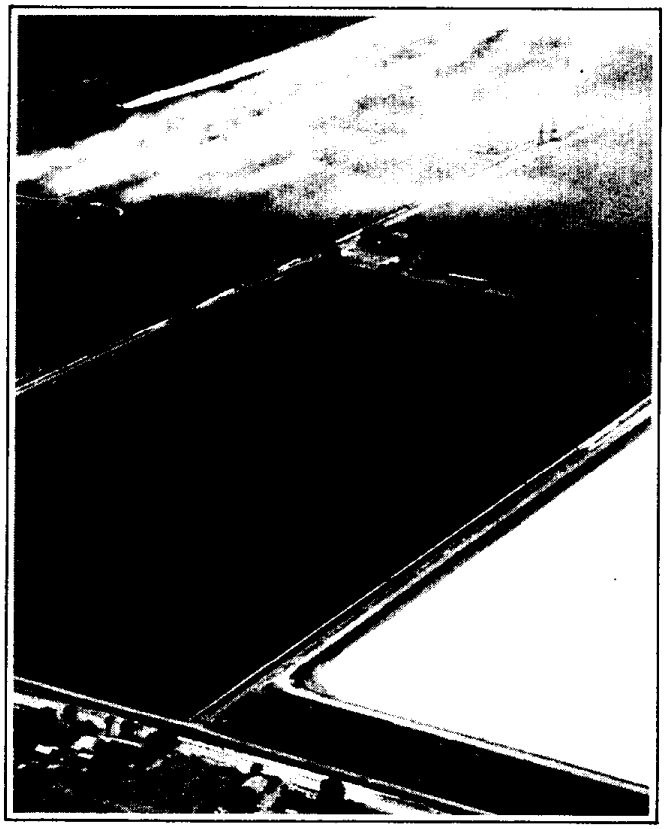
WATER QUALITY AND PESTICIDES



ENDOSULFAN

California State Water Resources Control Board

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ENDOSULFAN (THIODAN)

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PREFACE

This is one of a ten volume series of reports issued by the State Water Resources Control Board (SWRCB) on agricultural chemicals. Titles of volumes in this series: (1) Water Quality and Pesticides: A California Risk Assessment Program; (2) Toxaphene; (3) 1,2-Dichloropropane/1,3-Dichloropropene; (4) Rice Herbicides: Molinate and Thiobencarb; (5) Endosulfan; (6) Ethylene Dibromide; (7) Groundwater Contamination by Pesticides: A California Assessment; (8) Malathion; (9) 2,4-D; and (10) Glyphosate. These reports deal with priority chemicals of concern to water quality and the protection of beneficial uses of water in California.

On January 26, 1982, the State Board issued a Pesticide Guidance Document based on the premise that agricultural production and water quality protection can be compatible goals. A promising approach toward achieving these goals involves Integrated Pest Management (IPM) practices which encourage control of pests by natural predators, agricultural practice modifications and, where possible, reduction of use or substitution with less toxic pesticide. Other practices that support these goals include water and soil conservation. Agricultural resources are conserved by reducing soil erosion which, in turn, can also decrease runoff from pesticide containing soils to water.

Some current practices, e.g., simultaneous application of pesticide with irrigation water, may have an adverse impact on water quality. These activities can usually be modified to minimize adverse environmental effects. Where existing or potential water quality problems have been identified, the State Board will recommend appropriate measures to correct or prevent such adverse impacts.

ACKNOWLEDGEMENTS

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The Department of Fish and Game provided the fish kill reports, and assisted in field sampling and analysis of fish samples for endosulfan residues.

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The advice and comments of numerous individuals from state and federal agencies are acknowledged. A summary of selected comments and responses are presented in Appendix IX.

TABLE OF CONTENTS

TABLE OF CONTENTS (continued)

LIST OF APPENDICES

LIST OF FIGURES

LIST OF FIGURES (continued)

LIST OF TABLES

LIST OF TABLES (cont'd)

LIST OF TABLES (cont'd)

LIST OF ABBREVIATIONS

AGENCIES

Federal:

EPA	United States Environmental Protection Agency
FDA	United States Food and Drug Administration
FWS	United States Fish and Wildlife Service
NAS	National Academy of Sciences
NCI	National Cancer Institute
NIOSH	National Institute of Occupational Safety and Health
OSHA	Occupational Safety and Health Administration
USGS	United States Geological Survey

California:

DFA	Department of Food and Agricultrue
DFG	Department of Fish and Game
DHS	Department of Health Services
DWR	Department of Water Resources
RWQCB	Regional Water Quality Control Board
SWRCB	State Water Resources Control Board

Other:

FAO	Food Agricultural Organization, United Nations
NRCC	National Research Council of Canada

UNITS

A	Acres
C	Degrees celsius
cc	Cubic centimeter (milliliter)
g	Gram
Kg	Kilogram
l	Liter
lb	Pound
M	Molar
m	Meter
mg	Milligram (10^{-3} gram)
ng	Nanogram (10^{-9} gram)
pH	Measure of acidity (negative logarithm of hydrogen ion activity)
ppb	Part per billion (10^{-9} gram/gram or 10^{-6}gram/liter)
ppm	Part per million (10^{-6} gram/gram or 10^{-3}gram/liter)
ppt	Part per trillion (10^{-12} gram/gram or 10^{-9} gram/liter)
$t_{½}$	Half-life
torr	Unit of atmospheric pressure equivalent to millimeters of mercury
ug	Microgram (10^{-6} gram)
yr	Year

LIST OF ABBREVIATIONS (cont'd)

MISCELLANEOUS

ACGIH	American Conference of Governmental and Industrial Hygienists
ADI	Acceptable daily intake
BMPs	Best management practices
CFR	Code of Federal Regulation
EC50	Effective concentration of a toxicant that severely affects normal function of 50 percent of a test population within a specified time
FIFRA	Federal Insecticide, Fungicide and Rodenticide Act
FR	Federal Register
GLC	Gas-liquid chromatography
IPM	Integrated pest management
LC50	Lethal concentration of a toxicant that kills 50 percent of a test population within a specified time
LD50	Lethal dose of a toxicant that kills 50 percent of a test population within a specified time
MCL	Maximum contaminant level
NA	Not available
ND	Not detected
NOAEL	No observable adverse effect level
NPDES	National Pollutant Discharge Elimination System
RCRA	Resource Conservation and Recovery Act
UC	University of California
UV	Ultra violet

SUMMARY

BACKGROUND

Endosulfan is a broad-spectrum insecticide used to control insect pests that infest a large number of crops. It is registered for use on over 60 crops to control a variety of insect pests such as worms, borers, bugs, leafhoppers and aphids. It was developed and introduced in Germany by Farbwerke Hoechst, AG, in 1954 under the registered trade name 'Thiodan'. Endosulfan is extremely toxic to fish and other aquatic organisms. Its use in California has resulted in the largest number of pesticide-related fish kills reported by the Department of Fish and Game (DFG) during the past two decades. For this reason, DFG staff concurred with State Board selection of endosulfan as a priority chemical. The Pesticide Incidence Monitoring System of the U.S. Environmental Protection Agency (EPA) found over 90 reports of endosulfan-related human poisoning and ecological effects (e.g., fish and wildlife poisonings) between 1966 and 1979 (U. S. EPA, 1982). Recent information indicates that certain pests such as the potato beetle have developed resistance to endosulfan (FAO, 1981). This accentuates the need to accelerate the search for more efficaceous and ecologically safer alternative control measures.

EPA has designated endosulfan a "priority pollutant". The agency is currently in the process of re-evaluating the registration of this pesticide since much of the information on its toxicology and environmental fate is "invalid and not useful for registration" (EPA, 1982). Some of the animal toxicity data used to derive human health impacts were provided by the Industrial Biotest Laboratories (IBT). Several discrepancies in the IBT studies were discovered, and replacement studies were required, some of which are still pending.

The province of Ontario, Canada suspended endosulfan use on tobacco in 1977 because of high residues in cured tobacco leaves (Frank et al., 1979a). This was followed in 1977, by the rescinding of endosulfan use on tobacco by Agriculture Canada (Frank, 1981). In 1982, Canada decided to prohibit any new uses of endosulfan on all crops until the registrants provide new animal toxicity data to replace the invalid IBT data (Whelan, 1982). EPA has also asked the registrants of endosulfan to provide substitute studies, but has not prohibited any new or existing uses of endosulfan (EPA, 1982).

Technical grade endosulfan is a mixture of two cyclodiene stereoisomers (endosulfan I and II). Its water solubility (0.06 to 0.26 mg/l) is higher than many other common chlorinated hydrocarbon insecticides such as DDT. Endosulfan vapor pressure ($1x10^{-5}$ mm Hg) accounts for its volatilization and atmospheric transport.

Production of endosulfan in the U.S. was three million pounds in 1974. Production figures for more recent years are not available; appreciable quantities are imported from West Germany (Hoechst, 1984). According to EPA (EPA, 1983), the manufacturers of technical endosulfan in this country are Hooker Chemical Corporation, Velsicol Chemical Corporation, and Food Machinery and Chemical (FMC) Corporation. All three manufacturers of this chemical are located outside California.

In 1973, California Department of Food and Agriculture (DFA) placed endosulfan in the restricted use category. In 1982, over 535,000 pounds of endosulfan were reportedly sold in California, with over 350,000 pounds reportedly used, mostly on artichokes, lettuce, tomatoes, grapes, and alfalfa. Endosulfan ranked 11th among the major insecticides and 42nd among all pesticides used in the State during that year.

The environmental fate of endosulfan cannot be comprehensively assessed because of lack of information on hydrolysis, volatility, photodegradation, leaching, adsorption/desorption, aquatic and field dissipation, aerobic/anaerobic soil, and aquatic metabolism. EPA has asked endosulfan registrants to conduct additional studies to fill these data gaps. Past work on environmental fate dealt mainly with endosulfan I, and failed to account for endosulfan II and the metabolite, endosulfan sulfate. Both are more persistent than endosulfan I.

Based on available information, the main routes of endosulfan dispersion in the environment are volatilization and oxidation to endosulfan sulfate. Endosulfan and its metabolites persist in soil with a half-life ($t_{\frac{1}{2}}$) ranging from a few months (endosulfan I) to over two years (endosulfan II and sulfate). Single applications (0.25-15 lb/acre) of endosulfan during a growing season year after year can result in the accumulation of this pesticide and its degradation products in the soil.

The reported hydrolysis half-life of endosulfan in water ranges from one to six months. Bottom sediments may be a sink for accumulation of this pesticide in aquatic ecosystems. Runoff of endosulfan-laden soil can potentially contaminate surface waters.

Drinking water criteria expressed as maximum contaminant level or MCL for endosulfan and its metabolites have not been established. The EPA 1980 Clean Water Act criterion for ambient water is 74 ug/l (ppb) to protect human health from consumption of fish, shellfish and water. The instantaneous maximum criteria for freshwater and saltwater aquatic life are much lower (0.22 ug/l and 0.034 ug/l, respectively). The 24-hour average criteria for protection of freshwater and saltwater aquatic life are extremely low (0.056 ug/l and 0.008 ug/l, respectively).

The National Academy of Sciences' recommended guideline for protection of predators from endosulfan in fish is 0.1 mg/kg. EPA has established tolerances for endosulfan residues in raw agriculture commodities ranging from 0.1 to 2.0 mg/kg (ppm) but neither the Food and Drug Administration nor EPA have set an action level or tolerance for endosulfan in fish and shellfish for human consumption. In the absence of these guidelines, it is difficult to interpret endosulfan residue data in fish and shellfish.

MONITORING

The U.S. Fish and Wildlife Services' National Pesticide Monitoring Program does not monitor for endosulfan in fish. The California Department of Fish and Game (DFG) has historically monitored for endosulfan only when it is a suspected cause of fish kills. However, since 1982 the Department has monitored for endosulfan in Imperial Valley as part of the Hydrilla Eradication Study. Routine monitoring for endosulfan in California is conducted only by the State Water Resources Control Board (SWRCB). Endosulfan residues have been detected in water, fish, vegetable, and sediment samples from different sites in California.

In 1982, the State Board's Toxic Substances Monitoring Program (field sampling and chemical analysis conducted by DFG) found up to 25 ug/kg (ppb), wet weight, of endosulfan I residues in fillets of fish from three California rivers. Channel catfish caught from New River in 1979 had 160 ug/kg endosulfan I, which is greater than the NAS-recommended guideline to protect predators of 100 ug/kg. A water sample collected from Salinas River in 1981 had 5.8 ng/l (ppt) endosulfan I. The SWRCB Mussel Watch Program (field sampling and chemical analysis conducted by DFG) detected as high as 890 ug/kg of endosulfan I in whole bodies of mussels collected from Elkhorn Slough during the 1981-82 sampling season and up to 1,200 ug/kg in 1982-83. Stickleback collected from Old Salinas River Slough during 1982-83 had 1,200 ug/kg endosulfan I, which is twelve times the NAS guideline. Even higher values were found in 1983-84. Endosulfan II and sulfate were not analyzed in these mussel and fish samples in 1982-83 but were looked for in 1983-84. Sediment samples from Monterey County collected by the SWRCB Toxics Special Project in 1982 contained as high as 151 ug/kg of total endosulfan (endosulfan I, II and sulfate). Fish samples from this region had up to 52 ug/kg of total endosulfan residues.

The California Department of Food and Agriculture also detected endosulfan residues as high as 5 mg/kg in some leafy vegetables in 1976 (Coleman and Dolinger, 1978).

RISK ASSESSMENT

The most significant impact of endosulfan on beneficial uses of water is its extreme toxicity to fish and other aquatic organisms. The lowest reported acute toxicity (LC50) values are 0.17 ug/1(ppb) for a freshwater organism (rainbow trout) and 0.04 ug/1 for a saltwater organism (pink shrimp). Endosulfan sulfate is as toxic to aquatic life as its parent compounds. Bioaccumulation and adverse chronic effects have been observed in aquatic organisms including fish at low residue levels. It has been reported that chemicals used as "inert" emulsifiers may increase endosulfan toxicity to fish.

Endosulfan's acute toxicity to rodents, dogs and other mammals is also high, similar to parathion. The oral toxicity of endosulfan sulfate to mammals (LD50 for mice: 8 mg/kg) is higher than the parent compounds.

Earlier studies have indicated that endosulfan is not carcinogenic, mutagenic or teratogenic. However, these studies do not meet EPA's current requirements for risk assessment of cancer and other chronic effects. In 1982, EPA required the registrants to conduct additional studies to fill these data gaps. The long-term studies for carcinogenicity will not be completed before 1986.

RISK MANAGEMENT

Endosulfan nonpoint source losses to the atmosphere can occur by drift and volatilization. Drift losses can be reduced by substituting ground application for aerial application whenever possible. Volatilization losses can only be reduced, however, by reducing the amount applied. Aerial application of endosulfan in California in 1981 accounted for 66 percent of the total endosulfan used in the State. However, in some counties such as Imperial County, as high as 93 percent of the insecticide was applied aerially.

Section 208 of the Federal Water Pollution Control Act (now called "Clean Water Act") requires states to identify nonpoint sources of pollution, including runoff from agricultural fields, and to develop plans for their control. Agricultural Best Management Practices (BMPs) relevant to control of endosulfan discharges include soil and water conservation to minimize tailwater runoff and soil loss. Discharges of endosulfan adsorbed in sediment could be reduced by installing sediment traps below treated watershed areas. Runoff water from treated fields should be kept to a minimum.

Integrated Pest Management (IPM) programs are available from the University of California for tomatoes, alfalfa, and grapes which include nonchemical or safer chemical alternatives. The major crop pest for which an IPM program has not yet been established is the artichoke plume moth.

Endosulfan point source discharges can occur from manufacturers, formulators, and applicators. In California endosulfan is formulated at J.R. Simplot (formerly Occidental Chemical) in Lathrop and FMC Corporation in Fresno. Twenty one chemical companies have 87 different endosulfan products registered for use in California (DFA, 1984). As many as 400 pesticide applicator sites have been reported in the Central Valley. Endosulfan residues (up to 2,300 ug/l) have been detected in rinsewater discharges from some Imperial County applicator sites.

RECOMMENDATIONS

Extensive use of endosulfan in California has caused numerous problems including fish kills and bioaccumulation in aquatic organisms. Every effort should therefore be made to mitigate further contamination of the environment by this pesticide and its metabolites. To accomplish this objective, the following actions are recommended to the appropriate state or federal agencies:

Department of Food and Agriculture (DFA)

1. Reevaluate registration and use conditions for endosulfan in accordance with California Administrative Code, Title 3, Chapter 4, Subchapter 1, Group 2, Section 2367. (DFA Pesticide Registration and Evaluation Committee accepted this recommendation at the January 20, 1984, meeting and reevaluation is currently underway).

2. Improve label language as per recommendations 3(b) below.

3. Notify county agricultural commissioners that:

   (a) Endosulfan use permits should not be issued where safer substitutes (as described in IPM manuals of the University of California Cooperative Extension Service) are available.

   (b) Severely restrict endosulfan use by time, place and manner of use near water bodies where adverse impacts have been documented as well as where the potential for new adverse impacts on aquatic life is high, such as agricultural sites with high runoff potential. The best management practices include:

      (i) Field irrigation prior to endosulfan application. Endosulfan should never be applied to a field where irrigation is occurring and tailwater runoff to a receiving water is likely. In case of post-application irrigation, a waiting period of five days should be observed between the time of endosulfan application and irrigation. These recommendations are intended to prevent the discharges of endosulfan-containing irrigation return flows to fish-bearing waters.

      (ii) Ground application of endosulfan should be preferred over aerial application wherever possible. Where aerial application is unavoidable, wind velocity should not exceed 5 mph at the time of treatment, and a 100 ft. buffer strip should be observed adjacent to any fish-bearing waters.

(c) A high priority should be placed on reporting all fish and wildlife kills where pesticide use is known or suspected to be the cause.

4. Require the registrants of endosulfan to submit toxicity data for both the formulated pesticide products and the active ingredient since chemicals used as emulsifiers have been found to increase endosulfan toxicity to fish.

5. Require the manufacturer (Hoechst Chemical Co.) to conduct site-specific environmental monitoring in California, and acute and chronic toxicity studies with resident aquatic species including body-burden impacts. (Hoechst Co. letter of April 30, 1984, to SWRCB.)

U.S. Environmental Protection Agency (EPA)

1. Accelerate reevaluation of endosulfan registration.

2. Establish a drinking water maximum contaminant level (MCL) for total endosulfan (endosulfan I, II, and sulfate).

3. Establish a food additive tolerance for total endosulfan in fish and shellfish (for adoption by U.S. Food and Drug Administration).

4. Resolve the current discrepancy between the existing endosulfan food tolerances which yield a theoretical maximum residue contribution (TMRC) in diet which greatly exceeds the current ADI of 0.28 mg/day.

5. Include endosulfan analysis for isomers I, II, and sulfate in all EPA-sponsored research and monitoring programs.

U.S. Food and Drug Administration (FDA)

Adopt an action level for total endosulfan in fish and shellfish.

California State Water Resources Control Board (SWRCB)

1. Adopt, as an interim guideline, water quality criteria for total endosulfan residues as follows: (a) fresh water-0.22 ug/l (instantaneous maximum), and (b) salt water-0.03 ug/l (instantaneous maximum). When more specific toxicity information becomes available for California resident aquatic species, these guidelines should be revised.

2. Expand State Board Toxic Substances Monitoring and Mussel Watch analyses to include endosulfan II and endosulfan sulfate whenever endosulfan I residues are analyzed.

California Regional Water Quality Control Boards (RWQCB)

Identify all potential point sources of endosulfan discharge and develop appropriate control strategies to minimize discharges including self-monitoring reports, compliance inspections and discharge prohibitions.

California Department of Fish and Game (DFG)

1. Analyze for all endosulfan compounds (I, II, and sulfate) in fish from fish-kill incidents.

2. Reduce fish-kill incidences by increasing preventive surveillance during: (a) periods of highest use; (b) most sensitive life stages; and (c) in areas with highest potential for adverse impacts.

3. Validate the national ambient water quality criteria with resident species.

California Department of Health Services (DHS)

1. Establish an action level of total endosulfan in drinking water.

2. Include total endosulfan analysis in the monitoring programs conducted by DHS or required of local water purveyors.

University of California Cooperative Extension

1. Accelerate research on alternative control measures (including non-chemical methods of pest management) for those pest problems where endosulfan substitutes are not available.

2. Issue a Pesticide Management Manual - Endosulfan Notice to farm advisors, farmers, agricultural commissioners, pesticide advisors and applicators, and irrigation and soil conservation districts. This notice should contain the most current information on the toxicology, environmental fate, IPM substitutes, and recommended soil and water conservation's "Best Management Practices" to minimize impacts on human health and environment.

U.S. Fish and Wildlife Service

1. Include analyses of total endosulfan in the National Pesticide Monitoring Program.

2. Accelerate laboratory studies on chronic effects of total endosulfan such as growth, survival and reproduction of aquatic organisms.

3. Investigate the toxicity to aquatic life of endosulfan and its metabolites *per se* and the combined effects of endosulfan with other ubiquitous contaminants in aquatic ecosystems (e.g., petroleum hydrocarbons, PCB and DDT).

National Academy of Sciences (NAS)

Substantiate the scientific validity of the NAS guideline of 100 ug/kg of endosulfan and other chlorinated hydrocarbon pesticides in fish for the protection of predators.

I. INTRODUCTION

Endosulfan is a broad-spectrum chlorinated hydrocarbon insecticide which acts as a central nervous system poison. It was developed in Germany by Farbwerke Hoechst AG and introduced by the firm in 1954 under the registered trade name of 'Thiodan' (Maier-Bode, 1968). Technical grade endosulfan is a 7:3 mixture of two sterioisomers, endosulfan I and II, or α- and β-endosulfan (Figure I-1), which have similar chemical and toxicological properties.

Endosulfan is implicated in more known pesticide-related fish kills in California than any other pesticide (50 fish kill episodes during the last 21 years resulting in the loss of over 150,000 fish). In 1983, two endosulfan-related fish kills were reported to have caused the loss of approximately 1,000 carp, channel catfish, largemouth bass, and shad (Finalyson, 1983a and b). Canadian authorities observed several endosulfan-related fish kills during the period when total sales of endosulfan in that country doubled (NRCC, 1975).

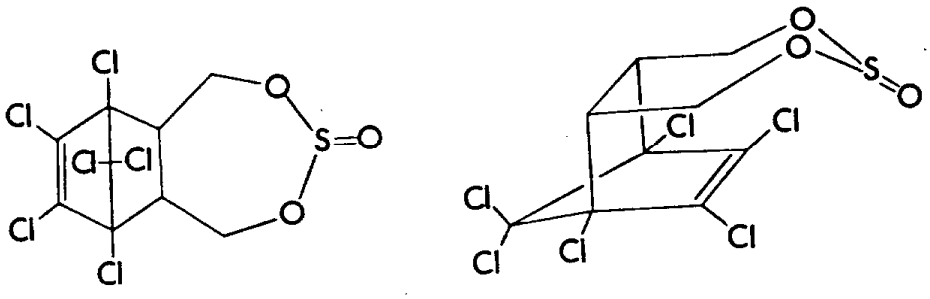
Endosulfan (I and II) and endosulfan sulfate (a degradation and metabolic product) are extremely toxic to fish and other aquatic organisms. For this reason, the ambient water quality criteria established by EPA for endosulfan are very low. The criteria to protect freshwater life against long-term chronic and short-term acute effects are 0.056 ug/l (ppb) (24-hour average) and 0.22 ug/l (instantaneous maximum), respectively. The corresponding saltwater criteria are 0.0087 ug/l and 0.034 ug/l, respectively (EPA, 1980). According to the Criteria Branch of EPA (Gostomski, 1984) these criteria are for total endosulfan (I, II and sulfate). The International Joint Commission of the United States and Canada has established a water quality objective of 0.003 ug/l for endosulfan in the Great Lakes Region (IJC, 1977).

Endosulfan is on the EPA's "Priority Pollutant" list. In 1982, EPA initiated a "Pesticide Registration Standard" review of endosulfan. The agency identified serious data gaps of information currently required for registration, such as hydrolysis, photodegradation, and aquatic field dissipation. EPA has asked the registrants of this insecticide to provide additional information within a specified time schedule in order to fill the existing data gaps (Appendix I).

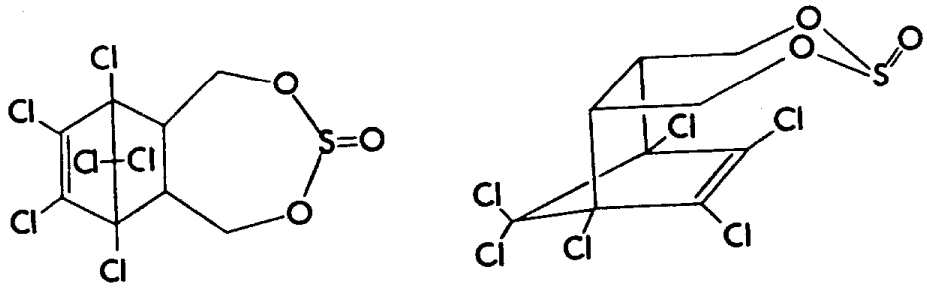
EPA's "Pesticide Incidence Monitoring System" found 91 reports of human poisoning and ecological incidents such as fish kill incidents related to the use of endosulfan during 1966 to 1979 (EPA, 1982).

Figure I-1

MOLECULAR STRUCTURES OF ENDOSULFAN I AND II



Endosulfan I



Endosulfan II

Some of the endosulfan animal toxicity data used to derive human health impacts were provided by the Industrial Biotest Laboratories (IBT). Several discrepancies in these studies were recently discovered. Information central to the assessment of human safety was found to be invalid by Agriculture Canada (NRCC, 1983). In 1982, Canada decided to prohibit any new uses of endosulfan on crops until the registrant provided new animal toxicity data to replace the invalid IBT data (Whelan, 1982).

In 1973, the California Department of Food and Agriculture (DFA) restricted most uses of endosulfan. Despite this restriction, endosulfan use remains high. Over 350,000 pounds were reportedly used in 1982 mostly on artichokes, celery, lettuce, tomatoes, and alfalfa (DFA, 1983).

Endosulfan is a fairly persistent pesticide. Endosulfan II and sulfate are more persistent than endosulfan I; the half-life of each in a sandy loam soil has been reported to be over two years (Stewart and Cairns, 1974). Since it is a persistent pesticide, its residues in water, sediment and fish have been frequently detected. Total endosulfan concentrations may be underestimated because most monitoring studies have not analyzed for endosulfan II and endosulfan sulfate. These two compounds are as toxic as endosulfan I.

In view of all this information, endosulfan was selected as a "priority" chemical for an in-depth study by the Toxics Program staff. Department of Fish and Game also requested the State Board to consider endosulfan as a priority chemical. This report is organized into chapters dealing with endosulfan monitoring (Chapter II), risk assessment (Chapter III), and risk management (Chapter IV). Supplementary information on physical and chemical properties, use trends, environmental fate, analytical methodology, and criteria and standards, is briefly reviewed in the Appendices II through VI. Appendices VII through IX contain information on the endosulfan use label; University of California Agricultural Extension Service: recommendations to reduce fish kills from thiodan; and selected review comments and responses.

II. MONITORING STUDIES

OVERVIEW

Endosulfan residues have been detected in air, rain, surface and ground water, sediment, fish, and other aquatic organisms. Unfortunately, most of the monitoring studies have analyzed for endosulfan I only; the equally toxic and persistent endosulfan II and endosulfan sulfate were not considered. Therefore, total endosulfan residues in the environment are underestimated. Endosulfan analytical methodology is briefly discussed in Appendix V.

EPA included endosulfan as one of the 129 priority pollutants because of its frequent detection in surface water samples. Shackleford and Keith (1976) reported that during the first six months of 1976, endosulfan isomers were found in nine water samples (four drinking water and five river) in the U.S. and elsewere. As no federal or state drinking water standard or advisory for endosulfan has yet been established, it is not generally looked for in a monitoring program.

The "National Pesticide Monitoring Program" of U.S. Fish and Wildlife Services does not monitor for endosulfan in fish samples. This data gap appears to be a serious omission since endosulfan is one of the most toxic pesticides to aquatic life; the lowest reported LC50 values for rainbow trout and striped bass are 0.17 and 0.1 ug/l (ppb), respectivly (EPA, 1980).

Most of the monitoring studies reported in the literature were conducted "reactively" after an accidental spill or disposal had contaminated the environment and impacted non-target organisms. In 1969, a massive fish kill in the Rhine River, Germany, was reported to be associated with contamination of a 75-mile stretch of the river with up to 5.5 ppb endosulfan. The discharge was due to an accidental spill, as well as effluent from an endosulfan manufacturer, Farbwerke Hoechst AG (Coleman and Dolinger, 1978). Very few other "investigative" monitoring data have been reported. One study by Rosales et al. (1979) found up to 0.4 ppb endosulfan II in oysters from Gulf of Mexico coastal lagoons.

NATIONAL PERSPECTIVE

Outside of California, the most intensively studied area for environmental impacts of pesticides and other chemicals has been the Great Lakes ecosystem. Eisenrich et al. (1981) found up to 10 and 12 ng/l (ppt) of endosulfan I and II, respectively, in precipitation over this region. The mean concentration of these two isomers in air was 1 ug/m . According to this study, nearly 55 tons per year of endosulfan I and II are deposited in Lakes

Superior, Michigan, Huron, Erie, and Ontario. Sonzogni et al. (1980) detected endosulfan in the drainage waters of the Great Lakes basin. Frank et al. (1979b) found up to 7.3 ppb endosulfan in the sediment of Georgian Bay.

Urban soils in Baltimore, Maryland, and Macon, Georgia were found to contain 170 and 60 ppb endosulfan sulfate, respectively (Carey et al., 1979).

Little information on endosulfan presence in the U. S. is available outside of these areas and California.

CALIFORNIA MONITORING RESULTS

The State Water Resources Control Board (SWRCB) has conducted both routine monitoring and special investigations for pesticides. A number of programs, such as the Priority Chemical Program, the fresh water Toxic Substances Monitoring Program, and marine Mussel Watch have monitored specifically for endosulfan. A number of other state and local agencies including Regional Water Quality Control Boards (RWQCB); the Departments of Fish and Game (DFG), Water Resources (DWR), Food and Agriculture (DFA); and local irrigation and water districts have periodically monitored for endosulfan residues in the environment.

