APPENDIX A

ATSDR MINIMAL RISK LEVEL AND WORKSHEETS

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) [42 U.S.C. 9601 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 99–499], requires that the Agency for Toxic Substances and Disease Registry (ATSDR) develop jointly with the U.S. Environmental Protection Agency (EPA), in order of priority, a list of hazardous substances most commonly found at facilities on the CERCLA National Priorities List (NPL); prepare toxicological profiles for each substance included on the priority list of hazardous substances; and assure the initiation of a research program to fill identified data needs associated with the substances.

The toxicological profiles include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations of a hazardous substance. During the development of toxicological profiles, Minimal Risk Levels (MRLs) are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean-up or action levels.

MRLs are derived for hazardous substances using the no-observed-adverse-effect level/uncertainty factor approach. They are below levels that might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs are derived for acute (1–14 days), intermediate (15–364 days), and chronic (365 days and longer) durations and for the oral and inhalation routes of exposure. Currently, MRLs for the dermal route of exposure are not derived because ATSDR has not yet identified a method suitable for this route of exposure. MRLs are generally based on the most sensitive chemical-induced end point considered to be of relevance to humans. Serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) are not used as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur.

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MRLs are intended only to serve as a screening tool to help public health professionals decide where to look more closely. They may also be viewed as a mechanism to identify those hazardous waste sites that are not expected to cause adverse health effects. Most MRLs contain a degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive (e.g., infants, elderly, nutritionally or immunologically compromised) to the effects of hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address this uncertainty consistent with the public health principle of prevention. Although human data are preferred, MRLs often must be based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive to the effects of hazardous substance than animals and that certain persons may be particularly sensitive. Thus, the resulting MRL may be as much as a hundredfold below levels that have been shown to be nontoxic in laboratory animals.

Proposed MRLs undergo a rigorous review process: Health Effects/MRL Workgroup reviews within the Division of Toxicology, expert panel peer reviews, and agencywide MRL Workgroup reviews, with participation from other federal agencies and comments from the public. They are subject to change as new information becomes available concomitant with updating the toxicological profiles. Thus, MRLs in the most recent toxicological profiles supersede previously published levels. For additional information regarding MRLs, please contact the Division of Toxicology, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road, Mailstop E-29, Atlanta, Georgia 30333.

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MRL WORKSHEET

Chemical Name: Beryllium and Compounds
CAS Number:
Date: May 16, 2002
Profile Status: Third Post Public Comment Draft
Route: [ ] Inhalation   [X] Oral
Duration: [ ] Acute   [ ] Intermediate   [X] Chronic
Key to Figure: 11
Species: Dogs

Minimal Risk Level: 0.002   [X] mg beryllium/kg/day   [ ] ppm

Reference: Morgareidge K, Cox GE, Gallo MA. 1976. Chronic feeding studies with beryllium in dogs. Food and Drug Research Laboratories, Inc. Submitted to the Aluminum Company of America. Alcan Research and Development, Ltd., Kawecki-Berylco Industries, Inc., and Brush-Wellman, Inc.

Experimental design: (human study details or strain, number of animals per exposure/control groups, sex, dose administration details): Groups of five male and five female Beagle dogs were fed a diet containing 0, 5, 50 or 500 ppm beryllium as beryllium sulfate tetrahydrate (0, 0.1, 1, and 12 mg beryllium/kg/day for males and 0, 0.2, 1, and 17 mg beryllium/kg/day for females) for 172 weeks. The basal diet was a commercial dog chow (Purina®) moistened with warm water; the dogs were given access to the food for 1 hour/day. After 33 weeks, a group of five male and five female dogs was added to the study and fed a diet containing 1 ppm (0.02 and 0.03 mg beryllium/kg/day for males and females, respectively); duration of exposure for this group was 143 weeks. The following parameters were used to assess toxicity: daily observations, food consumption, body weight, hematology and serum clinical chemistry (blood samples collected after 1, 3, 6, 16, 18, 24, 30, and 36 months of exposure), urinalysis (samples collected after 1, 3, 6, 18, 24, 30, and 36 months of exposure), organ weights (heart, liver, kidney, brain, spleen, pituitary, thyroids, adrenals, and gonads), and histopathology of the spleen, thymus, pancreas, lungs, gonads, stomach, small and large intestines, urinary bladder, heart, aorta, muscle, adrenals, thyroids, lymph nodes, salivary glands, gallbladder, liver, kidneys, pituitary, brain, spinal cord, skin, mammary gland, bone marrow, and eyes.

