APPENDIX A. ATSDR MINIMAL RISK LEVELS 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.

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 100-fold 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 and Human Health Sciences, expert panel peer reviews, and agency-wide 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 and Human Health Sciences, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road NE, Mailstop F-57, Atlanta, Georgia 30329-4027.
MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: DEET
CAS Numbers: 134-62-3
Date: September 2015
Profile Status: Final for Public Comment
Route: [ ] Inhalation  [X] Oral
Duration: [ ] Acute  [X] Intermediate  [ ] Chronic
Graph Key: 35
Species: Rats

Minimal Risk Level: 1.0 [X] mg/kg/day  [ ] ppm


Experimental design: Groups of Sprague-Dawley rats (28/sex/group) were fed diets containing 0, 500, 2,000, or 5,000 ppm DEET for at least 80 days before mating (EPA 1989). Using the standard conversion, 20 ppm=1 mg/kg/day (EPA 1998b), the diet provided doses of approximately 0, 25, 100, or 250 mg DEET/kg/day. Parental F0 animals were allowed to produce only one litter. Animals remained on the test diets during mating, gestation, and lactation. After weaning of F1 rats, 28 males and 28 females were randomly chosen to serve as parents for the F2 generation. F1 rats were mated after at least 93 days on the test diet. After weaning, 10 pups/sex/groups were subjected to gross necropsy. F0 females were killed after the selection of F1 parents. F1 females were sacrificed after weaning of their litters. The following parameters were used to assess toxicity: twice daily observations for deaths and clinical signs, body weight, and food consumption (not measured during the mating periods). Additionally gross necropsy and and histological examination of the ovaries, prostate, seminal vesicles, testes with epididymides, uterus, vagina, and all gross lesions were conducted in all parental rats and 10 weanlings/sex/group in the control and high-dose groups. Parameters used to assess developmental toxicity in the F1 and F2 litters included number of live and stillborn pups, external anomalies, sex and body weight grouped by sex on lactation days 0, 4, 7, and 14, sex and individual body weights (only group weights reported) on lactation day 21, viability, and behavioral abnormalities at least twice daily during lactation.

Effects noted in study and corresponding doses: There were no chemical-related deaths during the study. Hair loss appeared to be more prominent in high-dose F0 and F1 females than in other groups. Body weights of parental rats were lower in some of the mid- and high-dose groups at some points in the study, but the difference with controls was generally <10%. Changes in food consumption tended to parallel the changes in body weight and were generally <10% different than controls. F1 males showed mottled kidneys with incidences of 0/23, 2/23 (9%), 6/23 (26%), and 8/23 (35%) of the males (control and increasing dose groups). Microscopy revealed inflammation, hyaline droplet and granular cast formation, and regeneration of tubules. No explicit information was provided in the review regarding the other organs examined. The only significant reproductive/developmental effects reported were decreased F2 pup viability in the low- and high-dose groups on postnatal day 4 and reduced male and female F1 and F2 pups weight in the high-dose group on lactation days 14 and 21 (Table A-1).
Table A-1. Body Weight (g) of Rats on Lactation Day 21 in a 2-Generation Reproductive Study

<table>
<thead>
<tr>
<th>Doses (mg/kg/day)</th>
<th>0</th>
<th>25</th>
<th>100</th>
<th>250</th>
</tr>
</thead>
<tbody>
<tr>
<td>F1 males</td>
<td>46.2±6.50&lt;sup&gt;a&lt;/sup&gt;</td>
<td>46.8±5.06</td>
<td>45.7±4.53</td>
<td>40.1±4.22&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>F1 females</td>
<td>44.1±4.64</td>
<td>44.6±4.44</td>
<td>44.2±4.51</td>
<td>39.1±4.60&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>F2 males</td>
<td>50.4±4.31</td>
<td>49.4±6.07</td>
<td>47.9±4.02</td>
<td>44.5±3.93&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>F2 females</td>
<td>47.3±4.52</td>
<td>47.6±5.35</td>
<td>44.1±7.50</td>
<td>42.3±3.23&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a</sup>Mean±standard deviation.
<sup>b</sup>p<0.01.

