FINAL

SUPPLEMENTAL ENVIRONMENTAL ASSESSMENT (SEA) OF THE EFFECTS OF ELECTROMAGNETIC RADIATION FROM THE WSR-88D RADAR

PREPARED FOR

THE NEXT GENERATION WEATHER RADAR
JOINT SYSTEM PROGRAM OFFICE (JSPO)

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EXECUTIVE SUMMARY

FINAL SUPPLEMENTAL ENVIRONMENTAL ASSESSMENT (SEA) OF THE EFFECTS OF ELECTROMAGNETIC RADIATION FROM THE WSR-88D RADAR (FORMERLY NEXRAD)

The WSR-88D Program is currently being implemented by the Joint System Program Office (JSPO) formed by the U.S. Departments of Commerce, Transportation, and Defense (DOC, DOT, and DoD). The WSR-88D Program consists of the design, siting, construction, and operation of 116 National Weather Service (NWS) radars in the continental United States; 14 Federal Aviation Administration (FAA) radars in Alaska, Hawaii, and the Caribbean; and 22 DoD radars in the continental United States.

In 1984, JSPO published a Programmatic Environmental Impact Statement (PEIS) analyzing the potential environmental impacts of the system. The PEIS concluded that, "In general, construction and operation of the NEXRAD [now WSR-88D] system will have no significant adverse environmental impacts." In particular, it found that

> there is no reliable scientific evidence to suggest that, for the cases considered, exposure to RFR [radiofrequency radiation] from the NEXRAD radar will be deleterious to the health of even the most susceptible members of the population such as the unborn, infirm, or aged.

Since 1984, additional scientific research on the biological effects of exposure to RFR has been carried out. This Supplemental Environmental Assessment (SEA) focuses primarily on the results reported for that research to determine whether the earlier PEIS conclusion remains valid. In extending the earlier analysis, this SEA uses current radar performance specifications and field measurements made at the WSR-88D Operational Support Facility to recalculate and verify the strength of the electromagnetic field created by the radars during operation. The SEA also addresses the potential biological effects from exposure to the electromagnetic field created by power lines serving each radar. Finally, the document reexamines the potential for the creation of hazards to humans through electromagnetic interference with cardiac pacemakers, electroexplosive devices, and fueling operations.

Each WSR-88D unit will include a tower, ranging in height from 16.4 to 98.4 ft, topped by a 39-ft wide and 35-ft high radar dome; an equipment shelter; standby electrical generator; and a perimeter fence. The radars will emit electromagnetic signals at an assigned frequency in the range of 2700 to 3000 megahertz (MHz), a peak radiated power of 475 kilowatts (kW) or less, and an average radiated power of 1 kW or less. Sites for WSR-88D units are being selected to maximize the use of surplus or underutilized government property, reuse existing weather radar sites, achieve optimum network coverage, minimize construction costs, and prevent or reduce environmental impacts. The WSR-88D system will replace 43 existing WSR-57, 12 WSR-74S, and 1 WSR-74C radars operated by NWS, which are antiquated and unreliable. WSR-88Ds will be able to make velocity as well as reflectivity measurements; they will also have greater range

and resolution than the radars they replace because they use Doppler technology and improved data processing techniques.

The electromagnetic environment at any particular location consists of all natural and human contributions to electromagnetic fields arriving at that location. Naturally occurring nonionizing radiation includes the earth's magnetic field and electric fields in the atmosphere (most prominent during storms). Human sources include radio and television broadcast transmitters, radar and communications systems, home appliances such as microwave ovens, and power distribution lines. The U.S. Environmental Protection Agency (EPA) measured background levels of RFR (at 54 to 890 MHz, somewhat below the WSR-88D frequency band of 2700 to 3000 MHz) in 15 U.S. cities. It found that background RFR power density levels were generally below 0.000002 milliwatts per square centimeter (mW/cm^2).

The WSR-88D unit will emit a highly directed beam. During normal operation, the beam will be radiated in a pulse lasting either 1.57 or 4.71 microseconds (μs) and repeated up to 1,304 times per second as the radar antenna rotates through one 360° revolution every 15 to 75 seconds. At most locations, the WSR-88D unit will scan at elevation angles between +0.5° and +19.5°. However, at some mountaintop locations, scan angles may be as low as -0.5°.

During normal operation, based on the scan pattern that will cause the greatest power densities, WSR-88D units will generate a maximum average power density calculated at 0.6 mW/cm^2 at the surface of the radome. At 1000 ft from the radar at the antenna height, the average power density will decrease to 0.006 mW/cm^2. At the lowest tower height, the average power density at ground level of RFR emitted by the WSR-88D will not exceed 0.005 mW/cm^2. These calculations have been verified by field measurements.

Infrequently, WSR-88Ds will be operated for 5 minutes in a searchlight mode, that is, with the main beam aimed at a fixed point. This mode will be used only for maintenance and testing purposes. Because the radar beam will be fixed, average power densities will be higher than during normal operation. Within 800 ft of the antenna face, the power density averaged over 6 minutes will be 3.85 mW/cm^2. Averaged over 30 minutes, the power density will be 0.77 mW/cm^2.

In 1991, the Standards Coordinating Committee (SCC) 28 of the Institute of Electrical and Electronics Engineers (IEEE) adopted new guidelines for human exposure to RFR to replace the American National Standards Institute (ANSI) 1982 guidelines in the WSR-88D frequency range 2.7-3.0 GHz; the new limits for controlled environments (e.g., occupational exposure) are 9 to 10 mW/cm^2, averaged over any 6-minute period. Thus, WSR-88D emissions will not exceed those guidelines during either normal or searchlight-mode operation. The IEEE C95.1-1991 guidelines for exposure in uncontrolled environments (e.g., for the general population) to WSR-88D-frequency RFR are 1.8 to 2.0 mW/cm^2, averaged over any 30-minute period. During normal operation, WSR-88D signals will not exceed that level. Similarly, the guidelines will not be exceeded during searchlight-mode operation because operation in that mode will be restricted to one 5-minute period during any 30 minutes.

The scientific literature on possible biological effects (bioeffects) of exposure to WSR-88D RFR was carefully reviewed. Although RFR can heat molecules in the body and cause biological changes, RFR at the power densities of WSR-88D signals adds heat at rates well below the human body's ability to dissipate heat. Therefore, exposure to WSR-88D signals will not cause

the effects associated with high heat absorption. Close examination of numerous epidemiologic studies of human occupational or residential exposure to RFR yielded no convincing scientific evidence that chronic exposure to RFR at levels within the 1982 ANSI exposure guidelines or the IEEE (1991) revision is implicated in any detrimental human health effects. An even larger body of studies with laboratory animals also supports that conclusion. Ocular damage can result from excessive RFR exposure if the RFR levels cause tissue heating, which will not be the case for WSR-88D RFR emissions. Exposure to RFR that causes tissue heating has been shown to cause teratogenesis, developmental abnormalities, mutagenesis, effects on the immunological system, and effects on the endocrine system; however, the power densities of WSR-88D RFR will be too low to cause such heating and its associated effects. Early studies reported effects on the blood-brain barrier, the brain and the heart without heating in excess of the tissue's ability to dissipate heat. However, more recent studies with improved instrumentation and/or better controlled experimental conditions reveal that those earlier results probably were obtained erroneously. Subtle histochemical changes in the nervous system have been reported in animals at exposure levels as low as 0.5 mW/cm^2, the so-called "nonthermal" levels of RFR. However, there is no evidence that those changes are harmful. Thus, overall no scientifically valid evidence exists that chronic exposure to WSR-88D RFR will adversely affect the health of humans.

Cumulative effects, thermal or otherwise, from simultaneous exposure to multiple sources of RFR are not likely because measurements at WSR-88D sites have shown low background levels of electromagnetic radiation.

WSR-88D units will receive 3-phase, 208Y/120-volt (V), 200-ampere (A) electric service via overhead or underground power lines, similar to typical residential and commercial distribution lines, connected to the local electric grid. Underground power lines will generate negligible electric fields at ground level and magnetic fields at ground level that are comparable to those from above-ground power lines. Overhead power lines will generate an electric field level of about 1 volt per meter (V/m) and a magnetic field level of about 15 milligauss (mG) near ground level. This electric field level is considered to be harmless. The magnetic field level is similar to the upper value of levels measured in American homes and is far below 60 Hz magnetic-field exposure guidelines published by the International Radiation Protection Association (1000 mG for up to 24 hours per day for members of the general public). Several epidemiologic studies have found an association between the incidence of certain forms of cancer and the presence of nearby power lines. Other studies found an association between certain forms of cancer and job titles suggestive of occupational exposure to magnetic fields. A review of these studies indicates no conclusive evidence that magnetic fields *per se* are associated with cancer. However, considerable research into this question is underway. Based on current scientific evidence, no adverse health effects are expected from electric and magnetic fields from the WSR-88D power lines. Thus, given also that, in the majority of cases, the WSR-88D radar and its power lines will not be near businesses or residences, no adverse bioeffects are expected.

Operating frequencies and sites for individual WSR-88D units are being selected to minimize the potential for interference with other radars. The WSR-88D main beam could detonate electroexplosive devices such as blasting caps within 900 ft of the unit, but that is highly unlikely because the WSR-88D main beam will not strike the ground within that distance. Cardiac pacemakers or fuel handling operations are not expected to be adversely affected.

Therefore, the WSR-88D system will not cause significant adverse electromagnetic interference or hazards to systems.

The SEA review of research since 1984 found no scientific evidence that exposure to WSR-88D RFR will result in adverse biological impacts. Similarly, the review found that adverse effects are not expected to result from exposure to WSR-88D power-line fields. Finally, as concluded in 1984, no hazards will be created for operation of cardiac pacemakers, use of electroexplosive devices, or fuel handling.

In overall conclusion, therefore, JSPO finds that implementation of the WSR-88D Program will not cause significant adverse impacts on human health or hazards to electromagnetic systems. The earlier conclusion of the PEIS that construction and operation of the WSR-88D system would not cause significant adverse impacts on the human environment remains valid, and a Finding of No Significant Environmental Impact (FONSI) is warranted.

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FIGURES

TABLES

ABBREVIATIONS AND UNITS OF MEASUREMENT

AAMI	Association for the Advancement of Medical Instrumentation
ac	Alternating current
ACGIH	American Conference of Governmental Industrial Hygienists
AChE	Acetylcholinesterase
AF	Audiofrequency
AFOS	Automation of Field Operations and Services
AM	Amplitude-modulated
ANA	Automatic network analyzer
ANSI	American National Standards Institute
APTA	American Physical Therapy Association
ARMS	Adjusted root mean square
ARTCC	Air Route Traffic Control Center
ASR	Airport Surveillance Radar
ATP	Adenosine triphosphate
AWDS	Automated Weather Distribution System
AWIPS	Automated Weather Interactive Processing System
BBB	Blood-Brain Barrier
BER	Brainstem-evoked response
Bioeffects	Biological effects
BMR	Basal metabolic rate
BP	Benzopyrene
BRH	Bureau of Radiological Health
BS	Blue Spruce
Ca^{++}	Calcium ion
CB	Citizens band
CC&R	Covenants, Codes, and Restrictions
CEQ	Council on Environmental Quality
CI	Confidence interval
CIRRPC	Committee on Interagency Radiation Research and Policy Coordination
CL	Chemiluminescence
CNS	Central Nervous System
CP	Creatine phosphate
CPK	Creatine phosphokinase
CPM	Counts per minute
CR	Charles River
CR+	Complement-receptor-positive
CS 83	Corticosterone
CS 128	Conditional stimulus 128
CW	Continuous wave
DA	Dopamine
dc	Direct current
D/M	Mean dose (power density x duration}
DNA	Deoxyribonucleic acid
DOC	U.S. Department of Commerce
DoD	U.S. Department of Defense
DOT	U.S. Department of Transportation
DPC	Displayed pulse count
DPM/g	Disintegrations per minute per gram of tissue

DRL	Differential-reinforcement-of-low-rate
EA	Environmental assessment
ECAC	Electromagnetic Compatibility Analysis Center
EED	Electroexplosive device
EEG	Electroencephalogram
E-fields	Electric Fields
EHV	Extremely high voltage
EIS	Environmental impact statement
EKG	Electrocardiogram
ELF	Extremely low frequency
EMC	Electromagnetic compatibility
EMF	Electromagnetic field
EMI	Electromagnetic interference
EMP	Electromagnetic pulses
EMR	Electromagnetic radiation
EMS	Ethylmethane sulfonate
EPA	U.S. Environmental Protection Agency
F	Frequency
FAA	U.S. Federal Aviation Administration
FCC	U.S. Federal Communications Commission
FCMSSR	U.S. Federal Committee for Meteorological Services and Supporting Research
FRRS	Frequency Resource Record System
FI	Fixed-interval
FI-1	Fixed-interval, 1-min
FM	Frequency-modulated
FONSI	Finding of No Significant Impact
FR	Fixed-ratio
GCA	Ground control approach
GH	Growth hormone
GMF	Government Master File
Hb	Hemoglobin
HEW	U.S. Department of Health, Education, and Welfare
HF	High frequency
HFAL	High-frequency auditory limit
H-fields	Magnetic Fields
HMBA	Hexamethylene bisacetamide
5HT	Serotonin
HVA	Homovanillic acid
5HIAA	5-Hydroxyindole acetic acid
IEEE	Institute of Electrical and Electronics Engineers
IR	Infrared radiation
IRAC	Interdepartment Radio Advisory Committee
IRPA	International Radiation Protection Association
IRT	Interresponse-time
JDOP	Joint Doppler Operational Project
JPDP	Joint Program Development Plan
JSPO	Joint System Program Office
K+	Potassium ion
LD_{50}	Death in 50% of the subject test species
LDH	Lactic acid dehydrogenase
MEL	Murine erythroleukemic
MI	Myocardial ischemia
MLOS	Microwave line-of-sight
MOS	Military occupational specialtiy

MW	Microwave
Na+	Sodium ion
NE	Norepinephrine
NEPA	National Environmental Policy Act
NEXRAD	Next Generation Weather Radar
NIEMR	Nonionizing electromagnetic radiation
NIOSH	National Institute for Occupational Safety and Health
NK	Natural killer
NMC	National Meteorological Center
NOAA	National Oceanic and Atmospheric Administration
NOC	Naval Oceanography Command
NSPS	New Source Performance Standards
NTIA	National Telecommunication and Information Administration
NTR	NEXRAD Technical Requirements
NWS	National Weather Service
OSF	Operational Support Facility
OSTP	Office of Science and Technology Policy
OTP	Office of Telecommunications Policy
PCB	Polychlorinated biphenyl
PEL	Permissible exposure limits
PHA	Phytohemagglutinin
PMR	Proportionate mortality rate [or ratio]
PRF	Pulse repetition frequency
PRR	Pulse repetition rate
PSC	Posterior subcapsular cataract or Posterior subcapsular cortex
PSCI	Posterior subcapsular iridescence
PSD	Prevention of significant deterioration
PUF	Principal User Facility
PUP	Principal user Processing
RBC	Red blood cell
RDA	Radar data acquisition
RF	Radio frequency
RFEM	Radiofrequency electromagnetic
RFR	Radiofrequency radiation
RML	Radar microwave link
RMR	Resting metabolic rate
ROW	Right-of-way
RPG	Radar product generation
RR	Relative risk
RSEC	Radar spectrum engineering criteria
SA	Specific absorption
SAR	Specific absorption rate
SCC	Standards Coordinating Committee
SCE	Sister chromatid exchange
SE	Standard error of the means
SEA	Supplemental environmental assessment
SI	Stimulation index
SMR	Standardized mortality ratio
SRBC	Sheep red blood cell
SW	Shortwave
T4	Thyroxine
TD	Time-discrimination
TDWR	Terminal doppler weather radar
TEM	Transverse electromagnetic

TLV	Threshold limit value
TPA	12-O-tetradecanolyphorbol-12-acetate
UCS	Unconditional stimulus
USAF	U.S. Air Force
VI	Variable interval
WFO	Weather Forecast Office
WHO	World Health Organization
WSFO	Weather Service Forecast Office
WSO	Weather Service Office
WSR-88D	NEXRAD Radar

A	Ampere
A/m	Amperes per meter
cal/g	Calories per gram
cm	Centimeter
cm^2	Square centimeter
d/wk	Days per week
ft	Feet
G	Gauss
g	Gram
g/d	Grams per day
GHz	Giga-hertz
h	Hour
h/d	Hours per day
h/wk	Hours per week
J	Joule
J/kg	Joules per kilogram
kg	Kilogram
kHz	Kilohertz
kV	Kilovolt
kV/m	Kilovolts per meter
kW	Kilowatt
MHz	Mega-hertz
m	Meter
µg	Microgram
mG	Milligauss
min	Minute
min/d	Minutes per day
µl	Microliter
mJ	Millijoule
mJ/cm^2	Millijoules per square centimeter
ml	Milliliter
µs	Microsecond
ms	Millisecond
mW	Milliwatt
mW/cm^2	Milliwatts per square centimeter
nA	Nanoampere
nm	Nanometer
pps	Pulses per second
s	Second
V	Volt
V/m	Volts per meter
W	Watt

Wk	Week
W/kg	Watts per kilogram
W-min/g	Watt-minutes per gram

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I INTRODUCTION

I.A. WSR-88D PROGRAM

The WSR-88D Program is a joint effort by DOC, DOT, and DoD to improve the United States' electronic capabilities for detecting and tracking meteorological phenomena. The agencies cooperating to implement the WSR-88D Program are the NWS in DOC, the FAA in DOT, and the U.S. Air Force and Naval Oceanography Command (NOC) in DoD. The three departments have formed JSPO to manage the implementation of the WSR-88D Program. JSPO is responsible for:

- Development of engineering specifications for WSR-88D units
- Selection of sites for individual units
- System compliance with environmental regulations
- Engineering design for individual sites
- Procurement and installation of radar units
- Testing of units to ensure proper operation.

The WSR-88D system will consist of 116 NWS, 22 DoD, and 14 FAA weather radars in the continental United States, Alaska, Hawaii, and the Caribbean. The radar system will obtain information about the location, intensity, and movement of severe weather phenomena – tornadoes, thunderstorms, heavy precipitation, tropical cyclones, hail, high winds, and intense turbulence – throughout the United States and portions of the Caribbean.

I.B. ROLE OF THIS DOCUMENT

As a federal agency, JSPO is subject to the provisions of the National Environmental Policy Act (NEPA). To fulfill NEPA requirements, JSPO prepared a PEIS in 1984 that analyzed possible impacts to the human environment from implementation of the WSR-88D Program (then known as the Next Generation Weather Radar [NEXRAD]). JSPO has also prepared environmental assessments (EAs) that analyzed specific WSR-88D sites.

The 1984 PEIS examined the potential for human health effects to result from exposure to NEXRAD electromagnetic radiation (EMR), among other issues. Since 1984, additional scientific data on biological effects (bioeffects) from exposure to EMR have been generated. In addition, the engineering specifications for the WSR-88D units have been defined and applied, and electromagnetic measurements have been made at the WSR-88D Operational Support Facility (OSF).

In principle, the new research on EMR bioeffects and changes in WSR-88D design could change the conclusions of the PEIS. Therefore, in keeping with Chapter 2, Section 10 of the National Oceanic and Atmospheric Administration (NOAA) *Directives Manual* and the Council on Environmental Quality (CEQ) regulations implementing NEPA, JSPO is preparing this SEA,

which examines the potential for human health effects to result from implementation of the WSR-88D Program. The SEA focuses on the latest scientific research on radiofrequency radiation (RFR) and power-line field bioeffects.

II PURPOSE AND NEED FOR THE ACTION

The WSR-88D system will be designed, constructed, and operated to achieve the following objectives:

- Increase the average tornado warning time
- Improve the accuracy of descriptions about the location and severity of thunderstorms and the ability to distinguish between severe and less-than-severe storms
- Improve the detection of damaging winds and hail
- Improve the safety of aircraft operation by detecting and measuring the wind shear and turbulence associated with thunderstorms
- Provide improved rainfall estimates for use in flash flood warnings
- Reduce the size of warning areas to minimize unnecessary warnings
- Substantially reduce the number of false hazardous weather warnings
- Minimize failure to detect hazardous weather due to radar outages
- Help weather forecasters by improving distribution and display of radar information
- Detect hazardous weather conditions throughout the 50 states and at overseas locations specified by WSR-88D operators
- Maintain annual operations and maintenance costs at the same level as that of the radar systems to be replaced (excluding the cost for radars in areas not currently covered).

The weather information collected and disseminated by the WSR-88D system will benefit the aviation community and water resource managers. The safety and economy of air travel will increase as a result of improved means for avoiding severe weather and better route planning and flight control. The quality of weather advisories to pilots will improve. Advice about safe and optimum routing around hazardous weather will be provided, reducing encounters with severe weather.

The WSR-88D system will generate accurate rainfall estimates and storm path data, thereby enabling River Forecast Centers to quantify storm runoff over a watershed and to adjust reservoir levels in anticipation of increased runoff. Improved water management will benefit farmers through more efficient irrigation and reduced soil erosion, utilities through more relatively inexpensive hydroelectric power, and river commerce through better river stage forecasts. The WSR-88D system will also significantly improve flash flood warnings by estimating rainfall amounts more accurately, resulting in warnings that are expected to save 130 to 150 lives annually and reduce property losses by 15 to 20%.

The existing basic weather radar network operated by NWS consists of 43 WSR-57, 12 WSR-74S, and 1 WSR-74C radars. Because of difficulties in obtaining spare parts, most, if not all, of the existing radars are expected to be decommissioned when the WSR-88D system is fully operational. However, some interest has been expressed in retaining a few of the existing radars for research needs or to supplement the coverage of the WSR-88D system. The final number of the radars to be retained, if any, is unknown.

III PROPOSED ACTION AND ALTERNATIVES

III.A. THE WSR-88D PROGRAM

III.A.1. Program Development Phases

The WSR-88D Program consists of the major development and acquisition phases shown in Figure III.1. The system definition phase, which is now complete, resulted in the specification of system design requirements. The phase concluded with proposals from industry for the competitive validation phase, during which two contractors developed preproduction engineering models for test and evaluation. On the basis of evaluation of those models, Unisys Corporation was selected to build and install WSR-88D units. Initial test and evaluation have been completed on the prototype unit – the Operational Support Facility (OSF) – at Norman, Oklahoma.

Currently, locations for installation of WSR-88D radars are being selected. Site selection consists of three steps: (1) using readily available information, the suitability of one or more candidate sites in a each area of interest is initially assessed; (2) sites judged to be satisfactory are then visited to gather information; and (3) after analysis of that information, the most promising candidate site is selected for each area and an in-depth survey is conducted at each selected site.

III.A.2. Environmental Review

The 1984 PEIS constitutes the foundation for the environmental process for the WSR-88D Program. The basic facilities and equipment features of the proposed action were sufficiently well known in 1984 to judge their potential environmental impacts in general terms. However, because the actual sites for the radars had not been selected at that time, it was not possible to assess whether the potential impacts would occur at specific sites and, if they did, whether they would be significant. Therefore, the WSR-88D Program provides for preparing site-specific EAs. For many locations, specific sites have been selected, and JSPO has issued site-specific EAs. A site-specific EA is typically prepared after the in-depth site survey – the third step in the site selection process – using additional information pertinent to the impact, or impacts, of concern gathered during the survey. To date, no site-specific EISs have been found necessary.

III.B. THE WSR-88D SYSTEM

III.B.1. Overview

The WSR-88D system will consist of a network of WSR-88D units. Each WSR-88D unit is arranged in accordance with user area and point coverage requirements and the location and suitability of user facilities. Whenever feasible, units are being collocated with user facilities. For example, WSR-88D sites are often collocated with other NWS operational facilities, such as weather forecast offices (WFOs).

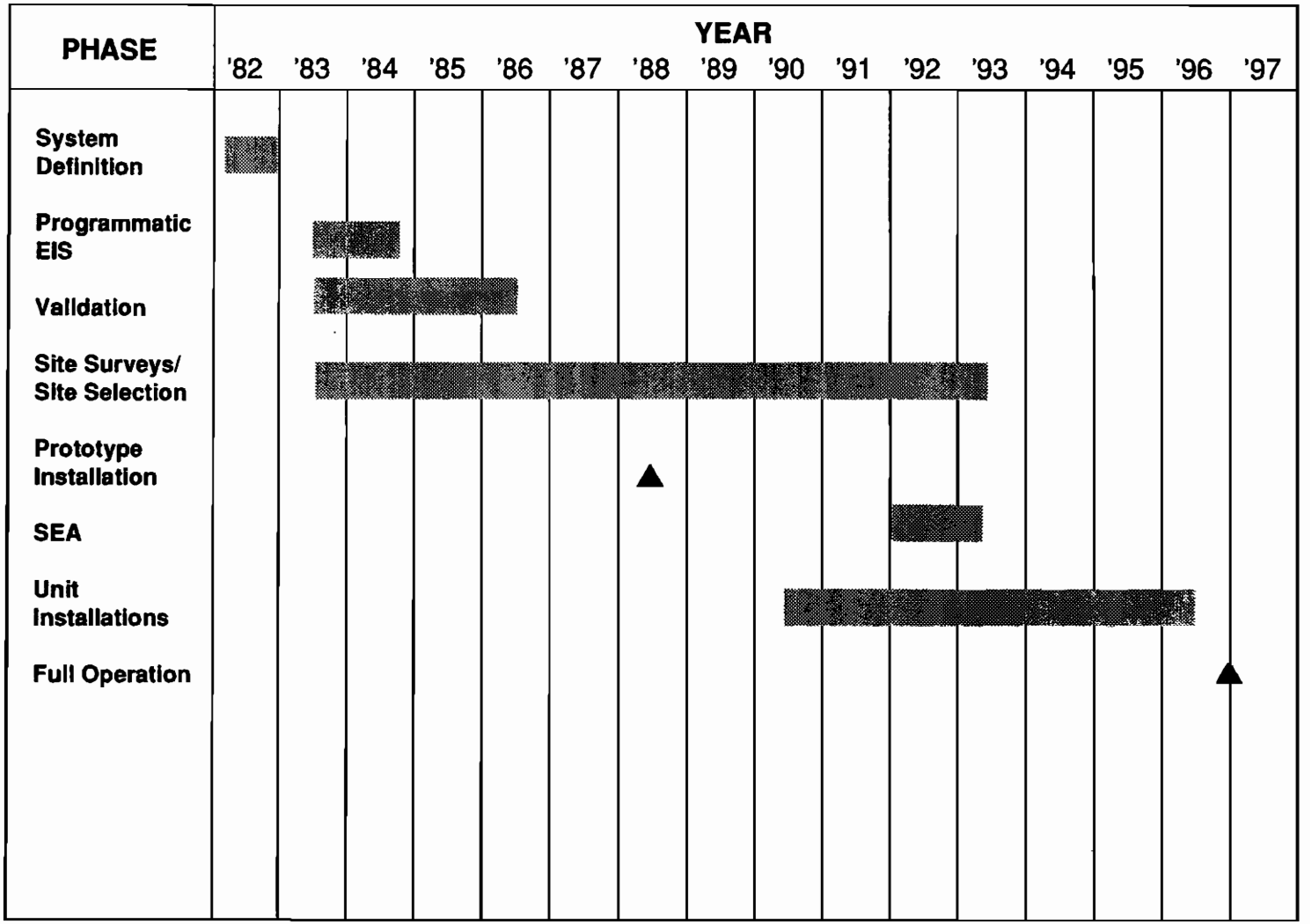


Figure III.1 WSR-88D Program Implementation Schedule

Where the radar is remotely located, radar data will be relayed to the WFO or other data receiver by T1 carrier systems, the standard means for high-speed digital data transmission used by telephone companies in the United States. Dedicated microwave line-of-sight (MLOS) systems, instead of T1, will transmit radar data from units at Los Angeles, California; Medford, Oregon; Missoula, Montana; and Oklahoma City, Oklahoma.

III.B.2. Description of the Network

Meeting radar coverage requirements is the primary consideration in selecting a radar site. In general, the meteorological site selection criteria call for locating radars where climatological records indicate that a significant threat of hazardous meteorological conditions occurs and where the radars have a clear view in the directions from which the most damaging weather typically approaches.

Existing weather radar sites are prime candidates. In most cases, they are at good locations for tracking weather conditions; the sites are available for government use; and the needed utilities, access, and compatible features exist. Generally, all existing user facilities and installations, including weather radar installations, WFOs, airports, military bases, and other government property in a given area are assessed and are given first consideration because of their prospective availability. Notwithstanding the likely suitability of existing sites as a whole, in studying specific areas, each site is scrutinized to determine its adequacy (e.g., availability of space and power) and the degree to which it meets user requirements.

The WSR-88D system will consist of 116 NWS radars in the continental United States, 14 FAA radars in Alaska, Hawaii, and the Caribbean; and 22 DoD radars in the continental United States. Table III.1 lists the general locations of the WSR-88D units. Figure III.2 shows the locations of NWS and DoD WSR-88D units. Figure III.3 shows the locations of FAA offshore WSR-88D units.

III.B.3. Description of the WSR-88D Unit

Like the radars that it is to replace, WSR-88D's transmitted signal consists of short pulses formed into the shape of a slender pencil beam by the antenna, which scans through 360° in azimuth by continuous rotation about a vertical axis. The pulses are separated by relatively long silent intervals for listening. WSR-88D differs from the older weather radars in its larger antenna, greater peak power, and shorter minimum time between pulses (roughly half as long). WSR-88D will operate in a frequency range of 2700 to 3000 MHz. Figure III.4 shows a typical WSR-88D unit.

Table III.1
PLANNED WSR-88D UNITS

NWS Locations (116)

Aberdeen SD	Des Moines IA	Louisville KY	Quad Cities IA/IL
Albany NY	Detroit MI	Lubbock TX	Raleigh/Durham NC
Albuquerque NM	Dodge City KS	Marquette MI	Rapid City SD
Alpena MI	Duluth MN	Medford OR	Reno NV
Amarillo TX	Elko NV	Melbourne FL	Riverton WY
Atlanta GA	El Paso TX	Memphis TN	Roanoke VA
Austin/San Antonio TX	Eureka CA	Miami FL	Sacramento Valley CA
Baltimore/WDC	Fargo/Great Falls ND	Midland/Odessa TX	Salt Lake City UT
Billings MT	Flagstaff AZ	Milwaukee WI	San Angelo TX
Binghamton NY	Glasgow MT	Minneapolis/St Paul MN	San Diego CA
Birmingham AL	Goodland KS	Missoula MT	San Francisco Bay Area CA
Bismark ND	Grand Island NE	Mobile AL	
Boise ID	Grand Junction CO	Morehead City NC	San Joaquin Valley CA
Boston MA	Grand Rapids MI	Nashville TN	Seattle/Tacoma WA
Brownsville TX	Great Falls MT	New Orleans/Baton Rouge LA	Shreveport LA
			Sioux Falls SD
Buffalo NY	Green Bay WI	New York NY	Spokane WA
Burlington VT	Greer, SC	Norfolk/Richmond VA	Springfield MO
Cedar City UT	Houston/Galveston TX	Norman OK	St Louis MO
Central Illinois	Indianapolis IN	North Platte NE	Tallahassee FL
Central PA	Jackson MS	Oklahoma City OK	Tampa Bay FL
Charleston SC	Jacksonville FL	Omaha NE	Topeka KS
Charleston WV	Kansas City MO	Paducah KY	Tucson AZ
Cheyenne WY	Key West FL	Pendleton OR	Tulsa OK
Chicago IL	Knoxville/Tri-Cities TN	Philadelphia PA	Wichita KS
Cincinnati OH	La Crosse WI	Phoenix AZ	Wilmington NC
Cleveland OH	Lake Charles LA	Pittsburgh PA	Yuma AZ
Columbia SC	Las Vegas NV	Pocatello ID	
Corpus Christi TX	Little Rock AR	Portland ME	
Dallas/Ft Worth TX	Loring/Caribou ME	Portland OR	
Denver CO	Los Angeles CA	Pueblo CO	

FAA Off-Shore Locations (14)

Anchorage AK	Grand Turk, BWI	Molokai HI	South Hawaii HI
Bethel AK	Kamuela HI	Nome AK	South Kauai HI
Fairbanks AK	King Salmon AK	San Juan PR	
Georgetown, BAH	Middleton Island AK	Sitka AK	

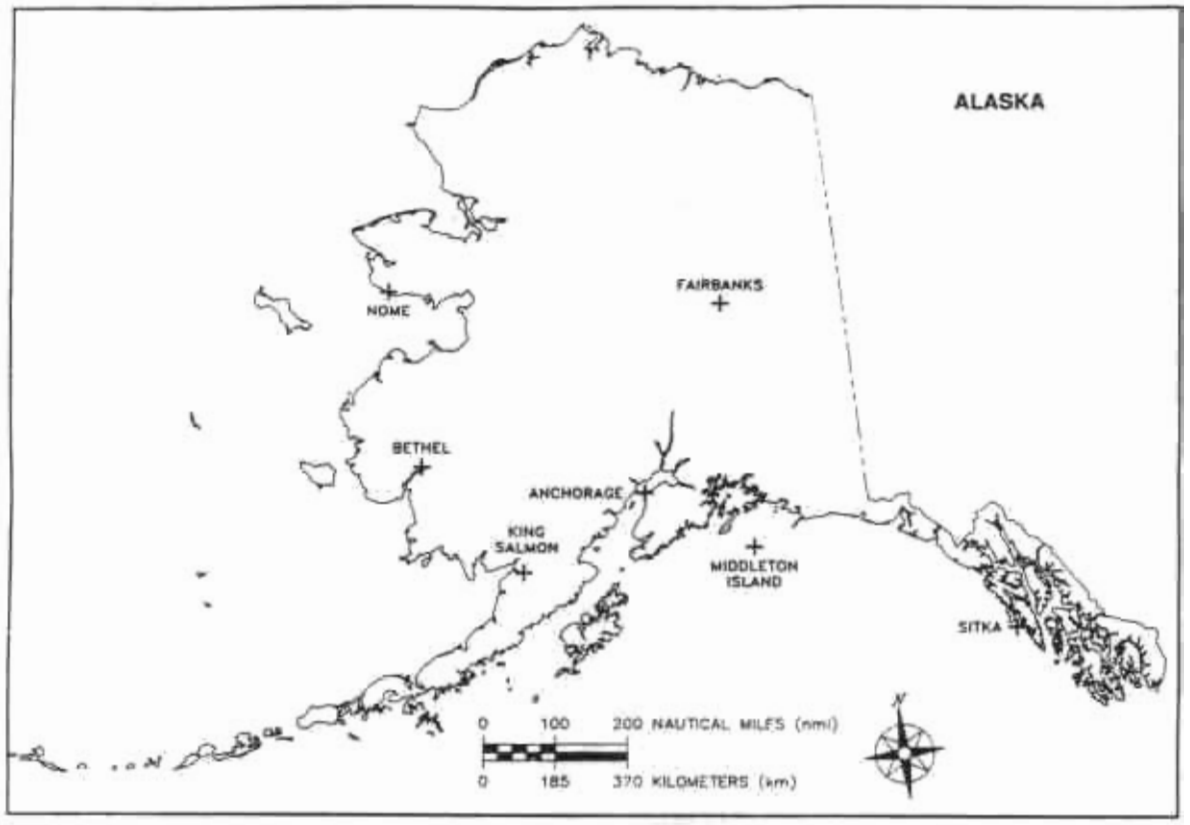
Table III.1 (Concluded)

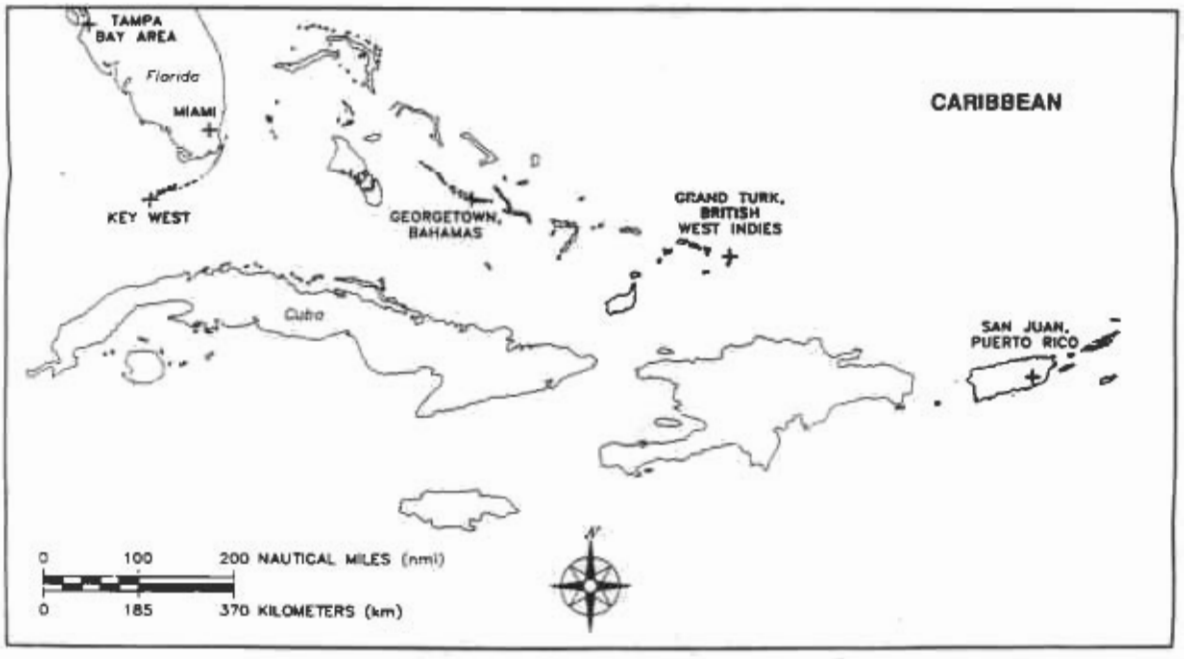
DoD Locations (22)

Beale AFB, CA	Frederick, OK	Vance AFB, OK
Cannon AFB, NM	Griffiss AFB, NY	Vandenburg AFB, CA
Central Texas, TX	Holloman AFB, NM	
Columbus AFB, MS	Keesler Training, MS	
Dover AFB, DE	Laughlin AFB, TX	
Dyess AFB, TX	March AFB, CA	
Eastern Alabama, AL	Minot AFB, ND	
Edwards AFB, CA	Moody AFB, GA	
Fort Campbell, KY	Northwest Florida, FL	
Fort Rucker, AL	Robins AFB, GA	

Source: JSPO

FIGURE III.2 WSR-88D System (locations for NWS and DoD Units) in Continental U.S.





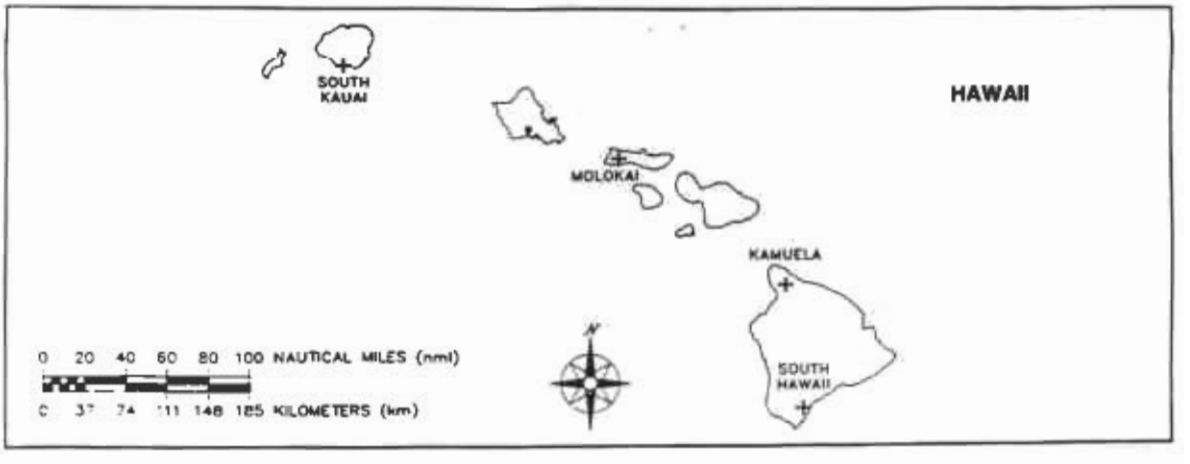


FIGURE III.3 Locations for FAA WSR-88D Units in Alaska, Hawaii, and the Caribbean

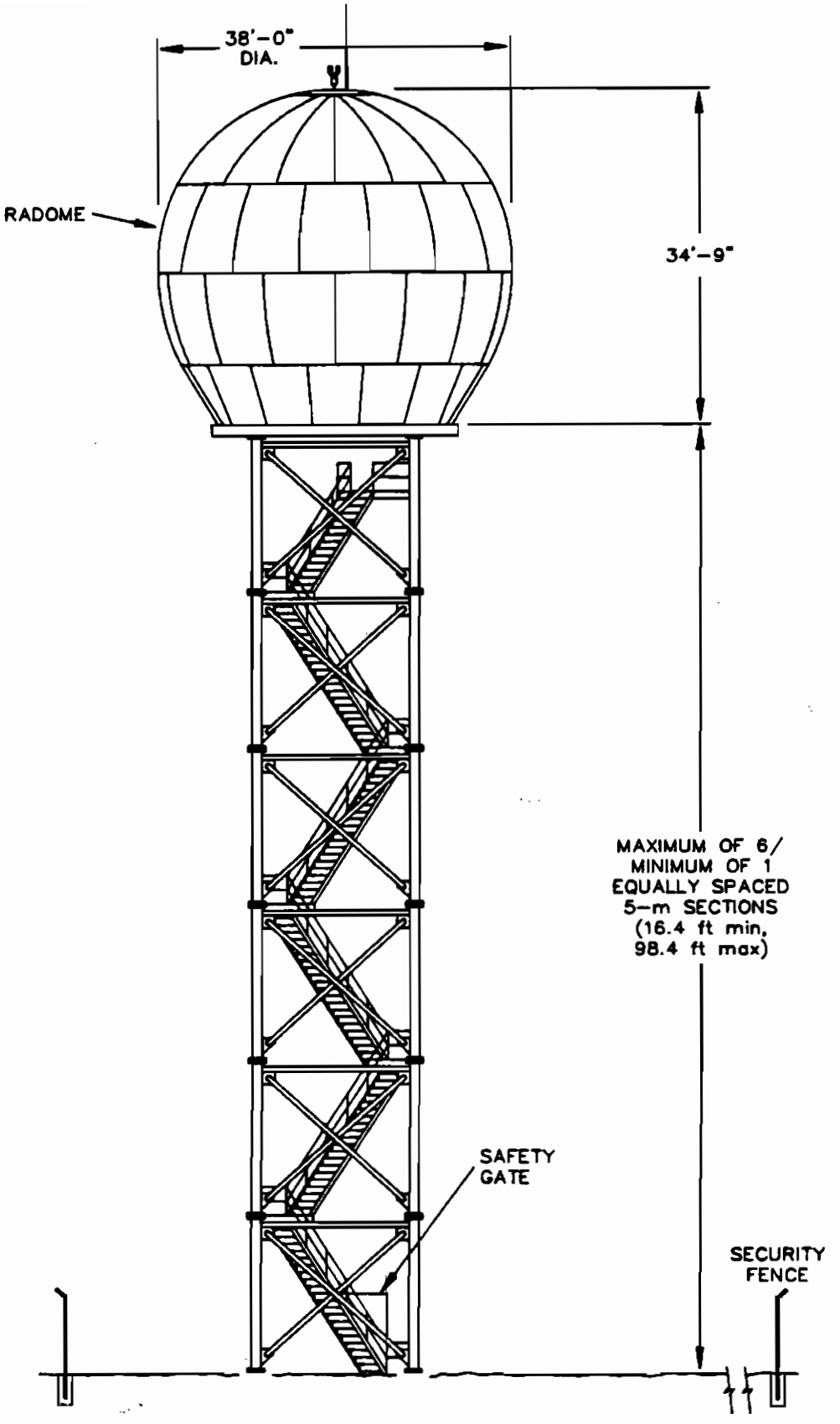


FIGURE III.4 Typical WSR-88D Unit (Profile)

The physical installation of a WSR-88D unit depends in large part on the site selected. Figure III.5 presents a conceptual site layout for a new site where the radar is not collocated with a user facility. The site contains an antenna tower, an equipment building that houses the transmitter and receiver, and utility lines. The tower, building, and adjacent equipment are surrounded by a 7-ft-high chain-link security fence. The radar antenna is enclosed in a nearly spherical radome about 35 ft in height mounted on a tower, which will range in height from 16.4 ft to 98.4 ft, depending on the surrounding terrain and nearby human obstructions. The location of the tower establishes the layout for the remaining facilities. Figure III.6 shows a schematic drawing of a typical collocated WSR-88D unit and a 5000-square-foot (ft^2) NWS WFO.

The peak power radiated by the radar will be 475 kW. The duration of each pulse will be either 1.57 or 4.71 microseconds (μs); depending on the pulse duration, pulses can be repeated at any rate between 318 and 1304 pulses per second (pps). The combination will be chosen so that the average radiated power will be about 1 kW. Section V.A provides more information about radar operation and the radiated beam.

III.C. ALTERNATIVES TO THE WSR-88D PROGRAM

The 1984 PEIS examined five alternatives to the WSR-88D system: continuation of the existing system, a new non-Doppler system, a new coherent non-Doppler system, a mixed system of new Doppler and non-Doppler radars, and satellite systems. No action and postponement of the action were also considered. That analysis determined that the WSR-88D system was preferable because none of the alternatives provided the advantages of an all-Doppler system (PEIS Section 2.4). There is no new information that would alter that conclusion.

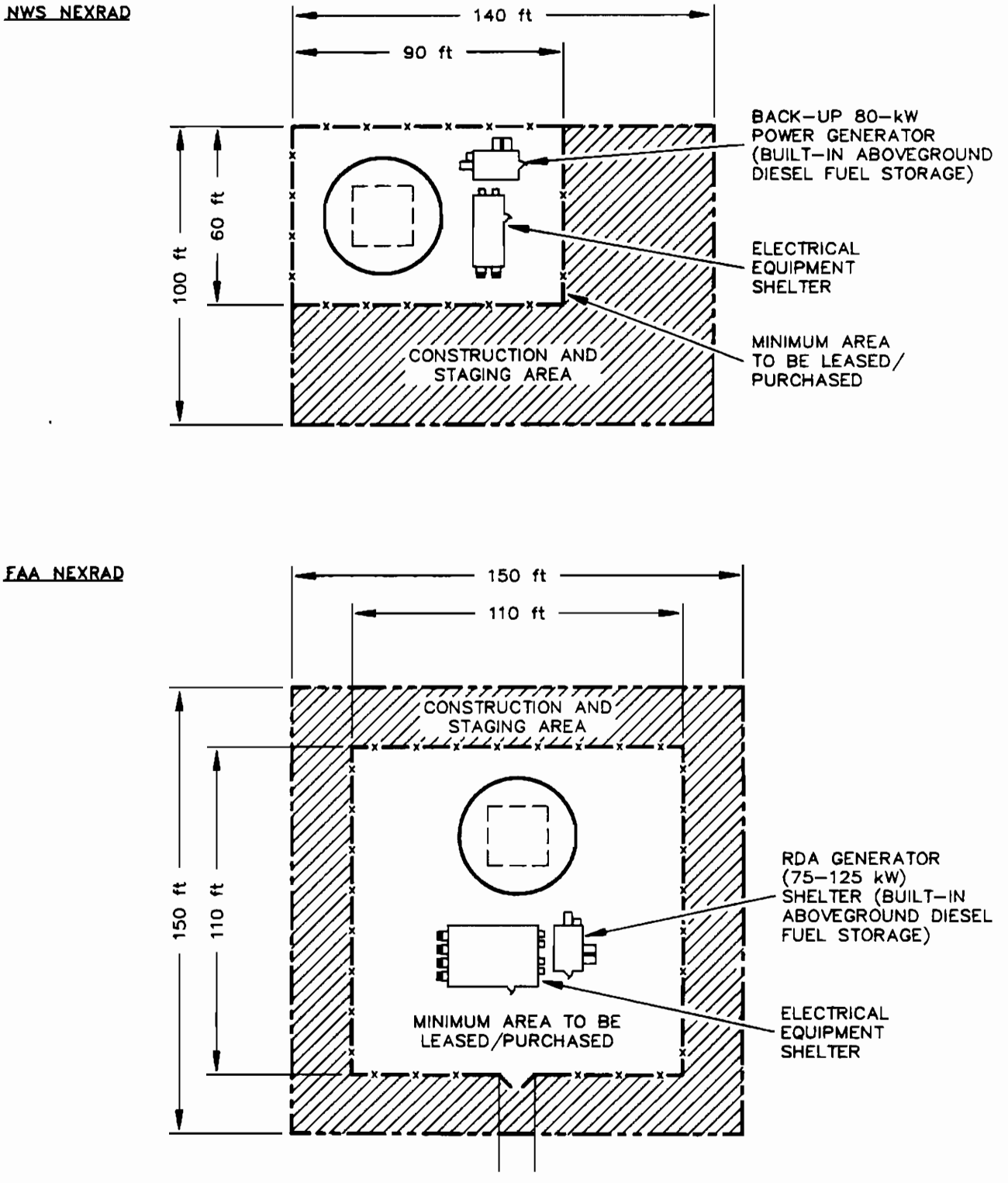


FIGURE III.5 Typical Layout Plans for NWS and FAA Radar Sites

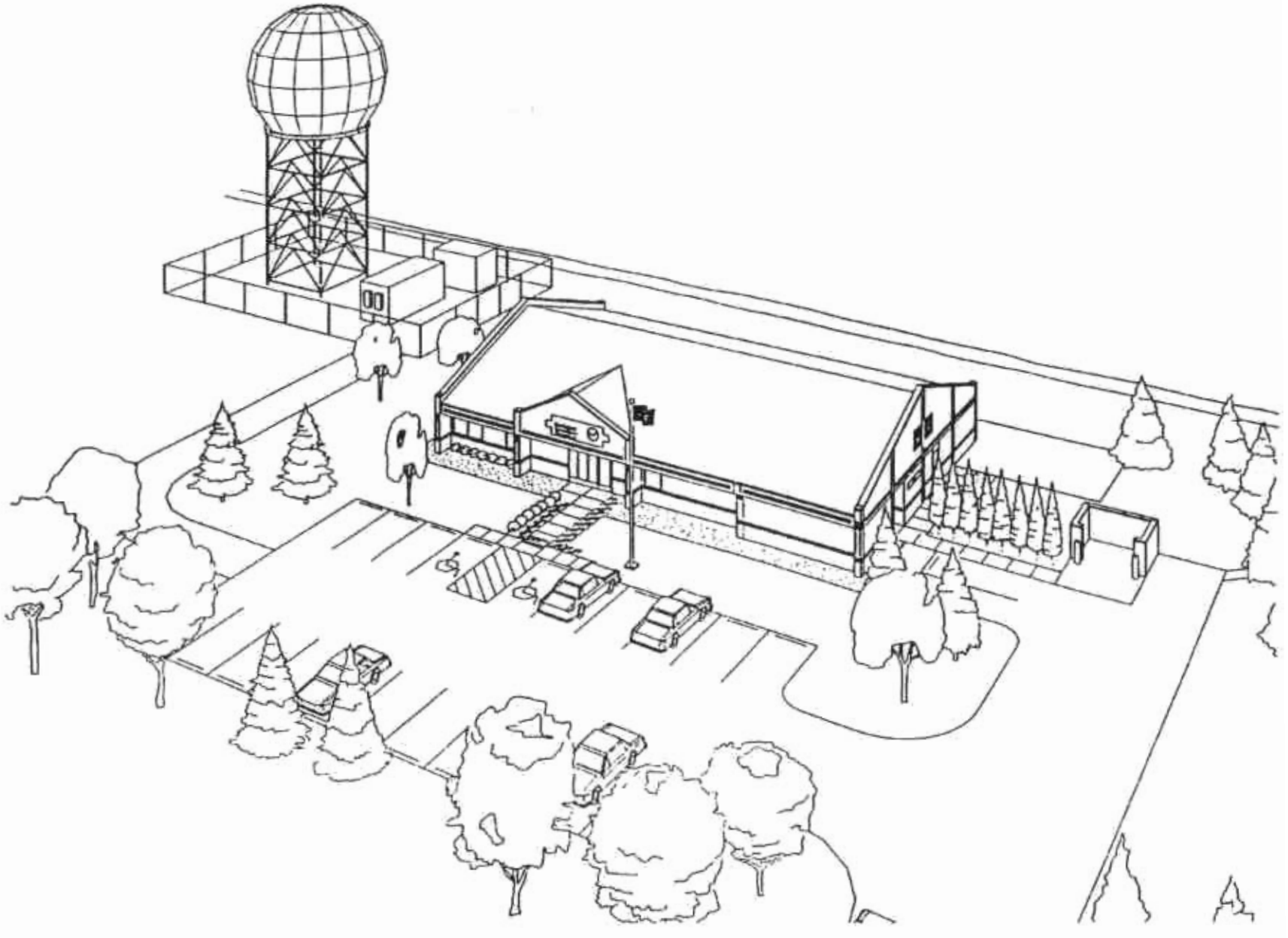


FIGURE III.6 Schematic of Typical Collocated Office/Radar Site

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IV AFFECTED ENVIRONMENT

IV.A. INTRODUCTION

The major focus of this SEA is the potential effects that could result from exposure of biological organisms to RFR, whether created by WSR-88D radars, microwave line-of-sight (MLOS) communications systems, or power lines supplying electricity to the radars. However, hazards to humans can also result from RFR effects on electrical and other systems. Section V.E analyzes the potential for WSR-88D RFR to interfere with normal operation of radios and televisions, other radars, electroexplosive devices (EEDs), fuel handling, and cardiac pacemakers. As background for Section V, this section presents information about the origins of the electromagnetic environment, the electromagnetic spectrum and its use, measurements of ambient electromagnetic conditions, and nonionizing vs. ionizing radiation.

IV.B. THE ELECTROMAGNETIC ENVIRONMENT

The electromagnetic environment consists of electric fields (E-fields) created by electric charges and of magnetic fields (H-fields) created by moving electric charges. The electromagnetic environment at a particular location and time consists of all the electromagnetic fields that are arriving there from numerous sources – both human and natural. Some of these fields are used for communication or for radiolocation (radar). The electromagnetic spectrum in an area is a continuously usable, renewable resource whose dimensions consist of amplitude, time, frequency, and space. In areas large enough to permit sufficient geographic separation of users, the spectrum will accommodate numerous users on the same frequency simultaneously. In smaller areas, the spectrum will accommodate a large number of simultaneous users only if they are sufficiently separated in frequency. A high-power signal can mask a low-power signal on the same frequency.

The electromagnetic environment at any single point can change almost instantaneously and, at a given instant, it will not be the same at two points a few feet distant from one another. Therefore, it is generally convenient to deal with averages over time and space. When sufficient incentive exists, certain features of the electromagnetic environment can be measured and documented. However, because of the high cost of doing so, attempts are seldom if ever made to define the electromagnetic environment simultaneously over wide frequency ranges, large geographic areas, and long periods. Most attempts at defining the spectrum are thus of limited scope and seek only to provide answers to particular questions such as, "Is the radio noise at this location low enough to successfully operate a sensitive receiving system?" or "Are the land-mobile radio bands in Chicago too crowded to accept additional users?" Some measurements have been made in the Los Angeles area of the portion of the electromagnetic spectrum that will be most affected by the WSR-88D radar. The measurements showed that in certain areas part of the spectrum is crowded with other radar signals.

Some of the human contributions to the electromagnetic environment in the vicinity of potential WSR-88D radar sites are intentional, but others are accidental and incidental to some

other activity. Radio (and radar) signals are intentional human contributions. The electromagnetic environment in any area consists in part of signals from broadcast radio and TV stations, from local or transient amateur and .i.citizens band (CB;) operators, from air navigation aids, from passing aircraft, from satellites that provide cable TV programming, and so on. Because some signals can be reflected back to the earth at great distances by high-altitude ionospheric layers, part of the electromagnetic environment in any area consists of transmissions propagated by sky wave from stations thousands of miles away. Signals from the WSR-88D radar and others that use the same part of the spectrum do not propagate by sky wave and generally do not propagate very far beyond the horizon.

The unintentional human contributions to the electromagnetic environment include noise radiated by power lines, fluorescent lights, household lighting dimmer switches, household appliance motors, computers, hand-held calculators, and so on. A major contributor is the automobile ignition system, which radiates a pulse of energy over all the communication bands with each spark-plug firing. Although such electromagnetic noise is a major feature of some parts of the spectrum, it is not a concern in the WSR-88D portion of the spectrum.

Nature contributes noise to the electromagnetic environment, and it can do so in a marked fashion. Lightning strokes in distant storms – in Africa and South America, for example – act as powerful transmitters and cover a wide frequency band. Their "signals" propagate by sky wave to regions far away, causing "static" in radios in the United States. This noise is a major, intermittent feature in the electromagnetic spectrum used by standard AM broadcast stations and by HF international broadcast stations. In the electromagnetic spectrum that WSR-88D will use, radio noise from the sun and from the stars (galactic noise) is, in the absence of signals from other radars, the predominant feature.

Humans are generally incapable of sensing the electromagnetic environment or changes in it. However, radio (and radar) receivers are designed for that purpose: They extract small amounts of energy from specific portions of the spectrum that they then amplify and convert to a signal meaningful to the receiver's operator. This signal might be a picture on a TV channel, music on a local FM broadcast station, a long-distance telephone conversation, an air navigation signal, or a radar signal reflected from an airplane or a heavy rainstorm.

To use the electromagnetic environment for communication, radiolocation, or radionavigation, signal power must usually exceed the noise at the receiving location. (Some systems' complex processing schemes permit use of signals weaker than the noise.) Unless signal power exceeds the sum of natural noise, man-made noise, the receiver's own background noise, and other interfering signals in the receiver's bandwidth, it is usually impossible to extract the desired signal. In WSR-88D's portion of the spectrum, natural and human contributions to electromagnetic noise will be less significant than the radar receiver's internal noise or signals from other radars. Thus, signals from other radars are a main concern.

IV.C. ELECTROMAGNETIC SPECTRUM

Electromagnetic fields are classified by the frequency, or number of cycles per second (hertz, Hz); frequency is inversely related to wavelength. The electromagnetic spectrum ranges from the extremely low frequency (ELF) band to the band containing gamma and cosmic rays, at frequencies greater than 10^{19} Hz, as shown in Figure IV.1.

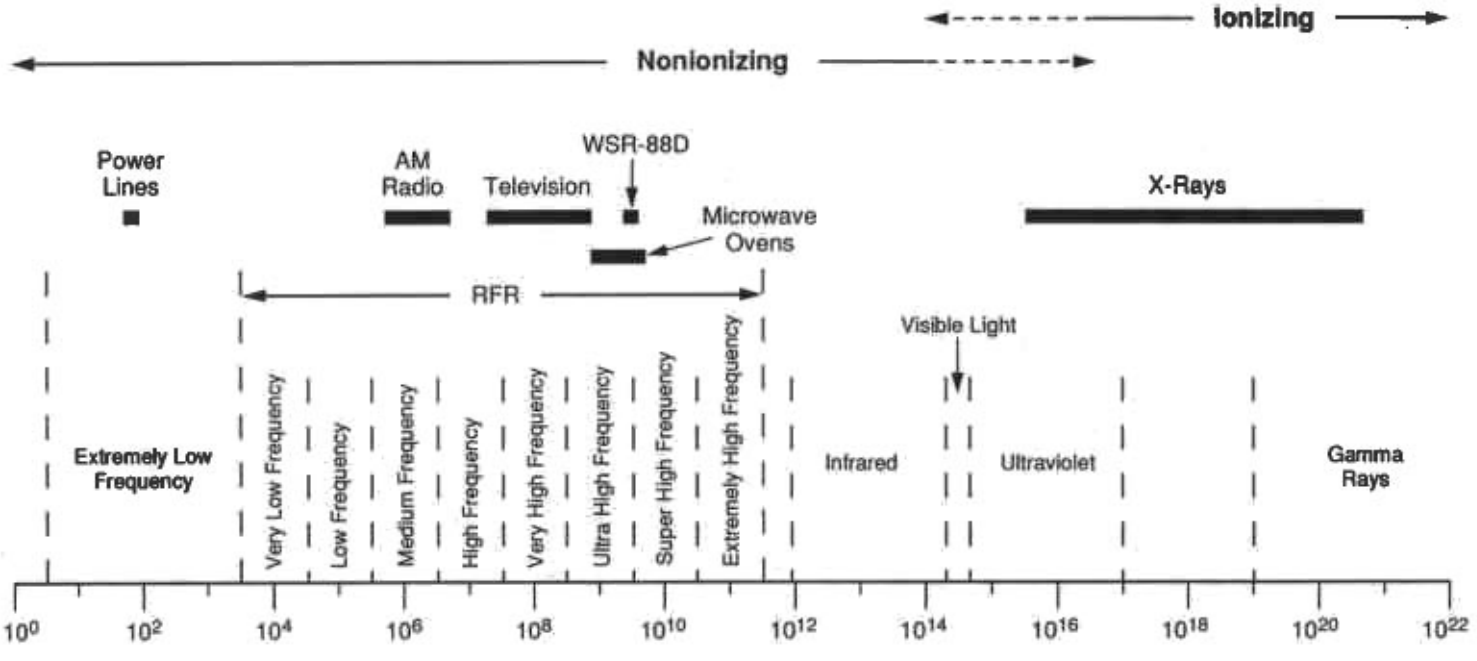

FIGURE IV.1 The Electromagnetic Spectrum

The WSR-88D radars will operate at frequencies between 2700 and 3000 MHz. Therefore, the band that is relevant to WSR-88D is the radiofrequency (RF) band between 3000 Hz and 300 gigahertz (GHz). The wave length of electromagnetic radiation at a frequency of 3000 MHz is about 4 in. In contrast, visible light at a frequency of 400,000,000 MHz, has a wavelength of 0.00003 in. MLOS units will operate at around 1800 MHz, which is also in the RF band. Equivalent terms sometimes used to describe RFR include radiofrequency electromagnetic (RFEM) fields, microwave radiation, and microwave fields.

WSR-88D radars will operate between 2700 and 2900 MHz when operationally feasible. In the United States, the 2700 to 2900 MHz band is allocated for the exclusive use of federal radars; air traffic control and weather radars are the band's primary users, and military radars are its secondary users. The band between 2900 and 3100 MHz is also allocated for use by radars. Government and nongovernment maritime radionavigation is the primary use, and military radionavigation is the secondary use. WSR-88D will use the lower half of the 2900 to 3100 MHz band when a frequency assignment in the 2700 to 2900 MHz band is not feasible. The upper half of that band – 3000 to 3100 MHz – is used by shipboard radars. The band below the WSR-88D band, from 2690 to 2700 MHz, is allocated for radioastronomy; to keep it free from interference, no U.S. radars are authorized to operate in that band.

The power lines supplying electricity to the WSR-88D units will operate at 60 Hz, which is in the ELF band. The wavelength of 60-Hz electromagnetic fields is about 3,100 miles.

IV.D. BACKGROUND CONDITIONS

EPA measured the intensities of RFR at 486 sites in 15 cities to estimate the population exposed to various power densities. The field strengths measured at each site were integrated over the frequency bands between 54 and 890 MHz and converted into equivalent power density (power density is the amount of energy intercepting a given area). The average equivalent power densities ranged from 0.000002 to 0.000020 mW/cm^2 (or 2 nanowatts/cm^2 [nW/cm^2]). EPA also measured RFR levels near FM radio and television transmitter towers and found that values averaged less than 0.01 mW/cm^2; the highest measured value was 1 to 7 mW/cm^2 (EPA, 1986).

The E-fields in the center of rooms of typical American homes, as measured by the World Health Organization (WHO), range from 0.8 to 13.0 V/m. H-fields may be fairly intense near certain appliances but decrease rapidly with distance away from the appliances. For instance, the H-field at 3 cm from a television set is up to 500 mG, which decreases to 2 mG at 1 m. At a distance of 1m from major appliances, residential magnetic fields vary between 0.1 and 8 mG (EPRI, 1991).

IV.E. IONIZING VS. NONIONIZING RADIATION

EMR with frequencies greater than about 10^{15} Hz have enough intrinsic energy to ionize (eject an electron from) molecules. EMR at lower frequencies, including the WSR-88D signal and power-line fields, cannot ionize molecules. Thus, the lower frequency EMR is called "nonionizing radiation." The examination of effects in this document is limited to nonionizing radiation.

Because of its relatively low frequency, nonionizing radiation has intrinsic energies far too low to ionize molecules within a body, but it can agitate molecules – the equivalent of adding

heat to the body. Absorption of RFR within a body as heat is usually not significant unless the rate at which heat is added is comparable to or higher than the body's metabolic rate. In high ambient temperatures and humidity, however, under which the body is close to the limits of its thermoregulatory system, absorption of considerably lower levels of RFR may cause thermal stress. Because molecular agitation ends shortly after RFR exposure ceases, the heat induced by successive low-level RFR exposures at short intervals is not cumulative.

By contrast, ionizing radiation has frequencies millions and trillions of times higher than those of RFR. The ejection of an electron from a molecule leaves the molecule positively charged, thereby greatly altering its own properties and enhancing its interactions with its neighboring molecules. The public is exposed in varying degrees to naturally occurring forms of ionizing radiation, including ultraviolet light from the sun, radioactive materials in the earth (including those released from mining and burning of coal and the release of radon gas), and gamma and cosmic rays from outer space. X-ray machines for various purposes (diagnosis, dentistry) and ultraviolet lamps for sun-tanning are two other artificial sources of ionizing radiation.

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V ENVIRONMENTAL CONSEQUENCES

V.A. INTRODUCTION

V.A.1. Scope

The WSR-88D unit will emit an electromagnetic signal at a frequency of 2700 to 3000 MHz in the radiofrequency band. In this section, we first calculate the power density of the signal and the decrease in power density with distance from the WSR-88D unit. (Appendix A describes the methodology used to calculate the intensity of the RFR.) Next, to assess the significance of possible human exposure to WSR-88D RFR, we summarize the findings of a review of the scientific literature on RFR bioeffects (Appendix B contains the review) and compare WSR-88D RFR levels to established standards for human exposure.

The power lines supplying electricity to the WSR-88D units will generate electromagnetic fields at a frequency of 60 Hz. Therefore, we calculate the strength of the electric and magnetic components of those fields. To assess the significance of possible human exposure to those fields, we then summarize the findings of the scientific literature on power-line field bioeffects and compare WSR-88D power-line field strengths to applicable existing standards. Because the WSR-88D power lines will not generate a focused, directed beam as the WSR-88D antenna will, the power-line field strength will diminish rapidly with distance and the potential for hazards to systems will be negligible.

This section closes with an analysis of the potential for indirect effects on human safety resulting from interference with other devices, including radios and televisions, radars, EEDs, fuel-handling activities, and cardiac pacemakers.

V.A.2. Assessment of Scientific Information and Risk

It is frequently asked whether guarantees can be given that no deleterious effects to people will result many years in the future from chronic exposure to low levels of RFR. In general, scientists believe that, if an experiment conducted many times indicates no effects, repeating it still more times will most likely show no effects as well. However, they have no way of fully guaranteeing that conclusion. To be completely sure about the absence of an effect of any agent would require that experiments be performed an infinite number of times on numerous biological systems and endpoints, with each experiment producing findings that indicate no deleterious effects. This is clearly an impossible task.

Instead, subject to practical considerations, experiments are conducted one or more times with specific numbers of subjects and stated conditions or assumptions regarding the agent(s) being investigated. Conclusions about the experimental findings are based on the use of appropriate statistical treatments of the data. The findings, including those indicative of no effects (negative results) related to the agent(s) studied, are usually given in probabilistic terms (confidence levels) because the data may contain varying degrees of uncertainty related to the

accuracy of the instrumentation used and/or uncontrolled variations in the populations and/or levels of the agents investigated.

The experimental evidence for any specific RFR bioeffect is derived primarily from the use of laboratory animals as surrogates for humans – a practice widely used in seeking or assessing possible effects of other agents. Thus, most projections about possible effects of RFR on humans are based on findings for species whose anatomies and functional characteristics differ significantly from those of humans; in addition, the RFR and exposure durations in the investigations may have differed considerably from those of any specific system (e.g., WSR-88D).

In conducting such experiments, animals must be housed, fed, and watered. Inappropriate cage materials or cage configurations can cause problems, including experimental artifacts that render findings questionable. Moreover, in evaluating reported results, it is also important to distinguish between effects and hazards. For example, if a laboratory animal is exposed to a strange noise of low or moderate intensity, several functional changes may occur in the hormonal secretions in its blood, in its sympathetic and parasympathetic nervous system, and in its behavioral orientation and alerting responses. If the noise stops or if the animal becomes acclimated to it, these effects may disappear. Exposure to noise, for example, has caused effects, but these are "normal" and fully reversible. If the noise is quite loud, on the other hand, the effects may include pathological changes, which have significant health implications.

Some investigations of human exposure to RFR have been made, either with volunteers or in the form of epidemiologic studies. For ethical reasons, very few of the former have been conducted. On the other hand, epidemiologic studies elucidate the distribution of death or disease – often for large human populations – and describe the probable factors that influence the distribution. However, for RFR as a possible cause of health effects, the values of the exposure parameters (especially the intensity levels and durations) vary widely with time for each individual and are highly variable from person to person. Moreover, in the absence of reliable information about exposure, those included in the "exposed" group are often selected by their occupation, a dubious method; it is also difficult to find a suitable control group that matches an exposed group in all important aspects except RFR exposure. Nevertheless, positive results of well-designed and conducted epidemiologic studies can point to the need for more specific research.

For other agents, possible effects at very low levels are predicted by extrapolating findings at higher levels on the basis of assumptions about the mathematical relationship between the level (or dose) of the agent and the degree of the effect. The existence or nonexistence of thresholds for deleterious effects of various agents has been debated at length. As a practical scientific matter, thresholds do exist for at least some substances, given that low concentrations of many natural substances are essential to maintain life, but are toxic at higher concentrations.

For RFR, such predictions by extrapolation from high to low levels are open to challenge. Many studies have yielded results showing that low-level exposures to RFR are not cumulative. In effect, the RFR energy that is continually absorbed at low incident power densities is readily dissipated and does not accumulate in the body. Those findings indicate that specific threshold levels must be exceeded to cause various RFR bioeffects. In a few studies, repetitive exposures within about 1 hour of one another at levels just below the thresholds for simple-exposure effects were reported to be cumulative, but those levels were relatively high. By contrast, certain

chemicals ingested in small quantities over time can accumulate in the body and become potentially harmful total doses.

V.B. WSR-88D RADAR AND ANTENNA CHARACTERISTICS

V.B.1. Introduction

A radar operates by transmitting a pulse of electromagnetic energy and then waiting to receive energy reflected back to it from some object (target) illuminated by the pulse. The radar interprets the interval between the transmission and the return as a measure of the distance from the radar to the target. To perform their basic functions, radars operate in very small units of time. The conventional unit is the microsecond (μs), or one-millionth of a second.

Most radar systems are designed and operated to detect objects such as ships, airplanes, or missiles. In contrast, the purpose of the WSR-88D system is to detect weather features such as rain or hail and storms such as hurricanes and tornadoes. This purpose has strongly influenced the design of the receiver's signal-processing components, but has had little effect on the transmitter design or on the antenna that radiates the transmitted pulses and receives echoes.

It is highly advantageous for a radar to concentrate its transmitted energy in (and to limit its receiving capability to) a relatively narrow beam. A narrow beam permits greater accuracy in regard to the direction in which the energy is sent and from which it returns; a narrow beam conserves available energy by concentrating most of it into a single direction; and a narrow beam permits reception of weaker return signals from a particular direction by discriminating against electromagnetic noise or extraneous, interfering signals that may arrive from other directions than can be achieved with wider beams.

Radar has long used parabolic reflectors, or dishes, to form beams in the same manner that the silvered reflector of an automobile headlight forms a beam from the light generated by the lamp's filament. To move the beam, the radar dish and the radiating element are typically rotated at a specified rate to sweep the beam past a given azimuth (radial direction) every few seconds to tens of seconds. Rotation about a second axis permits scanning at various angles of elevation.

V.B.2. Formation of the WSR-88D Beam

Power produced by a klystron amplifier tube is delivered by waveguide to a tapered feed horn, which is located at the focal point of the circular parabolic reflector. The power radiates from the feed horn, reflects from the parabolic surface, and passes out through the radome (radar dome) to form the beam. The radome is a nearly spherical, mostly plastic enclosure that protects the antenna and associated parts from dirt, wind, and weather, while providing unimpeded passage for the radar's signal.

WSR-88D's primary function is to detect weather conditions at distances up to almost 300 miles. To perform this function, the radar must radiate a strong, well-focused beam of electromagnetic energy and must provide a corresponding sophistication in receiving echoes that are returned. For WSR-88D these considerations have resulted in the design of a large antenna and concentration of most of the power in the main beam.

Figure V.1 illustrates the beam-forming process. Near the antenna face, the energy moves forward in an almost circular column of roughly constant diameter. At a greater distance, the

energy expands as a cone, with its apex at the center of the antenna face. A slender conical beam of this kind is commonly referred to as a "pencil beam." The intersection of the cone and cylinder occurs at about 640 ft from the antenna. A more detailed description of the beam is provided in the following sections.

The reflector forms the power radiated from the feed horn into a main beam with associated sidelobes, as indicated in the upper right-hand sketch in Figure V.1. The nulls of the main beam are separated by about 2.5°; the points at which the power density falls to half its maximum values are separated by no more than 1.0°. The sidelobes result from the inability of the reflector to concentrate all the energy into the main beam. The locations and relative intensities of the first few sidelobes are well known; their intensities are never greater than 0.32% [–25 decibels (dB)] that of the main beam. The large number of higher order (and very minor) sidelobes are distributed at various, almost random, angles. They have power densities no greater than 0.04% (–34 dB) that of the main beam and generally are smaller than 0.01% (–40 dB) that of the main beam.

To perform the surveillance function, the beam formed by the antenna scans continuously. Both the WSR-88D hardware and the software that controls it are flexible, and a wide range of scanning programs is possible. In all cases, the antenna will rotate continuously around the vertical axis as the elevation of the beam is held constant. Usually, the elevation of the beam will be changed at the end of each revolution.

WSR-88D can operate in any of several scanning modes. For our analysis we used scan pattern 31 because it leads to maximum possible values of time-averaged power densities at and near ground level. In scan pattern 31, the antenna rotates at a constant rate of 0.8 revolutions per minute (rpm) and makes 2 complete rotations at successive elevation angles of 0.5°, 1.5°, and 2.5°. It then makes one revolution each at elevation angles of 3.5° and 4.5° before repeating the entire cycle. Each cycle therefore includes 8 revolutions and lasts 10 minutes.

Another scan mode has a revisit time of 5 minutes, so that some locations could be struck by a total of 4 revolutions at a beam elevation of 0.5° in a 6-minute averaging period. However, the rotation speed in this mode is 3.1 rpm, so that the resulting average exposure is less than in pattern 31.

The WSR-88D antenna system is designed to prevent the transmitted beam from being directed below a minimum elevation angle of –1° or above a maximum elevation angle of 60°. At each site, the limit switches that control the minimum elevation angle will be set in accordance with the local terrain. Generally, the lower limit of the elevation angle will be set at 0.5°. In a few mountain-top locations, the lower limit may be -0.5°. System operators will routinely choose this limit so that the full strength of the main beam never strikes the ground; otherwise, operation would produce strong clutter signals that would greatly interfere with the desired observations.

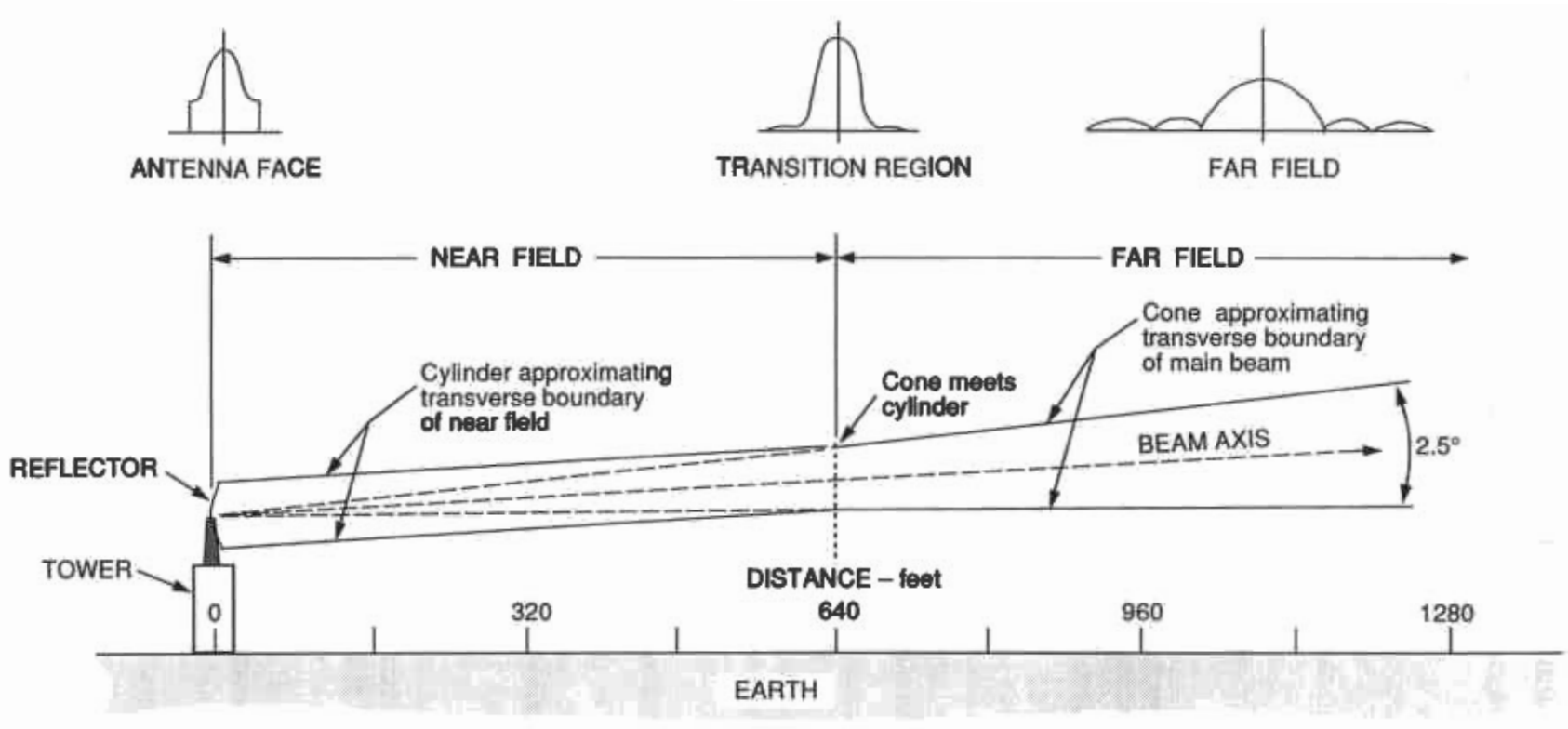

FIGURE V.1 Formation of the WSR-88D Beam

V.B.3. Pulse Shape, Duration, and Repetition Frequency

Many radar systems use the simple trapezoidal pulse shape shown in Figure V.2. By contrast, WSR-88D will use the more complicated waveform (also shown in Figure V.2) in which all the corners are deliberately rounded. The use of a shaped pulse narrows the frequency band occupied by the radar signal, thereby reducing the potential for interference with other systems that use the 2700- to 3000-MHz band. Of course, the waveforms of actual radars only approximate the idealized shapes shown in Figure V.2.

The pulse duration, T, indicated in Figure V.2 will be either 1.57 or 4.71 μs. The choice will depend on the range and nature of the weather feature that is of principal interest to system users at any given time.

The number of pulses transmitted per second is called the pulse repetition frequency (PRF). The value of PRF will be chosen in the range of 318 to 1304 pps. Long pulses will be associated with low values of PRF so that the average radiated power level will never exceed 1.0 kW.

The WSR-88D system is designed to operate at any fixed frequency in the 2700- to 3000-MHz range. The frequency used at a given site will be chosen on the basis of frequencies used by other radars (including other WSR-88D units) within about 150 miles of the site. Near major seaports, the frequency range of 2900 to 3000 MHz will be avoided to minimize the risk of mutual interference with marine radars that use this frequency range.

Table V.1 lists relevant WSR-88D characteristics. The WSR-88D system is often called a Doppler radar because it takes advantage of the Doppler principle to discriminate between moving targets – such as raindrops – and fixed objects. A moving target changes the frequency of the returned signal, and this frequency change is used in the Doppler processing unit of the WSR-88D receiver.

V.B.4. WSR-88D RFR Power Densities

The power density of the WSR-88D beam varies considerably between the near-field and the far-field regions. The near field is the area where the beam diameter is less than or equal to the diameter of the radiating antenna as shown in Figure V.1. In the near field, the power density varies about a mean value. In the far field, the beam diameter exceeds the antenna diameter and power density decreases as the inverse of distance away from the antenna. The distance at which the transmission from the near field to the far field occurs is not a precise distance, but a broad zone. It can be calculated either geometrically or mathematically. The geometrical approach yields a distance of 640 ft as shown in Figure V.1. The mathematical approach, explained in detail in Appendix A, yields a relatively similar distance of 800 ft.

The power density of the WSR-88D beam can be represented by either maximum pulse power – the maximum power level of a 1.57 to 4.71 μs pulse – or as the power averaged over a time period, usually minutes.

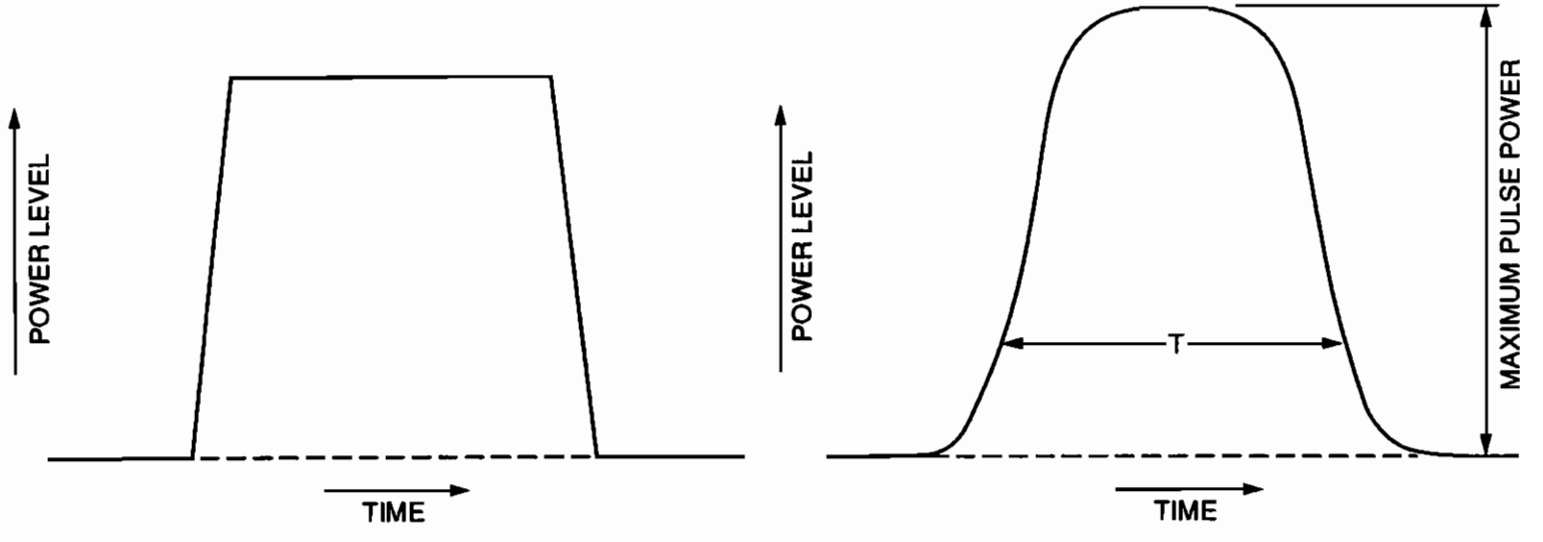

FIGURE V.2 Trapezoidal and Shaped Pulse Waveforms

Table V.1

CHARACTERISTICS OF THE WSR-88D RADAR

System Characteristics	Value
Frequency (MHz)	2700-3000
Wavelength[a] (ft)/(cm)	0.345/10.5
Maximum transmitter pulse power (kW)[b]	750
Maximum pulse power radiated (kW)[c]	475
Maximum duty cycle (%)[d]	0.21
Maximum average radiated power (kW)	1.0
Antenna diameter (ft)/(cm)	28/853
Antenna gain compared to nondirectional antenna (ratio)/(dB)[e]	35,500/45.5
Beam width at half power density (degrees)	1.0
First sidelobe relative power density – maximum (ratio)/(dB)	0.0032/-25
Other sidelobes maximum power density – relative to main beam (ratio)/(dB)	0.0004/-34
Azimuth scan rate, maximum (rpm)	5
Minimum elevation angle of beam (degrees)[f]	-1
Maximum elevation angle of beam (degrees)[g]	+60

Source: JSPO.

[a]At the midband frequency, 2850 MHz.

[b]Consistent with other sections of this document, the root-mean-square (RMS) value of the maximum value of the pulse is used when present.

[c]In typical systems, transmission line losses of 3 dB reduce the radiated power by a factor of 2 to 375 kW. The higher value 475 kW, corresponding to a 2-dB loss, is used to be conservative.

[d]The duty cycle is the percentage of time when the antenna is actually transmitting a signal.

[e]A gain of 45 dB is required by the system specification. The higher value, 45.5 dB, is used to obtain conservative estimates of RFR safety.

[f]In normal scanning, the beam elevation will not be less than +0.5°.

[g]In normal scanning, the beam elevation will not exceed +19.5°.

The maximum pulse power density of the WSR-88D beam in the near field is 2200 mW/cm^2. In the far field, maximum pulse power density decreases rapidly with distance from the antenna. Maximum pulse power density decreases to 100 mW/cm^2 at 3800 ft from the antenna and to 10 mW/cm^2 at 12,000 ft from the antenna.

The Institute of Electrical and Electronic Engineers (IEEE) Standards Coordinating Committee SCC C95.1-1991 guidelines for exposure to RFR use averaging periods of 6 and 30 minutes. Therefore, we present power density for the WSR-88D beam averaged over 6- and 30-minute periods. Figure V.3 shows the power density of the WSR-88D beam during normal operation in scan pattern 31, averaged over 6 minutes. In the main beam at 20 ft from the antenna – the distance of the protective radome – the average power density is 0.6 mW/cm^2; at 800 ft from the antenna, the near-field/far-field transition distance, the average power density is 0.008 mW/cm^2; at 1 mile, the power density is 0.0002 mW/cm^2. At ground level adjacent to the radar tower (for a tower height of 49.2 ft), the average power density is 0.001 mW/cm^2; at 800 ft from the antenna 0.0005 mW/cm^2; and at 1 mile, 0.00005 mW/cm^2.

A searchlight mode will be used infrequently for maintenance and testing purposes only. During searchlight-mode operation, the WSR-88D beam will be directed at a fixed location for up to 5 minutes at a time. Because the beam will be stationary, average power densities will be higher than during normal operation. Averaged over a 6-minute period, the maximum power density will be 3.85 mW/cm^2. Averaged over a 30-minute period, the maximum power density will be 0.77 mW/cm^2. If multiple 5-minute periods of searchlight-mode operation were conducted during a 30-minute interval, the IEEE (1991) guideline for uncontrolled environments could be exceeded. To prevent violation of the guideline, each time the searchlight mode is to be used, safety measures will be followed to make significant impacts very unlikely.

The validity of the methodology used to calculate WSR-88D RFR intensities was tested by comparing calculated RFR levels to RFR levels measured at the OSF in Norman, Oklahoma. The calculated values were somewhat greater than the measured values, which was expected because the calculated values are a worst-case analysis. Thus, the methodology used here to calculate RFR intensities accurately estimates worst-case RFR levels.

V.B.5. Comparison of WSR-57 and WSR-88D RFR

The WSR-57 radars that will be replaced by WSR-88D radars at some locations have a radiated pulse power similar to that of the WSR-88D unit. Because the duty cycle (percentage of time during which the radar transmits a signal) is much less than for the WSR-88D units, the WSR-57 has an average radiated power of 0.33 kW, about one-third that of the WSR-88D. The frequencies of the WSR-88D and WSR-57 signals are identical.

The maximum pulse power density of the WSR-57 signal is a constant 12,100 mW/cm^2 in the near-field (within 150 ft of the radar). In contrast, the WSR-88D unit, with a larger antenna, spreads its power over a larger area, resulting in a lower maximum pulse power in the near field. The maximum near-field pulse power density of WSR-88D units is 2200 mW/cm^2.

Table V.2 compares the average power density of WSR-57 and WSR-88D RFR at various distances within 1 mile of the radar. In the near field, the average power density of the WSR-88D is lower, while that of the WSR-57 is lower in the far field.

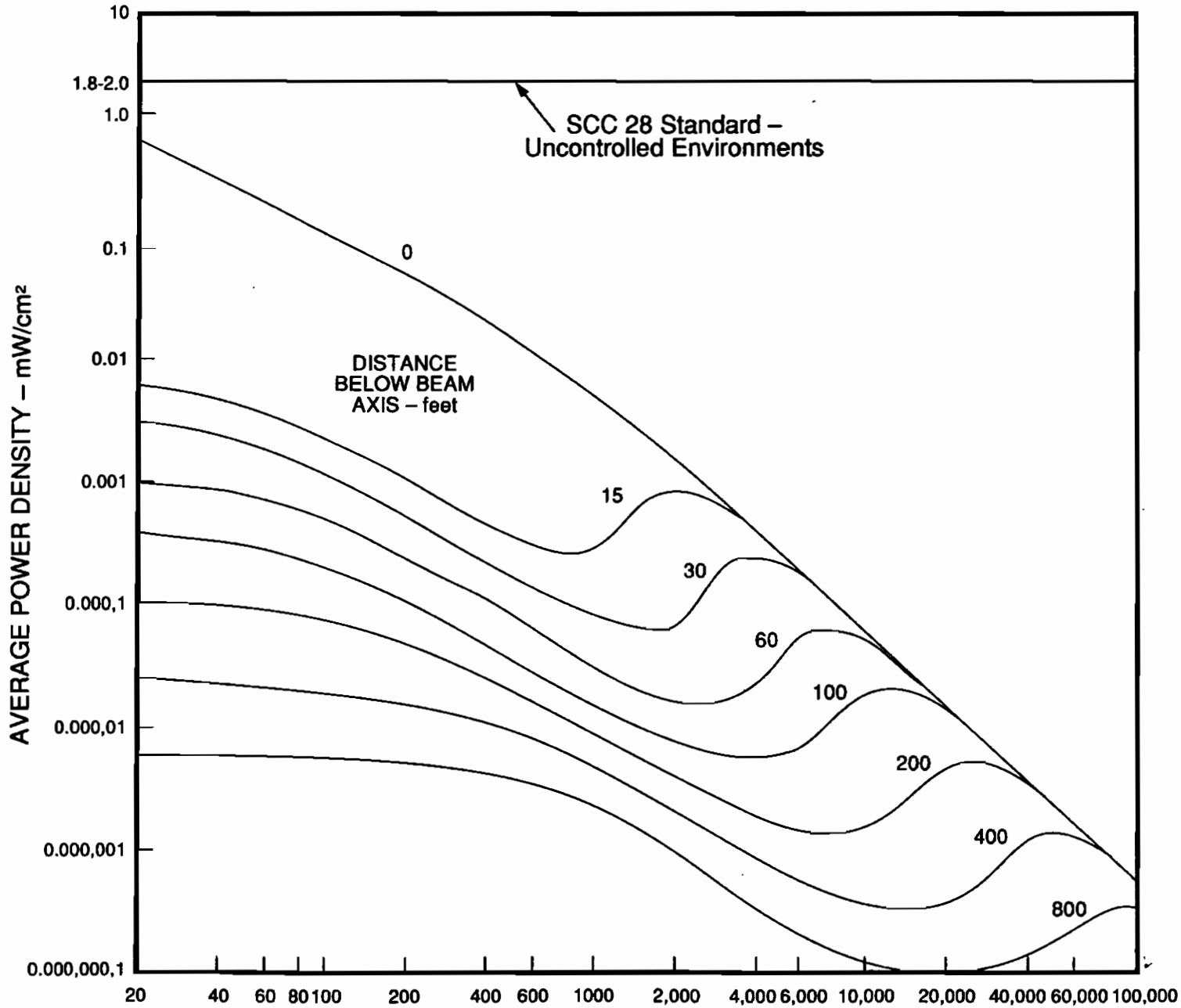

FIGURE V.3 Power Densities Near a WSR-88D Unit Operating in Coverage Pattern 31 (averaged over 6 min.)

Table V.2

COMPARISON OF AVERAGE POWER DENSITY OF WSR-57 and WSR-88D RFR

Distance from Antenna (ft)	Average Power Density (mW/cm^2)	
	WSR-57	WSR-88D
10-20 (surface radome)	1.28	0.60
150	0.085	0.07
800	0.003	0.009
2000	0.0005	0.0015
5280	0.00007	0.0002

Source: SRI International

The WSR-57 can be operated in searchlight-mode operation indefinitely. At 150 ft from the antenna, the average power density during searchlight mode operation is 8.0 mW/cm^2. At 800 ft from the antenna the WSR-57 RFR average power density during searchlight mode decreases to 0.28 mW/cm^2. In contrast, in the searchlight mode, the power density of WSR-88D RFR in the main beam, averaged over 6 minutes, will be 3.85 mW/cm^2 within 800 ft of the antenna.

V.C. HUMAN HEALTH EFFECTS OF RFR

V.C.1. Introduction

This section summarizes our analysis, contained in Appendix B, of the potential bioeffects of RFR and their relationship to human health. Our analysis is based on a review of papers selected from the many thousands of accounts published in scientific journals through about mid-1991. Appendix B also relies in part on *Critique of the Literature on Bioeffects of Radiofrequency Radiation: A Comprehensive Review Pertinent to Air Force Operations* (Heynick, 1987), that SRI International prepared for the U.S. Air Force. That document assessed more than 500 detailed reviews and analyses of research projects. Almost all of the papers selected for analysis had been published in scientific reviews, typically after peer review. Other general reviews of the literature on RFR bioeffects published from time to time were also investigated, among them an EPA report (Elder and Cahill, 1984), that was intended to serve as the primary reference for development of U.S. guidelines for general public exposure to RFR, but that was never promulgated. The conclusions in this SEA regarding possible effects of exposure of people to RFR were reached independently.

This section also compares WSR-88D RFR levels to exposure standards adopted or considered by a variety of entities, including professional organizations.

V.C.2. Summary of Current Knowledge About RFR Bioeffects

Most of the evidence for biological effects of RFR is derived from results of experiments in which various mammals (including human volunteers) and nonmammals (e.g., birds, insects, bacteria, other microorganisms) were exposed to RFR and specific biological effects were sought. Also studied were tissues such as excised organs and neurons, blood, single cells, cultures of cells, and subcellular components kept alive artificially (i.e., *in vitro*). Evidence is also derived from epidemiologic and occupational studies, but such results are indirect or inferential because the RFR-exposure levels and their durations are usually not known with any degree of accuracy.

This summary is organized around a set of RFR-bioeffects topics:

- Epidemiologic/occupational studies
- Congenital anomalies
- Ocular effects
- Auditory effects
- Shock and burn
- Mutagenesis, cytogenetic effects, and carcinogenesis
- Teratogenesis
- Nervous system effects
- Immunological and hematological effects
- Physiological and biochemical effects
- Behavior
- Cellular and subcellular effects.

V.C.2.1 Epidemiologic/Occupational Studies

Taken collectively, the epidemiologic studies reviewed yielded no reliable evidence that chronic exposure to RFR at levels within the U.S. exposure guidelines is hazardous.

Several of the epidemiologic studies found no evidence of detrimental effects associated with exposure to RFR. The studies by Robinette and Silverman (1977) on the deaths of Navy veterans falls in this category. It involved examination of the records of sizable "exposed" and control populations, but the findings are open to question because the members in each group were selected on the basis of military occupation titles. The Lilienfeld et al. (1978) search for possible effects of irradiation of Moscow Embassy personnel and dependents also yielded negative results.

Several other studies were flawed for various reasons, such as by the use of population samples that were too small, use of mailed self-administered questionnaires to acquire the data, inappropriate statistical treatment of the data, or incorrect assembly of data bases. Studies included in this category are those by Hamburger et al. (1983), Lester and Moore (1982a,b), Milham (1983, 1988), and Burr and Hoiberg (1988). The findings of such flawed studies, whether positive or negative, cannot be regarded as strong evidence.

V.C.2.2 Congenital Anomalies

Studies on congenital anomalies or perinatal infant deaths have not yielded scientifically valid evidence that such effects are caused by chronic exposure to RFR at levels below the U.S. exposure guidelines.

Sigler et al. (1965) obtained results suggestive of an association between the occurrence of Down's syndrome in children whose fathers were exposed to radar during military service. However, the study by Cohen et al. (1977) with a larger data base yielded negative findings, thereby superseding the earlier study. Similarly, the negative findings of a study by Burdeshaw and Schaffer (1977) on the incidence of birth defects in children born to mothers that live near military bases superseded the findings of two studies by Peacock et al. (1971, 1973).

The study of Källén et al. (1982) of infants born to physiotherapists, who were assumed to have been occupationally exposed to various agents such as chemicals, drugs, X-rays, and RFR, revealed fewer dead or malformed infants than in the general population. The data base for the cohort part of the study was large, thereby yielding statistically credible negative findings. However, the use of a questionnaire in the case-control part of the study renders questionable the finding of a weak association of malformed or perinatally dead infants with the use of shortwave equipment.

V.C.2.3 Ocular Effects

The cornea and lens of the eye are vulnerable to RFR at high levels because of their surface location and because any heat produced by the RFR is more effectively removed from other regions of the eye. Indeed, there are several documented early cases of inadvertent exposure to RFR at levels high enough to cause cataracts, and safety measures to avoid such exposure have been defined.

Taken collectively, neither animal studies nor epidemiologic studies on eye damage from RFR yield scientific evidence that prolonged exposure to low-level RFR is likely to cause eye damage to humans.

V.C.2.3.1 Animals

With the possible exception of the Kues et al. (1985) study, all animal experiments indicate that ocular damage from RFR exposure is a gross thermal effect. Especially noteworthy are the findings of Guy and coworkers that exposure to RFR at levels that yield a temperature rise within the eye of about 5°C or more are necessary for thermal eye damage and that no damage occurs from such RFR levels if the eye is cooled during exposure. Guy et al. (1975a) reported an average-power-density threshold for eye damage of roughly 150 mW/cm^2 for exposure durations of 100 minutes (or longer). A lower threshold (80 mW/cm^2) was found earlier by Carpenter et al. (1960), but errors in the power-density measurements were subsequently discovered.

Kues et al. (1985) reported increases in the numbers of corneal lesions observed by specular microscopy in the eyes of exposed monkeys. The adequacy of the exposure technique used and the use of the same monkeys in more than one aspect of the study have been questioned, as has the apparent reversibility of the corneal effect, even though the primate corneal endothelium is not known to repair itself through cell division. Resolution of such points awaits further studies or replication in other laboratories.

The results of Stewart-DeHaan et al. (1985) on exposure of lenses to pulsed RFR also are evidence for the thermal basis of RFR eye damage. Noteworthy is a similar study by Creighton et al. (1987) with continuous-wave (CW) as well as pulsed RFR because the pulsed RFR yielded almost 5 times greater depth of lens damage than the CW RFR under corresponding exposure conditions.

V.C.2.3.2 Humans

The findings of Cleary et al. (1965) were negative: of 2,644 veterans classified by military occupation specialties as radar workers, only 19 had cataracts, whereas 21 of the 1,956 nonradar veterans had cataracts. Because the populations of the two groups were adequate in size, the findings were negative, independent of how accurately the veterans were classified. By contrast, the findings of Cleary and Pasternack (1966) were unclear, in part because physiological aging of the lenses had occurred in both the exposed and the control groups and the two groups were not well matched in age distribution. In addition, in grading lens changes, the use of an arbitrary, subjective scale that did not represent reductions in visual acuity is questionable.

Three ophthalmologic studies of Army personnel by Appleton and coworkers on possible ocular damage from exposure to RFR yielded negative findings. However, the authors used a binary (yes or no) scale to score lens damage, and they did not treat their data statistically. The examination by Hollows and Douglas (1984) of the lenses of radiolinemen showed some statistically significant differences in eye changes between their exposed and control groups, but the presence of non-RFR factors could not be ruled out. Nor was evidence given that such changes affected visual acuity.

On rare occasions, accidental exposure to relatively high RFR levels occurs. Hocking et al. (1988) reported on such as exposure of nine radio linemen to RFR. In subsequent ophthalmic examinations, various eye abnormalities were seen in the "low-exposure" as well as the "high-exposure" groups, but vision was not affected in any of the subjects.

V.C.2.4 Auditory Effects

Humans near some types of pulsed radar transmitters have perceived single pulses or pulse trains of RFR as audible clicks (without the use of electronic receptors). Considerable experimental evidence supports the conclusion that an RFR pulse can produce a transient thermal gradient in the head large enough to generate a transient elastic wave at a boundary between regions of dissimilar dielectric properties and that this wave is transmitted by bone conduction to the middle ear, where it is perceived as a click. Persons with impaired hearing are unable to hear such clicks, and animals with nonfunctioning inner ears do not exhibit RFR-pulse-induced evoked responses in the brainstem. Thus, the preponderance of experimental results indicates that perception of RFR pulses as sound results from induction of thermoelastic waves in the head, rather than by direct brain stimulation by the RFR.

Frey and coworkers (e.g., 1961, 1962, 1967, 1968, 1973) were first to study the auditory effect in the U.S., but their hypothesis that the effect was due to direct brain stimulation by the RFR pulses was disproved by later studies. Among the latter were White (1963), who demonstrated that thermoelastic acoustic waves can be generated in various media by RFR. Foster and Finch (1974) confirmed White's findings in water, and proved that such acoustic waves are not generated in water at 4°C, where its thermal expansion coefficient is zero. Olsen

and Hammer (1981) and Olsen and Lin (1981) investigated RFR-pulse transduction in models of the head and obtained results that support the thermoelastic theory.

Taylor and Ashleman (1974) demonstrated that the effect does not occur in cats whose inner ears are nonfunctional. Guy et al. (1975b) confirmed the latter results, as did Chou and Galambos (1979). Guy et al. (1975b) also studied the hearing effect in two volunteers and found that their respective threshold peak energy densities for pulse perception were 40 and 135 microjoules/cm^2 per pulse irrespective of the pulse durations used. Cain and Rissman (1978), using 3.0-GHz pulsed RFR, determined peak-power-density thresholds in volunteers for various pulse durations. Tyazhelov et al. (1979), in studying the qualities of apparent sounds perceived by humans from exposure to 800-MHz pulsed RFR, showed that pulse perception as sound could be modulated by concurrent reception of acoustic tones.

V.C.2.5 RFR Shock and Burn

People can experience electric shock or tissue burns when in the vicinity of transmitters that emit RFR at frequencies below about 100 MHz, and the IEEE (1991) guidelines includes maximum exposure limits for avoidance of such effects. Such effects are not of concern with regard to the WSR-88D because its operating frequencies are much higher than 100 MHz.

V.C.2.6 Mutagenesis, Cytogenetic Effects, and Carcinogenesis

Mutagenesis and carcinogenesis are considered to be related, and many chemicals are screened for potential cancer-causing properties by testing whether they produce mutations in specific bacteria. RFR-induced mutagenic effects have also been sought in various plants and animals. Relatively few studies have been conducted to determine whether RFR *per se* (e.g., 3 GHz) induces or promotes cancer. On the other hand, a growing number of epidemiologic studies have reported a statistical association of cancer promotion with exposure to power line fields (60 Hz). However, most of these studies are subject to methodological and interpretative criticisms (Merritt and Jauchem, 1991). Despite this they cannot be totally dismissed.

Collectively, the scientific literature does not provide scientifically credible evidence that exposure of either mammalian or nonmammalian subjects to low levels of RFR produces mutations or cytogenetic effects, or that such RFR induces or promotes any form of cancer in mammals or cultures of mammalian cells. Thus, there is no experimental evidence from those studies that exposure to the RFR from WSR-88D will cause such effects in the general population.

V.C.2.6.1 Microorganisms and Fruit Flies

Blackman et al. (1976) found no significant differences in genetic activity in *E. coli* cultures exposed to RFR. Dutta et al. (1979) obtained similar results with *Salmonella* cultures. Anderstam et al. (1983) selected 27.12 MHz and 2.45 GHz to determine whether such RFR is mutagenic for *E. coli* or *Salmonella* bacteria and observed both increases and decreases in mutant counts relative to controls; however, most differences were nonsignificant.

A study by Pay et al. (1972) involving exposure of male fruit flies showed no significant differences between exposed and control groups in mean generation times or brood sizes. Hamnerius et al. (1979) exposed fruit fly embryos and found only 4 mutations in 7,512 RFR-

exposed males (0.05%) and 2 mutations in 3,344 control males (0.06%), a nonsignificant difference.

V.C.2.6.2 Mammals and Mammalian Tissues

A study by Skidmore and Baum (1974) seeking biological effects of EMP yielded negative findings: exposure of five pregnant rats in an EMP simulator during gestation produced no gross abnormalities in the fetuses. Almost continuous exposure of 20 female rats to EMP for 38 weeks produced no mammary tumors at age 1 year. Histologic examination of the 42 survivors of the 50 male mice exposed for 33 weeks showed that 9 (21%) were leukemic whereas 11 of the 24 survivors (46%) of the 50 control mice were leukemic. However, the sample sizes were too small to ascribe any validity to that difference in percentages. It is not clear why about half of the control mice had died, a possible indication that uncontrolled non-RFR factors were present. The authors, on mating EMP-exposed males with virgin females, also found no significant differences in the numbers of progeny and no anatomical abnormalities in newborns.

A study by Varma and Traboulay (1976) showed that exposure of male rodents to RFR levels that produce frank heating of the testes tend to reduce fertility, but that such levels were not mutagenic. Experiments on male fertility in rats by Berman et al. (1980) yielded no evidence of an increase of dominant lethal mutations from RFR at power densities up to 28 mW/cm^2.

McRee et al. (1981) found no statistically significant effects of RFR exposure of mice on the induction of sister chromatid exchanges, a sensitive technique for assaying genetic damage from mutagens and carcinogens, or on the rate of proliferation of bone-marrow cells.

A study by Meltz et al. (1990) on exposure of leukemic mouse cell cultures to pulsed RFR, either alone or in combination with the chemical mutagen proflavin, yielded negative findings: The RFR in combination with proflavin produced no statistically significant increase in induced mutant frequency relative to the results for treatment with proflavin alone. In addition, RFR exposure alone yielded no evidence of mutagenic action.

V.C.2.6.3 Cancer Induction and Promotion in Animals

In a study by Prausnitz and Susskind (1962) in which 200 mice were exposed to 9.3-GHz pulsed RFR at 100 mW/cm^2, the authors indicated occurrence of leukemia (a mistaken description) in both the exposed and control mice, but in more of the exposed than the control mice. Roberts and Michaelson (1983), in a subsequent reanalysis of the primary data, concluded that the Prausnitz and Susskind (1962) study provided no evidence that chronic RFR exposure does or does not induce cancer.

As previously mentioned, Skidmore and Baum (1974) found that continuous exposure to EMP did not lead to the development of mammary tumors and that exposure of a strain of mice prone to spontaneous leukemia to electromagnetic pulse (EMP) did not promote leukemia.

Szmigielski et al. (1982) studied whether exposure of mice of a strain known to have high spontaneous incidence of breast tumors would alter the incidence of such tumors. Four groups of mice were purposely confined during RFR exposure to stress them, and one group each was sham exposed (i.e., conditions mimicking RFR exposure without actual RFR), raised under chronic confinement stress without exposure to RFR, and maintained as cage controls. The mean times for tumor development for the groups exposed at 5 mW/cm^2 and for those stressed by

confinement were comparable, and were between the mean times for the groups exposed at 15 mW/cm^2 and the cage controls – a positive finding. However, because confinement stress alone was found to increase tumor incidence, it seems likely that the added heat stress from the higher RFR level was responsible for the increases in tumor incidence and not any carcinogenic properties of RFR *per se*.

Szmigielski et al. (1982) also similarly investigated whether the incidence of skin cancer in mice is increased by RFR exposure. Again, the RFR-induced increases in skin-cancer incidence were probably due to the heat stress at the higher RFR level rather than from any intrinsic properties of the RFR.

In a comprehensive study by the University of Washington on health and longevity, 100 rats were exposed to 2.45 GHz RFR at 0.5 mW/cm^2 for virtually their entire lifetimes (except those withdrawn for interim tests and those that expired before the end of the exposure regimen), and 100 rats were concurrently sham exposed. The findings on immunology and hematology are summarized in Section 2.9. Regarding longevity and cancer, the authors concluded that "no defendable trends in altered longevity, cause of death, or spontaneous aging lesions and neoplasia can be identified in the rats exposed to this long-term low-level radiofrequency exposure" (Guy et al. 1985).

A study by Santini et al. (1988) on the possible development of melanoma or survival times in black mice from RFR exposure yielded no significant differences in either tumor development or survival among the exposed, sham-exposed, and control groups.

Balcer-Kubiczek and Harrison (1991) exposed cultures of mouse-embryo-fibroblast cells to RFR alone or to RFR before or after exposure to X-rays. After such treatments, they incubated the cultures with or without a known tumor promotor (TPA) and assayed them for incidence of neoplastic transformations.

The results for RFR alone showed no evidence of tumor promotion. However, the mean neoplastic transformation incidence was higher for the RFR-exposed cultures incubated with TPA; the authors regarded these results as indicating that RFR acts synergistically in a dose-dependent manner with TPA to promote neoplastic transformation. Little credence can be given to the latter results because of the small numbers of foci per dish and the fact that the counting was apparently not done without prior knowledge of the treatment of each dish.

V.C.2.7 Teratogenesis

Teratogenesis refers to the causation of anatomical aberrations in a developing fetus, but more generally also includes fetal death and/or resorption and postnatal abnormalities in offspring. Such effects occur naturally at low rates in most mammals, and relatively little is known about their causes. In a few cases, however, specific agents have been shown to cause significant effects, and hence the possibility that such effects could occur from exposure to RFR is an appropriate matter of public concern. The term is usually applied to mammalian fetuses and infants, but effects on nonmammalian subjects have also been sought.

Taken collectively, the studies reviewed indicate that teratogenic effects can occur in both nonmammalian and mammalian subjects as a result of RFR exposure, but only at levels that produce significant temperature rises. The results for mammals show that increases in maternal

body temperature that exceed specific thresholds (41.5°C in rats) are necessary to cause teratogenic effects.

V.C.2.8 Nervous System

Concern has been expressed that direct (nonthermal) interactions of RFR with the central nervous system (CNS) could produce deleterious physiologic effects. It has been postulated that such effects may be manifested as alterations in behavior, passage of foreign agents from the blood vessels in the brain into the surrounding tissue by opening of the blood-brain barrier (BBB), changes in the histopathology and histochemistry of the nervous system and of the electroencephalogram (EEG), and changes in the efflux of calcium from brain tissue.

V.C.2.8.1 Blood-Brain Barrier Effects

In most organs and tissues of the body, molecules in the blood can freely diffuse into the tissues around capillaries. However, to protect the brain from invasion by various blood-borne microorganisms and toxic substances, the BBB in most regions of the brain allows little or no movement of large fat-insoluble molecules from the blood into the surrounding brain tissues. The BBB can be "opened" by certain agents (such as ionizing radiation or excessive heat) or by chemical substances (e.g., dimethyl sulfoxide). Studies have been conducted to determine if RFR can alter the permeability of the BBB in animals to substances of large molecular weight. The preponderance of negative scientific experimental findings indicates that exposure to pulsed or CW RFR at low levels would not alter the human BBB.

Early studies by Frey et al. (1975) and Oscar and Hawkins (1977) can be disregarded because of evidence of artifacts in the methods used in the experiments. Moreover, Merritt et al. (1978) and Ward and Ali (1985) were unable to reproduce those findings, and Preston et al. (1979) showed that certain specific RFR-induced changes in the brain could be interpreted wrongly as BBB alterations. Preston and Préfontaine (1980) and Gruenau et al. (1982) obtained negative findings regarding RFR-induced alterations of the BBB. Four comprehensive studies by Williams et al. (1984a, b, c, d) in which several different tracers and methods were used for detecting BBB penetration also yielded negative findings. Neilly and Lin (1986) showed that disruption of the rat BBB at high RFR levels is due to elevation of brain temperature. In addition, they found that high ethanol doses inhibit BBB disruption by moderating the increases in brain temperature produced by the RFR.

Many findings about the effects of RFR on the blood-brain barrier must be discounted because of the presence of significant artifacts due to the biological method used or to interpretation of RFR-induced changes in the relative sizes of vascular and extravascular volumes in the brain as alterations of the BBB. Results of recent studies, in which artifacts were substantially reduced and perhaps rendered negligible, indicate that RFR can affect the BBB only at hyperthermic levels.

V.C.2.8.2 Histopathology and Histochemistry

Although histochemical changes in the CNS have been seen at relatively low SARs*, their significance with regard to possible human health hazards is not clear, and questions about those studies remain open. However, overall, considering the other experimental results in which the effects were ascribable to increases in brain temperature, it is unlikely that the RFR at WSR-88D levels would cause histopathological or histochemical changes in the human central nervous system.

Albert et al. (1981) had reported lower mean counts of Purkinje cells in 40-day pups RFR-exposed for 5 days *in utero* to 2.45-GHz RFR at 10 mW/cm^2 relative to counts for sham-exposed pups. These results are open to question because of the large variations in SAR (0.8 to 6 W/kg, 2 W/kg estimated mean) due to movement of the dams during exposure. In addition, rat pups exposed to the RFR and euthanized 40 days later did not show such differences, and little credence can be given to their results for rat pups euthanized immediately after exposure because of Purkinje cell immaturity in neonates – a point previously mentioned by the authors. Moreover, in a similar study they conducted using squirrel monkeys that had been previously exposed perinatally elsewhere, they found no significant differences between RFR-exposed and sham-exposed monkeys in Purkinje cell counts.

Merritt and Frazer (1975) obtained negative findings in mean brain concentrations of various neurotransmitters and their metabolites assayed in mice exposed to predominantly electric or magnetic fields at 19 MHz. However, the authors noted that at 19 MHz, a mouse absorbs very little energy from either field.

Sanders and coworkers (1985) observed increases in NADH fluorescence and reductions of ATP and CP concentrations in the rat brain by RFR at levels characterized as not producing measurable brain hyperthermia. Although some points are open to question, those positive findings appear to be valid and are worthy of further study. The experiments were performed on anesthetized rats, with consequent lowering of brain temperatures. Whether similar results would occur in the absence of anesthesia has not been determined. It is noteworthy that the effects were higher at 591 MHz than at 200 MHz and that they were not observed at 2.45 GHz; these findings are suggestive of dependence on RFR frequency.

A study by Lai et al. (1988) with 2.45-GHz CW and pulsed RFR at whole-body SARs of about 0.6 W/kg on choline uptake in various regions of the rat brain yielded both positive and negative results. However, results for the two kinds of exposure chamber and between pulsed and CW RFR were apparently inconsistent.

In summary, histochemical changes in the CNS were seen at relatively low SARs by Sanders and coworkers (1985) and Lai et al. (1988), but their significance with regard to possible human health hazards is not clear and needs further investigation. Taken collectively with other experimental results in which the effects observed were ascribed to local increases in brain temperature, it seems unlikely that exposure to RFR levels that do not increase local brain temperatures would cause deleterious histopathologic or histochemical effects in the human central nervous system.

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* Specific absorption rate. In the RFR-bioeffects literature, the energy absorbed by a body from an incident electromagnetic field is usually quantified by the SAR. See Section V.C.4.1.

V.C.2.8.3 Electroencephalogram (EEG) and Evoked-Response Changes

Various studies have been undertaken to ascertain the effects of RFR on the EEG or on the responses evoked by visual or auditory stimuli. Experiments in which specially devised electrodes were used or in which electrodes were applied after exposure, yielded no evidence of significant differences in EEGs or in evoked responses between control and RFR-exposed animals. Thus, there is no evidence that the EEG or evoked responses of people will be affected by low-level RFR.

As demonstrated by Johnson and Guy (1972), the use of indwelling metallic electrodes, wires, or screws in the apparatus for measuring and recording EEGs is likely to induce artifactual effects in the animals under study as well as in the recordings themselves. The findings of early studies can be discounted because of such use. On the other hand, EEG measurements taken after completion of RFR exposure may be less definitive because of interpretation problems stemming from the time consumed in attaching the electrodes and the variability in their placement. Moreover, any transient effects that may occur during RFR exposure would disappear when exposure ceases. These points are applicable to the negative findings of Kaplan et al. (1982).

In several studies, such as those by Tyazhelov et al. (1977) and Chou and Guy (1979a), effort was made to minimize artifact occurrence by design of electrodes and leads from materials having high resistivities comparable to those for tissue. When such electrodes were implanted before exposure and were present during exposure, no significant differences in EEGs or evoked responses between control and RFR-exposed animals were obtained, as exemplified by the study of Chou et al. (1982).

Until relatively recently, studies directed toward the possible effects of RFR on EEGs and evoked responses suffered from artifacts introduced by the use of metallic electrodes during exposure that perturbed or were perturbed by the RFR. That problem was greatly ameliorated by the development of nonperturbing tissue-compatible electrodes, permitting measurements of EEGs and evoked responses during RFR exposure of unanesthetized animals. When such electrodes were used, the differences between responses of RFR- and sham-exposed animals were nonsignificant.

V.C.2.8.4 Calcium Efflux

Various studies have reported that exposure of samples of brain tissue from newly hatched chickens to 147-MHz or 450-MHz RFR at levels in the range 1-2 mW/cm^2 that were amplitude-modulated at low frequencies increases the rate of exchange of calcium ions between the tissue and the fluid bathing it (the "calcium-efflux effect"). The maximum effect was seen for modulation at 16 Hz, and no effect was seen for unmodulated 147-MHz or 450-MHz RFR.

Other researchers have been unable to confirm the existence of the phenomenon. Several of the recent studies that report positive findings suggest that magnetic fields, primarily at power line frequencies [as well as the earth's direct current (dc) field], contribute significantly to the effect. However, there is no experimental evidence that the effect, if it does exist, could be harmful to humans or intact animals.

V.C.2.9 Immunology and Hematology

Many reports indicate that RFR has specific effects on the immune systems of mammals. Most reported effects were detected after exposure at power densities of about 10 mW/cm^2 and higher; a few effects have been found from exposure to levels as low as about 0.5 mW/cm^2. In most of the studies, the mechanisms for the effects were not investigated, and many of the results were not consistent with one another.

Much early work investigating possible effects of RFR on white blood cells exposed *in vitro* suffered from inadequate control of cell temperature during exposure. In later studies in which effective control over culture temperature was exercised, nonsignificant differences were obtained with exposed cultures held at the same temperature as control cultures. In studies where the culture temperature was elevated by RFR, the effects were clearly of thermal origin.

In early studies of RFR exposure of red blood cells *in vitro*, hemoglobin and potassium-ion losses were found for rabbit red blood cells exposed at average power densities as low as 1 mW/cm^2. In subsequent studies, the hemoglobin and potassium-ion losses resulting from heating with RFR did not differ significantly from such losses obtained with conventional heating.

Studies seeking immunological effects of exposing animals to RFR *in vivo* yielded mixed results. Some investigators found that RFR exposure of mammals increased the proliferation of white blood cells or the production of antibodies (relative to controls), but with few exceptions the measured or estimated SARs were well in excess of 1 W/kg. More subtle effects on mammalian immune systems were sought in more recent studies, making use of significant advances in assay methods and with attention to possible effects of non-RFR stress. Some of those investigations were directed toward the effects of RFR on the activity of natural killer (NK) cells; their results again showed that SARs much higher than 1 W/kg were necessary for effect.

More directly relevant to possible RFR effects on the human immune system would be studies in which animals are chronically exposed to RFR (preferably over virtually their entire lifetimes) to determine whether such exposure adversely affects their health, longevity, and resistance to natural disease or experimental challenge with various microorganisms or toxins therefrom. Some studies indicated that animals exposed to RFR for relatively short periods can withstand bacterial infection better than sham-exposed animals. However, few studies involving chronic RFR exposure have been carried out or repeated by other laboratories.

Probably the most comprehensive chronic RFR exposure investigation to date was a University of Washington study in which the authors exposed 100 rats to 2.45-GHz RFR at 0.5 mW/cm^2 and concurrently sham-exposed 100 other rats for essentially their full lifetimes (except those withdrawn for interim tests and those that expired before the end of the exposure regimen). Tests of 10 each RFR- and sham-exposed rats withdrawn after 13 months of treatment showed counts of splenic T- and B-lymphocytes that were significantly higher for the RFR than the sham group, an effect ascribed to stimulation of the lymphoid system by the RFR. However, this effect was absent in similar tests on completion of treatment, with the absence ascribed to immunosenescence. Longevity of the rats was not affected by the RFR at corresponding times during the exposure regimen.

No primary malignancies were found at 13 months (in rats younger than 1 year). Probably the most controversial finding of the entire study, however, was that among rats older than 1

year, primary malignant lesions (of various kinds) were found in a total of 18 of the RFR-exposed rats but in only 5 of the sham-exposed rats. The authors gave several cogent arguments to discount the biological significance of this finding. Little credence can be given to this point because of the nonsignificant differences for each malignancy type and because combining those nonsignificant differences to attain statistical significance is an oncologically dubious procedure. Also, the results of the study showed no significant differences between RFR and sham groups in the incidence of benign tumors, a point of importance if the initiation process is assumed to be similar for both benign and malignant tumors. The authors stated "the findings here of excessive malignancies in the exposed animals is provocative; however, when this single finding is considered in light of other parameters evaluated, it is questionable if the statistical difference reflects a true biological activity".

V.C.2.10 Physiology and Biochemistry

V.C.2.10.1 Metabolism and Thermoregulation

The literature on physiological and biochemical effects associated with exposure to RFR is extensive. The thermal basis for various effects of RFR on the autonomic thermoregulatory systems of mammals and on their behavioral thermoregulatory responses to RFR is evident. Especially noteworthy are the results for primates because of their far greater similarities to humans than the other animals studied.

V.C.2.10.2 Endocrinology

Exposure of mammals to RFR has yielded rather inconsistent effects on the endocrine system. In general, effects are apparently related either to the heat load associated with RFR or to the stress induced by RFR, and possibly to other circumstances. Most of the studies of possible effects of RFR on endocrine systems were conducted on rodents. Studies that reported positive findings also yielded indications that the effects were largely due to increases in the thermal burdens of the animals. In many studies, observed alterations in endocrine function may have been significantly influenced by stresses in the animals. For this reason, the results of studies that reduced stress by acclimating animals to handling and the experimental situation are notable.

Although some effects of RFR exposure on the endocrine system appear to be straightforward and predictable from physiological considerations, other, more subtle effects may be worthy of additional study (e.g., those related to the interactions among the pituitary, adrenal, thyroid, and hypothalamus glands, and/or their secretions). Part of the problem in interpreting such results appears to be related to the uncertainties about stress mechanisms and various accommodations to such mechanisms. Animals placed in novel situations are much more prone to exhibit stress responses than those adapted to experimental situations.

Because the effects of RFR on the endocrine systems of animals are largely ascribable to increased thermal burdens, to stresses engendered by the experimental situation, or to both, there is no clear evidence that such effects would occur in humans exposed to RFR at levels which do not produce significant increases in body temperature.

V.C.2.10.3 Cardiovascular Effects

Few investigations have been carried out on possible effects of RFR on the human heart. However, various studies have been performed *in vitro* on hearts (or parts thereof) excised from animals, and others have been conducted on animal hearts *in vivo*.

The positive findings reported in early studies (bradycardia, tachycardia, or both) were suspect because of the use of attached or indwelling electrodes that probably introduced artifacts. Various kinds of electrodes were investigated, and special types were developed that were not perturbed by RFR or did not perturb the local RFR fields. Studies involving the use of such electrodes showed that heart rates were altered only at RFR levels that produced rises in temperature or otherwise added thermal burdens to the animal.

Also investigated was the possibility that pulsed RFR at repetition rates synchronous with various periodic characteristics of the EKG could alter heart rate. The authors of an early study reported induction of tachycardia in isolated frog hearts by RFR pulses in synchrony with the EKG, but other researchers could not confirm this finding in either isolated hearts or live animals.

Several researchers showed that CW RFR levels well in excess of 1 mW/cm^2 were necessary for significant alterations of heart rate. Slight bradycardia was seen in equilibrated conscious rats exposed to RFR at those levels, a finding ascribed to a compensating reduction in metabolic rate. The results of another study indicated that functioning of hearts damaged from other causes is not affected by exposure to CW RFR at 10 mW/cm^2 or lower.

V.C.2.11 Behavior

V.C.2.11.1 Behavior, Reflex Activity, Learning, and Task Performance

Numerous studies have been conducted of the possible effects of RFR exposure on various kinds of animal behavior. Many of the studies on avoidance behavior by animals indicate that RFR could be a noxious or unpleasant stimulus. There is much evidence, however, that changes in behavioral patterns induced by RFR are responses by animals' thermoregulatory systems, either to minimize absorption of heat in normal or warm ambient environments (including high levels of humidity) or to obtain warmth in relatively cold environments. Thus, other than possible auditory perception of RFR pulses, animals do not appear to sense RFR directly.

The results of studies on disruption of performance or learned behavior by RFR were variable; however, most of the findings showed that the behavioral changes were ascribable to the added thermal burden imposed by the RFR.

The behavioral findings of primate studies are more relevant than those with the other animal species with regard to possible effects of RFR on human behavior because the tasks the primates had to learn were far more complex and because their physiologies and intelligence are much closer to those of humans. In addition, reasonably accurate thresholds for RFR-induced behavioral changes were determined for each primate species studied, and those thresholds served as a basis for the IEEE (1991) human-exposure guidelines.

V.C.2.11.2 Drugs

Studies on the possible synergy between RFR and psychoactive drugs such as diazepam, chlorpromazine, chlordiazepoxide, and dextroamphetamine yielded unclear or inconsistent results. In some studies, the changes in drug dose-response relationship were subtle and not necessarily induced by the RFR. In most of the studies that yielded RFR-induced changes in drug response, average power densities of 1 mW/cm^2 or higher coupled with relatively high drug dosages were necessary. The results were negative (no effects) in still other studies. Noteworthy were the negative findings of synergistic effects between alcohol consumption and exposure to RFR, except at very high doses of alcohol.

