# APPENDIX A

## ATSDR MINIMAL RISK LEVELS AND WORKSHEETS

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) 142 U.S.C. 9601 et seq.], as amended by the Superfund Amendments and Reauthorization Act (SARA) [Pub. L. 9994991, 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, summaryI 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.

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, MREs 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, MRL,s 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.

APPENDIX A

# MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Sulfur Dioxide
CAS Number: 7446-09-5
Date: November 1998
Profile Status: Third Draft Post-Public
Route: [x] Inhalation [ ] Oral
Duration: [x] Acute [ ] Intermediate [ ] Chronic
Graph Key: 25
Species: Human

Minimal Risk Level: 0.01 [ ] mg/kg/day [x] ppm

Reference: Sheppard et al. 1981. Exercise Increases Sulfur Dioxide-induced Bronchoconstriction in Asthmatic Subjects. Am Rev Respir Dis 123:486-491

Experimental design: (human study details or strain, number of animals per exposure/control groups, sex, dose administration details):

Two separate sets of studies on two separate groups of mild asthmatics were conducted. In the first set of studies, the effects of exercise on sulfur dioxide-induced bronchoconstriction was assessed in seven subjects (six men and one woman). The study design included an examination of the changes in specific airway resistance ($SR_{aw}$) produced by moderate exercise (10 minute duration) alone, inhalation of 0.10, 0.25, and 0.50 ppm sulfur dioxide alone, and the combination of exercise and sulfur dioxide. Subjects breathed sulfur dioxide and/or air from a mouthpiece.

In the second set of studies, a comparison was made between the bronchoconstriction produced by breathing sulfur dioxide during exercise and that produced by eucapnic hyperventilation with sulfur dioxide in six subjects (four men and two women). In one experiment, subjects were exposed to 1.0 ppm sulfur dioxide from a mouthpiece while exercising for 5 minutes. In another experiment, subjects were exposed to 1.0 ppm sulfur dioxide and instructed to hyperventilate. The pattern of hyperventilation approximated the pattern of the breathing of subjects during exercise. In addition, the effect of increased tidal volumes on the measurements of $SR_{aw}$ after sulfur dioxide-induced bronchoconstriction was assessed in one subject since deep breathing may modify bronchoconstriction, and because hyperpnea occurs after exercise.

Effects noted in study and corresponding doses:

In the seven subjects with mild asthma, inhalation of 0.25 ppm sulfur dioxide during the performance of moderate exercise significantly increased $SR_{aw}$. Inhalation of 0.50 ppm during exercise significantly increased $SR_{aw}$ in all seven subjects ($p<0.05$), and three developed wheezing and shortness of breath. During the corresponding period of exercise alone and during inhalation of 0.50 ppm at rest, $SR_{aw}$ did not increase in any subject. After inhalation of 0.50 ppm of sulfur dioxide during exercise, $\Delta SR_{aw}$ (the difference between baseline specific airway resistance and specific airway resistance after inhalation of sulfur dioxide) was significantly greater than after exercise alone or inhalation of 0.50 ppm of sulfur dioxide at rest ($p<0.05$). Inhalation of 0.25 ppm sulfur dioxide during exercise significantly increased $SR_{aw}$ in three of the seven subjects, and the increase in $SR_{aw}$ for the group was significant ($p<0.05$). No subject developed wheezing or shortness of breath. During the corresponding period of exercise alone, $SR_{aw}$ did not increase in any subject. In the two most responsive subjects, inhalation of 0.10 ppm significantly increased $SR_{aw}$, and there was a dose-response relationship to 0.10, 0.25, and 0.50 ppm in the 2 subjects. $\Delta SR_{aw}$ at 0.10 ppm was slight and

was approximately 2.5 L x cm $H_20$/L/s (units for $\Delta SR_{aw}$). At 0.25 ppm, $\Delta SR_{aw}$ was approximately 5 L x cm $H_20$/L/s. At 0.5 ppm, the $\Delta SR_{aw}$ exceeded 15 L x cm $H_20$/L/s.

In the second set of studies, in all six subjects, inhalation of 1 ppm of sulfur dioxide dramatically increased $SR_{aw}$, both when it was delivered during exercise and during eucapnic hyperventilation (rapid, deep breathing to deplete arterial $CO_2$). In every case, the increase in $SR_{aw}$ was accompanied by dyspnea and audible wheezing. The magnitude of the increase in $SR_{aw}$ was the same when subjects inhaled sulfur dioxide while they exercised or while they performed eucapnic hyperventilation at the same minute ventilation.

