Aroclor 1016; CASRN 12674-11-2

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 Aroclor 1016

File First On-Line 01/01/1993

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	yes	01/01/1993
Inhalation RfC (I.B.)	not evaluated	
Carcinogenicity Assessment (II.)	not evaluated	

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

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

Substance Name — Aroclor 1016
CASRN — 12674-11-2
Last Revised — 01/01/1993

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also 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.A.1. Oral RfD Summary

Critical Effect	Experimental Doses*	UF	MF	RfD
Reduced birth weights **Monkey Reproductive Bioassay** **Barsotti and van Miller, 1984; Levin et al., 1988; Schantz et al., 1989, 1991**	NOAEL: 0.25 ppm in feed (0.007 mg/kg-day) LOAEL: 1 ppm in feed (0.028 mg/kg-day)	100	1	7E-5 mg/kg-day

*Conversion Factors: Dams received a total average intake of 4.52 mg/kg (0.25 ppm) or 18.41 mg/kg (1 ppm) throughout the 21.8-month (654 days) dosing period. These doses are equivalent to 0.007 mg/kg-day and 0.028 mg/kg-day for the identified NOAEL and LOAEL respectively.

I.A.2. Principal and Supporting Studies (Oral RfD)

Barsotti, D.A. and J.P. van Miller. 1984. Accumulation of a commercial polychlorinated biphenyl mixture (Aroclor 1016) in adult rhesus monkeys and their nursing infants. Toxicology. 30: 31-44.

Levin, E.D., S.L. Schantz and R.E Bowman. 1988. Delayed spatial alternation deficits resulting from perinatal PCB exposure in monkeys. Arch. Toxicol. 62: 267-273.

Schantz, S.L., E.D. Levin, R.E. Bowman et al. 1989. Effects of perinatal PCB exposure on discrimination-reversal learning in monkeys. Neurotoxicol. Teratol. 11: 243-250.

Schantz, S.L., E.D. Levin and R.E. Bowman. 1991. Long-term neurobehavioral effects of perinatal polychlorinated biphenyl (PCB) exposure in monkeys. Environ. Toxicol. Chem. 10: 747-756.

These are a series of reports that evaluated perinatal toxicity and long- term neurobehavioral effects of Aroclor 1016 in the same groups of infant monkeys. Aroclor 1016 is a commercial mixture of polyclorinated biphenyls (PCBs) devoid of chlorinated dibenzofurans (Barsotti and van Miller, 1984). Analysis of the commercial feed used for this study revealed contamination with congeners specific for Aroclor 1248, present in the parts per billion range. These congeners were present in the control as well as test diets. Aroclor 1016 was administered to groups of 8 adult female rhesus monkeys via diet in concentrations of 0, 0.25 or 1.0 ppm for approximately 22 months. Based on a reported total Aroclor intake of 4.52 and 18.41 mg/kg over the 22- month exposure period (Schantz et al., 1989, 1991), the low- and high-doses are estimated to be 0.007 and 0.028 mg/kg-day, respectively. Exposure began 7 months prior to breeding and continued until offspring were weaned at age 4 months. No exposure-related effects on maternal food intake, general appearance, hematology, serum chemistry (SGPT, lipid, and cholesterol analyses) or number of breedings were observed (Barsotti and van Miller, 1984). All monkeys had uncomplicated pregnancies, carried their infants to term and delivered viable offspring. Teratologic examinations were not performed. Mean birth weights of the infants in the control, 0.007 and 0.028 mg/kg-day dose groups were 521 g, 491 g and 442 g, respectively (Barsotti and van Miller, 1984). The decrease in birth weight in the high-dose group was significantly ($p<0.01$) lower than in controls. Further statistical analysis of the infant birth weight data by the Agency indicated that gestation length did not significantly affect birth weight and the distribution of male and female infants in the various dose groups could not account for the difference in birth weights among the dose groups. Agency reanalysis of the data confirmed the significant decrease in body weight for the high-dose infants, although slightly different average values were obtained. Males that had sired some infants were exposed to Aroclor 1248, so the birth weight data were also analyzed excluding these infants. The results for this adjusted data indicated that control infants weighed 528 g, low-dose infants weighed 486 g, and high-dose infants weighed 421 g. Even with this adjustment there was still a significant difference ($p<0.01$) in birth weight for the high-dose group when compared with controls. No significant differences between treatment and control groups were detected in neonatal head circumference or crown-to-rump measurements. Both exposure groups showed consistent weight gains, but infant weights in the high-dose group were still lower (864 g) at weaning, although not significantly different from the controls (896 g). Hyperpigmentation was present at birth in the low- and high-dose infants but did not persist once dosing was stopped. This clinical change was determined not to be a critical adverse effect. The concentration of Aroclor 1016 in breast milk was higher than the maternal dose. No exposure-related hematologic effects were observed in the infants during the nursing period (Barsotti and van Miller, 1984). One of the offspring in the high-dose group went into shock and died on the day following weaning for unknown reasons (Schantz et al., 1989, 1991).

