



Public Health Assessment for

Evaluation of Exposure to Contaminants from the
ZENECA/CAMPUS BAY SITE
1200 SOUTH 47TH STREET
RICHMOND, CONTRA COSTA COUNTY, CALIFORNIA

EPA FACILITY ID: CAD009123456
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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
Agency for Toxic Substances and Disease Registry

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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 (federal)

bgs—below ground surface

CAG—Community Advisory Group

Cal/EPA—California Environmental Protection Agency

CDPH—California Department of Public Health

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)

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

$\mu\text{g}/\text{m}^3$ —microgram per cubic meter of air

VOC—volatile organic compound

Summary

In this public health assessment (PHA), the California Department of Public Health (CDPH) looks at the possible ways people could come into contact with contaminants from the Zeneca/Campus Bay, and responds to 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 exposure pathways (scenarios): historic exposure to the residents of the Seaport warehousing apartments, nearby residents and off-site workers; contamination in the East Stege Marsh; airborne contaminants released during remedial work conducted between 2002 and 2005; impact on indoor air quality in Harborfront businesses from volatile contaminants in groundwater; and contaminants in dust in Building 240. Second, the PHA describes health concerns collected from community members and adjacent business owners and workers. Third, the PHA evaluates these health concerns based on environmental data review described above, and describes what is known about the cause of the health effects/concerns expressed to CDPH.

Stauffer Chemical Company (henceforth referred to as Stauffer) began operations at the site in 1897 with sulfuric acid production, which continued until about 1970. Sulfuric acid production generated a large volume of cinder waste from the roasting of iron pyrite ore (1). Cinders were deposited into low-lying areas on the site over the many years of operation. Pyrite cinders are generally acidic and contain high concentrations of metals, primarily arsenic, cadmium, copper, lead, selenium, and zinc.

From 1906-1971, Stauffer manufactured superphosphate fertilizer (2). Naturally-occurring radionuclides, such as uranium and radium, are found in phosphate rock and can be byproducts found in areas of superphosphate manufacturing, depending on the type of manufacturing process used.

Other manufacturing/production operations at Stauffer included carbon disulfide (1906-1961), aluminum sulfate (1923-1984), ferric sulfate (1949-1972), and titanium trichloride (1954-1976) (2). In the 1950s, Stauffer began formulating and producing various pesticides and herbicides and opened the Western Research Center. As the name implies, the Western Research Center was used for research and development of new agricultural chemicals.

Stauffer operations continued on the site until 1985. Between 1986 and 1992, the property was transferred between several owners. In 1993, Zeneca, Inc. took over operations at the site and continued manufacturing agricultural chemical products until 1997. In 2002, Cherokee Simeon Venture (CSV) land developers purchased the 86-acre site from Zeneca Inc. and renamed it Campus Bay (1). For the purpose of this report, the site will be referred to as Zeneca.

In 1998, investigations of manufacturing areas and clean-up activities were initiated at Zeneca under the oversight of the California Regional Water Quality Control Board (RWQCB), San Francisco Bay Region. In May 2005, the California Environmental Protection Agency

(Cal/EPA)'s Department of Toxic Substances Control (DTSC) took over as the lead oversight agency for Zeneca.

In April 2005, due to ongoing community concerns about the Zeneca site, DTSC and the Contra Costa County Health Services Department (CCCHSD) 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 Zeneca.

Contaminants detected on the Zeneca site (from Stauffer operations) include metals, pesticides, herbicides, polychlorinated biphenyls (PCBs), volatile organic compounds (VOCs), and petroleum hydrocarbons. As of this writing, radionuclides associated with the production of superphosphate fertilizer and other Stauffer-related work, are being investigated. The Radiologic Health Branch of CDPH is providing technical support to the Department of Toxic Substances Control (DTSC) regarding any radiological issues at Zeneca. This PHA addresses potential exposures to chemical contaminants found at the site. If future investigations indicate a need to evaluate potential exposure to radionuclides, an addendum to this PHA will be provided.

CDPH evaluated the possible exposure pathway/activities (past, current, and future) to contaminants at Zeneca, using environmental data collected from the site. The conclusions of this evaluation are presented below.

It is possible that during remedial work conducted between May 2002 and July 2005, nearby workers and Bay Trail users could have experienced mild irritant effects of the respiratory tract from breathing dust on the days when dust levels were elevated.

CDPH concludes that no public health hazard exists from the following:

- Current exposure to contaminants underlying the Zeneca site (Lots 1-3) under the site's current use.
- Current exposure to indoor air in businesses in the Harborfront Tract from vapor intrusion, as a result of VOC-contaminated groundwater.
- Past, current, and future exposure to metals, pesticides, and PCBs in sediment and surface water in the East Stege Marsh.
- Past exposure to students and staff from site-related contaminants in dust and indoor air in Building 240, used by the Making Waves Education Program from 2002 until 2006.
- Past exposure to site-related contaminants in dust during remedial activities conducted between 2002 and 2005.

CDPH was not able to determine the potential health impacts of historic exposure to Seaport residents, nearby workers, or residents of the Panhandle Annex or adjacent neighborhoods. Given the types of manufacturing that occurred at Stauffer, the history of emission control, and regulations, we recognize that exposures at levels of health concern could have occurred. However, there is no data available to evaluate the level and magnitude of these exposures.

Due to a lack of data, CDPH was not able to determine whether there is a past, current, or future health risk from exposure to elevated levels of naturally-occurring radionuclides that may be present in non-excavated portions of the marsh as a result of Stauffer operations.

CDPH conducted a number of outreach activities to collect and understand the health concerns community members believe are related to contamination at Zeneca. CDPH evaluated the health concerns/effects by investigating their known causes, including environmental or chemical agents. The majority of the health concerns expressed to CDPH cannot be linked to chemical exposures at the site, based on the exposure and toxicological information available—with the exception of irritation of the eyes, nose, and throat, and mild respiratory effects that may have occurred from exposure to airborne dust.

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

1. If the Zeneca site land use changes from industrial to residential or recreational, then the site should be cleaned up to levels consistent with residential standards. An increased residential population in this area may result in more human activity in tidal/shoreline areas adjacent to the site. Thus, areas south of the Bay Trail should also be characterized and the risk to potential receptors should be evaluated.
2. A robust air monitoring program and adequate dust suppression measures should be implemented during future remedial work at the site, as well as during any development activities where soil is disturbed.
3. Access to the East Stege Marsh should remain restricted until there is a complete understanding of the potential radiological issues at the site, and it can be determined that the non-excavated portions of the East Stege Marsh do not contain site-related radionuclides at levels of health concern.
4. Sediment and unfiltered surface water in the east Stege Marsh should be sampled annually until the site is remediated, to ensure that the marsh is not being re-contaminated from contaminant migration from other areas.

Background and Statement of Issues

The Environmental Health Investigations Branch (EHIB) within the California Department of Public Health (CDPH), under cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR), is conducting a public health assessment (PHA) related to the Zeneca/Campus Bay site (formerly Stauffer Chemical), in Richmond, California. The PHA includes a review of existing environmental data to evaluate the potential health impact from exposures to site-related contaminants. The PHA is an evaluation of the site to help determine what follow-up activities are needed, such as additional site characterization, health education, health study, or specific measures to reduce or eliminate exposure. Specifically, we address the following exposure pathways (scenarios): historic exposure to the residents of the Seaport warehousing apartments, nearby residents and off-site workers; contamination in the East Stege Marsh; airborne contaminants released during remedial work conducted between 2002 and 2005; impact on indoor air quality from volatile contaminants in groundwater; and contaminants in dust in Building 240.

Stauffer Chemical Company (henceforth referred to as Stauffer) began operations at the site in 1897 with sulfuric acid production, which continued until about 1970. Sulfuric acid production generated a large volume of cinder waste from the roasting of iron pyrite ore (1). Cinders were deposited into low-lying areas on the site over the many years of operation. Pyrite cinders are generally acidic and contain high concentrations of metals, primarily arsenic, cadmium, copper, lead, selenium, and zinc.

From 1906-1971, Stauffer manufactured superphosphate fertilizer (2). The manufacturing of phosphate and superphosphate fertilizer is well known for its negative effects on the environment, due to the type of processes involved and uncontrolled emissions (pollution) (3-5). The first environmental regulation to affect phosphate manufacturing was the Federal Air Pollution Control Act (Clean Air Act) of 1970 (6). Naturally-occurring radionuclides, such as uranium and radium, are found in phosphate rock and can be byproducts found in areas of superphosphate manufacturing, depending on the type of manufacturing process used.

Other manufacturing/production operations at Stauffer included carbon disulfide (1906-1961), aluminum sulfate (1923-1984), ferric sulfate (1949-1972), and titanium trichloride (1954-1976) (2). In the 1950s, Stauffer began formulating and producing various pesticides and herbicides and opened the Western Research Center. As the name implies, the Western Research Center was used for research and development of new agricultural chemicals.

From 1955-1965, research using electron beam furnaces was also conducted on the Stauffer site (2). The electron beam furnaces were used to melt various metals, including uranium (2). Some of the electron beam furnace work was done for National Lead of Ohio, a contractor to the Atomic Energy Commission. Historical photographs indicate that a battery manufacturing facility and a boat resin manufacturing facility operated on the Stauffer property in the 1960s and 1970s, respectively (7).

Stauffer operations continued on the site until 1985. Between 1986 and 1992, the property was transferred between several owners. In 1993, Zeneca, Inc. took over operations at the site and continued manufacturing agricultural chemical products until 1997. In 2002, Cherokee Simeon Venture (CSV) land developers purchased the 86-acre site from Zeneca Inc. and renamed it Campus Bay (1). For the purpose of this report, the site will be referred to as Zeneca.

From the 1970s through 2004, the California Regional Water Quality Control Board (RWQCB), San Francisco Bay Region, was the lead agency responsible for oversight of waste discharge requirements and environmental investigations at the Zeneca site. In 1980, investigations of soil and groundwater quality along the perimeter of the site were initiated. In 1998, environmental investigations of active manufacturing areas on-site were initiated.

In October 2001, RWQCB issued Zeneca a Site Cleanup Requirements Order for the 86-acre site, which includes the upland area (Lots 1-3) and the adjacent East Stege Marsh and freshwater lagoons (Appendix B, Figure B-1). Clean-up activities on Lot 1 were completed in 2001. Lot 1 has since been developed into a commercial space known as the Campus Bay Business Park.

Remedial activities have been underway at the site since 2000 and have included: the localized excavation and disposal of contaminated soil; the localized treatment of VOC-contaminated groundwater; the neutralization of cinder material; the installation of a temporary surface cap covering contaminated material; the neutralization of low pH groundwater; the installation of a Biological Active Permeable Barrier on the southern boundary to reduce metal concentrations in groundwater migrating to the Stege Marsh; and the installation of a new storm drain system (1). With the exception of Building 240 (unoccupied), the site buildings were destroyed in 2000. Recent remediation work conducted between October 2004 and March 2005 has included excavating sediments in the East Stege Marsh.

In April 2005, the Contra Costa County Health Officer requested the assistance of CDPH, in responding to exposure and health concerns related to remedial activities at the Zeneca site. Since that time, CDPH has been conducting PHA activities at the Zeneca site.

In May 2005, DTSC, of the California Environmental Protection Agency (Cal/EPA), formally became the lead regulatory agency overseeing environmental investigations and cleanup at the site.

Contaminants detected on the Zeneca site include metals, pesticides, herbicides, polychlorinated biphenyls (PCBs), volatile organic compounds (VOCs), and petroleum hydrocarbons. As of this writing, radionuclides associated with the production of superphosphate fertilizer and other Stauffer-related work, are being investigated. The Radiologic Health Branch of CDPH is providing technical support to the Department of Toxic Substances Control (DTSC) regarding radiological issues at Zeneca. This PHA

addresses potential exposures to chemical contaminants found at the site. If future investigations indicate a need to evaluate potential exposure to radionuclides, an addendum to this PHA will be provided.

Land Use

The site occupies approximately 86 acres and is bordered to the north by Interstate 580 (Appendix B, Figure B-1). The Richmond Field Station operated by the University of California borders the site to the west. Small businesses border the site to the east. The San Francisco Bay shoreline, which includes the East Stege Marsh (an 8-acre saltwater marsh), borders the site to the south. The site is divided into three lots (Lot 1, Lot 2, and Lot 3), with Lot 1 being the furthest upland (north), Lot 3 adjacent to the marsh and bay, and Lot 2 in the middle (Appendix B, Figure B-1).

There are a number of other contaminated sites in the area: University of California, Richmond Field Station, Liquid Gold Oil Corporation, Bio-Rad Laboratories, Marina Bay Project, Blair Landfill, and Stege Property Pistol Range.

Site Visits

CDPH first visited the site in May 2005. During the site visit, the following observations were made:

- Fencing surrounded the site and a temporary cap was present on the site;
- Non-remediated areas of the East Stege Marsh were accessible to the public; and
- Only an administrative building remained on the site. This building is currently unoccupied.

Subsequently, staff have visited the site a number of times to observe remedial activities and the integrity of the temporary cap, and to conduct dust sampling in the remaining building (Building 240) on the site. The East Stege Marsh is now fenced and posted warning people to stay out. The type of fencing and signs were selected by DTSC, in consultation with the East Bay Regional Park District.

Demographics

Zeneca is located within Census Tract 3800, which spans approximately 7 miles across, with an estimated population of 6,002 (8). The ethnic make-up is roughly 16% Asian, 17% Hispanic or Latino, 32% African American and 35% White (8).

The closest population to the Zeneca site is the Harborfront Business Tract, which consists of approximately 38 businesses, with roughly 250 full-time employees (S. Padgett, Harborfront Business Tract employee, personal communication, January 24, 2008).

Environmental Contamination/Pathway Analysis/Toxicological Evaluation

In this section CDPH examines the pathways for exposure to contamination from the Zeneca site. CDPH examines each of the media (groundwater, sediment in the East Stege Marsh, soil, and air) to determine whether or not contamination is present and if people in the community are exposed to (or in contact with) the contamination. If people are exposed to contamination in any of the media, we evaluate whether there is enough exposure to pose a public health hazard. This analysis systematically evaluates 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 them. This is called an exposure pathway. Exposure pathways are classified as either completed, potential, or eliminated.

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. 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 C-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, CDPH evaluates 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 completely evaluate exposure to multiple chemicals, or possible cancer and noncancer adverse effects from 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 CDPH uses 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 Zeneca site, CDPH compared contaminant concentrations to media-specific environmental guideline comparison values. Those concentrations that exceed the comparison values are identified as COCs for further evaluation of potential health effects. ATSDR, EPA, and Cal/EPA'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 comparison values 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 a significant 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 are considered carcinogenic and not all carcinogenic compounds have a CREG.
- Environmental Media Evaluation Guide (EMEG). EMEGs are estimates of chemical concentrations in air, soil, and water that are not likely to cause an appreciable risk of harmful, 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) (9).
- Reference Dose Media Evaluation Guides (RMEGs). RMEGs are estimates of chemical concentrations in soil and water that are not likely to cause an appreciable risk of harmful, noncancer health effects for chronic exposure. RMEGs are based on EPA's Reference Doses (RfDs) (see Glossary in Appendix A for a more complete description of EMEGs) (10).
- Reference Exposure Levels (RELs) and Reference Concentrations (RfCs). Cal/EPA's Office of Environmental Health Hazard Assessment RELs and EPA's RfCs are

estimates of chemical concentrations in air that are not likely to cause an appreciable risk of harmful, noncancer health effects for fixed durations of exposure.

- California Human Health Screening Levels (CHHSLs). Cal/EPA 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 (11).
- Preliminary Remediation Goals (PRGs). EPA's Region IX PRGs are risk-based concentrations used in initial screening-level evaluations of environmental measurements.

If a contaminant 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 No-observed-adverse-effect level (NOAEL) or the lowest-observed-adverse-effect level (LOAEL) (see Glossary in Appendix A for description of NOAEL and LOAEL) (9).
- 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 (10).

The toxicity studies used to determine the various health comparison values are usually conducted on adult animals or adult humans, typically 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, one chemical is enhancing the effect of the other chemical; this is known as synergism. In that case, the combined dose for the two

chemicals underestimates the potential toxicity of the mixture of two chemicals. Some chemical mixtures act in a less than additive manner, which is known as an antagonistic effect. In this scenario, 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) (12). 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. The model assumes that no matter how low, even for extremely low exposures, there is always the possibility that a 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 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, which 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.

Discussion of Environmental Contamination

The following table is included as a reference tool to help differentiate the units of measurement used in the reporting and discussion of sampling data.

Table 1. Units of Measurement Used in Environmental Sampling and Reporting

Environmental Media	Unit	Equivalent Unit
Water	mg/L (milligrams chemical per liter of water)	ppm (parts per million)
	µg/L (micrograms chemical per liter of water)	ppb (parts per billion)
Soil	mg/kg (milligrams chemical per kilogram soil)	ppm (parts per million)
	µg/kg (micrograms chemical per kilogram soil)	ppb (parts per billion)
Air	mg/m ³ (milligrams chemical per cubic meter of air)	ppmv (parts per million volume) = (24.45/molecular weight of chemical (mg/m ³))
	µg/m ³ (micrograms chemical per cubic meter of air)	ppbv (parts per billion volume) =(24.45/molecular weight of chemical (µg/m ³))

On-Site Soil and Groundwater Contamination

On-site soil and groundwater on the Zeneca site are contaminated, but exposure to these media is not occurring. Contaminants present in soil and groundwater include metals, pesticides, herbicides, PCBs, VOCs, and petroleum hydrocarbons. The majority of contamination on the site is contained under a temporary cap, thus, eliminating the risk of resuspension and migration of contaminants in soils. The site is fenced, prohibiting access to the public. The City of Richmond's drinking water comes from surface water

sources away from the site, not on-site groundwater wells; therefore, it is not threatened by the groundwater contamination at the site (13). These exposure pathways have thus been eliminated from further evaluation. However, if in the future, land use of the site changes to residential or recreational, then the site should be cleaned up to levels consistent with residential standards. An increased residential population in this area may result in more human activity in tidal/shoreline areas adjacent to site. Thus, areas south of the Bay Trail should also be characterized, and the risk to potential receptors evaluated.

On the basis of available information, CDPH concludes that on-site groundwater and soils do not pose a current health hazard to the public, under the site's current use.

Off-Site Contamination

Contamination has migrated off-site and is present in sediment in the East Stege Marsh and in groundwater beneath businesses located on 49th Street, adjacent to the eastern border of the Zeneca site.

CDPH evaluated six completed exposure pathways to Zeneca-related contamination (Appendix C, Table C-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. The toxicological evaluation of the completed exposure pathways involves the use of exposure assumptions. CDPH used conservative estimates and assumptions to ensure potential health hazards from chemicals are recognized.

Stauffer Operations and Historic Exposure

Residents Living in the "Seaport Warhousing Apartments"



Stauffer Chemical and Seaport Warhousing Apartments, 1945-1948 Richmond, California

During World War II, a large migration of workers from the South and Southwestern United States arrived in the City of Richmond to work at the Kaiser shipyards (14,15). According to the City of Richmond, approximately 60,000 persons lived in rapidly-constructed public housing as a result of this influx (14). One of these public housing complexes was the Seaport Warhousing Apartments (Seaport), built in 1944 and located immediately adjacent to the Stauffer Chemical Company. Seaport residents were primarily African American (16). A former Seaport resident first brought the Zeneca site to the attention of public health agencies (17,18).

Between 1944 and 1956, approximately 400 families lived in Seaport, which were located on the eastern side of Zeneca property (see photo above) (Appendix B, Figure B-1). Seaport consisted of about 50 apartment buildings with 494 units, and an elementary school (19). The Seaport Warhousing Apartments were torn down at some point between 1956 and 1957 and the area was utilized for commercial and industrial purposes, as it still is today.



CDPH staff spoke with a number of former Seaport residents who were children or adolescents during that time. They recollected smelling bad odors most of the time, as well as dust and smoke in the air from the operations at Stauffer. Many of the children played in open ponds and in the East Stege Marsh. Some residents (see photo above) grew their own vegetables in gardens, using water pumped from groundwater wells on the property. (A more detailed discussion on the health concerns expressed to CDPH by former Seaport residents is provided in the Community Health Concerns Section.)

During this time period, Stauffer operations included a number of chemical processes used to manufacture the following: sulfuric acid, superphosphate fertilizer, carbon disulfide, aluminum sulfate, and ferric sulfate (20).

These activities resulted in chemical releases to the air, soil, surface water, and groundwater. In particular, historic phosphate fertilizer manufacturing is known for having significant impacts on the environment (3,5). Some of the contaminants associated with these activities include releases of sulfur dioxide, carbon disulfide, particulate matter (dust), metals, phosphorus compounds, fluorides, inorganic acids, VOCs, and natural-occurring radionuclides (3,21-24).

CDPH has identified the following completed exposure pathways for Seaport residents, and on-site and nearby workers: breathing outdoor air; contacting and ingesting on-site surface soil; contacting and ingesting surface water and sediment in the open ponds in the East Stege Marsh; and eating vegetables containing chemical residue either on the exterior of the plant or from uptake of metals by the plant.

Seaport residents were likely exposed to air releases/emissions of sulfur dioxide particulate matter (dust), VOCs, metals, phosphorus compounds, fluorides, inorganic acids, naturally-occurring radionuclides (related to phosphate fertilizer manufacturing), and possibly others (3,21-24). The health effects associated with exposure to these contaminants include adverse respiratory, nervous system, developmental and/or reproductive and carcinogenic effects (cancer-causing) (24). It is probable that these exposures were at unhealthy levels, especially for Seaport children (24). However, it is not possible to quantitatively evaluate the magnitude of these exposures because of a lack of data and information. During this time period (1940-1950s), emissions were not regulated or characterized (measured). Without these data and a complete understanding of the manufacturing processes at Stauffer, we cannot model¹ or estimate the historical impact.

Similar to the air pathway discussion above, the available information is not adequate to evaluate exposures to soil, sediment, groundwater, surface water, or potentially contaminated vegetables.

On the basis of limited data, CDPH was not able to determine the potential health implications for Seaport residents from exposure to historic releases (1944-1956) from Stauffer operations. The site is classified as posing an indeterminate health hazard to residents living in the Seaport Warehousing Apartments.

Residents Living in the Richmond Panhandle Annex Area

The closest residential area known as the Richmond Panhandle Annex, developed in the 1940s, is located north of the I-580. It is possible that residents living in this area and possibly other adjacent neighborhoods could have been exposed to historic air releases from Stauffer operations. However, there is no information available to evaluate the extent or magnitude of potential exposures or if any health effects would have resulted.

¹ Computer modeling is a tool that can be used to estimate the amount of chemicals released from a particular industrial process. The accuracy of the model is reflective of the completeness of the site-specific information available.

There are a couple of qualitative points that can be made regarding potential exposure: the amount of exposures would decrease the further away from the site, and the amount of releases decreased over time, with the implementation of air pollution control laws and advancements in pollution control equipment.

Nearby Businesses (Harborfront Business Tract) and the Richmond Field Station

It is possible for workers of nearby businesses and at the Richmond Field Station to have been exposed to contaminants while Stauffer was in operation. After the Seaport Warehousing Apartments were torn down (around 1956-1957), various commercial and industrial businesses began operating in the area, now known as the Harborfront Business Tract. The area is bounded by East Montgomery Avenue to the north, Meade Street to the east, and South 49th Street to the west (Appendix B, Figure 1). The Richmond Field Station, located on the western boundary of the Zeneca site, at 1301 South 46th Street, has been owned and operated by the University of California since 1950.

Similar to the discussion above relating to Seaport Residents, nearby workers in the Harborfront Business Tract and at the Richmond Field Station could have been exposed to contaminants emanating from Stauffer, later known as Zeneca, primarily through aerial releases and, to a lesser extent, through incidental ingestion of soil.

Data are not available to evaluate exposures to airborne contaminants. Thus, CDPH was not able to determine the potential health implications for workers in the Harborfront Business Tract or at the Richmond Field Station, as a result of exposure to historic releases (1957-1997) from Stauffer operations, and later Zeneca (please see Background section for history of ownership).

Evaluation of East Stege Marsh Sediment and Surface Water

It is possible that children or adults who play or recreate in the East Stege marsh could come into contact with contaminated sediments and surface water. Anecdotal information provided to CDPH suggested the possibility for homeless people to be living in marsh. CDPH staff have visited the marsh on a number of occasions and have not seen evidence of anyone living in the East Stege Marsh. Additionally, the marsh is fenced and posted to warn people to stay out. However, we recognize the potential for a homeless person to enter and/or utilize the marsh. The assumptions used to evaluate exposure to an adult are conservative/health protective and will identify whether there is a health risk for a homeless person.

Sampling conducted between 1992-2004 of the East Stege Marsh has shown the sediments to be contaminated with heavy metals, pesticides, and PCBs (Appendix B, Figure B-2 and Appendix C, Table C-2) (25). Maximum concentrations of a number of contaminants (antimony, arsenic, cadmium, copper, lead, mercury, toxaphene, Alpha-Hexachlorocyclohexane, and PCBs) detected in sediment exceed comparison/screening values; these contaminants are considered COCs and will be

evaluated further. Average (mean) concentrations of some contaminants (arsenic, copper, PCBs, and toxaphene) exceed comparison/screening values.

Surface water sampling occurred on two occasions in September 1997 and October 1997, and once in 2000 near the border of Richmond Field Station, Western Stege Marsh (Appendix C, Table C-3) (25). Maximum concentrations of a number of contaminants (antimony, arsenic, cadmium, copper, manganese, and zinc) detected in surface water exceed comparison/screening values; these contaminants are considered COCs and will be evaluated further. Average (mean) concentrations of some contaminants (antimony, arsenic, cadmium, and copper) detected in surface water exceed comparison/screening values. In May 2007, surface water samples were collected from three locations in the East Stege Marsh (26). Arsenic is the only contaminant exceeding comparison/screening values (Appendix C, Table C-3A). It is worth noting that arsenic concentrations measured in the most recent surface water sampling are consistent with levels measured prior to remedial activities in 2004-2005.

Between October 2004 and April 2005, contaminated sediments from the East Stege Marsh were removed and replaced with dredged and terrestrial materials (sediment/soil) imported from other areas. The import materials were sampled according to regulatory guidelines to show that they were clean enough to be used for fill (27). In June 2006, LFR Inc. conducted sediment sampling in the East Stege (28).

Sediment samples were collected from 20 locations in areas that received imported fill, as well as in undisturbed areas (Appendix B, Figure B-3). Sediment samples were analyzed for heavy metals, general minerals, and pH. Maximum concentrations of arsenic, lead, and mercury exceed comparison/screening values and are considered COCs; they are further evaluated (Appendix C, Table C-4). The average concentration of arsenic exceeds comparison/screening values. Surface water was not sampled.

Historic Exposure to Adults and Children/Teenagers Playing in the East Stege Marsh Prior to 2004 and 2005 Removal Actions

CDPH estimated the potential historic exposure for a child/teenager (ages 8-18) who plays in the marsh and an adult who recreates (walks, hikes, etc.) or spends time in the marsh, from skin (dermal) contact and incidental ingestion of contaminants in surface water and sediments. In the dose estimations, it was assumed that an adult and a child/teenager would play in the marsh 100 days per year, for 30 years and 11 years, respectively. We estimated exposure doses using both the maximum and average contaminant concentration detected in surface water and sediment (Appendix C, Table C-5). The dose estimates derived from the average concentrations are more reflective of the exposure an individual might receive from years of recreating in the marsh. It is improbable that an individual would spend all of his/her time (11 years for a child and 30 years for an adult) in a specific location where the maximum concentrations were detected.

