United States Environmental Protection Agency | Pollution Prevention and Toxics (7407) | December 1994 EPA 749-F-95-002a

OPPT Chemical Fact Sheets

Aniline Fact Sheet: Support Document (CAS No. 62-53-3)

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 These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials.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 of 1995. No attempt has been made to verify information from these databases or secondary sources.

I. CHEMICAL IDENTITY AND PHYSICAL/CHEMICAL PROPERTIES

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

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

Characteristic/Property	Data	Reference
CAS No.	62-53-3	
Common Synonyms	aminobenzene; aminophen; phenylamine; benzeneamine	U.S. EPA 1994
Molecular Formula	C_6H_7N	
Chemical Structure		
Physical State	liquid	U.S. EPA 1985
Molecular Weight	93.12	U.S. EPA 1985
Melting Point	6.15 °C	U.S. EPA 1985
Boiling Point	184.4 °C @ 1 atm	U.S. EPA 1985
Water Solubility	34 g/L @ 20 °C; 35 g/L @ 25 °C	U.S. EPA 1985
Specific Gravity	1.02173 @ 20/4 °C	U.S. EPA 1985
Vapor Density (air = 1)	3.22	Verschueren 1983
K_{OC}	3870 @ pH 6.5 (measured)	U.S. EPA 1985
Log K_{OW}	0.90	U.S. EPA 1985
Vapor Pressure	0.3 mm Hg @ 20 °C; 0.67 mm Hg @ 25 °C	U.S. EPA 1985
Reactivity	flammable	U.S. EPA 1985; Budavari 1989
Flash Point	76 °C (closed cup)	U.S. EPA 1985
Henry's Law Constant	1.9×10^{-6} atm-m^3/mole @ 25 °C	U.S. EPA 1985
Fish Bioconcentration Factor	3.0 (calculated)	U.S. EPA 1985
Odor Threshold	perception, 0.34 mg/m^3	Verschueren 1983
Conversion Factors (in air)	1 ppm = 3.87 mg/m^3; 1 mg/m^3 = 0.259 ppm	U.S. EPA 1985

II. PRODUCTION, USE, AND TRENDS

A. PRODUCTION

In 1992, the estimated total United States production capacity of aniline was 1,380 million pounds. The 1994 capacity was expected to remain the same (Mannsville 1992). Table 2 shows the plant locations and plant capacities of producers of aniline in 1992. In 1992, the production volume of aniline was estimated to be 1,005 million pounds (457 million kilograms) (USITC 1994). The U.S. imported four million pounds of aniline in 1991, and was expected to import the same amount in 1992. In 1991, the U.S. exported 55 million pounds of aniline, but it is expected to export only 40 million pounds in 1992. Table 2 lists six producers of aniline in 1992 (Mannsville 1993). USITC (1994) lists an additional producer as Malinckrodt Specialty Chemicals Company of Raleigh, North Carolina.

TABLE 2. U.S. PRODUCERS OF ANILINE AND THEIR CAPACITIES IN 1992

Producer[1]	Plant Location	Plant Capacity (Millions of Pounds)
Aristech	Haverhill, OH	200
BASF Corporation[2]	Geismar, LA	170
DuPont	Beaumont, TX	270
First Chemical Corporation	Pascaqoula, MS	300
Miles, Inc. (formerly Mobay; now Bayer)	New Martinsville, WV	40
Rubicon (ICI/Uniroyal affiliate)[3]	Geismar, LA	400
TOTAL		1,380

Source: Mannsville 1992.

S))))))))))))))))))))Q

[1]USITC (1994) lists Malinckrodt Specialty Chemicals Company of Raleigh, NC as an additional producer of aniline.

[2]USITC's list (1994) was drafted before the opening of BASF Corporation's new 170 million pounds per year plant at Geismar, Louisiana in 1992.

[3]Rubicon is an affiliate of ICI and Uniroyal (Mannsville 1992).

B. USES

Isocyanate production accounts for approximately two-thirds of total U.S. demand for aniline (see Table 3). Isocyanates derived from anilines are used to produce urethanes (Mannsville 1992). The production of methyl diphenyl diisocyanate, an intermediate for the production of urethanes, is the largest end use for aniline (Windholz 1983).

Other principal applications of aniline include production of rubber accelerators and antioxidants to vulcanize rubber; the manufacture of intermediates for herbicides and other pesticides, especially fungicides; and the manufacture of dyes and pigments, especially azo dyes (Mannsville 1992; Sax and Lewis 1987; Windholz 1983) Aniline is also used to produce medicinals and pharmaceuticals, resins,

varnishes, perfumes, shoe blacks, photographic chemicals (hydroquinone), explosives, petroleum refining chemicals, diphenylamine, and phenolics (Mannsville 1992; Sax and Lewis 1987; Windholz 1983). Table 3 shows the end use pattern of aniline in 1992.