Behavioral testing of the infant monkeys was first performed at age 14 months and no overt signs of PCB toxicity were observed (Schantz et al., 1989, 1991). Two-choice discrimination-reversal learning was assessed using simple left-right spatial position, color and shape discrimination

problems, with and without irrelevant color and shape cues. One of the offspring in the low-dose group stopped responding early in testing for an unknown reason and could not be induced to resume; therefore, test results were obtained using 6, 7 and 6 infants in the control, low- and high-dose groups, respectively. The offspring in the high-dose (0.028 mg/kg-day) group were significantly ($p<0.05$) impaired in their ability to learn the spatial position discrimination problem (i.e., achieved 9 correct choices in 10 trials), requiring more than 2.5 times as many trials as their age-matched controls. There were no significant learning differences between these groups on this problem during overtraining (ability to achieve greater than or equal to 90% correct choices in two consecutive daily sessions) or position reversals. The only other exposure-related effect was significantly facilitated learning ability ($p<0.05$) on the shape discrimination problem at 0.028 mg/kg-day.

Performance on delayed spatial alternation (a spatial learning and memory task) was assessed in the offspring monkeys at age 4-6 years (Levin et al., 1988; Schantz et al., 1991). The two Aroclor-exposed groups were not significantly different from controls ($p<0.05$) in test performance. However, the exposed groups did significantly ($p<0.05$) differ from each other. The difference between the two exposed groups was due to a combination of facilitated performance at the low-dose (0.007 mg/kg-day) and impaired performance at the high-dose (0.028 mg/kg-day). Although these data are insufficient for establishing an exposure-effect relation due to the lack of difference between exposed and control groups, the investigators suggested that the performance deficit at 0.028 mg/kg-day may have been exposure- related. The investigators noticed that a paradoxical biphasic effect occurred on the same test when comparing low-dose and high-dose infants. This same effect has been observed for lead-exposed monkeys.

To summarize the above, adult monkeys that ingested 0.007 or 0.028 mg/kg- day doses of Aroclor 1016 for approximately 22 months showed no evidence of overt toxicity. Effects occurring in the offspring of these monkeys consisted of hairline hyperpigmentation at greater than or equal to 0.007 mg/kg-day, and decreased birth weight and possible neurologic impairment at 0.028 mg/kg-day. Based on the reduced birth weights of prenatally-exposed monkeys, the 0.007 mg/kg-day dose is the NOAEL and the 0.028 mg/kg-day dose is a LOAEL in monkeys.

The results of the neurobehavioral tests in the monkey offspring at 14 months and 4-6 years of age indicate adverse learning deficits at the 0.028 mg/kg-day maternal dose. Evaluation of these data is complicated by possible inconsistencies in the outcome of both the discrimination-reversal learning tests (learning was impaired and facilitated on different problems) and the delayed spatial alternation test (performance significantly differed between the two exposed groups, but not between either test group and the control). However, there is evidence suggesting that deficits in discrimination-reversal learning and delayed spatial alternation are related to

decreased brain dopamine (Schantz et al., 1991), which has been observed in monkeys orally exposed to Aroclor 1016 (Seegal et al., 1990, 1991). Behavioral dysfunctions, including deficits in visual recognition and short-term memory, also have been observed in infants of human mothers who consumed fish contaminated with PCB mixtures of unknown composition (Fein et al., 1984a,b; Jacobsen et al., 1985, 1990; Gladen et al., 1988).

