Final

Baseline Risk Assessment for Golf Course Impoundments at the Defense Distribution Depot, Memphis, Tennessee

May 1999

Prepared for:

U.S. Army Corps of Engineers
Mobile District

Timeline of Events
Baseline Risk Assessment of Golf Course Impoundments

1997: Radian contracted to conduct a baseline risk assessment using existing fish tissue and sediment sampling data; no new sampling.

1997: Upon receipt of draft Baseline Risk Assessment, the Tennessee Department of Environment and Conservation (TDEC) voiced concern about the use of 1985 and 1990 sampling data to determine current risk.

1998: Radian attempted to capture fish from Lake Danielson and the Golf Course Pond but captured only non-edible species. TDEC was not convinced that the necessary means to capture edible species were used and requested another attempt.

1998: The Tennessee Valley Authority assisted Radian to collect fish by electro-shocking Lake Danielson and verified that Lake Danielson and the Golf Course Pond did not contain edible species of fish. Non-edible species were captured, and Radian proceeded with the baseline risk assessment based on the non-edible species tissue samples.

FINAL

BASELINE RISK ASSESSMENT FOR GOLF COURSE IMPOUNDMENTS AT THE DEFENSE DISTRIBUTION DEPOT, MEMPHIS, TENNESSEE

Prepared for:

U.S. Army Corps of Engineers
Mobile District

Prepared by:

Radian International
1093 Commerce Park Drive, Suite 100
Oak Ridge, Tennessee 37830
F9708201.MW97

May 1999

This page intentionally left blank.

TABLE OF CONTENTS

		Page
LIST OF FIGURES		v
LIST OF TABLES		v
ACRONYMS		vii
EXECUTIVE SUMMARY		ix
1.0	INTRODUCTION	1-1
2.0	THE BASELINE RISK ASSESSMENT PROCESS	2-1
3.0	SITE DESCRIPTION	3-1
4.0	PREVIOUS INVESTIGATIONS	4-1
4.1	U.S. Army Environmental Hygiene Agency	4-1
4.2	1990 Remedial Investigation	4-1
4.3	Contaminant Fate and Transport	4-4
5.0	EXPOSURE SETTING	5-1
6.0	TOXICITY ASSESSMENT	6-1
6.1	4,4'-DDD,4,4'-DDE, and 4,4'-DDT	6-1
6.2	4,4'-DDD CAS No. 72-54-8	6-1
6.3	4,4'-DDE CAS No. 72-55-9	6-2
6.4	4,4'-DDT CAS No. 50-29-3	6-2
6.5	Chlordane	6-3
6.6	Dieldrin	6-5
6.7	Heptachlor Epoxide	6-7
7.0	INITIAL RISK CHARACTERIZATION	7-1
8.0	FOLLOW-UP INVESTIGATION	8-1
9.0	FINAL INVESTIGATION	9-1
10.0	UNCERTAINTY ANALYSIS	10-1
11.0	ECOLOGICAL RISK ASSESSMENT	11-1
12.0	CONCLUSIONS AND RECOMMENDATIONS	12-1
13.0	REFERENCES	13-1

TABLE OF CONTENTS (CONTINUED)

Appendix A: DETAILED TOXICITY SUMMARIES
Appendix B: RISK ASSESSMENT SPREADSHEETS
Appendix C: AUGUST 14, 1998, LETTER FROM TVA FISHERIES BIOLOGIST
Appendix D: PHOTOGRAPHS
Appendix E: CHAIN-OF-CUSTODY RECORDS
Appendix F: ANALYTICAL DATA

LIST OF FIGURES

		Page
1-1	Location Map of the Defense Distribution Depot, Memphis	1-2
1-2	Site Layout of the Defense Distribution Depot, Memphis	1-3
4-1	1990 Sediment Sample Locations, Lake Danielson and Golf Course Pond, Defense Distribution Depot Memphis, Tennessee	4-2
5-1	Conceptual Site Model, Lake Danielson and Golf Course Pond, Defense Distribution Depot, Memphis	5-3
8-1	1997 Sediment Sample Locations, Lake Danielson and Golf Course Pond, Defense Distribution Depot, Memphis	8-3

LIST OF TABLES

		Page
4-1	1990 RI Sediment Sampling Results, Lake Danielson and Golf Course Pond	4-3
7-1	Cancer Risk Estimates for Lake Danielson and Golf Course Pond Based on 1990 RI Data	7-1
8-1	Pesticide Concentration Reported for the 1997 Sediment and Fish Samples Collected from the Golf Course Pond at the Defense Distribution Depot, Memphis, Tennessee	8-5
8-2	Cancer Risk Estimates for Lake Danielson and Golf Course Pond Based on 1997 Data	8-6
9-1	Pesticide Concentrations Reported for the 1998 Fish and Frog Samples Collected from the Golf Course Pond at the Defense Distribution Depot, Memphis, Tennessee	9-2

This page intentionally left blank.

ACRONYMS

AEHA	U.S. Army Environmental Hygiene Agency
ATSDR	Agency for Toxic Substances and Disease Registry
BRA	Baseline Risk Assessment
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
DDD	Dichlorodiphenyldichloroethane
DDE	Dichlorodiphenyldichloroethene
DDT	Dichlorodiphneyltrichloroethane
EPA	U.S. Environmental Protection Agency
ERA	Ecological Risk Assessment
HEAST	Health Effects Assessment Summary Table
IRIS	Integrated Risk Information System
IRP	Installation Restoration Program
NOAEL	No Observed Adverse Effects Level
NOEL	No Observed Effect Level
NPDES	National Pollutant Discharge Elimination System
ppm	parts per million
RfC	Reference Concentration
RfD	Reference Dose
RI	Remedial Investigation
TVA	Tennessee Valley Authority
95 UCL	95% Upper Confidence Limit

This page intentionally left blank.

EXECUTIVE SUMMARY

In early 1997, a baseline risk assessment (BRA) was performed using all available data to evaluate human health and ecological risks associated with exposure to pesticide residues in the surface water impoundments on the golf course at the Defense Distribution Depot, Memphis, Tennessee (hereinafter referred to as the Depot). The Depot was scheduled for closure, but it was anticipated that the golf course would continue to be used as a golf course after the Depot closed.

The pesticide dichlorodiphneyltrichloroethane (DDT) and its degradation products, dichlorodiphenyldichloroethene (DDE) and dichlorodiphenyldichloroethane (DDD), were detected in sediment samples collected from the golf course impoundments during the 1990 Remedial Investigation (RI) (Law Environmental, Inc. 1990). Fishing and swimming in the impoundments is currently prohibited and will likely continue to be prohibited. However, it was assumed that a male youth would gain unauthorized access to the impoundments and would be exposed to contaminated sediments while swimming in the impoundments and as a result of eating fish caught from the impoundments.

No adverse health effects are anticipated from dermal contact and incidental ingestion of sediment while swimming. Ingestion of fish caught from the impoundments was conservatively estimated to increase the probability of developing cancer by almost 3 in 100,000.

In response to recommendations made in the 1997 risk assessment report (Radian 1997), additional sediment and fish samples were collected from the impoundments in late September/early October 1997 to provide more recent data for reevaluating risk. Again, the highest detected pesticide concentrations in sediment and fish muscle tissue were used to quantify human health risks via ingestion and dermal exposure, using the same exposure scenario. Except for the exposure concentrations of pesticides, the same values used to calculate contaminant intake and quantify toxic effects in the early 1997 risk assessment were used to reevaluate risk.

The only fish caught during the September/October 1997 sampling event were golden shiners (*Notropis girardi*), which are commonly used as bait fish. Although this fish species is not typically ingested by humans, there were no other, more appropriate data to use for

evaluating risk to humans via ingestion of fish from these impoundments. Analytical data on muscle tissue from a composite sample of several shiners were used as the representative exposure concentrations for pesticides in fish. Based on these data, the cancer risk associated with the modeled exposure is expected to be no greater than 7.3E-06 (i.e., a probability of 7.3 in a million of developing cancer). Most of the cancer risk (approximately 86%) is attributable to fish ingestion. The absence of fish species that are likely to be consumed by humans suggests that it is unlikely than anyone would actually incur a cancer risk of 7.3E-06 from eating fish from these impoundments.

U.S. Environmental Protection Agency (EPA) and State of Tennessee regulators expressed concern that the September/October 1997 sampling event might have failed to detect edible fish species that might possibly be present in the ponds. They recommended that electro-fishing be employed to definitively ascertain the presence or absence of edible fish. Radian subsequently obtained the services of the Tennessee Valley Authority (TVA) to electro-fish the golf course ponds.

This final sampling event took place 12–13 August 1998. TVA fisheries biologists used gill nets and a boat-mounted electro-fishing unit to make an exhaustive search of both ponds for fish and other aquatic vertebrates. Three gill nets were set in Lake Danielson and left overnight. No fish were captured by the gill nets. Electro-fishing revealed the presence of hundreds of golden shiners, the fish species that was observed and collected during the 1997 sampling event. After two circuits of Lake Danielson's shoreline and a series of transects that covered the entire pond surface area, for a total of more than 70 minutes of electro-fishing effort, no additional fish species were encountered. The TVA fisheries biologist with more than 20 years of experience with this type of sampling concluded that it is highly unlikely that any fish species other than the observed golden shiners are present in Lake Danielson.

Electro-fishing of the smaller pond revealed the presence of western mosquitofish (*Gambusia affinis*) and goldfish (*Carassius auratus*). Neither fish species is typically ingested by humans. The TVA biologist concluded that neither pond would be of interest to anglers. Shiners from Lake Danielson, goldfish from the smaller pond, and adult bullfrogs that were collected from each pond were analyzed for pesticides.

The analytical results from the August 1998 sampling event were used to reevaluate human health and ecological risks associated with exposure to contaminated media in the golf course ponds at the Defense Distribution Depot in Memphis, Tennessee. The pesticide concentrations in whole fish were used to evaluate the risk to piscivorous (fish-eating) birds. The pesticide concentrations in frog muscle tissue were used to evaluate the risk to humans who might ingest frog legs.

The same exposure scenario used for the previous human health risk assessment was applied to this quantification of risk. It was assumed that the ingestion rate of frog legs would be 10% the mean annual per capita fish ingestion rate for the United States since ingestion of frog legs is far less common than ingestion of fish. All other parameter values used in the previous risk assessment were applied to this quantification of risk. The total cancer risk due to ingestion of pesticides in frog legs is 4.9E-07. This is below the level of regulatory concern. The hazard index calculated for ingestion of all pesticides found in frog muscle tissue is 6E-04. A hazard index less than 1 indicates that non-cancer health effects are not expected to result from this exposure. Due to the absence of edible fish species in the golf course ponds, there is no other plausible exposure pathway that would result in unacceptable risk to human health.

Belted kingfishers (*Ceryle alcyon*) and great blue herons (*Ardea herodias*) are commonly occurring piscivorous birds whose geographic range includes the Memphis area, so these species were used to quantify ecological risks. Body weight and food ingestion rate values published for these birds by EPA (1993) were used to quantify risks associated with feeding from the golf course ponds.

The estimated intake of each pesticide for each receptor species was compared to the estimated No Observed Adverse Effects Levels (NOAEL) values published by Opresko et al. (1995). The NOAEL is the chemical-specific intake that has been experimentally observed to not cause adverse effects in the exposed species.

The estimated intake of DDT and its metabolites exceeded the estimated NOAEL values for both belted kingfishers and great blue herons. However, this analysis is based on the assumption that each bird obtains its entire food supply from the golf course ponds. Considering

This page intentionally left blank.

1.0 INTRODUCTION

The Depot is located in the city of Memphis in Shelby County, in the extreme southwestern portion of the state. The Depot is situated on 642 acres approximately 5 miles east of the Mississippi River and just northeast of the Interstate 240/55 junction. The Depot lies in the south-central section of Memphis, approximately 4 miles southeast of the central business district and 1 mile northwest of Memphis International Airport. Figure 1-1 is a map depicting the location of the Depot relative to the region, the city of Memphis, the Mississippi River, and the interstate highways.

Construction of the Depot began in June 1941, and operation of the Depot began in January 1942. The Depot's mission was to receive, store, maintain, and ship items such as food, clothing, electronic equipment, petroleum products, construction materials, and medical supplies to units of the U.S. military. The installation consists of 110 buildings, 26 miles of railroad track, and 28 miles of paved streets. Figure 1-2 is a site layout map. The Depot was closed in September 1997.

A nine-hole golf course is located on the southeast corner of the Depot. The golf course includes two surface water impoundments: Lake Danielson and the golf course pond. It is anticipated that the golf course will continue to be used for the foreseeable future.

The U.S. Department of Defense developed the Installation Restoration Program (IRP) in 1981 to evaluate and remediate the effects of past waste management and disposal practices at its facilities and to comply with the provisions of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), as amended. An RI was conducted for the Depot in 1990 as part of the IRP (Law Environmental, Inc. 1990). The purpose of the RI was to assess the nature and extent of contamination at the Depot, to examine the migration potential of detected contaminants, and to evaluate the risks associated with exposure to the contaminants. The RI Report suggested that pesticide residues in the surface water and bottom sediments in Lake Danielson and the golf course pond might pose a hazard to human health via ingestion of fish living in contact with the contaminated surface water/sediment. A BRA was conducted in

This page intentionally left blank.

N:\65572401\01\MAP.DWG

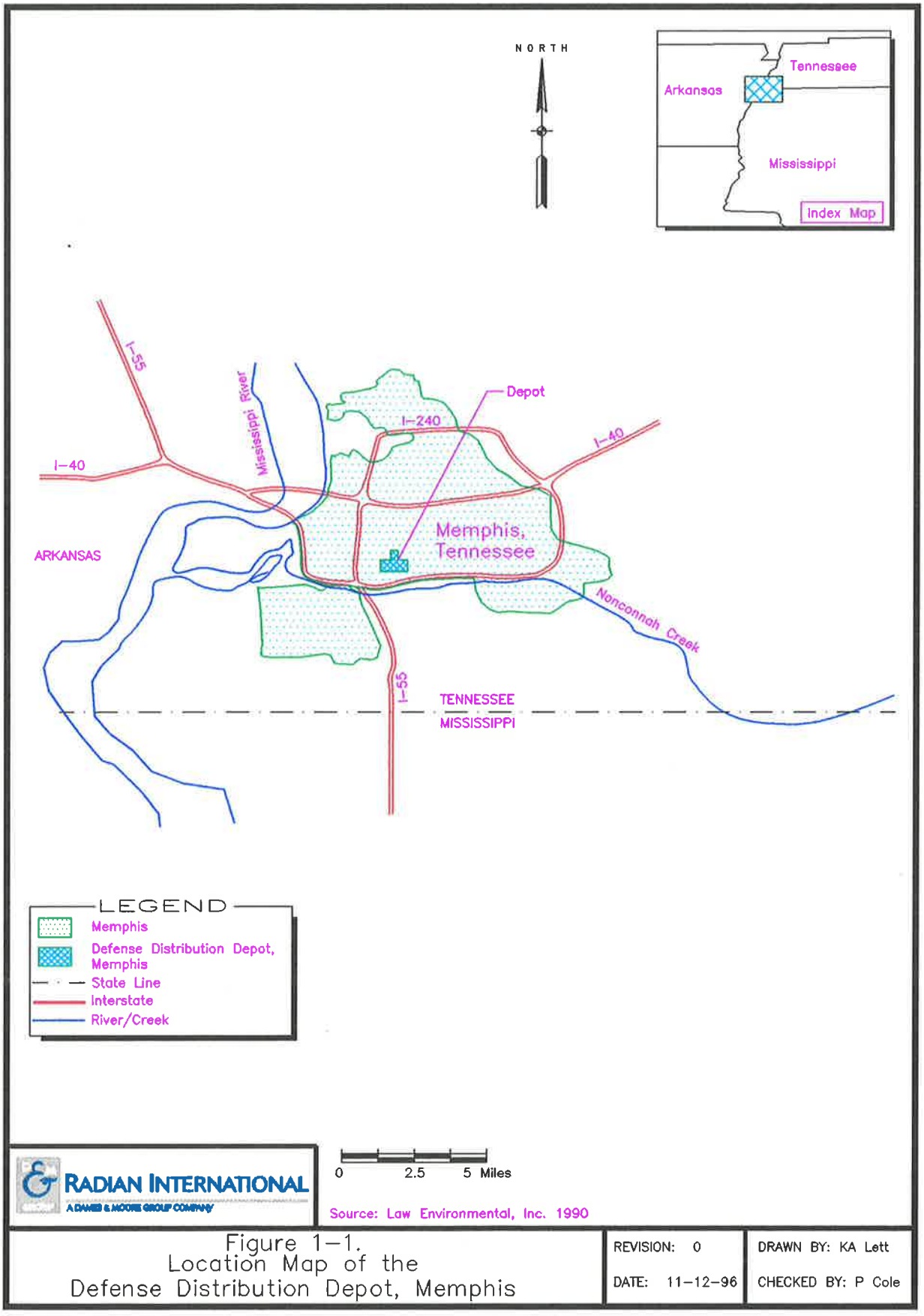
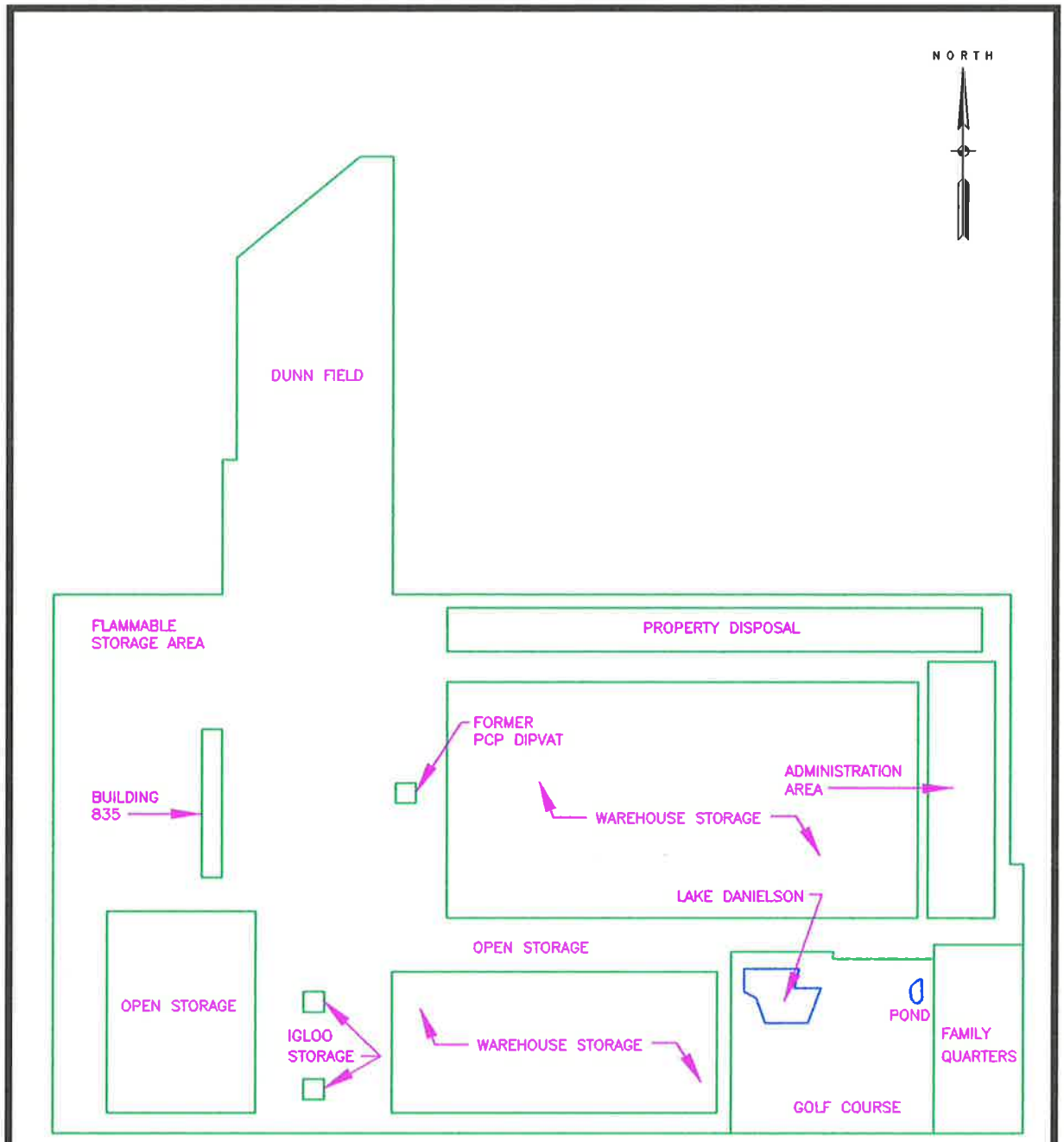


Figure 1-1.
Location Map of the
Defense Distribution Depot, Memphis

REVISION: 0
DATE: 11-12-96

DRAWN BY: KA Lett
CHECKED BY: P Cole

N:\65572401\01\SITE.DWG



RADIAN INTERNATIONAL
A DAMES & MOORE GROUP COMPANY

Source: Law Environmental, Inc. 1990

Figure 1-2.
Site Layout of the Defense Distribution Depot, Memphis

REVISION: 0	DRAWN BY: KA Lett
DATE: 11-12-96	CHECKED BY: P Cole

Lake Danielson and the golf course pond might pose a hazard to human health via ingestion of fish living in contact with the contaminated surface water/sediment. A BRA was conducted in early 1997 based on all historical data to evaluate the residual pesticide contamination in Lake Danielson and the golf course pond to determine whether remediation of sediments in those impoundments is warranted.

The following sections describe the BRA methodology that was used in early 1997 and the subsequent reevaluation of risks based on new contaminant data collected in September/October 1997 and in August 1998.

Following this introduction, Section 2.0 provides an overview of the BRA process. Section 3.0 outlines the history of the golf course impoundments' construction and use. Section 4.0 describes the investigations of the impoundments prior to 1997. Section 5.0 characterizes the exposure setting and provides the equations and input values used to quantify human health risks associated with exposure to contaminated media in the golf course impoundments. Section 6.0 summarizes the available toxicological information on the contaminants of concern. Section 7.0 presents the results of the initial human health risk characterization. Section 8.0 describes the follow-up investigation performed in September/October 1997 and presents the analytical data and risk characterization based on those new data. Section 9.0 describes the final field investigation performed in August 1998 and presents the analytical data and risk characterization based on those data. Section 10.0 discusses the various sources of uncertainty associated with the human health risk assessment. Section 11.0 evaluates potential risks to ecological receptors that might ingest contaminated prey from the golf course impoundments. Conclusions and recommendations are provided in Section 12.0. All information sources used in this BRA are referenced in Section 13.0.

This page intentionally left blank.

2.0 THE BASELINE RISK ASSESSMENT PROCESS

CERCLA requires that decisions regarding hazardous material release sites be protective of human health and the environment. Toward that end, a BRA is usually conducted to evaluate the nature and magnitude of human health and ecological risk posed by the hazardous material release site in the absence of remediation. Somewhat different approaches are used to evaluate human health risks versus ecological risks. This section discusses the human health evaluation process and the ERA screening methodology.

For a hazardous material release site to pose a risk to human health, there must be a means by which humans can come into contact with the contaminated media such that the contaminant(s) can enter the human body. Furthermore, there must be one or more modes of action by which the contaminant exerts a toxic effect on one or more organ systems of the exposed human. A conceptual site model is often used to depict the means by which a hazardous substance is released to the environment, transported to one or more environmental media (e.g., soil or groundwater), and contacted by humans via one or more exposure scenarios. The exposure scenarios are human activities that might lead to exposure and are based on current and reasonably anticipated future land use. Each exposure scenario is associated with one or more exposure pathway (i.e., the means by which an exposed individual might receive a contaminant "dose"). On-site recreation (e.g., swimming) is an example of an exposure scenario, and incidental surface water ingestion while swimming is an example of an exposure pathway. In this example, a surface water contaminant must be toxic by the oral exposure route in order for there to be a human health risk. The toxic effect might be cancer or some other adverse health effect.

The human health assessment methodology currently employed and recommended by EPA (1989) begins with a selection of those contaminants that are known to occur in the study area above background and/or health-based criteria. An exposure assessment is then performed to determine the receptors, activities, and exposure pathways that currently exist or that can reasonably be anticipated in the future at the site. Standard equations defined in applicable regulations and/or regulatory guidance are used to estimate the dose of each contaminant that a receptor might receive. Site-specific data are used when available to quantify

the dose. In the absence of site-specific data for the input variables, default values recommended in applicable regulations or regulatory guidance are used.

The estimated dose of each contaminant is then evaluated on the basis of available toxicity information for that contaminant. The reference dose (RfD) of a chemical is the chronic daily intake that is conservatively estimated to not cause adverse, noncancer health effects in even very sensitive individuals. An estimated intake that exceeds the RfD suggests that adverse, noncancer health effects may occur as a result of exposure as modeled and indicates the need for risk management.

Carcinogenic effects are evaluated by multiplying the calculated intake by a cancer slope factor that estimates the probability of developing cancer as a result of that contaminant intake. Carcinogenic effects are evaluated differently from noncancer effects, because it is believed that, in general, there is no threshold below which a carcinogenic substance does not pose some potential for causing cancer. An estimated cancer risk above one in a million (1E-6) is often used as the decision point for determining whether risk management is needed. The BRA usually concludes with a discussion of data gaps and the other sources of uncertainty inherent to the quantification of risk. The actual risk posed by contaminants at the site might be higher than the risk estimate but are usually believed to be much lower than the risk estimate when conservative assumptions are made regarding exposure conditions and toxicity.

Ecological risk can be evaluated in much the same way as human health risk, although the uncertainties associated with ERA are much greater. An ERA can focus on one or a few species that are known to occur in the area of the release site, that are highly susceptible to the contaminants of concern, and that are considered to have high ecological, economic, or societal importance. The toxic effects of concern in an ERA range from outright mortality of individual organisms to reduced reproductive success. ERA often begins with a screening process that compares on-site contaminant concentrations to toxicological benchmarks for wildlife. Toxicological benchmarks are environmental concentrations of toxicants that are

believed to be protective of specific ecological receptors. If the detected contaminant concentrations exceed the applicable toxicological benchmarks for the species of concern, a more detailed ERA analogous to the human health risk assessment might be warranted.

Risk management decisions can be made after the nature and magnitude of human health and ecological risk are estimated. Risk management for a site might involve remediation (e.g., excavation and removal of contaminated sediment), institutional controls (e.g., fencing, warning signs, deed restrictions), or other actions that serve to interrupt the transport, intake, or toxic effect of the contaminants of concern. In cases where the risks are conservatively estimated to be low and the risk management costs are expected to be high (in terms of dollars or other societal or ecological costs), the indicated course of action might be no further action.

This page intentionally left blank.

3.0 SITE DESCRIPTION

Lake Danielson and the golf course pond are the main surface water features at the Depot. Both are unlined, constructed impoundments that lie in the southeastern quadrant of the facility. Lake Danielson covers approximately 4 acres and is up to 14 ft deep in places. Lake Danielson receives surface run-off from most of the eastern half of the installation, primarily from the area around Buildings 470, 489, 490, 689, and 690. Surface run-off and direct precipitation are the only sources of water to Lake Danielson. Lake overflow is discharged through a drop inlet at the dam, via a concrete-lined channel, to a culvert extending beneath N Street and Ball Road. The culvert discharges at Outfall 004, as designated in the Depot's National Pollutant Discharge Elimination System (NPDES) permit, via unnamed tributaries to Nonconnah Creek approximately three-quarters of a mile south of the Depot. Nonconnah Creek drains into the Mississippi River at Lake McKellar.

The golf course pond is less than one-third acre in size and up to 3 ft deep. The pond receives drainage from the surrounding golf course; Buildings 249, 250, 251, 265, 270, and 271; and the south parking lot. Surface run-off and direct precipitation are the only sources of water to the pond. Pond overflow is directed to a culvert extending beneath N Street and Ball Road. The culvert discharges at Outfall 012, as designated in the Depot's NPDES permit, via unnamed tributaries to Nonconnah Creek.

Lake Danielson and the golf course pond have been used for a variety of purposes throughout the history of the Depot. Their primary function is storm water retention and sedimentation. Storm water is directed to the impoundments via swales, ditches, concrete-lined channels, and storm sewers. Most of the Depot is level with or above surrounding terrain, so the stormwater drainage system receives little or no run-off from areas outside the installation. Most of the main installation's land area has been graded, paved, and covered with buildings. The only significant vegetated area is the golf course.

Lake Danielson also serves as a fire protection reservoir, providing the required 1-hour additional fire fighting capacity beyond the 1-hour capacity provided by a 100,000-gal aboveground water storage tank. Lake Danielson was modified in the mid-1960s. A concrete/corrugated metal ("sheet piling") edge was added to stabilize and improve the appearance of the sides of the lake, and three ladders were added, probably to provide safe egress from the lake. Lake Danielson was periodically stocked with bluegill and bass. Catfish have also been observed in the lake in the past.

4.0 PREVIOUS INVESTIGATIONS

4.1 U.S. Army Environmental Hygiene Agency

Fish tissue samples (i.e., edible portions) were collected from Lake Danielson and the golf course pond and analyzed for pesticides in 1986 by the U.S. Army Environmental Hygiene Agency (AEHA). Chlordane, DDT, DDD, and DDE were detected in both sediment and fish tissue samples.

The use of DDT at the Depot was discontinued in 1980. Fishing was discontinued at Lake Danielson in 1986, and a continued ban on fishing and swimming at both impoundments was recommended in the 1990 RI Report (Law Environmental, Inc. 1990).

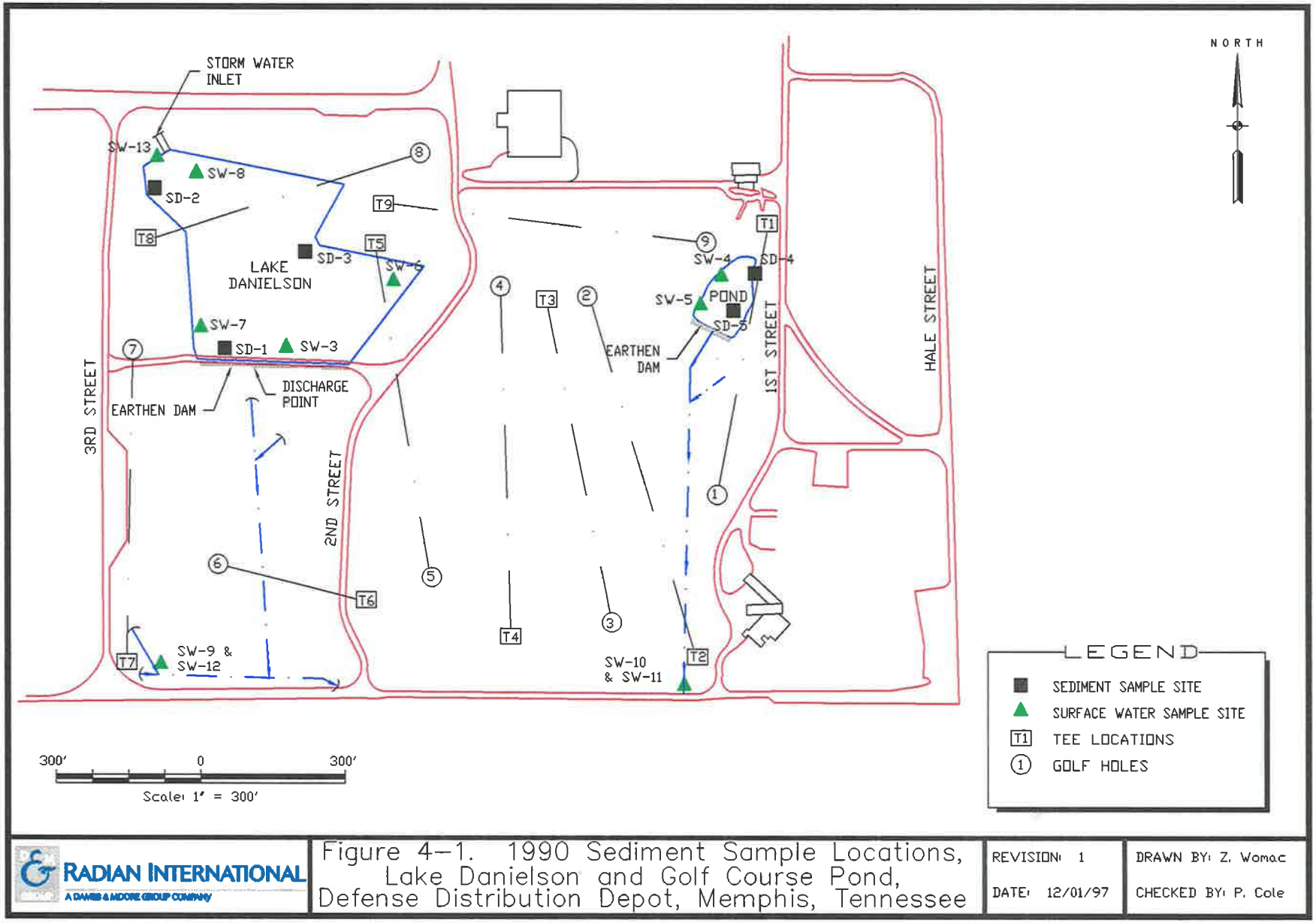
4.2 1990 Remedial Investigation

The golf course impoundments' surface water and sediment were sampled and analyzed in April 1989 and January 1990 as part of the 1990 RI. Sediment samples were collected from three locations in Lake Danielson (SD-1, SD-2, and SD-3) and two locations in the golf course pond (SD-4 and SD-5). Two sediment samples were collected from each location: one from the surface and one from a depth of 9 in. Surface water samples were also collected from Lake Danielson and from the golf course pond as part of the RI. Figure 4-1 shows the sample locations.