CDPH determined that an adult or child/teenager who engaged in activities in the East Stege Marsh on a regular basis prior to remediation would not have experienced noncancer health effects. The estimated exposure doses from exposure to the maximum and the average concentrations of COCs in sediment and surface water do not exceed health comparison values (Appendix C, Table C-6).

The hazard index for an adult or child/teenager from exposure to the maximum concentration of multiple contaminants (metals, PCBs, and pesticides) in sediment is estimated at 0.31 and 0.71, respectively (Appendix C, Table C-6). Since the estimated hazard index does not exceed 1, no adverse health effects are likely to have occurred or be occurring to adults or children/teenager from historic exposure to contaminants in sediment.

The hazard index from exposure to the maximum concentration of contaminants in surface water exceeds 1 for a child/teenager (Appendix C, Table C-6). As stated earlier, it is improbable that a person would have been exposed to the maximum level of COCs in surface water for a number of reasons: dose estimates based on the assumption that an individual is exposed to maximum concentrations of contaminants measured at specific locations in the marsh for multiple years (11 years—child, 30 years—adult); tidal and surface water influences on concentrations; and seasonal differences on surface water availability in the marsh. The hazard index is below 1 from exposure to the average concentration of contaminants in surface water (Appendix C, Table C-6). Thus, adverse health effects should not have occurred or be occurring from exposure to metals, pesticides, or PCBs in the East Stege Marsh.

Lead is evaluated based on an internal dose (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 recommended action level for lead exposure in children is 10 micrograms per deciliter ($\mu\text{g}/\text{dL}$). 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 $\mu\text{g}/\text{dL}$ (29). Childhood Lead Poisoning Prevention Branch of CDPH recommends exposure reduction/mitigation actions for pregnant women with BLLs of 10 $\mu\text{g}/\text{dL}$ or greater (30).

CDPH used the DTSC Lead Risk Assessment Spreadsheet (LeadSpread 7) to estimate BLL for adults. LeadSpread estimates BLL for children under the age of two². The exposure scenario being evaluated for this exposure pathway is for children 8-18 years

² As a point of reference, exposure to the highest level of lead (740 ppm) in the East Stege marsh prior to remediation would result in an estimated BLL for a 1-2 year old child of 12.2 $\mu\text{g}/\text{dL}$; the adult BLL is 6.2 $\mu\text{g}/\text{dL}$. It is reasonable to assume that the BLL for a child between 8-18 years old would fall between these two numbers, and below 10 $\mu\text{g}/\text{dL}$. If the average lead value (149.0) is used rather than the highest, the estimated BLL for a 1-2 year old is 5.0 $\mu\text{g}/\text{dL}$.

old. EPA's Adult Lead Model was used to estimate BLL for women of childbearing age, as it is protective of fetal health (31).

The estimated BLL for adults from exposure to the average level of lead (149 ppb) in the marsh (prior to remediation) is 3.1 µg/dL (95th percentile); exposure to the highest level of lead (740 ppb) would result in an estimated BLL for adults of 6.2 µg/dL. The BLL for women of childbearing age was estimated at 5.2 µg/dL (average level of lead) and 7.8 µg/dL (highest level of lead). These values include exposure to background sources of lead, such as ambient air, water, and produce. These levels are below 10 µg/dL for pregnant women and 25 µg/dL for all other adults, the levels at which exposure reduction actions are recommended (29,30).

CDPH estimated the theoretical increased cancer risk from historic exposure to the maximum and average concentration of contaminants considered carcinogenic in sediment and surface water. Carcinogenic contaminants exceeding screening values in surface water and/or sediment are arsenic, cadmium, PCBs, and toxaphene (Appendix C, Tables C-2 and C-3). We included all carcinogenic contaminants detected in sediment (above and below screening values) in the theoretical increased cancer risk estimates. The estimated increased cancer risk for adults and child/teenager from exposure to the maximum concentration of contaminants is 8 in 1,000,000 and 3 in 100,000, respectively. These are considered "no apparent to very low increased risks." The estimated increased cancer risk for adults and children/teenager from exposure to the average concentration of contaminants is 9 in 10,000,000 and 4 in 1,000,000, respectively. These are considered "no apparent increased risks" (32). Equations and cancer slope factors used to estimate increased cancer risks are provided in Appendix E.

In summary, CDPH concludes historic exposure metals, pesticides, and PCBs in sediments and/or surface water in the East Stege Marsh would not have caused noncancer adverse health effects in adults or children/teenagers. Exposure to the average and maximum concentrations of carcinogenic contaminants poses a "no apparent to very low" increased cancer risk. It was not possible to determine whether there was a potential health risk from exposure to elevated levels of naturally-occurring radionuclides in the marsh, because there is no data to make such a determination.

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

CDPH used the most recent sediment data collected in June 2006 and surface water data collected in 2007, to evaluate the current and future exposure to children from contaminants remaining in the East Stege Marsh.

CDPH estimated the potential current and future exposure for a child/teenager (ages 8-18) who plays in the marsh and an adult who recreates or spends time in the marsh from skin (dermal) from contact and incidental ingestion of contaminants in sediment and surface water (Appendix C, Table C-3A, Table C-4, Table C-7). In the dose estimations it was assumed that an adult and a child/teenager would play in the marsh 100 days per

year, for 30 years and 11 years, respectively. To be the most public health protective (precautionary), we used the highest contaminant concentration remaining in the marsh in the dose estimations (it is highly improbable that any person would be exposed to highest level of contaminants remaining in the marsh for the amount of time assumed). None of the estimated doses exceed health comparison values for adults or children/teenagers (Appendix C, Table C-7).

The hazard index is below 1 for adults and children/teenagers. Thus, exposure to all of the contaminants in the marsh would not result in noncancer health effects in adults or children/teenagers (Appendix C, Table C-7).

The estimated BLL for adults from exposure to the highest level of lead (250 ppm) remaining in the marsh is 3.6 µg/dL³. The BLL for women of childbearing age was estimated at 5.7 µg/dL from exposure to the highest level of lead. These levels are below 10 µg/dL for pregnant women and 25 µg/dL for all other adults, the levels at which exposure reduction actions are recommended (29,30).

For contaminants considered carcinogenic, CDPH calculated the theoretical lifetime increased cancer risk for an adult who recreates in the East Stege Marsh for 30 years and a child/teenager who plays in the marsh for 11 years. The theoretical lifetime increased cancer risk for an adult and a child/teenager is 1 in 100,000 and 5 in 100,000, respectively. These are considered “very low increased risks.” Equations and cancer slope factors used to estimate increased cancer risks are provided in Appendix E.

In summary, on the basis of available data, CDPH concludes current and future exposure to sediment in the East Stege Marsh would not result in noncancer adverse health effects in adults or children/teenagers, from exposure to metals. Exposure to the maximum concentrations of carcinogenic contaminants remaining in the marsh poses a “very low increased cancer risk.” To ensure that sediment and surface water in the marsh are not being impacted (re-contaminated) through groundwater or surface water runoff from other areas, sediment and unfiltered surface water should be sampled annually until remedial activities at the site areas are complete.

Due to a lack of data, CDPH was not able to determine whether there is a current or future health risk from exposure to elevated levels of naturally-occurring radionuclides that may be present in non-excavated portions of the marsh. The potential for radionuclide contamination at the Zeneca site, as a result of historic operations at Stauffer, is being investigated further by DTSC and the Radiologic Health Branch of CDPH. Until there is a complete understanding/characterization of the potential radiological issues at the site, access to the East Stege Marsh should remain restricted.

³ As a point of reference, exposure to the highest level of lead remaining in the marsh would result in an estimated BLL of 6.3 µg/dL for a 1-2 year old; the adult BLL is 3.6. It is reasonable to assume that the BLL for a child between 8-18 years old would fall between these two numbers, and below 10 µg/dL.

Evaluation of Potential Exposure to Dust and Site-Related Contaminants in Ambient Air During Remedial Work at Zeneca between October 2002 and October 2006

It is possible for workers in nearby businesses and at the Richmond Field Station and people who use the Marina Bay Trail to have been exposed to dust generated during remedial work at the site. Nearby workers expressed concerns to CDPH staff about inhalation (breathing) of site-related contaminants in dust generated during demolition of on-site buildings and during remedial activities at the site. These exposure groups are generally considered to spend 40 hours per week or less in the vicinity of the Zeneca site. CDPH reviewed available air monitoring data in an effort to understand exposures that may have occurred as a result of these activities.

Demolition of on-site buildings began in 1999 and was completed in 2000. Building 240 (administrative building) is the only remaining building (unoccupied) on the site. From 2000 through 2003, remedial activities were conducted in the upland areas of the site (33). There was no air monitoring conducted during the demolition of on-site buildings or during remedial activities (localized excavations) conducted in the between 2000 and 2001.

Air monitoring for dust and site-related contaminants was conducted during Phase I, Phase II, and Phase III remedial activities in the East Stege Marsh, the upper freshwater lagoon, and the Upland Remediation of Subunit 1 (1,33,34) (Appendix B, Figure B-4). Hydrogen sulfide, a byproduct of the decomposition process in sediments with little or no oxygen, was also measured. Remedial activities consisted of excavation and backfill of sediments from the marsh, and upper freshwater lagoon and localized excavation of soil in the upland portion of Subunit 1 (renamed Lots 1-3) (33,34).

Dust

Dust is made up of various sizes of particulate matter. Particulate matter less than 10 microns in aerodynamic diameter, known as PM 10, is considered among the most harmful of all air pollutants, because when these particles are inhaled, they can become deeply lodged in the lungs, potentially resulting in a number of respiratory and cardiovascular effects (35,36).

Between May 29, 2002, and September 7, 2003, total dust (total suspended particulates) was monitored at three locations around the perimeter of the site (33). PM 10 was not measured. Total dust was measured on 150 days. On 14 days, dust levels exceeded 500 $\mu\text{g}/\text{m}^3$ (limit set by DTSC on other site-related remedial work) at one or more monitoring location.

Between October 12, 2004, and July 17, 2005 (Phase I), real-time dust monitoring and laboratory analysis of total dust and PM 10 were conducted. There were a number of equipment malfunctions noted with the real-time dust monitors. Thus, CDPH focused on the analytical data, as it appears to be more reliable. The following paragraph discusses the analytical air monitoring data collected during the Phase I remedial work.

Total dust was measured at six locations along the perimeter of the site, for varying amounts of time, ranging from 2-160 days, depending on the type and location of work being conducted at the site. Three monitoring stations were located near the Harborfront Business Tract (49th Street businesses) and two stations along the border between Zeneca and the Richmond Field Station. During remedial activities at the site, total dust levels did not exceed $500\mu\text{g}/\text{m}^3$, the limit set by DTSC. PM 10 was measured at three locations along the perimeter of the site for varying amounts of time, ranging from 43-182 days. On three days, PM 10 levels were measured at or above the California 24-hour ambient air standard of $50\mu\text{g}/\text{m}^3$ (36). The highest PM 10 level was measured at $56\mu\text{g}/\text{m}^3$. While these levels do not appear to pose a significant health risk, there is the possibility that elevated PM 10 levels could cause some respiratory irritation, especially for people with pre-existing respiratory conditions.

Between September 12, 2005, and October 31, 2005 (Phase II), and between December 6, 2005, and December 23, 2005 (Phase III), total dust and PM 10 were measured. Total dust was measured at five locations and PM 10 was measured at one location. Two monitoring stations were located near the Harborfront Business Tract (49th Street businesses) and two stations along the border between Zeneca and the Richmond Field Station. Total dust levels did not exceed $500\mu\text{g}/\text{m}^3$, the limit set by DTSC or the California 24-hour ambient air standard of $50\mu\text{g}/\text{m}^3$, for PM 10.

In conclusion, it is possible that, during remedial work conducted between May 2002, 2005, and July 2005, nearby workers and Bay Trail users could have experienced mild irritant effects of the respiratory tract from breathing dust on the days when dust levels were elevated.

Site-related Contaminants in Dust

Between May 29, 2002, and September 7, 2003, air samples were analyzed for 20 COCs, including metals, VOCs, and pesticides. Action levels were set based on occupational standards, meant to be protective of workers exposed to chemicals over an 8-hour time period. None of the COCs were detected above action levels approved by the RWQCB for the remedial action. Metals (arsenic, copper, chromium, mercury, and lead) were detected at low concentrations, below $1\mu\text{g}/\text{m}^3$. VOCs and pesticides were not detected above method detection limits. Detection limits for VOCs and pesticides were below $0.5\mu\text{g}/\text{m}^3$. Potential exposure at these levels (or less) would not pose a health risk to nearby workers or Bay Trail users.

During remedial work conducted in 2004 and 2005 (Phase I-Phase III), samples were analyzed for 42 COCs, including metals, VOCs, aldehydes, pesticides, and PCBs (34). Action levels were set based on an increased cancer risk of 1 in 1,000,000 for carcinogens, and a hazard index of 1 for noncancer health effects. The action levels were approved by DTSC and are protective of both occupational and residential exposure (34). None of the site-related contaminants detected exceed site-specific action levels (Appendix C, Table C-8).

Hydrogen Sulfide

Hydrogen sulfide (H₂S), a byproduct of the decomposition process in sediments with little or no oxygen is commonly found in marsh areas. While Stauffer manufactured a number of sulfur-containing compounds, detections of H₂S appear to be associated with the natural processes occurring in the marsh.

During remedial work conducted in 2004 and 2005 (Phase I- Phase III), hydrogen sulfide (H₂S) levels were occasionally measured at concentrations that could result in temporary health effects (Appendix C, Table C-8). The average H₂S concentration (around 20 µg/m³) over the course of the three phases of remedial work (about 243 days) was below the intermediate (exposures occurring from 14-364 days) MRL of 27.9 µg/m³. There were 10 days during Phase I and 3 days during Phase II remedial work when H₂S concentrations in the marsh area exceeded the California Air Resources Board (CARB) 1-hour threshold of 42 µg/m³ (37). While these levels do not appear to pose a significant health risk, there is a possibility that exposures to elevated levels of H₂S levels could have occurred on several days. Health effects associated with H₂S exposure include irritation of mucous membranes and the respiratory tract, nausea, headaches, and skin and eye irritation. These effects are considered temporary and should subside once the exposure ceases.

Soil Gas/Vapor Intrusion

It is possible for indoor air in Harborfront Tract businesses and in Building 240 (formerly used for the youth afterschool program, Making Waves Education Foundation⁴) on the Zeneca site, to be affected by groundwater contaminated with VOCs in those areas. Building 240 is no longer occupied or utilized.

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 Zeneca 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 (38,39). Typical indoor air is not considered healthy and contains many chemical constituents, which come from various sources, such as household products, cooking, building materials, and influences from the outdoors.

⁴ Beginning in 2002, Making Waves held its afterschool program in Building 240 on the Zeneca site. Approximately 250 children participated in the program, which were held on weekdays and Saturdays. (Michael McCanta, Making Waves Education Foundation, personal communication, October 4, 2005). As of July 2006, the Making Waves program was no longer permitted to operate on the Zeneca site and has since relocated to another location in Richmond.

Several types of environmental data can be used to evaluate the potential for soil gas to migrate into buildings. These data include indoor air, groundwater, soil, and soil gas sampling. The Johnson and Ettinger (J&E) model can be used to evaluate the potential for soil gas migration into indoor air and whether risk-based exposure levels could be exceeded (40). The J&E soil gas model estimates indoor air concentrations from soil gas data. The J&E groundwater model estimates indoor air concentrations from chemicals measured in groundwater. The model does not predict precise concentrations to be used for interpretation of potential health effects, but rather concentrations for screening purposes to determine the need for further action (40).

CDPH reviewed available information to determine whether soil gas is impacting the indoor air quality in nearby businesses located in the Harborfront Tract and in Building 240 (B-240), at levels posing a health risk. First we will describe the data used to evaluate potential soil gas impacts for the Harborfront Tract and then B-240.

Evaluation of Potential Impacts to Indoor Air in Harborfront Business Tract from Vapor Intrusion

CDPH reviewed three sources of information to evaluate the potential for vapor intrusion to be impacting indoor air in the Harborfront Business Tract. The information includes results of soil gas sampling, groundwater sampling and modeling conducted by DTSC (41,42).

During October 2005 and November 2005, DTSC's environmental contractor, Weiss Associates, collected soil gas samples and groundwater in the Harborfront Tract area (41). Soil gas samples were collected from 29 locations, at depths ranging from 3-5 feet bgs (Appendix B, Figure B-5). Groundwater samples were also collected at these locations.

With the exception of benzene, soil gas results do not exceed soil gas screening values (Appendix C, Table C-9). However, none of the soil gas samples were collected from underneath the slabs (within footprint of building) of the businesses, which limit the utility of using these data alone for evaluating vapor intrusion into businesses. Soil gas samples collected outside of the building footprint provide information about diffusion (vapors migrating through the soil column), but they are not representative of vapor migration that can occur as a result of pressure differences caused by buildings that pull/draw vapors from the subsurface into buildings.

Several VOCs were measured in groundwater, with maximum detected concentrations as follows: 1,2-dichloroethane (DCA) 29 ppb; 1,1-dichloroethene (DCE) 7.8 ppb; 1,2,4-trimethylbenzene 0.8 ppb; trichloroethylene (TCE) 140 ppb; trichlorofluoromethane 9.2 ppb; and gasoline 110 ppb. We have included a figure showing the concentration contours of TCE in shallow groundwater, the highest VOC detected, as a point of reference (Appendix B, Figure B-6).

DTSC conducted vapor intrusion modeling using soil gas and groundwater models to evaluate the potential impacts to indoor air quality in businesses in the Harborfront Tract (42). DTSC's evaluation indicates that the air quality in businesses in the Harborfront Tract is not being impacted by contaminants through vapor intrusion at levels of concern for noncancer and cancer health effects. CDPH concurs with the findings presented by DTSC.

Evaluation of Potential Impacts to Indoor Air in Building 240 from Vapor Intrusion

In March 2005, the Making Waves Education Foundation hired PES Environmental, Inc. to conduct indoor air sampling in B-240 (43). Samples were analyzed for limited number of contaminants (Appendix C, Table C-10). Most of the VOCs analyzed were not detected above laboratory detection limits. The laboratory method used for the analysis was not very sensitive, resulting in relatively high detection limits. The limitations with these data prohibit our ability to make comparisons with concentrations of VOCs typically found in indoor air and those that may be due to soil gas migration. However, these data are adequate for assessing potential health risk. A number of site-related contaminants were not analyzed during the sampling of indoor air. Therefore, CDPH used soil gas data to augment the indoor data for contaminants not analyzed for in indoor air.

In August 2005, contractors for Cherokee Simeon conducted soil gas sampling around Building 240 (2). Two types of sampling analysis were conducted: 1) samples were analyzed using a mobile laboratory; and 2) samples were collected in a Summa canister and then sent to a fixed laboratory. A number of VOCs were detected in soil gas (Appendix C, Table C-11). Benzene was the only VOC detected above residential soil gas screening values (Appendix C, Table C-11).

To evaluate whether soil gas is in indoor air at levels posing a long-term health threat, CDPH compared the concentrations measured in indoor air to health comparison values (Appendix C, Table C-10). None of the measured VOCs, including benzene, exceed health comparison values, even if it is assumed that all of the VOCs analyzed were measured at the detection limit, which they were not. Thus, exposure to the VOCs analyzed in the indoor air would not be expected to have resulted in noncancer health effects in students or staff from Making Waves.

CDPH did not calculate a theoretical increased cancer risk for students and staff of Making Waves from VOCs considered carcinogenic (cancer-causing) because science does not support estimating cancer risks for short-term exposures, as these estimates may misrepresent the actual risk (44). One reason is that cancer slope factors are developed from studies that look at exposures over a long period of time (many years). CDPH used a minimum 9-year exposure duration as a basis for estimating theoretical increased cancer risks (45). Building 240 was used by Making Waves for 4 years.

In conclusion, on the basis of available data, it does not appear that indoor air in businesses in the Harborfront Tract or in B-240 poses a health hazard to workers from vapor intrusion, as a result of Zeneca operations.

Evaluation of Exposure to Contaminants in Indoor Dust in Building 240

It is possible that windblown dust generated during remediation of contaminated soils at the site could have entered B-240, where students and staff of the Making Waves program could have come into contact with the dust. Community members expressed concern that children attending Making Waves were being exposed to site-related contaminants in dust. There was no data available to address these concerns. As a result of this data gap, CDPH conducted an exposure investigation of indoor dust in B-240. The CDPH exposure investigation protocol, which described the selection of contaminants to be analyzed, the development of site-specific health comparison values for dust, and other sampling parameters, can be viewed at <http://www.ehib.org/cma/projects/ZenecaEI.pdf>.

In April 2006, CDPH conducted indoor dust sampling in B-240, consisting of both vacuum dust and surface wipe samples. Eleven samples were collected from areas utilized by students and staff of Making Waves (Appendix B, Figure B-7). CDPH compared the results of the dust sampling to health comparison values developed for the Exposure Investigation of B-240 dust (Appendix C, Table C-12) (46). None of the contaminants measured in vacuum dust or surface wipe samples exceed site-specific health comparison values derived for dust (Appendix C, Table C-12).

PCB analysis was not part of the original workplan. However, during the sample analysis, the laboratory reported observing “peaks” associated with PCBs. As a result, CDPH instructed the lab to run PCB congener-specific analysis. Each congener has two or more chlorine atoms located at specific sites on the PCB molecule. The PCB congener-specific analyses measure the concentration of each congener in the sample (Appendix C, Table C-13) (46). It is worth noting that while PCBs are a site-related contaminant, they are also often found in older buildings. Prior to 1977, PCBs were used in the manufacture of caulking used to seal joints around windows and between masonry joints.

Using the same protocol described in the exposure investigation protocol for the targeted pesticides and metals, we developed a dust health comparison value for PCBs to use as a comparison in evaluating the amount of PCBs found in the dust. Instead of having a health comparison value for each congener, CDPH used the toxic equivalent factor approach to obtain a single health comparison value for PCBs in the dust ($0.04 \mu\text{g}/\text{m}^2$). The toxic equivalent factor approach compares PCB congeners to the relative toxicity of dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin), since some PCB congeners behave like dioxin in the body. PCBs measured in vacuum and surface wipe samples do not exceed health comparison values derived for dust (Appendix C, Table C-14).

In conclusion, students and staff who attended the Making Waves program in B-240 were not exposed to site-related contaminants in dust at levels of health concern.

Quality Assurance and Quality Control

In preparing this PHA, CDPH used information in the referenced documents and assumed that adequate quality 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 PHA are prepared for regulatory agencies, which undergo review to ensure that proper quality control measures for laboratory procedures and reports were followed.

Community Health Concerns and Evaluation

Introduction and Purpose

The collection, documentation, and responses to community health concerns are a vital part of the PHA process. This section describes outreach efforts in more detail and characterizes the main past and present exposure and health concerns reported to CDPH. In addition, this section includes an evaluation the community's health concerns based on available scientific literature, within the framework and limitations of the PHA.

A variety of neighboring businesses and residents have been located next to the site through its operating and non-operating years. Efforts to document health concerns relevant to the Zeneca site included outreach to people who lived or worked near the site in the past, and people who currently live or work there.

In 2004, due to concerns about the rigor of oversight, community members advocated for a change in the regulatory agency overseeing the cleanup (47). In July 2004 and February 2005, the Contra Costa County Health Services Department requested DTSC's oversight of remediation at both the Zeneca and UC Richmond Field Station sites (48,49). In May 2005, DTSC became the lead agency overseeing cleanup (50). With the involvement of DTSC, a Community Advisory Group (CAG) was established (17).

Process for Gathering Community Health Concerns

In early 2005, the Contra Costa County Health Services Department requested the assistance of CDPH in evaluating potential risks to health from the Zeneca and adjacent UC Richmond Field Station site. While preparing the PHA, CDPH worked with the Contra Costa County Health Services Department to evaluate any immediate threats from the Zeneca site and the adjacent UC Richmond Field Station site in a Provisional Health Statement, which was updated as new information became available (51,52).

CDPH staff briefed the Zeneca CAG about the health assessment process in October of 2005, and worked with CAG members to identify nearby neighborhoods and businesses, and former residents of the Seaport apartments for outreach. In December 2005, CDPH placed a Public Notice in various local newspapers outlining the collection of past and

present exposure and health concerns related to the site. Throughout 2006 and 2007, CDPH continued to receive community concerns.

A variety of people reported health and exposure concerns, including former Seaport residents, former Stauffer workers, former workers of lessees of the Zeneca property, and former residents of nearby neighborhoods. The exposure and health concerns are described in chronological order: 1944-1956, when people resided at the Seaport apartments; 1957-1997, the period of time between the Seaport years and the 1997 on-site demolitions, including a timeframe during which employees of a neighboring facility maintained a log of concerns; and 1997-present, when neighboring businesses and residents were concerned about on-site demolitions and exposure during remedial activities.

1944-1956: Concerns of Residents of the Seaport Warhousing Apartments

Former residents of Seaport recalled chemical smells from Stauffer as a recurring event. Many noted that it was difficult to breathe the air. Several former Seaport residents recalled playing in the evaporation ponds as children; one particular incident involved boys who threw rocks into the ponds trying to splash each other with the water from what they called the “poison lakes.” One person recalled going fishing near Stauffer between 1951 and 1956, and was concerned about exposure to toxic chemicals via contaminated fish such as striped bass, sting ray, and jack smelt.

One former Seaport resident stated that Seaport residents suffered from skin breakouts and rashes, as well as eczema. Other ailments mentioned were rheumatic fever, scarlet fever with rashes, whooping cough, goiters, polio, tuberculosis, and emphysema.

Some former seaport residents reported ailments later in life such as non-Hodgkin lymphoma, lupus, endometriosis, uterine cancer, precancerous lesions on the face cheeks, nose, and arms, and muscular and joint conditions. Various former Seaport residents have described similar exposure and health concerns in other interviews (16,53,54).

1957-1997: Former Stauffer Workers

Several former Stauffer workers reported health and exposure concerns. One recalled the storage of rusting 55-gallon drums of chemical intermediates less than 100 feet from San Francisco Bay; the worker believed the drums were rusty and leaking. Another former Stauffer worker described poor occupational training in avoiding exposure to chemicals; the worker was concerned about the long term health impacts of exposure to Stauffer chemicals.

1957-1997: Former Neighbors and Residents

A former resident of Crescent Park reported smelling sulfuric acid in the air and a ‘chlorinating’ scent when the resident lived in the area between 1969 and 1970. Several business owners adjacent to the site reported that Stauffer routinely tested run-off water

from its site. In two cases, business owners reported that Stauffer took water samples from their water wells, inquiring how the water was used. In one of these cases, the business owner stated that Stauffer recommended that water from the wells not be used for drinking.

1961-1972: Historical Log of Odors and Health Concerns

Employees of a neighboring facility kept a log of Stauffer-related odors and health concerns from 1961 to 1972. It appears that approximately 68 unique individuals contributed to the log, although this is difficult to ascertain because entries were sometimes signed using full names and at other times using only initials. Entries varied from detailed descriptions of odors and health concerns to simply names of people reporting odors of unspecified nature. It appears that the log was more routinely updated during certain years, namely 1965 and 1966. It is unclear if this is due to stronger emissions that elicited a greater response during those years or to more diligent entries by staff during those years.

