



VOLUME II - PART D
DOSE RECONSTRUCTION FEASIBILITY STUDY

TASK 6

Hazard Summaries for
Important Materials at the Oak Ridge Reservation

Prepared by
ChemRisk®
A Division of McLaren/Hart

for the
Tennessee Department of Health and the
Oak Ridge Health Agreement Steering Panel



Oak Ridge Health Agreement Steering Panel

OAK RIDGE HEALTH STUDIES PHASE I REPORT

Volume II - Part D - Dose Reconstruction Feasibility Study

**Tasks 6: Hazard Summaries for Important Materials at the Oak Ridge
Reservation**

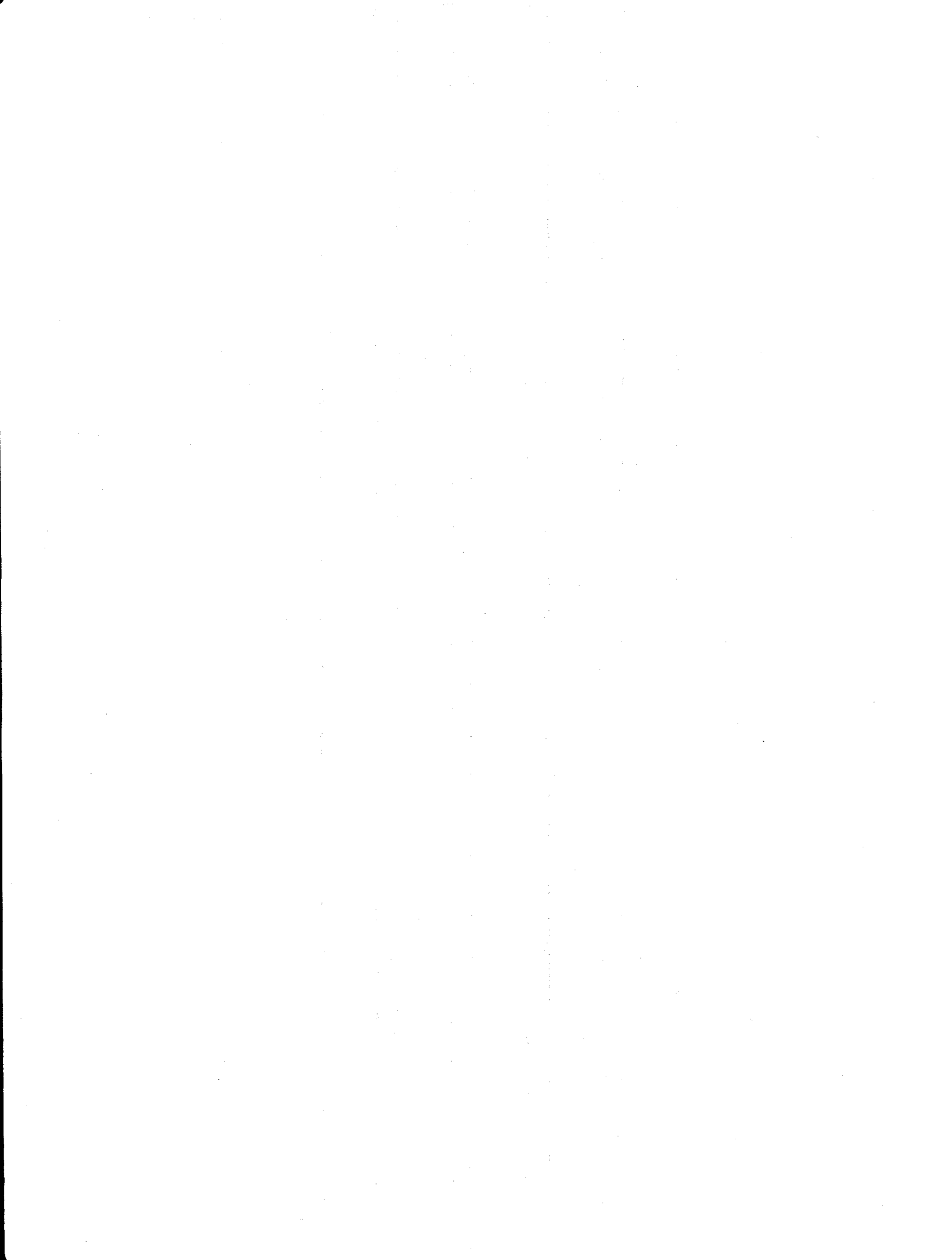
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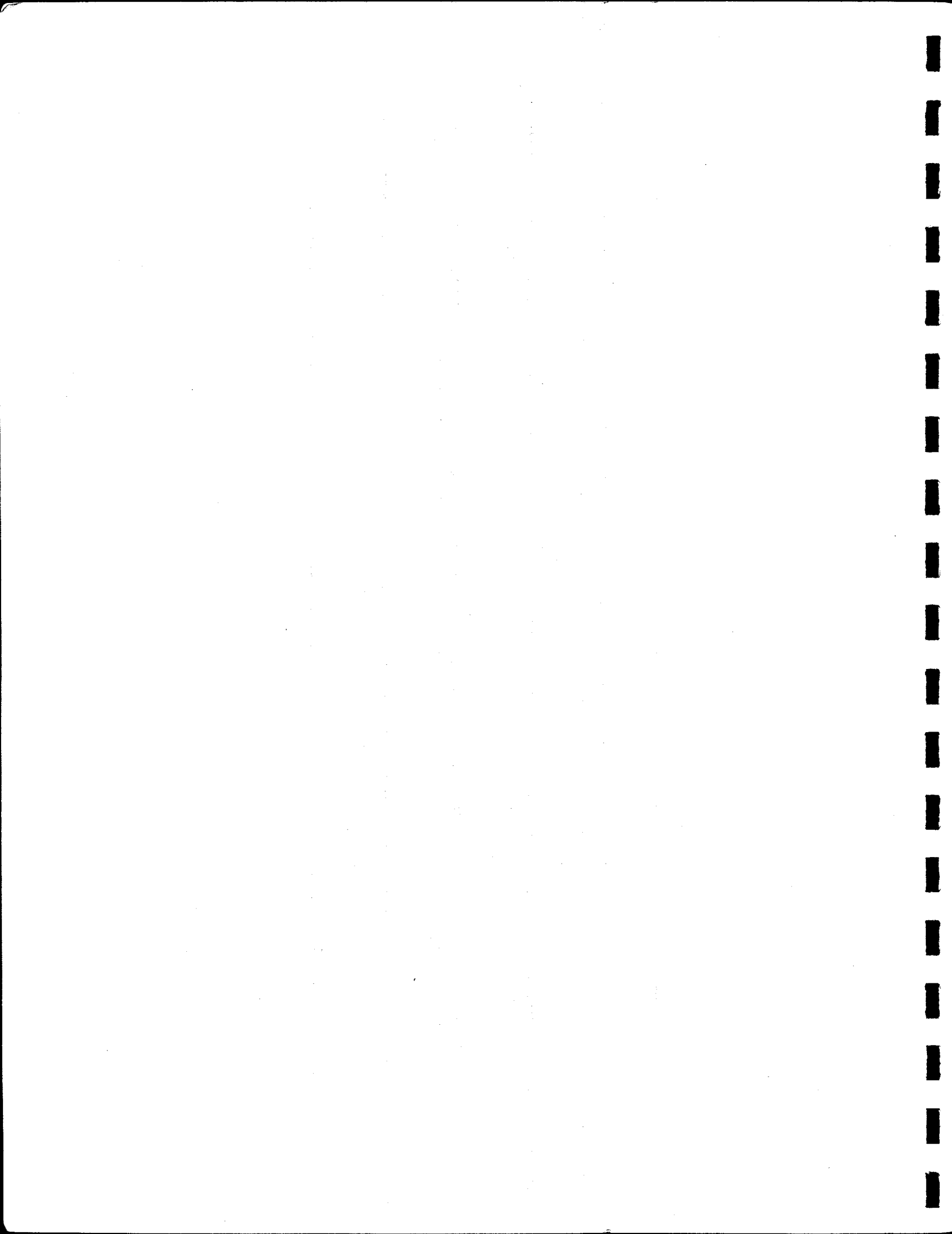
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CONTENTS OF THE OAK RIDGE HEALTH STUDIES PHASE I REPORT

Volume I summarizes the activities of the Oak Ridge Health Agreement Steering Panel, other than the Dose Reconstruction Feasibility Study, during Phase I of the Oak Ridge Health Studies. It includes four major topics:

- **Executive Summary of the Oak Ridge Health Studies Phase I Report**
- **Health Studies Background and Overview**
- **Phase I Goals**
- **Conclusions and Recommendations for Phase I**

Volume II documents the study (referred to as the Dose Reconstruction Feasibility Study) to find out if enough data exist to estimate historical doses of chemicals and radionuclides to the public living around the Reservation. It is comprised of four parts:

- **Part A** addressing project Tasks 1 and 2 to identify the historical operations and emissions at each of the complexes and characterize the availability of environmental sampling and research data
- **Part B** addressing Tasks 3 and 4 to identify important environmental exposure pathways and contaminants released from the Reservation
- **Part C** addressing Task 5 to identify information regarding historical locations and activities of off-site populations that could potentially be affected by releases from the Reservation

- **Part D** addressing Task 6 to identify the hazards associated with substances used at the reservation

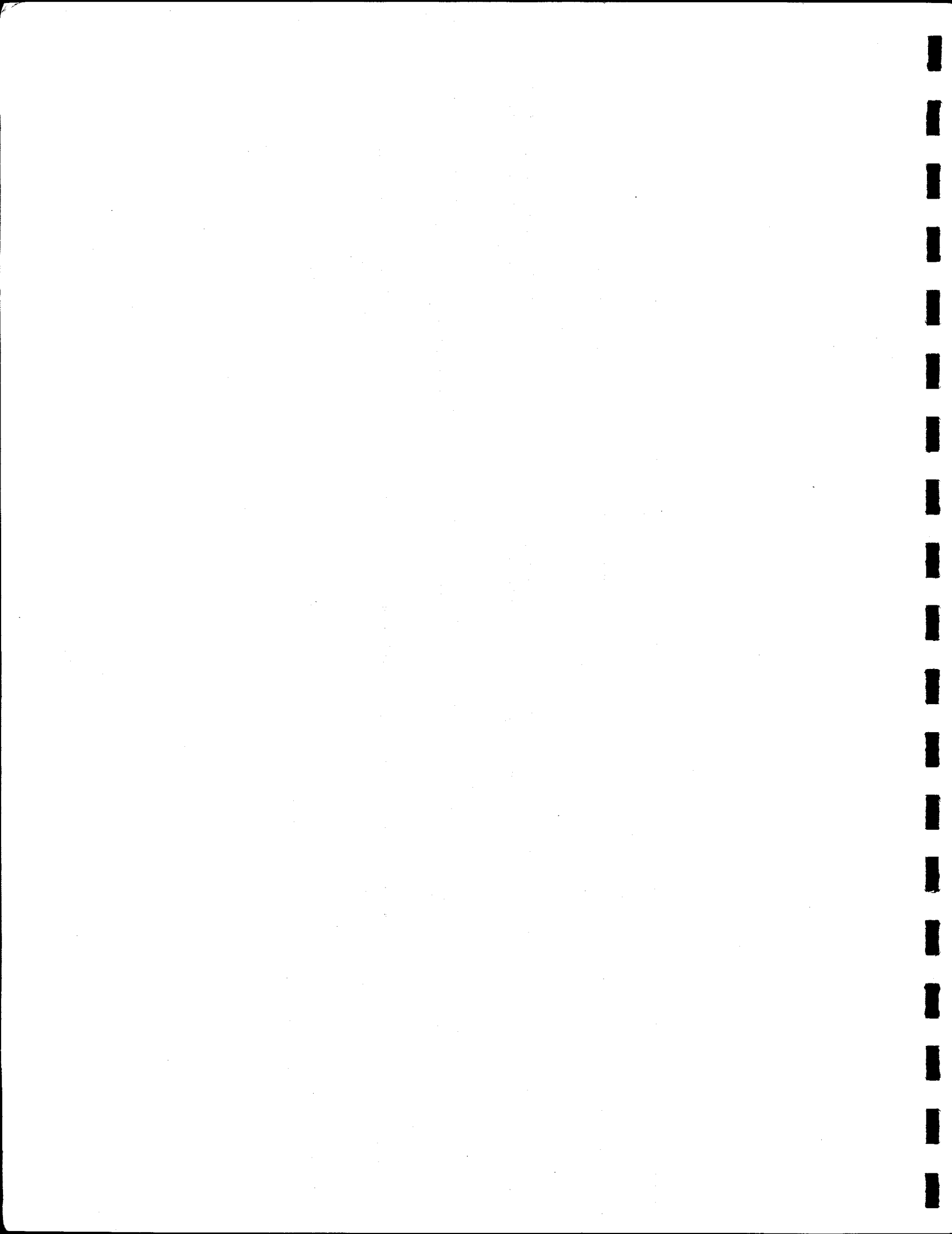


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3.0 SELECTED REFERENCE MATERIAL

Reference material is presented in the following order with blue dividers between each section.

Arsenic
Asbestos
Beryllium
Chlorinated Solvents
 Carbon Tetrachloride
 Methylene Chloride
 Tetrachloroethylene
 1,1,1-Trichloroethane
 Trichloroethylene
Chromium
Fluorine
Lead
Mercury
Polychlorinated Biphenyls (PCBs)
Polycyclic Aromatic Hydrocarbons (PAHs)
Radionuclides
Uranium

GLOSSARY OF TERMS

ABSORBED DOSE (of radiation)

The energy imparted by ionizing radiation per unit mass of irradiated material. The units of absorbed dose are the rad and the gray (Gy).

ACTIVATION PRODUCTS

Radionuclides that are created when substances are bombarded by neutrons. For example, the stable isotope cobalt-59 becomes the radioactive isotope cobalt-60 under neutron bombardment.

ACUTE

Of short duration and/or rapid onset. An acute toxic effect is one that develops during or shortly after a brief exposure to a toxic substance.

ANEMIA

A condition in which the blood is deficient in red blood cells, in hemoglobin, or in total volume of packed red blood cells.

BECQUEREL (Bq)

The International System of Units (SI) unit of radioactivity. One becquerel equals 1 disintegration per second. One curie equals 3.7×10^{10} Bq.

CARCINOGEN

A substance that is capable of causing cancer.

CHRONIC EXPOSURE

Repeated exposure to a chemical for over 3 months.

COMMITTED DOSE EQUIVALENT ($H_{T,50}$)

The dose-equivalent to organs or tissues of reference (T) that will be received from an intake of radioactive material by an individual during the 50-year period following intake.

COMMITTED EFFECTIVE DOSE EQUIVALENT ($H_{E,50}$ or CEDE)

The sum of the products of the weighting factors applicable to each of the body organs or tissues that are irradiated and the committed dose equivalent to these organs or tissues.

$$H_{E,50} = \sum W_T H_{T,50}$$

DERMAL

Related to the skin.

DOSE (for chemical toxicants)

The amount of a substance available for interaction with metabolic processes of an individual following exposure and absorption. The amount of a substance crossing the exchange boundaries of the skin, lungs, or digestive tract is termed absorbed dose.

GLOSSARY OF TERMS

DOSE (radiological dose)

A generic term that means absorbed dose, dose equivalent, effective dose equivalent, or committed effective dose equivalent.

DOSE EQUIVALENT (H_T)

The product of the absorbed dose in tissue, a quality factor, and all other necessary modifying factors at the location of interest. The units of dose equivalent are the rem and the sievert (Sv). $1 \text{ Sv} = 100 \text{ rem}$.

EFFECTIVE DOSE EQUIVALENT (H_E)

The sum of the products of the dose equivalent to the organ or tissue (H_T) and the weighting factors (W_T) applicable to each of the body organs or tissues that are irradiated ($H_E = \sum W_T H_T$)

Tissue	W_T
Gonads	0.20
Bone marrow	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Esophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05

The weighting factors represent the fraction of the total risk from irradiation of an organ when the total body is irradiated uniformly.

EPIDEMIOLOGY

The study of the distribution and causes of diseases and injuries in human populations.

EXPOSURE

In general terms, the amount of a chemical substance or radiation in the vicinity of a portal of entry to the body (e.g., the lungs, mouth, skin) that may be available for absorption. Exposure is also a measure of the ionization produced in air by X or gamma radiation. The special unit of radiation exposure is the roentgen (R).

GLOSSARY OF TERMS

FIBROSIS

A condition marked by an increase of tough, sinewy tissue, usually in reaction to an injury.

FISSION

The splitting of a nucleus into at least two other nuclei with the release of 1-5 neutrons and a relatively large amount of energy.

FISSION PRODUCTS

The nuclei (fission fragments) formed by the fission of heavy elements, plus the nuclides formed by the fission fragments' radioactive decay.

FRANK EXPOSURE LEVEL (FEL)

The level of exposure that produces a statistically or biologically significant increase in frequency or severity of unmistakable adverse effect such as irreversible functional impairment or mortality in an exposed population when compared with its appropriate control.

HALF-LIFE (physical)

The time in which half the atoms of a particular radioactive substance disintegrate to another substance.

HALF-LIFE (effective)

The time required for a radioactive element in an animal body to be diminished 50 percent as a result of the combined action of radioactive decay and biological elimination.

INORGANIC

Chemical substances which do not contain carbon linked to other elements by covalent bonds. Inorganic refers to chemical substances that are not hydrocarbons or their derivatives. For example, carbides, carbonates, and elemental metals are inorganic substances.

INSOLUBLE

Not able to be dissolved in a fluid (as in the term "water insoluble")

ISOTOPES

Nuclides having the same number of protons (same atomic number, and therefore same element) but differing in their number of neutrons and therefore their mass numbers (number of protons plus neutrons). Often, particularly in the past, improperly used as a synonym for nuclide.

LESION

Generic term for a type of damage or alteration. For example, abrasions, blisters, ulcers, dermatitis, and skin cancer are all types of skin lesions.

GLOSSARY OF TERMS

LETHAL CONCENTRATION₅₀ (LC₅₀)

A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population.

LOWEST OBSERVED ADVERSE EFFECT LEVEL (LOAEL)

The lowest dose of a chemical in a study or group of studies which produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

MALIGNANT

In reference to cancer, having the property of uncontrollable growth and dissemination, or recurrence after removal, or both.

MELANOMA

A malignant lesion that may occur in the skin of any part of the body, in the eye, or in mucous membranes. Melanomas often originate in a pigmented mole.

MINIMAL RISK LEVEL (MRL)

An estimate of daily human exposure to a chemical that is not likely to cause noncancerous toxicity over a specified duration of exposure.

NO OBSERVABLE ADVERSE EFFECT LEVEL (NOAEL)

The dose of a chemical at which there are no statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

NUCLIDE

An individual species of an element characterized by its particular mass number (number of protons plus neutrons), atomic number (number of protons), and the energy state of its nucleus.

ORGANIC

Chemical compounds which contain carbon atoms linked by covalent bonds.

PATHOLOGICAL

Altered or caused by disease

RADIOACTIVE

Possessing the property of spontaneously emitting electromagnetic rays or charged subatomic particles of matter with the release of energy.

RADIONUCLIDE

A radioactive nuclide.

GLOSSARY OF TERMS

REFERENCE CONCENTRATION (RfC)

A concentration (mg/m³) of a chemical in air that is not expected to cause adverse health effects over a lifetime of daily exposure. The term "reference concentration" refers to the concentration of a chemical in air that is *inhaled*.

REFERENCE DOSE (RfD)

A dose (mg/kg-day) of a chemical that is not expected to cause adverse health effects over a lifetime of daily exposure. In general, the term "reference dose" refers to the dose of a chemical that is *ingested*.

RISK

The nature and probability of occurrence of an unwanted, adverse effect on human life or health, or on the environment.

SIEVERT (Sv)

The special name for the International System of Units (SI) unit of dose equivalent. 1 Sv = 1 J/kg = 100 rem.

SLOPE FACTOR (SF)

The 95 percent upper confidence limit of the probability that a carcinogenic response will occur per unit daily intake of a chemical over a lifetime. Slope factors for most chemicals are expressed in units of (mg/kg-day)⁻¹. The slope factor for asbestos is expressed in units of (fibers/ml)⁻¹.

SOLUBLE

Able to be dissolved in a fluid (as in the term "water soluble").

SUBACUTE EXPOSURE

Repeated exposure to a chemical for one month or less.

SUBCHRONIC EXPOSURE

Repeated exposure to a chemical for 1 to 3 months.

TOXICITY

The degree to which a substance causes change in an organism which results in impairment of function or enhances susceptibility to the harmful effects of other environmental influences.

VOLATILIZE

To evaporate or cause to evaporate. The conversion of a solid or liquid into a gas.

GLOSSARY OF TERMS

WEIGHT-OF-EVIDENCE GROUPING FOR CARCINOGENICITY

A classification of a chemical's carcinogenic potential based on evidence of carcinogenicity from epidemiological studies and animal studies. Chemicals are placed into a weight-of-evidence group by the USEPA Human Health Assessment Group (HHAG).

Weight-of-evidence groups are defined as follows:

- Group A: Human Carcinogen (sufficient evidence of carcinogenicity in humans)
- Group B: Probable Human Carcinogen
 - B1 - limited evidence of carcinogenicity in humans;
 - B2 - sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
- Group C: Possible Human Carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data)
- Group D: Not Classifiable as to Human Carcinogenicity (inadequate or no evidence)
- Group E: Evidence of Noncarcinogenicity for Humans (no evidence of carcinogenicity in adequate studies)

ACRONYMS, INITIALISMS, AND ABBREVIATIONS USED IN THIS REPORT

ATSDR	Agency for Toxic Substances and Disease Registry
AVLIS	Atomic Vapor Laser Isotope Separation
CEDE	Committed Effective Dose Equivalent
d	Day
FEL	Frank Effect Level
h	Hour
ICRP	International Commission of Radiological Protection
K_d	Equilibrium Distribution Coefficient
LC ₅₀	Lethal Concentration ₅₀
LOAEL	Lowest Observed Adverse Effect Level
MRL	Minimal Risk Level
NCRP	National Council on Radiation Protection and Measurements
NOAEL	No Observed Adverse Effect Level
ORHASP	Oak Ridge Health Agreement Steering Panel
ORNL	Oak Ridge National Laboratory
ORR	Oak Ridge Reservation
PAHs	Polycyclic Aromatic Hydrocarbons
PCBs	Polychlorinated Biphenyls
RaLa	Radioactive Lanthanum
RfC	Reference Concentration
RfD	Reference Dose
SF	Slope Factor
Sv	Sievert
USEPA	United State Environmental Protection Agency
y	Year

EXECUTIVE SUMMARY

The Phase I feasibility study has focused on determining the availability of information for estimating exposures of the public to chemicals and radionuclides released as a result of historical operation of the facilities at the Oak Ridge Reservation (ORR). The estimation of such past exposures is frequently called dose reconstruction. The work of Phase I has examined both the feasibility of performing dose reconstruction and also attempted, in a limited way, to examine a portion of the enormous volume of historical data to identify the releases from the facilities in the past having the highest potential to have caused harm to the health of the public.

The project work was composed of a number of individual tasks designed to meet the overall objectives of the Phase I Studies. The study tasks are numbered 1 through 7. The initial project tasks, **Tasks 1 and 2** were designed to identify and collect information that documents the history of activities at the ORR that resulted in the release of contamination and to characterize the availability of data that could be used to estimate the magnitude of the contaminant releases or public exposures. **Task 7** was designed to support the collection of many of the documents and data identified in **Tasks 1 and 2** in a library that could then be used in any future health studies. These three tasks represent the information collection portion of the project and included qualitative evaluations of the potential for activities to have produced significant contaminant releases.

The **Task 1** investigations have led to the documentation of an overview of the activities that have taken place at each of the major complexes, including routine operations, waste management practices, special projects, and accidents and incidents. Historical activities that appear to warrant the highest priority in any further investigations were identified based on their likely association with off-site emissions of hazardous materials as indicated by the documentation reviewed or information obtained in interviews. Information that is available to support the reconstruction of historical releases of hazardous materials and possible off-site exposures for these high priority activities is summarized in the task report.

Task 2 focused on the development of an understanding of the environmental sampling and research data that are available to support any future dose reconstruction efforts. Information on the availability of environmental data was obtained from document reviews and personal interviews. Abstracts were developed to summarize approximately 100 environmental monitoring and research projects that characterize the historical presence and behavior of contaminants in areas outside of the ORR.

In structuring the Phase I studies, there was a desire to attempt to use the quantitative data on releases from the facilities and contamination present in the environment as another means of identifying those plant activities that should receive the highest priority in any further health studies. Project **Tasks 3 through 6** support a more quantitative evaluation of the potential impacts of facility releases. This quantitative evaluation represents a very rough and preliminary evaluation of the large quantity of information and data identified in **Tasks 1 and 2** to identify

those activities and contaminants having the greatest potential to cause harm to the public's health. The evaluation follows the following basic steps that are necessary to evaluate potential human health hazards:

- **Hazard identification**— identification of the contaminants that were released that are capable of causing harm to health.
- **Dose-response assessment**— characterization of the toxicity of the released contaminants by identifying the health effects that can result from exposure and the amount or dose of the contaminant required to produce the various health effects.
- **Exposure/Hazard Assessment**— quantification of the exposures that the public could have received.

The contaminants of concern to the Phase I health studies were identified in Tasks 1 and 2. The exposure and hazard assessment step of the process is covered in a preliminary fashion in Tasks 3 & 4. The Task 6 work represented in this report addressed the dose-response characterization step of the process. This step was accomplished by relying on, and, in large part, reproducing summary-level information available in documents produced by various regulatory agencies, government health agencies, and other authoritative bodies that publish guidelines and information on the toxicity and behavior of contaminants in the human body. This report presents information in the following formats to describe the hazards of materials that have been important at the Oak Ridge Reservation:

- A glossary of terms used in describing hazards of chemical and radiological hazards.
- A series of brief Material Hazards Summaries that address the current state of knowledge of the toxic properties of materials or groups of materials that were used at Oak Ridge.
- Selected reference material prepared by the Agency for Toxic Substances and Disease Registry and the National Council on Radiation Protection and Measurements. These pages provide considerably more detail concerning hazards of the materials of concern than can be provided in the brief summaries.

1.0 INTRODUCTION

A major focus of the Oak Ridge Phase I Health Studies is the description of historical uses and emissions of important materials. Project Task 1 has examined historical operations and emissions from the Oak Ridge facilities, and identified many materials which have been used in production, maintenance, and research applications. Not all of these materials warrant equal attention in terms of potential off-site health risks. The potential for off-site impact from a material are largely determined by its ability to travel off-site and its degree of toxicity or hazard.

The purpose of Task 6 of Oak Ridge Phase I Health Studies is to provide summaries of current knowledge of toxic and hazardous properties of materials that are important for the Oak Ridge Reservation. The information gathered in the course of Task 6 investigations will support the task of focussing any future health studies efforts on those operations and emissions which have likely been most significant in terms of off-site health risk. The information gathered in Task 6 efforts will likely also be of value to individuals evaluating the feasibility of additional health study efforts (such as epidemiological investigations) in the Oak Ridge area and as a resource for citizens seeking information on historical emissions.

1.1 Task 6 Scope of Work

The request for proposals for the Phase I Health Studies included a list of materials to be included in the evaluation of historical emissions. Task 1 investigations of historical operations and emissions have verified that many of the materials that were initially identified for consideration have been important materials used at the Oak Ridge plants. Through records review and personnel interviews, some additional materials that warrant consideration have been identified. At the same time, some materials that were initially identified have been shown to have been used in manners which made it unlikely that off-site health risks resulted that warrant primary focus at this time.

No new toxicological research is being conducted by the Phase I Health Studies investigators. In accordance with the recommendations of the Oak Ridge Health Agreement Steering Panel (ORHASP) and with the feasibility study nature of the Phase I work, existing information is being assembled from leading sources of toxicological information, namely the Agency for Toxic Substances and Disease Registry (ATSDR), the International Commission on Radiological Protection (ICRP), the National Council on Radiation Protection and Measurements (NCRP), and the U.S. Environmental Protection Agency (USEPA).

While the primary purpose of Task 6 is not to present information on how materials were used at the Oak Ridge facilities, brief summaries of the nature of the association that each material has had with the Oak Ridge Reservation are provided in order to help bridge the gap between historical operations information and current knowledge of the toxic properties of the materials involved.

1.2 Description of the Material Hazards Summaries

A series of brief Material Hazards Summaries has been prepared for a number of key materials that have been associated with historical Oak Ridge operations. To increase the usefulness of these summaries, certain materials have been grouped. These include chlorinated solvents, polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), and key radionuclides. These materials were grouped because of their similar physical or toxicologic properties in order that similarities could be indicated and that properties of the individual materials within classes could be compared.

The Material Hazards Summaries for chemicals have the following structure:

1. **Common Forms** - presents a brief list of commonly encountered forms of the material in question
2. **Nature of Use and Presence in the Environment** - presents a general overview of properties and uses of the material and facts relevant to its environmental presence and behavior.
3. **Use at Oak Ridge** - presents a very brief statement of the nature of the presence or uses of the material in question at the Oak Ridge Facilities. For more details, the report of Tasks 1 and 2 can be consulted.
4. **Toxic Effects** - presents a summary of the current knowledge of the toxic properties of the material in question, based on animal studies or human experience as stated.
5. **Indicators of Toxic Potency** - presents a summary of available information relevant to the degree in which a material can cause harm. Values given in this section include EPA Weight-of-Evidence classifications, cancer slope factors for carcinogens, and reference concentrations and/or reference doses for noncarcinogenic compounds.

The Material Hazards Summary for the key radionuclides at Oak Ridge has a unique format tailored to the information thought to be most appropriate at this point in the Health Studies. The radionuclide summary has the following structure:

1. **Key Radionuclides and their Sources and Uses** - presents a brief summary of the nature of each selected radionuclide's involvement at the Oak Ridge facilities.
2. **Radionuclide Carcinogenicity** - presents a brief summary of the recommended methodology for estimation of risk from radiation exposures.

3. **Factors in Environmental Mobility** - presents estimated values of distribution coefficients that indicate to what extent an element remains in solution or adsorbs on sediment.
4. **Factors in Environmental Persistence** - presents physical half-lives for selected radionuclides sorted in order of increasing magnitude and by nuclide name in alphabetical order.
5. **Relative Radiation Hazard when Ingested** - presents radiation dose conversion factors for selected radionuclides, ranked by dose per unit weight ingested or per unit radioactivity ingested.
6. **Relative Radiation Dose when Inhaled** - presents radiation dose conversion factors for selected radionuclides, ranked by dose per unit weight inhaled or per unit radioactivity inhaled.

1.3 Description of the Selected Reference Material

Section 3 of this report contains selected reference material for those individuals who desire additional toxicological information concerning the materials described in the Material Hazards Summaries. For most chemical toxins, the "Public Health Statement" sections of ATSDR Toxicological Profiles have been provided. To keep this document to a manageable size, complete copies of these documents are not provided. Draft toxicological profiles issued for public comment can be requested by writing to the following address:

Department of Health and Human Services
U.S. Public Health Service
Agency for Toxic Substances and Disease Registry
1600 Clifton Road N.E.
Mail Stop E-29
Atlanta, Georgia 30333
Telephone: (404) 639-6300

For information on how to receive copies of final toxicological profiles, call or write:

National Technical Information Service
5285 Port Royal Road
Springfield, Virginia 22161
Telephone: (800) 553-6846 or (703) 487-4650

For radionuclides, information from National Council on Radiation Protection and Measurements (NCRP) Report No. 65 has been provided. This report is entitled "Management of Persons Accidentally Contaminated with Radionuclides." Alternative information sources are International Commission on Radiological Protection (ICRP) publications, for example ICRP Publication Number 28, "The Principles and General Procedures for Handling Emergency and Accidental Exposure of Workers."

NCRP reports can be obtained by contacting the following office:

NCRP Publications
7910 Woodmont Avenue, Suite 800
Bethesda, MD 20814

Inquiries regarding ICRP publications can be directed to Pergamon Press, Inc., at the following address:

Pergamon Press Inc.
Maxwell House
Fairview Park
Elmsford, NY 10523

2.0 MATERIAL HAZARDS SUMMARIES

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Common Forms:

Metallic arsenic, arsenic acid, arsenic pentoxide, arsenic trioxide, arsenic trisulfide, calcium arsenate, sodium arsenate, sodium arsenite, arsenic acid, trimethylarsine, and other organic arsenic forms.

Nature of Use and Presence in the Environment:

Arsenic is a naturally occurring element and is widely distributed in the environment. It is usually found combined with one or more other elements such as oxygen, chlorine and sulfur, rather than as a pure metal. In these forms, it is referred to as inorganic arsenic, while when combined with carbon and hydrogen it is called organic arsenic. The inorganic forms of arsenic are more toxic than the methylated and organic forms. Elevated levels of arsenic are often associated with fossil fuel consumption, pesticide use, copper smelting, cigarette smoking, volcanic eruptions, and mineral weathering. High levels of the more inert organic form have been found in shellfish and other marine animals that store and excrete arsenic.

Use at Oak Ridge:

Arsenic was used in pesticides and wood treatment agents. It was also present in coal used for fuel.

Toxic Effects:

Inorganic arsenic is recognized as potentially toxic to humans, and ingestion of large doses can be lethal. Symptoms of acute exposure following ingestion are gastrointestinal irritation, anemia, neuropathy (disease of the nervous system), skin and vascular lesions, and liver or kidney lesions. Inhalation exposure to arsenic is not normally associated with acute lethality in animals or humans. The primary health effect associated with exposure to airborne arsenic is irritation of the skin and mucous membranes. Chronic exposure to arsenic via ingestion can result in gastrointestinal effects such as nausea, vomiting, and diarrhea. Long-term exposure may also lead to changes in the number of red and white blood cells and peripheral vascular disease, as well as irritation of the skin. The toxicity of arsenic does not appear to be cumulative, as small doses produce similar effects after both short- and long-term exposures.

Evidence suggests that arsenic is carcinogenic to humans by both the oral and inhalation routes; however, malignant tumors have not been observed in animal studies. Chronic oral exposure to elevated levels of arsenic has been shown to increase the risk of skin cancer in individuals exposed to water containing 30 $\mu\text{g}/\text{kg}/\text{day}$ or more. A number of epidemiologic studies have indicated an above-average incidence of lung cancer in populations exposed to arsenic through inhalation. The USEPA has classified arsenic as a Group A human carcinogen.

INDICATORS OF TOXIC POTENCY

ARSENIC

EPA Weight-of-Evidence Group	A (Human Carcinogen)
Carcinogenic Indicators:	
Slope Factor; inhalation	50 (mg/kg-day) ⁻¹
Slope Factor; ingestion	Not determined
Noncarcinogenic Indicators:	
Reference Concentration; inhalation	Not determined
Reference Dose; ingestion, chronic	0.0003 mg/kg-day

Common Forms:

Amosite, chrysotile, tremolite, actinolite, anthophyllite, crocidolite.

Nature of Use and Presence in the Environment:

Asbestos is the name applied to a group of minerals that occur naturally in the environment. These minerals are characterized by long, thin fibers that appear somewhat similar to fiber glass. Asbestos fibers are very strong and are resistant to heat and chemicals. Because of these properties, asbestos fibers are used in a wide range of products, especially building materials, friction products, and heat-resistant fabrics. Asbestos fibers are released to air from both weathering of natural outcroppings and the degradation of insulation, ceiling tiles, floor tiles, cement, and automobile brakes and clutches. Low levels of asbestos have been found in almost every air sample taken throughout the U.S. Asbestos fibers are also present in some drinking water sources and can be ingested.

Use at Oak Ridge:

Asbestos was used in insulating and fireproofing materials in both industrial buildings and residences.

Toxic Effects:

Information on the health effects of asbestos in humans comes mostly from studies of people exposed in the past to high levels of asbestos in the workplace. Numerous studies have shown that exposure of humans to asbestos fibers via inhalation can result in asbestosis, a disease characterized by a slow accumulation of scar-like tissue in the lungs and the membrane which surrounds the lungs. As a result of this formation of scar tissue, the tissue making up the lungs cannot expand and contract as it normally would and breathing becomes difficult. Severe cases of asbestosis may ultimately result in death. Because asbestos fibers are so resistant to breakdown, the inflammatory response triggered by the fibers is ongoing, even after exposure has ceased. Thus, the severity of pathological lesions and clinical symptoms tends to progress with time. No cases of acute or short-term lethality due to asbestos ingestion were located for either humans or animals. Evidence indicates that asbestos ingestion does not cause any significant noncarcinogenic effects in the gastrointestinal system.

Studies of workers exposed to asbestos in air have shown an increased risk of lung cancer and mesothelioma, a cancer of the thin membrane that surrounds the lung and other internal organs. Both forms of cancer are usually fatal; however, in people exposed to low levels of asbestos the cancer risks are small. Although some studies have suggested some forms of asbestos are more likely to cause cancer than others, most data indicate that the length of the fiber is the most important determinant in cancer-causing potential. It is not clear from the available information whether increased risk of cancer is associated with the ingestion of asbestos fibers. The USEPA has classified asbestos as a Group A human carcinogen.

INDICATORS OF TOXIC POTENCY**ASBESTOS**

EPA Weight-of-Evidence Group	A (Human Carcinogen)
Carcinogenic Indicators:	
Slope Factor; inhalation	0.23 (fibers/ml) ⁻¹
Slope Factor; ingestion	Not determined
Noncarcinogenic Indicators:	
Reference Concentration; inhalation	Not determined
Reference Dose; ingestion	Not determined

Common Forms:

Metallic beryllium, beryllium acetate, beryllium carbonate, beryllium chloride, beryllium fluoride, beryllium hydride, beryllium hydroxide, beryllium nitrate, beryllium oxide, beryllium sulfate

Nature of Use and Presence in the Environment:

Beryllium is a hard, brittle metallic element. It is used in ceramics, electron tubes, and high temperature reaction systems. It enters the environment primarily through coal combustion. The estimated typical human daily intake of beryllium from air, food and water is in the range of 0.4 $\mu\text{g}/\text{day}$. Beryllium has a natural abundance in the earth's crust of about 0.06-6 $\mu\text{g}/\text{g}$. It is accumulated and enriched in several plant species, including cultivated potatoes, tomatoes, and head lettuce.

Uses at Oak Ridge:

Beryllium was used in weapons manufacture and as a neutron reflector in nuclear reactors.

Toxic Effects:

The major toxicological effects following exposure to beryllium and beryllium compounds are to the lungs. In individuals exposed to varying levels of beryllium over varying exposure times, chronic pulmonary beryllium disease, otherwise known as berylliosis, has been observed. Symptoms of this condition include severe shortness of breath. Following short term exposure to high concentrations of beryllium, Acute Beryllium Disease, characterized by inflammation of the respiratory tract and chemical pneumonitis (a condition similar to pneumonia), has been observed. In sensitized individuals, dermal exposure to soluble beryllium compounds has caused allergic contact dermatitis. Ulcerated, noncancerous growths on the skin were observed to result after beryllium entered cuts in the skin of workers handling beryllium or beryllium compounds. No adverse reproductive effects have been observed following exposure of rats to beryllium. Data regarding adverse reproductive or developmental effects in humans are not available.

It is not clear if exposure to beryllium compounds is associated with an increase in the incidence of carcinomas in humans. No studies in animals or humans provide convincing evidence that ingestion of beryllium or its compounds causes cancer. However, experiments with laboratory animals indicate that breathing beryllium and some of its compounds, both soluble and insoluble, can cause lung cancer. Consequently, beryllium is classified by the USEPA as a Group B2 probable human carcinogen.

INDICATORS OF TOXIC POTENCY**BERYLLIUM**

EPA Weight-of-Evidence Group	B (Probable Human Carcinogen)
Carcinogenic Indicators:	
Slope Factor; inhalation	8.4 (mg/kg-day) ⁻¹
Slope Factor; ingestion	4.3 (mg/kg-day) ⁻¹
Noncarcinogenic Indicators:	
Reference Concentration; inhalation	Not determined
Reference Dose; ingestion, chronic	0.005 mg/kg-day
Reference Dose; ingestion, subchronic	0.005 mg/kg-day

Common Forms:

Some commonly used chlorinated solvents include methylene chloride (also known as dichloromethane), carbon tetrachloride, tetrachloroethylene (also known as perchloroethylene, or PCE), trichloroethylene (TCE), and 1,1,1-trichloroethane (1,1,1-TCA).

Nature of Use and Presence in the Environment:

Chlorinated solvents are frequently used as cleaning and degreasing agents, in the manufacture of organic chemicals and pharmaceuticals, and in laboratory extraction procedures. Because of their widespread use, chlorinated solvents are found in drinking water, surface water, surface water, and groundwater throughout the United States. Chlorinated solvents rapidly volatilize from surface waters and upper layers of soil into the atmosphere. The water solubility of chlorinated solvents varies from compound to compound. Most chlorinated solvents will readily leach from soil to groundwater and surface water. However, due to its high volatility and relatively low solubility and mobility in soil, carbon tetrachloride is not easily transported to groundwater.

Uses at Oak Ridge:

Chlorinated solvents were used for cleaning and degreasing equipment and manufactured components.

Toxic Effects:

The primary route of exposure of humans to chlorinated solvents is inhalation. Following inhalation of large quantities of chlorinated solvents, damage to the liver, kidney, lungs, cardiovascular system, and central nervous system has been observed. Effects on the central nervous system include anesthesia at very high concentrations and impairment of coordination, equilibrium, and judgement at lower concentrations. Irritation of the mucous membranes of the eyes, nose, and throat has also been observed, as has irritation of the skin following prolonged or repeated exposure. However, unless confined by clothing, chlorinated solvents will generally evaporate quickly from the skin.

The USEPA has classified methylene chloride, carbon tetrachloride, tetrachloroethylene, and trichloroethylene as probable (B2) carcinogens. Due to a lack of evidence from human or animal studies, the USEPA has not classified 1,1,1-TCA as to its carcinogenicity.

INDICATORS OF TOXIC POTENCY

CHLORINATED SOLVENTS

EPA Weight-of-Evidence Group	<u>B2 (Probable Human Carcinogens):</u> methylene chloride carbon tetrachloride tetrachloroethylene trichloroethylene	
EPA Weight of Evidence Group	<u>D (Not Classifiable as to Human Carcinogenicity):</u> 1,1,1-trichloroethane	
Carcinogenic Indicators:		
Slope Factor; inhalation	methylene chloride	0.0016 (mg/kg-day) ⁻¹
	carbon tetrachloride	0.053 (mg/kg-day) ⁻¹
	tetrachloroethylene	Not determined
	trichloroethylene	0.017 (mg/kg-day) ⁻¹
	1,1,1-trichloroethane	Not applicable
Slope Factor; ingestion	methylene chloride	0.0075 (mg/kg-day) ⁻¹
	carbon tetrachloride	0.13 (mg/kg-day) ⁻¹
	tetrachloroethylene	0.051 (mg/kg-day) ⁻¹
	trichloroethylene	0.011 (mg/kg-day) ⁻¹
	1,1,1-trichloroethane	Not applicable
Noncarcinogenic Indicators:		
Reference Concentration; inhalation	methylene chloride	Not determined
	carbon tetrachloride	Not determined
	tetrachloroethylene	Not determined
	trichloroethylene	Not determined
	1,1,1-trichloroethane	Not determined
Reference Dose; ingestion, chronic	methylene chloride	0.06 mg/kg-day
	carbon tetrachloride	0.0007 mg/kg-day
	tetrachloroethylene	0.01 mg/kg-day
	trichloroethylene	Not determined
	1,1,1-trichloroethane	0.09 mg/kg-day

Common Forms:

Chromic acetate, chromic acid, chromium chloride, chromium nitride, chromium oxychloride, chromium pentafluoride

Nature of Use and Presence in the Environment:

Chromium is a naturally occurring, environmentally pervasive element. The valence states of chromium of toxicological concern are III (trivalent chromium) and VI (hexavalent chromium). Trivalent chromium is the common stable form found in nature. It is an essential nutrient with a Recommended Daily Allowance (RDA) for adults of 0.05-0.2 mg/day. Hexavalent chromium is almost exclusively produced as the result of manufacturing activities. Elemental chromium does not occur in nature. There is much data to support the conclusion that reduction of hexavalent chromium to trivalent chromium commonly occurs under natural conditions, and that the reverse process, oxidation of trivalent chromium to hexavalent chromium, is not likely to occur. Chromium in soil can be transported to the atmosphere via soil particulate resuspension due to wind erosion or vehicle traffic. Chromium in soil is also transported via surface water runoff and leaching of water.

Use at Oak Ridge:

Chromium was used for corrosion inhibition in cooling tower waters and in plating of various components.

Toxic Effects:

Systemic effects have been demonstrated in the kidneys, liver, gastrointestinal tract, and circulatory system as a result of acute high dose exposure to trivalent and hexavalent chromium. High doses of chromate may lead to kidney damage.

