PUBLIC HEALTH ASSESSMENT

Evaluation of Exposure to Contaminants at the University of California, Berkeley, Richmond Field Station, 1301 South 46th Street

Richmond, Contra Costa County, California

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Prepared by

California Department of Public Health
Under Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

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List of Acronyms

ATSDR—Agency for Toxic Substances and Disease Registry

bgs—below ground surface

CAG—Community Advisory Group

Cal/EPA—California Environmental Protection Agency

CCCHSD—Contra Costa County Health Services Department

CDPH—California Department of Public Health

CSF—cancer slope factor

CHHSL—California Human Health Screening Levels

COCs—contaminants of concern

CREG—Cancer Risk Evaluation Guideline for one in a million excess cancer risk

DTSC—Department of Toxic Substances Control (of Cal/EPA)

EBRPD—East Bay Regional Parks District

EHIB—Environmental Health Investigations Branch

EMEG—Environmental Media Evaluation Guide (ATSDR)

EPA—U.S. Environmental Protection Agency

I.Q.—Intelligence Quotient

LOAEL—Lowest Observable Adverse Effect Level

ml—milliliter

MRL—Minimal Risk Level (ATSDR)

NA—not analyzed or not applicable

ND—not detected

NOAEL—No Observable Adverse Effect Level

NPL—National Priorities List (EPA)

NS—not sampled

NTP—National Toxicology Program

OEHHA—Office of Environmental Health Hazard Assessment (of Cal/EPA)

PCBs—polychlorinated biphenyls

PHA—public health assessment

PM 10—particulate matter that is less than 10 microns in aerodynamic diameter

ppm—parts per million

ppb—parts per billion

PRP—potentially responsible party

RCRA—Resource, Conservation, and Recovery Act

REL—Reference Exposure Level (OEHHA)

RFS—Richmond Field Station

RfC—reference concentration (EPA)

RfD—reference dose (EPA)

RI—remedial investigation

RI/FS—remedial investigation/feasibility study

RMEG—Reference Dose Media Evaluation Guide based on EPA's RfD (ATSDR)

RWQCB—Regional Water Quality Control Board (of Cal/EPA)

UC—University of California

$\mu g/m^3$—micro gram per cubic meter of air

VOC—volatile organic compound

Summary

This public health assessment (PHA) looks at the possible ways people could come into contact with contaminants at the Richmond Field Station (RFS), and responds to workers' health concerns related to the site. The purpose of the PHA is to help determine what follow-up activities are needed to reduce or eliminate exposure.

The PHA has three parts. The first is a review of existing environmental data to evaluate the potential health impact from exposures to contaminants found at the site. The review addresses the following: contamination in the Western Stege Marsh; metal contamination in on-site soils; airborne contaminants generated or released during remedial activities conducted in September 2002 and September 2003; and contaminants in indoor air. Second, the PHA describes health concerns collected from on-site workers and former workers. Third, the PHA evaluates these health concerns based on environmental data review described above, the health effects known to be associated with certain chemicals found on-site, and what is known about the cause of the health effects/concerns expressed by RFS workers.

RFS is operated by the University of California (UC), Berkeley, in Richmond, California. The RFS site is located at 1301 South 46th Street, Richmond, California. UC purchased the land in 1950. RFS is currently used as a research and teaching facility.

Between 1870 and 1950, much of RFS property belonged to the California Cap Company, which made explosives. The California Cap Company manufactured mercury fulminate on-site for the production of blasting caps. This resulted in mercury contamination to the soil and marsh sediments.

From 1897 to 1985, the adjacent property directly east, was owned and operated by Stauffer Chemical Company (later known as Zeneca). Stauffer produced/manufactured sulfuric acid, superphosphate fertilizer, pesticides, herbicides, and other chemicals. The production of sulfuric acid generated pyrite cinder wastes that were deposited on RFS (prior to 1950) and the Zeneca property. The pyrite cinders are a source of low pH conditions and metals including arsenic, cadmium, copper, lead, mercury, selenium, and zinc. Naturally-occurring radionuclides are associated with the production of superphosphate fertilizer and may also be elevated in soil, sediment, and groundwater on the RFS site. Other historic activities conducted on the Zeneca property involving radionuclides may also be present in soil, sediment, and groundwater. Zeneca is currently undergoing investigation and clean-up activities. At the time of this writing, radionuclides associated with Stauffer activities have not been characterized at the Zeneca site or the RFS.

From 1999 to 2005, investigations and clean-up activities were underway at RFS under the oversight of the California Regional Water Quality Control Board (RWQCB), San Francisco Bay Region. In May 2005, the California Environmental Protection Agency's Department of Toxic Substances Control (DTSC) took over as the lead oversight agency for RFS.

In April 2005, due to ongoing community concerns about the RFS, the Department of Toxic Substances Control (DTSC) and the Contra Costa County Health Services Department requested

assistance from the California Department of Public Health (CDPH) (formerly California Department of Health Services) to evaluate the potential health impact posed by the site. Since that time, CDPH has been conducting PHA activities at RFS.

CDPH evaluated the possible exposure pathway/activities (past, current, and future) to contaminants at RFS, using environmental data collected from the site. On the basis of available data, CDPH concludes past exposure to airborne mercury during remedial activities conducted between August 2003 and September 2003 did not pose a public health hazard.

CDPH concludes the following exposure pathways/activities pose an indeterminate public health hazard, due to a lack of data:

- current and future exposure to adults or youth from restoring the Western Stege Marsh in areas that have been excavated;

The available data do not indicate that people engaging in restoration activities are being exposed to levels of metals, pesticides or PCBs in the West Stege Marsh that would cause adverse health effects. However, there is a potential for elevated levels of natural occurring radionuclides associated with historic operations at the adjacent Zeneca site to have migrated into the West Stege Marsh. In addition, groundwater monitoring is needed to address the concern whether other site-related contaminants from the adjacent Zeneca site are migrating into the West Stege Marsh. Until these activities are completed, and a determination is made whether there is a need for further characterization of the West Stege Marsh, these pathways are classified as posing an indeterminate health hazard. Access to the marsh should remain restricted.

- current and future exposure to contaminants in indoor air as a result of vapor intrusion.

Limited indoor air sampling indicates a potential health risk from exposure to formaldehyde in indoor air that occurred between September 2005 and October 2005. These data are insufficient to draw conclusions about the source of formaldehyde in indoor air or the potential impact of future exposure.

CDPH concludes the following exposure pathways/activities pose a public health hazard:

- past, current, and future exposure to children/teenagers who regularly played/play in the West Stege Marsh;

CDPH identified a public health hazard for children/teenagers who regularly played/play in the West Stege Marsh, from exposure to metals and PCBs in surface water and/or sediment. The most sensitive (primary) noncancer endpoints associated with COCs include skin effects (arsenic), renal effects (cadmium), neurodevelopmental (methylmercury), gastrointestinal symptoms (copper), immune effects (PCBs), and decreases in erythrocyte copper, zinc-superoxide dismutase (ESOD) activity (zinc). COCs associated with an increased cancer risk are arsenic (skin, liver, bladder, and lung) and PCBs (liver, biliary). It is important to note that this conclusion is based on conservative assumptions meant to identify the possibility for exposures of health concern, so that steps can be taken to mitigate or prevent these exposures from

occurring. Actual exposures to children/teenagers are likely much less. Access to the marsh should remain restricted.

- past, current, and future exposure to RFS maintenance workers who regularly work in soil containing the highest levels of metals and PCBs in non-excavated areas of RFS.

CDPH identified a public health hazard for RFS maintenance workers who regularly work in soil containing the highest levels of metals and PCBs in non-excavated areas of RFS. The primary noncancer endpoints associated with COCs include skin effects (arsenic), immune changes (PCBs), renal effects (cadmium, inorganic mercury), and gastrointestinal symptoms (copper). COCs associated with an increased cancer risk are arsenic (skin, liver, bladder, and lung) and PCBs (liver, biliary). While this conclusion is based on conservative assumptions (actual ingestion and dermal exposure are likely much less), it does not include potential exposure from inhalation of contaminated soil particulates, which could be a significant route of exposure, adding to the worker's overall risk. Inhalation exposure can be mitigated if workers wear proper respiratory protection while working in RFS soil.

CDPH made efforts to collect and understand the health concerns that RFS workers believe are related to contamination at RFS. In the PHA, CDPH responds to these concerns by stating whether contaminants are associated with the health concern expressed, and whether these are present at levels where health effects have been seen in the scientific literature. The majority of the health concerns expressed by workers cannot be linked to chemical exposures at the site, based on the exposure and toxicological information available. Two exceptions are irritation of the eyes, nose, and throat, and mild respiratory effects that may have occurred from exposure to formaldehyde and airborne dust.

On the basis of these findings, CDPH and the federal Agency for Toxic Substances and Disease Registry recommend the following.

Site Characterization

- UC should conduct additional characterization of on-site groundwater at the east and northeast side of RFS, to better understand the potential for vapor intrusion to be affecting indoor air in buildings in that area.
- UC should conduct additional indoor air sampling in Buildings 163 and 175 to identify whether formaldehyde is elevated above levels typical of indoor air. Results of sampling will determine the need for further sampling or investigation.
- UC should analyze for radionuclides associated with historic activities at the Zeneca site (former Stauffer Chemical) in on-site upland soil and groundwater, and sediment from the West Stege Marsh, if radionuclide contamination is identified during investigations at the Zeneca site.
- UC should conduct additional characterization of on-site soil throughout RFS to identify other areas where potential contamination may exist. Chemicals used in research activities at RFS, as well as known contaminants from historic uses of RFS and Zeneca-related (former Stauffer Chemical) contaminants should be analyzed.

- Additional characterization of soil in the area where the Forest Products Laboratory is located is needed, and should include analyses of pentachlorophenol and chlorophenol byproducts.
- UC should provide all of RFS staff access to up to date maps showing locations of current and historic structures and soil sampling locations, along with the associated level of contamination.

Environmental Monitoring

- UC should annually sample sediment and unfiltered water in the marsh to identify whether contaminants are migrating from the non-remediated areas of the marsh, the uplands, and Zeneca site.
- Future soil disturbing/dust generating activities should be monitored for air quality along the perimeter of the site to ensure safe air quality for workers, residents, and other people in the area.

Training

- UC should offer Hazardous Waste Operations and Emergency Response training to workers whose work may involve handling or digging in soils on the RFS site.
- UC should train workers annually in how to identify cinders and what action to take if such material is identified.

Note: The Environmental Health Investigations Branch (EHIB), within CDPH, under a cooperative agreement with the federal Agency for Toxic Substance and Disease Registry (ATSDR), conducted this PHA of UC Richmond Field Station. In 2007, CDPH/ATSDR will release a PHA for the adjacent Zeneca site—that contains exposure information that may be applicable to RFS workers.

Background and Statement of Issues

The Environmental Health Investigations Branch (EHIB), within the California Department of Public Health (CDPH) (formerly the California Department of Health Services], under cooperative agreement with the federal Agency for Toxic Substance and Disease Registry (ATSDR), is conducting a public health assessment (PHA) of the Richmond Field Station (RFS), operated by the University of California (UC) in Richmond, California. The PHA will include a review of existing environmental data to evaluate the potential health impact from exposures to site-related contaminants, a collection of exposure and health concerns, and a response to these concerns based on review of the data. The PHA is an evaluation of the site to help determine what follow-up activities are needed: additional site characterization, health education, health study, or specific measures to reduce or eliminate exposure. Specifically, we will address the following exposure pathways (situations): contamination in the RFS marsh; metal contamination in on-site soils; airborne contaminants generated/released during remedial activities conducted in September 2002 and September 2003; and contaminants in indoor air. CDPH will be releasing a PHA for the adjacent Zeneca site—its current owners are Cherokee Simeon Ventures—that contains exposure information that may be applicable to RFS workers.

The RFS site is located at 1301 South 46th Street, Richmond, California. In 1950, UC Berkeley purchased the land known as RFS (Appendix B, Figure 1). The property is located along the Richmond shoreline and consists of tidal mudflats, marsh, grasslands, and the upland areas where most of the facilities/buildings are located. RFS is currently used as a research and teaching facility. The Northern Regional Library of the UC Office of the President and the U.S. Environmental Protection Agency (EPA)'s Regional Laboratory are also located at RFS.

Between 1870 and 1950, much of RFS property belonged to the California Cap Company, an explosives manufacturer. The California Cap Company manufactured mercury fulminate on-site for the production of blasting caps. Operations at the California Cap Company resulted in mercury contamination to the soil and marsh sediments (1).

From 1897 to 1985, the adjacent property directly east was owned and operated by Stauffer Chemical Company (later known as Zeneca). Stauffer produced/manufactured sulfuric acid, superphosphate fertilizer, pesticides, herbicides, and other chemicals. The production of sulfuric acid generated pyrite cinder wastes that were deposited on RFS (prior to 1950) and the Zeneca property. The pyrite cinders are a source of low pH conditions and metals including arsenic, cadmium, copper, lead, mercury, selenium, and zinc. Naturally-occurring radionuclides associated with the production of superphosphate fertilizer may also be elevated in soil, sediment, and groundwater on the RFS site. Other historic activities conducted on the Zeneca property involving radionuclides may also be present in soil, sediment, and groundwater. Zeneca is currently undergoing investigation and clean-up activities. At the time of this writing, radionuclides associated with Stauffer activities have not been characterized at the Zeneca site or the RFS.

In 1999, the California Regional Water Quality Control Board (RWQCB), San Francisco Bay Region, identified contamination (metals and low pH conditions) in sediments from the Western Stege Marsh (Appendix B, Figure 1). As a result, RWQCB requested that UC investigate the

extent of contamination in the marsh and the southern portion of the upland area. Elevated concentrations of polychlorinated biphenyls (PCBs) were also found in the sediment in and adjacent to Meeker Slough located along the western boundary of Western Stege Marsh. The source of PCB contamination is still under investigation.

Since 1999, investigations and clean-up activities have been underway at RFS (1). Clean-up activities include restoring the native marsh and creating additional marsh habitat. Three phases of excavation and removal of contaminated material from RFS have occurred.

- Phase 1. From August 2002 to January 2003, 28,000 cubic yards of contaminated soil (pyrite cinder waste and mercury) and marsh sediment were removed from an area bordered by Zeneca to the east and East Bay Regional Park Bay Trail to the south (Appendix B, Figure 2).

- Phase 2. From August 2003 to March 2004, 31,000 cubic yards of contaminated material (pyrite cinder waste and mercury-contaminated sediment) were removed. PCBs were also removed from an area at the outfall of a storm drain in Meeker Slough (Appendix B, Figure 2).

- Phase 3. From August 2004 to November 2004, 3,300 cubic yards of soil contaminated with metals and PCBs were removed from the upland areas.

Clean-up work is prohibited during the months of February through August, due to the presence in the marsh of the endangered California Clapper Rail.

In April 2005, due to ongoing community concerns about RFS, the Contra Costa County Health Services Department and the California Environmental Protection Agency's Department of Toxic Substances Control (DTSC) requested assistance from CDPH to evaluate the potential health impact posed by the facility. Since that time, CDPH has been conducting PHA activities at RFS. In May 2005, DTSC formally became the lead regulatory agency overseeing environmental investigations and cleanup at the site.

Land Use

RFS occupies approximately 150 acres in a primarily industrial area. The property is comprised of upland areas and offshore areas. The offshore area consists of an inner and outer portion of the Western Stege Marsh (Appendix B, Figure 1). The outer portion of the Western Stege Marsh is located south of the East Bay Regional Parks District (EBRPD) Bay Trail and includes approximately 60 acres of tidal mud flat, marsh, and open water; this portion of the RFS property is not been evaluated in this report. The upland area is located north of the Western Stege Marsh and occupies approximately 90 acres (1). Interstate 580 bounds RFS to the north.

The Richmond Redevelopment Agency owns the property on the western shore and most of Meeker Slough. The nearest residential area, Marina Bay, is located to the west of RFS. RFS is bounded to the east by the Zeneca property (Appendix B, Figure 1). Adjacent to the Zeneca property, to the east, are a number of small businesses.

There are a number of other contaminated sites in the area: Zeneca (formerly Stauffer Chemical Company), Liquid Gold Oil Corporation, Bio-Rad Laboratories, Marina Bay Project, Blair Landfill, and Stege Property Pistol Range.

Demographics

Approximately 400 people work in different departments at RFS, consisting of academics, researchers, laboratory staff, students, maintenance workers, security staff, and administrative staff. Approximately 50 people work at the EPA laboratory.

Environmental Contamination/Pathway Analysis/Toxicological Evaluation

This section examines the pathways for exposure to contamination from the RFS site. We will examine each of the media (groundwater, sediment, surface water in Western Stege Marsh, soil, and air) to determine whether or not contamination is present and if people in the community or at RFS are exposed to (or in contact with) the contamination. If people are exposed to contamination in any of the media, we will evaluate whether there is enough exposure to pose a public health hazard. This analysis will systematically evaluate each of the media. Table 1 in Appendix C presents a summary of the exposure pathways identified at this site.

Exposure pathways are means by which people in areas surrounding the sites could have been or could be exposed to contaminants from the site. For target populations to be exposed to environmental contamination, there must be a mechanism by which the contamination comes into direct contact with a human population. This is called an exposure pathway. Exposure pathways are classified as either completed, potential, or eliminated (2).

In order for an exposure pathway to be considered completed, the following five elements must be present: a source of contamination, an environmental medium and transport mechanism, a point of exposure, a route of exposure, and a receptor population. For a population to be exposed to an environmental contaminant, a completed exposure pathway (all five elements) must be present (2). The following is an example of a completed exposure pathway: a contaminant from a hazardous waste site (source) is released to the air (medium-transport mechanism); the wind blows the contaminant through air into the community (point of exposure) where community members breathe the air (route of exposure and receptor population) (Appendix C, Table 1).

Potential exposure pathways are either 1) not currently complete but could become complete in the future, or 2) indeterminate due to a lack of information. Pathways are eliminated from further assessment if one or more elements are missing and are never likely to exist.

Description of Toxicological Evaluation

In a toxicological evaluation, we evaluate the exposures that have occurred to site-related contaminants, based on the most current studies we can find in the scientific literature. There is not enough available information to thoroughly evaluate exposure to multiple chemicals or possible cancer and noncancer adverse effects of exposure to very low levels of contaminants

over long periods of time. Some introductory information follows to help clarify how we evaluate the possible health effects that may occur from exposure to the contaminants identified for follow-up.

When individuals are exposed to a hazardous substance, several factors determine whether harmful effects will occur and the type and severity of those health effects. These factors include the dose (how much), the duration (how long), the route by which they are exposed (breathing, eating, drinking, or skin contact), the other contaminants to which they may be exposed, and their individual characteristics such as age, sex, nutrition, family traits, lifestyle, and state of health. The scientific discipline that evaluates these factors and the potential for a chemical exposure to adversely impact health is called toxicology.

Environmental and Health Screening Criteria

The following section briefly discusses the method used to identify contaminants of concern (COCs) for further evaluation and to determine whether levels of contaminants in various environmental media pose a health hazard from adverse noncancer or cancer health effects.

As a preliminary step in assessing the potential health risks associated with contaminants at the RFS site, CDPH compared contaminant concentrations to media-specific environmental guideline comparison values (CVs). Those concentrations that exceed the CV are identified as COCs for further evaluation of potential health effects. ATSDR's comparison values are media-specific concentrations that are estimates of a daily human exposure to a contaminant that is unlikely to cause cancer or noncancer (health effects other than cancer) adverse health effects. The following CVs were applied in the current evaluation:

- Cancer Risk Evaluation Guide (CREG). CREGs are media-specific comparison values used to identify concentrations of cancer-causing substances that are unlikely to result in an increase of cancer rates in a population exposed over an entire lifetime. CREGs are derived from EPA's cancer slope factors, which indicate the relative potency of cancer-causing chemicals. Not all chemicals have a CREG (3).

- Environmental Media Evaluation Guide (EMEG). EMEGs are estimates of chemical concentrations that are not likely to cause an appreciable risk of deleterious, noncancer health effects for fixed durations of exposure. EMEGs might reflect several different types of exposure: acute (1-14 days), intermediate (15-364 days), and chronic (365 or more days). EMEGs are based on ATSDR's Minimal Risk Levels (MRLs) (see Glossary in Appendix A for a more complete description of EMEGs) (3, 4).

- Reference Dose Media Evaluation Guides (RMEGs). RMEGs are estimates of chemical concentrations that are not likely to cause an appreciable risk of deleterious, noncancer health effects for chronic exposure. RMEGs are based on EPA's References Doses (RfDs) (see Glossary in Appendix A for a more complete description of EMEGs) (5).

- California Human Health Screening Levels (CHHSLs). CHHSLs are screening levels for chemicals in soil and soil gas used to aid in clean-up decisions based on the protection of public health and safety (6).

- Reference Exposure Levels (RELs) and Reference Concentrations (RfCs). RELs and RfCs are estimates of chemical concentrations in air that are not likely to cause an appreciable risk of deleterious, noncancer health effects for fixed durations of exposure. The California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, RELs and EPA RfCs are used to evaluate inhalation exposure (7).

- Preliminary Remediation Goals (PRGs). EPA's Preliminary Remedial Goals (PRGs) are risk-based concentrations used in initial screening-level evaluations of environmental measurements. PRGs are used if there is no EMEG or RMEG available (8).

If a contaminant is not found at levels greater than its comparison/screening value, CDPH concludes the levels of corresponding contamination are not likely to cause illness and no further evaluation is conducted.

If a contaminant in soil or water is found at levels greater than its comparison value, CDPH designates the contaminant as a COC, and exposure doses are calculated. These values (exposure dose estimates) are then used to examine the potential human exposures in greater detail. CDPH uses the following health-based comparison values (or health guidelines) to identify those contaminants that have the possibility of causing noncancer adverse health effects (cancer health effects evaluation discussed later).

- Minimal Risk Level (MRL). MRLs are estimates of daily human exposure to a substance that is likely to be without an appreciable risk of adverse, noncancer health effects over a specified duration of exposure. MRLs are based on the NOAEL (No Observed Adverse Effect Level) or the LOAEL (Lowest Observed Adverse Effect Level) (see Glossary in Appendix A for description of NOAEL and LOAEL).

- Reference Dose (RfD). RfDs are estimates of daily human exposure to a substance that is likely to be without an appreciable risk of adverse, noncancer health effects over a specified duration of exposure. RFDs are based on the NOAEL or the LOAEL.

The toxicity studies used to determine the various health comparison values are usually conducted on adult animals or adult humans, mostly worker populations. In an effort to be protective of sensitive populations such as children, an uncertainty factor is included in the derivation of health comparison values.

COCs that exceed health comparison values are evaluated on an individual basis, relative to the concentrations shown to cause health effects. In situations when multiple COCs are present and none of the contaminants individually exceed their respective health comparison value, it is possible that exposure to multiple contaminants (chemical mixtures) may pose a noncancer health risk. Chemicals can interact in the body resulting in effects that might be additive, greater than additive, or less than additive. If additive, the dose of each chemical would have an equal

weight in its ability to cause harmful effects. In that case, the combined dose for the two chemicals is an indication of the degree to which possible harmful effects could occur in people. When the chemicals act in a greater than additive manner, which is known as synergism, one chemical is enhancing the effect of the other chemical. In that case, the combined dose for the two chemicals underestimates the potential toxicity of the mixture of two chemicals. For chemicals that act in a less than additive manner, which is known as an antagonistic effect, the combined dose overestimates the potential toxicity of the mixture of two chemicals.

Currently, the accepted methodology for evaluating noncancer exposure to chemical mixtures is by looking at the additive effect. For contaminants that do not exceed health comparison values, CDPH evaluated the additive effect of exposure to these contaminants by estimating the hazard index for those contaminants. If the hazard index is above 1, then exposure may pose a noncancer health risk and the mixture is evaluated further.

Cancer health effects are evaluated in terms of a possible increased cancer risk. Cancer risk is the theoretical chance of getting cancer. In California, 41.5% of women and 45.4% of men will be diagnosed with cancer in their lifetime (about 43% combined) (9). This is referred to as the background cancer risk. We say “excess cancer risk” to represent the risk above and beyond the background cancer risk. If we say that there is a “one-in-a-million” excess cancer risk from a given exposure to a contaminant, we mean that if one million people are chronically exposed to a carcinogen at a certain level over a lifetime, then one cancer above the background risk may appear in those million persons from that particular exposure. For example, in a million people, it is expected that approximately 430,000 individuals will be diagnosed with cancer from a variety of causes. If the entire population was exposed to the carcinogen at a level associated with a one-in-a-million cancer risk, 430,001 people may get cancer, instead of the expected 430,000.

Cancer risk numbers are a quantitative or numerical way to describe a biological process (development of cancer). This approach uses a mathematical formula to predict an estimated number of additional cancers that could occur due to the exposure modeled. The model is based on the assumption that there are no absolutely safe toxicity values for chemicals that can cause cancer, meaning that the model assumes that no matter how low, even for extremely low exposures, there is always the possibility that a true carcinogen could cause a cancer. The models typically use information from higher exposure scenarios and then extend an estimate of risk into lower exposure scenarios using the assumption that lower levels would still be carcinogenic. The calculations take into account the level of exposure, frequency of exposure, length of exposure to a particular carcinogen, and an estimate of the carcinogen’s potency.

EPA and OEHHA have developed cancer slope factors and unit risk values for many carcinogens. A slope factor/unit risk is an estimate of a chemical's carcinogenic potency, or potential, for causing cancer. Unit risk values or cancer slope factors are created from studies of persons (workers) or animals to see how much illness developed as a result of exposure. In order to take into account the uncertainties in the science (such as making predictions of health outcomes at lower levels when we only have information about high exposures), the risk numbers used are plausible upper limits of the actual risk, based on conservative assumptions. In other words, the theoretical cancer risk estimates are designed to express the highest risk that is plausible for the particular exposure situation, rather than aiming to estimate what is the most

likely risk. Given that there is uncertainty to these predictions, it is considered preferable to overestimate, rather than underestimate risk. If adequate information about the level of exposure, frequency of exposure, and length of exposure to a particular carcinogen is available, an estimate of the theoretical increased cancer risk associated with the exposure can be calculated using the cancer slope factor or unit risk for that carcinogen. Specifically, to obtain lifetime risk estimates from inhalation exposure, the contaminant concentration is multiplied by the unit risk for that carcinogen. To obtain lifetime risk estimates for other pathways, a chronic exposure dose is estimated, that is then multiplied by the slope factor for that carcinogen.

Cancer risk estimates are a tool to help determine if further action is needed and they should not be interpreted as an accurate prediction of the exact number of cancer cases that actually occur. The actual risk is unknown and may be as low as zero (10).

CDPH evaluated five completed pathways of exposure related to the RFS site (Appendix C, Table 1). Data are presented in tables in Appendix C. In the following pages, we describe our evaluation of these pathways. A brief summary of the toxicological characteristics of the COCs identified by CDPH is presented in Appendix D. Additional information on COCs is also provided in the Evaluation of Community Concerns section. The toxicological evaluation of the completed exposure pathways involves the use of exposure assumptions. The authors used conservative estimates and assumptions to ensure potential health hazards from chemicals are identified and evaluated.

