



# OPPT Chemical Fact Sheets

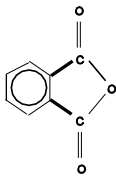
## (Phthalic Anhydride) Fact Sheet: Support Document (CAS No. 85-44-9)

This summary is based on information retrieved from a systematic search limited to secondary sources (see Appendix A). These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials. No attempt has been made to verify information in these databases and secondary sources.

### I. CHEMICAL IDENTITY AND PHYSICAL/CHEMICAL PROPERTIES

The chemical identity and physical/chemical properties of phthalic anhydride are summarized in Table 1.

**TABLE 1. CHEMICAL IDENTITY AND CHEMICAL/PHYSICAL PROPERTIES OF PHTHALIC ANHYDRIDE**

Characteristic/Property	Data	Reference
CAS No.	85-44-9	
Common Synonyms	1,2-benzenedicarboxylic acid anhydride; 1,3-dihydro-1,3-dioxoisobenzofurandione; 1,3-phthalandion; phthalandione	U.S. EPA 1994
Molecular Formula	$C_8H_4O_3$	
Chemical Structure		
Physical State	white solid;	
Molecular Weight	148.12	U.S. EPA 1986
Melting Point	131 °C	U.S. EPA 1986
Boiling Point	284 °C (sublimes)	Keith and Walters 1985
Water Solubility	6.2 g/L @ 25 °C	U.S. EPA 1986
Density	1.527 g/mL @ 20 °C	Keith and Walters 1985
Vapor Density (air = 1)	5.1	Keith and Walters 1985
$K_{oc}$	36 (estimated)	U.S. EPA 1986
Log $K_{ow}$	1.6 (measured)	Hansch and Leo 1987
Vapor Pressure	0.00052 mm Hg @ 25 °C	U.S. EPA 1986
Reactivity	hydrolyses in water	U.S. EPA 1986
Flash Point	152 °C (closed cup)	ACGIH 1991
Henry's Law Constant	$1.6 \times 10^{-8}$ atm·m <sup>3</sup> /mol (calculated)	U.S. EPA 1986
Fish Bioconcentration Factor	5 (calculated)	CHEMFATE 1994
Odor Threshold	0.053 ppm; air, v/v	U.S. EPA 1986
Conversion Factors	1 ppm = 6.06 mg/m <sup>3</sup> 1 mg/m <sup>3</sup> = 0.165 ppm	U.S. EPA 1986

## II. PRODUCTION, USE, AND TRENDS

### A. Production

USITC (1994) lists seven producers of phthalic anhydride in the United States in 1992 (see Table 3). In 1992, U.S. companies produced 896 million pounds (407 million kg) of phthalic anhydride. The U.S. imported 53 million pounds and exported 46 million pounds of phthalic anhydride in 1992 (Mannsville 1993).

According to Mannsville (1993), there were five US producers of phthalic anhydride in 1993, with a combined total production capacity of 990 million pounds. Table 2 shows U.S. phthalic anhydride producers, plant locations, and plant capacities for 1993.

**TABLE 2. U.S. PRODUCERS OF PHTHALIC ANHYDRIDE AND THEIR CAPACITIES IN 1993**

Producer (Millions of Pounds)	Location	Plant Capacity
Aristech (Mitsubishi)	Pasadena, TX	230
Exxon Chemical	Baton Rouge, LA	250
Koppers Industries	Cicero, IL	165
Stepan Chemical	Millsdale, IL	170
Sterling Chemical Company	Texas City, TX	175
TOTAL		990

Source: Mannsville 1993.

**TABLE 3. U.S. PRODUCTION AND SALES VALUE OF PHTHALIC ANHYDRIDE IN 1991**

Production (1,000 Kg)	Sales Quantity (1,000 Kg)	Sales Value (\$1,000)	Average Unit Value (Per Kg)
407,350	162,965	115,698	\$0.71

Source: USITC 1994.

## B. Uses

Phthalic anhydride is used principally to produce phthalate plasticizers used to compound flexible polyvinyl chloride. Phthalic anhydride is also used to make unsaturated polyesters that are used to manufacture fiberglass-reinforced plastics. Companies use these plastics to fabricate shower stalls, synthetic marble, translucent construction panels, boats, and recreation vehicles. Companies also use phthalic anhydride to make alkyd resins used primarily in solvent-borne protective coatings in the paint industry. Companies use phthalic anhydride to make halogenated anhydrides used as fire retardants; polyester polyols for urethanes; phthalocyanine pigments; dyes; perfumes; pharmaceuticals; tanning and curing agents; solvents; insect repellents; and various chemical intermediates.