Almost 300 entries in the log related to the odors of emissions coming from Stauffer (see Table 1 below). The odors were most commonly described as sulfur or sulfur-like. Other common descriptions were garlic- and onion-type odors. Odors were often characterized as obnoxious, offensive, and/or disagreeable, without further descriptions of their specific qualities. People reported tasting chemicals at various points; the reported tastes were described primarily as ‘metallic’ in the years 1965 and 1969, while the 1967 entries characterized the taste as sulfuric.

The most common health concerns reported were nose irritation and nose bleeds, headaches, throat irritation, and nausea. Eye irritation and sneezing were also frequently described. During 1965, the year in which the log appears to have been more routinely updated, a total of 195 health concerns were reported (see Table 2 below for a list of health concerns).

Table 1. Characteristics Used in Historical Log to Describe Emissions from Stauffer

	1961											1972	Total Types of Smells Described
Garlic		6				4	2	3	1	1			19
Onion		2		5	11	4							22
Garlic-Onion				2	11	14							25
Sulfur			1		20	8	8	5	7				49
Mercaptan					1	2	4	1					19
Tannery					6	5							11
Can taste smell	8		1		11	6	3	1	12				22
Obnoxious/ Offensive/ Disagreeable			1	3	10	6	5	20	1	1			47
Other			1	2	6	15	4	13	4	3	2	2	58
Total smell entries per year	0	22	3	11	78	64	26	43	14	7	2	2	

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Table 2. Health Concerns/Effects Noted in Historical Log

	1961											1972	Total Types of Health Concerns
Headaches			1	4	27	10	2	5	4	4	2	1	60
Throat irritation				1	30	15	2	3	2				53
Nose irritation/nose bleeds				2	42	20	10	2				1	77
Eye irritation				1	20	12	5	3				2	43
Sneezing / Coughing					21	10	1	1	2				35
Nausea					18	10	2	8	5	3	2		53
Vomiting		1	1	1	2	1		1					7
Difficulty breathing			5		4	3	2		1				10
Sick / Affected			1	4	6	7		3	1				22
Other					25	15	2	5	2	2			54
Total health concerns per year	0	1	3	21	195	103	26	31	17	9	4	4	

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In addition to describing odor and health concerns, those who updated the log sometimes included descriptions of unusually powerful emissions, communications with Stauffer staff, and other items worth noting. Table 3 provides some of the more vivid examples of these instances included in the log.

Table 3. Examples of Qualitative Accounts of Stauffer Emissions Reported in Historical Log

Date	Activity/Comment
1/8/1962	"leak in heat exchanger"
11/29/1962	"spill in ethylmercaptan"
5/14/1965	"had to wash windshield every day this week on account of fallout from material going over"
5/25/1965	"[Stauffer employee] came over (5/25/65) reported that the fumes were coming from superphosphate mill which will be shut down the 19 th , and asked if we would check on the situation during shutdown and after reopening."
6/9/1965	"Excessive fallout on parked cars."
6/10/1965	"cars are covered with white dust"
6/11/1965	"[Stauffer employee] phoned that they had a breakdown in their Mercaptan production but were decontaminating as rapidly as possible. However, fumes stayed. Very bad all day. All office staff coughed and reported headaches."
6/28/1965	"My clean car was coated with whitish dust that has to be washed off."
7/6/1965	"Wind bringing fumes and dust right over trees and down in parking area."
7/15/1965	"My husband said I smelled like Stauffer when I got home last night."
8/23/1965	"Heavy emission became very annoying and persisted for the next two hours...Large clouds were spuing forth from 3 or 4 sources."
8/24/1965	"Participants in NSF program have been very much affected and have remarked that they do not know how we stand it."
9/10/1965	"Now have very sore throat and nose is oozing blood."
11/1/1966	"Smell to the point where I am wondering if building should be evacuated." "Strongest smell ever in 3-1/2 years."

It is not possible to determine the amount and exact type of contaminants that were emitted during the years the log was maintained, due to a lack of data. However, the odors described in the historical log are consistent with descriptions of odors associated with chemicals (ammonia, mercaptans, sulfur compounds, thiophenol, etc.) used or produced in the manufacturing that occurred at the Stauffer facility (phosphate fertilizer and pesticides) (5,22,55,56).

There is a very limited understanding of the short and long term health effects from these types of exposures. A 1960 study found that children living near a large superphosphate manufacturing plant were 17 times more likely to have upper respiratory disease compared with children living further away from the plant (57). However, the long term implications of these types of exposures are not known.

1997-Present

Neighboring business owners and residents were concerned about exposure to dust during

remedial activities at the Zeneca site. Several business owners and workers were concerned about exposure to dust generated from activities. Many reported having to have their cars washed as a result, and many were concerned about potential exposure to dust among children and staff in the afterschool program operating out of Building 240 at the Zeneca site.

During the remedial activities, people reported feeling burning eyes, headache, sore throat, and pain in their lungs. Business owners and workers also became concerned about cases of endometriosis, ovarian cysts, destabilization of a previously-stabilized thyroid condition, and cancer.

Currently, community members are primarily concerned about potential exposure to contaminants during future remedial activities, and that adequate cleanup ensures the safety of future populations who interact with the land. Community members are also concerned about the lack of historical data outlining past activities of the site in detail.

Evaluation of Community Health Concerns

CDPH collected health concerns throughout late 2005 and 2006. CDPH collected concerns through personal interviews and via phone, mail and electronic mail. A health survey was not conducted; health concerns were documented in an open ended manner.

People reporting concerns were former residents of the Seaport Warehousing Apartments, former and current residents of other nearby neighborhoods (including Crescent Park, Richmond Annex, Panhandle, and Marina Bay), people who owned and worked at businesses that had leased land on the Zeneca property as well as neighboring businesses, and former Stauffer workers.

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 Zeneca. 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 Zeneca.

Some community members documented illnesses and deaths in the area. After removing identifying information, they shared that list with CDPH. The information was collected anecdotally and comprised of 25 cases.

CDPH evaluated the health effects by investigating their known causes, including environmental or chemical agents. The evaluation of cancer concerns includes an overview of cancer risk factors and health disparities. We are not able to draw a link between the health effects expressed to CDPH and contaminants at the Zeneca site for a number of reasons: first, the environmental data needed to understand potential exposures is not available; toxicological information on chemicals is limited; there is limited understanding of the effects from exposure to multiple chemicals; and there are many factors that contribute to causation of a disease, making it almost impossible to identify a specific or single factor, such as an environmental exposure.

Table 4 shows the health concerns and effects reported to CDPH; health effects are organized as either related or not related to cancer.

Table 4. Cancer and Noncancer Health Concerns Reported to CDPH

Cancer Concerns/Effects	Noncancer Concerns/Effects
<i>Bladder cancer</i> Breast Cancer Chondroma/Chondrosarcoma Kidney cancer Liver cancer Lung cancer <i>Multiple myeloma (cancer of the bone marrow)</i> Non-HodgkinLymphoma Pancreatic cancer <i>Prostate cancer</i> Rectal cancer <i>Throat cancer</i> <i>Thyroid papillary carcinoma</i> Skin Cancer Stomach Cancer Uterine cancer	<i>Amyotrophic lateral sclerosis</i> Anemia Asthma <i>Autoimmune disorders</i> Breathing difficulties Chicken Pox Eczema Elephantitis Emphysema Endometriosis Goiters <i>Headaches</i> Heart attack Kidney problems Lipoma fatty tumors in the abdomen Lupus <i>Pancreatitis</i> Polio Pre-cancerous lesions on skin <i>Ovarian cysts</i> Rapid weight loss Rheumatic Fever Thyroid Nodules <i>Thyroid disorders</i> Tuberculosis <i>Uterine bleeding</i> Uterine tumors <i>Uterine fibroid tumors</i> Whooping Cough

Items in *italics* denote health concerns/effects documented by community members.
 All other concerns listed were collected by CDPH.

Cancer Risk Factors and Health Disparities

Cancer as a whole is the second leading cause of death in the United States after heart disease. 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; poor diet; and exposure to another cancer-causing agent, like asbestos.

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 (58).

Age is another important risk factor. 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 a 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 (59). Cancer health disparities occur as a result of differences in income, education, access to healthcare, lifestyle, and/or environmental and biological factors (59). 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 (59).

Evaluation of Cancer Health Concerns at the Zeneca Site

As outlined in Table 4, the cancer concerns reported to CDPH were: chondroma/chondrosarcoma, bladder cancer, breast cancer, kidney cancer, liver cancer, lung cancer, multiple myeloma (cancer of the bone marrow), non-Hodgkin lymphoma, pancreatic cancer, pre-cancerous lesions on skin, prostate cancer, rectal cancer, throat cancer, thyroid papillary carcinoma, skin cancer, and uterine cancer. A description of each of these cancer types follows, along with its known causes and risk factors. A risk factor is something that may increase the chances that someone will develop an illness. However, having a risk factor does not guarantee that the person will develop an illness. Even if a person has several risk factors, he/she may never develop the illness (60). Some risk factors can be avoided or controlled, such as one's diet, level of physical activity, and use of tobacco. Other risk factors such as family history or genetics cannot be avoided.

In this section, CDPH evaluated potential environmental links to illnesses by searching for COCs in the Collaborative on Health and the Environment's Toxicant Disease Database. The database lists illnesses associated with contaminants and vice versa.

Current contaminants of concern for the Zeneca site are based on limited data about conditions at the site in the present and the recent past. It is likely that other contaminants may have been present in the past, particularly during the years Stauffer operated.

Compounds other than current Zeneca COCs may be associated with the health concerns evaluated here; because other compounds were not evaluated, the list of chemicals potentially associated with a health concern is not exhaustive.

The Collaborative on Health and the Environment categorized the amount and quality of evidence linking contaminants to health outcomes as “strong,” “good,” and “limited,” where strong means a causal association has been established; good means an association is being established; and limited means an association has begun to be suggested.

Bladder Cancer

The bladder is an organ that stores urine; it is located within the pelvis. Bladder cancer occurs in the lining of the bladder; it is the sixth most common type of cancer (61). Smoking is the greatest risk factor for bladder cancer because carcinogens in cigarettes are absorbed from the lungs into the blood, filtered by the kidney, and eventually end up in urine where they damage the lining of the bladder (61). Industrial chemicals sometimes used in the dye industry (such as benzdine and beta-naphthylamine) can cause bladder cancer. Other industries with high risk of bladder cancer include the rubber, leather, textile and paint industries (61). Painters, hairdressers, machinists, printers, and truck drivers also have an increased risk of developing bladder cancer (61). The Collaborative on Health and the Environment cites strong evidence linking bladder cancer to arsenic, and limited evidence linking bladder cancer to antimony and lead (62). Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with bladder cancer.

Breast Cancer

Breast cancer is the second most common type of cancer among women in the United States (63). The number of breast cancer cases among men is small. Breast cancer symptoms include a lump in the breast, a change in the shape or size of a breast, and/or nipple discharge (63). It is highly recommended that women perform breast self-exams and mammographies to identify breast cancer in its early stages, when it is more treatable. Treatment for breast cancer can include radiation, chemotherapy, hormone therapy, removal of a tumor, or removal of the entire breast (63). Some known risk factors for developing breast cancer are age (older women have a higher risk); genes; having one's first period before age 12; going through menopause after age 55; being overweight; using hormone replacement therapy, taking birth control pills; drinking alcohol; not having children; having a child after age 35; and having dense breasts (63). The Collaborative on Health and the Environment cites the strength of the evidence linking breast cancer and PCBs as “good” (62). Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with breast cancer.

Chondroma/Chondrosarcoma

Chondrosarcoma is a type of bone cancer that begins in the cartilage (64). Cancer that begins in the bones is also known as primary bone cancer. Primary bone cancer is rare (64). Secondary bone cancer is more common; it occurs when a cancer spreads to the bone from another part of the body (64). The most common symptom of bone cancer is pain; swelling or tenderness may also be present, along with fatigue, fever, weight loss, and anemia (64). Some bone cancers appear in youth (osteosarcoma and Ewing's sarcoma) (64). Chondrosarcoma occurs more

commonly after the age of 50, and is known to be located in the pelvis, upper legs, and shoulders (64).

Very little is known about the causes of bone cancer. Some risk factors are known for bone cancers that occur during childhood but data about chondrosarcoma is scarcer (64). A recent multinational study found increased bone cancer among blacksmiths, toolmakers, machine-tool operators, woodworkers, and construction workers, as well as people who reported using herbicides and pesticides; however, no data was available to determine what specific chemicals they might have been exposed to or in what amounts (65).

The Collaborative on Health and the Environment did not have a listing for chondrosarcoma.

Kidney Cancer

The kidneys are a pair of organs located in the lower abdomen, on either side of the spinal column (66). The kidneys remove waste and extra water from the blood, and turn this excess into urine. Cancer of the kidneys most often occurs in people over 40. The causes of kidney cancer are not known, but some risk factors include smoking, obesity, high blood pressure, long-term use of dialysis, gender (men are more likely to be diagnosed), Von Hippel-Lindau syndrome (a disease that runs in some families), and occupational exposure to asbestos and cadmium (66). Oven workers in the iron and steel industry are also at risk (66). Treatment for kidney cancer may include surgery, chemotherapy, radiation therapy, biological therapy, and arterial embolization, a process in which an artery is blocked by a foreign material to stop the flow of blood to a tumor (67).

The Collaborative on Health and the Environment states that there is a good amount of evidence linking kidney cancer to arsenic and a limited amount of evidence linking kidney cancer to arsenic, cadmium, lead, mercury, and nickel (62). Kidney cancer was associated with arsenic exposure in drinking water in a 2004 Taiwanese study, although exposure information is not available (68). Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with kidney cancer.

Liver Cancer

The liver filters harmful substances from the blood, digests fats from food, and stores sugar that the body uses for energy; it is the largest organ in the body (69). Symptoms of liver cancer can include yellowing of the skin and a lump or pain on the right side of the abdomen, although symptoms could be absent altogether until the cancer has reached later stages (69). Liver cancer can be treated through options such as surgery, radiation, chemotherapy, or liver transplantation (69). Some risk factors for liver cancer are cirrhosis of the liver, long term infection with Hepatitis B and C, smoking, and obesity (70). Hepatitis C and alcohol abuse are the leading causes of cirrhosis, and 80% of liver cancer cases are associated with cirrhosis (70).

The Collaborative on Health and the Environment cites a good amount of evidence linking liver cancer to arsenic and PCBs, and a limited amount of evidence linking liver cancer with toxaphene (62). Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with liver cancer.

Lung Cancer

Lung cancer is the leading cause of death among all cancers in the United States for both men and women (71). Lung cancer symptoms include a persistent cough, chest pain, hoarseness, sudden onsets of wheezing, shortness of breath, weight loss and diminished appetite, persistent respiratory infections, coughing up blood, and fatigue (71).

Most lung cancers (87%) are related to smoking and second-hand smoking (71). Other risk factors include exposure to arsenic, asbestos, radioactive dust, or radon, as well as radiation exposure. Family history of cancer is also considered a risk factor (71). An study funded by the Florida Phosphate Council found no large excess of lung cancer related to workplace exposures among Florida phosphate industry workers (72). Lung cancer treatment may include radiation therapy, chemotherapy, or surgery (71). The Collaborative on Health and the Environment cites a strong body of evidence associating lung cancer with nickel and arsenic, a good amount of evidence associating lung cancer with copper; and limited evidence associating lung cancer with lead and antimony. Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with lung cancer.

Multiple Myeloma

The bone marrow is the soft, inner part of bones where plasma cells are located (73). Plasma cells produce antibodies, which help remove bacteria and viruses that cause diseases (73). Multiple myeloma is cancer of the bone marrow that occurs when plasma cells reproduce in an out-of-control fashion, creating multiple tumors within the bone marrow (73). Multiple myeloma occurs primarily among adults in their early 60s, with only 1%-10% of cases occurring among people under the age of 40 (74).

The causes of multiple myeloma are unknown at this time. Some risk factors have been reported in the scientific literature. The American Cancer Society reports that “exposure to radioactivity has been suggested as a risk factor [for multiple myeloma] but accounts for a very small number of cases” (73). Workers in petroleum industries have also been found to have a higher risk of developing multiple myeloma (73). Multiple myeloma is twice as common among African Americans as among White Americans, though the reasons for this disparity are unknown (75). Multiple myeloma is also more prevalent among people who are overweight (73). The Collaborative on Health and the Environment lists a good body of evidence linking pesticides to multiple myeloma; the link between multiple myeloma and numerous other environmental compounds is also being established.

Non-Hodgkin's Lymphoma

The lymphatic system is composed of a vast drainage network that aids in immunity. The lymphatic system carries lymph—“a clear, watery fluid containing protein molecules, salts, glucose, urea, and other substances” (76). Small masses of tissue in the network, called lymph nodes, contain white blood cells that aid fight infections. One type of lymphatic cancer is called Hodgkin's disease. The others are known as “non-Hodgkin's” (77). About 54,000 new diagnoses of non-Hodgkin's lymphoma are made each year in the United States (78). Some symptoms of non-Hodgkin's lymphoma are swollen, painless lymph nodes in the neck, armpits, or groin; unexplained weight loss; fever; heavy night sweat; coughing, trouble breathing or chest pain;

chronic weakness or tiredness; and pain, swelling, or a feeling of fullness in the abdomen (77). Most people who are diagnosed with non-Hodgkin's lymphoma are over 60 years old (79).

Two known risk factors for developing non-Hodgkin's lymphoma are a weak immune system (such as from an inherited condition, HIV infection, or certain drugs), and having had certain infections (such as HIV, Epstein-Barr virus, H. pylori, hepatitis C, and Human T-cell leukemia/lymphoma virus) (79). According to the National Cancer Institute, workers routinely exposed to herbicides or other chemicals may be at risk of non-Hodgkin lymphoma (79). The Collaborative on Health and the Environment cites limited evidence linking toxaphene to non-Hodgkin's lymphoma and limited evidence associating non-Hodgkin's lymphoma to PCBs. Various other compounds are also associated with this disease.

Pancreatic Cancer

The pancreas is an organ located behind the stomach that releases enzymes that help in digestion as well as the hormones insulin and glucagon, which have an effect on blood sugar levels (80). Symptoms of pancreatic cancer include yellowing of the skin and eyes, abdominal pain, back pain, weight loss, and fatigue (81). Pancreatic cancer is difficult to detect early because the symptoms are vague. Also, pancreatic tumors cannot be seen or felt during routine medical exams because the pancreas is located behind other organs (81). Treatment of pancreatic cancer might include surgery, radiation, or chemotherapy (81).

Smoking, chronic inflammation of the pancreas, certain hereditary disorders, and having diabetes for a long time are all considered risk factors for developing pancreatic cancer (81). The Collaborative on Health and the Environment cites a good amount of evidence linking pancreatic cancer to PCBs and limited evidence associating it to cadmium (62). Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with pancreatic cancer.

Prostate Cancer

The prostate gland is found in the male body; it produces fluid for semen (82). Cancer of the prostate occurs most commonly in men over 40; among men of all ages, it is the third leading cause of cancer-related death. Most prostate cancers are diagnosed before symptoms appear, through the routinely performed prostate specific antigen test (82). Prostate cancer could cause symptoms such as painful urination, low back pain, and pain with ejaculation (82). Treatment for prostate cancer can include surgery, radiation therapy, chemotherapy, or hormone therapy (82).

Some known risk factors for prostate cancer are age (the chance of developing it increases as a man gets older); family history (there is a higher risk if one's father or brother had prostate cancer); race (it is more common in African American men and less common in Asian and American Indian men); diet (a diet high in animal fat and meat increases the risk); and having had high-grade prostatic intraepithelial neoplasia, a change in prostate cells (83). One recent study found an increased risk of prostate cancer among men who had been employed in chemical manufacturing (84). The Collaborative on Health and the Environment cites a limited amount of evidence linking prostate cancer to cadmium and limited body of evidence linking prostate cancer to nickel. Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with prostate cancer.

Rectal Cancer (also called colon cancer or colorectal cancer)

The colon is the first 4-5 feet of the large intestine, and the rectum is the last several inches; they are part of the digestive system (85). The colon removes water and nutrients and turns the leftover material into waste; waste exits the body through the rectum and then the anus (85). Cancer of the colon is the fourth most common type of cancer among both men and women (86).

Although the exact causes of colorectal cancer are unknown, some known risk factors include being over the age of 50, having growths on the inner wall of the colon or rectum (colorectal polyps), having a family history of colorectal cancer, having ulcerative colitis or Crohn's disease (conditions that cause inflammation of the colon over a period of years), smoking cigarettes, and having a diet high in animal fat and low in calcium, folate, and fiber (86). The Collaborative on Health and the Environment cites a limited amount of evidence linking prostate cancer to cadmium, nickel, and PCBs. Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with colo-rectal cancer.

Stomach Cancer

The stomach is a hollow organ located in the upper abdomen that contains and liquefies food as part of the digestive process (87). Often, stomach cancer does not present symptoms until it has grown. These symptoms include "discomfort in the stomach area, feeling full or bloated after a small meal, nausea and vomiting, and weight loss" (87). Although the cause of stomach cancer is unknown, some risk factors for developing stomach cancer include being older, being male, being Asian, Pacific Islander, Hispanic or African American, eating foods that are smoked, salted, or pickled, smoking, and having had stomach conditions such as inflammation and ulcers (87). The Collaborative on Health and the Environment cites a good amount of evidence linking stomach cancer to nickel and limited amount of evidence linking stomach cancer to lead. Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with stomach cancer.

Throat Cancer

Cancer of the throat refers to cancer of the vocal cords, voice box, or other areas of the throat (88). Some symptoms of throat cancer include chronic hoarseness and sore throat, neck pain and/or swelling, difficulty swallowing, coughing up blood, weight loss, and high-pitched breathing sounds (88). Most throat cancers occur among men, and throat cancers usually occur in people over the age of 50 (88). There is an increased risk of developing throat cancer among people who smoke or chew tobacco and among people who drink alcohol; people who do both have a much greater risk of developing throat cancer (88). The National Cancer Institute estimates that 85% of head and neck cancers are linked to tobacco use (89). Other possible risk factors include poor oral hygiene; Plummer Vinson syndrome—a rare syndrome that results from nutritional deficiency; and workplace exposure to asbestos (89). The Collaborative on Health and the Environment does not provide a listing for throat cancer.

Thyroid Papillary Carcinoma

Most diagnosed thyroid cancers are papillary carcinoma, and typically occurs in people between the ages of 20 and 40 (90). It appears more often in women than men (90). A small nodule in the thyroid gland is the first symptom. The cause of thyroid papillary carcinoma is unknown.

Papillary thyroid cancer has been linked to high dose external radiation to the neck (90). It has also been linked to people exposed to radioactive fallout in radiologic testing and disasters (90,91). Family histories of thyroid cancer, goiters, or colon growths are also risk factors (91). Iodine is being investigated as a possible risk factor; iodine is a substance found in shellfish and iodized salt (91). Too much iodine in the diet may be a risk factor for developing papillary thyroid cancer; on the other hand, too little iodine may increase the risk of another type of thyroid cancer (follicular thyroid cancer) (91). No COCs for the Zeneca site are listed as being associated with thyroid papillary carcinoma; however, the Collaborative on Health and the Environment cites limited evidence associating this cancer other compounds, including pesticides and polybrominated diphenyl ethers (PBDEs)._

Skin Cancer

The most common type of cancer in the United States is skin cancer (92). There are two types of skin cancer—melanoma and nonmelanoma. Nonmelanoma skin cancer is the more common type of skin cancer. Melanoma is less common, but more dangerous; it occurs when cancer forms in the skin cells that make pigment (92,93). Skin cancer occurs more frequently among people who are exposed to the sun, have light colored skin, hair and eyes, are over 50 years old, and have a family history of skin cancer (92). Exposure to ultraviolet radiation, which comes from the sun, sunlamps, tanning beds, or tanning booths, is a known risk factor for skin cancer (94). Other known risk factors are scars or burns on the skin, infection with some types of human papilloma viruses, workplace exposure to arsenic, chronic skin inflammation or ulcers, diseases that make the skin sensitive to the sun, radiation therapy; suppressed immune system, family history of skin cancer, actinic keratosis, and Bowen's disease—a disease in which the skin becomes scaly and thick (94). The Collaborative on Health and the Environment characterizes the evidence linking arsenic to non-melanoma skin cancer as strong and limited evidence linking PCBs to melanoma skin cancer. Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with skin cancer.

Uterine Cancer

The uterus is the place in a woman's body where a baby grows when a woman is pregnant. The lining of the uterus is known as the endometrium; the most common type of uterine cancer starts in this lining (95). Uterine cancer is also known as endometrial cancer (95). Some symptoms of uterine cancer are unusual vaginal bleeding or discharge, difficulty urinating, pain in the pelvic area, and pain during sexual intercourse (95).

Two risk factors for uterine cancer are: being obese and taking hormone replacement therapy in which only estrogen is used (95). Most cases of uterine cancer occur in woman over the age of 50 (96). Women who have had colorectal cancer and have a family history of colorectal cancer have a higher risk of developing uterine cancer (96). The use of the drug Tamoxifen has been linked to an increased risk of uterine cancer (96). Tamoxifen is used to prevent or treat breast cancer (96). Treatment for uterine cancer includes surgery, radiation therapy, hormonal therapy, or a combination of those (97). The Collaborative on Health and the Environment cites a limited amount of evidence linking arsenic to uterine cancer. The Collaborative also cites some evidence of association linking other compounds (not Zeneca COCs) to uterine cancer.

Evaluation of Noncancer Health Concerns at the Zeneca Site

CDPH documented community concerns not related to cancer. These included anemia, asthma, endometriosis, lipoma fatty tumors, lupus, menstrual disorders, ovarian cysts, thyroid nodules, uterine bleeding, uterine fibroid tumors, and unintentional weight loss. Noncancer concerns are evaluated next in alphabetical order.

Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease, is a disease of the nervous system (98). ALS weakens nerve cells that send messages from the brain to muscles that we normally control, such as leg and arm muscles (98). Early symptoms of ALS include mild muscle problems such as difficulty walking, running, writing, or speaking (98). Eventually, people suffering from ALS are unable to move, and when the muscles of the chest stop responding, the person cannot breathe (98). As a result, respiratory failure is the leading cause of death among people with ALS. ALS typically occurs in people between the ages of 40 and 60, and it occurs more frequently among men. The cause of ALS is unknown, although work-related exposure to agricultural chemicals, long-term exposure to lead, smoking, and working in crafts and trades are suspected risk factors (98-101). The Collaborative on Health and the Environment cites limited evidence associating ALS with lead, manganese, mercury, and pesticides. There is limited evidence of other compounds (that are not Zeneca COCs) as being associated with ALS.

Anemia

Anemia is a condition that occurs when the blood does not carry enough oxygen to the rest of the body, usually due to a lack of iron (102). The body needs iron to make hemoglobin, a protein that carries oxygen from the lungs to all parts of the body (102). Low levels of iron could be due to heavy periods, pregnancy, ulcers, colon polyps, colon cancer, inherited illnesses, or a diet that lacks iron, folic acid, or vitamin B12 (102). Some blood disorders can also lead to anemia. Anemia symptoms include weakness, coldness, numbness in the hands and feet, pale skin, dizziness, difficulty concentrating, and irritability (102,103). It is diagnosed with a blood test (102). Treatment of anemia depends on the cause (102,103). The Collaborative on Health and the Environment cites the strength of the evidence linking copper and lead to anemia as strong, and the evidence associating arsenic, cadmium, and mercury to anemia as good. Some other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with anemia.