Source: EPA (1989)

Dose and end point used for MRL derivation: NOAEL of 100 mg/kg/day (LOAEL of 250 mg/kg/day for reduced body weight in F1 and F2 male and female pups on lactation day 21).

[X] NOAEL  [ ] LOAEL

Uncertainty Factors used in MRL derivation:

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

Was a conversion factor used from ppm in food or water to a mg/body weight dose? Yes, 20 ppm=1 mg/kg/day was used as done in EPA (1998b).

If an inhalation study in animals, list conversion factors used in determining human equivalent dose: Not applicable.

Was a conversion used from intermittent to continuous exposure? No.

Other additional studies or pertinent information that lend support to this MRL: The intermediate-duration oral database showed relatively little toxicity for DEET. Most effects reported were of questionable toxicological significance except for the developmental effects that were used for MRL derivation. The fact that the reduction in body weight occurred in both male and female pups from both the F1 and F2 generations, provides strength to the MRL.

Intermediate-duration oral MRL is protective for chronic-duration exposure. The available chronic-duration oral database does not support derivation of a chronic-duration oral MRL for DEET. Long-term exposure, however, does not lead to more toxic effects than those reported for intermediate-duration exposure, so the intermediate-duration oral MRL of 1 mg/kg/day for DEET is protective for chronic-duration exposure.

Agency Contacts (Chemical Managers): Sam Keith

***DRAFT FOR PUBLIC COMMENT***
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 that they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, and 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.

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, ATSDR has derived 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.

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MRLs 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 that the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses (RfDs) for lifetime exposure.

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 no-observed-adverse-effect level (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 levels of significant exposure (LSE) tables.

Chapter 3

Health Effects

Tables and Figures for Levels of Significant Exposure (LSE)

Tables and figures 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, 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 NOAELs, 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 Sample LSE Table 3-1 (page B-6)

(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. Typically when sufficient data exist, 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 Tables 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 two "18r" data points in sample 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 regimens 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 “Chemical x” via inhalation for 6 hours/day, 5 days/week, for 13 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, one systemic effect (respiratory) was investigated.

(8) NOAEL. A 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").

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(9) **LOAEL.** A 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 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 that the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

**LEGEND**

See Sample Figure 3-1 (page B-7)

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 acute and intermediate 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/m³ or ppm and oral exposure is reported in mg/kg/day.

(16) **NOAEL.** In this example, the open circle designated 18r identifies a NOAEL critical end point in the rat upon which an intermediate inhalation exposure MRL is based. 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 38m is one of three studies for which CELs were derived. The diamond symbol refers to a CEL 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 (q₁*).

(19) **Key to LSE Figure.** The Key explains the abbreviations and symbols used in the figure.
Table 3-1. Levels of Significant Exposure to [Chemical x] – Inhalation

<table>
<thead>
<tr>
<th>Key to figure&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Exposed frequency/duration</th>
<th>System</th>
<th>NOAEL (ppm)</th>
<th>LOAEL (effect)</th>
<th>Reference</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Serious (ppm)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Less serious (ppm)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ppm</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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<td>ppm</td>
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**INTERMEDIATE EXPOSURE**

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<th>7</th>
<th>8</th>
<th>9</th>
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<tbody>
<tr>
<td>Systemic</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>18</th>
<th>Rat</th>
<th>13 wk</th>
<th>Resp</th>
<th>3&lt;sup&gt;b&lt;/sup&gt;</th>
<th>10 (hyperplasia)</th>
<th>Nitschke et al. 1981</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>5 d/wk</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>6 hr/d</td>
<td></td>
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**CHRONIC EXPOSURE**