**TABLE 4. END USE PATTERN OF PHTHALIC ANHYDRIDE--1992 ESTIMATE**

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Derivative (Typical Standard Industrial Classification (SIC) Code) <sup>1</sup>	Percent
Phthalate Plasticizers (production, SIC 2869)	53
Unsaturated Polyesters (production, SIC 2821)	22
Alkyd Resins (production, SIC 2821)	18
Miscellaneous (various SICs)	7

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Source: Mannsville 1993.

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<sup>1</sup> The Standard Industrial Classification (SIC) code is the statistical classification standard for all Federal economic statistics. The code provides a convenient way to reference economic data on industries of interest to the researcher. SIC codes presented here are not intended to be an exhaustive listing; rather, the codes listed should provide an indication of where a chemical may be likely to be found in commerce.

### C. Trends

The decline in production of phthalic anhydride from 1,040 million pounds in 1988, to 935 million pounds in 1989, was a result of product innovations, technology changes, and product downsizing such as that occurring in the automotive industry. However, annual output growth of phthalic anhydride is expected to recover and increase by two to three percent per year in the future. The production of phthalic anhydride based polyols and unsaturated polyesters used for reinforced plastics in transportation and construction is expected to increase three to five percent. The long-term demand for solvents based alkyd coatings or paints, on the other hand, will decline. In addition, use of phthalic anhydride as an intermediate for the production of dyes and saccharin will decline due to decreased demand for these products. The overall demand for plasticizers will remain even, growing by GNP rates. The demand for dioctyl phthalate, a primary plasticizer, will drop because of stronger environmental regulations regarding the use, handling, and disposal of the material; non-phthalate plasticizers marketed as substitutes will also affect demand for phthalic anhydride (Mannsville 1993).

**TABLE 5. U.S. PRODUCTION AND CAPACITY OF PHTHALIC ANHYDRIDE (MILLIONS OF POUNDS)**

Year	1990	1991	1992	1993 (Projected)	1995 (Projected)
Capacity	955	975	990	990	1070
Production	938	872	896	N/A	N/A

N/A: Not available

Source: Mannsville 1993.

### III. ENVIRONMENTAL FATE

#### A. Environmental Release

Phthalic anhydride is released to the environment from chemical plants, mainly those that manufacture the chemical or use it in the production of plastics and resins (U.S. EPA 1986). The major sources of these releases are process off-gases and industrial effluents; however, the use of catalytic oxidation now reduces the release of pollutants in off-gases (U.S. EPA 1986). Phthalic anhydride has been detected in arctic air at the concentration of  $\sim 10 \text{ ng/m}^3$  (however, the EPA review notes that the presence of the anhydride could have resulted from the hydrolysis of phthalate esters followed by dehydration in the GC injection port), and has been identified but not quantified in U.S. drinking water and in the volatile flavor components of baked Idaho potatoes (U.S. EPA 1986).

In 1992, environmental releases of the chemical, as reported to the Toxic Chemical release inventory by certain types of US industries, totaled about 756 thousand pounds, including 750 thousand to the atmosphere, 1 thousand pounds to land, and 5 thousand pounds to surface water (TRI92 1994).

## B. Transport

No information was found for the transport of atmospheric phthalic anhydride. However, the water solubility of the chemical suggests that wet deposition may occur prior to its conversion to phthalic acid, a less soluble chemical. Because vapor phase particle adsorption usually occurs with chemicals having a vapor pressure of  $\leq 10^{-7}$  mm Hg (U.S. EPA 1986), adsorption is not a likely vapor phase removal mechanism for phthalic anhydride.

No information was found for the transport of phthalic anhydride in the aquatic environment. However, the rapid hydrolysis of phthalic anhydride to phthalic acid that occurs in aqueous media, would preclude any significant transport of the chemical in the aquatic environment. Its low vapor pressure and Henry's Law constant ( $1.6 \times 10^{-8}$  atm·m<sup>3</sup>/mol) indicate that the chemical will slowly volatilize from water (U.S. EPA 1986). The chemical is soluble in water (6,200 mg/L @ 25 °C) and has a low  $K_{oc}$  (36, estimated) and therefore has a low potential for sedimentation or adsorption to particles, respectively.