SWRCB PRIORITY CHEMICAL PROGRAM

Endosulfan was selected as a 'priority chemical' because of its extreme toxicity to fish and because it has been associated with fifty fish kills in California during the last two decades. Samples were collected from Monterey and Imperial Counties, the two top endosulfan use counties in the State. Samples consisted of sediment (three to five cores per site) and fish (two to ten live fish of each species). The samples were composited and analyzed by Radian Corporation, Sacramento, using standard analytical methods (Appendix V).

All species collected from lower Elkhorn Slough, Monterey County, had residues of endosulfan I, II and endosulfan sulfate (up to 52 ug/kg) in the liver (Table II-1). Concentrations of endosulfan II and endosulfan sulfate were far greater than endosulfan I. This suggests an aged residue, where endosulfan I has been metabolized. If only endosulfan I and the fish fillet had been analyzed, the total residue would have been underestimated or undetected, and the potential for toxicological impacts, such as fish liver damage, would have been overlooked. The fillet of these fish did not have detectable levels of endosulfan. Liver is the organ most impacted by endosulfan (liver degeneration); endosulfan accumulates in the liver and is detoxified there. Persons consuming these fish are unlikely to be harmed since fish are cleaned and their livers discarded prior to cooking.

Table II-1

ENDOSULFAN IN FISH LIVER SAMPLES FROM ELKHORN SLOUGH, MONTEREY COUNTY[1/2/]
(SWRCB Toxics Special Project)

Species	Concentration (ug/kg, fresh wt)			
	Endosulfan I	Endosulfan II	Endosulfan sulfate	Total
Black surfperch	Trace [3/]	7.2	23	30.2
Shiner surfperch	"	8.6	18	26.6
Starry flounder	"	7.8	15	22.8
Pacific sanddab	9.4	7.0	4.1	20.5
Speckled sanddab	Trace	13	18	21.0
Staghorn sculpins	"	19	20	39.0
Fringehead	"	20	32	52.0

1/ Fish samples collected on February 1, 1983

2/ Fillets of these fish samples was also analyzed; no residues were detected

3/ The designation "Trace" indicates that some endosulfan was found at the detection limit (3 ug/kg), but the concentration was too low for quantification

The presence of endosulfan in fish indicates that these organisms were exposed to residues in water and/or sediment. Sediment samples taken from Salinas, Monterey County, had concentrations ranging from traces (< 0.2 ug/kg) to over 150 ug/kg total endosulfan (Table II-2). Endosulfan II and sulfate were higher than endosulfan I. Surface sediment (0-3 inches) at Old Salinas Channel had more residues than the subsurface (3-6 inches). However, the Salinas River sediment taken at Gonzales Bridge had only traces of endosulfan sulfate, whereas the subsurface sample contained 9.4 ppb of endosulfan I and II. Endosulfan-laden sediments have an adverse toxicological impact on benthic organisms and filter feeders. According to McLeese and Metcalfe (1980), who studied the toxicity of pesticide laden sediments to shrimp (Crangon septemspinosa), the 96-hour LC50 value for endosulfan was very low (6.9 ug/kg or ppb).

Endosulfan desorption from sediments serves as a source of chronic exposure to fish and other organisms. Sediments adsorb endosulfan, and are therefore a better indicator of endosulfan presence than a water sample. Chapman et al. (1982) stated that since endosulfan and endosulfan sulfate are readily absorbed by sediments, priority for environmental monitoring should be given to sediments. After a fish kill, Frank (1972) reported that, while endosulfan residues in the pond water were below the detection limit, over 3 ug/kg (dry wt.) of total endosulfan were present in the sediments. The pesticide dissipated from the water in this case by the time of sampling.

Four out of 15 (26.6 percent) composite fish samples collected from the Fillaree Canal, Imperial County, had detectable levels of endosulfan I, II and sulfate (Table II-3). Concentrations of the more persistent endosulfan II and endosulfan sulfate were higher than endosulfan I. Total endosulfan residues found in carp sampled in March, 1983 were higher (28 ppb) than those sampled in July, 1982 (20.1 ppb) (Figure II-1). In January, 1982, approximately 50 largemouth bass, channel catfish, and carp were found dead in the same canal. The canal water, as well as the tailwater from a nearby lettuce field, had 3.5 to 26 ug/l (ppb) of endosulfan. Gill tissue of dead carp contained 800 ppb of endosulfan I and II (Table II-12). Nudrin (Lannate) was also applied with endosulfan to lettuce but it was not detected in either water or dead fish.

All the sediment samples collected from Imperial County had traces of endosulfan (Table II-4). Three of these samples had measurable levels of endosulfan II ranging only from 0.21 to 1.8 ppb. In this instance, the persistent metabolite endosulfan sulfate was below the limit of quantification. This finding, however, is not unique. Frank et al. (1981) could not detect endosulfan sulfate while endosulfan II was present at 1 ppb in a sediment sample from St. Marys River in Canada.

Table II-2

ENDOSULFAN IN SEDIMENT SAMPLES FROM SALINAS, MONTEREY COUNTY[1/]
(SWRCB Toxics Special Project)

Site	Depth (inches)	Concentration (ug/kg, dry wt)			
		Endosulfan I	Endosulfan II	Endosulfan sulfate	Total
Pajaro River	0-3	Trace[2/]	ND[3/]	ND	-
Salinas River Twin Bridge	0-3	ND	Trace	"	-
Salinas River Gonzales Bridge	0-3	"	ND	Trace	-
Salinas River Gonzales Bridge	3-6	1.6	7.8	ND	9.4
Salinas River Chualar Bridge	0-3	ND	Trace	"	-
Old Salinas Channel	0-3	1.6	40	110	151.6
Old Salinas Channel	3-6	ND	1.2	5.5	6.7

1/ Sediment samples collected on September 29, 1982

2/ The designation "Trace" indicates that some endosulfan was found at the detection limit (0.2 ug/kg), but the concentration was too low for quantification

3/ Not detected

Table II-3

ENDOSULFAN IN FISH FILLET SAMPLES FROM FILLAREE CANAL, IMPERIAL COUNTY[1/]
(SWRCB Toxics Special Project)

Sampling date	Species	Concentration (ug/kg, fresh wt) Endosulfan I	Endosulfan II	Endosulfan Sulfate	Total
July, 1982	Largemouth Bass	ND[2/]	ND	ND	-
	Channel Catfish	"	"	7.8	7.8
	Carp	"	8.1	12.0	20.1
August, 1982	Largemouth Bass	"	ND	ND	-
	Carp	"	"	"	-
	Channel Catfish[3/]	"	"	"	-
October, 1982	"	"	"	"	-
	Carp	4.8	6.5	11.0	22.3
February, 1983	"	ND	ND	ND	-
	Channel Catfish	"	"	"	-
	Largemouth Bass	"	"	"	-
March, 1983	Carp	"	12.0	16.0	28.0

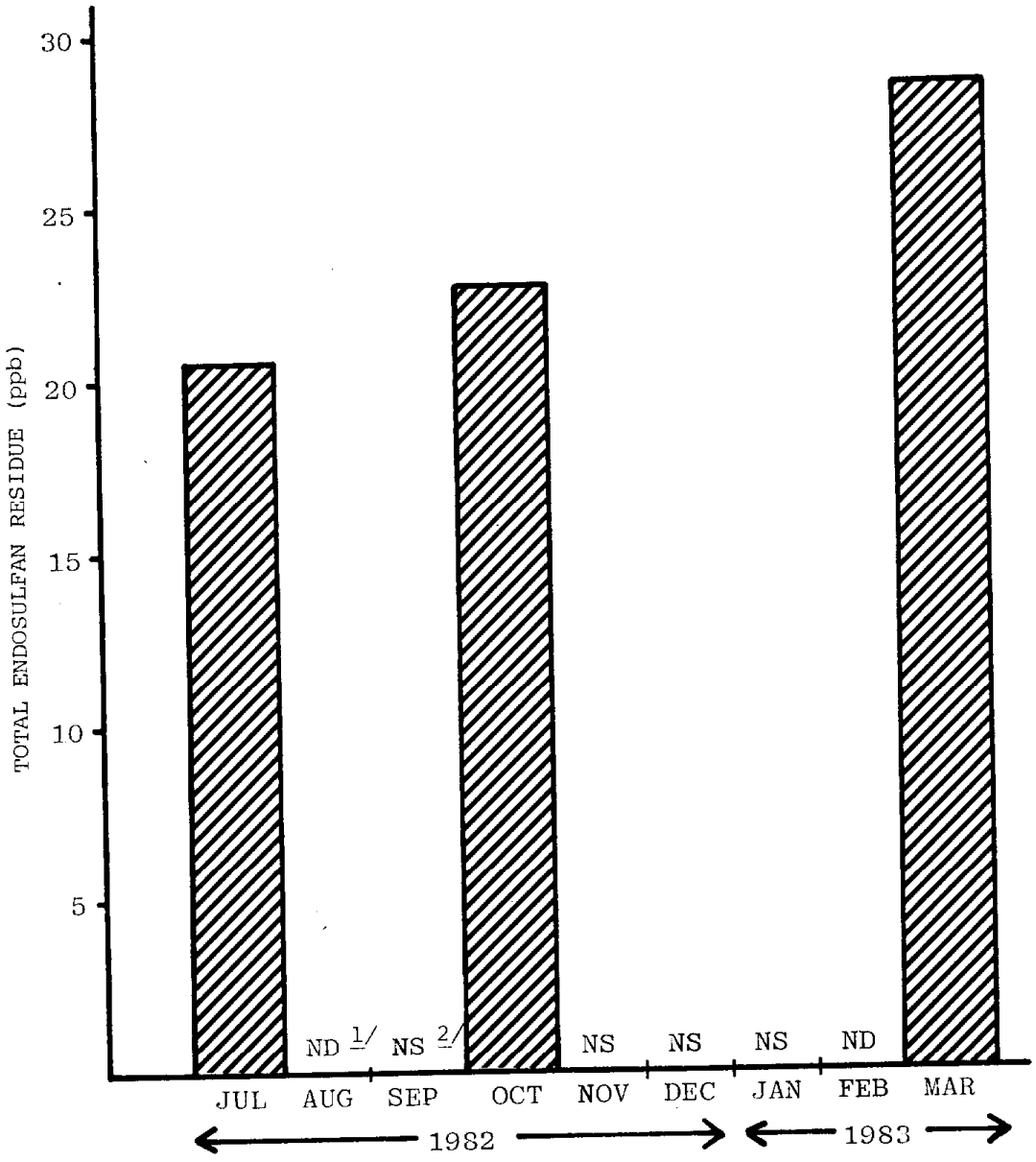
1/ Fish samples provided by Department of Fish and Game

2/ Not detected (Detection limit: 3 ug/kg)

3/ Liver was also analyzed; no residues were detected

Figure II-1

ENDOSULFAN RESIDUES IN CARP FROM
FILLAREE CANAL, IMPERIAL COUNTY
(July 1982 to March 1983)



1/ Not detected

2/ Not sampled

Table II-4

ENDOSULFAN IN SEDIMENT SAMPLES (SURFACE 3 INCHES) FROM IMPERIAL COUNTY[1/]
(SWRCB Toxics Special Project)

Site	Concentration (ug/kg, dry wt)			
	Endosulfan I	Endosulfan II	Endosulfan sulfate	Total
Lotus Canal	Trace[2/]	Trace	Trace	-
Spruce Main Drain at Outlet of New River	"	"	"	-
Vail Drain at New River	"	0.21	"	0.21
Salton Sea Outlet	"	0.54	"	0.54
Timothy Drain at Frederick and Elder	"	1.8	"	1.8
Best Drain near Outlet to New River	"	Trace	"	-

1/ Sediment samples collected on November 2, 1982

2/ The designation "Trace" indicates that some endosulfan was found at the detection limit (0.2 ug/kg), but the concentration was too low for quantification

SWRCB TOXIC SUBSTANCES MONITORING (TSM) PROGRAM

In this program live fish are collected from major California streams and analyzed for pesticides and toxic chemicals to serve as one indicator of water quality. Endosulfan residues in fish have been detected every year since the inception of this program in 1976. Endosulfan concentrations in composite fillet or whole-body samples of different fish and crayfish have ranged from 5 to 110 ppb (Table II-5). Total endosulfan concentrations are underestimated since endosulfan II and sulfate were not analyzed. The potential effects of endosulfan body burden on the physiological functions of fish have not been studied.

The National Academy of Sciences (NAS) recommended a guideline of 100 ppb endosulfan in fish (either singly or in combination with other chlorinated hydrocarbon pesticides) to protect predators from the effects of consuming contaminated fish (EPA, 1973). This guideline was exceeded in 1979 when 110 ppb endosulfan I were detected in New River channel catfish fillets. Since endosulfan II and sulfate were not included in the analysis, the reported endosulfan body burden is an underestimate.

The Food and Drug Administration (FDA) has yet to establish an action level and EPA a tolerance for endosulfan in fish and shellfish for human health protection.

SWRCB MUSSEL WATCH

The California State Mussel Watch, a marine water quality monitoring program, was initiated by SWRCB in 1977. It uses a bivalve mollusk, the mussel, as an indicator species. Endosulfan I residue data in resident mussels for the years 1979 through 1982 are given in Table II-6. Sandholt Bridge is the only station in the Moss Landing drainage area (Monterey County) which was sampled for four consecutive years. Concentrations of endosulfan I in mussels (*Mytilus edulis*) at this site increased from 170 ug/kg in 1980-81 to 3,800 ug/kg in 1983-84 (Figure II-2). Total endosulfan was measured for the first time in 1983-84, and was more than double (7,200 ug/kg) the endosulfan I concentration.

At certain locations, a resident mussel population is absent or unsuitable for study. Clean mussels are taken from their natural habitat at Bodega Bay and transplanted to such locations (Figure II-3). Endosulfan concentrations in mussels from some transplant stations are given in Table II-7. Mussels at a transplant station in Elkhorn Slough had up to 140 ppb endosulfan I. Since the more persistent endosulfan II and sulfate were not analyzed, total endosulfan residues are underestimated.

Table II-5

ENDOSULFAN I IN FISH FROM CALIFORNIA RIVERS
(SWRCB, Toxic Substances Monitoring Program)

Year	Frequency of Detection	Sampling Station	Fish Sampled	Tissue Analyzed	Concentration (ug/kg, fresh wt)
1982	3/20 (15%)	Stanislaus River	Channel catfish	Fillet	10
		Alamo River	"	"	25
		New River	"	"	12
1981	5/44 (11%)	San Joaquin River	"	"	17, 25
			Carp	"	8
		Alamo River	"	"	5
			Channel catfish	"	23
		New River	"	"	49
			Carp	"	19
		Sutter Bypass	"	"	16
			Channel catfish	"	22
		Colusa Drain	"	"	11
1980	3/29 (10%)	Santa Ana River	Crayfish	Tail flesh	7
		New River	Channel catfish	Fillet	7-27
		Reclamation Slough	Brown bullhead	"	22
1979	2/28 (7%)	Alamo River	Channel catfish	"	11
		New River	"	"	110
1978	1/26 (4%)	San Joaquin River	Carp	"	14
1977	3/26 (11%)	Feather River	"	Whole fish	20
			White catfish	"	8
		San Joaquin River	"	"	7
			Carp	"	12
		New River	Big mouth Buffalo	"	32
1976	2/27 (7%)	Alamo River	Channel catfish	"	82
		New River	Carp	"	88

Table II-6

ENDOSULFAN I IN RESIDENT MUSSELS ALONG CALIFORNIA COASTLINE
(SWRCB Mussel Watch Program)

Species	Site	Concentration (ug/kg, dry wt) 1979-80	1980-81	1981-82
Mytilus edulis				
	Elkhorn Slough (Duck Club)	NS[1/]	NS	260
	Moss Landing (Sandholt Bridge)	NS	170	770,890
	San Francisco Bay (Redwood Creek)	1.0	NS	NS
	Port Hueneme	NS	NS	3.0
	Channel Islands Marina	NS	NS	2.2
M. californianus	Trinidad Head	ND[2/]	2.3	ND
	Pygmy Forest	ND	2.0	NS
	Bodega Head	3.0	2.4	ND
	Pacific Grove	7.3	8.4	NS
	Oceanside	ND	6.3	ND

1/ Not Sampled

2/ Not Detected (Detection Limit: 1 ug/Kg)

Figure II-2

ENDOSULFAN IN MUSSELS (Mytilus edulis) AT SANDHOLT BRIDGE, MOSS LANDING (Monterey Co.)

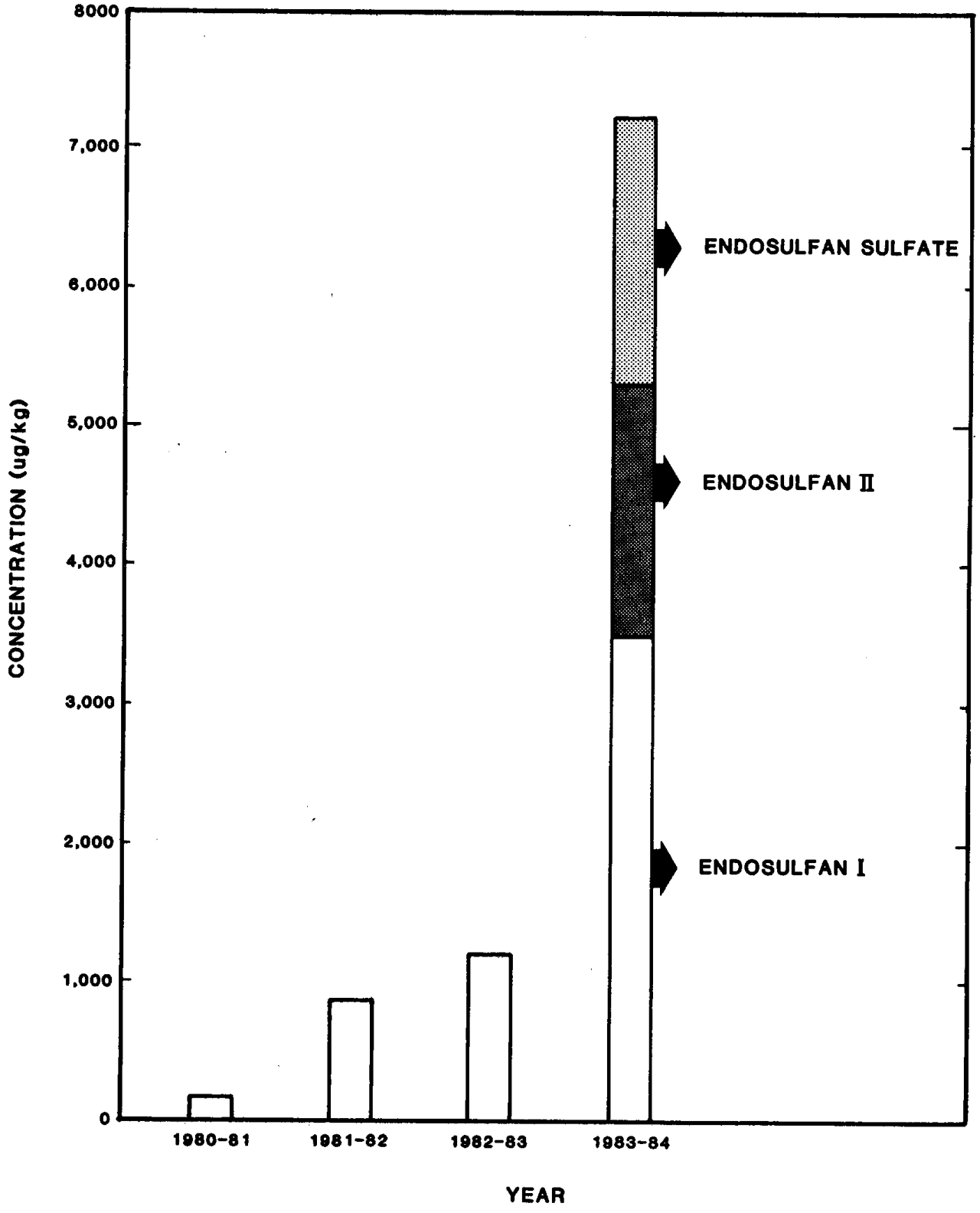


Figure II-3

MUSSEL TRANSPLANT SYSTEM USED IN CALIFORNIA MUSSEL WATCH (SWRCB, 1982)

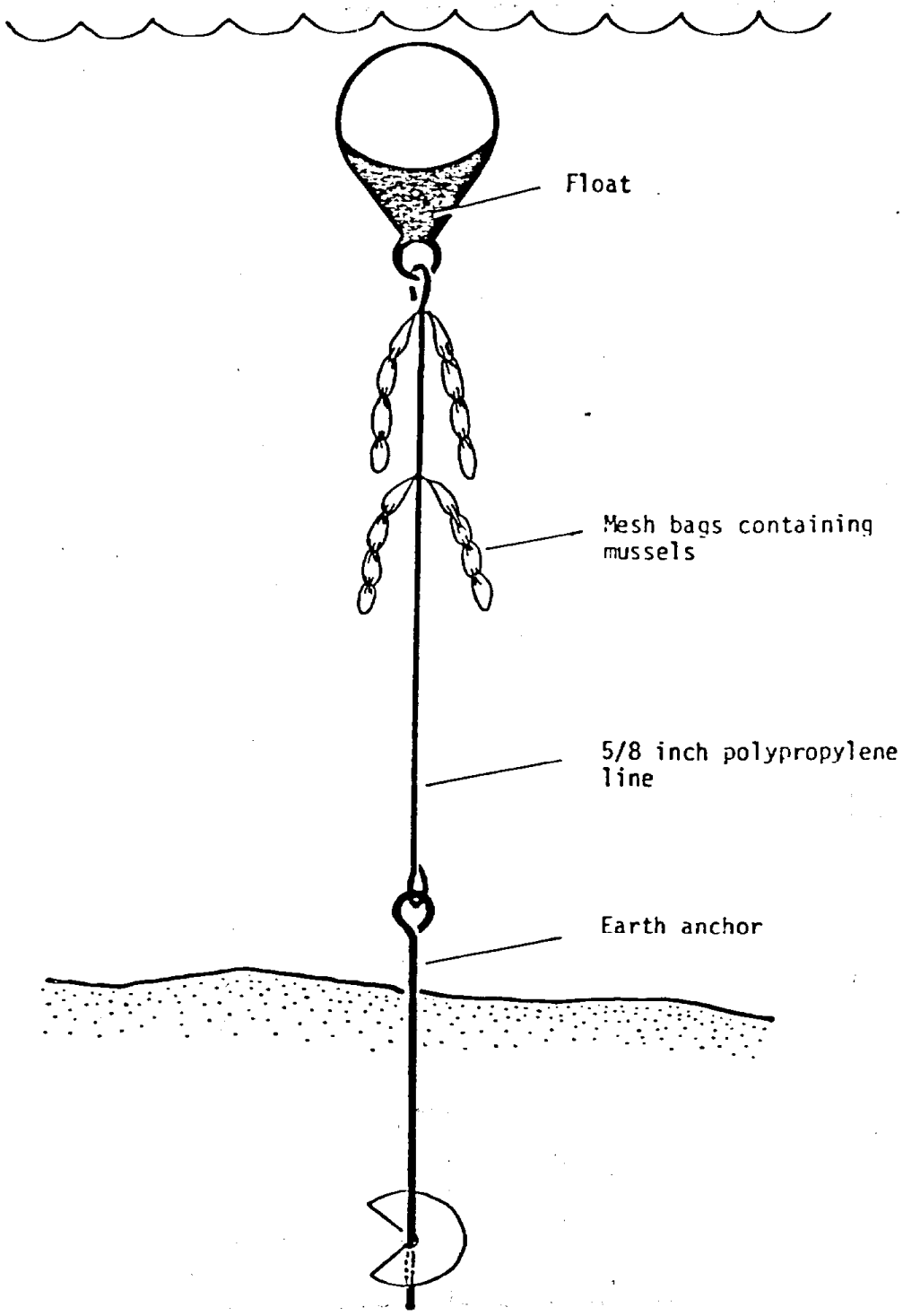


Table II-7

ENDOSULFAN I IN MUSSELS (*Mytilus californianus*) AT SELECTED CALIFORNIA ISLAND, BAY AND HARBOR TRANSPLANT STATIONS[1/]
(SWRCB Mussel Watch Program)

Site	Concentration (ug/kg, dry wt)		
	1979-80	1980-81	1981-82
Bodega Head	1.8	ND[2/]	ND[2/]
San Francisco Bay-Angel Island	ND	2.5	NS[3/]
Treasure Island	1.6	1.2	ND
Redwood Creek	NS	3.7	"
Dumbarton	NS	3.0	"
Bolinas Lagoon	1.7	ND	NS
Santa Cruz Harbor	3.0	26.0	"
Elkhorn Slough	24.0	140.0	"
Port Hueneme	2.3	11.0	"
Newport Bay - Island	ND	ND	3.2
Crows Nest	NS	NS	6.1

1/ Mussels were taken from their natural habitat at Bodega Head, transplanted to the identified sites and analyzed for toxicants after a 6-month period.

2/ Not Detected (Detection limit: 1 ug/kg)

3/ Not Sampled

Although mussels can depurate accumulated endosulfan residues with time (Figure III-1), even a short-term exposure to high residues of this pesticide can potentially impact the mussels and other aquatic organisms. Positive findings of endosulfan in mussels along the California shore indicate that the insecticide is not only entering the ocean but is also being concentrated to high levels by marine organisms which are generally more sensitive to endosulfan than fresh water aquatic species (Chapter III). For this reason, the EPA ambient water quality criteria for protection of saltwater species (24-hour average of 0.0087 ug/l, and instantaneous maximum of 0.034 ug/l) are lower than for freshwater species.

The SWRCB Mussel Watch Program has also found high residues of DDT and toxaphene in mussels from Monterey County sampling stations. For this reason, the County was designated as an "Action Site" for more intensive monitoring of pesticides. Endosulfan I concentrations in fish and mussel samples collected under this program are reported in Table II-8. Stickleback from Salinas River Slough had the highest concentration of endosulfan I (1,200 ppb) observed in any fish in California. Mussels also had significant amounts of endosulfan I (250 to 1,500 ppb). However, only two out of the seven water samples had detectable levels of endosulfan I (Table II-9), despite concentration through a resin column of a large volume of water (over 50 gallons). These results confirm that endosulfan residues are transient in a flowing water body, and that sediment and biota are therefore better media than water for endosulfan monitoring.

Large quantities of endosulfan are used in Monterey County (90,000 lb. in 1981) to control insect pests of artichokes, lettuce, celery, and strawberries. Burau et al. (1983) reported that 10 of the 50 environmental samples (soil, water and foliage) collected from Monterey County had detectable amounts of endosulfan (Table II-10). Some endosulfan concentrations found in surface water were greater than the EPA ambient water quality criteria (instantaneous maximum of 0.22 ug/l) for protection of aquatic life. High concentration (3.8 ug/l) of endosulfan in Salinas River suggests that the pesticide was being discharged to the river through drains and sloughs which receive endosulfan-laden irrigation return water. Endosulfan residues in soil ranged from 50 to 650 ppb. A strawberry foliage sample had a very high concentration (3000 ppb) of endosulfan. Critical evaluation of these soil and foliage residue data is not possible since the field treatment and sampling dates are not available. According to DFA staff (Leifson, 1984), the application rate of endosulfan to strawberries in 1982 averaged 1.8 lb/acre. If this amount were uniformly distributed in the top 4 inches of soil, the initial concentration of endosulfan in soil would be 2 ppm compared to the foliage concentration of 3 ppm (Table II-10).

Table II-8

ENDOSULFAN I IN FISH AND MUSSEL SAMPLES FROM MONTEREY COUNTY
(SWRCB Mussel Watch Program - "Action Site", 1982)

Species	Site	Concentration (ug/kg)[1/]
FISH		
Stickleback	Salinas River Slough	1,200
	Moro Cojo Slough	84
Sacramento blackfish	Tembladero Slough	110
MUSSEL		
Mytilus edulis (native)	Moro Cojo Slough	1,500
	Sandholt Bridge	1,200
M. californianus (transplant)	"	530
	Kirby Park	430
	Watsonville Slough	400
	Pearson's Slough	290
	PG&E Plant	250

1/ Fresh wt. basis for fish and dry wt. basis for mussels

Table II-9

ENDOSULFAN IN SURFACE WATER SAMPLES FROM MONTEREY COUNTY[1/]
(SWRCB Mussel Watch Program - "Action Site", 1982)

Site	Concentration (ng/l)	
	Endosulfan I	Endosulfan II
Blanco Drain	<48	<32
Old Salinas River Channel	< 1.2	<1
Elkhorn Slough	< 0.3	<0.5
Moro Cojo Slough	0.9	<0.5
Upper Tembladero Slough	< 6.2	<0.5
Espinoza Slough	<12	<5
Salinas River at Davis Road Bridge[2/]	5.8	NA[3/]

1/ Pesticides from a 50-gallon water sample were concentrated on a resin column prior to analysis.

2/ SWRCB Toxic Substances Monitoring Program, 1981

3/ Not Analyzed

Table II-10

ENDOSULFAN IN SELECTED MONTEREY COUNTY SAMPLES COLLECTED IN 1972
(Burau et al., 1983)

Date	Site	Medium	Concentration
			ug/l
10/25	Salinas Reclamation Canal at Airport Way	Surface water	2.5
8/22	Salinas River at Davis Road	"	3.8
"	Watsonville Slough near mouth	"	0.065
			ug/kg
3/20	NI Ranch	Soil	90
4/17	"	"	50
3/20	WN Ranch - east of Salinas	"	60
4/17	"	"	150
5/22	"	"	650
3/20	"	Strawberry (foliage)	3,000
7/17	WO Ranch near Greenfield Dump	Alfalfa	10

DEPARTMENT OF FISH AND GAME (DFG)

The Department of Fish and Game keeps a record of the fish and wildlife losses resulting from pesticides and other pollutants. Since 1963, 50 known endosulfan-related fish kills have been reported by DFG (Table II-11). This pesticide has been most often associated with fish kills in California. Fish kills caused by endosulfan may, however, be under reported for a variety of reasons. Fish kills may go unnoticed or be noticed only at an advanced stage. Under such conditions identifying the toxicant in decomposed fish or catching the band of endosulfan in flowing water may be impossible. Whenever a fish kill is reported, DFG attempts to collect and analyze samples of fish, water (at least 500 ml) and vegetation to determine the cause of death. Table II-12 gives the concentrations of endosulfan I in water and dead fish tissues reported by DFG. Even when concentrations of endosulfan I in water were very low (<1 ppb), fish had accumulated up to 17.5 ppm of the insecticide. Thus minute levels (<1 ppb) of endosulfan in water or sediment can result in a fish kill, even though by the time a water sample is collected, endosulfan concentrations may have declined from some acutely toxic peak to a non-detectable level.

