HUMAN HEALTH RISKS FROM CHEMICAL AND BIOLOGICAL COMPONENTS OF RECLAIMED WATER

SANTA ROSA SUBREGIONAL LONG-TERM WASTEWATER PROJECT

Prepared for

City of Santa Rosa

and

U.S. Army Corps of Engineers

March 1996

Prepared by

PARSONS ENGINEERING SCIENCE, INC.

PLANNING · DESIGN · CONSTRUCTION MANAGEMENT

1301 MARINA VILLAGE PARKWAY, ALAMEDA, CA 94501 · 510/769-0100

OFFICES IN PRINCIPAL CITIES

723129/94-14

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EXECUTIVE SUMMARY

This screening public health risk assessment evaluates the potential health risks to the public associated with exposure to reclaimed water from the Laguna Wastewater Treatment Plant (Laguna Plant). Risks from exposure to chemicals and to pathogens in the reclaimed water are considered.

APPROACH

The initial step of the screening methodology is designed to screen out chemicals and biological components that clearly do not present a hazard and do not warrant further analysis. Those components that do not pass the screen do not necessarily present a health hazard, but require further evaluation. The screening is accomplished by using "worst case" assumptions (e.g., maximum detected concentrations, direct exposure to reclaimed water as a drinking water source, continuous exposure period, etc.). Chemicals and biological components that do not present a hazard under these conditions would not present a hazard under more probable conditions (e.g., concentrations less than maximum due to dilution, degradation, or filtration, periodic exposure, etc.). The assumptions made for the screening step overestimate the risk for all components and these estimates of risk are used only as an indication that the component requires further evaluation.

The chemical and pathogen risk assessments consist of a summary and evaluation of existing chemical and biological analytical data, an exposure assessment, a toxicity assessment, and a risk characterization. The quantitative portion of this risk assessment is based on the conservative (health protective) assumption that an exposed human population would use undiluted reclaimed water as a domestic water supply and would receive additional exposure by consuming fish caught from the surface water bodies that receive reclaimed water. This approach overestimates the potential health risks posed by exposure to the reclaimed water through the discharge or reuse scenarios of the Subregional Wastewater Project. Although long term exposure to undiluted reclaimed water is not proposed and is not expected to occur, this approach was selected to screen out chemical and biological components that present a very low probability of human health impact.

CHEMICAL AND BIOLOGICAL ANALYTICAL DATA

Chemical components that may potentially occur in the Laguna Plant's reclaimed water were identified from water quality data obtained from the Laguna Plant's quarterly water monitoring program (1988-1995) and from data collected as part of the Subregional Wastewater Project (1994-1995). About 30 inorganic[DJB1] and 200 organic[DJB2] chemicals were assayed in at least one set of analyses during this 7 year period (not all chemicals were measured at each sampling event). Of these, 23 inorganic and 26 organic chemicals were detected at or above their reporting limits in at least one sample. Of the 49 chemicals detected at least once, eight inorganic chemicals (ammonia, calcium, fluoride, magnesium, nitrate, phosphate, potassium, and sodium) and one organic chemical (chloroform) were detected each time that they were assayed. The remaining

chemicals were detected in as few as one sampling event or as many as all-but-one sampling event. Maximum detected concentrations are used to screen chemicals for their human health effects.

Total coliform, heterotrophic bacteria, *Salmonella*, *Shigella*, *Legionella*, *Giardia*, *Cryptosporidium*, and enteric viruses were assayed in samples collected from the Laguna Plant effluent, Delta Pond, and the Russian River upstream of its confluence with the Laguna de Santa Rosa in October, November, and December 1994. In addition, data for total coliform were obtained from the Laguna Plant's waste discharge permit records (1991-1994). *Salmonella*, *Shigella*, *Legionella*, and enteric viruses were never detected above sample reporting limits in any samples. Total coliform and heterotrophic bacteria were detected in all three sources and their concentrations were highest in the Russian River and Delta Pond. *Giardia* cysts were detected only in the effluent during the late 1994 sampling event, although they have previously been detected in the Russian River (CH2M Hill 1993). *Cryptosporidium* oocysts were detected only in the Russian River.

EXPOSURE PATHWAYS

Exposure pathways considered for both the potential chemical and biological components of reclaimed water include domestic use of water for drinking and bathing, recreational use, irrigation use, and consumption of fish that have contacted reclaimed water. The potentially exposed population is assumed to be residents of Sonoma County or northern Marin County who may come into contact with the chemical or biological components in reclaimed water via these exposure pathways. Sensitive subpopulations (e.g., young children, pregnant or nursing women, the elderly, persons with suppressed immune systems) are identified and discussed.

TOXICITY ASSESSMENT AND RISK CHARACTERIZATION

Hazard quotients are used to evaluate the noncarcinogenic health effects of the chemical components. A hazard quotient of less than 1.0 indicates that a chemical is not expected to produce an adverse health effect. Excess cancer risks are used to evaluate carcinogenic health effects of the chemical components. In general, excess cancer risks greater than one in a million (1×10^{-6}) to one in one-hundred thousand (1×10^{-5}) are considered by the State of California to pose a significant threat to human health (Title 22, California Code of Regulations, §12703). For this assessment an excess cancer risk of 1.0×10^{-6} is used as a screening level for carcinogenic health effects.

The analysis of risk from the detected biological components in the Laguna Plant effluent is evaluated by comparing the data to a known infective dose (*Giardia*), to background concentrations (total coliform and heterotrophic bacteria), and to regulatory standards (total coliform).

Chemical and biological components that do not pass the screen are examined further and are evaluated as to their environmental fate (chemical or biological degradation), attenuation (loss of viability in the case of pathogens), filtration, dilution by groundwater or surface water,

background concentrations, and comparison to State and Federal drinking water standards (Maximum Containment Limits, MCLs) and reclaimed water standards.

Noncarcinogenic Effects

Of the detected chemicals, the maximum concentrations of ammonia, nitrate and nitrite in undiluted effluent yield hazard quotients greater than 1.0. The hazard quotients associated with the mean concentrations of these three chemicals are less than 1.0. No other chemical yields a hazard quotient greater than 1.0.

The maximum and mean concentrations for nitrate (maximum, 50.5 mg/L and mean, 16.3 mg/L) and combined nitrate and nitrite (maximum, 57.8 mg/L and mean 16.6 mg/L) in undiluted effluent are greater than the State and Federal drinking water standards of 10 mg/L. Plant treatment processes (i.e., nitrification and denitrification) would be upgraded before the project is built and are expected to lower the combined nitrate and nitrite level, although no specific design criteria for this level has been set. The mean concentrations currently yield a combined hazard index less than 1.0. Ammonia also would be reduced by the nitrification process and, based on historical data for the periods when the treatment plant was performing nitrification, both the maximum (14.9 mg/L) and mean (2.0 mg/L) concentrations for ammonia yield hazard quotients less than 1.0. Dilution of reclaimed water with surface waters and groundwaters upon release and uptake of these chemicals by terrestrial and aquatic plants would be expected to further reduce the potential human health hazard from nitrate, nitrite, and ammonia, and these chemicals would not present a significant human health hazard via most of the expected exposure pathways. However, in rural areas near some potential reservoir sites, where groundwater already contains elevated nitrate, some wells may yield water which contains nitrate levels that exceed the State and Federal drinking water standards if dilution alone is counted on to reduce nitrate levels.

Carcinogenic Effects

The excess cancer risks associated with the maximum concentrations of arsenic, chromium, chloromethane, 1,4-dichlorobenzene, methylene chloride, bromodichloromethane, chloroform, dibromochloromethane, aldrin, I-lindane, K-lindane, and heptachlor exceed the screening level of 1.0×10^{-6} (range: 1.1×10^{-6} to 8.9×10^{-5}). Because these risks are based on the maximum detected concentrations in effluent, they overestimate the potential risk from exposure to reclaimed water via the expected exposure pathways. Mean concentrations, which have been calculated from samples collected over several years, yield a better estimate of the risk associated with effluent, and thus undiluted reclaimed water. The excess cancer risks associated with the mean concentrations of arsenic, chromium, bromodichloromethane, chloroform, and aldrin exceed 1.0×10^{-6} (range: 2.2×10^{-6} to 5.4×10^{-5}) while the remaining chemicals yield excess cancer risks less than 1.0×10^{-6}. Dilution of undiluted reclaimed water with surface and ground waters, chemical and biological degradation (of organics), and adsorption of chemicals to soils and sediments would further reduce chemical concentrations in reclaimed water and the actual risk values.

Of the five chemicals which have mean concentrations that yield excess cancer risks greater than 1.0×10^{-6}, aldrin was detected infrequently (three times out of 19 samples) at a maximum concentration (0.00003 mg/L) just above its minimum analytical reporting limit (0.00001 mg/L). In addition, aldrin, an organochlorine pesticide, is no longer registered for use in the United States. The other four chemicals were detected frequently (50 to 100 percent of samples) but have both mean and maximum concentrations that are below drinking water standards. The combined maximum (0.0571 mg/L) and mean (0.0125 mg/L) concentrations of bromodichloromethane, chloroform, and dibromochloromethane (disinfection byproducts, or DBPs) are less than the State and Federal MCL (0.100 mg/L) for DBPs and the maximum concentrations of arsenic (0.004 mg/L) and chromium (0.014 mg/L) are less than their State (0.050 mg/L for both) and Federal (0.050 mg/L for arsenic and 0.100 mg/L for chromium) MCLs. The Laguna Plant has indicated that it is studying replacing chlorination with ultraviolet light disinfection, which could be expected to decrease the concentration of DBPs in the treatment plant effluent. It is therefore unlikely that these five chemicals would present a significant long-term human health risk.

No mean concentration for any potentially carcinogenic chemical exceeds a State or Federal MCL. Three chemicals (methylene chloride, heptachlor, and bis (2-ethylhexyl) phthalate) have a maximum concentration that exceeds a State and/or Federal drinking water standard. The maximum concentration of methylene chloride (0.006 mg/L) exceeds both its State and Federal MCL (0.005 mg/L); the maximum concentration of bis (2-ethylhexyl) phthalate (0.006 mg/L) exceeds its State MCL (0.004 mg/L) and equals its Federal MCL (the excess cancer risk associated with bis (2-ethylhexyl) phthalate is less than 1.0×10^{-6}); and the maximum concentration of heptachlor (0.00003 mg/L) exceeds the State MCL (0.00001 mg/L) but not the Federal MCL (0.00004 mg/L). Each chemical exceeds its MCL only once out of 19 samples and was otherwise detected infrequently (heptachlor was detected once and methylene chloride and bis (2-ethylhexyl) phthalate were detected five times each).

Given that no mean concentration exceeds an MCL and that additional factors (e.g., dilution, volatilization, biological and chemical degradation, changes in treatment plant processes) would reduce chemical concentrations in reclaimed water it is unlikely that the potentially carcinogenic chemical components of reclaimed water would present a significant long-term human health risk via the expected exposure pathways.

Fish Consumption

Water quality data and organismal data from the Kelly Farm Pond bioaccumulation study and from the Regional Water Quality Control Board's Toxic Substances Monitoring Program on the Russian River are used to evaluate the potential human health hazard associated with the consumption of fish. Water quality data are compared to water quality criteria proposed by the USEPA for the combined consumption of aquatic organisms and water and for the consumption of aquatic organisms alone. Organismal data are examined to evaluate whether chemicals are bioaccumulating in the food web. Although a number of chemicals exceed the water quality criteria that are based on the combined exposure of fish and drinking water ingestion, site-specific data collected from the Kelly Farm Pond bioaccumulation study and the Toxic

Substances Monitoring Program show no evidence that chemicals are bioaccumulating to concentrations that would present a significant human health hazard.

Biological Components

Total coliforms were detected only once in the effluent [2 most probable number (MPN)/100 mL] during the October through December 1994 sampling events, but were detected in all samples collected from Delta Pond (280 MPN/100 mL) and the Russian River. Total coliform counts from the Laguna Plant's historical data range from non-detect (<2 MPN/100 mL) to 170 MPN/100 mL. Heterotrophic bacteria were detected in all samples but were higher in Delta Pond [3,100 colony forming units (CFU)/1 mL] and the Russian River (31 to 610 CFU/1 mL) than in the Laguna Plant effluent (8 to 21 CFU/1 mL). The higher numbers for Delta Pond and the Russian River are not unexpected given that they are open bodies of water that would attract wildlife.

Salmonella, *Shigella*, *Legionella*, and enteric viruses were never detected above sample reporting limits in any samples. *Cryptosporidium* oocysts were not detected in the Laguna Plant effluent or Delta Pond but were present in the Russian River (0.4 to 2.7 oocysts/ 100 L). *Giardia* cysts were detected in two samples from the Laguna Plant effluent but not in Delta Pond or the Russian River. *Giardia* cysts have previously been detected in the Russian River upstream of the confluence with the Laguna de Santa Rosa (CH2M Hill 1993). Given their low concentrations, information on background concentrations in the Russian River, and the expected filtering effects of soils and base materials, the biological components of reclaimed water would not present a significant human health hazard via the expected exposure pathways.

1 INTRODUCTION

This document describes a screening public health risk assessment conducted for the Santa Rosa Subregional Long-Term Wastewater Project (Subregional Wastewater Project). It evaluates the potential health risks to the public associated with exposure to treated reclaimed water from the Laguna Wastewater Treatment Plant (Laguna Plant). Risks from exposure to chemicals and to pathogens potentially in the treated reclaimed water are considered.

SCOPE OF ASSESSMENT

The California Department of Toxic Substances Control's (DTSC) Preliminary Endangerment Assessment Manual (PEA Manual) was used as a source of guidance for the preparation of the chemical risk assessment (DTSC 1994a). However, the PEA Manual was designed to be used for the evaluation of hazardous waste sites and some modifications were made to adapt the methodology to the current project. To prepare the assessment the following tasks were performed:

- An evaluation of the chemical and biological analytical data to identify chemicals and biological agents in the fresh effluent from the Laguna Plant, in the Russian River upstream from its confluence with Mark West Creek (and thus not under the influence of discharges from the Laguna Plant), and in stored final effluent from the Delta Pond;

- An exposure assessment to evaluate the nature, duration, and magnitude of human exposure to the chemicals and biological agents in the Laguna Plant's effluent;

- A toxicity assessment to provide information on the potential for the detected chemicals and biological components to cause adverse health effects; and

- A risk characterization to integrate the exposure assessment and the chemical and pathogen toxicity information to quantitatively or qualitatively estimate chemical and pathogenic doses and potential carcinogenic and noncarcinogenic health risks due to the chemicals and biological agents in the Laguna Plant's effluent.

Although the PEA Manual does not address hazards associated with pathogens, a similar series of steps is used to evaluate these hazards. Section 3 summarizes the methodology used for assessing the potential human health risk from pathogens that may occur in reclaimed water.

The quantitative portion of this risk assessment is based on the conservative (health protective) assumption that an exposed human population would use undiluted reclaimed water as a domestic water supply and would receive additional exposure to chemicals in reclaimed water by consuming fish caught from surface water bodies that receive

reclaimed water. Although this is an unlikely scenario (it overestimates the potential health risks posed by exposure to the reclaimed water through the discharge or reuse scenarios of the Subregional Wastewater Project), it was selected to screen out chemicals that present a very low probability of human health impact. All detected chemicals and pathogens are evaluated in this screening process. Chemicals and pathogens that pass the screen for exposure to undiluted reclaimed water would not present a human health hazard and further analysis of their environmental fate (chemical or biological degradation), attenuation (loss of viability in the case of pathogens), dilution by groundwater or surface water, background concentrations, and comparison to regulatory standards is not necessary. Chemicals that do not pass the screen are examined further and these factors are considered in the appropriate sections on risk characterization (Sections 2.6 and 3.5).

The risk assessment report is divided into four sections. Section 2 follows this introduction and evaluates the human health risk from exposure to the chemicals contained in the Laguna Plant's effluent. Section 3 evaluates the human health risk from exposure to the biological agents contained in the Laguna Plant's effluent. Section 4 is a list of references.

2 RISK FROM CHEMICAL COMPONENTS

2.1 CHEMICAL COMPONENTS IN LAGUNA PLANT WASTEWATER

Chemical components that may potentially occur in the Laguna Plant's reclaimed water were identified from water quality data obtained from the Laguna Plant's quarterly water monitoring program (1988-1995) and from data collected as part of the Subregional Wastewater Project (1994-1995). A total of about 30 inorganic[DJB3] and 200 organic[DJB4] chemicals were assayed in at least one set of analyses during this 7 year period (not all chemicals were measured at each sampling event). Of these, 23 inorganic and 26 organic chemicals have been detected at or above their reporting limits in at least one sample (Table 2.1-1). Chemicals that were never reported at or above their respective reporting limits are not included in the table. A list of all chemical analytes and their reporting limits is presented in the Field Sampling and Quality Assurance Plan and the Reclaimed Water Quality Technical Report for this study (Merritt Smith Consulting 1995a, 1995b).

Of the 49 chemicals detected at least once, eight inorganic chemicals (ammonia, calcium, fluoride, magnesium, nitrate, phosphate, potassium, and sodium) and one organic chemical (chloroform) were detected each time that they were assayed. The remaining chemicals in Table 2.1-1 were detected in as few as one sampling event or as many as all-but-one sampling event. When a chemical was not detected at or above its reporting limit it is reported as a "non-detect." The reporting limits for the non-detects are presented in Table 2.1-1. The reporting limits have varied over the 7 years in which samples were collected and are usually listed as a range. For samples collected as described in the Field Sampling and Quality Assurance Plan, the reporting limits were set lower than the State and Federal drinking water standards (Maximum Contaminant Limit, MCL) when an MCL was available. Reporting limits can be affected by such things as the analytical method selected for the analysis, sample handling, and sample characteristics (e.g., presence of interfering compounds).

The mean concentration for each chemical in Table 2.1-1 was calculated using all quantified values and most non-detect values. Some non-detect values were excluded from the calculation of the mean when the reporting limits of the analytical method were several times greater than the highest detected value. The following criteria were applied to non-detects when calculating mean concentrations:

- When one-half of the reporting limit for a non-detect was more than twice the maximum detected concentration of any quantified value, that non-detect value was not used to calculate the mean;

- For values reported as non-detect, one-half of the reporting limit was used to calculate the mean.

Table 2.1-1

Potential Chemical Constituents of Reclaimed Water

Chemical	Concentration Range (mg/L)	Mean Concentration (mg/L)	Reporting Limit(s) (mg/L)	Number of Detects	Number of Samples
Inorganics					
aluminum	N.D. - 0.15	0.03	0.01 - 0.10	20	27
ammonia	N.D. - 40.3	4.1	0.1 - 0.5	49	49
arsenic	N.D. - 0.004	0.002	0.001 - 0.005	25	30
asbestos, MFL[(1)]	N.D. - 0.56	0.25	0.05 - 0.28	2	4
barium	N.D. - 0.11	0.02	0.02 - 0.05	4	27
boron	N.D. - 0.60	0.48	0.10	17	18
cadmium	N.D. - 0.007	0.001	0.0002 - 0.01	6	89
calcium	22 - 63	31	N/A	19	19
chromium	N.D. - 0.014	0.002	0.001 - 0.02	49	90
copper	N.D. - 0.04	0.01	0.005 - 0.10	88	90
cyanide	N.D. - 0.03	0.01	0.005 - 0.01	6	11
fluoride	0.18 - 0.31	0.22	N/A	4	4
lead	N.D. - 0.012	0.005	0.001 - 0.04	19	90
magnesium	15 - 23	19	N/A	18	18
mercury	N.D. - 0.0002	0.0001	0.0002 - 0.001	1	91
nickel	N.D. - 0.010	0.004	0.002 - 0.02	56	90
nitrate	0.3 - 50.5	16.3	N/A	49	49
nitrite	N.D. - 7.3	0.3	0.01	45	48
phosphate	0.1 - 8.4	4.3	N/A	49	49
potassium	7 - 24	11	N/A	28	28
silver	N.D. - 0.010	0.001	0.0001 - 0.01	40	88
sodium	58 - 150	80	N/A	28	28
zinc	N.D. - 0.28	0.03	0.01 - 0.10	82	90
Volatile Organics					
acetone	N.D. - 0.0060	0.0042	0.002 - 0.01	2	14
bromomethane	N.D. - 0.0014	0.0003	0.0001 - 0.0005	1	19
carbon disulfide	N.D. - 0.0370	0.0040	0.0005 - 0.005	3	14

Table 2.1-1 (continued)

Potential Chemical Constituents of Reclaimed Water

Chemical	Concentration Range (mg/L)	Mean Concentration (mg/L)	Reporting Limit(s) (mg/L)	Number of Detects	Number of Samples
chlorobenzene	N.D. - 0.0001	0.0001	0.0001	1	19
chloromethane	N.D. - 0.0050	0.0005	0.0001 - 0.001	1	19
1,4-dichlorobenzene	N.D. - 0.0009	0.0006	0.0005	10	13
ethylbenzene	N.D. - 0.0010	0.0002	0.0001 - 0.0005	1	19
methylene chloride	N.D. - 0.0060	0.0008	0.0001 - 0.003	5	19
tetrachloroethylene	N.D. - 0.0006	0.0002	0.0001 - 0.0005	2	19
toluene	N.D. - 0.0004	0.0002	0.0001 - 0.0005	2	19
1,1,1-trichloroethane	N.D. - 0.0002	0.0002	0.0001 - 0.0005	1	19
xylenes	N.D. - 0.0002	0.0002	0.0001 - 0.0005	1	18
Trihalomethanes					
bromodichloromethane	N.D. - 0.0110	0.0022	0.0005	22	23
chloroform	0.0024 - 0.0440	0.0099	0.0005	23	23
dibromochloromethane	N.D. - 0.0021	0.0004	0.0001 - 0.0005	4	22
total trihalomethanes[(2)]	0.0036 - 0.057	0.0129	N/A	23	23
Phthalates					
di-n-butyl phthalate	N.D. - 0.0019	0.0012	0.005 - 0.0005	2	19
bis (2-ethylhexyl) phthalate	N.D. - 0.0060	0.0028	0.0006 - 0.020	5	19
diethyl phthalate	N.D. - 0.0070	0.0009	0.0005 - 0.001	2	19
Pesticides					
aldicarb sulfone	N.D. - 0.0018	0.0011	0.0008	2	4
aldicarb sulfoxide	N.D. - 0.0019	0.0008	0.0005	2	4
aldrin	N.D. - 0.00003	0.00001	0.00001 - 0.00005	3	19
DCPA (Dacthal)	N.D. - 0.0003	0.0002	0.0002	2	4
Endosulfan II	N.D. - 0.00001	0.00001	0.00001 - 0.00005	1	19
α-lindane	N.D. - 0.00003	0.00001	0.00001 - 0.00005	2	19
γ- lindane	N.D. - 0.00009	0.00002	0.00001 - 0.00002	8	19

Table 2.1-1 (continued)

Potential Chemical Constituents of Reclaimed Water

Chemical	Concentration Range (mg/L)	Mean Concentration (mg/L)	Reporting Limit(s) (mg/L)	Number of Detects	Number of Samples
heptachlor	N.D. - 0.00003	0.00001	0.00001 - 0.00005	1	19
Radioactivity					
Gross alpha, GPV[3]	1.3 - 5.5 pCi/L	2.8 pCi/L	N/A	4	4
Gross beta, GPV	11.9 - 12.7 pCi/L	12.3 pCi/L	N/A	4	4

N/A - not available
N.D. - not detected
[1] Asbestos values are reported as millions of fibers per liter (MFL).
[2] Trihalomethanes include chloroform, bromoform, bromodichloromethane, and dibromochloromethane. Bromoform was not detected at or above the reporting limit for any sample. One half the reporting limit for bromoform was used to calculate the maximum and mean concentrations of trihalomethanes.
[3] Radioactivity values are reported as greatest probable value (GPV).

2.2 LOW TOXICITY INORGANICS

Five inorganic chemicals (calcium, magnesium, phosphate, potassium, and sodium) were detected, but are generally regarded as being of minimal environmental concern. They are of minimal concern because:

- all are naturally-occurring chemicals,
- none are carcinogenic, and all are of low toxicity to humans, most animals, and most plants,
- calcium, magnesium, potassium, and sodium are essential nutrients for humans and other animals and phosphate occurs naturally in various forms in humans and most other animals,
- these chemicals are rarely, if ever, quantitatively evaluated in risk assessments,

- although all are abundant in nature and widely used in industry, the Agency for Toxic Substances and Disease Registry (ATSDR) has not included any of them on its priority list of hazardous substances (ATSDR 1992b), and

- none are listed on the United States Environmental Protection Agency's (USEPA) Integrated Risk Information System (IRIS) or Health Effects Assessment Summary Tables (HEAST), two sources of toxicity information commonly used in risk assessments.

For these reasons, these five chemicals are not analyzed in the quantitative risk assessment.

2.3 POTENTIALLY EXPOSED POPULATION

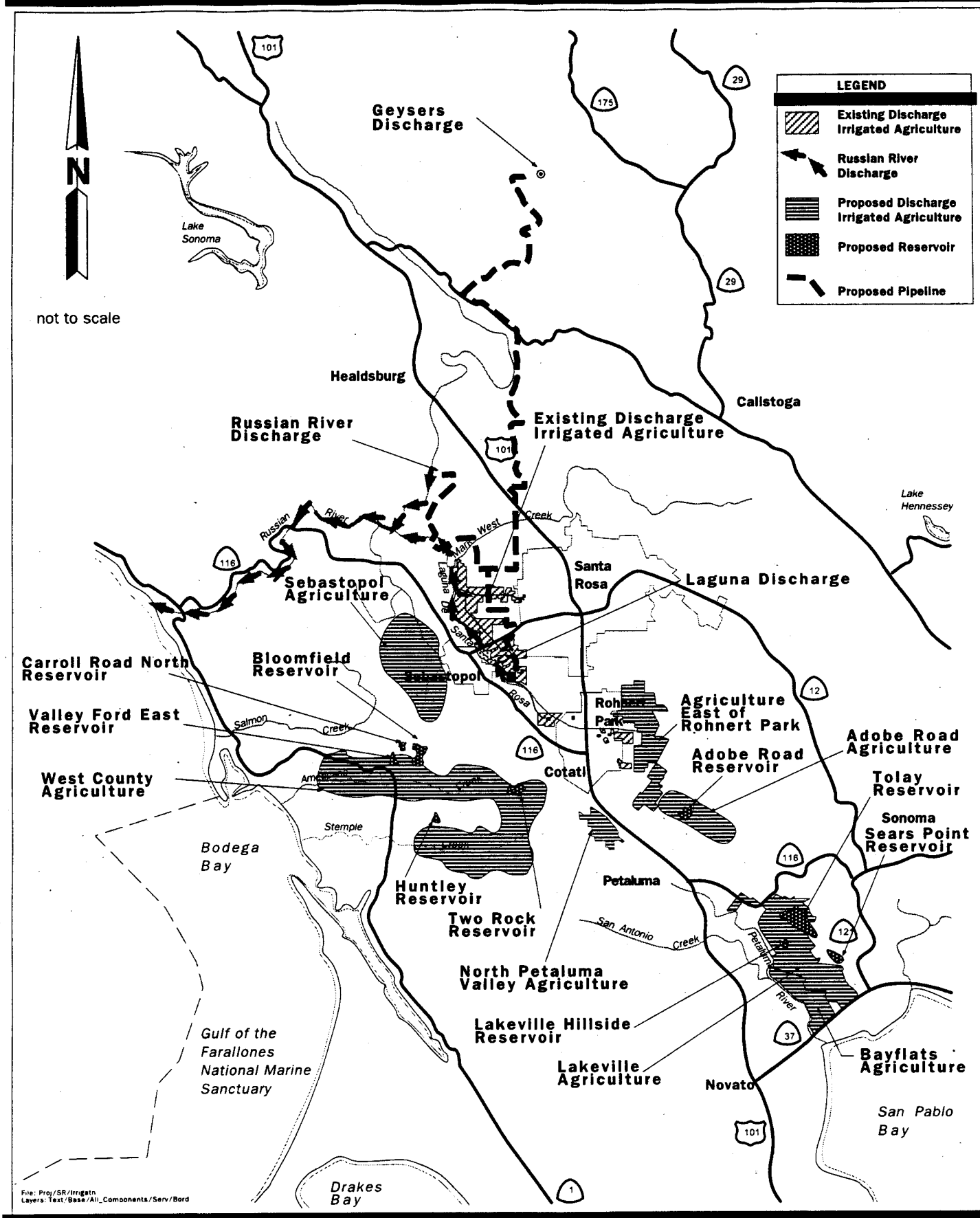
Components of the Subregional Wastewater Project (reservoirs, pipelines, irrigation areas, etc.) that may provide an exposure point for people to reclaimed water are located mostly within Sonoma County (Figure 2.3-1). Some reservoirs and agriculture irrigation areas are within watersheds that extend into rural areas of northern Marin County. Sonoma County is generally characterized by relatively compact cities and communities separated by areas of agricultural use and other open space. About two-thirds of the total county population lives in nine incorporated cities or towns and 24 unincorporated communities. Cities within the project area include Santa Rosa, Rohnert Park, Cotati, Sebastopol, and Petaluma.