TABLE 3. END USE PATTERN OF ANILINE IN THE UNITED STATES (1992)

Derivative (Typical Standard Industrial Classification (SIC) Code)[4]	U.S. Consumption (Millions of Pounds)	Percentage of U.S. Use
Isocyanates (SIC 2865)	659	68
Rubber Chemicals (SIC 2869)	155	16
Agricultural-Pesticides (SIC 2879)	68	7
Dyes and Pigments (SIC 2865)	39	4
Miscellaneous (Various SICs)	48	5
TOTAL	964	100

Source: Mannsville 1992.

[4] 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

United States demand for aniline is expected to increase three to four percent per year (Mannsville 1992). Increases in demand for aniline is dependent on increased demand for methyl diphenyl diisocyanate (MDI). MDI demand is dependent on general economic conditions due to its heavy use in the production of construction and transportation materials. MDI demand is expected to increase at four to five percent annually (Mannsville 1992). U.S. demand for rubber, agricultural chemicals, and dyes has moderated over the last several years.

III. ENVIRONMENTAL FATE

A. Environmental Release

Aniline is a colorless, oily liquid at room temperature that darkens on exposure to air and light. It has a characteristic pungent odor and burning taste (U.S. EPA 1985; Budavari 1989). It occurs naturally in some foods (e.g., in corn, grains, beans, and tea). It is released into the environment primarily from industrial uses. The largest sources of aniline release are from its primary uses as a chemical intermediate in the production of polymers, pesticides, pharmaceuticals and dyes (U.S. EPA 1985). The chemical has been detected, but not quantified, in ground water in a shallow aquifer known to be contaminated by coal-tar wastes. It has been measured at a maximum of 36 parts per billion (ppb) in an aquifer near an underground coal gasification site in Wyoming. Aniline has been found in industrial wastewater and leachates from disposal sites. One of two soil samples collected near the Buffalo River in New York contained 5 parts per million (ppm) aniline. One air sample near Raleigh, NC was found to contain 90 ppb aniline, and it has been detected in air about 4000 meters from a chemical factory (Howard 1989).

In 1992, releases of aniline to environmental media, as reported to the Toxic Chemical Release Inventory by certain types of U.S. industries, totaled about 1.6 million pounds. Of this amount, 400 thousand pounds (25%) were released to the atmosphere; 16 thousand pounds were released to surface water; 1.2 million pounds (74%) were released in underground injection sites; and 11 hundred pounds were released to land (TRI92 1994).

B. Transport

Aniline in solution adsorbs strongly to colloidal organic matter, which effectively increases its solubility and movement into ground water. It is also moderately adsorbed to organic material in the soil; adsorption is dependent upon soil pH (pKa of 4.596) (Howard 1989). It will slowly volatilize from soil and surface water (vapor pressure 0.67 mm Hg @ 25°C) and is subject to biodegradation. Although rapidly degraded in the atmosphere, aniline can be deposited in the soil by wet and dry deposition, and by adsorption on aerosol particles (U.S. EPA 1985).

C. Transformation/Persistence

1. Air — Aniline reacts with any free radicals produced by sunlight in the atmosphere. This radical scavenging reactivity has been shown to inhibit the production of photochemical smog by sunlight. Aniline apparently undergoes direct photolysis and has considerable absorption of wavelengths above 290 nanometers. Photoproducts formed from aniline in the atmosphere include N-methylaniline, N,N-dimethylaniline, isomeric hydroxyanilines, and phenols (U.S. EPA 1985). The half-life of atmospheric aniline due to photodegradation has been estimated at 3.3 hours (Howard 1989).

2. Soil — A number of microorganisms in soil can use aniline as a sole carbon and nitrogen source. Degradation of 44.2% of the incubated aniline to CO_2 in 10 days and 12% in 20 days, respectively, by different isolated soil microorganisms has been demonstrated in the laboratory (U.S. EPA 1985). Aniline bound to humic materials in the soil is subject to oxidation (U.S. EPA 1985; Howard 1989).

   Products apparently formed from oxidation include azobenzene, azoxybenzene, phenazine, formanilide, and acetanilide (U.S. EPA 1985). Photodegradation of aniline on the soil surface is also thought to be an environmentally important removal process (U.S. EPA 1985). The combination of these processes eventually results in the degradation of aniline to CO_2. The half-life for the mineralization of aniline to CO_2 has been estimated at 4 days, utilizing a model soil ecosystem. Information from studies

done obtained under environmental conditions indicate that the half-life of aniline in the soil is less than one week (U.S. EPA 1985).