Evaluation of Richmond Field Station Marsh Sediments and Surface Water

The RFS marsh/lagoon area is accessible by the Marina Bay trail, the connector trails from the Marina Bay residential neighborhood, and from RFS via a locked fence. There are many anecdotes about kids and sometimes adults going off the trail and playing in the water and mud. From the early 1990s, it has been known that the marsh area contained contamination from the former California Cap Company and from other nearby sources. In 2003, UC consultants conducted two remediation activities in the marsh area: 31,000 cubic yards of pyrite cinder waste and mercury-contaminated sediment were removed from an area of known PCB contamination, and 28,000 cubic yards of sediment and fill were removed from the marsh area closest to the RFS site (Appendix B, Figure 3). Fill from other parts of RFS, as well as sediments and soils from other locations, were brought in to fill the excavated area. The fill was sampled according to regulatory guidelines to show that it was clean enough to be used for fill. Children and adults have engaged in restoration of the remediated area, planting wetland grasses.

Surface and subsurface sediment samples taken in the non-remediated marsh area of RFS have elevated arsenic, cadmium, copper, lead, mercury, zinc, and total PCBs (surface sampling data shown in Table 2, Appendix C) (11). Many of these metals are naturally occurring in the environment, which contributes to the overall concentration. Recent surface soil/sediment data in the remediated area show low levels of PCBs and elevated levels of some metals (arsenic, cadmium, copper, lead, mercury, and zinc), perhaps indicating that chemicals may be migrating from the non-excavated areas as a result of the changing water levels in the marsh area (11, 12).

The most recent (2006) filtered surface water samples show low levels of the same chemicals, while unfiltered surface water data from the early 1990s show elevated levels of arsenic, cadmium, copper, chromium, and zinc (Appendix C, Table 3) (13, 14). These contaminants exceed comparison values and will be evaluated further.

Historic Exposure to Adults and Children/Teenagers Playing in the Marsh Prior to 2003 (Phase 1 and Phase 2 Excavations/Removals)

CDPH evaluated historic exposure to an adult and child/teenager (8-15 years of age; old enough to play unattended) playing in the Phase 1 and Phase 2 areas of the marsh, prior to remedial activities. We evaluated historic exposure to surface water in the marsh using data (unfiltered samples) collected in 1991. The amount of exposure a person might have received from playing in the marsh depends on how often that person might have come near to or in contact with the marsh. Exposure also depends on the types of play and activity, i.e., splashing, wading, etc. If an adult or child/teenager played in the marsh and splashed in the water, they may have absorbed contaminants through the skin, or accidentally/incidentally ingested some of the chemicals in the sediment and surface water. To estimate exposure we assumed an individual engaged in activities in the marsh during the warmer months (May to October), 4 days per week (100 days per year), for an hour at a time. We assumed the adults may have been exposed for the past 26 years and children for 10 years (15).

CDPH estimated the exposure dose from ingestion and dermal contact (touching) to children/teenager and adults from surface water and sediment in the marsh, prior to remediation. CDPH used the average concentration of contaminants in sediment and surface water from the Phase 1 and Phase 2 areas to estimate historic exposure (Appendix C, Tables 3 and 4). The other assumptions used in the dose estimations are shown in the footnotes to Tables 5 and 7 in Appendix C (15). It is important to note that the estimated exposure doses from surface water are very uncertain for a number of reasons: surface water data is limited; laboratory methods are not consistent between sampling events and; contaminant concentrations in surface water are not static due to the tidal influences and seasonal changes.

Prior to 2003, when remedial/removal activities occurred in the marsh, CDPH determined that an adult or child/teenager who engaged in activities in the marsh on a regular basis, would not have experienced noncancer health effects from exposure to individual COCs in sediment and surface water. Estimated exposure doses are below health comparison values for individual contaminants (Appendix C, Table 5).

The estimated hazard index for an adult from exposure to multiple contaminants/COCs (metals and PCBs) in sediment and surface water prior to 2003 is estimated at 0.5 (Appendix C, Table 6). Since the estimated hazard index is below 1.0, noncancer adverse health effects are not likely to have occurred or be occurring to adults from exposure to contaminants in sediment and surface water in the marsh.

The hazard index (1.6) for a child/teen from exposure to surface water exceeds 1.0, indicating the possibility for noncancer health effects (Appendix C, Table 6). (It is important to re-emphasize that the hazard index is based on surface water data from one sampling event, which makes this

analysis highly uncertainty.) Whenever the hazard index for a mixture of chemicals exceeds 1.0, exposures are evaluated further. The additional evaluation requires that the most sensitive health endpoint/organ system be determined or each chemical (16). For instance, when two chemicals both cause adverse effects to the liver, a liver target toxicity dose is derived for each chemical, added together and compared to the NOAEL or the LOAEL. As the estimated exposure doses approach the LOAEL for an organ system or endpoint the likelihood of specific adverse effects increases.

The most sensitive (primary) noncancer endpoints associated with COCs include skin effects (arsenic), neurodevelopmental (methylmercury[1]), gastrointestinal symptoms (copper), decreases in erythrocyte copper, zinc-superoxide dismutase (ESOD) activity (zinc), and immune effects (PCBs). Since the primary noncancer endpoints for COCs differ, target toxicity doses were not calculated. These COCs would not have an additive effect on the target organ, as these chemicals affect different organ systems at the lowest dose. There could be some additive effects from these chemicals through a mechanism not involving the target organ; however, that is not known at this time.

Lead is evaluated based on an internal dose, a blood lead level (BLL) that takes into account total exposure (includes exposure to background sources of lead). Young children (under 2 years old) are the most sensitive to lead exposure. The Centers for Disease Control and Prevention recommended action level for lead exposure in children is 10 micrograms per deciliter (µg/dL). Although children are at greatest risk from lead exposure, adult exposures can also result in harmful health effects. Most adult exposures are occupational and occur in lead-related industries such as lead smelting, refining, and manufacturing industries. The U.S. Department of Health and Human Services recommends that BLLs among all adults be below 25 µg/dL (17). The CDPH, Childhood Lead Poisoning Prevention Branch, recommends exposure reduction/mitigation actions for pregnant women with BBLs of 10 µg/dL or greater (18).

CDPH used the DTSC Lead Risk Assessment Spreadsheet (LeadSpread 7) to estimate BLL for adults. LeadSpread estimates BLL for children under the age of 2[2]. The exposure scenario being evaluated for this exposure pathway is for children 8-15 years old.

The estimated BLL for adults from exposure to the average level of lead of 156.1 parts per million (ppm) in the marsh (prior to remediation) is 3.1 µg/dL (95th percentile); exposure to the highest level of lead of 560 ppm would result in an estimated BLL for adults of 5.3 µg/dL. These values include exposure to background sources of lead, such as ambient air, water, and produce. This level is below 10 µg/dL for pregnant women and 25 µg/dL for all other adults, the levels at which exposure reduction actions are recommended (17, 18).

[1]The form of mercury present in sediment and soil at the RFS has not been analyzed. Comparisons to methylmercury were used based on the potential for methylization of mercury in sediments and surface water.
[2]As a point of reference, exposure to the highest level of lead (560 ppm) in the non-remediated area of the marsh would result in an estimated BLL for a 1-2 year old child of 10.2 µg/dL; the adult BLL is 5.3 µg/dL. It is reasonable to assume that the BLL for a child between 8-15 years old would fall between these two numbers, and below 10 µg/dL.

CDPH estimated the theoretical increased cancer risk from historic exposure to contaminants considered carcinogenic. Potentially carcinogenic contaminants exceeding health comparison values in surface water and/or sediment are arsenic and PCBs (Appendix C, Tables 2 and 3). The estimated cancer risk for adults and children/teenager is 2 in 100,000 and 1 in 10,000, respectively. Increased cancer risks in this range (1 in 10,000) are considered to be the upper-end of what is considered an acceptable risk (10).

Current and Future Exposure to Adults and Children/Teenagers Playing in the Marsh

CDPH evaluated current and future exposure to an adult and child/teenager to sediment and surface water in the marsh. The highest (maximum) concentration of contaminants remaining in the marsh was used to evaluate exposure (Appendix C, Tables 2 and 3). CDPH used the maximum concentration in order to identify whether there is a potential health risk under the worst-case scenario, requiring a need for further action. Actual exposures would be much less because an individual would not likely engage in activity in a single area of the marsh for the amount of time assumed (26 years for adults and 10 years for child/teenager) in the exposure dose estimates.

The estimated doses from dermal and ingestion exposure for an adult, are below levels that could result in noncancer adverse health. None of the contaminants in surface water and sediment exceed health comparison values and the hazard index does not exceed 1.0 (Appendix C, Tables 5 and 6).

The estimated dose (0.00005 mg PCBs/kg/day) for a child/teen from dermal and ingestion exposure to PCBs in sediment exceeds health comparison values, suggesting the noncancer health effects (Appendix C, Table 6). However, the estimated doses are below the LOAEL (Lowest Observed Adverse Effect Level) of 0.005 mg PCBs/kg/day shown to cause immune effects (decreased antibody response) in monkeys (19, 20). Since dose estimates are below LOAEL and estimated doses are based on exposure to the maximum concentration of PCBs found in sediment (actual exposures are probably much less), it is possible, but not probable that a child/teen would have experienced health effects from exposure PCBs in sediment. None of the other contaminants (metals) individually exceed their respective health comparison value.

The estimated hazard index (3.1) for a child/teen from exposure to COCs in sediment exceeds 1.0, indicating the possibility for noncancer health effects. Current toxicity information indicates that different parts of the body (organs) are affected by the lowest dose of each of the chemicals. The most sensitive (primary) noncaner endpoints associated with COCs include skin effects (arsenic), renal effects (cadmium), nerodevelopmental (methylmercury), gastrointestinal symptoms (copper), decreases in erythrocyte copper, zinc-superoxide dismutase (ESOD) activity (zinc), and immune effects (PCBs).

The estimated BLL for adults from exposure to the highest level of lead (410 ppm) in the marsh (after remediation), as well as other sources of lead in their life, is 4.5 µg/dL (95^{th} percentile). This level is below 10 µg/dL for pregnant women and 25 µg/dL for all other adults, the levels at which exposure reduction actions are recommended (17, 18).

CDPH estimated the theoretical increased cancer risk from current/future exposure to contaminants considered carcinogenic. Carcinogenic contaminants exceeding comparison values in surface water and sediment are arsenic and PCBs. The estimated cancer risk for adults and child/teenager is 3 in 100,000 and 5 in 100,000, respectively. Cancer risks in this range are considered a "very low increased risk."

Adults or Children/Teenagers Restoring the Excavated Areas of the Richmond Field Station Marsh

CDPH evaluated exposure to an adult and child/teenager (old enough to be part of a restoration project) planting or otherwise working on a restoration project in the excavated area (remediated) of the RFS marsh. They may have been exposed via absorption through the skin or accidentally/incidentally ingested some of the chemicals in the soil/sediment and surface water. To estimate exposure, we assumed the person engaged in some type of activity in the marsh for 2.6 hours per day, 100 days per year, for 8 years and are exposed to maximum level of contamination in the marsh. CDPH used the maximum concentration of the contaminant in sediment and surface water in order to identify whether there is a potential health risk under the worst-case scenario, requiring a need for further action. Actual exposures would be much less because an individual would not engage in activity in a single area of the marsh for the amount of time assumed for the dose estimates.

If an adult or child/teenager worked in the marsh on a regular basis in soil/sediment that contains the maximum concentration of chemicals found in the excavated area, this person should not experience noncancer adverse health effects (Appendix C, Tables 2 and 7). None of the contaminants exceed health comparison values and the hazard index does not exceed 1.0.

The estimated BLL for an adult from exposure to the highest level of lead of 410 ppm remaining in the marsh, as well as other sources of lead in their life, is 4.5 µg/dL. This level is below 10 µg/dL for pregnant women and 25 µg/dL for all other adults, the levels at which exposure reduction actions are recommended (17, 18).

CDPH estimated the theoretical increased cancer risk from current/future exposure to contaminants considered carcinogenic. Carcinogenic contaminants exceeding comparison values in surface water and sediment are arsenic and PCBs (Appendix C, Tables 2 and 3). The estimated cancer risk for adults and child/teenager is 1 in 100,000 and 2 in 100,000, respectively. Cancer risks in this range are considered "very low increased risks."

Conclusion of West Stege Marsh Evaluation

On the basis of available data, CDPH concludes that past and current exposure from ingestion and dermal contact with surface water and sediment poses a health hazard for both noncancer and cancer health effects (theoretical increased cancer risk) to children/teenager who regularly played/plays in the West Stege Marsh. It is important to note that this conclusion is based on conservative assumptions meant to identify the possibility for exposures of health concern, so that steps can be taken to mitigate or prevent these exposures from occurring. Actual exposures to children/teenagers are likely much less.

On the basis of available data, CDPH concludes ingestion and dermal exposure to metals, pesticides and PCBs in surface water and sediment would not result in noncancer adverse health effects for children and adults who participate in restoration activities in the West Stege Marsh. Participating in restoration activities on a regular basis for 8 years, results in a very low theoretical increased cancer risk.

Lastly, there is a possibility for radionuclides associated with superphosphate fertilizer production on the adjacent Zeneca site, to have migrated into the East and West Stege Marsh. At the time of this writing, investigations and characterization of radionuclides in soil, sediment, and groundwater on the Zeneca site are incomplete. In addition, it is possible that contamination may be migrating through surface and/or groundwater from non-remediated areas of the marsh, the uplands, and the adjacent Zeneca site. Thus, UC should periodically (bi-annually) sample the sediment and unfiltered surface water in the West Stege Marsh to identify whether contaminants are migrating into the marsh. Groundwater should also be monitored to identify whether contaminants are migrating into the marsh from the Zeneca site. Finally, until characterization of radionuclides on the Zeneca site is complete and a determination is made whether characterization of radionuclides in the West Stege Marsh is needed, CDPH has determined that restoring the West Stege Marsh poses an indeterminate health hazard currently and in the future. Access to the West Stege Marsh should continue to be restricted.

Soil at the University of California Richmond Field Station

Workers have expressed concerns about exposure to contaminants at RFS. Some workers at RFS maintain the facilities by performing landscaping, plumbing repairs, digging trenches, etc. For certain projects, outside contractors (PG&E, telephone company, etc.) work on RFS and dig in the soil. For the other projects, full-time employees of the university who work in the maintenance unit conduct these activities, that is, they dig in surface and subsurface soils.

CDPH reviewed the available soil data and evaluated possible exposure for the RFS worker who might dig in the soil in an area where contamination still exists. This type of activity presents the greatest risk for exposure to on-site soil.

Soil investigations in the past focused on those parts of the site associated with known past manufacturing processes or storage areas or suspected areas of contamination: the California Cap Company explosives storage area, California Cap Company test pit area, forest products area, California Cap Company shell manufacturing area, Zeneca-related pyrite cinders area, mercury-bearing area, Heron Drive area, and the western storm drain.

In 2004, contractors for UC removed soils from five of these areas (Appendix B, Figure 4) (21). Most of the soil samples were analyzed for metals and PCBs (measured as Aroclor mixtures). The main COCs in surface to near surface soils on RFS are arsenic, cadmium, copper, lead, mercury, Aroclor 1248, Aroclor 1254, Aroclor 1258, and Aroclor 1260 (Appendix B Figures 5a and 5b; Appendix C, Table 8) (21). It is important to note that soil at RFS has not been fully characterized, indicating the possibility for maintenance workers to be exposed to contaminants

at levels not yet identified. Further, with the exception of PCBs, the highest concentrations of metals (arsenic, cadmium, copper, lead, and mercury) detected in surface and near surface soil are found in areas that have not been remediated (Appendix C, Table 8).

CDPH estimated exposure for two lengths of employment: long-term (23 years) employment, and the past 7 years of employment (Appendix C, Table 9). Because of the lack of characterization at the RFS, we used the most public health protective approach by assuming short-term and long-term workers were/are exposed to the highest concentrations of metals measured in soil, which are present in non-remediated areas. With respect to PCBs, we assumed short-term workers would not have worked in any of the excavated areas or "PCB hot spot" areas since they were already identified (Appendix C, Table 8).

Evaluation of Past Exposure (Long-Term) to Maintenance Workers Prior to Soil Excavation/Removal

CDPH assumed that the RFS worker dug a trench or holes in the soil in an area that was contaminated with the highest concentrations of chemicals detected in the Field Station surface and near surface soil. CDPH assumed that they dug without protection for 2 hours a day, 100 days per year, for 23 years, and during the digging they were exposed through the skin and through incidental ingestion of the soil. Inhalation (breathing) of contaminated-dust (particulates) could be a significant route of exposure depending on the activity, the amount of dust generated, and if the worker was wearing respiratory protection. CDPH did not quantify this exposure because estimating inhalation exposure from resuspension of soil is highly uncertain and depends on a number of factors (type of activity, environmental conditions, use of protective equipment, dust suppression activities, etc.).

CDPH estimated an exposure dose for the field station worker routinely digging in soil containing the highest/maximum concentrations of contaminants found in soil, prior to remedial actions (soil excavation) (Appendix C, Tables 8 and 9). Dose estimates for ingestion and dermal exposure to arsenic and PCBs exceed health comparison values, suggesting the possibility for workers to have experienced noncancer health effects (Appendix C, Table 9). However, the estimated doses are below the LOAEL of 0.014 mg arsenic/kg/day shown to cause skin effects in people and the LOAEL of 0.005 mg PCBs/kg day shown to cause immune effects (decreased antibody response) in monkeys (19, 20). Since dose estimates are below LOAEL and estimated doses are based on exposure to the maximum concentration of arsenic and PCBs found in soil, it is possible, but not probable that workers would have experienced health effects from ingestion and dermal exposure to arsenic and PCBs in soil. None of the other contaminants (metals) individually exceed their respective health comparison value.

The hazard index for the field station worker from exposure to the remaining COCs (metals) is estimated at 1.4 indicating the possibility for noncancer health effects (Appendix C, Table 9). It is important to note that exposure estimates were based on the highest concentrations of contaminants measured in soil, which were not found in the same locations. Thus, in order for a worker's exposure to exceed the hazard index, she/he would have had to routinely (2 hours a day, 100 days per year) dig in soil from those areas at RFS where the maximum levels of each contaminant were measured.

The most sensitive (primary) noncancer endpoints associated with COCs include skin effects (arsenic), renal (kidney) effects (cadmium, inorganic mercury) and gastrointestinal symptoms (copper) (20, 22-25). Since renal effects are the most sensitive endpoint associated with cadmium and inorganic mercury exposure, the interaction of these metals is evaluated further. Studies have shown that interactions with metals can influence the absorption, distribution, and excretion of one of more of the metals involved. For example, supplementation with zinc has been shown to provide some protection from the nephrotoxic (damaging and/or toxic to the kidney) effects of inorganic mercury (24). Zinc supplementation has also been shown to reduce oral absorption of cadmium (25). It is unclear whether the interaction between cadmium and inorganic mercury has on an additive effect (acting together, that is, a sum of the individual doses), a synergistic effect (combined toxic effects are greater than each chemical alone), or an antagonistic effect (one chemicals counteracting the effect of the other chemical, creating a less toxic effect) on the kidney. In situations where the interactions between chemicals are not understood, it is assumed that the effects are additive.

CDPH estimated a kidney target toxicity dose from exposure to cadmium and inorganic mercury (Appendix C, Table 9). The kidney target toxicity dose (0.00023 mg/kg/day) from exposure to cadmium and inorganic mercury is below the NOAEL for both cadmium (0.0021 mg cadmium/kg/day) and inorganic mercury (0.23 mg mercury/kg/day) (24, 25). Thus, it is possible, but not probable, that long-term workers experienced renal effects from combined exposure to cadmium and inorganic mercury in soil at the RFS.

The primary noncancer endpoints for the remaining COCs (including PCBs) differ, thus target toxicity doses were not calculated. These three COCs (arsenic, copper, and PCBs) would not have an additive effect on the target organ, as these chemicals affect different organ systems at the lowest dose. There could be some additive effects from these chemicals through a mechanism not involving the target organ; however, that is not known at this time.

The estimated BLL level for workers from exposure to the highest level of lead in soil (1,140 ppm) prior to remediation, as well as other sources of lead exposure typical for an adult, is 6.5 µg/dL. This level is below 10 µg/dL for pregnant women and 25 µg/dL for all other adults, the levels at which exposure reduction actions are recommended (17, 18).

In conclusion, estimated exposure to RFS maintenance workers from ingestion and dermal contact with the highest level of contaminants in soil are not likely to have resulted in noncancer health effects, though the possibility of health effects cannot be ruled out. While this conclusion is based on conservative assumptions (actual ingestion and dermal exposures are likely much less), inhalation of contaminated particulates could be a significant exposure route, adding to a worker's overall risk. The primary endpoints associated with exposures are immune effects (PCBs), skin effects (arsenic), and to a lesser extent, renal effects (cadmium, inorganic mercury) and gastrointestinal symptoms (copper).

Evaluation of Current Exposure (Short-Term) to Maintenance Workers

CDPH estimated current exposure to the field station worker who routinely digs in soil that contains the highest/maximum amount of contaminants found in the non-excavated areas

(Appendix C, Tables 8 and 9). Arsenic is the only COC that exceeds health comparison values. The estimated dose (0.0047 mg arsenic/kg/day) is three times lower than the LOAEL (0.014 mg arsenic/kg/day) shown to cause skin effects. Since doses estimates are below LOAEL and estimated doses are based exposure to the maximum concentration of arsenic found in soil (actual exposures are probably much less), it is unlikely that workers would have experienced health effects from exposure to arsenic in soil. None of the other COCs individually exceed their respective health comparison value.

The hazard index for the field station worker from exposure to the remaining COCs (metals and PCBs) is estimated at 1.6, indicating the possibility for noncancer health effects (Appendix C, Table 9). It is important to note that exposure estimates were based on the highest concentrations of contaminants measured in soil that are not found in the same locations. Thus, in order for a worker's exposure to exceed the hazard index, she/he would have to routinely (2 hours a day, 100 days per year for 7 years) dig in soil from those areas at RFS where the maximum levels of each contaminant were measured. While these estimations are based on conservative assumptions (actual ingestion and dermal exposures are likely much less), inhalation of contaminated particulates could be a significant route of exposure, adding to a workers overall risk. It is also possible that contaminant levels in other non-excavated areas where sampling has not yet been conducted could be higher.

CDPH estimated a kidney target toxicity dose from exposure to cadmium and inorganic mercury (see discussion in the section above) (Appendix C, Table 9). The kidney target toxicity dose (0.00023 mg/kg/day) from exposure to cadmium and inorganic mercury is below both the NOAEL for cadmium (0.0021 mg cadmium/kg/day) and the NOAEL for inorganic mercury (0.23 mg mercury/kg/day) (24, 25). Thus, it is possible, but not probable, that short-term workers experienced renal effects from combined exposure to cadmium and inorganic mercury in soil at the RFS.

Target toxicity doses were not calculated for the remaining COCs, because the primary endpoints of concern are not the same (discussed in the section above). The primary noncancer endpoints associated with COCs include skin effects (arsenic), immune effects (PCBs), and gastrointestinal symptoms (copper). These COCs would not have an additive effect on the target organ, as these chemicals affect different organ systems at the lowest dose. There could be some additive effects from these chemicals through a mechanism not involving the target organ; however, that is not known at this time.

The estimated BLL level for workers from exposure to the highest level of lead (1,140 ppm) remaining in soil is 6.5 µg/dL, which is below the level (25 µg/dL) at which exposure reduction actions would be recommended.

Cumulative Theoretical Increased Cancer Risk from Past, Current, and Future Exposure

CDPH estimated the theoretical increased risk of cancer for a long-term worker digging in the excavated areas (prior to removals/excavations in 2004) to be 4 in 10,000. Digging in soil from non-excavated areas would add (7 in 100,000) to the cancer risk for a long-term worker; the

cumulative theoretical increased cancer risk for an RFS worker from 30 years[3] of exposure is estimated to be 5 in 10,000. Increased cancer risks in the range (greater than 1 in 10,000) are considered unacceptable risks (10). The increased cancer risks are based on exposure to maximum concentrations; the actual risk would likely be less. The chemicals associated with an increased cancer risk are arsenic (skin, liver, bladder, and lung) and PCBs (liver, biliary).

Conclusion of Soil Evaluation

CDPH concludes that exposure to RFS maintenance workers from ingestion and dermal contact with soil poses a health hazard for both noncancer and cancer health effects (unacceptable theoretical increased cancer risk). While this conclusion is based on conservative assumptions (actual ingestion and dermal exposure are likely much less), it does not include potential inhalation exposures to contaminated soil particulate, which could be a significant route of exposure, adding to a worker's overall risk. Inhalation exposure can be mitigated if workers wear proper respiratory protection while working in RFS soil. Additional characterization of on-site soil throughout the RFS is needed to identify other areas where potential contamination may exist. Chemicals used in research activities at RFS, as well as known contaminants from historic uses of RFS and the adjacent Zeneca (former Stauffer Chemical) site should be analyzed. Characterization of soil in the area where the Forest Products Laboratory is located should include analyses for pentachlorophenol and chlorophenol byproducts (26).

Evaluation of Ambient Air During Remedial Work

During discussions with RFS employees, CDPH was informed that a great deal of dust was generated during past remedial work at RFS and the adjacent Zeneca/Campus Bay, which is believed to have resulted in a number of health effects. Dust is made up of vairous sizes of particulate matter. Particulate matter less than 10 microns in aerodynamic diameter, known as PM 10[4], is considered among the most harmful of all air pollutants, because these particles are inhaled they can become lodged deep in the lungs (27). CDPH reviewed available air monitoring data in an effort to understand exposures that may have occurred as a result of these activities.

Air monitoring of total dust (PM 10 was not measured) and mercury vapor was conducted during Phase 1 and Phase 2 remedial activities at the RFS (1, 28). Phase 1 and Phase 2 activities consisted of removal and treatment of mercury-contaminated soils from the marsh and upland areas of RFS (Appendix B, Figure 2).

Dust

Between September 16, 2002, and December 6, 2002, total dust concentrations were measured from six locations along the site perimeter to monitor airborne dust leaving the remedial area of the mercury contamination. Dust monitors were placed on the site perimeter for the duration of

[3]Theoretical increased cancer risks assume 30 years of exposure and are calculated based on 23 years exposure to the highest concentrations of contaminants prior to remedial actions, plus 7 years of exposure to the highest concentrations of contaminants remaining in soil.
[4]As a point of reference, the 24-hour average California Ambient Air Quality Standard for PM 10 is 50 µg/ m^3.

each workday. The average dust concentrations did not exceed the site-specific dust action level[5] of 2 milligrams per cubic meter (mg/m^3) or 2,000 $\mu g/m^3$ (28). However, on several days, the maximum concentration of dust measured from at least one location exceeded the dust action level of 2 mg/m^3 (2,000 $\mu g/m^3$) (Note: there were a number of days when maximum dust concentrations were not recorded). Dust was measured as high as 39.75 mg/m^3 (39,750 $\mu g/m^3$) (29).