Asthma

Asthma is a chronic disease in which the airways become swollen and sensitive, reducing the amount of air flow into the lungs and making one react strongly to allergens; as a result, breathing is extremely difficult (104). The resulting symptoms include wheezing, coughing, and tightness of the chest. Most asthma cases begin in childhood (105). African Americans are more likely to be hospitalized for asthma attacks or die from asthma than Caucasians (105). There is no known cure for asthma, but there are ways to control asthma and reduce its severity (104). Asthma management includes avoiding things that create or worsen asthma symptoms, using asthma medication such as allergy medicine and shots, and monitoring asthma to be prepared for times when asthma symptoms may worsen (106). The only association between Zeneca COCs and asthma cited by the Collaborative on Health and the Environment was characterized as

strong and related to nickel; however, the link between asthma and numerous other environmental compounds is well established.

Chickenpox

Chickenpox is an infectious disease that spreads easily between people. Chickenpox is caused by the varicella virus and occurs primarily in children under age 15 (107). Chickenpox symptoms include fever, headache, and an itchy rash with blisters. In young children, the disease tends to be mild; adults and older children with chickenpox can experience more severe complications. People become infected with chickenpox through contact with contagious people. A chickenpox vaccine is recommended between the ages of 12 to 15 months and again between the ages of 4 to 6 to prevent infection. The Collaborative on Health and the Environment does not provide a listing for chickenpox.

Emphysema

Emphysema is a disease that affects the lungs; it damages the air sacs, preventing oxygen from being absorbed (108). As a result, it is difficult to breathe. Other symptoms include a chronic cough and difficulty breathing while exercising (108). Emphysema is most commonly caused by smoking and to a lesser extent by genetic factors (109). The Collaborative on Health and the Environment does not provide a listing for emphysema. Treatment for emphysema can include medications, oxygen, and surgery (108).

Endometriosis

Endometriosis occurs when tissue that lines the uterus grows somewhere else, such as the ovaries, behind the uterus, on the bowels, or on the bladder (110). As a result of endometriosis, a woman can experience infertility, very heavy periods, and pain in the abdomen, lower back and pelvic areas; however, some women have no symptoms (110). The Collaborative on Health and the Environment cites a good body of evidence associating endometriosis with PCBs. The Collaborative on health and the Environment cites limited evidence of association linking other compounds (that are not Zeneca COCs) with bladder cancer. The exact cause of endometriosis is unknown; scientists are investigating factors such as menstrual flow returning to the pelvis, genetics, hormones, immune response, and exposure to manmade chemicals (110,111). Endometriosis is not the same as endometrial cancer (111). Treatment for endometriosis may include pain medication, hormone therapy, or surgical treatment (111).

Goiters

Goiter is a growth of the thyroid gland that can interfere with swallowing or breathing (112). Other symptoms of goiter include swelling of the neck, tightness in the throat, and cough (112). Goiter appears most commonly among women and the elderly (112). One cause of goiters is a shortage of iodine in the diet. Other causes include low or excessive thyroid production, thyroid cancer, pregnancy, and inflammation (112). Treatment for goiter depends on the underlying cause and can include hormone therapy, surgery, radioactive iodine, or in mild cases, observation (112). The Collaborative on Health and the Environment does not provide a listing for goiter.

Lipoma

A lipoma is a benign (noncancerous) fatty tumor found just below the skin (113). More than one lipoma can develop in an area (113). Lipoma tumors may be present for many years (113). Lipoma tumors occur most often among people between 40 and 60 years of age (114). Lipomas

sometimes run in families; some are caused by injuries (114). Unless they are painful or growing rapidly, lipomas usually do not need treatment (114). If treatment is necessary, some options include steroid shots or surgery (114). The Collaborative on Health and the Environment does not provide a listing for lipomas.

Lupus

Lupus is a disease that occurs when the body's immune system attacks its own tissues and organs, causing inflammation in different parts of the body such as the joints, skin, kidneys, blood cells, heart, and lungs (115). There are four types of lupus: discoid lupus erythematosus, drug-induced lupus erythematosus, neonatal lupus, and the most common type, systemic lupus erythematosus (115). Lupus occurs more often among women, although the reason for this is unknown (115).

Lupus cases are different from each other. Because the disease can affect different body systems, signs and symptoms vary. Generally, some lupus symptoms are fatigue, fever, weight fluctuation; pain, stiffness, and swelling of the joints; a face rash that covers the cheeks and the bridge of the nose; skin lesions that result from sun exposure; skin lesions that worsen as a result of sun exposure; mouth sores; white or blue fingers or toes during cold or stressful periods; shortness of breath; chest pain; dry eyes; bruising easily; anxiety; depression; and memory loss (115). Some people may experience symptoms suddenly, while in other people symptoms develop at a slow pace (115). A common experience among people with lupus is an episode in which symptoms worsen and eventually improve or disappear; these episodes are called "flares" (115).

Although the exact causes of lupus are not known, some factors that may increase the risk of developing lupus are being a woman; being between the ages of 15 and 45; being African American or Asian; being exposed to ultraviolet radiation in sunlight; taking certain prescription medications; having recurring infections of Epstein-Barr Virus, which causes fever and sore throat; and being exposed to chemicals in the workplace such as mercury and silica (115). The Collaborative on Health and the Environment does not provide a listing for lupus.

Doctors diagnose lupus by identifying a set of symptoms and criteria and administering a series of urine and blood tests (115). It may take months or years to make a diagnosis (116). Lupus management depends on the symptoms, and can include anti-inflammatory medication and avoiding sun exposure (115). Lupus treatment has improved, and most people who have lupus are able to lead active lives (115).

Menstrual Disorders

Menstrual disorders here refer to abnormal bleeding, painful periods, and unusually short, long, or irregular cycles. These may be symptoms of endometriosis, uterine cancer or other conditions. The Collaborative on Health and the Environment cites a good amount of evidence associating menstrual disorders with lead, mercury, PCBs, and toxaphene; the evidence associating menstrual disorders with antimony and cadmium is limited.

Ovarian Cysts

The ovaries are two almond-shaped organs located in a woman's uterus. An ovarian cyst is a

fluid-filled sac that forms in a woman's ovary (117). Most women have ovarian cysts at some point in their lives. In rare occasions, ovarian cysts are cancerous among women under 50 (117). Ovarian cysts can affect fertility. Most ovarian cysts are not painful (118). If symptoms are present, they may include menstrual irregularities, nausea, vomiting, breast tenderness, fullness in the abdomen, pressure on the rectum or bladder, or pain the pelvic region (118). There are several types of cysts. Depending on the cyst type, symptoms, and woman's age, treatment may include observation, birth control pills, or surgery (118). The Collaborative on Health and the Environment does not provide a listing for ovarian cysts.

Polio

Polio (poliomyelitis) is a contagious disease caused by a virus that affects the nervous system (119). Polio can cause paralysis when the virus destroys nerve cells that feed into muscles, especially in the legs (119). The polio virus is spread through person-to-person contact, when the feces of infected people contacts other people in areas with poor sanitation systems (119). Polio affects primarily children under 3. There is no known cure for polio; however, it can be prevented through multiple immunizations. Polio epidemics existed in the United States until the late 1950s, when effective vaccines were introduced (119). Today, polio is still present in seven countries, including India, Nigeria, and Pakistan (119). The Collaborative on Health and the Environment does not provide a listing for polio.

Pre-cancerous Lesions on Skin

Pre-cancerous lesions on the skin are known as actinic keratosis, and are usually caused by sun exposure (120). Not all actinic keratosis cases develop into skin cancer; most cases (99%) are benign (120). Symptoms include a skin lesion that is rough and dry in texture, initially flat and scaly on the surface; with time, it becomes slightly raised and could become hard and wart-like (120). The patch of skin may be gray, pink, red, or the same color as the skin (120). It usually appears on areas that are exposed to the sun such as the face, scalp, and hands. Treatment can include removal by freezing, burning, or surgery, as well as medicine that prompts the skin to peel (120). Lasers are sometimes used in treatment (120). Exposure to sunlight is the most common cause of actinic keratosis among otherwise healthy Whites (120,121). One study found actinic keratosis among individuals who had been exposed to more than 0.13 mg/l (0.13 ppm) arsenic in drinking water for at least 20 years (122). The Collaborative on Health and the Environment does not provide a listing for actinic keratosis; however it cites a strong body of evidence associating arsenic and zinc with skin ulceration. Numerous other compounds (that are not Zeneca COCs) are cited by the Collaborative on Health and the Environment as being associated with skin ulceration.

Rheumatic Fever

Rheumatic fever is an inflammatory disease that can be caused by infection with *Streptococcus* bacteria (123). *Streptococcus* bacteria are the same bacteria responsible for strep throat and scarlet fever (123). Symptoms of rheumatic fever include fever, arthritis, joint swelling, pain in the abdomen, skin rashes or nodules, nosebleeds, and heart problems (123). Another symptom, known as Sydenham's chorea, is characterized by emotional instability, weak muscles, and rapid, erratic movements in the face, feet, and hands (123). Treatment includes anti-inflammatory medicine and antibiotics (123). Rheumatic fever can be prevented by prompt treatment of strep throat and scarlet fever. Some rheumatic fever outbreaks have occurred in the United States since

the 1980s; however, rheumatic fever is a lot less common today than it was in the 1800s (123). The Collaborative on Health and the Environment does not provide a listing for rheumatic fever.

Thyroid Nodules

The thyroid gland is located at the base of the neck; it makes hormones that regulate growth and how the body uses energy (124). Thyroid nodules develop when thyroid tissue begins to grow within the thyroid gland (124). Most thyroid nodules do not cause symptoms and are detected by doctors during routine medical exams (124). Other thyroid nodules can become large and press on the windpipe or esophagus, which can create discomfort or difficulty when swallowing (124). It is estimated that about 5% of thyroid nodules can be cancerous (124). Risk factors for developing thyroid nodules are heredity, being older, being a woman, having been exposed to therapeutic radiation, having been exposed to radioactive particles released during atomic weapons testing or nuclear power plant incidents, and having chronic inflammation of the thyroid gland (124). Aside from difficulty breathing or swallowing, some symptoms of thyroid nodules include sudden weight loss despite a normal appetite, trouble sleeping, muscle weakness, and nervousness or irritability (124). The Collaborative on Health and the Environment does not provide a listing for thyroid nodules.

Tuberculosis

Tuberculosis is a contagious disease caused by the bacteria *Mycobacterium tuberculosis* (125). The bacteria attack the lungs. Initial symptoms include weight loss, fever, night sweats, and loss of appetite; symptoms then can either stop or worsen, with chest pain, and cough, including coughing up blood (125). Tuberculosis is spread through the air, when people with tuberculosis cough, sneeze, or even sighs, releasing tiny droplets with the bacteria into the air, which can then enter and infect a healthy person's lungs (125). Tuberculosis is treated with antibiotic drugs; treatment is usually lengthy (125). The Collaborative on Health and the Environment does not provide a listing for tuberculosis.

Uterine Bleeding

Women can experience abnormal uterine bleeding as a result of too much estrogen or not enough progesterone, small and large growths in the uterus, cancer of the uterus, infection of the cervix, or thyroid conditions (126). Among women in their 20s and 30s, abnormal uterine bleeding may occur during pregnancy, or as a result of the use of birth control pills or the Norplant birth control device (126). Women in their 40s and early 50s may experience abnormal uterine bleeding during months when they do not ovulate during the years before menopause (126). Thickening of the lining of the uterus may also cause abnormal uterine bleeding among women in their 40s; in this case, it may be a warning sign of uterine cancer (126). Women who have gone through menopause may suffer from uterine bleeding as a result of hormone replacement therapy (126). Treatment of abnormal uterine bleeding depends on the cause, and could include medication or surgery (126). The Collaborative on Health and the Environment does not provide a listing for uterine bleeding.

Uterine Fibroid Tumors

Uterine fibroid tumors are non-cancerous tumors located in the wall of the uterus, either because they develop there or they attach to it (127). It is estimated that up to 40% of women in the

United States over the age of 30 have uterine fibroid tumors (127). Uterine fibroid tumors occur more frequently in African American women than Caucasian women (127).

The cause of these tumors is unknown, although oral contraceptives and pregnancy lower the risk of developing them (127). The size of uterine fibroid tumors varies from microscopic to several pounds (127). Uterine fibroid tumors often occur without symptoms; when symptoms are present, they may include pressure or fullness in the abdomen, pelvic cramping or pain with periods, gas, heavy menstrual bleeding, sudden, severe pain, and a need to urinate more often than usual (127). Diagnosis occurs through a pelvic examination; in cases where diagnosis is difficult, an ultrasound is conducted (127). Treatment of uterine fibroid tumors depends on several factors such as the woman's age, the severity of the symptoms, pregnancy, desire for future pregnancies, overall health status, and the characteristics of the fibroid tumors (127). Treatment options include anti-inflammatory drugs, hormone therapy, and surgical procedures (127). Uterine fibroid tumors are usually not cancerous. In rare instances, uterine fibroid tumors become cancerous; this usually occurs after menopause. The most common warning sign of a potentially cancerous uterine fibroid tumor is rapid growing. No Zeneca COCs are listed as being associated with uterine fibroids by the Collaborative on Health and the Environment. The only compound mentioned by the Collaborative on Health and the Environment as having limited evidence associating it with uterine fibroids is diethylstilbestrol (DES).

Whooping Cough

Whooping cough is a very contagious infection of the respiratory tract (128). It is also known as pertussis. In its early stages, it resembles a common cold, with symptoms such as runny nose or nasal congestion, sneezing, loss of appetite, dry cough, and red, watery eyes. After one or two weeks, symptoms worsen into severe coughing attacks that produce thick phlegm, coughing episodes of up to 15 coughs in a row, and fatigue (128). When coughing is severe, blood vessels in the skin surface can get ruptured, causing tiny red spots in the upper body and bleeding in the whites of the eyes (128). Whooping cough is passed from person-to-person, when a person with the pertussis bacteria coughs or sneezes into the air, and others breathe that air (128). Whooping cough can be prevented through the pertussis vaccine, which is typically given in a series of five shots. Whooping cough outbreaks occur regularly, and the whooping cough vaccine eventually wears off (128). Children 6 months old and younger are at greatest risk because they are not fully immune until they receive their third vaccination (128). Whooping cough is on the rise in the United States, from a low of about 1,000 cases in 1976 to more than 25,000 cases in 2004 (128). The Collaborative on Health and the Environment does not provide a listing for whooping cough.

Other Noncancer Health Concerns

Community members reported other health concerns unrelated to cancer such as breathing difficulties, kidney problems, chronic allergy with excessive mucus in the throat, and a condition in which muscles come off of joints. Because of the indistinct nature of these concerns, CDPH is unable to fully evaluate them. One person formerly exposed to Stauffer chemicals was experiencing rapid, unintentional weight loss, and was in the process of being evaluated by a physician. Unintentional weight loss could be a symptom of autoimmune disease, cancer, depression, diarrhea, drug abuse, infection, smoking or thyroid disorders, among other things (129,130). A medical provider can help determine the cause of the weight loss and appropriate

treatment (129). Other health concerns are more ubiquitous, like headaches. The most common type of headache is a tension headache, which results from tight muscles in the shoulders, neck, scalp, and jaw; headaches are usually related to stress, depression, or anxiety (131).

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 or recreate. A HOD review would not provide information about nearby workers, or Bay Trail users. The health outcome databases mentioned above were not in operation during the time period reflective of historic exposure to Seaport residents. 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. Children were considered in the pathways evaluated in this PHA. In addition, CDPH conducted an exposure investigation specifically to identify exposure to children from contaminants in indoor dust.

Conclusions

CDPH evaluated the completed exposure pathway/activities (past, current, and future) to contaminants from Zeneca, using environmental data collected from the site. The conclusions of this evaluation are presented below.

CDPH concludes that no public health hazard exists from the following:

- Current exposure to contaminants underlying the Zeneca site (Lots 1-3) under its current use.
- Current exposure to indoor air in businesses in the Harborfront Tract from vapor intrusion, as a result of VOC-contaminated groundwater.

- Past, current, and future exposure to metals, pesticides, and PCBs in sediment and surface water in the East Stege Marsh.
- Past exposure to students and staff from site-related contaminants in dust and indoor air in Building 240, used by the Making Waves Education Program from 2002-2006.
- Past exposure to site-related contaminants in dust during remedial activities conducted between 2002 and 2005.

It is possible that, during remedial work conducted between May 2002 and July 2005, nearby workers and Bay Trail users could have experienced mild irritant effects of the respiratory tract from breathing dust on the days when dust levels were elevated.

CDPH was not able to determine the potential health impacts of historic exposure to Seaport residents, nearby workers, or residents of the Panhandle Annex and adjacent neighborhoods. Given the types of manufacturing that occurred at Stauffer, history of emission control and regulations, we recognize that exposures at levels of health concern could have occurred. However, there are no data available to evaluate the level and magnitude of these exposures.

As of this writing, radionuclides associated with the production of superphosphate fertilizer and other Stauffer-related work are being investigated. The Radiologic Health Branch of CDPH is providing technical support to DTSC regarding any radiological issues at Zeneca. This PHA addresses potential exposures to chemical contaminants found at the site. If future investigations indicate a need to evaluate potential exposure to radionuclides, an addendum to this PHA will be provided.

Due to a lack of data, CDPH was not able to determine whether there is a past, current, or future health risk from exposure to elevated levels of naturally-occurring radionuclides that may be present in non-excavated portions of the marsh as a result of Stauffer operations.

CDPH conducted a number of outreach activities to collect and understand the health concerns community members believe are related to contamination at Zeneca. In the PHA, CDPH responds to these concerns by stating whether there is an association between chemical exposure and the health concern expressed. Given the gaps in exposure and toxicological data, we are not able to draw a link between Zeneca-related contaminants and the health concerns expressed, with the exception of irritation of the eyes, nose, and throat, and mild respiratory effects that may have occurred from exposure to airborne dust.

Recommendations

1. CDPH and ATSDR recommend the Zeneca site be cleaned up to levels consistent with residential standards if the land use changes from industrial to residential or recreational. An increased residential population in this area may result in more human activity in tidal/shoreline areas adjacent to site. Thus, areas south of the Bay Trail should be characterized and the risk to potential receptors evaluated. These activities should be carried out under the oversight of DTSC.

2. CDPH and ATSDR recommend a robust air monitoring program and adequate dust suppression measures be implemented during future remedial work at the site and during any development activities where soil is disturbed. These activities should be carried out under the oversight of DTSC.
3. CDPH and ATSDR recommend access to the East Stege Marsh remain restricted until there is a complete understanding of the potential radiological issues at the site, and it can be determined that the non-excavated portions of the East Stege Marsh do not contain site-related radionuclides at levels of health concern.
4. CDPH and ATSDR recommend annual sampling of sediment and unfiltered surface water in the East Stege Marsh until the site is remediated to ensure that the marsh is not being re-contaminated from contaminant migration from other areas.

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 activities that are on-going and the third section lists additional public health actions that are planned for the future.

Actions Completed

- CDPH gathered community concerns through meetings with local business owners, outreach efforts utilizing local media, and ongoing discussions with community members (May 2005-July 2007).
- CDPH and the Contra Costa County Health Services Department released a Provisional Joint Health Statement, providing an evaluation of current exposure from contaminants at the Zeneca and adjacent RFS sites (June 2005; update in August 2007).
- CDPH and ATSDR recommended that the East Stege Marsh be fenced and posted to eliminate exposure to contaminants remaining in the marsh (action completed in December 2005).
- CDPH contacted OEHHA regarding the posting of fish advisories relative to the San Francisco Bay, along the shoreline in the Marina Bay area (December 2007).

Ongoing Actions

- CDPH will continue to provide health outreach and education to the community and nearby business owners as needed.

Actions Planned

- CDPH will disseminate information summarizing the findings of this comprehensive PHA and discuss the results at a public meeting.
- CDPH will provide an addendum to this PHA if future investigations indicate a need to evaluate potential exposure to radionuclides.

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References

1. Levine Fricke. Current conditions summary report Lot 3, Campus Bay, 1200 South 47th Street, Richmond, California. Oakland (CA); 2005 Jul. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
2. Levine Fricke. Results of soil gas sampling and analysis adjacent to Building 240, Campus Bay, Richmond, California. 2005 Aug. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
3. Hodge C. Growth of the world fertilizer industry. In: Hodge C, Popovici N, eds. Pollution Control in Fertilizer Production. New York: Marcel Dekker, Inc., 1994;1-8.
4. Gorecki H. Granular triple superphosphate and single superphosphate. In: Hodge C, Popovici N, eds. Pollution Control in Fertilizer Production. New York: Marcel Dekker, Inc., 1994;269-98.
5. Popovici N. Pollutants, wastes, and by-products of the fertilizer industry. In: Hodge C, Popovici N, eds. Pollution Control in Fertilizer Production. New York: Marcel Dekker, Inc., 1994;9-22.
6. Hodge C. Regulation and pollution control in the fertilizer industry. In: Hodge C, Popovici N, eds. Pollution Control in Fertilizer Production. New York: Marcel Dekker, Inc., 1994;23-24.
7. Levine Fricke. Revised remedial investigations report, Lot 1 and Lot 2, Campus Bay, Richmond California. Oakland (CA); 2007 Jul. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
8. U.S. Census Bureau. Profile of General Demographic Characteristics: 2000, Census Tract 3800, Contra Costa County, California. Cited online at:
http://factfinder.census.gov/servlet/QTGeoSearchByListServlet?ds_name=DEC_2000_SF1_U&lang=en&ts=219241098228.
9. Agency for Toxic Substances and Disease Registry. Minimal risk levels (MRLs) for hazardous substances. Atlanta (GA): U.S. Department of Health and Human Services; 2006 Feb. Available online at <http://www.atsdr.cdc.gov/mrls/>.
10. U.S. Environmental Protection Agency. Integrated risk information system (IRIS). Available online at <http://www.epa.gov/iris/search.htm>. Last accessed 2007 Aug.
11. California Environmental Protection Agency. Use of California human health screening levels (CHHSLs) in evaluation of contaminated properties. Sacramento (CA); 2005 Jan. Available online at www.calepa.ca.gov/Brownfields/documents/2005/CHHSLsGuide.pdf.
12. California Cancer Registry. Cancer in California, 2000: A decade of cancer surveillance. Sacramento (CA); 2000 Jun. Available for public viewing at Sacramento (CA): California Cancer Registry.
13. East Bay Municipal Utilities District. Urban water management plan 2005. Available online at https://portal.ebmud.com/water_&_environment/water_supply/urban_water_management_plan/2005_uwmp/default.htm. Last accessed 2007 Nov 26.
14. City of Richmond. History of Richmond, California. Available online at <http://www.ci.richmond.ca.us/index.asp?NID=112>. Last accessed 2006 Jun.
15. Wilson Moore SA. To place our deeds: The African American community in Richmond, California, 1910-1963. University of California Press. 1999.

16. St. John K. Life in Seaport - Next door to a plant making pesticides. San Francisco Chronicle, 2004 Aug 31.
17. California Environmental Protection Agency, Department of Toxic Substances Control. May 5, 2005 News Release: Members of community advisory group selected. Available online at http://www.dtsc.ca.gov/PressRoom/upload/NEWS_2005_T-31-05.pdf. Last accessed 2006 Jun.
18. Ethel Dotson. Letter to Dr. Henry Falk requesting an investigation into health effects of Stauffer Chemical Company on residents of Seaport War Apartments. Richmond, California. April 24, 2002. Available for public viewing at Richmond (CA): California Department of Public Health.
19. Quivik F. Historic american engineering record, Kaiser's Richmond shipyards, July 2004. Available online at <http://www.rosietheriveter.org/home/shipyard3history.pdf>. Last accessed 2007 Apr.
20. Levine Fricke. Draft final remedial investigation report, Lot 3, Campus Bay, 1200 South 47th Street, Richmond, California. Oakland (CA); 2007 Jan. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
21. U.S. Environmental Protection Agency. Sector Notebooks: Profile of the agricultural chemical, pesticide and fertilizer industry. Washington, DC; 2000. Available online at <http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/index.html>.
22. Florida Institute of Phosphate Research. Phosphate primer. Available online at <http://www1.fipr.state.fl.us/PhosphatePrimer>. Last accessed 2007 Apr.
23. Bunus FT. Phosphoric acid by wet process: Radioactive components in phosphoric acid and their removal. In: Hodge C, Popovici N, eds. Pollution Control in Fertilizer Production. New York: Marcel Dekker, Inc., 1994;23-24.
24. Federal Register. National emission standards for hazardous air pollutants from phosphoric acid manufacturing plants and phosphate fertilizers production plants, Final Rule. Washington, DC: U.S. Environmental Protection Agency; 2002 Jun. Available online at <http://www.epa.gov/fedrgstr/EPA-AIR/2002/June/Day-12/a14757.htm>.
25. Levine Fricke. Human health and ecological risk assessment, East Stege Marsh, Meade Street Operable Unit, Richmond, California. Oakland (CA); 2002 Mar. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
26. Levine Fricke. Second annual monitoring report, East Stege Marsh monitoring plan implementation, Campus Bay, Richmond, California. Oakland (CA); 2007 Aug. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
27. Levine Fricke. Memorandum to Bill Carson from Shane Noreen concerning approved Bay Mud fill, analytical data. Oakland, California. July 19, 2005. Available for public viewing at Richmond (CA): California Department of Public Health.
28. Levine Fricke. Evaluation of year 1 sediment monitoring results from East Stege Marsh in June 2006, Campus Bay, Richmond, California. Oakland (CA); 2006 Nov. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
29. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health. Adult blood lead epidemiology and surveillance (ABLES). Available online at <http://www.cdc.gov/niosh/topics/ABELS/ables.html>. Last accessed 2006 Aug.

30. California Department of Public Health, Childhood Lead Poisoning Branch. Management guidelines for blood lead levels in children and adults. 2006 Nov. Available online at <http://www.dhs.ca.gov/childlead/html/POmatrix.html>.
31. U.S. Environmental Protection Agency. Recommendations of the technical review workgroup for lead: An approach to assessing risks associated with adult exposures to lead in soil. Washington, DC; 2003 Jan. Available online at <http://www.epa.gov/superfund/lead/products/adultpb.pdf>.
32. Agency for Toxic Substances and Disease Registry. Public health assessment guidance manual (update). Atlanta (GA): U.S. Department of Health and Human Services; 2005 Jan. Available online at <http://www.atsdr.cdc.gov/HAC/PHAManual/>.
33. Levine Fricke. Implementation report for upland remediation Subunit 1 and Subunit 2A, Meade Street Operable Unit, Richmond California. Oakland (CA); 2003 Oct. Available for public viewing at Berkeley (CA): Department of Toxic Substances Control.
34. Levine Fricke. Final implementation report for remediation of Habitat Enhancement Area Campus Bay, Richmond, California. Oakland (CA); 2007 Mar. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
35. American Lung Association of California. Particulate matter air pollution. Available online at http://www.californialung.org/spotlight/cleanair03_particulate.html. Last accessed 2006 Nov.
36. California Air Resources Board. Ambient air quality standards for particulate matter. Available online at <http://www.arb.ca.gov/research/aaqs/pm/pm.html>. Last accessed 2007 Nov.
37. California Air Resources Board. Hydrogen sulfide. Available online at <http://www.arb.ca.gov/research/aaqs/caaqs/h2s/h2s.htm>. Last accessed 2008 Jan.
38. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. Draft guidance for evaluating the vapor intrusion to indoor pathway from groundwater and soils (subsurface vapor intrusion guidance). Washington, DC; 2002 Nov. Available online at <http://www.epa.gov/correctiveaction/eis/vapor.htm>.
39. Interstate Technology and Regulatory Council. Vapor intrusion pathway: A practical guideline. Available online at http://www.itrcweb.org/gd_VI.asp. Last accessed 2007 Aug.
40. California Environmental Protection Agency, Department of Toxic Substances Control. Interim final guidance for evaluation and mitigation of subsurface vapor intrusion to indoor air. Sacramento (CA); 2007 Feb (revised). Available online at http://www.dtsc.ca.gov/AssessingRisk/upload/HERD_POL_Eval_Subsurface_Vapor_Int_rusion_interim_final.pdf.
41. Weiss Associates. Memorandum to the California Department of Toxic Substances Control concerning soil gas survey results (Harborfront Tract). Emeryville, California. January 18, 2006. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
42. California Department of Toxic Substances Control. Screening risk evaluation, Harborfront Tract, Richmond. 2008 Jan. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.
43. PES Environmental, Inc. Data sheets concerning indoor air data, Making Waves Building, Richmond, California. Novato, California. 2005 Mar. Available for public viewing at Richmond (CA): California Department of Public Health.

