DI-n-BUTYL PHTHALATE A-1

## **APPENDIX A**

# ATSDR MINIMAL RISK LEVEL AND WORKSHEETS

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

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

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

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

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

# MINIMAL RISK LEVEL (MRL) WORKSHEET

Chemical Name: Di-*n*-butyl phthalate

CAS Number: 84-74-2 Date: June 30, 2001

Profile Status: Final

Route: [] Inhalation [X] Oral

Duration: [X] Acute [] Intermediate [] Chronic

Key to Figure: 21 Species: Rats

Minimal Risk Level: 0.5 [X] mg/kg/day [] mg/m<sup>3</sup>

<u>Reference</u>: Mylchreest E, Wallace DG, Cattley RC, et al. 2000. Dose-dependent alterations in androgen-regulated male reproductive development in rats exposed to di(n-butyl) phthalate during late gestation. Toxicol Sci 55:143-151.

Experimental design (human study details or strain, number of animals per exposure/control groups, sex, dose administration details): Groups of 20 pregnant Sprague-Dawley rats (except 11 in the 500 mg/kg/day group) were treated with 0, 0.5, 5, 50, 100, or 500 mg/kg/day di-*n*-butyl phthalate by gavage in corn oil on gestation days 12–21. Dams were examined daily for clinical signs of toxicity, body weight was recorded daily during treatment, and food intake was recorded twice weekly. Dams were killed on postpartum day 21, examined grossly, and the weights of liver, kidneys, adrenals, uterus, and ovaries, and numbers of implantation sites were recorded. On postpartum day 1, numbers of live and dead pups, clinical signs of toxicity, pup weight, and ano-genital distance were recorded. Additionally, the location and number of nipples and areolas were recorded for male pups on postpartum day 14. From postpartum day 30 (females) or 38 (males), pups were examined daily for vaginal opening or preputial separation, respectively, until occurrence. Pups were killed at sexual maturity (postpartum day 110±10 days for males; 80±5 days for females), weighed, and internal and external genitalia were examined for malformations and undescended testes. Organs were weighed, and right testis and epididymides were examined histologically.

Effects noted in study and corresponding concentrations: No developmental effects or clinical signs of toxicity were seen in pups exposed to 50 mg/kg/day or less in utero. The only treatment-related effect seen in the 100 mg/kg/day group was retained areolas and nipples in males (normally seen only in females) on postpartum day 14. This effect was dose-dependent, being more prominent at 500 mg/kg/day. Permanence of retained areolas and nipples was not assessed. Other statistically significant effects, seen only in males at 500 mg/kg/day level, were decreased AGD (12% decrease) and ano-genital distance/body weight at birth; smaller epididymides, dorsolateral prostate, and levator ani-bulbocavernosus muscle at sexual maturity; and decreased testes weight (when animals with enlarged testes were excluded). Malformations of the male reproductive tract included absent or malformed epididymis, absent or malformed vas deferens, hypospadia, and unilaterally absent seminal vesicle. Histopathological lesions of the testis seen were seminiferous tubule degeneration, focal interstitial hyperplasia, and adenoma. In males, no significant changes were seen in age at preputial separation, or body, kidney, liver, adrenal, ventral prostate, vas deferens, seminal vesicle, or other organ weights (other than those listed above). In female pups, no significant differences from controls were seen in age of onset of vaginal opening; body, liver, kidney, adrenal, ovary, or uterus weight; or gross morphology of the reproductive organs at sexual maturity.

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<u>Concentration and end point used for MRL derivation</u> : Increased incidence of retained areolas and nipple in the male offspring of rats exposed to \$100 mg//kg/day; no effects were observed at 50 mg/kg/day.
[X] NOAEL [] LOAEL
<u>Uncertainty Factors used in MRL derivation</u> :
<ul> <li>[ ] 10 for use of a LOAEL</li> <li>[X] 10 for extrapolation from animals to humans</li> <li>[X] 10 for human variability</li> </ul>

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

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

Was a conversion used from intermittent to continuous exposure? NA

Other additional studies or pertinent information that lend support to this MRL: A number of developmental effects involving androgenic-dependent tissues have been observed in acutely or longer exposed rats; these include decreases in anogenital distance and anogenital distance/body weight ratio, increased number of retained nipples, and decreased androgen-dependent tissue weights (ventral prostate, epididymis, cauda epididymis, testes, glans penis, and levator ani-bulbocavernosus) (Ema et al. 1998, 2000b; Gray et al. 1999; Mylchreest et al. 1999, 2000).