The only surface water sample from either impoundment that contained a detectable amount of pesticide was sample SW-7, which contained 0.21 µg/L of 4,4'-DDE. DDD and DDE were detected in two of the sediment sample locations in Lake Danielson, and the maximum detected concentration of either pesticide was 110 µg/kg of DDE in the surface sediment sample from SD-3. DDD, DDE, and DDT were detected in both sediment sample locations in the golf course pond, and the maximum detected concentration was 3000 µg/kg of DDD in the surface sediment sample from SD-5. The sediments collected were described as firm clay (Law Environmental, Inc. 1990). Table 4-1 presents the sediment data from the RI Report.

This page intentionally left blank.

N:\65574110\03\FIG4-1



RADIAN INTERNATIONAL
A DAMES & MOORE GROUP COMPANY

Figure 4-1. 1990 Sediment Sample Locations, Lake Danielson and Golf Course Pond, Defense Distribution Depot, Memphis, Tennessee

REVISION: 1
DATE: 12/01/97

DRAWN BY: Z. Womac
CHECKED BY: P. Cole

Table 4-1

1990 RI Sediment Sampling Results, Lake Danielson and Golf Course Pond

Chemical	Concentration (µg/kg)									
	SD-1-SS	SD-1-9	SD-2-SS	SD-2-9	SD-3-SS	SD-3-9	SD-4-SS	SD-4-9	SD-5-SS	SD-5-9
4,4'-DDD	47	—	—	—	45	—	190	280	3000	960
4,4'-DDE	36	—	—	—	110	—	68	64	460	—
4,4'-DDT	—	—	—	—	—	—	—	—	2900	—

— = Not detected

Background levels of DDT, DDD, and DDE in U.S. and Canadian lake and river sediments range from 0.1 to 13 µg/kg (CH2M Hill 1996). Since these pesticides are not naturally occurring substances, and they are present in the golf course impoundments' sediment above background levels, all three compounds were evaluated quantitatively in the early 1997 BRA.

4.3 Contaminant Fate and Transport

DDD and DDE are degradation products of DDT, and all three compounds have similar properties. All are relatively insoluble in water and adsorb readily onto soil particles, so they tend to persist in soils and sediments. The presence of DDT, DDD, and DDE in the golf course impoundments' sediment is probably due to the past practice of direct application of these pesticides during routine golf course maintenance. Pesticides applied to the golf course and other parts of the Depot were likely transported to the golf course impoundments via soil particles in surface run-off. The low solubility of these compounds is the likely reason for the observed low concentrations in surface water samples. Leaching to groundwater is not likely to occur due to the low solubility of the pesticides (Law Environmental, Inc. 1990).

5.0 EXPOSURE SETTING

This section describes the exposure assessment that was used for the early 1997 BRA, the updated BRA based on data collected in September/October 1997, and the final assessment based on data collected in August 1998.

Land use in the area surrounding the Depot is a mixture of residential, commercial, and manufacturing establishments. The population for the Depot's zip code area is 40,352 according to the 1990 census. Several large, multifamily developments are in the area, ranging from an older apartment complex (Castalia Heights Apartments) located north of the Depot along Carver Avenue and Keltner Circle, to a newer development (Orchid Manor) located to the south of the Depot on Ball Road. Several schools are within 1.5 miles of the Depot. Dunn Elementary, Corry Junior High, and Alcy Road Elementary are within 0.5 mile of the Depot. Charjean Elementary, Airways Junior High, and Hamilton Elementary are within 1 to 1.5 miles of the Depot. Two neighborhood parks, Alcy Samuels Park and Lincoln Park, are in the vicinity of the Depot. No other sensitive land uses or receptors occur in the vicinity of the Depot (Law Environmental, Inc. 1990).

The Depot property is zoned light industrial, as are several contiguous parcels. With the exception of the golf course, most of the main installation is paved or covered with buildings, primarily warehouses and covered storage areas. Future land use on the installation is likely to remain industrial and/or commercial. The golf course is anticipated to remain in its current use.

The pesticide contamination in the golf course impoundments' sediment is unlikely to leach into surface water or groundwater, due to the low solubility of the pesticides and their strong affinity for soil and sediment particles. The sediments are covered with several feet of water, so direct human exposure to the sediments is unlikely to occur under current and reasonably anticipated future conditions. Swimming and fishing in the impoundments are likely to continue to be prohibited in the future. However, it is conceivable that an adolescent/teenage individual might gain unauthorized access to the ponds for swimming, wading, or fishing.

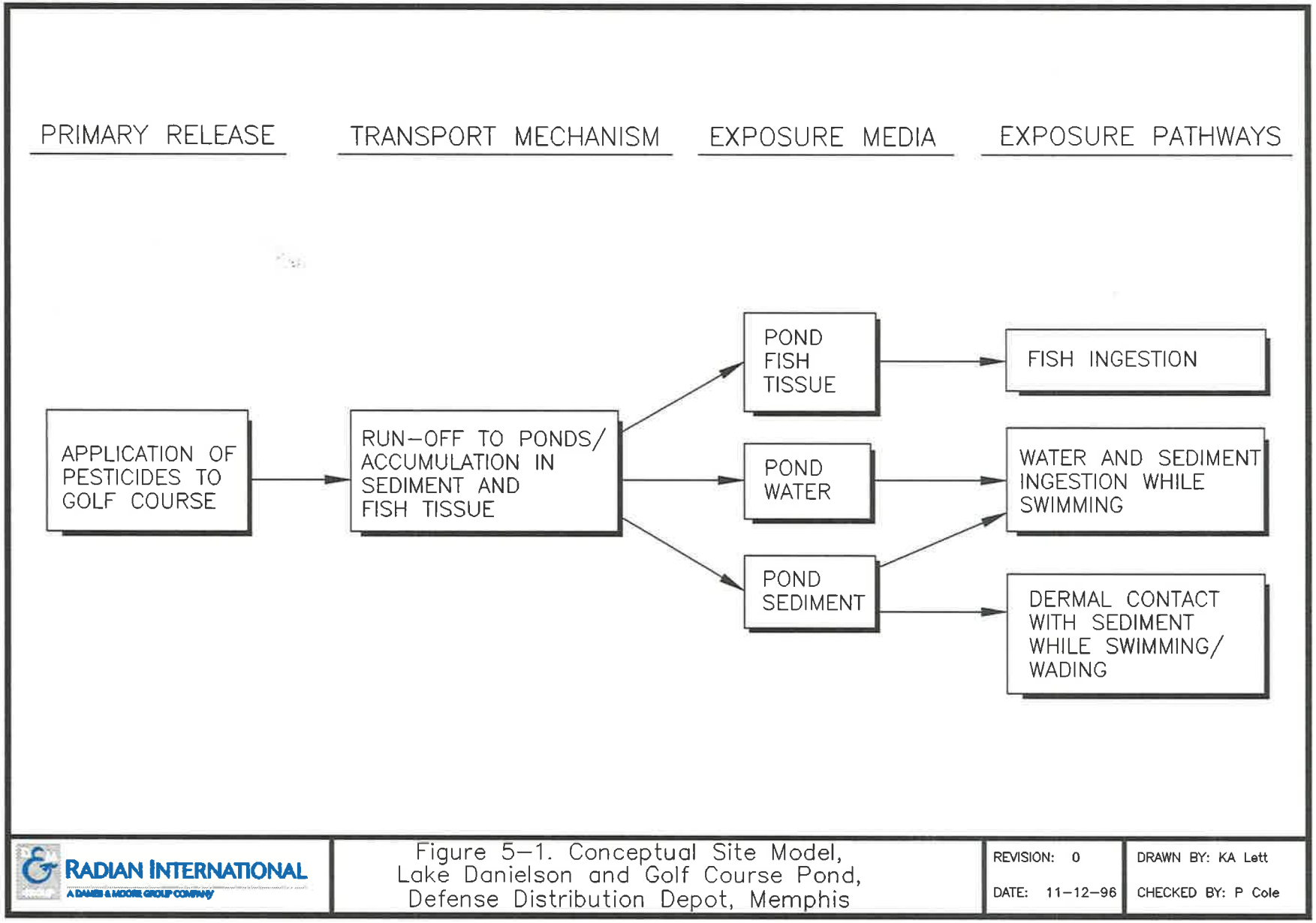
The exposure scenario used to quantify human health risk involves a male youth who gains unauthorized access to swim and fish in the impoundments. He was assumed to swim in the impoundments for 1 hour/day, 5 days/week during the summer months from the age of 13 to 18, attempting to retrieve golf balls from the bottoms of the impoundments. It was assumed that his hands and feet become covered with sediment in the process of attempting to retrieve golf balls. It was further assumed that a considerable amount of sediment becomes suspended in the water column while he swims and dives for golf balls. He was assumed to swallow a small amount of water containing suspended sediment while swimming and diving. He was assumed to be able to catch and eat catfish from the impoundments. Figure 5-1 is a conceptual site model diagram that summarizes the contaminant release mechanism, environmental transport mechanisms, exposure media, and exposure pathways that apply to the golf course impoundments.

The exposure duration and the age and gender of the receptor were chosen on the basis of the risk assessor's personal observation of behavior patterns. It seems that male youths are more likely than female youths to gain unauthorized access for recreational purposes. Before the age of 13, parental supervision tends to be greater, averting the opportunity for such activities. After the age of 18, other pastimes are likely to replace swimming and fishing to a large degree.

The mean skin surface area of the hands and feet of males age 13 to 18 was used as the contact area for sediment exposure (EPA 1990). The adherence factor recommended by EPA (1989) for kaolin clay was used to account for the amount of sediment that would adhere to the skin. The adsorption factor recommended by Ryan et al. (1987) for organic compounds was used to account for the amount of pesticide that would be transferred from the sediment to the receptor's blood through the skin. The mean body weight of males age 13 to 18 was used in the calculations of pesticide intake (EPA 1990).

The maximum concentration of each pesticide detected in any sediment sample collected in 1986 from the impoundments was used as the concentration to which the receptor would be exposed in the early 1997 BRA. Likewise, the maximum concentration of each pesticide detected in any sediment sample from the September 1997 sampling event was used as

N:\65572401\01\FIG4-1.DWG



RADIAN INTERNATIONAL
A DANIEL & MOORE GROUP COMPANY

Figure 5-1. Conceptual Site Model, Lake Danielson and Golf Course Pond, Defense Distribution Depot, Memphis

REVISION: 0
DATE: 11-12-96

DRAWN BY: KA Lett
CHECKED BY: P Cole

the representative exposure concentration for the updated BRA. EPA (1989) recommends the use of the 95% upper confidence limit (95 UCL) on the mean of the data set as the representative exposure concentration. However, the data sets for the impoundments' sediment are small and exhibit a high degree of variability, so the 95 UCL may be higher than the maximum detected concentration.

The amount of sediment suspended in the water column was assumed to be approximately 10 parts per million (ppm), which is very turbid water, so the maximum concentration of each pesticide was divided by 100,000 to estimate the pesticide concentration in water. The water ingestion rate recommended by EPA (1989) for contaminant exposure while swimming was used in the calculations of pesticide intake.

To quantify risks associated with ingestion of fish from the golf course impoundments, the same hypothetical youth was assumed to be able to catch and eat catfish from the impoundments as an activity independent of swimming. The catfish tissue pesticide data from the AEHA investigation (1986) were used as the representative exposure concentrations in fish. The fish ingestion rate (6.5 g/day) recommended by EPA (1989) as the mean annual per capita fish consumption rate for the United States was used along with an assumed exposure frequency of 365 days/year and an exposure duration of 6 years to quantify pesticide intake via ingestion of fish from the golf course impoundments. It was assumed that all fish tissue ingested was caught from the golf course impoundments, so a value of one was used for the fraction ingested variable.

The following equations and parameters were used to quantify contaminant intake:

Dermal Exposure to Sediment

$$\text{Absorbed Dose (mg/kg/d)} = (CS \times CF \times SA \times AF \times ABS \times EF \times ED)/(BW \times AT)$$

where:
- CS = chemical concentration in sediment (mg/kg),
- CF = conversion factor (1E-6 kg/mg),
- SA = surface area available for contact (cm^2/event),
- AF = sediment to skin adherence factor (mg/cm^2),
- ABS = absorption factor (unitless),

AF = sediment to skin adherence factor (mg/cm^2),
ABS = absorption factor (unitless),
EF = exposure frequency (events/year),
ED = exposure duration (years),
BW = body weight (kg),
AT = averaging time (period over which exposure is averaged, days).

Ingestion of Water and Sediment While Swimming

$$\text{Intake (mg/kg/d)} = (CW \times CR \times ET \times EF \times ED)/(BW \times AT)$$

where:
CW = chemical concentration in water (mg/L),
CR = contact rate (L/hour),
ET = exposure time (hours/event),
EF = exposure frequency (events/year),
ED = exposure duration (years),
BW = body weight (kg),
AT = averaging time (days).

Fish Ingestion

$$\text{Intake (mg/kg/d)} = (CF \times IR \times FI \times EF \times ED)/(BW \times AT)$$

where:
CF = contaminant concentration in fish (mg/kg),
IR = ingestion rate (kg/day),
FI = fraction ingested from contaminated source (unitless),
EF = exposure frequency (days/year),
ED = exposure duration (years),
BW = body weight (kg),
AT = averaging time (days).

For many noncarcinogenic effects, protective mechanisms are believed to exist that must be overcome before the adverse effect is manifested. For example, where a large number of cells perform the same or similar function, the cell population may have to be significantly depleted before the effect is seen. As a result, a range of exposures exists from zero to some finite value that can be tolerated by the organism with essentially no chance of expression of adverse effects. Because variability exists in the human population with regard to what that threshold is, attempts are made to identify a sub-threshold level protective of sensitive individuals in the population. This sub-threshold level is the RfD, expressed as a chronic daily

intake in mg of chemical per kg of body weight averaged over the number of days in the period of exposure. Thus, the averaging time variable used in the calculation of noncarcinogenic chemical intake is equal to the exposure duration in years multiplied by 365 days/year.

Carcinogenesis is generally thought to be phenomenon for which risk evaluation based on presumption of a threshold is inappropriate. For carcinogens, EPA assumes that a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation and eventually to a state of disease. This mechanism is referred to as "nonthreshold" because there is believed to be essentially no level of exposure to such a chemical that does not pose a finite probability, however small, of generating a carcinogenic response. Therefore, the toxicity of carcinogens is expressed as a cancer slope factor, which is the probability of cancer induction per unit intake. The unit intake is expressed as mg of chemical per kg of body weight averaged over a 70-year lifetime. Since carcinogens are believed to exert a toxic response anytime during an exposed individual's lifetime after the period of exposure, the averaging time variable for calculating carcinogenic chemical intake is equal to 365 days/year multiplied by an assumed 70-year lifetime.

This page intentionally left blank.

6.0 TOXICITY ASSESSMENT

This toxicity assessment summarizes the currently available information on the modes and magnitude of toxic action of DDD, DDE, DDT, chlordane, dieldrin, and heptachlor epoxide. The complete toxicity report from EPA's Integrated Risk Information System (IRIS) for each pesticide is provided in Appendix A.

6.1 4,4'-DDD, 4,4'-DDE, and 4,4'-DDT

DDT is a man-made compound that was widely used as an agricultural insecticide and to control disease carrying insects. DDD and DDE are common contaminants and metabolic products of DDT. DDD was also used to kill pests and as a chemotherapeutic agent in the treatment of adrenal cancer. DDT may no longer be used in the United States except in the case of public health emergencies to control disease vectors. It is still used regularly in other parts of the world. Because people are not typically exposed to DDT, DDD, or DDE individually, but rather to a mixture of all three, the toxicities of these compounds should be considered jointly [Agency for Toxic Substances and Disease Registry (ATSDR) 1994].

6.2 4,4'-DDD CAS No. 72-54-8

A NOAEL of 26 mg/kg-day was identified during short-term exposure (1 week) of mice to 4,4'-DDD in the diet. Exposure of rats to 1221 mg/kg-day of 4,4'-DDD for 16 days resulted in atrophy of the thymus. NOAELs of 165 and 107 mg/kg-day were identified in chronic studies (78 weeks) using rats and mice, respectively. However, at 85 mg/kg-day, exposure to 4,4'-DDD resulted in thyroid tumors in rats. In a separate study, exposure to 32.5 mg/kg-day of 4,4'-DDD caused lung tumors in mice (ATSDR 1994).

Neither EPA's IRIS nor the Health Effects Assessment Summary Table (HEAST) lists an oral RfD, inhalation RfD, or inhalation reference concentration (RfC).

4,4'-DDD is a Group B2–Probable Human Carcinogen. This classification is based on the induction of lung tumors in male and female mice, liver tumors in male mice, and

thyroid tumors in male rats. There are no human carcinogenicity data. The oral slope factor, as given by IRIS, is 2.4E-01 $(mg/kg\text{-}day)^{-1}$. The supporting study used an adequate number of animals, but the slope factor was derived using tumor incidence data from one dose. There is no inhalation unit risk at this time.

6.3 4,4'-DDE CAS No. 72-55-9

The health effects resulting from exposure of animals to 4,4'-DDE in water are not known. Exposure of mice (by gavage) to 26 mg/kg-day of 4,4'-DDE for 24 hours/day for one week caused alterations in the liver. When rats were exposed to 28 mg/kg-day of 4,4'-DDE by gavage on gestation days 15–19, a decrease in the weight of the ovaries was noted. A NOAEL of 42 mg/kg-day was identified in a long-term (78 weeks) study in which rats were fed 4,4'-DDE in the diet. Hamsters fed 41.5 mg/kg-day of 4,4'-DDE for 128 weeks exhibited necrosis of the liver, and when 4,4'-DDE was administered by gavage, tumors of the liver were observed. When mice were exposed to 19 mg/kg-day of 4,4'-DDE in the diet for 78 weeks, liver tumors were also observed. There is no RfD or RfC for DDE in IRIS or HEAST (ASTDR 1994).

4,4'-DDE is classified as a Group B2–Probable Human Carcinogen. This classification is based on increased incidence of liver tumors, including carcinomas in two strains of mice and in hamsters and thyroid tumors in female rats when 4,4'-DDE is given in the diet. Human data are not available. The oral slope factor is 3.4E-01 $(mg/kg\text{-}day)^{-1}$. This value is the geometric mean of six slope factors computed from incidence data by sex. There is no inhalation slope factor for DDE.

6.4 4,4'-DDT CAS No. 50-29-3

The primary effect of short-term exposure to high levels of 4,4'-DDT is on the nervous system. Oral ingestion of large quantities of 4,4'-DDT has resulted in excitability, tremors, and seizures in humans. Irritation of the eyes, nose, and throat has been reported by people who have come in contact with 4,4'-DDT. Exposure to low doses of DDT on a long-term basis has resulted in changes in the levels of liver enzymes involved in metabolism of drugs and chemicals, but there was no indication that 4,4'-DDT caused irreversible damage (ATSDR 1994).

Studies conducted in laboratory animals suggest that exposure to 4,4'-DDT may have harmful effects on reproduction and may result in an increased occurrence of liver tumors. However, five studies of 4,4'-DDT exposure in humans did not show increases in the number of deaths or cancers (ATSDR 1994). Increasing evidence indicates that pesticides, including 4,4'-DDT, can alter immune function in rodents, although studies in humans are limited and ambiguous. In a study of pesticide formulators in India, 73% of workers exposed to 4,4'-DDT had altered levels of serum immunoglobulins, although no increase in infections was noted.

The oral RfD for 4,4'-DDT is listed in IRIS as 5E-04 mg/kg-day. This value is based on a chronic rat feeding study in which 4,4'-DDT was provided in the diet. Weanling rats were fed commercial DDT in doses of 0, 1, 5, 10, or 50 ppm for 15 to 27 weeks. Increasing hepatocellular hypertrophy was seen at doses of 5 ppm and greater. Therefore, 5 ppm was established as a Lowest Observed Adverse Effects Level. A NOAEL of 1 ppm (converted to 0.05 mg/kg-day) was also established in the study. An uncertainty factor of 100 was used to account for interspecies conversion and to protect sensitive human subpopulations (10x each). An uncertainty factor for subchronic to chronic conversion was not included because of corroborating chronic data in the data base. A confidence rating of medium was associated with the RfD and reflects that the principal study was adequate but of shorter duration than desired. There are no values for the inhalation RfD or RfC at this time. HEAST lists the subchronic oral RfD as 5.0E-04 mg/kg-day.

4,4'-DDT is classified as a Group B2–Probable Human Carcinogen. This classification is based on tumors (usually liver) in various mouse strains and three rat studies. Human carcinogenicity data are inadequate. The oral slope factor listed in IRIS is 3.4E-01 $(mg/kg\text{-}day)^{-1}$. The inhalation unit risk is listed in IRIS as 9.7E-05 $(mg/m^3)^{-1}$.

6.5 Chlordane

Chlordane is a member of a class of chlorinated hydrocarbon pesticides called cyclodienes and has two main isomers (*cis* and *trans*). *Cis*-chlordane (alpha-chlordane) is more abundant than *trans*-chlordane (gamma-chlordane). In addition to the two chlordane isomers,

technical grade chlordane may also contain heptachlor, nonachlor, hexachlorocyclopentadiene, and other compounds (ATSDR 1994).

The health effects of chlordane are similar to other chlorinated hydrocarbon insecticides, especially other cyclodienes. The central nervous system is affected by inhalation of chlordane. Headaches, dizziness, vision problems, incoordination, irritability, excitability, weakness, muscle twitching, and convulsions have been reported in humans exposed acutely to chlordane via inhalation. Acute inhalation of chlordane may also cause respiratory irritation and congestion and gastrointestinal effects such as cramps, diarrhea, and nausea. Chronic exposure to chlordane has resulted in migraines, neuritis, and neuralgia. Chronic inhalation of chlordane may cause blood dyscrasias, adverse hepatic effects, and adverse reproductive effects. Available human data with regard to these effects is of limited use due to the fact that patients were not exposed solely to chlordane in most instances. Immunological effects have been observed in humans exposed to chlordane via inhalation. Adverse effects were seen in kidneys of animals exposed to chlordane by inhalation (ATSDR 1994).

Oral ingestion of chlordane affects the central nervous system in humans. Ataxia, headache, dizziness, irritability, excitability, confusion, incoordination, muscle tremors, seizures, convulsion, and coma have been noted with acute human oral exposure to chlordane. Oral ingestion of chlordane may also cause gastrointestinal effects such as nausea, cramps, and diarrhea. Hepatic, reproductive, and developmental effects have been observed in animals administered chlordane orally.

Dermal exposure to chlordane may result in systemic effects, including central nervous system effects. Burning of the skin, rashes, and pruritus have been reported in humans who were exposed to chlordane dermally. Conjunctivitis has been reported with accidental application of chlordane to the eyes.

The chronic RfD for chlordane is listed in IRIS as 6E-05 mg/kg-day. This is based on a chronic rat study using doses of 0, 1, 5, and 25 ppm technical grade chlordane in the diet. Clinical laboratory studies were performed and organ weights measured on eight animals/sex/group at 26 and 52 weeks, and on all survivors at 130 weeks. Gross and microscopic

pathology were performed on all tissues. Daily dose levels of 0.045, 0.229, and 1.175 mg/kg-day for males and 0.055, 0.273, and 1.409 mg/kg-day for females for the 1, 5, and 25 ppm treatment groups, respectively, were derived from food consumption and body weight data. It was concluded that liver hypertrophy occurred in female rats at 5 ppm, which was considered the lowest effect level. A NOAEL of 1 ppm was established. HEAST lists a subchronic RfD for chlordane as 6E-05 mg/kg-day.

An uncertainty factor of 1000 was used to derive the chronic oral RfD for chlordane. A factor of 100 was used to account for the inter- and intra-species differences (10 each). A factor of 10 was used to account for a lack of a second mammalian species, lack of chronic exposure data, and an insufficiently sensitive endpoint. These uncertainties resulted in a low confidence level. There are no values for the inhalation RfD or RfC at this time (IRIS 1999).

Chlordane is a Group B2–Probable Human Carcinogen. This classification is based on the development of benign and malignant liver tumors in four strains of mice (both sexes) and in male rats. This compound is also structurally related to other liver carcinogens. Human carcinogenicity data are inadequate. An oral slope factor is listed in IRIS as 1.3E+00 $(mg/kg\text{-}day)^{-1}$. Liver tumors were induced in mice of both sexes in two studies, an adequate number of animals was observed and dose response effects were reported. The inhalation unit risk is listed in IRIS as 3.7E-04 $(Fg/m^3)^{-1}$. HEAST lists an inhalation slope factor based on route to route extrapolation for chlordane as 1.3E+00 $(mg/kg\text{-}day)^{-1}$.

6.6 Dieldrin

Dieldrin is an agricultural insecticide that is no longer used in the United States. It was used extensively from the 1950s until its use was banned by the U.S. Department of Agriculture in 1970. EPA did allow the use of dieldrin to kill termites from 1972 to 1987. In 1987, the manufacturer of dieldrin voluntarily canceled the registration for use of dieldrin in controlling termites. In its pure form, dieldrin is a white powder that will evaporate slowly with a mild chemical odor. Technical grade dieldrin is a tan powder. Dieldrin is a product of aldrin degradation in the environment and in the body (ATSDR 1991).

Dieldrin is lipid-soluble and stored in adipose tissue of humans and other animals. Aldrin and dieldrin cause similar adverse health effects. No increase in mortality from any cause has been reported in workers who have been employed in the manufacture of dieldrin for more than 4 years. However, long-term exposure to moderate levels of dieldrin causes headaches, dizziness, irritability, vomiting, or uncontrollable muscle movements. Central nervous system excitation culminating in convulsions was the principal toxic effect noted in occupational studies of workers employed in the manufacture or application of dieldrin. Short-term exposure to high levels of dieldrin causes convulsion and kidney damage. Long-term exposures to lower levels may also cause convulsions as a result of the potential for dieldrin to accumulate within the body (ATSDR 1991).

The carcinogenic and reproductive/developmental effects of dieldrin in humans are currently unknown. Experimental studies indicate that animals born to mothers that were fed dieldrin do not live long. One study revealed detectable levels of dieldrin in the human placenta, amniotic fluid, and fetal blood. These results suggest that dieldrin can pass through the human placenta and accumulate in the developing fetus (ATSDR 1991).

The oral RfD for dieldrin is listed in IRIS as 5E-05 mg/kg-day. This value was based on a chronic (2-year) rat feeding study. The critical effect noted in the study was liver lesions. HEAST lists a value of 5.00E-05 mg/kg-day for the subchronic oral RfD.

The uncertainty factor used to derive the oral RfD for dieldrin is 100. This factor allows for the extrapolation of dose levels from animals to humans and the uncertainty in the threshold for sensitive humans. The confidence level for the RfD value is medium. The principal study is an older study for which detailed data are not available. The chronic toxicity evaluation is relatively complete and supports the critical effect. The RfD is given a medium confidence rating based on support for the critical effect from other dieldrin studies. Confidence in the study is low. However, confidence in the database is medium (IRIS 1996).

Dieldrin is a Group B2–Probable Human Carcinogen. This is based on the fact that dieldrin is carcinogenic in seven strains of mice when given orally. It is also structurally similar to aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid, which are

tumorgens. The oral slope factor listed by IRIS is 1.6E+1 $(mg/kg\text{-}day)^{-1}$ and is the geometric mean of 13 slope factors calculated from liver carcinoma data in both sexes of several strains of mice. The inhalation unit risk listed by IRIS is 4.6E-03 mg/m^3, based on oral data. HEAST lists a value of 1.6E+01$(mg/kg\text{-}day)^{-1}$ for the inhalation slope factor.

6.7 Heptachlor Epoxide

Upon entering the body, heptachlor is metabolized to heptachlor epoxide and other related chemicals. Heptachlor epoxide is more harmful than heptachlor, primarily because of its ability to be stored in fat for long periods of time. The breakdown products of heptachlor epoxide are generally are less toxic. Long-term exposure to heptachlor epoxide may adversely affect the liver. Animals fed heptachlor epoxide in an experimental setting have been reported to have enlarged livers, liver damage, kidney damage, and increased red blood cell count.

Placental transfer of heptachlor epoxide has been reported following inhalation exposure. Heptachlor epoxide has also been identified in breast milk. This compound has been detected in stillborn infant brain, adrenal, lung, heart, liver, kidney, spleen, and adipose tissues. However, the studies reporting these findings were limited by lack of data concerning route, duration, extent of exposure, and number of cases examined. No gross malformations were reported in any of the stillborn infants. Although a developing fetus could be exposed to heptachlor epoxide transplacentally, the existing data are inadequate to establish a relationship between exposure and human developmental toxicity (ATSDR 1992).

The oral RfD for heptachlor epoxide is listed as 1.3E-05 mg/kg-day in IRIS. This value is based on a chronic feeding study conducted in dogs fed diets containing 0, 0.5, 2.5, 5, or 7.5 ppm of heptachlor epoxide for 60 weeks. The critical effect noted in the study was treatment-related increases in liver-to-body weight ratios. Effects were noted in both males and females and a lowest effect level of 0.5 ppm was established. A no observed effect level (NOEL) was not established in this study.

An uncertainty factor of 1000 was used to account for inter- and intra-species differences and because a NOEL was not established in the study. The confidence associated with the oral RfD was low, reflecting that the principal study was of low quality and that the data base on chronic toxicity is complete but consists of low quality studies. The subchronic RfD listed in HEAST is the same as the chronic RfD (1.3E-05 mg/kg-day) listed in IRIS.