In general, it seems unlikely that the effects of psychoactive drugs prescribed by physicians or the effects of recreationally consumed alcohol would be altered by exposure to environmental levels of RFR.

V.C.2.12 Cellular and Subcellular Effects

Various studies of cellular and subcellular effects of RFR have been discussed above under other specific topics such as the BBB, immunology, and hematology. This subsection describes findings related to other RFR effects on cells and their constituents.

Many of the early studies on microorganisms produced results that were taken as evidence of nonthermal effects of RFR. The existence of resonances at frequencies above 30 GHz was postulated on theoretical grounds, and several studies by Webb and coworkers were conducted that appeared to confirm that hypothesis. However, later studies by Cooper and Amer (1983) and by Gandhi et al. (1988) that employed more sophisticated engineering and biological techniques and in which artifacts were reduced significantly, yielded results that did not confirm earlier findings of resonances or other evidence of nonthermal effects at such frequencies.

The apparent absorption resonances in the range of 2 to 9 GHz reported by Swicord and Davis (1983) for aqueous solutions of DNA molecules derived from *E. coli* were regarded as indicative of direct action of RFR with such molecules. Later attempts by Edwards et al. (1985) and Gabriel et al. (1987) to reproduce such findings, however, yielded negative results. Moreover, analytical and experimental results were obtained indicating that such resonances were most likely artifactual, associated with the probes and measurement methodology used.

In general, research on possible RFR effects on microorganisms or of RFR exposure *in vitro* of cells derived from macroorganisms is important for eliciting possible mechanisms of direct interaction of RFR with such biological entities or their constituents at levels that can be characterized as nonthermal. However, the relevance of such findings to possible effects of exposure of intact animals to RFR and ultimately the significance of such findings with regard to possible hazards of RFR to humans would have to be established. Such research to date has not provided any scientific evidence that humans chronically exposed to low-level RFR would experience cellular effects.

V.C.3. Findings

V.C.3.1 Epidemiologic/Occupational Studies

Given the problems and uncertainties in animal studies related to interspecies differences, considerable weight should be given to findings of well-performed studies involving actual or presumed human exposure to RFR, despite the previously stated limitations of such studies.

Relatively few epidemiologic studies have been done on the effects of RFR exposure, either occupationally or from residing in the vicinity of RFR emitters or experimental studies with human volunteers. Taken collectively, those studies yielded no scientific evidence that chronic exposure to RFR at levels within the IEEE (1991) guidelines would cause detrimental health effects.

V.C.3.2 Congenital Anomalies

The possible association of congenital abnormalities with prenatal exposure to RFR has been reported. Careful analyses of such reports do not substantiate such findings.

V.C.3.3 Ocular Effects

The results of animal experiments indicate that heating the eye to temperatures of about 41°C or more damages the lens. Another finding is that for continuous exposure for durations of 2 hours or more, the threshold power density for ocular damage is within the range 100-150 mW/cm^2. In the relatively few epidemiologic studies on possible ocular damage from chronic RFR exposure, some of the findings were negative and others were positive. Most of the positive findings were found to be explained more by aging of the lens than by RFR exposure. Exceptions were cases of possible occupational exposure at levels and for durations sufficient to heat the eye to damaging temperatures. Such cases occurred primarily during the first decade after the end of World War II, as exemplified in the paper by Hirsch and Parker (1952).

V.C.3.4 Auditory Effects

The RFR-auditory effect is not a concern with the WSR-88D, even for persons who may be exposed to its main beam. In a study with human volunteers by Cain and Rissman (1978), the perception threshold for 5-μs pulses (comparable to those from the WSR-88D) was found to be about 2500 mW/cm^2. The peak power density in the main beam of the WSR-88D will not exceed 2200 mW/cm^2. Thus, persons exposed to the main beam are unlikely to "hear" individual WSR-88D pulses.

V.C.3.5 Mutagenesis, Cytogenetic Effects, and Carcinogenesis

In many studies of possible mutagenic effects of RFR on bacteria, yeasts, or fruit flies, the findings indicated that mutations do not occur except under conditions in which the RFR produces significant temperature rises in the specimens. Regarding mutagenesis in mammals, several studies showed that exposure of male rodents to levels of RFR that produce frank heating of the testes tends to reduce fertility, but that such levels were not mutagenic. No scientifically valid evidence was found that chronic exposure to RFR at levels within the ANSI (1982) or IEEE (1991) guidelines induces or promotes any form of cancer in mammals.

V.C.3.6 Teratogenesis

RFR-induced teratogenesis was sought in various species of insects, birds, rodents, and nonhuman primates. In early studies with insects, the RFR levels used were usually high enough to significantly heat the subjects. Nevertheless, those investigators concluded that the abnormalities they found were not due entirely to the heat produced by the RFR. Subsequent investigators, however, were unable to confirm such nonthermal findings and suggested that the earlier findings could be ascribed to uncontrolled non-RFR factors. Thus, there is no valid scientific evidence establishing the occurrence of teratogenic effects in insects at nonthermogenic RFR levels.

A similar conclusion applies to studies of RFR-induced teratogenesis in quail eggs and developmental abnormalities in hatched quail. On the other hand, retardation of development in embryos of the domestic chicken was ascribed by others to exposure of eggs to RFR at relatively low RFR levels (about 3.5 mW/cm^2 average). Close examination of the research indicates that the reported nonthermal RFR teratogenesis in the chicken is questionable.

Mixed results were obtained in studies of RFR-induced teratogenesis and developmental abnormalities in rodents. With mice, both positive and negative findings were reported, but several of the more recent studies reported statistically significant retardation in postnatal growth from RFR exposure *in utero* at levels exceeding 10 mW/cm^2 (an effect found with hamsters as well). On the other hand, virtually all of the studies with rats yielded negative results and indications that the RFR levels that can cause significant prenatal abnormalities or retardation of postnatal growth or development are close to, or above, the lethal level for rat dams.

These findings for rats and mice led one group of researchers to conclude that the mouse may be more suitable than the rat as a model for investigations of possible teratogenic effects of RFR in humans. However, the positive findings in one rodent species and the negative findings in another rodent species suggest that neither rodent species is a suitable model for that purpose. Instead, investigations with nonhuman primates should yield much more definitive findings.

In a study primarily seeking effects of RFR exposure on mother-infant behavior in squirrel monkeys, a small number of unexpected infant deaths occurred in the RFR-exposure groups. Because fewer deaths occurred in the control groups, the results showed borderline statistical significance. However, in a follow-up study specifically directed toward possible RFR-induced infant mortality, which entailed a larger number of monkeys, no statistically significant difference in numbers of infant deaths between RFR-exposed and control groups was seen.

Overall, then, the studies performed on possible RFR-induced teratogenesis and developmental abnormalities support the conclusion that such effects can occur from temperature increases caused by the RFR, rather than from any special teratogenic properties of RFR. However, those findings also indicate that the likelihood is negligible that such effects would occur in humans from exposure to low-level RFR.

V.C.3.7 Nervous System

Many early BBB studies appear to have suffered from the presence of significant artifacts in the biological methodology used. In other studies, the results interpreted by the investigators as RFR-induced alterations of the BBB were more likely ascribable instead to changes in the relative sizes of vascular and extravascular volumes in the brain. In recent studies, however, in

which artifact was reduced substantially and perhaps rendered negligible, the results indicate that hyperthermic levels of RFR are necessary to alter the BBB.

Most of the positive findings of histopathological and histochemical changes in the nervous system by RFR were probably induced thermally. A notable exception was a recent study, which indicated that inhibition of the biochemical respiratory chain function within certain cells can be induced at RFR levels that do not cause measurable tissue hyperthermia. That effect is worthy of further study, but does not indicate a potential health hazard.

Problems associated with the use of metallic electrodes to record EEGs and evoked responses during RFR exposure led to discounting of the positive findings in studies involving such use. Such problems were essentially eliminated by development of high-resistance, carbon-loaded-Teflon electrodes that were tissue-compatible and thus implantable for chronic studies. When such electrodes were used to measure EEGs and/or evoked responses of conscious (unanesthetized) animals during exposure, the differences between responses of RFR-exposed and sham-exposed animals were nonsignificant. For rabbits, used frequently for such studies, the EEGs and evoked responses were found to vary widely among unanesthetized control animals, as well as with time in individual rabbits, thereby reducing the confidence in any positive or negative findings with rabbits.

V.C.3.8 Immunology and Hematology

Possible RFR effects on the immune system were sought in a variety of investigations. In many early studies, suspensions of the various classes of white blood cells were exposed to RFR *in vitro*, but such studies suffered from the lack of adequate control of cell temperature during exposure. In later studies, therefore, considerable effort was devoted to developing exposure systems that permitted maintenance of constant cell temperature at an optimum level during exposure or that provided means for deliberate temperature increases to specified values for purposes of comparison. Many studies with such systems were directed toward determining the effects of RFR on lymphocyte proliferation or on functional characteristics of lymphocytes as components of the immune system.

In studies of white blood cell cultures held at the same temperature during RFR exposure as control cultures, negative findings (nonsignificant differences between exposed and control cultures) were obtained. In those that yielded positive findings, the effects on the exposed cultures were clearly thermal.

Also sought in early studies were possible effects of exposure of red blood cells to RFR *in vitro*. Among the findings were significant cell breakdown (hemolysis) and potassium ion efflux for rabbit red blood cells exposed to 2.45-GHz RFR at average power densities as low as 1 mW/cm^2. In later studies, however, losses by heating with RFR from room temperature to 37°C did not significantly differ from losses due to conventional heating; the threshold SAR for effect was found to exceed 46 W/kg. Significant losses were not found for human cells heated by either means to 37°C, thus indicating that RFR may not induce similar changes in rabbit and human blood.

Exposure of animals to RFR *in vivo* for determining possible effects on the immune system yielded mixed results. Some of the studies showed an apparent diminution of immune responses to RFR, but with no clear dependence on RFR level. Results of other studies appeared to indicate

that survival was extended by exposure to RFR. In investigations with Japanese quail, RFR-related differences in antigenic responses were not found, except when temperature elevations were implicated. Some of the investigators reported that exposure of mammals to RFR increased the proliferation of leukocytes or the production of antibodies (relative to controls); however, with few exceptions, the measured or estimated thermal absorption was well in excess of 1 W/kg. In more recent studies that employed advances in assay methods, subtle effects on mammalian immune systems were sought, including the effects of RFR on the activity of natural killer (NK) cells, with attention to the possible effects of non-RFR stress in the animals. The results indicated that thermal absorption well in excess of 1 W/kg was necessary for such effects to occur.

More directly relevant to possible effects of RFR exposure on the human immune system would be studies in which animals are exposed to RFR continuously for long periods to determine whether such exposure would adversely affect their health, longevity, and resistance to natural disease or experimental challenge with various microorganisms or toxins therefrom. The results of many studies indicate the existence of threshold RFR levels for various bioeffects, thus providing confidence that exposure to levels that are appreciably below the threshold levels is unlikely to be harmful. However, most experimental data were obtained by use of single or repetitive exposures of relatively short durations. Although it is hard to conceive of mechanisms whereby exposure to RFR below threshold levels would accumulate to cause adverse effects, very few studies have been performed in which animal subjects were continuously exposed to low-level RFR (i.e. that does not cause significant heating of tissue) during most of their lifetimes.

The most comprehensive chronic study to date was a University of Washington rat study. In that study, 100 rats were exposed to 2.45-GHz RFR, and 100 rats were concurrently sham-exposed for virtually their full lifetimes (except those withdrawn for interim tests and those that died before the end of the exposure regimen). Tests of 10 rats withdrawn from each group after 13 months of exposure yielded significantly higher splenic T- and B-lymphocyte counts in the RFR subgroup than in the sham subgroup; the authors ascribed those higher counts to stimulation of the lymphoid system by the RFR. This effect, however, was not seen in similar tests conducted on completion of the exposure regimen, and the authors provisionally ascribed its absence to immunosenescence. The RFR did not affect longevity at corresponding times during the regimen.

No primary malignancies were found at the interim tests (in rats younger than 1 year). Primary malignant lesions were found in 2 RFR-exposed and 2 sham-exposed rats at ages 13-18 months, in 9 of the RFR group and 1 of the sham group at ages 19-24 months, and in 7 of the RFR group and 2 of the sham group at ages 26-30 months. The differences in the numbers for each specific malignancy type were all statistically nonsignificant, and the incidence of each specific malignancy in the RFR group was similar to that in the literature for untreated rats of the same strain. The authors stated "the finding here of excessive malignancies in the exposed animals is provocative; however, when this single finding is considered in light of other parameters evaluated, it is questionable if the statistical difference reflects a true biological activity."

In the RFR group 18 rats had malignancies, with 5 rats in the sham group – a difference noted by the authors to be statistically significant. Little credence can be given to this point, however, because of the nonsignificant differences for each malignancy type and because

combining those nonsignificant differences to attain statistical significance is an oncologically dubious procedure. Also, the results of the study showed no significant differences between RFR and sham groups in the incidence of benign tumors, a point of importance if the initiation process is assumed to be similar for both benign and malignant tumors.

V.C.3.9 Physiology and Biochemistry

In investigations of other possible physiological and biochemical effects of RFR, the thermoregulatory systems of nonhuman primates have been shown to readily compensate for high levels of RFR. This finding is most significant relative to possible hazards of human exposure because of the closer anatomical and physiological similarities among human and nonhuman primates than between those of humans and any other mammals.

Most of the studies of possible RFR-induced effects on the endocrine system were conducted on rodents. Those that yielded positive findings indicated that the effects were largely due to increases in the thermal burdens of the animals. In many of the studies, the changes observed in endocrine function may have been significantly influenced by stresses in the animals. For this reason, the studies in one laboratory are notable for the efforts taken toward reducing stress by acclimating the animals to handling and to the experimental situation. Nevertheless, some of the subtle effects of RFR on the endocrine system deserve further study.

In some early studies in which excised hearts were exposed to RFR and other studies in which the whole animal was exposed *in vivo*, the positive findings reported (bradycardia, tachycardia, or both) were suspect because of the use of attached or indwelling electrodes that probably introduced significant artifact. Subsequent studies that used electrodes that were not perturbed by RFR or did not perturb RFR yielded results indicating that heart rates were altered only at RFR levels that caused significant body-temperature rises or otherwise added to the thermal burden of the animal.

Also investigated, both in excised animal hearts and *in vivo*, was the possibility that pulsed RFR at repetition rates that are synchronous with various periodic characteristics of the EKG could alter heart rate. In one study, tachycardia was reported to have been induced in excised frog hearts by RFR pulses in synchrony with the EKG, but this finding could not be confirmed by other researchers.

Other investigators showed that for CW RFR, levels well in excess of 1 mW/cm^2 were necessary for significant alterations of heart rate. The results of another study indicated that the functioning of hearts already damaged from other causes is not affected by exposure to CW RFR at levels of 10 mW/cm^2 or lower.

V.C.3.10 Behavior

Many studies of avoidance behavior in animals appeared to indicate that RFR is a noxious or unpleasant stimulus. There is considerable evidence, however, that the RFR-induced changes in behavioral patterns observed in animals are the responses by their thermoregulatory systems to minimize absorption of heat in normal or warm ambient environments (including high levels of humidity) or to obtain warmth in relatively cold environments. Thus, other than auditory perception of RFR pulses, animals apparently do not directly sense RFR (other than as heat). The results of studies on RFR disruption of animal performance or learned behavior were quite

variable; however, most of the findings showed that the observed changes in behavior could be ascribed to the additional thermal burden imposed by the RFR, and specifically were significant at measured or estimated whole-body thermal absorption well in excess of 1 W/kg.

The many studies of possible synergy between RFR and various psychoactive drugs (diazepam, chlorpromazine, chlordiazepoxide, and dextroamphetamine) yielded unclear or inconsistent results. In some studies, the changes in drug dose-response relationship were subtle and not necessarily induced by the RFR. In most studies that yielded RFR-induced changes in drug response, average power densities of 1 mW/cm^2 or higher coupled with relatively high drug dosages were necessary. The results of still other studies showed no RFR-induced response changes. The absence of synergistic effects between the consumption of alcohol and RFR except at very high doses of alcohol were especially noteworthy. In general, it is most unlikely that the effects of psychoactive drugs or of alcohol would be altered by exposure to low-level RFR.

V.C.3.11 Cellular and Subcellular Effects

Excitation of resonances in cells exposed to submillimeter-wave RFR was postulated on theoretical grounds, and several studies were performed that apparently confirmed that hypothesis. Specifically, preparations of *E. coli* and of protein, RNA, and DNA isolated from such cells yielded apparent resonances in the range 65-75 GHz – results regarded as evidence of nonthermal RFR effects. However, results of later studies, in which more sophisticated engineering and biological techniques were used to reduce artifact, did not confirm the existence of such resonances or any other indications of nonthermal effects.

Apparent resonances in the range of 2 to 9 GHz were reported recently for aqueous solutions of DNA from *E. coli*, but in studies by other groups, true resonances were not found. Instead, use of better analytical and experimental methods produced results indicating that the apparent resonances were most likely artifactual and were associated with the methodology used in the earlier studies.

V.C.3.12 Overall Conclusion Based on the Scientific Review

On the basis of a rigorous analysis of the scientific literature, the RFR from WSR-88D units will be at levels that will not generate adverse health effects in the general population. There is no potential for significant effects to result from exposure to RFR from WSR-88D units.

V.C.4. RFR Safety Standards

V.C.4.1 Introduction and Findings

Terms such as "safety standards" and "exposure standards" generally refer to, and are frequently used interchangeably with, specifications or guidelines on maximum public or occupational exposure levels to electromagnetic fields. Such levels are usually expressed as permissible exposure limits (PELs), threshold limit values (TLVs), or maximum power densities or field intensities in specific frequency ranges for stated exposure durations. Exposure guidelines have been developed by private organizations such as the American National Standards Institute (ANSI), the National Council on Radiological Protection (NCRP) [now called the National Council on Radiation Protection and Measurements (NCRP)], and the American Conference of Governmental Industrial Hygienists (ACGIH) as voluntary guidelines for

occupational or general-public exposure or both. In some instances, however, governmental agencies such as the Federal Communications Commission and various state and municipal bodies have adopted such guidelines or more stringent versions thereof as enforceable standards.

Guidelines for human exposure to RFR have been selected on the basis of maximum values of SAR that were found to be not harmful in experimental animal studies. SAR is the rate at which RFR energy is absorbed in any small region of a body, expressed in watts per kilogram (W/kg). For any value of incident power density, the SAR usually varies with location of the region within the body. Because internal variations of SAR are difficult to determine for complex bodies, the term "whole-body SAR" is often used to represent the spatially averaged value of SAR for the body – a quantity that can be measured without the need for determining internal variations of local SAR. "Local SAR" denotes the rate of energy absorption at any local site within a biological object. The absorption does not necessarily occur as heat.

The following section describes relevant safety standards and compares the electromagnetic fields created by WSR-88D units with the standards. During normal operation, WSR-88D RFR will not exceed the standards or the guidelines adopted by ANSI, IEEE SCC 28, NCRP, or IRPA; nor will it exceed the lowest of the proposed, but not implemented, EPA limits. If limitations on the frequency of use are applied to searchlight-mode operation, the standards mentioned above will not be violated. WSR-88D RFR will exceed the human-exposure standards of the former USSR, but only within the near field of the radar.

V.C.4.2 IEEE (1991) GUIDELINES

In 1982, ANSI Subcommittee C95.IV adopted a frequency-dependent standard for both occupational and public exposure to RFR (ANSI, 1982). That standard was based on a maximum whole-body SAR of 4 W/kg, reduced by a safety factor of 10, to 0.4 W/kg. It covered the frequency range from 300 kHz to 100 GHz. Those limits were not to be exceeded for exposures averaged over any 6-minute period. In the 1982 ANSI standard, the incident power density limit for the range 2.7 to 3.0 GHz of WSR-88D was 5 mW/cm^2.

In 1988, the functions of ANSI Subcommittee C95.IV were transferred to Subcommittee IV of Standards Coordinating Committee 28 (SCC 28) on Non-ionizing Radiation Hazards, a new body under the jurisdiction of the IEEE. The subcommittee selected and analyzed the important research papers in an updated data base of the RFR-bioeffects literature and prepared a revision of the 1982 ANSI standard. That revision, the IEEE SCC 28 guidelines, has recently been approved by the IEEE.

The IEEE (1991) guidelines cover the frequency range from 3 kHz to 300 GHz and separately specify the maximum allowable RFR exposure in "uncontrolled environments" (accessible by the general population) and "controlled environments" (such as occupational exposure). In the range from 300 MHz to 1.5 GHz, the new limits have a safety reduction factor of 50 instead of 10, and are averaged over any 30-minute period instead of over 6 minutes. In the range from 1.5 to 3.0 GHz, the new limits increase in accordance with the formula f/1500 (with the frequency f in MHz), whereas the limit was fixed at 5.0 mW/cm^2 in the ANSI standard. (See Figures B.1 and B.2 in Appendix B). Thus, in the WSR-88D frequency range of 2700 to 3000 MHz, the new limits on average power density for uncontrolled environments are 1.8 to 2.0 mW/cm^2 instead of 5 mW/cm^2. The new IEEE (1991) guidelines limits for controlled environments are 9 to 10 mW/cm^2 averaged over any 6-minute period.

The duration of each WSR-88D pulse will be either 1.57 or 4.71 μs, depending on the weather feature of interest. The number of pulses to be transmitted per second (the pulse repetition frequency or PRF) will range from 318 to 1304 pps. The time-averaged power level from the WSR-88D antenna will never exceed 1 kW. During normal operation, the average power density of WSR-88D RFR at the surface of the radome would be 0.6 mW/cm^2 – a level considerably lower than the maximum exposure levels in either the 1982 ANSI standard or IEEE (1991) guidelines.

At the lowest planned tower height, the WSR-88D beam axis will be 32 ft above ground level. During normal operation, for most units, the RFR beam will be elevated at least 0.5°, and the highest average RFR power density at heights of 6 ft or less from the ground will not exceed 0.005 mW/cm^2. Thus, during normal operation with scan pattern 31, the near-ground average power density will not exceed the maximum values specified in the ANSI standard or the IEEE (1991) guidelines.

During a 5-minute period of searchlight mode operation, the WSR-88D unit with a -0.5° beam elevation will generate a maximum average power density (averaged over 6 minutes) in the main beam of 3.85 mW/cm^2 within 800 ft of the antenna. That power density will not exceed the IEEE (1991) guidelines for controlled environments of 9.0 to 10.0 mW/cm^2. For comparison with the IEEE (1991) guidelines for uncontrolled environments, a 30-minute averaging period is required. If multiple 5-minute periods of searchlight-mode operation occurred during a 30-minute period, the IEEE (1991) guidelines for uncontrolled environments – 1.8 to 2.0 mW/cm^2 – could be exceeded. To prevent that hazard, use of the searchlight mode operation will be limited to one 5-minute period per 30 minutes and a safety plan to minimize human exposure will be prepared and implemented for searchlight-mode operation.

Also included in the IEEE (1991) guidelines are maximum allowable values for RF current flow induced within the feet of a person immersed in an RFR field or by the person's contact with an inanimate object (e.g., a fence or vehicle) electrically charged by immersion in an RFR field. Such limits are applicable only within the frequency range from 3 KHz to 100 MHz (where such effects can occur) and thus are not relevant to WSR-88D.

V.C.4.3 International Radiation Protection Association Standard (IRPA)

In 1988, the International Non-Ionizing Radiation Committee of IRPA, with participants from Australia, France, West Germany, Italy, the Netherlands, Sweden, the United Kingdom, and the United States, published guidelines for occupational and public exposure to RFR in the frequency range 0.1-300 GHz (IRPA, 1988). The occupational exposure limits in the range 10 MHz upward were based on a whole-body SAR of 0.4 W/kg; the limits for public exposure are fivefold lower (based on 0.08 W/kg). WSR-88D RFR would not exceed that standard at any location outside the radome.

V.C.4.4 EPA Abandoned Guidelines

For several years the EPA had been planning to issue an exposure guidelines for the public. On July 30, 1986, EPA published a *Notice of Proposed Recommendations* in which 1 W/kg, instead of 4 W/kg, was given as the basis for a public guidelines (EPA, 1986). The rationale for the lower SAR was a literature review (Elder and Cahill, 1984). The three options EPA proposed, which were based on risk, benefit, and cost analyses, were a tenfold, fivefold, or no reduction

from 0.4W/kg (i.e., 0.04, 0.08, 0.4 W/kg). EPA decided not to issue guidelines and has not done so to date. The WSR-88D RFR would comply with any of the proposed, but not issued, EPA exposure guidelines.

V.C.4.5 National Council on Radiation Protection and Measurements (NCRP)

In a report by Scientific Committee 53 of the National Council on Radiation Protection and Measurements, the 1982 ANSI limits were recommended for occupational exposure only (NCRP, 1986). The exposure limits recommended for the general population were based on 0.08 W/kg, a fivefold lower value than in the 1982 ANSI guidelines. The corresponding lowest power-density limit in the NCRP report is 0.2 mW/cm^2, which is applicable to the frequency range 30 to 300 MHz. This reduction was based on the assumption that the public is exposed continuously (168 hours per week) and that the ratio of 40 hours in the work week to 168 hours is about 0.2.

The foregoing maximum permissible exposure limits in areas accessible to the general public are meant to apply to the sum of the RFR levels from all sources rather than to the levels from each source alone. Since the highest levels from the WSR-88D radar will be about 100 times lower than the lowest limit proposed in EPA (1986) and about 200 times lower than the NCRP (1986) limits, the emissions from the WSR-88D radar will not materially add to the emissions present in those areas from any other sources.

V.C.4.6 Former USSR Standard

Of interest are the exposure standards issued in the USSR before its recent transformation into a confederation of independent republics. Presumably, those standards remain in effect during the transition. For the WSR-88D range of 2700 to 3000 MHz, the exposure limit for the public is 0.01 mW/cm^2, a level much lower than in ANSI (1982) or IEEE (1991) guidelines. The military establishment of the USSR was exempt from exposure standards, but the U.S. military is not. However, during routine operation the average power density in the main beam of WSR-88D will be less than 0.01 mW/cm^2 beyond about 800 ft from the antenna.

V.D. HUMAN HEALTH EFFECTS OF POWER LINE FIELDS

V.D.1. Introduction

The frequency band of 30 to 300 Hz is known as the extremely low frequency (ELF) band. Power transmission systems in the United States and Canada operating at a frequency of 60 Hz (and European power lines at 50 Hz) are within the ELF band, as compared to the RF band of 3 kHz to 300 GHz (3,000 to 300,000,000,000 Hz). Because of the frequency differences, the power density concept discussed in the RFR bioeffects sections above is not applicable to power line fields. Instead, possible bioeffects are analyzed in terms of electric-field (E-field) strength and magnetic-field (H-field) flux density. This section describes actual levels of E- and H-fields near power transmission lines, summarizes findings from the scientific literature about the biological effects of exposure to such fields, and compares the field strengths with adopted standards.

Power line fields are characterized by voltages and currents. Voltage is a measure of the electrical potential energy that makes electric charges flow through a circuit. Current is a

measure of the rate of flow of electric charge in a circuit (e.g., a power line). The amount of power flowing is proportional to the product of voltage and current.

Voltages on any wire produce an E-field in the region surrounding the wire. Power line electric fields are characterized in space by the difference in voltage between the power line and the ground, expressed in volts over a distance of 1 m (V/m), usually at ground level. An H-field is produced when electric current flows in a wire. Power line H-fields are measured in units of milligauss (mG).

E- and H-fields are also created by household wiring and appliances. E-field levels in the centers of various rooms in a typical American home vary from 0.8 to 13.0 V/m, as reported by the World Health Organization. E-field levels 30 cm from appliances are reported as about 2 V/m for an incandescent bulb, 30 V/m for a color television set, 60 V/m for a refrigerator, and 250 V/m for an electric blanket. H-fields may be fairly intense close to appliances but decrease rapidly with distance. H-field levels at 30 cm from color television sets, hair dryers, and electric blankets are about 20 mG, 70 mG, and 15 mG, respectively. At 1 m from those appliances, the fields decrease to less than 3 mG (EPRI, 1991). Trees and building materials may greatly reduce the strength of power line E-fields. H-fields are not affected by trees or buildings, but are shielded by ferrous metals (iron or steel sheets).

The physics of the interaction of E- and H-fields with conductive objects having a complex dielectric constant is well understood, but the biological mechanisms of interaction of such fields with entities such as living cells and tissues are the subject of current research. Alternating-current (ac) E- and H-fields induce weak currents and E-fields in people and animals. The 60-Hz E-field strength in biological tissue is approximately 1-million times smaller than the E-field strength in the surrounding air because of the extremely large dielectric constant of the water composing much of the tissue. In any region of the body, the externally induced field adds to, and subtracts from, the internal field existing in the absence of the external field. Biologic tissue is permeable to magnetic fields, with the internal magnetic field in the body essentially equal to the outside field.

V.D.2. Field Strengths Near WSR-88D Power Lines

For a long isolated wire, the surrounding E-field is proportional to the voltage and varies inversely with the distance from the wire. Similarly, the surrounding H-field is proportional to the current and varies inversely with distance. For 3-phase 4-wire systems like the WSR-88D power supply line, the situation is much more complicated. The currents and voltages on the three wires are displaced in phase (relative time) by 120°, which tends to cancel the E- and H-fields that exist at moderate distances from the wires. In addition, the fourth wire is grounded, which further reduces the resulting E-fields. Moreover, the resulting fields depend strongly on the geometrical arrangement of the four wires. Thus, the calculation of 3-phase fields is complicated and difficult.

Power will be supplied to WSR-88D by a 200-A, 3-phase, 4-wire service with 120 V to ground and 208 V between wires. The power lines may be aboveground or underground. (The underground service would have a different physical configuration from aboveground service). The E- and H-field levels that will be produced by the incoming above-ground power line can be estimated from measurements made by the Southern California Edison Company (SCE) near a substation in Orange County, California (SCE, 1991).

One 3-phase line (with four wires) near the substation is 30 ft above the ground, and operates at 12,000 V (line-to-line). For a current of 180 A, the maximum H-field strength observed 3 ft above the ground directly below the line was 15.6 mG. The H-field fell to half this value about 20 ft on either side of the line (SCE, 1991). That value is independent of voltage. The WSR-88D overhead power lines will probably be closer together and closer to the ground than the SCE 12,000 V line. The two differences tend to cancel, yielding 15.6 mG as a reasonable estimate of the near-ground level H-field generated by the WSR-88D power line.

The situation is considerably different for WSR-88D power lines underground. In this case, the distance between the power line and humans at ground level is greatly reduced, but the current-carrying wires are grouped together and twisted into a cable. Those two effects tend to cancel each other, and the 15.6 mG estimate of H-field strength is reasonable.

The E-field at the same location in relation to the SCE power line was 60 V/m and again dropped to half that value at 20 ft from the below-the-line position. Because the WSR-88D power line will use only 208 V, that value for the E-field should be reduced by the factor 208/12,000 to obtain 1.04 V/m as the estimated value directly below the WSR-88D power line.

Electric fields are strongly attenuated by earth. Therefore, the strength of the electric field at near-ground level will be very low for an underground power line.

V.D.3. Summary of Current Knowledge About Power Line Frequency Biological Effects

Reports of possible health effects from long-term exposure to power-line electric fields were first reported in the Soviet Union in the mid-1960s. Soviet workers in 400- and 500-kV switchyards occupationally exposed to 50-Hz E-fields with intensities from 2 to 26 kV/m reported headaches, fatigue, reduced sexual potency, and other changes. As a result, regulations were established for Soviet substation workers that allowed unlimited exposure to fields less than 5 kV/m and limited exposure times to fields higher than 5 kV/m.

The Soviet workers' complaints have not been reported by substation workers in other countries, except for two reports from Spain in the early 1970s. At the time they were reported, Western scientists considered the Soviet effects speculative for three reasons. First, it is difficult to ascribe a causal relationship to field exposure instead of some other agent present; for example, 100-Hz audible noise, transformer oil vapors, and, importantly, microshocks. Second, there was an emphasis in Soviet medicine at that time on their so-called "neurasthenic syndrome"* (Gordon, 1966). Third, Soviet workers were entitled to protection from any and all workplace hazards, even if the symptoms might be judged psychosomatic by Western standards (Krylou and Yuchenkova, 1973; Gordon, 1966; and Umansky, 1989).

In the United States, Marino et al. (1976) conducted a pilot study of long-term exposure of mice to 60-Hz fields. They reported effects in fields of 15 kV/m, including decreased growth, increased mortality, and changes in blood chemistry. They stated that shocks received by the animals when attempting to eat or drink in the cages immersed in the fields may have been responsible for at least some of the effects.

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* In this medical concept, the CNS is considered as the initiating, monitoring, and controlling functionary over all life processes. The concept has been discounted in Western medical practice for more than 50 years.

Marino, Becker, and several other investigators presented their opinions concerning the potential health hazards of two proposed 765-kV transmission lines at a common record hearing before the New York State Public Service Commission, 1976-1978. Most of the expert testimony discounted the potential for hazardous effects (Scott-Walton et al., 1979), and construction of one of the power lines began. (The application for the second line was withdrawn for reasons unrelated to the hearing.) However, the concerns leading up to the hearing effectively marked the beginning of major research into power line-field bioeffects to attempt to resolve insufficient, inconclusive, or contradictory research findings. Most of this research has been funded by the U.S. Department of Energy (DOE) and the Electric Power Research Institute (EPRI).

The New York State Public Service Commission's decision based on the common record hearing resulted in the investor-owned utilities in New York State (as well as the New York Power Authority) funding a $5 million, 5-year research program on EMF health effects. While it was in existence, the New York State Power Lines Project was a third major program, including studies of the 60-Hz H-field and combined E-/H-field exposures.

To date, these research programs have not confirmed hazardous effects from exposure to power line E- or H-fields. However, several health effects have been described that require additional studies to elucidate mechanisms causing the observed effects and to determine the potential significance of the observed effects.

V.D.3.1 Literature Reviews

The scientific literature has been reviewed numerous times during the last decade. This subsection summarizes and compares the conclusions reached by a number of the most prominent reviews. The American Institute of Biological Sciences (1985), the Florida Electric and Magnetic Fields Science Advisory Commission (1985), and the Western Energy Supply and Transmission Associates (1986) all noted that E- and H-fields at power line frequencies can, under some circumstances, induce a variety of effects in biological systems. Many of those effects are temporary and reversible, and are therefore considered nonhazardous. The strongest evidence for adverse public health consequences from EMF exposure comes from two lines of epidemiologic investigation: the childhood cancer residential studies and the leukemia/CNS cancer/male breast cancer occupational studies. Those reviews concluded that more research is needed on confirmation of the epidemiologic findings and also on mechanisms of biologic action. These reviews also considered it unlikely that human health effects will result from exposure to power line fields.

The Florida Electromagnetic Field Advisory Panel (1987) examined epidemiologic studies and laboratory studies. They stated that the epidemiologic study by David Savitz "showed a potential for slight increase in childhood cancer from exposure to H-fields from distribution lines." The panel noted that exposure levels that cause effects in laboratories occur at field strengths higher than normally caused by power lines. However, it is not possible to conclusively determine the possible risks from long-term exposure to power line fields. Responding to the panel's findings, the State of Florida adopted standards for power line field levels that were higher than the panel's recommendations (EPRI, 1991).

The New York State Power Lines Project Scientific Advisory Panel (1987) found that most of the project-sponsored research indicated no effects of concern: however, some studies did identify several areas of concern. This report pointed out Savitz's work on the possible

association between childhood cancer and residential power lines. The panel recommended further study into that possible association and mechanism of interactions between power line fields and living systems, including residential exposure.

The Congressional Office of Technology Assessment (OTA) sponsored a background study (Nair et al., 1989). The authors determined that the results "most worthy of concern" are effects on the CNS and the possibility of cancer promotion. Interactions of power line-frequency fields with the CNS may be very specific with respect to frequencies and intensities, and may vary with the magnitude and orientation of the local geomagnetic field. For that reason, a carefully planned research agenda is required (Nair et al., 1989). They also determined that "no experiment or theory clearly proves that ELF fields promote cancer or growth enhancement." As a result, OTA recommended further research, including cellular, whole-animal, and epidemiologic studies (EPRI, 1991).

DOE's Bonneville Power Administration (BPA) prepared a thorough review of power line-field bioeffects literature in 1989. Although itself not a consensus scientific literature review, that report included a review of 21 other literature reviews on bioeffects of E- and H-fields. The conclusion of most of those reviews was that E- and H-fields:

> such as those produced by transmission lines have not been shown to be a health hazard to people or animals. Because of the uncertainty in the research findings, a number of reviews have included cautionary recommendations for measures to limit human exposures to electric and magnetic fields. (BPA, 1989)

V.D.3.2 Laboratory Animal Studies

Exposure measures are different for animals and humans because their presence perturbs the surrounding E-field. Humans are bipedal, whereas most laboratory animals are quadrupedal. In a vertical E-field, a person's presence causes the field lines to be concentrated on the head and upper torso. Most of the time laboratory rats have their lower body surface in contact with the ground, and the field is enhanced over their backs, but to a much lesser degree than for the human upper body. In a uniform 10-kV/m E-field, the introduction of a human perturbs the field, enhancing the E-field at the upper body surface to about 180 kV/m. For a rat, the enhancement is to about 40 kV/m (BPA, 1989).

Body-surface E-field exposure is only one of several exposure measures. Small currents that flow to ground are induced in the body from external fields. The "short-circuit" current for the human is 160 mA for a 10-kV/m E-field and 1.6 mA for the rat. Finally, internal body-current densities may be the parameter considered most relevant. In the same uniform 10-kV/m E-field, internal current densities at the center of the body are 190 nanoampere per square centimeter (nA/cm^2) for the human and 16 nA/cm^2 for the rat, an approximately 12-fold difference. These factors must be considered when assessing reported effects in rats and how these results may relate to humans. Comparable scaling problems for extrapolation to humans apply to all laboratory animal models.

Considerations for H-field exposure are different. Because humans and animals do not perturb H-fields, no field enhancement effect takes place as it does with E-fields. The H-field is also not diminished within the body. However, alternating H-fields induce alternating E-fields in

the biological specimens, also giving rise to alternating currents in the tissues. These currents do not flow to ground, but "circulate" within the body. Calculations show that magnetically induced E-fields (and hence currents) in the rat and the mouse are 5.6 and 12 times smaller, respectively, than those in a human, for a given external H-field strength (BPA, 1989). Thus, if an effect arises from a certain internal current density, a mouse would have to be exposed to an external H-field strength 12 times that needed to cause the effect in a human. Again, exposure-scaling considerations are important in assessing implications for human exposure.

During some early E-field exposure experiments with rats, the animals experienced electric shocks when they attempted to drink from a water bottle in the cage. More recent experiments have recognized such problems and corrected them. Another important experimental artifact problem is that of rats and mice rearing on their hind legs. In very strong E-fields, this can cause corona at the tip of the nose or vibrissae. For this reason, contemporary exposure chambers are designed to prevent the animals from rearing. Ozone may also be produced by equipment in studies using strong electric fields.

Other experimental factors, such as noise and vibration from the laboratory field generators must also be considered possible sources of artifact in E- and H-field laboratory animal studies.

Beginning in the 1970s, U.S. researchers began investigating 60-Hz E-field exposure at levels comparable with those produced by transmission lines. Marino et al. (1976) exposed mice over 3 generations to E-fields up to 15 kV/m. Mice were allowed to breed, gestate, and give birth in the E-field. Compared with controls, exposed animals experienced higher mortality rates and slower growth. However, it is likely that the effects observed in this pilot study were the result of microshocks from feeding and watering devices, rather than E-field exposure.

Marino et al. (1977) exposed rats to 15-kV/m for 1 month. In some of 10 experiments, water consumption and body weight were depressed, adrenal and pituitary gland weights increased, and some blood parameters changed. Although the researchers interpreted these observations as an indication that 60-Hz fields are biological stressors, the decrease in water consumption and weight loss also may have resulted from microshock problems.

The most extensive long-term U.S. laboratory research on 60-Hz fields was conducted by Battelle Pacific Northwest Laboratories. The researchers took great care to eliminate or minimize potential sources of artifact (e.g., corona, ozone, vibration, noise, spark discharge). Phillips et al. (1981) described results of 20 studies addressing 12 major areas of investigation using rats, mice, and chickens. No consistent effects were found for heart and blood, susceptibility to disease, growth and metabolism, and overall hormone production, including serum corticosterone levels (a stress marker). Plasma testosterone levels tended to decrease for male rats exposed to 100 kV/m for up to 120 days (Free et al., 1981). The authors suggested that this may have been related to the rats' ground-hugging characteristics and the contact of the males' scrotums with the ground-plate electrode, possibly causing large current densities around the testes. The few effects seen on the endocrine system did not threaten life or impair reproduction.

In a study marred by a faulty transformer that yielded exposures at field levels much lower than planned, 4 successive generations of mice (1400 animals) were conceived, born, and raised in a 125- to 250-V/m E-field. No consistent effects on reproduction or body weight were found.

Other Battelle studies did find effects – primarily in behavior changes. These changes entailed a preference for the shielded side of a shuttle box in field strengths of 75 kV/m and

above (Hjeresen et al., 1980). Chronic stimulation through vibrissae and/or body-hair vibration in the 60-Hz field was believed to be the likely mechanism for the observed effects. Later studies used a sensitive visual-evoked-response test to look for effects on the developing CNS. Continuous exposure of rats to a 65-kV/m field from conception to 20 days of age did not affect CNS development (Jaffe et al., 1983). Rats exposed to 2 or 65 kV/m for at least 3 weeks showed significant changes in the hormones melatonin and serotonin. Melatonin is produced by the pineal gland in the brain. Serotonin is a brain neurotransmitter associated with sleep and wakefulness. Melatonin level normally rises nocturnally. In animals exposed to the E-field, this did not occur. The daily rhythms of the hormones returned to normal about 3 days after they were removed from the field (Wilson et al., 1981, 1983, 1986). Anderson et al. (1988) showed that the threshold for the melatonin effect is between 200 V/m and approximately 2 kV/m.

Battelle researchers hypothesized that melatonin effects might also affect prolactin and estrogen, thereby increasing the risk of mammary cancer. Leung et al. (1988) conducted two experiments on rats. In each experiment, female rats were given a drug that induces mammary tumors and then were exposed to 40-kV/m 60-Hz E-fields. No differences were found between the exposed and control groups in regard to the number of animals with tumors. However, when the results of the two experiments were combined, the number of tumors per tumor-bearing animal was higher in the exposed group. The suggestion was that the E-field promoted the development of tumors. However, an earlier study in France found no increase in mammary tumors in rats exposed to 50 kV/m (LeBars et al., 1983).

Battelle researchers conducted a 6-year study with an inbred strain of female swine that were exposed to 30-kV/m E-fields for 20 h/d (Sikov et al., 1987). A second group not exposed served as a control. No adverse effects were noted in adults, even after long-term exposure.

When the animals were first bred with unexposed boars, the control group had slightly greater fetal mortality levels and malformations. A second breeding resulted in significantly higher fetal malformations in the exposed group. Half of the female progeny were raised in the E-field, the other half were controls. The first breeding of these second-generation females also showed more fetal malformations in the exposed group than in the controls. The second breeding showed no differences in malformation rates between exposed and control animals. The overall results of this major study indicated no adverse health effects. The reproduction findings were inconclusive. It is unclear whether the results were related to E-field exposure, to an outbreak of disease that occurred during the study, or to other factors. The lack of baseline data on malformation rates for the species also poses a major problem in interpreting the study's results.

A much larger study with rats was undertaken to try to answer the questions raised by the swine study (Rommereim et al., 1988). Groups of rats were exposed to 0, 10, 65, and 130 kV/m. Compared with the control animals, field-exposed animals showed no significant increase in the incidence of fetal malformations.

Experiments have been conducted in several other laboratories. Graves (1985) exposed more than 20,000 chick embryos to 60-Hz fields with intensities ranging up to 100 kV/m through the 21-day incubation period. No effects from exposure were found for mortality, deformity, or weights of the embryos and 1-day-old chicks. Sham exposure or exposure at several levels from 0 to 100 kV/m produced no evidence of an exposure-response relationship.

Thompson et al. (1988) examined the possible influence of 60-Hz H-fields on leukemia in mice. Adult mice were injected with leukemia cells at the start of field exposure to assess whether H-fields promote the development of leukemia. Results indicated no effect of 60-Hz H-fields of 14 mG, 2G, or 5G on survival time, spleen weight, or body weight, and no increase in leukemia was indicated.

Duffy and Ehret (1982) found that E-fields up to 100 kV/m caused phase shifts in circadian rhythms of laboratory mice and in some cases caused torpor, with decreased body temperature and metabolism, in wild-type mice. The high E-field values needed for the effects suggests that stimulation of vibrissae or body-hair vibration may have been involved.

Seto et al. (1984) conducted a long-term study of rats (over 4 generations) exposed to 80-kV/m E-fields. Large numbers of animals were used so that small effects could be detected. There were no effects on fertility, fecundity, survival, health, or food or water consumption. Slight increases (i.e., 2-6%) in weights of kidneys and testes and in corticosterone were found in exposed animals. Effects were also seen on white blood cells. Additionally, some transient effects on growth and some diminished sexual responsiveness in males were observed. Taken together, these mild effects suggested to the researchers an overall pattern of mild stress, most likely via detection of the 80 kV/m E-fields through vibrassee and/or body-hair vibration, leading to chronic stimulation.

The New York State Powerlines Project, completed in 1987, included five experiments involving laboratory animals (Ahlbom et al., 1987). One study found that rats exposed to a 60-Hz H-field had reduced mortality rates from drug-induced seizures. The study also found that a 1-h exposure to 60-Hz H-fields at night decreased the pain-reducing effect of morphine in mice.

Another study looked at the perinatal behavior of rat pups exposed to both E- and H-fields before birth, and for the first 8 days after birth. The exposed and control rats required considerable conditioning to learn a behavioral lever-pressing test, with the exposed animals showing about a 20% lower response rate than controls.

Another study investigated the effects of 1-kV/m E-field and 500-mG H-field on the rates and patterns of response of rats trained to press a lever for a food reward. No effects were seen. However, a significant increase in response rate was found in five animals when exposed simultaneously to a 500 mG, 60-Hz H-field and a dc H-field of 260 mG (about one-half of the earth's H-field). The effect disappeared within 24 hours after exposure stopped.

One of the New York State studies used squirrel monkeys to examine E- and H-field effects on circadian rhythms. At low field intensities, no effect on feeding rhythms was observed. With increasing intensities, a slight increase in the length of the feeding period was found. The researchers suggested that these effects may not occur under natural conditions where numerous environmental clues are available to time circadian rhythms. Another New York study used 10 pigtailed macaques; 6 were exposed to 3-kV/m and 100-mG fields, and 4 to 30-kV/m and 900-mG fields over three 21-day periods. No effects were found on weight, blood-cell counts, blood chemistries, tests of motor skills, daily rhythms, pathology, or general behavior. Significant declines were found in concentrations of two neurotransmitters in cerebrospinal fluid, and one of these remained depressed in some animals for up to 21 days after exposure ceased. Some of these effects may have resulted from low-level stress as a result of external sensory stimulation

induced by indirect mechanical interaction of fields with skin and hair (Weigel, et al., 1987; Miller, 1986).

Blackman et al. (1988) exposed incubating chicken eggs continuously to electric fields at 10 V(rms)/m at either 50 or 60 Hz for 21 days. Within 1-1/2 days after hatching, the chicks were sacrificed, and their brains removed and tested for response to 50- or 60-Hz electromagnetic (EM) fields (at 15.9 V(rms)/m and 73 μT(rms) in a local geomagnetic field of 38 μT, 85°C, using Blackman's calcium-ion-efflux assay, which is described in detail in the following section (Section V.C.3.3). For eggs exposed to 60-Hz fields, the chicken brains showed a statistically significant higher calcium-ion efflux ratio when exposed to a 50-Hz EM field, but no difference when exposed to a 60-Hz EM field. For eggs exposed to 50-Hz fields, the chicken brains showed no differences in calcium-ion efflux ratios when exposed to 50- or 60-Hz EM fields. The authors commented that, "It is unlikely that these data can be fully appreciated and extrapolated to other species before the underlying mechanism of action is established or a health consequence of an increased calcium efflux from brain tissue is identified." Thus, these data presently do not support any suggestion that such exposures constitute a health hazard.

Numerous additional studies have been conducted on the effects of 60-Hz E- and/or H-fields on various laboratory animal species. Most of the studies do not report hazardous effects from exposure. A variety of behavioral and some endocrine system and neurotransmitter effects similar to those described above have been reported. However, relatively few of those effects have been confirmed by multiple laboratory studies.

V.D.3.3 Tissue and Cellular Studies

In vitro studies of ELF bioeffects have entailed a wide spectrum of exposure conditions. Some studies have used 60-Hz fields. Others have used microwave or RF fields, amplitude modulated at 60-Hz or other ELF. Still others have used pulsed fields. Some studies on basic mechanisms have used currents directly injected into the samples. Difficulties then arise in comparing field strengths in the *in vitro* studies with those induced in humans and animals by power lines. The relevance of *in vitro* to *in vivo* (whole animal) effects is also difficult to assess because *in vivo* feedback control systems normally offset or correct changes observed in the *in vitro* case. Hence, the relevance (if any) of tissue and cellular study findings to the intact animal – including humans – is difficult to establish.

Several publications have reviewed *in vitro* studies (e.g., Sheppard, 1985; Adey, 1988; OTA, 1989). Some consistent effects have been described, but they do not appear to be pathological in nature. Scientific interest has focused on a series of studies claiming that E-and H-fields can influence the rate of calcium-ion (Ca^{++}) binding and release from brain tissue (e.g., Blackman et al., 1985, 1988). Such binding and release was originally called "calcium efflux" in the 1970s, although those early studies investigated effects on the outer surface of brain cells. This imprecise nomenclature has been misinterpreted to imply that the original studies investigated the flow of Ca^{++} through the cell membrane.

Calcium is involved in many important physiological functions. Effects influencing Ca^{++} function *in vitro* could therefore possibly be of significance if the effect also occurred *in vivo* (i.e., animals or humans). Only one study has reported evidence of an effect on Ca^{++} *in vivo*; in that study cats were exposed to a strong radiofrequency field modulated at 16 Hz and did not show a difference in average "efflux" rates between exposed and nonexposed preparations, only

a difference between standard deviations (i.e., "noisiness" of measurement values) (Adey, et al., 1982). Interpretation of effects not involving changes in mean values is problematic, and is not normally considered in most bioeffects studies.

A puzzling aspect of the so-called calcium efflux studies is that some effects are claimed to be related to certain frequency and amplitude "windows" of the fields. For example, the so-called calcium efflux effect is most pronounced at 16 Hz, which was originally interpreted as being in the natural frequency band of the EEG of the animal being studied (chickens) (Bawin et al., 1975). Some *in vitro* studies on frequency-specific responses have shown effects at specific frequencies (including modulation frequencies of RFR carriers), but not others. Other, *in vivo* studies on amplitude-specific responses have shown that hormone response of rat adrenal tissue was affected by a 60-Hz field of 10 kV/m, but not by fields of 5 kV/m, 100 kV/m, or 1000 kV/m (Lymangrover et al., 1983).

One mechanism, ion cyclotron resonance, was proposed to explain the frequency-dependent effects of H-field exposure on calcium efflux (McLeod and Liboff, 1986; Liboff and McLeod, 1988). This hypothesis started with the proven basis that Ca^{++} moved through the cell membrane via specialized channels. The hypothesis speculated that by controlling the "tuning" of externally applied ac and dc H-fields for the Ca^{++}, specific "gating" of ion flow might occur. Such a proposition was counter to the evidence from earlier (chick-brain) calcium efflux studies in which cyanide was used to poison the preparation and prevent transmembrane phenomena. The calcium efflux effect still occurred when cyanide poisoning was used, leading the investigators to conclude that the phenomenon was mediated on the outer surface of the chick brain cells (Bawin et al., 1975), not via transmembrane phenomena.

Notwithstanding this earlier evidence, in one study the cyclotron resonance model was used successfully to predict experimentally observed resonance leading to an observed effect on the behavior of unicellular diatoms (Smith et al., 1987). In a study using turtle colon cells, resonance conditions did not result in predicted effects on sodium ion movement (Liboff, 1988).

The Liboff and McLeod ion cyclotron resonance hypothesis was criticized by Halle (1988) on the grounds that it violates the laws of classical mechanics. Halle pointed out that certain parameters were severely underestimated in the model and that errors had been made in its formulation. He further demonstrated that the corrected model was untenable.

Adey (1988) and Grattarola et al. (1985) discussed mechanisms by which weak E- and H-fields could affect cell membrane functions. The cell membrane consists of a lipid bilayer with numerous imbedded protein structures. These may protrude outside the membrane into the extracellular (and intracellular) spaces. In the extracellular space, the protein structures are in intimate contact with the chemical and electrical environment of the narrow channels surrounding cells (Adey, 1988). Ends of the protruding protein strands include receptor sites for antibodies, hormones, neurotransmitters, etc., and may be negatively charged. Various ions, including Ca^{++}, also bind to these receptors, resulting in changes "signaling" the presence of a stimulus. Biochemical amplification of the stimulus effect frequently occurs causing changes within the cell that depend on the type of antibody or hormone stimulus.

The authors above believe that external E- or H-fields induce weak extracellular electrical currents flowing in the intercellular space and give rise to this signal-coupling and transduction process. This theory is not proven, despite the elaborate theoretical models.

A growing number of *in vitro* studies are investigating the possible influence of E- and (more recently) H-fields on tumor promotion, including cancer. One model of tumor development involves at least three stages – initiation, promotion, and progression. Tumor development caused by X-rays and certain chemicals seems to follow these stages. Studies to date have not found damaging effects of 60-Hz fields on DNA in cells. (This is the effect thought to cause tumor initiation.) Therefore, it seems unlikely that 60-Hz fields could initiate tumors or cancer. However, some *in vitro* studies suggest that the fields may in act as promoters, increasing the chance of tumor formation following initiation by some other agent.

One study found that the growth of human tumor cells *in vitro* was significantly increased following 24-hour exposure to a 60-Hz H-field of 1000 mG and an E-field of 30 kV/m (Phillips and Winters, 1987). When this study was repeated in research sponsored by the State of New York, the findings were not supported (Ahlbom et al., 1987).

Another study reported that 60-Hz E-fields (10 mV/m to 1 V/m in the medium, corresponding to several kV/m outside) caused an increase in activity of ornithine decarboxylase (ODC), an enzyme associated with cell growth that is also increased by all known chemical tumor promoters (Byus et al., 1987). However, other chemicals that are not tumor promoters can also cause increases in ODC. Therefore, this study is inconclusive. It did suggest to the researchers that E-fields *may* act as tumor promoters, but the study may not be relevant because of the field levels involved.

Lyle et al. (1988) showed that a 48-hour exposure to a 60-Hz E-field (1 V/m in the medium, corresponding to tens or hundreds of kV/m outside) resulted in a marked inhibition in the ability of lymphocytes to kill cancer cells *in vitro*. This inhibitory effect dropped to zero when the field was reduced to a level comparable with the maximum field that humans could be exposed to in the vicinity of power transmission lines. In addition, the researchers pointed out several reasons, separate from the exposure scaling consideration, why the results of their *in vitro* study could not be extrapolated to humans.

V.D.3.4 Epidemiologic Studies

Although some epidemiologic studies of power line field effects on humans have reported positive findings, the findings are open to question because not all of the criteria because of methodological deficiencies. Additional problems include: (1) relative risks that are sufficiently low (1.5-3.0) that epidemiology becomes a very coarse tool and (2) the fact that as exposure assessment becomes increasingly refined (thereby reducing misclassification), we fail to observe increased relative risks, as conventional epidemiologic wisdom would have us believe. In these circumstances, the positive studies may be said to be suggestive of an effect, but can never prove that it does (or does not) exist. Thus, the most important conclusion that can realistically be drawn is the need for more (and better) studies, particularly on mechanisms of biologic interaction.

Most people in modern society are normally exposed to E- and H-fields, either in the home from household wiring and appliances or at work from office or factory systems. Indeed, it would seem difficult to find large numbers of persons who do not receive E- and H-field exposures. For this reason, studies are often carried out that compare human populations identified as having "normal" exposure levels with populations presumed to have "above-normal" exposures.

Persons assumed to have above-normal exposures include those exposed by virtue of their occupation (e.g., utility linesmen, electrical switchyard workers), or in certain residential situations (e.g., residents of houses adjacent to high-voltage power transmission lines, those who sleep in electrically heated beds). Virtually all occupational studies have used job title as a surrogate exposure index.

An early French study (Strumza, 1970) reported that families living within 25 m of transmission lines sought medical consultation and bought medical supplies on the average about as frequently as families living more than 125 m away from such lines.

An English study (Reichmanis et al., 1979; Perry et al., 1981) reported a correlation between suicides and weak ac H-fields. Suicides over a 7-year period were related to the ac H-field measured near the front door of residences. The mean ac field for suicide residences was 0.87 mG, whereas that of control residences was 0.71 mG. It is difficult to give credence to these findings because of the closeness of the measured average field values and because the measurements were made at the doors of the residences. Other reasons for discounting this study are: (1) lack of a defensible *apriori* hypothesis; (2) the suicides occurred over an extended period, during which magnetic field levels could change due to changing load patterns; and (3) the cases had to live in their residence for only 14 days before their suicide. With the complex pathology of suicide, the latter requirement is absurdly short. A later large-scale, well-done British study (McDowall, 1986) found no indication of an association between suicide and proximity of residences to power lines or substations.

Perry and Pearl (1988) measured 50-Hz H-field strength in multistory buildings in Great Britain. Those who lived "near" power cables averaged exposures of 2.9 mG; those "distant" averaged 1.12 mG. No statistically significant differences were found between the two groups for illnesses involving pregnancy, cardiovascular disease, pediatric problems, malignancy, or psychiatric problems. The researchers said that their findings were not conclusive about an effect of H-fields (presumably because of the small values of the fields and the relatively small difference between exposure values of the two groups).

Severson et al. (1988) carried out a case-control study of 114 cases of adult acute nonlymphocytic leukemia and 133 controls with estimated and measured residential exposure to power-frequency magnetic fields in western Washington state. Magnetic field exposure was estimated from external electrical wiring configurations within 140 ft of each subject's residence: Magnetic fields were also measured inside each subject's residence at the time of interview. There was no consistent evidence of an increased risk of acute nonlympocytic leukemia associated with residential exposure to power-frequency magnetic fields, either from directly measured fields or from the surrogate values based on the wiring configurations.

Wertheimer and Leeper (1989) provided a reanalysis of Severson et al. (1988) that supposedly resulted in a strengthening of the association between acute nonlymphocytic leukemia and residential exposure to magnetic fields. Severson et al. (1989) responded that the post-hoc analyses were scientifically inappropriate and led to erroneous conclusions, and in any case indicated, "if anything, risk decreases somewhat as exposure increases."

The first study to suggest an association between childhood cancer and power-system magnetic fields was a case-control study in the Denver, Colorado metropolitan area (Wertheimer and Leeper, 1979). Residences of persons who died of cancer before age 19 in the greater Denver

area were compared with a group of control addresses. In-home exposure was assessed on the basis of the apparent (to an observer of the home) current-carrying capacity of nearby transmission and distribution lines and the distance of the home from these facilities. The authors found that children who died of cancer were 1.6-2.2 times more likely to have lived near high-current-configuration power lines than were the children without cancer. The Wertheimer and Leeper study was subjected to considerable scrutiny. Among the limits of the study were: biases due to survival aspects introduced by including only cancer deaths rather than all cancers diagnosed; the bias introduced by residential mobility (the study examined only the address at time of death, not when the cancer was first diagnosed); the validity of estimating exposure inside the house from the appearance of outside wiring (rather than measurement of fields); and the failure to account for other, possibly confounding, factors.

Four other studies on childhood cancer were undertaken. Two of them (Fulton, et al., 1980; Myers, et al., 1988) found no association between leukemia and estimated exposure to H-fields. The other two found positive results. The first of the studies reporting positive results considered children diagnosed with cancer in Stockholm County, Sweden, from 1958-1973 (Tomenius, 1986). The authors reported more electrical installations near dwellings of children with cancer than for a control group. H-fields of 3 mG and higher were found twice as frequently at cancer-incidence dwellings than at control dwellings. However, the author pointed out that this finding was statistically significant only for this (arbitrary) 3 mG cut-off point. Above or below that level, differences were statistically nonsignificant. The average H-field for all cancer-incidence and all control homes was identical (0.7 mG). The author concluded that the findings suggested an effect, but that the results might be due to other factors.

The second study with positive results (Savitz et al., 1988) was designed essentially to repeat the methods used by Wertheimer and Leeper (1979), but with correction of several weaknesses in the earlier study. The Savitz study included actual measurements of E- and H-fields inside study homes. It confirmed that there was some correlation between the Wertheimer and Leeper wiring-code classification and childhood cancer, but the correlation was only marginally statistically significant. When actual H-field measurements were used, that statistical significance disappeared. Four hypotheses have been advanced to explain the higher association observed with wire codes than with measured fields: (1) wire codes may better reflect long-term time-averaged magnetic flux density than do point-in-time measurements; (2) wire codes may be an index for some other characteristic of magnetic field exposure not captured by present measurement protocols (e.g., transients, intermittents); (3) wire codes may serve as an index for some other causal exposure (e.g., traffic density); and (4) the results may be artifactual due, for example, to socioeconomic bias introduced when using random-digit dialing for control recruiting. Thus, it remains unproved that the H-fields *per se* caused or promoted the childhood cancers.

In reviewing these studies for the New York State Power Lines Project, Ahlbom et al. (1987) stated that although there is some consistency among the results, the data are insufficient to conclude that exposure to H-fields increases the risk of childhood cancer.

London et al. (1991) used a population-based tumor registry for Los Angeles County, California, to identify 232 children, ages from birth to 10 years, diagnosed with leukemia between 1980 and 1987. These cases were carefully matched with 232 paired controls. London et al. obtained measurements of magnetic field in the child's bedroom over 24 hours or longer for

164 cases and 144 controls, spot measurements of magnetic and electric fields for 140 cases and 109 controls, and wiring configuration estimates for 219 cases and 207 controls. No clear associations between leukemia risk and actual measured magnetic or electric fields were seen. An association between leukemia risk and the Wertheimer-Leeper wiring configuration was observed (Odds Ratio = 2.15). The authors concluded that their results supported an association between childhood leukemia risk and wiring configuration but not between childhood leukemia risk and direct measurements of electric and magnetic fields.

As reported by Singewald et al. (1973), occupational health studies were first performed in the early 1960s on 10 linemen who routinely performed "hot-line" maintenance on 345-kV transmission lines. They were given complete physical examinations over a 42-month period. No significant health changes of any kind were found. The linemen were further observed by a team of physicians over a 9-year period. The physicians reported that the exposure to the fields from high-voltage transmission lines caused no long-term health effects in this small sample group.

At a 1975 US/USSR symposium, Lyskov et al. (1975) reported that some workers in extremely high-voltage (EHV) substations had experienced problems that might be attributed to long-term field exposure. The Soviets also conducted studies of the families of men working around substations and 330- to 750-kV lines (Savin et al., 1978), but they found no effects linked to field exposure for male/female offspring ratios, miscarriages, stillbirths, or hereditary illnesses.

The Soviet reports prompted two Canadian studies, one on those working in 735-kV substations (Roberge, 1976) and the other on men working around transmission lines up to 500 kV and in electrical switchyards and substations (Stopps and Janischewskyj, 1979). No health effects were found that could be related to 50/60-Hz electric-field exposure. (Magnetic-field exposures were not addressed.)

Similar negative findings for health effects were obtained by Malboysson (1976) for Spanish men working in electrical substations and near high-voltage lines up to 400 kV, and by Knave et al. (1979) for Swedish workers in 400-kV substations.

A larger Swedish study (Nordstrom et al., 1983) examined 542 electrical workers. Of these, 20 showed a slight tendency for increased chromosome damage in blood lymphocytes. A comparable study in Germany found no significant difference in the frequency of the chromosome breaks of 32 workers who had been exposed for more than 20 years to 50-Hz fields of up to 12 kV/m in 380-kV switchyards (Bauchinger et al., 1981).

In a follow-up to the Swedish study of chromosome breaks, Mild et al. (1982) reported that damage could occur in leucocytes as a result of high-voltage pulses that simulated spark discharge shocks sometimes received by substation workers. This explanation was given further credence by Nordenson et al. (1984), who showed that spark discharges increased the frequency of chromosomes breaks in the human blood samples used in their experiments, but that 50-Hz current (similar to that induced by field exposure) in the samples did not.

Broadbent et al. (1985) conducted an occupational health study in England and Wales. The study was based on health questionnaires (no medical examinations) administered to 390 high-voltage-line workers. Differences in health measures were found to be associated with such factors as working overtime, working alone, and change in shifts. No significant correlations were found between health measures and 50-Hz E-field exposure.

Milham (1983, 1985) analyzed the information on age and year of death in Washington State of 429,926 male decedents for 1950-1979 and 25,066 female decedents for 1974-1979 and presented cause-of-death analyses (160 causes) for 219 male and 51 female occupational categories. One finding was an increase in leukemia in workers exposed to electric and magnetic fields. The 1983 study (a technical report from which the 1985 paper was derived) is reviewed and critiqued in detail in Section 3.1.1 of Appendix B. That critique indicated there are numerous reasons why little credence can be given to the author's claim that the higher proportional mortality ratios (PMRs) for acute leukemia and all leukemia are associated with exposure to electric and magnetic fields.

On its own, Milham's study did not provide convincing evidence. However, Milham's reports prompted other epidemiologists to look at occupational data in their locales. In California, Wright et al. (1982), reported a trend for increased leukemia incidence in men in 11 electrical occupations in Los Angeles County. Calle and Savitz (1985) calculated PMRs for Wisconsin men using the same 10 electrical occupations as Milham (1982). No significant excess in leukemia mortality was found, although PMRs were elevated for electrical engineers and radio and telegraph operators. Savitz and Calle (1987) also performed an analysis by combining their results with 10 other cancer studies that had used Milham's occupational studies. The combined analysis resulted in a statistically significant 1.2- to 1.5-fold risk for various types of leukemia for telegraph, radio, and radar operators; power and telephone linemen; and electrical and electronic engineers. However, there are several weaknesses in these epidemiologic studies (Jauchem and Merritt, 1991): (1) associations, where they are seen at all, are weak (risk ratios of 2-3 at most), numbers of cases are small, and positive results are frequently at the limits of statistical significance; (2) exposure estimates are crude, based either on job categories, surrogate measures such as wiring configuration, or on a limited number of field measurements; (3) potential exposures to other carcinogenic or genotoxic agents in occupational studies are not well characterized or controlled for; (4) dose-response relationships are generally not observed; and (5) weak statistical analysis and experimental design flaws characterize many of the studies.

In a review of similar research, Coleman and Beral (1988) concluded that, taking all of the studies at face value, certain electrical workers seem to have about an 18% average increase in risk for leukemia. However, in the absence of other information such as exposure assessment and more rigorous analysis for other confounding factors, the cause or causes of the increased risk could not be determined.

In a case-control study, Thomas et al. (1987) assessed brain tumor risk associated with electrical and electronics jobs and with occupational exposure to ELF or microwave and radiofrequency radiation in 435 white men who died of a primary brain tumor and in 386 controls who died from other causes. Information on lifetime occupational history and other factors that might be related to excess brain tumor risk was obtained from interviews with next-of-kin. Thomas et al. reported that among electricians and power and telephone linemen combined (electrical tradesmen) who "are exposed to extremely low frequency electromagnetic radiation," relative risk (RR) for astrocytic tumors was slightly elevated, but was not statistically significant. This study therefore does not provide evidence that exposure to power line-frequency fields is associated with an increased risk of astrocytic or other brain tumors.

Matanoski et al. (1991) reported an excess of breast cancer in male telephone company workers having "histories of potential EMF exposure." Although the standardized incidence ratio (SIR) was 6.5, this finding was based on only two cases. The lower value of the confidence interval of the SIR was 0.79. In addition, the authors emphasized that the cases, employed as central office technicians, had a qualitatively different exposure from other technicians, who did not have breast cancer and whose major source of exposure was nearby 60-Hz ac currents. By contrast, the cases were exposed to "a fluctuating exposure pattern ... produced by the rapid switching environment" of electromechanical switches, "until the 1980s, the predominant technology in telephone switching offices." This study therefore seems to indicate that it is not the 60-Hz magnetic fields that are associated with incidence of male breast cancer, but rather unspecified fields from electromechanical switches. Matanoski et al. (1991) suggested that the report by Tynes and Anderson (1990) of an increase in the SIR for male breast cancer in electrical transport workers may also be due to "the magnetic environment around electric railway cars [that] may also be unusual when compared with the common 60 [sic] Hz ac currents." (Sweden uses a 50-Hz power system.) Neither study therefore provides evidence that 60-Hz magnetic fields *per se* cause male breast cancer.

Several studies have examined associations between the use of electric blankets or electric heaters (waterbed heaters or ceiling cable electric heat) and the occurrence of health effects. Wertheimer and Leeper (1986) conducted telephone interviews with 1318 families in the Denver, Colorado, area. Subjects were identified by published birth announcements. Additional information was obtained from birth records. Analysis of their data led Wertheimer and Leeper to report seasonal patterns in cases when electric blankets or heated waterbeds were used. For this group, for conceptions occurring from September through June, the gestation period was approximately 1 week longer than for conceptions in July and August. Miscarriages and spontaneous abortions were also stated to be more frequent in this group than in the nonuser group. Wertheimer and Leeper suggested that these findings could be related to field exposures, or, alternatively, to heat from the electrically heated beds.

Wertheimer and Leeper (1989) elaborated on the 1986 study in an attempt to determine whether heat or 60-Hz fields was the primary influence on the seasonal differences in fetal loss. They studied women in Eugene-Springfield, Oregon; one group lived in homes with ceiling cable heat, and the other lived in homes with electric baseboard heat. The cable-heated homes reportedly had H- and E-field strengths of about 10 mG and 10-50 V/m, respectively. The baseboard-heated homes had field strengths of about 1 mG and 10 V/m, respectively. They found no difference in the ratio of fetal losses to live births for the two groups of women.

Preston-Martin et al. (1982) studied whether there was a possible association between electric blanket use and myelogenous leukemia, a disease of the elderly who might be assumed to have greater lifetime exposure from more years of electric blanket use. They conducted telephone interviews of 224 cancer patients and matched controls in Los Angeles, California, to ascertain their use of electric blankets. No differences were found in the percentages of cases and controls in their use of electric blankets.

Two studies sponsored by the New York State Powerlines Project also included questions about the use of electric blankets by the subjects. Savitz et al. (1988) reported that only 5% of the case and control children in his study used electric blankets. Although children with cancer were 1.5 times more likely to have used electric blankets than controls, the ratio was not statistically

significant because of the small number of subjects involved. A study by Severson et al. (1988) in Seattle found no association between use of electrically heated beds and adult leukemia.

V.D.4. Findings

V.D.4.1 Laboratory Animal Studies

Laboratory animal research has not demonstrated that 60-Hz E- and H-fields pose a hazard to human health. Problems with this entire body of research include: (1) inconsistent research findings, (2) uncertainties about the mechanisms for claimed effects, and (3) inherent problems in extrapolating results from animal experiments to humans. The implications for human health of alterations in melatonin and certain neurotransmitter levels are unclear and are being investigated further.

V.D.4.2 Tissue and Cellular Studies

The major observation emerging from *in vitro* studies of cell and tissue preparations is that the reported effects exhibit a variety of exposure-response relationships. Some of the claimed effects seem highly questionable (e.g., those based on ion cyclotron resonance phenomenon through cell membranes). Other studies, in particular those investigating "calcium efflux" in chick brain preparations, postulate "window" responses (i.e., stronger fields have less effect than weaker fields). Although some scientists accept the findings of the calcium efflux studies, other scientists are skeptical about the findings and, indeed, all of the mechanistic work remains contentious to many bioelectromagnetics researchers. In any event, even if such effects do occur, no evidence exists that they are hazardous. Likewise, studies that purport to show mechanisms for tumor promotion by E- or H-fields have not been replicated or are considered unconvincing.

V.D.4.3 Epidemiologic Studies

Several studies among the many epidemiologic studies conducted to date have investigated a possible association between childhood cancer and power line H-fields and reported positive results. Only one study reported a possible association between adult cancer and power lines. About half of the approximately 30 studies in the scientific literature relating "electrical occupations" and cancer incidence or mortality claimed some statistically significant elevated risks.

The cancer types most frequently mentioned were leukemia and brain tumors. However, when each study is critically assessed, numerous technical, methodological, and interpretive questions remain, casting doubt on the validity of the study's conclusions. For example, actual exposures were either unknown or crudely estimated; subjects may have been exposed to other noxious or toxic agents; and the relative risk ratios were all small by standard epidemiologic risk criteria.

In spite of all the study design, analysis, and interpretation problems mentioned above, in this body of epidemiologic research half the studies report positive findings, and they cannot be dismissed. The occupational data are of concern and need further exploration. Given their methodological problems, the most that can be said is that the studies do not convincingly indict power-system electric and/or magnetic fields as a causal factor.

The studies to date do not provide sufficient evidence that power-line fields are associated with any adverse effects on general health, reproduction, or the induction of cancer. However, the consensus is that additional epidemiologic studies designed to address and correct shortcomings of previous studies are warranted. A further impediment to the scientific credibility of the causal relationship implied in the epidemiologic studies is the lack of any generally accepted mechanisms of biologic action mediating cancer promotion by magnetic fields.

V.D.4.4 Overall Conclusion on the Basis of the Scientific Review

Laboratory research has not demonstrated that power-line fields pose a human health threat. Some epidemiologic studies have reported associations between health effects and assumed exposure, but because of the methodological difficulties inherent in such studies, the validity of the findings has not been convincingly demonstrated. Nevertheless, further research is warranted.

Although the occurrence of health effects from exposure to power line fields is controversial, the requirements for siting the WSR-88D are such that the power lines serving the radar sites are not expected to create new exposures to power line fields. The WSR-88D sites will draw power from existing distribution lines, and extension of existing lines is generally expected to be necessary only in unpopulated or very lightly populated locales and therefore will probably not be near homes or businesses.

V.D.5. 60-Hz Safety Standards

There are no federal standards or guidelines for human exposure to 60-Hz electromagnetic fields. Several private organizations and states have adopted guidelines for human exposure to 60-Hz fields.

The American Conference of Governmental Industrial Hygienists (ACGIH) in 1991 recommended that occupational exposure to electric fields not exceed 25 kV/m and that magnetic fields not exceed 10 G (10,000 mG). These values are guides, not strict delineations between safe and unsafe exposures.

The International Radiation Protection Association (IRPA) published interim guidelines for exposure to 50 and 60 Hz fields in 1991. For electric fields, IRPA recommends that short-term public exposure not exceed field levels above 10 kV/m and that continuous (24 h/d) exposure not exceed field levels of 5 kV/m. The IRPA guidelines for H-fields recommend against short-term exposure to field levels above 10 G (10,000 mG) and continuous exposure to field levels above 1 G (1,000 mG). Australia has adopted the IRPA guidelines.

The Delmarva Power Company, which serves all of Delaware, has established the following limits for field levels at the edge of rights-of-way (ROWs) for 69-kV transmission lines: E-field, 2 kV/m; H-field, 200 mG (EPRI, 1991).

Table V.3 lists limitations adopted by some states to protect public health from exposure to 60-Hz E- and H-fields. Most states have not adopted limitations on E- and H-fields. These limitations generally apply to either fields measured at a height of 3.3 ft (1 m) above the ground at the edge of the power line ROW or to the maximum allowable value anywhere within the ROW. Brentwood, Tennessee has adopted a local ordinance restricting "spillage of EMFs associated with ... transmission lines" to 4 mG at the edge of ROW (EPRI, 1991).

The maximum levels expected directly below the WSR-88D power lines will be about 15 mG (H-field) and 1 V/m (E-field). Limitations adopted by states for power-line E-fields range from 1 to approximately 12 kV/m, or roughly 1000 to 10,000 times greater than the maximum E-field that WSR-88D power lines will generate. Limitations for H-fields adopted by states range from 150-200 mG at th edge of the right-of-way, or roughly 10 times the maximum H-field that WSR-88D power lines will generate. The WSR-88D power line H-field levels will be somewhat larger than average H-field levels of 0.5-10 mG in residential settings (EPRI, 1991). However, most individuals, when near appliances, are frequently exposed to 60-Hz H- and E-fields that are stronger than the fields generated by the WSR-88D power lines.

Table V.3

STATE LIMITATIONS ON POWER-LINE ROW E- AND H-FIELDS

State	Standards	
	Electric (kV/m)	Magnetic (mG)
Florida	2	150-200*
Minnesota	8	—
Montana	1	—
New Jersey	3	—
New York	1.6 – 11.8*	200
Oregon	9	—

Source: EPRI, 1991

*Varies with the size of the power line

V.E. ELECTROMAGNETIC INTERFERENCE AND HAZARDS TO SYSTEMS

V.E.1. Introduction

This section discusses the potential electromagnetic effects of the operation of a WSR-88D radar on other systems and activities. These other systems include some that use the electromagnetic spectrum, as well as others that are not designed to be users of the electromagnetic spectrum but may nevertheless be susceptible to radiated energy. The first group includes telecommunication and radionavigation systems, which are designed to sense electromagnetic energy. The second group includes cardiac pacemakers, fuel handling, and electroexplosive devices (EEDs), which may inadvertently respond to the radar energy.

To estimate the likelihood that an emitter of electromagnetic fields will interfere with some other system requires knowledge of the operating characteristics of both systems and of the means by which the electromagnetic energy is propagated from one to the other. It is common to speak of the threshold of susceptibility for a system that is subject to interference. It is the lowest level of undesired signal that will cause some perceptible effect on the susceptible system (or activity).

Typically, the threshold of susceptibility must be determined separately for each pair of interfering and potentially interfered-with systems, primarily because the threshold of susceptibility depends not only on the power density of the undesired signal at the potentially susceptible system (and therefore on the distance between them), but also on the frequency of the undesired signal and the characteristics of its modulation. Theory is useful in predicting likely modes of interference, and it can go far in helping to predict thresholds of susceptibility. Measurements, however, are often needed when theory is insufficient or to confirm theoretical results. Each new situation is usually unique in some way, and susceptibility thresholds applicable to that situation are often unavailable.

Susceptibility levels are either educated judgments or are based on measurements of only a very few units, generally selected in the hope that they are representative or typical. However, the units could be either more or less susceptible than the entire population of units of that type. The variation in the susceptibility levels of all the units of a type (taken as a group) may be quite large, but the variation is generally unknown. In addition, circuit designs change, and the susceptibilities of the systems change with them. The nature of RF propagation over irregular terrain is such that the level of the interfering signal will not be the same at all locations at the same distance from the source. At a given location, the level varies with time, and thus the use of expected, or median, values is common. That is also true of the desired signals, when they are applicable.

In some situations, it is not necessary to determine actual susceptibility; standards for maximum fields have been established so that the devices or systems are said to be safe if that field is not exceeded. Such is the case for EEDs and fuel handling.

Each WSR-88D site will differ from the others in terms of the numbers, types, locations, and operating frequencies of other nearby potentially affected systems. The operating frequency of each WSR-88D installation will be selected to reduce the likelihood of mutual interference between it and the other users of the radio spectrum.

At some WSR-88D locations, the unit and the principal user facility will not be collocated, and information will have to be passed between them using T1 communications links or microwave-line-of-sight (MLOS) links. MLOS links will be used only at Los Angeles, CA; Medford, OR; Missoula, MT; and Oklahoma City, OK. MLOS units will generate a signal 1000 times weaker than the WSR-88D signal. Thus, no effects will result.

Transmitters often radiate some power on frequencies, called harmonics, that are integer multiples of its fundamental frequency. (The second harmonic of a radar operating at 2800 MHz would fall at 5600 MHz and could potentially interfere with some other radar operating at 5600 MHz.) For that reason, some care will be taken when selecting the operating frequency of each WSR-88D to note whether any local systems operate on the WSR-88D harmonics and whether WSR-88D would operate on the harmonics of other local systems. As an example, the WSR-88D third harmonic could fall on a frequency used by the FAA for radar microwave links (RMLs). Katz et al. (1982) point out that interactions between the RML and the WSR-88D may occur if the WSR-88D frequency's third harmonic is the same as the operating frequency of an RML facility located in the immediate vicinity.

Transmitting systems can radiate noise and spurious signals that are generated somewhere within the transmitter and are far from the operating frequency. These will be at a very low

power level relative to the intended signal. For WSR-88D they will be at least 80 dB (a factor of 100,000,000) below the WSR-88D main beam power level and are often as far as 115 dB (a factor of 300,000,000,000) less powerful.

V.E.2. Interference with Other Radio and Radar Systems

V.E.2.1 Broadcast Radio and Television

Like all the other radars currently operating in the 2700- to 3000-MHz band, each WSR-88D radar may interfere with the reception of broadcast TV and radio in its immediate vicinity. When present, such interference is heard on the radio as a buzz at the radar's PRF (somewhere between 318 and 1304 pps for WSR-88D). As the radar antenna rotates, its beam is scanned past the radio receiver; given the pattern's lobing structure, the buzz becomes periodically more and less noticeable, sometimes disappearing entirely. Radar interference with television appears as dots on the screen. The presence or absence of the interference depends not only on characteristics of the radar, but also on the characteristics of the particular radio or TV receiver, and on the frequency and signal strength of the potentially interfered-with signal.

The extent to which WSR-88D may interfere with TV reception has been investigated by Kenney and Koppenhaver (1989). They calculated the interference susceptibility of typical television receivers. They also made calculations similar to those presented here of power levels generated by a WSR-88D unit. Interference predictions based on these calculations were compared with measurements on three television receivers. The tests were made at Norman, Oklahoma, on August 18, 1989. The television units tested were a 19-in. Daytron, a 25-in. Sharp, and a 12-in. black-and-white Panasonic. A laptop personal computer (PC), Zenith Supersport 286, and a camcorder were also tested. The power level of the WSR-88D transmitter was typical, but abnormal beam angles were used to obtain worst-case conditions. The distance was 2427 ft, and the frequency was 2995 MHz.

These tests indicate that television interference is unlikely, even at homes within a few hundred feet of a WSR-88D because the main-beam power remains substantially above the height of typical television antennas. Potential interference with residential radio sets was not investigated. However, the insensitivity of television receivers, which operate at higher frequencies than AM or FM radio suggest that radio interference is improbable. Results of these tests are summarized below:

1. During a normal 6-min WSR-88D scanning operation, using Volume Coverage Pattern 21, no interference of any kind occurred for any of the television sets, the PC, or the camcorder.
2. During a 1-min, 1 rpm, azimuth scan with antenna at an elevation of -0.5° (a condition not normal for WSR-88D) the color television sets experienced interference for just a fraction of a second. This slight interference was not objectionable.
3. The black and white television set was not affected by WSR-88D interference during the entire test.
4. The laptop PC experienced no interference.

5. The WSR-88D interfered with the audio and video circuits of the camcorder when the circuits were subjected to a field strength of 126 V/m.
6. The remote control functions of the Sharp television would not function properly when subjected to a field strength of 126 V/m.

The potential for significant interference with radio and television reception is extremely low – a finding supported by the experience to date with the Melbourne, Florida, WSR-88D. Operation of that unit, which is within several hundred feet of a residential subdivision, has not resulted in reports of interference.

V.E.2.2 Other Radar Systems

The 130 WSR-88D radars will share the band between 2700 and 2900 MHz with more than 600 other radars in the United States operated by FAA, the military, and NWS. Because some will be replaced by WSR-88D installations, the total number in service will remain about the same.

These radar systems are used for airport surveillance, airport ground-controlled approach, tracking weather features, and the like. Although a radar is commonly said to use a particular frequency, that is only the radar's center frequency; each radar actually uses a portion of the spectrum extending both above and below its center frequency. For example, the emission spectrum of an ASR-8 airport surveillance radar extends, at low levels, at least as far as 100 MHz above and below the radar's center frequency; however, the energy there is less than 1/100,000,000 as great as at the center frequency. Fortunately, these weak and dispersed signals do not seriously interfere with other nearby radars. Thus, more than 20 radars in the Los Angeles area now occupy the 2700-2900 MHz frequency band without serious interference.

Sangston and Schwartz (1984) and Edson (1984) analyzed the potential for interference between WSR-88D and other radars occupying the same or adjacent frequency bands. They made extensive calculations of the power spectra emitted and of the potential vulnerability of the radars involved. They also considered the possibility of front-end burnout if nearby radars point their antennas directly at each other. The Sangston and Schwartz report contains frequency-distance curves for a large number of radar combinations that indicate the frequency difference required to prevent interference between a particular pair of radars separated by a given distance.

The operating frequencies of the 130 FAA and NWS WSR-88D radars will be selected within the 2700- to 3000-MHz band to minimize mutual interference between each WSR-88D and the other radars already operating in its vicinity, as well as between adjacent WSR-88Ds. To prevent interference, a radar can be isolated from another radar by being separated sufficiently either in operating frequency or in geographical distance, or by an adequate combination of both. (An intervening mountain range will provide much better isolation than will an equal distance over flat terrain.) Thus, each installation will present a somewhat similar, but unique, situation. Where the available combinations of frequency and geographical separation are not adequate, special filtering will be required.

As a first step in the frequency selection process for each WSR-88D site the Government Master File (GMF) is used to identify existing equipment and their operating frequencies in the 2700-3000 MHz band. The second step is the measurement of the electromagnetic environment that exists at the candidate site. The results of these measurements are compared with those

predicted from knowledge of assigned sites and frequencies. Using these data the DoD Electromagnetic Compatibility Analysis Center (ECAC) selects two candidate frequencies with minimal risk of interfering with or being interfered with by existing systems. Finally, as the time for installation approaches, JSPO formally applies for permission to use one of the two candidate frequencies for operation of that particular site.

A large fraction of the designated WSR-88D sites have already been assigned frequencies that are not expected to interfere with other radars or systems that share the same frequency band. Results achieved to date indicate that allocation of frequencies for interference-free operation of the entire WSR-88D network will present no serious problem.

V.E.3. Potential Hazard Effects

This section discusses the potential effects of WSR-88D electromagnetic fields on equipment other than other radars and telecommunication systems. They are termed "hazard effects," because they describe three potentially dangerous situations that high-amplitude radiofrequency (RF) fields can cause under certain circumstances: (1) accidental detonation of EEDs, (2) ignition of liquid fuels as they are being handled, and (3) interference with the normal operation of implanted cardiac pacemakers. In addition to pacemakers, there are other implanted or attachable medical prosthetic devices; however, because they are principally in the developmental or prototype stage, little information exists about their susceptibility to interference. The implantable devices are to have the same resistance to interference as modern pacemakers (Toler, 1982).

V.E.3.1 Electroexplosive Devices

EEDs are commonly found at military air bases, where they are used to activate secondary explosive charges, to ignite propellant systems, and to actuate switches. They are also used in some military aircraft to jettison flares and wing tanks while in flight, to release externally carried missiles, and to activate ejection seats. There are still other applications, and the use of EEDs on modern military aircraft is common. EEDs are by no means limited to aircraft applications; a common electric blasting cap is an EED.

All EEDs are ignited electrically and hence are subject to accidental ignition from the following causes:

- Lightning discharge – Lightning-protective systems are normally provided to preclude the inadvertent ignition of EEDs by direct lightning strikes.
- Static electricity discharge – This is a hazard mainly for ground operations.
- Stray energy, such as transients and other forms of induced conducted energy from nearby electrical equipment.
- Radiated fields from RF emitters – If the RF field is strong enough, it can induce currents that will cause the EED to fire.

Although EEDs are susceptible to ignition by exposure to radiated fields, the degree of susceptibility depends on many variables: the sensitivity of the EED; the ability of the EED leads to capture RF energy; the frequency and power density of the RF energy; and the condition of

exposure of the EED – whether contained in a shielded canister, mounted inside an aircraft with shielding provided by the aircraft skin, or exposed to the environment with no shielding present.

Air Force safe exposure criteria are specified by AF Regulation 127-100(Cl), Explosives Safety Standards, (USAF, 1983). The safety criteria are based on a worst-case situation: the most sensitive EED currently in inventory, unshielded, and having leads or circuitry that could inadvertently be formed into a resonant antenna. The criteria apply generally to critical areas involving explosives assembly, disassembly, testing, loading, and unloading operations, and are based on the safe, no-fire threshold of the EED. This is intended to be a very conservative safety threshold, and exceeding it does not imply that the EED will fire. The actual firing threshold of the EED may be several orders of magnitude above the safe no-fire threshold. Table V.4 shows recommended maximum power densities at the WSR-88D frequency band, as well as the corresponding safe separation distances. Some of the recommended maximum power densities in the table are more conservative (i.e., lower) than the values derived from formulas given in AFR-127-100 (C1). EEDs are activated by heat and average the input over a substantial fraction of a second. The pulse duration and repetition rate of WSR-88D are such that the short-term average, rather than maximum pulse power, is appropriate for calculation of no-fire thresholds.

Table V.4

RECOMMENDED MAXIMUM SHORT-TERM AVERAGE POWER DENSITIES FOR EEDs AT WSR-88D FREQUENCIES

Exposure or Storage Condition	Maximum Power Density (W/m^2)	(mW/cm^2)
EEDs in exposed condition	31	3.1
EEDs in storage or transport, in metal containers, leads shorted	100	10
EEDs in storage or transport, in nonmetallic containers, leads shorted	100	10
Aircraft parked or taxiing with externally loaded weapons	100	10
Aircraft in flight with externally loaded weapons, or shipment of EEDs inside cargo aircraft	100	10

Source: USAF, 1983.

The most hazardous exposure situation is the handling of exposed EEDs, such as electric blasting caps, when setting up a blasting operation; this situation, then, rates the lowest recommended maximum power density and therefore the greatest safe separation distance. Table V.4 shows that the recommended maximum power density for exposed EEDs at 2.7 GHz, the lower edge of the WSR-88D band, is about 31 W/m^2 (or 3.1 mW/cm^2). Equation 15 of Section A.7 gives the equation:

$$U_f = 2.52 \times 10^6/R^2 \text{ mW/cm}^2$$

as the short-term average power density on the axis of the stationary beam. Substitution of 3.1 in this equation yields R = 900 ft as the safe separation distance for exposed EEDs that are struck by the main beam of WSR-88D.

Equation 14 of Section A.7 gives the value $U_n = 3.85$ mW/cm^2 as the short-term average power density in the near-field column. Because this value is substantially lower than the 10 mW/cm^2 value given in Table V.3 for all other conditions of EED exposure it follows that no point within the near field represents a hazard to EEDs unless they are in the exposed condition.

The use and handling of electric blasting caps are specifically addressed in a publication by the Institute of Makers of Explosives (1981), which may be more familiar or available to civilian users of blasting caps than the Air Force standard. The publication, which has been approved as a guide by American National Standards Committee C95 on Radiofrequency Radiation Hazards, does not provide safe exposure limits in terms of power densities. Rather, it recommends safe distances from common emitters of RFR; in some cases the distance is given as a function of transmitter power or effective radiated power (the product of the transmitter power and the antenna gain). The data in this document regarding radars are quite sparse and deal mainly with maritime radionavigation radar. The publication's best advice is:

> In cases where an uncertainty exists as to the nature of the radar signal as well as ground scatter and reflection of the radar signal, a recommended minimum distance of 1000 ft [about 300 m] should be maintained from the radar antenna.

This value is only slightly higher than the 900 feet calculated from the Air Force standard. Again, the WSR-88D radar will generally not be directed downward far enough to illuminate operations at ground level that close to the radar.

A document by Maine's Department of Public Safety (Maine, 1976) states that although

> The Institute of Makers of Explosives have not found it possible to detonate a commercial electric blasting cap by means of radar; nevertheless, it is advisable to keep them out of a radar beam.

Although WSR-88D RFR could theoretically detonate exposed EEDs within the near field, the EED would have to be illuminated by the main beam. Because the main beam of WSR-88D will generally not strike the ground in the near field, only airborne EEDs would be at risk. The likelihood of an airborne EED encountering the main beam is very remote.

V.E.3.2 Fuel Handling

Strong electromagnetic fields can induce currents in conductive objects and may produce open sparks when contact between conductive objects is made or broken. For that reason, there is concern about the handling of liquid fuel (such as when fueling aircraft) in the presence of strong electromagnetic fields. The Air Force, in T.O. 31Z-10-4, considers areas where the peak power is greater than 5 W/cm^2 to be hazardous for refueling operations, regardless of the source of the RF energy (USAF, 1971).

Because some WSR-88D radars will be situated at airports, the possibility that aircraft or vehicles being fueled would be subjected to unsafe power densities from the radar alone or from

the concerted action of WSR-88D and any other operating radars was considered. Equation (9) of Section A.4 shows 2200 mW/cm^2 as the maximum pulse power in the near field. This is less than one-half of the 5000 mW/cm^2 designated as hazardous by the Air Force. Thus, close-in direct main-beam exposure will not produce a power density hazardous to fuel handling.

V.E.3.3 Cardiac Pacemakers

Cardiac pacemakers are potentially subject to EMI, leading to the concern that the WSR-88D radar could affect pacemaker wearers in the air or on the ground in its vicinity. Whether WSR-88D will affect a pacemaker depends on the susceptibility of the individual device and on the level of the WSR-88D signal that reaches it. Although no directly applicable susceptibility data from which to make predictions of any hazardous regions for a pacemaker owner are available, the data that do exist suggest that the possibility of harmful interference is remote.

The heart can be considered to be an electrically operated pump. It is a set of muscles that contracts rhythmically in response to a periodic electrical impulse that originates naturally in a certain portion of the cardiac tissue. Some who suffer impaired operation of that natural pacemaker or of the conducting paths in the cardiac tissue rely on an artificial pacemaker, which supplies the electrical signal to make the heart beat when it should. Hundreds of thousands of people in the United States have pacemakers.

Four general types of cardiac pacemakers are employed, but by far the most common (80% to 90% of the pacemakers in use) is the R-wave inhibited type. The R-wave inhibited (synchronous) pacemaker supplies a pulse only on demand (i.e., when the heart requires it) and is often called a demand pacemaker. It senses the electrical signal of the main pumping action of the heart. If that signal fails to occur when it should, the pacemaker supplies the signal to trigger the heart's action. Although R-wave inhibited pacemakers are generally more susceptible to EMI than are other types, great progress has been made in recent years in reducing that susceptibility.

Pacemakers do not fail permanently when exposed to strong RF fields; instead, if the field is sufficiently intense, they may exhibit one of four types of dysfunction, of which the most common (for a synchronous pacemaker) is termed "reversion." This means that the pacemaker reverts to a benign fixed rate; it is designed to respond to RF by becoming, for the time being, an asynchronous pacemaker. Reversion is not always even considered a form of dysfunction. In fact, for purposes of monitoring the pacemaker's response (and thus the condition of its battery), it is routine to deliberately cause it to revert to fixed-rate pacing.

The preponderance of the available data on the susceptibility of pacemakers was developed more than 10 years ago at RFs around 450 MHz or at the ac power frequencies of 50, 60, and 400 Hz. A few measurements were made at frequencies around 3000 MHz. The ac power frequencies were chosen for experiments because exposure to them is virtually unavoidable; everyone, in day-to-day living, is exposed to fields or to physical contact with devices radiating at these power frequencies. For standardized testing, a pulse-modulated 450-MHz signal "was selected as a compromise frequency that represents good body penetration and has been used by expert personnel in the field" (AAMI, 1975). Some measurements were made at 3000 MHz, which was chosen because so many high-powered radars use that frequency. The frequency ranges between 400 Hz and 450 MHz, and between 450 and 3000 MHz, are very large, and few data have been published on them; the small amount of testing that has been done has typically been undertaken

for special purposes, such as investigating the safety of pacemaker owners in the vicinity of specific devices or systems.

The modulation of the RF field dictates, in large part, whether and how a field will affect a pacemaker. Pulse modulation is the form of modulation most likely to affect a pacemaker because the pacemaker is designed to sense electrical pulses, and a threshold of field intensity exists above which a given pacemaker will react to external RF pulses. According to Denny et al. (1977), at low PRFs (less than 10 pps) an R-wave inhibited pacemaker is likely to misinterpret such pulses as the heart's normal electrical activity and to become inhibited (i.e., to "feel" that it should not supply any pulses). At higher PRFs, it is more likely to revert to asynchronous operation. Long-term inhibition (for durations greater than about five normal heartbeats) may constitute a health hazard for some owners, whereas reversion to fixed-rate pacing is less serious.

Considerable research was conducted on pacemaker susceptibility to electromagnetic fields in the mid- and late 1970s, but research in this area has since decreased because a pacemaker susceptibility standard was developed in 1975 by the Association for the Advancement of Medical Instrumentation (AAMI, 1975). In accordance with that standard, the pacemakers now marketed operate in 450-MHz pulsed field-strengths in excess of 200 V/m without being affected. (Because the body penetration of 3000-MHz fields is less than that of 450-MHz fields, pacemakers would be unaffected by a 3000-MHz field of 200 V/m as well.) That 1975 draft standard also requires that the pacemaker be unaffected by unmodulated CW power-frequency signals directly coupled to the pacer at a 100-mV level. Susceptibility testing has now become routine, with the Biomedical Research Division of the Engineering Experiment Station of Georgia Institute of Technology conducting that work for all but one of the major U.S. manufacturers, as well as for many of the major foreign manufacturers (Toler, 1982). The latest (November 1981) draft version of the AAMI pacemaker standard (AAMI, 1981) describes performance tests, but no longer refers to EMI susceptibility testing. According to a cochairman of the AAMI pacemaker committee, the reference was omitted for commercial and political reasons. He agrees that the modern pacemakers are almost invulnerable to EMI (Flink, 1982).

Susceptibility levels based on pulsed 3200-MHz tests in 1975 were published by Mitchell and Hurt (1976). They used a pulse rate of 10 pps, and found that although some of the pacemakers tested reacted to pulse widths as long as 1 ms, none reacted to pulse widths of 2 μs, even when the field strength was increased to 1200 V/m – the maximum field strength available from their equipment. (WSR-88D pulse widths will range between 1.57 and 4.71 μs.) That report states that the levels reported therein "are believed most representative of the current state of technology" (for 1975). The report also states that:

> if pacemakers were designed and tested to be compatible with the minimum E-field level, viz 200 V/m, associated with the unrestricted 10 mW/cm^2 personnel exposure level, potential EMI situations would be substantially reduced or effectively eliminated.

Such a 200-V/m testing level, described in the 1975 draft standard prepared by the AAMI for the U.S. Food and Drug Administration, is now in use by independent university testing laboratories working under contract for manufacturers. Thus, the 10 mW/cm^2 threshold is no longer applicable. However, contemporary pacemakers are far less vulnerable than this standard implies.

Both Mitchell (1978) and Denny (1978) suggested that the manufacturers were then probably meeting the 450-MHz, 200-V/m level in their newer models. The 1975 measurements reported by Mitchell and Hurt in 1976 indicated that none that they measured were susceptible to 3200-MHz pulsed signals at levels as high as 1200 V/m. Denny stated in 1978 that the 450-MHz threshold for most of the newly released pacemakers was above 300 V/m. Toler stated in 1982 that none of the pacemakers even then being released were susceptible to pulsed 450-MHz fields of 200 V/m.

Manufacturers contacted in an informal 1978 survey stated that their newer pacemakers met the 1975 AAMI draft standard, and one manufacturer said that the manual for a particular model stated that it had been tested (at 450 MHz) to 295 V/m.

Few if any of the pacemakers described in the literature of 14 years ago are still in operation because new pacemakers must be implanted when the batteries of the old ones are exhausted; physicians implant pacemakers less susceptible to EMI.

Pacemakers are not particularly susceptible to signals about 3000 MHz; moreover, the pulse repetition rate of the WSR-88D radar is much beyond that of a normal cardiac signal, and a pacemaker does not confuse it with the naturally occurring electrical signals it is designed to sense. (If a pacemaker reacted at all, it would most likely revert to fixed-rate pacing.) The most relevant data are that of Mitchell and Hurt (1976), who tested older, probably more susceptible pacemakers near various types of radars. They stated that WSR-88D-like search radars operating in the frequency range 2400-2900 MHz "did not produce any significant pacemaker interference . . . primarily because such systems were located on a 50-75 ft tower and their operating frequency is significantly attenuated by the implant." They also noted that although air route surveillance radars might "cause many pacemakers to miss single beats as the radar beam scans past," this would be "an effect most likely unnoticed" by the pacemaker owner. Thus, although no directly applicable data on reactions of pacemakers exposed to WSR-88D signals exist, the available evidence strongly suggests that if reactions occurred, they would be unlikely to harm pacemaker owners.

V.E.4. Overall Conclusion

Because operating frequencies and sites for individual WSR-88D units will be selected to avoid interference with other radars, the potential for adverse effects on other radars is very low. WSR-88D signals could cause detonation of EEDs, such as blasting caps, in exposed condition within 900 ft of the unit, but that is unlikely because the WSR-88D beam will not strike the ground within that distance. No adverse effects are expected on cardiac pacemakers or fuel handling operations. Therefore, the WSR-88D system will not cause significant adverse EMI or hazards to systems.

VI POTENTIAL FOR SIGNIFICANT ENVIRONMENTAL IMPACTS

This SEA, which is based on the most recent available scientific information, has reexamined the potential for the WSR-88D Program to cause environmental impacts in the areas of RFR and power-line field bioeffects, EMI, and hazards to systems. With regard to RFR bioeffects, the WSR-88D unit in normal operation will not generate power densities exceeding standards established to protect human health. Further, there is no reliable evidence that exposure to RFR levels lower than existing guidelines poses a health threat. The searchlight mode will be used infrequently, and safety measures will be taken to prevent human exposure to the main beam of the radar to RFR levels exceeding health standards. Thus, no adverse effect on human health is expected to result from exposure to WSR-88D RFR.

Cumulative effects, thermal or otherwise, from simultaneous exposure to multiple sources of RFR are not likely because measurements at WSR-88D sites have shown low levels of background EMR. Because RFR exposures at levels below established guidelines have not been shown to cause bioeffects, accumulation of effects from a sequence of exposures at these levels from any RFR sources cannot occur.

The power lines providing electric service to WSR-88D units are similar to typical commercial and residential power distribution lines. The maximum electric field levels that they will generate will be about 0.1% that of state E-field limitations, while the maximum magnetic field level will be about 10% of state H-field limitations. The WSR-88D power line H-field levels will be somewhat larger than average residential H-field levels, although not larger than H-fields near electric appliances. Thus, the WSR-88D power lines will not add significantly to human exposure to E- and H-fields, and no human health effects are expected to result.

Tests of the WSR-88D unit at the OSF indicate that interference with radio and television broadcasts is very unlikely. The frequency band for specific units will be selected to avoid interference with other radars. Additionally, WSR-88D signals will not be a hazard to EEDs, fuel handling operations, or pacemakers. Therefore, indirect hazards to human health will not result from EMI caused by the WSR-88D.

With application of measures to mitigate unlikely but possible impacts – incorporation of safety measures into searchlight-mode operation and proper site and frequency selection – implementation of the proposed action will not result in significant environmental impacts. Therefore, JSPO finds that implementation of the WSR-88D Program will not cause significant environmental impacts.

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VIII LIST OF PREPARERS

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- Peter Polson, Ph.D., brain research, University of Adelaide, 20 years experience in biomedical engineering. Dr. Polson prepared the analysis of power-line field bioeffects and assisted in the analysis of RFR bioeffects.

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APPENDIX A

CALCULATION OF RADIOFREQUENCY RADIATION INTENSITIES

A.1. INTRODUCTION

In this appendix, an analytic procedure for calculating the intensity of radiofrequency radiation (RFR) in the vicinity of a WSR-88D radar is developed. Data obtained from the Next Generation Weather Radar Joint System Program Office (JSPO, 1983) and the National Telecommunications and Information Administration (Hinkle, 1983) have been combined with information available in textbooks and technical journals to develop mathematical expressions and graphical relationships that permit calculation of RFR intensity at specific locations. Because this document is general rather than specific to a particular site, the treatment is general. The results are presented in the form of analytic expressions and graphs, which make it easy to determine values for the peak electric field, maximum pulse power density, and average power density at selected points in the vicinity of the radar. Power densities at the center of the beam are also calculated to provide a basis for estimating their effect on personnel, on electrical and electronic systems, and on birds.

This analytic technique allows predictions that are quite accurate in free space; however, the results are affected by the presence of the ground and of objects such as trees, buildings, and power lines. In reality, terrain is irregular, and objects such as trees, buildings, and other structures are randomly distributed. When they block the line of sight to the antenna, they tend to absorb, reflect, and scatter the beam. In such circumstances, the strength of the field is lower than it would be in free space. In other situations, the power reflected from the earth or other objects adds to that propagated directly, thus increasing the intensity of the radiation. Under circumstances relevant to a WSR-88D radar, the electric field strength is rarely as much as doubled in this way. Field enhancement of this kind is much more important in calculations of maximum electric field strengths and power densities than of time-averaged power densities.

A.2. CONDITIONS AND ASSUMPTIONS

A large antenna that is many wavelengths in diameter produces a radiation field that is concentrated in a small volume of space and is commonly referred to as a narrow or pencil beam. The WSR-88D antenna falls into this class. The major characteristics of such a pencil beam (Hansen, 1976) are determined by the diameter of the antenna, the wavelength radiated, and the power distribution over the antenna surface.

The electromagnetic field generated by the antenna is normally described by dividing it into two regions – the near field and the far field – to which different sets of analytical conditions apply. The boundary between the two regions is not sharply defined; rather, RFR field conditions gradually change with increasing distance from the face of the antenna. In the near field, RFR power density is nearly constant; in the far field it, gradually diminishes with increasing distance

from the antenna. It is also necessary to distinguish between regions within or near the main beam and those at angles remote from it.

The mathematical description of the complete field produced by large antennas is complicated. Therefore, approximate expressions have been developed to facilitate calculation.

The following conditions and assumptions have been applied:

1. The antenna has a specific height, h, above ground level, and all elevations are referred to the center of the antenna.
2. The antenna rotates at a fixed speed about its vertical axis and follows the fixed scan pattern described in Section V.B.2. This pattern was used because it yields the highest (i.e., worst case) values of average power densities at and near ground level.
3. The main beam and its first five sidelobes have circular symmetry.
4. The intensity of the first sidelobe relative to the main beam is 0.0032 (-25 dB).
5. To simplify the analysis the transition between near-field and far-field conditions is taken as 800 ft. This approximation overstates power densities by factors as large as two to one for distances in the range of 400 to 2000 ft.
6. For distances less than 800 ft from the antenna, RFR power densities are assumed to be constant; at greater distances, power density decreases steadily with increasing distance.
7. During normal operation the greatest possible instantaneous field strength at any ground location will exist when the antenna main beam is at the azimuth of that location and has the minimum elevation angle of +0.5°. The results calculated on this basis can be easily adjusted to accommodate other values of minimum elevation angle.
8. The RFR duty cycle is taken as 0.0021 (0.21%), which is the maximum duty cycle for the system. The RFR duty cycle differs from duty cycles in other parts of the WSR-88D. In this appendix, duty cycle always refers to RFR duty cycle.
9. The calculation of RFR field strengths at any distance up to 20 miles from a WSR-88D radar is based on direct line-of-sight propagation because all other modes of propagation, such as ducting due to temperature inversion, diffraction, or tropospheric scatter or reflection, are weaker (Kerr, 1951). Ground-level areas that are shadowed by intervening terrain will be illuminated by the diffraction mode of propagation. The RFR field strengths in such areas will be overestimated because the calculations are based on direct line-of-sight propagation. The attenuation caused by trees and underbrush, which can reduce RFR values by a factor of 10 or more, is not included.

The calculations are intended to represent realistic estimates rather than precise scientific values. Many approximations are made; therefore, it is expected that the field strength at any

given location produced by operation of a WSR-88D radar may be either larger or smaller than the calculated value by as much as a factor of 2.

A.3. THE FAR-FIELD REGION*

The far field exists only at distances greater than 800 ft from the antenna; it is defined as a region over which the analytic conditions are constant and the fields vary inversely with distance (i.e., the power density varies inversely with the square of the distance). The conventional criterion for the distance from the antenna beyond which the far field exists is $2D^2/L$, where D is antenna diameter, and L is the radiation wavelength. For the WSR-88D antenna at 2850 MHz, this distance is 4550 ft.

A well-known and generally applicable equation for the power density on the beam axis in the far-field region of any antenna is

$$U = PG/4\pi R^2 \qquad (1)$$

where U is the power density, P is the radiated power, G is the antenna gain, and R is the distance; consistent units must be used. For WSR-88D, P = 475 kW and G = 35,500. To obtain results in the desired form of mW/cm^2 when the range is specified in feet, it is necessary to introduce suitable factors. To convert from kilowatts to milliwatts and from square feet to square centimeters, one must multiply by 10^6 and divide by $(30.48)^2 = 929$. Combination of these various terms leads to a key result: for the far field, the maximum pulse power density in the center of the main beam is:

$$U_1 = 1.44 \times 10^9/R^2 \ mW/cm^2 \qquad (2)$$

where R is the distance in feet.†

The size of the far-field main beam is limited by the system specification, which requires that the power density decline to half its maximum value in not more than 0.5° from the axis (see Figure A.1). The position of the first null is not specified. The value 1.25° shown in Figure A.1 was chosen from data on similar antennas (Silver, 1949). The 1° interval between successive nulls was chosen on the same basis.

Both the main beam and the many sidelobes of the WSR-88D radar will sometimes strike the ground. Therefore, it is necessary to consider the distribution of power in the first and higher order sidelobes as well as in the main beam.

System specifications require that the ratio of the power in the first sidelobe to the power in the main beam at the same distance be no greater than 0.0032 (-25 dB). The relative power densities in the first 5 sidelobes are controlled by the specification that the level shall decrease linearly from -25 dB in the first sidelobe to 0.0004 (-34 dB) at 10° from the axis. The distribution shown in Figure A.1 is consistent with this statement.

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*Here, and throughout this document, the term "pulse power" designates the maximum rms value of the pulse, when present.

† This formula is correct for a beam pointed in any direction. For most cases of interest in this document, the beam is nearly horizontal, and no substantial error results from taking R as a horizontal distance, which simplifies the discussion.

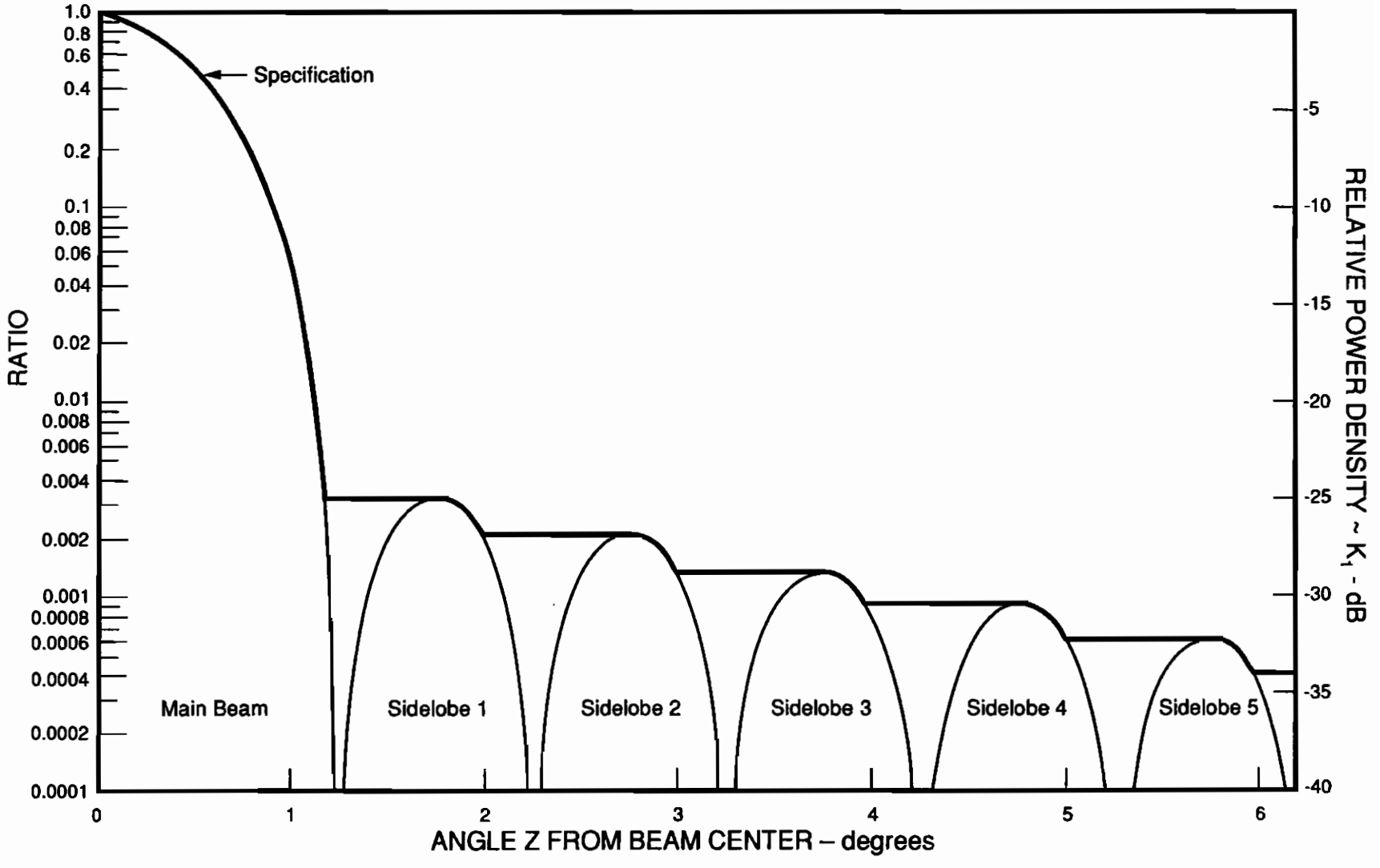

FIGURE A.1 Antenna Pattern for Far-Field Region of WSR-88D Beam

Locations that sometimes fall in one of the nulls shown in Figure A.1 are at other times subject to the higher power density of the adjacent peak. Therefore, all calculations of peak sidelobe power use the envelope function shown in the heavy line in Figure A.1. The envelope function is extended to the right at the value 0.0004 (-34 dB) because the system specification allows a few isolated peaks of this magnitude. Use of this value with reference to Figure A.1 and Eq. (2) leads to the equation

$$U_2 = 0.0004U_1 = 5.76 \times 10^5/R^2 \ \text{mW/cm}^2 \qquad (3)$$

which describes the maximum pulse power density at any location more than 6° from the axis of the main beam.

During normal operation, the main beam of a WSR-88D radar is in constant motion as a result of continuous rotation of the antenna around its vertical axis. This mobility has an averaging effect on the RFR power density. The result is to reduce the intensity of the main beam and near-in sidelobes and to fill in the nulls in the radiation pattern. The averaging factor will differ depending on whether the area is illuminated by the main beam or by some combination of sidelobes. The averaging factor becomes less important at close ranges, where the diameter of the radiation column is comparable to the distance through which it is swept.

A WSR-88D radar is capable of operating in many modes, and it is impractical to make a detailed calculation for each possible mode. However, a large fraction of the time is likely to be devoted to scanning modes similar to the one described in Section V.B.2. That mode has been chosen for analysis because it is typical and because it will produce maximum values of time-averaged power densities at locations at or near ground level.

Consider a location that is elevated 0.5° above the radar antenna and separated from it by a distance R. (The following analysis applies to beams at other elevations if all heights are referred to the center line of the beam.) At one moment during the 0.5° elevation scan, this point will be struck with the full power of the main beam with a maximum intensity of $U_1 = 1.44 \times 10^9/R^2$. At other instants it will be struck by all the sidelobes, as shown in Figure A.2. The exact shape of the power density curve, as sketched at the top of Figure A.2, is not known; however, no great error will result from assuming that each lobe has the shape of a half sinusoid. On this basis, the average value of each section of the lobe pattern is $2/\pi$ times the maximum value.

The technical specifications limit the maximum rotational speed of the antenna to 30°/s (5 rpm). Thus, even at the slowest pulse repetition frequency of 318 pps, the beam moves only 0.094° between successive pulses. Therefore, no significant error will result from treating the beam as continuous with a total power of 1 kW, calculated by multiplying pulse power, 475 kW, by duty cycle, 0.0021.

The 1991 IEEE SCC 28 exposure guidelines are stated in terms of averaging times not greater than 0.1 hour (6 minutes) for controlled environments and 0.5 hour (30 minutes) for uncontrolled environments. Therefore, as a worst case, we average over the 6-minute interval in which the beam elevation has the values 0.5°, 1.5°, and 2.5°. The relative power levels of the first five sidelobes of Figure A.1 are:

Sidelobe number	1	2	3	4	5
Relative level	0.0032	0.0021	0.0013	0.0009	0.0006

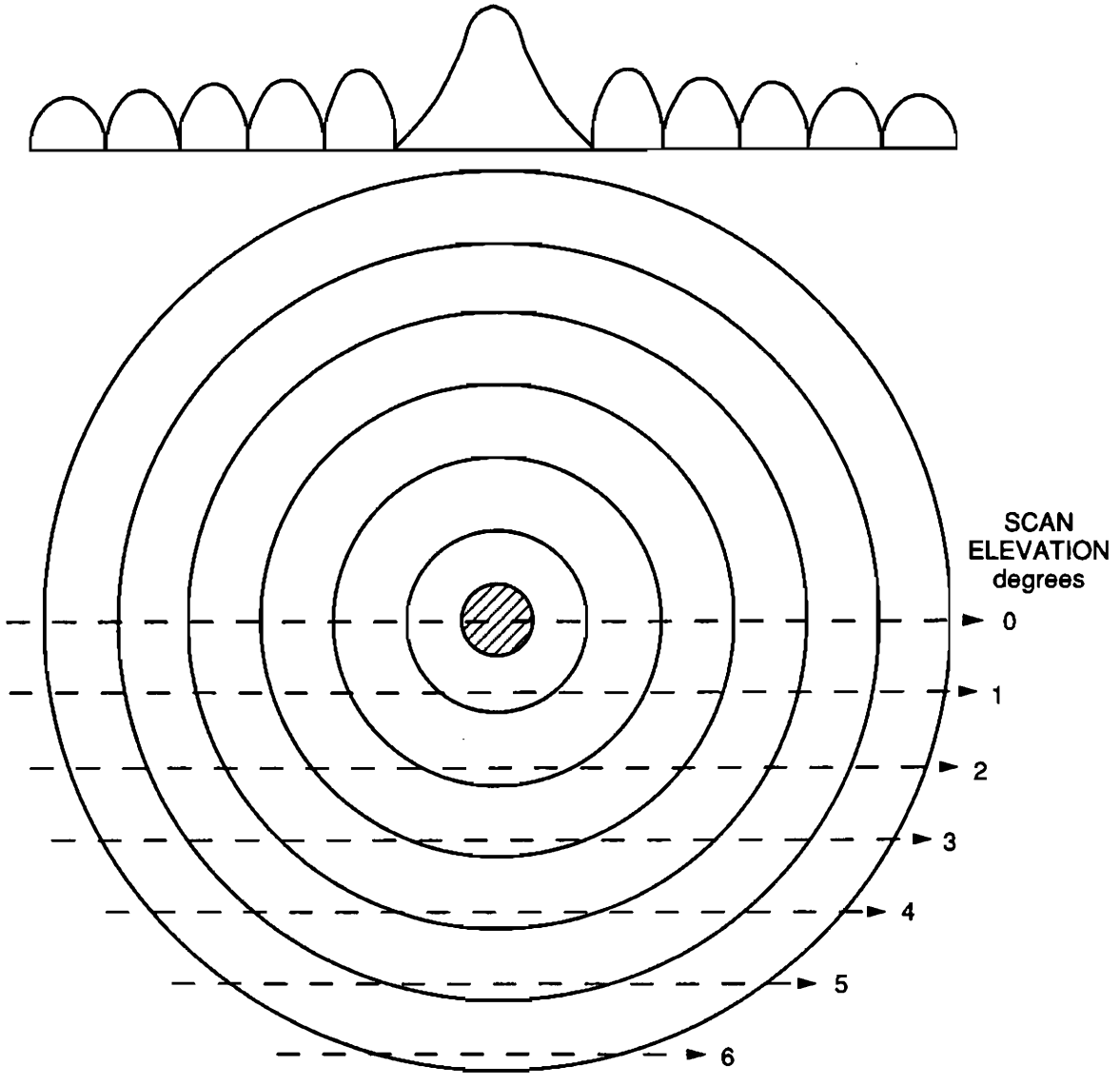

FIGURE A.2 Effect of Scanning by Main Beam and First Five Sidelobes

The width of each sidelobe is 1.0°; the half-width of the main lobe is 1.25°. Contributions from the 0.5°-level scan are listed in the first column of Table A.1. Contributions associated with other elevation angles are listed in successive columns. Table A.1 lists the product of the width and height of the energy pulse that results as the beam sweeps past the sampling point. These contributions are then added and finally referred to the zero-angle case to obtain the ratio K_2, which is plotted in Figure A.3. This curve, which applies to points either above or below the beam center, provides a basis for calculating time-averaged values of power density in the far field for any scanning sequence. In subsequent calculations the angular distance from the beam center is converted into a distance in feet.

Table A.1
FACTORS FOR CALCULATION OF AVERAGE POWER DENSITY

Sidelobe Number	Beam Elevation Above or Below 0.5°						
	0°	1°	2°	3°	4°	5°	6°
0	1.0 x 1.25 = 1.25	0.062 x 0.6 = 0.0372	0	0	0	0	0
1	0.0032 x 1.0 = 0.0032	0.0032 x 1.3 = 0.0042	0.0032 x 0.9 = 0.0029	0	0	0	0
2	0.0021 x 1.0 = 0.0021	0.0021 x 1.2 = 0.0025	0.0021 x 1.6 = 0.0034	0.0021 x 1.1 = 0.0023	0	0	0
3	0.0013 x 1.0 = 0.0013	0.0013 x 1.1 = 0.0014	0.0013 x 1.4 = 0.0018	0.0013 x 1.7 = 0.0022	0.0013 x 1.2 = 0.0016	0	0
4	0.0009 x 1.0 = 0.0009	0.0009 x 1.0 = 0.0009	0.0009 x 1.2 = 0.0011	0.0009 x 1.3 = 0.0012	0.0009 x 2.0 = 0.0018	0.0009 x 1.2 = 0.0011	0
5	0.0006 x 1.0 = 0.0006	0.0006 x 1.0 = 0.0006	0.0006 x 1.0 = 0.0006	0.0006 x 1.2 = 0.0007	0.0006 x 2.3 = 0.0014	0.0006 x 2.2 = 0.0013	0.0006 x 1.5 = 0.0009
Total	1.2581	0.0468	0.0098	0.0064	0.0048	0.0024	0.0009
Ratio K_2	1.00	0.0372	0.00078	0.0051	0.0036	0.0018	0.0007

The important conclusions to be drawn from Table A.1 are that the contribution of the main beam is much larger than the sum of the contributions of the first five sidelobes and that the power density decreases rapidly with increase of beam elevation angle. In particular, the contributions associated with a beam elevation angle above 4° are negligible.

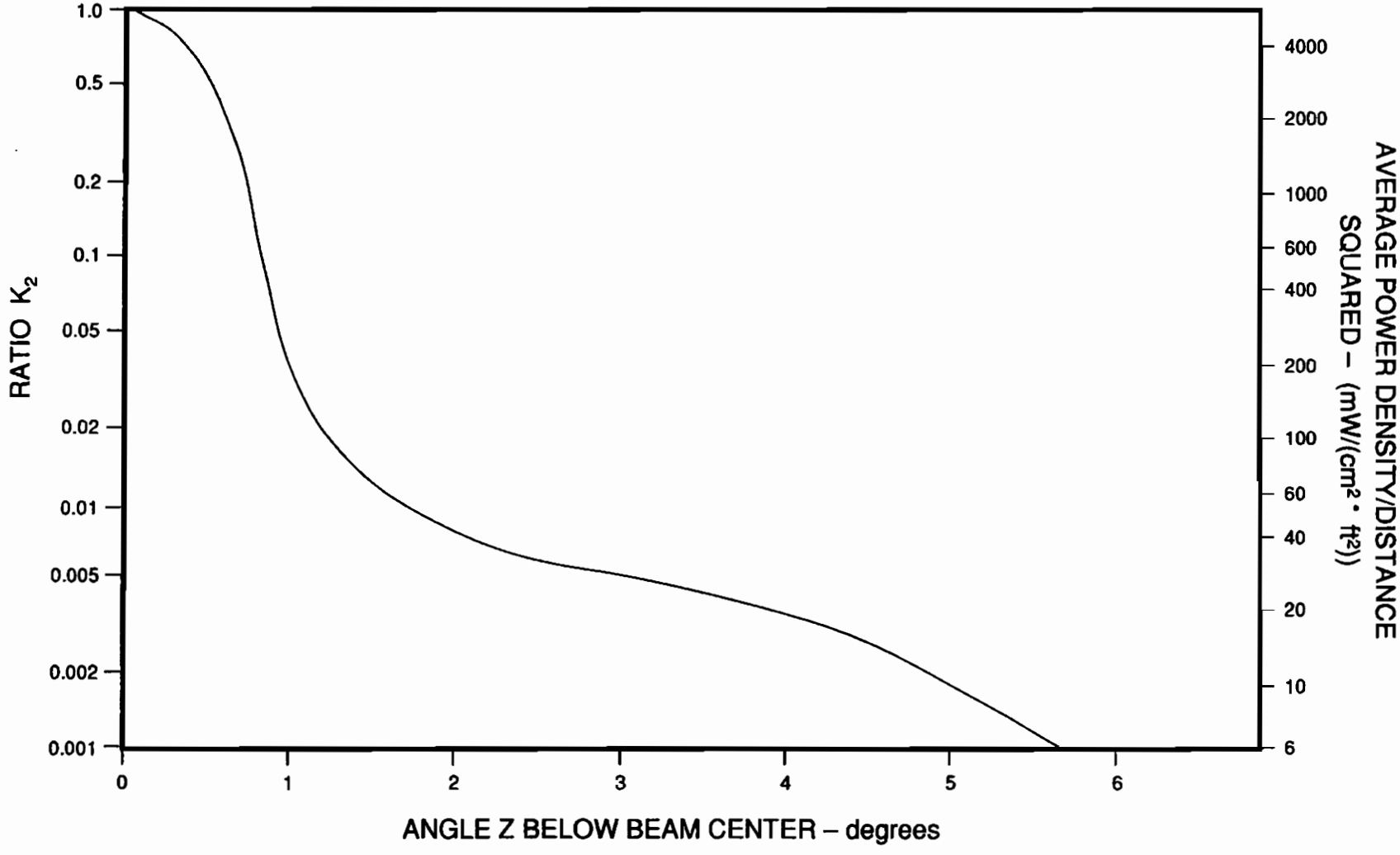

FIGURE A.3 Angular Variation of Far-Field Average Power Density

During one revolution of the antenna at 0.5° elevation the average power density at some point struck by the center of the beam is lower than the density at the center of the stationary beam by the factor

$$K_3 = (2/\pi)\ (1.2581/180) = 0.0044 \qquad (4)$$

where the coefficient $2/\pi$ represents averaging over a half sinusoid, 1.2581 is the total of Column 1 of Table A.1, and 180 represents one half revolution. The product

$$U_3 = 0.0021\ K_3\ U_1 = 1.35 \times 10^4 / R^2\ \text{mW/cm}^2 \qquad (5)$$

is the time-averaged value that would result from the main beam and the first five sidelobes if the antenna rotated continuously at 0.5° elevation.

