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THE MEMPHIS DEPOT
TENNESSEE

ADMINISTRATIVE RECORD
COVER SHEET

AR File Number 268

Baseline Risk Assessment Safety and Health Plan, and Sampling and Analysis Plan for Golf Course Impoundments at the Defense Distribution Depot, Memphis, Tennessee

December 1997

Prepared for:

U.S. Army Corps of Engineers
Mobile District

12 December 1997

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Mr. Ellis Pope
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Subject: **Draft Baseline Risk Assessment and Final Sampling and Analysis Plan and Safety and Health Plan for Golf Course Impoundments at the Defense Distribution Depot, Memphis, Tennessee**

Dear Mr. Pope:

Please find enclosed ten copies of the draft Baseline Risk Assessment, the final Sampling and Analysis Plan, and the final Safety and Health Plan for the Golf Course Impoundments at the Defense Distribution Depot, Memphis, Tennessee. The three reports are bound together. In the interest of efficiency, we hope to be able to provide replacement pages for any required changes, rather than resubmitting the entire bound document as the final version.

If you have any questions in this regard, please call Patrice Cole at (423) 220-8165.

Sincerely,

Lloyd A. Hinkle, P.E.
Principal Project Manager

LAH:rem

Enclosures

c: Mr. Kurt Braun, CESAM-PM-SP
Mr. Shawn Phillips
Ms. Patrice Cole, Radian International LLC
Project File

TAB

Baseline Risk Assessment

DRAFT

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:

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December 1997

TABLE OF CONTENTS

		Page
LIST OF FIGURES		iii
LIST OF TABLES		iii
ACRONYMS		iv
EXECUTIVE SUMMARY		v
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	4-3
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	6-4
6.6	Dieldrin	6-6
6.7	Heptachlor Epoxide	6-7
7.0	INITIAL RISK CHARACTERIZATION	7-1
8.0	FOLLOW-UP INVESTIGATION	8-1
9.0	UNCERTAINTY ANALYSIS	9-1
10.0	ECOLOGICAL RISK ASSESSMENT	10-1
11.0	CONCLUSIONS AND RECOMMENDATIONS	11-1

TABLE OF CONTENTS (CONTINUED)

Page

12.0 REFERENCES 12-1

Appendix A: RISK ASSESSMENT SPREADSHEETS
Appendix B: CHAIN-OF-CUSTODY RECORD
Appendix C: ANALYTICAL DATA
Appendix D: DETAILED TOXICITY SUMMARIES
Appendix E: PHOTOGRAPHS

LIST OF FIGURES

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

LIST OF TABLES

		Page
4-1.	1990 RI Sediment Sampling Results, Lake Danielson and Golf Course Pond	4-6
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	8-6
10-1.	Comparison of Maximum Detected Pesticide Concentrations in Golf Course Impoundments Surface Water and Sediment to EPA Region 4 Ecological Screening Values	10-2

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
PRE	Preliminary Risk Evaluation
RfC	Reference Concentration
RfD	Reference Dose
RI	Remedial Investigation
95 UCL	95% Upper Confidence Limit

EXECUTIVE SUMMARY

In early 1997, a baseline risk assessment 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.

The highest detected concentrations of DDT, DDE, and DDD in surface water and sediment samples collected from the golf course impoundments were below the U.S. Environmental Protection Agency (EPA) Region 4 ecological screening values, so further investigation or remediation based on ecological risk does not appear to be warranted.

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 1997 to provide more recent data for re-evaluating 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 for this risk assessment.

Based on the new pesticide 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 only fish caught during the September 1997 sampling event were Arkansas shiners (*Notropis girardi*), which are commonly used as bait fish. Analytical data on muscle tissue from a composite sample of several shiners were used as the representative exposure concentrations for pesticides in fish. 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. Remediation of contaminated sediments does not appear to be warranted.

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 is 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 land and buildings are owned by the U.S. Army and are leased by the Defense Logistics Agency. 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 after the Depot is closed.

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

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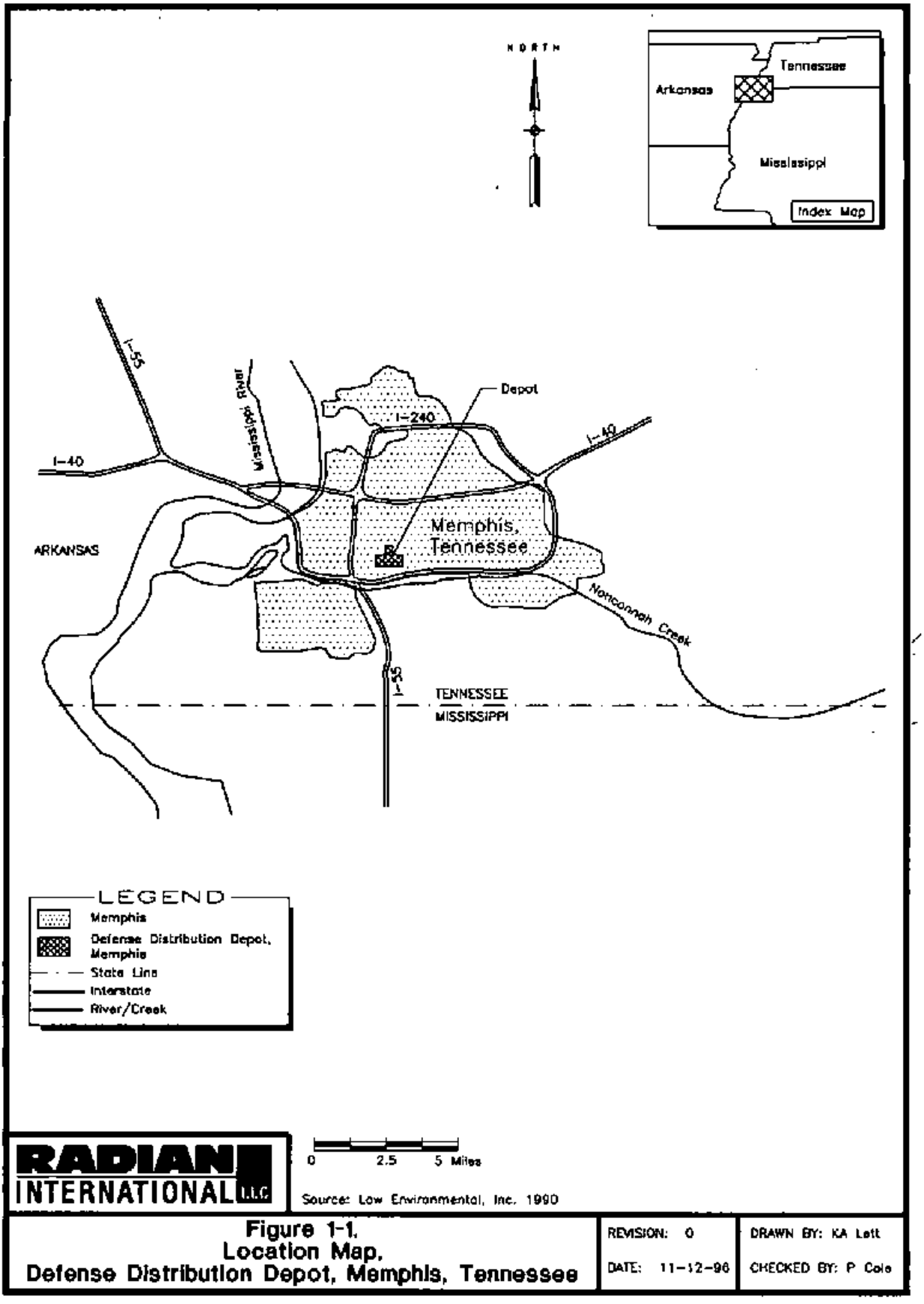

Figure 1-1.
Location Map.
Defense Distribution Depot, Memphis, Tennessee

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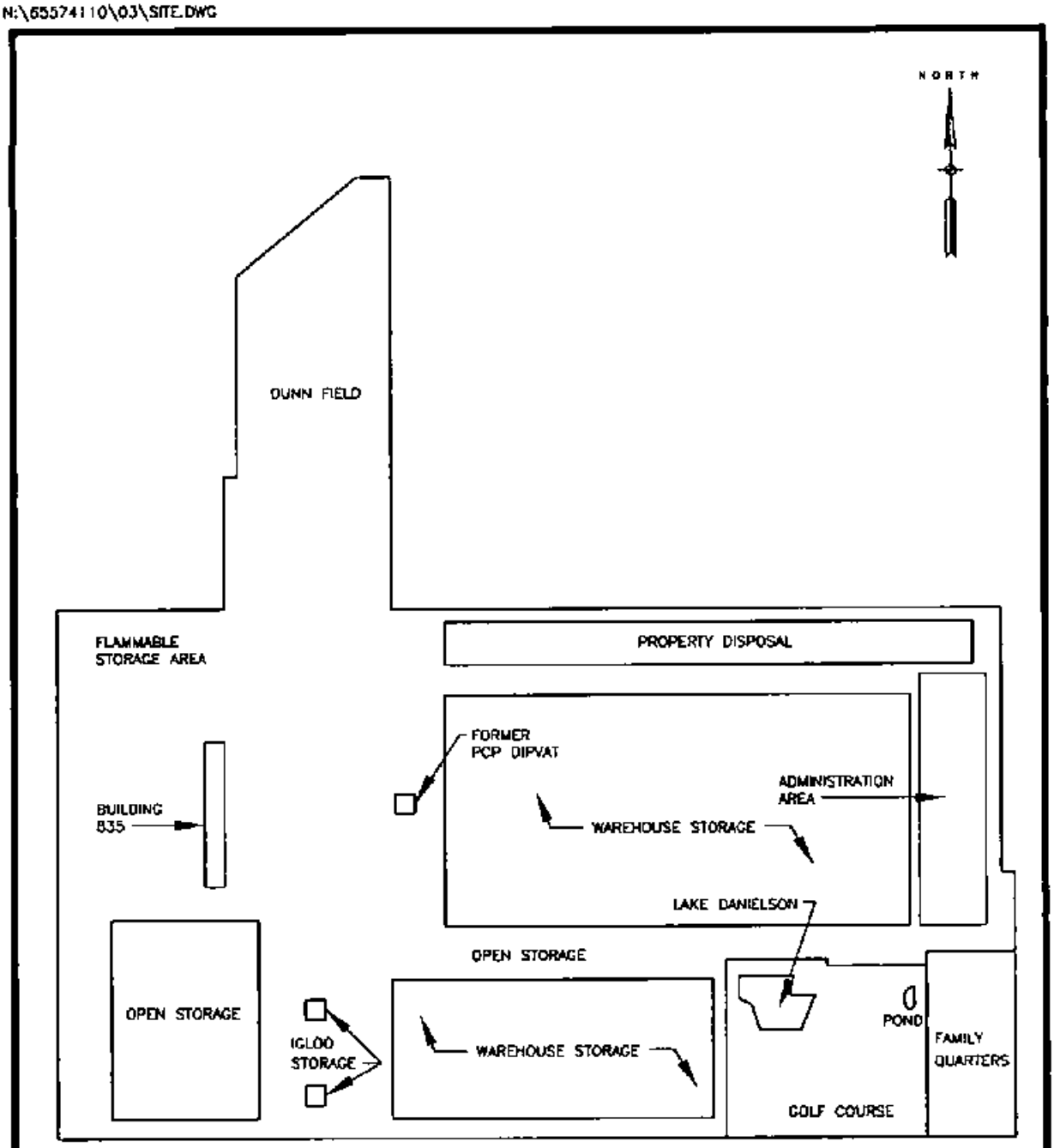

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Source: Low Environmental, Inc. 1990

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

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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 baseline risk assessment (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 re-evaluation of risks based on new contaminant data collected in September 1997.

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 previous investigations of the impoundments. 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 1997 and presents the analytical data and risk characterization based on those new data. Section 9.0 discusses the various sources of uncertainty associated with the human health risk assessment. Section 10.0 evaluates potential risks to ecological receptors that might be exposed to the surface water and sediment in the golf course impoundments. Conclusions and recommendations are provided in Section 11.0. All information sources used in this BRA are referenced in Section 12.0.

2.0 THE BASELINE RISK ASSESSMENT PROCESS

CERCLA requires that decisions regarding hazardous materials 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 materials released 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 ecological risk assessment (ERA) methodology.

For a hazardous materials 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 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 (10E-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.

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 has an area of approximately 4 acres and is up to 10 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 4 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 stormwater retention and sedimentation. Stormwater 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

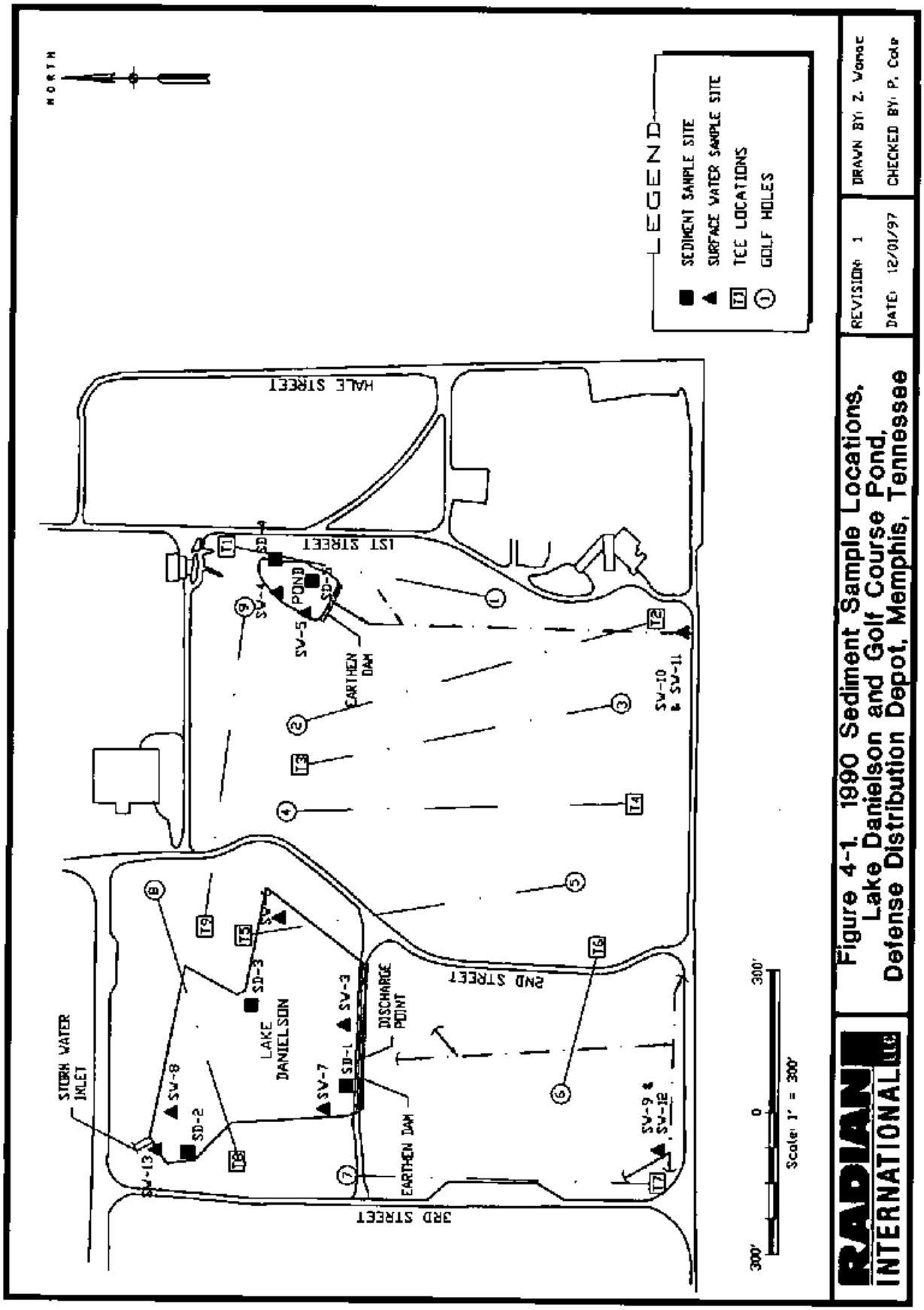
Fish tissue samples (i.e., edible portions) were collected from Lake Danielson and the golf course pond and analyzed for pesticides in 1986 by the U.S. Army Environmental Hygiene Agency (AEHA). Chlordane, DDT, DDD, and DDE were detected in both sediment and fish tissue samples.

