100 Pine Street, 10th Floor
San Francisco, CA 94111
(415) 434-9400 • FAX (415) 434-1365

GEOMATRIX

7 February 1994
Project 1886.03

Mr. Hugh Murphy
City of Hayward Fire Department
Hazardous Materials Office
25151 Clawiter Road
Hayward, California 94545-2731

Subject: Request for Case Closure
25066 Dania Lane
Sunnyside Commons II Parcel
Hayward, California

Dear Mr. Murphy:

As you requested, Geomatrix Consultants, Inc. (Geomatrix), is providing the following clarifications regarding environmental conditions in the vicinity of the former water supply well that was located in the northeast corner of the Sunnyside Commons II parcel. Geomatrix is providing these clarifications on behalf of The Plymouth Group (TPG), of Mountain View, California. We hope that the clarifications provided below help facilitate case closure for the subject site. As TPG has completed construction activities on this parcel, we would appreciate prompt attention to this matter.

BACKGROUND

The Sunnyside Commons II parcel covers approximately 1.8 acres and is adjacent to the former Sunnyside Nursery; the Sunnyside Nursery ceased operations in the fall of 1990. TPG has developed residential housing on this parcel after receiving approval for construction from the Alameda County Health Care Services Agency. The current address where the former water supply well was located is 25066 Dania Lane; the site address was formerly 25054 Mohr Drive.

Terratech, Inc. (Terratech), of San Jose, California performed a supplemental environmental investigation of the Sunnyside Commons II parcel for TPG, in early 1990. The results of this investigation are documented in Terratech's 26 June 1990 report entitled "Supplemental Environmental Investigation, Sunnyside Commons II, Hayward, California." Information regarding the water supply well presented in this report included the following:

- Terratech discovered the open water supply well in the southeastern corner of the Sunnyside Commons II parcel during an initial environmental investigation performed in early 1989; the water supply well reportedly had a

six-inch diameter casing and a measured depth of 24 feet below ground surface.

- Terratech collected a grab groundwater sample from the water supply well and had the sample analyzed for chlorinated pesticides, polychlorinated biphenols (PCBs), and volatile organics. According to the report, the analytical results indicated that the grab groundwater sample contained 14.6 micrograms per liter (μg/l) of endosulfan-family compounds.

- The water supply well was destroyed on 17 May 1990. (Copies of Terratech's well destruction documentation are included in appendix A.)

Terratech installed two groundwater monitoring wells (MW-2 and MW-3) around the former water supply well; these wells supplemented a pre-existing well MW-1 and were positioned so as to allow assessment of the groundwater gradient. A fourth well, MW-4, was subsequently installed in the downgradient direction from MW-3. Groundwater was initially encountered at about 13 to 13.5 feet below grade in each of the wells. Terratech collected groundwater samples from wells MW-3 and MW-4 on 11 June 1990; the groundwater samples were analyzed according to Environmental Protection Agency (EPA) Method 8080. Analytical results indicated that the sample from well MW-3 contained trace amounts (0.70 μg/l) of endosulfan-family compounds; no other EPA Method 8080 compounds were detected. Analytical results indicated that the sample from well MW-4 contained 0.14 μg/l of DDD, a DDT-family compound; no other EPA Method 8080 compounds were detected. The method detection limit for DDT-family compounds is 0.1 μg/l.

On 16 November 1990, Terratech advanced four exploratory borings around the vicinity of the former water supply well to assess the lateral extent of pesticides in groundwater. These activities are presented in Terratech's 4 December 1990 letter report entitled "Definition of the Lateral Extent of Ground Water Impact from Pesticides, Sunnyside Commons II, Hayward, California" included in Appendix A. Terratech collected a grab groundwater sample from each boring and had the groundwater samples analyzed according to EPA Method 8080. No EPA Method 8080 compounds were detected in any of the grab groundwater samples.

Terratech completed four consecutive quarterly groundwater monitoring events at the Sunnyside Commons II parcel in January 1991. A summary of the results from this quarterly monitoring program are presented in Terratech's 30 March 1993 letter report

What about MW-1 & MW-3

entitled "Request for Case Closure Review, Plymouth Group Sunnyside Development, 25054 Mohr Drive, Hayward, California" included in Appendix A. Additional information presented in this report indicates the following:

- The groundwater level elevations remained relatively constant during the duration of the monitoring activities, fluctuating by approximately one foot.

- Declining concentrations of endosulfan-family compounds were detected in well MW-3 for the first three quarterly events; no endosulfan-family compounds were detected in well MW-3 during the fourth quarterly sampling event. No endosulfan-family compounds were detected in well MW-4 during any of the four sampling events.

DEGRADATION OF ENDOSULFAN

Environmental Risk Sciences, Inc. (ERS), of San Francisco, California, prepared a health risk assessment report for TPG regarding the Sunnyside Nursery Site. This 22 June 1989 report, entitled "Health Risk Assessment, Sunnyside Commons Project, Hayward, California" discusses physical/chemical and environmental fate properties of pesticides detected in surface soil at the Sunnyside Nursery site including endosulfan-family compounds. According to the ERS report, endosulfan isomers biodegrade naturally in soil and water. Specifically, the report states that "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." A copy of this report is included in Appendix B.

In March 1993, Geomatrix conducted research regarding the environmental fate of endosulfan in surface soil. The results of this research indicated that endosulfan-family compounds break down in natural settings either biotically or abiotically, and have half lives in soil of several days to a few months. A copy of the memorandum summarizing our research is included in Appendix B.

SUMMARY AND CONCLUSIONS

DDD was detected just above laboratory detection limits during the first quarterly sampling event for well MW-4. DDT-family compounds were not detected in any of the subsequent

three quarterly monitoring events for well MW-4 and were not detected in any of the quarterly monitoring events for well MW-3. Based on these results, it is our opinion that the DDD detected in the groundwater sample collected during the first quarterly monitoring event was an anomaly and should not be considered as an indication that DDT-family compounds are a threat to groundwater in the vicinity of the former water supply well.

Relatively low concentrations of endosulfan-family compounds were detected in a grab groundwater sample collected from the former water supply well. Trace concentrations of endosulfan-family compounds detected in well MW-3 declined for the first three groundwater monitoring events; no endosulfan-family compounds were detected in the fourth quarterly monitoring event. Endosulfan-family compounds were not detected in well MW-4 in any of the four quarterly monitoring events. Endosulfan degrades naturally and has a relatively short half life. The groundwater elevation in the vicinity of the former groundwater supply well appears to be relatively constant. Based on this information, it is our opinion that endosulfan-family compounds are not a continuing threat to groundwater in the vicinity of the Sunnyside Commons II area.

Based on the above, we are requesting regulatory approval for site closure. If you have any questions regarding the above, please contact either of the undersigned.

Sincerely,

GEOMATRIX CONSULTANTS, INC.

Jeffrey C. Nelson, P.E.
Project Engineer

Tom Graf, P.E.
Vice President

JCN/TEG/lam
CONTR\18863RCC.LTR

cc: Dr. Ravi Arulanantham, ACHCSA/RWQCB-SF
Ms. Madhulla Logan, ACHCSA
Mr. Curtis Peterson, TPG
Mr. Eddy So, RWQCB-SF

GARCIA WELL & PUMP COMPANY
P.O. Box 52041
Palo Alto, California 94303
(415) 322-2803

State License No. 384167

Shop Location: 1045 Weeks Street
E. Palo Alto, Ca.
94303

Date: May 23, 1990

Client/Owner: TERRATECH, INC.
1365 Vander Way
San Jose, California 95112

Attention: Eric Lautenbach

Services and Supplies

Job Location: 25054 Mohr Drive, Hayward

Project: Well Development
Your Job Number: 4454/3

Date of Service: May 15, 1990

As arranged with Eric Lautenbach, Terratech, we visited the job location to abandon a shallow well set inside a 4' x 4' pit. The well itself consists of a 6-inch diameter transite casing with a total well depth of 24 feet. There is a standing water level of one foot in the well.

We filled the cased well with 1/4 inch pea gravel up to within 3 feet of the 4 x 4 pit bottom. A cement grout was then pumped into the remaining hole depth and mushroomed over the top of the casing. Native soil was placed in the pit and hand tamped. The 4 x 4 concrete lined pit was demolished and left beside the former well location.

Well Abandonment ---------

As requested, we are enclosing copies of the zone-7 ordinance, permit, and our driller's log.

---

10 May 1990

ZONE 7
WATER RESOURCES ENGINEERING
GROUNDWATER PROTECTION ORDINANCE

SUNNYSIDE NURSERY
25054 MOHR DRIVE
HAYWARD
WELL 3S/2W 29M80
PERMIT 90293

Destruction Requirements

1. Remove from the well any appurtenances and debris.

2. Remove any lining to 2 feet below finished grade or original ground, whichever is the lower elevation.

3. Remove small diameter casing to 3 feet below finished grade or original ground, whichever is the lower elevation.

4. Fill well to 3 feet below finished grade or original ground, whichever is the lower elevation, with pea gravel.

5. Fill the hole from 3 feet to 2 feet with neat cement, cement grout or concrete.

6. After seal has set, backfill the remaining hole with compacted material.

ALAMEDA COUNTY FLOOD CONTROL AND WATER CONSERVATION DISTRICT
5997 PARKSIDE DRIVE • PLEASANTON, CALIFORNIA 94566 • (415) 484-2600

GROUNDWATER PROTECTION ORDINANCE PERMIT APPLICATION

FOR APPLICANT TO COMPLETE

TION OF PROJECT Sunnyside Nursery
25054 Mohr Ln. Hayward, CA

NT
me Terra Tech
ess 1365 Vander Way Phone 408-297-6969
San Jose Zip 95112
Project No. 4454/3

PLICANT
Garcia Well & Pump
ddress 1045 Weeks St. Phone 415-322-2800
E. Palo Alto Zip 94303

PE OF PROJECT
e Construction
thodic Protection ___
ater Supply ___
Monitoring ___
Geotechnical Investigation
General ___
Contamination ___
Well Destruction X

POSED WATER SUPPLY WELL USE
omestic ___ Industrial ___ Other ___
icipal ___ Irrigation ___

RILLING METHOD:
Rotary ___ Air Rotary ___ Auger ___
e ___ Other ___

R LER'S LICENSE NO. 384167

L PROJECTS
Drill Hole Diameter ___ in. Maximum
Casing Diameter ___ in. Depth ___ ft.
Surface Seal Depth ___ ft. Number ___

ECHNICAL PROJECTS
Number of Borings ___ Maximum
Hole Diameter ___ in. Depth ___ ft.

