Environmental Risk Sciences, Inc.
381 Bush Street, Suite 600
San Francisco, California 94104

Phone: (415) 392-7422
Fax : (415) 296-9788

November 9, 1990

Mr. Hugh Murphy
Hayward Fire Department
22300 Hayward Boulevard
Hayward, CA 94541

Re: Toxicological Review of Verification Test Results
Laguna Park, Hayward, CA

Dear Mr. Murphy:

As requested, I have reviewed the verification sample test results for residual pesticides on the Laguna Park site in Hayward. A site plan documenting the locations of the sample points and the certified laboratory reports for the six most recent samples are presented in Terratech's "Confirmation Test Results,..." letter dated November 9, 1990.

Based on my statistical calculations and review of the U.S. EPA Manual SW-846, a sufficient number of random samples have been collected and analyzed at this site to provide a 95% confidence level that the residual onsite pesticide concentrations are well below the levels of concern identified in the health risk assessment prepared by my firm. Please note that the U.S. EPA generally recommends approximately an 80% confidence level for most environmental sampling, so that the 95% level your agency has required represents an additional degree of health conservatism.

Based upon the recent analytical results for these samples showing no detectable levels of organochlorine pesticides in soil at the Laguna Park site, the original conclusions of my 1989 health risk assessment report remain valid. Therefore, it is my professional judgment that according to the best available health risk assessment methodology supplied by the U.S. EPA, the residual soil concentrations of the organochlorine pesticides at the Laguna Park site pose an insignificant risk to human health.

Very truly yours,

ENVIRONMENTAL RISK SCIENCES, INC.

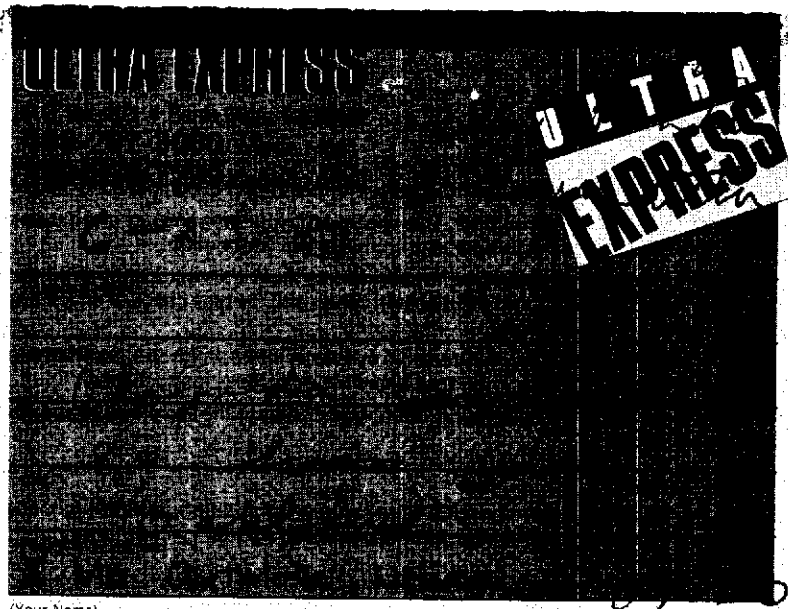
Scott Wolff

Scott K. Wolff
Principal

Enclosures

cc: Pam Evans, Alameda County Health Agency (w/ enc.)
Laura Rice, The Plymouth Group (w/o enc.)
Richard Hiett, Regional Water Quality Control Board (w/o enc.)

ERS



Waybill 131758

FROM (Your Name)

Phone Number (Very important) ( )

Company

Department/Floor No.

Environmental Risk Sciences

Street Address

381 Bush St

City

State

Zip Code

SF

Print When Picked Up

(Returned)

Time
A.M.
P.M. 07:18

X

YOUR BILLING REFERENCE INFORMATION

PAYMENT METHOD

Primary responsibility for payment lies with person or company ordering.

☐ BILL SHIPPER
☐ BILL RECIPIENT
☐ BILL THIRD PARTY
☐ BILL MASTERCARD, VISA

FROM ZONE

TO ZONE

☐ DIRECT PAK℠
(fastest service - direct from pick up to delivery)

☐ INSTA PAK℠
(2 Hour service. 3 Hours for destinations more than 30 miles)

☐ SHUTTLE SERVICE℠
(4 Hour service - must be ready by 2:00 P.M.)

☐ BUSINESS PAK℠
(delivery by 5:00 PM. - must be ready by 12:00 P.M.)

☐ OVERNIGHT PAK℠
(delivery by 11:00 A.M. following business day)

☐ AIRPORT SERVICE
(same day delivery to destinations nationwide)

☐ RESTRICTED DELIVERY
(only to the person listed on waybill)

☐ TELEPHONE CONFIRMATION

WAITING TIME _____ minutes
(applies to waits in excess of 5 minutes at pickup or destination)

TOTAL WEIGHT _____
(extra charge applies to anything over 25 pounds)

☐ RETURN
☐ Direct
☐ Same day by ____ A.M. ☐ Next Day
P.M. ☐ Mail

☐ DELIVER WEEKENDS OR AFTER HOURS
(extra charge applies)

☐ Envelope / s ☑ Parcel / s (

Driver: 93

HEALTH RISK ASSESSMENT
SUNNYSIDE COMMONS PROJECT
HAYWARD, CALIFORNIA

Prepared for

THE PLYMOUTH GROUP
2047 OLD MIDDLEFIELD WAY
MOUNTAIN VIEW, CALIFORNIA 94043

Prepared by

Scott K. Wolff
Principal

ENVIRONMENTAL RISK SCIENCES, INC.
381 BUSH STREET, SUITE 600
SAN FRANCISCO, CALIFORNIA 94104

JUNE 22, 1989

ERS

TABLE OF CONTENTS

ERS

LIST OF TABLES

ERS

EXECUTIVE SUMMARY

This health risk assessment evaluates the potential health risks attributable to low concentrations of pesticides in surface soils at the Sunnyside Nursery in Alameda County, California. This parcel is currently the proposed location for a residential housing development. Soil analyses have detected DDT, DDE, dieldrin, endrin and endosulfans in on-site surface soils. The chosen remedial alternative for the highest concentrations of pesticides (above California Title 22 TTLC's or equivalent -- see Table 2-3) is to bury them beneath the roadways to be constructed on-site.

The potential carcinogenic and noncarcinogenic health risks are evaluated using health criteria published by the Environmental Protection Agency (EPA). In addition, the health risk assessment has been prepared to follow EPA risk assessment guidelines. To ensure health conservatism, this analysis estimates the worst-case (upper-bound) and best-estimate (average-case) level of potential exposures and consequent health risks to a hypothetical maximum exposed individual (MEI) who is presumed to have access to the site everyday during his entire lifetime.

Using an estimate of the pesticide concentrations that will remain in the soils following the excavation activities, a worst-case total lifetime cancer risk level of 2.3E-06 and a best-estimate lifetime cancer risk of 3.9E-07 have been estimated for the MEI. The best-estimate cancer risk is generally regarded as well below risk levels of regulatory concern. Noncancer health hazard indices of 1.8E-01 and 8.7E-02 has been derived for the MEI for the worst-case and best-estimate cases. These noncancer risk levels are approximately one order of magnitude below unity indicating little probability for the occurrence of noncarcinogenic health risks.

In conclusion, based on the health risk values derived using

currently accepted risk assessment methods, the levels of pesticides that will be allowed to remain in on-site surface soils following the completion of excavation activities would pose an insignificant health risk to individuals who will have continual access to the Sunnyside Commons property. This conclusion is especially enlightened when considering that the health risk assessment has employed many health conservative assumptions throughout the entire analysis that are designed to overestimate the estimated health risks.

1.0 INTRODUCTION

Environmental Risk Sciences, Inc. (ERS) has been retained by The Plymouth Group to prepare a health risk assessment (HRA) for the Sunnyside Nursery located in Alameda County, California. This parcel was used primarily as agricultural land and later as a nursery and is currently the proposed location of a residential housing development. A preliminary soil characterization study completed by Terratech, Inc. has detected several organic pesticides including dieldrin, endosulfan, DDT, DDE and endrin in surface soils at residual concentration levels at the site (Terratech, 1989a). In addition to this initial study, Terratech Inc. has prepared a closure plan for the Sunnyside Nursery site (Terratech, 1989b). The reader is referred to both of these documents for further detail.

The objective of this health risk assessment is to estimate the level of potential health risk to the future residents of the proposed houses that are directly attributable to the chemical compounds detected in on-site surface soils. In following the currently acceptable health risk assessment methodologies published by the State of California Department of Health Services (DHS) and the U.S. Environmental Protection Agency (U.S. EPA), this report presents a screening level analysis designed specifically to estimate the upper-bound levels of health risk in the potentially exposed population. Upper-bound health risk estimates are derived by assuming that the maximally exposed individual (MEI) will have access to the soil at the proposed housing development every day of his/her entire lifetime. Using a screening level analysis allows the risk analyst to calculate health risk estimates that are not likely to be exceeded by any individual having continual access to the site.

The risk assessment methodology used in this analysis is based on the guidelines published by the U.S. EPA in several documents

including the cancer risk assessment guidelines, the Superfund Public Health Evaluation Manual, and the Superfund Exposure Assessment Manual (Federal Register, 1986; U.S. EPA, 1986a; U.S. EPA, 1988c). In addition, HRA guidance documents published by the California DHS, including The California Site Mitigation Decision Tree Manual, have been consulted during the preparation of this report (DHS, 1986; DHS, 1987).

The HRA is organized to follow the risk assessment guidelines published by the EPA. Chapter 2 introduces the risk assessment process in the hazard identification section. This section summarizes the results of the surface soil sampling programs and includes the concentrations of the chemical compounds detected on-site that will be included in the HRA.

The third chapter is called dose-response assessment. This section focuses on presenting the health criteria published by regulatory agencies for the compounds of concern. Health criteria for the potentially carcinogenic compounds DDE, DDT and dieldrin are presented as cancer potency factors (CPFs) that are derived by the EPA. Noncarcinogenic health criteria are presented as reference doses (RfDs) for DDE and DDT, endosulfan and endrin the compounds included in the health risk assessment that are known to induce noncarcinogenic health effects.

An integral part of dose-response assessment is the preparation of toxicological and environmental fate profiles for the compounds identified in on-site soils. These profiles present the salient chemical/physical and mammalian/human toxicology properties of the compounds included in the health risk assessment. These profiles are found in Appendix A.

The fourth chapter, exposure assessment, estimates the upper-bound daily exposures to the individuals who will be potentially exposed to the surface soil compounds. The exposure routes

analyzed specifically in this health risk assessment include the incidential ingestion of soil and the dermal absorption of the organic pesticide compounds via direct contact with soil. Other exposure pathways have not been included in the HRA because they present insignificant health risks compared with the direct exposure pathways. This philosophy towards HRA is consistent with a screening level approach specified for this study.

The results of the health risk assessment are presented as risk characterization in Chapter 5. Risk characterization provides numerical values of the upper-bound estimates of health risk that may be experienced by an individual who would be exposed to the highest levels of pesticides throughout an entire lifetime of potential exosure. The specific health criteria presented in the dose-response section are combined with the exposure estimates from the exposure assessment to derive the estimates of potential health risk in the maximally exposed individual.

Chapter 6 presents the literature references used to prepare the health risk assessment.

2.0 HAZARD IDENTIFICATION

The initial step in the preparation of a health risk assessment is to identify the potential human health hazards posed by the chemical compounds detected at the site. By definition, hazard identification includes a presentation of the analytical sampling data and a detailed description of the analytical soil data most relevant for the health risk assessment.