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. The sample locations are shown in Figure 4-1.

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


NORTH
LEGEND
SEDIMENT SAMPLE SITE
SURFACE WATER SAMPLE SITE
TEE LOCATIONS
GOLF HOLES
HALE STREET
1ST STREET
2ND STREET
3RD STREET
POND
EARTHEN DAM
LAKE DANIELSON
DISCHARGE POINT
STORM WATER INLET
SD-1
SD-2
SD-3
SD-4
SD-5
SV-1
SV-2
SV-3
SV-4
SV-5
SV-6
SV-7
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RADIAN INTERNATIONAL LLC
Figure 4-1. 1990 Sediment Sample Locations, Lake Danielson and Golf Course Pond, Defense Distribution Depot, Memphis, Tennessee
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DRAWN BY: Z. Vamac
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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.

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).

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

5.0 EXPOSURE SETTING

This section describes the exposure assessment that was used both for the early 1997 BRA and for this updated BRA based on data collected in September 1997.

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. There are several schools within 1.5 miles of the Depot. Dunn Elementary, Corry Junior High, and Alcy Road Elementary are within one-half 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 after the Depot closes.

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 in this BRA to quantify human health risk involves a male youth who gains unauthorized access to swim and fish in the impoundments. He is assumed to swim in the impoundments for 1 hour each 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 is assumed that his hands and feet become covered with sediment in the process of attempting to retrieve golf balls. It is further assumed that a considerable amount of sediment becomes suspended in the water column while he swims and dives for golf balls. He is assumed to swallow a small amount of water containing suspended sediment while swimming and diving. He is assumed to be able to catch and eat catfish from the impoundments. Figure 4-1 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).

N:\65574110\03\FIG5-1.DWG

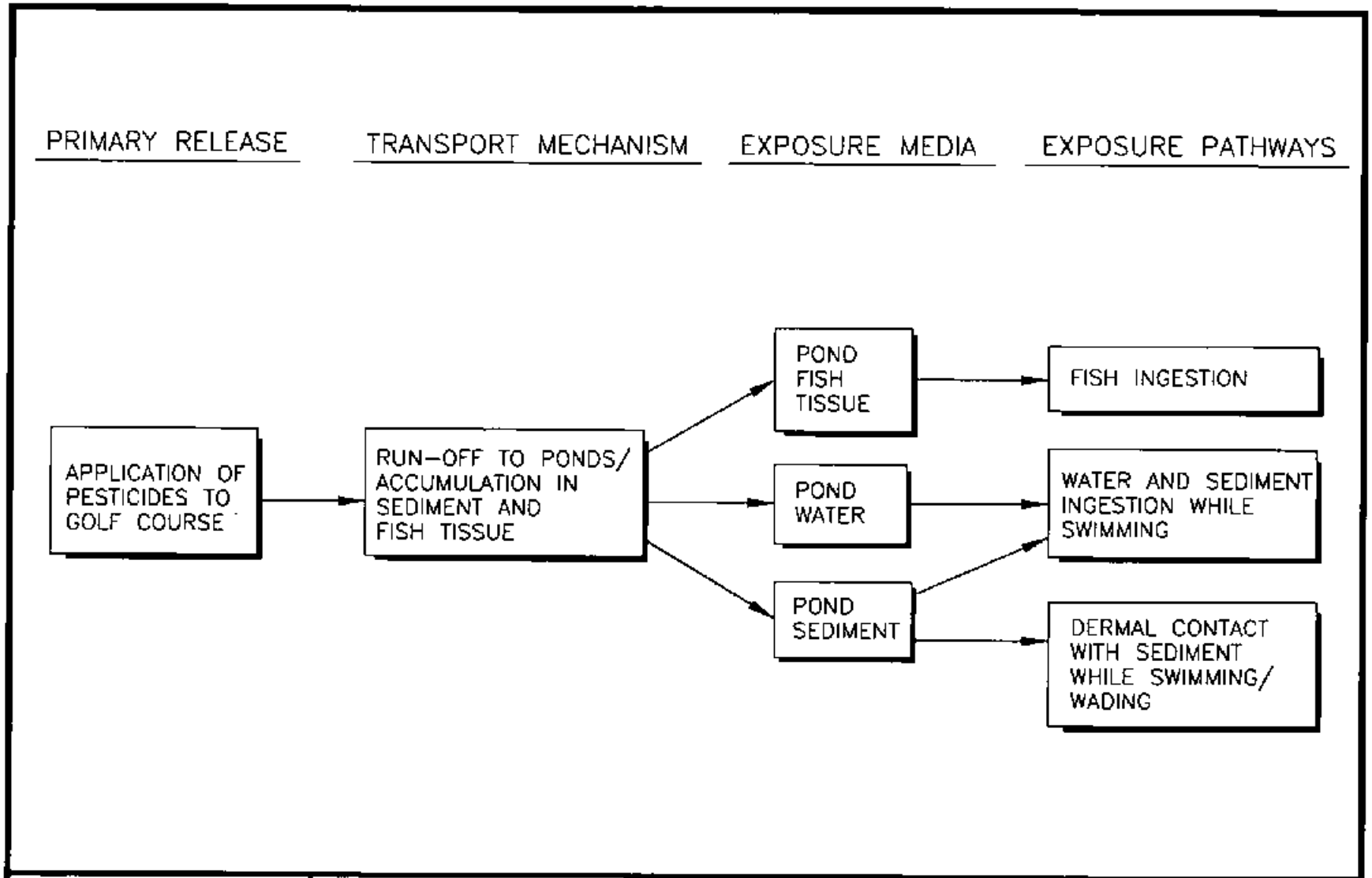

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Figure 5-1. Conceptual Site Model, Lake Danielson and Golf Course Pond, Defense Distribution Depot, Memphis, Tennessee

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DATE: 11-12-96

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CHECKED BY: P Cole

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 the representative exposure concentration for this updated BRA. EPA (1989) recommends the use of the 95% upper confidence limit on the mean of the data set (95 UCL) 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 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 is 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 1986 investigation by the AEHA 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

Absorbed Dose (mg/kg/d) = (CS x CF x SA x AF x ABS x EF x ED)/(BW x AT)

where:
- CS = chemical concentration in sediment (mg/kg)
- CF = conversion factor (10E-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)
- 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

Intake (mg/kg/d) = (CW x CR x ET x EF x ED) / (BW x 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

Intake (mg/kg/d) = (CF x IR x FI x EF x ED) / (BW x 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 individuals 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.

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 D.

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 No Observed Adverse Effects Level (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 $(mg/kg/day)^{-1}$. 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 $(mg/kg/day)^{-1}$. 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 $(mg/kg/day)^{-1}$. 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 1996).

Chlordane is a Group Be–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 F344 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 U.S. It was used extensively from the 1950's until its use was banned by the U.S. Department of Agriculture in 1970. The EPA did allow the use of dieldrin to kill termites from 1972 until 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 is 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 four 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 Be–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 database 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 the EPA as Group B2CProbable Human Carcinogen. 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. 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 A 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 time of those 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 re-evaluate the human health risks associated with exposure to pesticides in the impoundments. The recommended sampling was conducted in September and 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 Arkansas shiners (*Notropis 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

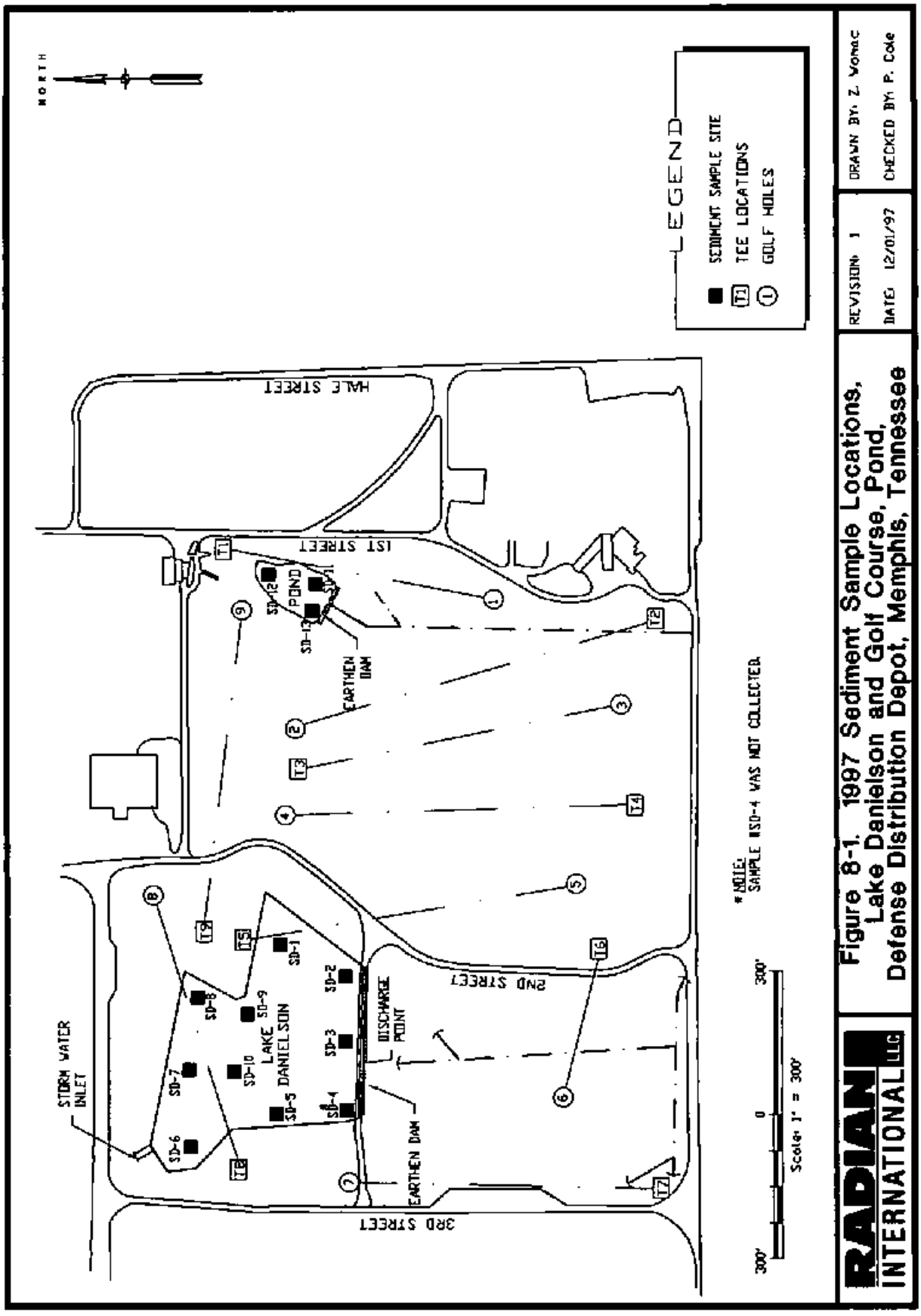
attached to the trot line near each end and in the middle to serve as floats. Lead sinkers were attached to the trot line about every 15 yards. The trot line was left in place for approximately 48 hours.

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 Arkansas 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.


NORTH
LEGEND
SEDIMENT SAMPLE SITE
T1 TEE LOCATIONS
1 GOLF HOLES
HALE STREET
1ST STREET
2ND STREET
3RD STREET
POND
SD-12
SD-13
EARTHEN DAM
SD-1
SD-2
SD-3
SD-4
SD-5
SD-6
SD-7
SD-8
SD-9
SD-10
LAKE DANIELSON
DISCHARGE POINT
STORM WATER INLET
EARTHEN DAM
*NOTE: SAMPLE NSD-4 WAS NOT COLLECTED.
300' 0 300'
Scale: 1" = 300'
RADIAN INTERNATIONAL LLC
Figure 8-1. 1997 Sediment Sample Locations, Lake Danielson and Golf Course, Pond, Defense Distribution Depot, Memphis, Tennessee
REVISION: 1
DATE: 12/01/97
DRAWN BY: Z. VONAC
CHECKED BY: P. COE
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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 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 rinseate blank was collected to evaluate the effectiveness of decontamination. The rinseate 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 rinseate 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 Arkansas 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 rinseate 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.

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 re-evaluate 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 Arkansas 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 Appendix A. 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 8-1

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	2020	883	234	2870	ND
Fish (µg/kg as received)						
1	ND	3190	490	12	732	45
2	ND	600	124	ND	166	19

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.

9.0 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 by the city of Memphis or other entity. 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 Arkansas 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.

10.0 ECOLOGICAL RISK ASSESSMENT

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. 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.

Lake Danielson has been stocked in the past with bass (*Micropterus* sp.) and bluegill sunfish (*Lepomis* sp.) and has also contained catfish (*Letalurus* sp.). The current condition of the aquatic community in the golf course impoundments is unknown.

To evaluate the ecological risk that might be associated with the pesticide residues in the impoundments' surface water and sediments, the maximum detected concentrations were compared to EPA Region 4 screening values (EPA 1997) for protection of ecological receptors. This ecological screening value comparison is the first step in the Preliminary Risk Evaluation (PRE) recommended by EPA Region 4 as the initial ecological risk screening assessment at a hazardous waste site. The last four steps of the PRE (i.e., problem formulation, ecological effects evaluation, exposure estimate, and risk calculation) are conducted only if comparisons of site analytical data with EPA Region 4 ecological screening values indicate a need for further ecological risk evaluation.