I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF — A 3-fold factor is applied to account for sensitive individuals. The results of these studies, as well as data for human exposure to PCBs, indicate that infants exposed transplacentally represent a sensitive subpopulation. A factor of 3 is applied for extrapolation from rhesus monkeys to human. A full 10-fold factor for interspecies extrapolation is not considered necessary because of similarities in toxic responses and metabolism of PCBs between monkeys and humans and the general physiologic similarity between these species. In addition, the rhesus monkey data are predictive of other changes noted in human studies such as chloracne, hepatic changes, and effects on reproductive function. A factor of 3 is applied because of limitations in the database. Despite the extensive amount of animal laboratory data and human epidemiologic information regarding PCBs, the issue of male reproductive effects is not directly addressed and two-generation reproductive studies are not available. As the study duration was considered as somewhat greater than subchronic, but less than chronic, a partial factor of 3 is used to account for extrapolation from a subchronic exposure to a chronic RfD.

MF — None

I.A.4. Additional Studies/Comments (Oral RfD)

Male pig-tailed macaques [Macaca nemistrina], (number not reported, age 3- 7 years, 5-9 kg initial body weight) were administered Aroclor 1016 dissolved in corn oil on bread in doses of 0, 0.8, 1.6 or 3.2 mg/kg-day for 20 weeks (Seegal et al., 1991). There were no overt signs of intoxication or exposure- related effects on body weight gain. Neurochemical analyses of various regions of the brain were performed following termination of exposure. Dose- related decreased concentrations of dopamine were observed in the caudate nucleus, putamen, substantia nigra, and hypothalamus, but not in the globus pallidus or hippocampus. There were no exposure-related changes in concentrations of norepinephrine, epinephrine, or serotonin. Other neurologic endpoints were not evaluated.

Subchronic oral studies of Aroclor 1016 have been performed in species other than monkeys. These species were tested at doses higher than the 0.007 and 0.028 mg/kg-day doses fed to monkeys in the principal studies.

Groups of 10 female Sprague-Dawley rats (age not reported, body weight approximately 225-250 g at start) were fed 0, 1, 5 or 50 ppm Aroclor 1016 in the diet for 5 months (Byrne et al., 1988). The Aroclor was dissolved in acetone that was evaporated from the diet prior to feeding. Using a rat food consumption factor of 0.05 kg food/kg bw (U.S. EPA, 1987), the doses are estimated to be 0, 0.05, 0.25 and 2.5 mg/kg-day. Serum levels of adrenal cortical hormones were evaluated four times throughout the treatment period. Adrenal dehydroepiandrosterone (DHEA) and dehydroepiandrosterone sulfate (DHS) levels were significantly ($p<0.05$) reduced at all treatment levels after approximately 100 days of exposure. Serum corticosterone (the principal glucocorticoid in rats), adrenal weight, adrenal histology, and nonadrenal endpoints other than food consumption were not evaluated. Food consumption did not significantly differ between and among control and treatment groups. Because insufficient information is available to determine whether the decreases in circulating adrenal hormones were physiologically significant, it is uncertain whether the doses are NOAELs or LOAELs for Aroclor 1016 in rats.

Male Balb/c mice (18-20 g body weight) were fed Aroclor 1016 mixed in diet at concentrations of 0 or 5 ppm for 3 or 6 weeks (Loose et al., 1978). Using a mouse food consumption factor of 0.13 kg food/kg bw (U.S. EPA, 1987), the dose is estimated to be 0.65 mg/kg-day. Sensitivity to Salmonella typhosa endotoxin (15 mice per endotoxin dose) and resistance to infection by Plasmodium berghei (malaria parasitemia; number of mice not reported) were evaluated. Sensitivity to the endotoxin was significantly ($p<0.05$) increased after 3 weeks of exposure as indicated by endotoxin LD50 values of 152 and 844 ug in the Aroclor-exposed and control groups, respectively. Sensitivity to the endotoxin after 6 weeks of Aroclor exposure was not evaluated. There were no significant ($p<0.05$) effects of Aroclor exposure for 3 or 6 weeks on malaria lethality as indicated by post-inoculation survival time. No other endpoints were evaluated in this study. When injected into neonates, splenic cells from C57Bl/6 male mice exposed to 167 ppm (21.71 mg/kg-day) dietary Aroclor 1016 for 3 weeks elicited a greater graft-versus-host reaction than controls (Silkworth and Loose, 1978). Based on the decreased resistance to infection leading to death, 0.65 mg Aroclor 1016/kg-day suggests a LOAEL for immunotoxicity for subchronic exposure in male mice.

