

Provisional Peer-Reviewed Toxicity Values for  
  
Aroclor 5460  
(CASRN 11126-42-4)

Superfund Health Risk Technical Support Center  
National Center for Environmental Assessment  
Office of Research and Development  
U.S. Environmental Protection Agency  
Cincinnati, OH 45268

## **AUTHORS, CONTRIBUTORS, AND REVIEWERS**

### **CHEMICAL MANAGERS**

Evisabel A. Craig, PhD  
National Center for Environmental Assessment, Cincinnati, OH

Q. Jay Zhao, PhD, DABT  
National Center for Environmental Assessment, Cincinnati, OH

### **PRIMARY INTERNAL REVIEWERS**

Paul G. Reinhart, PhD, DABT  
National Center for Environmental Assessment, Research Triangle Park, NC

Ambuja Bale, PhD, DABT  
National Center for Environmental Assessment, Washington, DC

Questions regarding the contents of this document may be directed to the U.S. EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300).

## TABLE OF CONTENTS

COMMONLY USED ABBREVIATIONS AND ACRONYMS.....	iii
PROVISIONAL PEER-REVIEWED TOXICITY VALUES FOR AROCLOR 5460 (CASRN 11126-42-4).....	1
BACKGROUND .....	1
DISCLAIMERS .....	1
QUESTIONS REGARDING PPRTVs.....	1
INTRODUCTION .....	2
REVIEW OF POTENTIALLY RELEVANT DATA (CANCER AND NONCANCER).....	5
Human Studies.....	8
Oral Exposures.....	8
Inhalation Exposures.....	8
Animal Studies.....	8
Oral Exposures.....	8
Other Data (Short-Term Tests, Other Examinations).....	10
Developmental Studies in Chickens .....	13
Short-term-Duration Studies.....	14
Metabolism/Toxicokinetic Studies .....	14
Mode of Action/Mechanistic Studies.....	15
DERIVATION OF PROVISIONAL VALUES .....	15
Derivation of Oral Reference Doses .....	17
Derivation of Inhalation Reference Concentrations .....	17
CANCER WEIGHT-OF-EVIDENCE (WOE) DESCRIPTOR.....	17
Derivation of Provisional Cancer Potency Values.....	18
APPENDIX A. PROVISIONAL SCREENING VALUES.....	19
APPENDIX B. DATA TABLES.....	22
APPENDIX C. BMD OUTPUTS .....	29
APPENDIX D. REFERENCES.....	30

## COMMONLY USED ABBREVIATIONS AND ACRONYMS

$\alpha$ 2u-g	$\alpha$ 2u-globulin	LOAEL	lowest-observed-adverse-effect level
ACGIH	American Conference of Governmental Industrial Hygienists	MN	micronuclei
AIC	Akaike's information criterion	MNPCE	micronucleated polychromatic erythrocyte
ALD	approximate lethal dosage	MTD	maximum tolerated dose
ALT	alanine aminotransferase	NAG	N-acetyl- $\beta$ -D-glucosaminidase
AST	aspartate aminotransferase	NCEA	National Center for Environmental Assessment
atm	atmosphere	NCI	National Cancer Institute
ATSDR	Agency for Toxic Substances and Disease Registry	NOAEL	no-observed-adverse-effect level
BMD	benchmark dose	NTP	National Toxicology Program
BMDL	benchmark dose lower confidence limit	NZW	New Zealand White (rabbit breed)
BMDS	Benchmark Dose Software	OCT	ornithine carbamoyl transferase
BMR	benchmark response	ORD	Office of Research and Development
BUN	blood urea nitrogen	PBPK	physiologically based pharmacokinetic
BW	body weight	PCNA	proliferating cell nuclear antigen
CA	chromosomal aberration	PND	postnatal day
CAS	Chemical Abstracts Service	POD	point of departure
CASRN	Chemical Abstracts Service Registry Number	POD <sub>[ADJ]</sub>	duration-adjusted POD
CBI	covalent binding index	QSAR	quantitative structure-activity relationship
CHO	Chinese hamster ovary (cell line cells)	RBC	red blood cell
CL	confidence limit	RDS	replicative DNA synthesis
CNS	central nervous system	RfC	inhalation reference concentration
CPN	chronic progressive nephropathy	RfD	oral reference dose
CYP450	cytochrome P450	RGDR	regional gas dose ratio
DAF	dosimetric adjustment factor	RNA	ribonucleic acid
DEN	diethylnitrosamine	SAR	structure activity relationship
DMSO	dimethylsulfoxide	SCE	sister chromatid exchange
DNA	deoxyribonucleic acid	SD	standard deviation
EPA	Environmental Protection Agency	SDH	sorbitol dehydrogenase
FDA	Food and Drug Administration	SE	standard error
FEV1	forced expiratory volume of 1 second	SGOT	glutamic oxaloacetic transaminase, also known as AST
GD	gestation day	SGPT	glutamic pyruvic transaminase, also known as ALT
GDH	glutamate dehydrogenase	SSD	systemic scleroderma
GGT	$\gamma$ -glutamyl transferase	TCA	trichloroacetic acid
GSH	glutathione	TCE	trichloroethylene
GST	glutathione-S-transferase	TWA	time-weighted average
Hb/g-A	animal blood:gas partition coefficient	UF	uncertainty factor
Hb/g-H	human blood:gas partition coefficient	UF <sub>A</sub>	interspecies uncertainty factor
HEC	human equivalent concentration	UF <sub>H</sub>	intraspecies uncertainty factor
HED	human equivalent dose	UF <sub>S</sub>	subchronic-to-chronic uncertainty factor
i.p.	intraperitoneal	UF <sub>D</sub>	database uncertainty factor
IRIS	Integrated Risk Information System	U.S.	United States of America
IVF	in vitro fertilization	WBC	white blood cell
LC50	median lethal concentration		
LD50	median lethal dose		

## PROVISIONAL PEER-REVIEWED TOXICITY VALUES FOR AROCLOR 5460 (CASRN 11126-42-4)

### BACKGROUND

A Provisional Peer-Reviewed Toxicity Value (PPRTV) is defined as a toxicity value derived for use in the Superfund Program. PPRTVs are derived after a review of the relevant scientific literature using established Agency guidance on human health toxicity value derivations. All PPRTV assessments receive internal review by a standing panel of National Center for Environment Assessment (NCEA) scientists and an independent external peer review by three scientific experts.

The purpose of this document is to provide support for the hazard and dose-response assessment pertaining to chronic and subchronic exposures to substances of concern, to present the major conclusions reached in the hazard identification and derivation of the PPRTVs, and to characterize the overall confidence in these conclusions and toxicity values. It is not intended to be a comprehensive treatise on the chemical or toxicological nature of this substance.

The PPRTV review process provides needed toxicity values in a quick turnaround timeframe while maintaining scientific quality. PPRTV assessments are updated approximately on a 5-year cycle for new data or methodologies that might impact the toxicity values or characterization of potential for adverse human health effects and are revised as appropriate. It is important to utilize the PPRTV database (<http://hhpprtv.ornl.gov>) to obtain the current information available. When a final Integrated Risk Information System (IRIS) assessment is made publicly available on the Internet ([www.epa.gov/iris](http://www.epa.gov/iris)), the respective PPRTVs are removed from the database.

### DISCLAIMERS

The PPRTV document provides toxicity values and information about the adverse effects of the chemical and the evidence on which the value is based, including the strengths and limitations of the data. All users are advised to review the information provided in this document to ensure that the PPRTV used is appropriate for the types of exposures and circumstances at the site in question and the risk management decision that would be supported by the risk assessment.

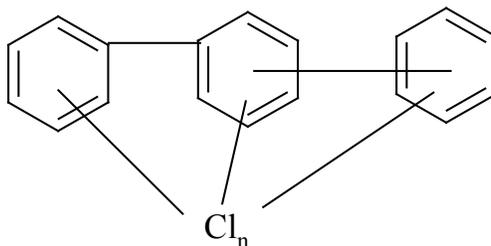
Other U.S. Environmental Protection Agency (EPA) programs or external parties who may choose to use PPRTVs are advised that Superfund resources will not generally be used to respond to challenges, if any, of PPRTVs used in a context outside of the Superfund program.

### QUESTIONS REGARDING PPRTVs

Questions regarding the contents and appropriate use of this PPRTV assessment should be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300).

## INTRODUCTION

Aroclor 5460 (CASRN 11126-42-4) belongs to a class of chlorinated aromatic compounds known as polychlorinated terphenyls (PCTs), which are structurally and chemically similar to polychlorinated biphenyls (PCBs). Currently, the production and use of PCTs are banned in the United States and Canada. PCTs are also severely restricted in 12 European countries where only preparations with a PCT content <0.01% by weight are permitted ([FAO, 1992](#)). However, between 1959 and 1972, the Monsanto Company was a major producer of millions of pounds of various PCT blends in the United States, including the ones termed “pure” PCT aroclors (aroclor 5460, aroclor 5442, and aroclor 5432). Among these, aroclor 5460 was the predominant PCT produced ([Jensen and Jørgensen, 1983](#)). Aroclor 5460 was manufactured for use as a paint additive, sealant, wax, hot-melt adhesive, fire retardant, plasticizer, hydraulic fluid, and lubricant. Aroclor 5460 is a mixture of polychlorinated terphenyl molecules in multiple configurations (*ortho*-, *meta*-, or *para*-) with 60% chlorination by weight in contrast to aroclor 5442 with 42% chlorination. The chemical formula of aroclor 5460 can be given as  $C_{18}H_{14-n}Cl_n$ , in which “n” is the number of chlorine atoms that can range from 1–14. The general chemical structure of aroclor 5460 is presented in Figure 1. A table of physicochemical properties for aroclor 5460 is provided below (see Table 1).