The Department of Fish and Game classifies a fish kill as "known" to be caused by a specific chemical when analysis of fish and/or water reveals lethal concentrations of the chemical. The Department attempts to infer the cause of fish kills where chemical analysis is not possible. If endosulfan is known to have been recently applied to a field near a canal where a fish kill occurred, the "certainty" of the kill being endosulfan-related is reported as "probable" or "possible" (Table II-11). According to DFG staff, the Department does not necessarily take legal action against the discharger causing the fish kill unless there is conclusive evidence to support the prosecution (Day, 1984). Most of the endosulfan-related fish kills in California reported by DFG were localized in drains and canals close to an endosulfan-treated field, and resulted from an acute exposure to agricultural return water runoff or drift (Day, 1984).

DEPARTMENT OF WATER RESOURCES (DWR)

DWR monitors for pesticides in the drains of the San Joaquin Valley. This monitoring effort developed into a special program in 1975, when DWR, SWRCB, and U.S. Bureau of Reclamation (USBR) formed the San Joaquin Valley Interagency Drainage Program (IDP).

One of the goals of the IDP was to develop a plan to protect the quality of ground and surface water in the valley by providing necessary facilities to dispose of agricultural wastewater. When the IDP was completed in 1979, monitoring was resumed by DWR as a separate program. The locations sampled by DWR are shown in Figure II-4. In 1975 and 1976 DWR reported findings of up to 0.035 ppb endosulfan I in the drain water. Drain water

Table II-11

REPORTED FISH KILLS IN CALIFORNIA INVOLVING ENDOSULFAN
(California Department of Fish and Game)

Year	Number of Episodes	Certainty of Cause			Cumulative Death Toll
		Known	Probable	Possible	
		(Number of dead fish)			
1983	2	1,000			1,000
1982	3	2,100			2,100
1981	2	3,100			3,100
1980	1	1,000			1,000
1978	4(1)[1/]	12,000	8,000		20,000
1977	3		9,030	1,200	10,230
1976	8(1)	2,700	3,000	3,100	8,800
1975	4(1)	2,000		500	2,500
1974	3	10,550		12,750	23,300
1973	2(1)	1,100			1,100
1972	3(1)			15,000	15,000
1971	2	7,535			7,535
1969	3	3,650			3,650
1966	6	19,150	1,325	1,000	21,475
1965	1	9,360			9,360
1964	2	5,050			5,050
1963	1			15,000	15,000
			GRAND TOTAL		150,200

[1/] Number in parenthesis refers to fish kill episodes without dead fish count

Table II-12

ENDOSULFAN (I/II) IN FISH AND WATER SAMPLES FROM FISH KILL EPISODES
(California Department of Fish and Game)

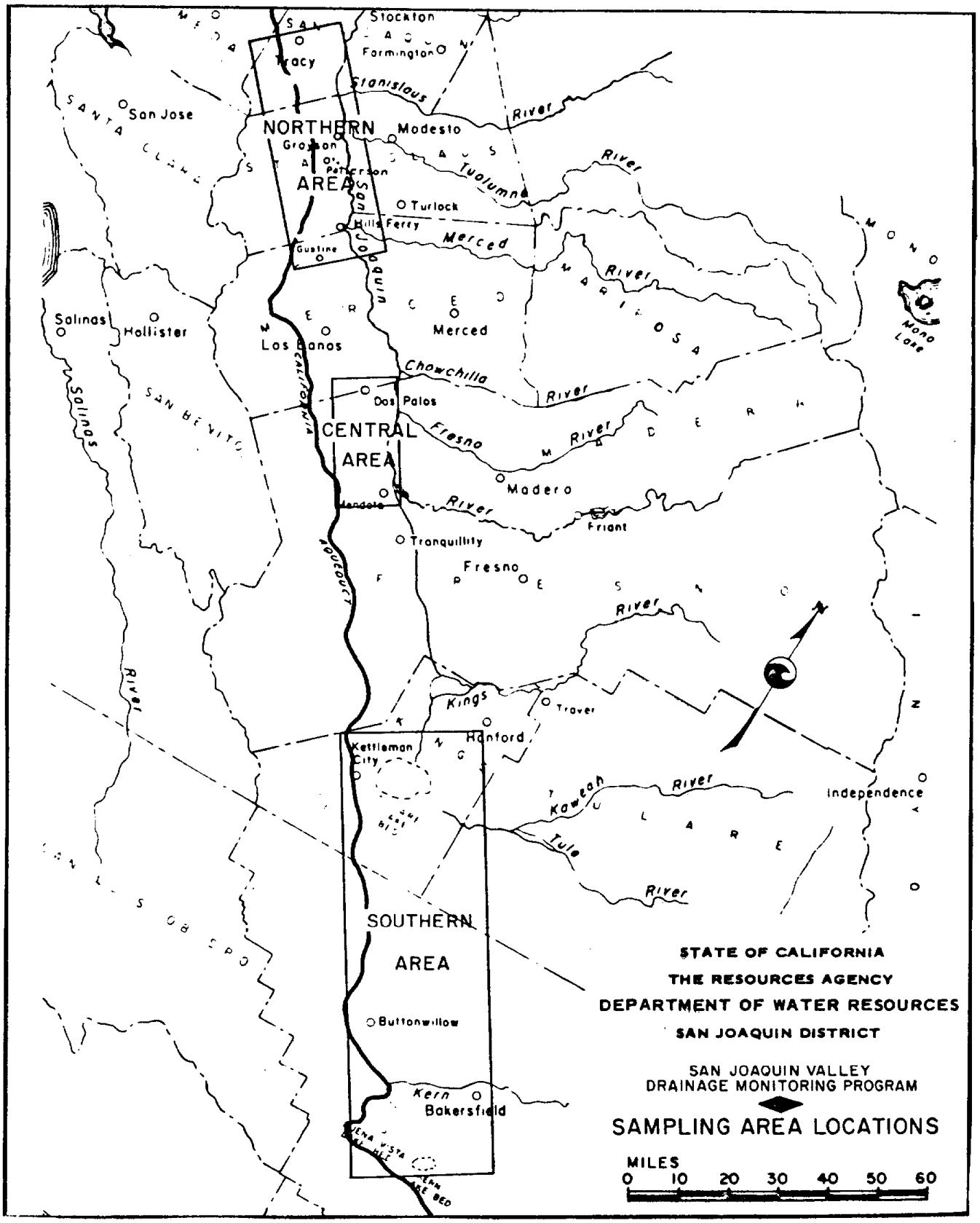
Site	Date (mo/yr)	Fish species and tissue analyzed	Concentration	
			Fish (ug/kg, fresh wt)	Water (ug/l)
Drain off 8th Ave. Riverside Co.	9/83	Carp gill	500	0.22 - 052
D23-1 Canal, Palo Verde Valley	8/83	Carp gill	602	1.02
		Largemouth bass gill	542	
Lotus Canal, Imperial County	10/82	Catfish gill	410	ND[1/]
		Goldfish gill	300	
Fillaree Canal, Imperial Co.	1/82	Carp gill	800	3.5 - 26
		Carp skeletal muscle tissue	200	
CO-3 Canal, Riverside Co.	1/78	Catfish liver	2,500	0.27 - 0.64
		Catfish gill	1,100	
		Largemouth bass gill and gut	930	
Vail Cutoff Drain Imperial Co.	11/77	Carp gill	2,650	1.33
		Carp GI tract	1,920	
2,047 Canal, Colusa Co.	6/77	Bass liver	1,400	0.07 - 7.9
		Catfish liver	200	
		Carp digestive tract	710	
Rice Drain #3, Imperial Co.	1/77	Carp liver and intestine	1,400	NA[2/]
Unspecified Canal, Imperial Co.	12/76	Carp digestive tract and liver	3,000	0.1 - 0.2
		Carp gills	1,200	
Canal 16, Palo Verde Valley	10/76	Carp gills and digestive tract	17,500	0.01 - 0.08

1/ Not detected

2/ Not analyzed

Figure II-4

SAMPLING LOCATIONS IN THE SAN JOAQUIN VALLEY IDP
(DWR, 1981)



monitoring data from DWR and other sources are given in Table II-13. The frequency of detection of endosulfan I in drain water samples ranged from 8.3 percent in San Joqaquin Valley to 45.8 percent in Imperial Valley sites shown in Figures II-5 and 6. The source of these residues may be irrigation tailwater.

Spencer et al. (1984) monitored pesticide concentrations in irrigation runoff water following the application of 20 pesticides to large fields of six different crops. As high as 104 ug/l of endosulfan (I and II) were detected in the runoff water from a melon field (Table II-14). The concentration of endosulfan in the irrigation runoff water was proportional to the amount of the pesticide applied (melons > lettuce > cotton) as well as time elapsed since the last application.

REGIONAL WATER QUALITY CONTROL BOARD (RWQCB)

The Regional Boards' monitoring activities are primarily for discharges from point sources such as pesticide formulation facilities and waste disposal sites. Regional Board monitoring data for endosulfan in ground water are presented in Table II-15. Up to 100 ppb of endosulfan were detected in 14 of 34 wells sampled; 11 of the positive wells were located in the vicinity of pesticide formulation/disposal facilities. According to DFA staff, the validity of the Riverside County GHT Lab data is questionable (Knaak, 1984).

Under normal agricultural practices, endosulfan use will not be expected to cause ground water contamination since the pesticide is adsorbed by soil components. The potential for ground water contamination could exist in situations where sandy soils were combined with a shallow water table, intense rainfall or irrigation and cumulative high use of the pesticide.

The Central Coast Regional Water Quality Control Board (Region 3) surveyed the pesticide rinse water disposal practices of retail businesses in Monterey and Santa Cruz Counties (Jones and Van Voris, 1980). Endosulfan residues of up to 250 ppm in soil and 60 ppb in storm drain effluent were measured at some sites.

The Colorado River Basin Region (Region 7) studied the methods utilized by pesticide applicators in the region for the disposal of pesticide spray equipment washwater. The survey indicated that waste liquid in the 15 evaporation basins sampled contained 4.9 to 2,300 ppb of endosulfan I (Table II-16). Surface soil sampled from disposal areas contained 4.7 to 5,900 ppm of endosulfan I. At a spray disposal site, 16 ppm of endosulfan I were detected in soil at a depth of four feet (RWQCB 7, 1982). This shows that endosulfan can potentially migrate to subsurface soil layers at waste disposal sites. Unfortunately, soil samples were not taken from below the four-foot level at this site.

Table II-13

ENDOSULFAN I IN DRAIN WATER SAMPLES

Drain Location	Year	Frequency of detection	Range of conc. (ug/l)	Reference
San Joaquin Valley[1/]	1976	1/18 (5.5%)Surface 2/80 (2.5%)Subsurface	0.005 0.005-0.01	DWR, 1977
	1975	1/12 (8.3%)Surface 1/114 (<1%)Subsurface	0.035 0.02	DWR, 1976
Imperial Valley (Mostly at boundaries, outlet and drops of New and Alamo rivers)	1978	22/48 (45.8%)	0.01 - 0.26	Imperial Irrigation District, 1978
South-eastern desert area (Imperial Coachella, Bard and Palo Verde Valleys)[2/]	1976/77	53/119 (44.5%)	0.01 - 1.7	Eccles, 1979

[1/] Sampling locations are shown in Figure II-4

[2/] Sampling locations are shown in Figures II-5 and 6

Figure II-5

LOCATION OF DRAIN SAMPLING SITES IN IMPERIAL VALLEY
(Eccles, 1979)

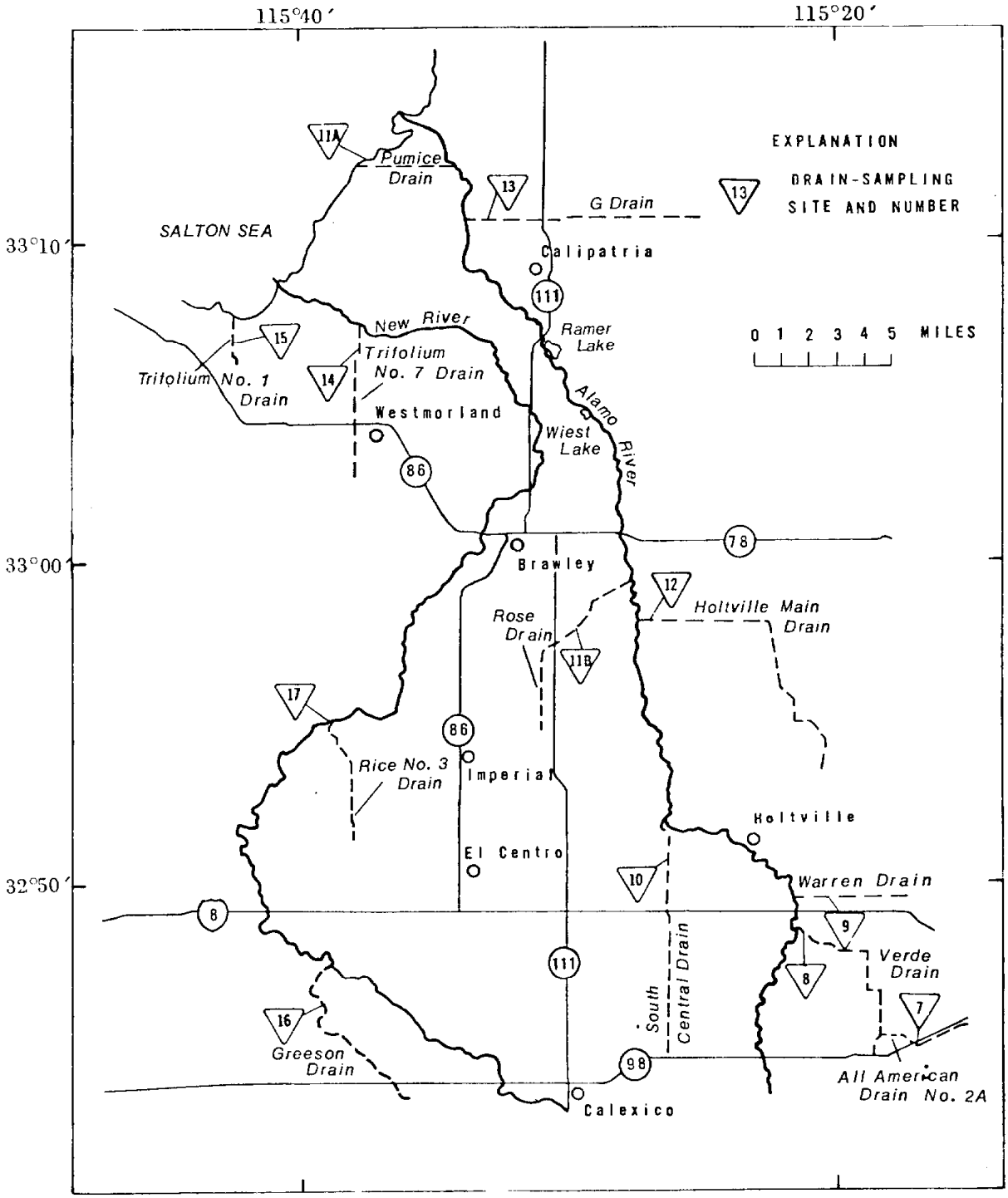


Figure II-6

LOCATION OF DRAIN-SAMPLING SITES IN COACHELLA, PALO VERDE AND BARD VALLEYS
(Eccles, 1979)

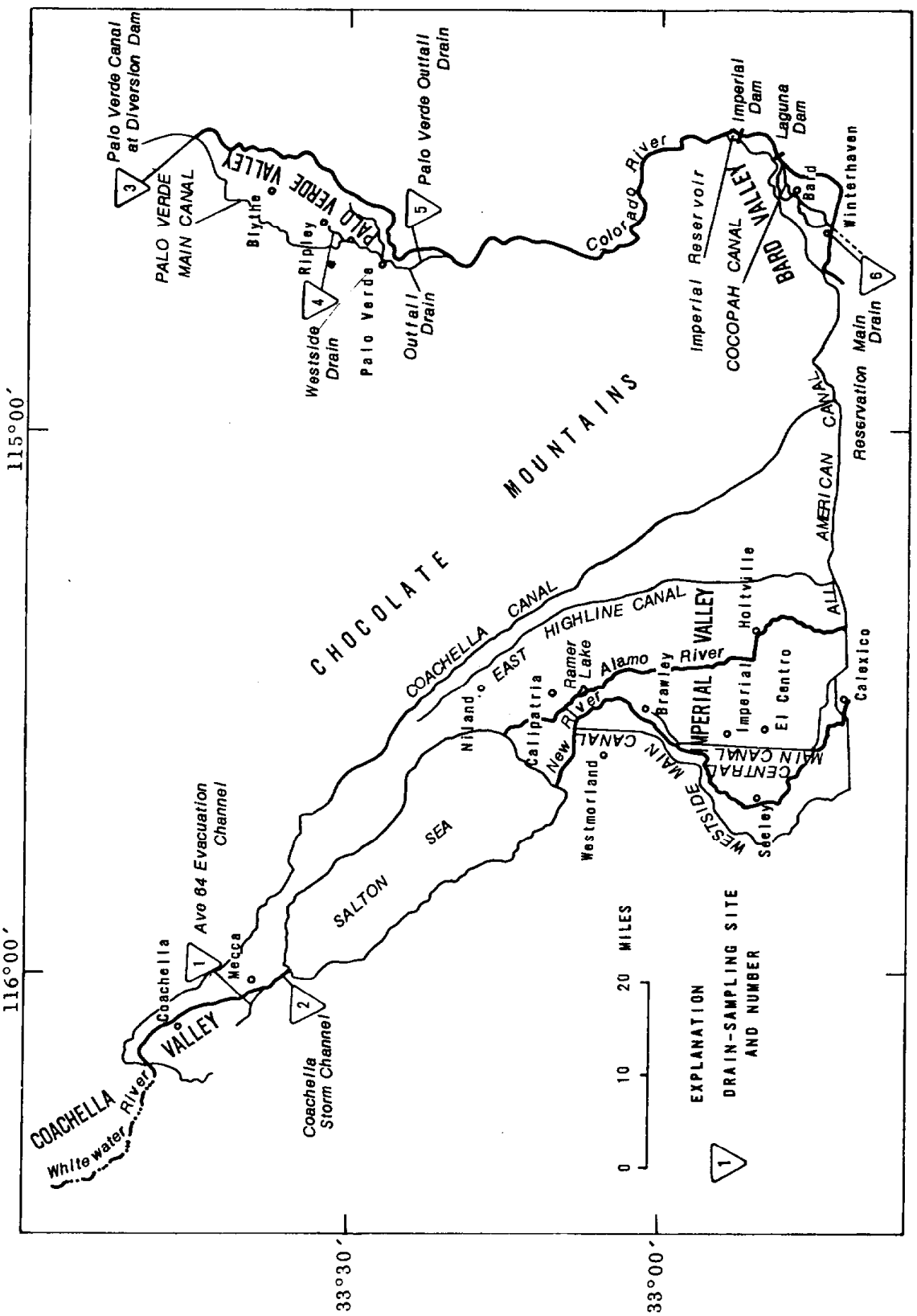


TABLE II-14

ENDOSULFAN IN IRRIGATION RUNOFF WATER
IN IMPERIAL VALLEY

(Spencer et al., 1984)

Crop	Endosulfan Applied (lb/A)	Days Since Last Pestic. Application	Residues in Irrigation Water runoff: Max. Conc. (ug/l)	Mean Conc. (ug/l)	Total Amt. (lb/A x 10^{-3})	Percent Total Applied
Melons	1.05	4	73	36	14.6	1.4
	2.10	2	99	51	15.2	0.5
	2.10	4	104	71	2.4	0.05
Total	5.25	-	- -	--	32.2	0.62
Lettuce	0.75	10	9.3	6.2	.54	0.07
		24	4.9	3.2	.24	0.03
	0.75	14	30	21.7	2.0	0.14
Total	1.5	-	- -	--	2.78	0.19
Cotton	0.7	110	0.7	0.4	0.63	0.8

Table II-15

ENDOSULFAN IN GROUND WATER SAMPLES [1/]

Year	County	No. of Wells sampled	No. of affected wells	Max. conc. found (ug/l)	References
1980[1/]	Monterey/ Santa Cruz	6	3	0.002[2/]	Jones and Van Voris, 1980
1979[1/]	Contra Costa	16	4	100	Todd, 1981
1979[1/]	Fresno	4	4	19	Lewis, 1983
1975[3/]	Riverside	8	3	0.75	GHT Lab, 1975

1/ Regional Board information on point-source discharges (e.g., pesticide formulation or disposal facilities)

2/ City water supply well in Watsonville

3/ Water supply wells

Table II-16

ENDOSULFAN I IN SOIL AND WASTE LIQUID AT OR NEAR PESTICIDE DISPOSAL FACILITIES IN IMPERIAL AND RIVERSIDE COUNTIES
(RWQCB 7, 1982)

Sampling Medium	No. of Samples	Conc. Range
		ug/l
Liquid from earthen basin	6	110 - 2,300
Liquid from concrete basin	9	4.9 - 2,100
		mg/kg
Bottom mud from evaporation basins	3	0.35 - 450
Soil from disposal (spreading) area	3	4.7 -5,900 (surface) 1.4 - 22 (1 ft. depth)
Soil from spray disposal area	3	280 -1,200 (surface) 3.5 - 9.7 (2 ft. depth) 3.1 - 16 (4 ft. depth)

Region 7 staff in cooperation with the U.S. Geological Survey (USGS) sampled agricultural drains for pesticides in the southeastern desert area of California (Figures II-5 and II-6). Residues for 29 of the 33 pesticides selected for monitoring were found in the drain waters (Eccles, 1979); nearly half of the samples (44.5%) contained endosulfan I at concentrations of up to 1.7 ug/l (Table II-13 and Figure II-7). This maximum concentration is significantly higher than the EPA recommended ambient water quality criterion for freshwater (instantaneous maximum) of 0.22 ug/l. According to Eccles (1979), the sources of pesticides in the drain waters are mainly from irrigation tailwater and drift from aerial applications.

San Diego Regional Water Quality Control Board (Region 9) detected as high as 146 ppb of endosulfan in irrigation return water discharges from a nursery in San Diego (Table II-17). This maximum value is over 600 times the EPA's criterion of 0.22 ppb (freshwater instantaneous maximum). At the Regional Board's recommendation, the nursery has terminated surface discharges and is currently discharging to the sewer system (Barker, 1984).

DEPARTMENT OF FOOD AND AGRICULTURE (DFA)

One of the DFA's pesticide monitoring programs is the retail food market survey. In 1976 DFA found endosulfan residues over the tolerance level of 2 ppm on several leafy vegetables (watercress, spinach, red leaf lettuce, and cabbage), and some of the produce was sold for human consumption (Table II-18). In the absence of information on total number of products sampled, analytical methodology, and pesticide use patterns on these commodities, the significance of these data cannot be completely assessed. It is apparent though that some consumers were potentially exposed to residues of endosulfan which were above the tolerance level. The Department has stated that in recent years endosulfan residues have not been detected in any food samples surveyed (Nash, 1983). However, Mott and Board (1984) found endosulfan residues below tolerance level in two of the 71 fruit and vegetable samples collected from four retail food stores in San Francisco. A strawberry sample had 0.14 ppm of endosulfan while a lettuce sample had 0.04 ppm.

Figure II-7

MEDIAN CONCENTRATION OF ENDOSULFAN AND FREQUENCY OF DETECTION, BY SITE, BASED ON SEVEN SAMPLES COLLECTED BETWEEN NOVEMBER 1976 AND OCTOBER 1977
(Eccles, 1979)

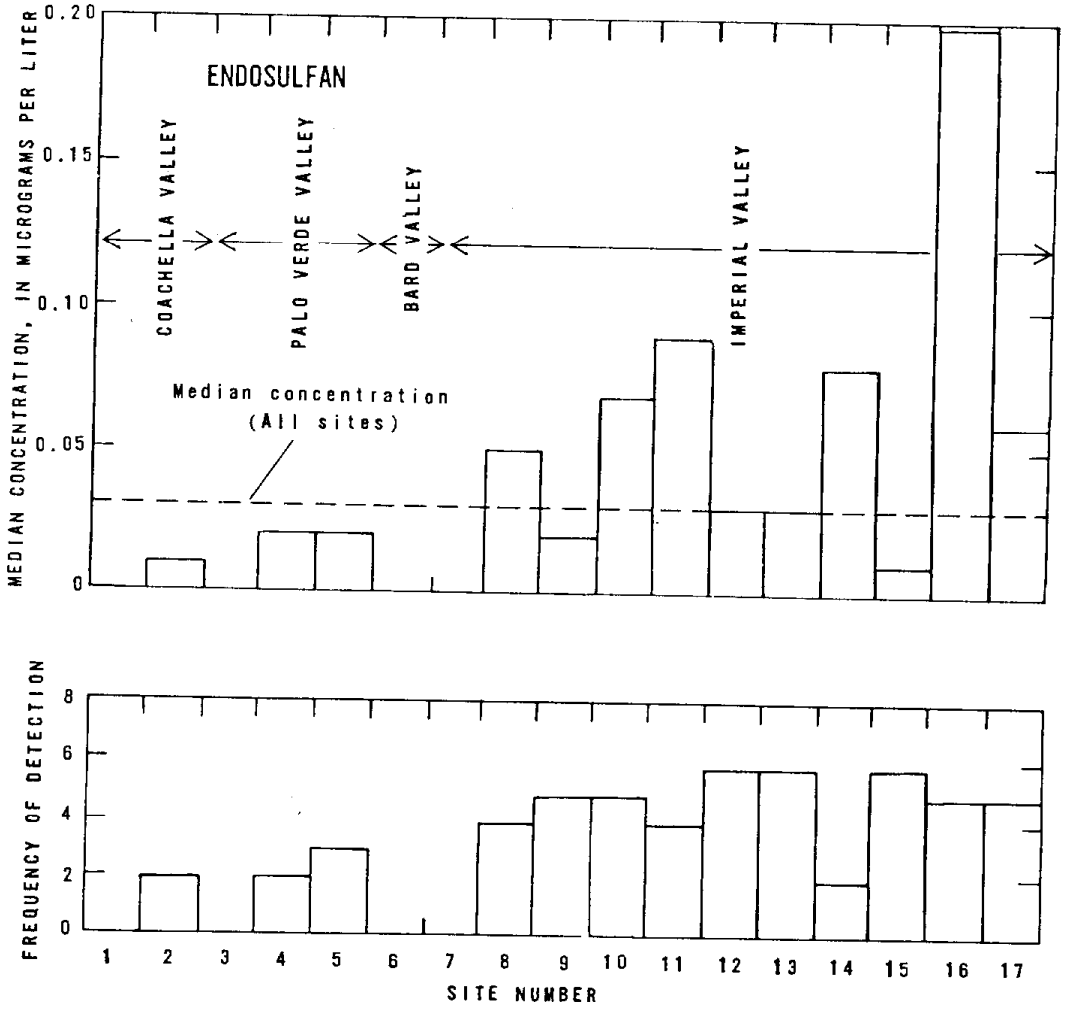


Table II-17

ENDOSULFAN IN IRRIGATION RETURN WATER
RUNOFF FROM A NURSERY IN SAN DIEGO[1/]

Sampling Date	Endosulfan Concentration (ppb)			
	I	II	Sulfate	Total
June 9, 1982	2.2	2.2	4.2	8.6
June 14, 1982	36.0	32.0	30.0	98.0
June 22, 1982	79.0	42.0	25.0	146.0
Aug 10, 1982	NR[2/]	NR	NR	0.1-0.2
Sept. 24, 1982	ND[3/]	ND	ND	-
Nov. 9, 1982	NR	26.4	NR	26.4
Nov. 9, 1982[4/]	NR	NR	NR	50.0
Nov. 9, 1982[5/]	34.8	NR	10.0	44.8
Nov. 9, 1982[6/]	17.5	NR	NR	17.5
April 26, 1983	11.0		9.0	20.0

1/ Samples collected by RWQCB (IX) and analyzed by Environmental Eng. Laboratory and Quality Assurance Laboratory, San Diego.

2/ Not reported

3/ Not detected (Detection Limit 0.02 ppb)

4/ Sample collected by neighborhood resident

5/ Road sediment sample

6/ Canyon sediment sample

Table II-18

ENDOSULFAN IN VEGETABLE SAMPLES
(Coleman and Dolinger, 1978)

Produce	District	Date	Quantity	Endosulfan residue (ppm)[1/]	Disposition
Watercress	Berkeley	2-18-76	29 crates	5.0	Destroyed
"	"	2-19-76	35 cartons	4.4	Destroyed
"	"	Not stated	5 cartons	2.8	Sold pending analysis
"	"	Not stated	23 crates	3.4	Sold pending analysis
Spinach	"	8-16-76	1 carton	2.14	Destroyed
"	"	Not stated	4 cartons	2.05	Sold pending analysis
Red leaf lettuce	Downey	Not stated	8 cartons.	5.0	Sold pending analysis
Cabbage	Downey	Not stated	80 cartons	2.10	Sold pending analysis

[1/] Tolerance level: 2 ppm

III. RISK ASSESSMENT

The potential risks associated with the use of endosulfan are assessed for the non-target organisms, aquatic life and humans.

AQUATIC TOXICOLOGY

Endosulfan is extremely toxic to fish and other aquatic species. It was found to be second in toxicity only to endrin in acute studies of fish species with both organochlorine and organophosphate insecticides (Macek et al., 1969). Korn and Earnest (1974) also reported that among 20 pesticides tested for their toxicity to striped bass, endosulfan was second in toxicity only to endrin.

