Aniline; CASRN 62-53-3

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR Aniline

File First On-Line 09/07/1988

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	not evaluated	
Inhalation RfC (I.B.)	yes	11/01/1990
Carcinogenicity Assessment (II.)	yes	09/07/1988

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Aniline
CASRN — 62-53-3

Not available at this time.

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Aniline
CASRN — 62-53-3
Last Revised — 11/01/1990

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.B.1. Inhalation RfC Summary

Critical Effect	Exposures*	UF	MF	RfC
Lack of toxicity (however, see study 2) **20-26 Week Inhalation Rat, Guinea Pigs and Mouse Study** **Oberst et al., 1956**	NOAEL: 19 mg/cu.m (5 ppm) NOAEL(ADJ): 3.4 mg/cu.m NOAEL(HEC): 3.4 mg/cu.m LOAEL: None	3000	1	1E-3 mg/cu.m
Mild spleen toxicity 2-Week **Rat Inhalation Study**	NOAEL: None LOAEL: 64.7 mg/cu.m (17 ppm) LOAEL(ADJ): 11.6 mg/cu.m LOAEL(HEC): 11.6 mg/cu.m			

Critical Effect	Exposures*	UF	MF	RfC
duPont deNemours, 1982				

*Conversion Factors -- MW = 93.12;

Oberst et al., 1956: Assuming 25C and 760 mmHg, NOAEL (mg/cu.m.) = 5 ppm x 93.12/24.45 = 19. NOAEL(ADJ) = NOAEL (mg/cu.m.) x 6 hours/24 hours x 5 days/7 days = 3.4 mg/cu.m. The NOAEL(HEC) was calculated for a gas:extra- respiratory effect in the rats assuming periodicity was attained. Since the b:a lambda values are unknown for the experimental species (a) and humans (h), a default value of 1.0 is used for this ratio. NOAEL(HEC) = 3.4 x (b:a lambda(a)/b:a lambda(h)) = 3.4 mg/cu.m;

duPont deNemours, 1982: Assuming 25C and 760 mmHg, LOAEL (mg/cu.m.) = 17 ppm x 93.12/24.45 = 64.7. LOAEL(ADJ) = LOAEL (mg/cu.m) x 6 hours/24 hours x 5 days/7 days = 11.6 mg/cu.m. The LOAEL(HEC) was calculated for a gas:extrarespiratory effect assuming periodicity was attained. Since the b:a lambda values are unknown for the experimental species (a) and humans (h), a default value of 1.0 is used for this ratio. LOAEL(HEC) = 11.6 x (b:a lambda(a)/b:a lambda(h)) = 11.6 mg/cu.m.

I.B.2. Principal and Supporting Studies (Inhalation RfC)

Oberst F.W., E. Hackley, C. Comstock. 1956. Chronic toxicity of aniline vapor (5 ppm) by inhalation. Arch. Ind. Health. 13: 379-384.

duPont deNemours and Company, Inc. 1982. Subacute inhalation toxicity study of aniline in rats. OTS No. 878220240. Fiche No. 0215025. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Co-principal studies are chosen to delineate effect levels. The study of Oberst et al. (1956), which demonstrated a NOAEL, was complemented by the duPont deNemours (1982) study which provided a LOAEL.

Oberst et al. (1956) exposed (whole body) 9 male Wistar rats and 2 dogs for 26 weeks, and 20 female albino mice and 10 guinea pigs for 20 weeks, to 5 ppm (19 mg/cu.m) of reagent grade aniline vapor for 6 hours/day, 5 days/week (duration-adjusted value = 3.4 mg/cu.m.). Analysis of whole blood, serum, and body weight was carried out on all animals and urinalysis, rectal temperatures, and blood pressure were determined for the dogs. Pathology and histopathology

including liver, lung, kidney, and spleen (personal communication with pathologist of the report), was conducted on the dogs and four of the smaller animals at termination of exposure. Mention is made that the rats began to lose weight during the last 3 weeks of exposure although no figures are given. Blood analysis indicated an increase in methemoglobin in rats only (0.6%, no statistics or control levels given). No organ pathologies in any species tested were attributable to aniline vapors. Study limitations include testing at only one dose, statistical reporting, incomplete histopathology, and autopsy of a small number of animals. Based on the slight increase of methemoglobin content and the absence of spleen toxicity, 3.4 mg/cu.m may be designated as a free-standing NOAEL.