**Figure 1. General Chemical Structure of Aroclor 5460**

<b>Table 1. Physicochemical Properties of Aroclor 5460 (CASRN 11126-42-4)<sup>a</sup></b>	
<b>Property (unit)</b>	<b>Value</b>
Distillation range (ASTM D-20 [Mod.] Corr.)	280–335°C at 5 mm Hg
Pour point (ASTM D-97)	46°C
Specific gravity (25°C)	1.470
Vaporization rate at 100°C and 760 mm Hg	0.000004 g/cm <sup>2</sup> /hr
Acidity-maximum (mg KOH/g)	0.05
Solubility in water (mg/L at 25°C)	Insoluble
Relative vapor density (air = 1)	ND; heavier than air
Molecular weight (g/mol)	ND; dependent on mixture composition

<sup>a</sup>Source: [Monsanto \(1960\)](#). Aroclor 5460 is a stable, nonflammable, yellow- to amber-colored resin that is insoluble in water and in low molecular weight alcohols. It is also nonoxidizing, noncorrosive, thermoplastic, and of low volatility.

ND = no data

A summary of available toxicity values for aroclor 5460 from EPA and other agencies/organizations is provided in Table 2.

**Table 2. Summary of Available Toxicity Values for Aroclor 5460 (CASRN 11126-42-4)<sup>a</sup>**

Source/Parameter <sup>a,b</sup>	Value (Applicability) <sup>c</sup>	Notes <sup>d</sup>	Reference	Date Accessed
<b>Noncancer</b>				
ACGIH	NV	NA	<a href="#">ACGIH (2013)</a>	NA
ATSDR	NV	NA	<a href="#">ATSDR (2013)</a>	NA
Cal/EPA	NV	NA	<a href="#">Cal/EPA (2013a, b)</a>	9-11-2013 <sup>e</sup>
NIOSH	NV	NA	<a href="#">NIOSH (2010)</a>	NA
OSHA	NV	NA	<a href="#">OSHA (2011, 2006)</a>	NA
IRIS/RfD, RfC	NV	NA	<a href="#">U.S. EPA</a>	9-11-2013
Drinking Water	NV	NA	<a href="#">U.S. EPA (2012a)</a>	NA
HEAST/RfD	NV	NA	<a href="#">U.S. EPA (2011a)</a>	NA
CARA HEHP	NV	NA	<a href="#">U.S. EPA (1994b)</a>	NA
WHO	NV	NA	<a href="#">WHO</a>	9-11-2013
<b>Cancer</b>				
IRIS/WOE, OSF	NV	NR	<a href="#">U.S. EPA</a>	9-11-2013
HEAST	NV	NR	<a href="#">U.S. EPA (2011a)</a>	NA
IARC	NV	NR	<a href="#">IARC (2013)</a>	NA
NTP	NV	NR	<a href="#">NTP (2011)</a>	NA
Cal/EPA	NV	NR	<a href="#">Cal/EPA (2013b, 2009)</a>	NA

<sup>a</sup>Sources: American Conference of Governmental Industrial Hygienists (ACGIH); Agency for Toxic Substances and Disease Registry (ATSDR); California Environmental Protection Agency (Cal/EPA); National Institute for Occupational Safety and Health (NIOSH); Occupational Safety and Health Administration (OSHA); Integrated Risk Information System (IRIS); Health Effects Assessment Summary Tables (HEAST); Chemical Assessments and Related Activities (CARA); Health and Environmental Effects Profile (HEEP); World Health Organization (WHO); International Agency for Research on Cancer (IARC); National Toxicology Program (NTP).

<sup>b</sup>Parameters: weight of evidence (WOE); reference dose (RfD); reference concentration (RfC); oral slope factor (OSF).

<sup>c</sup>NV = not available.

<sup>d</sup>NA = not applicable; NR = not relevant.

<sup>e</sup>The Cal/EPA Office of Environmental Health Hazard Assessment (OEHHA) Toxicity Criteria Database (<http://oehha.ca.gov/tcdb/index.asp>) was also reviewed and found to contain no information on aroclor 5460.

Literature searches were conducted on sources published from 1900 through September 2013 for studies relevant to the derivation of provisional toxicity values for aroclor 5460, using CASRN 11126-42-4. The following databases were searched by chemical name, synonyms, or CASRN: ACGIH, ANEUP, ATSDR, BIOSIS, Cal EPA, CCRIS, CDAT, ChemIDplus, CIS, CRISP, DART, EMIC, EPIDEM, ETICBACK, FEDRIP, GENE-TOX,

HAPAB, HERO, HMTc, HSDB, IARC, INCHEM IPCS, IPA, ITER, IUCLID, LactMed, NIOSH, NTIS, NTP, OSHA, OPP/RED, PESTAB, PPBIB, PPRTV, PubMed (toxicology subset), RISKLINE, RTECS, TOXLINE, TRI, U.S. EPA IRIS, U.S. EPA HEAST, U.S. EPA HEEP, U.S. EPA OW, and U.S. EPA TSCATS/TSCATS2. The following databases were searched for relevant health information: ACGIH, ATSDR, Cal EPA, U.S. EPA IRIS, U.S. EPA HEAST, U.S. EPA HEEP, U.S. EPA OW, U.S. EPA TSCATS/TSCATS2, NIOSH, NTP, OSHA, and RTECS.

### **REVIEW OF POTENTIALLY RELEVANT DATA (CANCER AND NONCANCER)**

Table 3 provides an overview of the relevant database for aroclor 5460 and includes all potentially relevant repeated-dose short-term-, subchronic-, and chronic-duration studies via the oral and inhalation routes. The phrase “statistical significance,” used throughout the document, indicates a *p*-value of <0.05.

**Table 3. Summary of Potentially Relevant Data for Aroclor 5460 (CASRN 11126-42-4)**

Category	Number of Male/Female, Strain, Species, Study Type, Study Duration, (Concentration)	Dosimetry <sup>a</sup>	Critical Effects	NOAEL <sup>a</sup>	BMDL/ BMCL <sup>a</sup>	LOAEL <sup>a</sup>	Reference (Comments)	Notes <sup>b</sup>
<b>Human</b>								
<b>1. Oral (mg/kg-d)<sup>a</sup></b>								
Acute <sup>c</sup>	ND							
Short-term <sup>d</sup>	ND							
Long-term <sup>c</sup>	ND							
Chronic <sup>f</sup>	ND							
<b>2. Inhalation (mg/m<sup>3</sup>)<sup>a</sup></b>								
Acute <sup>c</sup>	ND							
Short-term <sup>d</sup>	ND							
Long-term <sup>c</sup>	ND							
Chronic <sup>f</sup>	ND							
<b>Animal</b>								
<b>1. Oral (mg/kg-d)<sup>a</sup></b>								
Subchronic	10/10, Albino rat, diet, 7 d/wk, 95 d, (0; 100; 300; and 1,000 ppm)	M: 0, 7.13, 21.6, 65.6 (ADD)  HED: 0, 1.71, 5.18, 15.7  F: 8.06, 23.5, 77.8 (ADD)  HED: 0, 1.93, 5.64, 18.7	Increased absolute and relative liver weights	M: 7.13 (ADD)  F: 23.5 (ADD)	NC	M: 21.6 (ADD)  F: 77.8 (ADD)	<a href="#">Industrial Bio-Test Laboratories (1983b)</a>	NPR, PS

**Table 3. Summary of Potentially Relevant Data for Aroclor 5460 (CASRN 11126-42-4)**

Category	Number of Male/Female, Strain, Species, Study Type, Study Duration, (Concentration)	Dosimetry <sup>a</sup>	Critical Effects	NOAEL <sup>a</sup>	BMDL/ BMCL <sup>a</sup>	LOAEL <sup>a</sup>	Reference (Comments)	Notes <sup>b</sup>
Subchronic	6/0 (4 controls), Rhesus, monkey, diet, 7 d/wk, 12 wk, (0 and 5,000 ppm)	0, 690 (ADD) HED: 0, 311	Decreased body weight, alopecia, liver hypertrophy, swollen eyelids and lips, progressive generalized subcutaneous edema, purulent discharge from the eyes, acne-like lesions, and hypertrophy, hyperplasia, and dysplasia of the gastric mucosa	NI	NC	690 (ADD)	<a href="#">Allen and Norback (1973)</a>	PR
Developmental	ND							
Reproductive	ND							
Carcinogenicity	ND							
<b>2. Inhalation (mg/m<sup>3</sup>)<sup>a</sup></b>								
Subchronic	ND							
Chronic	ND							
Developmental	ND							
Reproductive	ND							
Carcinogenicity	ND							

<sup>a</sup>Dosimetry: NOAEL and LOAEL values are presented as an adjusted daily dose (ADD in mg/kg-d) and a human equivalent dose [HED in mg/kg-d, as calculated according to [U.S. EPA \(2011b\)](#) *Recommended Use of Body Weight<sup>3/4</sup> as the Default Method in Derivation of the Oral Reference Dose*] for oral noncancer effects. All long-term exposure values (4 wk and longer) are converted from a discontinuous to a continuous (daily) exposure.

ADD = adjusted daily dose; ADD = dose in ppm × (daily food consumption ÷ body weight).

HED = human equivalent dose; HED = animal dose (mg/kg-d) × (BW<sub>a</sub> ÷ BW<sub>h</sub>)<sup>1/4</sup>.

<sup>b</sup>Notes: PS = principal study; PR = peer reviewed; NPR = not peer reviewed; NA = not applicable.

<sup>c</sup>Acute = exposure for ≤24 hr ([U.S. EPA, 2002](#)).

<sup>d</sup>Short-term = repeated exposure for >24 hr ≤ 30 d ([U.S. EPA, 2002](#)).