Table 10-1 compares the maximum detected concentrations of DDT, DDE, and DDD in surface water and sediment to EPA Region 4 ecological screening values for chronic exposure. None of the screening values are exceeded; therefore, no further ecological risk evaluation is needed.

Table 10-1

Comparison of Maximum Detected Pesticide Concentrations in Golf Course Impoundments Surface Water and Sediment to EPA Region 4 Ecological Screening Values

	DDT		DDE		DDD	
	Maximum Concentration	Screening Value	Maximum Concentration	Screening Value	Maximum Concentration	Screening Value
Water	—	0.001	0.21	10.5	—	0.0064
Sediment	2900	3300	110	3300	3000	3300

— = not detected

All values are in parts per billion.

11.0 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 fish is approximately 7 in one million, assuming that there are edible fish in the impoundments, that they would be caught and eaten on a regular basis, and that the 1997 analytical data on pesticides in Arkansas shiners from the ponds are representative of the muscle tissue of edible fish that might occupy the ponds in the future. This level of cancer risk is within the range of Superfund site remediation goals in the National Contingency Plan (i.e., 1E-04 to 1E-06). Human health risks associated with ingestion of sediment and dermal contact with sediment are below the range of concern.

The maximum detected concentrations of DDT, DDE, and DDD in surface water and sediment were below EPA Region 4 ecological screening values, so further investigation and remediation based on ecological risk does not appear to be warranted.

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.

12.0 REFERENCES

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

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

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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.

Appendix A

RISK ASSESSMENT SPREADSHEETS

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	4.48E-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
									Total risk:	8.72245E-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	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	16	2.00842E-06
Chlordane	0.166	0.0065	365	1	6	57.7	25550	1.6E-06	1.3	2.08373E-06
									Total risk:	6.3493E-06

Appendix B

CHAIN-OF-CUSTODY RECORD

Analysis Request Environmental Services Chain of Custody

Lancaster Laboratories
A division of Thermo Analytical Inc.

For Lancaster Laboratories use only
Acct. # _____ Sample # _____

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

For lab use only
FSC: _____
SCR #: 101590

(2) Sample Identification	Date Collected	Time Collected	(3) Grab	Composite	(4) Matrix: Soil	Water (☐ Potable ☐ NPDES)	Other	Total # of Containers	(5) Analyses Requested: Pesticides (8081)	Remarks	(6) Temperature of samples upon receipt (if required)
Sediment #1-15	10/1/97		X				X	13	X	cooled to 4°C	
fish #1+2	10/1/97		X				X	2	X	frozen	
rinsate blank	10/1/97		X			X		1	X	cooled to 4°C	

298 56

(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

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:	Date	Time

(9)

Appendix C
ANALYTICAL DATA

268 58

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

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

See reverse side for explanation of symbols and abbreviations

2216 Rev 5 01 95

Lancaster Laboratories
A division of Thermo Analytical Inc.

268 59

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. SOG#: DE001-02

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	< 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

23

Lancaster Laboratories
A division of Thermo Analytical Inc.

268 60

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.
1859	Methoxychlor	< 100.	100.	ug/kg	< 520.	520.
1987	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

Lancaster Laboratories
A division of Thermo Analytical Inc.

268 61

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
SSED- SDG#: DEDD1-04

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

Lancaster Laboratories
A division of Thermo Analytical Inc.

268 62

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. DT-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	ODE	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.
1987	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

Lancaster Laboratories
A division of Thermo Analytical Inc.

268 G3

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

31

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

Lancaster Laboratories
A division of Thermo Analytical Inc.

268 64

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.
1987	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

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Lancaster, PA 17605-2425
717-656-2300 Fax: 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

268 65

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-01220-5-06
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

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268 66

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#: OED01-09

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.
1859	Methoxychlor	< 100.	100.	ug/kg	< 350.	350.
1987	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

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268 67

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
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Lancaster, PA 17605-2425
717 656-2300 Fax: 717-656-2681

Lancaster Laboratories
A division of Thermo Analytical Inc.

268 68

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.
1859	Methoxychlor	< 100.	100.	ug/kg	< 300.	300.
1987	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.

268 69

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: D6149
Radian International LLC
PO Box 201088
Austin TX 78720-1088

P.O. DT-01220-5-06
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

43

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Lancaster PA 17605-2425
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Lancaster Laboratories
A division of Thermo Analytical Inc.

268 70

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

45

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Lancaster, PA 17605-2425
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Lancaster Laboratories
A division of Thermo Analytical Inc.

268 71

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. 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	< 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

268 72

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 SOG#: 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

Appendix D

DETAILED TOXICITY SUMMARIES

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

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_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.

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_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).

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_VI. BIBLIOGRAPHY

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

__VI.A. ORAL RfD REFERENCES

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__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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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

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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

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_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.

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_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).

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_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.

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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.

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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.

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_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

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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

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_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.

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_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).

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_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.

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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.

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_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

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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

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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.

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_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 B6C3F1, and 71 C57B1/6J 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 chlorendic 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).

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_VI. BIBLIOGRAPHY

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

__VI.A. ORAL RfD REFERENCES

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__VI.B. INHALATION RfD REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_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

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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

268 110

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

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_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

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*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.

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_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).

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_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

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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.

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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

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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

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_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

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*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.

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_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).

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_VI. BIBLIOGRAPHY

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

__VI.A. ORAL RfD REFERENCES

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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.

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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.

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_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

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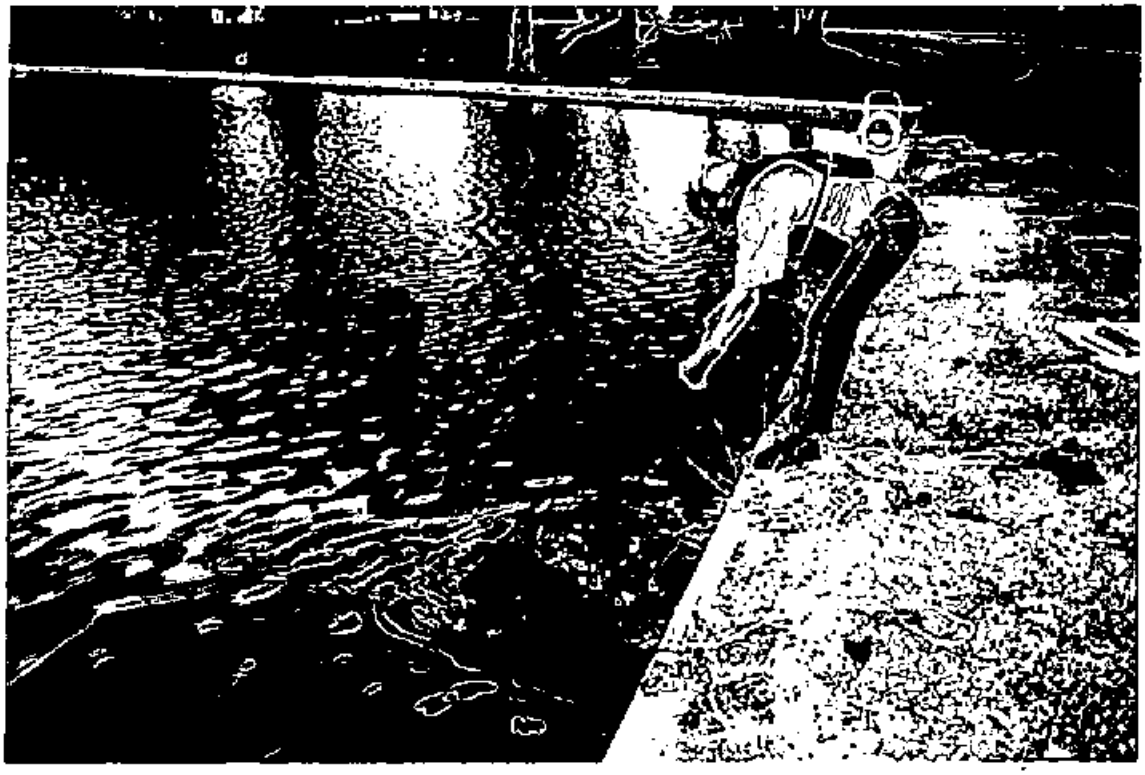
SYNONYMS

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

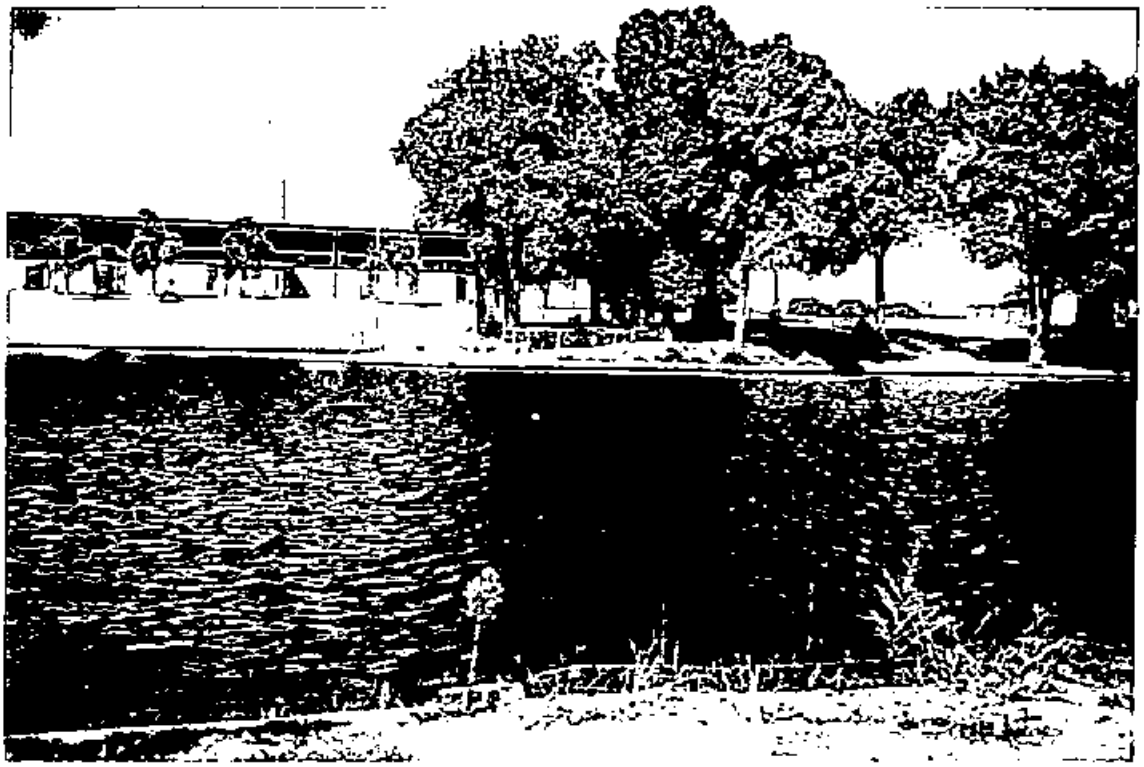
1024-57-3
ENT 25,584
EPOXYHEPTACHLOR
HCE
Heptachlor Epoxide
1,4,5,6,7,8,8-HEPTACHLORO-2,3-EPOXY-2,3,3a,4,7,7a-HEXAHYDRO-4,7-METHANOINDENE
1,4,5,6,7,8,8-HEPTACHLORO-2,3-EPOXY-3a,4,7,7a-TETRAHYDRO-4,7-METHANOINDAN
2,3,4,5,6,7,7-HEPTACHLORO-1a,1b,5,5a,6,6a-HEXAHYDRO-2,5-METHANO-2H-INDENO(1,2-b)OXIRENE
HIPTACHLOR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