MATED STARTING DATE 15 MAY 90
IMATED COMPLETION DATE ___

reby agree to comply with all requirements of this it and Alameda County Ordinance No. 73-68.

ICANT'S ATURE Fred Wong Date 5-10-90

FOR OFFICE USE

PERMIT NUMBER 90293
LOCATION NUMBER 3S/2W 29M80

PERMIT CONDITIONS

Circled Permit Requirements Apply

(A.) GENERAL
1. A permit application should be submitted so as to arrive at the Zone 7 office five days prior to proposed starting date.
2. Submit to Zone 7 within 60 days after completion of permitted work the original Department of Water Resources Water Well Drillers Report or equivalent for well projects, or drilling logs and location sketch for geotechnical projects.
3. Permit is void if project not begun within 90 days of approval date.

B. WATER WELLS, INCLUDING PIEZOMETERS
1. Minimum surface seal thickness is two inches of cement grout placed by tremie.
2. Minimum seal depth is 50 feet for municipal and industrial wells or 20 feet for domestic and irrigation wells unless a lesser depth is specially approved. Minimum seal depth for monitoring wells is the maximum depth practicable or 20 feet.

C. GEOTECHNICAL. Backfill bore hole with compacted cuttings or heavy bentonite and upper two feet with compacted material. In areas of known or suspected contamination, tremied cement grout shall be used in place of compacted cuttings.

D. CATHODIC. Fill hole above anode zone with concrete placed by tremie.

(E.) WELL DESTRUCTION. See attached.

24' Depth 1' STANDING WATER LEVEL
TRANSITE CASING

Approved Wyman Hong Date 10 May 90
Wyman Hong

121989

CONFIDENTIAL

STATE OF CALIFORNIA DWR
WELL COMPLETION REPORT
(WELL LOGS)

REMOVED

1365 VANDER WAY SAN JOSE, CALIFORNIA 95112 (408) 297-6969 FAX (408) 297-7716

December 4, 1990
Project 4454/3

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

Subject: Definition of the Lateral Extent of
Ground Water Impact from Pesticides
Sunnyside Commons II
Hayward, California

Dear Mr. Murphy:

The following report presents the findings of our recent investigation of the lateral extent of pesticide impact to the shallow ground water beneath Sunnyside Commons II in Hayward (see Figure 1). This study was required to document the absence of a more extensive ground water problem at the site. Our November 7th Work Plan for this investigation decribes the procedures involved and was approved by you in our meeting of November 15, 1990.

SUMMARY OF WORK PERFORMED

On November 16, 1990 four exploratory borings (WS-1 through WS-4) were advanced three to five feet into first ground water (~19 feet below grade) in the locations indicated on Figure 1. Drilling was performed by ASE, a C-57 licensed drilling contractor from San Ramon, with onsite professional guidance provided by an experienced environmental geologist from our staff.

Before travelling to the site, the drill rig and all drilling and sampling equipment was thoroughly steam cleaned at the drillers' yard. The rig was inspected upon arrival at the site and verified to be free of significant fluid leaks.

The combination of a 12+ inch diameter auger and hand shovelling were used to start each hole and remove the upper 2+ feet of surface soil. Eight-inch diameter, pre-cleaned hollow-stem augers were then used to open the holes to their final depth. Soil samples were collected for classification purposes at five-foot interval using an 18-inch split-spoon sampler. The split spoon soil sampler and drill bit will be thoroughly washed onsite in an Alconox™ solution then rinsed by potable water before each use.

Our geologist logged the soil conditions encountered using the Unified Soil Classification System with visual-manual procedures (ASTM 2488-84) and prepared an exploration drill hole log for each hole (see Appendix A).

A ground water grab sample was collected from each hole with a discreet Teflon bailer after purging two "hole volumes". The purge water was placed in labelled drums pending the outcome of test results. Each ground water sample was transferred from the bailer into two one-liter amber jars supplied by the testing laboratory. The sample jars were then immediately labelled and iced. A chain-of-custody record was completed to document sample collection, handling and analytical requests (see Appendix B). Our geologist wore a new pair of disposable gloves for each sampling process to minimize the risk of cross-contamination.

A "blank" sample of distilled water was collected from each bailer prior to sampling. These four blanks were refrigerated for potential testing but ultimately not analyzed as their corresponding ground water samples showed no pesticide impact.

Following the collection of ground water samples, the four holes were sealed with a cement/bentonite slurry.

The four ground water samples were submitted to Anametrix, a State certified laboratory in San Jose, and analyzed for organochlorine pesticides by EPA Method 608/8080.

FINDINGS

As expected, first ground water was encountered about 16 feet below grade and stabilized at a slightly higher level (see appended drill hole logs).

The laboratory report for the four ground water grab samples is presented in Appendix B. The results have been incorporated in the cummulative summary table (Table 1). No EPA Method 8080 pesticides were detected in the ground water at locations WS-1 through WS-4.

CONCLUSIONS AND RECOMMENDATIONS

The findings of the investigation described herein - teamed with the prior documentation from DH-1 (Sunnyside Nursery), DH-8 (Laguna Park) and MW-2 - demonstrate that the ground water impact is localized to the former open well (SW-1). No further ground water investigation is recommended.

The three rounds of ground water testing performed to date at MW-3, the well closest to the former open well, indicates a declining concentration of total Endosulfan - from 0.70 parts per billion (ppb) to 0.11 ppb. The trace amount of DDD at MW-4 has not been re-confirmed.

Based upon the encouraging signs of dissipation/degradation and the 1.75 ppb "safe" drinking water concentration determined by Environmental Risk Sciences (ERS), we recommend continuation of monitoring on a quarterly basis. For each monitoring event the local ground water should be verified and samples from MW-3 and MW-4 should be tested for chlorinated pesticides using EPA Method 608/8080. Monitoring should continue until both wells show no detectable pesticide residues for four consecutive quarters.

If, for some unexpected reason, the Endosulfan (or other EPA 8080 pesticide) residuals rise above drinking water standards, we recommend initiating a "pump and treat" cleanup process utilizing the existing well(s). Extracted ground water can be pumped through activiated carbon to remove the pesticides then either discharged to the storm sewer under a RWQCB permit or utilized on-site for dust control or landscape irrigation. In accordance with RWQCB guidelines, post-cleanup monitoring would have to continue for at least four quarters.

We see no technical reason that the public process steps of tentative and final map review/approval, as well as grading and construction permit issuance, cannot proceed simultaneously to the monitoring (or cleanup) work described above.

LIMITATIONS

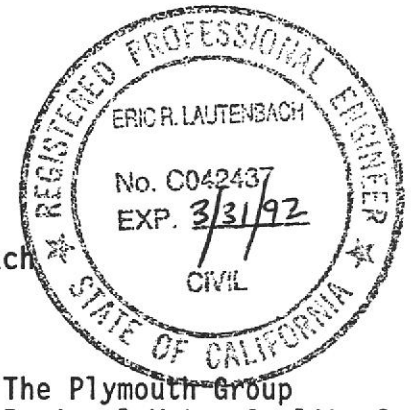
This report and the work associated with it have been provided in accordance with the general principles and practices currently employed in the environmental consulting profession. This is in lieu of all other warranties, express or implied.

Prepared by,

TERRATECH, INC.

E. R. Ltl

Eric R. Lautenbach
CE 42437



cc: Laura Rice, The Plymouth Group
Rich Hiett, Regional Water Quality Control Board
Pam Evans, Alameda County Health Agency