Between August 11, 2003, and November 26, 2003, total dust was measured at seven locations along the site perimeter, during Phase 2 remedial work. Average dust concentrations ranged from 0.000-0.125 mg/m^3 (1,250 $\mu g/m^3$) (1). Average dust concentrations did not exceed the site-specific dust action level of 2 mg/m^3 (2,000 $\mu g/m^3$). On numerous days (more than 35 days) the maximum dust concentration measured from at least one location exceeded the the site-specific dust action level of 2 mg/m^3 (2,000 $\mu g/m^3$). Dust levels were measured as high as 9.344 mg/m^3 (9,344 $\mu g/m^3$).

Part of the remedial work included mixing powdered activated carbon with excavated materials to neutralize pH and stabilize metals and mercury. During powdered activated carbon reagent addition, there were some detections of carbon dust outside the work area. Carbon dust levels did not exceed 2 mg/m^3. However, some of this dust did deposit on structures in the area (28).

In conclusion, it is possible for RFS workers to have experienced irritation of the eyes, nose, throat, and respiratory tract from breathing dust (particulate matter) generated during Phase 1 and Phase 2 remedial work. It is not known what chemicals were attached to the dust particles (except carbon) and thus not possible to evaluate health effects from potential exposure to other chemicals.

Mercury Vapor

Between November 21, 2002, and December 6, 2002, URS Corporation (URS) conducted air monitoring for mercury vapor during the Phase 1 remedial work at RFS. Mercury levels in the air at the work site were monitored using a Jerome Mercury Vapor Analyzer with a detection limit of 0.003 mg/m^3 (3 $\mu g/m^3$). According to a URS summary statement (data not provided), mercury was not detected above the detection limit (28). While this instrument may be appropriate for monitoring worker exposures to mercury vapor, non-worker (residential) exposure standards are set at lower levels. For example, the acute REL for inorganic mercury is 0.0018 mg/m^3 (1.8 $\mu g/m^3$).

During the early part of the Phase 2 remedial work, between September 12, 2003, and September 23, 2003, UC health and safety personnel monitored for mercury levels in the air at the work site using a Jerome Mercury Vapor Analyzer with a detection limit of 0.003 mg/m^3 (3 $\mu g/m^3$) (Appendix C, Table 10). Of the 125 samples collected during this sampling effort, 15 samples (collected at various times during each day) had detectable concentrations of mercury, ranging from 0.003-0.006 mg/m^3 (3-6 $\mu g/m^3$).

[5]Site-specific dust action level approved by the California Regional Water Quality Control Board, San Francisco Region, is based on PEL (permissible exposure level) for dust (5 mg/m^3), which was modified to be protective of the highest mercury level in soil. The level at which dust becomes visible (dust visibility threshold) is approximately 2 mg/m^3.

It is difficult to determine the level of mercury outside of the Phase 2 work area, either off-site or in other areas of RFS, since dilution with the ambient air would occur. In an effort to gain a better understanding of airborne mercury levels outside of the work area, CDPH obtained data collected by EPA Region 9 Laboratory located on the south west side of RFS (Appendix B, Figure 4).

EPA conducted air monitoring at the laboratory from August 26, 2003, until September 28, 2003 (30). The location of the excavation areas in relation to the laboratory ranged from approximately 150 feet to several hundred feet. Mercury was detected in air on several days at concentrations ranging from 0.01 µg/m3 to 0.9 µg/m^3 (30). Mercury levels did not exceed the acute REL of 1.8 µg/m^3. On two days (September 10 and September 12), mercury levels exceeded the chronic MRL of 0.2 µg/m^3 for time periods of less than an hour (Appendix B, Figure 6). The acute REL is the most appropriate comparison value for looking at short-term exposure; we included the chronic MRL as additional information to help put the exposure into context (e.g., chronic MRL: a constant exposure level occurring for greater than 365 days, without appreciable health risk). While these do not provide information about levels of airborne mercury in other areas of the RFS, particularly areas predominantly downwind of the excavation, they do show a decrease in levels outside the work area, at the EPA laboratory. The highest value (0.9 µg/m^3) was measured on September 10, 2003, and cannot be compared with data collected at the work area because there were no samples reported for that day (Appendix B, Figure 6; Appendix C, Table 10).

It appears that exposure to low level airborne mercury may have occurred in the vicinity of the Phase 2 work area. However, based on the available data, short-term exposures at the levels measured in air during the remedial work would not be expected to result in noncancer adverse health effects.

Future remedial activities at the site should include adequate dust suppression methods and perimeter air monitoring, with detection limits comparable to residential standards.

Evaluation of Indoor Air

During times when no remedial work is occurring, workers at RFS have expressed concerns that site-related contaminants are present in indoor air, either from soil being tracked indoors or through soil gas migration, resulting in health effects. In response to these concerns, UC health and safety personnel conducted indoor and outdoor air sampling at RFS, between August 16, 2005, and October 20, 2005.

Indoor Air Quality in General

Evaluating indoor air quality is complicated because indoor air typically contains many chemicals and is generally considered unhealthy (30). Several studies over the years have compared the overall quality of indoor versus outdoor air (30-32). The findings have consistently shown that the overall air quality indoors is invariably worse than the outdoor air quality. There are numerous reasons for the marked difference between indoor and outdoor air quality. Many

buildings have very poor air circulation and air turnover rates. This means that any chemical released into the air of a building will remain there. If chemicals are consistently released into buildings, the total concentration of that chemical will increase. Many of the construction materials used in home and office construction contain various substances (volatile chemicals) that continue to release chemicals into the air. Plywood, insulation, foam, and resins are examples of construction materials that have been shown to release, or off-gas, chemicals into the indoor air. (See Table 11 in Appendix C for a limited list of chemicals known be associated with household products) This is further complicated at RFS due to potential use of chemicals for research activities that may be impacting indoor air.

Metals in Indoor Air at the Richmond Field Station

It is possible for site-related contaminants present in soil to become airborne and enter buildings at RFS. On August 16, 2005, indoor air particulate samples were collected in Buildings 163 and 175, in an effort to address concerns expressed by workers in these buildings (29) (Appendix B, Figure 7; Appendix C, Table 12). A sample was also collected from the rooftop of Building 175. Samples were analyzed for metals (arsenic, cadmium, nickel, lead, mercury, selenium, and zinc). Arsenic was detected in Buildings 163 and 175 at 0.098 $\mu g/m^3$ and 0.085 $\mu g/m^3$, respectively (29). Arsenic is not a commonly found contaminant in indoor air. These levels do not exceed noncancer comparison values for acute exposure (0.19 $\mu g/m^3$). However, these levels exceed the cancer comparison value (0.0002 $\mu g/m^3$) for arsenic. On September 20, 2005, Buildings 163 and 175 were resampled, and arsenic was not detected above the detection limit (0.05 $\mu g/m^3$) (Appendix C, Table 12). No other metals were detected in indoor air during these sampling events.

On December 6, 2005, indoor air samples were collected in Building 478 and analyzed for arsenic. Arsenic was not detected at a laboratory detection limit of 0.05 $\mu g/m^3$ (33).

These data are too limited to quantify exposures and draw conclusions about potential health impacts from breathing arsenic in indoor air. These data show the potential for arsenic to enter Buildings 163 and 175 at levels of concern for prolonged/chronic exposure (greater than 365 days). UC should take steps to identify and mitigate the source of arsenic in indoor air. Additional indoor air sampling should be conducted on an intermittent basis to ensure workers are not being exposed to arsenic at levels of health concern.

Mercury Vapor in Indoor Air at the Richmond Field Station

Unlike other metals, mercury can be a vapor at room temperature. Due to worker concerns about mercury contamination affecting indoor air at RFS, mercury vapor samples were collected in Buildings 102, 163, and 175, during the August 2005 sampling event (Appendix B, Figure 7). Mercury was not detected at the laboratory detection limit of 0.52-0.84 $\mu g/m^3$ (0.00052-0.00084 mg/m^3) (29).

Volatile Organic Chemicals in Indoor Air at the Richmond Field Station

It is possible for the indoor air in buildings located along the east, northeast boundary of RFS to

be affected by groundwater contaminated with volatile organic chemicals (VOCs) as a result of past activities at the Forest Products Laboratory and through migration of VOCs in groundwater from the neighboring Zeneca site (26). The groundwater flow direction in the northeast portion of the RFS is under investigation. In cases when the groundwater is close to the surface (within 30 feet), VOCs in the groundwater can be pulled into buildings. This is known as soil gas migration/vapor intrusion. Groundwater in the RFS area is shallow, ranging from 6-15 feet below ground surface (bgs) (depending on location and the time of year), creating the potential for soil gas to migrate from VOC-contaminated groundwater into buildings. Once inside the building, these gases or vapors can be inhaled. While soil gas can be an important source of in-building air contaminants, it is only one of several contributors to the total air contaminants found inside a building (discussed above).

An important factor for evaluating soil gas migration is having an understanding of the extent of VOC contamination in groundwater. There has not been adequate characterization of the groundwater along the east and northeast side of RFS, which limits the ability for CDPH to evaluate the soil gas pathway. The following describes indoor air sampling that has been conducted at RFS.

On September 21, 2005, indoor air samples collected from Buildings 163 and 175 were analyzed for VOCs (Appendix B, Figure 7; Appendix C, Table 12). With the exception of formaldehyde, none of the VOCs exceed noncancer comparison values. Formaldehyde was measured in Building 163 at 410 $\mu g/m^3$, exceeding noncancer comparison values for acute exposure (exposure to a chemical for 14 days or less in duration). In studies of short-term exposure (less than 8 hours) to formaldehyde, irritant effects of the nasal passage and throat were seen at levels ranging from about 490 $\mu g/m^3$ to 3,700 $\mu g/m^3$ (34). Thus, it is possible for RFS workers to have experienced irritant effects of the nose and throat from exposure to formaldehyde in Building 163, between September 21, 2005, and October 20, 2005, when sampling indicated formaldehyde in Building 163 at levels below health concern (Appendix C, Table 12). In Building 175, formaldehyde levels exceeded comparison values for chronic exposure. It is not possible to determine whether workers in Building 175 are being chronically exposed to formaldehyde, based on one sampling event.

It is worth noting that formaldehyde was measured in outdoor air (roof of Building 175) at a level that exceeds chronic REL of 3 $\mu g/m^3$ (Appendix C, Table 12). This is not unusual since formaldehyde is a common contaminant in outdoor air, due to many sources. According to the California Air Resources Board, formaldehyde is present in outdoor air an average level of 3.7 $\mu g/m^3$, up to 14.7 $\mu g/m^3$, depending on the location (35). Formaldehyde is present at higher levels in outdoor air in urban areas compared to rural areas. For example, the Chevron refinery, located approximately 3.6 miles southwest of RFS, reports releasing 5,000-86,000 pounds of formaldehyde to the air each year (36).

Five contaminants (benzene, formaldehyde, methylene chloride, tetrachloroethylene, and trichloroethylene) detected exceed cancer comparison values that are developed assuming daily exposure for a lifetime (Appendix C, Table 12). Exceeding these values indicates the possibility of an increased risk of cancer greater than one-in-a-million. CDPH did not estimate the increased

cancer risks because one sampling event does not provide enough information to draw conclusions about exposure that is assumed to be over a lifetime (70 years).

On December 6, 2005, indoor air samples were collected inside Building 478, and analyzed for a limited number of VOCs: tetrachloroethylene (PCE), trichloroethylene (TCE), and vinyl chloride (33) (Appendix B, Figure 7). None of the VOCs were detected above laboratory detection limits. However, the detection limits were not sensitive enough to fully evaluate the health impact to employees working at the RFS.

Characterization of the groundwater along the east, northeast side of RFS (on site) is needed to evaluate the potential for VOCs to be affecting indoor air in buildings in these areas, as a result of soil gas migration. Past activities at the Forest Products Laboratory may have resulted in VOC-contamination of soil and groundwater in this area (26). It is also possible for VOC-contaminated groundwater to be migrating onto RFS from the adjacent Zeneca property. Additional indoor air sampling should be conducted in Building 163 and 175 to determine if formaldehyde is elevated above levels typical of indoor air.

Quality Assurance and Quality Control

In preparing this PHA, ATSDR and CDPH used information in the referenced documents and assumed that adequate assurance and quality control measures were followed, with regard to chain-of-custody, laboratory procedures, and data reporting. Most of the documents used in the health assessment are prepared for regulatory agencies, which undergo review to ensure that proper quality control measures were followed.

Community Health Concerns and Evaluation

Introduction and Purpose

Community members are often concerned about contaminated sites. The collection, documentation, and response to community health concerns are critical to the PHA process. This section outlines CDPH efforts to engage with workers at RFS and provides an overview of the health- and exposure-related concerns reported by RFS workers to CDPH. In addition, this section provides a response to the concerns with educational information and specifically addresses the health and other concerns within the framework and limitations of the PHA.

Background

RFS is adjacent to the former Stauffer Chemical Company site, an area currently referred to as Zeneca/Campus Bay. Remedial activities at Zeneca/Campus Bay created community and RFS worker concerns about exposure. The community was also concerned that RWQCB was not conducting rigorous oversight of the remediation of Zeneca/Campus Bay and RFS.

Community advocates petitioned the Richmond City Council to support a change in the regulatory agency overseeing site cleanup. In July 2004, the Contra Costa County Health Services Department (CCCHSD) supported the community's position, citing DTSC as the

agency with the adequate expertise to provide oversight for complex sites such as Zeneca/Campus Bay and RFS (37). It was reported that UC objected to the proposed transfer, contending that it was conducting an adequate cleanup of the site under RWQCB supervision and that the inclusion of the RFS in the transfer request was a result of confusion due to its proximity with the Zeneca/Campus Bay site (38). Community advocates were concerned when UC selected Cherokee Simeon Ventures (CSV) to develop an academic research complex at RFS because CSV was also involved at the Zeneca/Campus Bay site; this prompted the community to advocate that both sites be regulated by DTSC (39). After receiving input from the CCCHSD, the Richmond City Council and the Contra Costa County Board of Supervisors supported the transfer of regulatory oversight of both sites to DTSC that occurred in May 2005. In June 2005, DTSC formed a Community Advisory Group (CAG), as a result of a community petition for public involvement in the clean-up process at the Zeneca site. CAG obtained RFS worker representation in September 2005, after CAG members expanded their purview to include other sites in the area.

In May 2005, CDPH and CCCHSD attended an interagency briefing and coordination meeting at the RFS. At that meeting, both health agencies committed themselves to develop a provisional joint health statement that would provide an evaluation of any immediate exposure risks associated with the Zeneca/Campus Bay and RFS sites. The health statement was released in June 2005 and shared with RFS workers at a meeting on RFS. UC management and the unions also distributed the June 2005 provisional joint health statement. The provisional joint health statement was updated in February 2006. Highlights of the provisional joint health statement were shared with the community at DTSC CAG meetings on June 30, 2005, and February 8, 2006.

Aside from RFS-related concerns, RFS workers and union advocates were also concerned about possible exposures to workers that may have occurred during remedial activities at Zeneca/Campus Bay. Zeneca-related concerns will be addressed in a separate PHA to be released in 2007.

Process for Gathering Community Health Concerns

CDPH staff first became aware of workers' health concerns in April 2005, when contacted by DTSC about the site. Some community members had documented the illnesses and deaths of some RFS workers and they shared a list of those concerns (without identifying information) to CDPH.

EHIB staff worked with the Occupational Health Branch of CDPH to determine the appropriate mechanisms to reach workers and to prepare relevant health and safety information and referrals. In October 2005, EHIB staff met with UC management, and medical, health and safety personnel to provide an overview of the PHA process and receive input from UC regarding conditions at the RFS.

CDPH organized two public availability sessions. The first session, held on October 24, 2005, was held on site at RFS. The second session, held on October 25, 2005, was held at the CDPH Richmond campus, approximately 2 miles from the RFS site, in order to accommodate those

who could not attend the previous session or felt more comfortable speaking with CDPH staff off site. To publicize both sessions, CDPH worked with UC management and labor unions at the RFS. UC management sent electronic copies of the flyer to all managers and posted the flyer throughout the RFS campus. Labor union representatives sent the session flyers to union stewards and workers via e-mail. CDPH staff documented the concerns of 17 current and former workers of RFS at the two public availability sessions.

In addition, CDPH worked through labor union and community networks to invite workers who were not able to attend either session to contact CDPH via mail, e-mail, and telephone. CDPH staff also presented the PHA process at the October 2005 CAG meeting. After these outreach efforts, CDPH responded to phone calls and e-mails from several more current and former workers of RFS. Some family members of former RFS workers contacted CDPH to report some additional health concerns. These community members wanted to include the health problems suffered by the former RFS worker in their family in the PHA. CDPH staff documented the concerns of seven additional current and former workers of RFS through these outreach efforts.

Historical Concerns

CDPH received a compilation of health- and exposure-related concerns recorded by RFS workers between 1961 and 1972. The compilation is primarily focused on health concerns related to emissions from the Stauffer Chemical Company. Workers described strong chemical odors that resembled onions and garlic; some described the odors as sulfur. Workers reported health effects such as nausea, vomiting, irritation of the nose, throat, and eyes, nosebleeds and irritation of nasal membranes, and dull to severe headaches. A more complete overview of these historical concerns will be provided in the PHA for the Zeneca/Campus Bay, former Stauffer Chemical site, to be released in 2007.

In June 2005, a former RFS employee told a local radio station he was ordered to dump drums filled with what he believed to be radioactive waste from Lawrence Berkeley National Laboratories in the marshland area of RFS in the 1960s (40). A formal statement was filed with DTSC in August 2005, and the agency conducted a magnetometer survey in the area in October 2005 in an attempt to locate the drums. Although metal was detected, subsequent investigations revealed the metal to be concrete cylinders with steel casings. DTSC continues to investigate changes in RFS marshland topography from the 1960s to the present as they relate to the possible location of the drums.

The former RFS employee who stated he was ordered to dump drums filled with what he believed to be radioactive waste from Lawrence Berkeley National Laboratories at RFS in the 1960s maintained that, after handling the contents of the drums, he experienced severe health effects. The former worker described swelling of his feet and gums and bleeding through his ears, nose, and eyes.

Current/General Concerns

In addition to health and exposure concerns, workers expressed frustration to what they believe to be improper handling of the site by RWQCB and the lack of information about the

characterization and clean-up process. Workers called for a better characterization and proper remediation of the site.

In addition, some workers expressed distrust of UC medical providers and reported seeking outside care for health issues they considered as site-related. UC management and representatives were concerned that if workers did not report illnesses through UC mechanisms, UC would be unable to assist workers with health issues that might be related to the site. Some members of UC management were worried that workers were experiencing stress related to the site.

Participants asked for additional safety training and better access to protective gear for workers who spend the bulk of their time outdoors on RFS grounds. Some stated that they had asked for protective gear or equipment but were made to feel bad about asking. Others stated that there was a greater need for information dissemination beyond the RFS website that was not accessible by all. In general, workers wanted UC to develop a more responsive, prompt, and transparent approach to dealing with workers' concerns and requests.

Community Health Concerns Evaluation

CDPH documented the health concerns of 24 current and former RFS workers. These participants in the PHA process described a number of concerns and health effects that occurred or are occurring. This section discusses their health concerns in greater detail.

Some community members made an effort to document a list of illnesses and deaths of some RFS workers in 2005. The information was collected anecdotally and was comprised of 26 cases.

The following table presents the health effects and concerns expressed by workers to CDPH. In response to these concerns, CDPH provides a brief description about the health effects, their known causes, including environmental or chemical agents, in particular the ones associated with RFS.

Health Concerns/Effects Expressed to CDPH	
Noncancer health effects concerns	**Cancer health effects/concerns**
Headaches/migraines	Thyroid cancer
Inability to focus	Breast cancer
Allergies/sinus problems	Liver cancer
Eye irritation	*Pancreatic cancer*
Nose irritation/dryness/nose bleeds	*Kidney cancer*
Impaired sense of smell	*Throat cancer*
Coughing/sneezing/choking	
Dry mouth/loss of voice	
Skin irritation	
Stomach ache/diarrhea	
Weight gain	
Numbness in feet and hands	
Chronic fatigue	
Fertility concerns	
Developmental issues for children in utero	
Positive blood test for arsenic	
Positive blood test for mercury	
Swelling of feet, gums; bleeding of ears, nose and eyes*	
Heart disease	
Embolism	
Thyroid problems	
Asthma	
Abdominal pain	
Head and tongue tumors, not yet diagnosed	
Arsenic poisoning	
Bacterial meningitis	

*one-time incident in the past involving the possible handling of radioactive material.
Health effects are organized as either related or not related to cancer.
Items in *italics* denote health concerns/effects documented by community members.
Due to the possibility of overlap between CDPH- and community-collected health concerns, repeated concerns appear only once.

Cancer Risk Factors and Health Disparities

Cancer as a whole is the second leading cause of death in the United States after heart disease. However, grouping cancer together is very misleading because there are many different types of cancer, and each type has different causes and risk factors. It is rarely possible to know why a particular individual develops cancer, but studies have found certain risk factors to be associated with specific cancers. For example, prolonged exposure to sunlight is a risk factor for skin cancer and cigarette smoking is a risk factor for lung cancer. Usually, there are several factors that work together to cause cancer. For example, a number of factors may increase a persons risk for lung

cancer: cigarette smoking; having a genetic susceptibility; exposure to another cancer causing agent, like asbestos; and poor diet.

Gender is another factor that influences cancer risk. Lung cancer is now the leading cause of cancer in both men and women. With the exception of lung cancer, men and women differ in cancer risk. The second and third most common cancers in men are colon and prostate, respectively. For women, the second and third most common cancers are breast and colon, respectively (41).

Age is another important risk factor because people at different ages have different levels of risk for certain cancers. For example, in men the risk for testicular cancer decreases with age but the risk for prostate cancer increases with age. In general, the older a person gets, the more likely he/she will get cancer. Thus, more cancer cases will occur in populations that have greater proportion of elderly persons.

People of different ethnic and racial backgrounds get cancer following different patterns. These differences are known as cancer health disparities—they are inequalities that occur when members of one group of people do not enjoy the same health status as other groups (42). Cancer health disparities occur as a result of differences in lifestyle, income, education, access to healthcare, and/or environmental and biological factors (42). The American Cancer Society reports that African American men have the highest cancer related death rate of 339 deaths per 100,000 in the United States, followed by white men with a rate of 243 deaths per 100,000, and Hispanic men with a rate of 171 deaths per 100,000. African American women have the highest rate of cancer related death with a rate of 194 deaths per 100,000, followed by white women with a rate of 165 deaths per 100,000, and American Indian women with a rate of 114 deaths per 100,000 (41).

Evaluation of Cancer Health Concerns at the Richmond Field Station

Workers and former workers of RFS community reported receiving a diagnosis of cancer, or knowing someone diagnosed with cancer, or concern about the risk of cancer from exposures occurring while working at the field station. Diagnosing cancer related to environmental exposure is particularly difficult for a number of reasons: first, it is unknown how long someone must be exposed to cause a particular cancer; second, it is unknown how much time must pass between the environmental exposure and the development of the cancer (latency); lastly, it is difficult to quantify past exposure because we are exposed to numerous chemicals on a daily basis. In the absence of this information, it is difficult to make a diagnosis of cancer that is directly related to an environmental exposure. Doctors who treat cancer (oncologists) normally focus on treatment, rather than speculate about why their patient developed cancer.

It is important to note the current scientific understanding of exposure to chemicals and related health effects is limited. Most of the information has been derived from studies on animals or workers who have received much higher levels of exposure than typically seen at sites where environmental contamination exists, such as RFS. This is further complicated by the fact that most studies look at chemicals on an individual basis, not as mixtures (exposure to multiple chemicals). These limitations add uncertainty to the conclusions about potential health impact as a result of exposure to contaminants at RFS.

Former/current workers expressed concern about the following cancers: thyroid cancer, breast cancer, liver cancer, pancreatic cancer, kidney cancer, and throat cancer. This section describes the known causes of six different cancers with which members of the community have expressed concerns. The cancers will be addressed as they relate to the environmental contaminants of greatest concern identified by CDPH, based on a review of available site-related environmental data. The contaminants of greatest concern are mercury, arsenic, copper, lead, PCBs, and formaldehyde. The cancers will be described in context of known environmental causes and a determination of whether arsenic, copper, mercury, lead, PCBs, and formaldehyde are known causes, based on the current understanding/scientific knowledge.

Thyroid Cancer

The thyroid gland is an organ found in the front of the neck. The thyroid gland secretes the hormone thyroxin, essential for normal body growth in infancy and childhood. Thyroid cancer is cancer of the thyroid gland and is an uncommon form of cancer, with only about 30,000 new cases expected to occur in 2006 in the United States. Thyroid cancer occurs more frequently in women; most studies show that for every man with thyroid cancer, there are three women with thyroid cancer. Thyroid cancer mainly affects young people with two thirds of cases occurring between ages 25 and 55 years (43). The best known environmental risk factor for the development of thyroid cancer is from exposures to ionizing radiation[6], especially those exposures that occur 10 to 40 years prior to presentation or onset of disease (44). Studies have indicated that commercial PCB mixtures are carcinogenic in animals based on induction of tumors in the thyroid (19). No studies were located showing an association with exposure to mercury, arsenic, copper, lead, or formaldehyde, and thyroid cancer.

Breast Cancer

Until recently, breast cancer was the most common cancer in women. Over 212,900 women in the United States will be diagnosed with breast cancer in 2006 (41). There are three periods in a woman's life that affect breast cancer risk: age at the time of first menstrual period; age at first full-term pregnancy; and age of menopause (45).

Research is being done to learn how the environment might affect breast cancer risk. There are some links between breast cancer risk and exposure to estrogenic compounds, such as dioxin and diethylstilbestrol. However, a clear link between breast cancer and exposure to contaminants such as PCBs and pesticides, at levels commonly found in the environment, has not been shown at this time (46). Exposure to ionizing radiation is an established risk factor for breast cancer (47). There are some occupational risk factors for breast cancer. In large epidemiologic studies of occupation and cancer, jobs with higher education have increased breast and decreased cervical cancer rates; this finding may be confounded (influenced) by socioeconomic class and advanced maternal age (older age of mother) at first childbirth (47). A variety of studies have examined the possible relationship between breast cancer and exposure to permanent hair dyes. In two studies,

[6]Ionizing radiation is any one of several types of particles and rays given off by radioactive material, high-voltage equipment, nuclear reactions, and stars. The types that are normally important to your health are alpha particles, beta particles, X rays, and gamma rays.

regular use of permanent hair dyes was found among those with breast cancer as opposed to controls (47, 48). Case-control studies of the general population are inconclusive with respect to associations between environmental exposures to PCBs and risk of breast cancer (19). There is not strong evidence in the scientific literature showing an association between exposure to mercury, arsenic, copper, lead, or formaldehyde, and breast cancer.

