ATTACHMENT #2

ADAMS BROADWELL JOSEPH & CARDOZO
A PROFESSIONAL CORPORATION
ATTORNEYS AT LAW
651 GATEWAY BOULEVARD, SUITE 900
SOUTH SAN FRANCISCO, CA 94080-7059
TEL: (650) 589-1660
FAX: (650) 589-5062
rdrury@adamsbroadwell.com

SACRAMENTO OFFICE
1225 8th STREET, SUITE 550
SACRAMENTO, CA 95814-4810
TEL: (916) 444-6201
FAX: (916) 444-6209

DANIEL L. CARDOZO
RICHARD T. DRURY
THOMAS A. ENSLOW
TANYA A. GULESSERIAN
MARC D. JOSEPH
OSHA R. MESERVE
SUMA PEESAPATI

FELLOW
KEVIN S. GOLDEN

OF COUNSEL
THOMAS R. ADAMS
ANN BROADWELL

May 4, 2005

Secretary of the Planning Commission, and
Honorable Members of the
City of Long Beach Planning Commission
C/o Ms. Anita Garcia, Project Manager
Department of Planning and Building
City Hall, 5th floor
333 West Ocean Blvd.
Long Beach, CA 90802
VIA FACSIMILE: 562-570-6610

Re: Opposition to Certification of Long Beach Memorial Medical Center Expansion Environmental Impact Report and Request for Supplemental EIR.

Honorable Members of the City of Long Beach Planning Commission:

We are writing on behalf of the SEIU United Healthcare Workers – West ("SEIU") with regard to the City's Long Beach Memorial Medical Center Expansion (the "Project") Environmental Impact Report (State Clearinghouse No. 2004081142) ("the EIR"). As explained more fully below, the EIR does not comply with the requirements of the California Environmental Quality Act ("CEQA").[1] The City may not approve the Project or grant any permits for the Project until an adequate Environmental Impact Report ("EIR") is prepared and circulated for public review and comment.

Many members of SEIU live and work in areas in and around Long Beach and in the immediate vicinity of the Project. They are concerned about sustainable land use and development in the City. Poorly planned and environmentally detrimental projects may jeopardize future jobs by making it more difficult and more expensive for business and industry to expand in the region, and by making it less desirable for businesses to locate and people to live here. Continued degradation can, and has, caused construction moratoriums and other restrictions on growth that, in turn, reduce future employment opportunities. Additionally, the

[1] Public Resources Code §§ 21000 *et seq.*

members live in the communities that suffer the impacts of environmentally detrimental projects. Union members breathe the same polluted air that others breathe and suffer the same health and safety impacts.

Finally, SEIU members are concerned about projects that carry serious environmental risks without providing countervailing employment and economic benefits to local workers and communities. CEQA's most fundamental mandate is that an agency may only approve a project having significant impacts if it finds that "specific overriding economic, legal social technological, or other benefits of the project outweigh the significant effects on the environment." (CEQA section 21081(b)). Our goal is consistent with the legislative purpose embodied in CEQA to maximize the Project's economic and other benefits, while minimizing its impacts to the environment. Futhermore, SEIU members are also patients and caregivers in the Long Beach community. SEIU wishes to ensure that the hospital is constructed in a manner that safeguards the health and safety of patients and employees at the hospital.

Due to the deficiencies in the EIR, a supplemental EIR ("SEIR") should be prepared to analyze the Project's impacts and re-circulated for public review. CEQA requires re-circulation of an EIR when significant new information is added to the EIR following public review but before certification. (Pub. Res. Code § 21092.1.) The Guidelines clarify that new information is significant if "the EIR is changed in a way that deprives the public of a meaningful opportunity to comment upon a substantial adverse environmental effect of the project" including, for example, "a disclosure showing that ... [a] new significant environmental impact would result from the project." (CEQA Guidelines § 15088.5.) Significant new information will be required to analyze and mitigate the deficiencies identified in the EIR. An SEIR is therefore required.

We submit these comments pursuant to the ten-day review period commencing on April 25, 2005, but reserve the right to supplement these comments at any time prior to or through the date of final project approval by the City Council, and at any later hearings and proceedings for this Project.[2] We incorporate by reference all comments that have been or will be submitted by any other entities, agencies, organizations or individuals concerning the Project and/or the EIR.

---

[2] *Bakersfield Citizens for Local Control v. City of Bakersfield* (2004) 124 Cal. App. 4th 1184; *Galante Vineyards v. Monterey Water Dist.* (1997) 60 Cal. App. 4th 1109.

I. INTRODUCTION: LEGAL STANDARDS

CEQA generally requires that an agency analyze the potential environmental impacts of its proposed actions in an environmental impact report ("EIR"). (Pub. Res. Code § 21100.) The EIR is the very *heart* of CEQA.[3] "The 'foremost principle' in interpreting CEQA is that the Legislature intended the act to be read so as to afford the fullest possible protection to the environment within the reasonable scope of the statutory language."[4]

CEQA has two basic purposes, neither of which the Long Beach Memorial EIR satisfies. First, CEQA is designed to inform decision makers and the public about the potential, significant environmental effects of a project.[5] "Its purpose is to inform the public and its responsible officials of the environmental consequences of their decisions *before* they are made. Thus, the EIR 'protects not only the environment but also informed self-government.'"[6] The EIR has been described as "an environmental 'alarm bell' whose purpose it is to alert the public and its responsible officials to environmental changes before they have reached ecological points of no return."[7]

Second, CEQA directs public agencies to avoid or reduce environmental damage when possible by requiring alternatives or mitigation measures.[8] The EIR serves to provide public agencies and the public in general with information about the effect that a proposed project is likely to have on the environment and to "identify ways that environmental damage can be avoided or significantly reduced." (Guidelines §15002(a)(2).) If the project has a significant effect on the environment, the agency may approve the project only upon finding that it has "eliminated or substantially lessened all significant effects on the environment where feasible" and that any unavoidable significant effects on the environment are "acceptable due to

---

[3] *Dunn-Edwards v. BAAQMD* (1992) 9 Cal.App.4th 644, 652.
[4] *Communities for a Better Environment v. Calif. Resources Agency* (2002) 103 Cal. App. 4th 98, 109.
[5] 14 Cal. Code Regs. ("CEQA Guidelines") § 15002(a)(1).
[6] *Citizens of Goleta Valley v. Board of Supervisors* (1990) 52 Cal.3d 553, 564.
[7] Berkeley Keep Jets Over the Bay v. Bd. of Port Comm'rs. (2001) 91 Cal. App. 4th 1344, 1354 ("Berkeley Jets"); County of Inyo v. Yorty (1973) 32 Cal.App.3d 795, 810.
[8] CEQA Guidelines § 15002(a)(2) and (3). *See also, Berkeley Jets,* 91 Cal. App. 4th 1344, 1354; *Citizens of Goleta Valley v. Board of Supervisors* (1990) 52 Cal.3d 553, 564; *Laurel Heights Improvement Ass'n v. Regents of the University of California* (1988) 47 Cal.3d 376, 400.

overriding concerns" specified in CEQA section 21081. (Guidelines, § 15092, subd. (b)(2)(A) & (B).)

Standard of Review: While the court is to review an EIR using an "abuse of discretion" standard, "*the reviewing court is not to 'uncritically rely on every study or analysis presented by a project proponent in support of its position. A 'clearly inadequate or unsupported study is entitled to no judicial deference.'* "[9] As the court stated in *Berkeley Jets*[10]:

> A prejudicial abuse of discretion occurs " 'if the failure to include relevant information precludes informed decisionmaking and informed public participation, thereby thwarting the statutory goals of the EIR process.' [Citation.]" (*San Joaquin Raptor / Wildlife Rescue Center v. County of Stanislaus* (1994) 27 Cal. App. 4th 713, 722 [32 Cal. Rptr. 2d 704]; *Galante Vineyards v. Monterey Peninsula Water Management Dist.* (1997) 60 Cal. App. 4th 1109, 1117 [71 Cal. Rptr. 2d 1]; *County of Amador v. El Dorado County Water Agency* (1999) 76 Cal. App. 4th 931, 946 [91 Cal. Rptr. 2d 66].). . . "Our role here, as a reviewing court, is not to decide whether the board acted wisely or unwisely, but simply to determine whether the EIR contained sufficient information about a proposed project, the site and surrounding area and the projected environmental impacts arising as a result of the proposed project or activity to allow for an informed decision... [Citation.]" (*San Joaquin Raptor / Wildlife Rescue Center v. County of Stanislaus, supra,* 27 Cal. App. 4th at p. 718.)

---

[9] *Berkeley Jets,* 91 Cal. App. 4th 1344, 1355 (emphasis added), quoting, *Laurel Heights Improvement Assn. v. Regents of University of California,* 47 Cal. 3d 376, 391 409, fn. 12 (1988).

[10] 91 Cal. App. 4th at 1355.

II. THE EIR FAILS TO ACCURATELY DESCRIBE THE PROJECT OR ITS ENVIRONMENTAL SETTING.

The EIR is inadequate because it contains patently inconsistent Project descriptions throughout the document and fails to adequately describe the Project's environmental setting. "An accurate, stable and finite project description is the *sine qua non* of an informative and legally adequate EIR."[11] "[A] curtailed or distorted project description," on the other hand, "may stultify the objectives of the reporting process. Only through an accurate view of the project may affected outsiders and public decision-makers balance the proposal's benefit against its environmental costs, consider mitigation measures, assess the advantage of terminating the proposal (*i.e.*, the "no project" alternative) and weigh other alternatives in the balance."[12] As one analyst has noted:

> The adequacy of an EIR's project description is closely linked to the adequacy of the EIR's analysis of the project's environmental effects. If the description is inadequate because it fails to discuss the complete project, the environmental analysis will probably reflect the same mistake.[13]

The project description must include an accurate description of the project's environmental setting. An accurate description of the environmental setting is important because it establishes the baseline physical conditions against which a lead agency can determine whether an impact is significant.[14] Under CEQA, an EIR must include a description of the physical environmental conditions in the vicinity of the project, as they exist at the time the Notice of Preparation is published, from both a local and a regional perspective. (*Id.*) Knowledge of the regional setting is critical to an assessment of environmental impacts.[15]

---

[11] *County of Inyo v. City of Los Angeles* (1977) 71 Cal.App.3d 185, 192; *Berkeley Jets*, 91 Cal. App. 4th 1344, 1354; *Sacramento Old City Assn. v. City Council* (1991) 229 Cal. App. 3d 1011, 1023; *Stanislaus Natural Heritage Project v. County of Stanislaus* (1996) 48 Cal. App. 4th 182, 201.

[12] *Id. See also*, CEQA section 15124; *City of Santee v. County of San Diego*, 263 Cal.Rptr 340 (1989).

[13] Kostka and Zischke, "Practice Under the California Environmental Quality Act," §12.17.

[14] CEQA Guidelines § 15125(a).

[15] *Id.* at § 15125(c).

The courts are clear that an EIR must focus on impacts to the existing environment, not hypothetical situations.[16] The presentation of baseline information must be sufficiently detailed to make further analysis possible. (*Id.*) It must provide not only raw data but also analysis.[17] An EIR must provide an accurate description of the environmental baseline, because "[t]he impacts of the project must be measured against the 'real conditions on the ground.'"[18]

Here, the EIR's failure to correctly describe the existing physical conditions related to soil contamination precludes informed decisionmaking and informed public participation.

A. Inadequate Description of Existing Site Contamination

A CEQA document must disclose any existing toxic chemical contamination at the site so that the lead agency can propose ways to mitigate the contamination.[19] The EIR in this case fails even to characterize, quantify or specify the nature of very significant levels of toxic chemical contamination on the site.

Environmental expert John Paul Williams explains that the site of the proposed Project is heavily contaminated due to many old oil wells, and an abandoned "ravine" landfill. (Williams Research Letter, p.2, March 9, 2005). Contamination already discovered on site includes arsenic, lead, selenium, benzene, Freon, toluene, xylene, ethylbenzene, methane, hydrogen sulfide, and other volative organic compounds ("VOCs"). (Id. at p.3). Site contamination is so extensive that other portions of the site that have been developed, such as the Miller Children's Hospital, were required to install a methane mitigation system.

Despite this known contamination, the EIR fails to adequately define or describe the existing site contamination. As Mr. Williams explains, the EIR states that the concentration of total petroleum hydrocarbons ("TPH") as diesel and heavy

---

[16] *County of Amador vs. El Dorado County Water Agency* (1999) 76 Call. App. 4th 931, 954.
[17] *Id.* 76 Cal.App.4th at 955; see, *Environmental Planning & Information Council v. County of El Dorado* (1982) 131 Cal.App.3d 350, 355 (holding that an EIR should inventory and address the environment as it actually existed, not as it was proposed to be under the old General Plan.)
[18] *Save Our Peninsula Committee v. Monterey Board of Supervisors* (2001) 87 Cal.App.4th 99, 121.
[19] *McQueen v. Mid-Peninsula*,(1988) 202 Cal. App. 3d 1136.

hydrocarbons was 49,700 mg/kg, while a 1991 engineering report shows that levels are as high as 190,000 mg/kg. (Id. at p.5). The EIR nowhere explains this discrepancy.

The California Department of Toxic Substances Control ("DTSC") submitted written comments on the Project concluding that the EIR "did not provide sufficient description of the extent and nature of contamination existing at the site, or analysis of the potential impacts associated with potential RAW [remedial action workplan] activities. This is primarily due to the fact that information related to the extent and nature of the contamination is still being acquired and evaluated for the development of a draft RAW." (DTSC Comment, p. 2 (March 16, 2005)).

As discussed, CEQA requires a full disclosure and analysis of the existing environmental conditions. As DTSC concludes, the EIR patently fails to describe the extent and nature of substantial site contamination with highly toxic chemicals. An SEIR is therefore required to disclose this contamination and to propose feasible measures to remediate this impact.

B. Project Description is Internally Inconsistent.

As mentioned above, the Project description must be "accurate, stable and finite." By contrast, the EIR in this case contradicts itself repeatedly – often on the same page and concerning the same impacts. Such an internally inconsistent project description fails to meet the most basic requirements of CEQA.

For example, the EIR clearly states that "proposed project would be anticipated to have significant impacts to air quality during operations due to the exceedance of the SCACMD significance threshold for NOx," (EIR at 3.2-12,13,15), but then contradicts itself in the following cumulative impacts section by stating "the operational emissions from the proposed project are individually insignificant." (*Id.* at 3.2-16). The EIR again contradicts itself when dismissing the cumulative impacts for hazardous materials. The EIR explained that the "proposed project has the potential to result in significant impacts to the public or the environment related to the routine transport, use, or disposal of hazardous materials," (*Id.* at 3.5-9), and that "[o]ff-site transport and disposal routes for biomedical, radiological, hazardous, and nonhazardous may include the route . . . within 0.25 miles of the [Jackie Robinson Elementary] school." (*Id.* 3.5-11). The EIR contradicts itself when it states two pages later "hazards and hazardous materials impacts expected from

the implementation of the proposed project do no affect lands outside the boundaries of the proposed project site..." (*Id.* 3.5-13).

Professional engineer Tom Brohard points out numerous inconsistencies in the project description. For example:

- Page 2-9 indicates completion of construction for the Todd Cancer Institute Phase I in September 2006, while just four pages later, on page 2-13, the document states that the same facility will be completed in December 2007 – over one year later.
- Page 2-9 states that the Todd Cancer Institute Phase II will be 42,300 square feet while four pages later the same facility is described as being 45,500 square feet. (page 2-13).
- Page 2-10 states that the Miller Children's Hospital Phase I will be 129,220 square feet, but fives pages later, the EIR states that the same facility will be 124,500 square feet.
- Page 2-10 states that the Miller Children's Hospital Phase II will be 86,030 square feet, but at page 2-15, the same facility is described as being 73,500 square feet.

These internal inconsistencies must be clarified in a new SEIR.

C. The Environmental Setting Fails to Discuss New Ozone Standards.

The environmental setting must include a discussion of applicable environmental standards, regulatory frameworks and plans. (CEQA Guidelines §15125.) The EIR lists several state and federal air quality standards to apply in the area, but fails to mention the new 8-hour ozone standard adopted by the California Air Resources Board ("CARB") on April 28, 2005. Since the document fails entirely to mention this standard, there is no analysis of how the project may affect the standard, or the region's ability to comply with the standard.

On April 28, 2005, CARB adopted a new 8-hour ozone standard of 0.070 parts per million. (Exhibit 1). The EIR cites only the 1-hour ozone standard of 0.09 ppm and does not mention the 8-hour standard. (EIR, p. 3.2-3). Ozone presents very significant human health impacts, and the Los Angeles region has the worst ozone problem in the nation. The EIR admits that the Project will increase emissions of ozone precursors nitrogen oxides (NOx) and volatile organic compounds (VOCs).

(EIR section 3.2). Thus, the Project will exacerbate the region's already unacceptable ozone problem.[20] Since the EIR has not yet been certified, it should be revised to address the 8-hour ozone standard, including how the Project may affect the region's ability to meet that standard, and analyze the feasible measures that may reduce this impact.

An understanding of the nature of ozone pollution will help to understand why an individual and cumulative impacts analysis is so vitally important to understand the impacts of the Project. Ozone, the principal element of smog, is a secondary pollutant produced when two precursor air pollutants — volatile organic compounds ("VOCs") and nitrogen oxides ("NOx") — react in sunlight.[21] VOCs and NOx are emitted by a variety of sources, including cars, trucks, industrial facilities, petroleum-based solvents, and diesel engines.

The human health and associated societal costs from ozone pollution are extreme. In proposing a new rulemaking limiting emissions of NOx and particulate matter from certain diesel engines, EPA summarized the effects of ozone on public health:

> "A large body of evidence shows that ozone can cause harmful respiratory effects, including chest pain, coughing and shortness of breath, which affect people with compromised respiratory systems most severely. When inhaled, ozone can cause acute respiratory problems; aggravate asthma; cause significant temporary decreases in lung function of 15 to over 20 percent in some healthy adults; cause inflammation of lung tissue, produce changes in lung tissue and structure; may increase hospital admissions and emergency room visits; and impair the body's immune system defenses, making people more susceptible to respiratory illnesses."[22]

Moreover, ozone is not an equal opportunity pollutant, striking hardest the most vulnerable segments of our population: children, the elderly, and people with respiratory ailments. (*Id.*) Children are at greater risk because their lung capacity is still developing, because they spend significantly more time outdoors than adults — especially in the summertime when ozone levels are the highest, and because

---

[20] *Kings County Farm Bureau v. City of Hanford*, 221 Cal.App.3d 692 (1990).
[21] *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1181 (D.C. Cir. 1981).
[22] 66 Fed. Reg. 5002, 5012 (Jan. 18, 2001).

they are generally engaged in relatively intense physical activity that causes them to breathe more ozone pollution. (*Id.*)

Ozone has severe impacts on millions of Americans with asthma. While it is as yet unclear whether smog actually causes asthma, there is no doubt that it exacerbates the condition.[23] Moreover, as EPA observes, the impacts of ozone on "asthmatics are of special concern particularly in light of the growing asthma problem in the United States and the increased rates of asthma-related mortality and hospitalizations, especially in children in general and black children in particular."[24] In fact:

> "[A]sthma is one of the most common and costly diseases in the United States. . . . Today, more than 5 percent of the US population has asthma [and] [o]n average *15 people died every day* from asthma in 1995. . .. In 1998, the cost of asthma to the U.S. economy was estimated to be $11.3 billion, with hospitalizations accounting for the largest single portion of the costs."[25]

The health and societal costs of asthma are wreaking havoc here in California. There are currently 2.2 million Californians suffering from asthma.[26] In 1997 alone, nearly 56,413 residents, including 16,705 children, required hospitalization because their asthma attacks were so severe. Shockingly, asthma is now the leading cause of hospital admissions of young children in California. *Id.* at 1. Combined with very real human suffering is the huge financial drain of asthma hospitalizations on the state's health care system. The most recent data indicate that the statewide financial cost of these hospitalizations was nearly $350,000,000, with nearly a third of the bill paid by the State Medi-Cal program. (*Id.* at 4.)

The Los Angeles air basin has the worst ozone problem in the nation. The EIR admits that the Project will increase emissions of NOx and VOCs which create ozone. The EIR must discuss how the project may impact the new more stringent

---

[23] *See* 66 Fed. Reg. 5002, 5012 (Jan. 18, 2001) (EPA points to "strong and convincing evidence that exposure to ozone is associated with exacerbation of asthma-related symptoms").

[24] 62 Fed. Reg. at 38864.

[25] 66 Fed. Reg. at 5012.

[26] *California Department of Health Services*, California County Asthma Hospitalization Chart Book, August 1, 2000.

ozone standard, and propose feasible mitigation measures to reduce ozone precursor emissions.

In short, in light of the regional nature of the ozone problem, the failure of the Los Angeles area to meet ozone standards, the public health threat presented by ozone pollution, and the already serious respiratory problems in the area, ozone is precisely the type of pollutant that must be analyzed for its cumulative and individually-significant impacts.[27] Thus, the City must prepare an SEIR for the Project to fully analyze, disclose to the public and consider mitigation measures to address this important public health problem.

III. THE DEIR FAILS TO ANALYZE AND MITIGATE ALL POTENTIALLY SIGNIFICANT IMPACTS

An EIR must disclose all potentially significant adverse environmental impacts of a project.[28] CEQA requires that an EIR must not only identify the impacts, but must also provide "information about how adverse the impacts will be."[29] The lead agency may deem a particular impact to be insignificant only if it produces rigorous analysis and concrete substantial evidence justifying the finding.[30] The DEIR for this Project fails to do so.

As explained by a recent CEQA decision:

> "The EIR must demonstrate that the significant environmental impacts of the proposed project were adequately investigated and discussed and it must permit the significant effects of the project to be considered in the full environmental context." (Guidelines, § 15125, subd. (c).) We interpret this Guideline broadly in order to "afford the fullest possible protection to the environment." (*Kings County Farm Bureau, supra*, 221 Cal. App. 3d 692, 720.) In so doing, we ensure that the EIR's analysis of significant effects, which is generated from this description of the environmental context, is as accurate as possible.

---

[27] *See, Kings County, supra.*
[28] Pub. Res. Code § 21100(b)(1). CEQA Guidelines section 15126(a); *Berkeley Jets,* 91 Cal. App. 4th 1344, 1354.
[29] *Santiago County Water Dist. v. County of Orange*, 118 Cal.App.3d 818, 831 (1981).
[30] *Kings County Farm Bureau v. City of Hanford*, 221 Cal.App.3d 692 (1990).

(See also Remy et al., Guide to the Cal. Environmental Quality Act (CEQA) (10th ed. 1999), pp. 374-376.)[31]

A. EIR Fails to Accurately Describe or Mitigate Traffic Impacts.

Registered Professional Engineer Tom Brohard explains that the EIR vastly underestimates traffic impacts that will be generated by the Project. Mr. Brohard uses up-to-date traffic models to conclude that the Project 25% more daily trips than calculated in the EIR.

Despite acknowledging significant traffic impacts at eleven intersections (page 3.11-25), the EIR later omits one of the intersections (Pasadena Ave./ Willow Street) entirely from its mitigation measure discussion. (Brohard Comments, p.8). The EIR concludes that the impacts at five of ten intersections would not be mitigated below the level of significance for the year 2014. (Brohard Comments, p. 8-9).

The EIR concludes that no feasible mitigation measures are available to mitigate significant traffic impacts at Atlantic Ave./Willow Street, Long Beach Blvd./Willow Street, or Long Beach Blvd./Wardlow Road. The EIR states, "No physical mitigation measure is feasible; any additional turn lanes would require widening and additional right of way." However, as Mr. Brohard explains, there is nothing inherently infeasible about the purchase of additional right of way or the creation of additional turn lanes, and such measures are often required to mitigate traffic impacts. (Brohard Comment, p. 9).

An SEIR must be prepared to properly analyze and disclose the Project's traffic impacts and to propose feasible mitigation measures.

B. EIR Fails to Accurately Describe or Mitigate Toxic Contamination Impacts.

As discussed above, the site of the proposed Project is heavily contaminated with toxic chemicals. Environmental expert John Williams and DTSC have raised significant concerns about the unknown extent of the contamination, the potential risks posed by the contamination, and the lack of any adequate mitigation plan.

---

[31] *Friends of the Eel River v. Sonoma County Water Agency*, (2003) 108 Cal. App. 4th 859, 874.

Mr. Williams points out that it is possible that methane and other hazardous gases may migrate into buildings. In fact, at least one other building in the complex was required to install a methane gas mitigation system for this very reason.

Among the toxic chemicals identified on the site are arsenic, lead, selenium, benzene, freon, xylene, ethylbenzene, toluene, methane, hydrogen sulfide and other VOCs. Many of these chemicals are known to be highly toxic to humans.

- Benzene has been identified by the state as a chemical known to cause cancer in humans, and has been linked strongly to leukemia.[32]
- Ethylbenzene can cause eye and throat irritation, dizziness and weakness.[33]
- Xylene can cause irritation to the eyes, nose and throat, impaired memory, and dizziness. Xylene can damage the liver, kidneys, lungs, heart and nervous system, and can damage fetuses if pregnant women are exposed.[34]
- Lead has been identified by the State of California as a chemical known to cause cancer and reproductive toxicity in humans.[35] According to the United States Environmental Protection Agency (US EPA), lead can cause brain damage, learning deficits, hearing problems, headaches, difficulties during pregnancy, high blood pressure, memory and concentration problems, and muscle and joint pain.[36] Reduced IQ is one of the most common effects of lead poisoning in children. Each three microgram increase in lead poisoning has been found to result in a one-point drop in IQ.[37] Adults can be exposed to lead in soil through gardening or other outdoor activities, but children are at much greater risk of lead poisoning due to the fact that they often place their hands, yard toys, soil, and other objects into their mouths.[38]

---

[32] Proposition 65 Status Report, Exhibit 2; ATSDR, Public Health Statement for Benzene, Exhibit 3.
[33] ATSDR, Public Health Statement for Ethylbenzene, Exhibit 4.
[34] ATSDR, Public Health Statement for Xylene, Exhibit 5.
[35] Proposition 65 Status Report, Exhibit 2.
[36] US EPA Lead Fact Sheet, Exhibits 6 and 7.
[37] Lead Health Effects and Sources of Exposure, Exhibit 8.
[38] Exhibits 6-8.

- Arsenic is known to cause lung cancer, bladder cancer, skin lesions, and other ailments.[39]

DTSC submitted comments on the project, concluding that the EIR "did not provide sufficient description of the extent and nature of contamination existing at the site, or analysis of the potential impacts associated with potential RAW [remedial action workplan] activities. This is primarily due to the fact that information related to the extent and nature of the contamination is still being acquired and evaluated for the development of a draft RAW." (DTSC Comment, p. 2 (March 16, 2005)). DTSC also concludes that that "the specific impacts and mitigation measures associated with the removal/remediation of contaminated media that may be encountered during construction have not been outlined." (Id.) Since the site has not been adequately characterized, it is unclear whether the site exceeds applicable clean-up standards, and if so, by how much.[40]

Finally, and most significantly, DTSC states that "elements of the clean-up requiring mitigation including, but not limited to, soil excavation, onsite storage, off-site transportation, and backfill need to be adequately addressed. ***The actions that will be outlined in the draft RAW for the Project must be evaluated and incorporated in the final version of the EIR***." (Id.). DTSC also states that "specific impacts associated with the removal of contaminated soil, and corresponding mitigation measures ***must be outlined in the final EIR***." (Id. at p.3). ***However, the final EIR did not evaluate, incorporate, or even describe such remedial activities***.

Despite the extensive contamination, and clear routes of exposure to hospital workers, patients, construction workers and others, the EIR presents absolutely no mitigation proposal. Risks may be particularly pronounced given the certain presence of children on the site due to the children's hospital.

Instead of proposing mitigation, the EIR states that the toxic contamination will be mitigated in the future pursuant to a plan that will be developed by various agencies including the DTSC, the Long Beach Health Department and the South Coast Air Quality Management District. (Mitigation measures 1-15, pp. 3.5-14 – 3.5-17).

---

[39] Univ. of Calif. Berkeley, Program in Arsenic Health Effects Research, Exhibit 9.
[40] Calif. Regional Water Quality Control Board, Screening for Environmental Concerns at Sites with Contaminated Soil and Groundwater. Exhibit 10.

CEQA prohibits deferring the formulation of mitigation measures to post-approval studies.[41] An agency may only defer the formulation of mitigation measures when it possesses "'meaningful information' reasonably justifying an expectation of compliance."[42] A lead agency is precluded from making the required CEQA findings unless the record shows that all uncertainties regarding the mitigation of impacts have been resolved; an agency may not rely on mitigation measures of uncertain efficacy or feasibility.[43] This approach helps "insure the integrity of the process of decisionmaking by ***precluding stubborn problems or serious criticism from being swept under the rug***."[44]

Moreover, by deferring the development of specific mitigation measures, the Applicant has effectively precluded public input into the development of those measures. CEQA prohibits this approach. As explained by the *Sundstrom* court:

> An EIR ... [is] subject to review by the public and interested agencies. This requirement of "public and agency review" has been called "the strongest assurance of the adequacy of the EIR." The final EIR must respond with specificity to the "significant environmental points raised in the review and consultation process." . . . Here, the hydrological studies envisioned by the use permit would be exempt from this process of public and governmental scrutiny.[45]

The EIR suffers from the same fatal flaw. The EIR recognizes significant toxic chemical-related impacts, but fails to describe the scope or severity of those impacts, and fails to identify any specific mitigation measures to protect public health and the environment. By proposing that mitigation for this very significant

---

[41] CEQA Guidelines § 15126.4(a)(1)(B); *Sundstrom v. County of Mendocino* (1988) 202 Cal.App.3d 296, 308-309.
[42] *Sundstrom* at 308; *see also Sacramento Old City Association v. City Council of Sacramento* (1991) 229 Cal.App.3d 1011, 1028-29 (mitigation measures may be deferred only "for kinds of impacts for which mitigation is known to be feasible").
[43] *Kings County Farm Bureau v. City of Hanford* (1990) 221 Cal.App.3d 692, 727 (finding groundwater purchase agreement inadequate mitigation because there was no evidence that replacement water was available).
[44] *Concerned Citizens of Costa Mesa, Inc. v. 32nd Dist. Agricultural Assn.* (1986) 42 Cal.3d 929, 935.
[45] *Sundstrom*, 202 Cal.App.3d at 308.

impact be deferred until after the close of the CEQA process, the City is sweeping a very stubborn problem "under the rug" in violation of CEQA.

Also, by proposing that mitigation measures be developed by other agencies, (DTSC, the Long Beach Health Department and the South Coast Air Quality Management District), the City is abdicating its responsibility as CEQA lead agency. As CEQA lead agency, the City has a duty to ensure that all impacts are fully analyzed and mitigated, and the City may not pass this responsibility onto another agency.[46]

An SEIR is required to analyze significant toxic contamination impacts, and to propose mitigation measure. The SEIR must be circulated for full public review so that the public may review concrete mitigation measures to determine their adequacy.[47] As a leading CEQA treatise explains, "in *Perley v. Board of Supervisors* (1982) 137 Cal.App.3d 424, the court held that the public has a right to review a project described in a [CEQA document] in its final form and suggested that a [CEQA document] must be recirculated if mitigation measures are added."[48]

C. EIR Fails to Adequately Describe or Mitigate Significant Air Quality Impacts from Project Construction.

The EIR admits that the Project will have significant operational and construction air quality impacts. The EIR admits that construction emissions will exceed applicable significance thresholds for carbon monoxide (CO), nitrogen oxides (NOx), and reactive organic compounds (ROGs, also known as VOCs). (EIR, p. 3.2-11). The EIR also admits that the Project's operational emissions will combine with these construction emissions in 2010 to create cumulatively significant air impacts for CO, NOx and ROGs. (Id. p. 3.2-12). The EIR also admits that the Project's operational impacts a build-out will be significant for NOx and ROGs. (Id., p. 3.2-15).

---

[46] *Planning and Conservation League v. Dept. of Water Resources* (2000) 83 Cal.App.4th 892, 903; *Eller Media v. Community Redevel. Agency* (2003) 108 Cal.App.4th 25, 38.

[47] *Gentry v. City of Murrieta* (1995) 36 Cal.App.4th 1359, 1391-2, 1411, 1417.

[48] Kostka & Zischke, *Practice Under the Calif. Environ. Quality Act*, at §7.19.

Despite these admissions of significant air quality impacts, the EIR fails to require implementation of all feasible mitigation measures, and admits that the Project's air quality impacts will remain significant even after implementation of all mitigation measures set forth in the EIR. (Id., p. 3.2-20). While the EIR includes several construction emission mitigation measures, the list fails to include many feasible measures that are routinely required by other agencies.

The EIR includes almost no mitigation required for operational emissions other than to "encourage" carpooling and the use of public transportation. The EIR is silent on how the "encouragement" will be enforced or executed. Possible operational emission mitigations could include shuttle service to public transit stations, use of energy efficient windows, insulations and appliances, preferential parking for hybrid and low-emission vehicles, and other measures. The EIR considers none of these. An SEIR must be prepared to propose and require implementation of additional feasible mitigation measures.

1. EIR Fails to Include All Feasible Measures to Reduce Construction Particulate Emissions.

The EIR fails to consider numerous feasible measures to reduce construction emissions. For example the Bay Area Air Quality Management District (BAAQMD) suggests the following construction mitigations:

- Install wheel washers for all exiting trucks, or wash off the tires or tracks of all trucks and equipment leaving the site.
- Install wind- breaks, or plant trees/vegetative wind breaks at windward side(s) of construction areas.
- Suspend excavation and grading activity when winds (instantaneous gusts) exceed 25 mph.
- Limit the area subject to excavation, grading and other construction activity at any one time.

The EIR requires some but not all of these measures. They are all feasible, and CEQA requires their implementation. (BAAQMD CEQA Guidelines 1999 p. 15)

In addition, there are numerous additional relevant and reasonable measures contained in the CEQA guidelines and rules of air districts and other agencies that

should be required for this Project. Further, several agencies have conducted comprehensive studies of fugitive dust control measures to bring their region into compliance with federal ambient air quality standards on PM10.

The South Coast Air Quality Management District ("SCAQMD") has sponsored research, passed regulations (*e.g.*, Rule 403),[49] and published guidelines that identify best management practices for controlling fugitive dusts at construction sites. The *Rule 403 Implementation Handbook*[50] contains a list of such measures. Some of the feasible mitigation measures identified by the SCAQMD and other agencies include:

- For backfilling during earthmoving operations, water backfill material or apply dust palliative to maintain material moisture or to form crust when not actively handling; cover or enclose backfill material when not actively handling; mix backfill soil with water prior to moving; dedicate water truck or large hose to backfilling equipment and apply water as needed; water to form crust on soil immediately following backfilling; and empty loader bucket slowly; minimize drop height from loader bucket. (CCHD)[51]

- During clearing and grubbing, pre-wet surface soils where equipment will be operated; for areas without continuing construction, maintain live perennial vegetation and desert pavement; stabilize surface soil with dust palliative unless immediate construction is to continue; and use water or dust palliative to form crust on soil immediately following clearing/grubbing. (CCHD)

- While clearing forms, use single stage pours where allowed; use water spray to clear forms; use sweeping and water spray to clear forms; use

---

[49] South Coast Air Quality Management District ("SCAQMD"), *Revised Final Staff Report for Proposed Amended Rule 403—Fugitive Dust and Proposed Rule 1186—PM10 Emissions from Paved and Unpaved Roads, and Livestock Operations,* February 14, 1997.

[50] South Coast Air Quality Management District ("SCAQMD"), *Rule 403 Implementation Handbook,* January 1999.

[51] The following acronyms are used in this listing of mitigation measures: ADEQ = Arizona Department of Environmental Quality; BCAQMD = Butte County Air Quality Management District; CCHD = Clark County (Nevada) Health Department; MBUAPCD = Monterey Bay Unified Air Pollution Control District; SBCAPCD = Santa Barbara County Air Pollution Control District; SJVUAPCD = San Joaquin Valley Unified Air Pollution Control District; SLOCAPCD = San Luis Obispo County Air Pollution Control District.

industrial shop vacuum to clear forms; and avoid use of high pressure air to blow soil and debris from the form. (CCHD)

- During cut and fill activities, pre-water with sprinklers or wobblers to allow time for penetration; pre-water with water trucks or water pulls to allow time for penetration; dig a test hole to depth of cut to determine if soils are moist at depth and continue to pre-water if not moist to depth of cut; use water truck/pull to water soils to depth of cut prior to subsequent cuts; and apply water or dust palliative to form crust on soil following fill and compaction. (CCHD)

- For large tracts of disturbed land, prevent access by fencing, ditches, vegetation, berms, or other barrier; install perimeter wind barriers 3 to 5 feet high with low porosity; plant perimeter vegetation early; and for long-term stabilization, stabilize disturbed soil with dust palliative or vegetation or pave or apply surface rock. (CCHD)

- In staging areas, limit size of area; apply water to surface soils where support equipment and vehicles are operated; limit vehicle speeds to 15 mph; and limit ingress and egress points. (CCHD)

- For stockpiles, maintain at optimum moisture content; remove material from downwind side; avoid steep sides or faces; and stabilize material following stockpile-related activity. (CCHD)

- To prevent track-out, pave construction roadways as early as possible; install gravel pads; install wheel shakers or wheel washers, and limit site access. (CCHD)

- When materials are transported off-site, all material shall be covered, effectively wetted to limit visible dust emissions, or at least six inches of freeboard space from the top of the container shall be maintained. (BAAQMD, SJVUAPCD, Rule 403 Handbook, ADEQ)

- Where feasible, use bed-liners in bottom-dumping haul vehicles. (Rule 403 Handbook)

- Grade each phase separately, timed to coincide with construction phase or grade entire project, but apply chemical stabilizers or ground cover to

graded areas where construction phase begins more than 60 days after grading phase ends. (Rule 403 Handbook)

- All operations shall limit or expeditiously remove the accumulation of mud or dirt from adjacent public streets at least once every 24 hours when operations are occurring. (BAAQMD) *(The use of dry rotary brushes is expressly prohibited except where preceded or accompanied by sufficient wetting to limit the visible dust emissions.) (Use of blower devices is expressly forbidden.)* (SJVUAPCD)

- Following the addition of materials to, or the removal of materials from, the surface of outdoor storage piles, said piles shall be effectively stabilized of fugitive dust emissions utilizing sufficient water or chemical stabilizer/suppressant. (SJVUAPCD, ADEQ)

- During initial grading, earth moving, or site preparation, projects 5 acres or greater may be required to construct a paved (or dust palliative treated) apron, at least 100 ft in length, onto the project site from the adjacent site if applicable. (BCAQMD)

- Post a publicly visible sign with the telephone number and person to contact regarding dust complaints. This person shall respond and take corrective action within 24 hrs. (BCAQMD, MBUAPCD, CCHD)

- Prior to final occupancy, the applicant demonstrates that all ground surfaces are covered or treated sufficiently to minimize fugitive dust emissions. (BCAQMD)

- Gravel pads must be installed at all access points to prevent tracking of mud on to public roads. (SBCAPCD)

- The contractor or builder shall designate a person or persons to monitor the dust control program and to order increased watering, as necessary, to prevent transport of dust offsite. (SBCAPCD, SLOCAPCD)

- Prior to land use clearance, the applicant shall include, as a note on a separate informational sheet to be recorded with map, these dust control requirements. All requirements shall be shown on grading and building plans. (SBCAPCD, SLOCAPCD)

- All roadways, driveways, sidewalks, etc. to be paved should be completed as soon as possible. In addition, building pads should be laid as soon as possible after grading unless seeding or soil binders are used. (SLOCAPCD)

- Barriers with 50% or less porosity located adjacent to roadways to reduce windblown material leaving a site. (Rule 403 Handbook)

- Limit fugitive dust sources to 20% opacity. (ADEQ)

- Require a dust control plan for earthmoving operations. (ADEQ)

All of these measures are feasible and various combinations of them are routinely required elsewhere to reduce fugitive PM10 emissions. See the fugitive dust control program for the Big Dig (Kasprak and Stakutis 2000[52]), for the El Toro Reuse Draft EIR[53], and for the Padres Ballpark Final EIR.[54]

The EIR requires implementation of some, but not all of these measures. They are all feasible, and so must all be required under CEQA. The City must prepare a SEIR that includes all the above feasible measures to mitigate the significant adverse impact caused by fugitive PM10 pollution.

---

[52] A. Kasprak and P.A. Stakutis, A Comprehensive Air Quality Control Program for a Large Roadway Tunnel Project, *Proceedings of the Air & Waste Management Association's 93rd Annual Conference 7 Exhibition*, June 18-22, 2000.

[53] County of Orange, *Draft Environmental Impact Report No. 573 for the Civilian Reuse of MCAS El Toro and the Airport System Master Plan for John Wayne Airport and Proposed Orange County International Airport, Draft Supplemental Analysis*, Volume 1, April 2001, pp. 2-121 to 2-123.

[54] City of San Diego, *Final Subsequent Environmental Impact Report to the Final Master Environmental Impact Report for the Centre City Redevelopment Project and Addressing the Centre City Community Plan and Related Documents for the Proposed Ballpark and Ancillary Development Projects, and Associated Plan Amendments*, V. IV. Responses to Comments, September 13, 1999, pp. IV-254 to IV-256.

2. EIR Fails to Include All Feasible Measures to Reduce Construction Diesel Emissions.

The EIR fails to include any measures to reduce diesel emissions during construction. Many feasible measures are available, and would reduce NOx, sulfur, and particulate emissions. BAAQMD's CEQA Guidelines recommend the following measures to reduce diesel exhaust:

- Use of alternative fueled construction equipment
- Minimizing idling time
- Maintaining properly tuned equipment
- Limiting the hours of operation of heavy duty equipment and/or the amount of equipment in use

Further, the BAAQMD guidelines recommend that "[if] a project may result in public exposure to high levels of diesel exhaust, the Lead Agency should propose mitigation measures to reduce this impact" and recommend the following measures for construction equipment (*Id.*, p. 60.):

- Conversion to cleaner engines
- Use of cleaner (reduced sulfur) fuel
- Regular maintenance – keep equipment well tuned
- Add-on control devices, *e.g.*, particulate traps, catalytic oxidizers
- Buffer zone between facility and sensitive receptors

In addition, other feasible measures to reduce diesel emissions include:

- Requiring Aqueous Diesel Fuels
- Requiring Diesel Particulate Filters
- Requiring Cooled Exhaust Gas Recirculation (EGR)
- Requiring ultra low sulfur diesel
- Requiring the use of electric-powered equipment where possible
- Requiring alternative diesel formulations
- Requiring California Air Resources Board ("CARB")-certified construction equipment
- Requiring post-combustion controls

These measures are unquestionably feasible, and should be required. An SEIR should be prepared to analyze and implement such measures.

IV. EIR FAILS TO DESCRIBE OR MITIGATE THE PROJECT'S CUMULATIVE IMPACTS.

An EIR must discuss significant "cumulative impacts." CEQA Guidelines § 15130(a). This requirement flows from CEQA Section 21083, which requires a finding that a project may have a significant effect on the environment if,

> the possible effects of a project are individually limited but cumulatively considerable. . . . 'Cumulatively considerable' means that the incremental effects of an individual project are considerable when viewed in connection with the effects of past projects, the effects of other current projects, and the effects of probable future projects.

Public Resources Code § 21083.

As the court stated in *Communities for a Better Environment v. Cal. Resources Agency* ("*CBE v. CRA*")[55]:

> Cumulative impact analysis is necessary because the full environmental impact of a proposed project cannot be gauged in a vacuum. One of the most important environmental lessons that has been learned is that environmental damage often occurs incrementally from a variety of small sources. These sources appear insignificant when considered individually, but assume threatening dimensions when considered collectively with other sources with which they interact.

Cumulative impacts are defined as "two or more individual effects which, when considered together, are considerable or which compound or increase other environmental impacts." CEQA Guidelines § 15355(a). "[I]ndividual effects may be changes resulting from a single project or a number of separate projects." *Id.*

As set forth by the court in *CBE v. CRA,* 103 Cal.App.4th at 117:

> The cumulative impact from several projects is the change in the environment which results from the incremental impact of the project

[55] (2002) 103 Cal.App.4th 98, 114

> when added to other closely related past, present, and reasonably foreseeable probable future projects. Cumulative impacts can result from individually minor but collectively significant projects taking place over a period of time.

A legally adequate "cumulative impacts analysis" views a particular project over time and in conjunction with other related past, present, and reasonably foreseeable probable future projects whose impacts might compound or interrelate with those of the project at hand. "Cumulative impacts can result from individually minor but collectively significant projects taking place over a period of time." CEQA Guidelines § 15355(b).

To comply with CEQA, an EIR must contain either "a list of past, present, and probable future projects producing related or cumulative impacts, including, if necessary, those projects outside the control of the agency," or "a summary of projections contained in an adopted general plan or related planning document, or in a prior environmental document which has been adopted or certified, which described or evaluated regional or areawide conditions contributing to the cumulative impact."[56]

Here, the EIR violates CEQA by failing to provide any cumulative impact analysis at all for most subject areas, including air quality, aesthetics, geology, hazardous materials, land use planning and public services. However, the EIR admits that there are significant environmental impacts from air pollution, hazardous materials, and impacts to fire protection services. Instead of analyzing these and other potential environmental impacts, the EIR provides conclusory statements that there will be no cumulative impacts, contradicting its conclusions that there will be significant impacts, impermissibly limits the geographic scope of the cumulative impacts, and impermissibly relying on planning documents.