The chronic effects of chromium include changes in the skin and mucous membranes. Long-term inhalation or ingestion of trivalent chromium compounds suggest minimal adverse health effects. Indications are that trivalent chromium is not a carcinogen and that it is effectively non-toxic to humans at doses which would be encountered in environmental situations. Long-term high dose inhalation exposure to hexavalent chromium is associated with lesions of the mucous membranes of the respiratory tract in humans. Increased lung cancers among chromate workers in the United States have been reported. Inhalation studies in animals indicate that calcium(VI)chromate and sodium(VI)dichromate may be weak carcinogens. The USEPA has classified hexavalent chromium as a Group A human carcinogen.

INDICATORS OF TOXIC POTENCY

CHROMIUM

Trivalent (III) Chromium	
EPA Weight-of-Evidence Group	D (Not Classifiable as to Human Carcinogenicity)
Carcinogenic Indicators:	Not applicable
Noncarcinogenic Indicators:	
Reference Concentration; inhalation:	Not determined
Reference Dose; ingestion, chronic:	1 mg/kg-day
Hexavalent (VI) Chromium	
EPA Weight-of-Evidence Group	A (Human Carcinogen)
Carcinogenic Indicators:	
Slope Factor; inhalation	41 (mg/kg-day) ⁻¹
Slope Factor; ingestion	Not determined
Noncarcinogenic Indicators:	
Reference Concentration; inhalation	Not determined
Reference Dose; ingestion, chronic	0.005 mg/kg-day

Common Forms:

Ammonium fluoride, boron trifluoride, chlorine trifluoride, hydrogen fluoride, nitrogen trifluoride, uranium hexafluoride, and sulfur hexafluoride.

Nature of Use and Presence in the Environment:

Fluorine is a yellow gas which reacts with practically all elements and compounds to form fluorine derivatives. In air, fluorine combines with water or moisture to form hydrogen fluoride and possibly oxygen fluoride, which is more toxic than fluorine. Elemental fluorine is used primarily in the production of organic and inorganic compounds, as in the manufacture of fertilizer, glass, ceramics, and brick, production of pharmaceuticals, and the processing of aluminum.

Use at Oak Ridge:

Fluorine was used to make uranium hexafluoride for enrichment by the gaseous diffusion process.

Toxic Effects:

Fluorine gas is highly irritating to the eyes and respiratory tract. Long-term residents near fluoride-emitting industries and individuals living in hot areas with high, natural fluoride levels in drinking water have developed dental fluorosis, characterized by lusterless, opaque white patches in the enamel of teeth; these may become striated, mottled, or pitted. Additionally, high fluoride concentrations in drinking water have been observed to affect all mineralizing tissues under formation, a condition known as skeletal fluorosis. This condition is often associated with joint and muscle pains.

In occupational exposure studies of aluminum smelter workers, a higher incidence of articular pain and limitation of motion in comparison with foundry workers of the same age was observed. However, in a similar study, no definitive cases of skeletal fluorosis were reported. Studies of any association between water fluoridation and cancer mortality identified no increase in risk. The USEPA has not classified fluorine as to its carcinogenicity.

Airborne concentrations of 20-250 parts per million of fluorine can be dangerous, even for brief exposures. Fatalities from acute exposures to high levels of fluorine are documented.

INDICATORS OF TOXIC POTENCY**FLUORINE**

EPA Weight-of-Evidence Group	Not determined
Carcinogenic Indicators:	Not applicable
Noncarcinogenic Indicators:	
Reference Concentration; inhalation	Not determined
Reference Dose; ingestion, chronic	0.06 mg/kg-day (fluoride)

Common Forms:

Lead acetate, lead carbonate, lead chloride, lead chromate, lead dioxide, Lead(II) fluoride, lead monoxide, Lead(II) phosphate, Lead(II) sulfate

Nature of Use and Presence in the Environment:

Lead is a naturally occurring metal found in small quantities in the earth's crust. It is ubiquitous in the environment, primarily due to the past extensive use of leaded gasoline as a fuel. Lead is extremely persistent in both water and soil. It binds strongly to organic materials present in soil, so that very little lead is transported into surface waters or groundwater. Lead is not easily taken up by plants, and therefore its availability to humans and animals via plant consumption is limited.

Use at Oak Ridge:

Lead was used extensively for radiation shielding.

Toxic Effects:

Symptoms of acute lead poisoning in humans include fatigue, disturbance of sleep, and constipation, followed by colic, anemia, and inflammation of nerves. Target organs are the brain and central nervous system, the peripheral nervous system, the kidneys, and the hemopoietic (blood-forming) system.

Chronic exposure to lead has been reported to cause permanent brain damage. For infants and young children, lead exposure has been shown to decrease intelligence (IQ) scores, slow growth, and cause hearing problems. Extended exposure to extremely high concentrations can result in progressive kidney damage and possibly kidney failure. Common symptoms of chronic toxicity are loss of appetite, metallic taste, constipation, anemia, pallor, weakness, nervous irritability, fine tremors, and colic. Results of published studies regarding the carcinogenicity of lead in humans are not definitive, although in animal studies, lead has resulted in an increased incidence of kidney tumors. The USEPA has classified lead as a probable (B2) carcinogen.

INDICATORS OF TOXIC POTENCY**LEAD**

EPA Weight-of-Evidence Group	B2 (Probable Human Carcinogen)
Carcinogenic Indicators:	
Slope Factor; inhalation	Not determined
Slope Factor; ingestion	Not determined
Noncarcinogenic Indicators:	
Reference Concentration; inhalation	Not determined
Reference Dose; ingestion	Not determined

Common Forms:

Elemental mercury, mercuric chloride, mercuric sulfide, mercuric acetate, mercuric oxide, mercuric salicylate, methyl mercury

Nature of Use and Presence in the Environment:

Mercury is a naturally occurring element that is ubiquitous in the environment. Mercury and its compounds have numerous uses in industry, including textile printing, photography, and the manufacture of scientific instruments (barometers, thermometers, etc.), electrical equipment, dry batteries, electric lamps, and x-ray tubes. Mercury is fairly mobile in the environment. Elemental mercury volatilizes at room temperature and can be released into the atmosphere by degassing of the element from soils. Once in the atmosphere, it can be transported long distances before returning to soil and water. Many forms of inorganic mercury may be transformed into the more toxic methylated organic mercury by bacteria in soil or water. Certain marine fish also transform inorganic mercury to methyl mercury.

Use at Oak Ridge:

Mercury was used to enrich lithium in its Li-6 isotope for use in thermonuclear weapons at the Y-12 and X-10 plants. It was also used in much lower quantities in instrumentation at the three ORR plants.

Toxic Effects:

Routes of entry of mercury into the human body include inhalation of dust or vapor and absorption through the skin. Uptake of mercury in food is the largest source of intake from the environment. Methyl (organic) mercury is more toxic than inorganic mercury when ingested. Methyl mercury compounds are absorbed from the digestive tract very rapidly in contrast to inorganic mercury compounds. Methyl mercury does not accumulate in the blood; it is transferred there and then excreted or stored in other organs. Major target organs are the central nervous system and the kidney, although the liver, heart, pancreas, endocrine, and reproductive systems may be affected. Ingestion of inorganic mercury salts may cause severe kidney damage. Inhalation of elemental mercury may cause pneumonia, bronchitis, chest pains, inflammation of the gums, nausea, vomiting, and diarrhea. Fatalities from inhalation of mercury vapor are documented.

Mercury tends to accumulate in the kidney and brain. In addition, mercury may pass through the placental wall into the fetus, concentrating in the brain tissue. Chronic mercury exposures are associated with behavioral and neurological disturbances of the central nervous system. Data regarding the carcinogenicity of mercury in experimental animals are limited. As such, the USEPA has not classified mercury as to its carcinogenicity.

INDICATORS OF TOXIC POTENCY

MERCURY

Methyl Mercury	
EPA Weight-of-Evidence Group	Group D (Not Classifiable as to Human Carcinogenicity)
Carcinogenic Indicators:	Not applicable
Noncarcinogenic Indicators:	
Reference Concentration; inhalation, chronic	Not determined
Reference Dose; ingestion	0.0003 mg/kg-day
Inorganic Mercury	
EPA Weight-of-Evidence Group	Group D (Not Classifiable as to Human Carcinogenicity)
Carcinogenic Indicators:	Not applicable
Noncarcinogenic Indicators:	
Reference Concentration; inhalation,	0.0003 mg/m ³
Reference Dose; ingestion	0.0003 mg/kg-day

Material Hazards Summary

POLYCHLORINATED BIPHENYLS (PCBs)

Common Forms:

PCBs include Aroclor-1260, -1254, -1248, -1242, -1232, -1221, and -1016

Nature of Use and Presence in the Environment:

PCBs are a family of man-made chemicals that include 209 individual compounds. Aroclors, one of the most common commercial PCBs produced in the U.S., are mixtures of chlorinated biphenyls. PCBs have been used extensively as components of hydraulic fluids, plasticizers, adhesives, lubricants, flame retardants, and as dielectric fluids in capacitors and transformers due to their insulating and nonflammable properties. PCBs are very persistent in the environment and are widely distributed in air, soil, and water. Although Aroclors are no longer produced or used in the production of new products in the United States, they are still found in older capacitors and transformers.

Use at Oak Ridge:

PCBs were used as lubricants in the machining of metal parts and as dielectric fluids in electrical capacitors and transformers.

Toxic Effects:

Oral toxicity studies have established that the liver and skin are the primary target organs following exposure to PCBs. Inhalation and dermal exposure are considered the major routes of occupational exposure. In animal studies, liver lesions were observed following inhalation exposure and liver and skin lesions were observed following application to the skin. The general population is exposed to PCBs primarily through ingestion of food, particularly fish; by comparison, inhalation exposure is negligible. Adverse health effects have not been observed in humans following nonoccupational exposures.

Aroclor 1260 was found to be carcinogenic in rats following ingestion. Following application on skin, Aroclor 1254 has shown carcinogenic potential in mice. Additionally, an increased incidence of melanomas was reported in a group of workers exposed occupationally (from both inhalation and dermal contact) to Aroclor 1254. The USEPA classifies PCBs as probable (B2) carcinogens.

INDICATORS OF TOXIC POTENCY**PCBs**

EPA Weight-of-Evidence Group	B2 (Probable Human Carcinogen)
Carcinogenic Indicators:	
Slope Factor; inhalation	Not determined
Slope Factor; ingestion	7.7 (mg/kg-day) ⁻¹
Noncarcinogenic Indicators:	
Reference Concentration; inhalation	Not determined
Reference Dose; ingestion	Not determined

POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

Material Hazards Summary

Common Forms:

Common PAHs include acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, chrysene, dibenz(a,h)anthracene, fluoranthene, fluorene, indeno(1,2,3-cd)pyrene, 2-methylnaphthalene, naphthalene, phenanthrene, and pyrene.

Nature of Use and Presence in Environment:

PAHs are a group of over 100 compounds formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances. PAHs may be man-made or occur naturally, and are used in limited quantities for research and to make drugs, dyes, plastic, and pesticides. PAHs are widely distributed in the environment and have been detected in air, water, sediment, soil, and food, particularly charbroiled, broiled or pickled foods, and refined fats and oils.

Use at Oak Ridge:

PAHs were produced as a by-product of coal burning and were associated with coal gasification and coal liquification research at ORNL.

Toxic Effects:

Non-cancer adverse health effects associated with PAH exposure have been observed in animals, although generally, with the exception of adverse effects to the blood and skin, adverse non-cancer effects have not been seen in humans. In animal studies, PAHs have been shown to affect tissues with rapidly multiplying cells, such as bone marrow, organs of the lymphatic system, and intestinal epithelium. In animals, the major target organs are blood-forming and lymphoid systems.

No studies are available regarding cancer in humans following inhalation exposure to PAHs. However, studies have shown an increased likelihood of developing lung cancers following inhalation exposure to coke-oven emissions, cigarette smoke, and roofing-tar emissions, mixtures known to contain several PAHs, as well as other carcinogenic or potentially carcinogenic compounds. Skin cancers have also been observed following dermal exposure to mixtures. Because of the complex nature of these mixtures, it is difficult to determine the effect individual PAHs have on cancer production. PAHs have been shown to induce cancers in animals. The site of tumor induction is influenced by route of administration; i.e., stomach tumors are observed following ingestion, lung tumors following inhalation, and skin tumors following dermal exposure. The USEPA has classified benzo(a)pyrene as a probable (B2) carcinogen. All other PAHs have not been classified by the USEPA as to their carcinogenicity.

INDICATORS OF TOXIC POTENCY

PAHs

EPA Weight-of-Evidence Group	benzo(a)pyrene	B2 (Probable Carcinogen)
Carcinogenic Indicators:		
Slope Factor; inhalation		6.1 (mg/kg-day) ⁻¹
Slope Factor; ingestion	benzo(a)pyrene	7.3 (mg/kg-day) ⁻¹
Noncarcinogenic Indicators:		
Reference Concentration; inhalation	Not determined	
Reference Dose; ingestion, chronic	acenaphthene	0.06 mg/kg-day
	anthracene	0.30 mg/kg-day
	fluoranthene	0.04 mg/kg-day
	fluorene	0.04 mg/kg-day
	pyrene	0.03 mg/kg-day

KEY RADIONUCLIDES AT THE OAK RIDGE RESERVATION

Material Hazards Summary

Key Radionuclides at Oak Ridge and their Sources and Uses:

Barium-140 (12.8 d) is a fission product. It was recovered in large quantities at Oak Ridge from nuclear reactor fuel as a source of radioactive lanthanum-140 (RaLa) for weapons development work.

Cesium-137 (30.0 y) is a fission product. It is important because of its long half-life and the manner in which it readily enters the body. In the past it was separated from irradiated reactor fuel processed at ORNL and purified for commercial and medical uses. Most of the remaining Cs-137 present in reactor fuel processed at ORNL ended up in wastes, with relatively small portions in airborne and waterborne effluents. Cs-137 is a significant fission product that remains in sediments of the White Oak system and the Clinch and Tennessee Rivers after long-term accumulation from X-10 effluents.

Cobalt-60 (5.26 y) is an activation product. In the past it was purposefully formed for commercial and medical uses by neutron irradiation of stable cobalt in ORNL reactors. Co-60 is also formed in operating nuclear reactors when stable cobalt-59 contained in metal components is activated. When the metal corrodes, both Co-59 and Co-60 can be released. As a result, Co-60 was present in effluents of reactors operated at ORNL and in materials received from other sources for processing or disposal at ORNL.

Europium-152, 154, and 155 (12.7 y; 16 y; 1.811 y) are formed by neutron bombardment of stable europium or samarium. In the past europium isotopes were purposefully formed for various applications by neutron irradiation of stable europium in ORNL reactors. Eu-155 is also formed as a fission product.

Iodine-131 (8.05 d) is a fission product. When irradiated reactor fuels were processed at ORNL, iodine-131 was purified for medical and research applications. It was also released to the atmosphere in conjunction with radioactive lanthanum (RaLa) separation. Iodine-131 decays with an 8 day half-life, which tended to decrease the quantities of radioiodine released in effluents and wastes.

Lanthanum-140 (40.22 h) is a fission product and a decay product of barium-140. The requirement for La-140 for weapons development led to processing of short-decayed reactor fuel at Oak Ridge. Iodine-131 and other fission products were released as a result.

Phosphorus-32 (14.3 d) is an activation product. In the past it was purposefully formed for various applications by neutron irradiation of stable sulphur in ORNL reactors. P-32 is also formed in operating nuclear reactors, and as such was present in wastes and effluents of reactors operated at ORNL and in materials received from other sources for processing or disposal at ORNL.

Key Radionuclides at Oak Ridge and their Sources and Uses (continued):

Ruthenium-103 and -106 (39.5 d; 367 d) are fission products. They were present in the irradiated reactor fuel processed at ORNL from ORNL reactors and from off-site sources. The ruthenium present in reactor fuel and fuel processing wastes ended up in ORR effluents and wastes disposed of in ORNL pits, trenches, and burial grounds. Ruthenium is noteworthy because of its mobility in the environment, as was evidenced by significant leakage of Ru-106 from seepage pits in the late 1950s and early 1960s. Particles containing ruthenium were also released to the atmosphere from chemical processing operations.

Strontium-90 (28.1 y) is a fission product. It has been present in the irradiated reactor fuel processed at ORNL from ORNL reactors and from off-site sources. In the past it was separated from irradiated reactor fuel processed at ORNL and purified for commercial applications. Some of the remaining Sr-90 present in reactor fuel and fuel processing wastes ended up in ORR effluents and wastes disposed of in ORNL pits, trenches, and burial grounds. Sr-90 has been significant because of its mobility in the environment after release in liquid effluents or in wastes subjected to on-site disposal.

Technetium-99 (212,000 y) is a fission product. Some of the Tc-99 present in reactor fuel and fuel processing wastes ended up in ORR effluents and wastes disposed of in ORNL pits, trenches, and burial grounds. Tc-99 was also a concern at the K-25 Plant after reactor return material came into use as a uranium feed source for gaseous diffusion. Tc-99 accumulated in areas of the cascades and storage cylinders and appeared in K-25 feed plant and cascade vent gases and liquid effluents.

Transuranic Nuclides (e.g., neptunium, plutonium, americium, curium, and californium) are formed by neutron bombardment of lighter transuranic materials. This has occurred in the fuel of ORNL reactors and in targets placed in ORNL's High Flux Isotope Reactor (HFIR). Plutonium production by neutron bombardment of natural uranium was the primary purpose of the X-10 graphite pile. Some airborne emissions resulted. The transuranic radioactivity present in reactor fuel and fuel processing wastes ended up in ORR effluents and wastes disposed of in ORNL pits, trenches, and burial grounds.

Californium-252 (2.646 y) has been the primary transuranic radionuclide produced at the HFIR by neutron bombardment of plutonium-242 (379,000 y). Neptunium has been recovered from fluorination ash in ORNL's Metal Recovery Plant. Neptunium-237 (214,000,000 y) is often used in reactors to produce plutonium-238 (86.4 y). Am-241 (458 y), a decay product of Pu-241 (13.2 y), was received from other DOE facilities and prepared for commercial distribution.

Tritium (H-3; 12.3 y) is formed by neutron bombardment of a number of elements (such as lithium) and in relatively small quantities by fission of reactor fuel. Some of the H-3 present in reactor fuel and fuel processing wastes ended up in ORR effluents and wastes disposed of in ORNL pits, trenches, and burial grounds. ORNL has also received tritium from other DOE sites for repackaging for commercial distribution. H-3 has also been used in development of

Key Radionuclides at Oak Ridge and their Sources and Uses (continued):

radioluminescent light sources, fabrication of titanium tritide targets for the High Flux Isotope Reactor (HFIR) and research into helium embrittlement of metals.

Uranium Nuclides (e.g., U-233 (162,000 y), U-234 (247,000 y), U-235 (7.1×10^8 y), U-238 (4.51×10^9 y)): Uranium has been used in the fuels of reactors operated at ORNL and in reactors operated off-site that have sent fuel to ORNL for processing. ORNL has also fabricated fuel elements for numerous reactors and developed numerous methods for separation of desirable fission products from uranium-based fuels.

U-233 was also produced in various thorium-based "breeder" nuclear reactors, in which the very abundant thorium-232 absorbed neutrons to form desirable fissionable U-233.

On a weight basis, natural uranium consists of 99.276% U-238, 0.7196% U-235, and 0.0057% U-234. The K-25 Plant enriched uranium in its U-235 content by gaseous diffusion and supported development of centrifugal and atomic vapor laser isotope separation (AVLIS) enrichment methods. The Y-12 Plant in its early years enriched uranium by the electromagnetic process, and in later years chemically processed uranium and produced weapons components from uranium metal and other materials.

RADIONUCLIDE CARCINOGENICITY:

EPA classifies all radionuclides as Group A carcinogens based on their property of emitting ionizing radiation and on the extensive weight of evidence provided by epidemiological studies of radiation-induced cancers in humans.

The International Commission on Radiological Protection (ICRP) recommended in 1990 that an estimate of 5% per sievert (0.05% per rem) be used for the probability of induced fatal cancer in populations of all ages. A smaller value of about 4% per sievert (0.04% per rem) is recommended for a working population of age 20-64 years. These estimates are primarily made for exposure to low dose, low dose rate, and low linear energy transfer (LET) radiation. The probability of fatal cancer for other exposure conditions may be different.

ICRP also recommended in 1990 that an estimate of 7.3% per sievert (0.07% per rem) be used for "detriment" due to radiation exposure of the whole body at low doses. There are four main components of detriment considered by the ICRP: the risk of cancer in all relevant organs, expected years of life lost for induced cancer, morbidity resulting from induced non-fatal cancers, and risk of serious hereditary disease in all future generations descended from the exposed individual.

FACTORS IN ENVIRONMENTAL MOBILITY — The Extent to Which A Nuclide is Adsorbed on Sediment from Solution

The extents to which radionuclides are adsorbed on sediment from solution are indicated by published median freshwater equilibrium distribution coefficients, K_d , which are the ratios of amount adsorbed on sediment to amount left in solution. Values such as these reflect which elements tend to remain in solution, and which become bound in river-bottom, lake-bottom, or shoreline sediments. Elements that remain in solution are often washed away in rivers or diluted in the water contained in lakes. Elements which become sediment bound can in some cases provide a record of past releases in layers of accumulation. Radionuclides in sediments or in solution can result in exposure from drinking water, swimming, boating, or shoreline visitation. Dredged sediments can result in various exposure scenarios depending on placement of the spoils.

Selected Radionuclides in Order of Decreasing Tendency to Adsorb

<u>Element</u>	<u>Median K_d</u>
plutonium	100,000
cerium	10,000
thorium	10,000
americium	5,000
curium	5,000
cobalt	5,000
cesium*	1,000
strontium**	1,000
zirconium	1,000
europium	500
neptunium	0.2 to 127
iodine	10
technetium	5
ruthenium***	variable
tritium	0

* Site-specific research indicated values of 800-1000 for White Oak Creek and 1360 for the Clinch River.

** Site-specific research indicated a range of 100-150 for White Oak Creek.

*** Site-specific research indicated a value of 1 or less for White Oak Creek.

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FACTORS IN ENVIRONMENTAL PERSISTENCE — RADIONUCLIDE HALF-LIVES
Selected Radionuclides in Order of Increasing Half-Lives

La-140	1.7 d
Xe-133	5.3 d
I-131	8.1 d
Ba-140	13 d
P-32	14 d
Nb-95	35 d
Ru-103	40 d
Sr-89	52 d
Zr-95	66 d
Se-75	120 d
Cm-242	160 d
Co-57	270 d
Ce-144	280 d
Ru-106	370 d
Eu-155	1.8 y
Th-228	1.9 y
Cs-134	2.1 y
Cf-252	2.6 y
Co-60	5.3 y
Kr-85	11 y
H-3	12 y
Eu-152	13 y
Pu-241	13 y
Eu-154	16 y
Cm-244	18 y
Sr-90	28 y
Cs-137	30 y
Cm-243	32 y
U-232	72 y
Pu-238	86 y
Am-241	460 y
C-14	5700 y
Pu-240	6600 y
Pu-239	24,000 y
U-233	160,000 y
Tc-99	220,000 y
U-234	250,000 y
Np-237	2,100,000 y
U-235	710,000,000 y
U-238	4,500,000,000 y
Th-232	14,000,000,000 y

FACTORS IN ENVIRONMENTAL PERSISTENCE — RADIONUCLIDE HALF-LIVES

Selected Radionuclide Half-Lives

Radionuclides listed in alphabetical order

Am-241	460 y
Ba-140	13 d
C-14	5700 y
Ce-144	280 d
Cf-252	2.6 y
Cm-242	160 d
Cm-243	32 y
Cm-244	18 y
Co-57	270 d
Co-60	5.3 y
Cs-134	2.1 y
Cs-137	30 y
Eu-152	13 y
Eu-154	16 y
Eu-155	1.8 y
H-3	12 y
I-131	8.1 d
Kr-85	11 y
La-140	1.7 d
Nb-95	35 d
Np-237	2,100,000 y
P-32	14 d
Pu-238	86 y
Pu-239	24,000 y
Pu-240	6600 y
Pu-241	13 y
Ru-103	40 d
Ru-106	370 d
Se-75	120 d
Sr-89	52 d
Sr-90	28 y
Tc-99	210,000 y
Th-228	1.9 y
Th-232	14,000,000,000 y
U-232	72 y
U-233	160,000 y
U-234	250,000 y
U-235	710,000,000 y
U-238	4,500,000,000 y
Xe-133	5.3 d
Zr-95	66 d

RELATIVE RADIATION DOSE WHEN INHALED

INHALATION DOSE FACTORS: Committed Effective Dose Equivalent, Sv per microgram or becquerel inhaled

In Descending Order
Highest Organ Listed, but Dose Factors are Effective

Ranked by Dose per Unit Weight			Ranked by Dose per Unit Activity	
NUCLIDE	Sv/microgram Inhaled	Highest Organ*	NUCLIDE	CEDE, Sv/Bq Inhaled**
Cf-252	735	Bone Surface/Lung	Th-232	4.43E-04
Cm-242	574	Bone Surface	Np-237	1.46E-04
Cm-244	207	Bone Surface	Am-241	1.20E-04
Cm-243	141	Bone Surface	Pu-240	1.16E-04
Pu-238	68	Bone Surface	Pu-239	1.16E-04
P-32	44	Red Marrow/Lung	Pu-238	1.06E-04
I-131	41	Thyroid	Cm-243	8.30E-05
Ru-103	29	Remainder/Lung	Cm-244	6.70E-05
La-140	27	Lung/Remainder	Cf-252	3.70E-05
Ru-106	16	Lung/Lung	U-233	3.66E-05
Am-241	14	Bone Surface	U-234	3.58E-05
Ce-144	12	Lung/Lung	U-235	3.32E-05
Sr-89	12	Bone Surface/Lung	U-238	3.20E-05
Pu-241	9.2	Bone Surface	Cm-242	4.67E-06
Zr-95	5.0	Bone Surface/Lung	Pu-241	2.23E-06
Ba-140	2.7	Bone Surface	Sr-90	3.51E-07
Co-60	2.5	Lung/Lung	Ru-106	1.29E-07
Nb-95	2.3	Lung/Lung	Ce-144	1.01E-07
Sr-90	1.8	Bone Surface/Lung	Eu-154	7.73E-08
Se-75	1.2	Remainder/Lung	Eu-152	5.97E-08
Pu-240	1.0	Bone Surface	Co-60	5.91E-08
Co-57	0.80	Lung/Lung	Cs-134	1.25E-08
Cs-134	0.60	Remainder	Sr-89	1.12E-08
Eu-155	0.53	Bone Surface	Eu-155	1.12E-08
Eu-154	0.42	Bone Surface	I-131	8.89E-09
Eu-152	0.41	Bone Surface	Cs-137	8.63E-09
Pu-239	0.26	Bone Surface	Zr-95	6.39E-09
Cs-137	0.028	Remainder	P-32	4.19E-09
U-233	0.013	Bone Surface/Lung	Co-57	2.45E-09
H-3	0.0062	All Organs	Ru-103	2.42E-09
Np-237	0.0038	Bone Surface	Se-75	2.29E-09
C-14	0.000093	All Organs	Tc-99	2.25E-09
U-234	0.000028	Bone Surface/Lung	Nb-95	1.57E-09
Th-232	0.000018	Bone Surface	La-140	1.31E-09
U-235	0.000026	Bone Surface/Lung	Ba-140	1.01E-09
Tc-99	0.000014	Stomach Wall/Lung	C-14	5.64E-10
U-238	0.0000039	Bone Surface/Lung	H-3	1.73E-11

* Some nuclides have multiple highest organs depending on solubility.

** Notation: 4.43E-04 = 4.43 times 10 raised to the -4 power.

RELATIVE RADIATION DOSE WHEN INGESTED

INGESTION DOSE FACTORS: Committed Effective Dose Equivalent, Sv per microgram or becquerel ingested

In Descending Order

Highest Organ Listed, but Dose Factors are Effective

<i>Ranked by Dose per Unit Weight</i>			<i>Ranked by Dose per Unit Activity</i>	
NUCLIDE	Sv/microgram Ingested	Highest Organ*	NUCLIDE	Sv/Bq Ingested**
I-131	66.07	Thyroid	Np-237	1.20E-06
La-140	46.99	Remainder	Am-241	9.84E-07
P-32	24.99	Red Marrow	Pu-240	9.56E-07
Ru-103	9.79	Remainder	Pu-239	9.56E-07
Ba-140	6.91	Large Intestine Wall	Pu-238	8.65E-07
Cf-252	5.82	Bone Surface	Th-232	7.38E-07
Cm-242	3.81	Bone Surface	Cm-243	6.79E-07
Sr-89	2.61	Remainder/LLI Wall	Cm-244	5.45E-07
Cm-244	1.68	Bone Surface	Cf-252	2.93E-07
Se-75	1.39	Remainder	U-233	7.81E-08
Cm-243	1.16	Bone Surface	U-234	7.66E-08
Nb-95	1.01	Remainder	U-235	7.19E-08
Cs-134	0.95	Remainder	U-238	6.88E-08
Ru-106	0.92	LLI Wall	Sr-90	3.85E-08
Zr-95	0.79	Remainder	Cm-242	3.10E-08
Ce-144	0.67	LLI Wall	Cs-134	1.98E-08
Pu-238	0.56	Bone Surface	Pu-241	1.85E-08
Co-60	0.30	Remainder	I-131	1.44E-08
Sr-90	0.20	Bone Surface	Cs-137	1.35E-08
Am-241	0.12	Bone Surface	Ru-106	7.40E-09
Co-57	0.10	Remainder	Co-60	7.28E-09
Pu-241	0.08	Bone Surface	Ce-144	5.68E-09
Cs-137	0.04	Remainder	Se-75	2.60E-09
Eu-155	0.02	Bone Surface	Eu-154	2.58E-09
Eu-154	0.014	Remainder	Ba-140	2.56E-09
Eu-152	0.012	Remainder	Sr-89	2.50E-09
Pu-240	0.008	Bone Surface	P-32	2.37E-09
H-3	0.006	All Organs	La-140	2.28E-09
Pu-239	0.002	Bone Surface	Eu-152	1.75E-09
C-14	0.0001	All Organs	Zr-95	1.02E-09
Np-237	0.000031	Bone Surface	Ru-103	8.24E-10
U-233	0.000027	Bone Surface	Nb-95	6.95E-10
Tc-99	0.00000025	Thyroid	C-14	5.64E-10
U-234	0.00000001	Bone Surface	Eu-155	4.13E-10
Th-232	0.0000000030	Bone Surface	Tc-99	3.95E-10
U-235	0.0000000009	Bone Surface	Co-57	3.20E-10
U-238	0.0000000008	Bone Surface	H-3	1.73E-11

* Some nuclides have multiple highest organs depending on solubility.

** Notation: 1.20E-06 = 1.2 times 10 raised to the -6 power.

LLI Wall = lower large intestine wall.

Reference: USEPA Federal Guidance Report No. 11

Material Hazards Summary

URANIUM (AS A CHEMICAL TOXIN)

See also: Key Radionuclides at the Oak Ridge Reservation

Common Forms:

UF₆, U₃O₈, UO₂, UO₃, and UF₄

Nature of Use and Presence in the Environment:

Uranium is a naturally occurring element present at varying levels in soil and rock. Small amounts of natural uranium are used in ceramics, glass, and photographic material. Uranium may be present in groundwater and surface water due to leaching from rock and runoff from agricultural lands treated with uranium-containing fertilizers.

Toxic Effects:

Adverse health effects due to uranium exposure are of two types: radiological and chemical. Because of the low radioactivity of natural uranium, radiological effects are expected to be virtually absent. Studies have shown that lung cancers are the most common types of cancer among uranium miners. However, these cancers are thought to be due to exposure to radon and its decay products, which are also present in uranium mines, although one study did show an increase in deaths from lung cancer in workers not exposed to radon. With the exception of a single study which suggests that uranium produces changes in the lungs and lymph nodes of monkeys and dogs, no animal studies have demonstrated adverse health effects associated with exposure to natural uranium.

The primary acute toxic effect following exposure to soluble uranium compounds is to the kidneys. Increases in deaths due to kidney and liver damage were reported for uranium millers and miners. In animal studies, inhalation, oral, or dermal exposure to soluble uranium compounds resulted in kidney damage. Uranium has been shown to affect the respiratory system-- uranium miners and processors have shown an increase in deaths from respiratory disease. However, as with the development of lung cancers, this increase may be related to cigarette smoking or the effects of radon and its decay products. Lung fibrosis has been observed in animals following exposure to uranium.

Animal studies have shown that the toxicity of uranium compounds is dependent on solubility in body fluids.

INDICATORS OF TOXIC POTENCY

URANIUM

EPA Weight-of-Evidence Group	A (Human Carcinogen)
<i>Regarding carcinogenicity, see also uranium radionuclides</i>	
Carcinogenic Indicators:	
Slope Factor; inhalation	Not applicable (see radionuclide section)
Slope Factor; ingestion	Not applicable
Noncarcinogenic Indicators:	
Reference Concentration; inhalation	Not determined
Reference Dose; ingestion	0.003 mg/kg-day (soluble uranium salts)

3.0 SELECTED REFERENCE MATERIAL

All of the selected reference material presented hereafter has been photocopied directly from the cited sources. Because of this, there is some diversity of typeface and formatting between the sections. There has been an effort to minimize reference to other sections of the reports that are not provided here. However, some may still exist, and can be resolved by referring to the source document.

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ARSENIC

Source: ATSDR, 1991. "Draft Toxicological Profile for Arsenic." Agency for Toxic Substances and Disease Registry, Atlanta, GA. October, 1991.

1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about arsenic and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,300 sites on its National Priorities List (NPL). Arsenic has been found in at least 429 of these sites. However, we do not know how many of the 1,300 NPL sites have been evaluated for arsenic. As EPA evaluates more sites, the number of sites at which arsenic is found may change. This information is important for you to know because arsenic may cause harmful health effects and because these sites are potential or actual sources of human exposure to arsenic.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous chemical such as arsenic, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS ARSENIC?

Arsenic is a naturally-occurring element. Pure arsenic is a gray metal-like material, but this form is not common in the environment. Rather, arsenic is usually found combined with other elements such as oxygen, chlorine, and sulfur. Arsenic combined with these elements is called inorganic arsenic. Arsenic combined with carbon and hydrogen is called organic arsenic. You should know the difference between inorganic and organic arsenic because the organic forms are usually less harmful than the inorganic forms.

Most inorganic and organic arsenic compounds are white or colorless powders that do not evaporate. They have no smell, and most have no special taste. Thus, you usually cannot tell if arsenic is present in your food, water, or air.

Inorganic arsenic occurs naturally in many kinds of rock, especially in ores that contain copper or lead. When these ores are heated at smelters to get the copper or lead, most of the arsenic enters the air as a fine dust. Smelters collect this dust and purify the

1. PUBLIC HEALTH STATEMENT

arsenic for several uses. The main use is as a preservative for wood to make it resistant to rotting and decay. Arsenic is also used to make several types of insect killers and weed killers, such as Ansar®, Scorch®, Phytar®, Bueno®, Crab-E-Rad®, Premix®, and others.

You can find more information on the sources, properties, and uses of arsenic in Chapters 3 and 4.

1.2 WHAT HAPPENS TO ARSENIC WHEN IT ENTERS THE ENVIRONMENT?

Arsenic can enter the environment in several ways. Even though it does not evaporate, arsenic can get into air as dust. This can happen when smelters heat ores containing arsenic, when people burn any material containing arsenic, or when wind blows soil that contains arsenic into the air. Once in the air, the arsenic particles will travel with the wind for a while, but will then settle back to the ground. Most arsenic compounds can also dissolve in water. Thus, arsenic can get into lakes, rivers, or underground water by dissolving in rain or snow, or through the discharge of industrial wastes. Some of the arsenic will stick to the sediment on the bottom of the lake or river, and some will be carried along by the water.

Because arsenic is an element, it is not broken down or destroyed in the environment. However, it can change from one form to another by natural chemical reactions, and also by the action of bacteria that live in soil or water. Although some fish and shellfish build up arsenic in their tissues, most of this is in a form (often called "fish arsenic") that is not toxic.

You can find more information on how arsenic gets into the environment and how it behaves in air, soil, and water in Chapters 4 and 5.

1.3 HOW MIGHT I BE EXPOSED TO ARSENIC?

Because arsenic is a natural part of the environment, low levels of arsenic are present in soil, water, food, and air. Soil usually contains the most, with average levels of about 5,000 parts of arsenic per billion parts of soil (ppb). Levels in food are usually about 20-140 ppb and levels in water are about 2 ppb. Levels in air are usually about 0.02-0.10 micrograms per cubic meter. Thus, you normally take in small amounts of arsenic in the air you breathe, the water you drink, and the food you eat. Of these, food is usually the largest source. You are also likely to swallow small amounts of dust or dirt each day, so this is another way you can be exposed to arsenic. The total amount you take in from these sources is probably about 50 micrograms each day.

1. PUBLIC HEALTH STATEMENT

In addition to the normal levels of arsenic in air, water, soil, and food, you could be exposed to higher levels in several ways, such as the following:

- Some areas of the country contain unusually high natural levels of arsenic in rock, and this can lead to unusually high levels of arsenic in soil or water. If you live in an area like this, you could take in above-average amounts of arsenic from the soil or from the water.
- Some hazardous waste sites contain large quantities of arsenic. If the material is not properly disposed of, it can get into surrounding water, air, or soil. If you live near such a site, you could be exposed to above-average levels of arsenic from these media.
- If you work in an occupation that involves arsenic production or use (for example, copper or lead smelting, wood treating, pesticide application), you could be exposed to above-average levels of arsenic during your work. The government estimates that about 55,000 people may be exposed in this way.
- If you saw or sand arsenic-treated wood, you could inhale some of the sawdust into your nose or throat. Similarly, if you burn arsenic-treated wood, you could inhale arsenic in the smoke.
- In the past, several kinds of products used in the home (rat poison, ant poison, weed killer, some types of medicines) had arsenic in them. However, most of these uses of arsenic have ended, so you are not likely to be exposed from home products any longer.

You can find more information on how you may be exposed to arsenic in Chapter 5.

1.4 HOW CAN ARSENIC ENTER AND LEAVE MY BODY?

If you swallow arsenic in water, soil, or food, most of the arsenic quickly enters into your body. This is the most likely way for you to be exposed near a waste site. If you breathe air that contains arsenic dusts, many of the dust particles settle onto the lining of the lungs. Most of the arsenic in these particles is then taken up into the body. You might be exposed in this way near waste sites where arsenic-contaminated soils are allowed to blow into the air. If you get arsenic-contaminated soil or water on your skin, only a small amount will go through your skin into your body, so this is usually not of concern.

1. PUBLIC HEALTH STATEMENT

If you are exposed to arsenic, your liver changes some of this to a less harmful organic form. Both inorganic and organic forms leave your body in your urine. Most of the arsenic will be gone within several days, although some will remain in your body for several months or even longer.

You can find more information on how arsenic enters and leaves your body in Chapter 2.

1.5 HOW CAN ARSENIC AFFECT MY HEALTH?

Inorganic arsenic has been recognized as a human poison since ancient times, and large oral doses (above 60,000 ppb in food or water) can produce death. If you swallow lower levels of inorganic arsenic (ranging from about 300 to 30,000 ppb in food or water), you may experience irritation of your stomach and intestines, with symptoms such as pain, nausea, vomiting, and diarrhea. Other effects you might experience from swallowing arsenic include decreased production of red and white blood cells, abnormal heart function, blood-vessel damage, and impaired nerve function causing a "pins and needles" sensation in your hands and feet. Although there is no good evidence that arsenic can injure pregnant women or their fetuses, studies in animals show that doses of arsenic that are large enough to cause illness in pregnant females may cause low birth weight, fetal malformations, or even fetal death.

Perhaps the single most characteristic effect of long-term oral exposure to inorganic arsenic is a pattern of skin changes. This includes a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. While these skin changes are not considered to be a health concern in their own right, a small number of the corns may ultimately develop into skin cancer. Swallowing arsenic has also been reported to increase the risk of cancer in the liver, bladder, kidney, and lung. The Department of Health and Human Services has determined that arsenic and certain arsenic compounds are known carcinogens.

If you breathe high levels of inorganic arsenic, you are likely to experience a sore throat and irritated lungs. You may also develop some of the skin effects mentioned above. The exposure level that produces these effects is uncertain, but is probably above 100 micrograms per cubic meter. However, these effects are usually not serious. Of much greater concern is the ability of inhaled inorganic arsenic to increase the risk of lung cancer. This has been seen mostly in humans exposed to arsenic in or around smelters. People who live near smelters, chemical factories, or waste sites with arsenic may have increased risk of lung cancer as well.

If you have direct skin contact with inorganic arsenic compounds, your skin may become irritated with some redness and swelling. However, it does not appear that skin contact is likely to lead to any serious internal effects.

1. PUBLIC HEALTH STATEMENT

Despite all the adverse health effects associated with inorganic arsenic exposure, there is some evidence that the small amounts of arsenic in the normal diet (10-50 ppb) may be beneficial to your health. For example, animals fed a diet with unusually low concentrations of arsenic did not gain weight normally. They also became pregnant less frequently than animals fed a diet containing a normal amount of arsenic. Further, the offspring from these animals tended to be smaller than normal, and some died at an early age. However, no cases of arsenic deficiency in humans have ever been reported.

Almost no information is available on the effects of organic arsenic compounds in humans. Studies in animals show that most organic arsenic compounds are less toxic than the inorganic forms. However, high doses can produce some of the same effects. Thus, if you are exposed to high doses of an organic arsenic compound, you might develop nerve injury, stomach irritation, or other effects, but this is not known for certain.

You can find more information on the health effects of inorganic and organic arsenic in Chapter 2.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO ARSENIC?

Several sensitive and specific tests can measure arsenic in your blood, urine, hair, or fingernails, and these tests are often helpful in determining if you have been exposed to above-average levels of arsenic. These tests are not usually performed in a doctor's office, but require sending the sample to a testing laboratory.

Measurement of arsenic in your urine is the most reliable means of detecting arsenic exposures that you experienced within the last several days. Most tests measure the total amount of arsenic present in your urine. Sometimes this can be misleading, because the nonharmful forms of arsenic in fish and shellfish can give a high reading even if you have not been exposed to a toxic form of arsenic. For this reason, laboratories sometimes use a more complicated test to separate "fish arsenic" from other forms. Because most arsenic leaves your body within a few days, analysis of your urine cannot detect if you were exposed to arsenic in the past. Tests of your hair or fingernails can tell if you were exposed to high levels over the past 6-12 months, but these tests are not very useful in detecting low level exposures. If high levels of arsenic are detected, this shows that you have been exposed, but unless more is known about when you were exposed and for how long, it is usually not possible to predict whether you will have any harmful health effects.

You can find more information on how arsenic can be measured in your hair, urine, nails, and other tissues in Chapters 2 and 6.

1. PUBLIC HEALTH STATEMENT

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The federal government has taken several steps to protect humans from arsenic. First, EPA has set limits on the amount of arsenic that industrial sources can release into the environment. Second, EPA has restricted or canceled many of the uses of arsenic in pesticides and is considering further restrictions. Third, EPA has set a limit of 50 ppb for arsenic in drinking water. EPA is currently reviewing this value and may lower it. Finally, the Occupational Safety and Health Administration (OSHA) has established a maximum permissible exposure limit of 10 micrograms per cubic meter for airborne arsenic in various workplaces that use inorganic arsenic.

You can find more information on regulations and guidelines that apply to arsenic in Chapter 7.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. These clinics specialize in the recognition, evaluation, and treatment of illnesses resulting from exposure to hazardous substances.

ASBESTOS

Source: ATSDR, 1990. "Toxicological Profile for Asbestos." Agency for Toxic Substances and Disease Registry, Atlanta, GA. December, 1990.

1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about asbestos and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,177 sites on its National Priorities List (NPL). Asbestos has been found at 28 of these sites. However, we do not know how many of the 1,177 NPL sites have been evaluated for asbestos. As EPA evaluates more sites, the number of sites at which asbestos is found may change. The information is important for you to know because asbestos may cause harmful health effects and because these sites are potential or actual sources of human exposure to asbestos.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous substance such as asbestos, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS ASBESTOS?

Asbestos is the name applied to a group of six different minerals (amosite, chrysotile, tremolite, actinolite, anthophyllite, and crocidolite) that occur naturally in the environment. The most common mineral type is white (chrysotile), but others may be blue (crocidolite), gray (anthophyllite), or brown (amosite). These minerals are made up of long, thin fibers that appear somewhat similar to fiber glass. Asbestos fibers are very strong and are resistant to heat and chemicals. Because of these properties, asbestos fibers have been used in a wide range of products, mostly in building materials, friction products, and heat-resistant fabrics. Because the fibers are so resistant to chemicals, they are also very stable in the environment; they do not evaporate into air or dissolve in water, and they are not broken down over time.

Further information on the properties, uses, and behavior of asbestos in the environment may be found in Chapters 3, 4, and 5.