No information was found for the transport of phthalic anhydride in soil. In moist soil, the chemical will hydrolyze to phthalic acid and significant leaching is not expected to occur, other than in the case of a large spill (U.S. EPA 1994). Significant volatilization from soil is also unlikely based on the chemical's low vapor pressure (0.00052 mm Hg).

## C. Transformation/Persistence

1. Air — The rate constant for the reaction of phthalic anhydride with OH radicals is estimated to be  $5.0 \times 10^{-13}$  cm<sup>3</sup> molecule<sup>-1</sup> sec<sup>-1</sup> (U.S. EPA 1986). Assuming the concentration of OH<sup>-</sup> in the atmosphere to be  $10^6$  molecules per cubic centimeter, the estimated half-life for this reaction is 21 days (Chemfate 1994).
2. Soil — No information was found regarding the fate and transport of phthalic anhydride in the soil (U.S. EPA 1986). Based on its rapid hydrolysis in aqueous media, the chemical is expected to hydrolyze in moist soil. Phthalic anhydride is also expected to undergo biodegradation in soil, as it does in water (see below) (U.S. EPA 1986). Under aerobic soil conditions, phthalic anhydride probably has a half-life of >14 days (U.S. EPA 1988a).

Based on the presence of phthalic anhydride in regulated and unregulated waste streams and in contaminated soil, groundwater, or surface water resulting from hazardous waste mismanagement incidents, the U.S. EPA (1988b) (under section 4 of the Toxic Substances Control Act) required testing of phthalic anhydride for soil adsorption. The soil adsorption isotherm test was required, in accordance with §796.2750 (U.S. EPA 1988b). Optional biodegradation testing was also recommended.

3. Water — Hydrolysis and biodegradation are the two processes that may significantly degrade phthalic anhydride in water. The hydrolytic half-life for the chemical is about 1.5 minutes (calculated, based on the rate constant for the forward reaction in aqueous solution,  $7.9 \times 10^{-3}$  sec<sup>-1</sup> @ 25 °C) (U.S. EPA 1986). Biodegradation values, reported in U.S. EPA 1986 for various wastewater treatment conditions are as follows:
  - \* 44-78% mineralization in 5 days based on theoretical biological oxygen demand (incubation of 1-4 mg/L of phthalic anhydride with sewage as microbial inoculum);
  - \* ~21% degradation in 5 days using the standard dilution method (incubation of 2 mg/L with sewage);
  - \* ~18% degradation using the seawater dilution method;
  - \* 33% degradation in 24 hours using chemical oxygen demand removal (incubation of 9 mg/L phthalic anhydride with activated sludge); and
  - \* >30% degradation in 2 weeks using the Japanese MITI test (100 mg/L phthalic anhydride incubated with activated sludge, pH 7 @ 25 °C).

In addition, phthalic acid was 50% degraded in 1.5 weeks in the river die-away test (using water from the Mississippi River) and phthalic anhydride was almost completely converted to phthalic acid in a model aquatic ecosystem (U.S. EPA 1986).

4. Biota — Phthalic anhydride, having a calculated BCF (bioconcentration factor) value of 5, is not expected to bioaccumulate in aquatic organisms (U.S. EPA 1986). Studies using a model ecosystem reported log BCF values of 1.28-1.90 for daphnids, mosquito larvae, pouch snail, and mosquito fish 24-48 hours after exposure to radiolabeled phthalic acid (U.S. EPA 1986).

Plants and animals exposed to radiolabeled di-2-ethylhexyl phthalate (DEHP) in a microecosystem contained, but did not accumulate to any great extent, phthalic anhydride, a metabolite of DEHP. The initial concentrations of DEHP, 1-2 mg/L, declined to 0.001 mg/L after 28 days. The levels of phthalic anhydride in the various species (in mg/kg as di-2-ethylhexyl phthalate equivalents) are as follows: *Chara chara* (plant), 4.46; *Mentha aquatica* (plant), 3.66; *Gammarus pulex* (amphipod), 0.49; *Limnephilus* sp. (caddisfly), 18.99; *Helobdella* sp. (leech), 0.88; *Dendrocoelum lacteum* (flatworm), 0.21; and *Lampetra planeri* (lamprey), 0.56 (U.S. EPA 1986).