TABLE 1

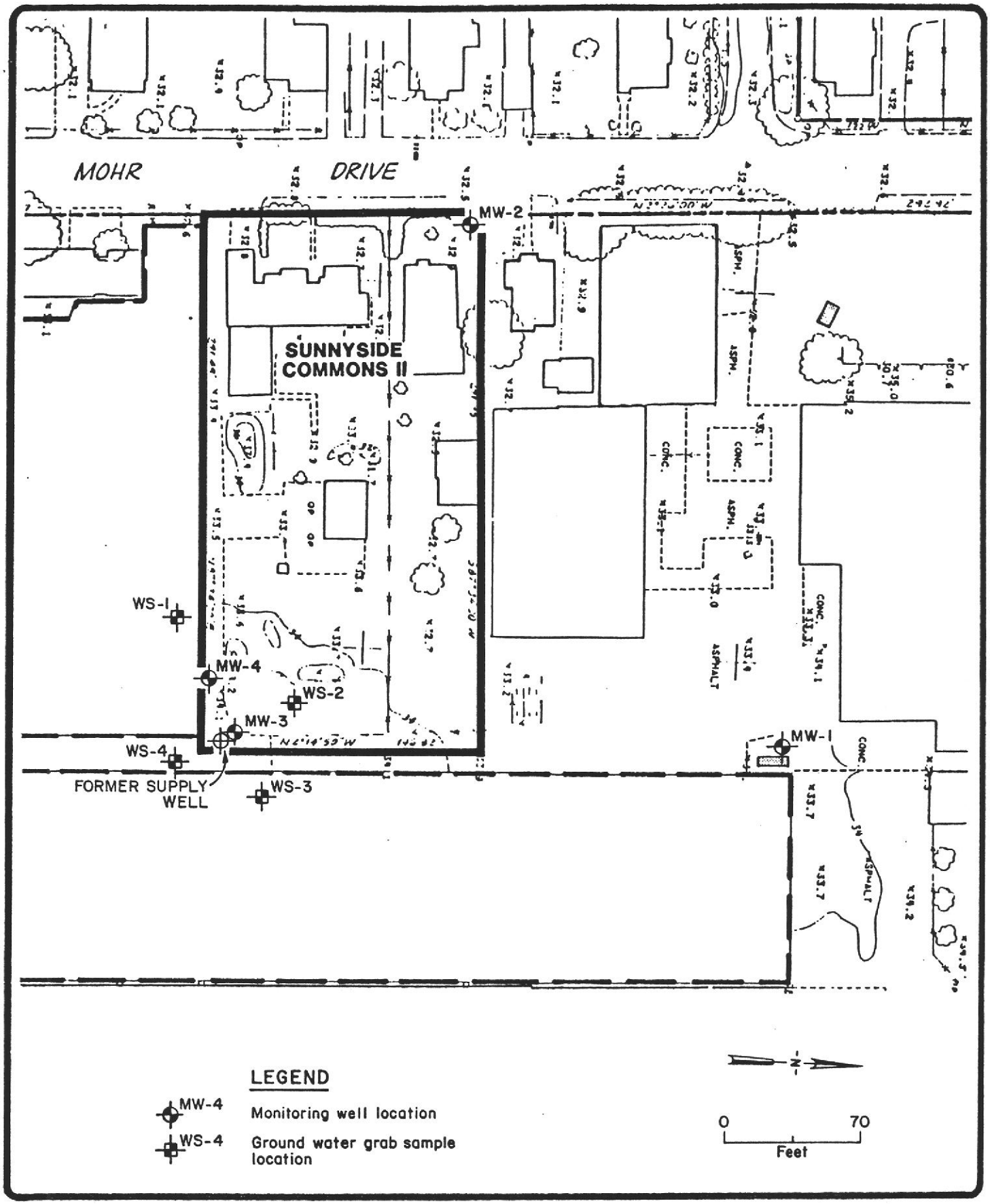
CUMULATIVE SUMMARY OF GROUND WATER SAMPLE ANALYSIS RESULTS

SUNNYSIDE COMMONS II
Hayward, California

SAMPLE LOCATION	DATE COLLECTED (1990)	DDD (ppb)	ENDOSULFAN I (ppb)	ENDOSULFAN II (ppb)	ENDOSULFAN SULFATE (ppb)	OTHERS * (ppb)
SW-1	1/25	< 0.5	5.0	4.5	5.1	N.D.
MW-2	7/17	< 0.1	< 0.05	< 0.1	< 0.1	N.D.
MW-3	5/18	< 0.1	0.37	0.17	0.16	N.D.
	7/17	< 0.1	0.15	0.50	< 0.1	N.D.
	10/17	< 0.1	0.11	< 0.1	< 0.1	N.D.
MW-4	6/11	0.14	< 0.1	< 0.1	< 0.1	N.D.
	7/17	< 0.1	< 0.1	< 0.1	< 0.1	N.D.
	10/17	< 0.1	< 0.1	< 0.1	< 0.1	N.D.
WS-1	11/16	< 0.1	< 0.1	< 0.1	< 0.1	N.D.
WS-2	11/16	< 0.1	< 0.1	< 0.1	< 0.1	N.D.
WS-3	11/16	< 0.1	< 0.1	< 0.1	< 0.1	N.D.
WS-4	11/16	< 0.1	< 0.1	< 0.1	< 0.1	N.D.

NOTES: * - EPA Method 8080 compounds.

N.D. - Not detected; see laboratory reports for detection limits.



TERRATECH

Dec. 1990

SUNNYSIDE COMMONS II
HAYWARD, CALIFORNIA

SITE PLAN AND GROUND WATER SAMPLING LOCATIONS

FIGURE 1

PROJECT 4454/3

SAN JOSE BLUEPRINT CO.
PRINTED ON JR 330

APPENDIX A

DRILL HOLE LOGS

EXPLORATION DRILL HOLE LOG

HOLE No. WS-1

PROJECT SUNNYSIDE COMMONS II | DATE 11-16-90 | LOGGED BY SMC

DRILL RIG Mobile B-61 - Hollow Stem | HOLE DIA. 8" | SAMPLER Std. Pen.

GROUNDWATER DEPTH INITIAL 16' | FINAL 14.5' | HOLE ELEV. ____

DESCRIPTION	SOIL TYPE	DEPTH	SAMPLE	BLOWS PER FOOT	POCKET PEN. (tsf)	TORVANE (tsf)		LIQUID LIMIT	WATER CONTENT	PLASTIC LIMIT	DRY DENSITY (pcf)	FAILURE STRAIN (%)	UNCONFINED SHEAR STRENGTH (psf)
CLAYEY SAND; brown, wet; Fill	CI												
FAT CLAY; black, moist, firm	CH	1											
		2											
CLAYEY SAND; brown, damp, very stiff; fine sand; minor black organics and rootlets	CI	3											
		4											
		5	/										
			/										
		6	/	26									
		7											
SILTY SAND; brown, damp, medium dense; fine sand; ~25%, silty fines	SM	8											
		9											
LEAN CLAY WITH SAND; brown, damp, stiff; ~25% fine sand, rootlets	CL	10	/										
			/										
		11	/	11									
		12											
		13											
		14											
		15	/				▼						
			/										
more plastic CI brown with gray mottles; rootlets, wet		16	/	7			▽						
		17											
		18											
		19											
BOTTOM OF BORING @ 19.5'		20											

Ground Water Sampled

EXPLORATION DRILL HOLE LOG

HOLE No. WS-2

PROJECT: SUNNYSIDE COMMONS II | DATE: 11-16-90 | LOGGED BY: SMC

DRILL RIG: Mobile B-61 - Hollow Stem | HOLE DIA.: 8" | SAMPLER: Std. Pen.

GROUNDWATER DEPTH INITIAL: 16' | FINAL: 14.9' | HOLE ELEV.: ____

DESCRIPTION	SOIL TYPE	DEPTH	SAMPLE	BLOWS PER FOOT	POCKET PEN. (tsf)	TORVANE (tsf)		LIQUID LIMIT	WATER CONTENT	PLASTIC LIMIT	DRY DENSITY (pcf)	FAILURE STRAIN (%)	UNCONFINED SHEAR STRENGTH (psf)
SILTY SAND WITH GRAVEL; brown, dry; Fill	SM	1											
		2											
FAT CLAY WITH SAND; black, dry; 20% fine sand	CH	3											
		4											
CLAYEY SAND; brown, damp, medium dense; fine sand	SC	5	/										
			/										
		6	/	30									
		7											
SANDY LEAN CLAY; brown, damp, stiff; 25% fine sand, minor organics	CL	8											
		9											
		10	/										
			/										
SILTY SAND, brown, damp, medium dense; fine sand, ~40% silty fines	SM	11	/	12									
		12											
		13											
CLAYEY SAND; brown with grey mottles, moist, firm; ~10% fine sand; rootlets	CI	14											
		15	/				▼						
			/										
wet		16	/	7			▽						
		17											
		18											
		19											
BOTTOM OF BORING @ 19.5'		20											

Ground Water Sampled

EXPLORATION DRILL HOLE LOG

HOLE No. WS-3

PROJECT SUNNYSIDE COMMONS II | DATE 11-16-90 | LOGGED BY SMC

DRILL RIG Mobile B-61 - Hollow Stem | HOLE DIA. 8" | SAMPLER Std. Pen.

GROUNDWATER DEPTH INITIAL 16' | FINAL 15.25' | HOLE ELEV. ____

DESCRIPTION	SOIL TYPE	DEPTH	SAMPLE	BLOWS PER FOOT	POCKET PEN. (tsf)	TORVANE (tsf)		LIQUID LIMIT	WATER CONTENT	PLASTIC LIMIT	DRY DENSITY (pcf)	FAILURE STRAIN (%)	UNCONFINED SHEAR STRENGTH (psf)
CLAYEY SAND; brown, moist, loose, wet, minor gravel, Fill	SC	1											
FAT CLAY; black, moist, stiff, trace fine sand	CH	2											
		3											
brown, very stiff; fine sand		4											
brown with black mottles													
CLAYEY SAND/SANDY CLAY; brown, damp, medium dense, very stiff; 50% fine sand, 50% clay	SC-CL	5	/										
			/										
		6	/	25									
		7											
		8											
		9											
SANDY LEAN CLAY; brown, damp, stiff; 30% fine sand, 6" lense of silty sand		10	/										
			/										
		11	/	9									
		12											
		13											
grey-brown, brown mottles; roootlets; less sand;		14											
		15	/				▼						
			/										
wet		16	/	8			▽						
		17											
		18											
		19											
BOTTOM OF BORING @ 19.5'		20											

Ground Water Sampled

EXPLORATION DRILL HOLE LOG

HOLE No. WS-4

PROJECT: SUNNYSIDE COMMONS II | DATE: 11-16-90 | LOGGED BY: SMC

DRILL RIG: Mobile B-61 - Hollow Stem | HOLE DIA.: 8" | SAMPLER: Std. Pen.

GROUNDWATER DEPTH INITIAL: 16.5' | FINAL: 15.75' | HOLE ELEV.: ____

DESCRIPTION	SOIL TYPE	DEPTH	SAMPLE	BLOWS PER FOOT	POCKET PEN. (tsf)	TORVANE (tsf)		LIQUID LIMIT	WATER CONTENT	PLASTIC LIMIT	DRY DENSITY (pcf)	FAILURE STRAIN (%)	UNCONFINED SHEAR STRENGTH (psf)
6" concrete slab													
CLAYEY SAND WITH GRAVEL; brown, moist; Fill	SC	1											
FAT CLAY; black, damp; very stiff; minor fine sand; organics, bluish sandy stringers	CH	2											
dark brown		3											
		4											
CLAY WITH SAND; brown; damp, very stiff, fine sand, black mottles and rootlets; 25% fine sand	CI	5	/										
			/										
		6	/	21									
		7											
sandy		8											
SANDY LEAN CLAY; brown, damp, stiff; 40% fine sand, minor organics and root holes, 3" lense of silty sand	CL	9											
		10	/										
			/										
		11	/	7									
		12											
POORLY GRADED SAND WITH SAND; brown, damp; minor coarse grains, sub angular (comes up on cuttings) predom; medium sand	SP	13											
		14											
		15	/										
LEAN CLAY WITH SAND; brown with rust mottles; moist to wet, firm, fine sand; organics	CL		/										
		16	/	5			▼						
							▽						
		17											
		18											
		19											
BOTTOM OF BORING @ 19.5'		20											

Ground Water Sampled

APPENDIX B

ANALYTICAL LABORATORY REPORT
AND
CHAIN-OF-CUSTODY RECORD

MY 17:00

701176 RC

TERRATECH

CHAIN OF CUSTODY RECORD

P.O. NO. 3343

TURNAROUND: 2-week

PROJECT NUMBER: 4454/3

SAMPLERS (signature): (Shiela Christy)

	Station Number	Date 1990	Time	Comp.	Grab	Station Location	Number of Containers	Analysis Required: EPA 608/8080 (pesticides)	REMARKS	SAMPLE DEPTH
01	WS-1	11-16			X		2 AMBERS	X	Bailer blank @ TT	
02	WS-2	11-16			X		2 AMBERS	X	"	
03	WS-3	11-16			X		2 AMBERS	X	"	
04	WS-4	11-16			X		2 AMBERS	X	"	

Samples iced, OK, no pres.

