United States Environmental Protection Agency | Pollution Prevention and Toxics (7407) | February 1995 EPA 749-F-95-003a

OPPT Chemical Fact Sheets

1,1'-Biphenyl Fact Sheet: Support Document (CAS No. 92-52-4)

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 These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials. The literature search was done in February 1995. 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 and chemical properties of biphenyl are summarized in Table 1.

TABLE 1. CHEMICAL IDENTITY AND CHEMICAL/PHYSICAL PROPERTIES OF BIPHENYL

Characteristic/Property	Data	Reference
CAS No.	92-52-4	
Common Synonyms	diphenyl; bibenzene; phenylbenzene	Budavari et al. 1989
Molecular Formula	$C_{12}H_{10}$	Budavari et al. 1989
Chemical Structure		
Physical State	Colorless leaflets	Budavari et al. 1989
Molecular Weight	154.20	Budavari et al. 1989
Melting Point	69-71°C	Budavari et al. 1989
Boiling Point	254-255°C	Budavari et al. 1989
Water Solubility (mg/L)	insoluble 7.5 mg/L @ 25°C	Budavari et al. 1989 Vershueren 1983
Density	1.041 g/mL	Budavari et al. 1989
K_{OC}	1.40×10^3 (calculated)	CHEMFATE 1994
Log K_{OW}	4.09	CHEMFATE 1994
Vapor Pressure	9.64×10^{-3} mm Hg @ 25°C	CHEMFATE 1994
Reactivity	incompatible with oxidizers	Keith and Walters 1985
Flash Point	109°C closed cup	Keith and Walters 1985
Henry's Law Constant	3×10^{-4} atm•m^3/mol	CHEMFATE 1994
Fish Bioconcentration Factor	436 (static test, rainbow trout)	CHEMFATE 1994
Odor Threshold	0.06 mg/m^3 pleasant, peculiar odor	Vershueren 1983 Budavari et al. 1989
Conversion Factors	1 ppm = 6.5 mg/m^3 1 mg/m^3 = 0.154 ppm	ACGIH 1991

II. PRODUCTION, USE, AND TRENDS

A. Production

USITC (1994) identified three companies in the United States that made biphenyl in 1992; these companies, along with the other identified (TRI92 1994) domestic producer, are listed in Table 2. The production capacities of these plants are not available. U.S. production of biphenyl was 53 million pounds in 1990 (Table 3). This was a 23 percent increase from the production volume of 43 million pounds in 1989.

TABLE 2. U.S. PRODUCERS OF BIPHENYL AND THEIR LOCATIONS

Producer	Plant Location
Chevron Corporation, Chevron Chemical Company	Baytown, TX
Koch Chemical Company, a division of Koch Refining Company, Specialties Group	Corpus Christi, TX
Monsanto Chemical Company	Anniston, AL
Sybron Chemicals Company	Wellford, SC
TOTAL	

Sources: USITC 1994, EPA TRI Database.

B. Uses

The primary use of biphenyl is in the formulation of dye carriers for textile dyeing (HSDB 1994). Biphenyl is used as an intermediate for polychlorinated biphenyls (HSDB 1994) and as a paper impregnant for citrus fruit where it acts as a fungicide (Grayson 1985). In the past, a major use of biphenyl has been as a component of heat-transfer fluids (Grayson 1985). Table 3 provides a list of applicable SIC Codes for biphenyl use. No estimates of percentages of domestic end use patterns are available.

C. Trends

Production of polychlorinated biphenyls (PCB's) was once the most important use of biphenyl. Recently, however, the use of PCB's in liquid-filled transformers and as dielectric fluids has been restricted by the U.S. Environmental Protection Agency. They are being replaced by liquids containing halogen and silicone (The Freedonia Group, Inc. 1995). U.S. demand associated with other uses of biphenyl are expected to remain constant.

TABLE 3. END USE PATTERN OF BIPHENYL--1992 ESTIMATE

Derivative (Typical Standard Industrial Classification (SIC) Code)[1]	Percentage of U.S. Use
Dye Carrier (SIC 2865)	N/A
Intermediate for Manufacture of Polychlorinated Biphenyls (SIC 2865)	N/A
Fungicide (SIC 2879)	N/A
Heat Transfer Agent (SIC 2899)	N/A

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[1] 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.

