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**HUMAN HEALTH RISK EVALUATION
FOR
PALOS VERDES SHELF**

April 1, 1999

(Corrected version)

Submitted To:

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NOTE TO READER: This "corrected version" of the April 1, 1999 Human Health Risk Evaluation incorporates the corrections listed in EPA's June 2, 1999 Errata Sheet for the Human Health Risk Evaluation for Palos Verdes Shelf.

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LIST OF ACRONYMS

ATSDR	Agency for Toxic Substances and Disease Registry
CDF	cumulative distribution function
CDFG	California Department of Fish and Game
CDI	chronic daily intake
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act
CFR	Code of Federal Regulations
COPCs	chemicals of potential concern
CPHF	California Public Health Foundation
CT	central tendency
DDD	1,1-dichloro-2,2-bis (p-chlorophenyl) ethane
DDE	2,2-bis (p-chlorophenyl) 1,1-dichloroethylene
DDMU	1-chloro-2,2-bis (p-chlorophenyl) ethylene
DDT	1,1,1-trichloro-2,2-bis (p-chlorophenyl) ethane
EE/CA	Engineering Evaluation/Cost Analysis
EPA	U.S. Environmental Protection Agency
EPC	exposure point concentration
FDA	United States Food and Drug Administration
FR	Federal Register
g/day	grams per day
HCB	hexachlorobenzene
HCH	hexachlorocyclohexane
HHRE	human health risk evaluation
HML	Hazard Materials Laboratory
HQ	hazard quotient
IRIS	Integrated Risk Information System
JWPCP	Joint Water Pollution Control Plant
kg	kilogram
LACSD	Los Angeles County Sanitation Districts
LADD	lifetime average daily dose
LOAEL	lowest-observed-adverse-effect level
MDL	method detection limit
mg/kg	milligrams per kilogram
mg/kg-day	milligrams per kilogram per day
NIEHS	National Institute of Environmental Health Sciences
NOAEL	no-observed-adverse-effect level
NPDES	National Pollutant Discharge Elimination System
NRDA	Natural Resource Damage Assessment
OEHHA	Office of Environmental Health Hazard Assessment
PAI	Pacific Analytical Incorporated
PCBs	polychlorinated biphenyls
PDFs	probability density functions
ppm	parts per million
QA	quality assurance

LIST OF ACRONYMS (Continued)

RAGS	Risk Assessment Guidance for Superfund
RfD	reference dose
RME	reasonable maximum exposure
RPD	relative percent differences
SCCWRP	Southern California Coastal Water Research Project
SDCDHS	San Diego County Department of Health Services
SFs	slope factors
SIF	summary intake factor
SMBRP	Santa Monica Bay Restoration Project
DDT	DDT and its metabolites
UCL95	95 percent upper confidence limit
USEPA	United States Environmental Protection Agency
USFDA	United States Food and Drug Administration
USGS	United States Geological Survey
$\mu\text{g}/\text{m}^3$	micrograms per cubic meter
$\mu\text{g}/\text{L}$	micrograms/liter
$\mu\text{g}/\text{day}$	micrograms per day
$\mu\text{g}/\text{kg}$	micrograms per kilogram

EXECUTIVE SUMMARY

A streamlined human health risk evaluation (HHRE) was performed to describe the human health risks posed by the presence of 1,1,1-trichloro-2,2-bis (p-chlorophenyl) ethane (DDT) and its metabolites, (referred to collectively as tDDT or total DDT) and polychlorinated biphenyls (PCBs or total PCB) in contaminated sediments on the Palos Verdes Shelf, focusing on the consumption of contaminated fish by boat anglers as the primary exposure pathway. Potential risks to human health are due to the consumption of fish which have bioaccumulated contaminants from sediments. The evaluation focused on risks for two types of exposure scenarios, a reasonable maximum exposure (RME) scenario and a central tendency scenario. The RME scenario is a high-end exposure scenario based on single-species fish consumption rates (i.e., consumption rates averaged over anglers who consume a particular species). The central tendency, or average, scenario assumes a mixed-species diet and uses median consumption rates averaged over all boat anglers. Single point estimates of risks were calculated and Monte Carlo simulation was employed to quantitatively evaluate uncertainty and variability in the risk estimates.

The HHRE considered consumption of the 12 species of fish most commonly consumed by Santa Monica Bay boat anglers, based on information collected as part of the *Santa Monica Bay Seafood Consumption Study* (SMBRP, 1994). Fish tissue concentrations of tDDT and PCBs for these 12 species are based on data collected by the Los Angeles County Sanitation Districts (LACSD) (white croaker, kelp bass, California halibut, surfperches) and for the Office of Environmental Health Hazard Assessment (OEHHA) Comprehensive Study (barred sand bass, California scorpionfish, California sheephead, chub mackerel, halfmoon, Pacific barracuda, Pacific bonito, and rockfishes; Pollock et al., 1991). Where possible, recent tissue data from the Palos Verdes Shelf were used. For example, white croaker tissue data from 1996 and 1997, collected from LACSD Zones 1, 2, and 3, were used in the HHRE.

Fish consumption rates are based on 338 boat anglers who reported consuming fish in the previous 4 weeks (28 days) in the *Santa Monica Bay Seafood Consumption Study* (SMBRP, 1994). An RME scenario was evaluated for each of the 12 fish species included in the HHRE; consumption rates were based on consumers of a particular fish species. For example, 13 people reported eating white croaker during the previous 28 days. The average consumption rate (estimated using the 95 percent upper confidence limit on the mean) of white croaker by these 13 white croaker consumers (27.9 g/day) was used to quantify the RME scenario for this species. This represents about six 150-gram meals per month. The central tendency scenario assumed that an angler would eat all 12 fish species, with consumption rates for each species calculated by multiplying the species diet fraction by the median fish consumption rate for all 338 boaters. For example, white croaker represents 2.2 percent,

or 0.48 g/day, of the overall median fish consumption rate (21.4 g/day) for boat anglers, based on the results of the SMBRP (1994) study. This represents about one 150-gram meal of white croaker every year.

Exposure durations used to quantify human health risks are based on the reported fishing durations of boat anglers in the *Santa Monica Bay Seafood Consumption Study* (SMBRP, 1994). Reported fishing duration reflects only the length of time the surveyed individuals had been fishing up to the time of the survey. Because no information is available on how long these individuals will continue to fish in the future, the reported fishing duration is not equivalent to total exposure duration. The 90th percentile reported fishing duration of 30 years was used to quantify the RME scenario; the mean reported fishing duration of 13.8 years was used to quantify the central tendency scenario.

Human health risks were evaluated in terms of both cancer risk and noncancer health hazard. Potential cancer risk was estimated by calculating the increased probability of an individual developing cancer during his or her lifetime as a result of exposure to tDDT and PCBs. PCBs and tDDT are both classified as probable human carcinogens. For the RME scenario, excess cancer risks exceed 1×10^{-4} for white croaker (cancer risk of 2×10^{-3} , or 1 in 500) and surfperches (cancer risk of 2×10^{-4} , or 1 in 5,000). Total DDT is the major contributor to cancer risk. For the central tendency scenario, the excess cancer risk was estimated to be 2×10^{-5} (or 1 in 50,000).

The potential for adverse health effects other than cancer was evaluated by comparing the average daily intake of tDDT and PCBs with a reference dose, below which no adverse health effects are expected to occur. The hazard quotient (HQ) is the ratio of the chemical intake to the reference dose. For the RME scenario, consumption of several fish species results in a potential noncancer hazard: white croaker (HQ = 32), surfperches (HQ = 5), barred sand bass (HQ = 3), California halibut (HQ = 3), California sheephead (HQ=2), and kelp bass (HQ = 2). For the central tendency scenario, HQs for tDDT and PCBs are less than 1. PCBs are the main contributor to the noncancer health hazard.

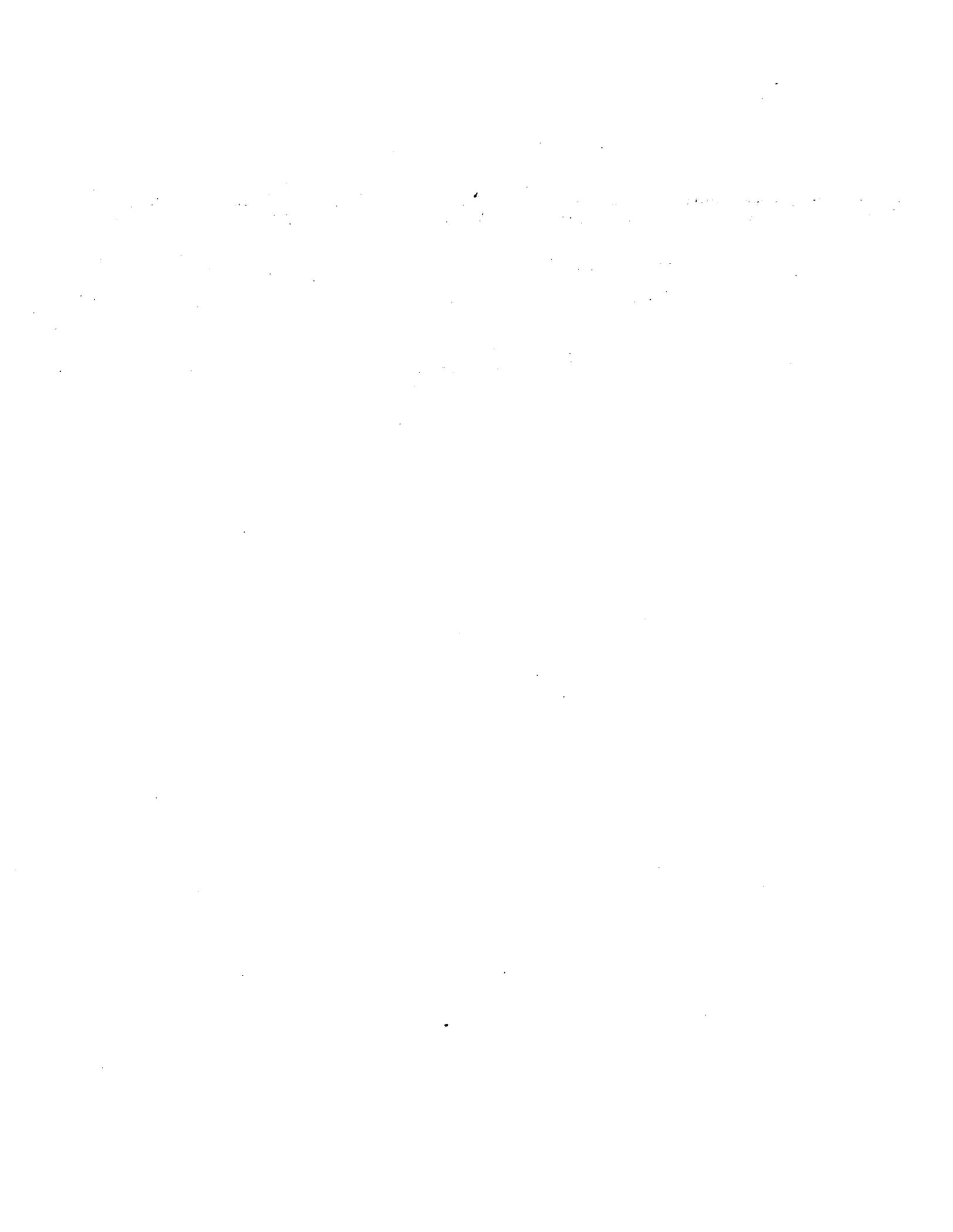
It should be noted, however, that boat anglers generally do not consume only a single species of fish. For example, since the UCL95 on the mean total fish consumption rate (i.e., all species) is 53.0 g/day, a consumer of white croaker (at the RME consumption rate of 27.9 g/day) may also be consuming a variety of other fish species. The contribution of tDDT and PCBs in these other fish species to human health risk is not reflected in the RME results.

In addition to the point estimate risk calculations described above, a Monte Carlo simulation was performed to evaluate uncertainty and variability in the consumption of white croaker by boat

anglers. Results of the Monte Carlo simulation indicated that the mean cancer risk is 3×10^{-4} , and the 95th percentile cancer risk is 1×10^{-3} . About 45 percent of simulation results were above 1×10^{-4} ; in other words, a cancer risk of 1×10^{-4} corresponds to a 55th percentile of the output distribution. The mean and median noncancer HQs (7 and 3, respectively) are greater than 1, the level above which there may be a concern for potential noncancer health effects. The 95th percentile HQ is 26. About 75 percent of simulation results exceeded a HQ of 1 (i.e., a HQ of 1 corresponds to a 25th percentile of the output distribution). Sensitivity studies were performed to identify those input parameters that represent the greatest contributors to variance in the cancer risk and noncancer hazard for recreational boat anglers consuming white croaker. Exposure duration is the largest contributor to variance in the cancer risk results, followed by tDDT and PCB concentrations in white croaker tissue. Tissue concentrations of tDDT and PCBs are the largest contributors to variance in the noncancer hazard, followed by the white croaker consumption rate. These exposure factors reflect both uncertainty and natural variability in a population. These results indicate that, based on available data on fish consumption rates, exposure duration, and white croaker tissue concentrations, both cancer and noncancer health effects are likely to occur for boat anglers who catch and consume white croaker collected at the Palos Verdes Shelf.

An evaluation of the potential risks to breast-fed infants due to consumption of tDDT and PCBs in breast milk was performed. The evaluation indicated that, based on a maternal fish consumption rate of one 150-gram meal of white croaker per month, breast milk concentrations of tDDT and PCBs could be as high as 0.8 mg/kg and 0.05 mg/kg, respectively. These breast milk concentrations correspond to infant hazard quotients of 220 and 370 for tDDT and PCBs, respectively.

A qualitative uncertainty analysis was presented which identified the assumptions and limitations in each phase of the risk evaluation (site data evaluation, toxicity assessment, exposure assessment, risk characterization), and their effect on the overall risks calculated for the site.



1.0 INTRODUCTION

This human health risk evaluation was conducted in conjunction with a non-time-critical removal action currently being considered by the United States Environmental Protection Agency (USEPA or EPA), Region IX at the Palos Verdes Shelf under the authority of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). The purpose of this report is to summarize, based on existing data, the human health risks posed by contaminated effluent-affected sediments on the Palos Verdes Shelf. In accordance with the *Guidance on Conducting Non-Time-Critical Removal Actions under CERCLA* (USEPA, 1993a), a streamlined human health risk evaluation (HHRE) report has been prepared. According to the guidance, a streamlined risk evaluation is intermediate in scope between the limited risk evaluation undertaken for emergency removal actions and the conventional baseline assessment normally conducted for remedial actions; can help justify taking a removal action and identify what current or potential risks should be prevented; and projects the potential risk of health problems occurring if no cleanup action is taken at the site. The results of this risk evaluation will be included in the Engineering Evaluation/Cost Analysis (EE/CA) currently being conducted. In this streamlined risk evaluation, only the most significant contaminants are included in the analysis; the exposure pathways are limited to those of greatest concern, in this case, fish consumption; and the analysis is based on existing, available data.

The HHRE is based on existing historically collected data from a variety of sources:

- Los Angeles County Sanitation Districts (LACSD) National Pollutant Discharge Elimination System (NPDES) bioaccumulation monitoring reports (LACSD, various years) and other data collected by LACSD, which include fish tissue concentration data for white croaker, kelp bass, black surfperch, and California halibut;
- California Office of Environmental Health Hazard Assessment (OEHHA) *Study of Chemical Contamination of Marine Fish from Southern California* (Pollock et al., 1991), which reports tissue concentration data in 16 fish species from 24 sites in southern California, including locations on the Palos Verdes Shelf;
- *Santa Monica Bay Seafood Consumption Study* (SMBRP, 1994), which describes fish consumption patterns and rates in areas including the Palos Verdes Shelf.

The HHRE has been performed consistent with USEPA and State of California risk assessment guidance, including:

- *Risk Assessment Guidance for Superfund (RAGS), Volume I, Human Health Evaluation Manual, Part A* (USEPA, 1989a);
- *Supplemental Guidance to RAGS: Standard Default Exposure Factors* (USEPA, 1991);
- *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories, Volume II, Risk Assessment and Fish Consumption Limits* (USEPA, 1994a);
- *Guidance for Risk Characterization* (USEPA, 1995b);
- *PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures* (USEPA, 1996a);
- *Exposure Factors Handbook* (USEPA, 1997a);
- *Guiding Principles for Monte Carlo Analysis* (USEPA, 1997c);
- *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 1996);

1.1 Scope of the Human Health Risk Evaluation

This HHRE presents the human health risks posed by the presence of polychlorinated biphenyls (PCBs or total PCBs) and 1,1,1-trichloro-2,2-bis (p-chlorophenyl) ethane (DDT) and its metabolites, (2,2-bis (p-chlorophenyl) 1,1-dichloroethylene (DDE) and 1,1-dichloro-2,2-bis (p-chlorophenyl) ethane (DDD)) in contaminated sediments on the Palos Verdes Shelf, focusing on the consumption of contaminated fish as the primary exposure pathway. Because a streamlined approach was used to develop the HHRE, only existing information was used to develop the risk estimates. The scope of this evaluation is limited to the quantitative assessment of the following:

- Human health risks from chemicals of greatest concern: although other contaminants are present in Shelf sediments and fish tissue, potential risks due to DDT and its metabolites, (referred to collectively as tDDT) and PCBs are significantly higher (see Section 2.2) and, therefore, the HHRE is focused on these compounds;
- Human health risks due to the most significant exposure route: although other routes of exposure to tDDT and PCBs in sediment or fish may be possible, consumption of

contaminated fish by recreational anglers is believed to be the most significant exposure pathway and, therefore, is evaluated quantitatively in this HHRE. Although subsistence fishing may occur in the Palos Verdes Shelf area, site-specific (e.g., Santa Monica Bay area) fish consumption data was available for recreational anglers only;

- Reasonable maximum exposure and central tendency (CT) scenarios: in accordance with USEPA guidance (USEPA, 1995b, 1995c), a high-end exposure scenario was evaluated to ensure the protection of human health. An RME scenario is not a worst case, but an estimate of exposure in the upper range of the risk distribution (i.e., above the 90th percentile of the distribution of risks to recreational anglers). In addition, a central tendency exposure scenario was evaluated, using average and/or median values for exposure parameters. The central tendency scenario reflects central estimates of exposure or dose, and does not necessarily represent a particular individual on the risk distribution (USEPA, 1995b);
- Variability and uncertainty in selected exposure parameters (fish ingestion rate, tissue concentrations of tDDT and PCBs, exposure duration, body weight) for the RME scenario using a Monte Carlo analysis.

The HHRE is not intended to address the following issues:

- consumption of commercially caught white croaker or other fish species;
- overall risks associated with multimedia exposure to tDDT and PCBs (e.g., exposure due to other food sources, air, water, and soil);
- potential risks to children, other than the evaluation of nursing infants described below; and
- effects of fish consumption advisories on the fishing/consumption habits of recreational anglers.

The following issues are included in the HHRE in a qualitative or semiquantitative manner:

- potential risks to nursing infants; and
- effects of cooking the fish on exposure to tDDT and PCBs.

1.2 Site Background

1.2.1 Site History

From 1947 to 1983, the Montrose Chemical Corporation of California, Inc. (Montrose) operated a DDT manufacturing plant in Los Angeles County, California. Wastewater containing DDT was discharged from the Montrose manufacturing plant to the Joint Outfall System and conveyed to the Joint Water Pollution Control Plant (JWPCP) owned and operated by LACSD. Wastewater from the JWPCP is discharged through submarine outfalls located offshore from Whites Point on the Palos Verdes Shelf. PCBs were also discharged from the Whites Point LACSD outfall. Historically, PCB contamination entered the LACSD system as the result of discharges from several sources in the greater Los Angeles area (USEPA, 1996b).

The discharge of DDT and PCBs in contaminated wastewater from the Whites Point outfall has resulted in tDDT and PCB contamination of the sediments on the Palos Verdes Shelf. The most significant discharges of DDT and PCBs to the Palos Verdes Shelf ceased in the early 1970s. Figure 1-1 presents the location of the contaminated sediments. The sediments most contaminated by tDDT and PCBs have gradually been buried, primarily by solids discharged to the Palos Verdes Shelf through Whites Point outfall and from the nearby Portuguese Bend landslide. In recent years, sediment input from both of these sources has been greatly reduced, and sedimentation rates are likely to be low into the future (USEPA, 1996b).

Currently, high levels of tDDT and PCBs are found in the Palos Verdes Shelf sediments; fish from the Palos Verdes Shelf are also contaminated with high levels of tDDT and PCBs, demonstrating an ongoing release of these hazardous substances to the food chain. Modeling by the United States Geological Survey (USGS) indicates that much of the mass of tDDT and PCBs is likely to remain on the Palos Verdes Shelf in near-surface sediments for 100 years or more (Drake, 1994).

Potential risks to human health from the Palos Verdes Shelf sediments are due to the bioaccumulation of tDDT and PCBs in fish. Recent studies related to fish contamination include a SMBRP (1992) study of white croaker, a SMBRP (1994) fish consumption study, and LACSD (various years) *Palos Verdes Ocean Monitoring Reports*. Results of the 1992 SMBRP fish contamination study show that the highest levels of PCBs in fish occur on or in the vicinity of the Palos Verdes Shelf, although there may be other sources in Santa Monica Bay. Extremely elevated levels of tDDT, however, are confined to the Palos Verdes Shelf area.

In 1985, the State of California issued a health advisory recommending limitations on the consumption of sport fish and discouraging consumption of white croaker caught in the Santa Monica Bay, the Palos Verdes Shelf, and Los Angeles/Long Beach Harbor area because of tDDT and PCB contamination. At that time, state agencies were directed to conduct a comprehensive study of chemical contaminants in sport fish and a risk assessment for their consumption. OEHHA completed the study in September 1991 (Pollock et al., 1991). Based on this study, OEHHA issued a health advisory (Table 1-1) recommending, in part, that recreational anglers not consume white croaker caught in most areas offshore of Los Angeles County and Orange County, and that anglers greatly limit consumption of a number of other fish species caught on or in the vicinity of the Palos Verdes Shelf. These warnings have been included in the California sport fishing regulations since March 1, 1992. However, recreational anglers continue to catch and consume white croaker and other species potentially contaminated with tDDT and PCBs in areas which are covered by the fish advisories.

In 1990, the California Department of Fish and Game (CDFG) closed commercial fishing of white croaker on the Palos Verdes Shelf because of the threat to human health posed by the tDDT and PCB contamination. The closure was adopted in Title 14 of the California Code of Regulations in May 1990, and became permanent in February 1991. The closure extends from Point Vicente to Point Fermin, in waters from zero to three nautical miles from shore extending oceanward (Figure 1-2). CDFG (1996) commercial catch data for 1996 indicate that over 100,000 pounds of white croaker were landed in the catch block areas located near Palos Verdes (719, 720, 740, 741). A 1996 study by Heal the Bay (Gold et al., 1997) indicated that levels of DDT and PCB found in some white croaker purchased in retail markets (primarily Asian markets) were similar to those in fish caught over the Palos Verdes Shelf. Some tissue DDT concentrations exceeded the Food and Drug Administration (FDA) Action Level for DDT, whereas measured PCB concentrations were below the FDA Tolerance Limit. Measured DDT concentrations corresponded to theoretical excess cancer risks of up to 1 in 420. Based on reviews of CDFG data, Gold et al. (1997) concluded that from 67 to 91 percent of the commercial white croaker landing from 1993 to 1996 was harvested within the closure area. CDFG (1998) commercial catch data reported since the study was released show overall declines in the white croaker catch; CDFG commercial catch data (blocks 719, 720, 740 and 741) reported in 1998 reflect only 22,995 pounds of white croaker having been landed. The Heal the Bay study, subsequent proposition 65 lawsuits filed against fish markets selling white croaker, and legislative efforts to ban commercial fishery of white croaker are the most likely explanations for the decrease in commercial catch of white croaker.

1.2.2 Relevant Studies

Summaries of pertinent studies related to contaminated seafood or bioaccumulation of contaminants in fish are provided below.

OEHHA's Study of Chemical Contamination of Marine Fish from Southern California, 1991.

This study consisted of a pilot phase and a comprehensive phase. During the pilot phase, OEHHA sampled and analyzed an extensive number of chemicals (selected trace metals and chlorinated hydrocarbons) in a limited number of fish samples. Using these results, potential contaminants of concern were identified for further evaluation in the comprehensive study described below (Pollock et al., 1991). The only chemicals of concern identified in the pilot study were DDT and its metabolites, and PCBs. Potential concerns were raised relative to methylmercury concentrations in higher trophic level fishes and tributyltin levels in fishes in sheltered marinas.

The comprehensive study, performed for OEHHA in 1987, involved collecting frequently caught fish species in southern California to determine the concentrations in edible muscle tissue of the chemicals of concern identified in the pilot study described above. Fish were collected from 24 sites in southern California from the northern tip of Santa Monica Bay to as far south as Dana Point and as far west as Catalina Island. Sampling sites represented areas fished by pier, private boat, and party boat anglers, and included locations on the Palos Verdes Shelf (northwest side of Palos Verdes, Point Vicente, and Whites Point). In all, 16 different species of fish were sampled in the study. Nearly 4,000 fish were sampled, and approximately 1,000 chemical analyses were performed on composite samples from the fish. Fish samples were analyzed for tDDT, PCBs, chlordane, mercury, and tributyltin. The species sampled were sculpin (also known as California scorpionfish), various rockfish species, barred sand bass, kelp bass, Pacific bonito, Pacific (or chub) mackerel, California halibut, Pacific sand dab, corbina, white croaker, queenfish, surfperch species, Pacific barracuda, opaleye, halfmoon, and black croaker. Total DDT concentrations in composite samples of fish on the Palos Verdes Shelf ranged from 4 micrograms per kilogram ($\mu\text{g}/\text{kg}$) to 8,052 $\mu\text{g}/\text{kg}$ wet weight; PCBs (sum of Aroclors 1254 and 1260) in composite samples of fish found near the Palos Verdes Shelf were at levels ranging from nondetectable to 589 $\mu\text{g}/\text{kg}$. The study found that the most contaminated sites appear to be off the Palos Verdes Peninsula and around the Los Angeles-Long Beach Harbors. In general, white croaker was found to be the most contaminated fish species; other relatively contaminated species were corbina, queenfish, surfperches, and sculpin.

The comprehensive study also calculated human health cancer risks from the recreational consumption of various fish species, as well as from a mixed-species diet, assuming a fish consumption rate of 23 grams per day (g/day). Cancer risks ranged from 1×10^{-6} to 2×10^{-3} for

individual fish species; highest risks were for consumption of white croaker from the Palos Verdes Shelf area. For a mixed-species diet, calculated cancer risks were 3×10^{-4} for boat anglers at Whites Point and 4×10^{-4} for anglers fishing from Cabrillo Pier.

In addition to tDDT and PCBs (Aroclors 1254 and 1260), Pollock et al. (1991) collected fish tissue data for chlordane, mercury, and tributyltin. Very few reported chlordane concentrations were above the method detection limit (MDL) of $3 \mu\text{g}/\text{kg}$. Mean chlordane concentrations in composite samples of fish tissue in the Palos Verdes area ranged from non-detectable to a high of $15 \mu\text{g}/\text{kg}$ in white croaker at Whites Point. Chlordane did not contribute significantly to the risk in any of the locations/species sampled in the Pollock study.

Fish tissue samples from four southern California sampling locations, including Whites Point, were analyzed for total mercury. The results for composite samples from Whites Point ranged from $50 \mu\text{g}/\text{kg}$ to $724 \mu\text{g}/\text{kg}$; mean mercury concentrations ranged from $136 \mu\text{g}/\text{kg}$ in white croaker to $219 \mu\text{g}/\text{kg}$ in kelp bass. Concentrations of mercury detected in the fish species sampled did not indicate a health concern.

One sampling of white croaker was collected from Marina del Rey and analyzed for tributyltin. The results for composite samples ranged from 52 to $105 \mu\text{g}/\text{kg}$; the mean concentration was $71.2 \mu\text{g}/\text{kg}$. Concentrations of tributyltin did not indicate a health concern, based on results of the comprehensive study.

Santa Monica Bay Seafood Contamination Study, 1992. In this study, white croaker and yellow rock crab samples were collected from various sites in Santa Monica Bay, including the Palos Verdes Shelf study area, and were analyzed for selected chlorinated hydrocarbons and metals. The highest concentrations of PCBs, tDDT, 1-chloro-2,2-bis (*p*-chlorophenyl) ethylene (a metabolite of DDE and DDD known as DDMU), hexachlorocyclohexane (HCH), hexachlorobenzene (HCB), and chlordane in white croaker were significantly higher on the Palos Verdes Shelf than elsewhere. Concentrations of HCH, HCB, and chlordane in white croaker were quite low compared to the concentrations of PCBs and tDDT. Concentrations of PCBs and tDDT in yellow crab were several orders of magnitude less than white croaker tissue concentrations. Aroclor 1254 was the dominant PCB mixture at all sites sampled. Total PCBs ranged from $314 \mu\text{g}/\text{kg}$ to $1,464 \mu\text{g}/\text{kg}$ at the Palos Verdes sampling locations (Palos Verdes North, Palos Verdes South, and Whites Point). Concentrations of tDDT ranged from $5,207 \mu\text{g}/\text{kg}$ to $18,336 \mu\text{g}/\text{kg}$ at the Palos Verdes Shelf. Maximum concentrations of HCH, HCB, chlordane, and selenium in white croaker near Palos Verdes were $1.5 \mu\text{g}/\text{kg}$, $0.4 \mu\text{g}/\text{kg}$, $29 \mu\text{g}/\text{kg}$, and $1,400 \mu\text{g}/\text{kg}$, respectively.

LACSD Monitoring Program. Required monitoring of contaminants in fish tissues began with the 1988 NPDES monitoring program. Sampling requirements were refined in the 1991 NPDES monitoring program. Since 1991, annual monitoring of white croaker has been conducted in three zones on the Palos Verdes Shelf; kelp bass are usually monitored biennially at three zones (some changes in schedule occurred due to participation in the regional monitoring program). Other fish species, such as California halibut and surfperch, have been collected outside of the NPDES required bioaccumulation monitoring. The most recent available LACSD data for white croaker were collected in 1997 from Zones 1, 2, and 3 (Figure 1-3). Kelp bass and black surfperch were collected in 1996 from Zones 1, 2, and 3; California halibut samples were collected from Zone B (between Zones 1 and 2) in 1991. White croaker muscle tissue concentrations reported for tDDT during the period 1994 to 1997 ranged from 470 to 135,000 $\mu\text{g}/\text{kg}$; total PCBs ranged from 70 to 10,200 $\mu\text{g}/\text{kg}$. Kelp bass muscle tissue concentrations in 1995/1996 ranged from 20 to 1,120 $\mu\text{g}/\text{kg}$ and nondetectable to 160 $\mu\text{g}/\text{kg}$ for tDDT and total PCBs, respectively. Black surfperch muscle tissue data from 1996 show concentrations of 40 to 1,310 $\mu\text{g}/\text{kg}$ tDDT and nondetectable to 150 $\mu\text{g}/\text{kg}$ PCBs. California halibut tissue concentrations of tDDT and PCBs from 1991 were reported as 35 to 1,360 $\mu\text{g}/\text{kg}$ and 30 to 180 $\mu\text{g}/\text{kg}$, respectively.

Santa Monica Bay Risk Assessment (SMBRP, 1997). Fish contamination and consumption data from previous OEHHA and SMBRP studies (described above) were used to construct exposure scenarios for anglers fishing in Santa Monica Bay, conduct deterministic and probabilistic exposure assessments, and calculate risks for these scenarios. Three types of scenarios were developed for each angler group: those using deterministic consumption rates and single-species diets; those using deterministic consumption rates with mixed-species diets; and those using probability distributions to estimate selected exposure variables and mixed-species diets. Single-species cancer risks were highest for white croaker and ranged from 1×10^{-3} to 8×10^{-3} for consumption rates ranging from 21 g/day to 140 g/day. Hazard quotients (HQs) ranged from 7 to 47 for white croaker consumption. Risks for mixed-species diets were calculated for 147 scenarios reflecting different fishing mode, ethnicity, and income variables. Cancer risks and hazard quotients were generally highest for hispanics at piers and jetties and blacks on private boats (cancer risk $>1 \times 10^{-3}$ and hazard quotient HQ > 10).

Heal the Bay Study, 1997. The purpose of this study was to determine if consumption of white croaker sold in community markets poses a significant health risk to consumers, despite the closure of fishing blocks immediately off the Palos Verdes Shelf. White croaker were collected from fish markets in the Chinatown, Koreatown, Monterey Park, South Bay, Westminster (including Little Saigon), and Garden Grove areas of southern California (Gold et al., 1997). In all, 132 fish tissue samples were analyzed for tDDT and PCBs. Concentrations of tDDT ranged from 19 $\mu\text{g}/\text{kg}$ to

32,600 $\mu\text{g}/\text{kg}$; total PCBs ranged from nondetectable (i.e., less than 5 $\mu\text{g}/\text{kg}$) to 1,470 $\mu\text{g}/\text{kg}$. The study concludes that contaminants pose significant risks to consumers who purchase and eat locally caught white croaker (up to 1×10^{-3} for consumption of fish muscle, assuming 50 g/day consumption of white croaker). The study also concludes that although information regarding where specific market fish were caught was unavailable, based on tDDT and PCB concentrations in some white croaker, it is highly probable that fish with significant quantities of tDDT were caught off the Palos Verdes Shelf or in the Los Angeles Harbor.