Appendix E
PHOTOGRAPHS

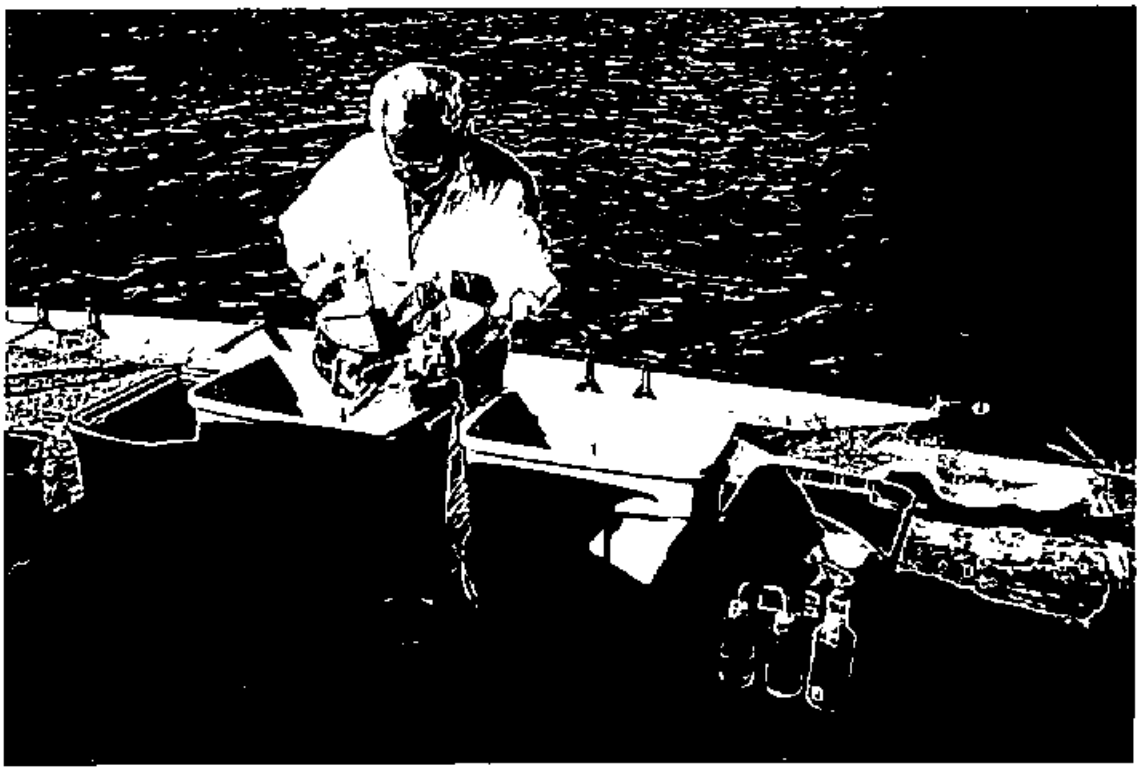


Retrieving Fish Trap from Lake Danielson

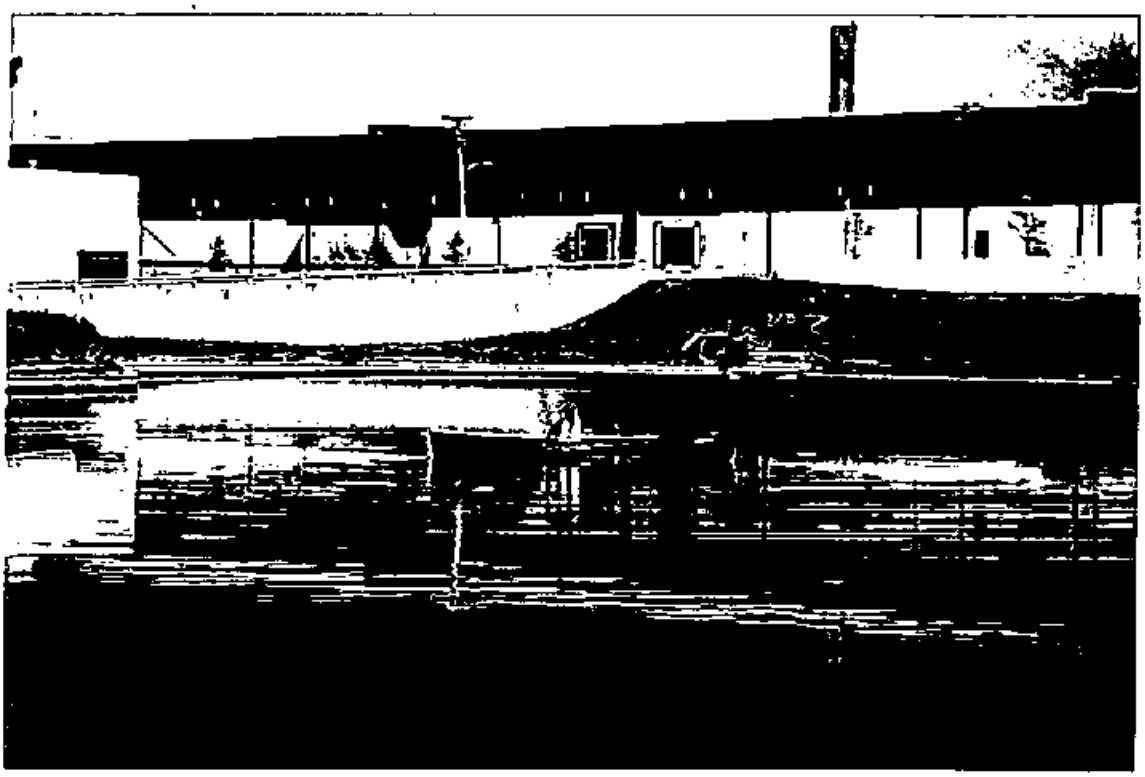
Arkansas Shiners in Fish Trap



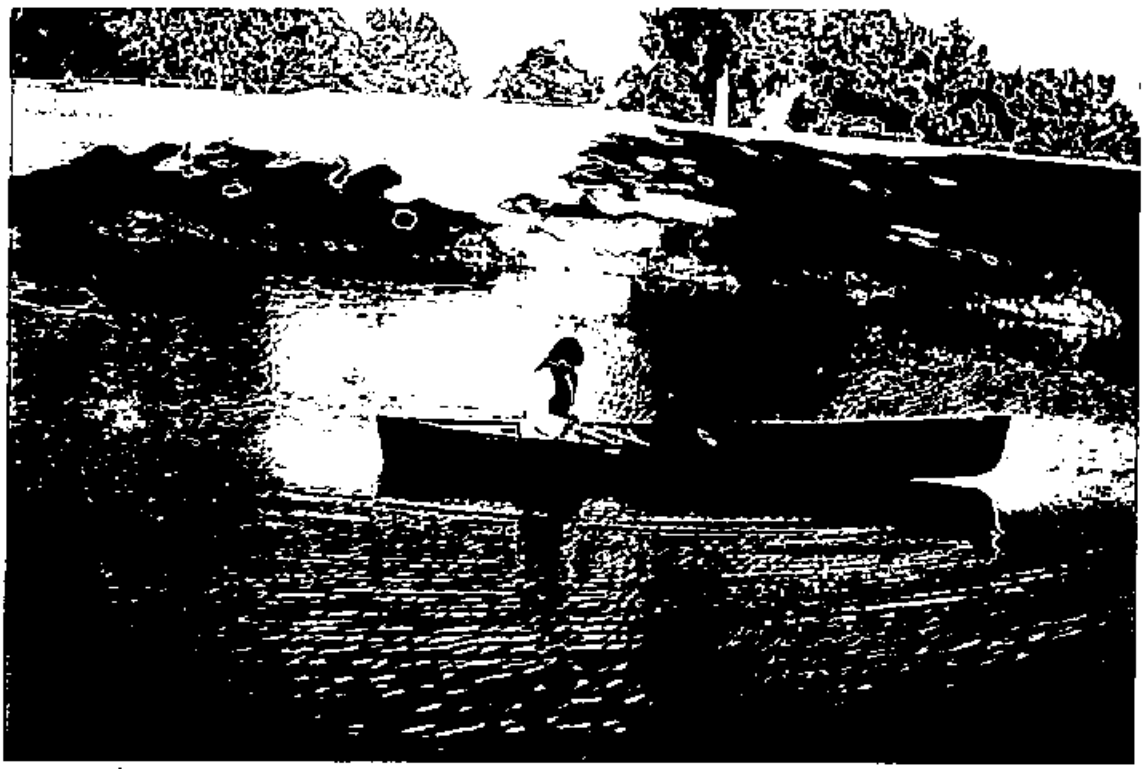
Trot Line Location, Lake Danielson



Decontaminating Sediment Sampling Equipment



Collecting Sediment From Lake Danielson



Collecting Sediment From Golf Course Pond

TAB

Sampling and Analysis Plan

SAMPLING AND ANALYSIS PLAN FOR FISH AND SEDIMENT SAMPLING AT THE DEFENSE DISTRIBUTION DEPOT, MEMPHIS, TENNESSEE

Prepared for:

U.S. Army Corps of Engineers
Mobile, Alabama

Prepared by:

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

December 1997

TABLE OF CONTENTS

		Page
LIST OF FIGURES		iii
LIST OF TABLES		iii
ACRONYMS		iv
1.0	INTRODUCTION	1-1
2.0	PREVIOUS INVESTIGATION	2-1
3.0	PROJECT ORGANIZATION AND RESPONSIBILIES	3-1
4.0	FIELD ACTIVITIES	4-1
	4.1 Fish Sampling	4-1
	4.2 Sediment Sampling	4-2
5.0	DECONTAMINATION PROCEDURES	5-1

Appendix A: CHAIN-OF-CUSTODY RECORD

LIST OF FIGURES

4-1. Proposed Sediment Sample Locations, Golf Course, Defense Distribution Depot, Memphis, Tennessee 4-3

LIST OF TABLES

4-1. Pesticides Analyzed by EPA SW-846 Method 8081 4-4

ACRONYMS

BRA	Baseline Risk Assessment
DDT	Dichlorodiphenyltichloroethane
EPA	U.S. Environmental Protection Agency
Radian	Radian International LLC
USACE	U.S. Army Corps of Engineers

1.0 INTRODUCTION

Radian International LLC (Radian) has been contracted by the U.S. Army Corps of Engineers (USACE) Mobile District to provide engineering services at the Defense Distribution Depot, Memphis, Tennessee (hereinafter referred to as the Depot). These services are being performed under USACE Delivery Contract No. DACA01-95-D-0015, Delivery Order 0041, funded by the U.S. Department of Defense.

The Depot is located in the south central section of Memphis, Tennessee. It is was closed in September 1997. A nine-hole golf course is located on the southeast corner of the Depot. It is anticipated that the golf course will continue to be used after the Depot is closed. The golf course includes two surface water impoundments: Lake Danielson and the golf course pond.

Lake Danielson is approximately 4 acres in size and approximately 8 ft deep. The golf course pond is approximately one-third acre in size and approximately 4 ft deep. Both ponds receive runoff from large areas of the Depot. Historical pesticide use at the Depot apparently led to contamination of sediments in the ponds.

Lake Danielson was periodically stocked with bluegill and bass. Catfish have also been observed in the lake.

2.0 PREVIOUS INVESTIGATION

Sediment, water, and fish tissue samples were collected from Lake Danielson and the golf course pond and analyzed for pesticides in 1986. Chlordane, dichlorodiphenyl-trichloroethane (DDT), dichlorodiphenyldichloroethane, and dichlorodiphenyldichloroethene were detected in sediment and fish samples [U.S. Army Environmental Hygiene Agency 1986]. Water in the ponds was found to be essentially uncontaminated. The use of DDT for pest control was discontinued in 1980. Fishing and swimming in the golf course impoundments have been banned since 1986.

In early 1997, Radian performed a baseline risk assessment (BRA) for the golf course impoundments to support remediation decisions for the ponds. The BRA was based on the 1986 contaminant data and the assumption that a male youth would routinely catch and eat fish from the ponds for several years. Very conservative assumptions were used in the quantification of human health risk resulting from this activity. The BRA concluded that direct exposure to water and sediment in the ponds would not result in unacceptable human health risks. However, the BRA further concluded that pesticide residues in fish tissue might pose an unacceptable risk to the health of humans ingesting the fish.

Data gaps regarding the method of fish tissue sample preparation during the 1986 sampling episode were a significant source of uncertainty for the risk assessment results. The small number of samples collected and the period of time elapsed since the samples were collected also contributed significantly to uncertainty in the BRA results. It was recommended that additional samples of sediment and fish tissue be collected and analyzed for pesticides and that the resulting data be used to re-evaluate human health risk. This Sampling and Analysis Plan describes the field activities that will be conducted to generate those data.

3.0 PROJECT ORGANIZATION AND RESPONSIBILITIES

Mr. Lloyd Hinkle will serve as the Project Manager. In this role, Mr. Hinkle will have overall responsibility, authority, and accountability for the project. He will function as the primary interface between the USACE, Radian management, and the project team. In executing these duties, he will:

- Have responsibility for meeting all contractual requirements for the task;
- Administer and supervise all contractual requirements for the task;
- Direct the formulation of work plans in accordance with client directions;
- Have responsibility for ensuring that required staffing levels and technical expertise are provided;
- Keep the USACE Technical Manager informed on all aspects of the project, including expenditures, progress, problems, and recommended solutions; and
- Review every technical project output prior to issue.

Ms. Patrice Cole will serve as the task leader for this project. In this capacity, she will be responsible for organizing and directing the technical activities of the project and for reporting the results of these activities. In execution of these duties, Ms. Cole will:

- Ensure that planned activities are executed in accordance with this and other applicable plans;
- Advise the Project Manager of technical progress, expenditure, program needs, potential problems, and recommended solutions;
- Ensure technical quality of reports, memoranda, and other communications; and
- Maintain contact with the USACE Technical Manager in areas that require decisions on technical matters.

Ms. Cole will also serve as the site health and safety officer during all field activities.

4.0 FIELD ACTIVITIES

The objective of the field work is to collect samples of fish tissue and sediment from the golf course impoundments for pesticide analysis. The analytical data will be used to re-evaluate the human health risk associated with ingesting fish from the ponds and to better characterize pesticide concentrations and distributions in the ponds' sediment. The fish sampling will also provide information on the current condition of fish populations in the ponds. This will help evaluate the degree to which the exposure assessment, which assumes that it is possible to routinely catch edible fish from the ponds, is realistic.

4.1 Fish Sampling

The number and species of fish currently in the golf course impoundments is unknown. Radian will attempt to collect at least five specimens of each edible species of fish from each pond. It is anticipated that at many as four pan fish species may reside in the ponds. These include sunfish (*Lepomis* sp.), smallmouth bass (*Micropteris dolomieui*), largemouth bass (*Micropteris salmoides*), and catfish (family Ameiruridae).

The smaller pond is approximately 4 ft deep with riprap sides. Lake Danielson is approximately 8 ft deep with vertical sides and no boat ramp. Due to the depth and configurations of Lake Danielson in particular, neither seining nor electrofishing are feasible fish collection methods for these impoundments. A portable (back pack) electroshocker cannot effectively shock to 8 ft of depth. A boat-mounted electroshocker requires a boat ramp for entry to the lake. Therefore, trotlines, hoop nets, and angling are the fish collection methods that will be used to collect any fish that might be present.

A commercial trotline will be baited, suspended across each pond at the water surface, and left undisturbed overnight. A commercial hoop net with a 3 to 4 ft opening will be baited with catfish bait, placed in Lake Danielson , and left undisturbed for at least 24 hours. A combination of cane poles, spin casters, live bait, and artificial lures will be used for angling in each pond.

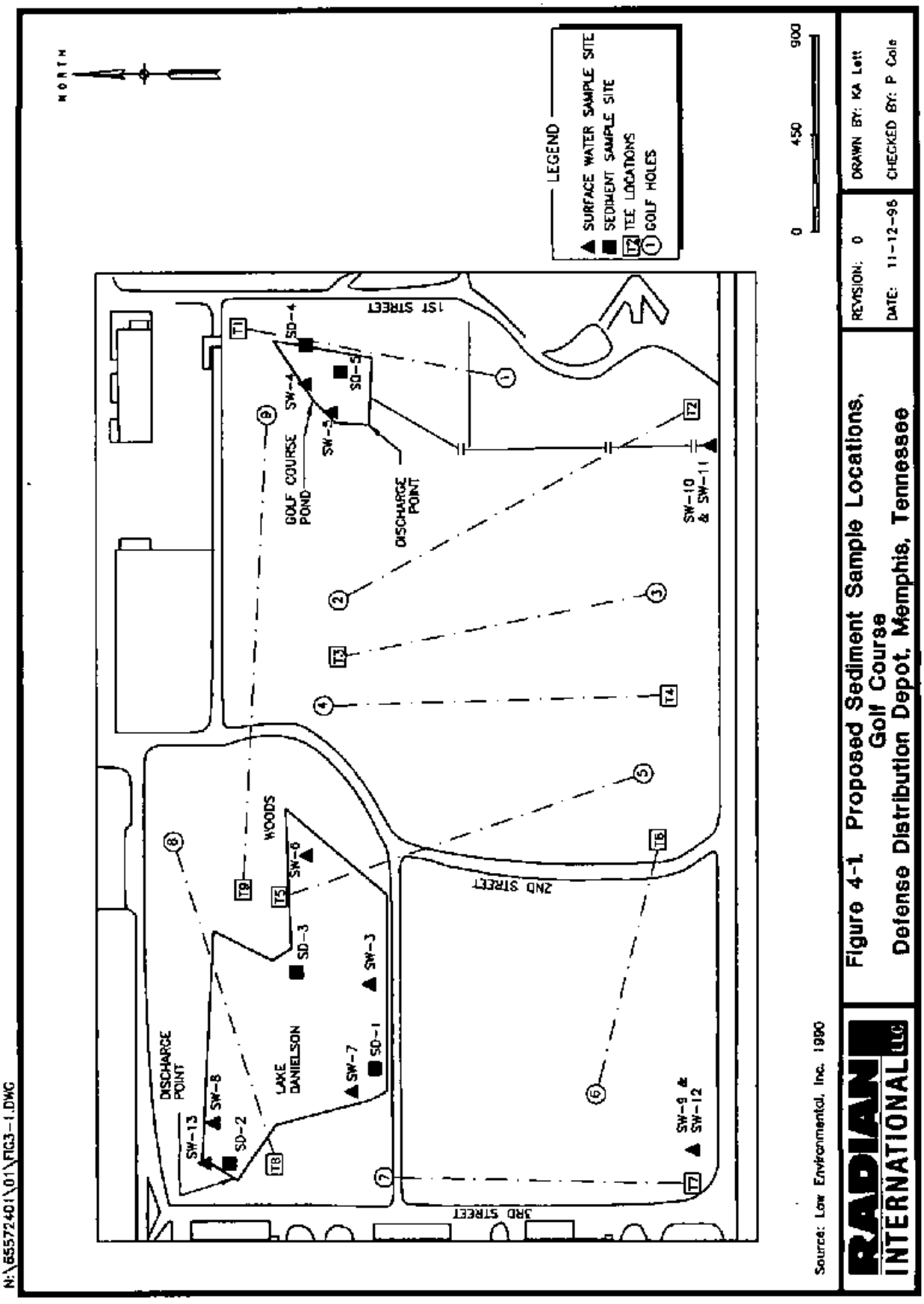
Each captured fish will be identified to species, weighed, and measured by length. A field notebook will be used to record the date, time, location, and method of capture for each fish, along with any other pertinent information regarding field conditions and handling of samples. Each whole fish belonging to one of the taxa listed above will be wrapped in aluminum foil, sealed in a plastic bag with tamper-resistant custody tape, labeled, and placed into an airtight, insulated container with dry ice. Fish samples will be numbered sequentially as they are collected, beginning with F-1. The samples will be shipped overnight to the analytical laboratory, which will be directed to filet the fish and analyze the filets, with skin, by U. S. Environmental Protection Agency (EPA) SW-846 Method 8081. Field personnel will maintain custody of all samples until shipment. Chain-of-custody records will be maintained to document that samples were not tampered with from the time they were collected until they were received by the analytical laboratory. A sample chain-of-custody form is included in Appendix A.