## IV. HEALTH EFFECTS

### A. Pharmacokinetics

1. Absorption — Oral toxicity data for animals, and other human and animal data provide indirect evidence that the phthalic anhydride or its hydrolysis product, phthalic acid, is absorbed from the gastrointestinal tract and lungs. No information was found concerning dermal absorption was found. Phthalic anhydride is a skin irritant, and skin damage is known to result in increased absorption of chemicals (U.S. EPA 1988a).
2. Distribution — Limited information was found on the distribution of phthalic anhydride or its hydrolysis product, phthalic acid. One study, using an exposure route of questionable relevance to human exposure, indicates that fetuses from CD-1 fetal mice injected intraperitoneally with 80 mg/kg <sup>14</sup>C-phthalic anhydride on days 11, 12, and 13 of gestation exhibited covalently bound radioactivity in all tissues (U.S. EPA 1986).
3. Metabolism — Phthalic anhydride is expected to undergo hydrolysis to phthalic acid in the aqueous media of the body (U.S. EPA 1988a). No information on the metabolism of phthalic anhydride was found in the secondary sources searched.
4. Excretion — Humans exposed to phthalic anhydride in workplace air excreted phthalic acid in the urine as free acid (U.S. EPA 1988a).

### B. Acute Effects

Acute exposure to phthalic anhydride produces irritation of the eyes, skin and respiratory tract and lung sensitization in humans. Phthalic anhydride causes sensitization in animals as well.

1. Humans — The acute toxicity of phthalic anhydride is characterized by irritation of the eyes and skin, allergic rhinitis, and asthma (U.S. EPA 1988a).
2. Animals — LD<sub>50</sub> values for phthalic anhydride administered orally to animals are 800 to 1600 mg/kg for rats and 2210 mg/kg for mice (ACGIH 1991). Guinea pigs were sensitized to 0.1% phthalic anhydride following both intracutaneous and inhalation exposure (U.S. EPA 1986).

## C. Subchronic/Chronic Effects

Workers exposed to moderate to high concentrations of atmospheric phthalic anhydride have experienced irritation of the eyes, skin, and respiratory tract, and have developed hypersensitivity, bronchial asthma, and emphysema. Animals exposed chronically to high concentrations of phthalic anhydride in the diet exhibited lung, kidney, and adrenal gland toxicity. EPA has derived a chronic reference dose of 2 mg/kg/day for phthalic anhydride.

1. **Humans** — Workers exposed to atmospheric phthalic anhydride powder at levels of ~24.9 to 77.3 ppm (21 to 67 mg/kg/day<sup>1</sup>) experienced higher incidences of irritation of the eye and respiratory tract than workers exposed to ≤1.1 ppm. Irritation of the skin has also been reported in workers (U.S. EPA 1986). Case studies document the induction of hypersensitivity and bronchial asthma in humans exposed to phthalic anhydride dust; specific antibody binding against the compound has been reported (U.S. EPA 1986). Workers currently or formerly employed in plants producing alkyd and/or polyunsaturated resins were evaluated for respiratory ailments (ACGIH 1991). Time-weighted-average breathing zone samples measured 3 to 13 mg/m<sup>3</sup> in areas where bags containing flaked phthalic anhydride were cut open and emptied manually into reactors several times a day during a 10- to 30-minute period. Of the 118 individuals examined, 24% had rhinitis, 11% had productive bronchitis, and 18% had work-associated asthma. The latent period for the symptoms ranged from 1 to 16 years (ACGIH 1991).

Workers exposed to mixtures of phthalic anhydride and phthalic acid developed conjunctivitis, bloody nasal discharge, atrophy of the nasal mucosa, hoarseness, cough, occasional bloody sputum, bronchitis, and emphysema (ACGIH 1991). Air concentrations of 30 mg/m<sup>3</sup> (5 ppm) and 25 mg/m<sup>3</sup> (4 ppm) were associated with conjunctivitis and mucous membrane irritation, respectively.