The study authors concluded that moderate exercise increases the bronchomotor effect of sulfur dioxide in subjects with asthma so the concentrations as low as 0.10 ppm can cause sigificant bronchoconstriction. However, the $\Delta SR_{aw}$ at 0.10 ppm was slight. In addition, the authors stated that the concentrations studied are sometimes equaled or exceeded in polluted urban air, and that their findings support the contention that sulfur dioxide is at least partially responsible for the observed association between air pollution and increased morbidity from asthma.

Dose and endpoint used for MRL derivation:

[] NOAEL [X] minimal LOAEL

0.1 ppm, bronchoconstriction in exercising asthmatics

Uncertainty Factors used in MRL derivation:

[X] 3 for use of a minimal LOAEL
[ ] 10 for extrapolation from animals to humans
[X] 3 for human variability

The uncertainty factor for human variability addresses varying sensitivity among asthmatics and possible increased sensitivity in children. There is concern of increased sensitivity in children but there is not sufficient data to confirm it.

Was a conversion used from ppm in food or water to a mg/body weight dose?
If so, explain: No, the doses provided are author-provided.

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

None

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

Lung function changes in asthmatics exposed by inhalation to sulfur dioxide have been reported by other investigators. In a chamber study of moderately exercising asthmatics, the concentration of sulfur dioxide required to produce an increase in airway resistance 100% greater than the response to clean air [designated as $PC(SO_2)$] has been determined (Horstman et al. 1986). Analysis of the cumulative percentage of subjects plotted as a function of $PC(SO_2)$ revealed that 25% of the subjects exhibited a $PC(SO_2)$ of 0.25 to 0.5 ppm sulfur dioxide. The study authors considered that the 25% of the mild asthmatics who were very sensitive to sulfur dioxide could possibly exhibit bronchoconstriction if they were to perform normal exercise routines in some highly industrialized areas of the United States. A dose-related increase in specific airway resistance

was seen in asthmatics following a 3 minute exposure (via mouthpiece) to ≥0.25 ppm sulfur dioxide (Myers et al. 1986a; Myers et al. 1986b).

Increases in specific airway resistance were observed in moderately exercising asthmatics exposed oronasally to 0.25 ppm sulfur dioxide for 5 minutes (Bethel et al. 1985). This study could have also been used to develop an MRL. An uncertainty factor of 30 would have been required (10 for the use of a LOAEL and 3 for human variability). Dividing the LOAEL of 0.25 ppm by an uncertainty factor of 30 results in an MRL of 0.01 ppm, a value consistent with the MRL derived from the Sheppard et al. (1981) study.
Some studies of asthmatics have reported a lack of significant lung function changes in asthmatics following exposures to 0.1-0.5 ppm (Jorres and Magnussen 1990; Koenig et al. 1990). Bronchoconstrictive responses to sulfur dioxide are highly variable among individual asthmatics (Horstman et al. 1986). In some studies asthmatics were preselected for sensitivity to sulfur dioxide and this may explain the range of sulfur dioxide-induced responses obtained by different investigators.

The dose level of 0.1 ppm sulfur dioxide can be considered a minimal LOAEL.

Agency Contact (Chemical Manager): Hana Pohl

Agency Review Date: 1° review:_____
2° review:_____

# 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

### Tables and Figures for Levels of Significant Exposure (LSE)

Tables (2-1,2-2, and 2-3) and figures (2-1 and 2-2) 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 2-1 and Figure 2-1 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 2-1

(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 2-1, 2-2, and 2-3, respectively). LSE figures are limited to the inhalation (LSE Figure 2-l) and oral (LSE Figure 2-2) 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 2-1).

(5) Species The test species, whether animal or human, are identified in this column. Section 2.5, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 2.3, "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 endpoint 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 8 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 2-1

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 endpoint 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) Estimated Upper-Bound Human Cancer Risk Levels 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 (ql*).

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

The Relevance to Public Health section 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 section 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 section. 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 Data Needs section.