Heptachlor epoxide is classified by EPA as Group B2–Probable Human Carcinogen. Sufficient evidence exists from rodent studies in which liver carcinomas were induced in two strains of mice of both sexes and in female rats. It is also structurally similar to several other liver carcinogens. There are no published epidemiologic evaluations of heptachlor epoxide. The oral slope factor listed in IRIS is 9.1E+00 $(mg/kg\text{-}day)^{-1}$. An inhalation unit risk of 2.63E-03 mg/m^{-3} was calculated from oral data. HEAST lists a value of 9.1E+00 $(mg/kg\text{-}day)^{-1}$ for the inhalation slope factor.

7.0 INITIAL RISK CHARACTERIZATION

Table 7-1 presents the results of the initial human health risk quantification. Appendix B contains the spreadsheet used to calculate pesticide intake and subsequent risk.

Table 7-1

Cancer Risk Estimates for Lake Danielson and Golf Course Pond Based on 1990 RI Data

Contaminant	Dermal Exposure	Sediment Ingestion	Fish Ingestion
DDD	9.98E-08	8.79E-11	1.07E-05
DDE	3.21E-09	2.82E-11	1.58E-05
DDT	1.37E-07	1.2E-10	3.13E-06
	Total Pathway Risk	**Total Pathway Risk**	**Total Pathway Risk**
	2.44E-07	2.4E-10	2.96E-05

Dermal exposure and ingestion of sediment while swimming were found to pose negligible degrees of cancer risk, according to the modeled exposure. The daily absorbed dose of DDT by the dermal exposure pathway was estimated to be 4.02E-07 mg/kg-day, and the chronic daily intake of DDT via sediment ingestion while swimming was estimated to be 1.2E-10 mg/kg-day. Both values are well below the RfD of 5E-04 mg/kg-day for DDT, so adverse noncancer health effects are not expected to occur as a result of the modeled exposure to DDT. No RfD values are available for DDD or DDE.

The total pathway cancer risk (i.e., the combined risk for all three pesticides) for fish ingestion was estimated to be 2.96E-05. This degree of cancer risk is within the range of Superfund site remediation goals in the National Contingency Plan [CFR 300.430(e)(2)(I)(A)(2)] (i.e., 1E-04 to 1E-06). The chronic daily intake of DDT via fish ingestion was estimated to be 9.2E-06 mg/kg-day, which is well below the RFD of 5E-04 mg/kg-day for DDT, so adverse noncancer health effects are not expected to occur as a result of the modeled exposure to DDT. As previously stated, no RFD values are available for DDE or DDD.

The May 1997 BRA (Radian 1997) concluded that the majority of the human health risk associated with the golf course impoundments was attributable to ingestion of pesticide residues that might be present in fish in the ponds. However, the current existence of

edible fish species in the ponds was uncertain. Furthermore, pesticide concentrations in fish and/or sediment appeared to be highly variable (based on 1986 and 1991 data) and may have changed since the previous investigations. The BRA recommended that additional sediment and fish samples be collected and analyzed while assessing the current condition of fish populations in the golf course impoundments. The new data could then be used to reevaluate the human health risks associated with exposure to pesticides in the impoundments. The recommended sampling was conducted in September/October 1997.

8.0 FOLLOW-UP INVESTIGATION

Fish and sediment sampling was conducted at the golf course impoundments beginning on 29 September and ending on 2 October 1997. The weather was sunny during the entire sampling event, with temperatures around 70°F.

Fish sampling was attempted before collecting sediment samples to avoid disturbing the fish (making them harder to catch) and to avoid suspending sediment that might further contaminate any fish that might be present. Several fishing methods and bait types were used. On the first day of the sampling event, four individuals spent a total of approximately 24 hours (an average of 6 hours of fishing per person) angling in Lake Danielson. Spin casters and cane poles were used together with live earthworms, crickets, and beetles; plastic worms, grubs, and lizards of various colors; chicken blood catfish dough; Uncle Ben's catfish bait; Worden's rooster tails; and Panther Martin and Mepps lures.

Several large golden shiners (*Notropus girardi*) were caught throughout the day, but no other fish species were caught or observed. No surface activity indicative of the presence of other fish species was observed.

The shiners ranged in length from 5¼ to 7 in., and the total weight of the 13 shiners caught on the first day was approximately 1 lb. The 13 fish were each rinsed in distilled water, and they were wrapped together in aluminum foil as a single, composite sample labeled "Fish Sample No. 1." The sample was placed into a freezer at the end of the first day of sampling.

On the second day of sampling, approximately 225 meters of commercial trot line was strung across Lake Danielson about 1/3 of the way from the south end of the lake, anchored on the dam and at a point jutting into Lake Danielson from the opposite side. The 48 trot line hooks were baited with shrimp, cut shad, and night crawlers. Empty plastic water bottles were attached to the trot line near each end and in the middle to serve as floats. Lead sinkers were attached to the trot line about every 15 yards. The trot line was left in place for approximately 48 hours.

Also on the second day a wire catfish trap, 19 in. in diameter and 60 in. long with 1-in. square mesh, was baited with cottonseed meal cake and placed into Lake Danielson near the dam (west wall) approximately 1/3 of the way from the south end of the lake. The trap was left in place overnight, with the open end facing such that fish swimming clockwise would encounter the open end.

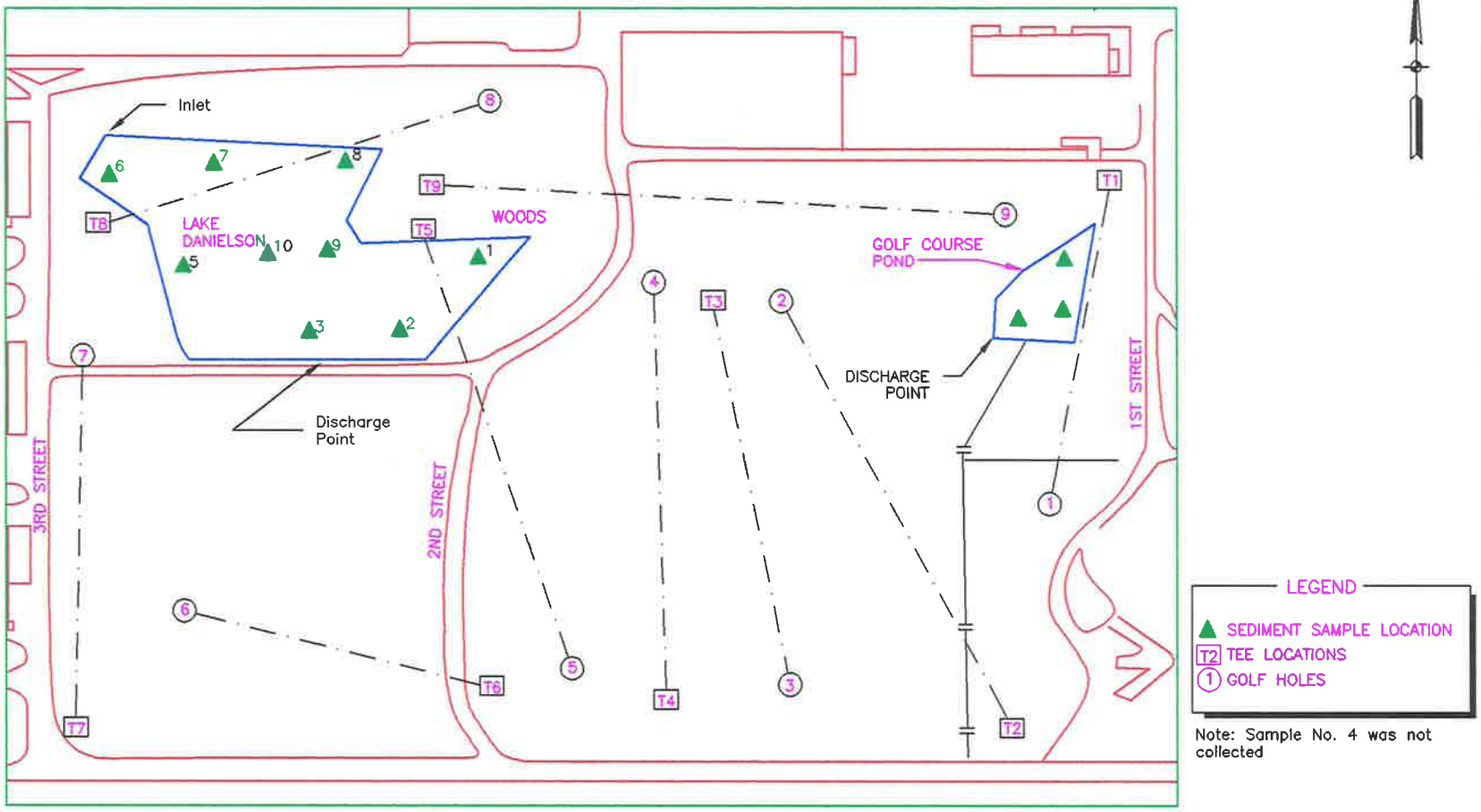
The trot line and catfish trap were checked on the morning of the third day of sampling. The trap contained several golden shiners but no other fish species or other aquatic organisms. All live fish (24 individuals weighing a total of approximately 2 lb) were rinsed with distilled water and wrapped in aluminum foil as a single sample. The sample was labeled as Fish Sample No. 2 and placed into a freezer.

Nothing had been captured by the trot line. The trot line was rebaited and left in place. The trap still contained bait and was also left in place.

All sediment samples were collected on the third day of sampling. A Petit Ponar stainless steel clamshell dredge was used to collect samples of sediment from the bottoms of both ponds. The approximate sample locations are shown in Figure 8-1. When possible, sediment samples were collected while standing on the sides of Lake Danielson. A few samples had to be collected by lowering the dredge from within a canoe. Nine of the 10 planned samples were collected from Lake Danielson. Sample No. 4 could not be collected due to an apparently thick layer of crushed rock lying on the bottom of Lake Danielson at that location. Three sediment samples were collected from the golf course pond by lowering the dredge from within a canoe.

When collecting sediment samples, the dredge was carefully lowered by hand from the end of a rope. The release of pressure when the dredge encountered the bottom would cause the discharge of a spring-loaded pin, allowing the dredge to close, encasing a portion of the material on the bottom of the ponds. In many cases, leaves from the trees surrounding the ponds would represent the majority of the material captured by the dredge. Repeated attempts were sometimes necessary to obtain an appropriate and adequate sample of sediment. Even after repeated attempts, Sediment Sample Location No. 2 yielded mostly leaf litter. The analytical

N:\65572410\01\FIG4-1.DWG



Source: Law Environmental, Inc. 1990

RADIAN INTERNATIONAL
A DAMES & MOORE GROUP COMPANY

Figure 8-1. 1997 Sediment Sample Locations
Lake Danielson and Golf Course Pond
Defense Distribution Depot, Memphis

REVISION: 0
DATE: 11-12-96

DRAWN BY: KA Lett
CHECKED BY: P Cole

laboratory was directed to sieve the leaves from the sediment samples before analyzing the sediment portion. The small amount of sediment obtained at Sample Location No. 2 resulted in higher detection limits for that sample.

Each sediment sample was transferred from the dredge to a clean, stainless steel bowl and mixed thoroughly with a clean, stainless steel spoon. The sample was then packed into a clean, wide-mouth glass jar provided by the analytical laboratory. The jar was immediately labeled, sealed with custody tape, and placed into a cooler with ice. All samples were kept in the custody of the sampling team or locked in the vehicle, until transferring the samples to the custody of Federal Express for shipment to the analytical laboratory.

Before and after collecting each sediment sample, the dredge, bowl, and spoon were decontaminated by washing with a tap water/low phosphate detergent solution, rinsing with tap water, rinsing with isopropanol, rinsing with distilled water, and air drying. A rinsate blank was collected to evaluate the effectiveness of decontamination. The rinsate blank was obtained by pouring distilled water over the decontaminated dredge into the decontaminated stainless steel bowl and transferring the water directly to a glass jar provided by the analytical laboratory. The rinsate blank was analyzed for pesticides. All results were below the detection limit of 10 µg/L.

On the fourth day of the sampling event, the trot line and trap were checked in the morning. No fish had been captured by the trot line, so it was removed. Only golden shiners were in the trap. All fish were removed from the trap, and the trap was removed from Lake Danielson. No fish were observed in or captured from the golf course pond.

The fish samples were packed with dry ice, and the sediment samples were packed with fresh ice, and all samples were shipped that day via Federal Express for overnight delivery to the analytical laboratory. The laboratory was directed to grind the whole fish in Fish Sample No. 1 for whole body analysis and to fillet the fish in Fish Sample No. 2 for muscle tissue analysis. All fish and sediment samples, as well as the rinsate blank, were analyzed by EPA SW-846 Method 8081 for pesticides. Pesticide concentrations in sediment were reported on a dry weight basis, whereas pesticide concentrations in fish were reported on an "as received" basis. The analytical data are shown in Table 8-1.

Table 8-1

Pesticide Concentrations Reported for the 1997 Sediment and Fish Samples Collected from the Golf Course Impoundments at the Defense Distribution Depot, Memphis, Tennessee

Sample Number	Concentrations					
	Heptachlor Epoxide	DDE	DDD	DDT	Chlordane	Dieldrin
Sediment (µg/kg dry weight)						
1	54	850	211	99	640	ND
2	ND	ND	ND	ND	ND	ND
3	87	1650	537	157	3890	ND
5	ND	386	123	ND	1030	ND
6	88	1470	712	166	2150	ND
7	ND	76	46	71	ND	ND
8	67	1170	448	164	2390	ND
9	ND	102	33	ND	210	ND
10	115	1780	1000	227	2440	ND
11	ND	95	48	ND	ND	ND
12	ND	95	38	ND	ND	ND
13	ND	134	65	35	ND	ND
15	114	2120	883	234	2870	ND
Fish (µg/kg as received)						
1	ND	3190	490	12	732	45
2	ND	600	124	ND	166	13

Notes:
Highlighted values were used in risk calculations.
Sediment Sample No. 2 had higher detection limits due to small sample size.
Sediment Sample No. 4 could not be collected due to gravel covering the pond bottom at that location.
Sediment Sample No. 15 was a duplicate of No. 6.
Fish Sample No. 1 was a whole-body analysis. Fish Sample No. 2 was filleted.
ND = Not detected

As expected, pesticide concentrations were much higher in the whole fish than in the fish muscle tissue, since these pesticides are highly lipophilic and partition preferentially to skin and internal organs. Pesticide concentrations in sediment were quite variable.

The data from this sampling event were used to reevaluate the human health risks associated with exposure to the golf course pond. The data were used in the same way that historical data had been used in the initial BRA. The maximum concentration of each pesticide detected in any sediment sample was used as the basis for the exposure concentration. The pesticide concentrations reported for Fish Sample No. 2 were used as the representative exposure concentrations for fish ingestion, since the primary interest is the risk association with human ingestion of the edible portion (i.e., muscle tissue). Humans are unlikely to eat golden shiners,

but the sample data were used as surrogates for edible fish species, since the shiners were the only fish obtained from the ponds. All other parameter inputs used to calculate intake and risk were the same as those used in the initial BRA.

The results of the risk calculations using the new analytical data are shown in Table 8-2 and in Appendix B. As before, sediment ingestion and dermal exposure to sediment while swimming were found to pose minimal risk. The risk associated with fish ingestion was conservatively estimated to be 6.3E-06. Combining the risks across pathways yields a total receptor risk of 7.3E-06, 80% of which is attributable to fish ingestion. This risk level is near the low end of EPA's range of concern (i.e., 10^{-4} to 10^{-6}).

Table 8-2

Cancer Risk Estimates for Lake Danielson and Golf Course Pond Based on 1997 Data

Contaminant	Dermal Exposure	Sediment Ingestion	Fish Ingestion
DDD	3.33E-08	2.93E-11	2.87E-07
DDE	9.90E-08	8.80E-11	1.97E-06
DDT	1.10E-08	9.71E-12	NA
Chlordane	7.01E-07	6.17E-10	2.08E-06
Heptachlor Epoxide	1.45E-07	1.28E-10	NA
	Total Pathway Risk	**Total Pathway Risk**	**Total Pathway Risk**
	9.90E-07	8.72E-10	6.35E-06

This page intentionally left blank.

9.0 FINAL INVESTIGATION

EPA and State of Tennessee regulators expressed concern that the September/October 1997 sampling event might have failed to detect edible fish species that might possibly be present in the ponds. They recommended that electro-fishing be employed to definitively ascertain the presence or absence of edible fish. Radian subsequently obtained the services of TVA to electro-fish the golf course ponds.

This final sampling event took place 12–13 August 1998. On August 12, two TVA fisheries biologists launched a 14-ft boat equipped with an electro-fishing unit into Lake Danielson. They set out three gill nets, one at the pond's inlet on the northwest corner, one at the northeast corner, and one in the middle of the pond. The gill nets were left in place overnight.

The gill nets were checked shortly after dawn on the morning of 13 August 1998. No fish were captured by this method. The TVA fisheries biologists electro-fished the entire perimeter of Lake Danielson, moving slowly within 5 ft of the shoreline. They collected dozens of shiners, approximately 3 in. in length, which were identical in appearance to the fish collected during the 1997 sampling event. They also collected a few larger fish (approximately 6 in. in length) of the same species. Three bullfrogs were collected from Lake Danielson as well. After two circuits of Lake Danielson's shoreline and a series of transects that covered the entire pond surface area, for a total of more than 70 minutes of electro-fishing effort, no additional fish species were encountered. The TVA fisheries biologist with more than 20 years of experience with this type of sampling concluded that it is highly unlikely that any fish species other than the observed shiners are present in Lake Danielson.

After completing sampling of Lake Danielson, the TVA fisheries biologists launched their boat in the small pond and began electro-fishing. They collected several western mosquitofish (*Gambusia affinis*) and observed hundreds more that were too small to capture in their dip net. They also collected 12 goldfish (*Carassius auratus*) and observed 5 other goldfish. They collected three adult bullfrogs as well. The TVA biologists concluded that neither pond would be of interest to anglers. A letter from TVA's Gary Jenkins expressing this conclusion is provided as Appendix C. Photographs of this sampling event are shown in Appendix D.

The fish and frogs that were collected were immediately rinsed with distilled water, wrapped in aluminum foil, packaged in ZipLoc bags, and placed in a cooler with ice. The three frogs from Lake Danielson were packaged together as a single composite sample designated "Frog Sample 1." The three frogs from the smaller pond were packaged together as a single composite sample designated "Frog Sample 2." All of the smaller shiners (2 to 4 in. in length) from Lake Danielson were packaged together as a single composite sample designated "Fish Sample 1." The larger shiners (5.5 to 7.5 in. in length) from Lake Danielson were packaged together as a single composite sample designated "Fish Sample 2." The six goldfish (6 to 8 in. in length) collected from the smaller pond were packaged together as a single composite sample designated "Fish Sample 3." All samples were kept on ice until being shipped with dry ice to Lancaster Laboratories for pesticide analysis the following business day. Chain-of-custody records are shown in Appendix E. The analytical results for all samples are shown in Appendix F and are summarized in Table 9-1.

Table 9-1

Pesticide Concentrations Reported for the 1998 Fish and Frog Samples Collected from the Golf Course Impoundments at the Defense Distribution Depot, Memphis, Tennessee

Sample Number	Concentrations				
	DDE	DDD	DDT	Chlordane	Dieldrin
Fish (µg/kg as received)					
1	762	257	17.9	400	36.7
2	1440	160	12.6	340	85.9
3	1570	690	109	560	167
Frog Leg Muscle (µg/kg as received)					
1	17	3.52	2.5	ND	31.4
2	1.85	ND	ND	ND	23.8

ND = Not detected

The analytical results from the August 1998 sampling event were used to reevaluate human health risks associated with exposure to contaminated media in the golf course ponds. Humans are unlikely to ingest golden shiners or gold fish but do occasionally eat frog legs, so the pesticide concentrations in frog tissue were used to evaluate the risk to humans who might ingest frog legs. The laboratory was directed to analyze the frog leg muscle only, since humans would eat only the legs of bullfrogs. Pesticide concentrations in Frog Sample 1 were

higher than in Frog Sample 2; therefore, results for Frog Sample 1 were used to reevaluate human health risk based on ingestion of frog legs.

The same exposure scenario used for the previous human health risk assessment was applied to this quantification of risk. It was assumed that the ingestion rate of frog legs would be 10% the mean annual per capita fish ingestion rate for the United States since ingestion of frog legs is far less common than ingestion of fish. All other parameter values used in the previous risk assessments were applied to this quantification of risk. The total cancer risk due to ingestion of pesticides in frog legs is 4.9E-07. This is below the level of regulatory concern. The hazard index calculated for ingestion of all pesticides found in frog muscle tissue is 6E-04. A hazard index less than 1 indicates that noncancer health effects are not expected to result from this exposure. Due to the absence of edible fish species in the golf course ponds, there is no other plausible exposure pathway that would result in unacceptable risk to human health.

This page intentionally left blank.

10.0 UNCERTAINTY ANALYSIS

The results of this risk assessment should be considered in light of the numerous uncertainties regarding the assumptions that had to be made to quantify risk in the absence of site-specific information. The greatest source of uncertainty is the assumption that a person would come into contact with the contaminated sediment in the golf course impoundments. Fishing and swimming in the impoundments is currently prohibited and would likely be prohibited under future ownership. Even if someone were to gain unauthorized access to wade, swim, or fish in the impoundments, it is unlikely that anyone would do so as often as described in the exposure assessment. Exposure frequency and duration values were chosen that are on the high end of the range of realistic possibilities in order to be conservative in the quantification of risk. Likewise, upper bound values were used for other exposure variables, as recommended by EPA. For example, the amount of sediment assumed to be suspended in the water column would result in very muddy looking water, which would not appeal to most swimmers, including children.

The maximum detected concentration of each pesticide was chosen as the representative exposure concentration in each risk assessment in order to avoid underestimating risk. The representative exposure concentrations used for fish tissue in the initial assessment were assumed to be equal to the maximum concentrations detected in fish tissue samples from a 1986 AEHA investigation.

The representative exposure concentrations used for fish tissue in the follow-up assessment were the pesticide concentrations measured in the muscle tissue of golden shiners, a bait fish not typically eaten by humans. The absence of other, edible fish in the impoundments further decreases the likelihood that the modeled exposure would occur and that the estimated cancer risk would actually be incurred by anyone.

The systemic toxicity and carcinogenicity of DDD, DDE, and DDT are largely based on laboratory studies using rats and mice. Extrapolating from rodents to humans and from high experimental doses to relatively low environmental doses may introduce uncertainty in the toxicity assessment by orders of magnitude. For example, in deriving the RfD for DDT, an

uncertainty factor of 10 was applied to the NOAEL from a laboratory study to account for interspecies conversion. This assumes that DDT is 10 times more toxic to humans than it is to rats. An additional uncertainty factor of 10 was applied to ensure that the most sensitive individual in the human population is protected. The average human might be able to tolerate a chronic daily intake several times higher than the RfD without experiencing adverse health effects.

The combination of several conservative (i.e., high end) assumptions regarding exposure and toxicity is more likely to have overestimated than underestimated risk for the golf course impoundments.

11.0 ECOLOGICAL RISK ASSESSMENT

It is a CERCLA requirement to consider risks to ecological receptors (i.e., plants and animals) when making remediation decisions. The ERA considers the plant and animal populations that are actually or potentially exposed to the contaminated media, the way in which exposure is likely to occur, and the toxicity of the contaminants in the exposed species. This ERA was conducted in accordance with *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessment (*EPA 1997a).

The Depot is located in a highly developed, urban area. Most of the facility is paved or covered with buildings, and there is little observable vegetation except on the golf course. The unsurfaced areas support Bermuda grass and a few deciduous black oak (*Quercus velutina*). Some decorative plant species have been used in landscaping the housing area, golf course, administrative areas, and the lake. No threatened or endangered species have been sighted on the installation. The area is generally poor ecological habitat (Law Environmental, Inc. 1990), because in this highly developed area there are few undisturbed wetlands, forest, or other natural wildlife habitat to provide food and shelter for wildlife species to live and raise their young.

However, various birds can access the golf course ponds. The pesticide DDT is known to cause eggshell thinning in many bird species as a result of eating DDT-contaminated forage or prey. For these reasons, the ERA focused on ingestion of pesticide-contaminated fish and frogs by piscivorous (fish-eating) birds.

Belted kingfishers (*Ceryle alcyon*) and great blue herons (*Ardea herodias*) are commonly occurring piscivorous birds whose geographic range includes the Memphis area, so these species were used to quantify ecological risks. Body weight and food ingestion rate values published for these birds by EPA (1993) were used to quantify risks associated with feeding from the golf course ponds. All fish samples collected during the 1998 sampling event were analyzed as whole fish, since piscivorous wildlife would eat the entire fish. The maximum reported pesticide concentrations in fish were used to evaluate the risk to piscivorous birds.

Belted kingfishers were assumed to eat only the smaller shiners from Lake Danielson because this relatively small bird would be unable to swallow the larger shiners or goldfish. Great blue herons were alternately assumed to eat frogs (from Lake Danielson) and goldfish (from the smaller pond). Goldfish from the smaller pond contained higher pesticide concentrations than either fish sample from Lake Danielson, and higher than either frog sample, so they represent the worst-case risk to great blue herons. The specific values used in the quantification of ecological risk are shown in the risk assessment spreadsheets in Appendix B.

Pesticide intake by birds was calculated by multiplying the pesticide concentration in food by the food ingestion rate, then dividing the product by body weight. The intake of each pesticide thus quantified for each receptor species was compared to the chemical-specific, species-specific NOAEL values published by Opresko et al. (1995). The NOAEL is the chemical-specific intake that has been experimentally observed to not cause detectable adverse effects in the exposed species. NOAEL values are not available for all chemicals or all species; however, Opresko et al. (1995) have used observed NOAEL values for certain bird species to estimate NOAEL values for other bird species. Estimated NOAEL values for belted kingfishers and great blue herons have been derived from DDT data on the brown pelican, dieldrin data on the barn owl, and chlordane data on the red-winged blackbird. The estimated NOAEL values for belted kingfishers and great blue herons are shown in the risk assessment spreadsheets in Appendix B. Toxicity profiles for wildlife are provided in Appendix A.

The estimated intake of DDT and its metabolites exceeded the estimated NOAEL values for both belted kingfishers and great blue herons. However, this analysis is based on the assumption that each bird obtains its entire food supply from the golf course ponds. Considering the much larger home ranges and feeding territories for these birds, it is extremely unlikely that any individual belted kingfisher or great blue heron would receive the dose of pesticides estimated in this analysis.

Unlike human health risk assessment, ecological risk assessment is concerned with population-level or community-level effects, rather than effects to individual organisms (Suter 1993, EPA 1997a). Adverse effects to individual organisms are of concern only in the case of threatened or endangered species. Neither belted kingfishers nor great blue herons are rare. In

fact, they are both fairly abundant throughout North America. The population-level effect recommended by Suter (1993) as a benchmark of unacceptable ecological risk is a 20% reduction in the size of the population.

As previously discussed, the Depot is located in a highly developed, urban area and provides generally poor ecological habitat. Significant use of the golf course ponds as feeding territory by any individual bird, much less an extensive population, is highly unlikely. Therefore, a 20% reduction in the population of either bird species as a result of ingestion of fish from the golf course ponds is implausible.

The uncertainty regarding the use of surrogate species (e.g., brown pelicans) to develop NOAEL values for belted kingfishers and great blue herons should also be considered in evaluating this quantification of risk. Furthermore, NOAEL values are those experimental contaminant doses that yielded no observed adverse effects. The actual dose that would cause an adverse effect (e.g., the Lowest Observed Adverse Effects Level) might be orders of magnitude higher than the NOAEL.

Considering the conservative nature of the ERA, the very low probability of an adverse effect to any individual organism, and the implausibility of population-level effects to either species, remediation on the basis of ecological risk is inadvisable.

This page intentionally left blank.

12.0 CONCLUSIONS AND RECOMMENDATIONS

The sediments in Lake Danielson and the golf course pond are a sink for pesticide contamination in the surrounding soils that resulted from pre-1980 use of DDT for pest control. The pesticide residues appear to be bound to sediment particles and are not likely to be mobilized to other environmental media by natural processes. Since fishing and swimming in the golf course impoundments are prohibited, there are no current exposure pathways. If recreational use of Lake Danielson and/or the golf course pond were to occur in the future as described in the exposure assessment, the probability of contracting cancer as a result of ingesting contaminated frog legs, ingestion of sediment, and dermal contact with sediment are below the range of concern. Noncancer adverse health effects are also unlikely.

Intake of pesticides by piscivorous birds, such as the belted kingfishes and the great blue heron, that might forage in the golf course ponds is unlikely to result in significant adverse effects.

The combination of several conservative (i.e., high end) assumptions regarding exposure and toxicity is more likely to have overestimated than underestimated risk. Based on the minimal human health and ecological risks that have been conservatively estimated for exposure to pesticide residues in the golf course impoundments, no further investigation or remediation of the impoundments is recommended.

This page intentionally left blank.

13.0 REFERENCES

AEHA 1986. *Water Quality Biological Study No. 32-24-0733-86, Investigation of Fire Reservoir, Defense Depot, Memphis, Tennessee.*

ATSDR 1991. *Toxicological Profile for Dieldrin.*

ATSDR 1992. *Toxicological Profile for Heptachlor Epoxide.*

ATSDR 1994. *Toxicological Profile for 4,4'-DDT, 4,4'-DDE, 4,4'-DDD (Update).*

CH2M Hill 1996. Proposal for Next Tier Evaluation for Lake Danielson at the Depot, Memorandum to Mark Corey, May 13.

EPA 1997a. *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessment.*

EPA 1997b. *Supplemental Guidance to RAGS*, Region 4 Bulletins, Office of Technical Services, March 19.

EPA 1990. *Exposure Factors Handbook*, EPA/600/8-89/043, Office of Health and Environmental Assessment, Washington, D.C.

EPA 1989. *Risk Assessment Guidance for Superfund, Vol. 1: Human Health Evaluation Manual*, EPA 540/1-89-002, Office of Emergency and Remedial Response, Washington, D.C.

Integrated Risk Information System (IRIS) 1999.

Law Environmental, Inc. 1990. *Remedial Investigation Report, Defense Depot, Memphis, Tennessee.*

Opresko, D. M., Sample, B. E., and G. W. Suter II 1995. *Toxicological Benchmarks for Wildlife: 1995 Revision*, ES/ER/TM-86/R2, Lockheed Martin Energy Systems, Inc.

Radian 1997. *Baseline Risk Assessment for Golf Course Impoundments at the Defense Distribution Depot, Memphis.*

Ryan, E. A., E. T Hawkins, B. Magee, and S. L. Santos 1987. "Assessing Risk from Dermal Exposure at Hazardous Waste Sites," *Superfund '87 Proceedings of the 8th National Conference*, Washington, D.C., Sponsored by the Hazardous Materials Control Research Institute, November 16-18.

Suter, G.W. II 1993. *Ecological Risk Assessment*, Lewis Publishers, Boca Raton.

This page intentionally left blank.

Appendix A
DETAILED TOXICITY SUMMARIES

This page intentionally left blank.

0147
p,p'-Dichlorodiphenyltrichloroethane (DDT); CASRN 50-29-3 (03/01/97)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR DDT

File On-Line 03/31/87

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/96
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	05/01/91

==========

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- p,p'-Dichlorodiphenyltrichloroethane (DDT)
CASRN -- 50-29-3
Last Revised -- 02/01/96

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
Liver lesions	NOEL: 1 ppm diet (0.05 mg/kg bw/day)	100	1	5E-4 mg/kg/day
27-Week Rat Feeding Study	LOAEL: 5 ppm			
Laug et al., 1950				

---

*Conversion Factors: Food consumption = 5% bw/day

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Laug, E.P., A.A. Nelson, O.G. Fitzhugh and F.M. Kunze. 1950. Liver cell alteration and DDT storage in the fat of the rat induced by dietary levels of 1-50 ppm DDT. J. Pharmacol. Exp. Therap. 98: 268-273.