The density of the cities and communities within the project area varies from highly urban to semi-rural. Agriculture occupies a major portion of the county lands outside of the cities and communities with a diversity of operations, including vineyards, orchards, dairies, forage crops, specialty crops and livestock. Along the Pacific Coast, fishery related uses are significant, while major recreation and tourism-related uses occur along the coast and the Russian River. Northern Marin County is primarily agricultural.

Because of the geographic size and the demographic diversity of the project area, the potentially exposed population is diverse and includes subpopulations (e.g., young children and infants, the elderly, pregnant women) that may be more sensitive to chemical exposures than the general population. These sensitive subpopulations are discussed in the following section. A discussion of subpopulations that may be sensitive to the biological components (e.g., people who have suppressed immune systems) of reclaimed water is included in Section 3.4.

Sensitive Subpopulations

Infants and children are not only particularly sensitive to the toxic effects of many chemicals but also may have elevated exposure rates. Children and infants generally have a greater absorptive capacity than adults, presumably for the enhanced uptake of nutrients to aid in development. Unfortunately, this characteristic also means that many



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Santa Rosa
Subregional Long-Term Wastewater Project

PROJECT COMPONENTS

Figure 2.3-1
March 13, 1996

toxic chemicals are also more readily absorbed. Chemicals that are fat soluble and bioaccumulate may be found in human breast milk. Thus infants may receive elevated doses of chemicals during nursing. Because children have the greatest potential exposure, the exposure equations used in this risk assessment to determine level of hazard were those derived by the DTSC for children (see Section 2.6). The cancer risk equations account for combined exposure during childhood and adult years averaged over a 70-year lifetime.

Pregnant and nursing women may be more susceptible to the toxic effects of chemicals due to the demands placed on their physiology by fetuses or nursing infants. For example, a woman's iron reserves may be depleted and this depletion of iron may facilitate absorption of toxic metals such as cadmium. People attempting conception may be susceptible to the toxic effects of certain chemicals, such as organochlorine pesticides which have been shown to affect sperm production and motility (Parsons Engineering Science 1995a and references therein). Elderly people may have increased susceptibility due to decreased function of detoxification and excretory processes, compromised bone integrity, declining cardiovascular and pulmonary function and generally poor health and inadequate nutrition. This risk assessment takes the increased sensitivity of these subpopulations into account by using toxicity values (derived by the USEPA or the DTSC) that have been adjusted to be protective of sensitive subpopulations (see Section 2.5).

2.4 PATHWAYS OF EXPOSURE

This section summarizes the potential pathways via which humans could be exposed to the chemical components potentially contained in reclaimed water. Reclaimed water has been proposed for discharge to the Russian River and for several reuse options, including urban and agricultural irrigation and recharge of the Geyser's geothermal steamfield, in the five alternatives being analyzed for the Subregional Wastewater Project. Each of the potential exposure pathways that may result from these reuse options is discussed in the following paragraphs.

Discharge to the Russian River

Currently, the North Coast Regional Water Quality Control Board (RWQCB) permits the Subregional System to discharge at a maximum rate of 1 percent of river flow except during periods of low river flow when discharge rates of 5 percent are allowed. Discharge occurs only from October 1 through May 14.

The five alternatives being analyzed for the Subregional Wastewater Project propose discharge rates to the Russian River which vary from 1 up to 20 percent of river flow. Design rates of 1, 5, 10, and 20 percent of river flow correspond to maximum rates of 1.0, 7.3, 13.4 and 28.3 percent, respectively (Parsons Engineering Science 1995b). River flows, which are used to calculate these percentages, would be based on measurements made downstream of the confluence of the Laguna de Santa Rosa with the Russian River

at the Hacienda Bridge. Thus, effluent as a percentage of surface water at the proposed discharge points in the Laguna de Santa Rosa or on the Russian River may exceed these percentages. All rates are expressed as monthly averages. Design rates are expected to be exceeded during 2 to 4 months over a 70-year period. The maximum rates are expected to occur only one month in 70 years. The actual volume and frequency of discharge would vary due to operational and seasonal considerations, including irrigation needs, storage levels, and weather. No change in the discharge season is proposed.

The discharge to the Russian River may occur through continued discharges into the Laguna de Santa Rosa creeks or through a direct discharge outfall on the Russian River. In both cases, water would be held in a storage pond(s) prior to release. The two principal discharge locations on the Laguna de Santa Rosa creeks are at Delta Pond, located south of Highway 12 and at the Meadow Lane Ponds west of Llano Road (Figure 2.3-1). These discharge points are located about 7 and 14 miles, respectively, upstream from the confluence of the Laguna de Santa Rosa (Mark West Creek) with the Russian River. The discharge point for the outfall on the Russian River would be located on the east bank of the Russian River, about 4 miles upstream of the river's confluence with the Laguna de Santa Rosa. The water would be stored in Delta Pond prior to discharge.

Possible exposure pathways that may result from discharge to the Russian River include movement of surface water to groundwater where it may be used as a domestic water supply (private or municipal), consumption of fish that have been exposed to surface water (for chemicals that bioaccumulate), and exposure to surface water during recreational use of the river.

Domestic Water Supply

Some reclaimed water may reach domestic wells that are near the Russian River. However, filtration by sands and gravels that underlie the Russian River and dilution of reclaimed water (by river water and then groundwater) would occur between the discharge point and any domestic water supply intakes. Previous water modeling work has indicated that both private and municipal wells will pump varying percentages of water from the river and from groundwater recharge from precipitation and infiltration, depending upon their distance from the river, their pumping rates, and local hydrogeologic conditions (CH2M Hill et al. 1993). The percentage of reclaimed water in the river water that is subject to uptake by these wells depends upon the discharge rate, river flows, and the distance between the discharge point and the domestic water intake.

The Sonoma County Water Agency (SCWA) withdraws water from under the Russian River in a series of caissons, or Ranney collectors (caissons 1 through 5). Caissons 1 and 2 are located about 1.7 miles upstream of the confluence with the Laguna de Santa Rosa and 2.3 miles downstream from the proposed Russian River outfall. Caissons 3, 4, and 5 are located on the north bank of the Russian River near the confluence with the Laguna de Santa Rosa. Caisson 5 is about 1,000 feet upstream of the confluence, caisson 4 is located a few hundred feet downstream of the confluence, and caisson 3 is located about

1,000 feet downstream of the confluence. The only SCWA drinking water intake along the Russian River that is under the influence of surface water is caisson 5 (Flugum 1995).

Because of the variety of the domestic water uses (e.g., drinking, bathing, showering, washing), all routes of exposure (ingestion, inhalation of volatile organics, and dermal absorption) would be possible for chemicals present in a domestic water supply.

Fish Consumption

Fish that are resident or migrate in the Russian River system downstream of the discharge points and that are subsequently consumed by humans, may be a source of chemical exposure for chemicals that bioaccumulate. Potential human exposure would vary depending on such factors as the type of fishing (sport or sustenance), location relative to discharge points, and the quantity and species of fish consumed.

The river provides resources for 46 species of fish, a variety of which are caught for sport and consumption. No sustenance-level fishing occurs on the Russian River, although warm water fishers within the Laguna de Santa Rosa (e.g., at the Occidental Road Bridge on the Laguna de Santa Rosa) probably consume a greater number and variety of fish species than sport fishers on the Russian River (Cox 1995). While fishing occurs year-round within the Russian River system, the type of fishing and the target species varies with the season.

During the wet season, the majority of fishing is for salmon and steelhead trout on the Russian River. In the spring, American shad are the primary target species, but most are caught for sport (catch and release). During the spring, summer, and early fall, warm water fishing in the Laguna de Santa Rosa accounts for the majority of the fishing activity. The target species during this period are varied (e.g., carp, suckers, bass, green sunfish, blackfish, and brown bullheads) and a greater proportion of the fish are believed to be caught for consumption (Cox 1995). Crayfish may also be caught and consumed from the Laguna de Santa Rosa during this period.

Recreational Use

Exposure to reclaimed water (diluted by river water) is possible for persons fishing, swimming or wading in the Russian River downstream of the proposed discharge point and/or the river's confluence with the Laguna de Santa Rosa. However, swimmers and waders are likely to have minimal exposure because the discharge season for the Subregional System runs from October 1 through May 14 whereas the recreational facilities on the river that are conducive to swimming and wading are generally operated only during the summer months. Sonoma County facilities, such as the Wohler Bridge boating access, the Forestville River Access and the proposed access at Steelhead Beach, are open only from Memorial Day through Labor Day (Sales 1995). Some recreational use for swimming and wading may occur at other times of the year but these events would be expected to be of limited duration and frequency. While fishing is permitted

year-round, fishers would be expected to ingest smaller quantities of river water and have reduced dermal contact.

Urban and Agricultural Irrigation

Alternatives 2 and 3 propose an expansion of existing urban irrigation areas. Potential irrigation areas include business parks, golf courses, parks and schools in the Fountaingrove and Bennett Valley areas. Alternatives 2 and 3 also propose an expansion of existing agricultural irrigation areas. About 18,000 acres of privately owned potential agricultural irrigation property is being considered in the West County area, 2,500 acres in the Sebastopol area, and 15,000 acres in the South County area. Of this total potential acreage, about 4,000 to 7,000 acres, depending upon the alternative, would actually be required. The irrigation season typically runs from March through November although there is a Winter Irrigation Program which can be implemented during dry winters.

State of California regulations restrict the use of reclaimed water for irrigation in areas where food is handled and drinking water fountains are located (Title 22, California Code of Regulations, §60310). The State also requires all publicly accessible areas where reclaimed water is used to be posted with conspicuous signs that include the warning, "RECLAIMED WATER - DO NOT DRINK." The use of reclaimed water is also restricted within 50 feet of domestic water supplies to prevent contamination of the wells. Some inhalation or dermal absorption from spray irrigation is possible, but these routes would be orders of magnitude lower than exposure to domestic water via these routes because of decreased exposure duration and because volatile organic chemicals will dissipate outdoors. In addition, some types of irrigation, such as drip, present essentially no exposure.

Prior to use for irrigation, reclaimed water would be stored in ponds or reservoirs. Existing ponds are fenced and do not present a complete exposure pathway for the general public. No new storage ponds are proposed as part of the Subregional Wastewater Project.

Reservoirs

Nine potential reservoir sites are proposed for the West and South County (Figure 2.3-1). One or two reservoirs may be needed, depending upon the project alternative selected. All reservoir sites are located in rural areas and would be constructed by damming a natural drainage or valley by means of an earth-filled embankment dam. Reservoir properties would be fenced with barbed wire cattle fence, although the reservoir itself would not be fenced. Because of the remote siting of the reservoirs and the limited access, exposure to surface water in the reservoirs is not considered a complete pathway.

Some potential exists for water to migrate from the reservoirs to groundwater via infiltration and into nearby domestic water supplies. The potential for this infiltration is currently unknown, however, hydrogeologic investigations are being conducted at each of the candidate reservoir sites to address this concern. The amount of reclaimed water

that reaches nearby wells, if any, depends upon the rate of infiltration, other local hydrogeologic conditions, and the pumping rates of the domestic wells. The quality of the water that reaches the domestic wells depends upon the initial quality of the reclaimed water and groundwater, the dilution of the reclaimed water with groundwater, and any natural degradation or filtration processes that might occur as water moves through the aquifer.

Geysers Injection

The Geysers Injection Alternative (Alternative 4) consists of pumping reclaimed water from the Delta Pond to the Geysers area north of Healdsburg (Figure 2.3-1). The water would be pumped through about 35 miles of pipeline to two 1-million-gallon storage tanks overlooking the Geysers steamfield area. From these tanks the water would be gravity fed through piping to about ten injection wells located around the central and northwest portion of the Geysers area. The injection wells inject water into the Geysers geothermal reservoir located in excess of 3,000 feet below ground surface. Because the piping and storage of reclaimed water for this Alternative is a closed system and because the water is injected at great depth, the geysers injection is not considered a complete pathway.

Some Russian River discharge is associated with the Geysers Injection Alternative. The pumping system to the Geysers would be designed to handle output from the Laguna Plant 95 percent of the time. During periods when output would exceed the pumping system capacity (5 percent of the time), a portion of the flow would be discharged to the Russian River. The maximum discharge rate as a percentage of river flow would be less than 1 percent.

Summary of Exposure Pathways

Potentially complete pathways have been identified for the Russian River discharge, urban and agricultural irrigation and for the storage of water in reservoirs (Table 2.4-1 and Figure 2.4-1). Most potential pathways are direct exposures to surface water or groundwater used as a domestic water source or for recreational purposes. All of these pathways would include a dilution of reclaimed water with surface water or groundwater between the release point (e.g., Russian River discharge, reservoir leakage) and the exposure point (e.g., domestic well intake, recreational water use). The consumption of fish presents an additional potential pathway for water released to the Russian River. This pathway could be potentially significant for chemicals that bioaccumulate.

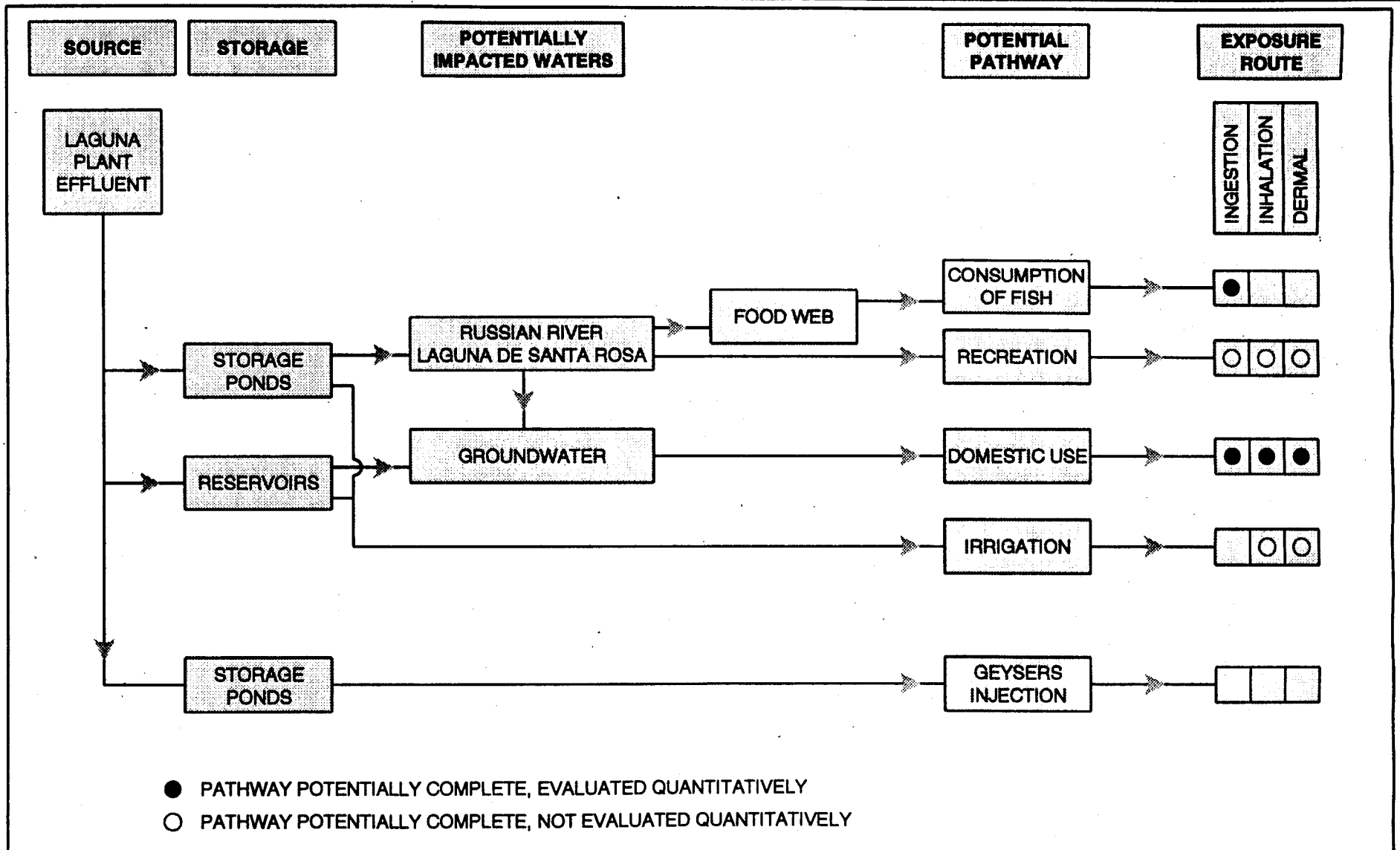
2.5 TOXICITY ASSESSMENT

This section summarizes the available toxicity information for all organic chemicals and most inorganic chemicals listed in Table 2.1-1. Some inorganic chemicals (i.e., calcium, magnesium, phosphate, potassium and sodium) that are relatively non-toxic (they are essential human nutrients) and that were detected at low concentrations have been

Table 2.4-1

Summary of Possible Exposure Pathways

Pathway	Comments
Russian River to Domestic Water Supply	Potentially complete pathway; Russian River flows will dilute discharge; Groundwater will dilute discharge; Discharge occurs for a maximum of 7.5 months per year
Fish Consumption	Potentially complete pathway; Potentially significant for chemicals that bioaccumulate
Recreational Use	Potentially complete pathway; Discharge occurs during portion of year when swimming and wading uses are low; Fishers protected by clothing
Urban and Agricultural Irrigation	Potentially complete pathway; Orders of magnitude smaller exposure than domestic water use scenario; Ingestion discouraged by State-mandated posting of warning signs
Storage Ponds	No probable complete pathway; Ponds are fenced and public access is restricted
Reservoirs to Domestic Water Supply	Potentially complete pathway; Groundwater will dilute water that infiltrates from reservoirs
Geyser Injection	No probable complete pathway except periodic river discharge; Closed system of pipes and tanks; Water injected in excess of 3,000 feet below ground surface



Santa Rosa
Subregional Long-Term Wastewater Project

CONCEPTUAL MODEL FOR POTENTIAL EXPOSURE PATHWAYS

FIGURE 2.4-1

REV. 3 SR904-11.DWG 03/05/96

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excluded for the reasons presented in Section 2.2. No organic chemicals have been excluded. Toxicity information for all other chemicals detected above the reporting limit in at least one sample is summarized in Table 2.5-1. Unless otherwise indicated, the information in this section was obtained from the National Library of Medicine's Hazardous Substances Database (HSDB) and the USEPA's IRIS (USEPA 1995a, 1995b, 1995d). Both information sources are available on the National Library of Medicine's data network system (TOXNET).

One widely held view among toxicologists is that toxic chemicals produce adverse human health effects only when an individual's exposure (via inhalation, ingestion, or dermal contact) exceeds some threshold level. The threshold level is expressed as a chemical dose. The chemical dose at the threshold level is the amount of chemical at which no effect is observed in the exposed individual. Only when the chemical dose exceeds the threshold are adverse health effects expected.

In practice, the chemical dose is expressed as the weight of chemical per kilogram of body weight per day (e.g., milligrams/kilogram/day or mg/kg/day). For non-carcinogenic effects this threshold level is a number greater than zero. For carcinogenic effects current DTSC and USEPA methods assume that no dose, however small, is risk-free. The assumed threshold level for carcinogenic effects is therefore zero. Because of this difference in the assumptions regarding threshold levels, the toxicity assessment information is derived differently for the two types of adverse health effects.

Assessment of Noncarcinogenic Effects

The toxicity assessment of noncarcinogenic effects is based on available toxicological information that has been derived from either animal or human studies. Chemical exposures in these studies may be acute (a brief exposure of a few minutes to a few days), subchronic (a few weeks to months), or chronic (usually includes at least a tenth of the life span of a species, generally six months or more). Chronic exposures have the lowest thresholds for adverse effects and are most commonly used to derive chemical reference doses (RfDs) for risk assessments.

The RfD is an estimate of the lowest daily intake level of a chemical that will not produce adverse health effects. The RfD incorporates a margin of safety (called an uncertainty factor) and has been calculated by the USEPA to protect the most sensitive members of the population. The RfD is derived from the experimentally determined no-observed-adverse-effect level (NOAEL) or the lowest-observed-adverse-effect-level (LOAEL). The NOAEL is a dose in a toxicity test that does not produce an observable adverse effect. The LOAEL is the lowest dose in a toxicity test that produces an observable adverse effect. The uncertainty factor (a number between 1 and 1,000) accounts for intraspecies and interspecies variations, for limited or incomplete data, for evaluating the significance of adverse effects, and for sensitive human subpopulations. In practice, an

Table 2.5-1

Potential Health Effects of Detected Chemicals[1]

Chemical	Target of Potential Health Effects
Inorganics	
aluminum	Nervous, respiratory, and skeletal systems, developing fetus
ammonia	Eye irritant and respiratory system
arsenic	Cancer (USEPA Group A), nervous, respiratory, gastrointestinal, and blood-forming systems, skin
asbestos	Cancer (USEPA Group A), respiratory and gastrointestinal systems
barium	Cardiovascular system (blood pressure)
boron	Reproductive and gastrointestinal systems, liver, kidneys, brain
cadmium	Cancer (USEPA Group B1), endocrine system, kidneys
calcium	Essential nutrient
chromium	Cancer (USEPA Group A) for chromium (VI), respiratory and gastrointestinal systems, essential nutrient for chromium (III)
copper	Gastrointestinal system, essential nutrient
cyanide	Red blood cells, inhibition of oxygen uptake
fluoride	Skeletal system (fluorosis)
lead	Cancer (USEPA Group B2), central nervous and endocrine systems, kidneys
magnesium	Essential nutrient
mercury	Nervous system, kidneys, brain
nickel	Respiratory, gastrointestinal and immune systems
nitrate	Methemoglobinemia (blue baby syndrome)
nitrite	Methemoglobinemia (blue baby syndrome)
phosphate	Essential nutrient
potassium	Essential nutrient
silver	Argyria (bluish-gray discoloration of the skin)
sodium	Essential nutrient
zinc	Gastrointestinal system, red blood cells, essential nutrient

Table 2.5-1 (continued)

Potential Health Effects of Detected Chemicals[1]

Chemical	Target of Potential Health Effects
Volatile Organics	
acetone	Central nervous system, kidneys, stomach
bromomethane	Central nervous and respiratory system, skin and eyes
carbon disulfide	Central nervous system, heart, liver, developing fetus
chlorobenzene	Nervous system, liver and kidneys
chloromethane	Cancer (USEPA Group C), central nervous system
1,4-dichlorobenzene	Cancer (USEPA Group C), respiratory irritant, liver
ethylbenzene	Respiratory irritant, liver, kidney, nervous system
methylene chloride	Cancer (USEPA Group B2), central nervous system, liver, kidneys
tetrachloroethylene	Suspected carcinogen, central nervous system, liver, kidneys
toluene	Central nervous system, liver, kidneys
1,1,1-trichloroethane	Central nervous system, skin and eye irritant
xylenes	Central nervous system
Trihalomethanes	
bromodichloromethane	Cancer (USEPA Group B2), central nervous system, liver, kidneys
chloroform	Cancer (USEPA Group B2), central nervous system, liver, kidneys
dibromochloromethane	Cancer (USEPA Group C), central nervous system, liver
Phthalates	
bis(2-ethylhexyl)phthalate	Cancer (USEPA Group B2), endocrine system, liver, kidneys, testes
diethyl phthalate	Reduced growth, endocrine system
di-n-butyl phthalate	Reproductive and endocrine systems
Pesticides	
aldicarb sulfone	Nervous and endocrine system
aldicarb sulfoxide	Nervous and endocrine system
aldrin	Cancer (USEPA Group B2), nervous and endocrine systems, liver
DCPA (Dacthal)	Respiratory and endocrine system, liver, kidneys, thyroid, eyes
α-lindane	Cancer (USEPA Group B2) nervous, reproductive, circulatory, and endocrine systems
γ-lindane	Cancer (USEPA Group B2) nervous, reproductive, circulatory, and endocrine systems
heptachlor	Cancer (USEPA Group B2), nervous and endocrine systems

Table 2.5-1 (continued)

Potential Health Effects of Detected Chemicals[1]

Chemical	Target of Potential Health Effects
Radioactivity	
Gross alpha	Cancer (USEPA Group A)
Gross beta	Cancer (USEPA Group A)

(1) Some of the health effects listed in this table occur only after exposure to elevated chemical concentrations. Reference doses (RfDs) are generally derived from chronic studies that examine health effects which occur at lower concentrations. A discussion of the health effects upon which the RfDs are based is presented in Appendix B.

experimentally derived NOAEL or LOAEL is divided by the uncertainty factor to determine the RfD. The RfD is therefore always equal to or less than the experimentally obtained threshold dose.

The oral and inhalation RfDs used in this risk assessment have been derived by the USEPA. An oral RfD is given for each chemical where information is available. An inhalation RfD is given for those organic chemicals that meet criteria for volatile organics set by the USEPA (USEPA 1995c). Inhalation RfDs were calculated from inhalation reference concentrations (RfCs). An inhalation RfC, expressed as the weight of a chemical per volume of air (e.g., milligram/cubic meter or mg/m^3), is an estimate of the lowest daily level of a chemical in air that will not produce adverse health effects. An RfC is converted to an equivalent inhalation RfD by multiplying the RfC by a breathing rate of 20 m^3/day and dividing it by an average body weight of 70 kg, a method recommended by the DTSC (DTSC 1994a). For some volatile chemicals, an inhalation RfC is not available. In these cases, the DTSC recommends the use of the same value for the inhalation RfD as for the oral RfD.

Assessment of Carcinogenic Effects

The toxicity assessment of carcinogenic effects is based on available toxicological information that has been derived from either animal or human studies. The DTSC and the USEPA consider the weight-of-evidence that a chemical is a carcinogen and have calculated slope factors to quantify the relative risk from exposure to a carcinogen. The weight-of-evidence classification is an objective assessment of each chemical that determines the level or strength of evidence that a substance is a human or animal carcinogen (Table 2.5-2). Slope factors have generally been calculated for chemicals in Groups A, B1, and B2 and some of the chemicals in Group C.

The oral or inhalation slope factor is an upper estimate of the probability of a response (the initiation or promotion of cancer) per unit intake of a chemical over a person's lifetime. An oral slope factor is given for each chemical where information is available. An inhalation slope factor is given for those organic chemicals that meet criteria for volatile organics set by the USEPA (USEPA 1995c). Both slope factors are expressed as the inverse of the dose. Slope factors are typically expressed as "$(mg/kg/day)^{-1}$." Most slope factors used in this report were derived by the USEPA. In some cases the DTSC has derived slope factors that differ from those derived by the USEPA (DTSC 1994b). The DTSC values have been used in deriving the "no significant risk levels" under the State's Safe Drinking Water and Toxic Enforcement Act of 1986 and the State drinking water Maximum Contaminant Levels (MCLs). DTSC values are used when they differ from the USEPA values.