---

[56] CEQA Guidelines § 15130(b)(1); *San Joaquin Raptor/Wildlife Rescue Ctr. v. County of Stanislaus* (1994) 27 Cal.App.4th 713, 740.

A. The Cumulative Impacts Analyses Are Impermissibly Conclusory, Contradictory, and Incomplete

Mere conclusory statements are not sufficient to satisfy the cumulative impacts analysis requirement.[57] A proper cumulative impact analysis must be supported by references to specific evidence. *Id.* As the Court in *Mountain Lion Coalition* explained, "it is vitally important that an EIR avoid minimizing the cumulative impacts. Rather, it must reflect a conscientious effort to provide public agencies and the general public with adequate and relevant detailed information about them." *Id.* at 1051. "A cumulative impacts analysis which understates information concerning the severity and significance of cumulative impacts impedes meaningful public discussion and skews the decisionmaker's perspective concerning the environmental consequences of the project, the necessity for mitigation measures, and the appropriateness of project approval." *Id.*

This EIR fails to support its conclusions with any evidence that there will be no cumulative impacts for almost every category of impact analyzed.

- **Air Quality**

The EIR clearly states that "proposed project would be anticipated to have significant impacts to air quality during operations due to the exceedance of the SCACMD significance threshold for NOx." (EIR at 3.2-13). However, the City then makes the contradictory claim that the project would not have significant cumulative air impacts because "the operational emissions from the proposed project are individually insignificant." (*Id.* at 3.2-16). The City, however, admits that the project's air emissions would be significant, leading to the conclusion that the cumulative impacts will also be significant. The City cannot now 'unring that bell.'[58]

Furthermore, the air quality cumulative impacts analysis is deficient because it fails to provide the necessary quantitative analysis, impermissibly limits the geographic scope considered and impermissably relies on planning documents to obviate the proper study of the cumulative air quality impacts. These issues are addressed in Section B below.

---

[57] *Mountain Lion Coalition v. Fish & Game Comm'n* (1989) 214 Cal.App.3d 1043, 1047.
[58] *Stanislaus Audubon v. Stanislaus* (1995) 33 Cal.App.4th 144, 154.

• **Aesthetics**

The EIR makes the bald conclusion that "due to the vicinity of the other development projects to the proposed project area, the proposed project would not result in cumulative impacts." (EIR at 3.1-8). However, the EIR does not provide any evidence, analysis or detail to substantiate this conclusion.

• **Geology and Soils**

EIR makes the bald conclusion that "[b]ecause the geology and soils impacts expected from the implementation of the proposed project do not affect lands outside the boundaries of the proposed project site, these impacts do not create any cumulative impacts on the environment outside of the proposed project boundaries." (*Id.* 3.4-15). However, the EIR does not provide any evidence, analysis or detail to substantiate this conclusion. Furthermore, while it may be true that no cumulative impacts will result "outside of the proposed project boundaries," the EIR failed to consider if there may be any cumulative impacts within the project boundaries as a result of this project. (*Id.*).

• **Hazardous Materials**

The EIR first admits that the Project may have significant environmental impacts: "the proposed project has the potential to result in significant impacts to the public or the environment related to the routine transport, use, or disposal of hazardous materials," (*Id.* at 3.5-9), and that "[o]ff-site transport and disposal routes for biomedical, radiological, hazardous, and nonhazardous may include the route . . . within 0.25 miles of the [Jackie Robinson Elementary] school." (*Id.* 3.5-11). The cumulative impacts analysis, however, contradicts this conclusion two pages later the bald conclusion that "[b]ecause the hazards and hazardous materials impacts expected from the implementation of the proposed project do no affect lands outside the boundaries of the proposed project site, these impacts do not create any cumulative impacts on the environment outside the proposed project boundaries." (*Id.* 3.5-13). Here the EIR not only fails to substantiate its conclusion that there will be no cumulative impacts, but it contradicts its own conclusion that there may be significant off-site impacts. Furthermore, the EIR fails to even consider any on-site cumulative impacts that may result from the use, transport and disposal of hazardous materials.

- **Land Use Planning**

The EIR makes the bald conclusion that the Project "would not cause significant impact to land use planning" because "[a]ll of the related projects occur outside of the Campus." (*Id.* at 3.7-8). However, the EIR does not provide any evidence, analysis or detail to substantiate this conclusion. Furthermore, the EIR explains that the Project will require a zoning amendment that "anticipates the likely increased future demand for expansion in the capacity of the region's medical service facilities." (*Id.* at 3.7-7). By its terms, this zoning amendment anticipates reasonably foreseeable probable future projects whose impacts might compound or interrelate with those of the project at hand. This EIR violates CEQA by failing to consider these anticipated future impacts.

- **Public Services**

The EIR draws the conclusion that there will be no cumulative impacts in part because the "proposed project would not require the provision of, or need for, new or physically altered fire protection." (*Id.* 3.10-8). However, the EIR does not provide any evidence, analysis or detail to substantiate this conclusion. In fact, the EIR stated two pages previously that the "proposed project would have a significant effect on fire protection and would require mitigation." (*Id.* 3.10-6). Thus, cannot claim the project to have no cumulative impacts on public services when its has already admitted the opposite. The City cannot now 'unring that bell.'[59]

B. Cumulative Air Quality Impacts From This Project Are Significant

As discussed above, this EIR admits that project operations will create significant impacts to air quality. (EIR at 3.2-13). Thus, the conclusion that there will be no cumulative impacts is incomprehensible. (*Id.* at 3.2-16).

The cumulative air quality impacts analysis is also deficient because it fails to provide the necessary quanitative analysis, imperssiably limits the geographic scope considered and impermissably relies on planning documents to obviate the proper study of the cumulative air quality impacts.

---

[59] *Stanislaus Audubon v. Stanislaus* (1995) 33 Cal.App.4th 144, 154.

1. The Air Quality Cumulative Impacts Analysis Lacks the Required Detail and Analytical Analysis.

The Air Quality Cumulative Impacts analysis is sorely deficient. The EIR merely contains one conclusory paragraph, which incorrectly concludes that there will be no cumulative air quality impacts. (EIR at 3.2-16). When conducting a cumulative impacts analysis, the EIR must consider past, present and reasonably future impacts.

An EIR must include objective measurements of a cumulative impact when such data are reasonably available or can reasonably be produced by further study, and is necessary to ensure disclosure of the impact.[60] It is impossible to evaluate the air quality impacts unless the EIR analyses and considers the data of other projects that must be considered. *Id.*

Here, the cumulative impact analysis contains no data whatsoever of other past, present, or reasonably future projects that may contribute to the cumulative air impacts. Simply referencing a list of other projects, without providing data and/or analysis explaining what type and magnitude of impact those projects may have is not an adequate cumulative impacts analysis.

2. The Air Quality Cumulative Impacts Analysis Impermissably Limits the Geographic Scope

In its air quality impacts analysis, the EIR considers forty-three related projects. (EIR Figure 2.6-1). Although the air quality cumulative impacts analysis fails to even mention a single other project in the vicinity, the conclusion that there are no cumulative air impacts implicitly considers these "related projects." Considering only these local projects, not more than approximately two miles from the Project location, impermissibly limits the geographic scope of the cumulative impacts analysis.

The courts have held that cumulative impacts analyses for air quality impacts must consider projects from the entire air basin.[61] The recent *Bakersfield Citizens* case demonstrates why the City has improperly limited the geographic

---

[60] *Kings Country Farm Bureau* (1990) 221 Cal.App.3d 692, 729.
[61] *Kings Country Farm Bureau*, 221 Cal.App.3dv692, 723.

scope.[62] In *Bakersfield Citizens*, two separate parties were each developing unrelated retail shopping centers 3.6 miles from one another.[63] Each shopping center failed to consider the cumulative impacts of the other shopping center.[64] The Court found that both EIRs were inadequate because the lead agency failed to properly define the geographic scope according to CEQA Guidelines Section 15130(b)(1)(B)(3).[65] The Court explained that "inaccurate minimization of the cumulative impacts on air quality" undermined the need for "[p]roper cumulative impacts analysis [as] absolutely critical to meaningful environmental review."[66]

The City of Long Beach cannot limit its cumulative impacts analysis to a few projects merely two miles away. It must consider other projects in the air basin that stand to have cumulative effects with this Project.

Furthermore, the South Coast Air Quality Management District (SCAQMD) has already provided its view of the geographic scope for cumulative impact analysis of projects in this area when it prepared its Paramount Refinery Clean Fuels Project EIR. (Attached as Exhibit 11). The Paramount EIR considered many projects up to 18 miles away, including two Long Beach projects – the City of Long reach Streetscape Improvements and the North Long Beach Redevelopment. (Paramount EIR, Figure 5-2, p. 5-4). For this Project EIR, however, the City failed to consider Paramount's emissions, or the emissions of any of the other facilities in the same vicinity.

The City is legally required to consider the cumulative impacts of other projects identified in the EIR, and the other projects identified in the Paramount Refinery EIR. All of those projects are in the same air basin, and that they all contribute to the same cumulative air pollution. If, as set forth in the Paramount Refinery EIR, Projects in Long Beach contribute to the cumulative emissions of the Paramount Refinery, then the Paramount Refinery and other projects described in SCAQMD's EIR for that refinery must contribute to the cumulative emissions of this Project.

---

[62] *Bakersfield Citizens v. City of Bakersfield* (2004) 124 Cal.App.4th 1184
[63] 124 Cal. App. 4th at 1184.
[64] *Id.* at 1193.
[65] *Id.*
[66] *Id.* (citing *Kings Country Farm Bureau*, 221 Cal.App.3d 692).

In the table below, we add the Project's air emissions as set forth in the EIR to the cumulative emissions set forth in the Paramount EIR. It is clear that the Project's cumulative emissions are significant for every pollutant.

Table 1
Cumulative Operational Emissions
Modified Based on Responses to Comments
(lbs/day)

SOURCE	CO	VOC	NOx	SOx	PM10
Ultramar CARB Phase 3 Project	514	156	2,164	2,678	287
ConocoPhillips Ethanol Import & Dist. Project	9	-54(1)	10	--	1
ConocoPhillips CARB RFG Phase 3	136	22	514	402	43
BP ARCO CARB Phase 3 Project	42	86	49	0	57
Shell CARB Phase 3 Project	2,213	482	2030	71	57
ExxonMobil CARB Phase 3 Project	29	288	138	12	103
ChevronTexaco CARB Phase 3 Project	393	347	3,103	2,498	843
Third Party Terminals	-	4	-	-	-
Paramount Clean Fuels Project	104	66	52	1	69
Industrial Warehouse Project (No. 10)(2)	76	7	10	<1	5
Recreational Center Project (No. 11)(2)	39	3	5	<1	3
Banco Popular Project (No. 13)(2)	109	9	14	<1	8
Residential Development (No. 14 and 15)(2)	80	25	5	<1	10
Long Beach Memorial	286	25.8	64	3.38	65
Cumulative Emissions	**4030**	**1,468**	**8,158**	**5,665**	**1,551**
SCAQMD Thresholds	**500**	**55**	**55**	**150**	**150**
Significant (?)	**YES**	**YES**	**YES**	**YES**	**YES**

(1) Negative numbers represent emission reductions.
(2) Based on URBEMIS2002 Model, using default assumptions.

Table 1 indicates that cumulative emissions of all criteria pollutants exceed the SCAQMD's emission significance thresholds (in bold). The EIR did not disclose that any emissions were cumulatively significant. These are new significant impacts that must be mitigated. An SEIR should be prepared to evaluate and mitigate these significant impacts.

3. The EIR Impermissibly relies on Planning Documents to Avoid a Valid Cumulative Impacts Analysis.

Relying on planning documents to avoid preparing a cumulative impacts analysis in an EIR does not satisfy CEQA's cumulative impact analysis requirement if summary projections from the planning document are inaccurate, outdated, or insufficient.[67] Reliance on planning document is also improper when the proposed project requires amendments to the plan that are not taken into account by the general plan EIR's cumulative impacts analysis. *Id.*

Here, the EIR simply states that because the project is consistent with land use plans and zoning, no cumulative impacts analysis are required. (EIR at 3.2-16). As stated in *Bakersfield*, this is inadequate without at the very least showing a summary of the data leading to this conclusion.

Additionally, the EIR states that land use zoning amendments will be necessary for this project. Thus, the EIR cannot rely on these planning documents and current zoning rules.

4. The City's Reliance on Air Quality Management Plan is Misplaced

The City claims that it does not need to conduct a cumulative impacts analysis for this project because the project complies with the Air Quality Management Plan (AQMP).

Reliance on the 2003 AQMP is misplaced, however. CEQA Guidelines Section 15064(h)(3) allows an agency to forgo cumulative analysis only when a plan addresses the cumulative problem with a mitigation program that contains "*specific requirements* that will avoid or substantially lessen the cumulative problem ... *within the geographic area in which the project is located.*" Here, the City fails to show any evidence that the AQMP satisfies this requirement.

---

[67] *Bakersfield Citizens v. City of Bakersfield* (2004) 124 Cal.App.4th 1184, 1217.

V. CONCLUSION.

The Project will have numerous highly significant impacts that are neither disclosed, analyzed, nor mitigated in the EIR. We urge the City to prepare an SEIR that fully complies with CEQA prior to approving the Project or certifying the EIR. Thank you for considering our comments.

Sincerely,

Richard Toshiyuki Drury

RTD:bh
Attachments

California Environmental Protection Agency

NEWS RELEASE

Air Resources Board

Release 05-10

FOR IMMEDIATE RELEASE
April 28, 2005

CONTACT: Jerry Martin
Gennet Paauwe Powell
(916) 322-2990
www.arb.ca.gov

California Adopts New Ozone Standard
Children's Health Focus of New Requirement

EL MONTE, CALIF. -- Today the California Air Resources Board (ARB) approved the nation's most health protective ozone standard with special consideration for children's health. The new 8-hour-average standard at 0.070 parts per million (ppm) will further protect California's most vulnerable population from the adverse health effects associated with ground-level ozone, or smog. The new 8-hour-average ozone standard is the first of its kind in the state.

"It is clear that children who grow up under smoggy skies have greater health problems than those who breathe clean air," said ARB Chairman Barbara Riordan. "California has a longstanding record of adopting the world's cleanest air quality standards and today's action continues our leadership in protecting public health."

The Children's Environmental Health Protection Act, passed in 1999, requires the ARB, in consultation with the Office of Environmental Health Hazard Assessment, to "review all existing health-based ambient air quality standards to determine whether these standards protect public health, including infants and children, with an adequate margin of safety." As a result of that requirement, the ARB today adopted the new ozone standard:

- A new 8-hour-average standard for ozone is established at 0.070 ppm, not to be exceeded.
- The 1-hour-average ozone standard is retained at 0.09 ppm, not to be exceeded.

Ozone, also known as urban smog, can affect human health in many ways including: itchy, watery eyes, scratchy throat, difficulty breathing, shortness of breath, coughs, heightened asthma rates, cardiopulmonary cases and premature deaths. Research has also shown that ozone is associated with increased hospital visits, emergency room admissions, student and worker absences, activity restrictions and premature death. ARB research has shown that ozone is associated with new cases of asthma.

Children are a particularly vulnerable population because their increased exposure to ozone can affect lung function. ARB research has also shown that children spend more time outside, are more active and breathe at a higher rate relative to their size than do adults.

Photochemical reactions between oxides of nitrogen (NOx) and volatile organic compounds (VOCs) form unhealthy ground-level ozone. California's geography and climate help with the creation of ozone because of its warm, sunny

days and mountains that trap air pollution.

The new standards amount to new clean air goals for the state and set the state's definition of healthy air. The standards will go into effect late this year or early next year, after going through California's review process for new regulations.

For further information, click here.

The Air Resources Board is a department of the California Environmental Protection Agency. ARB's mission is to promote and protect public health, welfare, and ecological resources through effective reduction of air pollutants while recognizing and considering effects on the economy. The ARB oversees all air pollution control efforts in California to attain and maintain health based air quality standards.

The energy challenge facing California is real. Every Californian needs to take immediate action to reduce energy consumption. For a list of simple ways you can reduce demand and cut your energy cost, see our web site at http://www.arb.ca.gov

#####

CONTRA COSTA TIMES

Posted on Fri, Apr. 29, 2005

California approves new ozone-level limits

By Gillian Flaccus
ASSOCIATED PRESS

LOS ANGELES - The state Air Resources Board unanimously adopted a new limit on ozone levels Thursday that gives California the toughest guidelines in the nation -- a standard that critics argue is largely symbolic.

Supporters estimate that, if fully effective, the new standard could save Californians millions of dollars each year in medical costs and productivity losses linked to smog-induced illness.

They insist that while it may take years for the state to meet the new standard, its existence will force individual air quality districts to implement long-term strategies to reduce pollution.

"It's definitely a goal that the air district will strive for," said Luna Salaver, spokeswoman for the Bay Area Air Quality Management District.

The majority of the state currently doesn't meet the less-stringent federal standard.

The eight-member board met for nearly 21/2 hours before approving the new limit. California has no authority to impose sanctions for violations of the rules.

Several board members said they supported the change but expressed concerns about the as-yet-unknown cost of implementing it statewide.

Janie Holmes-Gen, spokeswoman for the American Lung Association of California, said the new ozone standard is based on the latest research.

New evidence suggests pollution can cause a host of illnesses -- heart and lung disease, asthma, premature death -- and can exacerbate the symptoms of diabetes, she said.

Before the vote, she stressed to board members that they should only consider public health -- not expense -- when considering the new guideline.

"Today your job is to determine the level at which public health is protected," she said. "We should not settle for anything less."

Ozone pollution occurs when hydrocarbons and nitrogen oxides -- released as fossil fuels burn or chemicals evaporate -- combine with heat and sunlight.

Clean-air advocacy groups hope the upgraded California standard will influence new ozone standards currently under review at the federal level.

California is the only state that's allowed to have its own air pollution standards because it had emissions requirements in place before the federal Clean Air Act was passed in 1971, said Sonya Lunder, spokeswoman for the Environmental Working Group.

Other states can choose to follow the federal standards or California's tougher standards, she said.

The new standard passed Thursday calls for an average ozone level that doesn't exceed .07 parts per million over an eight-hour period. The federal eight-hour standard is .08 parts per million.

Seventy percent of California counties didn't meet the federal eight-hour standard between 2000-2003, said Lunder, and an estimated 92 percent of counties would fail the state standard, if implemented. The state already has a one-hour standard for ozone that is stricter than the federal rule.

The Environmental Protection Agency can withhold federal transportation funds from states that don't meet their ozone standards, but most states have until 2021 to fully comply, state officials said.

A coalition of groups representing the interests of the automobile and technology industries had opposed the new state eight-hour

guideline.

Bruce Magnani, legislative advocate for the California Chamber of Commerce, said the proposed standard is so restrictive, it approaches limiting the amount of ozone pollution to what occurs naturally in the air -- .04 parts per million.

"I think it could only have negative impacts on the economy, because it's so strict. No one knows how they're going to implement this," Magnani said.

Steven Douglas of the Alliance of Automobile Manufacturers said he was worried about a lack of information on the cost associated with the new standard. He also said he wanted to know how much the state would have to reduce ozone emissions to reach the new target.

"The very essence of good public policy is trying to find the balance between the costs and the benefits," Douglas said. "There isn't any discussion of the cost (here)."

Staff scientists said evaluating that cost would likely take at least until 2007 and possibly longer for areas around Los Angeles.

Air Resources Board

State of California

Governor Arnold Schwarzenegger

Review of the California Ambient Air Quality Standard For Ozone

Volume I of IV

Chapters 1-2

Appendix A—Proposed Amendments

Staff Report

Initial Statement of Reasons for Proposed Rulemaking

March 11, 2005

California Environmental Protection Agency

Air Resources Board

The energy challenge facing California is real. Every Californian needs to take immediate action to reduce energy consumption. For a list of simple ways you can reduce demand and cut your energy costs, see our Website: http://www.arb.ca.gov.

California Environmental Protection Agency

Alan C. Lloyd, Ph.D., Secretary

Printed on Recycled Paper

Project Coordinator

Deborah M. Drechsler, Ph.D.

Contributing Authors

Air Resources Board
Deborah M. Drechsler, Ph.D.
Cynthia Garcia
Hien Tran
Ahmed Mehadi Ph.D.
Marci Nystrom
Ralph Propper
Don Rake
Brent Takemoto, Ph.D
Tony VanCuren, Ph.D.
Whitney Webber

Office of Environmental Health Hazard Assessment
Bart Ostro, Ph.D.
Daryn Dodge

Department of Health Services
Michael Lipsett, M.D.

Contractors
Patrick L. Kinney, Sc.D., Columbia University
Laurel J. Gershwin, D.V.M., PhD., University of California, Davis
David A. Grantz, Ph.D., University of California, Riverside
Charles G. Plopper, Ph.D., University of California, Davis
Edward S. Schelegle, Ph.D., University of California, Davis

Reviewers

Air Resources Board
Linda Tombras Smith, Ph.D., Manager, Health and Ecosystem Assessment Section
Richard D. Bode, Chief, Health and Exposure Assessment Branch
Bart E. Croes, P.E., Chief, Research Division
Michael H. Scheible, Deputy Executive Officer

Office of Environmental Health Hazard Assessment
Melanie Marty, Ph.D., Chief, Air Toxicology and Epidemiology Section
George Alexeeff, Ph.D., D.A.B.T., Deputy Director for Scientific Affairs
Val F. Siebal, Chief Deputy Director

Acknowledgements

Air Resources Board
Jacqueline Cummins
Lawrence C. Larsen
Dane Westerdahl
In addition, staff wishes to acknowledges Jeff Cook , Richard Corey, Leon Dolislager, Robert Effa, Michael FitzGibbon, Shelley Green, Peggy Jenkins, Cliff Popejoy, and Ken Stroud

Disclaimer

This report has been reviewed by the staff of the Air Resources Board and the Office of Environmental Health Hazard Assessment. Mention of trade names or commercial products does not constitute endorsement or recommendation for their use. To obtain this document in an alternative format, please contact the Air Resources Board ADA Coordinator at (916) 322-4505, TDD (916) 324-9531, or (800) 700-8326 for TDD calls from outside the Sacramento area. This report is available for viewing or downloading from the Air Resources Board internet site at
http://www.arb.ca.gov/research/aaqs/ozone-rs/ozone-rs.htm

This staff report, the Notice of Public Hearing to consider Amendments to Regulations for the State Ambient Air Quality Standard for Ozone, and all subsequent regulatory documents, including the FSOR, when completed, are available on the ARB Internet site for this rulemaking at www.arb.ca.gov/regact/ozone05/ozone05.htm
Electronic copies on compacts discs or paper copies of this report may be obtained from the Public Information Office, Air Resources Board, 1001 I Street, Visitors and Environmental Services Center, 1st Floor, Sacramento, CA 95814, (916) 322-2990.

Table of Contents
Volume I

Table of Contents
Volume II

Table of Contents

Volume III

Table of Contents
Volume IV

Abbreviations and Definitions

abscission	the normal separation, involving a layer of specialized cells, of flowers, fruits and leaves of plants
AOT40	accumulated exposure over threshold of 40 ppb ozone
AQDA	air quality data action
ARB	Air Resources Board
AVG	aminoethoxyvinyl glycine
BSA	Broader Sacramento Area
Ca^{2+}	calcium ion
canopy	a cover of foliage that forms when the leaves on the branches trees in a forest overlap during the growing season
CEC	controlled environment chamber
CFR	Code of Federal Regulations
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
d	day
edaphic	the physical, chemical, and biological characteristics of soil
ESPACE	European Stress Physiology and Climate Experiment
FACE	Free Air Carbon Enrichment system, a chamber-free, open-air fumigation design
FEF25-75%	forced expiratory flow rate between 25 and 75% of forced vital capacity
FEM	federal equivalent method (for air monitoring)
FEV1	forced expiratory volume in one second
fine roots	roots with a diameter between 0.5 to 3 mm
foliar	of or referring to a plant leaf
FRM	federal reference method (for air monitoring)
full-sib	seedlings that have the same parents, but not necessarily from seed produced in the same year
FVC	forced vital capacity
g	gram
GBVAB	Great Basin Valleys Air Basin
gdw	gram dry weight
GIS	geographic information system

gfw	gram fresh weight
hr	hour
ha	hectare (= 10,000 m^2; an area that is 100 m x 100 m)
half-sib	seedlings that have one parent in common
hm	hourly mean
HNO_3	nitric acid
homeostasis	the tendency toward maintaining physiological stability within an organism (plant or animal)
H&SC	Health and Safety Code
IPM	Integrated Pest Management.
Jeffrey pine	*Pinus jeffreyi* Grev. and Balf.
k	allometric growth coefficient describing the distribution of dry weight gain between competing plant parts, defined as the ratio of the relative growth rates of the competing plant parts
K^+	potassium ion
kg	kilogram (= 1,000 g = 2.205 pounds)
km	kilometer (= 1,000 m = 0.6214 miles)
L	liter
LCAB	Lake County Air Basin
LST	local standard time
LTAB	Lake Tahoe Air Basin
m	meter (= 3.28 feet)
m^2	square meter, an area that is 1 m x 1 m
MCAB	Mountain Counties Air Basin
MDAB	Mojave Desert Air Basin
mesophyll cells	the internal cells of a leaf, distinct from cells at the leaf surface or from cell layers immediately adjacent to the leaf surface
mixed conifer	forests with a tree-layer dominated by a mixture of conifer species
montane	of or relating to a mountain or mountainous area
mRNA	messenger RNA (ribonucleic acid)
mycorrhizae	a biological association of a fungus (e.g., *Pisolithus tinctorius*) with the root cells of a plant (e.g., ponderosa pine tree)
mycorrhizal trees	trees with roots associated a mycorrhizae fungus

n	sample size
NARSTO	a public/private partnership to coordinate research in Canada, Mexico and the United States on tropospheric air pollution (formerly the North American Research Strategy for Tropospheric Ozone)
NCAB	North Coast Air Basin
NCCAB	North Central Coast Air Basin
NCLAN	National Crop Loss Assessment Network, a national study of ozone impacts on crops, undertaken during the 1980s
NEPAB	Northeast Plateau Air Basin
ng	nanogram (= 0.000000001 g = 10^{-9} g)
NH_4N_3	ammonium nitrate
nL	nanoliter (10^{-9} L)
nm	nanometer, or one billionth of a meter
NO	nitric oxide, the primary nitrogen-containing by-product of combustion
NO_2	nitrogen dioxide
NO_X	nitrogen oxides (or oxides of nitrogen)
ns	not statistically significant at p =0.05
O_3	ozone; triatomic oxygen
OII	ozone injury index
OTC	open top field exposure chamber
PAR	photosynthetically active radiation (400 – 700 nm)
phloem	the plant tissue through which sugars and other organic materials are transferred to different parts of the plant
photosynthesis	the production by green plants of organic compounds from water and carbon dioxide using energy absorbed from sunlight
Pisolithus tinctorius	a mycorrhizae-forming fungus that forms root-associations with a wide variety of pine and other tree species
ppb	parts per billion by volume
ppb-hr	parts per billion hours (i.e., sum of concentration times duration), a measure of exposure to ozone
ppm	parts per million by volume
ppm-hr	parts per million hours (i.e., sum of concentration times duration), a measure of exposure to ozone

process rates	the degree or amount at which specific actions or activities occur (e.g., water vapor loss from leaves of plants)
QAS	Quality Assurance Section (of ARB)
R:S	ratio of root biomass (dry weight) to shoot biomass
RGR	relative growth rate, defined as the difference in the dry weight of a plant or plant part over a time period, divided by the initial dry weight and the length of the time period
RH	relative humidity
RuBisCO	ribulose bisphosphate carboxylase-oxygenase
RuBP	ribulose bisphosphate
SCCAB	South Central Coast Air Basin
SCOIAS	Sierra Cooperative Ozone Impact Assessment Study
SDAB	San Diego Air Basin
senescence	the onset of aging -- a phase in plant development from maturity to the complete loss of organization and function in plants
SFBAAB	San Francisco Bay Area Air Basin
shoot	the aboveground portion of the plant (e.g., leaves, stems, flowers, and fruits)
sieve cells	the primary type of cell found in the phloem of plants
SIP	State Implementation Plan
SJVAB	San Joaquin Valley Air Basin
SoCAB	South Coast Air Basin
SSAB	Salton Sea Air Basin
sucrose	a disaccharide (with 12 carbon atoms) commonly found in plants
(sucrose) translocation	the movement of sucrose (or other soluble organic food materials) through plant tissues – most commonly from leaves to stems/roots
SUM06	an ozone exposure metric involving concentration weighting, defined as the sum of all hourly mean ozone concentrations equal to or greater than 70 ppb
terrain-effect winds	air currents influenced by the geographic features of the land that it passes over
TREEGRO	a physiologically based computer simulation model of tree growth and development

Ulmus americana	the scientific name for "American Elm"
UN-ECE	United Nations Economic Commission for Europe
USD	United States dollars
USDA	United States Department of Agriculture
USDI	United States Department of the Interior
USEPA	United States Environmental Protection Agency
USV	Upper Sacramento Valley
V_d	deposition velocity, defined as deposition flux of ozone divided by its concentration in air (usually in cm/s or m/s)
VPD	vapor pressure deficit, a measure of evaporative demand of air
whorl	the arrangement of leaves, petals, etc., at about the same place on a stem
wk	week
yr	year
ZAP	zonal application system, a chamber-free, open-air exposure system
µg	microgram (= 0.000001 g = 10^{-6} g)
µm	micrometer or micron (= 0.000001 m = 10^{-6} m)

1 Executive Summary

The California Health and Safety Code in section 39606, requires the Air Resources Board to adopt ambient air quality standards at levels that adequately protect the health of the public, including infants and children, with an adequate margin of safety. Ambient air quality standards are the legal definition of clean air. In December 2000, as a requirement of the Children's Environmental Health Protection Act (Senate Bill 25, Escutia, Stats. 1999, Health and Safety Code 39606 (d)(1)), the Air Resources Board (ARB or Board), approved a report, "Adequacy of California Ambient Air Quality Standards" (ARB and OEHHA, 2000) that contained a brief review of all of the existing health-based California ambient air quality standards.

Following this review, the standard for ozone, currently set at 0.09 parts per million (ppm) for one hour, was prioritized to undergo full review after review of the standards for particulate matter and sulfates. Staff from ARB and the Office of Environmental Health Hazard Assessment (OEHHA) have reviewed the scientific literature on public exposure, atmospheric chemistry, health effects of exposure to ozone, and welfare effects. This Staff Report or Initial Statement of Reasons (Staff Report) presents the findings of the review and the staff recommendations to revise the ozone standard in order to adequately protect public health. The proposed amendments to the ambient air quality standard for ozone are based on the health effects review contained in Volume III of this Report and the recommendation of OEHHA, as required by Health and Safety Code section 39606(a)(2).

1.1 Summary of the Staff Report/Initial Statement of Reasons

1.1.1 Health Effects of Ozone

Scientific studies show that exposure to ozone can result in reduced lung function, increased respiratory symptoms, increased airway hyperreactivity, and increased airway inflammation. Exposure to ozone is also associated with premature death, hospitalization for cardiopulmonary causes, emergency room visits for asthma, and restrictions in activity.

In controlled human exposure studies (see Chapter 9), exercising individuals exposed for 1 hour (hr) to an ozone concentration as low as 0.12 parts per million (ppm) or for 6.6 hours to a concentration as low as 0.08 ppm experienced lung function decrements and symptoms of respiratory irritation such as cough, wheeze, and pain upon deep inhalation. The lowest ozone concentrations at which airway hyperreactivity (an increase in the tendency of the airways to constrict in reaction to exposure to irritants) has been reported are 0.18 ppm ozone following 2-hour exposure in exercising subjects, 0.40 ppm following 2-hour exposure in resting subjects, and 0.08 ppm ozone in subjects exercising for 6.6 hr. Airway inflammation has been reported following 2-hour exposures to 0.20 ppm ozone and following 6.6-hour exposure to 0.08 ppm ozone.

Additional support for the exposure/response relationship for ozone health effects is derived from animal toxicological studies, which have shown that chronic ozone exposure can induce morphological (tissue) changes throughout the respiratory tract, particularly at the junction of the conducting airways and the gas exchange zone in the deep lung. In addition, the magnitude of ozone-induced effects is related to the inhaled dose (ozone concentration times breathing rate times exposure duration). Of these three factors ozone concentration is the most significant in predicting the magnitude of observed effects, followed by ventilation rate. Exposure duration has the least influence of the three factors.

Epidemiological studies (see Chapter 10) have shown positive associations between ozone levels and several health effects, including decreased lung function, respiratory symptoms, hospitalizations for cardiopulmonary causes, emergency room visits for asthma, and premature death. Children may be more affected by ozone than the general population due to effects on the developing lung and to relatively higher exposure than adults. There is little information available on the effects of ozone exposure on infants. Also, asthmatics may represent a sensitive sub-population for ozone. Since most California residents are exposed to levels at or above the current State ozone standard during some parts of the year, the statewide potential for significant health impacts associated with ozone exposure is large and wide-ranging.

1.1.2 Summary of Non-health Issues

The Staff Report contains reviews and discussions of non-health topics to provide a context for the health review and the staff recommendations for the State ozone standard. Almost all of the ozone in California's atmosphere results from reactions between substances emitted from sources including motor vehicles and other mobile sources, power plants, industrial plants, and consumer products. These reactions involve volatile organic compounds (VOC) and oxides of nitrogen (NO_X) in the presence of sunlight (Chapter 3). Ozone is a regional pollutant, as the reactions forming it take place over time, and downwind from the sources of the emissions. As a photochemical pollutant, ozone is formed only during daylight hours under appropriate conditions, but is destroyed throughout the day and night. Thus, ozone concentrations vary depending upon both the time of day and the location. Even in pristine areas there is some ambient ozone that forms from natural emissions that are not controllable (Chapter 4). This is termed "background" ozone. The average "background" ozone concentrations near sea level are in the range of 0.015 to 0.035 ppm, with a maximum of about 0.04 ppm.

The Staff Report includes an overview of statewide ozone precursor emissions that are involved in the formation of ozone (Chapter 5). The Staff Report also includes a discussion of the current ultraviolet photometry monitoring method, and a listing of approved samplers (Chapter 6). Although there are two measurement methods for ozone approved for use in the U.S. by the U.S. Environmental Protection Agency (USEPA), the method based on ultraviolet photometry is almost universally used in practice and is approved for use in California for state air quality standards.

The Staff Report includes a summary of current air quality in California, as well as long-term trends in statewide ozone concentrations (Chapter 7). Ozone is monitored continuously at approximately 175 sites in California. The highest number of exceedance days for both the State and federal 1-hour standards occurred in the San Joaquin Valley Air Basin and the South Coast Air Basin. Both areas had more than 115 State standard exceedance days and 31 or more federal standard exceedance days during each of the three years from 2001 through 2003. The Sacramento Metro Area, Mojave Desert Air Basin, and Salton Sea Air Basin all averaged more than 50 State standard exceedance days and averaged 6 or more federal standard exceedance days during 2001 through 2003. The remaining five areas (Mountain Counties Air Basin, San Diego Air Basin, San Francisco Bay Area Air Basin, South Central Coast Air Basin, and the Upper Sacramento Valley) averaged from 12 to 45 State standard exceedance days. The Upper Sacramento Valley area had no exceedances of the federal standard while the Mountain Counties Air Basin, San Diego Air Basin, San Francisco Bay Area Air Basin, and South Central Coast Air Basin each averaged 1 to 2 federal standard exceedance days for the three-year period.

The range of the measured maximum 1-hour concentrations tends to follow a similar pattern. The South Coast Air Basin showed the highest values, with measured concentrations of 0.169 ppm or higher during 2001 through 2003. The next highest 1-hour ozone concentrations occurred in the Salton Sea Air Basin and San Joaquin Valley Air Basin, which had concentrations of 0.149 ppm or higher during all three years. During 2001 through 2003, neither the State nor federal 1-hour standard was exceeded in the Lake County Air Basin, North Coast Air Basin, or Northeast Plateau Air Basin. Data for four additional areas, Great Basin Valleys Air Basin, Lake Tahoe Air Basin, North Central Coast Air Basin, and the Upper Sacramento Valley show exceedances of the State standard, but not the federal 1-hour standard (as described earlier, representative data for the Northeast Plateau Air Basin and Great Basin Valleys Air Basin are available for 2002 and 2003 only). Both the State and federal 1-hour standards were exceeded during at least two of the three years in all other areas.

Californians' indoor and personal exposures to ozone are largely determined by the outdoor ozone concentrations in their community. Nonetheless, some Californians experience a substantial exposure to ozone indoors, due to the increasing use of certain types of appliances and equipment that emit ozone. Children and those who are employed in outdoor occupations or exercise heavily outdoors, experience substantially greater exposures to ozone than the rest of the population, because they spend time outdoors during peak ozone periods.

A review of welfare effects, including effects of ozone on forest trees, agricultural crops, and materials is also discussed in this report (Chapter 8). Elevated concentrations of ozone can cause adverse effects on agricultural crops, forest trees and materials at current ambient levels, and the proposed health-based ozone standards should also provide protection to crops, forests and materials. In broad terms, impacts to crops are generally more severe than for forest trees owing to their inherently more vigorous rates of growth. Discussed in the

subsection on crops and the methods used to expose plants to ozone. This is followed by an examination of the physiological basis of ozone damage to plants, with special emphasis on carbon metabolism and the resulting impacts on crop growth and yield. Data collected since the 1950s on mixed conifer forests in the San Bernardino Mountains and the Sierra Nevada indicate that increasing numbers of ponderosa and Jeffrey pines exhibit ozone-specific needle damage due to the pollutant's cumulative effects. Also discussed are the impacts of ozone on materials, including building materials, rubber, paint, and fabrics. Although the proposed ozone standards are based on human health effects, progress toward attaining the proposed standards will provide welfare benefits.

1.2 Staff Recommendations for the Ozone Standard

California ambient air quality standards are defined in the Health and Safety Code section 39014, and 17 Cal. Code Regs. section 70101, and comprise four elements: (1) a definition of the air pollutant, (2) an averaging time, (3) a pollutant concentration, and (4) a monitoring method to determine attainment of the standard. The current California ambient air quality standard for ozone is 0.09 ppm averaged over one hour and was set by the Board in 1988. The data indicate that the current standard alone is not sufficiently protective of human health. Based on the review of the scientific literature and recommendations by OEHHA, the staff recommends that the following revisions be made to the California ambient air quality standard for ozone:

1. Ozone will continue to be the pollutant addressed by the standard.
2. Ozone 1-hour-average Standard – retain the current 1-hour-average standard for ozone at **0.09 ppm, not to be exceeded**.
3. Ozone 8-hour-average Standard – establish a new 8-hour-average standard for ozone at **0.070 ppm, not to be exceeded.**
4. Ozone Monitoring Method: retain the current monitoring method for ozone which uses the ultraviolet (UV) photometry method for determining compliance with the State ambient air quality standard for ozone. Incorporate by reference (17 Cal. Code Regs. section 70101) all federally approved UV methods (i.e., samplers) for ozone as "California Approved Samplers". This will result in no change in air monitoring equipment practices, but will align state monitoring requirements with federal requirements.

These recommendations are based on the following findings:

a. Reduced lung function and increased respiratory or ventilatory symptoms following 1-hour exposure to 0.12 ppm ozone with moderate to heavy exercise.

b. Increased airway hyperreactivity following 2-hour exposure to 0.18 ppm in exercising subjects.

c. Airway inflammation following 2-hour exposure to 0.20 ppm ozone in exercising subjects

d. Reduced lung function, increased respiratory and ventilatory symptoms, increased airway hyperreactivity, and increased airway inflammation following 6.6 to 8-hour exposure to 0.08 ppm ozone.

e. Evidence from epidemiological studies of several health endpoints including premature death, hospitalization, respiratory symptoms, and restrictions in activity and lung function.

f. Evidence from epidemiological studies of emergency room visits for asthma suggesting a possible threshold concentration between 0.075 and 0.11 ppm from analyses based on a 1-hour averaging time, and a possible threshold concentration between 0.070 and 0.10 ppm from analyses based on an 8-hour averaging time.

g. There is no evidence that children and infants respond to lower ozone concentrations than adults. Their risk is primarily related to their greater ventilation rate and greater exposure duration.

h. The dose-rate of ozone inhalation influences the magnitude of observed effects.

1.3 Other Recommendations

In light of the adverse health effects observed at current ambient concentrations and the lack of a demonstrated effect threshold for the population as a whole, staff makes the following comments:

1. Fund additional research investigating the responses of human subjects to multi-hour exposures to ozone concentrations between 0.04 and 0.08 ppm.
2. The standards should be revisited within five years, in order to re-evaluate the evidence regarding the health effects associated with ozone exposure.
3. In any air basin in California that currently attains the ambient air quality standards for ozone, air quality should not be degraded from present levels.

1.4 Estimated Health Benefits

Staff estimates that attainment of the proposed ozone standards throughout California would avoid a significant number of adverse health effects each year, specifically:

- 580 (290 – 870, probable range) premature deaths for all ages.
- 3,800 (2,200 – 5,400, 95% confidence interval (CI)) hospitalizations due to respiratory diseases for all ages.
- 600 (360 – 850, 95% CI) emergency room visits for asthma for children under 18 years of age.
- 3.3 million (430,000 – 6,100,000, 95% CI) school absences for children 5 to 17 years of age.

- 2.8 million (1.2 million – 4.6 million, 95% CI) minor restricted activity days for adults above 18 years of age.

As discussed in Appendix B, there are a several important assumptions and uncertainties in this analysis. Some have to do with study design, statistical methods, and choice of epidemiological studies used to develop the concentration-response (CR) functions used in the analysis. Few studies have investigated the shape of the CR function, or whether there is a population response threshold for health endpoints other than emergency room visits for asthma. Further uncertainty is added by assumptions in the statewide exposure assessment. It should also be noted that since several health effects related to acute exposure, and effects of chronic ozone exposure, are not included in the estimates, the health benefits associated with lowering ozone exposure are likely underestimated.

1.5 Public and Peer Review of the Staff Recommendations

The draft version of this Staff Report was released to the public on June 21, 2004 and presented for review and comment at public workshops during 2004 on July 14 in Sacramento, July 15 in El Monte, July 16 in Fresno, and August 25 in Sacramento.

The draft Staff Report was peer reviewed by the Air Quality Advisory Committee (AQAC). AQAC is a scientific peer review committee, appointed by the University of California, to independently evaluate the scientific basis of staff findings and recommendations in the draft Staff Report for revising the California ambient air quality standard for ozone. The AQAC held a public meeting to discuss its review of the draft Staff Report, comments submitted by the public, and staff responses to those comments. AQAC concluded that the report was well written and researched, and that the proposed revision to the State ozone standard was adequately supported. AQAC findings, public comments, and staff responses can be found in Appendices C-E. Following the meeting of the Air Quality Advisory Committee (AQAC), staff revised the draft Staff Report based on comments received from AQAC and the public.

1.6 Environmental and Economic Impacts

The proposed ambient air quality standards will in and of themselves have no environmental or economic impacts. Standards simply define clean air. Once adopted, local air pollution control or air quality management districts are responsible for the adoption of rules and regulations to control emissions from stationary sources to assure their achievement and maintenance. The ARB is responsible for adoption of emission standards for mobile sources and consumer products. A number of different implementation measures are possible, and each could have its own environmental or economic impact. These impacts must be evaluated when the control measure is proposed. Any environmental or economic impacts associated with the imposition of future measures will be considered if and when specific measures are proposed.

1.7 Environmental Justice Considerations

State law defines environmental justice as the fair treatment of people of all races, cultures, and incomes with respect to the development, adoption, implementation, and enforcement of environmental laws, regulations, and policies. The available literature suggests there appears to be no special vulnerability related to race, ethnicity or income level, although there may be higher exposure. Ambient air quality standards define clean air; therefore, all of California's communities will benefit from the proposed health-based standards.

1.8 Comment Period and Board Hearing

Release of this Staff Report opens the official 45-day public comment period required by the Administrative Procedure Act prior to the public meeting of the Air Resources Board to consider the staff's recommendations. Please direct all comments to either the following postal or electronic mail address:

Clerk of the Board
Air Resources Board
1001 "I" Street, 23rd Floor
Sacramento, California 95814
ozone05@listserve.arb.ca.gov

To be considered by the Board, written submissions not physically submitted at the hearing must be received at the ARB no later than 12:00 noon, April 27, 2005. Public workshops will be scheduled for April 2005 to present the final staff recommendations and receive public input on the Staff Report. Information on these workshops, as well as summaries of the presentations from past workshops and meetings are available by calling 1-916-445-0753 or at the following ARB website:
http://www.arb.ca.gov/research/aaqs/ozone-rs/ozone-rs.htm.

An oral report summarizing the staff recommendations for revising the ozone standard will be presented to the Board at a public hearing scheduled for April 28, 2005.