In a repeated exposure inhalation study, male Crl:CD rats (16/group) were exposed (head-only) to 0 (air-exposed controls), 17 ppm (64.7 mg/cu.m), 45 ppm (171.4 mg/cu.m), or 87 ppm (331.3 mg/cu.m) aniline vapors, 6 hours/day, 5 days/week, for 2 weeks (duPont, 1982). Duration-adjusted values for these exposures are 0, 11.6, 30.6, or 59.2 mg/cu.m. Toxicity was investigated utilizing urinalysis, hematology (including methemoglobin), organ and body weights, and gross pathology, and histopathology (including lungs and trachea), both following the last exposure and after a 13-day recovery period. Methemoglobin levels were elevated in a dose-dependent manner at 87 ppm (4.2 to 23%) and at 45 ppm (2.2 to 5.4%); but at 17 ppm the levels were not significantly different from the controls (0 to 2.9%). This increase was accompanied by clinical symptoms as the animals exposed to 87 ppm were judged as slightly cyanotic. The animals exposed to 87 or 45 ppm aniline vapors were anemic with decreases in RBC counts, hemoglobin content, mean corpuscular hemoglobin concentration (MCHC), and hematocrit, and accompanying increases in mean relative spleen weight. Also, exposure-dependent increases in erythropoietin foci, reticuloendothelial (RE) cell hypertrophy, and hemosiderin deposition were noted within the spleen of these high-exposure animals. Although judged as minimal or isolated, splenic histopathology was noted in the low-dose (17 ppm) group. Hemosiderin deposition occurred in 4 of 5 animals vs. 0 of controls, and RE cell hypertrophy and increases in erythropoietic foci both occurred in 1 of 5 treated, but in 0 of 5 controls. Based on these mild splenic effects, 17 ppm (HEC = 11.6 mg/cu.m) is designated as a LOAEL. Although the duration of this study is only 2 weeks, the critical effects (methemoglobin increase and splenic involvement) were already manifest (see Section I.B.4.).

I.B.3. Uncertainty and Modifying Factors (Inhalation RfC)

UF — Uncertainty factors of 10 are used for the protection of sensitive human subpopulations, 10 to allow for animal to human variability, 10 for use of a subchronic study, and 3 for lack of appropriate reproductive studies. A modifying factor of 1 is used.

MF — None

I.B.4. Additional Studies/Comments (Inhalation RfC)

The study of Kim and Carlson (1986) demonstrates in rats exposed to aniline that induced formation of methemoglobin occurs quickly after exposure, that methemoglobin does not accumulate at low doses, and that methemoglobin is removed from the blood with a measurable half-life. These authors exposed groups (maximum of 15/group) of male Sprague-Dawley rats to 0, 10, 30, 50, or 150 ppm aniline for 8 hours/day for 5 days or 12 hours/day for 4 days. The duration-adjusted values were 0, 9, 27, 45, or 136 mg/cu.m for the 5-day exposure and 0, 11, 33, 54, or 163 mg/cu.m for the 4-day exposures. An increase in methemoglobin values over control levels was noted at the beginning of the second day of exposure to 150 and 50 ppm, with methemoglobin levels tending to cumulate and become at least partially additive. Although a slight increase in methemoglobin was noted in the 30 ppm groups (HECs = 27 to 33 mg/cu.m and presumably in the 10 ppm groups (HECs = 9 to 11 mg/cu.m), no cumulative effects were noted. In additional experiments in which rats were intraperitoneally injected with aniline, the half-life of methemoglobin in blood was estimated at 177 minutes (almost 3 hours). The rapidity and reversibility of methemoglobin formation in humans in response to aniline was shown by Jenkins et al. (1972), who detected maximum levels of methemoglobin in humans at 2 hours after oral exposure, but normal levels at 3 hours. This response would indicate that methemoglobin has a longer half-life in rats than in humans (3 hours vs. <1 hour).