<sup>e</sup>Long-term = repeated exposure for >30 d ≤ 10% lifespan (based on 70-yr typical lifespan) ([U.S. EPA, 2002](#)).

<sup>f</sup>Chronic = repeated exposure for >10% lifespan ([U.S. EPA, 2002](#)).

NC = not calculated; ND = no data; NI = not identified.

## HUMAN STUDIES

### Oral Exposures

No studies were identified.

### Inhalation Exposures

No studies were identified.

## ANIMAL STUDIES

### Oral Exposures

The effects of oral exposure of animals to aroclor 5460 were evaluated in two subchronic-duration studies ([Industrial Bio-Test Laboratories, 1983b](#); [Allen and Norback, 1973](#)).

#### *Subchronic-Duration Studies*

##### *[Industrial Bio-Test Laboratories \(1983b\)](#)*

In the subchronic-duration toxicity study performed for the Monsanto Company by [Industrial Bio-Test Laboratories \(1983b\)](#), 10 Albino rats/sex/dose were exposed to aroclor 5460 in the diet continuously for 95 days. There is an inconsistency in the study report in which the study authors indicated that the animals were sacrificed after 90 days (page 4 of the report); however, body-weight data were provided through 95 days (page 7 of the report). Therefore, the study is considered to be 95 days in duration. The animals were treated with aroclor 5460 (purity not reported) at concentrations of 0; 100; 300; or 1,000 ppm. Adjusted daily doses are calculated for this PPRTV assessment based on body weights and food intake rates reported by the authors. Adjusted daily doses<sup>1</sup> were 0, 7.13, 21.6, and 65.6 mg/kg-day for males and 0, 8.06, 23.5, and 77.8 mg/kg-day for females. Animals were housed individually in steel, wire-bottomed cages. Pulverized Wayne Lab-Blox rat ration (Allied Mills, Chicago, IL) was provided ad libitum for most of the study. For the last 5 weeks of feeding, pulverized Purina Labena Chow (Ralston-Purina Co., St. Louis, MO) was provided. Dose formulations were prepared by blending appropriately weighed amounts of aroclor 5460 with a preweighed portion of the stock diet. No further information was reported concerning the animals, husbandry, and dosing. Body weights were determined prior to treatment, weekly during treatment, and at necropsy. Overall body-weight gain (Days 0–95) was calculated for each dose group and statistically analyzed. Food consumption was measured weekly. Clinical observations on mortality and abnormal behavior were made daily. Hematology, clinical chemistry, and urinalysis were conducted prior to the beginning of treatment, on treatment Day 34, and prior to the conclusion of treatment on Day 89 on five rats/sex from the control and 1,000-ppm groups. Hematological analyses included hemoglobin concentration, erythrocyte count, hematocrit value, and total and differential leukocyte counts. Clinical chemistry was limited to determining blood urea nitrogen concentrations and serum alkaline phosphatase activity. Urinalyses included determination of glucose, albumin, microscopic elements, urinary pH, and specific gravity. At the scheduled termination, all animals were necropsied, and selected tissues (not specifically listed by the study authors) were excised, processed, and examined microscopically. The liver, kidney, spleen, gonads, heart, and brain were weighed. This study was not subjected to peer review and was performed before EPA Good-Laboratory-Practice (GLP) guidelines became effective in 1984.

---

<sup>1</sup>Adjusted daily dose = dose in ppm × (daily food consumption ÷ body weight). For 100 ppm in male rats: Adjusted daily dose = 100 ppm × (0.0261 mg/day ÷ 0.366 kg) = 7.13 mg/kg-day.

No treatment-related effects were noted on mortality or behavior. Also, there were no effects on hematology, clinical chemistry, or urinalysis (see Tables B-1 through B-4). Though not statistically significant, absolute body weight in the high-dose group was decreased by approximately 10% throughout the treatment period in both sexes. A smaller decrease of approximately 5% was also observed in most of the weekly body-weight measurements in both sexes of the mid-dose group (see Table B-5). Total food consumption was decreased by 17% in males and 8% in females in the high-dose group (see Table B-6). As shown in Table B-7, males in the mid-dose group showed biologically significantly increased absolute (10%, not statistically significant) and relative liver weights (14%, liver-to-body weight ratio). Additionally, males in the high-dose group exhibited an increase in absolute liver weight (29%), liver-to-body weight ratio (40%), and liver-to-brain weight ratio (31%). Females in the high-dose group showed a statistically significant increase in absolute, liver-to-body, and liver-to-brain weight measurements (16–24%). Also, females in the low-dose group showed statistically significant decreases in relative spleen weight, but these changes were not dose dependent. No other treatment-related effects on organ weight were observed (see Tables B-8 through B-12). Gross and histopathological findings among treated animals were comparable to controls. The authors did not report a NOAEL or a LOAEL for the study. However, based on biologically significantly increased absolute and relative liver weights in the mid-dose male rats, a LOAEL of 21.6 mg/kg-day is identified, with a corresponding NOAEL of 7.13 mg/kg-day.

*Allen and Norback (1973)*

*Allen and Norback (1973)* conducted a peer-reviewed, subchronic-duration study in which aroclor 5460 (purity not reported) was administered in the diet to 6 male rhesus monkeys at 5,000 ppm for 12 weeks. An adjusted daily dose of 690 mg/kg-day is calculated for this PPRTV assessment utilizing the average body weight (2.9 kg) and daily food consumption (0.4 kg) provided in the study report. Four male rhesus monkeys served as untreated controls. All animals were provided 400 g of commercial feed (brand not stated) on a daily basis. Throughout the course of the study, the general appearance of the animals was evaluated daily. Blood was drawn biweekly for hematological and biochemical analysis of hemoglobin, hematocrits, differential white count, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), total protein, and serum electrophoresis. Additional parameters assessed included body weight, histological examination of the liver and gastrointestinal tract, and electron microscopy of the liver. Histopathological evaluation of other tissues was not described.

The study authors reported an average decrease in body weight of 19% in the treated group; however, raw body-weight data were not provided. Within 6 weeks, all of the aroclor 5460-fed animals had alopecia. Hematocrit, lymphocytes, and total serum protein levels were decreased after 12 weeks in the treated group (statistical significance not reported), but information on hematological measurements was not provided for the control group. Findings at necropsy included a purulent discharge exuded from the eyes, and isolated acneiform lesions present on skin areas devoid of hair. A progressive, generalized, subcutaneous edema of the face was manifested as swollen eyelids and lips. Liver weight was increased in the treated animals (2.3% of the body weight in controls versus 5.6% in the treatment group). Electron microscopy analysis showed that the hypertrophic state of the liver in treated animals was due to an increase in lipid droplets and proliferation of the smooth endoplasmic reticulum. Edematous thickening of the stomach wall, marked hypertrophy of the pyloric and fundic regions, and ulceration of the gastric mucosa were also noted in the aroclor 5460-treated animals. The study authors stated that

the gastric submucosa contained large cystic areas that occasionally ruptured and led to necrosis. However, the actual incidences of the clinical and pathological observations were not reported. A NOAEL or LOAEL for this study was not specified by the authors. However, based on the effects listed above, the 690 mg/kg-day dose is considered the LOAEL. Identification of a NOAEL is precluded.

***Chronic-Duration Studies***

No studies were identified.

***Developmental Studies***

No studies were identified.

**Inhalation Exposures**

No inhalation studies were found on the subchronic-duration, chronic-duration, developmental, or reproductive toxicity or on the carcinogenicity of aroclor 5460 in animals.

**OTHER DATA (SHORT-TERM TESTS, OTHER EXAMINATIONS)**

Table 4 summarizes other studies conducted with aroclor 5460 that are not appropriate for selection of a point of departure (POD) for derivation of a provisional RfD (p-RfD) but provide supportive data.

**Table 4. Other Studies**

Test	Materials and Methods	Results	Conclusions	References
Developmental	White Leghorn chickens (4 males/20 females per dose except 8 males at the high dose) were administered aroclor 5460 in the diet at concentrations of 0; 500; 1,000; or 2,000 ppm for 18 wk. Mortality, food consumption, behavioral reaction, body weight, egg production, and gizzard color were some of the parameters examined.	Maternal: Mortality as a frank effect was observed at the high dose in both sexes.  Fetal: Ambiguous results as presented.	Confounding factors in this study precluded conclusions on fetal toxicity.	<a href="#">Industrial Bio-Test Laboratories (1983c, d)</a>
Developmental	Twelve White Leghorn chicks (sex unspecified) were administered aroclor 5460 in the diet at doses of 0, 50, 200, or 400 ppm for 21 d. Chicks were examined for mortality, clinical signs of toxicity, body weight, and pericardial fluid volume.	No effects observed.	Aroclor 5460 was not developmentally toxic to chickens at dose levels up to 400 ppm for 21 d.	<a href="#">Industrial Bio-Test Laboratories (1983a)</a>
Short-term	LD <sub>50</sub> tests in rats	Oral LD <sub>50</sub> in rats was 19,200 mg/kg.	The short-term toxicity of aroclor 5460 in rats appeared minimal.	<a href="#">Fishbein (1974)</a> as cited in <a href="#">Jensen and Jørgensen (1983)</a>
Short-term	Dermal minimum lethal dose (MLD) tests in rabbits	MLD in rabbits was 7,940 mg/kg.	The short-term toxicity of aroclor 5460 in rabbits appeared minimal.	<a href="#">Fishbein (1974)</a> as cited in <a href="#">Jensen and Jørgensen (1983)</a>
Metabolism/ toxicokinetic	Wistar rats (6 males/dose) were treated with 0; 10; 100; or 1,000 ppm aroclor 5460 in the diet for 7 d. Tissue distribution, weights, and microsomal activity (aniline hydroxylase, aminopyrine <i>N</i> -demethylase and cytochrome P450), protein, and phospholipid levels were measured in the liver.	Liver weights were increased. Residues were found in all tissues analyzed but were highest in the liver. Increased levels of enzyme activity, microsomal protein, and phospholipids were observed.	Aroclor 5460 has an inductive effect on the hepatic microsomal enzyme system.	<a href="#">Sosa-Lucero et al. (1973)</a>
Mode of action/ mechanistic	Four male S-D rats received daily intraperitoneal injections of 0 or 300 mg/kg-d aroclor 5460 for 4 d and sacrificed on Day 5. Livers were weighed and microsomes prepared and analyzed using gel electrophoresis and microsomal enzyme assays.	Increases were observed in microsomal protein and cytochrome P450 levels. No changes in body or liver weight were observed.	Aroclor 5460 has an inductive effect on the hepatic microsomal enzyme system.	<a href="#">Nilsen and Toftgård (1981)</a>