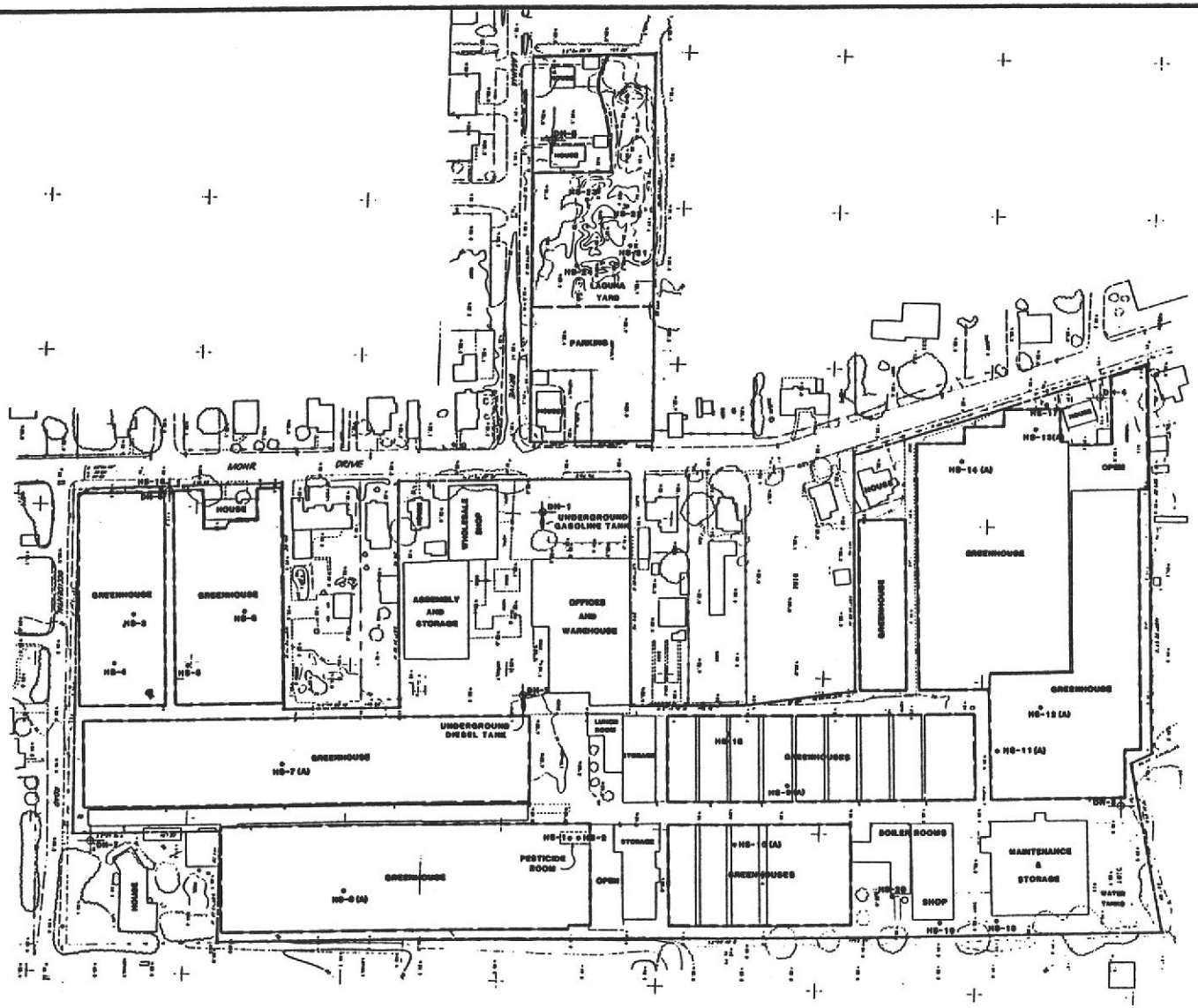
2.1 CHEMICAL ASSESSMENT OF THE SUNNYSIDE NURSERY SITE

The Sunnyside Nursery parcel is located near the City of Hayward in Alameda County, California. Since approximately 1955, the year the business was started, Sunnyside Nursery has grown ornamental plants on-site. Prior to having a nursery, the site was used as agricultural land.

Surface soil samples were collected at the site by Terratech, Inc personnel in January 1989 during a Phase I assessment (Terratech, 1989a). Figure 1 shows the approximate locations of these on-site samples. The soil samples were analyzed for metals (EPA Method 6010), volatile organics (EPA Method 8240), oil & grease (EPA Method 413.1), BETX (EPA Methods 8015 and 8020) and organochlorine pesticides/PCB's (EPA Method 8080). These soil samples included both composite and individual samples. In addition, one groundwater sample was obtained at the site during this phase. An additional soil sampling was conducted at the site in April 1989 to further characterize on-site soil at specific locations.

Analytical data from these surface soil samples represent the information that form the basis for determining the compounds of concern in this health risk assessment. Analytical results of the surface soil samples for the purpose of conducting a health risk assessment are described below. The reader is referred to

FIGURE 1



Base Map: "Boundary Topographic, Plymouth - Hayward", Prepared by Cornish Consulting Engineers, Dated February 1988.

REVISIONS					
NO.	DATE	DWN	CHKD	APPR	
1	6-16-89	DMK	BRL		MORE DETAIL ADDED

TERRATECH

SITE PLAN
SUNNYSIDE NURSERY
HAYWARD, CALIFORNIA

FIGURE 1
PROJECT 4454/2

the Phase I report for the chain-of-custody records, laboratory analysis reports and additional detail regarding the site sampling program and analytical results of the soil samples (Terratech, 1989a).

The purpose of the initial sampling activity on-site was to provide composite and individual soil samples to assess the presence of chemical compounds on-site. The specific EPA analytical methods recommended for analysis were based primarily on the historical uses of the site. Several chemicals were detected in the on-site soil samples. The organochlorine pesticides including 4-4'-DDE, 4-4'-DDT, dieldrin, endrin, endosulfan I, endosulfan II, and endosulfan sulfate were the chemical compounds detected in on-site soils. Low levels of recoverable grease & oil and TPH were detected in a few samples. In addition, metals were detected at soil levels consistent with natural background concentrations. No volatile organics or BETX compounds were detected in the soil samples.

Table 2-1 presents a summary of the analytical results from the January and April soil analyses. The endosulfan compounds have been detected in much greater frequency than the other organochlorine pesticides in on-site soil. Endrin, dieldrin, 4-4'-DDE and 4-4'-DDT have been detected relatively infrequently compared to the endosulfan compounds.

Table 2-2 identifies the State of California Title 22 TTLC and STLC values for these pesticides. Note that TTLC and STLC values are available for all of the compounds except the endosulfans. The values in parentheses have been derived for the endosulfans based on their known mammalian toxicity and TTLC/STLC values for the other pesticides. The derivation of these estimates for the endosulfans are explained in detail in the endosulfan toxicity profile in Appendix A.

Note that the TTLC values for dieldrin, DDE and DDT are greater than the highest detected concentrations on-site. The TTLC for endrin is lower than the highest detected on-site soil levels. In addition, the safe-soil level for the endosulfans derived by ERS, Inc. is lower than the highest detected on-site concentration for these compounds.

2.2 HEALTH RISK ASSESSMENT SOIL DATA

The closure plan prepared by Terratech, Inc. has indicated that surface soil will be excavated and placed under roadways to be constructed on-site (Terratech, 1989b). Verification samples will be obtained from the remaining soil at the site to assess the concentrations of pesticides (both organochlorines and carbamates) that will remain under residential yards following completion of the proposed soil excavation.

Since the objective of this health risk assessment is to estimate the level of health risk to the future residents of the site who may be in continual contact with the site's surface soil, it is necessary to estimate the pesticide concentrations that will remain in soil after excavation is completed. The soil concentrations of regulatory concern are the TTLC/STLC values published in the Title 22 regulations. It is assumed in this analysis that the TTLC soil levels and the safe-soil level estimated for the endosulfans will be the maximum soil concentrations for the pesticides remaining under residential yards.

For the purpose of estimating the excess lifetime health risk attributable to these on-site surface soil pesticides, the health risk assessment will utilize two estimates of soil pesticide levels for the remaining pesticides. For the detected pesticides with maximum on-site soil concentrations less than the TTLC (4-4'-DDE, 4-4'-DDT and dieldrin), the highest level of surface soil concentrations will be utilized in the worst-case health risk

scenario, while the site-weighted average of these compounds will be used in the best estimate (average case) analysis. For the remaining pesticides (endosulfans and endrin) health risk values will be estimated by assuming that these compounds will remain in soil at their respective TTLC safe-soil concentration levels for both the worst-case and best estimate exposure scenarios. The treatment of non-detectable soil concentrations follows a health conservative approach. All non detects are assumed to exist at one-half their respective detection limits for the compounds detected at least once at the site. DDD is not included in the health risk assessment because it was never detected in the Sunnyside Nursery surface soil samples. Table 2-3 presents the pesticide soil concentrations that will be assumed to remain in soil on-site following the completion of excavation activities for both the worst-case and best-estimate exposure scenarios.

TABLE 2-1

Organochlorine Pesticides Detected
in On-Site Surface Soil Samples

(All concentration values in mg/kg (ppm))

	N	Range of Concentration
4-4'-DDE	4	<0.005 - 0.21
4-4'-DDT	2	<0.01 - 0.64
Dieldrin	1	<0.005 - 0.041
Endosulfan I	12	<0.01 - 120.0
Endosulfan II	11	<0.005 - 44.0
Endosulfan Sulfate	14	<0.05 - 13.0
Endrin	2	<0.01 - 1.3

N = number of times detected in soil samples, includes composite and individual samples.

Range of concentration values include composite and individual soil samples.

ERS

TABLE 2-2

State of California Title 22
TTLC and STLC Values for the
Pesticides Detected in On-Site
Sunnyside Nursery Soils

	Total TTLC (mg/kg)	Soluble (STLC) (mg/l in extract)
4-4'-DDE	1.0	0.1
4-4'-DDT	1.0	0.1
Dieldrin	8.0	0.8
Endosulfan I	(3.5)*	(0.35)*
Endosulfan II	(3.5)	(0.35)
Endosulfan Sulfate	(3.5)	(0.35)
Endrin	0.2	0.02

*TTLC and STLC values for the endosulfan compounds in this table have been derived by ERS, Inc. Refer to the endosulfan toxicity and environmental profiles in Appendix for details. These values are called "safe-soil" concentration levels and have been derived using the best available methods at the present time.

TABLE 2-3

Assumed On-Site Soil Concentrations
for the Pesticides Detected
at the Sunnyside Nursery

	Worst-Case Exposure Scenario (mg/kg)	Best-Estimate Exposure Scenario (mg/kg)
4-4'-DDE	0.21	0.024
4-4'-DDT	0.64	0.044
Dieldrin	0.041	0.022
Endosulfan I*	3.5	3.5
Endosulfan II*		
Endosulfan Sulfate*		
Endrin	0.2	0.2

*The total concentrations of all the endosulfans combined (I, II and sulfate) is assumed as 3.5 mg/kg.

ERS

3.0 DOSE-RESPONSE ASSESSMENT

The dose-response assessment portion of a health risk assessment evaluates the potential toxicological effects of the compounds of concern detected in surface soil samples from the Sunnyside Nursery property. Historically, dose-response assessment has been designed to specify the quantitative relationship between the rate of chemical compound intake and the development of adverse health effects resulting from chemical exposures. The numerical estimates of toxicity required for dose-response assessment are called "health criteria".

The health criteria for chemicals are typically categorized into two broad categories -- carcinogens and noncarcinogens. Carcinogens are substances known to produce tumors in exposed animals, including mammals and/or humans. Due to their specific toxicological interactions at the cellular level, it is assumed primarily that carcinogens produce tumors only after long exposure durations, assumed to be as long as an entire lifetime of exposure in risk assessments. Chemical compounds that are noncarcinogenic are those substances that produce adverse health effects other than cancer in exposed individuals. Contrary to the carcinogens, noncarcinogenic health effects are known to occur following both short-term (acute) and long-term (chronic) exposure durations.