The staff recommends that the Board adopt the proposed amendments to the ambient air quality standards for ozone as stated above. The proposed amendments and their basis are described in detail in this Staff Report, which contains the findings of ARB and OEHHA staff's full review of the public health, scientific literature, and exposure pattern data for ozone in California. Due to the extensive nature of the literature review and the hundreds of studies reviewed, the Staff Report is divided into four volumes. Volume I contains the Executive Summary, Overview and Staff Recommendations, and Appendix A, the proposed amendments to the California Code of Regulations (amended regulatory text). Volumes II through IV present more detailed discussions of the material that is summarized in Volume I. Volume II includes background material on non-health topics, including chemistry of ozone formation and deposition, ozone precursor sources and emissions, ozone exposure and background levels, measurement methods, and welfare effects of ozone exposure. Volume III contains a summary

of ozone health effects and an in-depth discussion of the basis for the staff recommendation. Volume IV includes several appendices, including an analysis of the estimated health benefits associated with attainment of the proposed standards, summaries of Air Quality Advisory Committee and public comments and staff responses, and supplemental animal toxicologic data.

1.9 References

Air Resources Board and Office of Environmental Health Hazard Assessment (2000). Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act. Staff Report. Sacramento, CA. Available at http://www.arb.ca.gov/ch/programs/sb25/airstandards.htm.

2 Overview and Staff Recommendations

Ozone (O_3) can damage human cells upon contact, and has been implicated in a variety of adverse health effects. Scientific studies show that exposure to ozone can result in reduced lung function, increased respiratory symptoms, increased airway hyperreactivity, and airway inflammation. Exposure to ozone is also associated with premature death, hospitalization for cardiopulmonary causes, emergency room visits for asthma, and restrictions in activity. Ozone forms in the atmosphere as the result of reactions involving sunlight and two classes of directly emitted precursors. One class of precursors includes nitric oxide (NO) and nitrogen dioxide (NO_2), collectively referred to as nitrogen oxides or NO_X. The other class of precursors includes volatile organic compounds (VOCs, also called reactive organic gases or ROG), such as hydrocarbons. Ozone forms in greater quantities on hot, sunny, calm days. In metropolitan areas of California and areas downwind, ozone concentrations frequently exceed existing health-protective standards in the summertime. The current California ambient air quality standard for ozone is 0.09 ppm for one hour.

The sources of ozone precursor emissions within California have been grouped into three major categories: point sources, which are distinct facilities such as power plants and factories; mobile sources, which includes cars, trucks, and off-road mobile equipment; and area-wide sources, which include agricultural and construction activities, and consumer products. VOCs are emitted from vehicles, factories, fossil fuels combustion, evaporation of paints, and many other sources. NO_X is emitted from high-temperature combustion processes, such as at power plants or in motor vehicle exhaust.

The concentrations of ozone measured in the air vary both regionally and seasonally throughout California. For example, the Los Angeles area and the San Joaquin Valley experience highest ozone levels in the state. Ozone concentrations are typically higher during the summer months than the winter months.

To help understand which sources contribute to high ozone levels, the ARB has developed and maintains detailed facility and source specific estimates of the overall estimated ozone precursor emissions. Only the precursor gases are estimated. As a complement to emission inventory and routinely collected air quality monitoring data, the ARB conducts atmospheric modeling, using these precursor emission inventories and other appropriate information, to estimate ozone levels

2.1 Setting California Ambient Air Quality Standards

Ambient air quality standards (AAQS) represent the legal definition of clean air. They specify concentrations and durations of exposure to air pollutants that reflect the relationships between the intensities and composition of air pollution and undesirable effects (Health and Safety Code section 39014). The objective of an AAQS is to provide a basis for preventing or abating adverse health or welfare effects of air pollution (17 Cal. Code Regs. section 70101).

Health and Safety Code section 39606(a)(2) authorizes the Air Resources Board (Board) to adopt standards for ambient air quality "in consideration of public health,

safety, and welfare, including, but not limited to, health, illness, irritation to the senses, aesthetic value, interference with visibility, and effects on the economy." Standards represent the highest pollutant concentration for a given averaging time that is estimated to be without adverse effects for most people. Standards are set to ensure that sensitive population sub-groups are protected from exposure to levels of pollutants that may cause adverse health effects. A margin of safety is added to account for possible deficiencies in the data and measuring methodology. Health-based standards are based on the recommendation of the Office of Environmental Health Hazard Health Assessment (OEHHA).

Recent legislation requires that infants and children be given special consideration when ambient air quality standards are adopted. As part of its recommendation to the ARB, the statute requires OEHHA to use current principles, practices, and methods used by public health professionals to assess the following considerations for infants and children:

1. Exposure patterns among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.
2. Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.
3. The effects on infants and children of exposure to ambient air pollutants and other substances that have a common mechanism of toxicity.
4. The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

The law also requires that the scientific basis or the scientific portion of the method used to assess these considerations be peer reviewed (Health and Safety Code section 39606(c)). The draft Staff recommendations and their bases, including OEHHA's assessment and recommendation, is peer reviewed by the Air Quality Advisory Committee (AQAC). AQAC is an external peer review committee established in accordance with section 57004 of the Health and Safety Code and appointed by the President of the University of California a University of California. The AQAC meets to independently evaluate the scientific basis of draft recommendations for revising the California ambient air quality standards.

Ambient air quality standards should not be interpreted as permitting, encouraging, or condoning degradation of present air quality that is superior to that stipulated in the standards. Rather, they represent the minimum acceptable air quality. An AAQS adopted by the Board is implemented, achieved, and maintained by numerous rules and regulations that limit pollution from specific sources of ozone precursors. These rules and regulations are primarily, though not exclusively, emission limitations established by the regional and local air pollution control and air quality management districts for stationary sources, and by the Board for vehicular sources and consumer products (see generally, Health and Safety Code sections 39002, 40000, and 40001).

2.2 Current California Ambient Air Quality Standard for Ozone

The current California ambient air quality standard for ozone, established in 1988, is 0.09 ppm (180 $\mu g/m^3$) for a one-hour average. This value is not to be exceeded. This standard was established based on the following most relevant effects, which are listed in the table of standards (17 Cal. Code Regs. section 70200):

a. Short-term exposures:

(1) Pulmonary function decrements and localized lung edema in humans and animals.

(2) Risk to public health implied by alterations in pulmonary morphology and host defence in animals.

b. Long-term exposures: Risk to public health implied by altered pulmonary morphology in animals after long-term exposures and pulmonary function decrements in chronically exposed humans.

c. Welfare effects:

(1) Yield loss in important crops and predicted economic loss to growers and consumers.

(2) Injury and damage to native plants and potential changes in species diversity and number.

(3) Damage to rubber and elastomers and to paints, fabric, dyes, pigments, and plastics.

The US EPA has set national ambient air quality standards, as noted in the table below. The federal one-hour standard will be phased out beginning in June 2005. The Federal Clean Air Act gives California authority to set its own ambient air quality standards in consideration of statewide concerns. California has the largest number of exceedances of the Federal 8-hour ozone standard in the United States, supporting California's need to address a significant statewide public health issue.

Current Ambient Air Quality Standards for Ozone

Averaging Time	California Standard	Federal Standard
1 Hour	0.09 ppm (180 $\mu g/m^3$)	0.12 ppm (235 $\mu g/m^3$)
8 Hour	—	0.08 ppm (157 $\mu g/m^3$)

2.3 History of Ozone/Oxidant Standards

The first state oxidant standard was set in December 1959 by the state Department of Public Health (DPH), which had the responsibility for setting air pollution standards before the creation of the ARB. This standard was set at 0.15 ppm, averaged for one hour. The standard was for oxidant, rather than ozone, because the monitoring method available at that time, the potassium iodide (KI) method, measured all ambient oxidant

gases, including ozone and other oxidants such as peroxyacetyl nitrate (PAN) nitrogen dioxide, photochemical aerosols, and other unknown oxidants.

In 1969, the newly-created ARB reviewed the oxidant standard set by DPH and revised the standard to a concentration of 0.10 ppm, averaged over one hour, not to be equaled or exceeded. The information considered by the Board in 1969 included adverse effects upon: (1) the health of humans and animals; (2) vegetation; (3) materials; and (4) visibility. Eye irritation was listed as the most relevant effect of oxidant.

In 1974, the Board introduced ultraviolet photometry as the monitoring method for the standard. However, since ultraviolet photometry measures only ozone, the Board changed the designation of the standard from "oxidant" to "oxidant (as ozone)." Because only ozone was to be measured, the Board changed the most relevant effect from: "eye irritation" (which is caused primarily by peroxyacyl nitrates or PANs) to "aggravation of respiratory disease" (which is caused primarily by ozone).

In 1988, the Board changed the designation of the standard from "oxidant (as ozone)" to "ozone", and revised the standard to a concentration of 0.09 ppm, averaged over one hour, to reflect that the listed relevant effects were related to ozone exposure, rather than to oxidants in general.

For comparison, in 2000, the World Health Organization established a guideline value for ozone in ambient air of 120 $\mu g/m^3$ (0.061 ppm) for a maximum period of 8 hours per day (WHO 2000).

2.4 Review of the California Ambient Air Quality Standards

The Children's Environmental Health Protection Act (Senate Bill 25, Escutia, Stats. 1999, ch. 731) required the ARB, in consultation with the OEHHA, to evaluate all health-based standards by December 31, 2000, to determine whether the standards were adequately protective of the health of the public, including infants and children (Health and Safety Code section 39606 (d)). At its December 7, 2000 meeting, the Board approved a report, "Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act" (ARB, et al., 2000), prepared by ARB and OEHHA staffs. The Adequacy Report concluded that health effects may occur in infants and children and other potentially susceptible subgroups exposed to ozone at or near levels corresponding to the current standard. The report identified the standard for ozone as having the second highest priority for further detailed review and possible revision. The standard for PM10 (including sulfates) had the highest priority and was reviewed and revised in 2002, including establishment of a new standard for PM2.5.

2.5 Findings of the Standard Review

2.5.1 Chemistry and Physics

Most of the ozone in California's air results from reactions between substances emitted from sources including motor vehicles, power plants, industrial plants, consumer products, and vegetation. These reactions involve volatile organic compounds (VOCs, which the ARB also refers to as reactive organic gases or ROG) and oxides of nitrogen (NO_X) in the presence of sunlight. Ozone is a regional pollutant, as the reactions

forming it take place over time, and downwind from the precursor sources. As a photochemical pollutant, ozone is formed only during daylight hours under appropriate conditions, but is destroyed throughout the day and night. Thus, ozone concentrations vary depending upon both the time of day and the location. Ozone concentrations are higher on hot, sunny, calm days. In metropolitan and downwind areas of California, ozone concentrations frequently exceed regulatory standards during the summer.

2.5.2 Ozone Background

Even in pristine areas there is some ambient ozone that forms from natural emissions that are not controllable. This is termed "background" ozone. Overall, it appears that "background" ozone in California is dominated by natural tropospheric and stratospheric processes. The effects of occasional very large biomass fires and anthropogenic emissions are secondary factors. The foregoing discussion indicates that average "natural background" ozone near sea level is in the range of 0.015 to 0.035 ppm, with a maximum of about 0.04 ppm. Exogenous enhancements to "natural" levels generally are small (about 0.005 ppm), and are unlikely to alter peak concentrations.

At altitudes above 2 km stratospheric intrusions can push peak ambient concentrations to 0.045 to 0.050 ppm. The timing, spatial extent, and chemical characteristics of stratospheric air mass intrusions makes these events recognizable in air quality records, providing that the affected region has a fairly extensive monitoring network and that multiple air quality parameters (CO, VOC, PM, RH) are being measured as well.

Intermittent episodes of "natural" ozone from very large biomass fires in boreal forests (Alaska, Canada, Siberia) can produce short-lived pulses of ozone up to 0.020 ppm that may arrive during the North American ozone season. Present understanding suggests that these are infrequent events at latitudes below about 50N. There are no data documenting such an event in California. Long range transport of anthropogenic ozone may grow as Asian energy consumption increases the continent's NO_X emissions. Model studies indicate that the Asian ozone increment in North America could double over the next few decades. Assuming the temporal pattern of transport remains unchanged, such an impact could increase mean ozone concentrations by 0.002 to 0.006 ppm. The potential effect on peak transport events is unknown at this time.

2.5.3 Ozone Precursor Emissions

Ozone is an oxidant gas that forms photochemically in the atmosphere when nitrogen oxides (NO_X) and reactive organic gases (ROG) are present under appropriate atmospheric conditions (see Chapter 5). Carbon monoxide (CO) is also an ozone precursor. Both ROG and NO_X are emitted from mobile sources, point sources, and area-wide sources. ROG emissions from anthropogenic sources result primarily from incomplete fuel combustion, and from the evaporation of solvents and fuels, while NO_X and CO emissions result almost entirely from combustion processes.

2.5.4 Monitoring Method

Two measurement methods for ozone are approved for use in the U.S. by the USEPA: one is based on the chemiluminescence that occurs when ozone and ethylene react, and the other on the attenuation of ultraviolet (UV) radiation by ozone. The method based on UV spectrometry is almost universally used in practice. Specifications and

criteria for both methods exist in federal regulation. The UV photometry-based method is approved for use in California for state air quality standards. Both state and federal requirements are applied directly by the ARB and the air districts in the ozone monitoring network in California.

2.5.5 Exposure

During 2001 through 2003, neither the State nor federal 1-hour standard was exceeded in the Lake County Air Basin, North Coast Air Basin, or Northeast Plateau Air Basin. Data for four additional areas, Great Basin Valleys Air Basin, Lake Tahoe Air Basin, North Central Coast Air Basin, and the Upper Sacramento Valley show exceedances of the State standard, but not the federal 1-hour standard (as described earlier, representative data for the Northeast Plateau Air Basin and Great Basin Valleys Air Basin are available for 2002 and 2003 only). Both the State and federal 1-hour standards were exceeded during at least two of the three years in all other areas.

The highest 8-hour average values were found in the South Coast Air Basin and San Joaquin Valley Air Basin. Maximum 8-hour concentrations in the South Coast Air Basin ranged from 0.144 ppm to 0.153 ppm during 2001 through 2003, while maximum 8-hour concentrations in the San Joaquin Valley ranged from 0.120 ppm to 0.132 ppm during the same three-year period. Three other areas, the Mojave Desert Air Basin, the Sacramento Metro Area, and the Salton Sea Air Basin also had a maximum 8-hour concentration above 0.120 ppm during at least one of the three years.

With respect to the federal 8-hour ozone standard, Lake County Air Basin and North Coast Air Basin showed no exceedance days during 2001 through 2003. One area, the Lake Tahoe Air Basin, averaged only one exceedance day for the three-year period, while the North Central Coast Air Basin averaged three 8-hour exceedance days. In contrast, the San Joaquin Valley Air Basin showed the highest average number of exceedance days (123), followed by the South Coast Air Basin (99). The Sacramento Metro Area, Mojave Desert Air Basin, Mountain Counties Air Basin, and Salton Sea Air Basin each averaged between 42 and 68 exceedance days during 2001 through 2003. The remaining four areas averaged between 7 and 25 federal 8-hour exceedance days during the three-year period.

Californians' indoor and personal exposures to ozone are largely determined by the outdoor ozone concentrations in their community. Nonetheless, some Californians experience a substantial exposure to ozone indoors, due to the increasing use of certain types of appliances and equipment that emit ozone. Others, such as many children and those who are employed in outdoor occupations, may experience substantially greater exposures to ozone than the rest of the population, because they spend time outdoors during peak ozone periods.

2.5.6 Welfare Effects

A review of welfare effects, including effects of ozone on forest trees, agricultural crops, and materials is also discussed in this report (Chapter 8). Elevated concentrations of ozone can cause adverse effects on agricultural crops, forest trees and materials at current ambient levels, and the proposed health-based ozone standards should also provide protection to crops, forests and materials. In broad terms, impacts to crops are

generally more severe than for forest trees owing to their inherently more vigorous rates of growth. Discussed in the subsection on crops and the methods used to expose plants to ozone. This is followed by an examination of the physiological basis of ozone damage to plants, with special emphasis on carbon metabolism and the resulting impacts on crop growth and yield. Data collected since the 1950s on mixed conifer forests in the San Bernardino Mountains and the Sierra Nevada indicate that increasing numbers of ponderosa and Jeffrey pines exhibit ozone-specific needle damage due to the pollutant's cumulative effects. Also discussed are the impacts of ozone on materials, including building materials, rubber, paint, and fabrics. Although the proposed ozone standards are based on human health effects, progress toward attaining the proposed standards will provide welfare benefits.

2.5.7 Health Effects

Review of the controlled human exposure, animal toxicology and epidemiologic literature led to the following conclusions as to the health effects of ozone exposure:

1. The lowest ozone concentration at which reduced lung function and increased respiratory and ventilatory symptoms have been observed following 1-hour exposure is 0.12 ppm with moderate to heavy exercise.
2. The lowest ozone concentration at which increased airway hyperreactivity following 2-hour exposure has been reported is 0.18 ppm in exercising subjects.
3. The lowest ozone concentration at which airway inflammation following 2-hour exposure has been reported is 0.20 ppm ozone in exercising subjects
4. Reduced lung function, increased respiratory and ventilatory symptoms, increased airway hyperreactivity, and increased airway inflammation have been reported following 6.6- to 8-hour exposure to 0.08 ppm ozone.
5. Evidence from epidemiological studies of several health endpoints including premature death, hospitalization, respiratory symptoms, and restrictions in activity and lung function.
6. Evidence from epidemiological studies of emergency room visits for asthma suggests a possible threshold concentration between 0.075 and 0.11 ppm from analyses based on a 1-hour averaging time, and a possible threshold concentration between 0.070 and 0.10 ppm from analyses based on an 8-hour averaging time.
7. There is no evidence that children and infants respond to lower ozone concentrations than adults. Their risk is primarily related to their greater ventilation rate and greater exposure duration.
8. The dose-rate of ozone inhalation influences the magnitude of observed effects.

2.6 Summary of Recommendations

Following a detailed review of the scientific literature on the health and welfare effects of ozone, staff is proposing to revise the ambient air quality standard for ozone. The recommended ozone standards are based on scientific information about the health impacts associated with ozone exposure, recognizing the uncertainties in these data. The definition of California ambient air quality standards assumes a threshold below

which effects do not occur. However, the extremely wide range of individual responsiveness to ozone makes identification of a threshold on a population level somewhat problematic. In addition, the Children's Environmental Health Protection Act [Senate Bill 25, Escutia; Stats. 1999, Ch. 731, H&SC section 39606(d)(2)] requires a standard that "adequately protects the health of the public, including infants and children, with an adequate margin of safety." Recognizing the uncertainties in the database, staff makes the following recommendations.

1. Ozone will continue to be the pollutant addressed by the standard.

2. One-hour ambient air quality standard: staff recommends retaining the current 1-hour ozone standard at a concentration of **0.09 ppm**, not to be exceeded, based on several factors. First, at 0.12 ppm, in several studies 10 - 25% of the subjects experienced a decline of 10% of more in FEV1. In one study, these lung function changes were accompanied by increases in cough. At 0.24 ppm, increases were also observed in shortness of breath and pain on deep breath. These lung function and symptom outcomes have been demonstrated and replicated in several carefully controlled human exposure studies. The population at risk for these effects includes children and adults engaged in active outdoor exercise and workers engaged in physical labor outdoors. Thus, a margin of safety is necessary to account for variability in human responses. In addition, the chamber studies, by design, do not include potentially vulnerable populations (e.g., people with moderate to severe asthma, Chronic Obstructive Pulmonary Disease or COPD, and heart disease) who may be incorporated in the epidemiologic studies.

   Second, chamber studies indicate that bronchial responsiveness and pulmonary inflammation occur with 1-hour exposure to 0.18 to 0.20 ppm. Bronchial responsiveness can aggravate pre-existing chronic respiratory disease. The ultimate impact of the inflammatory response is unclear but repeated exposures to high ozone levels may result in restructuring of the airways, fibrosis, and possibly permanent respiratory injury. These latter outcomes are supported by animal toxicology studies, which also suggest the possibility of decreases in lung defense mechanisms.

   Third, epidemiological studies completed over the last 10 years indicate the potential for severe adverse health outcomes including premature death, hospitalizations, and emergency room visits. These studies include concentrations to which the public is currently being exposed. It is possible that some of these associations are due to relatively short-term exposures, for example less than two hours, since people at risk of experiencing these endpoints are unlikely to be engaged in multi-hour periods of moderate or heavy work or exercise outdoors. However, since there is high temporal correlation between 1-, 8-, and 24-hour average ozone concentrations, the averaging time of concern cannot be discerned from these studies.

   Viewing all of the evidence, staff recommends retention of the 1-hour standard of 0.09 ppm, not to be exceeded, as being protective of public health with an adequate margin of safety.

3. Eight-hour ambient air quality standard: We recommend establishing a new 8-hour average standard of **0.070 ppm**, not to be exceeded. Our recommendation for the 8-

hour standard is based primarily on the chamber studies that have been conducted over the last 15 years, supported by the important health outcomes reported in many of the epidemiologic studies. With exposure for 6.6 to 8-hours to an ozone concentration of 0.08 ppm, several studies have reported statistically significant group effects on lung function changes, ventilatory and respiratory symptoms, airway hyperresponsiveness, and airway inflammation in healthy, exercising individuals. A substantial fraction of subjects in these studies exhibited particularly marked responses in lung function and symptoms. Consequently, a concentration of 0.08 ppm ozone for an 8-hour averaging time can not be considered adequately protective of public health, and does not include any margin of safety, based on the definitions put forth in State law. The one published multi-hour study investigating a concentration below 0.08 ppm showed no statistically significant group mean decrement in lung function or symptoms at 0.04 ppm compared to a baseline of clear air. In addition, all individual subjects had changes in FEV1 of less than 10%. One unpublished multi-hour study at 0.06 ppm (Adams 1998) reported no statistically significant group mean changes, relative to clean air, in either lung function or symptoms including pain on deep inhalation and total symptom score. Therefore, staff has recommended an 8-hour concentration of 0.070 ppm. Many of the studies, and issues and concerns associated with the epidemiological studies listed above concerning the 1-hour standard are also relevant to the 8-hour standard. As discussed above, it may be that the health effects, often correlated with 1-hour exposures in the epidemiologic studies, are actually associated with 8-hour (or other) average exposures. Therefore, these epidemiologic findings were factored into the margin of safety for the 8-hour average.

It should be noted that the recommended 8-hour average concentration has three rather than two decimal places. Staff initially considered selection of 0.07 ppm. However, rounding conventions applied to air quality data (see Section 7.1.4) are such that any measured value up to and including 0.074 ppm would round down to 0.07 ppm. The available data suggested that selection of 0.07 ppm would not include an adequate margin of safety, as required by State law. The one available study at 0.06 ppm did not find a group mean effect. Staff is recommending that the 8 hour average standard have three decimal places, 0.070 ppm, to ensure an adequate margin of safety. Section 6.3 discusses issues related to precision and accuracy of the monitored data.

4. Monitoring method for ozone: Staff recommends retention of the current monitoring method for ozone which uses the ultraviolet (UV) absorption method for determining compliance with the state Ambient Air Quality Standard for ozone. Incorporate by reference all federally approved UV methods for ozone as California Approved Samplers for ozone. This will not change current air monitoring practices, but will align state monitoring requirements with federal requirements.

2.6.1 Consideration of Infants and Children

The Children's Environmental Health Protection Act [Health and Safety Code section 39606 (b)] requires that air pollution effects on children and infants be specifically considered in selection of ambient air quality standards. Children have a higher ventilation rate relative to body weight at rest and during activity than adults. Children

also tend to spend more time outside and be more active than adults. Consequently, virtue of their higher ventilation rates and outdoor behavior patterns, they are likely to inhale larger total doses of ozone than the general population. However, the chamber studies of exercising children suggest that they have responses generally similar to adults, pointing to a similar degree of responsiveness. Epidemiologic studies that have examined both children and adults do not show clear evidence for greater sensitivity in children. Studies in animals at high exposure concentrations (0.5 ppm and higher, 8 hrs/day for several consecutive days) indicate that developing lungs of infant animals are adversely affected by ozone. The recommended standards are well below that level of exposure. Two studies have shown evidence of lower lung function in young adults raised in high ozone areas (Kunzli et al. 1997; Galizia and Kinney 1999). The study by Kunzli et al. (1997) suggested that exposure to ozone prior to age 6 was associated with lower attained lung function. Examination of data for the Los Angeles basin from the early 1980s, show summer averages of the 1-hour maximum to be above 0.10 ppm. This is considerably above present levels and above the recommended 1-hour standard. There is also evidence that children who play three or more sports are at higher risk of developing asthma if they also live in high ozone communities in Southern California. This study needs to be repeated before the effect can be attributed to ozone exposure with greater certainty, but the finding is of concern. The warm season daily 8-hour maximum concentrations of ozone measured in these high ozone areas, over the four years of study, was 0.084 ppm. The proposed 8-hour standard of 0.070 ppm, therefore, should protect most children from asthma induction that may be associated with ozone exposure. Collectively, this body of evidence suggests that although children appear to be similarly responsive to a given dose of ozone as adults, they are at greater risk than adults of experiencing adverse responses to ozone by virtue of their higher level of outdoor activity, and consequently greater total exposure.

2.7 Estimated Health Benefits

It is estimated that attainment of the proposed ozone standards throughout California would avoid a significant number of adverse health effects each year, specifically:

- 580 (290 – 870, probable range) premature deaths for all ages.
- 3,800 (2,200 – 5,400, 95% confidence interval (CI)) hospitalizations due to respiratory diseases for all ages.
- 600 (360 – 850, 95% CI) emergency room visits for asthma for children under 18 years of age.
- 3.3 million (430,000 – 6,100,000, 95% CI) school absences for children 5 to 17 years of age.
- 2.8 million (1.2 million – 4.6 million, 95% CI) minor restricted activity days for adults above 18 years of age.

As discussed in Appendix B, there are a several important assumptions and uncertainties in this analysis. Some concern the study design, statistical methods, and choice of epidemiological studies used to develop the concentration-response (CR) functions used in the analysis. Few studies have investigated the shape of the CR function, or whether there is a population response threshold for health endpoints other

than emergency room visits for asthma. Further uncertainty is added by assumptions in the statewide exposure assessment. It should also be noted that since several health effects related to acute exposure, and effects of chronic ozone exposure, are not included in the estimates noted above, the health benefits associated with lowering ozone exposure are likely underestimated.

2.8 Public Outreach and Review

A draft Staff Report containing staff's preliminary findings was released to the public on June 21, 2004 titled, "Review of California Ambient Air Quality Standard for Ozone". Public outreach for the standard review involved dissemination of information through various outlets to include the public in the regulatory process. In an ongoing effort to include the public in the review of the ozone standard, the ARB and OEHHA integrated outreach into public meetings, workshop presentations, electronic "list serve" notification systems, and various web pages. Notification of release of the Staff Report, the schedule for public meetings and workshops, and invitations to submit comments on the Staff Report were made through the "list serve" notification system. Public workshops on the proposed ozone standard were held on July 14 – 16, 2004 in Sacramento, El Monte, and Fresno. An additional public workshop was held on August 24, 2004 in Sacramento.

Individuals or parties interested in signing up for an electronic e-mail "list serve" notification on the PM standards, as well as any air quality-related issue, may self-enroll at the following location: www.arb.ca.gov/listserv/aaqs/aaqs.htm. Additional information on the standards review process is also available at the ozone standards review schedule website at: www.arb.ca.gov/research/aaqs/ozone-rs/ozone-rs.htm.

2.9 Air Quality Advisory Committee Review

The Air Quality Advisory Committee, an external scientific peer review committee that was appointed by the President of the University of California, met January 11 and 12, 2005, in Berkeley, California to review the initial Staff Report and public comments, and to ensure that the scientific basis of the recommendations for the ozone standard are based upon sound scientific knowledge, methods, and practices. The AQAC held a public meeting, which provided time for oral public comments, and discussed their review of the draft Staff Report and the draft recommendations, and provided comments for improving the draft Staff Report. Final findings were received on February 24, 2005.

The AQAC determined that the staff recommendations were well founded on the scientific literature, and voted to endorse them. The Committee made suggestions for minor changes to the draft Staff Report to increase clarity, requested more detailed discussion of several topics, and inclusion of several additional scientific papers. The AQAC findings is included in this Initial Statement of Reasons as Appendix C, in Volume IV.

2.10 Environmental and Economic Impacts

The proposed ambient air quality standards are scientific in nature, and will in and of themselves have no environmental or economic impacts. Standards simply define clean air. Once adopted, local air pollution control or air quality management districts are

responsible for the adoption of rules and regulations to control emissions from stationary sources to assure their achievement and maintenance. The Board is responsible for adoption of emission standards for mobile sources. A number of different implementation measures are possible, and each could have its own environmental and/or economic impact. These impacts must be evaluated when the control measure is proposed. Any environmental or economic impacts associated with the imposition of future measures will be considered if and when specific measures are proposed.

2.11 Environmental Justice

State law defines environmental justice as the fair treatment of people of all races, cultures, and incomes with respect to the development, adoption, implementation, and enforcement of environmental laws, regulations, and policies (Senate Bill 115, Solis; Stats 1999, Ch. 690; Government Code §65040.12(c)). The Board established a framework for incorporating environmental justice into the ARB's programs consistent with the directives of State law (ARB, 2001). The policies developed apply to all communities in California, but recognize that environmental justice issues have been raised more in the context of low-income and minority communities, which sometimes experience higher exposures to some pollutants as a result of the cumulative impacts of air pollution from multiple mobile, commercial, industrial, areawide, and other sources.

Because ambient air quality standards simply define clean air, all of California's communities will benefit from the proposed health-based standards, as progress is made to attain the standards. Over the past twenty years, the ARB, local air districts, and federal air pollution control programs have made substantial progress towards improving the air quality in California. However, some communities continue to experience higher exposures than others as a result of the cumulative impacts of air pollution from multiple mobile and stationary sources and thus may suffer a disproportionate level of adverse health effects. Since the same ambient air quality standards apply to all regions of the State, these communities will benefit by a wider margin and receive a greater degree of health improvement from the revised standards than less affected communities, as progress is made to attain the standards. Moreover, just as all communities would benefit from new, stricter standards, alternatives to the proposed recommendations, such as not proposing an eight-hour ozone standard, would adversely affect many communities.

While it is possible that residents in environmental justice communities may be particularly sensitive to ozone, only one study investigated whether socioeconomic status (SES) alters responses to ozone exposure, and those results were difficult to explain. Hence, the study did not allow inferences as to whether socioeconomic status impacts on sensitivity to ozone. Moreover, other controlled studies investigating whether gender, ethnicity or environmental factors contribute to the responses to ozone exposure could not convincingly demonstrate a link with responsiveness. Therefore, the database is insufficient to conclude whether differences in ozone susceptibility exist in environmental justice communities. These studies are discussed in more detail in Section 9.6.8.

Once ambient air quality standards are adopted, the ARB and the local air districts will

propose emission standards and other control measures designed to result in a reduction of ambient ozone levels. The environmental justice aspects of each proposed control measure will be evaluated in a public forum at this time.

As additional relevant scientific evidence becomes available, the ozone standards will be reviewed again to make certain that the health of the public is protected with an adequate margin of safety.

2.12 References

Adams WC. 1998. Dose-response effects of varied equivalent minute ventilation rates on pulmonary function responses during exposure to ozone. Final Report to the American Petroleum Institute. Washington D.C.

Air Resources Board. Ambient Air Quality Standard for Ozone: Health and Welfare Effects. Staff Report. September 1987. Sacramento, CA.

Air Resources Board and Office of Environmental Health Hazard Assessment. Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act. Staff Report. 2000.

Air Resources Board (2001). Policies and Actions for Environmental Justice, December 13, 2001.

Galizia A, Kinney PL. 1999. Long-term residence in areas of high ozone: associations with respiratory health n a nationwide sample of nonsmoking young adults. Environ Health Perspect 107:675-679.

Kunzli N, Lurmann F, Segal M, Ngo L, Balmes J, Tager IB. 1997. Association between lifetime ambient ozone exposure and pulmonary function in college freshmen – results of a pilot study. Environ Res 72:8-23.

McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. 2002. Asthma in exercising children exposed to ozone: a cohort study. Lancet 359:386-391.

World Health Organization (2000). Air Quality Guidelines for Europe, Second Edition. (WHO regional publications, European series, No. 91.)

Appendix A

PROPOSED AMENDMENTS TO CALIFORNIA CODE OF REGULATIONS

AND

AIR MONITORING QUALITY ASSURANCE MANUAL VOLUME IV, PARTS A, B, & C (DOCUMENT INCORPORATED BY REFERENCE)

[PROPOSED] REGULATION ORDER

Section 70100. Definitions

~~(g) Oxidant. Oxidant is a substance that oxidizes a selected reagent that is not oxidizable by oxygen under ambient conditions. For the purposes of this section, oxidant includes ozone, organic peroxides, and peroxyacyl nitrates but not nitrogen dioxide. Atmospheric oxidant concentrations are to be measured with ozone as a surrogate by ultraviolet photometry, or by an equivalent method.~~

(g~~h~~) Carbon Monoxide ...

(h~~i~~) Sulfur Dioxide ...

(ij) Suspended Particulate Matter (PM10). Suspended particulate matter (PM10) refers to atmospheric particles, solid and liquid, except uncombined water as measured by a (PM10) sampler which collects 50 percent of all particles of 10 mm aerodynamic diameter and which collects a declining fraction of particles as their diameter increases and an increasing fraction of particles as their diameter decreases, reflecting the characteristics of lung deposition. ~~Suspended particulate matter (PM10) is to be measured by a California Approved Sampler (CAS) for PM10, for purposes of monitoring for compliance with the Suspended Particulate Matter (PM10) standards. Approved samplers, methods, and instruments are listed in Section 70100.1(a) below. A CAS for PM10 includes samplers, methods, or instruments determined by the Air Resources Board or the Executive Officer to produce equivalent results for PM10 with the Federal Reference Method (40 CFR, part 50, Appendix M, as published in 62 Fed. Reg. 38763, July 18, 1997).~~

(j~~k~~) Fine Suspended Particulate Matter (PM2.5). Fine suspended particulate matter (PM2.5) refers to suspended atmospheric particles solid and liquid, except uncombined water as measured by a PM2.5 sampler which collects 50 percent of all particles of 2.5 mm aerodynamic diameter and which collects a declining fraction of particles as their diameter increases and an increasing fraction of particles as their diameter decreases, reflecting the characteristics of lung deposition. ~~Fine suspended particulate matter (PM2.5) is to be measured by a California Approved Sampler (CAS) for PM2.5 for purposes of monitoring for compliamce with the Fine Particulate Matter (PM2.5) standards. Approved samplers, methods, and instruments are listed in Section 70100.1(b) below. A CAS for PM2.5 includes samplers, method, and instruments determined by the Air Resources Board or the Executive Officer to produce equivalent results for PM2.5 with the Federal Reference Method (40 CFR, part 50, Appendix L, as published in 62 Fed. Reg. 38763, July 18, 1997).~~

(kl) Visibility Reducing Particles ...

(lm) Hydrogen Sulfide ...

(mn) Nitrogen Dioxide ...

(no) Lead (particulate) ...

(op) Sulfates ...

(pq) Vinyl Chloride ...

(qr) Ozone ...

(rs) Extinction Coefficient ...

Section 70100.1. Methods, Samplers, and Instruments for Measuring Pollutants.

a) PM10 Methods. The method for determining compliance with the PM10 ambient air quality standard shall be the Federal Reference Method for the Determination of Particulate Matter as PM10 in the Atmosphere (40 CFR, Chapter 1, part 50, Appendix M, as published in 62 Fed. Reg., 38753, July 18, 1997). California Approved Samplers for PM10 are set forth in "Air Monitoring Quality Assurance Manual Volume IV, Part A: Monitoring Methods for PM10", adopted [insert date], which is incorporated by reference herein. Samplers, methods, or instruments determined in writing by the Air Resources Board or the Executive Officer to produce equivalent results for PM10 shall also be California Approved Samplers for PM10. These include those continuous samplers that have been demonstrated to the satisfaction of the Air Resources Board to produce measurements equivalent to the Federal Reference Method. ~~The following samplers, methods, and instruments are California Approved Samplers for PM10 for the purposes of monitoring for compliance with the Suspended Particulate Matter (PM10) standards:~~

~~(1) The specific samplers approved are:~~

~~(A) Andersen Model RAAS10-100 PM10 Single Channel PM10 Sampler, U.S. EPA Manual Reference Method RFPS-0699-130, as published in 64 Fed. Reg., 33481, June 23, 1999.~~

~~(B) Andersen Model RAAS10-200 PM10 Single Channel PM10 Audit Sampler, U.S. EPA Manual Reference Method RFPS-0699-131, as published in 64 Fed. Reg., 33481, June 23, 1999.~~

~~(C) Andersen Model RAAS10-300 PM10 Multi Channel PM10 Sampler, U.S. EPA Manual Reference Method RFPS-0669-132, as published in 64 Fed. Reg., 33481, June 23, 1999.~~

~~(D) Sierra (currently known as Graseby) Andersen/GMW Model 1200 High Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-1287-063, as published in 52 Fed. Reg., 45684, December 1, 1987 and in 53 Fed. Reg., 1062, January 15, 1988.~~

~~(E) Sierra (currently known as Graseby) Andersen/GMW Model 321B High Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-1287-064, as published in 52 Fed. Reg., 45684, December 1, 1987 and in 53 Fed. Reg., 1062, January 15, 1988.~~

~~(F) Sierra (currently known as Graseby) Andersen/GMW Model 321-C High Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-1287-065, as published in 52 Fed. Reg., 45684, December 1, 1987.~~

~~(G) BGI Incorporated Model PQ100 Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-124, as published in 63 Fed. Reg., 69624, December 17, 1998.~~

~~(H) BGI Incorporated Model PQ200 Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-125, as published in 63 Fed. Reg., 69624, December 17, 1998.~~

~~(I) Rupprecht & Patashnick Partisol Model 2000 Air Sampler, U.S. EPA Manual Reference Method RFPS-0694-098, as published in 59 Fed. Reg., 35338, July 11, 1994.~~

~~(J) Rupprecht & Patashnick Partisol FRM Model 2000 PM10 Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-126, as published in 63 Fed. Reg., 69625, December 17, 1998.~~

~~(K) Rupprecht & Patashnick Partisol Plus Model 2025 PM10 Sequential Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-127, as published in 63 Fed. Reg., 69625, December 17, 1998.~~

~~(L) Tisch Environmental Model TE-6070 PM10 High-Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-0202-141, as published in 67 Fed. Reg., 15566, April 2, 2002.~~

~~(2) Continuous samplers:~~

~~(A) Andersen Beta Attenuation Monitor Model FH 62 C14 equipped with the following components: louvered PM10 inlet, volumetric flow controller, automatic filter change mechanism, automatic zero check, and calibration control foils kit*.~~

~~(B) Met One Beta Attenuation Monitor Model 1020 equipped with the following components: louvered PM10 size selective inlet, volumetric flow controller, automatic filter change mechanism, automatic heating system, automatic zero and span check capability*.~~

~~(C) Rupprecht & Patashnick Series 8500 Filter Dynamics Measurement System equipped with the following components: louvered PM10 size selective inlet, volumetric flow control, flow splitter (3 liter/min sample flow), sample equilibration system (SES) dryer, TEOM sensor unit, TEOM control unit, switching valve, purge filter conditioning unit, and palliflex TX40, 13 mm effective diameter cartridge*.~~

b) PM2.5 Methods. The method for determining compliance with the PM2.5 ambient air quality standard shall be the Federal Reference Method for the Determination of Particulate Matter as PM2.5 in the Atmosphere, 40 CFR, Chapter 1, part 50, Appendix L, as published in 62 Fed. Reg., 38714, July 18, 1997 and as amended in 64 Fed. Reg., 19717, April 22, 1999. The samplers listed in the Federal Reference Method must use either the WINS impactor or the U.S. EPA-approved very sharp cut cyclone (67 Fed. Reg., 15566, April 2, 2002) to separate PM2.5 from PM10. California Approved Samplers for PM2.5 are set forth in "Air Monitoring Quality Assurance Manual Volume IV, Part B: Monitoring

Methods for PM2.5", adopted [insert date], which is incorporated by reference herein. Samplers, methods, or instruments determined in writing by the Air Resources Board or the Executive Officer to produce equivalent results for PM2.5 shall also be California Approved Samplers for PM2.5. These include those continuous samplers that have been demonstrated to the satisfaction of the Air Resources Board to produce measurements equivalent to the Federal Reference Method. ~~The following samplers, methods, and instruments are California Approved Samplers for PM2.5 for the purposes of monitoring for compliance with the Fine Particulate Matter (PM2.5) standards:~~

~~(1) The specific samplers approved are:~~

~~(A) Andersen Model RAAS 2.5-200 PM2.5 Ambient Audit Air Sampler, U.S. EPA Manual Reference Method RFPS-0299-128, as published in 64 Fed. Reg., 12167, March 11, 1999.~~

~~(B) Graseby Andersen Model RAAS 2.5-100 PM2.5 Ambient Air Sampler, U.S. EPA Manual Reference Method RFPS-0598-119, as published in 63 Fed. Reg., 31991, June 11, 1998.~~

~~(C) Graseby Andersen Model RAAS 2.5-300 PM2.5 Sequential Ambient Air Sampler, U.S. EPA Manual Reference Method RFPS-0598-120, as published in 63 Fed. Reg., 31991, June 11, 1998.~~

~~(D) BGI Inc. Models PQ200 and PQ200A PM2.5 Ambient Fine Particle Sampler, U.S. EPA Manual Reference Method RFPS-0498-116, as published in 63 Fed. Reg., 18911, April 16, 1998.~~

~~(E) Rupprecht & Patashnick Partisol FRM Model 2000 Air Sampler, U.S. EPA Manual Reference Method RFPS-0498-117, as published in 63 Fed. Reg., 18911, April 16, 1998.~~

~~(F) Rupprecht & Patashnick Partisol Model 2000 PM-2.5 Audit Sampler, as described in U.S. EPA Manual Reference Method RFPS-0499-129, as published in 64 Fed. Reg., 19153, April 19, 1999.~~

~~(G) Rupprecht & Patashnick Partisol Plus Model 2025 PM-2.5 Sequential Air Sampler, U.S. EPA Manual Reference Method RFPS-0498-118, as published in 63 Fed. Reg., 18911, April 16, 1998.~~

~~(H) Thermo Environmental Instruments, Incorporated Model 605 "CAPS" Sampler, U.S. EPA Manual Reference Method RFPS-1098-123, as published in 63 Fed. Reg., 58036, October 29, 1998.~~

~~(I) URG MASS100 Single PM2.5 FRM Sampler, U.S. EPA Manual Reference Method RFPS-0400-135, as published in 65 Fed. Reg., 26603, May 8, 2000.~~

~~(J) URG MASS300 Sequential PM2.5 FRM Sampler, U.S. EPA Manual Reference Method RFPS-0400-136, as published in 65 Fed. Reg., 26603, May 8, 2000.~~

~~(K) BGI Inc. Model PQ200 VSCC PM2.5 Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-142, as published in 67 Fed. Reg., 15567, April 2, 2002.~~

~~(L) BGI Inc. Model PQ200A VSCC PM2.5 Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-142, as published in 67 Fed. Reg., 15567, April 2, 2002.~~

~~(M) Rupprecht & Patashnick Partisol FRM Model 2000 PM2.5 FEM Air Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-143, as published in 67 Fed. Reg., 15567, April 2, 2002.~~

~~(N) Rupprecht & Patashnick Partisol Model 2000 PM2.5 FEM Audit Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-144, as published in 67 Fed. Reg., 15567, April 2, 2002.~~

~~(O) Rupprecht & Patashnick Partisol Plus Model 2025 PM-2.5 FEM Sequential Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-145, as published in 67 Fed. Reg., 15567, April 2, 2002.~~

~~(2) Continuous samplers:~~

~~(A) Andersen Beta Attenuation Monitor Model FH 62 C14 equipped with the following components: louvered PM10 size selective inlet, very sharp cut or~~

~~sharp cut cyclone, volumetric flow controller, automatic filter change mechanism, automatic zero check, and calibration control foils kit*.~~

~~(B) Met One Beta Attenuation Monitor Model 1020 equipped with the following components: louvered PM10 size selective inlet, very sharp cut or sharp cut cyclone, volumetric flow controller, automatic filter change mechanism, automatic heating system, and automatic zero and span check capability*.~~

~~(C) Rupprecht & Patashnick Series 8500 Filter Dynamics Measurement System equipped with the following components: louvered PM10 size selective inlet, very sharp cut or sharp cut cyclone, volumetric flow control, flow splitter (3 liter/min sample flow), sample equilibration system (SES) dryer, TEOM sensor unit, TEOM control unit, switching valve, purge filter conditioning unit, and palliflex TX40, 13 mm effective diameter cartridge*.~~

~~*Instrument shall be operated in accordance with the vendor's instrument operation manual that adheres to the principles and practices of quality control and quality assurance as specified in Volume I of the "Air Monitoring Quality Assurance Manual", as printed on April 17, 2002, and available from the California Air Resources Board, Monitoring and Laboratory Division, P.O. Box 2815, Sacramento CA 95814, incorporated by reference herein.~~

(c) Ozone Methods. The method for determining compliance with the ozone ambient air quality standard shall be the Federal Equivalent Method for the Determination of Ozone in the Atmosphere (40 CFR, part 53). California Approved Samplers for ozone are set forth in "Air Monitoring Quality Assurance Manual Volume IV, Part C: Monitoring Methods for Ozone", as adopted [insert date]. Samplers, methods, or instruments determined in writing by the Air Resources Board or the Executive Officer to produce equivalent results for ozone shall also be California Approved Samplers for ozone.