Weanling rats (25/sex/group) were fed commercial DDT (81% P,P isomer and 19% O,P isomer) at levels of 0, 1, 5, 10 or 50 ppm for 15-27 weeks. The diet was prepared by mixing appropriate amounts of DDT in corn oil solution with powdered chow. No interference with growth was noted at any level. Females stored more DDT in peripheral fat than did males, but pathologic changes were seen to a greater degree in males. Increasing hepatocellular hypertrophy, especially centrilobularly, increased cytoplasmic oxyphilia, and peripheral basophilic cytoplasmic granules (based on H and E paraffin sections) were observed at dose levels of 5 ppm and above. The effect was minimal at 5 ppm (LOAEL) and more pronounced at higher doses. No effects were reported at 1 ppm, the NOEL level used as the basis for the RfD calculation. The authors believe the effect seen at 5 ppm "represents the smallest detectable morphologic effect, based on extensive observations of the rat liver as affected by a variety of chemicals."

DDT fed to rats for 2 years (Fitzhugh, 1948) caused liver lesions at all dose levels (10-800 ppm of diet). A LOAEL of 0.5 mg/kg bw/day was established. Application of a factor of 10 each for uncertainty of estimating a NOEL from a LOAEL, as well as for interspecies conversion and protection of sensitive human subpopulations (1000 total) results in the same RfD level as that calculated from the critical study. DDT-induced liver effects were observed in mice, hamsters and dogs as well.

The Laug et al. (1950) study was chosen for the RfD calculation because: 1) male rats appear to be the most sensitive animals to DDT exposure; 2) the study was of sufficient length to observe toxic effects; and 3) several doses were administered in the diet over the range of the dose-response curve. This study also established a LOAEL and a NOEL, with the LOAEL (0.25 mg/kg/day) being the lowest of any observed for this compound.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- A factor of 10 each was applied for the uncertainty of interspecies conversion and to protect sensitive human subpopulations. An uncertainty factor for subchronic to chronic conversion was not included because of the corroborating chronic study in the data base.

MF --None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

In one 3-generation rat reproduction study (Treon and Cleveland, 1955), offspring mortality increased at all dose levels, the lowest of which corresponds to about 0.2 mg/kg bw/day. Three other reproduction studies (rat and mouse) show no reproductive effects at much higher dose levels.

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Medium
Data Base -- Medium
RfD -- Medium

The principal study appears to be adequate, but of shorter duration than that desired; therefore, confidence in the study can be considered medium to low. The data base is only moderately supportive of both the critical effect and the magnitude, and lacks a clear NOEL for reproductive effects; therefore, confidence in the data base can also be considered medium to low. Medium to low confidence in the RfD follows.

___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 -- None

Agency Work Group Review -- 12/18/85

Verification Date -- 12/18/85

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- p,p'-Dichlorodiphenyltrichloroethane (DDT)
CASRN -- 50-29-3

Not available at this time.

==========

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- p,p'-Dichlorodiphenyltrichloroethane (DDT)
CASRN -- 50-29-3
Last Revised -- 05/01/91

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water

or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen.

Basis -- Observation of tumors (generally of the liver) in seven studies in various mouse strains and three studies in rats. DDT is structurally similar to other probable carcinogens, such as DDD and DDE.

II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. The existing epidemiological data are inadequate. Autopsy studies relating tissue levels of DDT to cancer incidence have yielded conflicting results. Three studies reported that tissue levels of DDT and DDE were higher in cancer victims than in those dying of other diseases (Casarett et al., 1968; Dacre and Jennings, 1970; Wasserman et al., 1976). In other studies no such relationship was seen (Maier-Bode, 1960; Robinson et al., 1965; Hoffman et al., 1967). Studies of occupationally exposed workers and volunteers have been of insufficient duration to be useful in assessment of the carcinogenicity of DDT to humans.

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Twenty-five animal carcinogenicity assays have been reviewed for DDT. Nine feeding studies, including two multigenerational studies, have been conducted in the following mouse strains: BALB/C, CF-1, A strain, Swiss/Bombay and (C57B1)x(C3HxAkR). Only one of these studies, conducted for 78 weeks, showed no indication of DDT tumorigenicity (NCI, 1978). Both hepatocellular adenomas and carcinomas were observed in six mouse liver tumor studies (Walker et al., 1973; Thorpe and Walker, 1973; Kashyap et al., 1977; Innes et al., 1969; Terracini et al., 1973; Turusov et al., 1973). Both benign and malignant lung tumors were observed in two studies wherein mice were exposed both in utero and throughout their lifetime (Shabad et al., 1973; Tarjan and Kemeny, 1969). Doses producing increased tumor incidence ranged from 0.15-37.5 mg/kg/day.

Three studies using Wistar, MRC Porton and Osborne-Mendel rats and doses from 25-40 mg/kg/day produced increased incidence of benign liver tumors (Rossi et al., 1977; Cabral et al., 1982; Fitzhugh and Nelson, 1946). Another study wherein Osborne-Mendel rats were exposed in this dietary dose range for 78 weeks was negative (NCI, 1978) as were three additional assays in which lower doses were given.

Tests of DDT in hamsters have not resulted in increased tumor incidence. Unlike mice and humans, hamsters accumulate DDT in tissue but do not metabolize it to DDD or DDE. Studies of DDT in dogs (Lehman, 1951, 1965) and monkeys (Adamson and Sieber, 1979, 1983) have not shown a carcinogenic effect. However, the length of these studies (approximately 30% of the

animals' lifetimes) was insufficient to assess the carcinogenicity of DDT. DDT has been shown to produce hepatomas in trout (Halver, 1967).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

DDT has been shown to act as a liver tumor promoter in rats initiated with 2-acetylaminofluorene, 2-acetamidophenanthrene or trans-4-acetylaminostilbene (Peraino et al., 1975; Scribner and Mottet, 1981; Hilpert et al., 1983).

DDT has produced both negative and positive responses in tests for genotoxicity. Positive responses have been noted in V79 mutation assays, for chromosome aberrations in cultured human lymphocytes, and for sister chromatid exchanges in V79 and CHO cells (Bradley et al., 1981; Rabello et al., 1975; Preston et al., 1981; Ray-Chaudhuri et al., 1982). In one study, DDT was reported to interact directly with DNA; this result was not confirmed in the absence of a metabolizing system (Kubinski et al., 1981; Griffin and Hill, 1978).

DDT is structurally related to the following chemicals which produce liver tumors in mice: DDE, DDD, dicofol and chlorobenzilate.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 3.4E-1 per (mg/kg)/day

Drinking Water Unit Risk -- 9.7E-6 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	1E+1 ug/L
E-5 (1 in 100,000)	1E+0 ug/L
E-6 (1 in 1,000,000)	1E-1 ug/L

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- Liver, benign and malignant (see table)
Test Animals -- mouse and rat (see table)
Route -- diet
Reference -- see table

Species/Strain Tumor Type	Slope Factor Male	Slope Factor Female	Reference
Mouse/CF-1, Benign	0.80	0.42	Turusov et al., 1973
Mouse/BALB/C, Benign	0.082		Terracini et al., 1973
Mouse/CF-1, Benign, Malignant	0.52	0.81	Thorpe and Walker, 1973

Mouse/CF-1, Benign	1.04	0.49	Tomatis and Turusov, 1975
Rat/MRC Porton		0.084	Cabral et al., 1982
Rat/Wistar, Benign	0.16	0.27	Rossi et al., 1977

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

The estimate of the slope factor did not increase in the multigeneration feeding studies (Terracini et al., 1973; Turusov et al., 1973) but remained the same from generation to generation. A geometric mean of the above slope factors was used for the overall slope factor of 3.4E-1. This was done in order to avoid excluding relevant data (note that the appropriateness of this procedure is currently under study by U.S. EPA). All tumors were of the liver; there were no metastases. A few malignancies were observed in the Turusov study; possible neoplasms were indicated in the Terracini and Tomatis studies. The Turusov study was carried out over six generations, the Terracini assay for two. The slope factor derived from data of Tarjan and Kemeny (1969) was not included in the calculation of the geometric mean because the tumors developed at different sites than in any other studies. In addition, there was a problem in this study with possible DDT contamination of the feed.

DDT is known to be absorbed by humans in direct proportion to dietary exposure; t(1/2) for clearance is 10-20 years.

The unit risk should not be used if the water concentraion exceeds 1E+3 ug/L, since above this concentration the unit risk may not be appropriate.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Ten slope factors derived from six studies were within a 13-fold range. The slope factor derived from the mouse data alone was 4.8E-1 while that derived from the rat data alone was 1.5E-1. There was no apparent difference in slope factor as a function of sex of the animals. The geometric mean of the slope factors from the mouse and rat data combined was identical for the same tumor site as that for DDE [3.4E-1 per (mg/kg)/day], a structural analog.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 9.7E-5 (ug/cu.m)

Extrapolation Method -- Linear multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	1E+0 ug/cu.m
E-5 (1 in 100,000)	1E-1 ug/cu.m
E-6 (1 in 1,000,000)	1E-2 ug/cu.m

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

The inhalation risk estimates were calculated from the oral data presented in Section II.B.2.

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The unit risk should not be used if the air concentration exceeds 1E+2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

This inhalation risk estimate was calculated from the oral data presented in Section II.B.2.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1985

The U.S. EPA risk assessment document on DDT is an internal report and has not received external review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 10/29/86, 11/12/86, 06/24/87

Verification Date -- 06/24/87

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

==========

_VI. BIBLIOGRAPHY

Substance Name -- p,p'-Dichlorodiphenyltrichloroethane (DDT)
CASRN -- 50-29-3
Last Revised -- 05/01/91

__VI.A. ORAL RfD REFERENCES

Fitzhugh, O.G. 1948. Use of DDT insecticides on food products. Ind. Eng. Chem. 40(4): 704-705.

Laug, E.P., A.A. Nelson, O.G. Fitzhugh and F.M. Kunze. 1950. Liver cell

alteration and DDT storage in the fat of the rat induced by dietary levels of 1-50 ppm DDT. J. Pharmacol. Exp. Therap. 98: 268-273.

Treon, J.F. and F.P. Cleveland. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. J. Agric. Food Chem. 3(5): 402-408.

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Adamson, R.H. and S.M. Sieber. 1979. The use of nonhuman primates for chemical carcinogenisis studies. Ecotoxicol. Environ. Qual. 2: 275-296.

Adamson, R.H. and S.M. Sieber. 1983. Chemical carcinogenesis studies in nonhuman primates. Basic Life Sci. 24: 129-156.

Bradley, M.O., B. Bhuyan, M.C. Francis, R. Langenbach, A. Peterson and E. Huberman. 1981. Mutagenesis by chemical agents in V79 Chinese hamster cells: A review and analysis of the literature. Mutat. Res. 87: 81-142.

Cabral, J.R.P., R.K. Hall, L. Rossi, S.A. Bronczyk and P. Shubik. 1982. Effects of long-term intake of DDT on rats. Tumorigenesis. 68: 11-17.

Casarett, L.J., G.C. Fryer, W.L. Yauger, Jr. and H.W. Klemmer. 1968. Organochlorine pesticide residues in human tissue--Hawaii. Arch. Environ. Health. 17: 306-311.

Dacre, J.C. and R.W. Jennings. 1970. Organochlorine insecticides in normal and carcinogenic human lung tissues. Toxicol. Appl. Pharmacol. 17: 277.

Fitzhugh, O.G. and A.A. Nelson. 1946. The chronic oral toxicity of DDT [2,2-bis(p-chlorophenyl-1,1,1-trichloroethane)]. J. Pharmacol. 89: 18-30.

Griffin, D.E. and W.E. Hill. 1978. In vitro breakage of plasmid DNA by mutagens and pesticides. Mutat. Res. 52: 161-169.

Halver, J.E. 1967. Crystalline aflatoxin and other vectors for trout hepatoma. In: J.E. Halver and I.A. Mitchell, Ed. Trout Hepatoma Research Conference Papers. Bureau of Sport Fisheries and Wildlife Research Rep. No. 70. Dept. of the Interior, Washington, DC: p. 78-102.

Hilpert, D., W. Romen and H-G. Neumann. 1983. The role of partial hepatectomy and of promoters in the formation of tumors in non-target tissues of trans-4-acetylaminostilbene in rats. Carcinogenesis. 4(12): 1519-1525.

Hoffman, W.S., H. Adler, W.I. Fishbein and F.C. Bauer. 1967. Relation of pesticide concentrations in fat to pathological changes in tissues. Arch. Environ. Health. 15: 758-765.

Innes, J.R.M., B.M. Ulland, M.G. Valerio, et al. 1969. Bioassay of pesticides and industrial chemicals for tumorgenicity in mice: A preliminary note. J. Natl. Cancer Inst. 42(6): 1101-1114.

Kashyap, S.K., S.K. Nigam, A.B. Karnik, R.C. Gupta and S.K. Chatterjee. 1977. Carcinogenicity of DDT (dichlorodiphenyl trichloroethane) in pure inbred Swiss mice. Int. J. Cancer. 19: 725-729.

Kubinski, H., G.E. Gutzke and Z.O. Kubinski. 1981. DNA-cell-binding (DCB) assay for suspected carcinogens and mutagens. Mutat. Res. 89: 95-136.

Lehman, A.J. 1951. Chemicals in Foods: A Report to the Association of Food and Drug Officials on Current Developments. Part II, Pesticides. Section V. Pathology, Q. Bull. Assoc. Food Drug Office, U.S. 15(4): 126-132.

Lehman, A.J. 1965. Summaries of pesticide toxicity. Association of Food and Drug Officials of the United States, Topeka, Kansas.

Maier-Bode, H. 1960. Zur Frage der Herkunft des DDT im Koperfett des Menschen. Med. Exp. 3: 284-286. (Ger.)

NCI (National Cancer Institute). 1978. Bioassays of DDT, TDE and p,p'-DDE for possible carcinogenicity (CAS No. 50-29-3, 72-54-8, 72-55-9). NCI Report No. 131. DHEW Publ. No. (NIH) 78-1386.

Peraino, C., R.J.M. Fry, E. Staffeldt and J. P. Christopher. 1975. Comparative enhancing effects of phenobarbital, amobarbital, diphenylhydantoin, and dichlorodiphenyltrichloroethane of 2-acetylaminofluorene-induced hepatic tumorgenesis in the rat. Cancer Res. 35: 2884-2890.

Preston, R.J., W. Au, M.A. Bender, et al. 1981. Mammalian in vivo and in vitro cytogenetic assays: A report of the U.S. EPA's Gene-Tox Program. Mutat. Res. 87: 143-188.

Rabello, M.N., W. Becak, W.F. DeAlmeida, et al. 1975. Cytogenetic study on individuals occupationally exposed to DDT. Mutat. Res. 28: 449-454.

Ray-Chaudhuri, R., M. Currens and P.T. Iype. 1982. Enhancement of sister-chromatid exchanges by tumor promoters. Br. J. Cancer. 45: 769-777.

Robinson, J., A. Richardson, C.G. Hunter, A.N. Crabtree and H.S. Rees. 1965. Organo-chlorine insecticide content of human adipose tissue in south-eastern England. Br. J. Ind. Med. 22: 220-229.

Rossi, L., M. Ravera, G. Repetti and L. Santi. 1977. Long-term administration of DDT or phenobarbital-Na in Wistar rats. Int. J. Cancer. 19: 179-185.

Scribner, J.D. and N.K. Mottet. 1981. DDT acceleration of mammary gland tumors induced in the male Sprague-Dawley rat by 2-acetomidophenanthrene. Carcinogenesis. 2(12): 1235-1239.

Shabad, L.M., T.S. Kolesnichenko and T.V. Nikonova. 1973. Transplacental and combined long-term effect of DDT in five generations of A-strain mice. Int. J. Cancer. 11: 688-693.

Tarjan, R. and T. Kemeny. 1969. Multigeneration studies on DDT in mice. Food Cosmet. Toxicol. 7: 215-222.

Terracini, B., M.C. Testa, J.R. Cabral and N. Day. 1973. The effects of long-term feeding of DDT to BALB/c mice. Int. J. Cancer. 11: 747-764.

Thorpe, E. and A.I.T. Walker. 1973. The toxicology of dieldrin (HEOD). II. Comparative long-term oral toxicity studies in mice with dieldrin, DDT, phenobarbitone, beta-BHC and gamma-BHC. Food Cosmet. Toxicol. 11: 433-442.

Tomatis, L. and V. Turusov. 1975. Studies on the carcinogenicity of DDT.

Gann Monograph Cancer Res. 17: 219-241.

Turusov, V.S., N.E. Day, L. Tomatis, E. Gati and R.T. Charles. 1973. Tumors in CF-1 mice exposed for six consecutive generations to DDT. J. Natl. Cancer Inst. 51: 983-998.

U.S. EPA. 1985. The Carcinogenic Assessment Groups Calculation of the Carcinogenicity of Dicofol (Kelthane), DDT, DDE and DDD (TDE). Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC for the Hazard Evaluation Division, Office of Pesticide Programs, Office of Pesticides and Toxic Substances, Washington, DC.

Walker, A.I.T., E. Thorpe and D.E. Stevenson. 1973. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. Food Cosmet. Toxicol. 11: 415-432.

Wasserman, M., D.P. Nogueira, L. Tomatis, et al. 1976. Organochlorine compounds in neoplastic and adjacent apparently normal breast tissue. Bull. Environ. Contam. Toxicol. 15(4): 478-484.

==========

_VII. REVISION HISTORY

Substance Name -- p,p'-Dichlorodiphenyltrichloroethane (DDT)
CASRN -- 50-29-3

Date	Section	Description
09/30/87	I.A.6.	Documentation changed
08/22/88	II.	Carcinogen summary on-line
01/01/91	II.	Text edited
01/01/91	II.C.1.	Inhalation slope factor removed (global change)
05/01/91	II.A.3.	Change Lehman, 1952 to '1951'
05/01/91	VI.	Bibliography on-line
01/01/92	I.A.7.	Secondary contact changed
01/01/92	IV.	Regulatory actions updated
02/01/96	I.A.7.	Contact changed

==========

SYNONYMS

Substance Name -- p,p'-Dichlorodiphenyltrichloroethane (DDT)
CASRN -- 50-29-3
Last Revised -- 03/31/87

50-29-3
AGRITAN
ANOFEX
ARKOTINE
AZOTOX
BENZENE, 1,1'-(2,2,2-TRICHLOROETHYLIDENE)BIS(4-CHLORO-)
alpha,alpha-BIS(p-CHLOROPHENYL)-beta,beta,beta-TRICHLORETHANE
1,1-BIS-(p-CHLOROPHENYL)-2,2,2-TRICHLOROETHANE
2,2-BIS(p-CHLOROPHENYL)-1,1,1-TRICHLOROETHANE
BOSAN SUPRA

BOVIDERMOL
CHLOROPHENOTHAN
CHLOROPHENOTHANE
CHLOROPHENOTOXUM
CITOX
CLOFENOTANE
DDT
p,p'-DDT
DEDELO
DEOVAL
DETOX
DETOXAN
DIBOVAN
DICHLORODIPHENYLTRICHLOROETHANE
4,4'-DICHLORODIPHENYLTRICHLOROETHANE
Dichlorodiphenyltrichloroethane, p,p'-
DICOPHANE
DIDIGAM
DIDIMAC
DIPHENYLTRICHLOROETHANE
DODAT
DYKOL
ENT 1,506
ESTONATE
ETHANE, 1,1,1-TRICHLORO-2,2-BIS(p-CHLOROPHENYL)-
GENITOX
GESAFID
GESAPON
GESAREX
GESAROL
GUESAPON
GUESAROL
GYRON
HAVERO-EXTRA
HILDIT
IVORAN
IXODEX
KOPSOL
MICRO DDT 75
MUTOXIN
NA 2761
NCI-C00464
NEOCID
PARACHLOROCIDUM
PEB1
PENTACHLORIN
PENTECH
PPZEIDAN
R50
RCRA WASTE NUMBER U061
RUKSEAM
SANTOBANE
TECH DDT
1,1,1-TRICHLOOR-2,2-BIS(4-CHLOOR FENYL)-ETHAAN
1,1,1-TRICHLOR-2,2-BIS(4-CHLOR-PHENYL)-AETHAN
1,1,1-TRICHLORO-2,2-BIS(p-CHLOROPHENYL)ETHANE
TRICHLOROBIS(4-CHLOROPHENYL)ETHANE
1,1,1-TRICHLORO-2,2-DI(4-CHLOROPHENYL)-ETHANE
1,1,1-TRICLORO-2,2-BIS(4-CLORO-FENIL)-ETANO
ZEIDANE
ZERDANE

0328
p,p'-Dichlorodiphenyldichloroethylene (DDE); CASRN 72-55-9 (04/01/97)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR DDE

File On-Line 08/22/88

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	08/22/88

==========

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- p,p'-Dichlorodiphenyldichloroethylene (DDE)
CASRN -- 72-55-9

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- p,p'-Dichlorodiphenyldichloroethylene (DDE)
CASRN -- 72-55-9

Not available at this time.

==========

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- p,p'-Dichlorodiphenyldichloroethylene (DDE)
CASRN -- 72-55-9
Last Revised -- 08/22/88

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- increased incidence of liver tumors including carcinomas in two strains of mice and in hamsters and of thyroid tumors in female rats by diet.

___II.A.2. HUMAN CARCINOGENICITY DATA

Human epidemiological data are not available for DDE. Evidence for the carcinogenicity in humans of DDT, a structural analog, is based on autopsy studies relating tissue levels of DDT to cancer incidence. These studies have yielded conflicting results. Three studies reported that tissue levels of DDT and DDE were higher in cancer victims than in those dying of other diseases (Casarett et al., 1968; Dacre and Jennings, 1970; Wasserman et al., 1976). In other studies no such relationship was seen (Maier-Bode, 1960; Robinson et al., 1965; Hoffman et al., 1967). Studies of volunteers and workers occupationally exposed to DDT have been of insufficient duration to determine the carcinogenicity of DDT to humans.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. NCI (1978) administered DDE in feed at TWA doses of 148 and 261 ppm to 50 B6C3F1 mice/sex/dose for 78 weeks. After an additional 15 weeks, a dose-dependent and statistically significant increase in incidence of hepatocellular carcinomas was observed in males and females in comparison with controls. Increased weight loss and mortality was observed in females.

Tomatis et al. (1974) administered 250 ppm DDE in feed for lifetime (130 weeks) to 60 CF-1 mice/sex. A statistically significant increase in incidence of hepatomas was observed in both males and females in comparison with controls. In females, 98% of the 55 surviving exposed animals developed hepatomas, compared to 1% of the surviving controls.

Rossi et al. (1983) administered DDE in feed for 128 weeks to 40-46 Syrian Golden hamsters/sex/dose at doses of 500 and 1000 ppm. After 76

weeks, a statistically significant increase in incidence of neoplastic nodules of the liver were observed in both sexes in comparison with vehicle-treated controls.

NCI (1978) also fed DDE at TWA doses of 437 and 839 ppm for males and 242 and 462 ppm for females for 78 weeks to 50 Osborne-Mendel rats/sex/ dose, with an additional 35 week observation period. A dose-dependent trend in incidence of thyroid tumors was observed in females which was statistically significant by the Cochran Armitage trend test after adjustment for survival. The Fischer Exact test, however, was not statistically significant. Overall, the results of the bioassay were not considered by NCI to provide convincing evidence for carcinogenicity.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

DDE was mutagenic in mouse lymphoma (L5178Y) cells and chinese hamster (V79) cells, but not in Salmonella (ICPEMC, 1984). DDE is structurally similar to and a metabolite of DDT (Peterson and Robinson, 1964; Gingell and Wallcave, 1976; Morgan and Roan, 1977) which is a probable human carcinogen.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 3.4E-1/mg/kg/day

Drinking Water Unit Risk -- 9.7E-6/ug/L

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

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

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- hepatocellular carcinomas, hepatomas
Test Animals -- mouse/B6C3F1; mouse/CF-1; hamsters/Syrian Golden
Route -- diet
Reference -- NCI, 1978; Tomatis et al., 1974; Rossi et al., 1983

Administered Dose (ppm)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence female	male	Reference
Mouse/B6C3F1; hepatocellular carcinomas				
0	0.0	0/19	0/19	NCI, 1978
148	0.90	19/47	7/41	
261	1.584	34/48	17/47	
Mouse/CF-1; hepatomas				
0	0	1/90	33/98	Tomatis et

250	2.45	54/55	39/53	al., 1974

Hamsters/Syrian Golden; neoplastic nodules (hepatomas)

0	0	0/31	0/42	Rossi et
500	4.79	7/30	4/39	al., 1983
1000	9.57	8/39	6/39	

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

NCI (1978) used DDE of about 95% purity, while that used by Tomatis et al. (1974) and Rossi et al. (1983) was 99% pure. In the hamster study, Rossi et al. described the observed lesions as neoplastic liver nodules or hepatocellular tumors, using these terms interchangeably. The oral quantitative estimate is a geometric mean of six slope factors computed from incidence data by sex from the studies cited in Section II.A.3.

The unit risk should not be used if the water concentration exceeds 1E+3 ug/L, since above this concentration the slope factor may differ from that stated.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

An adequate number of animals was observed. The geometric mean obtained using the slope factors from the mouse studies alone is 7.8E-1/mg/kg/day. This is within a factor of 2 of that derived from the mouse and hamster studies combined. In addition, the slope factor for DDE was within a factor of 2 of the slope factors for liver tumors for three structurally similar compounds: DDT, 3.4E-1/mg/kg/day; DDD, 2.4E-1/mg/kg/day; and Dicofol, 4.4E-1/mg/kg/day.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1980, 1985

The 1985 Carcinogen Assessment Group's report has received Agency review. The 1980 Hazard Assessment Report has received peer review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 06/24/87

Verification Date -- 06/24/87

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

==========

_VI. BIBLIOGRAPHY

Substance Name -- p,p'-Dichlorodiphenyldichloroethylene (DDE)
CASRN -- 72-55-9
Last Revised -- 08/01/89

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfD REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Casarett, L.J., G.C. Fryer, W.L. Yauger, Jr. and H. Klemmer. 1968. Organochlorine pesticide residues in human tissue. Hawaii. Arch. Environ. Health. 17: 306-311.

Dacre, J.C. and R.W. Jennings. 1970. Organochlorine insecticides in normal and carcinogenic human lung tissues. Toxicol. Appl. Pharmacol. 17: 277.

Gingell, R. and L. Wallcave. 1976. Species differences in the acute toxicity and tissue distribution of DDT in mice and hamsters. Toxicol. Appl. Pharmacol. 28: 385.

Hoffman, W.S., H. Adler, W.I. Fishbein and F.C. Bauer. 1967. Relation of pesticide concentrations in fat to pathological changes in tissues. Arch. Environ. Health. 15: 758-765.

ICPEMC (International Commision for Protection Against Environmental Mutagens and Carcinogens). 1984. Report of ICPEMC Task Group 5 on the differentiation between genotoxic and nongenotoxic carcinogens. ICPEMC Publication No. 9. Mutat. Res. 133: 1-49.

Maier-Bode, H. 1960. DDT im Korperfett des Menschen. Med. Exp. 1: 146-152.

Morgan, D.P. and C.C. Roan. 1977. The metabolism of DDT in man. Essays Toxicol. 5: 39.

NCI (National Cancer Institute). 1978. Bioassay of DDT, TDE and p,p'-DDE for possible carcinogenicity. NCI Report No. 131. DHEW Publ. No. (NIH) 78-1386.

Peterson, J.E. and W.H. Robinson. 1964. Metabolic products of p,p'-DDT in the rat. Toxicol. Appl. Pharmacol. 6: 321-327.

Robinson, J., A. Richardson, C.G. Hunter, A.N. Crabtree and H.J. Rees. 1965. Organochlorine insecticide content of human adipose tissue in south-eastern England. Br. J. Ind. Med. 22: 220-224.

Rossi, L., O. Barbieri, M. Sanguineti, J.R.P. Cabral, P. Bruzzi and L. Santi. 1983. Carcinogenicity study with technical-grade DDT and DDE in hamsters. Cancer Res. 43: 776-781.

Tomatis, L., V. Turusov, R.t. Charles and M. Boicchi. 1974. Effect of long-term exposure to 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene, to 1,1-dichloro-2,2-bis(p-chlorophenyl)ethane, and the two chemicals combined on CF-1 mice. J. Natl. Cancer Inst. 52: 883-891.

U.S. EPA. 1980. Hazard Assessment Report on DDT, DDD, DDE. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 1985. The Carcinogen Assessment Group's Calculation of the Carcinogenicity of Dicofol (Kelthane), DDT, DDE and DDD (TDE). Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC for the Hazard Evaluation Division, Office of Toxic Substances, Washington, DC.

Wasserman, M., D.P. Nogueira, L. Tomatis, et al. 1976. Organochlorine compounds in neoplastic and adjacent apparently normal breast tissue. Bull. Environ. Contam. Toxicol. 15: 478-484.

==========

_VII. REVISION HISTORY

Substance Name -- p,p'-Dichlorodiphenyldichloroethylene (DDE)
CASRN -- 72-55-9

Date	Section	Description
08/22/88	II.	Carcinogen summary on-line
08/01/89	VI.	Bibliography on-line
01/01/92	IV.	Regulatory Action section on-line

==========

SYNONYMS

Substance Name -- p,p'-Dichlorodiphenyldichloroethylene (DDE)
CASRN -- 72-55-9
Last Revised -- 08/22/88

72-55-9
2,2-BIS(4-CHLOROPHENYL)-1,1-DICHLOROETHENE
2,2-BIS(p-CHLOROPHENYL)-1,1-DICHLOROETHYLENE

DDE
p,p'-DDE
DDT DEHYDROCHLORIDE
1,1-DICHLORO-2,2-BIS(p-CHLOROPHENYL)ETHYLENE
DICHLORODIPHENYLDICHLOROETHYLENE
Dichlorodiphenyldichloroethylene, p,p'-
1,1'-DICHLOROETHENYLIDENE)BIS(4-CHLOROBENZENE)
ETHYLENE, 1,1-DICHLORO-2,2-BIS(p-CHLOROPHENYL)-
NCI-C00555

0347
p,p'-Dichlorodiphenyl dichloroethane (DDD); CASRN 72-54-8 (03/01/97)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR DDD

File On-Line 08/22/88

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	08/22/88

==========

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- p,p'-Dichlorodiphenyl dichloroethane (DDD)
CASRN -- 72-54-8

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- p,p'-Dichlorodiphenyl dichloroethane (DDD)
CASRN -- 72-54-8

Not available at this time.

==========

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- p,p'-Dichlorodiphenyl dichloroethane (DDD)
CASRN -- 72-54-8
Last Revised -- 08/22/88

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

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- based on an increased incidence of lung tumors in male and female mice, liver tumors in male mice and thyroid tumors in male rats. DDD is structurally similar to, and is a known metabolite of DDT, a probable human carcinogen.

II.A.2. HUMAN CARCINOGENICITY DATA

None. Human epidemiological data are not available for DDD. Evidence for the carcinogenicity in humans of DDT, a structural analog, is based on autopsy studies relating tissue levels of DDT to cancer incidence. These studies have yielded conflicting results. Three studies reported that tissue levels of DDT and DDE were higher in cancer victims than in those dying of other diseases (Casarett et al., 1968; Dacre and Jennings, 1970; Wasserman et al., 1976). In other studies no such relationship was seen (Maier-Bode, 1960; Robinson et al., 1965; Hoffman et al., 1967). Studies of occupationally exposed workers and volunteers have been of insufficient duration to determine the carcinogenicity of DDT to humans.

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Tomatis et al. (1974) fed DDD for 130 weeks at 250 ppm (TWA) to 60 CF-1 mice/sex. A statistically significant increase in incidence of lung tumors was seen in both sexes compared with controls. In males, a statistically significant increase in incidence of liver tumors was also seen.

