THE PLYMOUTH GROUP

June 4, 1990

Hugh Murphy
City of Hayward Fire Department
Hazardous Materials Division
22300 Foothill Blvd. Suite 507
Hayward, CA 94541

Re: Sunnyside Commons II (Mohr Drive, Hayward)

Dear Hugh:

Enclosed here are copies of Appendix B and Appendix C, which supplement the original health risk assessment report for the proposed Sunnyside Commons project (Environmental Risk Sciences, June 1989). This supplemental report addresses the soils and groundwater analysis at Sunnyside Commons II. If you have any questions, please contact me at (415) 691-4314. Thank you.

Sincerely,

THE PLYMOUTH GROUP

Millie Allred

Millie Allred
Assistant Project Manager

cc: Pam Evans, Alameda County of Environmental Health (w/ encl)

1616 N. Shoreline Boulevard
Mountain View, CA 94043-1316
Telephone (415) 960-8570
FAX (415) 968-5619

APPENDIX B

APPENDIX B

SUNNYSIDE COMMONS II -- SOIL ANALYSIS

1. INTRODUCTION

Appendix B supplements the original health risk assessment for the proposed Sunnyside Commons project in Hayward, California (ERS, 1989). Recently, Terratech has collected soil samples from an adjacent property called Sunnyside Commons II. These samples were analyzed for the organochlorine pesticides (EPA Method 8080) and detected 4,4'-DDT, 4,4'-DDD, 4,4'-DDE and the PCB (Aroclor 1254) in soil. This appendix presents the sampling data and derives estimates of potential human health risk and appropriate soil clean-up levels for the detected PCB compounds. The methodology for this analysis follows precisely the methods used in the earlier health risk assessment. The reader should refer to the text of the previous HRA for a more detailed discussion of the health risk assessment methodology.

2. HAZARD IDENTIFICATION

Ten soil samples have been obtained by Terratech personnel at the Sunnyside Commons II property. Eight samples (HS-1 through HS-8) were collected at depths ranging from 12 to 18" below the ground surface. The remaining two soil samples (HS-4B and HS-9) were obtained at depths ranging from 36-42" and 30-36", respectively.

Analytical laboratory results for the ten soil samples are shown in Table 2-1. Note that only five of the soil samples (HS-1,2,3, 4, and 4B) have been analyzed for the EPA Method 8080 organochlorine pesticides, while the PCBs were analyzed in all ten soil samples. Four samples (HS-1 - HS-4) were analyzed for carbamate

TABLE 2-1

SUNNYSIDE COMMONS II -- SOIL ANALYSIS

Organochlorine Pesticides and PCBs

(All values in mg/kg)

LOCATION	DDD	DDE	DDT	PCBs (Aroclor 1254)
HS-1 (12-18")	<0.016	0.019	<0.016	<0.16
HS-2 (12-18")	0.018	0.023	0.019	<0.16
HS-3 (12-18")	<0.016	<0.016	<0.016	4.1
HS-4 (12-18")	0.04	0.40	0.36	<0.16
HS-5 (12-18")	NA	NA	NA	<0.16
HS-6 (12-18")	NA	NA	NA	0.34
HS-7 (12-18")	NA	NA	NA	4.1
HS-8 (12-18")	NA	NA	NA	1.2
Site Average Concentration (12-18")	0.02	0.11	0.10	1.3
DEEPER SOIL				
HS-4B (36-42")	0.041	0.17	0.12	<0.16
HS-9 (30-36")	NA	NA	NA	<0.16

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pesticides. No carbamates were detected in soil at the property.

Table 2-1 indicates that DDT, DDD and DDE are the only organochlorine pesticides detected in soil at the Sunnyside Commons II property. Site average soil concentrations at a depth of 12-18" range from 0.02 for DDD to 0.11 mg/kg for DDE. The non-detects are assumed as a soil concentration one-half of their respective detection limit. These average soil concentrations are less than the levels detected at the Sunnyside Commons property (page 10 --0.64 mg/kg for DDT and 0.21 mg/kg for DDE for the worst-case exposure scenario (ERS, 1989). Because the Sunnyside Commons property HRA estimated a total lifetime cancer risk for DDT/DDE as 7.0E-07 (page 29) using the worst-case soil levels, the lower DDD/DDE/DDT soil concentrations detected at the Sunnyside Commons II property would result in even lower lifetime risks to human health. Therefore, the DDT and DDE detected in soil at the Sunnyside Commons II property will not be considered further in this appendix.