If more than five individuals of each taxon are collected, the five largest fish of each taxon will be sent to the laboratory for analysis. The remaining fish will be returned to the ponds. If no fish are collected from either pond within 2 to 3 days, alternative means of determining the presence or absence of fish in the ponds will be considered.

4.2 Sediment Sampling

Sediment sampling will be conducted from a small boat. A clamshell dredge, specifically a Wildco Petite Ponar, will be attached to a sturdy rope and lowered by hand from the boat to obtain a sample of the upper 6 in. of sediment in the bottom of each pond. Ten samples will be collected from Lake Danielson, and three samples will be collected from the smaller pond. The approximate sample locations are shown in Figure 4-1.

Each sediment sample collected by the dredge will be transferred to a clean stainless steel bowl, thoroughly mixed with a clean stainless steel spoon, and packed into a glass jar supplied by the analytical laboratory. The samples will be labeled, sealed with tamper-resistant custody tape, and placed into a cooler with ice immediately upon being collected. The samples will be numbered sequentially as they are collected, beginning with S-1. The samples


NORTH
LEGEND
SURFACE WATER SAMPLE SITE
SEDIMENT SAMPLE SITE
TEE LOCATIONS
GOLF HOLES
0
450
900
1ST STREET
2ND STREET
3RD STREET
GOLF COURSE POND
DISCHARGE POINT
WOODS
LAKE DANIELSON
DISCHARGE POINT
SW-4
SW-5
SD-4
SD-5
SW-10 & SW-11
SW-6
SD-3
SW-3
SW-7
SD-1
SW-13
SD-2
SW-8
SW-9 & SW-12
T1
T2
T3
T4
T5
T6
T7
T8
T9
N:\65572401\01\FIG3-1.DWG
Source: Law Environmental, Inc. 1990
RADIAN INTERNATIONAL LLC
Figure 4-1. Proposed Sediment Sample Locations, Golf Course
Defense Distribution Depot, Memphis, Tennessee
REVISION: 0
DATE: 11-12-96
DRAWN BY: KA Lett
CHECKED BY: P Cole

will be shipped overnight to the analytical laboratory for pesticide analysis by EPA SW-846 Method 8081. The pesticides analyzed by EPA SW-846 Method 8081 are listed in Table 4-1. At one sample location, enough material will be collected for a blind duplicate sample to assess the analytical laboratory's reproducibility of results. A field notebook will be used to document the date, time, location, description, and handling of samples. Field personnel will maintain custody of all samples until shipment. Chain-of-custody records will be maintained to document that samples were not tampered with from the time they were collected until the time they were received by the analytical laboratory.

Table 4-1

Pesticides Analyzed by EPA SW-846 Method 8081

Aldrin	4,4′ -DDD	Methoxychlor
alpha-BHC	Dieldrin	Aroclor 1016
beta-BHC	Endosulfan I	Aroclor 1221
gamma-BHC (Lindane)	Endosulfan II	Aroclor 1232
delta-BHC	Endosulfan sulfate	Aroclor 1242
Chlordane	Endrin	Aroclor 1248
alpha-Chlordane	Endrine aldehyde	Aroclor 1254
gamma-Chlordane	Endrin ketone	Aroclor 1260
4,4′ -DDT	Heptachlor	Toxaphene
4,4′ -DDE	Heptachlor epoxide	

5.0 DECONTAMINATION PROCEDURES

A decontamination station will be required on-site to decontaminate all sampling equipment that comes into contact with sediment. The dredge, stainless steel bowl, and stainless steel spoon will be decontaminated prior to collecting each sample and after collecting the last sample. The hoop net, which will lie in contact with bottom sediments during fish sampling, will also be decontaminated after use. The decontamination station will be constructed so that all decontamination fluids and removed sediment will be retained inside the decontamination area. The procedure for all field decontamination of sampling equipment is as follows:

1. Wash equipment with a brush and a phosphate-free detergent solution.
2. Rinse with tap water.
3. Rinse with pesticide-grade isopropanol.
4. Rinse thoroughly with organic-free water.
5. Unless the equipment is going to be used immediately, it will be wrapped in aluminum foil.

Field personnel will wear latex gloves while handling sediment sampling equipment and fish and sediment samples. Additionally, field personnel will wear water-resistant paper suits and safety glasses while collecting and handling sediment samples. Personnel decontamination will consist of doffing gloves and paper suits, turning them inside out in the process, and disposing of them in a plastic trash bag. A clean pair of gloves will be donned before collecting each sediment sample and before handling each captured fish to avoid cross-contamination of samples.

A rinsate (equipment) blank will be collected and submitted to the analytical laboratory for pesticide analysis by EPA SW-846 Method 8081 to assess cross-contamination from the sampling equipment. The rinsate blank will consist of organic-free water poured over an item of decontaminated sediment sampling equipment and transferred to a glass sample

container. The sample will be labeled, sealed with custody tape, and immediately placed into the sample cooler with ice to ship to the analytical laboratory along with the sediment samples.

Decontamination fluids will be transferred to 55-gal drums already in place at Dunn Field for storage. Used personal protective equipment and sanitary trash will be transferred in suitable containers to Depot personnel for disposal.

Appendix A

CHAIN-OF-CUSTODY RECORD

CHAIN-OF-CUSTODY RECORD

PROJECT NAME:

PROJECT NUMBER:

SAMPLERS (SIGNATURE)

PRINTED NAME

CLIENT ID (9 CHARACTERS)									#	GC/MS					GC							INORGANICS				OTHER											SAMPLING/INFO		REMARKS
1	2	3	4	5	6	7	8	9	No. of Bottles/Vials:	624-8240	625-8270	CLP-VOA	CLP-SVOA	Other:	601-8010	602-8020	608-8080	8140	CLP Pest/PCBs	Herbicides	Other:	Metals	Cyanide	CLP Metals	Other:	TOC	TOX	Oil & Grease	Pet. Hydro.	Phenols	Neutron Activation					MATRIX: Water/Soil/Sediment	DATE	TIME	

RELINQUISHED BY:	Date/Time	RELINQUISHED BY:	Date/Time	RELINQUISHED BY:	Date/Time	SHIPPING INFORMATION
COMPANY NAME:		COMPANY NAME:		COMPANY NAME:		Number of Shipping Containers:
RECEIVED BY:	Date/Time	RECEIVED BY:	Date/Time	RECEIVED BY:	Date/Time	Method of Shipment
COMPANY NAME:		COMPANY NAME:		COMPANY NAME:		
RELINQUISHED BY:	Date/Time	RELINQUISHED BY:	Date/Time	RELINQUISHED BY:	Date/Time	Special Handling Requirements
COMPANY NAME:		COMPANY NAME:		COMPANY NAME:		

TAB

Safety and Health Plan

SAFETY AND HEALTH PLAN FOR FISH AND SEDIMENT SAMPLING AT THE DEFENSE DISTRIBUTION DEPOT, MEMPHIS. TENNESSEE

Prepared for:

U.S. Army Corps of Engineers
Mobile, Alabama

Prepared by:

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

December 1997

TABLE OF CONTENTS

		Page
LIST OF FIGURES		iv
LIST OF TABLES		iv
ACRONYMS		v
1.0	HEALTH AND SAFETY	1-1
1.1	Project Objectives	1-1
1.2	Site-Specific Safety and Health Plan Objectives	1-1
1.3	Radian Safety and Health Policy	1-1
2.0	REVIEW OF POTENTIAL HAZARDS	2-1
2.1	Site Hazard Assessment/Prevention	2-1
2.1.1	Chemical Hazards	2-1
2.1.2	Physical Hazards	2-1
2.1.3	Biological Hazards	2-3
2.1.4	Weather Conditions and Heat Stress	2-3
2.2	Field Tasks to be Performed and Hazard Prevention	2-4
3.0	KEY PERSONNEL AND RESPONSIBILITIES	3-1
3.1	Program Manager	3-1
3.2	Task Leader	3-1
3.3	Site Health and Safety Officer	3-2
4.0	TRAINING REQUIREMENTS	4-1
4.1	General Site Workers Training	4-1
4.2	Supervisors Training	4-1
4.3	Refresher Training	4-1
4.4	Documentation of Training	4-2
5.0	MEDICAL SURVEILLANCE AND EXPOSURE MONITORING	5-1
5.1	Applicability and Scope	5-2
5.2	Criteria for Medical Monitoring	5-2
5.2.1	Potential for Exposure to Chemical and Physical Hazards	5-2
5.2.2	Physical Agents	5-3
6.0	HEALTH AND SAFETY EQUIPMENT	6-1
6.1	Site-Specific Levels of Protection	6-1
6.2	Site-Specific Personal Protective Equipment	6-1
6.3	Decontamination Procedures	6-2

TABLE OF CONTENTS (Continued)

		Page
7.0	STANDARD OPERATING PROCEDURES	7-1
7.1	Site-Specific Work Practices	7-1
7.2	General Site Operating Procedures/Safety Guidelines	7-1
7.3	Drug Free Workplace Policy	7-2
8.0	EMERGENCY MEDICAL TREATMENT	8-1
9.0	ACCIDENT REPORTING PLAN	9-1
9.1	Applicability and Scope	9-1
9.1.1	Injuries and Illnesses	9-1
9.1.2	Accidents	9-1
9.1.3	Near Misses	9-2
9.2	Responsibilities	9-2
9.2.1	Corporate Director of Health and Safety	9-2
9.2.2	EAC	9-2
9.2.3	Technical Resource Manager	9-3
9.2.4	Project Manager	9-3
9.2.5	All Radian Employees	9-3
9.3	Reporting Procedures and Practices	9-3
9.3.1	Injuries and Illnesses	9-3
9.3.2	Accidents	9-6
9.3.3	Near Misses	9-7
9.3.4	Training	9-7
10.0	RECORDKEEPING REQUIREMENTS	10-1
11.0	APPROVAL BY CERTIFIED INDUSTRIAL HYGIENIST	11-1

Appendix A: MATERIAL SAFETY DATA SHEETS
Appendix B: ACCIDENT REPORT FORMS
Appendix C: MEDICAL SURVEILLANCE, HAZWOPER TRAINING, AND FIRST AID/CPR TRAINING DOCUMENTATION

LIST OF FIGURES

1-1. Radian Occupational Safety and Health Policy 1-2
2-1. Site Layout of the Defense Distribution Depot, Memphis, Tennessee 2-2
10-1. Sign-Off Sheet 10-2

LIST OF TABLES

2-1. Hazard Analysis List 2-5

ACRONYMS

AOE	Arising Out of Employment
CDHS	Corporate Director of Health and Safety
CFR	Code of Federal Regulations
COE	Course of Employment
CPR	Cardiopulmonary Resuscitation
DDD	Dichlorodiphenyldichloroethane
DDE	Dichlorodiphenyldichloroethene
DDT	Dichlorodiphneyltrichloroethane
EAC	Environmental Affairs Coordinator
HAZWOPER	Hazardous Waste Operations and Emergency Response
HRA	Human Resource Administrator
HSO	Health and Safety Officer
OSHA	Occupational Safety and Health Administration
PPE	Personal Protective Equipment
QA	Quality Assurance
Radian	Radian International LLC
SSHP	Site-Specific Safety and Health Plan
TLV	Threshold Limit Value
USACE	U.S. Army Corps of Engineers

1.0 HEALTH AND SAFETY

1.1 Project Objectives

The objective of this project is to conduct fish tissue and sediment sampling at the Defense Distribution Depot, Memphis, Tennessee (hereinafter referred to as the Depot) golf course pond. The samples will be analyzed for pesticide contamination, and the results will be used to determine whether remediation of contaminated sediment and/or fish is required to protect public health.

To ensure the health and safety of project personnel during this effort, this Site-Specific Safety and Health Plan (SSHP) was prepared in accordance with Occupational Safety and Health Administration (OSHA) requirements, U.S. Environmental Protection Agency hazardous waste requirements, and the U.S. Army Corps of Engineers (USACE) Safety and Health Requirements Manual (EM 385-1-1).

1.2 Site-Specific Safety and Health Plan Objectives

This SSHP contains safety and health guidelines to be followed by Radian International LLC (Radian) during field activities performed at the Depot golf course pond. Field activities will not be performed until the SSHP is reviewed and accepted by the Contracting Officer for USACE Mobile District. This plan identifies persons responsible for administering the plan and their specific duties, training and medical monitoring, health and safety equipment, and standard operating procedures.

1.3 Radian Safety and Health Policy

Figure 1-1 is the Radian Occupational Safety and Health Policy.

April 1, 1995

Radian believes that safety and property loss prevention are equal in importance to product quality, client responsiveness, and cost control.

The fundamental responsibilities of management in this area are to prevent injury and property loss through the identification and elimination of potential hazards. The ultimate responsibility for safety rests with management. Therefore, it is necessary that:

- all employees be encouraged through training, leadership, and example to appreciate the need for safety awareness on and off the job;
- equipment and processes in our facilities be properly designed and maintained;
- all supervisors accept responsibility for the enforcement of safety procedures; and
- all employees accept their responsibility to work safely and extend this concern to their fellow employees.