NOTE

Authority cited: Sections 39600, 39601 and 39606, Health and Safety Code. Reference: Sections 39014, 39606, 39701 and 39703(f), Health and Safety Code.

Section 70200. Table of Standards ***

Substance	Concentration and Methods*	Duration of Averaging Periods	Most Relevant Effects	Comments
Ozone	0.09 ppm**	1 hour	a. Short-term exposures: (1) ~~Pulmonary function decrements and localized lung edema in humans and animals.~~ One-hour and multi-hour exposures: lung function decrements, and symptoms of respiratory irritation such as cough, wheeze, and pain upon deep inhalation. (2) Multi-hour exposures: airway hyperreactivity and airway inflammation. ~~(2) Risk to public health implied by alterations in pulmonary morphology and host defence in animals.~~ (3) excess deaths, hospitalization, emergency room visits, asthma exacerbation, respiratory symptoms and restrictions in activity	a. The standard is intended to prevent adverse human health effects.
	0.070 ppm**	8 hour		b. The standard, when achieved, will not prevent all injury to crops and other types of vegetation, but is intended to place an acceptable upper limit on the amount of yield and economic loss, as well as on adverse environmental impacts.
	ultraviolet photometry using California Approved Sampler as set forth in section 70100.1 (c)		b. Long-term exposures: ~~Risk to public health implied by altered pulmonary morphology in animals after long-term exposures and pulmonary function decrements in chronically exposed humans.~~ Ozone can induce tissue changes in the respiratory tract, and is associated with decreased lung function and emergency room visits for asthma.	
			c. Welfare effects: (1) Yield loss in important crops and predicted economic loss to growers and consumers. (2) Injury and damage to forests ~~native plants and potential changes in species diversity and number.~~ ~~(3) Damage to rubber and elastomers and to paints, fabric, dyes, pigments, and plastics.~~	

Suspended Particulate Matter (PM10)	50 µg/m^3 PM10** 20 µg/m^3 PM10** using California Approved Sampler as set forth in section 70100.1(a)	24 hour sample 24 hour samples, annual arithmetic mean	Prevention of excess deaths, illness and restrictions in activity from short-and long-term exposures. Illness outcomes include, but are not limited to, respiratory symptoms, bronchitis, asthma exacerbation, emergency room visits and hospital admissions for cardiac and respiratory diseases. Sensitive subpopulations include children, the elderly, and individuals with pre-existing cardiopulmonary disease.	This standard applies to suspended mater as measured by PM10 sampler, which collects 50% of all particles of 10 µm aerodynamic diameter and collects a declining fraction of particles as their diameter increases, reflecting the characteristics of lung deposition.

* The list of California Approved Samplers may be obtained from the Air Resources Board, Monitoring and Laboratory Division, P.O. Box 2815, Sacramento, CA 95814. Any equivalent procedure which can be shown to the satisfaction of the Air Resources Board to give equivalent results at or near the level of the air quality standard may be used.

** These standards are violated when concentrations exceed those set forth in the body of the regulation. All other standards are violated when concentrations equal or exceed those set forth in the body of the regulation.

*** Applicable statewide unless otherwise noted.

****These standards are violated when particle concentrations cause measured light extinction values to exceed those set forth in the regulations.

NOTE

Authority cited: Sections 39600, 39601(a) and 39606, Health and Safety Code. Reference: Sections 39014, 39606, 39701 and 39703(f), Health and Safety Code; and Western Oil and Gas Ass'n v. Air Resources Bd. (1984) 37 Cal.3d 502.

HISTORY

1. Amendment filed 9-18-89; operative 10-18-89 (Register 89, No. 39). For prior history, see Register 88, No. 27.
2. Amendment filed 6-29-92; operative 7-29-92 (Register 92, No. 27).
3. Amendment filed 6-5-2003; operative 7-5-2003 (Register 2003, No. 23).

(1) The method for determining compliance with the State PM10 ambient air quality standard shall be the Federal Reference Method (FRM) for the Determination of Particulate Matter as PM10 in the Atmosphere (40 CFR, Chapter 1, part 50, Appendix M, as published in 62 Fed. Reg., 38753, July 18, 1997). When employed according to the FRM, the following are California Approved Samplers:

(A) Andersen Model RAAS10-100 PM10 Single Channel PM10 Sampler, U.S. EPA Manual Reference Method RFPS-0699-130, as published in 64 Fed. Reg., 33481, June 23, 1999.

(B) Andersen Model RAAS10-200 PM10 Single Channel PM10 Audit Sampler, U.S. EPA Manual Reference Method RFPS-0699-131, as published in 64 Fed. Reg., 33481, June 23, 1999.

(C) Andersen Model RAAS10-300 PM10 Multi Channel PM10 Sampler, U.S. EPA Manual Reference Method RFPS-0669-132, as published in 64 Fed. Reg., 33481, June 23, 1999.

(D) Sierra (currently known as Graseby) Andersen/GMW Model 1200 High-Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-1287-063, as published in 52 Fed. Reg., 45684, December 1, 1987 and in 53 Fed. Reg., 1062, January 15, 1988.

(E) Sierra (currently known as Graseby) Andersen/GMW Model 321B High-Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-1287-064, as published in 52 Fed. Reg., 45684, December 1, 1987 and in 53 Fed. Reg., 1062, January 15, 1988.

(F) Sierra (currently known as Graseby) Andersen/GMW Model 321-C High-Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-1287-065, as published in 52 Fed. Reg., 45684, December 1, 1987.

(G) BGI Incorporated Model PQ100 Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-124, as published in 63 Fed. Reg., 69624, December 17, 1998.

(H) BGI Incorporated Model PQ200 Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-125, as published in 63 Fed. Reg., 69624, December 17, 1998.

(I) Rupprecht & Patashnick Partisol Model 2000 Air Sampler, U.S. EPA Manual Reference Method RFPS-0694-098, as published in 59 Fed. Reg., 35338, July 11, 1994.

(J) Rupprecht & Patashnick Partisol-FRM Model 2000 PM10 Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-126, as published in 63 Fed. Reg., 69625, December 17, 1998.

(K) Rupprecht & Patashnick Partisol-Plus Model 2025 PM10 Sequential Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-127, as published in 63 Fed. Reg., 69625, December 17, 1998.

(L) Tisch Environmental Model TE-6070 PM10 High-Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-0202-141, as published in 67 Fed. Reg., 15566, April 2, 2002.

(2) The following continuous Californian Approved Samplers have been demonstrated to the satisfaction of the Air Resources Board to produce measurements equivalent to the FRM:

(A) Andersen Beta Attenuation Monitor Model FH 62 C14 equipped with the following components: louvered PM10 inlet, volumetric flow controller, automatic filter change mechanism, automatic zero check, and calibration control foils kit*.

(B) Met One Beta Attenuation Monitor Model 1020 equipped with the following components: louvered PM10 size selective inlet, volumetric flow controller, automatic filter change mechanism, automatic heating system, automatic zero and span check capability*.

(C) Rupprecht & Patashnick Series 8500 Filter Dynamics Measurement System equipped with the following components: louvered PM10 size selective inlet, volumetric flow control, flow splitter (3 liter/min sample flow), sample equilibration system (SES) dryer, TEOM sensor unit, TEOM control unit, switching valve, purge filter conditioning unit, and palliflex TX40, 13 mm effective diameter cartridge*.

---

*Instrument shall be operated in accordance with the vendor's instrument operation manual that adheres to the principles and practices of quality control and quality assurance as specified in Volume I of the "Air Monitoring Quality Assurance Manual", as printed on April 17, 2002, and available from the California Air Resources Board, Monitoring and Laboratory Division, P.O. Box 2815, Sacramento CA 95814, incorporated by reference herein.

(1) The method for determining compliance with the State PM2.5 ambient air quality standard shall be the Federal Reference Method (FRM) for the Determination of Particulate Matter as PM2.5 in the Atmosphere, 40 CFR, part 50, Appendix L, as published in 62 Fed. Reg., 38714, July 18, 1997 and as amended in 64 Fed. Reg., 19717, April 22, 1999. These must use either the WINS impactor or the U.S. EPA-approved very sharp cut cyclone (67 Fed. Reg., 15566, April 2, 2002) to separate PM2.5 from PM10. When employed according to the FRM, the following are California Approved Samplers:

(A) Andersen Model RAAS 2.5-200 PM2.5 Ambient Audit Air Sampler, U.S. EPA Manual Reference Method RFPS-0299-128, as published in 64 Fed. Reg., 12167, March 11, 1999.

(B) Graseby Andersen Model RAAS 2.5-100 PM2.5 Ambient Air Sampler, U.S. EPA Manual Reference Method RFPS-0598-119, as published in 63 Fed. Reg., 31991, June 11, 1998.

(C) Graseby Andersen Model RAAS 2.5-300 PM2.5 Sequential Ambient Air Sampler, U.S. EPA Manual Reference Method RFPS-0598-120, as published in 63 Fed. Reg., 31991, June 11, 1998.

(D) BGI Inc. Models PQ200 and PQ200A PM2.5 Ambient Fine Particle Sampler, U.S. EPA Manual Reference Method RFPS-0498-116, as published in 63 Fed. Reg., 18911, April 16, 1998.

(E) Rupprecht & Patashnick Partisol-FRM Model 2000 Air Sampler, U.S. EPA Manual Reference Method RFPS-0498-117, as published in 63 Fed. Reg., 18911, April 16, 1998.

(F) Rupprecht & Patashnick Partisol Model 2000 PM-2.5 Audit Sampler, as described in U.S. EPA Manual Reference Method RFPS-0499-129, as published in 64 Fed. Reg., 19153, April 19, 1999.

(G) Rupprecht & Patashnick Partisol-Plus Model 2025 PM-2.5 Sequential Air Sampler, U.S. EPA Manual Reference Method RFPS-0498-118, as published in 63 Fed. Reg., 18911, April 16, 1998.

(H) Thermo Environmental Instruments, Incorporated Model 605 "CAPS" Sampler, U.S. EPA Manual Reference Method RFPS-1098-123, as published in 63 Fed. Reg., 58036, October 29, 1998.

(I) URG-MASS100 Single PM2.5 FRM Sampler, U.S. EPA Manual Reference Method RFPS-0400-135, as published in 65 Fed. Reg., 26603, May 8, 2000.

(J) URG-MASS300 Sequential PM2.5 FRM Sampler, U.S. EPA Manual Reference Method RFPS-0400-136, as published in 65 Fed. Reg., 26603, May 8, 2000.

(K) BGI Inc. Model PQ200-VSCC PM2.5 Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-142, as published in 67 Fed. Reg., 15567, April 2, 2002.

(L) BGI Inc. Model PQ200A-VSCC PM2.5 Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-142, as published in 67 Fed. Reg., 15567, April 2, 2002.

(M) Rupprecht & Patashnick Partisol-FRM Model 2000 PM2.5 FEM Air Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-143, as published in 67 Fed. Reg., 15567, April 2, 2002.

(N) Rupprecht & Patashnick Partisol Model 2000 PM2.5 FEM Audit Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-144, as published in 67 Fed. Reg., 15567, April 2, 2002.

(O) Rupprecht & Patashnick Partisol-Plus Model 2025 PM-2.5 FEM Sequential Sampler, U.S. EPA Manual Equivalent Method EQPM-0202-145, as published in 67 Fed. Reg., 15567, April 2, 2002.

(2) The following continuous samplers have been demonstrated to the satisfaction of the Air Resources Board to produce measurements equivalent to the FRM:

(A) Andersen Beta Attenuation Monitor Model FH 62 C14 equipped with the following components: louvered PM10 size selective inlet, very sharp cut or sharp cut cyclone, volumetric flow controller, automatic filter change mechanism, automatic zero check, and calibration control foils kit*.

(B) Met One Beta Attenuation Monitor Model 1020 equipped with the following components: louvered PM10 size selective inlet, very sharp cut or sharp cut cyclone, volumetric flow controller, automatic filter change mechanism, automatic heating system, and automatic zero and span check capability*.

(C) Rupprecht & Patashnick Series 8500 Filter Dynamics Measurement System equipped with the following components: louvered PM10 size selective inlet, very sharp cut or sharp cut cyclone, volumetric flow control, flow splitter (3 liter/min sample flow), sample equilibration system (SES) dryer, TEOM sensor unit, TEOM control unit, switching valve, purge filter conditioning unit, and palliflex TX40, 13 mm effective diameter cartridge*.

---

*Instrument shall be operated in accordance with the vendor's instrument operation manual that adheres to the principles and practices of quality control and quality assurance as specified in Volume I of the "Air Monitoring Quality Assurance Manual", as printed on April 17, 2002, and available from the California Air Resources Board, Monitoring and Laboratory Division, P.O. Box 2815, Sacramento CA 95814, incorporated by reference herein.

Part C: Monitoring Methods for Ozone

The method for determining compliance with the State ozone ambient air quality standard shall be the Federal Equivalent Method (FEM) for the Determination of Ozone in the Atmosphere (40 CFR, part 53). The FEM (ultraviolet photometry) is considered equivalent to the Federal Reference Method (chemiluminescence) as described in 40 CFR, Chapter 1, Part 50, Appendix D as published in FR 62, 38895, July 18, 1997. When employed according to the FEM (40 CFR, part 53), the following are California Approved Samplers:

(A) Dasibi Models 1003-AH, 1003-PC, or 1003-RS Ozone Analyzers, USEPA Automated Equivalent Method EQOA-0577-019, as published in FR 42, 28571, June 03, 1977.

(B) Dasibi Models 1008-AH, 1008-PC, or 1008-RS Ozone Analyzers, USEPA Automated Equivalent Method EQOA-0383-056, as published in FR 48, 10126, March 10, 1983.

(C) DKK-TOA Corp. Model GUX-113E Ozone Analyzer, USEPA Automated Equivalent Method EQOA-0200-134, as published in FR 65, 11308, March 02, 2000.

(D) Environics Series 300 Ozone Analyzer, USEPA Automated Equivalent Method EQOA-0990-078, as published in FR 55, 38386, September 18, 1990.

(E) Environnement S.A. Model $O_3$41M UV Ozone Analyzer, USEPA Automated Equivalent Method EQOA-0895-105, as published in FR 60, 39382, August 02, 1995.

(F) Environnement S.A. Model $O_3$42M UV Ozone Analyzer, USEPA Automated Equivalent Method EQOA-0206-148, as published in FR 67, 42557, June 24, 2002.

(G) Environnement S.A. SANOA Multigas Longpath Monitoring System, USEPA Automated Equivalent Method EQOA-0400-137, as published in FR 65, 26603, May 08, 2000.

(H) Horiba Instruments Models APOA-360 and APOA-360-CE Ozone Monitor, USEPA Automated Equivalent Method EQOA-0196-112, as published in FR 61, 11404, March 20, 1996.

(I) Monitor Labs/Lear Siegler Model 8810 Ozone Analyzer, USEPA Automated Equivalent Method EQOA-0881-053, as published in FR 46, 52224, October 26, 1981.

(J) Monitor Labs/Lear Siegler Models ML9810, ML9811, or ML9812, Monitors Labs Model ML9810B, or Wedding & Associates Model 1010 Ozone Analyzers, USEPA Automated Equivalent Method EQOA-0193-091, as published in FR 58, 6964, February 03, 1993.

(K) Opsis Model AR 500 and System 300 Open Path Ambient Air Monitoring Systems for Ozone, USEPA Automated Equivalent Method EQOA-0495-103, as published in FR 60, 21518, May 02, 1995.

(L) PCI Ozone Corporation Model LC-12 Ozone Analyzer, USEPA Automated Equivalent Method EQOA-0382-055, as published in FR 47, 13572, March 31, 1982.

(M) Philips PW9771 O3 Analyzer, USEPA Automated Equivalent Method EQOA-0777-023, as published in FR 42, 38931, August 01, 1977; FR 42, 57156, November 01, 1977.

(N) Teledyne-Advanced Pollution Instrumentation, Inc. Model 400E Ozone Analyzer, Advanced Pollution Instrumentation, Inc. Model 400/400A Ozone Analyzer, USEPA Automated Equivalent Method EQOA-0992-087, as published in FR 57, 44565, September 28; 1992, FR 63, 31992, June 11, 1998; FR 67, 57811, September 12, 2002.

(O) Thermo Electron/Thermo Environmental Instruments Models 49, 49C, USEPA Automated Equivalent Method EQOA-0880-047, as published in FR 45, 57168, August 27, 1980

PROPOSITION 65 STATUS REPORT SAFE HARBOR LEVELS:

No Significant Risk Levels for Carcinogens and Maximum Allowable Dose Levels for Chemicals Causing Reproductive Toxicity

September 2003

Reproductive and Cancer Hazard Assessment Section
Office of Environmental Health Hazard Assessment
California Environmental Protection Agency

TABLE OF CONTENTS

Proposition 65 Safe Harbor Levels Development

The Office of Environmental Health Hazard Assessment (OEHHA) of the California Environmental Protection Agency is the lead agency for the implementation of the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65 or the Act). In that role, OEHHA has developed Proposition 65 safe harbor levels -- no significant risk levels (NSRLs) for carcinogens and maximum allowable dose levels (MADLs) for chemicals that cause reproductive toxicity. The NSRL is the daily intake level calculated to result in one excess case of cancer in an exposed population of 100,000, assuming lifetime (70-year) exposure at the level in question. The MADL is the level at which the chemical would have no observable adverse reproductive effect assuming exposure at 1,000 times that level. The NSRLs and MADLs are promulgated in Title 22, California Code of Regulations, (CCR) Sections 12705 and 12805 respectively to assist interested parties in determining whether warnings are required for exposures to listed chemicals, and whether discharges to sources of drinking water are prohibited.

Safe harbor levels may be based on risk assessments conducted outside OEHHA, as provided for in 22 CCR 12705(b), 12705(c), and 12805. In some cases, this can expedite safe harbor development. However, it should be noted that the process of review and consideration of existing risk assessments can be a lengthy one, and will depend on the complexity of the scientific information underlying the assessment, as well as on available resources.

This document provides the status of the development and adoption of intake levels calculated for all chemicals on the Proposition 65 list. In units of micrograms per day (µg/day), Part A reports NSRLs adopted in regulation for carcinogens and Part B reports MADLs adopted in regulation for chemicals that cause reproductive toxicity.

Parts C and D of this document give priority levels for development of dose response assessments for chemicals that cause cancer and reproductive toxicity, respectively. Interested parties are invited to recommend changes in priority levels. OEHHA retains the right to change priorities in response to the nature and availability of scientific information, and resources available, and requests from the public and the Attorney General's office.

Parts C and D also give draft levels, some of which have been available since the early 1990's and others of which have been updated recently. OEHHA will continue to review the basis for draft numbers and update analyses as needed, before proposing or finalizing levels for formal adoption in regulation.

This status report will be updated on a regular basis.

A. No Significant Risk Levels (NSRLs) Adopted in Regulation for Carcinogens

The table below lists NSRLs for Proposition 65 carcinogens in regulation (22 CCR §12705 and §12709). These levels are intended to provide "safe harbors" for persons subject to the Act, and do not preclude the use of alternative levels that can be demonstrated by their users as being scientifically valid.

A three-tiered procedure for development of NSRLs is currently in place. NSRLs may be based on a *de novo* dose response assessment conducted or reviewed by OEHHA (22 CCR §12705(b)), an assessment conducted by another state or federal agency (22 CCR §12705(c)), or an expedited process conducted by OEHHA (22 CCR §12705(d)). The last column of the table below indicates which of these processes was used to develop the NSRL for each chemical. NSRLs represent the daily intake level calculated to result in a cancer risk of one excess case of cancer in 100,000 individuals exposed over a 70-year lifetime.

NSRLs for chemicals in underline have been adopted since the last Status Report. As chemicals are removed from the Proposition 65 list, the regulatory process to remove the safe harbor level from regulation will be initiated.

Carcinogen	Level (µg/day)	22 CCR Section
A-alpha-C (2-Amino-9H-pyrido[2,3-b]indole)	2	12705(d)
Acetaldehyde	90 (inhalation)	12705(c)
Acetamide	10	12705(d)
2-Acetylaminofluorene	0.2	12705(d)
Acrylamide	0.2	12705(c)
Acrylonitrile	0.7	12705(b)
Actinomycin D	0.00008	12705(d)
AF-2; [2-(2-furyl)-3(5-nitro-2-furyl)acrylamide]	3	12705(d)
Aldrin	0.04	12705(b)
2-Aminoanthraquinone	20	12705(d)
o-Aminoazotoluene	0.2	12705(d)
4-Aminobiphenyl	0.03	12705(d)
3-Amino-9-ethylcarbazole hydrochloride	9	12705(d)
1-Amino-2-methylanthraquinone	5	12705(d)
2-Amino-5-(5-nitro-2-furyl)-1,3,4-thiadiazole	0.04	12705(d)
Amitrole	0.7	12705(d)
Aniline	100	12705(c)
o-Anisidine	5	12705(d)
o-Anisidine hydrochloride	7	12705(d)
Aramite	20	12705(d)
Arsenic	0.06 (inh) 10 (except inh)	12705(b) 12709
Asbestos NSRL for fibers ≥ 5 micrometers (mm) long and 0.3 wide, with a length/width ratio ≥ 3:1 as measured by phase contrast microscopy.	100 fibers/day (inh)	12705(b)
Auramine	0.8	12705(d)

Carcinogen	Level (µg/day)	22 CCR Section
Azaserine	0.06	12705(d)
Azathioprine	0.4	12705(d)
Azobenzene	6	12705(c)
Benzene	7	12705(b)
Benzidine	0.001	12705(b)
Benzofuran	1.1	12705(b)
Benzo[a]pyrene	0.06	12705(c)
Benzyl chloride	4	12705(c)
Benzyl violet 4B	30	12705(d)
Beryllium	0.1	12709
Beryllium oxide	0.1	12705(c)
Beryllium sulfate	0.0002	12705(c)
Bis(2-chloroethyl)ether	0.3	12705(b)
Bis(chloromethyl)ether	0.02	12705(b)
Bromodichloromethane	5	12705(c)
1,3-Butadiene	0.4	12705(c)
Butylated hydroxyanisole	4000	12705(b)
beta-Butyrolactone	0.7	12705(d)
Cadmium	0.05 (inh)	12705(b)
Captafol	5	12705(d)
Captan	300	12705(d)
Carbazole	4.1	12705(d)
Carbon tetrachloride	5	12705(b)
N-Carboxymethyl-N-nitrosourea	0.70	12705(b)
Chlorambucil	0.002	12705(d)
Chlordane	0.5	12705(c)
Chlordecone (Kepone)	0.04	12705(d)
Chlorendic acid	8	12705(d)
Chlorinated paraffins (Ave. chain length C12; approx. 60% chlorine by weight)	8	12705(d)
Chloroethane (Ethyl chloride)	150	12705(b)
Chloroform	20 (oral)	12705(c)
	40 (inh)	12705(c)
Chloromethyl methyl ether (technical grade)	0.3	12705(d)
3-Chloro-2-methylpropene	5	12705(d)
4-Chloro-ortho-phenylenediamine	40	12705(d)
Chlorothalonil	200	12705(d)
p-Chloro-ortho-toluidine	3	12705(d)
p-Chloro-*o*-toluidine, hydrochloride	3.3	12705(d)
Chlorozotocin	0.003	12705(d)
Chromium (hexavalent)	0.001 (inh)	12705(b)
C.I. Basic Red 9 monohydrochloride	3	12705(d)
Cinnamyl anthranilate	200	12705(d)
Coke oven emissions	0.3	12705(c)
p-Cresidine	5	12705(d)
Cupferron	3	12705(d)
Cyclophosphamide (anhydrous)	1	12705(d)
Cyclophosphamide (hydrated)	1	12705(d)

Carcinogen	Level (µg/day)	22 CCR Section
D&C Red No. 9	100	12705(d)
Dacarbazine	0.01	12705(d)
Daminozide	40	12705(d)
Dantron (Chrysazin; 1,8-Dihydroxyanthraquinone)	9	12705(d)
DDT, DDE, DDD (in combination)	2	12705(b)
DDVP (Dichlorvos)	2	12705(c)
2,4-Diaminoanisole	30	12705(d)
2,4-Diaminoanisole sulfate	50	12705(d)
4,4'-Diaminodiphenyl ether (4,4'-Oxydianiline)	5	12705(d)
2,4-Diaminotoluene	0.2	12705(d)
Dibenz[a,h]anthracene	0.2	12705(d)
1,2-Dibromo-3-chloropropane	0.1	12705(b)
p-Dichlorobenzene	20	12705(b)
3,3'-Dichlorobenzidine	0.6	12705(b)
1,1-Dichloroethane	100	12705(d)
1,2-Dichloroethane (Ethylene dichloride)	10	12705(b)
Dichloromethane (Methylene chloride)	200 (inh)	12705(b)
	50	12705(c)
Dieldrin	0.04	12705(b)
Di(2-ethylhexyl)phthalate (DEHP)	310	12705(b)
Diethylstilbesterol	0.002	12705(d)
Diglycidyl resorcinol ether (DGRE)	0.4	12705(d)
Dihydrosafrole	20	12705(d)
3,3'-Dimethoxybenzidine (o-Dianisidine)	0.15	12705(b)
3,3'-Dimethoxybenzidine dihydrochloride	0.19	12705(b)
4-Dimethylaminoazobenzene	0.2	12705(d)
trans-2-[(Dimethylamino)methylimino]-5-[2-(5-nitro-2-furyl)vinyl]-1,3,4-oxadiazole	2	12705(d)
7,12-Dimethylbenz(a)anthracene	0.003	12705(d)
3,3'-Dimethylbenzidine (o-Toluidine)	0.044	12705(b)
3,3'-Dimethylbenzidine dihydrochloride	0.059	12705(b)
Dimethylcarbamoyl chloride	0.05	12705(d)
1,2-Dimethylhydrazine	0.001	12705(d)
Dimethylvinylchloride	20	12705(d)
2,4-Dinitrotoluene	2	12705(c)
1,4-Dioxane	30	12705(b)
Direct Black 38 (technical grade)	0.09	12705(d)
Direct Blue 6 (technical grade)	0.09	12705(d)
Direct Brown 95 (technical grade)	0.1	12705(d)
Disperse Blue 1	200	12705(d)
Epichlorohydrin	9	12705(b)
Estradiol 17b	0.02	12705(d)
Ethyl-4,4'-dichlorobenzilate (Chlorobenzilate)	7	12705(d)
Ethylene dibromide	0.2 (oral)	12705(b)
	3 (inh)	12705(b)
Ethylene oxide	2	12705(b)
Ethylene thiourea	20	12705(d)
Ethyleneimine	0.01	12705(d)
Folpet	200	12705(c)

Carcinogen	Level (µg/day)	22 CCR Section
Formaldehyde (gas)	40	12705(c)
2-(2-Formylhydrazino)-4-(5-nitro-2-furyl)thiazole	0.3	12705(d)
Furmecyclox	20	12705(c)
Glu-P-1 (2-Amino-6-methyldipyrido[1,2-a:3',2'-d]imidazole)	0.1	12705(d)
Glu-P-2 (2-Aminodipyrido[1,2-a:3',2'-d]-imidazole)	0.5	12705(d)
Gyromitrin (Acetaldehyde methylformylhydrazone)	0.07	12705(d)
HC Blue 1	10	12705(d)
Heptachlor	0.2	12705(c)
Heptachlor epoxide	0.08	12705(c)
Hexachlorobenzene	0.4	12705(b)
Hexachlorocyclohexane		
alpha isomer	0.3	12705(c)
beta isomer	0.5	12705(c)
gamma isomer	0.6	12705(c)
technical grade	0.2	12705(b)
Hexachlorodibenzodioxin	0.0002	12705(b)
Hexachloroethane	20	12705(d)
Hydrazine	0.04	12705(c)
Hydrazine sulfate	0.2	12705(c)
Hydrazobenzene (1,2-Diphenylhydrazine)	0.8	12705(d)
IQ (2-Amino-3-methylimidazo[4,5-f]quinoline)	0.5	12705(d)
Isobutyl nitrite	7.4	12705(d)
Lasiocarpine	0.09	12705(d)
Lead	15 (oral)	12705(b)
Lead acetate	23 (oral)	12705(b)
Lead phosphate	58 (oral)	12705(b)
Lead subacetate	41 (oral)	12705(b)
Me-A-alpha-C (2-Amino-3-methyl-9H-pyrido[2,3-b]indole)	0.6	12705(d)
MeIQ (2-amino-3,4-dimethylimidazo-[4,5-*f*]quinoline)	0.46	12705(d)
MeIQx (2-Amino-3,8-dimethylimidazo[4,5-*f*]quinoxaline)	0.41	12705(d)
Melphalan	0.005	12705(d)
2-Methylaziridine (Propyleneimine)	0.028	12705(b)
Methyl carbamate	160	12705(d)
3-Methylcholanthrene	0.03	12705(d)
4,4'-Methylene bis(2-chloroaniline)	0.5	12705(d)
4,4'-Methylene bis(N,N-dimethyl)benzeneamine	20	12705(c)
4,4'-Methylene bis(2-methylaniline)	0.8	12705(d)
4,4'-Methylenedianiline	0.4	12705(d)
4,4'-Methylenedianiline dihydrochloride	0.6	12705(d)
Methylhydrazine	0.058 (oral)	12705(b)
	0.090 (inhalation)	12705(b)
Methylhydrazine sulfate	0.18	12705(b)
Methyl methanesulfonate	7	12705(d)
2-Methyl-1-nitroanthraquinone (of uncertain purity)	0.2	12705(d)
N-Methyl-N'-nitro-N-nitrosoguanidine	0.08	12705(d)
Methylthiouracil	2	12705(d)

Carcinogen	Level (µg/day)	22 CCR Section
Michler's ketone	0.8	12705(d)
Mirex	0.04	12705(d)
Mitomycin C	0.00009	12705(d)
Monocrotaline	0.07	12705(d)
5-(Morpholinomethyl)-3-[(5-nitrofurfurylidene)-amino] -2-oxazolidinone	0.18	12705(b)
MX (3-chloro-4-(dichloromethyl)-5-hydroxy-2(*5H*)-furanone)	0.11	12705(b)
Nalidixic acid	28	12705(d)
2-Naphthylamine	0.4	12705(d)
Nickel refinery dust	0.8	12705(c)
Nickel subsulfide	0.4	12705(c)
Nitrilotriacetic acid	100	12705(d)
Nitrilotriacetic acid, trisodium salt monohydrate	70	12705(d)
5-Nitroacenaphthene	6	12705(d)
5-Nitro-o-anisidine	10	12705(d)
Nitrofen (technical grade)	9	12705(d)
Nitrofurazone	0.5	12705(d)
1-[(5-Nitrofurfurylidene)-amino]-2-imidazolidinone	0.4	12705(d)
N-[4-(5-Nitro-2-furyl)-2-thiazolyl]acetamide	0.5	12705(d)
N-Nitrosodi-n-butylamine	0.06	12705(b)
N-Nitrosodiethanolamine	0.3	12705(c)
N-Nitrosodiethylamine	0.02	12705(b)
N-Nitrosodimethylamine	0.04	12705(b)
p-Nitrosodiphenylamine	30	12705(d)
N-Nitrosodiphenylamine	80	12705(b)
N-Nitrosodi-n-propylamine	0.1	12705(b)
N-Nitroso-N-ethylurea	0.03	12705(b)
4-(N-Nitrosomethylamino)-1-(3-pyridyl)-1-butanone	0.014	12705(d)
N-Nitrosomethylethylamine	0.03	12705(c)
N-Nitroso-N-methylurea	0.006	12705(b)
N-Nitroso-N-methylurethane	0.006	12705(d)
N-Nitrosomorpholine	0.1	12705(d)
N-Nitrosonornicotine	0.5	12705(d)
N-Nitrosopiperidine	0.07	12705(d)
N-Nitrosopyrrolidine	0.3	12705(c)
Pentachlorophenol	40	12705(c)
Phenacetin	300	12705(d)
Phenazopyridine	4	12705(d)
Phenazopyridine hydrochloride	5	12705(d)
Phenesterin	0.005	12705(d)
Phenobarbital	2	12705(d)
Phenoxybenzamine	0.2	12705(d)
Phenoxybenzamine hydrochloride	0.3	12705(d)
o-Phenylenediamine	26	12705(d)
o-Phenylenediamine dihydrochloride	44	12705(d)
Phenyl glycidyl ether	5.0	12705(b)
Phenylhydrazine	1.0	12705(b)
Phenylhydrazine hydrochloride	1.4	12705(b)
o-Phenylphenate, sodium	200	12705(d)

Carcinogen	Level (µg/day)	22 CCR Section
Polybrominated biphenyls	0.02	12705(b)
Polychlorinated biphenyls	0.09	12705(c)
Polygeenan	1200	12705(b)
Ponceau MX	200	12705(d)
Ponceau 3R	40	12705(d)
Potassium bromate	1	12705(d)
Procarbazine	0.05	12705(d)
Procarbazine hydrochloride	0.06	12705(d)
1,3-Propane sultone	0.3	12705(d)
beta-Propiolactone	0.05	12705(d)
Propylthiouracil	0.7	12705(d)
Reserpine	0.06	12705(d)
Safrole	3	12705(d)
Sterigmatocystin	0.02	12705(d)
Streptozotocin	0.006	12705(d)
Styrene oxide	4	12705(d)
Sulfallate	4	12705(d)
Tetrachlorodibenzo-p-dioxin	0.000005	12705(b)
1,1,2,2-Tetrachloroethane	3	12705(d)
Tetrachloroethylene	14	12705(c)
Tetranitromethane	0.059	12705(b)
Thioacetamide	0.1	12705(d)
4,4'-Thiodianiline	0.05	12705(d)
Thiourea	10	12705(d)
Tolnene diisocyanate	20	12705(d)
ortho-Toluidine	4	12705(d)
ortho-Toluidine hydrochloride	5	12705(d)
Toxaphene	0.6	12705(b)
Trichloroethylene	50 (oral)	12705(b)
	80 (inh)	12705(b)
2,4,6-Trichlorophenol	10	12705(b)
Trimethyl phosphate	24	12705(d)
Tris(1-aziridinyl)phosphine sulfide (Thiotepa)	0.06	12705(d)
Tris(2,3-dibromopropyl)phosphate	0.3	12705(d)
Trp-P-1 (Tryptophan-P-1)	0.03	12705(d)
Trp-P-2 (Tryptophan-P-2)	0.2	12705(d)
Urethane (Ethyl carbamate)	0.7	12705(b)
Vinyl chloride	3	12705(b)
Vinyl trichloride (1,1,2-Trichloroethane)	10	12705(d)
2,6-Xylidine	110	12705(b)

B. Maximum Allowable Dose Levels (MADLs) Adopted in Regulation for Chemicals Causing Reproductive Toxicity

The following table is a compilation of MADLs in regulation (22 CCR §12805) for Proposition 65 chemicals that cause reproductive toxicity. These levels represent the no observable effect level (NOEL) for the chemical, divided by 1,000. NOELs are set in accordance with procedures specified in 22 CCR §12803. MADLs for chemicals in underline have been adopted since the last Status Report.

Chemical Listed as Causing Reproductive Toxicity	Level (µg/day)
Benzene	24 (oral) 49 (inhalation)
Cadmium	4.1 (oral)
2,4-DB (2,4-dichlorophenoxybutyric acid)	910
m-Dinitrobenzene	38
Ethylene oxide	20
Hydramethylnon	120 (oral)
Lead	0.5
Linuron	460
N-Methylpyrrolidone	3200 (inhalation) 17000(dermal)
Quizalofop-ethyl	590
Toluene	7000[a]

[a] Level represents absorbed dose (rounded from 6,525 µg/day). Since 100% of ingested toluene is absorbed, oral dose is equivalent to administered dose. It is assumed that roughly 50% of the dose administered by the inhalation route is absorbed. Therefore the MADL for inhaled toluene is 13,000 µg/day (rounded from 13,050 µg/day), corresponding to an absorbed dose of 6,525 µg/day.

C. Priority List for the Development of NSRLs for Proposition 65 Carcinogens

OEHHA has developed the following priority list, which classifies into four priorities carcinogens for which dose-response assessments have not been completed. Priority levels reflect the availability and quality of scientific data for dose-response assessments, potential for exposure, resources available to perform the assessment, commitments made in settlement of the case of AFL-CIO v. Deukmejian (Sacramento Superior Court No. 3481295) and input from the public and Attorney General's office. OEHHA anticipates proposing NSRLs for the majority of chemicals in the first priority group within the next two years, and for second priority chemicals within the next two to four years. It is unlikely that NSRLs for third and fourth priority chemicals would be released within the next three years.

Any interested party may submit recommendations to OEHHA for revising the priority assignment for any of the chemicals listed. Recommendations should be accompanied by appropriate documentation supporting the alternative priority assignment suggested. OEHHA expects changes in priorities resulting from the availability of scientific information and resources, and requests from the public and Attorney General's office.

A three-tiered procedure for development of NSRLs is currently in place. NSRLs may be based on a *de novo* dose response assessment conducted or reviewed by OEHHA (22 CCR §12705(b)), an assessment conducted by another state or federal agency (22 CCR §12705(c)), or an expedited process conducted by OEHHA (22 CCR §12705(d)). The table below lists draft NSRLs and their year of release, along with the subsection of 12705 indicating the procedure used to develop the value. OEHHA will review the basis for draft numbers and update analyses as needed, before proposing or finalizing levels for formal adoption in regulation. Chemicals in bold font have been added to the Proposition 65 list or changed in priority status since the last Status Report.

---

1. First Priority for NSRL Development

Acetochlor	(1992 draft NSRL: 70 µg/day [12705(c)])
Acifluorfen	(1992 draft NSRL: 20 µg/day [12705(c)])
Alachlor	(1992 draft NSRL: 9 µg/day [12705(c)])
1-Amino-2,4-dibromoanthraquinone	
Aniline hydrochloride	
Antimony oxide	
Azacitidine	
Benz[a]anthracene	(2003 draft oral NSRL: 0.033 µg/day [12705(b)])
Benzo[b]fluoranthene	(2003 draft oral NSRL: 0.096 µg/day [12705(b)])
Benzo[j]fluoranthene	(2003 draft oral NSRL: 0.11 µg/day [12705(b)])
Benzo[k]fluoranthene	
Benzotrichloride	(1993 draft oral NSRL: 0.05 µg/day [12705(c)]) (1993 draft NSRL: 0.0002 µg/day [12705(b)])
2,2-Bis(bromomethyl)-1,3-propanediol	
Bromate	
Bromoform	(2003 draft NSRL: 64 µg/day [12705(b)])
Chlordimeform	(1992 draft NSRL: 0.5 µg/day [12705(c)])
p-Chloroaniline	
p-Chloroaniline hydrochloride	
Chrysene	(2003 draft oral NSRL: 0.35 µg/day [12705(b)])
C. I. Acid Red 114	
C.I. Direct Blue 15	
C.I. Direct Blue 218	

C.I. Solvent Yellow 14	
Dibenz[a,h]acridine	
Dibenz[a,j]acridine	
7H-Dibenzo[c,g]carbazole	(2003 draft oral NSRL: 0.0030 µg/day [12705(b)])
Dibenzo[a,e]pyrene	
Dibenzo[a,h]pyrene	(2003 draft oral NSRL: 0.0054 µg/day [12705(b)])
Dibenzo[a,i]pyrene	(2003 draft oral NSRL: 0.0050 µg/day [12705(b)])
Dibenzo[a,l]pyrene	
3,3'-Dichlorobenzidine dihydrochloride	
1,2-Dichloropropane	
1,3-Dichloropropene	(1993 draft oral NSRL: 4 µg/day [12705(b)]) (1993 draft inhalation NSRL: 20 µg/day [12705(c)])
Diepoxybutane	
Diethyl sulfate	(1993 draft NSRL: 0.7 µg/day [12705(b)])
Dimethyl sulfate	(1993 draft NSRL: 0.05 µg/day [12705(b)])
1,1-Dimethylhydrazine (UDMH)	(1992 draft NSRL: 0.3 µg/day [12705(b)])
1,6-Dinitropyrene	(1993 draft NSRL: 0.02 µg/day [12705(b)])
1,8-Dinitropyrene	(1993 draft NSRL: 0.01 µg/day [12705(b)])
2,6-Dinitrotoluene	
Estragole	
Ethinylestradiol	
Furan	
Glycidol	(1992 draft NSRL: 0.4 µg/day [12705(b)])
Griseofulvin	(1992 draft NSRL: 50 µg/day [12705(b)])
Hexamethylphosphoramide	(1992 draft NSRL: 0.01 µg/day [12705(b)])
Indeno[1,2,3-cd]pyrene	
Isoprene	
Lactofen	(1992 draft NSRL: 4 µg/day [12705(c)])
5-Methylchrysene	(2003 draft oral NSRL: 0.0084 µg/day [12705(b)])
Methyleugenol	
Methylmercury compounds*	
N-Methylolacrylamide	(1992 draft NSRL: 2 µg/day [12705(b)])
Metronidazole	(1992 draft NSRL: 4 µg/day [12705(b)])
Nafenopin	
Naphthalene	
Nickel carbonyl	
o-Nitroanisole	
Nitrobenzene	
4-Nitrobiphenyl	
6-Nitrochrysene	(1993 draft NSRL: 0.002 µg/day [12705(b)])
2-Nitrofluorene	(1993 draft NSRL: 0.09 µg/day [12705(b)])
2-Nitropropane	(1993 draft inhalation NSRL: 30 µg/day [12705(b)])
1-Nitropyrene	(1993 draft NSRL: 0.6 µg/day [12705(b)])
4-Nitropyrene	(1993 draft NSRL: 0.03 µg/day [12705(b)])
N-Nitrosomethylvinylamine	(1993 draft NSRL: 0.004 µg/day [12705(b)])
N-Nitrososarcosine	(1993 draft NSRL: 5 µg/day [12705(b)])
Ochratoxin A	(1992 draft NSRL: 0.03 µg/day [12705(b)])
Oxazepam	
o-Phenylphenol	
PhiP	
Progesterone	
Pronamide	

* For explanation of priority levels see discussion above.