Effects noted in study and corresponding doses: Two moribund animals in the 500 ppm group were sacrificed during week 26; the remainder of the animals in the 500 ppm group were killed during week 33. Overt signs of toxicity in the 500 ppm group included lassitude, weight loss, anorexia, and visibly bloody feces. A slight anemia (slight decreases in erythrocyte, hemoglobin, and hematocrit; statistical analysis not reported), more apparent in the females than in the males, was observed after 3 and 6 months of exposure; however, there were no alterations in the bone marrow and none of the animals was seriously affected. The study authors noted that the anemia may have been related to the gastrointestinal tract hemorrhages rather than a direct effect of beryllium on the hematological system. All animals in the 500 ppm group showed fairly extensive erosive (ulcerative) and inflammatory lesions in the gastrointestinal tract. These occurred predominantly in the small intestine, and to a lesser extent in the stomach and large intestine, and were regarded by the authors as treatment-related. All of the animals with stomach or large intestinal lesions also had lesions in the small intestine, except for one animal with stomach lesions only. This animal had stomach lesions that were very localized and not very severe. Erythroid hypoplasia of the bone marrow, bile stasis and vasculitis in the liver, and acute inflammation in the lymph nodes were also observed in the 500 ppm group; these effects are likely to be secondary to the gastrointestinal hemorrhages and a likely systemic bacterial invasion through the damaged intestinal mucosa.

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One dog in the 50 ppm group died after 70 weeks. Ulcerative gastrointestinal lesions were observed in
this animal. No beryllium-related hematological, serum chemistry, urinalysis, organ weight, or body
weight alterations were observed in the groups exposed to <500 ppm beryllium.

Dose and end point used for MRL derivation: THE CHRONIC-DURATION ORAL MRL FOR BERYLLIUM
CALCULATED USING A BENCHMARK DOSE METHOD THAT INVOLVES FITTING MATHEMAT
RESPONSE DATA FOR ULCERATIVE LESIONS OF THE SMALL INTESTINE OF DOGS EXPOSED
DIET (MORGAREIDGE ET AL. 1976). BECAUSE THERE WERE NO APPARENT SEX-RELATED DIF
OF THESE LESIONS, THE INCIDENCE DATA FOR THE MALE AND FEMALE DOGS WERE COM
DOSES FOR THE MALES AND FEMALES WERE AVERAGED. THE DOSE-RESPONSE DATA ARE F

DIETARY CONCENTRATION (PPM)	DOSE (MG BERYLLIUM/KG/DAY)	INCIDENCE
0	0	0/10
1	0.025	0/10
5	0.15	0/10
50	1	1/10
500	14.5	9/10

FOR THIS ANALYSIS, THE BENCHMARK DOSE (BMD) WAS DEFINED AS THE DOSE CORRESP
THE INCIDENCE OF SMALL INTESTINE LESIONS COMPARED TO CONTROLS; THE BMDL IS
LIMIT ON THE BMD. EPA'S BENCHMARK DOSE SOFTWARE (VERSION 1.3) WAS USED TO FIT
MODELS (GAMMA, LOGISTIC, LOG-LOGISTIC, MULTISTAGE, PROBIT, LOG-PROBIT, QUANTA
QUANTAL-QUADRATIC, AND WEIBULL) TO THE INCIDENCE DATA. MODEL FIT WAS JUDGE
ASSOCIATED WITH THE CHI-SQUARE GOODNESS-OF-FIT STATISTIC GENERATED BY THE M
INSPECTION OF THE PLOT OF OBSERVED AND PREDICTED VALUES. THE PROBIT MODEL I
THE INCIDENCE DATA; THE OBSERVED AND PREDICTED INCIDENCES ARE ILLUSTRATED
FITS THE DATA USING THE MAXIMUM LIKELIHOOD METHOD AND THE FOLLOWING EQU
BACKGROUND   (1-BACKGROUND) X CUMNORM(INTERCEPT   SLOPE X LOG(DOSE)), WHER
CUMULATIVE NORMAL DISTRIBUTION FUNCTION. A BMDL OF 0.56 MG BERYLLIUM/KG/D
MODEL.