The polychlorinated biphenyl (PCB) Aroclor 1254 was detected in four of the eight 12-18" soil samples at a range of 0.34 mg/kg to 4.1 mg/kg. The four samples indicating non-detected soil levels had detection limits of 0.16 mg/kg. Assuming the non-detects PCB soil levels at one-half the detection limit, a site average concentration of 1.3 mg/kg has been estimated for PCBs at a depth ranging from 12-18". PCBs were not detected in the two deeper soil samples. The objective of this appendix is to estimate the lifetime human health risks that may potentially result from direct contact with these PCB soil levels in the 12-18" soil samples.

3. DOSE-RESPONSE ASSESSMENT

Dose-response assessment presents the health criteria derived by regulatory agencies that are used to estimate potential human

health risks. As explained in detail in the earlier HRA, health criteria are available for both potential human carcinogens and noncarcinogens. PCB compounds are known to induce cancer in laboratory rats. It should be noted that the most positive carcinogenicity data has been derived for Aroclor 1260. Aroclor 1254, the compound detected at the Sunnyside Commons II property, has not been proved to be a positive animal carcinogen, however, to be health conservative, it is assumed that the Aroclor 1254 compound has a carcinogenic potency similar to Aroclor 1260. Based on the data for the Aroclor 1260 isomer, the U.S. EPA has derived a cancer potency factor (CPF) for PCBs of 7.7E+00 $(mg/kg\text{-}day)^{-1}$ (U.S. EPA, 1990).

A toxicity and environmental fate profile for PCBs is presented in section 6.

4. EXPOSURE ASSESSMENT

The exposure routes considered applicable for PCBs are the same direct contact routes used in the previous HRA, soil ingestion and dermal absorption. Exposure levels are estimated for the worst-case conditions and are assumed to remain constant over an entire lifetime of a potentially exposed individual. Table 4-1 presents the lifetime worst-case exposure levels for PCBs.

Soil Ingestion Exposures

Soil ingestion exposures will occur when individuals have direct contact with surface soils. The previous HRA has explained in detail the studies considered in estimating the rate of soil contact. The worst-case assessment uses the data published by the California Department of Health Services (DHS) to represent the most health conservative study published at this time (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. This method presents a lifetime daily soil consumption rate of approximately 150 mg/day (DHS, 1987). Note that this value is assumed to be an upper-bound estimate and has been derived specifically for a residential exposure scenario.

The daily exposure level for PCBs in soil that is assumed to occur over an entire lifetime of exposure for the maximum exposed individual (MEI) is estimated using the following equation:

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

where:

Soil concentration = the site average PCB concentration presented in Table 2-1 (1.3 mg/kg);

Soil consumption = estimated lifetime soil ingestion rate assumed to be 1.5E-04 kg/day (150 mg/day) for the worst-case scenario:

GI factor = absorption rate of PCBs via soil ingestion, assumed to be 100%;

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

Dermal Absorption Exposures

Dermal absorption exposures would occur when organic chemicals adsorbed to soil come in contact with exposed skin. The rate that

soil-bound organic compounds may penetrate the skin barrier is assumed as an upper-bound estimate based on the rate of 2,167 mg/day developed by the DHS (DHS, 1987).

The following equation estimates the upper-bound daily exposure levels of the PCBs via dermal absorption exposures for 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 = site average PCB soil concentration presented in Table 2-1 (1.3 mg/kg);

SLR = maximum skin loading rate = 2,167 mg/day for the worst-case scenario;

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

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

Table 4-1 presents the worst-case scenario PCB exposure levels for the PCBs detected in soil at the Sunnyside Commons II property for both the soil ingestion and dermal absorption exposure routes.

5. RISK CHARACTERIZATION

The risk characterization section presents the lifetime upper-bound estimates of human health risk to the hypothetical MEI resulting from potential lifetime exposure to the PCBs detected in soil at the Sunnyside Commons II property. The MEI is assumed to

TABLE 4-1

Summary Exposure Table

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

(All units = mg/kg-day)

Exposure Route	**PCBs**
Soil Ingestion	2.8E-06
Dermal Absorption	4.0E-07
TOTAL DAILY EXPOSURE	3.2E-06

Note: "E-" notation refers to powers of 10; e.g. 2.8E-06 = 2.8×10^{-6}.

