APPENDIX A

MINIMAL RISK LEVEL WORKSHEETS

Chemical Name: Di-n-octylphthalate
CAS Number: 117-84-0
Date: December 1995
Profile Status: Post-Public Comment - Draft 3
Route: [x] Oral
Duration: [x] Acute [ ] Intermediate [ ] Chronic
Graph Key: 5
Species: Rat

Minimal Risk Level: 3 [x] mg/kg/day [ ] ppm

Reference: Lake et al. 1986

Experimental design: Di-n-octylphthalate was administered (1,000 mg/kg/day) once/day for 14 days to a group of 4-6 male Sprague-Dawley rats. A group of male rats, administered the corn oil vehicle, served as controls. Following the last dose, animals were starved overnight and killed by cervical dislocation. Livers were excised to be weighed, then used for biochemical assays; histopathological examination was not performed.

Effects noted in study and corresponding doses: Liver effects were observed in treated rats. The hepatic effects consisted of a statistically significant (p < 0.01) increase (17 %) in relative liver weight and a reduction (approximately 30%) in 7-ethoxycoumarin O-deethylase activity relative to the vehicle control. Enzymatic indicators of peroxisome proliferation (KCN-insensitive palmitoyl-CoA oxidation or enoyl-CoA hydratase heat labile activity) were not significantly altered compared to controls. No significant changes in P-450 content or in the following activities were noted: ethylmorphine-N-demethylase, lauric acid 11-hydroxylation, and lauric acid 12-hydroxylation.

Dose and end point used for MRL derivation:

[ ] NOAEL [x] LOAEL (DOSE: 1000 mg/kg/day)

Uncertainty Factors used in MRL derivation:

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

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

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

Other additional studies or pertinent information which lend support to this MRL: The choice of liver toxicity as the basis for the acute oral MRL is supported by similar effects seen in other acute- and intermediate-duration studies in rats (DeAngelo et al. 1986; Lake et al. 1984; Mann et al. 1985).
Chemical Name:  Di-n-octylphthalate  
CAS Number:   117-84-0  
Date:    December 1995  
Profile Status:   Post-Public Comment - Draft 3  
Route:    [ ] Inhalation [x] Oral  
Duration:   [ ] Acute [x] Intermediate [ ] Chronic  
Graph Key:   2 1  
Species:   Rat  

Minimal Risk Level:   0.4 [x] mg/kg/day [ ] ppm  
Reference:  Poon et al. 1995  

Experimental design: Groups of 10 male and 10 female Sprague-Dawley rats were maintained on diets containing 0, 5, 50, 500, or 5,000 ppm di-n-octylphthalate (DNOP) in the diet for 13 weeks. The study authors determined that these dietary concentrations corresponded to doses of 0, 0.4, 3.5, 36.8, 350.1 mg/kg/day (males) and 0, 0.4, 4.1, 40.8, 402.9 mg/kg/day (females). Control animals received feed containing 4% corn oil. The rats were examined daily for clinical signs of toxicity, while food consumption and body weight data were collected weekly. At the end of the study, the animals were anesthetized with an i.p. injection of pentobarbital. Blood was collected from the aortic artery for hematomal and biochemical determinations; enzymatic activity assays and comprehensive histopathological examinations were performed (although only data on the liver, thyroid, testis, and epididymis were presented).

Effects noted in study and corresponding doses: No clinical signs of toxicity or reduction in food consumption or body weight gain were noted. No treatment-related changes in organ weights were noted. At 5,000 ppm, increased (p < 0.05) calcium was noted in males. Liver effects observed in rats administered di-n-octylphthalate in the diet at a concentration of 5,000 ppm; the study authors calculated the doses at this concentration to be 350.1 mg/kg/day (males) and 402.9 mg/kg/day (females) (Peon et al. 1995). The hepatic effects consisted of a significant (p < 0.05) increase in ethoxyresorufin-0-deethylase activity (12-fold, males; 3-fold, females); no significant changes were noted in liver aminopyrine-N-demethylase or aniline hydrolase activities. Also, at 5,000 ppm, histopathological changes in hepatic architecture were noted, including moderate accentuation of zonation and mild-to-moderate increases in perivenous cytoplasmic vacuolation. Mild histological changes in the thyroid were also noted at 5,000 ppm that consisted of reduction in the follicle size and decreased colloid density. No effects were observed at 500 ppm (36.8 mg/kg/day for males and 40.8 mg/kg/day for females). The MRL was derived by dividing the NOAEL value of 40.8 mg/kg/day for hepatic effects by an uncertainty factor of 100 (10 for extrapolation from animals to humans and 10 for human variability).