[ ] NOAEL   [ ] LOAEL   [X] BENCHMARK DOSE (BMDL)

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Probit Model with 0.95 Confidence Level
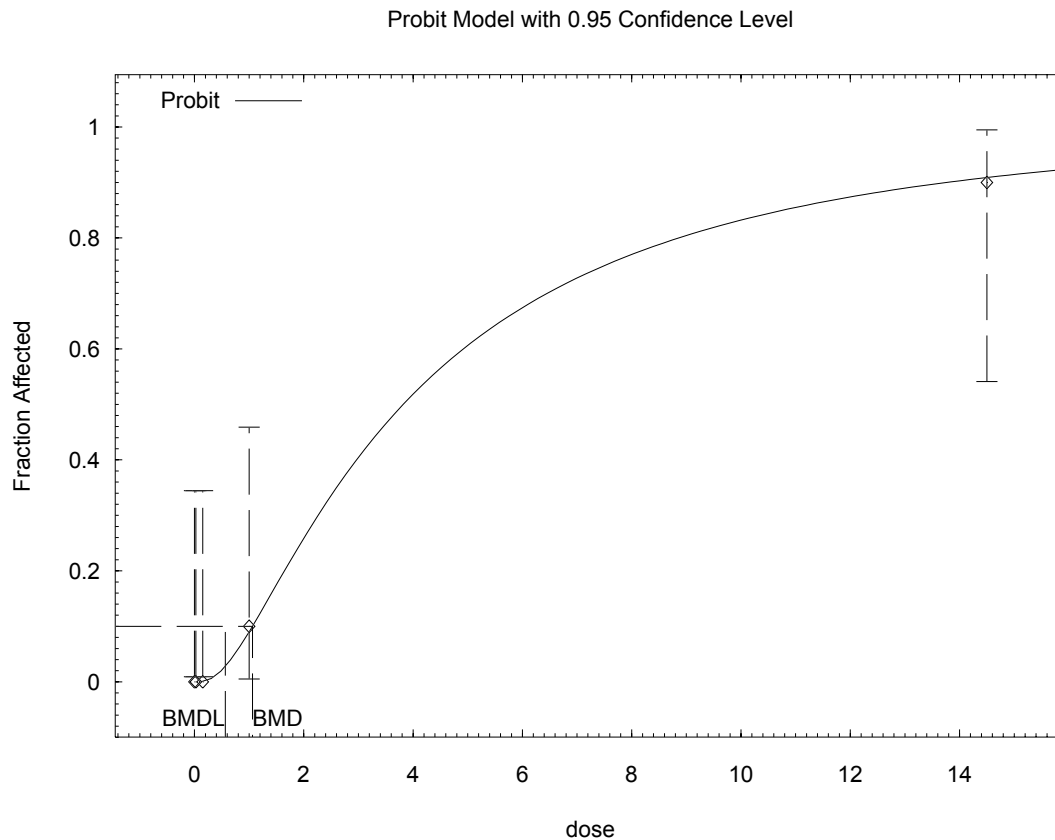
22:59 02/05 2002

Figure 1. Observed and Predicted (by Probit Model) Incidences of Intestinal Lesions in Dogs exposed to Beryllium in the Diet.

Uncertainty Factors used in MRL derivation:

       [X] 10 for use of a extrapolation from animals to humans
       [X] 10 for human variability
       [X] 3 modifying factor to account for the lack of a study that supports the gastrointestinal effects found in the Morgareidge et al. [1976] dog study and the uncertainty as to whether the benchmark dose level is the NOAEL.