Relinquished by(signature):	Date / Time	Received by (signature):	Relinquished by(signature):	Date / Time	Received by (signature):
Company or Agency:		Company or Agency:	Company or Agency:		Company or Agency:
Relinquished by(signature):	Date / Time	Received by (signature):	Relinquished by:	Date / Time	Received by (signature):
Company or Agency:		Company or Agency:	Company or Agency:		Company or Agency:
Relinquished by(signature):	Date / Time	Received for Laboratory by:	Date / Time	Remarks/Shipping Information	
Company or Agency: TERRATECH, INC.	11-16-90 4:30pm	(signature)	11-16-90 16:50	Send reports to: Eric Lautenbach 1365 VANDER WAY, SAN JOSE 95112	

ANAMETRIX INC
Environmental & Analytical Chemistry
1961 Concourse Drive Suite E. San Jose, CA 95131
(408) 432-8192 • Fax (408) 432-8198

REPORT

MR. ERIC LAUTENBACH
TERRATECH, INC. - SAN JOSE
1365 VANDER WAY
SAN JOSE, CA 95112

Workorder # : 9011168
Date Received : 11/16/90
Project ID : 4454/3
Purchase Order: 3343

The following samples were received at Anametrix, Inc. for analysis :

ANAMETRIX ID	CLIENT SAMPLE ID
9011168- 1	WS-1
9011168- 2	WS-2
9011168- 3	WS-3
9011168- 4	WS-4

This report is paginated for your convenience and ease of review. It contains 7 pages excluding the cover letter. The report is organized into sections. Each section contains all analytical results and quality assurance data related to a specific group or section within Anametrix. The Report Summary that precedes each section will help you determine which group at Anametrix generated the data. The Report Summary will contain the signatures of the department supervisor and a chemist, both of whom reviewed the analytical data. Please refer all questions to the department supervisor that signed the form.

If you have any further questions or comments on this report, please give us a call as soon as possible. Thank you for using Anametrix.

Burt Sutherland
Laboratory Director

12-04-90
Date

REPORT SUMMARY
ANAMETRIX, INC. (408)432-8192

MR. ERIC LAUTENBACH
TERRATECH, INC. - SAN JOSE
1365 VANDER WAY
SAN JOSE, CA 95112

Workorder # : 9011168
Date Received : 11/16/90
Project ID : 4454/3
Purchase Order: 3343
Department : GC
Sub-Department: PEST

SAMPLE INFORMATION:

ANAMETRIX SAMPLE ID	CLIENT SAMPLE ID	MATRIX	DATE SAMPLED	METHOD
9011168- 1	WS-1	H2O	11/16/90	8080
9011168- 2	WS-2	H2O	11/16/90	8080
9011168- 3	WS-3	H2O	11/16/90	8080
9011168- 4	WS-4	H2O	11/16/90	8080

REPORT SUMMARY
ANAMETRIX, INC. (408)432-8192

MR. ERIC LAUTENBACH
TERRATECH, INC. - SAN JOSE
1365 VANDER WAY
SAN JOSE, CA 95112

Workorder # : 9011168
Date Received : 11/16/90
Project ID : 4454/3
Purchase Order: 3343
Department : GC
Sub-Department: PEST

QA/QC SUMMARY :

- No QA/QC problems encountered for samples.

Stratos Dimas 12-4-90
Department Supervisor Date

Stefanie N Tran 12-4-90
Chemist Date

ORGANIC ANALYSIS DATA SHEET -- EPA METHOD 608/8080
ANAMETRIX, INC. (408) 432-8192

Sample I.D. : 4454/3 WS-1
Matrix : WATER
Date sampled : 11/16/90
Date ext. : 11/20/90
Date analyzed: 11/29/90
Dilution : NONE

Anametrix I.D. : 9011168-01
Analyst : ST
Supervisor : SD
Date released : 12/04/90
Volume ext. : 750mL
Instrument ID : HP5A

CAS #	Compound Name	Reporting Limit (ug/l)	Amount Found (ug/l)
319-84-6	alpha-BHC	0.05	ND
319-85-7	beta-BHC	0.05	ND
58-89-9	gamma-BHC (Lindane)	0.05	ND
319-86-8	delta-BHC	0.05	ND
76-44-8	Heptachlor	0.05	ND
309-00-2	Aldrin	0.05	ND
1024-57-3	Heptachlor epoxide	0.05	ND
959-98-8	Endosulfan I	0.05	ND
72-55-9	p,p'-DDE	0.1	ND
60-57-1	Dieldrin	0.1	ND
72-20-8	Endrin	0.1	ND
72-54-8	p,p'-DDD	0.1	ND
33212-65-9	Endosulfan II	0.1	ND
50-29-3	p,p'-DDT	0.1	ND
7421-93-4	Endrin aldehyde	0.1	ND
1031-07-8	Endosulfan sulfate	0.1	ND
72-43-5	p,p'-Methoxychlor	0.1	ND
53494-70-5	Endrin ketone	0.1	ND
12789-03-6	Technical chlordane	0.5	ND
8001-35-2	Toxaphene	1	ND
1104-28-2	Aroclor 1221	0.5	ND
11141-16-5	Aroclor 1232	0.5	ND
53469-21-9	Aroclor 1242	0.5	ND
12672-29-6	Aroclor 1248	0.5	ND
11097-69-1	Aroclor 1254	1	ND
11096-82-5	Aroclor 1260	1	ND
12674-11-2	Aroclor 1016	0.5	ND
	Dibutylchlorendate	24-154%	72%

ND : Not detected at or above the practical quantitation limit for the method.

ORGANIC ANALYSIS DATA SHEET -- EPA METHOD 608/8080
ANAMETRIX, INC. (408) 432-8192

Sample I.D. : 4454/3 WS-2
Matrix : WATER
Date sampled : 11/16/90
Date ext. : 11/20/90
Date analyzed: 11/29/90
Dilution : NONE

Anametrix I.D. : 9011168-02
Analyst : ST
Supervisor : SD
Date released : 12/04/90
Volume ext. : 1 LITER
Instrument ID : HP5A

CAS #	Compound Name	Reporting Limit (ug/l)	Amount Found (ug/l)
319-84-6	alpha-BHC	0.05	ND
319-85-7	beta-BHC	0.05	ND
58-89-9	gamma-BHC (Lindane)	0.05	ND
319-86-8	delta-BHC	0.05	ND
76-44-8	Heptachlor	0.05	ND
309-00-2	Aldrin	0.05	ND
1024-57-3	Heptachlor epoxide	0.05	ND
959-98-8	Endosulfan I	0.05	ND
72-55-9	p,p'-DDE	0.1	ND
60-57-1	Dieldrin	0.1	ND
72-20-8	Endrin	0.1	ND
72-54-8	p,p'-DDD	0.1	ND
33212-65-9	Endosulfan II	0.1	ND
50-29-3	p,p'-DDT	0.1	ND
7421-93-4	Endrin aldehyde	0.1	ND
1031-07-8	Endosulfan sulfate	0.1	ND
72-43-5	p,p'-Methoxychlor	0.1	ND
53494-70-5	Endrin ketone	0.1	ND
12789-03-6	Technical chlordane	0.5	ND
8001-35-2	Toxaphene	1	ND
1104-28-2	Aroclor 1221	0.5	ND
11141-16-5	Aroclor 1232	0.5	ND
53469-21-9	Aroclor 1242	0.5	ND
12672-29-6	Aroclor 1248	0.5	ND
11097-69-1	Aroclor 1254	1	ND
11096-82-5	Aroclor 1260	1	ND
12674-11-2	Aroclor 1016	0.5	ND
	Dibutylchlorendate	24-154%	80%

ND : Not detected at or above the practical quantitation limit for the method.

ORGANIC ANALYSIS DATA SHEET -- EPA METHOD 608/8080
ANAMETRIX, INC. (408) 432-8192

Sample I.D.	: 4454/3 WS-3	Anametrix I.D.	: 9011168-03
Matrix	: WATER	Analyst	: ST
Date sampled	: 11/16/90	Supervisor	: SD
Date ext.	: 11/20/90	Date released	: 12/04/90
Date analyzed	: 11/28/90	Volume ext.	: 1 LITER
Dilution	: NONE	Instrument ID	: HP5A

CAS #	Compound Name	Reporting Limit (ug/l)	Amount Found (ug/l)
319-84-6	alpha-BHC	0.05	ND
319-85-7	beta-BHC	0.05	ND
58-89-9	gamma-BHC (Lindane)	0.05	ND
319-86-8	delta-BHC	0.05	ND
76-44-8	Heptachlor	0.05	ND
309-00-2	Aldrin	0.05	ND
1024-57-3	Heptachlor epoxide	0.05	ND
959-98-8	Endosulfan I	0.05	ND
72-55-9	p,p'-DDE	0.1	ND
60-57-1	Dieldrin	0.1	ND
72-20-8	Endrin	0.1	ND
72-54-8	p,p'-DDD	0.1	ND
33212-65-9	Endosulfan II	0.1	ND
50-29-3	p,p'-DDT	0.1	ND
7421-93-4	Endrin aldehyde	0.1	ND
1031-07-8	Endosulfan sulfate	0.1	ND
72-43-5	p,p'-Methoxychlor	0.1	ND
53494-70-5	Endrin ketone	0.1	ND
12789-03-6	Technical chlordane	0.5	ND
8001-35-2	Toxaphene	1	ND
1104-28-2	Aroclor 1221	0.5	ND
11141-16-5	Aroclor 1232	0.5	ND
53469-21-9	Aroclor 1242	0.5	ND
12672-29-6	Aroclor 1248	0.5	ND
11097-69-1	Aroclor 1254	1	ND
11096-82-5	Aroclor 1260	1	ND
12674-11-2	Aroclor 1016	0.5	ND
	Dibutylchlorendate	24-154%	88%

ND : Not detected at or above the practical quantitation limit for the method.