Agency Contact (Chemical Manager): Jessilynn Taylor

DI-n-BUTYL PHTHALATE B-1

## **APPENDIX B**

#### **USER'S GUIDE**

# Chapter 1

#### Public Health Statement

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

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

## Chapter 2

### Relevance to Public Health

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

- 1. What effects are known to occur in humans?
- 2. What effects observed in animals are likely to be of concern to humans?
- 3. What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

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

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

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

## Interpretation of Minimal Risk Levels

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

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

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

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

# Chapter 3

# **Health Effects**

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

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

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

## **LEGEND**

#### See LSE Table 3-1

- (1) Route of Exposure One of the first considerations when reviewing the toxicity of a substance using these tables and figures should be the relevant and appropriate route of exposure. When sufficient data exists, three LSE tables and two LSE figures are presented in the document. The three LSE tables present data on the three principal routes of exposure, i.e., inhalation, oral, and dermal (LSE Table 3-1, 3-2, and 3-3, respectively). LSE figures are limited to the inhalation (LSE Figure 3-1) and oral (LSE Figure 3-2) routes. Not all substances will have data on each route of exposure and will not therefore have all five of the tables and figures.
- (2) Exposure Period Three exposure periods acute (less than 15 days), intermediate (15–364 days), and chronic (365 days or more) are presented within each relevant route of exposure. In this example, an inhalation study of intermediate exposure duration is reported. For quick reference to health effects occurring from a known length of exposure, locate the applicable exposure period within the LSE table and figure.
- (3) <u>Health Effect</u> 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) <u>Key to Figure</u> Each key number in the LSE table links study information to one or more data points using the same key number in the corresponding LSE figure. In this example, the study represented by key number 18 has been used to derive a NOAEL and a Less Serious LOAEL (also see the 2 "18r" data points in Figure 3-1).
- (5) Species The test species, whether animal or human, are identified in this column. Chapter 2, "Relevance to Public Health," covers the relevance of animal data to human toxicity and Section 3.4, "Toxicokinetics," contains any available information on comparative toxicokinetics. Although NOAELs and LOAELs are species specific, the levels are extrapolated to equivalent human doses to derive an MRL.
- (6) Exposure Frequency/Duration The duration of the study and the weekly and daily exposure regimen are provided in this column. This permits comparison of NOAELs and LOAELs from different studies. In this case (key number 18), rats were exposed to 1,1,2,2-tetrachloroethane via inhalation for 6 hours per day, 5 days per week, for 3 weeks. For a more complete review of the dosing regimen refer to the appropriate sections of the text or the original reference paper, i.e., Nitschke et al. 1981.
- (7) <u>System</u> 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) <u>NOAEL</u> 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) <u>LOAEL</u> A Lowest-Observed-Adverse-Effect Level (LOAEL) is the lowest dose used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects. These distinctions help readers identify the levels of exposure at which adverse health effects first appear and the gradation of effects with increasing dose. A brief description of the specific end point used to quantify the adverse effect accompanies the LOAEL. The respiratory effect reported in key number 18 (hyperplasia) is a Less serious LOAEL of 10 ppm. MRLs are not derived from Serious LOAELs.
- (10) <u>Reference</u> The complete reference citation is given in Chapter 9 of the profile.
- (11) <u>CEL</u> 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) <u>Footnotes</u> Explanations of abbreviations or reference notes for data in the LSE tables are found in the footnotes. Footnote "b" indicates the NOAEL of 3 ppm in key number 18 was used to derive an MRL of 0.005 ppm.

#### LEGEND

## See Figure 3-1

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

- (13) Exposure Period The same exposure periods appear as in the LSE table. In this example, health effects observed within the intermediate and chronic exposure periods are illustrated.
- (14) <u>Health Effect</u> These are the categories of health effects for which reliable quantitative data exists. The same health effects appear in the LSE table.
- (15) <u>Levels of Exposure</u> 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<sup>3</sup> or ppm and oral exposure is reported in mg/kg/day.
- (16) NOAEL In this example, 18r NOAEL is the critical end point for which an intermediate inhalation exposure MRL is based. As you can see from the LSE figure key, the open-circle symbol indicates to a NOAEL for the test species-rat. The key number 18 corresponds to the entry in the LSE table. The dashed descending arrow indicates the extrapolation from the exposure level of 3 ppm (see entry 18 in the Table) to the MRL of 0.005 ppm (see footnote "b" in the LSE table).
- (17) <u>CEL</u> 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 (q<sub>1</sub>\*).
- (19) Key to LSE Figure The Key explains the abbreviations and symbols used in the figure.