Summary of Toxicity Assessment

Table 2.5-3 summarizes the toxicity values (RfDs and slope factors) for each chemical listed in Table 2.1-1 except for the five low toxicity inorganic elements, which are not

Table 2.5-2

USEPA Weight-of-Evidence Cancer Classification

Group	Definition
A	Known human carcinogen based on sufficient evidence from epidemiological or other human evidence studies
B1	Probable human carcinogen based on limited evidence of carcinogenicity in humans
B2	Probable human carcinogen based on sufficient evidence in animals and inadequate or no data in humans
C	Possible human carcinogen based on limited evidence of carcinogenicity in animals in the absence of human data
D	Not classifiable as to human carcinogenicity based on lack of data or inadequate evidence of carcinogenicity from animal data
E	No evidence of carcinogenicity from at least two reliable tests in different species of laboratory animals

Table 2.5-3

Summary of Toxicity Assessment Values

Chemical	EPA Cancer Class	RfDo	RfDi	SFo	SFi	Kp
		mg/kg/day		$(mg/kg/day)^{-1}$		cm/hr
Inorganics						
aluminum	N/A	1.0				
ammonia	N/A	1.0				
arsenic	A	0.0003		1.5		
asbestos, MFL[(1)]	A	N/A		N/A		
barium	D	0.07				
boron	D	0.09				
cadmium	B1	0.0005		N/A		
chromium	D,A	0.005		0.42		
copper	D	0.04				
cyanide	D	0.02				
fluoride	N/A	0.06				
lead	B2	N/A		N/A		
mercury	D	0.0003				
nickel	D	0.02				
nitrate	N/A	1.6				
nitrite	N/A	0.1				
silver	D	0.005				
zinc	D	0.3				
Volatile Organics						
acetone	D	0.1	0.1			0.00072
bromomethane	D	0.0014	0.0014			0.0035
carbon disulfide	N/A	0.1	0.0029			0.024
chlorobenzene	D	0.02	0.0057			0.041
chloromethane	C	N/A	N/A	0.013	0.0063	0.0042
1,4-dichlorobenzene	C	0.23	0.23	0.04	0.040	0.062
ethylbenzene	D	0.1	0.29			0.074

Table 2.5-3 (continued)

Summary of Toxicity Assessment Values

Chemical	EPA Cancer Class	RfDo	RfDi	SFo	SFi	Kp
		mg/kg/day		$(mg/kg/day)^{-1}$		cm/hr
Volatile Organics						
methylene chloride	B2	0.06	0.86	0.014	0.0035	0.0045
tetrachloroethylene	N/A	0.01	0.01	0.051	0.021	0.048
toluene	D	0.2	0.11			0.045
1,1,1-trichloroethane	D	0.09	0.09			0.017
total xylenes	D	2.0	2.0			0.08
Trihalomethanes						
bromodichloromethane	B2	0.02	0.02	0.13	0.13	0.0058
chloroform	B2	0.01	0.01	0.031	0.019	0.0089
dibromochloromethane	C	0.02		0.094		0.0039
Phthalates						
di-n-butyl phthalate	D	0.1				0.033
bis (2-ethylhexyl) phthalate	B2	0.02		0.0084		0.033
diethyl phthalate	D	0.8				0.0048
Pesticides						
aldicarb sulfone	D	0.001				0.00043
aldicarb sulfoxide	D	0.001 (2)				0.00043
aldrin	B2	0.00003		17		0.0016
DCPA (Dacthal)	D	0.01				0.033
endosulfan II	N/A	0.006				0.01
α-lindane	B2	0.0003		6.3		0.014
γ-lindane	B2	0.0003		1.1		0.014
heptachlor	B2	0.0005		3.7		0.011

Table 2.5-3 (continued)

Summary of Toxicity Assessment Values

Chemical	EPA Cancer Class	RfDo	RfDi	SFo	SFi	Kp
		mg/kg/day		$(mg/kg/day)^{-1}$		cm/hr
Radioactivity						
Gross alpha, GPV[3]	A	N/A		N/A		
Gross beta, GPV	A	N/A		N/A		

N/A. - not available
RfDo Reference dose, oral.
RfDi Reference dose, inhalation.
SFo Cancer slope factor, oral
SFi Cancer slope factor, inhalation.
Kp Dermal permeability coefficient from water.
Shading indicates that the toxicity value is not applicable to the chemical (i.e., chemical is not a carcinogen, inhalation or dermal absorption are not significant routes of uptake).

(1) Asbestos values are reported as millions of fibers per liter (MFL).
(2) No RfD was available for aldicarb sulfoxide. The RfD for aldicarb sulfone is listed.
(3) Radioactivity values are reported as greatest probable value (GPV).

being carried forward in the risk assessment for the reasons identified in Section 2.2. Brief descriptions of the data on which the RfDs and slope factors are based are presented in Appendix B. To conduct the risk characterization, one additional set of values is needed, the chemical-specific dermal permeability coefficient from water (Kp). These values are used to account for uptake of chemicals that may occur through the skin and were obtained from the DTSC PEA Manual (DTSC 1994a). They are also listed in Table 2.5-3.

2.6 RISK CHARACTERIZATION

For each chemical that has been detected in the Laguna Plant effluent and that may potentially occur in reclaimed water, an "excess cancer risk" (for carcinogenic effects) and/or "hazard quotient" (for noncarcinogenic effects) for exposure to that chemical can be calculated. There are three exceptions, asbestos, lead and radioactivity, for which toxicity values are not available. These chemicals are discussed separately. For screening purposes the cancer risks and hazard quotients have been calculated assuming that the reclaimed water would be used as a primary domestic water source, without dilution (see Section 2.6, Uncertainty, for a discussion of how biological, chemical and physical processes would affect the conclusions based on this assumption). The potential natural and artificial sources and the environmental fate of chemicals that exceed the screening criteria are described in Appendix A. Formulae derived by the DTSC (Appendix C) were used to calculate these values (DTSC 1994a).

Calculation of Hazard Quotients

The noncarcinogenic human health risk from exposure to chemicals is expressed as a hazard quotient. The hazard quotient is the ratio of the estimated dose from exposure via all potential pathways, to a value which is believed not to produce adverse health effects. Generally, a hazard quotient of less than 1.0 would indicate that the chemical is not expected to produce an adverse health effect. However, when two or more chemicals manifest the same toxic effect(s) or target the same organ(s), their hazard quotients are added together. This sum is called a "hazard index." If the hazard index is less that 1.0, the combined effect of the chemicals is not expected to produce an adverse health effect. Hazard quotients based on maximum detected values are summarized in Table 2.6-1.

The equations used to calculate the hazard quotients for this risk assessment have been derived by the DTSC (DTSC 1994a). They are included in Appendix C. These equations have been simplified by the DTSC by incorporating default values to achieve a reasonable maximum estimation of exposure in a residential setting. There are different default values for children and adults and also for different exposure scenarios (i.e., residential and industrial). The water pathway considered in this screening assessment is a summation of ingestion exposure, inhalation of volatile organics from water released indoors, and dermal exposure. The equations are therefore different for volatile and nonvolatile chemicals. The hazard quotients are calculated for the first 6 years of childhood. The default values are such that if the hazard quotient (or hazard index) is not

Table 2.6-1

Summary of Hazard Quotients and Cancer Risk Values
Based on Maximum Detected Values[(1)]

Chemical	Maximum Hazard Quotient	Maximum Excess Cancer Risk
Inorganics		
aluminum	0.010	
ammonia	**2.575**	
arsenic	0.852	**8.9×10^{-5}**
asbestos		
barium	0.100	
boron	0.426	
cadmium	0.895	
chromium	0.179	**8.8×10^{-5}**
copper	0.064	
cyanide	0.096	
fluoride	0.330	
lead		
mercury	0.043	
nickel	0.032	
nitrate	**2.017**	
nitrite	**4.665**	
silver	0.128	
zinc	0.060	
Volatile Organics		
acetone	0.008	
bromomethane	0.128	
carbon disulfide	0.839	
chlorobenzene	0.001	
chloromethane		**1.4×10^{-6}**
1,4-dichlorobenzene	0.001	**1.1×10^{-6}**
ethylbenzene	0.001	
methylene chloride	0.007	**1.6×10^{-6}**
tetrachloroethylene	0.008	6.9×10^{-7}

Table 2.6-1 (continued)

Summary of Hazard Quotients and Cancer Risk Values
Based on Maximum Detected Values[1]

Chemical	Maximum Hazard Quotient	Maximum Excess Cancer Risk
Volatile Organics		
toluene	< 0.001	
1,1,1-trichloroethane	< 0.001	
total xylenes	< 0.001	
Trihalomethanes		
bromodichloromethane	0.070	**4.3×10^{-5}**
chloroform	0.565	**3.3×10^{-5}**
dibromochloromethane	0.007	**3.0×10^{-6}**
Pesticides		
aldicarb sulfone	0.118	
aldicarb sulfoxide	0.123	
aldrin	0.064	**7.6×10^{-6}**
DCPA	0.002	
endosulfan II	<0.001	
α-lindane	0.006	**9.0×10^{-6}**
γ-lindane	0.019	**1.5×10^{-6}**
heptachlor	0.004	**1.7×10^{-6}**
Phthalates		
di-n-butyl phthalate	0.001	
bis (2-ethylhexyl) phthalate	0.020	8.1×10^{-7}
diethyl phthalate	0.001	
Radioactivity		
Gross alpha and beta		

[1] Values in **bold text** indicate a hazard index that exceeds the screening value of 1.0 or an excess cancer risk that exceeds the screening value of 1.0×10^{-6}.

Shading in the "Hazard Quotient" column indicates that no reference doses were available for the chemical. Shading in the "Excess Cancer Risk" column indicates that the chemical is not a carcinogen or that no slope factor was available.

exceeded for the first 6 years of childhood, it will not be exceeded for any other age. These equations do not include exposure from ingestion of aquatic organisms in surface water.

Calculation of Excess Cancer Risk

The carcinogenic human health risk from exposure to chemicals is expressed as a probability of excess cancer risk. The excess cancer risk is an incremental probability, such as one in a million (1×10^{-6}) or one in ten thousand (1×10^{-4}), of developing cancer over a lifetime as a result of exposure to the potential carcinogen. In general, excess cancer risks greater than 1×10^{-6} to 1×10^{-5} are considered by the State of California to pose a significant threat to human health (Title 22, California Code of Regulations, §12703; DTSC 1994a). In addition, when two or more potential carcinogens target the same organ(s), their excess cancer risks are added together. If the summed excess cancer risk is greater than 1×10^{-6}, the combined effect of the chemicals would be expected to pose a significant threat to human health. Excess cancer risks are summarized in Table 2.6-1.

The equations used to calculate the excess cancer risk for this risk assessment have been derived by the DTSC (DTSC 1994a). They are included in Appendix C. As with the assessment of noncarcinogenic effects, these equations have been simplified by the DTSC by incorporating default values to achieve a reasonable maximum estimation of exposure in a residential setting. The water pathway considered in this screening assessment is a summation of ingestion exposure, inhalation of volatile organics from water released indoors (e.g., volatilization during showering, use of dishwasher, flushing of toilet), and dermal exposure. The equations are therefore different for volatile and nonvolatile chemicals. The equations estimate the lifetime excess cancer risk from a combined exposure duration of 6 years as a child and 24 years as an adult. These equations do not include exposure from ingestion of aquatic organisms in surface water.

Characterization of Noncarcinogenic Effects

A preliminary screening of the data for noncarcinogenic effects was performed using maximum reported values. The use of these values generates maximum hazard quotients and presents a worst-case scenario. Chemicals that have maximum hazard quotients less than 1.0 and that represent only a small percentage of the overall hazard are eliminated from further characterization. Chemicals with a hazard quotient greater than 1.0 or that may be summed with another chemical to give a hazard index greater than 1.0 are evaluated further (Table 2.6-2).

Ammonia, nitrate, and nitrite have maximum hazard quotients greater than 1.0. There are six additional chemicals, arsenic, boron, cadmium, fluoride, carbon disulfide, and chloroform, that could yield a combined hazard index greater than 1.0. No other single chemical contributes more than 2 percent of the total hazard index.

Table 2.6-2

Major Contributors to the Total Hazard Index[1]

Chemical	Maximum Hazard Quotient	Mean Hazard Quotient	Basis for RfD
ammonia	**2.575**	0.260	Eye irritant and respiratory system; RfD based on taste threshold in humans
arsenic	0.852	0.519	Nervous, respiratory, gastrointestinal, and blood-forming system; RfD based on dermal effects and possible effects on blood vessels in humans
boron	0.426	0.338	Reproductive and gastrointestinal systems, liver, kidney, brain; RfD based on testicular atrophy and lack of sperm production in dogs
cadmium	0.895	0.100	Endocrine system, kidneys; RfD based on abnormally high protein levels in urine in humans
fluoride	0.330	0.232	Skeletal system (fluorosis); RfD based on objectionable dental fluorosis in children
nitrate	**2.017**	0.652	Methemoglobinemia; RfD based on methemoglobinemia in infants
nitrite	**4.665**	0.220	Methemoglobinemia; RfD based on methemoglobinemia in infants
carbon disulfide	0.839	0.090	Central nervous system, heart, liver, developing fetus; RfD based on fetal toxicity in rabbits
chloroform	0.565	0.135	Central nervous system, liver, kidneys; RfD based on cyst formation in dogs

[1] Values in **bold text** indicate a hazard index that exceeds the screening value of 1.0.

Table 2.6-2 also contains the hazard quotients calculated from the mean chemical concentrations of the nine chemicals. While the maximum hazard quotients provide a worst-case scenario, the mean hazard quotients provide more representative estimations of the potential hazard associated with exposure to reclaimed water for all chemicals and especially for chemicals that have been detected only infrequently or that have been detected at an elevated concentration in only one analysis. For these chemicals the use of a maximum reported value greatly overestimates the potential hazard associated with long-term exposure to undiluted reclaimed water.

Cadmium, for example, was detected in six of 89 samples. In addition, most "non-detects" for cadmium (about 90%) had a reporting limit of 0.001 mg/L or less. This value, which is equal to the calculated mean concentration for cadmium (Table 2.1-1), yields a hazard quotient of 0.100. Carbon disulfide was detected in only three of 14 samples. The detected values were 0.037 mg/L, 0.018 mg/L and 0.0006 mg/L. The reporting limit was 0.005 mg/L or less for all remaining samples and was 0.0005 mg/L for five of these. The hazard quotient (0.090) associated with the mean carbon disulfide concentration (0.004 mg/L) is therefore a more representative estimate of the hazard associated with this chemical. The mean hazard quotients for these chemicals are well below 1.0 and indicate that there is no significant hazard associated with exposure to reclaimed water containing these concentrations of cadmium and carbon disulfide.

Each RfD for the remaining chemicals with hazard quotients less than 1.0 (arsenic, boron, fluoride, and chloroform) is based on a different and unique health effect and it is not appropriate to sum their RfDs. In addition, the mean hazard quotient for each of these chemicals is well below 1.0 and indicates that there is no significant hazard associated with exposure to reclaimed water containing the associated mean concentration. (Arsenic and chloroform, however, are potential carcinogens and are discussed further in the section on carcinogenic effects).

Hazard Posed by Ammonia, Nitrate, and Nitrite

Nitrate toxicity is due primarily to its conversion to nitrite in the body, which oxidizes the Fe(+2) form of iron in hemoglobin to the Fe(+3) state. The resulting form of hemoglobin, called methemoglobin, does not bind oxygen, resulting in reduced oxygen transport from lungs to tissues. Low levels of methemoglobin occur in normal individuals, with typical values usually ranging from 0.5 to 2.0% of total hemoglobin. These levels occur because nitrate is a normal component of the human diet. Over 85% of nitrate intake (or more for vegetarians) comes from the natural nitrate content of vegetables such as beets, celery, lettuce and spinach.

Due to the large excess capacity of blood to carry oxygen, levels of methemoglobin of up to around 10% are not associated with any significant clinical signs. Concentrations above 10% may cause a bluish color to skin and lips (cyanosis). Most cases of infant methemoglobinemia are associated with exposure to nitrate in drinking water used to prepare infants' formula at levels greater than 20 mg/L of nitrate-nitrogen (USEPA

1995d). Cases reported at levels of 11 to 20 mg/L nitrate-nitrogen are usually associated with concomitant exposure to bacteriologically contaminated water or excess intake of nitrate from other sources.

Clinical studies of healthy babies administered controlled doses of nitrate have reported no clinical signs of methemoglobinemia for infants who received water containing up to 34.5 mg/L nitrate-nitrogen. Methemoglobin levels in these infants ranged from about 1% to 3%. Several epidemiological studies of infants exhibiting cyanosis due to methemoglobinemia have reported that no symptoms occurred at concentrations less than 10 mg/L nitrate-nitrogen. A small number of cases (less than 2% of the total) has been reported for water containing 11 to 20 mg/L nitrate-nitogen, although the diagnosis of methemoglobinemia was considered questionable in some of these cases. Many of the wells in these studies, which were often shallow with inadequate protection from surface water, contained coliform bacteria, which may have been a complicating factor.

The maximum hazard quotients for ammonia, nitrate, and nitrite are greater than 1.0 while their mean hazard quotients are less than one (Table 2.6-2). Because nitrate and nitrite induce the same adverse health effect, methemoglobinemia in infants, it is appropriate to sum the hazard quotients for nitrate and nitrite. The combined maximum hazard index for nitrate and nitrite is 6.682 while the combined mean hazard index is 0.872. The maximum values provide a worst-case estimate of exposure to reclaimed water based on past operations of the Laguna Plant. However, plant treatment processes (i.e., nitrification and denitrification) would be upgraded before the project is built and are expected to reduce the concentrations of ammonia, nitrate and nitrite in the plant's effluent.

Currently, the Laguna Plant includes nitrification as a waste stream treatment process throughout the discharge season (1 October through 14 May) and for most of the remainder of the year. Nitrification removes most of the ammonia from the wastewater through a two-step conversion process. The ammonia is converted to nitrite which in turn is converted to nitrate. Based on historical data for periods when the Laguna Plant was nitrifying the waste stream, the maximum concentration of ammonia was lowered to 14.9 mg/L which yields a hazard quotient of 0.852; the mean concentration was lowered to 2.0 mg/L, which yields a hazard quotient of 0.127. These values indicate that there would be no significant human health hazard associated with exposure to reclaimed water containing these concentrations of ammonia.

Following nitrification the wastewater would be denitrified to reduce the nitrate level through conversion of the nitrate to nitrogen gas. Denitrification would be added with the current upgrade of the Laguna Plant. Design criteria for the addition of denitrification are intended to reduce the combined nitrate and nitrite level, but a specific target level has not been included in the design criteria. Dilution of reclaimed water with surface waters and groundwaters upon release and uptake of these chemicals by terrestrial and aquatic plants would be expected to further reduce the human health hazard from nitrate, nitrite, and ammonia, and these chemicals would not present a significant human health hazard via

most of the expected exposure pathways. However, in rural areas near some potential reservoir sites where groundwater already contains elevated nitrate, some wells may yield water which contains nitrate levels that exceed the State and Federal drinking water standards if dilution alone is counted on to reduce nitrate levels.

Hazard Posed by Lead

As noted in Section 2.5, the USEPA and DTSC do not publish RfDs or slope factors for lead. Instead, blood lead levels are predicted based on environmental data. These predicted blood lead levels are compared to the DTSC's and USEPA's threshold level for lead poisoning (10 micrograms of lead per deciliter of blood, µg/dl). Blood lead levels were modeled using the DTSC's LEADSPREAD model (the output is contained in Appendix D). The input required for the model includes background lead concentrations in air and soil, background dust concentrations, and the concentration of lead in drinking water.

Background lead concentrations in air and background dust concentrations were obtained from data collected by the California Air Resources Board from 1990 through 1993 in Sonoma County (CARB 1990-93). The background lead concentration in air (0.12 $\mu g/m^3$) was obtained from data collected at the Fifth Street monitoring station in Santa Rosa. The background dust concentration (72 $\mu g/m^3$) was obtained from data collected at the Matheson Street Station monitoring station in Healdsburg. These two values represent the highest 30-day averages for the 4-year period. The background lead concentration in soil (67 µg/g) was obtained from data collected by the United States Geologic Survey (USGS) and represents the 99th percentile (i.e., 99 percent of the background values in the western United States would be expected to be below this value) calculated on the geometric mean for the western United States (ATSDR 1992a). The concentration in drinking water (12 µg/L) was set to the maximum concentration in the Laguna Plant effluent in Table 2.1-1. All other input to the model was provided by the DTSC.

The model calculates the blood lead levels for exposed adults in residential and industrial settings and exposed children (including children with a tendency to eat soil, a behavior referred to as a pica) in a residential setting. The results are expressed as blood lead levels by percentile. The results for the 99th percentile indicate that the most sensitive receptor, a child exhibiting pica behavior, would be expected to have a blood lead level of 9.5 µg/dl, a value below the DTSC action level of 10 µg/dl. This result indicates that there is no significant hazard associated with exposure to reclaimed water containing this concentration of lead.

Characterization of Carcinogenic Effects

A preliminary screening of the data for carcinogenic effects was performed using maximum reported values (Tables 2.6-1 and 2.6-3). The use of these values generates maximum excess cancer risks and presents a worst-case scenario. Two chemicals,

Table 2.6-3

Summary of the Total Excess Cancer Risk[1]

Chemical	Maximum Excess Cancer Risk	Mean Excess Cancer Risk	EPA Cancer Class	Target Organ(s)[2]
arsenic	**8.9×10^{-5}**	**5.4×10^{-5}**	A	skin
chromium[3]	**8.8×10^{-5}**	**1.5×10^{-5}**	A	lung (by inhalation)
chloromethane	**1.4×10^{-6}**	1.3×10^{-7}	C	kidney
1,4-dichlorobenzene	**1.1×10^{-6}**	8.2×10^{-7}	C	liver
methylene chloride	**1.6×10^{-6}**	2.2×10^{-7}	B2	liver
tetrachloroethylene	6.9×10^{-7}	2.6×10^{-7}	N/A	liver
bromodichloromethane	**4.3×10^{-5}**	**8.4×10^{-6}**	B2	kidney
chloroform	**3.3×10^{-5}**	**7.4×10^{-6}**	B2	kidney
dibromochloromethane	**3.0×10^{-6}**	5.8×10^{-7}	C	liver
aldrin	**7.6×10^{-6}**	**2.2×10^{-6}**	B2	liver
α-lindane	**9.0×10^{-6}**	9.2×10^{-7}	B2	liver
γ-lindane	**1.5×10^{-6}**	4.0×10^{-7}	B2	liver
heptachlor	**1.7×10^{-6}**	4.7×10^{-7}	B2	liver
bis (2-ethylhexyl) phthalate	8.1×10^{-7}	3.2×10^{-7}	B2	liver

[1] Values in **bold text** indicate an excess cancer risk that exceeds the screening value of 1.0×10^{-6}.
[2] Target organ in test species identified by USEPA as basis for cancer slope factor.
[3] Cancer risk calculated assuming all chromium is present as chromium (VI).

tetrachloroethylene and bis (2-ethylhexyl) phthalate, have maximum excess cancer risks less than 1×10^{-6}. The remaining potential carcinogens have maximum excess cancer risks greater than 1×10^{-6} and of these, four chemicals, arsenic, chromium, bromodichloromethane, and chloroform, have maximum excess cancer risks greater than 1×10^{-5}. These four chemicals account for about 90 percent of the total excess cancer risk. Table 2.6-3 also lists the excess cancer risks associated with the mean chemical concentrations, the weight-of-evidence classification, and target organs of the potential carcinogens.

While maximum excess cancer risks provide a worst-case scenario, mean excess cancer risks provide more representative estimates of the potential risk associated with exposure to undiluted reclaimed water as it is discharged from the Laguna Plant. Mean risks are more representative for all chemicals and especially for chemicals that have been detected only infrequently or that have been detected at an elevated concentration in only one analysis. (Some studies discard these values as "outliers." However, all detections were retained for this risk assessment.) For infrequently detected chemicals, the use of a maximum reported value greatly overestimates the potential risk associated with long-term exposure to reclaimed water.

The risk equations assume that an individual will be exposed to a chemical for 30 years (6 years during childhood and 24 years as an adult) at an unchanging concentration, an assumption which is useful for screening purposes but which does not realistically describe long-term exposure. For example, the assumptions do not account for operations practices at the Laguna Plant such as the release of water to the Russian River only from 1 October through 14 May. This practice would limit the duration of an individual's exposure and would result in varying dilutions of reclaimed water with surface water. Factors such as volatilization and chemical and biological degradation during storage would also reduce the potential risk for many organic chemicals, especially volatile organics. Finally, historical concentrations may not be a good predictor of future concentrations because the use of some chemicals such as organochlorine pesticides has been restricted or banned and because proposed upgrades of plant operations (e.g., chlorination/dechlorination and nitrification/denitrification) would affect future chemical concentrations. These factors and their impact on the excess cancer risk estimates for the potential carcinogens are discussed below.

Tetrachloroethylene and bis (2-ethylhexyl) phthalate were detected infrequently (2 of 19 and 5 of 19 samples, respectively) and at concentrations very close to their respective reporting limits (Table 2.6-4). Tetrachloroethylene was never detected at concentrations above the State or Federal MCLs. Bis (2-ethylhexyl) phthalate was detected in one sample at a concentration in excess of the State MCL but equal to the Federal MCL. The maximum and mean excess cancer risks for both chemicals were less than 1×10^{-6} (Tables 2.6-3). These results indicate that tetrachloroethylene and bis (2-ethylhexyl) phthalate would not present a significant excess cancer risk. In addition, the DTSC and USEPA identify bis (2-ethylhexyl) phthalate as a potential laboratory contaminant that

Table 2.6-4

Comparison of Mean and Maximum Concentrations
to State and Federal Water Quality Standards

Chemical	Concentration[1]		State MCL (mg/L)	Federal MCL (mg/L)	Reporting Limit(s) (mg/L)
	Mean (mg/L)	Maximum (mg/L)			
Inorganics					
arsenic	0.002	0.004	0.050	0.050[2]	0.001 - 0.005
asbestos, MFL[3]	0.25	0.56	7	7	0.05 - 0.28
chromium	0.002	0.014	0.050	0.100	0.001 - 0.02
Volatile Organics					
chloromethane	0.0005	0.005	N/A	N/A	0.0001 - 0.001
1,4-dichlorobenzene	0.0006	0.0009	0.005	0.075	0.0005
methylene chloride	0.0008	**0.006**	0.005	0.005	0.0001 - 0.003
tetrachloroethylene	0.0002	0.0006	0.005	0.005	0.0001 - 0.0005
Trihalomethanes					
bromodichloromethane	0.0022	0.011	N/A	0.100[4]	0.0005
chloroform	0.0099	0.044	N/A	0.100[4]	0.0005
dibromochloromethane	0.0004	0.0021	N/A	0.100[4]	0.0001 - 0.0005
Pesticides					
aldrin	0.00001	0.00003	N/A	N/A	0.00001 - 0.00005
α-lindane	0.00001	0.00003	N/A	N/A	0.00001 - 0.00005
γ-lindane	0.00002	0.00009	0.0002	0.0002	0.00001 - 0.00002
heptachlor	0.00001	**0.00003**	0.00001	0.0004	0.00001 - 0.00005
Phthalates					
bis (2-ethylhexyl) phthalate	0.0028	**0.006**	0.004	0.006	0.0006 - 0.020

Table 2.6-4 (continued)

Comparison of Mean and Maximum Concentrations
to State and Federal Water Quality Standards

Chemical	Concentration[1]		State MCL (mg/L)	Federal MCL (mg/L)	Reporting Limit(s) (mg/L)
	Mean (mg/L)	Maximum (mg/L)			
Radioactivity					
Gross alpha, GPV[5]	2.8 pCi/L	5.5 pCi/L	15 pCi/L	N/A	N/A
Gross beta, GPV	12.3 pCi/L	12.7 pCi/L	50 pCi/L	N/A	N/A

N/A Not available

(1) Maximum concentrations in **bold text** indicate a concentration greater than a State or Federal MCL.

(2) The Federal MCL for arsenic is currently under review.

(3) Asbestos values are reported as millions of fibers per liter (MFL).

(4) MCL is for total trihalomethanes. The USEPA has proposed to lower the MCL to 0.08 mg/L.

(5) Radioactivity values are reported as greatest probable value (GPV) expressed as picoCuries per liter (pCi/L).

may be introduced accidentally into samples during their analysis (DTSC 1994a). Some of the reported detections may have occurred due to laboratory-introduced contamination.