2.0 DATA EVALUATION

This section describes the fish tissue data reviewed for this study, the identification of contaminants of concern, the generation of a data set for use in the human health evaluation, a discussion of the quality of the data set, and an evaluation of spatial and temporal trends of fish tissue concentrations. General sampling locations for fish tissue data used in the risk evaluation are shown on Figure 1-3.

Numerous studies have been conducted of contaminants in seafood off the southern California coast; many of these studies collected fish tissue data from the Palos Verdes Shelf. The most recent studies are described in Section 1.2.2. A summary of existing fish tissue data for areas near the Palos Verdes Shelf is presented in Table 2-1.

2.1 Chemicals of Potential Concern

The scope of this HHRE is to evaluate risks to anglers from tDDT and PCBs in fish tissue. To confirm that these compounds are likely to be the most significant contributors to human health risks at the Palos Verdes Shelf, a comparison of maximum detected concentrations of other contaminants detected in fish tissue near the Palos Verdes Shelf to human health screening values was conducted. Maximum detected concentrations are used for this comparison to provide a conservative means of identifying chemicals of concern. In other words, if the maximum detected concentration of a chemical does not exceed the risk-based screening values, then the chemical will not pose an unacceptable risk (assuming its presence has been sufficiently characterized) and can be eliminated from further consideration in the human health risk assessment.

The intent of this comparison, however, is not to conclude that chemicals other than tDDT and PCBs in fish pose no human health risk; rather, it is to evaluate the relative significance of chemicals detected in Palos Verdes Shelf fish tissue with respect to human health risks. This step allows the streamlined risk assessment to focus its quantitative evaluation on the greatest risk contributors. The comparison of detected concentrations to screening values is shown in Table 2-2.

The following screening values were used:

- United States Food and Drug Administration (USFDA or FDA) Action Levels (USFDA, 1994);
- FDA Tolerance Levels (USFDA, 1994);
- Fish tissue concentrations used as the basis for EPA Water Quality Criteria for protection of human health (40 Code of Federal Regulations (CFR) Part 131, 57 Federal Register (FR) 60848 12/22/92);
- Screening values listed in EPA's *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories, Volume I: Fish Sampling and Analysis*, Second Edition (USEPA, 1995a).

For carcinogens (tDDT, PCBs, chlordane, HCH, and HCB), some of the screening levels were developed to correspond to a cancer risk of 1×10^{-5} (USFDA, 1994, USEPA, 1995a) while others correspond to a cancer risk of 1×10^{-6} (EPA Water Quality Criteria values). Screening levels have been modified as necessary to correspond to a cancer risk of 1×10^{-6} , consistent with risk-based screening levels published by EPA Region 9 (USEPA, 1998a).

As shown in Table 2-2, tDDT and PCBs significantly exceed human health screening values (by several orders of magnitude). In addition, these chemicals exhibit the following critical parameters that contribute to human health risk (USEPA, 1994a):

- high persistence in the aquatic environment;
- high bioaccumulation potential;
- known sources of contaminant in areas of interest;
- high potential toxicity to humans; and
- high concentrations of contaminants in previous samples of fish from the areas of interest.

Although the maximum detected concentrations of chlordane and mercury slightly exceed a risk-based screening value, tDDT and PCBs clearly account for the majority of the human health risks

due to consumption of fish near the Palos Verdes Shelf area. Therefore, tDDT and PCBs have been identified as chemicals of potential concern (COPCs) and form the basis for the quantitative evaluation in this human health risk assessment.

2.2 Selection of Fish Tissue Data for Use in the HHRE

Based on the *Santa Monica Bay Seafood Consumption Study* (SMBRP, 1994), which forms the basis for the fish consumption rates used in this human health evaluation, the fish species most commonly consumed by boat anglers were identified (see Section 4.0). These species are barred sand bass, California halibut, California scorpionfish, California sheephead, chub mackerel, halfmoon, kelp bass, Pacific barracuda, Pacific bonito, rockfishes, surfperches, and white croaker. All available muscle tissue data for tDDT and PCBs for these 12 species were reviewed, and the following procedure was used to select a data set for the HHRE:

1. Data for the 12 commonly consumed species identified above were selected. No data were available for California sheephead, therefore, a similar species (kelp bass) was used as a surrogate species to represent fish tissue concentrations for California sheephead.
2. Data from the Palos Verdes Shelf were selected. For some species, no data from the Shelf were available. In these cases, the closest available data were selected. For example, fish tissue data from the Los Angeles Harbor area were used for barred sand bass because no Palos Verdes Shelf data were available. Also, Pacific barracuda were sampled only at 14-Mile Bank (offshore of Newport Beach) while halfmoon were sampled only at Catalina Island; therefore these data were used in the HHRE.

Total DDT and total PCB data were used rather than data for individual isomers, metabolites or Aroclors. Toxicity values for DDT, DDE, DDD and PCB Aroclor mixtures are not significantly different and would not be expected to affect the outcome of risk characterization.

For a given fish species, the most recent Palos Verdes Shelf area data were selected. Older data were incorporated as necessary. Sample sizes ranged from a minimum of five samples (halfmoon) to 60 samples (kelp bass, white croaker). Although LACSD white croaker data were available for several years from 1985 to 1997, only the two most recent years of tissue data were used in the HHRE. Wherever possible, data were selected to encompass an area that is consistent with the exposure scenario described in Section 4. For example, LACSD data from Zones 1, 2, and 3 were used, when available, to represent a large area consistent with the assumption that anglers fish exclusively in this area.

The HHRE data set selected for quantification of human health risks from tDDT and PCBs is provided in Appendix A. For data collected by Pollock et al. (1991), all analytical results reported by the laboratory were used; including those below the method detection limit (38 $\mu\text{g}/\text{kg}$ for tDDT, 50 $\mu\text{g}/\text{kg}$ for PCBs). When analytical results were reported as "ND" (not detected), one-half the method detection limit was assumed.

Tables 2-3 and 2-4 summarize the HHRE data sets for tDDT and PCBs, respectively.

A normality test was conducted on the fish tissue data for each species using the Shapiro-Wilk W test. If the hypothesis that the tissue data are lognormally distributed was not rejected, a 95 percent upper confidence limit (UCL95) on the mean tissue concentration was calculated assuming a lognormal distribution. If the hypothesis was rejected (i.e., the data are not lognormally distributed), a UCL95 was calculated assuming a normal distribution.

The following equations were used to calculate the UCL95 for each fish species:

Normal Distribution:

$$UCL95 = \mu + t \frac{s}{\sqrt{n}}$$

Where:

- μ = Sample mean concentration
- t = t statistic with specified probability level (0.05) and degrees of freedom
- s = Sample standard deviation
- n = Number of samples

Lognormal Distribution:

Where:

$$UCL95 = e^{(\mu + 0.5 s^2 + s \frac{H}{\sqrt{n-1}})}$$

- e = Base of natural logarithms
- μ = Arithmetic mean of the transformed data points
- s^2 = Arithmetic sample variance of log transformed data points
- s = Arithmetic sample standard deviation of log transformed data points
- H = H statistic with specified probability level (0.05) (Gilbert, 1987)
- n = Number of samples

Although tissue data for invertebrates (e.g., yellow crab, spiny lobster) are available, human health risks from ingestion of invertebrates were not evaluated in this study. Invertebrates make up a small proportion of the annual recreational catch from this area. In addition, consumption rates are lower for invertebrates than for finfish, and tissue concentrations of tDDT and PCBs are much lower than tissue concentrations in white croaker. Therefore, human health risks from fish ingestion are believed to bound the potential risks from ingestion of invertebrates.

2.3 Data Quality

For the HHRE data set, a review was conducted to determine if the data are adequate for use in the human health risk evaluation. The review is summarized below for the LACSD data (Section 2.3.1) and the Pollock et al. (1991) data.

2.3.1 LACSD Data

Quality assurance data generated during analyses of tissue chemistry samples for chlorinated pesticides and PCBs during the period July 1991 through February 1997 were provided by LACSD (Horvath, 1998). The tissues were extracted according to EPA Method 3540 and analyzed for pesticides and PCBs according to EPA Method 8081. The method detection limits specific to biological tissues were not determined by the laboratory; however, the sample quantitation limits reported were adequate for risk assessment purposes. Precision and accuracy quality assurance (QA) data were also reported. Precision was based on relative percent differences (RPD) in duplicate analyses, and accuracy was based on matrix spike recovery percentages for selected DDT isomers and PCB Aroclors. Even though specific data quality objectives for precision and accuracy were not precisely defined by LACSD in terms of acceptable limits, over 70 percent of the QA samples had RPDs less than 30. The majority of the matrix spike recovery results was within typical recovery range of 80-100 percent, with the exception of 2,4-DDD. Information on analyses of certified reference material was not available. No specific problems with the data set were identified. Consequently, it was determined that the LACSD tissue data are adequate for risk assessment.

2.3.2 OEHHA Study Data

An external quality control program was established for the study (Pollock et al., 1991). The California Public Health Foundation (CPHF) reviewed the data collected as part of the external quality control and provided a quality control report, Appendix A-6 to the *Study of Chemical Contamination of Marine Fish from Southern California: Comprehensive Study* report. Analytical chemistry support was provided by Pacific Analytical Incorporated (PAI). Fish samples were

collected by the Southern California Coastal Water Research Project (SCCWRP). A state reference laboratory, the Hazardous Materials Laboratory (HML) of the California Department of Health Services analyzed quality control split samples. CPHF's review of the quality control data concluded that the data were sufficiently reliable for use in human health risk assessment. One issue identified during the quality control review was that measured tDDT and PCB residues were typically two to three times higher in samples analyzed by the state reference laboratory. The differences were believed to result from the use of different, but valid, methods by the two laboratories. It is possible that the results produced by PAI are negatively biased and that actual tissue concentrations are two to three times higher than reported in the study. Another issue raised was that method detection limits for PCBs may not have been low enough. This would be of concern for samples that reported nondetectable levels of PCBs. It was concluded that these data are useable for purposes of this risk evaluation. However, the issues raised in the quality control review are addressed qualitatively in the uncertainties discussion presented in Section 8.0.

2.4 Spatial and Temporal Variations in Site Data

Earlier studies have shown that bottom-dwelling fish and invertebrates collected in the area of the Palos Verdes Shelf usually have the highest muscle concentrations of tDDT and PCBs in the Southern California Bight (SMBRP, 1992). Concentrations in pelagic fish, which travel over a wider range, were more uniform. White croaker had the highest tDDT and PCB residues at every location in studies conducted during 1975 to 1990. The high levels of contamination are attributed to their high lipid content and their feeding habits.

Sufficient information was available to evaluate spatial variations in tissue concentration data on the Palos Verdes Shelf for white croaker and kelp bass, based on LACSD data collected between 1985 and 1997. Mean tissue concentrations of tDDT in white croaker and kelp bass are presented in Figures 2-1 and 2-2, respectively. It is apparent from these figures that tDDT concentrations are highest in Zone 1 (nearest to the outfall) and get progressively lower in Zones 2 and 3 with increasing distance from the outfall. Similarly, concentrations of PCBs (Figures 2-3 and 2-4, respectively) also decrease with distance from the outfall.

Temporal variations in tissue concentration data are more difficult to evaluate. Decreases in fish concentrations from 1970 to the early 1980s have been described. In a review of historical data on seafood contamination for the Southern California Bight, including the Palos Verdes Shelf, SMBRP (1992) found generally that temporal trends of contamination in fish tissue for the Bight were difficult to assess due to variability in the data. It is difficult to predict trends based on data collected during the 1985 to 1997 time period with respect to tDDT concentrations in fish collected on the

Palos Verdes Shelf. Standardization of size protocols for collection of kelp bass data was established by LACSD in 1992 and the data are collected biennially. LACSD began collecting white croaker at all three zones on an annual basis in 1992. Thus, there are not enough data that have been consistently collected over a long enough time period to be able to predict trends in future tissue contaminant levels. Concentrations of COPCs in white croaker collected in Zone 1 appear to be increasing slightly in the 11-year period shown; this may be related to changes in lipid content in the fish. Concentrations of COPCs in kelp bass appear to be decreasing.

3.0 TOXICITY ASSESSMENT

The objective of a toxicity assessment is to identify and evaluate the toxicity of the chemicals of potential concern; this is a two-step process. The first step, hazard identification, is the process of determining whether exposure to a chemical can cause an increase in the incidence of a particular adverse effect (e.g., cancer, birth defects) and whether the adverse health effect is likely to occur in humans. Further, hazard identification characterizes the nature and strength of the evidence of causation.

The second step, dose-response evaluation, is the process of quantitatively evaluating the toxicity information and characterizing the relationship between the dose of the chemical administered or received and the incidence of adverse health effects in the exposed population. From this quantitative dose-response relationship, toxicity values (e.g., reference doses [RfDs] and slope factors) are derived that can be used to estimate the potential for adverse effects as a function of human exposure to the chemical. Toxicity values are combined with daily intakes and are used to calculate human health risks through previously identified scenarios. Exposure to chemicals can result in carcinogenic or noncarcinogenic pathologies; therefore, these two categories of adverse human health effects are characterized separately. Dose-response estimates are presented as RfDs for noncarcinogenic effects (those not related to cancer) and cancer slope factors (SFs) for carcinogenic effects (cancer). Some chemicals, including DDT and PCBs, may exhibit both types of effects.

Fish consumption patterns may not correspond well to the typical periods of exposure which are studied in toxicity tests (i.e., acute or chronic exposure). Many fish consumers ingest intermittent doses of varying sizes and may consume fish over a short period of time (e.g., a vacation) or on a regular basis over a lifetime (USEPA, 1994a). The potentially large, intermittent dose (also called a "bolus dose") may be a concern for those who are particularly susceptible to toxics (e.g., children, the elderly, persons taking certain medications, pregnant or lactating women); however, it has not been evaluated in most toxicity studies. This human health risk evaluation considers risks to anglers

based on multiple exposures over a significant period of time only and does not address shorter term or intermittent exposures. In addition, subchronic exposures to breast-fed infants are evaluated in this report.

The chemicals identified as COPCs in this risk assessment are tDDT and PCBs. These are discussed in more detail below.

DDT: DDT was one of the most widely used chemicals for controlling insect pests on agricultural crops and controlling insects that carry such diseases as malaria and typhus. Technical grade DDT is a mixture of three forms (p,p'-DDT, o,p'-DDT, and o,o'-DDT), all of which are white, crystalline, tasteless, and almost odorless solids. In addition, DDD and DDE are found in small amounts as contaminants in technical-grade DDT. DDD was also used as a pesticide, and one form of DDD was used medically to treat cancer of the adrenal gland.

DDT does not occur naturally in the environment. The presence of DDT is a result of contamination due to past production and use, and subsequent movement from sites of application to land, water, and air. Agricultural use of DDT was banned by the EPA in 1972. Currently DDT is widely distributed in the environment as a result of its extensive past use and its high stability and persistence.

DDT and its metabolites are highly persistent in the environment. DDT released to water adsorbs strongly to sediments and is subject to evaporation and photooxidation near the surface. It will not hydrolyze and will not significantly biodegrade in most waters. However, biodegradation may occur in sediments. DDT and its metabolites significantly bioconcentrate in fish.

Absorption of DDT has been demonstrated following oral and dermal exposure and inhalation. The primary route of exposure, however, is the oral route. In the United States, the average amount of DDT and DDE eaten daily in food in 1981 was 2.24 micrograms per day ($\mu\text{g}/\text{day}$) (0.000032 mg/kg-day; body weight standardized ingestion rate), with root and leafy vegetables containing the highest amount (ATSDR, 1994). Meat, fish, and poultry also generally contain low levels of these compounds.

PCBs: Polychlorinated biphenyls (PCBs) are mixtures of 209 synthetic organic chemicals, or congeners. Different mixtures can take on forms ranging from oily liquids to waxy solids. Although their chemical properties vary widely, different mixtures have many common components. Because of their inflammability, chemical stability, and insulating properties, commercial PCB mixtures have been used in many industrial applications, especially in capacitors, transformers, and other electrical

equipment. These chemical properties, however, also contribute to the persistence of PCBs after they are released to the environment. Because of evidence that PCBs persist in the environment and cause harmful effects, domestic manufacture of commercial mixtures was prohibited in 1977; existing PCBs, however, remain in use.

Aroclors are technical mixtures of several different PCB congeners that are made by the partial chlorination of biphenyl in the presence of a suitable catalyst. A set of four digits is used to designate the individual Aroclors. The first two digits identify the preparation as a mixture; the third and fourth numbers identify the approximate chlorine content by weight. For example, Aroclor 1254 is a mixture with an average chlorine content of 54 percent.

In the environment, PCBs also occur as mixtures of congeners, but their composition differs from the commercial mixtures. This is because after release into the environment, the composition of PCB mixtures changes over time, through partitioning, chemical transformation, and preferential bioaccumulation. PCBs adsorb to organic materials, sediments, and soils; adsorption tends to increase with chlorine content of the PCBs and organic content of the other material (USEPA, 1996a). PCBs can volatilize or disperse as aerosols, providing an effective means of transport in the environment. Congeners with low chlorine content tend to be more volatile and also more soluble in water.

PCBs can accumulate selectively in living organisms. PCBs are highly soluble in lipids and are absorbed by fish and other animals. Rates of metabolism and elimination are slow and vary by congener. Bioaccumulation through the food chain tends to concentrate congeners of higher chlorine content, producing residues that are considerably different from the original Aroclors. Because, in general, some toxic congeners are preferentially retained, bioaccumulated PCBs appear to be more toxic than commercial PCBs (USEPA, 1996a).

PCBs are widespread in the environment, and humans are exposed through multiple pathways. Levels in air, water, soil, sediment, and foods vary over several orders of magnitude, often depending on proximity to a source of release into the environment (ATSDR, 1993).

3.1 Noncancer Health Effects

Noncancer health effects, by definition, include all adverse health impacts other than cancer. For most noncancer effects, protective mechanisms within an individual are assumed to exist that must be overcome before an adverse effect is elicited. The level above which effects may occur is referred to as a threshold level. Examples of noncancer health effects include central nervous system

disorders (e.g., neurological damage or impairment), blood disorders (e.g., anemia), organ toxicity (e.g., kidney, liver, and heart effects), reproductive toxicity (e.g., infertility), and developmental effects (e.g., birth defects, miscarriage).

In developing dose-response values for noncarcinogenic effects (i.e., the RfD), the goal is to identify the highest no-observed-adverse-effect level (NOAEL) or the lowest-observed-adverse-effect level (LOAEL) from well-designed human or animal studies. Because of the limited number of adequate human studies, NOAELs and LOAELs are typically obtained from chronic toxicity studies in laboratory animals. The NOAEL is the highest dose administered that does not produce an adverse toxic effect in the test animal. The LOAEL may be used when a NOAEL is unavailable and is the lowest dose at which an adverse effect is observed (USEPA, 1989a).

The ultimate objective of using these values is to determine the safe dose in humans, not in laboratory animals. Therefore, a number of conservative extrapolations are required. The approach used by EPA for establishing safe dose levels for chemicals to which humans may be exposed is to reduce the NOAEL by a safety or uncertainty factor that takes into consideration both the interspecies and intraspecies variations in toxicological response to chemicals. Multiple uncertainty values, ranging from 1 to 10, are typically incorporated to adjust the NOAEL or LOAEL, based on some of the following considerations (USEPA, 1989a):

- the experiment lasted less than the animal's life span;
- a dose with no adverse health effects could not be identified (i.e., a LOAEL was used instead of a NOAEL);
- data were extrapolated from a laboratory species to humans; and
- protection of sensitive individuals in the population.

An additional modifying factor may be included to reflect weaknesses in the overall toxicological database. The uncertainty factors associated with the noncarcinogenic effects of chemicals included in this risk assessment range from 100 to 300, depending on the strength of the available toxicity database for each chemical.

The general formula to derive a RfD is as follows:

$$RfD = \frac{NOAEL \text{ or } LOAEL}{(UF_1 \times UF_2 \times \dots \times UF_n \times MF)}$$

where:

RfD	=	reference dose (mg/kg-day)
NOAEL	=	no-observed-adverse-effect level (mg/kg-day)
LOAEL	=	lowest-observed-adverse-effect level (mg/kg-day)
UF _i	=	uncertainty factors (unitless)
MF	=	modifying factor (unitless)

The RfDs have been developed by the EPA Reference Dose Work Group. Most RfDs developed by EPA are for chronic (i.e., greater than 7 years) oral exposures. Recently, EPA has begun to develop RfDs for subchronic (between 2 weeks and 7 years) and acute (less than 2 weeks) exposures. However, only chronic RfDs were used in this assessment because this report is focused on long-term exposures.

RfDs for DDT and PCBs are presented in Table 3-1 with supporting information (where available) on the uncertainty factors used in deriving the RfD, critical toxic effects, and reference sources. RfDs used in this risk assessment were taken from the Integrated Risk Information System (IRIS) database (USEPA, 1998b).

3.1.1 Noncancer Toxicity of DDT

The major adverse health effects of DDT involve the nervous system, the liver, and reproduction and development of offspring. Short-term exposures to high doses of DDT primarily affect the nervous system. People who either voluntarily or accidentally ingested very high amounts of DDT experienced excitability, tremors, and seizures. Volunteers who ate 6 to 10 milligrams per kilogram (mg/kg) body weight of DDT exhibited sweating, headache, and nausea, while a dose of 16 mg/kg body weight led to convulsions. These effects on the nervous system appeared to be reversible once exposures stopped (i.e., within 24 hours). In addition, rashes or irritation of the eyes, nose, and throat were also reported. DDT is eliminated relatively slowly, with a biological half-life of about one year; DDE is eliminated much more slowly, with a biological half-life of eight years.

Long-term exposures at low doses resulted in changes in liver enzymes, but there was no indication of irreversible noncancer effects. Volunteers ingested 0.31 to 0.61 mg DDT/kg-day for up to 21 months without any noticeable effects. Tests in laboratory animals confirm the effect of DDT on the nervous system. However, tests in animals suggest that exposure to DDT may have a harmful effect on reproduction, and long-term exposure may affect the liver. An oral reference dose, or RfD,

of 5×10^{-4} mg/kg-day has been developed and for DDT is based on the occurrence of liver lesions in exposed rats (USEPA, 1998b; Laug et al., 1950).

DDT causes embryotoxicity and fetotoxicity in experimental animals (USEPA, 1994a). Biomagnification of DDT in human milk has been observed. In lactating women with an intake of 5×10^{-4} mg/kg-day of DDT, the milk contained 0.08 ppm. This was calculated to result in infant doses of 0.0112 mg/kg-day, which is approximately 20 times the dosage to the mothers (USEPA, 1994a). DDT is suspected of causing spontaneous abortion in humans and cattle (USEPA, 1994a); it is not known whether this is related to the reproductive system toxicity of DDT or developmental toxicity. The average concentration of DDE in the blood of premature babies (weighing less than 2500 grams) was significantly greater than those of higher birth weight infants.

According to the Agency for Toxic Substances and Disease Registry (ATSDR), a recent developmental study in mice found behavioral abnormalities in offspring exposed prenatally at 0.5 mg/kg-day (ATSDR, 1994). Latent effects were observed after exposure was discontinued, and subsequent tissue evaluation found structural/functional alterations in the brain. The effects, including an abnormal increase in activity and probably altered learning ability, occurred at levels approximately 50-fold lower than those which were noted in adults; the effects did not cease when tissue levels had decreased or when dosing was discontinued. This information was used to support the hypothesis of permanent structural damage in the brain. Based on the results of this study, USEPA (1994a) suggests using an exposure limit of 5×10^{-4} mg/kg-day for developmental effects of DDT, DDE, and DDD. This is the same value as the current oral reference dose listed in IRIS (USEPA, 1998b).

Intensive research is underway on potential endocrine effects of DDT and its metabolites, including laboratory studies in mammals indicating that DDT metabolites can cause abnormalities in sex development (Kelce et al., 1995, as cited in USEPA, 1996b). However, the information is currently insufficient to determine whether consideration of endocrine effects will result in changes to the RfD.

3.1.2 Noncancer Toxicity of PCBs

Liver effects and skin irritations characterized by acne-like lesions and rashes are the only significant adverse health effects reported in PCB-exposed workers. Effects of PCBs in experimental animals include liver damage, skin irritations, low birth weights and other developmental effects, immunosuppression, and death (USEPA, 1998b).

All PCB mixtures tested have caused developmental effects in experimental animals (ATSDR, 1993). Several human studies have also suggested that PCB exposure may cause adverse effects in children and to developing fetuses. A study of pregnancy outcomes in women who had consumed PCB-contaminated fish from Lake Michigan over an average of 16 years found a correlation between maternal serum PCB levels and contaminated fish consumption. The average exposure was estimated at 5×10^{-4} mg/kg-day, although the quantification of exposure was not precise. Children whose mothers were exposed had significantly lower birth weights, smaller head circumferences, shorter gestational ages, and poorer neuromuscular maturity (USEPA, 1994a). A recent study in North Carolina of the children of women with background body burdens of PCBs noted more moderate effects of PCB exposure with no changes in birth weight or head circumference (ATSDR, 1993). Both studies (Michigan, North Carolina) indicate a strong association between PCB exposure and adverse reproductive outcomes. However, both studies suffer from serious deficits regarding exposure quantification and confounding exposures to other developmental toxics.

Exposure via lactation is a significant concern for neonates (USEPA, 1994a); animal studies indicate that lactational exposure may be more significant than prenatal exposure. In monkeys, signs of PCB intoxication were observed in offspring exposed via lactation, but not in offspring exposed prenatally only (ATSDR, 1993).

It has been suggested that PCBs in the environment can mimic the body's natural hormones, and that this endocrine disruption can lead to reproductive failure, developmental disorders, and impairments to the nervous and immune systems (NIEHS, 1998). Recent studies indicate that some coplanar PCBs can elevate the cellular levels of enzymes that the body normally uses to reduce estrogen levels, resulting in reduced intracellular levels of estrogen. However, some PCBs can also inhibit these same enzymes, an activity that could increase the intracellular levels of estrogen. These effects on estrogen metabolism are not toxic endpoints per se, but they indicate potential mechanisms that may be involved to varying degrees in the suspected endocrine-disrupting effects of PCBs (NIEHS, 1998).

An oral RfD of 2×10^{-5} mg/kg-day was derived for Aroclor 1254 from monkey clinical immunological studies. The critical effects following oral administration of Aroclor 1254 in gelatin capsules for more than five years were the production of ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger- and toenails, and decreased antibody response to sheep erythrocytes (USEPA, 1998b). A total uncertainty factor of 300 was applied to the LOAEL of 0.005 mg/kg-day. EPA assigns a medium confidence level in the RfD, reflecting medium confidence in both the primary study and the database (USEPA, 1998b).

No RfDs are available for Aroclors 1242 and 1260, the other PCB mixtures which are included in the “total PCB” data. Therefore, the oral RfD for Aroclor 1254 was applied to total PCBs.

3.2 Carcinogenic Effects

In 1986, EPA issued its Guidelines for Carcinogen Risk Assessment (51FR33992), which describe the general framework to be followed in developing an analysis of carcinogenic risk. The Guidelines also identified principles to be used in evaluating the quality of data and in formulating judgments concerning the nature and magnitude of the cancer hazard from suspect carcinogens (USEPA, 1987). The following discussion is based on the information presented in the 1986 Guidelines.

The development of cancer is not believed to be governed by the same threshold concept assumed for noncarcinogenic health effects. The general theory behind cancer development is 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 clinical state of disease (i.e., cancer). For carcinogens, therefore, it is assumed that there is no level of exposure to a chemical that does not pose “a finite probability, however small, of generating a carcinogenic response” (USEPA, 1989a).

Evaluation of chemicals as to their carcinogenicity is a two-step process. Initially, the toxicity database for a substance is evaluated as to its carcinogenic potential and is assigned a weight-of-evidence classification. The weight-of-evidence classification scheme is designed to determine the likelihood of a chemical to cause cancer in humans, according to the strength of the supporting human and/or animal data. The following are weight-of-evidence classifications defined by EPA (USEPA, 1989a):

Group A: Known human carcinogen;

Group B: Probable human carcinogen;

Group B1: Limited evidence of carcinogenicity in humans;

Group B2: Sufficient evidence in animals, but inadequate evidence in humans;

Group C: Possible human carcinogen;

Group D: Not classifiable as to human carcinogenicity because of lack of data; and

Group E: Evidence of noncarcinogenicity in humans (no evidence in at least two adequate animal tests in different species or in both epidemiological and animal studies).

In the second stage of the dose-response evaluation, a slope factor is derived for chemicals with weight-of-evidence classifications A, B1 or B2, and possibly C. The slope factors are derived from one of several mathematical models developed to extrapolate from carcinogenic responses observed at the high doses used in laboratory animal experiments to responses expected at the low doses to which humans are exposed in the environment. EPA most often derives slope factors using a linearized multistage model. This predictive model generally results in more conservative dose-response values than those derived from most other models (USEPA, 1989a). The slope factor represents the 95 percent upper confidence limit of the slope of the dose response curve for oral or inhalation exposure.

Since the publication of EPA's 1986 cancer risk guidelines, scientists have gained a better understanding of the variety of ways in which carcinogens can operate. For example, some chemicals act as initiators by inducing changes in an organism's genetic code; others act as promoters by stimulating cell replication. In April 1996, EPA issued *Proposed Guidelines for Carcinogen Risk Assessment* (USEPA, 1996d) which addresses limitations in the 1986 guidelines and accommodates new information on carcinogenesis. The proposed guidelines include the following changes:

- All relevant biological information is considered. In addition to modeling tumor data, the new guidelines call for the use and modeling of other kinds of responses if they are considered to be measures of carcinogenic risk;
- A chemical's mode of action is emphasized to reduce the uncertainty in describing the likelihood of harm and in determining the dose response approach;
- A weight-of-evidence narrative replaces the current alphanumeric classification. The narrative lays out a summary of the key evidence, describes the chemical's mode of action, characterizes the conditions of hazard expression, and recommends appropriate dose response approach(es). The overall conclusion as to the likelihood of human carcinogenicity is given by route of exposure;
- Three descriptors are used for classifying human carcinogenic potential: "known/likely," "cannot be determined," and "not likely." These replace the six alphanumeric categories (A, B1, B2, C, D, E) in the 1986 guidelines; and

- Three default approaches (linear, nonlinear, or both) for dose response assessment are provided.

Once finalized, the proposed guidelines will supersede the 1986 cancer guidelines.

3.2.1 Carcinogenic Effects of DDT

Studies in animals have shown that oral exposure to DDT can result in an increased occurrence of liver tumors. In the five studies of DDT-exposed workers, results did not indicate increases in the number of deaths or cancers. However, these studies had limitations so that possible increases in cancer may not have been detected. Because DDT caused cancer in laboratory animals, it is assumed that DDT could have this effect on humans. Therefore, EPA lists DDT, DDE, and DDD as probable human carcinogens (Group B2). An oral slope factor of $0.34 \text{ (mg/kg-day)}^{-1}$ has been derived for DDT and DDE, and an oral slope factor of $0.24 \text{ (mg/kg-day)}^{-1}$ has been derived for DDD; all are based on liver tumors in rats and mice exposed via diet (USEPA, 1998b). A slope factor of 0.34 is used in this assessment to evaluate carcinogenic risks for DDT and its metabolites, as recommended in USEPA (1994a).

DDT is on the State of California's Proposition 65 list of pollutants known to cause cancer.

3.2.2 Carcinogenic Effects of PCBs

Occupational studies show some increases in cancer mortality in workers exposed to PCBs (USEPA, 1996a). Bertazzi et al. (1987) found significant excess cancer mortality at all sites combined and in the gastrointestinal tract in workers exposed to PCBs containing 54 and 42 percent chlorine. Brown (1987) found significant excess mortality from cancer of the liver, gall bladder, and biliary tract in capacitor manufacturing workers exposed to Aroclors 1254, 1252, and 1016. Sinks et al. (1992) found significant excess malignant melanoma mortality in workers exposed to Aroclors 1242 and 1016. Some other studies, however, found no increases in cancer mortality attributable to PCB exposure (ATSDR, 1993). The lack of consistency overall limits the ability to draw definitive conclusions from these studies.

A new study of rats fed diets containing Aroclors 1260, 1254, 1242, or 1016 found statistically significant, dose-related, increased incidences of liver tumors from each mixture (Brunner et al., 1996). Partial lifetime studies found precancerous liver lesions in rats and mice ingesting PCB

mixtures of high or low chlorine content. EPA lists PCBs as Group B2, probable human carcinogens.

Evaluation of the toxicity of Aroclors and other commercial PCB mixtures is complicated by numerous factors, including differences in isomer/congener/mixture composition and toxicity, differences in species susceptibility, quantitatively inconsistent data, and varying degrees of contamination with toxic chemicals, such as chlorinated dibenzofurans. In addition, there is a lack of toxicological data for some of the Aroclors (most of the studies were conducted with the higher chlorinated Aroclors), and a paucity of data for the most sensitive species (monkey and mink). Also, it should be recognized that the PCBs to which people now may be exposed may be very different from the original PCB mixture because of changes in congener and impurity composition resulting from environmental and/or biological transformation.

A recent EPA publication (EPA, 1996a) estimates slope factors for environmental mixtures of PCBs on the basis of several factors:

- Persistence and bioaccumulation through the food chain (bioaccumulated PCBs appear to be more toxic than commercial PCBs);
- Presence or absence of congeners and metabolites that contribute to cancer induction;
- Number and position of chlorines.