Was a conversion factor used from ppm in food or water to a mg/body weight dose?

Yes. Daily doses were estimated using the estimated TWA body weights of 13.0, 12.7, 13.8, and 12.3 kg for males in the 1, 5, 50, and 500 ppm groups, respectively, and 10.2, 10.3, 11.2, and 8.6 kg for females, respectively and the reported average food intake of 300 g/day.

If an inhalation study in animals, list conversion factors used in determining human equivalent dose:

NA

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Was a conversion used from intermittent to continuous exposure?

No

Other additional studies or pertinent information that lend support to this MRL:

Gastrointestinal effects have not been reported in rats (Morgareidge et al. 1975; Schroeder and Mitchener 1976a) or mice (Schroeder and Mitchener 1976b) chronically exposed to dietary beryllium.

Agency Contact (Chemical Manager): Cassandra Smith, M.S.

APPENDIX B

USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in non-technical language. Its intended audience is the general public especially people living in the vicinity of a hazardous waste site or chemical release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the chemical.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 2

Relevance to Public Health

This chapter provides a health effects summary based on evaluations of existing toxicologic, epidemiologic, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1. What effects are known to occur in humans?

2. What effects observed in animals are likely to be of concern to humans?

3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The chapter covers end points in the same order they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, dermal) and within route by effect. Human data are presented first, then animal data. Both are organized by duration (acute, intermediate, chronic). *In vitro* data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this chapter. If data are located in the scientific literature, a table of genotoxicity information is included.

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency or perform cancer risk assessments. Minimal risk levels (MRLs) for noncancer end points (if derived) and the end points from which they were derived are indicated and discussed.

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Chapter 3 Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information is available, we have derived minimal risk levels (MRLs) for inhalation and oral routes of entry at each duration of exposure (acute, intermediate, and chronic). These MRLs are not meant to support regulatory action; but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans. They should help physicians and public health officials determine the safety of a community living near a chemical emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicologic information on which the number is based. Chapter 2, "Relevance to Public Health," contains basic information known about the substance. Other sections such as Chapter 3 Section 3.9, "Interactions with Other Substances," and Section 3.10, "Populations that are Unusually Susceptible" provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses for lifetime exposure (RfDs).

To derive an MRL, ATSDR generally selects the most sensitive end point which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential systemic, neurological, and developmental effects. If this information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest NOAEL that does not exceed any adverse effect levels. When a NOAEL is not available, a lowest-observed-adverse-effect level (LOAEL) can be used to derive an MRL, and an uncertainty factor (UF) of 10 must be employed. Additional uncertainty factors of 10 must be used both for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the LSE Tables.

Chapter 3

Health Effects

Tables and Figures for Levels of Significant Exposure (LSE)

Tables (3-1, 3-2, and 3-3) and figures (3-1 and 3-2) are used to summarize health effects and illustrate graphically levels of exposure associated with those effects. These levels cover health effects observed at increasing dose concentrations and durations, differences in response by species, minimal risk levels (MRLs) to humans for noncancer end points, and EPA's estimated range associated with an upper- bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. Use the LSE tables and figures for a quick review of the health effects and to locate data for a specific exposure scenario. The LSE tables and figures should always be used in conjunction with the text. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of No-Observed-Adverse-Effect Levels (NOAELs), Lowest-Observed-Adverse-Effect Levels (LOAELs), or Cancer Effect Levels (CELs).

The legends presented below demonstrate the application of these tables and figures. Representative examples of LSE Table 3-1 and Figure 3-1 are shown. The numbers in the left column of the legends correspond to the numbers in the example table and figure.

LEGEND

See LSE Table 3-1

(1)   Route of Exposure One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. When sufficient data exists, three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 3-1, 3-2, and 3-3, respectively). LSE figures are limited to the inhalation (LSE Figure 3-1) and oral (LSE Figure 3-2) routes. Not all substances will have data on each route of exposure and will not therefore have all five of the tables and figures.

(2)   Exposure Period Three exposure periods - acute (less than 15 days), intermediate (15–364 days), and chronic (365 days or more) are presented within each relevant route of exposure. In this example, an inhalation study of intermediate exposure duration is reported. For quick reference to health effects occurring from a known length of exposure, locate the applicable exposure period within the LSE table and figure.