Aulerich and Ringer (1977) performed a breeding study in which groups of 8 female and 2 male adult pastel mink were fed diets containing 0 or 2 ppm Aroclor 1016 for 39 weeks or until the kits were 4 weeks of age. The Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Using assumed values of 150 g/day for food consumption and 0.8 kg for body weight for female mink (Bleavins et al., 1980), the estimated dose of Aroclor 1016 is 0.4 mg/kg-day. Monthly determinations showed no statistically significant differences ($p<0.05$) between the control and treated mink in body weight gain, hemoglobin, and hematocrit. Additionally, tabulated data showed no treatment-related effects on female survival, numbers of females mated, number of females that gave birth, number of kits born alive or dead, number of births per female, average birth weight or number of kits alive at 4 weeks. The evidence for lack

of treatment-related effects on body weight, hematology, reproduction and survival suggests that 0.4 mg/kg-day is a NOAEL for Aroclor 1016 in mink.

Groups of adult Pastel mink were fed a diet containing 0 ppm (24 females and 6 males) or 20 ppm (12 females and 3 males) Aroclor 1016 during a 247-day breeding study (Bleavins et al., 1980). Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Using assumed values of 150 g/day for food consumption and 0.8 kg for body weight for female mink reported by the investigators, the estimated dose of Aroclor 1016 is 3.8 mg/kg-day. There were no deaths in the exposed or control males. Mortality was higher in the exposed females [25% (3/12) compared with 12.5% (3/24) in controls], but no clear difference in survival time was observed. Necropsies for gross abnormalities were performed on all control and treated mink that died; these showed effects only in the treated mink consisting of emaciation characterized by an almost complete absence of body fat. Histologic examinations were not performed. The incidence of mated females giving birth was reduced in the exposed group [44.4% (4/9) compared with 76.2% (16/21) in controls], but average gestation length, live births and birth weight did not significantly differ ($p>0.05$) between exposed and control groups. Body weight at age 4 weeks, average number of infants per lactating female and infant biomass (average body weight gain through age four weeks x average number of infants raised per lactating female) were significantly ($p<0.05$) reduced in the exposed group. Mortality during the first 4 weeks of life was increased in the exposed group [56.0% (14/25) compared with 24.1% (19/79) in controls]. The investigators noted that the adverse effects on reproduction do not appear to be due to an effect on spermatogenesis, since PCB-treated male mink have had acceptable levels of reproduction when mated to untreated females in other studies. The evidence for impaired reproduction and increased maternal and postnatal mortality suggests that 3.8 mg Aroclor 1016/kg-day is an FEL in mink. Although the FEL from this study and NOEL of 0.4 mg/kg-day from Aulerich and Ringer (1977) suggest that the dose-severity slope for Aroclor 1016 in mink is steep, neither study tested sufficient numbers of animals or dose levels to allow definitive conclusions to be drawn.

Dermal lesions including skin irritation, chloracne and increased pigmentation of skin and nails have been observed in humans occupationally exposed to Aroclor 1016 and other Aroclor formulations by both inhalation and dermal routes (Fischbein et al., 1979, 1982, 1985; Ouw et al., 1976; Smith et al., 1982). However, insufficient data are available to determine possible contributions of Aroclor 1016 alone, extent of direct skin exposure and possible contaminants in these occupational studies.