**Table 4. Other Studies**

<b>Test</b>	<b>Materials and Methods</b>	<b>Results</b>	<b>Conclusions</b>	<b>References</b>
Mode of action/ mechanistic	CD-1 mice (5/sex/dose) were given a single gavage dose of 0; 50; 100; 250; 500; or 1,000 mg/kg bw aroclor 5460. The mice were sacrificed 7 d after dosing. Intestines were examined microscopically, and intestinal sacs were prepared for the measurement of D-glucose absorption and fluid transfer.	Aroclor 5460 treatment reduced D-glucose absorption but not fluid transfer.	Malabsorption of D-glucose occurred and may have been attributed to intracellular metabolic effects of aroclor 5460.	<a href="#">Madge (1977)</a>

## Developmental Studies in Chickens

Three developmental studies were conducted in chickens ([Industrial Bio-Test Laboratories, 1983a, c, d](#)). EPA guidelines for acceptable developmental toxicity studies (870.3700) require testing in the “most relevant species.” The preferred species for developmental toxicity studies are rats and rabbits. No evidence was presented in the available studies to support the chicken as a relevant species for the assessment of the developmental toxicity of aroclor 5460 as it pertains to humans. Furthermore, these studies did not evaluate sufficient parameters for consideration as acceptable developmental toxicity studies. Other study-specific deficiencies include inadequate presentation of methods and inadequate number of dose groups. For these reasons, these studies are not considered acceptable developmental toxicity studies suitable for establishing reference toxicity values.

Two separate reports were issued by [Industrial Bio-Test Laboratories \(1983c, d\)](#) to Monsanto that describe different endpoints from what appears to be a single study. These studies were performed before EPA GLP guidelines were established. Although somewhat illegible, both reports appear to have the same study designation, IBT No. J5314. The report for the longer-term study, [Industrial Bio-Test Laboratories \(1983c\)](#), was designated as a chicken toxicity study with aroclor 5460. In this study, 12-week-old White Leghorn chickens were administered aroclor 5460 (purity not reported) in the diet at concentrations of 0; 500; 1,000; or 2,000 ppm (adjusted daily doses calculated for this PPRTV assessment are 0, 30.2, 62.6, and 126 mg/kg-day for males and 0, 40.9, 67.7, and 154.7 mg/kg-day for females).<sup>2</sup> Each group had 4 males and 20 females; however, all four males at 2,000 ppm died at early time points (cause of death not reported) and were replaced by four additional males. The duration of treatment was not specified, although it is assumed that the dosing formulations were administered throughout the evaluation period of 18 weeks. The chickens were weighed weekly beginning at Week 1 of the experiment (when chickens were 12 weeks of age) through Week 18, and observed daily for mortality and behavioral reaction. Food consumption measurements were made periodically and reported in weekly intervals (average grams/chicken/day) through Week 16; however, food consumption was not reported separately for each sex. As females in each group began laying eggs, the eggs were collected twice a day, numbered, and weighed. No treatment-related effects were observed on body weight, food consumption, or behavioral reaction (type of reaction assessed not specified). Increased mortality was observed at 2,000 ppm in males (7/8 treated versus 0/4 controls) and females (12/20 treated versus 4/20 controls), therefore, mortality confounded the interpretation of the data as reported. Egg production was decreased at 1,000 and 2,000 ppm; however, the average number of eggs per chicken per week was not presented. The content of the gizzard in many of the birds was green in color, but the significance of this finding is unclear. No other treatment-related pathological findings were reported.

The second report, [Industrial Bio-Test Laboratories \(1983d\)](#), was designated as an egg hatchability and chick viability study following aroclor 5460 treatment. The duration of treatment was not stated, but chick viability data were only reported for 4 weeks. In this study, 12-week-old White Leghorn chickens were administered aroclor 5460 (purity not reported) in the diet at concentrations of 0; 500; 1,000; or 2,000 ppm. At each dose, there were 4 males and 20 females; however, all 4 males at 2,000 ppm died (cause of death not reported) and were replaced by 4 other males. The weight and food consumption of these animals were not

---

<sup>2</sup>Adjusted daily dose = dose in ppm × (daily food consumption ÷ body weight). For 500 ppm in male chickens:  
Adjusted daily dose = 500 ppm × (0.117 mg/day ÷ 1.94 kg) = 30.2 mg/kg-day.

reported, thus adjusted daily doses could not be calculated. It was stated that food and water were offered ad libitum to the chicks and that they were observed for 30 days prior to sacrifice and necropsy to evaluate gross pathology. However, whether chicks were directly exposed to aroclor 5460 is unclear. Hatchability, behavior, and chick viability were reported. Although a treatment-related decrease in hatchability was observed, the lack of a clear understanding of the dosimetry and treatment duration precludes the identification of a NOAEL and LOAEL.

In a separate study performed for Monsanto by [Industrial Bio-Test Laboratories \(1983a\)](#), 12 single-comb White Leghorn chicks (cockerels)/group were administered aroclor 5460 in the diet at concentrations of 0, 50, 200, or 400 ppm ad libitum for 21 days. Adjusted daily doses could not be calculated because daily food consumption data were not available for this study. The chicks were received a day after hatching and were allowed a 48-hour acclimation period. Chicks were examined daily for mortality and evidence of toxicity. Body weights were measured prior to treatment and each week of treatment (Days 7, 14, and 21). After 21 days, the chicks were sacrificed and necropsied. Pericardial fluid was collected and the volume measured. Statistical analyses were focused on pericardial, subcutaneous, and peritoneal edema, and gross liver and kidney damage. There was no evidence of pericardial edema or adverse effects on mortality, behavioral reactions, body weight, or gross pathology. Based on the lack of observed effects, the highest concentration tested (400 ppm) is considered the NOAEL.

#### **Short-term-Duration Studies**

[Jensen and Jørgensen \(1983\)](#) reported data from [Fishbein \(1974\)](#) that showed that the oral LD<sub>50</sub> in rats was 19,200 mg/kg for aroclor 5460. Additionally, the dermal minimum lethal dose in rabbits was determined to be 7,940 mg/kg. Thus, the lethality of a single dose of aroclor 5460 is minimal.

#### **Metabolism/Toxicokinetic Studies**

[Sosa-Lucero et al. \(1973\)](#) evaluated the distribution of aroclor 5460 in rat tissues and its effect on hepatic microsomal mixed function oxidase. Six male Wistar Albino rats/dose group were sacrificed after treatment with 0; 10; 100; or 1,000 ppm (equivalent to adjusted daily doses of 0, 0.7, 6.5, and 64.7 mg/kg-day)<sup>3</sup> aroclor 5460 in the diet for 7 days. Blood, liver, brain, kidney, spleen, testes, heart, and omental fat were taken, weighed, and analyzed by electron capture gas chromatography to determine the concentration of aroclor 5460 found in each tissue. Microsomes were isolated from the liver and enzyme assays were conducted. No macroscopic manifestation of toxicity was observed, and body weights were not affected. Aroclor 5460 residues were found at dose-dependent concentrations in all tissues analyzed, with the greatest concentration in the liver and the least in the brain. In animals administered 1,000 ppm, the following statistically significant findings were observed: (1) absolute and relative liver weights were increased by 15–18%; (2) microsomal protein and phospholipids were increased by 28–36%; (3) cytochrome P450 was increased by 72%; (4) aniline hydroxylase activity was increased by 12%; and (5) aminopyrine *N*-demethylase activity was increased by 63%. Although glucose levels were decreased by 26%, these changes were not statistically significant. The

---

<sup>3</sup>Adjusted daily dose = dose in ppm × (daily food consumption ÷ body weight), where a default daily food consumption value of 0.02 kg ([U.S. EPA, 1988](#)) was used and body weight information was obtained from [Sosa-Lucero et al. \(1973\)](#).

increased levels of drug-metabolizing enzyme activities and the parallel increase in the content of microsomal protein, phospholipids, and cytochrome P450 indicated an inductive effect of aroclor 5460 on the hepatic microsomal enzyme system in the male rat.

#### **Mode of Action/Mechanistic Studies**

In a study by [Nilsen and Toftgård \(1981\)](#), four male Sprague-Dawley rats were injected intraperitoneally with aroclor 5460 (purity not reported) in corn oil at doses of 0 or 300 mg/kg body weight each day for 4 days. Animals were killed on the morning of the fifth day following a 24-hour fast. Livers were collected and weighed, and microsomes were prepared. Microsomes were subjected to SDS-polyacrylamide gel electrophoresis and microsomal enzyme assay analyses. Aroclor 5460 significantly increased (30%) the concentration of total microsomal cytochrome P450 protein content, although no changes in body or liver weights were observed. When the various forms of liver microsomal cytochrome P450 were analyzed separately, increases were noted in cytochrome P450 RLVMc P450<sub>55</sub> (275%), RLVMc P450<sub>54</sub> (40%), and RLVMc P450<sub>50</sub> (83%). The in vitro capacity of liver microsomes to metabolize biphenyl, benzo(a)pyrene, and androstene-3,17-dione was also increased (7- to 19-fold). These findings indicate that aroclor 5460 increased the metabolic activity of the liver in rats.