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have access to the Sunnyside Commons II property every day throughout his entire lifetime.

Because PCBs are known animal carcinogens and potential human carcinogens, the carcinogenic risk methodology is employed using the following equation that estimates lifetime cancer risk:

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

Cancer risks attributable to exposure to the detected PCBs in soil are calculated individually for each exposure pathway. The total lifetime cancer risk is estimated by summing the cancer risks for PCBs for the two exposure routes.

The cancer risks estimated for the maximally exposed individual (MEI) potentially exposed to soil contaminants for an entire lifetime are presented in Table 5-1. The estimated worst-case lifetime cancer risk to the MEI based solely on PCBs exposure is 2.5E-05, or approximately 2 and one-half cases of cancer per one hundred thousand exposed individuals.

Using the estimated lifetime cancer risk of 2.5E-05 for the MEI based on a soil PCB concentration of 1.3 mg/kg, a soil clean-up level of 0.05 mg/kg for soil-bound PCBs is estimated. Assuming that the PCBs at the Sunnyside Commons II is remediated to a level of 0.05 mg/kg, a lifetime cancer risk of 9.5E-07 would result for the maximum exposed individual (MEI) based on the risk assessment methodology used in this analysis.

TABLE 5-1

Lifetime Cancer Risks

Worst-Case Risk Estimates to the MEI

Exposure Route	**PCBs**
Soil Ingestion	2.2E-05
Dermal Absorption	3.1E-06
TOTAL LIFETIME CANCER RISK	2.5E-05

Note: "E-" notation refers to powers of 10; e.g. 2.2E-05 = 2.2×10^{-5}.

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6. TOXICITY AND ENVIRONMENTAL FATE PROFILE FOR PCBs

Introduction

First synthesized in 1881, polychlorinated biphenyls (PCBs) have been used commercially since the 1930s. Their low flammability, high heat-resisting capacity and low electrical conductivity made them valuable compounds in a wide range of products, from fluorescent light bulbs to hydraulic fluid, and most importantly, electric transformers and capacitors. Though the toxic effects of PCBs were first documented in 1936, it was not until the late 1960s, following a pollution incident in Japan, that their dangers became widely appreciated. In 1977, the manufacture, sale and distribution of PCB products in the U.S. was restricted to sealed systems, and they were banned in 1979 (U.S. EPA, 1984). A 1976 EEC directive banned the use of PCBs, except in sealed equipment, in Europe.

Physical/Chemical and Environmental Fate Properties

PCBs consist of a mixture of chlorinated biphenyls that contain a variable number of substituted chlorine atoms on two aromatic rings The commercial PCB mixtures manufactured in the U.S. are known as Aroclors, followed by a 4-digit number. The first two digits indicate the type of mixture (e.g. those with "12" are chlorinated biphenyls), and the last two indicate the percent weight of chlorine in the mixture. Aroclor 1254 is thus a chlorinated biphenyl mixture, containing approximately 54%, by weight, of chlorine.

The Aroclors are not very volatile, nor are they very soluble in water. They are, however, fat soluble. PCBs, therefore, adsorb readily to organic matter in soils and do not tend to leach from most soil matrices. PCBs may be removed from the atmosphere by wet

and dry deposition, and by reaction with $OH^{\cdot}$ radicals and ozone. PCBs can be degraded in soils and water, depending on their chlorine content and the characteristics of the environment. The salient physical/chemical properties of Aroclor 1254 relevant to potential human exposure are shown below.

Physical/Chemical Properties of Aroclor 1254

Molecular weight (g/mol)	Approx. 328
Vapor pressure (mm Hg @ 25°C)	7.7E-05
Water solubility (mg/l @ 25°C)	0.012
Log octanol/water partition coefficient	6.03
K_{oc}	5.3E+05

Source: U.S. EPA, 1984.