Dose and end point used for MRL derivation:

[x] NOAEL (DOSE:  40.8 mg/kg/day) [ ] 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
APPENDIX A

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

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

Other additional studies or pertinent information which lend support to this MRL: The choice of liver toxicity as the basis of the MRL is supported by necrosis and mild hepatic fatty changes seen in other acute- and intermediate-duration studies in rats (DeAngelo et al. 1986; Lake et al. 1984, 1986; Mann et al. 1985). Thyroid toxicity (decreased thyroxine levels and ultrastructural changes) was observed after rats were fed 2,000 mg/kg/day of di-n-octylphthalate in the diet for 3, 10, or 21 days (Hinton et al. 1986).
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)

A table (2-l) and figure (2-l) 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-l and Figure 2-l 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-l

(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,
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
(see the “18r” data points in Figure 2-1).

(5) Species The test species, whether animal or human, are identified in this column. Section 2.5,
“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 15), 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.,

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

(16) NOAEL In this example, 1% 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 (q¹*).

(19) Key to LSE Figure The Key explains the abbreviations and symbols used in the figure.
### TABLE 2-1. Levels of Significant Exposure to [Chemical x] – Inhalation

<table>
<thead>
<tr>
<th>Key to</th>
<th>Exposure</th>
<th>NOAEL</th>
<th>LOAEL (effect)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>figure</td>
<td>frequency/duration</td>
<td>(ppm)</td>
<td>Less serious (ppm)</td>
<td>Serious (ppm)</td>
</tr>
<tr>
<td>1</td>
<td>Systemic</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>2</td>
<td>INTERMEDIATE EXPOSURE</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>4</td>
<td>18</td>
<td>Rat</td>
<td>13 wk</td>
<td>Resp</td>
</tr>
</tbody>
</table>

**CHRONIC EXPOSURE**

<table>
<thead>
<tr>
<th>Cancer</th>
<th>Key to figure</th>
<th>Species</th>
<th>Exposure</th>
<th>NOAEL</th>
<th>LOAEL (effect)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>38</td>
<td>11</td>
<td>Rat</td>
<td>18 mo</td>
<td></td>
<td>20 (CEL, multiple organs)</td>
<td>Wong et al. 1982</td>
</tr>
<tr>
<td>39</td>
<td></td>
<td>Rat</td>
<td>89–104 wk</td>
<td></td>
<td>10 (CEL, lung tumors, nasal tumors)</td>
<td>NTP 1982</td>
</tr>
<tr>
<td>40</td>
<td></td>
<td>Mouse</td>
<td>79–103 wk</td>
<td></td>
<td>10 (CEL, lung tumors, hemangiosarcomas)</td>
<td>NTP 1982</td>
</tr>
</tbody>
</table>

<sup>a</sup> The number corresponds to entries in Figure 2-1.

<sup>b</sup> Used to derive an intermediate inhalation Minimal Risk Level (MRL) of $5 \times 10^3$ 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).