Figure A.3 indicates that for a beam elevation of 0.5° the average power density at a point level with the antenna will decrease by a factor of 2 from that at points struck by the beam center. This is consistent with Figure A.1, which shows that the main beam contribution will be cut in half. The other contributions are essentially unchanged but make up a negligible part of the total.

For the worst-case 6 minutes of the selected scan sequence, the beam spends 2.5 minutes at 0.5° elevation, 2.5 minutes at 1.5° elevation, and 1.0 minute at 2.5° elevation. At points 1.5° and 2.5° from beam center, the coefficients scaled from Figure A.3 are respectively 0.012 and 0.006. The corresponding power density at a point level with the antenna is found by taking the sum

$$U_4 = [(2.5 / 6)\ (0.5) + (2.5 / 6)\ (0.012) + (1/6)\ (.006)]\ U_3 \qquad (6)$$

$$= 2900 / R^2\ \text{mW/cm}^2 \quad .$$

Recalling that the contribution of the main beam is dominant and doubles at 0° vs 0.5° we may double U_4 and write

$$U_5 = 5800 / R^2 \quad . \qquad (7)$$

The contributions of all the other sidelobes that radiate in all directions are addressed by noting that the system specification requires that throughout this region the antenna have a "median gain" not greater than –13 dB (i.e., 0.05). Median gain is defined as "that level over an angular region at which the probability is 50% that the observed or measured gain at any position of the antenna will be less than or equal to that level." This specification suggests, but is not fully equivalent to, a statement that the average gain over the entire region outside the main beam and first five sidelobes is 0.05 (–13 dB), which means that 95% of the available power is concentrated in the main beam and first 5 sidelobes and that only 5% is distributed in sidelobes further out than the first 5 sidelobes.

This 5% value is used to obtain the total time-averaged power density at the designated point. From Eq. (l) the average power density due to higher order sidelobes is

$$U_a = (1.00 \times 10^6 \times 0.05) / (4\pi \times 929R^2) = 4/R^2\ \text{mW/cm}^2 \quad . \qquad (8)$$

A.4. THE NEAR-FIELD REGION

Hansen (1976) has studied the near-field region of antennas like that of the WSR-88D radar. Figure A.4, which is redrawn from one of his figures, shows that the power density on the axis of the beam is never higher than +15 dB (i.e., 32 times its value at the reference distance $2D^2/L$, which, as noted in Section A.3 of this appendix, is 4550 ft). Thus, from Eq. (2), the maximum value of pulse power density on the beam axis in the near field is

$$U_o = (1.44 \times 10^9 \times 32) / 4{,}550^2 = 2200 \text{ mW/cm}^2 \quad . \tag{9}$$

This value is used for all points within the circular column of the near field out to the distance of $0.18 \times 2D^2/L = 800$ ft, which corresponds reasonably well with the 640-ft value previously derived by a different method.

Additional information concerning power distributions in the near field is provided by Figure A.5, which is based on work at the National Bureau of Standards (Hu, 1961). At the antenna face, the WSR-88D beam is highly focused. At 3000 ft from the antenna face, the beam is more diffuse, and substantial power densities exist up to 50 ft from the beam axis.

The average power density in the near field is calculated with reference to Figure A.6, which shows the axis about which the antenna rotates as well as the lowest and highest beam positions. In this region the beam is represented as a circular cylinder with a 28-ft diameter and a power density distribution of the form shown in Figure A.6.

Consider a (mathematical) cylindrical surface of radius R concentric with the antenna vertical axis. The zone that is illuminated by one or more scans of the beam has a height Y given by the equation

$$Y = 28 + R \tan 2^\circ = 28 + 0.035R \tag{10}$$

where 2° is the angular variation between 0.5° and 2.5° in the worst case scan sequence. The circumference of such a cylinder is $2\pi R$ and the total area A is

$$A = 2\pi RY = 176R + 0.22R^2 \quad . \tag{11}$$

The average power radiated is equal to or less than 1 kW; therefore, the average power density over the cylindrical surface cannot exceed the total power divided by the area.

At the mid-height of this cylinder, the local power density will exceed the average value by a factor of 2 if the vertical power density distribution has either the sinusoidal form shown as a solid line or a triangular form shown in dashed lines. Introducing this factor, a factor of 10^6 to convert from kilowatts to milliwatts, and dividing by 929 to convert from square feet to square centimeters leads to the expression

$$U_6 = 1 \times 10^6 \times 2 / (929)\,(176R + 0.22R^2) = 9800 / (R^2 + 800R) \text{ mW/cm}^2 \tag{12}$$

which is accurate for values of R up to about 640 ft where the beam begins to spread.

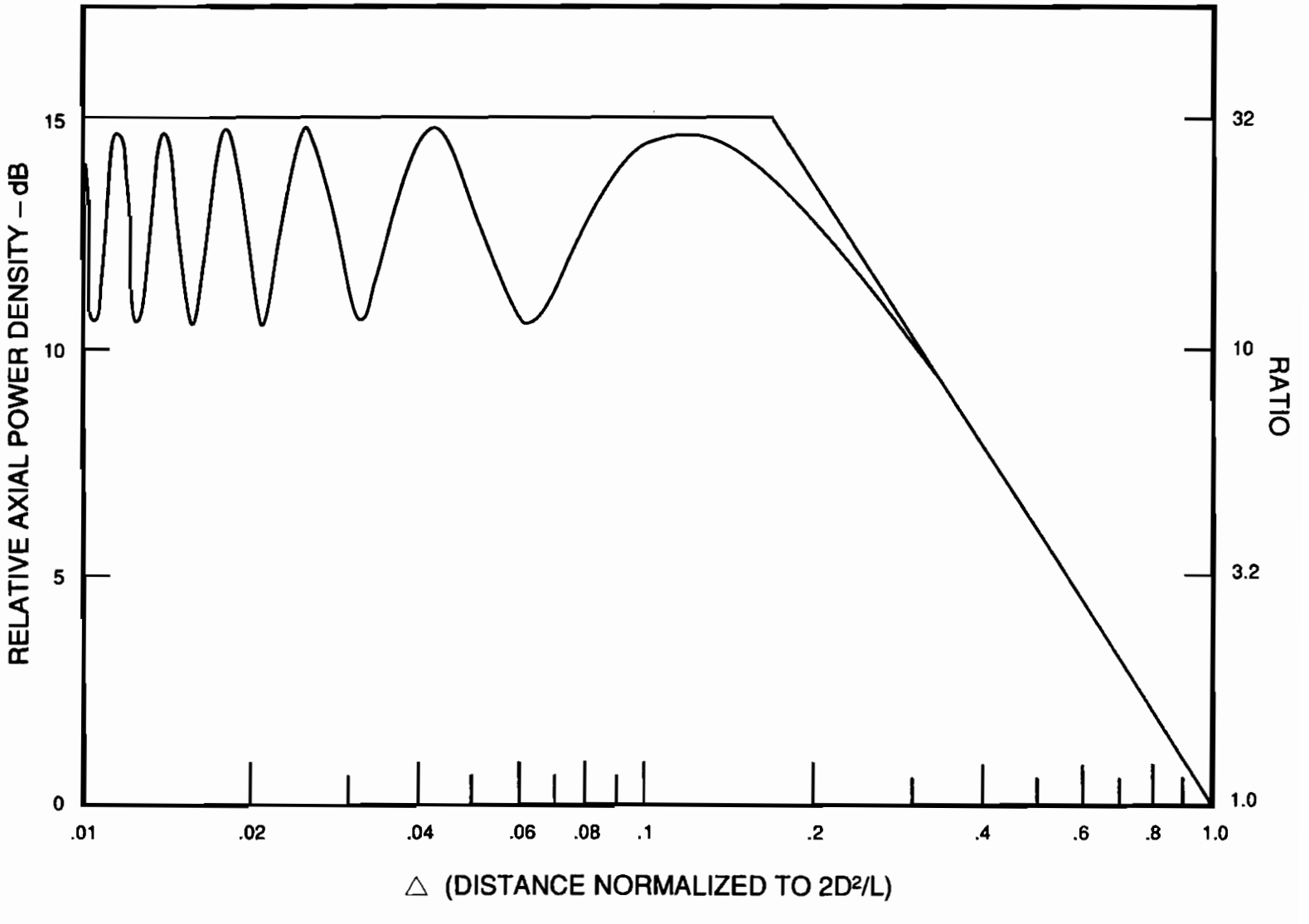

FIGURE A.4 Relative Power Densities on Axis of Main Beam

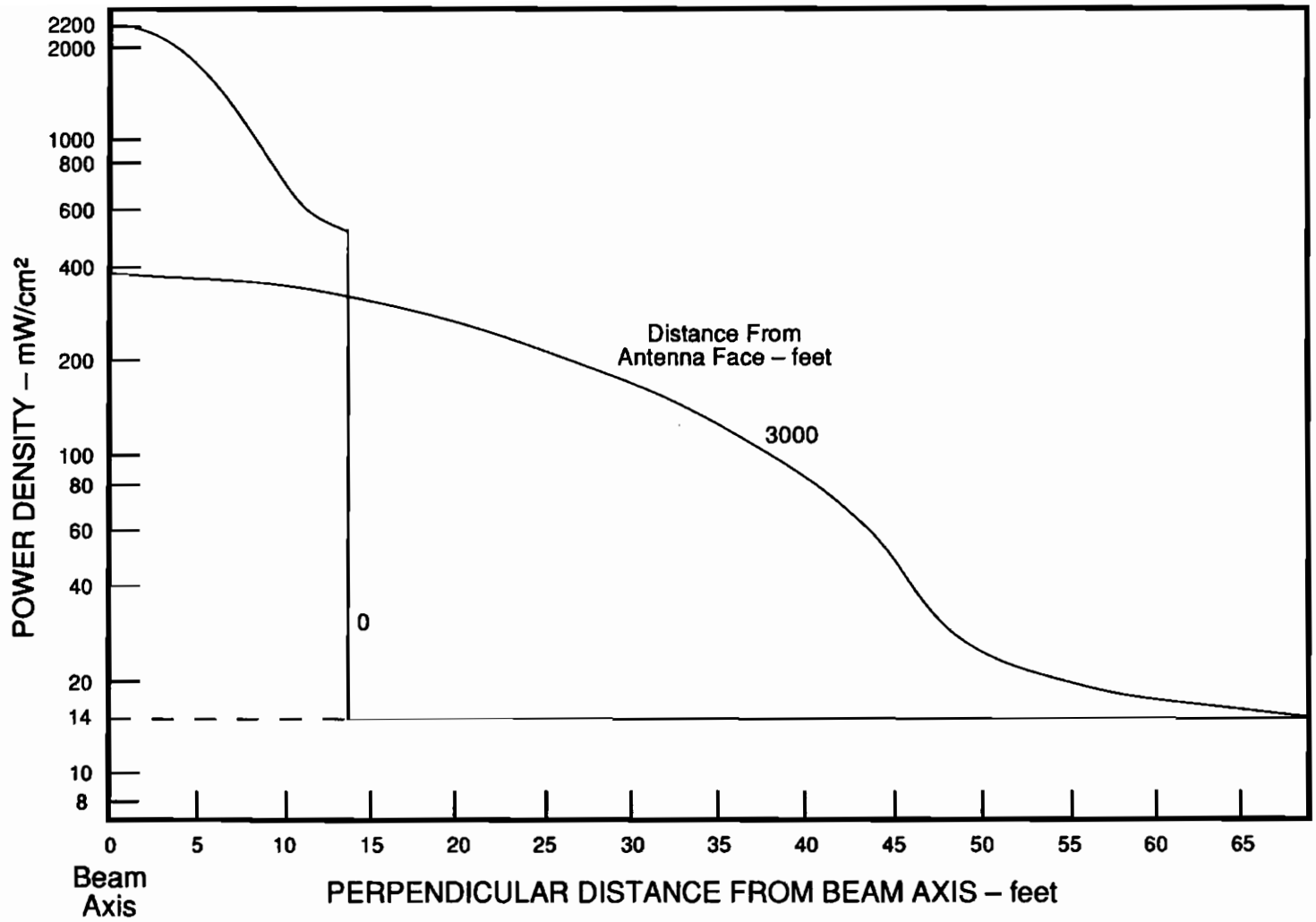

FIGURE A.5 Off-Axis Power Densities in Near Field of WSR-88D Antenna

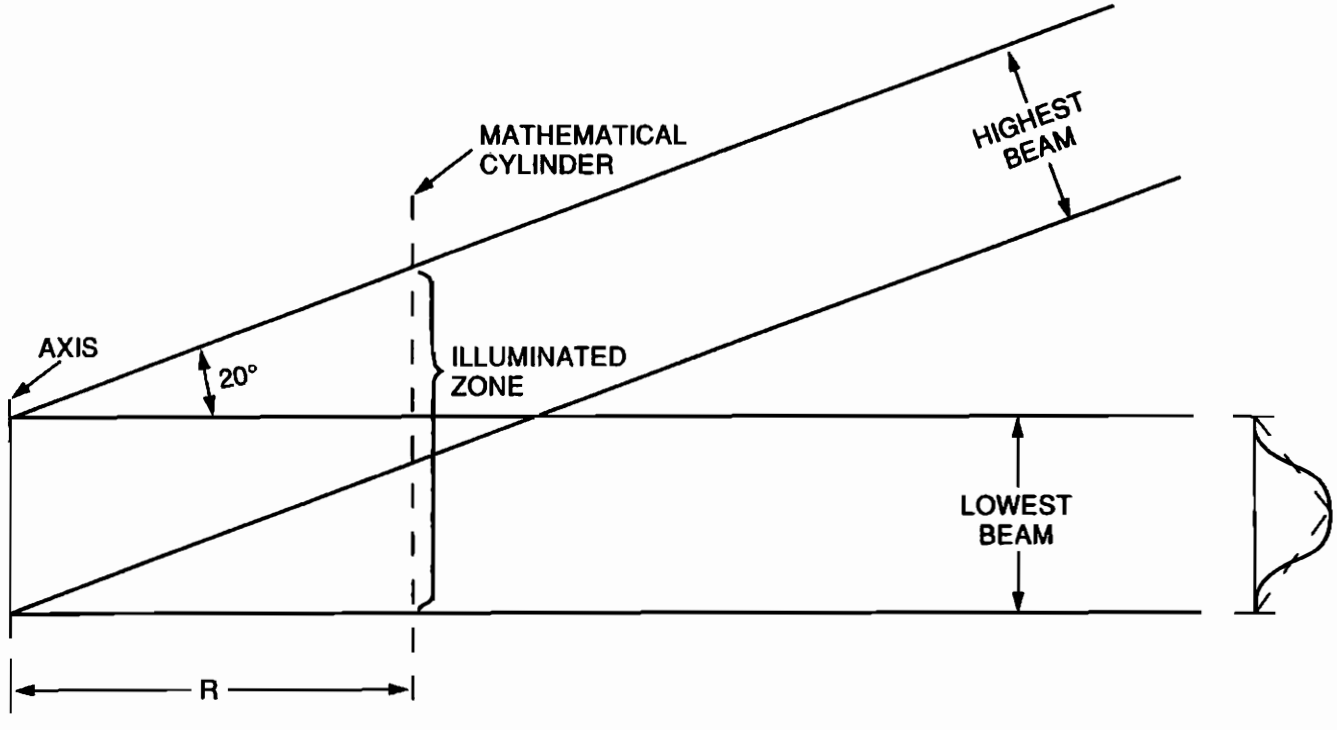

FIGURE A.6 Effect of Scanning By Near Field Column

Electromagnetic waves such as those generated by the WSR-88D radar are characterized by electric and magnetic fields, both of which are perpendicular to the direction of propagation. Of these, the electric field is of principal interest. Under all conditions relevant to the present calculations, the electric field strength (or intensity) E is related to the local power density U by the equation

$$E = (3{,}770\ U)^{1/2} \qquad (13)$$

where E is measured in volts per meter (V/m) and U is given in mW/cm^2. The relationship is used only for pulse values of E and U.

A.5. EFFECT OF FOLIAGE AND SCATTERING

Microwave energy is absorbed and scattered by trees and underbrush. The effect of foliage on microwave propagation has been studied extensively (Trevor, 1940; Head, 1960; Doeppner et al., 1972; Tamir, 1977; Nelson, 1980). Waves that are forced to propagate directly through a forest are attenuated in an exponential manner. For typical woods the rate is about 0.15 dB/ft at WSR-88D frequencies; that is, the signal loses half its power (3 dB) in traveling a distance of about 20 ft. It is reduced to one-tenth its original strength in 60 ft and one-one hundredth its original strength in 120 ft.

Over extended distances, waves find easier paths that curve around or skim over the tops of the trees. Such paths usually reduce the power to a value no larger than one-one hundredth of that which would exist over a direct free-space path. Waves that graze the treetops over a long distance behave somewhat like those that are guided over the surface of an imperfectly conducting earth, and the power density variation with distance includes a term of the form $1/R^4$.

When vegetation provides shielding between the radar antenna and the location of interest, the levels of RFR (both peak and average power densities) are likely to be reduced by a factor ranging from 10 to 100. That factor is not discussed here; rather, our purpose is to provide extremely conservative (i.e., overstated) estimates of RFR.

From some locations of interest, the WSR-88D antenna will not be visible. Under these circumstances it might be thought that no microwave power would reach such locations. Although this ideal is closely approached, a small residue of power does propagate to such locations by diffraction around the edges of the land masses or buildings that block the view, and by scattering from trees, fences, and other objects that are struck by the main beam or its principal sidelobes.

It is impractical to make precise calculations of the RFR that results from such effects. A conservative upper bound is obtained by the principles of diffraction theory (ITT, 1977), which indicate that neither the peak nor the average power density within such regions will exceed one tenth of that found in unshadowed regions at an equal distance from the radar.

Because the earth is nearly spherical, a horizontal beam of radiation is above the earth at all points. However, such a beam may be bent toward the earth by refraction in the atmosphere, which is caused by the decrease in atmospheric density associated with increase in altitude. The effect of such atmospheric refraction can be accounted for by using a fictitious earth radius that is 4/3 times the true radius. The result of such calculations leads to a simple formula for the height of a beam launched horizontally from a particular point on the surface of the earth

$$h = R^2 / 2 \quad (14)$$

where R is the horizontal distance in (statute) miles and h is the beam elevation in feet. Thus, at a distance of 10 miles the beam clearance above the ground is increased by 50 ft as a result of earth curvature. This effect reduces the intensity of RFR at locations distant from the radar site.

A.6. RESULTS

The preceding subsections have developed a set of formulas and graphs for calculating electric field strengths and power densities at various points relative to WSR-88D radars. To facilitate calculations of RFR levels at specific locations of interest, these relationships have been used to prepare Figures A.7 and A.8, which contain all the accumulated information.

A.6.1 Pulse Power Densities and Electric Field Intensities

Figure A.7 shows maximum pulse power densities and electric field intensities for distances and beam offset distances of interest. Maximum pulse power density and electric field intensity are independent of duty cycle and scan pattern, varying only with distance and beam offset. The two straight line segments near the top of the graph represent an envelope of maximum values of pulse power densities that could be encountered by airborne objects. The horizontal line segment corresponds to points that are nearly level with the antenna in the near-field zone. The sloping segment corresponds to the center of the beam in the far-field region, where the power density decreases steadily with increasing distance. These lines represent an upper bound on possible pulse power and electric field intensities. The intervening curves were obtained by graphical computation from Figures A.1 and A.5. The process was facilitated by use of Table A.2. The offset distances were arbitrarily chosen as convenient for this calculation and for subsequent use. The electric field intensities shown in the right-hand margin were obtained by use of Eq. (13).

A.6.2 Average Power Densities

Figure A.8, which is similar to Figure A.7, was derived in the same general way; however, it differs in several important respects. In particular, the power densities are much lower, an electric field scale is inappropriate, and the distance scale is extended down to 20 ft, approximately the surface of the radome.

Values in the right half of the figure were obtained from Eqs. (4) and (6), together with values of K_2 derived from Figure A.3 and listed in Table A.2. The maximum value displayed is 0.6 mW/cm^2; it occurs at R = 20 and h = 0, just outside the mid-height of the radome. Values along the R = 20 axis were obtained from Eq. (4) by substitution of h for R. In the range h <R, the values were obtained by using $h^2 + R^2$ in the denominator. Smooth curves were used to connect these points to those calculated from far-field relationships.

Figures A.7 and A.8 are based on a continuous scan with a minimum elevation angle of 0.5° and the scan sequence described in Section V.B.2 which has a revisit time of 10 minutes. The restriction to 0.5° elevation angle may be removed by referring the beam offset distance h to the center of the lowest beam. With this interpretation, Figures A.7 and A.8 apply equally to any small elevation angle, positive or negative.

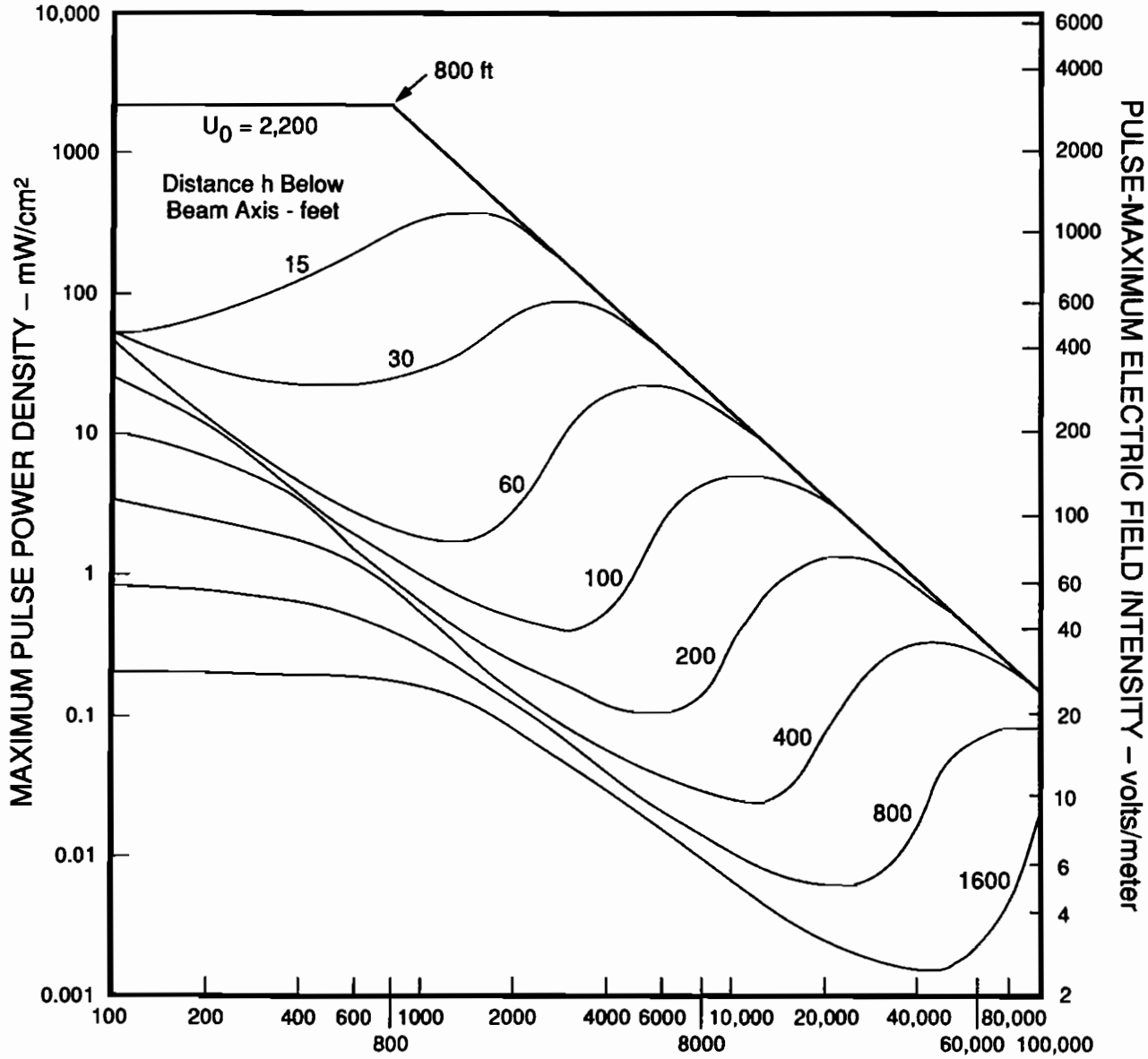

FIGURE A.7 Maximum Pulse Power Densities and Electric Field Intensities Near a WSR-88D Radar

Note: Pulse power designates the maximum rms value of the pulse, when present

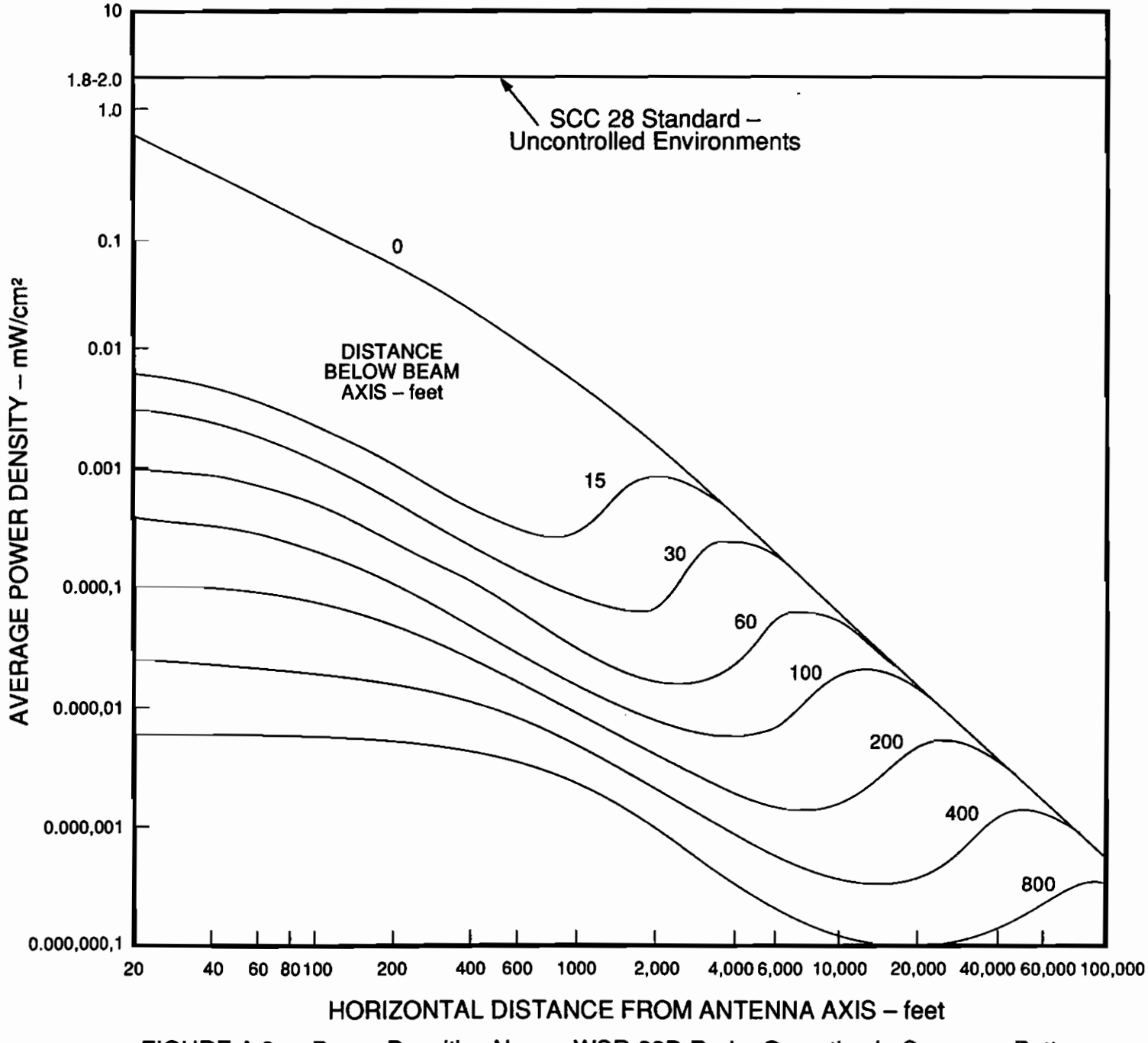

FIGURE A.8 Power Densities Near a WSR-88D Radar Operating in Coverage Pattern 31 (averaged over 6 min.)

Table A.2
DISTANCES D FOR VARIOUS ANGLES AND OFFSET DISTANCES

Offset Distance (feet)	Angle Z (degrees) 0.5°	1°	2°	4°
15	1,718	859	429	215
30	3,437	1,718	859	429
60	6,874	3,437	1,718	859
100	11,460	5,730	2,865	1,432
200	22,920	11,460	5,730	2,865
400	45,840	22,920	11,460	5,730
800	91,680	45,840	22,920	11,460
1,600	---	91,680	45,840	22,920
K_1	0.50	0.050	0.0021	0.0009
K_2	0.50	0.037	0.0078	0.0036

Values of average power density are proportional to the duty cycle and also depend on the scan pattern. Thus, Figure A.8 is specific to the designated scan sequence. However, no other proposed scan sequence will increase these values.

A.7. OTHER SCAN MODES

In most cases the WSR-88D tower will be located on fairly level ground, and best results will be obtained with the normal scan sequence in which the minimum beam elevation is +0.5°. However, a few systems will be in mountainous regions, where mountain tops will be the preferred sites because they will provide the best coverage. For such sites, JSPO has authorized the use of modified scan sequences in which the minimum beam elevation will be as low as -0.5°. Data presented in Figure A.8 and field measurements show that no personnel hazard will result from such operation.

In all operational modes the WSR-88D antenna will rotate continuously at a speed no less than 0.8 rpm. However, for diagnostic purposes, the beam will occasionally be held stationary for a period not exceeding 5 minutes – the "searchlight mode." The beam elevation to be used in these tests has not been specified, but it is appropriate to assume the worst case of -0.5°.

Under these circumstances, the effective power density on the beam axis for distances out to 800 ft, averaged over 6 minutes, will be

$$U_n = 0.0021\ (5/6)\ U_o = 3.85\ \text{mW/cm}^2 \quad . \qquad (14)$$

Here, U_o = 2200 and the factor 0.0021 is the duty cycle, which converts peak to short-term average power. In the WSR-88D frequency band of 2700 to 3000 MHz, the SCC 28 (1991) exposure guideline for controlled environments is 9 to 10 mW/cm^2, averaged over a 6-minute period. Thus, the average power density of WSR-88D emissions during operation in searchlight mode, averaged over 6 minutes, will not exceed the SCC 28 (1991) guideline for human

exposure in controlled environments. Beyond 800 feet the power density is still smaller, falling off inversely with the square of distance according to the expression

$$U_f = 0.0021\ (5/6)\ U_1 = 2.52 \times 10^6/R^2 \quad . \tag{15}$$

The 1991 SCC 28 guideline for human exposure to 2700 to 3000 MHz RFR in uncontrolled environments is 1.8 to 2.0 mW/cm^2, averaged over 30 minutes. To account for the 30-minute averaging period, Eq. (15) must be modified to

$$U_u = 0.0021\ (5/30)\ U_0 = 0.77\ \text{mW/cm}^2 \tag{16}$$

which is considerably below the SCC 28 (1991) exposure guideline. However, if multiple 5-minute periods of searchlight-mode operation were conducted during a 30-minute interval, the SCC 28 (1991) guideline for uncontrolled environments could be exceeded. To prevent violation of the SCC 28 (1991) guideline, each time the searchlight mode is to be used, a safety element will be prepared as part of the test plan. When the safety element is implemented, significant impacts will be very unlikely.

A.8. VALIDATION

The validity of the methods used to derive the results presented in Figures A.7 and A.8 has been confirmed by comparing the results of similar calculations with measurements made on August 16-18, 1989, at the prototype WSR-88D at the OSF. Measurement locations were selected on the basis of field intensity calculations and distances from the radar.

The radar operated at a frequency of 2995 MHz and a peak power level of 712 kW. The antenna gain was not given but was assumed to be 45.5 dB, as in Table V.1. The line losses were not given, but it is reasonable to assume that they reduced the actual radiation power to about 475 kW. Pulse widths of 1.5 and 4.7 μs were used, in combination with several pulse repetition frequencies. The combination of 4.7 μs and 446 pps, equivalent to a duty cycle of 0.21%, gave the largest average power densities.

Table A.3 sets forth the peak field intensity and (short-term) average power densities on the center of a horizontal stationary main beam as a function of distance (Air Force Communications Command, 1989).

The calculated values of average power were taken from Eqs. (14) and (15), omitting the factor (5/6) because time-averaging was not involved. The calculated values of peak field intensities were obtained from Eqs. (2), (9), and (13). The calculated values of power density and electric field are somewhat higher than the measured values except at the two closest locations.

Measurements were also made with the antenna rotating at a constant speed of 1 rpm at an elevation angle of 0°. Table A.4 presents the long-term time-averaged results of these measurements. At all distances, the measured values are substantially less than the calculated values; that finding is expected as the formulas used to calculate power density give conservative (worst-case) estimates.

Table A.3
MAIN BEAM MEASUREMENTS SUMMARY

Location Number	Distance (feet)	Average Power Density (mW/cm^2)		Peak Field Intensity (V/m)	
		Measured	Calculated	Measured	Calculated
1	1470	0.71	1.40	1126	1585
2	665	2.81	4.62	2246	2880
3	450	3.30	4.62	2435	2880
4	227	5.31	4.62	3090	2880
5	85	5.59	4.62	3170	2880

Source: U.S. Air Force Communications Command, 1989; SRI International

Note: For location number 1, average power density is calculated using the formula $U = 0.0021 \times 1.44 \times 10^9 \div (1,470)^2$ peak field intensity is calculated using the formula: $E = [1.44 \times 10^9 \times 3,770 \div (1,470)^2]^{1/2}$. For all other near-field locations the average power density is calculated using the formula: $U = 0.0021 \times 2,200$; the peak field intensity is calculated using the formula $E = (2,200 \times 3,770)^{1/2}$.

Table A.4
COMPARISON OF MEASURED AND CALCULATED LONG-TERM TIME-AVERAGED POWER DENSITIES (mW/cm^2)

Location Number	Distance (feet)	Measured	Calculated
1	1470	0.00085	0.0039
2	665	0.0086	0.018
3	450	0.0019	0.027
4	227	0.013	0.054
5	85	0.026	0.14

Source: U.S. Air Force Communications Command, 1989; SRI International

Note: For location number 1, the long-term, time-averaged power density is calculated using the formula: $U = 0.0021 \times 1.44 \times 10^9 \div (360 \times 1,470^2)$. For all other locations, the formula is $U = 2 \times 10^6 \div (2\pi R \times 28 \times 929)$, where the initial factor of 2 accounts for power concentration near the center of the main beam.

APPENDIX B

HUMAN HEALTH EFFECTS OF RFR

B.1. Introduction

This analysis of the potential bioeffects of RFR and their relationship to human health is based on a review of research papers selected from the many thousands of accounts published in scientific journals through about mid-1991. The papers reviewed were selected based on their relevance to WSR-88D RFR and the quality and originality of their findings. The papers discussed are grouped under a set of RFR-bioeffects topics selected to provide representative coverage of each topic. We describe differences in the findings of the papers within each topic, and, when possible, assess the quality of the research. We assume that most of the papers selected underwent peer review before publication. With a few exceptions, presentations at scientific symposia or abstracts thereof have been excluded from consideration because more complete and peer-reviewed accounts of such studies are likely to appear subsequently.

Some of the analyses and critiques here of papers published before 1987 have been derived from a general review entitled: *Critique of the Literature on Bioeffects of Radiofrequency Radiation: A Comprehensive Review Pertinent to Air Force Operations* (Heynick, 1987), that SRI International prepared for the U.S. Air Force. That review covered more than 500 detailed reviews and analyses of research projects. Almost all of the papers selected for analysis had been published in scientific articles, presumably after peer review. Other general reviews of the literature on RFR bioeffects published from time to time were also investigated, among them an EPA report (Elder and Cahill, 1984), intended to serve as the primary reference for development of U.S. guidelines for general public exposure to RFR which were never promulgated. The conclusions in this SEA regarding possible effects of exposure of people to RFR were reached independently.

The results of our research review are summarized in Section V.C.2, while our findings about RFR bioeffects are presented in Section V.C.3. In Section V.A.2, we discuss the challenges in assessing scientific information and risk, while Section IV provides extensive background information about the origins of the electromagnetic environment, the electromagnetic spectrum and its use, the ambient electromagnetic conditions, and nonionizing vs. ionizing radiation.

B.1.2 RFR Safety Standards

Relevant RFR safety standards are described in Section V.C.4. In this appendix subsection, we replicate the Section V.C.4 discussion of the recent IEEE (C95.1 - 1991) guidelines and supplement it with two graphs (Figures B.1 and B.2).

In 1982, American National Standards Institute (ANSI) Subcommittee C95.IV adopted a frequency-dependent standard for both occupational and public exposure to RFR (ANSI, 1982). That standard was based on a maximum whole-body SAR of 4 W/kg reduced by a safety factor

of 10, to 0.4 W/kg. It covered the frequency range from 300 kHz to 100 GHz. Those limits were not to be exceeded for exposures averaged over any 6-minute period. In the 1982 ANSI standard, the incident power density limit for the 2.7 to 3.0 GHz range of the WSR-88D was 5 mW/cm^2.

In 1988, the functions of ANSI Subcommittee C95.IV were transferred to Subcommittee IV of SCC 28, a new body under the jurisdiction of the IEEE. The subcommittee selected and analyzed the important research papers in an updated data base of the RFR-bioeffects literature and prepared a revision of the 1982 ANSI standard. That revision, the IEEE (1991) guidelines, has recently been approved by the IEEE.

The IEEE (1991) guidelines cover the frequency range from 3 kHz to 300 GHz and separately specify the maximum allowable RFR exposure in "uncontrolled environments" (accessible by the general population) and "controlled environments" (such as occupational exposure). Graphs of the guidelines for uncontrolled and controlled environments are displayed in Figures B.1 and B.2, respectively. In the range from 300 MHz to 1.5 GHz, the new limits for uncontrolled environments have a safety reduction factor of 50 instead of 10, and are averaged over any 30-minute period instead of over 6 minutes. In the range from 1.5 to 3.0 GHz, the new limits increase in accordance with the formula f/1500 (with the frequency f in MHz), whereas the limit was fixed at 5.0 mW/cm^2 in the 1982 ANSI standard. Thus, in the WSR-88D frequency range 2.7 to 3.0 GHz, the new limits on average power density for uncontrolled environments are 1.8 to 2.0 mW/cm^2 instead of 5 mW/cm^2. The new IEEE (1991) guideline limits for controlled environments are 9.0 to 10 mW/cm^2 averaged over any 6-minute period.

Also included in the IEEE (1991) guidelines are maximum allowable values for RF current flow induced within the feet of a person immersed in an RFR field or by the person's contact with an inanimate object (e.g., a fence or vehicle) electrically charged by immersion in an RFR field. Such limits are applicable only within the frequency range from 3 kHz to 100 MHz (where such effects can occur), and thus are not relevant to WSR-88D.

B.2. Interactions of RFR with Biological Entities

B.2.1 Thermal Interactions and SARs

The relative magnetic permeability of most organic substances is about unity. Therefore, thermal interactions of RFR with a biological body are dependent on the complex dielectric and thermal properties of the body's constituents and their distribution within the body, as well as on the RFR's characteristics (frequency, power density, polarization).

Because the index of refraction of any material is related to its dielectric constant, RFR is reflected and refracted at boundaries between regions of differing dielectric properties, such as at the air-surface interface of a body, for the same physical reasons as those that apply to light incident at an air-glass interface. This is also true at internal boundaries between constituents having different dielectric properties, thereby affecting the variation of electric field with internal location. Figure B.3 is a graph of the fraction of power transmitted (not reflected) at air-fat, fat-muscle, and air-muscle interfaces versus RFR frequency. At 3.0 GHz, for example, about 44% of the incident power density enters the body, with the remaining 56% being reflected at the surface.

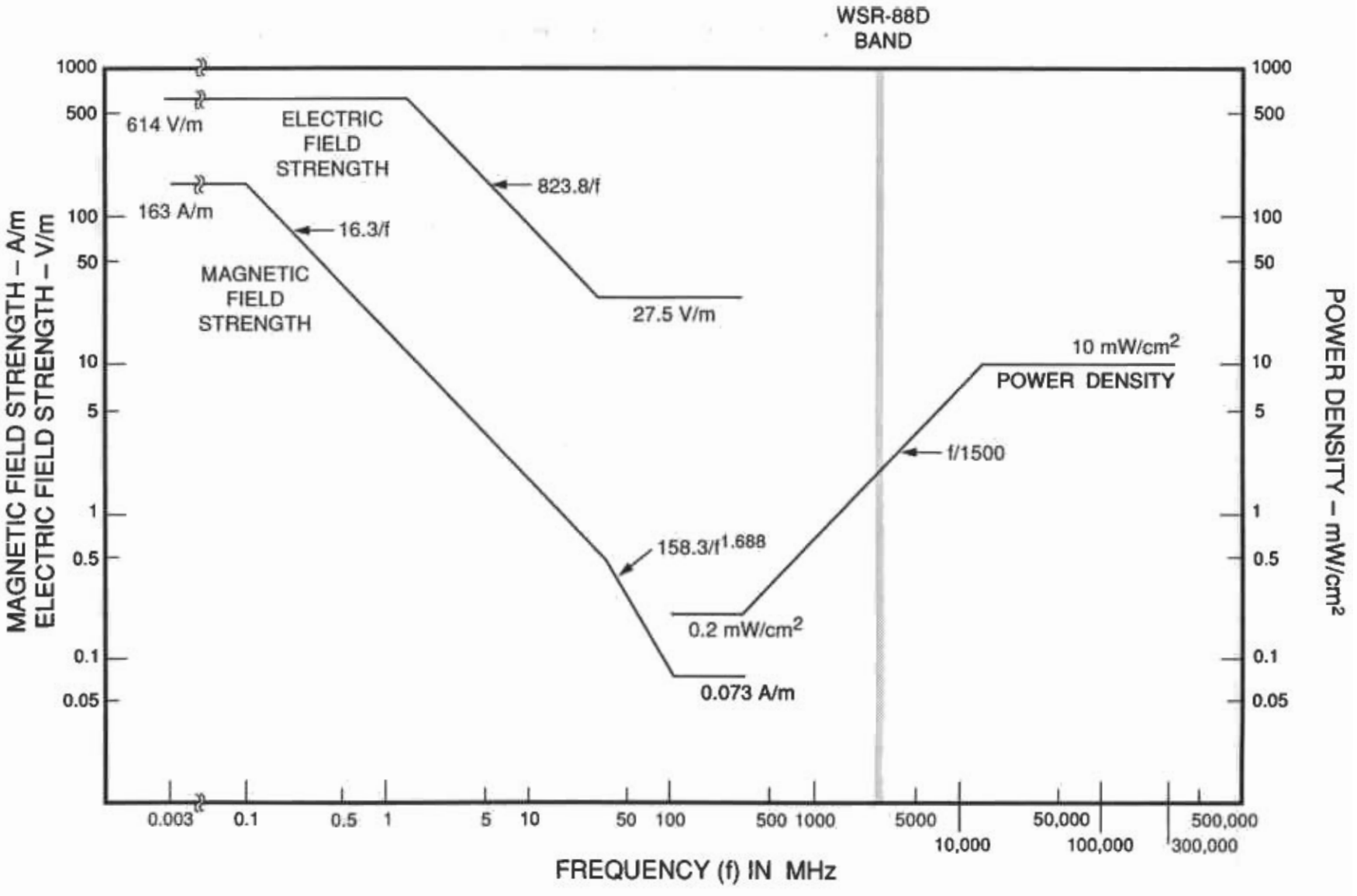

FIGURE B.1 Maximum Permissible Exposure for Uncontrolled Environments

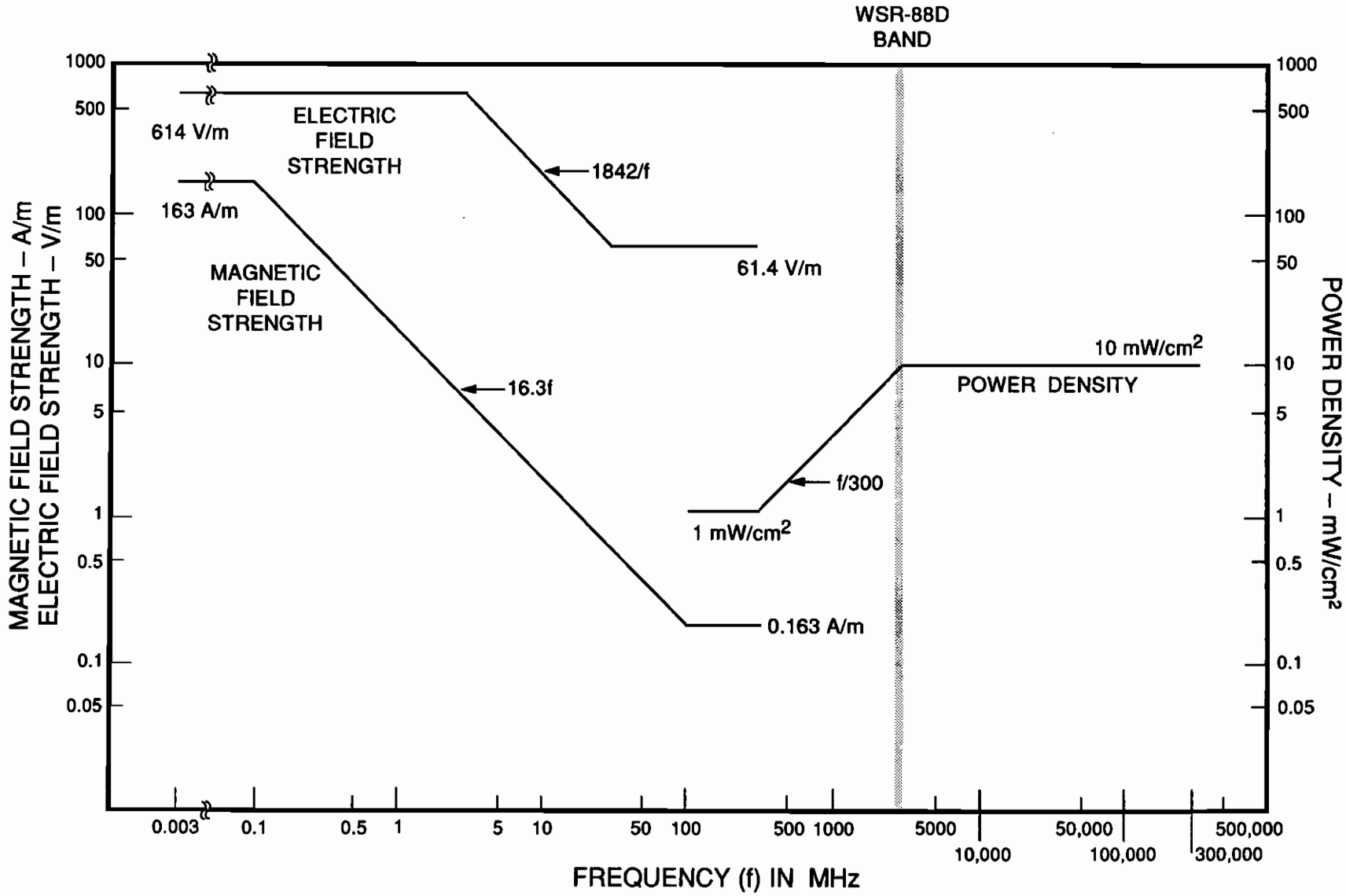

FIGURE B.2 Maximum Permissible Exposure for Controlled Environments

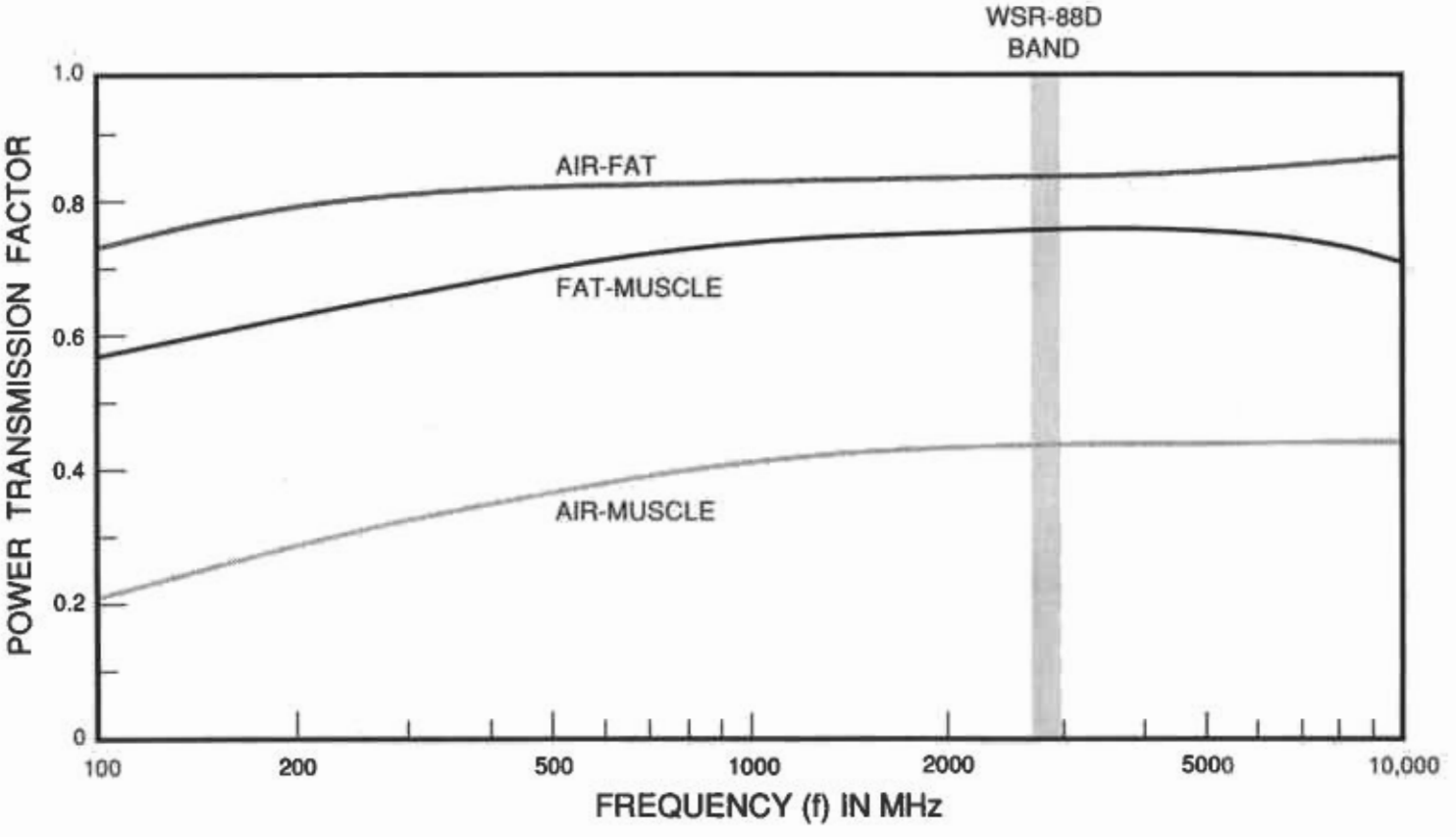

FIGURE B.3 Power Transmission Factors for Air, Fat, and Muscle Interfaces

The fraction of incident power density that enters a body undergoes progressive attenuation with depth because of energy absorption. The term "penetration depth" is usually used to quantify such attenuation. For homogeneous specimens and RFR incidence perpendicular to the surface, penetration depth is defined as the distance at which power density is decreased by absorption to about 14% of its value just within the body's surface. Graphs of penetration depth versus frequency for muscle, blood, and fat are shown in Figure B.4. At 3.0 GHz, the penetration depths for muscle, blood, and other high-water tissues are about 1.7 cm (0.66 in.); for fat, the value is about 7 cm (2.8 in.). Not shown in the figure is that at approximately 30 GHz and higher, penetration is largely confined to the outer layers of the skin (much like for sunlight).

In the RFR-bioeffects literature, the energy absorbed by a body from an incident electromagnetic field is usually quantified by the SAR. The local SAR at any site within a body depends on the characteristics of the incident RFR (carrier frequency, modulation, amplitudes and directions of its components) and on the properties of the body and location of the site. For bodies of complex shape and large internal spatial variations in properties, local SAR values are difficult to determine by experiment or calculation. Instead, the whole-body SAR, representing the spatial average SAR for the body, is often used because it can be determined in the absence of information about internal variations in local SARs.

Researchers have calculated whole-body SARs for models of relatively simple geometry such as spheroids, ellipsoids, and cylinders that have weights and dimensions approximately representative of various species, including humans. Others have experimentally verified such calculations by exposing physical models in various orientations to linearly polarized plane-wave RFR and determining distributions of heat produced therein.

For exposure of any given model to linearly polarized plane-wave RFR, the largest value of whole-body SAR occurs when the longest dimension of that model is parallel to the electric component of the RFR, called the E-orientation, and when the wavelength of the RFR is about 2.5 times the longest dimension of that model (or conversely, the longest dimension is 0.4 of a wavelength). The adjective "resonant" is used for that wavelength or for its corresponding frequency; at resonance, the model absorbs RFR energy much like a lossy half-wave-dipole antenna. Exposure at other orientations yields lower SARs.

Many of the important results of such theoretical and experimental investigations have been presented in handbooks issued by the U.S. Air Force. The last handbook (Durney, 1986) summarizes the data in previous editions and contains other pertinent information as well. Of particular interest are the plots of calculated whole-body SAR versus frequency for prolate-spheroidal models of an "average" man, woman, and 5-year-old child for exposure to 1 mW/cm^2 in three orientations (Durney, 1986), reproduced as Figures B.5, B.6, and B.7. Analogous plots for a prolate-spheroidal model of a medium rat are shown in Figure B.8 for comparison. Those plots all display the resonances noted for the E-orientation, with sharp reductions in SAR below each resonant frequency and slower decreases above the resonant frequency.

Specifically, the resonant frequency for the average man, taken to be 5 ft 9 in. tall and weighing about 154 lb, is about 70 MHz (when insulated from ground). At this frequency, the whole-body SAR is about 0.2 W/kg for an incident plane-wave power density of 1 mW/cm^2. This SAR is about one-sixth of his resting metabolic rate or one-twentieth to one-ninetieth of his

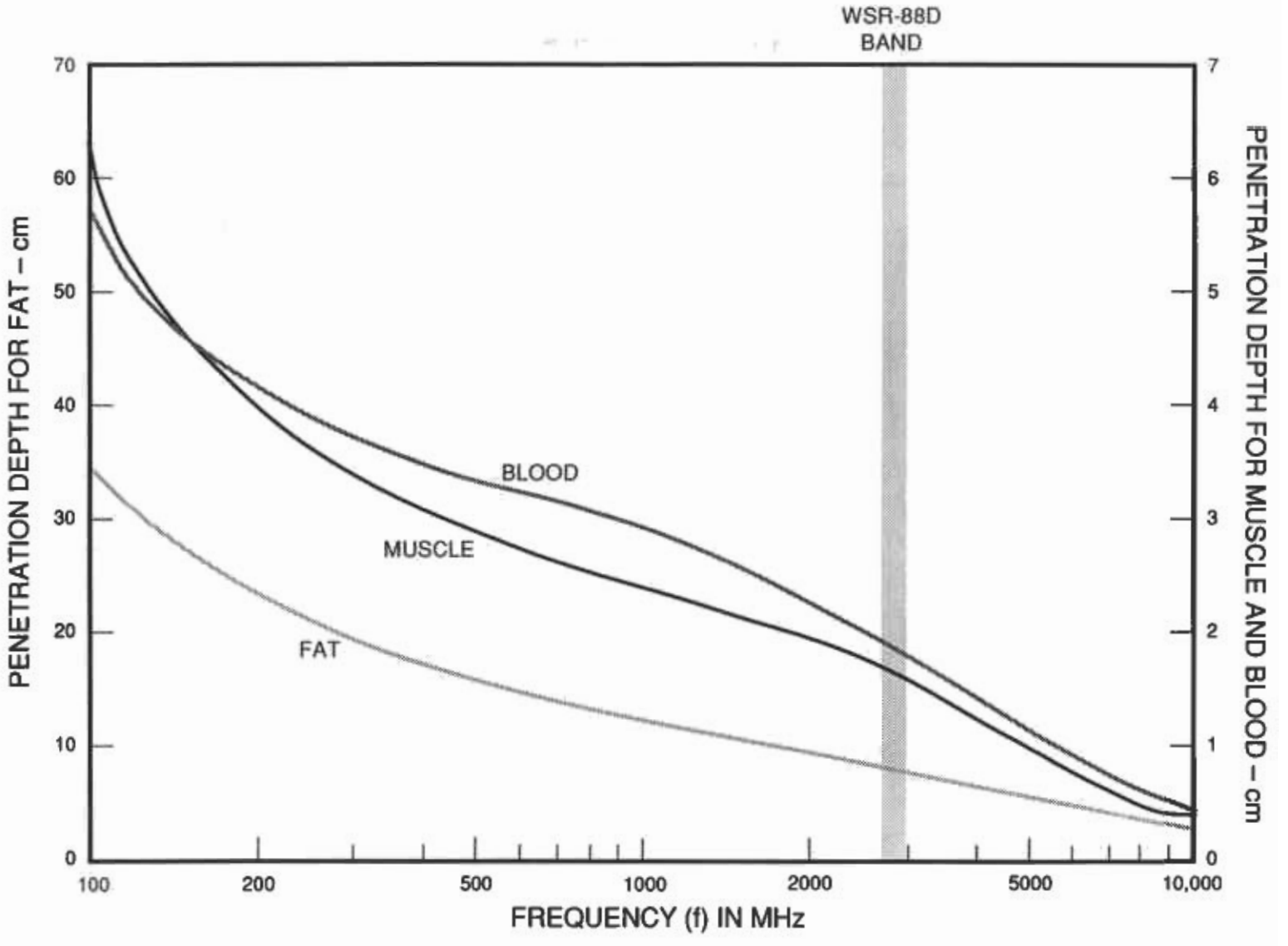

FIGURE B.4 Penetration Depth Versus Frequency for Muscle, Blood, and Fat

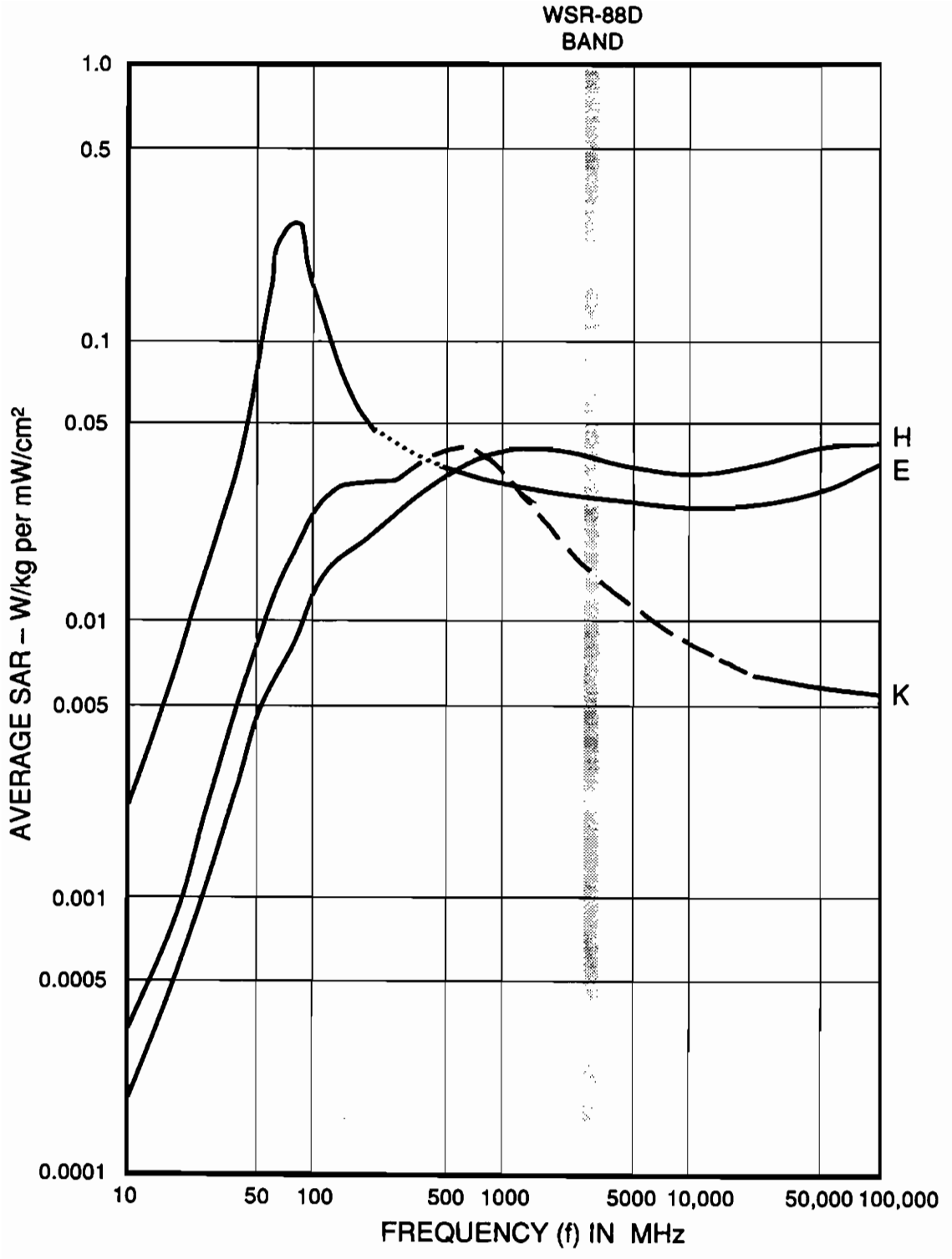

FIGURE B.5 Calculated plane-wave average SAR versus frequency for an average man in the E, H, K orientations. (Prolate spheroidal model, three polarizations; a = 0.875 m, b = 0.138 m, V = 0.07 m^3.)

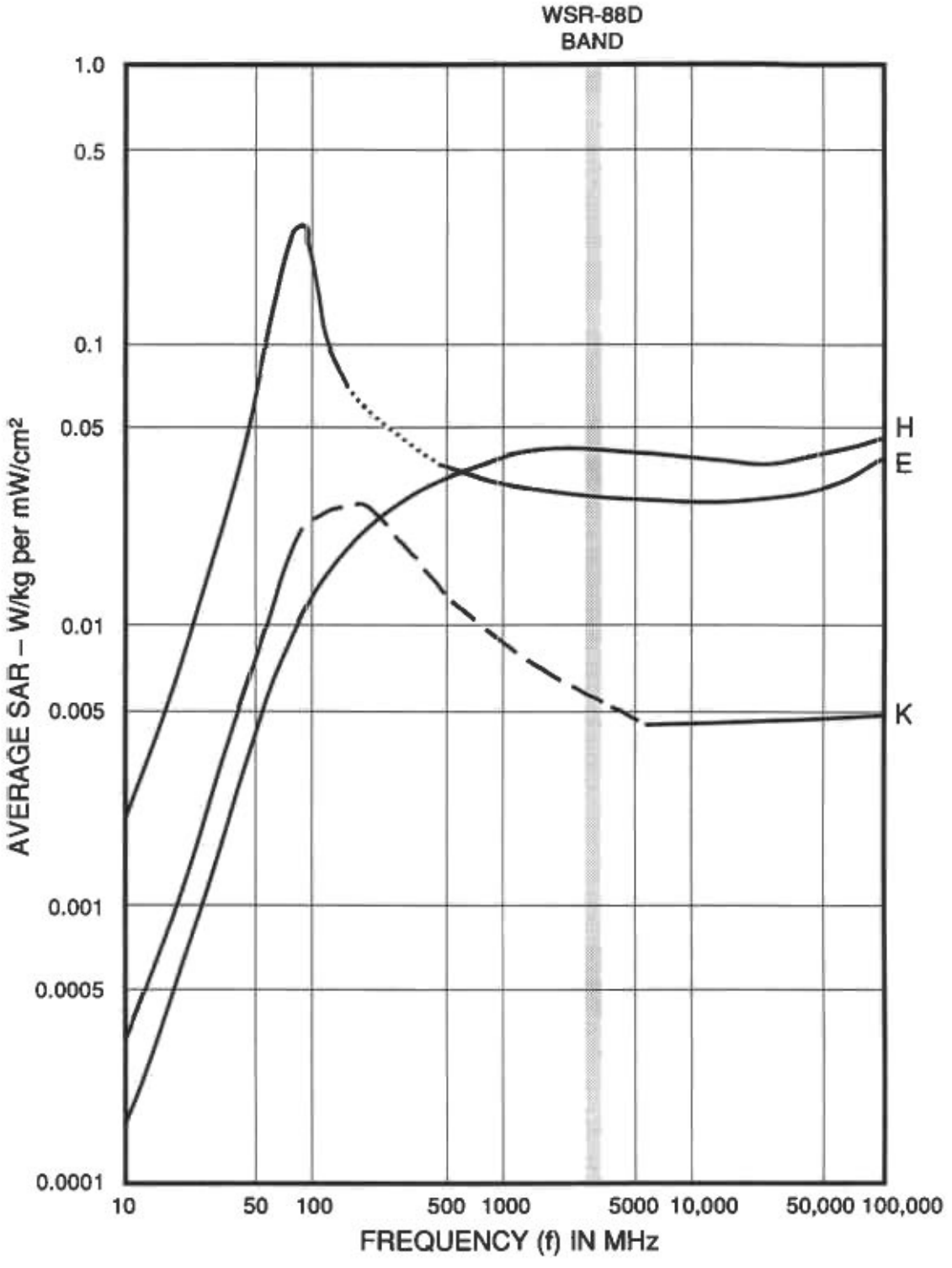

FIGURE B.6 Calculated plane-wave average SAR versus frequency for an average woman in the E, H, K orientations. (Prolate spheroidal model, three polarizations; a = 0.805 m, b = 0.135 m, V = 0.06114 m^3.)

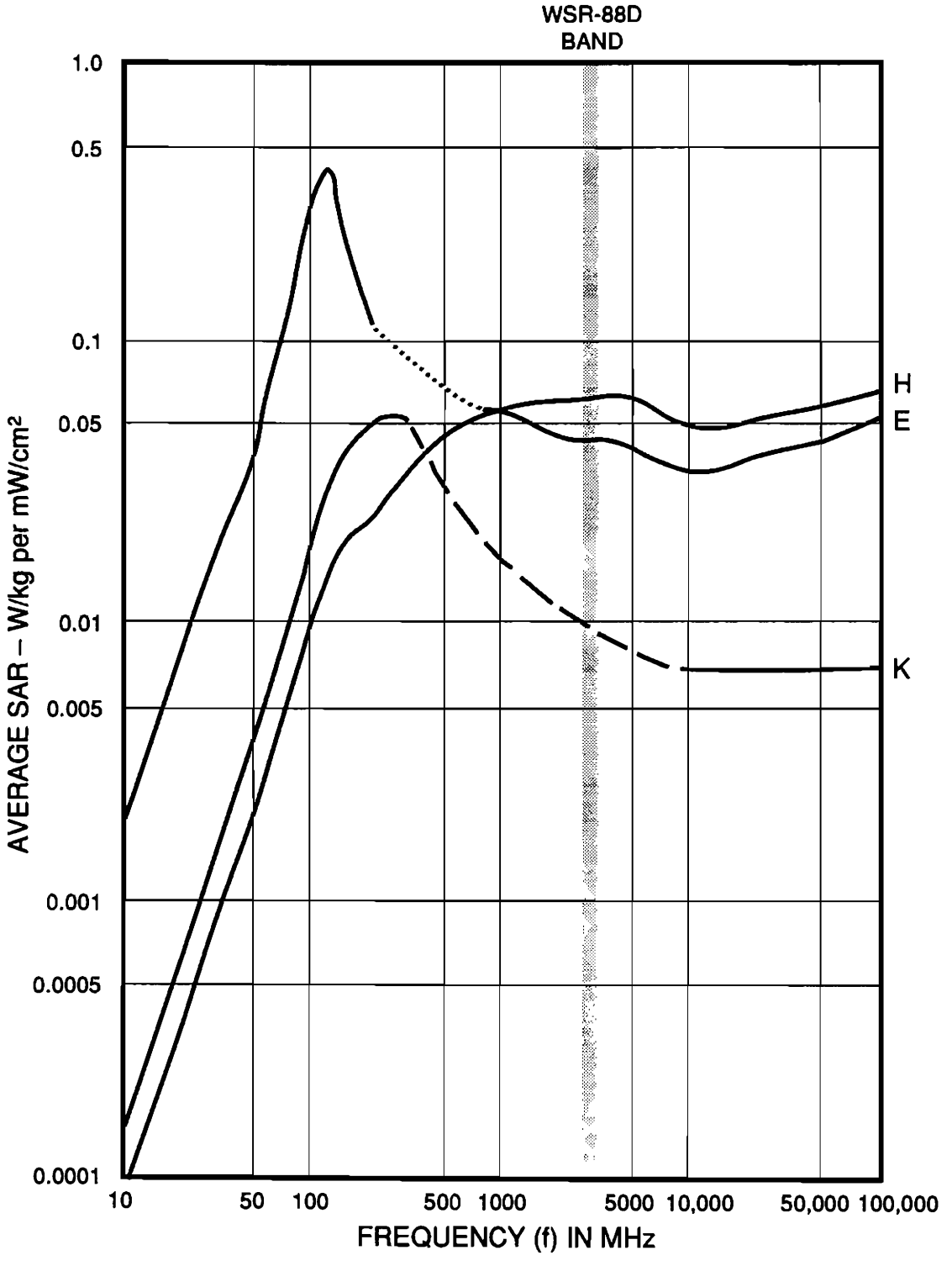

FIGURE B.7 Calculated plane-wave average SAR versus frequency for a prolate spheroidal model of an average 5-year-old child in the E, H, K orientations. (Prolate spheroidal model, three polarizations; a = 0.56 m, b = 0.091 m, V = 0.0195 m^3.)

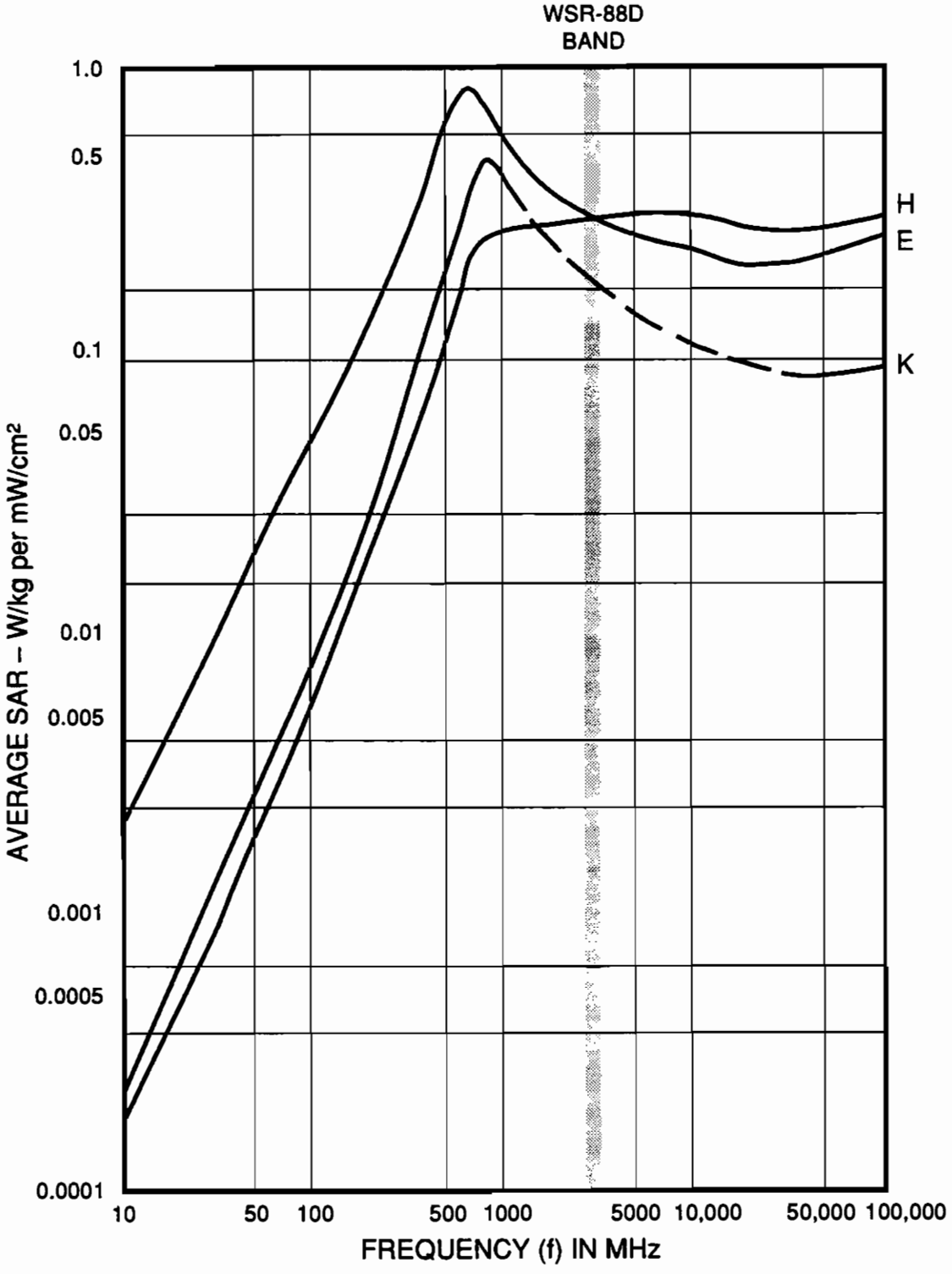

FIGURE B.8 Calculated plane-wave average SAR versus frequency for a medium rat in the E, H, K orientations. (Prolate spheroidal model, three polarizations; a = 0.1 m, b = 0.0276 m, V = 3.2 x 10^{-4} m^3.)

metabolic rate when exercising ranging from walking to sprinting. By calculation, exposure of a man at this SAR (to 1 mW/cm^2) for, say, 1 hour would produce a mean temperature increase of about 0.2°C with no heat-removal mechanisms (conduction, convection, radiation) operating. Actual temperature increases would be smaller with such heat-exchange mechanisms present. In addition, the compensation exercised by the thermoregulatory systems of live mammals may prevent any rises in body temperature.

At WSR-88D frequencies, which are well above the resonant frequency, the whole-body SAR in the E-orientation is only approximately 0.03 W/kg per mW/cm^2 (and about 0.04 W/kg in the H-orientation, in which the long axis of the body is at right angles to the electric component of the RFR). Therefore, a 1-h continuous exposure of the model man to 3 GHz at, say, 1 mW/cm^2 (a level much higher than from WSR-88D) would cause virtually no temperature rise.

Similarly, the resonant frequency for a prolate-spheroidal model of an average woman about 5 ft 3 in. tall (insulated from ground), is about 80 MHz, and her mean SAR is about the same as for the average model man. For the model of a 5-year-old child, the resonant frequency is about 110 MHz, and the resonant SAR is about 0.3 W/kg per 1 mW/cm^2.

By contrast, the resonant frequency for a prolate-spheroidal model of a medium rat is about 650 MHz, and the resonant whole-body SAR is about 0.8 W/kg per mW/cm^2. These values and those for other laboratory animals used in RFR-bioeffects studies are important in assessing the results of such animal studies relative to possible effects in humans.

Calculations indicate that if humans were to stand in bare feet on a wet surface, their resonant frequencies would be approximately halved, but their whole-body SARs (at the lower resonant frequency) would be higher. However, the SARs at the WSR-88D frequencies under such conditions would still be too small to cause any increase in body temperature.

Under some conditions, the SARs of various parts of the body, such as the head and limbs, also require consideration. In an important early study, Shapiro et al. (1971) calculated distributions of fields that would be induced in a multilayered spherical model of a primate head by exposure to 3.0-GHz plane-wave RFR. Their calculations indicated the existence of local internal regions of relatively high fields. Johnson and Guy (1972) obtained experimental results confirming the presence of such regions. Kritikos and Schwan (1975) did similar studies for frequencies in the range of 300 MHz to 12 GHz. In general, the locations of such regions depend on the size of the head, the electromagnetic and thermal properties of its layers, and the frequency of the incident field. Such regions have been dubbed "hot spots," even for combinations of power density and exposure duration that would produce biologically insignificant temperature rises at such spots.

Numerical calculations of internal spatial distributions of SAR have been made for "block" models. In such models, the shape of the body is approximated by an appropriate arrangement of many rectangular cells or blocks of various sizes, with each block assumed to be biologically homogeneous and to have constant internal field over its volume when the model is exposed to RFR. The biological properties ascribed to each block are also selected to approximate those of the tissues in the corresponding location of the body. By spatial averaging over such models, more accurate values of whole-body SAR have been derived than from simpler ones.

Rukspollmuang and Chen (1979), using a block model of an isolated multilayered spherical head, obtained results that were qualitatively similar to those of Kritikos and Schwan (1975).

They then studied, at 918 and 24,500 MHz, a block model with shape and internal structure more closely approximating that of the human head (including eyes, nose, skull bone, and brain), and found that much of the energy would be absorbed within the skull. In addition, frontal exposure of the model to 24,500 MHz would induce fields that are primarily concentrated near the front surface, and therefore energy dissipation within the brain would be relatively low.

Hagmann et al. (1979) calculated SAR distributions in the attached head of a block model of a human, and derived whole-head and whole-body SARs for three orientations of the model relative to the source of RFR. For front-to-back propagation with the long axis of the body parallel to the electric vector, they found a broad head resonance at about 350 MHz, with a whole-head SAR of about 0.12 W/kg per mW/cm^2; the corresponding whole-body SAR is about 0.05 W/kg per mW/cm^2. For propagation in the head-to-toe direction, a sharper head resonance at 375 MHz was obtained, with whole-head and whole-body SARs, respectively, approximately 0.22 and 0.07 W/kg per mW/cm^2.

Results of numerical analyses of whole-body SARs and internal SAR distributions have been subjected to experimental verification. Figurines of humans and animals constructed from synthetic biological materials with electromagnetic characteristics approximating their various biological constituents were exposed to RFR at power densities sufficient to produce accurately measurable temperature increases. Such temperature rises were determined immediately after exposure. An important qualitative result is that at frequencies near resonance, local internal fields for human figurines can be much higher for regions such as the neck and groin than for other locations in the body. Moreover, for nonprimate figurines, the variations of internal field with location are quite different from those for primate figurines, a finding that must be considered in attempting to extrapolate experimental results for laboratory animals to humans, or when comparing experimental results between two (or more) laboratory species.

B.2.2 Nonthermal Interactions and SARs

Under similar exposure conditions, the whole-body SARs obtained with amplitude-modulated (AM) RFR at any given carrier frequency and average power density are the same as those of CW RFR or frequency-modulated (FM-CW) RFR.

In the context of nonthermal interactions, the term "local SAR" denotes the rate of energy absorption at any local site within a biological object and does not necessarily indicate that such absorption occurs as heat. Rather, it is a useful measure of the local field strength resulting from RFR exposure, especially at internal field strengths too low to produce heat at biologically significant rates.

RFR pulses of appropriate characteristics are known to be perceived by some humans as apparent sound (the RFR-auditory effect). Pulsed RFR has also been reported to produce other effects, such as alterations of the blood-brain barrier and behavioral changes. Some researchers, using RFR that was amplitude-modulated at specific frequencies – primarily below about 30 Hz but up to about 400 Hz – have reported biological effects from the amplitude modulation *per se*, notably the calcium-efflux effect. Some researchers regard these reports as evidence of potentially harmful nonthermal RFR bioeffects. There is no experimental evidence that the RFR-auditory effects is harmful to humans or animals. Other researchers were unable to confirm that pulsed RFR at nonthermal time-averaged levels alters the BBB or adversely affects behavior. The calcium efflux effect and its purported mechanisms remain controversial (Myers and Ross,

1981; Albert et al., 1987; Halle, 1988; Sandweiss, 1990; Adair, 1991; Prasad et al., 1991). These topics are discussed more fully later.

B.2.3 RFR and Power Line-Frequency Fields

Nonionizing fields include several kinds of electric and magnetic fields. Such fields occur both naturally, among them the earth's magnetic field and the electric fields in the atmosphere (most prominent during storms), and by electric generation for various uses. Much controversy surrounds reports of the deleterious effects on humans from exposure to the power-line frequency electric and magnetic fields present in homes from the power lines supplying electricity to the house, the fields from operating appliances within the home, and those from any nearby high-voltage power lines. In considering possible bioeffects of the RFR from the WSR-88D radar, it is important to recognize the distinction between fields at power-line frequencies and RFR at the microwave (MW) frequencies such as from the WSR-88D radar.

U.S. power sources operate at 60 Hz. The corresponding wavelength is more than 3000 miles, meaning that people near a power line are in its induction zone, within which terms such as "propagation" and "radiation" do not apply. Rather, the electric and magnetic fields from such a source may induce currents in the body, and the effects of each field should be considered separately.

The WSR-88D radar will operate at frequencies about 50 million times higher than 60 Hz, and the corresponding wavelength will be only about 4 in. Thus, sources operating at such frequencies emit and propagate electromagnetic fields as true radiation, even at short distances. Calculations using the WSR-88D wavelengths and the size of its antenna indicate that people at distances about 800 ft from the antenna and beyond would be in the antenna's "far-field" region, in which the intensity falls off with the square of the distance. In such radiation, the electric and magnetic components are at right angles to one another and to the direction of propagation. In addition, because the ratio of the intensity of the electric component to that of the magnetic component has a constant numerical value, the intensity of the RFR can be stated in terms of the intensity of either component alone.

B.3. Current Knowledge About the Biological Effects of RFR

Most evidence for biological effects of RFR is derived from results of experiments in which various mammals (including human volunteers) and nonmammals such as birds, insects, and bacteria or other microorganisms were exposed to RFR, and specific biological effects were sought. Also studied were tissues such as excised organs and neurons, blood, single cells, cultures of cells, and subcellular components kept alive artificially (i.e., *in vitro*). Evidence is also derived from epidemiologic and occupational studies, but such results are regarded as indirect or inferential because the RFR-exposure levels and their durations most often are not known with any degree of accuracy.

B.3.1 Studies of Humans

B.3.1.1 Epidemiologic/Occupational Studies

Relatively few epidemiologic studies have been conducted on possible links between exposure to RFR and the incidence of chronic disease and all of those studies have been

retrospective. Retrospective studies consider disease or death distributions that have already occurred. Exposure is estimated. Prospective studies include measurements of exposure and follow the study population forward in time to see who develops specific diseases. The study groups for retrospective studies may be cohorts (groups of people) or "cases" (identified individuals with a particular disease). Cohorts must include groups identified as exposed to some agent and control groups, ideally identical to the exposed group in such factors as age, sex, and socioeconomic status, but not exposed to the agent. (Alternatively, "high" and "low" exposure groups may be used instead of "exposed" and "nonexposed" groups.) Cases are identified as people with some disease and they are matched with people having the same age, sex, etc., but not having the disease. Then comparisons are made to determine whether the amount of exposure to a agent results in a difference between the two groups. Because the onset of disease cannot be predicted in advance for individuals, case-control studies cannot be prospective.

Prospective cohort studies are lengthy (some may extend for decades) and expensive. They are employed only when the potential for scientific return is high. Most epidemiologic studies are retrospective, and the major problem lies in obtaining reliable and accurate estimates of past exposure (dose and fatal-dose rate).

It is common in epidemiologic studies to find apparent associations between exposure to an agent and the occurrence of some disease. However, such correlation, although suggestive of a cause-and-effect relationship, is insufficient to prove it. The association may be spurious as, for example, in the case of the correlation between the wearing of dresses and the incidence of breast cancer. Other criteria must be examined (Hill, 1965). These include:

1. The strength of association – for example, the incidence of lung cancer in smokers versus nonsmokers
2. Consistency – occurrence of effects in diverse groups; for example, increased lung cancer in smokers of both sexes, all age groups, and races
3. Specificity – for example, mesothelioma in smokers exposed to high levels of asbestos
4. Temporality – cause preceding effect in time
5. Biologic plausibility and coherence – a known or potential mechanism that is not in conflict with information about the history and biology of the disease
6. Biologic gradient – a dose-response relationship between the amount of exposure to the agent and the magnitude of the response
7. Experimental evidence – animal data that point to the same association.

These criteria are spelled out here in some detail because they are of considerable importance in analyzing the studies that are reported in the following sections.

For epidemiologic studies with RFR as the agent, the exposure characteristics (frequencies, levels, and exposure durations) are usually surmised or are coarsely estimated.

Often open to question is the extent to which the group of humans selected as unexposed controls matches the RFR-exposure group in all relevant factors except exposure, and whether unknown or uncontrolled non-RFR factors have substantially contributed to the results. Similar considerations are applicable to studies of occupational exposure, except possibly those in which

the exposures could be characterized better. In some epidemiologic studies, job title is used as the basis of estimating exposure, with varying degrees of uncertainty.

Robinette and Silverman (1977) chose 19,965 men who had served in the Navy during the Korean War who from their titles (electronics technician, fire control technician, or aircraft electronics technician) were regarded as repair technicians for electronic equipment and thus were assumed to have had occupational exposure to RFR. For the control group, the authors selected 20,726 Naval men considered operators of electronic equipment (titles: radioman, radarman, or aircraft electrician's mate) and were assumed to have had little occupational exposure to RFR. The records of the men in both groups were compared for mortality, in-service morbidity, morbidity in Veterans Administration hospitals, and granted and disallowed requests for disability compensation.

The decedent data showed no significant difference between exposed and control groups in deaths from all causes; the numbers of deaths in both groups were significantly lower than for corresponding groups of age-specific white males of the general population. The death rate from trauma was significantly higher in the exposed than in the control group. When deaths from trauma were subdivided into motor-vehicle and "other accidents," suicide, and homicide, the only significant difference between exposed and control groups was in the "other-accident" category. In that category, however, the death certificates and the other mortality data about the men in the exposed group showed that many had died in military-aircraft accidents after the Korean War, presumably because more of them later became flying officers. Thus, no statistical association was found between health effects and presumed RFR exposure.

The U.S. Embassy in Moscow was irradiated with low-level RFR from 1953 until February 1977. Lilienfeld et al. (1978) conducted a study on the health of U.S. personnel assigned to the Moscow embassy during that period. The authors identified 1827 employees and 1228 dependents as having been at that embassy during the 1953-1976 period. The controls consisted of 2561 employees and 2072 dependents assigned to embassies and consulates in Budapest, Leningrad, Prague, Warsaw, Belgrade, Bucharest, Sofia, and Zagreb during the same period. Periodic tests for RFR at those control sites showed only background levels.

Medical records were reviewed for 1209 of the Moscow employees and 834 of their dependents. The respective numbers for the control group were 1882 and 1507. Health questionnaires were returned by 969 Moscow employees and 1129 control employees. The number of questionnaires completed by the dependents was not clearly indicated in the report.

The questionnaire data indicated higher incidences of some health problems in Moscow employee groups than in controls: more correctable refractive eye problems; more cases of psoriasis in men; more cases of anemia in women; and more frequent cases of depression, irritability, difficulty in concentrating, and memory loss. The authors noted:

> In view of the possibilities which had been publicized of the increased danger to their health and that of their children, it is not at all surprising that the Moscow group might have had an increase in symptoms such as those reported. However, no relationship was found between the occurrence of these symptoms and exposure to microwaves; in fact, the four symptoms mentioned earlier, which showed the strongest differences between the Moscow and

> comparison groups, were all found to have occurred most frequently in the group with the least exposure to microwaves.

No discernible differences were found between Moscow and control groups in total mortality or mortality from specific causes, nor were there mortality differences between Moscow and control groups of adults or dependent children. The mortality rates for the Moscow and control groups were lower than for the U.S. population at large, except for cancer-related deaths, which were fractionally higher among Moscow-female (8 of 11 deaths) than control-female employees (14 of 31 deaths). The authors stated:

> It is difficult to attach any significance to the relatively proportion of cancer deaths in females because of the small numbers of deaths involved.

The authors recognized and commented on limitations of this study due to their inability to acquire complete sets of medical records, death certificates, and returned health questionnaires, and to the imprecision in classifying individual employees with regard to probable extent of RFR exposure. They also noted that for many of the medical conditions studied, the sizes of the study populations were too small for detecting less than twofold excess risks. In addition, they indicated that highest RFR levels were recorded late in the period of irradiation and therefore, for the subgroup with the highest potential exposure, the time during which health effects might have become apparent was the shortest. However, despite these acknowledged limitations, the authors were able to draw the following conclusions: for dependents, the authors found no differences between adults in the Moscow and control groups. The incidence of mumps in Moscow-based dependent children was twice that in control children. The incidences of congenital anomalies in children born after arrival of the parents at their duty stations were comparable for the Moscow and control groups.

The authors concluded:

> With very few exceptions, an exhaustive comparison of the health status of the State and non-State Department employees who had served in Moscow with those who had served in other Eastern European posts during the same period of time revealed no differences in health status as indicated by their mortality experience and a variety of morbidity measures. No convincing evidence was discovered that would directly implicate the exposure to microwave radiation experienced by the employees at the Moscow embassy in the causation of any adverse health effects as of the time of this analysis.

In one of a pair of studies, Lester and Moore (1982a) endeavored to establish an association between mortality from cancer and proximity of the decedents to Air Force bases. Polson and Merritt (1985) found this study to be flawed by incorrect assembly of the data base. When they independently assembled the data base correctly and analyzed it, they found that the cancer incidence for either sex in counties that had Air Force bases did not differ significantly from the incidence in counties that did not have Air Force bases.

In the second study, Lester and Moore (1982b) sought to determine whether there was a geographic pattern of cancer incidence in the city of Wichita, Kansas, and whether specific sources of RFR could be identified and related to any such pattern. Despite poor data on RFR exposure level, they reported finding a neighborhood pattern of cancer incidence, with a suggestion of a time element in its appearance, and noted that cancer tended to occur for persons

located on leading terrain crests relative to radar transmissions and occurred less frequently in the valleys. They derived a formula relating cancer incidence to the terrain and presence of RFR, leading to the overall finding that cancer incidence in Wichita appears to be related to the probability of RFR exposure from the radars at Wichita Mid-Continent Airport 15.5 miles southwest and 35 ft high, and McConnell Air Force Base 11.5 miles southeast and 130 ft high, both sites relative to the city center. The formula apparently bore no relation to actual exposure levels; the authors did not cite measurements to support their assumptions and gave no indication that the scan sectors of such radars were considered. A model of RFR exposure should have been used that is based on the physical laws of RFR propagation, particularly the inverse-square-law of attenuation with distance and the RFR shielding by artificial structures as well as terrain. Thus, any conclusions drawn in the paper are unwarranted.

Hamburger et al. (1983) noted that physical therapists are known to use various diathermy modalities (which they characterized as "microwave, shortwave, infrared, and ultrasound equipment") in the course of treating patients. They therefore sought to determine whether therapists might be suffering adverse health effects from exposure to the emissions from such units on a dose-related basis. They statistically analyzed the responses from male members of the American Physical Therapy Association (APTA) to a mailed questionnaire. The only consistent statistically significant finding was an apparent association between heart disease and exposure to shortwave (SW) radiation.

Although the authors considered other factors in the questionnaire, they emphasized that those health experiences reported in the RFR-bioeffects literature were associated with exposure to low levels of RFR. The responses requested from each subject included occupational history of diathermy use by length of employment in each position held since entering the clinical affiliation, and the number of treatments of each modality administered per typical work week. Other factors considered were the frequency of treatments, the years of work experience, and the use of infrared and ultrasound diathermy.

Three mailings of questionnaires were made, to reduce the number of nonresponses. The final population sample consisted of 3004 respondents from a total of 5187 therapists solicited. The respondents were divided into subgroups according to exposure across and within the energies of the four modalities above. The modalities were coded as U (ultrasound), I (infrared), M (microwave), and S (shortwave), and the authors initially distributed the population among the 15 exposure subgroups consisting of those exposed solely to each modality and those exposed to all possible combinations, plus a group that was not exposed. However, the small sizes of several groups necessitated merging them into other groups to ensure more meaningful statistical results, yielding nine subgroups.

Selected characteristics of the respondents were tabulated for the nine subgroups (age, race, marital status, present work setting, personal therapy with any modality, X-ray exposures) and prevalence among them of the following reported conditions: blood disorders, cataracts, diabetes, endocrine disorders, hearing disorders, heart disease, high and low blood pressure, nervous breakdowns, and "other."

The authors found that the reported prevalence rates for the entire cohort were below population rates in all instances, and that no single subgroup showed markedly higher rates relative to total rates. However, they noted that the all-four-modalities subgroup had significantly higher heart disease rates than those for the remaining population. They then formed new

subgroups: MW, SW, and joint MW/SW exposure, and further divided them into high- and low-exposure groups. A respondent with any exposure to MW was included in the MW group. Similar definitions were used for SW and joint exposure so the subgroups were not mutually exclusive, resulting in double-counting of subjects, a point recognized by the authors.

Contingency tables were constructed for the three types of exposure and for the three high-exposure versus low-exposure situations (treatment frequency, length of employment, and combination thereof), comprising 3 x 3 or 9 contingency tables for each of the 10 medical conditions, for a total of 90 contingency tables. The odds ratios for high exposure versus low exposure in the three exposure categories were also calculated for each contingency table, and the confidence intervals were determined for the odds ratios that were statistically significant after age adjustment. Heart disease was the only condition that remained significant, and only in 4 of the 9 type-of-exposure versus high-low situations (i.e., in 4 of the 90 contingency tables), a finding that is no better than chance.

The findings above may be cited by some as "proof" that exposure to MW/SW RFR causes heart disease. However, careful analysis of the paper did not yield convincing evidence that this is so. First, the paper illustrated the problems associated with attempts to uncover causal relationships between a purported health-effects agent (RFR in this case) and medical conditions in an identified population by using only the responses to a mailed, self-administered questionnaire. The response rate was 58%, with 2183 not responding. The authors did not mention attempts to reach a sample of nonrespondents by telephone or in person, to endeavor to characterize them as a group. (Statistical techniques exist to correct for bias when the nonrespondents group is large.) The 58% that did respond were self-selected in the sense that many of them may have responded because they had medical conditions and were curious about how such conditions may have arisen.

The major finding – that a statistical link exists between heart disease and self-reported recollection of one aspect of occupational exposure (frequency or number of treatments/week, but not employment duration) to SW and MW radiation (the correlation with SW was stronger than with MW), but not to joint SW/MW exposure – is dubious. The results indicate that SW exposure is a causal agent but that joint SW/MW exposure is not. Therefore, it can be inferred that MW exposure protects against possible adverse effects of SW exposure with respect to heart disease – an unlikely conclusion. In addition, duration of employment, which normally would be considered a factor in "cumulative exposure," had no statistically significant role.

On the basis of the RFR-bioeffects literature, the authors had classified heart disease into: (1) disorders of conduction/rhythm and ischemia, and (2) "other." They found statistical significance only for the first category. Heart disease, however, comprises many symptoms with various etiologies. For example, cigarette smoking is a widely known major risk factor and a strong predictor of heart disease in an aging population such as the 35+ group in the present study, for which the relationship with SW exposure was claimed. Inexplicably (but acknowledged by the authors), smoking history was not included in the questionnaire. Failure to consider this major biasing factor does not inspire great confidence in the sole positive finding of this study.

Milham (1983) analyzed the information on age and year of death in Washington State of 429,926 male decedents for 1950-1979 and 25,066 female decedents for 1974-1979, and presented cause-of-death analyses (160 causes) for 219 male and 51 female occupational

categories. One finding was an increase in leukemia in workers exposed to electric and magnetic fields.

The statistic used by the author was the 'proportionate mortality rate [or ratio]' (PMR). By definition, the PMR for each cause of death is the ratio of the number of deaths for that cause to the number of deaths from all causes, expressed as a percentage; accordingly, the sum of all PMRs must equal 100. A more commonly used statistic is the 'standardized mortality ratio' (SMR) because it represents the percentage of actual deaths for each cause relative to the expected number of deaths from that cause, independent of any other SMR (Lilienfeld and Lilienfeld, 1980, pp. 78-80).

The author examined the mortality patterns in selected occupations that appeared to have similar environmental exposures. It is only here that the categories of workers presumed to be occupationally exposed to magnetic and/or electrical fields were juxtaposed, and the PMRs for two categories of leukemia (acute leukemia, all leukemia) were given. The 11 occupations were: electrical engineers, electronic technicians, radio and telegraph operators, electricians, power and telephone linesmen, TV and radio repairmen, movie projectionists, aluminum workers, streetcar and subway motormen, power station operators, and welders and flame cutters.

Of the 22 categories (two leukemia categories in 11 occupations), 3 PMRs were high at the 1% significance level and 2 PMRs were high at the 5% significance level. Of the remaining 17 PMRs, 13 were elevated, 3 were depressed, 1 was unchanged, but none was statistically significant. As noted above, because the sum of PMRs for all occupations must be 100, the 5 significantly high PMRs might have been a consequence of abnormally low PMRs in three of the 11 occupations, a point explicitly recognized by the author. Thus, little credence can be given to the author's claim that the higher PMRs for acute leukemia and all leukemia are associated with exposure to electric and magnetic fields.

Another point to be considered is that a dose-response relationship must exist to conclude that cause-and-effect applies in this or any other epidemiologic/occupational study. Such a relationship was not established in this study. Without exposure data for the individuals or even for the occupations, no evidence exists that persons in these 11 occupations received more exposure to electrical and magnetic fields than those in other occupations. To illustrate this point, electricians – the occupation with the largest number of leukemia deaths (51) – do spend a large part of their time working on circuits that are not energized.

In a subsequent study, Milham (1988) examined mortality data for amateur radio operators presumably exposed to RFR while operating their transmitters. He extracted the names of 67,829 males in Washington State and California listed as licensed in the 1984 U.S. Federal Communications Commission Amateur Radio Station and/or Operator file between 1 January 1979 and 16 June 1984. Those names were searched for deaths during the 5-year period from 1 January 1979 to 31 December 1984, yielding a total of 2485 male decedents taken to have had 232,499 person-years at risk. Herein, the author did use the SMR.

Because the total expected deaths in both states from all causes was 3479, the 2485 deaths of licensees yielded an SMR of 71, with a 95% confidence interval (CI) of 69-74, indicating significantly lower death rates for licensees than for the general population. The category "all circulatory diseases" yielded the largest number of deaths, 1208, however, because 1732 were

expected, the SMR was only 70 (95% confidence interval: 66-74), also indicating a significantly lower death rate than in the general population.

The category "all malignant neoplasms" had 741 deaths, versus 839 expected, yielding an SMR of 89 (95% confidence interval: 82-95) – again a significantly lower death rate than for the general population. The only subcategory of malignant neoplasms that yielded an SMR that significantly exceeded 100 was "other lymphatic tissue." There were 43 deaths versus 27 expected; the SMR was 162, with a 95% CI of 117-218. The subcategory "leukemia" had 36 deaths versus 29 expected, for an SMR of 124, but the 95% CI was 87-172, thus rendering this result nonsignificant. The author considered nine subdivisions or subsubdivisions of "leukemia" and found that of the 36 deaths, 15 were for "acute myeloid" leukemia versus 8.5 of the 29 expected. These values yielded an SMR of 176 with a 95% CI of 103-285 for an apparently statistically significant result. However, little if any credence can be given to this finding, in view of the small numbers of deaths relative to the actual and expected totals. Thus, despite any claims to the contrary by the author, the results of this study do not offer any confirmation of those in Milham (1983).

Thomas et al. (1987) performed an analysis of brain tumor mortality risk for men occupationally exposed to RFR, lead, and soldering fumes in the petrochemical industry. They obtained death certificates of men who had died at age 30 years or older from brain tumors or other tumors of central nervous the system between 1 January 1979 and 31 December 1981 in northern New Jersey and in Philadelphia and its surrounding counties. They also acquired similar data for men who had died between 1 January 1978 and 30 June 1980 on the gulf coast of Louisiana. The lifetime work histories for the case men were obtained from next-of-kin. One control for each case was selected from men matched in age and year of death and area of residence, but who had died from causes other than brain tumor.

Case men were classified regarding RFR exposure by two methods. In the first method, the men were divided into two job-related categories: those engaged in the design, manufacture, installation, or maintenance of electronic or electrical equipment; and those exposed to RFR in other types of jobs (e.g., welding, radio broadcasting). In the second method, a certified industrial hygienist independently assigned codes to each job in the lifetime occupational histories for presumed exposure to RFR, to lead (high, moderate, low), and to soldering fumes (high, low). The authors noted that the classifications for RFR exposure in the two methods overlapped considerably, but that the second method included men in supervisory jobs not considered exposed in the first method.

Information was available on 435 cases and 386 controls. Of the 435 cases, 300 had astrocytic tumors, 90 had other recognized types of tumor cell, and 45 had unknown types of tumor cell. The authors estimated the maximum-likelihood relative risk (RR) and 95% CI for each exposure and job category, and adjusted the data for potential confounding influences of educational level. They regarded any RR as statistically significant if its 95% CI did not span 1.0.

The analyses showed significantly elevated RRs for astrocytic brain tumor among men classified as exposed to RFR in jobs involving design, manufacture, installation, or maintenance of electronic or electrical equipment. RRs were not elevated for exposure to RFR in other types of jobs. The highest RR was for the combined classifications of engineers, teachers, technicians, repairers, and assemblers. RR rose with exposure duration to tenfold for those in jobs associated with the manufacture and repair of electronics for 20 or more years, but the RR for "tradesmen"

(combined categories of electricians, and power and telephone linemen) showed no consistent pattern with increasing employment duration. On the other hand, the RRs were also higher for electronics workers classified as not having been exposed to RFR.

Elevated RRs were reported for those exposed to soldering fumes, but the variations with presumed exposure level were not large. However, nearly all of the men exposed to soldering fumes had such exposure in electronics manufacture and repair jobs. RRs were not elevated for lead exposure by level (low, medium, high) or overall.

On the basis of the results, the authors suggested that simple exposure to MW/RFR is not the responsible agent for excess brain tumor risk, and noted that exposure to such radiation in electronics jobs is probably intermittent and may be accompanied by exposures to lead, solder fluxes, solvents, and other chemicals. They also stated that the results should be interpreted with some caution because, when they calculated the risks for specific occupations and for individual strata by duration employed, they obtained very small numbers in single groupings.

Burr and Hoiberg (1988) compared the hospitalization rates of 1063 Naval pilots, who primarily flew "electronically modified aircraft" (the test group), with an age-matched control group of 2126 pilots who flew other aircraft. A major difference between the two groups was that the pilots of the test group were presumed to be subject to greater potential risks from exposure to ionizing radiation (such as at high altitudes) and nonionizing radiation (e.g., from onboard antennas, electronic equipment) than the control group.

The results showed that in the age range 21-26 years, control pilots had a significantly higher mortality rate for aviation-related injuries and higher hospitalization rates for accidents, poisonings, and violence than those in the test group. Also, in the age range 27-32 years, the pilots in the control group had a significantly higher hospitalization rate for mental disorders. The authors noted, however, that neither group had any hospitalizations for conditions related to either ionizing or nonionizing radiation.

Wertheimer and Leeper (1979) sought for and found statistically significant correlations between 60 Hz wiring configurations of homes in the Denver area and previous occurrence of cancer in children dwelling in them. The study was criticized for the absence of measurements of the fields in those homes, the possible presence of other agents known to contribute to cancer incidence, and possible bias because the researchers had known which homes had the cancer victims.

Savitz et al. (1986) conducted a similar but blind study on a different population in Denver. Those researchers measured the magnetic field at various locations within homes and did find a statistically significant correlation between the incidence of childhood cancer and proximity of their homes to wires carrying high currents. However, the association between the incidence of cancer and the magnetic fields actually measured was weaker than reported by Wertheimer and Leeper, and was within the margin for measurement error. Again it was suggested that agents other than the magnetic fields may have been responsible.

Other epidemiologic studies have been conducted on the possible association of cancer and exposure to power line fields, particularly magnetic fields. Taken collectively, the findings of those studies are inconclusive and the consensus is that further research is needed to determine whether such a linkage is real.

The EPA issued a preliminary report for external review (EPA, 1990) indicating a possible link between exposure to electromagnetic fields (RFR as well as those from power lines) and cancer. Recommended in the report was that electromagnetic fields in the extremely low frequency (ELF) range be classified as "probable human carcinogens," in a class with polychlorinated biphenyls (PCBs), DDT, and formaldehyde [see *Microwave News*, May/June 1990]. The report also recommended that RFR be designated as a "possible" carcinogen, in a class with saccharin.

The director of the EPA office that prepared the report ordered the ELF recommendation deleted in the absence of a mechanism of interaction and a dose-response relationship, and also deletion of the recommendation regarding RFR. Thus, the preface dated 13 December 1990 to the October 1990 draft of the report stated that:

> While there are epidemiologic studies that indicate an association between EM fields or their surrogates and certain types of cancer, other epidemiologic studies do not substantiate this association. There are insufficient data to determine whether or not a cause and effect relationship exists.
>
> Given the controversial and uncertain nature of the scientific findings of this report and other reviews of this subject, this review draft should not be construed as representing Agency policy or position.

Accounts of other activities regarding the EPA report, including comments by various individuals on both sides of the controversy, are contained in subsequent issues of *Microwave News*. *Microwave News* (September/October 1991), reported that the Committee on Interagency Radiation Research and Policy Coordination (CIRRPC) of the White House Office of Science and Technology Policy (OSTP) has urged the EPA to overhaul the report.

EPA's Radiation Advisory Committee of the Science Advisory Board established a subcommittee on Nonionizing Electric and Magnetic Fields to review the draft report. That subcommittee has issued a report, SAB (1992), indicating its findings. The subcommittee suggested numerous changes in emphasis, coverage, and wording, and concluded that the draft report [EPA, 1990] will have to be rewritten to accommodate all of the suggestions and comments.

The Science Advisory Board (SAB) of the EPA reviewed epidemiological studies on the possible link between cancer and exposure to EMFs and reached the following conclusion (EPA, 1992):

> 'Currently available information is insufficient to conclude that the electric and magnetic fields are carcinogenic. Some human epidemiologic data report an association between surrogates for electric and magnetic field exposure and an increased incidence of some types of cancer, but the conclusion of causality is currently inappropriate because of limited evidence of an exposure response relationship and the lack of a clear understanding of biologic plausibility.
>
> 'Nonionizing electric and magnetic fields should not be classified under EPA's chemical carcinogenesis system because of present major uncertainties. These involve an incomplete understanding of which aspects of field-tissue interactions give rise to biologic effects.'

As Section 2.3 of this appendix states, which discusses the basic differences between RFR fields and those from power lines, whatever the outcome of the controversy about a linkage between exposure to magnetic fields at power line frequencies and cancer incidence, the subject is not relevant with regard to the RFR from WSR-88D.

Several epidemiologic studies were conducted expressly on possible ocular effects of chronic exposure to RFR. The results of those studies are discussed in Section 3.1.3.2.

B.3.1.2 Congenital Anomalies

Two studies were conducted that sought a possible relationship between the occurrence of Down's syndrome and presumed exposure of the fathers to RFR from radars during military service. In the first study, Sigler et al. (1965) examined the data, derived from Baltimore hospital records and interviews with parents, on 216 Caucasian children with Down's syndrome. The case children were matched with 216 control children for hospital of birth (or birth at home), sex, and birth date (within 6 months), and nearly all were matched for maternal age (within 1 year) at time of birth. The parents were also matched for birthplace, residence, and hospital treatment.

Irradiation histories of the mothers were categorized as: diagnostic radiation excluding fluoroscopy, fluoroscopic exposure, radiation for therapy, and occupational contact. One statistically significant finding was that the percentage of case mothers that had received fluoroscopic examinations before the birth of the case child was significantly higher than for the control mothers. The percentage of case mothers who had at least one therapeutic radiation exposure (mostly for skin ailments) and the percentage of case mothers who worked in a professional or technical capacity in medical fields were also significantly higher than for the control mothers.

The difference in the percentages of case and control fathers that had served in the military was nonsignificant, but a higher percentage of case fathers reported close association with radars as technicians or operators than the control fathers. The authors thus ascribed the higher incidence of Down's syndrome primarily to greater exposure of the case mothers to ionizing radiation, but concluded: "The only truly puzzling association is the suggested relationship between Mongolism [Down's syndrome] and paternal radar exposure."

In the second study, Cohen et al. (1977) reexamined the data in the first study, denoted as the "Original Series," along with the data on 128 additional matched pairs denoted as the "Current Series." They concluded that the findings for the Current Series did not support the hypothesis that the fathers of the children with Down's syndrome had had excessive radar exposure or longer military experience.

Peacock et al. (1971) endeavored to assess whether the incidence of birth defects in Alabama could be associated with proximity to military bases. They examined a state-wide file of birth certificates by counties and found an overall rate of 10.3 newborns with anomalies per 1000 births, comparable to rates in other registries. A more detailed study of the data showed that there were 17 anomalies per 1000 births for the military personnel in the 6-county area surrounding Fort Rucker. By contrast, the anomaly rate for civilian births was 6.8 per 1000.

Peacock et al. (1973) reassessed the premise, but with data spanning 4 years rather than the 17 months examined previously. In addition, the data were corrected and rendered more accurate than previously, and a more precise test of the reliability of inferences was performed that did not

rely on the questionable use of a normal approximation. After accounting for "non-radar" factors, the authors repeated the analyses for the Fort Rucker area and specifically for Lyster Hospital (within Fort Rucker). In addition, as a "control" test, they compared the fetal death anomaly rates in the military hospitals at Fort Rucker and Eglin Air Force Base (which they designated as "radar bases") with those of three military hospitals in bases with minimal radar networks.

The results of the retests confirmed that the total anomaly rate and the rates for several specific anomalies were abnormally high at Lyster Hospital. The numbers of fetal deaths for Lyster hospital and the hospital at Eglin Air Force Base were also comparable and "constitute evidence that the problem may be associated with radar".

Burdeshaw and Schaffer (1977) reexamined the original Alabama birth records, but compared the data for Coffee and Dale Counties (within which Fort Rucker is located) with the data from each of the other 65 counties in Alabama on a score and rank basis instead of the statewide averages. They found little evidence that the incidence of congenital anomalies in the Fort Rucker area was unusually high. The overall rate at Lyster was well within the expectations for hospitals with characteristics similar to those of Lyster. When the addresses of mothers of anomalous infants were plotted on county road maps, no significant clustering was found, particularly in the vicinity of presumed radar sites.

Källén et al. (1982) hypothesized that physiotherapists (in Sweden) were likely to have had greater occupational exposure to various agents (chemicals, drugs, X-rays, RFR) than the general population. Therefore, they conducted a cohort study on 2043 infants born from 1973 to 1978 to 2018 women registered as physiotherapists during their pregnancies. They analyzed this cohort for perinatal mortality and the presence of malformations by comparing the data with information on all deliveries in the Swedish Medical Birth Register.

The results showed that for all endpoints, the expectation values for the total cohort were statistically better than, or comparable to, those for the general population. The authors noted that this excellent outcome could have been the result of a "healthy worker" effect; they thus hypothesized that if hazardous exposure exists, it should be more common among the few females who had dead or malformed infants than among those who had normal babies. Accordingly, they did a case-control study within the cohort, in which they selected 37 infants who had major malformations or those that did not but had died perinatally. Each infant was compared with two normal infants matched for maternal age, parity, and season of delivery (to compensate for work seasonality). Exposures for the case and control mothers were estimated from the answers to a questionnaire that asked (in part):

> Did you, during the pregnancy, work with or in close proximity to the following:
>
> - SW equipment: daily, often, seldom, never
> - MW equipment: daily, often, seldom, never
> - Ultrasonic equipment: daily, often, seldom, never
> - X-Ray equipment: daily, often, seldom, never
> - Electrostimulator: daily, often, seldom, never

Did you use hexachlorophene-containing soap (e.g., Phisohex): daily, often, seldom, never

On careful review and interpretation of the results, they concluded that the physiotherapists as a group had a slightly better than expected outcome for perinatal deaths and major malformations than did the general Swedish population for the same period. They reported that the use of SW equipment was higher among those who gave birth to a malformed or perinatally dead infant. In a critique of the paper, however, it was pointed out that those results would change from borderline significance to nonsignificance if one or two answers to the questionnaire were based on faulty recall.

B.3.1.3 Ocular Effects

The cornea and lens are the regions of the eye most vulnerable to RFR at high levels by their surface location and because any heat produced by the RFR is more effectively removed from other regions, particularly by blood circulation. Indeed, there have been several documented early cases of inadvertent exposure to RFR at levels high enough to cause cataracts, and appropriate safety measures have been taken to avoid such exposure.

B.3.1.3.1 Eye Damage by RFR in Animals

Carpenter et al. (1960) exposed predominantly the right eyes of 136 rabbits to 2.45-GHz CW RFR, one each at a level in the range 120 to 400 mW/cm^2 for respective durations of 60 to 10 min. After exposure, both eyes of each rabbit were examined at regular intervals with an ophthalmoscope and slit-lamp microscope.

The unexposed left eye of each rabbit was found to be clear in postexposure follow-up examinations. Level- and duration-dependent degrees of opacity were observed in the exposed right eyes for some exposure conditions. Those opacities were uniformly located in the posterior subcapsular cortex (PSC) and first appeared within 1 to 6 days after exposure, with a mean latency of 3.5 days.

The outcome of each exposure condition was shown as a circle on a graph of power density versus exposure duration, with solid circles for those that yielded lens opacities and open circles for those that yielded no effect. A curve that connected the circles representing the shortest duration at each power density that caused the development of an opacity was taken as the relation between the threshold power density for opacity induction by a single exposure versus exposure duration. The curve was a rectangular hyperbola, indicative of reciprocity (inverse relationship), with a nonzero power-density offset (asymptote) for exposures of long durations. The threshold exposure durations at 400 and 120 mW/cm^2 were about 3 and 35 min, respectively, with intermediate durations for power densities between those two values. By extrapolation for times longer than 60 min, the asymptotic threshold power density was roughly 80 mW/cm^2.

In a later study, Carpenter and Van Ummersen (1968) exposed one eye each of anesthetized rabbits to 8.2-GHz or 10-GHz RFR, also for a range of durations and input powers (power densities not indicated), and used the results to derive threshold-power versus exposure-duration curves for those frequencies. The curves at 8.2 and 10 GHz were similar to each other and to the curve for 2.45 GHz, but with an asymptote of 650 mW. For exposures to 8.2 and 10 GHz above

threshold levels, however, the cataracts always developed in the anterior cortex of the lens, whereas those for 2.45 GHz were typically located in the posterior cortex.

Guy and coworkers performed similar experiments at 2.45 GHz and obtained similar results, including threshold curves of incident power density versus exposure duration. As reported in Guy et al. (1975a), their threshold for exposure durations of 100 min (or longer) was roughly 150 mW/cm^2. In addition, they obtained the following results:

1. Increases in intraocular temperature of about 5°C or more are necessary for thermal eye damage.
2. Eye opacifications solely from exposure to RFR levels above the threshold are not produced at the same levels when the eye is cooled during exposure, showing that cataract causation by RFR is basically a gross thermal effect.

By contrast, Kues et al. (1985) reported that exposure of the eyes of anesthetized monkeys to 2.45-GHz CW RFR in weekly 4 h sessions at 30 mW/cm^2 yielded moderate-to-major changes in the numbers of corneal lesions seen with a specular microscope. Such lesions within fields of 1 mm^2 were counted in photographs of the central 6 mm of cornea. The local SAR was 7.8 W/kg, determined from temperature measurements in the eyes of live monkeys. Exposures at 5, 10, or 20 mW/cm^2 (1.3, 2.6, or 5.2 W/kg) caused no damage to the corneal endothelium. Representative results were presented for 1 monkey given 22 weekly sessions at 20 mW/cm^2, then 8 sessions at 30 mW/cm^2, and last, 2 series of 4 consecutive daily sessions at 20 mW/cm^2.

This study also appeared to indicate that exposure to 2.45-GHz pulsed RFR caused more effects than did exposure to 2.45-GHz CW RFR. Weekly 4-hour sessions of pulsed RFR at 10 mW/cm^2 average power density (2.6 W/kg) yielded minor or no corneal damage, but consecutive daily sessions at this level caused major damage – apparent evidence for effects of cumulative exposure. Moreover, one monkey showed major damage after only 1 session of pulsed RFR at 15 mW/cm^2 (3.9 W/kg).

Several representative specular micrographs were presented. Among those was one from a monkey before exposure, which clearly delineated the hexagonal cell boundaries and the nuclei of most cells, and exhibited no lesions. However, a photomicrograph, taken 48 h after another monkey was given a single 4 h exposure of pulsed RFR at 15 mW/cm^2, exhibited numerous lesions over areas of normal mosaic, each involving one cell or several contiguous cells. Also, a specular photomicrograph for a monkey 48 h after a session at 10 mW/cm^2 showed a similar distribution.

Kues and coworkers (Kues and Monahan, 1992; Kues, et al., 1992) conducted a similar study, which also showed that 2.45-GHz pulsed RFR (10-μs pulses at 100 pps) caused corneal lesions at a lower average power density (10 mW/cm^2) than 2.45-GHz continuous wave RFR (20-30 mW/cm^2). They also found that pulsed RFR at 10 and 15 mW/cm^2 caused iris damage in the form of increased vascular leakage through the blood-aqueous barrier. Leakage apparently occurred at some time during the 4-hour exposures and persisted up to 72 hours postexposure. The use of horseradish peroxidase (HRP) as the tracer in subsequent histopathologic examinations of some eyes confirmed the occurrence of such vascular leakage.

The authors then studied the effects of the drugs timolol maleate and pilocarpine, administered just before RFR exposure, on RFR-induced iris vascular leakage. Both drugs are clinically used to treat glaucoma (abnormally high intraocular pressure). They were selected to

test for possible thermal mechanisms for the observed vascular leakage; timolol was chosen because it had been shown to protect the eye against heat-induced disruption of the blood-aqueous barrier, and pilocarpine was chosen because it is known to increase the permeability of the barrier to the tracer sodium fluorescein when the iris is heated excessively. Exposures were to 2.45-GHz pulsed RFR for 4 hours a day on 3 consecutive days at a specific average power density for each sequence: 0 (sham), 5, 10, or 15 mW/cm^2 with or without drug application just before exposure. The corresponding SARs in the eye were 0, 1.3, 2.6, and 3.9 W/kg.

The results of treatment were scored for each eye as follows: 1 for no vascular leakage, 2 for minor leakage of fluorescein in tissue and the anterior chamber, 3 for moderate leakage consisting of partial filling of the anterior chamber with fluorescein more than 5 minutes after tracer injection, and 4 for significant fluorescein leakage within the first 3 minutes after tracer injection.

With neither drug applied, the mean vascular-permeability score was 1.0 (no effect) for 5 mW/cm^2 as well as sham-exposure; the mean scores for 10 and 15 mW/cm^2 were 2.1 and 2.8, both significantly higher than for sham-exposure (by t-test) but not significantly different from each other. For timolol, the mean scores for 0, 5, 10, and 15 mW/cm^2 were respectively 1.0, 2.7, 2.7, and 3.5; the latter three scores were all significantly higher than for sham-exposure, but the scores for 5 and 10 mW/cm^2 were the same. The corresponding scores for pilocarpine were 1.0, 2.4, 2.8, and 4.0. Again, the latter three scores were significantly higher than for sham-exposure, but the scores for 5 and 10 mW/cm^2 did not differ significantly from one another. Thus, for each drug, a mathematical relationship between the RFR-drug response and power density is not readily discernible.

Similar comparisons, at fixed power densities, of mean scores for either drug versus its absence also seem ambiguous. At 5 mW/cm^2, for example, the mean scores for timolol and its absence (2.7 and 1.0 above) differed significantly, but those at 10 mW/cm^2 (2.7 and 2.1) and at 15 mW/cm^2 (3.5 and 2.8) did not. Analogous results were obtained for corneal endothelial damage.

To estimate the threshold power density for the effects with timolol pretreatment, the authors exposed primates at 0.2 mW/cm^2 (0.05 W/kg) and 1.0 mW/cm^2 (0.26 W/kg). The mean scores for vascular permeability were 1.0 and 2.9, with the latter score significantly higher than for sham-exposure. However, the mean score for 1.0 mW/cm^2 (2.9) was comparable to the mean score for 5 and 10 mW/cm^2 (both 2.7). This result led the authors to conclude that timolol pretreatment reduced the threshold power density by a factor of 10.

As in Kues et al. (1985), the use of the same animals in different aspects of the study is open to question. Also unclear in this and the previous study was the dosimetry: the SARs were determined from *in vivo* temperature measurements at the corneal epithelium, but the authors gave no indication about the accuracy or reliability of those measurements. Another point not discussed was whether any significant differences occurred in results between the, presumably, equally treated left and right eyes of the subjects.

Stewart-DeHaan et al. (1985) excised the eye lenses from rats and exposed the lenses to 10-μs pulses of 918-MHz RFR at several SARs, repetition rates, and durations in a waveguide system while keeping the lenses at constant temperature. The lenses were fixed immediately after exposure, and depth of granular degeneration in the equatorial region, determined by scanning-

electron or light microscopy, was taken as the extent of lens damage. The pulses had a forward peak power of 24 kW and repetition rates that yielded mean forward powers in the range 65 to 0.5 W, with corresponding SARs in the range 1300 to 10 W/kg.

Statistical analyses indicated that lens damage increased with both exposure duration and SAR, with evidence for reciprocity. The authors stated that lens damage could be detected by scanning electron microscopy after 6 min of exposure at 40 and 20 W/kg, and by light microscopy after 1 h of exposure at 10 W/kg.

Creighton et al. (1987) reported on similar experiments, but with CW as well as pulsed RFR. Again, 10-μs pulses of 918-MHz RFR at 24 kW peak power were used; these were delivered at repetition rates that yielded SARs in the range 5.75-750 W/kg and durations of 6, 20, or 60 min. The range of total energy deposited in the lens was 0.23-15 watt-minutes per gram [W-min/g; 0.0138-0.90 joules per kilogram (J/kg)]. The results indicated that the pulsed RFR caused about 4.7 times greater depth of lens damage than the CW RFR at the same SAR for every combination of SAR and exposure duration except at 23 W/kg for 6 min. The authors suggested that the added damage could be ascribed to the thermoelastic effect of the pulses (see Section 3.1.4).

Foster et al. (1986) exposed the heads of anesthetized rabbits for 30 min each to 2.45-GHz RFR at various input powers in a waveguide system. The head of each rabbit was inserted in the waveguide with the left eye toward the RFR source. Measurements of input, reflected, and transmitted powers permitted determination of rates of energy absorption within the head. In each case, the transmitted power was negligible; as a result, the difference between the incident and reflected powers represented the power absorbed in the head. For the same reason, the right eye received virtually no power, thereby serving as the control for the left eye.

Immediate postexposure eye changes were noted, and each rabbit was given slit-lamp examinations for about 2 months. No changes were seen in the control eyes. The incidence of lens changes (in the left eyes) was plotted against both incident power and absorbed power (rate of energy absorption). Forward power and absorbed power were calculated at 8.7 W and 5.75 W, respectively. For a 375-g head, 5.75 W corresponds to a whole-head SAR of 15.3 W/kg. From measurements of rectal temperature, the authors found that 80% of the energy absorbed in the head was redistributed systemically, a finding that substantiates the concept that the thermoregulatory capabilities of animal influence thresholds.

Because the exposures in the waveguide were to the dominant electric field (TE_{10} mode), the authors calculated the power density corresponding to the 8.7-W threshold and obtained 285 mW/cm^2. They also calculated the equivalent free-space electric-field intensity to be 704 mV/cm. On the basis of these values, they noted that their waveguide power-density threshold compared well with those found with other kinds of exposure systems that provided quite different electric field intensities, and they concluded that the rate of energy absorption rather than the peak electric field is the significant parameter in RFR cataractogenesis.

B.3.1.3.2 Ocular Effects of RFR in Humans

Various epidemiologic studies expressly on possible ocular effects of RFR have been done. As with epidemiologic studies on other biological endpoints, the results were mixed. The following are representative.

Cleary et al. (1965) analyzed the incidence of cataracts in Army and Air Force veterans of World War II and the Korean War for possible association with occupational exposure to RFR. Examination of Veterans Administration hospital records produced 2946 veterans born after 1910 who had been treated for cataracts during the 1950-1962 period. A random sample of 2164 veterans hospitalized during the same period for other ailments was selected for control. By use of the military occupational specialties (MOSs), the authors classified each person as a radar worker, a nonradar worker, or one whose specialty could not be discerned.

In the radar group, the authors found only 19 with cataracts and 2625 without cataracts; in the nonradar group, only 21 had cataracts and 1935 did not. (Of the other 510 veterans, 202 of those with cataracts and 125 of those without cataracts had no indicated MOSs and the remaining 100 of those with cataracts and 125 of those without cataracts had MOSs that did not permit the determination of occupational category.) Evidently, at the outset, the small numbers of persons with cataracts in both groups yielded no basis for an association between RFR exposure and cataract causation. The authors indicated that the overall relative risk factor was 0.67, as compared with 1.0 for no increase in relative risk and values larger than 1.0 representing the degree of severity of the effect.