1.2 HOW MIGHT I BE EXPOSED TO ASBESTOS?

You are most likely to be exposed to asbestos by breathing in tiny asbestos fibers suspended in air. These fibers can come from natural outcroppings of asbestos, but many come from the degradation or breakdown of man-made products such as insulation, ceiling and floor tiles, roof shingles, cement, automotive brakes and clutches, and many others. Low levels of asbestos can be detected in almost any air sample. For example, in rural areas, there are usually an average of around 0.03 to 3 fibers present in a cubic meter (f/m^3) of outdoor air*. (A cubic meter is about the amount of air you breathe in 1 hour). Higher levels are usually found in cities, where there may be 3 to 300 f/m^3 . Close to an asbestos mine or factory, levels could reach 2,000 f/m^3 or higher. Levels could also be above average near a building that is being torn down or renovated, or near a waste site where asbestos is not properly covered up or stored to protect it from wind erosion.

In indoor air, the concentration of asbestos depends on whether asbestos was used for insulation, ceiling or floor tiles, or other purposes, and whether these asbestos-containing materials are in good condition or are deteriorated and easily crumbled. Concentrations measured in homes, schools, and other buildings that contain asbestos range from 30 to 6,000 f/m^3 . People who work with asbestos (e.g., miners, insulation workers, automobile brake mechanics) are likely to be exposed to much higher levels of asbestos particles in air.

You can also be exposed to asbestos by drinking fibers present in water. Even though asbestos does not dissolve in water, fibers can enter water by being eroded from natural deposits or piles of waste asbestos, or from cement pipes used to carry drinking water. Most drinking water supplies in the United States have concentrations less than 1 million fibers per liter (MFL)*. (A liter is about the same as a quart). However, in some locations, there may be 10 to 100 MFL or even higher.

*The number of fibers depends on how they are measured. Values in air are reported as phase contrast fibers per cubic meter. This is the same measure as used to describe health effects in Section 1.5. The values in water are reported as transmission electron microscope fibers. This method is more sensitive than phase contrast microscopy, so values in air and water are not comparable.

Further information on how you could be exposed to asbestos is presented in Chapters 2 and 5.

1.3 HOW CAN ASBESTOS ENTER AND LEAVE MY BODY?

If you breathe asbestos fibers into your lungs, some of the fibers will be deposited in the air passages and on the cells that make up your lungs. However, very few of these fibers move through your lungs into your body. Instead, most fibers are removed from your lungs by being carried away in a layer of mucus to the throat, where they are swallowed into the stomach. This usually takes place within a few hours, but fibers that are deposited in the deepest parts of the lung are removed more slowly, and some can remain in place for many years and may never be removed.

If you swallow asbestos fibers (either those present in water or those that are moved to your throat from your lungs), nearly all the fibers pass along your intestines within a few days and are excreted in the feces. A small number of fibers become stuck in the cells that line your stomach or intestines, and a few penetrate all the way through and get into the blood. Some of these become trapped in other tissues, and some are removed in the urine.

Further information on what happens to asbestos fibers in the lung and the stomach may be found in Chapter 2.

1.4 HOW CAN ASBESTOS AFFECT MY HEALTH?

Information on the health effects of asbestos in humans comes mostly from studies of people who were exposed in the past to high levels of asbestos in the workplace. These asbestos workers were found to have increased chances of getting two types of cancer: cancer of the lung tissue itself, and mesothelioma, a cancer of the thin membrane that surrounds the lung and other internal organs. Both lung cancer and mesothelioma are usually fatal. These diseases do not appear immediately, but develop only after a number of years. There is also some evidence from studies of workers that breathing asbestos can increase the chances of getting cancer in other locations (e.g., stomach, intestines, esophagus, pancreas, kidneys), but this is less certain. Members of the public who are exposed to lower levels of asbestos may also have increased chances of getting cancer, but the risks are usually small and are difficult to measure directly.

Besides causing cancer, breathing asbestos can also cause a slow accumulation of scar-like tissue in the lungs and in the membrane which surrounds the lungs. This scar-like tissue does not expand and contract like normal lung tissue, and so breathing becomes difficult. Blood flow to the lung may also be decreased, and this causes the heart to enlarge. When the injury is mostly in the lung itself, the disease is called asbestosis. This is a serious disease, and can eventually lead to

disability or death in people exposed to high levels of asbestos. However, asbestosis is not usually of concern to people exposed to low levels of asbestos. Similar injury to the membrane surrounding the lung is quite common in people exposed to asbestos, but effects on breathing are usually not serious.

The health effects from swallowing asbestos are unclear. Some groups of people who have been exposed to asbestos fibers in their drinking water have higher-than-average death rates from cancer of the esophagus, stomach, and intestines. However, it is very difficult to tell whether this is caused by asbestos or by something else. Animals that were given very high doses of asbestos in food did not get any more fatal cancers than usual, although some extra nonfatal tumors did occur in the intestines of rats in one study.

Further information on how asbestos can affect your health is given in Chapter 2.

1.5 WHAT LEVELS OF EXPOSURE HAVE RESULTED IN HARMFUL HEALTH EFFECTS?

The levels of asbestos in air that lead to lung disease depend on a large number of factors. The most important of these are 1) how long you were exposed, 2) how long it has been since your exposure started, and 3) whether you smoked cigarettes. Also, there is a scientific debate concerning the differences in the amount of disease caused by different fiber types and sizes. Some of these differences may be due to the physical and chemical properties of the different fiber types. There are several studies which suggest that amphiboles (tremolite, amosite, and especially crocidolite) may be more potent than chrysotile. However, most data indicate that fiber size is the most important factor for cancer causing potential. Most studies indicate that long fibers (where "long" means greater than about 1/5,000th of an inch) are more likely to cause injury than short fibers (where "short" means less than about 1/10,000th of an inch). Tables 1-1 and 1-2 show the levels of asbestos that have been found to cause noncancer lung injury in humans and animals, respectively. It is important to stress that the values shown in these tables are the lowest exposures known to have caused noncancer effects, and that not all people exposed to the doses in Table 1-1 would develop the effects.

As noted above, eating or drinking asbestos fibers may increase risk of cancer, but this is not certain. Eating or drinking asbestos fibers is not thought to cause any harmful noncancer effects. This is summarized in Tables 1-3 and 1-4.

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TABLE 1-1. Human Health Effects from Breathing Asbestos*

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of humans to air containing specific levels of asbestos are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Air (f/m³)</u>	<u>Length of Exposure</u>	<u>Description of Effects**</u>
1,000,000	20 years	Lung injury in some people.
3,400,000	20 years	Death from asbestosis in some people.

*See Section 1.2 for a discussion of exposures encountered in daily life.

**These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

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TABLE 1-2. Animal Health Effects from Breathing Asbestos

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of animals to air containing specific levels of asbestos are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Air (f/m³)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
54,000,000	2 years	Mild lung injury in rats.
1,100,000,000	2 years	More severe lung injury in rats.

*These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

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TABLE 1-3. Human Health Effects from Eating or Drinking Asbestos

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of humans to food containing specific levels of asbestos are not known.
<u>Levels in Water</u>		The health effects resulting from short-term exposure of humans to water containing specific levels of asbestos are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term exposure of humans to food containing specific levels of asbestos are not known.
<u>Levels in Water</u>		The health effects resulting from long-term exposure of humans to water containing specific levels of asbestos are not known.

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TABLE 1-4. Animal Health Effects from Eating or Drinking Asbestos

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of animals to food containing specific levels of asbestos are not known.
<u>Levels in Water</u>		The health effects resulting from short-term exposure of animals to water containing specific levels of asbestos are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term exposure of animals to food containing specific levels of asbestos are not known.
<u>Levels in Water</u>		The health effects resulting from long-term exposure of animals to water containing specific levels of asbestos are not known.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO ASBESTOS?

The most common test used to determine if you have been exposed to asbestos is a chest x-ray. The x-ray cannot detect the asbestos fibers themselves, but can detect early signs of lung disease caused by asbestos. While other things besides asbestos can sometimes produce similar changes in the lungs, this test is usually reliable for detecting asbestos-related effects.

It is also possible to test for the presence of asbestos fibers in urine, feces, mucus, or material rinsed out of the lung by a doctor. Low levels of asbestos fibers are found in these materials for nearly all people. Higher-than-average levels can show that you have been exposed to asbestos, but it is not yet possible to use the results to estimate how much asbestos you have been exposed to, or to predict whether you are likely to suffer any health effects.

Further information about how asbestos can be measured in people and in the environment is presented in Chapter 6.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

Despite the ongoing debate concerning health effects resulting from the different asbestos fiber types, ATSDR considers the different mineral forms of asbestos to be known human cancer causing substances with a prolonged latency period of between 10 and 30 years between exposure and the onset of disease. The federal government has taken a number of steps to protect citizens from exposure to asbestos. First, the U.S. Environmental Protection Agency (EPA) has established a very broad ban on the manufacture, processing, importation, and distribution of materials or products that contain asbestos. These regulations were initiated in 1990, and will be in full force by 1997. This ban will result in elimination of asbestos in insulation, brakes, floor and ceiling tiles, cement, paper, and nearly all other asbestos-containing materials. Second, EPA has established regulations that require school systems to investigate whether asbestos exposure is a problem inside their school buildings, and if so, to reduce or eliminate the exposure, either by removing the asbestos or by covering it up so it cannot get into air. In addition, EPA provides guidance and support for reducing asbestos exposure in other public buildings. Third, EPA regulates the release of asbestos from factories and during building demolition or renovation to prevent asbestos from getting into the environment. The EPA also regulates the disposal of waste asbestos materials or products, requiring these to be placed only in approved locations. Fourth, EPA has proposed a limit of 7 million fibers per liter on the concentration of long fibers that may be present in drinking water. Fifth, the Food and Drug Administration regulates the use of asbestos in the preparation of drugs, and restricts the use of asbestos in food-packaging materials.

Finally, the Occupational Safety and Health Administration has established a limit of 200,000 fibers/m³ on the average daily concentration of asbestos allowed in air in the workplace.

Further information about regulations and guidelines to protect people from asbestos is presented in Chapter 7.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns not covered here, please contact your State Health or Environmental Department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road, E-29
Atlanta, Georgia 30333

This agency can also give you information on the location of the nearest occupational and environmental health clinics. Such clinics specialize in recognizing, evaluating, and treating illnesses that result from exposure to hazardous substances.

BERYLLIUM

Source: ATSDR, 1991. "Draft Toxicological Profile for Beryllium." Agency for Toxic Substances and Disease Registry, Atlanta, GA. October, 1991.

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This Statement was prepared to give you information about beryllium and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,300 sites on its National Priorities List (NPL). Beryllium has been found in at least 56 of these sites. However, we do not know how many of the 1,300 NPL sites have been evaluated for beryllium. As EPA evaluates more sites, the number of sites at which beryllium is found may change. This information is important for you to know because beryllium may cause harmful health effects and because these sites are potential or actual sources of human exposure to beryllium.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous chemical such as beryllium, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS BERYLLIUM?

Beryllium is a hard, grayish element that does not occur naturally. The element does occur as a chemical component of certain rocks, soil, and volcanic dust. Two kinds of mineral rocks, bertrandite and beryl, are mined commercially for the recovery of beryllium. Very pure gem-quality beryl is better known as either aquamarine (blue or blue-green) or emerald (green). Beryllium is also present in a variety of compounds. They do not have any particular smell. There are two types of beryllium compounds, those that dissolve in water and those that do not.

Most of the beryllium ore that is mined is converted into alloys (mixtures of metals). Most of these alloys are used in making electrical and electronic parts or as construction materials for machinery and molds for plastics. Pure beryllium metal has applications in nuclear weapons and reactors, aircraft and space vehicle structures and instruments, X-ray machines, and mirrors. Beryllium oxide is also made from beryllium ores and is used to make specialty ceramics for electrical and high-technology applications. More information

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on the chemical and physical properties, and production and use is found in Chapters 3 and 4.

1.2 WHAT HAPPENS TO BERYLLIUM WHEN IT ENTERS THE ENVIRONMENT?

Beryllium enters the air, water, and soil as a result of natural and human activities. Emissions from burning coal and oil increase beryllium levels in air. Beryllium enters waterways from the wearing away of rocks and soil. Most of the beryllium that enters waterways comes when industry dumps waste water. Beryllium, as a chemical component, occurs naturally in soil; however, disposal of coal ash, incinerator ash, and industrial wastes may increase the concentration of beryllium in soil. In air, beryllium compounds are present mostly as fine dust particles. The dust eventually settles over land and water. Rain and snow aid in the removal of beryllium from air. Beryllium particles may remain airborne for about 10 days. Most of the beryllium in water settles in the material on the bottom. Beryllium compounds remain in ocean water for a few hundred years before settling to the bottom of the ocean. Fish do not accumulate beryllium from water into their bodies, to any great extent. A major portion of beryllium in soil does not dissolve in water but remains bound to soil, so it is not very likely to move deeper into the ground and enter groundwater. In the environment, chemical reactions can change the water-soluble beryllium compounds (substance formed by joining two or more chemicals) into insoluble forms. In some cases, water-insoluble beryllium compounds can change to soluble forms. Exposure to water-soluble beryllium compounds in the environment, in general, will pose a greater threat to human health than water-insoluble forms. More information about the fate and movement of beryllium in the environment is found in Chapter 5.

1.3 HOW MIGHT I BE EXPOSED TO BERYLLIUM?

You can be exposed to low levels of beryllium by breathing air, eating food, or drinking water that contains beryllium. In the United States, the average concentration of beryllium in air is 0.03 nanograms (ng) (1 ng = 1 billionth of a gram) in a cubic meter (ng/m^3) of air. In U.S. cities, the average air concentration is higher, and its value is $0.2 \text{ ng}/\text{m}^3$ of air. Beryllium is not found in 95% of 1,577 drinking water samples obtained throughout the United States. In the samples that were found to contain beryllium, the average beryllium concentration is only 190 ng in a liter (L) of water. Beryllium, as a chemical component, is naturally found in some food. The concentration of beryllium in both raw carrots and field corn grown in the United States is less than 25 micrograms (μg) (1 μg = 1 millionth of a gram) in a kilogram (kg) of the fresh vegetables. The intake of beryllium for most people will be very small.

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You can be exposed to higher than normal levels of beryllium, mostly in the form of beryllium oxide, that occur in certain workplaces. Occupational exposure to beryllium occurs at places where the chemical is mined, processed, and converted into metal, alloys, and other chemicals. Workers engaged in machining metals containing beryllium, in recycling beryllium from scrap alloys, or in using beryllium products may also be exposed to higher levels of beryllium. An estimated 11,000 workers may be exposed to beryllium and its compounds in the workplace.

As a member of the general public, you may be exposed to higher than normal levels of beryllium if you live near an industry that processes or uses beryllium. People who live near hazardous landfill sites that contain high concentrations of beryllium may also be exposed to higher than normal levels of beryllium. Beryllium, as a chemical component, occurs naturally in tobaccos and can be inhaled from cigarette smoke. People who smoke may breathe considerably more beryllium than people who do not smoke.

Beryllium metal and metal alloys may be found in consumer products such as electronic devices (e.g., televisions, calculators, and personal computers) and special nonsparking tools. More information about beryllium exposure can be found in Chapter 5.

1.4 HOW CAN BERYLLIUM ENTER AND LEAVE MY BODY?

Beryllium can enter your body if you breathe air, eat food, or drink water containing it. Very little beryllium enters your body from skin contact with the metal. Exposure to beryllium in the air is most likely to occur in factories where beryllium is used or processed. It is possible to be exposed to small amounts of beryllium in the air, water, or soil around waste sites. When you breathe air containing beryllium, beryllium particles can be deposited in the lungs. The beryllium that you breathe in moves slowly into the bloodstream. Some of the beryllium deposited in the lungs can be moved to the mouth and then swallowed; the rest can remain in your lungs for a long time. If you eat food or drink water that contains beryllium, less than 1% passes from your stomach and intestines into the bloodstream. Therefore, most of the beryllium that you swallow leaves your body through the feces without entering the bloodstream. The small amount of beryllium that moves from the lungs, stomach, and intestines into the bloodstream is carried by the blood to the kidneys. Beryllium leaves the kidneys by the urine. If you swallow beryllium, beryllium leaves the body in a few days. However, if you inhale beryllium, it may take months to years before your body rids itself of beryllium because it takes a long time before all the beryllium in the lungs enters the bloodstream or is swallowed. If your skin comes into contact with beryllium, not very much will enter your body unless your skin is scraped or cut. For more information, please read Chapter 2.

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1.5 HOW CAN BERYLLIUM AFFECT MY HEALTH?

Beryllium is a metal that can be harmful when you breathe it. The effects depend on how much and how long you are exposed to it. When you breathe it in, beryllium can damage your lungs. When you breathe in large amounts of beryllium over a short period of time, the lung damage resembles pneumonia with reddening and swelling of the lungs. This condition is called acute beryllium disease. In this case, if you stop breathing air with beryllium in it, the lung damage may heal. Some people can become sensitive to beryllium. This is known as hypersensitivity or allergy. If you are sensitive to beryllium, you develop an immune or inflammatory reaction to amounts of beryllium that do not cause effects in people who are not sensitive. When this occurs, white cells accumulate around the beryllium and form granulomas (granulomas are not tumors). This condition is called chronic beryllium disease. When you are exposed to smaller amounts of soluble or insoluble beryllium for long periods of time, you may become weak and short of breath.

Although the soluble and insoluble forms of beryllium can cause chronic beryllium disease, we do not know for certain what amount of beryllium is safe. Although most workers breathing in air containing beryllium at less than 0.002 milligrams (mg) (1 mg = 1 thousandth of a gram) in a cubic meter (mg/m^3) (a level that government rules permit in the workplace) will not have lung damage as a result of exposure, some workers will. Whether this level of exposure is absolutely safe is uncertain. Both the short-term, pneumonia-like disease and the chronic beryllium disease can be fatal. Long periods of exposure to beryllium have been reported to cause cancer in laboratory animals. The Department of Health and Human Services has determined that beryllium and certain beryllium compounds may reasonably be anticipated to be a carcinogen. We do not know whether breathing air, eating food, or drinking water that contains beryllium or whether skin contact with beryllium has any effects on reproduction or causes birth defects in humans or animals. Swallowing beryllium has not been reported to cause effects in humans because very little beryllium can move from the stomach and intestines into the bloodstream. Beryllium contact with skin that has been scraped or cut can cause rashes or ulcers. If you have developed an allergy to beryllium and have skin contact with it, you can get granulomas on the skin. These skin granulomas appear as a rash or as nodules. The skin granulomas are formed in the same way that lung granulomas are formed in sensitive people. For more information on how beryllium can affect your health, please read Chapter 2.

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1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO BERYLLIUM?

Beryllium can be measured in the urine and blood, but the amount of beryllium in the urine or blood may not reflect the amount to which you were exposed. The measurement of beryllium in urine and blood may not determine how recently you were exposed. Small amounts of human lung and skin can be removed from the body and examined to determine whether beryllium is present in these tissues. These tests can be done in a doctor's office or in a hospital. There is also a test that uses blood cells or cells washed out of the lung. If these cells start growing in the presence of beryllium, the possibility is strong that you have become sensitive to beryllium and have chronic beryllium disease. For more information, please read Chapters 2 and 6.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The National Institute for Occupational Safety and Health (NIOSH) recommends a standard for occupational exposure of $0.5 \mu\text{g beryllium}/\text{m}^3$ of workroom air during an 8-hour shift to protect workers from a concern that beryllium may cause cancer. The Occupational Safety and Health Administration (OSHA) has set a limit of $2 \mu\text{g beryllium}/\text{m}^3$ of workroom air for an 8-hour work shift. The Environmental Protection Agency restricts the amount of beryllium emitted into the environment by industries that process beryllium ores, metal, oxide, alloys, or waste to 10 grams (g) in a 24-hour period, or to an amount that would result in atmospheric levels of $0.01 \mu\text{g beryllium}/\text{m}^3$ of air, averaged over a 30-day period. For more information, please read Chapter 7.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. These clinics specialize in the recognition, evaluation, and treatment of illnesses resulting from exposure to hazardous substances.

CHLORINATED SOLVENTS

A. CARBON TETRACHLORIDE

Source: ATSDR, 1992. "Draft Toxicological Profile for Carbon Tetrachloride."
Agency for Toxic Substances and Disease Registry, Atlanta, GA. September,
1992.

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This Statement was prepared to give you information about carbon tetrachloride and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,300 hazardous waste sites as the most serious in the nation. These sites comprise the "National Priorities List" (NPL): Those sites which are targeted for long-term federal cleanup activities. Carbon tetrachloride has been found in at least 286 of the sites on the NPL. However, the number of NPL sites evaluated for carbon tetrachloride is not known. As EPA evaluates more sites, the number of sites at which carbon tetrachloride is found may increase. This information is important because exposure to carbon tetrachloride may cause harmful health effects and because these sites are potential or actual sources of human exposure to carbon tetrachloride.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You can be exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking substances containing the substance or by skin contact with it.

If you are exposed to a substance such as carbon tetrachloride, many factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, gender, nutritional status, family traits, life-style, and state of health.

1.1 WHAT IS CARBON TETRACHLORIDE?

Carbon tetrachloride is a clear liquid that evaporates very easily; therefore it is often found in the environment in gaseous form. Most carbon tetrachloride that escapes to the environment is found as a gas in the atmosphere. Carbon tetrachloride will not easily burn. Carbon tetrachloride has a sweet odor and most people can begin to smell it in air at 10 parts carbon tetrachloride per million parts of air (ppm). It is not known if people can taste it or, if they can, at what level.

Carbon tetrachloride does not occur naturally but has been produced in large quantities to make refrigeration fluid and propellants for aerosol cans. Since refrigerants and aerosol propellants have been found to affect the earth's ozone layer, the production of these chemicals is being phased out. Consequently, the manufacture and use of carbon tetrachloride will probably decline in the future.

In the past, carbon tetrachloride was widely used as a cleaning fluid, both in industry and dry cleaning establishments, where it served as a degreasing agent, and in the household.

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where it was used to remove spots from clothing, furniture, and carpeting. Carbon tetrachloride was also used in fire extinguishers and as a fumigant to kill insects in grain. These uses were discontinued in the mid-1960s. Until recently, carbon tetrachloride was used as a pesticide, but this was stopped in 1986.

Further information on the properties and uses of carbon tetrachloride can be found in Chapters 3, 4, and 5.

1.2 WHAT HAPPENS TO CARBON TETRACHLORIDE WHEN IT ENTERS THE ENVIRONMENT?

Because liquid carbon tetrachloride evaporates easily, most of the compound released to the environment during its production and use reaches the air where it is found mainly as a gas. It can remain in air for several years before it is broken down to other chemicals. Small amounts of carbon tetrachloride are found in surface water. Because it evaporates easily, much of it will travel from surface water to the air within a few days or weeks, but it may be trapped in groundwater. Carbon tetrachloride is not expected to stick to soil particles. Much of it will evaporate to the air and some of it will go to the groundwater where it can remain for months before it is broken down to other chemicals. It is not expected to build up in fish. We do not know if it builds up in plants.

Further information on what happens to carbon tetrachloride in the environment may be found in Chapters 4 and 5.

1.3 HOW MIGHT I BE EXPOSED TO CARBON TETRACHLORIDE?

Low background levels of carbon tetrachloride are found in air, water, and soil because of past and present releases. Concentrations of 0.1 part carbon tetrachloride per billion parts of air (ppb) are common around the world, with somewhat higher values (0.2-0.6 ppb) in cities. Carbon tetrachloride is also found in some drinking water supplies, usually at concentrations less than 0.5 ppb. Exposure to levels of carbon tetrachloride higher than these typical "background" levels is likely to occur only at specific industrial locations where carbon tetrachloride is still used or near chemical waste sites where emissions into air, water, or soil are not properly controlled. Exposure at such sites could occur by breathing carbon tetrachloride in air, by drinking water contaminated with carbon tetrachloride, or by getting soil contaminated with carbon tetrachloride on the skin. Young children may also be exposed if they eat soil that contains carbon tetrachloride. Carbon tetrachloride has been found in water or soil at about 22% of the waste sites investigated under Superfund, at concentrations ranging from less than 50 to over 1,000 ppb.

People who work with carbon tetrachloride are likely to receive the greatest exposure to the compound. The National Institute for Occupational Safety and Health (NIOSH) estimates that 58,208 workers are potentially exposed to carbon tetrachloride in the United States. The average daily intake of carbon tetrachloride for the general

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population is estimated to be 0.1 microgram (μg). The estimated average daily amount that the general population may drink in water is 0.01 μg .

Further information on the ways that humans can be exposed to carbon tetrachloride is presented in Chapter 5.

1.4 HOW CAN CARBON TETRACHLORIDE ENTER AND LEAVE MY BODY?

Carbon tetrachloride can enter your body through your lungs if you breathe air containing carbon tetrachloride, or through your stomach if you swallow food or water containing carbon tetrachloride. Carbon tetrachloride can also pass through the skin into the body. When you inhale carbon tetrachloride, over 30-40% of what you inhale enters your body where most of it accumulates in body fat. Some can enter the kidney, liver, brain, and skeletal muscle. About 85-91% of the carbon tetrachloride that you drink can enter your body when you drink water contaminated with it. Some of the compound that enters your body when you breathe it or drink water contaminated with it leaves your body quickly, and most of it can be found in your breath within a few hours. Animal studies indicated that 30-40% of carbon tetrachloride is excreted in expired air, 50-60% is excreted in feces, and low amounts are excreted in the urine. Animal studies also suggest that it may take weeks for the remainder of the compound in the body to be eliminated, especially those amounts that have entered the body fat. Most of the compound is eliminated from your body unchanged, but some of the carbon tetrachloride in your body may change to other chemicals (for example, chloroform, hexachloroethane, and carbon dioxide). Chloroform and hexachloroethane may be harmful in sufficiently high doses.

Further information on how carbon tetrachloride enters and leaves the body is presented in Chapter 2.

1.5 HOW CAN CARBON TETRACHLORIDE AFFECT MY HEALTH?

Most information on the health effects of carbon tetrachloride in humans comes from cases where people have been exposed only once or for a short period to relatively high levels of carbon tetrachloride. Experiments have not been performed on the effects of long-term exposure of humans to low levels of carbon tetrachloride, so the human health effects of such exposures are not known.

The liver is especially sensitive to carbon tetrachloride. In mild cases, the liver becomes swollen and tender, and fat tends to build up inside the tissue. In severe cases, many cells may be damaged or destroyed, leading to decreases in liver function. Such effects are usually reversible if exposure is discontinued.

The kidney is also sensitive to carbon tetrachloride. Less urine may be formed, leading to a buildup of water in the body (especially in the lungs) and buildup of waste products in the blood. Kidney failure often was the main cause of death in people who died after over-exposure to carbon tetrachloride.

Fortunately, if injuries to the liver and kidney are not too severe, these effects disappear after exposure stops. This is because both organs can repair damaged cells and replace dead tissue, and function is usually nearly normal within a few days or weeks after exposure.

At higher exposure levels, the nervous system, including the brain, is affected. Such exposure can be fatal. The immediate effects are usually signs of intoxication, including headache and dizziness, along with nausea and vomiting. These effects usually disappear within a day or two after exposure. In severe cases, stupor or even coma can result, and permanent damage to nerve cells can occur.

Carbon tetrachloride also causes effects on other tissues of the body, but these are not usually as important as the effects on the liver, kidney, and brain. Information from animal studies indicates that carbon tetrachloride does not cause birth defects but might decrease the survival rate of newborn animals.

Studies in animals have shown that carbon tetrachloride given by mouth can increase the frequency of liver tumors. Studies have not been performed to determine if breathing carbon tetrachloride causes tumors in animals, or whether swallowing or breathing carbon tetrachloride causes tumors in humans, but it should be assumed that carbon tetrachloride could have these effects. The Department of Health and Human Services (DHHS) has determined that carbon tetrachloride may reasonably be anticipated to be a carcinogen (i.e., cause cancer). The International Agency for Research on Cancer (IARC) has determined that carbon tetrachloride is possibly carcinogenic to humans. The EPA has determined that carbon tetrachloride is a probable human carcinogen.

Many of the cases of carbon tetrachloride toxicity reported above are associated with drinking alcohol. The frequent drinking of alcoholic beverages increases the danger from carbon tetrachloride exposure.

Further information on the health effects of carbon tetrachloride may be found in Chapter 2.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO CARBON TETRACHLORIDE?

Several very sensitive and specific tests can detect carbon tetrachloride in exposed persons. The most convenient way is simply to measure carbon tetrachloride in exhaled air, but carbon tetrachloride can also be measured in blood, fat, or other tissues. Because special equipment is needed, these tests are not routinely performed in doctors' offices; but your doctor can refer you to where you can obtain any test. Although these tests can show that a person has been exposed to carbon tetrachloride, the test results cannot be used to reliably predict whether any health effects might result. Because carbon tetrachloride leaves the body fairly quickly, these methods are best suited to detecting exposures that have occurred within the last several days. Further information on how carbon tetrachloride can be measured in exposed humans is given in Chapter 6.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

To protect citizens from exposure to carbon tetrachloride, the federal government has limited or banned the use of this compound in most common household products and fire extinguishers, and has discontinued its use as a pesticide. To protect workers who use carbon tetrachloride while on the job, the Occupational Safety and Health Administration (OSHA) has set a maximum concentration limit in workplace air of 2 ppm for an 8-hour workday over a 40-hour work week. EPA has also set limits on how much carbon tetrachloride can be released from an industrial plant into waste water and is preparing to set limits on how much carbon tetrachloride can escape from a plant into outside air. To ensure that drinking water supplies are safe, EPA has set a Maximum Contaminant Level (MCL) for carbon tetrachloride of 5 parts per billion (ppb), based on detection limits in drinking water. Because carbon tetrachloride is possibly carcinogenic, a Maximum Contaminant Level Goal (MCLG) of zero has been proposed. More detailed information on federal and state regulations regarding carbon tetrachloride may be found in Chapter 7.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333
(404) 639-6000

This agency can also provide you with information on the location of occupational and environmental health clinics. These clinics specialize in the recognition, evaluation, and treatment of illness resulting from exposure to hazardous substances.

CHLORINATED SOLVENTS

B. METHYLENE CHLORIDE

Source: ATSDR, 1991. "Draft Toxicological Profile for Methylene Chloride." Agency for Toxic Substances and Disease Registry, Atlanta, GA. October, 1991.

1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about methylene chloride and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,300 sites on its National Priorities List (NPL). Methylene chloride has been found in at least 311 of these sites. However, we do not know how many of the 1,300 NPL sites have been evaluated for methylene chloride. As EPA evaluates more sites, the number of sites at which methylene chloride is found may change. This information is important for you to know because methylene chloride may cause harmful health effects and because these sites are potential or actual sources of human exposure to methylene chloride.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous chemical such as methylene chloride, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS METHYLENE CHLORIDE?

Methylene chloride, also known as dichloromethane, is a colorless liquid that has a mild sweet odor, evaporates very quickly, and will not easily burn. It is widely used as an industrial solvent and as a paint stripper. It can also be found in certain aerosol and pesticide products and is used in the manufacture of photographic film. Methylene chloride does not appear to occur naturally in the environment. It is made from methane gas or wood alcohol. Most of the methylene chloride released to the environment results from its use as an end product by various industries and the use of aerosol products and paint removers in the home.

More information on the properties and uses of methylene chloride may be found in Chapters 3 and 4.

1. PUBLIC HEALTH STATEMENT

1.2 WHAT HAPPENS TO METHYLENE CHLORIDE WHEN IT ENTERS THE ENVIRONMENT?

Methylene chloride is mainly released to the environment in air and to a lesser extent in water and soil, due to industrial and consumer uses. Many chemical waste sites, including NPL sites, contain methylene chloride and these might act as additional sources of environmental contamination through spills, leaks, or evaporation. Because methylene chloride evaporates readily, most of it is released into the air. In air, it is broken down by sunlight and by reaction with other chemicals present in the air. About half of the methylene chloride disappears from air in 53—127 days. Although methylene chloride does not dissolve easily in water, small amounts may be found in some drinking water. Methylene chloride that is present in water is broken down slowly by reactions with other chemicals or by bacteria. Over 90% of the methylene chloride in the environment changes to carbon dioxide (CO₂), which is not toxic. It takes about 1 to 6 days for half the methylene chloride to break down in water. When methylene chloride is spilled on land, it attaches loosely to nearby surface soil particles. It moves from the soil into the air. Some may also move into groundwater. We do not know how long it remains in soil. We do not expect methylene chloride to build up in plants or animals.

More information on what happens to methylene chloride in the environment may be found in Chapters 4 and 5.

1.3 HOW MIGHT I BE EXPOSED TO METHYLENE CHLORIDE?

You may be exposed to methylene chloride in air, water, food, or from consumer products. Because methylene chloride evaporates easily, the greatest potential for exposure is when you breathe vapors of contaminated air. Background levels in air are usually at less than 1 part methylene chloride per billion parts (ppb) of air. Methylene chloride has been found in some urban air and at some hazardous waste sites at average concentrations of 11 ppb of air. The average daily intake of methylene chloride from outdoor air in three United States cities ranges from 33 to 309 micrograms per day ($\mu\text{g}/\text{day}$). Contact with consumer products such as paint strippers or aerosol cans that contain methylene chloride is another frequent source of exposure. Exposure occurs as a result of breathing the vapors given off by the product or from direct contact of the liquid material with the skin. Air concentrations resulting from the use of consumer products containing methylene chloride usually range from 1 to 23 ppb. The highest and most frequent exposures to methylene chloride usually occur in workplaces where the chemical is used. People who work in these places can breathe in the chemical or it may come in contact with their skin. Concentrations ranging from 1 to 1,000 parts methylene chloride per million parts (ppm; 1 ppm is 1,000 times more than 1 ppb) of air have been detected in general work areas, while higher concentrations (1,400 ppm) have been detected in samples in the breathing zone of some workers. The National Institute for

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Occupational Safety and Health (NIOSH) estimated that 1,000,000 workers may be exposed to methylene chloride. An average of 68 ppb of methylene chloride in surface water and 98 ppb methylene chloride in groundwater have been found at some hazardous waste sites. Less than 1 ppb has been found in most drinking water analyzed. We expect exposure from water and food to be low because very little methylene chloride has been detected in these sources.

More information on how you might be exposed to methylene chloride is given in Chapter 5.

1.4 HOW CAN METHYLENE CHLORIDE ENTER AND LEAVE MY BODY?

Methylene chloride may enter your body when you breathe vapors of contaminated air. It may also enter your body if you drink water from contaminated wells, or it may enter if your skin comes in contact with it. Since methylene chloride evaporates into air rapidly, exposure by breathing is the most likely source of exposure at hazardous waste sites, in the home, and in the workplace. When you breathe in methylene chloride, over 70% of it enters your bloodstream and quickly spreads throughout your body, with most of it going to the liver, kidney, brain, lungs, and fatty tissue. Increased physical activity or an increased amount of body fat tend to increase the amount of methylene chloride that remains or accumulates in your body tissue. About half of the methylene chloride in the blood leaves within 40 minutes. Some of the methylene chloride is broken down into other chemicals, including carbon monoxide (CO). Carbon monoxide is also toxic because it combines with hemoglobin to form carboxyhemoglobin (CO-Hb). Unchanged methylene chloride and its breakdown products are removed from your body mainly in the air you breathe out. Small amounts leave in your urine. This usually occurs within 48 hours after exposure. Although the rate of uptake through the skin and stomach have not been measured, uptake is likely to be fast.

More information on how methylene chloride enters and leaves the body is given in Chapter 2.

1.5 HOW CAN METHYLENE CHLORIDE AFFECT MY HEALTH?

If you breathe methylene chloride (300 ppm) or greater for short periods of time (e.g., 3-4 hours), you may not be able to hear faint sounds and your vision may be slightly impaired. If you breathe large amounts (800 ppm) you may not be able to react fast, remain steady, or perform tasks requiring precise hand movements. You may experience dizziness, nausea, tingling or numbness of the fingers and toes, and drunkenness if you breathe methylene chloride for a longer time. In most cases, effects disappear after exposure ends. Studies in animals suggest that exposure to higher concentrations (greater than 1,000 ppm) can lead to unconsciousness and death.

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Breathing methylene chloride also causes changes in the liver and kidney in animals, but similar effects have not been observed in humans. Studies in animals suggest that breathing methylene chloride does not cause birth defects or affect reproduction, even at high concentrations. Animal studies indicate that should you be exposed to vapors of methylene chloride in air, the vapors may irritate your eyes and affect your cornea. One study reported these effects at concentrations of 490 ppm; however the effects usually disappeared within a few days.

Methylene chloride has not been shown to cause cancer in humans exposed to vapors in the workplace. However, breathing high concentrations of methylene chloride for long periods of time did cause cancer in mice. The International Agency for Research on Cancer has determined that methylene chloride is possibly carcinogenic to humans. No information was found regarding the effects of methylene chloride in humans after oral exposure. Methylene chloride has caused death in rats following oral exposure to large amounts over a short period of time.

No information was found regarding the effects of methylene chloride in humans after skin exposure or direct contact with the eyes. In rabbits, effects were observed on the eyes (e.g., cornea), but they were reversible within a few days.

People can smell methylene chloride at about 200 ppm in air. Because people differ in their ability to smell various chemicals, odors may not be helpful in avoiding over-exposure.

The Agency for Toxic Substances and Disease Registry has calculated Environmental Media Evaluation Guides (EMEGs) for methylene chloride. EMEGs are derived from Minimal Risk Levels (MRLs) which are calculated from human or animal data for methylene chloride. The MRLs are further described in Chapter 2 and in the footnotes to Table 2-1 and 2-2. If a person is exposed to methylene chloride at a level below the EMEG for the period listed below, we do not expect harmful health effects to occur. Because these levels are based only on information currently available, some uncertainty is always associated with them. Also, an EMEG does not imply anything about the presence, absence, or level of risk for cancer because the methods for deriving EMEGs do not use any information about cancer. The EMEGs are provided as concentrations in order to allow for comparison to levels people might encounter in air, drinking water, and soil around homes or in other areas where children may play.

Air exposure

Drinking water EMEGs represent the lower end of a range and are protective for both children and adults.

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- An air EMEG of 0.4 ppm for methylene chloride was derived from human data for exposures of 14 days or less.
- An air EMEG of 0.03 ppm for methylene chloride was derived from animal data for exposures longer than 14 days but less than 1 year.

Drinking water exposure

Soil EMEGs represent the lower end of a range and are protective for both children and adults. However, this range is not protective for children (pica) who show increased desire for eating non-food items (such as soil).

- A drinking water EMEG of 0.6 ppm for methylene chloride was derived from animal data for exposures of 1 year or more.

Soil exposure

- A soil EMEG of 3,000 ppm for methylene chloride was derived from animal data for exposures of 1 year or more.

More information on how methylene chloride can affect your health is given in Chapter 2.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO METHYLENE CHLORIDE?

Several tests exist for determining whether you had measurable exposure to methylene chloride. The most direct method measures methylene chloride in the air you breathe out. Your blood can also be analyzed to determine if methylene chloride is present. However, these tests are only useful for detecting exposures which have occurred within a few days, because methylene chloride remains in the blood for only a very short time. A test to detect CO-Hb, a chemical formed in blood as methylene chloride breaks down in the body, can also be used as an indicator of exposure. However, this test is not specific, since smoking and exposure to other chemicals may also increase CO-Hb levels. Your urine can also be tested for methylene chloride itself or for other chemicals (such as formic acid) that are produced as methylene chloride breaks down in the body. These tests are not routinely available in a doctor's office; they require special equipment. The tests are useful to determine exposure to methylene chloride, but do not by themselves measure or predict health effects.

More information on how methylene chloride can be measured in exposed humans is presented in Chapters 2 and 6.

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1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The EPA requires that releases of methylene chloride of 1,000 pounds or more be reported to the federal government. The EPA has provided guidelines on how much methylene chloride you may be exposed to for certain amounts of time without causing risk to human health. It recommends that exposure of children to methylene chloride in water should not exceed 13.3 ppm for 1 day or 1.5 ppm for 10 days.

The Food and Drug Administration (FDA) has established limits on the amounts of methylene chloride that can remain in spice, hops extract, and decaffeinated coffee.

The Occupational Safety and Health Administration (OSHA) has adopted occupational exposure limits of 500 ppm for an 8-hour workday for persons who work with methylene chloride. OSHA is currently considering revising these standards based on the recent information available on cancer studies in animals.

NIOSH recommended a permissible limit of 75 ppm of methylene chloride over a 10-hour workday in the presence of CO concentrations less than or equal to 9.9 ppm. Because methylene chloride causes cancer in animals, NIOSH currently considers it a possible cancer-causing substance in the workplace and recommends that exposure be lowered to the lowest reasonable limit.

More information on government recommendations regarding methylene chloride can be found in Chapter 7.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. These clinics specialize in the recognition, evaluation, and treatment of illnesses resulting from exposure to hazardous substances.

CHLORINATED SOLVENTS

C. TETRACHLOROETHYLENE

Source: ATSDR “Draft Toxicologic Profile for Tetrachloroethylene.” Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about tetrachloroethylene and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,300 sites on its National Priorities List (NPL). Tetrachloroethylene has been found in at least 439 of these sites. However, we do not know how many of the 1,300 NPL sites have been evaluated for tetrachloroethylene. As EPA evaluates more sites, the number of sites at which tetrachloroethylene is found may change. This information is important for you to know because tetrachloroethylene may cause harmful health effects and because these sites are potential or actual sources of human exposure to tetrachloroethylene.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous chemical such as tetrachloroethylene, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS TETRACHLOROETHYLENE?

Tetrachloroethylene is a man-made substance that is widely used for dry cleaning fabrics and for metal-degreasing operations. It is also used as a starting material (building block) for making other chemicals and is used in some consumer products. Other names for tetrachloroethylene include perchloroethylene, perc, tetrachloroethene, perclene, and perchlor. It is a liquid at room temperature. Some of it evaporates into the air producing a sharp, sweet odor. For more information, see Chapters 3 and 4.

1.2 WHAT HAPPENS TO TETRACHLOROETHYLENE WHEN IT ENTERS THE ENVIRONMENT?

Tetrachloroethylene enters the environment mostly by evaporating into the air during use. It can also get into water supplies and the soil during disposal of sewage sludge and factory waste. Tetrachloroethylene may also get into the air, soil, or water by leaking or evaporating from storage and waste sites. It can last for several months in the air before

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it is broken down into other chemicals or is brought back down to the soil and water by rain. Some of the chemicals that are formed may also be harmful.

Much of the tetrachloroethylene that gets into water and soil will evaporate to the air. Some of it can travel through the soil and get into underground drinking water supplies. Tetrachloroethylene that gets into underground water may stay there for many months without being broken down. If conditions are right, bacteria will break down some of it and some of the chemicals formed may also be harmful. Under some conditions, tetrachloroethylene may stick to the soil and stay there. It does not seem to build up very much in animals that live in water, such as fish, clams, and oysters. We do not know if it builds up in plants grown on land. For more information on tetrachloroethylene in the environment, see Chapters 4 and 5.

1.3 HOW MIGHT I BE EXPOSED TO TETRACHLOROETHYLENE?

Humans can be exposed to tetrachloroethylene from environmental and occupational sources and from consumer products. Common environmental levels of tetrachloroethylene (called background levels) are several thousand times lower than levels found in some workplaces. Background levels are found in the air we breathe, in the water we drink, and in the food we eat. The chemical is found most frequently in air and, less often, in water. Tetrachloroethylene gets into air by evaporation from industrial or dry cleaning operations. One study showed tetrachloroethylene was present in 25% of drinking water samples tested in the study. In another study, 14 to 26% of groundwater samples contained tetrachloroethylene. There are no similar studies on how often the chemical is found in air samples, but we know it is widespread in the air. We do not know how often it is found in soil, but it was found in 5% of sediments sampled. Tetrachloroethylene also comes from releases from areas where chemical wastes are stored.

In general, tetrachloroethylene levels in air are higher in cities or industrial areas than in more rural or remote areas. The background levels of tetrachloroethylene in air are far less than 1 part in 1 million parts of air (ppm). You can smell it at levels of 5 ppm in air. The air close to dry cleaning shops and chemical waste sites has levels of tetrachloroethylene higher than background levels. These levels are still less than 1 ppm. Water, both above and below ground, may contain tetrachloroethylene. Levels in water are also usually much less than 1 ppm, but are higher than levels in air. Levels in water near disposal sites are higher than levels in water far away from those sites. Water with tetrachloroethylene pollution may have levels greater than 1 ppm. Background levels in soil are probably 100 to 1,000 times lower than 1 ppm.

You can also be exposed to tetrachloroethylene by using certain consumer products. Products that may contain tetrachloroethylene include auto brake quieters and cleaners,

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suede protectors, water repellents, silicone lubricants, and belt lubricants. Other products include specialized aerosol cleaners, ignition wire driers, fabric finishers, spot removers, adhesives, and wood cleaners. Although uncommon, small amounts of tetrachloroethylene have been found in food. Tetrachloroethylene may also be found in the breast milk of mothers who have been exposed to the chemical. For more information, see Chapter 5.