Humans appear to be more sensitive than rats to aniline exposure (as indicated by formation of methemoglobin). Jenkins et al. (1972) noted that after oral administration of aniline to volunteers and rats, the dose that produced increased levels of methemoglobin was much lower for humans than for rats. After correcting these values for inhalation exposure (assumed 100% absorption), the concentrations of aniline producing the same plasma levels of methemoglobin in humans were still less than in rats by a factor of 12 to 90. Other documentation indicates that this disparity in sensitivity may also apply to inhalation exposure. Whereas Vasilinko claims "definite" increases in methemoglobin in workers exposed to 0.5 to 1.0 mg/cu.m. aniline vapors (duration adjusted), the rat studies of duPont deNemours (1982) and Kim and Carlson (1986) indicate effects on methemoglobin level at 31 mg/cu.m and 45 to 54 mg/cu.m aniline, respectively. The reason for this increased sensitivity in humans is not known and does not appear to be related to the half-life of methemoglobin in the serum, which is three times longer in rats than in humans.

The occurrence of some non-neoplastic splenic lesions (fibrosis, mesothelial hyperplasia) in animals exposed to aniline is related to accumulation of hemosiderin deposits thought to be formed secondarily to methemoglobin (Goodman et al., 1984). On the other hand, Weinberger et al. (1985) showed a clear relationship between occurrence of splenic lesions and the development of splenic sarcomas in animals fed aniline HCl or the aniline- based food coloring D and C Red No. 9, even though this latter compound is not reported to produce

methemoglobinemia. Thus methemoglobin formation may not be an obligate precursor to splenic hyperplastic lesions for all aniline compounds.

An occupational study was conducted on workers in a plant producing diphenylamine in which aniline was used as the raw material (Vasilenko et al., 1972). The only harmful chemicals to which workers were exposed were aniline and hydrogen chloride (present at 5 mg/cu.m) under conditions that excluded liquid aniline as potential source of exposure to skin. Aniline concentrations ranged from 1.3 to 2.75 mg/cu.m (occupational adjustment = concentration x 10 cu.m air/ 20 cu.m air x 5 days/7 days = 0.5 to 1.0 mg/cu.m). (The method or frequency of measurement was not reported.) At the beginning of the study, the exposed group consisted of 47 men and 11 women (duration of employment was 3 to 5 years for 65.5% of the workers). The control group consisted of 67 men and 8 women. On reexamination 1 year later, 33 were evaluated from the exposed group (sex was not given). A "definite" increase in methemoglobin content was claimed during the first year (no data presented). A decrease in hemoglobin levels, erthyrocyte number, and coagulative factors was also reported although no data were presented. Lung parameters were not examined. No scientific judgment could be made upon the effects claimed in this study.

The potential for production of developmental and reproductive effects at 10, 30, and 100 mg/kg/day aniline HCl was studied in Fischer 344 rats (24- 27/group) gavaged on gestational days 7 to 20 (CIIT, 1981). A significant increase in methemoglobin (13.7% vs. 3.5% in controls) and altered hematological measures (decrease in RBCs and an increase in MCV) were observed in the dams of the 100 mg/kg group. Compared with controls, a dose-dependent increase in the relative spleen weight was observed in the dams of all treatment groups. No histopathology was performed in this study. No treatment-related alterations were observed in the reproductive parameters examined (including number of corpora lutea, implantations, resorptions, or dead fetuses). In the fetuses of the 100 mg/kg group, the relative liver weight and erythrocyte size were significantly elevated over control values. The methemoglobin levels in the fetuses were not significantly different from those of the controls. Aniline produced no other dose-related adverse effects in the fetuses and dams. In the next phase of this study, development of pups was observed from parturition to postnatal day 60. An increase in the number of postnatal deaths occurred in this phase, from 8% in the controls, to 9.6 % at 10 ppm, 20.8% at 30 ppm, and 12.5% at 100 ppm. The cause of death was not determined in any of these pups. No embryotoxicity or teratogenicity was observed at levels of aniline that caused maternal toxicity. Based on the increases in methemoglobin formation, a NOAEL of 30 mg/kg/day is designated for maternal effects, with a NOAEL of 100 mg/kg/day for fetal effects.

Feeding studies of aniline hydrochloride also note splenic pathology, methemoglobin formation, and hemosiderosis as principal non-neoplastic lesions (U.S. EPA, 1985).

I.B.5. Confidence in the Inhalation RfC

Study — Low
Database — Low
RfC -- Low

Although the duration of the Oberst et al (1656) study is longer than 13 weeks and is conducted with several species, it is poorly reported, used a single exposure concentration and small numbers of animals. Although of short duration, the study of duPont deNemours (1982) is well conducted and examines a number of valid endpoints. Confidence in the database is low as no appropriate reproductive studies were located. Low confidence in the RfC follows.