N/A Not available

III. ENVIRONMENTAL FATE

A. Environmental Release

Biphenyl is released to the atmosphere as a fume during its use as a heat transfer fluid and, to a lesser extent, by volatilization from soil and water (U.S. EPA 1984). Atmospheric levels of biphenyl measured 20-1500 ng/m^3 in Kingston, RI, an industry-free urban area (CHEMFATE 1994).

Biphenyl enters the aquatic environment in wastewater effluents from textile mills that use it as a dye carrier; from industrial processes; and from leaking heat exchangers (U.S. EPA 1984).

Because of its use as a fungicide, residues of biphenyl have been detected in tangerines (0.01-0.085 mg/kg, whole fruit), grapefruits (0-150 mg/kg, peel or whole fruit), oranges (0-0.012 mg/kg, edible portion), and lemons (0.02-0.12 mg/kg, edible portion) (U.S. EPA 1984). One investigator reported airborne concentrations of 3.2-250 pg biphenyl/m^3 from diesel exhaust (U.S. EPA 1984). In monitoring studies, biphenyl was detected at the following sites: in the Tennessee River (2 micrograms/L), in a roadside ditch in Kentucky (5000 mg/kg), in Lake Michigan at the mouth of the Galien River (4 micrograms/L), in effluent from industrial and water treatment facilities (0-130 micrograms/L), in Great Lakes Municipal drinking water (0.3-31.9 nanograms/L), in Athens, GA, drinking water (1-5 nanograms/L), and in groundwater at the site of an inactive underground coal gasification process (25-43 micrograms/L) (U.S. EPA 1984).

In 1992, environmental releases of biphenyl, as reported to the Toxic Chemical release inventory by

certain types of U.S. industries, totaled about 885 thousand pounds, including approximately 820 thousand pounds to the atmosphere, 50 thousand pounds to underground injection sites, 10 thousand pounds to surface water, and 5 thousand pounds to land (TRI92 1994).

B. Transport

No information was found in the secondary sources searched regarding the transport of biphenyl in air. Volatilization and sorption are important in the transport of biphenyl in water. The Henry's Law constant for biphenyl (3×10^{-4} atm·m^3/mol) suggests that the chemical could undergo volatilization. The volatilization half-life of 4.3 hours was estimated for biphenyl in a stream 1 meter deep, flowing 1 meter/second, with an air current of 3 meters/second (U.S. EPA 1984). The log octanol/water partition coefficient of 4.09 for biphenyl suggests that the chemical has potential for sorption to particulate matter (U.S. EPA 1984). One study demonstrated that 50% of the biphenyl applied to activated sludge in water remained as nonextractable residue (U.S. EPA 1984).

The high octanol/water partition coefficient and the low water solubility of biphenyl would probably preclude significant leaching of the chemical through soil (U.S. EPA 1984). However, soil microorganisms may metabolize biphenyl to the more polar hydroxy biphenyls and dihydroxy biphenyls which may leach into groundwater (U.S. EPA 1984). The volatilization of biphenyl from soil is not likely to be significant. Less than 2% of the applied radioactivity from (^{14}C)-biphenyl, incorporated into wet sand, loam, and humus, evaporated in 2 hours (U.S. EPA 1984).

C. Transformation/Persistence

1. Air — In the air, biphenyl reacts with hydroxyl radicals and undergoes photolysis (U.S. EPA 1984). The calculated half-life for the reaction of biphenyl with OH^- is 2.2 days at 25°C, assuming an OH concentration of 5×10^5 molecule/cm^3 (CHEMFATE 1994).

   In a photolysis experiment, biphenyl was impregnated into paper and exposed to UV irradiation (>300 nanometers) in the presence of NO_x. The chemical underwent "considerable" photodegradation, yielding 2- and 4-nitrobiphenyl as reaction products (U.S. EPA 1984). The half-life of biphenyl was ~2 hours. In another study, 80 ppb of ^{14}C-biphenyl was irradiated on silica gel for 17 hours at a wavelength of >290 nm (U.S. EPA 1984). Photodecomposition products included 9.5% $^{14}CO_2$ and <0.1% organic fragments.