Pyridine
Selenium sulfide
1,2,3-Trichloropropane
Tris(2-chloroethyl)phosphate
Vinyl bromide (1992 draft oral NSRL: 1 µg/day [12705(b)])
(1992 draft inhalation NSRL: 4 µg/day [12705(b)])
4-Vinylcyclohexene

It is anticipated that changes to NSRLs currently in regulation will be proposed or adopted during the next year for the following chemicals:

Acrylamide
Benzene (2003 draft oral NSRL: 6.4 µg/day [12705 (b)])
(2003 draft inhalation NSRL: 13 µg/day [12705 (b)])
Chromium (VI)
Ethylene thiourea
o-Phenylphenate, sodium
Pentachlorophenol
Safrole
Tetrachloroethylene

2. Second Priority for NSRL Development

Aflatoxins (1992 draft NSRL: 0.02 µg/day [12705(b)])
p-Aminoazobenzene
Bis(2-chloro-1-methylethyl)ether, technical grade
Bromoethane
Cacodylic acid
Catechol
Ceramic fibers (airborne particles of respirable size)
1-Chloro-4-nitrobenzene
Chloroprene
5-Chloro-o-toluidine and its strong acid salts
Cobalt metal powder
Cobalt [II] oxide
Cobalt sulfate heptahydrate
Diaminotoluene (mixed)
2,3-Dibromo-1-propanol
Dichloroacetic acid
1,4-Dichloro-2-butene
Diesel engine exhaust
Di-n-propyl isocinchomeronate (MGK Repellent 326)
Diuron
Ethoprop
Fenoxycarb
Indium phosphide
Iprodione
Isoxaflutole
Isosafrole
Metham sodium
Methyl iodide
1-Naphthylamine
Nickel and certain nickel compounds
Nitromethane

o-Nitrotoluene
Oxadiazon
Oxythioquinox
Polychlorinated dibenzo-p-dioxins
Primidone
Propachlor
Quinoline and its strong acid salts
Radionuclides
Salicylazosulfapyridine
Silica, crystalline (airborne particles of respirable size)
Testosterone and its esters
p-a,a,a-Tetrachlorotoluene
Tetrafluoroethylene
2,4,5-Trimethylaniline and its strong acid salts
Triphenyltin hydroxide
Trypan blue (commercial grade)
4-Vinyl-1-cyclohexene diepoxide

3. Third Priority for NSRL Development

Adriamycin (Doxorubicin hydrochloride)
Benzidine-based dyes
N,N-Bis(2-chloroethyl)-2-naphthylamine
Bischloroethyl nitrosourea (BCNU) (Carmustine)
1,4-Butanediol dimethanesulfonate (Busulfan)
Carbon black (airborne, unbound particles of respirable size)
Chloramphenicol
1-(2-Chloroethyl)-3-cyclohexyl-1-nitrosourea (CCNU)
1-(2-Chloroethyl)-3-(4-methylcyclohexyl)-1-nitrosourea
Chlorotrianisene
Ciclosporin (Cyclosporin A; Cyclosporine)
Cidofovir
Cisplatin
Clofibrate
Daunomycin
N,N'-Diacetylbenzidine
3,3'-Dichloro-4,4'-diaminodiphenyl ether
Dienestrol
1,2-Diethylhydrazine
Diisopropyl sulfate
2,4-/2,6-Dinitrotoluene mixture
Diphenylhydantoin (Phenytoin)
Diphenylhydantoin (Phenytoin), sodium salt
Estrone
Estropipate
Ethyl acrylate
Furazolidone
Fusarin C
Ganciclovir sodium
Gasoline engine exhaust (condensates/extracts)
Gemfibrozil
Glasswool fibers (airborne particles of respirable size)
Glycidaldehyde
Mancozeb

Maneb
Medroxyprogesterone acetate
Merphalan
Mestranol
Metiram
Mustard Gas
Niridazole
Nitrogen mustard (Mechlorethamine)
Nitrogen mustard hydrochloride (Mechlorethamine HCl)
Norethisterone (Norethindrone)
Oxymetholone
Panfuran S
Polychlorinated dibenzofurans
Procymidone
Propargite
Propylene oxide (1991 draft oral NSRL: 3 µg/day [12705(c)])
(1991 draft inhalation NSRL: 60 µg/day [12705(c)])
Spironolactone
Stanozolol
Strong inorganic acid mists containing sulfuric acid
Tamoxifen and its salts
Terrazole
Thiodicarb
Thorium dioxide
Treosulfan
Trichlormethine (Trimustine hydrochloride)
Uracil mustard
Vinclozolin
Vinyl fluoride
Zileuton

4. Fourth Priority for NSRL Development

Alcoholic beverages
2-Aminofluorene
4-Amino-2-nitrophenol
Analgesic mixtures containing phenacetin
Betel quid with tobacco
Bitumens, extracts of steam-refined
Bracken fern
Caffeic acid
Carbon-black extracts
Certain combined chemotherapy for lymphomas
Citrus Red No. 2
Conjugated estrogens
Creosotes
Cycasin
Cytembena
D&C Orange No. 17
D&C Red No. 8
D&C Red No. 19
3,7-Dinitrofluoranthene
3,9-Dinitrofluoranthene
Erionite

Ethyl methanesulfonate
Iron dextran complex
Lynestrenol
8-Methoxypsoralen with ultraviolet A therapy
5-Methoxypsoralen with ultraviolet A therapy
Methylazoxymethanol
Methylazoxymethanol acetate
Nitrogen mustard N-oxide
Nitrogen mustard N-oxide hydrochloride
3-(N-Nitrosomethylamino)propionitrile
Norethynodrel
Oil Orange SS
Oral contraceptives, combined
Oral contraceptives, sequential
Palygorskite fibers
Phenolphthalein
Residual (heavy) fuel oils
Shale-oils
Soots, tars, and mineral oils
Talc containing asbestiform fibers
Tobacco, oral use of smokeless products
Tobacco smoke
Tris(aziridinyl)-para-benzoquinone (Triaziquone)
Unleaded gasoline (wholly vaporized)

D. Priority List for the Development of MADLs for Chemicals Causing Reproductive Toxicity

OEHHA has developed the following priority list, which divides chemicals causing reproductive toxicity for which dose-response assessments have not been completed into three priorities. Priority levels reflect the availability and quality of scientific data for dose-response assessments, potential for exposure, resources available to perform the assessment, and input from the public and the Attorney General's office. OEHHA anticipates proposing MADLs for the majority of chemicals in the first priority group within the next two years, and for several chemicals in the second priority within the next two to four years. It is unlikely that MADLs for chemicals in the third priority group would be released within the next three years.

Any interested party may submit recommendations to OEHHA on revising the priority assignment for any of the chemicals listed. Recommendations should be accompanied by appropriate documentation supporting the alternative priority assignment suggested. OEHHA expects changes in priorities resulting from the availability of scientific information and resources and requests from the public and Attorney General's office.

Also given below are draft levels available and year of release. OEHHA will review the basis for draft numbers and update analyses as needed, before proposing or finalizing levels for formal adoption in regulation. Chemicals in bold font have been added to the Proposition 65 list or changed in priority status since the last Status Report.

1. First Priority for MADL Development

Arsenic (inorganic oxides)	(2003 draft oral MADL: 0.10 µg/day)
Carbon disulfide	(1994 draft oral MADL: 600 µg/day) (1994 draft inhalation MADL: 1000 µg/day)
1,2-Dibromo-3-chloropropane (DBCP)	(1994 draft MADL: 5 µg/day)
Ethylene glycol monoethyl ether	
Ethylene glycol monomethyl ether	
Ethylene glycol monoethyl ether acetate	
Ethylene glycol monomethyl ether acetate	
Mercury and mercury compounds*	
Metham sodium	
Methyl bromide as a structural fumigant	(1994 draft MADL: 1000 µg/day)
Methyl mercury*	(1994 draft MADL: 0.3 µg/day)
Nicotine	
Thiophanate-methyl	
Triphenyl tin hydroxide	
Vinclozolin	

2. Second Priority for MADL Development

Amitraz
Bromacil lithium salt
Bromoxynil
Bromoxynil octanoate
Chinomethionat (Oxythioquinox)
Chlorsulfuron
Cocaine

* For explanation of priority levels see discussion above.

Methyltestosterone
Midazolam hydrochloride
Minocycline hydrochloride (internal use)
Misoprostol
Mitoxantrone hydrochloride
Nafarelin acetate
Neomycin sulfate (internal use)
Netilmicin sulfate
Nickel carbonyl
Nifedipine
Nimodipine
Nitrofurantoin
Nitrogen mustard (Mechlorethamine)
Nitrogen mustard hydrochloride (Mechlorethamine hydrochloride)
Norethisterone (Norethindrone)
Norethisterone acetate (Norethindrone acetate)
Norethisterone (Norethindrone)/Ethinyl estradiol
Norethisterone (Norethindrone)/Mestranol
Norgestrel
Oxazepam
Oxymetholone
Oxytetracycline (internal use)
Oxytetracycline hydrochloride (internal use)
Paclitaxel
Paramethadione
Penicillamine
Pentobarbital sodium
Pentostatin
Phenacemide
Phenprocoumon
Pimozide
Pipobroman
Plicamycin
Polybrominated biphenyls
Polychlorinated biphenyls
Pravastatin sodium
Prednisolone sodium phosphate
Procarbazine hydrochloride
Propylthiouracil
Pyrimethamine
Quazepam
Retinol/retinyl esters, when in daily dosages in
excess of 10,000 IU, or 3,000 retinol equivalents.
Ribavirin
Rifampin
Secobarbital sodium
Sermorelin acetate
Streptomycin sulfate
Streptozocin (streptozotocin)
Sulfasalazine
Sulindac
Tamoxifen citrate
Temazepam
Teniposide

Testosterone cypionate
Testosterone enanthate
Tetracycline (internal use)
Tetracyclines (internal use)
Tetracycline hydrochloride (internal use)
Thalidomide
Thioguanine
Tobacco smoke (primary)
Tobramycin sulfate
Triazolam
Trientine hydrochloride
Trilostane
Trimethadione
Trimetrexate glucuronate
Uracil mustard
Urethane
Urofollitropin
Valproate (Valproic acid)
Vinblastine sulfate
Vincristine sulfate
Warfarin
Zileuton

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

RELATED RESOURCES
ToxFAQ™ 112k
ToxFAQ™ en Español 26k
Public Health Statement 159k
Toxicological Profile 6.5MB
MMG 57k

A-Z INDEX
A B C D E
F G H I J K
L M N O P
Q R S T U
V W X Y Z

ATSDR RESOURCES
ToxFAQs™
ToxFAQs™ en Español
Public Health Statements
Toxicological Profiles
Minimum Risk Levels
MMGs
MHMIs
Interaction Profiles

August 1995

Public Health Statement for Xylene

CAS# Mixed Xylene 1330-20-7

This Public Health Statement is the summary chapter from the Toxicological Profile for xylene. It is one in a series of Public Health Statements about hazardous substances and their health effects. A shorter version, the ToxFAQs™, is also available. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present. For more information, call the ATSDR Information Center at 1-888-422-8737.

This Statement was prepared to give you information about xylene and to emphasize the human health effects that may result from exposure to it. The Environmental Protection Agency (EPA) has identified 1,408 hazardous waste sites as the most serious in the nation. These sites comprise the "National Priorities List" (NPL): those sites which are targeted for long-term federal cleanup activities. Xylene has been found in at least 658 of the sites on the NPL. However, the number of NPL sites evaluated for xylene is not known. As EPA evaluates more sites, the number of sites at which xylene is found may increase. This information is important because exposure to xylene may cause harmful health effects and because these sites are potential or actual sources of human exposure to xylene.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You can be exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking substances containing the substance or by skin contact with it.

If you are exposed to a substance such as xylene, many factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route or pathway by which you are exposed (breathing, eating, drinking, or

Priority List of Hazardous Substances
Division of Toxicology

skin contact), the other chemicals to which you are exposed, and your individual characteristics such as age, gender, nutritional status, family traits, life-style, and state of health.

1.1 What is xylene?

In this report, the terms xylene, xylenes, and total xylenes will be used interchangeably. There are three forms of xylene in which the methyl groups vary on the benzene ring: *meta*-xylene, *ortho*-xylene, and *para*-xylene (*m*-, *o*-, and *p*-xylene). These different forms are referred to as isomers. The term total xylenes refers to all three isomers of xylene (*m*-, *o*-, and *p*-xylene). Mixed xylene is a mixture of the three isomers and usually also contains 6–15% ethylbenzene. Xylene is also known as xylol or dimethylbenzene. Xylene is primarily a synthetic chemical. Chemical industries produce xylene from petroleum. Xylene also occurs naturally in petroleum and coal tar and is formed during forest fires. It is a colorless, flammable liquid with a sweet odor.

Xylene is one of the top 30 chemicals produced in the United States in terms of volume. It is used as a solvent (a liquid that can dissolve other substances) in the printing, rubber, and leather industries. Along with other solvents, xylene is also used as a cleaning agent, a thinner for paint, and in varnishes. It is found in small amounts in airplane fuel and gasoline. Xylene is used as a material in the chemical, plastics, and synthetic fiber industries and as an ingredient in the coating of fabrics and papers. Isomers of xylene are used in the manufacture of certain polymers (chemical compounds), such as plastics.

Xylene evaporates and burns easily. Xylene does not mix well with water; however, it does mix with alcohol and many other chemicals. Most people begin to smell xylene in air at 0.08–3.7 parts of xylene per million parts of air (ppm) and begin to taste it in water at 0.53–1.8 ppm.

back to top

1.2 What happens to xylene when it enters the environment?

Xylene is a liquid, and it can leak into soil, surface water (creeks, streams, rivers), or groundwater, where it may remain for months or more before it breaks down into other chemicals. However, because it evaporates easily, most xylene (if not trapped deep underground) goes into the air, where it stays for several days. In the air, the xylene is broken down by sunlight into other less harmful chemicals.

Xylene can enter the environment when it is made, packaged, shipped, or used. Most xylene that is accidentally released evaporates into the air, although some is released into rivers or lakes. Xylene can also enter soil, water, or air in large amounts after an accidental spill or as a result of an environmental leak

during storage or burial at a waste site.

Xylene very quickly evaporates into the air from surface soil and water. Xylene stays in the air for several days until it is broken down by sunlight into other less harmful chemicals.

Most xylene in surface water evaporates into the air in less than a day. The rest of it is slowly broken down into other chemicals by small living organisms in the water. Only very small amounts are taken up by plants, fish, and birds. We do not know exactly how long xylene stays in water, but we do know that it stays longer in underground water than in lakes and rivers, probably because it can evaporate from the latter.

Xylene evaporates from soil surfaces. Xylene below the soil surface stays there for several days and may travel down through the soil and enter underground water (groundwater). Small living organisms in soil and groundwater may transform it into other less harmful compounds, although this happens slowly. It is not clear how long xylene remains trapped deep underground in soil or groundwater, but it may be months or years. Xylene stays longer in wet soil than in dry soil. If a large amount of xylene enters soil from an accidental spill, a hazardous waste site, or a landfill, it may travel through the soil and contaminate drinking water wells. Only a small amount of xylene is absorbed by animals that live in water contaminated with xylene.

back to top

1.3 How might I be exposed to xylene?

You may be exposed to xylene because of its distribution in the environment. Xylene is primarily released from industrial sources, in automobile exhaust, and during its use as a solvent. Hazardous waste disposal sites and spills of xylene into the environment are also possible sources of exposure. You are most likely to be exposed to xylene by breathing it in contaminated air. Levels of xylene measured in the air of industrial areas and cities of the United States range from 1 to 88 parts of xylene per billion parts of air (a part per billion [ppb] is one thousandth of a part per million [ppm]; one ppm equals 1,000 ppb). Xylene is sometimes released into water and soil as a result of the use, storage, and transport of petroleum products. Surface water generally contains less than 1 ppb, although the level may be higher in industrial areas. You can also be exposed to xylene by drinking or eating xylene-contaminated water or food. Levels of xylene in public drinking water supplies have been reported to range from 0 to 750 ppb. Little information exists about the amount of xylene in food. Xylene levels ranging from 50 to 120 ppb have been found in some fish samples. Xylene has been found in chicken eggs and in the polystyrene packaging in which they are sold.

You may also come in contact with xylene from a variety of

consumer products, including cigarette smoke, gasoline, paint, varnish, shellac, and rust preventives. Breathing vapors from these types of products can expose you to xylene. Indoor levels of xylene can be higher than outdoor levels, especially in buildings with poor ventilation. Skin contact with products containing xylene, such as solvents, lacquers, paint thinners and removers, and pesticides may also expose you to xylene.

Besides painters and paint industry workers, others who may be exposed to xylene include biomedical laboratory workers, distillers of xylene, wood processing plant workers, automobile garage workers, metal workers, and furniture refinishers also may be exposed to xylene. Workers who routinely come in contact with xylene-contaminated solvents in the workplace are the population most likely to be exposed to high levels of xylene.

back to top

1.4 How can xylene enter and leave my body?

Xylene is most likely to enter your body when you breathe xylene vapors. Less often, xylene enters the body through the skin following direct contact. It is rapidly absorbed by your lungs after you breathe air containing it. Exposure to xylene may also take place if you eat or drink xylene-contaminated food or water. The amount of xylene retained ranges from 50% to 75% of the amount of xylene that you inhale. Physical exercise increases the amount of xylene absorbed by the lungs. Absorption of xylene after eating food or drinking water containing it is both rapid and complete. Absorption of xylene through the skin also occurs rapidly following direct contact with xylene. Absorption of xylene vapor through the skin is lower than absorption of xylene vapor by the lungs. However, it is not known how much of the xylene is absorbed through the skin. At hazardous waste sites, breathing xylene vapors, drinking well water contaminated with xylene, and direct contact of the skin with xylene are the most likely ways you can be exposed. Xylene passes into the blood soon after entering the body.

In people and laboratory animals, xylene is broken down into other chemicals especially in the liver. This process changes most of the xylene that is breathed in or swallowed into a different form. Once xylene breaks down, the breakdown products rapidly leave the body, mainly in urine, but some unchanged xylene also leaves in the breath from the lungs. One of the breakdown products of xylene, methylbenzaldehyde, is harmful to the lungs of some animals. This chemical has not been found in people exposed to xylene. Small amounts of breakdown products of xylene have appeared in the urine of people as soon as 2 hours after breathing air containing xylene. Usually, most of the xylene that is taken in leaves the body within 18 hours after exposure ends. Storage of xylene in fat or muscle may prolong the time needed for xylene to leave the body.

back to top

1.5 How can xylene affect my health?

Short-term exposure of people to high levels of xylene can cause irritation of the skin, eyes, nose, and throat; difficulty in breathing; impaired function of the lungs; delayed response to a visual stimulus; impaired memory; stomach discomfort; and possible changes in the liver and kidneys. Both short- and long-term exposure to high concentrations of xylene can also cause a number of effects on the nervous system, such as headaches, lack of muscle coordination, dizziness, confusion, and changes in one's sense of balance. People exposed to very high levels of xylene for a short period of time have died. Most of the information on long-term exposure to xylene is from studies of workers employed in industries that make or use xylene. Those workers were exposed to levels of xylene in air far greater than the levels normally encountered by the general population. Many of the effects seen after their exposure to xylene could have been caused by exposure to other chemicals that were in the air with xylene.

Results of studies of animals indicate that large amounts of xylene can cause changes in the liver and harmful effects on the kidneys, lungs, heart, and nervous system. Short-term exposure to very high concentrations of xylene causes death in animals, as well as muscular spasms, incoordination, hearing loss, changes in behavior, changes in organ weights, and changes in enzyme activity. Long-term exposure of animals to low concentrations of xylene has not been well studied.

Information from animal studies is not adequate to determine whether or not xylene causes cancer in humans. Both the International Agency for Research on Cancer (IARC) and EPA have found that there is insufficient information to determine whether or not xylene is carcinogenic and consider xylene not classifiable as to its human carcinogenicity.

Exposure of pregnant women to high levels of xylene may cause harmful effects to the fetus. Studies of unborn animals indicate that high concentrations of xylene may cause increased numbers of deaths, decreased weight, skeletal changes, and delayed skeletal development. In many instances, these same concentrations also cause damage to the mothers. The higher the exposure and the longer the exposure to xylene, the greater the chance of harmful health effects. Lower concentrations of xylene are not so harmful.

back to top

1.6 Is there a medical test to determine whether I have been exposed to xylene?

Medical tests are available to determine if you have been exposed to xylene at higher-than-normal levels. Confirmation of xylene exposure is determined by measuring some of its breakdown

products eliminated from the body in the urine. These urinary measurements will determine if you have been exposed to xylene. There is a high degree of agreement between exposure to xylene and the levels of xylene breakdown products in the urine. However, a urine sample must be provided very soon after exposure ends because xylene quickly leaves the body. Alcohol or aspirin may produce false positive test results. Medical tests have been developed to measure levels of xylene in blood by the National Center for Environmental Health Laboratory and in exhaled breath by EPA's Total Exposure Assessment Methodology. These tests may be available in certain doctors' offices. Available tests can only indicate exposure to xylene; they cannot be used to predict which health effects, if any, will develop.

back to top

1.7 What recommendations has the federal government made to protect human health?

EPA estimates that, for an adult of average weight, exposure to 10 milligrams of xylene per liter (mg/L or ppm) of water each day for a lifetime (70 years) is unlikely to result in harmful noncancerous health effects. For a long-term but less-than-lifetime exposure (about 7 years), 27.3 ppm is estimated to be a level unlikely to result in harmful health effects in an adult.

Exposure to 12 ppm xylene in water for 1 day or to 7.8 ppm of xylene in water for 10 days or longer is unlikely to present a health risk to a small child. EPA has proposed a recommended maximum level of 10 ppm xylene in drinking water.

To protect people from the potential harmful health effects of xylene, EPA regulates xylene in the environment. EPA has set a legally enforceable maximum level of 10 mg/L (equal to 10 ppm) of xylene in water that is delivered to any user of a public water system. The Occupational Safety and Health Administration (OSHA) has set an occupational exposure limit of 100 ppm of xylene in air averaged over an 8-hour workday and a 15-minute exposure limit of 150 ppm. These regulations also match recommendations (not legally enforceable) of the American Conference of Governmental Industrial Hygienists. The National Institute for Occupational Safety and Health (NIOSH) has recommended an exposure limit (not legally enforceable) of 100 ppm of xylene averaged over a workday up to 10 hours long in a 40-hour workweek. NIOSH has also recommended that exposure to xylene not exceed 150 ppm for longer than 15 minutes. NIOSH has classified xylene exposures of 10,000 ppm as immediately dangerous to life or health.

EPA and the Food and Drug Administration (FDA) specify conditions under which xylene may be used as a part of herbicides, pesticides, or articles used in contact with food. The EPA has a

chronic drinking water health advisory of 27.3 ppm for an adult and 7.8 ppm for a 10-kilogram child.

EPA regulations require that a spill of 1,000 pounds or more of xylene or used xylene solvents be reported to the Federal Government National Response Center.

back to top

1.8 Where can I get more information?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop F-32
Atlanta, GA 30333

Information line and technical assistance:

Phone: 888-422-8737
FAX: (770)-488-4178

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

To order toxicological profiles, contact:

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22161
Phone: 800-553-6847 or 703-605-6000

back to top

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological profile for xylene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

back to top

ATSDR Home | Search | Index | Glossary | Contact Us
About ATSDR | News Archive | ToxFAQs | HazDat | Public Health Assessments
Privacy Policy | External Links Disclaimer | Accessibility
U.S. Department of Health and Human Services

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

CONTENTS

RELATED RESOURCES

ToxFAQ™ 47k
ToxFAQ™ en Español 91k
Public Health Statement 175k
Toxicological Profile 9.9MB
MMG 58k

A-Z INDEX

A B C D E
F G H I J K
L M N O P
Q R S T U
V W X Y Z

ATSDR RESOURCES

ToxFAQs™
ToxFAQs™ en Español
Public Health Statements
Toxicological Profiles
Minimum Risk Levels
MMGs
MHMIs
Interaction Profiles

September 1997

Public Health Statement for Benzene

CAS# 71-43-2

This Public Health Statement is the summary chapter from the Toxicological Profile for benzene. It is one in a series of Public Health Statements about hazardous substances and their health effects. A shorter version, the ToxFAQs™, is also available. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present. For more information, call the ATSDR Information Center at 1-888-422-8737.

This public health statement tells you about benzene and the effects of exposure.

The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup. Benzene has been found in at least 816 of the 1,428 current or former NPL sites. However, it's unknown how many NPL sites have been evaluated for this substance. As more sites are evaluated, the sites with benzene may increase. This information is important because exposure to this substance may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance or by skin contact.

If you are exposed to benzene, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals you're exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

1.1 What is benzene?

Benzene, also known as benzol, is a colorless liquid with a sweet

Priority List of Hazardous Substances
Division of Toxicology

odor. Benzene evaporates into air very quickly and dissolves slightly in water. Benzene is highly flammable. Most people can begin to smell benzene in air at 1.5–4.7 parts of benzene per million parts of air (ppm) and smell benzene in water at 2 ppm. Most people can begin to taste benzene in water at 0.5–4.5 ppm. Benzene is found in air, water, and soil.

Benzene found in the environment is from both human activities and natural processes. Benzene was first discovered and isolated from coal tar in the 1800s. Today, benzene is made mostly from petroleum sources. Because of its wide use, benzene ranks in the top 20 in production volume for chemicals produced in the United States. Various industries use benzene to make other chemicals, such as styrene (for Styrofoam® and other plastics), cumene (for various resins), and cyclohexane (for nylon and synthetic fibers). Benzene is also used for the manufacturing of some types of rubbers, lubricants, dyes, detergents, drugs, and pesticides. Natural sources of benzene, which include volcanoes and forest fires, also contribute to the presence of benzene in the environment. Benzene is also a natural part of crude oil and gasoline and cigarette smoke.

back to top

1.2 How might I be exposed to benzene?

Benzene is commonly found in the environment. Industrial processes are the main sources of benzene in the environment. Benzene levels in the air can increase from emissions from burning coal and oil, benzene waste and storage operations, motor vehicle exhaust, and evaporation from gasoline service stations. Since tobacco contains high levels of benzene, tobacco smoke is another source of benzene in air. Industrial discharge, disposal of products containing benzene, and gasoline leaks from underground storage tanks can release benzene into water and soil.

Benzene can pass into air from water and soil surfaces. Once in the air, benzene reacts with other chemicals and breaks down within a few days. Benzene in the air can attach to rain or snow and be carried back down to the ground.

Benzene in water and soil breaks down more slowly. Benzene is slightly soluble in water and can pass through the soil into underground water. Benzene in the environment does not build up in plants or animals.

back to top

1.3 How can benzene enter and leave my body?

Most people are exposed to a small amount of benzene on a daily basis. You can be exposed to benzene in the outdoor environment, in the workplace, and in the home. Exposure of the general population to benzene is mainly through breathing air that contains benzene. The major sources of benzene exposure are tobacco

smoke, automobile service stations, exhaust from motor vehicles, and industrial emissions. Vapors (or gases) from products that contain benzene, such as glues, paints, furniture wax, and detergents can also be a source of exposure. Auto exhaust and industrial emissions account for about 20% of the total nationwide exposure to benzene. About 50% of the entire nationwide exposure to benzene results from smoking tobacco or from exposure to tobacco smoke. The average smoker (32 cigarettes per day) takes in about 1.8 milligrams (mg) of benzene per day. This is about 10 times the average daily intake of nonsmokers.

Measured levels of benzene in outdoor air have ranged from 0.02 to 34 parts of benzene per billion parts of air (ppb) (1 ppb is 1,000 times less than 1 ppm). People living in cities or industrial areas are generally exposed to higher levels of benzene in air than those living in rural areas. Benzene levels in the home are usually higher than outdoor levels. People living around hazardous waste sites, petroleum refining operations, petrochemical manufacturing sites, or gas stations may be exposed to higher levels of benzene in air.

For most people, the level of exposure to benzene through food, beverages, or drinking water is not as high as through air. Typical drinking water contains less than 0.1 ppb benzene. Benzene has been detected in some bottled water, liquor, and food. Leakage from underground gasoline storage tanks or from landfills and hazardous waste sites containing benzene can result in benzene contamination of well water. People with benzene-contaminated tap water can be exposed from drinking the water or eating foods prepared with the water. In addition, exposure can result from breathing in benzene while showering, bathing, or cooking with contaminated water.

Individuals employed in industries that make or use benzene may be exposed to the highest levels of benzene. As many as 238,000 people may be occupationally exposed to benzene in the United States. These industries include benzene production (petrochemicals, petroleum refining, and coke and coal chemical manufacturing), rubber tire manufacturing, and storage or transport of benzene and petroleum products containing benzene. Other workers who may be exposed to benzene because of their occupations include steel workers, printers, rubber workers, shoe makers, laboratory technicians, firefighters, and gas station employees.

back to top

1.4 How can benzene affect my health?

Benzene can enter your body through your lungs when you breathe contaminated air. It can also enter through your stomach and intestines when you eat food or drink water that contains benzene. Benzene can enter your body through skin contact with

benzene-containing products such as gasoline.

When you are exposed to high levels of benzene in air, about half of the benzene you breathe in leaves your body when you breathe out. The other half passes through the lining of your lungs and enters your bloodstream. Animal studies show that benzene taken in by eating or drinking contaminated foods behaves similarly in the body to benzene that enters through the lungs. A small amount will enter your body by passing through your skin and into your bloodstream during skin contact with benzene or benzene-containing products. Once in the bloodstream, benzene travels throughout your body and can be temporarily stored in the bone marrow and fat. Benzene is converted to products, called metabolites, in the liver and bone marrow. Some of the harmful effects of benzene exposure are believed to be caused by these metabolites. Most of the metabolites of benzene leave the body in the urine within 48 hours after exposure.

back to top

1.5 What levels of exposure have resulted in harmful health effects?

To protect the public from the harmful effects of toxic chemicals and to find ways to treat people who have been harmed, scientists use many tests.

One way to see if a chemical will hurt people is to learn how the chemical is absorbed, used, and released by the body; for some chemicals, animal testing may be necessary. Animal testing may also be used to identify health effects such as cancer or birth defects. Without laboratory animals, scientists would lose a basic method to get information needed to make wise decisions to protect public health. Scientists have the responsibility to treat research animals with care and compassion. Laws today protect the welfare of research animals, and scientists must comply with strict animal care guidelines.

After exposure to benzene, several factors determine whether harmful health effects will occur and if they do, what the type and severity of these health effects might be. These factors include the amount of benzene to which you are exposed and the length of time of the exposure. Most data involving effects of long-term exposure to benzene are from studies of workers employed in industries that make or use benzene. These workers were exposed to levels of benzene in air far greater than the levels normally encountered by the general population. Current levels of benzene in workplace air are much lower than in the past. Because of this reduction, and the availability of protective equipment such as respirators, fewer workers have symptoms of benzene poisoning.

Brief exposure (5–10 minutes) to very high levels of benzene in air (10,000–20,000 ppm) can result in death. Lower levels (700–

3,000 ppm) can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. In most cases, people will stop feeling these effects when they stop being exposed and begin to breathe fresh air.

Eating foods or drinking liquids containing high levels of benzene can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, coma, and death. The health effects that may result from eating foods or drinking liquids containing lower levels of benzene are not known. If you spill benzene on your skin, it may cause redness and sores. Benzene in your eyes may cause general irritation and damage to your cornea.

Benzene causes problems in the blood. People who breathe benzene for long periods may experience harmful effects in the tissues that form blood cells, especially the bone marrow. These effects can disrupt normal blood production and cause a decrease in important blood components. A decrease in red blood cells can lead to anemia. Reduction in other components in the blood can cause excessive bleeding. Blood production may return to normal after exposure to benzene stops. Excessive exposure to benzene can be harmful to the immune system, increasing the chance for infection and perhaps lowering the body's defense against cancer.

Benzene can cause cancer of the blood-forming organs. The Department of Health and Human Services (DHHS) has determined that benzene is a known carcinogen. The International Agency for Cancer Research (IARC) has determined that benzene is carcinogenic to humans, and the EPA has determined that benzene is a human carcinogen. Long-term exposure to relatively high levels of benzene in the air can cause cancer of the blood-forming organs. This condition is called leukemia. Exposure to benzene has been associated with development of a particular type of leukemia called acute myeloid leukemia (AML).

Exposure to benzene may be harmful to the reproductive organs. Some women workers who breathed high levels of benzene for many months had irregular menstrual periods. When examined, these women showed a decrease in the size of their ovaries. However, exact exposure levels were unknown, and the studies of these women did not prove that benzene caused these effects. It is not known what effects exposure to benzene might have on the developing fetus in pregnant women or on fertility in men. Studies with pregnant animals show that breathing benzene has harmful effects on the developing fetus. These effects include low birth weight, delayed bone formation, and bone marrow damage.

The health effects that might occur in humans following long-term exposure to food and water contaminated with benzene are not known. In animals, exposure to food or water contaminated with benzene can damage the blood and the immune system and can

even cause cancer.

back to top

1.6 Is there a medical test to determine whether I have been exposed to benzene?

Several tests can show if you have been exposed to benzene. Some of these tests may be available at your doctor's office. All of these tests are limited in what they can tell you. The test for measuring benzene in your breath must be done shortly after exposure. This test is not very helpful for detecting very low levels of benzene in your body. Benzene can be measured in your blood. However, since benzene disappears rapidly from the blood, measurements may be accurate only for recent exposures. In the body, benzene is converted to products called metabolites. Certain metabolites of benzene, such as phenol, muconic acid, and S-phenyl-N-acetyl cysteine (PhAC) can be measured in the urine. The amount of phenol in urine has been used to check for benzene exposure in workers. The test is useful only when you are exposed to benzene in air at levels of 10 ppm or greater. However, this test must also be done shortly after exposure, and it is not a reliable indicator of how much benzene you have been exposed to, since phenol is present in the urine from other sources (diet, environment). Measurement of muconic acid or PhAC in the urine is a more sensitive and reliable indicator of benzene exposure. The measurement of benzene in blood or of metabolites in urine cannot be used for making predictions about whether you will experience any harmful health effects. Measurement of all parts of the blood and measurement of bone marrow are used to find benzene exposure and its health effects.

For people exposed to relatively high levels of benzene, complete blood analyses can be used to monitor possible changes related to exposure. However, blood analyses are not useful when exposure levels are low.

back to top

1.7 What recommendations has the federal government made to protect human health?

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA). Recommendations provide valuable guidelines to protect public health but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Institute for Occupational Safety and Health (NIOSH).

Regulations and recommendations can be expressed in not-to-

exceed levels in air, water, soil, or food that are usually based on levels that affect animals, then they are adjusted to help protect people. Sometimes these not-to-exceed levels differ among federal organizations because of different exposure times (an 8-hour workday or a 24-hour day), the use of different animal studies, or other factors.

Recommendations and regulations are also periodically updated as more information becomes available. For the most current information, check with the federal agency or organization that provides it. Some regulations and recommendations for benzene include the following:

EPA has set the maximum permissible level of benzene in drinking water at 5 parts per billion (ppb). Because benzene can cause leukemia, EPA has set a goal of 0 ppb for benzene in drinking water and in water such as rivers and lakes. EPA estimates that 10 ppb benzene in drinking water that is consumed regularly or exposure to 0.4 ppb benzene in air over a lifetime could cause a risk of one additional cancer case for every 100,000 exposed persons. EPA recommends a maximum permissible level of benzene in water of 200 ppb for short-term exposures (10 days) for children.

EPA requires that the National Response Center be notified following a discharge or spill into the environment of 10 pounds or more of benzene.

The Occupational Safety and Health Administration (OSHA) regulates levels of benzene in the workplace. The maximum allowable amount of benzene in workroom air during an 8-hour workday, 40-hour workweek is 1 part per million (ppm). Since benzene can cause cancer, the National Institute for Occupational Safety and Health (NIOSH) recommends that all workers likely to be exposed to benzene wear special breathing equipment.

back to top

1.8 Where can I get more information?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop F-32
Atlanta, GA 30333

Information line and technical assistance:

Phone: 888-422-8737
FAX: (770)-488-4178

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

To order toxicological profiles, contact:

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22161
Phone: 800-553-6847 or 703-605-6000

back to top

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1997. Toxicological profile for benzene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

back to top

ATSDR Information Center / ATSDRIC@cdc.gov / 1-888-422-8737

This page was updated on November 22, 2004

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

July 1999

Public Health Statement for Ethylbenzene

CAS# 100-41-4

This Public Health Statement is the summary chapter from the Toxicological Profile for ethylbenzene. It is one in a series of Public Health Statements about hazardous substances and their health effects. A shorter version, the ToxFAQs™, is also available. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present. For more information, call the ATSDR Information Center at 1-888-422-8737.

This public health statement tells you about ethylbenzene and the effects of exposure.

The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup activities. Ethylbenzene has been found in at least 720 of the 1,467 current or former NPL sites. However, the total number of NPL sites evaluated for this substance is not known. As more sites are evaluated, the sites at which ethylbenzene is found may increase. This information is important because exposure to this substance may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance or by skin contact.

If you are exposed to ethylbenzene, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals you're exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

CONTENTS

RELATED RESOURCES

A-Z INDEX

ATSDR RESOURCES

ToxFAQs™ en Español
Public Health Statements
Toxicological Profiles
Minimum Risk Levels
MMGs
MHMIs
Interaction Profiles
Priority List of Hazardous Substances
Division of Toxicology

1.1 What is ethylbenzene?

Ethylbenzene is a colorless liquid that smells like gasoline. You can smell ethylbenzene in the air at concentrations as low as 2 parts of ethylbenzene per million parts of air by volume (ppm). It evaporates at room temperature and burns easily. Ethylbenzene occurs naturally in coal tar and petroleum. It is also found in many products, including paints, inks, and insecticides. Gasoline contains about 2% (by weight) ethylbenzene. Ethylbenzene is used primarily in the production of styrene. It is also used as a solvent, a component of asphalt and naphtha, and in fuels. In the chemical industry, it is used in the manufacture of acetophenone, cellulose acetate, diethylbenzene, ethyl anthraquinone, ethylbenzene sulfonic acids, propylene oxide, and -methylbenzyl alcohol. Consumer products containing ethylbenzene include pesticides, carpet glues, varnishes and paints, and tobacco products. In 1994, approximately 12 billion pounds of ethylbenzene were produced in the United States.

back to top

1.2 What happens to ethylbenzene when it enters the environment?

Ethylbenzene is most commonly found as a vapor in the air. This is because ethylbenzene moves easily into the air from water and soil. Once in the air, other chemicals help break down ethylbenzene into chemicals found in smog. This breakdown happens in less than 3 days with the aid of sunlight. In surface water such as rivers and harbors, ethylbenzene breaks down by reacting with other compounds naturally present in the water. In soil, the majority of ethylbenzene is broken down by soil bacteria. Since ethylbenzene binds only moderately to soil, it can also move downward through soil to contaminate groundwater. Near hazardous waste sites, the levels of ethylbenzene in the air, water, and soil could be much higher than in other areas.

back to top

1.3 How might I be exposed to ethylbenzene?

There are a variety of ways you may be exposed to this chemical. If you live in a highly populated area or near many factories or heavily traveled highways, you may be exposed to ethylbenzene in the air. Releases of ethylbenzene into these areas occur from burning oil, gas, and coal and from discharges of ethylbenzene from some types of factories. The median level of ethylbenzene in city and suburban air is about 0.62 parts of ethylbenzene per billion parts (ppb) of air. In contrast, the median level of ethylbenzene measured in air in country locations is about 0.01 ppb. Indoor air has a higher median concentration of ethylbenzene (about 1 ppb) than outdoor air. This is because ethylbenzene builds up after you use household products such as cleaning products or paints.

Ethylbenzene was found in only one of ten U.S. rivers and

streams tested in 1982 and 1983. The average level measured was less than 5.0 ppb. Ethylbenzene gets into water from factory releases, boat fuel, and poor disposal of waste. Background levels in soils have not been reported. Ethylbenzene may get into the soil by gasoline or other fuel spills and poor disposal of industrial and household wastes.

Some people are exposed to ethylbenzene in the workplace. Gas and oil workers may be exposed to ethylbenzene either through skin contact or by breathing ethylbenzene vapors. Varnish workers, spray painters, and people involved in gluing operations may also be exposed to high levels of ethylbenzene. Exposure may also occur in factories that use ethylbenzene to produce other chemicals.

You may be exposed to ethylbenzene if you live near hazardous waste sites containing ethylbenzene or areas where ethylbenzene spills have occurred. Higher-than-background levels of ethylbenzene were detected in groundwater near a landfill and near an area where a fuel spill had occurred. No specific information on human exposure to ethylbenzene near hazardous waste sites is available.

You may also be exposed to ethylbenzene from the use of many consumer products. Gasoline is a common source of ethylbenzene exposure. Other sources of ethylbenzene exposure come from the use of this chemical as a solvent in pesticides, carpet glues, varnishes and paints, and from the use of tobacco products. Ethylbenzene does not generally build up in food. However, some vegetables may contain very small amounts of it.

back to top

1.4 How can ethylbenzene enter and leave my body?

When you breathe air containing ethylbenzene vapor, it enters your body rapidly and almost completely through your lungs. Ethylbenzene in food or water can also rapidly and almost completely enter your body through the digestive tract. It may enter through your skin when you come into contact with liquids containing ethylbenzene. Ethylbenzene vapors do not enter through your skin to any large degree. People living in urban areas or in areas near hazardous waste sites may be exposed by breathing air or by drinking water contaminated with ethylbenzene. Once in your body, ethylbenzene is broken down into other chemicals. Most of it leaves in the urine within 2 days. Small amounts can also leave through the lungs and in feces. Liquid ethylbenzene that enters through your skin is also broken down. Ethylbenzene in high levels is broken down slower in your body than low levels of ethylbenzene. Similarly, ethylbenzene mixed with other solvents is also broken down more slowly than ethylbenzene alone. This slower breakdown will increase the time it takes for ethylbenzene to leave your body.

back to top

1.5 How can ethylbenzene affect my health?

To protect the public from the harmful effects of toxic chemicals and to find ways to treat people who have been harmed, scientists use many tests.

One way to see if a chemical will hurt people is to learn how the chemical is absorbed, used, and released by the body; for some chemicals, animal testing may be necessary. Animal testing may also be used to identify health effects such as cancer or birth defects. Without laboratory animals, scientists would lose a basic method to get information needed to make wise decisions to protect public health. Scientists have the responsibility to treat research animals with care and compassion. Laws today protect the welfare of research animals, and scientists must comply with strict animal care guidelines.

At certain levels, exposure to ethylbenzene can harm your health. People exposed to high levels of ethylbenzene in the air for short periods have complained of eye and throat irritation. Persons exposed to higher levels have shown signs of more severe effects such as decreased movement and dizziness. No studies have reported death in humans following exposure to ethylbenzene alone. However, evidence from animal studies suggests that it can cause death at very high concentrations in the air (about 2 million times the usual level in urban air). Whether or not long-term exposure to ethylbenzene affects human health is not known because little information is available. Short-term exposure of laboratory animals to high concentrations of ethylbenzene in air may cause liver and kidney damage, nervous system changes, and blood changes. The link between these health effects and exposure to ethylbenzene is not clear because of conflicting results and weaknesses in many of the studies. Also, there is no clear evidence that the ability to get pregnant is affected by breathing air or drinking water containing ethylbenzene, or coming into direct contact with ethylbenzene through the skin. Two long-term studies in animals suggest that ethylbenzene may cause tumors. One study had many weaknesses, and no conclusions could be drawn about possible cancer effects in humans. The other, a recently completed study, was more convincing, and provided clear evidence that ethylbenzene causes cancer in one species after exposure in the air to concentrations greater than 740 ppm that were approximately 1 million times the levels found in urban air. At present, the federal government has not identified ethylbenzene as a chemical that may cause cancer in humans. However, this may change after consideration of the new data.

There are no reliable data on the effects in humans after eating or drinking ethylbenzene or following direct exposure to the skin. For this reason, levels of exposure that may affect your health after

eating, drinking, or getting ethylbenzene on your skin are estimated from animal studies. There are only two reports of eye or skin exposure to ethylbenzene. In these studies, liquid ethylbenzene caused eye damage and skin irritation in rabbits. More animal studies are available that describe the effects of breathing air or drinking water containing ethylbenzene.

back to top

1.6 How can ethylbenzene affect children?

This section discusses potential health effects from exposures during the period from conception to maturity at 18 years of age in humans. Potential effects on children resulting from exposures of the parents are also considered.

Since ethylbenzene is contained in many consumer products (including gasoline, paints, inks, pesticides, and carpet glue), it is possible for children to be exposed to ethylbenzene, especially by inhalation. Children might also be exposed to ethylbenzene from hazardous waste. Ethylbenzene vapors are heavier than air, and children generally spend more time on the floor or ground than do adults. We do not know whether children would be different than adults in their weight-adjusted intake of ethylbenzene.

No data describe the effect of exposure to ethylbenzene on children or immature animals. It is likely that children would show the same health effects as adults. Respiratory and eye irritation and dizziness are the most prevalent signs of exposure to high levels of ethylbenzene in adults, and children would probably also exhibit these effects. We do not know whether children differ in their susceptibility to the effects of ethylbenzene. We do not know whether ethylbenzene causes birth defects in people. Minor birth defects have occurred in newborn animals whose mothers were exposed by breathing air contaminated with ethylbenzene.

We do not know whether ethylbenzene can cross the placenta to an unborn child or accumulate significantly in breast milk.

back to top

1.7 How can families reduce the risk of exposure to ethylbenzene?

Ethylbenzene is found in the blood, urine, breath, and some body tissues of exposed people. Urine is most commonly tested to determine exposure to ethylbenzene. The test measures the presence of substances formed following an exposure to ethylbenzene. These substances are formed by the breakdown of ethylbenzene. You should have this test done within a few hours after exposure occurs because these substances leave the body very quickly. Although this test can prove your exposure to ethylbenzene, it cannot yet predict the kind of health effects that might develop from that exposure.

back to top

1.8 Is there a medical test to determine whether I have ben exposed to ethylbenzene?

If your doctor finds that you have been exposed to significant amounts of ethylbenzene, ask your doctor if children may also be exposed. When necessary your doctor may need to ask your state public heath department to investigate.

Ethylbenzene is found in consumer products including gasoline, pesticides, carpet glues, varnishes, paints, and tobacco products. Exposure to ethylbenzene vapors from household products and newly installed carpeting can be minimized by using adequate ventilation. Household chemicals should be stored out of reach of young children to prevent accidental poisonings. Always store household chemicals in their original labeled containers; never store household chemicals in containers children would find attractive to eat or drink from, such as old soda bottles. Gasoline should be stored in a gasoline can with a locked cap. Keep your Poison Control Center's number by the phone. To minimize exposure, children should be kept out of areas where products that contain ethylbenzene are being used. Sometimes older children sniff household chemicals in an attempt to get high. Your children may be exposed to ethylbenzene by inhaling products containing it, such as paints, varnishes, or gasoline. Talk with your children about the dangers of sniffing chemicals.

back to top

1.9 What recommendations has the federal government made to protect human health?

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA). Recommendations provide valuable guidelines to protect public health but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Institute for Occupational Safety and Health (NIOSH).

Regulations and recommendations can be expressed in not-to-exceed levels in air, water, soil, or food that are usually based on levels that affect animals; then they are adjusted to help protect people. Sometimes these not-to-exceed levels differ among federal organizations because of different exposure times (an 8-hour workday or a 24-hour day), the use of different animal studies, or other factors.