The people with the greatest chance of exposure to tetrachloroethylene are those who work with it. According to estimates from a survey conducted by the National Institute for Occupational Safety and Health (NIOSH) more than 650,000 U.S. workers may be exposed to tetrachloroethylene. The estimated amount that the general population might breathe in per day ranges from 0.04 to 0.2 milligrams. The estimated amount that most people might drink in water is less than 0.006 milligrams per day. These are very small amounts.

1.4 HOW CAN TETRACHLOROETHYLENE ENTER AND LEAVE MY BODY?

Tetrachloroethylene can rapidly enter your body when you breathe air containing it. How much enters your body by this route depends on how much of the chemical is in the air, how fast and deeply you are breathing, and how long you are exposed to it. Tetrachloroethylene may also rapidly enter your body through drinking water or eating food containing the chemical. How much enters your body depends on how much of the chemical you drink or eat. These two routes are the most likely ways people will take in tetrachloroethylene. These are also the most likely ways that people living near areas polluted with the chemical, such as hazardous waste sites, might take in tetrachloroethylene. Since tetrachloroethylene does not pass through the skin to any significant extent, entry into your body by this path is not of much concern.

Most tetrachloroethylene leaves your body rapidly when you breathe out the chemical in your breath. This is true whether you take up the chemical by breathing, drinking, eating, or touching it. Some of the tetrachloroethylene is changed into other chemicals in your body, and these are removed from your body in urine. One of these chemicals, trichloroacetic acid, is also thought to be harmful. Most of the changed tetrachloroethylene is removed in a few days. A small amount of the tetrachloroethylene that you take in is stored in tissues of your body. Part of the tetrachloroethylene that is stored in fat may stay in your body for several days or weeks. For more information on how tetrachloroethylene enters and leaves your body see Chapter 2.

1.5 HOW CAN TETRACHLOROETHYLENE AFFECT MY HEALTH?

When concentrations in air are high--particularly in closed, poorly ventilated areas--single exposures to tetrachloroethylene can cause dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, and possibly unconsciousness and death. Skin

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irritation may result from repeated or extended contact with the chemical. As you might expect, these symptoms occur almost entirely in work (or hobby) environments. Some people may be exposed to levels lower than those causing dizziness, sleepiness, and other nervous system effects. The health effects of breathing in air or drinking water with low levels of tetrachloroethylene are not known. The effects of exposing babies to tetrachloroethylene through breast milk are unknown. Results from some studies suggest that women who work in dry cleaning industries may have more menstrual problems and spontaneous abortions than women who are not exposed to tetrachloroethylene. However, we do not know if tetrachloroethylene was responsible for these problems because other possible causes were not considered. The chemical does not seem to cause birth defects in children whose parents are exposed.

Most people can smell tetrachloroethylene when it is present in the air at levels of 5 ppm or more. You can smell tetrachloroethylene in water if there is 0.3 ppm or more of it.

Animal studies, conducted with amounts much higher than those that most people are exposed to, show that tetrachloroethylene can cause liver and kidney damage and liver and kidney cancers. However, it has not been shown to cause cancer in people. The Department of Health and Human Services has determined that tetrachloroethylene may reasonably be anticipated to be a carcinogen. Tetrachloroethylene can be toxic to the fetuses of rats and mice. The only developmental effects seen in the offspring of rats that breathed very high levels of the chemical while they were pregnant were minor changes in the brain and behavior of the offspring. Since this was the only study showing developmental effects, we do not know how meaningful these results are at the present time.

For more information on the health effects of tetrachloroethylene, see Chapter 2.

The Agency for Toxic Substances and Disease Registry has calculated Environmental Media Evaluation Guides (EMEGs) for tetrachloroethylene. EMEGs are derived from Minimal Risk Levels (MRLs) which are calculated from human or animal data for tetrachloroethylene. The MRL(s) are further described in Chapter 2 and in the footnotes to Table 2-1 and 2-3. If a person is exposed to tetrachloroethylene at a level below the EMEG for the period listed below, we do not expect harmful health effects to occur. Because these levels are based only on information currently available, some uncertainty is always associated with them. Also, an EMEG does not imply anything about the presence, absence or level of risk for cancer because the methods for deriving EMEGs do not use any information about cancer. The EMEGs are provided as concentrations in order to allow for comparison to levels people might encounter in air, drinking water, and soil around homes or in other areas where children may play.

1. PUBLIC HEALTH STATEMENT

Air exposure

- An air EMEG of 0.6 ppm for tetrachloroethylene was derived from human data for exposures of 14 days or less.
- An air EMEG of 0.009 ppm for tetrachloroethylene was derived from animal data for exposures longer than 14 days but less than one year.

Drinking water exposure

Drinking water EMEGs represent the lower end of a range and are protective for both children and adults.

- A drinking water EMEG of 1 ppm for tetrachloroethylene was derived from animal data for exposures longer than 14 days but less than one year.

Soil exposure

Soil EMEGs represent the lower end of a range and are protective for both children and adults. However, this range is not protective for children (pica) who show increased desire for eating non-food items (such as soil).

- A soil EMEG of 5,000 ppm for tetrachloroethylene was derived from animal data for exposures longer than 14 days but less than one year.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO TETRACHLOROETHYLENE?

One way of testing for tetrachloroethylene exposure is to measure the amount of the chemical in the breath. This test has been used to measure levels of the chemical in persons living in areas where the air is contaminated with tetrachloroethylene or those exposed to the chemical through their work. This test is only useful, however, if the exposure is recent (less than a week) because tetrachloroethylene rapidly leaves the body. Tetrachloroethylene can also be detected in the blood. In addition, samples of blood and urine can be used to identify breakdown products of the chemical in persons suspected of being exposed to tetrachloroethylene. Some of the breakdown products can be identified in the blood and urine for only short periods after exposure. One product, trichloroacetic acid, can be detected for several days after exposure. Although these tests are relatively simple to perform, most physicians do not have the proper equipment and must rely on special laboratories to collect and test the samples. Because exposure to other chemicals can produce the same breakdown products in the urine and blood, these

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tests cannot determine if you have been exposed only to tetrachloroethylene. For more information on where and how tetrachloroethylene can be detected in your body after you have been exposed to it, see Chapters 2 and 6.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The government has developed regulations and guidelines for tetrachloroethylene. These are designed to protect the public from the potential adverse health effects of the chemical. The Environmental Protection Agency (EPA) has recommended limits on how much tetrachloroethylene can be present in drinking water. EPA advises that children should not have more than 2.0 milligrams tetrachloroethylene per liter of water (mg/L) (2 ppm) in 1 day or more than 1.4 mg/L (1.4 ppm) per day for long-term exposure. For long-term exposure in adults, EPA recommends that there should not be more than 5 mg/L (5.0 ppm) in the drinking water.

EPA considers tetrachloroethylene to be a hazardous waste. Many regulations govern its disposal. If amounts greater than 1 pound are released to the environment, The National Response Center of the federal government must be told immediately.

The Occupational Safety and Health Administration (OSHA) limits the amount of tetrachloroethylene that can be present in workroom air. This amount is now limited to 25 ppm for an 8-hour workday over a 40-hour workweek, but may be changed to 50 ppm in the near future. OSHA also proposed limiting the peak concentration for short-term exposure to not greater than 200 ppm. NIOSH recommends that tetrachloroethylene be handled as a chemical that might potentially cause cancer and states that levels of the chemical in workplace air should be as low as possible.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. These clinics specialize in the recognition, evaluation, and treatment of illnesses resulting from exposure to hazardous substances.

CHLORINATED SOLVENTS

D. 1,1,1-TRICHLOROETHANE

Source: ATSDR, 1990. "Toxicological Profile for 1,1,1-Trichloroethane." Agency for Toxic Substances and Disease Registry, Atlanta, GA. December, 1990.

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This Statement was prepared to give you information about 1,1,1-trichloroethane and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1177 sites on its National Priorities List (NPL). 1,1,1-Trichloroethane has been found at 244 of these sites. However, we do not know how many of the 1177 NPL sites have been evaluated for 1,1,1-trichloroethane. As EPA evaluates more sites, the number of sites at which 1,1,1-trichloroethane is found may change. The information is important for you because 1,1,1-trichloroethane may cause harmful health effects and because these sites are potential or actual sources of human exposure to 1,1,1-trichloroethane.

When a chemical is released from a large area such as an industrial plant, or from a container such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking, substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous substance such as 1,1,1-trichloroethane, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, or drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS 1,1,1-TRICHLOROETHANE?

1,1,1-Trichloroethane is a colorless man-made chemical which does not occur naturally. In the environment, it can be found as a liquid, as a vapor, or dissolved in water and other chemicals. When found as a liquid in an open container, it evaporates quickly and becomes a vapor in the air. 1,1,1-Trichloroethane has a sweet yet sharp odor.

1,1,1-Trichloroethane is made by industry and used in commercial products. Large amounts are produced each year; about 700 million pounds were made in 1987. We are not sure how much 1,1,1-trichloroethane will be made in the future.

1,1,1-Trichloroethane has many industrial and household uses. It is often used as a solvent to dissolve other substances, for example, glue and paint. In industry, it is widely used to remove oil or grease from manufactured metal parts. In the home, it might be in products such as spot cleaners, glues, and aerosol sprays. 1,1,1-Trichloroethane can also be found as a liquid in the soil and water, and as a vapor in the air at hazardous waste sites.

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Many home and workplace uses result in 1,1,1-trichloroethane vapor entering the air, where it lasts for about 2-10 years. It is not known how long 1,1,1-trichloroethane lasts in water or soil. From surface waters such as lakes and rivers, where 1,1,1-trichloroethane will partially mix with water, it will probably evaporate quickly to the air. It will not stick to soil, and can be carried by water through soil and into groundwater. Once there, it may be broken down by naturally occurring organisms, which take 200-300 days to remove half of the chemical.

You will find detailed information on the chemical properties of 1,1,1-trichloroethane in Chapter 3; the uses of 1,1,1-trichloroethane are described in Chapter 4. More information on environmental fate and sources of human exposure is in Chapter 5.

1.2 HOW MIGHT I BE EXPOSED TO 1,1,1-TRICHLOROETHANE?

You can be exposed to 1,1,1-trichloroethane daily from a wide variety of sources. 1,1,1-Trichloroethane is found in air samples taken all over the world. In the United States, urban air typically contains about 0.0001-0.001 parts of 1,1,1-trichloroethane per million parts air (ppm); rural air usually contains less than 0.0001 ppm. Because 1,1,1-trichloroethane is used so much in home and office products, there may be much more of it in inside air than outside air. New buildings can have high indoor levels, since this chemical is found in many building materials. Thus, you are more likely to be exposed to the vapor form of this chemical indoors.

Common consumer sources of 1,1,1-trichloroethane include glues, household cleaners, and aerosol sprays. If you eat foods contaminated with 1,1,1-trichloroethane, you will be exposed to it. Workplace exposure to 1,1,1-trichloroethane can occur during the use of metal degreasing agents, paints, glues, and cleaning products. You can be exposed to 1,1,1-trichloroethane by breathing the vapors from these products. Industrial uses of 1,1,1-trichloroethane probably make up the largest amount of release to the environment. High levels of exposure can occur during glue sniffing or solvent abuse.

1,1,1-Trichloroethane has also been found in rivers and lakes (up to 0.01 ppm), in soil (up to 120 ppm), in drinking water (up to 0.0035 ppm), and drinking water from underground wells (up to 12 ppm). These amounts vary widely by location, and can be caused by releases during manufacture and transportation and during industrial or household use. You can be exposed to 1,1,1-trichloroethane by drinking contaminated water. Release of large amounts of the chemical can be caused by spillage, improper disposal, or industrial emissions. High levels of 1,1,1-trichloroethane in soil, surface water, or groundwater can also be caused by water from landfills and hazardous waste sites. Further information on human exposure to 1,1,1-trichloroethane is discussed in Chapter 5.

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1.3 HOW CAN 1,1,1-TRICHLOROETHANE ENTER AND LEAVE MY BODY?

1,1,1-Trichloroethane can quickly enter your body if you breathe in air containing 1,1,1-trichloroethane vapor. It can also enter your body if you drink water or eat food containing 1,1,1-trichloroethane. If you were to spill it on your skin, most of it would quickly evaporate into the air and small amounts would enter your body through the skin. The most likely way you would be exposed to 1,1,1-trichloroethane at a hazardous waste site would be to breathe in 1,1,1-trichloroethane vapors in the air or drink water contaminated with 1,1,1-trichloroethane. Regardless of how 1,1,1-trichloroethane enters your body, nearly all of it will quickly leave your body in the air you breathe out. The small amount that is not breathed out changes in your body into other substances, known as metabolites. Most of the metabolites will leave your body in urine and breath in a few days. If you are exposed to 1,1,1-trichloroethane for a long time, some metabolites will begin to collect in your body. These metabolites rapidly leave your body when exposure stops. Further information on how 1,1,1-trichloroethane can enter and leave the body is found in Chapter 2.

1.4 HOW CAN 1,1,1-TRICHLOROETHANE AFFECT MY HEALTH?

If you were to breathe air containing high levels of 1,1,1-trichloroethane for a short time you might experience dizziness, lightheadedness, and loss of balance and coordination. These effects would rapidly "disappear" after you stop breathing in the contaminated air. If you were to breathe in much higher levels of 1,1,1-trichloroethane, either intentionally or accidentally, you might become unconscious, your blood pressure might decrease, and your heart might stop beating. We do not know whether any health effects would happen if you were to breathe in low levels of 1,1,1-trichloroethane for a long time. Studies in animals have shown that damage to the breathing passages and lungs, as well as mild liver effects, can result from breathing air with high levels of 1,1,1-trichloroethane. Exposure of pregnant rats to high levels of 1,1,1-trichloroethane in air slowed development of their offspring during pregnancy. Similarly, when pregnant rabbits breathed air containing high levels of 1,1,1-trichloroethane their offspring had changes in their bone structure. So far, other studies in animals have not shown 1,1,1-trichloroethane in the air or water to cause cancer or affect their ability to produce offspring, but some of these studies were not complete enough to be absolutely sure. There are no studies in humans that can tell us whether health effects will occur if you were to eat food or drink water contaminated with 1,1,1-trichloroethane. However, swallowing large amounts of 1,1,1-trichloroethane caused liver damage and death in animals and could hurt humans. You can find more information on the health effects of 1,1,1-trichloroethane in Chapter 2.

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1.5 WHAT LEVELS OF EXPOSURE HAVE RESULTED IN HARMFUL HEALTH EFFECTS?

You can smell 1,1,1-trichloroethane in the air at levels greater than 100 ppm. Tables 1-1 through 1-4 show how exposure to 1,1,1-trichloroethane can or might affect your health. The levels of 1,1,1-trichloroethane in the air that can affect your health, or might affect your health based on animal studies, are shown in Tables 1-1 and 1-2. Serious effects are more likely to occur after eating or breathing large amounts of 1,1,1-trichloroethane. How long you are exposed to 1,1,1-trichloroethane will also determine how serious the effects will be. Longer exposure times will result in more serious effects than short exposure times. A Minimal Risk Level (MRL) is also included in Table 1-1. This MRL was derived from human data for short-term exposure, as described in Chapter 2 and in Table 2-1. The MRL provides a basis for comparison to levels which people might encounter in the air. If a person is exposed to 1,1,1-trichloroethane at an amount below the MRL, it is not expected that harmful (noncancer) health effects will occur. Since this level is based on information that is currently available, there is always some uncertainty associated with it. Also, since the method for deriving MRLs does not use any information about cancer, an MRL does not imply anything about the presence, absence, or level of risk of cancer. Tables 1-3 and 1-4 show that we know little about how eating or drinking 1,1,1-trichloroethane affects people or animals. If you spilled concentrated 1,1,1-trichloroethane on your skin and it remained on your skin for 5 minutes you might experience mild skin irritation (for example, redness and itching) in the area of the spill. The irritation would disappear within a few hours. Further information on levels of exposure to 1,1,1-trichloroethane that can cause health effects can be found in Chapter 2.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO 1,1,1-TRICHLOROETHANE?

Samples of your breath and urine can be tested to determine if you have been exposed to 1,1,1-trichloroethane. These tests can also be used to estimate how much 1,1,1-trichloroethane has entered your body. Samples of your breath would have to be taken within hours of exposure, and samples of urine would have to be taken within 1 or 2 days after your exposure to be of any value. These tests will not tell you whether you will have any health effects from exposure to 1,1,1-trichloroethane. The tests are not routinely available in hospitals and clinics. See Chapters 2 and 6 for more information about tests for exposure to 1,1,1-trichloroethane.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The Environmental Protection Agency (EPA) regulates the levels of 1,1,1-trichloroethane in drinking water. The highest level of 1,1,1-trichloroethane allowed in drinking water is 0.2 ppm. The EPA has decided that the level of 1,1,1-trichloroethane in lakes and streams should

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TABLE 1-1. Human Health Effects from Breathing 1,1,1-Trichloroethane*

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects**</u>
0.255		Minimal Risk Level (see Section 1.5 for discussion).
175	3.5 hours	Mild nervous system effects.
1,000	15 minutes	Mild eye irritation.
1,900	5 minutes	Severe nervous system effects.
2,650	15 minutes	Mild liver effects.
10,000	2 minutes	Unconsciousness, markedly decreased blood pressure.
Long-term Exposure (greater than 14 days)		
<u>Levels in Air</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term exposure of humans to air containing specific levels of 1,1,1-trichloroethane are not known.

*See Section 1.2 for a discussion of exposures encountered in daily life.

**These effects are listed at the lowest levels at which they were first observed. They may also be seen at higher levels.

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TABLE 1-2. Animal Health Effects from Breathing 1,1,1-Trichloroethane

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
1,800	4 hours	Changes in behavior in monkeys.
3,116	4 hours	Decreased activity and loss of coordination in rats.
3,911	2 hours	Death in mice.
5,000	10 minutes	Serious heart effects in dogs.
6,000	12 days	Mild developmental delays and effects in the offspring of pregnant rats and rabbits.
6,644	4 hours	Behavioral change in mice.
8,000	10 minutes	Serious blood pressure depression in dogs.
8,000	7 hours	Mild liver effects in rats.
10,300	6 hours	Unconsciousness and death in rats.
Long-term Exposure (greater than 14 days)		
<u>Levels in Air (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
70	3 months	Permanent brain injury in gerbils.
820	4 weeks	Mild liver effects in rats.

*These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

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TABLE 1-3. Human Health Effects from Eating or Drinking
1,1,1-Trichloroethane*

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of humans to food containing specific levels of 1,1,1-trichloroethane are not known.
<u>Levels in Water</u>		The health effects resulting from short-term exposure of humans to water containing specific levels of 1,1,1-trichloroethane are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term exposure of humans to food containing specific levels of 1,1,1-trichloroethane are not known.
<u>Levels in Water</u>		The health effects resulting from long-term exposure of humans to water containing specific levels of 1,1,1-trichloroethane are not known.

*See Section 1.2 for a discussion of exposures encountered in daily life.

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TABLE 1-4. Animal Health Effects from Eating or Drinking
1,1,1-Trichloroethane

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
26,000	1 day	Mild liver effects in rats.
<u>Levels in Water (ppm)</u>	1 day	Death in rabbits.
52,500		
Long-term Exposure (greater than 14 days)		
<u>Levels in Food (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
15,000	78 weeks	Shortened lifespan in rats.
<u>Levels in Water</u>		The health effects resulting from long-term exposure of animals to water containing specific levels of 1,1,1-trichloroethane are not known.

*These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

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not be more than 18 ppm to prevent possible health effects from drinking water or eating fish contaminated with 1,1,1-trichloroethane. Any releases or spills of 1,1,1-trichloroethane of 1000 pounds or more must be reported to the National Response Center. 1,1,1-Trichloroethane levels in the workplace are regulated by the Occupational Safety and Health Administration (OSHA). The workplace exposure limit for an 8-hour workday, 40-hour workweek is 350 ppm. See Chapter 7 for more information on regulations and advisories regarding 1,1,1-trichloroethane.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns not covered here, please contact your State Health or Environmental Department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road, E-29
Atlanta, Georgia 30333

This agency can also give you information on the location of the nearest occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses that result from exposure to hazardous substances.

CHLORINATED SOLVENTS

E. TRICHLOROETHYLENE

Source: ATSDR, 1991. "Draft Toxicological Profile for Trichloroethylene." Agency for Toxic Substances and Disease Registry, Atlanta, GA. October, 1991.

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This Statement was prepared to give you information about trichloroethylene and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,300 sites on its National Priorities List (NPL). Trichloroethylene has been found in at least 614 of these sites. However, we do not know how many of the 1,300 NPL sites have been evaluated for trichloroethylene. As EPA evaluates more sites, the number of sites at which trichloroethylene is found may change. This information is important for you to know because trichloroethylene may cause harmful health effects and because these sites are potential or actual sources of human exposure to trichloroethylene.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous chemical such as trichloroethylene, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS TRICHLOROETHYLENE?

Trichloroethylene is also known as Triclene®, Vitran®, and other names used in industry. It is a nonflammable, colorless liquid at room temperature with an odor similar to ether or chloroform. It is a man-made chemical that does not occur naturally in the environment. Trichloroethylene is mainly used as a solvent to remove grease from metal parts. It is used as a solvent in other ways too and is used to make other chemicals. Further information can be found in Chapters 3 and 4.

1.2 WHAT HAPPENS TO TRICHLOROETHYLENE WHEN IT ENTERS THE ENVIRONMENT?

By far, the biggest source of trichloroethylene in the environment is evaporation from factories that use it to remove grease from metals. It can also get into the air and water when it is disposed of at chemical waste sites. It evaporates easily but can stay in the

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soil. Once it is in the air, about half will be destroyed within a week. When trichloroethylene is broken down in the air, phosgene, a lung irritant, is formed. Once it is in the water, much will evaporate into the air; again, about half will leave within a week. It will take days to weeks to break down in surface water and groundwater. Very little trichloroethylene breaks down in the soil. It is found in groundwater and in some foods. It does not build up in fish or other animals. For more information on trichloroethylene in the environment, see Chapters 4 and 5.

1.3 HOW MIGHT I BE EXPOSED TO TRICHLOROETHYLENE?

Trichloroethylene is in the outdoor air at levels far less than 1 part trichloroethylene per one million parts of air (ppm). When measured several years ago, some of the water supplies in the United States were found to have trichloroethylene in them. The most recent monitoring study found levels of 0.04 ppm in surface water and 0.03 ppm in groundwater. About 400,000 workers are exposed to trichloroethylene in the United States on a full-time basis. It can also get into the air or water at waste treatment facilities; by evaporation from paints, glues, and other chemicals; or by accidental release from factories where it is made. Another way you may be exposed is by breathing the air around factories that use the chemical to remove grease from metals. People living near hazardous waste sites may be exposed to it in the air or in their drinking water. Products that may contain trichloroethylene are some types of typewriter correction fluids, paints and paint removers, glue, spot removers, rug cleaning fluids, and metal cleaners. For more information on exposure to trichloroethylene, see Chapter 5.

1.4 HOW CAN TRICHLOROETHYLENE ENTER AND LEAVE MY BODY?

Trichloroethylene can easily enter your body when you breathe air or drink water containing it. You could be exposed to contaminated water or air if you live near or work in a factory that uses trichloroethylene or if you live near a waste site that contains trichloroethylene. If you breathe the chemical, about half the amount you breathe will get into your bloodstream; you will exhale the rest. If you drink it, most of it gets into your blood. It can also enter your body if you get a lot on your skin.

Once in your blood, your liver changes trichloroethylene into other chemicals. These leave your body in the urine within a day. You will also quickly breathe out much of the trichloroethylene that is in your bloodstream. It is not likely to build up in your body. For more information on trichloroethylene in your body, see Chapter 2.

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1.5 HOW CAN TRICHLOROETHYLENE AFFECT MY HEALTH?

Trichloroethylene was once used as an anesthetic for surgery. People who breathe very high amounts become unconscious. People who are exposed to high levels of trichloroethylene can become dizzy or sleepy. Many people have jobs where they work with trichloroethylene and can breathe it or get it on their skin. Some people exposed to high levels of trichloroethylene have damage to some of the nerves in the face. It is uncertain whether these people are at higher risk for cancer or if their children may have higher numbers of birth defects. The International Agency for Research on Cancer has determined that trichloroethylene is not classifiable as to its carcinogenicity to humans. People who used water for several years from a certain well that had high levels of trichloroethylene may have had more leukemia in their families than other people. However, since other chemicals were also in the water from this well, we do not know whether trichloroethylene alone can cause leukemia. We do not know if trichloroethylene will affect human reproduction. Some people who get trichloroethylene on their skin develop rashes.

The Agency for Toxic Substances and Disease Registry has calculated Environmental Media Evaluation Guides (EMEGs) for trichloroethylene. EMEGs are derived from Minimal Risk Levels (MRLs) which are calculated from human or animal data for trichloroethylene. The MRLs are further described in Chapter 2 and in the footnote to Table 2-2. If a person is exposed to trichloroethylene at a level below the EMEG for the period listed below, we do not expect harmful health effects to occur. Because these levels are based only on information currently available, some uncertainty is always associated with them. Also, an EMEG does not imply anything about the presence, absence or level of risk for cancer because the methods for deriving EMEGs do not use any information about cancer. The EMEGs are provided as concentrations in order to allow for comparison to levels people might encounter in air, water, and soil around homes or in other areas where children may play.

Drinking water exposure

Drinking water EMEGs represent the lower end of a range and are protective for both children and adults.

- A drinking water EMEG of 1 ppm for trichloroethylene was derived from animal data for exposures longer than 14 days but less than one year.

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Soil exposure

Soil EMEGs represent the lower end of a range and are protective for both children and adults. However, this range is not protective for children (pica) who show increased desire for eating non-food items (such as soil).

- A soil EMEG of 5,000 ppm for trichloroethylene was derived from animal data for exposures longer than 14 days but less than one year.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO TRICHLOROETHYLENE?

There is a test that can tell if you have been recently exposed to trichloroethylene, since this chemical can be measured in your breath. Also, the doctor can have a number of breakdown products of trichloroethylene measured in your urine or blood. None of these tests is routinely available at your doctor's office. These tests can also tell whether you have been exposed to a large amount of trichloroethylene, or only a small amount. Because one of the breakdown products is removed very slowly from the body, it can be measured in the urine for up to about 1 week after trichloroethylene exposure. However, exposure to other similar chemicals can produce the same breakdown products in your urine and blood. Therefore, these methods cannot tell you for sure that you have been exposed to trichloroethylene. For more information on medical tests, see Chapters 2 and 6.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The EPA has set a drinking water standard of 5 parts of trichloroethylene per one billion parts of water (ppb, 1 ppb is 1,000 times less than 1 ppm). This standard became effective on January 9, 1989, and applies to community water systems and those which serve the same 25 or more persons for at least 6 months.

Trichloroethylene levels in the workplace are regulated by the Occupational Safety and Health Administration (OSHA). The occupational exposure limit for an 8-hour workday, 40-hour workweek is an average concentration of 50 ppm in air. The 15-minute average exposure that should not be exceeded at any time during a workday is 200 ppm. The OSHA standards do not take into consideration the cancer-causing potential of trichloroethylene. EPA requires industry to report spills of 1,000 pounds or more of trichloroethylene. It has been proposed that this level be reduced to 100 pounds. For more information, see Chapter 7.

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1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. These clinics specialize in the recognition, evaluation, and treatment of illnesses resulting from exposure to hazardous substances.

CHROMIUM

Source: ATSDR, 1991. "Draft Toxicological Profile for Chromium." Agency for Toxic Substances and Disease Registry, Atlanta, GA. October, 1991.

1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about chromium and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,300 sites on its National Priorities List (NPL). Chromium (total) has been found in at least 564 of these sites. However, we do not know how many of the 1,300 NPL sites have been evaluated for chromium. As EPA evaluates more sites, the number of sites at which chromium is found may change. This information is important for you to know because chromium may cause harmful health effects and because these sites are potential or actual sources of human exposure to chromium.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to chromium or chromium compounds, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS CHROMIUM?

Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. Chromium is present in the environment in several different forms. The most common forms are chromium(0), chromium(III), and chromium(VI). Chromium(III) occurs naturally in the environment whereas chromium(VI) and chromium(0) are generally produced by industrial processes. No known taste or odor is associated with chromium compounds. The metal chromium, which is the chromium(0) form, is a steel-gray solid with a high melting point. Chromium(0) is used mainly for making steel and other alloys. The naturally occurring mineral chromite in the chromium(III) form is used as brick lining for high-temperature, industrial furnaces. Chromium compounds, mostly in chromium(III) or chromium(VI) forms, produced by the chemical industry are used for chrome plating, the manufacture of dyes and pigments, leather, wood preservative, and treatment of cooling tower water. Smaller amounts are used in drilling muds, textiles, and toner for copying machines. For more information on

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the physical and chemical properties and on the production and use of chromium, see Chapters 3 and 4.

1.2 WHAT HAPPENS TO CHROMIUM WHEN IT ENTERS THE ENVIRONMENT?

Chromium enters the air, water, and soil as a result of natural processes and human activities. Emissions from burning coal and oil, steel production, stainless steel welding, and chromium manufacturing and use can increase chromium levels in air. Waste streams from electroplating, leather tanning, and textile industries can discharge chromium into waterways. The level of chromium in soil increases mainly from disposal of chromium-containing products, chromium wastes from factories, and coal ash from electric utilities. In air, chromium compounds are present mostly as fine dust particles. This dust eventually settles over land and water. Rain and snow aid in removing chromium from air. Chromium compounds will usually remain in the air for less than 10 days. Although most of the chromium in water settles in the material on the bottom, a small amount may dissolve in the water. Soluble chromium compounds can remain in water for years before settling out. Fish do not accumulate chromium from water into their bodies to any great extent. Most of the chromium in soil is water-insoluble or is bound to the soil. A very small amount of the chromium in soil, however, will dissolve in water and be carried deeper in the soil to groundwater. The movement of chromium in soil depends on the type and condition of the soil and other environmental factors. For more information about the fate and movement of chromium compounds in the environment, see Chapters 4 and 5.

1.3 HOW MIGHT I BE EXPOSED TO CHROMIUM?

You can be exposed to chromium by breathing air, drinking water, or eating food containing chromium or through skin contact with chromium or chromium compounds. The level of chromium in air and water is generally low. The concentration of total chromium in air is generally ranges between 0.01 and 0.03 microgram (μg). ($1 \mu\text{g} = 1/1,000,000$ of a gram) per cubic meter of air ($\mu\text{g}/\text{m}^3$). Chromium concentrations in drinking water are generally lower than 2 parts of chromium in a billion parts of water (2 ppb). For the general population, eating foods that contain chromium is the most likely route of chromium exposure. Chromium(III) occurs naturally in many fresh vegetables, fruits, meat, yeast, and grain. Various methods of processing, storage, and preparation can alter the chromium content of food. Acidic foods in contact with stainless steel cans or cooking utensils might contain higher levels of chromium because of leaching from stainless steel. Refining processes used to make white bread or sugar can decrease chromium levels. Chromium(III) is an essential element that humans need to properly use sugar. On the average, adults in the United States take in an estimated 60 μg of chromium daily. The officially recommended level for adults is 50 to 200 $\mu\text{g}/\text{day}$.

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People who work in industries that process or use chromium or chromium compounds can be exposed to higher-than-normal levels of chromium. An estimated 305,000 workers in the United States are potentially exposed to chromium and chromium-containing compounds in the workplace.

Occupational sources of chromium exposure may occur in the following industries:

- Stainless steel welding
- Chromate production
- Chrome plating
- Ferrochrome alloys
- Chrome pigments
- Leather tanning

Examples of other occupations that may involve chromium exposure include:

- Painters
- Workers involved in the maintenance and servicing of copying machines, and the disposal of some toner powders from copying machines
- Battery makers
- Candle makers
- Dye makers
- Printers
- Rubber makers
- Ship and boat builders
- Automobile and truck mechanics

A list of other industries that may be sources of occupational exposure are given in Section 5.5.

You may be exposed to higher-than-normal levels of chromium if you live near the following:

- Landfill sites with chromium-containing wastes
- Industrial facilities that manufacture or use chromium and chromium-containing compounds
- Cement-producing plants, because cement contains chromium
- Industrial cooling towers that use chromium as rust inhibitors
- Waterways that receive industrial discharges from electroplating, leather tanning, and textile industries
- Busy roadways, because emissions from automobile brake lining and catalytic converters contain chromium

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In addition, you may be exposed to higher levels of chromium if you use tobacco products, since tobacco contains chromium. For additional information about chromium exposure, see Chapter 5.

1.4 HOW CAN CHROMIUM ENTER AND LEAVE MY BODY?

Chromium can enter your body when you breathe air, eat food, or drink water containing chromium. Chromium(VI) enters the body more easily than chromium(III), but once inside the body, chromium(VI) is changed to chromium(III). When you breathe air containing chromium, chromium particles can be deposited in the lungs. However, you might swallow some of the chromium you breathe in as your body removes the chromium from your lungs. Some of the chromium in your lungs will enter the blood, pass through the kidneys, and be eliminated in the urine within a few days. Everyone normally eats or drinks a small amount of chromium daily. Most of the chromium that you swallow leaves your body within a few days through the feces and never enters your blood. A small amount (about 0.4% to 2.1%) will pass through the kidneys and be eliminated in the urine in a few days. Chromium(III) present in food can attach to other compounds that make it easier for chromium to enter your bloodstream from your stomach and intestines. This form of chromium is used by your body to carry out essential body functions. If your skin comes into contact with chromium, not very much will enter your body unless your skin is scraped or cut. For more information, please read Chapter 2.

1.5 HOW CAN CHROMIUM AFFECT MY HEALTH?

Chromium(III) is an essential nutrient that helps the body use sugar, protein, and fat. An intake of 50 to 200 μg of chromium(III) per day is recommended for adults. Without chromium(III) in the diet, the body loses its ability to use sugars, proteins, and fat properly, which may result in weight loss or decreased growth, improper function of the nervous system, and a diabetic-like condition.

The health effects resulting from exposure to chromium(III) and chromium(VI) are fairly well characterized. Breathing in high levels (greater than 0.002 milligram [mg] chromium per cubic meter of air [0.002 mg chromium/ m^3]) of chromium(VI) can cause soreness of the nose, ulcers, nose bleeds, and holes in the nasal septum. These effects have primarily occurred in factory workers who make or use chromium(VI) every workday over a few months to years. Breathing in small amounts of chromium(VI) for short or long periods does not cause a problem in most people. However, chromium can cause asthma attacks in people who are allergic to chromium. Breathing in chromium(III) does not cause

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irritation to the nose or mouth in most people. In the same way, small amounts of chromium(VI) that you swallow will not hurt you; however, intentional or accidental swallowing have caused stomach upsets and ulcers, convulsions, kidney and liver damage, and even death. The levels of chromium(VI) that caused these effects are far greater than those that you might be exposed to in food or water. Although chromium(III) in small amounts is an important nutrient needed by the body, swallowing large amounts of chromium(III) may cause health problems. Workers handling liquids or solids that have chromium(VI) in them have developed skin ulcers. Some people have been found to be extremely sensitive to chromium(VI) or chromium(III). Allergic reactions consisting of severe redness and swelling of the skin have been noted. Exposure to chromium(III) is less likely than exposure to chromium(VI) to cause skin rashes in chromium-sensitive people. The metal, chromium(0), is less common, and we do not know much about how it affects your health.

Animals that breathed high levels of chromium had harmful effects on the respiratory system and a lower ability to fight disease. However, we do not know if similar effects could occur in humans or if chromium can lower a person's ability to fight disease. We have no information that any form of chromium, when swallowed, has harmful effects on reproduction or causes birth defects in humans. However, some of the female mice that were given chromium(VI) by mouth had fewer offspring and had offspring with birth defects. Some male mice that were given chromium(VI) or chromium(III) by mouth had decreased numbers of sperm in the testes. The harmful effects seen as birth defects or as a decrease in sperm occurred in mice at levels about 100 to 1,000 times higher than the normal daily intake by humans.-

Long-term exposure to chromium has been associated with lung cancer in workers exposed to levels in air that were 100 to 1,000 times higher than those found in the natural environment ($<0.1 \mu\text{g}/\text{m}^3$). Lung cancer may occur long after exposure to chromium has ended. It is not clear which form(s) of chromium is capable of causing lung cancer in workers. Chromium(VI) is believed to be primarily responsible for the increased lung cancer rates observed in workers who were exposed to high levels of chromium in air. The Department of Health and Human Services has determined that chromium and certain chromium compounds are known carcinogens. For more information, please read Chapter 2.

The Agency for Toxic Substances and Disease Registry has calculated environmental media evaluation guides (EMEGs) for chromium. EMEGs are derived from Minimal Risk Levels (MRLs) which are calculated from human or animal data for chromium. The MRL is further described in Chapter 2 and the footnote to Table 2-1. If a person is exposed to chromium at a level below the EMEG for the period listed below, we do not expect harmful health effects to occur. Because this level is based only on information currently available, some uncertainty is always associated with it. Also, an EMEG does

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not imply anything about the presence, absence or level of risk for cancer because the methods for deriving EMEGs do not use any information about cancer. The EMEG is provided as a concentration in order to allow for comparison to levels people might encounter in air.

- An air EMEG of 0.00002 mg chromium/m³ for chromium(VI) was derived from human data for exposures longer than 14 days but less than 1 year.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO CHROMIUM?

Chromium can be measured in the hair, urine, serum, red blood cells, and whole blood. However, since chromium(III) is an essential nutrient, low levels of chromium are found in the body tissues and urine. Tests for chromium exposure are most useful for people exposed to high levels. Chromium levels in the urine and red blood cells indicate exposure to chromium(VI) or chromium(III) compounds. Since the body changes chromium(VI) to chromium(III), the form of chromium that you were exposed to cannot be determined. Because red blood cells last about 120 days before they are replaced by newly made red blood cells, the presence of chromium in red blood cells can show whether a person was exposed to chromium 120 days prior to testing but not if exposure occurred longer than 120 days before testing. Skin patch tests may indicate whether a person is allergic to chromium. For more information, please read Chapters 2 and 6.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

EPA has set the maximum level of chromium(III) and chromium(VI) allowed in drinking water at 0.05 mg chromium per liter of water (mg/L). According to the EPA, the following levels of chromium(III) and chromium(VI) in drinking water are not expected to cause harmful effects: 1.4 mg chromium/L water for 10 days of exposure for children, 0.24 mg chromium/L water for longer-term exposure for children, 0.84 mg chromium/L for longer-term exposure for adults, and 0.12 mg chromium/L water for lifetime exposure of adults.

The Occupational Safety and Health Administration (OSHA) regulates chromium levels in the workplace air. The occupational exposure limits for an 8-hour workday, 40-hour workweek are 0.5 mg chromium/m³ for water-soluble chromic (chromium(III)) or chromous (chromium(II)) salts and 1 mg chromium/m³ for metallic chromium (chromium(0)), and insoluble salts. The level of chromic acid and chromium(VI) compounds in the workplace air should not be higher than 0.1 mg chromium(VI)/m³ for any period of time.

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For chromium(VI) compounds that do not cause cancer, the National Institute for Occupational Safety and Health (NIOSH) recommends an exposure limit of 0.025 mg chromium(VI)/m³ for a 10-hour workday, 40-hour workweek. The levels of the chromium(VI) compounds that do not cause cancer should not be greater than 0.05 mg chromium(VI)/m³ for any 15-minute period. For chromium(VI) compounds that do cause cancer, NIOSH recommends an exposure limit of 0.001 mg chromium(VI)/m³ for a 10-hour workday, 40-hour workweek.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. These clinics specialize in the recognition, evaluation, and treatment of illnesses resulting from exposure to hazardous substances.

FLUORINE

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Chapter 22

FLUORINE

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1. CHEMISTRY

1.1. Distribution and Production

Fluorine is the most electronegative and one of the most reactive elements. That is why it is found in nature not in pure form but rather as fluor-spar (CaF_2), cryolite (Na_3AlF_6), and apatite [$\text{Ca}_5(\text{PO}_4)_3(\text{OH}, \text{Cl}, \text{F})$]. Furthermore fluorine is a secondary component of silicates like topaz [$\text{Al}_2\text{SiO}_3(\text{OH}, \text{F})$]. The percentage of the earth's uppermost crust (approximately 16 km deep) is about 0.065% [1].

Hydrofluoric acid serves as a corrosive in the glass and metal industry, and the mineral cryolite is used as a solvent for the electrolytic production of aluminum from bauxite. The annual production in 1978 amounted to 241,500 tons (United States), 87,300 tons (Japan), and 78,500 tons (FRG) [1].

1.2. Chemical Properties

Fluorine is a yellowish green gas with an intensive corrodent effect under normal conditions. It reacts very violently with practically all elements and compounds to form fluorine derivatives in which fluorine has always a single negative valence. By means of the high electronegativity of fluorine the maximal oxidation number of other elements is often obtained in reaction products like SF_6 , OsF_8 , and AgF_2 . Even noble gases form with fluorine stable products. The small atomic radius and the weakness of formation bond occupy an exceptional position among the halogens. Thus fluorine gas decomposes water while forming hydrofluoric acid, oxygen, ozone, and dioxygen fluoride. In organic compounds hydrogen is substituted by fluorine leading to an uncalculable number of products with high chemical and thermal resistance due to the stability of the C-F bond.

Hydrogen fluoride is a colorless, pungent gas which is miscible with water in all ratios. The aqueous solution (hydrofluoric acid) contains oligomeric forms of HF and produces, in addition to neutral salts, even hydrogen fluorides such as KHF_2 . For storage of hydrofluoric acid plastic or lead containers, at concentrations above 60%, or even iron vessels may serve.

2. TECHNOLOGY

2.1. Environment

Because of the widespread presence of fluorine, manifold environmental sources contribute to the daily fluoride intake. Elemental fluorine is used above all for preparation of inorganic and organic compounds, i.e., uranium hexafluoride for the separation of uranium isotopes, sulfur hexafluoride, an inert gas with some physical and chemical properties like those of nitrogen. Fluorine and some of its derivatives (chlorine trifluoride, boron trifluoride, nitrogen trifluoride, etc.) are components of rocket propellants, for fluorine has an exceptionally high specific impulse value.

Fluorine-emitting industries, such as the production of aluminum, superphosphate fertilizer, glass, ceramics, fluor-spar, and bricks, expose above all livestock to the danger of contamination of forage by fluorine-containing airborne dust. Resorption and retention of fluoride from brick dust was studied in milkers [2]. Additional sources of fluorine emissions are beryllium and antimony industries as well as power stations using brown coal for energy production [3]. In brown coal a fluorine content of 2-178 mg/kg was measured [4]. The airborne dust coming out of brown coal contained 10-102 mg/m² fluoride in the district of Cottbus (GDR) over a period of 30 days [5]. The hydrogen fluoride emission was estimated at 90 g/t coal [6]. The annual fluoride emissions of the brown coal power stations of the GDR amount to 4500 tons [4]. Fluoride pollution caused by the factories processing natural phosphate 10 km south of Safi (Morocco) induced a high daily intake in animals

which showed important signs and lesions of dental fluorosis. Contaminated plants had concentrations 4-10 times higher than in plants from the nonpolluted areas [7].

Alternating periods of high and low exposure to fluoride seems to have more damaging effects on animals than the continuous intake of the same total amount. General undernutrition also enhances the toxicity [8].

Recommended fluoride tolerance levels for cattle were established [9]. Water with a fluoride concentration of 30 mg/L given to wethers had deleterious effects, but a high content of minerals such as aluminum chloride had an alleviating influence by decreasing gut absorption of fluorine and reducing retention in the bones [10]. Inorganic phosphate ingredients in mixed-feed or mineral supplements are looked on as the principal source of fluoride in the feed. A case in which cattle deaths occurred following grazing on pasture freshly top-dressed with superphosphate is described [11].

In long-term residents near fluoride-emitting industries and in hot areas with high, natural fluoride levels in the drinking water family cases of dental fluorosis have occurred, the clinical appearance of which is characterized by lusterless opaque white patches in the enamel which may become stratified, mottled, or pitted. The relationship between waterborne fluoride, dental fluorosis, and skeletal development was demonstrated in 11- to 15-year-old Tanzanian girls. Fluoride concentrations in drinking water above 3 mg/L seemed to affect all mineralizing tissues under formation [12]. Skeletal fluorosis is often associated with nonspecific joint and muscle pains. Cases of nonoccupational skeletal fluorosis are diagnosed by x-ray examination [13-16]. Using a bone mineral analyzer in persons which had evidently no occupational contact but were under influence of fluorine-emitting sources, no significant differences in bone mineral content and width of radius were detected [17].