SAMPLE

1 → **TABLE 2-1. Levels of Significant Exposure to [Chemical x] – Inhalation**

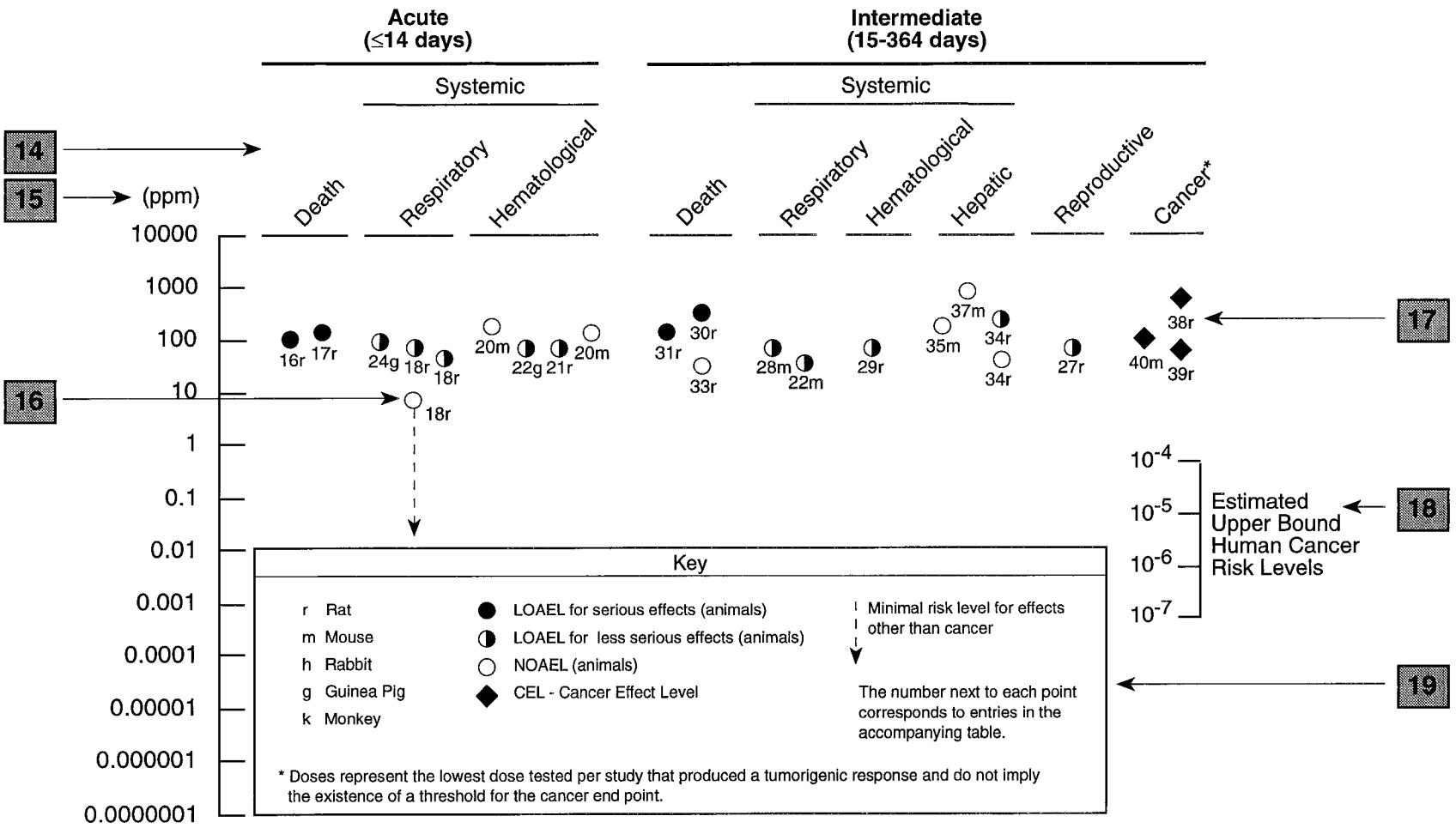
| Key to figure[a] | Species | Exposure frequency/ duration | System | NOAEL (ppm) | LOAEL (effect) Less serious (ppm) | | Reference |
|---|---|---|---|---|---|---|---|
| 2 → INTERMEDIATE EXPOSURE | | | | | | | |
| | 5 | 6 | 7 | 8 | 9 | | 10 |
| 3 → Systemic | ↓ | ↓ | ↓ | ↓ | ↓ | | ↓ |
| 4 → 18 | Rat | 13 wk 5d/wk 6hr/d | Resp | 3 [b] | 10 (hyperplasia) | | Nitschke et al. 1981 |
| CHRONIC EXPOSURE | | | | | | | |
| | | | | | 11 | | |
| Cancer | | | | | ↓ | | |
| 38 | Rat | 18 mo 5d/wk 7hr/d | | | 20 | (CEL, multiple organs) | Wong et al. 1982 |
| 39 | Rat | 89–104 wk 5d/wk 6hr/d | | | 10 | (CEL, lung tumors, nasal tumors) | NTP 1982 |
| 40 | Mouse | 79–103 wk 5d/wk 6hr/d | | | 10 | (CEL, lung tumors, hemangiosarcomas) | NTP 1982 |

[a] The number corresponds to entries in Figure 2-1.