EPA recommends a tiered approach to selecting an appropriate cancer slope factor. The default tier uses exposure pathway to choose appropriate potency values from the ranges described in Table 3-2. The highest observed potencies from these ranges are appropriate for food chain exposure, sediment or soil ingestion, and dust or aerosol inhalation. Lower potencies are appropriate for ingestion of water-soluble congeners or inhalation of evaporated congeners. In this HHRE, a cancer slope factor of $2.0 \text{ (mg/kg-day)}^{-1}$, appropriate for food chain exposure, was used. It should be noted that this slope factor is lower than the $7.7 \text{ (mg/kg-day)}^{-1}$ slope factor in effect at the time the EE/CA work began in 1996.

PCBs are on the State of California's Proposition 65 list of pollutants known to cause cancer.

4.0 EXPOSURE ASSESSMENT

This section assesses potential human exposure to tDDT and PCBs under reasonable land/water-use scenarios for the Palos Verdes Shelf. Future land/water use is assumed to be consistent with the current use. The exposure assessment identifies receptors at risk and estimates the type and magnitude of exposures to tDDT and PCBs. The results of the exposure assessment (this section) are then combined with the chemical-specific toxicity information in Section 3 to characterize potential risks (Section 5).

The following four steps make up an exposure assessment:

- characterization of the exposure setting and receptors at risk;
- identification of exposure pathways;
- development of exposure point concentrations; and
- quantification of chemical intakes.

4.1 Characterization of the Exposure Setting

This section describes the recreational and commercial uses of the Palos Verdes area. These include commercial and recreational fishing, and various recreational activities (e.g. boating, swimming, diving).

4.1.1 Recreational and Commercial Fisheries

Recreational fishing occurring in the vicinity of the Palos Verdes Peninsula has included sport fishing and shellfishing. Between 1978 and 1984, the Palos Verdes fishery accounted for 7 percent of California's total party boat catch. Recent recreational fisheries data for Palos Verdes (catch blocks 719 and 720) indicate that chub (Pacific) mackerel (*Scomber japonicus*), rockfish (*Scorpaenidae*), ocean whitefish (*Caulolatilus princeps*), barred sand bass (*Paralabrax nebulifer*) and kelp bass (*Paralabrax clathratus*) constitute a high proportion of the sport fish catch in the Palos Verdes area (CDFG, 1996). Chub (Pacific) mackerel represent more than 20 percent of the catch in 1996. Barred sand bass and kelp bass represent 20-30 percent of the catch. Fish which contribute at least 10 percent of the sport catch include rockfish and California barracuda (*Sphyraena argentea*). White croaker represent only a small percentage (about 1 percent) of the party boat catch. Recreational fishing is also conducted by private and rental boat anglers. Data comparable to the recreational fisheries party boat data collected by the CDFG are not available for private boat anglers on the Palos Verdes Shelf. Data collected by the National Marine Fisheries Service and presented

on the Recreational Fisheries Information Network website only indicate that a white croaker fishery exists for private and rental boats in Southern California (RecFIN, 1998).

Shellfishing had been a major activity in the Whites Point area, but stocks of abalone (*Haliotis* spp.) and spiny lobster (*Panulirus interruptus*) have been depleted by overexploitation, habitat loss, and competition with sea urchins (Tetra Tech, 1989).

Fish advisories have been issued for the Palos Verdes Shelf area (see Table 1-1 and Section 1.2.1). Since consumption guidelines were released, fish tissue levels of tDDT and PCBs have not decreased (Stull, 1995). It is likely that anglers continue to consume contaminated fish from the Palos Verdes Shelf area. The *Santa Monica Bay Seafood Consumption Study* (SMBRP, 1994) found that although 77 percent of anglers that fish in Santa Monica Bay were aware of posted health warnings regarding consumption of contaminated fish, many did not consider long-term health effects to be relevant.

In 1997, approximately 20 million pounds of fish and invertebrates were taken commercially in the Palos Verdes area (catch blocks 719 or 720). Pacific sardines (*sardinops sagax*) comprised nearly 78 percent of the harvest. Other commercial catches included Pacific mackerel, Northern anchovy (*Engraulis mordax*) Pacific bonito, white seabass (*Atractoscion nobilis*), California halibut (*Paralichthys californicus*), California barracuda, and California sheephead (*Semicossyphus pulcher*). Invertebrates harvested commercially included sea urchins (*Strongylocentrotus* spp.), rock crab (*Cancer* spp.), market squid (*Loligo opalescens*), and California spiny lobster (*Panulirus interruptus*) (CDFG, 1997). In 1991, the commercial white croaker fishery on the Palos Verdes Shelf was closed based on health risk evaluations of DDT and PCB tissue levels (Section 1.2.1).

4.1.2 Recreational Activities

Recreational activities that occur within the Palos Verdes area include fishing, swimming, surfing, diving, and boating. Recreational fishing has been described in Section 4.1.1. For the most part, the Palos Verdes coastline is too rugged for good swimming, except in protected coves. Surfing occurs at various locations including Palos Verdes Point, and there are reef breaks along the Palos Verdes shoreline from Malaga Cove to Lunada Bay (Surf Report, 1986). The Palos Verdes Peninsula is known for its good diving among rock reefs and kelp beds. Boating activities tend to be greater near harbors and over fishing areas. Sailing and power boating are popular in the southern part of Santa Monica Bay. One of the principal boating areas in Santa Monica Bay is located near King Harbor and on the north side of the Palos Verdes Peninsula. Private and charter boats may also be seen in the Palos Verdes area as gray whales migrate on their way to and from Baja California.

4.2 Identification of Exposure Pathways

An exposure pathway is the mechanism through which a receptor comes in contact with contaminated media. For exposure to occur, there must be a source and mechanism of chemical release, a retention or transport medium, a point of human contact with the exposure medium, and an exposure route (i.e., ingestion, inhalation, dermal contact). If all of these elements are present, the exposure pathway is complete, and a human exposure may occur.

The most important complete exposure pathway is consumption of fish caught on the Palos Verdes Shelf. Consumption of fish by recreational boat anglers was quantitatively evaluated in this risk assessment. Pier and jetty anglers are not included in this evaluation. Although it has been suggested that fish migrate between the Palos Verdes Shelf and the Los Angeles Harbor (LACSD, 1995), a tag and recapture study or other study has not been conducted to confirm that fish caught at the Cabrillo Pier are being contaminated by Palos Verdes Shelf sediments. Subsistence fishing is not specifically addressed in this evaluation in that there is no information available which demonstrates that subsistence fishing is occurring on the Palos Verdes Shelf. This risk evaluation does not address consumption of fish at the high ingestion rates that would be representative of subsistence fish consumers.

Anglers may bring contaminated fish home, where it may be consumed by other household members, including children and women of child-bearing age. As part of the *Santa Monica Bay Seafood Consumption Study*, anglers were asked whether there were other members of their household who eat the fish they catch. Two hundred and fourteen boat anglers reported that children (ages 1 to 17) in their households eat the recreationally caught fish they bring home. Although a potential exposure pathway for children exists, it was not evaluated here. Two hundred and sixty-six boat anglers between the ages of 20 and 45 said they brought fish home to other family members over 18. Women who have consumed recreationally caught contaminated fish may expose their infants to tDDT and PCBs through their breast milk. The exposure pathway for nursing infants is evaluated in Section 7.0 of this evaluation.

Human exposures to contaminated sediment or surface waters are believed to be minimal; therefore, these exposure pathways were not evaluated in this risk assessment.

4.3 Development of Exposure Point Concentrations

Exposure point concentrations are media-specific concentrations of a contaminant that an individual may plausibly come in contact with. Once exposure pathways have been identified, exposure point concentrations are developed for each COPC in each medium (in this case, DDT and PCBs in fish tissue).

Although some fish species are migratory (e.g., Pacific bonito), others are fairly resident. It is likely that contaminant levels found in fish tissue are related to sediment concentrations of tDDT and PCBs in the area. For the purposes of this risk assessment, it was assumed that fish in the Palos Verdes Shelf area collected most of their contaminant burden either directly or indirectly, from the shelf sediments.

Individuals often consume several species of fish in their diets. The proportion of each fish species in the exposed individual's diet was estimated as described in Section 4.4, below, and an exposure point concentration was developed for each fish species considered.

The data used in this risk assessment are summarized in Tables 2-3 and 2-4. The 95 percent upper confidence limit (UCL95) on the arithmetic mean was calculated as described in Section 2.2. The lesser of the UCL95 or the maximum detected concentration was used as a reasonable maximum exposure point concentration. The UCL95 is the value that a mean, calculated repeatedly from subsamples of the population, will not exceed 95 percent of the time. Therefore, there is a 95 percent probability that the true mean of the population does not exceed the UCL95. Using the UCL95 as the exposure point concentration accounts for uncertainties in knowledge of the true concentration of contaminants at the site. However, in cases where there is a limited amount of data or extreme variability in the data, the UCL95 can be greater than the maximum detected concentration.

Exposure point concentrations were assumed to remain constant for the selected exposure duration. Temporal trends in fish tissue concentrations are difficult to assess due to variability in the data. It is expected that over some period of time fish tissue concentrations of tDDT and PCBs would decline. If these tissue concentrations do decline significantly over time, then the human health risks calculated in this study may overestimate actual risks. If however, tissue concentrations remain relatively constant, then the assumption of a constant exposure point concentration is appropriate.

Exposure point concentrations are summarized in Table 4-1.

4.4 Consumption Patterns and Rates

In 1980, a creel survey was conducted in the Los Angeles metropolitan area (including Santa Monica Bay and the Palos Verdes Shelf) to assess noncommercial fish and shellfish consumption rates by local anglers and to identify subgroups having significantly larger consumption rates (Puffer et al., 1982). During the one-year study, 1,059 anglers were interviewed at 12 sites, including piers, jetties, and party boats. Average daily consumption rates were estimated based on the number of fish in the catch, the average weight of the fish in the catch, the edible portion of the species, the number of fish eaters in the family, and the frequency of fishing per year. Only English-speaking anglers were included in the study. The median and 90th percentile rates for total noncommercial fish consumption in the Puffer study are 36.9 and 224.8 g/day, respectively.

The San Diego County Department of Health Services conducted a survey of anglers in San Diego Bay (SDCDHS, 1990) to identify the demographics of this angler population and characterize their noncommercial fish consumption patterns. Only 59 anglers were interviewed; subsets of the 59 interviews were used to calculate species and ethnic-specific rates. Due to the small number of subjects in the study population, this study was not considered for use in this human health evaluation.

The most recent comprehensive study of noncommercial fish consumption in southern California is the *Santa Monica Bay Seafood Consumption Study* (SMBRP, 1994). This study was relied upon in determining fish consumption rates and exposure duration for the receptor identified in this risk evaluation, recreational boat anglers. The *Santa Monica Bay Seafood Consumption Study* (1994) is the largest study to date of California fishers and thus provides the most comprehensive database relevant to sport fishers in California. Gassel (1997) evaluated consumption surveys for use in California and describes it as the most representative and best available dataset for estimating sport fish consumption rates among California fishers. The SMBRP conducted this study to describe the demographic characteristics of recreational anglers who eat fish collected from Santa Monica Bay, assess their noncommercial seafood consumption rates, identify ethnic subgroups that may have high rates of seafood consumption, and determine the species that are being caught and consumed at the highest rates (SMBRP, 1994). From September 1991 to August 1992, 113 surveys were conducted at 29 sites on 99 days, at locations from Point Dume to Point Fermin, although Cabrillo Pier and Cabrillo Boat Ramp were also included. Anglers on piers and jetties, private boats, party boats, and beaches were interviewed using a questionnaire; interviewers were able to administer the questionnaire in English, Spanish, and Vietnamese. The majority of the those individuals surveyed were white (43 percent), male (93 percent) and between the ages of 21-40 (54 percent). A variety of fishing modes were included in the survey and all seasons were included in the year-long study

period. Because respondents reported consumption of fish for a one-month period of time, the variability in frequency of consumption among fishers would more likely be captured than in studies using short recall periods (Gassel, 1997). Interviewers used a fillet model to help anglers describe their own consumption, in addition to methods similar to those used by Puffer et al. (1982) to derive consumption rates based on estimates of the consumable portion weights divided by the number of consumers in a household. Interviewers also used pictures of fish when fish were not in hand to facilitate correct identification of species.

The study focused on consumption of eight common species of fish, but consumption of other types of fish was also quantified. Anglers were asked to estimate how much of a species they consumed per meal, compared to a wooden model representing a 150-gram (0.33 pound) portion of a fish fillet. In addition, anglers were asked the number of times they had consumed each species of fish in the four weeks prior to the interview. This latter estimate of noncommercial fish consumption was not limited to sport fish from Santa Monica Bay, but specifically excluded fish purchased from a store. Thirty-nine percent of the respondents stated that they had eaten fish from the Bay during the four weeks prior to the interview and most anglers had caught fish on the day of the interview. Anglers who had eaten any of the eight species in the survey in the four weeks prior to the interview were included in the consumption rate estimates. Surveys were conducted of people fishing from party boats, private boats, beaches, piers and jetties. The focus of this analysis for Palos Verdes Shelf is on anglers who caught fish from boats, both private and party. Although fish may migrate into Los Angeles Harbor from the Palos Verdes Shelf and may be caught at the Cabrillo Pier, this fishing mode was not evaluated. Of the 1,243 anglers interviewed, 554 provided information that could be used for calculating consumption rates and 338 were party and private boat anglers.

Party boat anglers were surveyed on half-day fishing boats that fished within the study area. Party boats departing from Malibu, Marina Del Rey, Redondo (boat and barge) and Los Angeles Harbor were included. Interviewers went out with the party boats during their fishing trip. Private boat surveys were conducted at Marina Del Rey boat ramp, Cabrillo Boat Ramp, and King Harbor Boat Hoist as anglers returned from fishing trips. Usually only one of the anglers could be surveyed during the time the boat was at the ramp. Since it was the end of the day and anglers were heading home, it is possible that private boat anglers were not as well represented in the survey as party boat anglers. Anglers who reported fishing outside the study area were not interviewed nor included in the census.

Average daily noncommercial fish consumption rates (g/day) were calculated by multiplying the angler's estimate of a typical meal size relative to the model, by the frequency of consumption in the four weeks prior to the interview, divided by 28 days. Santa Monica Bay anglers had a median

consumption rate of 21 g/day. Boat anglers had a similar median consumption rate to all anglers of 21.4 g/day, an average consumption rate of 49 g/day and a 95th percentile consumption rate of 154 g/day.

Fish advisories issued by OEHHA (see Section 1.0) recommend, in part, that recreational anglers not consume white croaker caught on or in the vicinity of the Palos Verdes Shelf, and that anglers greatly limit consumption of a number of other species caught in the same area. Despite these fish advisories, however, recreational anglers continue to catch and consume white croaker and other species covered by the fish advisories.

Based on the studies on consumption patterns and rates in the area (described above), a RME scenario and a CT scenario were selected for quantification in the HHRE. The RME scenario is based on single species consumption rates. These rates were calculated, based only on evaluating those anglers reporting consumption of that species. Table 4-2 presents various consumption rates by fish species, including the mean, UCL95 of the mean, 90th percentile and 95th percentile and the number of boat anglers reporting consumption of that species. Consumption rates based on a conservative estimate of the mean (the UCL95 consumption rate) are used in the RME risk characterization.

White croaker consumers are of particular concern due to the high tissue concentrations that have been reported in the white croaker. Existing fish advisories for the Palos Verdes Shelf recommend zero consumption of this fish species. Data on consumption of white croaker for boat-based anglers is based on thirteen surveyed boat anglers reporting white croaker consumption; ten of the thirteen boat anglers are private boat anglers. It has been reported that white croaker is generally not preferred by party boat anglers. The CDFG party boat recreational fish catch data reflect this, in that white croaker make up about 1 percent of the fish caught from catch blocks 719 and 720 in the Palos Verdes Shelf area (CDFG, 1996). No information on white croaker catch by private boat anglers for the Palos Verdes Shelf was available. Although other fish species are consumed at higher rates in the boat angler population, consumption of white croaker represents a RME exposure because of its high tissue concentrations. The white croaker ingestion rate of 27.9 grams per day represents consumption of about six 150-gram meals per month. A 150-gram meal represents about 5 ounces or one-third of a pound of raw fish.

A central tendency scenario based on a mixed-species diet of fish was also considered, using consumption rates for all boat anglers. Diet fractions were calculated for each fish species based on individual responses to the survey. Many boat anglers surveyed reported consuming more than one fish species. The proportion of each fish species in an individual's diet was estimated and

multiplied by the median total fish consumption rate. Table 4-3 presents the derivation of these consumption rates. First the diet fractions for each individual fish species for all 338 boat anglers were averaged. These species-specific diet fractions were then multiplied by the overall median consumption rate of 21.4 g/day to derive species-specific median consumption rates for the mixed species scenario.

Exposures to nursing infants were also considered. This evaluation is presented in Section 7.

4.5 Quantification of Chemical Intakes

Chemical exposure, or intake, was determined using exposure models that combine various parameters related to behavior and physiology, such as exposure frequency and body weight, with exposure point concentrations. The RME is the highest exposure that is reasonably expected to occur at a site. The intent of the RME is to provide a conservative estimate of exposure that is well above average, yet still within the range of possible exposures. By design, the estimated RME exposures are higher than will be experienced by most individuals in an exposed population. This provides a degree of protectiveness that encompasses individuals who have a higher likelihood of exposure.

The exposure model used to calculate intake for the fish ingestion pathway consists of a simple equation that is presented below. This equation is consistent with EPA guidance (USEPA, 1989a). Intake was calculated for adults only.

Exposure to tDDT and PCBs via ingestion in fish tissue was evaluated as follows:

$$\text{Intake (mg/kg-day)} = \text{EPC } (\mu\text{g/kg}) \times \text{SIF (kg/kg-day)} \times 1 \times 10^{-3} \text{ mg}/\mu\text{g}$$

where:

Intake = Chronic daily intake (CDI) for noncancer health effects, lifetime average daily dose (LADD) for cancer

EPC = exposure point concentration

SIF = summary intake factor

The summary intake factor, SIF, was calculated as shown below:

$$SIF \left(\frac{\text{kg}}{\text{kg-day}} \right) = \frac{IR \times EF \times ED}{BW \times AT}$$

where:

- IR = ingestion rate (kg/day)
- EF = exposure frequency (days/year)
- ED = exposure duration (years)
- BW = body weight (kg)
- AT = averaging time (days)

A summary of exposure parameters used to quantify this model is presented in Table 4-4. The derivation of ingestion rates is presented in Section 4.4 above. An exposure frequency of 365 days per year is used and reflects the fact that ingestion rates are based on annual consumption. An average body weight of 70 kilograms is used which is the EPA default value for adults (USEPA, 1991). Averaging time for cancer risk is equal to the average lifetime exposure duration of 70 years (USEPA, 1991). Averaging time for noncancer outcomes is equal to exposure duration, which is based on values for reported duration taken from the *Santa Monica Bay Seafood Consumption Study*. One of the questions asked of the survey respondents was how many years they had fished in the Santa Monica Bay. The responses of the 338 boat anglers in the study were compiled and mean and 90th percentile values calculated. The mean reported number of years fished is 13.8 years and the 90th percentile is 30 years. These values may underestimate total duration in that the numbers do not reflect those anglers who continue to fish after the survey was conducted. Calculated daily intakes based on the parameters presented here are presented in Table 4-5.

5.0 HUMAN HEALTH RISK CHARACTERIZATION

Risk characterization integrates the results of the exposure and toxicity assessments by combining estimates of intake with toxicity data to determine the likelihood of adverse effects in potentially exposed populations. Because of fundamental differences in the mechanisms through which carcinogenic and noncarcinogenic processes occur, risks are characterized separately for these two types of health effects.

This section presents point estimate risk results for two scenarios: a RME scenario for each fish species assuming a single-species diet, and a CT scenario assuming a mixed-species diet. In

addition, a quantitative evaluation of the uncertainty and variability in the risk estimates using Monte Carlo analysis was performed and is presented in Section 6.0.

5.1 Cancer Risk

Potential health risks associated with carcinogens were estimated by calculating the increased probability of an individual developing cancer during his or her lifetime as a result of exposure to a carcinogenic compound. Excess lifetime cancer risks were computed by combining estimated chemical intakes (or lifetime average daily dose) and available dose-response information in the following equation:

$$\text{Cancer Risk} = \text{LADD} \times \text{SF}$$

where:

LADD = daily chemical intake averaged over a lifetime of 70 years (mg/kg-day)

SF = oral cancer slope factor (mg/kg-day)⁻¹

Resulting cancer risk estimates represent incremental or excess lifetime cancer risks due to exposures to site contaminants. The observed national cancer incidence rate is approximately one in four, i.e., 25 percent of the United States population. An excess cancer risk of 2×10^{-5} represents a probability of 2 excess cases of cancer per 100,000 exposed individuals. Unlike noncarcinogenic effects, any exposure to a carcinogenic substance is assumed to be associated with some degree of risk. For this reason, EPA uses an excess cancer risk of 10^{-6} (one in 1,000,000) as the point of departure for cancer risk estimates that are of concern. EPA uses an acceptable risk range of 10^{-4} to 10^{-6} to determine whether a site poses a risk to human health (40 CFR §300.430).

5.2 Noncancer Hazard

The potential for adverse health effects other than cancer was evaluated by comparing the average daily intake of a chemical over a specified exposure period with the reference dose appropriate to that exposure period (i.e., subchronic or chronic; only chronic values were used in this risk assessment). An RfD is a dose below which no adverse health effects are expected to occur (USEPA, 1989a). The comparison is expressed as a HQ, the ratio of the chemical intake to the RfD:

$$HQ = \frac{CDI}{RfD}$$

where:

CDI = chronic daily intake of a contaminant (mg/kg-day)

RfD = oral reference dose (mg/kg-day)

An HQ is not a prediction of the probability or severity of effects, but rather a ratio that indicates whether the estimated exposure presents a potential threat to human health (USEPA, 1989a). When the CDI of a chemical exceeds the reference dose (i.e., $HQ > 1$), the potential for noncancer health effects exists (USEPA, 1989a).

Noncancer health effects are expected to be cumulative only when the toxic endpoints of each chemical are similar. Because the RfD for DDT is based on liver effects, while the RfD for PCBs is based on ocular (eye) effects, HQs were not summed across COPCs.

5.3 Point Estimate Risk Results

Cancer risk and noncancer hazard quotients were calculated for the RME (single-species diet) and CT (mixed-species diet) scenarios. Results of the point estimate risk calculations are presented in Tables 5-1 and 5-2.

The RME scenario represents the potential risks to boat anglers who consume a particular species of fish collected from the Palos Verdes Shelf, assuming mean tissue concentrations and consumption rates (as represented by the 95 percent upper confidence limit on the mean). Cancer risks exceed 1×10^{-4} for consumers of the following fish species: white croaker (2×10^{-3}) and surfperches (2×10^{-4}). Total DDT contributes about two-thirds of the cancer risk for consumption of white croaker. RME cancer risks are presented in Table 5-1 and Figure 5-1.

Consumption of several species of fish resulted in a potential noncancer hazard for the RME scenario (Table 5-1; Figure 5-2). These species are white croaker (HQ=32), surfperches (HQ=5), barred sand bass (HQ=3), California halibut (HQ=3), California sheephead (HQ=2), and kelp bass (HQ=2).

This scenario reflects consumption of a single species of fish using a conservative estimate of the mean consumption rate (i.e., the UCL95) for that species. It should be noted, however, that boat anglers generally do not consume only a single species of fish. For example, since the UCL95 on the mean total fish consumption rate (i.e., all species) is 53.0 g/day, a consumer of white croaker (at the RME consumption rate of 27.9 g/day) may also be consuming a variety of other fish species. The contribution of tDDT and PCBs in these other fish species to human health risk is not reflected in the RME results.

The CT scenario represents the potential risk to boat anglers who consume a mixed-species diet of fish collected from the Palos Verdes Shelf, assuming arithmetic mean tissue concentrations and median consumption rates for all boat anglers (rather than for consumers of a particular species). The total cancer risk (tDDT and PCBs combined) for anglers who fish from boats (mixed-species diet) is 2×10^{-5} . The noncancer HQs are 0.3 and 0.9 for tDDT and PCBs, respectively. These HQs indicate that noncancer health hazards are not likely to occur. CT point estimate cancer risks and noncancer hazard results are presented in Table 5-2.

5.4 Comparison of Point Estimate Risk Results to Previous Studies

Point estimate results for cancer risk and noncancer hazard developed in this HHRE were compared to results of previous risk assessments conducted for fish consumption from the Palos Verdes Shelf area. This comparison is presented in Table 5-3. For a single species diet consisting of white croaker, cancer risk results are very similar, generally in the range of 1×10^{-3} to 2×10^{-3} . Potential risks from consumption of commercially sold white croaker are also comparable for whole fish, with slightly lower cancer risk calculated for fillets. Noncancer hazard quotients are consistent, ranging from 10 to 17 for tDDT and 17 to 32 for PCBs.

Point estimate cancer risk results for the mixed-species (or CT) scenario in this HHRE are about an order of magnitude lower than in previous risk assessments; the difference is due primarily to the much lower white croaker consumption rate assumed for the mixed species diet in the current study. Noncancer hazard quotients are similar.

In summary, human health risk results of this HHRE are consistent with previous risk assessments conducted for fish consumption in the Palos Verdes Shelf area.

6.0 MONTE CARLO ANALYSIS

This section summarizes the results of a Monte Carlo analysis of the risks associated with the consumption of white croaker by recreational boat anglers. The Monte Carlo analysis was performed for the RME scenario (i.e., consumption of a single fish species). White croaker is by far the largest contributor to cancer risk and noncancer hazard, and therefore was selected for evaluation using the Monte Carlo technique.

The basic goal of a Monte Carlo simulation is to characterize, quantitatively, the uncertainty and variability in estimates of risk. A secondary goal is to identify key sources of variability and uncertainty and to quantify the relative contribution of these sources to the overall variance and

range of model results. The outcome of the Monte Carlo analysis provides additional information to be used, along with the results of the standard point estimate approach, in making risk management decisions.

As noted in EPA's 1997 policy on the use of probabilistic analysis in risk assessment (USEPA, 1997b), techniques, such as Monte Carlo analysis, given adequate supporting data and credible assumptions, can be viable statistical tools for analyzing variability and uncertainty in risk assessments. EPA has also issued *Guiding Principles for Monte Carlo Analysis* (USEPA, 1997c), which provides principles of good practice in the use of Monte Carlo analysis. Both of these documents have been followed in conducting this Monte Carlo analysis.

The term "uncertainty," as used in this report, refers to both variability and true uncertainty. Variability is the heterogeneity in the parameter of interest (e.g., contaminant concentrations in fish tissue), while uncertainty is the incomplete understanding of the true value of a particular variable or its heterogeneity. Additional data collection will typically reduce uncertainty, but will not affect variability.

Although the Monte Carlo simulation process is internally complex, commercial software (such as the Crystal Ball™ software used in this analysis) performs the calculations as a single operation, presenting results in simple graphs and tables. These results approximate the full range of possible outcomes and the likelihood of each.

Monte Carlo techniques have some important limitations, as described below:

- Available software cannot distinguish between variability and uncertainty. Current Monte Carlo software treats uncertainty as if it were variability, which may produce results that are difficult to interpret and use;
- Information on correlations between variables is seldom available. Ignoring these correlations, however, can bias Monte Carlo calculations;
- Distributions for exposure factors developed from short-term studies with large populations may not accurately represent long-term conditions in smaller populations; and
- The tails of Monte Carlo risk distributions, which are of greatest regulatory interest, are very sensitive to the shape of the input distributions.

6.1 Methodology

The Monte Carlo simulation consists of the following tasks:

- selection of exposure pathways or scenarios for quantification;
- selection of input variable distributions;
- performance of Monte Carlo simulations; and
- presentation of results and conclusions.

6.1.1 Selection of Exposure Pathways/Scenarios

A single-species diet involving the consumption of white croaker by Palos Verdes Shelf boat anglers was evaluated for this Monte Carlo analysis, consistent with the most significant contributor to risk identified by the point estimate risk calculations. The analysis focuses on exposures to recreational boat anglers. The following equations were used to calculate risk:

$$\text{Cancer Risk} = \text{Intake} \times \text{SF}$$

$$\text{HQ} = \frac{\text{Intake}}{\text{RfD}}$$

where:

Intake = average chronic daily intake (CDI) or lifetime average daily dose (LADD) of a contaminant (mg/kg-day)

$$\text{Intake} = \frac{\text{EPC} \times \text{IR} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

SF = cancer slope factor (mg/kg-day)⁻¹

RfD = oral reference dose (mg/kg-day)

EPC = exposure point concentration (mg/kg)

IR = ingestion rate (g/day)

EF = exposure frequency (days/year)

ED = exposure duration (years)

CF = conversion factor (kg/g)

BW = body weight (kg)

AT = averaging time (days)

6.1.2 Selection of Input Variable Distributions

A mix of point estimates and probability distributions for exposure variables was used in the Monte Carlo simulation. The exposure point concentration, ingestion rate, exposure duration, and body weight were defined by probability density functions (PDFs) rather than discrete values. The PDFs are based on site-specific data, published information, or professional judgment. The distributions and sources of these input variables are presented in Table 6-1.

Point estimate values were used for exposure frequency and averaging time. Exposure frequency is set at 365 days/year because the fish consumption rates are average daily rates of consumption measured during both summer and nonsummer months (SMBRP, 1994). Averaging time is defined as lifetime for cancer risks (70 years or 25,550 days) and equal to the exposure duration for noncancer hazard. For the estimation of noncancer hazard, exposure duration (in the numerator) and averaging time (in the denominator) are equal and thus cancel each other out. Therefore, the selected exposure duration has no effect on the noncancer hazard estimates.

PDFs for exposure point concentrations were developed based on site-specific data. First a normality test was conducted on the white croaker tissue data using the Shapiro-Wilk W test. Because the hypothesis that the tissue data are lognormally distributed was not rejected, the curve-fitting feature of Crystal Ball™ was used to fit a lognormal distribution to the tissue concentration data.

Fish tissue data collected by LACSD for the purposes of NPDES permit compliance are the most recently collected existing data for white croaker. The LACSD data from 1996 and 1997 were used in this Monte Carlo analysis; these data are presented in Appendix A. Tissue concentrations of tDDT and PCBs in white croaker were examined and found to be highly correlated (i.e., fish with high tDDT concentrations also had high PCB concentrations). A correlation coefficient of 0.96 was applied in the Monte Carlo analysis.

Fish consumption rates for the 13 boat anglers who reported consuming white croaker, as presented in data from the *Santa Monica Bay Seafood Consumption Study* (SMBRP, 1994), were used to define a lognormal distribution for consumption of white croaker. The mean consumption rate of white croaker is 16.7 g/day, with a standard deviation of 13 g/day. This corresponds to about three 150-gram meals of white croaker per month.

An empirical distribution was developed for exposure duration, using the reported fishing durations for boat anglers from the *Santa Monica Bay Seafood Consumption Study* (SMBRP, 1994). Reported fishing duration, as reported in the Consumption Study, reflects only the length of time the surveyed individuals had been fishing up to the time of the survey. Because no information is available on how long these individuals will continue to fish in the future, the reported fishing duration is not equivalent to total exposure duration. Several approaches have been proposed to relate reported duration to total exposure duration (Israeli and Nelson 1992, Price et al., 1998). A case study for the Price et al. approach, which evaluated reported and total durations for party boat and pier anglers in the Santa Monica Bay, found that although the mean total duration of the surveyed anglers was significantly higher than the mean reported duration, when corrected for a longevity bias (i.e., a bias towards sampling individuals who practice a behavior for a longer period of time) the mean total duration was similar to the mean reported duration. This relationship was also observed at the 90th and 95th percentiles of the distributions. Therefore, the reported durations for boat anglers from the *Santa Monica Bay Seafood Consumption Study* were used in this HHRE to represent total exposure duration. An empirical distribution was used rather than a lognormal or normal distribution because the duration data did not meet the criteria for these distributions, using the Shapiro-Wilk W test. The mean reported fishing duration is 13.8 years; the 90th percentile is 30 years.

The parameters for a lognormal distribution of body weight were based on literature values for the U.S. adult population. Toxicity values (i.e., slope factors and reference doses) were entered as point values. No other correlations between input variables (besides the tissue concentration correlation described above) were assumed.

6.2 Results of Monte Carlo Simulations

Monte Carlo simulations were conducted using Crystal Ball™ by Decisioneering, Inc. Results were based on 10,000 iterations using Latin Hypercube sampling (100 divisions) and a burst mode option of 5.

Detailed results of the Monte Carlo simulations are presented in Appendix B, including summary statistics for each simulation, percentiles of cancer risk and noncancer hazard, output frequency and cumulative distribution charts, and input assumption distributions. Figures 6-1 through 6-3 present the output frequency distribution graphs for cancer risk and noncancer hazard to boat anglers who consume white croaker. The figures show the results as both a PDF and a cumulative distribution function (CDF). The mean value shown on the frequency distribution graphs is the average of all output values generated by the Monte Carlo simulation model. The x-axis represents excess cancer risk (i.e., the probability that an individual exposed under the assumed conditions will develop

cancer during his or her lifetime as a result of exposures to site contaminants) in Figure 6-1, and the hazard quotient in Figures 6-2 and 6-3.

A PDF shows the number of values occurring in a given interval. A CDF shows the number or percentage of values greater than or equal to a given value. Percentiles, or cumulative percentiles, correspond to specific probabilities that the variable will be less than or equal to. For example, there is a 90 percent probability that cancer risk is less than the 90th percentile value, given the specified input assumptions. The cumulative percentiles plot together to form the CDF (Vose, 1996).