3. Water — Aniline in water is subject to biodegradation, photodegradation, and adsorption to sediment and humic materials. Low pH will increase the removal of aniline by adsorption; however, the adsorption to colloidal particles can extend the persistence of aniline in the aquatic environment (Howard 1989; U.S. EPA 1985). Although subject to oxidation when adsorbed to humic materials, aniline is resistant to hydrolysis. A half-life for aniline of 2.3 days has been reported in an industrial river (Howard 1989). The presence of humic acids and various species of algae in the water can increase the photodegradation rates of aniline up to 50 fold (Howard 1989).

4. Biota — The bioconcentration factor in two species of fish has been estimated at 0.78 and less than 1 (Howard 1989). A bioconcentration factor of 3 has also been calculated for fish (U.S. EPA 1985). Aniline is not expected to accumulate significantly in aquatic organisms; however, it is absorbed and metabolized by fish (Howard 1989).

IV. HUMAN HEALTH EFFECTS

A. Pharmacokinetics

1. Absorption — Studies in humans and animals have demonstrated that aniline is absorbed through the gastrointestinal tract and the lungs (see Sections IV.B. and C.); however, the only quantitative information found for the rates of absorption in humans were based on dermal exposures. Volunteers were exposed to solutions of 1-2% aniline for 30 or 60 minutes. The amount of aniline absorbed was quantitated by measuring the amount of *p*-aminophenol excreted in the urine in 24 hours. The absorption rates were 0.15-1.38 mg/cm^2/hour. The absorption rates increased with increasing concentrations and decreased with longer exposure times (U.S EPA 1985). Experiments with rats demonstrated that peak plasma levels of aniline were reached in 0.5, 1.0, and 2.0 hours following gavage administration of 10, 30, and 100 mg/kg ^{14}[C]-aniline hydrochloride, respectively (U.S. EPA 1985)

2. Distribution — The distribution of radioactivity after oral administration of ^{14}[C]-aniline to rats was highest in the kidney, liver, plasma, lung, heart, spleen, and brain. However, some radioactivity was reported in all tissues examined. Different doses of ^{14}[C]-aniline (10, 30, and 100 mg/kg) did not alter the distribution pattern of radioactivity (U.S. EPA 1985).

3. Metabolism — The primary metabolic route for aniline has been shown in several species to involve ring hydroxylation forming 4-aminophenol and 2-aminophenol, and N-hydroxylation forming phenylhydroxylamine. Other metabolites identified in a rat liver perfusate include acetanilide, 4-acetamidophenol, and nitrosobenzene (U.S. EPA 1985).

4. Excretion — Aniline metabolites are excreted in the urine. Conjugates of 4-aminophenol and glucuronic or sulfuric acid have been identified in the urine of all species tested. Phenylhydroxylamine may also be conjugated with glucuronate or sulfate, or it may form a mercapturic acid conjugate after reacting with cysteine. Forty-eight hours following oral administration of 10, 30 or 100 mg/kg body weight ^{14}[C]-aniline to rats, 96, 91, and 77%, respectively, of the radioactivity was recovered in the urine (U.S. EPA 1985).

B. Acute Toxicity

The primary toxic effect resulting from acute exposure to aniline by inhalation, oral or dermal routes is methemoglobinemia and accompanying anoxia, erythrocyte damage, and spleen effects. Adverse effects on the liver and spleen have also been reported.

1. Humans — Volunteers given single oral doses of 5, 15, 25, 35, 45, 55, and 65 mg of aniline developed increased methemoglobin formation at doses 25 mg or higher. The authors concluded that humans were much more sensitive to aniline exposure than rats, judging from methemoglobin formation (U.S. EPA 1994; U.S. EPA 1985). Liver cirrhosis and atrophy were reported in at least one fatal case of aniline exposure (ACGIH 1991). Inhalation exposure to 7-53 ppm aniline for several hours resulted in slight symptoms; exposure to 100-160 ppm for 1 hour (6.91-11.06 mg/kg)[5] resulted in (unspecified) serious disturbances (ACGIH 1991). Sitting in a car seat contaminated with aniline resulted in a methemoglobin level of 53% with cyanosis, dyspnea, fatigue, and dizziness in one individual. Recovery occurred within 24 hours following medical treatment (U.S. EPA 1985).

2. Animals — Increased spleen weight, splenic erythropoietic activity, splenic sinusoidal engorgement, and splenic hemosiderin content were reported in Colworth-Wistar male rats 12 days after receiving aniline in single oral doses of 20 mg/kg or above. A slight increase in hepatic hemosiderin content and erythropoietic activity was also reported but no gross pathological changes in the liver were seen (U.S. EPA 1985).

   Oral LD_{50} values of 440 mg/kg (males) and 1072 mg/kg have been reported for rats, and an oral LD_{50} of 841 mg/kg was reported for mice. An inhalation LC_{50} value of 950 mg/m^3 (250 ppm) for 4 hours was reported for rats, and a dermal LD_{50} of 1320 mg/kg was reported for guinea pigs (U.S. EPA 1985).