Recommendations and regulations are also periodically updated as

more information becomes available. For the most current information, check with the federal agency or organization that provides it. Some regulations and recommendations for ethylbenzene include the following:

The federal government has developed regulatory standards and guidelines to protect you from possible health effects of ethylbenzene in the environment. EPA's Office of Drinking Water (ODW) set 700 ppb (this equals 0.7 milligrams ethylbenzene per liter of water or mg/L) as the acceptable exposure concentration of ethylbenzene in drinking water for an average weight adult. This value is for lifetime exposure and is set at a level that is expected not to increase the chance of having (noncancer) adverse health effects. The same EPA office (ODW) set higher acceptable levels of ethylbenzene in water for shorter periods (20 ppm or 20 mg/L for 1 day, 3 ppm or 3 mg/L for 10 days). EPA has determined that exposures at or below these levels are acceptable for small children. If you eat fish and drink water from a body of water, the water should contain no more than 1.4 mg ethylbenzene per liter.

EPA requires that a release of 1,000 pounds or more of ethylbenzene be reported to the federal government's National Response Center in Washington, D.C.

OSHA set a legal limit of 100 ppm ethylbenzene in air. This is for exposure at work for 8 hours per day.

NIOSH also recommends an exposure limit for ethylbenzene of 100 ppm. This is for exposure to ethylbenzene in air at work for up to 10 hours per day in a 40-hour work week. NIOSH also set a limit of 125 ppm for a 15-minute period.

back to top

1.10 Where can I get more information?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop F-32
Atlanta, GA 30333

Information line and technical assistance:

Phone: 888-422-8737
FAX: (770)-488-4178

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from

exposure to hazardous substances.

To order toxicological profiles, contact:

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22161
Phone: 800-553-6847 or 703-605-6000

back to top

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological profile for ethylbenzene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

back to top

ATSDR Information Center / ATSDRIC@cdc.gov / 1-888-422-8737

This page was updated on November 22, 2004

U.S. Environmental Protection Agency

Lead in Paint, Dust, and Soil

Basic Information

Did you know the following facts about lead?

FACT: Lead exposure can harm young children and babies even before they are born.

FACT: Even children who seem healthy can have high levels of lead in their bodies.

FACT: You can get lead in your body by breathing or swallowing lead dust, or by eating soil or paint chips containing lead.

FACT: You have many options for reducing lead hazards. In most cases, lead-based paint that is in good condition is not a hazard.

FACT: Removing lead-based paint improperly can increase the danger to your

family.

If you think your home might have lead hazards, read on to learn about lead and some simple steps to protect your family.

Health Effects of Lead

In the United States, about 900,000 children ages 1 to 5 have a blood-lead level above the level of concern.

Even children who appear healthy can have dangerous levels of lead in their bodies.

- People can get lead in their body if they:
  - Put their hands or other objects covered with lead dust in their mouths.
  - Eat paint chips or soil that contains lead.
  - Breathe in lead dust (especially during renovations that disturb painted surfaces).
- Lead is even more dangerous to children than adults because:
  - Babies and young children often put their hands and other objects in their mouths. These objects can have lead dust on them.
  - Children's growing bodies absorb more lead.
  - Children's brains and nervous systems are more sensitive to the damaging effects of lead.
- If not detected early, children with high levels of lead in their bodies can suffer from:
  - Damage to the brain and nervous system
  - Behavior and learning problems (such as hyperactivity)
  - Slowed growth
  - Hearing problems
  - Headaches
- Lead is also harmful to adults. Adults can suffer from:
  - Difficulties during pregnancy
  - Other reproductive problems (in both men and women)
  - High blood pressure
  - Digestive problems
  - Nerve disorders
  - Memory and concentration problems
  - Muscle and joint pain

Back to Top

Where Lead is Found

*In general, the older your home, the more likely it has lead-based paint. *

- Paint. Many homes built before 1978 have lead-based paint. The

federal government banned lead-based paint from housing in 1978. Some states stopped its use even earlier. Lead can be found:
    - In homes in the city, country, or suburbs.
    - In apartments, single-family homes, and both private and public housing.
    - Inside and outside of the house.
- In soil around a home. (Soil can pick up lead from exterior paint, or other sources such as past use of leaded gas in cars.)
- Household dust. (Dust can pick up lead from deteriorating lead-based paint or from soil tracked into a home.)
- Drinking water. Your home might have plumbing with lead or lead solder. Call your local health department or water supplier to find out about testing your water. You cannot see, smell, or taste lead, and boiling your water will not get rid of lead. If you think your plumbing might have lead in it:
    - Use only cold water for drinking and cooking.
    - Run water for 15 to 30 seconds before drinking it, especially if you have not used your water for a few hours.
- The job. If you work with lead, you could bring it home on your hands or clothes. Shower and change clothes before coming home. Launder your work clothes separately from the rest of your family's clothes.
- Old painted toys and furniture.
- Food and liquids stored in lead crystal or lead-glazed pottery or porcelain.
- Lead smelters or other industries that release lead into the air.
- Hobbies that use lead, such as making pottery or stained glass, or refinishing furniture.
- Folk remedies that contain lead, such as "greta" and "azarcon" used to treat an upset stomach.

Back to Top

Where Lead is Likely to be a Hazard

Lead from paint chips, which you can see, and lead dust, which you can't always see, can be serious hazards.

- Peeling, chipping, chalking, or cracking lead-based paint is a hazard and needs immediate attention.
- Lead-based paint may also be a hazard when found on surfaces that children can chew or that get a lot of wear-and-tear. These areas include:
    - Windows and window sills.
    - Doors and door frames.
    - Stairs, railings, and banisters.
    - Porches and fences.

Note: Lead-based paint that is in good condition is usually not a hazard.

- Lead dust can form when lead-based paint is dry scraped, dry sanded, or heated. Dust also forms when painted surfaces bump or rub together. Lead chips and dust can get on surfaces and objects that people touch. Settled lead dust can re-enter the air when people vacuum, sweep, or walk through it.

- Lead in soil can be a hazard when children play in bare soil or when people bring soil into the house on their shoes. Contact the National Lead Information Center (NLIC) to find out about testing soil for lead.

Back to Top

Checking Your Family and Home for Lead

Get your children and home tested if you think your home has high levels of lead.

Just knowing that a home has lead-based paint may not tell you if there is a hazard.

To reduce your child's exposure to lead, get your child checked, have your home tested (especially if your home has paint in poor condition and was built before 1978), and fix any hazards you may have.

- Your Family
  - Children's blood lead levels tend to increase rapidly from 6 to 12 months of age, and tend to peak at 18 to 24 months of age.
  - Consult your doctor for advice on testing your children. A simple blood test can detect high levels of lead. Blood tests are important for:
    - Children at ages 1 and 2.
    - Children and other family members who have been exposed to high levels of lead.
    - Children who should be tested under your state or local health screening plan.
  - Your doctor can explain what the test results mean and if more testing will be needed.
- Your Home
  - You can get your home checked in one of two ways, or both:
    - A paint inspection tells you the lead content of every different type of painted surface in your home. It won't tell you whether the paint is a hazard or how you should deal with it.
    - A risk assessment tells you if there are any sources of serious lead exposure (such as peeling paint and lead dust). It also tells you what actions to take to address these hazards.
  - Have qualified professionals do the work. There are standards in place for certifying lead-based paint professionals to ensure the work is done safely, reliably, and effectively. Contact the National Lead Information Center (NLIC) for a list of contacts in your area.
  - Trained professionals use a range of methods when checking your home, including:
    - Visual inspection of paint condition and location.
    - A portable x-ray fluorescence (XRF) machine.
    - Lab tests of paint samples.
    - Surface dust tests.

Note: Home test kits for lead are available, but studies suggest that they are not always accurate. Consumers should not rely on these tests before doing renovations or to assure safety.

Back to Top

What You Can do to Protect Your Family

- If you suspect that your house has lead hazards, you can take some immediate steps to reduce your family's risk:
  - If you rent, notify your landlord of peeling or chipping paint.
  - Clean up paint chips immediately.
  - Clean floors, window frames, window sills, and other surfaces weekly. Use a mop, sponge, or paper towel with warm water and a general all-purpose cleaner or a cleaner made specifically for lead. REMEMBER: NEVER MIX AMMONIA AND BLEACH PRODUCTS TOGETHER SINCE THEY CAN FORM A DANGEROUS GAS.
  - Thoroughly rinse sponges and mop heads after cleaning dirty or dusty areas.
  - Wash children's hands often, especially before they eat and before nap time and bed time.
  - Keep play areas clean. Wash bottles, pacifiers, toys, and stuffed animals regularly.
  - Keep children from chewing window sills or other painted surfaces.
  - Clean or remove shoes before entering your home to avoid tracking in lead from soil.
  - Make sure children eat nutritious, low-fat meals high in iron and calcium, such as spinach and dairy products. Children with good diets absorb less lead.
- In addition to day-to-day cleaning and good nutrition:
  - You can temporarily reduce lead hazards by taking actions such as repairing damaged painted surfaces and planting grass to cover soil with high lead levels. These actions (called "interim controls") are not permanent solutions and will need ongoing attention.
  - To permanently remove lead hazards, you must hire a certified lead "abatement" contractor. Abatement (or permanent hazard elimination) methods include removing, sealing, or enclosing lead-based paint with special materials. Just painting over the hazard with regular paint is not enough.
  - Always hire a person with special training for correcting lead problems--someone who knows how to do this work safely and has the proper equipment to clean up thoroughly. Certified contractors will employ qualified workers and follow strict safety rules set by their state or the federal government.
  - Contact the National Lead Information Center(NLIC) for help with locating certified contractors in your area and to see if financial assistance is available.

Back to Top

Are You Planning to Buy or Rent a Home Built Before 1978?

Many houses and apartments built before 1978 have paint that contains lead (called lead-based paint). Lead from paint, chips, and dust can pose serious health hazards if not taken care of properly.

Federal law requires that individuals receive certain information before renting

or buying a pre-1978 housing:

- Residential Lead-Based Paint Disclosure Program
  - LANDLORDS have to disclose known information on lead-based paint and lead-based paint hazards before leases take effect. Leases must include a disclosure form about lead-based paint.
  - SELLERS have to disclose known information on lead-based paint and lead-based paint hazards before selling a house. Sales contracts must include a disclosure form about lead-based paint. Buyers have up to 10 days to check for lead hazards.
  - More information on the disclosure program.

Back to Top

Remodeling or Renovating a Home with Lead-Based Paint

If not conducted properly, certain types of renovations can release lead from paint and dust into the air.

Many houses and apartments built before 1978 have paint that contains lead (called lead-based paint). Lead from paint, chips, and dust can pose serious health hazards if not taken care of properly.

- Federal law requires that contractors provide lead information to residents before renovating a pre-1978 housing:
  - Pre-Renovation Education Program (PRE)
    - RENOVATORS have to give you a pamphlet titled "Protect Your Family from Lead in Your Home", before starting work.
    - More information on the Pre-Renovation Education Program.
- Take precautions before your contractor or you begin remodeling or renovations that disturb painted surfaces (such as scraping off paint or tearing out walls):
  - Have the area tested for lead-based paint.
  - Do not use a belt-sander, propane torch, heat gun, dry scraper, or dry sandpaper to remove lead-based paint. These actions create large amounts of lead dust and fumes.
  - Lead dust can remain in your home long after the work is done.
  - Temporarily move your family (especially children and pregnant women) out of the apartment or house until the work is done and the area is properly cleaned. If you can't move your family, at least completely seal off the work area.
  - Follow other safety measures to reduce lead hazards. You can find out about other safety measures in the EPA brochure titled "Reducing Lead Hazards When Remodeling Your Home". This brochure explains what to do before, during, and after renovations.
  - If you have already completed renovations or remodeling that could have released lead-based paint or dust, get your young children tested and follow the steps outlined to protect your family.

Back to Top

Additional Resources

- Documents and Brochures
  - Lead in Your Home: A Parent's Reference Guide
  - Testing Your Home for Lead in Paint, Dust, and Soil
  - Finding a Qualified Lead Professional for Your Home
  - Lead Poisoning and Your Children (English)
  - Lead Poisoning and Your Children (En Español)
  - Protect Your Family From Lead in Your Home (English)
  - Protect Your Family From Lead in Your Home (En Español)
  - Reducing Lead Hazards When Remodeling Your Home (English)
  - Reducing Lead Hazards When Remodeling Your Home (En Español)
  - Ten Tips to Protect Children from Pesticide and Lead Poisonings around the Home
  - The Lead-Based Paint Pre-Renovation Education Rule: A Handbook for Contractors, Property Managers, and Maintenance Personnel
  - Lead Paint Safety: A Field Guide for Painting, Home Maintenance, and Renovation Work
- Other Lead Resources

Back to Top

OFFICE OF THE SCIENCE ADVISOR

GUIDANCE

CHAPTER 7

ASSESSMENT OF HEALTH RISKS FROM INORGANIC LEAD IN SOIL

ABSTRACT

This guidance describes a mathematical model for estimating blood lead concentration resulting from contact with lead-contaminated environmental media. A lead concentration of concern of ten micrograms per deciliter of whole blood is established. A distributional approach is used, allowing estimation of various percentiles of blood lead concentration associated with a given set of inputs. The method has been adapted to a computer spreadsheet.

Principal Writer : James Carlisle, D.V.M., M.Sc.

TABLE OF CONTENTS

Assessment of Health Risks From Inorganic Lead in Soil

1 INTRODUCTION

1.1 Purpose

The purpose of this guidance is to provide a methodology for evaluating exposure and the potential for adverse health effects resulting from exposure to lead in the environment.

1.2 Application

Since most human health effects data are based on blood lead (Pb) concentration, this guidance presents a blood Pb concentration of concern for the protection of human health, and an alogrithm for estimating blood Pb concentrations in children and adults based on a multi-pathway analysis.

1.3 Limitations

It is anticipated that this guidance will be periodically revised to reflect the changing state of the science.

2 PRINCIPLES

2.1 Blood Lead Concentration Of Concern

The Pb concentration of concern in children and adults is ten micrograms (ug) per deciliter (dl) of whole blood. The point of departure for risk management is a 0.01 risk of exceeding this value.

2.2 Lead Exposure Pathways--Blood Lead Calculation

This method can be used to estimate blood lead concentrations resulting from exposure via the five pathways listed below. Each pathway is represented by an equation relating incremental blood lead increase to a concentration in a medium, using contact rates and empirically determined ratios. The contributions via the five pathways are added to arrive at an estimate of median blood lead concentration resulting from the multipathway exposure. Ninetieth, ninety-fifth, ninety-eighth, and ninety-ninth percentile concentrations are estimated from the median by assuming a log-normal distribution with a geometric standard deviation (GSD) of

1.42. The method has been adapted to a computer spreadsheet.

3 METHODS

Generalized equations describing uptake via the five exposure pathways are as follows:

Dietary Intake Equation

Pbb = dietary Pb * contact rate * dietary constant

where:

dietary Pb (ug Pb/kg diet) = (9.45 + 0.025 * mg Pb/kg soil) [1]
contact rate, adults = 2.2 kg diet/day [2]
contact rate, children = 1.3 kg diet/day [2]
dietary constant, children = 0.16 (ug Pb/dl blood)/(ug Pb/day)[3]
dietary constant, adults = 0.04 (ug Pb/dl blood)/(ug Pb/day)[4]

Drinking Water Intake Equation

Pbb = water Pb * contact rate * dietary constant

where:

drinking water Pb (ug Pb/l water) is a site-specific, measured value [5]
contact rate, adults = 1.4 l water/day [6]
contact rate, children = 0.4 l water/day [6]
dietary constant, children = 0.16 (ug Pb/dl blood)/(ug Pb/day)[3]
dietary constant, adults = 0.04 (ug Pb/dl blood)/(ug Pb/day)[4]

Soil and Dust Ingestion Intake Equation

Pbb = soil Pb * contact rate * soil constant

where:

soil Pb (ug/g) is a site-specific, measured value [15]
contact rate, children = 0.055 g/day [7]
contact rate, adults = 0.025 g/day [8]
soil constant, children = 0.07 (ug Pb/dl blood)/(ug ingested Pb/day)[9]
soil constant, adults = 0.018 (ug Pb/dl blood)/(ug ingested Pb/day)[9]

Inhalation Intake Equation

Pbb = atmospheric Pb * inhalation constant

where:

atmospheric Pb = local or regional ambient Pb (ug/m3) + (airborne dust * soil Pb)10
inhalation constant, children = 1.92 (ug/dl)/(ug/m3)11
inhalation constant, adults = 1.64 (ug/dl)/(ug/m3)11
airborne dust (g/m3) is a site-specific, measured value with a default value

of 0.00005.

Dermal Contact Intak e Equation

Pbb = soil Pb * contact rate * soil constant
where:

soil Pb (ug Pb/gm soil) is a site-specific, measured value
contact rate, children = 1.4 gm soil/day [12]
contact rate, adults = 1.85 gm soil/day [13]
soil constant = 0.0001 (ug Pb/dl blood)/(ug dermal Pb/day) [14]

---

1 Derived as follows: (0.945 * 10 ug/kg) ÷ (0.055 * 0.00045 * soil Pb in mg/kg * 1000 ug/mg). Assumes that 5.5% of the diet consists of home-grown produce with the other 94.5% supplied by a homogeneous source with a lead content of 10 ug/kg. If food production on the site can be ruled out, use 10 ug/kg for dietary lead (EPA, 1989b, Bolger, et.al., 1990). Home-grown produce is assumed to contain 0.045% of the lead level in the soil.

2 Based on a report by Pennington (1983). For this method, a one-year-old child shall represent all children, based on the assumption that protecting the one-year-old child will protect all children.

3 Based on a study by Ryu, et.al. (1983)

4 Based on a report by FDA (1990)

5 Pb concentrations in local water supplies as consumed. If site-specific data are unavailable, a value of 15 ug/l may be used.

6 EPA (1989b)

7 Based on Calabrese (1990). Deliberate soil ingestion (soil pica) is represented as 0.00079 kg soil/day average.

8 For residential exposures and most occupational exposures, based on Calabrese (1990). Occupations with a high potential for soil ingestion (such as construction) should be represented as 0 .00005 kg soil/day average.

9 These values are 44% of tha t for lead ingested with food or water, based on a study in rats which compared the bioavailability of lead acetate mixed with the diet to that of soil-bound lead (Chaney et.al., 1990).

10 The ambient air Pb concentration data are available from the California Air Resources Board, Technical Support Division. Data for the most recent year for the nearest monitoring station should be used. If monitoring data collected within the same air basin are unavailable, a value of 0.18 ug/m3 may be used, or consult with the DTSC project manager. Respirable airborne dust is assumed to be 0.00005 g/m3 unless site-specific data are available.

11 Based on EPA (1986)

12 Based on a soil adherence of 5 g/m2 and 0.28 m2 of exposed skin (EPA, 1989b).

13 Based on a soil adhere nce of 5 g/m2 and 0.37 m2 of exposed skin (EPA, 1989b).

14 This value is derived by multiplying the Pb ingestion:blood concentration ratio for adults (0.018 ug/dl per ug/day) by the ratio of dermal absorption [0.06% (Moore, et. al., 1980)] to oral absorption [11% (ATSDR, 1990)].

15 Developed according to Chapter 2 of this Guidance.

4 COMMENTS

4.1 Blood Lead Concentration Of Concern

The traditional reference dose approach to toxic chemicals is not applied to Pb because most human health effects data are based on blood Pb concentrations rather than external dose. Blood Pb concentration is an integrated measure of internal dose, reflecting total exposure from site-related and background sources. A clear no-observed-effect concentration has not been established for such Pb-related endpoints as birth weight, gestation period, heme synthesis and neurobehavioral development in children and fetuses, and blood pressure in middle-aged men. Dose-response curves for these endpoints appear to extend down to 10 ug Pb/dl or less (ATSDR, 1990).

4.2 Estimating Blood Lead Concentrations From Environmental Concentrations

Total Pb is generally used as the measure of Pb in various media, even though the disposition of Pb may differ according to its form. Insufficient data are available to justify differential treatment of different forms of inorganic Pb. However, if the lead at a particular site has been shown, in studies acceptable to DTSC, to be less bioavailable than the assumed values, lower bioavailability factors may be substituted for the default factors. Organic Pb is more readily absorbed through the skin and other membranes than inorganic Pb, and it must therefore be treated separately. Since it is less stable in the environment, it is usually a minor source of exposure.

In the absence of specific information about the population of interest, background exposures are estimated using norms developed from survey data.

4.3 Derivation Of Model Parameters

Unless the potential for on-site gardening can be ruled out, it is assumed that 5.5% of the diet consists of home-grown produce, based on EPA guidance (USEPA, 1991). Pb concentration in home-grown produce is calculated as 0.045% of that in the soil, based on plant uptake studies (Chaney, et.al., 1982). Background dietary Pb concentration (10 ug/kg) is based on a 1990 report based on FDA data (Bolger, et.al., 1990). The default drinking water Pb concentration is based on the federal action concentration of 15 ug/l at the tap (USEPA, 1991b).

The distribution of blood Pb concentrations for a given set of environmental inputs is a critical factor in protecting sensitive members of the population.

Based on a review of data from NHANES II and from several published studies of blood Pb concentrations in children living near point sources of lead, EPA concluded that blood Pb was generally log-normally distributed, that the geometric standard deviation (GSD) for children was between 1.3 and 1.53, and that 1.42 was a representative value for the GSD (USEPA, 1989c). Adult GSDs ranged from 1.34 to 1.40, which we do not consider to be sufficiently different from the range for children to justify using a different value for adults. The model assumes a log-normal distribution with a GSD of 1.42 and uses this information to estimate the fiftieth, ninetieth, ninety-fifth, ninety-eighth, and ninety-ninth percentile blood Pb concentration for a set of inputs. Since this distribution reflects the physiologic and behavioral variables including soil consumption, using upper bound values for contact rates would distort the percentiles corresponding to blood Pb concentrations.

The availability of Pb ingested with soil is based on a study which compared the absorption of soil Pb and Pb acetate incorporated into the diet of rats (Chaney, et.al., 1990). While the authors found a direct relationship between the Pb concentration in the soil and Pb bioavailability, the data did not define the shape of the concentration/ bioavailability curve sufficiently to allow extrapolation beyond the range studied. The highest observed bioavailability for soil lead concentrations less than 1000 ppm was 44% of that observed for Pb acetate, and this guideline adopts this value as a conservative estimate of bioavailability. To accurately assess the matrix effect, a variety of variables, including lead species, particle size, and soil type would have to be systematically examined at various Pb concentrations in soil.

The daily soil adherence to skin of 5 g/m2 (0.5 mg/cm2) is based on Driver et.al (1989). The dermal absorption factor of 0.0001 ug Pb/dl blood per ug dermal Pb/day was developed by multiplying the Pb ingestion:blood concentration ratio for adults (0.018 ug/dl per ug/day) by the ratio of dermal absorption [0.06% (Moore, et. al., 1980)] to oral absorption [(11% (ATSDR, 1990)]. Based on data in the Exposure Factors Handbook (USEPA, 1989b), the median skin area of arms, hands, feet, and legs of 1-year-old boys is estimated to be 0.28 m2, and the median skin area of arms and hands of men is estimated to be 0.37 m2.

The ratio of 0.16 ug/dl per ug/day ingested by children is a value derived from studies in infants by Ryu et.al. (1983). The ratio of 0.04 ug/dl per ug/day ingested by adults is an empirically-determined value recommended by EPA (1986) and FDA (1990). The default value for inadvertent soil/dust ingestion by children , 55 mg/day, is based on tracer studies reviewed by Calabrese, et.al. (1991). Adult soil consumption is 25 mg/day, based on EPA (1991a). DTSC uses soil consumption rates of 200 and 100 mg/day in calculating a reasonable maximum exposure for children and adults,

respectively. However, reasonable maximum inputs are not recommended for use with the lead model because the model already considers the distribution of blood lead, which reflects variation in soil ingestion along with other variables. Soil consumption representing pica is 0.79 g/day, based on estimates by Calabrese et.al. (1991).

The slopes of 1.92 and 1.64 ug/dl of blood per ug/m3 of continuously-breathed air at atmospheric Pb concentrations <5 ug/m3 are based on results of experimental exposures and epidemiological studies which adjusted for airborne lead contributions to pathways other than inhalation. These studies found slopes ranging from 1.52 to 2.46 ug/dl per ug/m3 in children and 1.25 to 2.14 in adults (USEPA, 1986). The default airborne lead concentration is the highest monthly mean 24-hour value recorded in California in 1990.

4.4 Using This Guidance

This guidance may be implemented using a computer spreadsheet, which may be obtained from DTSC. The spreadsheet is based on DTSC Guidance, Volume 4, Chapter 1, which should be consulted for more general aspects of spreadsheet application. For this spreadsheet, soil concentration in mg/kg (ppm w/w) is entered in cell E7. The spreadsheet uses it in each calculation that is affected by soil Pb. Atmospheric Pb is entered in cell E6. Drinking-water Pb is entered in cell E8. If omission of the site-grown produce pathway can be justified, a "0" is entered in cell E9. Airborne dust level is entered in cell E10. The remainder of the cells are protected and should not be altered without approval of DTSC. Any such changes will require sufficient justification and must be documented.

4.5 Other Standards And Guidance

USEPA (1991c) considers lead to be a class B-2 carcinogen, with sufficient evidence in animals and inadequate evidence in humans. A carcinogenic potency has not been assigned. The federal MCL is 15 ug/l maximum at the tap with a maximum of 5 ug/l as a system-wide average (USEPA, 1991b). The Centers for Disease Control has stated that prevention activities should be directed at reducing children's blood Pb concentrations at least to below 10 ug/dl (CDC, 1991). The EPA has set 1.5 ug/m3 as the Pb concentration limit for ambient air (quarterly average) (USEPA, 1978). California's standard is also 1.5 ug/m3, but is based on a monthly average. The threshold limit value is 50 ug/m3 for workplace air (ACGIH, 1989).

FDA (1990) considers the Lowest Observable Adverse Effect Level (LOAEL) to be 10 ug/dl in children and fetuses, and 30 ug/dl in adults. They use empirically-derived ratios of 0.16 and 0.04 ug/dl per ug/day ingested to predict concentrations in young children and adults, respectively. Applying an uncertainty factor of ten results in provisional

tolerable intake levels of 6 ug/day for children six or less, 15 ug/day for children over six, 25 ug/day for pregnant women, and 75 ug/day for men.

REFERENCES

ACGIH. 1989. American conference of Governmental Industrial Hygienists, Documentation of TLVs and BEIs.

ATSDR. 1990. Agency for Toxic Substances and Disease Registry, U.S. Public Health Service; Toxicological Profile for Lead.

Bolger, P. M., S. G. Capar, and M. A. Adams. 1990. Reductions in Dietary Lead Exposure in the United States. Presented at the Symposium on the Bioavailability and Dietary Exposure of Lead, Sept 24-27, 1990.

Calabrese, E. J., E. S. Stanek, and C. E. Gilbert. 1991. A Preliminary Decision Framework for Deriving Soil Ingestion Rates. In: Hydrocarbon-Contaminated Soils and Groundwater, Volume 1, Kostecki and Calabrese, eds ., Lewis, Chelsea, MI.

Carlisle, J. C., and Michael J. Wade. 1992. Predicting blood lead concentrations from environmental concentrations. Reg. Tox. and Pharmacology 16: 280-289.

CDC. 1991. Centers for Disease Control, U.S. Public Health Service ; Preventing Lead Poisoning in Young Children.

Chaney, R. L., H. W. Mielke, and S. B. Sterrett. 1990. Speciation, Mobility, and Bioavailability of Soil Lead; Environ. Geochem. and Health.

Chaney, R. L., S. B. Sterrett, and H. W. Mielke. 1982. The Potential for Heavy Metal Exposure from Urban Gardens and Soils; In : Proceedings of a Symposium on Heavy Metals in Urban Gardens, Agricultural Experiment Station, University of the District of Columbia, James M. Preer, ed.

DTSC. 1990. Default Risk Assessment Formulas for Determination of Soil Remediation Levels, California Environmental Protection Agency. Department of Toxic Substances Control. Draft.

Driver, J. H., J. J. Konz, and G. K. Whitmyre. 1989. Soil adherence to human skin. Bull. Environ. Contam. Toxicol. 43: 814-820.

FDA. 1990. Contaminants Team, Division of Toxicological Review and Evaluation, Food and Drug Administration, Public Health Service, U.S. Department of Health and Human services; Memorandum to Elizabeth Campbell, Division of Regulatory Guidance.

Moore, M. R., P. A. Meridith, W. S. Watson, D. J. Summer, M. K. Taylor, and A. Goldberg. 1980. The percutaneous absorption of lead-203 in humans from cosmetic preparations containing lead acetate, as assessed by whole-body counting and other techniques. Food Cosmet. Toxicol. 18: 636.

Pennington, J. A. T. 1983. Revision of the total diet study food list and diets. J. Am. Dietetic Association, 82: 166.

Ryu, J. E., E. E. Ziegler, S. E. Nelson, and S. J. Fomon. 1983. Dietary intake of lead and blood lead concentration in early infancy. Am. J. Dis. Early Child. 137: 886.

USEPA. 1978. U.S. Environmental Protection Agency ; National Primary and Secondary Ambient Air Quality Standards for Lead. Fed. Regist. 43(194): 46246-46261.

USEPA. 1986. Environmental Criteria And Assessment Office, U.S. Environmental Protection Agency. Air Quality Criteria for Lead, EPA 600/8-83-028, June 1986.

USEPA. 1989a. Office of Emergency and Remedial Response, U.S. Environmental Protection Agency. Risk Assessment Guidance for Superfund, Volume 1; EPA 540/1-89-002 December, 1989.

USEPA. 1989b. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. Exposure Factors Handbook. EPA/600/8-89/043

USEPA. 1989c. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency. Review of the National Ambient Air Quality Standards for Lead, EPA 450/2-89-011 June 1989.

USEPA. 1991a. Office of Solid Waste and Emergency Response U.S. Environmental Protection Agency. Human Health Evaluation Manual, Supplemental Guidance : Standard Default Exposure Factors; OSWER Directive 9285.6-03.

USEPA. 1991b. U.S. Environmental Protection Agency. National Primary Drinking Water Quality Standards for Lead and Copper. Fed. Regist. 56(110): June 7, 1991, p. 26460.

USEPA. 1991c. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. Health Effects Assessment Summary Tables, NTIS No. PB91-921199, January 1991.

■ General information

Lead: health effects and sources of exposure

WHAT ARE THE SOURCES?
The main sources are petrol and paint. The main pathway of children's exposure is ingestion of contaminated dust and soil via normal hand-to-mouth activity.

TRANSPORT SOURCES
Lead functions as an octane enhancer and valve lubricant for pre-1986 petrol vehicles. It is also used in lead acid batteries and some aviation fuels. The use of leaded petrol has contributed to approximately 90% of lead in air pollution worldwide. In Australia, emissions from motor vehicle exhausts remain a major source of exposure for young children and the major source of chronic (long-term mild to moderate) lead poisoning through contamination of dust, soil and, to a lesser extent, water and food.

PAINT SOURCES
Because of renovation involving lead-containing paint, contaminated homes and yards are the major source of acute (short-term high-dose) lead poisoning. All old (pre-1970s) paints (including on metal surfaces) should be assumed to contain lead unless tests prove otherwise.

INDUSTRY
Lead mining, smelting and to a lesser extent, manufacturing industries, are other major sources of acute poisoning for those in the nearby community. Problems include atmospheric fallout and contaminated effluent and sewage sludge.

FOOD SOURCES
Contamination can occur in eggs, and fruit and vegetables grown near traffic or smelting or mining activity; and lead-soldered tinned acidic foods and ham. The average two-year old gets 60% of their food lead from whole grain foods, possibly due to the use of lead-contaminated fertilisers.

DRINKING WATER
Atmospheric input to surface waters can contribute

about 15% of the lead in drinking water. Water which is acidic and low in dissolved salts can leach substantial quantities in the first five years from PVC pipes, brass or bronze fittings or (illegal) lead solder. Lead-lined holding tanks in water coolers and other containers are further sources of contamination.

OTHER SOURCES

These include contaminated soil from previous use of lead arsenic pesticides; lead crystal; exposure to fumes in glassmaking or lead lighting; swallowing of fishing sinkers, lead shot, bullets or small electronic parts; herbal remedies containing lead, and cosmetics; emissions and ash from incinerators or crematoria; burning lead-painted wood or coal; and seepage from landfill sites.

WHAT ARE THE HEALTH EFFECTS?

The most sensitive parts of the body are the kidneys, the blood and the central nervous system. Because children are developing, they are more susceptible to the effects of even low levels, once thought to be safe. These effects include birth defects, reduced IQ, learning disabilities, stunted growth, hearing loss and behaviour problems.

LEAD AND CHILDREN

Children absorb lead efficiently - up to 50% of ingested lead, which compares to 10-15% in adults (the rest is excreted). Even a moderate amount can contribute significantly to a child's lead uptake. Children are most at risk between the ages of one and four when hand-to-mouth activity is greatest.

The US definition of childhood lead poisoning is a blood lead level over 10 micrograms per decilitre (10 ug/dL). If your blood lead result is in micromoles per litre (umol/L) multiply the number by 20.7 to convert it to ug/dL. In 1993 the National Health and Medical Research Council (NH&MRC) of Australia set the goal of a blood level of less than 10 ug/dl for every Australian.
An American research team found on average that for each three microgram drop there was a corresponding one-point improvement in the children's performance on IQ tests.

Blood lead levels in children from rural areas are lower than in urban areas. A 1994 NSW Health Department study estimates that around 70,000 NSW children aged between 0 and 4 years suffer from lead poisoning.

Symptoms of long-term exposure in adults and children include lower IQ, difficulties with visual motor functions and reaction times, psychological impairment, tiredness, inability to concentrate and low overall functioning. Because these symptoms may only become evident years after the child has been lead poisoned, regular checks on young children's blood lead levels and due care are the only way to monitor lead poisoning and take avoidance action.

HOW LEAD POISONING OCCURS
It can be inhaled, ingested or absorbed through skin which is wet with sweat or saliva. The main sources for young children are leaded petrol fallout and paint, via ingestion of dust and soil.

Children in homes undergoing renovation are between 2 and 12 times more likely to have blood lead levels over 15 ug/dL. When leaded paint is removed from houses, bridges or cars by dry removal techniques, it disperses into the atmosphere as flakes, dust, ash or fumes. However in urban areas, up to 90% of lead in the air is due to leaded petrol exhaust fumes. Fallout from leaded petrol is a major source of contamination in house dusts and soil.

[Back to Details]

Proudly designed and programmed by:
Social Change Online

Last Modified: *Friday, 27-Mar-1998 16:25:20 EST*

Toxic Chemicals in Your Environment
- a community based program of the Total Environment Centre

Web Editor: Jo Immig, email
toencen@magna.com.au

UPDATED VERSION OF THE CALIFORNIA EPA LEAD RISK ASSESSMENT SPREADSHEET MODEL FOR PREDICTING BLOOD LEAD IN CHILDREN AND ADULTS

James Carlisle, John Christopher, Brian Davis, Kimiko Klein, Barbara Renzi, and Michael Wade

California Department of Toxic Substances Control, Sacramento, CA, U.S.A.

ABSTRACT

The California Department of Toxic Substances Control has revised and updated its lead risk assessment spreadsheet model (LeadSpread) for predicting distributions of blood lead for adults and for children 1-2 years old. Inputs to LeadSpread are central tendency values; output is converted to a lognormal distribution via an assumed geometric standard deviation. We increased this geometric standard deviation to 1.60, according to White et al. (1998). We decreased food consumption to 1.1 kg/day for children and 1.9 kg/day for adults (Bolger, 1996) and decreased our estimate of lead in the diet to 2.8 µg/kg of food for children and 1.6 µg/kg of food for adults (USFDA, 1996-97). Based on EPA guidance (USEPA, 1997), we increased soil ingestion rates to 100 mg/day for children and 50 mg/day for adults, decreased the ventilation rate for children to 6.8 m^3/day, and changed exposed skin surface to 2,900 cm^2 for children and 5,800 cm^2 for adults. Using recent guidance on dermal risk assessment (USEPA, 1998), we decreased soil-to-skin adherence from 1 mg/cm^2 for children and adults to 0.2 mg/cm^2 for children and 0.07 mg/cm^2 for adults. Using data from California Air Resource Board (CARB, 1999), we decreased our estimates of lead in air to 0.028 µg/m^3. Airborne respirable particulates were estimated at 1.5 µg/m^3, using emission modeling. Assuming 20 mg Pb/kg in soil and 15 µg Pb/L in drinking water, these revised inputs to LeadSpread predict a geometric mean blood lead concentration of 1.7 µg/dL for children 1-2 years old, with a 99th percentile of 5.2 µg/dL. The National Health and Nutrition Examination Survey III, Phase 2 (NHANES III; USDHHS, 1996) found the geometric mean blood lead concentration in the Western U.S. to be 2.2 for children 1-6 years old and 2.6 in children 1-2 years old. Restricting the data from NHANES III to children living in post-1973 housing, geometric mean blood lead concentration decreased to 1.7 and 1.9 µg/dL for children 1-6 and 1-2 years old, respectively. Thus, LeadSpread with its revised inputs agrees well with NHANES III data for children either 1-2 or 1-6 years old in post-1973 housing. We also predicted blood lead concentrations using LeadSpread with various combinations of possible site-specific inputs.

LEADSPREAD REVISIONS

The California Department of Toxic Substances Control maintains a lead risk assessment spreadsheet model (LeadSpread) for predicting distributions of blood lead concentration in adults and in children 1-2 years old. Inputs to LeadSpread are central tendency values; output is converted to a lognormal distribution via an assumed geometric standard deviation. The Department has recently revised the model by reformatting the spreadsheet and by replacing several default input parameters to reflect more recent information. The revised model parameters are shown below.

DEFAULT INPUT PARAMETER VALUES

General Parameters	Units	Previous	Revised	Reference
Geometric Std. Deviation	Unitless	1.42	1.60	White et al.,1998
Background airborne lead	µg/m^3	0.18	0.028	CARB,1999
Source-specific airborne dust	µg/m^3	50	1.5	Cowherd, 1985
Lead in drinking water	µg/L	15	15	MCL
% Diet home-grown (resident)	%	5.5	7	USEPA, 1997
% Diet home-grown (worker)	%	0	0	

Child Parameters	Units	Previous	Revised	Reference
Daily food consumption	kg/day	1.3	1.1	Bolger, 1996
Dietary lead	µg/kg	10	2.8	USFDA, 1996-97
Soil ingestion	mg/day	55	100	USEPA, 1997
Soil ingestion, pica child	mg/day	790	200	USEPA, 1997
Ventilation rate	m^3/day	10	6.8	USEPA, 1997
Exposed skin area	cm^2	2,800	2,900	USEPA, 1997
Soil-to-skin adherence	mg/cm^2	1	0.2	USEPA, 1998

Adult Parameters	Units	Previous	Revised	Reference
Daily food consumption	kg/day	2.2	1.9	Bolger, 1996
Dietary lead	µg/kg	10	1.6	USFDA, 1996-97
Soil ingestion	mg/day	25	50	USEPA, 1997
Exposed skin area, resid.	cm^2	3,700	5,800	USEPA, 1997
Soil-to-skin adherence	mg/cm^2	1	0.07	USEPA, 1998

RESULTS USING REVISED MODEL

We ran LeadSpread with various combinations of possible site-specific inputs to illustrate its responses to changes in key variables. The following tables illustrate some of these predictions. In each table, the non-default model inputs are highlighted. Poster 342 shows model response to stepwise changes in key input parameters.

TYPICAL CHILD

INPUTS					OUTPUTS			
					Blood lead (µg/dL)		Soil concentration (mg/kg) Corresponding to 10 µg/dL	
Lead in soil (mg/kg)	Home-grown food (% diet)	Lead in water (µg/L)	Airborne Lead (µg/m^3)	PM_{10} (µg/m^3)	95th percentile	99th percentile	95th percentile	99th percentile
20	7%	15	0.028	1.5	3.8	5.2	247	146
1000	7%	15	0.028	1.5	30.6	42.3	247	146
20	0%	15	0.028	1.5	3.6	5.0	435	255
20	7%	5	0.028	1.5	2.4	3.3	298	197
20	7%	15	0.1	1.5	4.0	5.5	240	139
20	7%	15	0.028	50	3.8	5.2	246	145

PICA CHILD

INPUTS					OUTPUTS			
					Blood lead (µg/dL)		Soil concentration (mg/kg) Corresponding to 10 µg/dL	
Lead in soil (mg/kg)	Home-grown food (% diet)	Lead in water (µg/L)	Airborne Lead (µg/m^3)	PM_{10} (µg/m^3)	95th percentile	99th percentile	95th percentile	99th percentile
20	7%	15	0.028	1.5	4.1	5.7	159	94
1000	7%	15	0.028	1.5	45.8	63.3	159	94
20	0%	15	0.028	1.5	3.9	5.4	218	128
20	7%	5	0.028	1.5	2.4	3.3	191	126
20	7%	15	0.1	1.5	4.3	5.9	154	89
20	7%	15	0.028	50	4.1	5.7	158	94

ADULT (RESIDENTIAL EXPOSURE)

INPUTS					OUTPUTS			
					Blood lead (µg/dL)		Soil concentration (mg/kg) Corresponding to 10 µg/dL	
Lead in soil (mg/kg)	Home-grown food (% diet)	Lead in water (µg/L)	Airborne Lead (µg/m^3)	PM_{10} (µg/m^3)	95th percentile	99th percentile	95th percentile	99th percentile
20	7%	15	0.028	1.5	2.5	3.5	1062	676
1000	7%	15	0.028	1.5	9.6	13.2	1062	676
20	0%	15	0.028	1.5	2.5	3.4	3793	2407
20	7%	5	0.028	1.5	1.3	1.8	1230	844
20	7%	15	0.1	1.5	2.8	3.8	1026	640
20	7%	15	0.028	50	2.5	3.5	1037	660

ADULT (OCCUPATIONAL EXPOSURE)

INPUTS				OUTPUTS			
				Blood lead (µg/dL)		Soil concentration (mg/kg) Corresponding to 10 µg/dL	
Lead in soil (mg/kg)	Lead in water (µg/L)	Airborne Lead (µg/m^3)	PM_{10} (µg/m^3)	95th percentile	99th percentile	95th percentile	99th percentile
20	15	0.028	1.5	2.4	3.3	5,452	3,468
1000	15	0.028	1.5	3.8	5.2	5,452	3,468
20	5	0.028	1.5	1.2	1.7	6,320	4,335
20	15	0.1	1.5	2.6	3.6	5,322	3,337
20	15	0.028	50	2.4	3.3	5,011	3,187

VALIDATION

We compared the revised LeadSpread predictions under baseline conditions (20 mg Pb/kg soil; 15 µg Pb/L drinking water) with National Health and Nutrition Examination Survey (NHANES III) regional survey data (USDHHS, 1996). The results, shown below, indicate reasonable agreement between LeadSpread predictions and NHANES III data for children 1-2 or 1-6 years of age living in post-1973 housing in the Western United States.

Indicator	Median Blood lead concentration (µg/dL)
LeadSpread with 20 mg Pb/kg soil and 15 µg Pb/L drinking water	1.7
NHANES III data for the Western United States:	
Children 1-6 years	2.2
Children 1-2 years	2.6
Children 1-6 living in post-1973 housing	1.7
Children 1-2 living in post-1973 housing	1.9

CONCLUSIONS

The California DTSC has revised its lead risk assessment spreadsheet model (LeadSpread) for predicting distributions of blood lead concentration in adults and in children 1-2 years old. The revised model predicts slightly lower blood lead concentrations with all parameters set at default values. Blood lead predictions using the revised version of LeadSpread agree reasonably well with NHANES III data for children 1-2 or 1-6 years of age living in post-1973 housing in the Western United States.

REFERENCES

Bolger, P. M., N.J. Yess, E.L. Gunderson, T.C. Troxell, and C. D. Carrington, 1996. Identification and Reduction of Sources of Dietary Lead in the United States, Food Additives and Contaminants 13: 53-60.

California Air Resource Board, Technical Support Division, 1999. California Air Quality Data for 1997.

Cowherd, C., G. Muleski, P. Englehart, and D. Gillette, 1985. Rapid Assessment of Exposure to Particulate Emission from Surface Contamination. EPA/600/8-85/002. (NTIS #PB85-1922197AS).

U.S. Department of Health and Human Services, 1996. National Health and Nutrition Examination Survey III, Phase 2.

U. S. Environmental Protection Agency, 1997. Exposure Factors Handbook.

U. S. Environmental Protection Agency, 1998. Guidance for Dermal Risk Assessment.

U.S. Food and Drug Administration, 1999. Personal Communication from M. Bolger.