Liver Cancer

The liver is the largest internal organ in the body. It is found just under the right lung and diaphragm. More than 500 vital functions have been identified with the liver. The liver regulates most chemical levels in the blood and excretes a product called bile that helps to break down fats, preparing them for further digestion and absorption. The American Cancer Society estimates there will be about 18,000 new cases of liver cancer in the United States in 2007. Liver cancer is twice as common in men as in women; this is probably due to greater male exposure to causative agents, such as alcohol, smoking, anabolic steroids and occupationally-related chemicals (vinyl chloride, etc.). There are two main types of malignant liver cancer: hepatocellular carcinoma and hemangiosarcoma. The more common cell type for liver cancer is hepatocellular carcinoma. The three primary risk factors for hepatocellular carcinoma worldwide include hepatitis B virus, alcohol, and aflatoxins (cancer-causing substances are made by a fungus that can contaminate peanuts, wheat, soybeans, groundnuts, corn, and rice) (49). A second form of liver cancer, hepatic hemangiosarcoma, is much more uncommon than hepatocellular carcinoma and is closely identified with occupational causes. The two major occupational and environmental causes include vinyl chloride and inorganic arsenic (49-51). Epidemiological studies have indicated that arsenic exposure is associated with liver cancer. Most commonly, the exposure to inorganic arsenic has been from the contamination of the drinking water.

There is conclusive evidence that commercial PCB mixtures are carcinogenic in animals based on the development of tumors in the liver (19). There is evidence showing an association between formaldehyde and cirrhosis of the liver, which can lead to liver cancer. No studies were located showing an association between exposure to mercury and liver cancer.

It is not possible to determine the cause of the liver cancer case expressed to CDPH. We identified potential exposures to arsenic, PCBs, and formaldehyde, which could have increased an individual's risk of developing liver cancer, if they were exposed under the conservative scenarios assumed. The estimated risk is considered a "low increased risk."

Pancreatic Cancer

The pancreas is a gland found behind the stomach and is about 6 inches long and less than 2 inches wide. The pancreas consists of separate glands that secrete enzymes which break down fats and proteins in foods, so the body can use them and make hormones (such as insulin) that help balance the amount of sugar in the blood. The American Cancer Society predicts that, in 2007, about 33,730 people in the United States will be found to have pancreatic cancer and about 32,300 will die of the disease. This kind of cancer is the fourth leading cause of cancer death. Pancreatic cancer is difficult to diagnose and tends to be diagnosed when the disease is

advanced. There is a strong association between tobacco smoke and pancreatic cancer (52). There is evidence that DDT and its metabolites, certain fungicides, herbicides, solvents, PCBs, and ionizing radiation could be associated with pancreatic cancer (19, 49). There is limited evidence suggesting a link between formaldehyde and pancreatic cancer. There is not adequate understanding of the levels of formaldehyde in indoor air at RFS, due to limited data. Thus, it is not possible to determine if there is a connection between the exposure and the case of pancreatic cancer. No studies were located showing a strong link between mercury, copper, or lead and pancreatic cancer.

Kidney Cancer

The kidneys are two bean-shaped organs. One is just to the left and the other to the right of the backbone. The lower rib cage protects the kidneys. The kidneys filter the blood and help the body get rid of excess water, salt, and waste products in the form of urine. Urine travels through long tubes (ureters) to the bladder where it is stored until the person passes the urine, or urinates. There are two main types of kidney cancers: renal cell and renal pelvis cancers. The American Cancer Society predicts that there will be about 38,890 new cases of kidney cancer in the year 2006 in this country. About 12,840 people will die each year from this disease. These numbers include both adults and children. Most people with this cancer are older. It is very uncommon among people under age 45. According to the National Institute of Cancer, some identified risk factors for fatal cancer include, smoking, alcohol consumption, obesity, and hypertension.

Epidemiologic studies from Taiwan and Argentina have found that arsenic ingestion from the drinking water can cause cancers of the kidney with prolonged exposure (53). It is unknown whether inhaling arsenic contaminated dust can cause kidney cancer. Occupational exposures suspected of causing kidney cancer include: polycyclic aromatic hydrocarbons, asbestos, lead salts, cadmium, petroleum products, distilled fuels, and aliphatic hydrocarbons. No studies were located showing a strong link between kidney cancer and exposure to copper, lead, mercury, PCBs, and formaldehyde.

Throat Cancer

Cancer of the throat may include many different anatomical regions such as the nasopharynx, esophagus, and nasal sinuses. CDPH is not aware of which throat cancer the former/current RFS worker developed. Nasopharyngeal cancer develops in the nasopharynx, an area in the back of the nose toward the base of the skull. The nasopharynx is a box-like chamber about 1½ inch on each edge. It lies just above the soft palate, just in back of the entrance into the nasal passages. Although it is considered an oral cancer, nasopharyngeal cancer is different from most oral cancers. It tends to spread widely, is not often treated by surgery, and has different risk factors from most oral cancers. Nasopharyngeal cancer is relatively rare in most parts of the world. In North America, it occurs in seven out of every one million persons (41). The International Agency for Research on Cancer has concluded that formaldehyde causes nasopharyngeal cancer (54). The additional risk of nasopharyngeal cancer from exposure to formaldehyde at RFS could not be quantified, due a lack of exposure data/air monitoring data.

The nasal sinus refers to the nasal cavity and the paranasal sinuses. The nose opens into the nasal

passageway, or cavity. This cavity runs along the top of the palate (the roof of the mouth, the shelf that separates the nose from the mouth) and turns downward to join the passage from the mouth to the throat. The term paranasal means "around or near the nose." Sinuses are cavities or small tunnels. The nasal cavity and paranasal sinuses help filter, warm, and humidify the air we breathe. They also give your voice resonance, lighten the weight of the skull, and provide a bony framework for the face and eyes. Cancers of the nasal cavity and paranasal sinuses are rare. About 2,000 people in the United States develop cancer of the nasal cavity and paranasal sinus each year. Men are about 50% more likely than women to get this cancer. Nearly 80% of the people who get this cancer are between the ages of 45 and 85 (41).

The esophagus is a muscular tube that connects the mouth to the stomach. It carries food and liquids to the stomach. It is about 10-13 inches long. In the United States, the American Cancer Society estimates that there will be about 14,550 new cases of this cancer in 2006. About 13,770 people will die of the disease. This cancer is 3 to 4 times more common among men than among women and 50% more common among African Americans than among whites.

There is no strong evidence in the scientific literature showing an association between exposure to arsenic, copper, lead, mercury, PCBs, and cancer of nasal sinuses or esophagus.

Evaluation of Noncancer Health Concerns at the Richmond Field Station

The RFS community also reported noncancer health concerns. These concerns included asthma, headaches, inability to focus, allergies, sinus problems, eye irritation, nose irritation, impaired sense of smell, cough, dry mouth, loss of voice, skin irritation, diarrhea, weight gain, numbness in hands and feet, chronic fatigue, cardiovascular disease, thyroid problems, bacterial meningitis, and fertility issues. This wide range of symptom complaints could have many possible explanations and all of the symptoms do occur in absence of an environmental exposure.

It is possible that exposure to dust contaminated with arsenic or mercury, or indoor formaldehyde exposure could contribute to one or many of these symptoms, but it is difficult to be certain. CDPH is unable to make definitive conclusions about the cause of these noncancer health concerns. The sampling data available provide an understanding of contaminant levels for limited time periods, and conditions may have varied on other days. Also, exposures to chemicals occur on a daily basis and it is not possible to assign health effects without considering everyday exposures. Formaldehyde, for example, is found in building materials, adhesives, pressed wood products, and some clothing and draperies (30).

In this section, we will provide a general background of some of the noncancer health concerns reported to CDPH. In addition, we will provide information regarding possible environmental factors that cause or exacerbate these noncancer health concerns. It is important to note that many studies analyzing the link between chemicals and health concerns do not characterize exposure levels. In other words, the dose is often unknown. Some studies involve exposing animals to high levels of chemicals and it is difficult to determine what dosage would exert the same effects in humans or if the same effects would occur in humans. Other studies involve populations exposed to varying amounts of chemicals in the past, such as through drinking water systems, and exposure levels are estimates. Some studies are case studies that describe unusual

circumstances such as individuals unknowingly eating contaminants in food, or suicide attempts involving high ingestion of a chemical. In this review, we will report dosages if they were available in the scientific literature.

Overall, the levels of arsenic, copper, lead, PCBs, and mercury detected at RFS are not expected to have caused the noncancer health concerns listed. It is possible that eye, nose and throat irritation, and respiratory effects could have occurred, if formaldehyde levels were consistently elevated.

Asthma

Asthma is a disorder of the airways in which they become inflamed, causing airflow in and out of the lungs to be restricted (55). This results in periodic attacks of wheezing, shortness of breath, chest tightness, and coughing. Asthma can be triggered by inhaling pet dander, dust mites, molds, pollens, and cockroach allergens. Respiratory infections, exercise, cold air, stress, food, drug allergies, and tobacco smoke can also trigger asthma attacks. Exposure to environmental pollutants also triggers asthma. Exposure to low levels of formaldehyde (100 $\mu g/m^3$) caused coughing among adult asthmatics who were later exposed to mite allergens (56). The level of formaldehyde measured in Building 163 (September 21, 2005) on one occasion could have caused similar effects in RFS workers who are asthmatic. Dust can also exacerbate asthma. No link has been established between asthma and arsenic, copper, lead, PCBs, or mercury.

Bacterial Meningitis

Meningitis is an infection that causes inflammation of the membranes covering the brain and spinal cord. Bacterial meningitis is caused by bacterial strains (such as streptococcus); about 17,500 cases of bacterial meningitis occur each year in the United States (57). Bacterial meningitis has not been linked with arsenic, copper, formaldehyde, lead, PCBs, or mercury.

Cardiovascular Concerns

Workers and former workers of RFS reported two types of cardiovascular concerns: heart disease and embolism. Heart disease is a term used to describe any disorder that affects the heart's ability to function normally (57). Heart disease is most commonly caused by the narrowing or blockage of the coronary arteries, a process that occurs over time. Other causes of heart disease are hypertension, abnormal function of the heart valves, abnormal electrical rhythm of the heart, and weakening of the heart's pumping function by infection or toxins (57). Lifestyle choices such as diet, physical activity, and smoking also affect one's chances of developing heart disease (58).

Embolism is the interruption of blood flow to an organ or body part due to one or more blood clots (59). This resulting lack of blood flow starves tissues of oxygen, resulting in tissue damage or death. Embolism can occur in the brain (causing a stroke) or in the heart (causing a heart attack), and occur less commonly in the kidneys, intestines, and eyes (59). Risk factors for

embolism include injury or damage to an artery wall, infection of the heart, and an increased amount of platelets in the blood (platelets are involved in blood clotting) (59).

Although several studies have looked at the possible relationship between increased levels of copper in the blood and risk of heart disease, it is unclear whether copper directly affects heart disease or is a marker of inflammation associated with heart disease (23). Data from animal studies suggests an increase in blood pressure in rats exposed to 14 mg copper carbonate/kg/day in the diet for 15 weeks (23). Exposure to copper at this high level is not expected to have occurred at RFS.

There is limited evidence that links arsenic exposure to ischemic heart disease after acute and long-term exposure, but exposure levels have not been well characterized (20, 60, 61). Intravenous doses of arsenic used in arsenic trioxide therapy for one type of leukemia showed effects on the cardiovascular system; intravenous doses were generally 0.15 mg arsenic /kg/day (20). Exposure to arsenic at this level is not expected to have occurred at RFS.

A number of occupational studies looking at PCB exposure and cardiovascular disease and blood pressure have produced inconsistent results. In animal studies, cardiovascular effects were not seen at levels ranging from approximately 5-10 mg/kg/day PCBs[7]. This implies that high doses of PCBs are not associated with cardiovascular problems.

Lead has been linked to cardiovascular effects in rats. Factors such as age, blood pressure, body mass, smoking, alcohol consumption, and family history of cardiovascular disease make human studies more complex and difficult to draw conclusions about cause-and-effect (22). An increase in blood pressure in women and men has been associated with a median blood lead of 2.3 ug/dL (22).

No link between formaldehyde exposure and heart disease appears in the scientific literature. One study investigating formaldehyde exposure levels in mice found no effects on the heart tissue of exposed mice (34).

Developmental Concerns for Children In Utero

In utero growth is a delicate process that is vulnerable to damage during the all trimesters of pregnancy (62). Damage can occur as a result of alcohol consumption, use of prescription and recreational drugs, infection, radiation (such as from X rays or radiation therapy), and nutritional deficiencies (62). Formaldehyde, lead, and copper are considered to have toxic effects on the fetus, based on findings from animals studies (20, 22, 23, 34, 52). However, an exposure dose that is associated with these effects in humans was not located. Studies of the effect of mercury exposure in utero focus primarily on exposure via ingestion of mercury contaminated fish (24). Animal studies have shown the possibility for developmental effects from exposure to PCBs at levels greater than 0.01 mg PCBs /kg/day. Exposure doses estimated for workers at RFS are much lower than levels shown to cause developmental effects (Appendix C, Table 9).

[7]The studies looked at various Aroclors.

Irritation of Eyes, Nose, and Sinuses

The sinuses are air-filled cavities within the bones of the face around the cheek, eyes, forehead, and near the middle of the skull (63). Each of the cavities has an opening that leads to the nose. Irritation of the sinuses can be caused by sinusitis—an inflammation caused by a viral, bacterial, or fungal infection (64). Each year, over 30 million adults and children get sinusitis. Some symptoms of sinusitis include nasal congestion and discharge, sore throat, cough, and the loss of the sense of smell (64).

Nosebleeds occur most commonly as a result of dryness, nose picking, injuries, allergies, or cocaine use, although the cause sometimes cannot be determined (65). The nose has many blood vessels close to the surface of the skin. These blood vessels help to warm and humidify the air that enters the lungs (65). Because of their proximity to the surface of the skin, these blood vessels are easy to injure (65).

The eyes are sensitive and can respond to irritation to any number of factors such as smoke, wind, dust, and fumes (66). Dry eyes are a common source of discomfort, and persons already suffering from dry eyes are more sensitive to irritants such as smoke or wind. Dry eyes are more likely to be experienced by adults over 40 (66).

It is possible that formaldehyde could have caused some of the symptoms reported by RFS workers (eyes, nose, and throat irritation), since elevated levels of formaldehyde were detected in Building 163, on one occasion. Exposure to airborne dust generated during Phase 1 and Phase 2 remedial activities could also result in irritation of the eyes, nose, and throat.

Irritation of Skin

Irritation of the skin can occur as a result of an allergic reaction or injury to the skin's surface (67). Detergents, soaps, cleaners, waxes, and chemicals can irritate the skin because they wear down the oily protective layer of the skin's surface (67). Restaurant, maintenance, and chemical workers may experience this condition more commonly because of their regular use of chemicals (67). Arsenic can cause skin lesions at chronic exposure doses ranging from 0.002 to 0.02 mg arsenic/kg/day (20). No effects on the skin have been seen in lower exposure levels ranging from 0.0004 to 0.01 mg arsenic/kg/day (20). Dermal effects have been observed via the inhalation route, but studies have not characterized the exposure concentrations required to produce dermal effects (20). Exposures estimated for RFS workers would not be expected to cause effects on the skin.

PCBs can cause skin conditions, such as acne and rashes, in people who were exposed to high levels of PCBs and dioxins in contaminated rice. Exposures estimated at RFS would not be expected to cause these effects (19).

Formaldehyde has also been linked with skin irritation (52). Studies have not found increased skin irritation symptoms for exposure to airborne formaldehyde at levels ranging from 490 $\mu g/m^3$-3,685 $\mu g/m^3$; however, subtle skin effects have been found among people who have increased sensitivity to formaldehyde (a condition called formaldehyde atopic eczema) in studies

of short-term exposure (34). During one sampling event (September 21, 2005), formaldehyde was measured at 410 µg/m^3 in Building 163, which is lower than the lowest level shown to cause skin irritation.

Skin reactions to inhalation of metallic mercury vapor (inorganic mercury) include skin rashes and heavy perspiration (24). Exposures estimated in this PHA would not be expected to cause these effects. Most mercury at RFS is likely inorganic mercury, though it is possible methylization of mercury to occur in sediment. No studies were located linking exposure to organic mercury to dermal effects.

Numbness in Feet and Hands

Numbness and tingling can be experienced in any part of the body, but are usually felt in the hands, feet, arms, or legs (68). There are many causes of numbness and tingling of the extremities, including remaining in the same position (sitting or standing) for a long period of time, injuring or pressuring a nerve, lack of blood supply to an area of the body, carpal tunnel syndrome, lack of vitamin B12, some medications, radiation therapy, and diabetes and other medical conditions (68). Toxic effects of lead, arsenic, and mercury on the nerves include numbness in the feet and hands, although exposure doses are not characterized (24, 52). In one study, neurological symptoms, including numbness, weakness, and neuralgia of limbs, were seen from exposure to high levels of PCBs (19). However, the findings from the studies of these groups cannot be attributed solely to exposure to PCBs since the victims also were exposed to dioxins and other chlorinated chemicals (19).

Diminished Mental Capacities (difficulty concentrating, fatigue)

Diminished mental capacities (such as difficulty concentrating) can be a result of a variety of factors. For example insomnia, depression, generalized anxiety disorder, chronic fatigue syndrome, poor nutrition, and inflammation of the thyroid can cause poor concentration (69-74). The Collaborative on Health and the Environment cites “limited” evidence of a link between arsenic exposure and cognitive impairment (52). Effects such as lethargy, mental confusion, hallucinations, seizure, and coma occurred in humans after exposure to over 2 mg arsenic/kg/day of inorganic arsenic via the oral route (20). Exposure to arsenic at RFS would not result in levels this high. Headache and fatigue have been reported at lower levels, between 0.004 and 0.006 mg arsenic/kg/day (20). Exposure doses estimated for to RFS maintenance workers range from 0.00023-0.00028 mg arsenic/kg/day, which are lower than the levels associated with headache and fatigue.

Lead is also associated with cognitive impairment, mostly I.Q. Children are at greater risk than adults because they are more likely to have contact with contaminated surfaces (by crawling on the floor or putting objects in their mouths) (22). Children also absorb a larger fraction of ingested lead than adults (22). Lead poisoning in adults can cause memory and concentration problems (75). Blood lead levels over 40 µg/dL are associated with neurobehavioral effects in adults (decreased cognitive function, verbal memory and learning, visual memory, manual dexterity) but exposure doses are not characterized (22). Blood lead levels estimated for RFS

workers are well below 40 µg/dL and thus would not be expected to cause neurobehavioral effects.

Some studies in workers suggest that exposure to PCBs may cause depression and chronic fatigue, but it is not known the exposure levels at which these effects occur (19).

Fertility Concerns

Infertility is the inability of a couple to become pregnant after 12 months of unprotected intercourse, either because the woman is unable to become pregnant or the man is unable to impregnate the woman (76). There is no single cause for infertility. Some causes are physical, such as pelvic infection, poor nutrition, hormone imbalance, and scarring of the uterine walls and fallopian tubes due to sexually transmitted disease (76). Other causes may relate to age, stress, smoking, and use of drugs or alcohol. For example, the heavy use of marijuana and some prescription drugs (cimetidine, spironolactone, and nitrofurantoin) affect sperm count (76).

Exposure to environmental toxins such as formaldehyde and lead has been linked to reduced fertility (52). One study with female wood workers found that exposure to formaldehyde was associated with delayed conception (77). Some studies suggest that lead can affect both female and male fertility. Alterations in sperm and decreased fertility have been observed in men whose blood lead level was in the range of 30-40 µg/dL (22). Lead levels in women's ovarian follicles were suspected of adversely affecting female reproduction, although exposure levels were not characterized (78). On the other hand, several studies have found no significant association between lead and pre-term delivery in women or alterations in sperm count in men (22). Lead exposure from RFS would result in blood lead levels (BLLs) below 30 µg/dL.

Limited information in humans does not suggest a link between PCB exposure and male reproductivity. Reproductive effects have been seen in women from exposure in the workplace and from eating contaminated fish. Reproductive impairment has been seen in animal studies (19). Further studies looking at a variety of reproductive outcomes are needed to understand the reproductive toxicity of PCBs.

Thyroid Problems

Thyroid conditions can involve either a change in the pace of the thyroid gland (causing it to be overactive or underactive) or thyroid nodules, which are small lumps (79). The term hyperthyroidism describes the condition of having an overactive thyroid gland. Hyperthyroidism speeds up the body's metabolism, resulting in the function of many body systems speeding up and producing too much heat. Hypothyroidism describes the condition of having an underactive thyroid gland. Hypothyroidism results in low levels of thyroid hormone, and most body functions slow down. With hypothyroidism, the body consumes less oxygen and produces less heat. Thyroid nodules occur in about 5% of the population, and it is estimated that almost half of the general population has thyroid nodules but many people are not aware of them until they grow in size (79). Thyroid nodules occur as a result of an enlargement of a collection of thyroid cells or because fluid collects and forms a cyst. Thyroid nodules can appear individually or in greater numbers (79). No reports were found describing the effects of arsenic or copper on the

thyroid (52). Some animal studies found no effects of formaldehyde on the thyroid (34). Changes in thyroid hormone levels occurred in workers who had blood lead levels greater than 40 µg/dL (22). Lead exposure estimate for RFS is well below 40 µg/dL.

Studies in animals, including rodents and nonhuman primates, provide strong evidence of thyroid hormone involvement in PCB toxicity. The levels of exposure in these studies range from 0.1 mg PCBs/kg/day (less serious effects) to 12.5 mg PCBs/kg/day (serious effects); these doses associated with PCB toxicity are higher than exposures estimated at RFS (Appendix C, Table 9) (19).

Other Health Concerns

RFS workers and former workers reported other health concerns such as abdominal pain, headaches and migraines, dry mouth, loss of voice, weight gain, stomachache, and diarrhea. These health concerns are common and occur as a result of a variety of reasons. Because of their ubiquitous nature, we are unable to assess their connection with exposures from RFS.

Toxicity by Chemical of Concern

To better understand the health concerns, we will describe some of the primary noncancer symptoms/health effects associated with COCs (arsenic, copper, formaldehyde, lead, PCBs, and mercury).

Arsenic

Arsenic is a naturally-occurring element that is normally found combined with other elements. Arsenic toxicity varies depending upon its form. The soluble inorganic forms are well absorbed from the digestive tract and distributed widely throughout the body. (Inorganic arsenic is most likely form of arsenic at RFS.) Arsenic is cleared rapidly from the blood (20). Most arsenic that is absorbed from the gastrointestinal tract and lungs is excreted in the urine within a couple of days (20). Although arsenic may concentrate in small amounts in the liver, kidney, lung, spleen, aorta, and upper gastrointestinal tract, it is also rapidly cleared from these tissues once exposure ceases. Arsenic that remains and accumulates in the body is stored mainly in the skin and hair (20). People who may show increased sensitivity to arsenic include those on protein-poor diets or those with choline (a B vitamin) deficiency. Inorganic arsenic is detoxified in humans by liver enzymes. Those individuals with low liver enzyme activity or liver damage such as alcoholic- or viral-induced cirrhosis may be more sensitive to the effects of arsenic than are people with normal liver enzyme activity (20). Studies of the chronic oral effects of arsenic show that although some people can ingest up to 150 µg arsenic/kg/day without noticeable effects, doses as low as 20 to 60 µg arsenic/kg/day may result in one or more signs of arsenic toxicity in more sensitive individuals. Adverse health effects from arsenic exposure include: digestive tract irritation, disturbances of the blood and nervous systems, skin and blood vessel injuries, and liver or kidney injury. The most sensitive effects are the changes in pigmentation of the skin and the appearance of calluses. The ATSDR MRL (0.3 µg arsenic/kg/day) is based on these effects.

CDPH calculated an exposure dose for a person (worker/teenager) who incidentally ingests and has dermal contact with on-site soil and marsh sediments using the maximum levels of arsenic detected in surface soil and near surface soil; exposure doses do not exceed the MRL, thus noncancer adverse health effects are not likely to have occurred or be occurring (Appendix C, Tables 5, 7, and 9). Some uncertainty exists in estimating the amount of worker exposure, since contaminant concentrations may have been higher or lower in areas not characterized at RFS. For instance, workers may have also received additional exposures from breathing arsenic-contaminated dust/particulate. CDPH did not evaluate inhalation exposure to contaminated dust/particulate due to uncertainties in the estimation.

Cadmium

Cadmium is a natural-occurring metal found in the earth's crust. The average level of cadmium in U.S. soil is about 250 ppb (0.25 ppm). The main source of cadmium exposure is from cigarette smoke and food (25). The average person eats about 30 µg of cadmium in food each day, but only 1-3 µg of cadmium is absorbed in the body each day. Cadmium from cigarette smoke is thought to be of greater health concern than cadmium taken in from food. There are no known benefits from cadmium intake. Breathing very high levels cadmium can cause severe lung damage and death. At lower levels, over long periods of time, breathing cadmium can damage the lung, kidneys, and bones. In animal studies, breathing cadmium has been shown to affect the liver and immune system (25). Lung cancer has been associated with inhalation of cadmium in some animal studies. It remains unclear whether breathing cadmium causes lung cancer in people. Eating or drinking cadmium over long periods of time can lead to cadmium buildup in the kidneys (25). Eating or drinking cadmium has not been shown to cause cancer, but more research is needed before definitive conclusions can be reached. Dermal (skin) contact with cadmium is not known to cause adverse health effects in people or animals (25).

CDPH calculated exposure doses for children and adults who engage in activities in the West Stege Marsh and RFS maintenance workers who are exposed to cadmium in soil. Exposure doses do not exceed the MRL, thus noncancer adverse health effects are not likely to have occurred or be occurring (Appendix C, Tables 5, 7, and 9). Some uncertainty exists in estimating the amount of worker exposure, since contaminant concentrations may have been higher or lower in areas not characterized at RFS. Workers may have also received additional exposures from breathing cadmium-contaminated dust.

Copper

Copper is a natural-occurring metal found in soil, rocks, water, and air. Copper is an essential nutrient for plants and animals, including people. The greatest potential source of copper exposure is through drinking water, especially in water that is first drawn in the morning after sitting in copper piping and brass faucets overnight (23). Copper is commonly use in agriculture and other industries.

Long-term exposure to copper dust can irritate your nose, mouth, and eyes, and cause headaches, dizziness. Ingesting high levels of copper (91 µg copper/kg/day) can cause nausea, vomiting, and

diarrhea (gastrointestinal effects) (23). At very high levels, copper can cause liver and kidney damage. It is not known whether copper causes cancer (23).

The exposure levels estimated for an RFS worker are below levels shown to cause gastrointestinal effects (Appendix C, Table 9).