12 → [b] an uncertainty factor of 100 (10 for extrapolation from animal to humans, 10 for human variability).

SAMPLE

13 → **Figure 2-1. Levels of Significant Exposure to [Chemical X] – Inhalation**



## Chapter 2 (Section 2.5)

## Relevance to Public Health

## 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 2.5, "Relevance to Public Health," contains basic information known about the substance. Other sections such as 2.8, "Interactions with Other Substances," and 2.9, "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 endpoint 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 endpoint 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.

# APPENDIX C

# ACRONYMS, ABBREVIATIONS, AND SYMBOLS

| | |
|---|---|
| ACGIH | American Conference of Governmental Industrial Hygienists |
| ADME | Absorption, Distribution, Metabolism, and Excretion |
| atm | atmosphere |
| ATSDR | Agency for Toxic Substances and Disease Registry |
| BCF | bioconcentration factor |
| BSC | Board of Scientific Counselors |
| C | Centigrade |
| CDC | Centers for Disease Control |
| CEL | Cancer Effect Level |
| CERCLA | Comprehensive Environmental Response, Compensation, and Liability Act |
| CFR | Code of Federal Regulations |
| CLP | Contract Laboratory Program |
| cm | centimeter |
| CNS | central nervous system |
| d | day |
| DHEW | Department of Health, Education, and Welfare |
| DHHS | Department of Health and Human Services |
| DOL | Department of Labor |
| ECG | electrocardiogram |
| EEG | electroencephalogram |
| EPA | Environmental Protection Agency |
| EKG | see ECG |
| F | Fahrenheit |
| $F_1$ | first filial generation |
| FAO | Food and Agricultural Organization of the United Nations |
| FEMA | Federal Emergency Management Agency |
| FIFRA | Federal Insecticide, Fungicide, and Rodenticide Act |
| fpm | feet per minute |
| ft | foot |
| FR | *Federal Register* |
| g | gram |
| GC | gas chromatography |
| gen | generation |
| HPLC | high-performance liquid chromatography |
| hr | hour |
| IDLH | Immediately Dangerous to Life and Health |

APPENDIX C

| | |
|---|---|
| IARC | International Agency for Research on Cancer |
| ILO | International Labor Organization |
| in | inch |
| Kd | adsorption ratio |
| kg | kilogram |
| kkg | metric ton |
| $K_{oc}$ | 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 |
| LOAEL | lowest-observed-adverse-effect level |
| LSE | Levels of Significant Exposure |
| m | meter |
| mg | milligram |
| min | minute |
| mL | milliliter |
| mm | millimeter |
| mmHg | millimeters of mercury |
| mmol | millimole |
| mo | month |
| mppcf | millions of particles per cubic foot |
| MRL | Minimal Risk Level |
| MS | mass spectrometry |
| NIEHS | National Institute of Environmental Health Sciences |
| NIOSH | National Institute for Occupational Safety and Health |
| NIOSHTIC | NIOSH's Computerized Information Retrieval System |
| ng | nanogram |
| 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 |
| NPL | National Priorities List |
| NRC | National Research Council |
| NTIS | National Technical Information Service |
| NTP | National Toxicology Program |
| OSHA | Occupational Safety and Health Administration |

APPENDIX C

| | |
|---|---|
| PEL | permissible exposure limit |
| pg | picogram |
| pmol | picomole |
| PHS | Public Health Service |
| PMR | proportionate mortality ratio |
| ppb | parts per billion |
| ppm | parts per million |
| ppt | parts per trillion |
| REL | recommended exposure limit |
| RfD | Reference Dose |
| RTECS | Registry of Toxic Effects of Chemical Substances |
| S | sulfur |
| sec | second |
| SCE | sister chromatid exchange |
| SIC | Standard Industrial Classification |
| SMR | standard mortality ratio |
| STEL | short term exposure limit |
| STORET | STORAGE and RETRIEVAL |
| Tg | teragrams = $10^{12}$ grams |
| TLV | threshold limit value |
| TSCA | Toxic Substances Control Act |
| TRI | Toxics Release Inventory |
| TWA | time-weighted average |
| U.S. | United States |
| UF | uncertainty factor |
| yr | year |
| WHO | World Health Organization |
| wk | week |

| | |
|---|---|
| > | greater than |
| ≥ | greater than or equal to |
| = | equal to |
| < | less than |
| ≤ | less than or equal to |
| % | percent |
| α | alpha |
| β | beta |
| δ | delta |
| γ | gamma |
| μm | micrometer |
| μg | microgram |