(3)   Health Effect The major categories of health effects included in LSE tables and figures are death, systemic, immunological, neurological, developmental, reproductive, and cancer. NOAELs and LOAELs can be reported in the tables and figures for all effects but cancer. Systemic effects are further defined in the "System" column of the LSE table (see key number 18).

(4)   Key to Figure Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to derive a NOAEL and a Less Serious LOAEL (also see the 2 "18r" data points in Figure 3-1).

(5)   Species The test species, whether animal or human, are identified in this column. Chapter 2, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 3.4, "Toxicokinetics," contains any available information on comparative toxicokinetics. Although NOAELs and LOAELs are species specific, the levels are extrapolated to equivalent human doses to derive an MRL.

(6)   Exposure Frequency/Duration The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to 1,1,2,2-tetrachloroethane via inhalation for 6 hours per day, 5 days per week, for 3 weeks. For a more complete review of the dosing regimen refer to the appropriate sections of the text or the original reference paper, i.e., Nitschke et al. 1981.

(7)   System This column further defines the systemic effects. These systems include: respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, and dermal/ocular. "Other" refers to any systemic effect (e.g., a decrease in body weight) not covered in these systems. In the example of key number 18, 1 systemic effect (respiratory) was investigated.

(8)   NOAEL A No-Observed-Adverse-Effect Level (NOAEL) is the highest exposure level at which no
       harmful effects were seen in the organ system studied. Key number 18 reports a NOAEL of 3 ppm
       for the respiratory system which was used to derive an intermediate exposure, inhalation MRL of
       0.005 ppm (see footnote "b").

(9)   LOAEL A Lowest-Observed-Adverse-Effect Level (LOAEL) is the lowest dose used in the study
       that caused a harmful health effect. LOAELs have been classified into "Less Serious" and
       "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse
       health effects first appear and the gradation of effects with increasing dose. A brief description of
       the specific end point used to quantify the adverse effect accompanies the LOAEL. The respiratory
       effect reported in key number 18 (hyperplasia) is a Less serious LOAEL of 10 ppm. MRLs are not
       derived from Serious LOAELs.

(10)   Reference The complete reference citation is given in Chapter 9 of the profile.

(11)   CEL A Cancer Effect Level (CEL) is the lowest exposure level associated with the onset of
       carcinogenesis in experimental or epidemiologic studies. CELs are always considered serious
       effects. The LSE tables and figures do not contain NOAELs for cancer, but the text may report
       doses not causing measurable cancer increases.

(12)   Footnotes Explanations of abbreviations or reference notes for data in the LSE tables are found in
       the footnotes. Footnote "b" indicates the NOAEL of 3 ppm in key number 18 was used to derive an
       MRL of 0.005 ppm.

LEGEND

See Figure 3-1

LSE figures graphically illustrate the data presented in the corresponding LSE tables. Figures help the
reader quickly compare health effects according to exposure concentrations for particular exposure
periods.

(13)   Exposure Period The same exposure periods appear as in the LSE table. In this example, health
       effects observed within the intermediate and chronic exposure periods are illustrated.

(14)   Health Effect These are the categories of health effects for which reliable quantitative data exists.
       The same health effects appear in the LSE table.

(15)   Levels of Exposure concentrations or doses for each health effect in the LSE tables are graphically
       displayed in the LSE figures. Exposure concentration or dose is measured on the log scale "y" axis.
       Inhalation exposure is reported in mg/m^3 or ppm and oral exposure is reported in mg/kg/day.

(16)   NOAEL In this example, 18r NOAEL is the critical end point for which an intermediate inhalation
       exposure MRL is based. As you can see from the LSE figure key, the open-circle symbol indicates
       to a NOAEL for the test species-rat. The key number 18 corresponds to the entry in the LSE table.
       The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see
       entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).

(17)   CEL Key number 38r is 1 of 3 studies for which Cancer Effect Levels were derived. The diamond
       symbol refers to a Cancer Effect Level for the test species-mouse. The number 38 corresponds to
       the entry in the LSE table.