C. Subchronic/Chronic Effects

Increased methemoglobin production and adverse splenic effects are the major non-neoplastic effects reported with extended exposure to aniline. Adverse effects on the liver and kidneys have been reported in some studies. A no-observed-adverse-effect level of 3.4 mg/m^3 was determined for continuous inhalation exposure in rats, dogs, mice, and guinea pigs. A lowest-observed-adverse-effect level of 64.7 mg/m^3 was identified in rats exposed by inhalation for 2 weeks based on increased methemoglobin production. EPA has derived an inhalation RfC[6] of 0.001 mg/m^3 for aniline exposure.

1. Humans — Increased methemoglobin and decreased hemoglobin, erythrocyte count, and coagulative factors were reported in an occupational study on workers exposed to 1.3 to 2.75 mg/m^3 (0.19-0.39 mg/kg/day) aniline for 3 to 5 years compared to an unexposed control group. An increase in methemoglobin was reported on reexamination after one year, however no numerical data were given (U.S. EPA 1994).

---

[5] For dose comparison purposes, this has been calculated using the factor, 3.87, to convert ppm to mg/m^3, which is multiplied by 0.0179 (the calculated occupational 1-hour breathing rate, 1.25 m^3, divided by the assumed adult body weight, 70 kg) to obtain the dose in mg/kg/hr assuming 100% absorption (U.S. EPA 1988).

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

2. Animals — Nine male Wistar rats, 2 dogs, 20 female albino mice, and 10 guinea pigs were exposed to 5 ppm (19 mg/m^3) aniline, 6 hours/day, 5 days/week for 26 weeks. The continuous exposure level was calculated to be 3.4 mg/m^3. Methemoglobin was slightly increased only in rats, but there were no pathological changes in any organs in any species tested attributed to aniline exposure. Although only one dose level was given in this study, it was identified as a no-observed-adverse-effect level (NOAEL) (U.S. EPA 1994). This value together with a 2-week study (see below for discussion) LOAEL of 17 ppm (64.7 mg/m^3) for increased methemoglobin levels and minimal splenic effects in rats were utilized by the U.S. EPA (1994) to calculate a chronic inhalation RfC of 0.001 mg/m^3 for aniline.

Male Crl:CD rats were exposed in an inhalation study to 0, 17, 45, or 87 ppm aniline vapors, 6 hours/day, 5 days/week for 2 weeks. A dose-related increase in methemoglobin level at 17 ppm and above was reported. The increase seen at 17 ppm (64.7 mg/m^3) was not statistically significant, compared to controls, and spleen histopathology was judged to be minimal. The higher doses resulted in cyanosis, anemia, increased relative spleen weight, and increases in erythropoietin foci, reticuloendothelial cell hypertrophy and hemosiderin deposition. The 17 ppm (64.7 mg/m^3) dose was identified as a lowest-observed-adverse-effect level (LOAEL) by U.S. EPA 1994

.

Dietary exposure of Fischer 344 rats to 3000 or 6000 ppm (150 or 300 mg/kg)[7] aniline hydrochloride for 103 weeks resulted in an increased incidence of splenic hyperplasia and erythropoiesis, and renal and hepatic hemosiderosis in both sexes (U.S. EPA 1985). B6C3F$_1$ mice were fed diets containing 0, 6000, or 12,000 ppm (480 or 960 mg/kg)[8] aniline hydrochloride for 103 weeks. Males developed diffuse inflammation of the bile duct at both dose levels, and females were found to have an increased incidence of splenic erythropoiesis and an accumulation of serous fluid in the uterine cavity at 12,000 ppm (U.S. EPA 1985).

Wistar rats were given 300-1200 ppm aniline in their drinking water for 80 weeks. No changes in body weight or liver weight were reported; however, there were slight but dose-related decreases in erythrocyte counts, hemoglobin levels, and hematocrits of treated rats (U.S. EPA 1985).

Daily oral doses of 110 mg aniline/kg for 5, 10, or 20 days resulted in increased spleen weight, splenic congestion, and hematopoiesis in Fischer 344 rats. Increased cellularity in bone marrow was also reported at all time points. Increased splenic hemosiderosis was seen but only after 20 days of exposure. No adverse effects were reported in the livers of the animals in this study (U.S. EPA 1985).

D. Carcinogenicity

Tumors of the spleen and body cavity were induced in two strains of rats in separate dietary studies. This information and the genotoxicity evidence of aniline were used by U.S. EPA as a basis for classifying aniline as a B2, probable human carcinogen.