ORGANIC ANALYSIS DATA SHEET -- EPA METHOD 608/8080
ANAMETRIX, INC. (408) 432-8192

Sample I.D. : 4454/3 WS-4
Matrix : WATER
Date sampled : 11/16/90
Date ext. : 11/20/90
Date analyzed: 11/28/90
Dilution : NONE

Anametrix I.D. : 9011168-04
Analyst : [illegible]
Supervisor : SP
Date released : 12/04/90
Volume ext. : 950mL
Instrument ID : HP5A

CAS #	Compound Name	Reporting Limit (ug/l)	Amount Found (ug/l)
319-84-6	alpha-BHC	0.05	ND
319-85-7	beta-BHC	0.05	ND
58-89-9	gamma-BHC (Lindane)	0.05	ND
319-86-8	delta-BHC	0.05	ND
76-44-8	Heptachlor	0.05	ND
309-00-2	Aldrin	0.05	ND
1024-57-3	Heptachlor epoxide	0.05	ND
959-98-8	Endosulfan I	0.05	ND
72-55-9	p,p'-DDE	0.1	ND
60-57-1	Dieldrin	0.1	ND
72-20-8	Endrin	0.1	ND
72-54-8	p,p'-DDD	0.1	ND
33212-65-9	Endosulfan II	0.1	ND
50-29-3	p,p'-DDT	0.1	ND
7421-93-4	Endrin aldehyde	0.1	ND
1031-07-8	Endosulfan sulfate	0.1	ND
72-43-5	p,p'-Methoxychlor	0.1	ND
53494-70-5	Endrin ketone	0.1	ND
12789-03-6	Technical chlordane	0.5	ND
8001-35-2	Toxaphene	1	ND
1104-28-2	Aroclor 1221	0.5	ND
11141-16-5	Aroclor 1232	0.5	ND
53469-21-9	Aroclor 1242	0.5	ND
12672-29-6	Aroclor 1248	0.5	ND
11097-69-1	Aroclor 1254	1	ND
11096-82-5	Aroclor 1260	1	ND
12674-11-2	Aroclor 1016	0.5	ND
	Dibutylchlorendate	24-154%	94%

ND : Not detected at or above the practical quantitation limit for the method.

ORGANIC ANALYSIS DATA SHEET -- EPA METHOD 608/8080
ANAMETRIX, INC. (408) 432-8192

Sample I.D.	: METHOD BLANK	Anametrix I.D.	: PWBL112090
Matrix	: WATER	Analyst	: [illegible]
Date sampled	: N/A	Supervisor	: SD
Date ext.	: 11/20/90	Date released	: 12/04/90
Date analyzed	: 11/29/90	Volume ext.	: 1 LITER
Dilution	: NONE	Instrument ID	: HP5A

CAS #	Compound Name	Reporting Limit (ug/l)	Amount Found (ug/l)
319-84-6	alpha-BHC	0.05	ND
319-85-7	beta-BHC	0.05	ND
58-89-9	gamma-BHC (Lindane)	0.05	ND
319-86-8	delta-BHC	0.05	ND
76-44-8	Heptachlor	0.05	ND
309-00-2	Aldrin	0.05	ND
1024-57-3	Heptachlor epoxide	0.05	ND
959-98-8	Endosulfan I	0.05	ND
72-55-9	p,p'-DDE	0.1	ND
60-57-1	Dieldrin	0.1	ND
72-20-8	Endrin	0.1	ND
72-54-8	p,p'-DDD	0.1	ND
33212-65-9	Endosulfan II	0.1	ND
50-29-3	p,p'-DDT	0.1	ND
7421-93-4	Endrin aldehyde	0.1	ND
1031-07-8	Endosulfan sulfate	0.1	ND
72-43-5	p,p'-Methoxychlor	0.1	ND
53494-70-5	Endrin ketone	0.1	ND
12789-03-6	Technical chlordane	0.5	ND
8001-35-2	Toxaphene	1	ND
1104-28-2	Aroclor 1221	0.5	ND
11141-16-5	Aroclor 1232	0.5	ND
53469-21-9	Aroclor 1242	0.5	ND
12672-29-6	Aroclor 1248	0.5	ND
11097-69-1	Aroclor 1254	1	ND
11096-82-5	Aroclor 1260	1	ND
12674-11-2	Aroclor 1016	0.5	ND
	Dibutylchlorendate	24-154%	86%

ND : Not detected at or above the practical quantitation limit for the method.

TERRATECH

1365 VANDER WAY, SAN JOSE, CA 95112, (408) 297-6969, FAX (408) 297-7716
7891 WESTWOOD DR., SUITE 101, GILROY, CA 95020, (408) 842-0236, FAX (408) 842-7314
12 THOMAS OWENS WAY, MONTEREY, CA 93940, (408) 372-3716, FAX (408) 372-7481
141 SUBURBAN RD., SUITE, SAN LUIS OBISPO, CA 93401, (805) 543-5493, FAX (805) 543-2748

HAYWARD FIRE DEPARTMENT

March 30, 1993
Project 4454/3

Eddy So
California Regional Water Quality Control Board
2101 Webster Street, Suite 500
Oakland, California 94612

Subject: Request for Case Closure Review
Plymouth Group Sunnyside Development
25054 Mohr Drive
Hayward, California

Dear Mr. So:

This letter transmits ground water monitoring data for 25054 Mohr Drive that we understand the RWQCB may not have on file, summarizes the past actions and presents rationale for case closure consideration.

As we discussed, The Plymouth Group is urgently seeking regulatory approval to permanently close the on-site monitoring wells as they are moving forward with a development of single family homes on the property. Hugh Murphy of the Hayward Fire Department referred us to you as the lead agency to make the decision since the issue involves a former open well and ground water quality.

WORK SUMMARY

In January 1990, while performing a pre-purchase, environmental assessment for the Plymouth Group, Terratech discovered an open well in the southeastern property corner. The well had a 6-inch diameter casing and was measured to be 24 feet deep. Neither the owner of the subject residential parcel nor the owner of the surrounding nursery (also now being redeveloped) knew the historic use of the shallow well. Analysis of the well water (SW-1) only found trace concentrations of Endosulfan (<20 parts per billion (ppb) total), a pesticide in common usage at the adjacent nursery. These initial test results are presented in Appendix A and summarized in the attached table. At Terratech's recommendation, the open well was formerly closed.

Terratech performed the first of several subsequent investigations regarding this ground water issue in May/June 1990. Two ground water monitoring wells were installed; MW-3 directly adjacent to the former open well and MW-4 approximately 35 feet downgradient (see attached set of gradient figures). Minute amounts of Endosulfan (<1 ppb total) were found in the ground water at MW-3 but none was detected at MW-4 (see attached table and Appendix B).

March 30, 1993 Project 4454/3

On behalf of the Plymouth Group we would appreciate your prompt consideration of the points summarized above. We will call next week to followup. If you have any questions in the interim, please call me at (408) 297-6969. Curt Peterson of The Plymouth Group can be reached at (415) 691-4300.

Sincerely,

TERRATECH, INC.

E. R. Lautenbach

Eric R. Lautenbach, CE 42437
Associate Environmental Engineer

Attachments (Cumulative Summary Table of Ground Water Analysis Results,
Set of Ground Water Gradient Figures and Appendices A-D)

cc (w/o appendices):
Hugh Murphy, Hayward Fire Department
Curt Peterson, The Plymouth Group

CUMULATIVE SUMMARY OF GROUND WATER ANALYSIS RESULTS
SUNNYSIDE COMMONS II, HAYWARD

LOCATION	DATE	DDD	ENDO. I	ENDO. II	ENDO. SULFATE	OTHER
SW-1	1/25/90	< 0.5	5.0	4.5	5.1	N.D.
MW-2	7/17/90	< 0.11	< 0.05	< 0.1	< 0.1	N.D.
MW-3	5/18/90	< 0.1	0.37	0.17	0.16	N.D.
	7/17/90	< 0.1	0.15	0.50	< 0.1	N.D.
	10/17/90	< 0.1	0.11	< 0.1	< 0.1	N.D.
	1/8/91	< 0.11	< 0.06	< 0.11	< 0.11	N.D.
MW-4	6/11/90	0.14	< 0.05	< 0.1	< 0.1	N.D.
	7/17/90	< 0.1	< 0.05	< 0.1	< 0.1	N.D.
	10/17/90	< 0.1	< 0.05	< 0.1	< 0.1	N.D.
	1/8/91	< 0.11	< 0.06	< 0.11	< 0.11	N.D.
WS-1	11/16/90	< 0.1	< 0.05	< 0.1	< 0.1	N.D.
WS-2	11/16/90	< 0.1	< 0.05	< 0.1	< 0.1	N.D.
WS-3	11/16/90	< 0.1	< 0.05	< 0.1	< 0.1	N.D.
WS-4	11/16/90	< 0.1	< 0.05	< 0.1	< 0.1	N.D.
MCL		---	1.75	1.75	1.75	---

NOTES: ENDO. = Endosulfan.
Other = EPA Method 608/8080 pesticides except for SW-1 which was also tested for carbamate & urea pesticides (EPA 632) and volatile organics (EPA 624/8240).
SW = Supply Well sample (prior to closure).
N.D. = Not Detected above laboratory's threshold of reliable quantitation (see appended laboratory reports for specifics).
MW = Monitoring Well sample.
WS = Water Sample from exploratory boring (subsequently grouted).
MCL = Maximium Contaminant Level for drinking water as calculated by project specialist - Environmental Risk Sciences; presented for relative comparison purposes only.