(18)   <u>Estimated Upper-Bound Human Cancer Risk Levels</u> This is the range associated with the upper-bound for lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. These risk levels are derived from the EPA's Human Health Assessment Group's upper-bound estimates of the slope of the cancer dose response curve at low dose levels (q_1^*).

(19)   <u>Key to LSE Figure</u> The Key explains the abbreviations and symbols used in the figure.

APPENDIX B

SAMPLE

Table 3-1. Levels of Significant Exposure to [Chemical x] – Inhalation

Key to figure[a]	Species	Exposure frequency/ duration	System	NOAEL (ppm)	LOAEL (effect) Less serious (ppm)	LOAEL (effect) Serious (ppm)	Reference
[5]	[6]	[6]	[7]	[8]	[9]		[10]
INTERMEDIATE EXPOSURE							
Systemic							
18	Rat	13 wk 5 d/wk 6 hr/d	Resp	3[b]	10 (hyperplasia)		Nitschke et al. 1981
CHRONIC EXPOSURE						[11]	
Cancer							
38	Rat	18 mo 5 d/wk 7 hr/d				20 (CEL, multiple organs)	Wong et al. 1982
39	Rat	89–104 wk 5 d/wk 6 hr/d				10 (CEL, lung tumors, nasal tumors)	NTP 1982
40	Mouse	79–103 wk 5 d/wk 6 hr/d				10 (CEL, lung tumors, hemangiosarcomas)	NTP 1982

[1] [2] [3] [4] [12]

a   The number corresponds to entries in Figure 3-1.
b   Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5 x 10⁻³ ppm; dose adjusted for intermittent exposure and divided by an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).