Acute Toxicity

Endosulfan concentrations acutely toxic to freshwater aquatic organisms are given in Table III-1. Fish are more sensitive to endosulfan than invertebrates. For protection of freshwater aquatic life from short-term acute effects of endosulfan, EPA in 1980 established an ambient water quality criterion of 0.22 ug/l as an instantaneous maximum. At this concentration, it is assumed that 95 percent of freshwater aquatic life would be protected from endosulfan acute toxicity. The remaining five percent would, however, be adversely affected. For instance, the lowest reported LC50 value for rainbow trout is 0.17 ug/l (Table III-1).

Table III-2 gives the endosulfan concentrations acutely toxic to saltwater species. The LC50 values indicate that saltwater species are generally more sensitive to endosulfan compared to freshwater species. The lowest LC50 for a saltwater fish is 0.09 ug/l. Saltwater invertebrates such as pink shrimp (LC50: 0.04 ug/l) appear to be more susceptible to endosulfan than saltwater fish. The EPA (1980) established criterion for the protection of saltwater life from short-term acute effects of endosulfan is 0.034 ug/l. This is an order of a magnitude lower than the corresponding freshwater criterion (0.22 ug/l).

Endosulfan toxicity to aquatic organisms may be irreversible. Schoettger (1970) reported that in a rainbow trout bioassay, individuals surviving after 120 hours in a 0.7 ug/l solution died within a week when removed to fresh water. Ludemann and Neuman (1960) found that carp exhibiting symptoms of endosulfan poisoning usually did not recover when removed to clean water.

Schoettger (1970) described the symptoms of endosulfan poisoning in trout and suckers. The fish at first seem overly excitable and swim rapidly about. Later they surface, lose equilibrium and move with spasmodic jerks.

Table III-1

ENDOSULFAN CONCENTRATIONS ACUTELY TOXIC TO FRESHWATER AQUATIC LIFE[1/]

Test Species	Number of Tests	LC50/EC50 Values (ug/l) Lowest	Highest	Mean
FISH				
Rainbow trout (Salmo gairdneri)	29	0.17	2.6	0.34
Fathead minnow (Pimephales promelas)	24	0.29	3.45	0.83
White sucker (Catostomus commersoni)	2	3.0	3.5	3.2
Guppy (Poecilia reticulata)	1	3.7	--	3.7
Bluegill (Lepomis macrochirus)	2	3.3	4.4	3.8
INVERTEBRATES				
Stonefly (Pteronarcys californica)	1	2.3	--	2.3
Scud				
(Gammarus lacustris)	1	5.8	--	5.8
(G. fasciatus)	1	6.0	--	6.0
Damselfly (Ischnura sp.)	2	71.8	107	88
Water flea (Daphnia magna)	15	62	740	261

1/ Summarized from U.S. EPA, 1980

Table III-2

ENDOSULFAN CONCENTRATIONS ACUTELY TOXIC TO SALTWATER AQUATIC LIFE[1/]

Test Species	Number of Tests	LC50/EC50 Values (ug/l) Lowest	Highest	Mean
FISH				
Striped bass (Morone saxatilis)	1	0.1	--	0.1
Spot (Leiostomus xanthurus)	1	0.09	--	0.09
Pinfish (Lagodon rhomboides)	1	0.3	--	0.3
Striped mullet (Mugil cephalus)	1	0.38	--	0.38
Sheephead minnow (Cyprinodon variegatus)	11	0.34	3.45	0.76
INVERTEBRATES				
Pink shrimp (Penaeus duorarum)	1	0.04	--	0.04
Copepod (Acartia tonsa)	6	0.032	0.45	0.14
Mysid shrimp (Mysidopsis bahia)	10	0.24	1.47	0.83
Grass shrimp (Palaemonetes pugio)	1	1.31	--	1.31
Korean shrimp (P. macrodactylus)	2	3.4	17.1	7.6
Eastern oyster (Crassostrea virginica)	2	65	380	157
Annelid worm (Neanthes arenaceodentata)	1	730		730

1/ Summarized from U.S. EPA, 1980

In time, the majority sink to the bottom and opercular (gill-flap) movements become eratic. Many of the trout appeared darker in color and the suckers appeared mottled before death.

Joshi and Rege (1980) reported that Thiodan 35EC (a formulated product containing endosulfan as an emulsifiable concentrate) was more toxic to mosquito fish than technical grade endosulfan. The lowest 96-hour LC50 for Thiodan 35EC was 2.8 ug/l as compared to 7.6 ug/l for technical endosulfan. It appears that chemicals used as emulsifiers may increase endosulfan toxicity to fish either directly or secondarily due to depletion of oxygen by microbial degradation (Fischer, 1984).

Toxicity of endosulfan to aquatic life depends on a number of factors such as: (1) species, (2) life stage, (3) flow-through versus static bioassay, (4) exposure period, (5) temperature, and (6) salinity.

Species and life stage: The LC50 values of endosulfan to freshwater fish range from 0.17 to 4.4 ug/l depending on the species (Table III-1). Knauf and Schulze (1973) reported that fish are 1,000 times more sensitive to endosulfan than worms and snails (Table III-3). The order of sensitivity was: fish>crabs>snails>worms. Age, weight and size of an organism also modify endosulfan toxicity. Shoettger (1970) found older or heavier fish more resistant to endosulfan. Usually the younger stages of an organism are more susceptible to a toxicant. Data presented in Table III-3 also suggest that the toxicity of endosulfan I, II and sulfate to aquatic organisms is in the following order:

Fish:	I>II>Sulfate
Crustacean:	Sulfate>II>I
Mollusca:	I>Sulfate>II

Flow-through versus static bioassay: It has been established by many investigators that the toxicity of endosulfan may be underestimated in static bioassays compared to constant flow testing. Unfortunately, most of the LC50 and EC50 values reported in Tables III-1 and 2 were derived from static tests. Lemke (1980) reported that endosulfan was three times more toxic to rainbow trout and two times more toxic to fathead minnows in flow-through tests compared to static tests. Nebeker et al. (1983) also found that the toxicity of endosulfan to rainbow trout in flow-through experiments (mean LC50: 0.35 ug/l) was five times higher than in static experiments (mean LC50: 1.65 ug/l). However, they reported that the differences between fathead minnow static and flow-through values were small.

Table III-3

ACUTE TOXICITY OF ENDOSULFAN AND ITS METABOLITES TO AQUATIC ORGANISMS

(Knauf and Schulze, 1973)

Species	LC50 (48-hour), ug/l							
	Technical Endosulfan	Endosulfan I	Endosulfan II	Endosulfan sulfate	Endosulfan lactone	Endosulfan diol	Endosulfan ether	Endosulfan alpha hydroxy ether
Fish								
Idus melanotus	1.8	1.3	1.5	12.5	750	500	-	-
Guppy (Lebistes reticulatus)	1.4	1.4	0.8	1.6	25,000	7,500	2,500	900
Goldfish (Carassius auratus)	1.8	1.5	10	17.5	5,000	7,500	3,500	-
Crustacea								
Water flea (Daphnia magna)	140	17.5	130	140	50,000	500	750	250
Brine shrimp (Artemia salina)	10,000	5,000	2,500	750	>100,000	10,000	20,000	-
Mollusca								
Planorbis corneus	1,000	2,500	5,000	4,000	100,000	100,000	100,000	10,000
Limnea stagnalis	1,200	3,000	7,500	6,000	50,000	10,000	3,000	-
Physa sp.	500	2,500	7,500	750	70,000	-	6,000	-
Annelida								
Sludge worm (Tubifex tubifex)	3,500	7,500	1,000	2,500	-	40,000	90,000	-

Herzel and Ludemann (1971) studied the effect of aeration on the water concentration of endosulfan in static tests. After a 96-hour period, endosulfan concentration decreased more than six-fold the initial concentration in the unaerated treatment. In the aerated treatment, however, the decrease was greater than 40 fold. This may be due to an increase in the rate of volatilization or degradation. The results of these studies indicate that the effective exposure concentration in aerated static tests may be considerably underestimated. The levels of endosulfan in water should be monitored during the bioassay. Without this data, a meaningful statistic such as LC50 cannot be calculated.

Exposure period: Endosulfan toxicity increases with an increase in exposure time (Schoettger, 1970). At the longer exposure periods, the LC50 (median tolerance limit) values were substantially lower than for shorter periods (Table III-4). For instance, the 24-hour LC50 value for rainbow trout was 5.9 ug/l compared to 0.7 ug/l for a 120-hour duration. This may be due to the accumulation of endosulfan in the test organism which, in turn, may lead to irreversible nerve damage over time (Day, 1984). The LC50 values generally reached a minimum in less than 120 hours, since the 96-hour LC50 for rainbow trout (0.8 ug/l) was not significantly different from the 120-hour LC50 (0.7 ug/l).

Temperature: Temperature plays a significant role in endosulfan toxicity as illustrated in Table III-4. Toxicity of endosulfan increases with an increase in temperature. The 24-hour LC50 for rainbow trout decreased from 5.9 ug/l at 1.5°C to 2.1 ug/l at 10°C, a three-fold increase in toxicity (Schoettger, 1970). The exception to this general rule was the endosulfan toxicity to damselfly (as well as sucker and Daphnia at 120-hour) which decreased with an increase in temperature (Table III-4).

Salinity: As noted earlier, saltwater aquatic life (Table III-2) is more sensitive to endosulfan than freshwater aquatic life (Table III-1). Greve and Verschuuren (1971) reported an increase in sensitivity of guppies to endosulfan with increase in water salinity. However, Oeser et al. (1971) found that when guppies were adapted to seawater, the toxicity of endosulfan in saltwater was not substantially different from fresh water. As with many other studies quoted in this report, due to the lack of statistical analysis of the data, it is not possible to determine the statistical significance of these differences.

Pickering and Henderson (1966) observed no significant effect of water hardness on endosulfan toxicity. The 96-hour LC50 values for bluegill exposed to technical grade endosulfan in soft and hard water were 3.3 and 4.4 ug/l,

A FUNCTION OF EXPOSURE TIME AND TEMPERATURE

(Schoettger, 1970)

Test Species	Temperature (°C)	Median Tolerance Limit (ug/l) 24-hour	72-hour	120-hour
Rainbow trout	1.5	5.9	1.4	0.7
	10	2.1	0.4	0.3
Western white sucker	10	8.1	4.9	2.5
	19	6.6	3.1	2.8
Daphnia	10	178	87.5	47.5
	19	68	60.5	53.5
Damselfy naiads	8	235	84.5	62
	19	275	150	75

respectively. Schoettger (1970) found that the presence of 500 mg/l of magnesium and calcium salts in test solutions did not affect the toxicity of endosulfan to western white suckers.

Chronic Toxicity

Chronic effects of chemicals, such as increase in susceptibility to disease and predators or decrease in adaptability to changes in the environment, probably occur more frequently and often go unnoticed. The few endosulfan chronic toxicity studies with observable effects (such as growth and survival) reported in the literature are summarized in Table III-5. Macek et al. (1976) studied the survival, growth and reproduction of fathead minnow in a chronic life-cycle bioassay which lasted for 40 weeks. They observed no statistically significant adverse effects on parental fish or offspring at 0.2 ug/l endosulfan. However, when three separate groups of eggs from control spawns were incubated in 0.4 ug/l endosulfan, only one percent of these eggs hatched successfully. Without endosulfan treatment, 83 percent of eggs in control tanks hatched. The chronic limits for fathead minnows are therefore between 0.2 and 0.4 ug/l. The geometric mean of these two numbers gives the chronic value of 0.28 ug/l (EPA, 1980).

In another chronic toxicity study, sheepshead minnows were continuously exposed to endosulfan for 28 days, starting with newly fertilized eggs to the juvenile stage (EPA, 1980). Survival of juveniles exposed to endosulfan concentrations greater than 1.3 ug/l was significantly less than that of the controls. Average standard lengths of fish exposed to concentrations greater than 0.6 ug/l were significantly less than that of controls.

Macek et al. (1976) reported that the survival of daphnids exposed to 7 ug/l endosulfan for 22 days was significantly reduced. This effect of endosulfan on survival of daphnids was cumulative since the survival of the second generation daphnids was significantly lower than that of the first generation.

In a 28-day life-cycle study with a saltwater mysid shrimp, survival and reproduction (number of young per female) were affected at 0.71 ug/l but not at 0.33 ug/l. The geometric mean of these two numbers gives the lowest assumed chronic value of 0.48 ug/l (EPA, 1980).

As discussed in Appendix VI (Criteria and Standards), the EPA (1980) ambient water quality criterion for protection of freshwater aquatic life from long-term chronic effects of endosulfan is 0.056 ug/l (24-hour average). The corresponding saltwater criterion is 0.0087 ug/l.

Table III-5

ENDOSULFAN CONCENTRATIONS CHRONICALLY TOXIC TO AQUATIC LIFE

Test Species	Effect 1/	Lowest Conc. Showing Effect (ug/l)	Length of Exposure (days)	Life Stage	Reference
Freshwater fish: Fathead minnow (Pimephales Promelas)	Survival (All fish died)	0.4	117-145	A month from onset of spawning	Macek et al., 1976
Saltwater fish: Sheephead minnow (Cyprinodon variegatus)	1. Length	0.6	28	Newly fertilized egg to juvenile	U.S. EPA, 1980
	2. Survival of juveniles	1.3			
Freshwater invertebrate: Water flea (Daphnia magna)	Survival	7.0	22 (Life Cycle)	Less than a day old	Macek et al., 1976
Saltwater invertebrate: Mysid shrimp (Mysidopsis bahia)	1. Survival	0.71	28 (Life cycle)	-	U.S. EPA, 1980
	2. Reproduction (number of young per female)	0.71			

1/ Statisticaly significant decrease or deviation from control

Effects on fish physiology and histopathology: Most of the studies on endosulfan effects on fish physiology and histopathology were done in India. Rao et al. (1981) reported that nitrogen excretion and oxygen consumption in the freshwater fish (Macrognathus aculeatum) decreased on exposure to 1 ug/l endosulfan for one hour. The decrease in total nitrogen excretion indicates that endosulfan interferes with fish protein metabolism (Rao et al., 1981) or it decreases fish activity (Day, 1984). The decrease in oxygen consumption was due to a progressive inactivity of the fish terminating in death, without any convulsions or muscular exertion. Oxygen uptake and carbon dioxide release increased in fish exhibiting symptoms of hyperaction, irritation and convulsions (Rao et al., 1980). Endosulfan, like other chlorinated hydrocarbon pesticides, acts as a neurotoxin and a change in the rate of oxygen consumption is one of the earliest symptoms of poisoning.

Shafi (1980) found that a four-hour sublethal exposure of 5 mg/l of endosulfan to fish increased alkaline phosphatase activity in liver, muscle, kidney and brain of nine freshwater teleost, while lethal doses (15 mg/l) decreased it. The reverse trend was observed with acid phosphatase. Verma et al. (1981) also reported similar effects on phosphatase activity in catfish on chronic and sub-chronic endosulfan exposure (Table III-6). They observed that glycogen metabolism was affected resulting in impaired carbohydrate metabolism. Changes in energy metabolism (Dalela et al., 1978) and hematology (Gopal et al., 1982) have been observed in fish exposed to sublethal concentrations of endosulfan. Haya and Waiwood (1983) found that sublethal levels of endosulfan decreased AEC (adenylate energy charge) in the polychaetes, Nerius virens. They suggested that endosulfan must have interfered with some energy-producing metabolic pathway which appeared to be more susceptible under anoxic conditions. Sastry and Siddiqui (1982) reported that the rate of absorption of glucose by the intestines of a teleost was reduced on exposure to 0.1 and 100 mg/l endosulfan for 30 and 4 days, respectively. It was suggested that structural damage of intestinal mucosa may be responsible for this effect.

Rao et al. (1980) reported that liver tissue of fish that survived in a 96-hour endosulfan toxicity bioassay showed the following histological changes: (1) cell boundaries became indistinct, (2) cytoplasm became hyaline and less dense, (3) nuclei were vacuolated and chromatin appeared scattered with one or two large dots, and (4) cell vacuolation was indicated. These changes were thought to result from a higher level of endosulfan metabolites in liver, compared to concentrations in other organs.

Table III-6

EFFECT OF ENDOSULFAN CHRONIC AND SUBCHRONIC EXPOSURE ON NATIVE FISH OF INDIA

Test Species	Effect	Lowest Conc. Showing Effect (ug/l)	Length of Exposure (days)	Reference
Catfish (*Mystus vittatus*)	1. Acid phosphatase activity in liver	0.045	30	Verma et al., 1981
	2. Alkaline and glucose 6-phosphatase activity in gills			
Murrel (*Channa gachua*)	Energy metabolism:			
	1. ATPase activity in liver	1.74	30	Dalela et al., 1978
	2. ATPase activity in kidney gill and brain	2.13		
Catfish (*Clarias batrachus*)	Hematological changes - increase in erythrocyte count, hemoglobin content and hematocrit value	2.0	10	Gopal et al., 1982

Studies with mussels: No studies were found in the literature relating the body burden of endosulfan to its effects on mussels. However, the effects of sublethal aquatic concentrations of endosulfan on mussels were studied by Roberts (1972, 1975a and b). Oxygen consumption of Mytilus edulis decreased at the concentration of 100 ug/l. Mussels exposed to 450 ug/l endosulfan for 24 hours showed a 50 percent reduction in the attachment to a substrate (byssus formation) due to a reduction in thread production (Roberts, 1975a). At endosulfan concentrations exceeding 500 ug/l, spawning time of mussels was protracted and some individuals showed a marked delay in the onset of spawning at 1,000 ug/l endosulfan (Roberts, 1972). It was suggested that endosdulfan interferes with the production of gamones, endocrine-like compounds secreted by the gametes to facilitate fertilization.

Uptake, Metabolism, and Depuration

Endosulfan I, II and sulfate are more soluble in water (Appendix II, Table AII-1) and conversely less soluble in lipids than many other chlorinated hydrocarbon pesticides. The log octanol-water partition coefficients of the endosulfan compounds range from 3.55 to 3.66, compared to 6.4 for toxaphene (Cohen et al., 1982). As endosulfan I is readily transformed to the sulfate by living organisms, it is less likely to accumulate in the aquatic organisms than the more persistent sulfate.

Uptake: The digestive gland was the major storage site of endosulfan in mussels and other bivalves (Roberts, 1972), endosulfan uptake in these organisms may result principally from the ingestion of the pesticide sorbed on/in food and particulate matter.

Bioconcentration: Endosulfan bioconcentration data are not available for freshwater fish, although several bioconcentration studies with saltwater organisms have been reported in the literature. Endosulfan bioconcentration factors for aquatic organisms range from 26 in scallop (exposed to 100 ug/l endosulfan for 14 days) to 2,755 in striped mullet (exposed to 0.035 ug/l endosulfan for 28 days) (Table III-7).

Schimmel et al. (1977) reported an average bioconcentration factor of 2,429 for the edible portion of striped mullet, compared to 2,755 for the whole body (Table III-7). Since the maximum bioconcentration factor was observed on the last day of the uptake portion of the study (day 28), it is possible that the equilibrium between endosulfan concentrations in water and mussels may not have been attained. Nearly all of the endosulfan measured in the fish was in the form of endosulfan sulfate. In contrast to this, all the detectable endosulfan in sheephead minnow,

Table III-7

BIOCONCENTRATION OF ENDOSULFAN BY AQUATIC ORGANISMS

Test Species	Water Conc. (ug/l)	Exposure Period (Days)	Bioconcentration Factor (Whole body)	Remarks	Reference
FISH					
Striped mullet	0.035	28	2,755	Endosulfan sulfate	Schimmel et al, 1977
(Mugil cephalus)	0.32	4	1,344	90% mortality	"
Pinfish (Lagodon rhomboides)	0.15	4	1,299	50% mortality	"
Spot (Leiostomus xanthurus)	0.076	4	895	45% mortality	"
Sheephead minnow (Cyprinodon variegatus)	-	28	328	Endosulfan I & II	U.S. EPA, 1980
INVERTEBRATES					
Grass shrimp (Palaemonetes pugio)	1.75	4	245	65% mortality	Schimmel et al., 1977
Mussel	100	14	29	-	Roberts, 1975
(Mytilus edulis)	0.22	2	600	Endosulfan I	Ernst, 1977
Scallop (Chlamys opercularis)	100	14	26	-	Roberts, 1975

bioconcentration factor: 328, was that of isomers I and II (EPA, 1980). It appears that mullet can metabolize endosulfan whereas minnows are unable to do so. After two days in an endosulfan-free environment, no endosulfan sulfate was detected in the exposed mullet (Schimmel et al., 1977).

Roberts (1972) found that mussels assimilated more endosulfan at higher exposure levels (Figure III-1), but the concentration factors were highest at the lowest exposure level (Table III-8). Mussels exposed to 100 ug/l endosulfan for 70 days concentrated the pesticide to a maximum of 22.5 times the exposure concentration. It is not known whether endosulfan sulfate, the more persistent metabolite, was also included in the analysis.

Ali (1978) studied the fate of endosulfan I, II and sulfate in separate terrestrial-aquatic microcosm experiments. The higher concentrations of endosulfan in snail may have resulted from both bioconcentration (i.e., from water) as well as bioaccumulation (i.e. from water and food) (Table III-9). Gorbach (1984) suggests that snails may differ from other organisms in being less able to degrade endosulfan rapidly with the aid of esterases, this being the reason for their higher concentration of endosulfan.

Metabolism: Endosulfan is readily metabolized in most aquatic and other organisms. Metabolic and degradation pathways and products of endosulfan in living organisms and environment are shown in Figure III-2. Endosulfan metabolites (sultate, lactone, diol, ether and hydroxy ether) have been isolated from various tissues of fish (Schoettger, 1970; Devi et al., 1981; Rao et al., 1980; Rao and Murty, 1980 and 1982). The findings of Rao et al. (1980) suggest that metabolism of endosulfan in fish occurs through different pathways and results in various end products (Table III-10). The liver and kidney ususally have the largest number and quantities of metabolites since these are the principal organs of storage and detoxification of endosulfan (Rao and Murty, 1982; Devi et al., 1981).

Endosulfan metabolites (except sulfate) are less toxic than the parent isomers (I and II). Knauf and Schulze (1973) reported that endosulfan I, II, and sulfate were all about equally toxic to the organisms studied (Table III-3). Formation of endosulfan sulfate is therefore not a detoxification process. The nonsulfur containing metabolites (i.e., diol, lactone, and ethers) had nearly equal LC50 values and were about 1,000 times greater than those for endosulfan I, II, and sulfate.

Figure III-1

ENDOSULFAN IN MUSSELS EXPOSED TO DIFFERENT CONCENTRATIONS OF THE PESTICIDE IN SEAWATER FOR 112 DAYS

(Roberts, 1972)

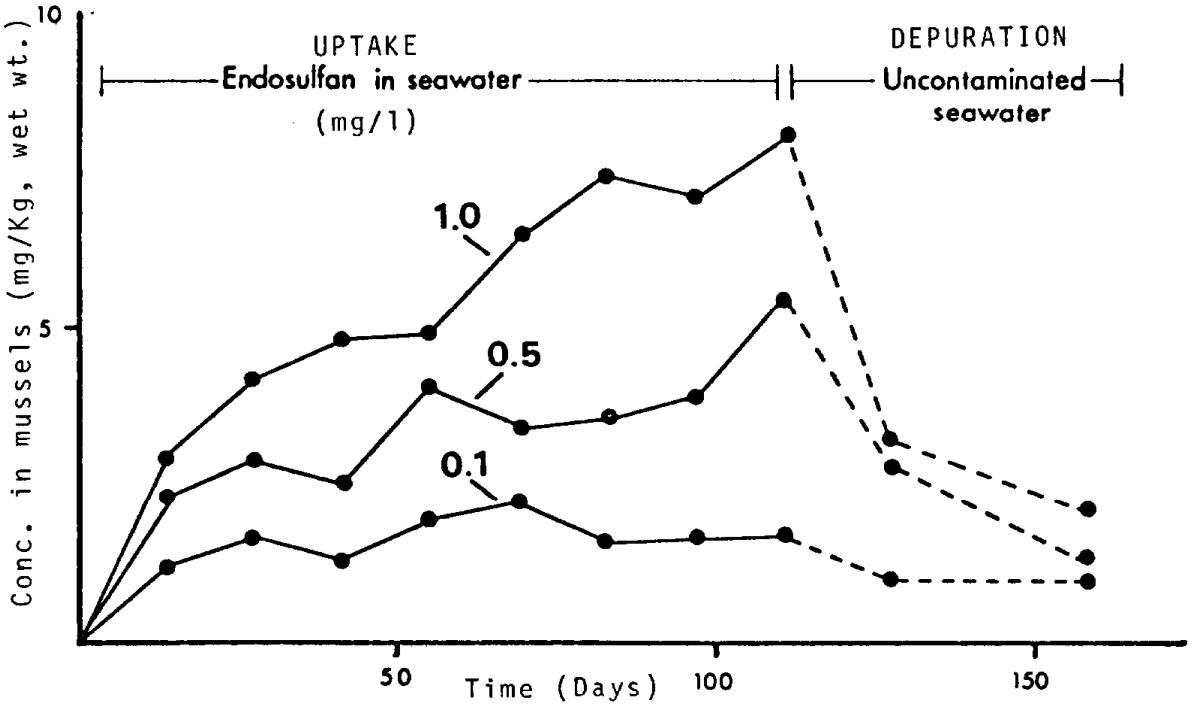


Table III-8

ENDOSULFAN BIOCONCENTRATION IN MUSSELS (*Mytilus edulis*) AS A FUNCTION OF EXPOSURE CONCENTRATION AND DURATION
(Roberts, 1972)

Length of Exposure (days)	Exposure Concentration (ug/l)		
	100	500	1,000
	(Bioconcentration Factors)		
14	13.0	4.7	2.8
42	13.5	4.9	3.7
70	22.5	6.9	6.5
112	17.0	11.0	8.1

Table III-9

MAXIMUM BIOMAGNIFICATION VALUES OF ENDOSULFAN ISOMERS BY ORGANISMS IN A TERRESTRIAL-AQUATIC MICROCOSM

(Ali, 1978)

Species	Biomagnification Factor		
	Endosulfan I	Endosulfan II	Endosulfan sulfate
Algae	999	3,863	1,654
Snail	5,763	39,457	29,430
Mosquito	831	1,508	763
Fish	304	388	1,741

Figure III-2

METABOLIC AND DEGRADATION PATHWAYS AND PRODUCTS
OF ENDOSULFAN IN BIOTA AND ENVIRONMENT

(Menzie, 1974; Knowles, 1974)

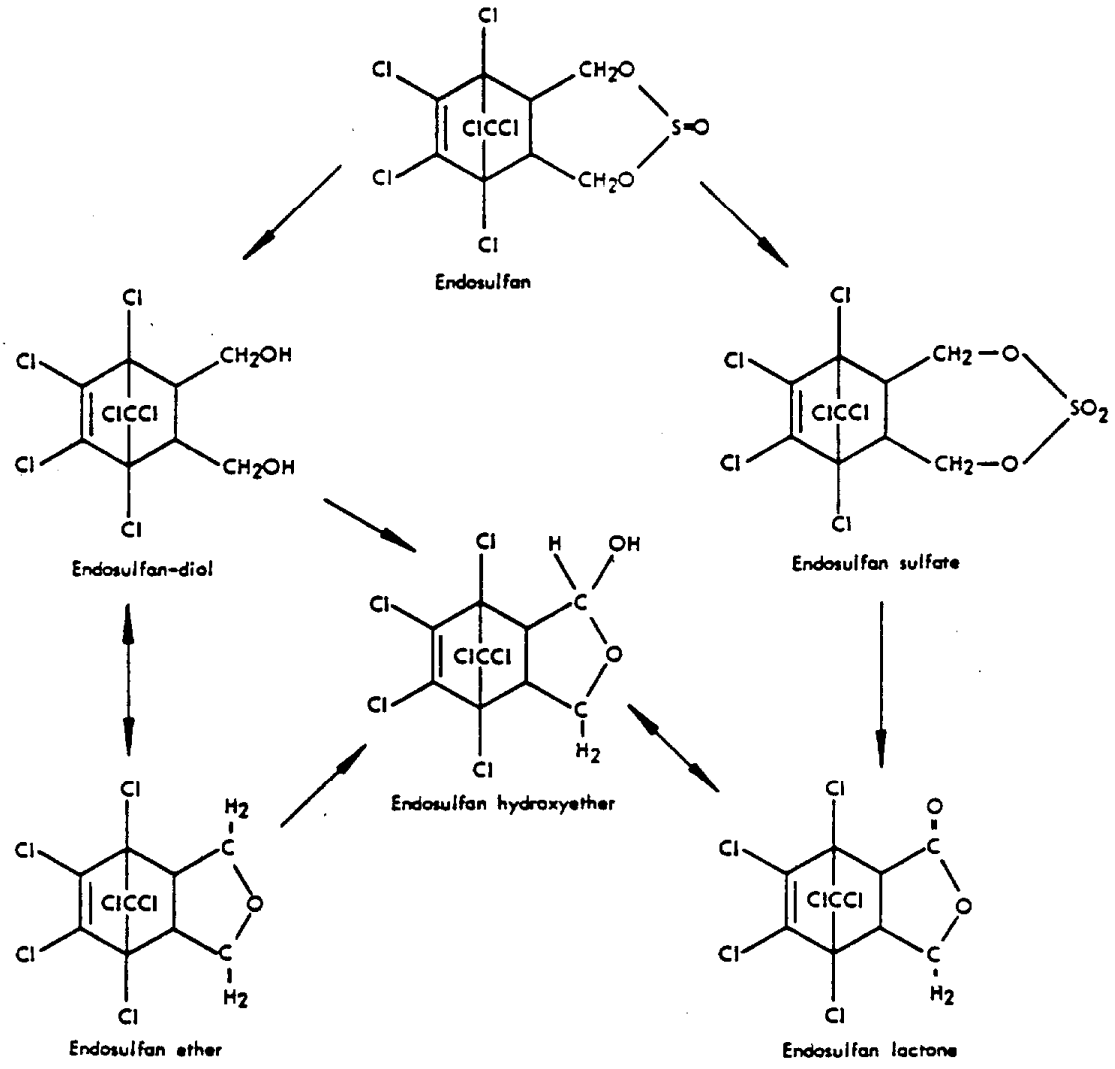


Table III-10

ENDOSULFAN AND ITS METABOLITES IN TISSUES OF FISH (Labeo rohita) EXPOSED TO 4 ug/L TECHNICAL ENDOSULFAN FOR 96HOURS*

(Rao et al., 1980)

Tissue	Endosulfan I	Endosulfan II	Endosulfan sulfate	Endosulfan lactone	Endosulfan alochol	Endosulfan alpha-hydroxy ether	Endosulfan ether
Gill	+	+	+	-	+	+	+
Muscle	+	+	+	-	-	+	+
Brain	-	-	-	-	-	-	-
Kidney	-	+	+	-	+	+	+
Liver	-	+	-	+	+	+	+
Anterior part of gut	+	+	+	-	+	-	-
Posterior part of gut	-	-	-	+	+	+	+

* A plus sign indicates presence and minus sign indicates absence of the chemical in the tissue

Elimination: Endosulfan residues can be rapidly eliminated from the body when the organism is placed in an endosulfan-free environment. As previously discussed, striped mullets which have accumulated 80 ppb of endosulfan sulfate (during a 28-day exposure to 0.035 ppb endosulfan in water) were able to depurate it to below detection limit (10 ppb) within two days (Schimmel et al., 1977). Roberts (1972) observed decline in endosulfan tissue residues when mussels were transferred to clean seawater (Figure III-1). He suggested that this may be due to excretion of the pesticide adsorbed on particulate matter in the gut. In most organisms, endosulfan metabolites are eliminated either through urine or feces, or both. Rao and Murty (1980) observed endosulfan metabolites in the bile and gut (with feces) of fish.