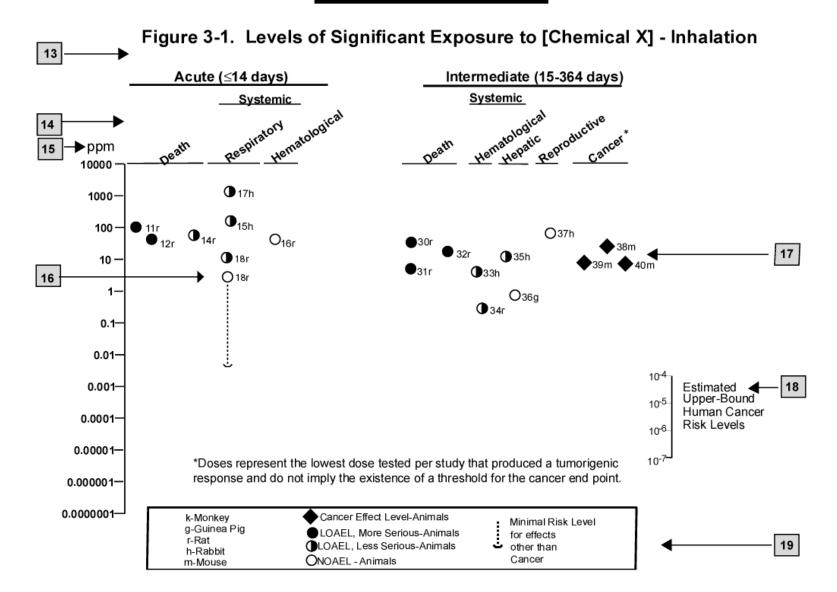
# SAMPLE

Table 3-1. Levels of Significant Exposure to [Chemical x] – Inhalation Exposure LOAEL (effect) Key to frequency/ **NOAEL** Less serious (ppm) Serious (ppm) figurea **Species** duration System (ppm) Reference INTERMEDIATE EXPOSURE 5 8 9 10 6 Systemic 9 9 9 9 9 9 18 Rat 13 wk  $3^{b}$ 10 (hyperplasia) Nitschke et al. Resp 5 d/wk 1981 6 hr/d CHRONIC EXPOSURE 11 Cancer 9 Wong et al. 1982 38 Rat 18 mo (CEL, multiple 5 d/wk organs) 7 hr/d (CEL, lung tumors, 39 89-104 wk Rat NTP 1982 5 d/wk nasal tumors) 6 hr/d 40 79-103 wk (CEL, lung tumors, NTP 1982 Mouse 5 d/wk hemangiosarcomas) 6 hr/d

<sup>&</sup>lt;sup>a</sup> The number corresponds to entries in Figure 3-1.

<sup>&</sup>lt;sup>b</sup> Used to derive an intermediate inhalation Minimal Risk Level (MRL) of 5 x 10<sup>-3</sup> 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).

# SAMPLE



DI-n-BUTYL PHTHALATE C-1

## APPENDIX C

# **ACRONYMS, ABBREVIATIONS, AND SYMBOLS**

ACGIH American Conference of Governmental Industrial Hygienists

ADI Acceptable Daily Intake

ADME Absorption, Distribution, Metabolism, and Excretion

AFID alkali flame ionization detector

AFOSH Air Force Office of Safety and Health

AML acute myeloid leukemia

AOAC Association of Official Analytical Chemists

atm atmosphere

ATSDR Agency for Toxic Substances and Disease Registry

AWQC Ambient Water Quality Criteria
BAT Best Available Technology
BCF bioconcentration factor
BEI Biological Exposure Index
BSC Board of Scientific Counselors

C Centigrade CAA Clean Air Act

CAG Cancer Assessment Group of the U.S. Environmental Protection Agency

CAS Chemical Abstract Services

CDC Centers for Disease Control and Prevention

CEL Cancer Effect Level

CELDS Computer-Environmental Legislative Data System

CERCLA Comprehensive Environmental Response, Compensation, and Liability Act

CFR Code of Federal Regulations

Ci curie

CL ceiling limit value

CLP Contract Laboratory Program

cm centimeter

CML chronic myeloid leukemia CNS central nervous system

CPSC Consumer Products Safety Commission

CWA Clean Water Act

d day Derm dermal

DHEW Department of Health, Education, and Welfare DHHS Department of Health and Human Services

DNA deoxyribonucleic acid DOD Department of Defense DOE Department of Energy DOL Department of Labor

DOT Department of Transportation

DOT/UN/ Department of Transportation/United Nations/

NA/IMCO North America/International Maritime Dangerous Goods Code

DWEL Drinking Water Exposure Level ECD electron capture detection

ECG/EKG electrocardiogram

# DI-*n*-BUTYL PHTHALATE APPENDIX C

EEG electroencephalogram

EEGL Emergency Exposure Guidance Level EPA Environmental Protection Agency

F Fahrenheit

F<sub>1</sub> 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

ft foot

FR Federal Register

g gram

GC gas chromatography Gd gestational day gen generation

GLC gas liquid chromatography GPC gel permeation chromatography

HPLC high-performance liquid chromatography

hr hour

HRGC high resolution gas chromatography HSDB Hazardous Substance Data Bank

IDLH Immediately Dangerous to Life and Health IARC International Agency for Research on Cancer