Cleary and Pasternack (1966) analyzed responses to a questionnaire on the occupational histories of personnel then currently employed at 16 MW installations, and used the histories to differentiate controls from exposure cases. They thereby selected 736 workers as occupationally exposed to RFR and 559 workers from the same locations and occupational environments (other than RFR) as controls. Exposure cases were grouped into occupational specialties by considering the types and functions of equipments used and the average generated powers, RFR frequencies, and modes of power termination. Exposure severity was classed by considering duration of work with each type of equipment, working distance from the equipment during normal operation, and specific type of work performed. Exposure scores were then determined by assigning appropriate weights to those factors (e.g., proportionality to average power, inverse proportionality to equipment distance).

The persons in both groups were examined with a slit lamp, and each was graded for subcataractous lens changes, classified as minute defects, opacification, reluctency, sutural defects, and posterior polar defects. A grade of 0 for "insignificant" to 3 for "large numbers or major degree of change (short of clinically recognized cataract)" was assigned in each category for each lens. An "eye score" consisting of the unweighted sum of scores for each type of defect was calculated.

A linear regression model was used for each group to relate mean eye score to age on the basis that the major increase in eye score with time was due to physiological aging of the lens. The slope of the regression line for the exposed group was significantly higher than for the control group; the lines crossed at 20 years of age with a mean score sum of 4.2. However, the authors remarked that no detrimental effects such as loss of visual acuity or higher propensity for cataract formation were associated with those subclinical eye changes, and that the results may indicate an acceleration of aging of lens tissue.

In addition to the usual uncertainties regarding actual exposure frequencies, levels, and durations, the grading of each worker for lens changes on an arbitrary 0-3 scale and the use of composite eye scores were open to question. These measures are subjective and not associated with actual reduction in visual acuity in the individuals examined; statistical analyses based on

such measures can be misleading. Other problems were a significant difference in age distribution between the exposed and control groups and the age-related lens changes in both.

In three studies (Appleton and McCrossan, 1972; Appleton, 1973; Appleton et al., 1975), surveys were conducted of the eyes of personnel at Army posts where various types of electronic communication, detection, guidance, and weather equipment were under development, test, and use. Examinations were conducted by ophthalmologists who had no prior knowledge of the histories of the individuals. The visual acuity of each person was determined. Then the pupil of each eye was dilated and the fundus was examined by direct ophthalmoscopy, with particular attention on the details of the posterior pole. The presence or absence of opacities or vacuoles and their location and number were recorded, as was the presence or absence of posterior subcapsular iridescence (PSCI), a manifestation claimed by others to be associated with RFR exposure.

The authors concluded that the available clinical evidence did not support the assumption that cataracts in personnel performing duties in the vicinity of MW generating equipment result from MW exposure, unless a specific instance of severe exposure can be documented and correlated with subsequent cataract development. However, they did not present any statistical treatment of the data.

Hollows and Douglas (1984) examined the lenses of 53 radio linemen who were occupationally exposed to RFR in erecting and/or maintaining radio, television, and repeater towers throughout Australia. The group included workers who had maximal cumulative RFR exposure, but excluded those known to have cataracts or who had cataracts removed. The RFR frequencies ranged from 558 kHz to 527 MHz. Power density measurements in and around work areas yielded values in the range 0.08-4000 mW/cm^2. The results of these examinations were statistically compared with those for 39 age-matched controls from the same Australian states who had never been radiolinemen.

The primary ocular finding was of posterior subcapsular cataract (PSC) in one or both eyes of 11 of the 53 radiolinemen (21%), compared with 3 of 39 controls (8%), or alternatively in 19 of the 106 eyes (18%) of the radiolinemen compared with 6 of the 78 control eyes (8%). The first result was not statistically significant, and the second was barely significant at the 5% level. The authors concluded that the excess of PSC radiolinemen may be work-related. However, the contribution of RFR exposure to those results is unclear because nuclear sclerosis, a type of lens opacity frequently attributed to exposure to solar irradiation, was reported in 50 (47%) of the eyes of the radiolinemen and 34 (44%) of the eyes of the controls. Also, the authors did not indicate the degree of vision degradation due to PSC and/or nuclear sclerosis.

Hocking et al. (1988) sought possible effects on the health of 9 radio linemen due to exposure to 4.1-GHz RFR from an open waveguide that was inadvertently activated. The men were divided into a "high-exposure" group comprising 2 individuals who had been exposed to 4.6 mW/cm^2 for up to 90 min, and a "low-exposure" group comprising the other 7 men who had been exposed to less than 0.15 mW/cm^2. By calculation, upper SAR limits for the skin, pituitary gland, and whole body for the high-exposure group were respectively 3.8, 0.06, and 0.13 W/kg. The range of SAR in their eye lenses was calculated to be 1.2-1.5 W/kg.

Both men in the high-exposure group reported loosening of their scalp hair, beginning about 2 weeks after exposure and lasting about a month. One of these individuals claimed temporary

sexual impairment and persistent symptoms of insomnia and irritability. The other individual had a moustache that was not affected.

The men in both groups were given medical examinations, their eyes were examined by ophthalmologists periodically for 9 months, and blood and semen samples were evaluated. One individual in each group exhibited significant elevation of creatine phosphokinase. No other significant biochemical or hematologic abnormalities were found in the high-exposure group. Three men in the low-exposure group exhibited mild abnormalities in liver function (ascribed to alcohol).

The authors tabulated the results of the ophthalmic examinations of each subject. In the first postexposure examination, the individuals in both groups had various eye abnormalities, but vision was normal in all. In the second post-exposure examination, eye changes were seen in both groups, but the third examination showed no further changes. The authors suggested that the changes could be ascribed to observational differences among the ophthalmologists. Their general conclusion was that the slight abnormalities seen in both groups were inconsistent and were unlikely to have been due to the RFR exposure.

B.3.1.4 Auditory Effects

Humans near some types of pulsed radar transmitters have perceived single pulses or pulse trains of RFR as audible clicks (without the use of electronic receptors). Considerable experimental evidence supports the hypothesis that an RFR pulse having a peak power density and duration within specific limits can produce a transient thermal gradient in the head large enough to generate a transient elastic wave at a boundary between regions of dissimilar dielectric properties and that this wave is transmitted by bone conduction to the middle ear, where it is perceived as a click. Persons with impaired hearing are unable to hear such clicks, and animals with destroyed cochleas (inner ears) do not exhibit RFR-pulse-induced evoked responses in the brainstem.

Frey (1961) exposed human volunteers to either 6-μs pulses of 1.3-GHz RFR at 244 pps or to 1-μs pulses of 3.0-GHz RFR at 400 pps. The mean average-power-density thresholds for perception were found to be 0.4 mW/cm^2 at 1.3 GHz, and 2 mW/cm^2 at 3.0 GHz. The corresponding peak power densities were 270 and 5000 mW/cm^2.

The author also tested four subjects having hearing loss (for air-conducted and/or bone-conducted sound) for perception of 1.3-GHz RFR. Subject 1, with both kinds of hearing loss above about 2 kHz, was unable to perceive the RFR pulses at intensities 30 times above the threshold. Subject 2, with bilateral severe air-conduction loss (about 50 dB) but moderate bone-conduction loss (about 20 dB), did perceive RFR pulses at about the threshold level. Subject 3, who suffered from tinnitus and bilateral hearing loss ranging from about 10 dB at 250 Hz to about 70 dB at 8 kHz for air conduction, more severe loss for bone conduction, and who had been diagnosed as having neomycin-induced nerve deafness, was not able to perceive the RFR pulses. Subject 4, with normal bilateral air-conduction hearing to about 4 kHz but severe bilateral bone-conduction loss, also could not perceive the pulses.

Frey (1962) found that some subjects with an audiogram notch around 5 kHz (and adequate hearing above and below 5 kHz) did not perceive RFR pulses as sound. The author also speculated about the possible sites and mechanisms of detection, including RFR-induced changes

of electrical capacitance between the tympanic membrane and the oval window, detection in the cochlea, and interaction of RFR with neuron fields in the brain. He discounted the first possibility because of insensitivity of the RFR-hearing effect to head orientation relative to the RFR source, and noted that the then-current experimental results were inconclusive with regard to the other two possibilities.

Frey (1967) subsequently tried to resolve this point by implanting an electrode in the cat brain stem and studying the potentials evoked by 10-μs pulses within the range 1.2-1.5 GHz. The results were not conclusive and may have contained artifact.

Frey and Messenger (1973) exposed humans to pulsed 1.245-GHz RFR at 50 pps in an RFR anechoic chamber. In one set, the average power density was held at 0.32 mW/cm^2 and the pulse width was varied from 10 to 70 μs in 10-μs increments, for peak power densities from 640 to 91 mW/cm^2. In another set, the peak power density was held at 370 mW/cm^2 and the pulse width was varied over the same range, for average power densities from 0.19 to 1.3 mW/cm^2. Four subjects with clinically normal hearing were each given three trials.

The results were shown as logarithmic plots of median values of perceived loudness versus peak power density and versus average power density. From their data, the authors calculated that the peak-power-density threshold for perception of RFR pulses is about 80 mW/cm^2, a value lower than those reported subsequently by Cain and Rissman (1978), discussed below.

White (1963) noted that when the surface of a body is transiently heated by RFR absorption (or electron bombardment), elastic waves are produced by surface motion due to thermal expansion. This process was analyzed theoretically, with emphasis on the case of an input heat flux varying harmonically with time, to relate the amplitude of the elastic waves to the characteristics of the input flux and thermal and elastic properties of the body. The results of experiments with both electron impact and RFR absorption verified the proportionality of the stress wave amplitude and the absorbed power density, and correlated well with the thermal and elastic properties of the heated medium.

Foster and Finch (1974) confirmed White's findings that RFR pulses can produce acoustic transients in water, and showed by calculation that for short pulses, the peak sound pressure is proportional to the energy per pulse, whereas for long pulses, it is proportional to the incident power density. Using 2.45-GHz RFR in several pulse-power-density and pulse-duration combinations and a hydrophone in saline, they found that the transition between the 2 regimes is for pulse durations between 20 and 25 μs. They also found that acoustic signals were not obtained in water at 4°C (where its thermal expansion coefficient is essentially zero) and that the polarity of the transient acoustic signal between 0° and 4°C was reversed from that for temperatures above 4°C. Those results support the thermoelastic expansion hypothesis.

Taylor and Ashleman (1974) surgically prepared three groups of cats for recording the potentials evoked by acoustic and RFR stimuli in three brain regions and for observing the effect of cochlear disablement. For presenting acoustic stimuli, a piezoelectric transducer was mounted on the dorsal surface of the frontal bone of each cat. In the first group, a glass microelectrode filled with physiological solution was inserted into the vestibulocochlear nerve and the round window on that side of the head was surgically exposed. In the second group, a microelectrode was inserted at a site that yielded acoustically evoked potentials, and both round windows were

surgically exposed. In the third group, a Teflon-covered carbon electrode was placed on the primary auditory cortex, and both round windows were surgically exposed.

The connections to the electrodes were made with carbon leads of high resistance. The acoustic stimuli were produced by feeding 10-μs electric pulses to the transducer at 1 pps. The RFR stimuli were 32-μs, 2.45-GHz pulses at 1 pps from a horn placed 10 cm from the cat's head.

The results indicated that cochlea destruction yielded a total loss of the evoked potentials to both acoustic and RFR stimuli. The authors concluded that in animals the transduction of RFR pulses into acoustic stimuli occurs in the cochlea in a manner similar to that of conventional acoustic stimuli.

In experiments similar to those of Taylor and Ashleman (1974), Guy et al. (1975b) studied the effect of cochlea disablement. Cats were prepared surgically for recording evoked potentials in the eighth cranial nerve, medial geniculate nucleus, and primary auditory cortex. After establishing that appropriate responses were obtained with RFR and acoustic pulses, the cochlea was disabled. Total loss of all evoked potentials resulted, even with the highest available peak acoustic and RFR powers and with computer averaging of large numbers of signals.

Guy et al. (1975b) also determined the power-density threshold and modulation characteristics for the RFR-hearing effect in two volunteers. The authors exposed the back of the head of the two humans to RFR at 15-30 cm from the aperture of a horn in an anechoic chamber at an ambient noise level of 45 dB, with RFR-absorbent material around the vicinity of the subject to eliminate reflections. The RFR consisted of 2.45-GHz pulses with durations that varied from 1 to 32 μs. For each duration, the RFR was presented in trains of 3 pps, with 100 ms between pulses. In each case, the subject signaled when perceiving an auditory sensation. From standard audiograms taken before exposure, the hearing threshold of subject 1 was normal and subject 2 had a deep notch at 3.5 kHz in both ears for both air and bone conduction.

For subject 1, the threshold for RFR perception was found to be a constant peak energy density (product of peak power density and pulse duration) of 40 mJ/cm^2 per pulse irrespective of pulse duration. At 3 pps, the corresponding average power density was 0.12 mW/cm^2. When subject 1 wore ear plugs, the threshold peak was only 28 mJ/cm^2 per pulse. The threshold for a pair of pulses spaced within several hundred μs was the same as for one pulse with the same total energy as the pair. Similar results were obtained for subject 2, but the threshold peak energy density was 135 mJ/cm^2 per pulse, about threefold higher than for subject 1.

The authors noted that each pulse was perceived individually as a click and that trains of short pulses were heard as chirps of tones that corresponded to the pulse repetition rate. In addition, when the pulse generator was keyed manually, digital (Morse) code transmitted thereby could be interpreted accurately by the subject.

Cain and Rissman (1978) used 3.0-GHz RFR pulses to study the RFR-auditory effect in 2 cats, 2 chinchillas, 1 beagle, and 8 human volunteers. For the animals, surface or brainstem-implanted electrodes were used to measure the responses evoked by audio clicks from a speaker and the responses to 5-, 10-, and 15-μs pulses.

For one cat, the threshold peak power densities were 2.2 W/cm^2 for 5-μs pulses, 1.3 W/cm^2 for 10-μs pulses, and 0.58 W/cm^2 for 15-μs pulses, and were respectively 2.8, 1.3, and 0.58 W/cm^2 for the other cat. For the beagle, the threshold values for the three pulse durations were 1.8, 0.30, and 0.20 W/cm^2. For the chinchillas, the values were 2.8, 2.0, and 0.58 W/cm^2 for one,

and 2.2, 1.0, and 0.50 W/cm^2 for the other. Thus, the beagle had the lowest absolute threshold, and compared to the other animals, the lowest thresholds at corresponding pulse durations.

The eight volunteers were given standard audiograms for both air-conducted and bone-conducted sound. In addition, because audiograms do not test hearing above 8 kHz, binaural hearing thresholds were determined for seven of the subjects for tone frequencies in the 1- to 20-kHz range. RFR pulses were presented at 0.5 pps. Each subject wore foam ear muffs during exposure, to reduce the ambient noise level, which was 45 dB.

Subjects 1-5 could hear 15-μs pulses as clicks; their peak power density thresholds were respectively 300 to 1000 mW/cm^2. Subjects 1-5 could also hear 10-μs pulses, with peak power density thresholds of 225 to 2000 mW/cm^2. Only subject 1 was able to perceive 5-μs pulses, with a threshold peak power density of 2500 mW/cm^2. By contrast, subjects 6-8 could not hear the 5-, 10-, or 15-μs pulses at the highest available peak power density but could perceive 20-μs pulses.

The authors found no correlation between the RFR results and the standard audiograms. However, they did note that a strong correlation existed between perception of RFR and hearing ability above 8 kHz as determined from the binaural thresholds. They also stated that their results are consistent with the hypothesis that an induced pressure wave in the human head in response to short RFR pulses contains a significant portion of its energy at frequencies above 8 kHz.

In summary, the results of Cain and Rissman's (1978) work with humans indicate that only subjects 1-3 could perceive 15-μs pulses, with a pulse-power-density threshold as low as 300 mW/cm^2 (energy-density threshold of 4.5 mJ/cm^2). Only subject 2 could hear 10-μs pulses, with 225 mW/cm^2 (2.3 mJ/cm^2) as the threshold; the thresholds for the other subjects were much higher than 300 mW/cm^2. Only subject 1 was able to perceive 5-μs pulses, but with a threshold of 2500 mW/cm^2. Those thresholds were for 45 dB of ambient noise and could be higher in noisier environments. Thus, the value 300 mW/cm^2 can be taken as the nominal human RFR-hearing pulse-power-density threshold for pulse durations of about 10 μs or longer.

It is noteworthy that Cain and Rissman (1978) had exposed the human volunteers to pulses of 3.0-GHz RFR at peak power densities as high as 2500 mW/cm^2 with no apparent ill effects.

Chou and Galambos (1979) investigated effects in 10 guinea pigs of external-ear blocking, middle-ear damping, and middle-ear destruction on brainstem-evoked responses (BERs) to both acoustic and RFR stimuli. The basic measurement technique was to record the amplitudes and latencies of the BERs to acoustic stimuli and to RFR pulses with a pair of carbon-loaded Teflon electrodes, one of which was attached to the left mastoid process and the other to the skin. The results showed strong evidence that activation of the cochlea is necessary for auditory perception of pulsed RFR and that perception is due to transduction into acoustic waves that travel via bone conduction to the cochlea or are generated directly in the cochlea itself.

Tyazhelov et al. (1979) studied the qualities of apparent sounds perceived by humans from exposure to 800-MHz pulsed RFR. The parietal area of the head was exposed to the open end of a waveguide fed from a 500-W source. Pulse durations ranged from 5 to 150 μs. The pulses were presented either continuously at 50 to 2000 pps (the latter for short pulse durations, to limit average power density) or in trains of duration 0.1 to 0.5 s at rates of 0.2 to 2.0 trains/s. Each person could be presented with sinusoidal audiofrequency (AF) sound waves independently of, or concurrently with, the pulsed RFR, and each person could adjust the amplitude, frequency, and phase of the AF signal to match the timbre and loudness of the perceived RFR.

The high-frequency auditory limit (HFAL) of each subject for tones from 1 kHz upward was tested first. Three subjects had HFALs below 10 kHz and could not perceive 10-30 μs RFR pulses, results that were consonant with those of Cain and Rissman (1978). Of 15 subjects with HFALs above 10 kHz, only one could not perceive the RFR pulses.

All of the perceptive subjects noted that 10- to 30-μs pulses delivered at 1,000 to 12,000 pps at peak power densities exceeding 500 mW/cm^2 produced sound of polytonal character that seemed to originate in the head, and that the quality of the sound changed with increasing pulse repetition rate (PRR) in a complex manner. Loudness diminished sharply and the sound became more monotonal as the PRR was increased from 6000 to 8000 pps, but no more than 3 distinguishable tonal transitions occurred. Subjects with HFALs below 15 kHz were unable to distinguish between the sounds perceived from a 5000-pps and a 10,000-pps signal, and subjects with more extended HFALs reported that the pitch for a 5000-pps signal was higher than for a 10,000-pps signal.

The subjects were able to detect small (5%) shifts of PRR only in the 8000-pps region; at lower PRRs, the subjects erred on 100% of tests to detect the direction of PRR change, indicating that increases of PRR were often perceived as decreases in frequency. For pulses of constant peak amplitude, loudness was perceived to: increase with duration from 5 to 50 μs, decrease from 70 to 100 μs, and increase again for 100 μs and upward. Such patterns of perception were exemplified by plots of threshold pulse power (normalized to the 10-kHz PRR threshold) versus PRR for a subject with a 14-kHz HFAL and another subject with a 17-kHz HFAL. The curves were roughly W-shaped, with a central relative maxima at about 8 kHz. A plot of mean threshold pulse power (normalized to the threshold at 50-μs pulse duration) was also presented for subjects unable to perceive sounds for pulses longer than 50 μs. This curve was also W-shaped, with a central relative maximum within the pulse range 100-120 μs.

When acoustic tones above 8 kHz were presented concurrently with 10- to 30-μs pulses at PRRs slightly above or below 8 kHz, the subjects reported hearing beat-frequency notes. For a PRR of 800 pps, similar beat frequencies were perceived when the acoustic frequency was set slightly above or below harmonics of the PRR. Moreover, when the tone and PRR frequencies were matched and the subjects were allowed to vary the phase of the acoustic tone, cancellation of perception of the two stimuli could be achieved. By proper phasing, the subjects with HFALs below 15 kHz could also achieve perception cancellation between a 10-kHz acoustic signal and a 5-kHz train of pulses.

The authors also reported that the sensory characteristics (pitch and timbre) evoked by RFR pulses shorter than 50 μs in duration were perceived as well when subjects' heads were lowered into seawater, with loudness diminishing roughly in proportion to the immersion depth and vanishing entirely with total immersion. For pulses longer than 50 μs, even partial immersion resulted in loss of perception.

Olsen and Hammer (1981) studied a spherical brain-equivalent model 10 cm in diameter. The model was exposed to 1.10-GHz RFR from an open section of waveguide, either as single pulses of 4-kW peak power and duration that was varied or as bursts of 3 pulses with an adjustable interpulse interval. For a nominal 10-μs pulse, the SAR was 824 W/kg at the center of the sphere and 653 W/kg at the surface facing the source. A hydrophone was placed at the center of the model to detect thermoelastic waves.

Exposure of the spherical model to single 14-μs pulses yielded ringing for each pulse, with a fundamental frequency of about 16 kHz and a time constant of about 500 μs. A plot of hydrophone response versus pulse duration over a 10- to 60-μs range showed maximum responses for 20-μs pulses. Bursts of 3 pulses each at burst frequencies ranging from 10 to 30 kHz yielded higher amplitudes than single pulses, with maximum enhancement at 16 kHz.

Olsen and Lin (1981) performed similar studies on spherical brain-equivalent models 6, 10, and 14 cm in diameter exposed to 10-kW, 1.10-GHz single pulses and to 3-pulse bursts from an open waveguide section. A plot of the peak hydrophone responses of the 6-cm sphere to bursts of 10-μs pulses versus burst frequency showed maximum response at 25.5 kHz. The corresponding data for the 10-cm sphere were the same as for those in Olsen and Hammer (1981). The response of the 14-cm sphere to single pulses was ringing at a fundamental frequency slightly above 10 kHz, and was maximum for 35-μs pulses. The responses of that sphere to bursts of 35-μs pulses had a peak at 11.5 kHz. Plots of the experimentally determined fundamental resonant frequencies for the three models occurred on the curve of resonant frequency versus head radius, derived from the thermoelastic theory for a homogeneous brain sphere with stress-free boundaries, thereby supporting that theory.

B.3.1.5 RFR Shock and Burn

People could experience electric shock or tissue burns when in the vicinity of transmitters that emit RFR at frequencies below about 100 MHz, and IEEE (1991) guidelines includes maximum exposure limits for avoidance of such effects. Such effects are of no concern with regard to WSR-88D because WSR-88D's 2700- to 3000-MHz RFR frequencies are much higher than 100 MHz.

B.3.2 Mutagenesis, Cytogenetic Effects, and Carcinogenesis

Mutagenesis and carcinogenesis are considered to be related, and many chemicals are screened for potential cancer-causing properties by testing whether such chemicals produce mutations in specific bacteria. RFR-induced mutagenic effects have been sought in various plants and animals. Relatively few studies have been conducted to determine whether RFR *per se* (e.g., 3 GHz) induces or promotes cancer. On the other hand, a few controversial epidemiologic studies have reported a statistical association of cancer promotion with exposure to power line fields (60 Hz). Some have wrongly inferred that such findings are applicable to RFR exposure, a subject that is discussed more fully later.

B.3.2.1 Microorganisms and Fruit Flies

Various strains of *Escherichia coli* (*E. coli*) bacteria, *Salmonella*, or yeast are often used for seeking mutagenic and cytogenetic effects because cultures of these organisms can be grown under well controlled conditions and examined for the effects of various agents on growth, survival rates, and genetic changes. The fruit fly is also commonly used for mutagenesis investigations.

Blackman et al. (1976) sought possible mutagenic effects of RFR in a strain of *E. coli* in which mutations can be detected readily. Cultures were held at constant temperature while exposed to either 1.7-GHz at 2 mW/cm^2 (3 W/kg) or 2.45-GHz RFR at 10 or 50 mW/cm^2 (15 or

70 W/kg) for 3 to 4 h. No significant differences in genetic activity were found between cultures exposed to either frequency for less than 3.2 h and sham-exposed cultures.

Dutta et al. (1979) obtained similar results with *Salmonella* cultures exposed to 2.45-GHz RFR at 20 mW/cm^2 (40 W/kg).

Anderstam et al. (1983) investigated whether RFR is mutagenic for *E. coli* or *Salmonella* bacteria (using a total of 11 strains). The RFR frequencies, 27.12 MHz and 2.45 GHz, were selected because of their wide use in industry. For some RFR treatments, some strains showed higher growth and others lower growth than their respective controls. Many of the changes were statistically nonsignificant, but the overall trend was toward RFR-induced increase in growth. Other results were both increases and decreases in mutant counts relative to controls, but most differences were nonsignificant.

In a study with the fruit fly, Pay et al. (1972) exposed male flies for 45 min to 2.45-GHz RFR at 6 mW/cm^2. Within 30 min after exposure, each male was placed in a vial with two virgin females. After initial mating, each male was placed in a new vial with two new virgin females every 24 h for 15 days after exposure. On day 8 after each mating, the females were removed and the number of days to the emergence of the first adult flies in each brood was recorded as the generation time. All broods were counted on day 17 of growth. No significant differences were found between the exposed and control groups in mean generation times or brood sizes.

Hamnerius et al. (1979) exposed embryos of the fruit fly to 2.45-GHz AM RFR. The embryos were of a sex-linked, genetically unstable stock in which the eye color is light yellow. The mutation sought was somatic, in which a shift in eye pigmentation results in eye sectors with normal red pigmentation clearly visible against the yellow background – a mutation that occurs at an early stage of eye development.

For exposure, embryos were immersed in 10 milliliters (ml) of water in a Teflon container, which was placed inside a larger Plexiglas container through which water held at 24.5°C flowed. The larger container was placed in the far-field region of a horn, with the RFR level measured with a power-density meter. SAR was calibrated by measuring the temperature rise in a biological sample due to a 30-s exposure at 900 mW/cm^2 with no water flowing through the larger container; the result was 0.5 W/kg per mW/cm^2.

Embryo exposures were at 100 W/kg (about 200 mW/cm^2) for 6 h. Controls were embryos similarly treated, except for exposure. Following treatment, the embryos were transferred to vials that contained standard medium and were maintained at 25°C and 75% relative humidity. The survival rate of the flies was determined from the number of male flies hatching from treated embryos, and the percentage of flies having red sectors constituted the mutation frequency. The mean survival rates for exposed and control flies were respectively 83% and 91%, a difference that was statistically nonsignificant. There were 4 mutations in 7512 RFR-exposed males (0.05%) and 2 mutations in 3344 control males (0.06%), also a nonsignificant difference.

Because confounding parameters, especially temperature rise, appear to have been controlled adequately, the finding of no RFR-induced mutagenic effect in this study is highly credible.

B.3.2.2 Mammals and Mammalian Tissues

Skidmore and Baum (1974) sought biological effects of exposure to electromagnetic pulses (EMP) resembling the RFR from a nuclear blast. They exposed 5 pregnant rats to a peak electric field of 447 kV/m in an EMP simulator during 17 days of gestation, a total of about 7 million pulses, with 5 unexposed pregnant rats as controls. On completion of exposure, the fetuses were examined for gross abnormalities. None were found. They exposed 20 female rats almost continuously to the EMP for 38 weeks (about 100 million pulses) and were observed for possible development of mammary tumors, together with 20 controls. At age one year, no mammary tumors were found in either group.

Those authors also exposed 50 male mice of strain AKR/J, known to be susceptible to spontaneous leukemia development between 6 and 12 months of age, to EMP. After 33 weeks of exposure (86 million pulses), 42 (84%) of the EMP-exposed mice and 24 (48%) of the unexposed control mice survived and were examined for leukemia. These results are difficult to interpret because it is not clear why more than half of the control mice died.

Of the survivors, 9 exposed mice (21%) and 11 control mice (46%) were leukemic, a nonsignificant difference. However, the sample sizes were too small to ascribe much if any validity to the latter finding.

The authors also sought possible effects of the EMP on fertility by exposing five 4-month-old male/female pairs of rats for 13 weeks. The males were separated from their partners for the first 8 days, but housed with their mates for the remainder of the period. Five unexposed pairs of rats were treated similarly. The numbers of progeny were recorded at term, and each neonate was examined for anatomical abnormalities. Also sought were effects of exposure on the fertility of five 2-year-old male rats by mating them on exposure termination with unexposed 4-month-old females. No anatomical abnormalities were found, and numbers of progeny were not significantly different.

Varma and Traboulay (1976) exposed the testes of 10 anesthetized 56-day-old mice to 1.7-GHz CW RFR at 50 mW/cm^2 for 30 min and the testes of 10 other mice at 10 mW/cm^2 for 80 min. Ten mice were sham-exposed with each RFR group. After 24 h for recovery, each mouse was caged for 1 week with three 56-day-old unexposed virgin females, and each subsequent week with new virgin females for 7 weeks. The authors used the "dominant lethal test" (mutations that result in the death of the embryo) to assess whether either RFR level was mutagenic. On gestation day 13, the females were euthanized and the numbers of live implants, late fetal deaths, and resorption sites were used to calculate the ratio of number of early fetal deaths to total number of implants.

The data contained tabulation errors and the statistical calculation was done incorrectly. With appropriate corrections, the differences in overall values between the RFR and sham groups were significant for both exposure conditions, but nonsignificant for most of the individual weeks postexposure. Exposure of the testes at 50 mW/cm^2 for 30 min caused a marked reduction in fertility, but exposure at 10 mW/cm^2 for 80 min caused only a marginal reduction.

As noted above, the mice were anesthetized during exposure. Only the testes were exposed, while their bodies were shielded from the RFR. However, anesthesia markedly reduces the ability of rodents to control their body temperature. Thus, the exposures may have caused

substantial rises in testicular temperature, so the results may not represent those for unanesthetized mice exposed to RFR at the same levels for the same durations.

Berman et al. (1980) conducted experiments on male fertility in rats by breeding RFR-exposed males with unexposed females. No evidence was found for an increase of dominant lethal mutations from 2.45-GHz RFR at power densities up to 28 mW/cm^2 (5.6 W/kg). Regarding male fertility, the authors noted that only under the most severe exposure regimen was there any hint of a deleterious effect: only 50% of the females bred to the exposed males 3-9 days after treatment became pregnant. Presumably, this temporary sterility was associated with the rises in rectal and intratesticular temperatures measured during exposure.

McRee et al. (1981), noting that analysis of induction of sister chromatid exchange (SCE) is a sensitive technique for assaying genetic damage from mutagens and carcinogens, used the technique to determine whether RFR is mutagenic in mice. They exposed twelve 10-week-old mice 8 h/d for 28 days to 2.45-GHz RFR at 20 mW/cm^2 (about 27 W/kg), with two other groups as controls. After exposure completion, the SCEs in bone marrow were determined, and the mitotic index for bone marrow cells was scored. The results for the three groups were comparable, an indication that there were no statistically significant effects of the RFR on SCEs or the proliferation rate of bone-marrow cells.

Meltz et al. (1990) investigated whether pulsed 2.45-GHz RFR alone can induce mutagenesis, chromosomal aberrations, and SCEs in mammalian cells, and whether the RFR can alter the genotoxic damage induced by a chemical mutagen alone when the RFR is administered simultaneously with the mutagen. The chemical agent used was proflavin, a DNA-intercalating drug (able to insert additional nucleotide bases into DNA sequences), and the mammalian cells studied were derived from a mouse leukemic cell line and purified of spontaneous mutations.

The six treatments listed below were conducted simultaneously on appropriately prepared and diluted cell samples in petri dishes, with each treatment involving two or four replicate, independent-treatment dishes, and with all treatments administered for 4 h in the dark. A sample treated with ethylmethane sulfonate (EMS) was run as a positive control for each set of replicates.

1. Controls at 37°C (no RFR and no proflavin)
2. RFR exposure alone, with no temperature control
3. RFR exposure with temperature control (heat convection to yield a temperature rise closely following that with RFR alone)
4. Proflavin treatment alone, at 37°C
5. Proflavin treatment with simultaneous RFR
6. Proflavin treatment with simultaneous temperature control.

The RFR exposures were to 10-μs, 2.45-GHz pulses at 25,000 pps (0.25 duty factor) in an anechoic chamber. The average power densities at the sample plane were 87 mW/cm^2 in the first 2 RFR experiments and 87 mW/cm^2 in the third RFR experiment. The corresponding SARs were 40.8 and 40.0 W/kg, respectively.

The combined RFR and proflavin treatment yielded no statistically significant increase in induced mutant frequency relative to the results for treatment with proflavin alone. Nor did exposure to RFR alone yield evidence of mutagenic action.

B.3.2.3 Cancer Induction and Promotion in Animals

Possible association between chronic RFR exposure and incidence of cancer has been reported in some epidemiologic studies (see Section 3.1.1), but for the reasons stated there, little credence can be given to such findings. On the other hand, few studies specifically directed toward determining whether RFR induces or promotes cancer in animals have been performed.

Prausnitz and Susskind (1962) exposed 200 mice to 9.3-GHz pulsed RFR for 4.5 min/d, 5 d/wk, for 59 weeks at an average power density of 100 mW/cm^2 (45 W/kg estimated SAR), with 200 other mice as controls. An unexpected finding was the development of leukosis in some of the mice, with incidence greater in the exposed mice than in the control mice. The authors mistakenly described leukosis as a "cancer of the white blood cells," thereby implying a link between RFR exposure and cancer incidence. In actuality, however, leukosis is defined basically as elevation of the numbers of circulating leukocytes, which in this study may have been due to the known occurrence of infection in the mouse colony or to other functional disturbances.

Roberts and Michaelson (1983), in a reanalysis of the primary data of Prausnitz and Susskind (1962), concluded that this study provided no evidence that chronic RFR exposure does or does not induce any form of cancer.

As discussed in Section 3.2.2, Skidmore and Baum (1974) found that continuous exposure of 20 females to EMP did not lead to the development of mammary tumors, and that EMP exposure of mice of the AKR/J strain prone to spontaneous leukemia did not promote leukemia; 21% of exposed mice versus 46% of control mice developed leukemia.

Szmigielski et al. (1982) investigated whether exposure to RFR: decreases the natural resistance of Balb/c mice to lung cancer cells injected intravenously before exposure; increases the incidence of breast tumors in female C_3H/HeA mice, a strain known to have high spontaneous incidence of such tumors; and increases the incidence of skin cancer in male Balb/c mice locally depilated and painted with the chemical carcinogen 3,4-benzopyrene (BP).

RFR exposures were to far-field 2.45-GHz RFR at 5 mW/cm^2 (SAR 2-3 W/kg) or 15 mW/cm^2 (6-8 W/kg). For exposure, 40 mice were placed in 4 polymethacrylate cages holding 10 mice each. Other groups of mice were sham exposed. The exposures were for 2 hours a day, 6 days a week, for periods of 1 to 6 months. The temperature (22-23°C) and humidity (60-70%) within the exposure chamber were held stable by external ventilation.

As additional controls to normal (cage-control) and sham-exposed mice, groups of male Balb/c mice were grown for 1 to 8 months, starting at age 6 weeks, within cages 20 x 30 x 10 cm in size containing 20 transparent 5 x 6 x 10-cm compartments, with one mouse in each compartment. The authors (citing appropriate references) noted that growth under such confinement causes a chronic stress syndrome with aggressiveness.

In the lung cancer study, Balb/c mice were intravenously injected with sarcoma L_1 cells. The mice were killed 14 days later, their lungs were infused with India ink in fixative, and the numbers of white neoplastic nodules (colonies originating from single cells) were counted. Based

on pilot experiments, the concentration of sarcoma L_1 cells used was 2×10^5 L_1 cells (in 0.1 ml of saline), which yielded a mean control value of 2.8 ± 1.6 (SD) nodules per mouse.

RFR exposure of injected mice for 3 months at 5 mW/cm^2 produced 6.1 ± 1.8 nodules, whereas 10.8 ± 2.1 nodules were seen for exposure at 15 mW/cm^2, a significant difference. Injection of mice that had been grown in confinement for 3 months showed 7.7 ± 2.0 nodules, a mean comparable to that for exposure at 5 mW/cm^2. Smaller but significant differences were seen after RFR exposure or confinement for 1 or 2 months.

In the breast-cancer investigation of C_3H/HeA mice, groups of 40 mice each were exposed to RFR from age 6 weeks up to age 12 months, and each mouse was checked every 2 weeks for palpable breast tumors. The cumulative numbers of mice with discernible tumors and their survival times were tabulated. By regression analysis, the results were summarized in terms of CDT_{50} (mean cancer development time in 50% of the mice) and MST_{50} (mean survival time of 50% of the mice). The CDT_{50} values were 219 days for 15 mW/cm^2, 255 days for confinement-stressed mice, 261 days for 5 mW/cm^2, 297 days for sham exposure, and 322 days for cage controls. Thus, the CDT_{50} values for 5 mW/cm^2 and confinement stress were comparable, and were between those for 15 mW/cm^2 and the cage controls. The results for MST_{50} were analogous.

Table B-1 of the paper, adapted below, showed the cumulative numbers of mice with tumors at 4, 6, 8, and 10 months:

Table B-1
CUMULATIVE NUMBERS OF MICE WITH TUMORS

	Tallied at:			
Treatment	**4 Months**	**6 Months**	**8 Months**	**10 Months**
Cage controls	0	0	2	16
Sham-exposed	0	0	3	14
RFR at 5 mW/cm^2	0	3	18†	32†
RFR at 15 mW/cm^2	1	11†	26*	37*
Confinement stress	0	2	16*	31*

*$p < 0.05$ relative to cage controls.
†$p < 0.01$ relative to cage controls.

Sham exposure did not significantly increase the numbers of mice with tumors, but both exposure at 5 mW/cm^2 and confinement stress yielded similar increases in the numbers of mice affected. Exposure at 15 mW/cm^2 yielded significantly higher increases.

At 5 mW/cm^2 (2-3 W/kg), no increase in rectal temperature was seen, but the authors noted that such SARs exceed the basal metabolic rate of the mice. In actuality, the basal metabolic rate of mice is about 9-10 W/kg (Durney et al., 1986), which is much higher than 2-3 W/kg estimated by the authors. At 15 mW/cm^2, (6-8 W/kg), no rectal-temperature increase was seen either, but the authors suggested the possible existence of "hot spots" within the mice.

In the skin-cancer experiments, to evaluate the effects of BP alone, 40 six-week-old male Balb/c mice had a 1-cm^2 of skin depilated, with the areas painted with BP (in a solvent) every other day for 5 months; controls were similarly depilated but painted only with the solvent. Cancer development was scored by histopathologic examination on a subjective 7-grade scale from 0 to 6. A score of 4 indicated that small papillomas had been found microscopically to contain cancer cells. Thus, mice with scores of 4-6 were regarded as having skin cancer, and those with scores 1-3 as having precancerous skin lesions. Skin cancer occurred within 7-10 months in more than 85% of those treated with BP.

Table B-2 of the paper, adapted below, shows the cumulative numbers of mice with skin cancer (scores 4-6) for cage controls (5 mice per cage), sham-exposure, RFR exposure at 5 mW/cm^2, or confinement stress for 1 and 3 months before BP treatment.

Table B-2
CUMULATIVE NUMBERS OF MICE WITH SKIN CANCER FROM EXPOSURE TO RFR BEFORE BP TREATMENT

	Tallied at:			
Treatment	**4 Months**	**6 Months**	**8 Months**	**10 Months**
Cage controls	0	0	3	18
Sham-exposure	0	0	4	19
RFR at 5 mW/cm^2 (for 1 month before BP)	0	2	18†	27*
Confinement stress (for 1 month before BP)	0	3	16†	24
RFR at 5 mW/cm^2 (for 3 months before BP)	1	22†	29†	36†
Confinement stress (for 3 months before BP)	0	16†	25†	31*

*$p < 0.05$ relative to cage controls.
†$p < 0.01$ relative to cage controls.

As in the breast-tumor study, the numbers of mice affected by exposure at 5 mW/cm^2 or confinement stress were comparable. It is not clear whether similar experiments were done for exposure at 15 mW/cm^2 for 1 and 3 months before BP treatment.

Regarding CDT_{50} values, control mice (treated only with BP) developed skin cancer (scores 4-6) in about 10 months (a CDT_{50} of 296 days). The results for other treatments were obscured by the order of their presentation, but presumably indicated that the CDT_{50} values for sham-exposure, confinement stress, exposure at 5 mW/cm^2, or exposure at 15 mW/cm^2 for 3 months before BP treatment were respectively 272, 201, 171, and 171 days, with the latter 3 values significantly lower those for the control mice. The reason for the lack of difference between the CDT_{50} values for 5 and 15 mW/cm^2 is not clear.

Table B-3 of the paper, adapted below, shows the results for concurrent RFR exposure and BP treatment. The only corresponding CDT_{50} value indicated was 131 days for mice exposed at 15 mW/cm^2. However, the MST_{50} values were 331, 268, 237, and 165 days for cage controls, exposure at 5 mW/cm^2, confinement stress, and exposure at 15 mW/cm^2, respectively.

Table B-3
CUMULATIVE NUMBERS OF MICE WITH SKIN CANCER FROM CONCURRENT RFR AND BP TREATMENT

	Tallied at:			
Treatment	**4 Months**	**6 Months**	**8 Months**	**10 Months**
Cage controls	0	0	3	18
Sham-exposure	0	0	5	21
Confinement stress	2	13*	26*	31*
RFR at 5 mW/cm^2	1	12*	23*	32*
RFR at 15 mW/cm^2	9†	28*	33*	38*

*$p < 0.01$ relative to cage controls.
†$p < 0.05$ relative to cage controls.

In their discussion, the authors stated: "It may be postulated that the differences in the appearance of tumors and the number [of] lung cancer colonies between animals irradiated with 2,450-MHz microwaves at 5 and at 15 mW/cm^2 may be due to local thermal effects evoked at 15 mW/cm^2."

They also noted that 2.45-GHz RFR is close to resonance for mice, that maximal absorption by humans at this frequency would be almost 2 orders of magnitude lower than for mice, and that RFR absorption by humans at their resonant frequencies (60-70 MHz) would be about 20% of the absorption by mice at their resonant frequencies. They stated: "Thus, the mouse model used in this study is of very limited value for concluding about the possible hazards from microwave radiation in human subjects."

In a University of Washington study of chronic exposure, discussed in 9 reports (Chou et al.,1983; Guy et al., 1983a, 1983b, 1985; Johnson et al., 1983, 1984; Kunz et al., 1983, 1984, 1985), 100 male rats were exposed unrestrained in individual cylindrical waveguides to 2.45-GHz RFR at average power densities of about 0.5 mW/cm^2 under controlled-environmental and specific-pathogen-free conditions. The exposure levels were selected to simulate, by scaling considerations, chronic exposure of humans to 450-MHz RFR at SARs of about 0.4 W/kg (the basis of the 1982 ANSI guidelines). The rats were exposed for virtually their entire lifetimes, except for those withdrawn for interim tests. The controls consisted of 100 sham-exposed male rats.

The exposure regimen began when the rats were 8 weeks old and continued for 25 months, which, except for the rats withdrawn for interim tests, encompassed virtually their entire lifetimes. After 13 months, 10 each of the RFR- and sham-exposed rats were euthanized (the

interim kill), as were 10 of the 12 RFR-exposed and 10 of the 11 sham-exposed rats that survived to the end of the 25-month exposure regimen.

No significant differences between groups were seen in daily body weight, food and water consumption, oxygen consumption, carbon dioxide production, or respiratory quotient at corresponding times. At each kill, the hearts, brains, livers, kidneys, testicles, and adrenal glands of the RFR- and sham-exposed rats were weighed, and the composition, fatty acid profile, and mineral content of the carcasses were analyzed. At interim kill, no significant differences between groups were seen in the mass of any organ. At terminal kill, mean adrenal mass for the RFR group was 75% higher than for the sham group, but the differences for the other organs were not significant.

During the exposure regimen, 157 rats had died spontaneously or were terminated *in extremis*. (Of the remaining 43 rats, 20 rats comprised the interim-kill groups; the other 23 were the survivors at the end of the exposure regimen and comprised the terminal-kill groups). Evaluation of the cumulative survival curves showed that the median survival times for the RFR- and sham-exposed rats were 688 and 663 days, respectively, but comparison of the curves by the log-rank statistic showed no significant difference between the groups at any age. The authors remarked that no significant infections had occurred to complicate or produce erroneous results in gross or histopathologic evaluations.

Section 3.5 of this appendix describes the findings of the immunologic and hematologic tests conducted.

In 20 rats necropsied at the interim kill, no adrenal tumors were found, but 7 of the 12 RFR-exposed rats and 4 of the 11 sham-exposed rats necropsied at the terminal kill were found to have benign adrenal tumors. The authors related the higher adrenal mass to the tumors. Exclusion of the rats with adrenal tumors from both groups rendered the difference in mean adrenal mass nonsignificant.

Gross and histopathological examinations were done. Primary causes of death (31 specific causes, 1 unknown-cause category, and the 2 kills) were tabulated separately for the RFR and sham groups.

Kidney failure (glomerulonephritis) was the largest cause, with 17 deaths in the RFR group and 15 in the sham group; next,was urinary tract blockage, with 9 and 19 deaths in the RFR and sham groups, respectively; third was atrial thrombosis, with 7 and 9 deaths; fourth was pituitary adenoma, with 4 and 8 deaths.

There were 22 RFR-exposed and 21 sham-exposed rats in the 2 kill groups. The all-other category had 41 deaths in the RFR group and 28 in the sham group. Chi-square analysis showed no association between cause of death and exposure condition. In addition, the log-rank statistic showed no significant differences between the RFR and sham groups in survival times for glomerulonephritis, atrial thrombosis, or pituitary adenoma; the RFR group had significantly longer survival times for urinary tract blockage than the sham group.

The lesions found in the various organs and tissues during necropsy were characterized as nonneoplastic or neoplastic, and the neoplastic lesions were subdivided into benign and malignant. Of the nonneoplastic lesions, glomerulonephropathy was the most prevalent. Analysis of the data by incidence, age, and treatment indicated that significantly fewer

glomerulonephropathic lesions had occurred in the RFR group. No significant differences occurred between the RFR and sham groups for nine other major types of nonneoplastic lesions.

Only 3 benign neoplasms occurred in rats younger than 1 year, and those were in the sham group. During the second year, benign neoplasm incidence rose rapidly with age for both the RFR and sham groups, but the differences between groups at each age of death were nonsignificant.

No primary malignant lesions were found in the rats younger than 1 year. Primary malignant lesions were found in 2 RFR-exposed and 2 sham-exposed rats at ages 13-18 months, in 9 of the RFR group and 1 of the sham group at ages 19-24 months, and in 7 of the RFR group and 2 of the sham group at ages 26-30 months.

Without regard to age, there were totals of 18 rats with malignancies in the RFR group and 5 rats in the sham group, a difference noted by the authors to be statistically significant. However, the authors indicated that the incidence of each specific primary malignancy in the RFR group was similar to that in the literature for untreated rats of the same strain. They stated that: "The finding here of excessive malignancies in the exposed animals is provocative; however, when this single finding is considered in light of other parameters evaluated, it is questionable if the statistical difference reflects a true biological activity."

The authors also noted that, from the standpoint of carcinogenesis, benign neoplasms have considerable significance under the assumption that the initiation process is similar for both benign and malignant tumors. The fact that the RFR and sham groups showed no significant difference in the incidence of benign tumors is an important element in defining the promotion and induction potential of RFR for carcinogenesis.

The conclusion of the investigators was as follows: "In summary, no defendable trends in altered longevity, cause of death, or spontaneous aging lesions and neoplasia can be identified in the rats exposed to this long-term low-level radiofrequency radiation exposure."

Santini et al. (1988) sought to determine whether low-level exposure of black mice (strain C57BL/6J) would affect the development of B16 melanoma or survival times. The authors exposed one group of 15 mice to 2.45-GHz CW RFR at 1 mW/cm^2 (SAR 1.2 W/kg) for 6 daily sessions per week, each 2.5 h/d, until death (up to 690 h total). Another group was similarly exposed to 2.45-GHz pulsed RFR at the same average power density. A third group was sham-exposed as controls. No statistically significant differences were found among the three groups either in tumor development or survival.

Balcer-Kubiczek and Harrison (1991) studied whether exposure to RFR of mouse-embryo-fibroblast-cell cultures induces malignant transformation in such cells. They exposed such cultures for 24 hours to 2.45-GHz RFR (amplitude-modulated at 120 Hz) at an SAR of 0.1, 1, or 4.4 W/kg alone, or to the RFR at 4.4 W/kg before or after exposure to X-rays at 0.5, 1, or 1.5 Gy. Control cultures were sham-exposed. After such treatments, cultures with or without incubation with the tumor promoter 12-*O*-tetradecanoylphorbol-13-acetate (TPA) at 0.1 μg/ml were assayed for incidence of neoplastic transformations by counting the number of transformed foci in culture dishes.

The sham-exposed cultures exhibited low incidences of neoplastic transformation; those incubated with TPA showed a slightly higher mean incidence than those not incubated with TPA. A plot of mean neoplastic transformation incidence (linear scale) versus SAR (exponential scale)

for the RFR-exposed cultures not incubated with TPA exhibited essentially no differences from sham-exposed cultures or any changes with increasing SAR, so the RFR alone did not promote transformation. However, the mean neoplastic transformation incidence rose with SAR for the RFR-exposed cultures incubated with TPA. The authors regarded those results as indicating that RFR acts synergistically in a dose-dependent manner with TPA to promote neoplastic transformation.

In graphs of mean neoplastic transformation incidence versus X-ray dose (Figure 2 of the paper), the cultures not incubated with TPA showed a relatively small rise with X-ray dose (0, 0.5, 1.0, 1.5 Gy), independent of whether the cultures were exposed to RFR (at 4.4 W/kg) or sham-exposed. For the cultures incubated with TPA, however, the mean transformation incidence rose linearly with X-ray dose for those exposed to 4.4 W/kg, and also linearly for sham-exposed cultures, but the latter with about half as many mean incidences as the 4.4-W/kg cultures at corresponding X-ray doses. Thus, exposure at 4.4 W/kg of cultures treated with X-rays plus TPA appeared to increase neoplastic transformation incidence relative to sham-exposure, but the differences in incidence at corresponding X-ray doses were statistically significant only for 0 and 0.5 Gy.

For cultures not treated with X-rays, sham-exposure yielded low incidences of neoplastic transformation, with a slightly higher mean incidence for those incubated with TPA than those not incubated with TPA; thus, TPA alone (at the dose used) did not promote transformation. On the other hand, RFR-exposed cultures not incubated with TPA showed essentially no differences from sham-exposed cultures or any significant changes with increasing SAR; thus, RFR alone also did not promote transformation. However, for the RFR-exposed cultures incubated with TPA, the mean neoplastic transformation incidence rose with SAR, an indication that RFR acted synergistically in a dose-dependent manner with TPA to promote neoplastic transformation. The numerical results were as follows:

a. The researchers found 14 foci in 1494 dishes of sham-exposed cultures incubated with TPA, and 4 foci in 887 dishes of sham-exposed cultures not incubated with TPA.

b. The researchers found 48 foci in 704 dishes of cultures exposed at 4.4 W/kg incubated with TPA; only 4 foci were found in 800 dishes exposed at 4.4 W/kg not incubated with TPA.

The study was well-conceived and conducted meticulously, but a number of aspects of the study make interpretation of the findings difficult and uncertain. First, a plot of mean neoplastic transformation incidence versus SAR (Figure 1) showed an apparently linear rise of incidence with SAR (0.1, 1.0, 4.4 W/kg). That result can be misleading because, unlike what was done for the plots of incidence versus x-ray dose (Figure 2), in which linear scales were used for both variables, the authors used a linear scale for incidence and an exponential scale for SAR. If the three SAR points had been plotted on a linear scale also, the graph would have displayed a fivefold sharper rise with SAR between 0.1 and 1.0 W/kg than between 1.0 and 4.4 W/kg.

The numbers of foci found relative to the numbers of dishes treated were small. The numbers of dishes used differed considerably for each treatment (see paragraphs a. and b. above). This point raises the question about whether the authors may have increased the number of dishes for each treatment until adequate percentages of foci for statistical analysis were obtained.

B.3.3 Teratogenesis

Teratogenesis refers to the causation of anatomical aberrations in a developing fetus, but more generally also includes fetal death and/or resorption and postnatal abnormalities in the offspring. Such effects occur naturally at low rates in most mammals, and relatively little is known about their causes. In a few cases, however, specific agents have been shown to cause significant effects, and hence the possibility that such effects could occur from exposure to RFR is an appropriate matter of public concern. The term is usually applied to mammalian fetuses and infants, but effects on nonmammalian subjects also have been sought.

B.3.3.1 Nonmammalian Species

Carpenter and Livstone (1971) exposed individual pupae of the darkling beetle to 10-GHz RFR for 2 h at 17 mW/cm^2 (40 W/kg estimated SAR) or at 68 mW/cm^2 (160 W/kg) for 20 or 30 min. As representative results, only about 20% of the pupae exposed at the lower RFR level developed into normal beetles; about 4% died and 76% had gross abnormalities. Exposure for 20 min at the higher RFR level yielded about 24% normal beetles, 25% dead ones, and 51% with gross abnormalities. By contrast, 90% of the sham-exposed pupae developed normally. Also, about 75% of the pupae heated conventionally to the temperature obtained with 17 mW/cm^2 emerged as normal beetles, leading the authors to conclude that abnormal development of RFR-exposed pupae could not be explained as a thermal effect. Liu et al. (1975) exposed pupae for 2 h to 9-GHz RFR at 0.17 mW/cm^2 (about 0.41 W/kg), which also yielded significant percentages of abnormal beetles.

On the other hand, Pickard and Olsen (1979) investigated pupae they had developed from larvae obtained from two sources (designated "colony-pupae" and "K-pupae"). Pupae from both groups were exposed for 2 h to a 6-GHz standing-wave electric field or magnetic field with their long axes parallel to the vector. The E-field exposures were at 91 V/m (130 W/kg) and the H-field exposures were at 1.53 ampere per meter (A/m) (54 W/kg). Pupae were also exposed for 13 h to far-field 6-GHz RFR at 11 mW/cm^2 (about 130 W/kg) and for 4 h to far-field 10-GHz RFR at 5 mW/cm^2 (45 W/kg).

For either E-field exposed group, the percentage of beetles with abnormalities did not differ significantly from that of its control group. However, the proportion of nonnormal beetles from control K-pupae was significantly higher than from control colony-pupae. H-field exposure also produced significant effects on K-pupae but not on colony-pupae. Exposures to the other forms of RFR yielded ambiguous results. The authors ascribed those variations to uncontrolled differences in such non-RFR factors as the source of the larvae, pupae maintenance regimes and handling protocols, the pupa containers used for pupation, and the ambient temperature.

Pickard and Olsen (1979) concluded that RFR could be teratogenic to the darkling beetle, but their results did not support the nonthermal hypothesis of Carpenter and Livstone (1971). In addition, Olsen and Hammer (1982) thermographically determined spatial distributions of SAR within pupae when exposed to 1.3-, 6-, and 10-GHz RFR, and found large variations of local SAR that would not be obtained by the conventional heating used by Carpenter and Livstone (1971).

In their studies of Japanese quail, McRee and coworkers found no significant differences between hatchlings from eggs exposed to 2.45-GHz RFR at a whole-egg SAR of 14 W/kg and

sham-exposed eggs in terms of average body weights, numbers and percentages of eggs hatched, numbers and percentages of hatched and unhatched live and dead birds, and blood parameters. In addition, no deformities were seen in the hatched quail. The authors concluded that RFR is not teratogenic except at hyperthermic levels.

Gildersleeve et al. (1987) on reproductive performance of Japanese quail from eggs that had been exposed to 2.45-GHz RFR at 5 mW/cm^2 (4 W/kg) during the first 12 days of embryogenesis also show that exposure to the RFR during embryogenesis does not affect the endpoints they studied, which included: hatchability, mortality after hatching, egg production, egg weight, fertility of the initial groups, and reproductive performance of the progeny.

B.3.3.2 Mammals

In some studies with mice, RFR exposure has been reported to cause teratogenic effects, but negative or inconsistent results have been obtained in others.

Rugh et al. (1974, 1975) exposed groups of female mice to 2.45-GHz RFR at 138 mW/cm^2 (SAR about 123 W/kg) for various durations to determine "D/M", the mean dose (power density x duration) per unit body mass, for lethality. The D/M for lethality was about 11 calories per gram (cal/g; 46.1 J/g). The authors exposed pregnant mice to the RFR at 123 mW/cm^2 (110 W/kg) for 2 to 5 min, corresponding to sublethal D/M values up to about 8 cal/g (33.5 J/g).

The authors found no teratogenesis threshold. However, a reanalysis of their data on the percentages of resorptions and of dead, stunted, and malformed fetuses versus D/M did indicate the existence of a threshold: at doses less than about 3 cal/g or power densities less than about 1 mW/cm^2, 100% of the fetuses examined were normal. Above that threshold, significant percentages of abnormal fetuses were obtained, but the dependence on RFR dose was obscure.

Chernovetz et al. (1975) found that absorption of about 5 cal/g of 2.45-GHz RFR is not teratogenic to mice, a threshold considerably higher than the 3-cal/g value above. In addition, they found a lethality D/M of about 5.7 cal/g (about half the Rugh et al. value), indicating that RFR teratogenesis would occur in pregnant mice only at levels that are close to lethality for the dams.

Stavinoha et al. (1975) exposed groups of 4-day-old mice in plastic containers for 20 min to 10.5-MHz, 19.27-MHz, or 26.6-MHz RFR pulses (pulse duration and duty cycle were not indicated) in a rectangular-coaxial transmission-line (TEM) system at an electric field strength of 5.8 kV/m. Control groups were maintained in similar containers outside the exposure chamber. The mice were weighed daily for the next 21 days. Graphs of weight versus age for each RFR frequency for exposed and control mice at corresponding ages showed virtually no differences between.

In another experiment, mouse pup litters were divided into 3 groups:

1. Control pups, kept in individual cages
2. Thermal-control pups, held at 3°C for 40 min/d on 5 consecutive days
3. Irradiated pups, exposed to 19-MHz CW RFR for 40 min/d on 5 consecutive days in a near-field synthesizer.

The electric field was 8 kV/m, the magnetic field was 55 A/m, and the two fields were parallel (vertical) in coincident planes. Following thermal or RFR treatment, the mean increase in rectal temperature was 1.5°C. The pups were weighed daily before each treatment and until they were 21 days old, at which time the males and females were separated. The mice were then weighed weekly for 13 additional weeks.

Statistical analyses of the growth curves showed no significant differences among the three groups of either sex. As the authors noted, although the fields were very intense, relatively little RFR energy was absorbed by the mice because their dimensions were much smaller than the wavelengths used. Thus, it would be inappropriate to apply such negative findings to humans exposed to RFR at frequencies in the same range.

Berman et al. (1978, 1982) found a consistent effect in mice: a significantly lower mean body weight of live fetuses from dams exposed to 2.45-GHz RFR at 28.0 mW/cm^2 (22.2 W/kg) than from sham-exposed dams. However, except for the negative results of Stavinoha et al. (1975) above, other researchers could not confirm such findings or found that growth retardation was thermally induced.

With rats, Berman et al. (1981) and Smialowicz et al. (1979) found no significant differences between RFR-exposed and control groups in any of the parameters commonly looked for in such studies, even at power densities capable of heating pregnant females to temperatures exceeding 40°C (104°F). This negative finding with rats led Berman et al. (1981) to conclude that mice may be more suitable than rats for seeking possible RFR-teratogenic effects in humans, a conclusion that seems specious in view of the large physiological differences between rodents and humans and among the rodent species themselves. Clearly, studies of nonhuman primates would be much more definitive.

Lary et al. (1983) treated 5 groups of rats on gestation day 9 as follows: The rats were euthanized on gestation day 20, at which time about two-thirds of them were found to be pregnant.

- Group I was sham-exposed for 2.5 h.
- Group II was exposed to 27.12-MHz fields at 55 A/m and 300 V/m (SAR about 11 W/kg), which produced relatively rapid colonic temperature rises; exposure was terminated when the temperature reached 41.0°C (14-22 min duration).
- For Group III, 41.0°C was held for an additional 2 h by on-off switching of the RFR (total exposure time 137-144 min).
- Exposure of Group IV was stopped when colonic temperature reached 42.0°C (13-33 min).
- In Group V, 42.0°C was maintained for an additional 15 min by on-off RFR switching (34-55 min total exposure time).

Comparing the groups in succession, steady increases took place in severity in both the percentage of malformed fetuses and ratio of litters affected, with by far the largest change for the prolongation of colonic temperature at 42.0°C. Similar results were obtained for percentages of live fetuses with visceral malformations, the largest change occurring again for prolonged

exposure at 42.0°C. The authors ascribed those teratogenic effects to the hyperthermia induced by the RFR.

Tofani et al. (1986) divided pregnant rats into four groups. The rats in group A (20 rats) were sham-exposed; those in group B (20 rats) were continuously exposed to 27.12-MHz RFR at field strengths of 20 V/m and 0.05 A/m (0.1 mW/cm^2 equivalent power density) during gestation days 0-20; and those in groups C and D (10 rats each) were similarly exposed respectively during gestation days 0-6 and 6-15. For exposure, 10 rats were co-housed in a plastic box of dimensions 80 x 60 x 35 cm, and two such boxes were used for concurrent exposure of group B, and similarly for groups C and D. The authors estimated the SAR to be about 0.00011 W/kg. They also noted that the basal metabolic rate (BMR) for such rats is 6.51 W/kg; accordingly, the SAR was insignificant relative to the BMR.

No dead fetuses were found. Total resorptions were found in half the dams of groups B and C and in 20% of the dams in group D, with none in sham-exposed group A. The values were statistically significant for groups B and C, and nonsignificant for group D, suggesting that this effect occurs during the early stage of egg development. Mean litter weights of the three RFR-exposed groups were significantly lower than for the sham group. The only significant teratologic finding was incomplete ossification of cranial bones in the three exposure groups.

In view of the low RFR level, the authors characterized the effects as nonthermal and due to long-term exposure. However, Lu and Michaelson (1987) took issue with the exposure methodology used. They remarked on the lack of description of the apparatus for providing food and water and for removing waste during exposure. They also noted that: the exposures were in the near field, that the use of RFR-absorbent materials was not discussed, and that the proximity of the rats to one another in the exposure boxes could have produced interactions that introduced large dosimetry uncertainties.

Lary et al. (1986) studied the dose-response relationship between RFR-induced maternal increases in body temperature and the incidence of birth defects in rats. The authors exposed groups of pregnant rats on gestation day 9 to 27.12-MHz RFR at field strengths of 55 A/m (magnetic) and 300 V/m (electric). The whole-body SAR was 10.8 W/kg. The exposures were terminated when colonic temperatures reached 41.0, 41.5, 42.0, 42.5, or 43.0°C (10-40 min). Exposed and control dams were euthanized on gestation day 20, and the uterine horns of each dam were examined for number of inplantations, live fetuses, and dead and absorbed conceptuses.

The numbers of various fetal abnormalities and of fetal mortality versus maternal colonic temperature on exposure termination were plotted (dose-response curves). The results indicated the existence of a colonic temperature threshold of 41.5°C for birth defects and prenatal death.

Kaplan et al. (1982), in a primate investigation designed primarily to determine whether chronic exposure of pregnant squirrel monkeys to 2.45-GHz RFR would alter usual mother-infant behavior patterns, found no differences between RFR-exposed and sham-exposed mothers in the number of live births or the growth rates of the offspring. Unexpectedly, however, a barely significant excess of infant deaths was found in the small group (5 animals) exposed at the highest level (equivalent power density 10 mW/cm^2). To investigate this unanticipated finding, a larger study was conducted with possible infant death as the primary endpoint. The exposure regimen used was similar, but with a sufficient number of dams to permit adequate statistical

treatment of the data. The results of this study did not confirm the previous finding of RFR-induced offspring mortality.

B.3.4 Nervous System

Concern has been expressed that direct (nonthermal) interactions of RFR with the central nervous system (CNS) could produce deleterious physiologic effects. It has been postulated that such effects may be manifested as alterations in behavior, passage of foreign agents from the blood vessels in the brain into the surrounding tissue by opening of the blood-brain barrier (BBB), changes in the histopathology and histochemistry of the nervous system and of the electroencephalogram (EEG), and changes in the efflux of calcium from brain tissue.

B.3.4.1 Blood-Brain-Barrier Effects

In most organs and tissues of the body, molecules in the blood can freely diffuse into the tissues around capillaries. However, to protect the brain from invasion by various blood-borne microorganisms and toxic substances, the BBB in most regions of the brain that allows little or no movement of large fat-insoluble molecules from the blood into the surrounding brain tissues. The BBB can be "opened" by certain agents (such as ionizing radiation or excessive heat) or by chemical substances (e.g., dimethyl sulfoxide). Studies have been done to determine if RFR can alter the permeability of the BBB in animals to substances of large molecular weight.

Frey et al. (1975) exposed rats to 1.2-GHz pulsed RFR at 0.2 mW/cm^2 average power density (estimated SAR 0.04 W/kg) or to 1.2-GHz CW RFR at 2.4 mW/cm^2 (about 0.5 W/kg). After exposure, the rats were injected with a tracer that fluoresces in ultraviolet light, and sections of various brain regions were scored for fluorescence. The authors reported higher fluorescence scores for the rats exposed to the pulsed RFR than the CW RFR, but noted that some brain sections of sham-exposed rats also fluoresced.

In another study, Oscar and Hawkins (1977) exposed rats to 1.3-GHz pulsed or CW RFR at levels up to 3 mW/cm^2 (about 0.6 W/kg), and measured the BBB-permeability changes to 3 substances that had been labeled with the radioactive tracer ^{14}C. For both RFR forms, significant levels of two of the substances were found in various brain regions, but not of the third substance.

Endeavors by Merritt et al. (1978) and by Ward and Ali (1985) to reproduce the findings of these studies were unsuccessful. The latter authors concluded that the earlier results were most likely due to artifacts introduced by the experimental techniques or to flaws in the basic methodology used. Preston et al. (1979) also showed that RFR-induced changes in the relative sizes of the vascular and extravascular volumes in the brain could be misinterpreted as BBB alterations.

Rapoport et al. (1979) developed a method for measuring permeability of the BBB to ^{14}C-labeled sucrose about 100 times more sensitive than the technique used by Oscar and Hawkins (1977), a method that yielded results independent of cerebral blood flow rate. With this method, Preston and Préfontaine (1980) showed that exposure of rats to 2.45-GHz RFR at 1 or 10 mW/cm^2 with their heads toward the source (0.1 or 1 W/kg head SAR) did not alter the permeability of the BBB to sucrose. Gruenau et al (1982) confirmed the negative findings of Preston and Préfontaine (1980), but for rat exposures to 2.8-GHz pulsed RFR (2-μs pulses, 500

pps) at average power densities up to 15 mW/cm^2 (3 W/kg) and CW RFR up to 40 mW/cm^2 (8 W/kg).

In other studies, such as that by Ward et al. (1982), in which results with RFR were compared with those for substances known to open the BBB as positive controls (e.g., urea), the findings were negative unless the brain had been rendered hyperthermic.

In four comprehensive studies, Williams et al. (1984a, b, c, d) exposed groups of conscious, unrestrained rats to 2.45-GHz RFR and used a variety of tracers and methods for detecting BBB penetration. They also determined the relationship of BBB changes to colonic temperature and the temperatures in several brain regions after exposure at levels up to 65 mW/cm^2 (13 W/kg) or to ambient heating at 42°C. At 20 mW/cm^2 (4 W/kg) and lower, no penetration was seen in those brain regions where the BBB is normally effective. The authors concluded that at the latter level, the thermoregulatory mechanisms of the rat are more than adequate for maintaining body temperatures well within tolerable limits, including brain temperatures, but that exposure to intense fields for long periods could approach or exceed such limits.

Neilly and Lin (1986) investigated the possible synergy between ethanol (which does not alter the BBB at normal intoxication levels) and levels of RFR sufficient to disrupt the BBB; they used Evans blue dye as the tracer. Groups of rats were anesthetized with pentobarbital. Each rat was placed on a heating pad to hold its colonic temperature near 37.5°C, and a catheter was inserted into its left femoral vein. When the colonic temperature of the rat reached between 37.2°C and 37.5°C, the rat was perfused through the catheter with a specified dose of warmed ethanol. Each group of rats was given a different ethanol dose within a specific range. When the colonic temperature stabilized at 37.0°C (usually 1-3 min after perfusion), the left side of the rat's head was exposed to 3.15-GHz CW RFR with an applicator at a net (forward minus reflected) power of 3000 mW/cm^2 for 15 min. A control group was perfused with saline instead of ethanol.

Following treatment, each rat was perfused with prewarmed Evans blue dye. After enough time had elapsed for dye circulation, the excess dye was washed out with saline, and the brain was removed. Each brain was scored for surface staining by the dye. The brain was then sliced into thin sections and each section was scored for internal staining.

In separate experiments, rats were similarly prepared but during the 15 min of RFR exposure and for 5 min afterward, brain temperature was measured with a nonperturbing probe inserted through a small hole burred in the cranium before exposure. Rectal temperatures were also recorded.

The brain temperature of each rat rose to a plateau within the first few minutes of exposure and remained there for the rest of the exposure. It then fell to preexposure level during the 5 min after exposure. The mean plateau temperature was highest (about 49°C) for the group given the lowest ethanol dose and it decreased steadily with increased ethanol dose to the lowest value (about 43°C) for the rats given the highest ethanol dose. The mean colonic temperatures of the various rat groups did not differ significantly from one another.

The results indicated that disruption of the BBB by the RFR was due to elevation of brain temperature. The authors concluded that, depending on dosage, ethanol can inhibit such disruption of the BBB by moderating the increases in brain temperature produced by the RFR.

They ascribed the action of ethanol to physiologic cooling of the RFR-exposed region of the brain, and cited references indicating that ethanol and pentobarbital can act synergistically.

B.3.4.2 Histopathology and Histochemistry of the Central Nervous System

Neural histopathologic and histochemical studies are, respectively, those of diseased or damaged neural tissues, and the chemical composition of such tissues. RFR effects have been sought *in vitro*, on preparations of neural tissues excised and kept alive in appropriate solutions while undergoing RFR exposure, and *in vivo*, from exposure of live animals.

B.3.4.2.1 Histological and Histochemical Studies *In Vitro*

In several histological studies, preparations of neural tissue were exposed to RFR *in vitro*. Courtney et al. (1975) excised superior cervical ganglia from white rabbits, stretched them singly across a vertical section of waveguide sealed at the bottom with a quarter-wave dielectric plate and filled with Ringer's solution, and exposed each ganglion to 2.45-GHz CW RFR from below. The calculated normalized SAR of the ganglion was 2.2 W/kg per mW/cm^2. The preganglionic end was connected to a set of stimulating electrodes outside the waveguide, and contact with the postganglionic end was made via a glass capillary through a wall of the waveguide. Ringer's solution at 37°C was pumped through the waveguide section.

Each ganglion was exposed to RFR at power densities up to about 300 mW/cm^2 (660 W/kg) for 1-minute intervals, with 1 minute between exposures for control measurements. The authors noted that only above about 100 W/kg did the temperature rise of the Ringer's solution exiting the waveguide exceed 0.1°C. During the exposure and control intervals, the ganglion was stimulated with 100-300 μs pulses at 1 pps, and the response latencies for synaptic transmission of the B fiber (short latency) and the C fiber (long latency) were determined. No significant changes occurred in the mean response latencies from the RFR at SARs up to 660 W/kg.

Chou and Guy (1978) similarly studied the rabbit vagus nerve and the cat saphenous nerve, as well as the rabbit superior cervical ganglion. As in Courtney et al. (1975), each ganglion or vagus nerve was mounted within a vertical waveguide section (parallel or perpendicular to the E-vector) through which Ringer's solution was pumped. The solution temperature at the fluid outlet of the waveguide was held constant at 37 ±0.02°C during exposure to the RFR. At the high RFR levels, however, the fluid temperature at the center of the specimen (measured with a nonperturbing probe) rose by as much as 1°C because of the limited circulation rate of the fluid pump.

Each specimen was stimulated with a 0.3-ms current pulse of 0.3-30 mA at 2-s intervals before, during, and after exposure to RFR. The compound action potentials (CAPs) were recorded, and their conduction velocity and amplitude were determined. Vagus nerves were exposed to 1-μs pulses of 2.45-GHz RFR at 1000 pps or 10-μs pulses at 100 pps for 10-minute periods at average SARs of 0.3, 3, 30, and 220 W/kg or to CW RFR at the same SARs, with 5 minutes between exposures. Superior cervical ganglia were exposed for only 5-minute periods because of their shorter lifetimes. Specimens were also exposed to pulsed RFR at 220 W/kg average (220 kW/kg peak) and to CW RFR at 1500 W/kg with no current-pulse stimulation, to test for the possibility of direct RFR stimulation.

No direct stimulatory effects of RFR exposure were observed at the highest available SARs (pulsed RFR at 220 kW/kg peak or CW RFR at 1500 W/kg) in the absence of stimulation by current pulses. With electrical stimulation, no changes in amplitude or conduction velocity of the CAPs were seen for exposures of rabbit superior cervical ganglia or vagus nerves to pulsed or CW RFR at SARs that did not increase the fluid temperature near the center of the specimen.

The conduction velocity and peak CAP amplitude versus time was shown for a representative cat saphenous nerve exposed to the CW RFR at 1500 W/kg, an SAR that increased the fluid temperature by 1°C. The conduction velocity rose by about 2% during exposure, a result reproduced by raising the temperature of the solution 1°C by non-RFR means. The variations in peak CAP amplitude were small and apparently not RFR-dependent. Similar data were shown for a representative stimulated rabbit vagus nerve before, during, and after exposure to 10-μs pulses (100 pps) at 220 kW/kg peak, and to CW RFR at 1500 W/kg. For the pulsed RFR, the fluid temperature rose 0.3°C, which increased the conduction velocity slightly (from 117 to 118 m/s). For the CW RFR, the fluid temperature rose by 1°C, which increased the conduction velocity from 117 to 135 m/s. An equivalent rise in fluid temperature by non-RFR means yielded the same velocity increase.

CAP recordings for a rabbit superior cervical ganglion exposed to 1-μs pulses (1000 pps) and to CW RFR at the same SARs as the vagus nerve were also made. For the pulsed RFR, the fluid-temperature rise was again 0.3°C, but the latency time remained unchanged. The rise in fluid temperature by the CW RFR was again 1°C, and the latency time decreased to 16 ms, a result reproduced by raising the fluid temperature 1°C by non-RFR means.

Among the histochemical effects sought *in vitro* were RFR-induced alterations in the activities of the enzymes acetylcholinesterase (AChE) and creatine phosphokinase (CPK) in rabbit blood. Olcerst and Rabinowitz (1978) found that 2.45-GHz RFR significantly decreased AChE activity, but only at a level sufficient to denature AChE (about 125 mW/cm^2). Galvin et al. (1981c) observed that 2.45-GHz RFR did not affect the activity of either AChE or CPK at SARs up to 100 W/kg.

B.3.4.2.2 Histological and Histochemical Studies *In Vivo*

Albert et al. (1981) indicated that prenatal development of the mammalian brain depends on migration of nerve cells, and specifically that a readily identifiable cell class in the cerebellum called Purkinje cells arises during the second half of gestation. They sought possible effects of prenatal and postnatal exposure to RFR of rats on such cells.

In 1 experiment, 2 groups of 3 pregnant rats each were exposed to 2.45-GHz RFR at 10 mW/cm^2 for 5 days starting on gestation day 17. The mean SAR was estimated as about 2 W/kg, with a range 0.8 to 6 W/kg due to rat movements. Six other rats were sham-exposed. Of the 6 pups in each group, 3 were euthanized on postnatal day 1, and the other 3 of each group were euthanized 40 days after birth. The cerebella from the day-1 pups were not mature enough for clear discernment of the Purkinje cells, but the mean Purkinje cell counts for the RFR-exposed 40-day pups were much lower than for the sham-exposed 40-day pups. The latter result was taken as indicating the permanence of this prenatal-exposure effect.

In another experiment, 1 pup each of 6 pairs of litter mates was exposed to RFR for 5 days, 7 h/d, whereas the other pup was sham-exposed. Three pups of each group were euthanized

immediately, and the other three of each group were euthanized 40 days later. In the groups euthanized immediately, the mean Purkinje-cell count for the rat pups exposed to the RFR was significantly lower than for those sham-exposed, but the mean counts for the 40-day RFR-exposed and sham-exposed pups did not differ significantly. The authors regarded those results as indicating the reversibility of the effect.

The statistical validity of the positive results above is open to question because of the large SAR variations noted by the authors. They also conducted a similar study on squirrel monkeys that previously had been exposed perinatally elsewhere, which yielded no significant differences between RFR- and sham-exposed groups in whole-body mass, brain mass or volume, or in counts of Purkinje cells.

Merritt and Frazer (1975) exposed mice for 10 minutes to 19-MHz RFR consisting of either an electric field at 6 kV/m with associated magnetic field of 6.4 A/m, or a magnetic field at 41 A/m with 2 kV/m associated electric field; these exposures did not increase rectal temperature. The objective was to determine whether such fields can alter the levels of specific neurotransmitters in the mouse brain.

Fifteen minutes after the 19-MHz exposure, the head of each mouse was exposed to intense microwave RFR to quickly inactivate its brain enzymes – a method that produced a rise in brain temperature of 40 to 50°C in 1 second. Sham-exposed mice were similarly treated. After such inactivation, the brains were assayed for the levels of serotonin (5HT) and its metabolite 5-hydroxyindole acetic acid (5HIAA), dopamine (DA) and its metabolite homovanillic acid (HVA), and norepinephrine (NE). No significant differences were found between the sham-exposed controls and those exposed to either the E-field or H-field in the mean whole-brain concentrations for any of the neurotransmitters or their metabolites.

Sanders et al. (1980) investigated the hypothesis whether exposure of brain tissue to RFR *in vivo* results in inhibition of the respiratory chain function, followed by decreases in concentrations of adenosine triphosphate (ATP) and creatine phosphate (CP). The authors noted that reduction of nicotinamide adenine dinucleotide (NAD) to NADH can be monitored *in situ* continuously by fluorescence measurements. Thus, if RFR exposure stresses the cells that inhibit respiratory chain function or cell functions that use ATP and CP, the NADH level will increase.

Rats were anesthetized, and a small aperture was made through the skull. The head of the rat was held rigidly in place, the light from a 366-nm excitation source was focused on a small spot on the cerebral cortex, and a fiber-optic bundle was directed toward the focal spot. The other end of the fiber-optic bundle was terminated at a wheel that housed a 460-nm filter to permit measurements of NADH fluorescence at that wavelength, and a 366-nm filter for reflectance measurements of the incident beam from the brain surface.

After preparation, each rat was exposed to 591-MHz CW RFR in the far field of a horn. The radiation pattern of the horn was such that only the head of the rat was exposed. Maximum SARs at the surface of a 2.0-cm diameter sphere and a semi-infinite plane of brain tissue were estimated to be 0.026 and 0.16 W/kg per mW/cm^2, respectively.

In a baseline experiment, after NADH fluorescence remained steady for 5 minutes in the absence of RFR, exposure to 591-MHz RFR was begun for 5 minutes at 13.8 mW/cm^2. In a representative graph of fluorescence, the intensity of the trace began to rise on RFR initiation. It reached a maximum of 12.5% above baseline level at 30 seconds, showed a compensatory partial

return toward baseline during the next 2.5 minutes, and than a slow rise during the remaining 2 minutes. The authors noted that the 30-s maxima for the rats tested were in the range 4% to 12.5% above baseline.

Groups of rats were then sham-exposed or exposed at 13.8 mW/cm^2 for 0.5, 1, 2, 3, or 5 minutes. Immediately after exposure, the head and neck of each rat were immersed in liquid nitrogen and the frozen head was removed and stored in liquid nitrogen. Subsequently, the frozen cerebral hemisphere near the aperture in the skull was extracted, pulverized, and assayed for ATP and CP. Other rats were sham-exposed or exposed at 5.0 mW/cm^2 for 0.5 or 1 minute and similarly processed.

Mean ATP and CP concentrations as percentages of baseline levels and their standard errors were graphed versus exposure duration. Sham-exposures yielded no statistically significant percentage changes in either mean ATP or mean CP level relative to baseline. For 13.8 mW/cm^2, however, 30-s exposures yielded the lowest mean ATP level – about 75% of baseline. For the longer exposures, the mean ATP levels were higher but did not exceed about 90% of baseline. The 30-se RFR exposures also yielded the lowest mean CP level – about 60% of baseline. The 3-minute exposures yielded a relative maximum of 85% of baseline and the 5-minute exposures showed a decrease to 60% again.

All of the percentage differences in mean ATP and CP between RFR and sham groups at corresponding times were significant, with most at the $p<0.005$ level. In particular, the mean ATP and CP levels for exposure at 5 or 13.8 mW/cm^2, each for 0.5 and 1 minute, differed significantly from their corresponding sham-exposure levels. For each duration, however, the difference in results between the two RFR levels was nonsignificant; these findings indicate that the effect may not be dose-dependent, or that it saturates below 5 mW/cm^2.

Temperatures at depths of 2-3 mm below the brain's surface were measured with a thermistor placed adjacent to the focal spot of the excitation light, with the leads at right angles to the electric vector. Rats other than those used in the NADH, ATP, and CP assays were exposed at 0, 13.8, 18.0, 30.0, 40.0, and 47.0 mW/cm^2 for 5 minutes. With sham-exposure, heat loss through the aperture in the skull was found to cause a decrease in brain temperature of 0.7°C at the end of the period. At 13.8 mW/cm^2, a decrease of 0.6°C was observed at exposure end; that value is close to that for sham-exposure, presumably an indication that the RFR at that level had added little heat. Decreases of 0.5 and 0.1°C were observed at 18.0 and 30.0 mW/cm^2, respectively, and increases of 0.2 and 0.1°C were obtained at 40.0 and 47.0 mW/cm^2. Rectal temperatures remained constant at all exposure levels.

From those results, the authors concluded that the observed changes in mean ATP and CP levels at 5 or 13.8 mW/cm^2 could not be ascribed to general tissue hyperthermia (but they did not exclude local hyperthermia). Instead, the data support the hypothesis that RFR inhibits electron transport chain function in brain mitochondria, thereby decreasing brain energy levels.

In another study, Sanders and Joines (1984) investigated the effects of hyperthermia alone and in conjunction with RFR. In the hyperthermia-only experiments, brain temperatures of sham-exposed rats maintained at 35.6°C yielded mean ATP and CP concentrations that did not differ significantly from those for sham-exposure reported in Sanders et al. (1980). Groups were then sham-exposed with brain temperatures held at 37.0, 39.0, or 41.0°C, and the mean ATP and CP concentrations versus brain temperature were plotted as percentages of the concentrations at

35.6°C. The percentages of both ATP and CP declined steadily with increases in brain temperature, with the CP rate of decline higher. At 39°C, ATP and CP decreased to about 90% and 70%, respectively; at 41°C, the decreases were to about 70% and 45%, respectively.

Rats were exposed at 13.8 mW/cm^2 for 0.5 to 5 minutes while their brain temperatures were held at 35.6°C, and the percentage increase in NADH and mean percentages of ATP and CP (relative to the concentrations at 35.6°C for sham-exposed rats) were plotted versus exposure duration. The results for 30-s exposures showed the largest decreases – to about 75% for ATP and to about 60% for CP. A similar set of experiments was conducted with brain temperatures held at 39.0°C before and during RFR exposure (RFR plus hyperthermia). At 0 minutes of RFR, the ATP and CP levels were respectively about 90% and 70%, with these decreases ascribed to the hyperthermia; the levels declined further to minima of about 60% and 45% for 1-minute RFR-exposures. For 5-minute exposures, the ATP level rose to about 80% and the CP level rose to about 50%. The general conclusion of the authors was:

> The decreases in ATP and CP in the 39°C brain during microwave exposure were significant and resulted in ATP and CP being much lower than observed at 35.6°C. Thus, at 39°C when the brain metabolic rate was increased, subsequent microwave exposure rapidly induced further decreases in ATP and CP, similar to the 35.6°C microwave exposure data, i.e., without a further increase in brain temperature; [these results] are consistent with the concept of direct microwave inhibition of energy metabolism.

The latter statement seems open to question, however, because an estimated local SAR of 8.5 W/kg at 13.8 mW/cm^2 must have produced considerable local heating, even though the mean brain temperature was held constant by external means.

Sanders et al. (1984) performed similar experiments, but at 200 MHz and 2,450 MHz as well as at 591 MHz. Local SARs in the brain of a dead rat were determined by measuring temperature rise versus time with a Vitek isotropic probe during exposure of the carcass to each frequency at 60 and 100 mW/cm^2. The normalized SARs at 200, 591, and 2,450 MHz were 0.046, 0.185, and 0.368 W/kg per mW/cm^2.

For the NADH fluorescence part of the study, a group of 6 rats was exposed at each RFR frequency. Each rat was given 2-minute exposures at increasing power densities in the range 0.5 to 20.0 mW/cm^2 (with 2 rats also given 40.0 mW/cm^2), and its NADH fluorescence was measured after each exposure. NADH fluorescence returned to baseline between exposures.

Because the differences in NADH results among the 6 rats of each group were large, their data were not combined for statistical treatment. Instead, the percentage changes in NADH versus power density were tabulated for each rat, with each serving as its own control. The 200-MHz data for each rat showed steady increases in NADH with power density, indicative of a dose-response relationship, with a trend toward saturation at the higher power densities. The results for 591 MHz were similar. However, no NADH changes were detected for 2,450 MHz, from which the authors suggested that the effect is frequency-dependent.

Whole-brain mean assays of ATP and CP for exposure to 200, 591, and 2,450 MHz at 13.8 mW/cm^2 versus exposure duration were shown graphically as percentages of baseline values. From the previously cited normalized SARs at the 3 frequencies, the SARs were about 0.6, 2.6,

and 5.1 W/kg, respectively. The largest effects were for 30-second exposures to either 200 MHz or 591 MHz; the levels of ATP decreased to about 80% and 75% of baseline for the two frequencies, respectively, and rose for exposures of longer durations. The mean CP assays showed no significant changes for 200 MHz, but a decrease to about 60% of baseline for 30-second exposures. No significant changes in either ATP or CP were seen at 2,450 MHz.

Given the negative findings at 2.45 GHz, the effect above is unlikely to be of importance relative to the RFR from the WSR-88D radar.

Lai et al. (1988) studied the results of single, 45-minute exposures of rats to 2.45-GHz CW or pulsed RFR (2-μs pulses at 500 pps) on choline uptake in several regions of the brain, with the uptake a measure of cholinergic activity. Exposures were done in groups of four individual cylindrical waveguide chambers (Guy et al., 1979) to circularly polarized RFR, or in a miniature anechoic exposure chamber (Guy, 1979) to plane-polarized RFR. The exposure levels were set to yield a whole-body SAR of 0.6 W/kg (1.0 or 2.07 mW/cm^2, respectively). Control rats were sham-exposed in the same chambers.

A dosimetry study by Chou et al. (1985) indicates that 0.6-W/kg whole-body SAR in a cylindrical waveguide yielded a spatial mean SAR in the head of 0.77 W/kg with the rat facing the RFR source and 0.91 W/kg with the tail facing the source. On the other hand, for a rat exposed dorsally in the anechoic chamber from above (with the top of its body toward the source) and its body axis parallel to the electric vector, the spatial mean SAR in the head corresponding to 0.6-W/kg whole-body SAR is 0.56 W/kg.

All four exposure conditions yielded decreases in choline uptake in the frontal cortex, with no significant differences between the CW and pulsed RFR. Decreases in choline uptake were seen in the hippocampus for exposures to the pulsed RFR in both types of chamber, but not to the CW RFR in either type of chamber. Choline uptake decreased in the striatum from exposure to pulsed or CW RFR in the anechoic chamber, but not in the waveguide chambers. On the other hand, no significant changes in choline uptake in the hypothalamus were seen for any of the exposure situations.

The authors suggested that the differences in results for the CW and pulsed RFR could be ascribed to the RFR auditory effect, noting that the characteristics of the pulsed RFR used were above the threshold for that effect. They did not suggest specific mechanisms for the other results.

B.3.4.3 EEG- and Evoked-Response Changes

Studies have been done to ascertain the effects of RFR on the EEG or on the response evoked by visual or auditory stimuli. EEG measurements done after completion of RFR exposure may be subject to interpretation problems stemming from the time consumed in attaching the electrodes and variability of their placement. Moreover, transient effects that may occur during exposure can disappear when exposure ceases. On the other hand, in studies to measure EEG changes during exposure, the electrodes and leads used to pick up the EEG signals also picked up electrical signals directly from the fields, introducing artifacts that rendered the recordings difficult to interpret. In addition, indwelling or chronically attached electrodes have perturbed the electric fields in their vicinity and yielded considerable enhancement of energy absorption, thereby creating still another artifact in the EEG data. However, in a few more recent studies,

those problems have been minimized by the use of specially designed indwelling electrodes of high-resistivity materials that do not cause field perturbation.

Experiments in which such specially devised electrodes were used, or in which electrodes were applied after exposure, yielded no evidence of significant differences in EEGs or in evoked responses between control and RFR-exposed animals. Therefore, there is no evidence that the EEG or evoked responses of people will be affected by the RFR emitted by WSR-88D units.

Takashima et al. (1979) sought the possible effects of amplitude-modulated electric fields on the EEGs of rabbits. They exposed rabbits to frequencies in the range 1-30 MHz between two 30 x 30 cm aluminum plates spaced 20 cm apart at fields in the range 0.5-1 kV/m. A circuit for impedance matching inserted between the plates and the source was used to minimize reflected powers. As noted below, EEGs were recorded before, after, and in some experiments, during exposure, with stainless-steel electrodes chronically implanted along the midline in the central and posterior regions of the brain; scalp electrodes were used in other experiments.

The EEG signals were fed to a preamplifier having a pass band of 3-100 Hz and a 60-Hz notch filter. In initial experiments, the authors found it difficult to interpret the unprocessed time-domain signals. In subsequent experiments, therefore, the signals were sampled at 5-ms intervals (1024 points), digitized, converted to complex spectra in the frequency domain by fast-Fourier-transform (FFT), and thence to power spectra. Smoothing the power spectra was required to resolve discrete frequency components, and sequential displays of the smoothed autocorrelation power spectra (usually 17 spectra at 3-min intervals) were examined for time-invariant features.

Typical preexposure power-spectra sequences from anesthetized rabbits showed frequency components between 5 and 15 Hz that varied during each sequence, indicating the absence of a dominant component. The authors denoted such EEGs as "normal".

To determine the effects of short-term exposure, anesthetized animals were exposed once (acute exposure) to fields modulated at 60 Hz. In one set of experiments (5 animals), stainless-steel electrodes had been implanted in the brain and were allowed to remain in the cranial cavity during exposure. The sequential set of power spectra obtained following exposure showed a clustering of amplitude peaks in the range 2-5 Hz that persisted over the postexposure recording period (40-60 min). Reduction of high-frequency components was also noted. Similar patterns were seen with no modulation, but to a lesser extent.

In a second set of experiments (2 animals), the EEG electrodes were removed before exposure and reinserted after exposure. (It was not clear whether surgery was used for such removal and reinsertion, and if so, what effect it had.) The resulting power spectra were said to resemble normal EEGs as defined above; no clustering of spectral components was seen. Therefore, the EEG alterations in the first set of acute-exposure experiments were attributed by the authors to the local field created by the presence of metal electrodes in the cranial cavity.

To investigate the effects of chronic exposure, 4 unanaesthetized animals were exposed 2 h/d for 6 weeks to 1.2-MHz fields modulated at 15 Hz. EEGs were recorded every 2 weeks with silver electrodes placed directly on the skull before and after exposure. A sequential display of the power spectra taken after 4 weeks of exposure showed ordering of low-frequency spectral peaks and reduction of high-frequency components similar to the acute-exposure data taken with intracranial electrodes. The abnormal patterns began to appear after 2 to 3 weeks of exposure. Histograms were constructed from power-spectrum sequences derived from 4-week exposures

and normal EEGs. The histogram for the exposed animals showed major peaks at 2 and 10 Hz, whereas the major peaks for normal EEGs were at 4.5, 8, and 11.5 Hz.

The authors assumed that the rabbit head (without the intracranial metal electrodes) could be modeled as a homogeneous conducting sphere immersed in a 10-MHz field of 500 V/m, and they calculated that the current density within the head was 0.082 mA/cm^2. Consequently, they regarded the positive results as a nonthermal effect. However, because they did not provide measurements or estimates of the SARs in the head, it is unclear whether the positive findings were thermal or nonthermal.

Even though enhancement of low-frequency components and increase in high-frequency activity after 3 weeks were reported, the data presented do not support this conclusion, and the authors themselves stated that the results presented were incomplete. However, they did note that for acute exposure, 'enhanced slow waves and unusually low high-frequency activities were due to the local field created by the presence of the metal electrodes in the cranial cavity.'

In this study, the rabbits were used as their own control group, in that data obtained during and after exposure were compared with "normal" (preexposure) data from the same group. However, the lack of a similarly treated sham-exposed group makes it difficult to assess whether the reported EEG changes were the result of exposure *per se*, or perhaps of adaptation to the repetitive aspects of the experimental procedures, such as handling and recording.

When power spectra for EEGs recorded at short time intervals (3 min) in a sequence are highly variable relative to one another, it is difficult to quantitatively assess the differences among the sequences. Autoregressive spectral estimation techniques may be more appropriate for analysis of EEG data than FFT techniques, because interval definition is problematic for nonstationary data. Qualitatively, nevertheless, the chronic-exposure data appear to show enhancement of low-frequency power-spectral components and reduction of high-frequency activities. On the other hand, comparison of data from the anesthetized animals used in the acute experiments with data from the unanesthetized animals used in the chronic experiments lacks analysis of the effects of anesthesia as a possible confounding factor. Moreover, it is not clear why the data taken after 4 weeks, and not after 2 and 6 weeks, of exposure were presented.

B.3.4.4 Calcium Efflux

Various studies have reported that exposure of samples of brain tissue from newly hatched chickens to 147-MHz or 450-MHz RFR at levels in the range 1-2 mW/cm^2 that were amplitude-modulated at low frequencies increases the rate of exchange of calcium ions between the tissue and the fluid bathing it (the "calcium-efflux effect"). The maximum effect was seen for modulation at 16 Hz, and no effect was seen for unmodulated 147-MHz or 450-MHz RFR. A similar effect was reported for exposure of the cortex of the paralyzed but awake cat.

Bawin et al. (1975) described the experimental protocol. After each neonate chick was decapitated, its forebrain was quickly excised and each of its cerebral hemispheres was incubated for 30 min in a mixture of physiologic medium and saline containing the radiotracer $^{45}Ca^{++}$. After incubation, the samples were washed 3 times in a solution without the tracer. They were then immersed in physiologic medium for 20 min during which 1 hemisphere of each chick was exposed to RFR, with the other hemisphere as the control. On treatment completion, an aliquot

of each bathing solution was processed and assayed for radioactivity by liquid scintillation counting.

The exposures were to a 147-MHz field between two large aluminum plates within an environmental chamber maintained at constant temperature and relative humidity. The field was amplitude-modulated at 0.5, 3, 6, 9, 16, 20, 25, or 35 Hz, or was not modulated. The RFR applied to the plates was adjusted to yield intensities in the range 1-2 mW/cm^2.

The results for the unmodulated field and each modulation frequency were plotted as bar graphs of the mean percentage concentration increases (effluxes) of $^{45}Ca^{++}$ relative to the corresponding mean for unexposed samples, with standard errors of the means (SEs). The unmodulated field and the fields modulated at 0.5, 3, 6, 9, and 16 Hz yielded progressively larger increases in calcium efflux, with a high of 19% (119% of control) for 16 Hz. Calcium efflux for 0 Hz (unmodulated), 0.5 Hz, and 3 Hz were nonsignificant ($p>0.05$); those for 6 and 9 Hz were significant at the 5% level, and those for 11 and 16 Hz were significant at the 1% level. For frequencies above 16 Hz, calcium efflux progressively declined with increasing frequency: the mean efflux was significant ($p<0.05$) for 20 Hz and was nonsignificant ($p>0.05$) for 25 or 35 Hz.

Other chick brains were used to compare the effects of RFR exposure on calcium efflux from brains with those from brains poisoned with sodium cyanide. Four sets were assayed, each set consisting of five poisoned samples and five normal samples: One set each was tested for the effects of exposure to fields modulated at 0, 0.5, and 16 Hz and one set was tested without exposure.

The results showed that although the mean effluxes from the normal and poisoned brains for 16-Hz modulation were significantly higher than their respective controls, the differences between poisoned and normal brains for each exposure condition were not significant. The authors stated:

> The field effects observed previously were not altered by the cyanide treatment, which strongly suggests that the $^{45}Ca^{++}$ effluxes from the cerebral tissues are independent of any ongoing metabolism.

On the other hand, the nonsignificant differences between the results for normal and poisoned brains appear to indicate that no calcium had moved across the cell membranes, (i.e., that calcium efflux is not an effect involving transmembrane calcium transport).

Bawin and Adey (1976) performed similar experiments to ascertain whether the previously observed calcium efflux from amplitude-modulated 147-MHz fields was due to the carrier frequency or the modulation itself. They exposed chick-brain preparations to sinusoidal fields at discrete frequencies of 1, 6, 16, or 32 Hz [in the extremely low frequency (ELF) and sub-ELF ranges] instead of to 147-MHz fields amplitude-modulated at such frequencies. Exposures were for 20 min at peak fields (in air) of 5, 10, 56, and 100 V/m.

The results indicated that the effect with ELF and sub-ELF fields was opposite to that with amplitude-modulated 147-MHz fields: decreases rather than increases of calcium efflux were obtained for all conditions, except with 1 Hz at 10 V/m, with maximum effect with 6 and 16 Hz at 10 V/m. In addition, the data indicated the existence of a field-amplitude "window" (as well as a frequency window). Specifically, for 6 and 16 Hz, the decreases were statistically significant at 10 and 56 V/m but not at 5 or 100 V/m.

Regarding their statistical treatment of the data, Bawin and Adey (1976) presented the rationale for discarding sample counts that were more than 40% above or below the mean of any set of 10 samples and for eliminating extreme counts in any set that were more than 1.5 standard deviations (SDs) from the mean. Despite the authors' rationale, the validity of discarding extreme values after the fact rather than from previous knowledge that an experimental error may have occurred in any specific sample is questionable and weakens the credibility of the results.

Sheppard et al. (1979) similarly prepared and exposed chick-brain halves to 450-MHz RFR sinusoidally modulated at 16 Hz only; this is the modulation frequency with which maximum effect occurred in prior studies with 147-MHz RFR. Exposures were for 20 min in a transverse electromagnetic (TEM) cell (rather than between two parallel plates) at power densities of 0.05, 0.10, 1.0, 2.0, or 5.0 mW/cm^2. After exposure, aliquots of bathing solution were assayed for calcium efflux. The differences in normalized radioactivity between the exposed and control samples were significant for 0.10 and 1.0 mW/cm^2, but not for the higher or lower levels, again indicating the existence of an intensity window.

Those authors stated:

> With attention to the important experimental steps (consistency in the pH and osmolarity of the physiological solution, and careful rinsing of the brains after incubation with the radioactive solution) it was possible to reduce the occurrence and magnitude of extreme values so that no data points were discarded.

Blackman et al. (1979) performed experiments to reproduce the 147-MHz results of Bawin et al. (1975). They also used a TEM cell to expose chick-brain samples for 20 min, but to 147-MHz RFR instead of 450-MHz RFR. Two series of exposures were made. In one series, power density was held constant at 0.75 mW/cm^2, and the modulation frequencies were 0, 3, 9, 16, and 30 Hz. In the other, the modulation frequency was held constant at 16 Hz, and the power densities were 0, 0.5, 0.75, 1.0, 1.5, and 2.0 mW/cm^2. The results seem to confirm the existence of the calcium-efflux phenomenon in excised chick brains, at least for 147-MHz RFR amplitude-modulated at 16 Hz. As the authors indicated, however, the power density window was narrower than those for modulated 450-MHz RFR and for sinusoidal sub-ELF fields.

Subsequently, Blackman et al. (1982) noted that their calcium-efflux changes (enhancements) were opposite in direction to the reductions found by Bawin and Adey (1976) at frequencies in the same range. They also noted that the alternating currents flowing in the walls of the TEM cell exposed their samples to a magnetic component as well as the alternating TEM field, whereas the parallel plates used by Bawin and Adey produced alternating electric fields only. Thus, Blackman and coworkers hypothesized that the magnetic component could influence changes in calcium efflux significantly and suggested that a direct current (DC) magnetic field such as that of the earth might also have a role.

Accordingly, the exposure apparatus used by Blackman et al. (1985) consisted of a transmission line that permitted use of an alternating electric field either alone or together with an alternating magnetic field. The transmission line was placed within a pair of large Helmholtz coils oriented to produce a DC magnetic field parallel to the earth's field, thereby permitting local alterations of its magnitude and polarity. The preparation of chick-brain samples was similar to that in previous studies.

Experiments in which the earth's magnetic field was altered by factors of 0.33, 0.5, 0.67, 1.33, or 2.0, or its polarity was reversed, yielded results that appeared to indicate a fundamental involvement of the earth's field in calcium-efflux changes. However, the relation between the sizes of such changes in DC magnetic field and the presence or absence of the calcium-efflux effect, if any, was obscure. It is noteworthy that the RMS value of alternating magnetic component used in most of the experiments was 59.5 nanoteslas, whereas the local value of the earth's DC magnetic field was 38 microteslas (or about 600 times larger).

In a more recent study, Blackman et al. (1991) reported that calcium efflux can be reduced, enhanced, or nullified by appropriately varying the temperature of the chick-brain samples before and during exposure. The authors hypothesized the existence of a temperature-window effect (an effect that occurs only within a narrow temperature range) and suggested that these results could account for the different outcomes (including those of opposite direction of effect) by various researchers.

Shelton and Merritt (1981) investigated whether pulses of RFR at repetition rates comparable to the modulation frequencies used in the chick-brain studies would elicit alterations in calcium efflux from the rat brain. In each experiment, pairs of samples of cerebral hemispheres from euthanized rats were processed together for RFR- and sham-exposure. Each sample was immersed in medium containing $^{45}Ca^{++}$ in a beaker, in which the sample was incubated for 20 min at 37°C. Then the samples were washed once with medium that was free of $^{45}Ca^{++}$ and were transferred to clean beakers containing radioactivity-free medium for RFR- or sham-exposure. Because the $^{45}Ca^{++}$ concentration could be affected by the extent of tissue washing, the samples in one experiment were washed with radioactivity-free medium 5 times instead of once.

In four experiments, exposures were for 20 min to 20-ms pulses of 1-GHz RFR, 16 pps, a repetition rate analogous to the 16-Hz amplitude modulation used in the chick-brain experiments. The average power densities were 0.5, 1.0, 2.0, or 15 mW/cm^2, selected to search for the reported power-density window. In two other experiments, samples were exposed for 20 min to 10-ms pulses, 32 pps, at 1.0 or 2.0 mW/cm^2. Sham-exposures for each experiment were conducted similarly. For each experiment, four groups each of RFR- and sham-exposed samples made up the population.

In a seventh experiment, samples washed 5 times instead of once were exposed to 20-ms pulses, 16 pps, at 1.0 mW/cm^2 (or sham-exposed). In an eighth experiment (with samples washed only once), the exposure parameters used were the same, but exposure was interrupted for about 1 minute each after 4, 8, and 12 min of exposure, and 0.5-ml aliquots of the incubation medium were taken and assayed for possible time-dependent effects and replaced with fresh medium.

On completion of RFR- or sham-exposure, the radioactivities of the media and tissue samples were assayed by liquid scintillation counting. These authors defined "efflux value" as the ratio of counts per minute (CPM) of the medium to the sum of the CPMs in tissue and medium, an efflux measure that differed from that used by other investigators. The cerebral tissues taken from 143 rats in the first 6 experiments yielded nonsignificant differences between any of the mean $^{45}Ca^{++}$ effluxes when RFR-exposed samples were compared with their corresponding sham-exposed control samples. The mean effluxes for RFR- and sham-exposure in the seventh experiment (24 rats, 5 washings instead of 1) also did not differ significantly from

one another, but both means were higher than those obtained in the first 6 experiments. In the eighth experiment (interruption of exposure), successive rises in efflux were seen at the three interruption intervals, but again the differences in the means for RFR- and sham-exposed samples at corresponding times were nonsignificant.

Thus, the findings of Shelton and Merritt (1981) were negative, but as the authors pointed out, no direct comparisons can be made between their results and those of the investigators on chick brains discussed previously because in addition to the difference in species, the samples were actually exposed (with a duty factor of 0.32) for only a third of the 20-min period (at correspondingly higher peak levels), and because the spectral energy distribution for the 16-pps repetition rate differed markedly from that in the sidebands for 16-Hz amplitude-modulated RFR.

Merritt et al. (1982) performed experiments *in vivo* as well as *in vitro* on possible pulsed-RFR-induced alterations of calcium efflux from the rat brain. For both types of experiment, brain tissue was loaded with $^{45}Ca^{++}$ by injection directly into the right lateral ventricle of ether-anesthetized rats.

In the *in vitro* experiments, the rats were euthanized by cervical dislocation after intraventricular injection of $^{45}Ca^{++}$. Six samples of brain tissue excised as in the previous study were concurrently exposed for 20 min to 20-ms pulses, 16 pps, of 1-GHz RFR at 1 or 10 mW/cm^2 (SAR 0.29 or 2.9 W/kg) or of 2.45-GHz RFR at 1 mW/cm^2 (SAR 0.3 W/kg), with a like number of samples sham-exposed as controls.

Although the radioactivities of the incubation media and the tissue samples were assayed by liquid scintillation counting, the results were not given in terms of the efflux values as defined in Shelton and Merritt (1981); instead, the mean disintegrations per minute per gram of tissue (DPM/g) and SDs were presented. By two-tailed t-test, the difference in mean DPM/g values between RFR- and sham-exposed samples was not significant for any of the three exposure conditions.

For the whole-animal exposures, 2 h after the rats were injected with $^{45}Ca^{++}$, each rat was gently squeezed between two sides of a holder to keep its body axis constant during exposure, and groups of 12 rats each were exposed for 20 min to 2.06-GHz RFR with their long axes parallel to the E-field. One group each was exposed to CW at 0.5, 1.0, 5.0, or 10.0 mW/cm^2, and one group each to 10-ms pulses at 8, 16, or 32 pps and average power density of 0.5, 1.0, 5.0, or 10.0 mW/cm^2 (16 groups total). A seventeenth group was sham-exposed. By calorimetry with rat models, the normalized SAR was 0.24 W/kg per mW/cm^2, yielding 0.12, 0.24, 1.2, and 2.4 W/kg for the average power densities above.

After exposure, the rats were euthanized, and their brains were removed and processed appropriately for $^{45}Ca^{++}$ assays. Statistical tests on the 17 treatment combinations (4 x 4 RFR-exposures, 1 sham-exposure) showed that the difference between the sham group and the combined RFR groups and the differences between the sham group and the individual RFR groups were nonsignificant.

This paper, like the previous one, described an attempt to determine whether changes in calcium efflux reported to be induced in chick brains by *in vitro* exposure to amplitude-modulated RFR might also be seen in rats given *in vitro* or *in vivo* exposure to pulse-modulated waveforms. No RFR-induced calcium-efflux changes were found. However, in addition to the differences previously noted about species, carrier frequency, and waveform, other important

ones occurred – most notably that the brain was loaded with $^{45}Ca^{++}$ by injection into the right ventricle of the brain of the intact animal, whereas external bathing media containing $^{45}Ca^{++}$ were used in the other studies.

Adey et al. (1982) presented the results of a study of $^{45}Ca^{++}$ efflux from the cortex of the paralyzed but awake cat. A hole centered over the right cortex had been drilled under ether anesthesia. The dura was removed and a plastic well was fitted into the aperture to make gentle contact with the surface of the pia. Nonradioactive physiologic medium was added to the well and all skin incisions, and pressure points were anesthetized locally. The use of ether was then discontinued, the cat was paralyzed, and artificial respiration was maintained with a tracheotomy.

During recovery from ether anesthesia, the fluid level in the well was replaced at 10-min intervals for 30 min, to ensure that the fluid was clear. Incubation was then begun with medium that contained $^{45}Ca^{++}$, and was continued for 90 min, at the end of which the fluid was replaced with a nonradioactive medium. At 10-min intervals during the remainder of the experiment, the fluid was exchanged completely with fresh medium, and aliquots of each solution were removed and assayed for $^{45}Ca^{++}$.

Starting at different times after completion of incubation of the cortex with $^{45}Ca^{++}$, 23 female cats were individually sham-exposed or exposed for 60 min to 450-MHz RFR amplitude-modulated at 16 Hz. Differences in efflux patterns were sought at intervals ranging from 80 to 120 min. Each cat was placed in a plastic stereotaxic headholder, with its body axis normal to the incident field and with the right cerebral cortex nearest the RFR source. The average power density was 3.0 mW/cm^2, for which the electric field within the interhemispheric fissure was found to be 33 V/m, corresponding to an SAR of 0.29 W/kg.

The description of the data-analysis methods used was obscure. The authors fit relative $^{45}Ca^{++}$ efflux data taken at 10-min intervals in the absence of the RFR by a time-dependent equation involving the sum of 2 exponential terms. They therefore log-transformed the data and used linear regression to obtain an idealized curve of relative efflux versus time. They then quantified the results for RFR-exposed cats in terms of the means of the relative squared deviations of experimental data from the idealized curve at sampling points (rather than comparing the slope of the regression line for the RFR-exposed cats with the slope of the idealized curve), and similarly for the sham-exposed cats. Presumably this was done because the mean values for the RFR-exposed and sham-exposed cats were comparable, but the SDs for the former were larger than for the latter.

Next, the authors paired experimental curves of relative $^{45}Ca^{++}$ efflux (defined in terms of relative squared deviations) versus time from RFR- and sham-exposed cats. They graphed the data for a representative pair of cats – one exposed to RFR for 60 min, starting after 90 min of preexposure measurements, and the other similarly sham-exposed. By eye, the average slopes of the two curves were about the same up to about the first 60 min of the preexposure measurement period. From about 60 min onward, which included the 60-min interval of RFR- or sham-exposure and afterward, both slopes were smaller, but the average slope for the RFR-exposed cat was less negative than for the sham-exposed cat, and showed cyclic variations ("waves of increased $^{45}Ca^{++}$ efflux") with a periodicity of about 25 min.

No experimental data for RFR- and sham-exposed animals were given (other than mention of their use in the curve-fitting method noted above) or were directly compared for statistically

significant differences. The absence of such data seemed to imply that the important differences in calcium efflux for the RFR- and sham-exposed animals were in the mean-squared deviations from the ideal and the cyclic variations about the idealized linear fits to the data, and not between the values themselves at corresponding times or their means.

B.3.5 Immunology and Hematology

Many reports indicate that RFR has specific effects on the immune systems of mammals. Most reported effects were detected after exposure at power densities of about 10 mW/cm^2 and higher; a few effects have been found from exposure to levels as low as about 0.5 mW/cm^2. In most of the studies, the mechanisms for the effects were not investigated, and many of the results were not consistent with one another. Representative studies are discussed in this section under appropriate topics.

B.3.5.1 *In Vitro* Studies

Early studies were performed *in vitro* to determine whether exposure to RFR can stimulate lymphocytes (one type of leukocyte or white blood cell) to become lymphoblasts, (i.e., active in mitosis – cell division), and to undergo mitosis. In such studies, samples of lymphocytes taken from the body were cultured, exposed to RFR (or exposed and then cultured), and examined for effects induced by the RFR. Usually, such cells were cultured with a mitogen, an agent that can stimulate transformation into lymphoblasts and mitosis. In recent studies, more subtle effects on various types of leukocytes were sought. Also sought were effects of exposure *in vitro* on red blood cells (erythrocytes).

B.3.5.1.1 Leukocyte Studies

Smialowicz (1976) exposed suspensions of mouse-spleen cells to 2.45-GHz RFR at 10 mW/cm^2 (a SAR of about 19 W/kg) for 1, 2, or 4 h. Similar suspensions held at 37°C (without RFR exposure) for the same durations served as controls. After treatment, specimens were cultured with and without each of four different mitogens. No significant differences were seen between RFR-exposed and control specimens treated for corresponding durations. This was true for specimens not stimulated with mitogen, as well as those stimulated with any of the four mitogens. The temperature and percentage viability of specimens immediately after each treatment were also measured, and no significant differences were seen between exposed and control samples for each treatment period.

Hamrick and Fox (1977) exposed cultures of rat lymphocytes with and without a mitogen (PHA) to 2.45-GHz RFR at 5, 10, or 20 mW/cm^2 (0.7, 1.4, or 2.8 W/kg) for 4, 24, or 44 h, and assayed them for lymphoblast transformation by the cellular uptake of tritium-labeled thymidine. The differences in thymidine uptake between mitogen-stimulated cultures and non-stimulated cultures for each RFR level and duration were large, but the differences between RFR- and control cultures were nonsignificant.

Roberts et al. (1983) exposed human mononuclear leukocyte cultures to 2.45-GHz RFR for 2 h at 4 W/kg, with no attempt to remove heating due to the RFR. Other cultures were sham-exposed, and untreated cultures were controls. The three groups of cultures were assessed for viability on days 1 through 7 after treatment, and assayed daily for DNA, RNA, and total protein synthesis. Mean viability of all three groups increased and decreased with time, but the

differences among the groups were not significant. Similar results were obtained at 0.5 W/kg and intermediate SARs. There were also no significant differences among the groups in DNA, RNA, and total protein synthesis.

The three groups were also assayed for spontaneous production of interferon, influenza-virus-induced production of alpha-interferon, and mitogen-induced production of gamma-interferon. Spontaneous production of interferon did not occur. Most of the virus-induced alpha-interferon was present by 24 h in the RFR group and the other two groups, with no significant differences among the three groups. The mitogen-induced gamma-interferon, usually produced in 48 to 72 h, was found in all stimulated cultures by 72 h, with no significant differences among the three groups.

In a later study, Roberts et al. (1987) infected human mononuclear leukocyte cultures with influenza virus and then exposed them to 2.45-GHz RFR, either CW, or pulsed at 60 or 16 Hz (duty cycle 0.5), all at 4 W/kg. Control cultures were sham-exposed. No significant differences due to RFR exposure relative to sham exposure were found in leukocyte viability of virus-infected leukocyte or uninfected cultures, or in DNA synthesis from mitogen stimulation.

Lyle et al. (1983) sought effects for 60-Hz-amplitude-modulated 450-MHz RFR at 1.5 mW/cm^2 (SAR not stated) on the toxicity of certain rodent T-lymphocytes (effector cells) against lymphoma cells (target cells) of a specific type. Exposure of specific mixtures of effector and target cells inhibited the cytotoxicity obtained in control mixtures by about 20%. Similar suppression percentages were seen in assays conducted in the absence of the RFR but in which the effector cells had been exposed before mixing them with the target cells. The authors therefore surmised that the cytotoxicity was due to the action of the RFR on the effector cells. They also found that the percentage of cytotoxicity inhibition diminished with elapsed time after exposure.

Those authors also performed similar cytotoxicity assays for 450-MHz RFR modulated at 0 (unmodulated), 3, 16, 40, 80, and 100 Hz, and compared the results with those at 60 Hz. Cytotoxicity inhibition was negligible with unmodulated RFR, maximal with 60 Hz, and significant (but smaller) with 80 and 100 Hz.

Sultan et al. (1983a) studied the effects of combinations of RFR with hyperthermia on the antigen-antibody activity of B-lymphocytes. Suspensions of normal mouse B-lymphocytes were exposed to 2.45-GHz RFR at levels in the range 5-100 mW/cm^2 (2.25-45 W/kg) at temperatures in the range 37-42.5°C. Suspensions at each temperature were sham-exposed as controls. Antigen-antibody activity was seen in more than 90% of cells exposed to RFR and heat-treated at 37°C, but in less than 60% of cells treated at 41°C, and in less than 5% of those treated at 42.5°C. The authors concluded that the mechanisms responsible for inhibition of such activity are thermal in origin, with no apparent effects of 2.45-GHz RFR if exposed and control samples were held at the same temperature.

Sultan et al. (1983b) reported similar results with cell suspensions exposed to 147-MHz RFR amplitude-modulated at 9, 16, or 60 Hz at levels in the range 0.1-48 mW/cm^2 (0.004-2.0 W/kg). Again, activity inhibition increased with temperature, and no significant differences were found between exposed and control specimens held at the same temperature.

Cleary et al. (1985) exposed rabbit neutrophils (another type of leukocyte) to 100-MHz CW RFR for 30 or 60 min at field strengths ranging from 250 to 410 V/m (120 to 341 W/kg), or for

60 min to 100-MHz RFR amplitude-modulated at 20 Hz (331 W/kg), within a temperature-controlled coaxial exposure chamber. For controls, they sham-exposed other samples and kept still others outside the exposure chamber at the same temperature as the sham-exposed samples. Results showed that the viability and phagocytotic ability of the neutrophils were not affected by such RFR exposures (i.e., no significant differences were found among the groups for each exposure regimen). However, these negative findings are questionable because of the relatively large variabilities among the two control groups in each case, an indication of the possible presence of uncontrolled non-RFR factors.

Kiel et al. (1986) sought effects of RFR on the nonphosphorylating oxidative metabolism of human peripheral mononuclear leukocytes (mostly lymphocytes). They noted that production of active oxygen metabolites is accompanied by generation of chemiluminescence (CL), and that CL can be enhanced by the addition of luminol and used as a sensitive detector of such effects.

Samples of peripheral blood from human volunteers were collected, and the leukocytes were separated, washed, and resuspended. Pairs of 1-ml aliquots within nitrocellulose tubes were used. In the RFR-exposure experiments, one aliquot of each pair was exposed for 30 min to 2.45-GHz CW RFR at 104 W/kg with the sample held at 37°C, and the other was held at 37°C in an incubator. Pairs of aliquots were used similarly for sham-exposure. RFR exposures were done for samples from 9 volunteers, and sham exposures from 6 volunteers.

Following treatment, half of each sample was used for measuring CL activity, and the other half for determining cell counts and viability. After the addition of luminol at time t = 0, CL was monitored at 10-s intervals from t = 40 to t = 180 s and the sum of those values divided by the cell concentration was taken as the total CL for each sample. The results were normalized in terms of a stimulation index (SI) for each treatment, defined as the ratio of the difference between treatment and paired control samples in total CL to the total CL in the control.

The authors noted that the CL variability among the 15 donors was large, but that nevertheless, the CLs for both the RFR- and sham-exposed samples significantly exceeded those of their respective incubator-held controls. The mean SIs for the RFR- and sham-exposed samples were 1.58± 1.38 (SD) and 1.86 ± 1.01, respectively. However, the difference in mean CL between the RFR- and sham-exposed samples was not significant. The authors ascribed the differences between treated and incubated samples to the slow rate of heating in the incubator; a rise from 22° to 36°C in the incubator took 39 min.

B.3.5.1.2 Erythrocyte Studies

In various studies of RFR interactions with *in vitro* samples of red blood cells (RBCs) taken from animals or humans alterations were sought in cell membrane function, and particularly effects on the movement of sodium ions (Na^+) and potassium ions (K^+) across the membrane.

In early Eastern European studies, the authors reported increased hemolysis (cell breakdown) and efflux of K^+ from rabbit RBCs exposed to 1-GHz or 3-GHz RFR at levels as low as 1 mW/cm^2. In a later U.S. study, Peterson et al. (1979) heated suspensions of rabbit RBCs conventionally or with 2.45-GHz RFR at 10-140 mW/cm^2 (46-644 W/kg). The suspension temperatures were monitored continuously, and each suspension was assayed for the loss of hemoglobin (Hb) and K^+ after either treatment. Higher losses of Hb or K^+ were observed for RFR-exposed suspensions than for conventionally heated suspensions. However, in all

experiments in which RFR-heated and conventionally heated erythrocytes were warmed at the same rate to the same final temperature, both Hb and K^+ were lost in equal amounts, indicating that the effect was thermal.

Those U.S. authors also heated samples of human RBCs to 37°C by exposing them to 2.45-GHz RFR at 90 mW/cm^2 (412 W/kg) for 8 min and then maintained them there for 37 min by exposure at 30 mW/cm^2 (137 W/kg). Unlike the results for rabbit RBCs, no significant differences among the groups were obtained in either hemolysis or K^+ release. Such absence of hemolysis and K^+ release for human RBCs can be taken as an indication that RFR-induced changes in rabbit blood may not be reflected in similar effects with human blood.

Brown and Marshall (1986) sought nonthermal effects of RFR on growth and differentiation of the murine erythroleukemic (MEL) cell line. Because they noted that in response to an inducer (hexamethylene bisacetamide – HMBA), MEL cells form Hb and exhibit other forms of erythroid differentiation, they exposed tubes of HMBA-cultured MEL cells for 48 h to 1.18-GHz RFR at an SAR of 18.5, 36.3, or 69.2 W/kg; incubation temperature was held at 37.4°C. Control cultures were held at the same temperature in a water bath.

The growth of exposed and control cultures was compared by measuring the elapsed times for cells to double in number; cell differentiation was compared by counting the percentages of cells stained by a hemoglobin-specific dye and by determining the amounts of hemoglobin produced. The results showed no significant differences in any of the three endpoints among the cultures exposed at each RFR level and their corresponding control cultures. Moreover, the mean values for each endpoint did not vary significantly with RFR level.

B.3.5.2 *In Vivo* Studies: Effects of Exposures on Immunological Parameters

Studies of immunological effects of RFR *in vivo* can be divided into those in which changes in specific immunological parameters were sought – the subject of this section – and those in which effects of RFR on the health of the subjects and their resistance to disease were examined, a topic discussed in the next section.

Huang et al. (1977) exposed groups of hamsters to 2.45-GHz RFR at levels in the range of 5-45 mW/cm^2 (2.3-20.7 W/kg). Blood samples were drawn 1 h after exposure and cultured with or without the mitosis stimulant (mitogen) PHA. The cultures not stimulated with PHA exhibited rises in percentage of transformed cells with increasing RFR level, with the largest changes at 30 mW/cm^2 (13.8 W/kg) followed by gradual return to control values. The cell counts at blood collection time showed no net lymphocyte increase from other sources such as lymph nodes or spleen. There were also no significant changes in differential leukocyte counts, thus supporting the contention that RFR does not cause lymphocytosis.

For PHA-stimulated cultures, the authors found that the mean value of the mitotic index (i.e., the percentage of cells in mitosis relative to the total number of lymphocytes) was 3% for controls but diminished significantly for the groups exposed at 30 and 45 mW/cm^2 (13.8 and 20.7 W/kg). The scatter of values, which was large for the controls, decreased rapidly with increasing RFR level, tending to further confirm that RFR inhibits mitogen-stimulated mitosis. Even at 5 mW/cm^2 (2.3 W/kg), the scatter was still sizable but smaller than for controls, an indication of the thermal basis for the effect.

Huang and Mold (1980) exposed mice to 2.45-GHz RFR at 5-15 mW/cm^2 (3.7-11 W/kg) 30 min/d for 1 to 17 days, after which spleen cells were cultured with or without either a T-lymphocyte mitogen or a B-lymphocyte mitogen. The radiotracer tritiated thymidine was added during culturing. After culturing, the cells were assayed for thymidine uptake, a measure of DNA synthesis during cell proliferation.

Plots of thymidine uptake versus exposure duration showed responses that varied cyclically with time for cells from both mitogen-stimulated and nonstimulated cultures. However, similar plots for sham-exposed mice also showed cyclical fluctuations, apparently due to factors other than RFR. Therefore, whether RFR *per se* has cell-proliferative effects could not be ascertained in this study. In another part of the study, exposure at 15 mW/cm^2 (11 W/kg) for 5 days (30 min/d) did not diminish the cytotoxic activity of lymphocytes on leukemic cells injected after, or concurrently with, the last exposure.

Lin et al. (1979) exposed mice to 148-MHz RFR at 0.5 mW/cm^2 (0.013 W/kg) for 10 weeks beginning on postpartum day 4, 5, 6, or 7. Control mice were sham-exposed. The exposed and control mice were weighed daily during the exposure period, and then weekly up to age 600 days. The mean weights of the two groups at corresponding times did not significantly differ. Blood was drawn at ages 28 and 70 days (4 and 10 weeks) and at ages 100, 250, 300, 360, and 600 days. No significant differences were found between the RFR and control groups for hematocrit, hemoglobin, leukocyte counts, erythrocyte counts, or differential blood-cell counts.

Wiktor-Jedrzejczak et al. (1977) exposed mice to 2.45-GHz RFR at 14 W/kg in a single 30-min session or in three such sessions, one per day, three days apart. Control mice were sham-exposed. After exposure, the numbers of T-lymphocytes and B-lymphocytes in the spleens of the two groups were compared. The total T-lymphocyte population was unaffected by either the single-session or triple-session exposures. However, the single sessions significantly increased the population of one subclass of B-lymphocytes (complement-receptor-positive, CR$^+$) but not of another subclass (immunoglobulin-positive, Ig$^+$), whereas the triple-session exposures yielded increases in both B-cell subclasses.

Next, spleen cells from RFR- and sham-exposed mice were cultured with various T-cell or B-cell mitogens, and the numbers of cells in lymphoblastic transformation were determined. Both single and triple exposures resulted in significant increases in blastic transformation of B cells but nonsignificant effects on T cells.

Last, mice were inoculated with the antigen sheep red blood cells (SRBC) that induces production of antibodies by B cells if T cells are also present, or with another antigen that does not require the presence of T cells for antibody production by B cells. The mice were then given triple-session RFR- or sham-exposures, after which their spleens were assayed for antibody production. RFR-induced decreases of antibody production in response to both antigens were observed, but only the difference for SRBC was statistically significant. Taken together, the results of this study show that thermogenic RFR levels (e.g., 14 W/kg) can have weak stimulatory effects on splenic B cells but none on T cells.

Sulek et al. (1980) found that the threshold for increases in CR$^+$ B cells was about 5 W/kg for 30-min exposures to 2.45-GHz RFR, yielding an energy-absorption threshold of 10 J/g. They also found that multiple exposures at levels below the threshold were cumulative if done within 1 h of one another, but not if spaced 24 h apart, even if the sum of the energy-absorption values

exceeded the threshold. Other authors obtained results indicating that the RFR-induced increases in CR^+ B cells depend on genetic factors; other authors could not confirm the RFR-induced CR^+ increases discussed above.

Liburdy (1977) exposed mice for 15 min to 26-MHz RFR at 80 mW/cm^2. This RFR level produced core-temperature rises of 2-3°C, and was called "thermogenic" by the author. The corresponding whole-body SAR was 5.6 W/kg, more than tenfold higher than the 0.4-W/kg basis for the 1982 ANSI standard. For comparison, other mice were heated in an oven at 63°C for the same period to obtain the same rise in core temperature.