1. Humans — The incidence of bladder tumors was investigated in British chemical dye workers exposed to aniline and other aromatic amines. There was inadequate evidence

---

[7] For dose comparison purposes this has been calculated by multiplying the ppm value by a factor of 0.05. the food consumption conversion factor for the rat.

[8] For dose comparison purposes this has been calculated by multiplying the ppm value by a factor of 0.13, the food conversion factor for the mouse.

to conclude that aniline alone was a cause of bladder tumors (U.S. EPA 1994).

2. Animals — Groups of 130 CD-F rats per sex were fed diets containing 0, 200, 600, or 2000 ppm (10, 30, or 100 mg/kg)[4] aniline hydrochloride for two years. An increased incidence of primary splenic sarcomas was reported in male rats at the 2000 ppm dose. Stromal hyperplasia and splenic red pulp fibrosis, probable precursors of sarcoma, were observed in both sexes at the high dose (U.S. EPA 1994).

   Groups of 50 Fischer 344 rats per sex were given aniline hydrochloride in the diet at 0, 3000, or 6000 ppm for 103 weeks (Section IV.C.2). The animals were killed and examined after a 4-7 week observation period. Significant, dose-related increases in the incidence of splenic hemangiosarcomas, multiple organ fibrosarcomas, and malignant pheochromocytomas were reported in treated males compared to controls. The incidences of splenic sarcomas and fibromas were also increased but were not dose dependent. Splenic sarcomas were slightly (3/50 at high dose, 0/24 in control) increased at the high dose in females. The incidence of sarcomas in a pooled group of control female Fisher 344 rats was 0/249. Likewise, the incidence of hemangiosarcomas, fibrosarcomas, and sarcomas in a pooled group of control male Fisher 344 rats was 0/250 (U.S. EPA 1994; 1985; NCI 1978). A parallel study was also conducted with $B6C3F_1$ mice in which the mice were fed diets containing 0, 6000, or 12,000 ppm aniline hydrochloride for 103 weeks. No significant increase in the incidence of any type of tumor was observed in either sex (U.S. EPA 1985; NCI 1978).

E. Genotoxicity

Results of short term mutagenicity testing of aniline are mixed. Positive results of a mouse bone marrow micronucleus assay of aniline have been submitted to EPA (Federal Register 1989b) in response to a request for testing under Section 4 of the Toxic Substances Control Act (TSCA). Aniline was positive in the mouse lymphoma forward mutation test and for sister-chromatid exchanges (SCE) in Chinese hamster ovary cells (ACGIH 1991) and in Swiss mice bone marrow cells (U.S. EPA 1985). Aniline did not increase the SCE in human fibroblasts; however, both 2-aminophenol and N-phenylhydroxylamine, potential metabolites of aniline, were positive for SCE (U.S. EPA 1985). Aniline was positive in the cell transformation assay in mouse Balb/3T3 cells, but was negative with Syrian hamster embryo cells or Fischer 344 rat embryo cells infected with murine leukemia virus (U.S. EPA 1994). Aniline has generally been reported negative in reverse mutation tests in multiple strains of *Salmonella typhimurium* with or without metabolic activation. In one assay, a weakly positive result was reported in *S. typhimurium* strains TA98 and/or TA100 with metabolic activation. Results of DNA interaction tests with *E. coli* are negative (U.S. EPA 1985).

F. Developmental/Reproductive Toxicity

Conclusive information on human and animal developmental/reproductive effects of aniline is not available. Limited evidence from animal and human studies suggest that repeat exposure to aniline may cause adverse effects on the reproductive system in female workers and fetal toxicity in animals.

1. Humans — A possible correlation has been reported between exposure to aniline and other organics encountered by Russian aniline dye industry workers and the incidence of menstrual disturbances and ovarian dysfunction. However, the effect was dependent on the nutritional status of the exposed individuals. The dye workers also experienced an increase in abortion rates, but the strenuous physical exertion required for the job complicates the interpretation of these results (U.S. EPA 1985).

2. Animals — Daily treatment of 50 pregnant CD-1 mice from day 7 to 14 of gestation with 560 mg/kg/day aniline by gavage resulted in slightly decreased pup weight gain and pup survival during the first 3 days postpartum. The average number of pups per litter was the same in the control and treated groups, however fewer litters were produced in the treated group. There was evidence of maternal toxicity. Six dams died and the mean weight gain was significantly decreased during treatment compared to control dams (U.S. EPA 1985).

   Aniline has been shown to cause malformations following injection into the inner shell membrane of 3-day-old chick embryos (U.S. EPA 1985). Embryonic malformations occurred when eggs from bass, goldfish, and catfish were exposed to aniline at 34-100 mg/L for 4 days. Exposure of clawed toad eggs to 10 mg aniline/L for 4 days resulted in 11% abnormal embryos and 28% mortality. No abnormalities or deaths were seen in the control animals (U.S. EPA 1985).