2.2. Industry

Skeletal fluorosis was first discovered as an occupational disease in cryolite workers in Copenhagen in 1932 [18]. Clinical examination of aluminum smelter workers showed a higher incidence of articular pain and limitation of motion in comparison with foundry workers of the same age. Urinary excretion and content of fluoride in bone obtained by biopsy were significantly increased [19]. Employees exposed to fluorine compounds showed increased calcium concentration but not higher magnesium levels in blood serum than a control group [20]. In another study clinical signs of vegetative vascular disorders were found: increased content of fluorine in blood and urine samples, reduction of brachial and temporal arterial pressure, contractile function of the heart and urinary excretion of adrenaline [21]. An epidemiological health study of 2066 workers in an aluminum smelter in Kitimat, British Columbia revealed no definitive cases of skeletal fluorosis, but in a few workers who had been employed in the aluminum smelters for more than 10 years periosteal changes, increased density, and calcification were found [22]. Investigations were undertaken with regard to nasal mucosa [23], periodontium and mouth mucosa [24-26], and cardiorespiratory system [27-29]. Some physical properties of bones altered by chronic industrial fluorosis were examined and compared with those of control persons [30].

Hydrofluoric acid is widely used in the glass, electronics, and chemical industries because of its ability to dissolve silica and etch glass. It is additionally a primary component of rust-removing agents. Toxicity depends on the concentration and total amount of acid, duration of contact, and portal of entry. Hydrofluoric acid has a severe action on body tissue and produces serious burns which are painful and characterized by progressive destruction and necrosis of tissue. Inhalation injuries produce oropharyngeal and respiratory distress and pulmonary edema [31]. Contact of liquid or vapor with the eyes can rapidly cause irritation and, if treatment is delayed, may produce burns with permanent visual defects or total loss of vision [32,33].

Hexafluorosilicate used as gelifier for the structural formation of foam rubber was the likely cause of pustular reactions observed earlier with ammonium fluoride and sodium fluoride [34]. The production of fluor-containing polymers is of particular importance because of the relatively high thermal stability, chemical and biochemical inertness, and low friction coefficients of these materials, in particular polytetrafluoroethylene (PTFE) and polyfluoroethylenepropylene (PFEP). The main pyrolysis products of PTFE and PFEP in a nitrogen stream are tetrafluoroethylene, hexafluoropropylene, octafluorocyclobutan, and octafluoroisobutylene. In air stream carbonyl fluoride and trifluoroacetyl fluoride are formed, which produce among other compounds hydrogen fluoride [35]. These products cause respiratory impairment in experiments with rats [36] and birds [37] and can result in an acute illness in man termed "polymer fume fever." Examples of occupational injuries have also been given recently [38-41].

2.3. Pharmaceuticals

Use of fluoride in the prevention of caries and in the treatment of osteoporosis without production of adverse effects requires cognizance of all sources of fluoride exposure. Some literature reviews give detailed surveys of toxic side effects, advantage and disadvantage of water fluoridation, and topical application of fluorides [42,43]. In the United States nowadays nearly 106 million persons receive fluoridated water [44]. Six cases of accidental overfluoridations of drinking water are described [42,45,46]. Severe complications occurred only in patients with renal insufficiency after hemodialysis with the overfluoridated water which contained 30 mg/L fluoride [45,47]. Many other methods of fluoridation procedures were tested such as drinking water fluoridation in schools, fluoride supplementation of food, milk, and salt; and ingestion of sodium fluoride tablets [48]. On the other hand, interest has shifted from systemic to topical applications of fluoride because of comparatively higher risk of accidental ingestion of tablets and since fluoridation of communal water supplies is still controversial [43].

From 1978 to 1983 in Brisbane, Australia, 20 children were admitted to hospitals after ingestion of sodium fluoride tablets. The highest recorded ingested dose was approximately 6.3 mg/kg of body weight which required a stay of 3 days in hospital [49]. 150 cases of such ingestions ranged from 1 to 5 mg/kg [50]. Reviews of the different applications of fluoride and its potential dangers have been published [51-53]. The toxicity of inorganic fluorine compounds used as anticariogenic agents, monofluorophosphate [54], and pentafluorostannite was examined more closely [55,56]. The risk of acute

toxicity is minimal if preparations for household usage are dispensed only in appropriate quantities and child-proof packaging.

The question as to whether there is any association between water fluoridation and cancer mortality has been heatedly discussed [57,58]. Recent studies have not identified any risk to all kinds of cancer associated with the additional daily fluoride intake of about 1 mg [59,60]. Likewise no correlation was revealed to levels of naturally occurring fluoride in water supplies [61] while a possible effect of heavy occupational fluoride exposure on cancer morbidity could not be excluded, in particular cancer of the respiratory system [62].

3. PHYSIOLOGY

3.1. Resorption and Elimination

Because of the widespread occurrence of fluoride in the biotic and abiotic nature, a complex regulating system in animals serves to equalize the fluctuations of the daily intake. Compartment analysis in rabbits showed that the serum fluoride is a good steady-state predictor of the concentrations in other compartments of the body. There is a lack of evidence of homeostatic control of serum fluoride levels, but daily fluctuations of fluoride in blood are usually held within a narrow range of about 30% change by the damping action of the very large bone compartment, which contains 99.7% of the fluoride in the body, if the nonabsorbed fluoride in the gastrointestinal tract is excluded. A direct relationship exists between intake and serum fluoride as well as bone, urine, milk, salivary, placental, and cord serum levels [63].

Fluoride is rapidly resorbed from the gastrointestinal tract and first distributed in the extracellular compartment and then in the whole body fluid. The elimination of low doses (2-5 mg) occurs with logarithmic elimination-characteristic and a half-life of 3-4 hr [64,65]. The plasma clearance of 0.1 mg/kg·hr contains the combination of the renal (50 ml/min) and the extrarenal clearance (about 110 ml/min) which applies mainly to the bone compartment [66,63]. Normal serum fluoride content ranges from 0.01 to 0.04 mg/L, soft organs contain 0.2-0.8 mg/kg dry tissue [67,69].

Long-term intake of fluoride in drinking water led to an increase of average plasma levels in humans from three cities in Texas and two in the State of New York with a linear relationship to drinking water concentration. A fluoride concentration in drinking water of 1 mg/L resulted in levels of about 0.02 mg/L plasma and that of 2 mg/L in 0.035 mg/L plasma [63].

A single dose of 5-10 g sodium fluoride (70-100 mg/kg) given to previously unexposed adults is regarded as lethal [50,52]. If fluoride is administered in solution or in an acid environment, absorption takes place more rapidly and because of the direct relationship of toxicity to plasma concentration its toxic effect will be greater. An intake of hydrofluoric acid can cause fatality after 1 hr, in the course of which severe irritation and damage to the gastrointestinal tract occurs which may be accompanied by necrotizing pancreatitis [68]. Solely as a result of a skin burn of 9-10% body surface area on the lower extremities did a systemic dissemination of fluoride cause fatality after severe hypocalcemia and recurrent ventricular arrhythmias had set in [69]. In previously described fatalities from hydrofluoric burns, in

one case with only a 2.5% body surface area, additional damage to the respiratory tract with pneumonitis and hemorrhagic pulmonary edema had occurred [70].

Symptoms of fluoride intoxication, usually occurring within 1 hr, range from salivation, abdominal pain, nausea, vomiting, and diarrhea in cases of minor poisoning, to hyperactive reflexes, tonic and clonic convulsions, and, ultimately, in severe cases, death occurs, usually from respiratory or cardiac failure [52]. If kidney function is impaired, the excretion of fluoride may be slowed; an apparently sublethal dose causes fatality under certain conditions.

Serum fluoride content rose to 56.2 mg/L after intake of hydrofluoric acid [68]. But fatalities occurred after hydrofluoric acid burns with serum levels of 3 mg/L [70] and 4.17 mg/L fluoride, respectively, [69] while a child survived owing to intensive therapy with a serum fluoride concentration of 14 mg/L [71]. Postmortem fluoride concentrations in soft organs lay in the same order of magnitude [68-70]. Extensively decreased calcium levels were observed in the case of a fatally burned person (22-35 mg/L; normal range 88-103) despite intravenous administration of calcium (280 meq) during the last 4 hr of his life [70]. In addition, serum concentrations of magnesium were reduced to 6 and 19 mg/L (normal range 19.9-27.5 [72]). A chronic inhalation of hydrofluoric acid fumes led to a mean urinary fluoride concentration of 12.02 mg/L in three employees; after the installation of a local exhaust ventilation the mean value was reduced to 1.41 mg/L [73] (normal range 0.43-3.5 mg/L [72]).

3.2. Osteosclerosis

Combined doses of calcium and fluoride are effective in the treatment of osteoporosis. A daily intake of 40-60 mg of sodium fluoride, i.e., approximately 10 times higher than that which is used for the prevention of tooth decay, leads to increasing bone formation [74]. Mechanism of action is rather an osteoblastic stimulation than the prevention of crystal dissolution [75]. At effective serum levels of 0.2-0.3 mg/L fluoride the normal fluorine content of bones (1.15 mg/kg [76]) is increased to multiple values by replacement of hydroxyl groups by fluoride in apatite. Diminution of pain was observed in steroid-induced osteoporosis at a rate of 90% [76]. The renal clearance can be reduced by renal disease causing severe osteosclerosis even in patients with moderate renal failure [77]. Thus a lower dosage of sodium fluoride should be given in such cases. X-ray controls in intervals of 6-8 months are essential for stating the osteoporosis and the potential stage of osteosclerosis [78]. Adverse effects during treatment of osteoporosis with sodium fluoride, calcium gluconate, and vitamin D were observed which led to osteomalacia [79]. An attempt to explain the contradictory effects of fluoride causing such different bone diseases—osteosclerosis, osteomalacia, secondary hyperparathyroidism and osteoporosis—was made by Franke [74].

3.3. Other Biochemical Effects

The best known example of an inhibitory effect is the competitive inhibition of enolase by fluoride concentrations exceeding 0.03 mM (0.6 mg/L) in the presence of 5 mM phosphate [80]. Other enzymes which are inhibited by

fluoride are acid phosphatase, ATPase, succinic dehydrogenase, and cholinesterase. The inhibition of protein synthesis is obviously the main cytotoxic effect of fluoride [81], while the cellular metabolism and in particular the glycolytic pathway are not affected in vivo by inhibition of enolase. Further cellular parameters that were affected by fluoride concentrations causing growth inhibitory effect were DNA synthesis and the activity of ornithine decarboxylase, and were assumed as results of decreased protein synthesis. A review of the cytotoxic effects of fluoride is given by Holland [81], and regarding the wide field of fluorinated substrate analogs, routes of metabolism and selective toxicity are covered by Walsh [82].

4. DETOXIFICATION

In the case of skin contamination with hydrofluoric acid first aid consists of washing all exposed areas with copious amounts of cold water. Calcium gluconate gel (2.5%) is massaged into all the affected parts [83,84]. The medical procedures have been reviewed in the literature [85-87]. Arterial perfusion of 20% calcium gluconate is preferred to the infiltration method previously used by some authors [88,89]. For burns involving a large area (more than 25 in.²) early and frequent monitoring of serum calcium and magnesium, with systemic replacement by calcium gluconate tablets or infusions when indicated, is essential in order to prevent hypocalcemia, which can lead to ventricular arrhythmias and QT interval prolongation in the electrocardiogram, though clinical signs of tetany may be absent [70]. In experiments with rats metabolic alkalosis produced by infusion of sodium bicarbonate showed a favorable influence in the course of acute fluoride toxicity. In particular, renal clearance was higher compared with a control group [90].

5. LEVELS OF TOLERANCE

The industrial threshold limit value (MAK) for elementary fluorine in the Federal Republic of Germany is now 0.2 mg/m³ and for hydrofluoric acid 2.5 mg/m³ [91]. Biological threshold values (BAT) have been fixed for hydrofluoric acid and its inorganic compounds with reference to urinary excretion. Threshold value before the work-shift is fixed at 4 mg fluoride/g creatinine; after the shift 7 mg fluoride/g creatinine [91].

In Great Britain a threshold limit value of 2 mg/m³ for hydrogen fluoride is given [73]. A revision of the existing industrial threshold values of 0.5 and 1 mg/m³ for hydrogen fluoride and inorganic salts of hydrofluoric acid, respectively, is recommended in the USSR, resulting in 0.05 mg/m³ for hydrogen fluoride, 0.2 mg/m³ for salts readily soluble in cold water, and 0.5 mg/m³ for slightly soluble fluorides [92]. The values in the United States are for fluorine 2 mg/m³ (TLV-TWA) and 4 mg/m³ (TLV-STEL), whereas for fluorides (calculated as F) the TLV-TWA is 2.5 mg/m³; the values for hydrogen fluoride (calculated as F) are 2.5 mg/m³ (TLV-TWA) and 5 mg/m³ (TLV-STEL) [103].

The daily intake with food ranges from 0.3 mg in districts using nonfluoridated water to about 2 mg fluoride in communities with fluoridated drinking water [93]. A fluoride concentration of 1 mg/kg in food and drinking water

is supposed to produce no adverse effects [94]. A phosphorus-to-fluoride ratio of 100:1 is regarded as a safe level of fluoride in supplemental feed phosphates [95].

6. SUMMARY OF ECOTOXICITY

Taves comes to the conclusion that evidence of an increase in fluoride intake is based on faulty analyses and that an increase of total intake is less than 10% comparing data obtained with reliable methods in urine specimens [93]. Because of the widespread occurrence of fluorine, a careful control of environmental sources has to be maintained for the daily intake within the small range essential for health. It is proved that the urinary fluoride excretion is suitable to monitor exposed persons [96].

7. ANALYTICAL CHEMISTRY

Analytical methods for quantification of fluorine in biological specimens were compared in detail by Guy [97]. Contamination can occur through the use of glassware for collection and storage of samples and theoretically by effect of heat on organic fluoro compounds contaminating laboratory air.

A direct measurement of fluoride in drinking water, urine, and serum is practicable with a fluoride ion-sensitive electrode [98]. In the case of analysis of blood and tissues containing levels in the low range, a microdiffusion after ashing with a fluoride fixative at 400-600°C is carried out. The observation that silicones facilitate separation of fluoride by diffusion is regarded as an important development [97]. The spectrophotometric method using lanthanum-alizarin complexone has recently been applied to the fluoride determination in organs and body fluids after microdiffusion from the biological matrix [68]. Latest values of fluoride levels especially in enamel and dentine have been published [99]. The content of fluoride in bones was determined by combustion at 450°C, steam distillation with perchloric acid, and colorimetric analysis using zirconium-eriochrome-cyanine-complexone [100]. The gas chromatographic method after extraction as fluorosilane developed by Fresen et al. [101] was used for the determination of fluoride in serum [102].

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LEAD

Source: ATSDR, 1991. "Draft Toxicological Profile for Lead." Agency for Toxic Substances and Disease Registry, Atlanta, GA. October, 1991.

1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about lead and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,300 sites on its National Priorities List (NPL). Lead has been found in at least 666 of these sites. However, we do not know how many of the 1,300 NPL sites have been evaluated for lead. As EPA evaluates more sites, the number of sites at which lead is found may change. This information is important for you to know because lead may cause harmful health effects and because these sites are potential or actual sources of human exposure to lead.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous chemical such as lead, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS LEAD?

Lead is a naturally occurring bluish-gray metal found in small amounts in the earth's crust. It has no characteristic taste or smell. Lead does not dissolve in water and does not burn. Some natural and man-made substances contain lead, but do not look like lead metal. Some of the substances that contain lead can burn.

Lead has many different uses. Its most important use is in the production of some types of batteries. Other uses include the production of ammunition and some kinds of products (such as sheet lead, solder, and pipes). Some chemicals containing lead, such as tetraethyl lead and tetramethyl lead, are used as gasoline additives. However, the use of these lead-containing chemicals in gasoline is much less than it used to be because these additives are being phased out. Other chemicals containing lead are used in paint. The amount of lead added to paints and ceramic products, roofing, caulking, ammunition, gasoline additives, and solder has been reduced in recent years because of lead's harmful effects in humans and animals.

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Most lead used by industry comes from mined ores or from recycled scrap metal or batteries. Human activities (such as use of "leaded" gasoline) have spread lead and substances that contain lead to all parts of the environment. For example, lead is in air, drinking water, rivers, lakes, oceans, dust, and soil. Lead is also in plants and animals that humans may eat. Please see Chapter 3 for more information on the physical and chemical properties of lead. Chapter 4 contains more information on the production and use of lead.

1.2 WHAT HAPPENS TO LEAD WHEN IT ENTERS THE ENVIRONMENT

Lead occurs naturally in the environment. However, most of the lead dispersed throughout the environment comes from human activities. Before the use of leaded gasoline was limited, most of the lead released into the environment came from car exhaust. Since the EPA has limited the use of leaded gasoline, the amount of lead released into the air has decreased. In 1979, cars released 94.6 million kilograms (kg) of lead into the air in the United States. In contrast, in 1989 cars released only 2.2 million kg to the air. Other sources of lead released to the air include burning fuel, such as coal or oil, industrial processes, and burning solid waste.

The release of lead to air is now less than the release of lead to soil. Most of the lead that enters soil comes from landfills. Landfills contain waste from lead ore mining, ammunition manufacturing, and from other industrial activities such as battery production. Very little lead goes directly into water.

High levels of lead from car exhausts can be measured near roadways. Very low levels of lead from car exhausts are found at distances of 25 meters from the road edge. However, once lead goes into the atmosphere, it may travel thousands of miles if the lead particles are small. Lead is removed from the air by rain as well as by particles falling to the ground or into surface water. Once lead deposits on soil, it usually sticks to soil particles. Small amounts of lead may enter rivers, lakes, and streams when soil particles wash in along with rainwater. Lead may remain stuck to soil particles in water for many years. Movement of lead from soil particles into groundwater or drinking water is unlikely unless the water is acidic or "soft."

Some of the chemicals that contain lead are broken down by sunlight, air, and water to other forms of lead. Lead is not broken down although it may become part of other chemicals that contain lead. Lead in water will combine with different chemicals depending on the acidity and temperature of the water.

The levels of lead may build up in plants and animals from areas where air, water, or soil are contaminated with lead. If animals eat contaminated plants or animals, most of the

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lead that they eat will pass through their bodies. Chapters 4 and 5 contain more information on the environmental fate of lead.

1.3 HOW MIGHT I BE EXPOSED TO LEAD?

People living near hazardous waste sites can be exposed to lead and chemicals that contain lead by breathing air, drinking water, eating foods, or swallowing dust or dirt that contains lead. For people who do not live near hazardous waste sites, most exposure to lead occurs by eating foods that contain lead. Foods such as fruits, vegetables, meats, grains, seafood, soft drinks, and wine may have lead in them. Cigarettes also contain small amounts of lead. In general, very little lead is in drinking water. Over 99% of all drinking water contains less than 0.005 parts of lead per million of water (ppm). However, the amount of lead taken into your body through drinking water can be higher in communities with acidic water supplies. Acid in water can make the lead found in lead pipes, solder, and brass faucets enter water.

Exposure to gasoline additives that contain lead can happen while you are pumping leaded gasoline, from sniffing leaded gasoline, and possibly during the use of some do-it-yourself fuel additives. For people who are exposed to lead at work, the largest source of exposure comes from breathing air that contains lead. Breathing or swallowing dust and dirt that has lead in it is another way you can be exposed to lead. Children, especially those who are preschool age, can have a lot of lead exposure because they put many things into their mouths. Their hands, toys, and other items may have lead-containing dirt on them. In some cases, children swallow nonfood items such as paint chips and dirt. These items may contain very large amounts of lead, particularly in and around older houses that were painted with lead-based paint. The paint in these houses often chips off and mixes with dust and dirt. Skin contact with dust and dirt containing lead occurs every day. However, not much lead can get into your body through your skin. During normal use of lead-containing products, very little lead gets on your skin.

The burning of gasoline has been the single largest source (90%) of lead in the atmosphere since the 1920s. A lot less lead in the air comes from gasoline now because EPA reduced the amount of lead that can be used in gasoline. Less than 35% of the lead released to the air now comes from gasoline. Other sources of lead in the air include releases to the air from industries involved in iron and steel production, lead-acid-battery manufacturing, and manufacturing of tetraethyl and tetramethyl lead. Lead released into air may also come from burning of solid waste, windblown dust, volcanoes, exhaust from workroom air, burning or weathering of lead-painted surfaces, and cigarette smoke.

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Sources of lead in drinking water include lead that can come out of lead pipes, faucets, and solder used in plumbing. Lead-containing plumbing may be found in public drinking water systems, in houses, apartment buildings, and public buildings. Sources of lead in surface water or sediment include deposits of lead-containing dust from the atmosphere, wastewater from industries that handle lead--primarily iron and steel industries and lead producers--urban runoff.

Sources of lead in food and beverages include deposition of lead-containing dust from the atmosphere on crops and during food processing and uptake of lead from soil by plants. Lead may also enter foods when foods are put into improperly glazed pottery and ceramic dishes. Illegal whiskey made using stills that contain lead-soldered parts (such as truck radiators) may also contain lead.

Sources of lead in dust and soil include deposition of atmospheric lead and weathering and deterioration of lead-based paint. Lead in dust may also come from windblown soil. Disposal of lead in municipal and hazardous waste dump sites also adds lead to soil.

Exposure to lead occurs in many jobs. People employed in lead smelting and refining industries, rubber products and plastics industries, steel welding and cutting operations, battery manufacturing plants, and alkyl lead manufacturing industries may be exposed to lead. People who work at gasoline stations, in construction work and at do-it-yourself renovations, or who work at municipal waste incinerators, pottery and ceramics industry, radiator repair shops and other industries that use lead solder may also be exposed. Between 0.5 and 1.5 million workers are exposed to lead in the workplace; in California alone over 200,000 workers are exposed to lead. Families of workers may be exposed to elevated levels of lead when workers bring home lead dust on clothes worn at work. Chapter 5 contains further information on sources of exposure to lead.

1.4 HOW CAN LEAD ENTER AND LEAVE MY BODY?

Lead can enter your body when you breathe in lead dust or chemicals that contain lead. Most of the lead that gets into your lungs goes quickly to other parts of the body in your blood.

You may swallow a lot of lead by eating food and drinking liquids that contain it. However, very little of the amount you swallow enters your blood and other parts of your body. The amount that gets into your body from your stomach partially depends on when you ate your last meal. It also depends on how well the lead particles you ate dissolved in your stomach juices. Experiments in adult volunteers showed that the amount of lead that got into the body from the stomach was only about 6% in adults who had just eaten. About 60-80% of the lead in the stomach of adults who had not eaten for a day got into

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their blood. On the other hand, 50% of the lead swallowed by children enters the blood and other body parts even if their stomachs are full.

You come in frequent skin contact with lead in the form of lead-containing dusts and soil. However, only a small portion of the lead will enter your body after skin contact.

Lead behaves the same once it gets into your body no matter what the route of exposure was. Shortly after it gets into your body, lead travels in the blood to the "soft tissues," (such as the liver, kidneys, lungs, brain, spleen, muscles, and heart). After several weeks most of the lead then moves into your bones and teeth. In adults, about 94% of the total amount of lead in the body is contained in their bones and teeth. Children, on the other hand, have only about 73% of the lead in their bodies stored in their bones. The rest is in their organs and blood. Part of the lead stays in your bones for decades. Part of the lead in your bones is available to reenter your blood and organs.

Your body does not change lead into any other chemical. Once it is taken in and distributed to your organs, the lead that is not stored in your bones leaves your body in your urine or your feces. About 99% of the amount of lead that you take into your body will leave in your waste within a couple of weeks, but only about 32% of the lead taken into the body of children will leave in the waste. For more information on how lead can enter and leave your body, please refer to Chapter 2.

1.5 HOW CAN LEAD AFFECT MY HEALTH?

Exposure to lead is particularly dangerous for unborn children, because of their great sensitivity during development. Exposure to lead is also dangerous for young children, because they swallow more lead through normal mouthing activity, take more of the lead that they swallow into their bodies, and are more sensitive to its effects. The American Academy of Pediatrics has concluded that lead continues to be a significant hazard to the health of children in the United States, and that most children in this country are exposed to lead due to pollution of the environment by the burning of leaded gasoline and by the weathering of lead-based paint. Unborn children can be exposed to lead through their mothers. This may cause premature births, smaller babies, and decreased mental ability in the infant. Lead exposure may also decrease intelligence quotient (IQ) scores and reduce the growth of young children.

In adults, lead exposure may decrease reaction time and possibly memory. Lead exposure may also cause weakness in your fingers, wrists, or ankles. Lead exposure may increase blood pressure in middle-aged men. It is not known whether lead has an effect on blood pressure in women. Lead exposure may also cause anemia. At high levels of exposure, lead can severely damage the brain and kidneys in adults or children. In addition, high

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levels of exposure to lead may cause abortion and damage the male reproductive system. The effects of lead are the same regardless of whether it enters the body through breathing or swallowing.

Tumors have developed in rats and mice given large doses of lead. The Department of Health and Human Services has determined that lead acetate and lead phosphate may reasonably be anticipated to be carcinogens. Please see Chapter 2 for more information on the health effects of lead.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO LEAD?

The amount of lead in the blood can be measured to determine if exposure to lead has occurred. Methods to measure lead in teeth or bones by X-ray techniques, although not widespread, also are available.

Exposure to lead can also be evaluated by measuring erythrocyte protoporphyrin (EP). EP is a part of red blood cells known to increase when the amount of lead in the blood is high. This method is commonly used to screen children for potential lead poisoning. The Centers for Disease Control (CDC) considers lead poisoning in children to exist if the amount of lead in the blood is at least 25 micrograms per deciliter ($\mu\text{g}/\text{dL}$). High levels in these tests suggest that adverse health effects may occur. Medical treatment to lower blood levels may be necessary if the lead concentrations in blood are high. For more information on tests to measure lead in the body, see Chapters 2 and 6.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The CDC recommends that all children should be screened for lead poisoning at least once a year. This is especially important for children between the ages of 6 months and 9 years. Children with blood lead levels of 25 $\mu\text{g}/\text{dL}$ or EP levels of 35 $\mu\text{g}/\text{dL}$ or greater should be tested by their doctors for symptoms of lead poisoning without delay. The CDC is currently considering lowering the blood lead level of concern below 25 $\mu\text{g}/\text{dL}$.

EPA requires that the concentration of lead in air that the public breathes shall not exceed 1.5 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) averaged over 3 months. EPA regulations now limit the level of lead in leaded gasoline to 0.1 grams per gallon (0.1 g/gal) and the level in unleaded gasoline to 0.001 g/gal.

EPA regulations also limit lead in drinking water to 0.015 milligrams per liter (mg/L).

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The Consumer Product Safety Commission (CPSC), EPA, and the states are required by the 1988 Lead Contamination Control Act to deal with the problem of lead in drinking water coolers by requiring that water coolers containing lead be recalled or repaired and that new coolers be lead-free. In addition, drinking water in schools must be tested for lead and the sources of lead in this water must be removed.

To help protect small children, the CPSC requires that the concentration of lead in most paints available through normal consumer channels be not more than 0.06%. The CDC recommends that inside and outside painted surfaces of dwellings be tested for lead, and that surfaces containing lead equal to or greater than 0.7 milligram per square centimeter (mg/cm^2) of surface area be stripped and repainted according to a four-step paint removal and replacement protocol. This is necessary because stripping can release fine particles of lead that can cause lead poisoning. The CDC has also warned that concentrations of lead in soil or dust greater than 500-1,000 micrograms per gram ($\mu\text{g}/\text{g}$) could lead to elevated blood lead levels in children who breathe or swallow the dirt.

The Department of Housing and Urban Development (HUD) requires that federally funded housing and renovations, public housing, and Indian housing be tested for lead-based paint hazards and that such hazards be fixed by covering the paint or removing it. HUD is carrying out demonstration projects to determine the best ways of covering or removing this paint in housing.

The Occupational Safety and Health Administration (OSHA) regulations limit the concentration of lead in workroom air to $50 \mu\text{g}/\text{m}^3$ for an 8-hour workday.

Please see Chapter 7 for more information on federal and state regulations and guidelines for lead.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

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This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. These clinics specialize in the recognition, evaluation, and treatment of illnesses resulting from exposure to hazardous substances.

MERCURY

Source: ATSDR, 1992. "Draft Toxicological Profile for Mercury." Agency for Toxic Substances and Disease Registry, Atlanta, GA. October, 1992.

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This Statement was prepared to give you information about mercury and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,300 hazardous waste sites as the most serious in the nation. These sites comprise the "National Priorities List" (NPL): Those sites which are targeted for long-term federal cleanup activities. Mercury has been found in at least 600 of the sites on the NPL. However, the number of NPL sites evaluated for mercury is not known. As EPA evaluates more sites, the number of sites at which mercury is found may increase. This information is important because exposure to mercury may cause harmful health effects and because these sites are potential or actual sources of human exposure to mercury.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You can be exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking substances containing the substance or by skin contact with it.

If you are exposed to a substance such as mercury, many factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, gender, nutritional status, family traits, life-style, and state of health.

1.1 WHAT IS MERCURY?

Mercury is a chemical (element) that occurs naturally in the environment in several forms (Table 1-1). In the metallic or elemental form, mercury is a shiny, silver-white, odorless liquid with a metallic taste. Mercury can also combine with other elements, such as chlorine, carbon, or oxygen, to form mercury compounds. These compounds are called "organic mercury" if they contain carbon, and "inorganic mercury" if they do not. In pure form, these mercury compounds are usually white powders or crystals. All forms of mercury are considered poisonous. One organic form of mercury, methylmercury, is of particular concern because it can build up in certain fish (see Section 1.2 below). For this reason, rather low levels of mercury in the oceans and lakes can contaminate these fish.

Mercury released into the environment stays there for a long time. Once in the environment, mercury can slowly be changed from organic to inorganic forms and vice versa by microorganisms and natural chemical processes. Methylmercury is the organic form of mercury created by these natural processes.

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TABLE 1-1. Sources and Uses of Mercury

Name	Form	Source or use
Mercury	Metallic or Elemental (Hg^0)	Chlorine-alkali manufacturing Dental fillings Gold mining Electrical equipment (batteries, switches) Instruments (thermometers, barometers)
Mercuric mercury	Inorganic (Hg^{+2})	Electrical equipment (batteries, lamps) Skin care products Medicinal products
Mercurous mercury	Inorganic (Hg^{+1})	Electrical equipment (batteries) Medicinal products
Methylmercury	Organic ($\text{CH}_3\text{Hg}^{+1}$)	Diet (e.g., contaminated fish) Polluted sediment
Phenylmercury	Organic ($\text{C}_6\text{H}_5\text{Hg}^{+1}$)	Fungicides Pigments (paints)

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There are many different uses for and sources of mercury. Metallic mercury is mined and is also a waste product of gold mining. Chemical factories that make chlorine use mercury and may release metallic mercury into the air. Thermometers, barometers, batteries, and tooth fillings all contain metallic mercury. Inorganic mercury compounds are commonly used in electrical equipment (for example, batteries, lamps) and skin care and medicinal products. Some inorganic mercury compounds are used in fungicides. Methylmercury is generally produced in the environment, rather than made by human activity. Fungicides and paints may contain other organic mercury compounds. Mercury compounds may be found in the air, soil, and water near hazardous waste sites.

Chapter 3 contains more information on the physical and chemical properties of mercury. Chapter 4 contains more information on the production and use of mercury.

1.2 WHAT HAPPENS TO MERCURY WHEN IT ENTERS THE ENVIRONMENT?

Mercury is a naturally occurring metal found throughout the environment as a result of normal breakdown of the earth's crust by wind and water. The total amount of mercury in the environment caused by natural processes throughout the world is far greater than the total amount caused by human activities. However, the amount of mercury that exists in any one place through natural processes is usually very low. In contrast, the amount of mercury that may be at a particular waste site because of human activity can be very high. Air, water, and soil can contain mercury from both natural sources and human activity.

The mercury in air, water, and soil is thought to be mostly inorganic mercury. This inorganic mercury can enter the air from deposits of ore that contain mercury, from the burning of fuels or garbage, and from the emissions of factories that use mercury. Inorganic mercury may also enter water or soil from rocks that contain mercury, releases of water containing mercury from factories or water treatment facilities, and the disposal of wastes. Organic compounds of mercury may be released in the soil through the use of mercury-containing fungicides.

Metallic mercury is a liquid at room temperature. It can evaporate easily into the air and be carried a long distance before returning to water or soil in rain or snow. As mentioned before, some microorganisms in the water or soil can change inorganic forms of mercury to organic forms. Organic forms of mercury can enter the water and remain there for a long time, particularly if there are particles in the water to which they can attach. If mercury enters the water in any form, it is likely to settle to the bottom where it can remain a long time. Mercury also remains in soil for a long time. Mercury usually stays on the surface of the sediments or soil and does not move through the soil to underground water.

Small fish and other organisms living in the water can take up the organic forms of mercury. When larger fish eat these small fish or other organisms that contain organic mercury, their bodies will store most of it. In this way, large fish living in contaminated waters can collect a relatively large amount of organic mercury. Plants may also have a

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greater concentration of mercury in them if they are grown in soil that contains higher than normal amounts of mercury. For further information on what happens to mercury in the environment, please refer to Chapters 4 and 5.

1.3 HOW MIGHT I BE EXPOSED TO MERCURY?

Because mercury occurs naturally in the environment, everyone is exposed to very low levels of mercury in air, water, and food. Sources of higher exposure to metallic mercury include breathing air containing mercury in the workplace or any place where mercury might have been spilled. Also, since amalgam dental fillings are about half metallic mercury, if you have them you can be exposed to mercury levels that are higher than the levels normally found in the environment. People with dental fillings containing mercury generally have more mercury in their breath than those who do not have these fillings. However, there is not enough evidence to prove that the mercury in amalgam fillings is causing health effects in humans.

Sources of exposure to inorganic mercury include swallowing or inhaling dust that contains mercury particles in the workplace and using skin care and medicinal products with small amounts of mercury in them. You can also be exposed to inorganic mercury by drinking water that is contaminated with mercury. For most people, eating contaminated fish is the major source of organic mercury exposure. Some fish contain such high levels of mercury that eating them has been prohibited. Other foods typically contain very little mercury. A greater risk of mercury exposure may occur in fetuses exposed to mercury in their mother's blood and in nursing children who may be exposed to mercury in their mother's milk. Exposure near hazardous waste sites is likely to occur by breathing contaminated air, having contact with contaminated soil, or drinking contaminated water.

The background or natural level of mercury found in outdoor air is generally between 10 and 20 nanograms of mercury per cubic meter of air (ng/m^3). Mercury levels found in surface water are generally less than 5 ng per liter of water. Levels normally found in soil range from 20 to 625 ng of mercury per gram of soil. The Food and Drug Administration (FDA) has estimated that, on average, most people are exposed to about 50 ng of mercury per kilogram of body weight per day in the food they eat. This translates to about 3.5 micrograms of mercury per day for an average weight adult. A large proportion of this mercury is likely to come from fish. Furthermore, people who eat a lot of fish are likely to have higher exposure to mercury.

Exposure to mercury can occur in many jobs. Most exposures on the job occur as a result of breathing air that contains mercury. Exposure occurs in the medical, dental, and other health services, and in the chemical, metal processing, electrical equipment, automotive, building, and other industries. Families of workers may be exposed to mercury in the home if the workers have mercury dust on their clothing. Dentists and their assistants may also be exposed to mercury from skin contact with dental fillings and breathing metallic mercury vapor released from these fillings.

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Exposure to mercury can be determined by measuring amounts in blood and urine. Levels found in blood and urine may show whether health effects are expected (see Section 2.5). Refer to Chapter 5 for more information on how you might be exposed to mercury.

1.4 HOW CAN MERCURY ENTER AND LEAVE MY BODY?

Mercury can easily enter your body when you breathe in air containing metallic mercury. Most of the mercury that gets into your lungs as metallic mercury goes rapidly to other parts of the body. Metallic mercury that you might swallow does not enter your bloodstream very easily, and most of it leaves the body in the feces. Some metallic mercury may stay in your body, mostly in the kidney and brain. Metallic mercury can also reach the fetuses of pregnant women easily. Metallic mercury that you breathe in will leave your body in the urine, feces, and breath.

Inorganic salts of mercury (mercurous or mercuric chloride, for example) that are inhaled do not enter your body as easily. However, these inorganic forms of mercury, if swallowed, enter the body more easily than metallic mercury. Inorganic mercury can also enter the bloodstream directly through the skin. However, only a small amount would pass through your skin compared with breathing or swallowing inorganic mercury. After entering the body, inorganic compounds of mercury can also reach many tissues. Some may stay in your body, mostly in the kidneys. However, inorganic mercury cannot reach the brain as easily as metallic mercury. Inorganic mercury leaves your body in the urine or feces after several weeks or months.

Organic compounds of mercury can probably enter your body easily through the lungs. Organic mercury in contaminated fish or other foods that you might eat enters your bloodstream easily and goes rapidly to other parts of your body. It can also enter the bloodstream directly through the skin, but only a small amount would pass through your skin. Organic mercury in the body is similar to metallic mercury because it can reach most tissues including the brain and fetus. Organic mercury can change to inorganic mercury in the brain and remain there for a long time. Organic mercury that you swallow or breathe leaves your body in the feces, mostly as inorganic mercury, within weeks.

For more information on how mercury can enter and leave your body, please refer to Chapter 2.

1.5 HOW CAN MERCURY AFFECT MY HEALTH?

Long-term exposure to either inorganic or organic mercury can permanently damage the brain, kidneys, and developing fetus. The most sensitive target of low-level exposure to metallic and organic mercury following short- or long-term exposures appears to be the nervous system. The most sensitive target of low-level exposure to inorganic mercury appears to be the kidneys. Short-term exposure to high levels of mercury can have

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similar effects. Full recovery is more likely after short-term exposures than long-term exposures, once the body clears itself of the contamination.

Short-term exposure to high levels of metallic mercury in the air can cause skin rashes and effects on the lungs and eyes. Long-term exposure to metallic mercury has been studied in workers at chlorine facilities. Some of them developed symptoms such as memory loss and shakiness. Levels of metallic mercury in air were greater than the levels normally encountered by the general population. Current levels of mercury in workplace air are lower than in the past. Because of this reduction, fewer workers have symptoms from mercury exposure. Studies in humans found there were no effects on the ability to reproduce after breathing metallic mercury for a long time.

Short- and long-term exposure to high levels of inorganic mercury can cause kidney effects in humans. However, recovery is likely, once the body clears itself of the contamination. There is no information on low-level exposures in humans. Short- and long-term exposure to low levels of inorganic mercury in animals can also cause kidney and brain effects. Long-term exposure to higher than normal levels of inorganic mercury from eating or drinking contaminated foods or water can lead to brain and kidney damage in some people. Long-term exposure to inorganic mercury has caused effects to the fetus in animals. The general population is generally not exposed to levels high enough to produce these effects.

People who eat fish containing organic mercury or grains treated with organic mercury for a long time can have permanent damage to the brain, kidneys, and the growing fetus. The amounts of organic mercury that cause these effects are higher than the amounts to which the general population is exposed daily. Exposure to organic mercury may cause brain damage in developing fetus. Exposure to organic mercury is also dangerous for young children because their nervous systems are more sensitive to these compounds. Kidney effects occur in animals exposed to low levels of organic mercury. Low-level exposure to organic mercury may also reduce the ability of animals to have babies. However, no studies are available to determine if this effect can occur in humans.

There is no information to show that mercury causes cancer in humans or animals. National Toxicology Program (NTP), EPA, and the International Agency for Research on Cancer (IARC) have not classified mercury as to its human carcinogenicity. Chapter 2 contains information on health effects of mercury in humans and animals.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO MERCURY?

There are reliable, accurate, and easily available ways to measure mercury levels in the body. However, the tests do not determine the form of mercury to which you might have been exposed. Blood or urine samples can be taken in a doctor's office and tested using special equipment in a laboratory. Levels found in blood and urine may be used to predict possible health effects. This test is useful during and after short- and long-term

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exposures. Several months after exposure ends, mercury levels in the blood and urine are much lower. Short-term exposure to mercury can also be evaluated by measuring mercury in the breath but only within a few days after exposure. For more information on testing for mercury levels in the body, see Chapters 2 and 6.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The government has developed regulations and guidelines for mercury. EPA has established many regulations to control air pollution. These are designed to protect the public from the possible harmful health effects of mercury.

EPA and the FDA have set a limit of 2 parts mercury per billion (ppb) parts of water in drinking water. EPA also recommends that the level of inorganic mercury in rivers, lakes, and streams should be no more than 144 parts mercury per trillion (ppt) parts of water to protect human health. EPA suggests that a daily exposure to 2 ppb of mercury in drinking water for an adult of average weight is not likely to cause any significant adverse health effects.

The Occupational Safety and Health Administration (OSHA) regulates levels in the workplace. It has set a limit of 1.2 ppb for organic mercury and 6.1 ppb for inorganic mercury in the workplace air to protect workers during an 8-hour shift and a 40-hour work week. The National Institute for Occupational Safety and Health (NIOSH) recommends that the amount of inorganic mercury in workplace air be limited to 6.1 ppb averaged over a 10-hour work shift.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333
(404) 639-6000

This agency can also provide you with information on the location of occupational and environmental health clinics. These clinics specialize in the recognition, evaluation, and treatment of illness resulting from exposure to hazardous substances.

**POLYCHLORINATED BIPHENYLS
(PCBs)**

Source: ATSDR, 1991. "Draft Toxicological Profile for PCBs." Agency for Toxic Substances and Disease Registry, Atlanta, GA. October, 1991.

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This Statement was prepared to give you information about polychlorinated biphenyls (PCBs) and to emphasize the human health effects that may result from exposure to them. The Environmental Protection Agency (EPA) has identified 1,300 sites on its National Priorities List (NPL). PCBs have been found in at least 286 of these sites. However, we do not know how many of the 1,300 NPL sites have been evaluated for PCBs. As EPA evaluates more sites, the number of sites at which PCBs are found may change. This information is important for you to know because PCBs may cause harmful health effects and because these sites are potential or actual sources of human exposure to PCBs.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, they enter the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to them in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to hazardous chemicals such as PCBs, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT ARE POLYCHLORINATED BIPHENYLS?

PCBs are a family of man-made chemicals that contain 209 individual compounds (known as congeners) with varying harmful effects. There are no known natural sources of PCBs in the environment. PCBs are either oily liquids or solids and are colorless to light yellow in color. They have no known smell or taste. PCBs enter the environment as mixtures containing a variety of individual components and impurities. Because the health effects of PCBs are difficult to evaluate, this document contains information on mainly seven types of commercially available PCB mixtures. These seven types of PCB mixtures include 35% of all of the different PCBs and 98% of PCBs sold in the United States since 1970. Some commercial PCB mixtures are known in the United States by their industrial trade name, Aroclor. Because they don't burn easily and are a good insulating material, PCBs have been used widely as coolants and lubricants in transformers, capacitors, and other electrical equipment. The manufacture of PCBs stopped in the United States in October 1977 because of evidence that PCBs build up in the environment and cause harmful effects. Consumer products that may contain PCBs are

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old fluorescent lighting fixtures and electrical devices or appliances containing PCB capacitors made before PCB use was stopped.

You will find further information on the physical properties and uses of PCBs in Chapters 3 and 4 of this profile.

1.2 WHAT HAPPENS TO POLYCHLORINATED BIPHENYLS WHEN THEY ENTER THE ENVIRONMENT?

In the past, PCBs entered the air, water, and soil during their manufacture and use. PCB-containing wastes generated during manufacture and use of PCBs were placed in dumpsites. PCBs also entered the environment from accidental spills and leaks, during the transport of the chemicals, or from leaks in transformers and capacitors or other products containing PCBs. Today, PCBs can be released into the environment from poorly maintained hazardous waste sites that contain PCBs; illegal or improper dumping of PCB wastes, such as transformer fluids; leaks or releases from electrical transformers containing PCBs; and disposal of PCB-containing consumer products into municipal landfills rather than into secured landfills.

PCBs in air can be present as both airborne solid and liquid particles and vapor that eventually return to the land and water by settling, snow, and rainwater. They may remain in the air for an average of more than 10 days depending on the type of PCB. Once in the air, PCBs can be carried long distances. They have been found in snow and seawater in areas far away from where they were released into the environment. In water, a small amount of the PCBs may remain dissolved but most tend to stick to particles and sediments. PCBs in water partially evaporate and then return to earth by rainfall, snow, or settling of dust particles. This cycle can be repeated many times. PCBs in water concentrate (build up) in fish and can reach levels hundreds of thousand times higher than the levels in water. Extremely small amounts of PCBs can remain in water for years. They bind strongly to soil and may remain there for several years. Although PCBs will not typically travel too deep into the soil with rainwater, PCBs from some waste landfills have been found in groundwater. PCBs will partially evaporate from soil surfaces to air. In general, the breakdown of PCBs in the water and soil occurs over several years. Sediments at the bottom of a body water like a lake, river, or ocean generally act as a reservoir from which PCBs may be released slowly over a long period of time to the water. More information about the fate and movement of PCBs in the environment can be found in Chapter 5.

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1.3 HOW MIGHT I BE EXPOSED TO POLYCHLORINATED BIPHENYLS?