3.1 DERIVATION OF HEALTH CRITERIA

All of the health criteria identified in this analysis have been derived by the U.S. EPA for the specific purpose of evaluating the relative health risks posed by environmental contaminants. These health criteria are derived by several offices within EPA, including the Carcinogen Assessment Group (CAG) in Washington D.C. and the Environmental Criteria and Assessment Office (ECAO)

in Cincinnati, OH . The CAG specializes in deriving health criteria for carcinogens, while the ECAO specializes in noncarcinogens.

Health criteria for carcinogens and noncarcinogens are expressed in different mathematical terms. Criteria for the carcinogenic compounds are expressed as the potential for inducing cancer per unit of chemical exposure and are called cancer potency factors (CPFs) by the EPA. These quantitative factors are derived from the experimental results of epidemiology and/or experimental animal bioassay studies and are typically expressed in units of $(mg/kg\text{-}day)^{-1}$.

Health criteria for the noncarcinogenic compounds have evolved more dramatically at the U.S. EPA in recent years. The most recent noncarcinogenic health criteria methodology promulgated by the agency expresses these values as reference doses (RfDs) or as acceptable intake chronic (AIC) levels. Both of these criteria values are expressed in units of mg/kg-day. The RfD has been more recently developed and represents the noncarcinogenic health criteria based on the best available toxicological information to agency scientists at this time. The EPA recommends that RfD values should be used in health risk assessments whenever available (U.S. EPA, 1988a).

Compounds that are relatively nonhazardous or have been poorly studied do not have health criteria published by the regulatory agencies. These compounds are only discussed on a qualitative basis, and are generally not included in health risk assessments.

3.2 HEALTH CRITERIA FOR SUNNYSIDE NURSERY COMPOUNDS

The carcinogenic and noncarcinogenic health criteria for DDE, DDT, dieldrin, endrin and endosulfan employed in this health risk assessment are derived by the CAG and ECAO, respectively.

Currently, the EPA's Integrated Risk Information System (IRIS) database is the best information source supplying the most recently developed health criteria data for chemical compounds (U.S. EPA, 1988a). The advantage that IRIS has over the other U.S. EPA databases is that it represents the most comprehensive effort to date to compile health criteria approved by all of the EPA program offices. Agency scientists recommend that health criteria published in IRIS should supersede all other health criteria values that have been published previously.

Currently, health criteria for the compounds detected in soil at the Sunnyside Nursery are available for DDT, dieldrin and endosulfan. Verified toxicity values are not available for DDE in IRIS or in any other EPA toxicity databases, including the Public Health Risk Evaluation Database (PHRED), the database commonly used in the Superfund program, (U.S. EPA, 1988b). A noncarcinogenic health criteria for endrin is available in PHRED.

For health conservatism, this health risk assessment assumes that the carcinogenic and noncarcinogenic health criteria for DDT are equally applicable to DDE. This assumption is justified for two reasons. First, a report published by the EPA in 1986 compared the relative cancer potencies of DDT, DDE and DDD (U.S. EPA, 1986b). This study estimated a cancer potency factor of 0.34 $(mg/kg\text{-}day)^{-1}$ for both DDT and DDE even though the corresponding animal bioassay data for the two compounds were different (U.S. EPA, 1986b). This result suggests that the carcinogenic potency for DDE is similar to the DDT value (U.S. EPA, 1986b). Using the DDT cancer potency factor as a surrogate for the potential potency of a DDE/DDT soil contaminant mixture assures health conservatism. The second reason why this assumption is warranted for health risk assessment is that DDT is metabolized in the mammalian system to form DDE as one of its ultimate metabolic products. The mammalian toxicity studies completed, to date,

have not had the sensitivity to distinguish between the adverse health effects produced by the parent compound or the metabolic products. Therefore, the toxic effects identified in laboratory animals presumed to be caused by DDT may, in fact, be attributable to DDE (U.S. EPA, 1986b).

The most recently published DDT cancer potency factor for the ingestion exposure route is used in this health risk assessment for both DDT and DDE. At the present time, this value has been reviewed by EPA but has not been placed in the IRIS database. To verify its use in this analysis, Dr. Christopher DeRosa, a scientist with the EPA Office of Research and Development in Cincinnati was consulted. Dr. DeRosa verified the use of this CPF. In addition, he stated that this cancer potency factor should be equally applicable for inhalation exposures to DDE and DDT (DeRosa, 1989).

Dieldrin is also a compound recognized as a potential human carcinogen by the EPA. Its most recent cancer potency factor published by EPA is 16.0 $(mg/kg\text{-}day)^{-1}$ based on liver tumors in mice. This CPF was verified by Dr. Robert McGaughy of the Carcinogen Assessment Group and has recently been added to IRIS (McGaughy, 1989). Comparing the CFPs for these potential carcinogens indicates that dieldrin is approximately 47 times more carcinogenic than either DDE or DDT.

Aside from its potential carcinogenic effects, DDT has been shown to induce noncarcinogenic liver lesions in rats exposed to chronic oral doses as low as 5 ppm in the diet over a 27 week period. A no observable effects level (NOEL) of 1 ppm (0.05 mg/kg-day) has been estimated based on this study. The NOEL was used by the EPA, after applying a safety factor of 100 to derive an oral RfD value of 5.0E-04 mg/kg-day for DDT. This value has been published in the IRIS database as being the best available noncarcinogenic health criteria for oral exposures to DDT (U.S.

EPA, 1988a). For health conservatism, this value is used for the potential noncarcinogenic effects attributable to DDE as well.

Endosulfan is a noncarcinogenic compound that is known to produce only noncarcinogenic health effects in the form of kidney toxicity in rats exposed for long durations. The U.S. EPA has derived a reference dose health criteria of 5.0E-05 mg/kg-day based on these potential kidney toxicity effects. This criteria is included in the IRIS database (U.S. EPA, 1988a). For health conservatism, this health risk assessment assumes that endosulfan I & II and endosulfan sulfate are all equally as toxic as endosulfan.

Endrin is a chemical compound that induces noncarcinogenic health effects in the form of adverse neurological effects in exposed mammals. The EPA has derived a reference dose health criterion of 3.0E-04 mg/kg-day based on these potential adverse health effects (U.S. EPA, 1988b). This value has been verified by Mr. Bruce Means of EPA's Superfund office in Washington, D.C. (Means, 1989). Tables 3-1 and 3-2 summarize the U.S. EPA derived carcinogenic and noncarcinogenic health criteria that will be employed in this health risk assessment.

3.3 TOXICITY AND ENVIRONMENTAL FATE PROFILES

Profiles summarizing the salient toxicological and environmental fate properties of the pesticides detected in surface soils are presented in Appendix A.

TABLE 3-1

U.S. EPA Carcinogenic Health Criteria*

	Ingestion CPF $(mg/kg\text{-}day)^{-1}$
DDE/DDT	0.34
Dieldrin	16.0

* Only health criteria published by the U.S. Environmental Protection Agency are included in this analysis. All health criteria are obtained from the Integrated Risk Information System (IRIS) (U.S. EPA, 1988a), or the Superfund Public Health Risk Evaluation Database (PHRED) (U.S. EPA, 1988b). Cancer potency factors were verified by Dr. Chris DeRosa and Dr. Robert McGaughy of the EPA.

TABLE 3-2

U.S. EPA Noncarcinogenic Health Criteria*

	Ingestion Reference Dose (mg/kg-day)
DDE/DDT	5.0E-04
Endosulfan	5.0E-05
Endrin	3.0E-04

* Only health criteria published by the U.S. Environmental Protection Agency are included in this analysis. All health criteria are obtained from the Integrated Risk Information System (IRIS) (U.S. EPA, 1988a), or the Superfund Public Health Risk Evaluation Database (PHRED) (U.S. EPA, 1988b). All values were verified by Dr. Chris DeRosa.

4.0 EXPOSURE ASSESSMENT

Exposure assessment is designed to estimate three variables in a health risk assessment: 1) the concentrations of the chemicals of concern at the specific points of potential human contact, 2) the rate of media contact that results in chemical uptake, and 3) the daily amount of contaminant uptake that results during normal daily activities. For this analysis, it is assumed that the potentially exposed population will consist primarily of individuals who will be living in the houses proposed to be constructed at Sunnyside Commons. Because this health risk assessment is specified as a screening level analysis, it is most appropriate to estimate exposures to a single hypothetical individual who is assumed to receive the highest level of exposures. This person, commonly called the Maximum Exposed Individual (MEI), represents that specific individual in the study area potentially receiving the highest exposures, out of all the potentially exposed individuals. This health risk assessment assumes that the MEI in the study area is a hypothetical male who will be living at Sunnyside Commons every day of his entire lifetime, assumed to be approximately 70 years. Two exposure estimates are derived for each exposure pathway: worst-case (upper-bound) and best-estimate (average-case) exposures. Obviously, using an MEI methodology will provide exposure estimates that are unlikely to be exceeded by anyone located within the study area, which is the primary purpose of conducting a screening analysis.

The human intake pathways considered in the exposure assessment are the direct exposure pathways:

- Soil Ingestion
- Dermal Absorption

Other potential exposure pathways including the ingestion of vegetables grown in gardens are not applicable to this specific

analysis since the surface soils at the site will be excavated to a depth of 18 inches, and therefore the remaining soil concentration will be less than the State of California TTLC or 1.0E-06 risk levels for direct contact. These soil concentrations would be expected to produce insignificant exposures compared to the direct contact pathways. In addition, other exposure pathways such as fugitive dust emissions and soil volatilization exposures are also generally lower than the direct contact exposure pathways and are not included in this screening level analysis.

4.1 SOIL INGESTION EXPOSURES

Individuals having contact with surface soils will ingest soil particles incidentally during daily activities via hand-to-mouth movement. This type of activity has been documented in several child observational and empirical studies that include estimates of the amount of soil ingested daily by people of varying ages (Binder et al., 1986; Clausing et al., 1987; Hawley, 1985; Kimbrough et al., 1984; LaGoy, 1987; Lepow et al., 1975). Generally, these soil consumption rates have been estimated based on data from children only. Most investigators extrapolate these child soil ingestion rates to estimate the potential for exposure to adults.

The data from LaGoy (1987) represent the most recent compilation of literature values deriving soil ingestion rates, however, the soil ingestion data published by the California Department of Health Services (DHS) represent the most health conservative data published to date and will be used in this analysis (DHS, 1987). This regulatory agency study presents a quantitative extrapolation method for estimating soil ingestion rates for adults in a residential setting based on the upper-bound ingestion rates in children and an estimate of the rate of decline in soil ingestion for individuals between the ages of 3-

19. Using this method, DHS staff have estimated a soil consumption rate of approximately 150 mg/day (DHS, 1987). Note that this value is assumed to be an upper-bound estimate for daily soil ingestion rates at the proposed site since it has been derived specifically for a residential exposure scenario. For comparative purposes, other daily lifetime soil consumption rates have been estimated in the literature: 67 mg/day (LaGoy, 1987) and 100 mg/day -- the EPA daily soil ingestion estimate from the Superfund Exposure Assessment Manual (U.S. EPA, 1988c). These other daily soil ingestion rate estimates indicate that the 150 mg/day quantity is a worst-case estimate. The daily soil ingestion estimate of 67 mg/day from LaGoy is used to represent the best-estimate exposure estimate for soil ingestion. Both scenarios are assumed to represent exposures to the maximum exposed individual (MEI), since it is assumed that this hypothetical person will ingest these amounts of soil every day throughout his entire lifetime.