NCI (1978) fed DDD at 411 and 822 ppm (TWA) to 50 B6C3F1 mice/sex/dose for 78 weeks. Actual doses were 350 or 630 ppm for 5 weeks, 375 or 750 ppm for 11 weeks, and 425 or 850 ppm for the next 62 weeks. After an additional 15 weeks, an increased incidence of hepatocellular carcinomas was seen in both sexes by comparison to controls, but the increase was not statistically significant.

NCI (1978) also fed DDD at 1647 and 3294 ppm TWA for males and 850 and 1700 ppm TWA for females for 78 weeks to 50 Osborne-Mendel rats/sex/dose. Males were fed 1400 or 2800 ppm for 23 weeks followed by 1750 or 3500 ppm for 55 weeks. Females were fed 850 or 1700 ppm for the entire 78 weeks. After an additional 35 weeks, an increased incidence of thyroid tumors (follicular cell adenomas and carcinomas) was observed in males. Due to a wide variation in incidence of these tumors in the control groups for DDD, DDE and DDT, the increased incidence was not statistically significant by comparison to concurrent controls. Although tumor incidence did not appear to be dose-related, the increase was significant at the low dose by comparison to historical controls. Thus, the pathologists' judgment and statistical results suggest a possible carcinogenic effect of DDD in male rats. NCI concluded that a definitive interpretation of the data was not possible.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

DDD is structurally similar to, and is a metabolite of, DDT, a probable human carcinogen, in rats (Peterson and Robinson, 1964), mice (Gingell and Wallcave, 1976), and humans (Morgan and Roan, 1977).

Positive effects were found with DDD in mammalian cytogenetic assays and a host-mediated assay (ICPEMC, 1984).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 2.4E-1/mg/kg/day

Drinking Water Unit Risk -- 6.9E-6/ug/L

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

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

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- liver
Test Animals -- mouse/CF-1, males
Route -- diet
Reference -- Tomatis et al., 1974

Administered Dose (ppm)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0	0	33/98
250	245	31/59

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

DDD used in the Tomatis study was 99% pure p,p'-isomer. In the NCI bioassay, technical grade DDD was used, in which 60% of the material consisted of the p,p'-isomer. The composition of the remaining 40% was unspecified, but it was stated that analysis by gas chromatography revealed at least 19 impurities.

The unit risk should not be used if the water concentration exceeds 1E+3 ug/L, since above this concentration the slope factor may differ from that stated.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

An adequate number of animals was tested. The slope factor was calculated using tumor incidence data from only one dose. The slope factor was similar to, and within a factor of 2, of the slope factors for this same site of three other structurally similar compounds: DDT, 3.4E-1/mg/kg/day; DDE, 3.4E-1/mg/kg/day; and dicofol, 4.4E-1/mg/kg/day.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

Not available

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1980, 1985

The 1985 Carcinogen Assessment Group's report has received Agency review.

The 1980 Hazard Assessment Report has received peer review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 06/03/87, 06/24/87

Verification Date -- 06/24/87

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

==========

_VI. BIBLIOGRAPHY

Substance Name -- p,p'-Dichlorodiphenyl dichloroethane (DDD)
CASRN -- 72-54-8
Last Revised -- 08/01/89

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfD REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Casarett, L.J., G.C. Fryer, W.L. Yauger, Jr. and H. Klemmer. 1968. Organochlorine pesticide residues in human tissue. Hawaii. Arch. Environ. Health. 17: 306-311.

Dacre, J.C. and R.W. Jennings. 1970. Organochlorine insecticides in normal and carcinogenic human lung tissues. Toxicol. Appl. Pharmacol. 17: 277.

Gingell, R. and L. Wallcave. 1976. Metabolism of 14C-DDT in the mouse and hamster. Xenobiotica. 6: 15.

Hoffman, W.S., H. Adler, W.I. Fishbein and F.C. Bauer. 1967. Relation of pesticide concentrations in fat to pathological changes in tissues. Arch. Environ. Health. 15: 758-765.

ICPEMC (International Commission for Protection Against Environmental Mutagens and Carcinogens). 1984. Report of ICPEMC task group 5 on the differentiation between genotoxic and nongenotoxic carcinogens. ICPEMC Publication No. 9. Mutat. Res. 133: 1-49.

Maier-Bode, H. 1960. DDT in Koperfett des Menschen. Med. Exp. 1: 132-137. (Russian)

Morgan, D.P. and C.C. Roan. 1977. The metabolism of DDT in man. Essays Toxicol. 5: 39.

NCI (National Cancer Institute). 1978. Bioassay of DDT, TDE and p,p'-DDE for possible carcinogenicity. NCI Report No. 131. DHEW Publ. No. (NIH) 78-1386.

Peterson, J.R. and W.H. Robinson. 1964. Metabolic products of p.p'-DDT in the rat. Toxicol. Appl. Pharmacol. 6: 321.

Robinson, J., A. Richardson, C.G. Hunter, A.N. Crabtree and H.J. Rees. 1965. Organochlorine insecticide content of human adipose tissue in south-eastern England. Br. J. Ind. Med. 22: 220-224.

Tomatis, L., V. Turusov, R.T. Charles and M. Boicchi. 1974. Effect of long-

term exposure to 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene, to 1,1-dichloro-2,2-bis(p-chlorophenyl)-ethane, and to the two chemicals combined on CF-1 mice. J. Natl. Cancer Inst. 52(3): 883-891.

U.S. EPA. 1980. Hazard Assessment Report on DDT, DDD, DDE. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 1985. The Carcinogenic Assessment Group's Calculation of the Carcinogenicity of Dicofol (Kelthane), DDT, DDE and DDD (TDE). Prepared by the Office of Health and Environmental Assessment, carcinogen Assessment Group, Washington, DC, for the Hazard Evaluation Division, Office of Toxic Substances, Washington, DC. (Internal Report) EPA-600/X-85-097.

Wasserman, M., D.P. Nogueira, L. Tomatis, et al. 1976. Organochlorine compounds in neoplastic and adjacent apparently normal breast tissue. Bull. Environ. Contam. Toxicol. 15: 478-484.

==========

VII. REVISION HISTORY

Substance Name -- p,p'-Dichlorodiphenyl dichloroethane (DDD)
CASRN -- 72-54-8

Date	Section	Description
08/22/88	II.	Carcinogen summary on-line
08/01/89	VI.	Bibliography on-line
01/01/92	IV.	Regulatory Action section on-line

==========

SYNONYMS

Substance Name -- p,p'-Dichlorodiphenyl dichloroethane (DDD)
CASRN -- 72-54-8
Last Revised -- 08/22/88

72-54-8
1,1-bis(4-chlorophenyl)-2,2-dichloroethane
1,1-bis(p-chlorophenyl)-2,2-dichloroethane
2,2-bis(p-chlorophenyl)-1,1-dichloroethane
DDD
4,4'-DDD
p,p'-DDD
1,1-dichloro-2,2-bis(p-chlorophenyl)ethane
dichlorodiphenyl dichloroethane
Dichlorodiphenyl dichloroethane, p,p'-
dilene
rothane
TDE
p,p'-TDE

0225
Dieldrin; CASRN 60-57-1 (03/01/97)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Dieldrin

File On-Line 09/07/88

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	09/01/90
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	07/01/93

==========

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Dieldrin
CASRN -- 60-57-1
Last Revised -- 09/01/90

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
Liver lesions 2-Year Rat Feeding Study Walker et al., 1969	NOAEL: 0.1 ppm (0.005 mg/kg/day) LOAEL: 1.0 ppm (0.05 mg/kg/day)	100	1	5E-5 mg/kg/day

---

*Conversion Factors: 1 ppm = 0.05 mg/kg/day (assumed rat food consumption)

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Walker, A.I.T., D.E. Stevenson, J. Robinson, R. Thorpe and M. Roberts. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-year oral exposures of rats and dogs. Toxicol. Appl. Pharmacol. 15: 345-373.

Walker et al. (1969) administered dieldrin (recrystallized, 99% active ingredient) to Carworth Farm "E" rats (25/sex/dose; controls 45/sex) for 2 years at dietary concentrations of 0, 0.1, 1.0, or 10.0 ppm. Based on intake assumptions presented by the authors, these dietary levels are approximately equal to 0, 0.005, 0.05 and 0.5 mg/kg/day. Body weight, food intake, and general health remained unaffected throughout the 2-year period, although at 10.0 ppm (0.5 mg/kg/day) all animals became irritable and exhibited tremors and occasional convulsions. No effects were seen in various hematological and clinical chemistry parameters. At the end of 2 years, females fed 1.0 and 10.0 ppm (0.05 and 0.5 mg/kg/day) had increased liver weights and liver-to-body weight ratios ($p<0.05$). Histopathological examinations revealed liver parenchymal cell changes including focal proliferation and focal hyperplasia. These hepatic lesions were considered to be characteristic of exposure to an organochlorine insecticide. The LOAEL was identified as 1.0 ppm (0.005 mg/kg/day) and the NOAEL as 0.1 ppm (0.005 mg/kg/day).

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The UF of 100 allows for uncertainty in the extrapolation of dose levels from laboratory animals to humans (10A) and uncertainty in the threshold for sensitive humans (10H).

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Data considered for establishing the RfD:

1) 2-Year Feeding - rat: Principal study - see previous description

2) 2-Year Feeding (oncogenic) - dog: Systemic NOEL=0.005 mg/kg/day; LEL= 0.05 mg/kg/day (increased liver weight and liver/body weight ratios, increased plasma alkaline phosphatase, and decreased serum protein concentration) (Walker et al., 1969)

3) 2-Year Feeding - rat: Systemic LEL=0.5 ppm (approximately 0.025 mg/kg/day), (liver enlargement with histopathology); (Fitzhugh et al., 1964)

4) 2-Year Feeding (oncogenic) - mouse: Systemic LEL=0.1 ppm (0.015 mg/kg/day), (liver enlargement with histopathology); (Walker et al., 1972)

5) 25-Month Feeding - dog: Systemic NOEL=0.2 mg/kg/day; LEL=0.5 mg/kg/day, (weight loss and convulsions); (Fitzhugh et al., 1964)

6) Teratology - mouse: Teratogenic NOEL=6.0 mg/kg/day (HDT, gestational days 7-16); Maternal LEL=6.0 mg/kg/day (HDT, decrease in maternal weight gain); Fetotoxic LEL=6.0 mg/kg/day (HDT, decreased numbers of caudal ossification centers and increases in supernumerary ribs); (Chernoff et al., 1975). This study was not considered since 41% of the test dams died at the highest dose tested.

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Low
Data Base -- Medium
RfD -- Medium

The principal study is an older study for which detailed data are not available and in which a wide range of doses was tested. The chronic toxicity evaluation is relatively complete and supports the critical effect, if not the magnitude of effects. Reproductive studies are lacking. The RfD is given a medium confidence rating because of the support for the critical effect from other dieldrin studies, and from studies on organochlorine insecticides in general.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1987

Other EPA Documentation -- None

Agency Work Group Review -- 04/16/87

Verification Date -- 04/16/87

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- Dieldrin
CASRN -- 60-57-1

Not available at this time.

==========

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Dieldrin
CASRN -- 60-57-1
Last Revised -- 07/01/93

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.

___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Two studies of workers exposed to aldrin and to dieldrin reported no increased incidence of cancer. Both studies were limited in their ability to detect an excess of cancer deaths. Van Raalte (1977) observed two cases of cancer (gastric and lymphosarcoma) among 166 pesticide manufacturing workers exposed 4-19 years and followed from 15-20 years. Exposure was not quantified, and workers were also exposed to other organochlorine pesticides (endrin and telodrin). The number of workers studied was small, the mean age of the cohort (47.7 years) was young, the number of expected deaths was not calculated, and the duration of exposure and of latency was relatively short.

In a retrospective mortality study, Ditraglia et al. (1981) reported no statistically significant excess in deaths from cancer among 1155 organochlorine pesticide manufacturing workers [31 observed vs. 37.8 expected, Standardized Mortality Ratio (SMR) = 82]. Workers were employed for 6 months or more and followed 13 years or more (24,939 person-years). Workers with no exposure (for example, office workers) were included in the cohort. Vital status was not known for 112 or 10% of the workers, and these workers were assumed to be alive; therefore additional deaths may have occurred but were not observed. Exposure was not quantified and workers were also exposed to other chemicals and pesticides (including endrin). Increased incidences of deaths from cancer were seen at several specific sites: esophagus (2 deaths observed, SMR = 235); rectum (3, SMR = 242); liver (2, SMR = 225); and lymphatic and hematopoietic system (6, SMR = 147), but these site-specific incidences were not statistically significantly increased.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Dieldrin has been shown to be carcinogenic in various strains of mice of both sexes. At different dose levels the effects range from benign liver tumors, to hepatocarcinomas with transplantation

confirmation, to pulmonary metastases.

The Food and Drug Administration (FDA) conducted a long-term carcinogenesis bioassay for dieldrin (Davis and Fitzhugh, 1962). Ten ppm dieldrin was administered orally to 218 male and female C3HeB/Fe mice for 2 years. The study was compromised by the poor survival rate, lack of detailed pathology, loss of a large percentage of the animals to the study, and failure to treat the data for males and females separately. A statistically significant increase in incidence of hepatomas was observed in the treated groups versus the control groups in both males and females. In FDA follow-up study, Davis (1965) examined 100 male and 100 female C3H mice which had been orally administered 10 ppm dieldrin. The same limitations as the previous study were reported. The incidence of benign hepatomas and hepatic carcinomas was significantly increased in the dieldrin group. A reevaluation of the histological material of both studies was done by Reuber in 1974 (Epstein, 1975a,b; 1976). He concluded that the hepatomas were malignant and that dieldrin was hepatocarcinogenic for male and female C3HeB/Fe and C3H mice.

Walker et al. (1972) conducted several studies of dieldrin in CF1 mice of both sexes. Dieldrin was administered orally at concentrations of 0, 0.1, 1.0, and 10 ppm. Treatment groups varied from 87 to 288 animals of each sex. Surviving animals were sacrificed during weeks 132-140. Incidence of tumors was related to the number of dose levels and the dose administered. Effects were detected at the lowest dieldrin level tested (0.1 ppm) in both male and female mice. Dieldrin also produced significant increases (<0.05) in the incidence of pulmonary adenomas, pulmonary carcinomas, lymphoid tumors, and "other" tumors in female mice.

Diets containing 10 ppm dieldrin were fed to groups of 30 CF1 mice of both sexes for 110 weeks (Thorpe and Walker, 1973). The control group consisted of 45 mice of both sexes. A statistically significant increase ($p<0.01$) in incidence of liver tumors was found in both sexes of treated animals relative to controls. The liver tumors appeared much earlier in treated animals than controls.

Technical-grade dieldrin (>96%) was fed to B6C3F1 mice (50/sex/dose) at TWA doses of 0, 2.5, or 5 ppm for 80 weeks followed by an observation period of 10 to 13 weeks (NCI, 1978a). Matched control groups consisted of 20 untreated males and 10 untreated females. No significant difference in survival was noted. A significant dose-related increase in hepatocellular carcinoma was found in male mice when compared with pooled controls.

Tennekes et al. (1981) fed groups of 19 to 82 male CF1 mice control or dieldrin-supplemented (10 ppm) diets or control diets for 110 weeks. Dieldrin produced a statistically significant increased incidence of hepatocellular carcinomas in the treated group.

Dieldrin (>99%) was continuously fed in the diet for 85 weeks to 50 C3H/He, 62 B6C3Fl, and 71 C57Bl/6J male mice (Meierhenry et al., 1983). Controls were 50 to 76 males of each strain. Dieldrin produced a significant increase in the incidence of hepatocellular carcinomas compared with controls in all three strains.

Seven studies with four strains of rats fed 0.1 to 285 ppm dieldrin varying in duration of exposure from 80 weeks to 31 months did not produce positive results for carcinogenicity (Treon and Cleveland, 1955; Fitzhugh et al., 1964; Song and Harville, 1964; Walker et al., 1969; Deichmann et al., 1970; NCI, 1978a,b). Three of these studies used Osborne-Mendel rats, two studies used Carworth rats, and one each used Fischer 344 and Holtzman strains. Only three of the seven studies are considered adequate in design and conduct. The others used too few animals, had unacceptably high levels of mortality, were too short in duration, and/or had inadequate pathology examination or reporting.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Dieldrin causes chromosomal aberrations in mouse cells (Markaryan, 1966; Majumdar et al., 1976) and in human lymphoblastoid cells (Trepanier et al., 1977), forward mutation in Chinese hamster V79 cells (Ahmed et al., 1977), and unscheduled DNA synthesis in rat (Probst et al., 1981) and human cells (Rocchi et al., 1980). Dieldrin did not produce responses in 13 other mutagenicity tests. Negative responses were given in assays for gene conversion in S. cerevisiae, back-mutation in S. marcesans, forward mutation (Gal Rz2 in E. coli), and forward mutation to streptomycin resistance in E. coli (Fahrig, 1974). Negative responses were produced in reverse mutation assays with six strains of S. typhimurium with or without metabolic activation (Bidwell et al., 1975; Marshall et al., 1976; Shirasu et al., 1976; Wade et al., 1979; Haworth et al., 1983). Majumdar et al. (1977), however, reported that dieldrin was mutagenic for S. typhimurium with and without metabolic activation.

Five compounds structurally related to dieldrin - aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorondic acid - have induced malignant liver tumors in mice. Chlorendic acid has also induced liver tumors in rats.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 1.6E+1 per (mg/kg)/day

Drinking Water Unit Risk -- 4.6E-4 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-1 ug/L
E-5 (1 in 100,000)	2E-2 ug/L
E-6 (1 in 1,000,000)	2E-3 ug/L

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- liver carcinoma
Test Animals -- mouse
Route -- diet
Reference -- see table

Sex/Strain	Slope Factor	Reference
Male, C3H	22	Davis (1965), reevaluated by Reuber, 1974 (cited in Epstein, 1975a)
Female, C3H	25	Davis (1965), reevaluated by Reuber, 1974 (cited in Epstein, 1975a)

Male, CF1	25	Walker et al. (1972)
Female, CF1	28	Walker et al. (1972)
Male, CF1	15	Walker et al. (1972)
Female, CF1	7.1	Walker et al. (1972)
Male, CF1	55	Thorpe and Walker (1973)
Female, CF1	26	Thorpe and Walker (1973)
Male, B6C3F1	9.8	NCI (1978a,b)
Male, CF1	18	Tennekes et al. (1981)
Male, C57B1/6J	7.4	Meierhenry et al. (1983)
Male, C3H/He	8.5	Meierhenry et al. (1983)
Male, B6C3F1	11	Meierhenry et al. (1983)

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

The slope factor is the geometric mean of 13 slope factors calculated from liver carcinoma data in both sexes of several strains of mice. Inspection of the data indicated no strain or sex specificity of carcinogenic response.

The unit risk should not be used if the water concentration exceeds 20 ug/L, since above this concentration the unit risk may not be appropriate.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

The individual slope factors calculated from 13 independent data sets range within a factor of 8.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 4.6E-3 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-2 ug/cu.m
E-5 (1 in 100,000)	2E-3 ug/cu.m
E-6 (1 in 1,000,000)	2E-4 ug/cu.m

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Calculated from oral data in Section II.B.2.

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The unit risk should not be used if air concentrations exceed 2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

This inhalation risk estimate was based on oral data.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1986

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 03/05/87

Verification Date -- 03/05/87

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

==========

_VI. BIBLIOGRAPHY

Substance Name -- Dieldrin
CASRN -- 60-57-1
Last Revised -- 09/01/90

__VI.A. ORAL RfD REFERENCES

Chernoff, N., R.J. Kavlock, J.R. Kathrein, J.M. Dunn and J.K. Haseman. 1975. Prenatal effects of dieldrin and photodieldrin in mice and rats. Toxicol. Appl. Pharmacol. 31: 302-308.

Fitzhugh, O.G., A.A. Nelson and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

U.S. EPA. 1987. Dieldrin: Health Advisory. Office of Drinking Water, Washington, DC. NTIS PB 88-113543/AS.

Walker, A.I.T., D.E. Stevenson, J. Robinson, E. Thorpe and M. Roberts. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-year oral exposures of rats and dogs. Toxicol. Appl. Pharmacol. 15: 345-373.

Walker, A.I.T., E. Thorpe and D.E. Stevenson. 1972. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. Food Cosmet. Toxicol. 11: 415-432.

__VI.B. INHALATION RfD REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Ahmed, F.E., R.W. Hart and N.J. Lewis. 1977. Pesticide induced DNA damage and its repair in cultured human cells. Mutat. Res. 42: 161-174.

Bidwell, K., E. Weber, I. Neinhold, T. Connor and M.S. Legator. 1975. Comprehensive evaluation for mutagenic activity of dieldrin. Mutat. Res. 31: 314. (Abstract)

Davis, K.J. 1965. Pathology report on mice fed aldrin, dieldrin, heptachlor or heptachlor epoxide for two years. Internal FDA memorandum to Dr. A.J. Lehman. July 19. (Cited in: U.S. EPA, 1986)

Davis, K.J. and O.G. Fitzhugh. 1962. Tumorigenic potential of aldrin and dieldrin for mice. Toxicol. Appl. Pharmacol. 4: 187-189.

Deichmann, W.B., W.E. MacDonald, E. Blum, et al. 1970. Tumorigenicity of aldrin, dieldrin and endrin in the albino rat. Ind. Med. Surg. 39: 426-434.

Ditraglia, D., D.P. Brown, T. Namekata and M. Iverson. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. Scand. J. Work. Env. Health. 7 (Suppl. 4): 140-146.

Epstein, S.S. 1975a. The carcinogenicity of dieldrin. Part 1. Sci. Total Environ. 4: 1-52.

Epstein, S.S. 1975b. The carcinogenicity of dieldrin. Part 2. Sci. Total Environ. 4: 205-217.

Epstein, S.S. 1976. Case study 5: Aldrin and dieldrin suspension based on experimental evidence and evaluation and societal needs. Ann. NY. Acad. Sci. 271: 187-195.

Fahrig, R. 1974. Comparative mutagenicity studies with pesticides. IARC Scientific Press No. 10.

Fitzhugh, O.G., A.A. Nelson and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

Haworth, S., T. Lawlor, K. Mortelmans, W. Speck and E. Zeigler. 1983.

Salmonella mutagenicity test results for 250 chemicals. Environ. Mutag. 5(Suppl. 1): 1-142.

Majumdar, S.K., H.A. Kopelman and M.J. Schnitman. 1976. Dieldrin-induced chromosome damage in mouse bone-marrow and WI-38 human lung cells. J. Hered. 67: 303-307.

Majumdar, S.K., L.G. Maharam and G.A. Viglianti. 1977. Mutagenicity of dieldrin in the Salmonella-microsome test. J. Hered. 68: 184-185.

Markaryan, D.S. 1966. Cytogenic effect of some chlorinated insecticides on mouse bone-marrow cell nuclei. Soviet Genetics. 2(1): 80-82.

Marshall, T.C., H.W. Dorough and H.E. Swim. 1976. Screening of pesticides for mutagenic potential using Salmonella typhimurium mutants. J. Agric. Chem. 24: 560-563.

Meierhenry, E.F., B.H. Reuber, M.E. Gershwin, L.S. Hsieh and S.W. French. 1983. Deildrin-induced mallory bodies in hepatic tumors of mice of different strains. Hepatology. 3: 90-95.

NCI (National Cancer Institute). 1978a. Bioassays of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-821. National Cancer Institute Carcinogenesis Technical Report Series, No. 21. NCI-CG-TR-21.

NCI (National Cancer Institute). 1978b. Bioassays of aldrin and dieldrin for possible carcinogenicity. DHEW Publication No. (NIH) 78-822. National Cancer Institute Carcinogenesis Technical Report Series, No. 22. NCI-CG-TR-22.

Probst, G.S., R.E. McMahon, L.W. Hill, D.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 chemicals. Environ. Mutagen. 3: 11-32.

Reuber, M.D. 1974. Exhibit 42. Testimony at hearings on aldrin/dieldrin. (Cited in: Epstein, 1975a)

Rocchi, P., P. Perocco, W. Alberghini, A. Fini and G. Prodi. 1980. Effect of pesticides on scheduled and unscheduled DNA synthesis of rat thymocytes and human lymphocytes. Arch. Toxicol. 45: 101-108.

Shirasu, Y., M. Moriya, K. Kato, A. Furuhashi and T. Kada. 1976. Mutagenicity screening of pesticides in the microbial system. Mutat. Res. 40(1): 19-30.

Song, J. and W.E. Harville. 1964. Carcinogenicity of aldrin and dieldrin in mouse and rat liver. Fed. Proc. Fed. Am. Soc. Exp. Biol. 23: 336.

Tennekes, H.A., A.S. Wright, K.M. Dix and J.H. Koeman. 1981. Effects of dieldrin, diet, and bedding on enzyme function and tumor incidence in livers of male CF-1 mice. Cancer Res. 41: 3615-3620.

Thorpe, E. and A.I.T. Walker. 1973. The toxicology of dieldrin (HEOD). Part II. Comparative long-term oral toxicology studies in mice with dieldrin, DDT, phenobarbitone, beta-BHC and gamma-BHC. Food Cosmet. Toxicol. 11: 433-441.

Treon, J.F. and F.P. Cleveland. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. Agric. Food Chem. 3: 402-408.

Trepanier, G., F. Marchessault, J. Bansal and A. Chagon. 1977. Cytological effects of insecticides on human lymphoblastoid cell line. In Vitro. 13: 201.

U.S. EPA. 1986. Carcinogenicity Assessment of Aldrin and Dieldrin. Prepared

by Carcinogen Assessment Group, Office of Health and Environmental Assessment, Washington, DC for Hazard Evaluation Division, Office of Pesticide Programs, Office of Pesticides and Toxic Substances. OHEA-C-205.

Van Raalte, H.G.S. 1977. Human experience with dieldrin in perspective. Ecotox. Environ. Saf. 1: 203-210.

Wade, M.J., J.W. Moyer and C.H. Hine. 1979. Mutagenic action of a series of epoxides. Mutat. Res. 66(4): 367-371.

Walker, A.I.T., D.E. Stevenson, J. Robinson, E. Thorpe and M. Roberts. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two year oral exposures of rats and dogs. Toxicol. Appl. Pharmacol. 15: 345-373.

Walker, A.I.T., E. Thorpe and D.E. Stevenson. 1972. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. Food Cosmet. Toxicol. 11: 415-432.

==========

_VII. REVISION HISTORY

Substance Name -- Dieldrin
CASRN -- 60-57-1

Date	Section	Description
09/07/88	I.A.	Oral RfD summary on-line
09/07/88	II.	Carcinogen summary on-line
03/01/90	II.A.2.	Ditraglia citation clarified
03/01/90	II.A.3.	Reuber citation year and Deichman spelling corrected
03/01/90	II.A.4.	Shirasu citation year corrected
03/01/90	II.B.2.	Reuber citation year corrected
03/01/90	VI.	Bibliography on-line
04/01/90	VI.C.	Treon and Cleveland, 1955 citation corrected
09/01/90	I.A.	Text edited
09/01/90	II.	Text edited
09/01/90	III.A.	Health Advisory on-line
09/01/90	VI.	Health Advisory references added
01/01/91	II.	Text edited
01/01/91	II.C.1.	Inhalation slope factor removed (global change)
01/01/92	IV.	Regulatory Action section on-line
07/01/93	II.D.3.	Secondary contact's phone number changed

==========

SYNONYMS

Substance Name -- Dieldrin
CASRN -- 60-57-1
Last Revised -- 09/07/88

60-57-1
ALVIT
COMPOUND 497
DIELDREX
Dieldrin

DIELDRINE
DIELDRITE
1,4:5,8-DIMETHANONAPHTHALENE, 1,2,3,4,10,10-HEXACHLORO-6,7-EPOXY-1,4,4a,5,6,7,
8,8a-OCTAHYDRO, endo,exo-
ENT 16,225
HEOD
HEXACHLOROEPOXYOCTAHYDRO-endo,exo-DIMETHANONAPHTHALENE
3,4,5,6,9,9-HEXACHLORO-1a,2,2a,3,6,6a,7,7a-OCTAHYDRO-2,7:3,6-DIMETHANONAPHTH
(2,3-b)OXIRENE
ILLOXOL
NA 2761
NCI-C00124
OCTALOX
PANORAM D-31
QUINTOX
RCRA WASTE NUMBER P037

0142
Chlordane; CASRN 57-74-9 (04/01/97)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Chlordane

File On-Line 03/31/87

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	07/01/89
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	07/01/93

==========

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Chlordane
CASRN -- 57-74-9
Last Revised -- 07/01/89

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
Regional liver hypertrophy in females	NOEL: 1 ppm (0.055 mg/kg/day)	1000	1	6E-5 mg/kg/day
30-Month Rat Feeding Study	LEL: 5 ppm (0.273 mg/kg/day)			

Velsicol Chemical Co.,
1983a

---

*Conversion Factors: Actual dose tested

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Velsicol Chemical Company. 1983a. MRID No. 00138591, 00144313. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Charles River Fischer 344 rats (80/sex/dose) were fed technical chlordane at dietary levels of 0, 1, 5, and 25 ppm for 130 weeks. Body weight, food consumption, and water uptake were monitored at regular intervals. Clinical laboratory studies were performed and organ weights measured on eight animals/sex/group at weeks 26 and 52, and on all survivors at week 130. Gross and microscopic pathology were performed on all tissues. Daily dose level of 0.045, 0.229, and 1.175 mg/kg/day for males and 0.055, 0.273, and 1.409 mg/kg/day for females for the 1, 5, and 25 ppm treatment groups, respectively, were calculated from food consumption and body weight data.

Following the submission of a 30-month chronic feeding/oncogenicity study in Fischer 344 rats, the Agency reviews by the Office of Pesticides Programs and the Cancer Assessment Group of these data indicated that male rats at the highest dosage exhibited an increase in liver tumors (ICF Clement, 1987). The registrant, Velsicol Chemical Company, subsequently convened the Pathology Working Group to reevaluate the slides of livers of the chlordane-treated rats reported in MRID No. 00138591. It was concluded that liver lesions had not occurred in male rats and that 25 ppm (0.1175 mg/kg/day) was the NOEL for males. Liver lesions (hypertrophy), however, had occurred in female rats at 5 ppm (0.273 mg/kg/day), which was considered an LEL. Therefore an NOEL of 1 ppm (0.055 mg/kg/day) (LDT) was established for female rats.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- An uncertainty factor of 100 was used to account for the inter- and intraspecies differences. An additional UF of 10 was used to account for the lack of an adequate reproduction study and adequate chronic study in a second mammalian species, and the generally inadequate sensitive endpoints studied in existing studies, particularly since chlordane is known to bioaccumulate over a chronic duration.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Data Considered for Establishing the RfD

1) 30-Month Feeding (oncogenic) - rat: Principal study - see previous description; core grade minimum

2) 24-Month Chronic Toxicity - mouse: NOEL=1 ppm (0.15 mg/kg/day); LEL=5 ppm (0.75 mg/kg/day) (hepatocellular swelling and necrosis in males; hepatocyte swelling in males, and increased live weight in males and females); At 12.5 ppm (1.875 mg/kg/day) (HDT); core grade minimum (Velsicol Chemical Co., 1983b)

Data Gap(s): Chronic Dog Feeding Study, Rat Reproduction Study, Rat Teratology Study, Rabbit Teratology Study

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Medium
Data Base -- Low
RfD -- Low

The critical study is of adequate quality and is given a medium rating. The data base is given a low confidence rating because of 1) the lack of an adequate reproduction study and adequate chronic study in a second mammalian species and 2) inadequate sensitive endpoints studied in existing studies, particularly since chlordane is known to bioaccumulate over a chronic duration. Low confidence in the RfD follows.