Formaldehyde

Formaldehyde is a colorless, flammable gas at room temperature. Formaldehyde is used in many industries. It is used in the production of fertilizer, paper, plywood, and urea-formaldehyde resins. Formaldehyde is found in many products used every day around the house, such as antiseptics, medicines, cosmetics, dish-washing liquids, fabric softeners, shoe-care agents, carpet cleaners, glues and adhesives, lacquers, paper, plastics, and some types of wood products (24). Most formaldehyde in the air also breaks down during the day. The breakdown products of formaldehyde in air include formic acid and carbon monoxide. Formaldehyde does not seem to build up in plants and animals, and although formaldehyde is found in small amounts in some food. It has a pungent, distinct odor.

The most common symptoms from exposure to formaldehyde include irritation of the eyes, nose, and throat, along with increased tearing. These symptoms occur at air concentrations of about 490-3700 $\mu g/m^3$ (34). Formaldehyde can also cause or exacerbate allergic asthma (56). Workers studies have shown increased nasal (nose) and throat cancer.

It is possible that workers in Building 163 could have experienced irritation of the eyes, nose, and throat based on September 21, 2005, when formaldehyde was measured at levels exceeding health-based standards. It is important to note that this conclusion is based on a single reading measured on September 21, 2005.

Lead

Lead is a natural-occurring metal found in all parts of the environment. Most of the lead found in the environment is due to human activities including burning fossil fuels, mining, and manufacturing.

The nervous system is the most sensitive target of lead exposure. Children are the most sensitive to the neurological effects of lead because their brains and nervous systems are still developing. Lead also affects renal function, blood cells, and the metabolism of vitamin D and calcium (17). Lead can also cause hypertension, reproductive toxicity, and developmental effects, in utero.

Studies on reproductive toxicity have shown increased miscarriages and stillbirths in women working in the lead industry at the turn of the century, when exposure levels were very high (17). The effect of low-level lead exposures on pregnancy outcomes is not clear, as studies have shown inconsistent findings (22). Exposure mitigation measures are recommended for pregnant women with BLLs of 10 μg/dL.

The lowest level at which lead has an adverse effect on the kidney remains unknown. Most documented renal effects for occupational workers have been observed in acute high-dose exposures and high-to-moderate chronic exposures (BLL greater than 60 μg/dL) (22). The estimated BLLs in each pathway evaluated were less than 10 μg/dL for youth and less than 25 μg/dL for adults and RFS workers. Thus, adverse kidney effects would not be expected.

Studies on developmental effects, including congenital abnormalities, and post birth effects on growth or neurologic development indicate that lead, that readily crosses the placenta, adversely affects fetus viability as well as fetal and early childhood development (22). There may be an increased risk of reduced birth weight and premature birth from prenatal exposure to low lead levels (e.g., maternal BLLs of 14 μg/dL) (22). The estimated BLL (7.6 μg/dL) for maintenance RFS workers maintenance is lower than levels shown to cause developmental effects.

It is unlikely that the average worker (not maintenance workers) at the RFS are being exposed to lead-contaminated soil at levels that would result in elevated BLLs. The estimated BLL (7.6 μg/dL) for maintenance workers was less the level at which exposure reduction actions are recommended (10 μg/dL for pregnant woman and 25 μg/dL for all other adults).

Mercury

Mercury is a natural-occurring metal in the environment. Metallic or elemental mercury is the main form of mercury released into the air by natural processes. Inorganic or elemental is probably the predominant form of mercury in soil at RFS, though sampling analyses to confirm this assertion were not available at the time of this writing. In the environment inorganic mercury can be methylated by microorganisms to form methylmercury (organic). It is possible for the mercury to be methylated in sediment from the Western Stege Marsh. Methylmercury will accumulate in the tissues of organisms. The most common ways people are exposed to mercury is through eating fish that may contain some methylmercury in their tissues and from the release of elemental mercury from dental fillings.

Inhalation of sufficient levels (below 1,000 $\mu g/m^3$) of metallic mercury vapor has been associated with systemic toxicity (kidney and central nervous system), respiratory, cardiovascular, and gastrointestinal effects in humans and animals (24). Commonly reported kidney effects from mercury exposure include blood in the urine and decreased urine output (24). Neurological symptoms could include weakness, numbness, tremors, and changes in balance (24). In animal studies, reproductive effects (subtle behavioral changes) were seen from exposure to metallic mercury at 50 $\mu g/m^3$. Airborne levels measured at RFS are well below levels shown to cause adverse health effects.

It is not likely that low-level mercury exposure in dust resulted in health effects reported by RFS workers. If mercury-related symptoms are suspected, it is possible to measure mercury in the blood and urine, near the time of exposure. However, determining the source of the mercury would be difficult, because mercury is a common contaminant found in blood (80).

CDPH calculated an exposure dose for a person (worker/teenager) who incidentally ingests and has dermal contact with on-site soil (inorganic) and marsh sediments (assumed to be

methylmercury) using the maximum levels of mercury detected in soil and sediment; exposure doses do not exceed MRL (Appendix C, Tables 5, 7, and 9)

PCBs

PCBs are complex mixtures of synthetic organic chemicals that vary in their degree of toxicity. PCBs stopped being manufactured in the United States in 1977, due to evidence that they accumulate and persist in the environment and can cause toxic effects. Small amounts of PCBs can be found in almost all outdoor and indoor air, soil, sediments, surface water, and animals. Some studies in workers suggest that exposure to PCBs causes irritation of the nose, lungs, gastrointestinal discomfort, changes in the blood and liver, depression, and chronic fatigue. Neurobehavioral and immunological changes in children have also been associate with exposure to PCBs. Animal studies have indicated that breathing high levels of PCBs for several months can result in liver and kidney damage (19). Other effects of PCBs in animals include changes in the immune system, behavioral alterations, and impaired reproduction. PCBs are not known to cause birth defects. In worker studies, PCBs were associated with certain types of cancer such as cancer of the liver and biliary tract (19).

PCBs were found in on-site soil in some areas of RFS. The exposure levels estimated for an RFS worker are below levels shown to cause noncancer adverse health effects. However, inhalation of contaminated-particulates could have added to a workers overall exposure.

Zinc

Zinc is one of the most common, naturally-occurring elements (metal) found in the environment. Zinc is found in soil, air, water, and is present in all food. It is an essential element needed by the body (81). The average person ingests about 5.2 -16.2 mg of zinc per day from dietary sources. Breathing high levels of zinc dust or fumes (generally associated with welding or smelting occupations) can develop a reversible disease known as metal fume fever. Not much is know about the long-term effects of breathing zinc dust or fumes. Ingesting high levels of zinc (10-15 times greater than the Recommended Daily Allowance of 11 mg/day) can result in stomach cramps, nausea, and vomiting (81). Long-term ingestion (several months) of high levels of zinc can damage the pancreas, cause anemia and decrease high-density lipoprotein (HDL) cholesterol levels. Certain zinc compounds have been shown to cause skin irritation in animal studies. It is likely that people would experience skin irritation as well. There is insufficient information to know whether zinc causes cancer (81).

CDPH calculated exposure doses for children and adults who engage in activities in the West Stege Marsh who are exposed to zinc in surface water and sediment. Exposure doses do not exceed the MRL, thus noncancer adverse health effects are not likely to have occurred or be occurring (Appendix C, Tables 5 and 7).

The following are general questions asked during CDPH discussions with RFS workers.

- What are the effects of the combination of chemicals?

Data on the health effects from exposure to multiple chemicals (chemical mixtures) are very limited. The effects of multiple chemical exposures can be additive, synergistic (combined toxic effects of two or more chemicals are greater than each chemical alone), or antagonistic (two chemicals interfere with each other's actions, leading to a less toxic compound). Inhibition effects occur when a chemical that does not have a toxic effect on a certain organ system decreases the apparent effect of a second chemical on that organ system.

- What is the effect of exposure to chemicals during pregnancy for the fetus and development of the child after birth?

The effect of chemical exposure to a fetus depends on the timing of exposure during pregnancy and the amount (dose) of exposure. Depending on the chemical, exposure can cause loss of fetus, abnormal skeletal growth, functional changes such as lesser thyroid hormone, and irreversible neurodevelopmental effects. Studies have shown developmental effects in children exposed to lead and mercury.

- Does the presence of chemicals in the environment decrease immune function because the immune system might be "distracted" dealing with the chemicals, and thus create a susceptibility to develop illnesses that run in one's family (such as thyroid problems)?

There is a great deal of debate on this topic within the scientific community. Animal studies clearly show exposure to chemical agents can suppress the immune system, which can result in disease. However, data on whether this is true for humans is much more limited (82).

Studies have shown that chemical exposure can affect immunity in three major ways:
by causing hypersensitivity reactions, including allergy, which can be harmful to organs and tissue and; autoimmunity, in which the immune cells attack themselves; or by immunosuppression—a reduction in immune response and activities of the immune system (82).

Some researchers who study immunotoxicology, specifically, adverse effects on the immune system as a result of exposure to environmental chemicals, contend that certain chemicals can affect immunity, increasing a person's susceptibility to disease. Age, genetics, preexisting disease, lifestyle, diet, drugs, stress, are all factors that play a role in immune function. These factors may compound the effects of chemical exposure by further compromising immune function and increasing the chance for disease. There is thought that some immunologic disorders appear only after toxic exposure from the environment invokes a previously undetected genetic condition, while other disorders appear under ordinary environmental conditions (82).

- Can people walk outside safely on RFS grounds?

Yes, it is safe for people to walk on RFS grounds. The main exposure concern is it to RFS maintenance workers who may dig and come into contact with contaminated soil. The primary route of exposure (way the contaminant gets into the body) is through incidental ingestion. Simply walking on the RFS grounds does not pose a health risk from exposure to contaminants present in soil.

- Is there a risk from walking/biking along the Bay Trail?

No, there is no health risk to Bay Trail users from exposure to contaminants at RFS. It is possible that Bay Trail users could have been exposed to contaminated dust generated during past remedial activities (cleanup and excavation work). DTSC will ensure that future remedial work will be conducted using adequate dust control measures.

- Is there radioactive waste at RFS?

DTSC is investigating allegations that drums containing radioactive waste were dumped in the bay. DTSC is also investigating potential radioactive contamination at the neighboring Zeneca site. At this time there is no evidence of radioactive contamination at the RFS site.

- Are there health risks from the power line (EMF) near RFS?

Exposure risks from EMFs are out of the scope of this health assessment. Information about EMFs can be obtained online at http://www.niehs.nih.gov/emfrapid/ and at http://www.dhs.ca.gov/ehib/emf/general.html.

Health Outcome Data

Health outcome data (HOD) record certain health conditions that occur in populations. These data can provide information on the general health of communities living near a hazardous waste site. They also can provide information on patterns of specified health conditions. Some examples of health outcome databases are the California Cancer Registry, birth defects registries, and vital statistics. Information from local hospitals and other health care providers also can be used to investigate patterns of disease in a specific population. These data are recorded based on the geographic area where a person lives, not where they work. A HOD review would not provide information reflective of the work force at RFS or visitors or people restoring the marsh. Thus, a review of HOD was not conducted for this site.

Children's Health Considerations

CDPH and ATSDR recognize that, in communities with contaminated water, soil, air, or food (or all of these combined, depending on the substance and the exposure situation), infants and children can be more sensitive than adults to chemical exposures. This sensitivity results from several factors: 1) children might have higher exposures to environmental toxins than adults because, pound for pound of body weight, children drink more water, eat more food, and breathe more air than adults; 2) children play indoors and outdoors close to the ground, which increases their exposure to toxins in dust, soil, surface water, and ambient air; 3) children have a tendency to put their hands in their mouths, thus potentially ingesting contaminated soil particles at higher rates than adults; some children even exhibit an abnormal behavior trait known as "pica," that causes them to ingest non-food items, such as soil; 4) children's bodies are rapidly growing and developing, thus they can sustain permanent damage if toxic exposures occur during critical growth stages; and 5) children and teenagers more readily than adults can disregard no trespassing signs and wander onto restricted property. CDPH considered children in the

pathways evaluated in this PHA.

Conclusions

CDPH evaluated five completed exposure pathways (past, current, and future) to contaminants at RFS, using available environmental data collected from the site. CDPH classifies each completed exposure pathways based on the pathways' potential for posing a health hazard.

No apparent public health hazard

- Past exposure to airborne mercury during remedial work.

The available data do not indicate that people were exposed to levels of airborne mercury between August and September 2003 that would be expected to cause adverse health effects.

Indeterminate health hazard

- Current and future exposure to adults or youth from restoring the West Stege Marsh in areas that have been excavated.

The available data do not indicate that people were/are being exposed to levels of metals, pesticides or PCBs in the West Stege Marsh that would not be expected to cause adverse health effects. However, there is a potential for elevated levels of natural occurring radionuclides associated with historic operations at the adjacent Zeneca site to have migrated into the West Stege Marsh. In addition, groundwater monitoring is needed to address the concern whether other site-related contaminants from the adjacent Zeneca site are migrating into the West Stege Marsh. Until these activities are completed, and a determination is made whether there is a need for further characterization of the West Stege Marsh, these pathways are classified as posing an indeterminate health hazard.

- Current and future exposure to RFS employees from contaminants in indoor air as a result of vapor intrusion.

Limited indoor air sampling indicates a potential health risk from exposure to formaldehyde in indoor air that occurred on between September and October 2005. These data are insufficient to draw conclusions about the source of formaldehyde in indoor air or the potential impact of future exposure.

Public health hazard

- Past, current, and future exposure to children/teenagers who regularly play in the West Stege Marsh.

CDPH identified a public health hazard for children/teenagers who regularly play in the West Stege Marsh, from exposure to metals and PCBs in surface water and/or sediment. The most sensitive (primary) noncancer endpoints associated with COCs include skin effects (arsenic),

renal effects (cadmium), neurodevelopmental (methylmercury), gastrointestinal symptoms (copper), immune effects (PCBs), and decreases in erythrocyte copper, zinc-superoxide dismutase (ESOD) activity (zinc). COCs associated with a theoretical increased cancer risk are arsenic (skin, liver, bladder, and lung) and PCBs (liver, biliary). It is important to note that this conclusion is based on conservative assumptions meant to identify the possibility for exposures of health concern, so that steps can be taken to mitigate or prevent these exposures from occurring. Actual exposures to children/teenagers are likely much less. Access to the marsh should remain restricted.

- Past, current, and future exposure to RFS maintenance workers.

CDPH identified a public health hazard for RFS maintenance workers who regularly work in soil containing the highest levels of metals and PCBs in non-excavated areas of RFS. The primary noncancer endpoints associated with COCs include skin effects (arsenic), immune changes (PCBs), renal effects (cadmium, inorganic mercury), and gastrointestinal symptoms (copper). COCs associated with an increased cancer risk are arsenic (skin, liver, bladder, and lung) and PCBs (liver, biliary). While this conclusion is based on conservative assumptions (actual ingestion and dermal exposure are likely much less), it does not include potential exposure from inhalation of contaminated soil particulate, which could be a significant route of exposure, adding to a worker's overall risk. Inhalation exposure can be mitigated if workers wear proper respiratory protection while working in RFS soil.

CDPH has conducted a number of outreach activities at RFS, in an effort to collect and understand the health concerns that RFS employees believe are related to contamination at RFS. The majority of the health concerns expressed by workers cannot be clearly linked to chemical exposures at the site, with the exception of eye, nose and throat irritation, and mild respiratory effects that may have occurred from exposure to formaldehyde and airborne dust. A number health and safety concerns expressed to CDPH has resulted in recommendations for worker training and better communication (maps, reports, etc.) by UC management to RFS workers.

Recommendations

1. CDPH/ATSDR recommend that future soil disturbing/dust generating activities be monitored for air quality along the perimeter of the site to ensure safe air quality for workers, residents, and other people in the area.

2. CDPH/ATSDR recommend UC conduct additional characterization of on-site groundwater at the east and northeast side of RFS to better understand the potential for vapor intrusion to be affecting indoor air in buildings in that area.

3. CDPH/ATSDR recommend UC annually sample the sediment and unfiltered water in the RFS marsh to identify whether contaminants are migrating from the non-remediated areas of the marsh, uplands, and adjacent Zeneca site. The sampling should continue until the site has been fully characterized and characterized and remediation completed in areas that could impact the marsh.

4. CDPH/ATSDR recommend UC analyze for radionuclides associated with historic activities at the Zeneca site (former Stauffer Chemical), in on-site soil, groundwater, and sediment from the West Stege Marsh, if radionuclide contamination is identified during investigations at the Zeneca site.

5. CDPH/ATSDR recommend UC conduct additional indoor air sampling in Buildings 163 and 175 to identify whether formaldehyde is elevated above levels typical of indoor air. Results of sampling will determine the need for further sampling or investigation.

6. CDPH/ATSDR recommend UC conduct additional characterization of on-site soil throughout RFS to identify other areas where potential contamination may exist, and that chemicals used in research activities at RFS, as well as known contaminants from historic uses of RFS and the adjacent Zeneca site be analyzed. Additional characterization of soil in the area where the Forest Products Laboratory is located is needed, and should include analyses of pentachlorophenol and chlorophenol byproducts.

7. CDPH/ATSDR recommend UC provide all of RFS staff access to up to date maps showing locations of current and historic structures and soil sampling locations, along with the associated level of contamination.

8. CDPH/ATSDR recommend UC offer Hazardous Waste Operations and Emergency Response (HAZWOPER) training to workers whose work may involve handling or digging in soils on the RFS site.

9. CDPH/ATSDR recommend UC train workers annually in how to identify cinders and what actions to take if such material is identified.

Public Health Action Plan

The Public Health Action Plan (PHAP) for this site contains a description of actions taken, to be taken, or under consideration by ATSDR and CDPH or others at and near the site. The purpose of the PHAP is to ensure that this PHA not only identifies public health hazards, but also provides a plan of action designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment. The first section of the PHAP contains a description of actions completed. The second section is a list of additional public health actions that are planned for the future.

Actions Completed

- CDPH/ATSDR worked with the Occupational Health Branch of CDPH to determine the appropriate mechanisms to reach workers and to prepare relevant health and safety information and referrals (May-September 2005).

- CDPH/ATSDR gathered community (RFS employees) concerns through meeting with workers at RFS and by conducting two public availability sessions (October 2005).

- CDPH/ATSDR and the Contra Costa County Health Services Department released a Provisional Joint Health Statement, providing an evaluation of current exposure from contaminants at RFS and adjacent Zeneca sites (June 2005; update in February 2006).
- CDPH/ATSDR recommended that RFS West Stege Marsh be fenced and posted to eliminate exposure to contaminants remaining in the marsh (action completed in December 2006).

Ongoing Actions

- CDPH/ATSDR will continue to provide health outreach and education to the community/RFS workers and recommend that health education activities be tailored to meet the workers needs.

Actions Planned

- CDPH will disseminate information summarizing the findings of this comprehensive PHA and hold a public meeting to discuss the results.

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Certification

This public health assessment, Evaluation of Exposure to Contaminants at the University of California Richmond Field Station, Richmond, California, was prepared by the California Department of Health Services under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the public health assessment was begun. Editorial review was conducted by the cooperative agreement partner.

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The Division of Public Health Assessment and Consultation, ATSDR, has reviewed this health consultation and concurs with the findings.

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Appendix A. Glossary of Terms

Absorption
How a chemical enters a person's blood after the chemical has been swallowed, has come into contact with the skin, or has been breathed in.

Acute Exposure
Contact with a chemical that happens once or only for a limited period of time. ATSDR defines acute exposures as those that might last up to 14 days.

Adverse Health Effect
A change in body function or the structures of cells that can lead to disease or health problems.

ATSDR
The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency with headquarters in Atlanta, Georgia, and ten regional offices in the U.S. ATSDR's mission is to serve the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and diseases related to toxic substances. ATSDR is not a regulatory agency, unlike the U.S. Environmental Protection Agency (EPA), which is the federal agency that develops and enforces environmental laws to protect the environment and human health.

Background Level
An average or expected amount of a chemical in a specific environment or, amounts of chemicals that occur naturally in a specific environment.

Cancer Risk
The potential for exposure to a contaminant to cause cancer in an individual or population is evaluated by estimating the probability of an individual developing cancer over a lifetime as the result of the exposure. This approach is based on the assumption that there are no absolutely "safe" toxicity values for carcinogens. U.S. EPA and the California EPA have developed cancer slope factors and inhalation unity risk factors for many carcinogens. A slope factor is an estimate of a chemical's carcinogenic potency, or potential, for causing cancer.

If adequate information about the level of exposure, frequency of exposure, and length of exposure to a particular carcinogen is available, an estimate of excess cancer risk associated with the exposure can be calculated using the slope factor for that carcinogen. Specifically, to obtain risk estimates, the estimated, chronic exposure dose (which is averaged over a lifetime or 70 years) is multiplied by the slope factor for that carcinogen.

Cancer risk is the theoretical chance of getting cancer. In California, 41.5% of women and 45.4% of men (about 43% combined) will be diagnosed with cancer in their lifetime (12). This is referred to as the "background cancer risk." The term "excess cancer risk" represents the risk above and beyond the "background cancer risk." A "one-in-a-million" excess cancer risk from a given exposure to a contaminant means that if one million people are chronically exposed to a carcinogen at a certain level, over a lifetime, then one cancer above the background risk may

appear in those million persons from that particular exposure. For example, in a million people, it is expected that approximately 430,000 individuals will be diagnosed with cancer from a variety of causes. If the entire population was exposed to the carcinogen at a level associated with a one-in-a-million cancer risk, 430,001 people may get cancer, instead of the expected 430,000. Cancer risk numbers are a quantitative or numerical way to describe a biological process (development of cancer). In order to take into account the uncertainties in the science, the risk numbers used are plausible upper limits of the actual risk, based on conservative assumptions.

Chronic Exposure
A contact with a substance or chemical that happens over a long period of time. ATSDR considers exposures of more than 1 year to be chronic.

Completed Exposure Pathway
See Exposure Pathway.

Concern
A belief or worry that chemicals in the environment might cause harm to people.

Concentration
How much or the amount of a substance present in a certain amount of soil, water, air, or food.

Contaminant
See Environmental Contaminant.

CREG (ATSDR Cancer Risk Evaluation Guide for 1 in 1,000,000 increased cancer risk)
Like EMEGs, water CREGs are derived for potable water used in homes, including water used for drinking, cooking, and food preparation. Soil CREGs apply only to soil that is ingested.

A theoretical increased cancer risk is calculated by multiplying the dose and the CSF. When developing CREG, the target risk level (10^{-6}), which represents a theoretical risk of one excess cancer case in a population of one million, and the CSF are known. The calculation seeks to find the substance concentration and dose associated with this target risk level.

To derive water and soil CREGs, ATSDR uses CSFs developed by the U.S. EPA and reported in the Integrated Risk Information System (IRIS). The IRIS summaries, available at http://www.epa.gov/iris/, provide detailed information about the derivation and basis of the CSFs for individual substances. ATSDR derives CREGs for lifetime exposures, and therefore uses exposure parameters that represent exposures as an adult. An adult is assumed to ingest 2 L/day of water and weigh 70 kg. For soil ingestion, ATSDR assumes a soil ingestion rate of 100 mg/day, for a lifetime (70 years) of exposure.

Dermal Contact
A chemical getting onto your skin. (See Route of Exposure.)

Dose
The amount of a substance to which a person may be exposed, usually on a daily basis. Dose is

often explained as “amount of substance(s) per body weight per day.”

Dose/Response
The relationship between the amount of exposure (dose) and the change in body function or health that result.

Duration
The amount of time (days, months, and years) that a person is exposed to a chemical.

EMEG (ATSDR Environmental Media Evaluation Guide)
Water EMEGs are derived for potable water used in homes. Potable water includes water used for drinking, cooking, and food preparation. Exposures to substances that volatilize from potable water and are inhaled, such as volatile organic compounds (VOCs) released during showering, are *not* considered when deriving EMEGs.

To derive the water EMEGs, ATSDR uses the chronic oral MRLs from the Toxicological Profiles, available at http://www.atsdr.cdc.gov/toxpro2.html. Ideally, the MRL is based on an experiment in which the chemical was administered in water. However, in the absence of such data, an MRL based on an experiment in which the chemical was administered by gavage or in food may have been used. The Toxicological Profiles for individual substances provide detailed information about the MRL and the experiment on which it was based.

Children are usually assumed to constitute the most sensitive segment of the population for water ingestion because their ingestion rate per unit of body weight is greater than the adults' rate. An EMEG for a child is calculated assuming a daily water ingestion rate of 1 liter per day (L/day) for a 10-kilogram (kg) child. For adults, a water EMEG is calculated assuming a daily water ingestion rate of 2 liters per day and a body weight of 70 kg.

Soil EMEGS: ATSDR uses the chronic oral MRLs from its Toxicological Profiles. Many chemicals bind tightly to organic matter or silicates in the soil. Therefore, the bioavailability of a chemical is dependent on the media in which it is administered. Ideally, an MRL for deriving a soil EMEG should be based on an experiment in which the chemical was administered in soil. However, data from this type of study is seldom available. Therefore, often ATSDR derives soil EMEGs from MRLs based on studies in which the chemical was administered in drinking water, food, or by gavage using oil or water as the vehicle. The Toxicological Profiles for individual substances provide detailed information about the MRL and the experiment on which it was based.

Children are usually assumed to be the most highly exposed segment of the population because their soil ingestion rate is greater than adults' rate. Experimental studies have reported soil ingestion rates for children ranging from approximately 40 to 270 milligrams per day (mg/day), with 100 mg/day representing the best estimate of the average intake rate (EPA 1997). ATSDR calculates an EMEG for a child using a daily soil ingestion rate of 200 mg/day for a 10-kg child.

Environmental Contaminant
A substance (chemical) that gets into a system (person, animal, or environment) in amounts higher than that found in Background Level, or what would be expected.

Environmental Media
Usually refers to the air, water, and soil in which chemicals of interest are found. Sometimes refers to the plants and animals that are eaten by humans. Environmental Media is the second part of an Exposure Pathway.

Exposure
Coming into contact with a chemical substance (for the three ways people can come in contact with substances, see Route of Exposure).

Exposure Assessment
The process of finding the ways people come in contact with chemicals, how often, and how long they come in contact with chemicals, and the amounts of chemicals with which they come in contact.

Exposure Pathway
A description of the way that a chemical moves from its source (where it began), to where, and how people can come into contact with (or get exposed to) the chemical. ATSDR defines an exposure pathway as having five parts: 1) a source of contamination, 2) an environmental media and transport mechanism, 3) a point of exposure, 4) a route of exposure, and 5) a receptor population. When all five parts of an exposure pathway are present, it is called a Completed Exposure Pathway.

Frequency
How often a person is exposed to a chemical over time; for example, every day, once a week, or twice a month.