Furthermore, Radian will comply with the Williams-Steiger Occupational Safety and Health Act, Resource Conservation and Recovery Act, and all federal, state, and local regulations involved in promoting safety and health in the workplace and the environment.

Donald M. Carlton
President
Radian Corporation

Figure 1-1. Radian Occupational Safety and Health Policy

2.0 REVIEW OF POTENTIAL HAZARDS

The golf course pond are located in the southeast corner of the Depot, as shown in Figure 2-1. Angling equipment, hoop nets, and trot lines will be used to collect fish from the ponds, and a hand-held clam shell dredge will be lowered from a small boat to collect sediment samples from the bottom of the ponds.

2.1 Site Hazard Assessment/Prevention

The evaluation of hazards is based on knowledge of the site background and anticipated risks posed by specific field activities. This section outlines the chemical and physical hazards that may be encountered while conducting field activities.

2.1.1 Chemical Hazards

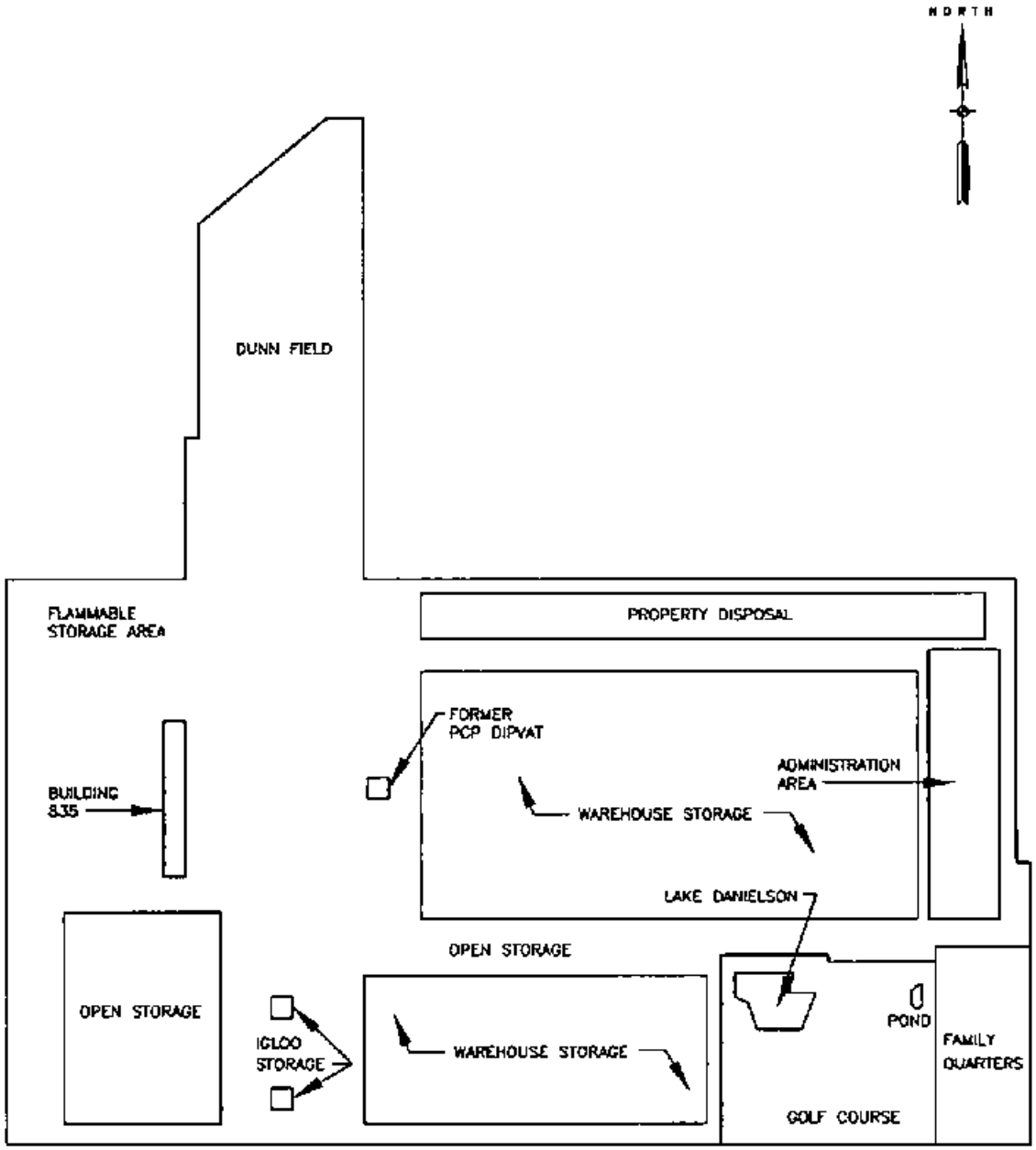
Dichlorodiphenyltrichloroethane (DDT), dichlorodiphenyldichloroethane (DDD), and dichlorodiphenyldichloroethene (DDE) are the contaminants of interest in the golf course pond. It is not anticipated that elevated levels of these or other chemicals will be detected while collecting samples. However, Radian personnel will wear latex gloves and chemical-resistant paper coveralls while handling sediment samples and fish.

A small amount of isopropanol for use in equipment decontamination will be brought to the site by the sampling team. A Material Safety Data Sheet for isopropanol is provided in Appendix A.

2.1.2 Physical Hazards

Physical hazards encountered while sampling at the site are primarily associated with the sampling equipment and working near a water body. The smaller pond is 4 ft deep, and Lake Danielson is 8 ft deep. Fish and sediment sampling will be conducted from a small, two-person boat with a trolling motor.

N:\65574110\03\SITE.DWG



RADIAN INTERNATIONAL LLC

Source: Law Environmental, Inc. 1990

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

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

Fish hooks may cause puncture wounds, and drowning could result from falling into the ponds from the sides of the ponds or from a boat. Radian personnel will exercise caution while handling fish hooks and will wear U.S. Coast Guard-approved personal floatation devices provided by Radian while working in a boat. Additionally, sampling personnel will be instructed to not stand in the boat, and they will be capable of swimming.

2.1.3 Biological Hazards

On-site workers must be aware of several potential natural hazards. Poisonous plants such as poison ivy, poison oak, and sumac are unlikely to be encountered on the golf course due to standard maintenance practices. However, stinging insects might be present. Insect stings from bees, wasps, and hornets can cause mild irritation to severe allergic reactions, depending on the kind of insect, number of stings, and reaction of the victim. Stings should immediately be treated with the first aid kit maintained on-site. If the victim indicates that he or she is allergic, or shows signs of allergic reaction, transport the victim to the nearest hospital emergency room for treatment. Workers who have known allergies to insect stings shall be identified before work starts.

Poisonous snakes in West Tennessee include the water Micatin, copperhead, and eastern diamondback rattlesnake. These snakes are classed as pit vipers and inject neurotoxins by biting. Adults in good health can die from the bites of these snakes but usually suffer illness, severe pain, and tissue necrosis. If someone is bitten, keep the victim calm and immobilize the affected limb. Administer first aid and transport the victim immediately to a hospital emergency room for treatment. It is important to identify the kind of snake, if this can be done without danger, so that proper treatment can be administered.

2.1.4 Weather Conditions and Heat Stress

Weather conditions will be monitored by the task leader. Any thunderstorms and/or high winds in proximity of the site will warrant shut down of all sample collection activities.

Heat stress is the aggregate of environmental and physical work factors that constitute the total heat load imposed on the body. The environmental factors of heat stress are air temperature, radiant heat exchange, air movement, and humidity. Physical work and personal protective equipment (PPE) worn by employees will add to the total heat load imposed on the body. To minimize heat stress, rest periods will be given to employees when temperatures exceed 85°F. This is particularly important for unacclimated workers. A 10-minute rest period each hour is recommended for unacclimated workers (i.e., workers who have not been working in high temperature conditions). Light-colored clothing, sunglasses, sunscreen, and hats will be used if weather conditions call for them.

Field activities will be temporarily discontinued in the event of high winds, heavy rain, or lightening in the sampling area. Field activities will resume after the threat of inclement weather has passed.

2.2 Field Tasks to be Performed and Hazard Prevention

Upon final approval of the required work plans, the field crew will be mobilized to the site to begin collecting necessary samples. Samples will be collected as outlined in the Sampling and Analysis Plan. Table 2-1 describes specific potential hazards and preventative measures that will be followed while conducting the sampling.

When angling and setting trot lines, field team members will use caution to avoid puncture wounds from fish hooks. When fishing or collecting sediment samples from the bank, caution will be exercised in ensuring sure footing to avoid falling into the water. Personal floatation devices will be worn at all times when in a boat. Caution will be exercised when in a boat (e.g., no standing or sudden movements) to avoid capsizing the boat. Outdoor work will be discontinued in the event of inclement weather.

Table 2-1

Hazard Analysis List

Potential Hazards	Recommended Controls
Trips, slips, and falls from uneven surfaces and heavy vegetation	Be alert and observe terrain while walking to minimize slips and falls. Remove trip hazards from walkways and be aware of wet surfaces
Allergic reaction to poisonous plants	Wear long-sleeved clothing and pants to minimize contact with irritant plants and protect against insect bites
Native wildlife such as snakes, ticks, insects, and rodents	Avoid wildlife when possible. In the case of an animal bite, administer first aid. Check for ticks when leaving wooded or vegetated areas. Determine whether staff members are allergic to bee stings and, if so, have medication available
Back strain from carrying instruments	Use proper lifting techniques; distribute heavy loads between two people
Accidents from driving vehicles on uneven or unsafe surfaces (overturned vehicles or flat tires)	Ensure maintenance has been performed on vehicles. A site surveillance on foot might be required to choose a clear driving path
	Wear seat belts
Heat stress from extreme weather conditions	Implement heat stress management techniques such as frequent breaks, monitoring fluid intake, and monitoring employees
Puncture wounds from fish hooks	Exercise caution when handling fish hooks
Drowning	Wear approved floatation device while working in the boat. Do not stand in boat. Discontinue field activities during inclement weather
Lightening strike	Discontinue field activities during inclement weather
Contact with pesticide-contaminated sediment	Wear water-resistant, chemical-resistant gloves and paper coveralls during sample collection and handling

3.0 KEY PERSONNEL AND RESPONSIBILITIES

3.1 Program Manager

The Program Manager for this task will be Mr. Lloyd Hinkle, P.E. He is responsible for the health and safety of all members of the project team. To carry out that responsibility, the program manager will ensure that all team members follow the health and safety guidelines provided in the *Radian International LLC Health and Safety Manual* (March 1996). He will ensure that project members are familiar with appropriate plans required to execute the field efforts and that these plans are in place and understood by all participants. He will ensure that required levels of training are provided to members of the team and that this training is up-to-date.

Mr. Hinkle will also ensure that health and safety is a high priority in planning field work, that appropriately trained project staff are selected, and that adequate resources are available to develop and implement this SSHP. He will ensure that the plan is reviewed/approved by an Environmental Affairs Coordinator (EAC). It is the responsibility of the task leader to respond to an unsafe condition reported by the project staff and to work with the staff to mitigate unsafe conditions.

3.2 Task Leader

Ms. Patrice Cole is the task leader for the project and, as such, will have responsibility for day-to-day management of the project, to include health and safety oversight. She will be responsible for performing a detailed hazard analysis of the work to be performed and ensuring that site-specific health and safety training is provided to team members prior to mobilization to the site. Ms. Cole has 8 hours of Hazardous Waste Operations and Emergency Response (HAZWOPER) supervisory training, and she will conduct site-specific health and safety training before field activities begin to ensure that each member is thoroughly familiar with and has signed this SSHP and other pertinent work plans (see Section 10.0). Ms. Cole will be responsible for monitoring compliance of this SSHP during project execution and reporting up

through the project manager, who has overall accountability. Ms. Cole will also coordinate activities with Radian personnel and subcontractors at the site to ensure safe completion of the project.

The task leader is responsible for managing the execution of each specific task. The responsibilities of the task leader are to:

- Ensure that activities planned are executed in accordance with this plan;
- Ensure that technical personnel are qualified by experience or training to perform assigned work and comply with the technical and quality assurance (QA) requirements applicable to the work being performed; and
- Ensure that proper PPE is available and used.

The task leader will also act as the QA officer. The QA officer is responsible for:

- Providing QA guidelines and directions to field personnel;
- Serving as the focal point for QA activities and ensuring that activities are conducted in accordance with the work plan objectives; and
- Reviewing, as appropriate, project documentation.

3.3 Site Health and Safety Officer

Ms. Cole will act as the Health and Safety Officer (HSO). Ms. Cole will be responsible for implementing field surveillance activities necessary to ensure that worker health and safety concerns are fully addressed, including adhering to the SSHP requirements. She will provide site-specific training to employees assigned to work at the site and enforce the requirements stated in the *Radian International LLC Health and Safety Manual* and this SSHP.

As the HSO, Ms. Cole has the authority to order the immediate evacuation of personnel from any area of the site that may be determined unsafe, require personnel to obtain immediate medical attention if warranted, and provide health and safety briefings to visitors; however, any member of the project team that identifies an unsafe act or situation has the authority to stop work.

4.0 TRAINING REQUIREMENTS

Personnel working at any hazardous waste site must recognize and understand the potential safety and health risks associated with work at that site. Workers involved in site activities must be thoroughly familiar with programs contained or referenced in this SSHP. Training requirements for personnel involved in hazardous waste operations will comply with 29 Code of Federal Regulations (CFR) 1910.120 (OSHA) regulations for HAZWOPER. Refer to Appendix B for individual employee training and medical certification dates.

4.1 General Site Workers Training

Site workers who are engaged in hazardous substance removal or other activities that expose or potentially expose them to hazardous substances will receive 40 hours of hazardous waste site training and 3 days of on-the-job training as described in the OSHA 29 CFR 1910.120 (HAZWOPER) standard.

4.2 Supervisors Training

On-site supervisors, such as field sampling team leaders, will receive the same 40-hour HAZWOPER training as the general site workers they supervise. Additionally, they will receive 8 hours of specialized training as described in the HAZWOPER standard.

4.3 Refresher Training

General site workers and supervisors will receive 8 hours of refresher training annually. The refresher training will include topics similar to those presented during the 40-hour course.

During the investigation and near its conclusion, the Radian team will review current conditions at the site to determine whether additional safety procedures and/or equipment are warranted.

4.4 Documentation of Training

Training activity must be documented. Accepted documentation includes a course certificate or a letter/memorandum signed by the trainer and subject to approval by a Radian EAC. Copies of the documentation will be forwarded to Radian's Corporate Director of Health and Safety (CDHS) in Austin, Texas. Site-specific training will be documented with a sign-up sheet and topics discussed. Formal training records will be maintained by the local EAC, the training recordkeeper in Radian's Austin office, and the CDHS for all company employees. Site-specific training records will be maintained in the project files. Appendix C contains documentation of training received by field team members.

5.0 MEDICAL SURVEILLANCE AND EXPOSURE MONITORING

Prior to mobilization to the site, personnel performing surveys and/or investigations are required to participate in the medical surveillance program as required by 29 CFR 1910.120.