The quantity of a chemical compound that would be ingested with soil is dependent upon the mass of soil consumed per day, the chemical soil concentration, and the fraction of the ingested chemical that is absorbed into the human body. Daily soil ingestion exposures assumed to occur over an entire lifetime are calculated using the following equation:

$$\text{Average Lifetime Dose (mg/kg-day)} = \frac{\text{Soil Concentration (mg/kg)} \times \text{Soil Consumption (kg/day)} \times \text{GI}}{\text{Body Weight (70 kg)}}$$

where:

Soil concentration = the pesticide concentration goals derived in Chapter 2 in mg/kg (refer to Table 2-3);

ERS

Soil consumption = estimated lifetime soil ingestion rate assumed to be 1.5E-04 kg/day (150 mg/day) for the worst-case and 6.7E-05 kg/day (67 mg/day) for the best-estimate scenario;

GI factor = absorption rate of pesticides via ingestion, assumed to be 100% for all pesticides;

Body Weight = average lifetime body weight, assumed to be 70 kg (U.S. EPA, 1986a).

Tables 4-1 and 4-2 present the worst-case and best-estimate estimates of daily exposures to the pesticides identified at the Sunnyside Nursery site via soil ingestion.

4.2 DERMAL ABSORPTION EXPOSURES

Exposure to pollutants via dermal absorption occurs when organic chemicals adsorbed to soil come in contact with exposed skin. The rate at which soil-bound organics may cross the skin barrier depends strongly upon the amount of skin in contact with contaminated soil, the amount of soil on the skin per unit area (skin surface loading), and the chemical-specific absorption efficiency of the skin for the organic compounds of concern.

In keeping consistent with the soil ingestion section, dermal absorption data derived by the State of California DHS represents the most health conservative estimates available at the present time (DHS, 1987). DHS used the observational/experimental results of Lepow as the basis for deriving a skin surface loading rate of 0.5 mg/cm^2-day (Lepow et al., 1975). DHS also used body surface area statistics to estimate a lifetime weighted average upper-bound exposed skin surface area of 4,333 cm^2. These values can be used to derive a total soil skin loading of 2,167 mg/day as an extreme upper-bound estimate. Comparing this value with

the LaGoy estimate of 529 mg/day shows that the DHS estimate is a very health conservative value. The DHS skin soil loading rate is used in the worst-case analysis, while the LaGoy estimate is employed in the best-estimate exposure scenario.

The fraction of soil-bound pesticides that would be expected to cross the skin barrier and enter the metabolic processes of exposed individuals has not been located in the literature for any of the pesticides identified at the Sunnyside Nursery site. An absorption rate of 1% for soil-bound organics is based on the data in Clement (1988). The actual absorption rate is more likely to be similar to the 0.2% absorption rate observed for TCDD bound to soil in a soil paste mixture (Poiger and Schlatter, 1980).

The following equation estimates the upper-bound daily exposure levels of the on-site pesticides via dermal absorption exposures in the MEI:

$$\text{Average Lifetime Dose (mg/kg-d)} = \frac{C_S \times SLR \times AF \times \frac{1\ kg}{1{,}000{,}000\ mg}}{BW}$$

where:

C_S = the pesticide concentration goals discussed in Chapter 2 in mg/kg (refer to Table 2-3);

SLR = maximum skin loading rate = 2,167 mg/day for the worst-case analysis and 529 mg/kg for the best-estimate scenario;

AF = fraction of ingested soil-bound pesticides that are absorbed by the skin, assumed to be 1% for pesticides (Clement, 1988);

BW = average lifetime body weight assumed to be 70 kg (U.S. EPA, 1986a).

Tables 4-1 and 4-2 present the worst-case and best-estimate estimates of the average daily lifetime exposures to the Sunnyside Nursery soil pesticides via dermal absorption.

4.3 SUMMARY OF EXPOSURES

Tables 4-1 and 4-2 present the worst-case and best-estimate daily exposure estimates for the pesticides remaining in surface soil at the Sunnyside Nursery site.

TABLE 4-1

Summary Exposure Table

Worst-Case Exposures to the Maximum Exposed Individual (MEI)

(All units = mg/kg-day)

Exposure Route	DDE/DDT	Dieldrin	Endosulfan	Endrin
Soil Ingestion	1.8E-06	8.8E-08	7.5E-06	4.3E-07
Dermal Absorption	2.6E-07	1.3E-08	1.1E-06	6.2E-08
TOTAL DAILY EXPOSURE	2.1E-06	1.0E-07	8.6E-06	4.9E-07

ERS

TABLE 4-2

Summary Exposure Table

Best-Estimate Exposures to the
Maximum Exposed Individual (MEI)

(All units = mg/kg-day)

Exposure Route	DDE/DDT	Dieldrin	Endosulfan	Endrin
Soil Ingestion	6.5E-08	2.1E-08	3.4E-06	1.9E-07
Dermal Absorption	5.1E-09	1.7E-09	2.7E-07	1.5E-08
TOTAL DAILY EXPOSURE	7.0E-08	2.3E-08	3.7E-06	2.1E-07

ERS

5.0 RISK CHARACTERIZATION

The health risk assessment is completed in risk characterization by calculating quantitative estimates of potential health risk in the potentially exposed population. Following the proposed screening level methodology, the worst-case (upper-bound) and best-estimate (average-case) lifetime estimates of health risk are derived for a hypothetical maximum exposed individual (MEI) assumed to have access to the Sunnyside Nursery site every day throughout his entire lifetime.

5.1 METHODOLOGY OF RISK CHARACTERIZATION

The health risk assessment methodology designated in this analysis provides conservative estimates of potential health risk. Exposures that would be expected to occur in a potentially exposed population are generally overestimated by using health conservative assumptions throughout the entire analysis. For example, it is assumed that the maximum exposed individual (MEI) in this analysis will be exposed to the soil-bound pesticides every day for an entire lifetime. In addition, it is assumed that soil pesticide concentrations will remain constant over the entire lifetime of the exposed individual, even though no sources of nursery pesticides will remain on-site during the exposure period. The compounding effect of using health conservative assumptions results in estimating health risk values that are expected to be upper-bound estimates of potential risk. Based on the health conservative nature of this methodology, it is highly probable that the actual health risk to the exposed population, who will be living in the houses proposed for construction on the site, as a whole, is lower than the numerical estimates estimated in this analysis. Likewise, the screening level HRA methodology is unlikely to predict risk estimates that are less than the actual risks to the potentially exposed population.

5.1.1 Carcinogenic Risk Characterization

Quantitative estimates of carcinogenic health risk are a function of both chemical exposure and the inherent toxicity of the particular chemicals of concern. The exposure levels, derived in units of mg/kg of body weight per day (mg/kg/day), are presented in Chapter 4 for the two modeled exposure pathways, soil ingestion and dermal absorption. The cancer potency factors (CPFs) presented in Chapter 3 are expressed in units of $(mg/kg/day)^{-1}$. Multiplying the exposure estimates by the CPFs results in calculating unitless estimates of cancer risk attributable to exposure to the pesticides in soil at the Sunnyside Nursery property.

$$\text{Lifetime cancer risk} = \begin{matrix}\text{total daily dose} \\ \text{(mg/kg-day)}\end{matrix} \times \begin{matrix}\text{cancer potency factor} \\ (\text{mg/kg-day})^{-1}\end{matrix}$$

Cancer risks attributable to exposure to the on-site pesticides are calculated individually for each chemical and each exposure pathway. The total lifetime cancer risk is estimated by summing the cancer risks for each compound and exposure route.

5.1.2 Noncarcinogenic Risk Characterization

The estimation of noncarcinogenic health risks proceeds by comparing the exposures levels for the noncarcinogens in this analysis, DDE/DDT, endosulfan and endrin, with their appropriate health criteria. Noncarcinogenic criteria identified in the current EPA literature are published as reference doses (RfDs). These values, expressed in units of mg/kg/day, are derived from animal bioassay and human epidemiology studies. The degree of noncarcinogenic health risk is estimated by comparing the health criteria values with the estimated exposure levels. Typically this comparison is expressed as a simple ratio.

Noncancer Health Hazard Index	=	daily exposure (mg/kg-day)	/	RfD (mg/kg-day)

In situations where the estimated exposure levels are greater than the noncarcinogenic health criteria (i.e. the ratio (exposure level / RfD) is greater than unity) a potential for the occurrence of noncarcinogenic adverse health effects may exist in the exposed population. The converse states that when the total noncancer risks are less than unity, it is presumed that noncancer health effects are not expected to occur in the potentially exposed population. Since both the exposure and the RfDs are derived in the same units, mg/kg/day, the resulting noncarcinogenic risk estimate is unitless.

5.2 RISK CHARACTERIZATION RESULTS

The cancer risks estimated for the maximally exposed individual (MEI) potentially exposed to soil contaminants for an entire lifetime are presented in Tables 5-1 and 5-2. The estimated worst-case (upper-bound) lifetime cancer risk to the MEI in this analysis is 2.3E-06, or approximately 2 cases of cancer per million exposed individuals. The best-estimate lifetime cancer risk to the MEI is estimated as 3.9E-07, or approximately 4 cases of cancer per 10 million exposed individuals. The best-estimate level of cancer risk is regarded as "de minimus" by regulatory agencies, especially considering the many health conservative assumptions provided throughout the analysis.

The noncancer health risks are presented in Tables 5-3 and 5-4. All exposures to the noncarcinogens are less than the EPA published criteria indicating little probability of noncarcinogenic health effects in the potentially exposed population.

TABLE 5-1

Lifetime Cancer Risks

Worst-Case Risk Estimates to the MEI

Exposure Route	DDE/DDT	DIELDRIN	Total Risk
Soil Ingestion	6.1E-07	1.4E-06	
	6.1/10,000,000		
Dermal Absorption	8.8E-08	2.1E-07	
	8.8/100,000,000		
TOTAL CANCER RISK	7.0E-07	1.6E-06	2.3E-06

Note: "E-" notation refers to powers of 10; e.g. 8.8E-08 = 8.8×10^{-8}.

ERS

TABLE 5-2

Lifetime Cancer Risks

Best-Estimate Risk Estimates to the MEI

Exposure Route	DDE/DDT	DIELDRIN	Total Risk
Soil Ingestion	2.2E-08	3.4E-07	
Dermal Absorption	1.7E-09	2.7E-08	
TOTAL CANCER RISK	2.4E-08	3.7E-07	3.9E-07

Note: "E-" notation refers to powers of 10; e.g. 8.8E-08 = 8.8×10^{-8}.

ERS

TABLE 5-3

Lifetime Noncancer Health Hazard Index (HHI)*

Worst-Case HHI to the MEI

Exposure Route	DDE/DDT	ENDOSULFAN	ENDRIN
Soil Ingestion	3.6E-03	1.5E-01	1.4E-03
Dermal Absorption	5.2E-04	2.2E-02	2.1E-04
TOTAL NONCANCER HEALTH HAZARD INDEX	4.1E-03	1.7E-01	1.6E-03
SUM	1.8E-01		

* Noncarcinogenic Risk = Exposure (mg/kg-day) / RfD (mg/kg-day)

Note: "E-" notation refers to powers of 10; e.g. 5.2E-04 = 5.2×10^{-4}.

ERS

TABLE 5-4

Lifetime Noncancer Health Hazard Index (HHI)*

Best-Estimate HHI to the MEI

Exposure Route	**DDE/DDT**	**ENDOSULFAN**	**ENDRIN**
Soil Ingestion	1.3E-04	6.8E-03	6.3E-04
Dermal Absorption	1.0E-05	5.4E-04	5.0E-05
TOTAL NONCANCER HEALTH HAZARD INDEX	4.8E-04	7.3E-03	7.9E-02
SUM	8.7E-02		

* Noncarcinogenic Risk = Exposure (mg/kg-day) / RfD (mg/kg-day)

Note: "E-" notation refers to powers of 10; e.g. 5.2E-04 = 5.2×10^{-4}.