Decreased birth weight has also been reported in infants born to women who were occupationally exposed to Aroclor 1016 and other Aroclor formulations (Taylor et al., 1984, 1989), ingested PCB-contaminated fish (Fein et al., 1984a,b) and ingested heated Kanechlor PCBs during the Yusho and Yu-Cheng incidents (Rogan, 1989; Yamashita, 1977). Due to

uncertainties regarding actual sources of PCB exposure, and other confounding factors and study limitations, the decreases in human birth weight cannot be solely attributed to PCBs, particularly specific PCB mixtures. However, due to the consistency with which the effect has been observed, the human data are consistent with the Aroclor 1016-induced decreased birth weight in monkeys reported in the principal studies.

The human data available for risk assessment of Aroclor 1016 are useful only in a qualitative manner. Studies of the general population exposed to PCBs by consumption of contaminated food, particularly neurobehavioral evaluations of infants exposed in utero and/or through lactation, have been reported, but the original PCB mixtures, exposure levels and other details of exposure are not known (Kreiss et al., 1981; Humphrey, 1983; Fein et al., 1984a,b; Jacobson et al., 1984a, 1985, 1990a,b; Rogan et al., 1986; Gladen et al., 1988). Most of the information on health effects of PCB mixtures in humans is available from studies of occupational exposure. Some of these studies examined workers who had some occupational exposure to Aroclor 1016, but in these studies concurrent exposure to other Aroclor mixtures nearly always occurred, exposure involved dermal as well as inhalation routes (the relative contribution by each route was not known), and monitoring data were lacking or inadequate (Fischbein et al., 1979, 1982, 1985; Fischbein, 1985; Warshaw et al., 1979; Smith et al., 1982; Lawton et al., 1985).

Information specifically on the oral absorption of Aroclor 1016 is not available, but studies of individual congeners and PCB mixtures of higher chlorine content in animals indicate, in general, that PCBs are readily and extensively absorbed. These studies have found oral absorption efficiency on the order of 75 to >90% in rats, mice, monkeys and ferrets (Albro and Fishbein, 1972; Allen et al., 1974; Tanabe et al., 1981; Bleavins et al., 1984; Clevenger et al., 1989). A study of a PCB mixture containing 54% chlorine provides direct evidence of absorption of PCBs in humans after oral exposure (Buhler et al., 1988), and indirect evidence of oral absorption of PCBs by humans is available from studies of ingestion of contaminated fish by the general population (Schwartz et al., 1983; Kuwabara et al., 1979). There are no quantitative data regarding inhalation absorption of PCBs in humans but studies of exposed workers suggest that PCBs are well absorbed by the inhalation and dermal routes (Maroni et al., 1981a,b; Smith et al., 1982; Wolff, 1985). PCBs distribute preferentially to adipose tissue and concentrate in human breast milk due to its high fat content (Jacobson et al., 1984b; Ando et al., 1985).

The metabolism of PCBs following oral and parenteral administration in animals has been extensively studied and reviewed, but studies in animals following inhalation or dermal exposure are lacking (Sundstrom and Hutzinger, 1976; Safe, 1980; Sipes and Schnellmann, 1987). Information on metabolism of PCBs in humans is limited to occupationally exposed individuals whose intake is derived mainly from inhalation and dermal exposure (Jensen and Sundstrom, 1974; Wolff et al., 1982; Schnellmann et al., 1983; Safe et al., 1985; Fait et al., 1989). In general, metabolism of PCBs depends on the number and position of the chlorine atoms on the phenyl

rings of the constituent congeners (i.e., congener profile of the PCB mixture) and animal species. Although only limited data are available on metabolism of PCBs following inhalation exposure, there is no reason to suspect that PCBs are metabolized differently by this route.

Data exist on the in vitro hepatic metabolism and in vivo metabolic clearance of 2,2',3,3',6,6'-hexachlorobiphenyl and 4,4'-dichlorobiphenyl congeners in humans, monkeys, dogs, and rats (Schnellmann et al., 1985). Both of these congeners are present in Aroclor 1016, but the hexachlorobiphenyl is only a minor constituent. For each congener, the Vmax values for metabolism in the monkey, dog and rat are consistent with the respective metabolic clearance values found in vivo. Thus, the kinetic constants for PCB metabolism obtained from the dog, monkey, and rat hepatic microsomal preparations were good predictors of in vivo metabolism and clearance for these congeners. In investigations directed at determining which species most accurately predicts the metabolism and disposition of PCBs in humans, the in vitro metabolism of these congeners was also studied using human liver microsomes (Schnellmann et al., 1983, 1984). Available data suggest that metabolism of PCBs in humans most closely resembles that of the monkey and rat. For example, the in vitro apparent Km and Vmax for humans and monkeys are comparable. These studies show consistency between the in vitro and in vivo findings and collectively indicate that metabolism of the two congeners is similar in monkeys and humans.