Bioaccumulation

The high octanol/water partition coefficients and low water solubilities of PCBs suggest that they have a high tendency to bioaccumulate in living organisms. This is confirmed by reported bioconcentration factors for PCBs in freshwater fish of 3,000 for brook trout (Salvelinus fontinalis) muscle, and 274,000 for the whole body of a fathead minnow (Pimephales promelas) (U.S. EPA, 1980).

Toxicokinetics

PCBs are readily absorbed from the gut following their ingestion. Studies have shown that greater than 90% of PCBs administered by gavage is absorbed by rats (Albro and Fishbein, 1972), and that they are extensively absorbed from the gut of Rhesus monkeys (Allen et al., 1974). Studies have also indicated that the absorption of

PCBs following inhalation exposure is comparable to that following ingestion (Benthe et al., 1972). The dermal absorption of Aroclor 1242 in a benzene/hexane solution has been studied in rhesus monkeys, and was found to be 15-34% of administered dose (Wester et al., 1983).

Following their absorption, PCBs tend to accumulate in adipose tissue, due to their high lipophilicity (Matthews and Dedrick, 1984).

Qualitative Description of Health Effects

Carcinogenic Potential

The available epidemiological data do not indicate a causal relationship between human exposure to PCBs and cancer (IARC, 1974; U.S. EPA, 1988).

The carcinogenic potency of PCBs to laboratory animals has been tested in several bioassays. Kimura and Baba (1973) exposed rats to dietary levels of Kanechlor-400 (PCB mixture similar to Aroclor 1260) for 22-80 weeks at levels for 38.5 to 616 ppm. Although early mortality made the study inconclusive, precancerous liver lesions were observed in all female rats that ingested more than a total dose of 1,200 mg Kanechlor. No such symptoms occurred in the male rats. Male mice exposed to 500 ppm Kanechlor-500 for 32 weeks developed hepatocellular carcinomas at a significant level (Ito et al., 1973). At lower dose levels, and with less chlorinated PCB mixtures, no such effect was noted. Female Sherman rats exposed to 100 ppm dietary Aroclor 1260 for 21 months suffered statistically significant increases in liver tumors (Kimbrough et al., 1975). The National Cancer Institute exposed male and female Fischer 344 rats to dietary levels of 0, 25, 50 and 100 ppm Aroclor 1254 for 728-735 days (NCI, 1978). Although four adenocarcinomas and one carcinoma of the gastrointestinal tract were observed in

the treated rats, the incidence did not seem to be dose-related. This led to the conclusion that Aroclor 1254 may not be carcinogenic under the conditions of the bioassay (NCI, 1978). In a further study, male and female Sprague-Dawley rats were exposed to 100 ppm Aroclor 1260 for 16 months, 50 ppm for eight months, and control diets for 5 months (Norback and Weltman, 1985). A statistical increase in the total tumor incidence rate in males and females was observed. Aroclor 1254 was tested for carcinogenicity in a mouse bioassay, the results of which were inconclusive (Kimbrough and Linder, 1974). Inconclusive results were also reported for a rat bioassay using Aroclor 1254 (Schaeffer et al., 1984). The last two studies suggest that lower chlorinated PCBs may not be carcinogenic.

Genotoxic Potential

The results of mutagenicity bioassays conducted on PCBs have been overwhelmingly negative. Schoeny et al. (1979) obtained negative results in the Salmonella typhimurium bioassay, both with and without rat liver enzyme activation. Further tests have also elicited simliar results (Schoeny, 1982; Wyndham et al., 1976). Studies using both rat and Drosophila mutagenicity bioassays have reported a similar lack of mutagenic potential in PCBs (Green et al., 1975a).

Reproductive Effects

Reproductive effects of PCB exposure have been reported in rhesus monkeys, rabbits, mice, and mink. Female rhesus monkeys exposed to 2.5 or 5 mg/kg dietary PCBs for 18 months suffered irregular menstrual cycle length and changes in progesterone levels (Barsotti and Allen, 1975). These effects have been confirmed in subsequent studies, along with fetotoxic effects such as reduced fetal body weight (Barsotti et al., 1976; Allen et al., 1979. Aroclor 1242 has been reported to cause complete reproductive failure in mink

at levels of 5 or 10 mg/kg, and Aroclor 1016, reduced reproductive performance at 20 mg/kg diet (Bleavins et al., 1980). Reproductive interference has also been noted for Aroclor 1254, at dietary levels of 2 mg/kg (Aulerich and Ringer, 1977).