Figure 3-1. Levels of Significant Exposure to [Chemical X] - Inhalation
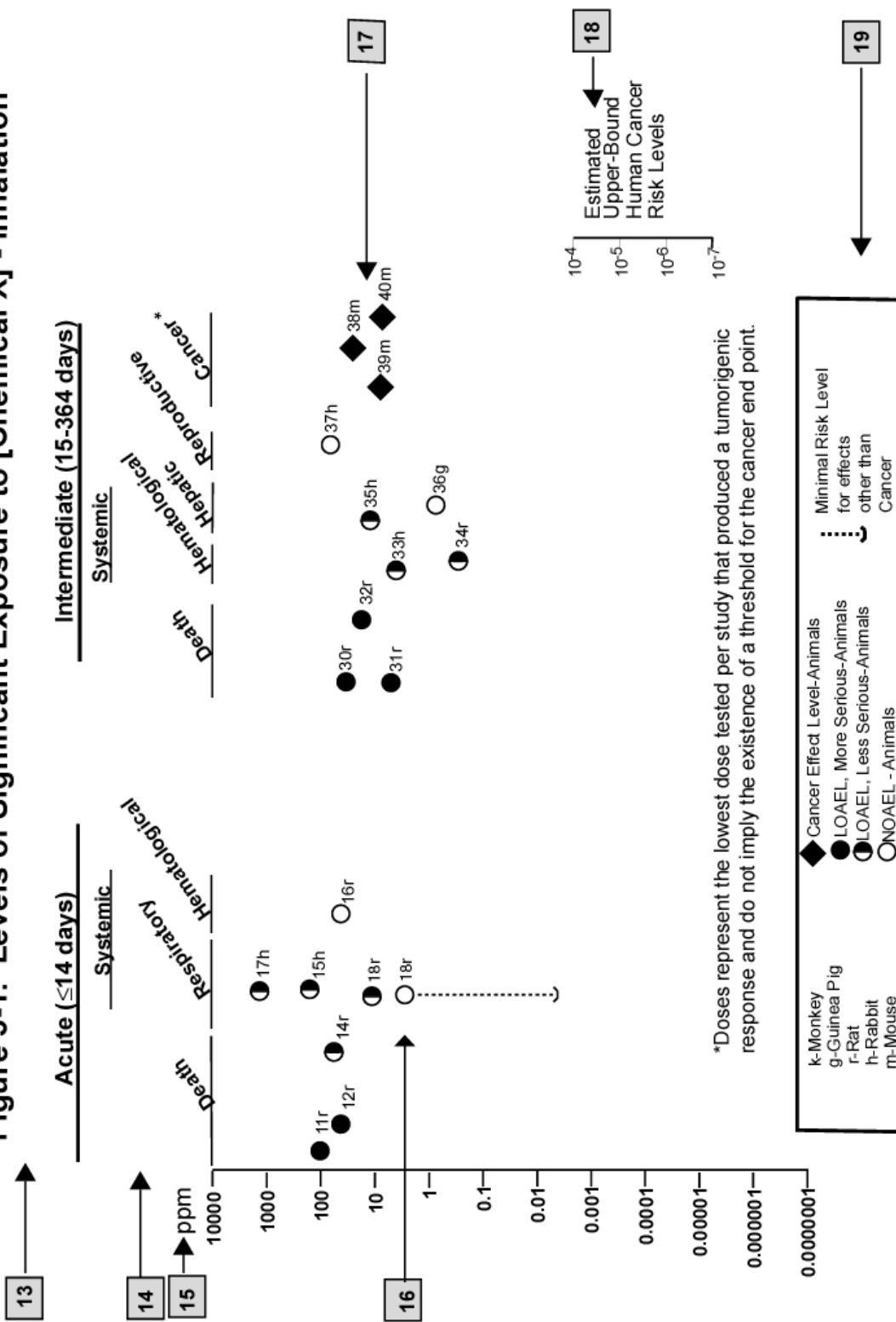
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ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACOEM	American College of Occupational and Environmental Medicine
ACGIH	American Conference of Governmental Industrial Hygienists
ADI	acceptable daily intake
ADME	absorption, distribution, metabolism, and excretion
AED	atomic emission detection
AOEC	Association of Occupational and Environmental Clinics
AFID	alkali flame ionization detector
AFOSH	Air Force Office of Safety and Health
ALT	alanine aminotransferase
AML	acute myeloid leukemia
AOAC	Association of Official Analytical Chemists
AP	alkaline phosphatase
APHA	American Public Health Association
AST	aspartate aminotranferase
atm	atmosphere
ATSDR	Agency for Toxic Substances and Disease Registry
AWQC	Ambient Water Quality Criteria
BAT	best available technology
BCF	bioconcentration factor
BEI	Biological Exposure Index
BSC	Board of Scientific Counselors
C	centigrade
CAA	Clean Air Act
CAG	Cancer Assessment Group of the U.S. Environmental Protection Agency
CAS	Chemical Abstract Services
CDC	Centers for Disease Control and Prevention
CEL	cancer effect level
CELDS	Computer-Environmental Legislative Data System
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
Ci	curie
CI	confidence interval
CL	ceiling limit value
CLP	Contract Laboratory Program
cm	centimeter
CML	chronic myeloid leukemia
CPSC	Consumer Products Safety Commission
CWA	Clean Water Act
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DNA	deoxyribonucleic acid
DOD	Department of Defense
DOE	Department of Energy
DOL	Department of Labor
DOT	Department of Transportation

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DOT/UN/	Department of Transportation/United Nations/
NA/IMCO	North America/International Maritime Dangerous Goods Code
DWEL	drinking water exposure level
ECD	electron capture detection
ECG/EKG	electrocardiogram
EEG	electroencephalogram
EEGL	Emergency Exposure Guidance Level
EPA	Environmental Protection Agency
F	Fahrenheit
F_1	first-filial generation
FAO	Food and Agricultural Organization of the United Nations
FDA	Food and Drug Administration
FEMA	Federal Emergency Management Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FPD	flame photometric detection
fpm	feet per minute
FR	*Federal Register*
FSH	follicle stimulating hormone
g	gram
GC	gas chromatography
gd	gestational day
GLC	gas liquid chromatography
GPC	gel permeation chromatography
HPLC	high-performance liquid chromatography
HRGC	high resolution gas chromatography
HSDB	Hazardous Substance Data Bank
IARC	International Agency for Research on Cancer
IDLH	immediately dangerous to life and health
ILO	International Labor Organization
IRIS	Integrated Risk Information System
Kd	adsorption ratio
kg	kilogram
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{Lo}	lethal concentration, low
LC_{50}	lethal concentration, 50% kill
LD_{Lo}	lethal dose, low
LD_{50}	lethal dose, 50% kill
LDH	lactic dehydrogenase
LH	luteinizing hormone
LT_{50}	lethal time, 50% kill
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
m	meter
MA	*trans,trans*-muconic acid
MAL	maximum allowable level
mCi	millicurie
MCL	maximum contaminant level
MCLG	maximum contaminant level goal