Chloromethane, aldrin, α-lindane, and heptachlor were detected in only a small number (one to three) of samples (Table 2.1-1); most samples were reported as below reporting limits. All but one non-detect for chloromethane had a reporting limit of 0.0005 mg/L or less and most non-detects for heptachlor (13 of 18), aldrin (12 of 16), and α-lindane (13 of 17) had reporting limits of 0.00001 mg/L or less. These reporting limits, which are also equal to the calculated mean concentrations for these chemicals (Table 2.1-1), yield excess cancer risks of 1.3×10^{-7} (chloromethane), 4.7×10^{-7} (heptachlor), 2.2×10^{-6} (aldrin), and 9.2×10^{-7} (α-lindane). Heptachlor was detected in one sample at a concentration in excess of the State MCL but less than the Federal MCL. There are no State or Federal MCLs for chloromethane, aldrin, and α-lindane. The mean excess cancer risk exceeds 1×10^{-6} only for aldrin. The mean concentrations (which incorporate one-half of the reporting limit as an estimated concentration for non-detects) of heptachlor, α-lindane and aldrin would overestimate risk because the use of these pesticides has been restricted since at least the 1980s and they would not be expected to occur at concentrations significantly above background in either the effluent or influent at the Laguna Plant. These results indicate that chloromethane, aldrin, α-lindane and heptachlor would not present a significant excess cancer risk.

Methylene chloride was detected in five samples. Eleven of thirteen non-detects had reporting limits of 0.0005 mg/L or less. The reporting limit of 0.0005 mg/L yields an excess cancer risk of 1.3×10^{-7}. The mean concentration of methylene chloride is well below the State and Federal MCL (only one sample, the maximum concentration detected, exceeded the State and Federal MCLs). These results indicate methylene chloride would not present a significant excess cancer risk. In addition, the DTSC and USEPA identify methylene chloride as a potential laboratory contaminant that may be introduced accidentally into samples during their analysis (DTSC 1994a). Some of the reported detections may have occurred due to laboratory-introduced contamination.

1,4-dichlorobenzene was detected in 10 of 13 samples. Both the maximum and mean excess cancer risks are about 1.0×10^{-6}. Although detected frequently in the Laguna Plant effluent (a possible source of 1,4-dichlorobenzene is its use in toilet bowl cleaners, as a deodorizer), 1,4-dichlorobenzene concentrations have been below both the State and Federal drinking water MCL. These factors indicate that 1,4-dichlorobenzene would not present a significant excess cancer risk.

The maximum and mean excess cancer risks for arsenic and chromium exceed 1×10^{-6}. However, both chemicals are present at concentrations well below their respective State and Federal MCLs. The Federal MCL for chromium is equal to the Maximum Contaminant Limit Goal (MCLG) for chromium set by the USEPA. An MCLG is defined by the USEPA as a non-enforceable concentration of a drinking water contaminant that is protective of human health and that allows an adequate margin of safety. The MCLG (and thus MCL) of 0.1 mg/L for total chromium (Cr III and Cr VI) is

based on the USEPA's reference dose methodology for Cr VI, the more toxic chromium species. The methodology assumes that humans will receive 20 percent of their daily chromium uptake from drinking water. An uncertainty (safety) factor of 500 was applied to derive this value from experimental data. The MCLG and MCL for chromium also fall into the safe and adequate daily dietary intake range of 50 to 200 mg/day for Cr III established by the National Research Council in the National Academy of Sciences (USEPA 1995b). Based on this information chromium would not present a significant excess cancer risk.

The MCL for arsenic is based on a value derived by the United States Public Health Service. Both the State and Federal MCLs for arsenic are equal to the USEPA's MCLG. Even though arsenic is potentially carcinogenic in humans by inhalation and ingestion, its potential essential nutrient value was considered in determining the MCLG. The basis for this evaluation is nutritional requirements identified by the National Academy of Sciences (USEPA 1995b). In addition, arsenic concentrations similar to that in the Laguna Plant's effluent have been reported for the City of Santa Rosa's domestic water supply, which it receives from the SCWA. The 1994 Annual Water Quality Report for the City of Santa Rosa Utilities Department reports that the water sample station at Meadowridge Drive had an arsenic concentration of 0.0064 mg/L on 30 September 1994 (City of Santa Rosa 1994). This value is greater than the maximum value (0.004 mg/L) reported in the Laguna Plant effluent, but well below the MCL. Based on this information arsenic would not present a significant excess cancer risk.

Cancer Risk Posed by Trihalomethanes (Bromodichloromethane, Dibromochloromethane and Chloroform)

Bromodichloromethane, chloroform, dibromochloromethane, and bromoform, known collectively as trihalomethanes, are disinfection by-products (DBPs) formed when water containing naturally-occurring organic matter is chlorinated. The maximum excess cancer risk exceeds 1×10^{-6} for the three trihalomethanes that were detected (bromoform was not detected above reporting limits). In addition, the mean excess cancer risk exceeds 1×10^{-6} for bromodichloromethane and chloroform. However, the current Federal MCL for total trihalomethanes is 0.1 mg/L (the USEPA has proposed to lower the MCL to 0.08 mg/L), well above the maximum total concentration of 0.057 mg/L (although bromoform was not detected in any sample, one half the reporting limit was used to calculate the maximum concentration of trihalomethanes).

The Laguna Plant has indicated that it is evaluating a plan to replace chlorination with ultraviolet light disinfection. It is expected that the mean concentration of DBPs would decrease if the new process is initiated. However, concentrations will depend on a number of factors, such as the need to add a chlorine residual to prevent bacterial growth in piping and the concentration of chlorination by-product precursors in the untreated water.

Cancer Risk Posed by Asbestos

As noted in Section 2.5, a chronic oral RfD and an oral slope factor for asbestos are not available on IRIS or HEAST and have not been derived by the DTSC. The mean asbestos concentration (0.25 MFL) in reclaimed water is below the California and Federal drinking water standard of 7 MFL. It is also well below the mean background concentration (1,438 MFL) measured in the Russian River. For these reasons asbestos concentrations would not present a significant excess cancer risk.

Cancer Risk Posed by Radioactivity

No slope factors have been derived by the DTSC or the USEPA for radioactivity expressed as pCi/L. However, water quality standards for the State of California have been promulgated for gross alpha (15 pCi/L) and gross beta (50 pCi/L) activity. Both the maximum and the mean gross alpha activity and gross beta activity in the Laguna Plant effluent are less than these standards. The background gross alpha and beta activities in the Russian River were 1.1 pCi/L and 1.8 pCi/L, respectively. Although the effluent activities are greater than the background activities, they are below the regulatory standards and would not present a significant excess cancer risk.

Fish Consumption

Many pollutants concentrate in fish tissues by accumulating in fatty tissues or selectively binding to fish muscle tissue (the fillet). Even extremely low concentrations of bioaccumulative pollutants in water or bottom sediments may result in fish tissue concentrations high enough to pose health risks to fish consumers. Some contaminants, particularly pesticides, tend to accumulate in the fatty tissues of fish. Consequently, fish species with a higher fat content, such as carp, bluefish, some species of salmon, and catfish, may pose greater risks from some contaminants than leaner fish such as bass, sunfish, and yellow perch. Although exposure to some contaminants may be reduced by removing the fat, skin, and viscera before eating, other contaminants, such as methylmercury, accumulate in the fillet, and therefore cannot be removed by trimming. In addition, some fish are consumed whole, or used whole in the preparation of fish stock for soups and other foods. Under these conditions the entire burden of bioaccumulative contaminants contained in the fish would be ingested.

In addition to the risks borne by the general population due to consumption of contaminated fish, populations eating higher-than-average quantities of fish are at greater risk of having higher body burdens of contaminants. Those at greatest risk include sport and subsistence fishers. Within these populations, pregnant women and children may be at greater risk of incurring adverse effects than other members of the populations, due to their proportionally higher consumption rates and/or increased susceptibility to adverse health effects.

An assessment of the potential for exposure to chemicals via fish consumption (and other aquatic organisms) is made by comparing the Laguna Plant effluent water quality data to

water quality criteria for the ingestion of aquatic organisms and water; by evaluating data from bioaccumulation/magnification studies performed in 1991 and 1994 at the Kelly Farm Demonstration Wetland; and by applying the USEPA's methodology for fish advisories to data collected for the Toxic Substances Monitoring Program (TSMP).

Water Quality Criteria for Ingestion of Aquatic Organisms and Water

Water quality criteria based on human health effects have been proposed by the USEPA for the combined consumption of aquatic organisms and water and for the consumption of aquatic organisms alone (Table 2.6-5). These criteria have been proposed to protect humans who eat fish and other aquatic species harvested from a contaminated body of water. In calculating these criteria, the USEPA estimated a chemical's bioconcentration factor (BCF) from experimental and field studies to account for contaminant uptake from ambient water by aquatic organisms. However, BCFs vary among species and may also vary according to age, size, fat content, and developmental stage of an organism. It is unlikely that the single estimated BCF could account for all of the various conditions and environments where contaminants may occur. Because many factors influence BCFs, it has been suggested that it is not possible to determine the water concentration of a contaminant that would protect humans who consume a variety of aquatic species living under varying conditions (ATSDR 1992a).

The water quality criteria assume that an individual eats 6.5 grams of fish and shellfish per day. This figure represents a national average, and it may over- or underestimate individual fish consumption, depending upon local conditions. Given the conditions described in Section 2.4, it is likely to overestimate the amount of fish consumed from any of the waterways within the project area.

Given the substantial uncertainties in BCFs and fish consumption rates, the ATSDR does not endorse using water quality criteria based on human consumption of aquatic organisms. Instead, it is recommended that actual concentrations in edible portions of aquatic species should be used where possible and that specific dietary habits of the potentially affected population be considered. The criteria are, however, a useful starting point for screening chemicals of concern. Any chemicals which pass this screen (i.e., that have water concentrations less than these criteria) would not be expected to have a significant human health impact.

Kelly Farm Demonstration Wetland Bioaccumulation/magnification Studies

The Kelly Farm Demonstration Wetland (Wetland) was constructed in 1990 and is owned by the Santa Rosa Subregional System. The water supply for the Wetland is the reclaimed water from the Santa Rosa Subregional Water Reclamation System on Llano Road. The first bioaccumulation/magnification studies were performed at the site during 1991. Freshwater clams, which had been deployed in the Wetland for about 3 months, sediments, plants, invertebrates and fish were collected and analyzed for trace elements and organochlorine compounds. Further investigation of bioaccumulation/magnification

Table 2.6-5

Water Quality Criteria Based on Human Health Effects
(Ingestion of Aquatic Organisms and Water)

	Concentration[1]		Criteria[2]	
Chemical	Mean (mg/L)	Maximum (mg/L)	Water & Organism Consumption (mg/L)	Organism Consumption (mg/L)
Inorganics				
arsenic	**0.002**	**0.004**	0.000018	0.00014
asbestos, MFL[3]	0.25	0.56	7	N/A
cadmium	0.001	0.007	w	w
chromium	0.002	0.014	w	w
copper	0.01	0.04	1.3	N/A
cyanide	0.01	0.03	0.7	220
lead	0.005	0.012	w	w
mercury	0.0001	**0.0002**	0.00014	0.00015
nickel	0.004	0.010	0.61	4.6
silver	0.001	0.01	N/A	N/A
zinc	0.03	0.28	N/A	N/A
Volatile Organics				
chlorobenzene	0.0001	0.0001	0.68	21
1,4-dichlorobenzene	0.0006	0.0009	0.4	2.6
ethylbenzene	0.0002	0.001	3.1	29
methylene chloride	0.0008	0.006	0.0047	1.6
tetrachloroethylene	0.0002	0.0006	0.0008	0.0085
toluene	0.0002	0.0004	6.8	200
1,1,1-trichloroethane	0.0002	0.0002	w	w
Trihalomethanes				
bromodichloromethane	0.0022	0.011	0.00056	0.046
chloroform	0.0099	0.044	0.0057	0.47
dibromochloromethane	0.0004	0.0021	0.00041	0.034

Table 2.6-5 (continued)

Water Quality Criteria Based on Human Health Effects
(Ingestion of Aquatic Organisms and Water)

	Concentration[1]		Criteria[2]	
Chemical	Mean (mg/L)	Maximum (mg/L)	Water & Organism Consumption (mg/L)	Organism Consumption (mg/L)
Phthalates				
di-n-butyl phthalate	0.0012	0.0019	2.7	12
bis (2-ethylhexyl) phthalate	<u>0.0028</u>	**0.006**	0.0018	0.0059
diethyl phthalate	0.0009	0.007	23	120
Pesticides				
aldrin	**0.00001**	**0.00003**	0.00000013	0.00000014
endosulfan II	0.00001	0.00001	0.110	0.240
α-lindane	**0.00001**	**0.00003**	0.0000039	0.000013
γ-lindane	<u>0.00002</u>	**0.00009**	0.000019	0.000063
heptachlor	**0.00001**	**0.00003**	0.00000021	0.00000021

N/A Not available.

(1) A concentration in **bold text** exceeds both the Organism Consumption criterion and the Water & Organism Consumption criterion. An <u>underlined concentration</u> exceeds the Water & Organism Consumption criterion, but is less than the Organism Consumption criterion.

(2) w - Criteria for cadmium, chromium, lead, and 1,1,1-trichloroethane were withdrawn in the National Toxics Rule.

(3) Asbestos values are reported as millions of fibers per liter (MFL).

in the food chain was conducted during August 1994. Cattail (*Typha latifolia*) rhizomes, crayfish (*Procambarus clarki*), mosquitofish (*Gambusia affinis*), bulrush (*Scirpus californicus*) seeds, and sediments were collected and analyzed for trace elements, and organochlorine and organophosphate compounds. The results of these studies are discussed in a technical report prepared for the Subregional Wastewater Project and are only summarized here (Merritt Smith Consulting, et al. 1995 and references therein).

Mosquitofish and sediment samples were analyzed for chlordane, DDT (and its metabolites, DDE and DDD), dieldrin, lindane and PCBs in both 1991 and 1994. Additional organochlorine compounds and organophosphate pesticides were analyzed in 1994. None of these chemicals were detected in any tissue or sediment sample collected in the Wetland during 1991 or 1994.

Eleven trace elements (metals and metalloids) were analyzed in animal and plant tissues, and sediments in 1991 and 1994. Aluminum, chromium, lead, nickel, and zinc had lower concentrations in 1994 than in 1991, both in sediments and in wetland vegetation. Concentration reductions ranged from 18 to 54 percent for sediments, and from 2 to 68 percent for plant tissues.

Arsenic and mercury had increased concentrations in sediments in 1994 relative to 1991. However, the average concentrations of these metals in Santa Rosa's reclaimed water were the same or slightly lower in 1994 than in 1991, indicating that the sediment increases were not due to increases in these metals in reclaimed water discharge. Changes in plant tissue concentrations for these two metals could not be documented because their concentrations were below analytical detection limits. Silver and copper had higher concentrations in plant tissues in 1994 relative to 1991.

A comparison of crayfish data showed that, similarly to sediments and vegetation, aluminum, nickel and zinc concentrations were lower in 1994 than in 1991 (32 to 69 percent reduction), while silver and copper were higher (89 to 107 percent). Crayfish data differed from vegetation data in that mercury decreased and lead increased in concentration from 1991 to 1994. Temporal changes in the concentration of arsenic, cadmium, chromium and selenium in crayfish tissues could not be documented because tissue concentrations reported in 1991 were below analytical detection limits.

Temporal changes in metals concentrations for mosquitofish largely mirrored those observed for crayfish tissues for mercury and nickel (lower in 1994 than in 1991), and for copper, lead, and silver (higher in 1994). The aluminum concentration in mosquitofish, unlike crayfish data, increased in 1994 relative to the previously collected data.

Based on these results, there is no evidence of bioaccumulation of metals in plant tissues collected from the Kelly Farm Demonstration Wetland (Merritt Smith Consulting, et al. 1995). Similarly, aquatic organisms showed no evidence of bioaccumulation for aluminum, arsenic, chromium, lead, and nickel. Average concentrations of these five

metals in crayfish and mosquitofish samples were less than 30 percent of the concentrations detected in sediments.

The concentration of copper in mosquitofish tissues was less than in sediment, but a bioaccumulation factor of 3.5 was calculated for crayfish (a bioaccumulation factor less than or equal to 1.0 indicates a low potential for chemical accumulation in an organism's tissues). Accumulation of copper in crayfish tissues is expected because copper is a major component of the respiratory pigment hemocyanine of crustaceans and other invertebrates.

Bioaccumulation factors for zinc in crayfish and mercury in crayfish and mosquitofish ranged from 0.56 to 1.24, indicating that the concentrations of these metals in animal tissues and in sediments are similar. The concentration in mosquitofish, however, was twice as high as that measured in sediments suggesting a possible accumulation, or concentration from water, in fish tissues.

Of the organisms studied, only crayfish would be considered a potential food source for humans although the other organisms may be potential components of the food web for species that are consumed by humans. With the exception of zinc in mosquitofish, there is no evidence that bioaccumulation of trace elements or organic chemicals is occurring in the species examined. This level of bioaccumulation of zinc, which is an essential nutrient for humans and other organisms, would not present a human health hazard via the food web.

Toxic Substances Monitoring Program

Monitoring programs for chemicals in fish generally include analysis of metals and bioaccumulative organics such as PCBs, and organochlorine pesticides and herbicides. The USEPA has recommended a list of target analytes for screening programs based on the chemical's usage and persistence in the environment (USEPA 1993). These target analytes include three metals (cadmium, mercury, and selenium), organochlorine pesticides, organophosphate pesticides, chlorophenoxy herbicides, PCBs, dioxins and dibenzofurans. The California EPA includes many of these chemicals as part of the TSMP, which is conducted by the State Water Resources Control Board (SWRCB) to detect and evaluate the occurrence of toxic substances in fresh, estuarine, and marine waters of the State through the analysis of fish and other aquatic life (SWRCB 1993). A presentation and discussion of TSMP data collected from within the project area has been prepared (Merritt Smith Consulting, et al. 1995).

The TSMP data were collected from several locations (both upstream and downstream from the Laguna Plant discharge) as well as for a variety of fish species and fish tissues (in some cases, the entire fish was analyzed and in others only fillets). Measurement endpoints (screening values) for this risk assessment are based on maximum chemical concentrations detected in fillets from fish caught downstream of the Laguna Plant discharge point. Several characteristics of the data set affect its usefulness for predicting risk. For example, TSMP data were collected at varying distances from the discharge

point. At increasing distances from the discharge point there is an increasing probability that water from other sources (e.g., discharges from other treatment facilities or surface water runoff) would contribute to (or reduce by dilution) any influence from discharges from the Laguna Plant. Data collected from Mark West Creek and the Russian River, just downstream from its confluence with Mark West Creek, were used for this risk assessment.

The USEPA, Office of Water currently recommends screening values that are based on methodology used to develop current water quality criteria and that were used in the National Study of Chemical Residues in Fish. The USEPA acknowledges that other methodologies exist, however this methodology is recommended because:

- It gives full priority to the protection of public health;
- It provides a direct link between fish consumption rate and risk levels (i.e., between dose and response);
- It generally leads to conservative (health protective) estimates of risk;
- It is designed to protect consumers of locally caught fish and shellfish, including susceptible subpopulations such as sport and subsistence fishermen who are at potentially greater risk than the general adult population because they tend to consume greater quantities of fish and because they frequently fish the same sites repeatedly.

As with the estimation of hazard and risk values for drinking water, the hazard and risk values for fish consumption are calculated differently for carcinogenic and non-carcinogenic effects. The following equations are used to calculate the screening values (SVs) for noncarcinogens and carcinogens.

Noncarcinogens:

$$\mathbf{SV_n = (RfD * BW)/CR}$$

where

SV_n = screening value for a noncarcinogen (mg/kg; ppm)
RfD = oral reference dose (mg/kg/day)
BW = body weight (kg), 70 kg
CR = mean daily consumption rate of the species of interest by the population of concern (kg/day), 0.0065 kg/day

Carcinogens:

$$\mathbf{SV_c = [(RL / SF) * BW] /CR}$$

where

SV_c = Screening value for a carcinogen (mg/kg; ppm)
RL = maximum acceptable risk level (dimensionless), 1×10^{-6}
SF = oral slope fact $(mg/kg/day)^{-1}$
BW = body weight (kg), 70 kg
CR = mean daily consumption rate of the species of interest by the population of concern (kg/day), 0.0065 kg/day

Default values for body weight and fish consumption rates are those recommended by the USEPA for a screening evaluation and are averages for adults in the United States general population. The maximum acceptable risk level for cancer is set at 1×10^{-6} (one excess case of cancer per 1,000,000 individuals exposed over a 70-year lifetime), a more conservative (health-protective) level than that (1×10^{-5}) recommended by the USEPA.

No concentrations exceed screening values (Table 2.6-6). Maximum values for mercury and DDT are nearest their respective screening values. For mercury, the fish (*Lepomis cyanellis,* green sunfish) associated with the maximum value was collected in 1990 on the Russian River at the Odd Fellows Park Bridge, downstream from the confluence of the Russian River and Mark West Creek. Other green sunfish collected at this location between 1978 and 1989 contained from 0.23 ppm to 0.41 ppm mercury. Two fish (both of the species *Lepomis macrochirus,* bluegill) collected from Mark West Creek contained lower concentrations of mercury (0.22 and 0.40 ppm). Mercury was detected at the reporting limit in one sample of the Laguna Plant effluent (Table 2.1-1). The hazard index associated with this detection is 0.043 (Table 2.6-1), well below the hazard index screening value of 1.0. Based on these values mercury would not present a significant adverse health hazard.

DDT and its metabolites were detected in only one fish tissue (fillet) sample (collected in 1978) on the Russian River at the Odd Fellows Park Bridge. DDT was not detected above reporting limits in the Laguna Plant effluent. Based on the reporting limit (0.001 mg/L) and the RfD and slope factor in Table 2.6-6 the hazard index and risk would be 0.128 and 5.1×10^{-6}. As with other organochlorine pesticides that have been banned, these values would be expected to overestimate the current discharge to the environment. Therefore, DDT would not be expected to present a significant adverse health hazard.

2.7 UNCERTAINTY

All risk assessment involves the use of assumptions, judgments, and imperfect data to varying degrees. This results in uncertainty in the final estimates of risk. Uncertainties are present in virtually each step of the risk assessment process including the selection of appropriate data, the identification of exposure pathways, the selection of toxicity values,

Table 2.6-6

Screening Values for Fish Consumption

Chemical	RfD (mg/kg/day)	Slope Factor $(mg/kg/day)^{-1}$	Screening Values (ppm)		Maximum Concentration (ppm)
			Non-carcinogens	Carcinogens (RL = 10^{-6})	
cadmium	0.0005		5		0.02
mercury	0.00006[(1)]		0.6		0.54
selenium	0.005		50		0.34
total DDT[(2)]	0.0005	0.34	5	0.031	0.026
total lindane[(3)]	0.0003	1.1	3	0.0098	0.0027

(1) For the purpose of calculating an SV, mercury is assumed to be present as methylmercury. The RfD for methylmercury was lowered by a factor of 5 from the value available on the USEPA IRIS database (3×10^{-4} mg/kg/day). This more conservative value (health protective) value was used because of concerns that the fetus, and possibly pregnant women, are at increased risk of adverse neurological effects from exposure to methylmercury.

(2) Total DDT includes DDD, DDE, and DDT.

(3) Total lindane includes α-, β-, δ-, and γ-lindane.

and the characterization of risk. Some of these uncertainties are inherent in the equations and values that are contained in guidance documents and information sources recommended by regulatory agencies. Toxicity values, for example, are often derived from animal data, which must be extrapolated to a human RfD or slope factor. The equations used to characterize risk are based on characteristics of the general population (e.g., food and water intakes, average weights and lifespans). These characteristics may not be appropriate for some subpopulations. Other sources of uncertainty are related to site-specific information. For example, there may be uncertainties associated with the completeness of an exposure pathway or the concentration of a chemical at the potential exposure point. This site-specific uncertainty, as it relates to the Subregional Wastewater Project is discussed below.

Data Quality

Quality control samples (e.g., field blanks, trip blanks and method blanks) were not available for most samples. The lack of such controls precludes an accurate quantitative evaluation of contaminants potentially introduced during sample collection, transport or analysis. For example, acetone, methylene chloride, toluene, and phthalate esters, four chemicals considered by the USEPA and DTSC to be potential laboratory contaminants (USEPA 1989d, DTSC 1994a), were detected in several samples (Table 2.1-1). Acetone, methylene chloride and toluene are used in analytical laboratories for chemical extractions and phthalates are commonly found in soft plastics (e.g., squeeze bottles, plastic wraps) used in laboratories. Some of these chemicals may have been introduced during the sample collection and analytical processes. The introduction of these chemicals during sampling and analysis would result in an overestimation of risk.

Chemicals for which all analyses were below the reporting limit were not included in the quantitative risk assessment. For chemicals which were detected at least once, non-detects were included in the sample mean by assigning a value of one-half the reporting limit. The actual concentration of a chemical that was not detected above the reporting limit may vary between zero and the reporting limit. The omission of these chemicals would result in an underestimation of risk if the chemical(s) were present at a concentration greater than zero. To account for some of this risk, reporting limits for this risk assessment were set lower than the drinking water standard for all chemicals for which an MCL has been promulgated (Merritt Smith Consulting 1995a, 1995b). Thus, for these chemicals a non-detect would indicate that the reported value was below the MCL. The use of one-half of the sample reporting limit to calculate the mean may either under- or over-estimate the risk.

Noncarcinogenic hazards and carcinogenic risks were calculated using DTSC guidance (DTSC 1994a), which states that maximum reported values should be used. This assumption overestimates risk.

Chromium was reported as total chromium. The toxicity values used to assess its effects were those for chromium VI, its more toxic form. In most environmental media,

chromium III is the predominant form. The use of toxicity values for chromium VI is likely to overestimate the potential health risks posed by chromium.

Exposure Pathway Assessment

Reclaimed water that is discharged to the Russian River or that moves from reservoirs to groundwater will be diluted by groundwater and/or surface water before it reaches a potential exposure point. In addition, the chemicals in reclaimed water will be subject to volatilization (from surface waters), degradation (biological and chemical) and adsorption to soils and sediments. Therefore, the health hazards and risks predicted by the quantitative assessment, which assumes that water reaching a domestic water intake would consist of 100% reclaimed water, would overestimate the potential health risks posed by exposure to the reclaimed water through one of the discharge or reuse scenarios of the Subregional Wastewater Project.

For domestic wells near the Russian River, the highest percentage of river water has been predicted to be 35 percent (CH2M Hill et al. 1993). This percentage was predicted to occur during dry weather conditions for a well 50 feet from the river with an average pumping rate of 10 gallons per minute (gpm). For municipal wells, the wells may pump river water almost exclusively under some conditions (e.g., a well located within 100 feet of the river with a pumping rate of 2,000 gpm). The shortest travel time for groundwater between the river and a municipal well 100 feet away was predicted to be 2 days.

Volatilization is likely to reduce the mean concentration of volatile organic chemicals in water released to reservoirs, storage ponds, the Laguna de Santa Rosa and the Russian River. The use of effluent data to estimate the concentration of volatile organic compounds in reclaimed water is likely to overestimate potential health risks from volatile organic chemicals.

Biological and chemical degradation may reduce the mean concentration of organic chemicals in water released to reservoirs, ponds or the Russian River. The use of effluent data to estimate the concentration of trihalomethanes in reclaimed water stored in ponds or reservoirs, or released to the Russian River, is therefore likely to overestimate potential health risks.

Toxicity Assessment

Some uncertainty is inherent in the toxicity values used to assess the noncarcinogenic hazards and carcinogenic risks. These uncertainties, which may lead to either under- or over-estimation of risk, are compounded by the assumption of dose additivity for multiple substance exposures (for example in the case of nitrates and nitrites). The assumption of additivity ignores possible synergisms or antagonisms among chemicals, and assumes similar mechanisms of action and metabolism. If synergistic effects occur the assumption of additivity would underestimate risk, while antagonistic effects would result in an overestimate of risk. Because a significant margin of safety is built into the

toxicity values derived by the USEPA and the DTSC (Section 2.5) the toxicity values generally would be expected to overestimate risk.

Current toxicity values that have been derived by the USEPA and DTSC are based on chronic effects and endpoints such as cancer (carcinogens), birth defects (teratogens), and genetic effects (genotoxicity or mutagens) or adverse effects on organ systems such as kidney and liver damage or neurotoxicity. It has recently been suggested that some chemicals may cause developmental or other reproductive damage by mimicking and/or disrupting the endocrine systems of humans (Parsons Engineering Science 1995a and references therein). These chemicals have been labeled "environmental estrogens," "environmental hormones," and/or "hormone mimics." RfDs based specifically on these types of developmental and reproductive effects have not yet been developed by the DTSC and USEPA for most analytes. Because some of these effects may occur at concentrations lower than those considered to establish current RfDs, the toxicity values may underestimate risk for some chemicals.