I.B.6. EPA Documentation and Review of the Inhalation RfC

Source Document — This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation — U.S. EPA, 1985

Agency Work Group Review — 09/20/1990

Verification Date — 09/20/1990

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfC for Aniline conducted in September 2002 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

I.B.7. EPA Contacts (Inhalation RfC)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Aniline
CASRN — 62-53-3
Last Revised — 09/07/1988

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. Evidence for Human Carcinogenicity

II.A.1. Weight-of-Evidence Characterization

Classification — B2; probable human carcinogen

Basis — Induction of tumors of the spleen and the body cavity in two strains of rat, and some supporting genetic toxicological evidence.

II.A.2. Human Carcinogenicity Data

Inadequate. Case et al. (1954) investigated the occurrence of bladder tumors among British workers in the chemical dye industry. These workers were generally exposed to a number of different aromatic amines including aniline, alpha- and beta-napthylamine, benzidine and auramine. Although little specific exposure information was available, it was the authors' judgment that there was not sufficient evidence to suggest that aniline itself is a cause of bladder tumors.

II.A.3. Animal Carcinogenicity Data

Sufficient. Aniline hydrochloride was administered in the diet for 2 years to CD-F rats (130 rats/sex/group) at levels of 0, 200, 600 and 2000 ppm (CIIT, 1982). An increased incidence of primary splenic sarcomas was observed in male rats in the high-dose group. Stromal hyperplasia and fibrosis of the splenic red pulp, which may represent a precursor lesion of sarcoma, was also observed in the high-dose males and, to a lesser degree, in the female rats.

Dietary aniline hydrochoride was administered at 0, 3000 or 6000 ppm to 50 male and 50 female Fischer 344 rats for 103 weeks (NCI, 1978). The animals were sacrificed at 107-110 weeks. The male rats showed statistically significant dose-related trends in incidence of hemangiosarcomas and sarcomas or fibrosarcomas. The males also had statistically significantly increased incidences of hemangiosarcoma in the spleen, fibrosarcoma and sarcoma (not otherwise specified) in the body cavity and spleen, and a significant dose-related trend in incidence of malignant pheochromocytoma. According to the authors, there was a possible association in the female rats between aniline hydrochloride treatment and the increased incidence of fibrosarcoma and sarcoma in multiple organs of the body cavity. None of the pooled control groups of 249 female and 250 male rats were observed to have fibrosarcoma or sarcoma in the spleen or multiple organs of the body cavity. Food containing 0, 6000 or 12,000 ppm aniline hydrochloride was also given to 50 male and 50 female B6C3F1 mice for 103 weeks (NCI, 1978). No statistically significant increase in any type of tumor in male or female mice was observed.

Hagiwara et al. (1980) administered 0, 0.03, 0.06 or 0.12% aniline alone or in combination with the comutagen norharman in the drinking water to 10 to 18 male Wistar rats for 80 weeks. The incidence of forestomach papillomas was low and did not appear to be dose-related. One pituitary adenoma was observed in the 0.03% aniline group. No tumors were observed in the controls. According to the authors, oral administration of aniline and norharman, separately or in combination, had no cocarcinogenic effect on the urinary bladder or other organs examined. Druckrey (1950) exposed 50 rats to a dose of 22 mg/day aniline hydrochloride in the drinking water for their lifetime. Fifty percent mortality occurred at day 450 and 100% at day 750. No tumors were observed in the bladder, liver, spleen or kidney, the only organs evaluated.

Syrian golden hamsters, 15 male and 15 female, received 52 weekly subcutaneous injections of 0 or 1.9 mmol/kg of aniline 1n peanut oil (Hecht et al., 1983). No increased incidence of tumors was observed. Mean survival was reduced in the aniline-treated groups.

II.A.4. Supporting Data for Carcinogenicity

Aniline produced generally negative results in reverse mutation assays using Salmonella typhimurium (Miyata et al., 1981; Simmon, 1979a; DeFlora, 1981; Parodi et al., 1981; McCann et al., 1975; Hecht et al., 1979; Haworth et al., 1983; Garner and Nutman, 1977). Nagao et al. (1977) and Sugimura et al. (1982) reported aniline to be mutagenic for S. typhimurium only

when assayed in the presence of the comutagen norharman. Aniline did not induce mitotic recombination in the presence or absence of metabolic activation in Saccharomyces cerevisiae (Simmon, 1979b). Aniline was reported to show positive results in the L5178+/- mouse lymphoma gene mutation assay (Amacher et al., 1980).