ERS

6.0 REFERENCES

Binder, S., D. Sokal and P. Maughan, 1986: Estimating Soil Ingestion: The Use of Tracer Elements in Estimating the Amount of Soil Ingested by Young Children, Archives of Environmental Health 41:341-345.

Clausing, J., B. Brunekreef and J. Van Wijnen, 1987: A Method for Estimating Soil Ingestion by Children, Int. Arch. Occup. Env. Health 59:73-82.

Clement Associates, Inc., 1988: Multi-Pathway Health Risk Assessment Input Parameters Guidance Document. Prepared for: South Coast Air Quality Management District, El Monte, California.

DHS, 1987. The Development of Applied Action Levels for Soil Contact: A Scenario for the Exposure of Humans to Soil in a Residential Setting-Final Draft, prepared by the California Department of Health Services, Toxic Substances Control Division.

DHS, 1986. The California Site Mitigation Decision Tree Manual, prepared by the California Department of Health Services, Toxic Substances Control Division.

DeRosa, 1989: Personal Communication with Dr. Chris DeRosa, U.S. Environmental Protection Agency, Office of Research and Development, Cincinnati, OH.

Federal Register, 1986. Guidelines for Carcinogen Risk Assessment. 51 Federal Register 33992-34003.

Hawley, J., 1985: Assessment of Health Risk from Exposure to Contaminated Soil, Risk Analysis 5(4):289-302.

Kimbrough, R., H. Falk, P. Stehr and G. Fries, 1984: Health Implications of 2,3,7,8-Tetrachlorodibenzodioxin (TCDD) Contamination of Residential Soil, Journal of Toxicology and Environmental Health 14:47-93.

LaGoy, P., 1987: Estimated Soil Ingestion Rates for Use in Risk Assessment, Risk Analysis 7:355-359.

Lepow, M.; Bruckman, L.; Rubino, R.; Markowitz, S.; Gillette, M.; Kapish, J. 1975: Investigations into Sources of Lead in the Environment of Urban Children, Environmental Research 10:415-426.

McGaughy, 1989: Personal Communication with Dr. Robert McGaughy, U.S. Environmental Protection Agency, Carcinogen Assessment Group, Washington, D.C.

Murphy, S., 1986: Toxic Effects of Pesticides, Casarett and Doull's Toxicology - The Basic Science of Poisons, 3rd ed., Chpt. 18, p. 132-133.

Poiger, H., and Schlatter, C. 1980: Influence of Solvents and Adsorbents on Dermal and Intestinal Absorption of TCDD, Food and Cosmetic Toxicology, 18:477-481.

Terratech 1989a: Phase I Environmental/Toxics Investigation, Sunnyside Nursery, Hayward, California, prepared for the Plymouth Group, February, 1989.

Terratech, 1989b: Closure Plan for Mitigating Agricultural Chemical Hazards, Sunnyside Nursery, Alameda County, California, prepared for the Plymouth Group, June 16, 1989.

U.S. EPA, 1988a: Integrated Risk Information System (IRIS). U.S. Environmental Protection Agency, Washington, D.C.

U.S. EPA, 1988b: Superfund Public Health Risk Evaluation Database (PHRED). U.S. Environmental Protection Agency, Superfund Office, Washington, D.C. (September 1988 version).

U.S. EPA, 1988c: Superfund Exposure Assessment Manual. Pre-Publication Edition, EPA/540/1-88-001.

U.S. EPA, 1986a: Superfund Public Health Evaluation Manual, U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C., EPA/540/1-86/060.

U.S. EPA, 1986b: The Assessment of the Carcinogenicity of Dicofol (Kelthane), DDT, DDE, and DDD (TDE). Prepared by J. Holder, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C., EPA/600/6-86/001.

APPENDIX A

This appendix presents the toxicological and environmental fate properties of the organochlorine pesticides detected in surface soils at the Sunnyside Nursery property. Individual profiles have been prepared for DDE/DDT, dieldrin, endosulfan I & II and endosulfan sulfate, and endrin. The profiles focus on the fate properties which affect the mobility of these pesticides in the environment. The toxicological properties of concern include mammalian toxicokinetics, their known chronic and acute health effects, genotoxic potential, reproductive health effects, and health criteria, both carcinogenic and noncarcinogenic, that have been published by the U.S. EPA for these pesticides.

ERS

Introduction

DDT and its degradation product DDE are organochlorine pesticides of similar chemical composition which have been used extensively all over the world for both agriculture and malaria control. Estimates indicate that more than 7 billion pounds have been utilized world-wide since 1940. Due to their relatively high stabilities and high lipid-water partitioning, DDT and DDE persist in the environment, so that even though most uses of DDT were banned in 1963, many soils contain residual levels to this day.

Persuasive evidence exists demonstrating that DDT and DDE build up in natural food chains via biologic accumulation in ecosystems (Dustman and Stickel, 1969; Edwards, 1970). DDT and DDE may adversely affect organisms at the top of these food chains by accumulating gradually in the lower organisms that constitute their food sources. According to field and laboratory studies, exposure to DDT or its metabolites hinders reproductive success in certain avian species (Klaassen et al., 1986). Furthermore, these pesticides are acutely toxic to fish and lower aquatic organisms (Pimental, 1971).

Physical/chemical and environmental fate properties

The major environmental and biological degradation product of DDT is DDE. In the degradation process, DDT goes through a dehydrochlorination reaction to form DDE, a compound much less toxic to insects and higher animals but of approximately equal solubility in water and high lipid-water partitioning capacity (NRC, 1977). The prominent chemical/physical properties of both DDT and DDE are presented below.

TABLE A-1

Physical/Chemical Properties of DDE/DDT

	DDE	DDT
Molecular weight (g/mole)	318.02	354.48
Henry's Law Constant ($atm\text{-}m^3/mole$)	6.80E-05	5.13E-04
Vapor Pressure (mm Hg)	6.50E-06	5.50E-06
Solubility (H_2O) (mg/l)	4.00E-02	5.00E-03
Log octanol/water partition coefficient	7.00	6.19
K_{OC}	4.4E+06	2.43E+05
Diffusion coefficient (cm^2/s)	0.048	0.046

Source: U.S. EPA, 1986a.

The environmental persistence of the chlorinated hydrocarbons is determined by their physicochemical boundaries, including their lipid solubility, low water solubility, chemical stability and absorption/desorption process. Because DDE is neither biologically nor environmentally degradable, it is more persistent in the environment than DDT (Gish, 1970; Kenaga, 1972). DDE is the prime DDT residue stored in living tissues, and it may increase in relative concentration in each trophic level (Woodwell et al., 1967). Distinctly different from DDT in environmental media, DDE

ERS

may break down as a result of direct photolysis to produce hydrochloric acid and carbon dioxide. This reaction occurs primarily in aqueous systems and the importance of this process in soil remains unclear at the present time.

The relative amount of soil organic matter largely determines the rate of absorption of hydrophobic chlorinated hydrocarbons such as DDT and DDE. In general, once absorbed, these compounds do not easily desorb from soils (Menzer and Nelson, 1986). This significant environmental fate property indicates that these compounds leach and disperse very slowly in most organic type soils. Ecological evidence suggests that the conveying of these compounds into the hydrosphere from contaminated soils occurs more readily via the erosion of soil particles or sediment rather than via desorption and dissolution (Menzer and Nelson, 1986).

Vaporization of these compounds from soil and water into air comprises another environmental fate migration. Temperature, the nature of the soil particles, total soil water content, water solubility and the degree of absorption all regulate the rate of vaporization from soil (Menzer and Nelson, 1986). The presence of a high level of organic material in the soil decreases the overall volatility as the compound is more firmly adsorbed to soil particles. Volatilization of these compounds from aqueous systems may be significant under certain conditions.

Bioaccumulation

The processes which result in the bioaccumulation of DDE and DDT are complex. The physicochemical properties of the chlorinated hydrocarbons such as lipid solubility, low water solubility and chemical stability seem to be of most significance in their bioaccumulation. The well-documented bioaccumulation effects of DDE and DDT are more evident at the highest levels of the food chain. The bioaccumulation ratio, the relationship of the

organism residue to the environmental residue levels, is higher in aquatic ecosystems than in terrestrial ecosystems for these compounds (Menzer and Nelson, 1986). The residue ratios of DDT and DDE vary throughout the environment (Fries, 1972; Kenaga, 1972).

The adipose tissue of both humans and animals is particularly susceptible to DDT and DDE bioconcentration due to the compounds' high lipid-water partition coefficients. Humans store DDT in the fat tissue at approximately ten times the intake concentration (NRC, 1977). DDE and DDT concentrations increase in relative amounts with each increase in trophic level (NRC, 1977). DDE exists in human fat tissue at approximately 70% of the DDE and DDT total concentration (Durham, 1969). The high lipid-water partition of DDT produces substantial fat accumulation. This fat storage occurs at approximately 20 times the dietary intake at equilibrium conditions (NRC, 1977). After consuming 1 ppm of DDT for 15 weeks, rats stored the pesticide in their fat at rates of 13 ppm in males and 18 ppm in females. The corresponding values for 50 ppm exposures were 284 and 588 ppm (Laug et al., 1950).

Toxicokinetics

Diffusion-controlled reaction rates remove lipophilic compounds such as DDT from metabolic environments. DDT, a highly lipophilic pesticide, is removed at exceptionally slow rates, with a $t_{1/2}$ of 300 minutes in the rat. A comparison of the pulmonary absorption rate with the physical properties of the compound, such as molecular weight and octanol/water partition coefficient, suggests that partitioning into the lipid of the lung membrane is the rate-determining factor for inhalation exposures (Klaassen et al., 1986).

Direct dietary exposure provides a ready means of absorption of DDE and DDT into the human body (WHO, 1979). Based on these

studies, approximately 100% of the ingested DDE/DDT compounds are absorbed. The human body usually retains the residues of these compounds in proportion to the percentage of fat in the various organ systems. The biological half-life for these compounds is long: DDT = 10 - 20 years, DDE = 60 - 70 years. Once exposed, the human body retains these residues for long periods. Further exposures add to the already existing body burden (U.S. EPA, 1986b).

Qualitative Description of Health Effects

Carcinogenic Potential

Convincing evidence exists that suggests that DDE and DDT are carcinogens in mice inducing primarily liver tumors, but also lung carcinomas and lymphomas (IARC, 1974; U.S. EPA, 1986b). Evidence is lacking from other studies since DDT and DDE have been tested several times under widely different conditions and have not proven to be carcinogenic in other experimental species. Although the pesticide has been in extensive use for 40 years, no proof exists which confirms a potential cancer risk in humans either in the general public, where trace amounts of DDT and DDE have been found in body fat, or individuals exposed to higher levels during production or spraying (Hayes, 1982; Klaassen et al., 1986). The potential carcinogenicity for humans resulting from DDT and DDE remains unclear, due to both the lack of relevant human data and the difficulties in associating test animal tumors to tumors in man (U.S. EPA, 1986b).

Genotoxic Potential

Because DDT has been tested thoroughly for genotoxicity with both positive and negative results, it is difficult at the present time to determine unequivocal genotoxicity for DDT and its metabolites (U.S. EPA, 1986b). For example, the results of the

Salmonella/microsome test did not show DDT to be mutagenic, but the pesticide caused chromosomal damage in mouse lymphoma cells (L5178Y cells) and in Chinese V79 hamster cells (ICPEMC, 1984).

Reproductive Effects

DDT is a known reproductive toxin that reduces fertility, stunts growth of offspring and increases fetal mortality. It is well-known that DDT in the environment has significantly decreased the populations of numerous species of water birds, raptors, and many other wild birds. A substantial decline in the reproductive capabilities of many fish-eating birds and their resultant population decrease is also linked to DDT exposure. Direct evidence of adverse reproductive effects of DDT in humans has yet to be established. In addition, evidence linking DDT exposure to teratogenic effects in exposed laboratory animals is lacking (Ware and Good, 1967).