Hazard Index
The sum of the Hazard Quotients (see below) for all chemicals of concern (COCs) identified, which an individual is exposed. If the Hazard Index (HI) is calculated to be less than 1, then no adverse health effects are expected as a result of exposure. If the Hazard Index is greater than 1, then adverse health effects are possible. However, an HI greater than 1.0, does not necessarily suggest a likelihood of adverse effects. The HI cannot be translated to a probability that adverse effects will occur, and is not likely to be proportional to risk

Hazard Quotient
The ratio of estimated site-specific exposure to a single chemical from a site over a specified period to the estimated daily exposure level, at which no adverse health effects are likely to occur. If the Hazard Quotient is calculated to be less than 1, then no adverse health effects are expected as a result of exposure. If the Hazard Quotient is greater than 1, then adverse health effects are possible. The Hazard Quotient cannot be translated to a probability that adverse health effects will occur, and is unlikely to be proportional to risk. It is especially important to note that a Hazard Quotient exceeding 1 does not necessarily mean that adverse effects will occur.

Hazardous Waste
Substances that have been released or thrown away into the environment and, under certain conditions, could be harmful to people who come into contact with them.

Health Comparison Value
Media specific concentrations that are used to screen contaminants for further evaluation.

Health Effect
ATSDR deals only with Adverse Health Effects (see definition in this glossary).

Ingestion
Swallowing something, as in eating or drinking. It is a way a chemical can enter your body (see Route of Exposure).

Inhalation
Breathing. It is a way a chemical can enter your body (see Route of Exposure).

LOAEL
Lowest-Observed-Adverse-Effect-Level (LOAEL). LOAEL is the lowest dose of a chemical in a study (animals or people), or group of studies, that produces statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control.

Noncancer Evaluation, ATSDR's Minimal Risk Level (MRL), U.S. EPA's Reference Dose (RfD) and Reference Concentration (RfC), and California EPA's Reference Exposure Level (REL)
MRL, RfD, RfC, and REL are estimates of daily exposure to the human population (including sensitive subgroups), below which noncancer adverse health effects are unlikely to occur. MRL, RfD, RfC, and REL only consider noncancer effects. Because they are based only on information currently available, some uncertainty is always associated with MRL, RfD, RfC, and REL. "Safety" factors are used to account for the uncertainty in our knowledge about their danger. The greater the uncertainty, the greater the "safety" factor and the lower MRL, RfD, RfC or REL.

When there is adequate information from animal or human studies, MRLs and RfDs are developed for the ingestion exposure pathway, whereas RELs and RfCs are developed for the inhalation exposure pathway.

Separate noncancer toxicity values are also developed for different durations of exposure. ATSDR develops MRLs for acute exposures (less than 14 days), intermediate exposures (from 15 to 364 days), and for chronic exposures (greater than 1 year). The California EPA develops RELs for acute (less than 14 days) and chronic exposure (greater than 1 year). EPA develops RfDs and RfCs for acute exposures (less than 14 days), and chronic exposures (greater than 7 years). Both MRL and RfD for ingestion are expressed in units of milligrams of contaminant per kilograms body weight per day (mg/kg/day). REL, RfC, and MRL for inhalation are expressed in units of milligrams per cubic meter (mg/m^3).

NOAEL
No-Observed-Adverse-Effect-Level. NOAEL is the highest dose of a chemical at which there were no statistically or biologically significant increases in the frequency or severity of adverse effects seen between the exposed population (animals or people) and its appropriate control. Effects may be produced at this dose, but they are not to be adverse.

PHA
Public Health Assessment. A report or document that looks at chemicals at a hazardous waste site and determines if people could be harmed from coming into contact with those chemicals. The PHA also recommends possible further public health actions if needed.

Plume
A line or column of air or water containing chemicals moving from the source to areas further away. A plume can be a column or clouds of smoke from a chimney, contaminated underground water sources, or contaminated surface water (such as lakes, ponds, and streams).

Point of Exposure
The place where someone can come into contact with a contaminated environmental medium (air, water, food, or soil). For example, the area of a playground that has contaminated dirt, a contaminated spring used for drinking water, the location where fruits or vegetables are grown in contaminated soil, or the backyard area where someone might breathe contaminated air.

Population
A group of people living in a certain area or the number of people in a certain area.

PRG
EPA Preliminary Remediation Goals (PRGs) are tools for evaluating and cleaning up contaminated sites. They are risk-based concentrations that are intended to assist risk assessors and others in initial screening-level evaluations of environmental measurements.

PRP
Potentially Responsible Party. A company, government, or person that is responsible for causing the pollution at a hazardous waste site. PRPs are expected to help pay for the cleanup of a site.
Health Hazard

ATSDR Hazard Categories
Depending on the specific properties of the contaminant(s), the exposure situations, and the health status of individuals, a public health hazard may occur. Sites are classified using one of the following public health hazard categories:

<u>Urgent Public Health Hazard</u>
This category applies to sites that have certain physical hazards or evidence of short-term (less than 1 year), site-related exposure to hazardous substances that could result in adverse health effects. These sites require quick intervention to stop people from being exposed. ATSDR will

expedite the release of a health advisory that includes strong recommendations to immediately stop or reduce exposure to correct or lessen the health risks posed by the site.

Public Health Hazard
This category applies to sites that have certain physical hazards or evidence of chronic (long-term, more than 1 year), site-related exposure to hazardous substances that could result in adverse health effects. ATSDR will make recommendations to stop or reduce exposure in a timely manner to correct or lessen the health risks posed by the site.

Indeterminate Public Health Hazard
This category applies to sites where critical information is lacking (missing or has not yet been gathered) to support a judgment regarding the level of public health hazard. ATSDR will make recommendations to identify the data or information needed to adequately assess the public health risks posed by this site.

No Apparent Public Health Hazard
This category applies to sites where exposure to site-related chemicals might have occurred in the past or is still occurring, but the exposures are not at levels likely to cause adverse health effects. ATSDR may recommend any of the following public health actions for sites in this category:

- Cease or further reduce exposure (as a preventive measure)
- Community health/stress education
- Health professional education
- Community health investigation

No Public Health Hazard
This category applies to sites where no exposure to site-related hazardous substances exists. ATSDR may recommend community health education for sites in this category.

For more information, consult Chapter 9 and Appendix H in the 2005 ATSDR Public Health Assessment Guidance Manual (http://www.atsdr.cdc.gov/HAC/PHAManual/index.html).

Qualitative Description of Estimated Increased Cancer Risks
The qualitative interpretation for estimated increased cancer risks are as follow:

Quantitative Risk Estimate	Qualitative Interpretation
Less than 1 in 100,000	No apparent increased risk
1 in 100,000 to 9 in 100,000	Very low increased risk
1 in 10,000 to 9 in 10,000	Low increased risk
1 in 1,000 to 9 in 1,000	Moderate increased risk
Greater than 9 in 1,000	High increased risk

Receptor Population
People who live or work in the path of one or more chemicals, and who could come into contact

with them (see Exposure Pathway).

RMEG (Reference Dose Media Evaluation Guides)
If no MRL is available to derive an EMEG, ATSDR develops RMEGs using EPA's reference doses (RfDs), available at http://www.epa.gov/iris/, and default exposure assumptions, which account for variations in intake rates between adults and children. EPA's reference concentrations (RfCs), available at http://www.epa.gov/iris/, serve as RMEGs for air exposures. Like EMEGs, RMEGs represent concentrations of substances (in water, soil, and air) to which humans may be exposed without experiencing adverse health effects. RfDs and RfCs consider lifetime exposures, therefore RMEGs apply to chronic exposures.

Route of Exposure
The way a chemical can get into a person's body. There are three exposure routes: 1) breathing (also called inhalation), 2) eating or drinking (also called ingestion), and 3) getting something on the skin (also called dermal contact).

Safety Factor
Also called Uncertainty Factor. When scientists do not have enough information to decide if an exposure will cause harm to people, they use uncertainty factors and formulas in place of the information that is not known. These factors and formulas can help determine the amount of a chemical that is not likely to cause harm to people.

Source (of Contamination)
The place where a chemical comes from, such as a smokestack, landfill, pond, creek, incinerator, tank, or drum. Contaminant source is the first point of an exposure pathway.

Sensitive Populations
People who may be more sensitive to chemical exposures because of certain factors such as age, sex, occupation, a disease they already have, or certain behaviors (cigarette smoking). Children, pregnant women, and older people are often considered special populations.

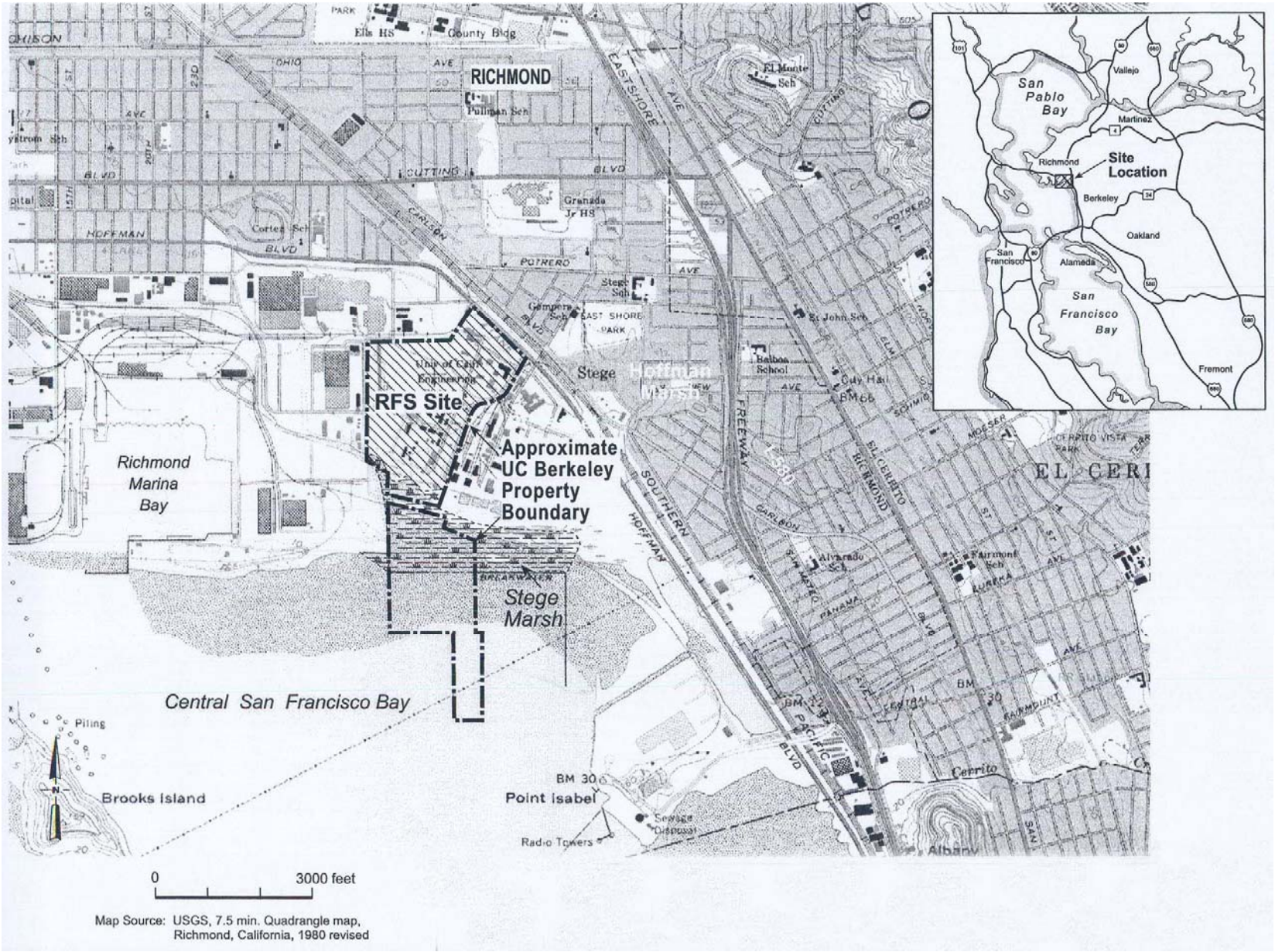
Toxic
Harmful. Any substance or chemical can be toxic at a certain dose (amount). The dose determines the potential harm of a chemical and whether it would cause someone to get sick.

Toxicology
The study of harmful effects of chemicals on humans or animals.

Volatile Organic Chemical (VOC)
Substances containing carbon and different proportions of other elements such as hydrogen, oxygen, fluorine, chlorine, bromine, sulfur, or nitrogen. These substances easily volatilize (become vapors or gases) into the atmosphere. A significant number of VOCs are commonly used as solvents (paint thinners, lacquer thinner, degreasers, and dry-cleaning fluids).

Appendix B. Figures

Figure 1. Site Location Map, University of California, Berkeley, Richmond Field Station, Richmond, California

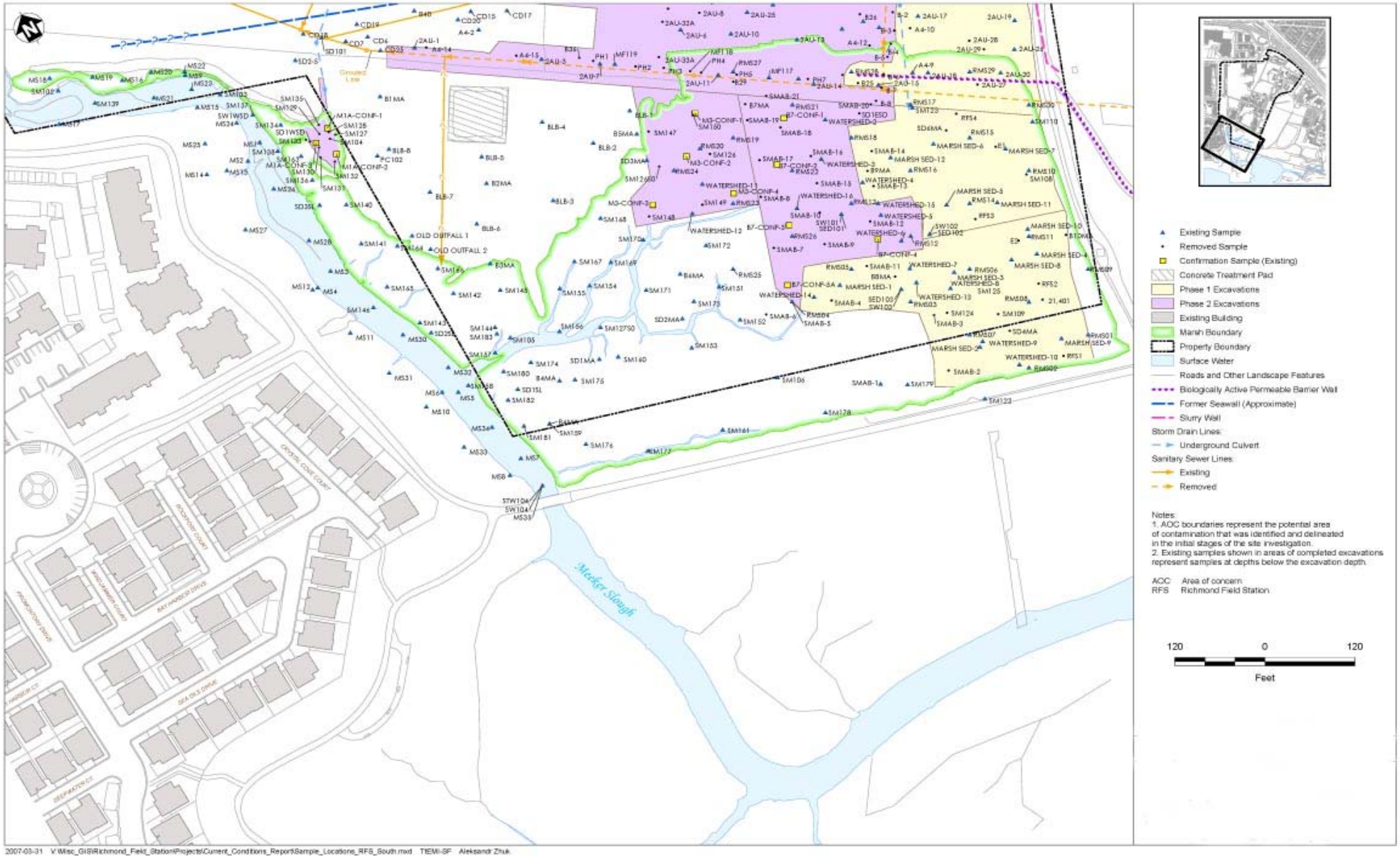


Data source (21)

Figure 2. Location of Phase 1 and Phase 2 Remedial Areas in the Western Stege Marsh, University of California, Berkeley, Richmond Field Station, Richmond, California

Data source (1)

Figure 3. Soil and Sediment Sampling Locations in the West Stege Marsh and Southern Portion of the site, University of California, Berkeley, Richmond Field Station, Richmond, California



Data source (83)

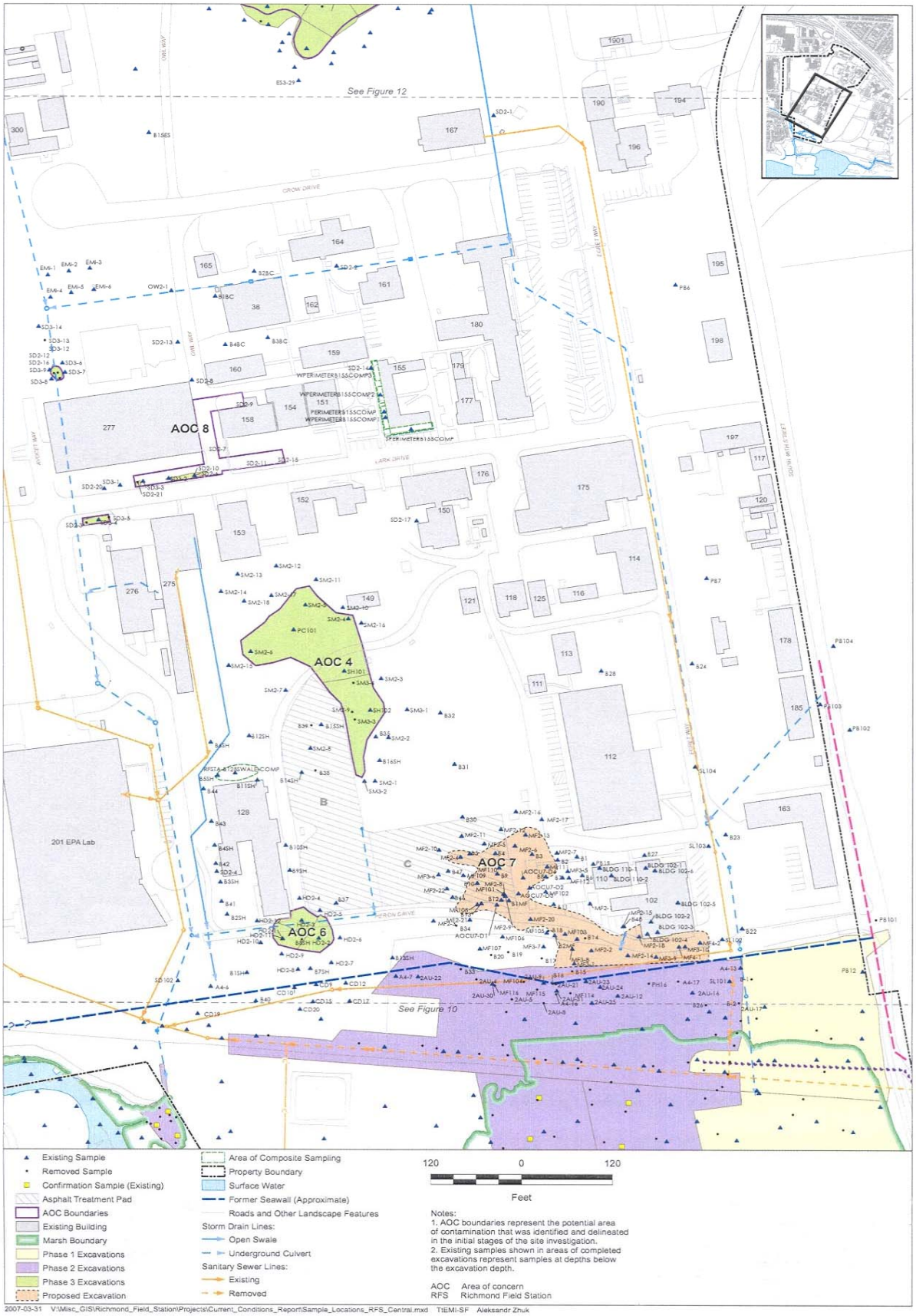
Figure 4. Location of Completed and Proposed Remediation Areas, University of California, Berkeley, Richmond Field Station, Richmond, California

Data source (83)

Figure 5a. Soil and Sediment Sampling Locations in the Northern Portion of the Site, University of California, Berkeley, Richmond Field Station, Richmond, California

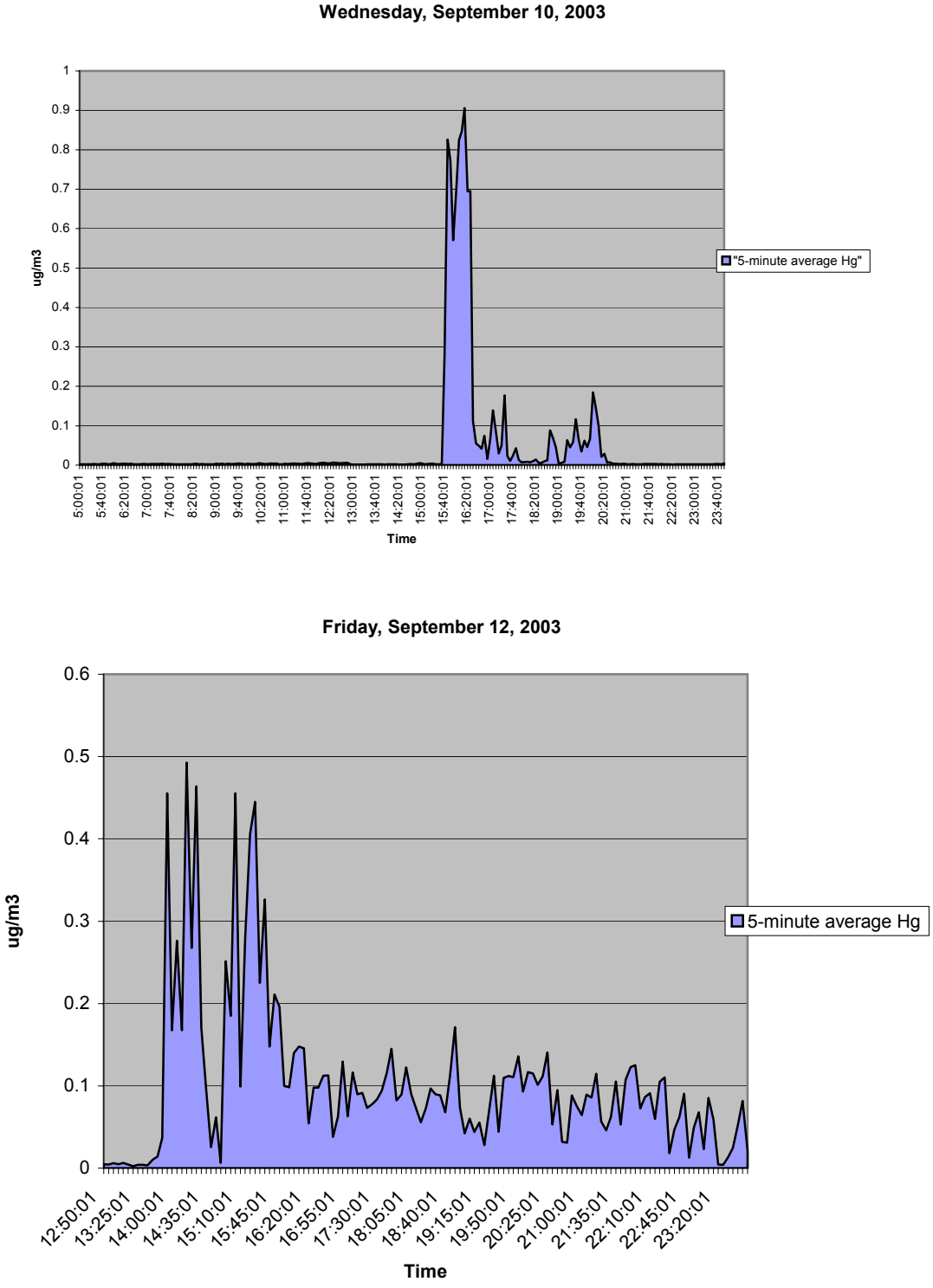
Data source (83)

Figure 5b. Soil and Sediment Sampling Locations in the Central Portion of the Site, University of California, Berkeley, Richmond Field Station, Richmond, California



Data source (83)

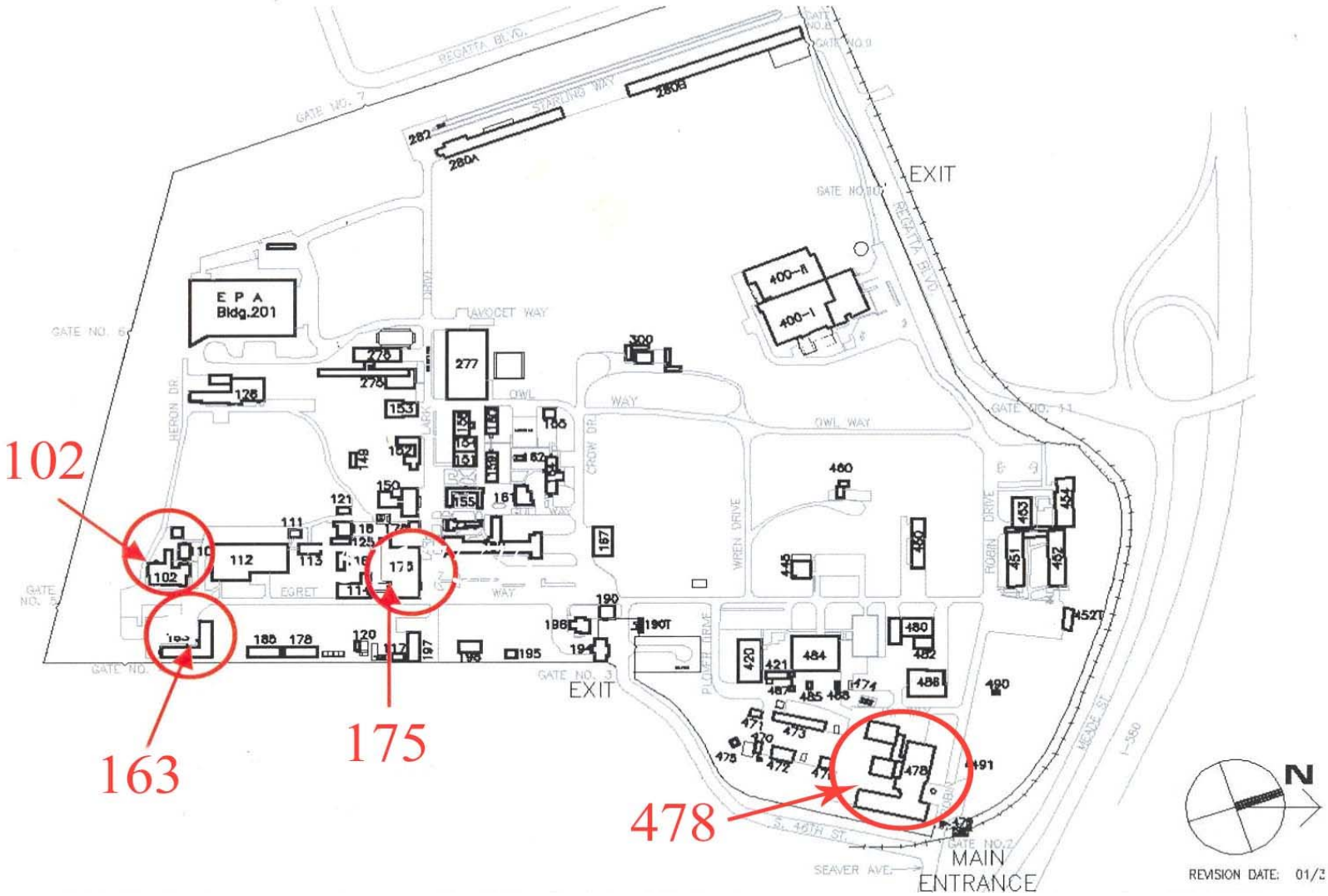
Figure 6. Monitoring Results For the Two Days When Airborne Mercury Exceeded the Chronic Minimal Risk Level (MRL) at the U.S. Environmental Protection Agency Laboratory, University of California, Berkeley, Richmond Field Station, Richmond, California



Data source (30)

MRL for mercury in air = 0.2 μg/m^3. The chronic MRL is a level at which exposure occurring for greater than 364 days would not be expected to result in noncancer adverse health effects. The Office of Environmental Health Hazard Assessment's Acute Reference Exposure Level for mercury in air = 1.8 μg/m^3. The acute REL is a level at which exposure occurring for 1-14 days would not result in noncancer adverse health effects.