Toxicity to Wild Fish and Other Aquatic Organisms

It has been suggested that wild populations may be more susceptible to environmental contaminants than laboratory animals. Laboratory bioassays may therefore underestimate toxicity of chemicals to fish and wildlife (Porter et al., 1984). Most of the endosulfan aquatic toxicity studies reported in the literature were conducted in the laboratory. SWRCB monitoring programs (Chapter II) provide information on pesticide residues in field fish and other aquatic organisms which is helpful in trend analysis. However, the data cannot be used to assess the harmful effects of these residues on the aquatic organisms since most aquatic bioassays measure the concentration of toxicant only in water. Analysis of tissue concentration in aquatic tests would help to determine correlations between these concentrations and potential effects.

In a few of the numerous endosulfan related fish kill episodes reported in California (Chapter II), concentrations of the pesticide in water as well as fish tissue were measured (Table II-12). The data indicate that when endosulfan concentrations in water ranged from nondetectable to 7.9 ug/l, the tissue concentration in the dead fish were from 200 (muscle tissue) to 17,500 (gills and digestive tract) ug/kg, respectively.

Endosulfan application of 46 ug/l to a 27-acre pond resulted in death of all minnows, perch, sunfish, bullheads, and suckers within seven days (FMC, 1958). Cuerier (1960) was able to eliminate blunt nose minnows, golden shiners, common suckers, bullheads, perch, smallmouth bass and sunfish from a lake with a concentration of 15 ug/l. Frogs and aquatic insects were also

killed in both these field trials. Gopal et al. (1981) found that in static bioassays, frog tadpoles (LC50: 1.8 ug/l) were more susceptible to endosulfan than aquatic insect Enallagma sp. (LC50: 17.5 ug/l) and catfish (LC50: 14 ug/l).

The field toxicity of endosulfan to amphibians is very high (EPA, 1982). Mulla (1962) reported that very low application rates (0.1 to 0.5 lb/A) of endosulfan were "toxic" to bullfrogs. With tadpoles, moderate mortality was observed at 0.1 lb/A endosulfan II, and complete kill at 0.5 lb/A endosulfan I (Mulla, 1963).

MAMALIAN TOXICOLOGY

Though mammals are not as sensitive to endosulfan as aquatic organisms, published data indicate that acute toxicity of endosulfan to mammals is high enough for EPA to assign it the Toxicity Category "I". For example, the lowest reported endosulfan acute oral toxicity to rat (LD50) of 9 mg/kg (Reno, 1975) is about the same as that of parathion.

Acute Toxicity

Reported acute LD50 values for rodents range from 6.9 to 130 mg/kg (Table III-11). Female rats are more sensitive to endosulfan than male rats regardless of the route (oral, intraperitoneal or dermal) and vehicle (alcohol, xylene, oil) of administration. The general order of endosulfan toxicity to rats according to the route of administration appears to be oral > intraperitoneal > dermal. However, this comparison is subject to differences in the toxicological experiments, particularly in regard to the vehicle used as well as to the susceptibility of the tested rat strains (Schutz and Leist, 1984). The lowest intraperitoneal LD50 for rat of 6.1 mg/kg was reported by Lendle (1956).

Rodents differ in their sensitivity to endosulfan toxicity. For instance, Truhaut et al. (1974) reported mean oral LD50 values of 64 mg/kg for rats and 118 mg/kg for hamsters. Further, they found that the biochemical effects of endosulfan on enzyme activity differed in these animals. Serum cholinesterase activity was inhibited in hamster while in rats hepatic cholinesterase activity was inhibited.

The toxicological mode of action of endosulfan has not been completely studied. Endosulfan, like other chlorinated hydrocarbon pesticides, acts as a central nervous system poison. It produced autonomic and somatic toxicity in cat brain tissue (Anand et al., 1981). The toxic symptoms included hypertension, pupillary dilation, increase in cardiac output and cerebral blood flow (Table III-12).

Table III-11

ACUTE TOXICITY OF ENDOSULFAN TO MAMMALS

Species	Sex	Route of Administration	Carrier (solvent)	Mean LD50 (mg/kg)	Reference
Rat	M	Oral	Peanut oil	43	Gaines, 1969
		Intraperitoneal	Alcohol	46.7	Guptar, 1976
			10% alcohol in peanut oil	89.4	"
		Dermal	Xylene	130	Gaines, 1969
		Inhalation (4 hours)	- -	350 (mg/m^3)	Ely et al, 1967
	F	Oral	Peanut oil	18	Gaines, 1969
		Intraperitoneal	Alcohol	22.1	Guptar, 1976
			10% alcohol in peanut oil	48.6	"
		Dermal	Xylene	74	Gaines, 1969
		Inhalation (4 hours)	- -	80 (mg/m^3)	Ely et al, 1967
Mouse	M	Intraperitoneal	Alcohol	6.9	Gupta, 1976
	F	"	"	7.5	"
Hamster	-	Oral	- -	64	Truhaut et al., 1974
Guinea pig	-	Dermal	Cottonseed oil	>1000	Hazelton Lab., 1964
Rabbit	F	"	"	147	Hazelton Lab., 1967
	F	"	Chloroform	175	Gupta and Chandra, 1975
Dog	-	Oral	- -	1-3[1/]	Hazelton Lab., 1959a

[1/] This value was reported by Coleman and Dolinger (1978). However, according to Hoechst AG., the oral LD50 value for dog established at Hazelton Lab. is 76.7 mg/kg (Schutz and Leist, 1984).

Table III-12

ENDOSULFAN-INDUCED CLINICAL, PHYSIOLOGICAL AND PATHOLOGICAL EFFECTS IN EXPERIMENTAL ANIMALS

Species	Dose	Effect	Reference
Rat	2.5 mg/kg (oral, 7 days)	Increase in liver weight	Gupta and Gupta, 1977
	2.5 mg/kg (oral, 14 days)	Increase in lipid peroxidation and enzyme activity	Agarwal et al., 1978
	5 mg/kg (oral, 15 days)	Liver damage (dilation of sinusoids around central veins, degenerated hepatocytes and mononuclears)	Gupta and Chandra, 1977
	30 mg/kg (intraperitoneal)	Decrease in brain acetylcholinesterase activity	Gupta, 1976
	40 mg/kg (single oral dose)	Increase in blood glucose, blood ascorbic acid and blood and brain glutathione	Gary et al., 1980
Cat	0.5 mg/kg (cumulative intravenous)	Hypertension, pupillary dilation, increase in cardiac output, increase in cerebral blood flow	Anand et al., 1981
	3 mg/kg (single, intravenous)	Increase in blood glucose level	Misra et al., 1980
Rabbit	100 mg/kg (single dermal)	Degeneration of liver tissue (hepatocytes with foamy cytoplasm and bile duct proliferation); damage to kidney tubules (necrosis of proximal tubules)	Gupta and Chandra, 1975

Endosulfan does not belong to the group of pesticides which inhibit cholinesterase activity. However, Gupta (1976) reported a decrease in brain acetylcholinesterase activity in female rats which were given a dose of 30 mg/kg endosulfan (Table III-12). Truhaut et al. (1974) also noted that endosulfan inhibited hamster serum and rat hepatic cholinesterase.

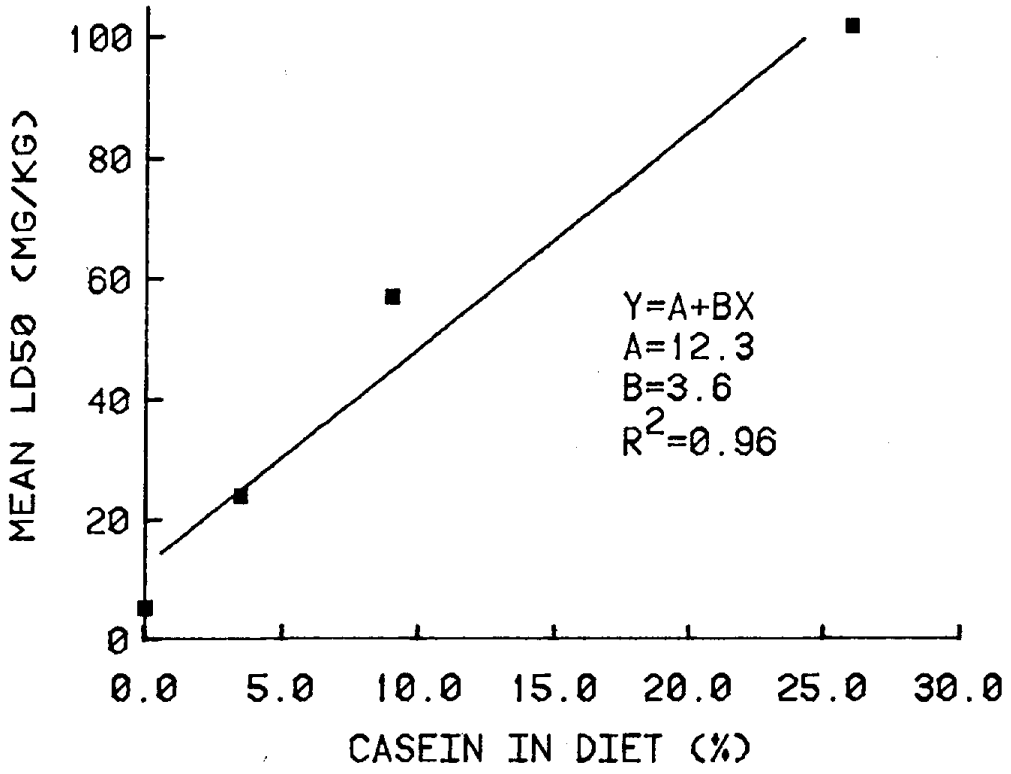
As with endosulfan aquatic toxicology, much of work on the mammalian toxicology of endosulfan was done in India. (See Table III-12 for a summary.) Endosulfan increased blood glucose levels in rats (Garg et al., 1980) and cats (Misra et al., 1981). Hyperglycemia may be a physiological response to meet the critical need of brain for increased energy in the form of glucose. Endosulfan-fed rats showed an increase in blood ascorbic acid and blood and brain glutathione (Garg et al., 1980), lipid peroxidation and enzyme activity (Agarwal et al., 1978).

Liver apparently is the organ most affected by endosulfan poisoning (Table III-12). Increases in liver weight (Agarwal et al., 1978; Gupta and Gupta, 1977) and liver damage (Gupta and Chandra, 1977) have been observed in rats. The symptoms of endosulfan toxicity in rabbits (Gupta and Chandra, 1975) are similar to those in rats and mice. Gupta and Chandra (1975) reported that a single dermal application of 100 mg/kg endosulfan to rabbits produced toxic effects in liver, kidney and adrenal tissues. No cutaneous (skin) abnormality was observed in the treated animals. Hyperexcitability, dyspnea, decreased respiration, discharge from the eyes, and tremors were followed by convulsions. The convulsions appeared at intermittent or regular intervals. The animals preferred to rest on the sternum with the forelimbs extended, and eventually lost response to painful stimuli, first in the hindlimbs, then the forelimbs, followed by loss of motility, loss of corneal reflex, a deep coma, and death (Gupta and Chandra, 1975).

Dietary protein has been reported to influence the toxicity of endosulfan in test animals. Das and Garg (1981) found that a daily dose of 0.5 ppm endosulfan in the diet was significantly more toxic to female rats receiving a low protein (5 percent) diet compared to those on a high protein (24 percent) diet. The toxic symptoms which developed exclusively in low protein-fed rats included growth retardation, low red blood cell counts, low RNA and protein levels in liver, and high glutathione levels in liver and blood. Boyd et al. (1979) reported that protein-deficient rats were four times as susceptible to endosulfan poisoning as rats having adequate protein nutrition (Figure III-3). Most toxicological tests are conducted with experimental animals having access to unlimited food

Figure III-3

EFFECT OF DIETARY PROTEIN (CASEIN) ON ENDOSULFAN TOXICITY IN RATS[1/2/]



1/ Figure developed from data of Boyd et al., 1970

2/ The highest treatment of 81 percent casein in diet gave an average LD50 of 98 mg/Kg

and water. However, non-experimental animals are subjected to changing natural conditions (e.g., need for food and water at certain times of the year) which can contribute to stress making them more sensitive to the effects of toxic chemicals.

Endosulfan sulfate is as toxic to mammals as endosulfan I and II. However, the LD50 values of non-sulfur containing metabolites (such as endosulfan alcohol, hydroxy ether and lactone) were higher in rats, and ranged from 150 to 1,500 mg/kg (Gorbach, 1972). Dorough et al. (1978) found that among the endosulfan isomers and metabolites, endosulfan sulfate was the most toxic compound to female mice (LD50: 8 mg/kg) and endosulfan diol the least toxic with an LD50 value of 2,000 mg/kg (Table III-13). As with female mice, endosulfan I was three times more toxic (oral LD50: 76 mg/kg) to rats than endosulfan II (oral LD50: 240 mg/kg) (Hoechst, 1967).

Chronic Toxicity

Very little information is available on the chronic toxicity of endosulfan, and particularly its metabolites in mammals. Table III-14 summarizes the chronic feeding experiments conducted with rats and dogs. The only study on rats by Keller (1959) suggests that male rats are more sensitive to chronic effects of endosulfan than female rats. Liver and kidney were the organs most affected. Histopathological examination of the livers of male rats fed 100 ppm endosulfan showed hydrophobic hepatic cells with pale eosinophilic cytoplasmic inclusions. The major kidney lesion manifested as renal tubule dilation, formation of albuminous cysts, focal intersititial nephritis, and degeneration of tubule epithelium. Gupta and Chandra (1977) observed similar effects in subchronic feeding experiments with rats (Table III-12).

Although endosulfan is acutely toxic to dogs (Table III-11), chronic toxicity does not appear to be a serious problem. Dogs tolerated 30 ppm endosulfan in diet for two years without any observable adverse effects (Baran, 1967). However, it is not known whether these animals were monitored after the two-year study period.

Carcinogenicity

Endosulfan carcinogenicity information has been difficult to obtain. High incidence of death among test animals precludes a definitive conclusion on the carcinogenic potential of endosulfan.

In one of two cancer bioassays (Kotin et al., 1968; Innes et al., 1969), male and female mice (Strains C57Bl/6 and C3H/AnfFl) were administered a 96 percent pure mixture of

Table III-13

LETHAL DOSE OF ENDOSULFAN AND
ITS METABOLITES TO FEMALE MICE
(Dorough et al., 1978)

COMPOUND	LETHAL DOSE (mg/kg)
Endosulfan I	11
Endosulfan II	36
Endosulfan sulfate	8
Endosulfan lactone	120
Endosulfan alpha-hydroxy ether	120
Endosulfan ether	270
Endosulfan diol	>2000

Table III-14

CHRONIC TOXICITY OF ENDOSULFAN TO MAMMALS

Species	Strain	Sex	Dose	Effect	Reference
Rats	Wistar	F	100 ppm in diet for two years	Decrease in survival rate	Keller, 1959
		M	"	Slight to moderate growth depression; increase in absolute and relative weights of kidney; liver and kidney damage	
Dogs	Beagle	M/F	30 ppm in diet for two years	No gross, clinical, hematological and histopathological effects	Baran, 1967
Dogs	Mongrel	- -	0.75 mg/kg/day in gelatin capsules, six days a week for one year	No significant gross and histopathological changes	Keller, 1959

the endosulfan isomers for nearly 18 months at levels of 3 and 6 mg/kg of feed. Although tumors were observed in both sexes of mice (Figure III-4), Innes et al. (1969) concluded that statistical analyses showed no evidence of endosulfan carcinogenicity. In a second bioassay (NCI, 1978), 50 Osborne-Mendel rats and 50 B6C3F1 mice of each sex were given technical grade endosulfan (98.8 percent purity) dissolved in corn oil and mixed with the feed for 78 weeks (Table III-15). Mice were observed for 14 additional weeks, and female and control male rats for 33 additional weeks. However, with endosulfan-fed male rats, the observations were terminated early; week 82 for high dose (952 mg/kg) and week 74 for low dose (408 mg/kg). The doses of endosulfan (2 to 952 mg/kg, time weighted average concentration in diets) used in this NCI study were toxic to the kidney of rats of both sexes and to male mice. Male rats also had testicular atrophy, and high early deaths were recorded in both species of male mice. Due to these early deaths, the bioassay was not conclusive with regard to males. However, enough females survived for the authors of this study to conclude that technical grade endosulfan is not a carcinogen to female B6C3F1 mice or to female Osborne-Mendel rats. Several lesions (nephropathy, parathyroid hyperplasia, and testicular atrophy) were observed in early male rat mortalities with no evident dose-response pattern.

These carcinogenicity studies do not meet the current EPA's requirements for oncogenic evaluation because of the route of exposure, strain of the test animals, and the model used, and the early mortality observed in these experiments. The agency has asked the registrants of endosulfan to conduct additional cancer tests with both rats and mice.

Teratogenicity/Reproductive Effects

A review and audit by EPA of some of the endosulfan teratogenicity studies (IBT, 1965; Raltech Sci. Serv., 1981; Haley, 1972) showed that the raw data do not support the conclusions drawn from these studies. For instance, in a three-generation reproduction study with rats (IBT, 1965), several discrepancies were found; five unreported rats died and were replaced during the pre-mating period, and certain pathology and histopathology data were not available. The auditor revised the data to reflect the discrepancies and found a possible kidney effect and a possible body weight effect at the high dose level (50 ppm) (DFA, 1982).

According to DFA (1982), an adverse effects disclosure was submitted concerning a teratology study with rats. There was a significant increase in small fourth and unossified fifth sternabrae at the high dose (6 mg/kg) and in

Figure III-4

ENDOSULFAN-INDUCED TUMORS IN MICE
(Kotin et al, 1968)

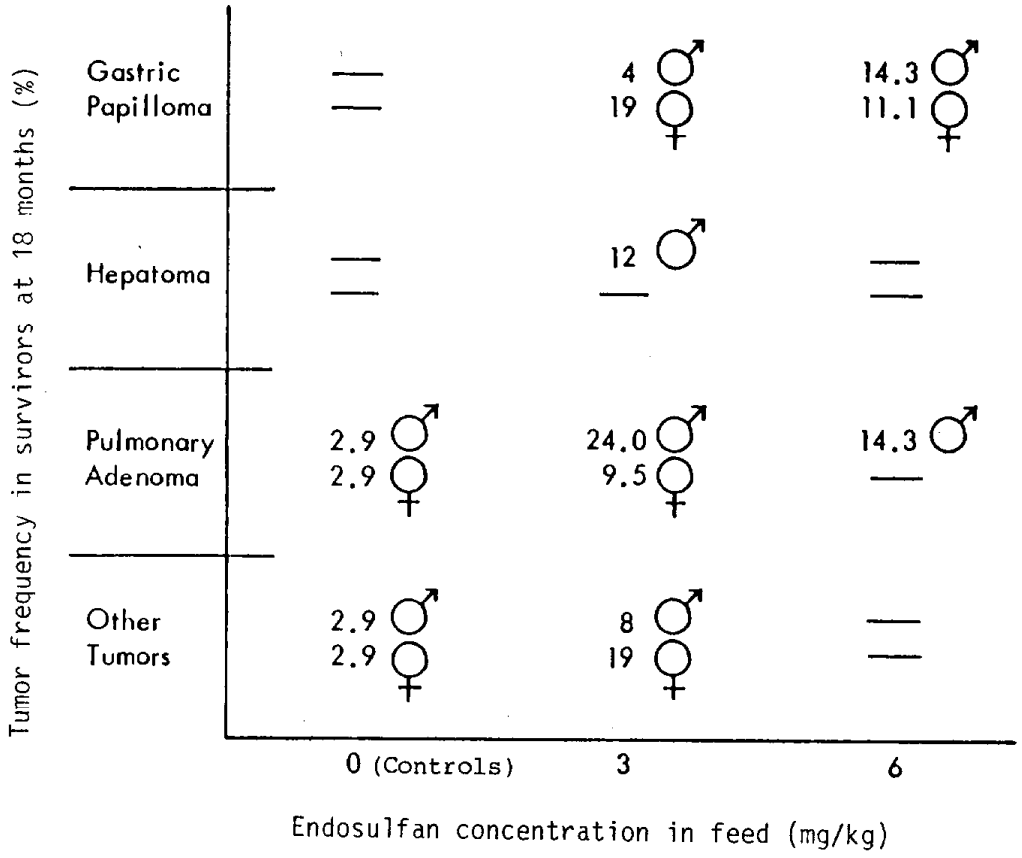


Table III-15

ENDOSULFAN-INDUCED TUMORS AND MORTALITY IN RATS AND MICE[1/]
(NCI, 1978)

Species (Strain)	Sex	DOSE (Time-Weighted Avg. Conc., mg/kg)	FREQUENCY OF TUMOR INDUCTION					
			Lung	Lymphomas/ Leukemias	Kidney	Liver	Endocrine	All Other Sites
Rats (Osborne-Mendel)	M	952	0/47	1/47	2/47	0/47	0/47	0/47
		408	0/50	2/50	3/50	0/50	1/50	4/50
		0	1/20	4/20	2/20	0/20	7/20	0/20
	F	445	1/50	1/50	3/50	1/50	11/50	15/50
		223	1/50	3/50	2/50	1/50	19/50	27/50
		0	0/20	1/20	1/20	0/20	13/20	14/20
Mice (B6C3F1)	M	6.9	2/50	0/50	0/50	2/50	0/50	6/50
		3.5	2/49	0/49	0/49	6/49	0/45	1/49
		0	0/20	0/20	0/20	1/20	0/20	3/20
	F	3.9	0/50	6/50	0/50	1/50	1/50	3/50
		2.0	6/50	10/50	0/50	0/50	0/50	4/50
		0	2/20	6/20	0/20	0/20	0/20	1/20

1/ Endosulfan was administered for 78 weeks. Observations were made in rats after 33 additional weeks, and in mice after 14 weeks (Trials with male rats were terminated early -- week 82 for high dose and week 74 for low dose)

misaligned sternabrae at the low (0.66 mg/kg) and medium (2 mg/kg) doses, but not the high dose. The misaligned sternabrae was therefore not dose related. The abnormalities at the high dose might be related to maternal toxicity as manifested by decreased body weight and central nervous system stimulation (Table III-16).

Gupta et al. (1978) investigated the teratogenic and embryotoxic effects of endosulfan in rats. At the high dose (10 mg/kg), there was a significant increase in the number of litters with resorptions (Table III-16). The authors concluded that endosulfan was not teratogenic to rats. However, it produced a dose-related increase in maternal toxicity of pregnant rats, which they attributed to a possible effect on the female sex hormones. A statistically significant decrease in the weight of the testes was found on autopsy of male rats which were fed 10 ppb endosulfan in the diet for 104 weeks (Hazelton Lab., 1959). The testes showed no histopathological damage.

In a teratogenic study with pregnant rabbits (Raltech Sci. Serv., 1982), maternal toxicity was evident in the animals fed 1.8 mg/kg (Table III-16). However, no significant differences were observed in the mean number of corbora lutea, implantation efficiency, litter size, sex ratio, mean fetal length and weight, or in the number and percent of live and resorbed fetuses.

The studies with chicken eggs listed in Table III-16 suggest that egg hatchability is affected by endosulfan. However, these studies (Dunachie and Fletcher, 1966 and 1969; Smith et al., 1970) are not relevant for evaluating the effects of endosulfan on the reproductive system of birds, since the route of application (injection) is artificial.

Lutz-Ostertage and Kantelip (1971) reported that exposure of excised chicken and quail gonads to endosulfan-containing culture media altered morphology of the reproductive organs. Although the hatchability of chick and quail eggs after endosulfan treatment (dose not mentioned) was normal, a high proportion of the resultant birds was sterile. The authors suggested that the sterility may be due to the antimitotic toxicity of endosulfan.

Mutagenicity

To support registration of a pesticide, EPA requires a battery of valid mutagenicity tests which determine the potency of the chemical to induce point mutations and chromosomal mutations either directly or indirectly. Since

Table III-16

TERATOGENIC, EMBRYOTOXIC AND REPRODUCTIVE EFFECTS OF ENDOSULFAN

SPECIES	DOSE	EFFECT	REFERENCE
Rat	6 mg/kg	Skeletal, visceral, external anomalies; insignificant reduction in size and weight of fetuses	Raltech Sci. Serv., 1981
	5 and 10 mg/kg in diet from day 6 through day 14 of gestation	Significant increase in fetal mortality and resorption sites	Gupta et al., 1978
	10 ppm in diet for 104 weeks	Statistically significant decrease in weight of testes	Hazelton Lab., 1959
Rabbits	0.3, 0.7 and 1.8 mg/kg/day on days 6 to 28 of gestation	Maternal toxicity at 1.8 mg/kg treatment; no significant differences in number and percent of live and resorbed fetuses	Raltech Sci. Serv., 1982
Chicken	1.5 mg/egg yolk injection	Hatchability 77.3 percent (80 percent hatchability in control	Smith et al., 1970
	5 mg/egg injection	Hatchability 60 percent	Dunachie and Fletcher, 1966
	10 to 500 mg/kg acetone injected at the center of the egg yolk	Hatchability 54 percent at 100 mg/kg treatment	Dunachie and Fletcher, 1969
	10 to 500 mg/kg in corn oil injected at the center of the egg yolk	Hatchability 24 percent at 100 mg/kg treatment	"

most of the studies available on endosulfan do not adequately define the mutagenic potential of the chemical, EPA has asked the registrant to conduct additional testing.

All but one of the mutagenicity studies listed in Table III-17 show that endosulfan is not mutagenic. Adams (1978) found that endosulfan (concentration, purity and other details not reported) was positive as a base-pair substitution mutagen in Salmonella typhimurium. Dorough et al. (1978) reported that endosulfan I, II, sulfate, and ether were not mutagenic in Ames bioassay with S. typhimurium. However, other metabolites (endosulfan diol, alphahydroxy ether, and lactone) severely inhibited bacterial growth at 10 mg per plate. Mutagenicity test results for these metabolites are, therefore, inconclusive.

Grover and Tyagi (1980) studied the cytological effects of endosulfan and other common pesticides in barley by soaking the seeds in pesticide solutions (0.025 to 0.1 percent) for two and four hours. Chromosomal abberations were observed in root tip cells at metaphase, anaphase and telophase at all concentrations. The authors suggested that endosulfan and the other pesticides are quite effective in producing abberant cells even at doses which are considerably less than those recommended for field use.

Uptake, Metabolism and Elimination

Very few studies have been reported on the uptake, metabolism and elimination of endosulfan in mammals.

Uptake: Endosulfan uptake in mammalian systems depends on the carrier or solvent used with it. Undiluted endosulfan is slowly and incompletely absorbed in the mammalian gastrointestinal tract (Maier-Bode, 1968). However, when it is dissolved in a carrier such as cotton seed oil, it is readily, though not completely, absorbed by rats (Boyd and Dobsos, 1969) and other animals (Maier-Bode, 1968).

Alcohols, oils, and emulsifiers also accelerate the dermal absorption of endosulfan. Gupta and Chandra (1975) reported that endosulfan is absorbed readily when it is dissolved in chloroform and painted on the shaven skin of rabbits. Demeter et al. (1977) found that absorption of endosulfan II was faster than endosulfan I.

Metabolism: Endosulfan metabolism in mammals has been adequately delineated. The generalized metabolic pathway for endosulfan in animals is shown in Figure III-2. Matsumura (1975) reported a slightly different pathway in which endosulfan alpha-hydroxy ether was formed directly from either the diol or the lactone, without the ether intermediate. It has been reported that after ingestion, endosulfan is first distributed to the liver and then to

MUTAGENICITY STUDIES WITH ENDOSULFAN

Test	Response	Comments	Reference
Mouse dominant lethal	-	IBT study	Arnold, 1972
Mouse micronucleus	-	Endosulfan treated mice had almost twice the number of polychromatic erythrocytes with micronuclei (0.52%) as compared to the control mice (0.28%). This increase was reportedly insignificant ($p>0.05$)	Rani et al., 1980
Cytogenetic assay of somatic and germinal cells in male rats	-	Chromatid breaks were observed in bone marrow cells but not in spermatogonial cells	Dikshit and Datta, 1978
Salmonella typhimurium base-pair substitution	+	Without microsomal activation	Adams, 1978
Ames test with *S. typhimurium*	-	With or without the S-9 liver homogenate	Dorough et al., 1978
E. coli forward mutation to streptomycin resistance	-		Fahrig, 1974
Yeast *Saccharomyces cerevisiae* mitotic gene conversion	-		"
Yeast *Serratia marcescens* back mutation to prototrophy	-		"

the brain, heart, kidneys, lungs, spleen, testes, thymus gland, suprarenal glands, mammary glands, skeletal muscles, and the remainder of the gastrointestinal tract (Boyd and Dobos, 1969; Maier-Bode, 1968).