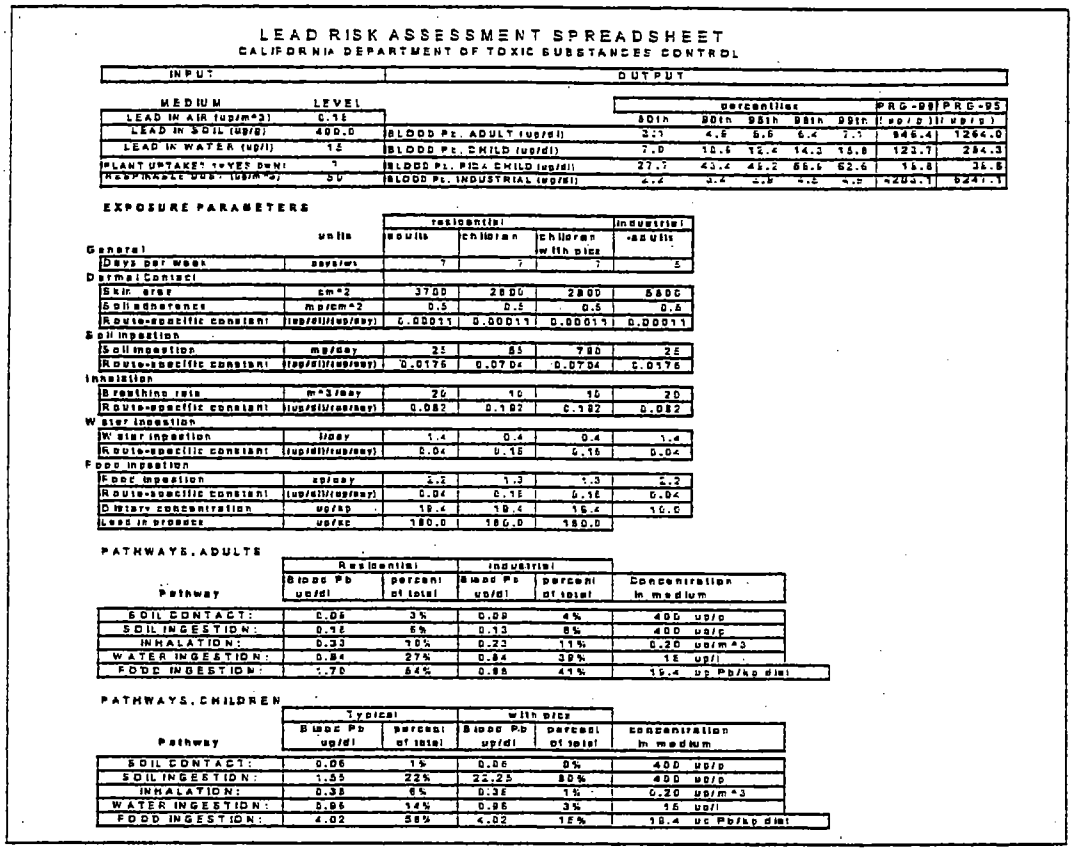
U.S. Food and Drug Administration, 1996-97. Total Diet Study.

White, P.D., P. VanLeeuwen, B.D. Davis, M. Maddaloni, K.A. Hogan, A.H. Marcus, and R.W. Elias, 1998. Environ. Health Perspectives 106, Suppl. 6: 1513.

SAMPLE SPREADSHEETS

Although the basic equations remain essentially the same, version 7 of the spreadsheet, employs new formatting and layout. It also collapses multiple terms into "pathway exposure factors" (PEF), and removes embedded factors from equations, making them visible in dedicated cells. The two versions of the spreadsheet are compared below.

Leadspread Version 6



LEAD RISK ASSESSMENT SPREADSHEET
CALIFORNIA DEPARTMENT OF TOXIC SUBSTANCES CONTROL

INPUT

MEDIUM	LEVEL
LEAD IN AIR (ug/m^3)	0.15
LEAD IN SOIL (ug/g)	400.0
LEAD IN WATER (ug/l)	15
%PLANT UPTAKE% (% YES OWN)	7
RESPIRABLE DUST (ug/m^3)	50

OUTPUT

	percentiles					PRG-98	PRG-95
	50th	90th	95th	98th	99th	(ug/g)	(ug/g)
BLOOD Pb, ADULT (ug/dl)	3.1	4.6	5.6	6.4	7.1	946.4	1264.0
BLOOD Pb, CHILD (ug/dl)	7.0	10.6	12.4	14.3	15.8	123.7	284.3
BLOOD Pb, PICA CHILD (ug/dl)	27.7	43.4	48.2	55.5	62.6	15.8	36.8
BLOOD Pb, INDUSTRIAL (ug/dl)	4.4	6.4	7.8	9.0	9.9	>2000.1	5241.1

EXPOSURE PARAMETERS

	units	residential: adults	residential: children	residential: children with pica	industrial: adults
General					
Days per week	days/wk	7	7	7	5
Dermal Contact					
Skin area	cm^2	3700	2800	2800	5800
Soil adherence	mg/cm^2	0.5	0.2	0.5	0.5
Route-specific constant	(ug/dl)/(ug/day)	0.00011	0.00011	0.00011	0.00011
Soil ingestion					
Soil ingestion	mg/day	25	85	700	25
Route-specific constant	(ug/dl)/(ug/day)	0.0175	0.07 Dx	0.0704	0.0175
Inhalation					
Breathing rate	m^3/day	20	10	10	20
Route-specific constant	(ug/dl)/(ug/day)	0.082	0.182	0.182	0.082
Water ingestion					
Water ingestion	l/day	1.4	0.4	0.4	1.4
Route-specific constant	(ug/dl)/(ug/day)	0.04	0.15	0.15	0.04
Food ingestion					
Food ingestion	kg/day	2.2	1.3	1.3	2.2
Route-specific constant	(ug/dl)/(ug/day)	0.04	0.15	0.15	0.04
Dietary concentration	ug/kg	19.4	19.4	19.4	19.0
Lead in produce	ug/kg	180.0	180.0	180.0	

PATHWAYS, ADULTS

Pathway	Residential: Blood Pb ug/dl	Residential: percent of total	Industrial: Blood Pb ug/dl	Industrial: percent of total	Concentration in medium
SOIL CONTACT:	0.09	3%	0.09	4%	400 ug/g
SOIL INGESTION:	0.15	5%	0.13	6%	400 ug/g
INHALATION:	0.33	10%	0.23	11%	0.20 ug/m^3
WATER INGESTION:	0.84	27%	0.84	39%	15 ug/l
FOOD INGESTION:	1.70	54%	0.88	41%	19.4 ug Pb/kg diet

PATHWAYS, CHILDREN

Pathway	Typical: Blood Pb ug/dl	Typical: percent of total	with pica: Blood Pb ug/dl	with pica: percent of total	Concentration in medium
SOIL CONTACT:	0.06	1%	0.06	0%	400 ug/g
SOIL INGESTION:	1.55	22%	22.25	80%	400 ug/g
INHALATION:	0.38	6%	0.38	1%	0.20 ug/m^3
WATER INGESTION:	0.96	14%	0.96	3%	15 ug/l
FOOD INGESTION:	4.02	58%	4.02	15%	19.4 ug Pb/kg diet

LeadSpread Version 7

LEAD RISK ASSESSMENT SPREADSHEET
CALIFORNIA DEPARTMENT OF TOXIC SUBSTANCES CONTROL

USER'S GUIDE to version 7

INPUT

MEDIUM	LEVEL
Lead in Air (µg/m³)	0.026
Lead in Soil/Dust (µg/g)	20.0
Lead in Water (µg/l)	15
% Home-grown Produce	7%
Respirable Dust (µg/m³)	1.5

OUTPUT

	Percentile Estimate of Blood Pb (ug/dl): 50th	90th	95th	98th	99th	PRG-98 (µg/g)	PRG-95 (µg/g)
Blood Pb, adult	1.2	2.1	2.5	3.1	3.5	675	1063
Blood Pb, child	1.8	3.2	3.8	4.6	5.2	146	247
Blood Pb, pica child	1.9	3.5	4.1	5.0	5.7	94	159
Blood Pb, occupational adult	1.1	2.0	2.4	2.9	3.3	3475	5464

EXPOSURE PARAMETERS

	units	adults	children
Days per week	days/wk	7	
Days per week, occupational		5	
Geometric Standard Deviation		1.6	
Blood lead level of concern (µg/dl)		10	
Skin area, residential	cm²	5700	2900
Skin area occupational	cm²	2900	
Soil adherence	µg/cm²	70	200
Dermal uptake constant	(ug/dl)/(ug/day)	0.0001	
Soil ingestion	mg/day	50	100
Soil ingestion, pica	mg/day		200
Ingestion constant	(ug/dl)/(ug/day)	0.04	0.15
Bioavailability	unitless	0.44	
Breathing rate	m³/day	20	6.8
Inhalation constant	(ug/dl)/(ug/day)	0.08	0.192
Water ingestion	l/day	1.4	0.4
Food ingestion	kg/day	1.9	1.1
Lead in market basket	ug/kg	3.1	
Pb in home-grown produce	ug/kg	9.0	

PATHWAYS

ADULTS	Residential: PEF	Residential: µg/dl	Residential: percent	Occupational: PEF	Occupational: µg/dl	Occupational: percent
Pathway		Pathway contribution			Pathway contribution	
Soil Contact	3.8E-5	0.00	0%	1.4E-5	0.00	0%
Soil Ingestion	8.8E-4	0.02	2%	6.3E-4	0.01	1%
Inhalation, bkgrnd		0.05	4%		0.03	3%
Inhalation	2.5E-6	0.00	0%	1.8E-6	0.00	0%
Water Ingestion		0.84	72%		0.84	75%
Food Ingestion, bkgrnd		0.22	19%		0.23	21%
Food Ingestion	2.4E-3	0.05	4%			0%

CHILDREN	typical: PEF	typical: µg/dl	typical: percent	with pica: PEF	with pica: µg/dl	with pica: percent
Pathway		Pathway contribution			Pathway contribution	
Soil Contact	5.5E-5	0.00	0%		0.00	0%
Soil Ingestion	7.0E-3	0.14	8%	1.4E-2	0.25	15%
Inhalation	2.0E-6	0.00	0%		0.00	0%
Inhalation, bkgrnd		0.04	2%		0.04	2%
Water Ingestion		0.96	55%		0.86	51%
Food Ingestion, bkgrnd		0.50	28%		0.50	27%
Food Ingestion	5.5E-3	0.11	6%		0.11	6%

PROGRAM IN ARSENIC HEALTH EFFECTS RESEARCH

School of Public Health and

Center for Occupational and Environmental Health

University of California, Berkeley

Program Director

Allan H Smith, MD, PhD, Professor of Epidemiology, ahsmith@uclink4.berkeley.edu

Research Team

Mary Lou Biggs, MPH, Research Associate, mlbiggs@u.washington.edu
Rajiv Bhatia, MD, MPH, Research Associate, rajiv_bhatia@dph.sf.ca.us
Joyce Chung, MPH, Doctoral Student in Epidemiology, jochung@uclink4.berkeley.edu
Reina Haque, MPH, Ph.D. Candidate, rhaque@socrates.berkeley.edu
Alexandra Hernandez, BA, Graduate Student Researcher, halexa@uclink4.berkeley.edu
Claudia Hopenhayn-Rich, PhD, Research Associate, cmhope0@pop.uky.edu
Peggy Lopipero, MPH, Research Associate, plopipero@earthlink.net
Lee Moore PhD, MPH Research Fellow, lmoore@cc.uscf.edu
Sandra Nuñez, BA, Research Associate, nunez@uclink4.berkeley.edu
Craig Steinmaus, MD, MPH, Research Associate, kingboho@pol.net

Co-Investigators at University of California, Berkeley

Gladys Block, PhD, Professor of Epidemiology and Public Health Nutrition
Steve Selvin, PhD, Professor of Biostatistics
Martyn Smith, PhD, Professor of Environmental Toxicology; Director, NIEHS Superfund Basic Research Program at the University of California, Berkeley

Collaborating Institutions and Research Scientists: (see page 3)

Mailing Address

School of Public Health
Division of Environmental Health Sciences
University of California, Berkeley
140 Warren Hall
Berkeley, California 94720-7360

Telephone: (510) 843-1736
Fax: (510) 843-5539

CONTENTS

I. INTRODUCTION

The purpose of this document is to present the Program in Arsenic Health Effects Research based at the University of California, Berkeley. These research activities began nearly ten years ago with a risk assessment for arsenic in drinking water. The realization that potential risks were high led to a program of arsenic research, including epidemiologic studies of various designs which are being undertaken among exposed populations in several countries.

II. MAJOR ACCOMPLISHMENTS

- Provided definitive evidence (from studies conducted in Argentina and Chile) that arsenic is a potent cause of human bladder cancer.

- Provided definitive evidence (from studies conducted in Argentina and Chile) that arsenic is a potent cause of human lung cancer.

- Demonstrated results which indicate that epidemiological and experimental human data do not support the methylation hypothesis.

- Showed that with exposure to water containing around 600 μg/L, 1 in 10 adult cancer deaths may be due to arsenic-caused cancers, the highest environmental cancer risk ever reported.

- Identified a dose-response relationship between arsenic exposure and bladder cell micronuclei, a genotoxic marker of effect.

- Identified a preliminary dose-response relationship between arsenic concentration in well water in India and the occurrence of keratoses and hyperpigmentation.

- Studies currently underway in India, Chile and the US, will allow projection of cancer risks with individual exposure data.

III. COLLABORATING INSTITUTIONS AND RESEARCH SCIENTISTS

United States

University of Washington, Seattle. *Professor David A. Kalman*, Director, Environmental Health Laboratory and Trace Organics Analysis Center, Department of Environmental Health.

University of California, San Francisco. *Professor Frederic Waldman*, Department of Laboratory Medicine, Division of Molecular Cytometry, and *Professor John K. Wiencke*, Department of Epidemiology and Biostatistics.

University of Colorado, Denver. *Professor Michael J. Kosnett*, Division of Clinical Pharmacology and Toxicology, Health Sciences Center.

Chile
Instituto de Salud Pública, Santiago, Chile. Ing. *Nella Marchetti*, Depto. de Salud Ocupacional y Contaminación Ambiental (currently at the Comisión Nacional del Medio Ambiente).

Dra. Catterina Ferreccio, Universidad Catolica, Santiago, Chile.

Servicio de Salud Antofagasta, Chile. *Dr. Mario Goycolea Chaparro and Dr. Alex Arroyo Meneses* (currently Secretario Regional del Ministerio de Salud in Region II).

Argentina
Universidad Católica de Córdoba, *Professor Ruben Sambuelli, Dean Esteban Trakal*

Dr. Omar Rey, Pathologist, Villa María; *Dr. Luis Sotelo,* Pathologist, Bell Ville; *Ing. Celia Loza,* Soil Chemist, Belle Ville, Cordoba, Argentina.

Dr. Analía Fuchs, Centro de Investigaciones Epidemiológicas, Academia Nacional de Medicina, Buenos Aires; *Dr. Remo Bergoglio*, Universidad Nacional de Córdoba and Academia de Ciencias Médicas de Córdoba, Córdoba; *Dr. Enrique E. Tello*, Universidad Nacional de Córdoba, Facultad de Ciencas Médicas, Córdoba; *Dr. Hugo Nicolli*, Instituto de Geoquímica, BuenosAires

India
Institute of Post Graduate Medical Education and Research, Calcutta, India. Dr. *D.N. Guha Mazumder, Dr. Nilima Gosh, Dr. Binoy K. De, Dr. Amal Santra.*

IV. FUNDING SOURCES

The main source of funding, which initiated the research program, has been the National Institute of Environmental Health Sciences (NIEHS) Superfund Basic Research Program at the University of California, Berkeley (Professor Martyn Smith, Director). NIEHS has funded the completed projects in Nevada and Chile and is currently funding the Argentina projects, No. P42-ES04705.

Seed funding for several projects has been provided through the NIEHS Center at Berkeley (Professor Bruce Ames, Director). No. P30-ES01896.

The initial risk assessment project was supported by the California Department of Health Services (Now the California Environmental Protection Agency or Cal/EPA).

The Nevada/California bladder cancer case-control study is funded by NIEHS Grant No: ES07459.

The planning of low exposure epidemiological studies was funded by the American Water Works Association Research Foundation (AWWARF).

The collaborative work with the Post Graduate Medical Institute in analysis of the cross-sectional study of arsenic-caused skin lesions was supported in part by the U.S. Environmental Protection Agency (EPA) National Center for Environmental Assessment.

The Dose-Response Study of Arsenic-Caused Skin Lesions in West Bengal, India, is funded by the U.S. EPA, No. R-826137-01-0.

The first planning of the Nevada/California bladder cancer case-control study was funded by a grant from the U.S. EPA.

Support for several students who worked on these projects was received from the Health Effects Component of the University of California Toxic Substances Teaching and Research Program.

Dr. Lee Moore has been supported by a research fellowship from the National Institute of Health (NIH) and the American Cancer Society.

The Center for Occupational and Environmental Health (COEH), University of California, Berkeley, provides salary support for Professors Allan Smith and Martyn Smith. COEH has also provided seed funding for early projects.

IV. CURRENT RESEARCH PROJECTS

1. Bladder cancer case-control study in Córdoba, Argentina

This study is in progress with an office and staff based in Villa María, Córdoba. The study is defined by 3 major components; 1) Arsenic and bladder cancer dose-response: Bladder cancer cases and age-sex matched population controls from the County of Unión are being interviewed in detail including lifelong residential histories, sources of drinking water and smoking histories. Water samples are being collected from both the current residences and previous residences where possible. Historical data on arsenic measurements in public water supplies are also being collected. We will conduct dose-response analyses incorporating individual exposure data, and examine the possible synergistic effect of cigarette smoking. 2) Metabolism: First-morning urine samples are being collected from cases and controls. Analysis of inorganic arsenic and its methlyated metabolites will be conducted in the laboratory of Professor David Kalman, University of Washington. Cases and controls will be compared to see if they differ in arsenic methylation patterns. 3) Molecular epidemiology: Tumor DNA is being analyzed for genetic alterations using a three-tiered approach: First, screening of the entire genome for gains and losses using comparative genomic hybridization (CGH); Second, specific analyses of chromosomes 9 and 17p for loss of heterozygosity using PCR-based methods; Third, analysis of the p53 gene for mutations using polymerase chain reaction-single-strand conformation (PCR-SSCP). The frequency and pattern of these genetic alterations in bladder tumors of arsenic

exposed and unexposed cases is being compared, and the potential synergistic action of arsenic on genotoxic effects of cigarette smoking is being assessed. In addition, susceptibility differences between cases and controls is being investigated by identifying the presence or absence of the glutathione S-transferases GSTM1 and GSTT1 null genotypes in buccal cells and by comparing urinary arsenic methylation patterns.

2. Bladder cancer case-control study in Nevada and California

The California/Nevada bladder cancer study is a population-based, case-control study that will examine the hypothesis that bladder cancer is caused by ingestion of arsenic in drinking water at relatively low concentrations. The study population includes residents of Kings County in California, and six counties in Nevada (Churchill, Mineral, Lyon, Douglas, Storey and Carson). These counties were chosen because they include water supplies containing close to 100 μg/L of arsenic, the highest level of arsenic found in major water supplies in the U.S.. Other water supplies in the study region contain less than 10 μg/L and thus provide a marked contrast in exposure. Two hundred bladder cancer cases diagnosed between 1994 and 2000 will be identified from the California and Nevada Tumor Registries. Random digit dial (RDD) will be used to identify 400 controls who will be frequency matched to cases by sex and 5-year age groups. Structured personal telephone interviews will be administered to obtain lifetime residential history and detailed information on current and past water consumption patterns. Information will also be obtained regarding cigarette smoking (which may be synergistic with arsenic in causing bladder cancer), chlorination of drinking water, diet, and occupational history. Although carcinogenicity of arsenic at 100 μg/L is uncertain, this study has over 90% statistical power to detect a relative risk of 2.0 which was predicted by linear extrapolation of data from studies in Taiwan.

3. Argentina mortality study

Mortality from internal cancers was identified in areas of the Province of Córdoba, Argentina, which in the past had high levels of arsenic in drinking water. The results concerning bladder cancer have been published (see publication 15). The analyses concerning mortality from other cancers is completed and a manuscript describing the results has been published (see publication 26). Increased rates of kidney and lung cancer were found in the exposed areas, as were the already reported increases in bladder cancer.

4. Dose-response study of arsenic-caused skin lesions in West Bengal, India

Research is being conducted in collaboration with Professor D.N. Guha Mazumder and his research team at the Institute of Post Graduate Education and Research (IPGMER) in Calcutta, India. Our group collaborated with the analysis of data from a large cross-sectional survey of about 7000 people in an arsenic-exposed region in West Bengal. The dose-response analysis linking cases of skin keratoses and hyperpigmentation to arsenic water levels has been recently published (see publication 27). The next phase is a case-control study nested in the same survey, which focuses on participants with skin lesions who had drinking water arsenic levels of less than 500 μg/L. Detailed interviews concerning water sources and fluid consumption, diet,

smoking and medical history are being completed for each participant. Water samples are obtained from all drinking water sources. Each participant receives a physical examination for skin lesions and other signs, portable spirometry, and blood and urine samples are obtained to assess micronutrients and arsenic metabolism. The study is funded by the U.S. EPA.

V. RESEARCH PUBLICATIONS WITH SUMMARIES OF KEY FINDINGS

1. Frost F, Harter L, Milham S, Royce R, Smith AH, Hartley J, Enterline P. Lung cancer among women residing close to an arsenic-emitting copper smelter. Arch Env Health 42:148-52, 1987.

Lung cancer mortality. This project was conducted with the Chronic Disease Epidemiology Section of the Washington State Division of Health. Overall lung cancer mortality rates were not increased among women living near the smelter. However, case-control analysis using an index of exposure based on distance of residence from the smelter showed increasing lung cancer odds ratios from 1 up to 1.6 for those in the highest quintile of potential exposure. The results are consistent with a small elevated lung cancer risk for women who resided close to the smelter for a period of over 20 years. (Note: There is an error in Table 6 - the lines for cases and controls are transposed).

2. Hertz-Picciotto I, Smith AH, Holzman D, Lipsett M, Alexeef G. Synergism between occupational arsenic exposure and smoking in the induction of lung cancer. Epidemiol 3:23-31, 1992.

Synergy. Data were assembled from epidemiological studies concerning inhalation of inorganic arsenic and cigarette smoking. It was concluded that the evidence for synergism between the two exposures was compelling. Various potential mechanisms for synergy were discussed.

3. Smith AH, Hopenhayn-Rich C, Bates MN, Goeden HM, Hertz-Picciotto I, Duggan HM, Wood R, Smith MT, Kosnett MJ. Cancer risks from arsenic in drinking water. Env Health Persp 97:259-67, 1992.

Risk assessment. Evidence that ingestion of inorganic arsenic in drinking water might cause bladder, kidney, lung and liver cancer was examined, and potential cancer risks were calculated for various levels of exposure. It was estimated that at the current standard of 50μg/L, the lifetime risk of dying from one of these cancers could be as high as 13 per 1000 persons. It was noted that existing studies did not support a threshold based on arsenic methylation. It was concluded that although further research was needed to validate the findings of the risk assessment, measures should be taken to reduce arsenic levels in drin king water.

4. Bates MN, Smith AH, Hopenhayn-Rich C. Arsenic ingestion and internal cancers: a review. Am J Epidemiol 135:462-76, 1992.

Internal cancers. A detailed review of epidemiological studies concerning arsenic ingestion and internal cancers was presented. The most informative studies were from Taiwan and it was concluded that these and other studies strongly suggest that ingested inorganic arsenic does cause cancers of the bladder, kidney, lung and liver, and possibly other sites.

5. Hopenhayn-Rich C, Smith AH, Goeden H. Human studies do not support the methylation threshold hypothesis for the toxicity of inorganic arsenic. Env Res 60:161-77, 1993.

Metabolism. The validity of the methylation threshold hypothesis was examined on the basis of published studies. The results indicated that epidemiological and experimental human data does not support the inorganic arsenic methylation threshold hypothesis. Regardless of the absorbed dose of inorganic arsenic, there was always some unmethylated inorganic arsenic present in the urine, even at background exposure levels. It was noted that lack of evidence for a methylation threshold based on the human exposure levels studied did not exclude the possibility of other threshold mechanisms. In addition, the considerable variation in methylation of inorganic arsenic observed between individuals was noted to warrant further study.

6. Hertz-Picciotto I, Smith AH. Observations on the dose-response curve for arsenic exposure and lung cancer. Scand J Work Env Health 19: 217-26, 1993.

Lung cancer dose-response. Information from published studies concerning arsenic inhalation and lung cancer risks was analyzed. It was found that all of the studies with quantitative data were consistent with a supralinear dose-response relationship. Various factors which might be distorting the true biological dose-response were assessed. These included the fact that the workers thought to be most highly exposed might actually have had lower exposures than previously quantified by air sampling as a result of non-random sampling and the possible use of respirators when air levels were highest. It was noted that there was a linear dose-response relationship in one study, which used urine arsenic measurements to assess exposure.

7. Smith AH, Hopenhayn-Rich C, Warner M, Biggs ML, Moore L, Smith MT. Rationale for selecting exfoliated bladder cells micronuclei as potential biomarkers for arsenic genotoxicity. J Toxicol Env Health 40: 223-34, 1993.

Molecular epidemiology. Biological markers of effect of toxic human exposures have the potential to allow exploration of dose-response relationships at levels of exposure lower than those which can be assessed by traditional epidemiological studies involving the ultimate disease end-point. In this paper we give reasons for proposing that exfoliated bladder cell micronuclei might be a good marker for carcinogenic effects of ingestion of inorganic arsenic. Based on studies in Taiwan, it was noted that the highest internal cancer relative risks involved bladder

cancer. Bladder cells can be collected from urine, and originate from a target organ of particular importance for arsenic effects. We described several studies from our group, which used bladder cell micronuclei as biomarkers, noting the important potential contribution of intervention studies incorporating cessation of exposure.

8. Warner M, Moore L, Smith MT, Kalman D, Fanning E, Smith AH. Increased micronuclei in exfoliated bladder cells of persons who chronically ingest arsenic contaminated water in Nevada. Cancer Epidemiol Biom & Prev 3:583-90, 1994.

Molecular epidemiology. This study involved 18 subjects in Nevada whose well water contained on average 1312 μg/L of arsenic, and 18 age and sex matched controls whose well water averaged 16 μg/L. Exposed subjects had a 1.8 fold increase in bladder cell micronuclei, but the differences were largely confined to males. The absence of findings for females was thought to be due to the fact that women exfoliate large numbers of cells into urine, while men exfoliate predominantly transitional cells, which are the cells involved in bladder cancer. No increase was found in buccal cell micronuclei among the arsenic exposed group.

9. Engel RR, Hopenhayn-Rich C, Receveur O, Smith AH. Vascular effects of chronic arsenic exposure: a review. Epidemiol Rev 16:184-209, 1994.

Vascular disease. Existing literature concerning vascular effects from chronic exposure to inorganic arsenic was reviewed in this publication containing 177 citations. It was concluded that there was good epidemiologic evidence indicating that chronic arsenic consumption at high levels is a cause of severe peripheral vascular disease with resulting gangrene and amputations of the limbs. We hypothesized that marginal zinc status might explain the differential occurrence of these conditions in populations ingesting large doses of arsenic. It was also concluded that it was plausible, though epidemiologic evidence is limited, that arsenic might cause increases in vascular mortality beyond that found in patients with severe peripheral vascular disease.

10. Engel RR, Smith AH. Arsenic in drinking water and mortality from vascular disease: an ecologic analysis in 30 U.S. counties. Arch Environ Hlth 49: 418-27, 1994.

Vascular disease. An investigation was made of the ecological relationship between arsenic concentrations in drinking water and mortality from circulatory disease in 30 U.S. counties from 1968 to 1984. Mean arsenic levels ranged from 5.4 to 91.5 μg/L. The standardized mortality ratios (SMRs) for diseases of arteries, arterioles, and capillaries for counties exceeding 20 μg/L were 1.9 (90% CI 1.7-2.1) for females and 1.6 (CI 1.5-1.8) for men. The SMRs for congenital anomalies of the heart and circulatory system were also elevated. Possible problems with the ecological study design and explanations for potentially spurious results were discussed. It was concluded that further investigation of vascular effects of arsenic exposure was warranted.

11. Smith AH, Hopenhayn-Rich C, Biggs ML, Moore L, Dale J, Warner M, Bates M, Engel R. Epidemiological study designs to address potential high bladder cancer risks from arsenic in drinking water. In: Chappell WR, Abernathy CO, Cothern CR, eds. Arsenic: Exposure and Health. Northwood: Science and Technology Letters, 109-17, 1994.

Epidemiological study designs. Various study designs were described which could be used to further investigate effects of arsenic ingestion from drinking water, including ecological studies, cohort studies, and biomarker studies. It was noted that small biomarker studies could be conducted relatively rapidly, and that the effect of interventions could be assessed for biomarkers in cells with short half-lives. However, interpretation of biomarker studies is difficult, consequently, traditional epidemiological study designs have an important role. It was concluded that the potential risks of bladder cancer from ingesting inorganic arsenic in drinking water warranted a concerted epidemiological approach using a variety of different study designs.

12. Bates MN, Smith AH, Cantor KP. Case-control study of bladder cancer and arsenic in drinking water. Am J Epidemiol 141: 523-30, 1995.

Bladder cancer. Cases and controls from the National Bladder Cancer Study were used in this project, which was conducted in collaboration with Dr. Ken Cantor of the National Cancer Institute. Information concerning arsenic levels in drinking water was added to this dataset for respondents from Utah. Water levels ranged from 0.5 to 160 μg/L, but only three towns were served with water containing over 20 μg/L of arsenic. There was no overall association of inorganic arsenic with the risk of bladder cancer at these levels of exposure. However, among cigarette smokers, time window analysis yielded some evidence for a dose-response relationship for exposure to arsenic in drinking water 10-39 years prior to diagnosis with bladder cancer. The possibility was raised that smoking potentiates the effect of arsenic in causing bladder cancer. However, the discrepancy between these findings at such low exposure levels, and predictions based on studies in Taiwan and England, also raised the possibility of bias in the data. It was concluded that further carefully conducted studies in exposed populations were needed.

13. Smith AH, Hopenhayn-Rich C, Biggs ML, Kalman D. Re: Arsenic risk assessment (letter). Env Health Persp 103:13-15, 1995.

Risk assessment. Heather Carlson-Lynch, Barbara Beck and Pamela Boardman of McLaren/Hart Environmental Engineering Corporation and Gradient Corporation wrote a letter which was highly critical of two of our published studies (Hopenhayn-Rich et al, 1993, and Smith et al, 1992, above). In the letter to the editor, we demonstrated that none of the criticisms raised was valid.

14. Moore L, Smith AH, Hopenhayn-Rich C, Biggs ML, Warner ML, Kalman D, Smith MT. Increased bladder cell micronuclei found in two populations environmentally exposed to arsenic in drinking water. Clin Chem 41:1915-17, 1995.

Molecular epidemiology. Summary findings from the Nevada bladder cell micronucleus study, with preliminary results from the Chile study, were reported. It was concluded that results from both the North and South American studies provided evidence that arsenic is genotoxic to human bladder epithelium. Further details are given in Warner et al, 1994 (publication 13) and Moore et al. 1997 (publication 15).

15. Hopenhayn-Rich C, Biggs ML, Fuchs A, Bergoglio R, Tello E, Nicolli H, Smith AH. Bladder cancer mortality associated with arsenic in drinking water in Argentina. Epidemiol. 7:117-124, 1996.

Bladder cancer. Bladder cancer mortality for the years 1986-1991 was investigated in Córdoba, Argentina in an ecological study comparing counties categorized as previously having high, medium and low water levels of arsenic. The average water arsenic level in the two high exposure counties for arsenic contaminated water sources was 178 μg/L. Clear trends in bladder cancer mortality were shown up to standardized mortality ratios (SMRs) of 2.14 for men (95% CI 1.78-2.53) and 1.82 for women (95% CI 1.19-2.64) in the two high exposure counties. The clear trends found in a population with a different ethnic composition and a high protein diet support the evidence from Taiwan that arsenic in drinking water is a cause of human bladder cancer. While it was made clear that exposure was not uniform within counties, it was noted the findings were roughly consistent with risks which might be predicted from the Taiwan studies.

16. Hopenhayn-Rich C, Biggs ML, Fuchs A, Bergoglio R, Tello E, Nicolli H, Smith AH. Arsenic and bladder cancer mortality. The Authors Reply. Epidemiol 7:557-58, 1996.

Bladder cancer. Kenneth G. Brown and Barbara D. Beck wrote a letter critical of the above study in which we were accused of making incorrect assumptions, errors and unwarranted conclusions. In this reply, we noted that we were surprised by their accusations of errors that did not, indeed, exist. However, we agreed with their statement, "the study does affirm the association of high concentrations of inorganic arsenic with increased mortality from bladder cancer, in this instance among the ethnically mixed Córdoba population, in the absence of nutritional deficiency or evidence of other substances such as humic or fluorescent substances".

17. Moore L, Warner ML, Smith AH, Kalman D, Smith MT. Use of the fluorescent micronucleus assay to detect the genotoxic effects of radiation and arsenic in human exfoliated epithelial cells. Env and Molecular Mutagen 27:176-84, 1996.

Molecular epidemiology. A new rapid method was used, which involves fluorescent in situ hybridization (FISH) to determine the mechanism of micronucleus formation in epithelial tissues

exposed to carcinogenic agents (as previously described in Titenko-Holland N, Moore LE, Smith MT. Measurement and characterization of micronuclei in exfoliated human cells by fluorescence in situ hybridization with a centromeric probe. Mutat Res 271:69-77, 1992.) The findings concerning micronuclei in exfoliated bladder cells obtained from arsenic-exposed subjects in Nevada suggested that arsenic may have both clastogenic and weak aneuploidogenic properties.

18. Hopenhayn-Rich C, Biggs ML, Smith AH, Kalman D, Moore LE. Methlyation study in a population environmentally exposed to high arsenic water. Env Health Persp 104:620-28, 1996.

Metabolism. Arsenic methylation patterns were investigated in this cross-sectional study of two towns in Chile. One hundred and twenty two people exposed to high levels of arsenic were compared to 98 people in a neighboring town with low levels of arsenic. Arsenic levels in drinking water were 600 ug/L and 15ug/L, respectively. The corresponding mean urinary arsenic levels were 580 μg/L and 60 μg/L, of which 18.4% and 14.9% were inorganic arsenic respectively. The main differences were found in the monomethyarsonate (MMA) to dimethylarsinate (DMA) ratio; high exposure, smoking, and being male were associated with higher MMA/DMA, while longer residence in the exposed town, Atacameno ethnicity, and being female were associated with lower MMA/DMA. Overall, there was no evidence of a threshold for methylation capacity, even at very high exposures. This study, which is the largest study conducted involving metabolites of arsenic to date, confirmed conclusions made in our earlier publications that the methylation threshold hypothesis was not valid.

19. Hopenhayn-Rich C, Biggs ML, Kalman D, Moore LE, Smith AH. Arsenic methylation patterns before and after changing from higher to lower concentrations of arsenic in drinking water. Env Health Persp 104:1200-07, 1996.

Metabolism. Presented are the results of an intervention study of 73 participants (from the above cross-sectional study in Chile), who were provided with water of lower arsenic content (45 μg/L) for two months. Total urinary arsenic levels fell from an average of 636 μg/L to 166 μg/L. There was a small decrease from 17.8% to 14.6% in the percent of urinary arsenic in inorganic form consistent with what might be predicted from the cross-sectional study. Other factors such as smoking, gender, age, years of residence, and ethnicity were associated mainly with changes in the MMA/DMA ratio. The main difference was found for smokers, where practically all of the smokers showed a decrease in the MMA/DMA ratio, while much more variability was seen for non-smokers. It was noted that the changes in the observed percent inorganic arsenic and in the MMA/DMA ratio did not support an exposure based threshold for arsenic methylation in humans. The last two studies (cross-sectional and intervention) also indicate that most of the inter-individual variability in the distribution of urinary metabolites remains unexplained.

20. Wright C, Lopipero P, Smith AH. Meta-analysis and Risk Assessment. In: Topics in Environmental Epidemiology. Eds. Steenland K and Savitz DA, Oxford University Press, 1996.

Risk assessment. Although arsenic is not discussed in this chapter, it is pertinent here because it includes issues and methods concerning the use of epidemiologic studies to estimate population risks at low levels of exposure. It was noted that apparent nonlinearity at low exposure points in studies can be fitted with statistical models that have a profound impact on risk extrapolations to lower doses. However, the empirical evidence for nonlinearity may be extremely weak, and there are often no good biological reasons for rejecting linearity. For these and other reasons, we stated that it would be preferable to use the linear relative risk model for quantitative risk assessment using epidemiologic data, *unless there are good reasons to reject it* (i.e. clear evidence of nonlinearity).

21. Moore LE, Smith AH, Hopenhayn-Rich C, Biggs ML, Kalman DA, Smith MT. Micronuclei in exfoliated bladder cells among individuals chronically exposed to arsenic in drinking water. Cancer Epidemiol Biom & Prev 6:31-6, 1997.

Molecular epidemiology. Using the same towns as the methylation study in Chile described in the previous publication summary, this cross-sectional study was confined to male participants in view of the extensive exfoliation of squamous cells as well as transitional bladder cells which occurs in females. There were 70 high-exposure participants (average urinary arsenic 616 μg/L) and 55 low-exposure participants (average urinary arsenic 66 μg/L). The prevalence of micronuclei increased three-fold (95% CI 1.9-4.6) from the lowest exposure quintile (less than 53.8 μg/L arsenic in urine) to those in the second highest exposure quintile (414-729 μg/L urinary arsenic). Surprisingly, those in the highest exposure quintile (more than 729 μg/L urinary arsenic) did not have any increase in micronucleus prevalence. This finding is not fully explained, but could be due to cytostasis or cytotoxicity at these high exposure levels. The centromeric probe classification of micronuclei suggested that chromosome breakage was the major cause of micronucleus formation. It is noteworthy that the prevalence of micronuclei in bladder cells was elevated even in the second to lowest quintile of exposure (urinary arsenic levels between 53.9 and 137.3 μg/L, prevalence ratio 2.1, 95% CI 1.4-3.4), which raises the possibility that arsenic has genotoxic effects on bladder cells at relatively low levels of exposure.

22. Biggs ML, Kalman DA, Moore LE, Hopenhayn-Rich C, Smith MT, Smith AH. Relationship of urinary arsenic to intake estimates and a biomarker of effect, bladder cell micronuclei. Mut Res 386:185-95, 1997.

Exposure assessment. The primary purpose of this study was to investigate methods for ascertaining arsenic exposure for use in biomarker studies. The study population was the same as the population in the metabolism and bladder cell micronucleus study conducted in Chile. Exposures were assessed by an interviewer-administered questionnaire concerning volumes and sources of fluid intake. Urinary inorganic arsenic measurements including methylated species

were measured in first-morning samples. Creatinine was measured to allow for adjustment for overly concentrated urine. As expected, creatinine adjusted urinary arsenic concentrations had a stronger relationship with the questionnaire-based estimates of arsenic intake than the unadjusted urinary concentrations. Interestingly, the unadjusted urinary arsenic measures had the stronger relationship with bladder cell micronucleus prevalence. This finding is plausible since the unadjusted urinary arsenic concentrations may better reflect target site dose to the bladder, which is exposed to the actual concentration of arsenic in urine.

23. Aposhian HV, Arroyo A, Cebrian M, Del Razo LM, Hurlbut KM, Dart RC, Gonzalez-Ramirez D, Kreppel H, Speiske H, Smith AH, et al. DMPS-Arsenic Challenge Test: I-Increased Urinary Excretion of Monomethylarsonic Acid in Humans Given Dimercaptopropane Sulfonate. J Pharm Exp Ther 282:192-200, 1997.

Chelation study. Directed by Professor Vasken Aposhian of the University of Arizona, this study involved a small subset of participants from our studies in Chile: 13 from the high-exposure town and 11 from the low-exposure town. Each participant was given 300 mg of the chelating agent 2,3-dimercaptone-1-sulfonic acid (DMPS). As expected, urinary arsenic concentrations increased in the 24-hour period after taking DMPS. Interestingly, the increase was considerably more pronounced for MMA than for inorganic arsenic and DMA. In our view, it is difficult to interpret these findings, since the tissue binding strengths of the various arsenic species may vary, and they may have different affinities for the chelating agent. For these and other reasons, urinary arsenic levels in chronically exposed persons remain the best indicators of body dose.

24. Moore, LE, Smith AH, Hopenhayn-Rich C, Biggs ML, Kalman DA, Smith MT. Decrease in bladder cell micronucleus prevalence after intervention to lower the concentration of arsenic in drinking water. Cancer Epidemiol Biomark and Prev 6:1051-6, 1997.

Molecular epidemiology. Water low in arsenic content (45 μg/L) was provided to 34 highly exposed participants in the cross-sectional study in Chile (publication 21 above). Mean urinary arsenic levels in this sub-group decreased from 742 to 225 μg/L during the intervention. Bladder cell micronucleus (MNC) prevalence decreased from 2.63/1000 to 1.79/1000 cells post-intervention ($p<0.05$). When the analysis was limited to individuals previously having subcytotoxic urinary arsenic levels (<700 μg/L), the change between pre- and post-intervention MNC was more pronounced: from 3.54 to 1.47/100 cells respectively ($p=0.002$). The primary changes occurred among smokers, suggesting that smoker's bladder cells could be more susceptible to genotoxic damage caused by arsenic. The reduction in bladder cell MNC prevalence with reduction in inorganic arsenic intake provides further evidence that arsenic is genotoxic to bladder cells.

25. Smith AH, Goycolea M, Haque R, Biggs ML. Marked increase in bladder and lung cancer mortality in a region of Northern Chile due to arsenic in drinking water. Am J Epidemiol, 147:660-69, 1998.

Cancer mortality. Studies in Taiwan and Argentina suggest that ingestion of inorganic arsenic from drinking water results in increased risks of internal cancers, in particular bladder and lung cancer. The authors investigated cancer mortality in a population of around 400,000 people in a region of Northern Chile (Region II) exposed to high arsenic levels in drinking water in past years. Arsenic concentrations from 1950 to the present were obtained. Population-weighted average arsenic levels reached 570 μg/L between 1955 to 1969, and decreased to less than 100 μg/L by 1980. Standardized mortality ratios (SMRs) were calculated for the years 1989 to 1993. Increased mortality was found for bladder, lung, kidney and skin cancer. Bladder cancer mortality was markedly elevated with an SMR of 6.0 [95% confidence interval 4.8-7.4] for men, and 8.2 [6.3-10.5] for women. Lung cancer SMRs were 3.8 [3.5-4.1] for men, and 3.1 [2.7-3.7] for women. Smoking survey data and mortality rates from chronic obstructive pulmonary disease provided evidence that smoking did not contribute to the increased mortality from these cancers. The findings provide additional evidence that ingestion of inorganic arsenic in drinking water is indeed a cause of bladder and lung cancer. It was estimated that arsenic might account for 7% of all deaths among those aged 30 and over. If so, the impact of arsenic on the population mortality in Region II of Chile is greater than any reported to date from environmental exposure to a carcinogen in a major population.

26. Hopenhayn-Rich C, Biggs ML, Smith AH. Lung and kidney cancer mortality associated with arsenic in drinking water in Cordoba, Argentina. Int J Epidemiol 27: 561-69, 1998.

Bladder cancer. Studies in Taiwan have found dose-response relations between arsenic ingestion from drinking water and cancers of the skin, bladder, lung, kidney and liver. To investigate these associations in another population, we conducted a study in Cordoba, Argentina, which has a well-documented history of arsenic exposure from drinking water. Mortality from lung, kidney, liver and skin cancers during the period 1986-1991 in Cordoba's 26 counties was investigated, expanding the authors' previous analysis of bladder cancer in the province. Counties were grouped a priori into low, medium and high arsenic exposure categories based on available data. Standardized mortality ratios (SMRs) were calculated using all of Argentina as the reference population. We found increasing trends for kidney and lung cancer mortality with arsenic exposure, with the following SMRs, for men and women respectively: kidney cancer, 0.87,1.33, 1.57 and 1.00, 1.36, 1.81; lung cancer, 0.92,1.54,1.77 and 1.24, 1.34, 2.16 (in all cases, $p<0.001$ in trend tests), similar to the previously reported bladder cancer results (0.80, 1.28, 2.14 for men, 1.22, 1.39, 1.81, for women). There was a small positive trend for liver cancer but mortality was increased in all three exposure groups. Skin cancer mortality was elevated for women in the high-exposure group, while men showed a puzzling increase in the low-exposure group. The results add to the evidence that arsenic ingestion increases the risk of lung and kidney cancers. In this study, the association between arsenic and mortality from liver and skin cancers was not clear.

27. Guha Mazumder DN, Haque R, Gosh N, De BK, Santra A, Chakraborty D, Smith AH. Arsenic levels in drinking water and the prevalence of skin lesions in West Bengal, India. Int J Epidemiol 27:871-77, 1998.

Skin lesions. A cross-sectional survey was conducted investigating the arsenic-caused skin lesions of keratoses and hyperpigmentation in West Bengal, India. There were 7683 participants who were examined and interviewed, and whose drinking water arsenic levels were measured. Water concentrations ranged up to 3400 ug/L of arsenic but over 80% of participants were consuming water containing less than 500 ug/L. The prevalence of keratoses was strongly related to water arsenic levels rising to 8.5 per 100 for females, and 10.7 per 100 for men, drinking water containing over 800 ug/L. However 12 cases with keratoses (2 females and 10 males) were drinking water containing less than 100 ug/L of arsenic. Findings were similar for hyperpigmentation with strong dose-response relationships, and with 29 cases drinking water containing less than 100ug/L. Calculation by dose per body weight showed that men had roughly two to three times the prevalence of both keratoses and hyperpigmentation compared to women ingesting the same dose of arsenic from drinking water. Subjects who were below 80% of the standard body weight for their age and sex had 1.6 fold increase in prevalence of keratoses, and a 1.2 fold increase in prevalence of hyperpigmentation suggesting that malnutrition might play a small role in increasing susceptibility. The surprising findings concerning cases with apparently low exposure need to be confirmed in studies with more detailed exposure assessment. Further research is also needed concerning susceptibility factors which might be present in the exposed population.