Although PCBs are no longer made, exposure can still happen. Many older transformers and capacitors still contain PCBs. These transformers can be used for 30 years or more. Old fluorescent lighting fixtures and electrical devices and appliances made before PCB use was stopped may contain PCBs. When these electric devices get hot during operation, small amounts of PCBs may leak into the air and indoor air.

The two main sources of exposure to PCBs are from the environment and from the workplace. PCBs are found throughout the environment and remain there a very long time. Small amounts of PCBs can be found in almost all outdoor air, in indoor air, on soil surfaces, and in surface water. PCBs enter the bodies of fish from water, sediment, particulates in water, and from eating prey that have PCBs in their bodies. The typical concentrations of PCBs are 5 to 10 nanograms per cubic meter of air (5 to 10 billionths of a gram in a cubic meter of air [ng/m^3]) in urban areas and $0.8 \text{ ng}/\text{m}^3$ in rural areas. The PCB concentrations in indoor air of seven public buildings (schools and offices) were 230 to $460 \text{ ng}/\text{m}^3$. The mean concentration of PCBs in waters of the Great Lakes is 0.5 to 17 nanograms per liter (0.5 to 17 billionths of a gram in a liter of water [ng/L]). Thirty-two of 163 well waters in industrial areas of New Jersey contained 60 to $1,270 \text{ ng}/\text{L}$ of PCBs. Typical concentrations of PCBs in soil are less than 10 to 40 micrograms per kilogram (less than 10 to 40 millionths of a gram in one kilogram of soil [$\mu\text{g}/\text{kg}$]). Average PCB concentrations as high as $4,331,000 \mu\text{g}/\text{kg}$ have been found in soil from a hazardous waste site. The mean concentration of PCBs in whole freshwater fish is $0.5 \mu\text{g}/\text{g}$ (0.5 millionths of a gram in 1 gram of fish). The concentrations of PCBs in air, water, soil, and food have generally decreased since PCB production stopped in 1977. Although PCBs are usually found at higher levels in the parts of fish that you do not eat, the amount of PCBs found in the parts of fish that you do eat is high enough to make eating fish a major source of exposure. Breathing indoor air in buildings that have electrical parts that contain PCBs may also be a major source of human exposure. You may be exposed to several micrograms of PCBs per day from air, water, and food.

People who live near hazardous waste sites that contain PCBs may be exposed primarily by breathing air that contains PCBs. Children playing at or near these sites may be exposed by touching and eating soil that contains PCBs. The most likely way infants will be exposed is from breast milk that contains PCBs.

Workplace exposure to PCBs can occur during repair and maintenance of PCB transformers, accidents or spills involving PCB transformers, and disposal of PCB materials. Contact with PCBs at hazardous waste sites can happen when workers breathe

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air and touch soil containing PCBs. Exposure in the workplace occurs mostly by breathing air containing PCBs and by touching substances that contain PCBs. Fewer than 2,500 people were thought to be exposed to PCBs in the workplace during 1981 to 1983. Information about PCB exposure can be found in Chapter 5.

1.4 HOW CAN POLYCHLORINATED BIPHENYLS ENTER AND LEAVE MY BODY?

If you breathe air that contains PCBs they can enter your body through your lungs and pass into the bloodstream, but we do not know how fast or how much of the PCBs will pass into the blood stream. If you swallow food, water, or soil contaminated with PCBs, most of the PCBs will probably enter your body and pass from the stomach into the blood stream quickly (in minutes). If you touch soil containing PCBs, such as might occur at a hazardous waste site, some of the PCBs will pass through the skin into the blood stream, but we do not know how much or how fast. The most common way for PCBs to enter your body is by eating fish and shellfish that contain PCBs. Exposure from drinking water than from food. For people living around waste sites or processing or storage facilities, and for people who work with or around PCBs, the most likely way PCBs will enter their bodies are from skin contact with contaminated soil and breathing PCB vapors. Once PCBs are in your body, some may change into other related chemicals called metabolites. Some metabolites of PCBs can be as harmful as unchanged PCBs. Some of the metabolites may leave your body in the feces in a few days, but others may stay in your body fat for months. Unchanged PCBs may also stay in your body and be stored for years in your body fat. PCBs build up in milk fat and can enter the bodies of infants through breast feeding. For more information on how PCBs can enter and leave your body, see Chapter 2.

1.5 HOW CAN POLYCHLORINATED BIPHENYLS AFFECT MY HEALTH?

Skin irritations, such as acne and rashes, can occur in workers exposed to PCBs. Studies suggest that exposure to PCBs in the workplace may also cause irritation of the nose and lungs or cancer in the liver and other organs. The Department of Health and Human Services has determined that PCBs may reasonably be anticipated to be carcinogens. The amount of PCBs in the workplace are much higher than at other places, such as in air in buildings that have electrical parts that contain PCBs or in outdoor air, including air at hazardous waste sites. Young children of women who ate foods that contain high levels of PCBs, such as fish, before and during their pregnancy might have some trouble learning. We do not know the possible effects in persons who are exposed to high levels of PCBs for a short period.

Rats that ate food containing large amounts of PCBs for a short period had mild liver damage, and some died. Animals that ate smaller amounts of PCBs in their food over

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several weeks or months had many serious health effects, including liver, stomach, and thyroid gland injuries; anemia; acne; and damaged reproduction. These effects have been seen in many different kinds of animals, including monkeys, as well as in the offspring of animals that ate PCBs. No birth defects have been found. Rats and mice that ate certain PCB mixtures throughout their lives developed cancer in their livers and other organs in their bodies. Only a small amount of information exists on the health effects in animals exposed to PCBs by skin contact or breathing. This information indicates that liver, kidney, and skin damage occurred in rabbits following repeated skin exposure, and that a single exposure to a large amount of PCBs on the skin caused death in rabbits and mice. Breathing PCBs over several months also caused liver and kidney damage in rats and other animals, but the levels necessary to produce these effects were very high. It is not known if the same effects would happen in people if they were exposed in the same way. For more information on how PCBs can affect your health, see Chapter 2.

The Agency for Toxic Substances and Disease Registry has calculated Environmental Media Evaluation Guides (EMEGs) for PCBs. The EMEGs are derived from a Minimal Risk Level (MRL), which is calculated from animal data for PCBs. The MRL is further described in Chapter 2 and in the footnotes to Table 2-2. If a person is exposed to PCBs at a level below the EMEG for the period listed below, we do not expect harmful health effects to occur. Because these levels are based only on information currently available, some uncertainty is always associated with them. Also, an EMEG does not imply anything about the presence, absence or level risk for cancer because the methods for deriving EMEGs do not use any information about cancer. The EMEGs are provided as concentrations of parts of PCBs per million parts of drinking water or soil (ppm) in order to allow for comparison to levels people might encounter in drinking water and soil around homes or in other areas where children may play.

Drinking water exposure

Drinking water EMEGs represent the lower end of a range and are protective for both children and adults.

- A drinking water EMEG of 0.00005 ppm for PCBs was derived from animal data for exposures of 1 year or more.

Soil exposure

Soil EMEGs represent the lower end of a range and are protective for both children and adults. However, this range is not protective for children (pica) who show increased desire for eating non-food items (such as soil).

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- A soil EMEG of 0.25 ppm for PCBs was derived from animal data for exposures of 1 year or more.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO POLYCHLORINATED BIPHENYLS?

There are tests to find out if PCBs are in the blood, body fat, and breast milk. These tests are not routine clinical tests, but they can detect PCBs in people exposed to them in the environment and at work. High levels of PCBs in these fluids will show that you have been exposed to high levels of PCBs. However, these measurements cannot determine the exact amount or type of PCBs you have been exposed to or for how long you have been exposed. Although these tests can indicate whether you have been exposed to PCBs, they do not predict whether you will develop harmful health effects. Blood tests are the easiest, safest, and possibly the best method for detecting recent exposures to large amounts of PCBs. Nearly everyone has been exposed to PCBs because they are found throughout the environment, and nearly all persons are likely to have amounts of PCBs in their blood, fat, and breast milk that can be measured. For more information on tests to determine whether you have been exposed to PCBs, see Chapters 2 and 6.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

For the maximum protection of human health from the possible cancer effects of drinking water or eating fish or shellfish that contain PCBs over a lifetime, the EPA recommends that the level of PCBs in lakes and streams not be more than 0.001 parts of PCBs per billion parts of water (0.001 ppb). For exposure through drinking water, EPA suggests that at 0.004 milligrams of PCBs per liter of water (0.004 mg/L) for adults, and 0.001 mg/L of water for children, noncancer harmful health effects would not be expected.

The Food and Drug Administration (FDA) says that infant foods, eggs, milk, and poultry fat should not contain more than 0.2 to 3 parts of PCBs per million parts of food (0.2 to 3 ppm) to protect infants from noncancer harmful health effects.

The National Institute for Occupational Safety and Health (NIOSH) recommends that workers not breathe air containing more than 0.001 milligrams of PCBs per cubic meter of air (0.001 mg/m³) for a 10-hour workday, 40-hour workweek. The Occupational Safety and Health Administration (OSHA) set the workplace exposure limits at 0.5 mg/m³ to 1 mg/m³ for different types of PCBs for an 8-hour workday to protect workers from noncancer harmful health effects.

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The EPA requires that companies that transport, store, or dispose of PCBs follow the rules and regulations of a federal hazardous waste management program. The EPA has also suggested standards that limit the amount of PCBs put into publicly owned waste water treatment plants. To minimize exposure to PCBs by humans, EPA says that industry must tell the National Response Center when 10 pounds or more of PCBs have been released to the environment.

For more information on federal and state recommendations, see Chapter 7.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

This agency can also provide you with information on the location of the nearest occupational and environmental health clinic. These clinics specialize in the recognition, evaluation, and treatment of illnesses resulting from exposure to hazardous substances.

**POLYCYCLIC AROMATIC HYDROCARBONS
(PAHs)**

Source: ATSDR, 1990. "Toxicological Profile for Polycyclic Aromatic Hydrocarbons."
Agency for Toxic Substances and Disease Registry, Atlanta, GA. December,
1990.

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This Statement was prepared to give you information about polycyclic aromatic hydrocarbons (PAHs) and to emphasize the human health effects that may result from exposure to them. The Environmental Protection Agency (EPA) has identified 1,177 sites on its National Priorities List (NPL). At this time, all of the polycyclic aromatic hydrocarbons discussed in this profile have been found at several of these NPL hazardous waste sites.

Dibenz(a,h)anthracene has been found at the lowest number of sites (6) and benzo(a)pyrene has been found at the highest number of sites (84). However, we do not know how many of the 1,177 NPL sites have been evaluated for PAHs. As EPA evaluates more sites, the number of sites at which PAHs are found may change. The information is important for you because PAHs may cause harmful health effects and because these sites are potential or actual sources of human exposure to PAHs.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to hazardous substances such as PAHs, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT ARE POLYCYCLIC AROMATIC HYDROCARBONS?

PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances. PAHs can be man-made or occur naturally. There is no known use for most of these chemicals except for research purposes. A few of the PAHs are used in medicines and to make dyes, plastics, and pesticides. They are found throughout the environment in the air, water, and soil. There are over one hundred different PAH compounds. Although the health effects of the individual PAHs are not exactly alike, the following 15 PAHs are considered as a group in this profile:

- acenaphthene
- acenaphthylene
- anthracene

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- benz(a)anthracene
- benzo(a)pyrene
- benzo(b)fluoranthene
- benzo(ghi)perylene
- benzo(k)fluoranthene
- chrysene
- dibenz(a,h)anthracene
- fluoranthene
- fluorene
- indeno(1,2,3-cd)pyrene
- phenanthrene
- pyrene

As pure chemicals, PAHs generally exist as colorless, white, or pale yellow-green solids. Most PAHs do not occur alone in the environment (including those found at hazardous waste sites), rather they are found as mixtures of two or more PAHs. They can occur in the air either attached to dust particles, or in soil or sediment as solids. They can also be found in substances such as crude oil, coal, coal tar pitch, creosote, and road and roofing tar. Most PAHs do not dissolve easily in water, but some PAHs readily evaporate into the air. PAHs generally do not burn easily and they will last in the environment for months to years.

More information on the chemical and physical properties of PAHs can be found in Chapter 3 and on their occurrence and break down in the environment in Chapter 5.

1.2 HOW MIGHT I BE EXPOSED TO POLYCYCLIC AROMATIC HYDROCARBONS?

PAHs are present throughout the environment, and you may be exposed to these substances at home, while outside, or at the workplace. Typically, you will not be exposed to an individual PAH alone, but to a mixture of PAHs.

In the environment, you are exposed to PAH vapors or PAHs that are attached to dust and other particles in the air. Other sources include vehicle exhausts, asphalt roads, coal, coal tar, wild fires, agricultural burning, and hazardous waste sites. Background levels of PAHs in the air are reported to be 0.02-1.2 mg/m³ in rural areas and 0.15-19.3 mg/m³ in urban areas. You may be exposed to PAHs in soil near areas where coal, wood, gasoline, or other products have been burned. You may be exposed to PAHs in the soil on or near hazardous waste sites, such as former manufactured-gas sites and wood-preserving facilities. PAHs have been found in some drinking water supplies in the United States. The background level of PAHs in drinking water ranges from 4 to 24 nanograms per liter (ng/L).

In the home, PAHs are present in tobacco smoke, smoke from home heating of wood, creosote-treated wood products, cereals, grains, flour, bread, vegetables, fruits, meat, processed or pickled foods, and beverages. Food

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grown in contaminated soil or air may also contain PAHs. Cooking meat or other food at high temperatures, which happens during grilling or charring, increases the amount of PAHs in the food. The level of PAHs in the typical U.S. diet is less than 2 parts of total PAHs per billion parts of food (ppb).

For many people, the greatest exposure to PAHs occurs in the workplace. PAHs have been found in coal-tar production plants, coking plants, bitumen and asphalt production plants, coal-gasification sites, smoke houses, aluminum production plants, coal-tarring activities, and municipal trash incinerators. PAHs have also been found in other facilities where petroleum, petroleum products, or coal are used or where wood, cellulose, corn, or oil are burned. For more information on human exposure to PAHs see Chapter 5.

1.3 HOW CAN POLYCYCLIC AROMATIC HYDROCARBONS ENTER AND LEAVE MY BODY?

PAHs can enter your body through your lungs when you breathe air that contains them. This is one of the most significant route of exposure for people living near hazardous waste sites. Drinking water or swallowing food, soil, or dust particles that contain PAHs are other routes for these chemicals to enter your body. Under normal conditions of environmental exposure, PAHs could enter your body if your skin comes into contact with soil that contains high levels of PAHs (this could occur near a hazardous waste site); or with heavy oils or other products (such as coal tar, roofing tar, or creosote) that contain PAHs.

PAHs enter your body quickly and easily by all routes of exposure. The rate at which PAHs enter your body is increased when they are present in oily mixtures. They go to all the tissues of your body that contain fat, and tend to be stored mostly in your kidneys, liver, and fat, with smaller amounts in your spleen, adrenal glands, and ovaries. Results from animal studies show that PAHs do not tend to be stored in your body for a long time; most PAHs that enter the body leave within a few days, primarily in the feces and urine. More information on how PAHs enter and leave your body can be found in Chapters 2 and 6.

1.4 HOW CAN POLYCYCLIC AROMATIC HYDROCARBONS AFFECT MY HEALTH?

PAHs can cause harm to your health. Several of the PAHs, including benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene, have caused tumors in laboratory animals when they ate them, when they were applied to their skin, or when they breathed them in the air for long periods of time. Reports in humans show that individuals exposed by breathing or skin contact for long periods of time to mixtures of other compounds and PAHs can also develop cancer.

Mice fed high levels of benzo(a)pyrene during pregnancy had difficulty reproducing and so did their offspring. The offspring from pregnant mice fed

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benzo(a)pyrene also showed other harmful effects, such as birth defects and decreased body weight. Similar effects could occur in humans, but we have no information to show that these effects do occur.

Studies in animals have also shown that PAHs can cause harmful effects on skin, body fluids, and the body's system for fighting disease after both short- and long-term exposure. These effects have not been reported in humans. More information on the health effects associated with exposure to PAHs can be found in Chapter 2.

1.5 WHAT LEVELS OF EXPOSURE HAVE RESULTED IN HARMFUL HEALTH EFFECTS?

Tables 1-1 through 1-4 show the relationship between exposure to PAHs and known health effects in humans and animals. As can be seen, no information is available from human studies to determine health effects resulting from exposure to specific levels of the individual PAHs, although inhalation and skin exposure to mixtures containing PAHs has been associated with cancer in humans.

A Minimal Risk Level (MRL) is also included in Table 1-3. This MRL was derived from animal data for short-term exposure, as described in Chapter 2 and in Table 2-2. The MRL provides a basis for comparison with levels that people might encounter in food. If a person is exposed to PAHs at an amount below the MRL, it is not expected that harmful (noncancer) health effects will occur. Because this level is based only on information currently available, some uncertainty is always associated with it. Also, because the method for deriving MRLs does not use any information about cancer, an MRL does not imply anything about the presence, absence, or level of risk for cancer.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO POLYCYCLIC AROMATIC HYDROCARBONS?

In your body, PAHs are changed into chemicals that can attach to substances within the body. The presence of PAHs attached to these substances can then be measured in body tissues or blood after exposure to PAHs. However, this test is still being developed and it is not known yet how well it works. PAHs or their breakdown products can also be measured in urine. Although these tests can tell that you have been exposed to PAHs, it is not yet possible to use these tests to predict the severity of any health effects that might occur or to determine the extent of your exposure to the PAHs. These tests are not routinely available at a doctor's office because they require special equipment for sampling and detecting these chemicals. More information on tests used to determine PAHs in your body is presented in Chapters 2 and 6.

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TABLE 1-1. Human Health Effects from Breathing PAHs*

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of humans to air containing specific levels of PAHs are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Air (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term exposure of humans to air containing specific levels of PAHs are not known.

*See Section 1.2 for a discussion of exposures encountered in daily life.

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TABLE 1-2. Animal Health Effects from Breathing PAHs

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term exposure of animals to air containing specific levels of PAHs are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Air (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
		The health effects resulting from long-term exposure of animals to air containing specific levels of PAHs are not known.

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TABLE 1-3. Human Health Effects from Eating or Drinking PAHs*

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
3.6		Minimal Risk Level (based on animal studies; see Section 1.5 for discussion).
<u>Levels in Water (ppm)</u>		The health effects resulting from short-term exposure of humans to water containing specific levels of PAHs are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Food (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term exposure of humans to food containing specific levels of PAHs are not known.
<u>Levels in Water (ppm)</u>		The health effects resulting from long-term exposure of humans to water containing specific levels of PAHs are not known.

*See Section 1.2 for a discussion of exposures encountered in daily life.

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TABLE 1-4. Animal Health Effects from Eating or Drinking PAHs

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
308	10 days	Birth defects caused by benzo(a)pyrene in mice.
<u>Levels in Water (ppm)</u>		The health effects resulting from short-term exposure of animals to water containing specific levels of PAHs are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Food (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
923	6 months	Adverse effects on the liver and blood in mice caused by benzo(a)pyrene.
<u>Levels in Water (ppm)</u>		The health effects resulting from long-term exposure of animals to water containing specific levels of PAHs are not known.

*These effects are listed at the level at which they were first observed. They may also be seen at higher levels.

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1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

Based on data on benzo(a)pyrene, the federal government has developed regulatory standards and guidelines to protect individuals from the potential health effects of PAHs in drinking water. The U.S. Environmental Protection Agency (EPA) has provided estimates of levels of total cancer-causing PAHs in lakes and streams associated with various risks of developing cancer in humans. EPA has also concluded that any release of PAHs of more than 1 pound should be reported.

PAHs are generally not produced commercially in the United States except as research chemicals. However, PAHs are found in coal, coal tar, and in the creosote oils and pitches formed from the distillation of coal tars. The National Institute for Occupational Safety and Health (NIOSH) concluded that occupational exposure to coal products can increase the risk of lung and skin cancer in workers and recommended an occupational exposure limit for coal tar products of 0.1 milligram of PAHs per cubic meter of air (0.1 mg/m^3) for a 10-hour workday, 40-hour work week. The Occupational Safety and Health Administration (OSHA) has established a legally enforceable limit of 0.2 milligrams of all PAHs per cubic meter of air (0.2 mg/m^3).

More information on regulations and advisories for PAHs exposure can be found in Chapter 7.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns not covered here, please contact your State Health or Environmental Department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road, E-29
Atlanta, Georgia 30333

This agency can also give you information on the location of the nearest occupational and environmental health clinics. Such clinics specialize in recognizing, evaluating, and treating illnesses that result from exposure to hazardous substances.

RADIONUCLIDES

Source: NCRP, 1980. Report No. 65: "Management of Persons Accidentally Contaminated with Radionuclides." National Council on Radiation Protection and Measurements. Bethesda, MD.

6. Resumé of Experience With Important Radionuclides

6.1 Americium

Americium, (Am), element number 95, is a member of the group of transuranic elements, and has isotopes of mass 237 to 246. It does not occur in nature. The two most important isotopes are americium-241 and americium-243. Americium-241 is a daughter product of plutonium-241 and therefore it is associated frequently with plutonium processing or handling. Americium-243 is produced from uranium-238 or plutonium-239 by multiple neutron capture.

Americium-241 has a physical half-life of 458 years and an effective half-life in bone of about 140 years. The assumed effective half-life in the whole body is 100 years and in liver 40 years (ICRP, 1972). Americium-241 decays by emitting alpha particles of 2 distinct energies [5.49 MeV (85 percent) and 5.44 MeV (13 percent)] to form neptunium-237. The principal photons emitted by ^{241}Am are gamma rays of 60 (36 percent) and 26 keV, and conversion L x rays of neptunium with energies centered at about 18 keV.

Americium-243 has a physical half-life of 7950 years and assumed effective half-life in bone of 195 years, in whole body of 100 years, and in liver of 40 years. Americium-243 decays by emitting alpha particles of 2 energies [5.28 MeV (87 percent) and 5.23 MeV (11 percent)], and two soft gamma rays (44 and 75 keV).

Americium exhibits all oxidation states from II to VII, but the trivalent state is the most common. The metal oxidizes slowly in air and dissolves readily in dilute HCl.

Reactor-grade plutonium contains a few percent of americium-241, depending on the age of the material and its radiation history in the reactor. Americium-241 is used as a radiation source for static eliminators, smoke detectors, thickness gauges, and calibration sources. Combined with beryllium, americium is used as a neutron source. It is also used as target material for producing ^{242}Cm in accelerators.

Depositions of americium in the body occur primarily by inhalation of particulates or through skin wounds. Absorption through the gas-

trointestinal tract is only about 0.03 percent in adult animals, although it is probably higher in newborns and the very young (Durbin, 1973). Absorption through intact skin is thought to be very small, but probably, as is the case with plutonium, is increased when it is dissolved in solutions having an acidity that destroys the integrity of the skin barrier. Skin absorption of various solutions of plutonium in man and animals has been observed to range from 0.002 to 0.25 percent for exposures of up to one day duration (Vaughan *et al.*, 1973). Similar studies have not been reported for americium.

Absorption through wounds will depend on the chemical form and volume of the material and probably the nature of the wound. Intramuscular injections of americium in rats resulted in the following absorption (redistribution) from the injection site in the first day: $^{241}\text{Am}(\text{NO}_3)_3$ —10 percent, $^{241}\text{Am}_2(\text{SO}_4)_3$ —24 percent, and $^{241}\text{AmCl}_3$ —58 percent. Rapid uptake continued to occur during the first few days (Durbin, 1973).

Inhalation of particles is an important internal exposure route in industry. Uptake depends markedly on the chemical and physical properties of the particulates (see Section 3.2). Studies in rats indicate 75 to 85 percent of the initial lung burden of americium compounds is absorbed into the body, about 10 percent is retained in the lung, and less than 15 percent is cleared from the lung and excreted in the feces. These percentages do not include the 50 to 90 percent of inhaled material that is promptly cleared from the lung (Durbin, 1973) and eliminated via the gastrointestinal tract.

Once absorbed, americium is deposited primarily in liver and skeleton with lesser amounts initially present in kidney and spleen. In most animal studies, 80 and 90 percent of parenterally administered ^{241}Am is partitioned initially with 20 to 35 percent in the skeleton and 50 to 70 percent in the liver. In rats, after deposition of ^{241}Am in the respiratory system and absorption into the circulation, approximately 35 percent was transported to the liver and 57 percent to the remaining carcass (Crawley and Goddard, 1976). The total activity transported to extrapulmonary tissue was greater after its administration as the citrate than as the nitrate. The skeleton is the probable critical organ for long-term effects due to the longer effective half-life in bone compared to other organs. The liver is also an organ of concern.

The pathology of ^{241}Am exposure by intraperitoneal injection in mice has been described (Nilsson and Broomé-Karlsson, 1976). Very high doses of 16 and 18 $\mu\text{Ci}/\text{kg}$ seriously damaged the hematopoietic tissues, bone tissue, and testes. The highest frequency of induced tumors, 27 percent in the skeleton and 10 percent in hematopoietic

tissue, was in the $8 \mu\text{Ci}/\text{kg}$ dose range. Liver tumors were not increased significantly in any group. Degenerative lesions of the liver, adrenal glands, kidney, and heart were found mainly in the higher dose groups.

Metabolism and dosimetry studies of inhaled ^{241}Am oxide in beagles (4.4 to $4.9 \mu\text{Ci}/\text{kg}$) showed that the greatest long-term doses are received by the tracheobronchial lymph nodes, liver, lung, bone, and thyroid in descending order (Thomas *et al.*, 1972). The total white cells, platelets, lymphocytes, and neutrophils of the blood were reduced, in number. Pathologic findings centered around fibrotic changes in lung and lymph nodes, fatty deposits, and cellular degeneration in liver, bone marrow depletion, glomerulosclerosis, and severely damaged thyroid. These dose levels, about 8,000 times the maximum permissible body burdens for workers, for time periods of 127 to 1022 days, do not necessarily indicate where long-term pathology, such as carcinogenesis, may develop.

In man, the mode of exposure determines the distribution in the body. In six persons chronically exposed to an unknown chemical form of americium by inhalation over a period of six years, most of the activity was in the skeleton with little activity in the soft tissues, except for the liver (Wrenn *et al.*, 1972). The ratios of activity found in the liver relative to the skeleton for a male adult and a 10-year old child were 0.1 and 0.3, respectively. In another instance, two men were studied for a period of nearly 4 years after accidental inhalation of americium oxide (Fry, 1976). At day 324, an estimated 41 percent was in lung, 47 percent in liver, and 12 percent in bone; at day 1392 the percentages were 18 in lung, 47 in liver, and 35 in bone. The long-term transfer from lung to blood, considered to be relatively small by the Task Group on Lung Dynamics (ICRP, 1966; 1972), appears to be a more important factor in lung clearance than ciliary mechanisms. It has been proposed (ICRP, 1972) that, for all americium compounds, the distribution be considered 45 percent in bone, 45 percent in liver, and 10 percent in other tissues or excreta.

Treatment for internal deposition of americium, regardless of the route of exposure, is the immediate administration of DTPA (see Section 7.3.5.3). If this agent is not immediately available, the use of EDTA (Section 7.3.5.2) can be substituted although it is less effective. In the event of a contaminated puncture wound, the local area should be excised promptly since the absorption rate of americium can be rapid (over 50 percent in the first day). CaDTPA (or CaEDTA) should be administered prior to surgical excision if possible. The effectiveness of DTPA has been documented by a number of investigators (Nenot *et al.*, 1972; Volf and Seidel, 1974; Lloyd *et al.*, 1975a; 1975b; Seidel, 1975; 1976; Cohen *et al.*, 1976). Dogs given ^{241}Am citrate intravenously

($0.3 \mu\text{Ci}/\text{kg}$) and treated 2 weeks later with daily subcutaneous injections of ZnDTPA had a liver content of ^{241}Am that was reduced after 203 days of treatment by a factor of 200 and a nonliver content reduced by a factor of 3, on the average, compared to untreated controls. After 387 days of treatment the nonliver content of ^{241}Am was about a factor of 10 less than in the untreated animals. The daily dose of ZnDTPA was equivalent to approximately 1 g DTPA daily in a 70-kg man (Lloyd *et al.*, 1975a). Daily injections of ZnDTPA also hastened the disappearance of ^{241}Am from a simulated wound site, prevented almost completely the translocation from the depot to the liver and skeleton, and reduced significantly the total body ^{241}Am content through increased excretion (Lloyd *et al.*, 1975b). Nenot *et al.* (1972) started treatment of rats 21 days after exposure to ^{241}Am nitrate by aerosol. CaDTPA, given twice a week intramuscularly ($50 \text{ mg}/\text{kg}$ body weight), had reduced the ^{241}Am content in the bone by a factor of 5.3 by day 100.

Seidel (1975, 1976) showed with rats that treatment of ^{241}Am exposure with DTPA is less effective if begun on day 4 than if started immediately after exposure. CaDTPA was clearly superior to ZnDTPA for the first dose given a few hours, or at most, one to two days after exposure, but otherwise ZnDTPA and CaDTPA were equally effective in enhancing the elimination of ^{241}Am .

Brodsky *et al.* (1968) treated a glovebox operator who had inhaled dust containing a mixture of ^{241}Am and ^{239}Pu oxides after a dry box explosion. One gram per day of CaDTPA given intravenously on days 5 through 8 increased the excretion of ^{241}Am in the urine to 50-100 times preinfusion levels. This marked increase indicates that CaDTPA can be effective even though the americium is in an oxide form, which was considered previously to be only slowly soluble in the lung.

Reasonable effectiveness of chelation therapy has been demonstrated in man even if started months or years after exposure to ^{241}Am . Fasiska *et al.* (1971) treated a person with a body burden of $1.8 \mu\text{Ci}$ ^{241}Am from exposures to oxides of americium that took place over a period of several years (see also Brodsky *et al.*, 1969). Chelation therapy, 1 g DTPA weekly over 30 months, removed about half the total body burden, mainly from liver and lung, but the bone component, about $1 \mu\text{Ci}$, was relatively unaffected. Continuation of DTPA administration at $0.5 \text{ g}/\text{week}$ appeared to have less effectiveness compared to the earlier regimen. No adverse side effects were observed in this long-term, low dose DTPA therapy (Slobodien *et al.*, 1973).

Cohen *et al.* (1976) have shown in both man and baboon that DTPA reduces the soft tissue and skeletal deposits of ^{241}Am much more effectively in the juvenile than in the adult. In baboons, the ^{241}Am

removed from the skeleton by DTPA is excreted primarily in urine, while increased ^{241}Am in the feces is from the liver.

The therapeutic effectiveness of chelation therapy was demonstrated in the case of a chemical operator, heavily contaminated with americium on his skin, including nitric acid burns, and contaminated wounds about the face and neck from flying debris (Heid *et al.*, 1979). Decontamination efforts on intact skin, wounds, and burns were extensive. During the first 935 days after the accident, a total of 548 g of ZnDTPA and 20 g of CaDTPA were administered intravenously. The dosages ranged from 1 g CaDTPA every 8 hours for several days in the first week after exposure to 1 g ZnDTPA daily until eleven months after exposure; then the drug was reduced to 3 times per week. No complications have been noted from this extensive course of therapy. It is estimated that the liver burden of ^{241}Am was reduced from 380 μCi to less than 0.2 μCi , and the bone burden from 380 μCi , estimated, to 25 μCi . Thus, the therapy is estimated to have been over 99 percent effective on the liver burden and over 90 percent effective on the bone burden. A total of 1100 μCi was excreted in urine and feces during the first 2 years following the accident.

6.2 Californium

Californium (Cf), element number 98, is a member of the actinide series. While there are thirteen isotopes with mass numbers from 242 to 254, the isotope ^{252}Cf is the most likely to be encountered and its most common valence is 3+. Californium-252, half-life 2.6 years, decays by emitting a 6.12 MeV alpha particle in 97 percent of decays, accompanied by gamma rays of 43, 100, or 160 keV. The property that makes ^{252}Cf especially interesting and useful is that it undergoes spontaneous fission with emission of 3.8 fast neutrons per fission; its fission half-life is 85.5 years.

Californium-252 is used primarily as a neutron source. One of the more attractive medical uses employs the neutron emission in the treatment of cancer. It is encapsulated and sealed in stainless steel and platinum and used in place of radium for interstitial or intracavitary applications (Seaborg, 1973; Wright, 1968). In industry, californium sources are used for neutron diffraction measurements, neutron radiography, neutron activation analyses, and as a neutron excitation source in nuclear reactors. It is also found in thickness gauges that use the alpha particle for measuring gas pressures and the thickness of very thin films. In geological prospecting, small ^{252}Cf sources are used in detecting gold, silver, and water in the soil.

Californium-252 presents serious external and internal radiological hazards. Inhalation and wounds are the most significant routes for accidental internal exposure. Absorption from the intestinal tract is negligible (Denham, 1969). Absorption of inhaled Cf compounds from the lung has not been studied experimentally, but Cf will probably be transported to the systemic circulation like other actinides, e.g., americium or curium. Uptake into the pulmonary lymph nodes is also of concern (Denham, 1969). Californium-252 in the systemic circulation is deposited rapidly in bone. In adult rats, over 60 percent of an intravenous or intramuscular injection of a citrate complex has been deposited in the skeleton within four days. In all species studied thus far, initial skeletal deposition of californium has been greater than that of americium or curium (Durbin, 1973). About 14 percent of the injected dose in rats was deposited initially in the liver, but by 90 days over 90 percent of the liver content was excreted via the bile into the intestine (Durbin *et al.*, 1973). Whole body retention, mainly in bone, lasts at least 11 years in the beagle; ^{252}Cf deposited in humans is estimated to have a biological half-time of about 175 years and an effective half-life of 2.2 years. The maximum permissible body burden is 0.01 μCi (0.000019 μg) and a permissible lung burden proposed to the ICRP is 0.004 μCi (Dolphin, 1973).

The excretion of californium in the urine (alpha counting) is used as a method of monitoring workers for possible internal exposures and is the principal measurement to use after accidents. *In vivo* counting is difficult to interpret because of the complex spectrum of gamma rays produced during fission. By use of special sensitive counters, 0.0003 to 0.005 μCi can be detected (Newton and Eagle, 1972).

There have been few reported ^{252}Cf exposures to humans. After inhalation of $^{252}\text{Cf}_2\text{O}_3$ particles by two workers, urinary clearance rate half-times of 10 to 12 days were observed after an initial clearance half-time of about one day (Poda and Hall, 1975). Treatment consisting of early aerosol DTPA chelation and saline catharsis was thought to have decreased deposition and enhanced clearance of the californium. Due to the paucity of human treatment data, the suggested therapy is based on animal experimentation.

Internal deposition of Cf as a result of an inhalation exposure or contaminated wounds should be treated immediately with CaDTPA (Section 7.3.5.3). Since ^{252}Cf , like other trivalent actinides, is transported rapidly from contaminated wounds into the systemic circulation (Morin *et al.*, 1974) and deposited in the skeleton, prompt treatment with CaDTPA is especially important (Durbin, 1973; Parker *et al.*, 1962). Wounds should be treated as described for plutonium in Section 7.2. Once bone deposition occurs, there is little prospect of removing

appreciable amounts with chelation therapy, although treatment started some days after exposure may help remove transuranics deposited in the liver, lung, and other soft tissues (Brodsky *et al.*, 1969; Fasiska *et al.*, 1971). DTPA treatment was still somewhat effective when started three weeks after ^{252}Cf injection (Morin *et al.*, 1974).

6.3 Cerium

The principal radioactive isotopes of cerium, element number 58, that are likely to be encountered are ^{144}Ce and ^{141}Ce . Cerium-144 is a fission product of uranium that emits beta rays (0.19, 0.24, and 0.32 MeV) and gamma rays (seven energies ranging from 0.034 to 0.133 MeV). Its physical half-life is 284 days. Cerium-141 is formed by exposing stable ^{140}Ce to neutron bombardment; it emits beta rays (0.44 and 0.58 MeV) and one 0.145 MeV gamma ray. The physical half-life of ^{141}Ce is 32 days. The physical, chemical, and biological properties of radiocerium have been compiled and evaluated recently by the NCRP in Report No. 60 (NCRP, 1978).

In the work place, radioactive cerium isotopes are most likely to be encountered as a component in mixed fission products. Exposure could occur around experiments in test reactors or at fuel reprocessing plants. Cerium in more purified forms may be used experimentally in chemical or biological laboratories. Its limited use as a separated isotope may account for the paucity of reports on cerium exposure in humans. Sill *et al.* (1969) described an exposure at a reactor facility where a mixture of cerium isotopes (36 μCi , ^{141}Ce ; 27 μCi , ^{144}Ce) and zirconium (13.5 μCi , ^{95}Zr - ^{95}Nb) was inhaled. A dose of about 10 rem was judged to have been given to the lower large intestine since practically all material was eliminated from the lung and gastrointestinal tract during the first four days. No activity was detected in the urine. Rundo (1965) followed the retention of radiocerium after an accidental inhalation of irradiated uranium particles. Six days after the accident, the subject's body burden was estimated to be 16.5 nCi of ^{141}Ce and 29 nCi of ^{144}Ce . The longer-lived ^{144}Ce appeared to have an effective half-life of about 280 days, nearly the same as the physical half-life.

Radiocerium is poorly absorbed from the intestinal tract in man and many species of animals. In mature rats the absorption of oral doses of radiocerium is less than 0.05 percent (Hamilton, 1947; Durbin *et al.*, 1956; Moskalev, 1959). Higher rates of absorption have been measured in young animals, such as weanling mice. Absorption of radioactive CeCl_3 from nasal membranes in Syrian hamsters was less than 4

percent (Cuddihy and Ozog, 1973). Studies in monkeys of the absorption of CeCl_3 after inhalation indicated that systemic absorption was always below 10 percent of the initial lung burden (Ducouso and Pasquier, 1974). The liver is the critical organ for the shorter-lived isotopes of cerium, whereas the bone is the critical organ for cerium-144, the radioisotope with the longest physical half-life (284 days). For material ingested or inhaled but not transported by the systemic circulation, the critical organs are the lower large intestine and the lung, respectively.

Treatment of radiocerium exposures should be started promptly by the use of CaDTPA or ZnDTPA (Section 7.3.5.3). Tombropoulos *et al.* (1969) were able to reduce the body burden in dogs after inhalation of $^{144}\text{CeO}_2$ by 90 percent within 30 days by use of CaDTPA compared to the 30 percent reduction in untreated controls. Treatment by aerosol or intramuscular injection (42-55 mg/kg body weight) was about equally effective. Effective DTPA therapy for a ^{144}Ce inhalation exposure in man was demonstrated by Glenn *et al.* (1979). The effectiveness of treatment depends markedly upon the promptness with which it is begun and the solubility of the cerium compound in the lungs. In mice, intraperitoneal injections of DTPA proved to be life saving in all animals given a lethal dose of 14.6 $\mu\text{Ci/g}$, of $^{144}\text{CeCl}_3$ by intraperitoneal injection (Il'in *et al.*, 1964).

6.4 Cesium

Cesium, (Cs), element number 55, is an alkali metal that has twenty-one radioactive isotopes. The two with the longest physical half-lives, ^{137}Cs (30 years) and ^{134}Cs (2.1 years), are the most likely to present contamination problems. Cesium-137 decays by emitting beta particles of two different energies, 0.51 MeV (95 percent) and 1.17 MeV (5 percent), and is accompanied by a 0.662 MeV gamma ray from its daughter product. Cesium-134 decays by emitting beta rays with six different energies ranging from 0.09 to 1.45 MeV [most abundant is 0.65 MeV (68.4 percent)] and gamma rays with seven different energies ranging from 0.48 to 1.37 MeV.

Cesium-137 is by far the more likely to be encountered because it is an important fission fragment produced during fissioning of either uranium or plutonium fuels. It has been the subject of many radiobiological and metabolic effects studies because it is one of the long-lived fission products found in the environment and in man as a result of worldwide fallout associated with atmospheric weapons tests. It is

used in industry as a sealed gamma source in thickness gauges, and in medicine and research as a sealed source for therapy and as a tracer substance.

Cesium and potassium have similar chemical and biochemical behavior, including distribution and metabolism in the body. Cesium is soluble in body fluids, is distributed more or less uniformly throughout the body, and is rapidly eliminated by the kidney. After ingestion, ^{137}Cs is absorbed rapidly and completely with about 10 percent being excreted within the first 2 days. The subsequent biological half-time, based on studies of contaminated cases occurring in industry or research laboratories, averages 109 days with a range from 68 to 165 days. These values are similar to those found on volunteer subjects after intravenous or oral intakes (Cohn *et al.*, 1963; Richmond *et al.*, 1962; Rosoff *et al.*, 1963; Van Dilla, 1965). The biological half-time is much shorter in children, ranging from 12 days in infants to 57 days in older children (Weng and Beckner, 1973; Lloyd, 1973); it is also shorter in women (84 ± 27 days) than in men (Lloyd *et al.*, 1966).

During the early 1960's, at least 19 human exposures to ^{137}Cs were reported in the literature (Hesp, 1964; Jeanmarie, 1964; Jordan *et al.*, 1964; Melandri and Rimondi, 1964; Miller, 1964; Taylor *et al.*, 1962). There were both inhalation and ingestion exposures, but most were of the order of 1 μCi or less; only one case exceeded the maximum permissible body burden of 30 μCi . These cases were not serious exposures and were followed primarily to study the metabolism and turnover rate of cesium and they were untreated. The number of reports of contaminated cases since 1965 has decreased, a trend that may indicate either better radiological protection procedures or a decreased interest in reporting minor contaminations. As a consequence of atmospheric weapons testing, there is a slowly decreasing level of ^{137}Cs in the environment and food that results in man now having a body burden of ~ 25 pCi/g of potassium, which delivers an annual radiation dose of about 0.5 mrad (NCRP, 1977a).

The most effective means for removing radioactive cesium in man is the oral administration of ferric cyanoferrate (II), commonly called Prussian blue, or Berlin blue, or ferric ferrocyanide (Madshus *et al.*, 1966; Madshus and Strömme, 1968; Strömme, 1968; Richmond, 1968; Stather, 1972; NCRP, 1977a). Although it is not available as an approved drug in the United States, it has been found to be relatively harmless and well tolerated by man (see Section 7.3.2.6). Other compounds related to Prussian blue, such as nickel ferrocyanide anion exchange resin, are also effective (Iinuma *et al.*, 1971) and without adverse reactions. Prussian blue is not absorbed from the intestine and it binds the cesium ions that are enterically cycled into the gastroin-

testinal tract so that the cesium is not reabsorbed. The biological half-time during such treatment is reduced to about one-third of its usual value and the body burden is likewise reduced. The effectiveness of the procedure is therefore dependent on the length of treatment and how soon after exposure it is started.

6.5 Cobalt

Cobalt, (Co), element number 27, has 10 radioactive isotopes, ^{54}Co to ^{64}Co . The radionuclides most likely to be encountered are ^{60}Co , ^{58}Co , and ^{57}Co . Cobalt-60 is the activation product produced by the bombardment of stable ^{59}Co by neutrons. Its half-life is 5.3 years and it decays by emitting a 0.31 MeV beta ray and gamma rays of two energies, 1.17 and 1.33 MeV. The other isotopes have shorter physical half-lives, ^{57}Co being 271 days, and ^{58}Co , 71 days. Both decay with the emission of penetrating gamma rays.

Cobalt-60 sealed sources are used in medical radiation therapy and industrial radiography. It is also used in industry for thickness gauges, calibration sources, and tracers. In biology, the radioactive cobalt isotopes have been used particularly for labeling vitamin B-12.

Small quantities of ^{60}Co and ^{58}Co have been detected in persons working around nuclear facilities, especially reactors, fuel-reprocessing plants, nuclear waste management operations, and laboratories using radioisotopes (Sill *et al.*, 1964; Edvardsson, 1972; Bhat *et al.*, 1973). Exposures usually have occurred by inhalation of particles. These are detected and evaluated more readily by whole-body counting techniques than by measurement for cobalt radioactivity in the urine. After inhalation exposure, about 80 percent of cobalt isotopes are eliminated with a biological half-time of one day or less (Edvardsson, 1972; Sill *et al.*, 1964). The remainder is eliminated much more slowly, varying considerably from case to case due presumably to chemical and particle size differences. Sill *et al.*, (1964) found biological half-times varying from 70 to 177 days. In one case the effective half-time was two years. Edvardsson (1972) found a second component in the elimination amounting to 5-10 percent of the total activity that had a biological half-time between 5 and 30 days. A third component in one case gave a biological half-time of about 200 days. Newton and Rundo (1971) studied 5 men who had inhaled cobalt metal or its oxide for periods of up to 11 years; the biological half-times in their chests ranged from 1.4 years to 17 years. Other studies by Cofield (1963) and Gupton and Bhat (1972) gave lung clearance half-times of 3 months to 2.5 years.

A typical excretion curve for inhaled ^{60}Co is shown in Figure 4.1 (page 53).