Acute/chronic effects

Many detrimental noncarcinogenic health effects develop from chronic DDT exposure, and these effects are particularly numerous in the central nervous system (CNS) and in the liver. DDT and DDE exposure induces behavioral effects in the CNS such as decreased aggression and decreased conditional reflexes. DDT exposure harms the mammalian liver by causing hypertrophy of the parenchymal cells and by increasing fat deposition. Seizures result from chronic exposure to lower doses or acute exposure to large doses. The oral LD_{50} for DDT varies from between 113 mg/kg and 450 mg/kg for the rat and is generally higher for most experimental animals (Hayes, 1963; Pimental, 1971).

Quantitative Description of Health Effects

Although no definitive examples exist which attribute any human

fatalities to the ingestion of DDT, a dosage of 10 mg/kg has caused illness in some but not all subjects. Convulsions have frequently occurred at dosages of 16 mg/kg or higher (NRC, 1977).

Because DDT/DDE and their metabolites contribute to carcinogenic activity in laboratory animals, the EPA has classified them as Group B2 carcinogens (U.S. EPA, 1986b). Cancer potency factors for both DDE and DDT have been estimated as 0.34 $(mg/kg\ day)^{-1}$. At present, the precise level of carcinogenicity of DDT to man is uncertain, since the appropriate epidemiologic studies do not exist. The EPA's classification indicates that there is sufficient evidence of carcinogenicity in animals to indicate the likelihood of potential carcinogenic effects in man.

The California Department of Health Services has established a TTLC level of 1.0 mg/kg and a STLC value of 0.1 (mg/l) for DDT and DDE.

REFERENCES for DDE/DDT

Durham, W., 1969: Body burden pesticides in man. Ann. N.Y. Acad. Sci. 160:183-195.

Dustman, E., and Stickel, L., 1969: The occurrence and significance of pesticide residues in wild animals. Ann. N.Y. Acad. Sci. 160:162-72.

Edwards, C., 1970: Persistent Pesticides in the Environment. CRC Monoscience Series, Chemical Rubber Co., Cleveland.

Fries, G., 1972: Degradation of Chlorinated Hydrocarbons under Anaerobic Conditions, p. 256-270 in American Chemical Society. Fate of Organic Pesticides in the Aquatic Environment, Washington, D.C.

Gish, C., 1970: Organochlorine Insecticide Residues in Soils and Soil Invertebrates from Agricultural Lands. Pesticides Monitoring Journal 3:241-252.

Hayes, W., Jr., 1982: Pesticides studied in man. Baltimore, MD: Williams & Wilkins Co., pp.234-247.

Hayes, W., Jr., 1963: Clinical Handbook of Economic Poisons. U.S. Department of Health, Education and Welfare, Public Health Service Communicable Disease Center, Atlanta, GA. Public Health Service Publication No. 476.

IARC, 1974: Monographs on the evaluation of carcinogenic risk of chemicals to man: Vol. 5 Some organochlorine pesticides. International Agency for Research on Cancer, 5:83-124.

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

Kenaga, E., 1972: Guidance for Environmental Study of Pesticides: Determination of Bioconcentration Potential, p.72-113 in Gunther, F.A. and J.D. Gunther (eds.). 1972. Residue Reviews, Residues of Pesticides and Other Contaminants in the Total Environment, Vol. 44, Springer Verlag, N.Y.; 192 pp.

Klaassen, C., Amdur, M., Doull, J., 1986: Casarett and Doull's Toxicology - The Basic Science of Poisons, 3rd ed., Macmillan Publishing House, N.Y., pp.543-547.

ERS

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

Menzer, R. and J. Nelson, 1986: Water and Soil Pollutants, Casarett and Doull's Toxicology - The Basic Science of Poisons, 3rd ed., Chpt. 26, p. 830-832.

NRC, 1977: Drinking Water and Health, Volume 1. National Academy of Sciences, Washington, D.C., pp.556-573.

Pimental, D., 1971: Ecologic Effects of Pesticides on Non-Target Species. Report to Executive Office of the President, Office of Science and Technology, U.S. Government Printing Office, Washington, D.C. pp.20-23.

U.S. EPA, 1986a: Superfund Public Health Evaluation Manual, U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C., EPA/540/1-86/060.

U.S. EPA, 1986b: The Assessment of the Carcinogenicity of Dicofol (Kelthane), DDT, DDE, and DDD (TDE). Prepared by J. Holder, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C., EPA/600/6-86/001.

Ware, G. and E. Good, 1967: Effects of insecticides on reproduction in the laboratory mouse II. Mirex, Telodrin, and DDT. Toxicol. Appl. Pharmacol. 10:54-61.

WHO, 1979: DDT and its derivatives, Environmental Health Criteria No. 9. World Health Organization, Geneva.

Woodwell, G., Wurster, C., Isaacson, P., 1967: DDT residues in an east coast estuary: A case of biological concentration of a persistent insecticide. Science 156:821-824.

Introduction

Dieldrin, an organochlorine pesticide, is structurally related to aldrin and is an aldrin breakdown product both via mammalian metabolic reactions and a variety of environmental conditions. Dieldrin was used extensively in the 1960s and early 1970s for a variety of pesticidal uses. Most uses were banned in the U.S. in 1974 by the U.S. EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Today, dieldrin is used in low volumes primarily as an insecticide for termite control.

Physical/chemical and environmental fate properties

Dieldrin is the most environmentally stable insecticide among the cyclodienes. The application of either aldrin or dieldrin to soil would result in the formation and environmental persistence of dieldrin in soil. In the degradation process, aldrin undergoes an epoxidation reaction to form dieldrin (U.S. EPA, 1987). This reaction is favored under a wide variety of environmental conditions.

Some of the chemical/physical properties of dieldrin are similar to those of aldrin, since they are structurally related. The salient physical/chemical properties for dieldrin relevant to mammalian toxicity and potential human exposure are presented below.

TABLE A-2

Physical/Chemical Properties of Dieldrin

Molecular weight (g/mole)	380.93
Henry's Law Constant ($atm\text{-}m^3/mole$)	4.58E-07
Vapor Pressure (mm Hg)	1.78E-07
Solubility (H_2O) (mg/l)	1.95E-01
Log octanol/water partition coefficient	3.50
K_{oc}	1700
Diffusion coefficient (cm^2/s)	0.044

Source: U.S. EPA, 1986

Environmental Fate and Persistence

Dieldrin is one of the most stable and persistent of the chlorinated hydrocarbons in both soil and water. Volatilization and photolysis reactions that forms the more environmentally stable compound, photodieldrin, are the dominant transport and fate processes of soil-bound and aqueous dieldrin. Adsorption to sediments, especially organic materials, and bioaccumulation are also important processes that remove dieldrin from water (U.S. EPA, 1979). Biotransformation and biodegradation reactions involving dieldrin occur very slowly, but may be the ultimate fate processes in sediment/soil. The half-life for dieldrin is soil ranges from approximately 7-25 years.

ERS

Before the banning of dieldrin as a pesticide, inhalation and skin adsorption were viable routes of exposure. During the period when there was extensive use of this pesticide, the potential for inhalation exposure was greatest for pesticide applicators or residents in buildings where termite treatment occurred. Atmospheric pollution was fairly common also. In 1976, more than 85% of atmospheric air samples tested by the EPA contained dieldrin or aldrin with levels as high as 2.8 ng/m^3 resulting in an intake of up to 0.098 ug/day (U.S. EPA, 1974).

Bioaccumulation

Bioaccumulation ratios compare tissue concentrations in exposed organisms to environmental concentrations. Dieldrin is a stable, highly persistent compound lipophilic that accumulates in the mammalian food chain (U.S, EPA, 1987). As a result, dieldrin concentrations in mammalian tissues are generally higher than other pesticides. Due to dieldrin's high lipid:water partition coefficient, this compound tends to accumulate in the adipose tissue of both humans and animals. The EPA estimated that 99.5% of all human beings in the United States had dieldrin residues in their tissues (U.S. EPA, 1980). These residues levels are believed to be due to contamination of foods of animal origin.

It has been estimated that dieldrin has one of the longest half-lives of the chlorinated hydrocarbons (U.S. EPA, 1979). In water at a depth of 1 meter, dieldrin has a half-life of approximately 723 days, compared to 3.5 days for DDT for instance (MacKay and Wolkoff, 1973). This long half-life enhances the potential hazard of dieldrin. The long soil half-life ranging from 7-25 years further increases dieldrin's potential for inducing potential adverse health effects.

Toxicokinetics

The primary routes of exposure for dieldrin include inhalation, ingestion and dermal absorption. The absorption of dieldrin following exposure of any of these routes has not been well characterized by experimental studies. The major source of dieldrin exposure is believed to be the ingestion of contaminated food (U.S. EPA, 1987). Since absorption rates have not been identified in the literature, this health risk assessment assumes that dieldrin will be absorbed entirely (100%) via ingestion and inhalation. The dermal absorption rate of absorption of soil-bound dieldrin is assumed as 1%.

There have been several studies on the tissue distribution of dieldrin following ingestion exposures. One study followed the distribution of [^{14}C]-dieldrin in rats. Upon first entering the body, dieldrin localizes in the liver and both dieldrin and its metabolites redistribute to other mammalian tissues (Hayes, 1974). The redistribution of radioactively labelled compounds suggests that accumulation and storage of unchanged dieldrin in body fats (Iatropoulos et al., 1975). No human studies have been identified that focus on the absorption and metabolic effects of dieldrin via inhalation.

Qualitative Description of Health Effects

Carcinogenic Potential

Dieldrin is a known animal carcinogen producing primarily liver tumors (hepatomas) in mice based on a two year feeding study (Walker et al., 1973). A positive dose-response relationship was observed in the three dose groups. Further dietary studies using rats and dogs have not shown dieldrin to be carcinogenic (U.S. EPA, 1987). No epidemiologic studies have been completed for a cohort exposed to dieldrin.

Genotoxic Potential

Dieldrin was not mutagenic in the Salmonella/microsome test (McCann et al., 1975). Three E. Coli reverse mutation survey studies with dieldrin further support the conclusion that the chemical is not mutagenic in procaryotes (U.S. EPA, 1987). All other assays reporting that dieldrin adversely affects genetic material were either flawed by inadequate study designs or showed greatest activity at cytotoxic doses, thereby confounding results.

Reproductive Effects

Evidence of the substantial effects of dieldrin on animal reproduction was presented at the 1974 dieldrin hearings conducted by the U.S. EPA. An example is a study in which raccoons were fed dieldrin at 2 and 6 ppm in their diet. The animals produced 20.0 and 20.2%, respectively, than did untreated controls (NRC, 1977). In another study, raccoons fed dieldrin at 2 ppm had abnormal estrous cycle, reduced ovulation rate, reduction of pregnancy to 25-30% of that in controls, increased resorption of embryos, and reduction in litter size.

In studies by Ottolenghi et al. (1974) using hamsters and mice, single oral doses of dieldrin at approximately one-half the respective LD_{50} doses were given on days 3, 7, or 9 of gestation in the hamsters and on day 9 of gestation in the mice. A significant number of defects were produced in both species (U.S. EPA, 1987). Evidence regarding potential reproductive effects in humans has not been reported, however, it is presumed that humans would be adverse affected by exposure to dieldrin.

Acute/Chronic Effects

Dieldrin is highly toxic by ingestion, inhalation and dermal

absorption. Hayes (1982) reported ingested dosages of about 10 mg/kg of dieldrin that resulted in fatalities. The lethal oral dose (LD_{50}) of aldrin/dieldrin for most species ranges from 3 to 100 mg/kg body weight. This includes mice, rats, hamsters, guinea pigs, dogs, rabbits, monkeys, and humans (Hodge et al., 1967; RTECS, 1985). For dieldrin, the signs of acute toxicity are primarily related to the central nervous system (CNS). The acute effects upon the CNS include intoxication, hyperactivity, hypersensitivity to auditory and tactile stimuli, loss of appetite (anorexia) and body weight, hyperexcitability, tremors, depression, convulsions, coma, and ultimate death (Hodge et al., 1967).