[Madge \(1977\)](#) evaluated in vitro intestinal absorption of different substrates in the CD-1 mouse (five/sex/dose) following single gavage doses of aroclor 5460 in corn oil at doses of 0; 50; 100; 250; 500; or 1,000 mg/kg bw. Seven days after dosing, the mice were sacrificed. The intestines were excised, examined microscopically, and in vitro inverted intestinal sacs were prepared to measure D-glucose absorption. The histology of the small intestine was unchanged, as was the wet intestinal weight. D-glucose absorption, but not fluid transfer, was significantly decreased following treatment with 250; 500; or 1,000 mg/kg body weight of aroclor 5460. Absorption of D-galactose, L-arginine, and L-histidine remained unaltered. When D-mannose, a compound known to diffuse through intestinal tissues, was added to the serosal fluid to provide absorptive cells with an added source of energy, differences in D-glucose absorption were not found between aroclor 5460-treated animals and controls. The study author concluded that malabsorption of D-glucose probably resulted from intracellular metabolic effects of aroclor 5460 and not malfunctions of the D-glucose carrier at the mucosal membrane.

### **DERIVATION OF PROVISIONAL VALUES**

Tables 5 and 6 present summaries of noncancer and cancer reference values, respectively.

<b>Table 5. Summary of Noncancer Reference Values for Aroclor 5460 (CASRN 11126-42-4)</b>							
<b>Toxicity Type (units)</b>	<b>Species/Sex</b>	<b>Critical Effect</b>	<b>p-Reference Value</b>	<b>POD Method</b>	<b>POD<sub>HED</sub></b>	<b>UF<sub>C</sub></b>	<b>Principal Study</b>
Screening Subchronic p-RfD (mg/kg-d)	Rat/M	Increased absolute and relative (liver-to-body) liver weight	$6 \times 10^{-3}$	NOAEL	1.71	300	<a href="#">Industrial Bio-Test Laboratories (1983b)</a>
Screening Chronic p-RfD (mg/kg-d)	Rat/M	Increased absolute and relative (liver-to-body) liver weight	$6 \times 10^{-4}$	NOAEL	1.71	3,000	<a href="#">Industrial Bio-Test Laboratories (1983b)</a>
Subchronic p-RfC (mg/m <sup>3</sup> )	NDr						
Chronic p-RfC (mg/m <sup>3</sup> )	NDr						

NDr = not determined.

<b>Table 6. Summary of Cancer Values for Aroclor 5460 (CASRN 11126-42-4)</b>				
<b>Toxicity Type</b>	<b>Species/Sex</b>	<b>Tumor Type</b>	<b>Cancer Value</b>	<b>Principal Study</b>
p-OSF	NDr			
p-IUR	NDr			

NDr = not determined.

## DERIVATION OF ORAL REFERENCE DOSES

No studies investigating the effects of aroclor 5460 are considered appropriate for the derivation of p-RfDs. The database for aroclor 5460 oral toxicity studies includes two subchronic-duration studies conducted in rats and monkeys ([Industrial Bio-Test Laboratories, 1983b](#); [Allen and Norback, 1973](#)). The subchronic-duration monkey study by [Allen and Norback \(1973\)](#) was peer reviewed and identified a LOAEL of 690 mg/kg-day based on skin, liver and stomach effects. However, this study is limited because incidences of the findings were not reported, only the liver and stomach were histopathologically analyzed, only young male monkeys were used, and only a single high dose was tested. Benchmark dose modeling of the data could not be performed due to the presence of a single treatment group.

From the subchronic-duration rat study by [Industrial Bio-Test Laboratories \(1983b\)](#), a NOAEL of 7.13 mg/kg-day and a LOAEL of 21.6 mg/kg-day are identified for male rats based on increased absolute and relative liver weight. Female rats were less sensitive, with a NOAEL of 23.5 mg/kg-day and a LOAEL of 77.8 mg/kg-day for the same liver effects. No histopathological changes in either sex were reported in the liver. This study was not peer reviewed, and it was performed before EPA GLP guidelines were established. Additionally, there are misgivings about the quality of the data presented because this study was performed during a time where critical errors were committed at Industrial Bio-Test Laboratories. Benchmark dose modeling was not possible because the study authors presented only mean values without standard deviation (SD) or individual data.

Liver changes, such as increased liver weight and liver hypertrophy, were commonly observed endpoints in several studies in multiple animal species treated with aroclor 5460 ([Industrial Bio-Test Laboratories, 1983b](#); [Shirai et al., 1978](#); [Allen and Norback, 1973](#)). These changes were associated with enhanced proliferation of liver cell smooth endoplasmic reticulum and liver enzyme induction ([Nilsen and Toftgård, 1981](#); [Allen and Norback, 1973](#); [Sosa-Lucero et al., 1973](#)). Similar liver toxicity was also seen in monkeys chronically exposed to the related PCB compound aroclor 1254. For example, animals treated with aroclor 1254, a compound similar in chlorination to aroclor 5460, exhibit increased liver weights and hepatocellular hypertrophy accompanied by progressive necrosis ([U.S. EPA, 1994a](#)). This observation further supports the liver as a target organ for aroclor 5460 toxicity.

From the available database for oral exposure to aroclor 5460, the rat study by ([Industrial Bio-Test Laboratories, 1983b](#)) is the only one that examines an adequate number of subjects of both sexes, and provides the most sensitive LOAEL for liver effects (LOAEL = 21.6 mg/kg-day for increased absolute and relative liver weight in male rats). However, because the report was not peer reviewed or published, the data were not clearly presented, and there are concerns regarding the quality of studies performed by Industrial Bio-Test Laboratories, this study is considered unsuitable for the derivation of p-RfDs. Instead, screening p-RfDs are derived in Appendix A.

## DERIVATION OF INHALATION REFERENCE CONCENTRATIONS

No suitable published studies investigating the effects of subchronic- or chronic-duration inhalation toxicity of aroclor 5460 in humans or animals were identified.

## CANCER WEIGHT-OF-EVIDENCE (WOE) DESCRIPTOR

Table 7 identifies the cancer WOE descriptor for aroclor 5460.

**Table 7. Cancer WOE Descriptor for Aroclor 5460**

<b>Possible WOE Descriptor</b>	<b>Designation</b>	<b>Route of Entry (oral, inhalation, or both)</b>	<b>Comments</b>
<i>“Carcinogenic to Humans”</i>	NS	NA	No human carcinogenicity data were identified.
<i>“Likely to Be Carcinogenic to Humans”</i>	NS	NA	No animal carcinogenicity studies were identified.
<i>“Suggestive Evidence of Carcinogenic Potential”</i>	NS	NA	No animal carcinogenicity studies were identified.
<b><i>“Inadequate Information to Assess Carcinogenic Potential”</i></b>	<b>Selected</b>	<b>Both</b>	<b>This descriptor is selected due to the lack of any information on the carcinogenicity of aroclor 5460.</b>
<i>“Not Likely to Be Carcinogenic to Humans”</i>	NS	NA	Although the genotoxicity studies were negative or equivocal, there are no data to indicate that aroclor 5460 is not carcinogenic.

NA = not applicable; NS = not selected.

#### **DERIVATION OF PROVISIONAL CANCER POTENCY VALUES**

The lack of data on the carcinogenicity of aroclor 5460 precludes the derivation of quantitative estimates for either oral (p-OSF) or inhalation (p-IUR) exposure.

## APPENDIX A. PROVISIONAL SCREENING VALUES

For the reasons noted in the main document, subchronic and chronic provisional reference doses (p-RfDs) for aroclor 5460 could not be derived. However, information is available for this chemical which, although insufficient to support derivation of a provisional toxicity value under current guidelines, may be of limited use to risk assessors. In such cases, the Superfund Health Risk Technical Support Center summarizes available information in an appendix and develops a “screening value.” Appendices receive the same level of internal and external scientific peer review as the main documents to ensure their appropriateness within the limitations detailed in the document. Users of screening toxicity values in an appendix to a PPRTV assessment should understand that there is considerably more uncertainty associated with the derivation of an appendix screening toxicity value than for a value presented in the body of the assessment. Questions or concerns about the appropriate use of screening values should be directed to the Superfund Health Risk Technical Support Center.

### DERIVATION OF SCREENING PROVISIONAL RfDs

The report by [Industrial Bio-Test Laboratories \(1983b\)](#) is selected as the principal study for derivation of the screening p-RfD. The critical effect is increased absolute and relative liver weight in male rats following exposure to aroclor 5460. Although this is an unpublished, non-peer-reviewed subchronic-duration rat study and there are misgivings regarding the quality of studies performed by Industrial Bio-Test Laboratories historically, this particular study did utilize an adequate number of animals and examined relevant tissues and parameters. Liver effects are further supported by the similar response observed in female rats in the same study and in monkeys in the only other subchronic-duration study ([Allen and Norback, 1973](#)). Although limited by their short-term duration and lack of comprehensive examination of tissues, two other studies also support the liver as a target organ of aroclor 5460 toxicity, with observed effects including increased liver weight, enhanced proliferation of liver cell smooth endoplasmic reticulum, and liver enzyme induction ([Nilsen and Toftgård, 1981](#); [Allen and Norback, 1973](#); [Sosa-Lucero et al., 1973](#)). The monkey study is limited because it utilized only young males, did not present quantitative histopathological information, and tested a single dose of aroclor 5460 (690 mg/kg-day), which is considerably higher than all doses tested in the rat study ([Industrial Bio-Test Laboratories, 1983b](#)). Thus, the NOAEL of 7.13 mg/kg-day identified from the report by [Industrial Bio-Test Laboratories \(1983b\)](#) constitutes the lowest point of departure (POD) among available studies and is used as the POD in the derivation of screening p-RfDs.