   Biphenyl also reacts slowly with ozone. The calculated half-life for the reaction is 57.3 days at 25°C, assuming an ozone concentration of 7×10^{11} molecule/cm^3 (CHEMFATE 1994).

2. Soil — The main removal process for biphenyl in soil appears to be biodegradation. The following organisms have been shown to degrade biphenyl: *Saccharomyces cerevisiae* (with the production of benzoic acid), *Streptomyces sp.*, *Achromobacter*, *Pseudomonas putida*, *Oscillatoria sp.*, gram negative bacteria, *Acaligenes sp.* 559, *Acaligenes* Y42 and *Acinetobacter* P6 (U.S. EPA 1984). Bacteria generally oxidize biphenyl via cytochrome P-450 to 2,3-dihydroxybiphenyl (U.S. EPA 1984). Fungi metabolize biphenyl to 4-hydroxy- or 2-hydroxybiphenyl and 4,4'-dihydroxybiphenyl (U.S. EPA 1984). In one study, 9.1% of the biphenyl was degraded by activated sludge in 2 days (U.S. EPA 1984).

3. Water — The main environmental fate processes for biphenyl in water are photolysis and microbial degradation (U.S. EPA 1984). Biphenyl in solution, irradiated with a germicidal UV lamp (UV spectra of ~250 nm), underwent 50% degradation in ~40 hours (U.S. EPA 1984).

   The biodegradation of biphenyl by aquatic microorganisms proceeds via aromatic hydroxylation to 2-, 3-, and 4-hydroxybiphenyl, with further hydroxylation to 2,3-dihydroxybiphenyl (U.S. EPA 1984). The degradation rates for biphenyl under various conditions were: 74% in 14 days (activated sludge), 100% in 7 days (anaerobic digester), 100% in 8 hours (aerated lagoon), 100% in 96 hours (retention pond), 79% in 5 days (domestic wastewater), 87% in 24 hours and 100% in 5 days (industrial wastewater) (CHEMFATE 1994).

4. Biota — The bioconcentration factors of biphenyl (436 for the rainbow trout, 540 for algae

[*Chlorella fusca*], and 282 for the orfe [*Leuciscus idus melanotus*] [U.S. EPA 1984]) suggest a moderate potential for the accumulation of the chemical in aquatic organisms. The octanol/water partition coefficient of 4.1 (CHEMFATE 1994) suggests that the chemical has an affinity for lipids.

IV. HEALTH EFFECTS

A. Pharmacokinetics

1. Absorption — The detection of urinary metabolites in animals following oral administration of biphenyl indicate the chemical is absorbed by this route (see section IV.A.4) (U.S. EPA 1984). Systemic effects in rabbits, rats, and mice, observed following repeated inhalation exposure to biphenyl-impregnated celite dust, indicate that the chemical is also absorbed by this route (U.S. EPA 1984). Similarly, repeated application of biphenyl to the depilated backs of rabbits resulted in systemic effects, indicating dermal absorption (U.S. EPA 1984).

2. Distribution — Little information was found in the secondary sources searched regarding the distribution of biphenyl. Following absorption, biphenyl is transported to the liver where it undergoes hydroxylation and conjugation, and becomes more polar (U.S. EPA 1984). In rats given ^{14}C-biphenyl orally, about 85, 7, 0.1, and 0.6% of the administered radioactivity was detected in the urine, feces, expired air (as CO_2), and tissues, respectively, within 96 hours of dosing (U.S. EPA 1984). Bile collected for 96 hours after the oral administration of biphenyl to rats contained 12 metabolites of biphenyl, accounting for 5.2% of the administered dose (U.S. EPA 1984). The octanol/water partition coefficient of 4.1 (CHEMFATE 1994) suggests that the chemical has an affinity for lipids, but no information was found to demonstrate this.

3. Metabolism — In the liver, biphenyl undergoes hydroxylation and conjugation (U.S. EPA 1984). More than 10 mono-, di-, and tri-hydroxybiphenyl metabolites have been identified in the urine of animals (U.S. EPA 1984). These metabolites may occur as conjugates of mercapturic acid and glucuronide. A major metabolite in the rat, mouse, guinea pig, rabbit and pig is 4-hydroxybiphenyl; other major metabolites include 4,4-dihydroxybiphenyl in the pig and rat and 2-hydroxybiphenyl in the mouse (U.S. EPA 1984). There is evidence to suggest that the metabolism of biphenyl is mediated by a cytochrome P-450 system and that an arene oxide intermediate capable of binding to biomacromolecules may be formed (U.S. EPA 1984).