APPENDIX C

MFO	mixed function oxidase
mg	milligram
mL	milliliter
mm	millimeter
mmHg	millimeters of mercury
mmol	millimole
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NAAQS	National Ambient Air Quality Standard
NAS	National Academy of Science
NATICH	National Air Toxics Information Clearinghouse
NATO	North Atlantic Treaty Organization
NCE	normochromatic erythrocytes
NCEH	National Center for Environmental Health
NCI	National Cancer Institute
ND	not detected
NFPA	National Fire Protection Association
ng	nanogram
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
NLM	National Library of Medicine
nm	nanometer
NHANES	National Health and Nutrition Examination Survey
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPD	nitrogen phosphorus detection
NPDES	National Pollutant Discharge Elimination System
NPL	National Priorities List
NR	not reported
NRC	National Research Council
NS	not specified
NSPS	New Source Performance Standards
NTIS	National Technical Information Service
NTP	National Toxicology Program
ODW	Office of Drinking Water, EPA
OERR	Office of Emergency and Remedial Response, EPA
OHM/TADS	Oil and Hazardous Materials/Technical Assistance Data System
OPP	Office of Pesticide Programs, EPA
OPPTS	Office of Prevention, Pesticides and Toxic Substances, EPA
OPPT	Office of Pollution Prevention and Toxics, EPA
OR	odds ratio
OSHA	Occupational Safety and Health Administration
OSW	Office of Solid Waste, EPA
OW	Office of Water
OWRS	Office of Water Regulations and Standards, EPA
PAH	polycyclic aromatic hydrocarbon
PBPD	physiologically based pharmacodynamic

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PBPK	physiologically based pharmacokinetic
PCE	polychromatic erythrocytes
PEL	permissible exposure limit
PID	photo ionization detector
pg	picogram
pmol	picomole
PHS	Public Health Service
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
PSNS	pretreatment standards for new sources
RBC	red blood cell
REL	recommended exposure level/limit
RfC	reference concentration
RfD	reference dose
RNA	ribonucleic acid
RTECS	Registry of Toxic Effects of Chemical Substances
RQ	reportable quantity
SARA	Superfund Amendments and Reauthorization Act
SCE	sister chromatid exchange
SGOT	serum glutamic oxaloacetic transaminase
SGPT	serum glutamic pyruvic transaminase
SIC	standard industrial classification
SIM	selected ion monitoring
SMCL	secondary maximum contaminant level
SMR	standardized mortality ratio
SNARL	suggested no adverse response level
SPEGL	Short-Term Public Emergency Guidance Level
STEL	short term exposure limit
STORET	Storage and Retrieval
TD_{50}	toxic dose, 50% specific toxic effect
TLV	threshold limit value
TOC	total organic carbon
TPQ	threshold planning quantity
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TWA	time-weighted average
UF	uncertainty factor
U.S.	United States
USDA	United States Department of Agriculture
USGS	United States Geological Survey
VOC	volatile organic compound
WBC	white blood cell
WHO	World Health Organization

>	greater than
$	greater than or equal to
=	equal to
<	less than

APPENDIX C

#	less than or equal to
%	percent
α	alpha
β	beta
γ	gamma
δ	delta
μm	micrometer
μg	microgram
q_1^*	cancer slope factor
−	negative
+	positive
(+)	weakly positive result
(−)	weakly negative result

APPENDIX D

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APPENDIX D