___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 -- Pesticide Registration Standard, November 1986; Pesticide Registration Files

Agency Work Group Review -- 12/18/85, 03/22/89

Verification Date -- 03/22/89

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- Chlordane
CASRN -- 57-74-9

Not available at this time.

==========

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Chlordane
CASRN -- 57-74-9
Last Revised -- 07/01/93

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Sufficient evidence in studies in which benign and malignant liver tumors were induced in four strains of mice of both sexes and in F344 male rats; structurally related to other liver carcinogens

___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. There were 11 case reports involving central nervous system effects, blood dyscrasias and neuroblastomas in children with pre-/postnatal exposure to chlordane and heptachlor (Infante et al., 1978). As no other information was available, no conclusions can be drawn.

There were three epidemiologic studies of workers exposed to chlordane and/or heptachlor. One study of pesticide applicators was considered inadequate in sample size and duration of follow-up. This study showed marginal statistically significant increased mortality from bladder cancer (3 observed) (Wang and McMahon, 1979a). The other two studies were of pesticide manufacturing workers. Neither of them showed any statistically significantly increased cancer mortality (Wang and McMahon, 1979b; Ditraglia et al., 1981). Both these populations also had confounding exposures from other chemicals.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Chlordane has been studied in four mouse and four rat long-term carcinogenesis bioassays. Dose-related incidences of liver carcinoma constitute the major finding in mice. Becker and Sell (1979) tested chlordane (90:10 mixture of chlordane to heptachlor) in C57B1/6N mice, a strain historically known not to develop spontaneous liver tumors. An unspecified number of mice were fed chlordane at 0, 25 and 50 ppm (0, 3.57, 7.14 mg/kg bw) for 18 months. None of the controls developed tumors or nodular lesions of the liver. Twenty-seven percent (16 mice) of the surviving treated mice developed primary hepatocellular carcinomas. Velsicol (1973) fed groups of 100 male and 100 female CD-1 mice diets with 0, 5, 25 or 50 ppm analytical grade chlordane for 18 months. A significant ($p<0.01$) dose-related increase in nodular hyperplasias in the liver of male and female mice was reported at the the two highest dose levels. A histological review by Reuber (U.S. EPA, 1985) reported a high incidence ($p<0.01$) of hepatic carcinomas instead of hyperplastic nodules at 25 and 50 ppm.

A dose-related increase ($p<0.001$ after lifetable adjustment) of

hepatocellular carcinomas was also observed in both sexes of B6C3F1 mice (NCI, 1977). Male and female mice were fed technical-grade chlordane (purity\= 94.8%) at TWA concentrations (TWAC) of 29.9 and 56.2 ppm and 30.1 and 63.8 ppm, respectively, for 80 weeks. In this study there were individual matched controls for the low and high dose groups. ICR male mice developed hepatocellular adenomas and hemangiomas when fed 12.5 ppm chlordane for 24 months. No tumors were observed in the female mice when tested at the same concentrations: 0, 1, 5, and 12.5 ppm (Velsicol, 1983a).

Velsicol (1983b) reported a long-term (130 weeks) carcinogenesis bioassay on 80 male and 80 female F344 rats fed concentrations of 0, 1, 5, and 25 ppm chlordane. A significant increase in adenomas of the liver was observed in male rats receiving 25 ppm. Although no tumors were observed in female rats, hepatocellular swelling was significantly increased at 25 ppm. The NCI (1977) reported a significant increase ($p<0.05$) of neoplastic nodules of the liver in low-dose Osborne-Mendel female rats (TWAC of 120.8 ppm) but not in the high-dose group (TWAC of 241.5 ppm). No tumor incidence was reported for the males fed TWAC of 203.5 and 407 ppm. Loss of body weight and a dose-related increase in mortality was observed in all treated groups. High mortality and reduced growth rates in Osborne-Mendel rats was also observed by Ingle (1952) when the rats were exposed to 150 and 300 ppm chlordane but not at 5, 10, and 30 ppm. No treatment-related incidence of tumors was reported. Significantly enlarged livers and liver lesions were found in male and female albino rats fed chlordane at greater than or equal to 80 ppm (Ambrose et al., 1953a,b). No treatment-related increase in tumors was found, but the study duration (400 days) was short.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Gene mutation assays indicate that chlordane is not mutagenic in bacteria (Wildeman and Nazar, 1982; Probst et al., 1981; Gentile et al., 1982). Positive results have been reported in Chinese hamster lung V79 cells and mouse lymphoma L5178Y cells with and without exogenous metabolism, as well as in plant assays. Chlordane did not induce DNA repair in bacteria, rodent hepatocytes (Maslansky and Williams, 1981), or human lymphoid cells (Sobti et al., 1983). It is a genotoxicant in yeast (Gentile et al., 1982; Chambers and Dutta, 1976), human fibroblasts (Ahmed et al., 1977), and fish (Vigfusson et al., 1983).

Five compounds structurally related to chlordane (aldrin, dieldrin, heptachlor, heptachlor epoxide, and chlorendic acid) have produced liver tumors in mice. Chlorendic acid has also produced liver tumors in rats.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 1.3E+0 per (mg/kg)/day

Drinking Water Unit Risk -- 3.7E-5 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E+0 ug/L

E-5 (1 in 100,000)	3E-1 ug/L
E-6 (1 in 1,000,000)	3E-2 ug/L

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- hepatocellular carcinoma
Test Animals -- mouse/CD-1 (Velsicol); mouse/B6C3F1 (NCI)
Route -- diet
Reference -- Velsicol, 1973; NCI, 1977

Administered Dose (ppm)	Human Equivalent Dose (mg/kg-day)	Tumor Incidence	Reference
female			
0	0.000	0/45	Velsicol,
5	0.052	0/61	1973
25	0.260	32/50	
50	0.520	26/37	
male			
0	0.000	3/33	Velsicol,
5	0.052	5/55	1973
25	0.260	41/52	
50	0.520	32/39	
male			
0	0.00	2/18	NCI, 1977
29.9	0.31	16/48	
56.2	0.58	43/49	
female			
0	0.00	0/19	NCI, 1977
30.1	0.31	3/47	
63.8	0.66	34/49	

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Four data sets for mice and one data set for rats showed a significant increase in liver tumors; namely hepatocellular carcinomas in mice (NCI, 1977; Velsicol, 1973) and hepatocellular adenomas in rats (Velsicol, 1983a). The quantitative estimate is based on the geometric mean from the four mouse data sets as mice were the more sensitive species tested and as risk estimates for a similar compound (heptachlor) were similarly derived from mouse tumor data. The slope factors for the data sets are these: 2.98 per (mg/kg)/day for CD-1 female mice, 4.74 per (mg/kg)/day for CD-1 male mice, 0.76 per (mg/kg)/day for B6C3F1 male mice, and 0.25 per (mg/kg)/day for B6C3F1 female mice. Low and high dose groups in the NCI (1977) study had individual matched controls.

The unit risk should not be used if the water concentration exceeds 300 ug/L, since above this concentration the unit risk may not be appropriate.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Liver carcinomas were induced in mice of both sexes in two studies. An adequate number of animals was observed, and dose-response effects were reported in all studies. The geometric mean of slope factors (0.25 to 4.74 per (mg/kg)/day for the most sensitive species is consistent with that derived from rat data (1.11/mg/kg/day).

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 3.7E-4 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E-1 ug/cu.m
E-5 (1 in 100,000)	3E-2 ug/cu.m
E-6 (1 in 1,000,000)	3E-3 ug/cu.m

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

The inhalation risk estimates were calculated from the oral data presented in II.B.2.

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The unit risk should not be used if the air concentration exceeds 30 ug/cu.m, above this concentration the unit risk may not be appropriate.

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

See II.B.4.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1986, 1985

The values in the 1986 Carcinogenicity Assessment for Chlordane and Heptachlor/Heptachlor Epoxide have been reviewed by the Carcinogen Assessment Group.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 04/01/87

Verification Date -- 04/01/87

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

==========

_VI. BIBLIOGRAPHY

Substance Name -- Chlordane
CASRN -- 57-74-9
Last Revised -- 07/01/89

__VI.A. ORAL RfD REFERENCES

ICF-Clement. 1987. MRID No. 40433701. Available from EPA. Write to FOI, EPA, Washington DC 20460.

Velsicol Chemical Co. 1983a. MRID No. 00138591, 00144313. Available from EPA. Write to FOI, EPA, Washington DC 20460.

Velsicol Chemical Co. 1983b. MRID No. 00144312. Available from EPA. Write to FOI, EPA, Washington DC 20460.

__VI.B. INHALATION RfD REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Ahmed, F.E., R.W. Hart and N.J. Lewis. 1977. Pesticide induced DNA damage and its repair in cultured human cells. Mutat. Res. 42: 161-174.

Ambrose, A.M., H.E. Christensen, D.J. Robbins and L.J. Rather. 1953a. Toxicological and pharmacological studies on chlordane. Arch. Ind. Hyg. Occup. Med. 7: 197-210.

Ambrose, A.M., H.E. Christensen and D.J. Robbins. 1953b. Pharmacological observations on chlordane. Fed. Proceed. 12: 298. (Abstract #982)

Becker, F.F. and S. Sell. 1979. Fetoprotein levels and hepatic alterations during chemical carcinogenesis in C57BL/6N mice. Cancer Res. 39: 3491-3494.

Chambers, D. and S.K. Dutta. 1976. Mutagenic tests of chlordane on different microbial tester strains. Genetics. 83: s13. (Abstract)

Ditraglia, D., D.P. Brown, T. Namekata and N. Iverson. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. Scand. J. Work Environ. Health. 7(4): 140-146.

Gentile, J.M., G.J. Gentile, J. Bultman, R. Sechriest, E.D. Wagner and M.J. Plewa. 1982. An evaluation of the genotoxic properties of insecticides

following plant and animal activation. Mutat. Res. 101: 19-29.

Infante, P.F., S.S. Epstein and W.A. Newton. 1978. Blooddyscrasis and childhood tumors and exposure to chlordane and heptachlor. Scand. J. Work Environ. Health. 4: 137-150.

Ingle, L. 1952. Chronic oral toxicity of chlordan to rats. Arch. Ind. Hyg. Occup. Med. 6: 357-367.

Maslansky, C.J. and G.M. Williams. 1981. Evidence for an epigenetic mode of action in organochlorine pesticide hepatocarcinogenicity: A lack of genotoxicity in rat, mouse, and hamster hepatocytes. J. Toxicol. Environ. Health. 8: 121-130.

NCI (National Cancer Institute). 1977. Bioassay of Chlordane for possible Carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 8. U.S. DHEW Publ. No. (NIH) 77-808. Bethesda, MD.

Probst, G.S., R.E. McMahon, L.E. Hill, C.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. Environ. Mutagen. 3: 11-31.

Sobti, R.C., A. Krishan and J. Davies. 1983. Cytokinetic and cytogenetic effect of agricultural chemicals on human lymphoid cells in vitro. Arch. Toxicol. 52: 221-231.

U.S. EPA. 1985. Hearing Files on Chlordane, Heptachlor Suspension (unpublished draft). Available for inspection at U.S. EPA, Washington, DC.

U.S. EPA. 1986. Carcinogenicity Assessment of Chlordane and Heptachlor/ Heptachlor Epoxide. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC.

Velsicol Chemical Corporation. 1973. MRID No. 00067568. Available from EPA. Write to FOI, EPA, Washington, DC. 20460.

Velsicol Chemical Corporation. 1983a. MRID No. 00144312, 00132566. Available from EPA. Write to FOI, EPA, Washington, DC. 20460.

Velsicol Chemical Corporation. 1983b. MRID No. 00138591. Available from EPA. Write to FOI, EPA, Washington, DC. 20460.

Vigfusson, N.V., E.R. Vyse, C.A. Pernsteiner and R.J. Dawson. 1983. In vivo induction of sister-chromatid exchange in Umbra limi by the insecticides endrin, chlordane, diazinon and guthion. Mutat. Res. 118: 61-68.

Wang, H.H. and B. MacMahon. 1979a. Mortality of workers employed in the manufacture of chlordane and heptachlor. J. Occup. Med. 21(11): 745-748.

Wang, H.H. and B. MacMahon. 1979b. Mortality of pesticide applicators. J. Occup. Med. 21(11): 741-744.

Wildeman, A.G. and R.N. Nazar. 1982. Significance of plant metabolism in the mutagenicity and toxicity of pesticides. Can. J. Genet. Cytol. 24: 437-449.

==========

_VII. REVISION HISTORY

Substance Name -- Chlordane
CASRN -- 57-74-9

Date	Section	Description
09/30/87	II.	Carcinogenicity section added
03/01/88	I.A.1.	Dose conversion clarified
03/01/88	I.A.2.	Text clarified in paragraph 3
03/01/88	II.A.1.	Basis for classification clarified
03/01/88	III.A.	Health Advisory added
04/01/89	I.A.	Withdrawn; new RfD verified (in preparation)
06/01/89	I.A.	Revised oral RfD summary added
06/01/89	VI.	Bibliography on-line
07/01/89	I.A.2.	Reference clarified in paragraph 2
07/01/89	II.	Velsicol (1983) references clarified
07/01/89	VI.C	Carcinogen references added
03/01/90	I.B.	Inhalation RfD now under review
08/01/90	III.A.5.	DWEL changed reflecting change in RfD
08/01/90	III.A.10	Primary contact changed
08/01/90	IV.F.1.	EPA contact changed
01/01/91	II.	Text edited
01/01/91	II.C.1.	Inhalation slope factor removed (global change)
01/01/92	IV.	Regulatory actions updated
07/01/93	II.D.3.	Secondary contact's phone number changed

==========

SYNONYMS

Substance Name -- Chlordane
CASRN -- 57-74-9
Last Revised -- 03/31/87

57-74-9
Belt
CD 68
Chlordane
Chlorindan
Chlor Kil
Corodan
Dowchlor
ENT 9,932
HCS 3260
Kypchlor
M 140
M 410
4,7-Methanoindan, 1,2,4,5,6,7,8,8-Octachloro-3a,4,7,7a-Tetrahydro-
4,7-Methano-1H-Indene, 1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-Hexahydro-
NCI-C00099
Niran
Octachlorodihydrodicyclopentadiene
1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-Hexahydro-4,7-Methano-indene
1,2,4,5,6,7,8,8-Octachloro-3a,4,7,7a-Hexahydro-4,7-Methylene Indane
Octachloro-4,7-Methanohydroindane
Octachloro-4,7-Methanotetrahydroindane
Octa-Klor
Oktaterr
Ortho-Klor
Synklor
TAT Chlor 4
Topiclor
Toxichlor

Velsicol 1068

0160
Heptachlor epoxide; CASRN 1024-57-3 (03/01/97)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Heptachlor epoxide

File On-Line 03/31/87

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	03/01/91
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	07/01/93

==========

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Heptachlor epoxide
CASRN -- 1024-57-3
Last Revised -- 03/01/91

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
Increased liver-to-body weight ratio in both males and females	NOEL: none LEL: 0.5 ppm (diet) (0.0125 mg/kg/day)	1000	1	1.3E-5 mg/kg/day

60-Week Dog Feeding

Study

Dow Chemical Co.,
1958

---

*Conversion Factors: 1 ppm = 0.025 mg/kg/day (assumed dog food consumption)

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Dow Chemical Company. 1958. MRID No. 00061912. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Beagle dogs from 23 to 27 weeks of age were divided into five groups (3 females and 2 males) and given diets containing 0, 0.5, 2.5, 5 or 7.5 ppm of heptachlor epoxide for 60 weeks. Liver-to-body weight ratios were significantly increased in a treatment-related fashion. Effects were noted for both males and females at the LEL of 0.5 ppm. A NOEL was not established.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- Based on a chronic exposure study, an uncertainty factor of 1000 was used to account for inter- and intraspecies differences and to account for the fact that a NOEL was not attained.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

None.

Data Considered for Establishing the RfD:

1) 60-Week Feeding - dog: Principal study - see previous description; no core grade

2) 2-Generation Reproduction - dog: NOEL=1 ppm (0.025 mg/kg/day); LEL=3 ppm (0.075 mg/kg/day) (liver lesions in pups); Reproductive NOEL=5 ppm (0.125 mg/kg/day); Reproductive LEL=7 ppm (0.175 mg/kg/day) (pup survival); no core grade (Velsicol Chemical, 1973a)

3) 3-Generation Reproduction - rat: NOEL=5 ppm (0.25 mg/kg/day); LEL=10 ppm (0.5 mg/kg/day) (pup mortality); no core grade (Velsicol Chemical, 1959a)

4) 2-Year Feeding - rat: LEL=0.5 ppm (0.025 mg/kg/day) (LDT) (females - vacuolar changes in central hepatic lobule); NOEL not established; no core grade (Velsicol Chemical, 1959b)

Other Data Reviewed:

1) Chronic Feeding Study - mouse: Heptachlor/Heptachlor Epoxide (1:3): NOEL=none; LEL=1 ppm (LDT) (vaculoation, enlarged nucleus, hepatocytomegaly); no core grade (Velsicol Chemical, 1973b)

2) Chronic Feeding Study - rat: Heptachlor/Heptachlor Epoxide (3:1): NOEL=none; LEL=5 ppm (LDT) (liver-to-body weight increase in females); no core grade (Velsicol Chemical, 1966)

3) 3-Generation Reproduction - rat: Heptachlor/Heptachlor Epoxide (3:1): NOEL=7 ppm (HDT); LEL=none; no core grade (Velsicol Chemical, 1967)

Data Gap(s): Rat Teratology Study; Rabbit Teratology

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Low
Data Base -- Medium
RfD -- Low

The principal study is of low quality and is given a low confidence rating. Since the data base on chronic toxicity is complete but consists of low-quality studies, the data base is given a medium to low confidence rating. Low confidence in the RfD follows.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Pesticide Registration Standard, August 1986

Agency Work Group Review -- 12/18/85, 09/16/86

Verification Date -- 09/16/86

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- Heptachlor epoxide
CASRN -- 1024-57-3

Not available at this time.

==========

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Heptachlor epoxide
CASRN -- 1024-57-3
Last Revised -- 07/01/93

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative

estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Sufficient evidence exists from rodent studies in which liver carcinomas were induced in two strains of mice of both sexes and in CFN female rats. Several structurally related compounds are liver carcinogens.

___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. There are no published epidemiologic evaluations of heptachlor epoxide. It is not commercially available in the United States, but is a product of heptachlor oxidation.

There were 11 case reports involving central nervous system effects, blood dyscrasias and neuroblastomas in children with pre-/postnatal exposure to chlordane and heptachlor (Infante et al., 1978). Since no other information was available, no conclusions can be drawn.

There were three epidemiologic studies of workers exposed to chlordane and/or heptachlor. One retrospective cohort study of pesticide applicators was considered inadequate in sample size and duration of follow-up. This study showed marginal statistically significant increased mortality from bladder cancer (3 observed) (Wang and McMahon, 1979a). Two other retrospective cohort studies were of pesticide manufacturing workers. Neither of them showed any statistically significant increased cancer mortality (Wang and McMahon, 1979b; Ditraglia et al., 1981). Both these populations also had confounding exposures from other chemicals.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Four long-term carcinogenesis bioassays of heptachlor epoxide have been reported. The major finding in mice has been an increased incidence of liver carcinomas. Davis (1965) fed groups of 100 male and 100 female C3H mice 0 or 10 ppm heptachlor epoxide for 2 years. Survival was generally low, with 50% of controls and 9.5% of treated mice living 2 years. A 2-fold increase in benign liver lesions (hepatic hyperplasia and benign tumors) over the controls was reported. Reevaluation by Reuber (1977b) revealed a significant increase in liver carcinomas in the dosed group (77/81 in females and 73/79 in males) over the controls (2/53 in females and 22/73 in males). The Velsicol Chemical Co. (1973) tested a 75:25 mixture of heptachlor epoxide:heptachlor in groups of 100 male and 100 female CD-1 mice. The mice were fed 0, 1, 5, and 10 ppm for 18 months. A statistically significant increase of hyperplasia was observed in the 5, and 10 ppm dose groups in both sexes; Reuber's reevaluation (U.S. EPA, 1985) resulted in a change in

diagnosis for benign to liver carcinomas, thereby increasing the incidence of hepatic carcinomas ($p<0.01$). Four independent pathologists concurred with Reuber's reevaluation.

The earliest bioassay with rats (Witherup et al., 1959) tested 25 male and 25 female CFN rats each at 0.5, 2.5, 5.0, 7.5, and 10 ppm for 108 weeks. The authors observed malignant and benign tumors randomly among test groups and controls. Reuber's reevaluation (1985) reported a significant increase of hepatic carcinomas above the controls at 5 and 10 ppm in the female rats. A reevaluation by Williams (1985) reported a significant increase of hepatic nodules at the 10 ppm level in the males over the controls. The Kettering Laboratory (Jolley et al., 1966) tested a mixture of 75:25 heptachlor:heptachlor epoxide in the diet of 25 female CD rats at 5, 7.5, 10, and 12.5 ppm for 2 years. Although no malignant lesions of the liver were observed, hepatocytomegaly was increased at 7.5, 10, and 12.5 ppm.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Gene mutation assays indicate that heptachlor epoxide is not mutagenic in bacteria (Moriya et al., 1983). In two mouse dominant lethal assays, heptachlor epoxide did not induce major chromosomal aberrations in male germinal cells (Arnold et al., 1977; Epstein et al., 1972). Ahmed et al. (1977) reported qualitative evidence of uuncheduled DNA synthesis response in SV40 transformed human fibroblasts in the presence of hepatic homogenates and heptachlor epoxide.

Five compounds structurally related to heptachlor epoxide (chlordane, aldrin, dieldrin, heptachlor and chlorendic acid) have produced liver tumors in mice. Chlorendic acid has also produced liver tumors in rats.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 9.1E+0 per (mg/kg)/day

Drinking Water Unit Risk -- 2.6E-4 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-1 ug/L
E-5 (1 in 100,000)	4E-2 ug/L
E-6 (1 in 1,000,000)	4E-3 ug/L

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- hepatocellular carcinomas
Test Animals -- mouse/C3H (Davis); mouse/CD1 (Velsicol)
Route -- diet
Reference -- Davis, 1965; Velsicol, 1973 (see table)

Administered Human Equivalent Tumor

Dose (ppm)	Dose (mg/kg/day)	Incidence	Reference
male			
0	0.0	22/73	Davis, 1965
10	0.108	73/79	as diagnosed
female			by Reuber, 1977
0	0.000	2/53	(cited in
10	0.108	77/81	Epstein, 1976)
female			
0	0.00	6/76	Velsicol, 1973
1	0.01	1/70	as evaluated
5	0.052	6/65	by Reuber, 1977
10	0.10	30/57	
male			
0	0.00	0/62	
1	0.01	2/68	
5	0.052	18/68	
10	0.10	52/80	

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

The Davis (1965) study was designed to be for lifetime exposure. Thus, although survival was low, no correction for duration of experiment was made. Five data sets (four in mice and one in rats) show an increased incidence of hepatocellular carcinomas in treated groups compared with controls. There are four slope factors, 27.7 per (mg/kg)/day for C3H male mice, 36.2 per (mg/kg)/day for C3H female mice, 1.04 per (mg/kg)/day for CD-1 female mice, and 6.48 per (mg/kg)/day for CD-1 male mice. Since mice were the more sensitive species tested and to avoid discarding relevant data, the quantitative estimate is based on the geometric mean of 9.1 per (mg/kg)/day. This geometric mean is consistent with the potency estimate from rats of 5.8 per (mg/kg)/day (CFN females).

The above unit risk should not be used if the water concentration exceeds 40 ug/L, since above this concentration the unit risk may not be appropriate.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Adequate numbers of animals were treated in both studies, but survival in the Davis (1985) study was low. A dose-related increase in tumor incidence was observed in CD-1 mice. Slope factors were consistent in two species of rodents.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 2.6E-3 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration

E-4 (1 in 10,000)	4E-2 ug/cu.m
E-5 (1 in 100,000)	4E-3 ug/cu.m
E-6 (1 in 1,000,000)	4E-4 ug/cu.m

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

The inhalation risk estimates were calculated from the oral data presented in II.B.2.

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The above unit risk should not be used if the air concentration exceeds 4 ug/cu.m, since above this concentration the unit risk may not be appropriate.

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

See II.B.4.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1985, 1986

The values in the 1986 Carcinogenicity Assessment for Chlordane and Heptachlor/Heptachlor Epoxide have been reviewed by the Carcinogen Assessment Group.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 04/01/87

Verification Date -- 04/01/87

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

==========

_VI. BIBLIOGRAPHY

Substance Name -- Heptachlor epoxide
CASRN -- 1024-57-3
Last Revised -- 03/01/91

__VI.A. ORAL RfD REFERENCES

Dow Chemical Company. 1958. MRID No. 00061912. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1959a. MRID No. 00062676. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1959b. MRID No. 00061911. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1966. MRID No. 00086208. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1967. MRID No. 00147057. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1973a. MRID No. 00050058. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1973b. MRID No. 000523262, 00062678, 00064943. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Davis, K.J. 1965. Pathology Report on Mice Fed Aldrin, Dieldrin, Heptachlor and Heptachlor Epoxide for Two Years. Internal FDA memorandum to Dr. A.J. Lehman, July 19.

Epstein, S.S. 1976. Carcinogenicity of heptachlor and chlordane. Sci. Total Environ. 6: 103-154.

Reuber, M.D. 1977. Histopathology of carcinomas of the liver in mice ingesting heptachlor or heptachlor epoxide. Exp. Cell Biol. 45: 147-157.

U.S. EPA. 1985. Hearing Files on Chlordane, Heptachlor Suspension (unpublished draft). Available for inspection at: U.S. EPA, Washington, DC.

U.S. EPA. 1986. Carcinogenicity Assessment of Chlordane and Heptachlor/Heptachlor Epoxide. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC. OHEA-C-204.

Velsicol Chemical Corporation. 1973. MRID No. 00062678. Available from EPA. Write to FOI, EPA, Washington, D.C. 20460.

==========

_VII. REVISION HISTORY

Substance Name -- Heptachlor epoxide
CASRN -- 1024-57-3

Date	Section	Description
09/30/87	II.	Carcinogen summary on-line
03/01/88	I.A.2.	Text clarified
03/01/88	I.A.5.	Confidence levels revised
03/01/88	II.B.4.	Confidence statement revised
03/01/88	III.A.	Health Advisory on-line
08/01/90	III.A.10	Primary contact changed
08/01/90	IV.F.1.	EPA contact changed
01/01/91	II.	Text edited
01/01/91	II.C.1.	Inhalation slope factor removed (global change)
03/01/91	I.A.4.	Citations added
03/01/91	VI.	Bibliography on-line
01/01/92	IV.	Regulatory actions updated
04/01/92	II.A.3.	Text revised
04/01/93	IV.C.2.	Freshwater and marine values corrected
07/01/93	II.D.3.	Secondary contact's phone number changed

==========

SYNONYMS

Substance Name -- Heptachlor epoxide
CASRN -- 1024-57-3
Last Revised -- 03/31/87

1024-57-3
ENT 25,584
EPOXYHEPTACHLOR
HCE
Heptachlor Epoxide
1,4,5,6,7,8,8-HEPTACHLORO-2,3-EPOXY-2,3,3a,4,7,7a-HEXAHYDRO-4,7-METHANOINDENE
1,4,5,6,7,8,8-HEPTACHLORO-2,3-EPOXY-3a,4,7,7a-TETRAHYDRO-4,7-METHANOINDAN
2,3,4,5,6,7,7-HEPTACHLORO-1a,1b,5,5a,6,6a-HEXAHYDRO-2,5-METHANO-2H-INDENO(1,2-b)OXIRENE
HIPTACHLOR EPOXIDE
4,7-METHANOINDAN, 1,4,5,6,7,8,8-HEPTACHLORO-2,3-EPOXY-3a,4,7,7a-TETRAHYDRO-2,5-METHANO-2H-OXIRENO(a)INDENE, 2,3,4,5,6,7,7-HEPTACHLORO-1a,1b,5,5a,6,6a-HEXAHYDRO-
VELSICOL 53-CS-17

Ecological Toxicological Profile for Chlordane (alpha, gamma)

Organism	Dose	Exposure Route	Exposure Period	Effect	Endpoint	Reference
Chlordane						
Earthworm (*Lumbricus terrestris)*		Artificial soil	16 days	Spermatozoa count reduction	25 ppm	4
Red-winged blackbird	10, 50, and 100 ppm	Oral - diet	84 days	Mortality	NOAEL = 2.14 mg/kg/day	3
Largemouth bass *(Micropterus salmoides)*		Medium	96 hours	Death	LC_{50} = 3.0 µg/l	1
Fathead minnow *(Pimephales promelas)*		Medium	96 hours	Death	LC_{50} = 25-115 µg/l	1
Sheepshead minnow *(Cyprinodon variegatus)*	0.8 µg/l	Medium	96 hours	Reduced hatch during continuous exposure		1
Mallard *(Anas platyrhyncos)*		Oral-diet		Death	LD_{50} = 709 mg/kg	1
Mouse		Gavage-oil	7 days, 1 time per day	Death	LOAEL = 300 mg/kg/day	2
Mouse		Oral-food	GD 1-19	Death of 55% offspring	LOAEL = 8.0 mg/kg/day	2
Rat		Oral-food	1 generation	Decreased fertility and survivability	LOAEL = 16 mg/kg/day	2
Freshwater aquatic life		Medium	Chronic	Protection of aquatic life	AWQC = 0.0043 µg/L	5
Mouse	25, 50, and 100 mg/kg	Oral - diet	6 generations	Reduced number of offspring	NOAEL = 4.58 mg/kg/day	3

Ecological Toxicological Profile for Chlordane (alpha, gamma) (Continued)

Chlordane is an organochlorine compound first introduced into the United States in 1947 in a variety of formulations for use as a broad-spectrum pesticide. Since 1983, chlordane use in the United States has been prohibited, except for control of underground termites. Technical chlordane consists of about 45 compounds, primarily cis-chlordane (19%), trans-chlordane (24%), heptachlor (10%), cis- and trans-nonachlor (7%), and various chlordane isomers (22%). Chlordane is readily absorbed by warm-blooded animals through skin, diet, and inhalation, and is distributed throughout the body. Food chain biomagnification is usually low, except in some marine mammals. In most mammals, the metabolite oxychlordane has proven much more toxic and persistent than the parent chemical. Many species of aquatic organisms are adversely affected by concentrations in water between 0.2 and 3.0 µg/L technical chlordane. Sensitive bird species have exhibited reduced survival on diets containing 1/5 mg chlordane per kilogram body weight in their diet, or after a single dose as low as 14.1 mg chlordane per kilogram body weight (1). Chlordane will bioconcentrate in both marine and freshwater organisms. The biomagnification from fish to seal was 7.3/4.7 and that between seal and bear was 6.6/9.5, resulting in an overall fish to bear biomagnification factor of 44.2. Chlordane is taken up b rooted aquatic vascular plants both from the water and from the sediment. Chlordane also bioconcentrates in the roots from contaminated sediment and translocates into the shoots.

Bioconcentration:

- Marine organism BCF = 3,000-12,000
- Rainbow trout BCF = 18,500
- Submerged vascular plant *(Hydrilla verticillata)* BCF = 1.06
- Earthworm BAF = 10.5366
- Plant Uptake Factor = 0.017 (7)

Environmental Fate:

Log K_{ow} = 5.54 (2)

Log K_{oc} = 3.49 - 4.64 (2)

Henry's Law Constant (25° C) = 4.85×10^{-5} atm - m^3/mol

References:

1. Eisler, R. 1990. Chlordane hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildlife Service, Contaminant Hazard Reviews Biological Report. 85(1.21).
2. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry (ATSDR). 1994. *Toxicological Profile for Chlordane.* U.S. Government Printing Office, Washington, D.C.
3. Oak Ridge National Laboratory, (ORNL) Toxicological Benchmarks for Wildlife: 1996 Revision. ES/ER/TM-86/R3. June 1996.
4. Citutovic, M.A., Fitzpatrick, L.C., Venables, B.J., and A.J. Goven. "Sperm Count in Earthworms (*Lumbricus Terrestris*) as a Biomarker for Environmental Toxicology: Effects of Cadmium and Chlordane." *Environmental Pollution* 81(1993)123-125.
5. Federal Ambient Water Quality Criteria.
6. Connell, D.W. and R.D. Markwell. "Bioaccumulation in the Soil to Earthworm system." *Chemosphere* Vol. 20, Nos 1-2, pp 91-100. 1990.
7. Sims, Ronald C., Judith L. Sims, and Steven G. Hansen. Soil Transport and Fate Database Ver. 2.0. Department of Civil and Environmental Engineering, Utah State University. April 1991.