44. Halmes N, Robert S, Tolson J. Reevaluating cancer risk estimates for short-term exposure scenarios. *Toxicological Sciences* 2000(58):32-42.
45. Office of Environmental Health Hazard Assessment. Technical support document for exposure assessment and stochastic analysis. Oakland (CA): California Environmental Protection Agency; 2000 Sep. Available online at http://209.85.173.104/search?q=cache:sPJFoHhrEncJ:www.oehha.org/air/hot_spots/pdf/c_hap6.pdf+Technical+support+document+for+exposure+assessment+and+stochastic+analysis&hl=en&ct=clnk&cd=1&gl=us.
46. Southwest Research Institute. Dust data package: laboratory analysis of metals, pesticides and PCBs in dust and wipe samples, Zeneca, Richmond, CA. 2006 May. Available for public viewing at Richmond (CA): California Department of Public Health.
47. Brennenman R. Richmond Council asks State to change oversight at two toxic sites. *Berkeley Daily Planet*, 2005 Mar 4.
48. Contra Costa Health Services. Letter to Terry Tamminen from Dr. Wendel Brunner concerning the oversight of the Zeneca site. Martinez, California. [Date missing]. Available for public viewing at Richmond (CA): California Department of Public Health.
49. Contra Costa Health Services. Letter to Bill Lindsay from Dr. Wendel Brunner concerning remediation and cleanup of the Zeneca-Campus Bay properties. Martinez, California. [Date missing]. Available for public viewing at Richmond (CA): City of Richmond Manager's Office.
50. Brennenman R. Activists win new oversight at Campus Bay, UC Field Station. *Berkeley Daily Planet*, 2005 May 13.
51. Contra Costa County Health Services Department and California Department of Public Health. Provisional joint health statement: Zeneca/Campus Bay site. Richmond (CA); 2005 Jun 22. Available for public viewing at Richmond (CA): California Department of Public Health.
52. Contra Costa County Health Services Department and California Department of Public Health. Provisional joint health statement: Zeneca/Campus Bay site. Richmond (CA); 2007 Aug. Available online at <http://ehib.org/cma/projects/FullJS.pdf>.
53. Rosen Lum R. Happy birthday, Richmond. *Contra Costa Times*, 2005 Aug 7.
54. Author unknown. Former residents of Richmond Calif toxic waste site have health concerns. *Contra Costa Times*, 2002 Jul 7.
55. International Finance Corporation. Environmental, health and safety guidelines for phosphate fertilizer manufacturing, 2007 Apr. Available online at: [http://www.ifc.org/ifcext/enviro.nsf/AttachmentsByTitle/gui_EHSGuidelines2007_PhosphateFertilizer/\\$FILE/Final++Phosphate+Fertilizer+Plants.pdf](http://www.ifc.org/ifcext/enviro.nsf/AttachmentsByTitle/gui_EHSGuidelines2007_PhosphateFertilizer/$FILE/Final++Phosphate+Fertilizer+Plants.pdf).
56. National Library of Medicine Hazardous Substances Database. Thiophenol. Available online at <http://toxnet.nlm.nih.gov/cgi-bin/sis/search>. Last accessed 2007 Oct.
57. Lindberg Z. Effect of superphosphate production discharges on children's health. *Gig. I Sanit* 1960;5:89-96.
58. American Cancer Association. Estimated U.S. cancer deaths. Available online at http://www.cancer.org/downloads/STT/Cancer_Statistics_2006_Presentation.ppt#313. Last accessed 2007 Oct 31.
59. National Cancer Institute. Center to reduce cancer health disparities. Available online at <http://crchd.nci.nih.gov/>. Last accessed 2006 Jun.

60. National Cancer Institute. What you need to know aboutTM - Cancer: An overview. Available online at <http://www.cancer.gov/cancertopics/wyntk/overview/page4>. Last accessed 2007 Nov 19.
61. American Cancer Society. Detailed Guide: Bladder cancer - What are the risk factors for bladder cancer? Available online at http://www.cancer.org/docroot/CRI/content/CRI_2_4_2X_What_are_the_risk_factors_for_bladder_cancer_44.asp?sitearea=. Last accessed 2007 Oct 3.
62. The Collaborative on Health and the Environment. CHE toxicant and disease database. Available online at <http://database.healthandenvironment.org/index.cfm>. Last accessed 2007 Oct 3.
63. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Breast cancer. Available online at <http://nlm.nih.gov/medlineplus/breastcancer.html>. Last accessed 2007 Nov 26.
64. National Heart Lung and Blood Institute, National Institutes of Health. Bone cancer: Questions and answers. Available online at <http://www.cancer.gov/cancertopics/factsheet/Sites-Types/bone>. Last accessed 2007 Oct 31.
65. Merleti F, Richiardi L, Bertoni F, Ahrens W, Buemi A, Costa-Santos C, Eriksson M, Guenel P, Kaerlev L. Occupational factors and risk of adult bone sarcomas: A multicentric case-control study in Europe. *Int J Cancer* 2006;118:721-727.
66. National Cancer Institute. What you need to know aboutTM - Kidney cancer: Who's at risk? Available online at <http://www.cancer.gov/cancertopics/wyntk/kidney/page4>. Last accessed 2007 Oct 3.
67. National Cancer Institute. What you need to know aboutTM - Kidney cancer: Treatment? Available online at <http://www.cancer.gov/cancertopics/wyntk/kidney/page8>. Last accessed 2007 Oct 3.
68. Chiu HF, Chuang HY, Ho SC, Wu TN, Yang CY. Reduction in kidney cancer mortality following installation of a tap water supply system in an arsenic-endemic area of Taiwan. *Arch Environ Health* 2004 Sep;59(9):484-488.
69. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Liver cancer. Available online at <http://www.nlm.nih.gov/medlineplus/livercancer.html>. Last accessed 2007 Oct 3.
70. American Liver Foundation. Liver cancer. Available online at <http://www.liverfoundation.org/education/info/livercancer/>. Last accessed 2007 Oct 31.
71. Zeller JL, Lynn C, Glass RM. Lung cancer. *JAMA* 2007;297(9):1022.
72. H Checkoway NH, PA Demers,. An updated mortality follow-up study of Florida phosphate industry workers. *American Journal of Industrial Medicine* 1998 6 Dec;30(4):452-460.
73. American Cancer Society. Detailed guide: Multiple myeloma - What is multiple myeloma? Available online at http://www.cancer.org/docroot/cric/content/cric_2_4_1x_what_is_multiple_myeloma_30.asp. Last accessed 2007 Oct 4.
74. American Cancer Society. Detailed guide: Multiple myeloma - What are the risk factors for multiple myeloma? Available online at http://www.cancer.org/docroot/CRI/content/CRI_2_4_2X_What_are_the_risk_factors_for_multiple_myeloma_30.asp?mav=cric. Last accessed 2007 Oct 4.

75. Benjamin M, Reddy S, Brawley OW. Myeloma and race: A review of the literature. *Cancer Metastasis Rev* 2003 Mar;22(1):87-93.
76. Teens Health, Nemours Foundation. Spleen and lymphatic system. Available online at http://kidshealth.org/teen/your_body/body_basics/spleen.html. Last accessed 2007 Oct 4.
77. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Lymphoma. Available online at <http://www.nlm.nih.gov/medlineplus/lymphoma.html>. Last accessed 2007 Nov 26.
78. National Cancer Institute. What you need to know aboutTM - Non-Hodgkin's Lymphoma: Risk factors. Available online at <http://www.cancer.gov/cancertopics/wyntk/non-hodgkins-lymphoma/page4>. Last accessed 2007 Nov 26.
79. National Cancer Institute. What you need to know aboutTM - Non-Hodgkin's Lymphoma: Risk factors. Available online at <http://www.cancer.gov/cancertopics/wyntk/non-hodgkins-lymphoma>. Last accessed 2007 Nov 26.
80. American Cancer Society. Detailed guide: Pancreatic cancer - What is cancer of the pancreas? Available online at http://www.cancer.org/docroot/CRI/content/CRI_2_4_1X_What_is_pancreatic_cancer_3_4.asp?rnav=cri. Last accessed 2007 Oct 4.
81. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Pancreatic cancer. Available online at <http://www.nlm.nih.gov/medlineplus/pancreaticcancer.html>. Last accessed 2007 Oct 4.
82. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Prostate cancer. Available online at <http://www.nlm.nih.gov/medlineplus/prostatecancer.html>. Last accessed 2007 Nov 1.
83. National Cancer Institute. What you need to know aboutTM - Prostate cancer: Risk factors. Available online at <http://www.cancer.gov/cancertopics/wyntk/prostate/page4>. Last accessed 2007 Nov 1.
84. Walschaerts M, Muller A, Auger J, Bujan L, Guerin JF, Le Lannou D, Clavert A, Spira A, Jouannet P, Thonneau P. Environmental, occupational, and familial risks for testicular cancer: A hospital-based case-control study. *Int J Androl* 2007 Aug;30(4):222-229.
85. National Cancer Institute. What you need to know aboutTM - Cancer of the colon and rectum: The colon and the rectum. Available online at <http://www.cancer.gov/cancertopics/wyntk/colon-and-rectal/page2>. Last accessed 2007 Nov 1.
86. National Cancer Institute. What you need to know aboutTM - Cancer of the colon and rectum. Available online at <http://www.cancer.gov/cancertopics/wyntk/colon-and-rectal>. Last accessed 2007 Nov 1.
87. National Cancer Institute. What you need to know aboutTM - Stomach Cancer. Available online at <http://www.cancer.gov/cancertopics/wyntk/stomach/page1>. Last accessed 2008 16 Jan.
88. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Medical encyclopedia: Cancer of the throat or larynx. Available online at <http://www.nlm.nih.gov/medlineplus/ency/article/001042.htm>. Last accessed 2007 Nov 19.
89. National Cancer Institute. Head and neck cancer: Questions and answers. Available online at: <http://www.cancer.gov/cancertopics/factsheet/sites-types/head-and-neck>. Last accessed 2007 Nov 19.

90. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Thyroid cancer - papillary carcinoma. Available online at: <http://www.nlm.nih.gov/medlineplus/ency/article/000331.htm>. Last accessed 2007 Nov 19.
91. National Cancer Institute. What you need to know aboutTM - Thyroid Cancer: Risk factors. Available online at <http://www.cancer.gov/cancertopics/wyntk/thyroid/page5>. Last accessed 2007 Nov 19.
92. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Skin Cancer. Available online at <http://www.nlm.nih.gov/medlineplus/skincancer.html>. Last accessed 2007 Nov 19.
93. National Cancer Institute. Skin Cancer. Available online at <http://www.cancer.gov/cancertopics/types/skin>. Last accessed 2007 19 Nov.
94. National Cancer Institute. What you need to know aboutTM - Skin cancer: Risk factors. Available online at <http://www.cancer.gov/cancertopics/wyntk/skin/page4>. Last accessed 2007 Nov 19.
95. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Uterine cancer. Available online at <http://nlm.nih.gov/medlineplus/uterinecancer.html>. Last accessed 2007 Nov 19.
96. National Cancer Institute. What you need to know aboutTM - Cancer of the uterus. Available online at <http://www.cancer.gov/cancertopics/wyntk/uterus/page4>. Last accessed 2007 Nov 19.
97. National Cancer Institute. What you need to know aboutTM - Cancer of the uterus: methods of treatment. Available at <http://www.cancer.gov/cancertopics/wyntk/uterus/page11>. Last accessed 2007 Nov 19.
98. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Amyotrophic lateral sclerosis. Available online at <http://www.nlm.nih.gov/medlineplus/amyotrophiclateralsclerosis.html>. Accessed 2008 Jan 7.
99. Kamel et al. Lead exposure as a risk factor for amyotrophic lateral sclerosis. *The Journal of Neurodegenerative Disease*;2(3-4):195-201.
100. Sutedja N, Veldink J, Fischer K, Kromhout H, Wokke J. Lifetime occupation, education, smoking and risk of ALS. *Neurology* 2007 69(15):1508-1514.
101. McGuire V, Longstreth W, Nelson L, Koepsell T, Checkoway H, Morgan M, VanBelle G. Occupational exposures and amyotrophic lateral sclerosis: A population-based case-control study. *American Journal of Epidemiology*. 1997;145 (12):1076-88.
102. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Anemia. Available online at <http://www.nlm.nih.gov/medlineplus/anemia.html>. Last accessed 2007 Nov 27.
103. Mayo Clinic. Anemia. Available online at <http://www.mayoclinic.com/print/anemia/DS00321/DSECTION=all&METHOD=print>. Last accessed 2007 Nov 27.
104. National Heart Lung and Blood Institute, National Institutes of Health. What is asthma? Available online at http://www.nhlbi.nih.gov/health/dci/Diseases/Asthma/Asthma_WhatIs.html. Last accessed 2007 Nov 27.

105. National Heart Lung and Blood Institute, National Institutes of Health. Who is at risk for asthma? Available online at http://www.nhlbi.nih.gov/health/dci/Diseases/Asthma/Asthma_WhoIsAtRisk.html. Last accessed 2007 Nov 27.
106. National Heart Lung and Blood Institute, National Institutes of Health. How is asthma treated? Available online at http://www.nhlbi.nih.gov/health/dci/Diseases/Asthma/Asthma_Treatments.html. Last accessed 2007 Nov 27.
107. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Chickenpox. Available online at <http://www.nlm.nih.gov/medlineplus/chickenpox.html>. Last accessed 2008 Jan 16.
108. Medline Plus. Emphysema. Available online at <http://www.nlm.nih.gov/medlineplus/emphysema.html>. Last accessed 2008 Jan 28.
109. American Lung Association. Emphysema. Available online at <http://www.lungusa.org/site/apps/s/content.asp?c=dvLUK9O0E&b=34706&ct=67284#causes>. Last accessed 2008 Jan 28.
110. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Endometriosis. Available online at <http://www.nlm.nih.gov/medlineplus/endometriosis.html>. Last accessed 2007 Nov 27.
111. National Institute of Child Health and Human Development, National Institutes of Health. Endometriosis: Here's what we do know about endometriosis. Available online at <http://www.nichd.nih.gov/publications/pubs/endometriosis/sub2.cfm>. Last accessed 2007 Nov 27.
112. Mayo Clinic. Goiter. Available online at <http://www.mayoclinic.com/health/goiter/DS00217/DSECTION=1>. Last accessed 2008 Jan 29.
113. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Skin lumps. Available online at <http://www.nlm.nih.gov/medlineplus/ency/imagepages/9896.htm>. Last accessed 2007 Nov 29.
114. American Academy of Family Physicians. Lipomas. Available online at <http://familydoctor.org/online/famdocen/home/articles/763.html>. Last accessed 2007 Nov 29.
115. Mayo Clinic. Lupus. Available online at <http://www.mayoclinic.com/print/lupus/DS00115/DSECTION=all&METHOD=print>. Last accessed 2007 Nov 29.
116. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Lupus. Available online at <http://www.nlm.nih.gov/medlineplus/lupus.html>. Last accessed 2007 Nov 29.
117. Medline Plus. Ovarian Cysts. Available online at <http://www.nlm.nih.gov/medlineplus/ovariancysts.html>. Last accessed 2008 Jan 29.
118. Mayo Clinic. Ovarian Cysts. Available online at <http://www.mayoclinic.com/print/ovarian-cysts/DS00129/DSECTION=all&METHOD=print>. Last accessed 2008 Jan 29.

119. World Health Organization, Global Polio Eradication Initiative. The disease and the virus. Available online at <http://www.polioeradication.org/disease.asp>. Last accessed 2008 Jan 29.
120. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Medical encyclopedia: Actinic keratosis. Available online at <http://www.nlm.nih.gov/medlineplus/ency/article/000827.htm>. Last accessed 2007 Nov 1.
121. Jeffes EW, Tang EH. Actinic keratosis: Current treatment options. *Am J Clin Dermatol* 2000 May;1(3):167-179.
122. Lerda D. Sister-chromatid exchange (SCE) among individuals chronically exposed to arsenic in drinking water. *Mutat Res* 1994 Apr;312(2):111-120.
123. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Rheumatic fever. Available online at <http://www.nlm.nih.gov/medlineplus/ency/article003940.htm>. Last accessed 2008 Jan 29.
124. Mayo Clinic. Thyroid nodules. Available online at <http://www.mayoclinic.com/print/thyroid-nodules/DS00491/DSECTION=all&METHOD=print>. Last accessed 2007 Nov 28.
125. National Institute of Allergy and Infectious Diseases, National Institutes of Health. Tuberculosis. Available online at <http://www3.niaid.nih.gov/topics/tuberculosis/WhatIsTB/>. Last accessed 2008 Jan 29.
126. American Academy of Family Physicians. Abnormal uterine bleeding. Available online at <http://familydoctor.org/online/famdocen/home/women/reproductive/menstrual/470.printreview.html>. Last accessed 2007 Nov 28.
127. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Uterine fibroids. Available online at <http://www.nlm.nih.gov/medlineplus/ency/article/000914.htm>. Last accessed 2007 Dec 10.
128. Mayo Clinic. Whooping cough. Available online at <http://www.mayoclinic.com/health/whooping-cough/DS00445>. Last accessed 2008 Jan 29.
129. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Weight loss - unintentional. Available online at <http://www.nlm.nih.gov/medlineplus/ency/article/003107.htm>. Last accessed 2007 Dec 10.
130. American Academy of Family Physicians. Hyperthyroidism: What it is and how to treat it. Available online at <http://familydoctor.org/online/famdocen/home/common/hormone/869.printerview.html>. Last accessed 2007 Dec 10.
131. Medline Plus, U.S. National Library of Medicine, National Institutes of Health. Headache. Available online at <http://www.nlm.nih.gov/medlineplus/headache.html>. Last accessed 2007 Nov 27.
132. Levine Fricke. Draft final revised removal action work plan remaining portions of East Stege Marsh to be remediated, Campus Bay, Richmond, California. Oakland (CA); 2005 Oct. Available for public viewing at Berkeley (CA): California Department of Toxic Substances Control.

133. U.S. Environmental Protection Agency, Office of Superfund Remediation and Technology Innovation. Risk assessment guidance for superfund (part E, supplemental guidance for dermal risk assessment). Washington, DC; 2004 Jul. Available online at <http://www.epa.gov/oswer/riskassessment/rage/index.htm>.
134. U.S. Environmental Protection Agency, Office of Research and Development. Exposure factors handbook. Washington, DC; 1997. Available online at <http://www.epa.gov/nceawww1/pdfs/efh/front.pdf>.
135. Oakridge National Laboratory. The risk assessment information center: Chemical-specific toxicity values. Available online at http://rais.ornl.gov/tox/tox_values.shtml. Last accessed 2007 Apr.
136. U.S. Environmental Protection Agency. Email memorandum to Rose Marie Caraway and Gerald Hiatt concerning August 2003 Pemaco data. San Francisco, California. November 17, 2003. Available for public viewing at Richmond (CA): California Department of Public Health.
137. California Department of Public Health. Exposure investigation protocol for dust sampling in Building 240, Zeneca/Campus Bay, Richmond, CA. 2006 Apr. Available online at: <http://www.ehib.org/cma/projects/ZenecaEI.pdf>.
138. Agency for Toxic Substances and Disease Registry. Toxicological profile for arsenic. Atlanta (GA): U.S. Department of Health and Human Services; 2007 Aug. Available online at <http://www.atsdr.cdc.gov/toxprofiles/tp2.html>.
139. U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS). Available online at: <http://www.epa.gov/iris/search.htm>. Last accessed 2007 Oct.
140. Agency for Toxic Substances and Disease Registry. Toxicological profile for antimony. Atlanta: U.S. Department of Health and Human Services; 1992 Dec. Available online at <http://www.atsdr.cdc.gov/toxprofiles/tp23.html>.
141. Office of Environmental Health Hazard, California Environmental Protection Agency. Toxicity criteria database. Available online at: <http://www.oehha.ca.gov/risk/ChemicalDB/index.asp>. Last accessed 2007 Oct.
142. Agency for Toxic Substances and Disease Registry. Toxicological profile for cadmium. Atlanta: US Department of Health and Human Services; 1999 Jul. Available online at <http://www.atsdr.cdc.gov/toxprofiles/tp5.html>.
143. Agency for Toxic Substances and Disease Registry. Toxicological profile for copper. Atlanta (GA): U.S. Department of Health and Human Services; 2004 Sep. Available online at <http://www.atsdr.cdc.gov/toxprofiles/tp132.html>.
144. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Acute and chronic reference exposure levels (RELS). Available online at <http://www.oehha.ca.gov/air.html>. Last accessed 2007 Dec.
145. Agency for Toxic Substances and Disease Registry. Toxicological profile for hydrogen sulfide. Atlanta: US Department of Health and Human Services; 2006 Jul. Available online at: <http://www.atsdr.cdc.gov/toxprofiles/tp114.html>.
146. Agency for Toxic Substances and Disease Registry. Toxicological profile for lead. Atlanta: U.S. Department of Health and Human Services; 2007 Aug. Available online at <http://www.atsdr.cdc.gov/toxprofiles/tp13.html>.
147. Agency for Toxic Substances and Disease Registry. Toxicological profile for mercury. Atlanta: U.S. Department of Health and Human Services; 1999 Mar. Available online at <http://www.atsdr.cdc.gov/toxprofiles/tp46.html>.

148. Agency for Toxic Substances and Disease Registry. Toxicological profile for polychlorinated biphenyls. Atlanta: U.S. Department of Health and Human Services; 2000 Nov. Available online at <http://www.atsdr.cdc.gov/toxprofiles/tp17.html>.
149. Agency for Toxic Substances and Disease Registry. Toxicological profile for toxaphene. Atlanta: U.S. Department of Health and Human Services; 1996 Aug. Available online at <http://www.atsdr.cdc.gov/toxprofiles/tp94.html>.
150. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. Guidelines for carcinogen risk assessment. Washington, DC; 2005 Mar. Publication No. 630/P-03/001F. Available online at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=116283>.

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 United States. 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, 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.

Benchmark Dose

A dose or concentration that produces a predetermined change in response rate of an adverse effect (called the benchmark response or BMR) compared to background.

California Human Health Screening Levels (CHHSLs)

Cal/EPA 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

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. The U.S. Environmental Protection Agency and the California Environmental Protection Agency have developed cancer slope factors and inhalation unit 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. 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. The Agency for Toxic Substances and Disease Registry 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’s Cancer Risk Evaluation Guide for 1 in 1,000,000 increased cancer risk)

CREGs are screening values for air, soil and water, developed by ATSDR. To derive water and soil CREGs, ATSDR uses CSFs developed by the U.S. Environmental Protection Agency 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 liters per day of water and weigh 70 kilograms. For soil ingestion, ATSDR assumes a soil ingestion rate of 100 milligram per day, for a lifetime (70 years) of exposure.

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 cancer slope

factor When developing CREGs, 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.

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 the “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’s Environmental Media Evaluation Guide)

EMEGs are screening values based on noncancer health endpoints, developed by ATSDR. EMEGS have been developed for air, soil and water. 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 released during showering, are not considered when deriving EMEGs.

To derive 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 for a 10-kilogram 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.

For 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, with 100 milligrams per day representing the best estimate of the average intake rate. ATSDR calculates an EMEG for a child using a daily soil ingestion rate of 200 milligrams per 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 Frequency

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

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.

Hazard Index

The sum of the Hazard Quotients (see below) for all contaminants of concern identified, to 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 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 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. "Uncertainty" factors are used to account for the uncertainty in our knowledge about their danger. The greater the uncertainty, the greater the "uncertainty" 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; RELs, MRLs 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/m³).

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. Some effects may be produced at this dose, but they are not considered adverse, nor precursors to adverse effects.

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 (U.S. Environmental Protection Agency's 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

Public Health Hazard Categories (ATSDR)

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 by ATSDR by using one of the following public health hazard categories:

Urgent Public Health Hazard

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. 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

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.

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 available at <http://www.atsdr.cdc.gov/HAC/PHAManual/>.