At low concentrations, dieldrin is acutely toxic to freshwater species. Tests in fish showed that the LC_{50} toxicity values ranged from 1 to 46 ug/liter for a variety of species. Final acute values for freshwater species were determined to be 2.5 ug/liter.

The results of dieldrin chronic feeding studies to laboratory animals is extraordinarily severe, and true no-adverse-effect dosages have never been determined since even the lowest dose group exhibited adverse health effects (Walker et al., 1972). Dieldrin has also been implicated in large-scale bird and mammal kills in treated areas. Experimental feeding studies have shown that the chemical is quite toxic to terrestrial wildlife and domestic animals in low levels.

Quantitative Description of Health Effects

Dieldrin was considered "positive" for tumor induction on the basis of tests conducted adequately in one or more species. In a review by Tomatis (1976) of the program on the evaluation of the carcinogenic risk of chemicals to humans of the International Agency for Research on Cancer (IARC), dieldrin was determined to

be carcinogenic in experimental animals only.

The EPA's Carcinogen Assessment Group has published a cancer potency factor of 16 $(mg/kg\text{-}day)^{-1}$ for dieldrin (U.S. EPA, 1988). Dieldrin is rated as a B2 "probable human" carcinogen by the EPA.

The California Department of Health Services has established a TTLC level of 8.0 mg/kg and a STLC value of 0.8 (mg/l) for dieldrin.

REFERENCES for DIELDRIN

Hayes, W., Jr., 1982: Pesticides Studied in Man. Baltimore, MD: Williams & Wilkins Co., pp. 234-247.

Hayes, W., Jr., 1974: Distribution of Dieldrin Following a Single Oral Dose. Toxicol. Appl. Pharmacol. 28:485-492.

Hodge, H., A. Boyce, W. Diechmann, and H. Kraybill, 1967: Toxicology and No-Effect Levels of Aldrin and Dieldrin. Toxicol. Appl. Pharmacol. 10:613-675.

Iatropoulos, M., A. Milling, W. Muller, G. Nohynek, K. Rozman, F. Coulston, and F. Korte, 1975: Absorption, Transport and Organotropism of Dichlorobiphenyl (DCB), Dieldrin, and Hexachlorobenzene (HCB) in Rats. Environmental Research 10:384-389.

MacKay, D. and A. Wolkoff, 1973: Rate of Evaporation of Low-Solubility Contaminants from Water Bodies to the Atmosphere. Environmental Science & Technology 7:611.

McCann, J., E. Choi, B.N. Ames, 1975: Detections of Carcinogens as Mutagens in the Salmonella/Microsome Test: Assay of 300 Chemicals. Proc. Nat. Acad. Sci. 72(12):5135-5139.

NRC, 1977: Drinking Water and Health, Volume 1. National Research Council, National Academy of Sciences, Washington, D.C.

Ottolenghi, A., J. Haseman and F. Suggs, 1974: Teratogenic Effects of Aldrin, Dieldrin and Endrin in Hamsters and Mice. Teratology 9:11-16.

RTECS, 1985: Registry of toxic effects of chemical substances, Database, National Institute for Occupational Safety and Health, Cincinnati, OH.

Tomatis, L., 1976: The IARC Program on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Ann. N.Y. Acad. Sci., 271:396-409.

U.S. EPA, 1988: Personal Communication with Dr. Robert McGaughy, U.S. Environmental Protection Agency, Carcinogen Assessment Group Washington, D.C.

U.S. EPA, 1987: Carcinogenicity Assessment of Aldrin and Dieldrin. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, , Washington, D.C., EPA/600/6-87/006.

ERS

U.S. EPA, 1986: Superfund Public Health Evaluation Manual, U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, EPA/540/1-86/060.

U.S. EPA, 1980: Ambient Water Quality Criteria for Aldrin/Dieldrin. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH. EPA-600/8-84-001F.

U.S. EPA, 1979: Water-Related Environmental Fate of 129 Priority Pollutants, Volume I, U.S. Environmental Protection Agency, Office of Water Planning and Standards, EPA-440/4-79-029a.

U.S. EPA, 1974: Respondent's First Proposed Findings of Fact on Environmental Effects of Aldrin and Dieldrin. U.S. Environmental Protection Agency, Office of Pesticide Programs, Washington, D.C.

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

ENDOSULFAN I & II & ENDOSULFAN SULFATE

Introduction

Endosulfan, which consists of two stereoisomers (endosulfan I (70-75%) & II (30-35%) also called Alpha & Beta endosulfan) is a broad-spectrum insecticide that belongs to the cyclodiene organochlorine chemical family. Endosulfan sulfate is the primary environmental breakdown product of endosulfan I & II. Endosulfan is used predominantly as a foliar insecticide to control several insect species, including beetles, aphids, and leafhoppers on a variety of fruit, nonfood crops, nuts, and vegetables. Although it is still used on a variety of crops, its total volume usage has diminished in recent years.

Physical/chemical and environmental fate properties

Both endosulfan isomers are highly absorbent to soils. The beta isomer adsorbs and concurrently remains stable in soils longer than the more rapidly degrading alpha isomer. Relative adsorbancies for specific soil types are currently not known (WHO, 1984). It is expected that because of their persistent binding and low water solubility, both endosulfan isomers and their primary metabolite, endosulfan sulfate, will leach only very slowly into most groundwater sources (WHO, 1984).

Endosulfan isomers in soil degrade primarily to form endosulfan sulfate, but may also form endosulfan diol and endosulfan lactone under many conditions. Experimental studies show that fungi may produce endosulfan sulfate as a metabolite, while a smaller percentage, approximately 10%, of that amount of endosulfan diol is formed from these same mechanisms (U.S. EPA, 1979). Predominant biodegradation mechanistic theory suggests that the beta isomer is isomerized to produce the alpha isomer which is subsequently degraded under many environmental conditions. This mechanism

ERS

TABLE A-3

Physical/Chemical Properties of Endosulfan

	Endosulfan I	Endosulfan II
Molecular weight (g/mole)	407	407
Henry's Law constant ($atm\text{-}m^3/mole$)	6.7E-07	6.7E-07
Vapor Pressure (mm Hg)	2.4E-07	4.9E-07
Solubility (H_2O) (mg/l)	0.53	0.28
Log octanol/water partition coefficient	3.55	3.62
K_{OC}	2,033 (calculated)	2,220 (calculated)
Diffusion coefficient (cm^2/s)	0.046	0.046

Source: U.S. EPA, 1986; Kenaga, 1980

accounts for the proposed half lives of 900 days for beta endosulfan and 60 days for the alpha isomer (WHO, 1984).

Thermolysis reactions have been proposed to be the primary soil degradation pathways for surface concentrations of the endosulfan compounds (WHO, 1984). Other potential degradations reactions such as oxidation, photolysis, and hydrolysis appear to play only a minor role. Endosulfan sulfate, the primary biodegradation product, itself degrades by primarily via photolysis and in water will tend to degrade to produce sulfur dioxide and endosulfan

alcohol (U.S. EPA, 1979). This reaction may be very important in soils as when endosulfan sulfate contacts with leaching rain water.

Bioaccumulation

Because endosulfan is easily transformed into other similarly structured compounds under most environmental conditions, this compound is not considered to have the extreme bioaccumulation potential as many of the other cyclodiene pesticides (WHO, 1984). While this condition is true primarily for soils, endosulfan in water may be removed readily from the aqueous phase by a variety of bioaccumulation processes (U.S. EPA, 1979). Bioaccumulation ratios for endosulfan are higher for aquatic organisms compared to terrestrial animals (Menzer and Nelson, 1986).

Toxicokinetics

The isomers of endosulfan are equally absorbed in mammals following ingestion, inhalation or skin contact. Endosulfan will accumulate to a plateau level in living systems during exposure, and is then metabolized to endosulfan sulfate, endosulfan diol and a number of less abundant compounds in the mammalian system (WHO, 1984). These metabolic products are rapidly excreted via the feces and the urine following the removal of the endosulfan source. Endosulfan has only a low affinity for lipids compared to other cyclodiene pesticides, and therefore, does not tend to remain in fat tissue (WHO, 1984).

Qualitative Description of Health Effects

Carcinogenic Potential

Endosulfan is not considered to be carcinogenic in mammals according to the World Health Organization (WHO, 1984). The most recent reports from the EPA indicate that the agency has not come out with an official position regarding endosulfan potential carcinogenicity at the present time, but has stated that the chemical has not been evaluated as yet regarding its potential for human carcinogenicity (U.S. EPA, 1988).

Genotoxic Potential

Very little information has been identified in the literature regarding the potential genotoxicity of endosulfan and endosulfan sulfate. Studies of mutagenicity induced by endosulfan exposure are inconclusive since some tests found increased incidence of mutation, while others saw no effect at all (WHO, 1984). It is presumed since the compound has not been shown to produce any carcinogenic effects in laboratory animals, endosulfan would be weakly mutagenic at best.

Reproductive Effects

Although no dramatic reproductive effects have been noted in the literature for the endosulfan compounds, smaller litter sizes have been noted in the second generation of rats exposed to levels of endosulfan as low as 3 mg/kg body weight (U.S. EPA, 1988).

Acute/chronic effects

Endosulfan has been judged to be non-carcinogenic based on the results from a German bioassay published in 1984 (U.S. EPA, 1988). It has been suggested that a very high mortality rate for mice in a carcinogenicity study prevented the collection of tumor data from these animals (WHO, 1984). Additional studies provide further evidence suggesting a higher mortality rate for exposed animals. In addition, other abnormalities such as weight increase in several organs and hematological effects were found in these animals.

The German rat bioassay was the first animal study that showed conclusive evidence of toxicity on a dose-response basis (U.S. EPA, 1987). This study fed endosulfan to rats at varying doses of 3, 15, and 75 ppm in their diet. Evidence of kidney toxicity was noted in all three exposure groups as exemplified by a yellowish discoloration in the cells of the proximal convoluted tubules in the kidney.

Subchronic feeding of endosulfan does not appear to induce any specific long term health effects in animals. Slight changes have been noted in the activity of an array of enzymes and in other metabolic processes. One study on mice found the activity of oxidase enzymes to be increased; another saw the weight of the liver increased. A subchronic feeding study on dogs caused temporary vomiting, tremors and convulsions to occur at doses of 2.5 mg/kg (WHO, 1984). Lower doses did not produce these effects.

Endosulfan administered in acute doses is moderately toxic in mammals. Symptoms of acute toxicity include hyperactivity, tremors, convulsions and ultimately death. LD_{50} values, that are equivalent for endosulfan Alpha & Beta and endosulfan sulfate, are approximately 40 mg/kg body weight for laboratory animals

(WHO, 1984).

Quantitative Description of Health Effects

At the present time, the U.S. EPA regulates endosulfan as a non-carcinogen. A reference dose (RfD) value of 5.0E-05 mg/kg-day has been published in IRIS based on potential kidney toxicity in mammals (U.S. EPA, 1988). This value was derived based on the presence of a no-observable-effects-level (NOEL) that could not be estab-lished from this particular bioassay and an uncertainty factor of 3000 to account for inter- and intraspecies differences, the lack of a NOEL and the lack of a complete database on chronic exposures.

The State of California has not established TTLC/STLC levels for endosulfan. While the methodology used to establish these levels approximately a decade ago was not a rigid exercise that can be applied from one substance to another, the present day practice in health risk assessment is to assume an acceptable health risk level and back-calculate out the soil concentration that would correspond to that acceptable level of risk.