The graphs also indicate the number of Monte Carlo simulations performed (10,000) and the number of "outliers," that is, values generated during the simulation that are not shown on the plot. "Outliers" are defined in this analysis as being more than two standard deviations from the mean; the range of values shown on the graphs includes about 96 percent of the simulation output values.

Sensitivity studies were performed to identify those input parameters that represent the greatest contributors to variance in the cancer risk and noncancer hazard for recreational boat anglers consuming white croaker. Results of the sensitivity analyses are shown in Figures 6-4 through 6-6.

The results of the Monte Carlo analysis are summarized below.

6.2.1 Cancer Risk Results

Table 6-2 presents the mean, 50th, 90th, 95th percentile, and point estimate cancer risks. The mean, median, and 95th percentile cancer risks are 3×10^{-4} , 8×10^{-5} , and 1×10^{-3} , respectively. About 45 percent of simulation results were above 1×10^{-4} (i.e., a cancer risk of 1×10^{-4} corresponds to approximately a 55th percentile of the output distribution). The point estimate cancer risk value of 2×10^{-3} corresponds to about a 97th percentile of the distribution of cancer risk. At the 95th percentile level, exposures to tDDT represent about two-thirds of the cancer risk; exposures to PCBs represent about one-third.

Results of sensitivity analyses (shown in Figure 6-4) indicate that the exposure duration is the largest contributor to variance in the cancer risk results, followed by tDDT concentration in white croaker and PCB concentration in white croaker. Exposure duration and fish tissue concentrations represent both natural variability in a population and uncertainty (e.g., the use of reported fishing duration to represent exposure duration, and the imprecision in measurement of tissue concentrations).

6.2.2 Noncancer Hazard

Table 6-2 presents the mean, 50th, 90th, and 95th percentile, and point estimate hazard quotients for exposures to tDDT and PCBs, respectively. The mean, median, and 95th percentile hazard quotients for exposures to tDDT (7, 3, and 26, respectively) are greater than 1, the level at which there may be a concern for potential noncancer health effects. About 75 percent of simulation results for noncancer hazard from tDDT exceeded a hazard quotient of 1 (i.e., a hazard quotient of 1 corresponds to approximately a 25th percentile of the output distribution). The point estimate hazard quotient for tDDT of 17 corresponds to a 91st percentile of the tDDT hazard quotient distribution.

For exposures to PCBs, the mean, median, and 95th percentile values are all greater than 1 (14, 7, and 52, respectively). About 95 percent of simulation results for noncancer hazard from PCBs exceeded a hazard quotient of 1 (i.e., a hazard quotient of 1 corresponds to approximately a 5th percentile of the output distribution). The point estimate hazard quotient for PCBs of 32 corresponds to a 90th percentile of the PCBs hazard quotient distribution.

Results of sensitivity analyses (shown in Figures 6-5 and 6-6) indicate that the tissue concentrations of tDDT and PCBs are the most significant contributors to variance in the results. The ingestion rate of white croaker is also a significant contributor to variance. Ingestion rates and fish tissue concentrations represent both natural variability in a population and uncertainty (e.g., the imprecision in measurement of tissue concentrations and estimation of fish ingestion rates).

6.3 Summary

A Monte Carlo analysis of the risks associated with the consumption of white croaker by recreational boat anglers was conducted. The goal of the Monte Carlo analysis was to characterize, quantitatively, the uncertainty and variability in estimates of risk. A secondary goal was to identify key sources of variability and uncertainty and to quantify the relative contribution of these sources to the overall variance and range of model results. Results of the simulations indicate that mean, median, 90th percentile, and 95th percentile cancer risks exceed 1×10^{-4} . The point estimate cancer risk of 2×10^{-3} corresponds to about a 97th percentile of the Monte Carlo output distribution.

Mean, median, 90th percentile, and 95th percentile noncancer hazard quotients for exposures to tDDT and PCBs in fish exceed a value of 1. Point estimate hazard quotients for both tDDT and PCBs correspond to about a 90th percentile of the Monte Carlo output distributions

Exposure duration and white croaker tissue concentrations of tDDT and PCBs were the most significant sources of variability/uncertainty in the cancer risk results. Tissue concentrations of tDDT and PCBs and the consumption rate of white croaker were the most significant sources of variability/uncertainty in the noncancer hazard results.

The probabilistic analysis performed here addresses variability and uncertainty in the exposure analysis only. Many potentially significant sources of uncertainty were not addressed, such as uncertainty in the toxicity of chemicals to humans. There is little quantified data available to develop distributions for toxicity variables, and the uncertainty associated with toxicity factors used in the risk calculations may be large. Use of point estimate toxicity values may contribute to an overestimation of risk.

7.0 RISKS TO NURSING INFANTS

7.1 Breast Milk Consumption Exposure Pathway

An evaluation of the potential risks to breast-fed infants due to consumption of tDDT and PCBs in breast milk was performed. Breast milk consumption can be an important exposure route for nursing infants to contaminants like DDT and PCBs that biomagnify and become concentrated in human adipose tissue and breast milk fat. Lipid-soluble, poorly metabolized chemical compounds, such as tDDT and PCBs, accumulate in body fat and may be transferred to breast-fed infants in the lipid portion of breast milk. These chemicals remain in adipose tissue and are only very slowly eliminated except during lactation (OEHHA, 1996). These chemicals appear to be in equilibrium with adipose tissue levels, and over time a significant portion of the maternal load may be transferred to the breast-fed infant (OEHHA, 1996). Evidence for the contamination of human milk by DDT metabolites and PCBs has been demonstrated in several studies: a national study conducted in 1975-1976 (Savage, 1977); and a study conducted in Hawaii during 1979 (Takei, 1983). Mean residues in parts per million (ppm) on a lipid basis for p,p'-DDE were similar in the two studies (1.9 ppm national; 2 ppm Hawaii). Residues for PCBs were also similar (0.80 ppm national; 0.97 ppm Hawaii).

Most American newborns are breast-fed. Breast-feeding patterns vary with maternal age and education, race/ethnicity and economic status, with the highest prevalence of breast-feeding occurring among college educated, higher income Caucasian women ages thirty and above (OEHHA, 1996). However, large increases in breast-feeding have occurred in recent years in populations where breast-feeding was among the lowest.

Infants are particularly vulnerable because they obtain most, if not all, of their dietary intake from breast milk. Estimating the magnitude of the potential risk to infants from breast milk requires information on the concentration of contaminants in breast milk, the quantity of breast milk consumed per day, the duration over which breast-feeding occurs, and the fat content of breast milk.

The equations used to quantify the breast milk pathway are presented in OEHHA's *Technical Support Document for Exposure Assessment and Stochastic Analysis* (OEHHA, 1996). The methodology is based on that presented in Smith (1987) and is consistent with the methodology used by EPA in the *Addendum to the Methodology for Assessing Risks Associated with Indirect Exposure to Combustor Emissions* (USEPA, 1993b).

Parameters used in this evaluation are from EPA's *Exposure Factors Handbook* (USEPA, 1997a), EPA's *Guidelines for Assessing Chemical Contaminant Data for Use in Fish Advisories* (USEPA, 1994a), Smith 1987, Butte 1984, and the OEHHA document. The following equations were used to calculate a breast-fed infant's average daily intake of contaminants:

$$\text{Daily Intake}_{\text{Infant}} \left(\frac{\text{mg}}{\text{kg}} \text{-day} \right) = \frac{C_{\text{milk}} \times IR_{\text{milk}} \times EF_{\text{Infant}} \times ED_{\text{Infant}} \times CF}{AT}$$

where:

C_{milk} = concentration of contaminant in breast milk (mg/kg)

$$= \text{Daily Intake}_{\text{maternal}} \times t_{1/2} \times f_1 \times \left(\frac{f_3}{[f_2 \times 0.693]} \right)$$

$\text{Daily Intake}_{\text{maternal}}$ = average maternal intake (calculated as the contaminant concentration in fish x maternal ingestion rate/body weight; mg/kg-day)

$t_{1/2}$ = half-life of a contaminant in the mother's body (days)

f_1 = fraction of contaminant that partitions to mother's fat

f_2 = fraction of mother's weight that is fat

f_3 = fraction of milk that is fat

IR_{milk} = infant's milk ingestion rate per body weight (g/kg-day)

EF_{Infant} = infant's exposure frequency

ED_{Infant} = infant's exposure duration

CF	=	conversion factor (kg/g)
AT	=	averaging time

7.2 Contaminant Concentrations in Breast Milk

There is no direct evidence available to substantiate that women of child-bearing age consume contaminated fish caught from the Palos Verdes Shelf and then nurse their infants. Only ten percent of respondents in the *Santa Monica Bay Seafood Consumption Study* were female. However, boat anglers did report taking fish home to their family members, both adults and children, where the adult portions were similar to those of the anglers themselves. There clearly is a potential for a complete exposure pathway to exist. A maternal ingestion rate equal to consumption of one 150-gram meal per month was used in the quantification of risk. This seems like a reasonable estimate of consumption, but may underestimate the actual ingestion rate. A 150-gram meal per month is equivalent to just under 5 grams of fish per day or one-third pound of fish per month. The median consumption rate of 21.4 grams per day for anglers surveyed in the SMBRP study is equivalent to consumption of 1.4 pounds of fish per month or four times the rate used in this evaluation.

Table 7-1 presents the parameters for the calculation of contaminant levels in maternal breast milk. The parameters presented reflect those variables influencing the partitioning of contaminants to breast milk lipid. Under these assumptions, the calculated contaminant concentration in breast milk due to consumption of contaminated white croaker is 0.8 mg/kg (for tDDT) and 0.05 mg/kg (for PCBs). Breast milk contaminant levels with a kelp bass scenario are 0.01 mg/kg for tDDT and 0.002 mg/kg for PCBs. These predicted breast milk levels represent a two to three order of magnitude biomagnification of the levels ingested in the diet. These levels are lower than the 2 and 1.9 mg/kg mean DDE and 0.97 and 0.8 mean PCB values reported in the national and Hawaiian studies from data collected during the 1970s (Savage 1977; Takei 1983).

7.3 Calculating Daily Intake for the Nursing Infant

Table 7-2 presents the quantification of the intake to the nursing infant based on the breast milk contaminant levels described above. The exposure duration used in this analysis is one year. Most American infants are weaned during the first year, although a portion of the population will wean infants well beyond a year. Breast milk ingestion rates are reviewed in OEHHA (1996). Their recommendations for point estimate intake rates of breast milk for breast-fed children during the first year of life are a mean of 102 g/kg-day for a central tendency estimate and 138 g/kg-day for a 95th percentile or RME estimate. The 95th percentile estimate was used here since the dominant pathway being evaluated for infants is breast milk consumption.

7.4 Quantification of Risk to Nursing Infants

Based on the assumptions described above and a white croaker consumption scenario, the noncancer hazard quotient for ingestion of tDDT by a breast-feeding infant is 220; for ingestion of PCBs the hazard quotient is 370. These results are presented in Table 7-3. Kelp bass hazard quotients exceed 1 for both tDDT and PCBs.

Lower fish ingestion rates by the mother would result in correspondingly lower estimated risks to the breast-feeding infant (e.g., a two-fold reduction in fish ingestion rate would result in a two-fold reduction in risk to the infant). Consumption at the median rate of 21.4 grams per day would result in a four-fold increase in risks. Consumption at a very low rate of fish ingestion from the Palos Verdes Shelf (e.g., one serving of white croaker per year or 0.4 g/day) would still result in a hazard quotient that exceeds 1. Hazard quotients at this rate of consumption would be 18 for tDDT and 30 for PCBs.

The simple model used for this preliminary evaluation contains a number of uncertainties. A tendency towards overestimation of the daily intake to the infant is caused by the assumption that reductions do not occur in maternal fat levels during breast feeding. Sullivan et al. (1991) estimated that the steady-state assumption may lead to overestimates of 20 percent.

8.0 QUALITATIVE UNCERTAINTY ANALYSIS

This uncertainty analysis provides a qualitative and, where possible, semiquantitative evaluation of the assumptions and limitations inherent in each step of the risk assessment process and their effects on the overall risks calculated for the site, particularly those uncertainties not addressed as part of the Monte Carlo analysis (Section 6). For each identified source of uncertainty, the direction and magnitude of the potential effect on the risk estimate and the steps taken to mitigate uncertainties are noted. In many cases, the only possible steps to mitigate uncertainties are use of professional judgment and best available data. Uncertainties in risk assessment are most often dealt with by using a conservative approach, resulting in a tendency to overestimate risks. This section describes the uncertainties associated with each step of the risk assessment process, including data evaluation, exposure assessment, toxicity assessment, and risk characterization.

8.1 Uncertainty in Site Data

There are many uncertainties associated with obtaining and evaluating data for use in risk assessment that are not specifically addressed here. Some of these uncertainties are associated with developing

the sampling plan, reviewing analytes detected at the site, and validation of site data. Because fish tissue data were collected by other agencies and as part of other studies, uncertainties related to sample collection and quality assurance of the data could not be comprehensively assessed. The data are believed to be acceptable for use in this risk assessment.

Recent fish tissue data for some of the fish species included in this evaluation were not available. Data for some species was up to 10 years old (e.g., Pacific bonito, chub mackerel, and rockfishes). Risks calculated using these data represent conditions present at the time the samples were collected, not the risks that may currently exist. On the other hand, because it does not appear that tissue concentrations of tDDT and PCBs are decreasing significantly with time, the uncertainty associated with the use of old fish tissue data is low.

Data collected by Pollock et al. (1991) for the OEHHA study may be negatively biased and actual tissue concentrations may have been two to three times higher than reported in the study. Risk estimates for consumption of fish species which relied on Pollock et al. data would be two to three times higher if the data are negatively biased. This would not impact the overall conclusions of the risk evaluation. The uncertainty associated with using the Pollock et al. data is therefore considered low.

In addition, for some species, very few samples were available from the area of concern (i.e., the Palos Verdes Shelf). Only five halfmoon and Pacific barracuda samples, and only 10 barred sand bass and California halibut samples were available. Small sample size has resulted in relatively high variance in the tissue data; for example, tDDT concentrations in halibut range from 35 to 1,360 $\mu\text{g}/\text{kg}$, resulting in a mean tissue concentration of 460 $\mu\text{g}/\text{kg}$ with a 95 percent upper confidence limit on the mean of 1,660 $\mu\text{g}/\text{kg}$. In this case, the maximum detected concentration was used as the exposure point concentration. The uncertainty associated with small sample sizes is moderate, particularly for California halibut.

No tissue data were available for California sheephead, and therefore tissue concentrations for a surrogate species (kelp bass) were used. In addition, exposures to contaminants in other species that may potentially be consumed were included in the risk evaluation by averaging tissue concentrations for the included species, with the exception of white croaker and California sheephead. This could result in an over or underestimation of the risks associated with consumption of this species.

The risk assessment focused strictly on tDDT and PCBs. Exclusion of other chemicals detected in Palos Verdes Shelf fish tissue could result in a low to moderate underestimation of risk.

8.2 Uncertainty in the Exposure Assessment

A number of uncertainties are associated with assumptions made for the exposure assessment. Areas of uncertainty include identification of receptors and exposure pathways, calculation of exposure point concentrations and intakes, and selection of exposure parameters.

The risk evaluation was limited to fish consumption by boat anglers. Although exposures to sediment and surface water may also occur, they are believed to be minimal in comparison to exposures from fish ingestion. Elimination of sediment and surface water pathways may result in minimal underestimation of risks. Although anglers may consume contaminated fish using fishing modes other than boating (e.g., piers, beaches), these scenarios were not evaluated because no piers are located directly on the Palos Verdes Shelf. While unacceptable risks to anglers at Cabrillo Pier may be present, it is not known whether fish at Cabrillo Pier are being contaminated by Palos Verdes Shelf sediments.

The assumption that fish tissue concentrations in fish collected near the Palos Verdes Shelf are the result of exposure to contaminants in shelf sediments is a source of uncertainty, particularly for fish that exhibit a wide range of movement such as chub mackerel.

Uncertainties regarding exposure assumptions stem from the natural variabilities of the different parameters, such as body weight or fish ingestion rate, as well as from insufficient data on the distribution of these parameters. These uncertainties were evaluated as part of the Monte Carlo analysis (Section 6).

An individual consuming extremely large amounts of seafood (e.g., commercial fishermen or ethnic subpopulations) may consume large amounts of a single species depending on personal preferences. Overall risks for heavy consumers, therefore, may be greater than the estimated risks using mean consumption rates for a particular species (i.e., the RME scenario), as presented in this HHRE.

Fish consumption rates are expressed in this HHRE in terms of number of meals per month or year. A 150-gram meal size was used as the standard meal size; this is consistent with the size of the fillet model used during the *Santa Monica Bay Seafood Consumption Study*. However, the median meal size reported by the 338 boat anglers for which fish consumption rates were calculated is 225 grams, with an average reported meal size of 290 grams. Therefore, assuming a meal size of 225 grams, the RME white croaker consumption rate of 27.9 grams/day corresponds to less than 4 meals per month, rather than 6 meals per month as presented in Section 4.4. In addition, a consumption rate

of one 150-gram meal per month was used to quantify risks from the breast milk pathway; if a meal size of 225 grams were used in this analysis, calculated health hazards would be 50 percent greater.

No attempt was made in this study to quantitatively evaluate the effects of fish preparation methods on human health risks; this may result in an overestimate of risk. Contaminant burdens in fish may decrease by 10 to 70 percent depending on how the fish is prepared and cooked (USEPA 1993c).

PCBs and tDDT are stored mainly in the fat of the fish (i.e., they are lipophilic), located along the back and the belly, and in the dark meat along the lateral line running along the side of the fish. Skinning fish will remove the thin layer of fat under the skin. Contaminant fish tissue data used in this human health evaluation is for raw, skin-off fillets. Therefore, a reduction in contaminant concentrations due to skinning and trimming is not applicable to this study.

The HHRE uses concentrations of tDDT and PCBs in fish fillets to calculate potential risks to human health from consumption of fish. However, the *Santa Monica Bay Seafood Consumption Study* (SMBRP 1994) shows that about 50 percent of the Asian anglers surveyed consumed whole, gutted fish, while 49 percent consumed fillet or steaks. A majority of white croaker consumers surveyed in the study reported eating their fish whole, gutted. Gold et al. (1997) measured concentrations of tDDT and PCBs in whole fish and fish fillets collected from Asian markets in the Los Angeles area. They found that concentrations of tDDT and PCBs are generally higher in whole fish than in fish fillets (by a factor of 2 to 10); this result was expected because DDT and PCBs are associated with the lipid fraction and whole fish have a higher lipid concentration. In addition, Gold et al. (1997) found that the average size of white croaker in the marketplace was quite small (20.5 cm and 161 grams), making it unlikely that consumers are eating individual fillets because of the difficulty in fish preparation and the small size of the individual fillets. Therefore, for consumers who eat whole fish, risks may be underestimated by up to an order of magnitude.

Cooking fish also may result in the reduction of halogenated hydrocarbon contaminants such as tDDT and PCBs. In a study of white croaker from Santa Monica Bay, cooking skin-off fillets resulted in a 74 percent loss of DDT and a 65 percent loss of PCBs (normalized to uncooked wet weight); the study indicated that the very high contaminant concentrations in Santa Monica Bay may have resulted in a greater percent reduction of contaminant concentrations than have been reported in other areas (Puffer and Gossett, 1983). Anderson et al. (1993) summarized data from the Great Lakes region and recommended using a contaminant reduction factor of 30 percent to account for the contaminant losses resulting from cooking skin-off fillets.

This evaluation assumes that a particular boat angler fishes exclusively in the vicinity of the Palos Verdes Shelf. This assumption is likely to result in overestimation of risk.

This study assumes 30 years of fish consumption for the RME scenario. This is the 90th percentile reported fishing duration for Santa Monica Bay boat anglers. The actual overall exposure duration may be higher or lower than the reported durations; this may result in over- or underestimation of risk.

A moderate to high level of uncertainty is associated with the assumption that current levels of exposure will remain constant over the duration of the exposure (i.e., 30 years), due to changes in activities in the area and changes in fish tissue and sediment concentrations.

A preliminary evaluation of risks to breast-fed infants was performed. This evaluation was intentionally conservative and is associated with a moderate to high level of uncertainty. For example, maternal consumption of one 150-gram meal per month of contaminated fish was assumed. Lower fish consumption rates would result in correspondingly lower risks to the breast-fed infant. Consuming at the overall median rate for boat anglers would result in higher risks to the breast-fed infant. The assumption that reductions in maternal fat levels do not occur during breastfeeding also leads to a potential overestimation of risk.

8.3 Uncertainty in the Toxicity Assessment

Numerous assumptions are required to develop toxicity values (slope factors, reference doses) from dose-response data. A critical assumption underlying all animal-human extrapolations is that there is a relationship between toxicity in test animals and the toxicity anticipated in humans. There may be significant differences in metabolism and other physiological aspects of study animals and the human population. Although many of these aspects are well-characterized, the relationship between interspecies differences and the toxicity of specific chemicals is not known.

Another important assumption is the existence of a threshold for noncarcinogenic effects and no threshold for carcinogens. The threshold issue is currently under evaluation for many chemicals and toxic endpoints.

A major source of uncertainty involves using toxicity values based on experimental studies that substantially differ from typical human exposure scenarios. The derivation of the toxicity values must take into account such differences as 1) using dose-response information from animal studies to predict effects in humans, 2) using dose-response information from high-dose studies to predict

adverse health effects from low doses, 3) using data from short-term studies to predict chronic effects, and 4) extrapolating from specific populations to general populations.

The cancer slope factors in particular are based on studies that may differ greatly from realistic situations. Experimental cancer bioassays typically expose animals to very high levels of chemicals (i.e., the maximum tolerated dose) for their entire lifetime. After the appropriate studies have been identified, the slope factor is calculated as the upper 95th percent confidence limit of the slope of the dose-response curve. This introduces significant conservatism into the risk assessment.

The derivation of RfDs generally involves the use of animal studies. Uncertainty factors ranging from 1 to 10,000 are incorporated into the RfD to provide an extra level of public health protection. The factors used depend on the type of study from which the value has been derived (e.g., animal or human, chronic or acute). The scientific basis for this practice is somewhat uncertain. In general, high uncertainty factors are meant to bias the results conservatively so that the RfD will not result in adverse health effects. Uncertainty factors for tDDT and PCBs are 100 and 300, respectively.

8.4 Uncertainty in Risk Characterization

Because the exposure scenarios were designed to represent the reasonable maximum exposure and were intentionally conservative, point estimate risks may overstate risks from typical (or average) exposures.

Because slope factors are typically upper 95 percent confidence limits on the mean probability of carcinogenic response (i.e., upper bound estimates), these slope factors are inherently conservative. In addition, the assumption that any exposure to a carcinogen produces some degree of risk is unproven; hence, it is possible that low levels of some carcinogens may not produce any excess risk at all.

9.0 SUMMARY

A human health risk evaluation was performed for DDT (and its metabolites) and PCBs in fish collected from the Palos Verdes Shelf. Two exposure scenarios were evaluated: a reasonable maximum exposure scenario based on single species consumption rates (based on only those anglers consuming a particular species), and a central tendency scenario based on a mixed-species diet. Consumption rates for all boat anglers derived from the *Santa Monica Bay Seafood Consumption Study* were the basis for the risk evaluation.

Point estimate cancer risks for the RME scenario are above 1×10^{-4} for consumption of white croaker (2×10^{-3}) and surfperches (2×10^{-4}). RME noncancer hazards exceeded a HQ of 1 for white croaker, surfperches, barred sand bass, California halibut, kelp bass, and California sheephead. The HQs for white croaker were particularly high (17 and 32 for tDDT and PCBs, respectively). In general, tDDT in white croaker tissue is the most significant contributor to cancer risk; PCBs in white croaker tissue are the most significant contributor to noncancer health hazards.

For the CT scenario, the cancer risk was 2×10^{-5} . In addition, the CT hazard quotients for both tDDT and PCBs were less than 1, indicating that noncancer health effects are unlikely to occur.

Results of a Monte Carlo simulation for consumption of white croaker by boat anglers showed that mean, median, 90th percentile, and 95th percentile cancer risks are above 1×10^{-4} ; the mean, median, and upper percentiles of the noncancer HQ exceed 1. This indicates that, based on available data on fish consumption rates, exposure duration, and white croaker tissue concentrations, both cancer and noncancer health effects are likely to occur for boat anglers who catch and consume white croaker collected at the Palos Verdes Shelf.

The potential risks to breast-fed infants due to consumption of tDDT and PCBs in breast milk were also evaluated. Results indicate that tDDT and PCB breast milk concentrations, based on maternal consumption of one 150-gram meal of white croaker per month, could be as high as 0.8 mg/kg and 0.05 mg/kg, respectively. This corresponds to noncancer HQs of 220 and 370 for tDDT and PCBs, respectively. Based on maternal consumption of kelp bass, noncancer HQs to an infant are 3 and 16 for tDDT and PCBs, respectively.

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TABLES

**Table 1-1
Fish Consumption Advisories
Southern California Locations Between Point Dume and Dana Point**

Site	Fish Species	Recommendations
Point Dume Malibu	White croaker	Do not consume
Malibu Pier	Queenfish	One meal a month
Short Bank	White croaker	One meal every two weeks
Redondo Pier	Corbina	One meal every two weeks
Point Vicente Palos Verdes – Northwest	White croaker	Do not consume
Whites Point	White croaker Sculpin Rockfishes Kelp bass	Do not consume One meal every two weeks*
Los Angeles/Long Beach Harbors (esp. Cabrillo Pier)	White croaker Queenfish Black croaker Surfperches	Do not consume One meal every two weeks*
Los Angeles/Long Beach Breakwater (ocean side)	White croaker Queenfish Surfperches Black croaker	One meal a month*
Belmont Pier Pier J	Surfperches	One meal every two weeks
Horseshoe Kelp	Sculpin White croaker	One meal a month*
Newport Pier	Corbina	One meal every two weeks

* Consumption recommendation is for all the listed species combined.

Sites bolded are located on the Palos Verdes Shelf.

Source: OEHHA, 1997.

Table 2-1
Summary of Available Fish Tissue Data for the Palos Verdes Shelf Area

Fish Species	Analytical Parameters ¹	Date	Source ²
White croaker			
muscle	tDDT, 2,4'-DDD, 2,4'-DDE, 2,4'-DDT, 4,4'-DDD, 4,4'-DDE, 4,4'-DDT, PCBs (Aroclors 1242, 1254, 1260)	1985, 1990, 1992, 1994, 1995, 1996, 1997	LACSD Palos Verdes Ocean Monitoring Reports
	tDDT, PCBs	1987	Pollock et al., 1991
	tDDT, PCBs congeners and Aroclors (1242, 1254, 1260), DDMU	1990	SMBRP, 1992
Dover sole			
muscle	tDDT, 2,4'-DDD, 2,4'-DDE, 2,4'-DDT, 4,4'-DDD, 4,4'-DDE, 4,4'-DDT, PCBs (Aroclors 1242, 1254, 1260)	1985 - 1993, 1995, 1996	LACSD Palos Verdes Ocean Monitoring Reports
liver	DDT isomers and metabolites, 12 PCB congeners, plus chlorinated pesticides	1994	SCCWRP 1994 (Southern California Bight Pilot Project)
Kelp bass			
muscle, liver	tDDT, 2,4'-DDD, 2,4'-DDE, 2,4'-DDT, 4,4'-DDD, 4,4'-DDE, 4,4'-DDT, PCBs (Aroclors 1242, 1254, 1260)	1985, 1988, 1990, 1992, 1995, 1996	LACSD Palos Verdes Ocean Monitoring Reports
muscle, liver	tDDT, PCBs, chlorinated pesticides	1985	Risebrough, 1987
muscle only	tDDT, PCBs	1987	Pollock et al., 1991
Surfperch, black surfperch			
muscle	tDDT, 2,4'-DDD, 2,4'-DDE, 2,4'-DDT, 4,4'-DDD, 4,4'-DDE, 4,4'-DDT, PCBs (Aroclors 1242, 1254, 1260)	1985, 1990, 1996	LACSD Data
muscle	tDDT, PCBs	1987	Pollock et al., 1991
Sanddabs			
muscle	tDDT, PCBs	1987	Pollock et al., 1991
liver	DDT isomers and metabolites, 12 PCB congeners plus chlorinated pesticides	1994	SCCWRP, 1994 (Southern California Bight Pilot Project)
Other species: sculpin, Pacific bonito, mackerel, queenfish, corbina, rockfish, barred sand bass			
muscle	tDDT, PCBs	1987	Pollock et al., 1991
California halibut			
muscle	tDDT, 2,4'-DDD, 2,4'-DDE, 2,4'-DDT, 4,4'-DDD, 4,4'-DDE, 4,4'-DDT, PCBs (Aroclors 1242, 1254, 1260)	1991	LACSD Data

(1) tDDT means total DDT metabolites; PCBs is the sum of aroclors/congeners sampled for.

(2) For sources other than LACSD all data are derived from the Southern California Bight NRDA data files, thus parameters are limited to those listed.

SMBRP - Santa Monica Bay Restoration Project; LACSD - Los Angeles County Sanitation Districts; NRDA - Natural Resource Damage Assessment; SCCWRP - Southern California Coastal Water Research Project

Table 2-2
Comparison of Maximum Detected Concentrations of Contaminants
in Fish Tissue to Human Health Screening Values

Chemical	Maximum Detected Concentration: PV Shelf Area ($\mu\text{g}/\text{kg}$)	Human Health Screening Value ($\mu\text{g}/\text{kg}$)	Ratio of Maximum Detected Concentration to Screening Value	Source of Screening Value
Total DDT	135,000 (LACSD, 1995, Zone 1)	500	270	FDA Action level for fish ¹
		30	4,500	USEPA 1995a ²
		32	4,200	Basis for EPA Water Quality Criteria ³
Total PCBs	10,200 (LACSD, 1995, Zone 1)	200	51	FDA Tolerance level for fish ¹
		1	10,000	USEPA 1995a ²
		1	10,000	Basis for EPA Water Quality Criteria ³
Chlordane	29 (SMBRP, 1990, Whites Point)	30	<1	FDA Action level for fish ¹
		8	4	USEPA 1995a ²
HCH (Lindane)	1.5 (SMBRP, 1990, PV North and South)	8	<1	USEPA 1995a ²
HCB	0.4 (SMBRP, 1990, PV South and Whites Point)	7	<1	USEPA 1995a ²
Selenium	1,400 (SMBRP, 1990, Whites Point)	50,000	<1	USEPA 1995a ²
Mercury	724 (Pollock et al., 1987, Whites Point)	3,000	<1	USEPA 1995a ² ; based on chronic systemic endpoints
		1000	<1	FDA Action level for methylmercury in edible portion of fish ¹
		600	1	USEPA 1995a ² ; based on developmental health endpoints
Tributyltin	105 (Pollock et al., 1987, Marina del Rey)	300	<1	USEPA 1995a ²

(1) From *FDA/CFSAN Action Levels for Poisonous or Deleterious Substances in Human Food and Animal Feed* (USFDA, 1994). For carcinogens (DDT, PCBs, chlordane), action levels were modified to correspond to a cancer risk of 1E-6. Action levels are based on an average fish and shellfish consumption rate in the general population (including both anglers and nonanglers) of 6.5 g/day.

(2) From *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories, Volume I: Fish Sampling and Analysis, Second Edition* (USEPA, 1995a). For carcinogens (DDT, PCBs, chlordane, HCH, and HCB), screening value was modified to correspond to a cancer risk of 1E-6. Screening levels are based on an average fish and shellfish consumption rate in the general population (including both anglers and nonanglers) of 6.5 g/day.

(3) From 40 CFR Part 131, 57 FR 60848 (12/22/92).

Table 2-3
Summary of Data Used in the Human Health Risk Evaluation:
tDDT in Fish Tissue

Fish Species	Sample Locations	Sample Date	Sample Type	No. of Samples	Range of Sample Conc'ns (µg/kg)	Mean Sample Conc'n (µg/kg)	UCL95 Conc'n (µg/kg)	Notes
Barred sand bass	L.A. Harbor Area	1987	Muscle	10 composite samples	29 to 190	74	110	
California halibut	LACSD Zone B (between Zones 1 and 2)	1991	Muscle	10 individual samples	35 to 1,360	460	1,360	a
California scorpionfish	Palos Verdes Shelf	1987	Muscle	15 composite samples	16 to 360	92	180	
California sheephead	LACSD Zones 1, 2, and 3	1995/96	Muscle	60 individual samples	20 to 1,120	220	290	b
Chub mackerel	Palos Verdes Shelf	1987	Muscle	15 composite samples	4 to 82	25	47	
Halfmoon	Catalina Island	1987	Muscle	5 composite samples	ND	19	19	c
Kelp bass	LACSD Zones 1, 2, and 3	1995/96	Muscle	60 individual samples	20 to 1,120	220	290	
Pacific barracuda	Fourteen-Mile Bank	1987	Muscle	5 composite samples	9 to 51	25	51	d
Pacific bonito	Palos Verdes Shelf	1987	Muscle	15 composite samples	7 to 150	33	49	
Rockfishes	Palos Verdes Shelf	1987	Muscle	25 composite samples	26 to 380	92	120	
Surfperches	LACSD Zones 1, 2, and 3	1996	Muscle	30 individual samples	40 to 1,310	340	530	
White croaker	LACSD Zones 1, 2, and 3	1996/97	Muscle	60 individual samples	470 to 59,600	13,400	21,300	

NOTES:

All distributions assumed lognormal except as indicated.