Figure 7. Indoor Air Sampling Locations, University of California, Berkeley, Richmond Field Station, Richmond, California



Data sources (29, 33)

Appendix C. Tables

Table 1. Completed Exposure Pathways (Situations), University of California, Berkeley, Richmond Field Station, Richmond, California

Pathway Name	Contaminants of Concern	Pathway Elements					
		Source	Environmental Media	Point of Exposure	Route of Exposure	Potentially Exposed Population	Time
Western Stege Marsh, sediment and surface water	Metals, PCBs	RFS	Sediment, Water	Marsh	Ingestion (drinking), dermal (skin)	Adults and children/teenagers who come into contact with marsh sediment and surface water	Past, current, future
Western Stege Marsh restoration, sediment and surface water	Metals, PCBs	RFS	Sediment, Water	Marsh	Ingestion (drinking), dermal (skin)	Adults and children/teenagers who come into contact with marsh sediment and surface water during restoration activities	Current, future
On-site soil	Metals, PCBs	RFS	Soil	Soil	Ingestion (eating), dermal (skin)	RFS workers who dig in the soil	Past, current, future
Outdoor air during remedial work	Metals, dust	RFS	Air	Outdoor air	Inhalation (breathing)	Bay Trail users, Marina Bay residents, RFS workers	Past, current, future
Indoor air	Metals, VOCs	RFS	Air	Indoor air	Inhalation (breathing)	RFS workers	Current, future

Table 2. Summary of Contaminants Detected in Sediments in the Western Stege Marsh, University of California, Berkeley, Richmond Field Station, Richmond, California

Chemical	Sediment in Marsh Still in Place (0-2 ft)	Sediment/Surface Soil in Marsh Removed (0-1 ft)	Post Restoration Removal Area (0-0.5 ft)	Comparison/Screening Value (ppm)
	Maximum Concentration at 0 ft (at 1-2 ft) (ppm)	Maximum Concentration (ppm)	Maximum Concentration (ppm)	
Metals				
Arsenic	260[1] (520[2])	2,210[15]	590[23]	20 Chronic EMEG (child) 200 Chronic EMEG (adult) 0.07 Residential CHHSL 0.39 Residential PRG (Background = 3.5)
Cadmium	<0.32 (9.8[3])	33.7[16]	6.6[23]	10 Chronic EMEG (child) 100 Chronic EMEG (adult) 1.7 Residential CHHSL (Background = 0.36)
Copper	740[4] (1,500[2])	1,330[17]	900[24]	500 Chronic EMEG (child) 7,000 Chronic EMEG (adult) 3,000 Residential CHHSL 3,100 Residential PRG (Background = 28.7)
Lead	560[5]	814[18]	410[24]	150 Cal-modified PRG (Background = 23.9)
Mercury	69[4] (100[2])	10.6[19]	34[23]	23 Residential PRG (Background = 0.26)
Zinc	1,100[6] (4,200[7])	3,930[17]	1,700[24]	20,000 Chronic EMEG (child) 200,000 Chronic EMEG (adult) 23,000 Residential CHHSL (Background = 149)
Pesticides				
α-BHC (hexachloro cyclohexane)	0.0049[8]	<0.0076-<0.5	NA	0.09 Residential PRG
α-Chlordane	0.12[5]	NA	NA	30 Chronic EMEG (child) 400 Chronic EMEG (adult)
γ-Chlordane	0.15[5]	<0.0076-<0.5	NA	1.6 Residential PRG
DDD	<0.05-<0.12	0.178[20]	NA	3 CREG 2.4 Residential PRG
DDE	0.11[9]	<0.005-<0.5	NA	1.7 Residential PRG
DDT	<0.05-<0.12	0.54	NA	2 CREG 400 Intermediate EMEG
Dieldrin	0.8[5]	<0.005-<0.5	NA	3 Chronic EMEG (child) 40 Chronic EMEG (adult)
Endosulfan	0.0044[10]	<0.005-<0.5	NA	100 Chronic EMEG (child) 1,000 Chronic EMEG (adult)

Table 2. Summary of Contaminants Detected in Sediments in the Western Stege Marsh, University of California, Berkeley, Richmond Field Station, Richmond, California

Chemical	Sediment in Marsh Still in Place (0-2 ft)	Sediment/Surface Soil in Marsh Removed (0-1 ft)	Post Restoration Removal Area (0-0.5 ft)	Comparison/Screening Value (ppm)
	Maximum Concentration at 0 ft (at 1-2 ft) (ppm)	Maximum Concentration (ppm)	Maximum Concentration (ppm)	
Methoxychlor	0.28	<0.005-<0.5	NA	300 Intermediate EMEG (child)
Pebulate	0.14[11]	NA	NA	33,800 Residential PRG
Polychlorinated Biphenyls (PCBs)				
PCBs-Aroclor 1248	39[12] (65[13])	1.4[21]	2.1[23]	0.50 Residential PRG
PCBs-Aroclor 1254	7.7[14] (25[13])	0.50[22]	0.39[6]	0.22 Residential PRG
PCBs-Aroclor 1260	0.69 (3.5[13])	<0.015-<1.9	0.096[6]	0.50 Residential PRG

Data sources (11, 83-85).
ft: feet; ppm: parts per million; NA: not analyzed; PCBs: Polychlorinated Biphenyls
PRG: U.S. Environmental Protection Agency Region 9 Preliminary Remediation Goal, based on noncancer health effects unless noted
EMEG: Agency for Toxic Substances and Disease Registry Environmental Media Evaluation Guide (see Glossary, Appendix A)
CREG: Agency for Toxic Substances and Disease Registry Cancer Risk Evaluation Guide for 1 in 1,000,0000 increased cancer risk (see Glossary, Appendix A)
RMEG: U.S. Environmental Protection Agency Reference Dose Media Evaluation Guide (see Glossary, Appendix A)
([1–24]) = Sample locations for contaminants exceeding screening values: [1]SM179 at 0 ft; [2]MS16 at 2 ft; [3]SM155 at 0 ft; [4]MS15 at 0 ft; [5]MS22 at 0-0.5 ft; [6]Watershed 11 at 0-0.2 ft; [7]MS16 at 2.0 ft; [8]MS28 at 0 ft; [9]MS35 at 0 ft; [10]MS1 at 0-0.5 ft; [11]SM172 at 0.05 ft; [12]SM138 at 0-0.5 ft; [13] MS22 at 1-1.5ft; [14]Old Outfall 2 at 0-0.2 ft; [15]B10MA at 0 ft; [16]SD6MA at 0ft; [17]RFS-1 at 0 ft; [18]SD6MA at 0ft: [19]SD6MA at 0ft; [20]B8MA; [21]SM135 at 0-0.5 ft; [22]SM123 at 0-0.5 ft; [23]RMS18 at 0-0.5ft; [24]RMS26 at 0-0.5ft

Table 3. Contaminants Detected in Surface Water in the Western Stege Marsh, University of California, Berkeley, Richmond Field Station, Richmond, California

Contaminant	Historic Concentrations (maximum / average concentrations detected in 1991 and 2002) (µg/L)	Current Concentrations (maximum concentration detected in 2006) (µg/L)	Comparison/Screening Value (Source) (µg/L)
Arsenic	1,570 / 744.2 (1991) † 59 (2002) †	18†	3 (child EMEG) 10 (adult EMEG)
Cadmium	53.8 / 6.1 (1991) † <5.0 (2002)	<5.0	1 (child EMEG) 17 (adult EMEG)
Copper	2,360 / 244.2 (1991) † 440† (2002)	23	100 (child EMEG) 400 (adult EMEG)
Chromium	132 / 15.0 (1991) <10 (2002)	<10	20,000 (child EMEG) 50,000 (adult EMEG)
Mercury	0.4 / 0.19 (1991) <0.2 (2002)	0.26	3 (child EMEG)* 10 (adult EMEG)*
Zinc	7,900 / 841.6 (1991) † 550 (2002)	470	3,000 (child EMEG) 10,000 (adult EMEG)
PCBs (as Aroclor 1248)	Not analyzed (1991) 1.4 / 0.0004 (2002)†	<0.96	0.02 (CREG)

Data sources (13, 14, 83)
µg/L: microgram per liter
CREG: Agency for Toxic Substances and Disease Registry Cancer Risk Evaluation Guide for 1 in 1,000,0000 increased cancer risk (see Glossary, Appendix A)
EMEG: Agency for Toxic Substances and Disease Registry Environmental Media Evaluation Guide (see Glossary, Appendix A)
*EMEG for methylmercury (based on the potential for methylization of mercury in sediments and surface water)
†Values exceed health comparison screening values and are evaluated further
PCBs: Polychlorinated biphenyls

Table 4. Range of Concentrations for Contaminants Exceeding Comparison Values in Sediment Removed During Phase 1 and Phase 2 Remedial Activities in the Western Stege Marsh, University of California, Berkeley, Richmond Field Station, Richmond, California

Contaminant	Range of Concentrations (0-1 ft) (ppm)	Average Concentration (ppm)	Comparison/Screening Value (Source) (ppm)
Arsenic	<2.60-2,210	251.7	20 Chronic EMEG (child) 200 Chronic EMEG (adult)
Cadmium	1.60-33.70	7.5	10 Chronic EMEG (child) 100 Chronic EMEG (adult)
Copper	13.0-1,330	273.7	500 Chronic EMEG (child) 7,000 Chronic EMEG (adult)
Lead	8.90-814	156.1	150 Cal-modified PRG
Mercury	<0.044-10.6	5.2	23 Residential PRG
Zinc*	40.0-3,930	764.6	20,000 Chronic EMEG (child) 200,000 Chronic EMEG (adult) (Background = 158)
Total PCBs	<0.015-1.54	0.22	0.4 (CREG)

ft: feet; ppm: parts per million
*Zinc concentrations do not exceed comparison values in sediment; however, since historic concentrations of zinc in surface water (Table 3) exceed comparison values, sediment was included in evaluation. Half the method detection limit was used for non-detects in calculating the average
CREG: Agency for Toxic Substances and Disease Registry Cancer Risk Evaluation Guide for 1 in 1,000,0000 increased cancer risk (see Glossary, Appendix A)
EMEG: Agency for Toxic Substances and Disease Registry Environmental Media Evaluation Guide (see Glossary, Appendix A)

Table 5. Noncancer Dose Estimates for Contaminants Exceeding Screening Values in Sediment and Surface Water in the Western Stege Marsh, University of California, Berkeley, Richmond Field Station, Richmond, California

Contaminant	Total Noncancer Dose Estimates Child/Teen (mg/kg/day)		Total Noncancer Dose Estimates Adult (mg/kg/day)		Toxicity/Health Comparison Value (mg/kg/day)
	Historic (prior to 2003)	**Current** (as of 2006)	**Historic** (prior to 2003)	**Current** (as of 2006)	
Arsenic	Sediment 0.00006	Sediment 0.00015	Sediment 0.00002	Sediment 0.00003	0.0003 (MRL)
	Surface water 0.00027	Surface water 0.000006	Surface water 0.00014	Surface water 0.0000005	
Cadmium	Sediment 0.0000004	Sediment 0.0000004	Sediment 0.0000001	Sediment 0.0000001	0.0002 (MRL)
	Surface water 0.000002	Surface water ND	Surface water 0.000001	Surface water ND	
Copper	Sediment 0.00003	Sediment 0.00004	Sediment 0.00002	Sediment 0.00003	0.01 (MRL)
	Surface water 0.00009	Surface water 0.000008	Surface water 0.00005	Surface water 0.000004	
Mercury	Sediment 0.000005	Sediment 0.000009	Sediment 0.0000002	Sediment 0.000002	0.0003 (MRL)*
	Surface water 0.00000007	Surface water 0.00000009	Surface water 0.00000004	Surface water ND	
Zinc	Sediment 0.00004	Sediment 0.0001	Sediment 0.00001	Sediment 0.00002	0.3 (MRL)
	Surface water 0.00029	Surface water 0.0002	Surface water 0.00016	Surface water 0.00009	
PCBs	Sediment 0.000002	Sediment 0.00005	Sediment 0.0000004	Sediment 0.00001	0.00002 (MRL)
	Surface water 0.00001	Surface water ND	Surface water 0.0000001	Surface water ND	

Data source (4)
Maximum surface sediment values used for estimating current exposure doses; "historic" calculation for surface water based on sample collected in 1991, prior to any remedial actions in the marsh; dose estimates include ingestion and dermal exposure; ND: not detected at laboratory detection limit; MRL: Agency for Toxic Substances and Disease Registry Minimal Risk Level; *MRL for methylmercury (based on the potential for methylization of mercury in sediments and surface water)

Exposure assumptions used in estimating dermal dose surface water (15, 86, 87)

CW = concentration in water (mg/L)
P = permeability constant (cm/hour) (chemical specific: arsenic 0.001, cadmium 0.001, copper 0.001, mercury 0.001, zinc 0.0006)
Conversion factor = liters to cm^2
SA = Skin surface area (cm^2) (adult = 5809 cm^2) from EPA exposure factors handbook, averaging the 50^{th} percentile for lower legs feet and hands of females and males with that of the forearms of males (data not supplied for women). Skin surface area (child = 5323 cm^2) from EPA exposure factors handbook, averaging the 50^{th} percentile for total body surface area for males and females ages 8-15 multiplied by the percentage of total surface area that the legs, hands, and feet.
ET = exposure time (1 hour/day)
EF = exposure frequency (100 days/year)
ED = exposure duration – years of exposure (child: 10 years) (Adult: 26 years)
BW = body weight (kg) (for child 41.9 kg: average of 50^{th} percentile of females and males ages 8-15) (for adult 71.8 kg: average of women and men)
AT = averaging time (days) (ED * 365 days/year) for non carcinogen; averaging time for carcinogen dose is equal to 70 years * 365 days/year

Equation: $(CW)(P)(0.001L/cm^2)(SA)(ET)(EF)(ED)/(BW)(AT)$

Exposure assumptions used in estimating dermal dose from sediment (2, 15, 86)

CS = concentration in sediment (mg/kg)
SSA = soil to skin adherence factor (0.2 mg/cm^2) child/teenager; (0.07 mg/cm^2) adult
CF = Conversion factor (10^{-6} kg/mg)
SA = Skin surface area (cm^2 /event) – Skin surface area (adult = 5809 cm^2) from U.S. Environmental Protection Agency, Exposure Factors Handbook, averaging the 50^{th} percentile for lower legs feet and hands of females and males with that of the forearms of males (data not supplied for women). Skin surface area (child = 5323 cm^2) from EPA exposure factors handbook, averaging the 50^{th} percentile for total body surface area for males and females ages 8-15 multiplied by the percentage of total surface area that the legs, hands, and feet.
AF = Absorption factor (unitless) (chemical specific: arsenic 0.03, copper 0.01, mercury 0.01, zinc 0.001, PCBs 0.15)
Skin surface area (adult) from the U.S. Environmental Protection Agency (EPA) exposure factors handbook, averaging the 50^{th}
EF = exposure frequency (100 events/year)
ED = exposure duration – years of exposure (child: 10 years) (adult: 26 years)
BW = body weight (for child 41.9 kg: average of 50^{th} percentile of females and males ages 8-15) (for adult 71.8 kg: average of women and men)
AT = averaging time (ED * 365 days/year) for non carcinogen

Equation: $(CS)(SSA)(CF)(SA)(AF)(EF)(ED)/(BW)(AT)$

Exposure assumptions used in estimating ingestion dose from surface water

Cw = chemical Concentration in Water (mg/L)
IR = ingestion rate (0.05 liter/hour)
ET = exposure time (1 hour/day)
EF = exposure frequency (100 days/year)
ED = exposure duration – years of exposure (child: 10 years) (adult: 26 years)
BW = body weight (kg) (for child 41.9 kg: average of 50^{th} percentile of females and males ages 8-15) (for adult 71.8 kg: average of women and men)
AT = averaging time (days) (ED * 365 days/year) for non carcinogen; averaging time for carcinogen dose is equal to 70 years * 365 days/year

Equation: $(CW)(IR)(ET)(EF)(ED)/(BW)(AT)$

Exposure assumptions used in estimating ingestion dose from sediment (2, 15)

CS = chemical concentration in sediment (mg/kg)
IR = ingestion rate (mg/day) – (adult 100 mg/day)(child 200 mg/day)
ET = exposure time (1 hour/day)
EF = exposure frequency (100 days/year)
ED = exposure duration – years of exposure (child: 10 years) (adult: 26 years)
CF = conversion factor (10^{-6} kg/mg)
BW = body weight (kg) (for child 41.9 kg: average of 50^{th} percentile of females and males ages 8-15) (for adult 71.8 kg: average of women and men)
AT = averaging time (days) (ED * 365 days/year) for non carcinogen

Equation: $(CS)(IR)(ET)(EF)(ED)(CF)/(BW)(AT)(24)$

Table 6. Estimated Hazard Quotients and Hazard Index for Children and Adults Recreating in the Western Stege Marsh, University of California, Berkeley, Richmond Field Station, Richmond, California

Contaminant	Hazard Quotients			
	Surface Water Historic (prior to 2003)	Surface Water Current/Future	Sediment Historic (prior to 2003)	Sediment Current/Future
Arsenic	0.9 (child/teen) 0.5 (adult)	0.02 (child/teen) 0.01 (adult)	0.2 (child/teen) 0.05 (adult)	0.5 (child/teen) 0.1 (adult)
Cadmium	0.01 (child/teen) 0.006 (adult)	ND	0.002 (child/teen) 0.0006 (adult)	0.002 (child/teen) 0.0006 (adult)
Copper	0.009 (child/teen) 0.005 (adult)	0.0008 (child/teen) 0.0004 (adult)	0.003(child/teen) 0.002 (adult)	0.001 (child/teen) 0.0007 (adult)
Mercury	0.0002 (child/teen) 0.0001 (adult)	0.03 (child/teen) 0.001 (adult)	0.002 (child/teen) 0.0005 (adult)	0.03 (child/teen) 0.007 (adult)
Zinc	0.001 (child/teen) 0.0005 (adult)	0.0005 (child/teen) 0.0003 (adult)	0.0001 (child/teen) 0.00004 (adult)	0.0003 (child/teen) 0.00007 (adult)
Total Polychlorinated biphenyls (PCBs)	0.7 (child/teen) 0.5 (adult)	ND	0.08 (child/teen) 0.02 (adult)	2.6 (child/teen) 0.6 (adult)
Hazard Index				
	1.6 (child/teen) 0.5 (adult)	0.05 (child/teen) 0.01 (adult)	0.3 (child/teen) 0.07 (adult)	3.1 (child/teen) 0.7 (adult)

Hazard quotient: intake dose/toxicity value
Hazard Index: sum of hazard quotients
Hazard quotients include ingestion and dermal exposure
ND: not detected at laboratory detection limit

Table 7. Noncancer Dose Estimates, Health Comparison Values and Hazard Quotient and Hazard Index for Adults and Children/Teenagers Restoring the Western Stege Marsh, University of California, Berkeley, Richmond Field Station, Richmond, California

Contaminant	Estimated Dose (mg/kg/day)	Toxicity/Health Comparison Value (source) (mg/kg/day)	Hazard Quotient
Arsenic	Sediment 0.0002 (child/teen) 0.0001 (adult)	0.0003 (MRL)	Sediment 0.7 (child/teen) 0.3 (adult)
	Surface water 0.00002 (child/teen) 0.000009 (adult)		Surface water 0.05(child/teen) 0.03 (adult)
Cadmium	Sediment 0.00000005 (child/teen) 0.00000003 (adult)	0.0002 (MRL)	Sediment 0.0002 (child/teen) 0.00003 (adult)
	None for surface water		
Copper	Sediment 0.0002 (child/teen) 0.00008 (adult)	0.01 (MRL)	Sediment 0.02 (child/teen) 0.008 (adult)
	Surface water 0.00002 (child/teen) 0.00001 (adult)		Surface water 0.002 (child/teen) 0.001 (adult)
Mercury	Sediment 0.000007 (child/teen) 0.000003 (adult)	0.0003 (MRL)*	Sediment 0.02 (child/teen) 0.01 (adult)
	Surface water 0.00002 (child/teen) 0.00001 (adult)		Surface water 0.001 (child/teen) 0.0008 (adult)
PCBs	Sediment 0.0000004 (child/t teen) 0.0000002 (adult)	0.00002 (MRL)	Sediment 0.02 (child/teen) 0.01 (adult)
	None for surface water		
Hazard Index			Sediment 0.8 (child/teen) 0.3 (adult)
			Surface water 0.1 (child/teen) 0.07 (adult)

Data source (11)
Dose estimates include ingestion and dermal exposure to sediment
MRL: Agency for Toxic Substances and Disease Registry Minimal Risk Level (http://www.atsdr.cdc.gov/mrls/)
*MRL for methylmercury (based on the potential for methylization of mercury in sediments)
Hazard Quotient: intake dose/toxicity value
Hazard Index: sum of hazard quotients

Exposure assumptions used in estimating dermal dose sediment (2, 15, 86, 87)
CS = concentration in sediment (mg/kg)
SSA = soil to skin adherence factor (0.2 mg/cm^2) child/teenager; (0.07 mg/cm^2) adult
CF = Conversion factor (10^{-6} kg/mg)
SA = Skin surface area (cm^2 /event) – Skin surface area (adult = 5809 cm^2) from U.S. Environmental Protection Agency, Exposure Factors Handbook, averaging the 50th percentile for lower legs feet and hands of females and males with that of the forearms of males (data not supplied for women). Skin surface area (child = 5323 cm^2) from EPA exposure factors handbook, averaging the 50th percentile for total body surface area for males and females ages 8-15 multiplied by the percentage of total surface area that the legs, hands, and feet.
AF = Absorption factor (unitless) (chemical specific: arsenic 0.03, copper 0.01, mercury 0.01, zinc 0.001, PCBs 0.15)
Skin surface area (adult) from the U.S. Environmental Protection Agency (EPA) exposure factors handbook, averaging the 50th
EF = exposure frequency (100 events/year)
ED = exposure duration – years of exposure (child: 8 years) (adult: 8 years)
BW = body weight (for child 41.9 kg: average of 50th percentile of females and males ages 8-15) (for adult 71.8 kg: average of women and men)
AT = averaging time (ED * 365 days/year) for non carcinogen
Equation: (CS)(SSA)(CF)(SA)(AF)(EF)(ED)/(BW)(AT)

Exposure assumptions used in estimating ingestion dose from sediment (2, 15)
CS = chemical concentration in sediment (mg/kg)
IR = ingestion rate (mg/day) – (adult 100 mg/day)(child 200 mg/day)
ET = exposure time (2.6 hour/day)
EF = exposure frequency (100 days/year)
ED = exposure duration – years of exposure (child: 8 years) (adult: 8 years)
CF = conversion factor (10^{-6} kg/mg)
BW = body weight (kg) (for child 41.9 kg: average of 50th percentile of females and males ages 8-15) (for adult 71.8 kg: average of women and men)
AT = averaging time (days) (ED * 365 days/year) for non carcinogen
Equation: (CS)(IR)(ET)(EF)(ED)(CF)/(BW)(AT)(24)

Exposure assumptions used in estimating dermal dose from surface water (2, 15, 86, 87)
CW = concentration in water (mg/L)
P = permeability constant (cm/hour) (chemical specific: arsenic 0.001, cadmium 0.001, copper 0.001, mercury 0.001, zinc 0.0006)
Conversion factor = liters to cm^2
SA = Skin surface area (cm^2) (adult = 5809 cm^2) from EPA exposure factors handbook, averaging the 50th percentile for lower legs feet and hands of females and males with that of the forearms of males (data not supplied for women). Skin surface area (child = 5323 cm^2) from EPA exposure factors handbook, averaging the 50th percentile for total body surface area for males and females ages 8-15 multiplied by the percentage of total surface area that the legs, hands, and feet.
ET = exposure time (2.6 hour/day)
EF = exposure frequency (100 days/year)
ED = exposure duration – years of exposure (child: 8 years) (adult: 8 years)
BW = body weight (for child 41.9 kg: average of 50th percentile of females and males ages 8-15) (for adult 71.8 kg: average of women and men)
AT = averaging time (ED * 365 days/year) for non carcinogen; averaging time for carcinogen dose is equal to 70 years * 365 days/year
Equation: (CW)(P)(0.001L/cm^2)(SA)(ET)(EF)(ED)/(BW)(AT)

Exposure assumptions used in estimating ingestion dose from surface water (2, 15)
CW = chemical concentration in water (mg/L)
IR = ingestion rate (0.05 liter/hour)
ET = exposure time (2.6 hour/day)
EF = exposure frequency (100 days/year)
ED = exposure duration – years of exposure (child: 8 years) (adult: 8 years)
BW = body weight (kg) (for child 41.9 kg: average of 50th percentile of females and males ages 8-15) (for adult 71.8 kg: average of women and men)
AT = averaging time (days) (ED * 365 days/year) for non carcinogen; averaging time for carcinogen dose is equal to 70 years * 365 days/year
Equation: (CW)(IR)(ET)(EF)(ED)/(BW)(AT)

Table 8. Summary of Contaminants Detected in the Richmond Field Station Soil and Comparison/Screening Values, University of California, Berkeley, Richmond Field Station, Richmond, California