Deema et al. (1966) studied the metabolism of ^{14}C-ring labeled endosulfan (58.3 percent endosulfan I, 35.6 percent endosulfan II, 6 percent ether, and 10 percent alcohol) in mice. Mice were fed 0.2 to 0.3 mg ^{14}C-endosulfan in a 300 mg diet, and after 24 hours the amount of radioactivity was determined in 1 g of an organ or excreta. The relative amounts of radioactivity were in the following sequence: Feces (98,452 counts per minute or cpm) > visceral fat (7,053 cpm) > urine (3,746 cpm) > liver (2,883 cpm) > small intestine and contents (2,080 cpm) > kidney (1,390 cpm) > brain (424 cpm) > blood (92 cpm). Respired air had some radioactivity (302 cpm), which indicates that animals are capable of metabolizing the cyclodiene ring, ultimately converting it to CO_2. Total recovery of the labeled endosulfan was about 66 percent, the remainder was unaccounted for. Under the conditions of this study, the principal metabolic products produced in the mouse were endosulfan sulfate and endosulfan alcohol.

Dorough et al. (1978) fed 5 ppm of ^{14}C-endosulfan isomers I or II in a diet to female rats for 14 days. Table III-18 gives the residue data of the isomers in rat tissues at various time intervals. The distribution of radioactivity did not vary significantly with endosulfan I and II. The highest residues were detected in kidneys where a steady increase occurred during the feeding period, and reached a maximum of about 3 ppm after 14 days. Another group of rats was fed 5 mg/kg endosulfan metabolites (the sulfate, diol, ether, alpha-hydroxyether, and lactone) for 14 days. The organs containing the greatest amounts of endosulfan metabolites were the liver (3 ug/g) and kidneys (1 ug/g).

Elimination: Endosulfan and its metabolites are almost completely eliminated from mammalian systems through feces and urine. Feces must be considered the principal route of elimination since the excreta has the highest residues (Deema et al., 1966; Dorough et al., 1978; Gorbach et al., 1968). Dorough et al. (1978) reported that fecal and urinary excretion of radiocarbon from rats given a single oral dose of ^{14}C-endosulfan I or II (2 mg/kg in corn oil) accounted for 88 and 87 percent of the administered doses after 120 hours (Table III-19), respectively. Biliary excretion was 47.2 percent of the single endosulfan I dose and 28.9 percent of endosulfan II dose over a 48-hour treatment. The half-life of the residues was approximately seven days when the insecticide was removed from the diet.

Table III-18

ENDOSULFAN RESIDUES IN TISSUES OF FEMALE RATS FED 5 ppm OF ^{14}C-ENDOSULFAN I OR II IN DIET (Dorough et al., 1978)

	Parts per million of [^{14}C] endosulfan equivalents per isomer in diet									
	Kidney		Liver		Visceral fat		Subcutaneous fat		Muscle[a/]	Brain[a/]
Days	I	II	I	II	I	II	I	II	I/II	I/II
On treatment										
1	0.38	0.47	0.26	0.32	0.34	0.24	0.32	0.30	0.02	0.03
2	1.26	1.21	1.02	0.79	0.85	1.02	0.23	0.34	0.02	0.03
7	1.77	1.87	0.96	0.75	0.74	0.53	0.51	0.30	0.02	0.04
10	2.28	2.08	1.11	0.94	0.94	0.55	0.15	0.28	0.03	0.04
14	3.00	3.26	1.08	1.06	0.62	0.50	0.15	0.32	0.05	0.07
Off treatment										
1	2.75	3.34	1.00	0.87	0.45	0.42	0.02	0.08	0.05	0.05
3	1.89	2.21	0.49	0.57	0.15	0.28	ND[b/]	ND	0.02	0.06
7	1.53	1.66	0.28	0.36	ND	ND	"	"	ND	0.04
14	0.94	0.92	0.11	0.19	"	"	"	"	"	0.02

a/ The low residues in muscle and brain represent both endosulfan I and II treatments

b/ Not Detected (detection limit: 0.02 ppm)

Table III-19

ENDOSULFAN ELIMINATION FROM RATS
(Dorough et al., 1978)

Treatment and time	Cumulative percentage of dose(s)		
	Feces	Urine	Total
Single dose, 2 mg/kg			
Endosulfan I			
24 hr	11.0	7.7	18.7
48 hr	61.6 (21.9)[a/]	11.1 (12.5)	72.7
96 hr	73.0	12.5	85.5
120 hr	74.8	13.2	88.0
Endosulfan II			
24 hr	12.5	12.3	24.8
48 hr	55.1 (15.2)	16.0 (10.4)	71.1
96 hr	66.5	17.7	84.2
120 hr	68.3	18.5	86.8
Dietary supplement			
Endosulfan I, 5 ppm			
14 days on	56.5	7.8	64.3
+14 days off	63.1	9.2	72.3
Endosulfan II, 5 ppm			
14 days on	57.0	8.0	65.0
+14 days off	63.5	9.3	72.8

a/ Values in parentheses are for animals having the bile duct cannulated; amounts in the bile collected for 48 hr were 47.2 and 28.9% for I and II, respectively.

Feces are the principal route of endosulfan elimination in dogs and sheep also (FMC, 1963). When dogs were fed endosulfan I and II for 28 days at 0.35 and 1.75 mg/kg/day, 13 to 25 percent of the dose was detected in the feces. Urine had only traces (0.02 to 0.1 ppm) of the endosulfan isomers. Kloss et al. (1966) reported that the half-life of radiolabeled endosulfan was about two days in feces and urine of sheep given a single oral dose of ^{14}C-labeled endosulfan at 14 mg/kg.

Endosulfan and its metabolites have been detected in milk of lactating animals treated with endosulfan. Gorbach et al. (1968) found 2 ug/l of endosulfan in the milk of sheep administered a single oral dose of 0.3 mg/kg ^{14}C-labeled endosulfan 22 days before. Between 0.1 and 0.2 mg/l endosulfan sulfate was detected in the milk of cows that had been given 2.5 mg/kg each of endosulfan I and II, and 5 mg/kg of endosulfan sulfate in their feed for 30 days (FMC, 1965). Twenty days after administration of the insecticide was stopped, less than 5 ug/l endosulfan sulfate were detected in the milk. Braun and Lobb (1976) reported a half-life of 3.9 days for endosulfan in the milk of cows that survived endosulfan poisoning.

HUMAN TOXICOLOGY

Exposure Assessment

Humans can be exposed to endosulfan residues in the following ways:

1. Ingestion from water: Endosulfan residues have been detected in surface and drinking water (EPA, 1980). No drinking water guideline has been established for endosulfan. However, in 1980 EPA set an ambient water quality criterion of 74 ug/l for the protection of human health from the toxic properties of endosulfan ingested through water and contaminated aquatic organisms.

2. Ingestion from food: Endosulfan is applied to over 60 food and nonfood crops to control over 100 different insect pests (EPA, 1980). Official U. S. tolerances for endosulfan residues in raw agricultural commodities range from 0.1 to 2 mg/kg (Appendix VI). Residues of endosulfan exceeding the tolerance limit have been detected in California (Table II-16). The acceptable daily intake (ADI) for endosulfan of 7.5 ug/kg was established by the Food and Agricultural Organization (FAO, 1975). The U.S. Food and Drug Administration (FDA) reported a daily intake of 10.22 ug total endosulfan in the western United States during fiscal year 1973-74 (U.S. FDA, 1977).

An "action level" of endosulfan in food has not been established by FDA. However, the National Academy of Sciences (NAS) has recommended that for the protection of predators, endosulfan residues in whole fish should not exceed 100 ug/kg, either singly or in combination with other persistent chlorinated hydrocarbon pesticides.

3. Cigarette smoking: Endosulfan is registered for use on tobacco crops; and, consequently, residues of up to 20 mg/kg of total endosulfan have been detected in commercial tobacco lots (Domanski and Sheets, 1973). Coleman and Dolinger (1978) computer that a two pack-a-day cigarette smoker will take in 6 ug/day of endosulfan. This is based on the assumption that an average cigarette weighing 0.5 g contains 2 ppm endosulfan, and the process of smoking transfers 15 percent of this residue to the smoker. The toxicological significance of inhaled or ingested endosulfan is unknown. In 1977, the province of Ontario, Canada, suspended endosulfan use on tobacco because of the presence of high residues of the pesticide in cured tobacco leaf (Frank et al., 1979a).

4. Inhalation: In addition to smoking, this route of exposure is important in work environment situations. However, endosulfan exposure limits have not been established by either the Occupational Safety and Health Administration (OSHA) or the National Institute for Occupational Safety and Health (NIOSH). Wolfe et al. (1972) evaluated the respiratory exposure of endosulfan to sprayers during application of the pesticide (0.08 percent solution) to orchards with tractor-drawn power air-blast equipment. The respiratory exposure was estimated to range from 0.01 to 0.05 mg/hour. Oudbier et al. (1974) found that workers are more sensitive to endosulfan exposure during mixing operations than during spraying. Over 180 ug of endosulfan were detected on the respirator pad during a five-minute mixing operation, whereas only 4.6 ug were found during a 30-minute spray operation. The American Council of Governmental and Industrial Hygienists (ACGIH, 1977) established a threshold limit value-time weighted average (TLV-TWA) for endosulfan of 0.1 mg/m3.

5. Dermal: This route of exposure is also significant for workers. Wolfe et al. (1972) estimated an endosulfan dermal exposure of 0.6 to 95.3 mg/hour to spraymen applying a 0.08 percent endosulfan solution. Kazen et al. (1974) found that endosulfan persisted on exposed worker's hands for as long as 112 days after exposure.

Toxicity

A small dose of endosulfan may be fatal. Hayes (1982) reported that one person died after swallowing only drops of a formulation. Worker-use experiences and accidental or intentional poisoning cases provide information on endosulfan toxicity in humans. Endosulfan acts on the central nervous system of humans resulting in convulsions and alterations in EEG (electroencephalogram) patterns (Tiberim et al., 1970).

Work-related cases: Ely et al. (1967) reported that nine workers suffered one or more convulsions following exposure to a 50 percent endosulfan powder. In one instance, a convulsion was followed by unconsciousness which lasted for an hour, and the fit was so violent that it resulted in fractures of the fourth and fifth dorsal vertebrae. Israeli et al. (1969) reported three cases of endosulfan toxicity in workers exposed to endosulfan in a factory. The symptoms appeared rapidly, within one to two hours in the lethal cases, and initially included headache, restlessness, and increased irritability, followed by vertigo, stupor, disorientation, and epileptiform convulsive seizures. Medical control at frequent intervals following anti-convulsant medication and discontinuation of exposure to endosulfan resulted in complete clinical recovery and cessation of seizures in all patients (Tiberin et al., 1970).

Accidental and intentional poisoning: Two cases of poisoning were reported by Demeter and Heyndrickx (1978). Both involved 20 percent endosulfan and alcohol (liquor), and resulted in death. Table III-20 gives the distribution of endosulfan and alcohol content in the tissue of the victims. Alcohol can increase the gastro-intestinal absorption of endosulfan, and, therefore, can act as a synergist.

Coutselinis et al. (1978) analyzed blood and viscera of three persons who died after an intentional ingestion of a 35 percent emulsifiable concentrate formulation of endosulfan. The average concentration of both endosulfan isomers ranged from 0.28 mg/kg in brain tissues to 6.3 mg/l in blood.

Circulatory disorders, protein dystrophy in the parenchymal organs, acute lung emphysema, and severe changes in the neurons were the most significant post-mortem findings described by Terziev et al. (1974) in five human deaths due to endosulfan poisoning (two accidental and three intentional).

Table III-20

ENDOSULFAN (I AND II) AND ALCOHOL LEVELS IN TISSUES, BLOOD AND URINE OF TWO HUMAN VICTIMS (Demeter and Heyndrickx,1978)

Organ/Tissue	Endosulfan Level	Alcohol Level (mg/l)
	mg/kg	
Small intestine	314	
	289	
Kidney	11.4	
	4.28	
Brain	4.1	
	NR[a]	
	mg/l	
Blood	<0.1	2.34
	0.075	1.81
Urine	<0.1	3.46
	2.65	2.47

a/ Not Reported.

The risk of endosulfan accidental poisoning is mainly limited to workers handling the emulsifiable concentrate during the loading and mixing operations and preparation of the end-use product (Schutz and Leist, 1984). The World Health Organization (WHO) has suggested that metabolic studies in man, with particular reference to storage of endosulfan and its metabolites should be investigated (Vettorazzi, 1979).

IV. RISK MANAGEMENT

According to Stewart and Cairns (1974), the half-life of endosulfan in soil ranges from a few months (endosulfan I) to over two years (endosulfan II and sulfate). This persistence, along with its potential for runoff and drift, and acute toxicity to fish and other aquatic life at extremely low levels are reasons for endosulfan having caused the greatest number of pesticide-related fish kills in California. According to DFG staff (Day, 1984), the fish kills were caused by acute exposure to endosulfan, usually from agricultural return water runoff or drift. Endosulfan's relative persistence (as opposed to organophosphates and carbamates) has caused the material to be found in aquatic organisms, although its chronic impacts have yet to be determined.

Nonpoint Source Discharges

Many of the crops covered under the existing endosulfan registrations are grown near or adjacent to water bodies containing valuable fisheries resources, which are exposed to contamination via runoff, soil erosion, and drift. A typical endosulfan product label (Appendix VII) states that: "This product is toxic to fish and wildlife. Keep out of lakes, ponds and streams. Do not apply when weather conditions favor drift from the areas treated. Do not apply when run-off is likely to occur. Do not contaminate water by cleaning of equipment, or disposal of wastes or containers." Fish and aquatic invertebrate kill reports suggest that levels of endosulfan resulting in acute mortality are observed even after use on some field crops has been restricted to label recommendations (EPA, 1982). To mitigate environmental contamination, the label language needs to be expanded. This subject is dealt with in more details in the "Recommendations" section.

Runoff: Most fish kills result from runoff or drainage of irrigation water from endosulfan-treated fields. A 1966 U.C. Cooperative Extension bulletin (Appendix VIII) stated that farmers should be warned about letting water from endosulfan treated fields drain into canals or ditches where fish may be present. The recommendations made in the bulletin to reduce the possibility of fish kills go beyond the recommendations on the label of endosulfan products, and include: (1) irrigation of the field before endosulfan application, when possible; (2) irrigation of the field following a waiting period of three to five days after endosulfan application; and (3) keeping runoff water from the treated field to a minimum.

Many fish kills have occurred since these management practices were first recommended in 1966. Additional use restrictions by County Agricultural Commissioners are therefore necessary.

The Federal Water Pollution Control Act (Section 208) requires states to identify nonpoint sources of pollution, including runoff from agricultural fields, and to develop plans for their control. Agricultural Best Managment Practices (BMPs) relevant to control of endosulfan discharges include soil and water conservation to minimize tailwater runoff and soil loss. Discharges of endosulfan adsorbed in sediment could be reduced by installing sediment traps below treated watershed areas. According to DFG staff (Day, 1984), runoff water from treated fields should not be allowed to be discharged if endosulfan is present.

Drift: Aerial application of endosulfan can potentially result in drift of the pesticide and subsequent redeposition on water bodies. In California during 1981 aerial application of endosulfan accounted for 66 percent of the total amount used in the state. However, in some counties, such as Imperial, as high as 93 percent of the insecticide was applied aerially. Drift losses can be minimized by following ground application whenever possible. If aerial spray is unavoidable, certain best management practices, i.e., optimal buffer strip, weather conditions, and spray nozzle sizes, should be adopted to minimize the drift.

Integrated Pest Management (IPM)

An integrated pest management program which includes non-chemical or safer chemical alternatives should be implemented to mitigate the impacts of endosulfan on aquatic organisms. Biological control agents have a great potential for the control of insect pests. For instance, Bari (1983) is exploring the potential of the bacterium Bacillus thuringiensis and entomogenous nematode (Neoaplectana arpocapsae Weiser) for use on a commercial scale to control the artichoke plume moth in Monterey County. Bari and Kaya (1984) have successfully demonstrated use of these two biological control agents in field trials. This research is of great significance considering (1) the high rate of endosulfan applied on artichokes (five to six applictions per year) in Monterey, where endosulfan use is high; (2) high residues of endosulfan detected in mussels and fish collected from this area; and (3) endosulfan's extreme toxicity to aquatic life.

Ryder et al. (1983) recommend the following approaches to reduce endosulfan and other pesticide use and residues on artichokes:

Scouting: The egg-laying activity of the artichoke moth should be monitored and fields should be treated only when egg density reaches levels of one egg per 100 leaves during winter and three eggs per 100 leaves in other seasons. This will keep the pest damage within tolerable limits and significantly reduce insecticidal use.

Pheromones: Commercially available female sex pheromone ((z)-11-hexadecenal) could be placed in plastic-laminated dispensers in the field. This disrupts the mating of adult artichoke plume moths and results in an acceptable level of pest control while reducing the insecticide use by 85 percent.

Mass trapping: Acceptable pest control could be achieved by the use of mass trapping along with half the usual amount of insecticide.

Other areas of research that are being investigated by Ryder et al. (1983) include the development of plume-moth resistent cultivars, and insect-growth regulators. For instance, diflubenzuron (Dimilin) is an insect growth regulator which acts by interfering with deposition of insect chitin. It has been effectively used to control cotton boll weevils in conjunction with trap-cropping techniques (Burris, 1984). According to Bari (1984), if Dimilin is registered for use in California on artichokes to control the plume moth, endosulfan use would decrease substantially.

The DFA staff have indicated that all these IPM programs, except monitoring (scouting), are still in experimental stage. Implementation of these programs are "many years down the road" and some, such as mass trapping, are not considered very feasible (Loughner, 1984).

Nevertheless, all current information on runoff, soil erosion, drift, and IPM must be evaluated by the Department of Food and Agriculture and the U.C. Extension Services, and revised recommendations should be prepared to ensure that no additional fish kills occur in California from the use of endosulfan.

Point Source Discharges

Endosulfan point source discharges can occur from manufacturers, formulators, and applicators. In California, endosulfan is formulated at J.R. Simplot (formerly Occidential chemical, Lathrop) and Food, Machinery and Chemical (FMC) Corporation in Fresno. EPA has proposed a zero discharge of endosulfan to surface waters from formulators/packagers facilities (Appendix VI). However, there are no regulations for discharges to ground water.

Up to 100 ppb of endosulfan were found in a monitoring well at a pesticide-manufacturing and formulating facility in Contra Costa County, California (Table II-14). The Central Valley Regional Water Quality Control Board (Region 5) has identified 400 pesticide applicator sites in the Central Valley. A survey by the Regional Board revealed that some practices for handling pesticides were not adequate to protect water quality, and that ground and surface waters at sites within the Central Valley may be threatened. To mitigate the water quality contamination potential, the Regional Board amended its water quality control plan to include a "Pesticide Rinse Water Management Program". Following are some of the guidelines listed in this program:

(i) Prohibition of discharge of diluted pesticide rinse water to any surface or ground water.

(ii) Prohibition of disposal of pesticide rinse water runoff where liquids and/or erosion of contaminated soils to surface waters is likely to occur.

(iii) Facilities developed for handling pesticide rinse waters shall not allow percolation to underlying soils and ground waters. This may be accomplished by lining of soil evaporation ponds with impermeable materials and/or providing documented tests by a registered engineer that the permeability of the storage area is 1×10^{-6} cm/sec or less.

(iv) Ultimate disposal of concentrated rinse waters and pesticide contaminated soils must take place at a Class I disposal site or an appropriate site approved by the Regional Board.

Petroleum base solvents (used as diluents in emulsifiable concentrate formulations of pesticides), when mixed with water, greatly decrease the normal evaporation rate of pesticides from disposal ponds. Some applicators tried to spray the rinsewater above the evaporation basins in order to increase the rate of pesticide evaporation. However, these attempts were unsuccessful and resulted in problems of odor and crop injury downwind of the spray area. (RWQCB 7, 1982).

Endosulfan residues (up to 2,300 ug/l) have been detected in washwater discharges from pesticide applicator sites. Jones and Van Voris (1980) studied the typical rinsewater disposal practices followed by pesticide applicators in Monterey and Santa Cruz counties (Table IV-1). They concluded that although pesticide rinsewaters were handled more carefully than fertilizer rinsewaters, disposal practices of both were typically inadequate to fully protect ground and surface water quality.

TABLE IV-1
SOME PESTICIDE RINSEWATER DISPOSAL PRACTICES
IN MONTEREY AND SANTA CRUZ COUNTIES
(Jones and Van Voris, 1980)

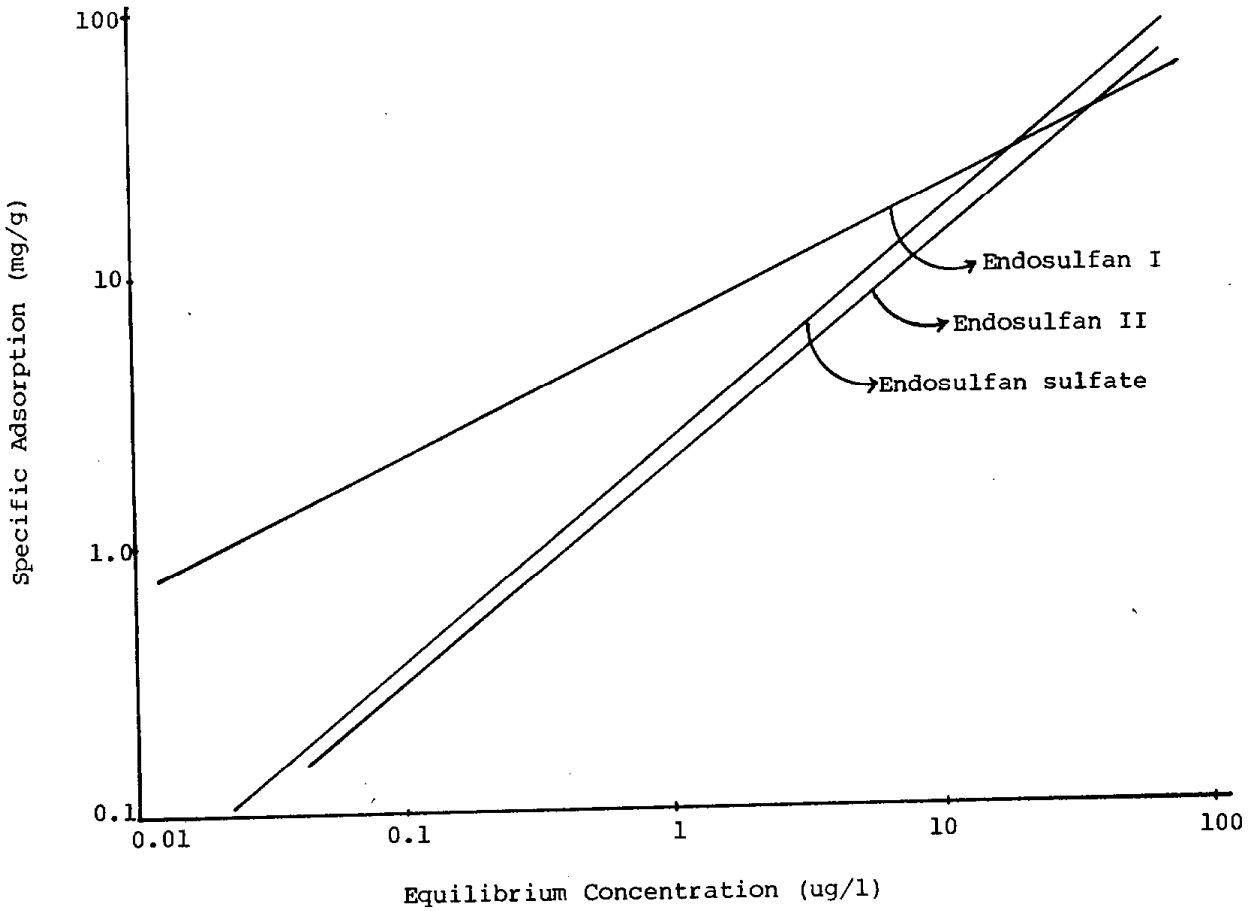
Name of Firm	Location	Method of Disposal
PureGro	Salinas	Discharges to City sewer
Western Farm Service	"	Holding tank to Class I site
Budsco	"	Discharges to City sewer
Soil Serv	"	Discharges to City sewer
Wilbur Ellis	"	Holding tank to Class I site
Crop Flight1/	"	Holding tank to Class I site
Pajaro Valley Aviation1/	Watsonville	Unlined ditch - drainage to Pajaro River
Western Farm Service	"	Discharges to City sewer - overflows to storm drain to Watsonville Slough
Moyer Chemicals	"	Holding tank to Class I site
PureGro	"	No Class I rinsing at this facility
Crop Flight1/	Greenfield	No Class I rinse at this facility
Western Farm Service	"	Holding tank to Class I site
Soil Serv	King City	Evaporation platform to lined sump to Class I site
Soil Serv Aerial1/	"	Evaporation platform to lined sump to Class I site
Soil Serv	Soledad	Evaporation platform to lined sump to Class I site
Soil Serv Aerial1/	"	No Class I rinse at this facility
Gomes Air Service1/	Castroville	Unlined ditch - to old Salinas River Channel
Castle Chemical	Pajaro	No rinsate at this facility

1/ Aerial Applicators

Carbon adsorption technology can be used to reduce endosulfan concentration in point source discharges. Adsorption isotherms of endosulfan I, II, and sulfate on activated carbon are shown in Figure IV-1. The affinity of these chemicals for adsorption is, in order, endosulfan sulfate > endosulfan II > endosulfan I. The adsorption capacity of activated carbon ranges from 194 mg/g for endosulfan I to 686 mg/g for endosulfan sulfate, when the initial concentration of these chemicals in water is 1 mg/l (Dobbs and Cohen, 1980).

Figure IV-1

ADSORPTION ISOTHERMS OF ENDOSULFAN I, II, AND SULFATE ON ACTIVATED CARBON[1/]



1/ Adapted from Dobbs and Cohen, 1980.

APPENDICES

APPENDIX I

DATA GAPS

Endosulfan and other pesticides such as DDT were registered for use years before the new stringent requirements for pesticide registration were enacted by EPA. Since these old pesticides were being sold and used without the same assurances of human and environmental safety as were being required for new products, Congress directed EPA in 1979 to re-register all previously registered pesticides.

In compliance with this Congressional order, EPA in April 1982 issued the "Pesticide Registration Standard" for endosulfan (EPA, 1982). This document sumarizes all the data available to the agency to support the registration of a pesticide, and lists all data gaps which have to be filled by the registrants. In the case of endosulfan, EPA found that much of the information on toxicology and environmental fate was invalid and not useful for registration. Important data gaps (as of April, 1982) identified by EPA are listed in Table AI-1. EPA has asked the registrants of endosulfan to conduct additional studies in order to fill these data gaps within a specified time (eight months for simple tests such as hydrolysis and photodecomposition to 50 months for complex toxicity tests such as carcinogenicity and chronic feeding).

State Board staff asked EPA to provide an update on the status of these tests, since this information was not available from the California Department of Food and Agriculture. According to EPA one-year extensions have been granted to complete tests on acute delayed neurotoxicity, subchronic oral toxicity, 21-day subchronic dermal toxicity, subchronic inhalation toxicity, and avian reproduction (La Rocca, 1984). Table AI-2 gives the data call-in status (as of March, 1984) for some important toxicological and environmental fate studies.

Table AI-1

SOME IMPORTANT ENDOSULFAN DATA GAPS
IDENTIFIED BY EPA
(U. S. EPA, 1982)

Toxicology	Environmental Fate
Acute delayed neurotoxicity	Hydrolysis
Subchronic oral toxicity	Photodegradation
Dermal sensitization	Aerobic soil metabolism
Subchronic dermal toxicity	Anaerobic soil metabolism
Subchronic inhalation toxicity	Aerobic aquatic metabolism
	Microbiological[4]
Subchronic neurotoxicity[1]	Leaching
Chronic feeding	Volatility
Oncogenicity	Adsorption-desorption
Reproduction	Activated sludge[4]
Mutagenicity	Water dispersal
Emergency treatment	Terrestrial field dissipation
Ecological Effects	Aquatic field dissipation
Avian single-dose oral LD 50	Dissipation-forestry
Avian reproduction	Aquatic impact uses
Fish acute LC50	Long-term field dissipation
Acute toxicity to estuarine and marine organisms[2]	Accumulation in irrigated crops
	Disposal and storage
Fish early life-stage, aquatic invertebrate life cycle[3]	
Fish life cycle[3]	
Aquatic organisms[3]	

1/ Requirement reserved pending the review of acute delayed neurotoxicity test.

2/ For crab and mollusc.

3/ Reserved pending the evaluation of required environmental fate data.

4/ Requirement reserved pending the review and modification of the testing protocols.