<table>
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</table>

<table>
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<tr>
<th>38</th>
<th>Rat</th>
<th>18 mo</th>
<th>20</th>
<th>(CEL, multiple organs)</th>
<th>Wong et al. 1982</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>5 d/wk</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>7 hr/d</td>
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<table>
<thead>
<tr>
<th>39</th>
<th>Rat</th>
<th>89–104 wk</th>
<th>10</th>
<th>(CEL, lung tumors, nasal tumors)</th>
<th>NTP 1982</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>5 d/wk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>6 hr/d</td>
<td></td>
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<table>
<thead>
<tr>
<th>40</th>
<th>Mouse</th>
<th>79–103 wk</th>
<th>10</th>
<th>(CEL, lung tumors, hemangiosarcomas)</th>
<th>NTP 1982</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>5 d/wk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>6 hr/d</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 3-1. Levels of Significant Exposure to [Chemical X] - Inhalation

*Doses represent the lowest dose tested per study that produced a tumorigenic response and do not imply the existence of a threshold for the cancer end point.
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APPENDIX C. ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH  American Conference of Governmental Industrial Hygienists
ACOEM  American College of Occupational and Environmental Medicine
ADI    acceptable daily intake
ADME  absorption, distribution, metabolism, and excretion
AED   atomic emission detection
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
AOEC  Association of Occupational and Environmental Clinics
AP    alkaline phosphatase
APHA  American Public Health Association
AST   aspartate aminotransferase
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
BMD/C benchmark dose or benchmark concentration
BMDX  dose that produces a X% change in response rate of an adverse effect
BMDEX 95% lower confidence limit on the BMDX
BMDS  Benchmark Dose Software
BMR   benchmark response
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

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DOT  Department of Transportation
DOT/UN/  Department of Transportation/United Nations/
    NA/IMDG  North America/Intergovernmental Maritime Dangerous Goods Code
DWEL  drinking water exposure level
ECD  electron capture detection
ECD/EKG  electrocardiogram
EEG  electroencephalogram
EEGL  Emergency Exposure Guidance Level
EPA  Environmental Protection Agency
F  Fahrenheit
F1  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
kkg  metric ton
Koc  organic carbon partition coefficient
Kow  octanol-water partition coefficient
L  liter
LC  liquid chromatography
LC50  lethal concentration, 50% kill
LCLo  lethal concentration, low
LD50  lethal dose, 50% kill
LDLo  lethal dose, low
LDH  lactic dehydrogenase
LH  luteinizing hormone
LOAEL  lowest-observed-adverse-effect level
LSE  Levels of Significant Exposure
LT50  lethal time, 50% kill
m  meter
MA  trans,trans-muconic acid
MAL  maximum allowable level
mCi  millicurie