Toxicity values (slope factors) for carcinogens are developed using a linearized model and an upper limit of the 95^{th}-percentile confidence interval. These simplified models do not allow use of information on the mechanism of action or cellular repair and are likely to overestimate risk. In addition, no threshold level (i.e., all concentrations greater than zero are expected to contribute to some incremental increase in cancer risk) is assumed and all carcinogens are assumed to be genotoxic (tumor-inducing). However, some chemicals may produce tumors only after continuous high exposure and may not be carcinogenic at relevant environmental concentrations. There is also evidence that cancer formation is a multistage process involving tumor induction (the genotoxic effect) and subsequent tumor promotion (tumor growth). Some chemicals may be complete carcinogens, that is capable of both inducing and promoting tumor formation, whereas other chemicals may either induce or promote tumor growth, but not both. The model assumptions may therefore, overestimate risk especially for chemicals which are not complete carcinogens. Finally, carcinogenic risks were summed for chemicals having various weight-of-evidence classifications as well as different target organs. This summation may tend to overestimate risk.

3 RISK FROM BIOLOGICAL COMPONENTS

This section presents the human health risk from biological components in reclaimed water from the Subregional Wastewater Project. It addresses the potential human health risks resulting from discharge of final treated effluent from the treatment plant to surface water and the subsequent use of surface water as a source of potable water as well as other purposes (e.g., irrigation).

The risk assessment follows the standard framework used for chemical risk assessment, which consists of:

- Biological components of reclaimed water
- Hazard identification
- Dose-response assessment
- Exposure Assessment
- Risk Characterization

3.1 BIOLOGICAL CONTAMINATION

Final effluent was sampled for microorganisms over a 3-month period from October through December 1994 and analyzed according the Field Sampling and Quality Assurance Plan for this study (Merritt Smith Consulting 1995a). Four samples were taken of fresh final effluent over this period and one sample was taken from Delta Pond of stored final effluent. Delta Pond is a large open impoundment adjacent to Santa Rosa Creek. These data are summarized in Tables 3.1-1 and 3.1-2. Table 3.1-1 presents the data for concentrations of pathogenic bacteria in the treated effluent. Table 3.1-2 presents the data for protozoa and viral agents. Four samples were also taken from the Russian River at Kaiser Beach, 5 miles upstream of the point of entry of the Laguna de Santa Rosa to the River. These data are included in the tables for comparison purposes.

The data in Table 3.1-1 include specific test results for *Salmonella*, *Shigella*, and *Legionella*, as well as results of tests for indicator organisms, including total coliform and heterotrophic bacteria. The term "indicator organism," as used in water microbiology, means a microorganism whose presence is evidence that pollution (associated with fecal contamination from man or other warm-blooded animals) has occurred. Indicator organisms may be accompanied by pathogens, but do not necessarily cause disease themselves (NRC 1977). Indicators have the following general characteristics: they are absent from unpolluted waters, are present in greater numbers than pathogenic organisms, have greater survival time than pathogens, and their detection is more reliable and less time-consuming.

Table 3.1-1

Analytical Data for Bacterial Parameters

le Type	Date	Total Coliform (MPN/100 mL)	Legionella (MPN/100 mL)	Salmonella (MPN/100 mL)	Shigella (MPN/100 mL)	Heterotrophic Bacteria (CFU/1 mL)
sh Effluent	27 Oct 94	<2	<7840	<2.2	<2.2	8
sh Effluent	8 Nov 94	<2	<7840	<2.2	<2.2	21
sh Effluent	30 Nov 94	2	<7840	<2.2	<2.2	20
sh Effluent	14 Dec 94	<2	<7840	<2.2	<2.2	18
red Effluent	30 Nov 94	280	<7840	<2.2	<2.2	3100
R. Water	27 Oct 94	23	<7840	<2.2	<2.2	166
R. Water	8 Nov 94	240	<7840	<2.2	<2.2	31
R. Water	30 Nov 94	30	<7840	<2.2	<2.2	110
R. Water	14 Dec 94	220	<7840	<2.2	<2.2	610

Table 3.1-2

Analytical Data for Protozoa and Viral Parameters

ole Type	Date	Giardia lamblia (Cysts/100 L)	Cryptosporidium (Oocysts/100 L)	Enteric Virus (PFU/Volume)
esh Effluent	27 Oct 94	0	0	NA
esh Effluent	8 Nov 94	0	0	NA
esh Effluent	30 Nov 94	5.1	0	NA
esh Effluent	14 Dec 94	13.8	0	< 1 / 129 L
red Effluent	30 Nov 94	NA	NA	0 / 22 L
n R. Water	27 Oct 94	0	0	< 1 / 45 L
n R. Water	8 Nov 94	0	2.7	NA
n R. Water	30 Nov 94	0	0	0 / 153 L
n R. Water	14 Dec 94	0	0.4	NA

The concentrations of *Salmonella*, *Shigella*, and *Legionella* in the final fresh effluent are all below their respective detection limits on all sample dates. Total coliform is below the detection limit for all final fresh effluent samples except one, which was at the detection limit of 2 most probable number (MPN) per 100 milliliters (mL). This concentration is below the facility's NPDES permit requirements for a daily maximum of 23 MPN/100 mL (Small 1995). Total coliform was detected at 280 MPN/100 mL in the one sample taken from Delta Pond of final stored effluent. Delta Pond is an open surface water impoundment. Sheep graze on land adjacent to the pond and ducks and other waterfowl utilize the pond as a stop-over along the Pacific flyway. It is not unexpected that total coliform counts would increase in stored effluent because of the contribution of feces from animals and wildlife. Total coliform was detected in all four samples taken from the Russian River, ranging from 23 MPN to 240 MPN/100 mL.

Heterotrophic bacteria plate counts in final fresh effluent ranged from 8 to 20 colony forming units per mL (CFU/mL). The one sample of final stored effluent detected heterotrophic bacteria at 3100 CFU/mL. Total coliform and heterotrophic plate count measurements are useful in assessing the effectiveness of the treatment system for drinking water purification. Heterotrophic bacteria plate counts in Russian River samples ranged from 31 to 610 CFU/mL.

The data in Table 3.1-2 include data for two protozoa, *Cryptosporidium* and *Giardia lamblia*. No *Cryptosporidium* oocysts were detected in the final fresh effluent samples. *Giardia lamblia* cysts were detected in two of the four final fresh effluent samples. The maximum *Giardia* concentration was 13.8 cysts/100 L. The analysis, which is performed by filtering at least 100 liters of the water and counting the cysts, does not distinguish between viable and inactivated cysts. The Delta Pond sample of stored final effluent was not analyzed for these two protozoa. *Cryptosporidium* oocysts were detected in two Russian River samples at concentrations of 0.4 and 2.7 oocysts/100 L. No *Giardia* cysts were detected in the four Russian River samples.

The two analyses for enteric viruses in the final effluent (See Table 3.1-2), one fresh and one stored, were below the detection limits. The lack of detectable enteric virus in the final effluent is expected since a system using chlorine to achieve 99.9 percent inactivation of *Giardia* cysts is expected to achieve greater than 99.99 percent inactivation of viruses (USEPA 1989a).

To summarize the quantitative analytical data for microbiological agents in the fresh final effluent, the only specific microorganism detected in the effluent above its detection limit is *Giardia lamblia* of unknown viability. Total coliform and heterotrophic bacteria were also detected in fresh and stored final effluent samples.

3.2 HAZARD IDENTIFICATION

The continuing outbreaks of waterborne pathogenic diseases in the United States clearly demonstrates a hazard from microbial contamination of drinking water. Water may

contain a wide variety of pathogenic microorganisms. A 1984 comparison of the microorganisms responsible for causing waterborne disease in the U.S. found bacteria caused the greatest number of outbreaks and cases of illness, followed by protozoans then viruses. They also found that the etiological agent had been identified in only half of the outbreaks reported (Lippy and Waltrip 1984).

The principal types of microorganisms considered in this risk assessment are those detected in fresh and stored final effluent samples, however, all microorganisms analyzed in samples taken for this study, even those not detected, are discussed.

Total Coliforms

The coliform group of bacteria is made up of a number of genera including *Klebsiella*, *Escherichia*, *Serratia*, *Erwinia* and *Enterobacteria*. Total coliform bacteria are all gram negative asporogenous rods and are associated with feces of warm-blooded animals. Although coliforms are usually considered nonpathogenic, enterotoxigenic and enteropathogenic variants of *Escherichia* are responsible for outbreaks of enteritis and gastroenteritis. Studies in different parts of the world have indicated that E. coli is a significant cause of bacterial diarrhea, and food and waterborne outbreaks of E. coli-caused illness have been documented (USEPA 1986).

Heterotrophic Bacteria

Heterotrophic bacteria are also considered an indicator of the general bacterial count in drinking water. The heterotrophic plate count (HPC) enumerates aerobic and facultative aerobic bacteria found in water that are capable of growth on simple organic compounds (primarily carbohydrates, amino acids and peptides) found in the culture medium, and under incubation time and temperature conditions specified. The HPC is useful for monitoring the efficiency of water treatment processes, including disinfection. Among other uses, it can be used to assess changes in finished water quality during distribution and storage, and distribution cleanliness (Reasoner 1990).

Although it is known that high concentrations of HPC bacteria can develop in favorable locations in a water distribution system, data on human health effects resulting from exposure to these organisms following ingestion or through inhalation of aerosols is lacking. It is also not known whether HPC bacteria present a risk to immunologically compromised people who are at risk to infection by organisms that are generally thought to be nonpathogenic (Reasoner 1990).

Giardia lamblia

Giardia lamblia is a flagellated parasitic protozoan. It causes illness by mechanically damaging the microvilous lining of the upper small intestine. The form of *Giardia* that causes the damage is the trophozoite. Trophozoites, shaped somewhat like horseshoe crabs, live and reproduce in the upper part of the small intestine. As they are excreted,

they form cysts, a dormant stage which is shed with the feces. Stomach acids activate ingested cysts, releasing new trophozoites into the small intestine. There, the trophozoites act like leaches, attaching to the intestinal epithelium by means of a suction cup-like structure, the ventral disc. As the trophozoites grow in the small intestine, they cause epithelial cells lining the small intestine to slough off. Although the body responds by producing new epithelial cells, these cells are not mature enough to produce digestive enzymes. This prevents digestion of food and leads to diarrhea.

Symptoms of giardiasis include, in addition to diarrhea, flatulence and vomiting. The illness usually continues for about a week, followed by recovery. Patients under stress or with immune deficiency may continue to be ill for months or even years. Severe giardiasis can cause dehydration and weight loss from malabsorption of nutrients. In children, malabsorption of fats and soluble vitamins may slow growth. Because many people can harbor *Giardia* without symptoms, asymptomatic carriers may spread the illness to others (Health & Environment Network 1988).

Non-Detected Microorganisms

Microorganisms that were analyzed for but not detected are discussed below by organism type. This discussion is intended to provided a brief explanation of the pathogenic microorganisms analyzed for in this study.

Bacteria

The principal bacterial agents that have been shown to cause human intestinal disease associated with drinking water are: *Salmonella typhi*, typhoid fever; *Salmonella paratyphi-A*, paratyphoid fever; *Salmonella* (other species and a great number of serotypes), salmonellosis, enteric fever; *Shigella dysenteriae*, *S. flexneri*, and *S. sonei*, bacillary dysentery; and *Legionella*, pneumonia. Several other organisms have been associated with diseases spread by drinking water, such as *Campylobacter jejuni*, enteritis; *Vibrio cholerae*, cholera; *Leptospira sp.*, leptospirosis; *Yersinia enterocolitica*, gastroenteritis; *Francisella tuarensis*, tularemia; and *Pseudomonas aeruginosa*, various infections; those in other genera of the Enterobacteriaceae: *Edwardssiella, Proteus, Serratia*, and *Bacillus*, gastroenteritis (NRC 1977, USEPA 1986).

Members of the genus *Salmonella* are the most widely recognized enteric pathogens. Often associated with food and waterborne outbreaks of illness, they are responsible annually for 1 to 2 million incidents of disease in the US population (USEPA 1986). *Salmonella* is responsible for over 50 percent of the waterborne outbreaks and illness over a 40-year period ending in 1986 (Lappenbusch 1986).

Shigella bacteria are responsible for approximately 3 percent of the reported diarrhea cases in the United States and is second to *Salmonella* in responsibility for waterborne illness (Lappenbusch 1986). The incidence of shigellosis in a community is clearly related to sanitation and water quality. Four pathogenic species of *Shigella* are

recognized, but little data are available on their presence in domestic waste and survival in the environment (USEPA 1986).

Legionella pneumophilia was first isolated in 1977 following an outbreak of pneumonia which occurred among 221 people attending the annual convention of the Pennsylvania American Legion. Following the discovery of this organism, other Legionella-like organisms were discovered. Microorganisms belonging to this newly classified family of organisms called Legionellaceae are gram-negative, aerobic bacilli.

The natural reservoir for *Legionella pneumophilia* appears to be aquatic habitats, including rivers and lakes. Outbreaks of legionnaires' disease have been linked to aerosol dissemination from water cooling towers. It is presumed that aerosols containing *Legionella pneumophilia* are taken into a building by the ventilation-dehumidification system.

The major manifestation of legionnaires' disease is pneumonia. Patients typically have fever and cough, usually feel lethargic, and can become disoriented. Unlike patients with other pneumonias, patients with legionnaires' disease often have severe gastrointestinal symptoms, including diarrhea, nausea, and vomiting. The disease rarely occurs in people with good health, but tends to afflict patients with preexisting illnesses, especially chronic lung disease. Cigarette smoking seems to be the most important risk factor, although heavy alcohol intake has also been noted as a factor (Muraca et al. 1988).

Protozoan Parasites

Cryptosporidium is classified as a coccidian parasitic protozoan. It is an obligate, intracellular parasite, whose life cycle involves both asexual and sexual multiplication. Infection results as a result of ingestion of an environmentally resistant stage referred to as an oocyst. Its primary route of transmission from host to host is fecal-oral. When the oocyst, an elliptical sphere 3 to 5 microns in diameter, is ingested by a compatible new host, the oocyst wall breaks down, releasing the four sporozoites contained inside. The sporozoites invade the intestinal epithelial cells of the host, where they undergo stages of asexual and sexual multiplication, finally producing new oocysts, of which 20 percent develop a single wall membrane in the host, while the other 80 percent develop environmentally-resistant, two layer walls and are excreted into the environment, where they are immediately infective (AWWA 1988, Sothern 1994).

Symptoms of cryptosporidiosis (the disease caused by *Cryptosporidium*) in humans appear within 2 to 12 days of exposure and generally include profuse watery diarrhea lasting for up to several weeks. Nausea, abdominal cramps and low grade fever may accompany the diarrhea. No known drug therapies are effective in treating the disease, but it is self-limiting in immuno-competent individuals. Individuals suffering from a viral illness, particularly measles or chicken pox, may be especially vulnerable to infection, as are malnourished children. The disease can become chronic in immuno-suppressed individuals, for example, in those who have AIDS or are HIV-positive (Sothern 1994).

Viruses

Viruses differ fundamentally from other microorganisms that may occur in water. They are transmitted as submicroscopic, inert particles that are unable to replicate or adapt to environmental conditions outside a living host. These particles, or virions, have the potential to produce infections, and sometimes disease, in people who ingest them with drinking water. A viral particle eventually loses its infectivity with the passage of time and with exposure to environmental conditions (NRC 1977). The maximum survival time of viruses in soil has been noted as six months (USEPA 1989c).

The viruses important to human health that are most likely to be transmitted by drinking water are the enteric viruses. These are primarily parasites of a portion of the intestinal tract. The stomach and duodenum are seldom affected by viruses, partly because of unfavorable conditions (NRC 1977).

The most commonly studied enteric viruses in sewage are the enteroviruses, which include polioviruses, coxsackie A and B viruses, echoviruses, hepatitis A virus and other more recently classified enterovirus types (USEPA 1986). Probably the most important viral disease sometimes transmitted by water is hepatitis A. Its most common route of transmission is fecal-oral, primarily by person-to-person contact. In addition to water and personal contact, the disease is sometimes transmitted by food (NRC 1977).

3.3 DOSE-RESPONSE ASSESSMENT

The dose-response assessment is the process of characterizing the relation between the dose of an agent administered or received and the incidence of an adverse health effect in exposed populations and estimating the incidence of the effect as a function of human exposure to the agent. In this risk assessment, the dose-response assessment identifies the probability of infection as a result of exposure to pathogenic microbial agents and, once infected, the conditional probability that an individual will contract a disease.

Development of disease depends on numerous factors, including the immune status of the host, age of the host, type, strain, and virulence of the microorganism, and route of infection. Uncertainties associated with the dose-response information available in the literature include:

- Experimentation with healthy individuals as opposed to individuals with poorer health status (aged, compromised immune system) and therefore greater susceptibility, and

- Experimentation with well-characterized strains of pathogens as opposed to indigenous pathogens (Glicker and Edwards 1991).

In order to assess the hazards from biological agents in drinking water, it is necessary to know how many viable pathogenic cells are necessary to initiate an infection. Dose-response experiments for microorganisms of concern in drinking water have been

conducted with human volunteers using bacteria, protozoans, and viruses. In these experiments, volunteers are typically exposed to dosages of microorganisms that were known to contain certain average concentrations. Then the resulting number of infected and unaffected individuals was determined. The number of infected individuals depends on the probability of the organism's occurrence in the water and the dose-response curve (Regli et al. 1991). The results of some of these dose-response studies are summarized below.

Total Coliforms

The number of bacteria required to produce disease is unknown but can range from 1 to 10^8 per 100 mL or more viable organisms. Total coliform are an indicator of microbial contamination and are usually not pathogenic in and of themselves. The Maximum Contaminant Level for total coliform under the National Primary Drinking Water Regulations are based on the presence/absence of total coliforms in samples, rather than on an estimated coliform density. Community water treatment systems are required to obtain routine total coliform samples at intervals during each month with the number of monthly samples based on the population served. When less than 40 samples per month are required, no more than one sample per month may be positive for total coliform. When 40 or more samples per month are required, no more than 5 percent of all monthly samples may be positive for total coliform (USEPA 1989b). Additionally, under these regulations, filtration of source water is not required if the total coliform concentration in water prior to disinfection is equal to or less than 100/100 mL in at least 90 percent of the samples (USEPA 1989b). The numerical criterion is set based on the premise that the number of coliforms in domestic wastewater far outnumber the number of pathogenic microorganisms since coliforms are contributed by the entire population while pathogens are contributed only by persons with enteric illnesses. The die-off rate of pathogenic bacteria is greater than the death rate of coliforms outside the intestinal tract, thus, exposure to treatment and residence in water reduces the number of pathogens relative to coliforms. Therefore, the MCL for coliforms is considered statistically safe for human consumption because of the improbability of ingesting pathogenic bacteria.

Heterotrophic Bacteria

Annual risks of disease and death from bacteria in drinking water appear to be substantially less as compared to risks from other microorganisms, specifically, enteric viruses. The number of ingested bacteria required to cause illness appears to be in the range from 1 to 10^8. Without the identification of the individual bacterial species, determination of the effective dose is not possible.

Giardia lamblia

When the USEPA promulgated the Surface Water Treatment Rule (SWTR), it suggested that water be treated for *Giardia* cyst removal with the goal of ensuring high probability that the population consuming the water would not be subjected to a risk of greater than

one infection of giardiasis per 10,000 people per year (1×10^{-4}). This is comparable to other acceptable microbiological risk levels (USEPA 1989a).

Based on a risk analysis by Rose et al. (1991), which assumed all cysts found were viable and infectious to humans, the incidence of infection from *Giardia* was predicted as a function of exposure to cyst concentrations in drinking water. Table 3.3-1 indicates the annual risk of *Giardia* infection for people consuming water with different concentrations of *Giardia* cysts. From the analysis performed by Rose et al., a concentration of 0.007 cysts per 100 L results in a risk of infection of 1×10^{-4}.

Table 3.3-1

Estimated Yearly Risk of *Giardia* Infections [a]

Yearly Risk [b]	Geometric mean cyst concentration per 100 L
31.6 infections per 100,000 persons ($1 \times 10^{-3.5}$)	0.002 (2.0×10^{-3})
10 infections per 100,000 persons($1 \times 10^{-4.0}$)	0.0007 (7.0×10^{-4})
3.2 infections per 100,000 persons($1 \times 10^{-4.5}$)	0.0002 (2.0×10^{-4})
1 infection per 100,000 persons(1×10^{-5})	0.00007 (7.0×10^{-5})

a Using the exponential risk assessment model of Rose et al. (1991).

b. Risk is a probability and is usually expressed in exponential form (1×10^{-4}). Expressing it as the number of infections per population exposed is often easier to understand.

Dose-Response Summary

Many of the pathogens present in wastewater are continuing causes of food and waterborne disease in the United States. Although the information on infectious dose for most pathogens is limited, it appears that low numbers (less than 50 organisms) protozoan cysts are capable of causing infection in a susceptible host. The infective dose of *Giardia lamblia* by the oral route appears to be as low as between 1 and 10 cysts. Minimum infectious doses for bacteria are generally higher than those for parasites. Virulence of the particular type and strain of microorganism and host factors may play a role in determining the actual number of microorganisms required to cause infection. The number of individuals who develop clinical illness will also depend upon the strain and type of organism as well as host factors such as age and immune status.

If the distribution of pathogens in the water consumed by a human population is known and dose-response relationships are established, the risk of infection, morbidity, and mortality can be estimated. However, dose-response data are not available for most waterborne pathogens.

3.4 EXPOSURE ASSESSMENT

This section presents a characterization of the population potentially impacted by the use of treated effluent, an evaluation of exposure pathways, following those identified in Figure 2.3-1, and estimates of potential exposure to microorganisms from the treated effluent.

Population of Potentially Exposed Individuals

Characteristics of populations potentially exposed are discussed in Section 2.3 of this assessment.

Sensitive Populations

Certain population groups are at greater risk of disease from waterborne microorganisms. The infecting dose of microorganisms and disease development varies with the age and general health of the host population. Infants and the aged may be particularly susceptible. Previous exposure to a specific pathogen is important, in that antibodies present in the intestinal tract, associated with immunity to enteric infection, may prevent infection with a strain that is generally present in the population, whereas a new strain introduced into the water supply may present an increased hazard (NRC 1977).

Immunosuppressed individuals are at increased risk of disease from waterborne microorganisms. These populations include the elderly, those who have AIDS, are HIV positive, are organ transplant recipients, are undergoing chemotherapy, or have leukemia. Diseases such as hepatitis may become chronic in these individuals. Among AIDS patients, for example, cryptosporidiosis is regarded as a leading cause of the chronic diarrhea and nutrient malabsorption associated with the wasting syndrome that frequently leads to death (Sothern 1994).

Exposure Pathways

Ingestion is the primary exposure pathway for contact with microorganisms in water. Although direct ingestion of final fresh or stored effluent does not occur, effluent would be discharged at the outfall on the Russian River or to the Laguna de Santa Rosa, which enters the Russian River at its confluence with Mark West Creek. The SCWA caissons are 2 (caissons 1 and 2) to 4 miles (caissons 3, 4, and 5) downstream from the Russian River outfall. Caissons 3 and 4 are downstream of the confluence with Mark West Creek, and caisson 5 is upstream of the confluence.

The National Primary Drinking Water Regulations (Title 40, Code of Federal Regulations §141) require that surface water and "groundwater under the direct influence of surface water" be treated to minimize the risk of disease from pathogenic organisms that may occur in these waters. Groundwater under the direct influence of surface water is defined as groundwater with (1) a significant occurrence of insects or other macroorganisms, algae, or large-diameter pathogens such as *Giardia lamblia*, or (2) significant and

relatively rapid shifts in water characteristics such as turbidity, temperature, conductivity, or pH which closely correlate to climatological or surface water conditions. Percolation of water through soil and/or base materials (e.g., sands and gravels) normally filters out macro- and micro-organisms and dampens the magnitude of changes in temperature, turbidity, conductivity and pH. The National Primary Drinking Water Regulations and the Surface Water Treatment Rule require additional treatment (e.g., chlorination or ozonation) of surface water and groundwater under the direct influence of surface water to minimize the risk of exposure to pathogens.

Primarily because of changes in turbidity that correlate with turbidity fluctuations in the Russian River, caisson 5 has been classified as under the direct influence of surface water (Flugum 1995). A 1993 study of this caisson showed that turbidity changes in water withdrawn from caisson 5 correlated with turbidity changes in the Russian River, although the changes in the caisson were at least two orders of magnitude (1×10^{-2}) smaller than the changes observed in the Russian River (CH2M Hill 1993). The report also notes that, in spite of the change in turbidity, the natural filtration system operates well in removing bacteria (only 11 of 60 samples from caisson 5 contained total coliform at 1 to 9 total coliform count) and that no *Giardia* were found in collector water. In contrast, all Russian River samples (62 of 62) were positive for total coliform (16 to 16,000 total coliform count) and two samples of Russian River water were positive for *Giardia* during this time.

Currently, caisson 5 has its own operational controls and is monitored for turbidity. As turbidity increases, the pump is shut down. The SCWA is attempting to modify the caisson so that it is no longer considered under the direct influence of surface water. None of the other four caissons are classified as under the direct influence of surface water. It is therefore unlikely that the biological components of reclaimed water would reach the SCWA's intakes.

A recent analysis of the effects of the proposed discharge to the Russian River has reaffirmed that the natural filtration provided by the river bed will effectively remove coliform bacteria, *Giardia*, *Cryptosporidium*, and viruses (CH2M Hill 1996). The analysis also concludes that implementation of a Russian River discharge upstream of the SCWA collectors would not necessitate additional treatment by the SCWA to comply with the requirements of the Surface Water Treatment Rule, although further analysis of collectors and additional sampling for *Giardia* and *Cryptosporidium* in the effluent and river may be required by the California Department of Health Services.

To provide a simple assessment of the potential risk from ingestion of final treated effluent, this evaluation considers ingestion of final effluent without dilution as the most conservative exposure pathway that could be quantified.

Additional exposure pathways exist from use of effluent as a source of domestic water. During showering, aerosolized microorganisms may be inhaled. Use of water containing microorganisms for irrigating home gardens could result in surface contamination of

fruits and vegetables with microorganisms. Eating prior to washing could result in the ingestion of microorganisms along with the skin of the produce. Watering gardens could also generate aerosols which could then be inhaled.

Many enteric microorganisms can be transmitted effectively by aerosols. In fact, the infectious dose by the aerosol route may be lower than the infectious dose for the ingestion route for some organisms. The organisms in aerosols can be transmitted by inhalation or the settling of the organisms onto surfaces with which humans come into contact (USEPA 1986). This exposure route may be important in an urban setting if water is used to irrigate public gardens and lawns.

Recreationists may also be exposed to microorganisms through direct contact with surface water while fishing, swimming or other water sports because of inadvertent ingestion of surface water. Ingestion of inadequately cooked fish caught from water containing pathogens is an indirect exposure pathway for recreationists.

Indirect exposure to pathogenic microbial agents may also occur through person-to-person contact and other means. For example, giardiasis is a fecal-oral disease and can be spread by person-to-person contact. Numerous outbreaks of giardiasis have occurred in daycare centers, and *Giardia* can also be transmitted through sexual contact. Viral and bacterial diseases can also be transmitted indirectly by person-to-person contact.

The agricultural use of treated effluent involves some of the same exposure pathways as domestic use of effluent. As with domestic use of effluent for irrigating home gardens, crop irrigation could result in surface contamination of agricultural products, leading to other indirect routes of exposure. Potential routes of exposure include:

- Inadvertent contact with effluent water during irrigation;
- Handling soil and raw produce from irrigated areas;
- Inhaling microorganisms that become airborne (via aerosols, dust, etc.) during and or after irrigation;
- Contact with dust raised by strong winds or by plowing or cultivating the soil;
- Consumption of pathogen-contaminated crops irrigated with effluent.