(a) The UCL95 concentration was higher than the maximum detected concentration, therefore the maximum detected concentration was used to represent an exposure point concentration.

(b) The data for kelp bass were used to represent an exposure point concentration for California sheephead.

(c) All 5 composite samples were ND; therefore, one-half the sample quantitation limit was used to represent the exposure point concentration.

(d) Because only five barracuda samples were collected, it was not possible to determine the distribution of the barracuda tissue data. The UCL95 was approximated by using the higher of the UCL95s calculated assuming a normal or lognormal distribution. For barracuda, however, the UCL95 assuming a lognormal distribution (the higher of the two) exceeded the maximum detected concentration. Therefore, the maximum detected concentration of tDDT in barracuda was used to represent the exposure point concentration.

Table 2-4
 Summary of Data Used in the Human Health Risk Evaluation:
 PCBs in Fish Tissue

Fish Species	Sample Locations	Sample Date	Sample Type	No. of Samples	Range of Sample Conc'n's (µg/kg)	Mean Sample Conc'n (µg/kg)	UCL95 Conc'n (µg/kg)	Notes
Barred sand bass	L.A. Harbor Area	1987	Muscle	10 composite samples	13 to 120	50	90	
California halibut	LACSD Zone B (between Zones 1 and 2)	1991	Muscle	10 individual samples	30 to 180	90	140	
California scorpionfish	Palos Verdes Shelf	1987	Muscle	15 composite samples	3 to 52	21	44	
California sheephead	LACSD Zones 1, 2, and 3	1995/96	Muscle	60 individual samples	ND to 160	46	68	a
Chub mackerel	Palos Verdes Shelf	1987	Muscle	15 composite samples	1 to 57	16	42	
Halfmoon	Catalina Island	1987	Muscle	5 composite samples	ND	25	25	b
Kelp bass	LACSD Zones 1, 2, and 3	1995/96	Muscle	60 individual samples	ND to 160	46	68	
Pacific barracuda	Fourteen-Mile Bank	1987	Muscle	5 composite samples	12 to 24	16	23	c
Pacific bonito	Palos Verdes Shelf	1987	Muscle	15 composite samples	2 to 110	17	34	
Rockfishes	Palos Verdes Shelf	1987	Muscle	25 composite samples	6 to 63	21	28	
Surfperches	LACSD Zones 1, 2, and 3	1996	Muscle	30 individual samples	ND to 150	35	70	
White croaker	LACSD Zones 1, 2, and 3	1996/97	Muscle	60 individual samples	70 to 5,630	1,170	1,620	

NOTES:

All distributions assumed lognormal except as indicated.

(a) The data for kelp bass were used to represent an exposure point concentration for California sheephead.

(b) All 5 composite samples were ND; therefore, one-half the sample quantitation limit was used to represent the exposure point concentration.

(c) Because only five barracuda samples were collected, it was not possible to determine the distribution of the barracuda tissue data. The UCL95 was approximated by using the higher of the UCL95s calculated assuming a normal or lognormal distribution. For barracuda, the UCL95 assuming a lognormal distribution was higher and was therefore used to represent the exposure point concentration.

Table 3-1
Toxicity Values: Potential Noncancer Effects

Chemical	Chronic Oral RfD	Confidence Level	Critical Effect	Uncertainty Factor	Modifying Factor	RfD Source
PCBs (Based on Aroclor 1254)	2×10^{-5}	Medium	Ocular exudate; inflamed and prominent Meibomian glands; distorted growth of finger and toenails; decreased antibody response to sheep erythrocytes; based on monkey clinical and immunological studies.	300	1	IRIS
DDT	5×10^{-4}	Medium	Liver lesions; based on a 27-week rat feeding study.	100	1	IRIS

IRIS: Integrated Risk Information System (USEPA, 1998b)

Table 3-2
Tiers of Human Potency and Slope Estimates for Environmental Mixtures of PCBs
(from USEPA, 1996a)

High Risk and Persistence				
ED10 ^a	LED10 ^b	Central Slope ^c	Upper-bound Slope ^d	Criteria for Use
0.086	0.067	1.	2.	Food chain exposure Sediment or soil ingestion Dust or aerosol inhalation Dermal exposure, if an absorption factor has been applied to reduce the external dose Presence of dioxin-like, tumor-promoting, or persistent congeners in other media Early life exposure (all pathways and mixtures)
Low Risk and Persistence				
ED10 ^a	LED10 ^b	Central Slope ^c	Upper-bound Slope ^d	Criteria for Use
0.38	0.27	0.3	0.4	Ingestion of water-soluble congeners Inhalation of evaporated congeners Dermal exposure, if no absorption factor has been applied to reduce the external dose
Lowest Risk and Persistence				
ED10 ^a	LED10 ^b	Central Slope ^c	Upper-bound Slope ^d	Criteria for Use
2.4	1.4	0.04	0.07	Congener or isomer analyses verify that congeners with more than 4 chlorines comprise less than ½% of total PCBs

^a Estimated dose associated with 10% increased incidence of cancer, in mg/kg-d

^b 95% lower bound on ED10, in mg/kg-d

^c Per mg/kg-d, computed as 0.10/ED10 and rounded to one significant digit

^d Per mg/kg-d, computed as 0.10/LED10 and rounded to one significant digit.

Table 4-1
Exposure Point Concentrations in Fish Tissue

Fish Species	RME Scenario		CT Scenario	
	Total DDT ($\mu\text{g}/\text{kg}$)	Total PCBs ($\mu\text{g}/\text{kg}$)	Total DDT ($\mu\text{g}/\text{kg}$)	Total PCBs ($\mu\text{g}/\text{kg}$)
Barred sand bass	110	90	74	50
California halibut	1,360	140	460	90
California scorpionfish	180	44	92	21
California sheephead ^a	290	68	220	46
Chub mackerel	47	42	25	16
Halfmoon	19	25	19	25
Kelp bass	290	68	220	46
Pacific barracuda	51	23	25	16
Pacific bonito	49	34	33	17
Rockfishes	120	28	92	21
Surfperches	530	70	340	35
White croaker	21,300	1,620	13,400	1,170
Other species ^b	280	56	140	34

(a) Because no tissue data were available for California sheephead, the exposure point concentrations (EPCs) in kelp bass were used to represent sheephead (see also Section 2.2).

(b) The EPC for "Other species" was calculated by averaging the EPCs for all other included species except white croaker and California sheephead.

RME – Reasonable maximum exposure

CT – Central tendency

Table 4-2
Fish Consumption Rates - RME Scenario

Fish Species	No. of Boaters Consuming this Species^a	Mean Consumption Rate (g/day)^b	UCL95 Consumption Rate (g/day)^c	90th Percentile Consumption Rate (g/day)^d	95th Percentile Consumption Rate (g/day)^d
Barred sand bass	128	33.1	38.8	69.4	99.6
California halibut	48	23.8	30.6	47.6	64.4
California scorpionfish	46	16.0	19.3	29.6	38.0
California sheephead	11	24.1	47.4	46.9	61.8
Chub mackerel	50	19.0	24.1	37.9	51.5
Halfmoon	18	15.5	23.3	29.5	38.3
Kelp bass	129	29.6	34.9	62.3	88.4
Pacific barracuda	66	27.3	34.1	56.0	78.2
Pacific bonito	57	15.9	18.9	30.0	38.9
Rockfishes	53	25.2	32.1	50.9	68.5
Surfperches	8	33.8	94.8	67.4	90.0
White croaker	13	16.7	27.9	31.8	40.7
Other species	44	18.9	23.6	36.3	48.0

(a) Includes all boaters (private and party) surveyed in the *Santa Monica Bay Seafood Consumption Study* who reported eating fish caught in Santa Monica Bay within the last 28 days of being surveyed, or who had fish in hand at the time of the survey.

(b) Mean consumption rate of each species for boaters who reported consuming that species (e.g., the white croaker consumption rate of 16.7 g/day is the mean consumption rate for white croaker consumers only).

(c) Calculated assuming consumption rates are lognormally distributed; the UCL95 values were used in the HHRE to represent reasonable maximum exposures.

(d) 90th and 95th percentile consumption rates are presented for comparison only.

RME - Reasonable maximum exposure; UCL 95 - 95 percent upper confidence limit of the mean.

Table 4-3
Fish Consumption Rates - CT Scenario

Fish Species	No. of Surveyed Boaters Consuming this Species ^a	Individual Diet Fraction ^b	Median Fish Consumption Rate (g/day) ^c	Median Consumption Rate by Species (g/day)
Barred sand bass	128	18.0%	21.4	3.85
California halibut	48	6.9%	21.4	1.48
California scorpionfish	46	4.9%	21.4	1.05
California sheephead	11	1.1%	21.4	0.23
Chub mackerel	50	8.5%	21.4	1.81
Halfmoon	18	2.0%	21.4	0.42
Kelp bass	129	19.6%	21.4	4.20
Pacific barracuda	66	10.3%	21.4	2.21
Pacific bonito	57	10.1%	21.4	2.16
Rockfishes	53	9.6%	21.4	2.05
Surfperches	8	1.0%	21.4	0.21
White croaker	13	2.2%	21.4	0.48
Other species	44	5.9%	21.4	1.26
TOTAL	338	100.0%		21.4

(a) Includes all boaters surveyed in the *Santa Monica Bay Seafood Consumption Study* who reported eating fish caught in Santa Monica Bay within the last 28 days of being surveyed, or who had fish in hand at the time of the survey which they planned to consume.

(b) Diet fractions were calculated for each fish species reported consumed by each individual boat angler, then averaged by species over all 338 boaters.

(c) The median (or 50th percentile) total fish consumption rate for the 338 boaters.

CT - Central tendency

Table 4-4
Summary of Exposure Assumptions

Variable	Units	RME Value	CT Value	Comment
Ingestion Rate (IR)	g/day	See Table 4-2	see Table 4-3	Specific to fish species
Exposure Frequency (EF)	days/yr	365	365	From USEPA, 1991; fish ingestion rates are based on annual consumption, therefore a consistent exposure frequency must be used.
Exposure Duration (ED)	yrs	30	13.8	RME and CT values are 90th percentile and mean, respectively, reported fishing duration of boaters based on data from the Santa Monica Bay Seafood Consumption Study (SMBRP, 1994).
Conversion Factor (CF)	kg/g	1.00×10^{-3}	1.00×10^{-3}	
Body Weight	kg	70	70	From USEPA, 1991; EPA default value for adults.
Averaging Time (AT)	days	25,550 (cancer)	25,550 (cancer)	Equal to 70 years, or lifetime (USEPA, 1991)
	days	10,950 (noncancer)	5,037 (noncancer)	Equal to exposure duration (USEPA, 1991)

RME - Reasonable maximum exposure

CT - Central tendency

Table 4-5
Summary of Chemical Intakes for Fish Ingestion

Fish Species	Lifetime Average Daily Dose		Chronic Daily Intake	
	Cancer		Noncancer Health Effects	
	tDDT mg/kg-day	PCBs mg/kg-day	tDDT mg/kg-day	PCBs mg/kg-day
<i>RME Scenario</i>				
Barred sand bass	2.61E-05	2.14E-05	6.10E-05	4.99E-05
California halibut	2.55E-04	2.62E-05	5.95E-04	6.12E-05
California scorpionfish	2.13E-05	5.20E-06	4.96E-05	1.21E-05
California sheephead	8.42E-05	1.97E-05	1.96E-04	4.60E-05
Chub mackerel	6.93E-06	6.20E-06	1.62E-05	1.45E-05
Halfmoon	2.71E-06	3.57E-06	6.32E-06	8.32E-06
Kelp bass	6.20E-05	1.45E-05	1.45E-04	3.39E-05
Pacific barracuda	1.06E-05	4.80E-06	2.48E-05	1.12E-05
Pacific bonito	5.67E-06	3.93E-06	1.32E-05	9.18E-06
Rockfishes	2.36E-05	5.50E-06	5.50E-05	1.28E-05
Surfperches	3.08E-04	4.06E-05	7.18E-04	9.48E-05
White croaker	3.64E-03	2.77E-04	8.49E-03	6.46E-04
Other species	3.98E-05	8.15E-06	9.29E-05	1.90E-05
<i>CT Scenario</i>				
Barred sand bass	8.03E-07	5.42E-07	4.07E-06	2.75E-06
California halibut	1.91E-06	3.74E-07	9.70E-06	1.90E-06
California scorpionfish	2.72E-07	6.20E-08	1.38E-06	3.15E-07
California sheephead	1.46E-07	3.05E-08	7.40E-07	1.55E-07
Chub mackerel	1.28E-07	8.20E-08	6.50E-07	4.16E-07
Halfmoon	2.29E-08	3.01E-08	1.16E-07	1.53E-07
Kelp bass	2.60E-06	5.43E-07	1.32E-05	2.76E-06
Pacific barracuda	1.55E-07	9.93E-08	7.87E-07	5.04E-07
Pacific bonito	2.01E-07	1.03E-07	1.02E-06	5.25E-07
Rockfishes	5.32E-07	1.22E-07	2.70E-06	6.16E-07
Surfperches	2.05E-07	2.11E-08	1.04E-06	1.07E-07
White croaker	1.78E-05	1.55E-06	9.01E-05	7.87E-06
Other species	4.91E-07	1.20E-07	2.49E-06	6.08E-07
<i>TOTAL</i>	2.52E-05	3.68E-06	1.28E-04	1.87E-05

RME - Reasonable maximum exposure

CT - Central tendency

Table 5-1
Summary of Point Estimate Risks
RME Scenario (Single Species Diet)

Fish Species	Cancer Risk tDDT	Cancer Risk PCBs	Cancer Risk Combined	Noncancer HQ tDDT	Noncancer HQ PCBs
Barred sand bass	9×10^{-6}	4×10^{-5}	5×10^{-5}	0.1	3
California halibut	9×10^{-5}	5×10^{-5}	1×10^{-4}	1	3
California scorpionfish	7×10^{-6}	1×10^{-5}	2×10^{-5}	0.1	0.6
California sheephead	3×10^{-5}	4×10^{-5}	7×10^{-5}	0.4	2
Chub mackerel	2×10^{-6}	1×10^{-5}	2×10^{-5}	0.03	0.7
Halfmoon	9×10^{-7}	7×10^{-6}	8×10^{-6}	0.01	0.4
Kelp bass	2×10^{-5}	3×10^{-5}	5×10^{-5}	0.3	2
Pacific barracuda	4×10^{-6}	1×10^{-5}	1×10^{-5}	0.05	0.6
Pacific bonito	2×10^{-6}	8×10^{-6}	1×10^{-5}	0.03	0.5
Rockfishes	8×10^{-6}	1×10^{-5}	2×10^{-5}	0.1	0.6
Surfperches	1×10^{-4}	8×10^{-5}	2×10^{-4}	1	5
White croaker	1×10^{-3}	6×10^{-4}	2×10^{-3}	17	32
Other species	1×10^{-5}	2×10^{-5}	3×10^{-5}	0.2	1

Species in bold have cancer risk greater than 1×10^{-4} or HQ greater than 1

HQ = Hazard quotient

RME = Reasonable maximum exposure

Table 5-2
Summary of Point Estimate Risks
CT Scenario (Mixed Species Diet)

Fish Species	Cancer Risk tDDT	Cancer Risk PCBs	Cancer Risk Combined	Noncancer HQ tDDT	Noncancer HQ PCBs
Barred sand bass	3×10^{-7}	1×10^{-6}	1×10^{-6}	0.008	0.1
California halibut	7×10^{-7}	8×10^{-7}	1×10^{-6}	0.02	0.1
California scorpionfish	9×10^{-8}	1×10^{-7}	2×10^{-7}	0.003	0.02
California sheephead	5×10^{-8}	6×10^{-8}	1×10^{-7}	0.002	0.008
Chub mackerel	4×10^{-8}	2×10^{-7}	2×10^{-7}	0.001	0.02
Halfmoon	8×10^{-9}	6×10^{-8}	7×10^{-8}	0.0002	0.008
Kelp bass	9×10^{-7}	1×10^{-6}	2×10^{-6}	0.03	0.1
Pacific barracuda	5×10^{-8}	2×10^{-7}	3×10^{-7}	0.002	0.03
Pacific bonito	7×10^{-8}	2×10^{-7}	3×10^{-7}	0.002	0.03
Rockfishes	2×10^{-7}	2×10^{-7}	4×10^{-7}	0.005	0.03
Surfperches	7×10^{-8}	4×10^{-8}	1×10^{-7}	0.002	0.005
White croaker	6×10^{-6}	3×10^{-6}	9×10^{-6}	0.2	0.4
Other species	2×10^{-7}	2×10^{-7}	4×10^{-7}	0.005	0.03
Total	9×10^{-6}	7×10^{-6}	2×10^{-5}	0.3	0.9

HQ = Hazard quotient
 CT = Central Tendency

Table 5-3
Comparison of Risk Results with Previous Studies

	Single-Species Diet				Mixed-Species Diet			
	Current HHRE RME Scenario ¹	Comprehensive Study ²	SMBRP Risk Assessment ³	Heal the Bay Study (whole fish) ⁴	Heal the Bay Study (fish fillets) ⁴	Current HHRE CT Scenario ⁵	Comprehensive Study ⁶	SMBRP Risk Assessment ⁷
Cancer Risk								
White croaker	2 x 10 ⁻³	1 x 10 ⁻³ to 2 x 10 ⁻³	2 x 10 ⁻³	9 x 10 ⁻⁵ to 2 x 10 ⁻³	6 x 10 ⁻⁵ to 9 x 10 ⁻⁴	9 x 10 ⁻⁶	NA	NA
All fish species	NA	NA	NA	NA	NA	2 x 10 ⁻⁵	4 x 10 ⁻⁴	2 x 10 ⁻⁴
Noncancer Hazard Quotient								
White croaker (tDDT)	17	NA	9.9	NA	NA	0.2	NA	NA
White croaker (PCBs)	32	NA	17	NA	NA	0.4	NA	NA
All fish species (tDDT)	NA	NA	NA	NA	NA	0.3	NA	0.5
All fish species (PCBs)	NA	NA	NA	NA	NA	0.9	NA	1.3

- 1 - Assumes a white croaker ingestion rate of 28 g/day, UCL95 tissue concentrations from PV Shelf; boaters only
 - 2 - Assumes a white croaker ingestion rate of 23 g/day, mean tissue concentration from PV Shelf; from Pollock et al. (1991).
 - 3 - Assumes a white croaker ingestion rate of 30 g/day, mean tissue concentrations from PV Shelf area, all fishing modes; from SMBRP (1997)
 - 4 - Assumes a white croaker ingestion rate of 50 g/day, mean tissue concentrations; from Gold et al. (1997).
 - 5 - Assumes a total ingestion rate of 21.4 g/day, 2.2 percent of diet is white croaker (0.48 g/day), mean tissue concentrations from PV Shelf, boaters only.
 - 6 - Assumes a total ingestion rate of 23 g/day, white croaker consumption of 4.6 g/day, mean tissue concentrations from Cabrillo Pier; from Pollock et al. (1991).
 - 7 - Assumes a total ingestion rate of 21 g/day, 7 percent of diet is white croaker (1.5 g/day), mean tissue concentrations from PV Shelf area, all fishing modes; from SMBRP (1997)
- HHRE - Human health risk evaluation; RME - Reasonable maximum exposure; CT - Central tendency; SMBRP - Santa Monica Bay Restoration Project
NA - Not available, or analysis not performed in the referenced study.

Table 6-1
Exposure Assumptions for Monte Carlo Analysis

Exposure Variable	Distribution Type	Parameters of Distribution	Point Estimate Value (RME Scenario)	Source for Distribution and Parameters
Conc'n of tDDT in white croaker tissue (mg/kg)	Lognormal	Mean=14.0; SD=25.4	21.3	Based on LACSD data from Zones 1, 2, and 3 for the years 1996 and 1997 ^a
Conc'n of PCBs in white croaker tissue (mg/kg)	Lognormal	Mean=1.2; SD=1.6	1.6	Based on LACSD data from Zones 1, 2, and 3 for the years 1996 and 1997 ^a
White croaker ingestion rate (g/day)	Lognormal	Mean=16.7; SD=13.0	27.9	Adapted from <i>Santa Monica Bay Seafood Consumption Study</i> (SMBRP 1994); based on white croaker consumption by boat anglers who reported eating white croaker in the previous 28 days (13 anglers)
Exposure frequency (days/yr)	Determinate value	365	365	Corresponds to an annual average fish consumption rate
Exposure duration (yrs)	Empirical	Median=10; 90th percentile=30	30	Based on reported durations of fishing by boat anglers in the <i>Santa Monica Bay Seafood Consumption Study</i> (SMBRP 1994)
Slope factor (mg/kg-day) ¹	Determinate value	tDDT - 0.34; PCBs - 2.0	tDDT - 0.34; PCBs - 2.0	From USEPA 1998b; upper 95 percent confidence limit of the dose-response curve; slope factor for tDDT is based on oral slope factor for 4,4'-DDE and 4,4'-DDT
Reference dose (mg/kg-day)	Determinate value	tDDT - 5×10^{-4} ; PCBs - 2×10^{-5}	tDDT - 5×10^{-4} ; PCBs - 2×10^{-5}	From USEPA 1998b; RfD for PCBs is based on oral RfD for Aroclor 1254; RfD for tDDT is based on oral RfD for 4,4'-DDT
Body weight (kg)	Lognormal	Mean=71; SD=15.9	70	From Brainard and Burmaster (1992); distribution truncated at 40 kg and 150 kg
Averaging time (days)	Determinate value	25,550	25,550	Corresponds to a lifetime of 70 years

NOTE: A correlation coefficient of 0.96 was applied to fish tissue concentrations of tDDT and PCBs.

a - Fish tissue concentrations used to develop this distribution are presented in Appendix A.

SF - Cancer slope factor

SD - Standard deviation

RfD - Reference dose

Table 6-2
Monte Carlo Simulation Results for Cancer Risk and Noncancer Hazard
White Croaker Consumption by Boat Anglers

	Cancer Risk - tDDT	Cancer Risk - PCBs	Cancer Risk - tDDT and PCBs Combined	HQ - tDDT	HQ - PCBs
Mean	2×10^{-4}	1×10^{-4}	3.4×10^{-4}	6.8	14
50th Percentile	5×10^{-5}	3×10^{-5}	8.0×10^{-5}	2.6	6.6
90th Percentile	5×10^{-4}	3×10^{-4}	8×10^{-4}	16	32
95th Percentile	9×10^{-4}	5×10^{-4}	1×10^{-3}	26	52
RME Point Estimate (white croaker)	1×10^{-3}	6×10^{-4}	2×10^{-3}	17	32
CT Point Estimate (mixed-species diet)	9×10^{-6}	7×10^{-6}	2×10^{-5}	0.3	0.9

RME - reasonable maximum exposure

CT - central tendency

Table 7-1
Contaminant Concentrations in Breast Milk

Variable	Units	tDDT	PCBs	Comment
Daily Intake _{maternal} - average daily intake based on white croaker consumption	mg/kg-day	1.8×10^{-3}	1.3×10^{-4}	Based on assumption that mother consumes fish at the rate of one 150 gram meal per month or 4.9 grams/day; Body weight of 60 kg used, EPA default value for females (USEPA, 1997a)
Daily Intake _{maternal} - average daily intake based on kelp bass consumption	mg/kg-day	2.4×10^{-5}	5.6×10^{-6}	Based on assumption that mother consumes fish at the rate of one 150-gram meal per month or 4.9 grams/day; body weight of 60 kg used, EPA default value for females (USEPA, 1997a)
$t_{1/2}$ - half-life of contaminant in the mother's body	days	2,920	2,555	Value for tDDT from USEPA, 1994a (8 yrs); value for PCBs from USEPA, 1996a (7 years)
f_1 - fraction of contaminant that partitions to mother's fat	unitless	0.9	0.9	Smith 1987
f_2 - fraction of mother's weight that is fat	unitless	0.33	0.33	Butte 1984
f_3 - fraction of milk that is fat	unitless	0.04	0.04	Butte 1984, USEPA, 1997a
C_{milk} - concentration of contaminant in breast milk	mg/kg			$C_{milk} \text{ (mg/kg)} =$ $Daily \ Intake_{maternal} \times t_{1/2} \times f_1 \times \left(\frac{f_3}{[f_2 \times 0.693]} \right)$
White croaker scenario		0.8	0.054	
Kelp bass scenario		0.011	0.0022	

**Table 7-2
Infant's Average Daily Intake Due to Breast Milk Consumption**

Variable	Units	Fish Species	tDDT	PCBs	Comment
C _{milk} - concentration of contaminant in breast milk	mg/kg	White croaker	.8	.054	Values taken from Table 7-1
		Kelp bass	.011	.0022	
IR _{milk} - Infant's milk ingestion rate per body weight	g-kg/day		138	138	OEHHA, 1996; 95th percentile breast milk intake for infants presumed to be exclusively breast-fed during their first year of life
EF _{Infant} - Exposure Frequency	days/year		365	365	Assumes infant's consume breast milk on a daily basis
ED _{Infant} - Exposure Duration	year		1	1	OEHHA 1996 default value; conservative assumption
CF - Conversion Factor	kg/g		0.001	0.001	
AT - Averaging Time	days		365	365	Averaging time = exposure duration for noncancer health effects (in days)
Daily Intake _{Infant} - Breast fed Infant's Daily Intake	mg/kg-day	White croaker	1.1 x 10 ⁻¹	7.4 x 10 ⁻³	Daily Intake _{Infant} = $\frac{(C_{milk} \times IR_{milk} \times EF_{Infant} \times ED_{Infant} \times CF)}{AT}$
		Kelp bass	1.5 x 10 ⁻³	3.1 x 10 ⁻⁴	

Table 7-3
Summary of Point Estimate Hazard Quotients
Infant Consumption of Contaminated Breast Milk

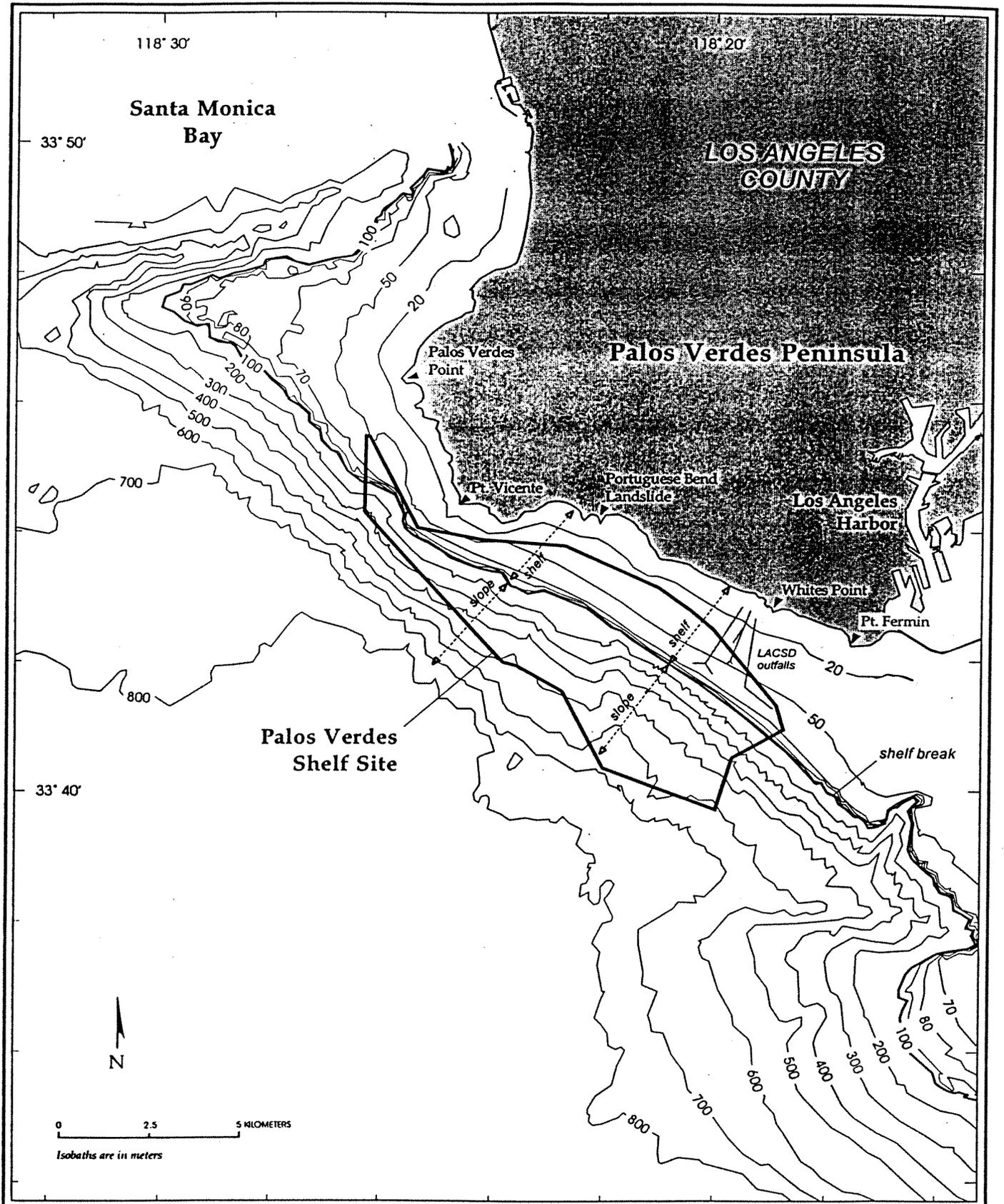
Fish Species	Noncancer HQ tDDT	Noncancer HQ PCBs
White croaker	220	370
Kelp bass	3	16

$$\text{Noncancer tDDT} = \frac{\text{Dose}_{\text{Infant}} \text{ mg/kg-day}}{\text{Reference Dose [0.0005 mg/kg-day]}}$$

$$\text{Noncancer PCBs} = \frac{\text{Dose}_{\text{Infant}} \text{ mg/kg-day}}{\text{Reference Dose [0.0002 mg/kg-day]}}$$

FIGURES

Figure 1-1
Palos Verdes Shelf Site Map



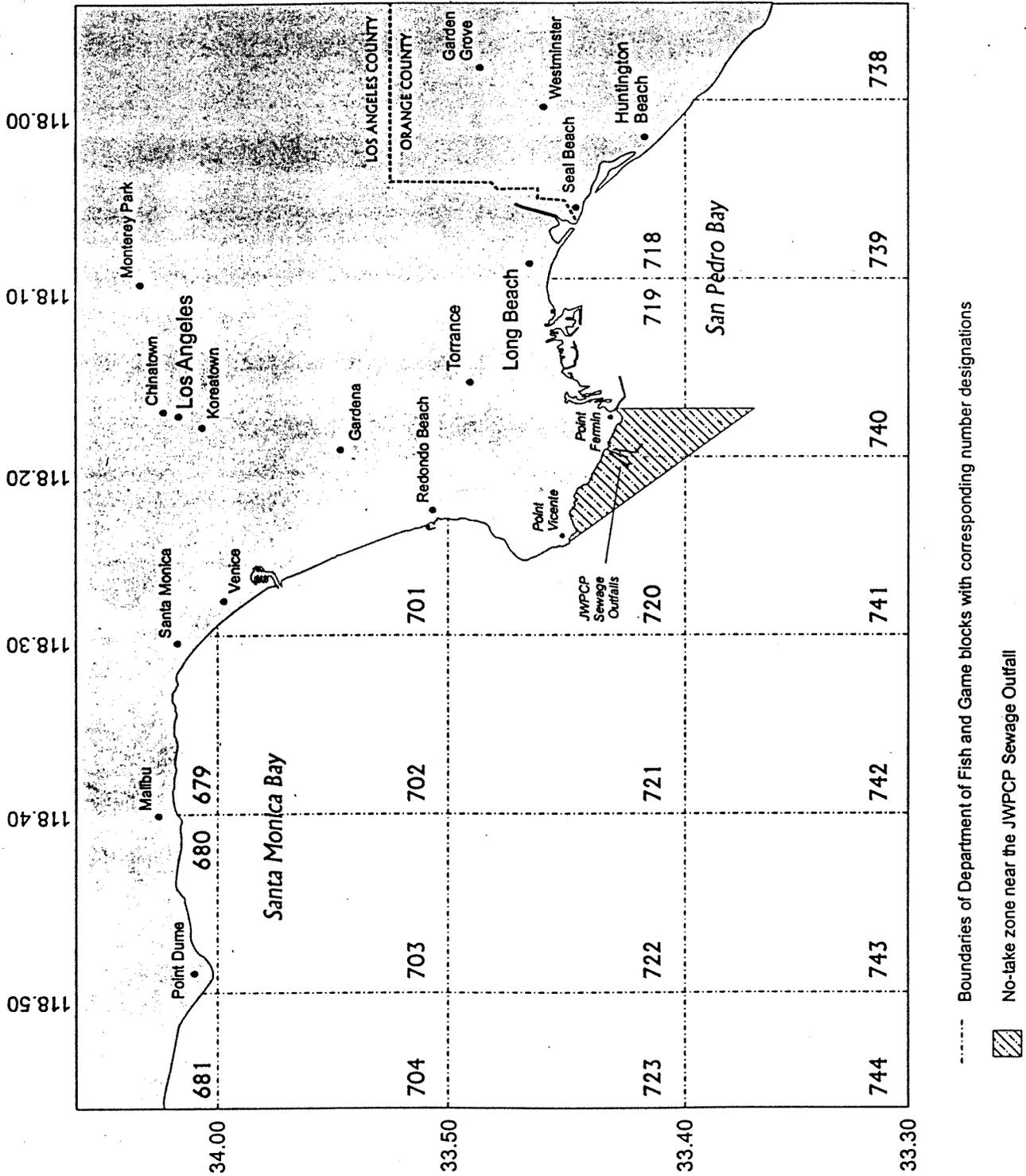


Figure 1-2. Location of the Commercial Fishing Closure Area for White Croaker (adapted from Gold et al., 1997)