ILO International Labor Organization

in inch

IRIS Integrated Risk Information System

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

 $\begin{array}{ccc} LD_{Lo} & & lethal\ dose,\ low \\ LD_{50} & & lethal\ dose,\ 50\%\ kill \\ LT_{50} & & lethal\ time,\ 50\%\ kill \end{array}$ 

LOAEL lowest-observed-adverse-effect level LSE Levels of Significant Exposure

m meter

MA trans, trans-muconic acid MAL Maximum Allowable Level

mCi millicurie

MCL Maximum Contaminant Level MCLG Maximum Contaminant Level Goal

mg milligram min minute mL milliliter

# DI-*n*-BUTYL PHTHALATE APPENDIX C C-3

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

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 NCI National Cancer Institute

NIEHS National Institute of Environmental Health Sciences NIOSH National Institute for Occupational Safety and Health NIOSHTIC NIOSH's Computerized Information Retrieval System

NFPA National Fire Protection Association

ng nanogram

NLM National Library of Medicine

nm nanometer

NHANES National Health and Nutrition Examination Survey

nmol nanomole

NOAEL no-observed-adverse-effect level
NOES National Occupational Exposure Survey
NOHS National Occupational Hazard Survey

NPD nitrogen phosphorus detection

NPDES National Pollutant Discharge Elimination System

NPL National Priorities List

NR not reported

NRC National Research Council

NS not specified

NSPS New Source Performance Standards
NTIS National Technical Information Service

NTP National Toxicology Program ODW Office of Drinking Water, EPA

OERR Office of Emergency and Remedial Response, EPA

OHM/TADS Oil and Hazardous Materials/Technical Assistance Data System

OPP Office of Pesticide Programs, EPA

OPPTS Office of Prevention, Pesticides and Toxic Substances, EPA

OPPT Office of Pollution Prevention and Toxics, EPA
OSHA Occupational Safety and Health Administration

OSW Office of Solid Waste, EPA OTS Office of Toxic Substances

OW Office of Water

OWRS Office of Water Regulations and Standards, EPA

PAH Polycyclic Aromatic Hydrocarbon

PBPD Physiologically Based Pharmacodynamic PBPK Physiologically Based Pharmacokinetic

PCE polychromatic erythrocytes PEL permissible exposure limit PID photo ionization detector

# DI-*n*-BUTYL PHTHALATE APPENDIX C C-4

pg picogram pmol picomole

PHS Public Health Service PMR proportionate mortality ratio

ppb parts per billion ppm parts per million ppt parts per trillion

PSNS Pretreatment Standards for New Sources
REL recommended exposure level/limit

RfC Reference Concentration

RfD Reference Dose RNA ribonucleic acid

RTECS Registry of Toxic Effects of Chemical Substances

RQ Reportable Quantity

SARA Superfund Amendments and Reauthorization Act

SCE sister chromatid exchange

sec second

SIC Standard Industrial Classification

SIM selected ion monitoring

SMCL Secondary Maximum Contaminant Level

SMR standard mortality ratio

SNARL Suggested No Adverse Response Level

SPEGL Short-Term Public Emergency Guidance Level

STEL short term exposure limit STORET Storage and Retrieval

TD<sub>50</sub> toxic dose, 50% specific toxic effect

TLV threshold limit value
TOC Total Organic Compound
TPQ Threshold Planning Quantity
TRI Toxics Release Inventory
TSCA Toxic Substances Control Act
TRI Toxics Release Inventory
TWA time-weighted average

U.S. United States
UF uncertainty factor

VOC Volatile Organic Compound

yr year

WHO World Health Organization

wk week

> greater than

 $\geq$  greater than or equal to

= equal to less than

 $\leq$  less than or equal to

 $\begin{array}{lll} \% & & \text{percent} \\ \alpha & & \text{alpha} \\ \beta & & \text{beta} \\ \gamma & & \text{gamma} \\ \delta & & \text{delta} \\ \mu m & & \text{micrometer} \end{array}$ 

#### C-5 DI-n-BUTYL PHTHALATE APPENDIX C

microgram cancer slope factor negative positive weakly positive result weakly negative result μg q<sub>1</sub>\* -+

(+) (-)

DI-*n*-BUTYL PHTHALATE

# **APPENDIX D**

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