Intravenous $^{60}\text{CoCl}_2$ administered to human subjects was retained for long periods, as much as 9 to 16 percent of the dose being eliminated with biological half-times of about 2 years (Smith *et al.*, 1972). The absorbed fraction of an oral dose was retained by the whole body similarly to ^{60}Co given intravenously. The absorption of orally administered $^{60}\text{CoCl}_2$ was 5 percent or less when only small amounts of stable cobalt ($<1 \mu\text{g Co}$) accompanied the dose, but increased to more than 20 percent when larger quantities of stable cobalt (1.2 mg) were given. About $\frac{1}{2}$ of the total body content of ^{60}Co after oral or IV administration is retained in the liver.

These various studies in humans suggest that an appreciable fraction of cobalt isotopes is retained with a much longer biological half-time than the value of 10 days used by the ICRP (1968).

There have been no cases of significant radiation exposures resulting from internal deposition of cobalt radionuclides. No treatment of cases has been necessary. For ingestion, however, stomach lavage followed by a small or medium sized meal is indicated. Chelating drugs do not penicillamine (Section 7.3.5.5) can be considered in the case of a serious exposure where even small gains would be helpful. It should be used as soon as possible after exposure.

6.6 Curium

Curium, (Cm), element number 96, has thirteen isotopes, ^{238}Cm to ^{250}Cm . Because of their good power-to-weight ratio, high specific activity, and insensitivity to temperature variations, curium isotopes have been used in thermoelectric generators for unmanned meteorological stations and some aerospace satellites. Their principal use now is as a source material to be irradiated in high flux neutron fields in order to produce transplutoniums such as berkelium and einsteinium.

Curium's high specific activity causes spontaneous heat release in addition to the hazard of its energetic alpha particles, neutrons, and gaseous daughter products. Heat production by ^{242}Cm is 120 watts/g and solid compounds may generate enough heat to reach a red glow due to self-irradiation. It also undergoes spontaneous fission with the release of two to eight fast neutrons per fission; the fission half-life is 1.3×10^7 years.

Curium-244 has a wider distribution in industry than most of the other curium isotopes because it has been used in thermoelectric

generators. It is produced in nuclear reactors during the irradiation of ^{242}Pu and can be recovered from the processed waste.

In general, curium compounds tend to be more soluble than their plutonium analogues. The critical organ for soluble curium compounds is bone, where the effective half-life for ^{244}Cm is 16.7 years. The effective half-life for ^{242}Cm is only about 155 days due to its short 163-day physical half-life, while ^{245}Cm with an extremely long physical half-life, 9300 years, is assumed to have an effective half-life of 199 years.

The absorption of soluble curium salts from the lung is rapid. Fifteen minutes after rats completed an inhalation exposure, 15 to 45 percent of the amount retained in the lung had passed through the alveoli and 10 percent had been deposited in bone (Nenot, 1971). The plasma clearance rate of curium is more rapid than for plutonium and less influenced by chemical form (Turner and Taylor, 1968).

After injection of the soluble ^{244}Cm citrate in beagles, initial excretion was almost entirely in the urine but later, reflecting liver uptake and excretion, it was excreted almost equally in the feces and urine (Lloyd *et al.*, 1973).

In general, curium, like the other transplutonic elements, seems to be more mobile than plutonium and thus irradiates soft tissues to a greater degree (Stannard, 1975). The large uptake by the liver is distributed diffusely within the organ. The uptake in the bones is greatest on surface mucoproteins in areas of enchondral ossification (Simon, 1972).

Since curium compounds tend to be soluble, DTPA therapy should be started as quickly as possible after exposure. Desferrioxamine (DFOA) does not reduce the retention of ^{244}Cm (Taylor, 1967; Volf *et al.*, 1977) and is not recommended.

Two cases of human exposure to ^{244}Cm have been reported (Sanders, 1974). One worker inhaled airborne particles of a poorly identified but relatively soluble aerosol containing curium. Four and one-half hours after the incident, 14 nCi were estimated to be in the lung by using the technique of *in vivo* chest counting of the 40 keV x-ray emission. One gram of CaDTPA was administered 2½ hours after the incident and the lung burden dropped to 5 nCi within four days.

In the other case, a worker inhaled mixed oxides of curium and americium. Three hours after the incident, the lung was estimated to contain 456 nCi, but 24 hours later it appeared to contain 1523 nCi, even though no further exposure had occurred. This delayed increase in activity is not an unusual finding when low-energy x rays from alpha emitters are being counted in the thorax. Such a change may represent merely a shift in counting geometry due to the relocation of the

radioactivity in the lungs. During the first seven days, 202 nCi were excreted in the feces. DTPA was given promptly and may have increased the excretion. DTPA was not given again until the fiftieth day after the incident, when 99.8 percent of the soluble ^{244}Cm had already been excreted. At that late time it was not particularly effective.

6.7 Gold

There are twenty-four radioactive isotopes of gold, (Au), element number 79, but ^{198}Au is the only one that is used widely in medical therapy, biological research, and occasionally as a calibration source. It is produced by bombardment of stable ^{197}Au with neutrons. Gold-198 emits 0.97 MeV beta particles and, in 95 percent of disintegrations, 0.412 MeV gamma rays; its half-life is 2.7 days.

For medical uses, ^{198}Au was formerly used primarily in a colloidal form. The principal therapeutic use of ^{198}Au colloid was in the palliative management of malignant pleural, abdominal, and pericardial effusions. Approximately 90 percent of the radiation dose is delivered by the beta particles which have a mean penetration of about 0.4 mm and a maximum of less than 4 mm in tissues.

When non-colloidal ^{198}Au solutions are taken in by mouth, about 10 percent is absorbed into the blood. Of this about one quarter goes into the liver and kidneys (Simon, 1972). Gold is eliminated from the body via the feces and the urine.

Colloidal gold injected intravenously is distributed differently and little is excreted within the effective half-life of 2.6 days. The phagocytic reticulo-endothelial (R-E) cells of the liver, spleen, bone marrow, and lymph nodes quickly remove the colloid from circulation (Säterborg, 1973). While there is some dependence upon particle size, the R-E cells of the liver retain about 80 to 85 percent, the spleen 5 to 10 percent, and the other tissues the remainder of the usual commercial preparation.

In accident situations, the organ dose calculations must take into consideration the widely varying distribution of gold depending on whether the material is in the ionic or colloidal form and the route of administration. It was not possible to do this in the simple presentation of doses in Table 2.6 (page 14); therefore, dose must be recalculated to account for the estimated organ distribution in an accidental exposure case.

If a therapeutic dose of ^{198}Au had been injected into a patient shortly prior to his death, the pathologist would have to evaluate the quantity present in the peritoneal or pleural fluids prior to proceeding with an autopsy. Proper procedures can be found in NCRP Report 37 (1970).

No industrial type accidents have occurred that resulted in ^{198}Au exposures. Two instances of misadministrations of ^{198}Au resulted from carelessness in ordering the ^{198}Au colloid and in administering intravenously the therapeutic preparation in place of the diagnostic (Baron *et al.*, 1969). The doses were 200 and 120 millicuries instead of the intended 200 and 120 microcuries, respectively. The calculated dose to the liver and spleen from 200 millicuries was 7,300 rads; the bone marrow received an estimated 440 rads. It was bone marrow failure that led to death 70 days later from intracerebral hemorrhages due to severe thrombocytopenia. The thrombocytes, in contrast to the other cellular elements of the blood, showed little evidence of recovery. Liver function tests gave no signs of deterioration of that organ, but with further time hepatic damage may have occurred.

There is no known therapy that is useful for ^{198}Au colloid, once the particles are phagocytized by R-E cells. Bone marrow failure might be treated with transfusions of suitable blood components. For gold in ionic solution, dimercaprol (Section 7.3.5.4) or penicillamine (7.3.5.5) may be tried although the short effective half-life of ^{198}Au suggests that the dose reduction resulting from these agents will be limited.

6.8 Iodine

Of the more than 20 radioactive isotopes of iodine, (I), element number 53, about half occur as fission products, and among them, ^{131}I contributes an increasingly important portion of the total activity starting at several hours after fission. The dominant internal exposure after a reactor accident or nuclear weapons test or any incident involving fresh fission products is likely to be ^{131}I (Roberts, 1966). However, the short-lived radioisotopes, ^{132}I , ^{133}I , ^{134}I , and ^{135}I , with half-lives from 52 minutes to 6.7 hours, can contribute significantly if the person is in close proximity to a fresh fission product release.

Iodine-131 has a physical half-life of about 8 days and an effective half-life in humans of about 7.6 days. It decays by emitting beta particles of four energies [0.25 to 0.81 MeV, the predominant one is 0.61 MeV (87.2 percent)] and gamma rays of five energies [0.08 to 0.72 MeV, the predominant one being 0.36 MeV (79 percent)]. The effective energy is generally taken as 0.22 MeV. An important shorter-lived iodine found in fresh mixed-fission products is ^{132}I , which is derived via ^{132}Te , a 78-hour half-life beta emitter. Iodine-132 has a 2.3-hour half-life and decays with six betas (0.80 to 2.14 MeV) and many gamma rays (0.38 to 1.39 MeV). Although the following discussion centers on ^{131}I , the metabolism and treatment is applicable to the other radioac-

tive iodines. Only the risk (dose) estimate changes as the mixtures of iodine isotopes shift according to their effective half-lives and energies.

In a reactor accident involving fuel rod leakage, a sizable fraction of the inventory of ^{131}I becomes available because of its volatility. In a major reactor accident, the containment vessel is designed to retain nearly all of such vapors. In the Windscale reactor fire in England in October 1957, it was estimated that 20,000 Ci of ^{131}I were released through a 410-foot stack (Dunster *et al.*, 1958). This air-cooled reactor had no containment features to retain volatile products. In weapons tests, each kiloton of fission energy produces 30,000 Ci of ^{131}I (Holland, 1963). Iodine may be released from ruptured fuel elements, during dissolution of spent fuel elements (Sill and Flygare, 1960), from leaks (Bhat *et al.*, 1973), and malfunctioning ventilation systems (Frederickson, 1970). Exposures may also occur during manufacture of iodine radiopharmaceuticals and sources (Soldan, 1968), during research, and in transportation accidents (Paas, 1967). Due to its volatile character and ease of absorption, potentially exposed persons should be monitored after any accident where release of radioactive iodines is suspected.

Small exposures usually occur as the result of inhalation, but ingestion through mouth pipetting (Haas, 1970) and absorption through the skin have been reported (Low, 1970; Harrison, 1963). When extensive environmental contamination occurred during the Windscale reactor accident in 1957, and after the Sedan and Dominic II nuclear tests in Nevada (Bernhardt *et al.*, 1971), contamination of the milk supply by cattle grazing on contaminated grasslands was a major problem. Fall out from atmospheric nuclear weapons testing exposed the residents of certain of the Marshall Islands to external radiation and to radioiodine by ingestion (Conard *et al.*, 1970; 1975).

In view of the quantity produced, transported, and used, it is a tribute to the care and precaution used in industry that only 20 cases of internal exposure to radioiodine were reported to the AEC by its contractors during the period 1957-66. Sixteen of these cases occurred as the result of two incidents, one at a chemical processing facility and the other after an underground nuclear detonation. Only 11 exceeded the permissible dose and none caused obvious injury (Ross, 1968). During the same period, six cases occurred among AEC licensees. All but one of these were the result of processing activities; the one exception happened during a gas release from a defective fuel element (Roeder, 1968).

Most of the iodine in accidents will be soluble and quickly absorbed via inhalation, ingestion, or the skin or any combination of these.

Inhaled iodine reaches equilibrium with body fluids in about $\frac{1}{2}$ hour (Ramsden *et al.*, 1967). The mean values for normal 24-hour ^{131}I thyroid uptake in six test groups in the United States ranged from 12 to 20 percent of the total oral dose (Ghahremani *et al.*, 1971). The percentage of the dose of radioiodine that is present in the thyroid gland one day after ingestion is similar for children and adults (Van Dilla and Fulwyler, 1964). Even though the adult thyroid gland is considered a relatively radioresistant organ, radiation exposure has resulted in an increased frequency of nodules and cancers (Conard *et al.*, 1970; 1975; UNSCEAR, 1977). Patients who received 3 mCi or less for the treatment of hyperthyroidism have developed hypothyroidism, some as late as 17 years after treatment (Glennon *et al.*, 1972).

The maximum permissible organ burden for continuous exposure to ^{131}I is 0.7 μCi in the thyroid (NCRP, 1959; ICRP, 1960) or a thyroid dose of 15 rems per year. In cases of environmental contamination with radioiodine where the ^{131}I is transferred via grass \rightarrow cow \rightarrow milk \rightarrow man, the physician may be called upon to advise patients on the safety of drinking milk after a reactor accident. In general, there is about a 5-day effective half-life of ^{131}I on vegetation (eight-day physical half-life combined with a 14-day vegetation half-life, varying with growth rate and weathering) (FRC, 1964). An infant drinking one liter of milk per day contaminated to an initial level with 1 $\mu\text{Ci/l}$ will receive a total cumulative dose to the thyroid of about 16 rems (FRC, 1964).

When environmental contamination of pastureland occurs, effective protective actions include changing the cow forage from contaminated to uncontaminated feed, withholding milk from consumption, and diverting milk from direct use to milk products, such as cottage cheese, firm cheese, condensed milk, or powdered milk (Bernhardt *et al.*, 1971; White and Moghissi, 1971). Emergency reference levels have been suggested that can be used to determine when countermeasures may be indicated. These levels are 0.25 $\mu\text{Ci/l}$ as a peak level in milk and 1.5 $\mu\text{Ci/m}^2$ on pasture (Bryant, 1969).

Preventive actions to reduce exposure to radioactive iodine from milk must be taken promptly if they are to be effective. Sensitive and rapid field methods for monitoring contamination of milk have been developed (Porter and Carter, 1965). Techniques for the routine monitoring of milk from cattle grazing in pastures surrounding a reactor are now required to detect the very low concentration of 0.25 pCi/l (USAEC, 1973). Means of protecting the thyroid gland after nuclear reactor accidents are discussed in NCRP Report 55 (1977b).

When a person has been exposed to radioiodine the thyroid gland can be monitored for radioactivity by holding a beta-gamma detector

close to the suprasternal notch. If the detector is a NaI crystal plus pulse height analyzer and calibrated with a phantom, reasonably accurate estimates of thyroid uptake can be made. Counts made soon after exposure often may be unreliable because of skin contamination. A scan of the thyroid in the Nuclear Medicine Department of a hospital will give a qualitative estimate of exposure and properly calibrated instruments used for radioiodine uptake studies can be used for a quantitative figure. The usual limit of detection is below the 1-2 μCi level. Urine bioassay can be used to measure excretion. Whole body counting will detect 3×10^{-4} to 3×10^{-3} μCi (Mehl and Rundo, 1963).

Individuals who have had an accidental occupational exposure to radioiodine, regardless of the route of exposure, should immediately be given a 300 mg KI or NaI tablet,¹ which provides 230 and 255 mg respectively of the stable iodide. Five or six drops of SSKI, Saturated Solution of Potassium Iodide, (1 g/ml) in a small glass of water is another convenient means to administer the stable iodide. Iodates, such as KIO_3 or $\text{Ca}(\text{IO}_3)_2$, are also effective (Auxier and Chester, 1972), but are not available as FDA-approved drugs. See Section 7.3.4.2 for additional discussion on the use of antithyroid drugs for exposure to radioactive iodine.

Daily administration of 300 mg KI should be continued for 7 to 14 days. This continuation of the blocking agent is needed to prevent recycling of the radioiodine (Bernhardt *et al.*, 1971). A combination of KI and thyroid stimulating hormone has been used but this offers little advantage over simple KI given promptly (Blum and Eisenbud, 1966).

Individuals exposed to large amounts of ^{131}I should be observed periodically for evidence of hypothyroidism, which may not develop for several years (Glennon *et al.*, 1972). The amount required to produce early myxedema in a patient with normal thyroid function is in excess of 150 μCi per gram of estimated thyroid gland weight (Chapman, 1965). For thyroid exposures in excess of 100 rads, an estimate of residual thyroid function should be made within two or three months after exposure by measurements of plasma T_4 and TSH by radioimmunoassay. At six months to yearly intervals thereafter, measurements of plasma T_4 and a clinical evaluation should be made.

¹ In general population exposures to ^{131}I , the NCRP (1977b) recommends a daily dose of 130 mg of potassium iodide for adequate blocking of the thyroid. One-half of that dose should be given to children under one year of age. In known occupational exposures to radioactive iodine discussed here, a slightly improved therapeutic effect can be gained by the higher stable iodide dose recommended here without a significant increase of side effects.

6.9 Mercury

Five radioactive isotopes of mercury, (Hg), element number 20, are produced by exposing mercury or mercury oxide to neutron bombardment. All but two have half-lives of less than a day. The longest-lived mercury radioisotopes are ^{203}Hg and ^{197}Hg with physical half-lives of 47 days and 65 hours, respectively. Mercury-203 decays by emitting a 0.21 MeV beta particle and a 0.279 MeV gamma ray. Mercury-197 decays by orbital electron capture with emission of two x rays (0.077 and 0.19 MeV).

Both isotopes have been used in nuclear medicine for scintigraphy of the kidneys or brain, but have been replaced now by other agents. Mercury radionuclides are used in biological or medical research laboratories. They are found occasionally in industrial settings, such as areas related to mercury isotope production around nuclear reactors or in radiopharmacies.

The effective half-life for ^{203}Hg is usually considered to be 11 and 8.2 days for the kidneys and whole body, respectively (ICRP, 1960). Investigations of exposure to different chemical forms of ^{203}Hg and by various means of administrations have indicated effective half-lives from 19 to 30 days (Edvardsson, 1972; Johnson and Johnson, 1968; Roedler *et al.*, 1972; Rahola *et al.*, 1972; and Scott, 1969). After accidental inhalation of elemental ^{203}Hg by two laboratory workers, the effective half-lives were found to be 16.6 ± 0.6 and 17.5 ± 2.0 days (Brown *et al.*, 1975).

Accidental exposures to mercury radionuclides have thus far not involved doses large enough to require treatment. In view of the relatively short effective half-lives, the available therapy may not prove to be of much value. In case of ingestion, gastric lavage should be the single most useful procedure if done within the first hour. Dimercaprol (Section 7.3.5.4) and penicillamine (Section 7.3.5.5) can enhance excretion of mercury. Sodium-2,3-dimercaptopropane-1-sulphonate was found to be clearly superior to various other chelating agents in the case of inorganic mercury (Gabard, 1976a) and methyl mercury (Gabard, 1976b) in rats.

6.10 Phosphorus

Phosphorus-32 (^{32}P) was the first radioactive isotope to be prepared in a cyclotron for biologic and therapeutic research purposes and was produced by irradiating red phosphorus, element number 15, with deuterons (Cohn and Greenberg, 1938; Lawrence *et al.*, 1939). This

product had a low specific activity, whereas practically carrier-free ^{32}P now is separated chemically from a target of sulfur-32 irradiated with neutrons. The physical half-life of ^{32}P is 14.2 days. It decays by emitting a beta particle with a maximum energy of 1.71 MeV and an effective mean energy of 0.69 MeV corresponding to a maximum range in tissues of 7 mm and a half-value layer in tissue of 2 mm.

Phosphorus is an essential element in living cells and, in standard man, the phosphorus content is listed as 1.1 percent of the body weight, or 780 g (ICRP, 1975). It is eliminated from the body principally via the urine. For soluble compounds of ^{32}P , the critical organ is the bone, which receives about 20 percent of the dose ingested or inhaled. Tissues with rapid cellular turnover rates also show higher retention due to the concentration of phosphorus in the nucleoproteins. This concentration has been used to provide a possibly useful differential localization pattern for certain nuclear medicine procedures. A long biological half-time for phosphorus, ~257 days for the whole body and 1155 days for bone, makes the effective half-life for ^{32}P about the same as its physical half-life, 14 days.

Phosphorus-32 is used widely in medicine, biochemical research, industry, and agricultural research. It is used in medicine for both diagnostic and therapeutic purposes. Although once used in the diagnosis of intraocular, intracranial, skin and breast tumors, this use has been supplanted by improved techniques designed around the specific characteristics of other radionuclides. Phosphorus-32 is used as a therapeutic agent for the treatment of polycythemia vera in initial doses of 50 to 100 $\mu\text{Ci}/\text{kg}$ of body weight of an isotonic solution of $\text{Na}_2\text{H}^{32}\text{PO}_4$, given intravenously or orally. In biochemistry, industry, and agriculture, ^{32}P is used as a tracer to study phosphorus-containing processes, such as nucleotide biochemistry or fertilizer utilization.

Serious accidental exposures to ^{32}P have not been reported from its use except due to misadministration in medical use. An accidental overdose of ^{32}P to a patient being treated for thrombocytosis occurred because the month of the assay of the stock solution was recorded on the bottle erroneously as 9 for September instead of 10 for October with the result that 16.2 mCi were administered instead of 4.05 mCi (Cobau *et al.*, 1967). The characteristic fall of white and red cells developed progressively for six weeks after exposure. Recovery began on about the forty-fifth day and was not quite complete 300 days later. The patient was free of symptoms throughout the entire course of the syndrome. Treatment in this case was begun on the ninth day after ^{32}P administration, shortly after the error was discovered. The therapy, continued over an 18-day period, included large doses (5 g) of phosphate by mouth daily as the buffered sodium salt (Section 7.3.3.4),

calcium (540 mg) given intravenously daily (Section 7.3.3.6), and 200 units of parathyroid extract intramuscularly every 6 hours (Section 7.3.4.6). This regimen, although started late, resulted in an estimated 38 percent reduction of radiation dose to the bone marrow.

Recommendations for treatment of non-radioactive phosphorus poisoning by ingestion are included here for consideration in the case of accidental ^{32}P ingestion. Treatment immediately after ingestion of stable phosphorus, according to Arena (1976), should include thorough gastric lavage with potassium permanganate (1:5000) or 3 percent hydrogen peroxide. Copper sulfate, which forms insoluble copper phosphide, may be given in a dose of 0.25 g in a glass of water. Mineral oil (100 ml) will help prevent absorption and hasten elimination. This can be repeated in 2 hours. Use of aluminum hydroxide gel or aluminum phosphate gel (Section 7.3.2.7), or a mixture of aluminum and magnesium hydroxide, can also be used to help prevent gastrointestinal absorption.

6.11 Plutonium

Plutonium, (Pu), atomic number 94, a transuranic element, is a silvery-white reactive metal that melts at 639.5°C and oxidizes readily on warming in moist air. In powdered form the metal may be pyrophoric, igniting spontaneously in the range of 300° to 350°C. Of its 15 isotopes (^{232}Pu to ^{246}Pu), all of which are radioactive, ^{238}Pu and ^{239}Pu are the most likely to be encountered.

Plutonium-239 is an alpha emitter with a 24,400-year physical half-life. It emits two principal alpha rays [5.16 MeV (88 percent) and 5.11 MeV (11 percent)], which are accompanied by infrequent gamma rays [e.g., 0.039 MeV (0.0012 percent)]. A mass of ~16 g of ^{239}Pu equals one curie of radioactivity. Plutonium-239 has the property of fissioning when exposed to a slow neutron flux. This is the basis for its use as a fissile fuel for nuclear explosives and for generating heat for power production (Weast, 1975).

Plutonium-238 has an 86-year physical half-life and is finding increased use as a heat source because of its high rate of emission of alpha particles. A mass of ~57 mg equals one curie of radioactivity. The principal alpha emissions have energies of 5.50 MeV (72 percent) and 5.46 MeV (28 percent). Plutonium-238 has been used in thermoelectric generators as a power source for lunar space missions, communications satellites, heart pacemakers, and experimental power sources for artificial hearts (Bair and Thompson, 1974). On a mass basis ^{238}Pu

is 280 times more hazardous than ^{239}Pu because of its greater radioactivity per unit mass.

From a biological standpoint the high chemical reactivity of plutonium is an important characteristic. Plutonium can exhibit five oxidation states from a valence of +3 to +7 (Taylor, 1973). It has a marked tendency to hydrolyze and to form complex ions under physiologic conditions. The compounds formed may be monomeric, in which state any particulates are less than about $0.01\ \mu\text{m}$ in diameter, or polymeric with particle diameters ranging from about $0.01\ \mu\text{m}$ to over $1\ \mu\text{m}$. In the body, monomeric compounds become converted to at least minimally polymeric forms. Hydrolytic reactions also can change the chemical form after intake. Biological ligands to which plutonium may bind in the body include proteins, apoferritin, amino acids, phospholipids, hydroxy acids, and other metabolites (Taylor, 1973). Polymers and particulates formed by hydrolysis lead to binding on cell surfaces and phagocytic uptake of plutonium.

Because of the unique history of plutonium as a man-made element, it was possible to consider its potential biological hazards and to impose controls for personnel protection from the start. Radiobiological research on the effects of plutonium has been extensive (Bair and Thompson, 1974; Vaughan *et al.*, 1973; Bair *et al.*, 1973). The principal routes of entry to the body are through inhalation and contaminated wounds; ingestion and contaminated intact skin result in little absorption and are not important modes of exposure. After entry into the body some or all of the plutonium is solubilized by the body fluids, including blood, and redistributed within the body. The rate and amount of plutonium translocation will be markedly influenced by the deposition site, the physical and chemical form of the deposited compound, and the specific activity of the material. Ultimately, the plutonium will be distributed by the blood to the skeleton, liver, and all other tissues in the proportion 45: 45: 10 percent, respectively (ICRP, 1972). The retention half-time in the whole body has been estimated to be about 200 years in man (Langham, 1959; Durbin, 1972), and the half-times in the skeleton and liver are assumed to be 100 years and 40 years, respectively (ICRP, 1972).

The portal of entry of plutonium into the body is the chief determinant of the course of the subsequent contamination and appropriate therapeutic efforts. The unbroken skin surface offers high resistance to penetration by plutonium except mechanically as by a contaminated metal splinter or glass chip. Experiments on the unbroken skin of animals and man with various solutions of plutonium being applied for one day indicated that 0.002 to 0.25 percent of the applied plutonium was absorbed (Vaughan *et al.*, 1973). Subdermal or intradermal pene-

tration, such as in contaminated wounds, may result in long-term localization of the plutonium at that site and the possible appearance of a fibrous nodule (Lushbaugh and Langham, 1962; Lushbaugh *et al.*, 1967). The possible development of a sarcoma or carcinoma in such nodules is a matter of concern, although none has been reported to date. Contamination of wounds that have penetrated the skin may also lead to translocation of some of the material to the liver and skeleton. Ingestion of plutonium results in the absorption of approximately 0.003 percent by the intestine (ICRP, 1972).

Inhalation is a particularly important route of intake as it accounts for about 75 percent of industrial exposures (Ross, 1968). The amount retained in the lung is highly variable for it depends on the particle sizes and chemical form of the aerosol. If the compound is soluble, e.g., nitrate, citrate, fluoride, this retained plutonium may be largely absorbed into the blood circulation within a few weeks and translocated to the ultimate deposition sites, principally bone and liver. If the inhaled plutonium particles are relatively insoluble, e.g., in the case of high-fired oxides, their retention in lung tissue, pulmonary lymph nodes, and tracheobronchial lymph nodes will be high with a gradual translocation of small amounts over a period of months or years. In experimental animals, the pulmonary retention half-time is about 150 days for plutonium chloride and ammonium plutonium-pentocarbonates, 200 days for plutonium fluoride and citrate, 250 to 300 days for pulmonary nitrate, and up to 1000 days for PuO_2 (Bair *et al.*, 1973). Data on lung retention for plutonium compounds in man are not available in similar detail, but plutonium analyses of autopsy tissues from occupationally exposed workers show the highest concentrations per gram of tissue to be in tracheobronchial lymph nodes followed by lung and liver many years after inhalation exposure (Campbell *et al.*, 1973; 1974; Lagerquist *et al.*, 1973; and Norwood *et al.*, 1973).

There has been considerable experience in the management of persons exposed to plutonium. The cases can be classified as intact skin contamination, contaminated wounds, and inhalations; many cases are mixtures of these. Ingestion of plutonium has not been encountered as an exposure problem in industry, although cases of skin contamination or inhalation always involve the probability of some ingestion. Treatment is not required after ingestion because the low absorption rate does not result in any appreciable uptake in the body.

Plutonium contamination of intact skin is easily managed by washing with water, detergents, and occasionally other agents (see Section 7.1). Thousands of such cases have been cleaned up with no significant systemic absorption as long as the skin remains intact. Areas that are

not completely cleansed can be left alone after all loose material has been removed. Usually gloves or a temporary covering of some type are placed over the contaminated skin that resists cleanup attempts to retain the plutonium while the individual is at home or at work. It is not necessary to isolate the individual.

Contaminated puncture wounds and burns are a more serious problem because significant body burdens can result from these types of injuries. All wounds with possible plutonium contamination should first receive simple decontamination, such as washing and irrigating. The need for more extensive treatment by excision requires clinical judgment considering the skin area involved, ease of excision, and the quantity of plutonium in the wounds. Wounds containing over 4 nCi of plutonium should be serious candidates for such additional treatment. The treatment consists of immediate chelation therapy with CaDTPA, prior to surgical excision of the wound, to prevent possible systemic absorption (see Section 7.2). In wound decontamination, much attention is paid to locating the contaminating material as precisely as possible so that complete removal by surgical excision is accomplished with as little functional loss as possible. In burn cases, the burn surface shall be flushed with sterile saline or water. A large portion of plutonium in the burn area is likely to be removed later when the eschar sloughs off.

Review of human cases suggests that CaDTPA given immediately after wound deposition may result in urinary excretion of 50 percent or more of the material reaching the systemic circulation. The amount of plutonium that may transfer to the rest of the body within the first day or two will vary from only a few percent of that in the wound to well over 50 percent. Since this uptake cannot be predicted reliably in the usual accident situation, the use of CaDTPA is advised whenever the quantity in the wound is judged to be significant. A few illustrative cases are listed to show variations in treatment.

Schofield *et al.* (1974) reported the case of a wound in the right hand that was contaminated with an estimated 14 μ Ci of plutonium oxalate. Surgical excisions on days 1 and 14 were successful in reducing the content in the wound to about 1.5 μ Ci. Systemic CaDTPA therapy, a total of 13 grams during the first 14 days and at intervals throughout the next six months, is estimated to have diverted about half (~ 0.6 μ Ci) of the systemic plutonium absorbed from the wound into the urine. The CaDTPA intravenous administrations were usually given as 0.25 g per day with an upper dose of 1 g per day given at the time of the second excision.

In a review of several plutonium wound cases, Dolphin (1976)

concluded that CaDTPA therapy appeared to be more successful in cases involving contaminated wounds than in those involving inhalation.

Lagerquist *et al.* (1965) reported the treatment of a worker sprayed with an acid solution of plutonium chloride and plutonium nitrate that resulted in inhalation and ingestion of plutonium as well as skin and burn contamination. The skin, except for the burned areas, was decontaminated with dilute sodium hypochlorite solution. The patient received 11 one-gram doses of DTPA by intravenous injection beginning 1 hour after the accident and at intervals through day 17. Burn eschars were removed 2 weeks after the accident and were found to contain most of the plutonium. Treatments used in this case were considered highly effective. Prompt CaDTPA treatment was reported to be similarly effective in another contaminated acid burn incident (Lagerquist *et al.*, 1967a). Twenty-seven daily one-gram intravenous CaDTPA treatments, beginning 1 hour after the accident, resulted in elimination of more than 96 percent of the systemic burden. As in the previous case, much of the contamination was removed with the eschar.

The effectiveness of CaDTPA, using a regimen similar to those reported above, was considered inconclusive in the case of a wound in the thumb from a plutonium-contaminated metal sliver (Lagerquist *et al.*, 1965). Initially, the sliver was removed without tissue excision. On day 4, tissue excisions were performed at the points of entrance and exit of the sliver; the entry site contained about 98 percent of the plutonium removed by those excisions. Wound counting performed over several months indicated a movement of embedded plutonium toward the skin surface. Nodules that formed concurrently with increased wound counts were excised and found to contain essentially all the plutonium estimated to have remained in the thumb. While CaDTPA was effective in removing plutonium from the system, the continued presence of plutonium in the wound site indicates that the remaining relatively insoluble particles were probably not influenced by chelation therapy. McClanahan and Kornberg (1968) showed, in rats, that washing with CaDTPA would probably not accelerate greatly the removal of plutonium from contaminated wounds, nor would it increase the movement into the blood and so increase the amount retained systemically.

In the case of a puncture wound contaminated with plutonium-238 nitrate (Jolly *et al.*, 1972), CaDTPA was administered intravenously four days per week for about 11 weeks followed by a 32-week period of no treatment and then by a 90-week period in which CaDTPA (1 g) was administered intravenously or by aerosol on 2 successive days

each month. The wound area was excised 2 hours after the accident. The ^{239}Pu content in the body after wound excision was estimated to be 103 nCi. The CaDTPA treatment for more than two years was credited for the further reduction in the body burden to an estimated 31 nCi.

Additional cases of puncture wound injury have been reported (Lagerquist *et al.*, 1967b; Swanberg and Henle, 1964). In each instance of puncture wound, prompt CaDTPA therapy and one or more tissue excisions appeared to be effective.

The use of CaDTPA or ZnDTPA chelation is indicated for cases of plutonium aerosol inhalation, but the results have been disappointing in many instances. This is due to the fact that the chemical form most commonly encountered in aerosols is PuO_2 . This compound is transferred at a relatively slow rate from the lung into the systemic circulation over many weeks or months. Thus there is little systemic burden of plutonium available for chelation in the early period after exposure and there is never a time when a sizable systemic burden is available in extracellular spaces for effective chelation.

In spite of this experience, CaDTPA should be used as soon as possible after significant inhalation exposures because the chemical form usually is not known in these accidents. The therapeutic effect on soluble forms is manifest principally in the first 12 to 24 hours and a therapeutic trial should be initiated immediately to insure that the patient will benefit from the treatment if some systemic uptake has occurred.

An estimate of the possible effectiveness of CaDTPA chelation therapy in persons who have inhaled plutonium particulates can be made based on the results of a relatively few cases treated to date. Norwood (1960, 1962a) showed the therapeutic effect of giving CaDTPA intravenously to seven individuals starting several years after inhalation exposure to plutonium. The rate of elimination of ^{239}Pu via the urine was increased 45 to 120 times and fecal elimination increased sixfold. Long-term administration in one case showed a gradually decreasing effectiveness until at the end of 50 weeks of intermittent therapy, it was only 20 percent as effective as it was at the beginning. About 20 percent of the estimated body burden of plutonium was removed by this long-term therapy when started 5 years after deposition (Norwood and Fuqua, 1969).

Data from two puncture wound cases and several inhalation cases treated with CaDTPA were used to derive an empirical urinary excretion model for single and multiple DTPA treatments (Hall *et al.*, 1978). The percentage of inhaled soluble plutonium excreted in urine both after a single and after five treatments is shown in Table 6.1. Figure 6.1 gives a plot of the predicted urinary excretion rates after a single

TABLE 6.1—Predicted plutonium excretion in urine after inhalation of a soluble plutonium aerosol with and without CaDTPA treatment

Week	Percent of initial systemic Pu deposition excreted in urine		
	No treatment	Single treatment ^a	Five treatments ^b
1	0.64	38	42
2	0.24	11	13
3	0.17	5.5	7.7
4	0.13	2.8	3.6
5	0.11	1.4	1.8
6	0.09	0.71	0.91
7	0.08	0.37	0.46
8	0.07	0.19	0.24
9	0.07	0.11	0.13
10	0.06	0.07	0.07
TOTAL	1.7	60	70
→			

^a CaDTPA therapy (1 g in 4 ml by aerosol) on day one immediately after inhalation.

^b CaDTPA therapy (1 g in 4 ml by aerosol) with optimal schedule on days 1, 2, 4, 7, and 15. (From Hall *et al.*, 1978.)

administration of CaDTPA immediately after or two days after an acute intake of soluble plutonium.

The Pu-DTPA excretion model has been used to evaluate bioassay data on five other workers exposed to airborne ^{239}Pu contamination and treated immediately with CaDTPA by aerosol. The excretion curves for the various treatment regimens predicted by empirical equations fit the bioassay results obtained for these cases reasonably well (Hall *et al.*, 1978). Good correlation was also found in a case of inhaled ^{239}Pu fluoride treated with 1 g of CaDTPA intravenously at 3 hours post-exposure and days 2, 5, and 8 post-exposure (Voelz, 1979). These models indicate that the DTPA therapy initiated immediately after a contamination incident may reduce the body burden of soluble plutonium by a factor of about 3.

In other accidental inhalation cases, chelation therapy with CaDTPA has failed to achieve a significant increase in the elimination of plutonium. These cases generally involve PuO_2 , particularly the high-fired oxides. The gradual transfer of plutonium from the lung to other organs through the systemic circulation is reflected in a rising urinary excretion pattern of several months to years. Low plutonium values in urine for the first few weeks after this type of inhalation exposure can lead to a significantly low estimate of the lung exposure unless periodic urine samples are taken over a period of months and years. Figure 6.2 shows several such predicted excretion curves based on lung clearance half-times of 70 and 700 days. Schofield and Lynn (1973) found little effect in three cases given CaDTPA within a few hours following PuO_2

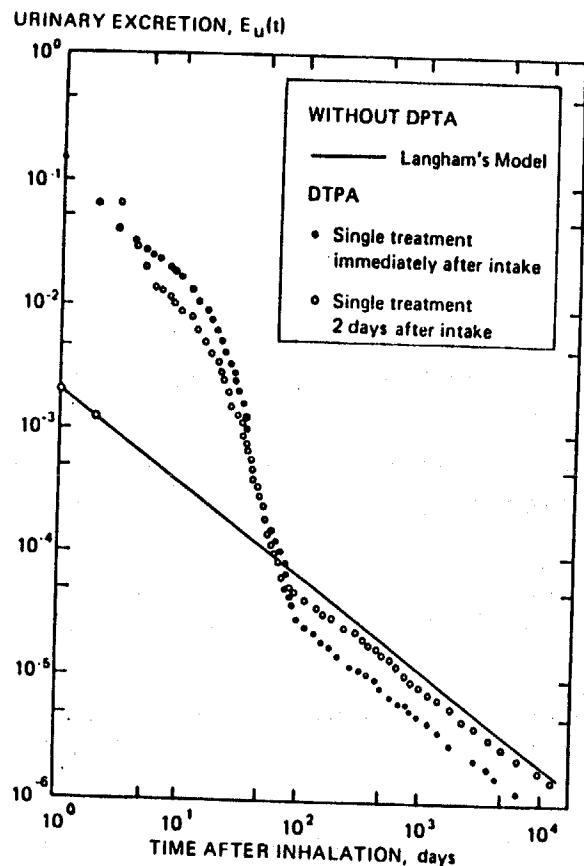


Fig. 6.1. Predicted urinary excretion rates (portion of body burden excreted per day) after single intake of soluble plutonium with DTPA administered immediately after and two days after exposure compared to that predicted by Langham's equation for untreated persons (Hall *et al.*, 1978).

inhalation. Catsch (1976) notes that "the efficacy of DTPA is extremely poor or even nil in the case of polymeric or highly insoluble compounds of the radionuclides. For instance, it is absolutely impossible by DTPA to remove PuO_2 from an intramuscular deposit or from the lung; this holds both for systemic and local administration of DTPA." Attempts to stimulate phagocytosis and the mucus-transport mechanism or to use expectorant drugs have not been successful in animal studies (Tombropoulos, 1964; Bair and Smith, 1969).

The only procedure that is useful in enhancing the clearance of insoluble particles, like PuO_2 , from the lung is bronchopulmonary

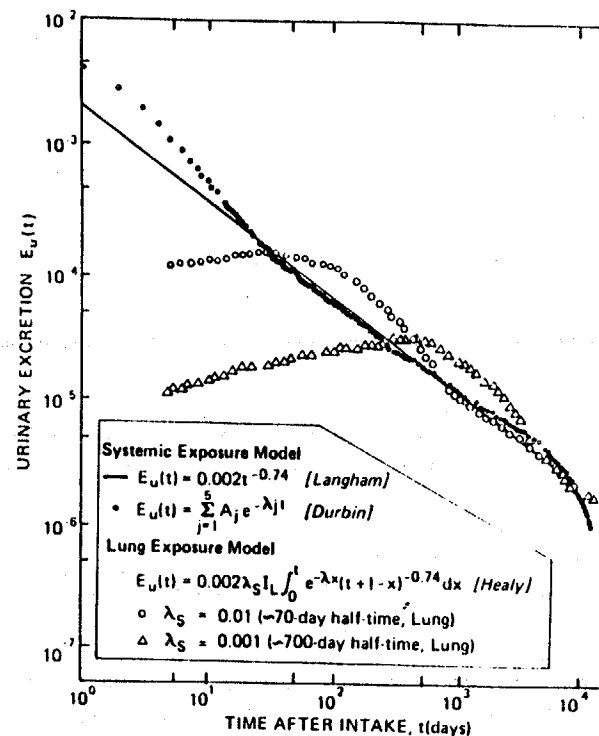


Fig. 6.2. Predicted urinary excretion rates (portion of body burden excreted per day) plotted as a function of time after an acute plutonium inhalation. The plots of Langham's and Durbin's equations represent excretion of the lung burden transfers to the blood immediately after exposure. The curves of Healy show predicted excretion when the lung to blood transfer occurs with half-times of ~70 and ~700 days (Hull *et al.*, 1978).

lavage (Section 7.4). Here the physician must balance carefully the risk of this procedure against the risk of future health effects created by the estimated lung burden (Section 5.2). Use of repeated lavages should be able to remove 25 to 50 percent of plutonium that would otherwise be retained in the lung.

6.12 Polonium

Polonium-210 was used extensively in early atomic weapons manufacture and later was employed briefly in thermoelectric generators in space satellites. The first communications satellite was powered by a

^{210}Po source. Polonium-beryllium neutron sources have now largely been replaced by plutonium-beryllium or transplutonic sources. Polonium-210 is used as a static eliminator (Robertson and Randle, 1974) in a variety of applications.

Polonium, element number 84, is a soft silvery-gray metal, much like lead in appearance. It volatilizes readily in a vacuum at elevated temperatures. When deposited on Pyrex glass or quartz, it eventually produces small irregular cracks called crazing (Goode, 1956). Therefore, old polonium ampoules should be considered hazardous. Polonium readily forms halides and many polonium compounds are relatively soluble.

The atmosphere normally contains polonium. It arises from radium-226, which occurs widespread in nature in the earth's crust (Hill, 1965). Contributions have been added by such man-made activities as burning fossil fuels, nuclear weapons testing in the atmosphere, and accidents such as the Windscale reactor accident. Grazing animals take up ^{210}Po from contaminated grass and concentrations greater than 1000 pCi/kg have been found in caribou in the Arctic (Hill, 1965). Marine organisms ranging from plankton to shellfish, crabs, and fish (Hoffman *et al.*, 1974) are often contaminated with ^{210}Po . Cigarettes have been found to contain 0.49 ± 0.07 pCi per cigarette (Hill, 1965) and it has been suggested as a possible cause of lung cancer (Marsden, 1964). Such information can be of some importance to the physician when he receives a bioassay report stating a low-level ^{210}Po content that cannot be explained on the basis of an occupational exposure.

Polonium-210, formerly called radium F, is the last radioactive member of the uranium-radium radioactive series and has therefore been extensively studied in uranium miners. After years of exposure, about 78 percent of the polonium body burden will be found in the skeleton with important deposits in the lung, liver, muscle, lymph nodes, kidney, spleen, and blood (Blanchard and Moore, 1971). This distribution probably is derived more from the preceding isotopes in the chain than from ^{210}Po itself.

Polonium-210 decays by alpha emission with a physical half-life of about 138 days. The biological half-time in the whole body is about 40 days (ICRP, 1968), while for the spleen and kidneys it is somewhat longer, about 60 and 70 days, respectively (ICRP, 1960). The longest effective half-life, 46 days, is in the kidneys. The critical organs are considered to be the spleen and kidneys (ICRP, 1960). Studies on several species of laboratory animals have shown the highest concentrations of ^{210}Po to be in kidney, ranging from 5 to 10 percent of the injected radionuclide (ICRP, 1968).

The metabolism of polonium chloride has been studied in terminal human cancer patients (Silberstein *et al.*, 1950). After intravenous

injection of 0.17 to 0.3 $\mu\text{Ci}/\text{kg}$, ^{210}Po was eliminated in the feces at levels ten to twenty times higher than in the urine. Polonium chloride is poorly absorbed from the intestine after ingestion. In one experiment, the daily absorption through the skin was found not to exceed 2 percent of the amount applied.

Urine and fecal bioassays are required for monitoring of exposures since *in vivo* counting cannot detect less than 100 μCi and the maximum permissible body burden is 0.03 μCi . Urine radiochemistry can detect 0.1 pCi/l (ICRP, 1968).