Qualitative Description of Estimated Increased Cancer Risks

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)

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

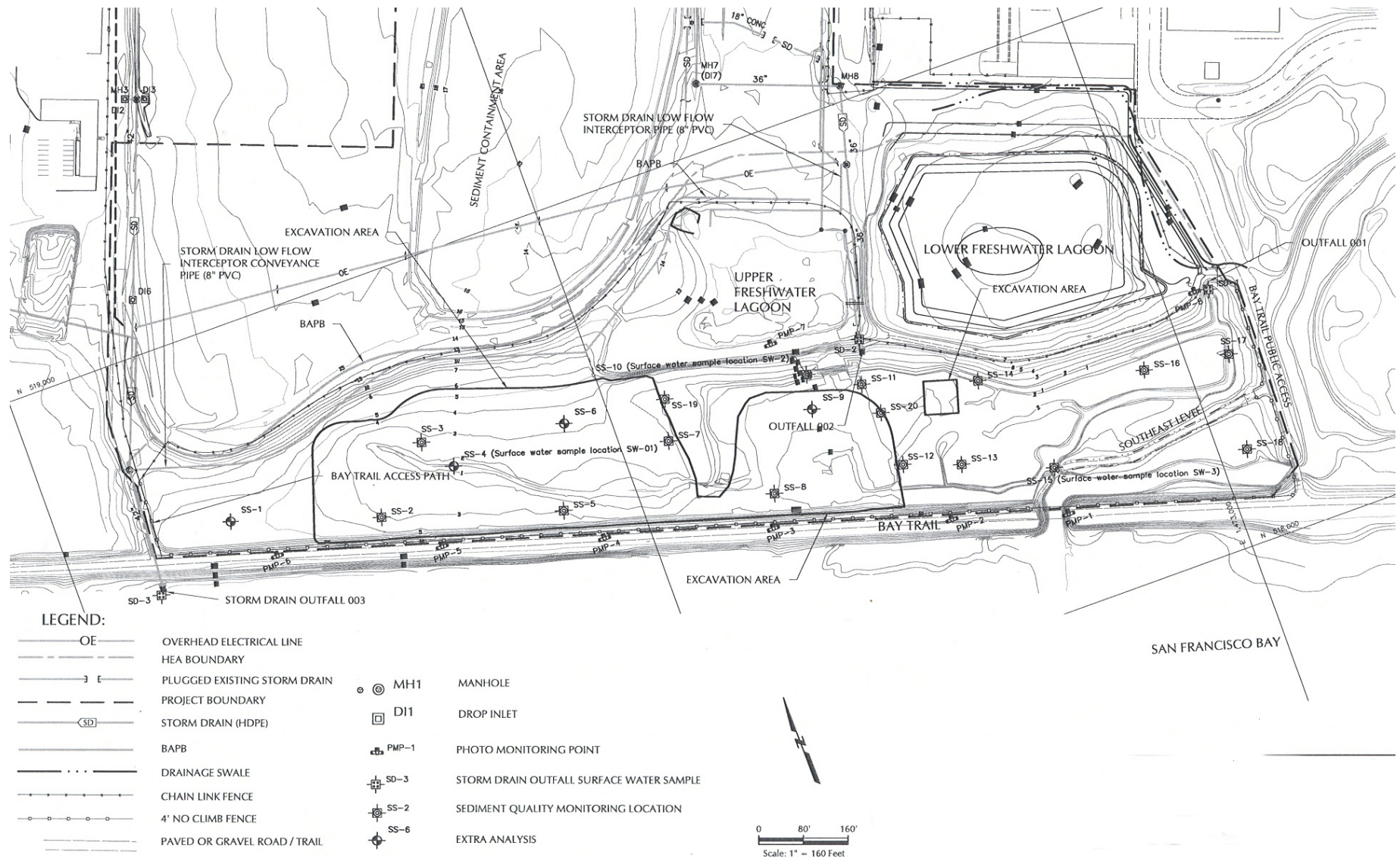
Figure B-1. Site Location Map, Zeneca/Campus Bay, Richmond California



HEA: Habitat Enhancement Area
Source (1)

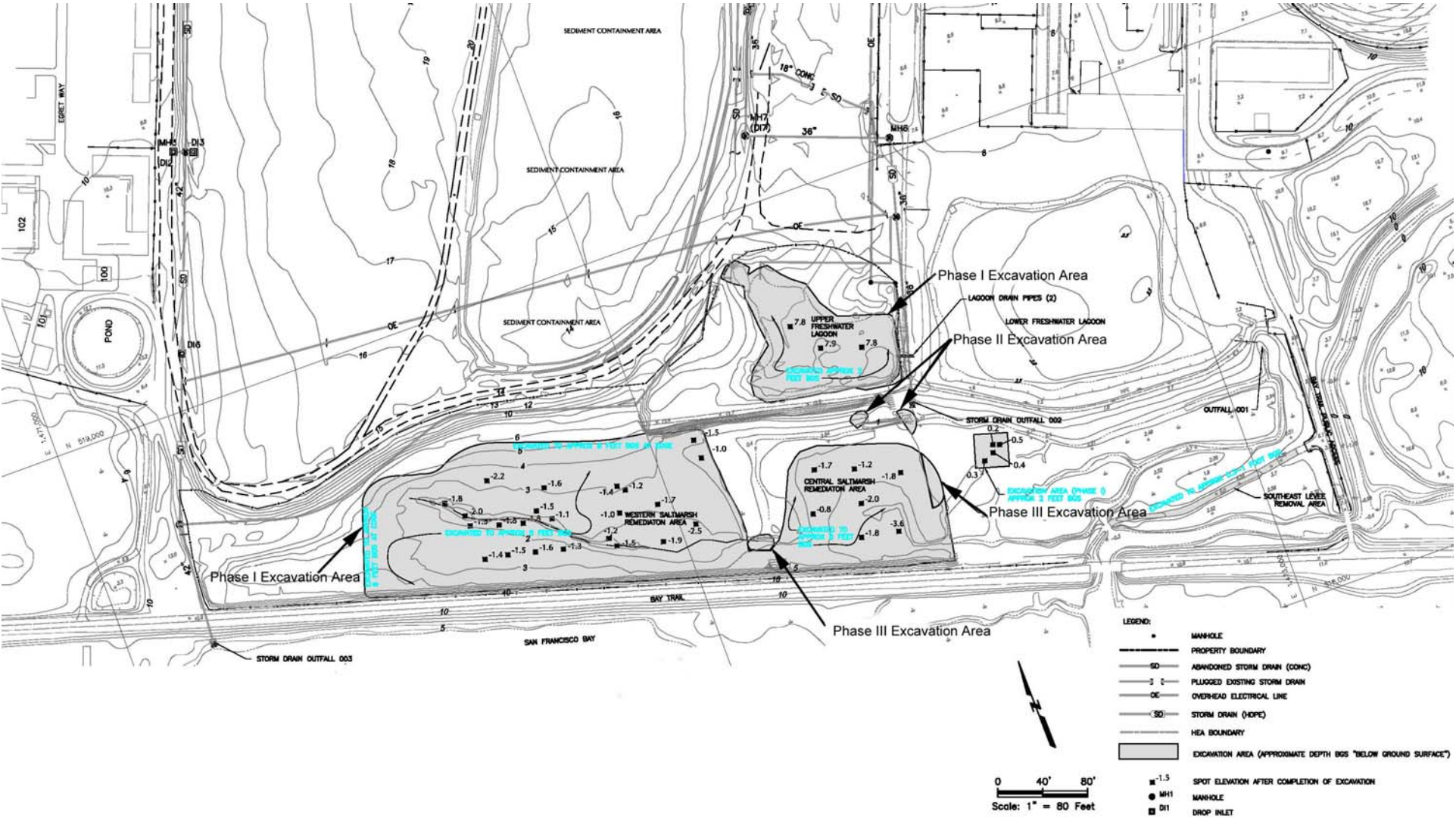
74

Figure B-3. Location of Sediment Samples Collected in the East Stege Marsh in 2006, After Remediation, Zeneca/Campus Bay, Richmond, California



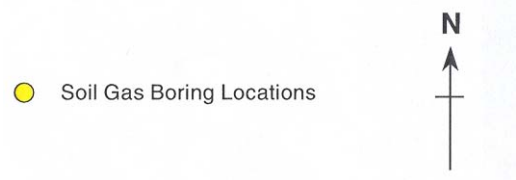
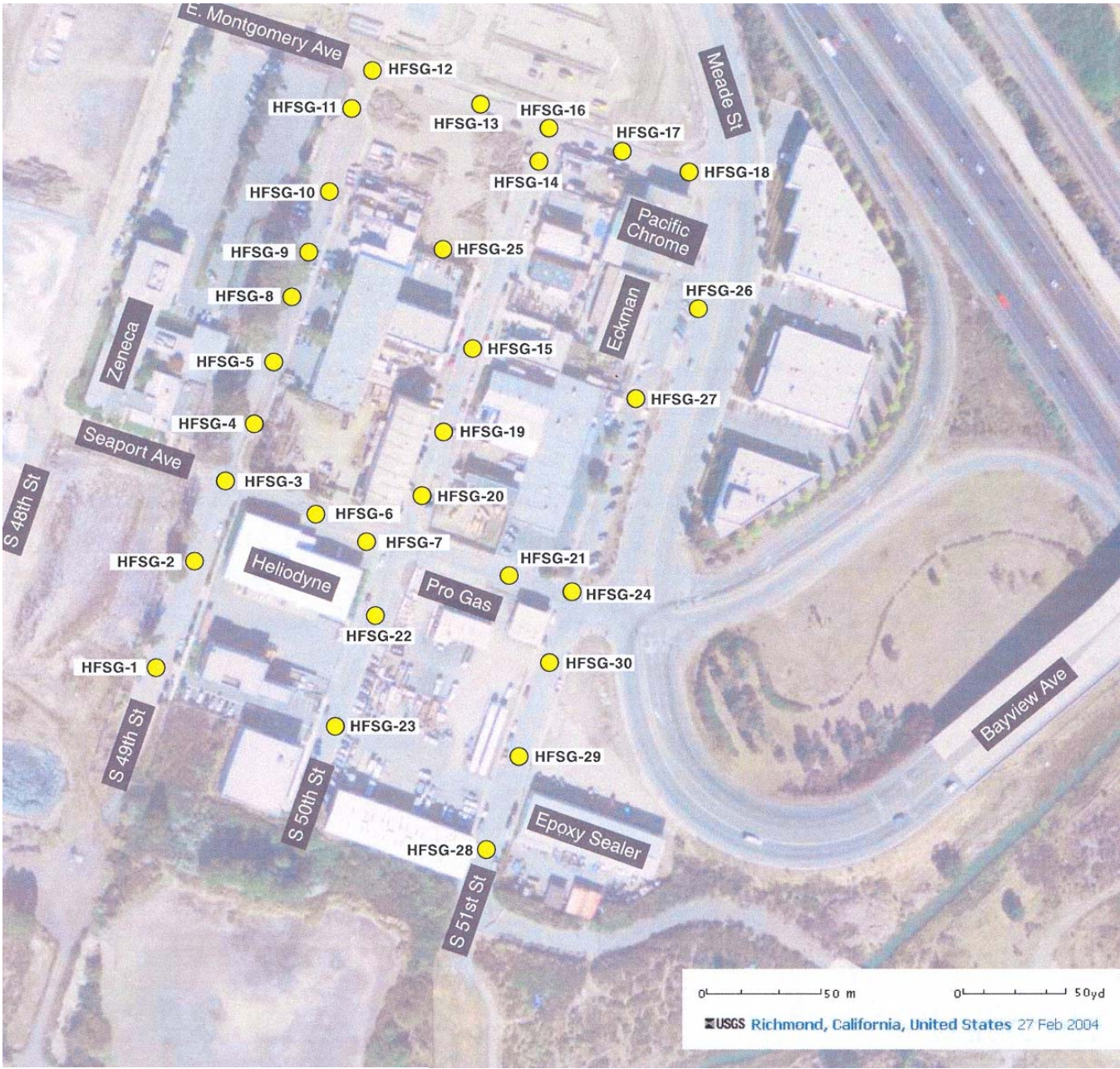
HEA: Habitat Enhancement Area; BAPB: Biologically Active Permeable Barrier
Source (28)

Figure B-4. Location of Phase 1-Phase III Excavation Areas, Zeneca/Campus Bay, Richmond, California



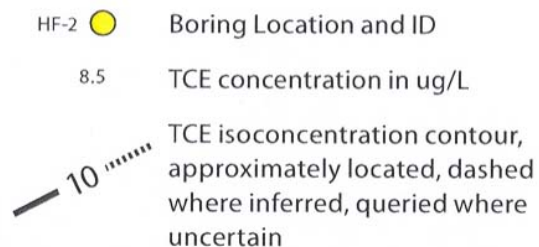
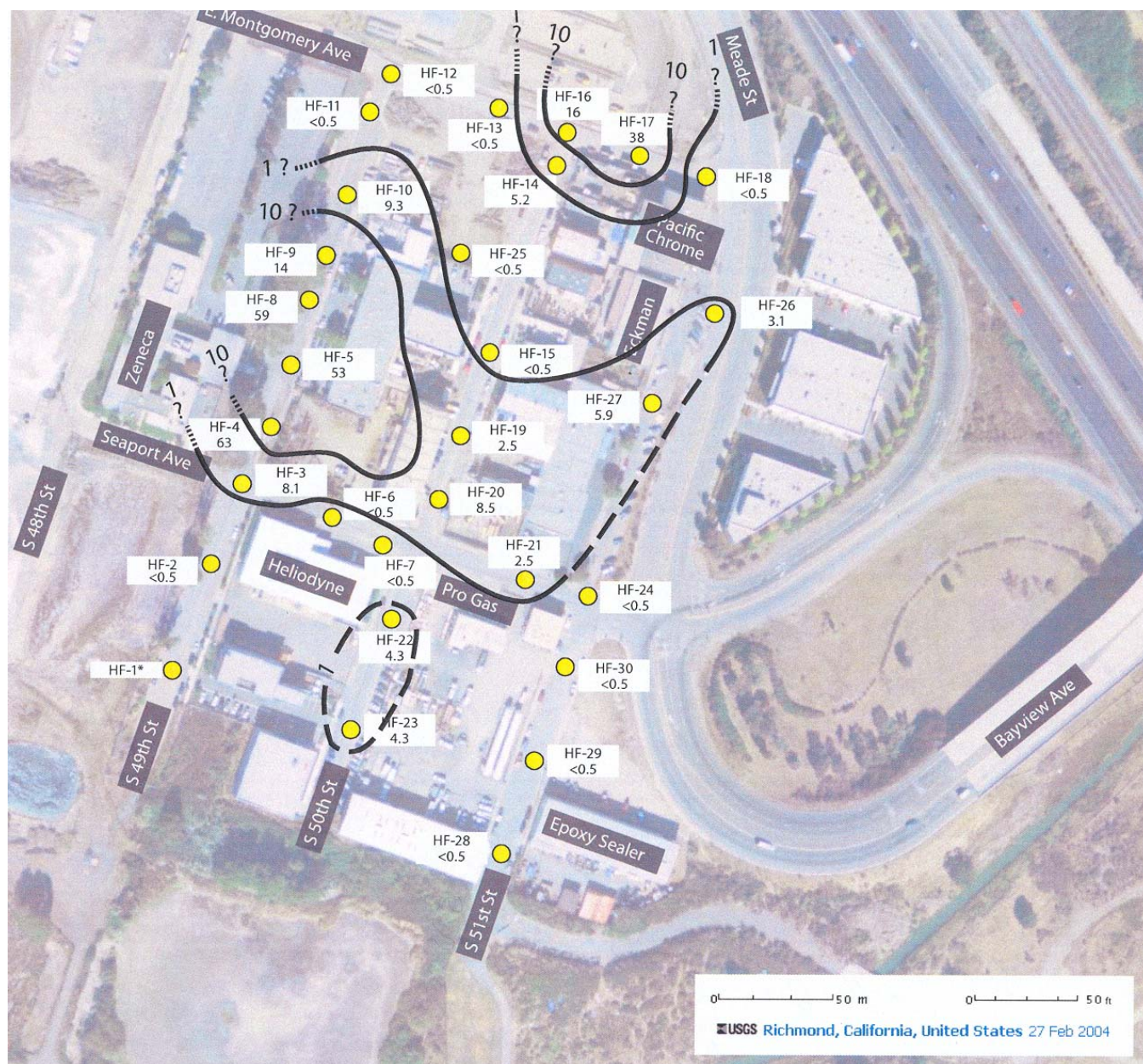
Source (34)

Figure B-5. Location of Soil Gas Samples Collected in the Harborfront Business Tract, Zeneca/Campus Bay, Richmond, California



Source (41)

Figure B-6. Trichloroethylene Concentration Contours in Shallow Groundwater in the Harborfront Business Tract, Zeneca/Campus, Richmond, California

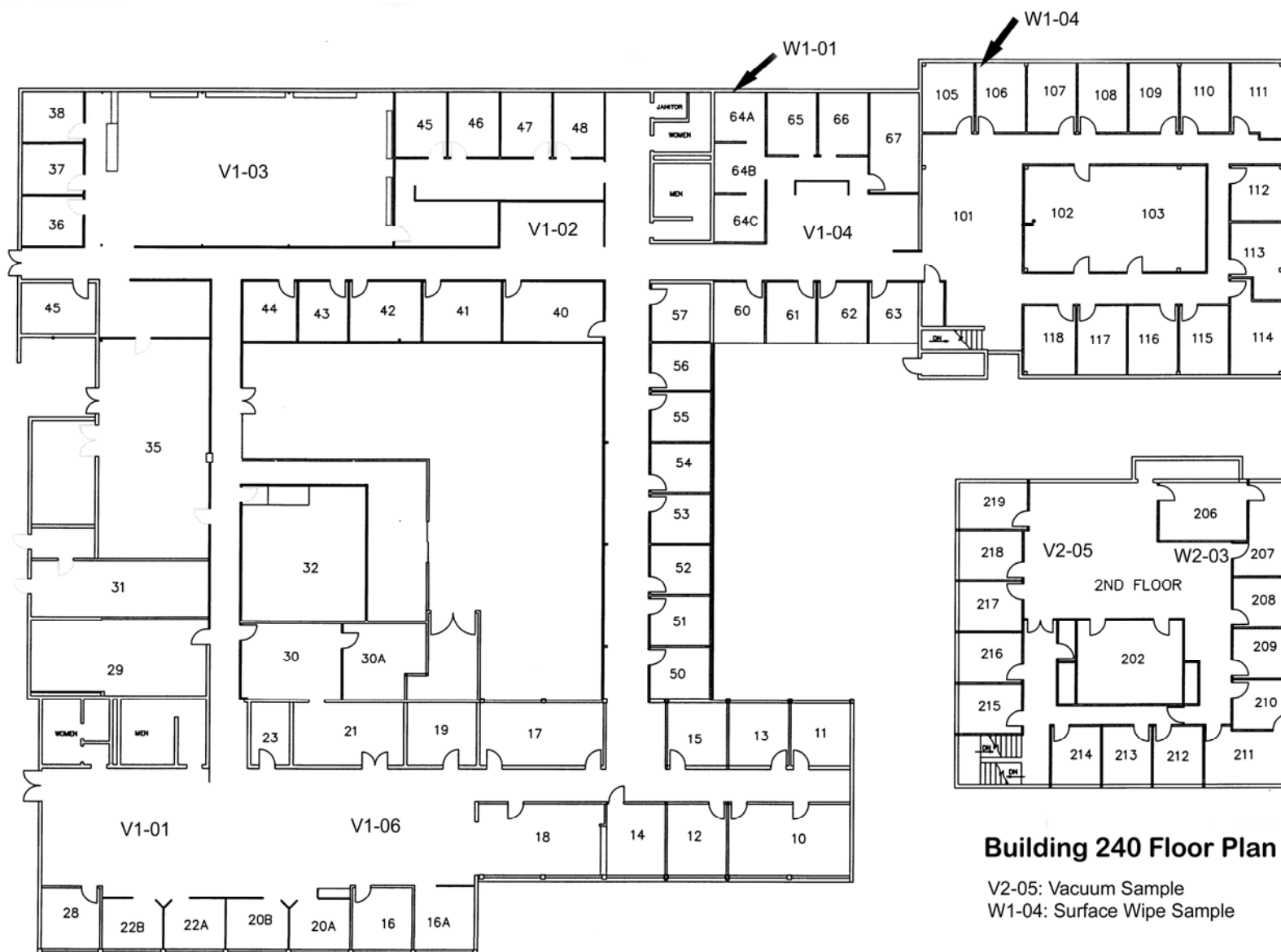


ug/L: microgram per liter
MCL: maximum concentration level
Source (41)

Note:
Bold indicates concentrations above the 5 ug/L MCL

*HF-1 not sampled due to flooding

Figure B-7. Approximate Location of Vacuum Dust and Surface Wipe Samples Collected in Building 240, Zeneca/Campus Bay, Richmond, California



Appendix C. Tables

Table C-1. Completed Exposure Pathways (Scenarios), Zeneca/Campus Bay, Richmond, California

Pathway Name	Contaminants of Concern	Pathway Elements					
		Source	Environmental Media	Point of Exposure	Route of Exposure	Potentially Exposed Population	Time
Seaport Residents	Metals, VOCs, particulates (dust) pesticides, inorganic acids, fluorides, radionuclides	Zeneca (formerly Stauffer)	Air, soil, surface water, food chain	Outdoor air, soil, surface water	Inhalation, ingestion, dermal contact	Adults and children residents	Past (1944-1956)
Panhandle Annex and nearby workers	Metals, VOCs, particulates (dust), inorganic acids, fluorides, radionuclides	Zeneca (formerly Stauffer)	Air	Outdoor air	Inhalation	Adults and children residents, nearby workers	Past (1950s-1997)
East Stege Marsh sediment and surface water	Metals, PCBs, pesticides	Zeneca	Sediment, water	East Stege Marsh	Ingestion, dermal contact	Adults and children/teenagers who come into contact with marsh sediment and surface water	Past, current, future
Indoor Air	VOCs	Zeneca	Air	Indoor air	Inhalation	Staff and students of Making Waves Education Program and Harborfront Tract businesses	Past, current, future
Outdoor air during remedial work	Metals, particulates (dust)	Zeneca	Air	Outdoor air	Inhalation	Bay Trail users, Harborfront Tract workers/residents, RFS workers	Past, future
Dust in Building 240	Metals, PCBs, pesticides	Zeneca	Dust	Indoor surfaces	Ingestion, dermal contact	Staff and students of Making Waves afterschool program	Past

VOC: volatile organic compound

PCB: polychlorinated biphenyls

Table C-2. Contaminants Detected in Sediment from the East Stege Marsh Prior to Removal Activities in 2004-2005 and Comparison/Screening Values, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (ppm)	Location of Maximum Detected Concentration	Average (Mean) Concentration (ppm)	Comparison/Screening Value/Source (ppm)
M e t a l s				
Aluminum	1,200-64,700	VC-6	23,200	100,000 Intermediate EMEG (child) 1,000,000 Intermediate EMEG (adult)
Antimony	41		8.5	30 Residential CHHSL 31 Residential PRG
Arsenic	4.8- 1,660		199	0.5 CREG 20 Chronic EMEG (child) 200 Chronic EMEG (adult)
Barium	3.8-103	E-16	54	30,000 Chronic EMEG (child) 400,000 Chronic EMEG (adult)
Beryllium	VC-10	M-4	0.66	100 Chronic EMEG (child) 1,000 Chronic EMEG (adult)
Cadmium	30 E-10		6.5	10 Chronic EMEG (child) 100 Chronic EMEG (adult)
Chromium	3.2-146	VC-6	75	210 Residential PRG
Cobalt 0.10-	2.2-16	M-2, M-4	9.0	500 Intermediate EMEG (child) 7,000 Intermediate EMEG (adult)
Copper	1.0- 5,390		581	500 Chronic EMEG (child) 7,000 Chronic EMEG (adult)
Lead	2.3- 740 M-11	VC-10	149	150 Cal-modified PRG
Manganese 0.15-1.6	8.0-329	E-9	207	3,000 RMEG (child) 40,000 RMEG (adult)

0.2-

SX-4

Table C-2. Contaminants Detected in Sediment from the East Stege Marsh Prior to Removal Activities in 2004-2005 and Comparison/Screening Values, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (ppm)	Location of Maximum Detected Concentration	Average (Mean) Concentration (ppm)	Comparison/Screening Value/Source (ppm)
Mercury	0.19-73	VC-10	3.7	23 Residential PRG 5 RMEG (child)* 70 RMEG (adult)*
Molybdenum	1.1-4.3	TM-10	2.2	300 RMEG (child) 4,000 RMEG (adult)
Nickel	2.0-115	CC-1	64	1,000 RMEG (child) 10,000 RMEG (adult)
Selenium	0.28-130	VC-4	11	300 Chronic EMEG (child) 4,000 Chronic EMEG (adult)
Silver	0.29-27	VC-10	1.3	300 RMEG (child) 4,000 RMEG (adult)
Thallium	0.10-1.7	VC-4	0.67	5 Residential CHHSL 5.2 Residential PRG
Vanadium	14-110	M-10	60	200 Intermediate EMEG (child) 2,000 Intermediate EMEG (adult)
Zinc	21-5,320	VC-10	1,202	20,000 Chronic EMEG (child) 200,000 Chronic EMEG (adult)
Pesticides				
4,4'-DDD	0.0097-1.8	SM-3	0.36	3 CREG 2.4 Residential PRG
4,4'-DDE	0.0015-0.23	SM-3	0.11	1.7 Residential PRG
4,4'-DDT	0.0021-0.54	21403	0.20	2 CREG 400 Intermediate EMEG
Aldrin	0.0003-0.0020	VC-10	0.01	2 Chronic EMEG (child) 20 Chronic EMEG (adult)

Table C-2. Contaminants Detected in Sediment from the East Stege Marsh Prior to Removal Activities in 2004-2005 and Comparison/Screening Values, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (ppm)	Location of Maximum Detected Concentration	Average (Mean) Concentration (ppm)	Comparison/Screening Value/Source (ppm)
Alpha-Hexachlorocyclohexane (BHC)	0.00054- 0.20	E-6	0.077	0.09 PRG (cancer)
Alpha-Chlorodane	0.00072-0.061	SM-3	0.016	30 Chronic EMEG (child) 400 Chronic EMEG (adult)
Beta- Hexachlorocyclohexane (BHC)	0.00054-0.13	SM-113	0.049	0.32 Residential PRG (cancer)
Delta-Hexachlorocyclohexane (BHC)	0.00014-0.070	E-6	0.03	0.32 Residential PRG (cancer)
Dieldrin	0.00088-0.037	E-8	0.0091	0.09 Residential PRG (cancer) 3 Chronic EMEG (child) 40 Chronic EMEG (adult)
Endosulfan I	0.00014-0.0097	SM-9	0.0013	100 Chronic EMEG (child) 1,000 Chronic EMEG (adult)
Endosulfan II	0.00033-0.0072	SM-9	0.0011	
Endosulfan Sulfate	0.00023-0.0053	VC-10	0.0013	none
Endrin	0.000080-0.019	E-9	0.00028	20 Chronic EMEG (child) 200 Chronic EMEG (adult)
Endrin Aldehyde	0.00025-0.018	E-9	0.0041	none
Endrin Ketone	0.00015-0.0020	E-5	0.00059	none
Gamma-Hexachlorocyclohexane (BHC)	0.0010-0.029	VC-2	0.0041	1.1 PRG (cancer)
Gamma-Chlorodane	0.00066-0.074	SM-6	0.021	1.6 Residential PRG (cancer) [†] 0.43 Residential CHHSL [†]
Heptachlor Epoxide	0.00015-0.0014	VC-1	0.00033	0.17 Residential PRG (cancer)
Mirex	0.00088-0.0026	SM-7	0.0014	40 Chronic EMEG (child) 600 Chronic EMEG (adult)

Table C-2. Contaminants Detected in Sediment from the East Stege Marsh Prior to Removal Activities in 2004-2005 and Comparison/Screening Values, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (ppm)	Location of Maximum Detected Concentration	Average (Mean) Concentration (ppm)	Comparison/Screening Value/Source (ppm)
Tetraethyl Pyrophosphate (TEPP)	0.011-0.11	E-16	0.018	none
Toxaphene	0.0039- 68	SM-10	6.8	0.6 CREG 50 Intermediate EMEG (child) 700 Intermediate EMEG (adult)
Proprietary Pesticides				
Bensulfide	0.028-4.5	VC-6	0.26	none
Butylate	0.016-0.43	SX-1	0.13	3,000 RMEG (child) 40,000 RMEG (adult)
Captan	0.35-0.89	VC-6	0.15	7,000 RMEG (child) 90,000 RMEG (adult)
Carbophenothion	0.029-0.16	VC-10	0.090	none
Cycloate	0.078-0.33	VC-6	0.12	none
EPTC	0.033-1.3	VC-6	0.28	1,000 RMEG (child) 20,000 RMEG (adult)
Fluorochloridone	0.020-0.12	SM-3	0.096	none
Fonofos	0.026-0.73	VC-5	0.082	100 RMEG (child) 1,000 RMEG (adult)
Metam sodium	0.53-1.2	TM-04	0.60	none
Molinate	0.028-2.3	VC-6	0.19	100 RMEG (child) 1,000 RMEG (adult)
Napropamide	0.018-0.46	SM-3	0.16	6,100 Residential PRG
Pebulate	0.035-6.8	VC-6	0.29	33,800 Residential PRG
R-25788	0.018-0.67	SX-1	0.13	none

Table C-2. Contaminants Detected in Sediment from the East Stege Marsh Prior to Removal Activities in 2004-2005 and Comparison/Screening Values, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (ppm)	Location of Maximum Detected Concentration	Average (Mean) Concentration (ppm)	Comparison/Screening Value/Source (ppm)
Vernolate	0.017-0.25	VC-6	0.10	50 RMEG (child) 700 RMEG (adult)
Polychlorinated biphenyls (PCBs)				
PCB #8	0.00008-0.0063	VC-10	0.0017	0.06 Residential PRG (cancer) 0.09 CHHSL
PCB #18	0.00045-0.018	VC-10	0.0066	
PCB #28	0.00049-0.054	VC-10	0.018	
PCB #44	0.00045-0.044	VC-10	0.017	
PCB #52	0.00051-0.049	VC-10	0.016	
PCB #66	0.00048-0.042	VC-10	0.016	
Total PCBs (based on location of max concentration)	0.00032-0.213	VC-10	0.075	
PCB #101	0.00045- 0.061	CC-1	0.018	0.06 Residential PRG (cancer) 0.09 CHHSL
PCB #105	0.00028-0.022	CC-1	0.0084	
PCB #118	0.00040-0.042	CC-1	0.016	
PCB #128	0.00043-0.022	CC-1	0.0040	
PCB #138	0.00047-0.087	CC-1	0.015	
PCB #153	0.00030-0.052	CC-1	0.010	
PCB #170	0.00045-0.019	CC-1	0.0031	

Table C-2. Contaminants Detected in Sediment from the East Stege Marsh Prior to Removal Activities in 2004-2005 and Comparison/Screening Values, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (ppm)	Location of Maximum Detected Concentration	Average (Mean) Concentration (ppm)	Comparison/Screening Value/Source (ppm)
PCB #180	0.00017-0.035	CC-1	0.0059	
PCB #187	0.00012-0.018	CC-1	0.0032	
PCB #195	0.00010-0.0032	CC-1	0.00054	
PCB #206	0.00045-0.0046	CC-1	0.0011	
Total PCBs (based on location of max concentration)	0.0008-0.3658		0.0852	
PCB #209	0.00024-0.0078	SM-7	0.0032	0.06 Residential PRG (cancer) 0.09 CHHSL

Data source (25,132)

ppm: parts per million

*Indicates comparison value for methylmercury (based on the potential for methylation of mercury in sediments)

†Indicates comparison value for chlorodane

Contaminants exceeding screening values in bold

PRG: EPA Region IX Preliminary Remediation Goal (based on noncancer health effects unless noted)

EMEG: ATSDR Environmental Media Evaluation Guide

CREG: ATSDR Cancer Risk Evaluation Guide for 1 in 1,000,000 increased cancer risk using EPA's cancer slope factors.