4. Excretion — Following the hydroxylation and conjugation of biphenyl in the liver, the chemical is excreted mainly in the urine as mercapturic acid or glucuronide conjugates (U.S. EPA 1984). Small amounts have also been detected in the feces, in expired air as CO_2, and in the tissues (see section IV.A.2) (U.S. EPA 1984). Rabbits, guinea pigs, and pigs given biphenyl by gavage excreted at least 20% of the administered dose in the urine within 24 hours; rats administered ^{14}C-biphenyl orally excreted 75-80% of the dose in 24 hours (U.S. EPA 1984).

B. Acute Effects

Workers exposed acutely to biphenyl in air have experienced nausea, vomiting, bronchitis, and irritation of the eyes and mucous membranes.

1. Humans — Workers exposed to biphenyl fumes experienced irritation to the eyes and mucous membranes at concentrations of 3-4 ppm or 19-25 mg/m^3 (Sandmeyer 1981). The concentration of 3-4 ppm is roughly equivalent to 2-3 mg/kg over an 8-hour exposure period[1]. Others, exposed (concentrations not available) during paper impregnation, had transient nausea, vomiting, and bronchitis (ACGIH 1991). At concentrations below 1 mg biphenyl/m^3 there were no differences between exposed and unexposed workers in a

[1]For dose comparison purposes this has been calculated by multiplying 19-25 mg/m^3 by 0.143 (the occupational standard breathing rate, 10 m^3, divided by the assumed adult body weight, 70 kg, and assuming 100% absorption) to obtain the dose in mg/kg/day (U.S. EPA 1988).

comparison of blood pressure, pulmonary function tests, serum creatinine values, urinary protein levels, and standard blood cell counts (ACGIH 1991).

2. Animals — Oral LD_{50} values for biphenyl are 3280 mg/kg for the rat and 2410 mg/kg for the rabbit (U.S. EPA 1984)).

C. Subchronic/Chronic Effects

The EPA has derived a chronic oral reference dose (RfD[2]) of 0.05 mg/kg/day for biphenyl, based on kidney damage in rats. Workplace exposure to biphenyl has caused the death of one worker and adverse effects on the liver and nervous system (see section IV.G) of others. In animals, oral doses of large amounts of biphenyl produced adverse effects on the kidneys, the liver, the blood and spleen, and growth and longevity. Subchronic inhalation exposure of animals to biphenyl-impregnated dust produced lesions of the respiratory tract.

1. Humans — A Finnish paper mill worker died in 1969, following 11 years of "heavy exposure" to biphenyl (ACGIH 1991). The cause of death was reported as "yellow atrophy" of the liver. During the 11 years, average concentrations of biphenyl in the workplace air ranged from 0.6 to 128 mg/m^3 (0.09 to 19.7 ppm; 0.086 to 18.3 mg/kg/day). Eight other workers experienced signs of toxicity that were characterized by damage to the liver and nervous system (see section IV.G).

2. Animals — The following study was the basis for EPA's derivation of the chronic oral RfD for biphenyl, 0.05 mg/kg/day (U.S. EPA 1994). Albino rats (15/sex/group) received diets containing biphenyl concentrations of 0.0, 0.001, 0.005, 0.01, 0.05, 0.1, 0.5 or 1% for 700 days (U.S. EPA 1984; 1994). Growth retardation, reduced hemoglobin levels, decreased food intake, kidney damage (that included irregular scarring, lymphocytic infiltration, tubular atrophy and patchy tubular dilation), and decreased longevity were observed in rats given $\geq$0.5% biphenyl ($\geq$250 mg/kg/day; the lowest-observed-adverse-effect level [LOAEL]). Sporadic effects were noted at lower doses (2 male rats receiving 0.1 and 0.05% biphenyl had disintegrated blood cells in the renal pelvis and two others had small basophilic concretions in the renal medullas); 0.1% biphenyl (50 mg/kg/day) was selected as the no-observed-adverse-effect level (NOAEL) for the study (U.S. EPA 1994). In the calculation of the RfD the EPA applied a modifying factor of 10 to account for intraspecies variability demonstrated by uncertainty in the threshold of the study, in addition to the uncertainty factor of 100 (U.S. EPA 1984; 1994). Another study from the same laboratory demonstrated that the renal damage occurring in rats fed >0.5% could be reversed by placing animals on a control diet (U.S. EPA 1984).