2. **Animals** — In an NCI bioassay, male and female F344 rats and B6C3F<sub>1</sub> mice were treated with dietary phthalic anhydride for 105 and 104 weeks, respectively (50/sex/group, treated; 20/sex/group, controls) (NCI 1979). The rats were exposed to 0, 7,500 or 15,000 ppm (375 or 750 mg/kg/day<sup>2</sup>). The mice were exposed to 0, 25,000, or 50,000 ppm for 32 weeks; however, these concentrations were reduced to 12,500 and 25,000 ppm for the males and 6,250 or 12,500 ppm for the females for the remainder of the study because of severe weight loss. The time-weighted-average doses were 16,346 or 32,692 ppm (2,215 or 4,250 mg/kg/day<sup>3</sup>) for the male mice and 12,019 and 24,038 ppm (1,562 or 3,125 mg/kg/day) for the female mice. Treatment did not affect the survival of either species and did not induce non-neoplastic gross or microscopic pathology in the rats (see section IV.D. for neoplastic effects).

At the end of the study, the mice exhibited weight loss (12 and 25% for low- and high-dose males, respectively; and 12 and 27% for low- and high-dose females, respectively), along with significantly increased incidences of lung and kidney lymphocytosis (both doses, males and females), chronic bile duct inflammation (high-dose males and females), dose-related adrenal atrophy (low- and high-dose males)(U.S.EPA 1994). Based on these findings, the EPA identified the Lowest observed adverse effect (LOAEL of 12,019 ppm for the female mice of the study (U.S. EPA 1994). Using a factor of 0.13 kg food/kg body weight to convert the dose to 1562 mg/kg/day, and dividing by an uncertainty factor of 1000, the EPA calculated

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<sup>1</sup>For dose conversion purposes the inhalation (ppm) dose has been given as an oral dose (mg/kg/day) estimate using the following:  $\text{mg/m}^3 \times \text{air breathed/day (m}^3) \times 1/\text{body weight} = \text{dose (mg/kg/day)}$ ;  $= 145.4 \text{ mg/m}^3 \times 10 \text{ m}^3/\text{day} \times 1/70 \text{ kg} = 21 \text{ mg/kg/day}$ .

<sup>2</sup>For rats, mg/kg/day calculated from ppm x 0.05 food factor (U.S. EPA 1985).

<sup>3</sup>For mice, mg/kg/day converted from ppm x 0.13 food factor.

the chronic oral RfD (reference dose) to be 2 mg/kg/day<sup>4</sup> for phthalic anhydride.

In a range-finding study for the NCI bioassay, groups of 5 rats and 5 mice per sex received up to 50,000 ppm dietary phthalic anhydride for 7 weeks (U.S. EPA 1986). Male and female rats given 50,000 ppm exhibited a ~25% weight loss and males given 25,000 ppm had slight centrilobular vacuolation in the liver. There were no other lesions in the rats and none in the mice.

Cats exposed by inhalation for seven days to 3,700 mg/m<sup>3</sup> (611 ppm) phthalic anhydride exhibited liver and kidney effects (U.S. EPA 1988a).

#### **D. Carcinogenicity**

No information was found for the carcinogenicity of phthalic anhydride in humans. Phthalic anhydride was not carcinogenic in a lifetime dietary study in mice and rats.

1. Humans — No information was found in the secondary sources searched for the carcinogenicity of phthalic anhydride in humans.
2. Animals — In an NCI bioassay, male and female F344 rats and B6C3F<sub>1</sub> mice were treated with dietary phthalic anhydride for 105 and 104 weeks, respectively (50/sex/group, treated; 20/sex/group, controls) (NCI 1979). The rats were exposed to 0, 7,500 or 15,000 ppm. The mice were exposed to a time-weighted average concentration of 16,346 or 32,692 ppm (see section IV.C for further details of exposure). There were no treatment-related effects on survival of either species and no increased tumor incidences in male rats or male and female mice. The female rats showed increases in alveolar/bronchiolar adenoma (0/20, controls; 0/50, low dose; 5/50, high dose) and in lymphomas (1/20, controls; 11/50, low dose; 4/50, high dose). However, the interpretation of the results of the study was complicated by nonsignificant results for lung tumors using the Fisher exact test and an occasionally elevated spontaneous incidence of lymphomas in historical controls. The NCI concluded that the evidence for an association between exposure to phthalic anhydride and the occurrence of these tumors was questionable, and that phthalic anhydride was not carcinogenic for F344 rats and B6C3F<sub>1</sub> mice under the conditions of this study.