Ecological Toxicological Profile for DDT, DDD, and DDE

Organism	Dose	Exposure Route	Exposure Period	Effect	Endpoint	Reference
4,4-DDT						
Mouse		Oral	70 wks	Decreased survival	LOAEL=13mg/kg/d	6
Rabbit		Oral		Death	LD_{50}=300mg/kg	7
Dog		Oral	5 days/wk for 14 months	Maternal and fetal death	LOAEL=12 mg/kg	6
Japanese quail	500 ppm 700 ppm	Diet		No eggs hatched No eggs laid		7
Rat		Gavage	One time	Death	LD_{50} = 113 mg/kg	1
Mouse		Oil-gavage	One time	Death	LD_{50} = 1,466 mg/kg	1
Mouse		Oil-gavage	One time	Death	LD_{50} = 810 mg/kg	1
Mouse		Oral-food	3-12 weeks	Decreased IgM antibody titer	LOAEL = 13 mg/kg/day	1
Rat		Oral-food	60 days	Decreased fertility	LOAEL = 0.35 mg/kg/day	1
Rat		Oral-food	36 weeks	Sterility	LOAEL = 7.5 mg/kg/day	1
Mouse		Oral-food	Life	Increase in preweaning death	LOAEL = 32.5 mg/kg/day	1
Mouse		Oral-food	70 weeks	Decreased survival	LOAEL = 13 mg/kg/day	1
Freshwater aquatic organisms		Medium	Chronic	Protection of aquatic life	AWQC = 0.001 µg/L	11
Mallard					LD_{50} = 2240 mg/kg	12
California quail					LD_{50} = 595 mg/kg	12
Rat	1, 10, 25 mg/kg/d	Oral	7 months	Pup death by 10 days	LOAEL = 25mg/kg/d	1

Organism	Dose	Exposure Route	Exposure Period	Effect	Endpoint	Reference
4,4-DDT						
	of DDT				NOAEL = 1 mg/kg/d	
Earthworm	7 ppm	Oral-soil	11 years	Conc. in worm hazardous to bird health	Dose = 7 ppm	18
Japanese quail					LD_{50} = 841 mg/kg	12
Pheasant					LD_{50} = 1334	12
Sandhill crane					LD_{50} > 1200	12
Rock dove					LD_{50} > 4000	12
Bullfrog					LD_{50} > 2000 mg/kg	12
Sheep	250 ppm	Oral	10-16 weeks	Increased hepatic microsomal enzyme activity		2
Rat	10, 50, 100 and 600 ppm	Oral-diet	2 years	Fertility reduced	NOAEL = 0.8 mg/kg/day	13
Brown pelican		oral-diet	5 years	Reduced fledgling rate	NOAEL = 0.0028 mg/kg/day	13

Ecological Toxicological Profile for DDT, DDD, and DDE (Continued)

Organism	Dose	Exposure Route	Exposure Period	Effect	Endpoint	Reference
4,4-DDE						
Rat		Gavage	One time	Death	LD_{50} = 880 mg/kg	1
Mouse		Oil-gavage	One time	Death	LD_{50} = 810 mg/kg	1
Mouse		Food	78 weeks	Liver carcinomas	LOAEL = 19 mg/kg/day	1
Rat		Oral	One time	Death	LD_{50} = 880mg/kg	8
Hamster		Gavage	128 weeks	Adrenal tumors	LOAEL = 41.5 mg/kg/day	1
Lesser kestrel		Environmental		Reproductive	NOAEL in eggs = 5ppm	9
American sparrow hawk		Diet	Eggs laid in the 2nd year of experiment	10% decrease in shell thickness	2-8 ppm in eggs	7
Ringed turtle dove	20-30 mg total	Diet	3 wks, starting 6 wks before pairing	Decrease in various reproductive endpoints such as reproductive performance.		10
Bald eagle	> 5 mg/kg in egg	trophic	Lifetime	Reduced reproduction	> 5 mg/kg in egg	4
Cooper's hawk	> 5 mg/kg	trophic	Lifetime	Egg breakage	> 5 mg/kg	4
Bald eagle		trophic	Lifetime	Successful reproduction	NOEL = 2 mg/kg in egg	5
Bald eagle		trophic	Lifetime	15% eggshell thinning	16 µg/g in egg	6
Rat		Oral	9 weeks		NOAEL = 10 mg/kg/d	1
Earthworm	7 ppm	Oral-soil	11 years	Conc. in worm hazardous to sensitive bird species	Dose = 7 ppm	18

Ecological Toxicological Profile for DDT, DDD, and DDE (Continued)

Organism	Dose	Exposure Route	Exposure Period	Effect	Endpoint	Reference
4,4'-DDD						
Rat		Oral	One time	Death	LD_{50} = 400 mg/kg	1
Rat		Oral		Death	LD_{50}=113mg/kg	8
Mouse		Oil-gavage	One time	Death	LD_{50} = 1466 mg/kg	1
Rabbit		Oral	One time	Death	LD_{50}=1200mg/kg	
Dog		Capsule	36-150 days	Adrenal necrosis	LOAEL = 50 mg/kg/day	1
Rat		Oral-diet	78 weeks	Thyroid adenomas, carcinomas	LOAEL = 85 mg/kg/day	1
Bobwhite quail		Oral-diet	5 days	Death	LC_{50} = 2178 ppm Adjusted NOAEL = 4.75 mg/kg/day	15
Ring-necked pheasant		Oral-diet	5 days	Death	LC_{50} = 445 ppm	15
Daphnia magna		Media	96 hours	Death	LC_{50} = 3.2 µg/L	16
Mouse	53-107 mg/kg/d	Oral	130 weeks	Lung, liver tumors	LOAEL = 32.5 mg/kg/d	1

DDT was used extensively as a pesticide and for vector control until its ban in the U.S. in 1972. DDT and its primary metabolites, DDE and DDD, are man-made chemicals and are not none to occur naturally in the environment. The p,p'-isomer of DDT is the most prevalent in the environment (85%) with the o,p'-isomer accounting for the remaining 15%. DDT, DDE and DDD bind to soil and sediment as predicted by their organic carbon partition coefficients (K_{oc}) of 2.4×10^5, 4.4×10^6 and 7.7×10^5 for DDT, DDE and DDD respectively. DDT, DDE and DDD are highly lipid soluble with log octanol-water partition coefficients (log K_{ow}) of 6.19, 7.00, and 6.20 respectively. This lipophilic property, combined with an extremely long half-life, has resulted in bioaccumulation. Biomagnification of residues may result in high levels of residues in organisms at the top of the food chain as has been documented with fish-eating birds (3). The steady-state bioconcentration factor in rainbow trout is estimated to be 12,000. Because of the extensive past use of DDT worldwide and the persistence of DDT and its metabolites, these materials are virtually ubiquitous and are continually being transformed and redistributed in the environment (1).

DDT produces embryotoxicity and fetotoxicity but not teratogenicity in animals. Intermediate oral exposure to DDT in animals has been shown to produce developmental effects such as infertility, mortality, and slow development. The reproductive capability of both males and females is adversely affected by DDT. DDT enhances the metabolism of estrogen. This creates an endocrine imbalance that affects the egg-laying and nesting cycle in birds in such a way that total reproductive success and survival of young during the nesting season may be reduced (3). DDE is far more persistent then DDT or DDD in birds, having a half-life in excess of 200 days. DDE has been significantly correlated with reduced eggshell thickness and brood sizes of ospreys, bald eagles and some species of falcons (4). Rat study with simultaneous exposure of DDT (7ppm), aldrin (5ppm), endrin (5ppm) or heptachlor (5ppm) increased the adverse effects on conception rate and pup survival, but effects appeared less than additive. Carcinogenicity has been shown in some laboratory animals (2).

Ecological Toxicological Profile for DDT, DDD, and DDE (Continued)

Bioconcentration:
- Plant Uptake Factor for 4,4'-DDD = 0.0025 (17)
- Plant Uptake Factor for 4,4'-DDE = 0.00087 (17)
- Plant Uptake Factor for 4,4'-DDT = 0.0026 (17)

References:

1. Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for DDT, DDE, and DDD*. 1994.
2. NIOSH Special Occupational Hazard Review for DDT. U.S. Department of Health, Education and Welfare, 1978.
3. Klassen, C.D., Amdur, M.O., and J. Doull (Eds.) *Casarett and Doull's Toxicology, Third Edition*. Macmillian Publishing, 1986.
4. Wiemeyer, S.N., "Effects of Environmental Contaminants on Raptors in the Midwest. In: Proc. Raptor Management Symposium and Workshop." Natl. Wildl. Fed., Washington, D.C., pages 168-181, 1991.
5. Heinz, G.H. and S.N. Wiemeyer. "Effects of contaminants on birds." In: Funderburk, S.L., J.A. Mihursky, S.J. Jordan and D. Riley (Eds.). *Habitat Requirements for Chesapeake Bay Living Resources*, 2nd Edition. Living Resources Subcommittee, Chesapeake Bay Program. Annapolis, MD. 1991.
6. Wiemeyer, S.N., C.M. Bunck, and C.J. Stafford. "Environmental Contaminants in Bald Eagle Eggs-1980-84-and Further Interpretations of Relationships to Productivity and Shell Thickness." *Arch. Environ. Contam. Toxicol.* Vol 24, 213-227, 1993.
7. Hazardous Substances Data Bank. (HSDB) on-line computer search, 1994.
8. Sax, I. and Lewis, R. J. Sr. *Dangerous Properties of Industrial Materials*. 7th ed., Vol. II. Von Nostrand Reinhold,: New York, 1989.
9. Negro, J. J., Donazar, J. A., Hernandez, L. M., Hernandez, M. A., and Hiraldo, F. "Organochlorine and Heavy Metal Contamination in Non-Viable Eggs and Its Relation to Breeding Success in a Spanish Population of Lesser Kestrels (*Falcan nanmanni*)." *Environmental Pollution*. 82, 201-205 (1993).
10. Keith, J. O. and Mitchell, C. A. "Effects of DDE and Food Stress on Reproduction and Body Condition of Ringed Turtle Doves." *Arch. Environ. Contam. Toxicol.* 25, 192-203 (1993).
11. U.S. Environmental Protection Agency (USEPA). "Water Quality Criteria Summary." *Federal Register* Notice 57FR60914. Office of Sciences and Technology, Health and Ecological Criteria Division: Washington, D.C. 1991.
12. Hudson, R.H., Tucker, R.K., and M.A. Haegele. *Handbook of Toxicity of Pesticides to Wildlife*. 2nd Edition. U.S. Department of Interior. Fish and Wildlife Service. Resource Publication 153. Washington, D.C., 1984.
13. Oak Ridge National Laboratory (ORNL). *Toxicological Benchmarks for Wildlife: 1996 Revision*. ES/ER/TM-86/R3. June 1996.
14. Beyer, W.N. "Evaluating Soil Contamination." *U.S. Fish Wildl. Serv., Biol. Rep.*, 90 (2), 25 pp. 1990.
15. Hazardous Substance Data Bank (HSDB) on-line computersearch, 1996.
16. Hazardous Substance Data Bank (HSDB) on-line computer search, 1996.
17. Sims, Ronald C., Judith L. Sims, and Steven G. Hansen. *Soil Transport and Fate Database Ver. 2.0*. Department of Civil and Environmental Engineering, Utah State University. April 1991.
18. Beyer, Nelson and Charles D. Gish. "Persistence in Earthworms and Potential Hazards to Birds of Soil Applied DDT, Dieldrin, and Heptachlor." *Journal of Applied Ecology*. 17, 295-307. 1980.

This page intentionally left blank.

Appendix B

RISK ASSESSMENT SPREADSHEETS

This page intentionally left blank.

Quantification of Dermal Exposure to Sediment While Swimming
Lake Danielson and Golf Course Pond
Defense Depot Memphis Tennessee

Contaminant	Maximum Concentration (mg/kg)	Surface Area (sq.cm)	Adherence Factor (mg/sq.cm)	Absorption Factor (unitless)	Exposure Frequency (events/yr)	Exposure Duration (years)	Body Weight (kg)	Averaging Time (days)	Conversion Factor (kg/mg)	Absorbed Dose (mg/kg/d)	Cancer Slope Factor (/mg/kg/d)	Cancer Risk (unitless)
DDD	3	2050	2.77	0.1	60	6	57.7	25550	0.000001	4.16E-07	0.24	9.98E-08
DDE	0.068	2050	2.77	0.1	60	6	57.7	25550	0.000001	9.43E-09	0.34	3.21E-09
DDT	2.9	2050	2.77	0.1	60	6	57.7	25550	0.000001	4.02E-07	0.34	1.37E-07

Quantification of Sediment Ingestion (with surface water) While Swimming
Lake Danielson and Golf Course Pond
Defense Depot Memphis Tennessee

Contaminant	Maximum Concentration (mg/L)*	Ingestion Rate (liters/hour)	Exposure Time (hours/event)	Exposure Frequency (events/yr)	Exposure Duration (years)	Body Weight (kg)	Averaging Time (days)	Intake (mg/kg/d)	Cancer Slope Factor (/mg/kg/d)	Cancer Risk (unitless)
DDD	0.00003	0.05	1	60	6	57.7	25550	3.66E-10	0.24	8.79E-11
DDE	0.0000068	0.05	1	60	6	57.7	25550	8.3E-11	0.34	2.82E-11
DDT	0.000029	0.05	1	60	6	57.7	25550	3.54E-10	0.34	1.2E-10

* assumes 0.001% sediment suspended in water

Quantification of Pesticide Exposure via Fish Ingestion
Lake Danielson and Golf Course Pond
Defense Depot Memphis Tennessee

Contaminant	Maximum Concentration (mg/kg)	Ingestion Rate (kg/day)	Exposure Frequency (days/year)	Fraction Ingested from ponds	Exposure Duration (years)	Body Weight (kg)	Averaging Time (days)	Intake (mg/kg/d)	Cancer Slope Factor (/mg/kg/d)	Cancer Risk (unitless)
DDD	5.09	0.0059	365	1	6	57.7	25550	4.46E-05	0.24	1.07E-05
DDE	5.31	0.0059	365	1	6	57.7	25550	4.65E-05	0.34	1.58E-05
DDT	1.05	0.0059	365	1	6	57.7	25550	9.2E-06	0.34	3.13E-06

Quantification of Dermal Exposure to Sediment While Swimming
Lake Danielson and Golf Course Pond
Defense Depot Memphis Tennessee

Contaminant	Maximum Concentration (mg/kg)	Surface Area (sq.cm)	Adherence Factor (mg/sq.cm)	Absorption Factor (unitless)	Exposure Frequency (events/yr)	Exposure Duration (years)	Body Weight (kg)	Averaging Time (days)	Conversion Factor (kg/mg)	Absorbed Dose (mg/kg/d)	Cancer Slope Factor (/mg/kg/d)	Cancer Risk (unitless)
DDD	1	2050	2.77	0.1	60	6	57.7	25550	0.000001	1.38666E-07	0.24	3.32798E-08
DDE	2.1	2050	2.77	0.1	60	6	57.7	25550	0.000001	2.91198E-07	0.34	9.90074E-08
DDT	0.234	2050	2.77	0.1	60	6	57.7	25550	0.000001	3.24478E-08	0.34	1.10323E-08
Chlordane	3.89	2050	2.77	0.1	60	6	57.7	25550	0.000001	5.3941E-07	1.3	7.01233E-07
Heptachlor E	0.115	2050	2.77	0.1	60	6	57.7	25550	0.000001	1.59466E-08	9.1	1.45114E-07
											Total risk:	9.89666E-07

Quantification of Sediment Ingestion (with surface water) While Swimming
Lake Danielson and Golf Course Pond
Defense Depot Memphis Tennessee

Contaminant	Maximum Concentration (mg/L)*	Ingestion Rate (liters/hour)	Exposure Time (hours/event)	Exposure Frequency (events/yr)	Exposure Duration (years)	Body Weight (kg)	Averaging Time (days)	Intake (mg/kg/d)	Cancer Slope Factor (/mg/kg/d)	Cancer Risk (unitless)
DDD	0.00001	0.05	1	60	6	57.7	25550	1.22E-10	0.24	2.93033E-11
DDE	0.0000212	0.05	1	60	6	57.7	25550	2.59E-10	0.34	8.80077E-11
DDT	0.00000234	0.05	1	60	6	57.7	25550	2.86E-11	0.34	9.71406E-12
Chlordane	0.0000389	0.05	1	60	6	57.7	25550	4.75E-10	1.3	6.17446E-10
Heptachlor E	0.00000115	0.05	1	60	6	57.7	25550	1.4E-11	9.1	1.27775E-10
* assumes 0.001% sediment suspended in water									Total risk:	8.72245E-10

Quantification of Pesticide Exposure via Fish Ingestion
Lake Danielson and Golf Course Pond
Defense Depot Memphis Tennessee

Contaminant	Maximum Concentration (mg/kg)	Ingestion Rate (kg/day)	Exposure Frequency (days/year)	Fraction Ingested from ponds	Exposure Duration (years)	Body Weight (kg)	Averaging Time (days)	Intake (mg/kg/d)	Cancer Slope Factor (/mg/kg/d)	Cancer Risk (unitless)
DDD	0.124	0.0065	365	1	6	57.7	25550	1.2E-06	0.24	2.87358E-07
DDE	0.6	0.0065	365	1	6	57.7	25550	5.79E-06	0.34	1.96979E-06
Dieldrin	0.013	0.0065	365	1	6	57.7	25550	1.26E-07		
Chlordane	0.166	0.0065	365	1	6	57.7	25550	1.6E-06	1.3	2.08373E-06
									Total risk:	6.3493E-06

Risk Calculations, 1998 Data

Quantification of Pesticide Intake by Humans via Ingestion of Frog Legs
Lake Danielson and golf course pond
Defense Distribution Depot, Memphis, Tennessee

Chemical	Maximum Conc. (mg/kg)	Ingestion Rate (kg/d)	Fraction Ingested from ponds	Exposure Frequency (days/year)	Exposure Duration (years)	Body Weight (kg)	Averaging Time (days	Contaminant Intake (mg/kg/d)	Cancer Slope Factor (/mg/kg/d)	Cancer Risk (unitless)	Reference Dose (mg/kg/d)	Hazard Quotient (unitless)
DDD	0.00352	0.00065	1	365	6	57.7	25550	3.3989E-09	0.24	8.16E-10		
DDE	0.017	0.00065	1	365	6	57.7	25550	1.6415E-08	0.34	5.58E-09		
DDT	0.0025	0.00065	1	365	6	57.7	25550	2.414E-09	0.34	8.21E-10	0.0005	4.83E-06
Dieldrin	0.0314	0.00065	1	365	6	57.7	25550	3.0319E-08	16	4.85E-07	0.00005	0.000606

Quantification of Pesticide Intake by Belted Kingfishers
Lake Danielson and golf course pond
Defense Distribution Depot, Memphis, Tennessee

Chemical	Maximum Conc. (mg/kg)	Food Ingestion Rate (g/g-d)	Body Weight (g)	Conversion Factor (kg/g)	Body Weight (kg)	Estimated NOAEL (mg/kg-day)	Estimated Intake (mg/kg-day)	Intake Exceeds NOAEL?
Chlordane	0.4	0.5	158	0.001	0.158	1.623	0.2	no
DDT and metabolites	1.0369	0.5	158	0.001	0.158	0.0008	0.51845	yes
Dieldrin	0.0367	0.5	158	0.001	0.158	0.112	0.01835	no

Quantification of Pesticide Intake by Great Blue Herons (fish)
Lake Danielson and golf course pond
Defense Distribution Depot, Memphis, Tennessee

Chemical	Maximum Conc. (mg/kg)	Food Ingestion Rate (g/g-d)	Body Weight (g)	Conversion Factor (kg/g)	Body Weight (kg)	Estimated NOAEL (mg/kg-day)	Estimated Intake (mg/kg-day)	Intake Exceeds NOAEL?
Chlordane	0.56	0.18	2230	0.001	2.23	0.648	0.1008	no
DDT and metabolites	2.369	0.18	2230	0.001	2.23	0.0032	0.42642	yes
Dieldrin	0.167	0.18	2230	0.001	2.23	0.045	0.03006	no

Quantification of Pesticide Intake by Great Blue Herons (frogs)
Lake Danielson and golf course pond
Defense Distribution Depot, Memphis, Tennessee

Chemical	Maximum Conc. (mg/kg)	Food Ingestion Rate (g/g-d)	Body Weight (g)	Conversion Factor (kg/g)	Body Weight (kg)	Estimated NOAEL (mg/kg-day)	Estimated Intake (mg/kg-day)	Intake Exceeds NOAEL?
Chlordane	ND	0.18	2230	0.001	2.23	0.648		NA
DDT and metabolites	0.02302	0.18	2230	0.001	2.23	0.0032	0.0041436	yes
Dieldrin	0.0314	0.18	2230	0.001	2.23	0.045	0.005652	no

Appendix C

AUGUST 14, 1998, LETTER FROM TVA FISHERIES BIOLOGIST

This page intentionally left blank.

August 14, 1998

To: Patrice G. Cole, Radian International, 1093 Commerce Park Drive,
Suite 100, Oak Ridge, TN 37830-8029

From: Gary D. Jenkins, TVA Water Management - Environmental Compliance,
202 West Blythe Street, PO Box 280, Paris, TN 38242-0280

Subject: Fish Sampling at the Defense Distribution Depot, Memphis, TN

Per your request, the following is a brief report on our findings in the two ponds at the Defense Distribution Depot in Memphis, TN.

Robert Pickett and I arrived on site at approximately 1620 Wednesday, August 12, 1998. We set three 100-foot long experimental gill nets (with five 25-foot panels of varying bar mesh size from ½ in. up to 2½ in.) in the four acre pond and fished them overnight. At approximately 0610 the following morning, we ran all three nets. There were no fish in any of the nets. Prior to running the nets, we observed small fish surfacing in the pond. We reset the nets and began electrofishing about 0645. With the electrofishing gear, we collected approximately 100 golden shiners (*Notemigonus crysoleucas*) of various sizes ranging from about one inch up to six inches. We observed several hundred other golden shiners which were not collected. We also collected three adult bullfrogs. We ran the nets again and caught only two adult golden shiners. All specimens kept were given to you for further analysis.

We exerted over 70 minutes of electrofishing sampling effort in this pond, making two shoreline runs and a series of transects that covered the entire pond surface area. Additionally, we exerted over 46 net-fishing hours on the pond. The only fish species encountered was golden shiner. With the amount of sampling effort exerted in this pond, I am almost certain no other fish species were present in this pond at the time of our sampling. If other fish species are present in the pond, their numbers are so low they would be of little interest to anglers.

After completing our sampling of the larger pond, we launched the boat in the small pond and began electrofishing. We collected several Western mosquitofish (*Gambusia affinis*) and observed hundreds more that were too small to capture in the dip net. We also collected 11 goldfish (*Carassius auratus*) and observed five other goldfish. We collected three adult bullfrogs. Six goldfish and the bullfrogs were given to you for further analysis. As with the larger pond, I feel the fish community of this pond is of no interest to anglers.

If you have any questions or need additional information, please call me at (901) 641-2012. If I can be of assistance in future projects, please feel free to call me.

This page intentionally left blank.

Appendix D

PHOTOGRAPHS

This page intentionally left blank.

Photo 1. Setting Gill Nets in Lake Danielson

Photo 2. Electro-Fishing Lake Danielson

Photo 3. Electro-Fishing Lake Danielson

Photo 4. Electro-Fishing Lake Danielson

Photo 5. Electro-Fishing Lake Danielson

Photo 6. Collecting and Identifying Fish from Lake Danielson

Photo 7. Golden Shiners Collected from Lake Danielson

Photo 8. Checking Gill Nets in Lake Danielson

Photo 9. Retrieving Boat from Lake Danielson

Photo 10. Launching Boat in Golf Course Pond

Photo 11. Electro-Fishing Golf Course Pond

Photo 12. Specimens Collected from Lake Danielson and Golf Course Pond

Appendix E

CHAIN-OF-CUSTODY RECORDS

This page intentionally left blank.

Analysis Request / Environmental Services Chain of Custody

Lancaster Laboratories
A division of Thermo Analytical Inc.

For Lancaster Laboratories use only
Acct. # 6149 Sample # 2792920-35

Please print. Instructions on reverse side correspond with circled numbers.

(1)
Client: Radian International Acct. #:
Project Name/#: Defense Depot PWSID #:
Project Manager: Patrice Cole P.O.#
Sampler: Patrice Cole Quote #:
Name of state where samples were collected: Tennessee

(2) (3) (4) (5) (6)

Sample Identification	Date Collected	Time Collected	(3) Grab	(3) Composite	(4) Matrix: Soil	(4) Matrix: Water (Potable / NPDES)	(4) Matrix: Other	Total # of Containers	(5) Analyses Requested: Pesticides (1828)	Remarks	(6) Temperature of samples upon receipt (if requested)
Sediment #1-15	10/1/97		X				X sediment	13	X	Cooled to 4°C	
Fish #1+2	10/1/97		X				X	2	X	Frozen	
rinseate blank	10/1/97		X			X		1	X	Cooled to 4°C	

For lab use only
FSC:
SCR #: 101590

(7) **Turnaround Time Requested** (TAT) (please circle): Normal Rush
(Rush TAT is subject to Lancaster Laboratories approval and surcharge.)
Date results are needed:
Rush results requested by (please circle): Phone Fax
Phone #: Fax #:

(8) **Data Package Options** (please circle if requested)
QC Summary Type VI (Raw Data)
Type I (Tier I) GLP
Type II (Tier II) Other
Type III (NJ Red. Del.)
Type IV (CLP)

SDG Complete? Yes No

Site-specific QC required? Yes No
(If yes, indicate QC sample and submit triplicate volume.)
Internal Chain of Custody required? Yes No

(9)

Relinquished by:	Date	Time	Received by:	Date	Time
Patrice Cole	10/2/97	3:00pm			
Relinquished by:	Date	Time	Received by:	Date	Time
Relinquished by:	Date	Time	Received by:	Date	Time
Relinquished by:	Date	Time	Received by:	Date	Time
Relinquished by:	Date	Time	Received by: Marlan Collins	10-3-97	0900

RADIAN INTERNATIONAL LLC

Acct# 6149
cc# 298517-21

Chain of Custody Record

PROJECT: Defense Distribution Depot, Memphis
SITE: Golf Course Ponds
PREPARED BY (Signature): Patricia Cole

FIELD SAMPLE I.D.	SAMPLE MATRIX	DATE/TIME	MS/MSD	NO. OF CONTAINERS	ANALYSES: Pesticides (8081)	REMARKS
Fish Sample 1	fish	8-13-98		1		Frozen / dry ice
Fish Sample 2	fish	8-13-98		1		Frozen / dry ice
Fish Sample 3	fish	8-13-98		1		Frozen / dry ice
Frog Sample 1	frog	8-13-98		1		Frozen / dry ice
Frog Sample 2	frog	8-13-98		1		Frozen / dry ice

REMARKS: Analyze frog leg muscle tissue only (composite samples).
Please analyze whole fish as composite samples.

RECEIVED BY:	DATE	TIME	RELINQUISHED BY:	DATE	TIME	RECEIVED BY:	DATE	TIME	RELINQUISHED BY:	DATE	TIME
A Hunter	8/17	1528									

LAB USE ONLY

RECEIVED FOR LABORATORY BY:	DATE	TIME	AIRBILL NO.	OPENED BY:	DATE	TIME	TEMP°C	SEAL #	CONDITION
Harla Collins	8-18-98	1030		Harla Collins	8-18-98	1120	-18.0	NA	Good

REMARKS: 804838444183

IEG-4\FH\3-96-30337coc.FH5

Appendix F
ANALYTICAL DATA

This page intentionally left blank.

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792920
Collected: 10/ 1/97 at 11:35 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #1 Grab Sediment Sample

Defense Depot - TN
1SED- SDG#: DED01-01

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 27.	27.
1982	Beta BHC	< 10.	10.	ug/kg	< 27.	27.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 27.	27.
1983	Delta BHC	< 10.	10.	ug/kg	< 27.	27.
1219	Heptachlor	< 10.	10.	ug/kg	< 27.	27.
1220	Aldrin	< 10.	10.	ug/kg	< 27.	27.
1984	Heptachlor Epoxide	20.	10.	ug/kg	54.	27.
1985	DDE	310.	100.	ug/kg	850.	270.
1986	DDD	78.	10.	ug/kg	211.	27.
1221	DDT	37.	10.	ug/kg	99.	27.
1222	Dieldrin	< 10.	10.	ug/kg	< 27.	27.
1223	Endrin	< 10.	10.	ug/kg	< 27.	27.
1859	Methoxychlor	< 50.	50.	ug/kg	< 140.	140.
1987	Chlordane	236.	50.	ug/kg	640.	140.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 5,400.	5,400.
1989	Endosulfan I	< 10.	10.	ug/kg	< 27.	27.
1990	Endosulfan II	< 10.	10.	ug/kg	< 27.	27.
1991	Endosulfan Sulfate	< 30.	30.	ug/kg	< 81.	81.
1992	Endrin Aldehyde	< 100.	100.	ug/kg	< 270.	270.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

21

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792921
Collected: 10/ 1/97 at 09:35 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #2 Grab Sediment Sample

Defense Depot - TN
2SED- SDG#: DED01-02

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 480.	480.
1982	Beta BHC	< 10.	10.	ug/kg	< 480.	480.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 480.	480.
1983	Delta BHC	< 10.	10.	ug/kg	< 480.	480.
1219	Heptachlor	< 10.	10.	ug/kg	< 480.	480.
1220	Aldrin	< 10.	10.	ug/kg	< 480.	480.
1984	Heptachlor Epoxide	< 10.	10.	ug/kg	< 480.	480.
1985	DDE	< 10.	10.	ug/kg	< 480.	480.
1986	DDD	< 10.	10.	ug/kg	< 480.	480.
1221	DDT	< 10.	10.	ug/kg	< 480.	480.
1222	Dieldrin	< 10.	10.	ug/kg	< 480.	480.
1223	Endrin	< 10.	10.	ug/kg	< 480.	480.
1859	Methoxychlor	< 50.	50.	ug/kg	< 2,400.	2,400.
1987	Chlordane	< 50.	50.	ug/kg	< 2,400.	2,400.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 95,000.	95,000.
1989	Endosulfan I	< 10.	10.	ug/kg	< 480.	480.
1990	Endosulfan II	< 10.	10.	ug/kg	< 480.	480.
1991	Endosulfan Sulfate	< 30.	30.	ug/kg	< 1,400.	1,400.
1992	Endrin Aldehyde	< 100.	100.	ug/kg	< 4,800.	4,800.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792922
Collected: 10/ 1/97 at 10:10 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #3 Grab Sediment Sample

Defense Depot - TN
3SED- SDG#: DED01-03

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 52.	52.
1982	Beta BHC	< 10.	10.	ug/kg	< 52.	52.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 52.	52.
1983	Delta BHC	< 10.	10.	ug/kg	< 52.	52.
1219	Heptachlor	< 10.	10.	ug/kg	< 52.	52.
1220	Aldrin	< 10.	10.	ug/kg	< 52.	52.
1984	Heptachlor Epoxide	17.	10.	ug/kg	87.	52.
1985	DDE	316.	10.	ug/kg	1,650.	52.
1986	DDD	103.	10.	ug/kg	537.	52.
1221	DDT	30.	10.	ug/kg	157.	52.
1222	Dieldrin	< 10.	10.	ug/kg	< 52.	52.
1223	Endrin	< 10.	10.	ug/kg	< 52.	52.
859	Methoxychlor	< 100.	100.	ug/kg	< 520.	520.
987	Chlordane	747.	50.	ug/kg	3,890.	260.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 10,000.	10,000.
1989	Endosulfan I	< 10.	10.	ug/kg	< 52.	52.
1990	Endosulfan II	< 10.	10.	ug/kg	< 52.	52.
1991	Endosulfan Sulfate	< 60.	60.	ug/kg	< 310.	310.
1992	Endrin Aldehyde	< 100.	100.	ug/kg	< 520.	520.