Two physicists who inhaled ^{210}Po after the rupture of a polonium/beryllium source were observed to excrete ten times more ^{210}Po in the feces than in the urine (Foreman *et al.*, 1958). Another contamination accident that occurred in a university print shop resulted from the cleaning of a device to eliminate static; in this incident the operators incurred no significant radiation exposures (Caruthers and Maxwell, 1971). Another small exposure was reported to have been due to accidental inhalation of ^{210}Po during the handling of an encapsulated source (Scott and West, 1975). No treatment was required. Approximately 3 percent of the 0.015 μCi burden was excreted in the urine with a biological half-time of 33 days. The initial fecal to urine ratio of 65 dropped to 20 about 20 days post-exposure.

Dimercaprol (Section 7.3.5.4) has been suggested as a treatment (Hursh, 1951; 1952). When it was given intramuscularly after a single intravenous dose of ^{210}Po , the total excretion in a ten-day period was twice that of the control animals and the polonium was shifted from the bone marrow, spleen, and testes into muscle. When rats were injected with a lethal dose of ^{210}Po (36 $\mu\text{Ci}/\text{kg}$), the median survival time was 22 days, but when promptly treated with dimercaprol the median survival time was 89 days. The untreated animals died of hemopoietic failure, while much less effect was demonstrated on the white cell and platelet levels of treated animals. Russian investigators have reported some success with the use of dimercaprol derivatives in animal experiments, but the compounds are not available in the United States (Parfenov *et al.*, 1974; Erleksova, 1959).

Neither DTPA nor EDTA is effective in treating polonium internal contamination (Foreman *et al.*, 1958).

6.13 Radium

Radium, (Ra), element number 88, is a radioactive element that occurs in each of the major series of natural radionuclides and transuranic elements. Radium-226, a member of the decay chain of ura-

mium-238, has a physical half-life of 1620 years. It decays to radon-222 by emitting alpha particles of two energies [4.589 MeV (5.7 percent) and 4.777 MeV (94.3 percent)]. The ^{222}Rn then decays to polonium-218 followed by a series of active daughter elements that emit mixtures of alpha, beta, and gamma rays. The most important daughter products of Radium-226 are radon-222 (3.8-day physical half-life, alpha emitter, gaseous), bismuth-214 (20-minute physical half-life, alpha and gamma emitter), and lead-210 (22-year physical half-life, beta and gamma emitter).

Radium-226 is used as a radioactive source in medical practice and industry. Its primary value has been in the treatment of cancers by insertion of encapsulated needle sources directly into the tumor or by means of moulded applicators that hold the source next to the tumor. Industrial applications have included radium-beryllium neutron sources, radium for radiography, and luminous paints. The use of radium in all of these applications has been reduced greatly with the availability of safer and cheaper radioactive materials although many radium applications still exist.

After ingestion, about 30 percent of the radium-226 is absorbed (ICRP, 1960). Most of that absorbed is excreted within a few days after exposure; 95-98 percent is eliminated in the feces and 2-5 percent in the urine (Oberhausen, 1963). The radium remaining in the body is almost entirely deposited in the skeleton (Neuman *et al.*, 1955; Lloyd, 1961; Rowland, 1963). Norris *et al.* (1955) proposed that the retention of radium in the body can be described by a power function, $R_t = 0.54t^{-0.52}$ where R_t = amount of radium retained after the time t and t = time in days after injection. The effective half-life of radium is about 4.5 years for bone and 900 days for the whole body. About 65 percent of the ^{222}Rn formed as a decay product of radium in the body is exhaled (Oberhausen, 1963). This value is time dependent and the 65 percent value is for long times (years) after deposition. The expired air can be used in "radon breath analyses" for estimating the quantity of radium in the body. A review on the metabolism of ^{226}Ra in man has been published recently (Marshall *et al.*, 1973).

In the past, radium was the major radioisotope leading to serious levels of internal emitter exposure in man. Observations on this element have been the keystone to setting permissible burdens of other bone-seeking radionuclides (Evans, 1967). Ingestion of luminous paints containing radium occurred about the time of World War I and for several years thereafter as a result of workers pointing their brushes with their lips. The report of Martland *et al.* (1925) described the clinical effects of radium poisoning in the dial painters and led to the classic paper by Martland and Humphries (1929) on the development

of osteogenic sarcoma in 2 of 15 cases. This observation was the start of additional studies by Martland, R. D. Evans, and J. C. Aub of radiation oncogenesis due to radium.

In 1941, a task group assembled by the U.S. National Bureau of Standards selected 0.1 μg as a tolerance dose of ^{226}Ra for workers. The 0.1 μg , and, by definition, 0.1 μCi , of radium was that amount that could be contained in the body without clinical evidence of harm, and this residual body burden remains the standard maximum permissible body burden to the present time.

Several decades ago radium was used as a form of therapy in a group of patients in a state mental hospital (Miller and Finkel, 1968). Radium was also used in repeated doses for the treatment of hyperthyroidism (Loucks, 1930). Follow-up studies on nearly 300 radium dial painters over a period of many years revealed several types of malignant tumors, including osteogenic sarcoma, fibrosarcoma, carcinoma of the paranasal sinuses and mastoid, leukemia, and aplastic anemia (Hasterlik *et al.*, 1969; Finkel *et al.*, 1969). Cumulative mean skeletal radiation doses below 1000 rads were not associated with clinically significant radiobiological injury according to the data in the Massachusetts Institute of Technology series, which covers a time span of 40-50 years in more than 500 persons (Evans, 1974).

Immediate stomach lavage with a 10 percent magnesium sulfate solution is recommended in patients who have just ingested radium. This should be followed by daily saline purgatives with magnesium sulfate.

Little is known about the removal of ^{226}Ra once it is absorbed into the human body. Compounds that induce skeletal demineralization have been shown to increase the fecal excretion of ^{226}Ra (Aub *et al.*, 1938). This increased excretion of ^{226}Ra was induced by the concurrent use of ammonium chloride, thyroid extract, and parathyroid extract. The urinary ^{226}Ra excretion was only slightly increased. Each of these agents is a potent demineralizer that usually causes an increased excretion of the urinary calcium and this effect should be associated with an increase of excretion of ^{226}Ra in the urine. Since ammonium chloride is an effective demineralizing agent, this type of compound alone may be useful in increasing the urinary excretion of ^{226}Ra , but no data have been reported to support or deny this presumption. Orally administered calcium has only a slight effect on increasing the urinary ^{226}Ra excretion (Aub *et al.*, 1938; Spencer *et al.*, 1973a). One study showed that intravenously administered ACTH increases the urinary ^{226}Ra excretion but does not affect the fecal ^{226}Ra excretion (Spencer *et al.*, 1973a). In mice the excretion of ^{226}Ra could be increased by the use of sodium alginate (Humphreys *et al.*, 1972; Van der Borgh *et al.*,

1971) but no data are available on the effect of alginates on the intestinal absorption or excretion of ^{226}Ra in man.

6.14 Strontium

Six of the 16 radioisotopes of strontium, (Sr), element number 38, are direct fission products of uranium. By far the most important is ^{90}Sr because of its long physical half-life, 28 years. It decays by emitting a 0.54 MeV beta particle and gives rise to yttrium-90 which emits a 2.25 MeV beta particle. Strontium-89, an indirect fission product of uranium, has a physical half-life of 51 days and decays by emitting a 1.46 MeV beta particle and 0.009 percent of the time a 0.91 MeV gamma ray. Another important isotope of strontium is ^{85}Sr , which is produced by bombarding a ^{84}Sr target with neutrons to produce an $^{84}\text{Sr}(n,\gamma)^{85}\text{Sr}$ reaction. Its physical half-life is 65 days and it decays by orbital electron capture giving rise to $^{85\text{m}}\text{Rb}$, which immediately emits an 0.513 MeV gamma ray. With the shorter physical half-life and gamma emission, ^{85}Sr incorporation in the body results in a much lower absorbed dose for comparable activity than does ^{90}Sr incorporation.

Strontium-90 applied in the form of plaques is used for treatment of cutaneous lesions that are only a few millimeters in depth. Sources have been used in industry for thickness gauges by measuring the beta-ray backscatter from relatively thin sheets of paper, rubber, or metal. Strontium-90 has also been employed inappropriately in paints for luminous dials. Other industrial applications include sources used for static dust elimination by air ionization and ^{90}Sr titanate sources as compact heat sources. Large amounts are used for thermoelectric sources in buoys and similar devices where a long-lived, independent power source is needed.

Strontium-85 has been preferred to ^{90}Sr as a tracer in medical and agricultural research. Its principal use in nuclear medicine has been for study of the metabolism of strontium and for diagnostic bone scans.

Extensive studies have been carried out on the metabolism of radioactive strontium in animals and man because of the risks from the presence of ^{90}Sr in nuclear fallout due to atmospheric weapons tests and from the possible effect of escape of strontium into the biosphere during or after the reprocessing of used fuel elements. Information published by the ICRP (1968) suggests that after a single intake by mouth about one-quarter, and after inhalation one-third, of the radiostrontium taken in is absorbed into extracellular fluid and one-half of this is deposited in bone. Because of the high energy of the

beta particles emitted by the $^{90}\text{Sr} + ^{90}\text{Y}$ sequence, the ^{90}Sr deposited in bone irradiates both calcified bone and the adjacent bone marrow. Information on the potential hazards of ^{90}Sr in causing bone tumors, leukemia, and genetic effects are presented by Copp *et al.*, 1947; Nilsson, 1962; Van Putten and DeVries, 1962; Barnes *et al.*, 1970; Loutit, 1967; McClellan and Jones, 1969; Frolen, 1970; Nilsson, 1970; and UNSCEAR, 1977.

The biological half-time of ^{85}Sr in man was found to be less than 250 days during the first 160 days after a single ingestion (Furchner *et al.*, 1962). The average long-term retention in bone was estimated to be about 8.4 percent of the administered dose. The biological half-time determined by the use of intravenous tracer doses of ^{85}Sr averaged 843 days (Cohn *et al.*, 1962); the biological half-time after accidental inhalation of ^{90}Sr was estimated to be 500 days (Cowan *et al.*, 1952). The ICRP dose calculations use much longer half-times of about 50 years for bone and 36 years for the whole body (ICRP, 1960). The resultant effective half-life for ^{90}Sr is about 15 years.

The number of industrial or laboratory cases of accidental incorporation of ^{90}Sr into man is small. Several accidental ^{90}Sr inhalations have been reported (Cowan *et al.*, 1952; Rundo and Williams, 1961; Stewart *et al.*, 1958; Fisher and Kellehar, 1963; Bradley *et al.*, 1964; and Volf, 1963). In a case of accidental inhalation of $^{90}\text{SrCO}_3$ (Rundo and Williams, 1961), the ^{90}Sr body burden was estimated to be 0.364 μCi at 2 days after exposure. Accidental exposure to ^{90}Sr -titanate powder (Bradley *et al.*, 1964) resulted in an initial body burden estimated to be 5.2 μCi . However, several days after the accident the ^{90}Sr body burden had markedly decreased to 0.16 μCi . Two cases of accidental ^{90}Sr nitrate inhalation resulted in nasal smears containing 10^{-4} and 10^{-2} μCi ^{90}Sr (Volf, 1963).

Treatment was given in three of these ^{90}Sr inhalation exposures. Bradley *et al.* (1964) gave oral ammonium chloride and intravenous calcium. Volf (1963) treated two persons with orally administered barium sulphate. In none of these cases did the retained ^{90}Sr body burden exceed the maximum permissible body burden of 2 μCi ^{90}Sr .

Since ^{90}Sr that is ingested is eliminated mainly via the intestine (Spencer-Laszlo *et al.*, 1963; Spencer-Laszlo *et al.*, 1964), it is helpful to determine the fecal as well as the urinary excretions of ^{90}Sr after accidental exposures. In the case of ^{90}Sr -titanate inhalation (Bradley *et al.*, 1964), the fecal ^{90}Sr accounted for 94 percent of the total ^{90}Sr excretion. Volf (1963) found the largest amount of ^{90}Sr in feces 1 to 2 days after accidental inhalation of ^{90}Sr nitrate.

The amount of ^{90}Sr that has accidentally entered the human body can be estimated by a simple method in which only a single 24-hour

measurement of the urinary excretion of ^{90}Sr and of calcium is needed (Samachson and Spencer, 1965). Data obtained with tracer doses of ^{90}Sr in man (Spencer-Laszlo *et al.*, 1963; Samachson and Spencer-Laszlo, 1962; Spencer *et al.*, 1960) were used to determine various factors based on the time after exposure up to 12 days and the 24-hour calcium excretion in urine for the individual. The radiostrontium 24-hour urinary excretion, multiplied by the appropriate factor, gives a reasonable estimate of the radiostrontium absorbed.

Several methods of treatment can be used to reduce the amount of radiostrontium absorbed from the intestine and to assist in its removal. Immediately after ingestion, aluminum phosphate (Section 7.3.2.7) can reduce absorption of radiostrontium as much as 85 percent (Spencer *et al.*, 1967; 1969a; 1969b) and is considered the drug of choice. Barium sulfate (Section 7.3.2.9) is a risk-free alternative drug that also causes significant reduction of intestinal absorption (Volf, 1963). Sodium alginate (Section 7.3.2.8) inhibits the absorption of acutely ingested radiostrontium, but its heavy viscosity makes it difficult to administer.

After absorption of radiostrontium has occurred, several compounds of stable strontium (Section 7.3.3.3) can be used as isotopic diluting agents to reduce uptake of radiostrontium. They must be given shortly after exposure. Large doses of calcium (Section 7.3.3.6) orally or intravenously will increase excretion rates and may be used as a substitute if strontium drugs are not available. The above treatments should be combined with administration of oral ammonium chloride to achieve maximum effect (Section 7.3.4.3).

6.15 Technetium

Of the isotopes of Technetium, (Tc), element number 43, ^{99m}Tc , is used more frequently in nuclear medicine now than the radioiodine isotopes. It was selected and introduced because of its suitability for diagnostic scanning. Its gamma ray (0.14 MeV), emitted as it decays to technetium-99, is easily detected with a collimated detector at any body surface. Its short physical half-life, 6.0 hours, makes the radiation dose very small for each use in diagnostic procedures.

Technetium-99m results from the decay of molybdenum-99, which has a 67-hour physical half-life and decays by emitting several beta particles of energy ranging from 0.45 to 1.23 MeV plus seven gamma rays with energies of 0.04 to 0.95 MeV. The ^{99m}Tc then decays by isomeric transition to ^{99}Tc which has a physical half-life of 212,000 years. The ingrowth and decay of the ^{99}Tc is of no concern because of the low specific activity resulting from its long physical half-life.

Technetium, at least as the pertechnetate, has a very short biological half-time, only 1 day for the whole body, 20 days in the kidney, and up to 30 days in the liver. After intravenous administration, approximately 30 percent of the ^{99m}Tc -pertechnetate is excreted in the urine over the first 24 hours. Animal studies have shown that pertechnetate ion is selectively concentrated in the thyroid gland, salivary glands, and stomach (Andros *et al.*, 1965).

Pharmaceutically prepared solutions and suspensions of ^{99m}Tc compounds can be obtained, but loss of activity between manufacture, dispatch, and use is a major disadvantage. Development of an aluminum hydroxide absorption column loaded with ammonium molybdate-99 solved the transport problem. Nuclear medicine laboratories now order a column every week or two and elute its ^{99m}Tc in the form of pertechnetate as needed. The ^{99m}Tc must then be converted into various chemical forms suitable for specific diagnostic tests. It is used in a great many chemical forms such as the pertechnetate.

There have been no reports of misadministrations or accidents with ^{99m}Tc that caused recognizable effects, probably because the radiation doses from millicurie amounts are so small. There have been instances of misadministrations due to confusion of the millicurie with the microcurie. In one instance, a molybdenum-99 charged alumina column was assembled in the inverted position, in spite of explicit instructions to the contrary, with the result that patients received chiefly ^{99}Mo instead of ^{99m}Tc . No effects were observed. It seems unlikely that accidental exposures to ^{99m}Tc will require treatment, but steps must be taken to be sure that the activity detected is due to ^{99m}Tc and not some other radioisotope, e.g., radioiodine, for which immediate treatment might be indicated. Exposures to the very long-lived ^{99}Tc , such as might occur during nuclear fuel processing or waste storage, seem to have low hazard potential. The effects of long standing body burdens, however, have not been examined by animal experimentation.

Administration of potassium perchlorate (KClO_4) is effective in displacing pertechnetate-99m ions from sites of concentration in the thyroid gland, salivary glands, and stomach (Andros *et al.*, 1965).

6.16 Thorium

Thorium-232, with a physical half-life of 1.39×10^{10} years, is the starting element for a decay series that ends with ^{208}Pb . The series is analogous to that of ^{238}U except that the daughters have short physical half-lives, with two exceptions, (R^{226}Ra -6.5 y and ^{228}Th -1.94), and consequently, relatively larger amounts of ionizing radiation are deliv-

ered to the tissue surrounding a particle. Thorium, (Th), element number 90, is used in ceramic glazes, optical glass, gas mantles, tungsten welding electrodes, and various metal alloys.

Thorium-232 was used formerly in the chemical form of $^{232}\text{ThO}_2$ as a radioopaque substance, ideal for angiography. The preparation sold under the trade name "Thorotrast" was a 25 percent suspension of finely dispersed ThO_2 . It contained varying amounts of daughter radioisotopes and, consequently, was more radioactive than it would have been had the thorium ore been refined to yield pure ^{232}Th for preparing the $^{232}\text{ThO}_2$.

Since Thorotrast was a fine particulate, the reticulo-endothelial cells of the liver, spleen, and marrow as well as hepatic cells phagocytized it quickly. Enhanced contrast, especially of fine vessels, can be obtained with Thorotrast but the risk of hepatomas, angiosarcomas, osteosarcomas, and subpleural mesotheliomas caused Thorotrast to be supplanted by other radioopaque media.

No effective treatment is available to modify the distribution or absorption of injected Thorotrast. For other thorium forms, the chelating agents DTPA (Section 7.3.5.3) or DFOA (Section 7.3.5.6) may enhance excretion but are not likely to be sufficiently effective to warrant long-term therapy.

6.17 Tritium (Hydrogen-3)

Tritium is the only radioactive isotope of hydrogen, (H), element number 1. It decays to ^3He by emitting a beta particle with a maximum energy of 18 keV and an average energy of 5.7 keV. Its half-life is 12.3 years. Small amounts of the isotope are present normally in the atmosphere and biosphere, being produced by cosmic-ray reactions with nitrogen in the stratosphere and also from the spontaneous fission of elements on the surface of the earth. Atmospheric weapons testing has increased the tritium present in the environment. Before the nuclear age, water contained a ratio of 1 tritium atom per 10^{18} hydrogen atoms but this is now about 10 to 100 per 10^{18} hydrogen atoms.

Tritium has not been widely used in clinical medicine, probably because of the difficulty in detecting its weak beta particles. Experimental uses include total body water measurements and the *in vivo* labeling of proliferating cells by injection of tritium-labeled thymidine. Tritium labeling is also used in a variety of metabolic tracer studies. When it is incorporated in chemical compounds, the distribution and retention of that tritium in the body can be influenced markedly. Tritium is used as a target material in accelerators for production of

fast neutrons. It is also used as a radiation source in thickness gauges and is finding much commercial use in making luminous paints (Lambert and Vennart, 1972). It can be used to trace the movement of water in soils.

The absorption, and therefore the hazard, of tritium inhaled in air is much less when it is present as elemental tritium than as tritiated water, HTO. This is recognized in the maximum permissible concentration limits for a 40-hour work week, which are $5 \times 10^{-6} \mu\text{Ci}/\text{cm}^3$ ($5 \mu\text{Ci}/\text{m}^3$) for tritiated water and $2 \times 10^{-3} \mu\text{Ci}/\text{cm}^3$ ($2000 \mu\text{Ci}/\text{m}^3$) for molecular or gaseous tritium in air. The oxidation of tritium gas in air is usually slow, less than one percent per day, unless burning occurs. Tritium penetrates the skin, lungs, and gastrointestinal tract, either as tritiated water or in the gaseous form. As gaseous hydrogen, tritium is not significantly absorbed into the body and does not exchange significantly with the hydrogen in body compounds. As water the tritium entering the lungs or gastrointestinal tract is completely absorbed, and is rapidly dispersed throughout the body. Radiological control requires periodic biological monitoring of workers through measurements of tritium in urine (Lambert and Vennart, 1972).

As soon as a probable acute exposure is recognized, the individual should void his urine as completely as possible. The first sample must be saved as a control against the possibility of previous unrecognized exposures. The next, and subsequent voidings, are used to measure the tritium excretion. A rough rule of thumb, based on the peak urine concentration after a single acute exposure, is that $1 \mu\text{Ci}/\text{l}$ of tritium in urine is indicative of a total integrated whole-body dose of about 10 mrem in the average person, if no treatment is instituted. Tritium exposures are comparable in their biological effect to whole-body exposure to external x or gamma radiations.

Tritium contamination of surfaces, including skin, cannot be measured except by special survey instruments because of the low energy of the beta particles. A special survey instrument has been developed to estimate the amount of tritium deposited in the body based on measurements made over the skin, such as the forearm (Powell *et al.*, 1977). While these data are less accurate and less sensitive than the urine assay, this monitor system may be useful as an early indicator of tritium exposure. Counting of surface smears in a gas-flow proportional counter is the usual method for detecting surface contamination. Air concentrations can be detected by air ionization chambers called "sniffers". The HTO content of air can be sampled by absorbing or freezing out the water. The residue is then measured in a liquid scintillation counter. Techniques for measuring tritium are described in NCRP Report No. 47 (NCRP, 1976).

Health effects in man have not been reported from single acute

exposures to tritium. There have been many instances of single exposure that were treated by forcing fluids as soon as possible. This has the dual value of diluting the tritium and physiologically increasing its excretion. Five instances have been reported of repeated exposures over periods of one to seven years to multicurie doses of tritium during preparation of batches of tritiated luminous compounds (Seelentag, 1973; Minder, 1969). Urine concentrations of tritium varied from 0.1 to 40 times the maximum permissible values throughout the periods of exposure according to the limited data that are available. The two men working most closely with the chemical preparation developed symptoms of nausea, lassitude and exhaustion, and a progressive anemia combined with a poorly defined physical deterioration that ended with aplastic panmyelocytopenia and death. A clear statement in these cases is difficult because both men at one time or another had worked with other radionuclides, received medication such as steroids, antibiotics, and transfusions, or worked in a manufacturing process involving polymeric plastics and organic solvents.

Between batches and when taken off the job, each man showed two- or three-component exponential decreases of tritium concentration in the urine with time. Tissue samples from the two men that died had specific activities of tritium bound to dried tissues (presumably organically-bound tritium) that were six to twelve times higher than concentrations in the urine samples. Thus, some of the tritium is incorporated into cellular components and has a turnover rate of several hundred days (Moghissi *et al.*, 1971; 1972). This long-term component, however, contributes only about 2 percent of the total dose (Snyder *et al.*, 1968).

The biologic half-time is about 10 to 12 days. Forcing fluids to tolerance, at least 3 to 4 liters per day, will reduce the half-time to about $\frac{1}{3}$ to $\frac{1}{2}$ of the normal value (Section 7.3.3.5). The dose is reduced proportionately to the effective half-life. Daily urine samples analyzed for tritium can be used to judge the effectiveness of the treatment by calculating the resultant effective half-life.

6.18 Uranium

Uranium, (V), element number 92, occurs only in radioactive form. Natural uranium (U-nat) is a mixture of ^{238}U , (~99.3 percent), ^{235}U (~0.7 percent), and ^{234}U (~0.006 percent). Uranium-238 is the head of the uranium/radium series and ^{235}U starts the uranium/actinium series. Uranium isotopes are also found in other series of transuranic

elements. The isotopes of U-nat have extremely long physical half-lives: 4.5×10^9 years for ^{238}U , 7.1×10^8 years for ^{235}U , and 2.5×10^5 years for ^{234}U . U-nat and its daughter products emit alpha, beta, and gamma radiations.

Each of the three isotopes of U-nat also undergoes spontaneous fission at low rates. These are well below criticality even with the pure metal, but evidence of fission is found in the emission of fast fission neutrons and the presence of fission product isotopes. The daughter elements include two noble gases, radon-222 and radon-219; a third, radon-218, occurs in very low frequency and has no biological significance. These gaseous radionuclides are released in uranium and other mines and decay then to alpha- and beta-emitting isotopes of polonium, bismuth, thallium, astatine, and lead. The radon and the radon daughters adhere to atmospheric dust particles and constitute a serious inhalation hazard.

Most exposures to uranium and its daughters have occurred during the mining, processing, and fabrication of uranium into fuel elements for nuclear reactors or weapons. During this process, the uranium exists in several different physical states and chemical compounds. Raw ores contain from 0.1 percent to 1.0 percent uranium, chiefly U_3O_8 . During the milling operation, the ore is concentrated, leached, and processed to ammonium diuranate and U_3O_8 , a mixture called "yellowcake". The oxide is converted to UF_6 by fluorination and then processed through a gaseous diffusion plant, where the ^{235}U content is enriched to the required level for use as fuel elements for nuclear reactors. Enrichment to above 90 percent is possible for use in research and experimental reactors or nuclear weapons. The enriched UF_6 is converted to UO_2 and formed into pellets for manufacture of fuel elements. Standard metallurgical processing converts the oxide into the dense, silvery white, uranium metal. When fuel is reprocessed the uranium is dissolved in nitric acid, the fission products and trans-uranium elements are removed, and the uranium then is reprocessed to UF_6 for recycling.

Explosion and fire are the hazards associated with the handling of uranium. At room temperature, finely divided uranium metal may ignite spontaneously in air, oxygen, and even water (Wilkinson, 1962). The rapid oxidation of uranium under suitable conditions may cause a chemical explosion. The lower explosive limit for suspended uranium metal dust is 55 mg/l (Gindler, 1973).

A practical classification of the various uranium compounds is useful because differences in their absorption, transport within the body, deposition, and excretion affect their toxicity. Three levels of biologic mobility, or "transportability", for inhaled uranium have been devel-

oped (Scott, 1973). This term refers to the rate at which inhaled materials leave the lung without regard for the route of the movement. It is determined primarily by solubility but is also influenced by particle size. Highly transportable compounds take from weeks to months; moderately transportable, months; and slightly transportable from months to years. Table 6.2 lists this classification of uranium compounds.

In addition to transportability, the isotopic composition of the uranium must be considered in determining the hazard from different radiation properties. In uranium compounds enriched in ^{235}U to less than 5 to 8 weight percent and not irradiated in a reactor, the chemical toxicity is probably the limiting factor (Ford, 1964). In uranium enriched in ^{235}U to more than 8 weight percent or after irradiation, the radiation hazard predominates.

Uranium thus is considered either a chemical or radiologic hazard depending on its isotopic composition and its radiation history. With U-nat, the total quantity of metal absorbed is the determinant regardless of the compounds involved. One to five percent of an oral dose is absorbed and most is excreted by the kidneys (Hursh and Spoor, 1973). In acute or sub-acute poisonings, the kidney is the first organ to show chemical damage in the form of nephritis and proteinuria. Renal damage by U(VI) ion is produced by concentrations of the order of 0.1 mg/kg body weight. Uranium absorbed into the systemic circulation is eliminated principally via urine, about 60 percent of U(VI) and 20 percent of U(IV) within the first 24 hours. Fixation in the skeleton also occurs rapidly with about 8 percent of uranyl salts [U(VI)], but less than 1 percent of uranous salts [U(IV)], being retained in the bone (Simon, 1972). Since soluble uranium is eliminated so rapidly from the body, only a urine sample collected during or immediately after exposure provides a reliable index of the extent of the exposure.

The critical organ for less soluble compounds of uranium, especially when enriched with ^{235}U and ^{234}U , is likely to be the bone, or when

TABLE 6.2—Classification of uranium compounds according to transportability from the lung after inhalation (Scott, 1973)

Highly transportable (weeks to months)	Moderately transportable (months)	Slightly transportable (months to years)
UF_6	UO_2	UO_2
UO_3	U_3O_8	U_3O_8
$\text{UO}_2(\text{NO}_3)_2$	UO_3	Uranium oxides
UF_4	UF_4	Uranium hydrides
Uranium sulfates	Uranium nitrates	Uranium carbides
Uranium carbonates		Salvage ash

inhaled, the lung. The biological half-time is usually considered to be 300 days for the bone, 100 days for the whole body, and 15 days for the kidney (ICRP, 1960). In cases of ingestion, the slightly transportable compounds, the oxides and carbides, are eliminated principally via the feces. After inhalation, the retention depends on particle size and chemical form. Long biological half-times of 120 days or more can be anticipated from slightly transportable compounds having particle sizes under 2 micrometers. Although the nominal biological half-time for uranium in the lung is 120 days (ICRP, 1959), the uranium oxides have biological half-times of up to 1470 days (West and Scott, 1969).

Uranyl nitrate, uranyl fluoride, uranium pentachloride, uranium trioxide, sodium diuranate, and ammonium diuranate are absorbed through the skin of experimental animals. Several of these compounds also cause mild to moderate skin irritation. Contamination of the eyes can be a serious hazard since extensive absorption via the cornea occurs rapidly.

Exposures of about 0.1 mg/kg or more of soluble U-nat results in chemical injury of the cells of the lower portion of the proximal convoluted tubule of the kidney (Luessenhop *et al.*, 1958). There is usually a lag period of 6 hours to several days followed by necrosis (Yuile, 1973). Mild glomerular damage also develops with signs such as albuminuria, hematuria rarely, and hyaline and then granular casts in large numbers (Stone *et al.*, 1961). Urinary catalase is usually elevated. Other renal function tests, especially those that measure tubular excretory capacity, may be abnormal and azotemia may occur after a severe exposure. However, even after exposure to levels that cause necrosis, the kidney shows evidence of regeneration within 2 to 3 days, depending on the severity of the initial injury. The injured kidney, upon recovery, usually shows evidence of a tolerance to subsequent exposures at the same or higher levels. This tolerance does not develop unless the kidney has been sufficiently injured to require regeneration of tubular epithelium.

A number of the reported accidental exposures to soluble uranium compounds have been reviewed by Hursh and Spoor (1973). Most of the exposures were by inhalation of UF_6 that leaked from enclosed systems. One accident (Howland, 1953) resulted in the death of two employees and serious injury to three others. The deaths and injuries were caused by the hydrofluoric acid that was released when steam reacted with UF_6 ; this reaction accounts for the highly corrosive features of the exposure. In another accident (Boback and Heatherton, 1966), an employee inhaled 13 mg of uranium as UF_6 but recovered after several days' hospitalization; he showed evidence of only transient kidney damage.

In view of extensive industrial experience, it appears that natural uranium is less toxic to man than was expected on the basis of animal experiments. There has been no evidence of chronic chemical toxicity after years of exposure to low levels (Scott *et al.*, 1970).

Because of the rapid excretion of soluble uranium compounds, the primary value of routine urinalyses for uranium is to provide a general guide as to the effectiveness of exposure control in a group of workers in a specific area of a plant. A guide to the various types of samples and where and when they should be collected has been published (Scott, 1973).

When a single large exposure has occurred, continuous measurement of the urinary excretion of uranium is recommended, especially when the solubility of the compound is known or can be estimated. *In vivo* counting of the 186 keV gamma from ^{235}U , however, is superior to urinalyses in evaluating body burdens and is the best method available to assess the inhalation of insoluble material. Care must be taken when evaluating exposures to uranium enriched in its content of ^{235}U because the ^{234}U content is also increased. When the enrichment is above a few percent, much of the radiation comes from ^{234}U because of its shorter half-life and higher specific activity. It is necessary to determine the percent ^{234}U and the specific activity of the material because the ^{234}U content varies with its processing history (Scott, 1973).

Treatment with sodium bicarbonate produces a uranyl bicarbonate complex in tubular urine that is less nephrotoxic; it also promotes migration to extracellular fluids and deposition in the bone. Oral doses or infusions of sodium bicarbonate are regulated so as to keep the urine alkaline as determined by frequent pH measurements. Use of a diuretic drug (Section 7.3.4.4) has been advocated (ICRP, 1978). If the chemical toxicity becomes severe enough, renal dialysis would presumably be useful since the damage of the proximal convoluted tubule is temporary and recovery is fairly rapid.

Both EDTA and DTPA have been used in experimental animals (Catsch, 1964) to increase excretion. DTPA increased the LD_{50} in mice injected intraperitoneally with uranyl nitrate in doses of 6.7 mg U/kg to 16.2 mg U/kg (Catsch, 1959). The effectiveness of both chelating drugs is strongly time dependent and no protective action is observed with a delay of 4 hours or more after exposure (Catsch and Harmuth-Hoene, 1979).

URANIUM

Source: ATSDR, 1990. Toxicological Profile for Uranium. Agency for Toxic Substances and Disease Registry, Atlanta, GA. December, 1990.

1. PUBLIC HEALTH STATEMENT

This Statement was prepared to give you information about uranium and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1177 sites on its National Priorities List (NPL). Uranium has been found above background levels at 26 of these sites. However, we do not know how many of the 1177 NPL sites have been evaluated for uranium. As EPA evaluates more sites, the number of sites at which uranium is found may change. The information is important for you because uranium may cause harmful health effects and because these sites are potential or actual sources of human exposure to uranium.

When a chemical is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment as a chemical emission. This emission, which is also called a release, does not always lead to exposure. You can be exposed to a chemical only when you come into contact with the chemical. You may be exposed to it in the environment by breathing, eating, or drinking substances containing the chemical or from skin contact with it.

If you are exposed to a hazardous substance such as uranium, several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health.

1.1 WHAT IS URANIUM?

Natural uranium is a silver-colored metal that is radioactive. Small amounts of uranium are present in rocks, soil, water, plants, and animals and contribute to the weak background radiation from these sources. Soil commonly contains variable amounts, but the average is about 2 parts uranium per million parts of soil (2 ppm). This is equivalent to a tablespoon of uranium in a truckload of dirt. Fertilizers made from phosphate rocks contain higher amounts of uranium than natural soils. Some rocks and minerals in underground and open pit mines also contain uranium in a more concentrated form. After these rocks are mined, uranium is extracted and chemically converted into uranium dioxide or other usable forms. The remaining rock from which uranium has been extracted is called depleted ore or mill tailings.

Natural uranium is composed of three forms (called isotopes) of uranium: uranium-234, uranium-235, and uranium-238. The amount of uranium-238 in natural uranium is more than 99%. Uranium-235 is present at just 0.72% in natural uranium, but it is more radioactive (and therefore more hazardous) than uranium-238. Uranium-235 is used in nuclear bombs and nuclear reactors. An industrial process by which the percent of uranium-235

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is concentrated is called enrichment, and the uranium obtained this way is called enriched uranium. Uranium-234 is even less abundant than uranium-235, so it can be ignored for most practical purposes.

Uranium-238 is not stable but breaks down into two parts. This process of breaking down is called decay. The decay of uranium-238 produces a small part called "alpha" radiation and a large part called the decay product. The break down of uranium-238 to its decay products happens very slowly. In fact, it takes about 4.5 billion years for one-half of the uranium-238 to break down (4.5 billion years is the half-life of uranium-238). Thorium, the decay product of uranium, is also not stable, and it continues to decay until stable lead is formed. During the decay processes, the parent uranium-238, its decay products, and their subsequent decay products release a series of new elements and radiation, including such elements as radium and radon, alpha and beta particles, and gamma radiation. Alpha particles cannot pass through human skin, whereas, gamma radiation passes through more easily.

Because of the slow rate of decay, the total amount of natural uranium in the earth stays almost the same, but it can be moved from place to place through natural processes or by human activities. When rocks are broken up by water or wind, uranium becomes a part of the soil. When it rains, the soil containing uranium can go into rivers and lakes. Mining, milling, manufacturing and other human activities also move uranium around natural environments.

We use uranium mainly in nuclear power plants and nuclear weapons. Very small amounts are used in making some ceramics, light bulbs, photographic chemicals, and household products. For more information on the properties and use of uranium, see Chapters 3, 4, and 5.

1.2 HOW MIGHT I BE EXPOSED TO URANIUM?

Since uranium is found nearly everywhere, you can be exposed to it in the air, water, food and soil. We know, roughly, the average amounts of uranium in food [0.08 to 70 micrograms per kilogram ($\mu\text{g}/\text{kg}$)] and drinking water [0.4 to 1.4 micrograms per liter ($\mu\text{g}/\text{L}$)] (1 microgram = 1/100,000th of 1 gram). Most people in the United States take in some uranium with their food every day. Root vegetables, such as beets and potatoes, tend to have a little more uranium than other foods. In a few places, the concentration of uranium is higher in the water than in the food. People in these areas take in more uranium from their drinking water than from their foods. Your daily intake of uranium may be greater than average if you live near uranium mines or processing plants or an uncontrolled waste site containing uranium, eat food grown in contaminated soil, or drink water that contains unusually high levels of uranium. Normally, very little of the uranium in lakes, rivers, or oceans gets into the fish or seafood we eat. The amount in air is usually so small that it can be safely ignored. However, people who work at factories that process uranium, work with phosphate fertilizers, or live

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near uranium mines have a greater chance of being exposed to uranium in the air than most other people. Larger-than-normal amounts of uranium might also enter the environment from accidental discharges from uranium processing plants. For more information on the potential for exposure to uranium, see Chapter 5.

1.3 HOW CAN URANIUM ENTER AND LEAVE MY BODY?

If you were to breathe in uranium dust, most of it would leave the lungs when you cough or breathe out. However, you might swallow some of the uranium you breathe in as your body removes the uranium from your lungs. Some of the uranium in your lungs will enter your blood, pass through the kidneys, and be eliminated in the urine within a few days. A small amount may stay in your lungs for years.

Since uranium is present all over the earth, everyone normally eats or drinks a small amount of uranium daily. When it enters your body this way, about 99% of it leaves within a few days in your feces and never enters your blood. A small amount of uranium (about 1%) will enter the blood. Most of this will pass through the kidney and be eliminated in the urine in a few days. A small amount goes to your bones and may stay in your bones for years. A very small amount, about 1/5000th of the weight of an aspirin tablet, is found in most people, mainly in their bones.

Although uranium is radioactive, the type of radiation it gives off cannot go through your skin, so natural uranium that is outside the body is not hazardous. When uranium gets inside your body, after breathing it in, eating or drinking it, or through cuts in your skin, radiation and chemical toxicity are of concern to health. For more information, see Chapter 2.

1.4 HOW CAN URANIUM AFFECT MY HEALTH?

We do not know for certain if natural uranium is dangerous to human health, although evidence of kidney effects were seen in people who work in uranium mines. Animals have developed kidney disease after they have been exposed to large amounts of natural uranium in the food, in the drinking water, in the air, or on the skin.

There is always a concern about getting cancer from any radioactive material. Natural uranium has very low levels of radioactivity and has not definitely been shown to cause cancer in humans or animals. Nevertheless, it is possible that you could develop cancer from swallowing or breathing large amounts of natural uranium because the greater your exposure to a radioactive material, the greater your chance of developing cancer. This is particularly true for enriched uranium that has been made more radioactive. Cancer may develop many years after swallowing or breathing a radioactive material. Just being near natural uranium is of very little danger to your health because most of the radiation given off by uranium cannot go through your skin.

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We do not know if natural uranium causes reproductive effects or birth defects in humans, but animal studies suggest that uranium may affect reproduction and the developing fetus. See Chapter 2 for more information on the health effects associated with uranium.

1.5 WHAT LEVELS OF EXPOSURE HAVE RESULTED IN HARMFUL HEALTH EFFECTS?

Small amounts of uranium are always in your body and these amounts are not known to affect your health. Some uranium miners have developed lung cancer. This cancer is not from the uranium itself, but from the high levels of radioactive radon gas, which is formed when uranium decays. For more information about radon, see the ATSDR Toxicological Profile on Radon.

Animals that ate food, drank water, or breathed air that had high levels of uranium dust have developed kidney damage. The extent of kidney damage depends on how much uranium gets into their bodies and on the chemical form to which the animals are exposed. Animals can eat or breathe large amounts of some forms of uranium without having any health problems at all.

Tables 1-1 through 1-4 show the relationship between uranium and known health effects. See Chapter 2 for more information.

1.6 IS THERE A MEDICAL TEST TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO URANIUM?

There are medical tests that can be performed to determine the amount of uranium in your urine and feces. If you are exposed to a larger-than-normal amount of uranium, some uranium may appear in your urine and feces. Since most uranium leaves the body in the feces within a few days, the amount in the feces only shows whether you have been exposed to a larger amount than normal within the last week or so. Uranium can be found in your urine for up to several months after exposure. The amount of uranium in your urine and feces does not always accurately show how much uranium you were exposed to.

Since uranium is known to cause kidney damage in humans and animals, urine tests can be used to see if you have kidney damage that may have been caused by exposure to uranium. Some of these tests include measuring the amount of protein, sugar, or enzymes in the urine, or detecting the presence of damaged kidney cells in the urine. These tests, however, are not specific for uranium and are only useful to determine if kidney damage has occurred.

If you breathe large amounts of radioactive uranium, the amount of radioactivity in your body can be measured by a special test. This test is only useful if you have been exposed to certain types of uranium that stay

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TABLE 1-1. Human Health Effects from Breathing Uranium*

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term human exposure to air containing specific levels of uranium are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Air</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term human exposure to air containing specific levels of uranium are not known.

*See Section 1.2 for a discussion of exposures encountered in daily life.

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TABLE 1-2. Animal Health Effects from Breathing Uranium

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Air (mg/m³)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
630	10 min	Slight kidney damage in rats.
12,000	10 min	Death in rats.
18,000	2 min	Slight kidney damage in guinea pigs.
62,000	2 min	Death in guinea pigs.
Long-term Exposure (greater than 14 days)		
<u>Levels in Air (mg/m³)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
0.05	1 year	Slight kidney effects in rats.
0.20	7.5 months	Kidney damage in guinea pigs.
0.25	6.5 months	Kidney damage and death in rabbits.
0.25	1 year	Kidney damage and death in dogs.
5.0	5 years	Lung damage in monkeys.

*These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

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TABLE 1-3. Human Health Effects from Eating or Drinking Uranium*

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from short-term human exposure to food containing specific levels of uranium are not known.
<u>Levels in Water</u>		The health effects resulting from short-term human exposure to water containing specific levels of uranium are not known.
Long-term Exposure (greater than 14 days)		
<u>Levels in Food</u>	<u>Length of Exposure</u>	<u>Description of Effects</u>
		The health effects resulting from long-term human exposure to food containing specific levels of uranium are not known.
<u>Levels in Water</u>		The health effects resulting from long-term human exposure to water containing specific levels of uranium are not known.

*See Section 1.2 for a discussion of exposures encountered in daily life.

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TABLE 1-4. Animal Health Effects from Eating or Drinking Uranium

Short-term Exposure (less than or equal to 14 days)		
<u>Levels in Food (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
9480	1 dose	Rats had fewer pups.
<u>Levels in Water (ppm)</u>	Days 6-15 of pregnancy	Deformities in the pups of mice and weight loss in the mothers.
16		
716	1 dose	Death in mice.
820	1 dose	Death in rats.
Long-term Exposure (greater than 14 days)		
<u>Levels in Food (ppm)</u>	<u>Length of Exposure</u>	<u>Description of Effects*</u>
94	30 days	Kidney damage in rabbits.
469	30 days	Death in rabbits.
1940	2 years	Death in rats.
2315	48 weeks	Death in mice.
<u>Levels in Water (ppm)</u>	8-14 weeks (during and after pregnancy)	Decreased weight of pups in mice.
16		
21	Day 13 of pregnancy to day 21 of nursing	Maternal death in mice.
64	4 weeks	Kidney and liver damage and blood effects in rats.
471	4 months	Damage to the testes in rats.

*These effects are listed at the lowest level at which they were first observed. They may also be seen at higher levels.

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in the lungs for a long time, or to enriched uranium that is more radioactive than normal. See Chapters 2 and 6 for more information.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The EPA states that long-term exposure to 0.003 milligrams of uranium/kilogram of body weight/day in the food or drinking water is safe for humans. This value is for compounds of uranium that dissolve easily in water. EPA requires industries to report discharges of more than 0.1 curie for most uranium isotopes, including uranium-238, and to report spills of 100 pounds or more of two uranium compounds, uranyl nitrate and uranyl acetate. Uranium levels in the workplace are regulated by the Occupational Safety and Health Administration (OSHA) and recommended by the National Institute for Occupational Safety and Health (NIOSH). Both organizations set the occupational exposure limit for an 8-hour workday, 40-hour workweek at 50 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) for uranium compounds that dissolve easily in water. The limits for compounds that do not dissolve easily in water are 200 $\mu\text{g}/\text{m}^3$ (OSHA) and 250 $\mu\text{g}/\text{m}^3$ (NIOSH). See Chapter 7 for more information.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns not covered here, please contact your State Health or Environmental Department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road, E-19
Atlanta, Georgia 30333

This agency can also give you information on the location of the nearest occupational and environmental health clinics. Such clinics specialize in recognizing, evaluating, and treating illnesses that result from exposure to hazardous substances.



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