PCB's have no known or clearly defined teratogenic effects in mammals, although they have been shown to cross the placenta (Kimbrough et al., 1978). Maternal ingestion of PCBs has been linked to dark brown staining of the skin of newborn babies (Funatsu et al., 1972; Miller, 1971).

Acute/Chronic Effects

Several chronic and subchronic studies of the effects of exposure to PCBs have been carried out. The predominant effect is liver toxicity, with some effects noted in the gut, skin, and blood lipid biochemistry (U.S. EPA, 1984).

Quantitative Description of Health Effects

The Carcinogen Assessment Group (CAG) of U.S. EPA considers PCBs to be Group B2, or probable human, carcinogens, based on sufficient evidence of carcinogenicity in animals, and inadequate or lacking human data (U.S. EPA, 1990). Based on the liver tumor incidence observed in rats (Norback and Weltman, 1985), CAG has derived a Cancer Potency Factor for ingested PCBs of 7.7 $(mg/kg\text{-}day)^{-1}$ (U.S. EPA, 1990). It is important to note that this CPF is based on exposure to Aroclor 1260. There are no data to support the derivation of a CPF for inhaled PCBs (U.S. EPA, 1990).

No Reference Doses for noncarcinogenic effects of exposure to PCBs have been derived by U.S. EPA or the DHS (U.S. EPA, 1990).

7. REFERENCES

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APPENDIX C

APPENDIX C

SUNNYSIDE COMMONS II -- GROUND WATER ANALYSIS

1. INTRODUCTION

Appendix C supplements the Sunnyside Commons HRA by presenting results for the two ground water samples collected in monitoring wells at the Sunnyside Commons II property. Terratech has collected ground water samples from two wells at the property. The monitoring wells (SW-1 and MW-3) are located approximately 6 feet apart. The ground water samples detected only endosulfan I, endosulfan II, and endosulfan sulfate from the EPA Method 8080 organochlorine pesticide analysis. In addition, no volatile organic compounds (VOCs) have been detected in well SW-1. This appendix derives a health based ground water clean-up level for endosulfan consistent with the approach used in the HRA.

2. HAZARD IDENTIFICATION

Ground water samples have been obtained from two monitoring wells located on the Sunnyside Commons II property (SW-1 and MW-3). Each well has been sampled only once and analyzed for the organochlorine pesticides/PCBs (EPA Method 8080). Only endosulfan I, endosulfan II, and endosulfan sulfate were detected in these analyses. In addition, no volatile organic compounds were detected in SW-1. Table 2-1 presents the ground water data from these two sampling rounds.

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TABLE 2-1

SUNNYSIDE COMMONS II -- GROUND WATER ANALYSIS

Organochlorine Pesticides and PCBs

(All values in ug/L - ppb)

LOCATION	ENDOSULFAN I	ENDOSULFAN II	ENDOSULFAN SULFATE
SW-1 Sampling date: (1/25/90)	5.0	4.5	5.1
MW-3 Sampling date: (5/18/90)	0.37	0.17	0.16

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3. DOSE-RESPONSE ASSESSMENT

Endosulfan I, endosulfan II, and endosulfan sulfate are known to exhibit noncarcinogenic effects in exposed laboratory animals. The U.S. EPA has published a reference dose (RfD) of 5.0E-05 mg/kg-day based on potential kidney toxicity in mammals (U.S. EPA, 1990). The reader should refer to the original HRA for the toxicity profile for endosulfan.

4. ESTIMATE OF GROUND WATER CLEAN-UP LEVEL FOR ENDOSULFAN

The RfD of 5.0E-05 for endosulfan can be used to estimate a ground water clean-up level assuming that the water beneath the property would be used as a primary source of drinking water for an entire lifetime. This clean-up level is estimated as:

5.0E-05 mg/kg-day x 70 kg x 1 day/2 liters x 1000 ug/1 mg = 1.75 ug/l.

This analysis assumes that all the endosulfan compounds are equally toxic and, thus, the summation of the three endosulfan compounds should not exceed a ground water concentration of 1.75 ug/l in order to minimize the potential impact to public health.

5. REFERENCES

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