RMEG: Reference Dose Media Evaluation Guide based on EPA's Reference Dose.

CHHSL: Cal/EPA Human Health Screening Levels

Table C-3. Contaminants Detected in Surface Water from the East Stege Marsh (1997 and 2000), Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (µg/L)	Average (Mean) Concentration (µg/L)	Comparison/Screening Value/Source (µg/L)
Aluminum	730-4,300	1,873	20,000 Intermediate EMEG (child) 70,000 Intermediate EMEG (adult)
Antimony	<60- 170	48	4 RMEG (child) 10 RMEG (adult)
Arsenic	<5.0- 27.0	7.8	3 Chronic EMEG (child) 10 Chronic EMEG(adult)
Barium	24.0-37.0	31.5	6,000 Chronic EMEG (child) 20,000 Chronic EMEG (adult)
Beryllium	<2.0-3.2	2.9	20 Chronic EMEG (child) 70 Chronic EMEG (adult)
Cadmium	<5.0- 220	19.9	1 Chronic EMEG (child) 17 Chronic EMEG (adult)
Chromium III	<10.0-45.0	19.1	50 MCL
Copper	<10.0- 23,000	1,402	100 Intermediate EMEG (child) 400 Intermediate EMEG (adult)
Lead	<3.00-4.3	2.2	15 MCL/TT
Magnesium	10,000-1,100,000	806,154	Not available
Manganese	24.0- 550	227	500 RMEG (child) 2,000 RMEG (adult)
Mercury	<0.2-0.31	0.1	3 Chronic EMEG (child)* 10 Chronic EMEG (adult)*
Nickel	<20.0- 490	71.4	200 RMEG (child) 700 RMEG (adult)
Selenium	<5.0-22	12.6	50 Chronic EMEG (child) 200 Chronic EMEG (adult)
Zinc	25.0- 28,000	2,974	3,000 (child EMEG) 10,000 (adult EMEG)

Data source (25)

µg/L: microgram per liter

Contaminants exceeding screening values in **bold**

EMEG: ATSDR Environmental Media Evaluation Guide

RMEG: Reference Dose Media Evaluation Guide based on EPA's Reference Dose

MCL: Maximum Contaminant Level allowable in drinking water

TT: Treatment Technique. Lead is regulated by a Treatment Technique that requires systems to control the corrosiveness of their water. If more than 10% of tap water samples exceed the action level, water systems must take additional steps.

*EMEG for methylmercury (based on the potential for methylation of mercury in sediments and surface water)

Table C-3A. Contaminants Measured in Surface Water from the East Stege Marsh in 2007, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (µg/L)	Average (Mean) Concentration (µg/L)	Comparison/Screening Value/Source (µg/L)
Antimony	<10		4 RMEG (child) 10 RMEG (adult)
Arsenic	15-27	19.7	3 Chronic EMEG (child) 10 Chronic EMEG(adult)
Barium	32-43	36.3	6,000 Chronic EMEG (child) 20,000 Chronic EMEG (adult)
Beryllium	<2.0		20 Chronic EMEG (child) 70 Chronic EMEG (adult)
Cadmium	<5.0		1 Chronic EMEG (child) 17 Chronic EMEG (adult)
Chromium III	<5.0-5.3	3.4	50 MCL
Copper	<11-15	13	100 Intermediate EMEG (child) 400 Intermediate EMEG (adult)
Lead	<3.0		15 MCL/TT
Mercury	<0.2		3 Chronic EMEG (child)* 10 Chronic EMEG (adult)*
Nickel	<6.5-8.2	7.3	200 RMEG (child) 700 RMEG (adult)
Selenium	<10		50 Chronic EMEG (child) 200 Chronic EMEG (adult)
Zinc	23-25	24.3	3,000 (child EMEG) 10,000 (adult EMEG)

Data source (25,26)

µg/L: microgram per liter

Contaminants exceeding screening values in **bold**

EMEG: ATSDR Environmental Media Evaluation Guide

RMEG: Reference Dose Media Evaluation Guide based on EPA's Reference Dose

MCL: Maximum Contaminant Level allowable in drinking water

TT: Treatment Technique. Lead is regulated by a Treatment Technique that requires systems to control the corrosiveness of their water. If more than 10% of tap water samples exceed the action level, water systems must take additional steps.

*EMEG for methylmercury (based on the potential for methylation of mercury in sediments and surface water)

Table C-4. Contaminants Detected in Sediment from the East Stege Marsh After Removal Activities in 2004-2005 and Comparison/Screening Values, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (ppm)	Location of Maximum Detected Concentration	Average (Mean) Concentration (ppm)	Comparison/Screening Value/Source (ppm)
M e t a l s				
Antimony	4.7	SS-1	3.3	30 Residential CHHSL
Arsenic	6.1- 240	SS-20	36.6	0.5 CREG 0.39 Residential PRG 20 Chronic EMEG (child) 200 Chronic EMEG (adult)
Barium	34-130	SS-6	89.5	30,000 Chronic EMEG (child) 400,000 Chronic EMEG (adult)
Beryllium	0.29-0.97	SS-4	0.7	100 Chronic EMEG (child) 1,000 Chronic EMEG (adult)
Cadmium	0.37-3.1	SS-20	0.8	10 Chronic EMEG (child) 100 Chronic EMEG (adult)
Chromium	25-130	SS-11	77.1	210 Residential PRG
Cobalt	5.6-19	SS-9	12.6	500 Intermediate EMEG (child) 7,000 Intermediate EMEG (adult)
Copper	11-650	SS-11	138.3	500 Chronic EMEG (child) 7,000 Chronic EMEG (adult)
Lead	18- 250	SS-11	72.7	150 Cal-modified PRG

Table C-4. Contaminants Detected in Sediment from the East Stege Marsh After Removal Activities in 2004-2005 and Comparison/Screening Values, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (ppm)	Location of Maximum Detected Concentration	Average (Mean) Concentration (ppm)	Comparison/Screening Value/Source (ppm)
Mercury	7.5		1.1	5 RMEG (child)* 70 RMEG (adult)*
Molybdenum	1.1-6.5	SS-12	2.0	300 RMEG (child) 4,000 RMEG (adult)
Nickel	23-110	SS-12	70.3	1,000 RMEG (child) 10,000 RMEG (adult)
Selenium	0.05- SS-20	SS-20	1.6	300 Chronic EMEG (child) 4,000 Chronic EMEG (adult)
Thallium	0.34-1.1	SS-20	0.4	5 Residential CHHSL
Vanadium	29-120	SS-13	71.5	200 Intermediate EMEG (child) 2,000 Intermediate EMEG (adult)
Zinc	1.3-12 45-820	SS-20	291.3	20,000 Chronic EMEG (child) 200,000 Chronic EMEG (adult)

Data source (28)

*Indicates comparison value for methylmercury (based on the potential for methylization of mercury in sediments)

Contaminants exceeding screening values in **bold**

PRG: EPA Region IX Preliminary Remediation Goal (based on noncancer health effects unless noted)

EMEG: ATSDR Environmental Media Evaluation Guide

CREG: ATSDR Cancer Risk Evaluation Guide for 1 in 1,000,000 increased cancer risk

RMEG: Reference Dose Media Evaluation Guide based on EPA's Reference Dose

CHHSL: Cal/EPA Human Health Screening Levels

Table C-5. Noncancer Dose Estimates for Historic Exposure (prior to 2004) to Contaminants Exceeding Screening Values in Sediment and/or Surface Water in the East Stege Marsh, Zeneca/Campus Bay, 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)
	Maximum Concentration	Average Concentration	Maximum Concentration	Average Concentration	
Antimony	Sediment 0.000003	Sediment 0.0000006	Sediment 0.000001	Sediment 0.0000002	0.0004 (RfD)
	Surface water 0.00005	Surface water 0.00001	Surface water 0.00004	Surface water 0.00001	
Arsenic	Sediment 0.0001	Sediment 0.00001	Sediment 0.00005	Sediment 0.000006	0.0003 (MRL)
	Surface water 0.000008	Surface water 0.000002	Surface water 0.000005	Surface water 0.000001	
Cadmium	Sediment 0.000002	Sediment 0.0000005	Sediment 0.0000007	Sediment 0.0000002	0.0002 (MRL)
	Surface water 0.00007	Surface water 0.000006	Surface water 0.00004	Surface water 0.000004	
Copper	Sediment 0.0004	Sediment 0.00004	Sediment 0.0001	Sediment 0.00001	0.01 (MRL)
	Surface water 0.007	Surface water 0.0004	Surface water 0.004	Surface water 0.0003	
Manganese	Sediment 0.00002	Sediment 0.00001	Sediment 0.000008	Sediment 0.000005	0.14 (RfD)
	Surface water 0.0002	Surface water 0.00006	Surface water 0.0001	Surface water 0.00004	
Mercury	Sediment 0.000005	Sediment 0.0000003	Sediment 0.000002	Sediment 0.0000001	0.0003 (MRL)*
	Surface water ND	Surface water ND	Surface water ND	Surface water ND	

Table C-5. Noncancer Dose Estimates for Historic Exposure (prior to 2004) to Contaminants Exceeding Screening Values in Sediment and/or Surface Water in the East Stege Marsh, Zeneca/Campus Bay, 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)
	Maximum Concentration	Average Concentration	Maximum Concentration	Average Concentration	
Nickel	Sediment 0.00001	Sediment 0.0000005	Sediment 0.000005	Sediment 0.0000002	0.02 (RfD)
	Surface water 0.0002	Surface water 0.00002	Surface water 0.00009	Surface water 0.00001	
Zinc	Sediment 0.0004	Sediment 0.00009	Sediment 0.0001	Sediment 0.00003	0.3 (MRL)
	Surface water 0.008	Surface water 0.0008	Surface water 0.005	Surface water 0.0006	
Polychlorinated biphenyls (PCBs) [†]	Sediment 0.00000004	Sediment 0.000000009	Sediment 0.00000002	Sediment 0.000000004	0.00002 (MRL)
	Surface water NA	Surface water NA	Surface water NA	Surface water NA	
Toxaphene	Sediment 0.000006	Sediment 0.0000006	Sediment 0.000002	Sediment 0.00000006	0.001 (MRL)
	Surface water NA	Surface water NA	Surface water NA	Surface water NA	

Data source (9,10,133)

Maximum surface sediment values used for estimating current exposure doses; “historic” calculation for surface water based on sample collected in 1997 and 2000, prior to any remedial actions in the marsh; dose estimates include ingestion and dermal exposure

ND: not detected at laboratory detection limit

NA: not analyzed

MRL: ATSDR Minimal Risk Level

*MRL for methylmercury (based on the potential for methylation of mercury in sediments and surface water)

[†]The highest maximum and average values for total PCBs measured at location CC-1

Exposure assumptions used in estimating dermal dose surface water (133-135)

CW = concentration in water (mg/L)

P = permeability constant (cm/hour) (chemical specific: antimony 0.001, arsenic 0.001, cadmium 0.001, copper 0.001, manganese 0.001, mercury 0.001, nickel 0.001, zinc 0.0006)

Conversion factor = liters to cm²

SA = exposed surface body area (cm²) adult = 5809 cm²; child = 5323 cm². Skin surface area (adult) from the U.S. Environmental Protection Agency (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) from the 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 (1 hour/day)

EF = exposure frequency (100 days/year)

ED = exposure duration – years of exposure (child: 11 years) (Adult: 30 years)

BW = body weight (kg) (for child 48.9 kg: average of females and males ages 8-18) (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/cm²)(SA)(ET)(EF)(ED)/(BW)(AT)

Exposure assumptions used in estimating dermal dose from sediment (32,133-135)

CS = concentration in sediment (mg/kg)

AF = soil to skin adherence factor (0.2 mg/cm²)

CF = conversion factor (10⁻⁶ kg/mg)

SA = exposed surface body area (cm²)

ABS = absorption factor (unitless) (chemical specific: antimony 0.001 arsenic 0.03, copper 0.01, manganese 0.001, mercury 0.01, nickel 0.01, toxaphene 0.05, zinc 0.001, PCBs 0.15)

Skin surface area (adult) 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) 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.

SA adult = 5803 cm²

SA child = 5323 cm²

ET = exposure time (1 hour/day)

EF = exposure frequency (100 days/year)

ED = exposure duration – years of exposure (child: 11 years) (Adult: 30 years)

BW = body weight (for child 48.9 kg: average 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)(ABS)(SA)(CF)(SA)(AF)(ET)(EF)(ED)/(BW)(AT)

Exposure assumptions used in estimating ingestion dose from surface water (32,134)

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 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)

Exposure assumptions used in estimating ingestion dose from sediment (32,134)

CS = chemical concentration in sediment (mg/kg)

IR = ingestion rate (mg/day) – (adult 100 mg/day)(child 200 mg/day – averaged over 16 hours/day (time spent awake)

ET = exposure time (1 hour/day)

EF = exposure frequency (100 days/year)

ED = exposure duration – years of exposure (child: 11 years) (adult: 30 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/10)(EF)(ED)(CF)/(BW)(AT)

Table C-6. Estimated Hazard Index from Exposure to Contaminants in Surface Water and Sediment in the East Stege Marsh Prior to 2004, Zeneca/Campus Bay, Richmond, California

Contaminant	Hazard Index Child/Teen		Hazard Index Adult	
	Maximum Concentration	Average Concentration	Maximum Concentration	Average Concentration
Metals	Sediment 0.5	Sediment 0.06	Sediment 0.2	Sediment 0.02
	Surface water 1.3	Surface water 0.1	Surface water 0.7	Surface water 0.05
Polychlorinated biphenyls (PCBs)	Sediment 0.003	Sediment 0.0009	Sediment 0.002	Sediment 0.0004
	Surface water NA	Surface water NA	Surface water NA	Surface water NA
Pesticides	Sediment 0.006	Sediment 0.0008	Sediment 0.003	Sediment 0.0003
	Surface water ND	Surface water ND	Surface water ND	Surface water ND
Total Hazard Index	Sediment 0.5	Sediment 0.06	Sediment 0.2	Sediment 0.02
	Surface water 1.3	Surface water 0.1	Surface water 0.7	Surface water 0.05

Hazard index = Sum (intake dose of each contaminant/toxicity value)

NA: not analyzed

ND: not detected at laboratory detection limit

Table C-7. Noncancer Dose Evaluation for Current and Future Exposure to Contaminants Exceeding Screening Values in Sediment and Surface Water in the East Stege Marsh, Zeneca/Campus Bay, Richmond, California

Contaminant	Dose Estimates (mg/kg/day)		Health Comparison/Toxicity Value (source) (mg/kg/day)	Hazard Quotient
	Child/Teen	Adult		
Arsenic	Sediment 0.00002	Sediment 0.000007	0.0003 (MRL)	0.09 (child) 0.04 (adult)
	Surface Water 0.000008	Surface Water 0.000005		
Mercury	Sediment 0.0000005	Sediment 0.0000002	0.0003 (MRL)*	0.002 (child) 0.0006 (adult)
	Surface Water not detected	Surface Water not detected		
			Hazard Index	0.09 (child) 0.04 (adult)

Dose estimations based on maximum concentrations of contaminants remaining in the East Stege Marsh, and include dermal and ingestion exposure.

mg/kg/day: milligram per kilogram per day

MRL: ATSDR Minimal Risk Level; *MRL for methylmercury (based on the potential for methylization of mercury in sediments and surface water)

Hazard quotient: intake dose/toxicity value; Hazard Index: sum of hazard quotients

Exposure assumptions used in estimating dermal dose from sediment (32,133-135)

CS = concentration in sediment (mg/kg)

AF = soil to skin adherence factor (0.2 mg/cm²)

CF = conversion factor (10⁻⁶ kg/mg)

ABS = absorption factor (unitless) (chemical specific: arsenic 0.03, mercury 0.01)

SA = exposed surface body area (cm²) Skin surface area (adult) 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) 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.

SA adult = 5803 cm²

SA child = 5323 cm²

ET = exposure time (1 hour/day)

EF = exposure frequency (100 days/year)

ED = exposure duration – years of exposure (child: 11 years) (Adult: 30 years)

BW = body weight (for child 48.9 kg: average 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)(ABS)(CF)(SA)(AF)(ET)(EF)(ED)/(BW)(AT)

Exposure assumptions used in estimating ingestion dose from sediment (32,134,135)

CS = chemical concentration in sediment (mg/kg)

IR = ingestion rate (mg/day) – (adult 100 mg/day)(child 200 mg/day – averaged over 16 hours/day [time spent awake])

ET = exposure time (1 hour/day)

EF = exposure frequency (100 days/year)

ED = exposure duration – years of exposure (child: 11 years) (adult: 30 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/10)(EF)(ED)(CF)/(BW)(AT)

Table C-8. Summary of Contaminants Detected in Ambient Air During Remedial Activities Conducted in 2004 and 2005, Zeneca/Campus Bay, Richmond California

Contaminant	Action Level ($\mu\text{g}/\text{m}^3$)	Phase I Average (Maximum) Detected Concentration ($\mu\text{g}/\text{m}^3$)	Phase II Average (Maximum) Detected Concentration ($\mu\text{g}/\text{m}^3$)	Phase III Average Detected Concentration ($\mu\text{g}/\text{m}^3$)
Copper	260	0.10 (0.57)	0.03 (0.06)	0.08 (0.33)
Lead	1.0*	0.07 (0.09)	Not detected above MDL of <0.04	Not detected above MDL of <0.05
Nickel	0.760	0.051 (0.051)	0.03 (0.12)	Not detected above MDL of <0.05
Zinc	2.3	0.12 (0.30)	0.05 (0.14)	0.08 (0.16)
Hydrogen Sulfide	42*	21.6 (60.4)	18.4 (61)	6.96 (21.8)
Formaldehyde	35	1.26 (6.0)	1.56 (3.80)	1.63 (5.60)

$\mu\text{g}/\text{m}^3$: microgram per cubic meter

*Bay Area Air Quality Management District regulatory levels for lead and hydrogen sulfide.

The action levels for lead and hydrogen sulfide correspond to a 24-hour average and 1-hour average, respectively. Averages were calculated using 1/2 method detection limit (MDL) for non-detects.

Table C-9. Summary of Contaminants Detected in Soil Gas Samples Collected in the Harborfront Business Tract, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (µg/m ³)	Number of Detections	Location of Maximum Detection	Soil Gas Screening Values (Source) (µg/m ³)
Acetone	48-1,500	35	HFSG-20-5	36,500 (Residential soil gas PRG)
Benzene	<6.4- 190		HFSG-4-5	122 (Industrial CHHSL)
2-Butanone (MEK)	<29-440	31	HFSG-4-5	104,286 (Residential soil gas PRG)
Carbon Disulfide	<31-120	1	HFSG-26-5	73,000 (Residential soil gas PRG)
Chloroform	<9.8-62 ³¹	2	HFSG-15-5	313 (Residential soil gas PRG)
1,1-Dichloroethane	<8.1-100	2	HFSG-20-5	118 (Residential soil gas PRG)
1,1-Dichloroethylene	<7.9-410	3	HFSG-20-5	20,805 (Residential soil gas PRG)
cis-1,2-Dichloroethylene	<7.9-77	1	HFSG-8-5	44,400 (Industrial CHHSL) 3,650 (Residential soil gas PRG)
trans-1,2-Dichloroethylene	<7.9-12	1	HFSG-8-5	88,700 (Industrial CHHSL) 7,300 (Residential soil gas PRG)
Ethylbenzene	<8.7-24	12	HFSG-30-5	175 (Residential soil gas PRG)
4-Ethyltoluene	<9.8-51	3	HFSG-28-5	Not available
Isopropanol	<2.7-4,300	28	HFSG-9-5	Not available
Styrene	<8.5 – 10	1	HFSG-18-5	105,805 (Residential soil gas PRG)
Toluene	<7.5 – 230	31	HFSG-18-5	378,000 (Industrial CHHSL)

Table C-9. Summary of Contaminants Detected in Soil Gas Samples Collected in the Harborfront Business Tract, Zeneca/Campus Bay, Richmond, California

Contaminant	Range of Concentrations (µg/m ³)	Number of Detections	Location of Maximum Detection	Soil Gas Screening Values (Source) (µg/m ³)
Trichloroethylene	<11 – 220	3	HFSG-5-5	1,770 (Industrial CHHSL)
Trichlorofluoromethane	<11 – 100	1	HFSG-28-5	73,000 (Residential soil gas PRG)
1,1,2-Trichloro-1,2,2-trifluoroethane	<15 – 48	3	HFSG-3-5	3,128,050 (Residential soil gas PRG)
1,2,4-Trimethylbenzene	<9.8 – 92	1	HFSG-28-5	Not available
1,3,5-Trimethylbenzene	<9.8 – 27	1	HFSG-28-5	Not available
Vinyl acetate	<11 – 220	4	HFSG-5-5	Not available
Vinyl chloride	<5.1 – 21	1	HFSG-20-5	44.8 (Industrial CHHSL)
m-p-Xylene,	<8.7 – 430	21	HFSG-19-5	887,000 (Industrial CHHSL)
o-Xylene	<8.7 – 66	16	HFSG-11-5	879,000 (Industrial CHHSL)
Xylenes (total)	<8.7 – 190	20	HFSG-11-5	879,000 (Industrial CHHSL)

Data source (41)

Contaminants exceeding soil gas screening values in **bold**.