Lymphopenia (decrease in lymphocyte count) and neutrophilia (rise in neutrophil count) were seen in the RFR-exposed mice, which persisted for about 12 h after exposure. Those effects could be sustained and the recovery period prolonged by more RFR exposures at 3-h intervals. By contrast, the oven-heated mice exhibited only slight effects. Injection of corticosteroid as a positive control yielded a similar time course for lymphopenia and neutrophilia, but it also led to a decrease in the total leukocyte population.

The effects above were absent for mice exposed to 26-MHz RFR at 50 mW/cm^2 or to 5-MHz RFR at 800 mW/cm^2, called "nonthermogenic" RFR, both of which yielded a whole-body SAR of 0.36 W/kg or about 1/16 of the SAR used previously. (This SAR is about the same as the basis for the 1982 ANSI standard.)

Smialowicz et al. (1981) exposed 16 rats almost continuously for 69-70 consecutive days to 970-MHz RFR at 2.5 W/kg. Another group of 16 rats was similarly sham-exposed. Blood samples were taken from 8 rats of each group on day 69, after which their spleens were removed. The other rats were treated similarly on day 70.

No significant differences were found between RFR- and sham-exposed rats in erythrocyte count, total or differential leukocyte counts, mean cell volume of erythrocytes, Hb concentration, or hematocrit. Nor did spleen cells removed from RFR- and sham-exposed rats and cultured with various mitogens exhibited significant differences in responses. However, blood serum analysis yielded significantly higher concentrations of triglyceride, albumin, and total protein for the RFR group. Those higher albumin and protein concentrations were within the normal ranges for this strain of rat and not consonant with the absence of changes in erythrocyte assays, indicating that the rats may have been dehydrated.

The authors noted that an SAR of 2.5 W/kg is about half the basal metabolic rate of an adult rat, and they suggested that the increases in triglyceride level may have been due to thermal stress induced by RFR exposure. At 970 MHz, there probably were regions within the rat where local SARs were much higher than 2.5 W/kg, and such higher SARs could have affected the endocrinologic system.

Smialowicz et al. (1982a) exposed pregnant mice to 2.45-GHz RFR at 28 mW/cm^2 (16.5 W/kg) for 100 min daily from gestation day 6 to day 18. At 3 and 6 weeks of age, the pups were assessed for development of primary immune response to the antigen SRBC, proliferation of lymphocytes *in vitro* by stimulation with mitogens, and *in vitro* activity of natural killer (NK) cells against lymphoma cells. No consistent significant differences were found between RFR- and sham-exposed mice in any of the endpoints.

Smialowicz et al. (1982b) exposed mice to either CW or pulsed 425-MHz RFR at SARs up to 8.6 W/kg. No differences in mitogen-stimulated responses of lymphocytes or in primary antibody response to sensitization with SRBC or another antigen (PVP) were observed between RFR- and sham-exposed mice, or between mice exposed to the CW- or pulse-modulated RFR.

Smialowicz et al. (1983) exposed groups of mice for 1.5 h/d on 9 consecutive days to 2.45-GHz RFR at several levels. For positive controls, other mice were injected with either hydrocortisone or saline. Splenic cells were then assayed *in vitro* for NK-cell activity by their cytotoxicity against mouse-lymphoma cells. Significant suppression of NK-cell activity was seen for 30 mW/cm^2 (21 W/kg), but such activity returned to normal within 24 h after the last RFR exposure. However, this transient effect was not seen at 15 or 5 mW/cm^2 (10.5 or 3.5 W/kg).

NK-cell activity was also assayed *in vivo*. Suppression of activity was seen in mice exposed at 30 mW/cm^2, but with a return to normal activity several days after the last exposure. Hydrocortisone injection caused activity suppression of both *in vitro* and *in vivo*.

Ortner et al. (1981) exposed groups of rats to 2.45-GHz RFR for 8 h at 2 or 10 mW/cm^2 (0.44 or 2.2 W/kg). A sham-exposed group served as controls. Within 5-15 min after treatment, peritoneal mast cells were extracted from rats of each group, and mitogen-induced histamine releases therefrom were determined. The results showed no significant differences among the three groups in percentage of cell viability, percentage of cells, amount of histamine per cell, and cell diameter.

For groups of rats similarly exposed, the total red and white cell counts were not affected by 8-h exposure at either level, nor were blood-hemoglobin levels or percentages of lymphocytes and neutrophils relative to those of the sham group. The other types of cells were also unchanged by the RFR. Serum biochemistry parameters were not affected by either level.

Wong et al. (1985) noted that relatively few studies had been done in the HF band (3-30 MHz), that most such studies showed that thermogenic levels of RFR were necessary for significant effects of acute exposure, and that possible effects of prolonged exposure to low RFR levels in that frequency range had not been investigated. To address the last issue, they conducted two experiments with rats at 20 MHz.

In the first experiment, 200 rats were caged in 40 groups of 5 rats; 20 of the groups were exposed to 20-MHz RFR at 1920 mW/cm^2 (about 0.3 W/kg) for 6 h/d, 5 d/wk, and the other 20 groups were sham-exposed as controls. After 8 days of treatment, 6 groups each of exposed and control rats were euthanized and examined for histopathology. This was also done for 7 groups each after 22 days and for the remaining 7 groups each after 39 days.

In the second experiment, 24 rats were divided into exposed and control groups of 12 each, but each rat was housed separately and all rats were euthanized after 6 weeks. On termination, blood samples were collected and the routine counts and blood-chemistry assays were performed. The spleens were excised and weighed, and suspensions of spleen cells were prepared. Various other tissues were also examined for histopathology.

The first experiment yielded a significantly higher mean RBC count and a significantly lower mean Hb content for the rats terminated after 39 days of exposure than for the control group, but the statistical analysis showed that those differences were not RFR-related. In the second experiment, however, no significant differences were seen in RBC count, Hb, or any of the blood-chemistry parameters.

In regard to histopathology, the authors noted that rats examined for quality control before the start of the first experiment were normal, but by day 8, pulmonary congestion and edema, rhinitis, and peribronchiolar and pulmonary perivascular lymphoid proliferation were evident. Emphysema and incomplete lung expansion were also present in some rats killed on day 22. However, no pattern of lesions could be associated with RFR exposure. Focal disseminated pneumonia was present in 4 exposed and 4 control rats of the first experiment euthanized on day 39, but of the 200 rats used in that experiment, only 1 had any clinical signs of illness. All 24 rats in the second experiment were histologically normal at the end of the 6-week period of exposure.

B.3.5.3 *In Vivo* Studies: Effects of Chronic Exposure on Health, Longevity, and Resistance to Disease

Relatively few investigations have been undertaken to determine whether or not chronic RFR exposure affects the general health or longevity of animals, or alters the resistance to, or the severity of, diseases accidentally acquired or purposely given to animals. Representative examples of such investigations follow.

Prausnitz and Susskind (1962) exposed 200 male mice in groups of 10 for 4.5 min/d, 5 d/wk, for 59 weeks to 9.3-GHz pulsed RFR at 100 mW/cm^2 average power density (roughly 45 W/kg). Exposures of that duration yielded a mean rise in body temperature of 3.3°C. For a test group, the rise that caused death in 50% of the mice (LD_{50}) was 6.7°C, attained in 12 min at 100 mW/cm^2. Thus, exposure for 4′ min at 100 mW/cm^2 was sublethal. Controls were 100 sham-exposed mice.

Some deaths occurred in both groups during the exposure series and were attributed to a pneumonia infection introduced accidentally into the colony during the experiment. However, the death rate was found to be higher in the sham-exposed mice than in the RFR-exposed mice: On completion of the series, 50% of the control mice and 65% of the RFR-exposed mice were still alive. The authors ascribed the better survival of the exposed mice to the protective effect of the daily rise in temperature ("fever") induced by the RFR. That explanation seems plausible, but is not proven. Among the results of tissue examination were liver abscesses in some mice, but because of tissue breakdown (autolysis), the relative incidence in RFR-exposed and control mice could not be determined.

The authors reported that some mice had developed leukosis [also spelled leucosis], which they described as a "cancer of the white blood cells," and that leukosis incidence was higher in the exposed than the control mice. This effect was real, but the interpretation by the authors was probably faulty. In dictionaries of medicine and pathology, leukosis is defined as an abnormal rise in the number of circulating white blood cells, and is not regarded as a form of cancer. Various factors can give rise to leukosis, including stress, disturbances of the endocrine system, and infections such as pneumonia. The stated presence of pneumonia in the mouse colony may have caused the observed liver abscesses.

Roberts and Michaelson (1983) reanalyzed the data of Prausnitz and Susskind (1962) with appropriate statistical treatment. They found that the results do not support a link between exposure to RFR and cancer development. They also remarked that the greater longevity of the RFR-exposed mice could be taken equally plausibly as indicating that the RFR was beneficial.

Szmigielski et al. (1980) exposed mice to CW or pulsed 2.45-GHz RFR at 5 or 15 mW/cm^2 (2-3 or 6-9 W/kg) 2 h/d for 6 or 12 weeks before injecting the mice with staphylococcal bacteria at a dose selected to yield a 3-day survival rate of 60% for control mice. For the mice exposed at 5 mW/cm^2 for 6 weeks before injection, the survival rate was 80%. For those exposed at the same RFR level for 12 weeks, the survival rate was 45%. The differences among the two RFR groups and the control group were not significant. The survival rates for those exposed at 15 mW/cm^2 for 6 or 12 weeks were only 25% and 5%, respectively.

Liddle et al. (1987) sought effects of exposure to RFR at various ambient temperatures on the survival of mice given an LD_{50} dose of another type of *Staphylococcus*. They injected mice with that *Staphylococcus* bacterium and exposed them to 2.45-GHz RFR at 10 mW/cm^2 (6.8 W/kg) for 5 days (4 h/d) at 8 ambient temperatures in the range 19-40°C and 50% relative humidity. Equal numbers of mice were injected and sham-exposed.

In the temperature range 19-31 °C, the percentages of RFR-exposed mice that survived the *Staphylococcus* challenge were significantly higher than for the corresponding sham-exposed mice. Above 31 °C, the survival values of the RFR-exposed mice dropped sharply, to 0% at 37 °C. Similar results were seen for the sham-exposed mice above 34 °C; survival dropped to 0% at 40°C. The results also indicated that most of those deaths were due to hyperthermia, and showed that exposure to RFR may be beneficial to infected animals at low and moderate ambient temperatures, in consonance with the findings of several other studies.

In the University of Washington study discussed in Section 3.2.3, a group of 100 male rats was exposed unrestrained to 2.45-GHz RFR at an average power density of about 0.48 mW/cm^2 in individual cylindrical waveguides under controlled-environmental and specific-pathogen-free conditions. Another group of 100 male rats was similarly sham-exposed. The exposures were started at age 8 weeks and continued for 25 months. At 13 months, 10 each of the RFR- and sham-exposed rats were euthanized (the interim kill), as were 10 of the 12 RFR-exposed and 10 of the 11 sham-exposed rats that survived the 25-month regimen.

Suspensions of splenic cells from each group were assayed for populations of T cells and B cells, complement-receptor-positive (CR^+) cells, and plaque-forming cells in response to immunization with SRBC versus saline. Such suspensions were also stimulated with various mitogens, and the stimulation index relative to unstimulated cultures was determined.

The results for the interim kill showed significantly higher counts of splenic T- and B-lymphocytes for the RFR-exposed rats than the sham-exposed rats, indicating that the RFR had stimulated the lymphoid system. By contrast, however, there were no significant differences in T- and B-cell populations between the RFR and sham groups of the terminal kill, a possible indication of the onset of immunosenescence.

The CR^+ values for the RFR groups of both the interim and terminal kills were lower than for the sham groups, but the differences were not significant, indicating no differences between RFR and sham groups in lymphocyte maturation. The percentages of plaque-forming cells for the SRBC-immunized rats were nonsignificantly higher for the RFR group than the sham group in the interim kill, but was nonsignificantly lower in the terminal kill.

The mitogen-stimulation results for the RFR group of the interim kill showed higher responses than the sham group to T-cell mitogens PHA and Con A, but only the difference for Con A was significant. The RFR group's responses to the B-cell mitogens LPS and PPD were

significantly higher and lower, respectively, than for the sham group. The RFR group also yielded a significantly higher response to the nonspecific mitogen pokeweed than the sham group. No mitogen-stimulation results could be obtained for the terminal kill because the lymphocyte cultures failed to grow and respond to any of the mitogens.

Blood samples drawn periodically from all of the rats were analyzed. The samples were taken under light anesthesia to avoid stress-induced corticosterone elevation. The first samples were drawn 4 weeks before the start of the exposure regimen to provide baseline data. The other samples were taken after 7 weeks of exposure, then at subsequent 6-week intervals during the first year of exposure, and at 12-week intervals during the second year of exposure. However, the numbers of rats sampled decreased with time because of the withdrawals and mortality.

The blood samples were assayed for various hematologic parameters and serum chemistry. Multivariate analyses revealed no overall significant differences between the RFR- and sham-exposed rats in regard to hematologic parameters. Differences in thyroxine (T_4) levels between the RFR- and sham-exposed rats were nonsignificant, indicating that the RFR had no effect on the hypothalamic-pituitary-thyroid feedback mechanism. As expected, however, T_4 levels of both groups decreased significantly with age.

Overall, no significant differences were found between the RFR- and sham-exposed rats at periodic behavioral test sessions. However, as discussed in Section 3.2.3, primary malignant lesions of several types were found in both groups, with the numbers of rats exhibiting each type of malignancy similar to those reported in the literature for untreated rats of the same strain, and the differences in numbers of rats for each specific type of malignancy all statistically nonsignificant.

Adding the numbers for each malignancy type without regard for age yielded totals of 18 RFR-exposed and 5 sham-exposed rats, a statistically significant difference. However, grouping the different malignancy types in that manner is not considered valid by most members of the toxicology community.

Toler et al. (1988) implanted cannulas in the aortas of 200 male white rats. After the rats recovered, 100 of them were concurrently exposed to 435-MHz pulsed RFR (1-μs pulses at 1000 pps) in a special facility, described by Bonasera et al. (1988). The facility consisted of four circular parallel-plate waveguides stacked vertically. Each such waveguide radiated horizontally outward in the TE_{10} mode (with horizontal polarization). Placed around the periphery of each waveguide were 25 rats in individual cages that permitted unfettered movement.

Exposures were at 1 mW/cm^2 average power density for about 22 h/d, 7 d/wk, for 6 months. Because of rat movements, the whole-body SARs varied with time, ranging from 0.04 to 0.4 W/kg, with a mean of about 0.3 W/kg. An identical stack of waveguides was used for concurrent sham-exposure of the other 100 rats.

Small samples of blood were drawn cyclically without restraint or anesthesia and assayed for the stress hormones ACTH, corticosterone, prolactin in the plasma, for plasma catecholamines, and for hematologic parameters, including hematocrit and various blood cell counts. Also monitored were heart rates and arterial blood pressure. The results showed no significant RFR-induced differences between groups in any of those endpoints.

B.3.6 Physiology and Biochemistry

The literature on physiological and biochemical effects associated with exposure to RFR is extensive. Many of the reported effects were associated with other events (e.g., changes in hormonal levels or stress adaptation), some are questionable for various reasons, and the medical significance of others is unclear.

B.3.6.1 Metabolism and Thermoregulation

Several studies have been done in which primates were exposed to RFR in the HF region (3-30 MHz). Bollinger (1971) exposed rhesus monkeys to 10.5-MHz or 19.3-MHz RFR for successive intervals at successively higher power densities up to 600 mW/cm^2 (SARs of about 0.2 and 0.6 W/kg, respectively), or to 26.6-MHz RFR at up to 300 mW/cm^2 (about 0.6 W/kg). Deep-body temperatures and EKGs were taken during exposure. No obvious indications of thermal stress, heart-rate increases, or other influences on the electrical events of the heart cycle due to the RFR were found.

The author also exposed rhesus monkeys to 10.5- or 26.6-MHz RFR for 1 hour at 200 or 105 mW/cm^2 (0.06 or 0.2 W/kg), or to 19.3-MHz RFR for 14 days (4 h/d) at 115 mW/cm^2 (0.1 W/kg). Hematologic and blood-chemistry analyses were done before and after exposure. There were no significant differences between exposed and control monkeys for most of the cellular components of blood. Significant differences in mean counts of monocytes and eosinophils were seen, but were ascribed to conditions unrelated to RFR. The conclusions were similar for the blood-chemistry parameters. No abnormalities ascribable to exposure were seen in gross pathological and histopathological examinations.

In another study, Frazer et al. (1976) exposed rhesus monkeys to 26-MHz RFR at 500, 750, or 1000 mW/cm^2 (1.0, 1.5, or 2.0 W/kg) for 6 h, during which skin and rectal temperatures were measured. The results showed that the monkeys were in thermal equilibrium even at the highest RFR level; they were able to dissipate the additional heat from the RFR, and their thermoregulatory mechanisms were quite efficient in doing so. The authors noted that exposing a 3.6-kg monkey to 26-MHz RFR at 1000 mW/cm^2 (2.0 W/kg) is equivalent to exposing a human 1.8 m (about 5 ft 11 in.) tall to the same frequency at 400 mW/cm^2.

In a third study, Krupp (1977) exposed rhesus monkeys for 3 h to 15- or 20-MHz RFR at levels in the range 760-1270 mW/cm^2 (0.6-1.3 W/kg). Again, the results showed that the additional heat from the RFR was readily accommodated by their thermoregulatory mechanisms. Exposure to 20-MHz RFR at 1270 mW/cm^2 was calculated to be equivalent to human exposure to 20-MHz at 225 mW/cm^2.

Krupp (1978) conducted a follow-up study on 18 rhesus monkeys that had been exposed 1-2 years previously at least twice to 15-, 20-, or 26-MHz RFR for up to 6 h at levels in the range of 500-1270 mW/cm^2. No RFR-related variations from normal values of hematologic and biochemical blood indices or of physical conditions were found.

Ho and Edwards (1979) used oxygen-consumption rate as an indicator of stress in mice. Mice were exposed to 2.45-GHz RFR in a waveguide system that permitted continuous monitoring of SAR during exposure. The exposures were for 30 min, during which oxygen-consumption rates and SARs were determined at 5-min intervals. The oxygen-consumption rates were converted into specific metabolic rates and expressed in the same units as the SARs (W/kg).

Oxygen-consumption rates were also measured at 5-min intervals for 30 min before and after exposure.

At the highest input power, the mean SAR decreased from 56 to 39 W/kg during 30 min of exposure, and the mean specific metabolic rate decreased from 17.5 to 14 W/kg, thereby decreasing the total thermal burden from 74 to 54 W/kg. Apparently the mice sought to diminish their thermal burdens by altering their body configurations during exposure to minimize the RFR-absorption rates; they also reduced their oxygen consumption. Smaller changes were seen at SARs of 23.6 W/kg and 10.4 W/kg, and insignificant changes at 5.5 and 1.6 W/kg. The specific metabolic rate decreases for SARs of 10.4 W/kg and higher were ascribed to thermal stress, and the onset level was about the same as the basal metabolic rate of the mouse (9 W/kg). After exposure stopped, oxygen consumption rates returned to normal.

To study voluntary thermoregulation in the rat, Stern et al. (1979) trained fur-clipped rats in a cold chamber to press a lever that turned on an infrared lamp. When the rats were exposed to 2.45-GHz RFR for 15-min periods, the rate at which they turned on the lamp decreased as a function of the RFR level, which ranged from 5 to 20 mW/cm^2 (1-4 W/kg). The rats responded to maintain a nearly constant thermal state. In the absence of the RFR, the lamp was the sole heat source. With the RFR present, the rats compensated by reducing their response rate, thereby lowering the IR heat contribution. Thus, voluntary thermoregulation is an indicator of the additional thermal burden of RFR.

Adair and Adams (1980) trained three squirrel monkeys to regulate their environmental temperature (T_a) behaviorally by adjusting the flows of air at various temperatures into an exposure chamber. The monkeys were then exposed to 2.45-GHz RFR for 10-min periods at levels in the range 1-22 mW/cm^2 (0.15-3.3 W/kg). They were also sham-RFR-exposed and exposed to infrared radiation (IR) of equivalent power densities. At an RFR level of about 7 mW/cm^2 and higher, all were stimulated to select a lower T_a. This RFR level represents a whole-body-SAR threshold of 1.1 W/kg or 20% of the resting metabolic rate of the squirrel monkey. The thermoregulatory behavior was highly efficient, and the skin and rectal temperatures remained stable, even at 22 mW/cm^2 (3.3 W/kg), where the preferred T_a was lower by as much as 4°C. Comparable reductions in the T_a selected did not occur for exposure to IR.

Bruce-Wolfe and Adair (1985) investigated the ability of squirrel monkeys to vary the level of 2.45-GHz RFR as a source of thermalizing energy. First, they trained four monkeys to regulate the temperature of the air in the exposure chamber, T_a, by selecting air streams at 10° and 50°C successively (i.e., temperatures below and above the thermoneutral level of about 30°C). The resulting mean T_a was about 35°C.

After stable performance was achieved, the 50°C air source was replaced with 2.45-GHz RFR at 20 mW/cm^2 (3 W/kg) and thermoneutral 30°C air. Thus, only the RFR and air source were activated whenever the monkeys demanded heat, and only the 10°C air source was activated when cooling was demanded. Sessions with RFR at 25 and 30 mW/cm^2 (3.75 or 4.5 W/kg) were also conducted. The results indicated that the monkeys were readily able to use the thermal energy from the RFR for thermoregulation instead of the 50°C air source, and were thereby able to maintain normal rectal temperature.

Adair et al. (1985) examined the effects of long-term RFR-exposure on behavioral and physiological thermoregulation in the squirrel monkey. The exposures were for 15 weeks, 40

h/wk, to 2.45-GHz CW RFR at 1 or 5 mW/cm^2 (0.16 or 0.8 W/kg) at environmental temperatures of 25°, 30°, or 35°C. Fourteen monkeys were trained to select a preferred T_a, and were treated concurrently in fours, one pair each for RFR- and sham-exposure. The pairs used for sham-exposure in a given experiment were RFR-exposed in the next experiment, with 2-3 months between experiments for the dissipation of any residual effects.

Physiologic changes related to thermoregulation were determined periodically before, during, and after such treatment, including body mass, blood properties, skin temperatures, oxygen consumption, colonic temperature, and foot sweating. Each monkey was also trained to select its preferred environmental temperature T_a as in the study above, and their corresponding colonic temperatures and mean skin temperatures were measured.

The results for environmental temperature 25°C or 30°C showed no change in preferred T_a during exposure at 1 mW/cm^2 (0.16 W/kg). At 35°C and 1 mW/cm^2 or at all three environmental temperatures and 5 mW/cm^2 (0.8 W/kg), however, the monkeys selected cooler environments (T_as 1° to 3°C lower). Sweating was higher at 35°C, but was not enhanced by exposure to the RFR at either level. Colonic temperature was not affected, but skin temperature varied with environmental temperature and RFR exposure in an unpredictable way.

Mean body mass during sham-exposure, with proper consideration of seasonal variations, increased at 25° and 30°C, and decreased slightly at 35°C. Decreases in body mass at all three temperatures were found for exposure at 5 mW/cm^2 (0.8 W/kg). However, the authors could not ascribe those decreases to reductions in food intake, because precise measurements of the food and water intake by the monkeys were not possible. No significant changes in blood indices or in other physiological characteristics due to the RFR were found.

Lotz (1985) exposed 5 rhesus monkeys to 225-MHz RFR in the E-orientation (near whole-body resonance) at levels in the range 1.2-15.0 mW/cm^2 (0.8-10.2 W/kg) in an anechoic chamber in 4-hour day and night sessions. The monkeys were also exposed in the H-orientation at 5 mW/cm^2 (1.2 W/kg), and to 1.29-GHz RFR (a frequency well above resonance) at 20, 28, and 38 mW/cm^2 (2.9, 4.0, and 5.4 W/kg). Rectal temperatures were monitored continuously during every session, and blood samples for cortisol analysis were taken hourly during the 225-MHz E-orientation sessions. The criterion for tolerance of RFR was defined as a rectal temperature not exceeding 41.5°C.

Average rectal-temperature increases for exposures to 225-MHz RFR in the E-orientation at 2.5 and 5.0 mW/cm^2 (1.7 and 3.4 W/kg) were 0.4 and 1.7°C, but the monkeys could not tolerate exposure without rectal temperature exceeding the prescribed maximum to 225-MHz RFR in the E-orientation at 7.5 mW/cm^2 (5.1 W/kg) or higher for more than 90 minutes. No changes were observed in circulating cortisol levels for exposures at 5 mW/cm^2 (3.4 W/kg) or less. The exposures to 225-MHz RFR at 5 mW/cm^2 in the E-orientation (3.4 W/kg) and the H-orientation (1.2 W/kg) produced mean rectal-temperature increases of 2.1 and 0.2°C, respectively. The mean rectal-temperature rises for exposures to 1.29-GHz RFR at 20, 28, and 38 mW/cm^2 (2.9, 4.0, and 5.4 W/kg) were 0.4, 0.7, and 1.3°C. These results confirmed that RFR exposure near resonance is most effective for producing hyperthermia.

Lotz and Saxton (1987) studied the vasomotor and metabolic responses of five rhesus monkeys exposed to 225-MHz CW RFR with body axis parallel to the electric component (E-orientation). Two protocols were used. The first protocol was designed to determine the

threshold for the onset of vasomotor responses: after the monkeys equilibrated for 120 min, each was given repetitive 10-min exposures at successively higher power densities until a marked vasomotor response was evidenced by a rapid change in the temperature of the tail skin. After each 10-min exposure, the monkey was allowed to reequilibrate to within 0.3°C of its preexposure value.

RFR levels in the range 1.2-12.5 mW/cm^2 (0.3-3.6 W/kg) were used. The time interval needed for reequilibration varied from about 10 to 60 min, depending on the RFR level. Each monkey was used in 4 such experiments, 2 each at 20° and 26°C.

Results for the first protocol showed that metabolic response occurred before tail vasodilation was manifested (i.e., at a lower RFR level). At 20°C, metabolic heat production was not altered at 1.2 mW/cm^2 (0.3 W/kg) but declined with increasing RFR level. At 26°C, the rate of metabolic heat production before exposure was 28% below that at 20°C, and was not altered by the 10-min exposures. Analysis of the data indicated that the lowest RFR level that reliably altered metabolic heat production during such 10-min exposures was in the range 5-7.5 mW/cm^2 (1.4-2.1 W/kg).

In the second protocol, thermoregulatory adjustments in the steady state during exposure were evaluated. The monkeys were equilibrated for 120 min at 20°C and then given single 120-min exposures at levels in the range 0-10 mW/cm^2 (0-2.9 W/kg). Data were collected for 10-60 min after exposure. The monkeys were also similarly treated at 26°C, but at levels in the range 0-7.5 mW/cm^2 (0-2.1 W/kg).

During the last 30 min of preexposure, the mean metabolic heat production was 2.26 W/kg at 26 °C and 3.13 W/kg at 20 °C; it declined sharply during RFR exposure at 20 °C and remained essentially unchanged during RFR exposure at 26 °C. Also evident was progressive recruitment of metabolic and vasomotor responses at 20 °C. At both ambient temperatures, the mean colonic temperature during the last 30 min of RFR exposure was higher than for the last 30 min of sham exposure, even at 2.5 mW/cm^2 (0.7 W/kg), which was below threshold for thermoregulatory effector action. This result indicated that the thermoregulatory responses could not fully compensate for the heat generated by the RFR even in the cooler environment.

In summary, the thermal basis for various effects of RFR on the autonomic thermoregulatory systems of mammals and on their behavioral thermoregulatory responses to RFR is evident. Especially noteworthy are the results for primates because of their far greater similarities to humans than the other animals studied.

B.3.6.2 Endocrinology

Exposure of mammals to RFR has yielded rather inconsistent effects on the endocrine system. In general, effects are apparently related either to the heat load associated with RFR or to the stress induced by RFR, and possibly to other circumstances.

Cairnie et al. (1980) sham-exposed or exposed unanesthetized mice for 16 h to 2.45-GHz RFR at 50 mW/cm^2 (60 W/kg), after which they were euthanized. Their rectal and testis temperatures versus time after exposure cessation were measured, and the resulting cooling curves were used to determine the temperatures at exposure end. The mean rectal temperature for the RFR group was significantly higher than for the sham group, but the mean testis temperatures of the two groups did not differ significantly, indicating that the thermoregulatory system of the

testes was able to compensate fully for the increased thermal burden from RFR at close to lethal level.

The authors also conducted experiments in which arrays of conscious mice were exposed for various durations to 2.45-GHz RFR at levels in the range 21-37 mW/cm^2, after which testicular cells were examined for damage, and abnormal sperm were counted. The corresponding ranges of whole-body and testicular SARs were 25.3-44.5 W/kg and 8.4-14.8 W/kg, respectively. The results showed no significant differences between RFR- and sham-exposed mice in mean percentages of damaged testicular cells, sperm counts, or percentages of abnormal sperm.

Lebovitz and Johnson (1983) sham-exposed or exposed unanesthetized male rats to 1.3-GHz RFR for 9 days (6 h/d) during a 2-week period. The whole-body SAR was 6.3 W/kg, which produced a mean rise in core temperature of 1.5°C. After treatment completion, groups of rats were weighed and decapitated at intervals corresponding to ´, 1, 2, and 4 cycles of spermatogenesis. Spermatids resistant to homogenization were counted in the right testis, and daily sperm production was calculated. The left testis was processed for examination by light microscopy.

RFR-exposed rats yielded 87.6% normal sperm after a half-cycle of spermatogenesis versus 95.8% for sham-exposed rats. The difference was significant, but most of the abnormal sperm in the RFR group were from 1 rat with 45.5% abnormal sperm, which rendered the finding suspect. There was no significant difference in the weight of seminal vesicles between RFR- and sham-exposed rats, indicating that exposure at 6.3 W/kg was not deleterious to the production of testosterone. This finding was supported by histological evaluations by light microscopy.

Lebovitz and Johnson (1987) subsequently exposed 16 male rats once for 8 h unrestrained in individual cylindrical waveguides to 1.3-GHz CW RFR at 9 W/kg, a level selected to yield a core-temperature rise of 4.5°C and stated to be lethal for chronic exposure. Sixteen other rats were sham-exposed. Subgroups at ´, 1, 2, and 4 cycles of spermatogenesis after treatment were analyzed for testis mass and daily sperm production as in the previous study. Also, trunk blood was assayed for follicle-stimulating hormone and leutinizing hormone.

No significant differences were found between the RFR- and sham-exposed rats in any of the endpoints, except for a decline in epididymal sperm count 26 days (2 cycles of spermatogenesis) after RFR exposure. However, the authors remarked that, in view of the negative results in the many other endpoints examined, the single positive result is highly questionable. They also noted that a differential sensitivity of germ cells at this stage of maturation had been reported for conventional heating of the testes.

In the first of several studies, Lotz and Michaelson (1978) first "gentled" rats for 2 weeks before exposure by weighing and handling them at least 4 times a week, and behaviorally equilibrating each rat by taking its colonic temperature and putting it into an exposure cage for 3-5 h for several days before use. Adrenal-axis activity during equilibration was determined by placing groups of rats in the exposure chamber for 30, 60, 90, 120, 150, or 180 min, measuring colonic temperature before and after each interval, and assaying the blood for corticosterone (CS) level. The results showed rapid rise of colonic temperature and CS level during the first 30 min to an approximate plateau, followed by return to baseline values by the end of 180 min, thus demonstrating the need for such equilibration prior to exposure.

The authors then exposed groups of unanesthetized gentled rats to 2.45-GHz RFR for 30, 60, or 120 min at levels up to 60 mW/cm (9.6 W/kg), and measured colonic temperatures and CS levels after exposure. A plot of mean postexposure colonic temperature versus exposure duration showed a small but significant rise after 30 min at 13 mW/cm^2 (2.1 W/kg); exposures for 30 min at higher levels yielded mean temperature rises approximately proportional to RFR level. The mean CS level rose nonsignificantly for durations up to 120 min at 13 mW/cm^2 (2.1 W/kg), up to 60 min at 20 mW/cm^2 (3.2 W/kg), and 30 min at 30 mW/cm^2 (4.8 W/kg). All other CS rises were significant and highly correlated with rises in colonic temperature. The estimated threshold values for adrenal-axis stimulation were 30-50 mW/cm^2 (4.8-8.0 W/kg) for 60-min exposures and 15-20 (2.4-3.2 W/kg) mW/cm^2 for 120-min exposures. The latter range is somewhat less than half the rat's resting metabolic rate.

Lu et al. (1985), noting inconsistencies in the findings of various investigators, described their studies with male Long-Evans rats from two suppliers, Blue-Spruce (BS) and Charles River (CR). Before treatment, the rats were acclimated and gentled, which included observation for 4 to 11 days, 2 weeks of handling, and 3 to 5 days of simulated exposure.

Treatment consisted of concurrently exposing groups of 4 rats each in individual cages were from above to 2.45-GHz RFR (or sham exposure) in an anechoic chamber. The minimum separation between rats was 18 cm. The normalized SAR was 0.19 W/kg per mW/cm^2. The seven protocols below were used, with each group of rats equilibrated for 3 h before the start of treatment.

1. Single exposures of one group each of BS rats only, for 1 h at 0 (sham), 1, 5, 10, 20, 40, 50, 60, or 70 mW/cm^2
2. Single 2-h exposures of BS rats at 0, 5, 10, or 20 mW/cm^2, and of CR rats at 25, 30, or 40 mW/cm^2
3. Single 4-h exposures of BS rats at 0, 1, 5, 10, or 20 mW/cm^2, and of CR rats at 0, 0.1, 1, 10, 25, or 40 mW/cm^2
4. Single 8-h exposures of BS rats only, at 0, 1, 5, 10, or 20 mW/cm^2
5. Single 4-h exposures of CR rats only, at 0, 0.1, 1, 10, 25, or 40 mW/cm^2, followed by sham exposure for 7 h starting 17 h after RFR exposure, with endpoints measured at the end of sham exposure (24 h after RFR-exposure completion)
6. Three consecutive daily 4-h exposures of CR rats only, at 0, 1, 10, 20, 30, 40, or 55 mW/cm^2
7. Ten consecutive daily 4-h exposures of CR rats only, 5 days a week, for 2 weeks at 1, 10, 20, 25, 30, or 40 mW/cm^2.

The rats were decapitated immediately after treatment, and their blood was assayed for serum T_4 concentration. Results for the 1-h exposures showed no apparent dose dependence: significantly higher T_4 levels were obtained for rats exposed at 40 or 70 mW/cm^2, but not at 50 or 60 mW/cm^2 or at 20 mW/cm^2 or lower. For the 2-h exposures, the T_4 levels for both BS and CR rats were higher at 25, 30, or 40 mW/cm^2 but were not significantly affected at 20 mW/cm^2 or lower. However, because the authors found that the normal T_4 concentrations for CR rats were higher than for BS rats, those and the later results were reevaluated by separately comparing T_4

levels for the RFR-exposed rats from each supplier with sham-exposed rats from the same supplier. When that was done for the 2-h exposures, no significant RFR-induced changes were found in T_4 concentration.

For the 4-h exposures, only results for BS rats were displayed. Their T_4 levels, when compared with those for sham-exposure, were significantly higher at 1 mW/cm^2, not significantly changed at 5 or 10 mW/cm^2, and significantly lower at 20 mW/cm^2. For the remaining protocols, there were no significant RFR-induced alterations of T_4 level, except for CR rats given the 3 consecutive daily 4-h exposures at 40 mW/cm^2, for which the T_4 level was significantly lower than for shams.

Because of their protocol design, the authors believed that they had avoided nonspecific reactions to stressful stimuli, but not those from repeated sham-exposures. However, they remarked:

> From the viewpoint of environmental health, changes in serum thyroxine cannot be used as an indicator of a past history of microwave exposure due to its limited magnitude of response and its sensitivity to extraneous factors.

Lu et al. (1986) assessed the influence of confounding factors in studies of effects of RFR on the adrenal cortex. After acclimation and gentling, groups of rats were subjected to 10 protocols involving single or multiple 2-h or 4-h exposures to 2.45-GHz RFR at levels in the range 0.1-55 mW/cm^2 (0.02-11 W/kg) and sham exposures. Their protocols included: (1) concentrations of CS in urine and its excretion rates, measurements of colonic temperature, and inhalation of ether to determine responsiveness of the hypothalamic-hypophysial-adrenocortical axis to an intensive stimulant; (2) injection of ethanol to lower the preexposure body temperature and limit the heat-dissipation ability of the rat; and (3) hair removal to increase heat dissipation during RFR exposure.

Rises in colonic temperature and CS concentration were found to be dependent on RFR level with distinct thresholds, but showed acclimation (diminution of effect with repetition). For repetitive 4-h exposures, the threshold for colonic-temperature rise was 10 mW/cm^2 (2 W/kg) at the first exposure, 30 mW/cm^2 (6 W/kg) at the third exposure, and 25 mW/cm^2 (5 W/kg) at the tenth exposure. The thresholds for the change of the CS concentration was 40 mW/cm^2 (8 W/kg) at the first exposure and 55 mW/cm^2 (11 W/kg) at the third exposure, however, rats exposed 10 times at levels up to 40 mW/cm^2 (8 W/kg) showed no changes. The changes observed were no longer present 24 h after exposure. For the sham exposures, baseline colonic temperature was higher after the tenth than the first treatment, but the CS concentration did not change.

Injection of ethanol lowered the baseline colonic temperature and raised the CS concentration – effects not observed for saline-injected controls. Exposures for 2 h at 1 or 10 mW/cm^2 following ethanol injection yielded lower colonic temperatures than for similar exposures after saline injection, but were higher for ethanol-injected rats exposed at 40 or 50 mW/cm^2. Interaction of ethanol and RFR at 10 and 20 mW/cm^2 also yielded higher CS concentrations than in saline-injected rats. A single exposure at 50 mW/cm^2 was lethal in 2 rats given ethanol.

Hair removal did not affect baseline colonic temperatures and CS concentrations significantly, but it decreased RFR-induced hyperthermia and CS stimulation.

The authors concluded that the adrenal response of the rat to RFR is quantifiable with respect to RFR level or colonic temperature, with no response to RFR in the absence of a rise in colonic temperature. They indicated that adrenal stimulation minimally required an RFR level of 20 mW/cm^2 (4 W/kg) or a 0.7°C increase in colonic temperature. They also remarked that RFR at less than 4 W/kg in ambient temperatures well above 24°C may induce hyperthermic stimulation of adrenal secretion.

Lotz and Podgorski (1982) implanted a catheter in the jugular vein of 6 rhesus monkeys for monitoring levels of cortisol, thyroxine (T_4), and growth hormone (GH). They collected blood samples from each monkey hourly for 24 h and measured colonic temperature with an indwelling probe. At the same clock time during the 24-h period, each monkey was exposed for 8 h to 1.29-GHz RFR at 20, 28, or 38 mW/cm^2 (2.1, 3.0, or 4.1 W/kg). The authors noted that the resting metabolic rate (RMR) of a rhesus monkey is 2.4 W/kg. Three sessions at each level were alternated with sham-exposure sessions at intervals of 10-14 days for recovery.

Hematocrit and Hb, monitored before and after each session, showed no significant decline. Samples of plasma from the blood were assayed for cortisol, T_4, and GH. The data collected for the same clock periods of the three sessions at each RFR level were averaged to yield a 24-h temporal series of mean values for each condition.

For the sham-exposure sessions, the mean rectal temperature showed a slight 24-h periodicity, with minimum values during the times when the chamber lights were off. It rose within 2 h after the beginning of RFR exposure to plateaus that were dependent on RFR level, but returned to the control profile within 2 h after exposure end.

For sessions at 20 and 28 mW/cm^2, the mean plasma-cortisol levels did not differ significantly from those for sham-exposure sessions, but rose significantly above control level during sessions at 38 mW/cm^2. The levels then diminished to control values, indicating that the effect was transient. The authors suggested that a threshold existed between 28 and 38 mW/cm^2 (3.0 and 4.1 W/kg), that the rises were associated with rectal-temperature elevations of about 1.7 °C, and that the results support the hypothesis that adrenocortical effects of RFR are thermally induced. For all RFR levels, no significant differences in mean GH or T_4 levels were seen at corresponding times during RFR and sham sessions.

In summary, although some effects of RFR exposure on the endocrine system appear to be straightforward and predictable from physiological considerations, other, more subtle effects may be worthy of additional study, such as those related to the interactions among the pituitary, adrenal, thyroid, and hypothalamus glands, and/or their secretions. Part of the problem in interpreting such results appears to be related to the uncertainties about stress mechanisms and various accommodations to such mechanisms. Animals placed in novel situations are much more prone to exhibit stress responses than those adapted to experimental situations.

Because the effects of RFR on the endocrine systems of animals are largely ascribable to increased thermal burdens, to stresses engendered by the experimental situation, or to both, there is no clear evidence that such effects would occur in humans exposed to RFR at levels that do not produce significant increases in body temperature.

B.3.6.3 Cardiovascular Effects

Few investigations have been carried out on possible effects of RFR on the human heart. However, various studies have been performed *in vitro* on hearts (or parts thereof) excised from animals, and others have been conducted on animal hearts *in vivo*.

B.3.6.3.1 *In Vitro* Studies

Frey and Seifert (1968) exposed 22 excised beating frog hearts to 10-μs pulses of 1.425-GHz RFR at 60 mW/cm^2 peak. Because the pulses were triggered at the peak of the P wave of the electrocardiogram (EKG) and at 100 and at 200 ms after the peak, the average power density was negligible. The results for zero and 100-ms delays were inconclusive, but a significant increase in heart rate (tachycardia) was seen for the 200-ms delay.

Clapman and Cain (1975) tried to obtain the results above. They exposed 3 of 14 groups of frog hearts to the 1.425-GHz pulsed RFR, but each group was triggered at only one of the delays. Three other groups were exposed with 15-μs instead of 10-μs pulses. The other 8 groups were exposed to 3-GHz RFR at 5500 mW/cm^2 peak, using 10-μs or 2-μs pulses. For one 2-μs group, the pulses were triggered at the initial rise of the QRS complex of the EKG and another group was exposed to unsynchronized pulses at 500 pps (5.5 mW/cm^2 average power density). No significant differences in heart rate were seen among any of the RFR-exposed groups and a control group, in contrast with the results of the study above.

Liu et al. (1976) also sought effects similar to those of Frey and Seifert (1968), but obtained negative results with excised hearts. They also opened the thorax of frogs and exposed the heart *in situ* to 100-μs pulses of 1.42-GHz or 10-GHz RFR, with negative results.

Galvin et al. (1981b) isolated cardiac muscle cells from the quail heart and exposed them in suspension to 2.45-GHz RFR at 37°C for 90 min at SARs of 1, 10, 50, or 100 W/kg. After exposure, samples of the suspensions were examined for integrity of the cells. (Intact cells exclude trypan blue, a stain.) The remainder of each suspension was centrifuged, and the supernatant was assayed for the release of the enzymes creatine phosphokinase (CPK) and lactic acid dehydrogenase (LDH). The residue pellets from the centrifugation were resuspended and assayed for bound enzyme. Some pellets were examined by electron microscopy.

Cardiac-cell integrity was unaffected by exposure at 1 W/kg, but the suspensions exposed at 10, 50, and 100 W/kg showed successively larger increases in percentages of cells permeable to trypan blue relative to their respective control suspensions. CPK release was unaffected at any SAR. Release of LDH increased with SAR, but the increases relative to the controls were statistically nonsignificant, except at 100 W/kg. By electron microscopy, the structural appearance of heart cells exposed at 1, 10, and 50 W/kg, as well as control cells, appeared normal. However, cells exposed at 100 W/kg showed increased intracellular changes, but the intercellular junctions remained intact.

Because Yee et al. (1984) were concerned about possible electrode artifacts, they distributed 102 isolated frog hearts in physiological (Ringer's) solution into 10 groups, and exposed each heart to 2.45-GHz RFR at 2 or 8.55 W/kg. Heart rates were recorded for each group with one of the following: a glass electrode filled with a potassium-chloride solution, an ultrasound probe, a tension transducer, a glass electrode filled with Ringer's solution, or a metal wire within the glass electrode containing Ringer's solution. Faster than normal decreases in heart rate were detected

only in those groups recorded with the potassium-chloride or metal-wire electrodes; no effect was found in the other groups. Those results show that electrode-caused field intensification can induce bradycardia.

Yee et al. (1986) divided 81 frog hearts into 10 groups. After each heart was excised and immersed in Ringer's solution to remove blood clots, its arteries were tied to prevent washing out of neurotransmitters at the nerve endings. Each heart was mounted within a waveguide filled with Ringer's solution. One group served as controls, 8 groups were exposed to 2.45-GHz RFR, and one group was heated by circulation of hot Ringer's solution through the waveguide. The beat rate of each heart was monitored for 60 min at 5-min intervals. For the hearts exposed to RFR, exposure was begun 10 min into the monitoring period and was terminated 30 min later. The groups were treated as follows:

Group A comprised the controls. The hearts in Group B were exposed individually to 10-μs pulses at 100 pps in 0.5-s trains of 50 pulses, with each train triggered by the EKG; the average SAR was 8.55 W/kg during each train. Group C was similarly exposed, but to random 0.5-s trains not triggered by the EKG. Group D was exposed to EKG-triggered trains, but at 2 W/kg. Group E was exposed to 10-μs pulses, 100 pps, at 8.55 W/kg, but continuously instead of pulse trains. Group F was also exposed continuously, but at 200 W/kg. Group G was continuously exposed at 200 W/kg, but was cooled by circulating bathing solution. Hot circulating bathing solution was used instead of RFR to treat Group H.

The authors noted that Schwartz et al. (1983) had reported a 19% increase in calcium efflux from isolated frog hearts exposed for 30 min to 16-Hz-modulated, 1-GHz RFR at 0.15-3 W/kg – an effect not observed for CW RFR or RFR amplitude-modulated at natural heart-beat rates. To investigate that finding, they exposed Groups I and J to CW and pulsed RFR, both amplitude-modulated at 16 Hz, at 3 W/kg average SAR.

The mean heart rate of Group A (controls) decreased linearly to about 67% of initial rate at the end of the 60-min monitoring period. The decrease for Group B was similar, with no significant differences between the groups at corresponding monitoring times. Similar negative results were obtained for Groups C, D, and E, relative to Group A. The authors remarked that the RFR-induced bradycardia observed by other researchers could be ascribed to loss of neurotransmitters via the open arteries, a problem avoided in their experiments by tying the arteries.

Exposure of Group F at 200 W/kg yielded heart-temperature increases of 2.5°, 5.5°, and 8 °C respectively at 5, 15, and 30 min of exposure; the mean heart rate decreased sharply to about 50% of the initial value by 20 min of exposure and decreased further to about 25% at the end of the monitoring period. Group H, heated by bathing solution to yield a temperature-versus-time rise similar to that with RFR, showed a linear decrease in heart rate to final values comparable with those of Group F. By contrast, the differences between Group G, cooled during exposure at 200 W/kg, and Group A were nonsignificant.

The results for Groups I and J, exposed to 16-Hz-modulated CW and pulsed RFR at 3 W/kg, were also not significantly different from those of Group A. The authors remarked that, given the increase in concentration of free Ca^{++} in the cytoplasm of heart cells triggers rapid changes in heartbeat, the absence of such changes in Groups I and J did not support the findings of Schwartz et al. (1983).

Yee et al. (1988) performed a similar study with rat hearts. However, to avoid arrythmia frequently observed previously in hearts exposed to air at temperatures less than 20 °C, and to increase the survival time of the excised hearts, the authors used a double circulation system, one part for coronary circulation of Ringer's solution and the other part for circulating Ringer's solution through the waveguide. As in the study of frog hearts, the beat rate of each heart was monitored for 60 min at 5-min intervals, but exposure was begun 20 min into the monitoring period and was terminated at 50 min.

In control Group A, the solution flowing through the heart was held at 37.7 °C; the temperature of the waveguide solution was initially at 19 °C, was increased within 10 min to 37.7 °C, and was held there for the remainder of the experiment. For this group, mean beat rate increased linearly to a maximum of 70% above initial value at 45 min, and then decreased sharply to 40% below initial value at 60 min. The hearts of Group B were not exposed to RFR but to air while the Ringer's solution through the coronary was held at 19 °C. This group frequently exhibited arrythmia, and its mean beat rate declined to about 20% of initial value.

Group C was exposed to 10-μs pulses, 100 pps, of 2.45-GHz RFR amplitude-modulated at 16 Hz, at a mean SAR of 2 W/kg, and otherwise treated the same as Group A. The graphs for heart rate versus time for Groups C and A were essentially the same, with no significant differences in heart rates at corresponding times. Group D was treated in the same manner as Group C, but at 10 W/kg. The heart rates also did not differ significantly at corresponding times, but were slightly higher than those of Group A for the first 15 min of exposure, results ascribed to a small increase in heart temperature (<0.2 °C).

Group E was exposed at 200 W/kg; it exhibited a sharp increase in mean heart rate during the first 10 min of exposure, followed by an abrupt cessation of beating after 20 min of exposure. The mean heart temperature rose to 41.7 °C during exposure, so no RFR was used for Group F, but the temperature of the bathing solution was raised to 41.7 °C, to approximate the temperature-time profile of Group E. The graph of heart rate versus time of Group F was similar to that of Group E. The authors concluded that exposure to RFR at 2 or 10 W/kg had no specific influence on the myocardium or its neural components.

B.3.6.3.2 *In Vivo* Studies

Among the early studies on the live rabbit were two by Presman and Levitina (1963a, b), one with 2.4-GHz CW RFR at 7-12 mW/cm^2 and the other with 3-GHz pulsed RFR at 3-5 mW/cm^2 average power density, 4.3-7.1 W/cm^2 pulse power density. In both studies, the rabbits were exposed for 20-min periods each in various aspects. During each exposure and for 10 min before and afterward, the EKGs of the rabbits were recorded with plate electrodes. The rabbits were sham-exposed once each before and after the series of RFR exposures.

Exposing the entire dorsal surface produced neither tachycardia nor bradycardia during exposure. However, tachycardia was seen during the first half of the postexposure period, changing to bradycardia toward the end of that period. By contrast, dorsal exposure of the head only or of the back only produced significant tachycardia during exposure, with head exposure yielding the greater effect. The tachycardia rose to maximum values about 5 min postexposure and then declined to nonsignificant values. Bradycardia was seen during exposure in three ventral aspects; it persisted to exposure end and was followed by returns toward normal heart

rates during postexposure. The effect was most pronounced and was manifested earliest for exposure of only the head.

Other authors tried to repeat that study. The results were negative except for RFR levels that were clearly hyperthermic.

Phillips et al. (1975) exposed rats to 2.45-GHz RFR for 30 min at 0, 4.5, 6.5, or 11.5 W/kg. Nonsignificant bradycardia was seen at 4.5 W/kg; mild but significant bradycardia developed within 20 min at 6.5 W/kg, followed by recovery in 2 h; pronounced bradycardia occurred abruptly at 11.1 W/kg, after which heart rates rose to values well above those of controls and persisted at the higher levels to the end of the test period. For most of the rats exposed at 11.5 W/kg, the bradycardia was accompanied by irregular heart rhythms. Incomplete heart block was evident, but with recovery within 60 min after exposure cessation. The authors surmised that the heart block was caused by the release of toxic materials, elevated serum potassium, or myocardial ischemia, all from excessive heat.

Galvin and McRee (1981a) studied the effect of RFR exposure *in vivo* on the functioning of the cat heart with and without myocardial ischemia (MI). The MI was produced surgically by occlusion of a coronary artery. One group of MI hearts was exposed to 2.45-GHz CW RFR at 30 W/kg with an applicator for 5 h, and another MI group was sham-exposed. At this RFR level, heart temperature of dead cats rose at an initial rate of 0.43 °C per minute, but no increases in aortic blood temperature occurred in live cats. For comparison, two groups of non-MI hearts were similarly treated. Before and during the treatment, mean arterial blood pressure, cardiac output, heart rate, and EKG were measured, and blood samples were assayed for plasma protein concentration and CPK activity. After the treatment, the hearts were excised and assayed for tissue CPK activity.

The results for both the MI and non-MI cats showed no significant differences in mean arterial blood pressure, cardiac output, or heart rate between RFR- and sham-exposed groups, and no synergy of ischemia and RFR exposure for those cardiovascular indices. The RFR and sham groups yielded no significant differences in either plasma or tissue CPK activity. Thus, localized exposure of the undamaged or ischemic heart to the RFR *in vivo* had no effect on the myocardium or its neural components – results at variance with those for excised hearts exposed to RFR.

Galvin and McRee (1986) studied the effects of exposure to 2.45-GHz CW RFR on conscious rats, including cardiovascular, biochemical, and hematologic indices. Before treatment, the right femoral artery of each rat was cannulated under anesthesia to permit the continuous recording of mean arterial blood pressure and the removal of blood samples. Following surgery, the leg wound was sutured and infiltrated with Lidocaine and the rat was allowed to recover for 2 h, during which stable heart rate, blood pressure, and colonic temperature were usually achieved.

No significant differences between RFR- and sham-exposed rats were found in any of the blood parameters assayed. Initial mean heart rates of the RFR and sham groups did not differ significantly. However, during the first hour of exposure, the mean heart rate of the RFR group decreased to about 90% of its initial value, a significant drop, and remained there (with smaller variations) during the rest of the period.

The occurrence of bradycardia led the authors to study another group of rats. The rats were exposed to the RFR for 2 h and monitored for 2 h after exposure cessation. Those rats also exhibited bradycardia after the first hour of exposure, but heart rates returned to preexposure values during the postexposure period (no data presented). The authors noted that the bradycardia was small, and they surmised that it was due to reduction of metabolic rate to compensate for the heat from the RFR.

B.3.7 Behavior

Numerous studies have been conducted on the possible effects of RFR exposure on various kinds of animal behavior. Representative papers on this topic have been selected and are discussed in Section 3.7.1. Possible interactive effects of exposure to RFR and drugs on the behavior and physiologic responses of animals are discussed in Section 3.7.2.

B.3.7.1 RFR Effects on Naturalistic Behavior, Reflex Activity, Learning, and Performance of Trained Tasks

B.3.7.1.1 Rodents

Justesen and King (1970) trained food-deprived rats to lick a nozzle 40 successive times to obtain a drop of dextrose-water solution. The authors then exposed the rats within a modified commercial microwave oven to 2.45-GHz RFR at levels up to 1.5 mW/cm^2 (4.6 W/kg) in 1-h sessions consisting of alternating 5-min intervals of RFR and no RFR. The task was rendered more complex with an audio tone, with "tone-on" or "tone-off" presented at random intervals during a session as a signal of reward availability.

The mean number of responses by the rats diminished with increasing RFR level, but the decreases at higher levels were related to cessation of responding rather than lower licking rates, most likely associated with warming.

In a three-part study, Hunt et al. (1975) exposed rats to 2.45-GHz RFR in a holder within a modified microwave oven for 30 min. In the first part, each rat was sham-exposed or exposed to the RFR at 6.3 W/kg, after which it was placed in a chamber in which its exploratory movements were recorded. The mean activities after both treatments decreased with time, but the values were generally lower during most of the period after RFR exposure than after sham-exposure and became comparable for the two treatments toward session end. The RFR-exposed rats were frequently seen sleeping during the middle parts of sessions.

In the second part, rats were trained to swim a 6-m channel forth and back repeatedly during a 24-h period, with rests of 20-30 s at each end of the channel. Each rat's performance versus time was scored as its median swim speed for each successive block of 20 traverses. After training, the rats were given a pretreatment test and distributed into RFR and sham groups on the basis of equal proficiency.

In the first of 3 experiments in this second part, rats were sham-exposed or exposed at 6.3 W/kg for 30 min and tested immediately after treatment to determine if any effects were present. In the other 2 experiments, the RFR level was 11 W/kg and the rats were tested immediately after treatment or after a 1-day delay.

The performance of the 6.3-W/kg group was similar to that of the sham group for about 200 traverses, but was below mean control speed for about the next 100 traverses, after which both groups again performed comparably. Colonic temperatures measured immediately after treatment showed that those exposed at 11 W/kg were rendered severely hyperthermic (41 °C or higher), with partial relief by immersion for those tested right after exposure. At 11 W/kg, the performance of the group tested immediately after exposure was clearly impaired by the hyperthermial, despite the partial relief from immersion. However, the 11-W/kg group tested 1 day later showed recovery from the hyperthermia and yielded results similar to those of the 6.3-W/kg group.

In the third part of the study, water-deprived rats were trained to press a lever in a complex vigilance-discrimination task to obtain 0.08-ml quantities of saccharin-flavored water. After stable performance was achieved, each rat was tested on 5 successive days. On the first day, all were sham-exposed. In the next 4 days, each group was exposed for one 30-min period each at 6.5 and 11 W/kg and 2 of sham-exposure. Performance was tested for 30 min immediately after each treatment, and the results were expressed as percentage errors of omission and commission versus elapsed time at 5, 15, and 25 min of testing.

For the test sessions following sham-exposures, the mean omission errors were in the 10-15% range at all 3 times. For the session after exposure at 6.5 W/kg, the mean of omission errors was 36% at 5 min, but dropped to the sham-exposure range at 15 and 25 min. After exposure at 11 W/kg, however, the percentages were all much higher than sham-exposure values. No significant differences were found in the mean commission-error results among treatments.

Monahan and Ho (1976) described experiments to determine whether mice would orient themselves to minimize absorption of RFR under conditions that did not allow them to escape. Each mouse was exposed for 15 min in a holder that permitted relatively free movement within a waveguide system to 2.45-GHz CW RFR at forward powers in the range 0.4-4.8 W in an ambient temperature held at 24 °C by air flow through the waveguide. The mean SAR and the percentage of forward power absorbed were measured at 5-min intervals. In a second experiment, exposures were limited to 10 min, and the absorptions were recorded at 12-s intervals. The mice could not be watched within the waveguide, but the results of both experiments showed that they oriented themselves to reduce their percentages of RFR energy absorbed and SARs when the forward power was about 1.7 W (initial SAR 28 W/kg) or higher (at 24 °C).

Lin et al. (1977) sham-exposed or exposed food-deprived rats to 918-MHz RFR at 10, 20, or 40 mW/cm^2 (2.1, 4.2, or 8.4 W/kg) in 30-min sessions. The rat holder was a truncated cone of rods designed to allow the rat to poke its head through the narrower end and move it freely. A small upward head movement interrupted a horizontal light beam. The rat was required to execute 30 such movements rapidly and regularly for a food pellet. A downward movement gave access to the pellets delivered.

The baseline cumulative responses versus time for three rats showed virtually uniform rates. One of those rats was exposed for 30 min each at 2.1, 4.2, and 8.4 W/kg on consecutive days, another was exposed at the same levels on alternate days, and the third was given 30-min sessions of sham-exposure. No significant performance-rate changes were seen at 2.1 or 4.2 W/kg. At 8.4 W/kg, the first rat's performance did not change for the first 5 min, at which time its rate dropped to almost zero; the second rat performed at baseline rate for the first 5 min, at a

slowly decreasing rate for the next 15 min, and then ceased performing for the remaining 10 min. Both showed heat stress, including panting, fatigue, and foaming of the mouth.

Another rat was exposed in increments of 3 mW/cm^2 (0.63 W/kg) up to 32 mW/cm^2 (6.7 W/kg), at which level it exhibited similar signs of heat stress. At that level, its response rate remained at baseline for about the first 13-14 min, diminished slightly during the next 5-6 min, and then dropped significantly for the rest of the session. The results appear to be straightforward and indicate the existence of a threshold between 30 and 40 mW/cm^2 (6.3 and 8.4 W/kg) at 918 MHz for that task.

Computerized thermography was used in the study above to determine energy absorption rates in rat carcasses. An important general finding was the large spatial range of local SARs. As the authors noted, high values of local SAR ("hot spots") could occur from exposure to RFR at seemingly thermally insignificant power densities.

Schrot et. al. (1980) exposed 3 rats to pulsed 2.8-GHz RFR at average power densities in the range 0.25-10 mW/cm^2 (0.04-1.7 W/kg), and observed their behavior acquisition. The test apparatus was a standard two-lever rat chamber augmented with a third lever. Each rat was trained to respond to auditory stimuli with chains of presses on the 3 levers until a predetermined sequence of 4 presses of the 3 levers was learned. Each rat was required to learn a different four-member chain of responses during each session. Just before behavior assessment, the rats were sham-exposed or exposed at one of the RFR levels for 30 min. The sessions were conducted daily, 5 days a week, and were terminated after 150 reinforcements or 2 h, whichever occurred first.

Presession exposure at 10 mW/cm^2 (1.7 W/kg) of all 3 resulted in higher error-responding rates, lower sequence-completion rates, and alteration in the normal acquisition pattern. Similar effects were seen at 5 mW/cm^2 (0.7 W/kg) but to a lesser extent. Below 5 mW/cm^2, most data points were within the control range, but a few were outside that range. The significance of the latter points is uncertain.

Gage and Guyer (1982) trained rats to perform on a reinforcement schedule in which the opportunity to obtain a food pellet was presented on the average of once each minute in a preplanned sequence of intervals without cueing. The rat lost a reinforcement opportunity if it did not respond before the next opportunity. After training, groups were exposed to 2.45-GHz RFR for 15.5 h at 8 or 14 mW/cm^2 (1.6 or 2.8 W/kg) and 22, 26, or 30 °C ambient temperature without access to food or water. The rats were given water for 10 min after treatment, after which the test sessions were begun. The response rates at each ambient temperature diminished directly with increasing RFR level, but the effects of ambient temperature *per se* were not consistent.

Lebovitz (1981), concurrently sham-exposed and exposed groups of 15 rats to circularly polarized 1.3-GHz pulsed RFR (1-μs pulses at 600 pps) unrestrained in individual waveguides. Each waveguide contained a vertical displacement bar (behavioral operandum), a means for illuminating the operandum as a cue (visual discriminative stimulus), and means for delivering a food pellet. The rats faced the RFR source during operant performance.

Before exposure, groups of 46 rats each were initially deprived of food for 2 days and trained for 10 days to press the bar for food pellets at increasing fixed-ratio (FR) schedules to FR-5 (requiring 5 successive lever presses for a pellet). The 30 rats of each group that performed at the highest and most stable rates were selected and trained on a multiple FR, extinction

schedule of reinforcement that involved visual discriminative stimuli, in which only the responses when an operandum was illuminated (SD) were reinforced by pellet delivery. Such responses were tabulated. Reinforcements were done on an FR schedule that was gradually increased to FR-25 during several weeks of training. Responses when the operandum was not illuminated (Sd), which yielded no pellets, were tabulated separately.

Each set of 30 rats that achieved high and steady performance at FR-25 was given a baseline period of sham exposures and testing, after which half were randomly assigned to the RFR group and half to the sham group, with the 2 groups matched by baseline FR-25 performance. RFR exposures and sham exposures were for 3 h daily, 5 days a week. The behavioral sessions were begun 15 min after exposure start and were terminated 15 min before exposure end, for a 150-min session duration. The rats were also tested during a 2-week recovery period after the exposure regimen. Each behavioral session was divided into 6 sequential blocks of 25 min each; each block consisted of a 15-min SD interval (with the operandum illuminated) followed by a 10-min Sd interval (operandum illumination extinguished). The response rates of each rat for SD and Sd during baseline, exposure, and recovery periods were summed weekly by block number, and the results for the rats in each group were averaged.

One group each was exposed at 1.5, 3.6, and 6.7 W/kg (3.9, 9.2, and 17.2 mW/cm^2 average; 6.4, 15.4, and 28.7 W/cm^2 peak). When the rats were not in their waveguides, they were held in their home cages with water constantly available. Each rat was given 8 g/d of food irrespective of its operant performance, and was weighed 3 times per week. All rats maintained satisfactory growth curves.

Results for 8 weeks of sham exposure and exposure at 1.5 W/kg (3.9 mW/cm^2) showed stable response rates and no significant SD differences between RFR and sham groups for corresponding blocks and weeks. Modest declines in rates during sessions (blocks 1-6) were seen in both groups. The response rates for Sd were more variable than for SD. The decline in Sd rates during sessions, which was also evident for the baseline and recovery weeks, was sharper than for SD, but no significant differences were found between the RFR and sham groups.

Groups sham-exposed and exposed for 9 weeks at 3.6 W/kg (9.2 mW/cm^2) also showed no significant differences in SD response rates. Marginally significant changes occurred in the weekly rates, and the intrasession decline in rate was significant. For the Sd response rates (which again showed sharp intrasession declines for both groups), analysis revealed an apparently transient difference between groups: the Sd response rate by the RFR group was significantly lower than for the sham group only for blocks 2 and 3 of the first exposure week. The author remarked that similar results were obtained with another group of rats exposed at the same RFR level.

Results for 6 weeks of exposure at 6.7 W/kg (17.2 mW/cm^2) showed no significant differences between groups in overall SD response rates, but there were significant block-dependent differences between groups. In particular, the RFR group's block-6 SD rate was significantly lower than the sham group's rate for week 2, and was marginally significantly lower for weeks 1, 3, and 4. The differences were ascribed to reductions in bar pressings near the end of behavioral sessions (blocks 4-6) during those weeks. An analysis by rat showed that the differences between SD rates during the last baseline week and the first exposure week were not significant, indicating that the decline was gradual rather than immediate.

At 6.7 W/kg (17.2 mW/cm^2), the Sd rates showed significant intrasession declines for both groups during the baseline period, with nonsignificant intergroup differences. For unknown reasons, however, the Sd rates of both groups varied up and down during sessions, rendering it difficult to interpret the results. The block-6 rates of both groups were already low during the baseline period, but the RFR group's rate dropped to almost zero during exposures, with only slight increases seen during the recovery period.

On the basis of the negative results for SD and Sd at 1.5 W/kg and the doubtfully significant decline in Sd rate at 3.6 W/kg, the author suggested that the latter RFR level could be the approximate threshold for modifying the rate of operant responding in the absence of visual cues or food reinforcement.

The author also indicated that 6.7 W/kg is the approximate resting metabolic rate for a 240-g rat; accordingly, such RFR exposure represented a virtual doubling of the heat dissipation requirements of the rat. He therefore concluded that thermal factors were probably involved in the behavioral effects. By his calculation, the energy deposited in the rat during each pulse exceeded the threshold for the RFR-auditory effect, but he questioned whether the loudness perceived by the rat would constitute an adequate acoustic cue or how the presence of such a cue could account for the observation that the major decline in Sd responding was gradual rather than immediate with the onset of RFR exposure. He indicated that other studies with CW RFR yielded essentially the same findings.

As noted, the operant data for each rat consisted of the number of bar presses during each of the 6 blocks or pairs of cued (SD) and uncued (Sd) response intervals sequentially numbered 1-6 daily. However, the rationale was not clear for summing the responses for correspondingly numbered blocks to obtain weekly block SD and Sd response totals as the "primary descriptive variables" for each rat, and why the time-dependent data for the successive blocks during daily sessions were not described or treated more explicitly, because even the baseline SD and Sd rates both exhibited intrasession diminutions. Thus, it is difficult to assess the contribution of this time-dependent non-RFR factor despite the extensive statistical treatment.

Lebovitz (1983) sham-exposed or exposed trained groups of 15 rats each to CW or pulsed 1.3-GHz RFR (1-μs pulses at 600 pps). As in the previous study, the rats were trained to press the bar for pellets at increasing fixed-ratio schedules to FR-5, and then were trained daily on a multiple schedule that started with a 15-min interval (called S+ instead of SD) of bar illumination and pellet availability at FR-25, followed by a 10-min time-out interval (called S- instead of Sd) of no illumination or pellet availability. Each session consisted again of 6 contiguous pairs of S+ and S- (25-min periods) numbered 1 through 6.

After a week of baseline performance by 2 groups, 1 was exposed to CW RFR at 5.9 W/kg (15.2 mW/cm^2), and the other was sham-exposed for a week. For the baseline week, the S+ results for both groups exhibited a trend toward decreasing response rates of 10% from period 1 to period 6 but no significant differences between the groups. During the week of exposure, both groups also showed a downward trend in S+ rates, but the RFR group's decline in response rate was significantly faster.

During the baseline week, the S- response rates of this RFR group were initially higher than for the sham group, but they declined faster between period 4 and period 5; thus the S- rates for the two groups were comparable for periods 5 and 6. During the week of exposure, the sham

group exhibited higher rates for periods 1 and 2 than they did for the same periods of the baseline week, and approximately the same rate of decline. However, the rates of the RFR group for periods 1-3 dropped sharply to almost zero for periods 4-6.

For exposure to pulsed RFR at 6.7 W/kg (17.2 mW/cm^2), response rates during both S+ and S- were similar to those with CW RFR at 5.9 W/kg (15.2 mW/cm^2). (Limitations of equipment did not permit closer match of SARs.) The author remarked that the results with pulsed RFR at this level were similar to those obtained in the previous study with pulsed RFR at the same level and that occurrence of similar changes in S+ rates with CW RFR at a comparable SAR showed that the effect was not ascribable to the pulsed character of the RFR.

For a group exposed to CW RFR at 3.6 W/kg (9.2 mW/cm^2), the S+ rates during the baseline week were consistently lower than those for the corresponding sham group, but the rates of decline for periods 1-6 were essentially the same. Similar results were also obtained for the week of exposure except that the initial (period-1) response rates for both groups were higher than the initial rates for the baseline week.

The S- rates of the 3.6-W/kg group were consistently higher than those of the sham group for the baseline week, but with comparable rates of decline, thus yielding no significant differences between the groups. The S- results for the week of exposure showed that the response rates of the RFR group were consistently higher than of the sham group; the response rates of both groups declined for periods 1-6, but the decline was much faster for the RFR group. These results were again consonant with those of the previous study.

Separate groups of 5 rats each were used to determine rises in core temperature due to RFR exposure. Each rat was exposed for 1 hour or 3 h and its temperature was measured just before it was placed in the waveguide and within 10 min after it was removed. Exposures to CW or pulsed RFR at 3.5 W/kg, the approximate threshold for the behavioral effects above, yielded no significant differences in rectal-temperature changes compared with rats similarly sham-exposed. However, exposures at 6.3 W/kg, CW or pulsed, yielded increases of 0.5-1 °C, with no significant duration-dependent differences.

As was true for the previous study, the engineering aspects were excellent, and the statistical treatment of the data provided a sound basis for the conclusions reached. Moreover, the presentation of the data at 5.9 W/kg (CW) by operant days (a format lacking in the previous paper) provided greater insight into the time-dependent aspects of the results. Especially noteworthy was that the daily S+ response rates for period 1 were not significantly affected by the entire week of exposure to RFR and that the declines in those rates occurred progressively in the subsequent periods of each session. Also more clearly evident were the virtually immediate sharp declines in S- response rates for all periods at the onset of RFR exposure. In the absence of light cue and pellet rewards, it is possible that the rats were thoroughly confused by the presence of the RFR. Another possibility the author suggested was that without such reinforcement, the rats tried to reorient themselves so as to redistribute the thermal burden added by the RFR.

As indicated by the author, the thermal basis for the behavioral changes is evident, with a threshold of about 3.5 W/kg irrespective of whether the RFR is CW or pulsed. Also, even though the pulse width (1 μs) and peak power density (estimated as about 28.7 W/cm^2) were sufficient to produce the RFR-auditory effect, there was little doubt that perception of the pulses as sound (if it occurred) was not a factor in the results obtained.

D'Andrea et al. (1986a) adapted 28 rats to exposure chambers and divided them into two groups of 14 each. One group was exposed for 7 h/d on 90 consecutive days, totaling 630 h, to 2.45-GHz CW RFR at 0.5 mW/cm^2 (0.14 W/kg); the other group was sham-exposed. Body masses and intake of food and water were measured daily. Each rat was tested monthly for its threshold reactivity to footshock by observing its paw movements in response to electric shocks of varied intensity within a gridded-floor chamber. The differences in body masses, food and water intake, or threshold footshock reactivities between the groups were not significant.

After the 90 days of treatment, 7 rats of each group were assessed for open-field behavior, shuttlebox performance, and lever pressing for food pellets on an interresponse time schedule. In the open-field tests, each rat was placed in the center square of a floor of 20-cm squares, and its crossings into adjacent squares and its rearings during a 1-min period on each of 3 successive days were counted. Major changes were seen in both tests over the daily trials, but no significant differences were ascribable to RFR exposure. The open-field tests 60 days after treatment yielded similar results.

Shuttlebox performance was tested using a tone and white light as a compound conditional stimulus (CS) and electric shock as an unconditional stimulus (UCS). Each rat was trained on trials consisting of presenting the CS for 10 s immediately followed by presentation of the UCS. By crossing to the other side of the shuttlebox during the 10 s of CS, the rat could prevent presentation of the UCS, a behavior termed an avoidance response. A crossing during a UCS presentation was termed an escape response. The latencies for avoidance and escape responses were recorded. Two days after shuttlebox testing, the rats were deprived of food and trained daily to press a lever twice for a food pellet, with a specific time interval between the presses. The training was rendered more difficult until the rats were required to do the second press only between 12 and 18 s after the first press to obtain a food pellet.

The shuttlebox responses were highly variable: four RFR-exposed rats showed relatively long escape latencies and poor avoidance, whereas the other three RFR-exposed rats did as well as those sham-exposed. Overall, the differences were not statistically significant. The shuttlebox test done 60 days after treatment also showed no significant differences in mean latencies or their variances. During training in the interresponse-time tests, the RFR group earned fewer pellets than the sham group, but the differences at corresponding times and overall were nonsignificant.

The results of the study above and of two similar studies in the same laboratory (D'Andrea et al., 1986b; DeWitt et al., 1987) were not fully consistent and exhibited little if any statistically significant differences between RFR-exposed and sham-exposed rats. However, they suggested that the threshold for behavioral responses to 2.45-GHz RFR in rats may be in the range 0.5-2.5 mW/cm^2 (0.14-0.70 W/kg).

Mitchell et al. (1988) exposed rats from above to 2.45-GHz CW RFR at 10 mW/cm^2 for 7 h within an anechoic chamber in individual plastic cages that permitted free movement. Concurrently, 2 cages were exposed to the RFR and 2 others were sham-exposed in another anechoic chamber, with 10 replications of the experiment (20 each RFR-exposed and sham-exposed rats). Calorimetric measurements with rat carcasses yielded a spatial mean whole-body SAR of 2.7 W/kg, assuming that the rats spent equal times in horizontal orientations parallel and perpendicular to the electric component of the RFR. For 8 days before treatment, the rats were adapted to the chambers for 8 h daily. Right after treatment, each rat was tested for vertical and

horizontal spontaneous locomotor activity, acoustic startle response, and retention of a shock-motivated passive avoidance task.

The vertical and horizontal locomotor activities of each rat were assessed as photoelectric detections of light-beam interruptions during 5-min intervals of a 30-min test session. Lower activity was seen in the RFR-exposed rats than the sham-exposed rats, especially during the second half of test sessions.

In the startle-response test, given at the end of the locomotor test session, each rat was subjected to 20 intense 8-kHz, 0.2-s acoustic pulses at variable intervals in the range 20-60 s (mean interval 40 s), and the response of the rat during each acoustic pulse was determined. The mean of startle responses of the RFR-exposed rats was significantly lower than for the sham-exposed rats.

Immediately after the startle-response test, each rat was placed in the lighted smaller part of a gated, two-chamber shuttle box, the larger part of which was dark and equipped to deliver an electric shock. After 1 minute for adaptation, the gate was opened, and if the rat moved into the larger chamber within 2 min, it was given a 1-s shock; if it remained in the smaller chamber for more than 2 min, it was removed and not tested further (17 RFR-exposed and 16 sham-exposed rats were tested). One week later, retention of the shock experience was tested in the box by allowing each rat 5 min (instead of 2 min) within the smaller chamber to react. The differences in passive avoidance activity between the two groups were not significant.

Akyel et al. (1991) trained 12 rats to press a lever to obtain food pellets. The rats were then trained on reinforcement schedules: 4 rats on an FR schedule, another 4 rats on a variable interval (VI) schedule, and the remaining 4 rats on a differential-reinforcement-of-low-rates (DRL) schedule.

After training and subsequent adaptation to the exposure chamber, each rat was exposed once a week for 10 min to 10-μs pulses of 1.25-GHz RFR at 1-MW peak forward power, with its long axis parallel to the electric component of the RFR. The average forward power was held constant, during any session, at 4, 12, 36, or 108 W, obtained by using a pulse repetition frequency of 240, 720, 2160, or 6480 pps. Each rat was administered all four RFR levels in a weekly quasirandom order. The corresponding whole-body total doses or specific absorptions (SAs), and the whole-body SARs respectively ranged from 0.5 to 14.0 kJ/kg and 0.84 to 23.0 W/kg. Testing of each rat was begun within less than 80 s after exposure end.

At the three lower RFR levels, no significant differences in any of the three behavior schedules were seen. At the highest level (14.0 W/kg, 23.0 W/kg), however, the rats trained on the FR and VI schedules failed to reach baseline performance, and those on the DRL schedule exhibited variable effects. Exposures at that level caused an average rise in colonic temperature of 2.5°C, and the rats did not respond at all for about 13 min after exposure completion. The authors concluded that those behavioral changes were thermally induced.

B.3.7.1.2 Nonhuman Primates

Galloway (1975) trained rhesus monkeys to press one or more of three levers on panels when the panels were selectively lit in order to obtain food pellets. After training, the head of each monkey was exposed with an applicator to 2.45-GHz RFR at estimated mean head SARs of 7, 13, 20, 27, and 33 W/kg, and the effects on their performance were examined.

For studying discriminative behavior, the RFR was administered for 2 min just before each behavioral session but was terminated earlier if the monkey began to convulse. Convulsions occurred for all exposures at 33 W/kg, and often at 20 W/kg. Each monkey was exposed at least twice at each level during a 9-month period. In addition, 3 were exposed at 13 W/kg for 5 daily 1-h schedules of 2 min on and 1 min off, totaling 40 min of exposure per day. No effects on discriminative behavior were evident for either exposure regimen.

In a repeated-acquisition test, each monkey had to press the correct lever for each of four illumination stimuli in the proper sequence. An incorrect lever press caused a 15-s timeout, during which any lever press had no effect. Sessions of 60 trials were conducted daily before exposure, with the correct sequence changed each day. In sessions just preceding RFR exposure, a slight learning trend (diminishing error rate) was seen, but the changes were too small to ascribe significance to. This was also true for the results at all RFR levels, except 33 W/kg, for which the error rate at session start was highest. Thus, except possibly for the latter result, the RFR had no effect on this behavioral paradigm.

Cunitz et al. (1975) trained a 3-kg and a 5-kg rhesus monkey on a 4-choice, forced-choice serial reaction program. Each monkey's head was inserted through a hole in the bottom of a 383-MHz resonant cavity, with the monkey facing a diamond array of the ends of 4 light pipes mounted through the cavity's side wall. Lighting any pipe required the monkey to move a lever to the left, right, up, or down to correspond with the position of that pipe end in the diamond. For criterion performance, 100 correct lever presses were required to obtain a food pellet. During performance, the light stimuli were presented in random order. A correct response produced an immediate stimulus change and presentation of a tone for 0.75 s. An incorrect response yielded a 3-s timeout during which all lights were off and lever movements had no consequences.

In each session, the monkey was restrained in a chair for 1 h before the behavioral program was started; the program was then conducted for 1 hour (or halted sooner when the monkey obtained its entire daily food ration). Each monkey was exposed to 383-MHz RFR during the entire 2-h session at a fixed power input in the range 0-15.0 W. Head SARs were estimated to range up to 33 W/kg and 20 W/kg respectively for the 3-kg and 5-kg monkeys. The larger monkey was also exposed at 17.5 W (23 W/kg). The sessions were conducted on 5 consecutive days at each level, with sham-exposure sessions before the RFR was raised to the next level.

Exposures below 10 W did not alter either monkey's performance. The rate of correct responses of the 3-kg monkey at 10 W (22 W/kg) decreased sharply during exposure days 1-3, and recovered partially on days 4 and 5 and the subsequent sham-exposure sessions. At 15 W (33 W/kg), the drop was very severe (to almost zero on day 5), with recovery to about a third of baseline rates during the subsequent sham-exposure sessions. The 5-kg monkey's performance was not affected significantly at 15 W (20 W/kg). At 17.5 W (23 W/kg), its performance dropped sharply, but recovered to baseline in the subsequent sham-exposure sessions, indicating that the effect was reversible. The lowest head SARs for diminished performance by the 2 monkeys were about the same: 22 and 23 W/kg.

Scholl and Allen (1979) trained three rhesus monkeys in a visual-tracking task that required each monkey, seated in a restraining chair, to move a lever to hold a continuously moving spot within a prescribed clear area on the screen of a display monitor. The spot was moved electronically in a specific pattern, and the lever responses generated continuous difference signals (errors). The central 15% of the screen was clear and made up the on-target area. That

area was surrounded by a 35% area of light blue, and the remaining 50% area was dark blue. The monkey received a 0.1-s electric shock for each 1 s accumulated outside the clear on-target area.

After training, the monkeys were exposed to horizontally polarized, 1.2-GHz CW RFR at 10 and 20 mW/cm^2 (measured at the center of the head in the absence of the monkey) for 2 h/d at 2-day intervals until each was exposed for 120 min at each level. This polarization and frequency were chosen to provide half-wave resonant absorption in the monkey head. The corresponding head SARs were 0.8 and 1.6 W/kg. Each daily session comprised 40 work trials of 1.5 min each, alternating with similar rest periods. Baseline runs were done for 26 consecutive days to ensure performance stability, and the results of the last 6 runs were used for statistical analysis.

The endpoint scored was the adjusted root mean square (ARMS) of the tracking error for each trial, expressed as a percentage of the total target area. The 95% simultaneous confidence limits were calculated for each monkey's baseline runs, and the ARMS was plotted for each of the 40 trials in each 2-h session during RFR exposure at each level. Of 720 data points collected during a total of 36 h of RFR, only 4 points were outside the confidence limits, fewer than expected by chance. It seems clear that the performance of the monkeys was not diminished by the RFR exposure. Whether the apparent performance improvement observed was RFR-related could not be ascertained.

De Lorge (1976) trained five rhesus monkeys to perform the following task while seated: each monkey was required to press a lever in front of its right arm, thus producing either a low-frequency tone for 0.5 s to signal that no food pellet will be coming, or a higher-frequency tone for which the monkey had to press a lever in front of its left arm to receive a pellet. Training sessions lasted for 1 or 2 h. During 1-h sessions, pellets were made available at VIs around an average of 30 s (VI-30-s schedule). For example, presses of the right lever would yield the high tone once about every 30 s and the low tone at other times. During the 2-h training sessions, pellets were made available on a VI-60-s schedule.

The monkeys were exposed frontally to vertically polarized, 120-Hz-modulated 2.45-GHz RFR at levels in the range 4-72 mW/cm^2 measured at head height. Superposed in some experiments were 0.1-s pulses at 1 pps. Estimated head SARs were 0.4-7.2 W/kg (0.1 W/kg per mW/cm^2).

After stable VI-30-s behavior was achieved, 1-h sessions were conducted on each monkey, during which the monkey was exposed to the 120-Hz-modulated RFR with superposed 0.1-s pulses at 4 or 16 mW/cm^2 (0.4 or 1.6 W/kg head SAR) for 30 min. Similar sessions were conducted with the unpulsed modulated RFR and with no RFR. At either RFR level, the performances of the monkeys were not affected by either the unpulsed or pulsed RFR, which led to the use of only the unpulsed RFR and of the VI-60-s schedule during the 2-h test sessions. Only 3 of the monkeys were tested in the 2-h sessions, during which they were exposed for 1 hour at levels in the range 16-72 mW/cm^2. One of them was also exposed at 16 mW/cm^2 during the entire 2-h test sessions.

The VI-60-s performances showed no significant departures from the control rates for all 3 monkeys up to 52 mW/cm^2 (5.2 W/kg) and for 2 of them at 62 mW/cm^2 (6.2 W/kg). The mean performance of the third monkey at the higher level was about 80% of its mean control performance. At 72 mW/cm^2 (7.2 W/kg), all 3 monkeys performed at approximately 50% of their respective control values. The results suggest that the monkeys had reacted to body heating

by the RFR at the higher levels and that their performances were diminished because of such heat.

De Lorge (1979) trained four squirrel monkeys in 1-h sessions to press either the right or the left lever on top of a chair to obtain a food pellet. Initially, each successive lever press resulted in the alternate activation of a red light and a blue light in front of the monkey. When the monkeys achieved consistent performance in such lever presses, the contingencies were changed so that press of the left lever was rewarded only when the blue light was on, with right-lever presses continuing to alternate the red and blue lights.

Training progressed in stages, with each stage requiring a higher number of right-lever presses to turn on the blue light. The last stage was a schedule in which each right-lever response yielded either 0.5 s of red light or 10 s of blue light, and only a left-lever press during the latter yielded a pellet.

After stable behavior was achieved, each monkey was exposed from above to 2.45-GHz RFR at levels in the range 10-75 mW/cm^2. SARs were estimated to have been 0.5 to 3.75 W/kg. In 41 daily sessions, exposures were done during the middle 30 min of 1-h testing sessions, with the other two 15-min periods for obtaining baseline data. In the next 53 sessions, only 3 of the monkeys were tested, the session duration was 2 h, and the RFR exposures were during the middle 1 hour. The number of sessions at each level ranged from 2 to 5, with sham exposures between sets. Neither the 30-min nor the 60-min exposure regimens caused any obvious permanent physical changes in any of the monkeys.

Among the various performance measures, only the rate of right-lever responses showed an RFR-induced change. This measure exhibited a slight trend toward lower rates with increasing RFR level, to a minimum of about 90% of mean control value at 60 mW/cm^2 (3.0 W/kg), and a slightly higher value (92%) at 70 mW/cm^2 (3.5 W/kg). However, the response rate never exceeded one standard deviation from 100%. The behavioral effects of 1-h exposures were similar to those of 30-min exposures but were more pronounced. No consistent behavioral changes occurred below 50 mW/cm^2 (2.5 W/kg); above that level, the effects increased with RFR level. The right-lever-response rate versus RFR level varied widely among the 3 monkeys, but at 60 mW/cm^2 (3.0 W/kg), all showed decrements to about 60%.

The author concluded that the observed behavioral changes in the squirrel monkeys were temporary and clearly related to hyperthermia. Consistent changes were seen when rises in rectal temperature exceeded 1 °C, which corresponded to a threshold between 40 and 50 mW/cm^2 (2.0-2.5 W/kg). The author noted that similar results had been obtained with rhesus monkeys tested for the same behavioral task during exposure to 2.45-GHz RFR, but with a threshold 10 to 20 mW/cm^2 higher, and suggested that RFR-induced behavioral changes in different species may be scaled on the basis of body mass.

The findings of this study, reinforced by the similar results with rhesus monkeys, are important because the measurements of performance of a complex behavioral task during exposure to RFR were carried out with two species much closer to human physiology and intelligence than more commonly used nonprimate laboratory animals, and because reasonably accurate RFR thresholds for each primate species were determined.

De Lorge (1984) similarly trained food-deprived rhesus monkeys to perform a task in which each monkey was to press a lever in front of its right hand (an observing response), which

produced a 0.7-s low tone (860-1000 Hz) to signal that no food pellet will be delivered, or a high tone (1250-3703 Hz) for up to 1.2 s to signal the availability of a pellet. If the monkey pressed a lever in front of its left hand while the high tone was on (a detection response), the tone would cease and a pellet would be delivered. A left-lever response at other times produced a 5-s interval during which presses of the right lever yielded only the low tone. If the left lever were not pressed during 1.2 s of the high tone, that tone would cease and the reinforcement schedule would recycle. No tones were presented without a lever press, and right-lever presses during the presence of either tone had no consequences.

The low tone was delivered most frequently and the high tone was sounded at random times at an average of about once every 30 s. Reinforcement was at random intervals of about 1 minute initially, and the intervals were shortened as the responses became more efficient.

After several sessions of stable performance, each monkey, while seated in a Styrofoam restraining chair, was frontally exposed, during 1-h sessions, to vertically polarized 225-MHz CW RFR (near the whole-body resonant frequency), or to pulsed RFR at 1.3 GHz or 5.8 GHz (both above whole-body resonance).

Estimates of normalized SAR were derived from exposure of saline-filled models to 225 MHz and 1.3 GHz, and of models filled with tissue-simulating materials to 5.8 GHz. The results, in W/kg per mW/cm^2, were 0.4 for 225 MHz, 0.13 for 1.3 GHz, and 0.03 for 5.8 GHz. The exposures to 225 MHz were at 5-11 mW/cm^2 (2.0-4.4 W/kg). The 1.3-GHz RFR consisted of 3-μs pulses, 370 pps, at 20-95 mW/cm^2 average (2.6-12.4 W/kg); the 5.8-GHz RFR consisted of 0.5-μs or 2-μs pulses, 662 pps, at 11-150 mW/cm^2 (0.34-4.7 W/kg). Rectal temperature was monitored continuously during each session with a nonperturbing probe.

For each frequency, each monkey was exposed three times at each RFR level, with the levels usually sequenced in ascending order, but all RFR sessions were followed with sham-exposure sessions. Four of the monkeys reduced their rates of incorrect detection responses (on the left lever) to low, stable levels. The fifth, subject 10, made excessive numbers of incorrect detection responses throughout the study, which were sometimes greater than its observing-response rate (on the right lever).

Reductions in observing-response rates occurred at RFR levels above threshold. For example, one monkey (subject 13) exhibited decreases in observing-response rates during exposure to 1.3-GHz RFR at 50 mW/cm^2 and higher, and the rate reduction became larger toward the latter part of each session as the RFR level was raised. Also observed were response patterns that became increasingly erratic during sessions – an effect most pronounced at 225 MHz, at which the monkeys paused for as much as 15 min and often stopped responding at all for the last half of a session at 10 mW/cm^2.

As a more definitive index of behavioral change than the individual cumulative records, plots of the mean and standard error of the ratio of observing-responses during each RFR-exposure session at each frequency to the values during the preceding sham-exposure session versus the power density were shown. The results for each frequency yielded a threshold power density for statistically significant behavioral alterations that increased with frequency: 7.5 mW/cm^2 at 225 MHz, 63 mW/cm^2 at 1.3 GHz, and 140 mW/cm^2 at 5.8 GHz. However, the corresponding threshold whole-body SARs varied up and down with frequency: respectively 3, 8.2, and 4.3 W/kg, presumably because of differences in penetration depth.

The detection-response rate on the food lever was not consistently affected by RFR exposure at any frequency. No effect was observed for 225 MHz or 5.8 GHz; for 1.3 GHz, a decreased response rate was observed occasionally, but only at 83 mW/cm^2 or higher. However, plots of mean ratio of detection-response latencies during RFR exposure to detection-response latencies during sham exposure versus power density exhibited values slightly but significantly higher than 1 at all three frequencies and at most power densities. For each frequency, however, the mean ratio changed both upward and downward with power density, but with an overall downward trend.

Postreinforcement pause (a pause following a reinforced detection-response) was also affected. The mean ratio during exposure to 225-MHz RFR to that during sham exposure was 1.0 in the range 5-7.5 mW/cm^2, but rose significantly to 1.5 at 10 mW/cm^2. The changes for 1.3 GHz were both upward and downward, but nonsignificant up to 63 mW/cm^2, at which the mean ratio was 1.3. Above 63 mW/cm^2, the mean ratio decreased to 1.1 at 93 mW/cm^2; the latter ratio was still significantly larger than 1.0. The only significant change for 5.8 GHz was at 150 mW/cm^2, to 1.06, a smaller increase than for the other frequencies.

The mean colonic temperature at the start of the 1-h sessions rose an average of 0.15°C during the sham-exposure sessions. For 225 MHz, the temperature rises were linear with RFR level, from 0.8°C at 5 mW/cm^2 to 2.1°C at 10 mW/cm^2. With 1.3 GHz, the rises were less than linear, from 0.4°C at 20 mW/cm^2 to 1.9°C at 93 mW/cm^2. With 5.8 GHz, the rises were even more gradual, from 0.2°C at 10 mW/cm^2 to 1.0°C at 150 mW/cm^2.

The author's estimates of absolute thresholds for the disruption of observing-response rates for each frequency were 8.1 mW/cm^2 for 225 MHz, 57 mW/cm^2 for 1.3 GHz, 67 mW/cm^2 for 2.45 GHz (from de Lorge, 1976), and 140 mW/cm^2 for 5.8 GHz – values that rose with frequency. However, the corresponding SARs, 3.2, 7.4, 6.7, and 4.3 W/kg, varied both upward and downward with frequency, perhaps reflecting penetration-depth differences again.

The results led the author to remark that predictions of biological effects based only on power density are poor and that predictions from normalized whole-body absorption of energy are not very useful. He also noted that because the ratio of highest-to-lowest threshold SAR is much smaller than the ratio of the corresponding power densities, SAR is a more efficient predictor than power density, but that both are frequency-dependent. Thus, he concluded that an increase of about 1 °C in colonic temperature is a more reliable single index of behavioral disruption.

The author speculated that the 225-MHz results reflect a resonance heating effect of the blood in the entire body, causing great difficulty in thermoregulation, because heated blood cannot be replaced with cooler blood. He also suggested that the results at 1.3 and 5.8 GHz illustrate normal thermoregulation, because the limbs or skin are heated much more at those frequencies than the interior of the head.

The author did not discuss of the possible occurrence of the RFR-auditory effect with the 1.3-GHz and 5.8-GHz pulsed RFR. He presumably discounted this effect as a factor in the results, because the pulse repetition rates used (370 and 662 pps) were lower than the tones used in the behavioral paradigm.

D'Andrea et al. (1989) trained five food-deprived rhesus monkeys to operate three levers (left, right, center) in various sequences to obtain food pellets. The sessions were 60 min long.

The task during each session comprised three successive 10-min schedules of lever presses, followed by repetition of the same 3 schedules. About 60 baseline sessions were given before the start of sham- or RFR exposures.

In the first 10-min schedule, the monkey was required to withhold responding for 8 s after the start of a 1070-Hz tone, and then to respond only within the next 4 s; the correct response during those 4 s was 2 presses of the left lever within 2 s of each other. The authors called this an interresponse-time (IRT) schedule.

The second 10-min period was devoted to a time-discrimination (TD) schedule, in which a press of the center lever in the presence of blue light randomly yielded white light of short duration (1-3 s) or long duration (8-10 s). At the end of either period, the white light was replaced with red and green light. When the red and green light were present, the monkey, to obtain a pellet, had to press the right lever if the preceding white light was of short duration or the left lever if the preceding white light was of long duration.

During the third 10-min period, a fixed-interval (FI) schedule was used: The monkey was presented with a continuous 2740-Hz tone, and its first press of the right lever after 55 s yielded a pellet.

During the 60-min behavioral test sessions, each monkey was sham-exposed or exposed from above to 3-μs pulses of 1.3-GHz RFR at a RMS pulse power density of 131.8 W/cm^2. The peak SAR was 15.0 W/kg in the head and 8.3 W/kg whole-body. The pulse repetition rate was 2, 4, 8, 16, or 32 pps, with corresponding average power densities of 0.92, 1.85, 3.70, 7.40, or 14.80 mW/cm^2.

The results showed no significant differences between sham- and RFR-exposures in any of the behavioral responses. The authors noted that the the energy absorbed in the head by each pulse (280 mJ/kg) was well above the threshold for the RFR-auditory effect, and remarked that if such auditory stimulation did occur, it produced no obvious effect on the trained behavior (see Section .

B.3.7.2 RFR and Drugs

Various studies have been conducted on possible interactive effects of exposure to RFR and medications or other drugs taken or administered. Those discussed below are representative.

Thomas et al. (1979) trained four food-deprived rats on a fixed-interval, 1-min (FI-1) schedule to press a bar for a pellet. After stable baseline patterns were achieved, an effect-versus-dose function for the psychoactive drug chlordiazepoxide (tradename Librium), given 30 min before a session, was established. This function showed that the responding rate rose with increased drug dose up to 10 mg/kg, attaining 2-3 times the baseline rate at that dose. At still higher doses, the responding rate decreased, attaining zero at 40 mg/kg.

After training, the rats were exposed to 2.45-GHz RFR at 1-W/cm^2 peak, 1-mW/cm^2 average (0.2 W/kg) during the 30 min before each bar-pressing session (starting immediately after drug injection). RFR exposure yielded the same shape of effect-versus-dose function, but the magnitudes were generally higher by a factor of about 2. By contrast, RFR exposure in the absence of any drug injection produced no difference in responding rate.

The results of this investigation are unequivocal, but the mechanisms are obscure. For example, although average power density and whole-body SAR were low, local SARs in brain regions that are target areas for central actions of chlordiazepoxide may have been high enough for a thermally potentiating effect. It is also conceivable that the pulse parameters produced the RFR-hearing effect during the 30-min pre-session period. If so, however, it is not clear whether or how any influence of this effect would have carried over into sessions during which RFR was absent.

Thomas and Maitland (1979) also trained 6 food-deprived rats to depress a lever on a schedule in which a second response at least 18 s after a first response was rewarded with a pellet, but a second response in less than that time interval reset the timing period. After such training, effects of exposure to the 2.45-GHz pulsed RFR (0.2 W/kg) used previously were sought on the dose-response function of the psychoactive drug d-amphetamine.

Three of the rats were dosed with the drug once per week and exposed for 30 min (single-exposure condition). Their behavior was observed for 1 hour right after exposure for any direct drug-RFR interaction. For detection of possible cumulative action of the RFR, the other three rats were dosed with d-amphetamine once a week and exposed for 4 d/wk, 30 min/d (multiple-exposure condition), except on drug-injection days. On those days, their behavior was observed for 30 min after injection. The sessions were conducted for 13 weeks, and included sham exposures and saline injections for all 6 rats.

For the three rats studied under the single-exposure condition, the mean response rates (in total responses per minute) after injection of saline and sham exposure, and after injection of saline and RFR exposure, were comparable to baseline performances. When those rats were given d-amphetamine and sham-exposed, their mean response rates rose with drug dose to a maximum at 2.0 mg/kg, with consequent reductions in the frequency of correct responses that yielded reinforcement. At higher doses, the mean response rates dropped sharply, to zero for 4.5 mg/kg.

By contrast, the mean total response rate of those rats dosed with the drug and exposed to the RFR rose to values significantly higher than for the corresponding doses with sham exposure, with maximum at 0.5 mg/kg. Above 0.5 mg/kg, the mean total response rate declined sharply, to zero for 1.5 mg/kg. Those results show that RFR exposure after injection of a given dose of d-amphetamine yielded behavior similar to that obtained with a larger dose without RFR exposure.

For the three rats studied under the multiple-exposure condition, the mean baseline performance and mean performances for saline injection followed by sham- or RFR exposure did not differ significantly from the values for the other saline-injected rats. The dose-response functions of the rats with and without multiple RFR exposures were qualitatively similar to those with and without single RFR exposures, even though the performances of the former group were determined 24 h after the final exposure. With the multiple sham exposures, maximum responses were obtained for 2.0 mg/kg, with a sharp decline to zero for 4.5 mg/kg. The maximum responses for the multiple RFR exposures were obtained with 0.5 mg/kg, and the responses declined sharply to zero for 2.0 mg/kg.

The authors remarked that the modest average power density (1 mW/cm^2) may have produced relatively high local SARs, particularly by resonant absorption in the head, which could have selectively heated the brain. In addition, head resonance could have yielded energy

values above the threshold for the RFR-hearing effect. However, the authors discounted those possibilities because they would not account for the persistence of behavioral effects for 1 hour after the single exposures and for 25 h after the last of the multiple exposures.

The effects of body restraint, which could synergize with low RFR levels and stressful events to produce considerable elevations of body temperature, were considered and discounted because restraint of the rats injected with saline and exposed to RFR yielded no significant deviations from the baseline values, and the dose-effect functions of unrestrained and restrained rats not exposed to RFR were the same.

Because d-amphetamine has been reported to heighten human perception with various senses, it could be hypothesized that rats can perceive lower levels of RFR under the drug's influence than without the drug, and that such drug-induced perception would change their behavior. However, this hypothesis would not account for the 24-h persistence of RFR influence seen in the multiple-exposure group.

Thomas et al. (1980) described similar research with the drugs diazepam and chlorpromazine. Diazepam (tradename Valium) has been widely prescribed as a tranquilizer and muscle relaxant. Chlorpromazine is used as a sedative and as an antiemetic.

Four food-deprived rats of two strains were trained on a FI-1 schedule of reinforcement. After training, the dose-effect functions for diazepam were determined in one strain and for chlorpromazine in the other strain. One dose was injected 30 min before each session and the doses were administered in mixed order, with at least 3 replications for each dose. Response rates and patterns were compared with their corresponding baseline performances.

Chlorpromazine lowered performance with increasing dose for all four rats administered that drug. The response rates stayed within baseline variability for doses up to about 1 mg/kg and declined for higher doses. For those given diazepam, the drug caused slight increases in response rate at doses up to about 2.5 mg/kg, with decline at higher doses.

Having determined dose-effect functions for the drugs, the authors exposed each rat for 30 min to 2.8-GHz pulsed RFR at 0.2 W/kg immediately after administering each drug, and tested the rats at exposure end. The RFR did not alter the effects of chlorpromazine or diazepam, in contrast with the results with chlordiazepoxide and d-amphetamine. Such differences in findings are difficult to reconcile.

Pappas et al. (1983) exposed rats in separate waveguides to pulsed 2.45-GHz RFR for 45 min at 1 mW/cm^2 average power density (SAR 0.6 W/kg), and studied the effect of the RFR on stereotypy (persistent senseless movements) induced by d-amphetamine. During a 1-h session following exposure and starting 4 min after d-amphetamine injection, each rat was observed for 3 normal behaviors (immobility, rearing, forward walking) and 3 abnormal behaviors (backward walking, circling, head swaying) for 1 minute every 5 min. The occurrence of each behavior was recorded on an all-or-none basis, and the total incidence of each was determined for each rat. The difference in average score for each of the six behaviors between RFR and sham groups was nonsignificant.

Lai et al. (1984) described experiments to determined the effects of the same RFR used by Pappas et al. (1983) on ethanol-induced hypothermia and ethanol consumption. For the ethanol-hypothermia experiment, 15 rats were RFR exposed and 14 were sham-exposed for 45 min.

Immediately after exposure, each rat was removed from its waveguide, its colonic temperature was measured, and it was injected with a solution of ethanol (3 g per kg of body weight in 25% of water by volume). The rats were then housed 6 to a cage, and their colonic temperatures were measured with a thermistor inserted and removed at 15-min intervals for 120 min.

Mean colonic temperatures of the RFR- and sham-exposed rats immediately after exposure were respectively 38.2 ° and 38.3 °C, a nonsignificant difference. Ataxia developed within 5 min of ethanol injection, but righting reflex remained intact. The mean temperature changes versus time after ethanol injection for the two groups showed that hypothermia had occurred in the RFR group at a slower rate than in the sham group. For example, the temperature depressions at 15 min after injection were about 0.4° and 0.9°C for the RFR and sham groups, respectively, a significant difference; at 60 min, the corresponding depressions were about 1.5° and 1.8°C, also a significant difference; at 90 min, the depressions were 1.9°C for both groups and did not differ significantly at subsequent times.

In the ethanol-consumption study, rats were given 90-min sessions in the waveguides daily for 9 days. Drinking water was removed from the home cages 24 h before the first session. On session days 1, 2, and 3, the rats were inserted in the waveguides for 45 min with the RFR source on "standby." At this time, a bottle containing a 10% sucrose solution was inserted in each waveguide and the amount consumed during the remaining 45 min was measured. The procedure on day 4 was the same except that half the rats (24) were selected randomly and exposed to the RFR, and the other half were sham-exposed for the full 90 min. On days 5-7, the procedure was the same as on days 1-3, except that a 15% ethanol + 10% sucrose solution was used to render the ethanol more palatable.

On day 8, half the rats (group I, randomly selected) were exposed to RFR and the remaining rats (group II) were sham-exposed for 90 min, and the amounts of sucrose-ethanol solution consumed during that period were measured. On day 9, the group roles were reversed: group I was sham-exposed, group II was RFR exposed, and fluid consumption was noted.

For days 1, 2, and 3 (during which all 48 rats were sham-exposed and offered the sucrose solution), the mean sucrose consumption rose significantly each successive day. On day 4, however, (when half were RFR exposed and the others sham-exposed for 90 min), the mean sucrose consumptions for the 2 groups did not differ significantly from each other or from the day-3 value.

For days 5, 6, and 7 (when all 48 rats were sham-exposed and offered the sucrose-ethanol solution), the mean sucrose-ethanol consumption varied up and down with time. For day 8 (when group I was RFR exposed and group II was sham-exposed), sucrose-ethanol consumption by group II did not change significantly but that of group I significantly increased. For day 9 (when group II was RFR exposed and group I was sham-exposed), consumption by group II significantly increased. Thus, RFR exposure had no apparent effect on sucrose consumption, but increases in sucrose-ethanol consumption were linked to RFR exposure.

B.3.8 Cellular and Subcellular Effects

Various studies on cellular and subcellular effects of RFR have been discussed above under other specific topics such as the BBB, immunology, and hematology. This subsection describes representative studies of other RFR effects on cells and their constituents.

B.3.8.1 Structures and Constituents of Microorganisms and Other Single-Cell Systems

Webb and Dodds (1968) sought effects of RFR at frequencies above 30 GHz (millimeter waves) on the growth of *E. coli* bacteria. The results appeared to indicate that bacterial growth was inhibited by 136-GHz RFR. No statistical treatment was given, but examination of the data indicated the likelihood that non-RFR factors were present. Webb and Booth (1969) also reported RFR absorption by *E. coli* cells, and by preparations of *E. coli* protein, and ribonucleic acid (RNA) deoxyribonucleic acid (DNA) at specific (resonant) frequencies in the range 65-75 GHz. The latter findings were difficult to evaluate because of absence of adequate information on methodology, instrumentation, and statistical treatment.

Several studies sought to confirm predictions by Fröhlich (1975) of resonances above 30 GHz. In one study, Webb and Stoneham (1977) reported the detection of resonances in the range 70-5000 GHz in active cells of *E. coli* and *Bacterium megatorium*, using laser Raman spectroscopy. They found no resonances in resting cells, cell homogenates, or nutrient solutions, and therefore associated resonances for active cells with metabolic processes.

Grundler et al. (1977) investigated the effects of RFR in the range 40-60 GHz on growth of yeast cells (*Saccharomyces cerevisiae*). After appropriate preparation, 2.5 ml of yeast suspension in an aqueous medium was placed in a standard rectangular glass receptacle, and cell growth was monitored by transmission photometry. Extinction (decrease of light transmission through the suspension) was recorded versus time and was found to increase exponentially over approximately three generations (about 4 hours). The data were replotted semilogarithmically to obtain the growth rate (slope of the new plot).

Growth curves with no RFR were obtained at suspension temperatures in the range 30.5-34°C. (The authors noted that in any experiment, suspension temperature never varied by more than ±0.5°C.) The growth rates therefrom were plotted versus suspension temperature; these plots showed that the growth rate decreased from 4.0% per 1°C at 31°C to 1.3% at 33°C, and that the results were reproducible to within ±3%.

The RFR source was a backward-wave oscillator in the 40-60 GHz range, with frequency stability and resettability, respectively, of ±1 MHz and 3 MHz (equivalent to ±25 and 75 parts per million at 40 GHz). The output of the oscillator was fed through a metallic waveguide to a vertical horn terminated by a Teflon structure that was immersed in the aqueous suspension. For depth of penetration into water of about 0.2 mm at 42 GHz, the RFR power emitted into the suspension ranged between 11 and 27 mW. Because the total area of emission was stated to be 10 cm^2, the power densities were in the range 1.1-2.7 mW/cm^2, which increased suspension temperature by roughly 0.4°C. The relative or normalized growth rate was then defined as the ratio of the growth rate in the presence of RFR to the rate in its absence at the corresponding suspension temperature.

The authors performed 67 experiments; they discarded the results of 5 because the growth rates were very small in the samples studied and in a monitor beaker, probably because of bacterial infection or chemical poisoning. Those of the other 62 experiments were tabulated as normalized growth rate at specific frequencies, suspension temperatures, and absorbed RFR powers (which varied from 11 to 27 mW). The normalized growth rate was also plotted versus frequency. Evident in that figure were several sharp maxima and minima spanning unity growth rate ("a multiplet of biological resonances") in the frequency region 41.83-41.96 GHz, with little

effect below or above the region. The largest increase in growth rate was 15% at 41.682 GHz ±3 MHz, for 17 mW absorbed power and 32.4°C suspension temperature. The largest decrease was 29% at 41.712 GHz ±3 MHz, for 23 mW and 31.7°C. (The figure showed one smaller minimum and maximum at intermediate frequencies.)

The authors noted that on an absolute frequency scale, a systematic offset of up to ± 20 MHz was possible. They also stated:

> 'The strong absorption of the radiation in water means that in our geometry only a small part of the total volume is subjected to the full intensity. We have not yet measured the dependence on intensity and thus would not know how to correct the results for the varying power. Inspection of the figure shows, however, that this can cause only minor alterations even if linear dependence on power is assumed.'

The results of this study seem to indicate that RFR in the resonant range either enhances or inhibits cell growth, depending on the specific frequency, but the authors did not speculate on possible mechanisms for such reversals of effect.

Cooper and Amer (1983) disputed the findings above, indicating that cell suspensions yield spurious Raman lines in the frequency range of interest under certain conditions, notably by Mie scattering from cell clumps, and that they thereby were able to reproduce many of the spectra.

Gandhi et al. (1980) used a stable, computer-controlled system to measure RFR absorption in various biological specimens at frequencies in the range 26.5-90.0 GHz in small steps. They studied solutions of DNA from salmon sperm and RNA from whole yeast and yeast-like fungi, and suspensions of *E. coli* cells and baby-hamster-kidney cells transformed with mouse sarcoma virus. Those results showed no resonances at any frequency sampled, strongly suggesting that none of those biological materials absorb significant RFR energy in that range.

Swicord and Davis (1983) used a new method for measuring absorption by optically transparent liquids and for studying interactions between cellular constituents and RFR at frequencies below (as well as above) the millimeter range. They measured RFR absorption in the range 8-12 GHz by aqueous solutions of DNA extracted from *E. coli*. A plot of attenuation coefficient for DNA versus frequency exhibited no resonances, but the attenuation increased linearly with frequency and its values were much higher than for physiologic (Ringer's) solution or deionized water at the same frequencies.

Edwards et al. (1985) noted that biochemical analysis of the DNA solution used in the previous study indicated the presence of significant amounts of RNA and protein impurities, and that the DNA had been sheared extensively by improper handling. In addition, the enhanced absorption observed for such samples in the range 8-12 GHz was absent for carefully prepared DNA samples of high molecular weight that were free of protein and RNA.

Gabriel et al. (1987) described the efforts in London and Uppsala by two independent laboratories to detect resonances in the range 1-10 GHz for aqueous solutions of circular DNA molecules of the same form studied by Edwards et al. (1985). The dielectric measurements in London were done on an automated time domain spectrometer using a reflection technique; those in Uppsala were done with a similar spectrometer, but with a transmission technique. The authors noted that a common and most important feature of such measurements is the use of a reference

sample to normalize reflection or transmission coefficients, thus eliminating systematic experimental artifacts, such as slight impedance mismatches.

Plots of relative permittivity and loss factor of a 0.1% plasmid DNA solution at 20 °C versus frequency obtained in London were displayed on linear scales, to permit direct comparison with those of Edwards et al. (1985). Those from Uppsala were shown as the more common log-log plots. Each point was the average of up to 36 measurements on samples from 4 plasmid preparations; no variances were shown, but the authors estimated the measurement uncertainties to be about 1% for permittivity and 2% for loss factor. Also shown on each plot were lines of relative permittivity and loss factor for pure water. Within those limits, the measured values of permittivity and loss factor for DNA were all close to those for pure water.

The values of attenuation coefficient and incremental attenuation coefficient relative to water were plotted versus frequency; also displayed were those of Edwards et al. (1985). Unlike the results for the latter, no resonances were evident; the incremental attenuation values were scattered above and below the zero line, with the largest deviations less than ±0.1/cm, primarily in the upper 20% of the frequency range. By contrast, the incremental attenuation values by Edwards et al. (1985) at their reported resonant frequencies ranged from 0.28 to 0.7/cm.

Foster et al. (1987), in another endeavor to reproduce the findings of Edwards et al. (1985), used two techniques. In one technique, which was a variant of that used by Edwards et al. (1985), a probe consisting of the open end of a length of coaxial line was immersed in the sample, and the complex reflection coefficient at its tip was measured with an automatic network analyzer (ANA) in the frequency range 0.045-18 GHz in 0.045-GHz increments. The authors surmised that the previously reported apparent resonances could be ascribed to two sources of error associated with such probe measurements. First, the coaxial connector to the probe could be a source of artifactual reflection. Accordingly, they performed the measurements with and without use of a time-domain-gating procedure for removing connector artifact. Second, analyses of the measurements did not account for radiation from the probes they and Edwards et al. (1985) used . In the other technique, the sample was placed inside a section of 7-mm coaxial transmission line between a Teflon disk and a short circuit terminating the line, thus avoiding probe radiation, and the reflection coefficient from the sample was measured with an ANA.

With the probe method and no time-domain gating to remove connector artifact, solutions of DNA having about threefold higher concentration than those used by Edwards et al. (1985) yielded reflection-coefficient oscillations crudely resembling the resonances reported by the latter; however, those oscillations were eliminated by the time-domain gating. No apparent resonances were seen with the transmission-line technique.

Sagripanti et al. (1987) reported that plasmid DNA (derived from *E. coli* and purified to ensure the absence of protein contaminants), when exposed to low-levels of RFR in the frequency range 2.00 to 8.75 GHz, exhibited both single-strand and double-strand breaks, but only if small amounts of copper ions (cuprous but not cupric) were present. Samples consisted of 10 micrograms (μg) of plasmid DNA in 28 microliters (μl) of buffer, with each sample contained within a 1.5-ml micro test tube.

Exposures were made by immersing an open-ended coaxial probe into each sample. The probe consisted of a solid outer conductor 3.58 mm in diameter and a central conductor 1 mm in diameter, both of copper, with solid dielectric between them and with a flush open end.

Attenuation and standing-wave ratio were measured with a dual-directional coupler and a slotted line inserted between a generator of CW RFR and the probe. The results were used to determine the maximum and minimum SARs (SAR_{max} and SAR_{min}). Those data indicated that SAR_{max} was about 5 times larger than SAR_{min}, with SAR_{true} somewhere between the two. The experimental results were referenced to the values of SAR_{max}.

In the first experiments, samples were sham-exposed or exposed for 20 min to 2.55-GHz RFR at an SAR_{max} of 10 W/kg. The results of six experiments showed that the mean number of double-strand breaks in the RFR-exposed samples was significantly higher than for the sham-exposed samples. The authors regarded such exposures as nonthermal, because of the large surface-to-volume ratio of the samples and thus their ability to dissipate heat readily; the authors indicated that exposures at levels of about 1 kW/kg were needed to detect any significant temperature rises in the samples.

In other experiments seeking frequency specificity of the effect, samples were exposed to RFR at 8.75 GHz (a frequency along with 2.55 GHz that had been found to produce maximum resonant absorption by DNA) and to 2.00-GHz, 3.45-GHz, and 7.64-GHz RFR, which were frequencies of minimum absorption (Edwards et al., 1985). The authors stated that they could not find any variation in double-strand breaks attributable to resonant absorption by DNA.

For statistical analysis, the authors pooled data on 12 experiments at the 5 frequencies above. The results showed a significantly higher mean percentage of double-strand breaks for the RFR-exposed samples than the sham-exposed samples. However, the mean percentage of double-strand breaks for the sham-exposed samples was also significantly higher than for control samples, for which the copper probe was close to the sample but not in contact with it. When the probe was covered with a thin plastic coating, the difference between sham-exposed and control samples vanished, but also no strand breaks were detected in RFR-exposed samples.

In other experiments, samples were incubated in either cupric or cuprous chloride, or in the storage buffer (controls), and not RFR- or sham-exposed. The results indicated that only incubation in cuprous chloride mimicked the strand breaking seen with RFR exposure. On the basis of linear increases of damage with exposure duration, the authors concluded that the presence of cuprous chloride (in the probe) causes the strand breaking and that the RFR increases the effect.

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APPENDIX C

COMMENT LETTERS

Many letters commenting on the Draft Supplemental Environmental Assessment (SEA) of the Effects of Electromagnetic Radiation from the WSR-88D were received during the comment period held from March 28 through May 26, 1992. Those letters that contained comments requiring responses or that provided information augmenting the Draft SEA are reprinted here. Appendix D sets forth responses to comments in those letters. Letters that do not contain substantive comments and do not require a response are preceded by an asterisk (*) in the list below, and are not reprinted in this document.

1. Norman C. McLaughlin, Bennett Heights, New York, April 21, 1992
2. Rev. Robert L. Smith, Pastor, New Hope Fellowship, Batavia, New York, April 15, 1992
3. Mary Beth Hoffarth, Sonshine Patch Child Care, Batavia, New York, April 1992
4. *Anthony Deni, Bennett Heights, New York [No date]
5. Arlene Bow, RN, North Bennett Heights, New York, April 16, 1992
6. David T. Powell, National President, NWSEO, [No address or date]
7. Susan Lukaszewicz, Batavia, New York, [No date]
8. *John J. Lukaszewicz, Batavia, New York, [No date]
9. Sarah C. Lukaszewicz, Batavia, New York, [No date]
10. *Christina Petrone, Elba, New York, April 17, 1992
11. *Donald C. Nelson, Elba, New York, April 17, 1992
12. Dianne M. Puccio, [No address], April 18, 1992
13. Gloria McLaughlin, Bennett heights, New York, April 21, 1992
14. Florence C. Gioia, [No address], April 17, 1992
15. June C. Vukman, Supervisor, Town of Batavia, New York, April 14, 1992

16. Gary C. Plan, [No address], April 21, 1992

17. Catherine J. Dusen, Batavia, New York, [No date]

18. Dan Voltura, CONRAD III Project Coordinator, [No address], April 21, 1992

19. D. Keith Bow [No Return Address], April 21, 1992

20. Roshan Shaikh, East Yaphank Civic Association, May 25 ,1992

21. *_____, State of Illinois, Office of Governor, April 24, 1992

22. *_____, State of Vermont, Office of Governor, April 24, 1992

23. *_____, State of Mississippi, State Clearinghouse for Federal Programs, April 29, 1992

24. *Linda Wise, State Clearinghouse, State of Ohio Office of Management and Budget, [No date]

25. *William T. Quigg, Intergovernmental Review Coordinator, Policy Development and Planning Division, State of Connecticut, April 13, 1992

26. *Carol Whiteside, Assistant Secretary, Intergovernmental Relations, The Resources Agency of California, May 15, 1992

27. *Louis Pohl, Coordinator, Missouri Clearinghouse, May 4, 1992

28. *Kathy Reis, Assistant Grants Coordinator, State of South Carolina Office of the Governor, April 22, 1992

29. Harold S. Masumoto, Director, Office of State Planning, Office of the Governor, State of Hawaii, May 5, 1992

30. *Margaret Dubas, Staff Assistant, Colorado State Clearinghouse, State of Colorado, May 21, 1992

31. Chrys Baggett, Director, North Carolina State Clearinghouse, May 22, 1992

32. Don N. Strain, Director, Oklahoma Department of Commerce Office of Federal Assistance Management, May 14, 1992

33. *Charles H. Badger, Administrator, Georgia State Clearinghouse, Office of Planning and Budget, May 15, 1992

34. *Charles H. Brown, Director, State Clearinghouse Tennessee State Planning Office, May 18, 1992

35. Barry J. and Catherine M. Debbins, Batavia, New York, May 19, 1992

36. *Kenneth W. Holt, M.S.E.H., Special Programs Group, National Center for Environmental Health and Injury Control, Atlanta, Georgia, May 6, 1992.

37. Joel Ray, Ithaca, New York, May 12, 1992

38. Richard E. Sanderson, Director, Office of Federal Activities, United States Environmental Protection Agency, Washington D.C., May 26, 1992

39. George Morgan and Cindy Kushner, Elba, NY, May 17, 1992

40. Cindy Kushner, Elba, NY, May 17 ,1992

41. *Jami Owens, Illinois State Clearinghouse, May 26, 1992

42. Danna G. Sturm, Coordinator, State Clearinghouse, State of Nevada, May 21, 1992

43. *Kathy Reis, Assistant Grants Coordinator, State of South Carolina Office of the Governor, May 28, 1992

44. *Jeanette Tomczak, North Carolina State Clearinghouse, [No date]

45. Joe A. Elder, Ph.D., and Carl F. Blackman, Ph.D., U.S. Environmental Protection Agency Health Effects Research Laboratory, October 8, 1992

Letter # 1

April 21, 1991

TO: The National Oceanic and Atmospheric Administration

FROM: Norman C. McLaughlin
4421 North Bennett Heights
Batavia, NY 14020
716-343-9116

Thank you for the invitation to this informal public hearing on the DRAFT SEA regarding the Next Generation Weather Radar, also designated as WRS-88D. Thanks also to the people at the National Weather Service who have been most gracious in facilitating my presence here today.

I am here representing myself and the members of CONRAD III, a citizen's coalition opposed to the proposed siting of a weather radar tower at the Batavia NY airport.

On page 1 of the EXECUTIVE SUMMARY of the SEA, the text reads: "The WSR-88D Program consists of the design, siting, construction, and operation of the 115 NWS radars in the continental United States."

We take no exception to the need for better weather reporting. We are not happy that in our area the NWS is using equipment from 1957. We heard, however accurately, that until recently the vacuum tubes for this system were available only from a factory in the former Soviet Union. That's sad.

..."the design, construction and operation of the 115 NWS radars in the continental United States" (Supplemental Environmental Assessment, p.1) are not an issue for us. We are concerned solely with SITING.

The proposed site for the radar tower in Batavia NY is partially on Genesee County Airport property. The site is within a few hundred feet of the New Hope Fellowship Church and its Day Care Center. It is within one and half miles of approximately 262 residences.

So that you may clearly understand our stance, we say that if there is a question about the health risks from this non-ionizing radar source, it is better to err on the side of safety. If people may be harmed, move the tower away from people.

We are totally astonished that, although we admit there are two sides to this argument about safety from the electromagnetic field, SRI of California, the author of this study has said, "no significant impact." If SRI worked in the US mint, they might produce the first one sided coin.

We say, if there is to be an error in the siting of these radiating sources, let it be on the side of safety for people who live, attend school or work proximate to the WSR-88D tower. This tower will be radiating 24 hours per day for the next 20-25 years.

My wife and I have been studying this issue for 26 months. We have gathered data from innumerable sources and, inductively, have arrived at our conclusions. No one has reviewed my comments, except my wife. I speak as a layman on these matters.

THE MOSCOW STORY

STATEMENT IN SEA: pp. B-16 and B-17

"The U. S. Embassy in Moscow was irradiated with low-level RFR from 1953 until February 1977. Lilienfeld et al (1978) conducted a study on the health of U. S. personnel assigned to the Moscow embassy during that period. The authors identified 1827 employees and 1228 dependents as having been at that embassy during the 1953 - 1976 period.

The controls consisted of 2581 employees and 2072 dependents assigned to embassies and consulates in Budapest, Leningrad, Prague, Warsaw, Belgrade, Bucharest, Sofia and Zagreb during the same period. Periodic tests for RFR at those control sites showed only background levels.

No convincing evidence was discovered that would directly implicate the exposure to microwave radiation experienced by the employees at the Moscow Embassy in the causation of any adverse health effects as of the time of this analysis."

COMMENT: More than 50% of the Americans studied when the Embassy was irradiated with low-level RFR (certainly not at the 88D peak pulse levels) lived and worked there for a short period of time. WSR-88D residents will have NEXRAD radar operated 24 hours per day for the next 20 - 25 years. Even to hint that this study, if valid, would apply to the weather radar tower, is invalid. 1-1

David B. Lehrman, M.D. and Chief of Radiation Therapy and Radiation Oncology, New Rochelle Hospital, writing for the NYS ASSEMBLY hearing of May 10, 1990 in West Sayville said: "It may take a period of five years for a leukemia to develop and a period of twenty to thirty years for solid tumors to develop after exposure to various forms of radiation." The Lilienfeld study was completed 1 year after the radiating stopped (1978).

Incidentally, our whole issue is not cancer problems. I'll quote Dr. Lehrman again: "Other health hazards such as the hastening of athero-sclerosis, cataract formation and infertility problems among others have yet to be fully researched."

A further comment by Dr. Joel Ray in an article titled "Invisible Energies (The Bookpress, Vol 2, #1) said the Moscow evaluations, done in secrecy and embroiled in controversy, showed blood samples with significant numbers of chromosone abnormalities.

CONCLUSION: The Lilienfeld study used by SRI in developing this SEA to prove there was no problem, does not bear transfer to the Weather Service's radar for the reasons mentioned. It is not a valid argument in the SEA's "Finding of No Significant Impact (hereafter referred to as FONSI).

THE MILITARY STORY

STATEMENT IN SEA B-16:

On p. B-16, the SEA uses a study of military personnel exposed to RFR in its argumentation that radar in 88-D's will pose no health problems.

"Robinette and Silverman (1977) chose 19,965 men who had served in the Navy during the Korean War whose job titles of Electronics Technician, Fire Control Technician, or Aircraft Electronics Technician appeared to classify them as electronic-equipment repair technicians...For the control group, the authors selected 20,726 Naval men...considered...to presumably have had little occupational exposure to RFR...no statistical association was found between health effects and presumed RFR exposure."

COMMENT: Dr. Stanislaw Szmigielski, who in 1985 was at the Center of Radiobiology and Radioprotection in Warsaw, Poland, worked with a team of researchers in studying radar's impact on servicemen. Dr. Szmigielski and his collaborators spent two years analyzing all cancer cases reported from 71 - 80 in the total population of career servicemen in the Polish military. They also ascertained the length of time each patient was exposed to RF/MW radiation and the location of each type of tumor. As of 1985, this was, by far, the largest epidemiological study of the effects of non-ionizing radiation ever completed.

The results: a tripling of the incidence of cancer among military personnel exposed to radiofrequency and microwave (RF/MW) radiation as compared to unexposed servicemen. "The observed cancer risk was greatest for the blood-forming organs, the lymphatic tissues and thyroid gland." According to the results, the odds of developing cancer increase with length of exposure and are greatest for younger age groups.

1-2

Overall, servicemen who worked with RF/MW radiation were 3.1 times more likely to get cancer than those in other military occupations. The research team also noted a "high correlation" between the chance of developing cancer and the number of years of exposure.

Statistical analysis revealed that the relative incidence of cancer was highest for the youngest age group. The 20 - 29 year-olds who were exposed to RF/MW radiation had a 550 percent greater chance of getting cancer than those in the same age who were not exposed. Cancer risks for the exposed group decreased with age but were still statistically significant for all age groups, especially for those under 50. Source: MICROWAVE NEWS, Vol. V No. 2, March 1985.