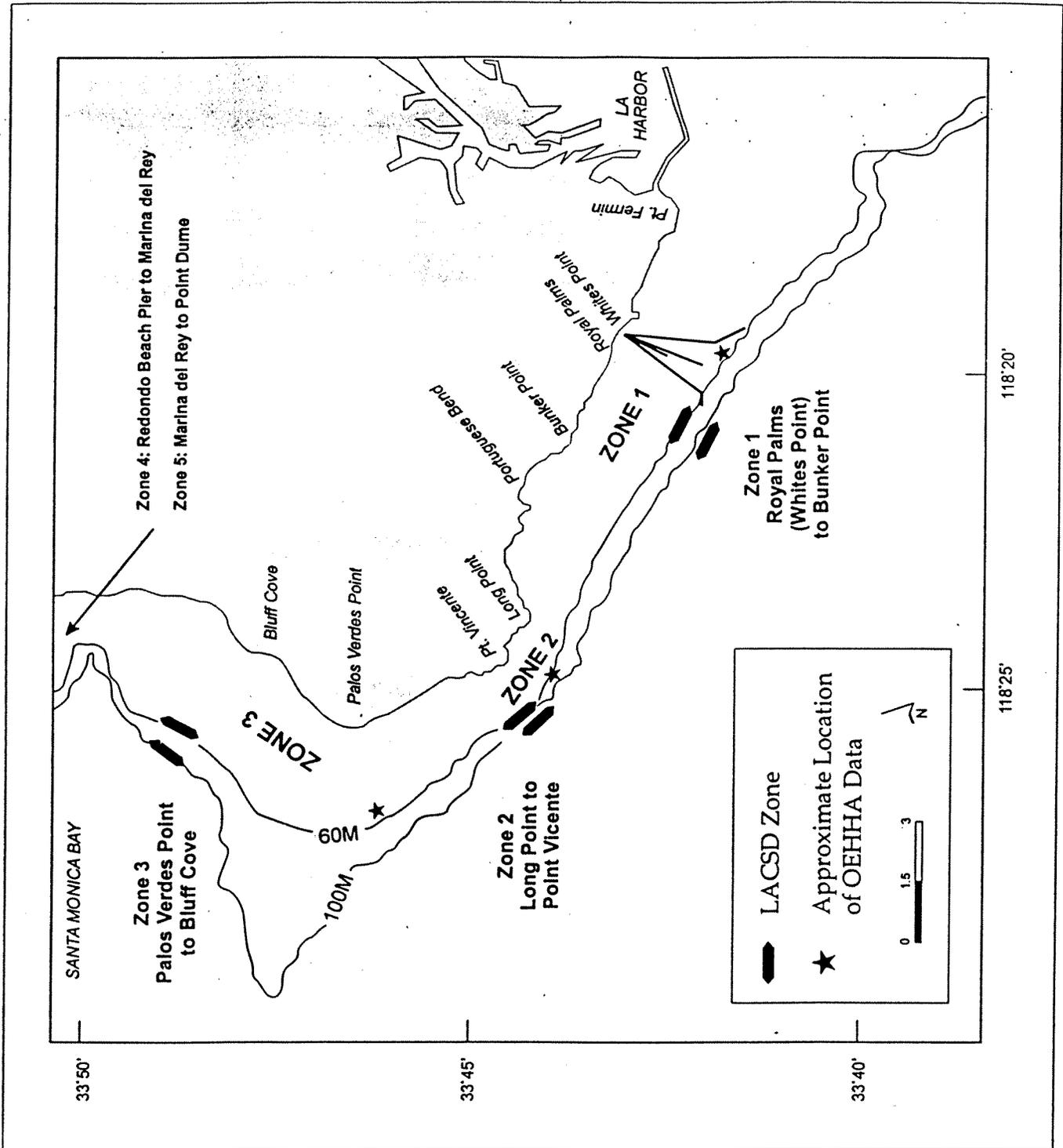


Figure 1-3. Fish Sampling Locations (LACSD, 1998; Pollock et al., 1991)

Figure 2-1
Mean tDDT Concentrations in White Croaker
1985 to 1997 (LACSD)

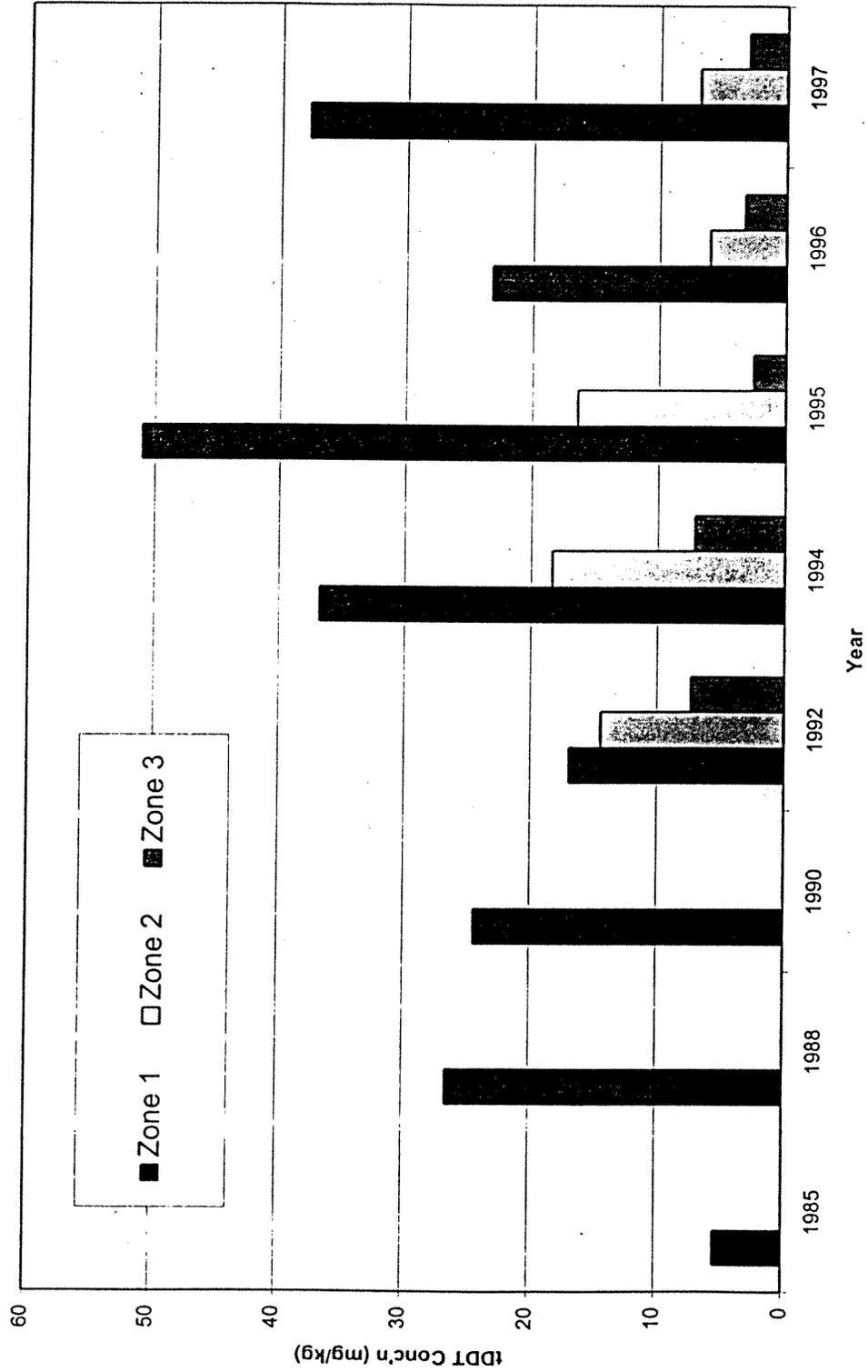


Figure 2-2
Mean tDDT Concentrations in Kelp Bass
1985 to 1996 (LACSD)

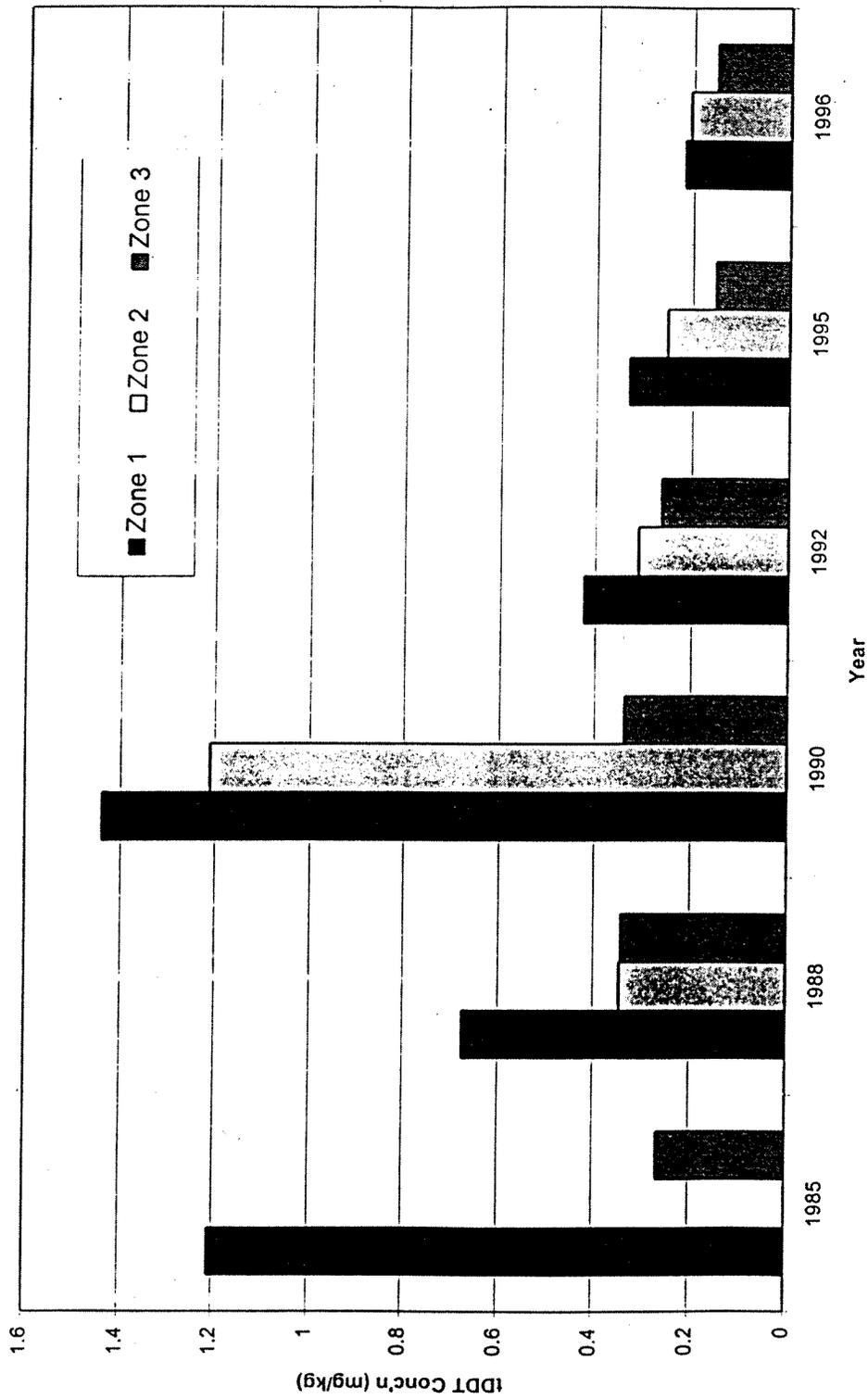


Figure 2-3
Mean PCB Concentrations in White Croaker
1985 to 1997 (LACSD)

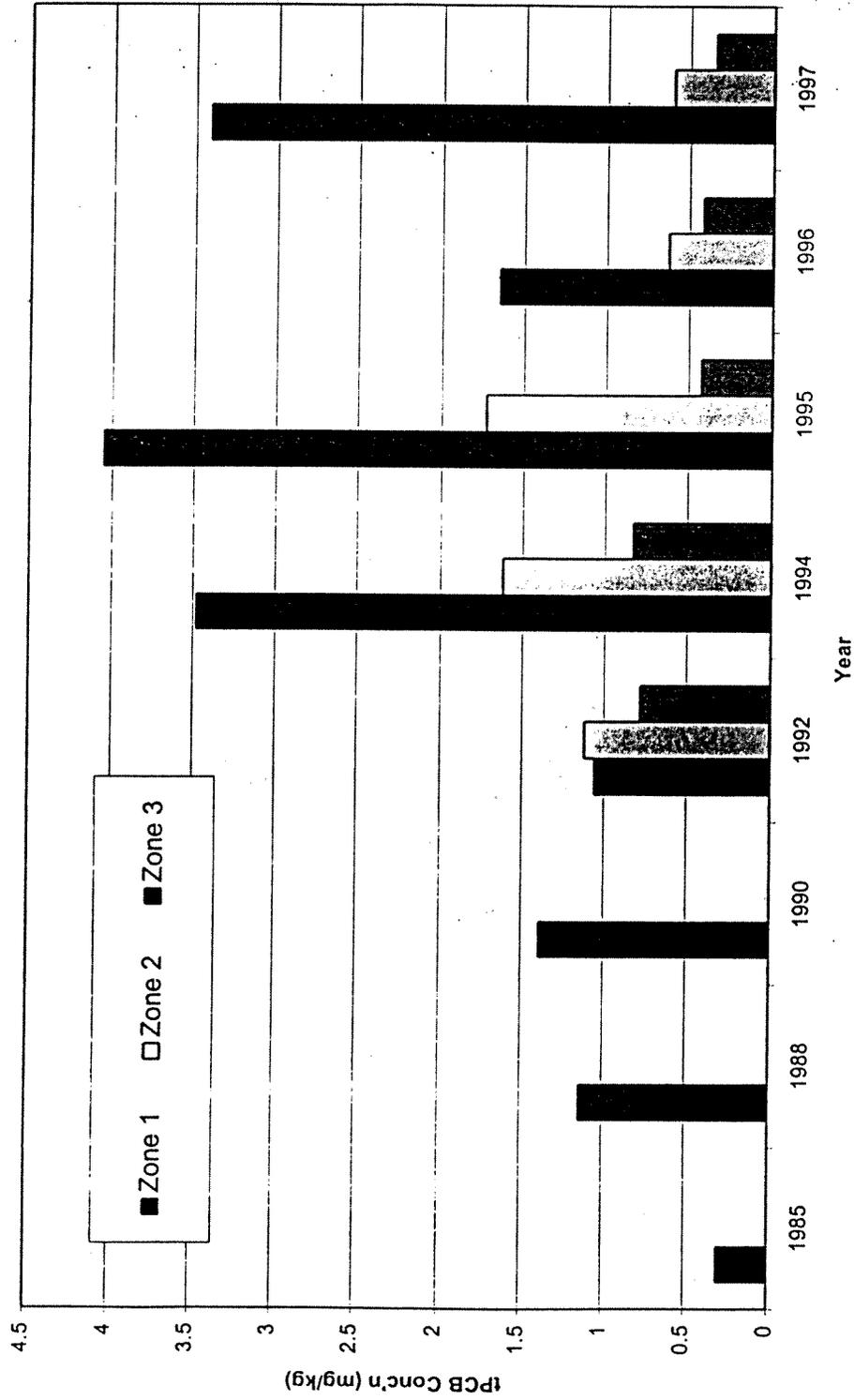


Figure 2-4
Mean PCB Concentrations in Kelp Bass
1985 to 1996 (LACSD)

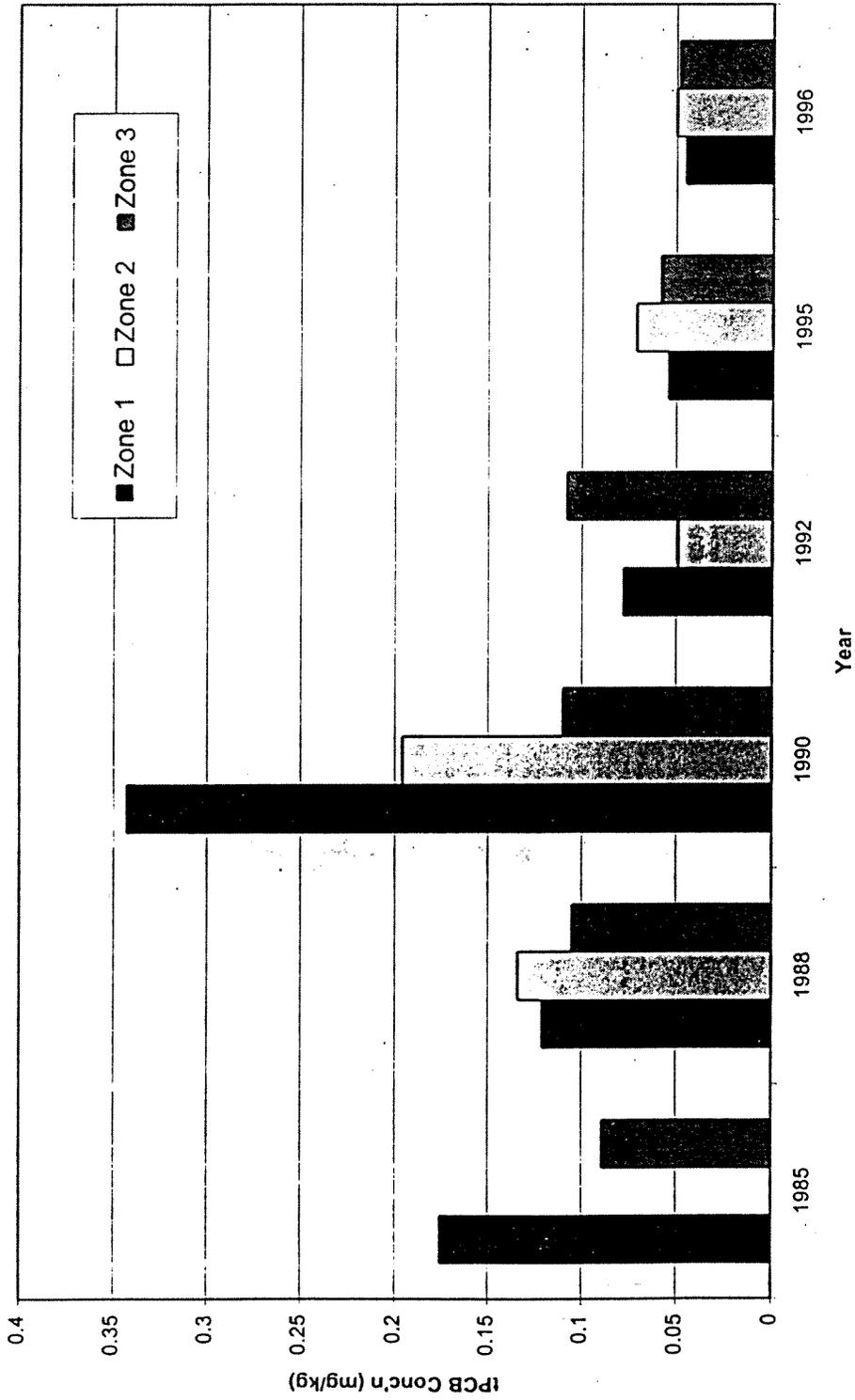


Figure 5-1
Point Estimate Cancer Risk
(RME Scenario)

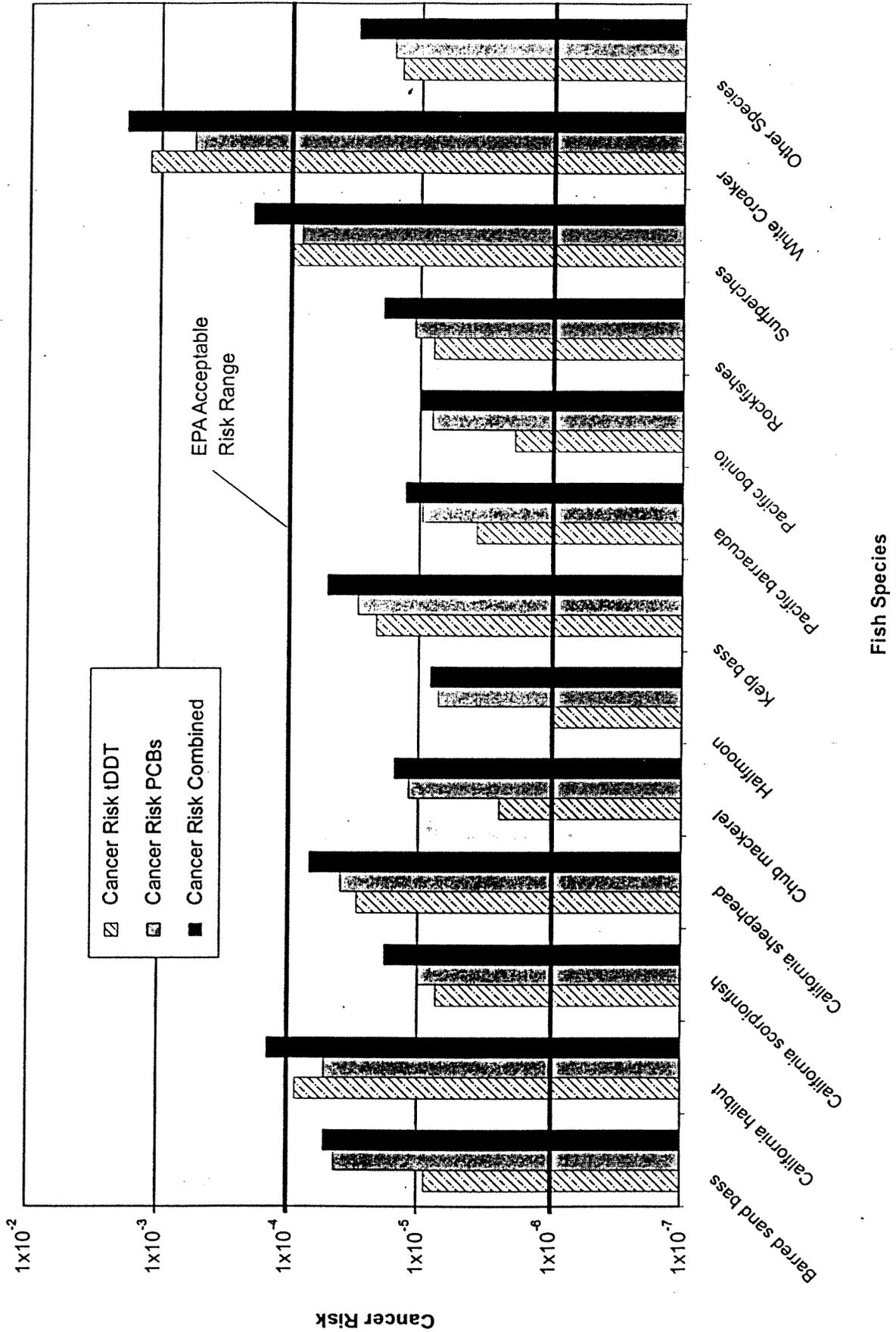


Figure 5-2
Point Estimate Noncancer Hazard
(RME Scenario)

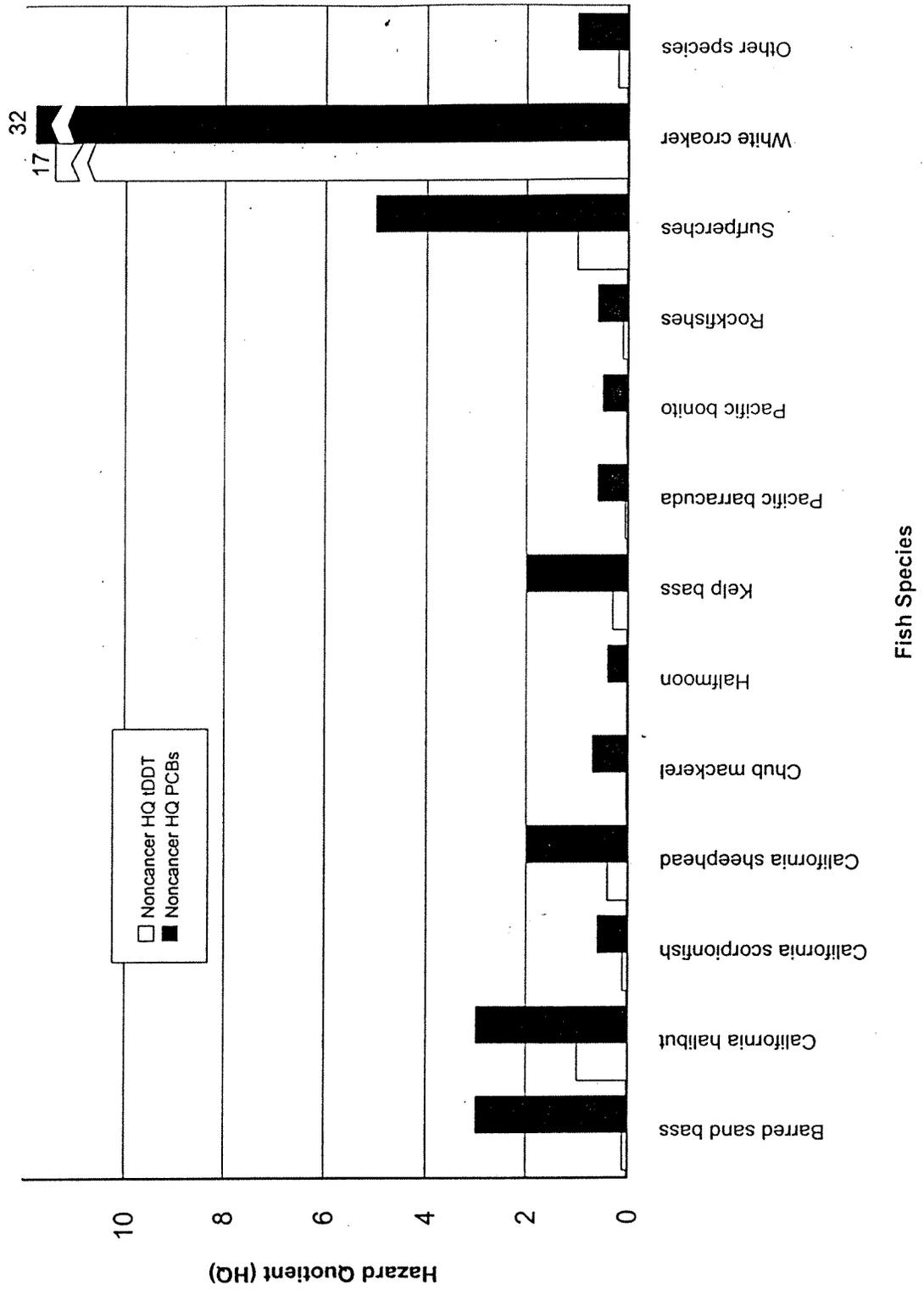
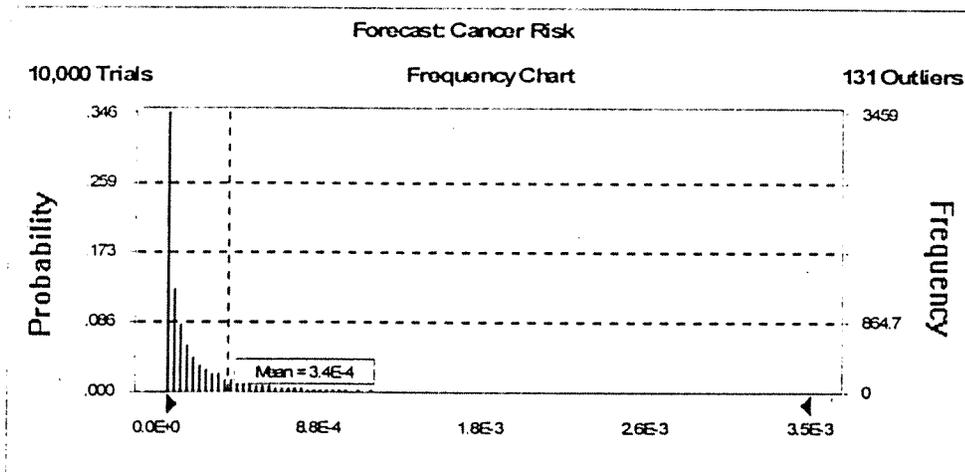


Figure 6-1
Frequency Distribution Graphs for Cancer Risk (tDDT and PCBs Combined)
White Croaker Consumption by Boat Anglers

Summary: Display Range is from 0 to 3.5E-3
 Entire Range is from 3.3E-10 to 5.2E-2
 After 10,000 Trials, the Std. Error of the Mean is 1.1E-5

Statistics:	Trials	10,000
	Mean	3.4E-04
	Median	8.0E-05
	Mode	---
	Standard Deviation	1.1E-03
	Variance	1.2E-06
	Skewness	20.48
	Kurtosis	730.27
	Coeff. of Variability	3.28
	Range Minimum	3.3E-10
	Range Maximum	5.2E-02
	Range Width	5.2E-02
	Mean Std. Error	1.12E-05

Probability
Density
Function
(PDF)



Cumulative
Distribution
Function
(CDF)

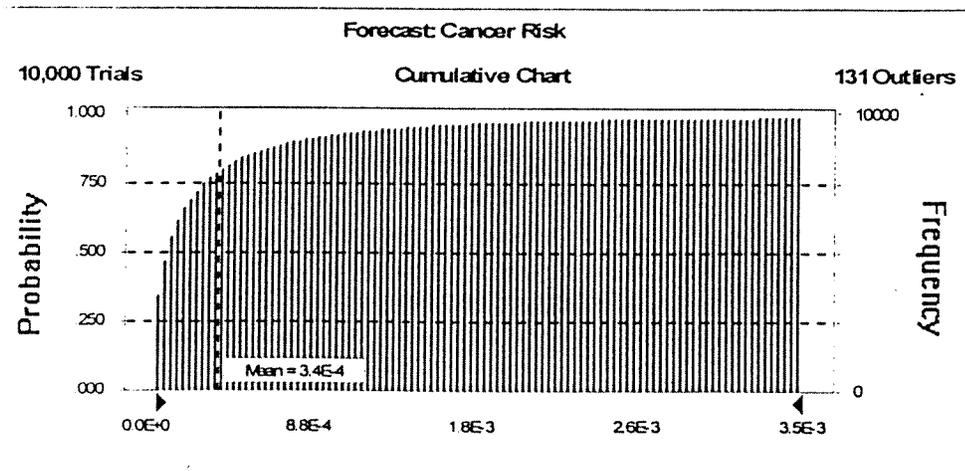
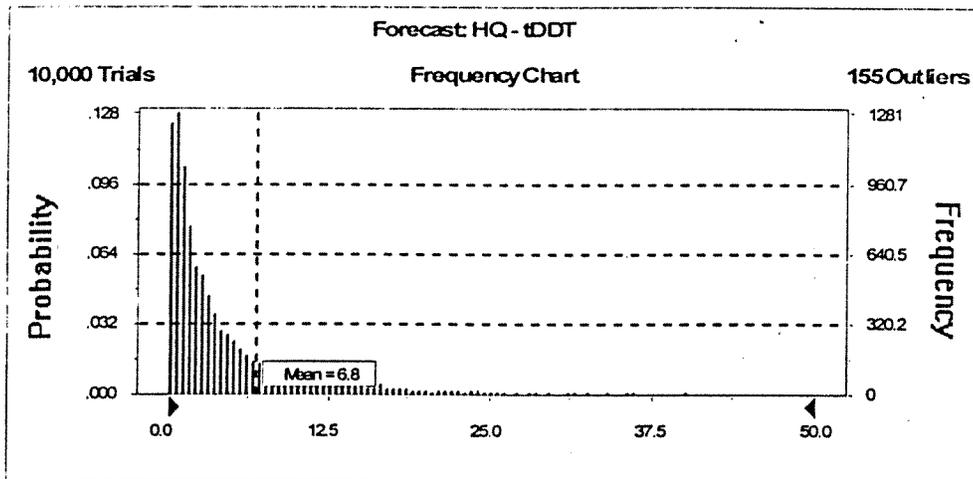


Figure 6-2
Frequency Distribution Graphs for Noncancer Hazard from tDDT
White Croaker Consumption by Boat Anglers

Summary: Display Range is from 0 to 50 µg/kg
 Entire Range is from 0 to 571.5 µg/kg
 After 10,000 Trials, the Std. Error of the Mean is 0.2

Statistics:	Trials	10,000
	Mean	6.8
	Median	2.6
	Mode	---
	Standard Deviation	16.4
	Variance	269.0
	Skewness	12.0
	Kurtosis	261.1
	Coeff. of Variability	2.4
	Range Minimum	0.0
	Range Maximum	571.5
	Range Width	571.5
	Mean Std. Error	0.2

Probability
Distribution
Function
(PDF)



Cumulative
Distribution
Function
(CDF)