I.A.5. Confidence in the Oral RfD

Study — Medium
Database — Medium
RfD — Medium

Confidence in the critical studies is rated medium since essentially only one group of monkeys has been examined. The initial study was well conducted in a sensitive animal species (rhesus monkeys) that closely resembles humans for many biological functions. These studies evaluated many sensitive endpoints of PCB toxicity and the effects observed have also been documented for human exposure. Many sophisticated reproductive and neurologic tests were performed over 6 years and many clinical chemistry determinations were conducted on the dams during the exposure period. Very extensive analyses of feed samples and tissue samples from dosed monkeys were performed. Although contamination of the control laboratory primate diet with PCBs other than those found in Aroclor 1016 was detected, the level of contamination was at the level of parts per billion and dosing of Aroclor 1016 was in the parts per million range. Because the contamination was consistent across all treatment groups and controls, quantitative comparison of adverse effects can be made. The investigators carefully documented the levels of test material and contaminant throughout the exposure and post-exposure period in animal tissues. Because the system of placentation, hemothelial-chorial with bidiscoidal distribution, is similar for Rhesus monkeys and humans, it is felt that toxic events that are induced during

gestation for Rhesus monkeys will be highly predictive of similar events in humans. Historically, developmental neurobehavioral effects observed in rhesus monkeys are predictive of similar effects in humans. Although these studies were performed in an academic setting prior to the era of Good Laboratory Practices- Quality Control-Quality Assurance, the study report provides ample documentation of the experimental protocol and quality of data collected. While the group sizes for this study are small (8 monkeys/group) when compared with the standards for rodent studies they are within the acceptable range for studies of large mammalian species as determined by EPA.

The database for PCBs in general is extensive. Studies examining Aroclor 1016 have been performed in rhesus monkeys, mice, rats and mink. However, despite the extensive amount of data available only medium confidence can be placed in the database at this time. It is acknowledged that mixtures of PCBs found in the environment do not match the pattern of congeners found in Aroclor 1016, therefore the RfD is only given medium confidence. For those particular environmental applications where it is known that Aroclor 1016 is the only form of PCB contamination, use of this reference dose may rate high confidence. For all other applications only medium confidence can be given. The U.S. EPA recognizes that there is a diversity of opinion among scientists concerning the use of the monkey studies for determining PCB toxicity. However, all of the studies in the vast database for this chemical mixture support the conclusions reached in this document.

I.A.6. EPA Documentation and Review of the Oral RfD

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

Other EPA Documentation — U.S. EPA, 1980, 1984, 1989, 1990

Agency Work Group Review — 02/21/1990, 03/25/1992, 06/23/1992, 09/24/1992, 10/15/1992, 11/04/1992, 02/11/1993

Verification Date — 11/04/1992

I.A.7. EPA Contacts (Oral RfD)

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).

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

Substance Name — Aroclor 1016
CASRN — 12674-11-2

Not available at this time.

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Aroclor 1016
CASRN — 12674-11-2

This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.

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

VI. Bibliography

Substance Name — Aroclor 1016
CASRN — 12674-11-2

VI.A. Oral RfD References

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VI.B. Inhalation RfC References

None

VI.C. Carcinogenicity Assessment References

None

VII. Revision History

Substance Name — Aroclor 1016
CASRN — 12674-11-2

Date	Section	Description
01/01/1993	I.A.	Oral RfD assessment on-line

VIII. Synonyms

Substance Name — Aroclor 1016
CASRN — 12674-11-2
Last Revised — 01/01/1993

- 12674-11-2
- AROCLOR 1016
- HSDB 6352