Medical exams will be conducted by a licensed physician who is certified in occupational medicine or who, by necessary training and experience, is considered board-eligible by the American Board of Preventive Medicine Incorporated. The physical should categorize the individuals as fit for the specific tasks to be assigned and able to wear respiratory equipment if deemed necessary. Medical monitoring documentation for the site team members is provided in Appendix B.

Radian has established a medical monitoring program for employees engaged in potentially hazardous activities as described in the *Radian International LLC Health and Safety Manual*. The medical monitoring program provides for regular physical exams for employees in certain job profiles, assessment of his or her medical status over the course of his or her employment at Radian, as well as exams or consultations in the event of an exposure or suspected exposure.

Radian will ensure that this program is based on current occupational medicine practices and that it complies with applicable government regulations by:

- Contracting physicians competent in occupational medicine;
- Monitoring of program compliance on an ongoing basis by the CDHS, the Administrator of Health Services, and the local EAC;
- Performing periodic evaluation of the program by a Radian management team; and
- Modifying/updating the program as necessary.

5.1 Applicability and Scope

Medical monitoring is conducted on those employees whose work has the potential to expose them to chemicals or agents at work sites. Employees involved in work at hazardous waste sites will comply with the medical monitoring requirements of the OSHA 1910.120 standard. Candidates for medical monitoring will be selected based on the potential for chemical exposure, environmental conditions, physical requirements, regulatory requirements, and the potential use of PPE.

The basic concepts used to develop this program are based on the following OSHA regulations:

- Access to Employee and Medical Records (29 CFR 1910.20);
- HAZWOPER (29 CFR 1910.120);
- Asbestos (29 CFR 1910.1001);
- Respiratory Protection (29 CFR 1910.134);
- Occupational Noise Exposure (29 CFR 1910.95); and
- Occupational Exposure to Hazardous Chemicals in Laboratories (29 CFR 1910.1450).

5.2 Criteria for Medical Monitoring

In general, there are two criteria that determine whether an employee should be enrolled in the medical monitoring program: potential for exposure to hazards and job profile.

5.2.1 Potential for Exposure to Chemical and Physical Hazards

OSHA-Regulated Material—Employees who work with or around the OSHA-regulated materials listed in 29 CFR 1910.1000 at or above the indicated action levels, will be entered into the medical monitoring program and will receive annual exams.

Unplanned Exposure to Hazardous Substances—Employees who are suspected of having been exposed to concentrations of hazardous substances above permissible exposure limits or threshold limit values (TLVs) will be included in the medical monitoring program. Title 29 CFR 1910.1000 and the American Conference of Governmental Industrial Hygienists TLVs pamphlet can be consulted for specific exposure limits.

5.2.2 Physical Agents

Exposure to the following physical agents or hazards requires enrollment in the medical monitoring program.

Noise Levels—Employees whose exposure to noise equals or exceeds an 8-hour time-weighted average of 85 dBA for greater than 30 days/year will be included in the medical monitoring program and will receive annual audiometric testing and training as required by OSHA 29 CFR 1910.95.

Job Profiles—Employees engaged in work at hazardous waste sites, who have the potential to be exposed to chemicals above regulatory or guidance levels, or who use respirators in their work will be enrolled in the Radian medical monitoring program.

6.0 HEALTH AND SAFETY EQUIPMENT

This section describes the PPE to be used during sample collection. OSHA defines protection levels ranging from A to D; for this project, only modified Level D is discussed as this is the site-specific level that may be used during this effort.

6.1 Site-Specific Levels of Protection

Employees will be supplied with and wear modified Level D protective equipment; however, the level of protection provided by PPE may be upgraded or downgraded based upon a change in site conditions. The task leader will determine whether a change in PPE level is warranted or additional safety procedure changes are needed. No conditions are anticipated that would require an upgrade of PPE beyond Level D.

The following constitute modified Level D protective equipment:

- Work clothes/coveralls;
- Safety glasses with side shields; and
- Latex gloves.

6.2 Site-Specific Personal Protective Equipment

Site-specific PPE for this project will be selected, used, and maintained in accordance with the requirements contained in 29 CFR 1910.132, 133, 134, 135, 136, and 138. PPE is designed to provide protection to team members when engineering and administrative controls are not feasible for controlling hazards. PPE will be used in conjunction with appropriate mitigation measures to ensure full protection against identified hazards.

6.3 Decontamination Procedures

Personnel decontamination will consist of removing disposable PPE (i.e., paper suits, paper shoe covers, and latex gloves), turning each piece of PPE inside out, and ending with removal of latex gloves. All used PPE will be placed into a plastic bag and transferred to Depot personnel for disposal.

7.0 STANDARD OPERATING PROCEDURES

7.1 Site-Specific Work Practices

While on-site, Radian team members will follow the site-specific practices established in this SSHP. These practices are described below and should be adhered to at all times for the safety of the project team members.

7.2 General Site Operating Procedures/Safety Guidelines

The following are general guidelines for safe operations in areas that are potentially contaminated.

- Wear required PPE at all times.
- Never work alone in an isolated area of the site.
- Practice contamination avoidance. Never sit, kneel, or lay equipment on potentially contaminated surfaces. Avoid obvious sources of contamination.
- No eating, drinking, or smoking is permitted in areas of sites that are suspected of being contaminated.
- In the event PPE is ripped or torn, replace it as soon as safety will allow.
- Be alert to any unusual changes in your own condition; never ignore warning signs. Notify the task leader of suspected exposures or accidents.
- A vehicle will be readily available for emergency use at all times during field efforts. Personnel working on-site shall be familiar with the most direct route to the nearest hospital.
- In the event of direct skin contact with contaminants, immediately wash the affected area with soap and water.
- Copies of the SSHP will be readily accessible at the work site.
- Hands and face should be thoroughly washed before eating or drinking.

- Any substantial modifications to this plan that could affect health and safety must be approved by the EAC or designee.

7.3 Drug Free Workplace Policy

Radian's Drug Free Workplace Policy obligates employees to perform their work free of the influence of alcohol or drugs. As part of this policy each new employee is required to submit to and pass a urine drug screen prior to beginning work. Job offers are made contingent on passing the drug test. The policy has provisions to conduct random drug testing on employees. Upon client request, Radian will provide the client a copy of the Radian Drug Free Workplace Policy.

Any employee who is impaired on the job will not be allowed to continue working. The task leader will be responsible for determining whether an employee should not be allowed on the job site.

8.0 EMERGENCY MEDICAL TREATMENT

Any person who becomes ill or injured should have first aid and or cardiopulmonary resuscitation (CPR) administered while awaiting an ambulance or paramedics. The task leader is trained in CPR and first aid. A first aid kit will be on-site. Injuries must be reported and follow the accident reporting plan in Section 9.0. Any person being transported to a hospital should take a copy of the SSHP. Additionally, if the injured's condition is serious, at least partial decontamination should be considered.

The nearest medical facility is Baptist Hospital in downtown Memphis. When leaving the Depot, take I-55 north to the Downtown Union exit, which leads directly to Baptist Hospital.

9.0 ACCIDENT REPORTING PLAN

9.1 Applicability and Scope

The accident reporting requirements apply to all incidences involving Radian personnel arising out of employment (AOE) or in the course of employment (COE) that result in personal injury, illness, or property damage or incidences that, strictly by chance, did not result in personal injury, illness, or property damage ("near misses").

9.1.1 Injuries and Illnesses

Injuries and illnesses that require reporting include those injuries and illnesses AOE/COE that result in any of the following: lost work time, restrictions in performing job duties, the need for first aid or outside medical attention, permanent physical bodily damage, or death.

Examples of "non-reportable" injuries and illnesses include small minor cuts such as paper cuts, common colds, and small bruises not resulting in work restriction or requiring first aid or medical attention. Examples of "reportable" injuries and illnesses include heat exhaustion from working outside, strained back muscles from moving objects, acid burns on fingers, chronic bronchitis from chemical exposure, and fingers crushed while conducting field activities.

9.1.2 Accidents

Accidents that require reporting include those accidents AOE/COE that result in any of the following: injury or illness damage to a Radian-operated vehicle (rented, leased, or owned), damage to a personal vehicle AOE/COE, fire/explosion, property damage of more than $100, or release of substances requiring evacuation of at least the immediate release/spill area. All lost time accidents and property damage accidents over $2000 shall be reported to the Contracting Officer Representative within 24 hours using Engineer Form 3394.

9.1.3 Near Misses

Other incidences that, strictly by chance, do not result in actual or observable injury, illness, death, or property damage are also required to be reported. The information obtained from such reporting can be extremely useful in identifying and mitigating problems before they result in actual personal or property damage. Thus, these incidences will be treated as if they did result in personal or property damage so that they can be reviewed and corrective actions implemented.

9.2 Responsibilities

All Radian employees and subcontractors have a responsibility to report accidents, injuries, illnesses, and near misses under the Radian Accident Reporting Program. Supervising personnel also have a responsibility to ensure that unsafe working practices or conditions that affected personnel under their supervision are promptly corrected.

9.2.1 Corporate Director of Health and Safety

The CDHS is responsible for ensuring that Radian's health and safety programs effectively minimize accidents and injuries, meet health and safety regulatory requirements, and provide consistency of practices and procedures among Radian offices. The CDHS has overall responsibility for implementing the accident reporting program, including review of accident reports, investigation of accidents, and recommendations of changes in practices, procedures, or the program. The CDHS is responsible for completing all regulatory compliance reports.

9.2.2 EAC

The EAC will review all accident reports and will summarize these reports to the CDHS as needed. Furthermore, the EAC will investigate the accidents if he or she deems it necessary and make recommendations for program improvement, if warranted.

9.2.3 Technical Resource Manager

The Technical Resource Manager has responsibility for ensuring that accident reports for Radian employees are complete and sent to the appropriate human resource administrator (HRA).

9.2.4 Project Manager

The Project Manager shares the responsibility with the affected employee's administrative supervisor for accident reporting. In some instances, when timeliness of reporting is not practicable for an administrative supervisor, such as an injury occurring at a field site or when an administrative supervisor is not available, the Project Manager should complete the necessary accident report forms and submit them to the appropriate HRA.

9.2.5 All Radian Employees

All Radian employees have responsibility to initiate the accident reporting sequence by communicating with their supervisors as soon as possible after an incident they observe or to which they fell victim. To effectively accomplish this, all employees must be familiar with the Radian Accident Reporting Program, including the criteria defining reportable incidents.

9.3 Reporting Procedures and Practices

This section describes the specific procedures and practices that will be followed by Radian personnel to effectively conduct accident reporting. A telephone will be available on-site for use in case of emergency.

9.3.1 Injuries and Illnesses

Serious injury or illness posing a life-threatening situation will be reported immediately to the local emergency response medical services (typically, a local fire department or paramedic service).

Injuries and illnesses will be reported by the victim to his or her administrative supervisor in person or by phone as soon as possible after any life-threatening situation has been addressed. If the victim is unable to report, the supervisor of the activity in which the victim was involved will notify the victim's administrative supervisor.

The supervisor will immediately notify the local EAC verbally of the incident and will complete an Incident Report Form (Appendix B) within 48 hours of the reported incident. This form asks for the following information:

- Date and time of incident,
- Location of incident,
- Description of incident,
- Direct cause of incident,
- Nature of injury/illness (be specific),
- Type of medical treatment provided,
- Name of treating physical or hospital and address, and
- Number of lost work days after date of injury (if already returned to work).

The local HRA will notify the local EAC within 24 hours of the incident. Within 5 days of the incident, the local HRA will complete and submit an Employer's First Report of Injury to the local Workmens' Compensation insurance carrier and send copies, along with copies of the Incident Report Form, to the local EAC and the Health Services Administrator.

Any fatality or incident where three or more employees are hospitalized must be reported to OSHA within 8 hours of any Radian employee becoming aware of the incident.

The first Radian employee becoming aware of such an incident becomes responsible for reporting the incident to the Director of Environmental Affairs or a Technical Resource Manager or company officer. The following information will be required:

- Location of incident,
- Time of incident,
- Number of fatalities or hospitalized employees,
- Contact person,
- Phone number, and
- Brief description of the incident.

When contact is made, the contacted person assumes responsibility for notifying OSHA and for convening an investigation team.

If contact cannot be made within 7 hours, then the responsible employee should contact OSHA directly either by calling the nearest OSHA office or by calling 1-800-321-6742. The report should be confined to the items listed above with no speculation (cause, blame, etc.). The responsible party should continue to try and contact the Director of Environmental Affairs or management until someone has been reached. At this point, the contacted person assumes responsibility for convening an investigation team.

The Health Services Administrator will maintain the OSHA log and summary of recordable injuries on OSHA Form 200 (a separate form will be kept for each office) and will forward copies of the updated Form 200 to the applicable office. A supplementary record will also be maintained by filing the Employer's First Report of Injury (equivalent to OSHA Form 101). The Health Services Administrator will notify the CDHS for each new entry into the reporting system.

The EAC will review each reported accident and determine whether further investigation is required and make recommendations to minimize future similar occurrences.

The CDHS is responsible for reviewing each new accident reported. At the beginning of each calendar year, the CDHS Health Services Administrator will review and sign (certify) the annual summary of OSHA Form 200 for the prior year so that local offices can post the summaries by February 1 following the reporting calendar year.

9.3.2 Accidents

Accidents not involving injury of illness, but resulting in property damage, must be reported to the local EAC on a Radian Accident/Injury Report Form within 48 hours of the accident.

In cases of fire of explosion that cannot be controlled by one person, vehicular accident resulting in injury or more than $500 worth of damage, or chemical release requiring a building evacuation, the involved party must immediately report the incident to the outside agency emergency response services in the area.

Accidents involving a Radian-operated vehicle must be reported as soon as practicable (i.e., after emergency agency reporting is completed) to the local EAC or office/facilities manager with the following information:

- Employee's name,
- Vehicle identity,
- Date and time of accident,
- Location of accident (street address),
- Name and driver's license number of other driver (if applicable),
- Other driver's insurance carrier and policy number,
- Employee's account of accident, and
- Whether police report was filed.

The local EAC or office/facilities manager will immediately notify the Corporate Insurance Clerk and relay the above information.

9.3.3 Near Misses

All near miss incidences are also required to be reported on the Radian Accident/Injury Report Form within 48 hours and submitted to the local EAC. In place of indicating the result of the incident (i.e., actual personal or property damage), the reporting person will indicate the avoided injury or damage.

9.3.4 Training

To ensure that Radian employees are cognizant of the Radian Accident Reporting Program, and are aware of their own and other's responsibilities, a series of informational and instructional training opportunities exist. The employees who will work at this site will be briefed on the Radian Accident Reporting Program during the site-specific training.

Attendance at a New Employee Orientation session, for Radian organization, resources, and procedures information, is required of all new Radian employees. This orientation ensures that new employees are aware of the existence of the *Radian International LLC Health and Safety Manual* and of its contents and who the responsible persons in their organization (office or department) are.