### Derivation of Screening Subchronic p-RfD

The screening subchronic p-RfD for aroclor 5460, based on the NOAEL of 7.13 mg/kg-day ([Industrial Bio-Test Laboratories, 1983b](#)), is derived as follows:

The U.S. EPA endorses body-weight scaling to the  $3/4$  power ( $BW^{3/4}$ ) to extrapolate toxicologically equivalent doses of orally administered agents from all laboratory animals to humans for the purpose of deriving an RfD under certain exposure conditions. The use of  $BW^{3/4}$  scaling for deriving an RfD is recommended when the observed effects are associated with the parent compound or a stable metabolite but not for portal-of-entry or developmental endpoints. Thus, following the [U.S. EPA \(2011b\)](#) guidance, the POD for increased absolute and relative liver weight in male rats is converted to a human equivalent dose (HED) through an application of a dosimetric adjustment factor (DAF) as follows:

$$DAF = (BW_a^{1/4} \div BW_h^{1/4})$$

Where:

DAF = dosimetric adjustment factor

BW<sub>a</sub> = animal body weight

BW<sub>h</sub> = human body weight

Using a BW<sub>a</sub> of 0.25 kg for rats and a default BW<sub>h</sub> of 70 kg for humans ([U.S. EPA, 2011b](#)), the resulting DAF is 0.24. Applying this DAF to the NOAEL identified in the rat subchronic-duration study yields a NOAEL<sub>HED</sub> as follows:

$$\begin{aligned} \text{POD}_{\text{HED}} &= \text{NOAEL (mg/kg-day)} \times \text{DAF} \\ &= \text{NOAEL (mg/kg-day)} \times 0.24 \\ &= 7.13 \text{ (mg/kg-day)} \times 0.24 \\ &= 1.71 \text{ mg/kg-day} \end{aligned}$$

$$\begin{aligned} \text{Screening Subchronic p-RfD} &= \text{NOAEL}_{\text{HED}} \div \text{UF}_C \\ &= 1.71 \text{ mg/kg-day} \div 300 \\ &= \mathbf{6 \times 10^{-3} \text{ mg/kg-day}} \end{aligned}$$

Table A.1 summarizes the uncertainty factors for the screening subchronic p-RfD for aroclor 5460.

<b>Table A.1. Uncertainty Factors for the Screening Subchronic p-RfD for Aroclor 5460</b>		
<b>UF</b>	<b>Value</b>	<b>Justification</b>
UF <sub>A</sub>	3	A UF <sub>A</sub> of 3 (10 <sup>0.5</sup> ) has been applied to account for uncertainty in characterizing the toxicodynamic differences between rats and humans following oral aroclor 5460 exposure. The toxicokinetic uncertainty has been accounted for by calculation of a HED through application of a DAF as outlined in the EPA's <i>Recommended Use of Body Weight<sup>3/4</sup> as the Default Method in Derivation of the Oral Reference Dose</i> ( <a href="#">U.S. EPA, 2011b</a> ).
UF <sub>D</sub>	10	A UF <sub>D</sub> of 10 has been applied because there are no acceptable two-generation reproductive toxicity or developmental toxicity studies via the oral route.
UF <sub>H</sub>	10	A UF <sub>H</sub> of 10 has been applied for inter-individual variability to account for human-to-human variability in susceptibility in the absence of quantitative information to assess the toxicokinetics and toxicodynamics of aroclor 5460 in humans.
UF <sub>L</sub>	1	A UF <sub>L</sub> of 1 has been applied for LOAEL-to-NOAEL extrapolation because the POD is a NOAEL.
UF <sub>S</sub>	1	A UF <sub>S</sub> of 1 has been applied because a subchronic-duration study was selected as the principal study.
UF <sub>C</sub>	300	Composite UF.

### Derivation of Screening Chronic p-RfD

Based on the same database and similar considerations, and utilizing the NOAEL<sub>HED</sub> of 1.74 mg/kg-day ([Industrial Bio-Test Laboratories, 1983b](#)) as the POD, the screening chronic p-RfD for aroclor 5460 is derived as follows:

$$\begin{aligned}
 \text{Screening Chronic p-RfD} &= \text{NOAEL}_{\text{HED}} \div \text{UF}_{\text{C}} \\
 &= 1.71 \text{ mg/kg-day} \div 3,000 \\
 &= \mathbf{6 \times 10^{-4} \text{ mg/kg-day}}
 \end{aligned}$$

Table A.2 summarizes the uncertainty factors for the screening chronic p-RfD for aroclor 5460.

UF	Value	Justification
UF <sub>A</sub>	3	A UF <sub>A</sub> of 3 (10 <sup>0.5</sup> ) has been applied to account for uncertainty in characterizing the toxicodynamic differences between rats and humans following oral aroclor 5460 exposure. The toxicokinetic uncertainty has been accounted for by calculation of a HED through application of a DAF as outlined in the EPA's <i>Recommended Use of Body Weight<sup>3/4</sup> as the Default Method in Derivation of the Oral Reference Dose</i> ( <a href="#">U.S. EPA, 2011b</a> ).
UF <sub>D</sub>	10	A UF <sub>D</sub> of 10 has been applied because there are no acceptable two-generation reproductive toxicity or developmental toxicity studies via the oral route.
UF <sub>H</sub>	10	A UF <sub>H</sub> of 10 has been applied for inter-individual variability to account for human-to-human variability in susceptibility in the absence of quantitative information to assess the toxicokinetics and toxicodynamics of aroclor 5460 in humans.
UF <sub>L</sub>	1	A UF <sub>L</sub> of 1 has been applied for LOAEL-to-NOAEL extrapolation because the POD is a NOAEL.
UF <sub>S</sub>	10	A UF <sub>S</sub> of 10 has been applied to account for the extrapolation from less than chronic exposure because no chronic-duration toxicity studies are available to evaluate chronic systemic toxicity.
UF <sub>C</sub>	3,000	Composite UF.

APPENDIX B. DATA TABLES

Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Total Leukocyte Count ( $10^3/\text{mm}^3$ ) on Day			Erythrocyte Count ( $10^6/\text{mm}^3$ ) on Day		
			0	33	89	0	33	89
0	0	M	15.6	18.7	16.6	5.90	6.66	7.73
		F	14.5	14.6	8.1	5.78	6.78	6.94
1,000	65.6 77.8	M	13.6 (-13)	21.2 (13)	16.3 (-2)	6.16 (4)	6.34 (-5)	6.87 (-11)
		F	13.6 (-6)	21.4 (47)	13.5 (67)	6.46 (12)	6.09 (-10)	6.27 (-10)
Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Hemoglobin Concentration (g/100 mL) on Day			Hematocrit Value (%) on Day		
			0	33	89	0	33	89
0	0	M	12.5	14.2	14.4	47	48	50
		F	12.9	14.2	14.4	47	47	47
1,000	65.6 77.8	M	12.8 (2)	12.4 (-13)	12.8 (-11)	48 (2)	43 (-10)	44 (-12)
		F	14.5 (12)	12.2 (-14)	12.7 (-12)	49 (4)	42 (-11)	43 (-9)

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table IV on page 11.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Differential Leukocyte Count (# cells/100)								
			Lymphocytes on Day			Neutrophils on Day			Monocytes on Day		
			0	33	89	0	33	89	0	33	89
0	0	M	87.6	92.8	85.0	9.8	6.6	14.4	2.4	0.2	0.4
		F	90.0	94.4	92.8	6.2	4.8	6.2	3.2	0.0	0.2
1,000	65.6 77.8	M	87.6 (0)	79.0	84.2 (-1)	8.8 (-10)	18.4	15.0 (4)	3.2 (33)	0.2 (0)	0.4 (0)
		F	84.8 (6)	(-15)	81.6	11.6 (87)	(179)	17.4	3.0 (-6)	0.0 (0)	1.0 (400)
				93.6 (1)	(-12)		4.6 (-4)	(181)			
Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Eosinophils on Day			Basophils on Day					
			0	33	89	0	33	89			
0	0	M	0.2	0.4	0.2	0.0	0.0	0.0			
		F	0.4	0.6	1.0	0.2	0.2	0.0			
1,000	65.6 77.8	M	0.4 (100)	2.0 (400)	0.4 (100)	0.0 (0)	0.4	0.0 (0)			
		F	0.6 (50)	1.6 (167)	0.0 (-100)	0.0 (-100)	0.2 (0)	0.0 (0)			

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table V on page 12.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

**Table B-3. Clinical Blood Chemistry Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a,b</sup>**

Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Serum Alkaline Phosphatase Activity (King-Armstrong Units) on Day			Blood Urea Nitrogen Concentration (mg Urea N/100 mL) on Day		
			0	33	89	0	33	89
0	0	M	39	40	12	18	19	17
	0	F	40	29	12	24	23	21
1,000	65.6	M	28 (-28)	36 (-10)	13 (8)	18 (0)	20 (5)	21 (24)
	77.8	F	27 (-33)	25 (-14)	12 (0)	18 (-25)	21 (-9)	26 (24)

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table VI on page 14.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

**Table B-4. Urinalysis Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a</sup>**

Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Glucose Concentration on Day			Albumin Concentration on Day			Specific Gravity on Day			pH on Day			Examination of Microscopic Elements on Day		
			0	33	89	0	33	89	0	33	89	0	33	89	0	33	89
0	0	M	n	n	n	n	n	t	1.015	1.031	1.037	6	7	7	N	N	N
	0	F	n	n	n	n	n	n	1.020	1.034	1.033	6	7	6	N	N	N
1,000	65.6	M	n	n	n	n	n	t	1.026	1.027	1.022	7	7	7	N	N	N
	77.8	F	n	n	n	n	n	t	1.015	1.035	1.028	6	7	6	N	N	N

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table VII on page 16.

n = negative; N = normal; t = trace: less than 10 mg albumin/100 mL.