   Supporting the results of the critical study for the RfD, a 0.1% NOAEL was also found in a subchronic feeding study and in a 3-generation reproduction study; both studies were conducted in rats and were unpublished (U.S. EPA 1994).
   Another study investigated the effect of dietary biphenyl on the induction of polycystic renal lesions in the rat. Rats fed biphenyl in commercial rat chow for 21 days exhibited increased kidney weight, urine volume, and urine specific gravity at $\geq$500 mg/kg body weight/day and polycystic renal changes at 1000 mg/kg/day. The no-effect level for renal effects was 300 mg/kg/day. When a semisynthetic diet was substituted for the commercial chow, increased kidney weight occurred at 50 mg/kg/day (the lowest dose tested) and polycystic changes and other renal effects occurred at $\geq$150 mg/kg/day (U.S. EPA 1984).

   In an inhalation study, three albino rabbits and 10 Sprague-Dawley rats inhaled biphenyl-impregnated celite dust (300 mg biphenyl/m^3), 7 hours/day, 5 days/week over a period of 90 days (U.S. EPA 1985a). Similarly, 3 rabbits and 6 rats were exposed to 40 mg biphenyl/m^3 over 64 days. Control information was not available. All animals had severe bronchopulmonary lesions characterized by emphysema, lobular pneumonia, bronchitis and multiple abscesses of the lungs. Minor liver and kidney damage also occurred (U.S. EPA

[2]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.

1985a). The concentration of 40 mg/m^3 is roughly equivalent to 7.43 mg/kg over the 7-hour exposure period[3]. In the same study, mice exposed to 5 mg/m^3 for 64 days exhibited respiratory difficulty, whereas rats exposed similarly did not (ACGIH 1991). In the absence of control data, the pulmonary effects could not be attributed solely to biphenyl (U.S. EPA 1985a).

Rabbits exposed by dermal application to 0.5 g biphenyl/kg, 2 hours/day for 5 days exhibited growth depression; slight cardiac, hepatic, and renal changes; and follicular atrophy, necrosis, and leukocytic infiltration of the spleen; and there were "some" deaths (Sandmeyer 1981). Repeated application of 25% biphenyl in olive oil to the skin of rabbits did not produce irritation, but resulted in the death of 1 animal after 8 applications, and produced weight loss in 3 others after 20 applications (ACGIH 1991).

D. Carcinogenicity

Based on no information on humans and inadequate studies in rats and mice, the EPA has classified biphenyl as D, not classifiable as to human carcinogenicity. There is some evidence that biphenyl may be a tumor promoter.

1. Humans — No information was found in the secondary sources searched for the carcinogenicity of biphenyl in humans.

2. Animals — Treatment-related tumors were not found under the following conditions: (1) in B6AKF$_1$ and B6C3F$_1$ mice (18/sex/strain) treated with 215 mg/kg biphenyl/day by gavage from day 7 to day 28 of age, then with 517 ppm dietary biphenyl for the subsequent 18 months (U.S. EPA 1994); (2) in albino rats given ≤500 mg/kg biphenyl/day in the diet for 700 days (U.S. EPA 1994); and (3) in groups of 12 male and 12 female Sprague-Dawley rats given ≤1% dietary biphenyl for 2 years. Several deficiencies limit the validity of the last study (U.S. EPA 1994).

   Groups of 25 male F344 rats were given 0.05% N-butyl-N-(4-hydroxybutyl)nitrosamine (BBN) in drinking water for 4 weeks followed by a basal diet or a diet containing 0.5% biphenyl for 32 weeks (U.S. EPA 1994). A group of 5 rats received only 0.5% biphenyl. In the 18 surviving rats treated with BBN and biphenyl, the incidences of hyperplasia, papillomas and carcinomas in the urinary bladder were 94, 83 and 61%, respectively. These were statistically significant in comparison to incidences of 25, 12, and 0%, respectively, in animals treated with BBN alone (there were no signs of hyperplasia, papillomas and carcinomas in the rats fed biphenyl alone). The results of this study suggest that 1,1-biphenyl is a tumor promoter (U.S. EPA 1994).