10.0 RECORDKEEPING REQUIREMENTS

The following records are to be maintained in the project files:

- Copy of the SSHP, original sign-off sheet (Figure 10-1), and a copy of the Certification of Hazard Assessment;
- Documentation of the PPE used during sampling (can be in field logbook);
- Copy of any accident or injury reports; and
- Copy of air monitoring results (field logbook or final reports).

By signing below, I acknowledge that I have read and understand the requirements of this Site-Specific Safety and Health Plan, that I have been briefed on the potential hazards involved with this work, and that I will abide by the provisions of this plan.

Signature	Date	Company
Signature	Date	Company
Signature	Date	Company
Signature	Date	Company
Signature	Date	Company
Signature	Date	Company

Figure 10-1. Sign-Off Sheet

11.0 APPROVAL BY CERTIFIED INDUSTRIAL HYGIENIST

This SSHP has been reviewed and approved by Robert Hayes, CIH.

Appendix A

MATERIAL SAFETY DATA SHEETS

DOD Hazardous Materials Information System
DoD 6050.5-LR
AS OF July 1995
Proprietary Version - For U.S. Government Use Only

FSC: 6810
NIIN: 00N006588
Manufacturer's CAGE: 52519
Part No. Indicator: A
Part Number/Trade Name: ISOPROPYL ALCOHOL

General Information

Item Name:
Company's Name: KEPRO CIRCUIT SYSTEMS,INC (CHEMTECH IND. INC)
Company's Street: 630 AXMINISTER DR
Company's P. O. Box:
Company's City: FENTON
Company's State: MO
Company's Country: US
Company's Zip Code: 63026-2906
Company's Emerg Ph #:
Company's Info Ph #:
Distributor/Vendor # 1:
Distributor/Vendor # 1 Cage:
Distributor/Vendor # 2:
Distributor/Vendor # 2 Cage:
Distributor/Vendor # 3:
Distributor/Vendor # 3 Cage:
Distributor/Vendor # 4:
Distributor/Vendor # 4 Cage:
Safety Data Action Code:
Safety Focal Point: N
Record No. For Safety Entry: 001
Tot Safety Entries This Stk#: 001
Status:
Date MSDS Prepared: 01JAN87
Safety Data Review Date: 02DEC85
Supply Item Manager:
MSDS Preparer's Name:
Preparer's Company:
Preparer's St Or P. O. Box:
Preparer's City:
Preparer's State:
Preparer's Zip Code:
Other MSDS Number:
MSDS Serial Number: BCNDB
Specification Number:
Spec Type, Grade, Class:
Hazard Characteristic Code:
Unit Of Issue:
Unit Of Issue Container Qty:
Type Of Container:
Net Unit Weight:

• Report for NIIN: 00N006588

NM[redacted]ate License Number: N/A
Net Explosive Weight:
Net Propellant Weight-Ammo: N/A
Coast Guard Ammunition Code:

Ingredients/Identity Information

Proprietary: NO
Ingredient: ISOPROPYL ALCOHOL (SARA III)
Ingredient Sequence Number: 01
Percent: 100
Ingredient Action Code:
Ingredient Focal Point: N
NIOSH (RTECS) Number: NT8050000
CAS Number: 67-63-0
OSHA PEL: 400 PPM/500 STEL
ACGIH TLV: 400 PPM/500STEL;9192
Other Recommended Limit:

Physical/Chemical Characteristics

Appearance And Odor: CLEAR,COLORLESS LIQUID; ODOR CHARACTERISTIC
Boiling Point: 177-182F
Melting Point:
Vapor Pressure (MM Hg/70 F): 33
Vapor Density (Air=1): 2.1
Specific Gravity: 0.7863
De[redacted]sition Temperature:
Evaporation Rate And Ref: 1.7 (BU AC)
Solubility In Water: COMPLETE
Percent Volatiles By Volume: 100
Viscosity:
pH:
Radioactivity:
Form (Radioactive Matl):
Magnetism (Milligauss): N/P
Corrosion Rate (IPY):
Autoignition Temperature:

Fire and Explosion Hazard Data

Flash Point: 53F (TCC)
Flash Point Method: N/P
Lower Explosive Limit: 2.1
Upper Explosive Limit: 12
Extinguishing Media: CO*2,DRY CHEMICAL,ALCOHOL FOAM
Special Fire Fighting Proc: H*2O SPRAY POSS INEFFECTIVE,MAY BE USED TO COOL CLOSED CNTNR
Unusual Fire And Expl Hazrds: KEEP AWAY FROM HEAT,SPARKS & OPEN FLAME

* Report for NIIN: 00N006588

Reactivity Data

Stability: YES
Cond To Avoid (Stability):
Materials To Avoid: STRONG OXIDIZERS (I.E. PERMANGANATE)
Hazardous Decomp Products: CARBON MONOXIDE FROM BURNING
Hazardous Poly Occur: NO
Conditions To Avoid (Poly): NONE

Health Hazard Data

LD50-LC50 Mixture:
Route Of Entry - Inhalation: N/P
Route Of Entry - Skin: N/P
Route Of Entry - Ingestion: N/P
Health Haz Acute And Chronic:
Carcinogenicity - NTP: N/P
Carcinogenicity - IARC: N/P
Carcinogenicity - OSHA: N/P
Explanation Carcinogenicity:
Signs/Symptoms Of Overexp: SKIN & EYE IRRIT. BREATHING OF VAPS MAY IRRIT NOSE & THROAT. IN HIGH CONC,MAY CAUSE (SEE SUPP DATA)
Med Cond Aggravated By Exp:
Emergency/First Aid Proc: SKIN: WASH W/SOAP & H*2O. EYES: FLUSH W/H*2O FOR 15 MIN. GET MEDICAL ATTENTION. INGESTION: INDUCE VOMITING. GET MEDICAL ATTENTION IMMEDIATELY. INHALATION: REMOVE TO FRESH AIR,GIVE ARTIFICIAL RESPIRATION IF NECESSARY. CALL A PHYSICIAN.

Precautions for Safe Handling and Use

Steps If Matl Released/Spill: ELIMINATE ALL SOURCES OF IGNITION. AVOID BREATHING VAPORS. VENTILATE AREA. REMOVE W/INERT ABSORBENT & NON-SPARKING TOOLS.
Neutralizing Agent:
Waste Disposal Method: WASTE DISPOSAL MUST BE IAW FEDERAL, STATE & LOCAL REGULATIONS.
Precautions-Handling/Storing: DONT STORE ABOVE 120F. STORE LRG AMTS IN STRUCTURES MADE FOR NFPA CLASS 1B LIQUIDS. CNTNRS SHOULD BE GROUNDED WHEN POURING. AVOID FREE FALL OF LIQUID.
Other Precautions: FLAMMABLE. DONT FLAME CUT,BRAZE,WELD. USE ONLY W/ ADEQUATE VENT. AVOID PRLNG BREATHING OF VAP/SPRAY MIST. AVOID CONT W/EYES, SKIN. DONT TAKE INTERNALLY. KEEP CLOSURES TIGHTEUPRIGHT TO PREV LEAK.

Control Measures

Respiratory Protection: FOR EMERGENCY: NIOSH/MSHA APPRVD ORGANIC CANISTER OR SCBA
Ventilation: LOCAL EXHAUST PREFERABLE
Protective Gloves: RUBBER GLOVES
Eye Protection: CHEMICAL SAFETY GOGGLES
Other Protective Equipment: PROTECTIVE GLOVES SHOULD BE SOLVENT RESISTANT
Work Hygienic Practices:

Appendix B

ACCIDENT REPORT FORMS

RADIAN
CORPORATION

INCIDENT REPORT

DATE: _____ LOCATION: _____

TIME: _____

Description of incident: _____

_____

_____

_____

Personnel involved: _____

_____

_____

Describe injuries (if applicable): _____

_____

_____

DESCRIBE TREATMENT:

Company first aid: _____

_____

Physician's treatment: _____

_____

Further treatment: _____

_____

RADIAN
CORPORATION

CORRECTIVE ACTIONS: _____

_____

_____

ADDITIONAL COMMENTS: _____

_____

_____

REPORTED BY: _____ DATE: _____

DISTRIBUTION:
CDHS: JTC EAC: SKT GRP LDR: _____

EMPLOYEE(S): _____

These forms are intended to help identify and correct conditions or practices which result in or could result in injury to personnel and/or property damage. Please complete forms for "near miss" as well as employee injury incidents. Employees involved in the incident, and their supervisors, should complete these forms within two days. Supervisors are responsible for ensuring timely distribution.

c:\skt\incident

Appendix C

MEDICAL SURVEILLANEC, HAZWOPER TRAINING, AND FIRST AID/CPR TRAINING DOCUMENTATION

RADIAN INTERNATIONAL LLC

HEALTH STATUS MEDICAL REPORT

Employer Copy

Type of Examination: Baseline Examination

Employee:	Kilroy, Marshal	Position:	Sr. Environmental Scientist
SSN:	435-58-6687	Location:	Oakridge (OKR)
Date of Exam:	09/24/1997	Site:	Oak Ridge
Expiration Date:	09/24/1998		

The following recommendations are based on a review of one or all of the following: a base history questionnaire, supporting diagnostic tests, physical examination, and the essential functions of the position applied for or occupied by the individual named above. The recommendations comply with Federal OSHA 29 CFR 1910.120 Hazardous Waste Operations and Emergency Response Standard and 29 CFR 1910.134 Respiratory Protection Standard.

	Yes	No	Undecided
Has the employee any detected medical conditions that would increase his/her risk of material health impairment from occupational exposure in accordance with 29 CFR §1910.120?	☐	☑	☐
Does the employee have any limitations in the use of respirators in accordance with 29 CFR §1910.134?	☐	☑	☐

STATUS

1. ☑ QUALIFIED — The examination indicates no significant medical impairment. Employee can be assigned any work consistent with skills and training.

2. ☐ QUALIFIED — The examination indicates non-occupational medical impairment(s) and employee has been referred to his/her personal physician for follow-up. Employee can be assigned to any work consistent with skills and training.

3. ☐ QUALIFIED - WITH LIMITATIONS — The examination indicates that a medical impairment currently exists that limits work assignments on the following basis:

4. ☐ NOT QUALIFIED

5. ☐ DEFERRED — The examination indicated that additional information is necessary. The employee has been given the following instructions.

COMMENTS:

I have reviewed the medical data of the above named employee, and informed the employee of the results of the medical examination and any medical conditions that require follow-up examination or treatment.

Name of Physician: Wesley P Chan, M.D. Date: 09/30/97

Signature: _____

GMG WorkCare
333 S. Anita Drive, Suite 630
Orange, CA 92868
(714) 978-7488 - (800) 455-6155 - FAX (714) 456-2154

GMG WorkCare™

TYPE OF EXAMINATION
☐ Baseline ☒ Termination / Exit
☐ Annual ☐ Special Occupational
☐ Other: _____ Specify

WORK STATUS REPORT

Employee Name: Patrice G. Cole	Position: Environmental Scientist	Date of Exam: 05/20/97
Employer Name: Radian International	Location: Oak Ridge	Social Security Number: 227-78-6554

The following recommendation is based on a review of a base history questionnaire, diagnostic tests, physical examination, and the essential functions of the position applied for or occupied by the individual named above.

STATUS:

1. ☑ The examination indicates no significant medical impairment. Can be assigned any work consistent with skills and training.

2. ☐ The examination indicates that a medical impairment currently exists that limits work assignments:

   - ☐ Cannot perform an essential job function (s): _____
   - ☐ Not to lift over _____ pounds
   - ☐ Not to work at a specific job or area: _____
   - ☐ No work with chemicals or irritants
   - ☐ No work requiring filter type respiratory protective device
   - ☐ No work in confined spaces
   - ☐ No SCBA use; No work requiring impermeable protective clothing
   - ☐ Not to work with volatile organic compounds, organic solvents, or hepatotoxins
   - ☐ Must wear hearing protection in areas with noise levels greater than 85 decibels
   - ☐ Sitting work only
   - ☐ Day work only (no shift work)
   - ☐ No overtime
   - ☐ No repeated waist bending
   - ☐ Suggested accommodations: _____
   - ☐ Not to operate commercial vehicle: _____

3. ☐ Deferred, the examination indicated that additional information is necessary. Employee given the following instructions:

_____

_____

The following recommendations comply with Federal OSHA standards:

	YES	NO	UNDECIDED
Has the employee any detected medical conditions that would increase his/her risk of material health impairment from occupational exposure?	☐	☑	☐
Does the employee have any limitations in the use of personal protective equipment, (e.g. clothing or respirators)?	☐	☑	☐

Name of Physician: Wesley P Chan, M.D. Date: 05/23/97

Signature: _____

* The employee has been informed of the results of this examination.

333 S. Anita Drive, Suite 630, Orange, CA 92868 • (714) 978-7488 • (800) 455-6155 • FAX (714) 456-2154

American Red Cross

This certifies that

Marshall Kilroy

has completed the requirements for

ADULT CPR

sponsored by

APPALACHIAN CHAPTER

Date completed

1/25/96

American Red Cross

This certifies that

Marshall Kilroy

has completed the requirements for

STANDARD FIRST AID

sponsored by

APPALACHIAN CHAPTER

Date completed

1/25/96

American Red Cross

This certifies that

Patrice Cole

has completed the requirements for

STANDARD FIRST AID

sponsored by

Appalachian Chapter

Date completed

8/5/93

American Red Cross

This certifies that

Patrice Cole

has completed the

ADULT CPR

course of instruction sponsored by

Appalachian Chapter

Date course completed

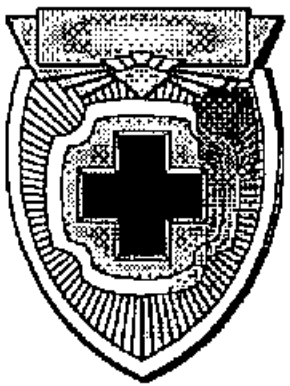
8/5/93

Certificate of Completion

This Certifies that

Patrice Cole

has met the annual 8 hour refresher training requirements for Hazardous Waste Site Operations and Emergency Response - OSHA Standard 1910.120



Radian Corporation, Technical Resources Manager

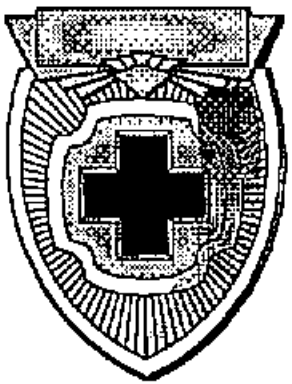
December 1996

Certificate of Completion

This Certifies that

Marshall Kilroy

has met the annual 8 hour refresher training requirements for Hazardous Waste Site Operations and Emergency Response - OSHA Standard 1910.120



Radian Corporation, Technical Resources Manager

December 1996

FINAL PAGE

ADMINISTRATIVE RECORD

FINAL PAGE

FINAL PAGE

ADMINISTRATIVE RECORD

FINAL PAGE