The potential for exposure through these pathways diminishes over time as environmental conditions such as heat, sunlight, desiccation, and other microorganisms destroy pathogens that may be present in areas irrigated with wastewater. Table 3.4-1 summarizes the survival rates of bacteria, viruses and protozoan cysts in soil and on plants. Because protozoan cysts are rapidly killed by environmental factors, the public health threat from protozoa in wastewater-irrigated land is minimal. Bacteria and viruses are of greater concern. Some bacteria are unique among pathogens in their ability to regrow. Even very small populations of certain bacteria can rapidly proliferate under the

Table 3.4-1

Survival Time of Pathogens in Soil and on Plant Surfaces

	Soil		Plants	
Pathogen	**Absolute Maximum**	**Common Maximum**	**Absolute Maximum**	**Common Maximum**
Bacteria	1 year	2 months	6 months	1 month
Protozoan cysts(1)	6 months	3 months	2 months	1 month
Viruses	10 days	2 days	5 days	2 days

Source: USEPA, 1989c

(1) Little if any data are available on the survival time of *Giardia lamblia* cysts and *Cryptosporidium* oocysts.

right conditions. Viruses and protozoa cannot regrow outside their specific host organism(s). Once reduced by treatment, their populations stay reduced (USEPA 1989c).

Concentrations of Pathogens in Water

In this risk assessment, it is assumed that the final treated effluent is used directly as a drinking water source. Exposure point concentrations of the microorganisms detected are assumed to be equal to the maximum detected concentration in final effluent. However, because there is no mechanism to determine the viability of *Giardia* cysts in a sample, an assumption of viability must be made considering wastewater treatment effectiveness. Under the conditions used at the Santa Rosa wastewater treatment facility, the wastewater is disinfected with chlorine at a sufficiently high concentration and adequate time to inactivate greater than 99.99 percent (4-log) of *Giardia* cysts. See Appendix F for this calculation and a discussion of the requirements for inactivation of *Giardia lamblia* cysts by chlorine in water. The National Primary Drinking Water Regulations for *Giardia lamblia* (40 CFR 141.72) require disinfection treatment to ensure 99.9 percent (3-log) inactivation of *Giardia lamblia* for public water systems that do not provide filtration (USEPA 1991). Using an inactivation rate of 99.99 percent (4-log), the maximum concentration (13.8 cysts/100 L) detected in any sample of effluent, and the assumption that all cysts are viable, the *Giardia* cyst concentration would be no more than 1.4×10^{-3} cysts/100 L. If the assumption is made that only 10 percent of the cysts detected were viable, as stated by Regli et al. (1991), and a 4-log inactivation rate was used, the concentrations of *Giardia* drops to 1.4×10^{-4} cysts/100 L.

3.5 RISK CHARACTERIZATION

This section integrates the information from the hazard identification, dose-response assessment, and exposure assessment to characterize risk.

Total coliform levels have been used for decades as the primary measure of the microbial quality of drinking water. Coliforms are usually present in water contaminated with human and animal feces and are often associated with outbreaks of disease. Although total coliforms are usually not pathogenic themselves, their presence in drinking water indicates that pathogens may also be present. It is generally accepted that treatment which provides total coliform-free water will reduce pathogens to minimum levels (USEPA 1989b). The total coliform data for the final effluent indicates the absence of coliform in three of the four samples taken over a period of three months. The sample in which total coliform was detected (2 MPN/100 mL) was below the daily maximum (23 MPN/100 mL) required by the facility's NPDES permit (Small 1995) and below the concentration that requires filtration (100 MPN/100 mL). By contrast, samples of Russian River water upstream from the Laguna de Santa Rosa discharge plant were all positive for total coliform (range 23 MPN to 240 MPN/100 L).

The sewage treatment facility is required to meet wastewater discharge standards as part of their NPDES permit. This requires them to take daily samples that must be below a concentration of 23 MPN/100 mL. Data available from January 1991 through June 1994 indicates they have generally met this requirement for total coliform counts (Small 1995). During this period there were five exceedances of this concentration (170, 130, 50, 49 and 49) that would be considered above the margin of error for the coliform test (+/- 50%). All exceedances were below the concentration detected in the Russian River upstream of the Laguna de Santa Rosa discharge.

The estimated maximum concentration of viable *Giardia lamblia* cysts in the final effluent, based on four analytical tests and a 99.99 percent disinfection rate, is 1.4 x 10^{-3}/100 L. The concentration of *Giardia lamblia* cysts which corresponds to an "acceptable" annual risk of infection of 1 x 10^{-4}, according to the risk analysis of Rose et al. (1991), is 7.0 x 10^{-4} cysts/ 100 L (see Table 3.3-1). Therefore, the estimated maximum *Giardia lamblia* concentration in the final effluent is twice the acceptable average concentration as defined by Rose, et al. Assuming that the cyst concentration in the effluent is the maximum concentration, the risk to an individual or a population using the final effluent as their sole drinking water source, without dilution, is twice the acceptable risk. A more reasonable scenario is that only 10 percent of the *Giardia lamblia* cysts in the final effluent after disinfection are viable, dropping the concentration in the undiluted effluent to 1.4 x 10^{-4} cysts/100 L, which is below the concentration that results in an annual risk of infection of 1 x 10^{-4}. Under such a scenario, the *Giardia lamblia* cyst concentration in the effluent does not pose an infection risk above what is considered acceptable by USEPA for drinking water. Dilution of effluent with surface and groundwater and filtration by soils and underlying base materials would further reduce this risk.

Although no *Giardia lamblia* cysts were detected in the Russian River samples during this study, a previous study has reported cysts in the Russian River (CH2M Hill 1993). Most samples collected for the CH2M Hill study were negative for *Giardia* but two samples collected in November 1992 and December 1993 each contained one cyst per 100 gallons of water. This is equivalent to 2.6×10^{-1} cysts/100 mL, assuming all cysts were viable, considerably higher than the viable concentrations calculated for the treatment plant effluent.

The three specific bacteria, *Legionella*, *Salmonella*, and *Shigella* were below the respective detection limits in all final effluent samples. These results coupled with the low total coliform concentrations indicate that the risk of disease from ingestion of enteric bacteria in final effluent is low.

No *Cryptosporidium* oocysts were detected in the final effluent, indicating the risk of disease from ingesting effluent as a source of drinking water is low. *Cryptosporidium* was detected on two testing dates in surface water samples taken from the Russian River at Kaiser Beach at a maximum concentration of 2.7 oocysts per 100 L. The concentration of *Cryptosporidium* which causes disease is not known (USEPA 1994d). However, epidemiological data on an outbreak of cryptosporidiosis in Carrollton, Georgia in 1987 provided an indication that the ingestion of a very small number of viable oocysts, possibly as low as one, may be sufficient to cause disease symptoms in healthy individuals. In the Carrollton outbreak, measurements taken during the second week detected concentrations averaging 0.63 oocysts/L within the distribution system (post-filtration) (Sothern 1994). The concentrations detected in surface water from the Russian River, 0.027 oocysts/L, are an order of magnitude below this value.

The limited test data for enteric viruses in the final effluent from the treatment process indicate that the final effluent does not contain enteric viruses above the detection limit. Furthermore, the wastewater treatment process, including chlorine disinfection, is expected to attain greater than 99.99 percent disinfection of enteric viruses. Therefore, exposure to the final effluent is not likely to pose a risk of enteric viral disease.

Risk Summary

The analysis of microbiological data collected for this study leads to the conclusion that it is unlikely that the treated effluent discharged from the treatment system poses a significant human health risk. However, considering the uncertainty in estimating minimum infectious doses for some pathogens, it is not possible to accurately assess the risk from direct ingestion of treated effluent, let alone the risk through pathways other than ingestion. It is known that waterborne disease occurs as a result of inadequately treated drinking water supplies, and that outbreaks of waterborne diseases have been attributed to bacterial, viral, and parasitic microorganisms. The potential disease risk from exposure to the treated effluent depends on the concentration of the organism in the exposure medium, the virulence of the organism, the health of the receptor, the extent and duration of exposure and the exposure route. The exposure pathway that presents the

greatest risk of disease is from use of the treated effluent for potable water without further treatment or dilution. Ingestion of the water would constitute the largest potential dose of microorganisms present in the treated effluent. However, other exposure routes, including inhalation of aerosols and contact with irrigated garden plants and crops would also pose a potential disease risk if pathogenic organisms are present in the treated effluent. Other factors which enter into the determination of risk include survival times of the microorganisms in water and soil, further treatment of the water before its use, and dilution rates.

3.6 UNCERTAINTY

Uncertainties are associated with the collection and analysis of microbial data. Sampling and analytical procedures may or may not have accurately characterized the organisms present and their concentrations. In addition, sample size affects the accuracy of both the identification and quantification of microorganisms. For example, the specific bacterial species, *Shigella* and *Salmonella*, were not detected in samples taken. These species are responsible, nation-wide, for over 50 percent of waterborne outbreaks. They can be highly virulent and there may be significant risk of disease even when present in low numbers. The risk of death from drinking 2 liters of water per day with one *Shigella dysenteriae* in 10 L of water (0.01 organism in 1 liter of water) could be as high as 1.3×10^{-6} (USEPA 1986). The limited number of samples adds uncertainty to the absence of these organisms in the effluent, although the low total coliform count tends to corroborate the lack of these pathogenic bacteria in effluent.

Viruses often require large sample sizes for positive identification (NRC 1977). Although none were identified in samples taken for this study, viruses have very low levels of infectivity. Haas and his colleagues have studied the dose-response relationships for viruses present in sources of drinking water which are believed to be responsible for gastroenteritis and other diseases. The beta-Poisson model has been identified as the one which fits most data and which provides a conservative method for low dose extrapolation (Regli et al. 1991). Table 3.6-1 lists the concentrations of several viruses, computed from dose-response curves and based on a daily water consumption of 2 liters, which correspond to an annual risk of infection of 1×10^{-4}. Note that there is about a four order of magnitude difference among the various viruses. If rotavirus is regarded as being conservatively representative of the indigenous pathogens in water, then the acceptable risk of 1×10^{-4} limits the mean virus density to 2.2×10^{-7} (Regli et al. 1991).

The risk of enteric viral disease is judged to be low based on the test data and treatment technology. However, monitoring data are not organism-specific and the test results only show that the virus concentration in the final effluent is below the detection limit. The actual concentration could be anywhere in the range from zero to the detection limit. Therefore, it is not possible to compare the virus concentration in the final effluent with the risk estimates for infection in Table 3.6-1.

Table 3.6-1

Acceptable Concentrations [1] of Viruses [2]

Organism	Water concentration (number/2L)
Rotavirus	2.2×10^{-7}
Polio III	2.7×10^{-7}
Polio I	1.5×10^{-5}
Polio I	1.9×10^{-3}

(1) An acceptable concentration leads to an annual risk of infection of 1×10^{-4}
(2) From dose-response curves of Regli et al., 1991.

A major uncertainty in the available dose-response data is that human experiments were performed using healthy adult volunteers. The general population has a lower overall health status and a greater susceptibility to adverse effects from infection. The development of clinical illness depends on many factors, including the immune status of the host, age of the host, virulence of the organism, strain of the organism, and route of infection. Because of this, the dose required for infection does not correlate with illness for all receptors. In addition, dose-response data available have been obtained using well-characterized laboratory strains of pathogens. The intrinsic infectivity may differ between laboratory maintained cultures and indigenous viruses; however, the magnitude of these differences is currently not clear.

One major area of uncertainty in the exposure assessment is the assumption of ingestion of effluent without dilution. Although the degree of dilution cannot be adequately predicted on a daily basis, this assumption leads to an over-estimation of the concentration of microorganisms in drinking water. This in turn, over-estimates risk.

The assumption of percent viability for *Giardia* cysts adds uncertainty to the estimate of risk through ingestion. Bracketing the possible viability between 10 and 100 percent allows for a range of estimated risk.

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Page: 4

[DJB1] My count is 31 inorganic chemicals; includes routine constituents such as cyanide, fluoride, phosphate, forms of nitrogen as well as metals.

Page: 4

[DJB2] My count is 211.

Page: 1

[DJB3] My count is 31 inorganic chemicals; includes routine constituents such as cyanide, fluoride, phosphate, forms of nitrogen as well as metals.

Page: 1

[DJB4] My count is 211.

APPENDIX A

This appendix describes the natural and artificial sources and the environmental fate of the chemicals whose maximum detected concentration yields a hazard quotient greater than 1.0 or an excess cancer risk greater than 1×10^{-6}.

Inorganics

Five chemicals in Table 2.6-1 that exceed the health screening criteria are metals or other inorganic chemicals. All have natural as well as anthropogenic sources. Because of their diversity, their sources and environmental fates vary greatly depending upon the chemical.

Ammonia

Ammonia occurs naturally in animal waste, primarily urine (Merck 1989). Anthropogenic sources include fertilizers, explosives, fiber and plastic manufacturing, and use as a bactericide.

Ammonia is expected to adsorb readily to soil and to sediment particles in water. Under anaerobic conditions, adsorption to sediments is reduced, resulting in the release of ammonia to water or to an oxidized sediment layer. In water, ammonia is rapidly converted to nitrate via nitrification by bacteria (primarily of the genus *Nitrosomonas*) (USEPA 1995d). Nonionized ammonia (NH_3) is the principal toxic form of ammonia (Prager 1989). It is a lethal toxin at very low concentrations to many aquatic life forms.

Arsenic

Arsenic is a naturally-occurring element. Its concentration in minimally disturbed soils varies from less than 5.2 ppm to 100 ppm (USGS 1984). Anthropogenic sources include pottery glazes, metal smelting, pesticides, and chemical manufacturing.

Elemental arsenic is persistent in both water and soil. Environmental processes may transform one arsenic compound to another, however, arsenic itself is not degraded. Soluble forms of arsenic tend to be quite mobile in water, while less soluble species adsorb to clay or soil particles (ATSDR 1991a). Bioconcentration of arsenic occurs in aquatic organisms, primarily in algae and lower invertebrates. Biomagnification in aquatic food chains does not appear to be significant, although some fish and invertebrates contain high levels of arsenic compounds which are relatively inert toxicologically. Plants may accumulate arsenic, subject to various factors including soil arsenic concentration, plant type, and soil characteristics (ATSDR 1991a).

Chromium

Chromium is a naturally-occurring element which is dispersed throughout the environment primarily as a result of anthropogenic activities (e.g., combustion of coal and oil) (ATSDR 1991b). The concentration of chromium in minimally disturbed soils varies from less than 37 ppm to 700 ppm (USGS 1984). Chromium occurs in the environment in primarily its trivalent (III) and hexavalent (VI) forms. The most toxic form is chromium (VI), which is produced by chemical manufacture, primary metal production, chrome plating, and cooling towers (ATSDR 1991b).

There are no known chromium compounds that can volatilize from water. Most chromium (III) is expected to precipitate in sediments. Chromium (VI) will be present predominantly in the soluble form. Chromium (VI) will eventually be reduced to the trivalent form by the organic materials present in surface water. Chromium in soil may become airborne due to fugitive dust emissions, while runoff and leaching may transport it to surface water and groundwater. Flooding of soils and the subsequent anaerobic decomposition of plant material may increase the mobilization of chromium from soils. The half-life of chromium in soils may be several years. The residence time of chromium in lake water is estimated to be in the range of 4.6 to 18 years. Bioconcentration is expected to be minimal (ATSDR 1991b). In general, chromium (VI) is more toxic than chromium (III).

Nitrate

Natural sources of nitrate include vegetables such as beets, celery, lettuce, and spinach, as well as mineralization of soil organic matter (Sittig 1985). Anthropogenic sources include farm fertilizer and animal wastes, lawn fertilizer, leachate from waste disposal in sanitary landfills and dumps, atmospheric sources, and nitric oxide and nitrite discharges from automobile exhausts (Sittig 1985).

Nitrates may be found in the environment bound with organic and/or inorganic matter. The fate and transport of nitrates, therefore, is dependent upon those properties associated with the nitrate-bound material. Any discussion attempting to encompass all properties of nitrate-bound materials is beyond the scope of this assessment.

Nitrite

Naturally-occurring nitrite is found bound to organic and/or inorganic matter in the environment. Anthropogenic sources include sodium nitrite used in the manufacture of diazo dyes, and in numerous processes involving the manufacture of organic chemicals; textile fabric dyeing and printing; bleaching processes of silk, flax, and linen; photography; and meat curing, coloring and preserving (Merck 1989).

Because nitrites in the environment are generally bound with organic and/or inorganic matter, the fate and transport of nitrites is dependent upon those properties associated

with the nitrite-bound material. Any discussion attempting to encompass all properties of nitrite-bound materials is beyond the scope of this assessment.

Volatile Organic Chemicals

Six chemicals in Table 2.6-1 that exceed the health screening criteria are volatile organic chemicals. Volatile organic chemicals are a diverse group of organic compounds whose common characteristics are that they have a relatively low molecular weight and readily volatilize. They may have both natural (several of the chlorinated chemicals are produced by marine algae or plants) and anthropogenic sources. Because of their diversity their environmental fate varies greatly.

Chloromethane

Natural sources of chloromethane include volcanoes, plant volatiles, forest fires, and seawater. Anthropogenic sources include the manufacture of silicones, agrochemicals, methyl cellulose, quaternary amines, butyl rubber, and tetraethyl lead; tobacco smoke; turbine exhaust; wood, field, and backyard burning; chlorination of waters; solvents; propellants; and fumigants (Howard 1989).

Chloromethane released to surface soils or water will readily volatilize. Chloromethane shows little ability to adsorb to soils and/or sediments, but there is a potential for it to leach to groundwater where it may biodegrade and hydrolyze very slowly. The half-life of chloromethane in soil and surface water is one to four weeks. It is not expected to bioconcentrate or bioaccumulate in aquatic organisms.

1,4-Dichlorobenzene (1,4-DCB)

There are no natural sources of 1,4-dichlorobenzene. Anthropogenic sources include chemical manufacture and disposal, room deodorants, moth fumigants, and polyphenylene sulfide resin-production[DJB1] intermediate (Howard 1989, Merck 1989).

Volatilization of 1,4-DCB is an important mechanism for transport in soil, as well as being the dominant mechanism for removal from water. Tight adsorption of 1,4-DCB to soils and sediments attenuates volatilization. 1,4-DCB will undergo aerobic biodegradation in soils and waters, but is not expected to hydrolyze, photolyze, oxidize, or anaerobically biodegrade. The half-life[DJB2] of 1,4-DCB in soil and surface water is one to six months. Significant bioconcentration of 1,4-DCB in aquatic organisms is not expected (Howard 1989).

Methylene Chloride

There are no natural sources of methylene chloride. Anthropogenic sources include aerosol propellant, paint remover, metal degreaser, urethane foam blowing agent, paint and ink industries, aluminum forming, coal mining, photographic equipment, the

pharmaceutical, organic chemicals and plastics, and rubber processing industries, foundries, and laundries (Howard 1990, Merck 1989).

Methylene chloride released to soil will volatilize quickly from near-surface soils. That which does not volatilize is expected to leach through soils to groundwater. Under normal environmental conditions, hydrolysis in soils and/or groundwaters is not expected. Aerobic biodegradation of methylene chloride is reported to be complete (within six hours to seven days) and anaerobic biodegradation will proceed after a variable-length acclimation period. The primary removal process of methylene chloride from surface waters is volatilization. Biodegradation of methylene chloride is possible in natural waters, but will be a slow process relative to volatilization. Hydrolysis in surface waters, under normal environmental conditions, is not expected. The half-life of methylene chloride in soil and surface water is one to four weeks (Howard et al. 1991). Methylene chloride is not expected to bioconcentrate (BCF = 5) in aquatic biota (Howard 1990).

Trihalomethanes

Three chemicals in Table 2.6-1 that exceed the health screening criteria belong to a subgroup of volatile organic chemicals known as trihalomethanes (bromoform, which was not detected in samples, is also classified as a trihalomethane). They are found in drinking water and wastewater primarily because of their formation as by-products of the chlorination process. Their environmental fates are similar. They volatilize relatively quickly from surface waters and do not significantly bioaccumulate.

Bromodichloromethane

A natural source of bromodichloromethane is marine microalgae. Anthropogenic sources include chlorination treatment processes of drinking water, wastewater, and cooling waters (Howard 1989).

Bromodichloromethane will volatilize from soils. Where volatilization does not occur readily, bromodichloromethane is expected to leach to groundwater. Once leached to subsurface soils and/or groundwater, anaerobic biodegradation may be the major removal mechanism. For releases to surface waters, volatilization is also the dominant removal mechanism. Hydrolysis, oxidation, photolysis, and adsorption are not considered environmentally important. In addition, bioconcentration of bromodichloromethane in aquatic organisms is not considered an important removal mechanism (log BCF = 1.37) (Howard 1989). The volatile half-life in rivers and streams has been estimated at 33 minutes to 12 days, with a typical half-life being 35 hours (Micromedex, Inc. 1992). Aquatic hydrolysis half-life at 25°C and pH 7 is 137 years, so it is an unlikely removal mechanism (Sax 1984).

Chloroform

Natural sources of chloroform include plants. Anthropogenic sources include the chemical industry, chlorination of drinking water, municipal sewage, power plants, auto exhaust, the dry cleaning industry, fumigation, and manufacturing (Howard 1990).

Chloroform will volatilize from soil and water. It is not adsorbed significantly on soils or sediment. Chloroform in soils will leach to groundwater, where it may remain for long periods of time or until discharged. Since it is substantially denser than water, when it occurs as a separate phase it tends to sink to the bottom of the aquifer. Releases to surface soils and water will be dissipated primarily by volatilization. It is subject to significant biodegradation. It is not expected to bioconcentrate in aquatic organisms (Howard 1990). The half-life of chloroform in soil and surface water is one to six months (Howard et al. 1991).

Dibromochloromethane

Natural sources of dibromochloromethane include marine algae. Anthropogenic sources include chlorination treatment processes of drinking water, wastewater, and cooling water (Howard 1990).

Dibromochloromethane will volatilize from soils. Where volatilization does not occur, dibromochloromethane is expected to leach to groundwater. Once leached to subsurface soils and/or groundwater, anaerobic biodegradation may be the major removal mechanism. For releases to surface waters, volatilization is the dominant removal mechanism. Hydrolysis, oxidation, photolysis, and adsorption are not considered environmentally important. In addition, bioconcentration of dibromochloromethane in aquatic organisms is not considered an important removal mechanism (log $BCF_{[DJB3]}$ = 1.47) (Howard 1990). The half-life of dibromochloromethane in soil and surface water is one to six months (Howard et al. 1991).

Phthalates

One chemical in Table 2.6-1 that exceeds the health screening criteria is a phthalate. Phthalates are a widely used group of chemicals found in a variety of consumer and commercial products. They are used primarily as plasticizers for polyvinyl and cellulosic resins (e.g., polyvinylchloride), and may also be found in insect repellents, cosmetics, rubbing alcohol, liquid soap, detergents, decorative inks, lacquers, munitions, industrial and lubricating oils, and defoaming agents during paper and paperboard manufacture. Their environmental fate varies (some degrade or volatilize more readily than others) and they have a low to moderate tendency to bioaccumulate.

Bis(2-ethylhexyl) phthalate (DEHP)

Natural sources of bis(2-ethylhexyl) phthalate may include animal and/or plant life. Anthropogenic sources include use as a plasticizer for polyvinylchloride (PVC) and other

polymers, disposal or incineration of plastics or polymers, and use in a wide variety of consumer products (Howard 1989).

DEHP has a strong tendency to adsorb to soils and sediments, suggesting low likelihood of leaching to groundwaters. Volatilization from soils and waters is unlikely. DEHP may bioconcentrate in aquatic organisms. Hydrolysis (from aquatic systems), photolysis (in the water and atmosphere), and photo-oxidation (in atmospheric systems) are not expected to be important removal processes. In water, aerobic biodegradation occurs rapidly following acclimation, but no anaerobic biodegradation occurs. Some slight biodegradation in soils is expected (Howard 1989). The half-life of DEHP in soil and surface water is five to 23 days. DEHP is the most well studied of the phthalate esters. Most information reported in the technical literature deals with phthalate esters as a group. Autian (1973) suggests there is evidence phthalate esters are degraded by microbes and metabolized by fish and animals. As a result, phthalate esters are not likely to biomagnify. According to Arthur D. Little, Inc. (1985), phthalate esters readily complex with natural organic substances (e.g., fulvic acid) to form complexes which are very soluble in water. DEHP is nonvolatile, strongly adsorbed, and may bioaccumulate (BCF = 100 to 10,000). However, it is metabolized by fish.

Pesticides

Four chemicals listed in Table 2.6-1 that exceed the health screening criteria are pesticides. All belong to the organochlorine group of pesticides. Because of their known adverse effects on human health and the environment the use of aldrin, heptachlor, and α-lindane has been canceled or greatly reduced in the United States. γ-lindane is an active ingredient in currently registered pesticides.

As a group, organochlorine pesticides are slow to degrade and persist in the environment. They also bioaccumulate.

Aldrin

There are no natural sources of aldrin. It was formerly used as a pesticide. Biodegradation or metabolism of aldrin produces dieldrin, which is also a manufactured pesticide.

Aldrin is considered to be moderately persistent. Biodegradation of aldrin should be slow and it should not leach to groundwaters. Photooxidation in water is significant. The half-life of aldrin in soil and surface water is three weeks to 1.6 years. Dieldrin is an extremely persistent compound and may persist in soils for periods exceeding seven years. Dieldrin's low water solubility make leaching into groundwaters unlikely. Soil runoff may carry particle-adsorbed dieldrin to the water system. Dieldrin in water systems will not undergo hydrolysis or appreciable biodegradation; photoarrangement to photodieldrin is a possibility. Adsorption to sediments/suspended solids in waters, and moderate to significant bioconcentration in aquatic organisms are predicted to be important transport/fate mechanisms. Volatilization from waters may be an important

process. Volatilization from soils, slight in any case, will increase as the moisture content of the soils increases (Howard 1991). The half-life of dieldrin in soil and surface water is 175 days to three years.

Both chemicals have a high environmental toxicity for invertebrates and are also quite toxic to fish, birds, and mammals. They are expected to bioaccumulate, with BCFs of about 10^5 in fish tissue, ostracods[DJB4], and snails; 10^3 in algae, freshwater vascular plants (*Elodea*), and clams; and 10^2 for crabs.

Endosulfan

There are no natural sources of endosulfan. Technical grade endosulfan is composed of α- and β-endosulfan (endosulfan I and II, respectively). It is a pesticide used to control various insects and mites on cereal, cotton, fruits and vegetables (USEPA 1995d).

In soil, endosulfans will most likely biodegrade and hydrolyze, especially under alkaline conditions. Endosulfans on the soil surface may photodegrade. Volatilization and leaching are not expected to be significant because endosulfan adsorbs strongly to soils. In water, endosulfans are expected to hydrolyze readily under alkaline conditions, and more slowly at neutral and acidic pH values (alpha half-lives are 35.4 and 150.5 days for pH 7 and 5.5, respectively; beta half-lives are 37.5 and 187.3 days for pH 7 and 5.5, respectively). Volatilization and biodegradation are also expected to be significant. Photolysis and oxidation may also be important. Bioconcentration of endosulfan is expected to be significant.

Lindane (α- and γ-hexachlorocyclohexane)

There are no natural sources of lindane. Anthropogenic sources include its use in insecticides, pediculicides, scabicides, and ectoparasiticides (Howard 1991).

Lindane in soil is not expected to bind tightly to organic matter. Given its low solubility in water, lindane is expected to leach slowly to groundwater. Lindane is expected to volatilize from moistened soils. Some biodegradation is expected to occur, with anaerobic biodegradation proceeding more rapidly than aerobic biodegradation. Lindane released to water may adsorb to sediments and suspended solids. Lindane is expected to bioconcentrate in aquatic organisms in degrees ranging from slight to significant (BCF = 63 to 1,622). Lindane will volatilize from waters, but the degree of volatilization is greatly dependent upon the depth of the water (i.e., much slower volatilization at greater depths). Biodegradation, hydrolysis, and photolysis are all loss mechanisms for lindane in aqueous environments. In addition, since products of lindane hydrolysis are more susceptible to photolysis, it is expected that lindane in the aqueous environment will undergo a series of reactions (Howard 1991). The half-life of lindane in soil and surface water is 13.8 to 240 days (Howard et al. 1991). Bioconcentration factors range from 100 to 1,000 in aquatic invertebrates and fish.

Heptachlor

There are no natural sources of heptachlor. Heptachlor was manufactured in the past for use as an insecticide. Since 1983 its use has been restricted to termite control.