Table AI-2

ENDOSULFAN DATA CALL-IN STATUS AS OF MARCH 1984
(La Rocca, 1984)

Type of Study	Completion Date	EPA Received or Due Date
TOXICITY		
Dermal sensitization	7/15/83	12/27/83
Acute delayed neurotoxicity		Extended to 11/84
Subchronic oral toxicity		Extended to 11/84
21-Day subchronic dermal toxicity		Extended to 11/84
Subchronic inhalation toxicity		Extended to 11/84
Chronic feeding		Due 11/86
Oncogenicity		Due 11/86
Reproduction		Due 11/85
Mutagenicity		Due 11/84
Special studies: Emergency treatment	11/15/83	12/27/83
FISH & WILDLIFE		
Avian single-dose oral LD50	9/8/83	12/27/84
Avian reproduction		Extended to 11/84
Fish acute LC50	4/8/83	12/27/83
Acute toxicity to estuarine and marine organisms	4/13/83	5/31/83
ENVIRONMENTAL FATE		
Hydrolysis	9/29/82	5/31/83
Photodegradation	4/20/83	5/31/83
Aerobic soil metabolism		Due 11/84
Anaerobic soil metabolism		Due 11/84
Anaerobic aquatic metabolism		Due 11/84
Aerobic aquatic metabolism		Due 11/84
Leaching	10/14/83	12/27/83
Adsorption/Desorption	11/25/82	12/27/83
Terrestrial field dissipation	9/26/83	12/27/83

APPENDIX II

PROPERTIES

Physical and chemical properties of endosulfan (I, II, and sulfate) are given in Table AII-1. Endosulfan I is the low melting point isomer which constitutes 70 percent of technical endosulfan. The solubility of these chemicals range from 60 ug/l (ppb) for endosulfan II to 220 ug/l for endosulfan sulfate, and is sufficient to be acutely toxic to all aquatic organisms tested. Technical endosulfan has a moderate vapor pressure of 1×10^{-5} torr (mm Hg), and so volatilization might be a significant dissipation pathway. However, according to EPA, the data provided by the registrants of endosulfan suggest that the chemical has no measurable vapor pressure at 20 to 75°C (EPA, 1982). The differences in the values of both vapor pressure and solubility of endosulfan which have been reported in the literature may be due to the differences in experimental and analytical methodologies. The octanol-water partition coefficient of endosulfan sulfate is slightly higher than either endosulfan I or II (Table AII-1). This suggests that endosulfan sulfate might have a greater tendency for bioaccumulation.

Table AII-1

PHYSICAL AND CHEMICAL PROPERTIES OF ENDOSULFAN I, II AND SULFATE

Empirical Formula	
Endosulfan	$C_9H_6Cl_6O_3S$
Endosulfan sulfate	$C_9H_6Cl_6O_4S$
Molecular Weight	
Endosulfan	406.95
Endosulfan sulfate	422.95
Melting Point (°C)	
Technical (NRCC, 1975)	70-100
Endosulfan I (Ali, 1978)	108-110
Endosulfan II (Ali, 1978)	207-209
Endosulfan sulfate (Ali, 1978)	198-201
Aqueous Solubility (ug/l;ppb)	
Endosulfan I (NRCC, 1975)	150
Endosulfan II (NRCC, 1975)	60
Endosulfan sulfate (Callahan et al., 1979)	220
Vapor Pressure (torr)	
Technical Endosulfan (at 25°C) (Martin and Worthing, 1977)	1×10^{-5}
Log Octanol/Water Partition Coefficient	
Endosulfan I (Ali, 1978)	3.55
Endosulfan II (Ali, 1978)	3.62
Endosulfan sulfate (Ali, 1978)	3.66
Specific Gravity	
Technical Endosulfan (NRCC, 1975)	1.745

APPENDIX III

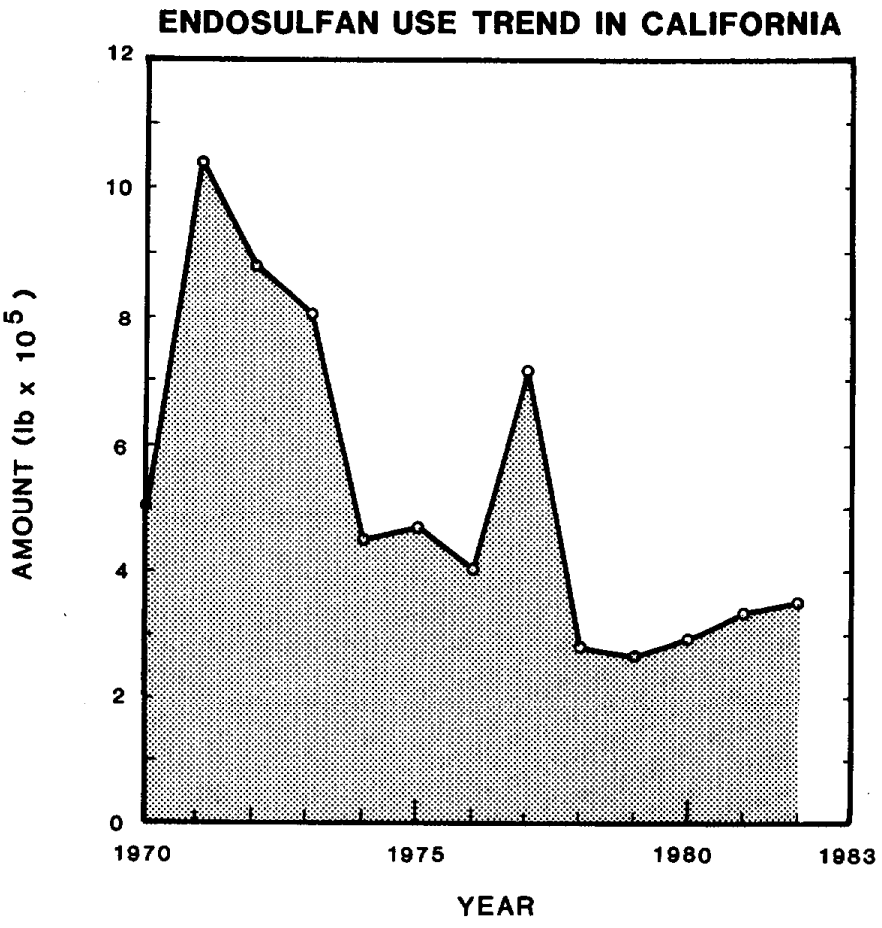
USE PATTERN IN CALIFORNIA

Endosulfan is a powerful contact and stomach insecticide used to control a wide spectrum of insects. In 1973, the California Department of Food and Agriculture (DFA) placed endosulfan in the restricted use category. This means that a permit is required from County Agricultural Commissioners for its agricultural use and that all uses must be reported. Endosulfan is, however, widely used in the state. Figure AIII-1 shows endosulfan use trend in California from 1970 to 1982. The total reported use ranged from 269,210 pounds in 1979 to over a million pounds in 1971. The most recent year for which the data are available for both the amounts of pesticide sold and used in California is 1982. During this year over 535,000 pounds of endosulfan were reportedly sold and over 350,000 pounds reportedly used in the state. Endosulfan ranked 11th among the major insecticides and 42nd among all the pesticides used in the state during 1982.

Lettuce, tomatoes, alfalfa and artichokes are the top endosulfan use crops (Table AIII-1). Reported use of endosulfan in California during 1982 on all the commodities is given in Table AIII-2. Celery and grapes were also high endosulfan use crops during that year.

The endosulfan use map of California (Figure AIII-2) illustrates that the pesticide is used mainly in the central valley as well as Monterey and Imperial counties. Figure AIII-3 shows the location of endosulfan use during 1981 in Monterey county, which is consistently the top endosulfan use county in California. Most of the insecticide is applied along the Salinas river. At the northern tip of the county endosulfan use is concentrated near the coast along the Pacific Ocean. The peak period of endosulfan application in this county is September through November (Figure AIII-4). Imperial county is another top endosulfan use county where several fish kills have resulted from its use. Endosulfan use in this county is concentrated in the Imperial, Bard and Palo Verde Valleys (Figure AIII-5). Most of the insecticide in this county is applied during August through January (Figure AIII-6) for winter lettuce. Endosulfan use in the five California counties in and around the San Francisco Bay-Delta, particularly Solano County, is quite high (Figure AIII-7). The long-term effects of endosulfan on striped bass and other fishery resources of the Bay-Delta have not been evaluated.

Figure A III-1



REPORTED USE OF ENDOSULFAN IN CALIFORNIA ON THE TOP THREE ENDOSULFAN USE CROPS
(DFA PESTICIDE USE REPORTS, 1970-1982)

Year	Lettuce	Tomatoes	Alfalfa	Other	Total of All Uses
			(lb)		
1970	153,440	88,820		66,960[1/]	507,680
1971	430,530	233,510	109,360		1,042,210
1972	192,350	167,760		186,170[2/]	882,580
1973	267,480	193,260	64,940		808,290
1974	134,200	110,620	55,590		455,730
1975	174,470	126,820	26,110		471,080
1976	142,070	107,700		23,870[3/]	401,300
1977	148,380	164,970		156,980[4/]	718,590
1978	82,240	72,440		18,430[5/]	285,490
1979	46,520	77,050	31,655		269,210
1980	40,520	51,260		51,150[3/]	294,630
1981		58,260	47,190	81,990[3/]	337,360
1982	56,780	66,860		48,130[3/]	352,730

1/ Potatoes

2/ Broccoli

3/ Artichokes

4/ Cotton

5/ Grapes

Table AIII-2

REPORTED USE OF ENDOSULFAN IN CALIFORNIA DURING 1982
(DFA, 1983)

Commodity	No. of Applications	Amount Applied (lb)	Acreage (acres)
ALFALFA	296	45,120.50	44,986.00
ALMONDS	28	744.38	1,483.50
ALMONDS	1	4.00	10.00-U- 1/
APPLE	30	1,566.71	244.50
APRICOT	9	320.78	327.00
APRICOT	1	10.00	20.00-U-
ARTICHOKE	1,045	48,132.25	56,437.87
BEANS	53	1,692.24	2,067.00
BROCCOLI	81	1,158.94	1,209.87
BRUSSELS SPROUTS	24	169.64	365.80
CABBAGE	277	1,686.83	2,061.13
CARROT	13	356.52	438.58
CAULIFLOWER	72	878.65	1,234.10
CELERY	393	37,051.40	7,587.90
COLLARD	2	17.00	34.00
COMMERCIAL TURF/LANDSCAPE	9	2.91	
CONIFERS	4	151.99	3.37
CONIFERS	1	5.00	11,000.00-U-
CORN	87	2,829.94	6,998.50
COTTON	25	2,589.17	3,124.70
CUCUMBER	43	904.74	809.00
EGGPLANT	14	54.67	126.20
FLOWERS	48	216.37	309.50
FLOWERS	6	11.56	40,019.25-U-
GRAPES	507	35,886.16	29,711.96
LETTUCE (HEAD)	1,667	54,150.59	65,330.32
LETTUCE (LEAF)	291	2,629.24	3,019.70
MELONS	288	15,524.67	20,142.65
ORANGE	1	93.50	75.00
ORIENTAL VEGETABLES	19	49.03	54.25
ORNAMENTALS	119	538.94	439.39
ORNAMENTALS	8	8.84	9,763.00-U-
PEACH	14	738.81	517.75
PEAR	56	4,624.96	2,431.25
PEAS	2	.55	4.00
PEPPERS (BELL)	54	1,624.53	1,941.25
PEPPERS (CHILI)	2	76.32	92.00
PLUM	4	152.75	192.00
POTATO	3	636.08	631.00
PRUNE	7	75.87	347.00
PUMPKINS	12	118.40	152.33
RESIDENTIAL PEST CONTROL	1	2.00	
SAFFLOWER	1	144.00	180.00
SHRUBS	1	2.50	3.00
SPINACH	95	733.50	1,060.30
SQUASH	82	2,729.45	3,074.25
SQUASH, GENERAL	2	60.00	60.00
STRAWBERRIES	172	6,617.84	3,684.25
SUGARBEET	85	3,487.44	6,666.00
SUNFLOWER	93	4,643.65	5,310.00
SWEET POTATO	8	1,717.50	1,580.00
TOMATO	1,134	66,855.09	79,517.36
WALNUT	21	2,157.23	1,787.00
WATERMELONS	20	913.76	1,070.75
	TOTAL Amount (lb)	352,729.39	

1/ Miscellaneous units.

Figure AIII-2

Map of Endosulfan Use in California (1982)

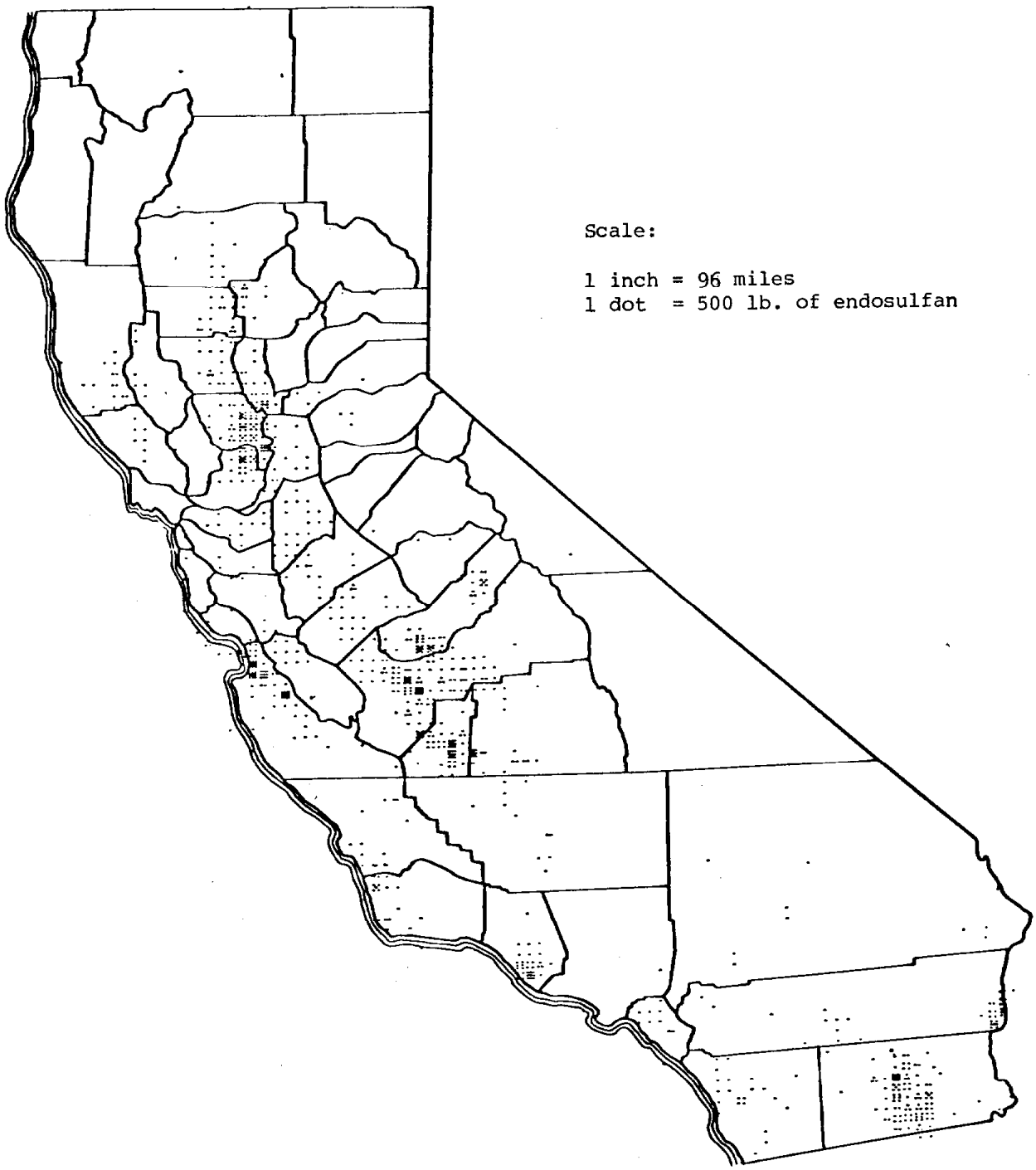


Figure AIII-3

MONTEREY COUNTY ENDOSULFAN USE MAP (1981)

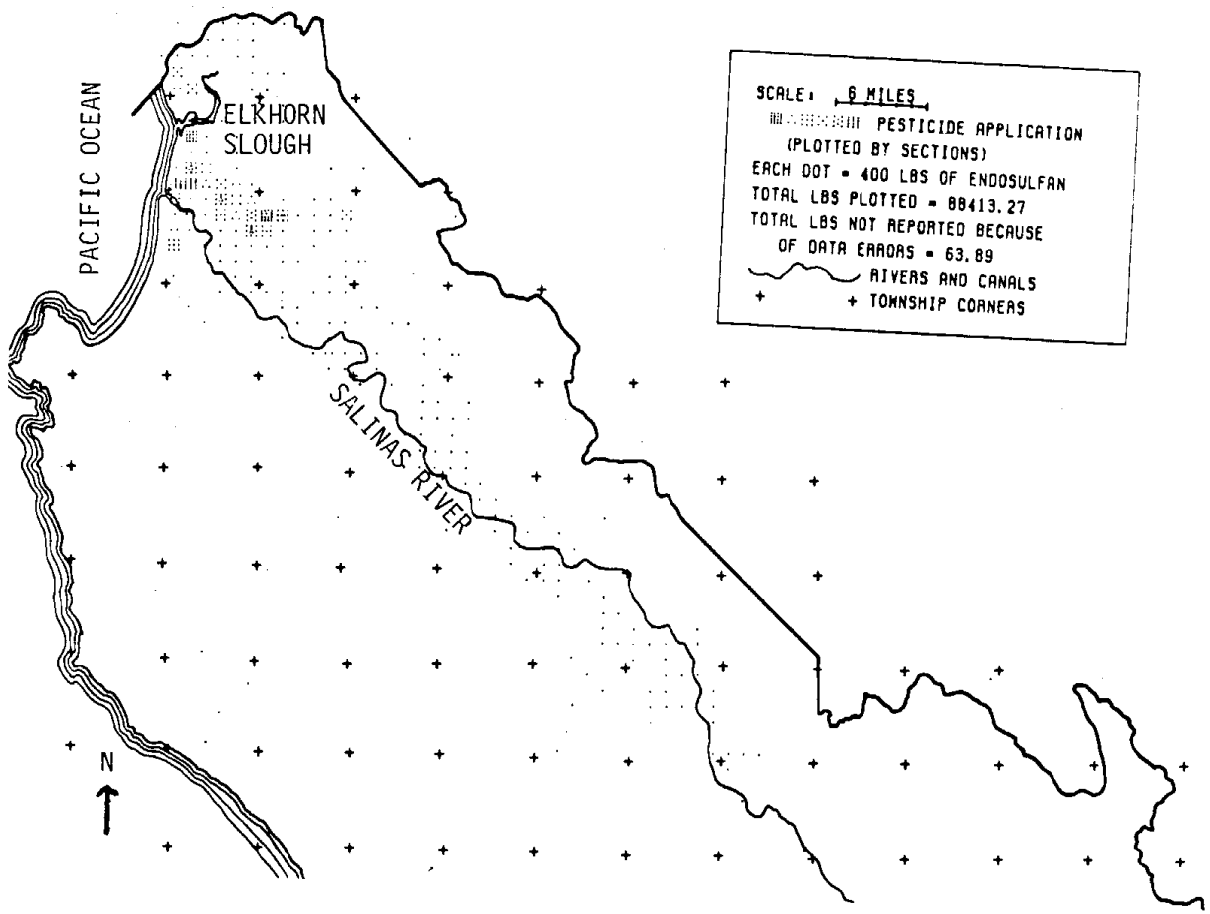


Figure AIII-4

MONTHLY USE OF ENDOSULFAN IN MONTEREY COUNTY
(1981-1982)

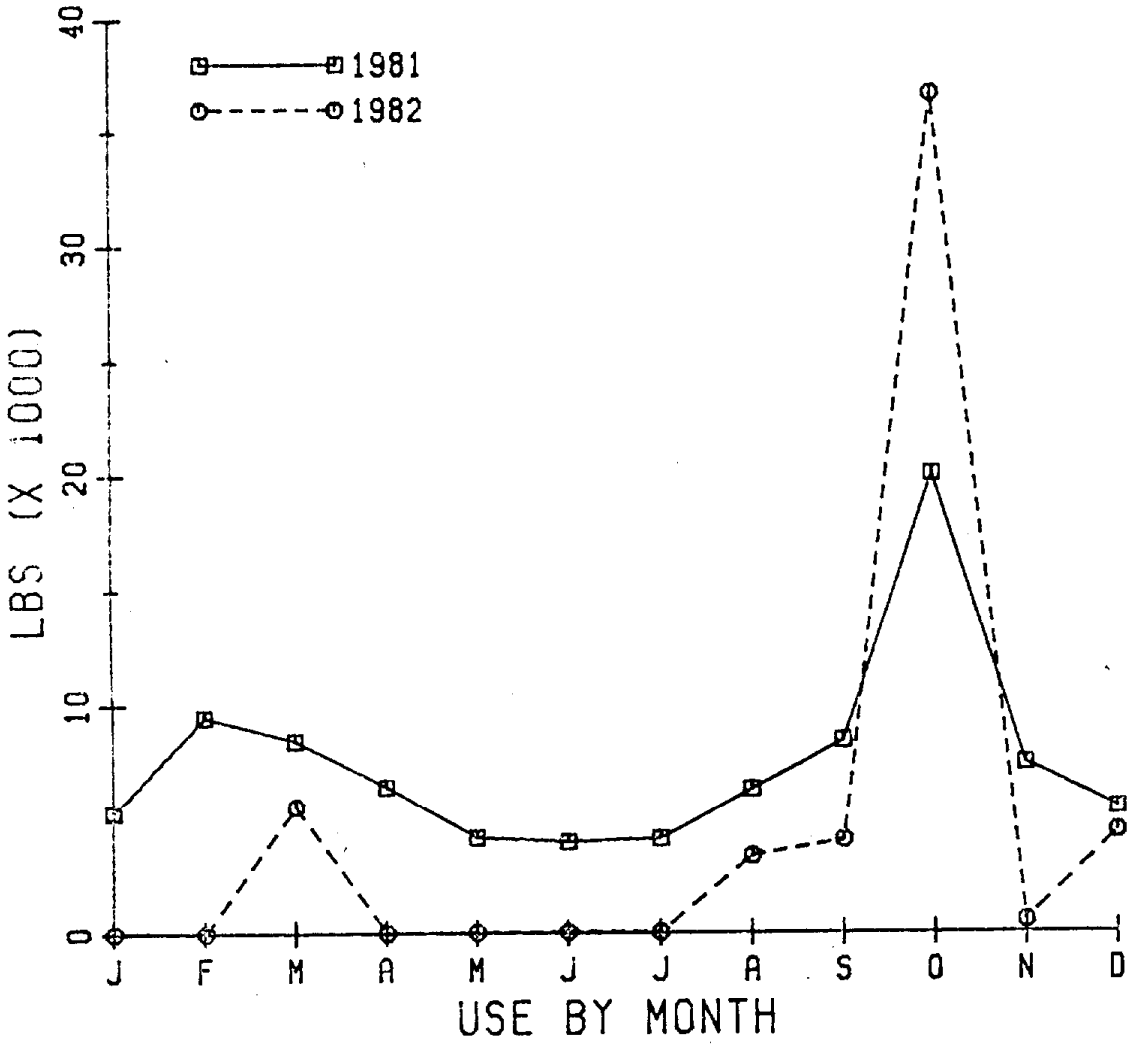


Figure AIII-5

IMPERIAL COUNTY ENDOSULFAN USE MAP (1972-1981)

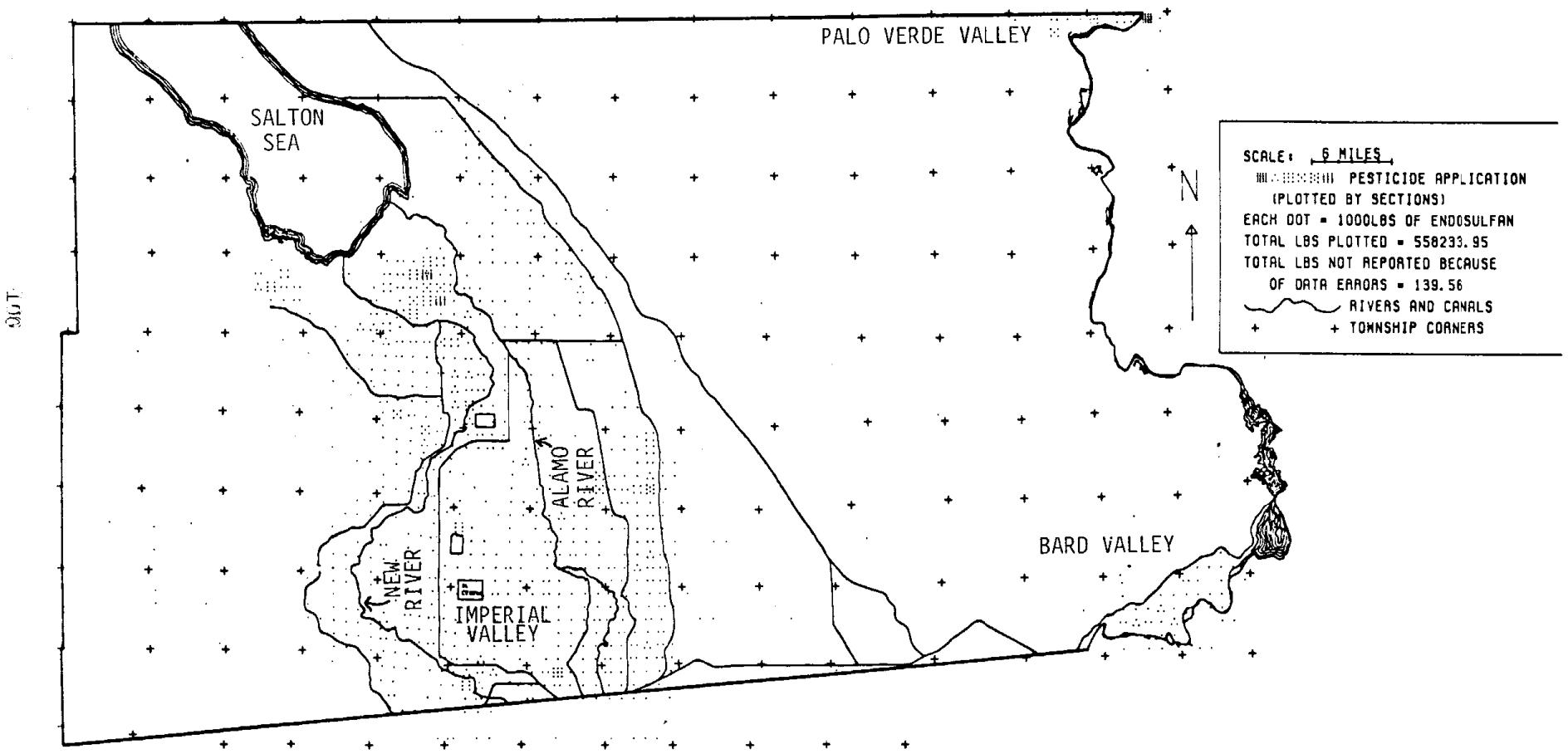


Figure AIII-6

MONTHLY USE OF ENDOSULFAN IN IMPERIAL COUNTY (1981)

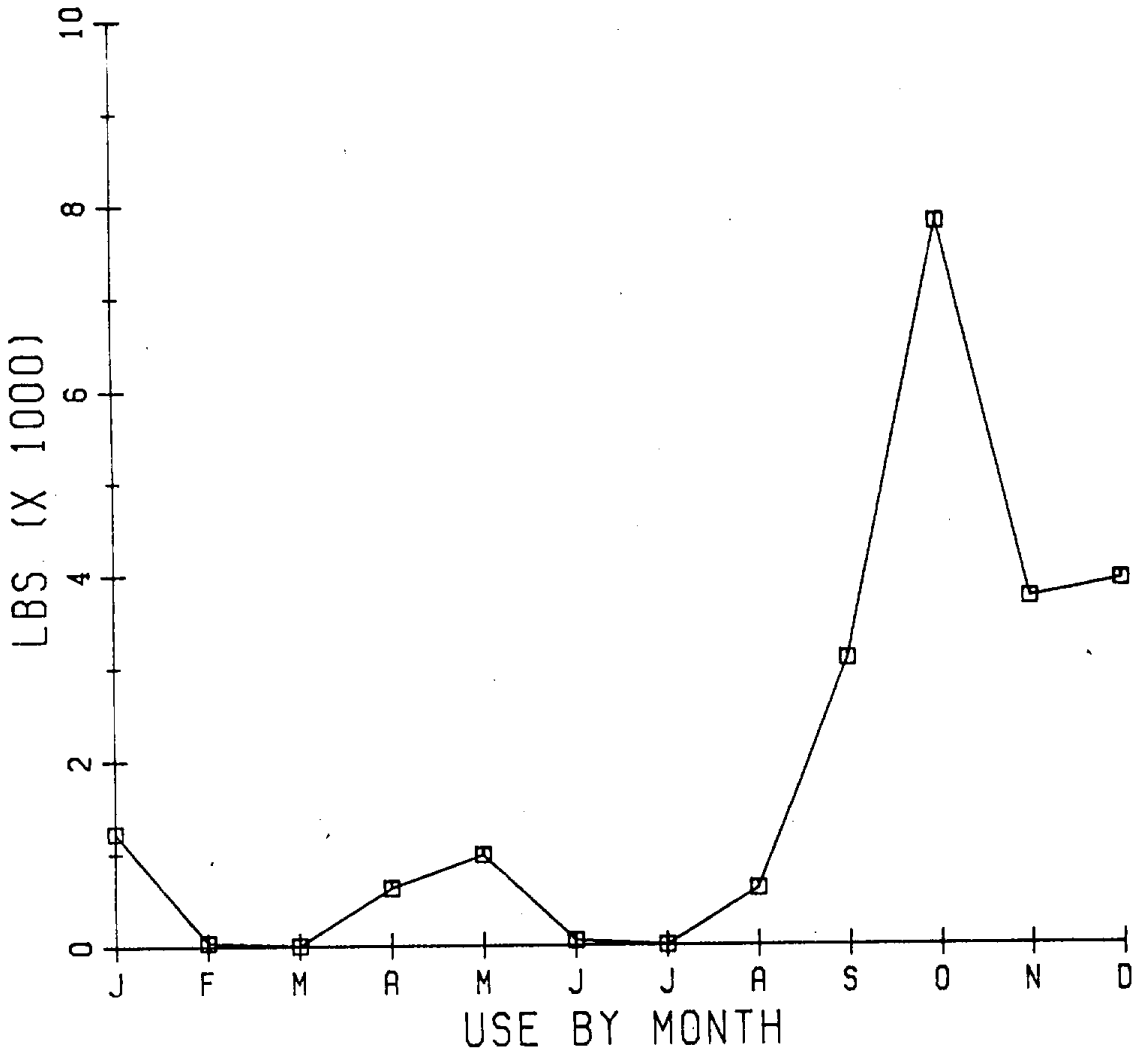
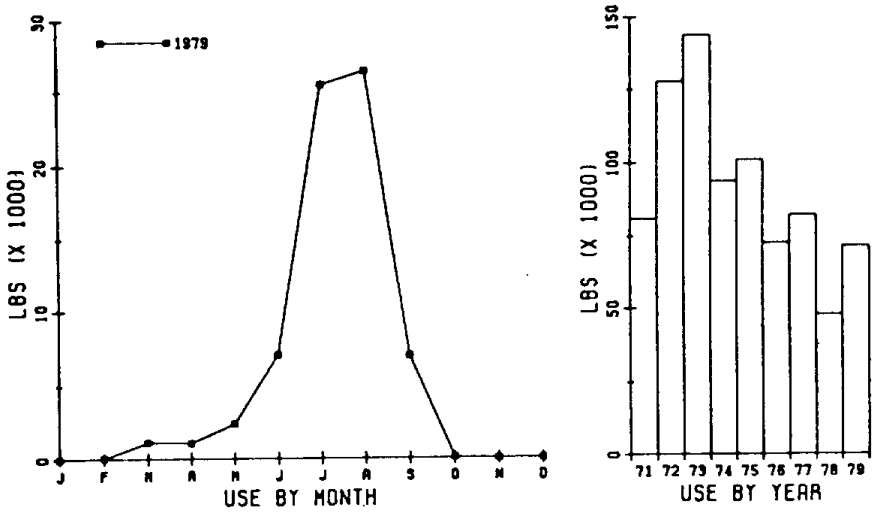


Figure AIII-7

ENDOSULFAN USE IN FIVE CALIFORNIA COUNTIES (1971-1979) 1/



1/ Sacramento, San Joaquin, Yolo, Solano and Contra Costa Counties

APPENDIX IV

ENVIRONMENTAL FATE

The environmental fate of endosulfan cannot be completely assessed because of data gaps in the literature. The Environmental Protection Agency (EPA, 1982) has asked the registrants of this insecticide to conduct additional studies on the environmental fate of endosulfan including hydrolysis, photodegradation, volatility, leaching, adsorption/desorption, aquatic and terrestrial field dissipation, aerobic/anaerobic soil and aquatic metabolism (Appendix I). Most of the research reported on endosulfan failed to analyze for endosulfan II and sulfate, which are more persistent than endosulfan I.