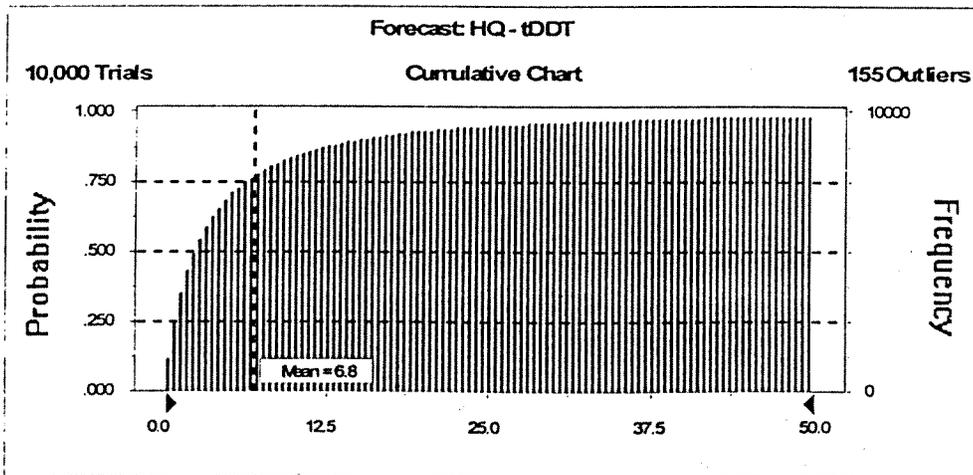
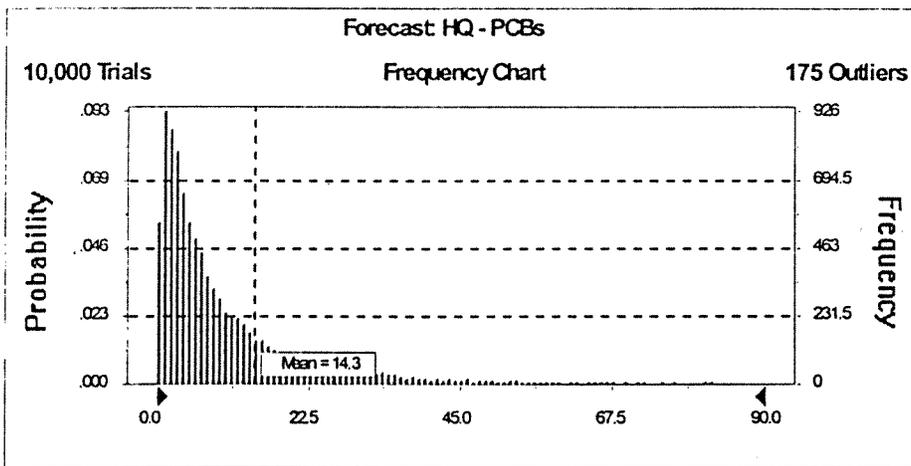


Figure 6-3
Frequency Distribution Graphs for Noncancer Hazard from PCBs
White Croaker Consumption by Boat Anglers

Summary: Display Range is from 0.0 to 90.0
 Entire Range is from 0.1 to 953.4
 After 10,000 Trials, the Std. Error of the Mean is 0.3

Statistics:	Trials	10,000
	Mean	14.3
	Median	6.6
	Mode	---
	Standard Deviation	28.5
	Variance	811.5
	Skewness	11.3
	Kurtosis	245.3
	Coeff. of Variability	2.0
	Range Minimum	0.1
	Range Maximum	953.4
	Range Width	953.3
	Mean Std. Error	0.3

Probability
Density
Function
(PDF)



Cumulative
Distribution
Function
(CDF)

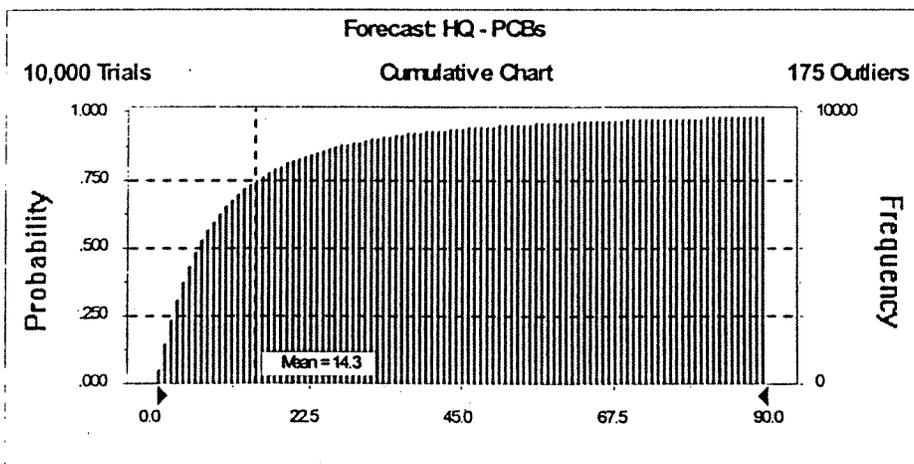
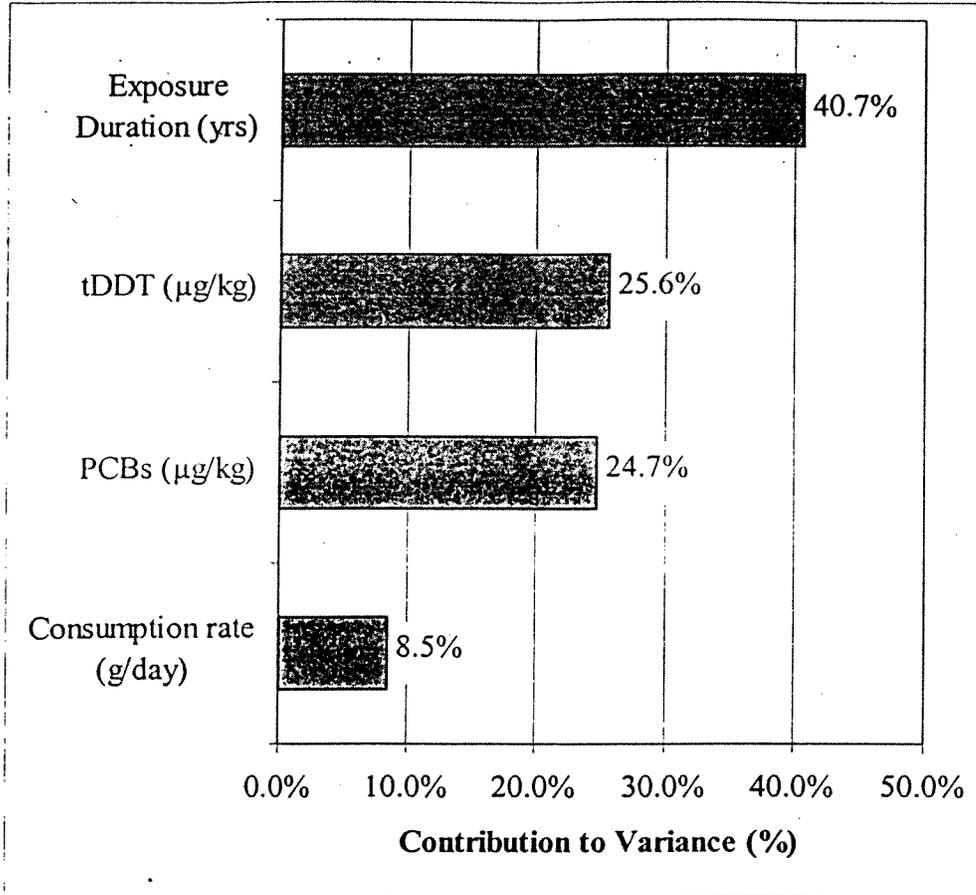
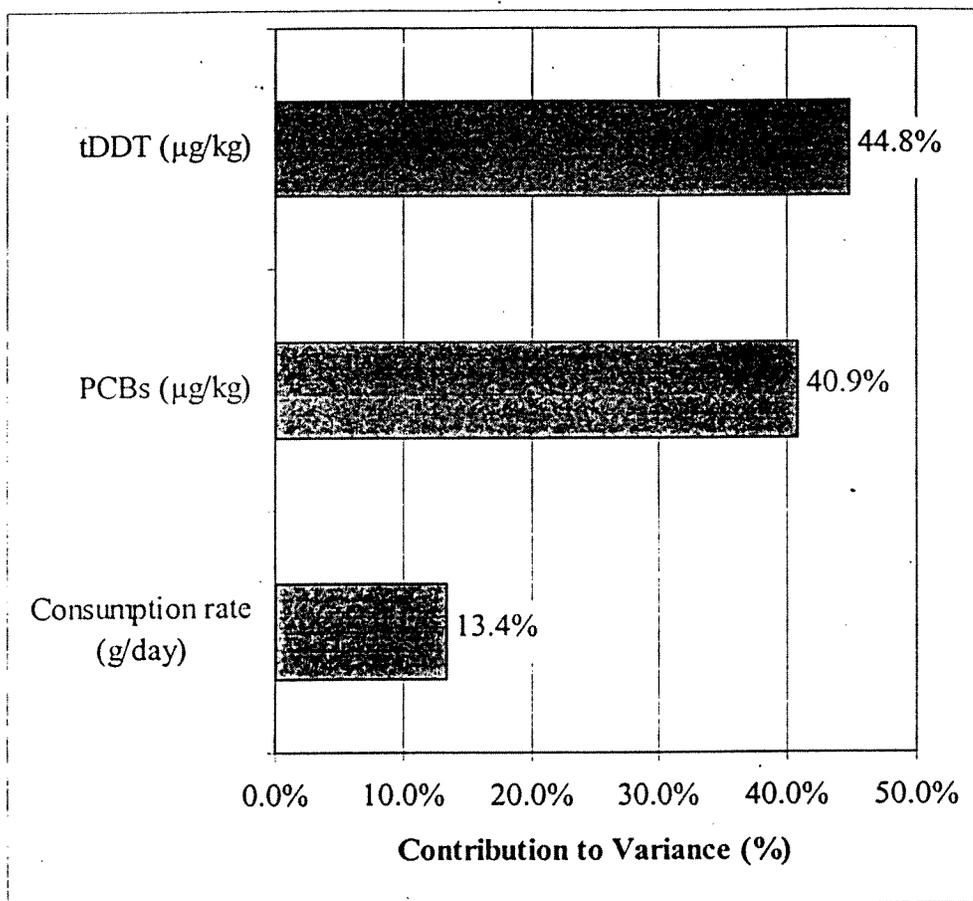


Figure 6-4
Sensitivity Chart for Cancer Risk
White Croaker Consumption by Boat Anglers



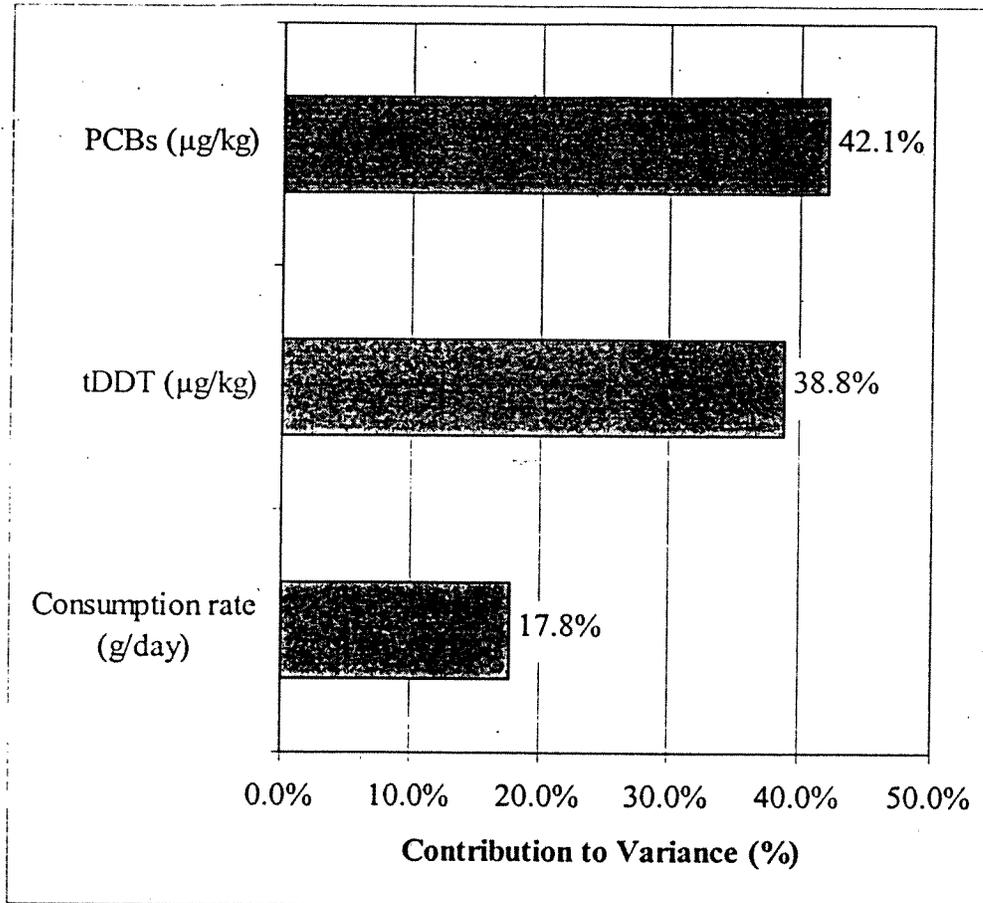
NOTE: tDDT and PCBs are correlated assumptions. Figure shows all parameters that contribute at least 5% to variance.

Figure 6-5
Sensitivity Chart for Noncancer Hazard from tDDT
White Croaker Consumption by Boat Anglers



NOTE: tDDT and PCBs are correlated assumptions. Figure shows all parameters that contribute at least 5% to variance.

Figure 6-6
Sensitivity Chart for Noncancer Hazard from PCBs
White Croaker Consumption by Boat Anglers



NOTE: tDDT and PCBs are correlated assumptions. Figure shows all parameters that contribute at least 5% to variance.

APPENDIX A

FISH TISSUE CONCENTRATION DATA BY SPECIES

Table A-1
Fish Tissue Concentrations - Barred Sand Bass

Sample Date	Sample Location	Composite ID	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1987	LA/Long Beach Harbor Breakwater	CA512	29.5	35.9	Pollock et al. 1991
1987	LA/Long Beach Harbor Breakwater	CA513	81.5	41.1	Pollock et al. 1991
1987	LA/Long Beach Harbor Breakwater	CA514	52.9	20.3	Pollock et al. 1991
1987	LA/Long Beach Harbor Breakwater	CA515	68.7	36.1	Pollock et al. 1991
1987	LA/Long Beach Harbor Breakwater	CA516	56.1	28.6	Pollock et al. 1991
1987	Pier J (Queen Mary)	CA147	47.7	117	Pollock et al. 1991
1987	Pier J (Queen Mary)	CA148	73.9	108	Pollock et al. 1991
1987	Pier J (Queen Mary)	CA149	187	12.9	Pollock et al. 1991
1987	Pier J (Queen Mary)	CA150	97.2	49.3	Pollock et al. 1991
1987	Pier J (Queen Mary)	CA151	48.0	49.9	Pollock et al. 1991

NOTES:

Samples are composites of five fish each.

Table A-2
Fish Tissue Concentrations - California Halibut

Sample Date	Sample Location	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1991	ZoneB	143	30	LACSD
1991	ZoneB	572	100	LACSD
1991	ZoneB	159	70	LACSD
1991	ZoneB	257	40	LACSD
1991	ZoneB	1364	140	LACSD
1991	ZoneB	751	140	LACSD
1991	ZoneB	284	60	LACSD
1991	ZoneB	249	60	LACSD
1991	ZoneB	35	80	LACSD
1991	ZoneB	772	177	LACSD

NOTE: Samples are based on a single fish.

Table A-3
Fish Tissue Concentrations - California Scorpionfish

Sample Date	Sample Location	Composite ID	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1987	Palos Verdes (Northwest Side)	CA487	53.8	16.0	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA488	15.8	5.1	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA489	30.3	8.6	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA490	28.4	9.8	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA491	42.7	6.1	Pollock et al. 1991
1987	White Point	CA167	173.8	21.7	Pollock et al. 1991
1987	White Point	CA168	254.4	52.3	Pollock et al. 1991
1987	White Point	CA169	53.2	27.7	Pollock et al. 1991
1987	White Point	CA170	142.4	42.3	Pollock et al. 1991
1987	White Point	CA171	357.9	44.6	Pollock et al. 1991
1987	Point Vicente	CA777	52.8	5.1	Pollock et al. 1991
1987	Point Vicente	CA778	92.8	ND	Pollock et al. 1991
1987	Point Vicente	CA779	15.5	ND	Pollock et al. 1991
1987	Point Vicente	CA780	27.8	2.5	Pollock et al. 1991
1987	Point Vicente	CA781	41.7	ND	Pollock et al. 1991

NOTES:

ND = Not detected

Samples are composites of five fish each.

For results reported as "ND", one-half of the method detection limit of 38 µg/kg for tDDT, 50 µg/kg for PCBs was used

Table A-4
Fish Tissue Concentrations - Chub Mackerel

Sample Date	Sample Location	Composite ID	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1987	Palos Verdes (Northwest Side)	CA357	4.4	1.5	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA358	21.3	7.7	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA359	15.7	4.2	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA360	15.1	5.5	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA361	21.7	8.7	Pollock et al. 1991
1987	White Point	CA747	70.0	53.0	Pollock et al. 1991
1987	White Point	CA748	10.9	5.7	Pollock et al. 1991
1987	White Point	CA749	5.4	ND	Pollock et al. 1991
1987	White Point	CA750	6.7	1.4	Pollock et al. 1991
1987	White Point	CA751	45.9	34.8	Pollock et al. 1991
1987	Point Vicente	CA622	82.2	57.2	Pollock et al. 1991
1987	Point Vicente	CA623	22.5	8.6	Pollock et al. 1991
1987	Point Vicente	CA624	19.9	9.3	Pollock et al. 1991
1987	Point Vicente	CA625	25.2	9.6	Pollock et al. 1991
1987	Point Vicente	CA626	5.8	2.7	Pollock et al. 1991

NOTES:

ND = Not detected

Samples are composites of five fish each.

For results reported as "ND", one-half of the method detection limit of 38 µg/kg for tDDT, 50 µg/kg for PCBs was used

Table A-5
Fish Tissue Concentrations - Halfmoon

Sample Date	Sample Location	Composite ID	Total DDT ($\mu\text{g}/\text{kg}$)	Total PCB ($\mu\text{g}/\text{kg}$)	Source
1987	Twin Harbor, Catalina	CA982	ND	ND	Pollock et al. 1991
1987	Twin Harbor, Catalina	CA983	ND	ND	Pollock et al. 1991
1987	Twin Harbor, Catalina	CA984	ND	ND	Pollock et al. 1991
1987	Twin Harbor, Catalina	CA985	ND	ND	Pollock et al. 1991
1987	Twin Harbor, Catalina	CA986	ND	ND	Pollock et al. 1991

NOTES:

ND = Not detected

Samples are composites of five fish each.

For results reported as "ND", one-half of the method detection limit of 38 $\mu\text{g}/\text{kg}$ for tDDT, 50 $\mu\text{g}/\text{kg}$ for PCBs was used

Table A-6
Fish Tissue Concentrations - Kelp Bass

Sample Date	Sample Location	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1995	Zone 1	60	ND	LACSD
1995	Zone 1	190	40	LACSD
1995	Zone 1	680	90	LACSD
1995	Zone 1	200	40	LACSD
1995	Zone 1	170	30	LACSD
1995	Zone 1	180	30	LACSD
1995	Zone 1	100	30	LACSD
1995	Zone 1	180	30	LACSD
1995	Zone 1	450	80	LACSD
1995	Zone 1	1120	120	LACSD
1995	Zone 2	720	140	LACSD
1995	Zone 2	430	80	LACSD
1995	Zone 2	620	100	LACSD
1995	Zone 2	70	30	LACSD
1995	Zone 2	120	40	LACSD
1995	Zone 2	160	30	LACSD
1995	Zone 2	200	80	LACSD
1995	Zone 2	100	ND	LACSD
1995	Zone 2	80	ND	LACSD
1995	Zone 2	30	ND	LACSD
1995	Zone 3	80	ND	LACSD
1995	Zone 3	30	ND	LACSD
1995	Zone 3	70	ND	LACSD
1995	Zone 3	20	ND	LACSD
1995	Zone 3	170	50	LACSD
1995	Zone 3	240	40	LACSD
1995	Zone 3	160	10	LACSD
1995	Zone 3	150	30	LACSD

Table A-6
Fish Tissue Concentrations - Kelp Bass (Continued)

Sample Date	Sample Location	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1995	Zone 3	570	160	LACSD
1995	Zone 3	30	ND	LACSD
1996	Zone 1	110	20	LACSD
1996	Zone 1	810	120	LACSD
1996	Zone 1	220	50	LACSD
1996	Zone 1	180	50	LACSD
1996	Zone 1	50	20	LACSD
1996	Zone 1	170	40	LACSD
1996	Zone 1	100	20	LACSD
1996	Zone 1	210	40	LACSD
1996	Zone 1	220	50	LACSD
1996	Zone 1	110	40	LACSD
1996	Zone 2	190	50	LACSD
1996	Zone 2	800	130	LACSD
1996	Zone 2	270	60	LACSD
1996	Zone 2	30	10	LACSD
1996	Zone 2	180	60	LACSD
1996	Zone 2	80	30	LACSD
1996	Zone 2	50	10	LACSD
1996	Zone 2	170	60	LACSD
1996	Zone 2	70	30	LACSD
1996	Zone 2	220	60	LACSD
1996	Zone 3	330	70	LACSD
1996	Zone 3	140	50	LACSD
1996	Zone 3	120	40	LACSD
1996	Zone 3	60	20	LACSD
1996	Zone 3	20	20	LACSD
1996	Zone 3	130	50	LACSD

Table A-6
Fish Tissue Concentrations - Kelp Bass (Continued)

Sample Date	Sample Location	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1996	Zone 3	130	60	LACSD
1996	Zone 3	30	20	LACSD
1996	Zone 3	90	20	LACSD
1996	Zone 3	460	130	LACSD

NOTES:

ND = Not detected

Samples are based on a single fish.

For results reported as "ND", one-half of the method detection limit of 38 µg/kg for tDDT, 50 µg/kg for PCBs was used

Table A-7
Fish Tissue Concentrations - Pacific Barracuda

Sample Date	Sample Location	Composite ID	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1987	Fourteen Mile Bank	CA192	34.0	24.0	Pollock et al. 1991
1987	Fourteen Mile Bank	CA193	50.7	13.7	Pollock et al. 1991
1987	Fourteen Mile Bank	CA194	11.5	18.8	Pollock et al. 1991
1987	Fourteen Mile Bank	CA195	8.7	12.4	Pollock et al. 1991
1987	Fourteen Mile Bank	CA196	20.0	13.1	Pollock et al. 1991

NOTE: Samples are composites of five fish each.

Table A-8
Fish Tissue Concentrations - Pacific Bonito

Sample Date	Sample Location	Composite ID	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1987	Palos Verdes (Northwest Side)	CA412	16.7	6.6	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA413	24.5	10.5	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA414	17.6	6.8	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA415	13.9	5.3	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA416	12.5	4.3	Pollock et al. 1991
1987	White Point	CA612	7.1	1.7	Pollock et al. 1991
1987	White Point	CA613	27.6	15.1	Pollock et al. 1991
1987	White Point	CA614	147.1	111.5	Pollock et al. 1991
1987	White Point	CA615	28.0	2.1	Pollock et al. 1991
1987	White Point	CA616	26.1	21.5	Pollock et al. 1991
1987	Point Vicente	CA667	64.7	29.1	Pollock et al. 1991
1987	Point Vicente	CA668	17.8	7.0	Pollock et al. 1991
1987	Point Vicente	CA669	27.3	10.6	Pollock et al. 1991
1987	Point Vicente	CA670	20.6	7.9	Pollock et al. 1991
1987	Point Vicente	CA671	35.7	12.4	Pollock et al. 1991

NOTE: Samples are composites of five fish each.

Table A-10
Fish Tissue Concentrations - Surfperches

Sample Date	Sample Location	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1996	Zone 1	350	50	LACSD
1996	Zone 1	580	60	LACSD
1996	Zone 1	360	40	LACSD
1996	Zone 1	150	20	LACSD
1996	Zone 1	200	30	LACSD
1996	Zone 1	200	10	LACSD
1996	Zone 1	240	30	LACSD
1996	Zone 1	100	ND	LACSD
1996	Zone 1	530	60	LACSD
1996	Zone 1	470	40	LACSD
1996	Zone 2	380	40	LACSD
1996	Zone 2	1310	150	LACSD
1996	Zone 2	170	10	LACSD
1996	Zone 2	1130	120	LACSD
1996	Zone 2	330	40	LACSD
1996	Zone 2	690	70	LACSD
1996	Zone 2	510	40	LACSD
1996	Zone 2	450	60	LACSD
1996	Zone 2	130	ND	LACSD
1996	Zone 2	520	50	LACSD
1996	Zone 3	120	ND	LACSD
1996	Zone 3	180	ND	LACSD
1996	Zone 3	310	80	LACSD
1996	Zone 3	220	10	LACSD
1996	Zone 3	80	ND	LACSD
1996	Zone 3	40	ND	LACSD
1996	Zone 3	40	ND	LACSD
1996	Zone 3	40	ND	LACSD
1996	Zone 3	140	ND	LACSD
1996	Zone 3	130	ND	LACSD

NOTES: ND = Not detected Samples are based on a single fish.
For PCBs, the lowest reported value is 10 µg/kg; therefore, all NDs were assumed to be present at ½ of 10 µg/kg, or 5 µg/kg.

Table A-9
Fish Tissue Concentrations - Rockfishes

Sample Date	Sample Location	Composite ID	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1987	Palos Verdes (Northwest Side)	CA567	158.4	21.6	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA568	67.7	15.0	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA569	48.1	7.5	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA570	39.5	6.5	Pollock et al. 1991
1987	Palos Verdes (Northwest Side)	CA571	40.4	7.0	Pollock et al. 1991
1987	White Point	CA877	27.7	ND	Pollock et al. 1991
1987	White Point	CA878	28.4	ND	Pollock et al. 1991
1987	White Point	CA879	189.5	16.6	Pollock et al. 1991
1987	White Point	CA880	194.8	37.9	Pollock et al. 1991
1987	White Point	CA881	65.7	ND	Pollock et al. 1991
1987	White Point	CA882	382.6	53.4	Pollock et al. 1991
1987	White Point	CA883	25.9	ND	Pollock et al. 1991
1987	White Point	CA884	28.3	10.0	Pollock et al. 1991
1987	White Point	CA885	51.0	36.3	Pollock et al. 1991
1987	White Point	CA886	135.9	62.9	Pollock et al. 1991
1987	Point Vicente	CB023	72.6	16.5	Pollock et al. 1991
1987	Point Vicente	CB024	62.7	14.6	Pollock et al. 1991
1987	Point Vicente	CB025	86.3	14.6	Pollock et al. 1991
1987	Point Vicente	CB026	66.8	19.5	Pollock et al. 1991
1987	Point Vicente	CB027	108.6	21.6	Pollock et al. 1991
1987	Point Vicente	CB028	57.8	7.1	Pollock et al. 1991
1987	Point Vicente	CB029	110.8	17.7	Pollock et al. 1991
1987	Point Vicente	CB030	117.9	20.4	Pollock et al. 1991
1987	Point Vicente	CB031	96.1	16.9	Pollock et al. 1991
1987	Point Vicente	CB032	37.3	7.2	Pollock et al. 1991

NOTES: ND = Not detected Samples are composites of five fish each
For results reported as "ND", one-half of the method detection limit of 38 µg/kg for tDDT, 50 µg/kg for PCBs was used.

Table A-11
Fish Tissue Concentrations - White Croaker

Sample Date	Sample Location	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1996	Zone 1	30,300	1,880	LACSD
1996	Zone 1	42,000	2,510	LACSD
1996	Zone 1	27,300	2,050	LACSD
1996	Zone 1	20,490	1,360	LACSD
1996	Zone 1	59,600	4,490	LACSD
1996	Zone 1	4,500	520	LACSD
1996	Zone 1	3,950	310	LACSD
1996	Zone 1	20,950	1,550	LACSD
1996	Zone 1	2,460	250	LACSD
1996	Zone 1	21,800	1,580	LACSD
1996	Zone 2	10,130	990	LACSD
1996	Zone 2	9,850	870	LACSD
1996	Zone 2	2,130	490	LACSD
1996	Zone 2	3,780	330	LACSD
1996	Zone 2	8,090	900	LACSD
1996	Zone 2	6,560	700	LACSD
1996	Zone 2	6,100	710	LACSD
1996	Zone 2	6,130	640	LACSD
1996	Zone 2	1,950	210	LACSD
1996	Zone 2	5,240	420	LACSD
1996	Zone 3	4,350	600	LACSD
1996	Zone 3	4,780	590	LACSD
1996	Zone 3	1,310	210	LACSD
1996	Zone 3	470	90	LACSD
1996	Zone 3	3,220	340	LACSD
1996	Zone 3	3,210	480	LACSD
1996	Zone 3	2,620	370	LACSD
1996	Zone 3	4,660	590	LACSD
1996	Zone 3	6,720	730	LACSD
1996	Zone 3	1,200	150	LACSD
1997	Zone 1	58,700	3,500	LACSD

Table A-11
Fish Tissue Concentrations - White Croaker (Continued)

Sample Date	Sample Location	Total DDT (µg/kg)	Total PCB (µg/kg)	Source
1997	Zone 1	44,300	3,210	LACSD
1997	Zone 1	33,800	3,180	LACSD
1997	Zone 1	24,600	2,430	LACSD
1997	Zone 1	51,900	5,610	LACSD
1997	Zone 1	57,000	5,630	LACSD
1997	Zone 1	32,660	2,910	LACSD
1997	Zone 1	19,880	1,880	LACSD
1997	Zone 1	21,800	2,700	LACSD
1997	Zone 1	32,900	3,050	LACSD
1997	Zone 2	3,330	300	LACSD
1997	Zone 2	11,770	1,130	LACSD
1997	Zone 2	2,480	260	LACSD
1997	Zone 2	17,990	970	LACSD
1997	Zone 2	6,210	680	LACSD
1997	Zone 2	2,020	190	LACSD
1997	Zone 2	7,630	770	LACSD
1997	Zone 2	9,040	810	LACSD
1997	Zone 2	4,710	520	LACSD
1997	Zone 2	3,260	330	LACSD
1997	Zone 3	4,670	500	LACSD
1997	Zone 3	1,160	260	LACSD
1997	Zone 3	9,940	860	LACSD
1997	Zone 3	2,750	350	LACSD
1997	Zone 3	3,420	320	LACSD
1997	Zone 3	2,270	350	LACSD
1997	Zone 3	1,120	200	LACSD
1997	Zone 3	1,670	310	LACSD
1997	Zone 3	2,130	230	LACSD
1997	Zone 3	560	70	LACSD

NOTE: Samples are based on a single fish.