G. Neurotoxicity

Central nervous system effects following aniline exposure are secondary to effects associated with the anoxia resulting from methemoglobinemia.

1. Humans — Exposure to aniline caused central nervous system symptoms such as euphoria and headache. Continued exposure increases the symptoms to lightheadedness, ataxia, and weakness. These symptoms are concurrent with signs of anoxia resulting from methemoglobinemia (Beard and Noe 1981).

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

V. ENVIRONMENTAL EFFECTS

The aniline industry has completed aquatic toxicity studies in response to an EPA request for testing. These tests show that aniline is highly toxic to aquatic life. Reported LC_{50} values for daphnids are less than 1 mg/L. Reported chronic values for daphnids are less than 0.1 mg/L.

A. Toxicity to Aquatic Organisms

Ninety-six-hour LC_{50} values for fish are: 41 mg/L in hard water and 20 mg/L in soft water for *Salmo gairdneri* (rainbow trout), and 32-53 mg/L for *Brachydanio rerio* (zebrafish). Forty-eight-hour LC_{50} values for fish are: 43 mg/L for *Salmo gairdneri*, 100 mg/L for *Poecilia reticulata* (guppy), 165 mg/L for *Oryzias latipes* (medaka), 65 mg/L for *Pimephales promelas* (fathead minnow), and 61-78 mg/L for *Leuciscus idus* (golden orfe) (U.S. EPA 1985). A 96-hour EC_{50} for aniline in algae is 19 mg/L; a 48-hour EC_{50} for aniline in daphnids is 0.65 mg/L (Rabert 1991). The geometric average concentration 48-hour LC_{50} for aniline in daphnids is 0.57 mg/L, based on three tests (Newsome 1995).

Results of acute and chronic aquatic testing of aniline in invertebrates have been submitted to EPA (Federal Register 1989a and 1989c) in response to a request for testing under Section 4 of The Toxic Substance Control Act. The 96-hour LC_{50} of aniline is 2.3 mg/L in the amphipod *Gammarus fasciatus*. Results of a 21-day chronic flow through assay of daphnids shows decreased reproductive capacity at 0.027 mg/L. Test results from the chronic daphind toxicity study have been described as unreliable because of several deficiencies, including unstable test concentrations and poor replication of test results (Rabert 1991).

B. Toxicity to Terrestrial Organisms

Aniline is unlikely to exist in U.S. terrestrial environments in sufficient concentrations to cause serious acute or chronic effects to terrestrial organisms. The toxicity data reported for rats, mice, and guinea pigs (see sections IV.B.2. and IV.C.2.) suggest that no effects would be seen at normally expected U.S. environmental concentrations.

C. Abiotic Effects

Aniline acts as a radical scavenger in the atmosphere and inhibits the formation of photochemical smog (Howard 1989).

VI. EPA/OTHER FEDERAL/OTHER GROUP ACTIVITY

The Clean Air Act Amendments of 1990 list aniline as a hazardous air pollutant. Occupational exposure to aniline is regulated by the Occupational Safety and Health Administration (OSHA). The OSHA permissible exposure limit (PEL) is 5 parts per million parts of air (ppm) as an 8-hour time-weighted average (TWA) (29 CFR 1910.1000). 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 aniline.

TABLE 4. EPA OFFICES AND CONTACT NUMBERS INFORMATION ON ANILINE

EPA Office	Statute	Contact Number
Pollution Prevention & Toxics	PPA[a]	(202) 260-1023
	EPCRA (§313/TRI)[b]	(800) 535-0202
	TSCA (§4, 8D)[c]	(202) 554-1404
Air	Clean Air Act (111, 112B)[d]	(919) 541-0888
Water	Clean Water Act (311)[e]	(202) 260-7588
Solid Waste & Emergency Response	RCRA (Action levels: water, 6E-3 mg/L; soils, 1E+2 mg/kg)[f]	(800) 535-0202
	CERCLA (RQ: 5000 pounds)[g]	(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]Listed as hazardous air pollutant under § 112 of Clean Air Act [42 U.S.C. 7401 et seq.]

[e]**CWA**: Clean Water Act; regulates waters of the United States, including surface waters, ground waters, and wetlands [40 CFR Part 131 (1994)].

[f]**RCRA**: Resource Conservation and Recovery Act of 1976, (codified as amended at 42 U.S.C. §6901 *et seq*). **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 Measures Study (U.S. EPA 1990).

[g]**CERCLA**: Comprehensive Environmental Response, Conpensation, 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 Naltional Response Center [40 CFR Part 302 (1991)].