#### **E. Genotoxicity**

Phthalic anhydride was non-mutagenic in *Salmonella typhimurium* strains TA98, TA100, TA1535 and TA1537, with and without metabolic activation, at concentrations of 3 µmol/plate in the spot test and at concentrations of 1 to 666 µg/plate in the plate incorporation test (U.S. EPA 1986). Phthalic anhydride was also negative in a *Salmonella* reverse mutation assay and in chromosomal aberration and sister chromatid exchange assays (U.S. EPA 1986).

#### **F. Developmental/Reproductive Toxicity**

Information on potential developmental and reproductive effects of phthalic anhydride in humans and in animals is limited and is not sufficient to support conclusions on these effects. Workers exposed to phthalic anhydride have reported sexual dysfunction.

1. Humans — No information was found in the secondary sources searched to indicate that phthalic anhydride causes developmental toxicity in humans. A report of sexual dysfunction among workers engaged in the manufacture of phthalic anhydride prompted the male reproductive toxicity study described below (an abstract from a Russian study cited in U.S.

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<sup>4</sup>The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during the time period of concern.



EPA 1988a).

2. Animals — Information on possible developmental effects of phthalic anhydride is limited to several intraperitoneal studies in animals. I.p. injection of phthalic anhydride has produced developmental toxicity (mainly dysmorphogenesis) in mice. In one study, injection of CD-1 mice with a minimum of 55.5 mg/kg phthalic anhydride on gestation days 11, 12, and 13 produced "fetal abnormalities", whereas in another, injection of 80 mg/kg on gestation days 8, 9, and 10 produced rib and vertebral malformations in 25.6% of the fetuses (U.S. EPA 1986). In addition, cleft palate was also reported in fetuses treated on days 11-13, but doses were not specified (U.S. EPA 1986). Information on the developmental toxicity of phthalic anhydride administered by other routes of exposure was not found in the secondary sources searched.

An abstract from the Russian literature reports that phthalic anhydride, administered continuously to male rats for 45 days at concentrations of 0.2 or 1 mg/m<sup>3</sup>, decreased the levels of ascorbic, dehydroascorbic, and/or nucleic acids in the seminiferous tubules (U.S. EPA 1988a). No effects were reported for the lowest concentration administered, 0.02 mg/m<sup>3</sup>. Exposures were 24 hours/day for 45 days. Following a 2-week recovery period, the motility of spermatozoa was one-half to one-third that of controls for high- and mid-dose animals, respectively (U.S. EPA 1988a).

The NCI (1979) chronic dietary studies did not demonstrate testicular effects in rodents.

## G. Neurotoxicity

Information is insufficient to characterize the neurotoxicity of phthalic anhydride in humans. Cats exposed to high concentrations of the chemical in air exhibited loss of appetite and drowsiness. Mice exposed to high concentrations of phthalic anhydride in their diet exhibited mineralization of the thalamus.

1. Humans — Six workers employed for an average of 6.6 years in a phthalic anhydride plant exhibited normal neuromuscular activity, with the exception of one worker who experienced a bilateral Achilles hyporeflexia (U.S. EPA 1986).
2. Animals — In a 2-year dietary study (see Section IV.C for details about the study), mice exhibited mineralization of the thalamus (both doses, males) (U.S. EPA 1994).

Cats exposed by inhalation for seven days to 3,700 mg/m<sup>3</sup> (611 ppm) phthalic anhydride exhibited loss of appetite and drowsiness (U.S. EPA 1988a).

## V. ENVIRONMENTAL EFFECTS

Studies with phthalic acid, the hydrolysis product of phthalic anhydride, suggest that the chemical is toxic to aquatic organisms only at moderate to high concentrations. Experimental studies suggest that phthalic anhydride is of low acute toxicity to terrestrial animals.

### A. Toxicity to Aquatic Organisms

The 96-hour LC<sub>50</sub> for phthalic anhydride in the fathead minnow (*Pimephales promelas*) is >50 mg/L (U.S. EPA 1986). Sea lampreys (*Petromyzon marinus*) were not adversely affected by exposure to 5 mg/L for 24 hours (U.S. EPA 1986). No-effect concentrations (NOECs) reported for the hydrolysis product of phthalic anhydride, phthalic acid, in various species are as follows: 640 mg/L for daphnids (48 hour); 56 mg/L for fathead minnows ("acute"); 40 mg/L for Japanese frogs (24 hours); and 5 mg/L for rainbow trout (*Salmo gairdneri*) and bluegill sunfish (*Lepomis macrochirus*) (24 hours) (U.S. EPA 1988a).