CEL = cancer effect level; d = days(s); hr = hour(s); LOAEL = lowest-observed-adverse-effect level; mo = month(s); NOAEL = no-observed-adverse-effect level; Resp = respiratory; wk = week(s)
Figure 2-1. Levels of Significant Exposure to [Chemical X] – Inhalation

**Acute**
(≤14 days)

- **Systemic**
  - Death
  - Respiratory
  - Hematological

**Intermediate**
(15-364 days)

- **Systemic**
  - Death
  - Respiratory
  - Hematological
  - Hepatic
  - Reproductive

**Cancer**

- *(ppm)*

### Key
- r Rat
- m Mouse
- h Rabbit
- g Guinea Pig
- k Monkey

- **LOAEL for serious effects (animals)**
- **LOAEL for less serious effects (animals)**
- **NOAEL (animals)**
- **CEL - Cancer Effect Level**

* 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.

**Estimated Upper Bound Human Cancer Risk Levels**

- $10^{-4}$
- $10^{-5}$
- $10^{-6}$
- $10^{-7}$
Chapter 2 (Section 2.5)

Relevance to Public Health

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.

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 2.7, “Interactions with Other Substances,” and 2.8, “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
AML  acute myeloid leukemia
atm  atmosphere
ATSDR  Agency for Toxic Substances and Disease Registry
BCF  bioconcentration factor
BEI  Biological Exposure Index
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
Ci  curie
CLP  Contract Laboratory Program
cm  centimeter
CML  chronic myeloid leukemia
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
F₁  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
IARC  International Agency for Research on Cancer
ILO  International Labor Organization
in  inch
Kd  adsorption ratio
kg  kilogram
kg  metric ton
K_{oc}  organic carbon partition coefficient
K_{ow}  octanol-water partition coefficient
L  liter
LC  liquid chromatography
LC_{50}  lethal concentration, low
LC_{50}  lethal concentration, 50% kill
LD_{10}  lethal dose, low
LD_{50}  lethal dose, 50% kill
LOAEL  lowest-observed-adverse-effect level
LSE  Levels of Significant Exposure
m  meter
MA  trans,trans-muconic acid
mCi  millicurie
mg  milligram
min  minute
mL  milliliter
mm  millimeter
mm Hg  millimeters of mercury
mmol  millimole
mo  month
mppcf  millions of particles per cubic foot
MRL  Minimal Risk Level
MS  mass spectrometry
NCE  normochromatic erythrocytes
NIEHS  National Institute of Environmental Health Sciences
NIOSH  National Institute for Occupational Safety and Health
NIOSH/TIC  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
PEL  permissible exposure limit
PCE  polychromatic erythrocytes
pg  picogram
pmol  picomole
PHS  Public Health Service
PMR  proportionate mortality ratio
ppb  parts per billion
ppm  parts per million
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>ppt</td>
<td>parts per trillion</td>
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<tr>
<td>REL</td>
<td>recommended exposure limit</td>
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<tr>
<td>RfD</td>
<td>Reference Dose</td>
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<tr>
<td>RTECS</td>
<td>Registry of Toxic Effects of Chemical Substances</td>
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<tr>
<td>sec</td>
<td>second</td>
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<tr>
<td>SCE</td>
<td>sister chromatid exchange</td>
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<td>SIC</td>
<td>Standard Industrial Classification</td>
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<td>SMR</td>
<td>standard mortality ratio</td>
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<td>STEL</td>
<td>short term exposure limit</td>
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<td>STORET</td>
<td>STORAGE and RETRIEVAL</td>
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<tr>
<td>TLV</td>
<td>threshold limit value</td>
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<tr>
<td>TSCA</td>
<td>Toxic Substances Control Act</td>
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<tr>
<td>TRI</td>
<td>Toxics Release Inventory</td>
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<tr>
<td>TWA</td>
<td>time-weighted average</td>
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<tr>
<td>UMDNJ</td>
<td>University of Medicine and Dentistry New Jersey</td>
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<tr>
<td>U.S.</td>
<td>United States</td>
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<tr>
<td>UF</td>
<td>uncertainty factor</td>
</tr>
<tr>
<td>yr</td>
<td>year</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
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<table>
<thead>
<tr>
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<tr>
<td>&gt;</td>
<td>greater than</td>
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<td>≥</td>
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<tr>
<td>≈</td>
<td>approximately equal to</td>
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<td>&lt;</td>
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