Heptachlor strongly adsorbs to soils and should not leach extensively to groundwater. In soil, heptachlor will degrade to heptachlor epoxide, which is more persistent than heptachlor, and other chemical species. Significant biodegradation of heptachlor occurs under both aerobic and anaerobic conditions. On the soil and water surface, heptachlor may photodegrade or volatilize. The volatilization half-life of heptachlor in water is estimated to range from two to ten days (Howard 1991). Heptachlor epoxide adsorbs strongly to soils and sediments/suspended solids in waters. It is not expected to leach significantly to lower soil layers or to groundwater. Little or no biodegradation, under aerobic or anaerobic conditions, is expected to occur in either soil or water. Bioconcentration of both chemicals (BCF greater than 10,000 in some species) is expected to occur readily. The half-life of heptachlor in soil and surface water is about 23 hours to five days. The half-life of heptachlor epoxide in soil and surface water is 33 days to 1.5 years (Howard et al. 1991).

APPENDIX B

This appendix describes toxicity data for the chemicals detected in the effluent samples collected from the Laguna Plant. Toxicity data were obtained from the USEPA's Integrated Risk Information System (IRIS) and the National Library of Medicine's Hazardous Substances Database (HSDB) unless otherwise indicated. Both information sources are available on the National Library of Medicine's data network system (TOXNET).

Inorganics

Because of their diversity, the toxicity of this group varies greatly depending upon the chemical.

Aluminum

The primary targets of aluminum toxicity are the central nervous system, skeletal system, respiratory system and the developing fetus. Aluminum has a relatively low toxicity and daily doses of several grams of aluminum are not unusual in individuals using antacids. Excess aluminum has been associated with neurodegenerative diseases (e.g., Alzheimer's disease), although the current scientific literature indicates that excess aluminum in the brains of affected patients is a symptom of the disease, not its underlying cause. [DJB5]

The USEPA is evaluating the oral RfD for aluminum and does not currently publish a value on IRIS or HEAST. A chronic oral RfD of 1 mg/kg/day was obtained from Region IX, San Francisco of the USEPA (USEPA 1995c). The experimental evidence on which this value is based was not available, although the high RfD (relative to most other metals) is consistent with aluminum's low toxicity.

Aluminum is not known to cause cancer in humans or animals, and has not been placed in a USEPA weight-of-evidence cancer group.

Ammonia

The major targets of ammonia toxicity are the respiratory system and the eyes.

A chronic oral RfD of 1 mg/kg/day is based on a NOAEL of 34 mg/L for the taste threshold in humans. Because the RfD is based on the study of a human that includes sensitive subpopulations, no uncertainty factor was applied to convert the NOAEL to the RfD.

The USEPA has not placed ammonia in a weight-of-evidence cancer group.

Arsenic

Arsenic is a long-recognized human poison capable of producing a lethal reaction and cancer. The major targets of arsenic toxicity are the respiratory system, gastrointestinal system, nervous system, blood-forming (hematological) system and skin (ATSDR 1991a). Animal studies suggest that low levels of arsenic may be necessary to maintain good health, but this has not been shown in humans (ATSDR 1991a).

A chronic RfD of 0.0003 mg/kg/day is based on a NOAEL of 0.009 mg/L for dermal effects and possible effects on blood vessels. An uncertainty factor of three (to account for a lack of data to preclude reproductive toxicity and sensitive individuals) was used to convert the NOAEL to the chronic RfD.

Arsenic is considered a weak mutagen and has been placed in weight-of-evidence cancer Group A, indicating that it is a human carcinogen. An oral slope factor of 1.5 $(mg/kg/day)^{-1}$ is based on the occurrence of skin cancer in humans. The DTSC has not derived an oral slope factor for arsenic. Arsenic has been identified as presenting no significant risk of cancer by ingestion by the State of California so long as the anticipated level of exposure does not exceed 10 μg/day [Title 22, California Code of Regulations, §12709 (22 CCR 12709)].

Asbestos

The major targets of asbestos toxicity are the lungs and gastrointestinal system. The USEPA has placed asbestos in weight-of-evidence Group A, indicating that it is a human carcinogen. A chronic oral RfD or an oral slope factor are not available on IRIS or HEAST and have not been derived by the DTSC. Asbestos has been identified as presenting no significant risk of cancer by ingestion by the State of California (22 CCR 12707).

Barium

The primary target of barium toxicity is the cardiovascular system.

A chronic oral RfD of 0.07 mg/kg/day is based on a NOAEL of 0.21 mg/kg/day for increased blood pressure in a long-term drinking water study in humans. The study concentrated on the most sensitive population and was supported by other studies. An uncertainty factor of three (to protect the most sensitive individuals) was used to convert the NOAEL to the RfD.

Information regarding the genotoxicity of barium is equivocal and the USEPA has placed barium in weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity (USEPA 1994c).

Boron

The primary targets of boron exposure are the testes, gastrointestinal system, liver, kidneys, and brain. Boron is also a nose, throat, and eye irritant.

A chronic oral RfD of 0.09 mg/kg/day is based on a NOAEL of 8.8 mg/kg/day for testicular atrophy and lack of sperm production in a chronic study in dogs. An uncertainty factor of 100 (to account for interspecies variability) was used to convert the NOAEL from the lifetime animal study to the RfD for humans.

The USEPA has placed boron in weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity (USEPA 1994c).

Cadmium

High cadmium levels in the diet irritate the digestive tract, while lower levels consumed over a long period of time may cause kidney damage.

A chronic oral RfD of 0.0005 mg/kg/day for water is based on a NOAEL of 0.005 mg/kg/day for abnormally high levels of protein in the urine following chronic exposures in humans. An uncertainty factor of ten (to account for intrahuman variability) was used to convert the NOAEL to the RfD.

The USEPA has placed cadmium in weight-of-evidence Group B1, indicating that it is a probable human carcinogen. An oral slope factor was not available on IRIS or HEAST and has not been derived by the DTSC. Cadmium has been identified as presenting no significant risk of cancer by ingestion by the State of California (22 CCR 12707).

Chromium

Chromium (III) is an essential nutrient which helps to maintain normal glucose, cholesterol, and fat metabolism. A daily ingestion of 0.05 to 0.20 mg/day (0.0007 to 0.003 mg/kg/day) is estimated to be safe and adequate (ATSDR 1991b).

The major targets of chromium toxicity are the respiratory system and the gastrointestinal system.

A chronic oral RfD of 1 mg/kg/day for chromium (III) is based on a NOEL of 1,468 mg/kg/day for adverse effects in a chronic feeding study in rats. An uncertainty factor of 100 (to account for interhuman and interspecies variability) was used to convert to the NOEL to the RfD. An oral RfD of 0.005 mg/kg/day for chromium (VI) is based on a NOAEL of 2.4 mg/kg/day for adverse effects in a 1-year drinking study in rats. An uncertainty factor of 500 (to account for interhuman and interspecies variability and to compensate for the less-than-lifetime exposure duration) was used to convert the NOAEL to the RfD.

Chromium (III) has not been placed in a cancer class by the USEPA. Chromium (VI) is considered to be genotoxic and the USEPA has placed it in weight-of-evidence cancer Group A, indicating that it is a human carcinogen. An oral slope factor is not available on IRIS or HEAST. Although chromium (VI) has been identified as presenting no significant risk of cancer by ingestion by the State of California (22 CCR 12707), an oral slope factor of 0.42 $(mg/kg/day)^{-1}$ has been derived by the DTSC.

Copper

Copper is an essential trace element; therefore, toxic effects can result if too much or too little is taken into the body. Because it is an essential nutrient, it is strongly accumulated by all plants and animals, but is probably not biomagnified. The Recommended Dietary Allowance (RDA) for copper is 2 to 3 mg/day (0.03 to 0.04 mg/kg/day).

The major targets of copper toxicity are the gastrointestinal tract following oral exposure and the lungs following inhalation exposure.

A chronic oral RfD of 1.3 mg/L (0.04 mg/kg/day) is based on a LOAEL of 5.3 mg/L for gastrointestinal irritation in humans. Case studies of human suicides indicate that doses of 6 to 637 mg/kg have been fatal.

Information regarding the genotoxicity of copper is equivocal and the USEPA has placed copper in weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity.

Cyanide

Cyanide is highly toxic to humans following all routes of exposure. Cyanide inhibits enzymes that are needed to use oxygen efficiently, resulting in respiratory arrest. The major targets of cyanide toxicity are the central nervous system, the lungs and the heart. The lowest reported fatal dose in humans was 0.56 mg/kg. The average fatal dose of cyanide in humans following dermal exposure was estimated to be 100 mg/kg.

A chronic oral RfD of 0.02 mg/kg/day is based on the NOAEL of 10.8 mg/kg/day for weight loss, thyroid effects and nervous system effects in a chronic study in rats. An uncertainty factor of 100 (to account for interhuman and interspecies variability) was used to convert the NOAEL to the RfD.

Cyanide is not mutagenic and has been placed in weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity.

Fluoride

The major targets of fluoride toxicity are the teeth and the bones.

Fluoride is added to municipal water supplies to prevent dental caries. In California, the maximum amount of added fluoride that is allowed by law varies from 0.8 mg/L to 1.7

mg/L depending upon temperature (the higher concentrations are allowed at lower temperatures).

A chronic oral RfD of 0.06 mg/kg/day is based on a NOAEL of 0.06 mg/kg/day for objectionable dental fluorosis in an epidemiological study in children. In humans, a recent study reported that a dose of 16 mg sodium fluoride/kg was fatal to a child.

Fluoride has been found to be genotoxic only at high doses and the USEPA has not placed fluoride in a weight-of-evidence cancer group.

Lead

The major health threat from lead arises from the damage it causes to the brain, especially in fetuses, infants, and young children. Fetal exposure may also result in preterm birth, reduced birth weight, and decreased IQ. Decreased IQ and reduced growth may result from childhood exposure. Lead exposure may increase blood pressure in middle-aged men. High-level exposure can severely damage the brain and kidneys in adults or children. In addition, high doses of lead will cause abortion and damage the male reproductive system.

The Federal Centers for Disease Control recently lowered the threshold at which children are considered to have lead poisoning from 25 to 10 micrograms (μg) of lead per deciliter (dl) of blood, a level also recognized by the DTSC (DTSC 1992). Some of the health effects of lead, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood levels so low as to be essentially without a threshold.

The traditional RfD approach to toxic chemicals is not applied to lead because most human health effects data are based on blood lead concentrations rather than external dose. Therefore, the USEPA and DTSC currently do not provide any toxicity values for lead.

The USEPA has placed lead in weight-of-evidence Group B2, indicating that it is a probable human carcinogen. However, the USEPA and DTSC currently do not provide slope factors for lead.

Mercury

The primary targets of mercury toxicity are the brain, kidneys and developing fetus. Methylmercury is a potent neurotoxin.

A chronic RfD of 0.0003 mg/kg/day is based on LOAELs and NOAELs for kidney and autoimmune disease in subchronic studies of rats. An uncertainty factor of 1,000 (to account for subchronic studies, inter- and intra-species variability, and sensitive subpopulations) was used to calculate the RfD.

The USEPA has placed inorganic mercury (mercuric chloride) in weight-of-evidence Group C, indicating that it is a possible human carcinogen, and metallic mercury in weight-of-evidence Group D, indicating that it is not classifiable as to human carcinogenicity. The USEPA and DTSC currently do not provide slope factors for any form of mercury.

Nickel

The primary targets of nickel toxicity are the respiratory, gastrointestinal and immune systems. Studies in animals suggest that low levels of nickel may be necessary to maintain good health, but this has not been shown in humans.

A chronic oral RfD of 0.02 mg/kg/day is based on a NOAEL of 5 mg/kg/day for decreased body and organ weights in a chronic oral study in rats. An uncertainty factor of 300 (to account for interhuman and interspecies variability and to account for inadequacies in the reproductive studies) was used to convert the NOAEL to the RfD. A fatal oral dose in humans of approximately 570 mg/kg has been reported.

Some forms of nickel are considered to be genotoxic but metallic nickel has been placed in weight-of-evidence cancer Group D by the USEPA, indicating that it is not classifiable as to human carcinogenicity (USEPA 1994c). Only nickel refinery dust and nickel subsulfide have been placed in Group A, indicating that they are human carcinogens. Nickel has been identified as presenting no significant risk of cancer by ingestion by the State of California (22 CCR 12707).

Nitrate

Nitrate is a normal component of the diet, with a typical daily intake of 75 mg/day (0.2 to 0.3 mg nitrate-nitrogen/kg/day) reported for U.S. adults. Over 85% of the intake comes from the natural nitrate content of vegetables, such as beets, celery, lettuce and spinach.

The primary target of nitrate toxicity is the blood, with methemoglobinemia occurring, especially in infants. Methemoglobinemia occurs when nitrate is converted in the body to nitrite, and the nitrite oxidizes hemoglobin to a form that is unable to transport oxygen. This condition results in reduced oxygen transport to tissues. Methemoglobin (MetHb) concentrations above 10% may cause cyanosis (bluish color to skin and lips). MetHb levels above 25% lead to weakness, rapid pulse and breathing, and levels exceeding 50-60% may be fatal. Infants aged less than three months are most sensitive to this condition because the infant gastrointestinal system has a normally high pH which favors the growth of nitrate-reducing bacteria, and because infants have hemoglobin F, which is more susceptible to oxidation.

A chronic oral RfD of 1.6 mg/kg/day is based on a NOAEL of 1.6 mg/kg/day for methemoglobinemia in infants (dose based upon the amount of nitrogen within the nitrate molecule).

Information regarding the genotoxic potential of nitrate was not located and the USEPA has not placed nitrate in a weight-of-evidence cancer group.

Nitrite

The toxic effects of nitrite are similar to those of nitrate, with the primary concern being methemoglobinemia.

A chronic oral RfD of 0.1 mg/kg/day is based on a NOEL of 1.0 mg/kg/day for methemoglobinemia in infants. A modifying factor of 10 was used to convert the NOEL to the RfD because of the direct toxicity of nitrite.

Information regarding the genotoxic potential of nitrite was not located and the USEPA has not placed nitrite in a weight-of-evidence cancer group.

Silver

The major targets of silver toxicity are the respiratory system following inhalation exposure and the skin following inhalation, oral and dermal exposure.

A chronic oral RfD of 0.005 mg/kg/day is based on a LOAEL of 0.014 mg/kg/day for skin discoloration in a long-term study in humans. An uncertainty factor of three (to account for minimal effects in a subpopulation which has exhibited an increased propensity for skin discoloration) was used to convert the LOAEL to the RfD.

Data suggest that silver is a mutagen but the USEPA has placed silver in weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity.

Zinc

Zinc is an essential trace element, therefore, toxic effects can result if too much or too little is taken into the body. Because it is essential it is bioaccumulated by all organisms, but does not biomagnify in terrestrial or aquatic food chains. The Recommended Dietary Allowances (RDAs) for zinc are 15 mg/day for men and 12 mg/day for women.

The major targets of zinc toxicity are the gastrointestinal tract following oral exposure and the lungs following inhalation exposure.

A chronic oral RfD of 0.3 mg/kg/day is based on a LOAEL of 1 mg/kg/day for effects on red blood cells in human females. An uncertainty factor of three was used to convert the LOAEL, based on a minimal LOAEL from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential nutrient, to the RfD.

Zinc is not mutagenic and has been placed in weight-of-evidence Group D, indicating that it is not classifiable as to human carcinogenicity, by the USEPA.

Volatile Organic Chemicals

Because of their diversity the toxicity of the volatile organic chemicals varies greatly.

Acetone

Acetone acts primarily as an irritant and as a central nervous system depressant. Fatal oral doses in humans have not been reported, but oral exposure to 200 ml (2,860 mg/kg/day) acetone has resulted in inflammation or irritation of the stomach and intestines, drowsiness, and possible kidney injury (Arthur D. Little, Inc. 1987).

A chronic oral RfD of 0.1 mg/kg/day is based on a NOEL of 100 mg/kg/day for reduced kidney and liver weights of rats. An uncertainty factor of 1,000 (to account for inter- and intraspecies extrapolation and subchronic to chronic exposure) was used to convert the NOEL to the RfD. No RfC has been derived by the USEPA, therefore the inhalation RfD is set to the same value as the oral RfD.

Acetone is not considered to be mutagenic and the USEPA has placed acetone in weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity.

Bromomethane

The primary targets of bromomethane toxicity are the central nervous system, respiratory system, skin and eyes. Data regarding the potential effects of bromomethane on reproduction and development indicate that bromomethane does not cause birth defects.

A chronic oral RfD of 0.0014 mg/kg/day is based on a NOAEL of 1.4 mg/kg/day for hyperplasia (abnormal growth by an increase in cell numbers) of the forestomach of rats. An uncertainty factor of 1,000 (to account for interspecies and intrahuman variability and to extrapolate to lifetime exposure) was used to convert the NOAEL to the RfD. A chronic inhalation RfD of 0.0014 mg/kg/day (RfC of 0.003 mg/m^3) is based on a LOAEL of 2.08 mg/m^3 for lesions in the nasal cavities of rats. An uncertainty factor of 100 (to account for intraspecies uncertainty, the use of a LOAEL, and interspecies extrapolation) was used to convert the LOAEL to the RfD.

Bromomethane has been shown to be genotoxic but has been placed in USEPA weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity.

Carbon Disulfide

The major targets of carbon disulfide toxicity are the central nervous system, heart, liver, and the developing fetus.

A chronic oral RfD of 0.1 mg/kg/day is based on a NOEL of 11 mg/kg/day fetal toxicity (malformations) in rabbits exposed to carbon disulfide via inhalation. An uncertainty

factor of 100 (to account for inter- and intra-species variability) was used to convert the NOEL to the RfD. A chronic inhalation RfD of 0.0029 mg/kg/day (RfC of 0.01 mg/m^3) is based on a NOAEL of 10 mg/m^3 for birth defects in rats. An uncertainty factor of 1,000 (to account for inter- and intraspecies variation and the use of subchronic data) was used to convert the NOAEL to the RfD.

Carbon disulfide is considered to be nonmutagenic and carbon disulfide has not been placed in a weight-of-evidence cancer group by the USEPA.

Chlorobenzene

Humans exposed to chlorobenzene have experienced headaches, numbness, sleepiness, nausea, and vomiting. Chlorobenzene has been shown to affect the brain, liver, and kidneys in animals.

A chronic oral RfD of 0.02 mg/kg/day is based on a NOAEL of 19 mg/kg/day for effects on the live of dogs exposed to chlorobenzene via inhalation. An uncertainty factor of 1,000 (to account for extrapolation from laboratory animals to humans, an unknown threshold for sensitive humans and extrapolation from subchronic to chronic exposure) was used to convert the LOAEL to the RfD. A chronic inhalation RfD of 0.0057 mg/kg/day (RfC of 0.02 mg/m3) is based on a LOAEL of 75 ppm for liver and kidney effects on rats. An uncertainty factor of 10,000 (to account inter- and intra-species variability, extrapolation from subchronic to chronic exposure and non-standard methodology) was used to convert the LOAEL to the RfD.

The USEPA has placed chlorobenzene in weight-of-evidence Group D, indicating that it is not classifiable as to human carcinogenicity.

Chloromethane

The primary target of chloromethane toxicity is the central nervous system. Chloromethane has been found to be fatal in humans following exposure to vapors from leaks in refrigeration units, but the fatal concentration is not known (ATSDR 1990b).

Chronic oral and inhalation RfDs have not been set by the USEPA.

Data indicate that chloromethane is mutagenic (ATSDR 1990b) and the USEPA has placed chloromethane in weight-of-evidence cancer Group C, indicating that it is a possible human carcinogen. An oral slope factor of 0.013 $(mg/kg/day)^{-1}$ and in inhalation slope factor of 0.0063 $(mg/kg/day)^{-1}$ are based on the occurrence of kidney tumors in mice.

1,4-Dichlorobenzene (1,4-DCB)

1,4-DCB is an eye, nose, throat and skin irritant. The lowest reported fatal dose in humans is 857 mg/kg (Arthur D. Little, Inc. 1989).

Because a chronic oral RfD has not been set by the USEPA, the inhalation RfD has been set to the same value as the inhalation RfD. A chronic inhalation RfD of 0.23 mg/kg/day (RfC of 0.8 mg/m3) is based on a NOAEL of 50 ppm for increased liver weights in male rats. An uncertainty factor of 100 (to account for interspecies variability, sensitive human subpopulations, and the use of subchronic data) was used to convert the NOAEL to the RfD.

1,4-DCB is not considered to be mutagenic (Arthur D. Little, Inc. 1989, ATSDR 1991c) but the USEPA has placed 1,4-DCB in weight-of-evidence cancer Group C, indicating that it is a possible human carcinogen. An oral and inhalation slope factor of 0.04 $(mg/kg/day)^{-1}$ has been derived by the DTSC.

Ethylbenzene

Humans exposed to ethylbenzene may experience eye and throat irritation, decreased movement, and dizziness. Animal studies have shown liver and kidney damage, nervous system changes, and blood changes.

A chronic oral RfD of 0.1 mg/kg/day is based on a NOEL of 97.1 mg/kg/day and a LOAEL of 291 mg/kg/day determined for liver and kidney toxicity in rats. An uncertainty factor of 1,000 (to account for inter- and intra-species variability and extrapolation from a subchronic effect level to a chronic equivalent) was used to convert the NOEL to the RfD. A chronic inhalation RfD of 0.29 mg/kg/day (RfC of 1 $mg/m^{3)}$ is based on a NOAEL of 434 mg/m^3 determined for developmental toxicity in rats and rabbits exposed via inhalation. An uncertainty factor of 300 (to account for interhuman variability, interspecies variability, and the absence of multigenerational reproductive and chronic studies) was used to convert the NOAEL to the RfD.

The USEPA has placed ethylbenzene in weight-of-evidence Group D, indicating that it is not classifiable as to human carcinogenicity.

Methylene Chloride

The major targets of methylene chloride toxicity are the central nervous system, the liver, and the kidneys (ATSDR 1990c).

The chronic oral RfD of 0.06 mg/kg/day is based on a NOAEL of 6 mg/kg/day for liver toxicity in a chronic oral study in rats. An uncertainty factor of 100 (to account for intra- and inter-species variability) was used to convert the NOAEL to the RfD. The chronic inhalation RfD of 0.86 mg/kg/day (RfC of 3 mg/m^3) is based on a NOAEL of 694.8 mg/m^3 for liver toxicity in a chronic study in rats. An uncertainty factor of 100 was applied for the same reasons as above.

Information regarding the mutagenicity of methylene chloride are equivocal but the USEPA has placed methylene chloride in weight-of-evidence cancer Group B2, indicating that it is a probable human carcinogen. An oral slope factor of 0.014

$(mg/kg/day)^{-1}$ and an inhalation slope factor of 0.0035 $(mg/kg/day)^{-1}$ have been derived by the DTSC.

Tetrachloroethylene

The primary targets of tetrachloroethylene toxicity are the central nervous system, the liver, and the kidneys.

A chronic oral RfD of 0.01 mg/kg/day is based on a NOAEL of 14 mg/kg/day for liver toxicity in mice and weight gain in rats following subchronic administration of tetrachloroethylene. An uncertainty factor of 1,000 (to account for interhuman and interspecies variability and use of a subchronic effect level) was used to convert the NOAEL to the RfD. No RfC has been derived by the USEPA, therefore the inhalation RfD is set to the same value as the oral RfD.

Tetrachloroethylene is not considered to be mutagenic. The USEPA has not adopted a final position on the weight-of-evidence cancer classification for tetrachloroethylene, but an oral slope factor and inhalation unit risk have been derived. An oral slope factor of 0.051 $(mg/kg/day)^{-1}$ and an inhalation slope factor of 0.021 $(mg/kg/day)^{-1}$ have been derived by the DTSC.

Toluene

Toluene acts primarily on the central nervous system.

A chronic RfD of 0.2 mg/kg/day is based on a NOAEL of 223 mg/kg/day for changes in liver and kidney weights in a subchronic oral study in rats. An uncertainty factor of 1,000 (to account for inter- and intra-species variability, the use of subchronic data, and the lack of reproductive and developmental data) was used to convert the NOAEL to the RfD. The chronic inhalation RfD of 0.11 mg/kg/day (RfC of 0.4 mg/m^3) is based on a LOAEL of 88 ppm for central nervous system effects observed in humans following inhalation exposure. An uncertainty factor of 300 (to account for interhuman and interspecies variability, and for data base deficiencies) was used to convert the LOAEL to the RfD.

The USEPA has placed toluene in weight-of-evidence Group D, indicating that it is not classifiable as to human carcinogenicity.

1,1,1-Trichloroethane (TCA)

TCA is generally regarded as being of moderate to low toxicity. The primary target of TCA toxicity in humans is the central nervous system. TCA is also a skin and eye irritant.

A chronic RfD of 0.09 mg/kg/day is based on a NOAEL of 500 ppm for liver toxicity in guinea pigs. An uncertainty factor of 1,000 (to account for inter- and intra-species variability, and the use of subchronic data) was used to convert the NOAEL to the RfD.

This value is currently under review by the USEPA. There is no current RfC for TCA, therefore the inhalation RfD is set to the same value as the oral RfD.

Information regarding the mutagenicity of TCA are equivocal. The USEPA has placed TCA in weight-of-evidence Group D, indicating that it is not classifiable as to human carcinogenicity.

Xylenes

The primary target of xylenes toxicity is the central nervous system. Death in humans has been reported following the ingestion of xylenes, but the fatal dose is not known. Death following inhalation of approximately 10,000 ppm xylenes has been fatal.

A chronic oral RfD of 2 mg/kg/day is based on a NOAEL of 250 mg/kg/day for hyperactivity, decreased body weight and increased male mortality in a chronic study in rats. An uncertainty factor of 100 (to account for interspecies variation and to protect sensitive individuals) was used to convert the NOAEL to the RfD. There is no current RfC for xylenes, therefore the inhalation RfD is set to the same value as the oral RfD.

Xylenes are not considered to be genotoxic and the USEPA has placed xylenes in weight-of-evidence cancer Group D, indicating that they are not classifiable as to human carcinogenicity.

Trihalomethanes

The toxic effects of the trihalomethanes are similar. At effective doses they may adversely affect the central nervous system, liver and kidneys. They are classified by the USEPA as possible or probable carcinogens.

Bromodichloromethane

Oral studies in animals suggest that the liver, kidneys, and the central nervous system are the targets of bromodichloromethane toxicity.

A chronic oral RfD of 0.02 mg/kg/day is based on a LOAEL for kidney effects in a chronic study in mice. An uncertainty factor of 1,000 (to account for interspecies extrapolation, sensitive human subpopulations, the use of a LOAEL, and the lack of reproductive studies) was used to convert the LOAEL to the RfD. No RfC has been derived by the USEPA, therefore the inhalation RfD is set to the same value as the oral RfD.

Bromodichloromethane appears to be mutagenic and the USEPA has placed it in weight-of-evidence cancer Group B2, indicating that it is a probable human carcinogen. Oral and inhalation slope factors of 0.13 $(mg/kg/day)^{-1}$ have been derived by the DTSC.

Chloroform

Chloroform exerts adverse effects on the central nervous system, liver, and kidneys. High doses have been found to cause liver and kidney cancer in experimental animals (ATSDR 1987). Reported fatal oral doses for humans ranged from 212 to 3,755 mg/kg.

A chronic oral RfD[DJB6] of 0.01 mg/kg/day is based on a LOAEL[DJB7] of 12.9 mg/kg/day determined for fatty cyst formation following chronic administration to dogs. An uncertainty factor[DJB8] of 1,000 (to account for interspecies extrapolation, sensitive human subpopulations, and the use of a LOAEL) was used to convert the LOAEL to the RfD. No RfC has been derived by the USEPA, therefore the inhalation RfD is set to the same value as the oral RfD.

The USEPA has placed chloroform in weight-of-evidence Group B2, indicating that it is a probable human carcinogen. An oral slope factor[DJB9] of 0.031 $(mg/kg/day)^{-1}$ and an inhalation slope factor of 0.019 $(mg/kg/day)^{-1}$ have been derived by the DTSC.

Dibromochloromethane

The major target of dibromochloromethane toxicity is the central nervous system (ATSDR 1990a).

A chronic oral RfD of 0.02 mg/kg/day is based on a NOEL of 21.4 mg/kg/day for liver lesions in a subchronic study in rats. An uncertainty factor of 1,000 (to account for interspecies variation, sensitive human subpopulations, and use of subchronic assay) was used to convert the NOEL to the RfD. Dibromochloromethane does not meet the USEPA's criteria for volatility and no RfC has been derived by the USEPA.