APPENDIX B

MONTE CARLO SIMULATION RESULTS

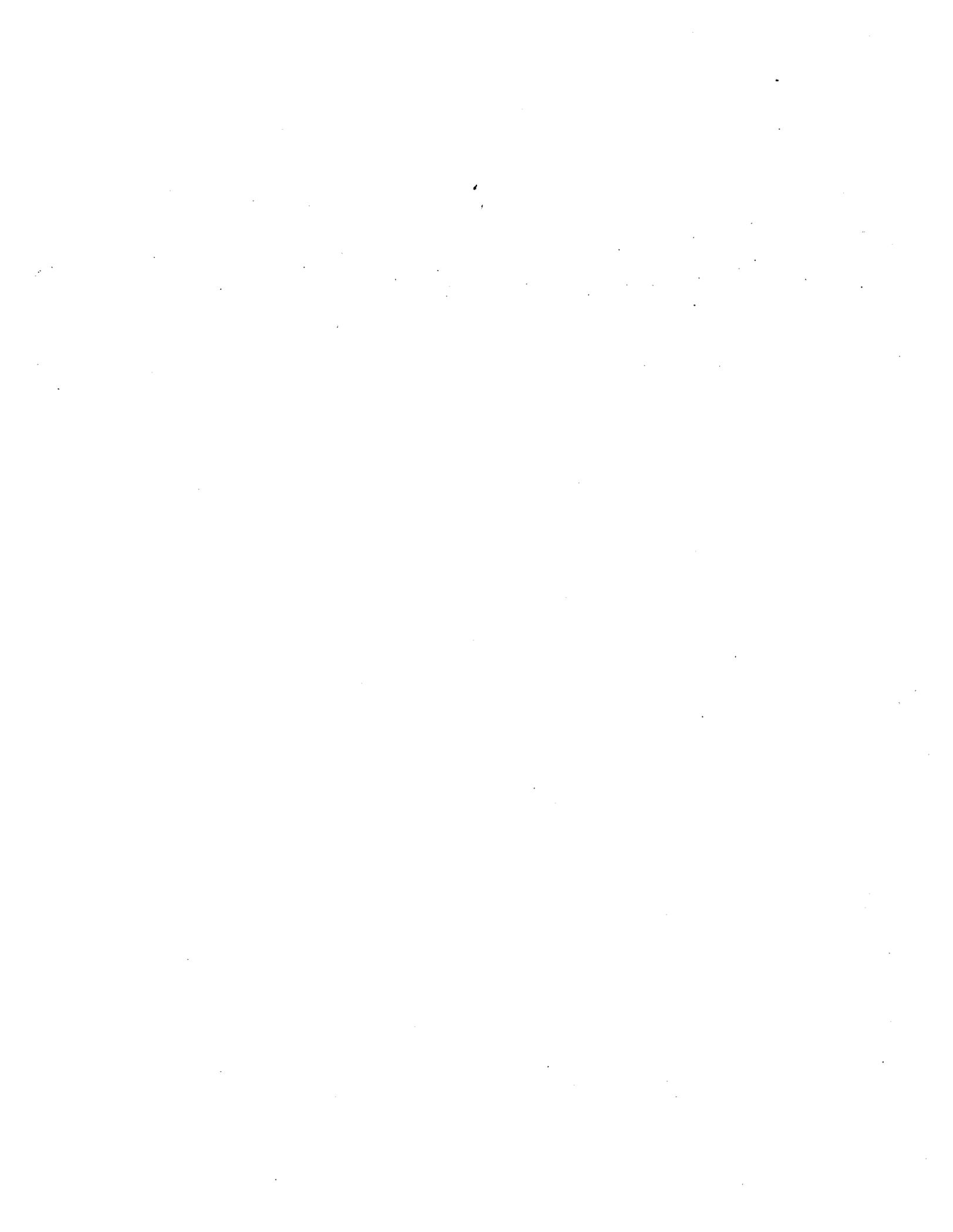


Table B-1
Input Distribution Assumptions

Assumption: tDDT Conc'n in White Croaker ($\mu\text{g}/\text{kg}$)

Lognormal distribution with parameters:

Mean 14,007.12

Standard Dev. 25,793.71

Selected range is from 0.00 to +Infinity

Mean value in simulation was 14,038.73

Correlated with:

PCB ($\mu\text{g}/\text{kg}$) (C9) 0.96

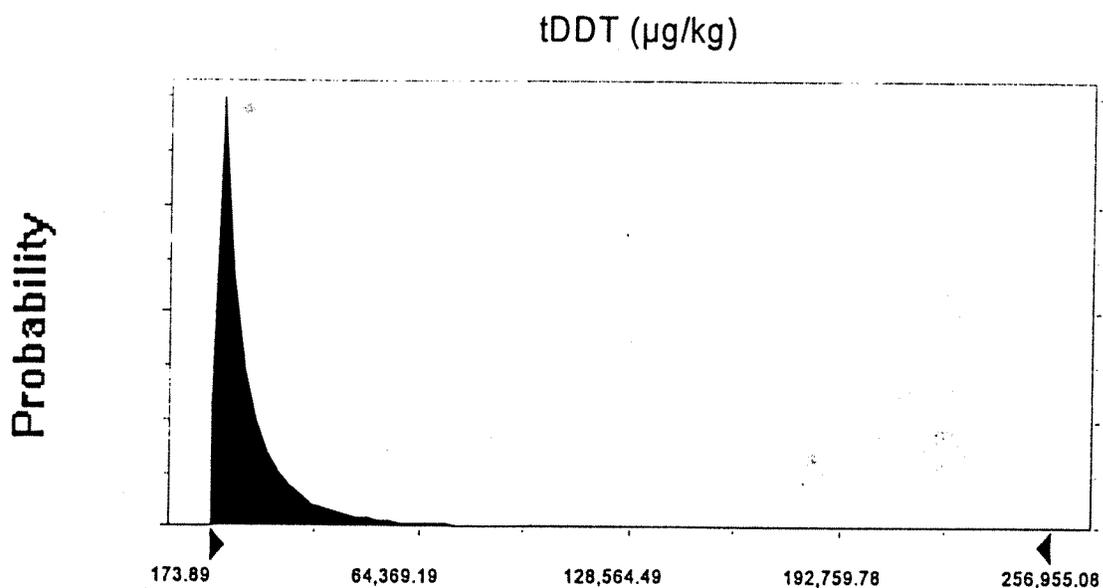


Table B-1
Input Distribution Assumptions (continued)

Assumption: PCB Conc'n in White Croaker ($\mu\text{g}/\text{kg}$)

Lognormal distribution with parameters:

Mean 1,174.68

Standard Dev. 1,599.25

Selected range is from 0.00 to +Infinity

Mean value in simulation was 1,176.17

Correlated with:

tDDT ($\mu\text{g}/\text{kg}$) (B9) 0.96

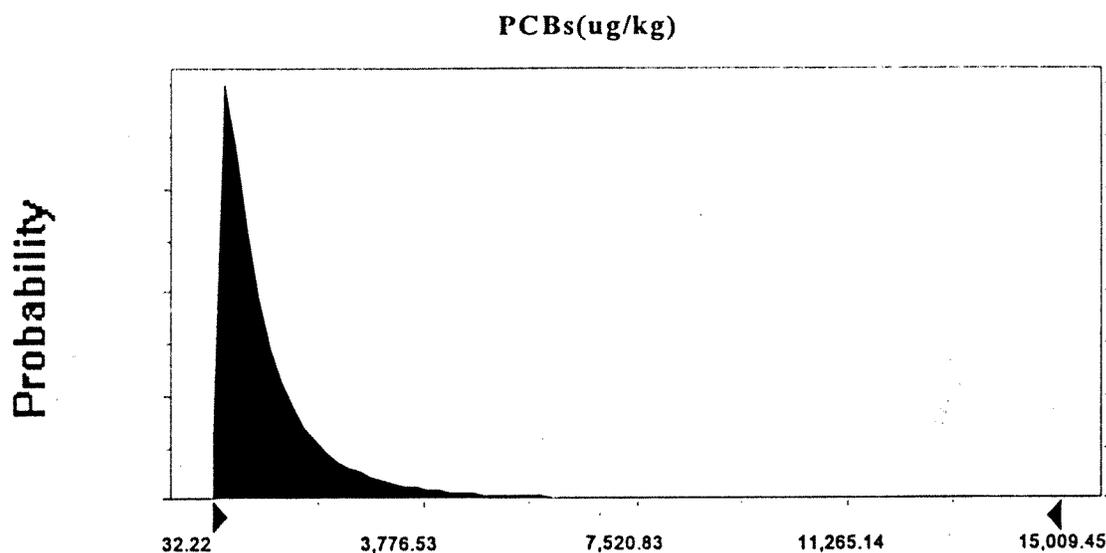


Table B-1
Input Distribution Assumptions (continued)

Assumption: White Croaker Consumption Rate (g/day)

Lognormal distribution with parameters:

Mean 16.70

Standard Dev. 12.92

Selected range is from 0.00 to +Infinity

Mean value in simulation was 16.74

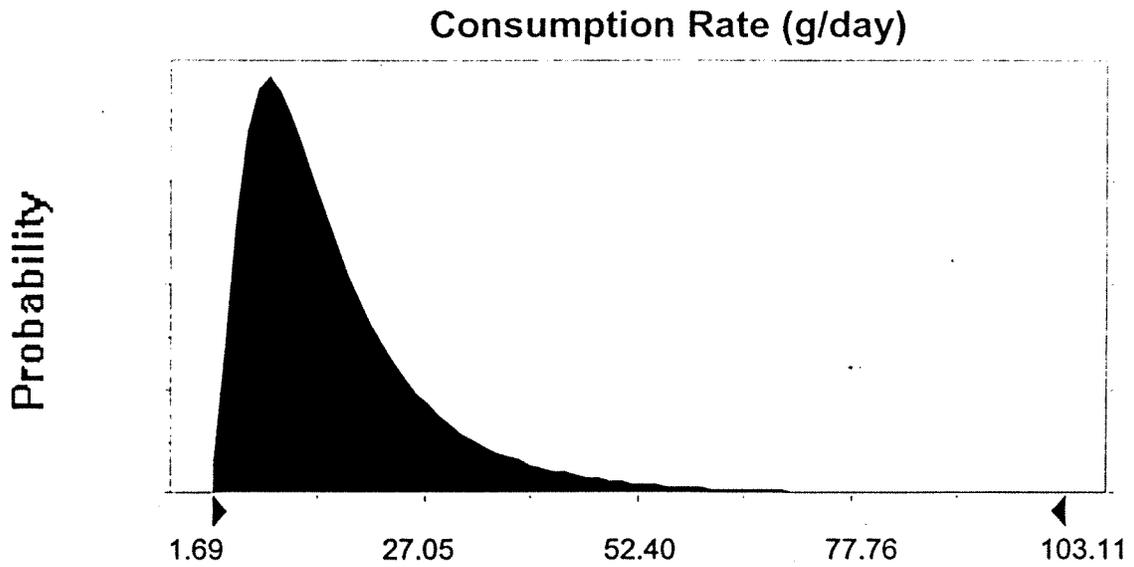


Table B-1
Input Distribution Assumptions (continued)

Assumption: Exposure Duration (yrs)		<u>Relative Prob.</u>
Custom distribution with parameters:		
Single point	0.00	0.02
Single point	0.00	9.00
Single point	0.04	1.00
Single point	0.08	3.00
Single point	0.17	4.00
Single point	0.25	6.02
Single point	0.50	1.00
Single point	0.75	1.00
Single point	1.00	26.08
Single point	1.50	2.01
Single point	2.00	18.06
Single point	2.50	2.01
Single point	3.00	24.07
Single point	3.50	1.00
Single point	4.00	15.05
Single point	5.00	17.05
Single point	6.00	5.02
Single point	7.00	8.02
Single point	8.00	5.02
Single point	9.00	1.00
Single point	10.00	33.10
Single point	11.00	3.01
Single point	12.00	8.02
Single point	13.00	3.01
Single point	14.00	4.01
Single point	15.00	20.06
Single point	16.00	3.01
Single point	17.00	4.01
Single point	18.00	1.00
Single point	20.00	29.09
Single point	21.00	1.00
Single point	22.00	2.01

Assumption: Exposure Duration (yrs)

Custom distribution with parameters:		<u>Relative Prob.</u>
Single point	23.00	1.00
Single point	25.00	14.04
Single point	26.00	1.00
Single point	27.00	1.00
Single point	28.00	1.00
Single point	29.00	2.01
Single point	30.00	27.08
Single point	31.00	1.00
Single point	32.00	2.01
Single point	33.00	2.01
Single point	35.00	7.02
Single point	40.00	6.02
Single point	42.00	1.00
Single point	45.00	1.00
Single point	50.00	3.01
Single point	60.00	5.02
Single point	65.00	1.00
Total Relative Probability		337.00

Mean value in simulation was 13.77

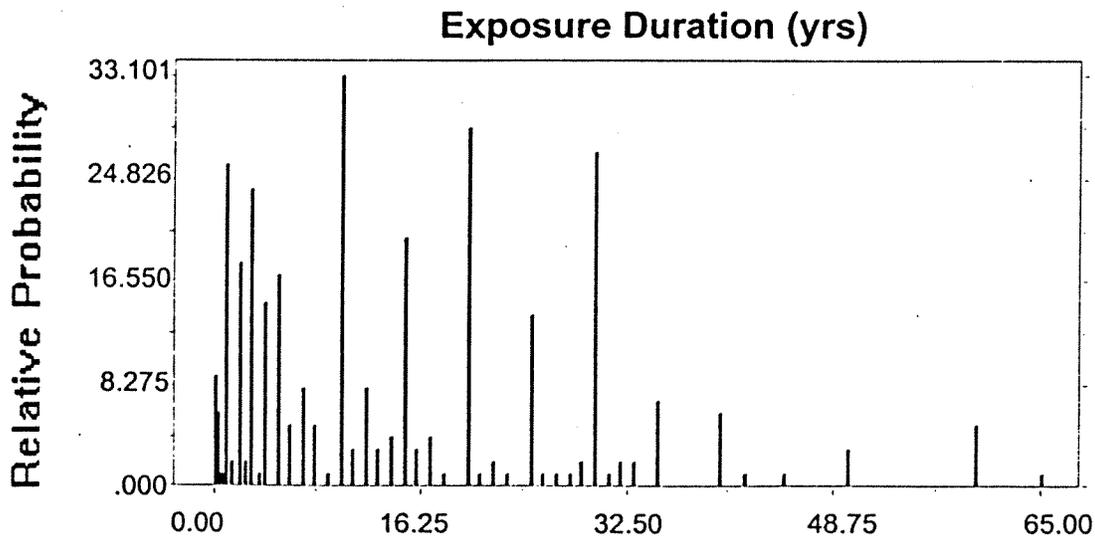


Table B-1
Input Distribution Assumptions (continued)

Assumption: Body Weight (kg)

Lognormal distribution with parameters:

Mean 71.00

Standard Dev. 15.90

Mean value in simulation was 71.21

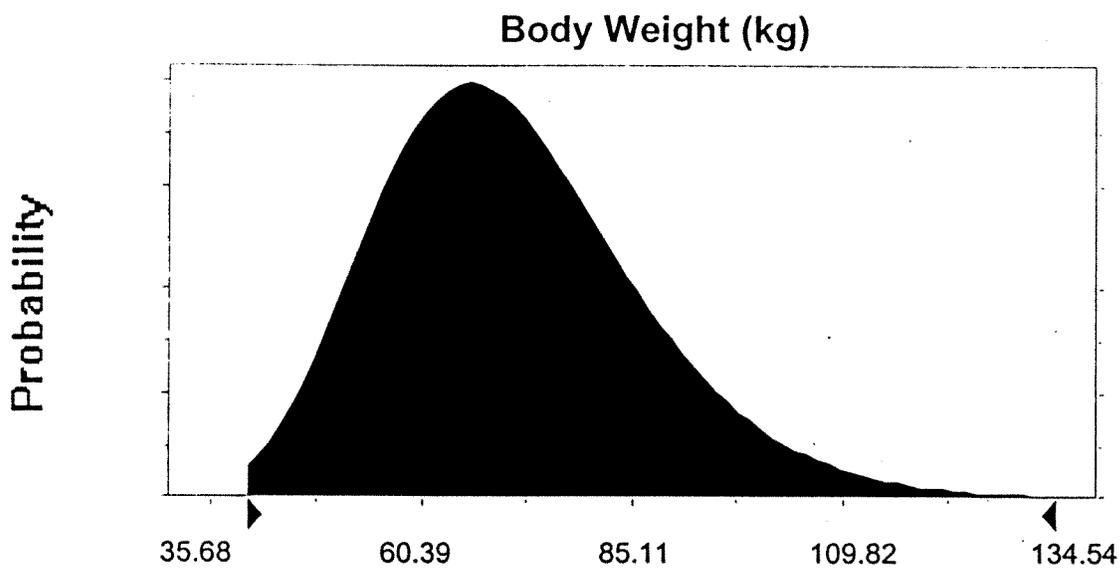


Table B-2
Summary Statistics - Output Parameters

Statistics	Cancer Risk - tDDT	Cancer Risk - PCBs	Cancer Risk - Combined	HQ - tDDT	HQ - PCBs
Trials	10,000	10,000	10,000	10,000	10,000
Mean	2.28×10^{-4}	1.13×10^{-4}	3.40×10^{-4}	6.8	14.3
Median	4.86×10^{-5}	3.07×10^{-5}	8.01×10^{-5}	2.6	6.6
Mode	---	---	---	---	---
Standard Deviation	8.07×10^{-4}	3.24×10^{-4}	1.12×10^{-3}	16.4	28.5
Variance	6.50×10^{-7}	1.05×10^{-7}	1.25×10^{-6}	269.0	811.5
Skewness	22.71	16.33	20.48	12.00	11.35
Kurtosis	890.07	467.24	730.27	261.13	245.28
Coeff. of Variability	3.54	2.88	3.28	2.40	1.99
Range Minimum	1.90×10^{-10}	1.37×10^{-10}	3.28×10^{-10}	0.0	0.1
Range Maximum	4.06×10^{-2}	1.23×10^{-2}	5.22×10^{-2}	571.5	953.4
Range Width	4.06×10^{-2}	1.23×10^{-2}	5.22×10^{-2}	571.5	953.3
Mean Std. Error	8.07×10^{-6}	3.24×10^{-6}	1.12×10^{-5}	0.16	0.28

HQ - Hazard quotient

Table B-3
Percentiles - Output Parameters

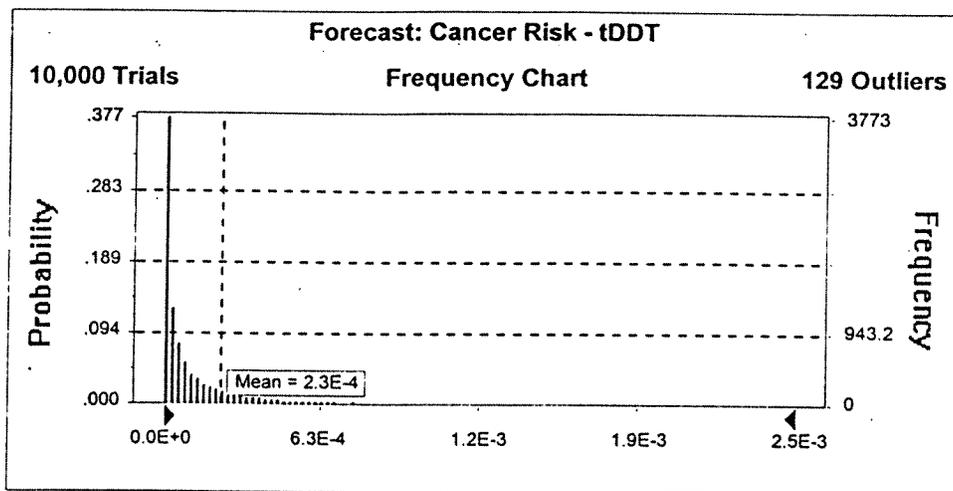
Percentiles	Cancer Risk - tDDT	Cancer Risk - PCBs	Cancer Risk - Combined	HQ - tDDT	HQ - PCBs
0%	1.90×10^{-10}	1.37×10^{-10}	3.28×10^{-10}	0.0061	0.094
5%	6.74×10^{-7}	4.96×10^{-7}	1.22×10^{-6}	0.25	0.85
10%	2.41×10^{-6}	1.73×10^{-6}	4.27×10^{-6}	0.41	1.35
15%	4.79×10^{-6}	3.34×10^{-6}	8.34×10^{-6}	0.58	1.82
20%	7.76×10^{-6}	5.24×10^{-6}	1.34×10^{-5}	0.77	2.30
25%	1.15×10^{-5}	7.68×10^{-6}	1.95×10^{-5}	0.99	2.86
30%	1.59×10^{-5}	1.07×10^{-5}	2.71×10^{-5}	1.22	3.44
35%	2.15×10^{-5}	1.41×10^{-5}	3.57×10^{-5}	1.47	4.06
40%	2.83×10^{-5}	1.83×10^{-5}	4.76×10^{-5}	1.77	4.85
45%	3.76×10^{-5}	2.38×10^{-5}	6.24×10^{-5}	2.12	5.69
50%	4.86×10^{-5}	3.07×10^{-5}	8.01×10^{-5}	2.57	6.61
55%	6.23×10^{-5}	3.83×10^{-5}	1.01×10^{-4}	3.03	7.68
60%	8.03×10^{-5}	4.84×10^{-5}	1.30×10^{-4}	3.60	9.04
65%	1.04×10^{-4}	6.18×10^{-5}	1.67×10^{-4}	4.39	10.7
70%	1.37×10^{-4}	7.88×10^{-5}	2.18×10^{-4}	5.34	12.7
75%	1.81×10^{-4}	1.03×10^{-4}	2.88×10^{-4}	6.54	15.2
80%	2.42×10^{-4}	1.35×10^{-4}	3.82×10^{-4}	8.35	18.7
85%	3.42×10^{-4}	1.85×10^{-4}	5.26×10^{-4}	11.0	23.8
90%	5.10×10^{-4}	2.71×10^{-4}	7.77×10^{-4}	15.5	32.4
95%	9.38×10^{-4}	4.58×10^{-4}	1.39×10^{-3}	25.5	51.5
100%	4.06×10^{-2}	1.23×10^{-2}	5.22×10^{-2}	572	953

Table B-4
Probability Density Functions - Output Parameters

Forecast: Cancer Risk - tDDT

Summary:

Display Range is from 0.0E+0 to 2.5E-3
Entire Range is from 1.9E-10 to 4.1E-2
After 10,000 Trials, the Std. Error of the Mean is 8.1E-6



Forecast: Cancer Risk - PCBs

Summary:

Display Range is from 0.0E+0 to 1.0E-3
Entire Range is from 1.4E-10 to 1.2E-2
After 10,000 Trials, the Std. Error of the Mean is 3.2E-6

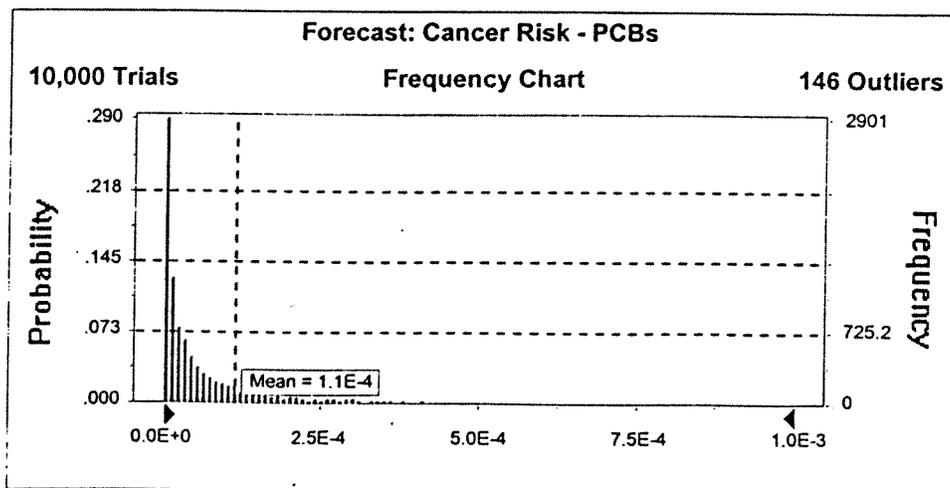


Table B-4
Probability Density Functions - Output Parameters (continued)

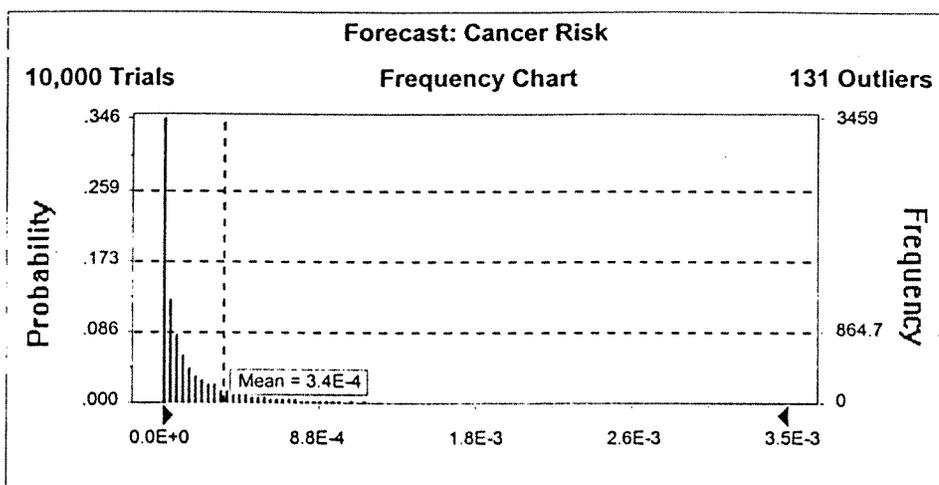
Forecast: Cancer Risk (Combined)

Summary:

Display Range is from 0.0E+0 to 3.5E-3

Entire Range is from 3.3E-10 to 5.2E-2

After 10,000 Trials, the Std. Error of the Mean is 1.1E-5



Forecast: HQ - tDDT

Summary:

Display Range is from 0.0 to 50.0

Entire Range is from 0.0 to 571.5

After 10,000 Trials, the Std. Error of the Mean is 0.2

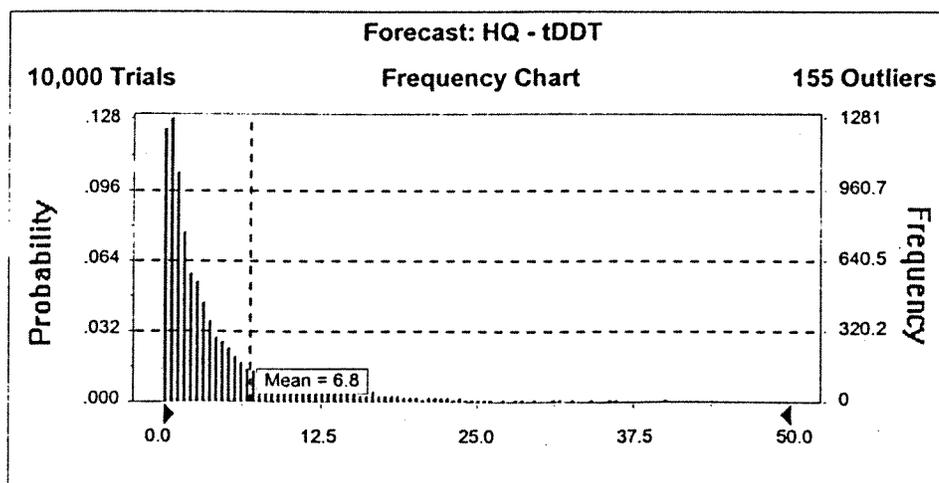


Table B-4
Probability Density Functions - Output Parameters (continued)

Forecast: HQ - PCBs

Summary:

Display Range is from 0.0 to 90.0

Entire Range is from 0.1 to 953.4

After 10,000 Trials, the Std. Error of the Mean is 0.3

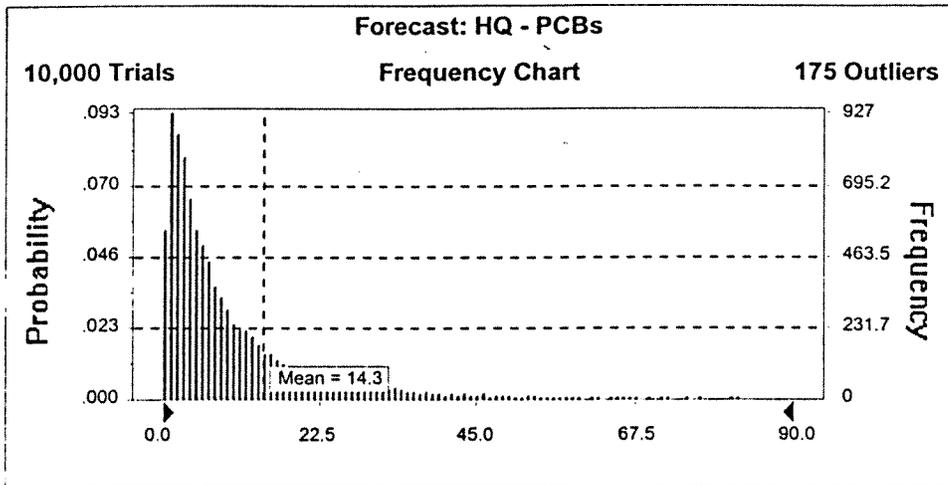
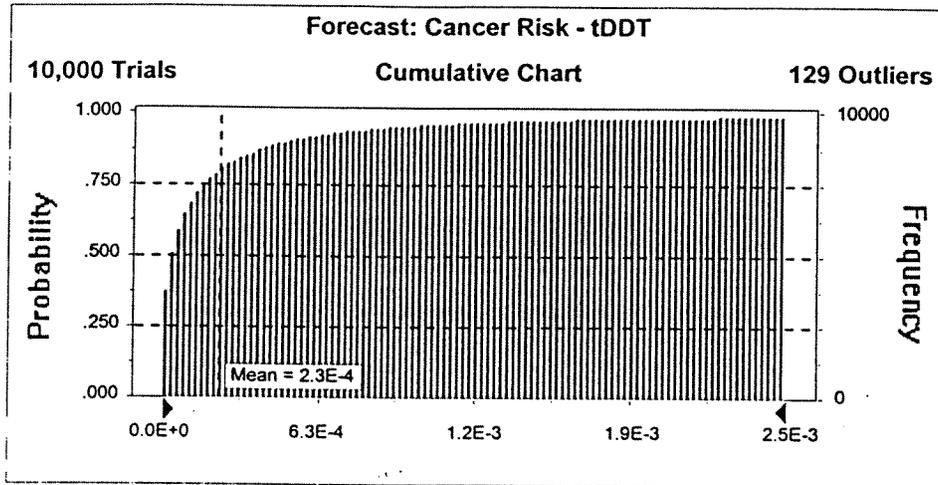


Table B-5
Cumulative Distribution Functions - Output Parameters

Forecast: Cancer Risk - tDDT



Forecast: Cancer Risk - PCBs

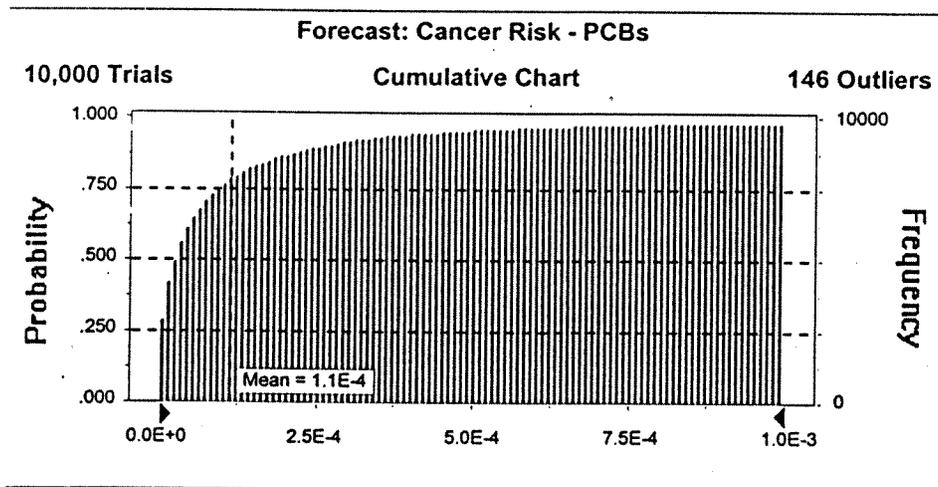
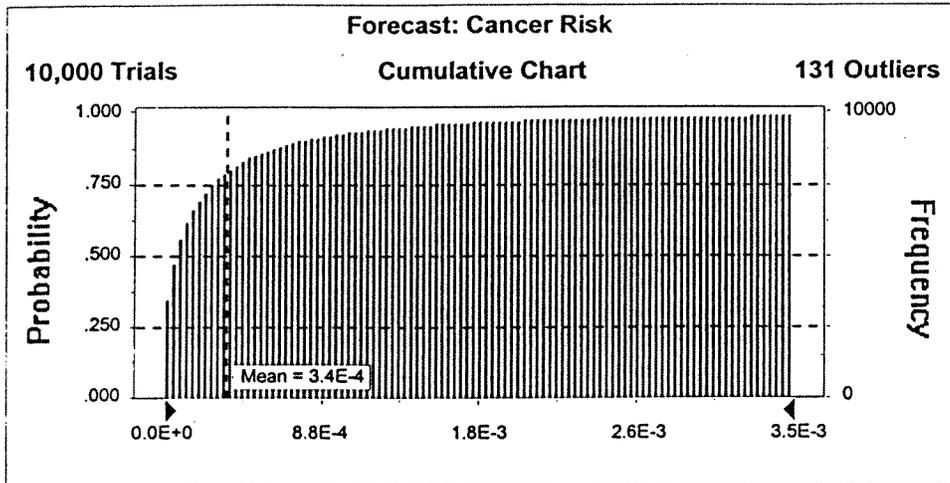


Table B-5
Cumulative Distribution Functions - Output Parameters (continued)

Forecast: Cancer Risk (Combined)



Forecast: HQ - tDDT

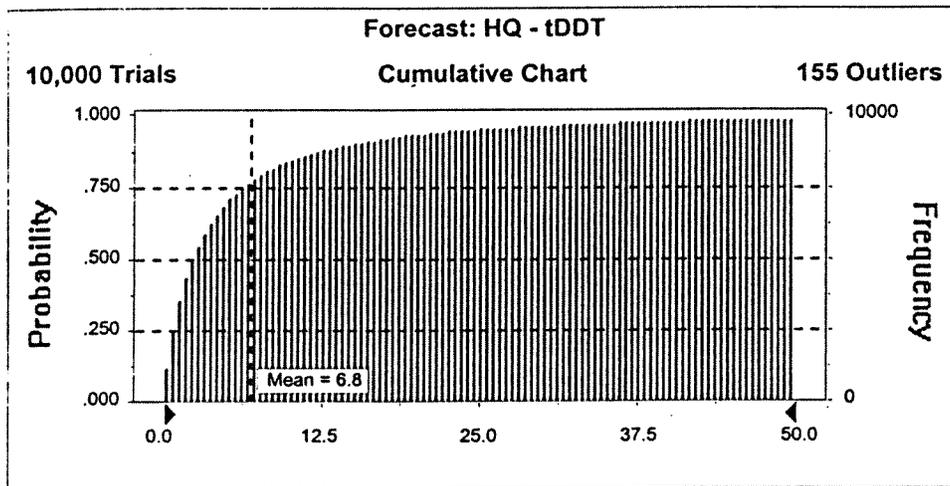


Table B-5
Cumulative Distribution Functions - Output Parameters (continued)

Forecast: HQ - PCBs