Chemical	Surface and Near Surface Soil in Exposed, Non- excavated Areas (0-4 ft bgs)		Surface Soil and Near Surface Soil in Excavated Areas (0-4 ft bgs)		Comparison/Screening Value (ppm) (Background Level)
	Maximum Concentration (ppm)	Average Concentration (ppm)	Maximum Concentration (ppm	Average Concentration (ppm)	
Metals					
Antimony	4.8	4.1	ND (<3.1)		380 CHHSL 342 Industrial PRG
Arsenic	1,300[1]	15.9	150[2]	10.7	200 Chronic EMEG 0.24 CHHSL 1.6 Industrial PRG (Background = 3.5)
Barium	310	226	Not analyzed		63,000 CHHSL 175,000 Industrial PRG
Beryllium	2.5	0.47	1.0	0.45	1,700 CHHSL 1,300 Industrial PRG
Cadmium	437[3]	3.34	6.1[4]	1.84	100 chronic EMEG 7.5 CHHSL 450 Industrial PRG (Background = 0.36)
Chromium	110	36.2	170	39.7	10,000 CHHSL 734,000 Industrial PRG
Copper	13,000[5]	104	4,000[6]	286	3,800 CHHSL 3,100 Industrial PRG (Background = 28.7)
Lead	1,140[7]	35.1	1,000[10]	57.5	800 Industrial PRG (23.9 Background)
Mercury	270[9]	26.7	140[10]	4.24	180 CHHSL 310 Industrial PRG (Background = 0.26)
Molybdenum	3.6	2.44	Not analyzed		4,800 CHHSL 5,580 Industrial PRG

Table 8. Summary of Contaminants Detected in the Richmond Field Station Soil and Comparison/Screening Values, University of California, Berkeley, Richmond Field Station, Richmond, California

Chemical	Surface and Near Surface Soil in Exposed, Non- excavated Areas (0-4 ft bgs)		Surface Soil and Near Surface Soil in Excavated Areas (0-4 ft bgs)		Comparison/Screening Value (ppm) (Background Level)
	Maximum Concentration (ppm)	Average Concentration (ppm)	Maximum Concentration (ppm	Average Concentration (ppm)	
Nickel	230	45.2	78	48.5	16,000 CHHSL 22,000 Industrial PRG
Selenium	4.5	0.85	3.1	0.76	4,800 CHHSL 5,590 Industrial PRG
Silver	1.9	0.66	1.1	0.13	4,800 CHHSL 5,480 Industrial PRG
Thallium	9.4	1.23	2.7	0.67	63 CHHSL 87.9 Industrial PRG
Vanadium	60	46.4	Not analyzed		6,700 CHHSL 4,790 Industrial PRG
Zinc	2,150	115	480	108	100,000 CHHSL 330,000 Industrial PRG
Pesticides					
α-BHC (hexachloro-cyclohexane)	ND (<0.058)		0.0418	0.0418	0.36 Industrial PRG
γ-Chlordane	0.092	0.089	ND (<0.038)		1.7 CHHSL 5.6 Industrial PRG
DDD	ND (<0.0075)		0.33	0.22	3 CREG 9.0 CHHSL 12.1 Industrial PRG
DDE	0.047	0.073	ND (<0.0075)		6.3 CHHSL 8.5 Industrial PRG
DDT	0.38	0.13	0.22	0.13	400 Intermediate EMEG 6.3 CHHSL 7.2 Industrial PRG 2 CREG

Table 8. Summary of Contaminants Detected in the Richmond Field Station Soil and Comparison/Screening Values, University of California, Berkeley, Richmond Field Station, Richmond, California

Chemical	Surface and Near Surface Soil in Exposed, Non- excavated Areas (0-4 ft bgs)		Surface Soil and Near Surface Soil in Excavated Areas (0-4 ft bgs)		Comparison/Screening Value (ppm) (Background Level)
	Maximum Concentration (ppm)	Average Concentration (ppm)	Maximum Concentration (ppm	Average Concentration (ppm)	
Dieldrin	0.0082	0.036	ND (<0.0075)		40 Chronic EMEG (adult) 0.13 CHHSL 0.16 Industrial PRG
Polychlorinated biphynels (PCBs)					
PCBs-Aroclor 1248	5.2[11]	1.46	430[11]	15.2	0.78 Industrial PRG
PCBs-Aroclor 1254	0.69[12]	0.13	7.1[13]	0.47	0.74 Industrial PRG
PCBs-Aroclor 1260	0.33[14]	0.07	15[11]	0.55	0.73 Industrial PRG

Data sources (6, 21, 83, 85)
Average concentration calculated using ½ the detection limit for non-detects.
ft: feet; bgs: below ground surface; ppm: parts per million
ND: not detected; detection limit not available; NA: not analyzed;
CHHSL: California Environmental Protection Agency Human Health Screening Level for industrial/commercial land use
PRG: U.S. Environmental Protection Agency Region 9 Preliminary Remediation Goal
EMEG: Agency for Toxic Substances and Disease Registry Environmental Media Evaluation Guide for an adult resident (intermediate: exposure duration lasting between 14-365 days; chronic: exposure duration lasting longer than 365 days) (see Glossary, Appendix A)
CREG: Agency for Toxic Substances and Disease Registry Cancer Risk Evaluation Guide for 1 in 1,000,0000 increased cancer risk (see Glossary, Appendix A)
RMEG: U.S. Environmental Protection Agency Reference Dose Media Evaluation Guide (see Glossary, Appendix A)

([1–14]) = Sample locations for contaminants exceeding screening values: [1] WTA45 at 0-0.5ft; [2] FP2-5 at 0ft; [3] B2MF at 1.5 ft; [4] SH2-7 at 0 ft; [5] BI6SH at 1-3 ft; [6] TP2-7 at 0 ft; [7] B2MF at 1 ft; [8] SM2-4 at 0 ft; [9] AOCU7-D1 at 0ft; [10] SH101 at 0 ft; [11]SD2-10 at 0.5-1 ft; [12]HD2-9 at 0 ft; [13]HD2-1 at 0 ft; [14]SD2-9 at 0.5-1 ft;

Table 9. Non Cancer Dose Estimates, Health Comparison Values and Hazard Index for Richmond Field Station Workers Who Dig in On-Site Soil, University of California, Berkeley, Richmond Field Station, Richmond, California

Contaminant	Estimated Dose Long-Term Past Exposure (maximum concentration) (mg/kg/day)	Estimated Dose Short-Term Current Exposure (maximum concentration) (mg/kg/day)	Toxicity/Health Comparison Value (Source) (mg/kg/day)	Hazard Quotient
Arsenic	0.00047	0.00047	0.0003 (MRL)	1.6 (long-term) 1.6 (short-term)
Cadmium	0.00014	0.00014	0.0002 (MRL)	0.7 (long-term) 0.7 (short-term)
Copper	0.0043	0.0043	0.01 (MRL)	0.4 (long-term) 0.4 (short-term)
Mercury	0.00009	0.00009	0.002 (RfD)*	0.3 (long-term) 0.3 (short-term)
			Hazard Index (metals) →	3.0 (long-term) 3.0 (short-term)
Total PCBs	0.00025	0.000003	0.00002 (MRL)	1.2 (long-term) 0.2 (short-term)

Dose estimates include ingestion and dermal exposure; *RfD for mercuric chloride
Hazard quotient: intake dose/toxicity value; Hazard Index: sum of hazard quotients

Exposure assumptions used in estimating dermal dose (15, 86, 87)
CS = concentration in soil (mg/kg)
SSA = soil to skin adherence factor (0.07 mg/cm^2)
CF = conversion factor (10^{-6} kg/mg)
AF = absorption factor (unitless) (chemical specific: arsenic 0.03, copper 0.01, mercury 0.01, PCBs 0.15)
SA = Skin surface area (cm^2 /event) – Skin surface area (adult = 5809 cm^2) from U.S. Environmental Protection Agency, Exposure Factors Handbook, averaging the 50th percentile for lower legs feet and hands of females and males with that of the forearms of males (data not supplied for women).
EF = exposure frequency (100 events/year)
ED = exposure duration – years of exposure (long-term: 23 years) (short-term: 7 years)
BW = body weight (71.8 kg: average of women and men)
AT = averaging time (ED * 365 days/year) for non carcinogen

Equation: (CS)(SSA)(CF)(SA)(AF)(EF)(ED)/(BW)(AT)

Exposure assumptions used in estimating ingestion dose (15, 88)
CS = chemical concentration in soil (mg/kg)
IR = ingestion rate (330 mg/day): estimated intake for adults engaged in outdoor activities
ET = exposure time (2 hours/day)
EF = exposure frequency (100 days/year)
ED = exposure duration – years of exposure (long-term: 23 years) (short-term: 7 years)
CF = Conversion Factor (10^{-6} kg/mg)
BW = body weight (kg) (71.8 kg: average of women and men)
AT = averaging time (days) (ED * 365 days/year) for non carcinogen

Equation: (CS)(IR/4)(ET)(EF)(ED)(CF)/(BW)(AT)

Table 10. Mercury Levels Measured in Ambient Air On-Site During the Phase 2 Remedial Work (2003), University of California, Berkeley, Richmond Field Station, Richmond, California

Date	9/12	9/13	9/15	9/15	9/15	9/15	9/15	9/15	9/16	9/16	9/16	9/16	9/17	9/17	9/17	9/19	9/19	9/22	9/22	9/23	9/23	
Time →			9:30	10:30	11:30	13:30	14:30	16:30	8:30	11:30	13:30	16:30	9:30	11:30	14:00	10:30	14:30	11:30	14:30	11:30	13:30	MEAN
Station																						
1	**0.004**	<0.003	0.006	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003		<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	**0.003**	0.00065
2	**0.004**	<0.003	**0.004**	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.00038
3	**0.006**	<0.003	**0.006**	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	**0.003**	<0.003	**0.004**	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.00090
4	<0.003	<0.003	**0.004**	<0.003	**0.005**	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	**0.005**	<0.003	<0.003	<0.003	<0.003	<0.003	**0.004**	<0.003	<0.003	**0.004**	0.00105
5	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003		<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.00000
6	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003		<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.00000
7	<0.003	<0.003																				
8	**0.004**	<0.003																				
9	<0.003	<0.003																				
10	<0.003	<0.003																				

Detected values in **bold**
Samples collected using a Jerome Mercury Vapor Analyzer (field instrument) with a detection limit of 3 $\mu g/m^3$ (0.003 mg/m^3)
Acute Reference Exposure Level (REL) = 1.8 $\mu g/m^3$

Table 11. Common Sources of Chemicals Found in Indoor Air, University of California, Berkeley, Richmond Field Station, Richmond, California

Chemical Name	Sources
Acetone	Used as a common solvent.
Acetonitrile	Found in certain lithium batteries. Used to make plastics, synthetic rubber, and acrylic fibers. Used as a common solvent in laboratories.
Acrolein	Used in plastics, perfumes, aquatic herbicides. Also found in cigarette smoke and automobile exhaust.
Benzene	Found in cigarette smoke, gasoline, crude oil, and used as a solvent. May be an ingredient of household products such as glues, paints, furniture wax, and detergents.
2-Butanone	Found in paints, coatings, glues, cleaning agents, and cigarette smoke. It occurs naturally in some fruit and trees. Also known as Methyl Ethyl Ketone or MEK.
tert-Butyl alcohol	Found as flavors, in perfumes, in paint remover, as a gasoline booster, and in solvents.
Carbon disulfide	Used in the manufacturing of rayon, in soil disinfectants, and in solvents.
Chlorobenzene	Used as a solvent for paints, pesticides.
Chloroethane	Used as a refrigerant, solvent. Also used in making cellulose, dyes, medicinal drugs.
Chloromethane	Byproduct of burning grasses, wood, cigarettes, charcoal, or plastic. Found in styrofoam insulation, aerosol propellants, and chlorinated swimming pools.
Dichlorodifluoromethane	Used as a refrigerant, aerosol propellant, and solvent. Also known as Freon 12.
cis-1,2-Dichloroethene	Found in perfumes, dyes, lacquers, solvents, and products made from natural rubber.
Ethylbenzene	Used as a common solvent, and found in gasoline, inks, insecticides, and paints. Also found in cigarette smoke.
4-Ethyltoluene	Used as a solvent, found in kerosene and light vapor oil.
Formaldehyde	Used in production of adhesives and binders for wood, plastics, textiles, leather and related industries. Also found in vehicle emissions, cigarette smoke, disinfectants and food.

Table 11. Common Sources of Chemicals Found in Indoor Air, University of California, Berkeley, Richmond Field Station, Richmond, California

Chemical Name	Sources
Heptane/Hexane	Found in petroleum products, is often mixed with other solvents, and is used as a filling for thermometers.
Isooctane	Found in petroleum, gasoline, solvents, and thinners. A component of the "odor" of gasoline.
Methyl t-butyl ether	Used as an additive in unleaded gasoline.
Pentane	Found in petroleum, gasoline.
Propene	A flammable propellant, produced from petroleum cracking.
Styrene	Found in synthetic rubbers, resins, insulators.
Tetrachloroethylene	Used in dry cleaning and as a degreaser. When clothes are brought home from the drycleaners, they often release small amounts of tetrachloroethylene into the air.
Toluene	Used as a common solvent, and found in gasoline, paints and lacquers. Also found in cigarette smoke.
1,1,1-Trichloroethane	Used as a degreaser, in solvents, and as an aerosol propellant.
Trichloroethylene	Used as a degreasing agent. It is also a common ingredient in cleaning agents, paints, adhesives, varnishes, and inks.
Trichlorofluoromethane	Used as refrigerant, aerosol propellant, and solvent. Also known as Freon 11.
1,2,4-Trimethylbenzene	Used to make drugs and dyes, in gasoline and certain paints and cleaners.
1,3,5-Trimethylbenzene	Component in diesel exhaust.
Xylenes	Used as a solvent, cleaning agent, and thinner for paints, and in fuels and gasoline.

Data source (89)

Table 12. Contaminants Detected in Indoor and Outdoor Air on the Richmond Field Station, and Health Comparison Values, University of California, Berkeley, Richmond Field Station, Richmond, California

Contaminant	Date Sampled	Sample Location Sample Results (μg/m^3)			Health Comparison Value (μg/m^3)
		Building 163	Building 175	Building 175 Roof	
Arsenic (metal)	8/16/05 9/20/05	0.098* < 0.05	0.085* < 0.05	< 0.08	0.19 (acute REL) 0.03 (chronic REL) 0.0002 (CREG)
Volatile Organic Chemicals					
Acetone	9/21/05	25	17	7.6	365 (PRG) 30,881 (MRL)
Benzene	9/21/05	1.3	< 0.30	< 0.30	60 (REL) 0.10 (CREG) 160 (MRL)
Bromoethane	9/21/05	11	< 0.68	< 0.68	19.4 (MRL)
Bromoform	9/21/05	6.3	< 1.2	< 1.2	0.9 (CREG)
Carbon Disulfide	9/21/05	19	< 0.40	< 0.40	800 (chronic REL)
Chloroform	9/21/05	< 0.62	< 0.62	0.78	300 (REL) 0.04 (CREG)
Chloromethane	9/21/05	< 0.39	2.1	< 0.39	90 (RfC)
1,2-Dibromoethane	9/21/05	1.8	< 0.90	< 0.90	9 (RfC) 0.002 (CREG)
Dichlorodifluoromethane	9/21/05	2.5	3.7	2.5	200 (RfC)
Formaldehyde	9/21/05 10/20/05	410* 0.16, 0.12, 0.16	37* not sampled	12* not sampled	94 (acute REL) 3 (chronic REL) 40 (acute MRL) 0.08 (CREG)
Freon 11 (Trichlorofluoromethane)	9/21/05	1.2	1.7	1.2	730 (PRG)
Hexane	9/21/05	< 0.49	1.9	< 0.49	7,000 (REL) 210 (PRG) 2,100 (MRL)

Table 12. Contaminants Detected in Indoor and Outdoor Air on the Richmond Field Station, and Health Comparison Values, University of California, Berkeley, Richmond Field Station, Richmond, California

Contaminant	Date Sampled	Sample Location Sample Results (μg/m^3)			Health Comparison Value (μg/m^3)
		Building 163	Building 175	Building 175 Roof	
Methylene chloride	9/21/05	4.0	2.0	0.45	4.1 (PRG†) 3.0 (CREG)
Propene	9/21/05	6.2	3.6	< 0.17	not available
Styrene	9/21/05	0.73	< 0.27	< 0.27	900 (REL) 1,100 (PRG) 260 (MRL)
Tetrachloroethylene	9/21/05	< 0.79	< 0.79	2.6	0.32 (PRG†) 35 (REL) 270 (MRL)
Toluene	9/21/05	2.8	7.3	< 0.45	300 (REL) 400 (PRG) 300 (MRL)
Trichlororethylene	9/21/05	1.4	< 0.59	< 0.59	0.017 (PRG†) 40 (RfC) 540(REL)
1,2,4-Trimethylbenzene	9/21/05	< 0.37	0.65	< 0.37	6.2 (PRG)
m,p-Xylene	9/21/05	< 0.93	2.2	< 0.93	700 (chronic REL)
o-Xylene	9/21/05	< 0.45	0.61	< 0.45	700 (chronic REL)

*exceeds noncancer health comparison values
REL: Office of Environmental Health Hazard Assessment Reference Exposure Level
CREG: Agency for Toxic Substances and Disease Registry Cancer Risk Evaluation Guide for 1 in 1,000,0000 increased cancer risk (see Glossary, Appendix A)
PRG: U.S. Environmental Protection Agency Region 9 Preliminary Remediation Goal (exposure occurring for greater than 364 days
PRG† is based upon cancer endpoint (level reflects 1 in 1,000,000 increased cancer risk, considered no apparent increased risk)
MRL: Agency for Toxic Substances and Disease Registry Chronic Minimal Risk Level (http://www.atsdr.cdc.gov/mrls/)
RfC: U.S. Environmental Protection Agency Reference Concentration (http://www.epa.gov/iris/search.htm)

Appendix D. Toxicological Summaries

This appendix provides background information from toxicological profiles published by the Agency for Toxic Substances and Disease Registry, information developed by the California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, and the U.S. Environmental Protection Agency. It highlights the toxicological effects of chemicals of concern (chemicals exceeding health comparison or screening values) detected in air, soil, surface water, or groundwater, in and around the Richmond Field Station site.

Arsenic (20)

- Naturally-occurring element commonly found in surface soil and surface water.
- Arsenic trioxide is the primary form marketed and consumed, with 90% used in the production of wood preservatives (copper chromated arsenic).
- Various organic arsenicals are still used in herbicides and as antimicrobials in animal and poultry feed.
- Long-term exposures of lower levels of arsenic through drinking water (170-800 ppb) can lead to a condition known as "blackfoot disease."
- Other effects include gastrointestinal irritation, and contact with skin can cause discoloration (hypo-or hyper-pigmentation), wart-like growths, and skin cancer.
- Acute oral minimal risk level (MRL) = 0.005 mg/kg/day (gastrointestinal effects in humans).
- Chronic oral minimal risk level (MRL) = 0.0003 mg/kg/day (dermal effects in humans).
- Oral reference dose (RfD) = 0.0003 mg/kg/day (dermal effects in humans).
- Acute reference exposure level (REL) = 0.19 $\mu g/m^3$ (reproductive, developmental effects in mice).
- Chronic reference exposure level (REL) = 0.03 $\mu g/m^3$ (developmental, cardiovascular, nervous system in mice).
- Oral cancer slope factor = 1.5 mg/kg/day.
- Inhalation unit risk (U.S. Environmental Protection Agency) = 0.0043 $\mu g/m^3$.
- Carcinogenicity: known human carcinogen due to its ability to cause skin cancer, with oral exposures increasing the risks of liver, bladder, and lung cancer (U.S. Environmental Protection Agency); carcinogenic to humans (International Agency for Research on Cancer).

Cadmium (25)

- Naturally-occurring element (metal); also occurs as a result of industrial processes.
- Not usually found as a pure metal, but as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulfur (cadmium sulfate, cadmium sulfide).
- Enters the body primarily through inhalation and ingestion; people are exposed to cadmium mostly from food and cigarette smoke.
- Inhalation of high levels of cadmium can severely damage the lungs and cause death.
- Chronic exposure (inhalation) to low levels can cause kidney (renal) damage.
- Chronic oral minimal risk level (MRL) = 0.0002 mg/kg/day (kidney damage in humans).
- Carcinogenicity: probable human carcinogen (limited human, sufficient animal evidence) (U.S. Environmental Protection Agency); human carcinogen (sufficient human evidence) (International Agency for Research on Cancer).

Copper (23)

- Naturally-occurring metal found in rocks, soil sediment, and water.
- Occurs naturally in all plant and animals.
- Essential element for humans, plants and other animals.

- Long-term exposure to copper dust can irritate your nose, mouth, and eyes, and cause headaches, dizziness, nausea, and diarrhea.
- Common effects from ingestion of higher than normal levels of copper include nausea, vomiting, stomach cramps, or diarrhea.
- Intermediate oral minimal risk level (MRL) = 0.01 mg/kg/day (gastrointestinal effects in humans).
- Carcinogenicity: not classifiable as a human carcinogen due to a lack of studies (U.S. Environmental Protection Agency); not reviewed (International Agency for Research on Cancer).

Formaldehyde (34)

- Colorless flammable gas at room temperature.
- Commonly contaminant found in indoor and outdoor air.
- Common health effects include irritation of the eyes, nose, and throat, along with increased tearing, which occurs at air concentrations of about 400-3,000 parts per billion (491-3655 $\mu g/m^3$).
- Acute inhalation minimal risk level (MRL) = 40 $\mu g/m^3$ (respiratory effects in humans).
- Intermediate inhalation minimal risk level (MRL) = 30 $\mu g/m^3$ (respiratory effects in monkeys).
- Acute reference exposure level (REL) = 94 $\mu g/m^3$ (eye irritation in humans).
- Chronic reference exposure level (REL) = $\mu g/m^3$ (respiratory effects in humans).
- Inhalation unit risk (U.S. Environmental Protection Agency) = 0.000013 $\mu g/m^3$.
- Carcinogenicity: probable human carcinogen, based on limited evidence in humans (site-specific respiratory neoplasms) and sufficient evidence in animals (nasal squamous cell carcinomas in mice and rats) (U.S. Environmental Protection Agency).

Lead (17, 22)

- Naturally-occurring metal found in small amounts in the earth's crust; most of the high levels of lead found in the environment are from human activities.
- People may be exposed to lead by eating foods or drinking water that contains lead, spending time in areas where leaded paints have been used or are deteriorating, lead pipes, and drinking from leaded-crystal glassware.
- People who live near hazardous waste sites may be exposed to lead and chemicals containing lead by breathing the air, swallowing dust and dirt containing lead, or drinking lead-contaminated water
- Lead affects the nervous system, the blood system, the kidneys, and the reproductive system.
- Low blood levels (30 µg/dL) may contribute to behavioral disorders; lead levels in young children have been consistently associated with deficits in reaction time and with reaction behavior. These effects on attention occur at blood lead levels extending below 30 µg/dL, and possibly as low as 15-20 µg/dL; the developing nervous system of a young child can be adversely affected at blood lead levels below 10 µg/dL.
- Health effects associated with lead are not based on an external dose, but on internal dose that takes into account total exposure.
- Federal agencies and advisory groups have defined childhood lead poisoning as a blood lead level of 10 µg/dL.
- Occupational Safety and Health Administration requires workers with a blood lead level above 50 µg/dL be removed from the workroom where lead exposure is occurring.
- Carcinogenicity: probable human carcinogen (renal tumors in mice) (U.S. Environmental Protection Agency); possibly carcinogenic to humans (limited evidence of kidney, brain and lung cancer) (International Agency for Research on Cancer).

Mercury (24)

- Mercury occurs naturally in the environment and exists in several forms; these forms can be organized under three headings: metallic mercury (also known as elemental mercury), inorganic mercury, and organic mercury. Toxicity depends on the form of mercury.
- Metallic mercury is used in a variety of household products and industrial items, including thermostats, fluorescent light bulbs, barometers, glass thermometers, and some blood pressure devices.
- Spills of metallic mercury from broken thermometers or damaged electrical switches in the home may result in exposure to mercury vapors in indoor air that could be harmful to health; microorganisms (bacteria, phytoplankton in the ocean, and fungi) convert inorganic mercury to methylmercury.
- Ingestion of fish one of the most common ways people are exposed to methylmercury.
- Exposure to high levels (above 500 $\mu g/m^3$ and above 1.9 mg/kg/day) of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing fetus.
- Chronic inhalation minimal risk level (MRL) = 0.2 $\mu g/ m^3$ (neurological effects in humans).
- Intermediate oral minimal risk level (MRL) (inorganic mercury/mercuric chloride) = 0.002 mg/kg/day (renal effects in mice).
- Chronic minimal risk level (MRL) (methylmercury) = 0.0003 mg/kg/day (neurodevelopment effects in humans).
- Carcinogenicity: mercury chloride and methylmercury are possible human carcinogens (U.S. Environmental Protection Agency); not classified (International Agency for Research on Cancer).

Polychlorinated Biphenyls (PCBs) (19)

- Produced in the United States between 1933-1977 for use as coolants and lubricants.
- Mixtures of up to 209 individual chlorinated compounds (known as congeners).
- Though no longer manufactured, PCBs are still released during some industrial processes, from hazardous waste sites; illegal or improper disposal of industrial wastes, consumer products; leaks from old electrical transformers containing PCBs; and burning of some wastes in incinerators.
- Food most common source of PCBs uptake in the general population.
- Bioaccumulate in food chains and are stored in fatty tissues.
- Do not readily break down in the environment and thus may remain there for very long periods of time.
- Most common health effect observed from exposure to PCBs are skin rashes and acne.
- Reproductive effects have been shown in women exposed to high levels of PCBs in the work place or from eating contaminated fish.
- High levels of PCBs may cause liver damage.
- Intermediate minimal risk level (MRL) for Aroclor 1254 = 0.00003 mg/kg/day (developmental effects).
- Chronic minimal risk level (MRL) for Aroclor 1254 = 0.00002 mg/kg/day (immunological effects).
- Limited human (workers) and animal studies have shown an association with liver and biliary cancer.
- Carcinogenicity: probable human carcinogen, based on sufficient evidence of carcinogenicity in animals (U.S. Environmental Protection Agency); probably carcinogenic to humans (International Agency for Research on Cancer).

Zinc (81)

- Naturally-occurring metal found in rocks, soil sediment, and water.
- Essential element for humans and animals.
- Ingestion of high levels of zinc can cause stomach cramps, nausea and vomiting.
- Inhalation of high levels of zinc dust or fumes can cause metal fume fever.
- Intermediate minimal risk level (MRL) (zinc and zinc compounds) = 0.3 mg/kg/day (decreases in erythrocyte SOD and serum ferritin levels in humans).
- Carcinogenicity: not classifiable as a human carcinogen due to a lack of studies (U.S. Environmental Protection Agency); not reviewed (International Agency for Research on Cancer).