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

Other Agency/Department/Group	Contact Number
American Conference of Governmental Industrial Hygienists [TLV-TWA, 2 ppm (7.6 mg/m^3); skin exposure][a]	(513) 742-2020
Consumer Product Safety Commission	(301) 504-0994
Food & Drug Administration	(301) 443-3170
National Institute for Occupational Safety & Health [TWA, 2 ppm (8 mg/m^3); IDLH, 100 ppm; skin exposure, Ca, LF][b]	(800) 356-4674
Occupational Safety & Health Administration [TWA, 5 ppm (19 mg/m^3][c] Check local phone book for phone number under Department of Labor	

[a]**TLV-TWA** : Time-Weighted-Average concentration for a normal 8-hr workday and a 40-hr workweek to which nearly all workers may be repeatedly exposed without adverse effects. **Skin exposure** : air sampling alone is insufficient to accurately quantitate exposure. Measures to prevent significant cutaneous absorption may be required (ACGIH 1993-1994).

[b]**TWA**: Time-Weighted-Average concentration for up to a 10-hour workday during a 40-hour workweek. **IDLH**: immediate danger to life and health. **Ca**: potential human carcinogens. **LF**: reduce exposure to lowest feasible concentration; when Ca designation accompanies lowest feasible designation, use of only the most reliable and protective respirators is recommended. **Skin exposure** : air sampling alone is insufficient to accurately quantitate exposure. Measures to prevent significant cutaneous absorption may be required (NIOSH 1990, 1992).

[c]**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).

VII. CITED REFERENCES

ACGIH. 1991. Documentation of the Threshold Limit Values and Biological Exposure Indices, Sixth edition. American Conference of Governmental Industrial Hygienists, Inc., Cincinnati, OH, pp. 256-257.

ACGIH. 1993-1994. American Conference of Governmental Industrial Hygienists. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. ACGIH, Cincinnati, OH.

Beard RR and Noe JT. 1981. Aromatic Nitro and Amino Compounds. In: Clayton GD, Clayton FE. 1981-1982. Patty's Industrial Hygiene and Toxicology, 3rd ed., Vol. 2A. John Wiley & Sons, New York, pp. 2413-2446.

Budavari S, O'Neil MJ, Smith A, Heckelman PE (Eds.). 1989. The Merck Index, 11th ed. Merck & Co., Inc., Rahway, N.J., p. 686.

Federal Register. 1989a. 54 FR 25167. June 13, 1989.

Federal Register. 1989b. 54 FR 32117. August 4, 1989.

Federal Register. 1989c. 54 FR 33773. August 16, 1989.

Grayson, M. (ed.). Kirk-Othmer Concise Encyclopedia of Chemical Technology, Third edition. New York: John Wiley and Sons, 1985.

Howard PH. 1989. Handbook of Environmental Fate and Exposure Data for Organic Chemicals, Vol. I, Large Production and Priority Pollutants. Lewis Publishers, Inc., Chelsea, MI. pp. 44-52.

Mannsville. 1992. Mannsville Chemical Products Corporation. Aniline. Chemical Products Synopsis, December 1992.

Newsome L. 1995. Memorandum from Larry Newsome, HERD/EEB, to Richard Wormell, CSRAD/AIMB. Subject: Comments on OPPT Fact Sheet for Aniline and Others. July 13, 1995.

NCI. 1978. National Cancer Institute. Bioassay of Aniline Hydrochloride for Possible Carcinogenicity. CAS No.: 142-04-1. ITS Carcinogenesis Technical Report Series 130. U.S. DHEW, PHS, NIH, Bethesda, MD. DHEW Publ. No. (NIH) 78 to 1385. (as cited in U.S. EPA 1985).

NIOSH. 1990. National Institute for Occupational Safety and Health. Pocket Guide to Chemical Hazards. NIOSH, Cincinnati, OH.

NIOSH. 1992. National Institute for Occupational Safety and Health. Recommendations for Occupational Safety and Health. Compendium of Policy Documents and Statements, NIOSH, Cincinnati, OH.

OSHA. 1993. Occupational Safety and Health Administration. Air Contaminants. Final Rule. 29 CFR part 1910. Fed. Reg. 58:35338-35351.

Rabert WS. 1991. Memorandum from William Rabert, Environmental Effects Branch, to John Harris, Chemical Testing Branch. Subject: Review of *Daphnia magna* 21-Day Life Cycle Toxicity Test on Aniline. Jan 8, 1991.

Sax, N.I., and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary, Eleventh edition. New York: Van Nostrand Reinhold Company, 1987.

TRI92. 1994. 1992 Toxics Release Inventory, Public Data Release. Office of Pollution Prevention and Toxics, U.S. EPA, Washington, D.C., p. 87.

U.S. EPA. 1985 Health and Environmental Effects Profile for Aniline. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response Washington, D.C., Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-P136.