BASE MAP: Thomas Brothers maps, Alameda County, 1986

N

0 1/2 1 mile
Scale

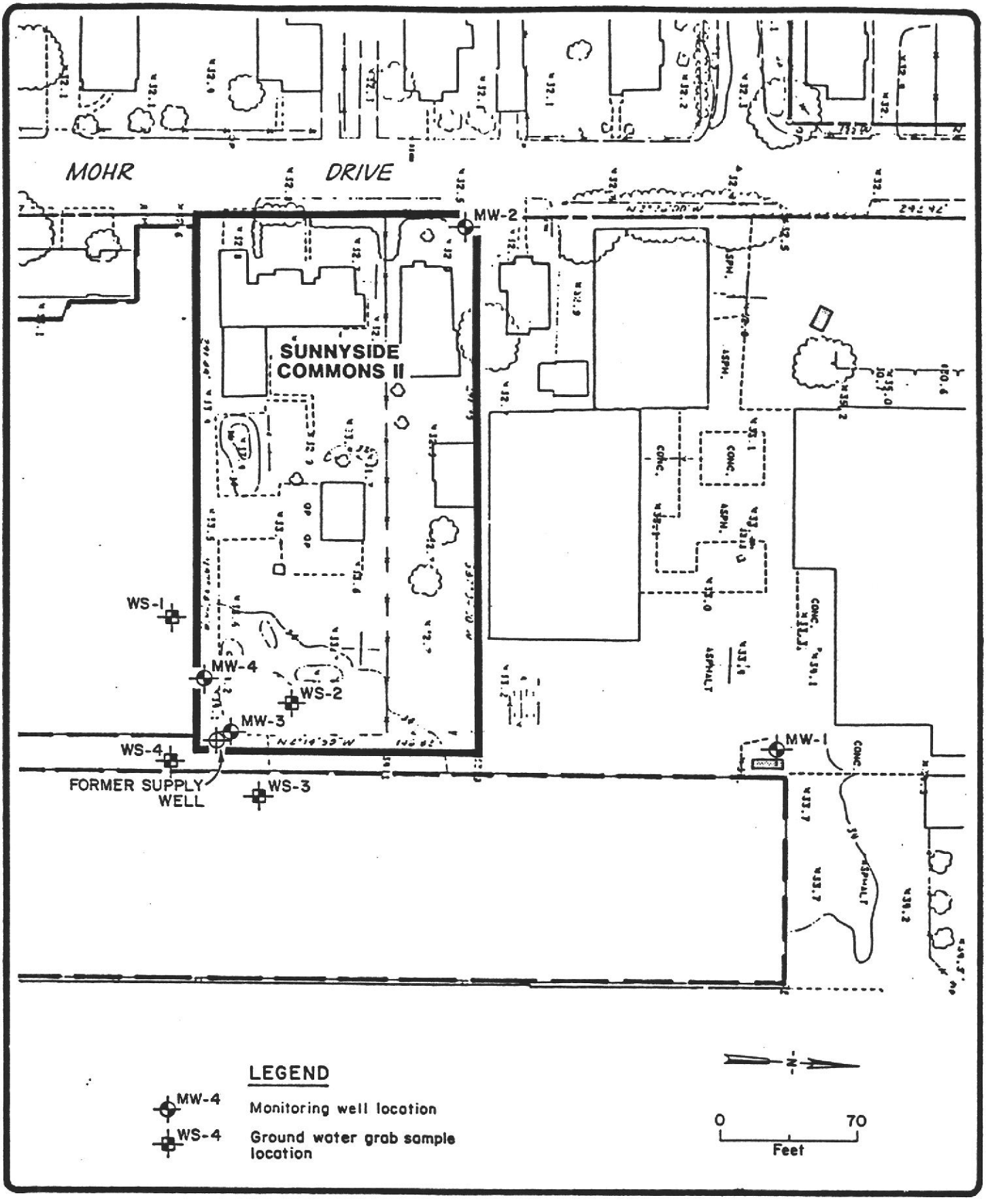
TERRATECH

SUNNYSIDE DEVELOPMENT II
HAYWARD, CALIFORNIA

VICINITY MAP

FIGURE 1

PROJECT 4454/3



TERRATECH

Dec. 1990

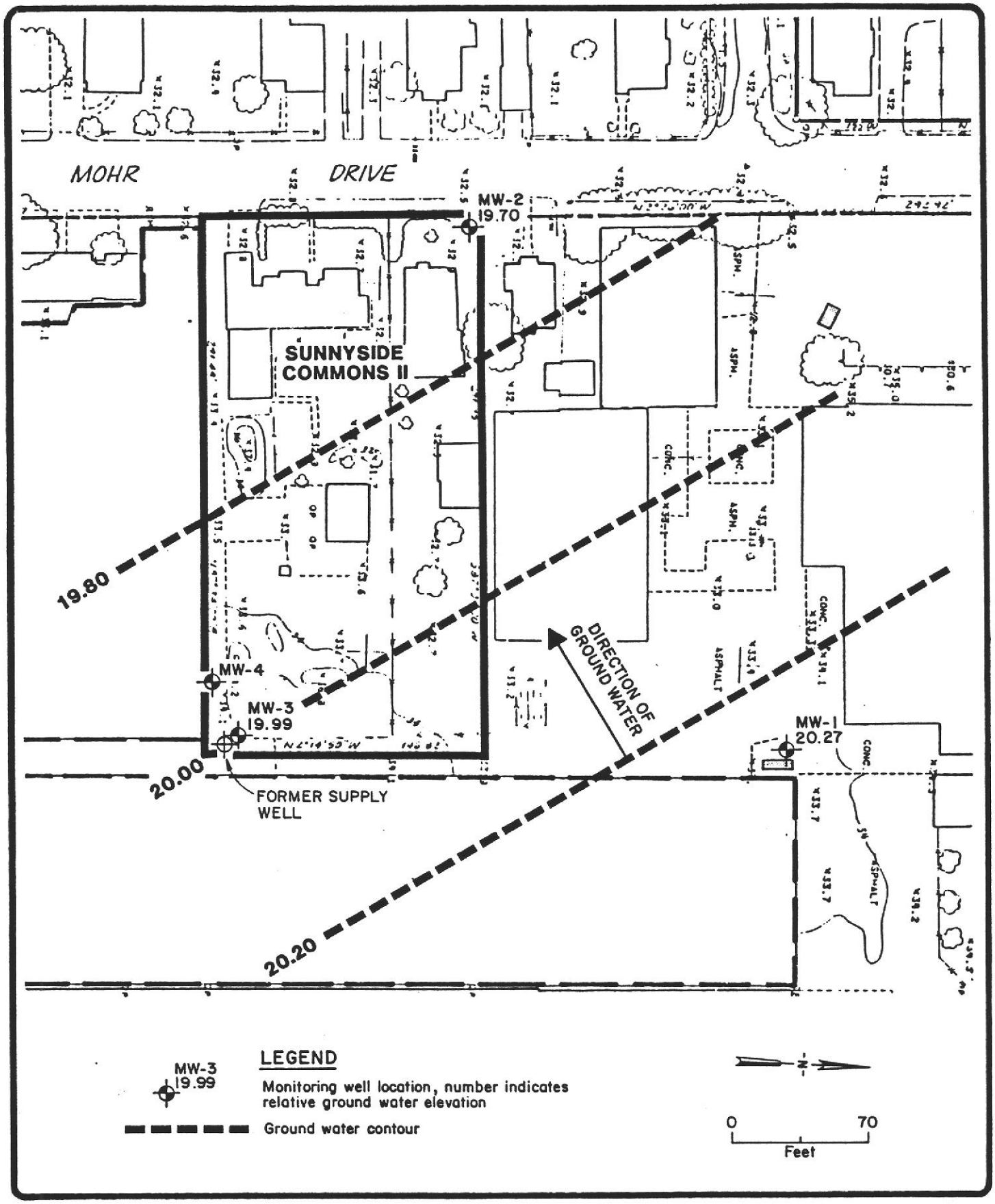
SUNNYSIDE COMMONS II
HAYWARD, CALIFORNIA