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MCL maximum contaminant level
MCLG maximum contaminant level goal
MF modifying factor
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
NHANES National Health and Nutrition Examination Survey
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
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
OPPT Office of Pollution Prevention and Toxics, EPA
OPPTS Office of Prevention, Pesticides and Toxic Substances, EPA
OR odds ratio
OSHA Occupational Safety and Health Administration
OSW Office of Solid Waste, EPA
OTS Office of Toxic Substances
<table>
<thead>
<tr>
<th>Acronym</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>OW</td>
<td>Office of Water</td>
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<tr>
<td>OWRS</td>
<td>Office of Water Regulations and Standards, EPA</td>
</tr>
<tr>
<td>PAH</td>
<td>polycyclic aromatic hydrocarbon</td>
</tr>
<tr>
<td>PBPD</td>
<td>physiologically based pharmacodynamic</td>
</tr>
<tr>
<td>PBPK</td>
<td>physiologically based pharmacokinetic</td>
</tr>
<tr>
<td>PCE</td>
<td>polychromatic erythrocytes</td>
</tr>
<tr>
<td>PEL</td>
<td>permissible exposure limit</td>
</tr>
<tr>
<td>pg</td>
<td>picogram</td>
</tr>
<tr>
<td>PHS</td>
<td>Public Health Service</td>
</tr>
<tr>
<td>PID</td>
<td>photo ionization detector</td>
</tr>
<tr>
<td>pmol</td>
<td>picomole</td>
</tr>
<tr>
<td>PMR</td>
<td>proportionate mortality ratio</td>
</tr>
<tr>
<td>ppb</td>
<td>parts per billion</td>
</tr>
<tr>
<td>ppm</td>
<td>parts per million</td>
</tr>
<tr>
<td>ppt</td>
<td>parts per trillion</td>
</tr>
<tr>
<td>PSNS</td>
<td>pretreatment standards for new sources</td>
</tr>
<tr>
<td>RBC</td>
<td>red blood cell</td>
</tr>
<tr>
<td>REL</td>
<td>recommended exposure level/limit</td>
</tr>
<tr>
<td>RfC</td>
<td>reference concentration</td>
</tr>
<tr>
<td>RfD</td>
<td>reference dose</td>
</tr>
<tr>
<td>RNA</td>
<td>ribonucleic acid</td>
</tr>
<tr>
<td>RQ</td>
<td>reportable quantity</td>
</tr>
<tr>
<td>RTECS</td>
<td>Registry of Toxic Effects of Chemical Substances</td>
</tr>
<tr>
<td>SARA</td>
<td>Superfund Amendments and Reauthorization Act</td>
</tr>
<tr>
<td>SCE</td>
<td>sister chromatid exchange</td>
</tr>
<tr>
<td>SGOT</td>
<td>serum glutamic oxaloacetic transaminase</td>
</tr>
<tr>
<td>SGPT</td>
<td>serum glutamic pyruvic transaminase</td>
</tr>
<tr>
<td>SIC</td>
<td>standard industrial classification</td>
</tr>
<tr>
<td>SIM</td>
<td>selected ion monitoring</td>
</tr>
<tr>
<td>SMCL</td>
<td>secondary maximum contaminant level</td>
</tr>
<tr>
<td>SMR</td>
<td>standardized mortality ratio</td>
</tr>
<tr>
<td>SNARL</td>
<td>suggested no adverse response level</td>
</tr>
<tr>
<td>SPEGL</td>
<td>Short-Term Public Emergency Guidance Level</td>
</tr>
<tr>
<td>STEL</td>
<td>short term exposure limit</td>
</tr>
<tr>
<td>STORET</td>
<td>Storage and Retrieval</td>
</tr>
<tr>
<td>TD₅₀</td>
<td>toxic dose, 50% specific toxic effect</td>
</tr>
<tr>
<td>TLV</td>
<td>threshold limit value</td>
</tr>
<tr>
<td>TOC</td>
<td>total organic carbon</td>
</tr>
<tr>
<td>TPQ</td>
<td>threshold planning quantity</td>
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<tr>
<td>TRI</td>
<td>Toxics Release Inventory</td>
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<tr>
<td>TSCA</td>
<td>Toxic Substances Control Act</td>
</tr>
<tr>
<td>TWA</td>
<td>time-weighted average</td>
</tr>
<tr>
<td>UF</td>
<td>uncertainty factor</td>
</tr>
<tr>
<td>U.S.</td>
<td>United States</td>
</tr>
<tr>
<td>USDA</td>
<td>United States Department of Agriculture</td>
</tr>
<tr>
<td>USGS</td>
<td>United States Geological Survey</td>
</tr>
<tr>
<td>VOC</td>
<td>volatile organic compound</td>
</tr>
<tr>
<td>WBC</td>
<td>white blood cell</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
</tbody>
</table>
DEET

APPENDIX C

>  greater than
\( \geq \)  greater than or equal to
=  equal to
<  less than
\( \leq \)  less than or equal to
%  percent
\( \alpha \)  alpha
\( \beta \)  beta
\( \gamma \)  gamma
\( \delta \)  delta
\( \mu m \)  micrometer
\( \mu g \)  microgram
\( q_1^* \)  cancer slope factor
–  negative
+  positive
(+)  weakly positive result
(−)  weakly negative result