U.S. EPA. 1988. U.S. Environmental Protection Agency. Methodology for Evaluating Potential Carcinogenicity in Support of Reportable Quantity Adjustments Pursuant to CERCLA Section 102. Carcinogen Assessment Group, Office of Health and Environmental Assessment, U.S. EPA, Washington, D.C., pp. 21,22. OHEA-C-073.

U.S. EPA. 1990. Examples of concentrations meeting criteria for action levels. Fed. Reg. 55:30865-30867.

U.S. EPA. 1994. U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) Online. Coversheet for Aniline. Office of Health and Environmental Assessment, U.S. EPA, Cincinnati, OH. Retrieved 10/94.

USITC. 1994. United States International Trade Commission. Synthetic Organic Chemicals: United States Production and Sales, 1992, 76th edition. USITC Publication 2720, February 1994.

Verschueren, K. 1983. Aniline. In: Handbook of Environmental Data on Organic Chemicals, Second Edition. Van Nostrand Reinhold Co., New York, pp. 201-203.

Windholz, M. (ed.). The Merck Index, Tenth edition. Rahway, N.J.: Merck and Company, Inc., 1983.

APPENDIX A. SOURCES SEARCHED FOR FACT SHEET PREPARATION

ACGIH. Most recent. American Conference for Governmental Industrial Hygienists, Inc. TLVs®. Documentation of the Threshold Limit Values and Biological Exposure Indices, ... ed. ACGIH, Cincinnati, OH.

AQUIRE. 1994. Aquatic Information Retrieval online data base. Chemical Information Systems, Inc., a subsidiary of Fein-Marquart Assoc.

ATSDR. 1989-1994. Agency for Toxic Substances and Disease Registry. Toxicological Profiles. Chamblee, GA: ATSDR.

Budavari S, O'Neil MJ, Smith A, Heckelman PE (Eds.). 1989. The Merck Index, 11th ed. Rahway, N.J.: Merck & Co., Inc.

Clayton GD, Clayton FE. 1981-1982. Patty's Industrial Hygiene and Toxicology, 3rd ed., Vol. 2C. New York: John Wiley & Sons. (Soon to be updated)

Clean Air Act. 1990. As amended. 42 U.S.C. 7412.

GENETOX. 1994. U.S. EPA GENETOX Program, computerized database.

Howard, P.H., Ed. 1989. Handbook of Environmental Fate and Exposure Data. Lewis Publishers, Chelsea, MI.

HSDB. 1994. Hazardous Substances Data Bank. MEDLARS Online Information Retrieval System, National Library of Medicine.

IARC. 1979-1994. International Agency for Research on Cancer. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Lyon: IARC.

IPCS. 19.... International Programme on Chemical Safety. Environmental Health Criteria. World Health Organization, Geneva, Switzerland.

NIOSH (National Institute for Occupational Safety and Health). 1992. NIOSH Recommendations for Occupational Safety and Health. Compendium of Policy Documents and Statements. Cincinnati, OH: NIOSH.

NTP. 199... National Toxicology Program. Toxicology and Carcinogenesis Studies. Tech Rep Ser.

NTP. 199... National Toxicology Program. Management Status Report. Produced from NTP Chemtrack system. April 8, 1994. National Toxicology Program, Research Triangle Park, NC.

OSHA. 1993. Occupational Safety and Health Administration. Table Z-2. Limits for Air Contaminants.

TRI92. 1994. 1992 Toxics Release Inventory. Public Data Release. Office of Pollution Prevention and Toxics (7408), U.S. Environmental Protection Agency, Washington, D.C.

TSCATS. 199... MEDLARS Online Information Retrieval System, National Library of Medicine.

U.S. Air Force. 1989. The Installation Restoration Toxicology Guide, Vols. 1-5. Wright-Patterson Air Force Base, OH.

U.S. EPA . 1991. U.S. Environmental Protection Agency. Table 302.4 List of Hazardous Substances and Reportable Quantities 40 CFR, part 302.4:3-271.

U.S. EPA. U.S. Environmental Protection Agency. Appendix A. Examples of Concentrations Meeting Criteria for Action Levels. 40 CFR Part 264.521 (a)(2)(i-iv). Fed. Reg. 55:30865-30867.

U.S. EPA. Most current. Drinking Water Regulations and Health Advisories. Office of Drinking Water, U.S. Environmental Protection Agency, Washington, D.C.

U.S. EPA. Most Current. Health Effects Assessment Summary Tables. Cincinnati, OH: Environmental Criteria and Assessment Office, U.S.EPA.

U.S. EPA reviews such as Health and Environmental Effects Documents, Health and Environmental Effect Profiles, and Health and Environmental Assessments, HERD Analogue Profiles, ITC Documents.

U.S. EPA. 1994. Integrated Risk Information System (IRIS) Online. Cincinnati, OH: Office of Health and Environmental Assessment.