SITE PLAN AND
GROUND WATER SAMPLING LOCATIONS

FIGURE
1

PROJECT
4454/3

SAN JOSE BLUEPRINT CO
PRINTED ON JR 330



June 1990

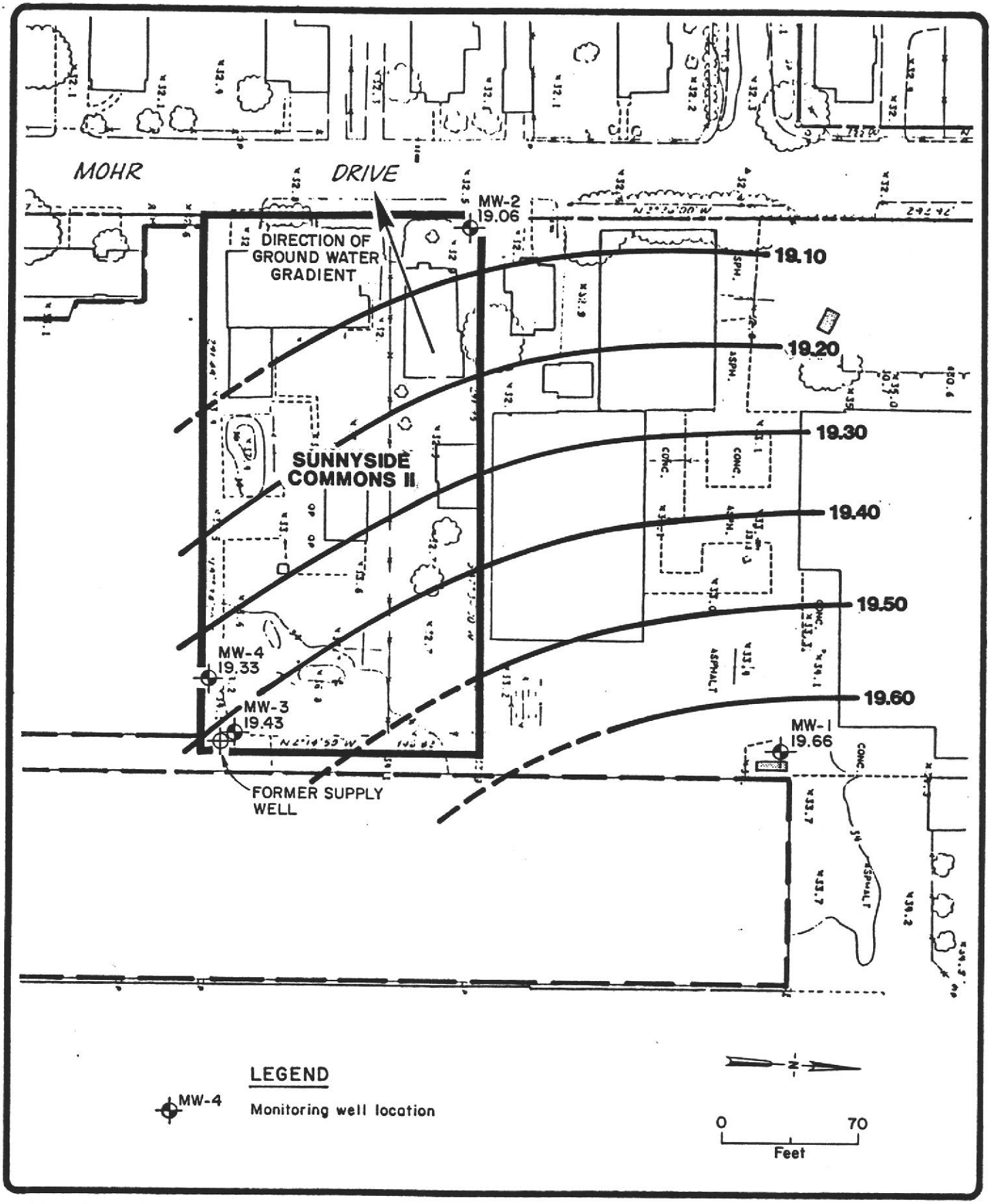
TERRATECH

SUNNYSIDE COMMONS II
HAYWARD, CALIFORNIA

GROUND WATER GRADIENT 5-18-90

FIGURE
1
PROJECT
4454/3

SAN JOSE BLUEPRINT CO
PRINTED ON JR 330



TERRATECH

Sept. 1990

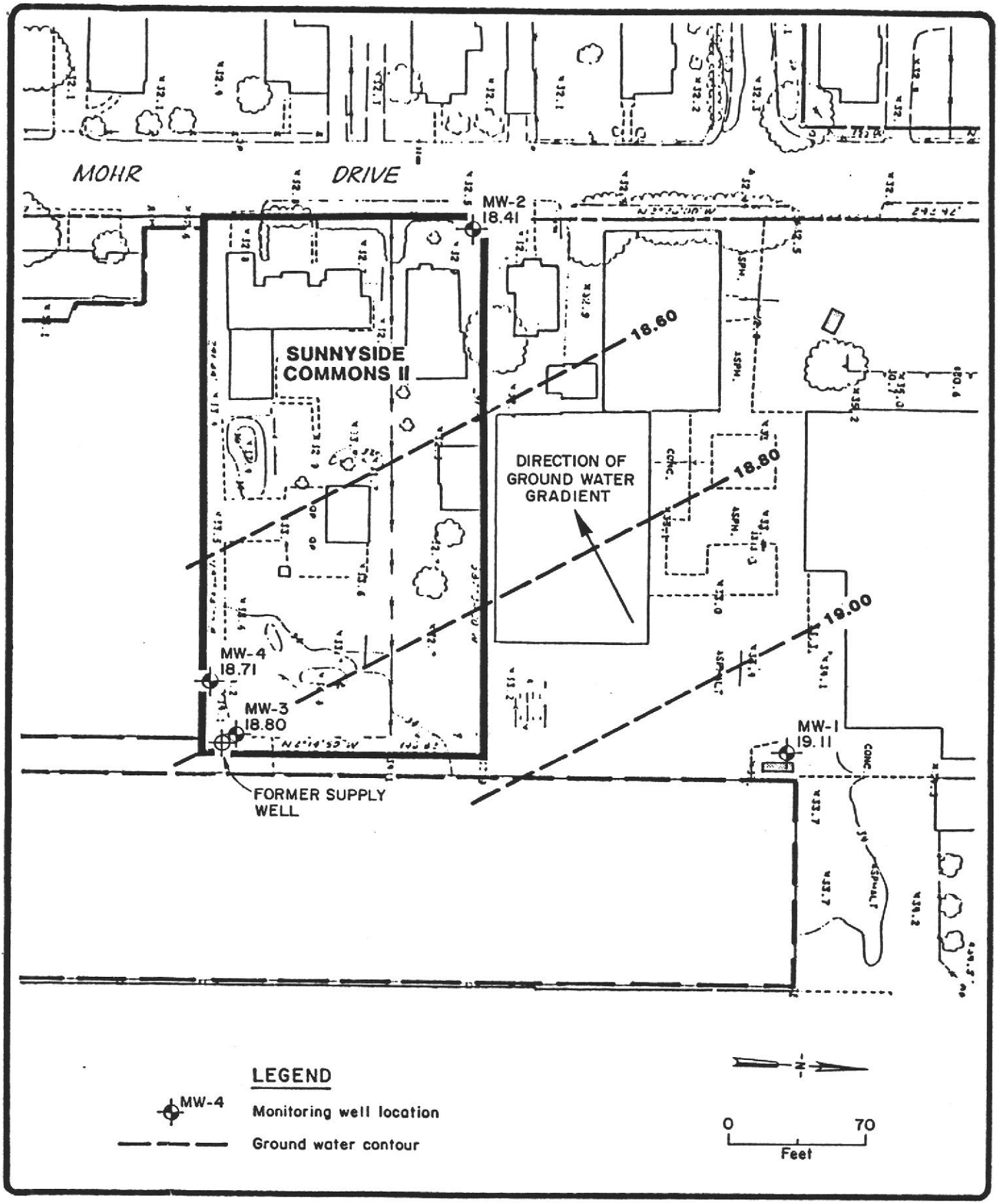
SUNNYSIDE COMMONS II
HAYWARD, CALIFORNIA

SITE PLAN WITH GRADIENT 8/29/90

FIGURE 1

PROJECT 4454/3

SAN JOSE BLUEPRINT CO.
PRINTED ON JR 330



TERRATECH

Dec. 1990

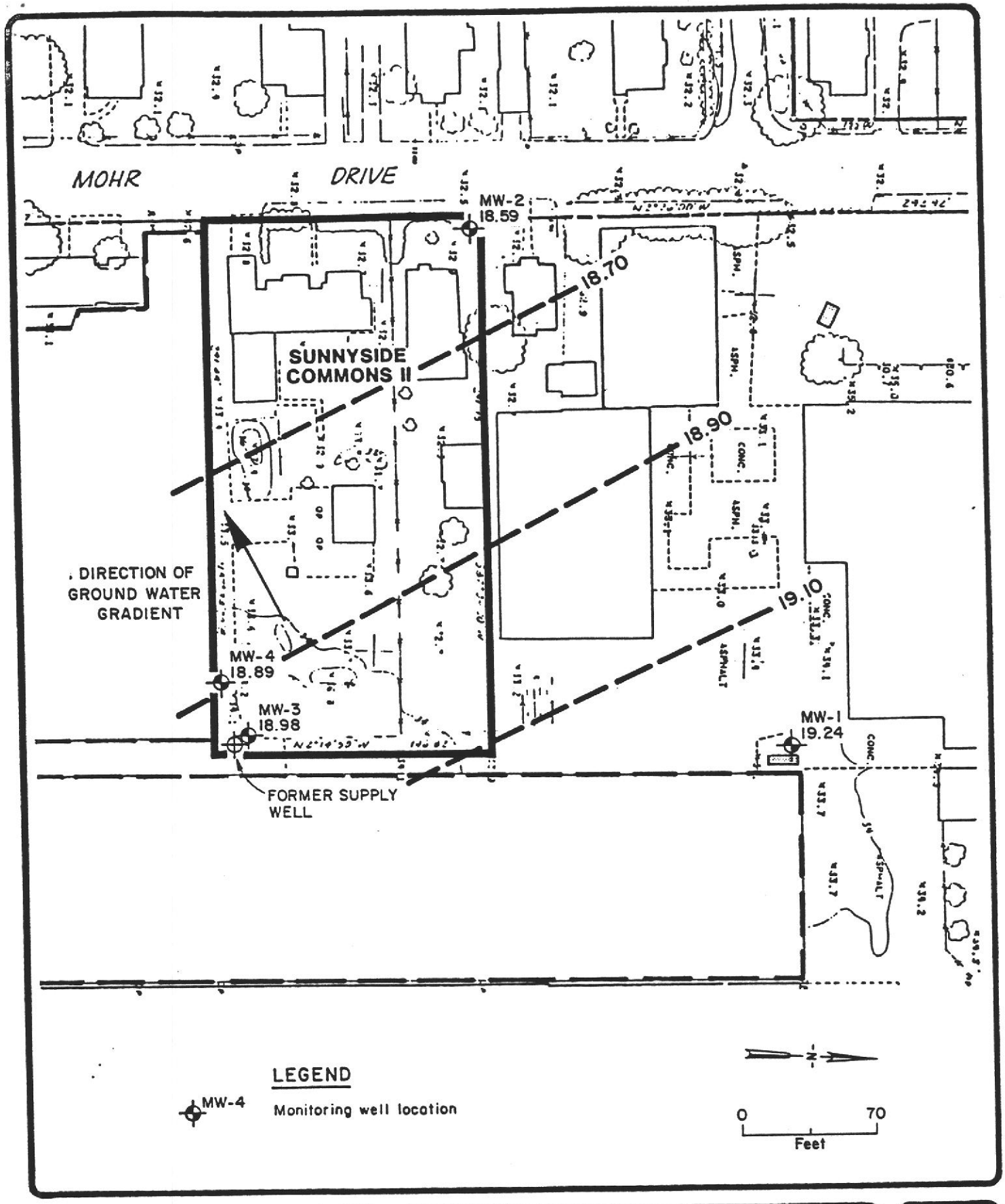
SUNNYSIDE COMMONS II
HAYWARD, CALIFORNIA