Information regarding the mutagenicity of dibromochloromethane is equivocal, but suggests that the compound is a mutagen and the USEPA has placed dibromochloromethane in weight-of-evidence Group C, indicating that it is a possible human carcinogen. An oral slope factor of 0.094 $(mg/kg/day)^{-1}$ has been derived by the DTSC.

Phthalates

The toxicity of phthalates is generally low to moderate, although DEHP has been classified as a probable carcinogen by the USEPA.

Bis(2-ethylhexyl) phthalate (DEHP)

Animal studies indicate that the liver, kidneys and testes are targets of DEHP exposure.

A chronic oral RfD of 0.02 mg/kg/day is based on a LOAEL of 19 mg/kg/day for increased relative liver weight in a chronic oral study in guinea pigs. An uncertainty factor of 1,000 (to account for interspecies extrapolation, sensitive human subpopulations, less-than-lifetime exposure, and use of a minimally adverse LOAEL)

was use to convert the LOAEL to the RfD. DEHP has not been found to be fatal to humans at doses up to 143 mg/kg. Mild abdominal pain and diarrhea were the only effects reported at this dose (ATSDR 1991d).

Information regarding the genotoxicity of DEHP is equivocal but indicates that DEHP may act as a co-carcinogen in rodents (ATSDR 1991d). The USEPA has placed DEHP in weight-of-evidence cancer Group B2, indicating that it is a probable human carcinogen. An oral slope factor of 0.0084 $(mg/kg/day)^{-1}$ has been derived by the DTSC.

Diethyl phthalate (DEP)

Both the acute and chronic toxicity of DEP appear to be very low.

A chronic oral RfD of 0.8 mg/kg/day is based on a NOAEL of 750 mg/kg/day for decreased growth rate and food consumption, and altered organ weights in a subchronic study in rats. An uncertainty factor of 1,000 (to account for interspecies extrapolation, sensitive human subpopulations and use of subchronic data) was used to convert the NOAEL to the RfD.

DEP is considered to be nonmutagenic and information regarding the carcinogenicity of DEP are not available (Arthur D. Little, Inc. 1987). The USEPA has placed DEP in weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity.

Di-n-butyl phthalate (DBP)

Animal studies suggest that DBP interferes with normal reproduction.

A chronic RfD of 0.1 mg/kg/day is based on a NOAEL of 125 mg/kg/day and a LOAEL of 600 mg/kg/day for increased mortality in a rat subchronic to chronic oral bioassay. An uncertainty factor of 1,000 (to account for interspecies extrapolation, sensitive human subpopulations, and for deficiencies in the study and use of less-than-chronic data) was used to convert the NOAEL to the RfD.

The USEPA has placed DBP in weight-of-evidence Group D, indicating that it is not classifiable as to carcinogenicity.

Pesticides

As a group organochlorine pesticides are both acutely toxic at high concentrations and may cause a variety long-term adverse health effects at low concentrations. Aldrin, lindane and heptachlor are classified as probable carcinogens by the USEPA. Aldicarb and its degradation products are acutely toxic at high doses and are not classified as a carcinogens by the USEPA.

Aldicarb

The available toxicity values are for aldicarb sulfone, one of the oxidative metabolites of aldicarb. Aldicarb sulfone inhibits the enzyme cholinesterase, which is found in the brain and blood.

An oral RfD of 0.001 mg/kg/day was based on a NOAEL of 0.11 mg/kg/day and a LOAEL of 0.58 mg/kg/day for brain enzyme inhibition in a one-year dog feeding study. An uncertainty factor of 100 (to account for inter- and intraspecies variation) was used to convert the NOAEL to the RfD.

The USEPA has placed aldicarb in weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity (USEPA 1994c).

Aldrin

Exposure to aldrin by oral, inhalation, and dermal routes may cause convulsions and/or kidney damage. Human deaths following exposure have been documented.

A chronic RfD for aldrin of 0.00003 mg/kg/day is based on a LOAEL of 0.025 mg/kg/day for liver toxicity in a chronic feeding study in rats. An uncertainty factor of 1,000 (to account for interhuman variability, interspecies variability, and use of a LOAEL) was applied to convert the LOAEL to the RfD.

The USEPA has placed aldrin in weight-of-evidence Group B2, indicating that it is a probable human carcinogen. An oral slope factor of 17 $(mg/kg/day)^{-1}$ for aldrin is based on liver carcinomas observed in mice maintained on a treated diet.

DCPA (Dacthal)

Target organs for DCPA include the lungs, liver, kidney, thyroid, and eyes. Human volunteers have ingested 50 mg with no observed ill effects (USEPA 1995a).

An oral RfD of 0.01 mg/kg/day is based on a NOAEL of 1 mg/kg/day and a LOAEL of 10 mg/kg/day for adverse effects to these organs in a two-year rat feeding study. An uncertainty factor of 100 (to account for interspecies extrapolation and interhuman variability) was used to convert the NOAEL to the RfD.

The USEPA has placed DCPA in weight-of-evidence cancer Group D, indicating that it is not classifiable as to human carcinogenicity (USEPA 1994c).

Endosulfan

The primary target of endosulfan is the nervous system.

A chronic oral RfD of 0.006 mg/kg/day is based on a NOAEL of 0.6 mg/kg/day for reduced body weight and adverse effects on the circulatory system in a 2-year feeding

study in rats. An uncertainty factor of 100 (to account for inter- and intraspecies variation) was used to convert the NOAEL to the RfD.

Endosulfan has not been placed in a weight-of-evidence cancer group by the USEPA.

Lindane (α- and γ-hexachlorocyclohexane)

The major effects of exposure to lindane and/or its various isomers in humans include lung irritation, heart disorders, blood disorders, headache, convulsions, and changes in the levels of sex hormones (ATSDR 1989a). High level exposure has caused death in both humans and animals. Several forms of lindane have been associated with liver cancer in rodents.

A chronic oral RfD for γ-lindane of 0.0003 mg/kg/day is based on a NOAEL of 0.33 mg/kg/day for liver and kidney toxicity following subchronic exposure in rats. An uncertainty factor of 1,000 (to account for interhuman variation, interspecies variation, and for use of subchronic data) was used to convert the NOAEL to the RfD. No chronic oral RfD for α-lindane has been derived by the USEPA. Because α- and γ-lindane are related chemically the RfD for γ-lindane will be used as a surrogate for α-lindane.

The USEPA has placed both α- and γ-lindane in weight-of-evidence Group B2, indicating that they are probable human carcinogens. An oral slope factor of 6.3 $(mg/kg/day)^{-1}$ for α-lindane is based on liver tumors observed in mice maintained on a treated diet. An oral slope factor of 1.1 $(mg/kg/day)^{-1}$ for γ-lindane has been derived by the DTSC.

Heptachlor

Tremors and convulsions have been observed in humans and animals exposed to heptachlor. No reports of human fatalities were located (ATSDR 1991e). Humans and animals metabolize heptachlor to heptachlor epoxide, which is also toxic.

A chronic RfD for heptachlor of 0.0005 mg/kg/day is based on a NOEL of 0.15 mg/kg/day and an LEL of 0.25 mg/kg/day determined for increased liver weight in a chronic feeding study of rats. An uncertainty factor of 300 (to account for interhuman and interspecies differences and for the lack of chronic toxicity data in a second species) was used to convert the NOEL to the RfD.

The USEPA has placed heptachlor in weight-of-evidence Group B2, indicating that it is a probable human carcinogen. An oral slope factor of 3.7 $(mg/kg/day)^{-1}$ has been derived by the DTSC.

Radioactivity

Data for two types of radioactive emissions, gross alpha and gross beta particles, were available for analysis.

Alpha and Beta Particles

An alpha particle is a helium nucleus (consisting of 2 protons and 2 neutrons) with a charge of +2 that is ejected from the nucleus of an atom. Alpha particles are the naturally-occurring decay products of elements such as radium and radon. Beta particles are emitted when a neutron decays into a proton and an electron. The electron, which is the beta particle, is ejected. Beta particles are naturally emitted from a variety of radioactive isotopes of elements, including copper and lead (Harley 1991).

Alpha and beta particles can be emitted from a variety of naturally radioactive elements, and therefore their half-life, mobility, and presence in the environment will depend upon their parent element. Both particles are classified as ionizing radiation, because their kinetic energy is lost through the ionization of surrounding molecules. Alpha and beta particles vary in the amount of energy they contain, but alpha particles lose their energy at a faster rate as they travel through the environment. Alpha particles will not travel as far as beta particles with an equivalent energy, but their energy loss will be concentrated in a smaller amount of space (USEPA 1981). Alpha particles can lose approximately 10 to 20 percent of their energy traveling through 10 μm of tissue or 1 cm of air (Harley 1991). Thus, alpha particles are likely to cause more severe but more localized damage to human tissue than beta particles, per unit of energy.

Elements emitting alpha or beta particles may be ingested, inhaled, or encountered externally. Internal exposure is of particular concern because radioactive material may become lodged in the body and could potentially emit radioactive particles for the lifetime of the recipient, depending on the half-life of the element involved. Ionizing radiation may damage any part of the body through disruption of DNA or other cellular molecules. The primary health risk of exposure to alpha or beta particles is cancer. Other health effects include developmental defects in fetuses and cataracts in adults. All ionizing radiation is mutagenic (USEPA 1981).

As with chemical exposure the amount of radioactive exposure is expressed as a dose. For radioactive materials the exposure may be expressed as an absorbed dose, the amount of energy (e.g., joules) absorbed per kilogram of matter, or a dose equivalent, the product of the absorbed dose and a quality or modifying factor. Common units of absorbed dose include Grays (1 Gray = 1 joule/kg) and rads (1 rad = 0.01 Grays). Common units of dose equivalents include Sieverts and rems (1 rem = 0.01 Sievert).

Another common unit of radiation measurement is the Curie. A Curie is 3.7×10^{10} nuclear disintegrations per second (dps); a common unit of measurement is the picoCurie 3.7×10^{-2} dps or 2.2 disintegrations per minute (dpm). While this unit of measurement reveals nothing about the potential absorbed dose it is easily determined and commonly measured. Water quality standards for the State of California have been promulgated for gross alpha (15 pCi/L) and gross beta (50 pCi/L) activity.

APPENDIX C

Table C-1

Derivation of Risk Equation for Non-VOCs in Water

$$Risk_{water} = SF_o \times C_w \times \frac{IR_{w,adult} \times EF \times ED_{adult}}{BW_{adult} \times AT \times 365\ day/yr}$$

$$+ SF_o \times C_w \times \frac{IR_{w,child} \times EF \times ED_{child}}{BW_{child} \times AT \times 365\ day/yr}$$

$$+ SF_o \times C_w \times \frac{SA_{adult} \times K_p \times EF \times ED_{adult} \times ET_{adult} \times 1\ L/1000\ cm^3}{BW_{child} \times AT \times 365\ days/yr}$$

$$+ SF_o \times C_w \times \frac{SA_{child} \times K_p \times EF \times ED_{child} \times ET_{child} \times 1\ L/1000\ cm^3}{BW_{child} \times AT \times 365\ days/yr}$$

Default exposure factors:

BW	=	body weight (70 kg adult; 15 kg child)
AT	=	averaging time, 70 yr
EF	=	exposure frequency, 350 days/yr
ED	=	exposure duration (24 yr adult; 6 yr child)
IR_w	=	intake rate (adult = 2 L/day; child = 1 L/day)
ET	=	exposure time during showering/bathing (adult, 15 min/shower = 0.25 hr/day; child, four 15 min baths/week = 0.14 hr/day)
SA	=	skin surface area available for contact (adults, 23,000 cm^2; child, 7,200 cm^2)
K_p	=	chemical-specific dermal permeability coefficient from water, cm^2/hr

Reduced Equation:

$$Risk_{water} = (SF_o \times C_w \times 0.0149) + (SF_o \times C_w \times 0.0325 \times K_p)$$

Ref: PEA Manual (DTSC 1994a)

Table C-2

Derivation of Risk Equation for VOCs in Water

$$Risk_{voc,water} = Risk_{water} + \frac{SF_i \times C_w \times IR_{voc,adult} \times EF \times ED_{adult}}{BW_{adult} \times AT \times 365\ days/yr}$$

$$+ \frac{SF_i \times C_w \times IR_{voc,child} \times EF \times ED_{child}}{BW_{child} \times AT \times 365\ day/yr}$$

Default exposure factors:

BW	=	body weight (70 kg adult; 15 kg child)
AT	=	averaging time, 70 yr
EF	=	exposure frequency, 350 days/yr
ED	=	exposure duration (24 yr adult; 6 yr child)
IR_{voc}	=	intake from inhalation of VOCs from domestic use of water is equivalent to the amount of ingested water

Reduced Equation:

$$Risk_{water} = [0.0149 \times ((SF_o \times C_w) + (SF_i \times C_w))] + (SF_o \times C_w \times 0.0325 \times K_p)$$

Ref: PEA Manual (DTSC 1994a)

Table C-3

Derivation of Hazard Equation for Non-VOCs in Water

$$Hazard_w = (1/RfD_o) \times \frac{C_w \times IR_w \times EF \times ED}{BW \times AT \times 365\ day/yr}$$

$$(1/RfD_o) \times \frac{C_w \times SA \times K_o \times ET \times EF \times ED \times (1\ L/1000\ cm^3)}{BW \times AT \times 365\ days/yr}$$

Default exposure factors (for childhood exposure from birth to six years of age):

BW	=	body weight, 15 kg
AT	=	averaging time, 6 yr
EF	=	exposure frequency, 350 days/yr
ED	=	exposure duration, 6 yr
IR_w	=	daily intake of water, 1 L/day
ET	=	exposure time, 0.14 hr/day, based on the assumption of four 15 minute baths taken weekly
SA	=	skin surface area (cm^2) exposed during bathing (child, 7,200 cm^2
K_p	=	chemical-specific dermal permeability coefficient from water, cm^2/hr

Reduced Equation:

$$Hazard_{water} = ((C_w/RfD_o) \times 0.0639) + ((C_w/RfD_o) \times 0.0644 \times K_p)$$

Ref: PEA Manual (DTSC 1994a)

Table C-4

Derivation of Hazard Equation for VOCs in Water

$$Hazard_w = (1/RfD_o) \times \frac{C_w \times IR_w \times EF \times ED}{BW \times AT \times 365\ days/yr}$$

$$(1/RFD_i) \times \frac{C_w \times IR_{w,voc} \times EF \times ED}{BW \times AT \times 365\ days/yr}$$

$$(1/RfD_o) \times \frac{C_w \times SA \times K_p \times ET \times EF \times ED \times 1\ L/1000\ cm^3)}{BW \times AT \times 365\ days/yr}$$

Default exposure factors (for childhood exposure from birth to six years of age):

BW	=	body weight, 15 kg
AT	=	averaging time, 6 yr
EF	=	exposure frequency, 350 days/yr
ED	=	exposure duration, 6 yr
$IR_{w,voc}$	=	intake from inhalation of VOCs ≈ ingestion rate = 1L/day (A chemical is a VOC if it has Henry's Law constant greater than 1 x 10^{-5} atm-m^3/mole and molecular weight less than 200g/mole). The increased intake for VOCs is to account for the additional exposure via inhalation of volatilized compounds from domestic use of water
ET	=	exposure time, 0.14 hrs/day, based on the assumption of four 15 minute baths taken weekly
SA	=	skin surface area (cm^2) exposed during bathing, 7,200 cm^2
K_p	=	chemical-specific dermal permeability coefficient from water, cm^2/hr

Reduced Equation:

$$Hazard_{water} = [0.0639 \times ((C_w/RfD_o) + (C_w/RFD_i))] + [(C_w/RfD_o) \times 0.0644 \times K_p)$$

Ref: PEA Manual (DTSC 1994a), Region IX (USEPA 1995c)

APPENDIX D

LEAD RISK ASSESSMENT SPREADSHEET

CALIFORNIA DEPARTMENT OF TOXIC SUBSTANCES CONTROL

INPUT		OUTPUT					
			percentiles				
MEDIUM	LEVEL		50th	90th	95th	98th	**99th**
LEAD IN AIR (ug/m^3)	0.12						
LEAD IN SOIL (ug/g)	67.0	BLOOD Pb, ADULT (ug/dl)	1.9	3.0	3.4	3.9	**4.3**
LEAD IN WATER (ug/l)	12	BLOOD Pb, CHILD (ug/dl)	3.4	5.3	6.0	6.9	**7.6**
PLANT UPTAKE? 1=YES 0=NO	1	BLOOD Pb, PICA CHILD (ug/dl)	4.2	6.6	7.5	8.7	**9.5**
AIRBORNE DUST (ug/m^3)	72	BLOOD Pb, INDUSTRIAL (ug/dl)	1.7	2.7	3.1	3.6	**3.9**

EXPOSURE PARAMETERS

		residential			industrial
General	units	adults	children	children with pica	adults
Days per week	days/wk	7	4	2	5
Dermal Contact					
Skin area	cm^2	3700	2800	2800	5800
Soil adherence	mg/cm^2	0.5	0.5	0.5	0.5
Route-specific constant	(ug/dl)/(ug/day)	0.00011	0.00011	0.00011	0.00011
Soil ingestion					
Soil ingestion	mg/day	25	55	790	25
Route-specific constant	(ug/dl)/(ug/day)	0.0176	0.0704	0.0704	0.0176
Inhalation					
Breathing rate	m^3/day	20	10	10	20
Route-specific constant	(ug/dl)/(ug/day)	0.082	0.192	0.192	0.082
Water ingestion					
Water ingestion	l/day	1.4	0.4	0.4	1.4
Route-specific constant	(ug/dl)/(ug/day)	0.04	0.16	0.16	0.04

Food ingestion

Food ingestion	kg/day	2.2	1.3	1.3	2.2
Route-specific constant	(ug/dl)/(ug/day)	0.04	0.16	0.16	0.04
Dietary concentration	ug/kg	11.1	11.1	11.1	10.0
Lead in produce	ug/kg	30.2	30.2	30.2	

PATHWAYS, ADULTS

	Residential		Industrial			
Pathway	Blood Pb ug/dl	percent of total	Blood Pb ug/dl	percent of total	Concentration in medium	
SOIL CONTACT:	0.01	1%	0.01	1%	67	ug/g
SOIL INGESTION:	0.03	2%	0.02	1%	67	ug/g
INHALATION:	0.20	11%	0.15	8%	0.12	ug/m^3
WATER INGESTION:	0.67	35%	0.67	39%	12	ug/l
FOOD INGESTION:	0.98	52%	0.88	51%	11.1	ug Pb/kg diet

PATHWAYS,CHILDREN

	Typical		with pica			
Pathway	Blood Pb ug/dl	percent of total	Blood Pb ug/dl	percent of total	concentration in medium	
SOIL CONTACT:	0.01	0%	0.00	0%	67	ug/g
SOIL INGESTION:	0.15	4%	1.06	25%	67	ug/g
INHALATION:	0.14	4%	0.07	3%	0.12	ug/m^3
WATER INGESTION:	0.77	23%	0.77	18%	12	ug/l
FOOD INGESTION:	2.31	69%	2.31	55%	11.1	ug Pb/kg diet

APPENDIX E

Table E-1

Chemical Constituents in the Russian River
Above the Confluence with the Laguna de Santa Rosa

Chemical	Concentration Range (mg/L)	Mean Concentration (mg/L)	Reporting Limit(s) (mg/L)	Number of Detects	Number of Samples
Inorganics					
aluminum	ND - 0.3	0.15	0.2	1	4
ammonia	0.025 - 0.25	0.036	N/A	N/A[1]	131
arsenic	ND	ND	0.005	0	18
asbestos, MFL[2]	27.1 - 5,490	1,438	N/A	4	4
barium	0.06 - 0.08	0.07	N/A	4	4
boron	N/A	N/A	N/A	N/A	N/A
cadmium	ND	ND	0.0005 - 0.005	0	20
calcium	16 - 26	21	N/A	11	11
chromium	ND - 0.036	0.008	0.0005 - 0.02	4	19
copper	ND - 0.019	0.008	0.005 - 0.02	2	20
cyanide	ND	ND	0.005	0	4
fluoride	ND	ND	0.1	0	4
lead	ND - 0.007	0.002	0.002 - 0.007	3	12
magnesium	10-17	13	N/A	11	11
mercury	ND	ND	0.0005 - 0.001	0	18
nickel	ND - 0.051	0.017	0.005 - 0.05	4	12
nitrate	ND - 5.2	0.21	N/A	N/A[1]	137
nitrite	ND - 0.12	0.009	N/A	N/A[1]	128
phosphate	0.005 - 0.66	0.021	N/A	N/A[1]	128
potassium	N/A	N/A	N/A	N/A	N/A
silver	ND - 0.003	0.0014	0.01 - 0.001	1	12
sodium	N/A	N/A	N/A	N/A	N/A
zinc	ND - 0.04	0.015	0.01 - 0.05	4	16

Table E-1

Chemical Constituents in the Russian River
Above the Confluence with the Laguna de Santa Rosa

Chemical	Concentration Range (mg/L)	Mean Concentration (mg/L)	Reporting Limit(s) (mg/L)	Number of Detects	Number of Samples
Organics					
acetone	N/A	N/A	N/A	N/A	N/A
bromomethane	ND	ND	0.0004 - 0.0005	0	5
carbon disulfide	N/A	N/A	N/A	N/A	N/A
chlorobenzene	ND	ND	0.0004 - 0.0005	0	5
chloromethane	ND	ND	0.0004 - 0.0005	0	5
1,4-dichlorobenzene	ND	ND	0.0004 - 0.0005	0	5
ethylbenzene	ND	ND	0.0005 - 0.0006	0	5
methylene chloride	ND	ND	0.0005 - 0.001	0	5
tetrachloroethylene	ND	ND	0.0004 - 0.0005	0	5
toluene	ND	ND	0.0005	0	5
1,1,1-trichloroethane	ND	ND	0.0004 - 0.0005	0	5
xylenes	ND	ND	0.0005 - 0.0006	0	5
Trihalomethanes					
bromodichloromethane	ND	ND	0.0004 - 0.0005	0	5
chloroform	ND	ND	0.0004 - 0.0005	0	5
dibromochloromethane	ND	ND	0.0004 - 0.0005	0	5
total trihalomethanes[3]	N/A	N/A	N/A	N/A	N/A
Phthalates					
di-n-butyl phthalate	ND	ND	0.0005	0	4
bis (2-ethylhexyl) phthalate	ND - 0.0007	0.0004	0.0006	1	4
diethyl phthalate	ND	ND	0.0005	0	4
Pesticides					
aldicarb sulfone	ND	ND	0.0005 - 0.0008	0	4
aldicarb sulfoxide	ND	ND	0.0005	0	4
aldrin	ND	ND	0.00001 - 0.00003	0	12
DCPA (Dacthal)	ND	ND	0.0002	0	4

Table E-1

Chemical Constituents in the Russian River Above the Confluence with the Laguna de Santa Rosa

Chemical	Concentration Range (mg/L)	Mean Concentration (mg/L)	Reporting Limit(s) (mg/L)	Number of Detects	Number of Samples
α-lindane	ND	ND	0.00001 - 0.00003	0	12
γ- lindane	ND	ND	0.00001 - 0.00002	0	5
heptachlor	ND	ND	0.00001 - 0.00005	0	12
Radioactivity					
Gross alpha, GPV[4]	-0.1 - 2.0 pCi/L	1.1 pCi/L	N/A	4	4
Gross beta, GPV	1.0 - 2.7 pCi/L	1.8 pCi/L	N/A	4	4

N/A - not available
N.D. - not detected

(1) Data were obtained from the North Coast Regional Water Quality Control Board. Non detects in the historical data set were reported as one-half of the reporting limit, but were not distinguished from detections of equal value. Therefore, the number of detects could not be determined.

(2) Asbestos values are reported as millions of fibers per liter (MFL).

(3) Trihalomethanes include chloroform, bromoform, bromodichloromethane, and dibromochloromethane. Bromoform was not detected at or above the reporting limit for any sample. One half the reporting limit for bromoform was used to calculate the maximum and mean concentrations of trihalomethanes.

(4) Radioactivity values are reported as greatest probable value (GPV).

APPENDIX F

INACTIVATION OF *GIARDIA* CYSTS BY CHLORINE

This appendix presents a discussion of the requirements for inactivation of *Giardia lamblia* cysts by chlorine in water. Under the Surface Water Treatment Requirements (SWTR) of the National Primary Drinking Water Regulations, all community and non-community public water systems using surface water are required to provide minimum disinfection to control *Giardia lamblia*, enteric viruses, and bacteria (USEPA 1989a). The treatment is required to achieve at least 99.9 percent removal and/or inactivation of *Giardia lamblia* cysts and at least 99.99 percent removal and/or inactivation of viruses (i.e., virus of fecal origin and infectious to humans). Unfiltered systems are required to demonstrate that disinfection alone achieves the minimum performance requirements by monitoring disinfectant residual, disinfectant contact time, pH (if chlorine is used), and water temperature. These data must be applied to determine if their "*Ct*" values [the product of disinfectant concentration (mg/L) and disinfectant contact (minutes)] equals or exceeds the Ct values for *Giardia lamblia* specified in the SWTR. With the exception of chloramines, where ammonia is added prior to chlorine, these *Ct* values are also adequate to achieve greater than 99.99 percent inactivation of viruses (USEPA 1991).

In the Guidance Manual to the SWTR (USEPA 1991), USEPA recommends *Ct* values for different disinfectants to achieve levels of inactivation for unfiltered systems. The destruction of pathogens by chlorination is dependent on a number of factors, including water temperature, pH, disinfectant contact time, degree of mixing, turbidity, presence of interfering substances, and concentration of chlorine available. The pH has a significant effect on inactivation efficiency because it determines the species of chlorine found in solution, each of which has a different inactivation efficiency. The impact of temperature on disinfection efficiency is also significant. Disinfection by chlorination can inactivate *Giardia lamblia* cysts, but only under rigorous conditions. The cysts are among the most resistant pathogens known, disinfection at low temperatures is especially difficult, and treatment processes prior to disinfection are important (Clark and Regli 1991).

The SWTR Guidance manual provides tables of *Ct* values for 99.9 percent inactivation of *Giardia lamblia* by free chlorine at different temperatures and pH values. Data are also available in the literature on the inactivation efficiency of chlorine under various conditions. Clark and Regli (1991) have developed an equation that can be used to predict *Ct* values for the inactivation of *Giardia lamblia* by free chlorine based on the interaction of disinfectant concentration, temperature, pH, and inactivation level (*I*). The parameters for this equation have been derived from a set of animal infectivity and excystation data. The equation can be used to predict *Ct* values for achieving 0.5 to 4 logs (68.37 percent to 99.99 percent) of inactivation, within a temperature range of 0.5 to 5 °C, chlorine concentration ranges up to 4 mg/L, and pH levels of 6 to 8. Ct values above 5 °C can be estimated by using the equation, then applying the assumption that there is a twofold decrease in *Ct* values for every 10 °C increase in temperature. While

the model was not based on pH values above 8, the model is still applicable up to pH level of 9. The equation for the estimated *Ct* values at 0.5 and 5 °C is as follows:

$$Ct = 0.36 pH^{2.69} temp^{-0.15} C^{0.15} (-\log I)^{1.00}$$

where:

Ct	=	concentration in mg/L times time in minutes
pH	=	pH in the water treatment system
temp	=	temperature in °C
C	=	concentration of disinfectant (mg/L)
I	=	level of inactivation

This equation can be used to determine if 99.99 percent (4 log) inactivation of *Giardia lamblia* can be achieved by the wastewater treatment conditions of the Santa Rosa Subregional Wastewater Treatment System. The following conditions are met by the system:

- pH: 6.9
- temperature: 18 - 25 °C (monthly average)
- chlorine concentration: 6.7 mg/L (annual average 1994)
- chlorine contact time: 1.3 to 1.9 hours (monthly average)

Solving the equation using a temperature of 5 °C, a chlorine concentration of 4 mg/L (maximum of applicable range), and pH 6.9, the calculated *Ct* is 251. At 15 °C, the *Ct* is 251/2 or 125. The *Ct* attained by the system is 6.7 mg/L x 78 minutes (1.3 hour) or 522. Therefore the system exceeds the *Ct* requirements for 99.99 percent inactivation of *Giardia lamblia* cysts by greater than a factor of 2.