E. Genotoxicity

Biphenyl induced forward mutations in mouse lymphoma cells and sister chromatid exchanges (SCE) in Chinese hamster cells; however, a dose-response relationship was not observed in the SCE assay (U.S. EPA 1994). Biphenyl did not induce reverse mutations in *Salmonella typhimurium* and *Escherichia coli*, DNA repair in *E. coli*, chromosomal aberrations in Chinese hamster cells, or unscheduled DNA synthesis in rat hepatocytes (the presence or absence of metabolic activation was not specified) (U.S. EPA 1994).

F. Developmental/Reproductive Toxicity

Limited information indicates that biphenyl is not teratogenic and does not produce significant fetal toxicity, even at maternally toxic doses.

1. Humans — No information was found in the secondary sources searched regarding the

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[3]For dose comparison purposes, this has been calculated by multiplying 40 mg/m^3 by 0.186 (the 7-hour breathing rate, 0.065 m^3 [standard 24-hour breathing rate, 0.223 m^3] divided by the assumed adult rat body weight, 0.350 kg, and assuming 100% absorption) to obtain the dose in mg/kg/day (U.S. EPA 1985b).

developmental/reproductive toxicity of biphenyl in humans.

2. Animals — Pregnant Wistar rats received biphenyl in oral doses of 0, 125, 250, 500 or 1000 mg/kg/day on days 6-15 of gestation (U.S. EPA 1994). No fetal or maternal toxicity occurred in animals receiving doses $\leq$500 mg/kg. There was some (but not statistically significant) evidence of toxicity in both dams (death) and fetuses (reduced fetal weights, decreased number of live fetuses, and increased resorptions) at 1000 mg/kg (U.S. EPA 1994). In one study, no significant effects occurred in rats given 0, 0.1, or 0.5% dietary biphenyl from 60 days before mating through weaning of their offspring (U.S. EPA 1994); however, in a 3-generation study on rats, 1% dietary biphenyl produced "unspecified adverse effects" (U.S. EPA 1994).

G. Neurotoxicity

Workers exposed chronically to concentrations of biphenyl ranging from low to high developed persistent damage to the central and peripheral nervous systems.

1. Humans — In a Finnish paper mill, levels of biphenyl in the workplace air ranged from 0.6 to 128 mg/m^3 from 1959 to 1970 (ACGIH 1991). Central and peripheral nerve damage was observed in workers who were exposed in this plant. Neurological findings in 24 workers were as follows: (1) electroencephalographic abnormalities (10 workers) of a generally diffuse nature, persisting for 1 to 2 years; and (2) electromyographic abnormalities (9 workers), including fibrillation of muscles (7 of the 9 workers) and long rhythmic series of fasciculations resembling that in infantile spinal muscular atrophy (1 worker). These abnormalities also persisted upon reexamination (ACGIH 1991).

2. Animals — No information was found in the secondary sources searched on the neurotoxicity of biphenyl in animals.

V. ENVIRONMENTAL EFFECTS

In laboratory studies, biphenyl is highly toxic to aquatic organisms; an acute toxicity value of <1 mg/L has been reported. EPA has requested chronic aquatic toxicity testing for biphenyl under Section 4 of the Toxic Substance Control Act (TSCA). Results of chronic tests in fish and invertebrates suggest low chronic toxicity for biphenyl.

A. Toxicity to Aquatic Organisms

Acute toxicity values as low as 0.36 mg/L (for daphnids) and 1.3 mg/L (for rainbow trout) have been reported (Fenner-Crisp 1988). Other ninety-six-hour LC_{50} values for biphenyl in fish are as follows: 1.5 mg/L for *Oncorhynchus mykiss* (rainbow trout); 4.7 mg/L for *Lepomis macrochirus* (bluegill); 4.6 mg/L for *Cyprinodon variegatus* (sheepshead minnow); 6 mg/L for *Pimephales promelas* (fathead minnow) (this concentration is near the water solubility of biphenyl) (U.S. EPA 1984). Other estimated 48-hour LC_{50} values for biphenyl in *Daphnia magna* are 4.7 and 2.1 mg/L (U.S. EPA 1984).