Aniline caused an increased frequency of SCE in vivo in mouse bone marrow cells (Parodi et al., 1982, 1983) and in two in vitro assays with Don cells (Abe and Sasaki, 1977) and lung fibroblasts (Kawachi et al., 1980). In another in vitro assay, only the two metabolites of aniline, 2-aminophenol and N-phenylhydroxylamine, increased the frequency of SCE in human fibroblasts (Wilmer et al., 1981).

Aniline transformed the mouse cell line Balb/3T3, but not Syrian hamster embryo cells or Fischer 344 rat embryo cells infected with murine leukemia virus (Dunkel et al., 1981). DNA damage assays in E. coli (Mamber et al., 1983) and B. subtilis (McCarroll et al., 1981) were negative.

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

II.B.1. Summary of Risk Estimates

Oral Slope Factor — 5.7E-3/mg/kg/day

Drinking Water Unit Risk — 1.6E-7/ug/L

Extrapolation Method — Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	6E+2 ug/L
E-5 (1 in 100,000)	6E+1 ug/L
E-6 (1 in 1,000,000)	6 ug/L

II.B.2. Dose-Response Data (Carcinogenicity, Oral Exposure)

Tumor Type — spleen, combined fibrosarcoma, stromal sarcoma, capsular sarcoma and hemangiosarcoma
Test animals — rat/CD-F, male
Route — diet
Reference — CIIT, 1982

Administered Dose (ppm)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0	0	0/64
200	1.23	0/90
600	3.69	1/90
2000	12.29	31/90

II.B.3. Additional Comments (Carcinogenicity, Oral Exposure)

Calculation of the transformed doses for aniline included a correction for the difference in molecular weights of aniline and aniline hydrochloride, the form in which the compound was administered in the NCI and CIIT bioassays.

Using data from the male rats (NCI, 1978), slope factors were derived for the incidence of hemangiosarcoma in the spleen and body cavity (2.6E-2/mg/kg/day), and individually for the combined incidence of fibrosarcoma and sarcoma in the spleen (1.0E-2/mg/kg/day), in the body cavity (6.2E-3/mg/kg/day), and in the spleen and body cavity combined (1.1E-2/mg/kg/day). Most of the sarcomas observed in the spleen and body cavity combined were in the spleen; as a consequence, the slope factors for sarcoma in the spleen and for the spleen and body cavity are very close.

The unit risk should not be used if the water concentration exceeds 6E+4 ug/L, since above this concentration the slope factor may differ from that stated.

II.B.4. Discussion of Confidence (Carcinogenicity, Oral Exposure)

An adequate number of animals were dosed for an adequate length of time. Slope factors derived from tumor data at other sites were with one exception within a factor of 2.

Studies in rats by Bus and Sun (1979) have indicated that erythrocytes preferentially bind aniline, and that at high doses (100 mg/kg), aniline selectively accumulates in the spleen due to scavenging of damaged erythrocytes. The deposition of debris from erythrocytes results in hemosiderosis, which in turn induces a fibrotic response in the spleen that may be involved in sarcoma production. Robertson et al. (1983) have shown that aniline accumulation in the spleen is nonlinear; only minimal accumulation of aniline and no hemosiderosis is observed at doses below 10 mg/kg. Since hemosiderosis may be important in the induction of splenic sarcoma, the linearized multistage procedure may not be the most appropriate method for the derivation of the slope factor.

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document — U.S. EPA, 1985

The 1985 Health and Environmental Effects Profile for Aniline has received Agency Review.

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review — 05/13/1987, 06/03/1987

Verification Date — 06/03/1987

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the cancer assessment for Aniline conducted in September 2002 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — Aniline
CASRN — 62-53-3

VI.A. Oral RfD References

None

VI.B. Inhalation RfD References

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VII. Revision History

Substance Name — Aniline
CASRN — 62-53-3

Date	Section	Description
09/07/1988	II.	Carcinogen summary on-line
11/01/1990	I.B.	Inhalation RfC summary on-line
12/03/2002	I.B.6., II.D.2.	Screening-Level Literature Review Findings message has been added.

VIII. Synonyms

Substance Name — Aniline
CASRN — 62-53-3
Last Revised — 09/07/1988

- 62-53-3
- aminobenzene
- aminophen
- Aniline
- aniline-oil
- kyanol
- phenylamine