## B. Toxicity to Terrestrial Organisms

No information was found in the available literature for the toxicity of phthalic anhydride to terrestrial organisms. The LD<sub>50</sub> values of 800 to 1600 mg/kg for rats and 2210 mg/kg for mice (ACGIH 1991) suggest that the chemical would not be acutely toxic to terrestrial animals unless present in very high concentrations. Acute and chronic toxicity to terrestrial plants is expected to be low (U.S. EPA 1988a).

## C. Abiotic Effects

No information was found in the secondary sources searched on the abiotic effects of phthalic anhydride.

## VI. EPA/OTHER FEDERAL/OTHER GROUP ACTIVITY

The Clean Air Act Amendments of 1990 list phthalic anhydride as a hazardous air pollutant. Occupational exposure to phthalic anhydride is regulated by the Occupational Safety and Health Administration (OSHA). The OSHA permissible exposure limit (PEL) is 2 parts per million of air (ppm) as an 8-hour time-weighted average (TWA) (29 CFR 1910.000). In addition to OSHA, other federal agencies and groups may develop recommendations to assist in controlling workplace exposure. These agencies and groups (listed in Tables 6 and 7) should be contacted regarding workplace exposures and for additional information on phthalic anhydride.

**TABLE 6. EPA OFFICES AND CONTACT NUMBERS INFORMATION ON PHTHALIC ANHYDRIDE**

EPA Office	Statute	Contact Number
Pollution Prevention & Toxics	PPA <sup>a</sup> EPCRA (§313/TRI) <sup>b</sup> TSCA (§4 and §12b) <sup>c</sup>	(202) 260-1023 (800) 535-0202 (202) 554-1404
Air	Clean Air Act (§111 and §112B) <sup>d</sup>	(919) 541-0888
Solid Waste &	RCRA (Action levels: 7 mg/L, water; 200 g/kg, soil) <sup>e</sup>	(800) 535-0202
Emergency Response	CERCLA (RQ: 5000 lbs) <sup>f</sup>	(800) 535-0202

<sup>a</sup>PPA: Pollution Prevention Act

<sup>b</sup>EPCRA: Emergency Planning and Community Right to Know Act of 1986

<sup>c</sup>TSCA: Toxic Substances Control Act

<sup>d</sup>Listed as hazardous air pollutant under §111 and §112B of **Clean Air Act** [42 U.S.C. 7401 et seq.]

<sup>e</sup>RCRA: Resource Conservation and Recovery Act (40 CFR 264.94). **Action Level:** Health and environmental-based levels used by the EPA as indicators for the protection of human health and the environment and as triggers for a Corrective Measure Study.

<sup>f</sup>CERCLA: Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended; **RQ:** level of hazardous substance, which, if equaled or exceeded in a spill or release, necessitates the immediate reporting of that release to the National Response Center (40 CFR Part 302).

**TABLE 7. OTHER FEDERAL OFFICES/CONTACT NUMBERS FOR INFORMATION ON PHTHALIC ANHYDRIDE**

<b>Other Agency/Department/Group</b>	<b>Contact Number</b>
American Conference of Governmental Industrial Hygienists (TLV-TWA, 1 ppm) <sup>a</sup>	(513) 742-2020
Consumer Product Safety Commission	(301) 504-0994
Food & Drug Administration	(301) 443-3170
National Institute for Occupational Safety & Health (TWA, 1 ppm; IDLH, 1650 ppm) <sup>b</sup>	(800) 356-4674
Occupational Safety & Health Administration (TWA: 2 ppm) <sup>c</sup> (Check local phone book under Department of Labor)	

<sup>a</sup>**TLV-TWA:** Time-weighted-average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed without adverse effects (ACGIH 1993-1994).

<sup>b</sup>**TWA:** Time-weighted-average concentrations for up to a 10-hour workday during a 40-hour workweek.

**IDLH:** Immediate danger to life and health.

<sup>c</sup>**TWA:** Time-weighted-average that must not be exceeded during any 8-hour work shift of a 40-hour workweek. Standard promulgated pursuant to the Occupational Safety and Health Act, 29 CFR 1910 (OSHA 1993).

## VI. CITED REFERENCES

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