The growth of *Chlorella autotrophica* (green algae) was slightly inhibited (4 mm zone of inhibition at 1.0 mg biphenyl/plate) and totally inhibited (36 mm zone of inhibition) at 10 mg/plate (U.S. EPA 1984).

Results from aquatic chronic toxicity testing in rainbow trout and daphids suggest low chronic toxicity for biphenyl (Fenner-Crisp 1988). Chronic toxicity values were 230 micrograms/L and 275 micrograms/L for fish and daphnids, respectively. Chronic values greater that 100 micrograms/L are indicative of low toxicity.

B. Toxicity to Terrestrial Organisms

No information was found in the secondary sources searched regarding the toxicity of biphenyl to terrestrial organisms; however, studies with experimental animals have demonstrated low acute toxicity and low to moderate chronic toxicity. Biphenyl is not expected to be toxic to terrestrial organisms at levels normally found in the U.S. U.S. environment.

C. Abiotic Effects

No information was found regarding the abiotic effects of biphenyl.

VI. EPA/OTHER FEDERAL/OTHER GROUP ACTIVITY

The Clean Air Act Amendments of 1990 list biphenyl as a hazardous air pollutant. Occupational exposure to biphenyl is regulated by the Occupational Safety and Health Administration (OSHA). The OSHA permissible exposure limit (PEL) is 0.2 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 other groups (listed in Tables 4 and 5) should be contacted regarding workplace exposures and for additional information on biphenyl.

TABLE 4. EPA OFFICES AND CONTACT NUMBERS FOR INFORMATION ON BIPHENYL

EPA Office	Statute	Contact Number
Pollution Prevention & Toxics	PPA[a]	(202) 260-1023
	EPCRA (§313/TRI)[b]	(800) 535-0202
	TSCA (§4, §8A, §8D, §12B)[c]	(202) 554-1404
Pesticides	FIFRA[d]	(800) 858-7378
Air	Clean Air Act (§111, §112B)[e]	(919) 541-0888
Solid Waste & Emergency Response	CERCLA[f]	(800) 535-0202

[a]PPA = Pollution Prevention Act

[b]EPCRA = Emergency Planning and Community Right to Know Act of 1986

[c]TSCA = Toxic Substances Control Act

[d]FIFRA = Federal Insecticide and Rodenticide Act. Biphenyl is a list "B" pesticide (as of 1989), according to IRIS (U.S. EPA 1994) (54 FR 22706 [5/25/89]).

[e]Listed as hazardous air pollutant under §111 and §112 of Clean Air Act [42 U.S.C. 7401 et seq.]

[f]CERCLA = Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended.

TABLE 5. OTHER FEDERAL OFFICES/CONTACT NUMBERS FOR INFORMATION ON BIPHENYL

Other Agency/Department/Group	Contact Number
Agency of Toxic Substances & Disease Registry[a]	(404) 639-6000
American Conference of Governmental Industrial Hygienists (TLV-TWA, 0.2 ppm)[b]	(513) 742-2020
Consumer Product Safety Commission	(301) 504-0994
Food & Drug Administration	(301) 443-3170
National Institute for Occupational Safety & Health (TWA, 0.2 ppm; IDLH, 300 ppm)[c]	(800) 356-4674
Occupational Safety & Health Administration (TWA, 0.2 ppm)[d] (Check local phone book for phone number under Department of Labor)	

[a]Newly listed hazardous substance subject to Toxicological Profile (55 FR 46131, Nov. 1, 1990)

[b]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 1994-1995).

[c]TWA: Time-Weighted-Average concentration for up to a 10-hour workday during a 40-hour workweek. IDLH: immediate danger to life and health.

[d]TWA: Time-Weighted-Average concentrations that must not be exceeded during any 8-hour work shift of a 40-hour workweek. OSHA standards promulgated pursuant to the Occupational Safety and Health Act, 29 CFR 1910 (OSHA 1993).

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