Residential soil gas PRG (Preliminary Remediation Goal): EPA soil vapor screening value = ambient air PRG x 100 (136)

CHHSL: Cal/EPA Human Health Screening Levels

Table C-10. Summary of Indoor Air Sampling Results Collected in Building 240 (Making Waves Afterschool Program) and Health Comparison Values, Zeneca/Campus Bay, Richmond, California

Contaminant	Concentrations ($\mu\text{g}/\text{m}^3$)	Health Comparison Values ($\mu\text{g}/\text{m}^3$)
Benzene	<30	1,300 (acute REL) 60 (chronic REL) 30 (RfC)
Chlorobenzene	<37	1,000 (chronic REL)
Formaldehyde	11.3 9.1 (ppbv)	94 (acute REL) 27 ppbv (CARB/CDPH)*
Hydrogen Sulfide	<6.3	42 (acute REL) 10 (chronic REL)
Tetrachloroethylene	<35	20,000 (acute REL) 35 (chronic REL) 30 (RfC)

Data source (43)

$\mu\text{g}/\text{m}^3$: microgram per cubic meter

REL: OEHHA Reference Exposure Level

RfC: EPA Reference concentration

ppbv: parts per billion volume

*California Air Resources Board (CARB) and California Department of Public Health (CDPH) screening value for formaldehyde in classrooms

Table C-11. Volatile Organic Chemicals Detected in Soil Gas Samples Collected Near Building 240, Zeneca/Campus Bay, Richmond, California

Contaminant	Residential Soil Gas Screening Values (µg/m ³)	Soil Gas Sampling Locations							
		Sample Results (µg/m ³)							
		Mobile Laboratory Result/Fixed Laboratory Result							
		Lot 3-SGT-1	Lot 3-SGT-2	Lot 3-SGT-3	Lot 3-SGT-7	Lot 3-SGT-8	Lot 3-SGT-11	Lot 3-SGT-12	Lot 3-SGT-16
Acetone	36,500*	NA	NA	NA	NA/ 150	NA	NA	NA	28
Benzene	36.2 (CHHSL)	< 25	27	< 25	60 / 16	170	48	31	20
1,3-Butadine	Not available	NA	NA	NA	NA / 7.8	NA	NA	NA	6.4
2-Butanone	104,286*				NA / 44				12
Carbon Disulfide	73,000*	NA	NA	NA	NA / 4.2	NA	NA	NA	< 3.4
Ethyl Benzene	174.6*	< 80	< 80	< 80	< 80 / 18	< 80	< 80	< 80	15
4-Ethyltoluene	Not available	NA	NA	NA	NA / 5.1	NA	NA	NA	18
Heptane	Not available	NA	NA	NA	NA / 7.5	NA	NA	NA	< 4.4
Hexane	20,857*	NA	NA	NA	NA / 7.8	NA	NA	NA	7.0
4-Methyl-2-pentone	Not available	NA	NA	NA	NA / 5.1	NA	NA	NA	< 4.4
Tetrachloroethylene	180 (CHHSL)	< 80	< 80	< 80	< 80/ < 7.5	< 80	< 80	< 80	88
Toluene	135,000 (CHHSL)	94	< 80	110	150 / 57	150	110	100	51
1,3,5-Trimethylbenzene	Not available	NA	NA	NA	NA / 6.9	NA	NA	NA	7.2
1,2,4-Trimethylbenzene	Not available	NA	NA	NA	NA / 30	NA	NA	NA	20
m, p-Xylene	315,000 (CHHSL)	< 80	< 80	< 80	< 80 / NA	< 80	< 80	< 80	56
o-Xylene	315,000 (CHHSL)	< 80	< 80	< 80	< 80 / 22	< 80	< 80	< 80	19

Data source (2); Contaminants exceeding soil gas screening values in **bold**; µg/m³: microgram per cubic meter

*EPA soil vapor preliminary remedial goal (PRG) (ambient air PRG x 100) (136)

CHHSL: Cal/EPA Human Health Screening Levels

Table C-12. Results of Surface Wipe and Vacuum Dust Samples and Site-Specific Dust Health Comparison Values Collected in Building 240, Zeneca/Campus Bay, Richmond, California

Surface Wipe and Vacuum Dust Samples Collected and Health Comparison Values (µg/m ²)												
Dust Health Comparison Values	DDD	DDE	DDT	Dieldrin	Molinate	Toxaphene	Arsenic	Cadmium	Lead	Mercury	Selenium	Zinc
C: cancer NC: noncancer	5,000 C	3,500 C	3,600 NC 3,000 C	420 NC 74 C	17,000 NC	8,500 NC 1100 C	2,200 NC 108 C	1,800 NC 1,100 C	430 NC	2,700 NC	46,000 NC	2,700,000 NC
S a m p l e I . D .												
W1-01	<0.15	<0.15	<0.15	<0.15	<0.59	<7.41	48.9	2.96	69.3	2.52	2.74	351
W1-02	<0.19	<0.19	<0.19	<0.19	<0.76	<9.52	0.52	2.86	20.0	0.62	0.38	114
W1-04	<0.15	<0.15	<0.15	<0.15	<0.59	<7.41	0.26	0.15	17.4	0.07	0.11	67.4
W1-05	<0.17	<0.17	<0.17	<0.17	<0.67	<8.33	0.33	0.67	16.7	0.25	0.25	55.0
W2-03	<0.15	<0.15	<0.15	<0.15	<0.59	<7.41	0.19	0.07	13.3	0.04	0.11	52.2
V1-01	<0.004	<0.004	<0.004	<0.004	<0.016	<0.19	1.59	0.12	3.32	0.16	4.68	38.4
V1-02	<0.010	<0.010	0.018	<0.010	<0.041	<0.51	3.53	27.1	24.6	0.28	1.29	296
V1-03	<0.021	<0.021	0.038	<0.021	<0.083	<1.04	12.9	4.80	59.6	0.90	1.79	365
V1-03 (duplicate)	<0.021	<0.021	0.030	<0.021	<0.083	<1.04	13.8	1.32	55.9	1.16	2.53	423
V1-04	<0.003	<0.03	<0.003	<0.003	<0.013	<0.16	0.36	1.79	10.7	0.03	0.39	61.4
V1-06	<0.004	<0.004	0.004	<0.004	<0.017	<0.21	0.86	0.26	3.72	0.13	1.58	40.4
V2-05	<0.007	<0.007	0.008	<0.007	<0.029	<0.36	2.51	0.52	6.27	0.08	8.14	93.8

Data source (46,137)

µg/m³: microgram per cubic meter

Table C-13. Polychlorinated Biphenyls (PCBs) Congener Analysis and Toxic Equivalent Concentrations of Dust Wipe Samples Collected in Building 240, Zeneca/Campus Bay, Richmond, California

PCB No.	Target Analyte	W1-01			W1-02			W1-04			W1-05			W2-03		
		Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)		TEC (µg/m ²)
8	2,4'-Dichlorobiphenyl	0.0146			<0.0381						<0.0333			<0.0296		
18	2,2',5-Trichlorobiphenyl	0.0746			<0.0381			0.0127			0.0102			<0.0296		
28	2,4,4'-Trichlorobiphenyl	0.1974			0.0194			0.0338			0.0182			0.0117		
44	2,2',3,5'-Tetrachlorobiphenyl	0.6058			0.0190			0.0161			0.0192			<0.0296		
52	2,2',5,5'-Tetrachlorobiphenyl	0.9371			0.0402			0.0385			0.0304			0.0145		
66	2,3',4,4'-Tetrachlorobiphenyl	0.5110			0.0168			<0.0296			0.0125			<0.0296		
77	3,3',4,4'-Tetrachlorobiphenyl	0.0727	0.0001	7.27E-06	<0.0381	0.0001	1.91E-06	<0.0296			<0.0333	0.0001	1.65E-06	<0.0296	0.0001	
81	3,4,4',5-Tetrachlorobiphenyl	0.0600	0.0001	6.00E-06	<0.0381	0.0001	1.91E-06	<0.0296			<0.0333	0.0001	1.65E-06	<0.0296	0.0001	
101	2,2',4,5,5'-Pentachlorobiphenyl	3.2678			0.0758			0.0191			0.0517			0.0068		
105	2,3,3',4,4'-Pentachlorobiphenyl	0.8897	0.0001	8.90E-05	0.0770	0.0001	7.70E-06	<0.0296			0.0217	0.0001	2.17E-06	<0.0296	0.0001	
114	2,3,4,4',5-Pentachlorobiphenyl	0.0469	0.0005	2.34E-05	<0.0381	0.0005	9.53E-06	<0.0296			<0.0333	0.0005	8.25E-06	<0.0296	0.0005	
118	2,3',4,4',5-Pentachlorobiphenyl	2.5527	0.0001	2.55E-04	0.1684	0.0001	1.68E-05	<0.0296			0.0504	0.0001	5.04E-06	<0.0296	0.0001	
123	2',3,4,4',5-Pentachlorobiphenyl	<0.0296	0.0001	1.48E-06	<0.0381	0.0001	1.91E-06	<0.0296			<0.0333	0.0001	1.65E-06	<0.0296	0.0001	
126	3,3',4,4',5-Pentachlorobiphenyl	0.0308	0.1	3.08E-03	<0.0381	0.1	1.91E-03	<0.0296			<0.0333	0.1	1.65E-03	<0.0296	0.1	
128	2,2',3,3',4,4'-Hexachlorobiphenyl	0.4316			0.0437			<0.0296			0.0096			<0.0296		
138	2,2',3,4,4',5'-Hexachlorobiphenyl	2.8817			0.2593			<0.0296			0.0556			<0.0296		
153	2,2',4,4',5,5'-Hexachlorobiphenyl	2.0482			0.1631			<0.0296			0.0356			<0.0296		
		0.1539	0.0005	7.70E-05			-	<0.0296	0.0005				-	<0.0296	0.0005	

Table C-13. Polychlorinated Biphenyls (PCBs) Congener Analysis and Toxic Equivalent Concentrations of Dust Wipe Samples Collected in Building 240, Zeneca/Campus Bay, Richmond, California

PCB No.	Target Analyte	W1-01			W1-02			W1-04			W1-05			W2-03		
		Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)		TEC (µg/m ²)
157	2,3,3',4,4',5'-Hexachlorobiphenyl	0.0405	0.0005	2.03E-05	<0.0381	0.0005		<0.0296	0.0005		<0.0333	0.0005	8.25E-06	<0.0296		
167	2,3',4,4',5,5'-Hexachlorobiphenyl	0.0547	0.00001	5.47E-07	0.0083	0.00001	8.30E-08	<0.0296			<0.0333	0.00001	1.65E-07	<0.0296	0.00001	
169	3,3',4,4',5,5'-Hexachlorobiphenyl	<0.0296	0.01	1.48E-04	<0.0381	0.01		<0.0296			<0.0333	0.01	1.65E-04	<0.0296	0.01	
170	2,2',3,3',4,4',5-Heptachlorobiphenyl	0.2540	0.0001	2.54E-05	0.0499	0.0001	4.99E-06	<0.0296			0.0069	0.0001	6.93E-07	<0.0296	0.0001	
180	2,2',3,4,4',5,5'-Heptachlorobiphenyl	0.6467	0.00001	6.47E-06	0.1086	0.00001	1.09E-06	<0.0296			0.0150	0.00001	1.50E-07	<0.0296	0.00001	
187	2,2',3,4',5,5',6-Heptachlorobiphenyl	0.6571			0.0669			<0.0296			0.0126			<0.0296		
189	2,3,3',4,4',5,5'-Heptachlorobiphenyl	0.0296	0.0001	2.96E-06	<0.0381	0.0001	1.91E-06	<0.0296			<0.0333	0.0001	1.65E-06	<0.0296	0.0001	
195	2,2',3,3',4,4',5,6-Octachlorobiphenyl	0.0304						<0.0296			<0.0333			<0.0296		
206	2,2',3,3',4,4',5,5',6-Nonachlorobiphenyl	0.0106						<0.0296			<0.0333			<0.0296		
209	Decachlorobiphenyl							<0.0296			<0.0333			<0.0296		
Sum TECs		3.74E-03			2.17E-03			<0.03			1.84E-03			<0.03		

Data Source (46)

µg/m³: microgram per cubic meter

The table above show the PCB congener concentrations (µg/m²), the TEFs (toxic equivalent factors), the TECs (toxic equivalent concentrations), which are calculated by multiplying the TEF by PCB congener concentration.

The sum/total TEC is the value compared with the PCB health comparison value for dust (0.04 µg/m²) (see Table C-15 below).

½ the method detection limit (MDL) was used in calculating the TEC for non-detects. PCB congeners detected in **bold**.

Table C-14. Polychlorinated Biphenyls (PCBs) Congener Analysis and Toxic Equivalent Concentrations of Vacuum Dust Samples Collected in Building 240 in 2006, Zeneca/Campus Bay, Richmond, California

PCB No.	Target Analyte	V1-01			V1-02			V1-03		
		Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)		TEC (µg/m ²)	Result (µg/m ²)	TEF	TEC (µg/m ²)
8	2,4'-Dichlorobiphenyl	<0.0008			0.0049			0.0023		
18	2,2',5-Trichlorobiphenyl	0.0014			0.0202			0.0118		
28	2,4,4'-Trichlorobiphenyl	0.0025			0.0306			0.0289		
44	2,2',3,5'-Tetrachlorobiphenyl	0.0030			0.0216			0.0178		
52	2,2',5,5'-Tetrachlorobiphenyl	0.0070			0.0632			0.0408		
66	2,3',4,4'-Tetrachlorobiphenyl	0.0018			0.0260			0.0094		
77	3,3',4,4'-Tetrachlorobiphenyl	<0.0008	0.0001	4.00E-08	<0.00205	0.0001	1.00E-07	<0.0042	0.0001	2.05E-07
81	3,4,4',5-Tetrachlorobiphenyl	<0.0008	0.0001	4.00E-08	<0.00205	0.0001	1.00E-07	<0.0042	0.0001	2.05E-07
101	2,2',4,5,5'-Pentachlorobiphenyl	0.0103			0.0631			0.0445		
105	2,3,3',4,4'-Pentachlorobiphenyl	<0.0008	0.0001	4.00E-08	0.0189	0.0001	1.89E-06	<0.0042	0.0001	2.05E-07
114	2,3,4,4',5-Pentachlorobiphenyl	<0.0008	0.0005	2.00E-07	<0.00205	0.0005	5.00E-07	<0.0042	0.0005	1.03E-06
118	2,3',4,4',5-Pentachlorobiphenyl	0.0094	0.0001	9.41E-07	0.0529	0.0001	5.29E-06	0.0294	0.0001	2.94E-06
123	2',3,4,4',5-Pentachlorobiphenyl	<0.0008	0.0001	4.00E-08	<0.00205	0.0001	1.00E-07	<0.0042	0.0001	2.05E-07
126	3,3',4,4',5-Pentachlorobiphenyl	<0.0008	0.1	4.00E-05	<0.00205	0.1	1.00E-04	<0.0042	0.1	2.05E-04
128	2,2',3,3',4,4'-Hexachlorobiphenyl	<0.0008			0.0118			<0.0042		
138	2,2',3,4,4',5'-Hexachlorobiphenyl	0.0118			0.0525			0.0327		
153	2,2',4,4',5,5'-Hexachlorobiphenyl	0.0097			0.0458			0.0348		
156	2,3,3',4,4',5-Hexachlorobiphenyl	<0.0008	0.0005	2.00E-07	<0.00205	0.0005	5.00E-07	<0.0042	0.0005	1.03E-06
157	2,3,3',4,4',5'-Hexachlorobiphenyl	<0.0008	0.0005	2.00E-07	<0.00205	0.0005	5.00E-07	<0.0042	0.0005	1.03E-06
167	2,3',4,4',5,5'-Hexachlorobiphenyl	<0.0008	0.0000	2.00E-07	<0.00205	0.0005	5.00E-07	<0.0042	0.00001	2.05E-08
169	3,3',4,4',5,5'-Hexachlorobiphenyl	<0.0008	0.01	2.00E-07	<0.00205	0.01	1.00E-04	<0.0042	0.01	2.05E-05
170	2,2',3,3',4,4',5-Heptachlorobiphenyl	0.0014	0.0001	1.37E-07	0.0066	0.0001	6.64E-07	0.0036	0.0001	3.59E-07
180	2,2',3,4,4',5,5'-Heptachlorobiphenyl	0.0021	0.00001	2.11E-08	0.0094	0.00001	9.36E-08	0.0091	0.00001	9.13E-08
187	2,2',3,4',5,5',6-Heptachlorobiphenyl	0.0014			0.0058			0.0081		
189	2,3,3',4,4',5,5'-Heptachlorobiphenyl	<0.0008	0.0001	4.00E-08	<0.00205	0.0001	1.00E-07	<0.0042	0.0001	2.05E-07
195	2,2',3,3',4,4',5,6-Octachlorobiphenyl	<0.0008			<0.00205			<0.0042		
206	2,2',3,3',4,4',5,5',6-Nonachlorobiphenyl	<0.0008			<0.00205			<0.0042		
209	Decachlorobiphenyl	<0.0008			<0.00205			<0.0042		
Sum TECs		4.55E-05			1.19E-04			2.31E-04		

Table C-14. Polychlorinated Biphenyls (PCBs) Congener Analysis and Toxic Equivalent Concentrations of Vacuum Dust Samples Collected in Building 240 in 2006, Zeneca/Campus Bay, Richmond, California

PCB No.	Target Analyte	V1-04			V1-06			V2-05		
		Result (µg/m ²)	TEF	TEC (µg/m ²)	Result (µg/m ²)		TEC (µg/m ²)	Result (µg/m ²)	TEF	TEC (µg/m ²)
8	2,4'-Dichlorobiphenyl	0.0009			<0.0008			<0.0015		
18	2,2',5-Trichlorobiphenyl	0.0032			0.0010			0.0020		
28	2,4,4'-Trichlorobiphenyl	0.0062			0.0019			0.0117		
44	2,2',3,5'-Tetrachlorobiphenyl	0.0041						0.0017		
52	2,2',5,5'-Tetrachlorobiphenyl	0.0120			0.0049			0.0048		
66	2,3',4,4'-Tetrachlorobiphenyl	0.0021			0.0011			<0.0015		
77	3,3',4,4'-Tetrachlorobiphenyl	<0.0006	0.0001	3.00E-08	<0.0008	0.0001	4.00E-08	<0.0015	0.0001	7.50E-08
81	3,4,4',5-Tetrachlorobiphenyl	<0.0006	0.0001	3.00E-08	<0.0008	0.0001	4.00E-08	<0.0015	0.0001	7.50E-08
101	2,2',4,5,5'-Pentachlorobiphenyl	0.0127			0.0064			0.0037		
105	2,3,3',4,4'-Pentachlorobiphenyl	<0.0006	0.0001	3.00E-08	<0.0008	0.0001	4.00E-08	<0.0015	0.0001	7.50E-08
114	2,3,4,4',5-Pentachlorobiphenyl	<0.0006	0.0005	1.50E-07	<0.0008	0.0005	2.00E-07	<0.0015	0.0005	3.75E-07
118	2,3',4,4',5-Pentachlorobiphenyl	0.0067	0.0001	6.69E-07	0.0059	0.0001	5.94E-07	0.0032	0.0001	3.19E-07
123	2',3,4,4',5-Pentachlorobiphenyl	<0.0006	0.0001	3.00E-08	<0.0008	0.0001	4.00E-08	<0.0015	0.0001	7.50E-08
126	3,3',4,4',5-Pentachlorobiphenyl	<0.0006	0.1	3.00E-05	<0.0008	0.1	4.00E-05	<0.0015	0.1	7.50E-05
128	2,2',3,3',4,4'-Hexachlorobiphenyl	0.0012			<0.0008			<0.0015		
138	2,2',3,4,4',5'-Hexachlorobiphenyl	0.0065			0.0065			0.0028		
153	2,2',4,4',5,5'-Hexachlorobiphenyl	0.0072			0.0058			0.0031		
156	2,3,3',4,4',5-Hexachlorobiphenyl	<0.0006	0.0005	1.50E-07	<0.0008	0.0005	2.00E-07	<0.0015	0.0005	3.75E-07
157	2,3,3',4,4',5'-Hexachlorobiphenyl	<0.0006	0.0005	1.50E-07	<0.0008	0.0005	2.00E-07	<0.0015	0.0005	3.75E-07
167	2,3',4,4',5,5'-Hexachlorobiphenyl	<0.0006	0.00001	3.00E-09	<0.0008	0.00001	4.00E-09	<0.0015	0.00001	7.50E-09
169	3,3',4,4',5,5'-Hexachlorobiphenyl	<0.0006	0.01	3.00E-06	<0.0008	0.01	4.00E-06	<0.0015	0.01	7.50E-06
170	2,2',3,3',4,4',5-Heptachlorobiphenyl	0.0007	0.0001	7.34E-08	0.0009	0.0001	9.08E-08	<0.0015		7.50E-08
180	2,2',3,4,4',5,5'-Heptachlorobiphenyl	0.0012	0.00001	1.23E-08	0.0015	0.00001	1.50E-08	0.0009	0.00001	9.27E-09
187	2,2',3,4',5,5',6-Heptachlorobiphenyl	0.0012			0.0010			0.0007		
189	2,3,3',4,4',5,5'-Heptachlorobiphenyl	<0.0006	0.0001	3.00E-08	<0.0008	0.0001	4.00E-08	<0.0015		7.50E-08
195	2,2',3,3',4,4',5,6-Octachlorobiphenyl	<0.0006			<0.0008			<0.0015		
206	2,2',3,3',4,4',5,5',6-Nonachlorobiphenyl	<0.0006			<0.0008			<0.0015		
209	Decachlorobiphenyl	<0.0006			<0.0008			<0.0015		
Sum TECs		3.41E-05			4.51E-05			8.37E-05		

Data Source (46)

The table above show the PCB congener concentrations (µg/m²), the TEFs (toxic equivalent factors), the TECs (toxic equivalent concentrations), which are calculated by multiplying the TEF by PCB congener concentration.

The sum/total TEC is the value compared with the PCB health comparison value for dust (0.04 µg/m²) (see Table C-15 below). ½ the method detection limit (MDL) was used in calculating the TEC for non-detects. PCB congeners detected in **bold**.

Table C-15. Summary of Results for Polychlorinated Biphenyl (PCB) Analyses of Surface Wipe and Vacuum Dust Samples Collected in Building 240, Zeneca/Campus Bay, Richmond, California

Sample ID	Sum Toxic Equivalent Concentration ($\mu\text{g}/\text{m}^2$)	Dust Health Comparison Value ($\mu\text{g}/\text{m}^2$)
W1-01	0.004	0.04
W1-02	0.002	0.04
W1-04	<0.03	0.04
W1-05	0.002	0.04
W2-03	<0.03	0.04
V1-01	0.0005	0.04
V1-02	0.0001	0.04
V1-03	0.0002	0.04
V1-03 (duplicate)	0.0002	0.04
V1-04	0.00003	0.04
V1-06	0.00005	0.04
V2-05	0.00008	0.04

Data source (46,137)

$\mu\text{g}/\text{m}^2$: micrograms per square meter

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 contaminants of concern (chemicals exceeding health comparison or screening values) detected in air, soil, surface water, or groundwater, around the Zeneca site.

Arsenic (138)

- 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 $\mu\text{g}/\text{m}^3$ (reproductive, developmental effects in mice).
- Chronic reference exposure level (REL) = 0.03 $\mu\text{g}/\text{m}^3$ (developmental, cardiovascular, nervous system in mice).
- Oral cancer slope factor = 1.5 mg/kg/day.
- Inhalation slope factor = 12 mg/kg/day.
- Inhalation unit risk (U.S. Environmental Protection Agency) = 0.0043 $\mu\text{g}/\text{m}^3$.
- 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).

Antimony (139,140)

- Naturally-occurring element (metal) found in small amounts in the earth’s crust.
- Antimony ores are mined and then either changed into antimony metal or combined with oxygen to form antimony oxide.
- Inhalation of high levels of antimony can damage the lungs and cardiovascular system.
- Ingestion of antimony can result in gastrointestinal effects.
- Oral reference dose (RfD) = 0.0004 mg/kg/day (decreased longevity, decreased blood glucose and alteration in cholesterol in animals).
- Carcinogenicity: not evaluated (U.S. Environmental Protection Agency); possibly carcinogenic to humans (International Agency for Research on Cancer).

Cadmium (139,141,142)

- 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).
- Chronic reference exposure level (REL) = 0.02 $\mu\text{g}/\text{m}^3$ (kidney and respiratory damage in humans).
- Inhalation slope factor = 15 mg/kg/day.
- 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 (143)

- 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).

Hydrogen Sulfide (H₂S) (144,145)

- H₂S released primarily as a gas and remains in the atmosphere for 18 hours.
- Naturally-occurring in crude petroleum, natural gas, volcanic gases, and hot springs.
- Can be produced during bacterial breakdown of organic matter
- Byproduct of the decomposition process in sediments with little or no oxygen is commonly found in marsh areas.
- Brief exposures to high concentrations of H₂S (greater than 500 ppm) can cause a loss of consciousness and possibly death.
- Exposure to low concentrations of H₂S may cause irritation to the eyes, nose, or throat. It may also cause difficulty in breathing for some asthmatics.
- Acute inhalation minimal risk level (MRL) = 0.07 ppm (respiratory effects in humans).
- Intermediate inhalation minimal risk level (MRL) = 0.02 ppm (nasal effects in rats).

- Acute reference exposure level (REL) = $42 \mu\text{g}/\text{m}^3$ (respiratory irritation).
- Chronic reference exposure level (REL) = $10 \mu\text{g}/\text{m}^3$ (respiratory irritation).
- The Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), and the EPA have not classified hydrogen sulfide for carcinogenicity

Lead (29,146)

- 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 ($30 \mu\text{g}/\text{dL}$) 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 have been shown to occur at blood lead levels extending below $30 \mu\text{g}/\text{dL}$, and possibly as low as $15\text{-}20 \mu\text{g}/\text{dL}$; the developing nervous system of a young child can be adversely affected at blood lead levels below $10 \mu\text{g}/\text{dL}$.
- 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 $10 \mu\text{g}/\text{dL}$.
- The State of California recommends exposure reduction/mitigation actions for pregnant women with BBLs of $10 \mu\text{g}/\text{dL}$ or greater.
- Occupational Safety and Health Administration requires workers with a blood lead level above $50 \mu\text{g}/\text{dL}$ 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 (147)

- 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 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$ (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) (139,141,148)

- 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.
- Historically used as a component in caulking compounds.
- 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 effects 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).
- Oral cancer slope factor = 2 mg/kg/day (liver cancer).
- Inhalation cancer slope factor = 5 mg/kg/day (liver cancer).
- 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).

Toxaphene (149)

- One of the most heavily used insecticides until 1982.
- Breaks down slowly in the environment; quickly broken down and excreted from the body.

- Breathing, eating, or drinking high levels of toxaphene can damage the lungs, nervous system, liver, and kidneys.
- Intermediate oral minimal risk level (MRL) = 0.001 mg/kg/day (hepatic effects in animals)
- Inhalation unit risk (U.S. Environmental Protection Agency) = 0.00032 $\mu\text{g}/\text{m}^3$ (hepatocellular tumors in mice and thyroid tumors in rats-derived from oral studies).
- Oral cancer slope factor (U.S. Environmental Protection Agency) = 1.1 mg/kg/day (hepatocellular tumors in mice and thyroid tumors in rats).
- Carcinogenicity: probable human carcinogen, based on sufficient evidence of carcinogenicity in animals (U.S. Environmental Protection Agency); possibly carcinogenic to humans (International Agency for Research on Cancer).

Appendix E. Exposure Assumptions and Equations Used for Estimating Increased Cancer Risk and Cancer Slope Factors

Exposure assumptions used in estimating increased cancer risk from dermal contact with sediment (32,133,134,150)

CS = concentration in sediment (mg/kg)

SSA = soil to skin adherence factor (0.2 mg/cm²) child/teenager; (0.07 mg/cm²) adult

CF = Conversion factor (10⁻⁶ kg/mg)

SA = Skin surface area (cm²/event) – Skin surface area (adult = 5809 cm²) from U.S.

Environmental Protection Agency (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 cm²) 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, PCBs 0.15, captan 0.1, remaining pesticides 0.05)

Skin surface area (adult) from the EPA Exposure Factors Handbook, averaging the 50th

EF = exposure frequency (100 events/year)

ED = exposure duration – years of exposure (child: 11 years) (adult: 30 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 (days) – (365 days/year)(70 years)

Equation for estimating theoretical increased cancer risk:

$[(CS)(SSA)(CF)(SA)(AF)(EF)(ED)/(BW)(AT)]$ (cancer slope factor)

Exposure assumptions used in estimating increased cancer risk from ingestion of sediment (32,134,150)

CS = chemical concentration in sediment (mg/kg)

IR = ingestion rate (mg/day) – (adult 100 mg/day) (child 200 mg/day) over 16 hours (time spent awake) (IR adjusted to account for 1 ET)

ET = exposure time (2.6 hour/day)

EF = exposure frequency (100 days/year)

ED = exposure duration – years of exposure (child: 11 years) (adult: 30 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) – (365 days/year)(70 years)

Equation for estimating theoretical increased cancer risk:

$[(CS)(IR/16)(ET)(EF)(ED)(CF)/(BW)(AT)]$ (cancer slope factor)

Exposure assumptions used in estimating increased cancer risk from dermal contact with surface water (133-135,150)

CW = concentration in water (mg/L)

P = permeability constant (cm/hour) (chemical specific: arsenic 0.001)

Conversion factor = liters to cm²

SA = Skin surface area (cm²) (adult = 5809 cm²) 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 cm²) 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 (1.0 hour/day)

EF = exposure frequency (100 days/year)

ED = exposure duration – years of exposure (child: 11 years) (adult: 30 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 (days) – (365 days/year)(70 years)

Equation for estimating theoretical increased cancer risk:

$[(CW)(P)(0.001L/cm^2)(SA)(ET)(EF)(ED)/(BW)(AT)](\text{cancer slope factor})$

Exposure assumptions used in estimating increased cancer risk from ingestion of surface water (32,134)

CW = chemical concentration in water (mg/L)

IR = ingestion rate (0.05 liter/hour)

ET = exposure time (1.0 hour/day)

EF = exposure frequency (100 days/year)

ED = exposure duration – years of exposure (child: 11 years) (adult: 30 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) – (365 days/year)(70 years)

Equation for estimating theoretical increased cancer risk:

$[(CW)(IR)(ET)(EF)(ED)/(BW)(AT)](\text{cancer slope factor})$

Cancer slope factors used to estimate increased cancer risk (135,139,141)

Contaminant	Oral Cancer Slope Factor (mg/kg/day)
Arsenic	1.5
4,4'-DDD	0.24
4,4'-DDE	0.34
4,4-DDT	0.34
Aldrin	17
Alpha-BHC	6.3
Alpha Chlorodane	1.2
Beta-BHC	1.8
Dieldrin	16
Gamma-BHC	1.3
Gamma Chlorodane	1.2
Heptachlor Epoxide	13
Toxaphene	1.2
Polychlorinated biphenyls	5

Note: There is no oral cancer slope factor for cadmium.