CONCLUSION: Understandably, SRI did not even mention this study in the bibliography because it would not logically lead to a FONSI. The Szmigielski work has both national and international credibility - and our citizen's group stands with its conclusions.

(Incidentally, Dr. Szmigielski initiated a 5 year prospective study - the prior one was retrospective - on the same topic around 1985. In an interim report after a year and a half, there was support for the earlier finding indicating higher morbidity from all malignancies with relation to duration and intensity of RF/MW exposure.)

THE ANSI GUIDELINES

STATEMENT IN SEA P. 34 *"Taken collectively, the epidemiological studies reviewed yielded no reliable evidence that chronic exposure to RFR at levels within U. S. exposure guidelines are hazardous."*

COMMENT: The American National Standards Institute (ANSI) standards issued in 1984, like their 1966 predecessors, were based on the faulty premise that thermal effects alone can cause adverse health conditions. If it doesn't heat, it doesn't hurt! There is a growing body of knowledge affirming that there are deleterious health effects from non-thermal exposure to non-ionizing radiation. Since ANSI standards do not even take into consideration this possibility, they are not an appropriate criterion for assuring no risk to health. Most probably, no one presently knows what adequate standards are or what a safe level of exposure is.

Dr. Arthur Guy's study of 1985, a $5 million project in which rats were exposed to microwaves for periods of over 25 months or so, actually "disproved" the theory that only thermal effects were possible. (Dr. Robert O. Becker, CROSS CURRENTS, 1991, p. 197). Of course that was scarcely the intent of the research - but it turned out to be a by-product. Still our point remains, that ANSI standards prove nothing about safety because they are built on a faulty premise.

The Congressional Office of Technology Assessment, in June of 1989, issued a 103 page document. The authors were Drs. Nair, Morgan and Florig of Carnegie Mellon in Pittsburg. They stated that setting a safe limit at this time cannot be scientifically supported and might potentially do "more harm than good." (Quoted in MICROWAVE NEWS, Vol.. IX, #4). So when
1-3 the SEA asserts no evidence of hazard at levels within U.S standards, we're totally unimpressed.

In a letter to our Batavia NY group on March 27, 1990, Dr. Stephen Cleary from the Medical College of Virginia said: "Epidemiological studies have revealed an unexpected relationship of exposure to electromagnetic radiation at microwave, and lower frequencies and cancer incidence. To date, questions regarding the types of exposures that may affect cancer incidence remain unanswered. In other words, it is not possible to state a safe level of exposure in the absence of adequate information."

And further on in his letter he tells of..."the evidence indicating that microwave radiation affects living systems via mechanisms other than heating. The potential significance of this is to draw into question the adequacy of safety guidelines such as ANSI C95.1 that are based upon the thermal effects premise."

Dr. Leo Birenbaum, in a letter for the N.Y. State Assembly hearing in West Sayville, New York, stated that "No one is yet able to say just what a 'safe' exposure level is. The ANSI standard is a consensus number arrived at by agreement among many people, rather than a threshold number, clearly defined by medical evidence, below which safety lies." (Dr. Birenbaum is a Research Associate Professor, Electrical Engineering and Electrophysics, New York Polytechnic University.)

CONCLUSION: We reject the false assurance offered by the ANSI standards because they rely on a faulty premise and unverifiable numbers.

4

THE SELECTIVITY STORY

STATEMENT IN SEA p. 34:

"Taken collectively, the epidemiological STUDIES REVIEWED yielded no reliable evidence that chronic exposure to RFR at levels within U.S. exposure guidelines are hazardous."

COMMENT: The emphasis here is on the words STUDIES REVIEWED. From our reading, we certainly had expected that the following authors and studies would have been included in any objective study of the issue.

1. Dr. Stanislaw Szmigielski, previously quoted, had by far the largest epidemiological study of the effects of non-ionizing radiation ever completed as of 1985. The exposed group was 3.1 times more likely to get cancer than those in other military occupations. (Quoted in MICROWAVE NEWS, Vol. V #2)

2. Dr. Szmigielski's prospective study, in its preliminary report in 1987, affirmed the retrospective study's results. Couldn't there been some updated report on these data in the 1992 SEA?

3. Dr. A. W. Guy's 1983-1985 nine volume work on the effects of long-term low level radio frequency radiation exposure on rats was analyzed by the Environmental Protection Agency in 1990. In part, the EPA report said this "long term animal study provides evidence that pulses (EMF's) in the absence of an appreciable tissue heating effect, induce carcinomas generally across all tissues of the body without being localized to any site..... The study can be said to demonstrate that pulsed RF fields are carcinogenic." (Quoted in MICROWAVE NEWS, Vol X, #4)

1-4

Of Guy's nine volumes, Volume 2 alone was cited in the bibliography of this new SEA, although I did not find any reference to it in the text. (It may be there; I did not find it.) Certainly, the conclusion that the EPA evaluation gave to Dr. Guy's research could not possibly have led to the FONSI nor to the sweeping statement quoted on p. 34 of SEA.

4. Dr. David Thomas, with Paul Demers from the Fred Hutchinson Cancer Research Center in Seattle, WA, uncovered evidence of an association between occupational exposure to EMF's and the development of male breast cancer. As quoted in MICROWAVE NEWS, Vol. X #4, the study "supports the preliminary findings of a Johns Hopkins University study, reported in late 1989, showing an increased risk of male breasts cancer among New York telephone workers." Obviously, no allusion to this type of study was included in the pages of the 1992 SEA.

5. The 1989 book, ELECTROMAGNETIC BIOINTERACTION by Franceschetti, Gandhi and Grandolfo, Plenum Press, New York and London, does not merit a citation in the SEA. Since the SEA document resulted in a FONSI, the contributors certainly did not wish to quote from Dr. Stephen Cleary's chapter in that book, but I will. "Review of the bioeffects literature, especially the results of <u>in vitro</u> cellular studies provide convincing evidence that RF radiation and other types of electric or magnetic fields, can alter living systems via direct non-thermal mechanisms, as well as by heating."

Cleary's chapter mentions eleven (11) direct alterations of living systems

via non-thermal mechanisms. He concludes: "Consequently the current basis for setting Western safety standards, namely of restricting exposure to minimize thermally-induced alterations, must be re-examined in light of the emerging evidence of non-thermal RF bioeffects...Current standards must be viewed as interim health protection expedients." I can understand why SRI didn't want to quote him in the SEA. It wouldn't be congruent with their conclusion.

6. Our Batavia citizen's group had a bibliography prepared for us by Dr. Joel Ray. In this document, Ray has listed 59 studies which cover the frequency range of NEXRAD. They basically affirm our stance - that there are potential health risks from non-thermal radiation, the type that would result from the radar tower's emissions.

Only 7 of the 59 studies made their way into the SEA bibliography. Since the other 52 offer an argument for our side, they naturally were not what SRI would wish to use in deciding, "no significant impact."

As noted, these 52 ignored works are at or near the WSR-88D frequency. But even if they weren't in or near that frequency, we have reached the conclusion that the whole EM spectrum is capable of bioeffects. The results may well be the same, no matter where the exposure is on the non-ionizing continuum - just as the results might be the same whether one is hit by a Colt 45 or an M16.

7. Now I will cluster these last few studies which we would have hoped to have been included in SRI's research.

1-4 (Cont.)

7.1. Drs. Jack McLean and Maria Stuchly (Health and Welfare Dept.,Canada). Finding: ELF emissions, in concert with toxic chemicals, promote the development of cancers in live animals. (Microwave News, July/Aug '91)

7.2 Drs.Balcer-Kubiczek and Harrison (Univ. of Maryland School of Medicine). Finding: Chemicals and microwaves can initiate cancer in normal cells. (Microwave News, July/Aug '91)

7.3. Drs. Wendy Mack, Susan Preston-Martin and John Peters. Finding: Men working more than 10 years in jobs with low frequency EMF exposure had a ten-fold increased risk of brain tumors. (Microwave News, Sept/Oct '91)

7.4. Dr. Sam Koslov from Johns Hopkins Applied Physics Laboratory in 1986. Finding: Alzheimer-like symptoms in primates exposed to microwaves. This was connected with a blood-brain barrier leakage.

7.5. Drs. Salford and Person (Lund University Hospital in Sweden). Finding: Evidence that microwave radiation can increase permeability of the blood-brain barrier. (Microwave News, Jan/Feb '92)

7.6. Dr. Jocelyne Leal (Spain), reporting at the first European Conference of the Biomagnetic Society. Finding: Weak magnetic fields had a profound effect on developing chick embryos. (Microwave News, Jan/Feb '92)

CONCLUSION: The selectivity of the studies contained in the SEA has prejudiced its outcome. The handful of studies cited above are illustrative of the proof of our thesis, that there is a definite possibility of health risk from this exposure.

THE OCULAR STORY

Statement in SEA: pp. 35 and 47

"Taken collectively, neither animal studies nor epidemiologic studies on eye damage from RFR yield scientific evidence that prolonged exposure to low-level RFR is likely to cause eye damage to humans...several epidemilolgic studies were performed to determine whether ocular damage could be statistically associated with occupational exposure to RFR.

Most of the positive findings in humans were found to be explained more by aging than by RFR exposure. Some exceptions were cases of possible occupational exposure of individuals at levels and durations likely to have been sufficient to heat the eye to temperatures well in excess of those found damaging in animals experiments."

COMMENT: Speaking of eye problems from RFR, Dr. Milton Zaret says, "Terms like radar, microwave or broadcast radiation, although frequently used synonymously, are not necessarily interchangeable...Although a few effects (ocular eye damage) depend only upon specific frequencies, most other harmful effects like radiant energy cataracts are not frequency specific but instead can result from exposure to any portion of the spectrum."

Because this radiation from the WSR-88D would be over homes for 20 or 25 years, Dr. Zaret's following statement is most fitting. "...little of the ambient radiofrequency radiation enters the body at any single exposure, and whatever harm might be done would be microscopic and therefore go unnoticed in the beginning. However, if the exposure is repeated often over a prolonged period of times, it would eventually result in delayed
1-5 appearing pathological changes.

By discovering this, I was able to establish the scientific basis for 'microwave' and similar 'radiant energy' cataracts becoming compensable diseases and to develop techniques for preventing injury." (Law Enforcement News, John Jay College of Criminal Justice, City University of New York, Feb. 28, 1991). I hope Dr. Zaret has time to respond to the SEA.

No threat of ocular harm? Won't SRI even temper its statement on the top of this page? Would SRI review the first (1948) report that microwaves could produce cataracts (A.W. Richardson et al.)

Would SRI review the 1973 report by E. Auell and B. Tengroth which said that microwaves can produce cataracts at nonthermal levels and can damage the retina itself.

And what of the report of R. Birge in the Journal of the American Chemical Society (1987) concluding that certain chemicals in the retina absorb microwaves to a high degree?

These last three studies are in the bibliography, Chapter eight, of Dr. Robert O. Becker's work, "Cross Currents," 1990.

CONCLUSION: My reading, that of a layman, gives me concern, especially for the little children in our area. To say there'll be no significant impact re vision from their proximity to this radiation is a pretty strong statement. I hope that within the next 30 days some ophthalmologists who have more vigorous and provable convictions than I am presently able to discover, will convey them to the National Weather Service.

7

I have a few authentic questions. I'm not trying a Lt.Columbo approach. I don't know the answers in advance of posing these questions.

1-6 1. Did I miss any printed statement about weather factors causing reflection of the radar's pencil beam, e.g. heavy cloud cover, storms, etc? What are the implications for the power density in the near field in case of these weather factors being present?

1-7 2. What are the implications on the pencil beam if the radar is in a 5 minute searchlight mode when there is a heavy cloud cover?

3. What are the implications of possible radiation leaks in the near field?

1-8 4. If children from the Sonshine Patch Day Care Center were playing on their Church property, about 600 feet from the radar, would you try to avoid being in the searchlight mode if the beam were over them? If there were a heavy cloud cover while in the searchlight mode, would you still keep it away from the play area? I will publish your answer.

5. If the answer to # 4 is negative, I ask you why? I will publish your answer.

1-9 6. Why, except for your own convenience, do you so often use averaged power densities (and don't say because IEEE's Standards Coordinating Committee does)? You don't <u>emit</u> the radiation in averages, why <u>measure</u> it in 6 or 30 minute periods?...Averaging bothers me...I know that if I give my thumb a good hit with a hammer, the pain is very strong. If I average the minute of extreme pain over the 1440 minutes in the day, it would be much more bearable. But banged thumbs and pulsed radar don't work that way. Averaging as used here is a convenient concept, not reality.

1-10 7. How would SRI respond to Dr. Leo Birenbaum, cited earlier, who asked, "Is it really safe to expose a person repeatedly many hundreds of times per second, over a long period of time, to those (NEXRAD) power densities?" Would SRI unequivocally say it's safe? I'll publish the answer.

8. Birenbaum asked, "do our bodies respond to peaks or to averages?" What answer does SRI give? (Birenbaum:"No one knows for sure.")

My next 4 questions are rhetorical, the previous 8 weren't.

1.1. If the EMF offers no health problem, why is there pending Federal legislation (HR 1483, Pallone) to have a five year study of the issue?

2.1 If there is no significant impact from EMF's, why is there a two day seminar at The Univ. of Michigan Law School next month to discuss the most recent developments in EMF research and litigation? (If you wish to take shots at attorneys, don't do it within hearing of the Judge.)

3.1. If radar can be used safely, why did two Connecticut Police departments, the Connecticut State police as well as least two departments in Florida ban hand held radar units last year?

4.1. Why has the Electric Power Research Institute been given $350 million by power companies to study the issue?
'Nuf said. Thank you

8

Letter #2

NEW HOPE FELLOWSHIP

Jeremiah 29:11

April 15, 1992

Robert L. Smith, Pastor

Gentlemen,

My name is Robert L. Smith and I am Pastor of New Hope Fellowship Church located at 8052 Bank St. Rd. in Batavia, New York. We are an extremely busy, growing Church involved in many ministries to reach out to the poor, needy and the hurting in our locality. One of the many ways our Church tries to meet the needs of this area is the operation of a child care center. When our center first opened, the Batavia area virtually had no child care centers available. With the desire to be a blessing to our community, we went to great expense to bring our building to meet state and local codes. Not long after opening, we began to hear of plans to build a NEXRAD weather facility, including radar tower within just a few hundred feet of our Church. After taking a lot of time before choosing sides, we came upon quite a bit of information, some assuring us of the safety of the radar tower and some (actually a lot) warning us of the **POSSIBILITIES** of harmful effects of electromagnetic radiation. I watched in disbelief as it seemed endless numbers of elected officials at all levels were willing to take the risk with the children who attend our day care or who were unwilling to become involved.

Not just our child care ministry, but as I said, we are a busy Church and there is almost always something going on. Therefore, the concern for all our people and their children and any ministries we may hope to move into in the future (i.e. pregnant teenagers, single mothers, etc..) is very real.

As some of us have had an opportunity to read the newest supplemental environmental assessment done by S.R.I. of California, we cannot help but feel that it's just more of the same. Extolling NEXRAD while pointing out that there is no conclusive evidence that there are harmful effects from electromagnetic radiation, instead of showing the proper concern for human life that there should be when there is doubt.

2-1 I stand by my conviction that for a study to have any credibility, it **MUST** be performed by a neutral, non-government agency.

I remain convinced that unless there is irrefutable documented evidence of the safety of NEXRAD and electromagnetic radiation both short and long term, it does not belong near a church, day care or residential neighborhoods.

Thank you for your time.

Yours in Christ,

Rev. Robert L. Smith

EXHIBIT 18 pm

McLaughlin

8052 Bank Street Road, Batavia, New York 14020 • 716/344-2997

Letter #3

Sonshine Patch Child Care

(Teaching Christian Principles)

Robert L. Smith, Pastor

April 1992

Gentlemen:

My name is Mary Beth Hoffarth and I am the Director of the Sonshine Patch Child Center. The day care which is less than a 1000 feet from the proposed NEXRAD weather station in Batavia, New York. We have 102 registered children in our day care center, and average 50-60 children in attendance per day. We have been operating for almost three years now, and have established a wonderful rapport with our parents and a fine reputation in the community.

3-1 It is my understanding that the NEPA have reported a negative declaration regarding the aforementioned weather station. I have read enough scientific literature to show that evidence exists to substantiate a re-evaluation of the situation and
3-2 we will not be satisfied until there is and independent study done. In a letter from Dr. Robert O. Becker, he states: ..
3-3 "the population is not homogenous in this respect, the fetus, the infant, and the young child are much susceptible to harmful effects." My concern is that this is precisely those who are entrusted to our care on a daily basis. I have talked with many parents, who are also becoming increasingly aware of the potential risks, and have voiced that they will seek other child care arrangements for their children if NEXRAD goes through plans to build the weather station on the land adjacent to us.

I wonder if the county legislature members have considered the implications that could arise if our day care is forced to close. We have 23 employees - ALL of whom reside in Genesee County and most reside in Batavia. Are they willing to sacrifice OUR jobs for the 22 jobs of Erie County residents that the weather station will maintain? We have made appeals to the county legislature, and to the Batavia town board and have ahd very little acknowledgement. I feel that the parents of the children at our day care have been ignored and they need us to be their voice and fight for their children.

I thank you for understanding my concern and my position of this very delicate matter. Any support you can give us will be greatly appreciated.

Sincerely,

Mary Beth Hoffarth

An Outreach of New Hope Fellowship, 8052 Bank St. Rd., Batavia, N Y 14020
716-344-2997

Arlene R. Bow
4423 North Bennett Heights
Batavia, NY 14020

April 16, 1992

To Whom It May Concern:

I am appealing to active listeners. I believe it is time for the U. S. Government to investigate all the facts regarding the Next Generation Radar System, NEXRAD, before jumping in with both feet - just to pay the financial and possible health consequences later.

The objective world of science shows a "buyer beware" attitude towards these pulsed radar systems. Is government too large that the hand purchasing this system is not the hand approving the program? Is this U. S. Citizen to believe its elected officials are not intereseted enough in its individual constituents? Is it okay to sacrfice a few to save the masses?

We are a great nation of entrepreneurs - of inventors - of creators; yet there have been many times when this nation has acted in haste to develop, with no foresight for the future. Radiation, in a different form, used 40 some odd years ago, shrunk thymus glands on newborns. Twenty years after its use, these people were developing cancer of the skin and/or thyroid. This group will need continual monitoring, and at whose expense? Yes, all of ours.

I'm a member of this radiated thymus gland infant group. I'm not opposed to new developments using radar. I do know from all I've read that its proximity to homes, schools, and day cares is critical. I am aware of studies focusing on cancer growths in people who are in close proximity to radar systems. Cancers may take <u>years</u> to develop. A study today needs follow-up in five-ten-twenty or more years. There are inidcations of safe distances today that THE GOVERNMENT, if you will, is not taking into account today.

5-1

I ask you to please read. Please study. Please act responsibly. It is my opinion that the only error that would be irreversible is the error of overlooking every safety factor.

Sincerely,

Arlene R Bow

(Mrs.) Arlene R. Bow, RN

Letter #6

NWSEO POLICY STATEMENT

ON

NEXRAD AND RADIO FREQUENCY (RF) RADIATION

NWSEO is not against new technology such as NEXRAD. NWSEO has testified before Congress on this point, most recently on March 29, 1990. We feel that this nation should have the most advanced weather forecasting and early warning services of any nation in the world. We feel strongly however, that new technology such as NEXRAD, should be tested and proven before it becomes operational resulting in closed weather offices and services reduced.

NWSEO is also concerned of possible health and safety problems that may be connected with NEXRAD, specifically, if electromagnetic fields resulting from NEXRAD would have an environmental impact on community residents, and NWS employees.

6-1 Scientists have debated the mechanism by which RF can cause changes in the body, and that RF may affect cells previously damaged by a carcinogen. The exact mechanism of damage is unknown, but there is some evidence to suggest that a prudent course of action is to avoid as much radiation as possible.

6-2 Very strong Radio Frequency (fields or radiation) generated by radars prompted the military to prudently limit refueling; blasting cap storeage and personnel exposure. These standards are indentical to the American National Standards Institute which assumes that there is no accumulative effect from repeated exposure in humans. The NEXRAD prototype mainbeam power tests indicate these standards are exceeded up to 400 feet from the antenna. A peak power density of less than 5W/cm2 can be considered safe.

NWSEO believes that while RF from NEXRAD may, in future studies prove harmful, current studies on the long range effects are inconclusive.

6-3 NWSEO will continue to monitor possible RF hazards that may be connected with NEXRAD. NWSEO will request that additional environmental impact and health studies be conducted on the long term effects of RF emissions. Until such studies are completed, NWSEO cannot conclude that NEXRAD does or does not create health AND safety hazards.

David T. Powell Jr.,
National President

To Whom It May Concern:

I am a member of CONRAD III, citizens opposed to NEXRAD in Batavia, New York and I have great concerns about placing a NEXRAD radar tower within a mile of residential homes and 1000 feet from a day care center.

CONRAD III is not opposed to having a NEXRAD tower, but it is opposed to the proximity to homes and day care centers. Since it has not proven to be safe, the government is taking chances with long term health of our population. Not all scientists are in agreement with Dr. Friday, the director of National Weather Service, in stating that there is not a health risk. Dr. Leo Berenbaum and Dr. Robert Becker both made statements that in not knowing long term health risks and with NEXRAD a pulsed radar system of 100 times per second at 500 times the average value of power density that it is inappropriate to place the NEXRAD tower in a residential area.

7-1

We need a new weather system and it seems as though NEXRAD is the weather system of the future. However if you do place value on human life, you will consider the future of the children's health in these areas and find another NEXRAD site.

Sincerely,

Susan Lukaszewicz

Susan Lukaszewicz
8057 Bennett Heights
Batavia, N.Y. 14020

TO WHOM IT MAY CONCERN:

The proposal by the U.S. Weather Service to build a high-teck Nexrad Weather Forecast station near the Genesee County Airport has triggered much controversy for the residents of Bennett Heights.

We are not opposed to Nexrad, but its location within one mile of residential homes and 600 feet from a church and a day care center.

9-1 The health risks of building a radar tower so close to residents must be taken into consideration. Since no recent studies have been done, and when there is doubt, caution should be exercised.

So is it right to place the radar tower within the residential area? By so doing, to take chances with the long term health of the population, I feel it is inappropriate to do so.

Sarah C. Lukaszewicz

SARAH C. LUKASZEWICZ
8057 Bennett Heights
Batavia, New York 14020

Letter #12

April 10, 1992

Dear Sir,

12-1 After following NEXRAD for over two years, I'm convinced more than ever, that it is potentially harmful and a complete mechanical and financial disaster. Testing has proved as much.

As a mother, I feel it is my duty to try to protect my children in all ways possible. Putting a stop to this farce you call WSR-88D (NEXRAD) will probably be my biggest battle, but I'm not willing to give up their right to a healthy and safe up-bringing.

We have enacted laws for use of safety belts in vehicles, helmets on bikes, etc. to protect our children and adults, and now you want to install WSR-88D. It's like holding a loaded gun to their heads.

Enough studies have been, and are being done to warrant restriction of electromagnetism near homes, schools etc.

It's about time to save the taxpayers money on foolish health risks and start over with something that is practical, safe, and works.

I have read the ~~XXX~~ 1984 PEIS done for NEXRAD 3 times and scanned the April 1992 SEA. I will follow this letter with another by the May deadline rebutting this issue.

Sincerely,

Mrs. Dianne M. Puccio

Mrs. Dianne M. Puccio

STATEMENT FOR HEARING ON THE SUPPLEMENTAL ENVIRONMENTAL ASSESSMENT

APRIL 21, 1992

PLEASE ANSWER THE FOLLOWING QUESTIONS CONCERNING THE SEA OF 1992:

1. Why were the following studies not even studied by SRI when preparing the SEA and why were their findings not considered when arriving at a conclusion?

DATE	RESEARCHER	EXPOSURE FREQUENCY/INTENSITY	RESULT
1973	Bychkov	100uw - 3 Ghz	effects on EEG
1974	Dumanskiy	1-10.5uw-2.5 Ghz	effects on EEG
1978	Grin	50uw-2.4 Ghz	brain enzyme reduction
1975	Roberti	-- --	lowered blood cholinesterase
1973	Tolgskaya	60-320uw-3 Ghz	atrophy of hypothalamus
1974	Dumanskiy	5-10uw- 3 Ghz	elevation of excreted corticoids
1973	Demokidova	--- - 3 Ghz	altered adrenal weight
1973	Demokidova	153uw - 3 Ghz	altered thyroid weight
1976	Baranski	5000uw	increased incorporation of iodine, increased number of cytosomes, enlarged Golgi apparatus
1973	Markov	1153uw - 3 Ghz	short-term hypertension, long-term hypotension
1974	Kartsovnykh	25-50uw- 3 Ghz 500uw - 3 Ghz	white blood cell increase white blood cell decrease
1978	Gonchav	10-50uw, 2.4 Ghz 500 uw - 2.4 Ghz	increased glycogen decreased glycogen
1974	Czerski	3000uw - 2.95 Ghz	decreased erythrocyte production, effect on iron metabolism
1975	Szmigielski	3000uw - 3 Ghz	immune system impairment
1979	Shandala	1-300uw - 2.4 Ghz	immune system impairment
1971	Dronov	50 uw -	immune system impairment

13-1

	1979	Shandala	500uw --	immune system impairment
	1975	Czerski	500uw - 2.95 Ghz	immune system impairment
	1977	Vinogradov	5-50uw - 2.4 Ghz	immune system impairment
	1974	Czerski	1000uw - 2.9 Ghz	altered cell mitosis in bone marrow
	1978	Dumanskiy	100-1000uw-2.4 Ghz	altered liver metabolism
	1976	Dumanskiy	10-1000uw - 2.4 Ghz	effects on liver mitochondria
	1976	Shandala	50 - 500uw - 2.4 Ghz	altered kidney function
	1974	Miro	2000uw - 3 Ghz	increased protein synthesis in liver, thymus and spleen
	1975	Minayev	570 uw - 2 Ghz	decrease in vitamin B6 levels in majoe organs
	1975	Gabovich	10,100,1000uw -2.4 Ghz	altered distribution of trace metals in major organs
13-1 (Cont.)	1975	Ilchevich	10-50uw - 2.4 Ghz	disturbed ovarian morphology and fertility, altered postembryonic development
	1973	Markov	153uw - 3 Ghz	decreased weight gain
	1984	Szmigielski	10,000uw - 2.45 Ghz	fetal abnormalities from synergy with teratogen
	1976	Kapustin	50-500uw - 2.4 Ghz	chromosome abnormalities
	1972	Baranski	3500uw - 3 Ghz	disorders in lymphocyte
	1970	Yao	100-10,000uw - 9.4 Ghz	(in vitro) chromosome aberrations, disrupted RNA synthesis, reduced protein production
	1969	Baranski	7000uw - 2.9 Ghz	(in vitro) chromosome abnormalities
	1983	Kues	--- 2.45 Ghz	cornea abnormalities

	1973	Sadchikova	20-30uw - -	headache, fatigue, irritability, cardio-vascular change; abnormal blood pressure and ECG .
	1974	Sadchikova	-- --	similar results
	1969	Fofanov	10-170uw - 30 Ghz	bradycardia, decrease in pump-ing ability of the heart
	1966	Monayenkova	-- --	similar results
	1966	Droogichiva	-- --	cardiovascular disorders
	1974	Klimkova	-- 3-30 Ghz	headache, fatigue, EEG changes
	1973	Sokolov	-- --	decreased leukocytes increase in red blood cells
	1975	Lancrangan	10-100uw - 3.6-10 Ghz	sperm abnormalities
13-1 (Cont.)	1968	Majewski	-- - .6-10.7 Ghz	lens opacities
	1970	Kheifets	-- - .3 -300 Ghz	lens opacities
	1973	Odland	-- military radar	eye defects
	1974	Tengorth	-- --	lens opacities and retinal lesions
	1974	Zydecki	100-1000uw --	lens opacities
	1969-80	Zaret	-- --	cataracts
	1981	Friedman	-- --	polycythemia (rare blood disorder)
	1988	Szmielgski	-- --	lymphatic cancer and leukemia
	1982	Hannson	-- - 1-10 Ghz	abnormal proteins in cerebrospinal fluid

COMPILED 2/92 BY DR. JOEL RAY, CO-AUTHOR, THE ELECTRIC WILDERNESS (SAN FRANCISCO PRESS)

N.B. ALL OF THE ABOVE STUDIES ARE AT OR NEAR THE FREQUENCY OF THE NEXRAD (WSR88D) WEATHER RADAR TOWER.

N.B. MOST OF THESE STUDIES INVOLVE EXPOSURES WELL BELOW THE ANSI 5

MILLIWATT STANDARD. THE READER SHOULD BE REMINDED THAT EASTERN EUROPEAN STANDARDS AS IN THE FORMER SOVIET UNION, AND PRESENTLY IN CHINA, ARE A HUNDRED TIMES AS STRICT AS THE AMERICAN STANDARDS (25 - 50 MICROWATTS/CM SQUARED). MANY OF THE STUDIES CITED ABOVE SERVED AS THE BASIS FOR THOSE STANDARDS.

13-1 (Cont.)

N.B. SINCE THE GOVERNMENT OF THE U.S. HAS ALLOCATED LITTLE MONEY FOR RESEARCH IN THIS FIELD, WE NEED TO RELY HEAVILY ON THE STUDIES OF THE EUROPEAN AND CHINESE SCIENTISTS.

13-2 2. Will the Supplemental Environmental Assessment of 1992 prepared by six men from SRI (under contract to the NATIONAL WEATHER SERVICE) be PEER-REVIEWED by independent scientists and researchers at government expense?

3. On page ii of the SEA, power densities were listed as:

MAXIMUM AVERAGE POWER	0.6 mWcm squared
1000 ft. from radar at antenna height	0.006 mWcm squared
1000 ft. from radar at ground level	0.0004 - 0.00002 mWcm squared

13-3

followed by the statement: These measurements have been verified by field measurements.

Will there be any independent, peer-reviewed measurements done to verify these figures?

4. Professor Leo Birenbaum, a Research Associate Professor from the POLYTECHNIC UNIVERSITY wrote to Mrs. Barbara Pettick for the West Sayville New York State Assembly hearing held on May 10, 1990. (His letter is attachment #1) In his letter, he calculates the power densities and his results are substantially different from the ones indicated in the SEA.

E. g.

4000 ft. from the antenna	0.22 mWcm squared
5000 ft. from the antenna	0.14 mWcm squared
5280 ft. from the antenna	0.13 mWcm squared

13-4

Why are your calculations so different from those of Dr. Leo Birenbaum?

5. In light of the above information, will the NATIONAL WEATHER SERVICE hire Dr. Leo Birenbaum to review SRI calculations and make comments about them?

6. On page i of the SEA, it is stated:

"There is no 'reliable scientific evidence to suggest that, for the cases considered exposure to RFR from the NEXRAD radar will be deleterious to the health of even the most susceptible members of the population such as the unborn, infirm, or aged."

13-5 Are you willing to examine the opposing view of Dr. Robert O. Becker of Syracuse, New York who says "those most at risk will be the fetus, infant and younger child..." ? (His letter is attachment #2)

7. On page i of the SEA the statement is made :
"WSR-88D will be able to make velocity as well as reflectivity measurements."

13-6 Does making "reflectivity measurements" mean that the radar beam will at times be reflected from the clouds to earth? If so, in the case of low, heavy cloud cover (which is so common in Western New York weather) will this change the calculations of the power densities given above and/or the power from either the main beam or the sidelobes?

8. On page i of the SEA; "The highest values which were measured near radio transmission towers were 1-7 mWcm squared."

What are the main differences between the typical radio transmission tower and WSR-88D?

13-7

Since we already have radio and T.V. transmitters in our city, how much will the addition of the power from WSR-88D add to the ambient power with which we will be surrounded?

Is there any way of measuring this ambient power?

9. When describing "searchlight mode" on p. ii, it is said: " Average power densities will be higher than during normal operation. Within 800 ft. of the antenna face, the power density averaged over 6 min. will be 3.85 mWcm squared. Averaged over 30 min., the power density will be 0.77 mWcm squared."

What are the power densities BEFORE AVERAGING?

How were the averages calculated?

13-8 The Sonshine Patch Day Care Center is within 700 - 800 ft. from the proposed radar tower.

What will be the impact on the children who will be very close to the "near field" during normal operations?

How will they be impacted during the "searchlight mode"?

13-8 (Cont.) Give us the power calculations for the "searchlight mode" and the normal operational power densities UNAVERAGED please.

10. On page ii, it is stated: "SCC 1991 standards 28 guidelines for exposure in uncontrolled environments (e.g. for general population) to WSR-88D frequency RFR are 1.8 - 2.0 mWcm squared averaged over 30 minute periods."

What are the measurements BEFORE AVERAGING?

13-9 Please list the women and men who were on the committee which developed these new SCC standards.

Were these standards based on the same premise that generated the ANSI standards, namely that the only harmful effects are thermal effects?

Will the NATIONAL WEATHER SERVICE hire Dr. Robert O. Becker to review and comment on the standards developed by the SCC committee?

11. On page iii of the SEA it states: "...however, the power densities of WSR-88D RFR will be too low to cause such heating and its associated effects."

Does this mean that the committee from SRI which prepared the SEA, rejects any data on the health effects from the studies on non-ionizing radiation?

13-10 Are they still accepting that there are thermal and/or ionizing effects only?

Will NATIONAL WEATHER SERVICE hire Dr. Milton Zaret to comment on this theory for us?

12. On page iii of the SEA: "However, more recent studies with improved instrumentation and/or better controlled experimental conditions reveal that those earlier results PROBABLY were obtained erroneously."

How scientific is the use of the word PROBABLY in an issue of this magnitude?

We note the frequent and continual use of the words: PROBABLY, IN SOME CASES, MAYBE throughout this document. This gives the SEA a sophomoric tone and decreases its credibility as a scientific document to be respected.

13-11 Will the NATIONAL WEATHER SERVICE hire an independent, outside expert of our choosing to comment on the statement given in #12 above?

Were there ANY studies which showed some deleterious effects which were not "OBTAINED ERRONEOUSLY."?

In order for us to comment on this sweeping statement, we need specificity.

List the studies which were in error due to faulty instrumentation.

13-11 (Cont.)

List the studies which were in error due to problems with the experimental conditions.

13. Page 33 says that papers were "selected" for study.

Who selected the papers to be studied and on what criteria was the selection based?

The selectivity of this SEA committee displays a bias against any studies which would have given a fair hearing to the "other side", the side which urges caution. A similar biased document could have been prepared by the scientists who see danger to the use of radar near people. This highlights our conviction that THE VERY EVIDENT CONTROVERSY LEAVES CONFUSION IN THE MINDS OF LAY PEOPLE SUCH AS OURSELVES. WE BELIEVE THAT IT IS REASONABLE TO TAKE EITHER SIDE IN A SCIENTIFIC ARGUMENT SUCH AS THIS. WE OPT FOR THE SIDE OF SAFETY. WHEN THE SCIENTIFIC COMMUNITY MAKES UP ITS MIND DEFINITIVELY FOR EITHER THE SAFETY OR THE DANGER OF RADAR, WE WILL ACCEPT ITS DECISION. UNTIL THEN, WE STICK TO OUR FIRM CONVICTION. WE WILL NOT BE A CASE STUDY!!

13-12

14. On page 34 of the SEA: " Taken collectively, the epidemiologic studies reviewed yielded no reliable evidence that chronic exposure to RFR at levels within the U. S. exposure guidelines is hazardous."

"Several of the epidemiologic studies reviewed yielded no evidence of detrimental effects associated with exposure to RFR."

We do not doubt that "several studies" yielded no evidence of detrimental effects; however, were there also several which did?

13-13

If so, what ratio of those which did to those which didn't is needed before a reasonable person would say there is need for prudent avoidance?

Are we looking for all black or all white in an issue which at this time is yielding nothing but gray?

15. On page 34 of SEA: " Several other studies were flawed for various reasons such as by use of population samples too small, use of mail self-administered questionnaires to acquire data, inappropriate statistical treatment of the data, incorrect assembly of data bases."

By whose criteria were these studies termed "flawed"?

Were there also similarly "flawed" studies which yielded negative results?

13-14

How can one draw any definitive conclusion when we are pitting negative flawed studies against positive flawed studies?

16. On page 35 of the SEA: " Taken collectively, neither animal studies nor epidemiologic studies on eye damage from RFR yield scientific evidence that prolonged exposure to low level RFR is likely to cause eye damage to humans."

Will the NATIONAL WEATHER SERVICE hire Dr. Milton Zaret review this statement and supporting data and render an opinion on the its accuracy?

17. On page 35 of the SEA: " The findings of Cleary et al (1965) were 2644 veterans classified as radar workers produced 19 cataracts....." and " Cleary and Pasternack (1966) findings unclear because physiological aging of lenses had occurred in both groups."

Will the NATIONAL WEATHER SERVICE hire Dr. Stephen Cleary tc review this statement and supporting data and render an opinion on its accuracy?

13-15 We have a letter from Dr. Cleary which leads us to think that he believes there are potential health effects from non-ionizing radiation from radar. (Attachment #3)

18. On page 35 of SEA: " Relatively few studies have been conducted to determine whether RFR per se (e.g. 3 Ghz) induces or promotes cancer. On the other hand, a few controversial epidemiologic studies have reported a statistical association of cancer promotion with exposure to power line fields (60 hz)."

By your own admission, there have been FEW STUDIES conducted to determine whether RFR induces or promotes cancer. This is our very point- if there have been few tests, how can one be confident of the conclusions drawn based on this lack of evidence either way?

13-16 That would be like saying that none of my theory students failed the test; but I did not give them a test!

The very lack of studies convinces us that our stand for safety until either side can be proven more conclusively is a reasonable one. At this time, the picture is gray not BLACK OR WHITE.

19. On page 35 SEA: " Collectively scientific literature does not provide scientifically credible evidence that exposure of either mammalian or nonmammilian subjects to low levels of RFR produces mutations or cytogenetic effects or that such RFR induces or promotes any form of cancer in mammals or culture of mammalian cells. Thus there is no experimental evidence from those studies that exposure to RFR from WSR-88D will cause such effects in the general population."

13-17 Strange that you are able to reach that conclusion so strongly after admitting that there have been FEW STUDIES. If there have been few studies and therefore a reasonable person cannot conclude that there is danger in promoting mutations or cancer, how can one conclude that there is no danger from the same lack of studies?

In closing, may I quote from Dr. Robert O. Becker's book CROSS CURRENTS. He says what we believe.

"I have yet to see a scientific question over which there was no

difference of opinion among reputable scientists. Such differences may be honest, motivated by some uncertainty about the data or by a valid but different outlook. When this occurs, the actual decision-making authorities have a difficult time. ....."

" Unfortunately, expressed differences of scientific opinion are not always so noble. Scientists are, after all, human beings, and some can be tempted to shade their opinions for certain considerations. It is quite possible to slant a presentation by ignoring positive data indicating risk, or to require of such data a much higher standard of scientific rigor than for negative data. The usual recommendation from this type of expert consultant is that some risk may be present, but that it requires more study in order to be clarified and, in any event, is not significant enough to prevent an installation from being constructed. The citizen groups in opposition to the planned construction generally LACK THE RESOURCES TO HIRE SCIENTIFIC EXPERTS WHO SHARE THEIR VIEWS."

" The answer to a valid risk/benefit determination appears to me to be quite clear. All of the data needed for making a decision should be made available to everyone concerned. This seems simple enough, but in our technological world it often means the expenditure of considerable amounts of money. If the data are inadequate or incomplete, a major benefit over any possible risk should be demonstrated before a conclusion is made that the benefit justifies the risk. If the data are adequate, the decision should be based upon the open and unbiased evaluation of these data--something that is more easily said than done. Of equal importance to adequate data is the principle that THE ONLY PERSONS WHO CAN MAKE THE FINAL DECISION AS TO RISK VERSUS BENEFITS ARE THOSE PERSONS WHO WOULD BE AT RISK."

DUE TO THE LACK OF TIME TO REVIEW THE ONE COPY OF THE SEA WHICH OUR GROUP RECEIVED, THESE ARE THE ONLY QUESTIONS WHICH I HAVE TO PRESENT AT THIS TIME. I WILL;HOWEVER, HAVE MORE TIME TO READ THE ENTIRE DOCUMENT AND DESIGN MORE QUESTIONS FOR YOUR CONSIDERATION BEFORE MAY 26,1992.

LAST YEAR, WE DID A SIMILAR, THOROUGH STUDY OF THE SITE SURVEY AND SUBMITTED 30 PAGES OF COMMENTS TO NWS AND SRI. THAT WAS 15 MONTHS AGO AND WE HAVE YET TO HEAR ANYTHING IN RESPONSE TO OUR COMMENTS. WE RECEIVED NO CORRESPONDENCE FROM THE NATIONAL WEATHER SERVICE (OTHER THAN DOCUMENTS RECEIVED UNDER FREEDOM OF INFORMATION ACT) SINCE WE SUBMITTED THE COMMENTS FOR THEIR REVIEW. WE HAD 30 DAYS TO PREPARE THEM; THEY WITH ALL OF THEIR MONEY AND EXPERTS. HAVE NOT YET, AFTER 15 MONTHS, WRITTEN US A SINGLE WORD ABOUT THESE. I HOPE THAT THESE REMARKS AND QUESTIONS WILL BE GIVEN BETTER TREATMENT THAN OUR PREVIOUS ONES.

IF NEPA PROVIDES A COMMENT PERIOD FOR QUESTIONS AND COMMENTS, I PRESUME

THAT THE SPIRIT OF THE LAW WOULD INDICATE THAT THESE BE TAKEN SERIOUSLY. I HOPE THAT IS SO.

Respectfully submitted:

Gloria M. Laughlin

4421 N. Bennett Hyte.

Batavia, N.Y. 14020

Attachment #1

333 Jay Street, Brooklyn, New York 11201 718-260-3600/FAX 7182603136

MR. N. MCLAUGHLIN
4421 N. BENNETT HEIGHTS
BATAVIA, NY 14020

May 4, 1990

Polytechnic UNIVERSITY

Ms. Barbara Pettick
CONRAD
20 Milton Street
Sayville, NY 11782

Dear Ms. Pettick:

This letter concerns the planned installation of a NEXRAD weather surveillance doppler radar unit on an FAA site in Sayville, Long Island. I understand that this is one of a network of 165 units to be deployed in the continental U.S. by the National Weather Service.

From the material that you sent me, I made a list of some of the characteristics of the NEXRAD radar:

Frequency	2710 MHz		wavelengt. 11.06 cm
Beam	conical	1° wide	elevated 0.5° - 20° above horizon
Modes	short pulse	1.57 μs	repetition rate 318 - 1304 Hz typical operation 322, ... 1282 Hz
	long pulse	4.5 μs	repetition rate 318 - 452 typical operation 332, 446 Hz
Peak Power	750 kW		
Average Power	short pulse	1510 W at rep. rate of 1282 Hz (duty cycle of .00201)	
	long pulse	1510 W at rep. rate of 446 Hz (duty cycle of .00201)	
Antenna	28-foot diameter parabolic dish, continuously rotating		
Tower	82 feet high; 39-foot diameter radome on top of tower houses radar		

The proximity of the proposed radar site to a residential area makes it appropriate to consider whether the radar could affect any of the 2600 neighboring homes, 7 schools, several nursing homes and residential communities located within a one mile radius of the site. Accordingly, an estimate of the average power density within the beam is made below (expressed in milliwatts per square centimeter, mW/cm^2) as a function of the distance from the radar location.

Average power density within the beam "near" the antenna: "Near" the antenna; that is, for distances closer than

$$\frac{2d^2}{\lambda}$$

(d is the dish diameter and λ is the wavelength); or about 4000 feet, the average power density is approximately

$$p = \frac{\text{average radiated power}}{\text{dish area}} = \frac{1500 \times 1000 \text{ mW}}{\pi (14 \times 12 \times 2.54)^2} = 2.64 \text{ mW/cm}^2,$$

although it may be higher or lower at some locations.

Gain of the antenna: The gain of the parabolic dish antenna above that of an isotropic antenna is

$$G = \frac{27{,}000}{\theta}$$

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FAX 7182603136

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516-755-4400
FAX 5167554404

Westchester:
36 Saw Mill River Road
Hawthorne, NY 10532
914-347-6940
FAX 9143476939

where θ is the beam width expressed in degrees*. (An isotropic antenna is one that radiates in all directions with equal intensity, as would a light bulb.) For a 1° beam, the gain is G = 27,000 (44.3dB).

Average power density within the beam "far" from the antenna:

$$p\left(\frac{mW}{cm^2}\right) = G \cdot \frac{\text{average radiated power in mW}}{\text{Area of spherical surface a radial distance R from the antenna}}$$

$$= G \cdot \frac{1510 \times 1000\ mW}{4\pi\left[R_{ft} \times 12 \times 2.54\right]^2\ cm^2} = G \cdot \frac{129.3}{R_{ft}^2}\ \frac{mW}{cm^2}$$

$$p = \frac{3.49 \times 10^6}{R_{ft}^2}\ \frac{mW}{cm^2}$$

R_{ft} is the radial distance from the radar tower expressed in feet. Thus, the average power density in the beam "far" from the antenna is given in the table below:

R(ft)	p (mW/cm^2)
4000	0.22
5000	0.14
5280	0.13

Suppose that the beam is stationary (rather than rotating) and is misdirected downward (rather than 0.5° – 20° above the horizon). Although this situation is not expected to occur, it may. In that case, if a person were directly in the beam, then these would be the approximate power densities. (See also attached graph.) For comparison, the 1982 ANSI (American Natural Standards Institute) C95.1 exposure guide at 2.71 GHz is 5 mW/cm^2.

No one is yet able to say just what a "safe" exposure level is. The ANSI standard is a consensus number arrived at by agreement among many people, rather than a threshold number, clearly defined by medical evidence, below which safety lies. For example, during the pulse on-time, the power density of the weather radar in the example cited here is $\frac{1}{.00201} \approx 500$ times the average value. This raises the question: Is it really safe to expose a person repeatedly, many hundreds of time per second, over a long period of time, to these power densities? Under these circumstances, do our bodies respond to peaks or to averages? No one knows for sure.

Of course, it is desirable to have better weather forecasting. But is it all right to place the radar set within a residential area, and by so doing, to take chances with the long-term health of the population? I feel it is inappropriate to do so.

Sincerely,

Les Braunstein

Research Associate Professor,
Electrical Engineering and Electrophysics
(718) 260-3319

LB/vcv

* Reference Data for Radio Engineers, Howard Sams, 1968, page 29-2.

Attachment #2
Dr. Robert Becker

ROBERT O. BECKER, M.D.
CONSULTANT IN BIO MEDICAL SCIENCES

BOX 278 STAR ROUTE
LOWVILLE NY 13367
315-376-8072

3 April 1990

Mr. Norman McLaughlin
Batavia, NY 14020

Dear Mr. McLaughlin,

Your letter arrived today and I will try to get this to you in time for your meeting but doubt that I will succeed. The fact is that no one knows with any level of authority what the safe level for chronic exposure to microwave radiation is. The standard that Dr. Elder suggests is based upon the now discredited concept that only heating effects are possible and that field levels below those capable of causing heating are entirely without bioeffects.

In the absence of any definitive information, and to avoid appearing over alarmist, I made the statement re. 0.1 mW/cm2. This is far below the government "standard" that is presently extant. If you would read on however, you would note that I state that it is the total level of ambient radiation, from all sources that counts, not the level from any one source. Dr. Elder frequently refers to household appliances as being more dangerous because of their much higher fields. This is incorrect; first of all, you have the choice of using these devices and secondly exposure times are short (except for electric blankets). Ambient radiation, however, is imposed upon you, you have no choice, and it is present all day, every day. All evidences we have for hazards now clearly show that exposure time plays a very large role, with residential and occupational exposures to very small field strengths being productive of major bioeffects. In addition, the population is not homogenous in this respect, the fetus, infant and younger child are much more susceptible to harmful effects. All of these statements are not fabricated out of thin air but are based upon laboratory and epidemiological studies published in the peer revieved scientfic literature.

It is very common to calculate the field strength of such devices and then point to the number of zero's calculated with the suggestion that anyone who would think that harmful is not only incorrect but mentally unsound as well. Again, such calculations are not valid indications of the real field that would result, nor do they take into consideration the factors noted above.

The government has been aware of this situation, both in regard to fields from various components of the electric power system as well as radio frequency and microwave. To date, no adequate, valid, unbiased studies have been done in this country. With the exception of the New York Dept of Health study on power line radiation, studies reporting potentially hazardous bioeffects have been done by individual scientists with their own resources. The great majority of well-funded studies reporting no bioeffects have been supported or performed by "parties of interest" such as the DOD, DOE and industry.

I am opposed to the installation of any additional radiating sources that would irradiate residential area until adequate facts have been obtained that reveal the actual levels of harm. I believe that the concept of NEXRAD is a good one, however, because of its continuous operation. I suggest that it be located where it would not expose residential areas or such installations as schools, etc.

Sincerely,

Medical College of Virginia
Virginia Commonwealth University

MR. N. MCLAUGHLI
4421 N. BENNETT HEIGHT
BATAVIA, NY 14020

March 27, 1990

Mr. Jim Dusen
8049 State Street Road
Batavia, New York 14020

Dear Mr. Dusen:

In response to our telephone conversation of March 26, 1990 I will react to your concern regarding the siting of a weather radar in your community at Batavia, New York.

It is not possible for me to express an opinion about the potential health effects of the microwave radiation from this source since I have no specific information about it. However, based upon recently emerging issues I understand your concern and can offer a few general comments that hopefully may put the problem into a more appropriate context.

Epidemiological studies have revealed an unexpected relationship of exposure to electromagnetic radiation at microwave, and lower frequencies and cancer incidence. To date, questions regarding the types of exposures that may affect cancer incidence remain unanswered. In other words, it is not possible to state a safe level of exposure in the absence of adequate information. A current review of this issue is provided in the reference:

Szmigielski, S. and Gil, J.: Electromagnetic Fields and Neoplasms, in Electromagnetic Biointeraction (Franceshetti, G., Gandhi, O.P. and Grandolfo, M., eds.), Plenum Press, New York, 1989, pp. 81-98.

I am enclosing a preprint of a chapter I wrote for this same volume in which I discussed some of the evidence indicating that microwave radiation affects living systems via mechanisms other than heating. The potential significance of this is to draw into question the adequacy of safety guidelines such as ANSI C95.1 that are based upon the thermal effects premise. Finally I enclose a reprint and a preprint of our recent research results which are further evidence of nonthermal microwave effects on cells.

I hope this information is useful in drawing attention to your problem.

Sincerely,

S.F. Cleary

Stephen F. Cleary, Ph.D.
Professor of Physiology

SFC/is/9994

Enclosures

Department of Physiology • Box 551 • MCV Station • Richmond, Virginia 23298-0551 • (804) 786-9756

Attachment #4

Letter from Dr. Lehrman for West Lapvile Area

NEW ROCHELLE RADIOLOGY ASSOCIATES, P.C.

THE CENTER FOR RADIATION ONCOLOGY

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DAVID B LEHRMAN, M.D.

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ISAIAH J. BELIGMAN, M.D.
IRA S. NOVICH, M.D.
ALVIN J. CHISOLM, M.D.
SHELLEY HAN WEINER, M.D.

May 7, 1990

To Whom It May Concern

I regret that I am unable to attend this evening's meeting concerning the proposed installation of the NEXRAD unit. I think that it is important to realize that sound concepts regarding the use and management of radiation are being ignored in the discussion of this installation.

History has taught medical science important lessions regarding all forms of radiation extending from ionizing radiation right through the entire electromagnetic spectrum including microwaves. The public should realize that our use and understanding of radiation in all of its forms is very incomplete. We should never forget that in the early part of this century that test tubes of ionizing radioactive material where held in the hands of some of this country's foremost scientists including Alexander Graham Bell who simply did not realize their danger. On a more modern scale, the danger of ultra-violet radiation in the development of skin cancer and other non-cancer problems such as cataracts is first being realized. Medical radiation including fluoroscopy and plain xray has undergone a marked reformation from the 1950's to the present as our concepts regarding its safety and application are constantly being reformulated. In Suffolk County we are still debating the hazards of video display terminals and their emissions. It comes as no surprise therefore to see a controversy emerge over other forms of electromagnetic radiation especially microwave radiation. Questions are beginning to arise from geographical locations in the United States and Canada concerning the possible linkage of this type of radiation to cancer and other health ailments.

It is very possible that we do not have at this time the sufficient biologic expertise to correctly measure the nature and type of damage being caused by these rays. Indeed, the damage may not be expressed in forms which we can clinically recognize at this time. It is important to realize that microwave radiation and other forms of radiation should not simply be dismissed as safe simply because they are not clear initiators of carcinogenic events. They must be fully investigated as possible co-promoters of these events as well as other non-cancer health hazards.

....continued....

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SHELLEY NAN WEINER M.D.

This data is clearly not available in any creditable form at the present time. The criteria of molecular and genetic judgement which we are currently using may very well be outmoded over the next five to ten years.

This does not mean that NEXRAD should not be built. It means however that careful consideration should be given as to where it is placed. In the use of ionizing radiation, all of us subscribe to the ALARA principle. This principle should be applied to all forms of radiation. ALARA stands for As Low As Reasonably Achievable. There are two basic concepts fundamental to this rule. The first is that exposure should only be of necessity and at the lowest level possibly attainable. The second is that when exposure is necessary that only the fewest number of people should be exposed. The NEXRAD concept, while commendable for its desire to achieve very low doses of radiation within currently acceptable "thresholds", violates the second concept. Clearly, it is ridiculous to expose a large population of people to any form of radiation when simply changing the location of the installation can improve the risk versus benefit equation dramatically.

It is important that this committee realize that there is no clear threshold for the development of biologic damage from any form of radiation. These equations are linear quadratic in nature. That is, they are increasing straight lines with no known threshold. As such using the ALARA principle is not only science, but basic common sense. It would be a tragedy to see a NEXRAD unit installed in a highly populated area and have to look back at a period of ten to twenty years to find out to our shock that there are indeed health hazards present. It is also important to realize however, that health hazards may not be present. We simply do not know.

....continued....

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ALVIN J. CHISOLM, M.D.
SHELLEY NAN WEINER, M.D.

NEXRAD may have the potential to significantly help our civilization. Its ability to predict weather and help in navigation may save numerous lives. Yet, health questions are emerging and until they are satisfactorly answered, it is important for the NEXRAD representatives to place these sites at locations where only the fewest number of residents will have any exposure. We can not rely on their so called "scientific judgement". The questions are first being asked and the data is not in. It may take a period of five years for a leukemia to develop and a period of twenty to thirty years for solid tumors to develop after exposure to various forms of radiation. Other health hazards such as the hastening of atherosclerosis, cataract formation, and infertility problems among others have yet to be fully researched. I strongly urge that these units be placed in sites that are appropiate as to population exposure and not merely allocated due to governmental costs and convenience.

Thank you for your attention.

Sincerely,

David B. Lehrman M.D

David B. Lehrman, M.D.

(1) Chief of Radiation Therapy & Radiation Oncology New Rochelle Hospital
(2) Former co-chairman American Cancer Society's Committe on Professional Education
(3) Member of New York State Radiologic Society's Committee on Radiation Oncology and Theraputic Radiology

DBL: jmk

Attachment # 5
Dr. Rodman's
letter
for
Clarence Hearing
(used with
permission)

16 June 1989
368 Hedstrom Drive
Buffalo, NY 14226

Councilman John Love
Clarence Town Hall
One Town Place
Clarence, NY 14031

Dear Councilman Love,

During May 1989, I was approached by members of CONRAD (Citizens Opposed to NEXRAD Radar) to investigate possible health effects from the proposed installation of NEXRAD in the Town of Clarence, New York.

This correspondence is not intended to supplant the extensive scientific literature on the issue of possible health effects from electromagnetic fields, especially microwave radar energy. The information here is as non-technical as I can make it. I do however, wish to inform you of my personal opinions and many well documented facts on this subject in order to better inform your community as to the controversy surrounding your request of me.

I regret that I was not previously approached on the logistics of the meeting schedule, as my professional obligations to attend the Bioelectromagnetics Society meeting in Arizona prevent me from being in attendance. In my place I have arranged for Mrs. Diane M. Rodman to deliver the contents of my opinions to the Town of Clarence on 21 June 1989.

Before discussion of the data in question, a brief outline of my technical qualifications to address these issues would perhaps be in order. Following my Bachelor of Science degree in Biology from Siena College, I attended Union College and followed course work for a BS/MS degree in Electrical Engineering and Computer Science. I have been involved in broadcasting as Chief Engineer of an FM broadcast station where I obtained experience with microwave systems. During my education process, I have been self-employed as a consulting broadcast engineer. I completed my MD degree from SUNY/Buffalo in 1981 and completed a postgraduate residency program in Ophthalmology. I have maintained interests in Amateur Radio and the effects of electromagnetic fields (EMF) on biological systems. These interests have led me to lecture on various occasions to groups on the possible health hazards of EMF as well as other areas of interest to me such as the design of high performance communication antennas.

I must mention at the outset I am deeply gratified that there exists in our society today a growing sense of interest of possible harmful health effects to exposure by

EMF. Numerous articles have appeared in the press and concurrently the New Yorker is publishing a three part article on these subjects. Amongst my own professional colleagues there is an increasing opinion that previously known, but not publicized, health effects after both brief and chronic exposure to microwave radiation are now much more than isolated anecdotical stories. For example, several scientists working with microwave systems in the laboratory have been reported to develop or succumb to rare and unusual cancers at premature ages. In another case, two laboratory workers were exposed to intense radar signals in the near field, one developed epilepsy and the other had a new onset of Parkinson's Disease. Additionally, in a epidemiological study of electronics technicians published in 1987, those personnel associated with assembly and testing of microwave equipment had a 10 fold increase in the risk of developing brain tumors after employment of 20 or more years. It is my opinion that such information may lead to an increasing suspicion that exposure to EMF, that was previously considered safe or unable to demonstrate harmful health effects, may be playing a pivotal part of the aforementioned devastating and catastrophic health effects.

Outside of professional communications with scientists in the Bioelectromagnetics Society I have carefully reviewed recent scientific literature concerning the biological effects of EMF on cells and animal models, including both low frequency and microwave signals. After comparison of these data, including epidemiological reports and studying the document entitled Next Generation Weather Radar Programmatic Environmental Impact Statement along with topography information on the proposed location, including adjacent dwellings, and public buildings, I have serious questions that the proposed NEXRAD system will not be without potential health hazard to the general public. I predicate this believe on various information that I would like to elaborate on briefly, before I conclude my remarks on this subject.

Since the publication of the 1984 description of NEXRAD, considerable scientific literature has been devoted to describing how weak EMF interacts with biological systems. This understanding, has brought forth evidence on the first significant change in our comprehension of possible mechanisms for the development of cancer in 75 years. Evidence is mounting that carcinogenic compounds and weak EMF, including pulsed microwave energy, are capable of molecular interaction with the cell membrane. This interaction is amplified and can modulate enzyme systems within cells, including enzymes associated with cell growth. In this way, both chemical and EMF stimuli are thought to act as cancer promoters. That is to say, interaction with the cell does not cause cancer, but may help to promote tumor formation in tissues that were previously exposed to an initiating event that left damage and the potential of cancer formation. It is suggested that EMF, such as pulsed

radar energy, even at levels far below the 5000 microwatts/sq cm designated in ANSI C95.1-1982, by virtue of the ability to influence cell growth may be implicated in the future as a more ominous health hazard than it is presently represented to be.

Studies on the effects of pulsed radar systems with biological systems such as the immune system and cell growth are presently being conducted. Recent experimental evidence on the ability of pulsed radar signals of similar description to NEXRAD, indicate that cells are capable of demonstratable changes in growth at microwave signals of less than 100 microwatts/sq cm. Furthermore, enhancement of effects were noted to be related to Pulse Repetition Frequencies in the range of 300 to 500 pulses per second. One will note that that those rates are well within the normal operating range of NEXRAD. It is felt that exposure to power densities of such systems above 10 microwatts/sq cm should be avoided as this seems to be the lower limit of present demonstratable biological interactions.

I will now make an effort to correlate my study of the location of NEXRAD with regards to the local topography in Clarence, New York and the avoidance of exposure to energies from the system in the 10 to 100 microwatts/sq cm range. Based on my interpretation of data supplied, I would recommend that personnel avoid elevations of approximately 100 feet above local grade within a radius of 1400 feet of the transmitting antenna. Lower elevations in the closest proximity to the transmitting antenna afford protection of exposure to the transmitted signal from the various sidelobes. The topography in the general area to the South and Southeast of the proposed site presents an opportunity for many local residents to be subjected to repetitive firstlobe exposure of to NEXRAD up to 40,000 microwatts/sq cm, depending on local height above mean sea level and distance from NEXRAD. This is due to the up hill terrain in the general direction of an area of significant population density. Paradoxically, it appears that residents living with in the immediate vicinity of NEXRAD are more protected from power density exposure in excess of 10 to 100 microwatts/sq cm than those residing up to one to two miles away, based on terrain. To further illustrate this fact, there appears to be an area of dwellings that are approximately at level with the antenna system on a radial of 170 degrees with a distance of 6000 feet. These residents would likely be subjected to mainlobe signals containing power densities up to 70,000 microwatts/sq cm.

Unfortunately, there is no present data to demonstrate adverse health effects on humans from intermittent, continual exposure to extremely high pulsed radar signals such as proposed by the location of NEXRAD. With increasing evidence that low amplitude EMF is capable interaction with cells to promote cancer formation and retard mechanisms of cellular immunity, including our own cancer fighting

abilities, it is felt that a prudent decision dictate that intermittent exposure to such signals be avoided.

In conclusion, scientific data on the mechanisms of interaction of low amplitude electromagnetic fields with cell membranes indicates that athermal exposure of various signals may be ultimately shown to be intimately associated with cancer promotion. The present ANSI C95.1-1982 Standard is incapable of addressing the significance of these data. There is strong doubt in my mind that proposed NEXRAD system would be completely without potential health hazard over the life of local residents. I definitely urge that the proposed site be changed due to the high local population density at elevations above mean sea level which would tend to expose those residents to repetitive power densities of unusually high magnitude from both mainlobe and firstlobe energies.

Sincerely yours,

David J. Rodman, MD
President, Buffalo Professional Consultants
716-845-2640

DJR:ei

Letter #14

April 19, 1993

, Letter to the Joint Systems Program Office
United States Departments of Commerce, Transportation, Defense

The Nexrad radar - Next Generation Weather Radar Programmatic Environmental Impact Statement was published in 1984, prepared by the Nexrad Joint System Program Office and was developed to, acquire, process and distribute improved weather information....
"The 1984 impact analysis indicates that the construction and operation of the Nexrad environmental impacts and all anticipated impacts are/or can be limited to minor local effects. Supplements to this Programmatic EIS will be prepared for those sites selected for radars where significant impacts are possible". The Nexrad radar is a very costly undertaking, being designed primarily for the detection of phenomena which cause severe weather and for flooding. This system which is being planned in the Town of Batavia, Genesee County

for the City of Buffalo, Erie County, New York. The People in Clarence, Sayville and the residents of Bennett Heights and other believers in Genesee County, requested through their representatives, that the Federal government leave this area, when the people no longer believed that the Departments of Commerce, Transportation, Defense, who said, ...

> "that upon examination of the environmental impacts of electromagnetic fields and non-ionizing radiation that might be emitted by the proposed Nepsad (Next Generation of Radar) facility on the flora, the fauna, and especially the people of the surrounding communities."

In this costly search something more than ~~[illegible]~~ was lost along the way.

where potentially significant adverse impacts are frozen, but sites are nevertheless highly desirable for other seasons, the impacts will be analyzed in depth. In these cases mitigation measures will be developed and supplements to the Programmatic EIS will be prepared."

... because there is always a beginning please allow me to introduce myself to the members of the Joint Programmatic System Office and all invited guests. My name is Miss. Flosen C. Gioia. I reside at 9 Cherry Street, Batavia (Genesee County), New York. Before any discussion of the data in question, I would like to inform you of my involvement in the controversy known as Nexrad, the potential dangerous health effects, and the proposed installation of the Nexrad Facility in the Town of Batavia,

... because there is always a beginning please allow me to introduce myself to the members of the Joint Programmatic System Office and all invited guests.

My name is Mrs. Florence C. Gioia. I reside at 9 Cherry Street, Batavia (Genesee County), New York. Before any discussion of the data in question, I would like to inform you of my involvement in the controversy known as Nexrad, and my belief of the potential dangerous effects this facility emitts on the environment, and my concern and frustration of the proposed installation of the Radar Facility in the Town of Batavia (Genesee County), New York.

Following the completion of an Associates in Arts degree, and a Bachelor's degree ... finally I completed the requisite

thirty credit hours for teacher certification. I became a candidate for the Office of Councilperson in the City of Batavia, and served three (3) years in this capacity, and became a candidate for the Office of Legislator representative of the 9th District in Genesee County, of which I have served for 12 years in this capacity.

I learned of the National Weather Service in November, 1989. January of 1990, at the Annual Genesee County Industrial Agency Dinner Meeting a Mrs. Donald Wunsch, a meteorologist in charge of the National Weather Service Office at the Greater Buffalo International Airport revealed that after "careful consideration Batavia had been selected for the Weather Services new high-tech

forcast office. I expressed my concern immediately to the press, the Executive Director, Neil T. Burns, of the Genesee County Industrial Development Agency and to Mr. Donald Wuesch from the National Weather Service that the Genesee County (I D A) Industrial Development Agency had "pushed" the project on the citizens of the County and secretely negotiated with the National Weather Service and some elected Legislators of the Genesee County Legislature without formal disclosure of the plans.

Immediately I released a statement to the people of Genesee County that an information meeting would be held as soon as pertinent data on this project could be obtained. A response from citizens in Genesee County, who had relatives in [illegible] New York and where a

a similiar attempt had been proposed for the installation of a Nexrad Facility.

A meeting was immediately arranged with the members of Conrad I (Citizens opposed to Nexrad Radar) and with the elected officials of Clarence. In a televised Committee meeting, especially arranged for some people from Genesee County. We were informed that a Public Hearing was held and when the residents learned of the potential hazards of electromagnetic radiation, and the debate within this scientific community with members of the National Weather Service, a decision was reached by the elected officials of Clarence and a unanimous vote was taken that the National Weather Service would not be welcome in this Community. With the video-cassette of the Public Hearing, and printed material from The New Yorker Magazine in February 1990, an informal

meeting was held to inform citizens of the public exposure from pulsed doppler radar systems and that there are no studies have been done guaranteeing long-term safety from such installations, neither are there studies that clearly demonstrate known risks from the levels the radar systems produce. There is an increasing opinion in the scientific community that cells communi-
14-1 cate with each other at the membrane level and low level electromagnetic fields can influence tumor formation or affect normal activities by disrupting the cellular communication. Moreover there is experimental laboratory data
14-2 that indicates pulsed microwave energy has additional biological effects over non-pulsed microwave sources.

These facts have made me very skeptical of the safety in locating

a doppler radar system near populated area. I believe that continual exposure to the public near such facilities should be considered a possible hazard until proved otherwise.

David J. Rodman M. D.

14-3 This letter I read at the informal meeting, and later at the formal meeting of the Legislature of Genesee County we all were privileged to hear the renowned Dr. David Rodman discuss the potential health risks. You have received or will receive Dr. David Rodmans statement at your meeting.

March of 1990 the Citizens of Bennett Height. in the Town of Batavia, Genesee County formed CoRad (Citizens opposed to Nexrad Radar) at the airport site since it was well within one (1) mile

35 homes.

A number of people from Clarence, New York contacted me to say that they had learned that another community, Sayville New York were informed that a Nexrad Facility was proposed to be installed in their community. A Rev. and Mrs. Don Malone, 24 Mobile Street, Sayville New York would be my contact. I quickly called the Malones' and we exchanged all pertinent information for months by phone, faxed and letters. We were all encouraged here in Genesee County by the quick developments that had occurred in Sayville regarding the proposed Nexra Radar Facility on Cherry Avenue in West Sayville. The citizens organization called themselves Consad II.

The citizens of Bennett Heights, now known as Consad III and myself were

aware of the representation afforded to the citizens of Sayville, New York. The Islip Town Officials and the Islip Supervisor, Frank Jones demonstrated their strong support and voiced their concern about a facility (radar) that would emitt electro-magnetic radiation and cause possible health effects upon the citizens of their community. We were impressed by Patrick G Halpin, Suffolk County Executive who wrote a letter to President Bush, "... to strongly request that the President direct the National Oceanic and Atmospheric Administration to to abandon its misguided attempts to construct a Nexrad installation in Sayville, New York... Executive Halpin stated, the next Generation Radar system or Nexrad is based on the Doppler radar principal This system, includes the transmission and reading of electro-magnetic radiation

... and long term exposure to electro-magnetic radiation emitted by such a facility could lead to a range of health problems despite the assurances of the National Weather Service to the contrary.. Because of the potential health consequences to the thousands of Suffolk County residents living within a mile of the proposed site, I call on you to put an immediate halt to this safety process... a carbon copy was sent to Long Island Delegation, House of Representatives and the United States Senate Now that is what one calls representation The letter had to be and deserved to be addressed. We were once again impressed of Congressman Thomas Downy's immediate concern and quick response to the people he represents. In a letter to Don Malone Congressman Downy voiced his concern about this facility from the very beginning but wanted to defer final judgement until the residents had a chance to voice their concerns to the Weather Service. Congressman [illegible] made his position very clear ... and

did oppose the construction of this Radar Facility in any residential area within the Second Congressional District.

That is how a "network of people" who had concern for each other had made things happen."

Even Senator Al D'Amato made a special trip to Sayville and agreed with the residents in Sayville that he would do x in his power to Remove the Federal Facility from this area — and that he did — the next thing we knew the facility had been scheduled for Brookhaven, N.Y.

What a sorry lot of Representatives we the people of Genesee County have. First the elected legislator from the Town of Batavia, and the Legislature's liaison of the Genesee County Industrial Agency supported the entrance of the National Weather Service here in Genesee County. By the way his name is Stephen Hawley

In fact 7 of the 9 legislators supported the National Weather Service in Genesee County after Legislators Borelli and Goia. There were letters and phone calls sent to Assemblyman Steve Hawley and Senator Hoffer, complete silence. Letters and phone calls to U.S. Senator Daniel P. Moynihan, just about a vampire and a letter was sent. Phone calls and letters to Congresswoman Louise Slaughter, although she did have a personal meeting with the Conrad III group also turned her face away. U.S. Senator Al D'Amato said he endorsed the National Weather Service in Genesee County, although the residents lived less than a mile to the proposed facility — as the citizens did in Sayville, New York. Maybe the life here is not as important as the life in Sayville, or maybe the approximate 3 million voters in Suffolk elected Senator Al D'Amato for Genesee County might have a mere 20 thousand. Senator Al D'Amato disagreed of course and stated that he supported the 7 votes of

Genesee County Legislator—. At the time we were unaware of the Senator's possible conflict of interest — his brother who is the attorney representing the Unisys Corporation, the Radar Systems' Manufacturer. Now we also have the many articles published in the New York Times that the "Weather Service Cites Radar Flaws" and that "Best Solution May be to End Unisys Contract For New System, all in 199. We also have press releases, "in the face of stern questioning at a Congressional sub committee meeting — hearing. Not after we all heard and saw the NBC "Dateline" anchored by Jane Pauley on April 8, 1993, about the National Weather Service and Representative James T. Scheuer.

Previously, I had stated that the Next Generation Weather Radar Programmatic Environmental Impact Statement published in 1984, prepared by the Joint Systems Program Office and developed to acquire process and distribute improved weather information ... Supplements to this Programmatic EIS will be prepared for those sites where significant impact are possible ... that the Draft Supplemental Environmental (SEA) of the Effects of Electromagnetic Radiation from the WSR-88D Radar is to be heard at the hearing of today. What is most amusing is the change of the name "Nexrad" — I guess it was psychologically dangerous. I also stated that this system now called WSR-88D (Nexrad) is being placed in the Town of Batavia, Genesee County, for the City of Buffalo, Erie County, New York.

In 1984, JSPO published a Programmatic Environmental Impact Statement (PEIS) analyzing the potential environment impact of the system. The PEIS concluded, in general, construction and operation of the Nexrad (now WSR-88D) system will have no significant adverse environmental impacts ... in particular, it found...

"that there is no reliable scientific evidence to suggest that, for the cases considered, exposure to RFR (radiofrequency radiation) from the Nexrad Radar will be deleterious to the health of even the most susceptable members of the population such as the unborn, infirmed or aged".

Sites for WSR-88D units are being selected to maximize the use of surplus or underutilized government property, reuse existing weather radar sites, achieve optimum network coverage,

minimize construction costs, and prevent
reduce environmental impacts.

The SEA review of research since 1984
found no scientific evidence that exposure
to WSR-88D Program will cause no
significant adverse biological impacts.
Similarly, the review found no adverse
effects will result from exposure to
WSR-88D power-line fields.... In
overall conclusion, therefore, ISPO proposes
that implementation of the WSR-88D
Program will not cause significant
adverse impacts on human health, or
hazards to electromagnetic systems. Thus
the earlier conclusion that construction
and operation of the WSR-88D system
would not cause significant adverse
impacts on the human environment
remains valid, and a Finding of
No Significant Environment Impact
(FONSI) is warranted."

Now, why doesn't this surprise no one!

Why was the statement of Dr. Rodman on page 7, "there is an increasing opinion in the scientific community that cells communicate with each other at the membrane level and low level electric magnetic fields can influence tumor formation or effect normal activities by disrupting the cellular communication. Where was this tested.

14-4 As this is a fact finding scientific hearing it is most ludicrous that a hearing was scheduled a couple of day. after the Christian Easter Celebration and the Passover. Why were some people informed just a few days before this hearing?

Why was a budget meeting of the sub-committee scheduled for April 28, 1992 was cancelled?

"In the Buffalo, New York Area the Genesee County Airport Site was selected because it is within a radius of 62 nmi, a Nexrad, oops WSR-88D, the proposed Genesee County Airport site should reliably detect weather beyond 2 to 4 nmi, except over hills to the southwest and north without interference from near-in clutter returns. That is, near-in clutter should not obscure weather returns...

14-5 I would suggest to you that this site could not and would not protect Buffalo at all because Buffalo is West of the Genesee County Airport, and most of the weather moves in from the West — therefore, if you have Radar Cast of the location your trying to protect, the effect is diminished.

14-6 ... understanding that the Doppler

14-6 (Cont.)

was tested in March, 1991 in Norman, Oklahoma but the National Weather Service has refused to devulged the results. Although it has been said that Ms. Findley has stated that Nexrad passed with flying colors which must be very doubtful to believe it is understood that the National Weather Service as an agency no longer has system procurement and that the responsibility is strictly with NOAA because the National Weather Service really "screwed up" in the Nexrad fiasco.

It is also rumored that the Office of Inspector General (Dept. of Commerce) must report out on how much is spent on software — and it is doubtful that the software could and would operate in a working environment.

I had previously stated that the site for Nexrad to be placed in the

County of Genesee. Yet this site is within less than 1 mile of the homes in Bennett Heights.

At the Public Hearing held in Sayville, New York, a presenter Mr. Leonard Solon, the Director of the New York City Bureau for Radiation Control stated that, "at the 17 Annual National Conference of 1985, "our current state of knowledge with respect to the psychological

14-7 effects of non-ionizing stood the same as it did in 1934." So, we dont really know enough about it. The only thing that's clear about the potential health effects of non-ionizing radiation is that the evidence is unclear, that it is inconclusive ~~all~~ and that we really need further research.

Another presenter a Ms. Holly Bixler, a mother of two children, is educated with

14-8 [illegible]

14-8 (Cont.)

how a study conducted of the long-range effects of low power electromagnetic radiation. By long range, I mean 10 to 20 years. It is ludicrous that the National Weather Service, or anyone could fathom the thought of placing this type of equipment in a residential area — Sayville or any residential area". Miss Bialer also said that Stanley Wasserman of the National Weather Service stated at the IEEE meeting at Polytechnic University in Farmingdale on April 18 1990: "I personally think its safe, but no, these are no guarantees for 20 years from now."

14-9

Another presenter Dr. Louis Bozzi, Deputy Assistant Administrator for modernization at the National Weather Service stated, "I want to recognize the ongoing public and scientific debate regarding whether or not there is a verifiable link between non-ionizing emission and human health Hazards. The presence of new

scientific theories indicates a possible indirect link between electro magnetic fields and existing known carcinogens. These studies are only five years old, and it could be perhaps ten years or more before any results can be consistently reproduced." (Has Mr. Baczzi heard the results from his own department. Mr. Baczi was questioned where would other locations be for Nepsad? He responded that they have Albany, Batavia, Binghamton and Long Island.

I would like to continue with the presenters at the Sayville, New York Public Hearing, and maybe in the future you will allow me to do just that, because there are dozens of citizens who were present.

Now, I would like the opportunity

to discuss some pertinent data that appeared in "U.S. News" on July 24, 1989. In fact the front cover which displays the title, Predicting Storms and in a picture we see a researcher in "Oklahoma" probe the "secrets" of a severe thunderstorm. Well into the story, it is said, "Initially, meteorologists thought they could infer upper-air temperature and wind pattern from satellite images. But according to John Brown of NOAA, the data while better than nothing, are not very accurate. "The glorious promise of satellite data simply has not materialized," he says.

A $1 billion overhaul of the National Weather Service promises to remedy many of the shortcomings of atmospheric samplings of today. By the mid-1990's a $700 million nationwide network of

Doppler radars will replace today's W
War II vintage conventional radars. A
prototype for the new system, dubbed
"NexRad" for Next-generation Radar, (NEXRA
is being tested in Norman. The Dopplers
promise to dramatically improve the de-
tection of severe weather, including
tornadoes. Both Doppler and conventional
radar pick up a so-called hook echo,
the signature of a tornadic vortex
inside a storm. But the hook echo
appears only 5 minutes before a torna
touches down and does its damage.
Dopplers however, also spot mesocyclones,
which develop up to half an hour
before a tornado descends. With
Doppler radar, forecasters should be
able to predict a strong tornado
as much as 20 minutes before it
occurs. The network also includes

a network of specialized radars called "wind profilers," which measure wind currents up to 10 miles high, and 1,000 completely automated surface-observation stations, based primarily at airports, that will collect data on clouds, wind, temperature and moisture every 6 minutes. How accurate will forecasters get at predicting damaging thunderstorms? Even next-generation sensors will not catch everything. Downbursts, for instance, are notoriously difficult to spot. They last only a couple of minutes; Doppler radar scans every 5 or 6 minutes. And Doppler radars can't measure the vertical movement of air. According to Ron Alberty, director of the National support facility in Norman, most tornadoes will escape detection in the Doppler era as well. While the really big [illegible] the easiest to spot, 90

percent are too short-lived and dim initiate to be picked up by a Doppler unless the funnel descends less than 25 miles from the radar.

The experts concur that they may never be able to forecast an individual storm much more than an hour in advance .... the new atmospheric sensors, especially Nexrad and the wind profilers should help forecasters issue more-focused severe storm warnings, however.

Currently the Weather Service alerts whole counties or large parts of counties. These were warnings in effect for most of three counties when severe winds wreaked havoc at the Yorkams'. If the new technologies had been available, forecasters might have been able to limit the warnings to the immediate environs of Bloomfield, for example.

That would do much to improve public confidence in warnings and help to ensure that they actually achieve their intended effect of limiting the damage, economic loss, injuries and fatalities that inevitably ensue when storms strike the unprepared. As it stands now, people frequently ignore warnings altogether or run outside to see what is going on instead of taking shelter. Meteorologists included. "There's no way I'd run my family into a shelter every time NOAA issued a warning [illegible] county admits," NSSL Director Doswell. The real sign that forecasting has come of age may be when weathermen start heeding their own advice.

It is too bad that Betty Carpenter did not do a follow up story. In December, 1984 in Norman, Oklahoma

14-10

14-10 (Cont.)

the new prototype for the new system dubbed Nexrad was an 85% failure

It has been stated recently that a new test was given in March of 1992 and Nexrad passed with flying colors so says Dr. Friday of the National Weather Service. Yet on the NBC program titled "Dateline" which was shown on April 8, 1992, it was stated to Representative James H. Scheuer, Chairman of the Sub Committe for the Department of Commerce admitted that not one test had been ever independently tested ever...."

I stated previously that the Nexrad radar, and another name cannot change it, and that this search cost more than money and "something" was lost along the way — its

integrety, which includes the members of the National Weather Service members of NOAA, the Departments of Commerce, Transportation, Defense and menembers (representatives) of Congress and the Senate.

I do apologize for not having this paper typed but all the typists were gone for the Holeday Weekend.

I do hope that this paper was able to be read at your hearing, and although, "I myself am not a scientist I am not ignorant either, I am an educated person and perhaps my degree is not in the field of electromagnetic radiation, but am qualified to know what "integrety" means.

Respectfully

Florence C. Gibson

Letter #15

TOWN OF BATAVIA

TOWN HALL
4165 WEST MAIN STREET ROAD
BATAVIA, NEW YORK 14020-1299
PHONE: (716) 343-1729

FOUNDED 1802

April 14, 1992

Mr. David Smiley
National Oceanic & Atmospheric Administration
Systems Program Office (JSPO 121)
1325 E. W. Highway
Silver Spring, MD 20910

Dear Mr. Smiley:

At the March 14, 1990 Batavia Town Board meeting, the Town Board adopted a resolution opposing the location of the National Weather Service on East Saile Drive in the Town of Batavia. The vote was unanimous.

While the Board is not opposed to a weather station per se, it is opposed to locating one within a two mile radius of a residential or otherwise populated area because of the potential health risks involved. It has not been made clear to the Board that the lov level microvave radiation emitted from the tover will not have an adverse affect on human health and until it is made clear the Board will continue to maintain its present position. To make it clear that it will not pose a health risk a non-governmental study in an uncontrolled environment must be conducted.

15-1

Enclosed is the certified resolution adopted by the Batavia Town Board opposing the location of the weather station on East Saile Drive in the Town of Batavia.

Sincerely,

June C. Vukman

June C. Vukman
Supervisor

JCV:lc
Enclosure

OPPOSING THE NATIONAL WEATHER SERVICE

WHEREAS, the Genesee County Legislature in conjunction with the Genesee County Industrial Development Agency has proposed to locate the National Weather Service on the south side of East Saile Drive, Town of Batavia, and

WHEREAS, this land and the surrounding area is "prime land" for residential and industrial development in the Town, and

WHEREAS, development in this area will significantly benefit the Town by broadening its tax base, and

WHEREAS, the National Weather Service will not broaden the Town's tax base because it will be tax exempt, and

WHEREAS, the negative impact of low level microwave radiation from NEXRAD on human health has neither been proven or disproven thus determining the health effects on humans as an "unknown", and

WHEREAS, this "unknown" may have a negative impact on development in this area thereby limiting growth and ultimately adversely effecting the tax base, and

WHEREAS, the close proximity of the NEXRAD tower to the Genesee County Airport may jeopardize expansion of the airport in the future,

NOW, THEREFORE, BE IT

RESOLVED, the Batavia Town Board hereby goes on record as opposing the location of the National Weather Service on East Saile Drive for the above stated reasons and be it further;

RESOLVED, that upon adoption of this Resolution the Town Clerk is directed to forward a certified copy to the Genesee County Legislature, the Genesee County Industrial Development Agency and the National Weather Service.

Offered by: Councilman Severyniak

Second by: Supervisor Vukman

APPROVED BY UNANIMOUS VOTE. (5-0).

CERTIFICATION

STATE OF NEW YORK)
:
COUNTY OF GENESEE)

I hereby certify that the foregoing is a true and accurate copy of a resolution adopted by the Batavia Town Board on March 14, 1990.

Dated: Batavia, New York
April 14, 1992

(SEAL)

Rubie K. Levins

Rubie K. Levins
Batavia Town Clerk

STATEMENT FOR HEARING APRIL 21, 1992 ON SEA OF 1992

FOR NEXRAD (WSR-88D)

Lest you be tempted to consider us and our opposition to the proposed siting of NEXRAD in Genesee County radical, unreasonable or fanatic, let me remind you that similar stands have been taken by numerous citizens and public officials before us.

In May of 1990 in West Sayville, New York, the New York State Assembly held a hearing organized by Maurice Hinchey, chair of the Assembly Committee on Environmental Conservation, to examine the controversy surrounding the safety of radar in general and the NEXRAD tower in particular. Several public officials, school superintendents and citizens spoke in opposition to the proposed site so close to schools and homes. Speakers read information and statements of support from several scientists and medical doctors whose views were in agreement with that taken by the citizens opposed to the siting. I'm sure you recall that NWS, at the suggestion of Senator Alfonse D'Amato, changed the location of the Long Island NEXRAD to a site away from a residential neighborhood.

We are asking no more and no less. Since our bodies are the same as those of the people of West Sayville, it remains a mystery to us why Sen. D'Amato (or NWS) has refused to take a supportive stand in Batavia as he did in West Sayville. We can only guess at his motivation for this. The information which the people of West Sayville read and on which they relied is the same data which persuaded us to believe that "prudent avoidance" was the correct way for us to proceed. Our convictions were similarly determined by the material proclaimed at that hearing and additional information and contacts with other scientists and medical doctors whom we sought out on our own.

Our citizens' group has no quarrel with the goals of the NWS or its need for newer, more accurate weather radar. We applaud their efforts to save lives through this Master Plan; we merely request that the locations they choose for the radar tower in Genesee County and across the country allow us and others to avoid the potential dangers of the long-term exposure of the radar until DEFINITIVE conclusions and a CONVINCING consensus is reached in the scientific community. We are a long way from that in 1992.

To support my statement that we are not the only ones concerned about the uncertainties and unanswered questions surrounding the issue of the health risks associated with non-ionizing radiation, I wish to mention the statements and recommendations found in the NEXRAD hearing report promulgated by NYS ASSEMBLY.

Allow me to cite to you some of the recommendations found on pp. 20 and 21 of the report. I urge you to study all eight (8) of the recommendations; they are consistent with our stand and supportive of our cause.

Especially important and numbers four (4), five (5), six (6) and seven (7). I quote:

16-1 4. The New York State Assembly, through resolution, should ask the U.S. EPA to develop a standard for occupational and general public exposure to non-ionizing electromagnetic radiation based on scientific

16-1 (Cont.) conclusions reached from comprehensive INDEPENDENT research.

5. The New York State Assembly, through resolution, should ask the National Weather Service to consider postponing additional siting of NEXRAD until the question of potential health effects of nonionizing electromagnetic radiation is resolved.

6. The New York State Assembly, through resolution, should ask the National Weather Service to develop a list of alternate sites for siting of the NEXRAD radar, taking into account potential environmental and community health impact as well as meteorological considerations.

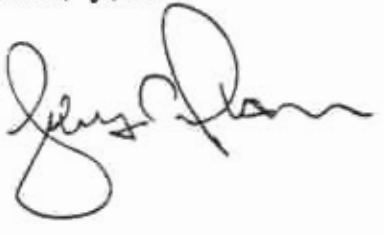
7. The New York State assembly, through resolution, should ask the National Weather Service to hold public hearings and coordinate siting of the NEXRAD facilities at the local and state levels, with an eye toward improved community involvement and input.

In addition, there is currently a bill presented to the NYS Assembly by Maurice Hinchey (A4657) and the Committee on Environmental Conservation which would set exposure standards for the state and request that NWS new NEXRAD tower be banned in New York State.

During the course of our two plus years of opposition, we have repeatedly stated our case simply and firmly: We have no opposition to NEXRAD, but until the scientific controversy can be solved, we do not want the radar tower built within two miles of homes, schools, churches or any place to which the general public has access. We have cited scientists and medical doctors who agree with our stand and who have continually encouraged us in our efforts.

I trust that these comments serve to show that our opposition group is not the lone voice "crying in the wilderness"; others who have studied the issue but who have a less personal interest in the siting problem have taken the same stand as ours.

Thank you.



8049 State St. Rd.
Batavia, NY 14020

To Whom It May Concern:

It is very clear to anyone who has done any reading at all about the bioeffects of RFR that the newly compiled SEA, prepared by SRI for the National Weather Service, is an inadequate document. The research that was selected by SRI "experts" is only that which serves to prove the point that the NWS has so well paid them to make. The point being, of course, that weather radar is totally harmless. So much of the cited research is "old news" and so little of the newer research is included. Further, no independent researchers were asked to comment.

17-1

Some longterm researchers, such as Dr. Stephen Cleary, Dr. Robert Becker, and Dr. Zaret are inadequately represented or omitted entirely. Dr. Szmigielski's Polish military radar study and Dr. Hansson's Swedish study of radar workers are conspicuously absent from the references.

17-2

More specifically as regards Dr. Cleary, with whom we have been in contact, the more recent studies are the most significant. In a personal letter to a member of our group, Dr. Cleary stated "Epidemiological studies have revealed an unexpected relationship of exposure to electromagnetic radiation at microwave, and lower frequencies and cancer incidence." Also "it is not possible to state a safe level of exposure in the absence of adequate information." In an overview for the 1989 book, Electromagnetic Biointeractions, Cleary states "Review of the bioeffects literature... provides convincing evidence that RF radiation, and other types of electric and magnetic fields, can alter living systems via direct nonthermal mechanisms." This does not seem to be the same conclusion SRI has reached. Dr. Cleary is a well-respected researcher, yet only a few (3) of his older studies are included in SRI's references. Are SRI "experts" perhaps afraid to admit what more recent research is finding?

17-3

To be truly fair to the average citizen who has neither the financial

means nor the scientific expertise to respond in an appropriate way to this document, the National Weather Service should request written responses to said document from some independent scientists who have not been under government contract or grants recently. The NWS should be prepared to pay these researchers for their time and effort. Members of our group would be glad to offer some suggestions as to those researchers who are not so easily swayed by government dollars.

Prudent avoidance is the best course to take in the case of so much uncertainty and scientific disagreement. As researchers find more and more bioeffects of RFR, we must make responsible decisions as to where to site new facilities that may soon be proven to have long term harmful effects to the residents of the surrounding areas.

Sincerely,

Catherine J. Dusen

Catherine J. Dusen

April 21,1992

To whom it may concern,

A great number of scientists feel that there is a potential health risk with exposure to the electromagnetic fields associated with the proposed Nexrad weather radar facility. That risk is said to be highest in the range of a 1 1/2 mile radius from the tower, and that information has been widely publicized from the beginning in our fight to keep this installation out of the shadow of our homes. Unfortunately, in our society today, a great deal of apathy exists when it is someone else's problem. Residents who are located well beyond what has been stated as the area of potential harmful exposure saw no need to be either well informed on the subject, nor alarmed should it come to our county. This was to be expected.

We however, who will have to live or die in the shadow of this tower, and have the most to lose, see things differently. While we do not dispute the need for new and improved weather radar, we feel strongly that the health and safety of citizens should come first. In the case of this radar facility, controlled by the Federal Government and the National Weather Service, we do not believe that this is the case. While there is no evidence to support either its absolute safety or possible hazard, there is substantial confirmation on record from respected scientists, that suggest that we are facing a significant health risk with radar
18-1 exposure. The documents we have been provided with, namely the Nexrad site survey for Genesee County, and the recently completed SEA study are nothing more than a whitewash. Both reports indicate a callous disregard for getting to the truth and insuring public safety. As often is the case, the federal government cannot afford the risk that a thorough, independent study could indicate a health problem with a system such as radar.

It has been stated by the some of our local legislators that the installation of the Nexrad weather radar facility in our county is welcomed by a majority of the people who reside here. It nas also been their contention that there is a general feeling that there is no potential health risk to residents associated with this radar facility. A county wide survey, organized by the legislative supporters of Nexrad, and conducted by the Jaycees was used to support their claims of acceptance. In a county of over 58,000 people, 248 were said to have been polled with barely a majority indicating that they would want the radar facility to come to Genesee County.

In the Site Survey and Enviormental Assessment for the Genesee County site the chosen location was described as "farm land, open fields, scattered farmhouses and barns. and a residential sub-division located 4500 ft. away from the tower". While the radar tower itself is located on land zoned for agriculture, the fact remains that along with that sub-division there are a total of 67

families residing within a one mile radius, and 262 families within a one and one half mile radius. It had been stated that in addition to the county wide survey, a majority of the residents located in the vicinity of the proposed site favored Nexrad. This was a statement that CONRAD III disputed and in order to determine the validity of that statement, we found it necessary to conduct our own study.

Although it was not to be a scientific survey, it was however a polling of the opinion of all residents most affected by the installation: those within the 1 1/2 mile radius. To poll everyone in the area thought to be most at risk is a far better indicator on public opinion than questioning those who will not be affected in any way by the radar.The following is the overall result of that poll with a complete copy of the poll attached for your review.

Of the 67 homes located within 1 mile 57, (85%) participated in the poll.

18-2

41 out of 57 (71.9%) were well informed about the proposal and:
47 out of 57 (82.5%) were against the proposed site.

When you expand the area to a 1 1/2 mile radius, 178 out of 262 (67.9%) responded.

106 out of 178 (59.6%) were well informed about the proposal and:
128 out of 178 (71.9%) were against the proposed site.

What did this show? That an overwhelming majority of the residents within the proximity of the radar tower are opposed to the site. Dr. Friday, in his visit to Batavia to promote the Nexrad site made two significant statements.

18-3

1. He cannot guarantee that no health problems will ever occur as a result of this Nexrad facility.
2. The National Weather Service has a policy that they will not go where they are not wanted.

The citizens who are convinced that the scientists are correct in stating that there is a potential health risk, especially to children, have spoken. The National Weather Service and Nexrad will not be welcome in our neighborhood.

Very truly yours,

Dan Voltura

Dan Voltura
CONRAD III Project Coordinator

RATIONALE FOR A SURVEY CONDUCTED BY CONRAD III IN DECEMBER 1990

Due to the significance placed on the two surveys conducted in Genesee County, one by the JAYCEES and the other by Stephen M. Hawley, we felt it was in our own best interest and in the interest of fairness that we conduct our own OPIONNAIRE of the residents within a one mile radius and a one and a half mile radius of the proposed site at the airport.

To accomplish this task, fourteen two-person teams were organized to deliver a one page letter and information sheet about the potential health risks to each home which would later be involved in the opionnaire. These teams delivered the information sheet one week in advance of their visit to ask six questions of the residents in each home.

Each opionnaire was identified with the name and address of the respondents and each respondent was asked to sign the document on which opinions were checked. These were then collated and the statistics which follow were calculated.

No pressure was put on the respondents and several chose not to answer the six questions at all. These were designated as NO POLL. People who were not available when the teams visited them several times, were listed as NO CONTACT.

The statistics speak for themselves and it is obvious that they refute the statements about the previous two surveys.

SUMMARY OF SURVEY RESULTS

CONDUCTED BY CONRAD III AND NEW HOPE FELLOWSHIP CHURCH

DECEMBER 1990

SURVEY OF RESIDENCES WITHIN A ONE MILE RADIUS OF THE PROPOSED SITE

ARE YOU AWARE OF THE NWS' PLANS TO INSTALL A WEATHER RADAR FACILITY AT THE GENESEE COUNTY AIRPORT?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
57	0	0	4	6

ARE YOU AWARE THAT THIS FACILITY WILL BE WITHIN 1 MILE OF HOMES AND WITHIN 1,000 FT. OF A CHURCH AND DAY CARE CENTER?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
56	1	0	4	6

ARE YOU AWARE THAT MANY PHYSICIANS AND SCIENTISTS ARE CONVINCED THAT DAILY EXPOSURE TO THIS TYPE OF RADAR CAN PRODUCE NEGATIVE HEALTH EFFECTS, ESPECIALLY TO CHILDREN?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
51	5	1	4	6

DID YOU PARTICIPATE IN THE SURVEY CONDUCTED BY MR. STEPHEN HAWLEY WHEN RESIDENTS WITHIN ONE MILE OF THE SITE WERE POLLED?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
35	21	1	4	6

DO YOU FEEL YOU CAN MAKE AN INFORMED OPINION ON THIS RADAR FACILITY?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
41	14	2	4	6

DO YOU AGREE WITH THE CITIZENS AND CHURCH MEMBERS WHO ASK THAT THIS RADAR FACILITY NOT BE PLACED NEAR RESIDENCES OR THE DAY CARE CENTER?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
47	4	6	4	6

STATISTICAL SUMMARY OF ONE MILE RADIUS POLL

POSSIBLE CONTACTS 67

COMPLETED THE POLL 57

85% COMPLETED THE POLL

ON QUESTION 6:

DO YOU AGREE WITH THE CITIZENS AND CHURCH MEMBERS WHO ASK THAT THIS RADAR FACILITY NOT BE PLACED NEAR RESIDENCES OR THE DAY CARE CENTER?

47 OUT OF 57 RESPONDENTS ARE AGAINST THE PROPOSED SITE AT THE GENESEE COUNTY AIRPORT

82.5% AGAINST THE PROPOSED SITE

47 OUT OF THE 51 WHO ANSWERED EITHER YES OR NO TO QUESTION 6

92% AGAINST THE PROPOSED SITE

SUMMARY OF SURVEY RESULTS

CONDUCTED BY CONRAD III AND NEW HOPE FELLOWSHIP CHURCH

DECEMBER 1990

SURVEY OF RESIDENCES WITHIN A 1 1/2 MILE RADIUS OF THE PROPOSED SITE

ARE YOU AWARE OF THE NWS' PLANS TO INSTALL A WEATHER RADAR FACILITY AT THE GENESEE COUNTY AIRPORT?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
170	5	3	26	58

ARE YOU AWARE THAT THIS FACILITY WILL BE WITHIN 1 MILE OF HOMES AND WITHIN 1,000 FT. OF A CHURCH AND DAY CARE CENTER?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
157	18	3	26	58

ARE YOU AWARE THAT MANY PHYSICIANS AND SCIENTISTS ARE CONVINCED THAT DAILY EXPOSURE TO THIS TYPE OF RADAR CAN PRODUCE NEGATIVE HEALTH EFFECTS, ESPECIALLY TO CHILDREN?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
147	24	7	26	58

DID YOU PARTICIPATE IN THE SURVEY CONDUCTED BY MR. STEPHEN HAWLEY WHEN RESIDENTS WITHIN ONE MILE OF THE SITE WERE POLLED?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
54	119	5	26	58

DO YOU FEEL YOU CAN MAKE AN INFORMED OPINION ON THIS RADAR FACILITY?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
106	68	4	26	58

DO YOU AGREE WITH THE CITIZENS AND CHURCH MEMBERS WHO ASK THAT THIS RADAR FACILITY NOT BE PLACED NEAR RESIDENCES OR THE DAY CARE CENTER?

YES	NO	NO OPINION/UNDECIDED	REFUSED POLL	NO CONTACT
128	18	32	26	58

STATISTICAL SUMMARY OF 1 1/2 MILE RADIUS POLL

POSSIBLE CONTACTS 262

COMPLETED THE POLL 178

68% COMPLETED THE POLL

ON QUESTION 6:

DO YOU AGREE WITH THE CITIZENS AND CHURCH MEMBERS WHO ASK THAT THIS RADAR FACILITY NOT BE PLACED NEAR RESIDENCES OR THE DAY CARE CENTER?

128 OUT OF 178 RESPONDENTS ARE AGAINST THE PROPOSED SITE AT THE GENESEE COUNTY AIRPORT

72% AGAINST THE PROPOSED SITE

128 OUT OF THE 146 WHO ANSWERED EITHER YES OR NO TO QUESTION 6

88% AGAINST THE PROPOSED SITE

STATEMENT FOR APRIL 21, 1992 HEARING

I would like to make some comments about standards since the SEA relies so heavily on comparisons between NEXRAD calculations and standards. First, there seems to be disagreement about standards. The following chart will show the proof of this statement:

19-1

SOURCE OF STANDARD	MILLIWATTS PER CENTIMETER SQUARED
OSHA	10.00
ANSI (revised)	1.8 - 2.0
EPA	.1
JOHN HOPKINS MED. SCHOOL	.1
CHINESE GOVERNMENT	.05
Dr.Leonard Solon, Director of the New York City Health Department's Bureau of Radiation Control	.05

In addition, I wish to quote the following pertinent statements from the Report on the PROPOSED NEXRAD from the NYS Hearing on May 10, 1990 in West Sayville, New York. The quotes are taken from the official document provided by Assemblyman Maurice Hinchey, chair of the Committee on Environmental Conservation.

19-2 - " The standards development process for non-ionizing radiation occurs through the review of the scientific literature and debate among scientists with differing views. The standards are set through a process of consensus, not on the basis of objective, scientific fact or experimentation. And there is a great deal of debate within the scientific community over the bio-effects of exposure to this radiation. The standards in the Soviet Union indeed are 1,000 times lower than the current ANSI standards. And in 1985, The New York City Department of Health's Bureau of Radiation Control, recommended a general population exposure of .05 milliwatts/centimeter squared--100 lower than the current ANSI standard"

19-3 - " The Institute of Electrical and Electronic Engineering itself, in a 1989 position paper states-- 'there is insufficient information to define safe and unsafe field levels...There is general agreement that more research is needed to define safe limits of human exposure... The roles of field strength, duration of exposure, and intermittent versus continuous exposure are also unknown...It will take additional years for the biological sciences to determine what component of exposure, if any, is a factor in health risks."

19-4 - "Dr. Solon also states :'It should be emphasized that those standards (ANSI) are for occupational guidance and not for protection of the general public, including potentially exceptionally vulnerable persons, children, pregnant women and the embryos they carry."

19-5 - "It is not possible at this time to determine safe limits for human

19-5 (Cont.) exposure to power frequency electromagnetic fields." Source of this quote is "The Human Considerations in Bio-effects of Electric Fields" by William Harrison Mehn, Commonwealth Edison Company and Northwestern University Medical School, Chicago, Illinois.

19-6 - "Even with improved coordination of research, it will not be possible to prove the absence of hazard, which means that regulators cannot guarantee total safety when they set standards. Standards are one way by which society balances the benefits of technology against potential risks." This quote was taken from "The Microwave Problem" by Kenneth Foster and Arthur W. Guy, 1986, Scientific American.

- Mr. Joseph Verdone, the Superintendent of Schools of Sayville, New York: "It is incredible to me that the NATIONAL WEATHER SERVICE and our federal government would plan installation less than 400 yards (1200 ft.) from an elementary school that houses 600 students."

N.B. The Sonshine Patch Day Care Center run by NEW HOPE FELLOWSHIP CHURCH is approximately 700 - 800 FEET from the proposed radar tower in Batavia, New York.

19-7 - "According to the FCC, evidence of harmful biological effects is less clear at low power RF radiation intensities, those intensities which produce heating. These non-thermal effects include changes in the immune system, neurological effects, behavioral effects, evidence for a link between microwave exposure and the action of certain drugs and compounds, a 'calcium afflux' effect in brain tissue, and a suggestion that microwaves might be involved in cancer promotion under certain conditions." However, contradictory experimental results have also been reported in many of these cases, and further experiments are needed to determine the generality of these effects and whether they constitute a threat to human health."

Just the word CONTRADICTORY makes us firm in our stand for safety until the conclusive evidence is found.

19-8 - "For example, researchers at John Hopkins University's have found that relatively modest levels of radio-frequency radiation, l;levels which are below those required to produce thermal effects,are capable of causing cellular damage in the cornea of rhesus monkeys. If the length of exposure increases, so does the damage. These exposure levels are felt by professional radar workers and not by the general public. However, the findings are far from conclusive, and definitive answers aren't likely to be found until there is MORE FUNDING FOR RESEARCH."

19-9 - "Samuel Milham studied all of the death certificates of all adult-male deaths in Washington State between 1950 and 1979 to see if deaths from non-Hodgkins' lymphoma, as well as leukemia, might be linked to occupations with exposure to electromagnetic fields. The results of that analysis were the findings that were published in The New England Journal of Medicine in the summer of 1982. Since then, Samuel Milham has learned that non-Hodgkin's lymphoma is a type of tumor that often develops in people who have impaired immune systems, so it may turn out that magnetic fields promote cancer by weakening the immune system."

19-10 - "In 1965, Dr. Robert O. Becker and Howard Friedman, a psychologist at the V.A. Hospital in Syracuse, New York, exposed human volunteers to pulsed magnetic fields of similar frequency and considerably greater

19-10 (Cont.) strength than those associated with magnetic storms, and found that doing so significantly slowed the volunteer's ability to react to the appearance of a light by pressing and relensing a telegraph key. A year earlier, however, a Soviet investigator named Yuri Alexandrie Kholodov had reported that exposure to even stronger magnetic fields caused areas of cell death in the brains of rabbits, and when Becker and Friedman learned of this they discontinued their experiments and advised others to do the same."

19-11 - "Almost all scientific studies have researched the relationship of high power lines to cancer. Epidemiological studies have focused on a search for cancer because of a historical observation and the public saliency for cancer, rather than because cancer is thought to be the most likely effect. Epidemiological studies suggest that, if there is a link, it is that exposure to 60 Hz fields does not initiate cancer, but rather, may serve to promote the growth of cancerous cells, once the cancer has been initiated by other causes."

19-12 - "According to information presented by Ms. Pettick, 90% of the research on the health effects of electromagnetic radiation is funded by groups which have a vested interest: The Department of Energy, the Department of Defense, the Air Force and the power companies."

19-13 - "At this point, it remains unclear in what mode and what power density the NEXRAD radar would actually operate. Of major concern to the New York State Assembly Committee on Environmental Conservation, after reviewing possible power density levels of NEXRAD and New York City guidelines, is the fact that there are three public schools located within 1/2 mile of the proposed NEXRAD site. This includes an elementary school, housing 600 children, located 1200 ft. from the proposed radar tower. In contrast to the testimony of Dr. Boezi, the power density of the main beam at 4,000 ft. from the tower, according to Mr. Leo Birenbaum, is .220 milliwatts per centimeter squared which is well above the Solon recommendation of .05 milliwatts per centimeter square."

D. Keith Bow

May 25, 1992

Roshan Shaikh
East Yaphank Civic Association
Box 566,
Yaphank, NY 11967

Mrs. Jo Ann Wargo
Secretary to Program Manager
NEXRAD Joint Systems Program Office
National Oceanic and Atmospheric Administration
1325 East-West Highway,
Room 15146
Silver Spring, Maryland 20910

RE: COMMENTS ON SEA DRAFT FOR THE NEXRAD JSP WSR-88D RADAR

Dear Ms. Wargo,

Attached please find some comments prepared by the undersigned and approved by the board members of the Civic Association of East Yaphank, Long Island, New York.

This document was written primarily, to inform the members of our association about the contents of the SEA Draft mailed to Mr. John Wolfe of East Yaphank Civic Association.

On behalf of the president and the members of the board of our association, I would appreciate, if you could include these comments in your records. Would you please, let us know if you have any further information or comments to address some of the concerns raised in our comments.

Thank you very much for your cooperation.

Sincerely,

Roshan Shaikh.

May 17, 1992 **Comments on SEA Draft**

By

Roshan Shaikh

After reviewing the draft on Supplemental Environmental Assessment (SEA) of the effects of electromagnetic radiation from the proposed WSR-88D Radar, I have outlined some comments as under:

1. The proposed radar is going to emit radiations known as RFR (Radio Frequency Radiations). It has been established that these radiations heat up the molecules of living cells that in turn can cause several diseases and malfunctions in humans and animals some of these include: 20-1

   a. Biological and developmental abnormalities.
   b. Disruption of immunological systems.
   c. Nervous system disorders.
   d. Subtle histopathological and histochemical changes.

2. Since almost all magnetic and electro-magnetic systems today, do emit some levels of RFR, a "safe" doze of these radiation levels that the humans and animals can take has been also established by the researchers and regulatory authorities such as EPA etc. 20-2

   a. Typically, it has been suggested that we can take 9 to 10 milli watts per square centimeter of radiation for 6 minutes without any harm.

   b. We can also take 1.8 to 2.0 milli watts per square centimeter of radiation for 30 minutes without any harm.

3. It has been suggested that the proposed radar WSR-88D will only emit radiations in the following order: 20-3

   a. At 2700-3000 Mhz frequency and at 475 Kilo Watt peak radiated power will emit 3.85 milli watts per square centimeter of radiation for 30 minutes in its so called "search light mode".

   b. In its "standard mode" the radar will constantly emit 0.6 milli watts per square centimeter of radiation for 1.57-4.71 micro second 1304 times per second.

4. The draft document prepared by the government (Joint System Program Office - JSPO) suggests that since the radar is
20-4 operating much below the dangerous levels of radiations, there is absolutely no harm to humans and animals. This is also been supported by an EPA study. [4]

5. Based upon the above observations, I find the following:

   a. The radar is probably not harmful. However, the draft document has failed to show what are the potential effects of small dozes of sustained RFR for a very long duration.

   b. If what the JSPO draft suggests is correct, then the "standard mode" radiations may not be at a harmful level, but who is to say that these levels will be maintained through out the life of the radar. What is the working life of the radar? Not given in the draft
20-5 any way.

   c. The " search light mode" is the high radiation mode. We need to know little bit more about it:

      i. how many times in 24 hours?
      ii. what are the supporting facts that during a 30 minute period radiation at 3.85 milli watts per square centimeter for 7.5 minutes is really not dangerous to humans and animals?

   d. Since there are 3 other sites in New York state out of the total of 153 radar sites [5], we may like to contact residents of these sites to form a joint and much informed opinion.

==========

References:

1. Page 34. JSPO Draft.
2. Page 30, 32. JSPO Draft.
3. Page A8. JSPO Draft Appendix.
4. Page 52. JSPO Draft.
5. Page 8. JSPO Draft.

Letter #29

OFFICE OF STATE PLANNING

Office of the Governor

MAILING ADDRESS: P.O. BOX 3540, HONOLULU, HAWAII 96811-3540
STREET ADDRESS: 250 SOUTH HOTEL STREET, 4TH FLOOR
TELEPHONE: (808)587-2846, 587-2800

FAX: Director's Office 587-2848
Planning Division 587-2824

Ref. No. P-3014

May 5, 1992

Mr. Mark A. McCloy
Program Manager
Systems Program Office, JSPO (SPO121)
National Oceanic and Atmospheric
Administration
1325 East-West Highway
Silver Spring, Maryland 20910

Attention: Mr. David R. Smiley
Deputy Program Manager

Dear Mr. McCloy:

Subject: Hawaii Coastal Zone Management (CZM) Program Federal Consistency for the WSR-88D Weather Radar Facilities in Hawaii

Upon reviewing the Draft Supplemental Environmental Assessment of the Effects of Electromagnetic Radiation from the WSR-88D Radar, we have determined that pursuant to the Coastal Zone Management Act (16 U.S.C. 1451 et. seq.) and 15 Code of Federal Regulations 930, CZM Federal consistency concurrence is required for developing and operating radar facilities at the three locations proposed within the State of Hawaii--Kamuela, Island of Hawaii; Molokai; and South Kauai. Even if the radar facilities will be on Federal land, the facilities and radar operations will directly affect Hawaii's coastal zone.

29-1

We are enclosing copies of our Federally approved CZM Program and our CZM Federal consistency guide. Thank you for the opportunity to review the environmental assessment. If you need any assistance or have any questions, please call our CZM Program at (808) 587-2878.

Sincerely,

Harold S. Masumoto
Director

Enclosures

State of North Carolina
Department of Environment, Health, and Natural Resources
Division of Radiation Protection
P.O. Box 27687 • Raleigh, North Carolina 27611-7687

James G. Martin, Governor
William W. Cobey, Jr., Secretary

Dayne H. Brown, Director
Telephone 919/571-4141

MEMORANDUM

TO: Melba McGee, Project Review Coordinator
Division of Planning and Assessment

FROM: Lanny L. Watkins, Chief
X-Ray Control Section

SUBJECT: Project Review for the WSR-88D Radar,
Project Number 92-0822

DATE: May 5, 1992

As an In-House Review of the environmental assessment from the effects of electromagnetic radiation, associated with the WSR-88D Radar, it is substantiated that there are no known effects of non-ionizing radiation at the levels which this unit will operate. Therefore, there is no professional reason why this project could not go forward as proposed. The benefits of the use of this radar would certainly be capable of maintaining and improving the quality of life for our citizens. (See page 3 of the report, II. Purpose and need for the Action.)

There are, however, a number of scientific statements and conclusions completed by the researchers that are misleading, inaccurate and incomplete which could certainly be misinterpreted during public review. Some examples are as follows:

31-1 1. One example of misleading scientific analysis is found on page 21, IV. Affected Environment, part E. When comparing ionizing and non-ionizing radiation characteristics the last sentence on this page combines dental x-ray machines with sun-tanning lamps. This paragraph compares apples and oranges, and ends up with apples or oranges; whichever you prefer. I believe the researcher is trying to demonstrate there are exposures to ionizing radiation by the public from a number of sources as well as exposures to non-ionizing sources, and we must evaluate the benefit versus the risk to justify the exposure. The entire section (E, p. 20 - 21) should be rewritten because they tried to condense the information too much.

2. On pages 37 and 38 under V. Environmental Consequences, part C. Human Health Effects of RFR, paragraph 2.6.3 the data is fractionated and fails to reflect the intent of the scientific analysis. In demonstrating the significant effects of radio-frequency-radiation (RFR) the data does not give the basis for the percentages in comparing non-ionizing radiation exposure versus ionizing (X-Ray) and chemical exposure to fruit flies. In paragraph 2.6.2 the researcher states that male mice exposed to electromagnetic pulses appeared to benefit from the exposure. There is no determination made as to what is the benefit, but this should have been left out or concluded. Under paragraph 2.6.3 on page 39, the researcher determines that stress from confinement is more harmful than the RFR levels, but does not detail the comparison of the control groups.

31-2

The referenced examples were given so that researchers can either give complete synopsis on their subjects of discussion or give their summation and list references.

In summation as an In-House-Reviewer, the proposal for the WSR-88D Radar at the three locations in North Carolina would be of great benefit to our citizens. There are no known hazards associated with the levels to which this unit is restricted.

Letter #32

State Clearinghouse

OKLAHOMA
DEPARTMENT OF COMMERCE
OFFICE OF FEDERAL ASSISTANCE MANAGEMENT

May 14, 1992

ATTN: MR. DAVID R. SMILEY, DEPUTY PROGRAM MANAGER
NATIONAL OCEANIC & ATMOSPHERIC ADMINISTRATION
SYSTEMS PROGRAM OFFICE, JSPO (SPO121)
1325 EAST-WEST HIGHWAY
SILVER SPRING, MARYLAND 20910

RE: 14D211 - DRAFT SUPPLEMENTAL ENVIRONMENTAL ASSESSMENT OF THE EFFECTS OF ELECTROMAGNETIC RADIATION FROM THE WSR-88D RADAR BY THE NEXT GENERATION WEATHER RADAR JOINT SYSTEM PROGRAM, (SAI #920514-010)

The environmental information for the above referenced project has been reviewed in accordance with State of Oklahoma Review procedures and Section 102 (2)(C) of the National Environmental Policy Act by the reviewing agencies charged with enforcing environmental standards in the State of Oklahoma.

The state agencies, comprising the Pollution Control Coordinating Board, have reviewed the proposed project and offer the following specific comments:

The Oklahoma Archeological Survey - Comments are attached and made a part of this letter.

The Office of Federal Assistance Management requires no further review.

for Lucille McShane
Don N. Strain
Director

DNS:lrm

pc: LSB
DOC

6601 Broadway/P O. Box 26980 ● Oklahoma City, Oklahoma 73126-0980
Telephone (405) 841-9375

APR 27 1992

The University of Oklahoma

OKLAHOMA ARCHEOLOGICAL SURVEY
1808 Newton Drive
Norman, Oklahoma 73019-0540
(405) 325-7211

April 24, 1992

Don Strain
Office of Federal Assistance
Management
P.O. Box 26980
Oklahoma City, OK 73126-0989

Re: Proposed "Supplemental Environmental Assessment of the Effects of Electromagnetic Radiation from the WSR-88D Radar" by the NEXT Generation Weather Radar Joint System Program Office. State Clearinghouse No. 14D211.

Dear Mr. Strain:

I have evaluated the above referenced project for its potential impact on Oklahoma's archaeological resources. It does not appear that the radiation will effect cultural resources. However, it is possible that proposed locations for NEXT radar stations could result in disturbance of archaeological resources. If the proposed locations for NEXT stations at Enid, Oklahoma City, and Tulsa would result in construction at previously undisturbed locations, then further consultation regarding potential impact on archaeological resources will be necessary. 32-1

This review has been conducted in cooperation with the State Historic Preservation Office, Oklahoma Historical Society.

Sincerely,

Robert L. Brooks

Robert L. Brooks
State Archaeologist

RLB/lw

cc: SHPO

7635 Oak Orchard Road
Batavia, New York 14020
May 19, 1992

Mr. David Smiley
SEA Coordinator
NOAA Systems Program Office
SSMC-2, Room 15166
1325 East West Highway
Silver Spring, Maryland 20910
Attn: Mrs. Jo Ann Wargo, (301) 713-0144

Dear Mr. Smiley:

Thank you for the copy of the Draft Supplemental Environmental Assessment of the Effects of Electromagnetic Radiation from the WSR-88D Radar, received on May 15, 1992.

We feel that several significant studies demonstrating adverse effects of exposure to RFR have been omitted from the SEA. Our comments follow.

Section III.B.2 - Description of the Network

Site selection criteria are discussed in this section. We feel that health, safety and remoteness from residential areas should also be considerations.

David B. Lehrman, M.D., Chief of Radiation Therapy and Radiation Oncology at New Rochelle Hospital, and a member of the New York State Radiologic Society's Committee on Radiation Oncology and Therapeutic Radiology wrote the following comments for the New York State Assembly Hearing on May 10, 1990 on the environmental impacts of the Next Generation Radar (NEXRAD) Doppler Effect Radar System:

35-1 "...all of us should subscribe to the ALARA principle. This principle should be applied to all forms of radiation. ALARA stands for As Low As Reasonably Achievable ... it is ridiculous to expose a large population of people to any form of radiation when simply changing the location of the installation can improve the risk versus benefit equation dramatically."

"It may take a period of five years for a leukemia to develop after exposure to various forms of radiation and a period of twenty to thirty years for solid tumors to develop after exposure to various forms of radiation ... I strongly urge that these units be placed in sites that are appropriate as to population exposure and not merely allocated due to governmental costs and convenience."

Section III.C - Alternatives to the WSR-88D Program

Satellite systems are listed as one alternative to the WSR-88D. In regard to this alternative, the 1984 PEIS states the need for "new sensors to provide the necessary spatial resolution and
35-2 information on internal storm intensity and dynamics." The NASA space program and electronic technology in general have seen great advances in the past eight years and such sensors may now be available, making satellite systems a more viable alternative. The SEA should not assume that the sensor limitations present in 1984 still exist today.

Section V.C.2.6 - Mutagenesis, Cytogenetic Effects, and Carcinogens

The SEA states that "the scientific literature does not provide scientifically credible evidence that exposure of either mammalian or nonmammalian subjects to low levels of RFR produces mutations or cytogenetic effects, or that such RFR induces or promotes any form of cancer in mammals or cultures of mammalian cells."

We feel that it is important to consider the opinions of experts like Dr. Lehrman. In a letter dated May 6, 1990, Dr. Lehrman stated:

> "It is very possible that we do not have at this time the sufficient biologic expertise to correctly measure the nature
> 35-3 and type of damage being caused by these rays. Indeed, the damage may not be expressed in forms which we can clinically recognize at this time. It is important to realize that microwave radiation and other forms of radiation should not simply be dismissed as safe simply because they are not clear initiators of carcinogenic events. They must be fully investigated as possible co-promoters of these events as well as other non-cancer health hazards."

In his book, Cross Currents, Robert O. Becker, M.D. sites a 1983 report by E. Manikowska-Czerska, P. Czerska and W. Leach of FDA's Center of Devices and Radiological Health:

> "The researchers concluded that chromosomal abnormalities were produced by the microwave exposure at dose rates far below those producing a heating effect. Furthermore, they noted that the mechanism appeared to be a direct effect of the microwaves on the chromosomes themselves."

Section V.C.2.9 - Immunology and Hematology

The WSR-88D will chronically expose humans to low level RFR. Unfortunately, few studies have been done to investigate the effects of such chronic exposure. A study by the University of Washington, described in this section of the SEA, involved chronic exposure of rats to RFR over a 25 month period. 18 exposed rats developed malignancies compared to only 5 sham exposed rats. Although the exposed group had nearly four times the incidence of malignancies, the results were considered to be nonsignificant.

Dr. Becker includes this experiment in his book, Cross Currents and discusses aspects of the experiment that were not considered in the SEA:

> "In the early 1980s, the U.S. Air Force School of Aerospace Medicine funded a very large, very expensive study at the University of Washington, under the direction of Dr. Arthur W. Guy. In this study, rats were continuously exposed to high-frequency microwaves of 2.45 gigahertz (with one gigahertz equaling one billion hertz) at approximately 0.5 mW/cm^2, twenty times lower than the 'safe' thermal level. The exposures lasted for as long as twenty-five months, and 155 different measures of health and behavior were collected.
>
> 35-4 This appeared to be a well-designed study that would finally answer the question of whether there were any potential hazards to human beings from chronic exposure to microwave radiation. According to Guy, 'The results revealed few differences between the exposed and control rats, and those differences for the most part were either not statistically significant or came and went, suggesting that they may be due to chance.'
>
> However, one striking observation was noted: 'Primary malignant tumors developed in eighteen of the exposed animals but in only five of the controls.' Guy hastened to explain that the incidence of cancers even in the experimental group was actually *lower* than normally expected for the strain of rat used in the experiment. He suggested that no hasty conclusions should be drawn, and that a 'consensus among most investigators that the only strong evidence for the hazards of microwaves is found at high levels of exposure' was still valid.
>
> This project was widely reported in the press and discussed at scientific meetings, and it was the subject of a major article in the September 1986 issue of *Scientific American* (from which the above quotes have been drawn). A significant aspect of the experiment was not reported either in that article or in the popular press - but at the scientific

meeting at which the results of the study were first reported, it was revealed that all of the animals used, both experimental and control, were *gnotobiotic* (a term meaning germ and virus free). This circumstance alone was responsible for a major part of the $5 million cost of the project.

To produce gnotobiotic animals, the young must be delivered by cesarean section under the strictest possible sterile operating-room conditions (much more stringent than those in use in operating or delivery rooms for people). Following delivery, the animals must be raised and then housed in totally sterile environments for the entire duration of the experiment. This type of environment is akin to the decontamination rooms used to house the astronauts after they returned from the moon, or the 'bubbles' within which children born without immune systems are housed.

The use of gnotobiotic animals seems to be not only totally unnecessary, but undesirable as well. Neither we nor the laboratory rat normally live in a sterile world, devoid of bacteria or viruses. On the contrary, we live surrounded by uncountable numbers of organisms. We generally do not get sick unless we are injured and bacteria enter the body through the wound, or unless our immunity is inadequate and we get a communicable disease or infection. An experiment on germ- and virus-free animals has no relevance to the real world.

35-4 (Cont.)

This point becomes even more apparent when two established facts are considered. First, present evidence shows that at least 20 percent of human cancers are caused by viral infection, and the percentage is considerably higher in animals. Therefore, animals that are maintained in a germ- and virus-free state have an incidence of cancer that is much lower than expected. Second, it is well established that exposure to any abnormal electromagnetic field produces a stress response. If the exposure is prolonged, the stress-response system becomes exhausted, and the competency of the immune system declines to below normal. In such a state, animals and humans are more susceptible to cancer and infectious diseases.

One can only conclude that the experiment at Washington was deliberately designed to sharply reduce the incidence of cancer and infectious diseases in the exposed animals. There can be no other reason for the requirement that the animals be gnotobiotic.

Therefore, if we knew these facts in advance, and we wanted to set up a 'scientific' project to expose animals to microwaves for a long time but were required to get negative results, we would have only one choice - to use germ- and

virus-free test animals. Being gnotobiotic, both the unexposed control animals and the exposed experimental animals would be protected against the usual dangers of infection and cancer. In Guy's study, the fact that the experimental animals had a lower-than-normal incidence of cancer was totally expected. What was unexpected and highly significant was that even with this protection, the cancer incidence in the animals exposed to microwaves was four times that in the control animals.

The well-designed experiment that should have 'proved' that microwaves are safe fell into a trap, and the nature of the trap is revealed by the types of cancer that occurred in the experimental group. These were mainly limited to cancers of the pituitary, thyroid, and adrenal glands; these cancers were accompanied by a significant number of pheochromocytomas, which are benign tumors of the adrenal glands. There were no significant cancers of any of the usual tissues.

35-4 (Cont.)

The experiment was designed to prevent the results of stress, but the planners forgot that it would *produce* stress. Because stress resistance is mediated chiefly through the three glands just mentioned, we must conclude that the microwave exposure produced an extremely high level of stress - so much so that the resultant prolonged hyperactivity of these glands led to their becoming cancerous. Considering the extreme stress experienced by the exposed animals, if the animals had been normal (rather than gnotobiotic) the entire experimental group would have died of infection or cancer long before the close of the experiment.

Some of the 155 biochemical determinations done by Guy in the course of the experiment confirm this interpretation. Plasma cortisol is one of the chemical substances produced by the adrenal glands under conditions of stress, and it was one of the substances measured in the experiment. At the start, the plasm cortisol was equal in both the contrcl and experimental groups; in the early months of microwave exposure, however, cortisol in the experimental group was elevated above that in the control group, indicating that the experimental animals were reacting to stress. By the latter phase of the experiment, the plasma cortisol of the exposed animals was depressed below that of the controls, indicating that the stress-response systems of the experimental animals had become exhausted. This result is exactly as expected for a condition of chronic stress.

These data, which are buried in the multivolume official Air Force report of the project, were first published in the July-August 1984 issue of *Microwave News*. The experiment was planned cleverly, but not cleverly enough. It clearly

indicated that chronic exposure to microwaves, at levels twenty times below the established safe thermal level, produced profound stress and ultimately exhaustion of the stress-response system. Because the experiment involved gnotobiotic animals, this resulted only in a significant increase in cancers of the stress-response glands. Had the experiment been performed under real-world conditions, the results would have been catastrophic for the exposed group of animals."

35-4 (Cont.)

We feel that the data obtained from this experiment should be reevaluated in light of the information provided by Dr. Becker.

General Comments

i) All thresholds and limits are given as averages. The effect of peak exposure values should also be explored. Leo Birenbaum, a research associate professor at Polytechnic University in New York City raised this point in a letter to West Sayville citizens opposed to NEXRAD:

35-5

"Is it really safe to expose a person repeatedly, many hundreds of times per second, over long periods of time, to these power densities? Under these circumstances, do our bodies respond to peaks or to averages? No one knows for sure."

ii) The SEA stresses thermal effects of RFR and states that "RFR at the power densities of WSR-88D signals adds heat at rates well below the human body's ability to dissipate heat". Little is said, however, about the stress effects of exposure to RFR. Dr. Becker, in Cross Currents, states:

35-6

"The scientific data at this time indicate that microwaves have major biological effects at power levels far below those required to cause heating. ... The hazard comes from the fact that exposure to microwaves, like exposure to any abnormal electromagnetic field, produces stress, a decline in immune-system competency, and changes in the genetic apparatus. Thus the levels of exposure that the government says are 'safe' are in fact not safe at all."

iii) Effects attributable to RFR are sometimes mentioned and dismissed without sufficient explanation. An example is found in section V.C.2.8.1, in which the SEA states that "certain specific RFR induced changes in the brain could be interpreted wrongly as BBB alterations". We feel that any biological changes attributable to RFR warrant further investigation.