**Table B-5. Body Weight and Weight Gain Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a</sup>**

Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Body Weight (g) <sup>b</sup> on Day							
			0	7	14	21	28	35	42	49
0	0	M	109	163	218	280	311	341	372	394
	0	F	107	146	171	200	205	223	227	239
100	7.13	M	110 (1)	171 (5)	222 (2)	286 (2)	312 (0)	353 (4)	365 (-2)	400 (2)
	8.06	F	107 (0)	148 (1)	170 (-1)	198 (-1)	206 (0)	219 (-2)	224 (-1)	242 (1)
300	21.6	M	109 (0)	166 (2)	218 (0)	269 (-4)	292 (-6)	327 (-4)	343 (-8)	374 (-5)
	23.5	F	107 (0)	143 (-2)	163 (-5)	190 (-5)	203 (-1)	216 (-3)	221 (-3)	234 (-2)
1,000	65.6	M	110 (1)	159 (-2)	200 (-8)	254 (-9)	280 (-10)	309 (-9)	328 (-12)	357 (-9)
	77.8	F	107 (0)	143 (-2)	161 (-6)	189 (-6)	196 (-4)	208 (-7)	214 (-6)	221 (-8)
Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Body Weight (g) <sup>b</sup> on Day							95-Day Weight Gains (g)
			56	63	70	77	84	91	95	
0	0	M	431	444	452	488	503	510	516	407
	0	F	251	257	254	274	280	276	282	175
100	7.13	M	424 (-2)	440 (-1)	442 (-2)	476 (-2)	487 (-3)	496 (-3)	501 (-3)	391 (-4)
	8.06	F	247 (-2)	259 (1)	263 (4)	279 (2)	283 (1)	285 (3)	289 (2)	182 (4)
300	21.6	M	396 (-8)	423 (-5)	433 (-4)	462 (-5)	480 (-5)	491 (-4)	494 (-4)	385 (-5)
	23.5	F	240 (-4)	256 (0)	258 (2)	267 (-3)	272 (-3)	270 (-2)	278 (-1)	171 (-2)
1,000	65.6	M	387 (-10)	406 (-9)	417 (-8)	446 (-9)	463 (-8)	463 (-9)	474 (-8)	364 (-11)
	77.8	F	230 (-8)	238 (-7)	243 (-4)	252 (-8)	257 (-8)	259 (-6)	262 (-7)	155 (-11)

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table II on page 7.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

**Table B-6. Food Consumption Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a</sup>**

Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Food Consumed (g/rat/week) on Week						
			1	2	3 <sup>b</sup>	4 <sup>c</sup>	5 <sup>d</sup>	6	7
0	0	M	127	153	215	174	82	229	209
		F	99	112	177	138	62	139	138
100	7.13 8.06	M	130 (2)	158 (3)	231 (7)	182 (5)	109 (33)	199 (-13)	184 (-12)
		F	104 (5)	124 (11)	164 (-7)	125 (-9)	70 (13)	121 (-13)	133 (-4)
300	21.6 23.5	M	135 (6)	158 (3)	218 (1)	178 (2)	105 (28)	182 (-21)	181 (-13)
		F	101 (2)	117 (4)	158 (-11)	121 (-12)	72 (16)	112 (-19)	122 (-12)
1,000	65.6 77.8	M	97 (-24)	126 (-18)	181 (-16)	151 (-13)	72 (-12)	142 (-38)	177 (-15)
		F	100 (1)	113 (1)	154 (-13)	119 (-14)	54 (-13)	143 (-3)	114 (-17)
Dose (ppm)	Adjusted Daily Dose (mg/kg-d)	Sex	Food Consumed (g/rat/week) on Week						Total Food Consumption (g/rat)
			8	9	10	11 <sup>e</sup>	12	13	
0	0	M	221	213	234	146	198	195	2,393
		F	134	133	157	93	120	115	1,617
100	7.13 8.06	M	170 (-23)	184 (-14)	213 (-9)	132 (-10)	179 (-10)	177 (-9)	2,346 (-2)
		F	141 (5)	144 (8)	169 (8)	98 (5)	133 (11)	127 (10)	1,653 (2)
300	21.6 23.5	M	190 (-14)	187 (-12)	231 (-1)	144 (-1)	188 (-5)	186 (-5)	2,283 (-5)
		F	129 (-4)	134 (1)	161 (3)	91 (-2)	121 (1)	116 (1)	1,555 (-4)
1,000	65.6 77.8	M	163 (-26)	185 (-13)	218 (-7)	131 (-10)	173 (-13)	173 (-11)	1,989 (-17)
		F	108 (-19)	124 (-7)	149 (-5)	86 (-8)	116 (-3)	104 (-10)	1,484 (-8)

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table III on page 9.. Data are presented as mean values (absolute % change from controls); % change is calculated.

<sup>b</sup>Values represent a 9-d period.

<sup>c</sup>Values represent an 8-d period.

<sup>d</sup>Values represent a 4-d period.

<sup>e</sup>Values represent a 6-d period.

<b>Table B-7. Liver and Body Weight Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a,b</sup></b>									
<b>Dose (ppm)</b>	<b>Adjusted Daily Dose (mg/kg-d in M/F)</b>	<b>Terminal body weight (g)</b>		<b>Absolute Liver Weight (g)</b>		<b>Liver/Body Weight Ratio (g/100 g)</b>		<b>Liver/Brain Weight Ratio (g/g)</b>	
		<b>Males</b>	<b>Females</b>	<b>Males</b>	<b>Females</b>	<b>Males</b>	<b>Females</b>	<b>Males</b>	<b>Females</b>
0	0/0	516	282	22.6	11.7	4.39	4.16	11.8	5.98
100	7.13/8.06	501 (-3)	289 (2)	22.7 (0)	12.4 (6)	4.52 (3)	4.29 (3)	11.1 (6)	6.08 (2)
300	21.6/23.5	494 (-4)	278 (-1)	24.8 (10)	11.7 (0)	5.02 (14)*	4.23 (2)	12.3 (4)	6.07 (2)
1,000	65.6/77.8	474 (-8)	262 (-7)	29.1 (29)**	13.6 (16)**	6.14 (40)**	5.16 (24)**	15.5 (31)**	7.06 (18)**

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table VIII on page 18.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

\* $p < 0.05$ ; \*\* $p < 0.01$

**Table B-8. Kidney Weight Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a,b</sup>**

Dose (ppm)	Adjusted Daily Dose (mg/kg-d in M/F)	Absolute Kidney Weight (g)		Kidney/Body Weight Ratio (g/100 g)		Kidney/Brain Weight Ratio (g/g)	
		Males	Females	Males	Females	Males	Females
0	0/0	3.95	2.17	0.766	0.770	2.06	1.10
100	7.13/8.06	3.70 (-6)	2.21 (2)	0.738 (-4)	0.767 (0)	1.81 (-12)	1.09 (-1)
300	21.6/23.5	3.87 (-2)	2.36 (9)	0.785 (2)	0.848 (10)	1.92 (-7)	1.22 (11)
1,000	65.6/77.8	3.70 (-6)	2.19 (1)	0.782 (2)	0.833 (8)	1.97 (-4)	1.14 (4)

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table IX on page 19.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

**Table B-9. Spleen Weight Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a,b</sup>**

Dose (ppm)	Adjusted Daily Dose (mg/kg-d in M/F)	Absolute Spleen Weight (g)		Spleen/Body Weight Ratio (g/100 g)		Spleen/Brain Weight Ratio (g/g)	
		Males	Females	Males	Females	Males	Females
0	0/0	0.773	0.627	0.151	0.222	0.402	0.320
100	7.13/8.06	0.684 (-12)	0.498 (-21)	0.137 (-9)	0.172 (-23)**	0.336 (-16)	0.24 (-25)**
300	21.6/23.5	0.695 (-10)	0.546 (-13)	0.14 (-7)	0.196 (-12)	0.344 (-14)	0.282 (-12)
1,000	65.6/77.8	0.701 (-9)	0.563 (-10)	0.148 (-2)	0.214 (-4)	0.373 (-7)	0.293 (-8)

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table X on page 20.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

\*\* $p < 0.01$

**Table B-10. Gonad Weight Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a,b</sup>**

Dose (ppm)	Adjusted Daily Dose (mg/kg-d in M/F)	Absolute Gonad Weight (g)		Gonad/Body Weight Ratio (g/100 g)		Gonad/Brain Weight Ratio (g/g)	
		Males	Females	Males	Females	Males	Females
0	0/0	3.35	0.125	0.651	0.0444	1.74	0.0638
100	7.13/8.06	3.48 (4)	0.126 (1)	0.698 (7)	0.0438 (-1)	1.70 (-2)	0.0622 (-3)
300	21.6/23.5	3.53 (5)	0.110 (-12)	0.719 (10)	0.0398 (-10)	1.75 (1)	0.0571 (-11)
1,000	65.6/77.8	3.36 (0)	0.130 (4)	0.713 (10)	0.0494 (11)	1.80 (3)	0.0675 (6)

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table XI on page 21.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

**Table B-11. Heart Weight Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a,b</sup>**

Dose (ppm)	Adjusted Daily Dose (mg/kg-d in M/F)	Absolute Heart Weight (g)		Heart/Body Weight Ratio (g/100 g)		Heart/Brain Weight Ratio (g/g)	
		Males	Females	Males	Females	Males	Females
0	0/0	1.54	1.00	0.301	0.357	0.803	0.513
100	7.13/8.06	1.56 (1)	0.981 (-2)	0.313 (4)	0.341 (-4)	0.767 (-4)	0.485 (-5)
300	21.6/23.5	1.57 (2)	0.965 (-4)	0.318 (6)	0.348 (-3)	0.778 (-3)	0.499 (-3)
1,000	65.6/77.8	1.55 (1)	0.930 (-7)	0.328 (9)	0.355 (-1)	0.827 (3)	0.485 (-5)

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table XII on page 22.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

**Table B-12. Brain Weight Data from Albino Rats Exposed to Aroclor 5460 in the Diet for 95 Days<sup>a,b</sup>**

Dose (ppm)	Adjusted Daily Dose (mg/kg-d in M/F)	Absolute Brain Weight (g)		Brain/Body Weight Ratio (g/100 g)	
		Males	Females	Males	Females
0	0/0	1.92	1.96	0.375	0.699
100	7.13/8.06	2.04 (6)*	2.13 (9)	0.410 (9)	0.742 (6)
300	21.6/23.5	2.02 (5)	1.94 (-1)	0.411 (9.6)	0.699 (0)
1,000	65.6/77.8	1.88 (-2)	1.92 (-2)	0.399 (6)	0.735 (5)

<sup>a</sup>Values were obtained from [Industrial Bio-Test Laboratories \(1983b\)](#) from Table XIII on page 23.