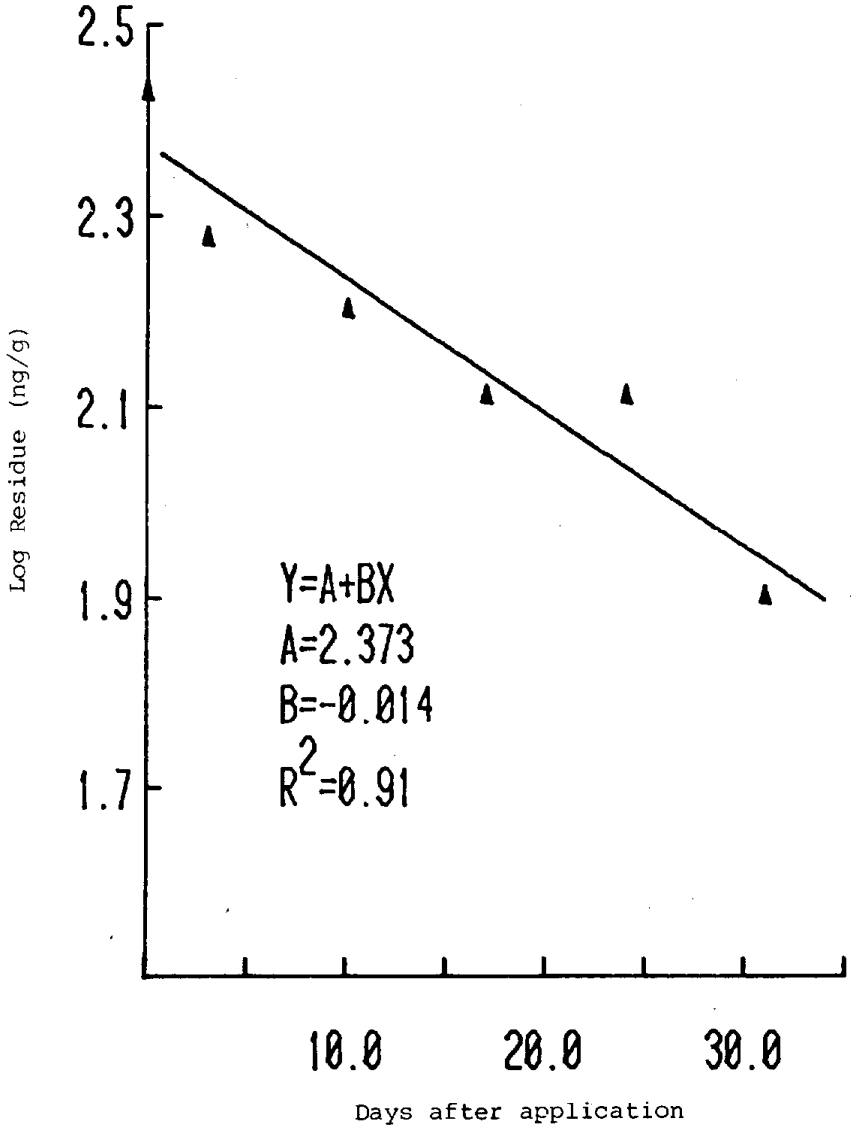
PERSISTENCE IN THE ENVIRONMENT:

Endosulfan and its metabolites, particularly endosulfan sulfate, tend to accumulate in the terrestrial environment for several years when applied annually or several times during a single growing season (EPA, 1982). The only Californias data on endosulfan persistence are available from the study of Niagara Chemical Division of FMC Corporation, which produces the insecticide. In July 1971, endosulfan (2EC) was applied at the rate of one pound per acre to soil at the company's research farm in Davis (Goebbel et al., 1982). Endosulfan residues in soil were monitored for a month, and 80 ppb of the insecticide were detected in the soil 31 days after treatment. It is not known whether endosulfan II and sulfate were also analyzed. For this report, study data were fitted to first-order decay kinetics (Figure AIV-1). From the regression analysis of the data, a half-life (t1/2) of 49.5 days for endosulfan in soil was established. Stewart and Cairns (1974) reported that the $t_{1/2}$ of endosulfan I in a sandy loam soil was 60 days. However, the degradation of endosulfan II was slower ($t_{1/2}$= 800 days), and the sulfate residues appeared to be stable for several years.

Eichelberger and Lichtenberg (1971) studied the persistence of endosulfan and other pesticides in water samples taken from a tributary of the Ohio river. Both the isomers of endosulfan (I and II) disappeared completely in four weeks. However, the authors noted that the gas chromatographic identification and quantification of endosulfan was extremely difficult after one week. The environmental conditions in a naturally flowing stream are different from the conditions of this laboratory study (i.e., dosed raw river water in closed glass containers, standing at room temperature, exposed to natural and artificial light). The rates of endosulfan degradation in the field will be different, although the mechanisms and degradation pathways will be the same.

Figure AIV-1

ENDOSULFAN DISSIPATION IN A YOLO SOIL 1/



1/ Figure developed from FMC data (Goebbel et al.,1982)

DISSIPATION PATHWAYS

Dissipation of endosulfan in the environment can occur by different mechanisms such as photodecomposition, chemical hydrolysis, microbial metabolism, leaching, volatilization and runoff.

Photodecomposition: Photodecomposition of endosulfan has been studied by Archer (1973) and his coworker (1972), and Putnam et al. (1975). The data of Archer et al. (1972) suggests that endosulfan (isomers I and II) has a photolytic half-life of approximately seven days. Endosulfan sulfate was found not to be affected by light.

Hydrolysis: Endosulfan is stable to hydrolysis in neutral or slightly acidic aqueous solutions (Coleman and Dolinger, 1978). However, at pH 8 or above 90 percent of endosulfan was hydrolozed at the ester linkage (EPA, 1982). Singh et al. (1984) reported that at pH 7 and 30°C, the hydrolysis half-life of endosuslfan I and II in water was short and of same order of magnitude (7.3 and 8.1 days, respectively). However, the rate of hydrolysis of endosulfan I under similar conditions in a 1:1 methanol/water mixture was slow (Table AIV-1).

Microbial metabolism: Studies reported in the literature (Martens, 1976; Miles and Moy, 1979) indicate that endosulfan can be metabolized by soil microorganisms. However, Tabak et al. (1981) reported that endosulfan I, II and sulfate were resistant to bio-oxidative activity of wastewater microorganisms.

Leaching: Endosulfan is adsorbed by the soil and its components (Byers et al., 1965; Richardson and Epstein, 1971). It would not therefore be expected to leach in significant quantities to ground water. El Beit et al. (1981) found that even after prolonged leaching (60+ days), endosulfan isomers did not leach below 5-inch depth in a Sudanese soil. Under normal agricultural practices the potential for ground water contamination with endosulfan will not exist except under situations of sandy soil, high rainfall, and a shallow ground water table.

Volatilization: Endosulfan and its metabolites can volatilize from water. However, endosulfan volatilization data available in the literature are limited. Callahan et al. (1979) calculated a theoretical volatilization half-life of 11 days for a quiescent water body using the equations and assumptions of MacKay and Leinonen (1975). They also stated that the volatilization half-life of endosulfan would be less in more turbulent water bodies.

Runoff: Endosulfan residues have been detected in runoff water and sediments (Epstein and Grant, 1968; Miles and Harris, 1971). The Canadian document on endosulfan (NRCC, 1975) cites a California study conducted by the Niagara Chemical Division of FMC Corporation in 1972. Endosulfan was monitored in irrigation

Table AIV-1.
RATE CONSTANT AND HALF-LIFE OF ENDOSULFAN HYDROLYSIS AT pH 7 AND 30°C
(Singh et al., 1984)

Isomer	Medium	Rate Constant (day^{-1})	Half-Life (days)
I	Water	0.095	7.3
	Methanol/water (1:1)	0.019	36.5
II	Water	0.086	8.1

runoff from a field in California which had received a single aerial application of 1 lb/acre. Approximately 15 ug/l (ppb) of endosulfan I and II were detected in the runoff water following the first irrigation. However, the residues dissipated readily to below the detection limit (5 ug/l) after the third irrigation (15 days post treatment).

Adsorption-Desorption: Sediments tend to act as a sink and accumulate endosulfan and then release the insecticide once the concentration in surrounding water drops below the equilibrium value. A dynamic equilibruim is established between its concentration in water and sediments:

$$\text{Endosulfan in water} \underset{\text{desorption}}{\overset{\text{adsorption}}{\rightleftharpoons}} \text{Endosulfan in sediments}$$

Aquatic organisms therefore will be exposed to endosulfan which may desorb from the sediments. This will prolong the exposure of organisms to the insecticide. Spencer et al. (1984) calculated the partition coefficient, (concentration in sediment/ concentration in water), k_d=249 for endosulfan. The partition coefficient based on the soil organic carbon content, K_{oc} (K_d x 100/% org. carbon), was 44,500, which indicates that organic matter has a high affinity for endosulfan adsorption.

APPENDIX V

RESIDUE ANALYSIS

OVERVIEW

Environmental monitoring for endosulfan should include the analysis of both the isomers I and II, as well as the major degradation or metabolic products, especially endosulfan sulfate. Unfortunately, most of the endosulfan studies reported in the literature have not analyzed for endosulfan II and sulfate, which are as toxic as endosulfan I, and more persistent. Therefore, the total endosulfan residues in the environment might be underestimated.

In general, multiresidue analytical methods for organochlorine pesticides are suitable for the determination of endosulfan residues. Endosulfan I, II and sulfate in water, fish, soil and sediment can be analyzed simultaneously by using appropriate extraction, cleanup and detection techniques. The December 3, 1979 issue of Federal Register (Vol. 44. No. 233, p. 69501-69504) gives a detailed multiresidue analytical method for endosulfan and other organochlorine pesticides in water. The detection limits of this procedure are: 0.005, 0.01 and 0.3 ug/l (ppb) for endosulfan I, II and sulfate, respectively. A summary of endosulfan analytical methodology can be found in the Canadian National Research Council document on endosulfan (NRCC, 1975).

Extraction

Endosulfan can be extracted from water with methylene chloride (44 FR. p. 69501). The Niagara Chemicals Division of FMC Corporation, which produces endosulfan, recommends extraction of soil and sediments with a 2:1 mixture of hexane-acetone (NRCC, 1975). California Department of Fish and Game (DFG) uses acetonitrile to extract endosulfan from fish and other aquatic organisms (Morgan, 1983).

Cleanup

Cleanup and separation of endosulfan components particularly II and sulfate can be difficult. The problem is mostly with endosulfan sulfate analysis in fish extracts. The sulfate elutes with lipids, and at least two Florisil column cleanups are required for a satisfactory resolution.

Detection

Endosulfan can be analyzed with a gas-liquid chromatograph equipped with either electron capture or microcoulometric detector. Typically, the peaks on the chromatogram are in the order of endosulfan I, endosulfan II and endosulfan sulfate. In a review paper on endosulfan, Maier-Bode (1968) has given the

retention times of endosulfan and its metabolites on three different gas chromatographic columns. A gas chromatography mass spectrometry (GC-MS) system can be used to confirm the identity of endosulfan and its metabolites.

Radian Corporation Analytical Methodology

Fish: Approximately 20 g of fish and 100 g of anhydrous sodium sulfate were blended in a stainless steel blender until the two were well mixed. To this, 150 ml of pesticide grade hexane were added and the sample were blended for two more minutes. The hexane was decanted and vacuum filtered through solvent washed filter paper. The extraction procedure was repeated twice. After the last blending, the residue was filtered, and the jar was rinsed with three 50 ml portions of hexane. The combined extracts were poured through anhydrous sodium sulfate into a 500 ml Kuderna-Danish concentrator. The extract was concentrated to 1 ml for injection into a Varian 3700 gas chromatograph under the following conditions:

Column: 6 ft x 2 mm i.d. glass
packed with 3% OV-1
on 100/120 Supelcoport
Detector: Electron capture
Carrier gas: Helium at 30 ml/min
Column temperature: 185°C isothermal
Injector and detector temperatures: 220°C

Sediment: Sediment samples were filtered to remove the water. Endosulfan residues were extracted from a 50 g sediment sample with 300 ml of 1:1 hexane-acetone mixture in a Soxhlet apparatus for 16 hours. The volume was concentrated to 10 ml before injection into the gas chromatograph. The GC conditions were the same as for the fish analysis. Positive results for endosulfan I, II and sulfate were confirmed by reanalysis with a second GC column packing material.

APPENDIX VI

CRITERIA AND STANDARDS

Although endosulfan has been registered for use as a pesticide for thirty years, very few criteria and standards have been established for protection of human health or aquatic life. Table AVI-1 summarizes the existing limits for endosulfan in water.

Aquatic Life Protection

For the protection of freshwater life, EPA in 1980 developed ambient water quality criteria of 0.056 ug/l as a 24-hour average, and 0.22 ug/l as an instantaneous maximum (EPA, 1980). The corresponding criteria for saltwater aquatic life, are 0.0087 and 0.034 ug/l. These criteria are similar to the the endosulfan water quality objective of 0.003 ug/l established by the International Joint Commission of the United States and Canada for the Great Lakes region (IJC, 1977). For the protection of predators, the National Academy of Sciences (NAS) suggested a residue value in whole fish of 100 ug/kg (ppb) endosulfan either singly or in combination with other chlorinated pesticides (EPA, 1973).

Human Health Protection Water Criteria: EPA established in 1980 two different ambient water quality criteria to protect human health: (i) 74 ug/l for consumption of water and aquatic organisms living in the water, and (ii) 159 ug/l for ingestion of contaminated aquatic organisms alone (EPA, 1980). However, these criteria are based on an ADI (Acceptable Daily Intake) which was calculated using the data of a rodent feeding bioassay. Gravitz (1984) indicated that this ADI may be underprotective since cattle and man appear to be the most sensitive species according to EPA.

A drinking water advisory for endosulfan has not been set by EPA. However, using the ADI of 0.28 mg/day for a 70 kg person (EPA, 1980), a "no-observable-adverse-effect-level" (NOAEL) of 28 ug/l was calculated based on the following assumptions: (i) two liters of water are consumed by a person in a day; and (ii) 20 percent of daily endosulfan intake is through water, while the rest is from other sources such as food.

Food Tolerances: Residue limits for endosulfan have been set for food by the federal government. These values range from 0.1 to 2.0 mg/kg (ppm), and are listed in Table AVI-2. The existing food tolerances yield a theoretical maximum residue contribution (TMRC) of 0.7364 mg/day for a 70 Kg person (EPA, 1982). This value is 263 percent of the acceptable daily intake (ADI) of 0.28 mg/day for a 70 kg person. Further, this does not include any potential contributions from drinking water, fish and other seafood.

Table AVI-1

WATER LIMITS FOR PROTECTION OF HUMAN HEALTH AND AQUATIC LIFE FROM ENDOSULFAN
(U.S. EPA, 1980)

Endosulfan Concentration	Aquatic Life Protection	Human Health Protection
ppb (ug/l)		
0.0087	Saltwater 24-hr average	
0.034	Saltwater instantaneous maximum	
0.056	Freshwater 24-hr average	
0.22	Freshwater instantaneous maxium	
28		Drinking water guideline based upon ADI [1/]
74		Consumption of contaminated water and aquatic life
159		Consumption of only aquatic life

[1/] Calculated by the SWRCB Staff

Table AVI-2

ENDOSULFAN TOLERANCES IN OR ON RAW AGRICULTURAL COMMODITIES
(40 CFR Parts 100-399)

Residue Level ug/g (ppm)	Agricultural Commodity
0.1	Grains: Barley, oat, rye, wheat. Misc: Blueberries, sugar beets (without tops)
0.2	Fat, meat and by products: Cattle, hog, goat, sheep, horse Nuts: Almond, macadamia, filbert, walnut, pecan Seeds: Safflower, mustard, grape Straw: Barley, oat, rye. wheat Vegetables: carrot, sweet corn, potato, sweet potato.
0.3	Alfalfa (fresh)
0.5	Milk fat, sugarcane
1.0	Alfafa (hay), almond hull, cotton seed
2.0	Fruits: Apple, apricot, cherry, grape, melon, nectarine, peach, pear, pineapple, plum, prune, pumpkin, strawberry Vegetables: Artichoke, bean, broccoli, brussel sprout, cabbage, cauliflower, celery, collard, cucumber, eggplant, kale, lettuce, mustard green, pea (succulent), pepper, squash, tomato, turnip (green), watercress

A pre-harvest interval of 14 days should be followed between the last application of endosulfan to lettuce and the harvest of the heads (UC, 1979). Pre-harvest intervals are established to lower the pesticide residues on harvested produce below current food tolerances.

Work Environment: Exposure limits for endosulfan in work environment situations have not been established by the U.S. Occupational Safety and Health Administration (OSHA) or National Institute of Occupational Safety and Health (NIOSH). The American Conference of Governmental and Industrial Hygienists (ACGIH) has recommended a threshold limit value-time weighted average (TLV-TWA) of 0.1 mg/m3 (ACGIH, 1977). This value suggests that occupational uptake of about 14 ug/kg/day is considered safe. ACGIH also proposed a TLV of 0.3 mg/m^3 for short-term exposure (15 minutes). A 2-day worker reentry interval for endosulfan has been established by DFA and EPA. This safety period is the time between pesticide application and when workers are allowed to enter the field to engage in an activity requiring substantial body contact with treated foliage.

Effluent Standards

The Federal Water Pollution Control Act Amendments of 1972 established a comprehensive program to "restore and maintain the chemical, physical, and biological integrity of the Nation's waters". New industrial direct discharges of pollutants should comply with New Source Performance Standard (NSPS) effluent limitations based on best available demonstrated technology. New and existing indirect discharges to publicly owned treatment works should comply with pretreatment standards. The proposed standards applicable to discharges of endosulfan I and II resulting from the manufacture of endosulfan active ingredients are (49FR, p. 24503, June 13, 1984):

	Daily max.	4-day avg.	30-day avg.
		(mg/l)	
Endosulfan I	0.009	0.032	0.02
Endosulfan II	"	"	"

Since EPA assumes that endosulfan sulfate is not present in the discharges, it is not regulated (47FR, p. 53994, Nov. 30, 1982). EPA has proposed a zero discharge of endosulfan I and II for formulators/packagers (47FR, p. 24499, June 13, 1984).

APPENDIX VII

ENDOSULFAN LABEL

READ THE LABEL

WARNING: KEEP OUT OF REACH OF CHILDREN.
Hazardous if Swallowed, Inhaled, or Absorbed through skin. Do not breathe spray mist. Do not get in eyes, on skin or on clothing. Wash thoroughly with soap and water after handling and before eating or smoking; wear clean clothing. During commercial or prolonged exposure in spray-mixing and loading operation, wear clean synthetic rubber gloves and a mask or respirator of a type passed by the U.S. Bureau of Mines for Thiodan protection. Do not apply or allow drift to areas occupied by unprotected humans or beneficial animals. Workers entering areas within 24 hours of application should wear protective clothing.

This product is toxic to fish and wildlife. Keep out of lakes ponds and streams. Birds feeding in treated areas may be killed. Do not apply when weather conditions favor drift from the areas treated. Do not apply when run-off is likely to occur. Do not contaminate water by cleaning of equipment, or disposal of wastes and containers. Apply only as specified on the label.

This product is toxic to bees. Do not apply when bees are actively visiting the area.

Destroy containers by perforating or crushing. Bury or discard in a safe place away from water supplies. Do not use, pour or store near heat or open flame. Do not store at temperatures below 20°F.

ANTIDOTE

EXTERNAL - In case of contact, immediately remove contaminated clothing and flush skin or eyes with plenty of water; for eyes get medical attention.

INTERNAL:- If swallowed give a tablespoon of salt in a glass of warm water and repeat until vomit fluid is clear. Have victim lie down and keep quiet. Call a physician immediately.

NOTE TO PHYSICIAN: Endosulfan is a central nervous system stimulant and may cause convulsions. There is no specific antidote. Barbitutic acid derivatives may be used in treatment.

AA

TOXO TOXODAN 3 EC

Active Ingredients:	BY WT.
*Endosulfan(Hexachlorohexahydro-methano-2,4,3-benzodioxathiepin oxide)	33.70%
Xylene	60.50%
Inert Ingredients:	5.80%

*Thiodan (R) Products of Canadian Hoechst Ltd. U.S. Pat. No.2,799,685

STATE REG. NO. 11219-50086-AA

E.P.A. Est. No. 35296-CA-2

NET GALLONS _____

Manufactured By

TOXO SPRAY DUST, INC.
12651 E. LOS NIETOS ROAD
SANTA FE SPRINGS, CALIF.

PHONE: 714-544-6300

APPENDIX VIII

AGRICULTURAL EXTENSION SERVICE
UNIVERSITY OF CALIFORNIA

UNIVERSITY HALL
2200 UNIVERSITY AVENUE
BERKELEY 4, CALIFORNIA 94720

May 23, 1966

THIODAN and FISH KILLS

Recently two fish "die-offs" have occurred because Thiodan (endosulfan) was carried into drainage canals by drainage water from treated fields. In each case the field was irrigated on the day following the Thiodan treatment.

Thiodan, like other pesticides, is toxic to fish, and farmers should be warned about letting water from treated fields drain into canals or ditches where fish may be present.

The following suggestions are being made to reduce the possibility of killing fish:

1. When possible the field should be irrigated before the pesticide is applied.
2. If irrigation is necessary following a treatment, a waiting period of 3 to 5 days should be observed between the time of application and irrigation.
3. Keep the run-off water from treated fields to a minimum.

This release is directed at Thiodan, but the same general principles apply to all insecticides. All of these materials are toxic to fish to one degree or another.

This information should be gotten to farmers, applicators and others, but it is not advisable to do this by the use of newspapers, radio or television. The information can be given to them through county newsletters, personal contact or at various meetings.

Please make every effort to help farmers adopt one of these practices when Thiodan or other chemicals are involved as we want to stop the killing of fish and avert any additional unfavorable publicity or legislation.

John E. Swift

John E. Swift, Extension Entomologist
Statewide Coordinator--Pesticides

APPENDIX IX

SELECTED REVIEW COMMENTS AND RESPONSES

The draft endosulfan report was reviewed by scientists recognized in the fields of environmental fate and toxicology of endosulfan. In addition, review comments were received from regional water quality control boards, state and federal agencies, and Hoechst AG., primary manufacturer of endosulfan. The report was revised, where appropriate, to reflect the comments of the reviewers. Responses to selected comments are listed below.

Comment 1:

The EPA ambient water quality criteria for endosulfan were dervied from a data base which lacks true chronic toxicity test with fish. Site specific data are not available to warrant recommending the EPA water quality criteria.

Response:

A number of data gaps have been identified for endosulfan (Appendix I), including chronic toxicity test with fish. Chronic toxicity data are required to develop the "24-hour average" ambient water quality criteria to protect aquatic life from long-term effects of a pesticide. These data are sparse for endosulfan. This report therefore recommends that the State Board adopt only as a guideline the "instantaneous maximum" criteria which were derived from acute toxicity tests. There is a much broader data base for the acute toxicity values compared with the chronic toxicity data base.

Site specific acute toxicity tests with California resident species are not available. Hoechst Chemical Co., the manufacturer of endosulfan, has offered to conduct site specific studies in California to fill these data gaps.

In 1981, State Board staff developed ambient water quality criteria for 2,4-D propylene glycol butyl ether ester (40 ppb instantaneous maximum and 2 ppb 24-hour average) using the EPA methodology and aquatic toxicity data from published literature. Site specific toxicity studies with resident aquatic species were subsequently conducted to validate the 2,4-D PGBEE criteria. The water quality criteria developed from these bioassays were similar to the criteria based on the national toxicity data base. In view of this experience, interim California criteria for endosulfan acute toxicity can be based on existing information in the literature, pending development of site-specific data.

Comment 2:

There is no data base to substantiate the NAS claim that residues in prey of 0.1 ppm endosulfan (or total chlorinated hydrocarbons) will cause harm to predators.

Response:

In the absence of any other guideline such as EPA's tolerance level or FDA's action level, the NAS guideline is the only available criterion to compare monitoring data for endosulfan residues in fish. The report recommends that the National Academy of Sciences substantiate the validity of the NAS guideline.

Comment 3:

Table II-11 lists "certainty of cause" for fish kills. What constitutes a "known" fish kill?

Response:

The Department of Fish and Game assigns a fish kill as "known" to be caused by endosulfan when chemical analysis of dead fish tissue or water sample reveals lethal concentrations of the pesticide.

Comment 4:

The measurement unit of ug/kg (ppb), used in the report for endosulfan monitoring data, has the effect of unduly magnifying the residue values.

Response:

Endosulfan is extremely toxic to fish and other aquatic life at low parts per billion (ppb) levels. Lowest reported LC50 values for pink shrimp, striped bass and rainbow trout are below 1 ppb (0.04, 0.1 and 0.17 ppb, respectively). The EPA ambient water quality criteria for endosulfan ranges from 8.7 parts per trillion (ppt) to 0.22 ppb (see Appendix VI - Criteria and Standards). For these reasons, it is quite appropriate to use the units of ppb in reporting monitoring data.

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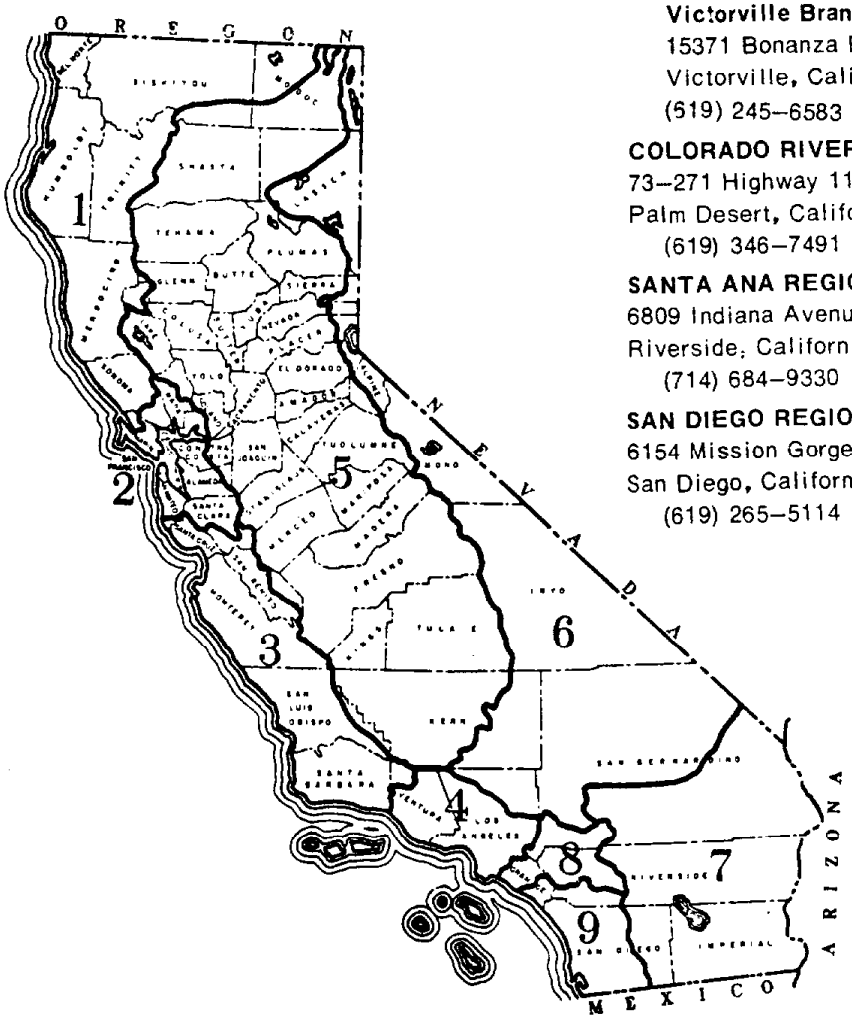

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11/84