35-7

Conclusion

Based on the preceding comments, we must disagree with the conclusion reached in section V.C.3.12 that "There is no potential for significant effects to result from exposure to RFR from WSR-88D units". We feel that there is sufficient doubt about the safety of exposure to RFR at the levels expected for the WSR-88D. More research is necessary.

Thank you for the opportunity to review and respond to the Draft SEA. We hope that our comments will be helpful to you, and we look forward to hearing from you soon.

Sincerely,

Barry J. Debbins

Catherine M. Debbins

96 Besemer Rd.
Ithaca, NY 14850
May 12, 1992

Mr. David R. Smiley, Deputy Program Manager
Next Generation Weather Radar
Joint System Program Office
National Oceanographic and Atmospheric Administration
U.S. Department of Commerce
Washington. DC 20230

Dear Mr. Smiley:

Enclosed please find my response to the draft SEA on the WSR-88D weather radar. I have included also several attachments which I wish to be included in the public record of the review of this document.

I trust you will share with me the record of comments on this draft SEA, once those documents have been recorded. Thank you for the opportunity to respond.

Sincerely yours,

Joel Ray

May 12, 1992

Response to April 1992 Draft of the Supplemental Environmental Assessment (SEA) of the Effects of Electromagnetic Radiation from the WSR--88D Radar

Submitted to: David R. Smiley, Deputy Program Manager, NOAA

Submitted by: Joel Ray, 96 Besemer Rd., Ithaca, NY 14850

The SEA on the WSR-88D radar is one of the most intellectually indefensible documents purporting to review the literature on radiofrequency (microwave) and electric and magnetic radiation that, in fifteen years of reading such reviews, I have ever encountered.

Were its implications not so potentially noxious for human safety and for long-respected ideals of ethical integrity in American science, it would hardly bear rational comment. Yet the burden this report places on largely uninformed citizens to respond appropriately, and the implications that follow from the possibility that the SEA might be accepted at face value by government evaluators, requires that it be addressed, in however incomplete and general a fashion.

The patterns of intellectual sloth and dishonesty I have perceived in this report fall into several categories:

37-1 It uses the passive voice, "probably," "unlikely," "seems," "apparently" and other such fudge words so often in describing the invalidity of scientific studies reporting bioeffects of EM radiation that on the face of it no astute scientific evaluator could justify this document

as convincing, even if he or she knew nothing about the literature. The report relies on simple assertion to deny that credible or reliable scientific information exists to raise the possibility of potential hazard to humans, when in fact ample evidence does exist. It ignores many important studies that have found deleterious biological effects. It misrepresents other studies that included cautions about their own data, interpreting those cautions as ground for disqualification as legitimate science. It presumes artifacts to be responsible for certain reported effects, without identifying them.

37-1 (Cont.) This pattern of evasions, misrepresentations, distortions, and omissions can lead to only one conclusion: The contracted SRI analysts have been given at least implicit marching orders from NSW and the DOD (consistent with the Bush administration's political determination to undermine regulatory protections for American citizens, and with the US military's refusal to admit there is a nonthermal RFR hazard) to find no significant impact. The strain this injunction engenders, when the analysts are faced with numerous studies finding clear effects at nonthermal levels of RFR and from low magnetic fields, is palpable. The sense of unreality it causes, when the reader realizes how much important work has been ignored, is absolutely dislocating.

To review some of the more glaring omissions (with the understanding that my use of the passive voice in this summary always implies the SRI

37-2 analysts as the active party): Zaret, who has found numerous clinical

37-2 (Cont.) cases of nonthermal microwave cataracts over 25 years, is completely ignored. Frey's blood-brain-barrier work, which continues to be 37-3 confirmed, is trashed. His work on effects of magnetic fields on the opiate and dopamine systems of the brain, finding clear behavioral effects, is completely ignored. Dumanskiy's work on EEG effects is ignored. Marino's three-generation study, finding clear growth effects 37-4 in 2nd and 3rd generations, is misrepresented. The Battelle studies which found confirmations of Marino's work are misrepresented. Shandala's 37-5 findings of altered kidney function and immune system effects are ignored.

37-6 Zinaida Gordon's findings of clincal microwave effects are ignored. Becker, who is a founder of the science of biology and EM fields, is virtually ignored; specifically, his work on behavorial effects related to the earth's magnetic fields is ignored, as are his reports of effects 37-7 on biological cycles, his report of a cancer cluster near microwave towers, and his report of cancer cell proliferation under exposure to DC current (roughly confirmed by Winters, and again by Akamine, both

37-8 using AC fields). Friedman, who found an increase in polycythemia among

37-9 radar workers, is ignored. Czerski's findings of immune system impairment are ignored. Szmigielski's report of a threefold increase in cancer 37-10 among Polish military exposed to microwaves (especially in blood-forming

37-11 organs) is ignored. Stevens' provocative thesis on the relation between breast cancer and EMFs is ignored. Reiter's and Wilson's separate work on the pineal gland and EMFs is ignored. The many and various early 37-12 studies finding effects at or near the WSR-88D frequency (see Attachment A), especially in Eastern bloc countries, are presumed not to exist.

37-13 The histories of the microwave irradiation of the Moscow embassy, and the New York hearing on biological effects of electric and magnetic fields, are grossly misrepresented (see Brodeur's Zapping of America, Steneck's Microwave Debate, and Marino and Ray's Electric Wilderness).

37-14 The numerous Leal/Delgado-"Henhouse" reports of effects of weak magnetic
37-15 fields on developing chick embryos are ignored. The Lotz/Saxton report of hormonal effects is ignored. Hicks' report on second-generation
37-16 increase in eye cancer is ignored, as is Spitz's report on 2nd-generation nervous system tumors, Rivas' report on 2nd-generation decrease in body weight, and Salzinger's report on delayed response in the 2nd generation.

37-17 Swerdlow's report on eye cancer increase is ignored. Numerous reports
37-18 on leukemia increase are ignored: Gilman; Flodin; Pearce; Stern;
37-19 Juutilainen; Linet. Hannson's clinical report on abnormal proteins in
37-20 the cerebrospinal fluid of microwave workers is ignored. Koslov's report on Alzheimer symptoms in a primate is ignored. To anyone who has followed the developments in this field over 15 years, this report has an Alice-in-Wonderland unreality, where fact is denied and reality stood on its ear.

37-21 The report is based on the theory that only thermal effects from RFR can harm. This theory has been conclusively shown to be flawed by, among others, the hundreds of studies showing effects of 60 hz radiation on animals and humans. As 60 hz fields are incapable of creating thermal effects at any intensity, the effects they elicit in animals and humans are fraught with implications for the nonthermal bioeffects of microwaves.

The SEA is written as if in a historical vacuum. Never is it suggested, 37-22 as is the case, that other authorities besides ANSI, EPA, BPA, and the military have seriously called into question the present federal RFR 37-23 guidelines for humans. Never is any anecdotal information, such as provided by numerous lawsuits, local siting battles, and the increasing reports of individuals, included. Never is it pointed out that the 37-24 present safety limit within the Johns Hopkins Applied Physics Lab is 150 microwatts/cm squared, or that numerous experiments have been done in the US and the former Eastern bloc countries showing possibly hazardous effects from exposures as low as 50 microwatts, or that the Soviet and Chinese population standards are 25 and 50 microwatts respectively, or 37-25 that American cities and states have established population exposure levels of 150-200 microwatts or less. Never is it recognized that Soviet scientists asserted on the basis of experimental and clinical work <u>two decades ago</u> that non-thermal microwaves could have deleterious effects.

The strategy of this report is to arrogantly call into question <u>any</u> study that has found troubling effects, either by lapsing conveniently into the passive voice by saying the study "has been questioned," without identifying the source; by baldly misrepresenting the authors' own conclusions without any substantiation that an objective evaluator could judge; by presuming that the effect reported isn't relevant to the technology under question; by wrongly and perversely presuming that animal effects cannot be extrapolated to human hazard; and by raising

protocol questions that no evaluator can contest without further evidence.

37-26 The report was written by engineers and physicists, none of whom appears to have serious credentials in biology. It simply ignores the enormous intellectual ferment in the field of electromagnetic bioeffects by pretending it doesn't exist. It has nothing to say about the millions now being spent by governments around the world, and in independent research efforts, to identify the mechanisms by which nonthermal EM
37-27 radiation bioeffects are mediated in biological systems. The report could have been written in the 1950s for all the notice it affords the many new developments in the field, especially in the last decade.

Obviously the economic imperative driving the WSR-88D system is simply too great to allow its sponsors to admit that there is even a question about the human impacts of RFR radiation.

A finding of "no significant impact" on the basis of this evaluation, especially when many of the sites proposed by NWS have humans living within a half-mile of a radar that will emit 2200 milliwatts peak pulsed power at 800 feet, would be a travesty of science and public policy.

37-28 I append to these comments my own listing of about 60 studies, conducted at or near the WSD frequency, most showing effects that cannot possibly be thermal (Attachment A), and another listing of 80 or so studies at

37-28 (Cont.) other frequencies, all showing hazardous effects from nonthermal EM radiation (Attachment B).

Finally, I wish to draw attention to the ethical issue here. The hardened position of this SEA is meant to deflect questions about ethics; were there even possible harmful effects, clearly the writers would have to suggest caution, and thus raise the possibility of the resiting

37-29 of many of the 115 WSR-88D radars. It is because of this ethical dimension--involving the possibility, given current knowledge, of being accused of experimenting on humans--that the writers of this document have taken their extraordinary steps to ignore the real evidence, discredit the rest, and assert that the WSR-88D facility cannot cause health effects in humans.

Yours sincerely,

Joel Ray

Microwave exposure studies at or near the frequency of the NEXRAD weather radar--1969-88

In some cases the intensity or frequency or both are not specified (see note below on human studies).

Most of these studies involve exposures well below the US ANSI 5-milliwatt standard. The reader should be reminded that eastern European standards, as in the former Soviet Union, and presently in China, are a hundred times as strict as the American standards (25 and 50 microwatts/cm squared). Many of the studies cited below served as the basis for those standards.

All of the studies are in uw (microwatts/centimeter squared--a microwatt is one millionth of a watt, or one thousandth of a milliwatt). This indicates the absorbed power level per unit of square area.

Calculations by Professor Leo Birenbaum (see NYS Assembly report, Jan. 1992) show that the NEXRAD antenna will create a power level of 220 microwatts/cm squared at a distance of 4000 feet; 140 uw at 5000 feet, and 130 uw at one mile. Some of the studies cited below found effects in animals and humans at even weaker levels. Hazardous effects have been found at the entire range of microwave frequencies.

Animal studies

Date	Investigator	Exposure Frequency, intensity	Result
1973	Bychkov	100 uw, 3 Ghz	effects on EEG
1974	Dumanskiy	1-10.5 uw, 2.5 Ghz	effects on EEG
1975	Prey	200 uw, 1.2 Ghz	increased permeability of blood-brain barrier
1977	Oscar	30 uw --	increased permeability of blood-brain barrier
1978	Grin	50 uw, 2.4 Ghz	increased norepinephrine in brain
1978	Dumanskiy	100, 1000 uw, 2.4 Ghz	brain enzyme reduction
1975	Roberti	-- --	lowered blood cholinesterase
1973	Tolgskaya	60-320 uw, 3 Ghz	atrophy of hypothalamus neurons
1977	Mitchell	600 uw, 2.45 Ghz	hyperactivity

1979	Thomas	1000 uw, 2.45 Ghz	potentiated effect of chlordiazepoxide on behavior
1974	Dumanskiy	5-10 uw, 3 Ghz	elevation of excreted corticoids
1973	Demokidova	— 3 Ghz	altered adrenal weight
1973	Demokidova	153 uw, 3 Ghz	altered thyroid weight
1976	Baranski	5000 uw --	increased incorporation of iodine, increased number of cytosomes, enlarged Golgi apparatus
1973	Markov	153 uw, 3 Ghz	short-term hypertension, long-term hypotension
1974	Kartsovnykh	25-50 uw, 3 Ghz 500 uw, 3 Ghz	white blood cell increase white blood cell decrease
1978	Gonchav	10-50 uw, 2.4 Ghz 500 uw, 2.4 Ghz	increased glycogen decreased glycogen
1974	Czerski	3000 uw, 2.95 Ghz	decreased erythrocyte production, effect on iron metabolism
1975	Szmigielski	3000 uw, 3 Ghz	immune system impairment
1979	Shandala	1-500 uw, 2.4 Ghz	immune system impairment
1971	Dronov	50 uw --	immune system impairment
1979	Shandala	500 uw --	immune system impairment
1975	Czerski	500 uw, 2.95 Ghz	immune system impairment
1977	Vinogradov	5-50 uw, 2.4 Ghz	immune system impairment
1974	Czerski	1000 uw, 2.9 Ghz	altered cell mitosis in bone marrow
1978	Dumanskiy	100-1000 uw, 2.4 Ghz	altered liver metabolism
1976	Dumanskiy	10-1000 uw, 2.4 Ghz	effects on liver mitochondria
1976	Shandala	50, 500 uw, 2.4 Ghz	altered kidney function

1974	Miro	2000 uw, 3 Ghz	increased protein synthesis in liver, thymus, and spleen
1975	Minayev	570 uw, 2 Ghz	decrease in vitamin B6 levels in major organs
1975	Gabovich	10, 100, 1000 uw, 2.4 Ghz	altered distribution of trace metals in major organs
1975	Ilchevich	10-50 uw, 2.4 Ghz	disturbed ovarian morphology and fertility, altered postembryonic development
1973	Markov	153 uw, 3 Ghz	decreased weight gain
1984	Szmigielski	10,000 uw, 2.45 Ghz	fetal abnormalities from synergy with teratogen
1976	Kapustin	50, 500 uw, 2.4 Ghz	chromosome abnormalities
1972	Baranski	3500 uw, 3 Ghz	disorders in lymphocyte mitosis
1970	Yao	100-10,000 uw, 9.4 Ghz	(in vitro) chromosome aberrations, disrupted RNA synthesis, reduced protein production
1969	Baranski	7000 uw, 2.9 Ghz	(in vitro) chromosome abnormalities
1985	Guy	480 uw, 2.45 Ghz	malignancies, enlarged adrenal glands
1982	Szmigielski	500, 1500 uw, 2.45 Ghz	lung, breast and skin cancer inducement
1983	Kues	-- 2.45 Ghz	cornea abnormalities

Human studies

These are clinical and epidemiological, and often the strengths and frequencies are not known; they are studies of radar workers and microwave repairmen known to be exposed, usually chronically.

1973	Sadchikova	20-30 uw	--	headache, fatigue, irritability; cardiovascular change; abnormal blood pressure and ECG
1974	Sadchikova	--	--	similar results
1969	Fofanov	10-170 uw, 30 Ghz		bradycardia, decrease in pumping efficiency of heart
1966	Monayenkova	--	--	similar results
1966	Drogichiva	--	--	cardiovascular disorders
1974	Klimkova	--	3-30 Ghz	headache, fatigue, EEG changes
1973	Sokolov	--	--	decreased leukocytes, increase in red blood cells
1975	Lancrangan	10-100 uw, 3.6-10 Ghz		sperm abnormalities
1968	Majewska	--	.6-10.7 Ghz	lens opacities
1970	Kheifets	--	.3-300 Ghz	lens opacities
1973	Odland	--	military radar	eye defects
1974	Tengorth	--	--	lens opacities and retinal lesions
1974	Zydecki	100-1000 uw	--	lens opacities
1969-80	Zaret	--	--	cataracts
1981	Friedman	--	--	polycythemia (rare blood disorder)

1988	Szmigielski	--	--	lymphatic cancer and leukemia
1982	Lester	--	air force radar	cancer
1985	Hannson	--	1-10 Ghz	abnormal proteins in cerebrospinal fluid

Sources: Electromagnetism and Life, Becker and Marino (State University of New York Press, 1982); Modern Bioelectricity, ed. Marino (Dekker, 1988); Microwave News; Journal of Bioelectricity; EPA Review Draft 600/6-90/005B, "Evaluation of the Potential Carcinogenicity of Electromagnetic Fields," October 1990.

Compiled 2/92 by Joel Ray, co-author, The Electric Wilderness (San Francisco Press, 1986)

JR 2/92

Extremely low frequency (ELF): 1-100 Hz
(power lines, electric transport systems, submarine communications, bone stimulation, appliances, electric blankets, motors, video terminals, electrically heated waterbeds, theft detection systems, etc.)

date	study results	type	investigators
1979	childhood cancer increase	epid.	Wertheimer, Leeper
1980	mortality in 3 generations	animal	Marino, et al.
1981	suicide increase	epid.	Perry, et al.
1981	pineal gland effects	animal	Wilson, et al.
1982	leukemia increase	epid.	Milham
1982	childhood cancer increase	epid.	Tomenius, Hellstrom, Enander
1982	embryological defects	animal	Delgado, et al.
1982	leukemia increase	epid.	Wright, Peters, Mack
1982	cancer increase	epid.	Wertheimer, Leeper
1983	leukemia increase	epid.	Coleman, Bell, Skeet
1983	leukemia increase	epid.	MacDowall
1983	suppression of T-lymphocyte cytotoxicity	in vitro	Lyle, et al.
1983	cancer increase	epid.	Howe, Lindsay
1983	eye cancer increase	epid.	Swerdlow
1984	DNA synthesis effects	in vitro	Liboff, et al.
1984	tumor cell proliferation	in vitro	Winters, J. Phillips
1984	weight gain, testosterone increase	animal	Lotz, Saxton
1984	eye cancer in 2d generation	epid.	Hicks, et al.
1985	impaired timing behavior (effect on neurotransmitters)	animal	Thomas, Schrot, Liboff
1985	nervous system tumors in 2d generation	epid.	Spitz, Johnson

1985	brain tumor increase	epid.	Lin, et al.
1985	leukemia increase	epid.	Gilman, Ames, McCawley
1985	effect on neurotransmitters	animal	Seegal, Dowman, Wolpaw
1985	effect on RNA transcription	in vitro	Henderson, Goodman
1985	leukemia increase	epid.	Calle, Savitz
1986	fetal loss	epid.	Wertheimer, Leeper
1986	carcinoma cell proliferation	in vitro	Akamine, et al.
1986	growth effects, malformations in 3d generation	animal	R. Phillips, et al.
1986	tumor cell proliferation	in vitro	J. Phillips
1986	brain tumor increase	epid.	Coggon, et al.
1986	AML (Lou Gehrig's disease) increase	epid.	Deapen, Henderson
1986	leukemia increase	epid.	Flodin, et al.
1986	leukemia increase	epid.	Pearce, et al.
1986	leukemia increase	epid.	Stern
1987	childhood cancer increase	epid.	Savitz
1987	leukemia increase	epid.	Juutilainen, Pukkala, Laara
1987	allergic reactions	clinical	Choy, Munro, Smith
1987	delayed response in 2d generation	animal	Salzinger, et al.
1987	growth effects in tumor cells	in vitro	Byus, Pieper, Adey
1987	decreased body weight in 2d generation	animal	Rivas, Orozo, Delgado
1988	developmental effects in embryos in 5 separate, coordinated laboratories	animal	1-Leal 2-Martin 3-Monahan 4-Hansson Mild 5-Martucci
1988	brain tumor increase	epid.	Speers, et al.

1988	synergy with carcinogen	animal	Leung, et al.
1988	menstrual cycle effects	clinical	Wilson
1988	heart disease and depression increase	epid.	Perry, Pearl
1988	cell malignancies	in vitro	Parola, Kiesow
1988	leukemia increase	epid.	Linet, et al.
1989	leukemia increase	epid.	Pearce, et al.
1989	suppression of T-lymphocyte cytotoxicity	in vitro	Lyle, et al.
1989	reduction of nighttime melatonin levels	animals	Reiter, et al.
1989	cancer increase	epid.	Matanoski, Breysse
1989	brain tumor increase	epid.	Loomis, Savitz
1990	brain tumor increase	epid.	Preston-Martin
1990	brain and CNS tumor increase	epid.	Johnson and Spitz

Very low frequency (VLF): 300 Hz-30 Khz
(computer terminals, television sets, military communications, etc.)

date	study results	type	investigators
1982	noradrenaline release	in vitro	Dixey, Rein
1986	fetal abnormalities	epid.	Ericson, Kallen
1986	fetal abnormalities	animal	Tribukait, Cekan, Paulsson
1987	fetal abnormalities	animal	Walinder, et al.
1987	genetic effects	in vitro	Nordenson, Hansson Mild
1988	miscarriages and possible birth defects	epid.	Goldhaber, Polen, Hiatt
1988	fetal death, resorptions	animal	Frolen

Radiofrequency/microwave (RF/MW): 300 Khz-300 Ghz
(radio and TV transmission, microwave relay, diathermy, ham radio, citizens band radio, microwave ovens, air navigation, police radios and radar, military radar, microwave heaters and sealers, etc.)

date	study results	type	investigators
1979	enhancement of librium effect	animal	Thomas, Burch, Yeandle
1981	polycythemia increase	epid.	Friedman
1982	tumor growth from synergy of microwaves and benzopyrene	animal	Szmigielski, et al.
1982	leukemia increase	epid.	Morton
1982	cancer increase	epid.	Lester, Moore
1982	fetal loss and malformations	epid.	Kallen, Malmquist
1983	chromosome change	animal	Czerski, et al.
1983	cornea abnormalities	animal	Kues
1984	fetal malformations from synergy with teratogen	animal	Szmigielski, et al.
1984	DNA absorption of microwaves	in vitro	Swicord, Davis, Edwards
1984	malignancies and enlarged adrenal glands	animal	Guy, et al.
1984	cataracts	epid.	Hollows, Douglas
1984	heart disease increase	epid.	Hamburger, Logue, Silverman
1985	cancer increase	epid.	Szmigielski, et al.
1985	abnormal proteins in cerebrospinal fluid	clinical	Hansson, et al.
1985	leukemia increase	epid.	Milham
1986	reduced aggression	animal	Frey, Spector
1986	Alzheimers	animal	Koslov

1986	cell death in eye from synergy with glaucoma medicine	animal	Kues, et al.
1986	reproductive and developmental effects	animal	Tofani, et al.
1988	cancer increase	epid.	Milham
1989	synergy with cancer promoter	in vitro	Balcer-Kubiczek, Harrison

Sources: Microwave News; Transmission/Distribution Health and Safety Report; Journal of Bioelectricity; Bioelectromagnetics Society Newsletter; Electromagnetism and Life (Becker and Marino, 1982); Modern Bioelectricity (ed. Marino, 1988); Cross Currents (Becker, 1989)

JR 6/90

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAY 26 1992 OFFICE OF ENFORCEMENT

Mr. David R. Smiley
Deputy Program Manager
National Oceanic and Atmospheric Administration
Systems Program Office, JSPO (SPO121)
1325 East-West Highway
Silver Spring, Maryland 20910

Dear Mr. Smiley:

In accordance with Section 309 of the Clean Air Act and the National Environmental Policy Act (NEPA), the U.S. Environmental Protection Agency (EPA) has reviewed the "Draft Supplemental Environmental Assessment (SEA) of the Effects of Electromagnetic Radiation from the WSR-88D Radar" (formerly known as NEXRAD).

The WSR-88D radar system will consist of 153 weather radars which will obtain information about the location, intensity, and movement of severe weather phenomena throughout the United States and portions of the Caribbean. The SEA contains a review of the literature on the biological and health effects of radio frequency radiation (RFR) as well as electric and magnetic fields from power lines. Our general comments are highlighted below with detailed comment enclosed for your consideration.

The approach to the analysis of RF exposure levels from radar is one on which there is general agreement. Although there are specific differences between the analytical model used in calculating near and far field exposures, the agreement in calculated exposures is good, i.e., within a factor of 2 in the near field. The SEA model does not consider the discontinuity at the near field/far field plane. The report shows an obvious effort to update and discuss available information on the biological effects of radio frequency (RF) fields. However, the SEA should present a more up-to-date and balanced treatment of the research in this area.

We agree with the overall conclusion of the SEA, i.e., that the RF radiation exposures from the WSR-88D radar will not cause adverse health effects in the general population, because the measured far field exposures, with antenna rotation, at locations below the main beam, are less than 1 microwatt per square centimeter (uW/cm squared).

38-1 The rationale for exclusion or inclusion of studies should be stated, particularly where more recent data exists. Recent reviews are not cited, including those of EPA. Although these studies have flaws, it is more the lack of analytical studies that is the problem facing any RF risk assessment.

Based on our review of the SEA and in accordance with our national environmental assessment (EA) and environmental impact statement (EIS) rating criteria, we have classified this SEA as category EC-2, Environmental Concerns - Insufficient Information (See enclosed "Summary of Rating Definitions and Follow-Up Action"). The EPA review has identified potential environmental impacts requiring additional information to be included in the SEA.

Thank you for the opportunity to review the SEA. Should you have any questions regarding the detailed comments in the enclosure, please contact Dr. Carl Blackman at (919-629-2543) or Dr. Joe Elder (919-629-2542). You may also reach me at (202-260-5053) or have your staff contact Susan Hamilton, of my staff, at (202-260-5052).

Sincerely,

Richard E. Sanderson
Director
Office of Federal Activities

Enclosure

Environmental Impact of the Action

LO—Lack of Objections
The EPA review has not identified any potential environmental impacts requiring substantive changes to the proposal. The review may have disclosed opportunities for application of mitigation measures that could be accomplished with no more than minor changes to the proposal.

EC—Environmental Concerns
The EPA review has identified environmental impacts that should be avoided in order to fully protect the environment. Corrective measures may require changes to the preferred alternative or application of mitigation measures that can reduce the environmental impact. EPA would like to work with the lead agency to reduce these impacts.

EO—Environmental Objections
The EPA review has identified significant environmental impacts that must be avoided in order to provide adequate protection for the environment. Corrective measures may require substantial changes to the preferred alternative or consideration of some other project alternative (including the no action alternative or a new alternative). EPA intends to work with the lead agency to reduce these impacts.

EU—Environmentally Unsatisfactory
The EPA review has identified adverse environmental impacts that are of sufficient magnitude that they are unsatisfactory from the standpoint of environmental quality, public health or welfare. EPA intends to work with the lead agency to reduce these impacts. If the potential unsatisfactory impacts are not corrected at the final EIS stage, this proposal will be recommended for referral to the Council on Environmental Quality (CEQ).

Adequacy of the Impact Statement

Category 1—Adequate
EPA believes the draft EIS adequately sets forth the environmental impact(s) of the preferred alternative and those of the alternatives reasonably available to the project or action. No further analysis or data collection is necessary, but the reviewer may suggest the addition of clarifying language or information.

Category 2—Insufficient Information

The draft EIS does not contain sufficient information for EPA to fully assess environmental impacts that should be avoided in order to fully protect the environment, or the EPA reviewer has identified new reasonably available alternatives that are within the spectrum of alternatives analyzed in the draft EIS, which could reduce the environmental impacts of the action. The identified additional information, data, analyses, or discussion should be included in the final EIS.

Category 3—Inadequate
EPA does not believe that the draft EIS adequately assesses potentially significant environmental impacts of the action, or the EPA reviewer has identified new, reasonably available alternatives that are outside of the spectrum of alternatives analyzed in the draft EIS, which should be analyzed in order to reduce the potentially significant environmental impacts. EPA believes that the identified additional information, data, analyses, or discussions are of such a magnitude that they should have full public review at a draft stage. EPA does not believe that the draft EIS is adequate for the purposes of the NEPA and/or Section 309 review, and thus should be formally revised and made available for public comment in a supplemental or revised draft EIS. On the basis of the potential significant impacts involved, this proposal could be a candidate for referral to the CEQ.

*From: EPA Manual 1640, "Policy and Procedures for the Review of Federal Actions Impacting the Environment."

Detailed Comments on the Draft Supplemental Environmental Assessment of WSR-88D (formerly known as NEXRAD)

Appendix B offers several areas that need to be changed for a more balanced presentation. We agree with the final conclusions that, given the current data on biological effects of pulsed RFR, there is no known health hazard from WSR-88D Radar (formerly known as NEXRAD). Comments follow below with reference to the page they refer to.

Nonthermal Interactions and SARs (B-13 to B-14)

Page B-14 - Top of page. Other researchers who have failed to confirm the calcium release finding have been shown in a series of subsequent reports by Blackman et al. to have either failed to replicate the conditions of exposure or to have not reported vital exposure information so that definitive conclusions could not be made. Further, in an abstract, Guy and colleagues (Lee QP, Guy AW, Lai H, Horita A (1987): The effects of modulated radiofrequency radiation on calcium efflux from chick brains in vitro. Abstract D1, Ninth Annual Meeting of the Bioelectromagnetics Society, Portland, OR, June 21-25, 1987. Available from BEMS, P.O. Box 3729, Gaithersburg, MD 20878) have reported another duplication of the calcium-ion release effect using modulated 147-MHz fields. The statement that later studies have failed to confirm is misleading and incorrect. The essential details of the finding have been sufficiently replicated to establish it as a phenomenon. It is not known what the result means physiologically; it must be studied further. However, EPA is modifying its EMF Cancer Assessment Document because of the non-classical dose-dependence shown by this phenomenon. See detailed description below.

38-2

Calcium Efflux (B-55 to B-61)

Pages B-57 & B-58 - The review misses a series of 17 papers describing various aspects of exposure or extending the results of the initial calcium efflux studies, including conditions that minimize the impact of the negative effect experiments that are noted. Studies of both RFR and ELF fields are included because of text on calcium efflux found on page 42, for RFR, and pages 62-63, for ELF.

38-3

Blackman CF, Benane SG, Elder JA, House DE, Lampe JA, Faulk JM (1980a): Induction of calcium-ion efflux from brain tissue by radiofrequency radiation; effect of sample number and modulation frequency on the power-density window. Bioelectromagnetics 1:35-43.

Joines WT, Blackman CF, Hollis MA (1980): Broadening of the RF power-density window for calcium-ion efflux from brain tissue. IEEE Trans. Biomed. Eng. 28:568-573.

Blackman CF, Benane SG, Joines WT, Hollis MA, House DE (1980b):

Calcium-ion efflux from brain tissue: Power-density vs. internal field-intensity dependencies at 50-MHz RF radiation. Bioelectromagnetics 1:277-283.

Joines WT, Blackman CF (1980): Power density, field intensity, and carrier frequency determinants of RF-energy-induced calcium-ion efflux from brain tissue. Bioelectromagnetics 1:271-275.

Blackman CF, Joines WT, Elder JA (1981): Calcium-ion efflux induction in brain tissue by radiofrequency radiation. Biological Effects of Nonionizing Radiation, Illinger KH ed. ACS Symposium Series, 157:299-314.

Joines WT, Blackman CF (1981): Equalizing the electric field intensity within chick brain immersed in buffer solution at different carrier frequencies. Bioelectromagnetics 2:411-413.

Blackman CF, Benane SG, Kinney LS, House DE, Joines WT (1982): Effects of ELF fields on calcium-ion efflux from brain tissue, in vitro. Radiat. Res. 92:510-520.

Lin-Liu S, Adey WR (1982): Low frequency amplitude modulated microwave fields change calcium efflux rates from synaptosomes. Bioelectromagnetics 3:309-322.

38-3 (Cont.)

Dutta SK, Subramonian A, Ghosh B, Parshad R. (1984): Microwave radiation-induced calcium-ion efflux from human neuroblastoma cells in culture. Bioelectromagnetics 5:71-78.

Joines WT, Blackman CF, Spiegel RJ (1986): Specific absorption rate in electrically coupled biological samples between metal plates. Bioelectromagnetics 7:163-176.

Wood AW, Joines WT, Blackman CF (1987): Characteristics of transverse electric and magnetic field Transmission Cells at extremely low frequencies. Bioelectromagnetics 8(4):407-413.

Spiegel RJ, Joines WT, Blackman CF, Wood AW (1987): A method for calculating electric and magnetic fields in TEM calls at ELF. IEEE Trans. Electromagnetic Compatibility EMC-29:266-272.

Blackman CF, Benane SG, House DE, Joines WT, Spiegel RJ (1988): Effect of ambient levels of power-line-frequency electric fields on a developing vertebrate. Bioelectromagnetics 9(2):129-140.

Blackman CF, Benane SG, Elliott DJ, House DE, Pollock MM (1988):

Influence of electromagnetic fields on the efflux of calcium-ions from brain tissue in vitro: a three-model analysis consistent with the frequency response up to 510 Hz. Bioelectromagnetics 9:215-227.

Blackman CF, Kinney LS, House DE, Joines WT (1989): Multiple power density windows and their possible origin. Bioelectromagnetics 10(2): 115-128.

Dutta SK, Ghosh B. Blackman CF (1989): Radiofrequency radiation-induced calcium-ion efflux enhancement from human and other neuroblastoma cells in culture. Bioelectromagnetics 10(2):197-202.

38-3 (Cont.)

Blackman Cf, Benane SG, House DE, Elliott DJ (1990): Importance of alignment between local C magnetic field and an oscillating magnetic field in responses of brain tissue in vitro and in vivo. Bioelectromagnetics 1(2):159-167.

Schwartz J-L, House DE, Mealing GAR (1990): Exposure of frog hearts to CW or amplitude-modulated VHF fields: selective efflux of calcium-ions at 16 Hz. Bioelectromagnetics 11:349-358.

38-4 Page B-57 - Last line in second to last paragraph. This line does not appear to refer to the paper being cited.

Beginning of last paragraph. The proper citation is 1982, see list above.

38-5 Page B-58 - Last sentence of the second complete paragraph. This statement is highly implausible. The exposure conditions are very similar to those found in simple NMR spectrometers. The AC and DC fields interact in a resonant manner; there is no "swamping out."

Mutagenesis, Cytogenetic Effects, and Carcinogenesis (B-36 to B-44)

38-6 Page B-37 - The reviewers failed to mention Bevin et al. who reported microwave induced mutagenesis. The citation is Blevins et al. (1980): Radiation Research 82:511-517.

38-7 Page B-40 and B-41 - There should be a better statement to support the claim that the animals used by Szmigielski are stressed, and to define the amount of stress because most animals used in experiments are stressed to some extent.

38-8 Taking the reviewers' assumption that the animals are heat stressed, and this was the cause of the increases in breast and skin cancers under RFR exposure, should not this be a bigger issue than work stoppage and other endpoints that are used in the

Institute of Electronic and Electrical Engineers (IEEE) SCC 28 guideline? I recommend that the thermoregulation section cross reference these results and indicate whether this kind of effect has been looked for in chronic exposure situations. Further, see the Guy study, below.

38-8 (Cont.)

Page B-43 - With reference to tumors of the endocrine system (Guy study), it seems that an analysis should be done comparing additional heat load to endocrine stress. There may be a connection that would allow the reviewers to break out of the chemical toxicology reference frame they espouse.

38-9

Page B-44 - The review of the Balcer-Kubiczek & Harrison (1991) paper is uninformed. The data cited shows a strong synergism between RFR and TPA, to wit: 0 RFR and 0 TPA - 6.8 foci normalized to 1500 dishes; 0 RFR and TPA - 14.0; RFR and 0 TPA - 8.5; and RFR and TPA - 102.3. Furthermore, the foci are quite easily scored, so the "blind" requirement is unnecessary, and frequently there are more than one foci on a dish and many dishes with no foci. The data that are missing in this review are the values for positive control treatments; how many foci compared to historical data? The zero treatment controls indicate that the system is very stable. This paper needs a more objective treatment. The statements on pages 37 and 39 need to be modified.

38-10

Metabolism and Thermoregulation (B-71 to B-74)

It is uninformed to think that only when the RFR-generated heat is comparable to the body's metabolic rate, there might be a health problem. There are reports that conclude that humans, under conditions of reduced perspiration-based heat loss, may be much more susceptible to RFR-induced heat input because of the reduced cooling due to decreased surface-area-to-mass ratio of humans compared to rodents. These reports, pioneered by Christopher Gordon, should be acknowledged and addressed in any review of heat stress. Further, the review should evaluate the health problems of RFR exposure to thermal-regulation-compromised individuals, particularly in view of the analysis of Szmigielski's work (B-40 to B-41), which has been interpreted as heat-stressed-induced increased incidence of cancer of the breast and of the skin.

38-11

In view of these comments, changes should be made on pages ii-iii, 21, and 44, to introduce these alternative views of this issue.

Cellular and Subcellular Effects (B-99 to B-102)

Page B-100 - A research group left out of this section, Grundler, Kellmann and colleagues, needs to be included because they have published a series of extensive tests, with different antennas

38-12

and endpoints measured to document frequency specific effects above 30 GHz. In fact, Grundler has presented some of Gandhi's data as entirely consistent with a zero crossing point within a given frequency range found by Grundler. Gandhi did not object
38-12 (Cont.) to this interpretation and agrees that more studies need to be performed before adequate conclusions can be drawn (personal communication).

Corrections to text are also needed on pages 46 and 52 to include this information.

Ocular Effects (B-26 to B-28)

Page B-27 - In view of potential health consequences, it is reasonable to compare the worse case exposure scenario (see A-18 to A-19) to reports of ocular damage in monkeys exposed to pulsed 2.45 GHz radiation. In reports subsequent to 1985, Kues and colleagues have shown a potentiating effect of a drug used to treat glaucoma on RFR-induced ocular injury. The reviewers
38-13 should compare those exposure levels for peak power densities with the worst case example for NexRad, namely, 2200 mW/cm2. The margin of error present in the NexRad exposure situation should be assessed.

This concern needs to be addressed on pages iii and 35.

Nervous System (B-48 to B-).

Blood-Brain-Barrier Effects (B-49 to B-50) - The reviewers have failed to acknowledge the potential importance of the pulsed RFR compared to CW, and of the pulse repetition rate, in causing changes in the endpoints examined by Frey and by Oscar and Hawkins. These values should be stated and compared with exposures used by later groups, for example, Merritt et al., Ward
38-14 and Ali, Rapoport et al., Ward et al., Williams et al. and Neilly and Lin. It appears that true replications were not done in most, if not all, of the cases. In that case, the issue still remains open and particularly relevant for the pulsed fields used in NexRad.

Other pages that need to address this viewpoint are iii and 40.

Histological and Histochemical Studies In Vivo - The work of
38-15 Henry Lai et al. is treated fairly on pages B-54 to B-55, but is reported in a bias manner on page 41. There are no "apparent inconsistencies" in his data.

EEG- and Evoked-Response Changes (B-55) - Work that should be
38-16 cited here includes that of Bawin and colleagues, using cats, and of Takashima and Schwan, using rabbits; changes in EEg patterns were reported during or subsequent to exposure.

38-16 (Cont.) Changes should also be made on page 49 to incorporate these findings.

38-17 Laboratory Animal Studies (page 61) - Blackman et al. (1988) exposed incubating eggs to electric fields at 10 V/m (50 or 60 Hz) and showed a change in response of the brain tissue of the hatched chickens when tested in the calcium-efflux test. The report is in the bibliography, but the results are not stated. They should be discussed here.

The following SEA statements are incorrect or misleading:

38-18 p.34 In the Moscow Embassy exposure situation, the frequencies, power densities and signal characteristics were so different from the ones generated by NexRad, that a comparison is inappropriate.

38-19 p.38 The report by Skidmore and Baum appears to show no differences between exposed and controlled. It is strange that other reports showing statistically significant detrimental effects are discounted for various reasons, but this report is described as showing "beneficial" value from exposure in spite of the "sample size...(being)...too small to ascribe validity to that finding."

38-20 p.39 The Balcer-Kubiczek & Harrison data is significant. The reason for having small numbers of foci per dish, is to facilitate counting them. Secondly, the need for double blind experiments has been exaggerated. This is a standard assay that the original authors were familiar with. The important issue is whether the results can be independently replicated.

38-21 p.68 In second paragraph. If this critique of Milham's work has not been peer-reviewed, and if Milham has not had an opportunity to reply, then it is inappropriate for this publication. Rather similar to omitting reports presented as abstracts.

38-22 p.68 In third paragraph. What are the credentials of the reviewers to claim that "each study was open to serious methodological criticism"? If this is in a peer-review publication the evidence should be cited.

The following assessments of the SEA are divided into two parts. In the first part, a comparison is made between two published guidelines and the RFR levels in areas accessible to the public. The second part comments on the adequacy of the literature review.

I. Comparison of RFR levels to published guidelines

There are no Federal rules to control exposure of the public to RFR, but two American organizations have published voluntary guidelines that can be used to judge the safety of the WSR-88D radar emissions. In 1986, the National Council on Radiation Protection and Measurements (NCRP) published recommendations to limit public exposure at 2.7-3.0 GHz to 1 mw/cm2. The "IEEE Standard for Safety Electromagnetic Fields 3 kHz to 300 GHz" published in 1991 recommends that exposure to the public not exceed 1.8-2.0 mW/cm2 at 2.7-3.0 GHz. The SEA provides information that shows that RF radiation levels in areas accessible to the public will be significantly below the recommended values for both limits. In fact, the average RF
38-23 radiation level (0.6 mW/cm2) at the surface of the radome is below both limits (p. 53). Exposure levels more distant from the radome in areas accessible to the public will be significantly below 0.6 mw/cm2. The SEA states that "During normal operation, for most units...the highest average RFR power density at heights of 6 ft or less from the ground will not exceed 0.005 mW/cm2" (p. 53). This value is 200 times lower than the more restrictive limit recommended by the NCRP. Based on this information, no significant public health effects should result from the operation of the WSR-88D radar as described in the draft Supplemental Environmental Assessment. This conclusion is supported by the additional observation that the RFR levels in areas accessible to the public are 100 times lower than the lowest limit proposed by the EPA in 1986 (Federal Register, Vol. 51, No. 146, pp. 27318-27339, July 30, 1986).

Both the NCRP and IEEE guidelines are based on the sum of all RF emissions and not on a single, specific source such as the WSR-88D radar. This is based on the assumption that the sum of all
38-24 RF emissions in areas accessible to the public does not exceed the NCRP and IEEE limits. The final EA should show that the RFR from the WSR-88D radar and from all other RF sources does not result in levels that sum to values equal or greater than the NCRP limit in areas accessible to the public.

EPA is in partial agreement with the overall conclusion stated in Section 3.12 (p.52). We agree that it is logical to conclude that the RFR from WSR-88D units will be at levels that will not generate adverse health effects in the general population, but we do not agree that this conclusion is based on a "rigorous analysis of the scientific literature" presented in the SEA. We would favor a more comprehensive and objective analysis.

II. Comments on the Literature Review

The SEA relied in large measure on the CRITIQUE OF THE LITERATURE ON BIOEFFECTS OF RADIOFREQUENCY RADIATION: A COMPREHENSIVE REVIEW PERTINENT TO AIR FORCE OPERATIONS (Heynick 1987) that has not been subjected to peer review. The use of the CRITIQUE contributed to errors of omission and commission that should be corrected before the SEA can be accepted as a comprehensive and
38-25 objective review of the literature.

One problem in this approach is illuminated by a statement on p. 38. It states that the results of a study with mice "appeared to indicate exposure to the EMP was beneficial". This conclusion is based on "a nonsignificant difference" and the "sample sizes were too small to ascribe any validity to that finding" (same paragraph, p. 38). There is a marked contrast between the comment, particularly, on those reporting positive findings, e.g., Milham 1983, 1988 (p. 34 and B021) and Byus et al. 1987 (p. 64). Consider the following overstated negative critiques: (1) "a few controversial epidemiologic studies have reported a statistical association of cancer promotion with exposure to power line fields" (p.37). While the papers may be
38-26 controversial, there are more than a few of them as stated correctly on p. 70 of the SEA; "In spite of all the study design, and interpretation problems mentioned above, in this body of epidemiologic research half the studies report positive findings, and they cannot be dismissed."

(2) The criticism of the results by Balcer-Kubiczek and Harrision (1991) (p. 39) reads: "Little credence can be given to the latter results because of the small numbers of foci per dish and
38-27 the fact that the counting was apparently not done without prior knowledge of the treatment of each dish". While "blind" analysis of samples may be preferable, it is not improper to do otherwise. This criticism in the SEA is questionable.

(3) At least three labs have observed in calcium efflux or related effects and have published in the peer reviewed literature (Bawin et al., Blackman et al. and Dutta et al.). EPA
38-28 assumes that this conclusion is based on preliminary results from the Universities of Rochester and Washington that have not been published in the peer reviewed literature. If the authors cannot provide a reference in the peer reviewed literature to support their conclusion, the conclusion should be revised or deleted.

(4) The concluding comment on the study by Byus et al. (1987) is "It also has been seriously challenged by oncologists" (p. 64). The conclusion is unsubstantiated; no references are cited to support the author's opinion. As stated above, if the authors cannot provide a reference in the peer reviewed literature to support their conclusion, the conclusion should be revised or deleted.

38-30 Contrary to the statement that "...most of the papers selected underwent peer review before publication" (p. B-1), the citations for a number of Air Force reports are not in the peer-reviewed literature, i.e, Bollinger (1971), Frazer (1976) and Krupp (1977).

38-31 The Executive Summary should contain a statement describing the worst case exposure situation for publicly accessible areas. The statement on p. 53 seems appropriate: "During normal operation, for most units, the RFR beam will be elevated at lease 0.5, and the highest average RFR power density at heights of 6 ft. or less from the ground will not exceed 0.005 mW/cm2." This statement should replace the following sentence on p. ii: "At 1000 ft. from the radar at ground level, the average power density will decrease to between 0.0004 mW/cm2 and 0.0002 mW/cm2, depending on tower height." The letter statement describes the exposure at an arbitrary distance and is not helpful; it does not address the worst case exposure scenario.

38-32 p. iii: "Because effects from exposure to RFR below established guidelines have not been demonstrated, accumulation of effects cannot occur." This sentence is technically and logically incorrect. Effects, not necessarily adverse, have been reported below guideline levels.

38-33 p. iii: The statement that "Underground power lines will generate negligible fields" is not correct for magnetic fields.

38-34 p. iii: The statement that the "radar and its power lines will not be near businesses or residences, no adverse bioeffects are expected" is not helpful because "near" is a relative term. This sentence should be replaced with information on the exposure levels of the closest businesses and residences.

38-35 p. 25: EPA does not agree with the conclusion "As will be evident later in this document, much experimental evidence exists to affirm that chronic exposure to RFR at low levels is not harmful to humans." There are few reports of chronic exposure studies, especially at low levels, and it is certain that these do not constitute "much experimental evidence."

38-36 Section C. HUMAN HEALTH EFFECTS OF RFR should be changed to BIOLOGICAL EFFECTS OF RFR or HEALTH AND BIOLOGICAL EFFECTS OF RFR because the text includes animal data.

38-37 The report should clearly explain that the guidelines developed by American organizations such as the American National Standards Institute and the National Council on Radiation Protection and Measurements are voluntary recommendations.

38-38 p. 37: EPA recommends that Section 2.5 RFR Shock and Burn be moved to the end of Section 2. It seems inappropriate to include

38-38 (Cont.) this subsection between text on auditory effects and mutagenesis.

38-39 The conclusion to Section 2.8.2 ("Most of the findings on histopathological and histochemical changes in the CNS induced by RFR were clearly thermally based, with the exception of the recent work on changes in ATP and CP levels in the rat brain"), is not supported by the literature cited in this section. Only three studies are mentioned other than the rat brain biochemistry experience. Of these three, two report negative effects and the results of the third study by Lai et al. (1988), cannot be ascribed to clearly thermal effects.

38-40 The discussion (p. 43) of malignancies in the University of Washington study belongs in Section 2.6.3 Cancer Induction and Promotion in Animals (p.38). Similarly, the discussion (p.50) of malignancies in the University of Washington study belongs in Section 3.5 (p.47).

38-41 p. 44: It is not true that "There are scientifically credible experimental data to show that the thermoregulatory systems of nonhuman primates can readily compensate for high RFR levels." The converse is true. High RFR levels kill animals and the cause of death is attributed to thermal stress. Lotz (1985) had to terminate exposure of rhesus monkeys to 225 MHz at 5 W/kg in less than two hours to prevent irreversible thermal stress leading to death. A deficiency in the SEA is the absence of a section summarizing the literature on lethality. The draft report, in general, does not adequately review and summarize the thermoregulatory effects of RFR. The treatment of the thermoregulatory effects of RFR is inadequate on two counts. One, it is inappropriate that the draft report does not include a single reference to C.J. Gordon, who has published extensively on the thermoregulatory effects of RFR. Two, the authors demonstrate a general lack of understanding of the current state of knowledge of the thermoregulatory effects of RFR as evidenced by the above statement quoted from p. 44 and other statements on pp. ii, iii and 21. It is known that high levels of RFR cause death in mammals at normal conditions of temperature and humidity. Moderate and low RFR levels can cause adverse thermal effects at extreme conditions of temperature and humidity. The statement on p. 21 that "absorption of RFR within a body as heat is not significant unless the rate at which heat is added is comparable to or higher than the body's metabolic rate" needs to be qualified by consideration of ambient environmental conditions. We do not support the conclusion on p. iii that levels as low as 0.5 mW/cm2 are the "so-called 'nonthermal' levels of RFR." As explained above, manipulation of environmental conditions could make this level thermally significant, especially under resonant frequency exposure conditions.

38-42 p. 52: The statement that WSR-88D RFR will not exceed the

38-42 (Cont.) proposed, but not implemented, EPA standard should be revised to read that the radar emissions will not exceed the lowest of three limits proposed by EPA in 1986.

38-43 p. 54, Section 4.4: Delete the phrase "Although some favored the five-fold reduction to (0.08 W/kg)." One could make the same statement for no reduction and for ten-fold reduction.

38-44 p. 55: The statement that "The physics of the interaction of E- and H-fields with conductive objects, including biological organisms, is well understood" does not recognize the current debate on the mechanism(s) of interaction of EMP with cells and tissues. This debate clearly underscores the lack of understanding on the interaction of E- and H-fields on biological organisms.

38-45 p. 57: The SEA should delete or cite a reference to support the conclusion that "as part of Marxist-Lenin doctrine, workers were entitled to protection from any and all workplace hazards, even if actions of these agents were psychosomatic."

38-46 p. 62: "Significant declines were found in concentrations of two neurotransmitters in cerebrospinal fluid, and one of these remained depressed in some animals for up to 21 days after exposure ceased. Some of these effects may have resulted from chronic stimulation of body hair by the 60-Hz 3-field." The SEA should cite a reference to support the second sentence or delete this conclusion.

38-47 p. B-4: Insert "for uncontrolled environments" at the end of line 13 to read "In the range from 300 MHz to 1.5 GHz, the new limits for uncontrolled environments have a safety factor of 50..."

38-48 p. B-4: "The thermal interaction of RFR with living tissue is important for interpretation of animal studies because the size of the animal determines the percentage of energy absorbed." The amount of absorbed energy is dependent on many factors including body size, frequency, power density, polarization, etc., as stated in the last sentence in this paragraph.

38-49 p. B-23: The text should be revised to incorporate the conclusions of the Science Advisory Board Subcommittee of 17 experts who reviewed the EPA draft report on cancer and EMF.

38-50 p. B-70: The first sentence in paragraph 6 contradicts information in the first full paragraph.

38-51 The SEA sections called "Summary of Current Knowledge About RFR Bioeffects" that are summaries of the 102 page review in Appendix B called "Human Health Effects of RFR" and a section called "Findings" that summarized the summary, are not organized

effectively. This format resulted in much redundancy that could be avoided by combining the summary and Appendix B. The organization for RFR would then be consistent with that for power line fields that have only two sections, namely, the "Summary of Current Knowledge About Power Line Frequency" and "Findings." Another problem with the current organization of the report is that sections in "Findings," the summary of the summary, are longer than sections in the "Summary"; see for example, Sections 3.6 and 3.8.

38-51 (Cont.)

The text would be improved by a discussion of the emission standard for the microwave oven that operates at a frequency (2450 MHz) near the WSR-88D radar band (2700-3000 MHz). A discussion of the microwave oven standard and a comparison of the microwave emissions from the oven, a very familiar household appliance, to the emissions from the weather radar in areas accessible to the public should aid the public's understanding of the RFR exposure levels. EPA recommends that Figure IV.1 be revised to show the frequency of the microwave oven.

38-52

Minor comments:

38-53 p. 20: change 2 nanowattscm/2 to 2 nanowatts/cm2

38-54 p. 53, line 3: 300 KNz to 300 kHz

38-55 p. 54, Section 4.5: Change National Council on Radiological Protection (NCRP) to National Council on Radiation Protection and Measurements (NCRP).

38-56 p. 54: The last line is repeated on the first line on p. 55.

38-57 p. 55: Delete "at ground level" in the sentence "power line H-fields are measured in units of milligauss (mG) at ground level."

38-58 The first sentence in Section 2.8.2 regarding in vitro studies is out of place; the studies summarized here are all in vivo studies.

38-59 p. 60: Edit the last sentence reading "A second breeding..."

38-60 p. 62: The last two lines are repeated on top of next page.

38-61 p. 64: Section 3.4: The first sentence needs editing.

38-62 p. 71, Section 5: Change 60-HZ to 60-Hz. Insert "and states" in the second sentence to read "Several private organizations and states have adopted guidelines..."

38-63 p. B-16: Edit the sentence on lines 10-13.

38-64 p. B-63: Insert "of" to read "Samples of peripheral blood..."

38-65 p. B-65: Change 4' min to 4.5' min (see line one of same paragraph).

38-66 p. B-73: Change "that" to "the" on line 7 to read "Thus, only the RFR and air source..."

The following comments pertain to the treatment of RF exposure levels, biological effects, the overall conclusion of environmental impact and epidemiology.

38-67 The SEA should present a more up-to-date and balanced treatment of the research in the area of biological effects in RF fields. For example, one significant and continuing RF research project involves exposure of the eyes of monkeys to non-thermal levels of pulsed microwave radiation (Henry Kues, Johns Hopkins Applied Physics Laboratory). The significance is that pulsed 2.45 G Hz radiation exposures at non-thermal levels, representing a specific absorption rate (SAR) as low as .13 W/kg, has produced damage to the eye. The only SEA reference to the work by Kues was a 1985 paper; more recent papers by Kues, i.e., 1989 and 1991, (reprints are available) were overlooked.

38-68 The discussion of the 1985 paper questions the adequacy of the exposure technique and the experiment methods used, and as a result lessens the reader's perception of the possible significance of the work. However, that paper was subjected to peer review and published in the reputable scientific journal Bioelectromagnetics and the later work confirmed and extended the earlier results to show damage at even lower non-thermal exposures.

38-69 With respect to epidemiology, there are inadequacies in the SEA. For example, positive studies are heavily criticized but negative are rarely criticized. The overall conclusion that the general population will not suffer adverse health effects can be reached by relying on comparative exposure assessments and accessibility to the source.

38-70 Readers would be aided by a revised format and framework i.e., cancer, non-cancer endpoints, children, adults, etc. Recent reviews are not cited, including those of EPA. The lack of epidemiologic research on RF radiation and chronic disease is not stated. This review leads one to conclude that all RF epidemiology studies are badly done and show negative results.

38-71 The section on EPA's regulatory activities should be revised. Where the SEA uses the term "standard" the wording should be changed to "guidelines."

The following are further page specific comments on the SEA:

38-72 Page iii - The statements, "Early studies reported effects on the blood-brain barrier...those earlier results probably were obtained erroneously." need justification and citation.

38-73 Page iii - The statement, "The magnetic field level...power lines rights of way in some states (150 to 200 mG)" is not health based but is status quo; the standards are irrelevant.

38-74 Page iii - In the second paragraph, "Other studies claim" should be changed to "Other studies found" to avoid bias.

38-75 Page iv - In the statement, "Similarly, the review found that no adverse effects will result..." "will" should be changed to "should."

38-76 Page 46 - Section 3.1 states "Given the problems...despite the previously stated limitations." This unnecessarily reflects bias. In the analysis that "studies with human volunteers produced no valid scientific evidence..." the studies are not scientifically invalid but yield no evidence. The relative lack of studies should be noted for balance.

38-77 Page 47 - Section 3.3 has methodological problems. All of the studies, with the exception of one, were cross-sectional and therefore did not examine incidence. It is also possible that there is bias in the case selection.

38-78 Page 57 - Section 3.1. should note the EPA review because it is more current.

38-79 Page 64 - Section 3.4 the first sentence is invalid. The section should note that Great Britain's exposure experience is different than the United States'. We suggest that problems with negative studies be incorporated for balance.

6/1

5119 Bridge Road
Elba, New York 14058
May 17, 1992

Andrew Anderson
NEXRAD Program Managers
Systems Program Office
Room 15146
1325 East West Highway
Silver Spring, Maryland 20910

Dear Mr. Anderson:

We are writing to you to express our concern in having a NEXRAD unit located in the Batavia/Elba area. Knowing about the studies regarding health issues, it disturbs us to think one of these units will be located in the midst of a populated area.

The "new" location (1 mile north of the airport) would be detrimental to even a larger portion of the population as it would be dangerously close to Elba Central School. There are already many signatures from the citizens of Elba opposed to the original site and if the new location continues to be discussed, hundreds more will be obtained. Our children (kindergarten-12) do not need these electromagnetic waves bombarding them. No one does!

39-1 The NEXRAD technology is already outdated (invented in 1976). What is worng with satellites? If the NWS feels it must use these old units to "save face" - put them up in the wilderness!

Sincerely,

George Morgan
Cindy Kushner

Letter #40

5119 Bridge Road
Elba, New York 14058
May 17, 1992

David Smiley
Deputy Program Manager
Joint System Program Office, NOAA
1325 East West Highway
Silver Spring, Maryland 20910

Dear Mr. Smiley:

We are writing to you to express our concern in having a NEXRAD unit located in the Batavia/Elba area. Knowing about the studies regarding health issues, it disturbs us to think one of these units will be located in the midst of a populated area.

The "new" location (1 mile north of the airport) would be detrimental to even a larger portion of the population as it would be dangerously close to Elba Central School. There are already many signatures from the citizens of Elba opposed to the original site and if the new location continues to be discussed, hundreds more will be obtained. Our children (kindergarten-12) do not need these electro-magnetic waves bombarding them. No one does!

40-1 The NEXRAD technology is already outdated (invented in 1976). What is worng with satellites? If the NWS feels it must use these old units to "save face" - put them up in the wilderness!

Sincerely,

Cindy Kushner

Cindy Kushner

Letter #42

BOB MILLER
Governor

STATE OF NEVADA

JUDY MATTEUCCI
Director

DEPARTMENT OF ADMINISTRATION

Capitol Complex
Carson City, Nevada 89710
Fax (702) 687-3983
(702) 687-4065

May 21, 1992

Mr. David R. Smiley (SPO1)
SEA Coordinator
National Oceanic and Atmospheric Administration
1325 East-West Highway, Room 13166
Silver Spring, Maryland 20910

Re: SAI NV # 92300155 Project: SEA, Effects of Electromagnetic Radiation from the WSR-88D Radar

Dear Mr. Smiley:

Attached are the comments from the Nevada State Communications Board concerning the above referenced project. These comments constitute the State Clearinghouse review of this proposal as per Executive Order 12372. Please address these comments or concerns in your final decision.

Sincerely,

Danna G. Sturm

Danna G. Sturm, Coordinator
State Clearinghouse/SPOC

DGS/gd
Enclosure

BOB MILLER
Governor

WAYNE R. TEGLIA
Chairman

STATE OF NEVADA

MARK BLOMSTROM
Coordinator of Communications

STATE COMMUNICATIONS BOARD
555 Wright Way
Carson City, Nevada 89711-0550
(702) 687-5125
Fax (702) 687-6798

MAY 12 1992

May 5, 1992

M E M O R A N D U M

TO: Danna G. Sturm, Coordinator
Nevada State Clearinghouse

FROM: Mark Blomstrom
Coordinator of Communications

RE: FEDERAL NEXRAD PROJECT
SAI # 89300069 AND 89300071

Thank you for forwarding a copy of the Draft Supplemental Environmental Assessment (EA) of the Effects of Electromagnetic Radiation from the WSR-88D Radar.

After review, the Board has only one reservation. The State operates numerous point-to-point microwave systems in the 1900-2200 MHz band. The specific effect of the proposed radar system on point-to-point communications systems --as opposed to broadcast systems-- is not specifically addressed. The State operates several communications sites in the vicinity of the proposed radar locations; the existing Virginia Peak state communications site is very close to the proposed Virginia Peak radar.

The specific question we have is: what sort of interference or increase in the noise level can we expect at 2200 MHZ? This answer may take the form of 42-1 a specification identifying noise or out-of-band emissions which would be seen within the band of interest, i.e. 1900-2200 MHz. We are interested in knowing how far down, in decibels, from the peak radiated power of 475 KW the noise level would be over the band of interest; or alternately, how much, in decibels, the noise floor over the band of interest is raised.

The proposed radar system project is viewed by the State Communications Board as being generally beneficial to the State. However, the Board is also obligated to maintain and protect existing communications systems. The above requested information will, hopefully, assure the non-interference of the proposed system with existing state communications.

The opportunity to comment is appreciated and we will look for the response from the FAA.

HMB:lm

L-72

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
HEALTH EFFECTS RESEARCH LABORATORY
RESEARCH TRIANGLE PARK, NC 27711

OFFICE OF
RESEARCH AND DEVELOPMENT

October 8, 1992

Mr. David R. Smiley
Deputy Program Manager
National Oceanic and Atmospheric Administration
Systems Program Office, JSPO (SPO121)
1325 East-West Highway
Silver Spring, MD 20910

Dear Mr. Smiley:

Enclosed are our comments on the DRAFT RESPONSES to the ENVIRONMENTAL PROTECTION AGENCY (EPA) COMMENTS ON THE DRAFT SUPPLEMENTAL ENVIRONMENTAL ASSESSMENT (SEA) OF THE EFFECTS OF ELECTROMAGNETIC RADIATION FROM THE WSR-88D RADAR. We believe our comments demonstrate our intent to provide information and comments useful to the preparation of an objective and comprehensive SEA. Once the latter is achieved, we will support a more favorable reclassification of the SEA by the EPA Office of Federal Activities.

In general, the authors of the SEA are advised to summarize published reports without including unsubstantiated criticisms, present a balanced view of controversies where such exist in the literature, and cite published interpretations of the literature by expert panels where appropriate.

We are available to discuss and, if necessary, clarify any of the enclosed comments. Our telephone numbers are given below.

Sincerely yours,

Joe A. Elder

Joe A. Elder, Ph.D.
(919/541-2542)

Carl F. Blackman

Carl F. Blackman, Ph.D.
(919/541-2543)

Enclosure

cc: R.E. Sanderson, Director, OFA
Ann Miller, OFA

COMMENTS
on
DRAFT RESPONSES
to the
ENVIRONMENTAL PROTECTION AGENCY (EPA) COMMENTS ON THE DRAFT SUPPLEMENTAL ENVIRONMENTAL ASSESSMENT (SEA) OF THE EFFECTS OF ELECTROMAGNETIC RADIATION FROM THE WSR-88D RADAR

Comments are numbered by the same format used in the DRAFT RESPONSES.

38-1: OK. The authors state that papers were selected for review based on relevance to the weather radar system, originality and quality of findings; preference was given to peer-reviewed articles published in respected scientific journals; and reports were added where they contribute substantially to treatment of the subject.

38-2: Revision of first paragraph on p. B-14 is good.

Our understanding of the draft response to 38-2 is that the above revision to the first paragraph on p. B-14 is the only change. The rest of the draft response seems to be written for information only. We have the following comments on this informational text.

Paragraphs 3 and 4 are inadequate responses to a serious issue, that is, the independent and exact replication of research protocols to test research reports. What should be cited is the published abstract of Q.H. Lee, and not unpresented data, that is, personal communication. Close scrutiny of the response presented by the authors identifies significant similarities with the report by Dutta et al. (1984), in which unmodulated higher intensity RFR caused increased calcium ion release from neuroblastoma cells in culture, and of the report by Blackman et al. (1991) showing the need to carefully monitor and control the temperature history of the sample before and during the exposure to obtain the reported results. The authors should evaluate the findings in light of the peer-reviewed publications mentioned above.

38-3: The authors propose to ignore the extensive literature on RFR- and ELF-induced calcium-ion-efflux changes because, presumably, the effect is not harmful to human health. However, other groups including EPA and EEPA, have recognized the potential for the mechanism responsible for the efflux changes to also influence biological responses associated with health issues. It is important to recognize that the first fundamental criterion for any unusual research result, which includes the reported calcium efflux changes, is that the results be replicated and extended in the laboratory where initially discovered. That research has been published by Bawin, Adey, and colleagues. The second fundamental criterion is that other research laboratories also demonstrate the phenomenon. A brief summary of the published literature listed on the initial EPA review of this SEA should be included in the SEA for the explicit acknowledgment that there are important reasons for believing that research uncertainties exist that temper any statements about absolute safety of RF radiation. The calcium efflux phenomenon is important because

the effects occur at low intensities and display a nontraditional dose-response that prevents the assignment of threshold intensity values below which no effects would be expected. As stated above, the interpretation of this literature is tempered by the conclusion that there is no evidence that these effects are harmful to human beings.

38-4: Correction made.

38-5: Deletion acceptable.

38-6: OK

38-7: The review of the papers by Szmigielski et al. ignores some critical points important to the interpretation of the data. Examples follow.

(1) On page 4, paragraph 2, of the draft response, it is noted that no rectal temperature increases were observed even at the highest intensity used, 15 mW/cm^2 (6-8 W/kg), and that biological changes could have "plausibly" occurred due to assumed radiation-induced but unidentified "hot spots" within the mice. (See Comment 38-8.) Thus, RFR exposure causes a unique type of heating, one in which the animals, and presumably humans, have not evolved to accommodate.

If the statement that "it is plausible that the heat from the RFR (also affecting the immune system) caused the increases in tumor incidence" cannot be supported by a peer reviewed reference, the statement should be deleted.

(2) On page 5, paragraph 2, the draft response appears to confine acceptable responses to those in which there is always a statistically significant difference between various intensities of exposure. An equally probable alternative view is that the intensities are too close together to separate changes and that the physiological responses of the animal due to exposure are altering the linear dose-response model apparently adopted by the authors. The last sentence of paragraph 2 should be deleted.

(3) On page 6, paragraph 1, the authors equate a generalized heat stress model with local thermal effects. This correspondence is not generally true as demonstrated by the microwave hearing phenomenon. Delete the entire paragraph. See comment 38-8.

On page 4, paragraph 2, of the draft response, the authors correctly quote a statement from the original paper by Szmigielski et al. that no increase in rectal temperature was seen at 2-3 W/kg and "that such SARs exceed the basal metabolic rate of the mice". This statement is incorrect because the basal metabolic rate of mice is much higher, about 9-10 W/kg (See RF Dosimetry Handbook, second edition, 1978, p. 47).

38-8: The issue raised by the authors in their evaluations of the world's literature should not be assigned to some other group for analysis. By raising the possibility of local heating being the cause of increases in cancer promotion processes, the authors owe to the readers of the SEA an

examination of the literature to determine the seriousness of the proposal, its potential flaws, and future research needs to resolve the uncertainties. These need to be explicitly discussed in the text, or the alternative is to delete from the text the discussion of local heating from RFR exposure causing cancer [see comment 38-7(1)].

38-9: Revision acceptable with the following modification. On page 7, paragraph 3 of revised text, the numbers for each of the categories should be added, as was done in paragraph 4.

38-10: On page 9, paragraph 2, of the draft response, the authors raise an issue about the difference between an effect seen as a function of a linear change versus an exponential change in an independent variable but without justifying why the reader should agree that a scale different from one used by the original authors should be used. The data should be reported without these unsubstantiated comments.

On page 9, paragraph 3, there is the mistaken implication that a point mutation is not chromosomal damage. Ionizing radiation and chemicals have been used for decades to produce chromosomal damage. There is nothing wrong with the X-ray agent selected by the researchers. Paragraph 3 should be deleted.

On page 9, paragraph 4, the authors speculate on the motives of the researchers in selection of numbers of dishes to test. The zero EMF and positive TPA treatment had approximately twice as many dishes as the other combinations, which were approximately equal (704, 800 & 887). This speculation by the reviewers is unfounded and if included would only provide support for the contention that the SEA was biased. The last sentence of the paragraph should be deleted.

On page 9, paragraph 5, the text suggests that ionizing radiation and chemicals cannot produce different types of damage to the chromosome. This assertion is not true, e.g., x-rays can break chemical bonds and chemicals can produce DNA adducts. Moreover, some types of DNA damage may interact with EMF and TPA in the promotion process and others may not. Thus, the suggestion that a "more standard" chemical initiator be used is not convincingly presented. Paragraph 5 should be deleted.

The revision to the sixth paragraph on p. 39 is acceptable.

38-11: In paragraph 1 of the draft response, the authors use an hypothesis to limit their evaluation of the potential impact of RFR exposure. Specifically, they assume that the RFR only causes an added thermal burden that is less than changes in metabolic rate produced by simple wakeful activities. There are two schools of thought about the potential impact of increased thermal load introduced by RFR, the Adair and the Gordon schools. Both should be explicitly mentioned (we do not agree that the Gordon references should be left absent from the SEA).

Gordon has proposed two significant ideas. The first involves extrapolation of animal data to human data. In his study of thermal effects of RF radiation in four different species, he found that the SAR that produced a 1 °C increase in core temperature decreased with increase in body mass. Extrapolation of the data to 70 kg, the mass of the average adult male human being, predicts that an SAR of about 0.1 W/kg will increase human body temperature by 1 °C (see C.J. Gordon. Effect of radiofrequency radiation exposure on thermoregulation. ISI Atlas of Science: Plants and Animals 1, 245-250, 1988). The accuracy of the prediction based on Gordon's data is not known because of uncertainties in our ability to extrapolate thermoregulatory effects in laboratory animals to those in human beings. The appropriate approach would be to state that the model offered by Gordon is an hypothesis that needs to be further investigated. The second Gordon proposal is that expression of dose rate in terms of body surface area rather than body mass would allow more reliable prediction of thermal effects across species (see C.J.Gordon. Normalizing the thermal effects of radiofrequency radiation: Body mass versus body surface area. Bioelectromagnetics 8, 111-118, 1987).

On page 10, paragraph 6 (part 1), the second line shows evidence of bias by selecting one curve out of four for special treatment. The authors offer no explanation for the selection of a linear scale of the independent variable. As stated above, the appropriate approach would be to state that the model offered by Gordon is an hypothesis that needs testing.

With reference to page 10, paragraph 7 (part 2), MRI experiments on sheep were assumed by some researchers to be relevant to responses of humans under similar exposures, and thus should be included in the SEA text. Further, MRI exposure data on human volunteers should be included in the SEA [see Schaefer, D.J. Safety aspects of magnetic resonance imaging. In: Principles, Methodology, and Applications of Biomedical Magnetic Resonance Imaging (F. Wehrli and D. Shaw, editors). Deerfield Beach, Florida Publishers, 1988, chapter 13; and Shellock, F.G. and Crues, J.V. 1987. Temperature, heart rate, and blood pressure changes associated with clinical MR imaging at 1.5 T. Radiology 163, 259-262].

If the authors do not delete paragraph 1 on page 6 in the draft response as recommended, a discussion should be included in this section on the relationship between general heat stress, localized heating, and increased cancer incidence as inferred from their discussion of the Szmigielski et al. paper. See comment 38-7(3). In comment 38-8, we recommended the alternative that all mention of a relation between heating and cancer incidence be deleted.

38-12: Addition acceptable.

38-13: On page 13, paragraphs 1-4, of the draft response, the authors display an unwillingness to accept any exposure-response relation other than a strict linear response. By claiming the results are ambiguous, the authors are biasing an objective reading of the results. There is no a priori reason that a linear response is expected when the experimental endpoints could be ameliorated by various physiological responses available in whole animals.

Further, there are accepted practices when using sub-human primates to retreat the animals because of their value and scarcity. Delete the last line of paragraph 1 (Thus, for each drug ... ambiguous.) through to the second to the last sentence of paragraph 4.

On page 13, paragraph 6 (At a duty cycle ...), in the last sentence add the phrase "of 4 hours" between "... durations" and "comparable ... ".

38-14: The draft response is inappropriate. It is very important to establish whether subsequent studies replicated the exposure conditions used by Frey et al. (1975) and by Oscar and Hawkins (1977). If the subsequent studies were not true replicates including all aspects of the exposure parameters, then the issue remains open. This point must be addressed.

38-15: Revision acceptable.

38-16: On page 15, paragraph 4, of the draft response, the calculation of induced currents of 0.082 mA/cm^2 due to a 500-V/m 10-MHz field demonstrates ample dosimetry, which was done by a highly respected biophysical group working in the EMF research area. The last two sentences of that paragraph should be deleted.

On page 15, paragraph 5, the authors need to justify their claim that data obtained with chronic exposures, without intracranial metal electrodes, do not support the conclusions of the authors and presumably peer reviewers for the journal. Further, the authors should not mix in the same paragraph comments about results obtained with metal electrodes in place during exposure with those results obtained when the electrodes were absent.

On page 16, paragraph 1, the last two sentences are not relevant and should be deleted.

38-17: Revision acceptable.

38-18: It is appropriate to provide relevant exposure information in studies invoked to demonstrate the lack of a health hazard from the weather radar. The relevant exposure parameters closest to weather radar exposures, especially power density and pulse characteristics, should be extracted from the NTIA report and included in this SEA. The maximal power density was only 18 $microwatts/cm^2$. As a comparison, it would be useful to list the ambient exposure intensities recorded in many US metropolitan areas by Tell in EPA reports (R.A. Tell and E.D. Mantiply. Population exposure to VHF and UHF broadcast radiation in the United States. Proceedings of the IEEE 68(1), 6, 1980).

38-19: The revised text is acceptable if it replaces the third and fourth sentences and not the second and third sentences as stated.

38-20: See comment 38-10 above.

38-21: Draft response not adequate. On pages 68 and B-20 to B-21, the authors should summarize the reports by Milham without inclusion of the critique from a report that has not been peer reviewed. At the end of the Section 3.4 Epidemiologic Studies and Section 3.1.1, Epidemiologic/ Occupational Studies, the authors should cite the conclusions of two expert panels that have reviewed the literature on cancer. These two panels are the EPA Science Advisory Board (Loehr et al., 1992) and the Advisory Group on Non-ionising Radiation to the UK National Radiological Protection Board (Stather, 1992). Copies are enclosed of the reports by Loehr et al. (R.C. Loehr, O.F. Nygaard, G.M. Matanoski, and D.V. Bates, "An SAB Report: Potential Carcinogenicity of Electric and Magnetic Fields", letter dated January 29, 1992 to W.K. Reilly, EPA Administrator) and Stather (J.W. Stather, Electromagnetic fields and the risk of cancer. Radiological Protection Bulletin, No. 131, pp. 8-14, 1992).

38-22: Revision accepted.

38-23: Addition accepted with the following revision. Replace the last phrase ("the emissions from the WSR-88D radar will not materially add to the emissions present in those areas from any other sources") by "it is improbable that ambient RFR levels when increased by the RFR generated by the WSR-88D radar will exceed the guidelines."

38-24: Comment covered by 38-23 above.

38-25: We accept your statement that the Heynick (1987) document was reviewed by RFR-bioeffects researchers at the U.S. Air Force School of Aerospace Medicine before it was released, but this type of intramural review of a contractor's report does not equate to peer review. Peer review is a critical and unbiased analysis by independent experts and therefore excludes those associated with the organization funding the project when extramural independent experts exist. Our position is unchanged. The use of the 1987 report contributed to errors of omission and commission that should be corrected before the SEA can be accepted as a comprehensive and objective review of the literature. Our comments are provided to assist the authors in preparing a balanced report. In general, the authors are advised to report out the findings in published reports in summary form, and where controversies exist, present both sides of the controversy. Published interpretations of the literature by expert panels should be included where appropriate.

Regarding the EMP study, see comment 38-19 above.

38-26: Change acceptable if the change modifies the last sentence of the first paragraph in Section 2.6 to read "On the other hand, a growing number of epidemiologic studies have reported a statistical association of cancer promotion with exposure to power line fields (60 Hz)."

In place of the proposed addition, add "Two expert panels, one in the US and one in the UK, have reviewed the literature on cancer and both concluded that currently available information is insufficient to conclude that the electric and magnetic fields are carcinogenic (Loehr et al., 1992; Stather, 1992)."

Copies of the two references (Loehr et al., 1992; Stather, 1992) are enclosed.

38-27: See comments for 38-10.

38-28: See comments for 38-2.

38-29: Deletion accepted.

38-30: OK

38-31: Revision accepted.

38-32: Deletion accepted.

38-33: Revision accepted.

38-34: Since the draft response states that "The siting of many WSR-88R units is undetermined at this time" the following sentence is inappropriate and should be deleted: "Thus, given also that, in the majority of cases, the WSR-88D radar and its power lines will not be near businesses or residences, no adverse bioeffects are expected" (last sentence, last full paragraph on p. iii).

In this same paragraph, the sentence "A review of these studies indicates no conclusive evidence that magnetic fields per se are associated with cancer" should cite two recent reviews: (1) EPA Science Advisory Board report dated January 22, 1992 (Loehr et al., 1992) and (2) the report of the Advisory Group on Non-ionising Radiation to the UK National Radiological Protection Board (J.W. Stather, Radiological Protection Bulletin, No. 131, pp. 8-14, 1992).

38-35: Deletion accepted.

38-36: Title to Section C is not appropriate to text and should be changed as recommended.

38-37: Addition accepted.

38-38: OK, but no section was found on cutaneous perception.

38-39: The revision of Section 3.4.2.2 should be modified by reducing the five pages of text describing the four studies by Saunders and colleagues to one page or less.

Revision to second through sixth paragraphs on p. 41 acceptable.

38-40: The second and third sentences of revised text should be changed to read: "The findings on immunology and hematology are summarized in Section 2.9. Regarding longevity and cancer, the authors concluded that "no defendable trends in altered longevity, cause of death, or spontaneous aging lesions and neoplasia can be identified in the rats exposed to this long-term low-level radiofrequency exposure" (Guy et al., 1985).

Delete last paragraph on p. 43 or move the paragraph to Section 2.6.3.

Move last two paragraphs in Section 3.8 on p. 50 to Section 3.5.

Correct spelling of "virtually" in first sentence of revised text.

38-41: Revisions acceptable except for inadequate response to C.J. Gordon's contributions to the literature on thermoregulation in laboratory animals exposed to RF radiation. A summary of his more important papers should be included in Section 3.6.1 Metabolism and Thermoregulation (pp. B-71 to B-74) (see comment 38-11 for Gordon references).

38-42: Revision accepted.

38-43: Revision accepted.

38-44: Revision accepted.

38-45: Revision accepted.

38-46: Revision accepted.

38-47: Correction made.

38-48: Revision accepted.

38-49: The authors missed the point. The text on p. B-23 (beginning with the fifth paragraph reading "The EPA issued..." through the paragraph ending on top of p. B-24) should be deleted and replaced by text describing the following. The EPA issued a draft review of the literature on cancer and exposure to electromagnetic fields (RFR as well as those from power lines) entitled EVALUATION OF THE POTENTIAL CARCINOGENICITY OF ELECTROMAGNETIC FIELDS. The report was reviewed by 17 experts on the EPA Science Advisory Board Subcommittee on Nonionizing Electric and Magnetic Fields. The panel concluded that "Currently available information is insufficient to conclude that the electric and magnetic fields are carcinogenic." Furthermore, in response to the question: Does the animal or biological effects information provide a basis for postulating that there is a human hazard from exposure to extremely low-frequency fields or either modulated or unmodulated radiofrequency radiation?, the SAB concluded that "Hypothetical constructs relating observed biological effects to possible health effects (specifically, increased cancer risk) have been delineated. However, there are at present insufficient data on many of the critical steps in the linkage to infer causality on the basis of animal or cellular data" (R.C. Loehr, O.F. Nygaard, G.M. Matanoski, and D.V. Bates, "An SAB Report: Potential Carcinogenicity of Electric and Magnetic Fields", letter dated January 29, 1992 to W.K. Reilly, EPA Administrator). A similar conclusion concerning RF radiation was reached recently by the Advisory Group on Non-ionising Radiation to the UK National Radiological Protection Board. They stated that "It can be concluded only that the evidence for a co-carcinogenic or tumour promoting effect of such relatively high frequency fields is not convincing" (J.W. Stather,

Electromagnetic fields and the risk of cancer. Radiological Protection Bulletin, No. 131, pp. 8-14, 1992).

38-50: Move the sentence "Overall, no significant differences were found between the RFR- and sham-exposed rats at periodic behavioral test sessions" to the appropriate section, namely Section 3.7.1.1 Rodents (Behavior).

38-51: OK

38-52: The draft response is good and should be inserted in an appropriate place in the SEA.
We recommend again that Figure IV.1 be revised to show the frequency of the microwave oven.

38-53: Correction made.

38-54: Correction made.

38-55: Correction made.

38-56: Deletion made.

38-57: Deletion made.

38-58: Deletion made.

38-59: Correction made.

38-60: Deletion made.

38-61: Delete the first paragraph in Section 3.4 Epidemiologic Studies (p. 64) and insert the first four sentences of the draft response, namely "There is a growing number of epidemiologic studies of power line field effects in humans. Approximately half of these report positive findings for an association between some indicator or estimate of the presence of electric and magnetic fields and the increased incidence of several types of cancer or other biological endpoints, including outcomes of pregnancy after use of electric blankets, infertility of exposed male workers, suicides, and measures of general health. The other half do not. Several of these studies are discussed below."

Delete the remaining part of the draft response.

In Section 4. Findings, specifically Section 4.3 Epidemiologic Studies, revise the text to incorporate the information in 38-49. In addition, add the appropriate conclusion of the Advisory Group on Non-ionising Radiation to the UK National Radiological Protection Board that reads "It cannot be concluded either that electromagnetic fields have no effect on the physiology of cells, even if the fields are weak, or that they produce effects that would, in other circumstances, be regarded as suggestive of potential carcinogenicity...The results of some whole animal and cellular studies suggest the possibility that

electromagnetic fields might act as co-carcinogens or tumour promoters but, taken overall, the data are inconclusive" (J.W. Stather, Electromagnetic fields and the risk of cancer. Radiological Protection Bulletin, No. 131, pp. 8-14, 1992).

38-62: Corrections made.

38-63: Revision should replace the first three, not two, sentences.

38-64: Correction made.

38-65: Correction made.

38-66: Correction made.

38-67: See comment 38-13.

38-68: See comment 38-13.

38-69: See comments 38-61 and 38-49 above.

38-70: 1) Recommendation to reorganize draft SEA withdrawn because of the need to complete the final SEA as quickly as possible.

2) The EPA comment that "Recent reviews are not cited, including those of EPA" refers to the recent EPA cancer report on EVALUATION OF THE POTENTIAL CARCINOGENICITY OF ELECTROMAGNETIC FIELDS (October 1990) and not to the 1984 report on the BIOLOGICAL EFFECTS OF RADIOFREQUENCY RADIATION as indicated in the draft response.

3) To be judged a comprehensive review, especially in regard to RF radiation, the SEA must include a statement that few epidemiologic studies on RF radiation and chronic disease have been done; the recent EPA cancer review mentioned above cites only nine in Table 3-39 (Lilienfeld et al., 1978; Robinette and Silverman, 1977; Robinette et al., 1980; Milham, 1985, 1988a,b; Environmental Epidemiology Program, State of Hawaii 1986; Hill, 1988; and Szmigielski et al., 1987).

4) To be judged an objective report, again in regard to RF radiation, the report cannot give the wrong impression that all RF epidemiologic studies are badly done and report negative effects. Two investigators, namely Milham and Szmigielski, have authored 4 of the 9 papers; these 4 papers describe positive effects such as leukemia. Both positive and negative effects studies suffer from the same limitations (exposure data, confounding factors, statistical power, etc.) These limitations prevent one from defining a cause-and-effect relation between exposure to RF radiation and cancer (Loehr et al., 1992, Stather, 1992).

5) The authors of the SEA will make a major error in judgment if they do not incorporate the conclusions of the EPA Science Advisory Board (Loehr et al., 1992) and the Advisory Group on Non-ionising Radiation to the UK NRPB

(Stather, 1992) (see comments 38-49 and 38-61 above). The interpretations of the literature by these two expert panels are essential to the conclusions drawn in the SEA. Both reports are available and copies are enclosed.

38-71: Correction accepted.

38-72: See comment 38-14 above.

38-73: Revision acceptable.

38-74: Correction made.

38-75: Revision acceptable.

38-76: Revision acceptable.

38-77: Revision of Section 3.3 acceptable if a reference(s) is added to support the last sentence concerning eye damage.

38-78: Revision not adequate; see comment 38-49 above.

38-79: See comment 38-61.
The addition to the last paragraph on p. 64 is acceptable.

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APPENDIX D

RESPONSES TO COMMENTS ON THE DRAFT SEA

This appendix provides responses to substantive comments on the Draft SEA received by the NEXRAD JSPO during the public and government review period. All modifications to the Draft SEA text needed to respond to the comments have been incorporated into the text of this Final SEA.

Response to Comments from Norman C. McLaughlin

1-1

The Lilienfeld (1978) study is one of many studies examined in reaching the conclusions of this document. Copies of that report and other relevant documents such as a report by Pollack (1979) and another by the National Telecommunications and Information Administration of the U.S. Department of Commerce (NTIA, 1981) are available to the public. See also the response to Comment 37-13.

Although the conditions of RFR exposure at the U.S. Embassy in Moscow differ from the RFR exposure that would result from the WSR-88D, the Lilienfeld study is informative because it reports on one of the few situations in which humans have been exposed to known doses of RFR for extended periods. The irradiation of the U.S. Embassy began in 1953 and ended in 1977.

Pollack (1979) described the embassy irradiation situation, indicating that the U.S. Embassy in Moscow had been subjected to RFR from 1953, the year after the United States moved its chancery to Chekovsky Street, until February 1977. The presence of RFR had been detected intermittently before 1962 during routine surveillance of the building. Continuous monitoring of the signals was instituted during that year.

In NTIA (1981), details regarding signal frequencies, characteristics, irradiation durations, and average power densities or equivalent field intensities at various locations within and on the roof of the chancery were given. The signals were in the frequency range from 0.5 to 10 GHz, which encompass those of the WSR-88D radar, and thus renders the Lilienfeld et al. (1978) study relevant.

It is noteworthy that the average power densities during various periods ranged from less than 1 $\mu W/cm^2$ to 15 $\mu W/cm^2$. In rooms with windows or doors in outside walls toward the RFR sources, typical levels were about 4 $\mu W/cm^2$ within 2 ft of a door or window and 2.5 $\mu W/cm^2$ elsewhere therein. The highest level cited was 24 $\mu W/cm^2$, which was found in one room during

a 2-hour period of unusual signal strength on 24 January 1976. Because his maximum level was found in only one part of the embassy, the exposure levels of most embassy personnel for most of the time were probably well below the then maximum permissible level (5 $\mu W/cm^2$) specified in the USSR standard for exposure of the general population.

Therefore, despite the limitations of the Lilienfeld et al. (1978) study noted by the authors themselves, it is most unlikely that any of the illnesses suffered by embassy personnel or their dependents were associated with the irradiation of the embassy.

Dr. Lehrman's clinical statements regarding the long latencies in the development of leukemia and solid tumors are correct with respect to well-established carcinogens, but his implied association of such long latencies with exposure to low levels of nonionizing radiation is unsubstantiated. It is well known that exposure to ionizing radiations such as ultraviolet light, X-rays, and the emissions from radioactive materials is cumulative, even at very low dose rates. Such radiations can induce cancer or cause other harmful effects at excessive cumulative total doses; as a result, film badges are used to ensure that total doses do not exceed specific maxima. By contrast, much experimental evidence exists for the existence of threshold RFR-exposure levels for various effects, an indication that chronic exposure to RFR below such thresholds is not cumulative.

A recent study by Balcer-Kubiczek and Harrison (1991) appears to show that RFR alone does not initiate cancer but is a copromoter, meaning that it stimulates the growth of a cancer that was already initiated by a carcinogen, but only in conjunction with a known promoter. As discussed on p. B-43+ of the SEA and in response to comment 38-10, the findings of that study are questionable for several reasons. For a discussion of this study, see the response to comment 38-10.

Regarding Dr. Lehrman's statement on other possible health hazards of RFR, the large body of research discussed in the SEA indicates otherwise.

1-2

The study of Polish military personnel is discussed by Dr. Szmigielski in *Modern Bioelectricity* (Marino, 1988, p. 906). Dr. Szmigielski noted that the report, in Polish, had received only limited distribution and will be published in the future (date unspecified) only in part. The information provided by Dr. Szmigielski in *Modern Bioelectricity* on that study was not peer-reviewed; therefore, independent analysis of its scientific merits is unavailable.

The prospective study by Dr. Szmigielski is mentioned in both *Modern Bioelectricity* and the book, *Electromagnetic Biointeraction* (Franceschetti et al., 1989), neither of which presents preliminary results. Only brief anecdotal accounts, such as those in *Microwave News*, appear to be available at the present time.

1-3

The former ANSI guidelines for RFR exposure have been replaced by the IEEE SCC 28 guidelines (see section V.C.4.2 of the Draft SEA).

The 1991 SCC-28 RFR-exposure guidelines (IEEE, 1991) were recently published (May 1992) by the IEEE. As stated by IEEE, it is untrue that only thermal effects were considered in setting those guidelines, or that the guidelines were established solely on the basis of thermal effects. Had credible scientific evidence existed that exposure of humans to SARs of 4 W/kg or

lower would be hazardous to their health, the 1991 guidelines would not have been based primarily on 4 W/kg with a safety factor of 10 for exposure in controlled (occupational) environments and a safety factor of 50 in uncontrolled environments (accessible to the public).

Regarding nonthermal effects, the results of some scientific studies, such as those with cell cultures or cellular constituents, indicate that nonthermal effects do indeed occur. Such studies are important in elucidating basic interaction mechanisms of electric fields, magnetic fields, or electromagnetic fields (RFR) with biological objects. Thus, it is untrue to claim that only thermal effects have been considered in the SEA. However, no credible scientific evidence indicates that such nonthermal findings have possible effects on the health of humans (or other mammals).

Many of the assertions in the book *Cross Currents* by Dr. R.O. Becker are highly speculative and in large measure not in accord with the findings of most of the scientific literature on nonionizing radiation. Specifically, on pages 194-197, Dr. Becker describes his views on the long-term study by Dr. A.W. Guy and coworkers at the University of Washington. To minimize possible uncontrolled extraneous factors that might obscure effects of the radiation itself, the rats used were delivered by cesarean section and were barrier-reared. In addition, both groups were maintained throughout the study under the same controlled-environmental and specific-pathogen-free conditions. Among Dr. Becker's criticisms of the study was the use of cesarean-derived rats, an opinion that apparently disregards the findings of several other RFR-bioeffects studies that were considered questionable because of lack of meaningful control over non-RFR factors. Dr. Becker's incorrect conclusion (p. 195) was that the study "was deliberately designed to sharply reduce the incidence of cancer and infectious diseases in the exposed animals."

Dr. Becker's remark (pages 196-7 of *Cross Currents*) that the data "are buried in the multivolume official Air Force report of the project..." implies that those researchers deliberately attempted to hide the results. In fact, before the study began, Guy and coworkers published an article entitled "Study of Effects of Long-Term Low-Level RF Exposure on Rats: A Plan", in the *Proceedings of the IEEE* (Vol. 68, No. 1, 1980). In that article the rationale and experimental design of the study were described. In addition, when the study was completed, the results were reported at various technical society meetings. The Air Force School of Aerospace Medicine published nine technical reports (cited in the SEA) and distributed them widely to RFR-bioeffects researchers. Copies of those reports were available to all persons requesting them.

Professor S.F. Cleary, in his letter to Mr. J. Dusen dated 27 March 1990, cited the chapter "Electromagnetic Fields and Neoplasms" by Szmigielski and Gil in *Electromagnetic Biointeraction* (Franceschetti et al., 1989). In that chapter, Szmigielski and Gil noted several papers, that on critical examination, do not support Dr. Cleary's views. The following are two examples:

Lester and Moore (1982) (cited on p. 85 of *Electromagnetic Biointeraction*) reported a significantly higher incidence of cancer mortality in U.S. counties with Air Force bases than in counties without such bases. The analysis of that paper by Polson and Merritt (1985), also cited by Szmigielski and Gil, clearly demonstrated that the data base used by Lester and Moore was incorrectly assembled. After the data base was correctly assembled, no significant differences in cancer incidence between the two sets of counties were found.

Szmigielski and Gil also mentioned the study by Prausnitz and Susskind (1962) (p. 82 of *Electromagnetic Biointeraction*). In that study, mice exposed to RFR at high intensity were reported to have had a higher incidence of leukosis than control mice. Prausnitz and Susskind had mistakenly described leukosis as "cancer of the white blood cells," thereby implying a link between RFR exposure and cancer incidence. Leukosis, however, is an elevation of circulating leukocytes (white blood cells), which in this study may have due to a known infection in the colony of mice or to other functional disturbances. In addition, Roberts and Michaelson (1983) reanalyzed the primary data of Prausnitz and Susskind (1962) and concluded that that study provided no evidence that chronic RFR exposure does or does not induce any form of cancer.

Although Szmigielski and Gil did cite papers that reported positive findings, they omitted several that reported negative findings. For example, they made no mention of Skidmore and Baum (1974), who found that continuous exposure of 20 females to "electromagnetic pulse (EMP)" (a form of RFR designed to simulate the RFR from a nuclear blast) did not lead to the development of mammary tumors, and that exposure to EMP of the AKR/J strain of mice, which are prone to spontaneous leukemia, did not promote leukemia; 21% of the exposed mice versus 46% of the control mice developed leukemia.

Although Dr. Cleary's chapter in *Cross Currents*, "Biological Effects of Radiofrequency Radiation: An Overview" contains important aspects about RFR-interaction mechanisms at the cellular level and the problems associated with extrapolation of results with animals to humans, it does little toward clarifying whether or not RFR can induce or promote cancer in humans.

Professor Birenbaum questions the generally accepted basis for setting U.S. guidelines for maximum human exposure to nonionizing radiation. The maximum permissible levels of exposure in the 1982 ANSI guidelines were derived from examination of the then current body of experimental literature; the guidelines included a safety factor of 10 below the RFR absorption levels experimentally found to cause significant effects in laboratory animals, and the 1991 SCC-28 revision thereof (IEEE, 1991) includes safety factors of 10 for occupational exposure and 50 for exposure of the general population, with averaging times of 30 or 6 minutes as appropriate. As noted in "Safety Factors" (pp. 28-29 of IEEE, 1991), the use of such safety factors is consonant with the existence of threshold levels for RFR bioeffects, and compensates for uncertainties in measurement techniques and instrumentation, as well as in variations of experimental conditions.

The Congressional Office of Technology Assessment (OTA) did issue a report in June of 1989. The report is entitled "Biological Effects of *Power Frequency* [emphasis added] Electric and Magnetic Fields." The relationship between power-frequency fields and the RFR from the WSR-88D system is discussed in Sections IV.C and V.D.1 of this document. It is true that the authors of the OTA report found the scientific literature on power-frequency bioeffects to be confusing in its conclusions and lacking what is needed for scientifically based evidence of harm to humans, and that they indicated that setting a safe limit at this time for power frequency electric- and magnetic-field exposures cannot be scientifically supported. The RFR bioeffects literature, however, is much more comprehensive. The IEEE C95.1-1991 standard shows that it does support scientifically based exposure limits.

1-4

Regarding the research by Szmigielski, see responses to Comments 1-2 and 1-3 above.

Regarding the University of Washington study, see the response to Comment 1-3 above. Note also that all nine reports of that study are cited on p. B-43 of the Final SEA.

In independently examining the scientific literature on RFR bioeffects, only papers by the researchers themselves were analyzed, rather than other accounts by authors of review articles or in books such as *Electromagnetic Biointeraction* or *Modern Bioelectricity*. Hence, such review publications have not been cited in the SEA. For similar reasons, accounts such as the chapter by Dr. Cleary would not be cited in Appendix B of the SEA. Likewise, review articles that do not cite peer-reviewed articles, such as the studies described in subcomments 4, 7.1, and 7.3 were not examined.

In response to Dr. Cleary's remarks about nonthermal safety standards and the basis of safety standards for RFR exposure, the existence of nonthermal biological effects resulting from RFR exposure is not questioned by the SEA. The likelihood that those effects would result from exposure to RFR from a WSR-88D and the significance with regard to human health of those effects is analyzed in the SEA, which determines that harmful effects are unlikely to result from WSR-88D RFR. See also the responses to Comment 1-3 above.

The quotation from Dr. Cleary concerning the supposed thermal basis of the ANSI (1982) standard is dated 1989. That standard was revised in 1991. The current standard is not based solely on the consideration of thermal effects. See also the response to Comment 1-3 above.

Concerning the bibliography prepared by Dr. Joel Ray, see the response to Comment 37-28 by Dr. Ray.

Concerning subcomment 7.2 about the studies by Drs. Balcer-Kubiczek and Harrison, see the response to Comment 38-10.

Concerning subcomment 7.4 about studies by Dr. Sam Koslov, Dr. Becker (p. 261 of *Cross Currents*) briefly discussed the preliminary results Dr. Koslov had described at a public meeting. Dr. Koslov cited one case of what he claimed to be Alzheimer's disease in an RFR-exposed chimpanzee, "out of frustration with the lack of funding for the whole area of electromagnetic fields," a very indefinite result. A search of abstracts of papers presented at various symposia by Dr. Koslov and his Johns Hopkins University colleagues who are performing ocular studies (Kues et al.) yielded no references to any prior or subsequent work on a link between RFR exposure and Alzheimer's disease. In addition, as far as is known no chimpanzees were ever used in those ocular studies.

Concerning subcomment 7.5 about studies by Drs. Salford and Person, *Microwave News* does not cite references to a published article on this study. Therefore, the applicability of that finding to the WSR-88D situation cannot be assessed.

Concerning subcomment 7.6 about studies by Dr. Leal, presentations at symposia such as that made by Dr. Leal are not peer-reviewed accounts, but usually contain only preliminary results. For that reason, that presentation is not considered in the SEA.

1-5

The quoted statement by Dr. Zaret is contrary to experimentally established thresholds for RFR bioeffects. Section 3.1.3 of Appendix B to the Draft SEA summarizes the research on ocular effects of RFR exposure and specifically the threshold for ocular effects, about 150

mW/cm^2. The power density of WSR-88D RFR would be more than two orders of magnitude below that threshold, and no ocular effects would result.

Dr. Becker mentioned (*Cross Currents*, pp. 191-192) a study of RFR cataractogenesis done at Northwestern University shortly after the end of World War II (no citation given), in which the eyes of animals were briefly exposed to high RFR levels; examination immediately after exposure showed no cataracts. According to Dr. Becker, Richardson et al. (1948) had remarked that if the researchers had waited 3 days after exposure, they would have seen cataracts. The paper by Richardson et al. (1948) made no mention of such a study. In any case, many subsequent studies at high RFR levels have been performed, rendering the Richardson et al. (1948) study primarily of historical interest.

A discussion of the Aurell and Tengroth (1973) occupational study was not included in the SEA because the numbers of individuals involved were too small to place any statistical credence in the findings.

Concerning the findings of R. Birge (1988) of Carnegie-Mellon University mentioned by Dr. Becker (*Cross Currents*, p. 192), no published reports appear to be available, presumably because the work was done as part of the classified Stealth-aircraft program, as noted by Dr. Becker. Dr. Becker's statement that the medical implications are far more serious than the production of cataracts is speculative and is not supported by the large body of literature on the biological effects of RFR.

In summary, no credible evidence exists that long-term exposure to the RFR from the WSR-88D radar will produce cataracts.

1-6

A principal purpose of the NEXRAD system is to detect clouds and rain. To achieve this goal the operating frequency band has been chosen as a compromise between two considerations; the frequency must be high enough to respond to clouds and rain, but low enough to provide penetration so that near-in weather features do not obscure observation of more distant events. As a result of this frequency choice, it follows that only a small fraction of the power in the near-field column and resulting pencil beam is scattered by local cloud cover. Specifically, ground-level power densities near the radar are not significantly affected by local weather conditions and will not exceed the power densities given in Section V.B.4 of the SEA.

1-7

The analysis is given in the response to Comment 1-6 above. The power density of the WSR-88D RFR will not be significantly affected by local weather conditions during searchlight-mode operation.

The term "radiation leaks in the near field" used by N. McLaughlin is unfamiliar. The WSR-88D incorporates a number of safety systems to prevent improper operation; it will not "leak" RFR.

1-8

The searchlight mode operation will be used only once every several years for calibration and adjustment of the WSR-88D or in response to a request from the FAA. Operation in searchlight mode will occur for 5-minute periods, limited to one period every 30 minutes. The

use of searchlight mode will last no more than 1 day. Technicians will be present during searchlight mode operation and will direct the beam away from people and inhabited structures. The main beam of the Buffalo area WSR-88D will not be directed at the Sonshine Patch Day Care Center during any mode of operation.

1-9

The SEA presents information on both pulse-power and average-power densities because RFR effects have been shown to be caused by exposure to pulses and also longer exposure to average power densities. The WSR-88D emits RFR in short pulses with durations of 4.71 μs or less (i.e., less than 5 one-millionths of a second). The interval of time between pulses ranges as long as 3.14 ms, or more than 600 times as long as the duration of each pulse. The instantaneous maximum power density during emission of a pulse is 2,200 mW/cm^2; maximum power density occurs only in the main beam in the near field within about 800 ft of the radar. The maximum pulse power density does not vary with mode of operation. The maximum average power that will occur within the near field is 0.6 mW/cm^2 during normal operation and 3.85 mW/cm^2 during searchlight-mode operation. See also Section V.B of the SEA.

The 1991 SCC-28 guidelines do include limits on pulsed RFR of high peak power densities, specifically in the section "Peak Power Exposures (p. 33+), which discusses the reasons for doing. In response to the cited comments of Leo Birenbaum, we note that the body can respond to either form of RFR. However, the only well established human response to pulsed RFR (at low average SARs) is the RFR auditory effect, and there is no scientific evidence that exposure to peak levels of RFR below the limits given in the 1991 SCC-28 guidelines would affect human health. For a more complete discussion of RFR auditory effects, see Section V.C.2.4, of the Draft SEA.

1-10

The human body responds in a variety of ways to both RFR pulses and long-term RFR exposure that exceed perception thresholds. Section V.C and Appendix B of the SEA summarize the current state of knowledge concerning the human health effects of exposure to RFR at various power densities and pulse repetition rates. The SEA concludes that "no scientifically valid evidence exists that chronic exposure to WSR-88D RFR will adversely affect the health of humans."

As is indicated in Section V.A.2 of the SEA (p. 23+) it is not possible to prove scientifically that anything is absolutely safe. However, the overall evidence from analyses of published investigations on bioeffects of RFR indicates that human harm from indefinitely long exposure to the RFR from the WSR-88D radar would be most unlikely.

Response to Comments from Rev. Robert L. Smith

2-1

The SEA was prepared by in an objective and neutral manner by SRI International under contract to the NEXRAD JSPO. The SEA preparers and their qualifications are listed in Section VIII of the SEA.

The potential benefits of the WSR-88D radar with regard to earlier warnings about impending storms and other possibly destructive natural phenomena were indeed described in the

SEA. However, the NWS is concerned about possible harm to the health of humans in the vicinity of the radar. It has been and continues to be a policy of SRI International, as a not-for-profit organization, to study such questions of public concern impartially, with no bias toward the outcome of such studies. The conclusions in the SEA regarding human health were based on critical analyses of the scientific literature to determine what is known and what is not known about the subject, always with the understanding that knowledge on any scientific subject is never complete.

Questions of policy and the balancing of benefits and possible risks are in the province of the decision makers, who must consider factors beyond the scope of the environmental review.

Response to Comments from Mary Beth Hoffarth

3-1

The NEXRAD JSPO prepared the Draft SEA to update the information and analyses of the 1984 PEIS. Comments on the Draft SEA were received from the public and government agencies during a 60-day review period. This Final SEA contains responses to substantive comments on the Draft SEA and revisions to the Draft SEA text made necessary by those responses. This document concludes that implementation of the proposed action is unlikely to significantly affect the human environment. Therefore, a finding of no significant impact (FONSI) is appropriate and will be issued by the NEXRAD JSPO.

3-2

See the response to Comment 2-1 above.

3-3

Section V.C and Appendix B of the SEA summarize the current state of knowledge concerning the potential for human health effects to result from exposure to RFR. Sections 3.2 and 3.3 of Appendix B of the SEA specifically discuss possible RFR-induced genetic effects and developmental abormalities in fetuses and infants.

The unborn, infants, ill people, and elderly persons are usually more susceptible to adverse conditions than the remainder of the population. With regard to exposure to RFR, however, many experimental findings indicate the existence of threshold levels above which effects are known to occur. The levels of RFR from the WSR-88D radar will be far below such thresholds and would not be cumulative. Thus, there is no definitive scientific evidence that the health of those more susceptible members of the general population will be adversely affected.

Response to Comments from Arlene Bow

5-1

Very few studies have been performed in which laboratory animals been exposed to RFR for long periods of time relative to their lifespan. In some cases, multiple generations of laboratory animals have been exposed to RFR to ascertain the potential for mutagenic or carcinogenic effects. See Section V.C.2.6.3 and Section 3.2 of Appendix B of the SEA.

Response to Comments from David T. Powell

6-1

The design WSR-88D unit incorporates a number of features that minimize the level of RFR to which the public may be exposed. First, the radiating antenna is mounted on a relatively tall tower within a secured compound. Second, the main beam of the WSR-88D is directed above the horizontal, except at a few remote ridgetop locations where the beam may be directed downward at an angle of 0.5° below horizontal. Third, WSR-88D units are being sited so that the main beam will not illuminate tall buildings or elevated ground within the near field where RFR power densities are greatest (and in most cases in the far field for many miles out from the radar as well). The radar will also not illuminate the ground. Fourth, the WSR-88D is equipped with an interlock to prevent emission of RFR when personnel enter the radome. All of these features reduce the exposure of humans to RFR.

As discussed in the SEA, neither the epidemiologic/occupational studies nor the investigations with laboratory animals to date have provided valid scientific evidence that the 3.0-GHz RFR of the WSR-88D radar will initiate or promote cancer. See also the response to Comment 1-1. Most of the current controversy on this topic is concerned with possible effects from presumed exposure to powerline frequencies (60 Hz), a subject also discussed in the SEA. Prudence does suggest avoidance of as much ionizing radiation as possible (e.g., X-rays, radioactive substances), consistent with appropriate medical diagnosis and treatment. This premise is not applicable to low levels of RFR such as those from the WSR-88D radar because of the fundamental differences between nonionizing and ionizing radiation.

6-2

For military radars such as the PAVE PAWS installations, operational limitations were recommended and subsequently imposed to avoid possible exposure of blasting caps and fuels to excessive levels of RFR. However, the EISs for such military systems treated the topic of human exposure to RFR separately.

Section V.E of the SEA analyzes the potential for the WSR-88D signal to create hazards to fuel handling and electroexplosive devices (including blasting caps), and concludes that the WSR-88D conforms to existing guidelines and standards. The IEEE (1991) revision of the ANSI (1982) guidelines for human exposure to RFR includes separate limits for occupational exposure and exposure of the general public. Section V.B and Appendix B of the SEA analyze the potential for health effects to result from exposure to the WSR-88D signal and conformance with existing guidelines for human exposure to RFR. WSR-88D RFR will not exceed the IEEE (1991) guidelines for uncontrolled environments (1.8 to 2.0 mW/cm^2 at 2.7 to 3.0 GHz) during normal operation. However, during searchlight mode operation, the maximum level of exposure allowed by the IEEE (1991) guidelines could be exceeded. To prevent that hazard from occurring, use of the searchlight mode will be limited so that the resulting RFR power levels conform to the IEEE guideline levels.

6-3

The NEXRAD JSPO believes that there is sufficient scientific evidence to conclude that the implementation of the NEXRAD program will not create a significant potential for human health effects from exposure to WSR-88D RFR. JSPO plans to keep abreast of any new scientific findings that are relevant to the WSR-88D system.

Response to Comments from Susan Lukaszewicz

7-1

See the response to Comment 1-9.

Response to Comments from Sarah C. Lukaszewicz

9-1

The purpose of this document is to describe the environmental consequences that could result from implementation of the WSR-88D system and the level of significance of those impacts. The document includes analyses of numerous scientific studies on RFR bioeffects conducted worldwide for the purpose of assessing whether RFR from the WSR-88D could be harmful to human health. Many of these studies were conducted after the 1984 PEIS was prepared. This document focuses on the findings of more recent studies. The government will consider the information contained in this document while making decisions on the future of this program.

Response to Comments from Dianne M. Puccio

12-1

The WSR-88D Operational Support Facility (OSF, a preproduction model of the WSR-88D) was subject to operational testing from March to August 1989. Excellent data based on Doppler capabilities were obtained for a distance out to 144 miles from the OSF. Conventional reflectivity data were obtained for distances out to 288 miles. The OSF demonstrated superior ability to penetrate intervening weak weather signals as compared to previous weather radars. The OSF was also able to detect light rain, fine drizzle, mist, snow, and other weather phenomenon that were seldom detected by previous radars. During the 5-month trial period, the OSF demonstrated 91% accuracy for severe storm warnings, compared with the current national average of 58% for existing radars. Moreover, the OSF's false-alarm rate of 21% was much lower than the existing national average of 57%. Finally, the OSF was able to detect mesoscale mid-atmospheric conditions that precede tornadoes at distances out to 92 miles. Previous weather radars could not detect those phenomena. In short, the WSR-88D is highly effective at accomplishing its mission.

Response to Comments from Gloria McLaughlin

13-1

Concerning the list of studies referred to by the comment author, please see the response to Comment 37-28 below.

In reference to standards and guidelines for human exposure to RFR, Section V.B.4 of the SEA summarizes RFR safety guidelines, including guidelines promulgated by the IEEE SCC 28, International Radiation Protection Association, National Council on Radiological Protection, the former USSR and standards previously proposed but not adopted by the EPA.

13-2

The Draft SEA was distributed to agencies and individuals who commented on the 1984 PEIS, members of the public expressing interest in the NEXRAD program, responsible federal agencies, and state clearinghouses for environmental documents in all 50 states. Comments on the Draft SEA were accepted during a 60-day period and at a public hearing held in Washington D.C. on April 21, 1992. The Final SEA will be distributed to those who received the Draft SEA and additional individuals or agencies commenting on the Draft SEA. Comments on the Final SEA will be accepted for a 30-day period before any decision-making by the JSPO on implementing the proposed action takes place.

13-3

WSR-88D RFR power levels were measured at the prototype OSF in Norman, Oklahoma. The results of those measurements are described in Section 8 of Appendix A of the SEA (pp. A-19 to A-20). The tests were performed by the Air Force Communications Command, 1839 Engineering Installation Group, Engineering Division, Keesler Air Force Base, MS 39534-6348. The tests were planned and monitored by Mr. Dane E. Ericksen, a professional engineer of an independent commercial firm, Hammett and Edison, Inc., Consulting Engineers, P.O. Box 280068, San Francisco, CA 94128. The tests are described in 1839 EIG Engineering Report, which was endorsed in a separate report issued September 5, 1989, by Hammett and Edison.

13-4

Professor Birenbaum made independent calculations of the peak and average power densities on the axis of the WSR-88D main beam at various distances from the antenna. The discrepancy between his calculations and those presented in the SEA arises from a comparison of two different scan modes. The calculated power densities of 0.6, 0.006, and 0.0004-0.0002 mW/cm^2 apply to the normal scan mode. Professor Birenbaum treated the so-called searchlight mode discussed in Section V.B.4 and in Section 7 of Appendix A of the SEA (see pp. A-18 to A-19). His numbers and the comparable numbers from the SEA are tabulated here:

Condition	SEA	Birenbaum
Near field	3.85	2.64
4000 ft	0.16	0.22
5000 ft	0.10	0.14
5280 ft	0.09	0.13

Some of Professor Birenbaum's figures are higher and some are lower than those presented in the SEA. In all cases, the discrepancy is lower than the 2:1 ratio typical of such calculations. In brief, no disagreement exists in regard to the predicted values.

13-5

Many of the assertions in Dr. Becker's book *Cross Currents* are highly speculative and in large measure not in accord with the findings in most of the scientific literature on nonionizing radiation. For example, Dr. Becker states (p. 214): "*All* [his italics] abnormal, man-made

electromagnetic fields, regardless of their frequencies, produce the same biological effects." Apparently he has ignored the large body of engineering and biological literature indicating the dependence of interactions of fields with biological entities on frequency and, in particular, the differences between low-frequency induction fields and nonionizing far-field radiation.

In his references for Chapter 8, "Man-Made Electromagnetic Fields" (pp. 317-320), Dr. Becker primarily cites his own papers and a few papers that appear to support his views; he omits the many that do not accept his viewpoint. In that context, he appears to give greater credence to anecdotal accounts than to results of scientifically designed experiments.

See also the response to Comment 3-3 above.

13-6

See response to Comment 1-6 above.

13-7

It is quite possible to measure ambient power densities at any chosen location. In 1976, the EPA made such measurements at many locations in 15 cities and in various frequency bands. As stated in Section IV.D of the SEA, the ambient levels were generally below 0.000,002 mW/cm^2. Power densities up to 7 mW/cm^2 were found near some radio transmission towers.

A typical AM radio transmission tower radiates 50,000 W continuously, as compared to the 1,000 W average power of WSR-88D. Radiation from a single AM tower is nondirectional, is strong near ground level, and decreases rapidly with increasing distance. At a frequency of 1 MHz (the center of the broadcast band) the wavelength is 300 m and the antenna height is approximately 100 m. Near the tower and at a fixed distance from it, the power density decreases from a maximum near ground level to a low value at points level with the top of the tower. WSR-88D uses a frequency of 2710 MHz (2710 times higher than the typical AM station) and radiates its power from a large parabolic reflector high above ground level forming a circular beam that is nearly horizontal. The beam passes over nearby buildings and trees, and little of the power reaches the ground. Thus, its radiation is quite different from that of a broadcast station.

See also the response to Comment 38-24.

13-8

The methodology used to calculate the power density of WSR-88D RFR is described in Appendix A of the SEA. At ground level at a distance of 700 ft from a WSR-88D on a 15-m tower, the average power density will be 0.000,05 mW/cm^2, a factor 36,000 times less than the IEEE SCC 28 guideline for exposure in uncontrolled environments. The maximum instantaneous pulse power density will be about 0.6 mW/cm^2. See also the responses to Comments 1-8 and 1-9 above for information on averaged and unaveraged power densities and on use of the searchlight mode.

13-9

In the searchlight mode within 800 ft of the antenna and on the axis of the main beam, the power density of the WSR-88D signal will be as follows:

Averaging Time	Power Density (mW/cm^2)
1.57 to 4.5 μs[a]	2,200
0.00078 to.0022 s[b]	4.62
6 min	3.85
30 min	0.77
[a]Duration of one pulse [b]Duration of a pulse and subsequent "quiet" interval	

The first value applies to a single pulse, when present, or to the instantaneous power density of the RFR from the WSR-88D when it is transmitting a pulse. The second value applies to a single pulse and the silent interval between successive pulses. The third and fourth values correspond to the averaging times for uncontrolled and controlled environments, respectively, designated by the IEEE SCC 28 (1991) guidelines for human exposure to RFR. See also Appendix A of the SEA for a description of the method used to calculate these values.

Those who contributed to the IEEE C95.1-1991 standards are listed in the Foreword of IEEE C95.1-1991 (unnumbered pages 4 and 5). The IEEE standard is not based solely on thermal effects.

See also the responses to Comments 1-3, 1-8, and 1-9.

13-10

The SEA preparers evaluated the evidence concerning RFR health effects on the basis of scientific merit. Some biological effects (e.g., subtle histopathological and histochemical changes of the nervous system) have been reported as occurring at nonthermal levels. However, there is no evidence that those effects are harmful. Thus, in overall conclusion, no valid evidence exists that the exposure to WSR-88D RFR will adversely affect the health of humans. See also the response to Comment 1-3.

13-11

To determine with 100% certainty that no adverse health effects will result from exposure to WSR-88D RFR, many detailed studies under all possible conditions would be required. It is obviously impossible to duplicate every possible real-world condition in the laboratory; therefore, a 100% guaranty is not possible. However, the preponderance of scientific information indicates that adverse health effects are extremely unlikely.

The data obtained in scientific studies are rarely if ever absolutely conclusive, and therefore the findings of each study on a given subject must be weighed against those of the other studies on that subject. Thus, an overall conclusion based on a collection of studies not in full agreement with one another will necessarily contain words indicating judgment or probability.

In response to the question about listing studies by deficiency, Appendix B of this Final SEA provides detailed discussions of hundreds of scientific studies and their relevance to the

analysis of the potential for WSR-88D RFR to cause bioeffects. The findings of some of those studies are questionable for many reasons, including, but not limited to, methodological deficiencies, incorrect statistical treatment of data, and failure of later experiments to replicate results.

Problems with experimental conditions (and methodology) are too numerous to list. One important consideration, for example, is the need to acclimate animals to the experimental situation, to minimize stresses that affect their endocrinologic responses, particularly when possible endocrinologic effects of RFR are sought. As another example, some of the studies seeking RFR effects on cell cultures failed to hold the temperature of the cultures constant during exposure. As still another example, in various studies that reported effects of RFR on the blood-brain barrier, the biological methodologies used were found to yield erroneous positive findings. Measuring temperatures in animals, during exposure, with instruments that perturb the local RFR fields or are perturbed by those fields were serious flaws in some early studies; however, these problems that were virtually eliminated by the subsequent development of nonperturbing instrumentation. Similarly, the development of high-resistance electrodes for measuring EEGs of animals during RFR exposure largely removed the perturbation problems found in early studies.

13-12

The preparers of the SEA are listed in Section VIII of the SEA. Mr. L.N. Heynick and Dr. P. Polson selected the papers for analysis. The criteria for paper selection are discussed in Section V.C.1 and B-1 of the SEA. see also the response to Comment 1-4 above.

13-13

This comment refers to Section V.C.2.1 of the document, which summarizes Section 3.1.1 of Appendix B. Those sections describe in detail a number of epidemiologic studies on RFR exposure. There is no ratio of negative to positive findings that dictates a conclusion in this document.

Detrimental effects of RFR were reported in several epidemiologic studies, and negative findings were reported in others. The uncertainties in assigning individuals to the exposed or control groups (as well as in the levels and durations of exposure) and/or inadequacies in the numbers in each group and statistical treatment of the data did not permit absolute judgments of either the positive or negative findings in each study. However, as noted on page 34 of the SEA, consideration of the studies in both categories showed, on balance, no scientifically reliable evidence that chronic exposure to RFR levels within U.S. exposure guidelines is hazardous.

13-14

Studies are reviewed in this SEA for adherence to established scientific methodology, including proper use of controls, correct statistical treatment of data, etc. Flawed studies have generated both positive and negative findings. The conclusions presented herein are based as much as possible on well-designed and conducted studies.

In general, one can conclude from a collection of imperfect studies containing both kinds of findings that the predominance of the evidence indicates that further research is desirable but not essential for the well being of the general population, or that further research is crucial and that prudent avoidance should be practiced pending the findings of such research. As noted in 13-14(b), the epidemiologic studies indicate the former course.

13-15

Dr. Cleary states:

"It is not possible for me to express an opinion about the potential health effects of the microwave radiation from this source since I have no specific information about it."

Dr. Cleary goes on to discuss several issues concerning RFR exposure and possible health effects, including possible cancer incidence and the adequacy of the ANSI (1982) exposure guidelines. Cancer incidence is discussed in Section V.C.2.6.3 of the SEA and Section 3.2 of Appendix B. The ANSI (1982) guidelines and the recent revision thereof (IEEE, 1991) are discussed in Section 1.2 of Appendix B.

13-16 and 13-17

The quotation from the Draft SEA is meant to highlight the fact that relatively few studies have examined the potential for RFR exposure to cause or promote cancer, as compared with the number of studies examining other possible effects of RFR exposure. Although relatively few in number, existing studies do provide important data on possible effects. The conclusion given in the response to Comment 13-15 above is also applicable to cancer induction or promotion by RFR exposure. Current evidence indicates that a problem is not likely to occur.

The text cited in the comment is meant to contrast the limited number of studies of RFR cancer induction or promotion with the far greater number of studies on other possible RFR effects (e.g., ocular, blood-brain barrier, calcium-efflux effects). It is not meant to imply that research in this area is lacking. See section B.3.2 of this document.

Response to Comments from Florence C. Gioia

14-1

Although there is speculation about a possible connection between exposure to low-level electromagnetic fields and tumor induction, no credible experimental evidence exists for such a connection. See also the response to Comment 1-1 above.

14-2

Section 2.2 of Appendix B analyzes the potential for pulsed RFR to produce biological effects .

See also the responses to Comments 1-9 and 13-9(b).

14-3

The only letter received from Dr. Rodman is one dated June 16, 1989, and attached to the letter from Ms. Gloria McLaughlin. Dr. Rodman is correct in stating that much experimental work has been done since the 1984 NEXRAD PEIS was issued. Studies undertaken since that time are discussed in the 1992 SEA. However, his letter is dated 16 June 1989, almost 3 years before the latter document was issued, and before the publication of IEEE-C95.1-1991, the revision of the 1982 ANSI standard. Thus, his comments in that letter are outdated.

14-4

A public hearing was held on Tuesday, April 21, 1992 – 2 days after Easter Sunday – at the Department of Commerce building in Washington D.C. That public hearing was announced in

the *Federal Register* on Thursday, March 26, 1992. Comments on the Draft SEA postmarked on or before May 26, 1992, were accepted, and all substantive comments are responded to in this Final SEA.

14-5

Genesee County Airport was chosen for the Buffalo, New York, area WSR-88D on the basis of several considerations. One of the most important of these is the fit of the WSR-88D at that site with the nationwide network of weather radars, each of which has an effective range of up to 288 miles. WSR-88D units are being the located to the west, south, and southwest of Buffalo – near Cleveland and Pittsburgh, and in Central Pennsylvania. All of the proposed WSR-88D units are linked together and are mutually supportive. The network as a whole, with the Buffalo area WSR-88D as a crucial link, will provide superb coverage of weather events approaching Buffalo from the west.

The quality of coverage that would be provided by a WSR-88D at the Genesee County Airport site is analyzed in the following report: SRI International (1992), "NEXRAD Expanded Site Survey and Environmental Assessment Report for the Buffalo, New York, Area (Genesee County Airport Site)," Expanded Site Survey Report 276. The report concludes that a WSR-88D at the proposed Genesee County Airport site "...would provide excellent coverage in all directions except toward the southeast through the southwest because of rising terrain. This blockage is unavoidable. The site is well positioned to provide low-level detection of severe-weather approaching the Buffalo, New York area."

14-6

See response to Comment 12-1 above.

14-7

The scientific literature on possible psychological effects of RFR exposure are discussed in Section V.C.2.11 of the SEA and Section 3.7 of Appendix B of the SEA. Those sections describe studies on the behavioral effects on rodents and nonhuman primates. The IEEE SCC 28 guidelines were based partially on the scientific data concerning thresholds for RFR-induced behavioral changes in primates. The WSR-88D will conform with the IEEE SCC 28 exposure levels and will be unlikely to cause behavioral effects in humans.

The scientific literature on the behavioral effects of RFR on animals is extensive (see Section B.3.7 of the SEA). It is also noteworthy that the 4-W/kg criterion on which the 1991 SCC-28 guidelines are based was derived from behavioral studies with animals. With regard to human behavior, there is no experimental evidence that, except for the RFR-auditory effect (see 1-9), humans can perceive low levels of pulsed RFR or time-averaged RFR.

14-8

The epidemiologic/occupational studies discussed in the SEA (Section B.3.1) were retrospective studies, in which a possible link was sought between various forms of diminished human health and assumed previous exposure to RFR, usually based on occupation. On that basis, the presumed RFR exposures were for relatively long periods, but not for 10 to 20 years. It would seem that prospective epidemiologic studies would be preferable that monitor groups of people who are presumed to be exposed to a specific agent over long periods into the future for the occurrence of specific ailments. However, apart from the ethics of doing so, the practicality,

and the cost of such prospective studies, the results would be inconclusive to the extent that the groups exposed to the agent are also concurrently undergoing uncontrolled exposure to other agents, a problem that worsens with increasing exposure duration. Thus, the inherent limitations of prospective, as well as retrospective epidemiologic studies must be recognized.

See the responses to Comments 5-1 and 6-1 above.

14-9

See the responses to Comments 1-10 and 13-11.

14-10

See the response to Comment 12-1 above.

Response to Comments from June C. Vukman

15-1

The Final SEA describes and evaluates the findings of numerous studies on health effects of RFR exposure. The laboratory studies require controlled environments because studies conducted in uncontrolled environments have many variables and the results are difficult to interpret. Epidemiologic studies examine the effects of presumed or measured RFR exposure on large groups of people and do not control for other environmental influences. Both laboratory and epidemiologic studies are evaluated in the SEA.

See also the responses to Comments 2-1 and 13-2 above.

Response to Comments from Gary C. Plan

16-1

Section V.C.4.4 of the SEA describes the draft standard for RFR exposure which was proposed by the EPA, but which was ultimately not adopted.

Response to Comments from Catherine J. Dusen

17-1

The SEA includes reviews of dozens of scientific articles published in the last 5 years. The authors attempted to include information from recent studies consistent, insofar as possible, with the need to review the most important and relevant scientific research applicable to the WSR-88D emissions. Some earlier studies have been rendered obsolete by later studies on the same topic and were discussed in the SEA as background information, but other earlier studies remain relevant. Moreover, the results of some of the more recent studies may not necessarily be correct. Concerning independent review of the SEA, see also the response to Comment 13-2 above.

Regarding selection of studies for review in the Draft SEA, see the responses to Comments 1-4 and 2-1 above.

17-2

Regarding studies by Dr. Cleary, see the response to Comment 1-3 above. About work by Dr. Becker, see the responses to Comments 1-3, 1-4, 3-3, and 13-5 above. Regarding studies by

Dr. Zaret, see the response to Comment 1-5 above. Dr. Szmigielski's work is discussed in responses to Comments 1-2 and 1-3 above.

The comment author gave no specific reference to any study with Hansson as the first author. There was a study with H.A. Hansson as a secondary author:

> R. Nillson, Y. Hamnerius, Kjell Hansson Mild, H.-A. Hansson, E. Hjelmqvist, S. Olanders, and L.I. Persson (1989), "Microwave Effects on the Central Nervous System – A Study of Radar Mechanics," *Health Phys.*, Vol. 56, No. 5, pp. 777-779 (1989).