<sup>b</sup>Data are presented as mean values (absolute % change from controls); % change is calculated.

\* $p < 0.05$

## **APPENDIX C. BMD OUTPUTS**

BMD analysis is not performed for this assessment.

## APPENDIX D. REFERENCES

- ACGIH (American Conference of Governmental Industrial Hygienists). (2013). 2013 TLVs and BEIs. Based on documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, OH.
- Allen, JR; Norback, DH. (1973). Polychlorinated biphenyl- and triphenyl-induced gastric mucosal hyperplasia in primates. *Science* 179: 498-499.
- ATSDR (Agency for Toxic Substances and Disease Registry). (2013). Minimal risk levels (MRLs) for hazardous substances. Atlanta, GA. Retrieved from <http://www.atsdr.cdc.gov/mrls/mrllist.aspID> - 2
- Cal/EPA (California Environmental Protection Agency). OEHHA toxicity criteria database. Sacramento, CA: Office of Environmental Health Hazard Assessment. <http://www.oehha.ca.gov/tcdb/>. Accessed 9-11-2013.
- Cal/EPA (California Environmental Protection Agency). (2009). Appendix A: Hot spots unit risk and cancer potency values. Sacramento, CA: Office of Environmental Health Hazard Assessment. [http://www.oehha.ca.gov/air/hot\\_spots/2009/AppendixA.pdf](http://www.oehha.ca.gov/air/hot_spots/2009/AppendixA.pdf)
- Cal/EPA (California Environmental Protection Agency). (2013a). All OEHHA acute, 8-hour and chronic reference exposure levels (chRELs) as of August 2013. Sacramento, CA: Office of Environmental Health Hazard Assessment. <http://www.oehha.ca.gov/air/allrels.html>  
<<http://www.oehha.ca.gov/air/allrels.html>
- Cal/EPA (California Environmental Protection Agency). (2013b). Current proposition 65 list [07/26/13]. Sacramento, CA: Office of Environmental Health Hazard Assessment. [http://oehha.ca.gov/prop65/prop65\\_list/Newlist.html](http://oehha.ca.gov/prop65/prop65_list/Newlist.html)
- FAO (Food and Agriculture Organization of the United Nations). (1992). Operation of the prior informed consent procedure for banned or severely restricted chemicals in international trade : decision guidance documents : crocidolite, polybrominated biphenyls, polychlorinated biphenyls, polychlorinated terphenyls, tris(2,3-dibromopropyl) phosphate. Geneva, Switzerland.
- Fishbein, L. (1974). Toxicity of Chlorinated Biphenyls. *Annu Rev Pharmacol Toxicol* 14: 139-156. <http://dx.doi.org/10.1146/annurev.pa.14.040174.001035>
- IARC (International Agency for Research on Cancer). (2013). Bitumens and bitumen emissions, and some N- and S-heterocyclic polycyclic aromatic hydrocarbons. IARC monographs on the evaluation of carcinogenic risks to humans, vol 103. Geneva, Switzerland: WHO. <http://monographs.iarc.fr/ENG/Monographs/vol103/mono103.pdf>
- Industrial Bio-Test Laboratories, Inc., (1983a). 21-day chick edema study aroclor 5460 [TSCA Submission]. (PB-848211260). St. Louis, MO: Monsanto Company.

- Industrial Bio-Test Laboratories, Inc. (1983b). 90-day subacute oral toxicity of aroclor 5460 - albino rats with cover letter [TSCA Submission]. (PB878211258). St. Louis, MO: Monsanto Company.
- Industrial Bio-Test Laboratories, Inc. (1983c). Report to Monsanto Company: Chicken toxicity study on aroclor 5460 - white leghorn chickens [TSCA Submission]. (PB-878211259). St. Louis, MO: Monsanto Company.
- Industrial Bio-Test Laboratories, Inc. (1983d). Report to Monsanto Company: Egg hatchability and chick viability study aroclor 5460 white leghorn chickens [TSCA Submission]. (PB-878211261). St. Louis, MO: Monsanto Company.
- Jensen, AA; Jørgensen, KF. (1983). Polychlorinated terphenyls (PCTs) use, levels and biological effects. *Sci Total Environ* 27: 231-250.
- Madge, DS. (1977). Polychlorinated terphenyls and intestinal transport in mice. *Gen Pharmacol* 8: 43-46.
- Monsanto. (1960). Exhibit 58: The Aroclor Compounds. St Louis, Missouri: Monsanto.
- Nilsen, OG; Toftgård, R. (1981). Effects of polychlorinated terphenyls and paraffins on rat liver microsomal cytochrome P-450 and in vitro metabolic activities. *Arch Toxicol* 47: 1-11.
- NIOSH (National Institute for Occupational Safety and Health). (2010). NIOSH pocket guide to chemical hazards. Index of chemical abstracts service registry numbers (CAS No.). Atlanta, GA: Center for Disease Control and Prevention, U.S. Department of Health, Education and Welfare. <http://www.cdc.gov/niosh/npg/npgdcas.html>
- NTP (National Toxicology Program). (2011). Report on carcinogens: Twelfth edition (12th ed.). Research Triangle Park, NC. <http://ntp.niehs.nih.gov/ntp/roc/twelfth/roc12.pdf>
- OSHA (Occupational Safety & Health Administration). (2006). Table Z-1 limits for air contaminants. Occupational Safety and Health Administration. [http://www.osha.gov/pls/oshaweb/owadisp.show\\_document?p\\_table=STANDARDS&p\\_id=9992](http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=9992)
- OSHA (Occupational Safety & Health Administration). (2011). Air contaminants: occupational safety and health standards for shipyard employment, subpart Z, toxic and hazardous substances. (OSHA Standard 1915.1000). Washington, DC: U.S. Department of Labor. [http://www.osha.gov/pls/oshaweb/owadisp.show\\_document?p\\_table=STANDARDS&p\\_id=10286](http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=10286)
- Shirai, T; Miyata, Y; Nakanishi, K; Murasaki, G; Ito, N. (1978). Hepatocarcinogenicity of polychlorinated terphenyl (PCT) in ICR mice and its enhancement by hexachlorobenzene (HCB). *Cancer Lett* 4: 271-275.
- Sosa-Lucero, JC; De la Iglesia, FA; Thomas, GH. (1973). Distribution of a polychlorinated terphenyl (PCT) (Aroclor 5460) in rat tissues and effect on hepatic microsomal mixed function oxidases. *Bull Environ Contam Toxicol* 10: 248-256.

- U.S. EPA (U.S. Environmental Protection Agency). Integrated risk information system (IRIS). Available online at <http://dx.doi.org/10.1177/1091581812466418>. Accessed 9-11-2013.
- U.S. EPA (U.S. Environmental Protection Agency). (1988). Recommendations for and documentation of biological values for use in risk assessment [EPA Report]. (EPA/600/6-87/008). Cincinnati, OH: U.S. Environmental Protection Agency, National Center for Environmental Assessment. <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=34855>
- U.S. EPA (U.S. Environmental Protection Agency). (1994a). Aroclor 1254 (CASRN 11097-69-1). Washington, DC: Integrated Risk Information System (IRIS). Retrieved from <http://www.epa.gov/iris/subst/0389.htmID - 1>
- U.S. EPA (U.S. Environmental Protection Agency). (1994b). Chemical assessments and related activities (CARA) [EPA Report]. (600R94904; OHEA-I-127). Washington, DC. <http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=60001G8L.txt>
- U.S. EPA (U.S. Environmental Protection Agency). (2002). A review of the reference dose and reference concentration processes [EPA Report]. (EPA/630/P-02/002F). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=51717>
- U.S. EPA (U.S. Environmental Protection Agency). (2011a). Health Effects Assessment Summary Tables (HEAST). Available online at <http://epa-heast.ornl.gov/>
- U.S. EPA (U.S. Environmental Protection Agency). (2011b). Recommended use of body weight 3/4 as the default method in derivation of the oral reference dose [EPA Report]. (EPA/100/R11/0001). Washington, DC. <http://www.epa.gov/raf/publications/interspecies-extrapolation.htm>
- U.S. EPA (U.S. Environmental Protection Agency). (2012a). 2012 Edition of the drinking water standards and health advisories. (EPA 822-S-12-001). Washington, DC: Office of Water. <http://water.epa.gov/action/advisories/drinking/upload/dwstandards2012.pdf>
- WHO (World Health Organization). (2012). Online catalog for the Environmental Health Criteria Series. Available online at <http://www.who.int/ipcs/publications/ehc/en/>