GROUND WATER GRADIENT 11/27/90

FIGURE
1

PROJECT
4454/3

SAN JOSE BLUEPRINT CO
PRINTED ON JR 330



Jan. 1991

TERRATECH

SUNNYSIDE COMMONS II
HAYWARD, CALIFORNIA

SITE PLAN WITH GRADIENT 1/8/91

FIGURE
1

PROJECT
4454/3

SAN JOSE BLUEPRINT CO
PRINTED ON JR 330

GEOMATRIX

MEMORANDUM

TO: Jeff Nelson **DATE:** 7 April 1993

FROM: Larry Edwards LE **FILE:** 1886.02

SUBJECT: Environmental Fate (Lifetime) of Endosulfan

Some question has arisen about the probable lifetime of endosulfan in the natural environment. The question arose when trying to reconcile the results of two different samples of the same soil believed to have been contaminated with endosulfan. One set of samples was taken immediately after a nursery had stopped using the site; the other set was taken about three years later. Is it possible that some endosulfan could have been found in the first sampling effort and not in the second? Endosulfan is a compound likely to have been used by the nursery operation.

Calling upon the Hazardous Substances Data Base (an electronic data base much used by toxicologists), the following information was reported on the terrestrial fate of endosulfan[1]:

1) In one study, the half-life of endosulfan applied to soil at 0.35 kg/ha was 5 days (based on concentration in runoff). [Wauchope, R.D. and Leonard, R.A., Journal of Environmental Quality, 9, 665-72 (1980)]

2) In another study , 0.38 ppm of endosulfan was applied to soil, and three years later, the most that could be found in several samples was 0.04 ppm. [Mullins, D.E., et al., Journal of Pesticide Monitoring, 5, 268-275 (1971)]

3) In another study, endosulfan "persisted" for 100 days in dry soils and for 120 days in wet soils. [Rao, D.M.R. and A.S. Murty, Journal of Agricultural Food Chemistry, 28, 1099-1101 (1980)] In this case, "persisted" is taken to mean that it was still present in levels above the method detection limit.

4) In another study, the half-life of alpha endosulfan was 60 days and the half-life of beta endosulfan was 800 days. [Stewart, D.K.R. and K.G. Cairns, Journal of Agricultural Food Chemistry, 22, 984-986 (1974)]

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[1] The same information may be found in "Handbook of Environmental Fate and Exposure Data for Organic Chemicals: Volume III Pesticides", Howard, Philip H., Editor. Lewis Publishers, Chelsea, MI., 1991, pp 327-343.

5) In another study, endosulfan was applied at a rate of 0.5 to 1.5 lb/acre to tobacco; time to zero residue level was estimated to be ten days. [Metabolism of Pesticides, pg. 191 (1974)]

6) In another study, at 20°C, the abiotic hydrolysis half-life was reported to be 35.4 days and 150.6 days for alpha endosulfan at pH 7.0 and 5.5, respectively; for beta endosulfan, the hydrolysis half-life was reported to be 37.5 and 187 days for pH 7.0 and 5.5, respectively. [Greve, P.A. and S.L. Wit, Journal of Water Pollution Control 42, 2338-2348 (1971)]

In an unreferenced hydrolysis study, the lifetime of endosulfan in surface waters has been reported to be four weeks (i.e., none was detectable after four weeks).

The most likely degradation mechanisms (not in any particular order) are biodegradation, hydrolysis (especially under alkaline conditions), oxidation and photolysis. The most common byproduct is endosulfan sulfate; this compound would not be reported as endosulfan if analyzed by common laboratory practices (i.e., SW-846).

In general, the picture emerges that endosulfan breaks down in natural settings either biotically or abiotically; while the degradation is pH dependent, most studies report half-lives either on soil or in water of several days to a few months. In three years, this would translate into greater than 99% degradation. Citation #2 above did find endosulfan remaining in a few samples after three years at levels up to 10% of the original concentration. Thus, it is possible to persist in the environment for several years, although such persistence is not favored. In this case, if the pH was above 6, it is unlikely that endosulfan remained in detectable quantities three years after its initial application. However, it is probable that endosulfan was detectable 60 days after application and could have been found in the earlier testing.

HEALTH RISK ASSESSMENT
SUNNYSIDE COMMONS PROJECT
HAYWARD, CALIFORNIA

Prepared for

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

Prepared by

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June 22, 1989

ERS Environmental Risk Sciences, Inc.

HEALTH RISK ASSESSMENT
SUNNYSIDE COMMONS PROJECT
HAYWARD, CALIFORNIA

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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.

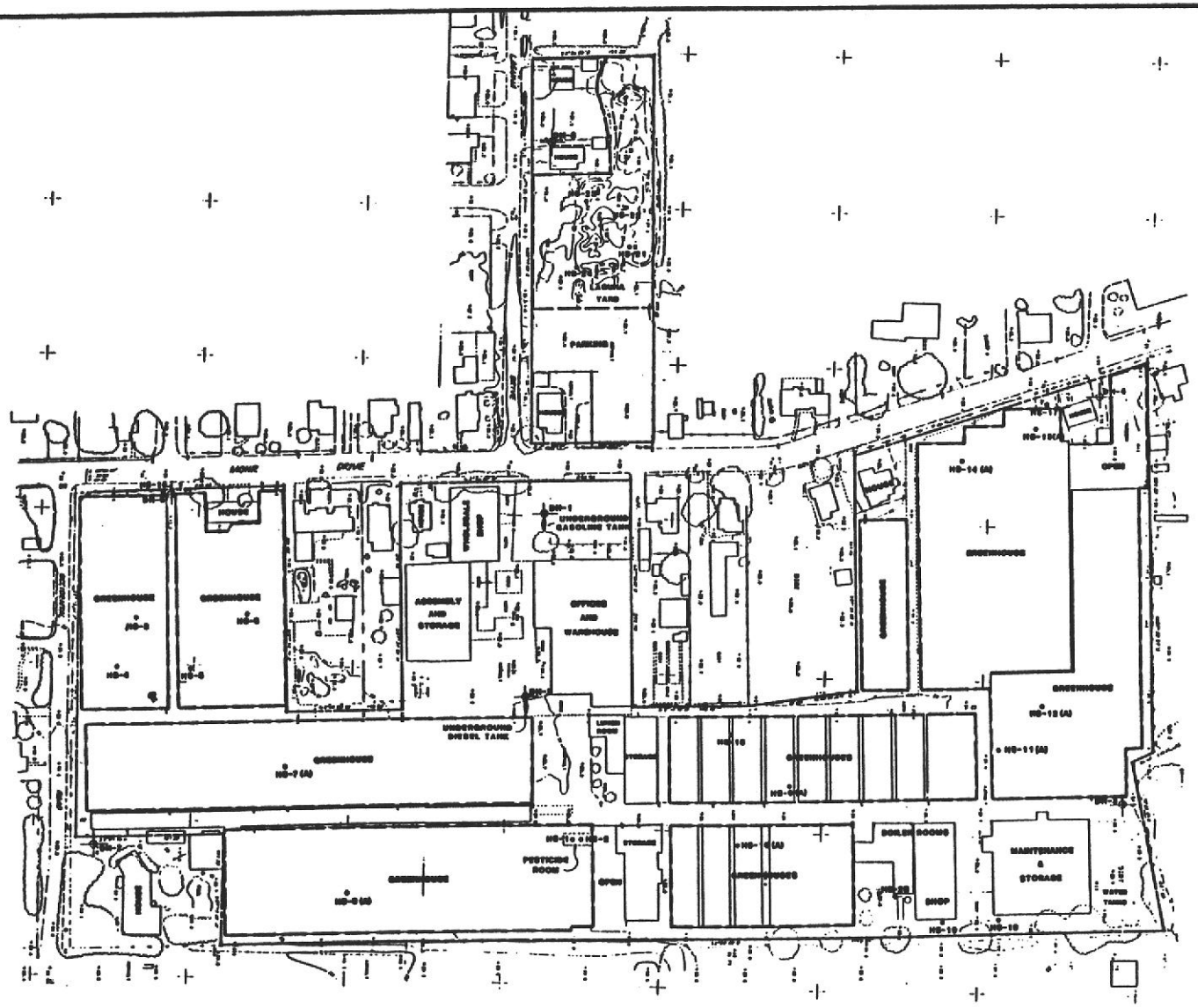
2.1 CHEMICAL ASSESSMENT OF THE SUNNYSIDE NURSERY SITE

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


N
LEGEND
EXPLORATION DRILL HOLE (ENVIRONMENTAL)
EXPLORATION DRILL HOLE (GEOTECHNICAL)
SHALLOW SOIL SAMPLE ("A" DESIGNATES RESAMPLE)
PROPERTY BOUNDARY
FENCE
MITIGATION BOUNDARY (APPROX.)
SCALE (IN FEET)
1" = 70'
TERRATECH
SITE PLAN
SUNNYSIDE NURSERY
HAYWARD, CALIFORNIA
FIGURE 1
PROJECT 4484/2

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.

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.

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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.

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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.

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:

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

where:

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

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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

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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

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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	
Dermal Absorption	8.8E-08	2.1E-07	
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}.

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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}.

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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}.

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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}.

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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.

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

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.

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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.

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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.

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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

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

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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.

REFERENCES for ENDOSULFAN I & II & ENDOSULFAN SULFATE

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ENDRIN

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.

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.

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