The findings of that study were essentially negative: "No clinically significant abnormalities were noted in the exposed subjects, nor was psychological performance significantly different from the referents." However, that paper was not discussed in the SEA because the numbers of subjects studied (17 exposed and 12 unexposed) were too small to be statistically meaningful.

17-3

See the response to Comment 1-1 above and the second paragraph of the response to Comment 6-1. In addition, the quoted statement of Dr. Cleary: "Review of the bioeffects literature...provides convincing evidence that RF radiation, and other types of electric and magnetic fields..." can be misleading because he does not distinguish possible interactions of RFR from those for powerline fields – a distinction discussed on page B-14 of the Draft SEA. The reports of cancer from exposure to powerline frequencies (if validated experimentally) are not relevant to the RFR from the WSR-88D radar, and there is no definitive scientific evidence for cancer induction by RFR at the frequencies of the WSR-88D radar.

One of Dr. Cleary's more recent papers (Cleary et al., 1985) was discussed on page 351 of the review by Heynick (1987) cited in the SEA. In that study, preparations of rabbit neutrophils exposed to 100-MHz RFR at high SARs (up to 341 W/kg) showed no significant differences in the viability or phagocytotic ability of such neutrophils from sham-exposed preparations. That study was not included in the SEA because of the large variabilities in the control groups – an indication of the possible presence of uncontrolled non-RFR factors.

Response to Comments from Dan Voltura

18-1

See the responses to Comments 2-1 and 13-2 above.

18-2

Although this poll was not conducted in accordance with scientific principles of polling, it is an indicator of public sentiment regarding the proposed Genesee County Airport site for the Buffalo area WSR-88D.

18-3

See the response to Comment 13-11 above.

Response to Comments from D. Keith Bow

19-1

The cited EPA guideline was proposed but not adopted. The power density of the WSR-88D will not exceed 0.6 mW/cm^2 outside the radome. Thus the OSHA standards and IEEE (1991) guidelines will not be exceeded. At ground level adjacent to the a 15-m WSR-88D tower, the RFR power density during normal operation will be less than 0.001 mW/cm^2, which will conform with all of the guidelines listed in the comment.

The 10 mW/cm^2 value was the basis of the 1974 ANSI standard, a value independent of frequency. Although that standard did not discuss occupational or general-public exposure, OSHA had promulgated its 10-mW/cm^2 value as a voluntary occupational standard, but found it legally unenforceable. The 1982 ANSI standard had superseded the 1974 ANSI standard and, in turn, was superseded by the 1991 IEEE guidelines (see also the response to Comment 1-3 above).

For the frequency range of the WSR-88D radar, the 1991 IEEE guidelines for uncontrolled environments (accessible to the public) are 1.8-2.0 mW/cm^2 as stated in the comments. For controlled environments (not usually accessible to the public; e.g., occupational environments), the guidelines are 9.0-10.0 mW/cm^2.

19-2

See the response to Comment 19-1 above. The RFR power density at the base of the radar tower will not exceed the levels set by the former USSR or the standard recommended by the New York City Department of Health's Bureau of Radiation Control.

It is incorrect to state that the 1991 IEEE standard was not based on objective scientific fact or experimentation. The bibliography of studies used in the development of the standard is given on pp. 35-39 of the document describing the standard. Those studies were selected from the larger list of studies in Appendix A (pp. 41-67) of that document. Various subcommittees of the IEEE SCC 28 used those studies to develop the specific provisions of the 1991 IEEE standard, and acceptance of the standard was by majority vote of the parent body of SCC 28. The IEEE Standards Board then reviewed and approved the standard.

19-3

The position of IEEE with respect to thresholds for safe exposure of humans to RFR is given in the SCC 28 standard issued in 1992. Please see section B.1.2 of the SEA.

19-4

The IEEE (1991) guidelines that superseded the ANSI (1982) guidelines recommend maximum exposure levels for controlled environments (i.e., workplace exposure) and uncontrolled environments (exposure of the public). The latter guidelines are intended to protect the entire population, including the unborn, children, and pregnant women. The WSR-88D will conform with the guidelines for uncontrolled environments. See also Section 1.2 of Appendix B of the SEA.

19-5

U.S. electric-power systems operate at a frequency of 60 Hz, also known as "power frequency." The WSR-88D radar will emit electromagnetic radiation at 2,700 to 3,000 MHz, which is about 45 million times greater than power frequency. The power line serving the WSR-88D will operate at power-line frequencies. Section V.D of the SEA analyzes the potential for human health effects to result from electromagnetic fields generated by the power lines serving WSR-88D units.

19-6

See the response to Comment 13-11 above.

The quoted statements regarding standards are correct. As discussed in the SEA, it is scientifically impossible to prove the negative and thus offer an absolute guarantee of safety. The risk of harm to human health from exposure to the RFR from the WSR-88D radar is negligible.

19-7

All of the nonthermal effects listed in this comment were treated in the SEA, with the conclusion stated in 19-6 above. Further experimental research is desirable, but primarily to learn more about the basic interaction mechanisms between nonthermal electromagnetic fields and biological entities than for determining whether such fields are potentially hazardous. See also the viewpoint of Foster and Guy (1986) in the article cited by the comment author.

19-8

See response to Comment 38-13 below.

19-9

The studies by Samuel Milham referred to by the comment author are analyzed on pages B-21 and B-22 in Appendix B of the Draft SEA.

The cited 1982 paper was a letter to the *New England Journal of Medicine* (Vol. 307, No. 4, p. 249), and the claims therein were questioned in a subsequent letter to the same journal by R.P. Liburdy (Vol. 307, No. 22, p. 1402). A more extensive account of the study was given in a report (Milham, 1983), discussed on pages B-20 and B-21 of the SEA, and the material was subsequently published (Milham, 1985). As noted in the SEA, little credence can be given to the results of this study.

19-10

Several studies on possible behavioral effects of exposure to RFR are reviewed in the SEA. Please see section B.3.7.

19-11

Epidemiologic studies examining the possible connection between power-line electromagnetic fields and cancer are summarized in Section V.D.3.4 of the SEA.

19-12

The SEA preparers reviewed studies conducted by numerous well-respected researchers. Regardless of the source of funding, there is no evidence of widespread fraud in the reporting of

study results. To the contrary, we believe that most, if not all, the studies reviewed were accurately and honestly reported.

19-13

To provide maximum flexibility the NEXRAD system is capable of operating in many scan modes. However, the one that produces maximum power density at and near ground level, and is therefore of principal interest concerning possible effects on human health, is designated pattern 31 and is described in the fourth paragraph on page 26 of the SEA. Professor Birenbaum's calculations of the power density on the axis of the main beam are essentially correct for searchlight-mode operation. However, searchlight-mode operation will be used only once every several years at most and during that operation the main beam of the WSR-88D will not be pointed at schools or any other inhabited buildings. The RFR power density at a distance of 1,200 ft generated by a WSR-88D in normal operation will be about 0.000,05 mW/cm^2, which is a factor of 1,666 times less than 0.05 mW/cm^2 of the "Solon recommendation."

See also the responses to Comments 13-3 and 13-4 about Mr. Birenbaum's calculations.

Response to Comments from Rosan Shaikh

20-1

Paragraph 1 is correct for levels of RFR that far exceed those to be emitted by the WSR-88D radar, but it is most unlikely that any of the effects mentioned in that paragraph will occur at the WSR-88D RFR levels. This document contains detailed discussions of the research studies on RFR bioeffects and the levels at which such effects might occur. For further information on these issue areas, see sections V.C.2.7, V.C.3.8, and V.C.2.8.2, as well as appendix B of the Draft SEA.

20-2

The IEEE SCC 28 guideline for uncontrolled environments in the WSR-88D frequency band is 1.8 to 2.0 mW/cm^2. During normal operation, the maximum level of RFR generated outside the WSR-88D radome will be 0.6 mW/cm^2, which is well below the IEEE SCC 28 guideline.

The RFR levels noted above are the highest permissible levels in the exposure guidelines published recently by the IEEE (1991). Exposures are to be averaged over all 6-minute and 30-minute periods. They do not indicate that exposure for longer durations at those levels would be harmful.

Currently, the 1991 IEEE guidelines are voluntary rather than official ones from regulatory agencies. As noted in the SEA, the EPA has not issued RFR-exposure guidelines for the public.

20-3

The quoted numbers are correct. However, use of the searchlight-mode operation will be limited to 5 minutes during any 30-minute period to reduce the average power density to 0.77 mW/cm2. In addition, the searchlight mode will be used only during initial testing of the WSR-88D, after major equipment repairs, and when requested by the FAA. For a particular radar, the searchlight mode will be used only once every few years.

20-4

Comment noted.

20-5

Regarding paragraph 5a, given the many RFR-bioeffects studies discussed in the SEA, including those that have yielded knowledge about basic mechanisms of interactions, there is no reason to believe that continuous exposure to the low levels of RFR from the WSR-88D radar would have cumulative effects.

The expected useful life of the WSR-88D is 20 years, and the radars will be maintained so that the level of RFR emitted is very close to that described in the SEA. See also the response to Comment 20-3 above.

Response to Comments from Harold S. Masumoto

29-1

The WSR-88D radars planned for Hawaii will be sited in conformance with all applicable environmental laws and regulations, including the Coastal Zone Management Act. An environmental assessment will be prepared analyzing each of the three proposed radars in conformance with the NEPA implementing guidelines adapted by the Council on Environmental Quality and the FAA.

Response to Comments from Chrys Baggett

31-1

The text in question has been revised.

In paragraph 1 on page 21 of the Draft SEA, the sentence "However, absorption...body's metabolic rate." has been replaced with the following two sentences:

> "Absorption of RFR within a body as heat is usually not significant unless the rate at which heat is added is comparable to or higher than the body's metabolic rate. In high ambient temperatures and humidity, however, under which the body is close to the limits of its thermoregulatory system, absorption of considerably lower levels of RFR may cause thermal stress."

The last sentence on page 21 has been changed to:

> "X-ray machines for various purposes (diagnosis, dentistry) and ultraviolet lamps for suntanning are two other artificial sources of ionizing radiations."

31-2

See the responses to Comments 38-7 and 38-19.

Response to Comments from Don N. Strain

32-1

The potential for an individual WSR-88D unit to cause environmental impacts is analyzed in a site-specific environmental assessment. As part of that analysis, the potential for effects on

cultural resources is analyzed through consultation with the state historic preservation officer (SHPO) and performance of site surveys by a qualified archaeologist or historian.

Five WSR-88D units exist in or are planned for Oklahoma. The OSF, Oklahoma City, and Frederick WSR-88Ds have been constructed at sites approved by the SHPO. No adverse effects on cultural or archaeological resources resulted from construction of those radars. The Tulsa WSR-88D will be constructed in 1993 at a site that has been approved by the SHPO. The WSR-88D planned at Vance AFB was analyzed in an environmental assessment prepared by the Air Force. That assessment determined that no potential exists for impact on cultural resources.

Response to Comments from Barry J. and Catherine M. Debbins

35-1

See the response to Comment 1-1 above. Because the worst-case levels of RFR from the WSR-88D radar would be too low to affect human health in any way, no logical reason exists for locating the radars at sites remote from human habitation. In addition, because the bioeffects of RFR are phenomena that do not occur in live mammals below specific threshold levels that are much higher than the levels from the WSR-88D radar, the ALARA principle would not apply.

See also the first paragraph of the response to Comment 6-1 above.

35-2

See response to comment 39-1 below.

35-3

In response to Dr. Lehrman's comment, the results of the study by Balcer-Kubiczek and Harrison (1991), discussed in the response to Comment 1-1 above, indicate that although RFR by itself is not an initiator of tumors, it may be a copromoter with the chemical carcinogen TPA. However, the copromoter results were obtained for cell cultures exposed to RFR levels (4.4 W/kg) far exceeding those from the WSR-88D radar. See also the response to Comment 38-10 below.

The 1983 report by Manikowska-Czerska, Czerski, and Leach was an abstract of a paper presented at a meeting. A search of the Biological Effects of Nonionizing Electromagnetic Radiation (BENER) data base yielded no indication of subsequent formal publication of that study. Because of the lack of information available about this study, its significance cannot be evaluated.

35-4

Dr. Becker criticism of the use of cesarean-derived, barrier-reared rats disregards the questioning of many other RFR-bioeffects studies because of a lack of meaningful control over non-RFR factors. Dr. Becker's incorrect conclusion [p. 195] was that the study "was deliberately designed to sharply reduce the incidence of cancer and infectious diseases in the exposed animals." Dr. Becker is also in error in implying that use of cesarean-section, barrier-reared rats was not clear at the outset. In fact, well before the study was begun, an article by Guy and coworkers entitled "Study of Effects of Long-Term Low-Level RF Exposure on Rats: A Plan" was published in the *Proceedings of the IEEE* (Vol. 68, No. 1, 1980), in which the rationale and

experimental design of the study were described, and the planned use of such rats was stated explicitly (p. 95 of that article).

Dr. Becker's statement that the pituitary, thyroid, and adrenal glands of the rats in the University of Washington study were highly stressed at the RFR levels used (about 0.4 W/kg) and thereby became cancerous is speculative and not in accord with the findings of other studies of RFR effects on the endocrine and immune systems.

Dr. Becker's remark (pp. 196-197 of his book) that the data "are buried in the multivolume official Air Force report of the project" seems to imply that those researchers deliberately attempted to hide their results. As noted above, details of the proposed study were published before its initiation. Moreover, when the study was completed, the results were reported at various technical society meetings, and the Air Force School of Aerospace Medicine published nine technical reports (cited in the SEA) and distributed them widely to RFR-bioeffects researchers. Copies of those reports were readily available to anyone requesting them.

In view of the above, we do not believe that a reevaluation of the data based on Dr. Becker's assertions is necessary.

See also response to Comment 1-3.

35-5

See the responses to Comments 1-9 and 13-9 above.

When appropriate, peak power levels and limits are cited in the SEA and elsewhere. As examples, the threshold peak power densities and energy densities for the RFR auditory effect are discussed in Appendix B, Section 3.1.4 (page B-31 et seq.) of the Draft SEA, and the IEEE (1991) exposure guidelines contain a section on peak power exposure (pp. 33-34).

35-6

As indicated in Section A.2 of the Final SEA it is not possible to prove scientifically that anything is absolutely safe. However, the overall evidence from analyses of published investigations on bioeffects of RFR indicates that human harm from indefinitely long exposure to the RFR from the WSR-88D radar would be most unlikely. Regarding Dr. Becker's remarks on stress, see the response to Comment 35-4 above on the University of Washington study relative to the endocrine and immune systems. In addition, there is no valid scientific evidence of genetic effects.

35-7

As indicated on pages 40 and B-49 of the SEA, the comment refers to a study by Preston et al. (1979). That study was discussed in more detail on page 230 of Heynick (1987), a primary reference cited in the SEA. The following statement on page B-49 of the SEA was inadvertently misleading: "Preston et al. (1979) also showed that RFR-induced changes in the relative sizes of the vascular and extravascular volumes in the brain could be misinterpreted as BBB alterations." That statement was a surmise by the authors, based on the results of a non-RFR experiment they had done on uneven blood-flow distributions of tritium in the brain, rather than on experimental evidence with RFR. The point about relative vascular and extravascular volumes became moot with the development of a better method for measuring BBB permeability by Rapoport et al. (1979), as discussed on page B-49 of the SEA.

Response to Comments from Joel Ray

37-1

The SEA authors have attempted to review the scientific literature in an unbiased and objective manner to determine the potential for human health effects to result from exposure to WSR-88D RFR. The review of the scientific literature examined both the scientific credibility of the studies as judged against accepted norms for scientific research, and the applicability of the study findings to WSR-88D RFR emissions. Because the mechanisms by which RFR may interact with living tissue under many types of environmental conditions are extremely complex, scientific research often provides incomplete or qualified results. As a consequence, findings are often presented in a probabilistic manner.

As discussed in Section V.A.2 of the Final SEA, Assessment of Scientific Information and Risk, the findings of most published reports on RFR bioeffects, even in studies conducted meticulously, contain various sorts of uncertainties. The preparers of the draft SEA analyzed the basis for the various findings in the published reports cited, and derived from them an assessment of each RFR-bioeffects topic based on both the positive and negative findings reported. Because such assessments cannot be rendered in absolute terms, words such as "probable" and "unlikely" were used.

Studies with inadequacies in biological methodology, apparatus and instrumentation, or lack of control over extraneous factors, were given relatively little weight. Discussions of accounts of studies that provided few details were not included in the SEA, a point particularly applicable to many of the early reports from Eastern European countries. Anecdotal accounts were also excluded because they are analogous to hearsay and do not provide a scientific basis for assessing their findings, although they may be useful in directing further investigation.

Dr. Ray's assertion that the preparers of the SEA (those who used the passive voice) were dishonest or were coopted by the NWS or the DoD is without basis. The individuals who prepared the Draft SEA (see section VIII of the Final SEA for a list of preparers and their qualifications) have had many years of experience on RFR bioeffects. They are independent contractors hired by the government to conduct an unbiased evaluation of possible hazards to human health of the WSR-88D radar.

The existence of nonthermal RFR bioeffects is described in the SEA. Also indicated, however, is that there is no scientific evidence that such nonthermal effects would be manifested from exposure of humans to the levels of nonionizing electromagnetic fields from WSR-88D radars or that chronic exposure to such levels would adversely affect human health.

37-2

Most of the publications by Dr. Zaret on RFR cataractogenesis refer to cases of individuals who have developed cataracts, very likely from exposure to RFR at high levels. Such individual cases were not relevant to whether chronic exposure to the RFR from the WSR-88D would be hazardous to human health. The progress of research on RFR cataractogenesis is described in Section 3.1.3 of Appendix B of the Final SEA. The findings of Kues et al. (1985) on corneal damage at relatively low RFR levels (but far above those from the WSR-88D radar) were discussed therein, and a discussion of the results of recent work by Kues and coworkers (published in 1992) has been added to the SEA (see the response to Comment 38-13).

37-3

As discussed in the SEA (see Appendix B, Section 3.4.1 of the Final SEA), Frey's research on RFR-induced changes in the blood-brain barrier (BBB) was not given much credence because the tracer used, fluorescein, was found in the control animals as well as in those exposed to RFR. Nevertheless, on the basis of Frey's findings, other researchers also sought effects of RFR on the BBB, but were unable to confirm such findings. On the basis of better experimental methods, such as those used by Williams et al. (1984), there is no scientifically valid evidence that RFR at nonthermal levels alters the permeability of the BBB. See also the response to Comment 38-14.

37-4

The work of Dumanskiy and Shandala (1974) was discussed on pages 297-298 of Heynick (1987), a primary reference for the SEA. Discussion of that work was not included in the SEA because, as stated on page 298 of that reference:

> The rather sketchy description of the study precluded any definitive evaluation of the results. Absent in this paper were details on the exposure arrangements and the method used to record and process the EEGs. Specifically, not indicated was whether indwelling electrodes had been used. Presumably they were, however, because such electrodes were used in a later study, Shandala et al. (1979), discussed below.

37-5

The EEG findings of Shandala et al. (1979) are not credible because of the use of implanted metallic electrodes. Their other findings are also suspect.

37-6

The SEA authors are unaware of the specific study by Zinaida Gordon on clinical microwave effects. A detailed citation of that study was not provided by the comment author. Therefore, the significance of that work cannot be evaluated.

37-7

See the responses to Comments 1-3 and 35-4 above.

37-8

This 1981 letter publication by Friedman did not provide sufficient information to evaluate the findings. The BENER database shows no later publication by Friedman on this study.

37-9

Czerski's 1975 work on the immune system was discussed on page 344 of Heynick (1987), a primary reference for the SEA. As noted therein, that study was an attempt to reproduce results obtained by Stodolnik-Baranska (1974), discussed on page 343 of Heynick (1987). Neither study was included in the SEA because in the few positive results obtained, Baranski and Czerski (1976) implicated uncontrolled RFR-induced temperature increases in the specimens exposed to RFR, and found no effects at power densities below 5 mW/cm^2, as compared to the maximum power density of 0.6 mW/cm^2 that will be generated by the WSR-88D radar.

37-10

The study of Polish military personnel is discussed by Szmigielski in the book *Modern Bioelectricity* by A.A. Marino. On page 906 of the book, Dr. Szmigielski notes that the report on that study, Bielec (1985), in Polish, had received only limited distribution and will be published in the unspecified future only in part. The information given in the book does not permit independent analysis of its scientific merits.

37-11

See the first paragraph on page 70, section V.D.3.4 of the SEA for an analysis of studies on possible relationship between exposure to EMFs and breast cancer. Stevens' thesis has not yet been subject to experimental testing and as such is speculative.

37-12

Early studies in Eastern Europe were discussed in Heynick (1987) if the information available was adequate. For the most part, the reports on those studies are lacking in detail on methodology, statistical analysis, etc., and have been superseded by newer studies.

37-13

Neither the Moscow Embassy situation nor of the New York State hearing on power line fields were misrepresented. See the response to Comment 1-1 above.

37-14

The "Leal-Delgado henhouse reports" were initially given considerable attention by scientists. Follow-up studies gave mixed results and indicated that observed effects may have been due to variability in incubation temperatures, and in time between purchase of eggs and incubation. Possible developmental effects of RFR exposure are discussed in Section B.3.3 of the SEA.

37-15

This reference is to a 1984 abstract; no later full publication was identified; therefore, the significance of this study cannot be evaluated.

37-16

The reports referenced by the comment author were not published in peer-reviewed journals. For a summary of studies on ocular effects, see section B.3.1.3; on nervous-system effects, see Section B.3.4; on developmental effects, see Section B.3.3; and on behavioral effects, see Section B.3.7.

37-17

See Section B.3.1.3 for a summary of ocular effects research.

37-18

The most recent and well-published research on possible association between leukemia and EMF is summarized in Section V.D.3.4.

37-19

This citation is to an abstract of a paper presented at a meeting. Because complete details are lacking, the significance of this study cannot be evaluated.

37-20

See the response to Comment 1-4 above.

37-21

See the response to Comment 1-3 and the fifth paragraph of the response to Comment 37-1. The vast differences between electromagnetic fields at radiofrequency, such as the WSR-88D emissions, and power-line frequencies are described below:

For electromagnetic radiation, wavelength and frequency are related in simple inverse fashion: at low frequencies, the wavelengths are long; at high frequencies, the wavelengths are short. In particular, a 60-Hz wave is more than 3,000 miles long. Thus people near a 60-Hz powerline source are in the "induction zone," within which terms such as "propagation" and "radiation" do not apply. Rather, the electric and magnetic fields from such a source, either separately or together, may induce currents in the body that alternate in direction at the 60-Hz rate. The strengths of the fields within the induction zone of a source decrease with distance from the source. In seeking possible biological effects at such low frequencies or long wavelengths, the electric and magnetic fields should be considered separately.

In the WSR-88D frequency band (2,700 to 3,000 MHz), a distance equal to one wavelength is only about 4 in. Thus, it is correct to say that sources operating at such frequencies emit and propagate electromagnetic fields as far-field radiation, for which the intensity falls off with the square of the distance even at short distances from such sources. In such radiation, the electric and magnetic components are at right angles to each other and to the direction of propagation, and the ratio of their amplitudes is a constant.

Based partly on the foregoing, a primary difference between power-line and microwave fields is the way in which each interacts with a biological body in terms of penetration and absorption. A 60-Hz magnetic field pervades the body with little change and a 60-Hz electric field is greatly attenuated internally (up to 100,000 times smaller than it would be outside) in a complicated way that is related to the electrical properties of the constituents of the body. The level of each field at any small region within the body adds and subtracts, at the 60-Hz rate, from the internal fields existing in that region. Whether such fields can cause any effects that are biologically significant largely depends on whether the levels of the 60-Hz external fields substantially alter the internal fields.

Microwave radiation incident on a body is not all pervasive, but is partially reflected at the surfaces of the body facing the source. The percentage of the incident power density that is reflected depends on the microwave frequency. Most if not all of the unreflected percentage is absorbed within the body, but not uniformly. Instead, absorption is highest near the surface and it progressively diminishes with depth at a rate that also depends on the frequency. For the simple case of a flat slab facing a 3-GHz source, about 56% of the incident power density is reflected at the surface and the remainder decreases to about 14% of the surface value at a depth of about 2/3 of an inch, commonly called the "penetration depth." For radiation at higher and higher

frequencies, the penetration depth is progressively more shallow, and above about 10 GHz, absorption is largely confined to the skin (much like sunlight).

In sum, because of the basic differences above, the controversy regarding bioeffects of power-line frequencies is not relevant to possible bioeffects of the RFR from the WSR-88D radar.

37-22

No federal standards or guidelines currently apply to RFR exposure. Section V.C.4 of the SEA summarizes guidelines developed by IEEE, IRPA, NCRP, and the former USSR, as well as the proposed but not adopted EPA guideline.

37-23

Section 1 of Appendix B of the SEA sets forth the criteria used to select scientific articles for review in the SEA. The SEA concentrated on peer-reviewed articles because they meet accepted standards for scientific research and present the detailed information necessary to judge the validity of the findings and their applicability to the WSR-88D. Anecdotal information is not presented in a scientific manner and generally lacks experimental controls or detailed description of conditions under which the incident occurred. As a result, it is much less usable than peer-reviewed articles.

37-24

The RFR exposure guideline adopted by the Johns Hopkins Applied Physics Laboratory (JHAPL) is 0.15 mW/cm^2, or 12 times, lower than the IEEE SCC 28 guideline for uncontrolled environments. During normal operation, power density specified by JHAPL will be exceeded only within the main beam of the WSR-88D within 75 ft of the radar. That level will never be exceeded at ground level.

RFR safety limits, including those of the former Soviet Union, are discussed in Section V.C.4, page 52+ and in Section 1.2 of Appendix B, page B-1+ of the SEA. The IEEE (1991) guidelines developed by SCC 28 were officially published after the Draft SEA was completed, and that section of the SEA has been revised accordingly. The safety limits of organizations such as those of the Johns Hopkins Applied Physics Laboratory were not specifically cited in the SEA because the differences in criteria for such guidelines from those of ANSI (1982) or IEEE (1991) were not clearly indicated.

In the absence of a governing federal standard (but not necessarily for that reason), various state, county, and municipal bodies have issued ordinances on exposure of the general population to RFR that are usually more stringent than those of ANSI (1982) or SCC 28 (1991). Most such standards refer to the 4 W/kg SAR used as the basis of the 1982 ANSI guidelines, but with a safety reduction factor of 50 [to 0.08 W/kg] instead of 10.

37-25

The power density of RFR emitted by a WSR-88D on a 15-m high tower during normal operation will be 0.001 mW/cm^2 (1 microwatt/cm^2) at the base of the tower. Thus, it is extremely unlikely that any members of the public will be exposed to RFR power densities exceeding the former Soviet or Chinese standards.

The literature on U.S. studies at low (nonthermal) levels was treated extensively in the Draft SEA. Those studies offer no credible evidence that such low RFR levels would be hazardous to human health. The findings in the literature on early Eastern European studies were largely supplanted by subsequent studies reported in Western publications.

37-26

The qualifications of the Draft SEA authors are shown in Section VIII of the Draft EA. Dr. Peter Polson, one of the SEA authors, holds a Ph.D. in brain research.

37-27

The scientific currency of the SEA is evident in the many recent references cited therein. See also the response to Comment 9-1 above.

37-28

The reference lists appended to Dr. Ray's letter were examined carefully. For the reasons stated previously, many of the citations from Eastern European countries have not been commented on herein; exceptions were those that Dr. Ray specifically alluded to in the body of his letter. A comparison of his lists of Western studies with the reference list the SEA will show that many of the studies he alluded to are discussed in the SEA. Regarding the existence of nonthermal effects, see the fifth paragraph of the response to Comment 37-1.

37-29

The preparers of the SEA presented an objective analysis of the RFR-bioeffects literature to determine whether the health of humans exposed to RFR from the WSR-88D radar would be adversely affected. A primary purpose of the SEA is to evaluate the most recent available scientific literature on this subject. For situations in which there is reason to believe that exposure to RFR may adversely affect human health, measures to prevent that hazard are recommended (e.g., the recommended limitations on use of the searchlight mode given on page 31 of the SEA). However, for operation in normal mode, no scientific basis exists for concluding that the radar would be a threat to human health.

Response to Comments from Richard E. Sanderson

38-1

Section 1 (page B-1) of Appendix B of the SEA describes the criteria used to select papers for review for the SEA analysis of the potential for human health effects to result from exposure to WSR-88D RFR. In general, papers were selected for review on the basis of relevance to the WSR-88D system, originality, and quality of the findings. Preference was given to peer-reviewed articles published in respected scientific journals. Several comment authors have suggested additional papers for review. The Draft EA analysis has been revised as described in the responses to comments herein to include the findings from relevant additional papers where they contribute substantially to treatment of the subject.

38-2

The last three sentences of the first paragraph on page B-14 of the Draft SEA have been revised as follows:

Some researchers regard these reports as evidence of potentially harmful nonthermal RFR bioeffects. There is no experimental evidence that the RFR-auditory effects is harmful to humans or animals. Other researchers were unable to confirm that pulsed RFR at nonthermal time-averaged levels alters the BBB or adversely affects behavior. The calcium efflux effect and its purported mechanisms remain controversial (Myers and Ross, 1981; Albert et al., 1987; Halle, 1988; Sandweiss, 1990; Adair, 1991; Prasad et al., 1991). These topics are discussed more fully later.

The EPA comment that other researchers who reported negative findings on the calcium efflux effect did not replicate the experimental conditions is true in several instances, such as in the study by Shelton and Merritt (1981). However, the pulsed waveforms used by Shelton and Merritt more closely resemble the WSR-88D waveforms than the amplitude-modulated waveforms of Blackman and coworkers, therefore Shelton and Merritt's negative findings are more relevant to the WSR-88D radar than the positive findings of Blackman and coworkers.

In response to the EPA comments on the abstract by Lee et al. (1987), note that the abstract contains little information other than that the rate of calcium efflux was not affected by 16-Hz-modulated RFR when the physiological washing medium was held at 37 °C, but was affected at 26.5 °C. Those results were clearly preliminary, with no subsequent publication of that study in any journal. Dr. A.W. Guy, the director of the laboratory involved, who was also one of the coauthors of the Lee et al. (1987) abstract, provided several clarifying points (Guy, 1992).

In the experimental protocol used by Blackman and coworkers (and their predecessors), the chick-brain hemispheres to be RFR-exposed and sham-exposed were incubated in a solution having a prescribed concentration of $^{45}Ca^{++}$, after which the samples were washed several times in a similar solution without $^{45}Ca^{++}$ to remove any unattached $^{45}Ca^{++}$. Guy (1992) indicated that in the Lee et al. (1987) study, they had found no significant differences between RFR-exposed and sham-exposed samples that had been washed in such solutions held at 37 °C. They also saw no significant differences between samples that had been washed in solutions held at 26.5 °C if the RFR-exposed and sham-exposed samples were inserted in their respective chambers concurrently. However, they did see a significant difference between RFR-exposed and sham-exposed samples were inserted in their respective chambers concurrently. However, they did see a significant difference between RFR-exposed and sham-exposed samples if, after the washing, there was a time lag between inserting the RFR-exposed and sham-exposed samples in their respective chambers, but only if the RFR were present in the RFR-exposure chamber. The authors had provisionally ascribed that difference in response to differences in the temperature-versus-time profiles of the samples because of the time lag.

Guy (1992) stated that the effect above was observed for one RFR level within the reported amplitude window but also at two levels considerably higher than the upper boundary of the window, and it occurred whether the RFR was amplitude modulated or not. He also noted that those results led the authors to conclude that they had seen no evidence for the existence of an amplitude window or for an effect ascribable to amplitude modulation per se.

In a study by Dutta et al. (1984) on calcium efflux for 16-Hz-modulated 915-MHz RFR that was cited by EPA, the SARs that yielded statistically significant increases in calcium efflux from human neuroblastoma cells were 0.05, 0.75, and 1.0 W/kg (54.7%, 11.6%, and 16.6%, respectively); no significant chambes were seen for intermediate SARs or for other SARs as low

as 0.01 W/kg or as high as 5.0 W/kg. Efflux increases (3.5% and 19.8%) were also obtained for unmodulated RFR at 0.05 and 1.0 W/kg. The finding of significant increases in calcium efflux for both modulated and unmodulated RFR art 1.0 W/kg appears to vitiate a basic aspect of the effect: the need for amplitude modulation (at 16 Hz), at least for human neuroblastoma cells. Moreover, the validity of comparing the Dutta et al. (1984) findings on human neuroblastoma cells with those on chick brains is questionable.

In general, the scientific validity of using the chick-brain preparation is further brought into question by the finding by Blackman (1990) that the "[chick-brain] tissue is electrically dead when it is removed from the animal; within 15 min after removal, no electrical signal from it can be detected, and when electrically shocked there is no response that would indicate an electrically active tissue." Even though the tissue may be utilizing oxygen, use of such a "brain-dead" preparation as a guide to what might happen to electrically viable tissue is highly questionable.

Further impediments to the calcium-efflux model have been reported (Blackman et al., 1991). The "release" of calcium ions from the chick-brain preparation has been shown to be dependent on the temperature of the tissue samples before and during exposure. Control over tissue-sample temperature before exposure was not exercised in all of the previous studies, thereby indicating the possibility that many of the previously reported effects were artifactual.

38-3

Because there is no evidence that the calcium efflux effect is harmful to human health, the SEA described the effect briefly and how it had evolved historically since its discovery, rather than having undertaken an exhaustive treatment of the topic. The papers identified in the comment were examined, but the findings of those papers do not indicate the need to alter observations of the SEA with respect to the calcium efflux effect.

38-4

The last sentence of the sixth paragraph on page B-57 of the Draft SEA has been deleted. The citation "Blackman et al. (1985)" in the first sentence of the last paragraph of page B-57 of the Draft SEA has been changed to "Blackman et al. (1982)".

38-5

The last sentence of the third paragraph on page B-58 of the Draft Sea has been deleted.

38-6

The Blevins et al. (1980) paper was not included in the SEA because it does not describe the microwave dosimetry in sufficient detail to permit an adequate evaluation of the findings. Specifically, it was not clear how the five points of "approximately the same amount of radiation energy" (p. 512, second paragraph) within the microwave oven were found and quantified, and to what extent the authors had accounted for any differences in the heating profiles among the groups of five 2-ml top agar tubes exposed concurrently to the microwaves, and between the microwave-exposed tubes and those heated conventionally.

Concerning this paper, EPA reviewers stated (EPA, 1984, p. 5-102): "Their conclusion that microwave radiation is a potent mutagen, because it caused mutations in excess of those expected from the radiation-induced temperature rise, is not supported by their data."

38-7

The type of heating produced by RFR is not unique, but its distribution within the body may differ considerably from the internal distribution from conventional external heating of the surface because the RFR heating depends strongly on the dielectric and thermal properties of the various constituents of the body (as well as on the characteristics of the incident RFR). The point here is that, depending on the internal locations of significant temperature increases from RFR exposure, the functioning of various organs or interactive systems (e.g. the endocrine and immune systems) within the body may be altered in a manner different from those due to elevated external temperatures and relative humidities.

The plausibility of a possible relationship between tumor incidence and excessive local heat—i.e., between tumor incidence and stress—stems from a study by Riley (1981). Among the conclusions of that paper was the following: "the forces of stress are limited, and in many circumstances are unable to exert any significant pathological effects. In other cases, however, the physiological and hormonal changes induced by emotional or anxiety sgress are capable of shifting an immunological equipoise to produce lethal consequences. Various tumor-host models have been used to illustrate these circumstances."

The consequences of RFR exposure were not specifically addressed in that paper. However, experimental verification of a relationship between RFR heating and increases in tumor volume in a tumor-heat model was reported in an abstract (Riley et al., 1980). Work on the subject of concern and RFR exposure as a stressor was not continued, presumably because of Riley's untimely death.

The relevance of the microwave hearing effect to local thermal effects, which is suggested in this EPA comment, is questionable. In that effect, an RFR pulse of appropriate characteristics produces a sharp temperature differential across a boundary between dissimilar dielectrics. The duration of that temperature gradient is short (comparable to the pulse duration), and the spatially averaged temperature increase is negligible. That temperature gradient (which launches an acoustic wave that is perceived by the auditory system as sound) is far different from local but macroscopic temperature rises at "hot spots," discussed on pages B-12 and B-13 of the SEA.

The EPA comment about the basal metabolic rate of mice is correct; the authors of the paper were in error. The applicable text in the SEA has been revised.

The discussion of the Szmigielski et al. (1982) paper in the last three paragraphs on page B-40 and the first three paragraphs of page B-41 of the Draft SEA has been replaced by the following:

> Szmigielski et al. (1982) investigated whether exposure to RFR: decreases the natural resistance of Balb/c mice to lung cancer cells injected intravenously before exposure; increases the incidence of breast tumors in female C_3H/HeA mice, a strain known to have high spontaneous incidence of such tumors; and increases the incidence of skin cancer in male Balb/c mice locally depilated and painted with the chemical carcinogen 3,4-benzopyrene (BP).
>
> RFR exposures were to far-field 2.45-GHz RFR at 5 mW/cm^2 (SAR 2-3 W/kg) or 15 mW/cm^2 (6-8 W/kg). For exposure, 40 mice were placed in 4 polymethacrylate cages holding 10 mice each. Other groups of mice were sham exposed. The exposures were for 2 hours a day, 6 days a week, for

periods of 1 to 6 months. The temperature (22-23°C) and humidity (60-70%) within the exposure chamber were held stable by external ventilation.

As additional controls to normal (cage-control) and sham-exposed mice, groups of male Balb/c mice were grown for 1 to 8 months, starting at age 6 weeks, within cages 20 x 30 x 10 cm in size containing 20 transparent 5 x 6 x 10-cm compartments, with one mouse in each compartment. The authors (citing appropriate references) noted that growth under such confinement causes a chronic stress syndrome with aggressiveness.

In the lung cancer study, Balb/c mice were intravenously injected with sarcoma L_1 cells. The mice were killed 14 days later, their lungs were infused with India ink in fixative, and the numbers of white neoplastic nodules (colonies originating from single cells) were counted. Based on pilot experiments, the concentration of sarcoma L_1 cells used was 2×10^5 L_1 cells (in 0.1 ml of saline), which yielded a mean control value of 2.8 ± 1.6 (SD) nodules per mouse.

RFR exposure of injected mice for 3 months at 5 mW/cm^2 produced 6.1 ± 1.8 nodules, whereas 10.8 ± 2.1 nodules were seen for exposure at 15 mW/cm^2, a significant difference. Injection of mice that had been grown in confinement for 3 months showed 7.7 ± 2.0 nodules, a mean comparable to that for exposure at 5 mW/cm^2. Smaller but significant differences were seen after RFR exposure or confinement for 1 or 2 months.

"In the breast-cancer investigation of C_3H/HeA mice, groups of 40 mice each were exposed to RFR from age 6 weeks up to age 12 months, and each mouse was checked every 2 weeks for palpable breast tumors. The cumulative numbers of mice with discernible tumors and their survival times were tabulated. By regression analysis, the results were summarized in terms of CDT_{50} (mean cancer development time in 50% of the mice) and MST_{50} (mean survival time of 50% of the mice). The CDT_{50} values were 219 days for 15 mW/cm^2, 255 days for confinement-stressed mice, 261 days for 5 mW/cm^2, 297 days for sham exposure, and 322 days for cage controls. Thus, the CDT_{50} values for 5 mW/cm^2 and confinement stress were comparable, and were between those for 15 mW/cm^2 and the cage controls. The results for MST_{50} were analogous.

Table B-1 of the paper, adapted below, showed the cumulative numbers of mice with tumors at 4, 6, 8, and 10 months:

Table B-1
CUMULATIVE NUMBERS OF MICE WITH TUMORS

	Tallied at:			
Treatment	**4 Months**	**6 Months**	**8 Months**	**10 Months**
Cage controls	0	0	2	16
Sham-exposed	0	0	3	14
RFR at 5 mW/cm^2	0	3	18†	32†
RFR at 15 mW/cm^2	1	11†	26*	37*
Confinement stress	0	2	16*	31*

*p < 0.05 relative to cage controls.
†p <0.01 relative to cage controls.

Sham exposure did not significantly increase the numbers of mice with tumors, but both exposure at 5 mW/cm^2 and confinement stress yielded similar increases in the numbers of mice affected. Exposure at 15 mW/cm^2 yielded significantly higher increases.

At 5 mW/cm^2 (2-3 W/kg), no increase in rectal temperature was seen, but the authors noted that such SARs exceed the basal metabolic rate of the mice. In actuality, the basal metabolic rate of mice is about 9-10 W/kg (Durney et al., 1986), which is much higher than 2-3 W/kg estimated by the authors. No rectal-temperature increase was seen at 15 mW/cm^2 (6-8 W/kg), either, but the authors suggested the possible existence of 'hot spots' within the mice.

In the skin-cancer experiments, to evaluate the effects of BP alone, 40 six-week-old male Balb/c mice had a 1-cm^2 of skin depilated, with the areas painted with BP (in a solvent) every other day for 5 months; controls were similarly depilated but painted only with the solvent. Cancer development was scored by histopathologic examination on a subjective 7-grade scale from 0 to 6. A score of 4 indicated that small papillomas had been found microscopically to contain cancer cells. Thus, mice with scores of 4-6 were regarded as having skin cancer, and those with scores 1-3 as having precancerous skin lesions. Skin cancer occurred within 7-10 months in more than 85% of those treated with BP.

Table B-2 of the paper, adapted below, shows the cumulative numbers of mice with skin cancer (scores 4-6) for cage controls (5 mice per cage), sham-exposure, RFR exposure at 5 mW/cm^2, or confinement stress for 1 and 3 months before BP treatment.

Table B-2
CUMULATIVE NUMBERS OF MICE WITH SKIN CANCER FROM EXPOSURE TO RFR BEFORE BP TREATMENT

	Tallied at:			
Treatment	**4 Months**	**6 Months**	**8 Months**	**10 Months**
Cage controls	0	0	3	18
Sham-exposure	0	0	4	19
RFR at 5 mW/cm^2 (for 1 month before BP)	0	2	18†	27*
Confinement stress (for 1 month before BP)	0	3	16†	24
RFR at 5 mW/cm^2 (for 3 months before BP)	1	22†	29†	36†
Confinement stress (for 3 months before BP)	0	16†	25†	31*

*$p < 0.05$ relative to cage controls.
†$p < 0.01$ relative to cage controls.

As in the breast-tumor study, the numbers of mice affected by exposure at 5 mW/cm^2 or confinement stress were comparable. It is not clear whether similar experiments were done for exposure at 15 mW/cm^2 for 1 and 3 months before BP treatment.

Regarding CDT_{50} values, control mice (treated only with BP) developed skin cancer (scores 4-6) in about 10 months (a CDT_{50} of 296 days). The results for other treatments were obscured by the order of their presentation, but presumably indicated that the CDT_{50} values for sham-exposure, confinement stress, exposure at 5 mW/cm^2, or exposure at 15 mW/cm^2 for 3 months before BP treatment were respectively 272, 201, 171, and 171 days, with the latter 3 values significantly lower those for the control mice. The reason for the lack of difference between the CDT_{50} values for 5 and 15 mW/cm^2 is not clear.

Table 3 of the paper, adapted below, shows the results for concurrent RFR exposure and BP treatment. The only corresponding CDT_{50} value indicated was 131 days for mice exposed at 15 mW/cm^2. However, the MST_{50} values were 331, 268, 237, and 165 days for cage controls, exposure at 5 mW/cm^2, confinement stress, and exposure at 15 mW/cm^2, respectively.

Table 3 CUMULATIVE NUMBERS OF MICE WITH SKIN CANCER FROM CONCURRENT RFR AND BP TREATMENT				
	Tallied at:			
Treatment	4 Months	6 Months	8 Months	10 Months
Cage controls	0	0	3	18
Sham-exposure	0	0	5	21
Confinement stress	2	13*	26*	31*
RFR at 5 mW/cm^2	1	12*	23*	32*
RFR at 15 mW/cm^2	9†	28*	33*	38*
*$p < 0.01$ relative to cage controls. †$p < 0.05$ relative to cage controls.				

In their discussion, the authors stated: 'It may be postulated that the differences in the appearance of tumors and the number [of] lung cancer colonies between animals irradiated with 2,450-MHz microwaves at 5 and at 15 mW/cm^2 may be due to local thermal effects evoked at 15 mW/cm^2.'

They also noted that 2.45-GHz RFR is close to resonance for mice, that maximal absorption by humans at this frequency would be almost 2 orders of magnitude lower than for mice, and that RFR absorption by humans at their resonant frequencies (60-70 MHz) would be about 20% of the absorption by mice at their resonant frequencies. They stated: 'Thus, the mouse model used in this study is of very limited value for concluding about the possible hazards from microwave radiation in human subjects.'

The relevance of the microwave hearing effect to local thermal effects, suggested in this EPA comment, is questionable. In microwave hearing effect, an RFR pulse of appropriate characteristics produces a sharp temperature differential across a boundary between dissimilar dielectrics. The duration of that temperature gradient is short (comparable to the pulse duration), and the spatially averaged temperature increase is negligible. That temperature gradient (which launches an acoustic wave that is perceived by the auditory system as sound) is far different from local but macroscopic temperature rises at "hot spots," discussed on pages B-12 and B-13 of the SEA.

38-8

The purpose of this document is to analyze the potential for WSR-88D radars to cause environmental effects, including health effects in humans. To that end, the IEEE SCC 28 guidelines are summarized and compared to RFR levels generated by the WSR-88D radar.

EPA's original comment was whether cancer promotion would be a better basis for RFR exposure guidelines than the work-stoppage criterion used by Subcommittee IV of SCC 28 in deriving the relevant sections of the IEEE (1991) guidelines. Criteria recommendations for future guidelines—e.g., revision of IEEE (1991)—are are not within this document's scope of assessing

the potential environmental consequences of the WSR-88D radar, and it would not be appropriate to recommend that criterion or other criteria for setting exposure guidelines therein.

In the development of the IEEE (1991) guidelines, the paper by Szmigielski et al. (1982) was one of the many cited in the data base (comprising Appendix A of those guidelines). That paper was also one of those finally selected (and listed in Appendix B of those guidelines) for review by the appropriate SCC 28 subcommittees.

38-9

In the University of Washington study, the additional heat load from the RFR did not exceed about 0.4 W/kg or about one-fifteenth of the basal metabolic rate of a rat (Durney et al., 1986, Table 10.4, p. 10.20), and thus was most unlikely to have caused endocrine stress.

Although the rats used in the University of Washington study may have been stressed initially by handling and the novelty of the experimental situation, during the study they were removed from their individual waveguides for only about 1 hour per day for tests and routine animal care, and spent the remaining 23 hours per day in their waveguides for their entire lifetimes, thereby becoming fully acclimated. In addition, the environmentally controlled room that contained the exposure waveguides was equipped to perform those tests and the animal care routine, thus avoiding stress from transporting the rats to another room, as has been the practice in other animal studies.

The last four paragraphs on page B-42 and the first three paragraphs on page B-43 of the Draft SEA have been replaced by the following:

> There were 22 RFR-exposed and 21 sham-exposed rats in the 2 kill groups. The all-other category had 41 deaths in the RFR group and 28 in the sham group. Chi-square analysis showed no association between cause of death and exposure condition. In addition, the log-rank statistic showed no significant differences between the RFR and sham groups in survival times for glomerulonephritis, atrial thrombosis, or pituitary adenoma; the RFR group had significantly longer survival times for urinary tract blockage than the sham group.
>
> The lesions found in the various organs and tissues during necropsy were characterized as nonneoplastic or neoplastic, and the neoplastic lesions were subdivided into benign and malignant. Of the nonneoplastic lesions, glomerulonephropathy was the most prevalent. Analysis of the data by incidence, age, and treatment indicated that significantly fewer glomerulonephropathic lesions had occurred in the RFR group. No significant differences occurred between the RFR and sham groups for nine other major types of nonneoplastic lesions.
>
> Only 3 benign neoplasms occurred in rats younger than 1 year, and those were in the sham group. During the second year, benign neoplasm incidence rose rapidly with age for both the RFR and sham groups, but the differences between groups at each age of death were nonsignificant.
>
> No primary malignant lesions were found in the rats younger than 1 year. Primary malignant lesions were found in 2 RFR-exposed and 2 sham-exposed

rats at ages 13-18 months, in 9 of the RFR group and 1 of the sham group at ages 19-24 months, and in 7 of the RFR group and 2 of the sham group at ages 26-30 months.

Without regard to age, there were totals of 18 rats with malignancies in the RFR group and 5 rats in the sham group, a difference noted by the authors to be statistically significant. However, the authors indicated that the incidence of each specific primary malignancy in the RFR group was similar to that in the literature for untreated rats of the same strain. They stated that: 'The finding here of excessive malignancies in the exposed animals is provocative; however, when this single finding is considered in light of other parameters evaluated, it is questionable if the statistical difference reflects a true biological activity.'

The authors also noted that, from the standpoint of carcinogenesis, benign neoplasms have considerable significance under the assumption that the initiation process is similar for both benign and malignant tumors. The fact that the RFR and sham groups showed no significant difference in the incidence of benign tumors is an important element in defining the promotion and induction potential of RFR for carcinogenesis.

The conclusion of the investigators was as follows: 'In summary, no defendable trends in altered longevity, cause of death, or spontaneous aging lesions and neoplasia can be identified in the rats exposed to this long-term low-level radiofrequency radiation exposure.'

38-10

The discussion of the Balcer-Kubiczek and Harrison (1991) in the fifth paragraph of page B-43 through the fifth paragraph on page B-44 of the Draft SEA has been replaced by the following:

Balcer-Kubiczek and Harrison (1991) studied whether exposure to RFR of mouse-embryo-fibroblast-cell cultures induces malignant transformation in such cells. They exposed such cultures for 24 hours to 2.45-GHz RFR (amplitude-modulated at 120 Hz) at an SAR of 0.1, 1, or 4.4 W/kg alone, or to the RFR at 4.4 W/kg before or after exposure to X-rays at 0.5, 1, or 1.5 Gy. Control cultures were sham-exposed. After such treatments, cultures with or without incubation with the tumor promoter 12-*O*-tetradecanoylphorbol-13-acetate (TPA) at 0.1 μg/ml were assayed for incidence of neoplastic transformations by counting the number of transformed foci in culture dishes.

The sham-exposed cultures exhibited low incidences of neoplastic transformation; those incubated with TPA showed a slightly higher mean incidence than those not incubated with TPA. A plot of mean neoplastic transformation incidence (linear scale) versus SAR (exponential scale) for the RFR-exposed cultures not incubated with TPA exhibited essentially no differences from sham-exposed cultures or any changes with increasing SAR, so the RFR alone did not promote transformation. However, the mean neoplastic transformation incidence rose with SAR for the RFR-exposed

cultures incubated with TPA. The authors regarded those results as indicating that RFR acts synergistically in a dose-dependent manner with TPA to promote neoplastic transformation.

In graphs of mean neoplastic transformation incidence versus X-ray dose (Figure 2 of the paper), the cultures not incubated with TPA showed a relatively small rise with X-ray dose (0, 0.5, 1.0, 1.5 Gy), independent of whether the cultures were exposed to RFR (at 4.4 W/kg) or sham-exposed. For the cultures incubated with TPA, however, the mean transformation incidence rose linearly with X-ray dose for those exposed to 4.4 W/kg, and also linearly for sham-exposed cultures, but the latter with about half as many mean incidences as the 4.4-W/kg cultures at corresponding X-ray doses. Thus, exposure at 4.4 W/kg of cultures treated with X-rays plus TPA appeared to increase neoplastic transformation incidence relative to sham-exposure, but the differences in incidence at corresponding X-ray doses were statistically significant only for 0 and 0.5 Gy.

For cultures not treated with X-rays, sham-exposure yielded low incidences of neoplastic transformation, with a slightly higher mean incidence for those incubated with TPA than those not incubated with TPA; thus, TPA alone (at the dose used) did not promote transformation. On the other hand, RFR-exposed cultures not incubated with TPA showed essentially no differences from sham-exposed cultures or any significant changes with increasing SAR; thus, RFR alone also did not promote transformation. However, for the RFR-exposed cultures incubated with TPA, the mean neoplastic transformation incidence rose with SAR, an indication that RFR acted synergistically in a dose-dependent manner with TPA to promote neoplastic transformation. The numerical results were as follows:

a. The researchers found 14 foci in 1494 dishes of sham-exposed cultures incubated with TPA, and 4 foci in 887 dishes of sham-exposed cultures not incubated with TPA.

b. The researchers found 48 foci in 704 dishes of cultures exposed at 4.4 W/kg incubated with TPA; only 4 foci were found in 800 dishes exposed at 4.4 W/kg not incubated with TPA.

The study was well-conceived and conducted meticulously, but a number of aspects of the study make interpretation of the findings difficult and uncertain. First, a plot of mean neoplastic transformation incidence versus SAR (Figure 1) showed an apparently linear rise of incidence with SAR (0.1, 1.0, 4.4 W/kg). That result can be misleading because, unlike what was done for the plots of incidence versus x-ray dose (Figure 2), in which linear scales were used for both variables, the authors used a linear scale for incidence and an exponential scale for SAR. If the three SAR points had been plotted on a linear scale also, the graph would have displayed a fivefold sharper rise with SAR between 0.1 and 1.0 W/kg than between 1.0 and 4.4 W/kg.

> The numbers of foci found relative to the numbers of dishes treated were small. The numbers of dishes used differed considerably for each treatment (see paragraphs a. and b. above). This point raises the question about whether the authors may have increased the number of dishes for each treatment until adequate percentages of foci for statistical analysis were obtained.

In response to the relevant EPA comment, the phrase above, "unlike what was done for the plots of incidence versus x-ray dose (Figure 2), in which linear scales were used for both variables," was omitted inadvertently. Thus, the intent was not so much to question the use of a logarithmic scale for SAR per se, but to ask why the authors did not use a linear dose-rate scale for RFR as they did for x-rays.

38-11

Healthy people are less able to bear high RFR levels at high ambient temperature and/or relative humidity than under normal conditions, and those with compromised thermoregulatory systems are even less capable. However, it is unlikely that, for those with compromised thermoregulatory systems, exposure to low RFR levels such as those from the WSR-88D radar will adversely affect their health. The added thermal burden from the RFR of the WSR-88D radar will be much less than the changes in metabolic rate produced by simple wakeful movements by such individuals.

To illustrate, the resting metabolic rate (RMR) for a normal adult male (20-24 years old) is about 1.3 W/kg, and the act of sitting upright increases the RMR to 1.5 W/kg (Durney et al., 1986, Table 10.3, p. 10.17). In addition, the RMR decreases only by about 0.1 W/kg from age 20 years to 80 years (Durney et al. 1986, Fig. 10.6, p. 10.19). The highest RFR average power density from the WSR-88D radar will be about 0.6 mW/cm^2 (at the surface of the radome), and the corresponding SAR for a person at that location would be about 0.03 W/kg, roughly 15% of the above movement-induced increase in RMR. For similar reasons, the RFR-induced increase in thermal burden for persons with compromised thermoregulatory systems (who are most unlikely to be next to the radome) would be vanishingly small. In this context, the work of Szmigielski is not relevant.

Gordon (1982) presented data on whole-body evaporative water loss (EWL) in mice exposed to 2.45-GHz RFR at 0-44 W/kg in an ambient temperature of 20°C as a measure of whole-body evaporative heat loss (EHL), and in unexposed mice maintained at 20, 25, 30, 33, and 35°C. From the data on the RFR-exposed mice, the author concluded that 29 W/kg of RFR is a threshold for loss of thermoregulatory ability by mice at 20°C, and from the data on the unexposed mice, he concluded that the threshold ambient temperature for loss of thermoregulatory ability in mice in the absence of RFR is 30°C.

Gordon (1982) also used published data on reported RFR-induced effects on thermoregulation and the endocrine system of various laboratory animals to predict similar effects in humans. From the results for several biological endpoints in various species studied at ambient temperatures in the range 20-30°C and mostly at 2.45 GHz, the author abstracted and plotted the minimum SAR reported to alter the specific physiological response studied versus the representative body mass of each species. The best fit was a regression line indicating the existence of an inverse relationship between the logarithm of threshold SAR and the logarithm of body weight. The author suggested that this inverse relationship could be used to extrapolate a

threshold SAR measured in a small mammal to a threshold SAR for humans. As an example, the author predicted that a threshold SAR of 29 W/kg in a 0.034-kg mouse would be equivalent to an SAR of 0.25 W/kg in a 70-kg man. However, the validity of extrapolation over such a large mass range is dubious.

Several problematic aspects of this research are addressed in the literature. For example, Adair et al. (1983) took issue with the basic premises of Gordon (1982), as well as with some of the numerical data. The Gordon paper (1982), the critique by Adair et al. (1983), a rebuttal by Gordon (1983), and a counter-rebuttal by Adair et al. (1984b) were discussed in Heynick (1987).

Although discussions of review papers were not included in the SEA, we offer the following comments on Gordon (1992):

1. The log-log graphs of SAR versus body mass for various animal species displayed in Figure 3 of the paper, like the similar graphs in Gordon (1992), were based on linear regression. A replot of the data points shown in that figure, specifically those for the threshold T_a = 30°C, yields a curve that resembles a rectangular hyperbola more than a straight line, which calls into question the validity of using linear regression. In addition, the dashed lines in Figure 3, representing extrapolation of body mass from about 4 W/kg to about 100 W/kg, is difficult to justify on a scientific basis – a criticism also applicable to Gordon (1992). That extrapolation implies that, for a 70-kg human, the threshold SAR for increasing the core temperature by 1°C at a T_a of 30°C would be about 0.02 W/kg. The replot of the data points suggested above yields a threshold SAR about an order of magnitude higher (assuming that such an extrapolation can be justified).

2. Regarding the MRI experiments with animals, unshorn sheep were used because their sizes approximate those of humans. However, as noted by the author, the thermoregulatory responses of sheep differ distinctly from those of humans. Thus, the findings may be of intrinsic interest, but their applicability to human MRI exposure is unclear.

3. The review in Gordon (1992) of MRI studies (by others) with human volunteers contains important data. However, because MRI techniques involve sequential scans of various cross-sections of the body, the term "whole-body SAR" differs in MRI usage from its usage in RFR-exposure guidelines (i.e., IEEE SCC 28) in which the entire body is exposed at the same time. Therefore, the comparisons of MRI findings on the basis of SAR with the SARs used in defining RFR-exposure guidelines may be misleading.

At issue regarding Gordon's animal experimental data on RFR-induced core-temperature increases is how valid are extrapolations from his findings to humans, especially given that the thermoregulatory responses of humans differ from those of various animal species, a point recognized in the EPA comment. In addition, extrapolations over such large ranges of body mass (e.g., from mice weighing a fraction of a kg) to 70 kg (for humans) is questionable without supporting experimental data in the upper part of that mass range, such as for non-human primates.

The result cited in this EPA comment (an SAR of about 0.1 W/kg for a 1°C rise in core temperature) appears to conflict with the extrapolated human threshold SAR (0.25 W/kg) predicted in Gordon (1982), discussed in the draft response.

Gordon's hypothesis about body surface area versus body mass may have some validity at RFR levels high enough to require the dissipation of considerable RFR-caused heat at the surface, but it is confounded by differences in the mechanisms for dissipating excessive heat among various species, as well as differences in surface configurations. At low RFR levels (such as those from the WSR-88D radar), Gordon's hypothesis is not applicable because the rate of heat generation by the RFR would be negligible relative to the metabolic heat generation occurring normally in the body. Thus, the Gordon papers are not relevant to human-health hazards of WSR-88D RFR.

Regarding the EPA comment on selecting one plot for discussion, note that all four plots were curved lines rather than the straight lines expected from a linear-regression analysis. To emphasize that point, the plot that showed the greatest curvature was selected for discussion.

38-12

The following discussion of Grundler et al. (1977) has been inserted after the second paragraph on page B-100 of the Draft SEA:

> Grundler et al. (1977) investigated the effects of RFR in the range 40-60 GHz on growth of yeast cells (*Saccharomyces cerevisiae*). After appropriate preparation, 2.5 ml of yeast suspension in an aqueous medium was placed in a standard rectangular glass receptacle, and cell growth was monitored by transmission photometry. Extinction (decrease of light transmission through the suspension) was recorded versus time and was found to increase exponentially over approximately three generations (about 4 hours). The data were replotted semilogarithmically to obtain the growth rate (slope of the new plot).
>
> Growth curves with no RFR were obtained at suspension temperatures in the range 30.5-34°C. (The authors noted that in any experiment, suspension temperature never varied by more than ±0.5°C.) The growth rates therefrom were plotted versus suspension temperature; these plots showed that the growth rate decreased from 4.0% per 1°C at 31°C to 1.3% at 33°C, and that the results were reproducible to within ±3%.
>
> The RFR source was a backward-wave oscillator in the 40-60 GHz range, with frequency stability and resettability, respectively, of ±1 MHz and 3 MHz (equivalent to ±25 and 75 parts per million at 40 GHz). The output of the oscillator was fed through a metallic waveguide to a vertical horn terminated by a Teflon structure that was immersed in the aqueous suspension. For depth of penetration into water of about 0.2 mm at 42 GHz, the RFR power emitted into the suspension ranged between 11 and 27 mW. Because the total area of emission was stated to be 10 cm^2, the power densities were in the range 1.1-2.7 mW/cm^2, which increased suspension temperature by roughly 0.4°C. The relative or normalized growth rate was then defined as the ratio of the growth

rate in the presence of RFR to the rate in its absence at the corresponding suspension temperature.

The authors performed 67 experiments; they discarded the results of 5 because the growth rates were very small in the samples studied and in a monitor beaker, probably because of bacterial infection or chemical poisoning. Those of the other 62 experiments were tabulated as normalized growth rate at specific frequencies, suspension temperatures, and absorbed RFR powers (which varied from 11 to 27 mW). The normalized growth rate was also plotted versus frequency. Evident in that figure were several sharp maxima and minima spanning unity growth rate ("a multiplet of biological resonances") in the frequency region 41.83-41.96 GHz, with little effect below or above the region. The largest increase in growth rate was 15% at 41.682 GHz ±3 MHz, for 17 mW absorbed power and 32.4°C suspension temperature. The largest decrease was 29% at 41.712 GHz ±3 MHz, for 23 mW and 31.7°C. (The figure showed one smaller minimum and maximum at intermediate frequencies.)

The authors noted that on an absolute frequency scale, a systematic offset of up to ± 20 MHz was possible. They also stated:

> 'The strong absorption of the radiation in water means that in our geometry only a small part of the total volume is subjected to the full intensity. We have not yet measured the dependence on intensity and thus would not know how to correct the results for the varying power. Inspection of the figure shows, however, that this can cause only minor alterations even if linear dependence on power is assumed.'

The results of this study seem to indicate that RFR in the resonant range either enhances or inhibits cell growth, depending on the specific frequency, but the authors did not speculate on possible mechanisms for such reversals of effect.

38-13

The sixth paragraph on page B-27 of the Draft SEA has been replaced by the following:

Kues and coworkers (Kues and Monahan, 1992; Kues et al., 1992) conducted a similar study, which also showed that 2.45-GHz pulsed RFR (10-μs pulses at 100 pps) caused corneal lesions at a lower average power density (10 mW/cm^2) than 2.45-GHz continuous wave RFR (20-30 mW/cm^2). They also found that pulsed RFR at 10 and 15 mW/cm^2 caused iris damage in the form of increased vascular leakage through the blood-aqueous barrier. Leakage apparently occurred at some time during the 4-hour exposures and persisted up to 72 hours postexposure. The use of horseradish peroxidase (HRP) as the tracer in subsequent histopathologic examinations of some eyes confirmed the occurrence of such vascular leakage.

The authors then studied the effects of the drugs timolol maleate and pilocarpine, administered just before RFR exposure, on RFR-induced iris vascular leakage. Both drugs are clinically used to treat glaucoma (abnormally high intraocular pressure). They were selected to test for possible thermal

mechanisms for the observed vascular leakage; timolol was chosen because it had been shown to protect the eye against heat-induced disruption of the blood-aqueous barrier, and pilocarpine was chosen because it is known to increase the permeability of the barrier to the tracer sodium fluorescein when the iris is heated excessively. Exposures were to 2.45-GHz pulsed RFR for 4 hours a day on 3 consecutive days at a specific average power density for each sequence: 0 (sham), 5, 10, or 15 mW/cm^2 with or without drug application just before exposure. The corresponding SARs in the eye were 0, 1.3, 2.6, and 3.9 W/kg.

The results of treatment were scored for each eye as follows: 1 for no vascular leakage, 2 for minor leakage of fluorescein in tissue and the anterior chamber, 3 for moderate leakage consisting of partial filling of the anterior chamber with fluorescein more than 5 minutes after tracer injection, and 4 for significant fluorescein leakage within the first 3 minutes after tracer injection.

With neither drug applied, the mean vascular-permeability score was 1.0 (no effect) for 5 mW/cm^2 as well as sham-exposure; the mean scores for 10 and 15 mW/cm^2 were 2.1 and 2.8, both significantly higher than for sham-exposure (by t-test) but not significantly different from each other. For timolol, the mean scores for 0, 5, 10, and 15 mW/cm^2 were respectively 1.0, 2.7, 2.7, and 3.5; the latter three scores were all significantly higher than for sham-exposure, but the scores for 5 and 10 mW/cm^2 were the same. The corresponding scores for pilocarpine were 1.0, 2.4, 2.8, and 4.0. Again, the latter three scores were significantly higher than for sham-exposure, but the scores for 5 and 10 mW/cm^2 did not differ significantly from one another. Thus, for each drug, a mathematical relationship between the RFR-drug response and power density is not readily discernible.

Similar comparisons, at fixed power densities, of mean scores for either drug versus its absence also seem ambiguous. At 5 mW/cm^2, for example, the mean scores for timolol and its absence (2.7 and 1.0 above) differed significantly, but those at 10 mW/cm^2 (2.7 and 2.1) and at 15 mW/cm^2 (3.5 and 2.8) did not. Analogous results were obtained for corneal endothelial damage.

To estimate the threshold power density for the effects with timolol pretreatment, the authors exposed primates at 0.2 mW/cm^2 (0.05 W/kg) and 1.0 mW/cm^2 (0.26 W/kg). The mean scores for vascular permeability were 1.0 and 2.9, with the latter score significantly higher than for sham-exposure. However, the mean score for 1.0 mW/cm^2 (2.9) was comparable to the mean score for 5 and 10 mW/cm^2 (both 2.7). This result led the authors to conclude that timolol pretreatment reduced the threshold power density by a factor of 10.

As in Kues et al. (1985), the use of the same animals in different aspects of the study is open to question. Also unclear in this and the previous study was the dosimetry: the SARs were determined from *in vivo* temperature measurements at the corneal epithelium, but the authors gave no indication

about the accuracy or reliability of those measurements. Another point not discussed was whether any significant differences occurred in results between the, presumably, equally treated left and right eyes of the subjects.

Regarding animal re-use, note that Mr. Kues, in his written statement dated August 4, 1992, to the Senate Committee on Government Affairs Ad Hoc Subcommittee on Consumer and Environmental Affairs, stated (*Ongoing Research*, page 4):

> During our earlier investigation into the effects of microwave radiation on the corneal endothelium, several primate eyes were removed at sacrifice for histopathological analysis. Preliminary analysis of the retiinas demonstrated photoreceptor change and retinal detachment. To date, a clear cause-effect relationship has not been established. The mixed exposure protocols of the primates, the use of halothane gas anesthesia during microwave exposure, and the possibility of pre-existing retinal pathology contributing to the effects must be addressed.

Mr. Kues also briefly discussed the measures being taken to avoid such confounding factors in their current research, such as restraining the primates and not anesthetizing them during irradiation, and the addition of ocular electrophysiologic testing to the ophthalmic examination. He then noted: "Both electrophysiological and retinal changes have been observed following microwave exposure; however, the data are preliminary in nature." When published, the new findings should be analyzed.

At a duty cycle of 0.001 (10-μs pulses at 100 pps), the peak power densities corresponding to 1, 5, 10, and 15 mW/cm^2 would be 1,000, 5,000, 10,000, and 15,000 mW/cm^2, respectively. The worst case peak power density for the WSR-88D radar would be 2,200 mW/cm^2, a level about double the 1,000-mW/cm^2 threshold reported above. However, the likelihood of exposure of humans to that level for durations of 4 hours, comparable to those used in this study, is negligible.

38-14

The methodology used by Frey et al. (1975) to seek possible alterations of the blood-brain barrier (BBB) by detecting the tracer fluorescein in thin brain slices after RFR- or sham-exposure was prone to artifact because not all regions of the brain have a BBB. Thus, brain slices of sham-exposed rats that included regions with no BBB would display the tracer fluorescein as well as similar slices from RFR-exposed rats. Frey et al. (1975) did report detection of the tracer in sham-exposed rats, thereby confounding the results. For that reason, replication of the study would serve no useful purpose.

In contrast, the study by Oscar and Hawkins (1977) was well conducted. However, their method of radioactive-tracer introduction, injection into the carotid in the form of a bolus, was seriously questioned because such rapid injection can alter the relative vascular and extra-vascular volumes in the brain, and thereby yield false findings of BBB alterations. As noted in the SEA, that method was later supplanted by that of Rapoport et al. (1979). Thus, "exact" replication of the study by Oscar and Hawkins (1977) would be pointless as well.

Scientific evidence for BBB alterations by either pulsed or continuous wave RFR (except possibly at hyperthermic levels) is lacking, given the negative findings of Preston and Préfontaine (1980) with pulsed RFR, who used the technique developed by Rapoport et al.

(1979); the similar negative findings by Gruenau et al. (1982) [with Oscar as a coauthor]; and the subsequent negative findings by others (e.g., Williams et al., 1984).

38-15

The fifth paragraph on page 41 of the Draft SEA has been replaced with the following:

> Lai et al. (1988) determined choline uptake (a measure of cholinergic activity) in various regions of the rat brain following exposure of rats to continuous wave or pulsed 2.45-GHz RFR in cylindrical waveguides (circularly polarized RFR) or in a miniature anechoic chamber (plane-wave RFR) at whole-body SARs of about 0.6 W/kg. Decreases of choline uptake were seen in the frontal cortex for exposure in either type of chamber, with no significant differences for pulsed and continuous wave RFR. The pulsed RFR in both types of chamber yielded decreases of choline uptake in the hippocampus, but not for the continuous wave RFR in either type of chamber – a difference possibly ascribable to RFR auditory effect. Decreases of choline uptake in the striatum were seen for both pulsed and continuous wave RFR in the anechoic chamber, but not for waveguide exposure. No significant changes of choline uptake were seen in the hypothalamus for any of the exposure conditions.

38-16

The following discussion of the study by Takashima et al. (1979) has been added after the sixth paragraph on page B-55 of the Draft SEA:

> Takashima et al. (1979) sought the possible effects of amplitude-modulated electric fields on the EEGs of rabbits. They exposed rabbits to frequencies in the range 1-30 MHz between two 30 x 30 cm aluminum plates spaced 20 cm apart at fields in the range 0.5-1 kV/m. A circuit for impedance matching inserted between the plates and the source was used to minimize reflected powers. As noted below, EEGs were recorded before, after, and in some experiments, during exposure, with stainless-steel electrodes chronically implanted along the midline in the central and posterior regions of the brain; scalp electrodes were used in other experiments.
>
> The EEG signals were fed to a preamplifier having a pass band of 3-100 Hz and a 60-Hz notch filter. In initial experiments, the authors found it difficult to interpret the unprocessed time-domain signals. In subsequent experiments, therefore, the signals were sampled at 5-ms intervals (1024 points), digitized, converted to complex spectra in the frequency domain by fast-Fourier-transform (FFT), and thence to power spectra. Smoothing the power spectra was required to resolve discrete frequency components, and sequential displays of the smoothed autocorrelation power spectra (usually 17 spectra at 3-min intervals) were examined for time-invariant features.
>
> Typical preexposure power-spectra sequences from anesthetized rabbits showed frequency components between 5 and 15 Hz that varied during each sequence, indicating the absence of a dominant component. The authors denoted such EEGs as "normal".

To determine the effects of short-term exposure, anesthetized animals were exposed once (acute exposure) to fields modulated at 60 Hz. In one set of experiments (5 animals), stainless-steel electrodes had been implanted in the brain and were allowed to remain in the cranial cavity during exposure. The sequential set of power spectra obtained following exposure showed a clustering of amplitude peaks in the range 2-5 Hz that persisted over the postexposure recording period (40-60 min). Reduction of high-frequency components was also noted. Similar patterns were seen with no modulation, but to a lesser extent.

In a second set of experiments (2 animals), the EEG electrodes were removed before exposure and reinserted after exposure. (It was not clear whether surgery was used for such removal and reinsertion, and if so, what effect it had.) The resulting power spectra were said to resemble normal EEGs as defined above; no clustering of spectral components was seen. Therefore, the EEG alterations in the first set of acute-exposure experiments were attributed by the authors to the local field created by the presence of metal electrodes in the cranial cavity.

To investigate the effects of chronic exposure, 4 unanaesthetized animals were exposed 2 h/d for 6 weeks to 1.2-MHz fields modulated at 15 Hz. EEGs were recorded every 2 weeks with silver electrodes placed directly on the skull before and after exposure. A sequential display of the power spectra taken after 4 weeks of exposure showed ordering of low-frequency spectral peaks and reduction of high-frequency components similar to the acute-exposure data taken with intracranial electrodes. The abnormal patterns began to appear after 2 to 3 weeks of exposure. Histograms were constructed from power-spectrum sequences derived from 4-week exposures and normal EEGs. The histogram for the exposed animals showed major peaks at 2 and 10 Hz, whereas the major peaks for normal EEGs were at 4.5, 8, and 11.5 Hz.

The authors assumed that the rabbit head (without the intracranial metal electrodes) could be modeled as a homogeneous conducting sphere immersed in a 10-MHz field of 500 V/m, and they calculated that the current density within the head was 0.082 mA/cm^2. Consequently, they regarded the positive results as a nonthermal effect. However, because they did not provide measurements or estimates of the SARs in the head, it is unclear whether the positive findings were thermal or nonthermal.

Even though enhancement of low-frequency components and increase in high-frequency activity after 3 weeks were reported, the data presented do not support this conclusion, and the authors themselves stated that the results presented were incomplete. However, they did note that for acute exposure, 'enhanced slow waves and unusually low high-frequency activities were due to the local field created by the presence of the metal electrodes in the cranial cavity.

In this study, the rabbits were used as their own control group, in that data obtained during and after exposure were compared with "normal"

(preexposure) data from the same group. However, the lack of a similarly treated sham-exposed group makes it difficult to assess whether the reported EEG changes were the result of exposure *per se*, or perhaps of adaptation to the repetitive aspects of the experimental procedures, such as handling and recording.

When power spectra for EEGs recorded at short time intervals (3 min) in a sequence are highly variable relative to one another, it is difficult to quantitatively assess the differences among the sequences. Autoregressive spectral estimation techniques may be more appropriate for analysis of EEG data than FFT techniques, because interval definition is problematic for nonstationary data. Qualitatively, nevertheless, the chronic-exposure data appear to show enhancement of low-frequency power-spectral components and reduction of high-frequency activities. On the other hand, comparison of data from the anesthetized animals used in the acute experiments with data from the unanesthetized animals used in the chronic experiments lacks analysis of the effects of anesthesia as a possible confounding factor. Moreover, it is not clear why the data taken after 4 weeks, and not after 2 and 6 weeks, of exposure were presented.

38-17

The following has been added as the second-to-last paragraph of Section 3.2 on page 62 of the Draft SEA:

> Blackman et al. (1988) exposed incubating chicken eggs continuously to electric fields at 10 V(rms)/m at either 50 or 60 Hz for 21 days. Within 1-1/2 days after hatching, the chicks were sacrificed, and their brains removed and tested for response to 50- or 60-Hz electromagnetic (EM) fields (at 15.9 V(rms)/m and 73 μT(rms) in a local geomagnetic field of 38 μT, 85°C, using Blackman's calcium-ion-efflux assay, which is described in detail in the following section (Section V.C.3.3). For eggs exposed to 60-Hz fields, the chicken brains showed a statistically significant higher calcium-ion efflux ratio when exposed to a 50-Hz EM field, but no difference when exposed to a 60-Hz EM field. For eggs exposed to 50-Hz fields, the chicken brains showed no differences in calcium-ion efflux ratios when exposed to 50- or 60-Hz EM fields. The authors commented that, 'It is unlikely that these data can be fully appreciated and extrapolated to other species before the underlying mechanism of action is established or a health consequence of an increased calcium efflux from brain tissue is identified.' Thus, these data do not support any suggestion that such exposures constitute a health hazard.

38-18

In NTIA (1981), details regarding signal frequencies, characteristics, irradiation durations, and average power densities or equivalent field intensities at various locations within and on the roof of the chancery were given. The signals were in the frequency range from 0.5 to 10 GHz. See also the response to Comment 1-1.

As discussed in the NTIA (1981) report, the RFR beams directed toward the U.S. Embassy in Moscow varied widely in character during the several calendar periods of irradiation, rendering it difficult to abstract exposure levels at frequencies close to those of the WSR-88D radar. An important point about the irradiations was that the higher exposure levels exceeded USSR exposure standards in force at that time for the general population, but were lower than the ANSI guidelines in force at that time. Discussion of the embassy incident was included in the SEA as general background information because of the claims of adverse health effects from the radiation, a subject covered in the study by Lilienfeld et al. (1978).

38-19

The use of the word "beneficial" is misleading. To clarify the point, the third and fourth sentences of the second paragraph on page 38 of the Draft SEA have been replaced by the following:

> Histologic examination of the 42 survivors of the 50 male mice exposed for 33 weeks showed that 9 (21%) were leukemic, whereas 11 of the 24 survivors (46%) of the 50 control mice were leukemic. However, the sample sizes were too small to ascribe any validity to that difference in percentages. It is not clear why about half of the control mice died, a possible indication that uncontrolled non-RFR factors were present.

38-20

See the response to Comment 38-10 above.

38-21

The analysis of the Milham (1983) study appeared in Heynick (1987), pp. 53-57; the Heynick report that was reviewed by its sponsor and was widely distributed to the RFR-bioeffects community and to others who requested copies.

The second through fourth full paragraphs on p. B-20 of the Draft SEA are replaced with the following paragraph:

> The statistic used by the author was the 'proportionate mortality rate [or ratio]' (PMR). By definition, the PMR for each cause of death is the ratio of the number of deaths for that cause to the number of deaths from all causes, expressed as a percentage; accordingly, the sum of all PMRs must equal 100. A more commonly used statistic is the 'standardized mortality ratio' (SMR) because it represents the percentage of actual deaths for each cause relative to the expected number of deaths from that cause, independent of any other SMR (Lilienfeld and Lilienfeld, 1980, pp. 78-80).

The following text is added after the first sentence on p. B-24 of the Draft SEA:

> The Science Advisory Board (SAB) of the EPA reviewed epidemiological studies on the possible link between cancer and exposure to EMFs and reached the following conclusion (EPA, 1992):
>
> > 'Currently available information is insufficient to conclude that the electric and magnetic fields are carcinogenic. Some human epidemiologic data report an association between surrogates for electric and magnetic field

> exposure and an increased incidence of some types of cancer, but the conclusion of causality is currently inappropriate because of limited evidence of an exposure response relationship and the lack of a clear understanding of biologic plausibility.
>
> 'Nonionizing electric and magnetic fields should not be classified under EPA's chemical carcinogenesis system because of present major uncertainties. These involve an incomplete understanding of which aspects of field-tissue interactions give rise to biologic effects.'

The British National Radiological Protection Board (NRPB), which advises the government of the United Kingdom on standards for protection for exposure to nonionizing radiation, has also reviewed the epidemiological evidence for a link between electromagnetic fields and cancer (NRPB, 1992). The NRPB states:

> Animal studies conducted at frequencies above about 100 kHz have provided some evidence for effects on tumour incidence but, for some data, indirect temperature-mediated effects could not be excluded. In other studies, an increased rate of *in-vitro* malignant transformation was demonstrated in a chromosomally abnormal cell line but the *in-vivo* implications of these findings remain uncertain. No epidemiological study has been carried out that would indicate whether exposure to such fields cause an increased risk of cancer in humans. It can be concluded that the evidence for a co-carcinogenic or tumour promoting effect of such relatively high frequency fields is not convincing.
>
> At low frequencies, the experimental evidence provides no reason to suggest that a carcinogenic effect of any sort is at all likely, except possibly for a small risk attributable to an effect on the secretion of melatonin. Some human observations have, however, been interpreted as implying that a variety of carcinogenic hazards might result from exposure at work, or, in relation to childhood cancers, exposure at home from proximity to power lines or the use of some electrical appliances, or exposure of a parent at work before the child was conceived.
>
> In summary, the epidemiological findings that have been reviewed provide no firm evidence of the existence of a carcinogenic hazard from exposure of parental gonads, the fetus, children, or adults to the extremely low frequency electromagnetic fields that might be associated with residence near major sources of electric supply, the use of electrical appliances, or work in the electrical, electronic, and telecommunications industry. Much of the evidence that has been cited is inconsistent, or derives from studies that have been inadequately controlled, and some is likely to have been distorted by bias against the reporting or publishing of negative results. The only finding that is at all notable is the consistency with which the least weak evidence relates to a small risk of brain tumours. This consistency is, however, less impressive than might appear as brain tumours in childhood and adult life are different in origin, arising from different types of cells.

38-22

The qualifications of the SEA preparers are given in Chapter VIII of the Draft SEA. The last two sentences of the fourth paragraph on page 68 of the Draft SEA have been replaced with the following:

> However, these epidemiologic studies (Jauchem and Merritt, 1991) have several weaknesses: (1) associations, when seen at all, are weak (risk ratios of 2-3 at most), numbers of cases are small, and positive results are frequently at the limits of statistical significance; (2) exposure estimates are crude, based either on job categories, surrogate measures such as wiring configuration, or on a limited number of field measurements; (3) potential exposures to other carcinogenic or genotoxic agents in occupational studies are not well characterized or controlled for; (4) dose-response relationships are generally not observed; and (5) weak statistical analysis and experimental design flaws characterize many of the studies.

38-23

The following paragraph has been added at the end of the fifth paragraph on page 54 of the Draft SEA:

> The foregoing maximum permissible exposure limits in areas accessible to the general public are meant to apply to the sum of the RFR levels from all sources, rather than to the levels from each source alone. Because the highest levels from the WSR-88D radar will be about 100 times lower than the lowest limit proposed in EPA (1986) and about 200 times lower than the NCRP (1986) limits, the emissions from the WSR-88D radar will not materially add to the emissions present in those areas from any other sources.

38-24

The total power density due to all sources is very unlikely to exceed the NCRP and IEEE limits. Because the power density of RFR decreases with distance from the transmitting antenna (at a rate of $1/r$ or $1/r^2$, depending on the type of antenna), the total power densities in space are almost everywhere dominated by the nearest transmitter. The ambient power density is many orders smaller than the designated limits, except at a limited number of locations centering on recognized transmitters.

As noted on page ii of the SEA, the 1976 EPA measurements in 15 cities yielded average values below 0.000,002 mW/cm^2. The Spectrum Engineering Division of the FAA measured the power density of background nonionizing radiation at Sayville, New York, about 50 miles east of New York City in March 1990. That study identified three main sources of RFR – FAA communications transmitters used for air traffic control, FM radio broadcasts, and television broadcasts. The highest power density measured was 0.000,265 mW/cm^2 at one of the 3 measurement locations. At the other 2 measurement locations, the highest power density was found to be less than 0.000,000,022 mW/cm^2.

It is instructive to consider the radiation from a 10-W CB hand-held transmitter. If nondirectional radiation is assumed, the distance at which the power density from such a device will drop to 2 mW/cm^2 (the upper limit of permissible exposure level in the IEEE (1991) guidelines for uncontrolled environments) can be found from the equation:

$$2 = 10{,}000/4\pi R^2 \text{ or}$$
$$R = (5000/4\pi)^{0.5} = 20 \text{ cm.}$$

Thus, individual transmitters typically contribute significant power densities only over very limited areas. As a more extreme example, consider a typical AM broadcasting tower that radiates 50,000 W at 1.0 MHz through a single vertical tower 100 m tall. Near the tower the power must pass through a cylindrical surface of radius R and 100 m = 10^4 cm tall. The power density (p) on this surface is given by:

$$p = 5 \times 10^7/(2\pi R \times 10^4) \text{ mW/cm}^2$$

The radius at which p = 2 mW/cm^2 is given by:

$$R = 5 \times 10^7/(4\pi \times 104) = 400 \text{ cm} = 4 \text{ m.}$$

The foregoing calculation is a rough approximation, but it indicates that RFR from most types of emitters is extremely local in nature.

Radars other than WSR-88Ds have focused power distribution patterns similar to that of the WSR-88D. They generate average power densities above 0.01 mW/cm^2 within very restricted areas near the radar antenna. Because the performance of the WSR-88D could be adversely affected by other nearby radars, steps are taken during the WSR-88D siting process to prevent siting near another radar. Government master files that show the locations of all fixed radars in the United States are consulted, and RFR levels at the proposed site are measured. Thus, the siting process for the NEXRAD program ensures that a WSR-88D will not be sited near another radar or other major RFR emitter. The overall conclusion drawn from these examples is that ambient RFR levels will add insignificantly to the RFR levels generated by the WSR-88D. Because the RFR levels generated by the WSR-88D are less than one-third of the permissible exposure level of the IEEE (1991) guidelines for uncontrolled environments, it is improbable that ambient RFR levels, when increased by the RFR generated by WSR-88D, will exceed those guidelines.

38-25

The Heynick (1987) document was critically reviewed by RFR-bioeffects researchers at the U.S. Air Force School of Aerospace Medicine before it was released for printing and distribution. It has served as the primary reference on RFR bioeffects for the EISs or EAs of a number of Air Force radar systems (e.g. PAVE PAWS, OTH-B) reviewed by interested members of the public and various governmental agencies, including EPA.

The report was cited in the SEA primarily as a reference for the RFR-bioeffects literature through about 1986, and EPA comments and the corresponding responses have corrected the errors noted, thereby enhancing its usefulness. The RFR-bioeffects sections of the SEA also

emphasize the studies done after that report was issued. Here again, comments by EPA have been considered carefully and appropriate changes have been made.

Regarding the EMP study, see the response to Comment 38-19 above. Also note that the last sentence on page 34 of the Draft SEA states "The findings of such flawed studies, whether positive or negative, cannot be regarded as strong evidence."

38-26

In the last sentence of the third paragraph on page 37 of the Draft SEA, "a few controversial epidemiologic studies ..." has been changed to "a growing number of epidemiologic studies ..."

The following sentences have been added at the end of that paragraph:

> However, most of these studies are subject to methodological and interpretative criticisms (Merritt and Jauchem, 1991). Despite this, they cannot be totally dismissed.

38-27

See the response to Comment 38-10 above.

38-28

See the response to Comment 38-2 above.

38-29

The last sentence of the fourth paragraph on page 64 of the Draft SEA has been deleted.

38-30

There is no contradiction. The quoted statement says "most of the papers," not all of them.

38-31

The third sentence of the fourth paragraph on page ii of the Draft SEA has been replaced by the following:

> At the lowest tower height, the average power density at ground level of RFR emitted by the WSR-88D will not exceed 0.005 mW/cm^2.

38-32

The last sentence of the second paragraph on page iii of the SEA has been deleted. See also the response to Comment 38-24.

38-33

The second sentence of the last full paragraph on page iii of the Draft SEA has been changed to the following:

> Underground power lines will generate negligible electric fields at ground level and magnetic fields at ground level that are comparable to those from above-ground power lines.

38-34

The siting of many WSR-88D units has yet to be determined at this time. For the overwhelming majority of units that have been sited, power lines exist along roads or utility rights-of-way (ROWs) very near the site, and only a short connection to the site is required. Because the strength of electric and magnetic fields decreases rapidly beyond the ROW, the installation of a power line connecting the site to an existing distribution line should not affect the levels of electric and magnetic fields experienced by nearby homes and businesses.

38-35

The second sentence of the first paragraph on page 25 of the Draft SEA has been deleted.

38-36

The title of Section C is meant to reflect that the findings of animal studies (as well as those involving humans) are being assessed for indications of possible effects on human health, as explained in Subsection 2 (see page 34 of the Draft SEA).

38-37

The following sentences have been added at the end of the fourth paragraph on page 52 of the Draft SEA:

> Exposure guidelines have been developed by private organizations such as the American National Standards Institute (ANSI), the National Council on Radiological Protection (NCRP) [now called the National Council on Radiation Protection and Measurements (NCRP)], and the American Conference of Governmental Industrial Hygienists (ACGIH) as voluntary guidelines for occupational or general-public exposure or both. In some instances, however, government agencies such as the Federal Communications Commission and various state and municipal bodies have adopted such guidelines or more stringent versions thereof as enforceable standards.

8-38

The section on Shock and Burn was placed near the sections on ocular effects and auditory effects because they all involve effects of RFR on sensory perception.

38-39

Section 3.4.2 of Appendix B and Section 2.8.2 in the SEA has been expanded considerably. The revisions to both sections are given below. Section 3.4.2 of Appendix B on pages B-50 through B-55 of the Draft SEA has been replaced by the following:

> 3.4.2 HISTOPATHOLOGY AND HISTOCHEMISTRY OF THE CENTRAL NERVOUS SYSTEM
>
> "Neural histopathologic and histochemical studies are, respectively, those of diseased or damaged neural tissues, and the chemical composition of such tissues. RFR effects have been sought *in vitro*, on preparations of neural tissues excised and kept alive in appropriate solutions while undergoing RFR exposure, and *in vivo*, from exposure of live animals.

3.4.2.1 HISTOLOGICAL AND HISTOCHEMICAL STUDIES *IN VITRO*

In several histological studies, preparations of neural tissue were exposed to RFR *in vitro*. Courtney et al. (1975) excised superior cervical ganglia from white rabbits, stretched them singly across a vertical section of waveguide sealed at the bottom with a quarter-wave dielectric plate and filled with Ringer's solution, and exposed each ganglion to 2.45-GHz CW RFR from below. The calculated normalized SAR of the ganglion was 2.2 W/kg per mW/cm^2. The preganglionic end was connected to a set of stimulating electrodes outside the waveguide, and contact with the postganglionic end was made via a glass capillary through a wall of the waveguide. Ringer's solution at 37°C was pumped through the waveguide section.

Each ganglion was exposed to RFR at power densities up to about 300 mW/cm^2 (660 W/kg) for 1-minute intervals, with 1 minute between exposures for control measurements. The authors noted that only above about 100 W/kg did the temperature rise of the Ringer's solution exiting the waveguide exceed 0.1°C. During the exposure and control intervals, the ganglion was stimulated with 100-300 μs pulses at 1 pps, and the response latencies for synaptic transmission of the B fiber (short latency) and the C fiber (long latency) were determined. No significant changes occurred in the mean response latencies from the RFR at SARs up to 660 W/kg.

Chou and Guy (1978) similarly studied the rabbit vagus nerve and the cat saphenous nerve, as well as the rabbit superior cervical ganglion. As in Courtney et al. (1975), each ganglion or vagus nerve was mounted within a vertical waveguide section (parallel or perpendicular to the E-vector) through which Ringer's solution was pumped. The solution temperature at the fluid outlet of the waveguide was held constant at 37 ±0.02°C during exposure to the RFR. At the high RFR levels, however, the fluid temperature at the center of the specimen (measured with a nonperturbing probe) rose by as much as 1°C because of the limited circulation rate of the fluid pump.

Each specimen was stimulated with a 0.3-ms current pulse of 0.3-30 mA at 2-s intervals before, during, and after exposure to RFR. The compound action potentials (CAPs) were recorded, and their conduction velocity and amplitude were determined. Vagus nerves were exposed to 1-μs pulses of 2.45-GHz RFR at 1000 pps or 10-μs pulses at 100 pps for 10-minute periods at average SARs of 0.3, 3, 30, and 220 W/kg or to CW RFR at the same SARs, with 5 minutes between exposures. Superior cervical ganglia were exposed for only 5-minute periods because of their shorter lifetimes. Specimens were also exposed to pulsed RFR at 220 W/kg average (220 kW/kg peak) and to CW RFR at 1500 W/kg with no current-pulse stimulation, to test for the possibility of direct RFR stimulation.

No direct stimulatory effects of RFR exposure were observed at the highest available SARs (pulsed RFR at 220 kW/kg peak or CW RFR at 1500 W/kg) in the absence of stimulation by current pulses. With electrical stimulation, no changes in amplitude or conduction velocity of the CAPs were seen for

exposures of rabbit superior cervical ganglia or vagus nerves to pulsed or CW RFR at SARs that did not increase the fluid temperature near the center of the specimen.

The conduction velocity and peak CAP amplitude versus time was shown for a representative cat saphenous nerve exposed to the CW RFR at 1500 W/kg, an SAR that increased the fluid temperature by 1°C. The conduction velocity rose by about 2% during exposure, a result reproduced by raising the temperature of the solution 1°C by non-RFR means. The variations in peak CAP amplitude were small and apparently not RFR-dependent. Similar data were shown for a representative stimulated rabbit vagus nerve before, during, and after exposure to 10-μs pulses (100 pps) at 220 kW/kg peak, and to CW RFR at 1500 W/kg. For the pulsed RFR, the fluid temperature rose 0.3°C, which increased the conduction velocity slightly (from 117 to 118 m/s). For the CW RFR, the fluid temperature rose by 1°C, which increased the conduction velocity from 117 to 135 m/s. An equivalent rise in fluid temperature by non-RFR means yielded the same velocity increase.

CAP recordings for a rabbit superior cervical ganglion exposed to 1-μs pulses (1000 pps) and to CW RFR at the same SARs as the vagus nerve were also made. For the pulsed RFR, the fluid-temperature rise was again 0.3°C, but the latency time remained unchanged. The rise in fluid temperature by the CW RFR was again 1°C, and the latency time decreased to 16 ms, a result reproduced by raising the fluid temperature 1°C by non-RFR means.

Among the histochemical effects sought *in vitro* were RFR-induced alterations in the activities of the enzymes acetylcholinesterase (AChE) and creatine phosphokinase (CPK) in rabbit blood. Olcerst and Rabinowitz (1978) found that 2.45-GHz RFR significantly decreased AChE activity, but only at a level sufficient to denature AChE (about 125 mW/cm^2). Galvin et al. (1981c) observed that 2.45-GHz RFR did not affect the activity of either AChE or CPK at SARs up to 100 W/kg.

3.4.2.2 HISTOLOGICAL AND HISTOCHEMICAL STUDIES *IN VIVO*

Albert et al. (1981) indicated that prenatal development of the mammalian brain depends on migration of nerve cells, and specifically that a readily identifiable cell class in the cerebellum called Purkinje cells arises during the second half of gestation. They sought possible effects of prenatal and postnatal exposure to RFR of rats on such cells.

In 1 experiment, 2 groups of 3 pregnant rats each were exposed to 2.45-GHz RFR at 10 mW/cm^2 for 5 days starting on gestation day 17. The mean SAR was estimated as about 2 W/kg, with a range 0.8 to 6 W/kg due to rat movements. Six other rats were sham-exposed. Of the 6 pups in each group, 3 were euthanized on postnatal day 1, and the other 3 of each group were euthanized 40 days after birth. The cerebella from the day-1 pups were not mature enough for clear discernment of the Purkinje cells, but the mean Purkinje cell counts for the RFR-exposed 40-day pups were much lower than

for the sham-exposed 40-day pups. The latter result was taken as indicating the permanence of this prenatal-exposure effect.

In another experiment, 1 pup each of 6 pairs of litter mates was exposed to RFR for 5 days, 7 h/d, whereas the other pup was sham-exposed. Three pups of each group were euthanized immediately, and the other three of each group were euthanized 40 days later. In the groups euthanized immediately, the mean Purkinje-cell count for the rat pups exposed to the RFR was significantly lower than for those sham-exposed, but the mean counts for the 40-day RFR-exposed and sham-exposed pups did not differ significantly. The authors regarded those results as indicating the reversibility of the effect.

The statistical validity of the positive results above is open to question because of the large SAR variations noted by the authors. They also conducted a similar study on squirrel monkeys that previously had been exposed perinatally elsewhere, which yielded no significant differences between RFR- and sham-exposed groups in whole-body mass, brain mass or volume, or in counts of Purkinje cells.

Merritt and Frazer (1975) exposed mice for 10 minutes to 19-MHz RFR consisting of either an electric field at 6 kV/m with associated magnetic field of 6.4 A/m, or a magnetic field at 41 A/m with 2 kV/m associated electric field; these exposures did not increase rectal temperature. The objective was to determine whether such fields can alter the levels of specific neurotransmitters in the mouse brain.

Fifteen minutes after the 19-MHz exposure, the head of each mouse was exposed to intense microwave RFR to quickly inactivate its brain enzymes – a method that produced a rise in brain temperature of 40 to 50°C in 1 second. Sham-exposed mice were similarly treated. After such inactivation, the brains were assayed for the levels of serotonin (5HT) and its metabolite 5-hydroxyindole acetic acid (5HIAA), dopamine (DA) and its metabolite homovanillic acid (HVA), and norepinephrine (NE). No significant differences were found between the sham-exposed controls and those exposed to either the E-field or H-field in the mean whole-brain concentrations for any of the neurotransmitters or their metabolites.

Sanders et al. (1980) investigated the hypothesis whether exposure of brain tissue to RFR *in vivo* results in inhibition of the respiratory chain function, followed by decreases in concentrations of adenosine triphosphate (ATP) and creatine phosphate (CP). The authors noted that reduction of nicotinamide adenine dinucleotide (NAD) to NADH can be monitored *in situ* continuously by fluorescence measurements. Thus, if RFR exposure stresses the cells that inhibit respiratory chain function or cell functions that use ATP and CP, the NADH level will increase.

Rats were anesthetized, and a small aperture was made through the skull. The head of the rat was held rigidly in place, the light from a 366-nm excitation source was focused on a small spot on the cerebral cortex, and a fiber-optic

bundle was directed toward the focal spot. The other end of the fiber-optic bundle was terminated at a wheel that housed a 460-nm filter to permit measurements of NADH fluorescence at that wavelength, and a 366-nm filter for reflectance measurements of the incident beam from the brain surface.

After preparation, each rat was exposed to 591-MHz CW RFR in the far field of a horn. The radiation pattern of the horn was such that only the head of the rat was exposed. Maximum SARs at the surface of a 2.0-cm diameter sphere and a semi-infinite plane of brain tissue were estimated to be 0.026 and 0.16 W/kg per mW/cm^2, respectively.

In a baseline experiment, after NADH fluorescence remained steady for 5 minutes in the absence of RFR, exposure to 591-MHz RFR was begun for 5 minutes at 13.8 mW/cm^2. In a representative graph of fluorescence, the intensity of the trace began to rise on RFR initiation. It reached a maximum of 12.5% above baseline level at 30 seconds, showed a compensatory partial return toward baseline during the next 2.5 minutes, and than a slow rise during the remaining 2 minutes. The authors noted that the 30-s maxima for the rats tested were in the range 4% to 12.5% above baseline.

Groups of rats were then sham-exposed or exposed at 13.8 mW/cm^2 for 0.5, 1, 2, 3, or 5 minutes. Immediately after exposure, the head and neck of each rat were immersed in liquid nitrogen and the frozen head was removed and stored in liquid nitrogen. Subsequently, the frozen cerebral hemisphere near the aperture in the skull was extracted, pulverized, and assayed for ATP and CP. Other rats were sham-exposed or exposed at 5.0 mW/cm^2 for 0.5 or 1 minute and similarly processed.

Mean ATP and CP concentrations as percentages of baseline levels and their standard errors were graphed versus exposure duration. Sham-exposures yielded no statistically significant percentage changes in either mean ATP or mean CP level relative to baseline. For 13.8 mW/cm^2, however, 30-s exposures yielded the lowest mean ATP level – about 75% of baseline. For the longer exposures, the mean ATP levels were higher but did not exceed about 90% of baseline. The 30-se RFR exposures also yielded the lowest mean CP level – about 60% of baseline. The 3-minute exposures yielded a relative maximum of 85% of baseline and the 5-minute exposures showed a decrease to 60% again.

All of the percentage differences in mean ATP and CP between RFR and sham groups at corresponding times were significant, with most at the $p<0.005$ level. In particular, the mean ATP and CP levels for exposure at 5 or 13.8 mW/cm^2, each for 0.5 and 1 minute, differed significantly from their corresponding sham-exposure levels. For each duration, however, the difference in results between the two RFR levels was nonsignificant; these findings indicate that the effect may not be dose-dependent, or that it saturates below 5 mW/cm^2.

Temperatures at depths of 2-3 mm below the brain's surface were measured with a thermistor placed adjacent to the focal spot of the excitation light, with the leads at right angles to the electric vector. Rats other than those used in the NADH, ATP, and CP assays were exposed at 0, 13.8, 18.0, 30.0, 40.0, and 47.0 mW/cm^2 for 5 minutes. With sham-exposure, heat loss through the aperture in the skull was found to cause a decrease in brain temperature of 0.7°C at the end of the period. At 13.8 mW/cm^2, a decrease of 0.6°C was observed at exposure end; that value is close to that for sham-exposure, presumably an indication that the RFR at that level had added little heat. Decreases of 0.5 and 0.1°C were observed at 18.0 and 30.0 mW/cm^2, respectively, and increases of 0.2 and 0.1°C were obtained at 40.0 and 47.0 mW/cm^2. Rectal temperatures remained constant at all exposure levels.

From those results, the authors concluded that the observed changes in mean ATP and CP levels at 5 or 13.8 mW/cm^2 could not be ascribed to general tissue hyperthermia (but they did not exclude local hyperthermia). Instead, the data support the hypothesis that RFR inhibits electron transport chain function in brain mitochondria, thereby decreasing brain energy levels.

In another study, Sanders and Joines (1984) investigated the effects of hyperthermia alone and in conjunction with RFR. In the hyperthermia-only experiments, brain temperatures of sham-exposed rats maintained at 35.6°C yielded mean ATP and CP concentrations that did not differ significantly from those for sham-exposure reported in Sanders et al. (1980). Groups were then sham-exposed with brain temperatures held at 37.0, 39.0, or 41.0°C, and the mean ATP and CP concentrations versus brain temperature were plotted as percentages of the concentrations at 35.6°C. The percentages of both ATP and CP declined steadily with increases in brain temperature, with the CP rate of decline higher. At 39°C, ATP and CP decreased to about 90% and 70%, respectively; at 41°C, the decreases were to about 70% and 45%, respectively.

Rats were exposed at 13.8 mW/cm^2 for 0.5 to 5 minutes while their brain temperatures were held at 35.6°C, and the percentage increase in NADH and mean percentages of ATP and CP (relative to the concentrations at 35.6°C for sham-exposed rats) were plotted versus exposure duration. The results for 30-s exposures showed the largest decreases – to about 75% for ATP and to about 60% for CP. A similar set of experiments was conducted with brain temperatures held at 39.0°C before and during RFR exposure (RFR plus hyperthermia). At 0 minutes of RFR, the ATP and CP levels were respectively about 90% and 70%, with these decreases ascribed to the hyperthermia; the levels declined further to minima of about 60% and 45% for 1-minute RFR-exposures. For 5-minute exposures, the ATP level rose to about 80% and the CP level rose to about 50%. The general conclusion of the authors was:

> The decreases in ATP and CP in the 39°C brain during microwave exposure were significant and resulted in ATP and CP being much lower than observed at 35.6°C. Thus, at 39°C when the brain metabolic rate was increased, subsequent microwave exposure rapidly induced further

> decreases in ATP and CP, similar to the 35.6°C microwave exposure data, i.e., without a further increase in brain temperature; [these results] are consistent with the concept of direct microwave inhibition of energy metabolism.

The latter statement seems open to question, however, because an estimated local SAR of 8.5 W/kg at 13.8 mW/cm^2 must have produced considerable local heating, even though the mean brain temperature was held constant by external means.

Sanders et al. (1984) performed similar experiments, but at 200 MHz and 2,450 MHz as well as at 591 MHz. Local SARs in the brain of a dead rat were determined by measuring temperature rise versus time with a Vitek isotropic probe during exposure of the carcass to each frequency at 60 and 100 mW/cm^2. The normalized SARs at 200, 591, and 2,450 MHz were 0.046, 0.185, and 0.368 W/kg per mW/cm^2.

For the NADH fluorescence part of the study, a group of 6 rats was exposed at each RFR frequency. Each rat was given 2-minute exposures at increasing power densities in the range 0.5 to 20.0 mW/cm^2 (with 2 rats also given 40.0 mW/cm^2), and its NADH fluorescence was measured after each exposure. NADH fluorescence returned to baseline between exposures.

Because the differences in NADH results among the 6 rats of each group were large, their data were not combined for statistical treatment. Instead, the percentage changes in NADH versus power density were tabulated for each rat, with each serving as its own control. The 200-MHz data for each rat showed steady increases in NADH with power density, indicative of a dose-response relationship, with a trend toward saturation at the higher power densities. The results for 591 MHz were similar. However, no NADH changes were detected for 2,450 MHz, from which the authors suggested that the effect is frequency-dependent.

Whole-brain mean assays of ATP and CP for exposure to 200, 591, and 2,450 MHz at 13.8 mW/cm^2 versus exposure duration were shown graphically as percentages of baseline values. From the previously cited normalized SARs at the 3 frequencies, the SARs were about 0.6, 2.6, and 5.1 W/kg, respectively. The largest effects were for 30-second exposures to either 200 MHz or 591 MHz; the levels of ATP decreased to about 80% and 75% of baseline for the two frequencies, respectively, and rose for exposures of longer durations. The mean CP assays showed no significant changes for 200 MHz, but a decrease to about 60% of baseline for 30-second exposures. No significant changes in either ATP or CP were seen at 2,450 MHz.

Given the negative findings at 2.45 GHz, the effect above is unlikely to be of importance relative to the RFR from the WSR-88D radar.

Lai et al. (1988) studied the results of single, 45-minute exposures of rats to 2.45-GHz CW or pulsed RFR (2-μs pulses at 500 pps) on choline uptake in several regions of the brain, with the uptake a measure of cholinergic activity.

Exposures were done in groups of four individual cylindrical waveguide chambers (Guy et al., 1979) to circularly polarized RFR, or in a miniature anechoic exposure chamber (Guy, 1979) to plane-polarized RFR. The exposure levels were set to yield a whole-body SAR of 0.6 W/kg (1.0 or 2.07 mW/cm^2, respectively). Control rats were sham-exposed in the same chambers.

A dosimetry study by Chou et al. (1985) indicates that 0.6-W/kg whole-body SAR in a cylindrical waveguide yielded a spatial mean SAR in the head of 0.77 W/kg with the rat facing the RFR source and 0.91 W/kg with the tail facing the source. On the other hand, for a rat exposed dorsally in the anechoic chamber from above (with the top of its body toward the source) and its body axis parallel to the electric vector, the spatial mean SAR in the head corresponding to 0.6-W/kg whole-body SAR is 0.56 W/kg.

All four exposure conditions yielded decreases in choline uptake in the frontal cortex, with no significant differences between the CW and pulsed RFR. Decreases in choline uptake were seen in the hippocampus for exposures to the pulsed RFR in both types of chamber, but not to the CW RFR in either type of chamber. Choline uptake decreased in the striatum from exposure to pulsed or CW RFR in the anechoic chamber, but not in the waveguide chambers. On the other hand, no significant changes in choline uptake in the hypothalamus were seen for any of the exposure situations.

The authors suggested that the differences in results for the CW and pulsed RFR could be ascribed to the RFR auditory effect, noting that the characteristics of the pulsed RFR used were above the threshold for that effect. They did not suggest specific mechanisms for the other results.

The second through sixth paragraphs on page 41 of the Draft SEA have been replaced by the following:

Albert et al. (1981) had reported lower mean counts of Purkinje cells in 40-day pups RFR-exposed for 5 days *in utero* to 2.45-GHz RFR at 10 mW/cm^2 relative to counts for sham-exposed pups. These results are open to question because of the large variations in SAR (0.8 to 6 W/kg, 2 W/kg estimated mean) due to movement of the dams during exposure. In addition, rat pups exposed to the RFR and euthanized 40 days later did not show such differences, and little credence can be given to their results for rat pups euthanized immediately after exposure because of Purkinje cell immaturity in neonates – a point previously mentioned by the authors. Moreover, in a similar study they conducted using squirrel monkeys that had been previously exposed perinatally elsewhere, they found no significant differences between RFR-exposed and sham-exposed monkeys in Purkinje cell counts.

Merritt and Frazer (1975) obtained negative findings in mean brain concentrations of various neurotransmitters and their metabolites assayed in mice exposed to predominantly electric or magnetic fields at 19 MHz.

However, the authors noted that at 19 MHz, a mouse absorbs very little energy from either field.

Sanders and coworkers (1985) observed increases in NADH fluorescence and reductions of ATP and CP concentrations in the rat brain by RFR at levels characterized as not producing measurable brain hyperthermia. Although some points are open to question, those positive findings appear to be valid and are worthy of further study. The experiments were performed on anesthetized rats, with consequent lowering of brain temperatures. Whether similar results would occur in the absence of anesthesia has not been determined. It is noteworthy that the effects were higher at 591 MHz than at 200 MHz and that they were not observed at 2.45 GHz; these findings are suggestive of dependence on RFR frequency.

A study by Lai et al. (1988) with 2.45-GHz CW and pulsed RFR at whole-body SARs of about 0.6 W/kg on choline uptake in various regions of the rat brain yielded both positive and negative results. However, results for the two kinds of exposure chamber and between pulsed and CW RFR were apparently inconsistent.

In summary, histopathologic and histochemical changes in the central nervous system were seen at relatively low SARs by Sanders and coworkers (1985) and Lai et al. (1988), but their significance with regard to possible human health hazards is not clear and needs further investigation. Taken collectively with other experimental results in which the effects observed were ascribed to local increases in brain temperature, it seems unlikely that exposure to RFR levels that do not increase local brain temperatures would cause deleterious histopathologic or histochemical effects in the human central nervous system.

38-40

The comment is well taken; the University of Washington study is discussed in proper order in Appendix B (page B-40+), and the appropriate sections of the main body of the Draft SEA have been revised similarly. The third paragraph on p. 39 of the Draft SEA is replaced with the following:

> In a comprehensive study by the University of Washington on health and longevity, 100 rats were exposed to 2.45 GHz RFR at 0.5 mW/cm^2 for virtually their entire lifetimes (except those withdrawn for interim tests and those that expired before the end of the exposure regimen), and 100 rats were concurrently sham-exposed. The findings on immunology and hematology are summarized in Section 2.9. Regarding longevity and cancer, the authors concluded that 'no defendable trends in altered longevity, cause of death, or spontaneous aging lesions and neoplasia can be identified in the rats exposed to this long-term low-level radiofrequency exposure' (Guy et al. 1985).

38-41

The third sentence of the first paragraph on page 44 of the Draft SEA – "There are scientifically credible…human exposure to RFR." – does not reflect the more detailed discussion

of the topic in Section 3.6.1 of Appendix B, and has been deleted. In addition, the next sentence, "This finding...any other species." has been revised to read:

> Especially noteworthy are the results for primates because of their far greater similarities to humans than the other animals studied.

The following discussion of Lotz (1985) has been inserted after the sixth paragraph on page B-73 of the Draft SEA:

> Lotz (1985) exposed 5 rhesus monkeys to 225-MHz RFR in the E-orientation (near whole-body resonance) at levels in the range 1.2-15.0 mW/cm^2 (0.8-10.2 W/kg) in an anechoic chamber in 4-hour day and night sessions. The monkeys were also exposed in the H-orientation at 5 mW/cm^2 (1.2 W/kg), and to 1.29-GHz RFR (a frequency well above resonance) at 20, 28, and 38 mW/cm^2 (2.9, 4.0, and 5.4 W/kg). Rectal temperatures were monitored continuously during every session, and blood samples for cortisol analysis were taken hourly during the 225-MHz E-orientation sessions. The criterion for tolerance of RFR was defined as a rectal temperature not exceeding 41.5°C.
>
> Average rectal-temperature increases for exposures to 225-MHz RFR in the E-orientation at 2.5 and 5.0 mW/cm^2 (1.7 and 3.4 W/kg) were 0.4 and 1.7°C, but the monkeys could not tolerate exposure without rectal temperature exceeding the prescribed maximum to 225-MHz RFR in the E-orientation at 7.5 mW/cm^2 (5.1 W/kg) or higher for more than 90 minutes. No changes were observed in circulating cortisol levels for exposures at 5 mW/cm^2 (3.4 W/kg) or less. The exposures to 225-MHz RFR at 5 mW/cm^2 in the E-orientation (3.4 W/kg) and the H-orientation (1.2 W/kg) produced mean rectal-temperature increases of 2.1 and 0.2°C, respectively. The mean rectal-temperature rises for exposures to 1.29-GHz RFR at 20, 28, and 38 mW/cm^2 (2.9, 4.0, and 5.4 W/kg) were 0.4, 0.7, and 1.3°C. These results confirmed that RFR exposure near resonance is most effective for producing hyperthermia.

Inclusion of a section on lethality studies, though perhaps of general interest, did not seem appropriate in an environmental assessment for a system that would emit low levels of RFR.

In the third to last sentence of the first paragraph on page iii of the Draft SEA, the phrase: "the so-called 'non-thermal' levels of RFR" has been deleted.

Regarding the studies by Gordon and coworkers, see response to Comment 38-11.

See also the response to Comment 31-1.

38-42

In the second sentence of the last paragraph on page 52 of the Draft SEA, the phrase "nor will it exceed the proposed, but not implemented, EPA standard." has been changed to

> nor will it exceed the lowest of the proposed, but not implemented, EPA limits.

38-43

In the fourth sentence of the fourth paragraph on page 54 of the Draft SEA, the phrase "Although some favored..." has been deleted.

38-44

The first sentence of the last paragraph on page 55 of the Draft SEA has been changed to the following:

> The physics of the interaction of E- and H-fields with conductive objects having a complex dielectric constant is well understood, but the biological mechanisms of interaction of such fields with entities such as living cells and tissues are the subject of current research.

38-45

The final sentence of the first paragraph on page 57 of the Draft SEA has been replaced by the following:

> Third, Soviet workers were entitled to protection from any and all workplace hazards, even if the symptoms might be judged psychosomatic by Western standards (Krylov and Yuchenkova, 1973; Gordon, 1966; and Umansky 1989).

38-46

The last sentence of the second paragraph on page 62 of the Draft SEA has been changed to the following:

> Some of these effects may have resulted from low-level stress as a result of external sensory stimulation induced by indirect mechanical interaction of the fields with skin and hair (Weigel et al., 1987; Miller, 1986).

38-47

The suggested correction has been made.

38-48

The first two sentences of the fifth paragraph on page B-4 of the Draft SEA have been deleted.

38-49

The phrase in the first sentence on page B-24 of the Draft SEA "a recommendation similar to an earlier one by EPA's Science Advisory Board (SAB)" has been deleted, and the following paragraph has been added at the end of that paragraph:

> EPA's Radiation Advisory Committee of the Science Advisory Board established a subcommittee on Nonionizing Electric and Magnetic Fields to review the draft report. That subcommittee has issued a report, SAB (1992), indicating its findings. The subcommittee suggested numerous changes in emphasis, coverage, and wording, and concluded that the draft report [EPA, 1990] will have to be rewritten to accommodate all of the suggestions and comments.

38-50

In the first sentence of the seventh paragraph on page B-70 of the Draft SEA, the phrase "periodic test sessions" has been changed to "periodic behavioral test sessions".

38-51

The SEA is organized into a main text and technical appendices to minimize the amount of technical discussion in the main body of the document.

38-52

The standard for RFR emissions from new microwave ovens is 1 mW/cm^2 at a distance of 5 cm. That level is permitted to rise to 5 mW/cm^2 during the lifetime of the oven as a result normal wear of the appliance. (Food and Drug Administration, 21 CFR 1030). A person at a distance of 6 ft from a microwave oven that emits 1 mW/cm^2 would receive whole body exposure of about 0.5 $\mu W/cm^2$, which is similar to the exposure level for a person at ground level about 1,000 ft from a WSR-88D.

38-53

The suggested correction has been made.

38-54

The suggested correction has been made.

38-55

The suggested correction has been made.

38-56

The first line on page 55 of the Draft SEA has been deleted.

38-57

The phrase "at ground level" at the end of the fourth paragraph on page 55 of the Draft SEA has been deleted.

38-58

The last sentence on page 40 of the Draft SEA has been deleted.

38-59

The first word - "showed" - on page 63 of the Draft SEA has been deleted.

38-60

The first two lines at the top of page 63 have been deleted.

38-61

The first paragraph of Section 3.4 on page 64 of the Draft SEA has been replaced with the following:

> The number of epidemiological studies of power line field effects in humans is growing. Approximately half of these report positive findings for and

association between some indicator or estimate of the presence of electric and magnetic fields and the increased incidence of several types of cancer, or of other biological endpoints including outcomes of pregnancy after use of electric blankets, infertility of exposed male workers, suicides, and measures of general health. The other half do not. Several these studies are discussed below. However, most if not all of them have recognized weaknesses (Connecticut Academy of Science and Engineering, 1992; Joachem and Merritt, 1991), making interpretation difficult. In these circumstances, the positive findings may be said to be suggestive of an effect, but do not prove that it does exist. Likewise, negative studies cannot prove that it does not exist. Acceptance of the positive findings of epidemiological studies requires the concurrent demonstration of scientifically acceptable mechanisms (e.g., for carcinogenesis, and much of the latter is currently speculative for EMFs). The most that can be said at the present is that more research is needed.

38-62

The suggested correction has been made.

38-63

The first three sentences of the second paragraph on page B-16 of the Draft SEA have been replaced by the following:

> Robinette and Silverman (1977) chose 19,965 men who had served in the Navy during the Korean War who from their titles (electronics technician, fire control technician, or aircraft electronics technician) were regarded as repair technicians for electronic equipment and thus were assumed to have had occupational exposure to RFR. For the control group, the authors selected 20,726 Naval men considered operators of electronic equipment (titles: radioman, radarman, or aircraft electrician's mate) and were assumed to have had little occupational exposure to RFR.

38-64

The suggested correction has been made.

38-65

The error referred to by the comment author does not occur on the cited page.

38-66

The suggested correction has been made.

38-67

See the response to Comment 38-13 above. During preparation of the Draft SEA, a search of the Biological Effects of Nonionizing Electromagnetic (BENER) database by Information Ventures, Inc. for published papers on the work of Kues and colleagues yielded only the Kues (1985) paper; the other citations made there were abstracts. A discussion of Kues and Monahan (1992) and Kues, et al. (1992) has been added to the SEA text.

38-68

Even though the 1985 paper was peer-reviewed, the findings of the study are open to question, as are those of the 1992 paper. See also the response to Comment 38-13.

38-69

See the responses to Comments 38-22 and 38-61.

38-70

Major reorganization of the Draft SEA would make it difficult for the interested public to compare the Draft and Final SEA to determine the changes made to respond to comments on the Draft SEA. For that reason the SEA has not been reorganized.

The availability of other reviews, and specifically the EPA report by Elder and Cahill (1984), was noted on page B-1 of the SEA. However, the preparers of the SEA directly reviewed the scientific literature, as opposed to reviewing other reviews of the RFR bioeffects literature, to reach their conclusions independently. See also the response to Comment 38-69.

It is true that relatively few epidemiologic studies were done on RFR and chronic disease. However, in addition to those cited in the EPA comment, the SEA includes discussions of those by Lester and Moore (1982a, 1982b), Hamburger et al. (1983), Thomas et al. (1987), and Burr and Hoiberg (1988) (pp. B-17 to B-23), as well as the epidemiologic studies on ocular effects. In addition, Heynick (1987) includes several early Eastern European studies not discussed in the Draft SEA. Nevertheless, the first sentence of the first paragraph of Section 3.1.1 (p. B-15 of the Draft SEA) has been deleted and the following sentence inserted:

> Relatively few epidemiologic studies have been conducted on possible links between exposure to RFR and the incidence of chronic disease, and all of those studies have been retrospective.

38-71

The suggested correction has been made.

38-72

See the response to Comment 38-14.

38-73

The latter portion of the fifth sentence of the third paragraph on page iii of the Draft SEA is changed to the following:

> ...in American homes and is far below 60-Hz magnetic-field exposure guidelines published by the International Non-Ionizing Radiation Committee of the International Radiation Protection Association (1000 mG for up to 24 h/d for members of the public).

This is a health-based guideline.

38-74

The suggested correction has been made.

38-75

The second sentence of the second paragraph on page iv of the Draft SEA has been replaced by the following:

> Similarly, the review found that adverse effects are not expected to result from exposure to WSR-88D power line fields.

38-76

The first sentence of the fifth paragraph on page 46 of the Draft SEA has been deleted. The second sentence of that paragraph has been revised to:

> Relatively few epidemiologic studies have been conducted on the effects of RFR exposure, either occupationally or from residing in the vicinity of RFR emitters or experimental studies with human volunteers. Taken collectively, those studies yield no scientific evidence that chronic exposure to RFR at levels within the 1991 SCC-28 guidelines would cause detrimental health effects.

38-77

Section 3.3, Ocular Effects, on page 47 of the Draft SEA has been revised as follows:

> The results of animal experiments indicate that heating the eye to temperatures of about 41°C or more damages the lens. Another finding is that for continuous exposure for durations of 2 hours or more the threshold power density for ocular damage is within the range 100-150 mW/cm^2. In the relatively few epidemiologic studies on possible ocular damage from chronic RFR exposure, some of the findings were negative and others were positive. Most of the positive findings were found to be explained more by aging of the lens than by RFR exposure. Exceptions were cases of possible occupational exposure at levels and for durations sufficient to heat the eye to damaging temperatures. Such cases occurred primarily during the first decade after the end of World War II, as exemplified in the paper by Hirsch and Parker (1952).

38-78

The following paragraph has been added following the last paragraph of Section 3.1 on page 58 of the Draft SEA:

> The EPA issued a preliminary report for external review (EPA, 1990) indicating a possible link between exposure to electromagnetic fields (RFR as well as power lines) and cancer. It recommended that electromagnetic fields in the extremely low frequency (ELF) range be classified as "potential human carcinogens," in a class with polychlorinated biphenyls (PCBs), DDT, and formaldehyde. The report also recommended that RFR be designated as a possible carcinogen, in a class with saccharin. The ELF and RFR recommendations were subsequently deleted in the absence of a mechanism of interaction and a dose-response relationship. The preface, dated 13 December 1990, to the October 1990 draft report stated that:

> While there are epidemiological studies that indicate an association between EM fields or their surrogates and certain types of cancer, other epidemiological studies do not substantiate this association. There are insufficient data to determine whether or not a cause and effect relationship exists.

EPA's Radiation Advisory Committee of the Science Advisory Board set up a subcommittee on Nonionizing Electric and Magnetic Fields to review the draft report. That subcommittee has issued a report, SAB (1992), indicating its findings. The subcommittee suggested numerous changes in emphasis, coverage, and wording, and concluded that the draft report [EPA, 1990] will have to be rewritten to accommodate all of the suggestions and comments.

38-79

The sixth paragraph on page 64 of the Draft SEA has been replaced with the following:

> The number of epidemiologic studies of powerline field effects in humans is increasing. Approximately half report positive findings for an association between some indicator or estimate of the presence of electric and magnetic fields and the increased incidence of several types of cancer, or of other biological endpoints including outcomes of pregnancy after use of electric blankets, infertility of exposed male workers, suicides, and measures of general health. The other half do not. A number of these studies are discussed below. However, most if not all of them have recognized weaknesses (Connecticut Academy of Science and Engineering, 1992; Jauchem and Merritt, 1991), making interpretation difficult. In these circumstances, the positive studies may be said to be suggestive of an effect, but do not prove that it exists. Likewise, negative studies do not prove that it does not exist. Acceptance of the positive findings of epidemiologic studies requires the concurrent demonstration of scientifically accepted mechanisms (e.g., of carcinogenesis), and much of the latter is presently speculative for EMFs. The most that can be said at present is that more research is needed.

The following sentence has been added at the beginning of the last paragraph on page 64 of the Draft SEA:

> In North America, electric transmission and distribution is at a frequency of 60 Hz as compared to the 50 Hz used in Europe and most of the rest of the world.

Response to Comments from George Morgan and Cindy Kushner

39-1

The NEXRAD system can make highly detailed measurements of wind velocity and rainfall intensity. Miniaturization of sensors is not sufficiently advanced to allow placement of NEXRAD components in a satellite platform. Thus, satellites cannot provide the measurement and geographic accuracy that will result with the NEXRAD system. NOAA uses satellites to track large-scale meteorological phenomena, and they will complement the NEXRAD system. See also the response to Comment 12-1.

Response to Comments from Cindy Kushner

40-1

See the responses to Comments 12-1 and 39-1.

Response to Comments from Danna G. Sturm

42-1

This comment concerns possible interference between the proposed NEXRAD transmitter on Virginia Peak and existing point-to-point communications systems that operate in the 1900-2200 MHz band. The same question was raised in a letter dated April 12, 1989, from Mr. Gene E. Oakley of the same agency and is addressed in the following documents prepared by SRI International:

- Memo from Bill Edson to Dave Leone, revised 17 June 1987, "Possible Radio Interference at Reno, Virginia Peak."
- Memo from W.A. Edson to Juris Petriceks dated May 2, 1989, "Virginia Peak NV."
- Letter dated May 16, 1989 from Juris Petriceks to Mr. James Barrows of NOAA, Seattle.
- Memo from W.A. Edson to Juris Petriceks dated May 24, 1989, "Nevada Bell Equipment at Reno, NV, (Virginia Peak)."

Measurements on an ASR-8 radar, which is similar to WSR-88D, show that at a frequency 300 MHz below the carrier the power level is reduced by about 120 dB. The communications systems of concern to the State Communications Board operate in the frequency band between 1,900 and 2,200 MHz, as compared to the WSR-88D band of 2,700 to 3,000 MHz. Because those systems will operate at least 500 MHz below the lowest NEXRAD frequency, it is appropriate to use the 120 dB value for attenuation of the WSR-88D signal. [See also R.L. Hinkle (1983) "Background Study on Efficient Use of the 2,700-2,900 MHz Band," NTIA Report 83-177.]

Reduced by 120 dB, the 475 kW output of the WSR-88D becomes 0.475 mW, which is the total power delivered by the klystron in the 1900-2200 MHz band. Not all of this power will be radiated because of waveguide losses and antenna inefficiency. Additionally, most of the radiated power will follow the main beam and thus be directed away from the communication antennas. Thus, it is safe to conclude that the WSR-88D at Virginia Peak will not contribute significantly to the electromagnetic noise floor experienced by the links in the Nevada State Communications System.

Response to Comments from Joe A. Elder and Carl F. Blackman

These comments in this letter (Comment Letter #45) directly refer to the comments in the letter from Richard E. Sanderson (Comment Letter #38). To assist the reader, the responses to comments by Elder and Blackman have been incorporated into the responses for Sanderson's comments.

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APPENDIX E

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