Due to interfering peaks on the chromatogram, the values reported represent the lowest quantitation limits obtainable.
Despite numerous cleanup methods, we were unable to reach our usual quantitation limits.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

25

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792923
Collected: 10/ 1/97 at 14:45 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #5 Grab Sediment Sample

Defense Depot - TN
5SED- SDG#: DED01-04

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 53.	53.
1982	Beta BHC	< 10.	10.	ug/kg	< 53.	53.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 53.	53.
1983	Delta BHC	< 10.	10.	ug/kg	< 53.	53.
1219	Heptachlor	< 10.	10.	ug/kg	< 53.	53.
1220	Aldrin	< 10.	10.	ug/kg	< 53.	53.
1984	Heptachlor Epoxide	< 10.	10.	ug/kg	< 53.	53.
1985	DDE	73.	10.	ug/kg	386.	53.
1986	DDD	23.	10.	ug/kg	123.	53.
1221	DDT	< 10.	10.	ug/kg	< 53.	53.
1222	Dieldrin	< 10.	10.	ug/kg	< 53.	53.
1223	Endrin	< 10.	10.	ug/kg	< 53.	53.
1859	Methoxychlor	< 50.	50.	ug/kg	< 270.	270.
1987	Chlordane	193.	50.	ug/kg	1,030.	270.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 11,000.	11,000.
1989	Endosulfan I	< 10.	10.	ug/kg	< 53.	53.
1990	Endosulfan II	< 10.	10.	ug/kg	< 53.	53.
1991	Endosulfan Sulfate	< 30.	30.	ug/kg	< 160.	160.
1992	Endrin Aldehyde	< 100.	100.	ug/kg	< 530.	530.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

27

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792924
Collected: 10/ 1/97 at 14:00 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #6 Grab Sediment Sample

Defense Depot - TN
6SED- SDG#: DED01-05

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. 0T-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 30.	30.
1982	Beta BHC	< 10.	10.	ug/kg	< 30.	30.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 30.	30.
1983	Delta BHC	< 10.	10.	ug/kg	< 30.	30.
1219	Heptachlor	< 10.	10.	ug/kg	< 30.	30.
1220	Aldrin	< 10.	10.	ug/kg	< 30.	30.
1984	Heptachlor Epoxide	29.	10.	ug/kg	88.	30.
1985	DDE	490.	100.	ug/kg	1,470.	300.
1986	DDD	236.	10.	ug/kg	712.	30.
1221	DDT	55.	10.	ug/kg	166.	30.
1222	Dieldrin	< 10.	10.	ug/kg	< 30.	30.
1223	Endrin	< 10.	10.	ug/kg	< 30.	30.
1859	Methoxychlor	< 100.	100.	ug/kg	< 300.	300.
987	Chlordane	713.	50.	ug/kg	2,150.	150.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 6,000.	6,000.
1989	Endosulfan I	< 10.	10.	ug/kg	< 30.	30.
1990	Endosulfan II	< 10.	10.	ug/kg	< 30.	30.
1991	Endosulfan Sulfate	< 60.	60.	ug/kg	< 180.	180.
1992	Endrin Aldehyde	< 200.	200.	ug/kg	< 600.	600.

Due to interfering peaks on the chromatogram, the values reported represent the lowest quantitation limits obtainable.
Despite numerous cleanup methods, we were unable to reach our usual quantitation limits.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

29

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792925
Collected: 10/ 1/97 at 15:45 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #7 Grab Sediment Sample

Defense Depot - TN
7SED- SDG#: DED01-06

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. 0T-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 15.	15.
1982	Beta BHC	< 10.	10.	ug/kg	< 15.	15.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 15.	15.
1983	Delta BHC	< 10.	10.	ug/kg	< 15.	15.
1219	Heptachlor	< 10.	10.	ug/kg	< 15.	15.
1220	Aldrin	< 10.	10.	ug/kg	< 15.	15.
1984	Heptachlor Epoxide	< 10.	10.	ug/kg	< 15.	15.
1985	DDE	51.	10.	ug/kg	76.	15.
1986	DDD	31.	10.	ug/kg	46.	15.
1221	DDT	48.	10.	ug/kg	71.	15.
1222	Dieldrin	< 10.	10.	ug/kg	< 15.	15.
1223	Endrin	< 10.	10.	ug/kg	< 15.	15.
1859	Methoxychlor	< 100.	100.	ug/kg	< 150.	150.
1987	Chlordane	< 50.	50.	ug/kg	< 74.	74.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 3,000.	3,000.
1989	Endosulfan I	< 10.	10.	ug/kg	< 15.	15.
1990	Endosulfan II	< 10.	10.	ug/kg	< 15.	15.
1991	Endosulfan Sulfate	< 60.	60.	ug/kg	< 89.	89.
1992	Endrin Aldehyde	< 200.	200.	ug/kg	< 300.	300.

Due to interfering peaks on the chromatogram, the values reported represent the lowest quantitation limits obtainable.
Despite numerous cleanup methods, we were unable to reach our usual quantitation limits.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792926
Collected: 10/ 1/97 at 16:00 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #8 Grab Sediment Sample

Defense Depot - TN
8SED- SDG#: DED01-07

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 40.	40.
1982	Beta BHC	< 10.	10.	ug/kg	< 40.	40.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 40.	40.
1983	Delta BHC	< 10.	10.	ug/kg	< 40.	40.
1219	Heptachlor	< 10.	10.	ug/kg	< 40.	40.
1220	Aldrin	< 10.	10.	ug/kg	< 40.	40.
1984	Heptachlor Epoxide	17.	10.	ug/kg	67.	40.
1985	DDE	296.	10.	ug/kg	1,170.	40.
1986	DDD	113.	10.	ug/kg	448.	40.
1221	DDT	41.	10.	ug/kg	164.	40.
1222	Dieldrin	< 10.	10.	ug/kg	< 40.	40.
1223	Endrin	< 10.	10.	ug/kg	< 40.	40.
1859	Methoxychlor	< 100.	100.	ug/kg	< 400.	400.
987	Chlordane	602.	50.	ug/kg	2,390.	200.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 7,900.	7,900.
1989	Endosulfan I	< 10.	10.	ug/kg	< 40.	40.
1990	Endosulfan II	< 10.	10.	ug/kg	< 40.	40.
1991	Endosulfan Sulfate	< 60.	60.	ug/kg	< 240.	240.
1992	Endrin Aldehyde	< 200.	200.	ug/kg	< 790.	790.

Due to interfering peaks on the chromatogram, the values reported represent the lowest quantitation limits obtainable.
Despite numerous cleanup methods, we were unable to reach our usual quantitation limits.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

33

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792927
Collected: 10/ 1/97 at 15:15 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #9 Grab Sediment Sample

Defense Depot - TN
9SED- SDG#: DED01-08

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 21.	21.
1982	Beta BHC	< 10.	10.	ug/kg	< 21.	21.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 21.	21.
1983	Delta BHC	< 10.	10.	ug/kg	< 21.	21.
1219	Heptachlor	< 10.	10.	ug/kg	< 21.	21.
1220	Aldrin	< 10.	10.	ug/kg	< 21.	21.
1984	Heptachlor Epoxide	< 10.	10.	ug/kg	< 21.	21.
1985	DDE	49.	10.	ug/kg	102.	21.
1986	DDD	16.	10.	ug/kg	33.	21.
1221	DDT	< 10.	10.	ug/kg	< 21.	21.
1222	Dieldrin	< 10.	10.	ug/kg	< 21.	21.
1223	Endrin	< 10.	10.	ug/kg	< 21.	21.
1859	Methoxychlor	< 100.	100.	ug/kg	< 210.	210.
1987	Chlordane	102.	50.	ug/kg	210.	100.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 4,100.	4,100.
1989	Endosulfan I	< 10.	10.	ug/kg	< 21.	21.
1990	Endosulfan II	< 10.	10.	ug/kg	< 21.	21.
1991	Endosulfan Sulfate	< 60.	60.	ug/kg	< 120.	120.
1992	Endrin Aldehyde	< 200.	200.	ug/kg	< 410.	410.

Due to interfering peaks on the chromatogram, the values reported represent the lowest quantitation limits obtainable.
Despite numerous cleanup methods, we were unable to reach our usual quantitation limits.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

35

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792928
Collected: 10/ 1/97 at 10:30 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #10 Grab Sediment Sample

Defense Depot - TN
10SED SDG#: DED01-09

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 35.	35.
1982	Beta BHC	< 10.	10.	ug/kg	< 35.	35.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 35.	35.
1983	Delta BHC	< 10.	10.	ug/kg	< 35.	35.
1219	Heptachlor	< 10.	10.	ug/kg	< 35.	35.
1220	Aldrin	< 10.	10.	ug/kg	< 35.	35.
1984	Heptachlor Epoxide	33.	10.	ug/kg	115.	35.
1985	DDE	510.	100.	ug/kg	1,780.	350.
1986	DDD	289.	10.	ug/kg	1,000.	35.
1221	DDT	66.	10.	ug/kg	227.	35.
1222	Dieldrin	< 10.	10.	ug/kg	< 35.	35.
1223	Endrin	< 10.	10.	ug/kg	< 35.	35.
859	Methoxychlor	< 100.	100.	ug/kg	< 350.	350.
987	Chlordane	704.	50.	ug/kg	2,440.	170.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 6,900.	6,900.
1989	Endosulfan I	< 10.	10.	ug/kg	< 35.	35.
1990	Endosulfan II	< 10.	10.	ug/kg	< 35.	35.
1991	Endosulfan Sulfate	< 60.	60.	ug/kg	< 210.	210.
1992	Endrin Aldehyde	< 200.	200.	ug/kg	< 690.	690.

Due to interfering peaks on the chromatogram, the values reported represent the lowest quantitation limits obtainable.
Despite numerous cleanup methods, we were unable to reach our usual quantitation limits.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

37

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792929
Collected: 10/ 1/97 at 17:00 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #11 Grab Sediment Sample

Defense Depot - TN
11SED SDG#: DED01-10

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 36.	36.
1982	Beta BHC	< 10.	10.	ug/kg	< 36.	36.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 36.	36.
1983	Delta BHC	< 10.	10.	ug/kg	< 36.	36.
1219	Heptachlor	< 10.	10.	ug/kg	< 36.	36.
1220	Aldrin	< 10.	10.	ug/kg	< 36.	36.
1984	Heptachlor Epoxide	< 10.	10.	ug/kg	< 36.	36.
1985	DDE	26.	10.	ug/kg	95.	36.
1986	DDD	13.	10.	ug/kg	48.	36.
1221	DDT	< 10.	10.	ug/kg	< 36.	36.
1222	Dieldrin	< 10.	10.	ug/kg	< 36.	36.
1223	Endrin	< 10.	10.	ug/kg	< 36.	36.
1859	Methoxychlor	< 100.	100.	ug/kg	< 360.	360.
1987	Chlordane	< 50.	50.	ug/kg	< 180.	180.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 7,200.	7,200.
1989	Endosulfan I	< 10.	10.	ug/kg	< 36.	36.
1990	Endosulfan II	< 10.	10.	ug/kg	< 36.	36.
1991	Endosulfan Sulfate	< 60.	60.	ug/kg	< 220.	220.
1992	Endrin Aldehyde	< 200.	200.	ug/kg	< 720.	720.

Due to interfering peaks on the chromatogram, the values reported represent the lowest quantitation limits obtainable.
Despite numerous cleanup methods, we were unable to reach our usual quantitation limits.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

39

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792930
Collected: 10/ 1/97 at 17:15 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #12 Grab Sediment Sample

Defense Depot - TN
12SED SDG#: DED01-11

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 30.	30.
1982	Beta BHC	< 10.	10.	ug/kg	< 30.	30.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 30.	30.
1983	Delta BHC	< 10.	10.	ug/kg	< 30.	30.
1219	Heptachlor	< 10.	10.	ug/kg	< 30.	30.
1220	Aldrin	< 10.	10.	ug/kg	< 30.	30.
1984	Heptachlor Epoxide	< 10.	10.	ug/kg	< 30.	30.
1985	DDE	32.	10.	ug/kg	95.	30.
1986	DDD	13.	10.	ug/kg	38.	30.
1221	DDT	< 10.	10.	ug/kg	< 30.	30.
1222	Dieldrin	< 10.	10.	ug/kg	< 30.	30.
1223	Endrin	< 10.	10.	ug/kg	< 30.	30.
859	Methoxychlor	< 100.	100.	ug/kg	< 300.	300.
987	Chlordane	< 50.	50.	ug/kg	< 150.	150.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 6,000.	6,000.
1989	Endosulfan I	< 10.	10.	ug/kg	< 30.	30.
1990	Endosulfan II	< 10.	10.	ug/kg	< 30.	30.
1991	Endosulfan Sulfate	< 60.	60.	ug/kg	< 180.	180.
1992	Endrin Aldehyde	< 200.	200.	ug/kg	< 600.	600.

Due to interfering peaks on the chromatogram, the values reported represent the lowest quantitation limits obtainable.
Despite numerous cleanup methods, we were unable to reach our usual quantitation limits.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

41

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792931
Collected: 10/ 1/97 at 17:30 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #13 Grab Sediment Sample

Defense Depot - TN
13SED SDG#: DED01-12

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 31.	31.
1982	Beta BHC	< 10.	10.	ug/kg	< 31.	31.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 31.	31.
1983	Delta BHC	< 10.	10.	ug/kg	< 31.	31.
1219	Heptachlor	< 10.	10.	ug/kg	< 31.	31.
1220	Aldrin	< 10.	10.	ug/kg	< 31.	31.
1984	Heptachlor Epoxide	< 10.	10.	ug/kg	< 31.	31.
1985	DDE	43.	10.	ug/kg	134.	31.
1986	DDD	21.	10.	ug/kg	65.	31.
1221	DDT	11.	10.	ug/kg	35.	31.
1222	Dieldrin	< 10.	10.	ug/kg	< 31.	31.
1223	Endrin	< 10.	10.	ug/kg	< 31.	31.
1859	Methoxychlor	< 50.	50.	ug/kg	< 150.	150.
1987	Chlordane	< 50.	50.	ug/kg	< 150.	150.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 6,200.	6,200.
1989	Endosulfan I	< 10.	10.	ug/kg	< 31.	31.
1990	Endosulfan II	< 10.	10.	ug/kg	< 31.	31.
1991	Endosulfan Sulfate	< 30.	30.	ug/kg	< 93.	93.
1992	Endrin Aldehyde	< 100.	100.	ug/kg	< 310.	310.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2792932
Collected: 10/ 1/97 at 14:25 by PC

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

SP #15 Grab Sediment Sample

Defense Depot - TN
15SED SDG#: DED01-13

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 30.	30.
1982	Beta BHC	< 20.	20.	ug/kg	< 60.	60.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 30.	30.
1983	Delta BHC	< 20.	20.	ug/kg	< 60.	60.
1219	Heptachlor	< 20.	20.	ug/kg	< 60.	60.
1220	Aldrin	< 20.	20.	ug/kg	< 60.	60.
1984	Heptachlor Epoxide	38.	20.	ug/kg	114.	60.
1985	DDE	710.	100.	ug/kg	2,120.	300.
1986	DDD	296.	20.	ug/kg	883.	60.
1221	DDT	78.	20.	ug/kg	234.	60.
1222	Dieldrin	< 20.	20.	ug/kg	< 60.	60.
1223	Endrin	< 20.	20.	ug/kg	< 60.	60.
1859	Methoxychlor	< 100.	100.	ug/kg	< 300.	300.
1987	Chlordane	960.	100.	ug/kg	2,870.	300.
1988	Toxaphene	< 4,000.	4,000.	ug/kg	< 12,000.	12,000.
1989	Endosulfan I	< 20.	20.	ug/kg	< 60.	60.
1990	Endosulfan II	< 20.	20.	ug/kg	< 60.	60.
1991	Endosulfan Sulfate	< 60.	60.	ug/kg	< 180.	180.
1992	Endrin Aldehyde	< 200.	200.	ug/kg	< 600.	600.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. G5 2792933
Collected: 10/ 1/97

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

Fish #1 Grab Sample

Defense Depot - TN
FISH1 SDG#: DED01-14

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 10.	10.	ug/kg	< 37.	37.
1982	Beta BHC	< 10.	10.	ug/kg	< 37.	37.
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg	< 37.	37.
1983	Delta BHC	< 10.	10.	ug/kg	< 37.	37.
1219	Heptachlor	< 10.	10.	ug/kg	< 37.	37.
1220	Aldrin	< 10.	10.	ug/kg	< 37.	37.
1984	Heptachlor Epoxide	< 10.	10.	ug/kg	< 37.	37.
1985	DDE	3,190.	100.	ug/kg	11,900.	370.
1986	DDD	490.	100.	ug/kg	1,820.	370.
1221	DDT	12.	10.	ug/kg	46.	37.
1222	Dieldrin	45.	10.	ug/kg	169.	37.
1223	Endrin	< 10.	10.	ug/kg	< 37.	37.
1859	Methoxychlor	< 50.	50.	ug/kg	< 190.	190.
1987	Chlordane	732.	50.	ug/kg	2,740.	190.
1988	Toxaphene	< 2,000.	2,000.	ug/kg	< 7,500.	7,500.
1989	Endosulfan I	< 10.	10.	ug/kg	< 37.	37.
1990	Endosulfan II	< 10.	10.	ug/kg	< 37.	37.
1991	Endosulfan Sulfate	< 30.	30.	ug/kg	< 110.	110.
1992	Endrin Aldehyde	< 100.	100.	ug/kg	< 370.	370.

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

47

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. G5 2792934
Collected: 10/ 1/97

Submitted: 10/ 3/97 Reported: 10/22/97
Discard: 11/ 6/97

Fish #2 Grab Sample

Defense Depot - TN
FISH2 SDG#: DED01-15

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. OT-01220-S-06
Rel..

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	LIMIT OF QUANTITATION	UNITS
Pesticides/PCBs in Solids				
1981	Alpha BHC	< 10.	10.	ug/kg
1982	Beta BHC	< 10.	10.	ug/kg
1218	Gamma BHC - Lindane	< 10.	10.	ug/kg
1983	Delta BHC	< 10.	10.	ug/kg
1219	Heptachlor	< 10.	10.	ug/kg
1220	Aldrin	< 10.	10.	ug/kg
1984	Heptachlor Epoxide	< 10.	10.	ug/kg
1985	DDE	600.	100.	ug/kg
1986	DDD	124.	10.	ug/kg
1221	DDT	< 10.	10.	ug/kg
1222	Dieldrin	13.	10.	ug/kg
1223	Endrin	< 10.	10.	ug/kg
1859	Methoxychlor	< 50.	50.	ug/kg
1987	Chlordane	166.	50.	ug/kg
1988	Toxaphene	< 2,000.	2,000.	ug/kg
1989	Endosulfan I	< 10.	10.	ug/kg
1990	Endosulfan II	< 10.	10.	ug/kg
1991	Endosulfan Sulfate	< 30.	30.	ug/kg
1992	Endrin Aldehyde	< 100.	100.	ug/kg

Questions? Contact your Client Services Representative
Lisa M. Hetrick at (717) 656-2300

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

49

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2983517
Collected: 8/13/98

Submitted: 8/18/98 Reported: 9/ 4/98
Discard: 9/15/98

Fish 1 Composite Sample
Golf Course Ponds
Defense Distribution Depot Memphis, TN
FSH-1 SDG#: DDD01-01

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O.
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	AS RECEIVED LIMIT OF QUANTITATION	UNITS	DRY WEIGHT RESULTS	DRY WEIGHT LIMIT OF QUANTITATION
Pesticides/PCBs in Solids						
1981	Alpha BHC	< 3.3	3.3	ug/kg	< 15.	15.
1982	Beta BHC	< 3.3	3.3	ug/kg	< 15.	15.
1218	Gamma BHC - Lindane	< 3.3	3.3	ug/kg	< 15.	15.
1983	Delta BHC	< 3.3	3.3	ug/kg	< 15.	15.
1219	Heptachlor	< 3.3	3.3	ug/kg	< 15.	15.
1220	Aldrin	< 3.3	3.3	ug/kg	< 15.	15.
1984	Heptachlor Epoxide	< 3.3	3.3	ug/kg	< 15.	15.
1985	DDE	762.	67.	ug/kg	3,540.	310.
1986	DDD	257.	67.	ug/kg	1,200.	310.
1221	DDT	17.9	6.7	ug/kg	83.	31.
1222	Dieldrin	36.7	6.7	ug/kg	171.	31.
1223	Endrin	< 6.7	6.7	ug/kg	< 31.	31.
1859	Methoxychlor	< 33.	33.	ug/kg	< 150.	150.
1987	Chlordane	400.	170.	ug/kg	1,840.	790.
1988	Toxaphene	< 330.	330.	ug/kg	< 1,500.	1,500.
1989	Endosulfan I	< 3.3	3.3	ug/kg	< 15.	15.
1990	Endosulfan II	< 6.7	6.7	ug/kg	< 31.	31.
1991	Endosulfan Sulfate	< 6.7	6.7	ug/kg	< 31.	31.
1992	Endrin Aldehyde	< 6.7	6.7	ug/kg	< 31.	31.
1993	PCB-1016	< 170.	170.	ug/kg	< 790.	790.
1994	PCB-1221	< 170.	170.	ug/kg	< 790.	790.
1995	PCB-1232	< 170.	170.	ug/kg	< 790.	790.
1996	PCB-1242	< 170.	170.	ug/kg	< 790.	790.
1997	PCB-1248	< 170.	170.	ug/kg	< 790.	790.
1998	PCB-1254	< 170.	170.	ug/kg	< 790.	790.
1999	PCB-1260	< 170.	170.	ug/kg	< 790.	790.

A disparity of >40% between the primary and confirmatory analysis occurred. Due to suspected interference, the lower result was reported for Chlordane.

The % difference for the calibration verification standard fell outside the +/- 15% criteria for the compounds listed below. Since the average of the % difference values met the criteria, the results were reported for 4,4'-DDE.

Questions? Contact your Client Services Representative
Kay G. Hower at (717) 656-2300

10

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax: 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2983518
Collected: 8/13/98

Submitted: 8/18/98 Reported: 9/ 4/98
Discard: 9/15/98

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O.
Rel.

Fish 2 Composite Sample
Golf Course Ponds
Defense Distribution Depot Memphis, TN
FSH-2 SDG#: DDD01-02

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	LIMIT OF QUANTITATION	UNITS
Pesticides/PCBs in Solids				
1981	Alpha BHC	< 3.3	3.3	ug/kg
1982	Beta BHC	< 3.3	3.3	ug/kg
1218	Gamma BHC - Lindane	< 3.3	3.3	ug/kg
1983	Delta BHC	< 3.3	3.3	ug/kg
1219	Heptachlor	< 3.3	3.3	ug/kg
1220	Aldrin	< 3.3	3.3	ug/kg
1984	Heptachlor Epoxide	< 3.3	3.3	ug/kg
1985	DDE	1,440.	67.	ug/kg
1986	DDD	160.	6.7	ug/kg
1221	DDT	12.6	6.7	ug/kg
1222	Dieldrin	85.9	6.7	ug/kg
1223	Endrin	< 6.7	6.7	ug/kg
1859	Methoxychlor	< 33.	33.	ug/kg
1987	Chlordane	340.	170.	ug/kg
1988	Toxaphene	< 330.	330.	ug/kg
1989	Endosulfan I	< 3.3	3.3	ug/kg
1990	Endosulfan II	< 6.7	6.7	ug/kg
1991	Endosulfan Sulfate	< 6.7	6.7	ug/kg
1992	Endrin Aldehyde	< 6.7	6.7	ug/kg
1993	PCB-1016	< 170.	170.	ug/kg
1994	PCB-1221	< 170.	170.	ug/kg
1995	PCB-1232	< 170.	170.	ug/kg
1996	PCB-1242	< 170.	170.	ug/kg
1997	PCB-1248	< 170.	170.	ug/kg
1998	PCB-1254	< 170.	170.	ug/kg
1999	PCB-1260	< 170.	170.	ug/kg

Questions? Contact your Client Services Representative
Kay G. Hower at (717) 656-2300

12

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax: 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2983519
Collected: 8/13/98

Submitted: 8/18/98 Reported: 9/ 4/98
Discard: 9/15/98

Fish 3 Composite Sample
Golf Course Ponds
Defense Distribution Depot Memphis, TN
FSH-3 SDG#: DDD01-03

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O.
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	LIMIT OF QUANTITATION	UNITS
Pesticides/PCBs in Solids				
1981	Alpha BHC	< 6.6	6.6	ug/kg
1982	Beta BHC	< 6.6	6.6	ug/kg
1218	Gamma BHC - Lindane	< 6.6	6.6	ug/kg
1983	Delta BHC	< 6.6	6.6	ug/kg
1219	Heptachlor	< 6.6	6.6	ug/kg
1220	Aldrin	< 6.6	6.6	ug/kg
1984	Heptachlor Epoxide	< 6.6	6.6	ug/kg
1985	DDE	1,570.	130.	ug/kg
1986	DDD	690.	130.	ug/kg
1221	DDT	109.	13.	ug/kg
1222	Dieldrin	167.	13.	ug/kg
1223	Endrin	< 13.	13.	ug/kg
1859	Methoxychlor	< 66.	66.	ug/kg
1987	Chlordane	560.	340.	ug/kg
1988	Toxaphene	< 660.	660.	ug/kg
1989	Endosulfan I	< 6.6	6.6	ug/kg
1990	Endosulfan II	< 13.	13.	ug/kg
1991	Endosulfan Sulfate	< 13.	13.	ug/kg
1992	Endrin Aldehyde	< 13.	13.	ug/kg
1993	PCB-1016	< 340.	340.	ug/kg
1994	PCB-1221	< 340.	340.	ug/kg
1995	PCB-1232	< 340.	340.	ug/kg
1996	PCB-1242	< 340.	340.	ug/kg
1997	PCB-1248	< 340.	340.	ug/kg
1998	PCB-1254	< 340.	340.	ug/kg
1999	PCB-1260	1,240.	340.	ug/kg

Questions? Contact your Client Services Representative
Kay G. Hower at (717) 656-2300

15

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax: 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2983520
Collected: 8/13/98

Submitted: 8/18/98 Reported: 9/ 4/98
Discard: 9/15/98

Frog 1 Composite Sample
Golf Course Ponds
Defense Distribution Depot Memphis, TN
FRG-1 SDG#: DDD01-04

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O.
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	LIMIT OF QUANTITATION	UNITS
Pesticides/PCBs in Solids				
1981	Alpha BHC	< 0.33	0.33	ug/kg
1982	Beta BHC	< 0.33	0.33	ug/kg
1218	Gamma BHC - Lindane	< 0.33	0.33	ug/kg
1983	Delta BHC	< 0.33	0.33	ug/kg
1219	Heptachlor	< 0.33	0.33	ug/kg
1220	Aldrin	< 0.33	0.33	ug/kg
1984	Heptachlor Epoxide	< 0.33	0.33	ug/kg
1985	DDE	17.0	0.67	ug/kg
1986	DDD	3.52	0.67	ug/kg
1221	DDT	2.50	0.67	ug/kg
1222	Dieldrin	31.4	6.7	ug/kg
1223	Endrin	< 0.67	0.67	ug/kg
1859	Methoxychlor	< 3.3	3.3	ug/kg
1987	Chlordane	< 17.	17.	ug/kg
1988	Toxaphene	< 33.	33.	ug/kg
1989	Endosulfan I	< 0.33	0.33	ug/kg
1990	Endosulfan II	< 0.67	0.67	ug/kg
1991	Endosulfan Sulfate	< 0.67	0.67	ug/kg
1992	Endrin Aldehyde	< 0.67	0.67	ug/kg
1993	PCB-1016	< 17.	17.	ug/kg
1994	PCB-1221	< 17.	17.	ug/kg
1995	PCB-1232	< 17.	17.	ug/kg
1996	PCB-1242	< 17.	17.	ug/kg
1997	PCB-1248	< 17.	17.	ug/kg
1998	PCB-1254	< 17.	17.	ug/kg
1999	PCB-1260	< 17.	17.	ug/kg

The % difference for the calibration verification standard fell outside the +/- 15% criteria for the compounds listed below. Since the average of the % difference values met the criteria, the results were reported for 4,4'-DDD.

Questions? Contact your Client Services Representative
Kay G. Hower at (717) 656-2300

18

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax: 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

LLI Sample No. SW 2983521
Collected: 8/13/98

Submitted: 8/18/98 Reported: 9/ 4/98
Discard: 9/15/98

Frog 2 Composite Sample
Golf Course Ponds
Defense Distribution Depot Memphis, TN
FRG-2 SDG#: DDD01-05*

Account No: 06149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O.
Rel.

CAT NO.	ANALYSIS NAME	AS RECEIVED RESULTS	LIMIT OF QUANTITATION	UNITS
Pesticides/PCBs in Solids				
1981	Alpha BHC	< 0.33	0.33	ug/kg
1982	Beta BHC	< 0.33	0.33	ug/kg
1218	Gamma BHC - Lindane	< 0.33	0.33	ug/kg
1983	Delta BHC	< 0.33	0.33	ug/kg
1219	Heptachlor	< 0.33	0.33	ug/kg
1220	Aldrin	< 0.33	0.33	ug/kg
1984	Heptachlor Epoxide	< 0.33	0.33	ug/kg
1985	DDE	1.85	0.67	ug/kg
1986	DDD	< 0.67	0.67	ug/kg
1221	DDT	< 0.67	0.67	ug/kg
1222	Dieldrin	23.8	0.67	ug/kg
1223	Endrin	< 0.67	0.67	ug/kg
1859	Methoxychlor	< 3.3	3.3	ug/kg
1987	Chlordane	< 17.	17.	ug/kg
1988	Toxaphene	< 33.	33.	ug/kg
1989	Endosulfan I	< 0.33	0.33	ug/kg
1990	Endosulfan II	< 0.67	0.67	ug/kg
1991	Endosulfan Sulfate	< 0.67	0.67	ug/kg
1992	Endrin Aldehyde	< 0.67	0.67	ug/kg
1993	PCB-1016	< 17.	17.	ug/kg
1994	PCB-1221	< 17.	17.	ug/kg
1995	PCB-1232	< 17.	17.	ug/kg
1996	PCB-1242	< 17.	17.	ug/kg
1997	PCB-1248	< 17.	17.	ug/kg
1998	PCB-1254	< 17.	17.	ug/kg
1999	PCB-1260	< 17.	17.	ug/kg

Questions? Contact your Client Services Representative
Kay G. Hower at (717) 656-2300

20

Respectfully Submitted
Jenifer E. Hess, B.S.
Group Leader Pesticides/PCBs

MEMBER ACIL
Lancaster Laboratories
2425 New Holland Pike
PO Box 12425
Lancaster, PA 17605-2425
717-656-2300 Fax: 717-656-2681