Since endosulfan is a noncarcinogenic compound, risk assessment methodology can be used to estimate the soil concentration of endosulfan that when ingested would result in a daily intake level of 5.0E-05 mg/kg-day (the current RfD published by the EPA). Assuming an average daily soil consumption rate of 100 mg/day, the following endosulfan soil concentration corresponding to a "safe" dose level is estimated:

5.0E-05 mg/kg-day X 70 kg X 1,000,000 mg/1 kg X 1 day/100 mg = 35 mg/kg

If we apply a safety factor of 10 to represent the potential exposures that may occur via skin absorption and other potential

exposure routes, a "safe soil" concentration of endosulfan is estimated to be 3.5 mg/kg. This soil concentration value is equivalent, in theory, to the TTLC levels stipulated in Title 22. Likewise, an STLC value for endosulfan is estimated as 0.35 mg/l.

ERS

REFERENCES for ENDOSULFAN I & II & ENDOSULFAN SULFATE

Kenaga, E.E., 1980: Predicted Bioconcentration Factors and Soil Sorption Coefficients of Pesticides and Other Chemicals, Ecotoxicology and Environmental Safety, 4:26-38.

Menzer, R. and J. Nelson, 1986: Water and Soil Pollutants, Casarett and Doull's Toxicology - The Basic Science of Poisons, 3rd ed., Chpt. 26, p. 830-832.

U.S. EPA, 1988: Integrated Risk Information System (IRIS), U.S. Environmental Protection Agency, Washington, D.C.

U.S. EPA, 1987: Health Effects Assessment for Alpha- and Beta-Endosulfan. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office.

U.S. EPA, 1986: Superfund Public Health Evaluation Manual, U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C., EPA/540/1-86/060.

U.S. EPA, 1979. Water-Related Environmental Fate of 129 Priority Pollutants, Volume 1. Prepared by Versar, Inc. for the U.S. Environmental Protection Agency, Office of Water Planning and Standards, U.S. EPA-440/4-79-029a.

World Health Organization (WHO), 1984. Endosulfan, Environmental Health Criteria 40, Geneva.

Introduction

Endrin is a pesticide belonging to the organochlorine cyclodiene chemical family. In the 1950s and 1960s, endrin was a commonly used insecticide and rodent control agent. It was most commonly used as a pesticide on cotton crops. Due to its persistence in soil and potential toxicity, endrin use has become restricted in recent years (U.S. EPA, 1980). Most of the major uses of endrin in the United States were banned by the Environmental Protection Agency in 1979 (Federal Register, 1979). Endrin is a persistent compound in soil environments, migrates slowly and is known to produce only noncarcinogenic health effects in exposed mammals.

Physical/chemical and environmental fate properties

Endrin is a cyclodiene pesticide consisting of a cyclic hydrocarbon with a chlorine-substituted methanobridge structure. Formulations containing pure endrin are typically 96.6% pure that include dieldrin, isodrin, and aldrin as impurities. Pure endrin is a white crystal compound. The physical/chemical properties of endrin are presented below:

TABLE A-4

Physical/Chemical Properties of Endrin

Molecular Weight (g/mole)	381
Henry's Law Constant ($atm\text{-}m^3/mole$)	3.86E-07
Vapor Pressure (mm Hg)	2.0E-07
Water Solubility @ 25°C (mg/l)	0.26
Log octanol/water partition coefficient	5.3 - 5.6
K_{OC}	34,000
Diffusion Coefficient (cm^2/s)	0.044

Sources: U.S. EPA, 1987, Kenaga, 1980.

Environmental Fate and Persistence

Endrin is known to be highly persistent in soil owing to its relatively high soil/organic carbon partition coefficient (K_{OC}) value (34,000). The pesticide binds quickly and is generally resistent to most migration mechanisms, including leaching. Because it biodegrades so slowly, endrin has a tendency to bioaccumulate (U.S. EPA, 1987). Endrin in soil and aqueous environments will oxidize when exposed to ambient air and forms a variety of products including endrin aldehyde under many conditions.

Although no information is available in the literature regarding photolysis of endrin aldehyde, endrin has been shown to undergo photolysis in its solid state and in organic solutions. However, no quantitative data are available to evaluate endrin photolysis

in both soil and aqueous conditions (U.S. EPA, 1980).

Endrin has a hydrolysis half-life of approximately four years (U.S. EPA, 1979). Studies have not evaluated the hydrolysis of endrin aldehyde, but in comparison with endrin, the half-life of endrin aldehyde is also assumed to be a minimum of four years (U.S. EPA, 1980). No information is available in the literature regarding the volatilization rates of endrin or endrin aldehyde adsorbed to soil. It is assumed that the potential for volatilzation is low due to endrin's low Henry's Law constant and vapor pressure.

Bioaccumulation

Aquatic system studies indicate that endrin is taken up rapidly and completely by aquatic microorganisms, plants and fish. Endrin bioaccumulation in water results in bioconcentration factors ranging from 1,000 to 10,000 in microcosm experiments (U.S. EPA, 1980). In both mammals and birds, endrin accumulates in fatty tissues although bioaccumulation ratios have not been derived for terrestrial species. In mammals, endrin is distriuted and concentrated throughout many vital organs including the brain, liver and kidneys (U.S. EPA, 1987).

Toxicokinetics

Absorption rates for endrin via ingestion, inhalation and dermal absorption have not been identified in the literature. It is known that endrin is absorbed by humans and other mammals because tissue residue levels have been detected following exposure (U.S. EPA, 1985). Since definitive absorption rates are not published, it is assumed that absorption via ingestion and inhalation would be 100%. Absorption via dermal absorption of soil-bound endrin would be considerably lower at approximately 1%.

Endrin metabolism is complex and dependent upon the specific species involved. Although, they have not been studied extenively, neurotoxins like photodieldrin are known to be among endrin's metabolic byproducts (Brooks, 1973). Other cyclodiene compounds are commonly formed during metabolism. The metabolic pathway common in mammals involves degradation of the methylene bridge followed by oxidation to form 12-ketoendrin. This strucure is considered to be the major toxic component of endrin and possibly endrin aldehyde (U.S. EPA, 1987). Endrin excretion occurs rapidly in mammals in the form of a hydrophilic metabolite (U.S. EPA, 1979). The efficiency of endrin excretion in humans is indicated by the relatively short half-life in blood serum, estimated to be 1 to 2 days. The most common excretion route is via the urine (U.S. EPA, 1987).

Humans do not generally store large amounts of endrin following exposure. Following accidental endrin poisoning, the pesticide has been detected in urine and blood samples. Blood endrin levels have been shown to decline rapidly in these victims, indicating efficient excretion of the toxin by humans (U.S EPA, 1979).

Qualitative Description of Health Effects

Carcinogenic Potential

Studies of endrin carcinogenicity in laboratory rats and mice did not find any oral carcinogenic potential or any increase in tumors following endrin consumption. Long-term studies of dogs consuming maximum dose levels of endrin also failed to reveal any carcinogenicity of endrin (U.S EPA, 1987). Based on these results, endrin is not considered to be an animal or a human carcinogen.

Genotoxic Potential

Endrin is a relatively nongenotoxic compound. Laboratory studies of the mutagenicity of endrin resulted in negative results in the Salmonella typhimurium reverse mutation assay and in Escherichia coli WP2 hcr. Additionally, endrin did not cause an increase in DNA synthesis in the published rat and hamster studies (U.S. EPA, 1987).

Reproductive Effects

Endrin is considered to be a moderate reproductive toxin based on evidence that repeated exposures have led to maternal mortality in a study involving hamsters. Reduced fetal weight and diminished skeletal formation were both produced at doses one-half the maximum (Chernoff et al., 1979). Single exposures to maximal doses in a second study were associated with an increase in fused ribs and cleft palate formation in addition to a significant increase in fetal deaths (Ottolenghi et al., 1974). In a study with rats, increased infant motor activity was associated with maternal endrin consumption during lactation, and the same animals showed diminished activity levels at maturity (U.S. EPA, 1987). Mice have been shown to produce smaller litters following endrin consumption during lactation, although rat studies have not seen the same effect. Low dose endrin exposure in quails stopped egg production, and pheasants consuming 10 times the dose were shown to suffer reduced egg production and increased chick mortality (U.S. EPA, 1973).

Acute/chronic Effects

Acute endrin toxicity onset is rapid and severe although for those animals that survive, recovery also occurs quickly. Initial symptoms of endrin toxicity include central nervous system stimulation including tremors and seizure activity.

Bradycardia, hypertension, increased body temperature and increased cerebrospinal fluid pressure are among the other symptoms of acute endrin toxicity (U.S. EPA, 1980). The acute LD_{50} of endrin for mammals ranges from 2.3 mg/kg to 43.4 mg/kg. Repeated exposure causes cardiac arrhythmias in monkeys and dysrhythmias and convulsions in rats (U.S. EPA, 1987). No information regarding the potential adverse health effects in humans following acute exposure has been identified in the literature.

Long term exposure to endrin results in the production of noncarcinogenic health effects specifically. Monkeys exposed to endrin produced convulsions and characteristic EEG changes. An additional study in rats demonstrated severe seizure activity leading to tetany and death resulting from long-term consumption of endrin (U.S. EPA, 1980). Histological changes in renal epithelium of laboratory rats have also been identified as a chronic effect of endrin consumption (U.S. EPA, 1987).

Quantitative Description of Health Effects

Exposure to endrin produces noncarcinogenic adverse health effects primarily as adverse impacts upon neurologic function. The only health criterion available for endrin is an oral reference dose (RfD) value of 3×10^{-4} mg/kg-day published in the U.S. EPA's Superfund Public Health Risk Evaluation Database (PHRED) (U.S. EPA, 1988). This noncarcinogenic health criterion is used for in the inhalation, ingestion and dermal absorption exposure routes in this health risk assessment.

The California Department of Health Services has established a TTLC level of 0.2 mg/kg and a STLC value of 0.02 (mg/l) for endrin.

REFERENCES for ENDRIN

Brooks, G.T., 1973: Chlorinated Insecticides, CRC Press, Cleveland, OH.

Chernoff, N., R. Kavlock and R. Hanisch, 1979: Perinatal toxicity of Endrin in Rodents. I. Fetotoxic Effects of Prenatal Exposure in Hamsters, Toxicology 13:153-157.

Federal Register, 1979: Endrin Intent to Cancel Registrations and Denial of Applications for Registration of Pesticide Products Containing Endrin, and Statement of Reasons, 44:43632-43657.

Kenaga, E.E., 1980: Predicted Bioconcentration Factors and Soil Sorption Coefficients of Pesticides and Other Chemicals, Ecotoxicology and Environmental Safety, 4:26-38.

Ottolenghi, A., J. Haseman and F. Suggs, 1974: Teratogenic Effects of Aldrin, Dieldrin, and Endrin in Hamsters and Mice, Teratology 9:11-16.

U.S. EPA, 1988: Superfund Public Health Risk Evaluation Database (PHRED), U.S. Environmental Protection Agency, Superfund Office (September 1988 release).

U.S. EPA, 1987: Endrin Health Advisory, U.S. Environmental Protection Agency, Office of Drinking Water, March 31, 1987 edition, Washington, D.C.

U.S. EPA, 1985: Drinking Water Criteria Document for Endrin, U.S. Environmental Protection Agency, Office of Drinking Water, Criteria and Standards Division, Washington, D.C.

U.S. EPA, 1980: Ambient Water Quality Criteria for Endrin, U.S. Environmental Protection Agency, Office of Water Regulations and Standards, EPA/440/5-80-047.

U.S. EPA, 1979: Water-Related Environmental Fate of 129 Priority Pollutants, U.S. Environmental Protection Agency, Office of Water Planning and Standards, Volume I, EPA-440/4-79-029a.

U.S. EPA, 1973: Aspects of Pesticidal Use of Endrin on Man and the Environment, U.S. Environmental Protection Agency, Office of Toxic Substances.