28. Steinmaus C, Moore LE, Hopenhayn-Rich C, Biggs ML, Smith AH. Arsenic in drinking water and bladder cancer. Cancer Invest. In press 1998.

Millions of people throughout the world are drinking water containing inorganic arsenic. Although initially controversial, the association between high exposures to ingested arsenic and bladder cancer is now well established. Unfortunately, the dose-response relationship, especially at low to moderate doses such as those found in the U.S., remains unclear. Attempts to define these risks and establish new drinking water regulations have been controversial, primarily due to questions regarding the risk assessment process used to establish these standards. Epidemiological studies involving low- to moderate- dose exposures will help to define these risks and aid in the establishment of appropriate drinking water regulations. In addition, genetic biomarker studies may provide information on the mechanistic and susceptibility issues of arsenic induced carcinogenesis, and thus may also help elucidate dose-response relationships at low doses. However, until a new arsenic drinking water standard is implemented, most evidence suggests that populations currently exposed to arsenic in drinking water will continue to have substantially elevated cancer risks. Waiting for more precise data before a new standard is applied will only prolong these risks. Therefore, until further research can be completed, an interim drinking water arsenic standard similar to the World Health Organization recommendation of 10 µg/L, may be appropriate.

29. Smith, AH, Arroyo A, Guha Mazumder DN, Kosnett MJ, Hernandez A, Beeris M, Smith MT, More LE. Arsenic-induced skin lesions among Atacameño people in Northern Chile despite good nutrition and centuries of exposure. Submitted, 1999.

It has been suggested that the indigenous Atacameño people in Northern Chile might be protected from the health effects of arsenic in drinking water because of many centuries of exposure. Here we report on the first intensive investigation of arsenic-induced skin lesions in this population. Eleven families were selected from the village of Chiu Chiu which is supplied with water containing between 750 and 800 *u*g/L of inorganic arsenic. For comparison, 8 families were also selected from a village where the water contains around 10 *u*g/L. After being transported to the nearest city so that assessment could be done blind as to drinking water source, participants were examined by four physicians with experience in studying arsenic-induced lesions. Four of the six men from the exposed village who had been drinking the contaminated water for more than 20 years were diagnosed with skin lesions due to arsenic, but no women were found to have definite lesions. A 13 year old girl was found to have definite skin pigmentation changes due to arsenic, and a 19 year old boy had both pigmentation changes and keratoses on the palms and soles. Family interviews identified a wide range of fruit and vegetable consumption among affected participants, plus weekly intake of red meat and chicken. However, the prevalence of skin lesions found among men and children was as high or higher than reported with corresponding arsenic drinking water concentrations in both Taiwan and West Bengal, India, populations in which extensive malnutrition has been thought to increase susceptibility.

Last updated August 17,1999

Screening For Environmental Concerns At Sites With Contaminated Soil and Groundwater

Volume 1: Summary Tier 1 Lookup Tables

Prepared by:
California Regional Water Quality Control Board
San Francisco Bay Region
1515 Clay Street, Suite 1400
Oakland, California 94612

INTERIM FINAL - July 2003
(updated 9/4/03)

Contact:
Roger D. Brewer, Ph.D.
Associate Engineering Geologist
Telephone: 1-510-622-2374
E-mail: rdb@rb2.swrcb.ca.gov

OR

Sampath Rangarajan
Water Resources Control Engineer
Telephone: 1-510-622-2381
E-mail: sr@rb2.swrcb.ca.gov

California Regional Water Quality Control Board
San Francisco Bay
1515 Clay Street, Suite 1400
Oakland, California 94612

DISCLAIMER

This document, *Screening For Environmental Concerns at Sites With Contaminated Soil and Groundwater* (July 2003), is a technical report prepared by staff of the California Regional Water Quality Board, Bay Area Region (Board staff). This document is not intended to establish policy or regulation. The Environmental Screening Levels presented in this document and the accompanying text are specifically not intended to serve as: 1) a stand-alone decision making tool, 2) guidance for the preparation of baseline ("Tier 3") environmental assessments, 3) a rule to determine if a waste is hazardous under the state or federal regulations, or 4) a rule to determine when the release of hazardous chemicals must be reported to the overseeing regulatory agency.

The information presented in this document is not final Board action. Board staff reserve the right to change this information at any time without public notice. This document is not intended, nor can it be relied upon, to create any rights enforceable by any party in litigation in the State of California. Staff in overseeing regulatory agencies may decide to follow the information provided herein or act at a variance with the information, based on an analysis of site-specific circumstances.

This document will be periodically updated as needed. Please send comments, edits, etc. in writing to the above contacts. Board staff overseeing work at a specific site should be contacted prior to use of this document in order to ensure that the document is applicable to the site and that the user has the most up-to-date version available. This document is not copyrighted. Copies may be freely made and distributed. It is cautioned, however, that reference to the screening levels presented in this document without adequate review of the accompanying narrative could result in misinterpretation and misuse of the information.

Table of Contents

FIGURES

TABLES (ENVIRONMENTAL SCREENING LEVELS)

APPENDICES (IN VOLUME 2)

Executive Summary

This document presents Environmental Screening Levels (ESLs) for chemicals commonly found in soil and groundwater at sites where releases of hazardous chemicals have occurred. The ESLs replace screening levels presented in the previous edition of this document, entitled *Application of Risk-Based Screening Levels (RBSLs) And Decision Making to Sites With Impacted Soil and Groundwater* (December 2001). The change in terminology from "Risk-Based" screening levels to "Environmental" screening levels is intended to better convey the broad scope of the document and clarify that some screening levels are not "risk-based" in a strict toxicological definition of this term.

The ESLs are considered to be conservative. Under most circumstances, and within the limitations described, the presence of a chemical in soil, soil gas or groundwater at concentrations below the corresponding ESL can be assumed to not pose a significant, long-term (chronic) threat to human health and the environment. Additional evaluation will generally be necessary at sites where a chemical is present at concentrations above the corresponding ESL. Active remediation may or may not be required, however, depending on site-specific conditions and considerations. This document may especially be beneficial for use at sites with limited impacts, where the preparation of a more formal environmental assessment may not be warranted or feasible due to time and cost constraints.

The ESLs were developed to address environmental protection goals presented in the *Water Quality Control Plan for the San Francisco Bay Basin* ("Basin Plan," RWQCBSF 1995) of the San Francisco Bay Area Regional Water Quality Control Board (RWQCB). These goals include:

Surface Water and Groundwater:
- Protection of drinking water resources;
- Protection of aquatic habitats;
- Protection against adverse nuisance conditions.

Soil:
- Protection of human health;
- Protection of groundwater;
- Protection of terrestrial biota;
- Protection against adverse nuisance conditions.

The ESLs are presented in a series of four lookup tables. Each table reflects a specific combination of soil, groundwater and land-use characteristics that strongly influence the magnitude of environmental concerns at a given site. This allows the user to select ESLs that are most applicable to a given site.

The ESL document presents a "tiered" approach to environmental risk assessments. Under "Tier 1", sample data are directly compared to ESLs selected for the site and decisions are made regarding the need for additional site investigation, remedial action or a more detailed risk assessment. In a "Tier 2" risk assessment, a selected component(s) of the Tier 1 ESL is modified with respect to site-specific considerations. An example may be the adjustment of a screening level for direct exposure with respect to an approved, alternative target risk level. Site data are then compared to the revised screening level as well as the remaining, unmodified components of the Tier 1 ESL. This provides an intermediate but still relatively rapid and cost-effective option for preparing more site-specific risk assessments. Risk assessment models and assumptions that depart significantly depart from those used to develop the Tier 1 ESLs are described in a more traditional, "Tier 3" risk assessment. The Tier 1 methodology can, however, still provide a common platform to initiate a Tier 3 risk assessment and help ensure that all potentially significant environmental concerns are considered.

The Tier 1 ESLs presented in the lookup tables are NOT regulatory "cleanup standards". Use of the ESLs and this document in general is intended to be entirely optional on the part of the regulated facility and subject to the approval of the case manager in the overseeing regulatory agency. The presence of a chemical at concentrations in excess of an ESL does not necessarily indicate that adverse impacts to human health or the environment are occurring; this simply indicates that a potential for adverse risk may exist and that additional evaluation is warranted. ESLs presented for chemicals that are known to be highly biodegradable in the environment may in particular be overly conservative for use as final cleanup levels (e.g., many petroleum-related compounds). Use of the ESLs as cleanup levels should be evaluated in view of the overall site investigation results and the cost/benefit of performing a more site-specific risk assessment.

Reliance on only the Tier 1 ESLs to identify potential environmental concerns may not be appropriate for some sites. Examples include sites that require a detailed discussion of potential risks to human health, sites where physical conditions differ drastically from those assumed in development of the ESLs (e.g., mine sites, landfills, etc., with excessively high or low pH) and sites where impacts pose heightened threats to sensitive ecological habitats. The latter could include sites that are adjacent to wetlands, streams, rivers, lakes, ponds or marine shoreline or sites that otherwise contain or border areas where protected or endangered species may be present. Potential impacts to sediment are also not addressed. (e.g., presence of endangered or protected species). The need for a detailed ecological risk assessment should be evaluated on a site-by-site basis for areas where significant concerns may exist. Notification to the Natural Resource Trustee Agencies (including the state Department of Toxics Substances Control and Department of Fish and Game and the federal Fish and Wildlife Service, Department of the Interior and National Oceanic and Atmospheric Administration) may also be required, particularly if the release of a hazardous substance may impact surface waters.

The ESLs should NOT be used to determine when impacts at a site should be reported to a regulatory agency. All releases of hazardous substances to the environment should be reported to the appropriate regulatory agency in accordance with governing regulations. The lookup tables will be updated on a regular basis, as needed, in order to reflect changes in the referenced sources as well as lessons gained from site investigations and field observations.

1

Introduction

1.1 Purpose

Preparation of detailed environmental risk assessments for sites impacted by releases of hazardous chemicals can be a time consuming and costly effort that requires expertise in a multiple of disciplines, including toxicology, geology, ecology, chemistry, physics and engineering, among others. For small-business owners and property owners with limited financial resources, preparation of such risk assessments can be time and cost-prohibitive.

As a means to partially address this problem, this document presents a series of conservative Environmental Screening Levels (ESLs) for soil, groundwater and soil gas that can be directly compared to environmental data collected at a site. Correlative screening levels for surface water are also provided. Screening levels for over 100 commonly detected contaminants are given in a series of "lookup" tables. The tables are arranged in a format that allows the user to take into account site-specific factors that help define environmental concerns at a given property.

Within noted limits, risks to human health and the environment can be considered to be insignificant at sites where concentrations of chemicals of concern do not exceed the respective ESLs. The presence of chemicals at concentrations above the ESLs does not necessarily indicate that a significant risk exists at the site. It does, however, generally indicate that additional investigation and evaluation of potential environmental concerns is warranted.

The introductory text of this document is kept intentionally brief with a focus on theh use of the ERLs rather than technical details about their derivation. The latter is provided in the appendices of Volume 2.

1.2 Tiered Approach to Environmental Risk Assessments

This document presents a three-tiered approach to environmental risk assessment. Under "Tier 1", sample data are directly compared to ESLs selected for the site and decisions are made regarding the need for additional site investigation, remedial action or a more

detailed risk assessment. A detailed understanding of the derivation of the screening levels is not required for use at this level.

Under "Tier 2", selected components of the models used to develop the Tier 1 ESLs are modified with respect to site-specific data or considerations. Examples include adjustment of the assumed depth to impacted groundwater in the Tier 1 indoor-air impact model or use of an approved, alternative target risk level for direct-exposure concerns. Site data are then compared to the revised screening level as well as the remaining, unmodified components of the Tier 1 ESLs. This provides an intermediate but still relatively rapid and cost-effective option for preparing more site-specific risk assessments.

Under Tier 3, the user employs alternative models and modeling assumptions to develop site-specific screening or final cleanup levels or quantitatively evaluate the actual risk posed to human and/or ecological receptors by the impacted media. Consideration of the methodologies and potential environmental concerns discussed in this document is still encouraged, however. This will help increase the comprehensiveness and consistency of Tier 3 risk assessments as well as expedite their preparation and review.

1.3 Comparison To Existing Screening Levels

Both Region IX of the U.S. Environmental Protection Agency (USEPA 2002) and the City of Oakland (Oakland 2000) have prepared lookup tables of Environmental Screening Levels for soil and water. The lookup tables presented in this document represent an expansion of this work to reflect the broader scope of environmental concerns put forth in the Regional Water Quality Control Board (RWQCB) Basin Plan (RWQCBSF 1995). Differences and similarities between the ESL document and lookup tables prepared by the other programs are summarized below.

1.3.1 USEPA Region IX PRGs

The U.S. Environmental Protection Agency (USEPA) Region IX "Preliminary Remediation Goals" or "PRGs" are intended to address human health concerns regarding direct exposure with impacted soils (USEPA 2002). The equations used to develop the USEPA PRGs are generally consistent with human health risk assessment guidance prepared by the Department of Toxic Substances Control, including the CalTOX model (CalEPA 1994a) and the documents *Preliminary Endangerment Assessment Guidance Manual* (CalEPA 1994b) and *Supplemental Guidance For Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities* (CalEPA 1996a). As noted in Chapter 3, use of the CalTOX model and other CalEPA guidance documents and models may be necessary where more detailed risk assessments are required.

As discussed in the USEPA Region IX document, the PRGs are intended to address human direct-exposure with impacted soil and "...do not consider impact to groundwater or address ecological concerns." (USEPA 2002). Expansion of the USEPA PRGs in the lookup tables presented in this document includes:

- Modification of soil PRGs to reflect CalEPA-specific toxicity factors;
- Adjustment of PRGs for noncarcinogens to reflect a target hazard quotient of 0.2 to address potential cumulative health concerns;
- Addition of direct-exposure screening levels for construction and trench workers' exposure to subsurface soils;
- Addition of soil and groundwater screening levels for indoor-air impact concerns;
- Addition of groundwater screening levels for the protection of aquatic habitats/surface water quality;
- Use of a more rigorous leaching model to develop soil screening levels for protection of groundwater quality;
- Addition of soil screening levels for urban area, ecological concerns;
- Addition of soil and groundwater "ceiling levels" to address gross contamination and general resource degradation concerns; and
- Addition of soil and groundwater screening levels for Total Petroleum Hydrocarbons (TPH).

Use of the USEPA Region IX PRGs in the RWQCB lookup tables is discussed further in Section 3.2 of Appendix 1. A copy of the PRG background document is provided in Appendix 2.

1.3.2 City of Oakland Screening Levels

A brief comparison of the RWQCB and the City of Oakland approaches to the development of environmental screening levels is provided in Table 1-1. Since 1999, the City of Oakland has presented environmental screening levels for soil and groundwater through its Urban Land Redevelopment (ULR) Program. The ULR Program is a collaborative effort by the City of Oakland and the principal agencies charged with enforcing environmental regulations in Oakland to facilitate the cleanup and redevelopment of contaminated properties (Oakland 2000). It includes innovative institutional mechanisms for tracking residual contamination and ensuring long-term compliance with risk management plans. The ULR Program is coordinated by the City and is specific to Oakland sites.

The City of Oakland approach is based on the guidelines prescribed in *Standard Guide for Risk-Based Corrective Action Applied at Petroleum Release Sites* (ASTM 1995). The Guidance Document, Technical Background Document and other information on the Oakland ULR program is available on the internet at www.oaklandpw.com/ulrprogram. Modifications have been made to better address child exposure and recreational water use scenarios. In addition, many input values reflect Oakland-specific geologic, hydrogeologic and climatic conditions (Oakland Technical Background 2000 and

updates). These values may not be appropriate for other areas within the RWQCB's jurisdiction.

The RWQCB has agreed that the Oakland look-up tables are appropriate for use at Oakland sites under the conditions and limitations discussed in the ULR Program Guidance (memo dated August 3, 2001; RWQCBSF 2001b). In particular, sites where surface or groundwater conditions present ecological, aesthetic, taste or odor concerns may require additional analysis. Active remediation to address these concerns may not be necessary at most sites in Oakland that are not near sensitive water bodies, however, due to its highly-developed, urban setting

1.3.3 Hazardous Waste Regulations

California Total Threshold Limit Concentrations (TTLC) criteria for solids and Soluble Threshold Limit Concentration (STLC) criteria for liquids should not in most cases be used as soil and groundwater screening or cleanup levels. The TTLC and STLC criteria are intended to determine the type of landfill a waste material must be sent to (Title 22, Section 66699 - Persistent and Bioaccumulative Toxic Waste). Where TTLC or STLC criteria are exceeded, the waste must in general be sent to a Class I, hazardous waste landfill. The criteria, developed in the 1980s, are only loosely based on human health and environmental considerations. STLC values in general reflect drinking water or surface water goals of the time, although some are clearly out-of-date (e.g. trichloroethylene STLC value of 204 mg/L). TTLC values were derived by simply multiplying the STLC value by ten (organic substances) or one hundred (metals).

In most cases, TTLC values exceed the most conservative environmental screening levels presented in this document. In the case of Endrin and DDT/DDE/DDD, however, the TTLC is somewhat lower than the screening levels for human health concerns. For example, the TTLC for combined DDT/DDE/DDD is 1.0 mg/kg while the residential, direct-exposure soil screening is 1.7 mg/kg. This presents the enigma that while soil impacted below 1.7 mg/kg is not considered to pose a significant risk to human health, it could be classified as a "hazardous waste" if it were excavated and transported offsite for disposal. Again, this is not a difference of opinion about the potential toxic effects of these chemicals, it is merely a reflection of the less rigorous development of the TTLC values.

Unfortunately, it is not anticipated that the TTLC and STLC values will be revised in the near future. To avoid potential future problems with soil disposal and even public perception, it may be prudent to use TTLCs as final cleanup values for sites where the TTLC is less than cleanup values based on actual risk to human health and the environment.

1.3.4 OSHA Standards Permissible Exposure Levels

The National Institute for Occupational Safety and Health (NIOSH) is the Federal agency responsible for conducting research and making recommendations for the prevention of work-related disease and injury, including exposure to hazardous chemicals in air (NIOSH 2003). NIOSH develops and periodically revises Recommended Exposure Limits (RELs) for hazardous substances in the workplace. The RELs are used to promulgate Permissible Exposure Levels (PELs) under the Occupational Safety and Health Act (OSHA).

OSHA Permissible Exposure Levels (PELs) for indoor air are intended for use in controlled, industrial work areas where employees are aware of potential health hazards associated with the chemicals they are using and are trained to take proper precautions and minimize exposure (NIOSH 2003). OSHA PELs are **not** appropriate for use at commercial/industrial sites where the chemical is not currently being used. This includes sites affected by the migration of offsite releases (e.g., via emissions from a moving plume of contaminated groundwater). Indoor-air protection goals for these sites should be based on long-term (chronic) health risk to workers. Such risk-based goals levels are typically much more stringent than OSHA PELs.

For example, the current OSHA PEL for trichloroethylene (TCE) is 678,000 ug/m^3 (100 ppmv, NIOSH 2003). Comparable risk-based screening levels for uncontrolled, commercial/industrial settings included in this document fall between 2.0 ug/m^3 and 10 ug/m^3 (carcinogenic effects vs noncarcinogenic effects, respectively; refer to Table E and Appendix 1, Table E-3). The PEL is applicable to work areas where TCE is being used and the employees have been properly trained to minimize exposure. The risk-based goals are applicable to all other areas.

1.3.5 RWQCB Basin Plan

The RWQCB Basin Plan ("Basin Plan") presents generic soil screening levels of 1.0 mg/kg total volatile organic compounds (VOCs) and 10 mg/kg semi-volatile organic compounds (SVOCs, RWQCBSF 1995). The Basin Plan states that the need to develop chemical-specific screening is to be evaluated on a site-by-site basis. As can be inferred from the detailed ESLs provided in Appendix 1, the Basin Plan screening level for total VOCs is probably adequate to overly conservative for gasoline-range petroleum fuel mixtures at most sites. Chemical-specific ESLs for benzene and MTBE are less than 1 mg/kg, due to their human toxicity and/or mobility in soil. The prevalence of less toxic and mobile VOCs in gasoline-range fuel mixtures (e.g., toluene, ethylbenzene, xylenes, etc.), however, would generally ensure that a total VOC screening level of 1 mg/kg adequately addresses concerns regarding these compounds in the absence of chemical-specific ESLs. The total VOC screening level is in all likelihood overly conservative for

most heavier fuel mixtures that lack significant amounts of benzene and MTBE (e.g., diesel fuel).

For direct-exposure, human health concerns, the Basin Plan screening level of 1 mg/kg for total VOCs as presented in the Basin Plan is adequate to marginally over-conservative for the most commonly detected chlorinated solvents (e.g., tetrachloroethylene, trichloroethane, trichloroethylene, etc.). From a modeling perspective, the screening level may be somewhat under-conservative for potential leaching and groundwater protection concerns (e.g., see Appendix 1, Table G). The model used to generate screening levels for leaching of chemicals from soil conservatively assumes, however, that the impacted soil was situated within one meter of groundwater. At the vast majority of sites where this is the actual case, groundwater has already been impacted by the main mass of chemicals and direct monitoring provides a more accurate evaluation of leaching impacts. For sites where impacted soil is situated greater than 10 meters from groundwater, model-generated screening levels developed by other agencies suggest that a screening level of 1 mg/kg (or more) may be adequate for chlorinated VOCs (e.g., HIDOH 1995).

The Basin Plan screening level of 10 mg/kg for total semi-volatile organic compounds (SVOCs) is probably overly conservative for these compounds for groundwater protection purposes. For soils impacted with carcinogenic SVOCs, the Basin Plan screening level has traditionally been used in conjunction with human-health screening levels presented in the USEPA PRGs. The PRGs are also referenced in this document although with some modifications.

The Basin Plan references a total petroleum hydrocarbon (TPH) soil screening level of 100 mg/kg for the protection of drinking water resources. A similar screening level was developed for use in this document. As noted in the lookup tables and discussed in Appendix 1, however, this screening level is considered to be overly conservative for heavy, residual fuels (fuel oil #6, motor oil, etc.) as well as for use at sites that do not pose a direct threat to drinking water or surface water resources.

1.4 Chemicals Not Listed In Lookup Tables

The lookup tables list 100-plus chemicals most commonly found at sites with impacted soil or groundwater. Inclusion of ESLs for additional chemicals is a relatively straightforward process, provided that adequate supporting data are available. To obtain ESLs for chemicals not listed in the lookup tables, the interested party should contact the RWQCB staff noted at the beginning of this document. Development of ESLs will be carried out in the same manner as done for the listed chemicals. As an alternative, ESLs may be developed by qualified persons and submitted to the overseeing regulatory agency for review (refer to Section 3.0).

1.5 Limitations

The Tier 1 ESLs presented in the lookup tables are NOT required, regulatory "cleanup standards". Use of the ESLs as actual cleanup levels should be evaluated in view of the overall site investigation results and the cost/benefit of performing a more detailed environmental risk assessment. The ESLs are intended to be conservative for use at the vast majority of impacted sites in developed areas. As discussed in Chapter 3, however, use of the Environmental Screening Levels may not be appropriate for final assessment of all sites. Examples include:

- Sites that have a high public profile and warrant a detailed, fully documented environmental risk assessment;
- Sites with less than 3.0m (ten feet) of low permeability soils (clay, silt, etc.) between impacted groundwater and the ground surface (including potential downgradient areas; applies only to use of groundwater screening levels for sites with low permeability, vadose-zone soils);
- Sites with high rainfall and subsequent high surface water infiltration rates (i.e., infiltration >28 inches (720mm) per year),
- Sites where inorganic chemicals (e.g., metals) are potentially mobile in leachate due to soil or groundwater conditions different than those assumed in development of the lookup tables (e.g., low pH at mine sites);
- Conservation areas where impacts pose heightened threats to ecological habitats (e.g., presence of endangered or protected species); and
- Sites where more than three known or suspected carcinogens or more than five chemicals with similar noncarcinogenic health effects have been identified.
- Sites affected by tides, rivers, streams, etc. where there is a potential for erosion and concentration of contaminants in aquatic habitats.

Examples of other site characteristics that may warrant a more detailed environmental risk assessment are discussed in Chapter 3 (refer also to discussion of screening levels in Appendix 1). In such cases, the information provided in this document may still be useful for identification of potential environmental concerns and development of strategies for preparation of a more site-specific risk assessment.

ESLs for chemicals that are known to be highly biodegradable in the environment may in particular be overly conservative for use as final cleanup levels. For example, final soil

ESLs for Total Petroleum Hydrocarbon (TPH) and many noncarcinogenic, petroleum-related compounds (e.g., xylenes) are driven by the protection of groundwater quality. If long-term monitoring demonstrates that actual impacts to groundwater are insignificant then less stringent soil (and groundwater) screening levels may be warranted. Additional guidance regarding the management of impacted soil and groundwater at petroleum-release sites is provided in the following documents (refer also to overseeing regulatory agency):

- *Interim Guidance on Required Cleanup at Low-Risk Fuel Sites* (RWQCBSF 1996);
- Guidelines for Investigation and Cleanup of MTBE and Other Ether-Based Oxygenates (SWRCB 2000).

Copies of these documents can be obtained from the RWQCB.

Soil ESLs do not consider potential water- or wind-related erosion and deposition of contaminants in a sensitive ecological habitat. This may especially be of concern for metals and pesticides that are only moderately toxic to humans but highly toxic to aquatic and terrestrial biota (e.g., copper). The RWQCB *Erosion and Sediment Control Field Manual* provides practical information on the mitigation of erosion and runoff concerns.

It is conceivable that soil, groundwater and soil gas screening levels for the emission of chlorinated, volatile organic compounds to indoor air concerns may not be adequately conservative in some cases. This is most likely to occur at sites where the vapor permeability of vadose-zone soils is exceptionally high (e.g., highly fractured bedrock, gravels, etc.) and/or where building designs, ventilation systems and local environmental conditions otherwise lead to higher-than-expected vapor flow rates through foundations (e.g., houses with heating systems in basements). As discussed in Appendix 1, conservative target risks are used in part to address these uncertainties.

Table 1-1. Comparison of RWQCB and Oakland Risk-Based Approaches

		RWQCB	[1]Oakland
General Approach	Tiers	One tier of look-up tables. Includes separate screening levels for indoor air concerns based on soil type.	Two tiers of look-up tables: Tier 1 table applicable at any Oakland site; Tier 2 tables (3) account for site-specific soil types (Merritt Sands, sandy silts, and clayey silts) and alternate target risk. Tier 3 spreadsheets provided.
	Target Cancer Risk Level	10^{-6}	10^{-6} for Tier 1; 10^{-5} for Tier 2.
	Target Noncancer Hazard Quotient	0.2 (with option for site specific adjustment)	1.0 (with requirement to address cumulative risk as necessary)
	Ceiling/Nuisance Levels	"Ceiling levels" to address gross contamination concerns, nuisances, free-product mobility, and general resource quality	No "ceiling levels"; recommends removal of mobile or potentially-mobile free product.
	Total Petroleum Hydrocarbons	Screening levels for TPH included	No TPH screening levels.
Soil Pathways	Definition of "Shallow" Soils	0-3 meters below ground surface.	0-1 meter below ground surface.
	Direct Exposure, Inhalation of Volatiles	USEPA PRG model (USEPA 2002). Assumes "infinite" source thickness for volatile organic compounds.	ASTM (1995) model. Assumes infinite source unless mass balance conditions violated based on 1.0 m thick source.
	Ecological Concerns	Screening levels for terrestrial biota included (shallow soils only).	Recommends site-specific analysis when significant ecological habitats are threatened.
	Deep Soils	Direct-exposure soil screening levels for Construction/ Trench Worker exposure scenario.	No screening levels for this scenario; recommends a site-specific analysis as warranted.
Groundwater	Leaching Model	Employs the SESOIL model.	Employs the ASTM (1995) model.
	Leaching of Inorganic Compounds	No soil screening levels; recommends laboratory tests.	Soil screening levels for inorganic compounds, based on a neutral pH.
	Surface Water Protection	Groundwater screening levels for the ecological and aesthetic protection of surface water.	Screening levels for recreational use of groundwater and surface water. Recommends site-specific analysis of ecological and aesthetic concerns as warranted.
Indoor Air	Thickness of Soil Source	Assumes five meters. Recommends site-specific analysis as warranted.	Assumes "infinite" source thickness.
	Convective Flow	Incorporates convective flow in indoor-air impact model.	Does not incorporate convective flow (i.e., assumes no pressure differential) in indoor-air impact model.
	Surface Soil Screening Levels	Includes screening levels for protection of indoor air for both surface and subsurface soils.	Recommends site-specific analysis and controls for shallow soils (<1m) and use of screening levels for deeper soils.
	Soil Gas	Includes screening levels for soil gas.	Not included.

1. *Oakland Risk-Based Corrective Action: Technical Background Document: City of Oakland, Environmental Services Division*, January 2000 (and updates), www.oaklanddpw.com/urlprogram.

2

Tier 1 Lookup Tables

2.1 Organization of Lookup Tables

Environmental risk assessments may be carried out in either a "forward" mode, where actual risks are quantified based on concentrations of a chemical in an impacted media, or "backward" mode, where acceptable concentrations of a chemical in a given media are developed based on specified, target goals. The Environmental Screening Levels (ESLs) presented in this document represents an example of the latter. Tier 1 ESLs for soil and groundwater are summarized in Tables A through E. Each ESL in the tables collectively addresses environmental concerns stated or inferred in the *Water Quality Control Plan for the San Francisco Bay Basin* ("Basin Plan," RWQCBSF 1995), prepared by the San Francisco Bay Area Regional Water Quality Control Board (RWQCB). These concerns include:

Groundwater Quality:
- Protection of human health
  - Current or potential drinking water resource;
  - Emission of subsurface vapors to building interiors;
- Protection of aquatic habitats (discharges to surface water);
- Protection against nuisance concerns (odors, etc.) and general resource degradation.

Soil Quality:
- Protection of human health
  - Direct/indirect exposure to impacted soil (ingestion, dermal absorption, inhalation of vapors and dust in outdoor air);
  - Emission of subsurface vapors to building interiors;
- Protection of groundwater quality (leaching of chemicals from soil);
- Protection of terrestrial (nonhuman) habitats;
- Protection against nuisance concerns (odors, etc.) and general resource degradation.

Shallow Soil Gas:
- Protection of human health
  - Emission of subsurface vapors to building interiors.

For the purpose of this document, "soil" refers to any unlithified material in the vadose zone that is situated above the capillary fringe of the shallowest saturated unit. A

summary of environmental concerns considered in the ESLs is depicted schematically in Figure 1. This is correlative to a "conceptual site model" prepared for a detailed environmental risk assessment. The degree to which any given concern will "drive" environmental risk at a site depends on the actual potential for exposure and the toxicity and mobility of the chemical.

Site characteristics that play an important role in evaluating potential environmental concerns or developing site-specific cleanup levels include:

- Physical location of the impacted soil (e.g., currently or potentially exposed at the ground surface versus isolated in the subsurface);
- Beneficial use of the groundwater immediately underlying the site or otherwise potentially threatened by the release (e.g., drinking water resource threatened versus no drinking water resource threatened);
- Current and anticipated future use of the site (e.g., residential land use permitted or commercial/industrial land use only).

In order to include consideration of these site characteristics in the ESLs, four different tables were prepared (Tables A through D). Each table reflects varying combinations of site characteristics:

- Table A – Shallow soils, potential drinking water resource threatened;
- Table B – Shallow soils, potential drinking water resource not threatened;
- Table C – Deep soils, potential drinking water resource threatened;
- Table D – Deep soils, potential drinking water resource not threatened;

Each of the tables provides separate soil screening levels for residential (i.e., unrestricted) and commercial/industrial land-use scenarios.

For each chemical listed in the lookup tables, screening levels were selected to address each applicable environmental concern under the specified combination of site characteristics. The lowest of the individual screening levels for each concern was selected for inclusion in the summary Tier ESL tables presented in Volume 1 of this document. This ensures that the ESLs presented in these tables are protective of all potential environmental concerns and provides a tool for rapid screening of site data. Where ESLs are exceeded, the detailed tables provided in Appendix 1 can be used to identify the specific environmental concerns that may be present at the site.

An example of the selection of summary, Tier 1 ESLs for tetrachloroethylene (PCE) is presented in Figure 2 (surface soils, drinking water resource threatened, unrestricted land use desired). A more detailed discussion of this example is provided in Appendix 1.

2.2 Use of Lookup Tables

The step-by-step use of the lookup tables is summarized below and discussed in more detail in the following sections. A summary of the process is also provided in Figure 3. An outline and discussion of information that should be included in a Tier 1 environmental risk assessment is provided in Section 2.11.

Step 1 - ESL Updates and Applicability
Check with the overseeing regulatory agency to determine if the ESLs can be applied to the subject site. Ensure that the most up-to-date version of this document is being used (updated every 1-2 years in general).

Step 2: Identify All Chemicals of Potential Concern
An environmental risk assessment must be based on the results of a thorough site investigation, where all chemicals of potential concern have been identified. A summary of the site investigation results should be included in the risk assessment in order for it to be reviewed as a "stand alone" document." A general outline of site investigation information that should be included in a Tier 1 risk assessment is provided in Section 2.11.

Step 3: Select Lookup Table(s)
Determine the designated beneficial use of impacted or threatened groundwater beneath the site. In general, all groundwater must initially be treated as a current or potential source of drinking water (see Section 2.3). Next, determine the depth below ground surface to the top of impacted soil (see Section 2.4). This site information is then used to select the most appropriate lookup table (see Figure 3).

Steps 4: Determine Desired Land Use (soil ESLs only)
ESLs for soil are selected based on the present and desired future use of the site. Two options are provided in the lookup tables, "Unrestricted Land Use Permitted" or "Commercial/Industrial Land Use Only". Screening levels for unrestricted land used are considered to be adequate for residential use of a property. **For evaluation of commercial/industrial properties, it is highly recommended that site data be compared to ESLs for both unrestricted/residential and commercial/industrial land use.** Reference only to ESLs for commercial/industrial land use will in most cases require that a covenant to the deed be prepared that restricts use of the property to these purposes only (see Section 2.9).

Steps 5 and 6: Select Soil and/or Groundwater ESLs

Based on the desired land use(s), select appropriate soil ESLs. ESLs for groundwater are provided in the adjacent column of each table and are not dependent on land use or depth to impacted soil. Correlative screening levels for surface water are also provided. Replace ESLs with naturally occurring, background concentrations of chemicals of concern (e.g., arsenic) or laboratory method reporting levels if higher (see Section 2.8).

Step 7: Determine Extent of Impacted Soil and/or Groundwater

Using the selected ESLs, determine the extent of impacted soil or groundwater and areas of potential environmental concern at the site and offsite, as required. Soil data should be reported on a dry-weight basis (see Appendix 1, Section 6.2). For sites where sample data are limited, it will be most appropriate to compare the maximum-detected concentrations of chemicals of concern to the ESLs. For sites where an adequate number of data points are available, the use of statistical methods to estimate more site-specific exposure point concentrations and evaluate environmental risks may be appropriate. The exposure point concentration is generally selected as the lesser of the maximum-detected concentration and the 95% upper confidence interval of the arithmetic mean of sample data. Guidance for the estimation of exposure point concentrations, use of "non-detect" data, and other issues is provided in the CalEPA documents *Preliminary Endangerment Assessment Guidance Manual* (CalEPA 1994b) and *Supplemental Guidance For Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities* (CalEPA 1996a), among other sources. As discussed in these documents, sample data collected outside of impacted areas should generally not be included in estimation of exposure point concentrations. **For residential land use scenarios, sample data should be averaged over no more than a 1,000 ft^2 area.**

Steps 8 and 9: Evaluate The Need For Additional Investigation or Corrective Actions; Submit Appropriate Reports

Based on a comparison of available site data to the ESLs, evaluate the need for additional action at the site (e.g. additional site investigation, remedial action, preparation of a more site-specific risk assessment, etc.). This is then summarized in the Tier 1 Environmental Risk Assessment report and workplans for additional corrective actions as needed (see Section 2.11). Decisions for or against additional actions should always be made in conjunction with guidance from the overseeing regulatory agency.

Note that impacts to soil and water from petroleum mixtures are evaluated in terms of both Total Petroleum Hydrocarbon (TPH) and target "indicator chemicals" for the given petroleum mixture. Indicator chemicals typically recommended for petroleum mixtures include (after CalEPA 1996a):

Monocyclic Aromatic Compounds (primarily gasolines and middle distillates)

- benzene
- ethylbenzene
- toluene

- xylene

Fuel additives (primarily gasolines)

- MTBE
- other oxygenates as necessary

Polycyclic Aromatic Compounds (primarily middle distillates and residual fuels)

- methylnaphthalene (1- and 2-)
- acenaphthene
- acenaphthylene
- anthracene
- benzo(a)anthracene
- benzo(b)fluoranthene
- benzo(g,h,i)perylene
- benzo(a)pyrene
- benzo(k)fluoranthene
- chrysene
- dibenzo(a,h)anthracene
- fluoranthene
- fluorene
- indeno(1,2,3)pyrene
- naphthalene
- phenanthrene
- pyrene

The TPH ESLs should be used in conjunction with ESLs for these chemicals. As discussed in Appendix 1, the "middle distillates" category of TPH includes diesel fuel kerosene, stoddard solvent, home heating fuel, jet fuel and similar petroleum mixtures. "Residual fuels" includes heavy petroleum products such as No. 6 fuel oil ("Bunker C"), lubricating oils, "waste oils" and asphalts. Soil and groundwater impacted by releases of waste oil may also require testing for heavy metals and chemicals such as chlorinated solvents and PCBs. Screening levels for these chemicals are included in the lookup tables.

2.3 Groundwater Beneficial Use

As stated in the San Francisco Bay Region *Water Quality Control Plan* ("Basin Plan", RWQCBSF 1995), "Unless otherwise designated by the Regional Board, all groundwaters are considered suitable, or potentially suitable, for municipal or domestic water supply." All groundwater beneath a given site should be initially treated as a potential source of drinking water unless otherwise approved by the RWQCB office. For the purposes of this document, it is also assumed that all shallow groundwater will ultimately discharge to a body of surface water and potentially impact aquatic organisms (see Section 2.7). Soil and groundwater ESLs were therefore developed to be protective of both drinking water resources and aquatic habitats. This is discussed in greater detail in Chapters 2 and 3 of Appendix 1.

The Basin Plan recognizes that site-specific factors may render groundwater unsuitable for potential drinking water purposes. Tables B and D in this document are intended for use at such sites. The ESLs presented in these tables consider the potential discharge of groundwater to surface water but do not consider potential impacts to sources of drinking water. The ESLs also consider "gross contamination" issues such as the presence of free product and aesthetic or odor problems. Use of these tables for screening level environmental risk assessments must be approved by the RWQCB but may not necessarily require regulatory "de-designation" of groundwater beneficial use.

Hydrogeologic criteria presented in the Basin Plan for potential exclusion of a given occurrence of groundwater from consideration as a potential source of drinking water include:

- Total dissolved solids in groundwater is greater than or equal to 3,000 mg/L; OR

- Water bearing unit is not sufficiently permeable to produce an average, sustained yield of 200 gallons of water per day.

Groundwater in coastal areas, geothermal fields, etc., may contain levels of dissolved solids that make the water unsuitable as a potential source of drinking water. In addition, the permeability of soils and sediments that lack a significant amount of coarse-grained material (or fractures, in the case of bedrock) may be too low to allow for an adequate, sustained yield of groundwater. Unconsolidated geologic units that are comprised of less than 20% sand-size (or larger) material or more than 30% clay-size material are typically not considered to be viable "aquifers" or potential sources of useable groundwater (inferred from Fetter 1994). The potential for a given unit of bedrock to serve as a viable source of groundwater similarly depends on the primary and secondary porosity in the rock and the quality of the groundwater. Consideration must also be made for the potential migration of groundwater out of a geologic unit that in itself is insufficiently permeable to be considered to be an aquifer and into a more permeable unit that could serve as a viable source of drinking water.

In general, soil and groundwater screening levels are more stringent for sites that threaten a potential source of drinking water (e.g., compare Tables A and B). This is particularly true for chemicals that are highly mobile in the subsurface and easily leached from impacted soil. For chemicals that are especially toxic to aquatic life (e.g., several long-chain hydrocarbons, pesticides and heavy metals), however, screening levels for sites that threaten drinking water resources may be driven by surface water/aquatic habitat protection concerns. This is discussed in more detail in Appendix 1.

2.4 "Shallow" Versus "Deep" Soils

For the purposes of this document, a depth of three meters (approximately 10 feet) was used to delineate between "shallow" soils, where a potential exists for regular direct exposure of residents and/or office workers, and "deep" soils where only periodic exposure during construction and utility maintenance work is considered likely. This is consistent with guidance presented in the CalEPA document *Supplemental Guidance For Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities* (CalEPA 1996a) and is regarded as the maximum, likely depth that impacted soil could at some point in the future be excavated and left exposed at the surface during typical redevelopment activities. The potential for deeper soils to be brought to the surface in the future should be evaluated on a site-by-site basis based on planned redevelopment or maintenance activities.

The full suite of environmental concerns noted in Figure 1 was considered in development of ESLs for shallow soils. For deep soils, regular exposure of residents or commercial/industrial workers and impacts to terrestrial flora and fauna was not considered. As a result, ESLs for relatively non-mobile chemicals are generally less stringent for deep soils than correlative ESLs for shallow soils (e.g., compare PCB ESLs in Tables A and C). For chemicals that are easily leached from soil or potentially emitted to the air as a volatile gas, however, groundwater and indoor-air protection concerns usually drive selection of the final ESL regardless of the depth of the impacted soil. This is the case for several of the highly volatile, chlorinated organic compounds. As a result, correlative shallow and deep soil ESLs are identical (e.g., compare trichloroethylene ESLs in Tables A and C).

If impacted soil extends across the three-meter dividing line between shallow soil and deep soil, it may be appropriate to use a separate set of screening levels for each zone (e.g., Table A for the shallow soils and Table C for the deep soils). As discussed in Section 2.9, however, the pros and cons of remediating deep soils to shallow soil criteria should be evaluated on a site-by-site basis. This may help avoid concerns regarding future disturbance and reuse of deeper soils.

As another alternative, the less stringent ESLs for deep soils could be applied to shallower soils under a Tier 2 or Tier 3 risk assessment (refer to Chapter 3), provided that appropriate actions to prevent future exposure and unmanaged reuse are taken. Such controls may include (but not necessarily be limited to):

- placement and maintenance of adequate cap or other risk-management measures to eliminate potential direct exposure;
- modeling and/or direct field measurement to evaluate potential impacts to indoor air due to vapor emissions; and

- preparation of a risk management plan and other appropriate institutional controls (e.g., deed restrictions) in order to prevent unauthorized disturbance of the soil in the future and allow for appropriate management of the soil if it is exposed.

Capping of shallow, contaminated soil and other engineered controls used in place of full cleanup are generally not allowed for properties that are to be used for single-family homes. The need to consider these actions at sites with impacted soils situated more than three meters below the ground surface should be discussed with the overseeing regulatory agency on a site-by-site basis.

2.5 Land Use

Land uses are categorized based on the assumed length, duration and magnitude of potential human exposure. The category "Residential Land Use" is intended for use at sites where future land-use restrictions are not desirable or allowed. This includes sites to be used for residences, hospitals, day-care centers and other sensitive purposes (e.g., refer to DTSC 2002). ESLs listed under this category incorporate conservative assumptions regarding long-term, frequent exposure of children and adults to impacted soils in a residential setting (see Appendices 1, Section 3.2 and Appendix 2). In contrast, the land-use category "Commercial/Industrial Use Only" assumes that only working age adults will be present at the site on a regular basis. Direct-exposure assumptions incorporated into the soil ESLs are somewhat less conservative than assumptions used in the residential land-use scenario.

Land use should be selected with respect to the current and foreseeable future use of the site in question. Reference to adopted General Plan zoning maps and local redevelopment plans is an integral part of this process. Use of the lookup tables for sites with other land uses (e.g., agriculture, parkland, etc.) should be discussed with and approved by the overseeing regulatory agency. As the category heading implies, use of the soil ESLs listed under "Commercial/Industrial Use Only" places implicit land-use restrictions on the affected property. While this may be considered acceptable for properties currently zoned for such purposes, the need for such restrictions in the future should be seriously weighed against the cost-benefit of remediating the property to meet the sometimes more conservative but less restrictive ESLs for unrestricted land use. Implications for land-use restriction are discussed in more detail in Section 2.9.

A 2003 amendment to the Porter-Cologne Act (Section 13307.1(c)) requires that formal land-use restrictions be placed on sites that are not remediated to an extent that allows unrestricted future use (e.g., residential, day care, etc.). This rule does not currently apply to sites regulated under the state underground storage tank program. It is anticipated that this rule will be especially applied to non petroleum-impacted sites.

2.6 Threat To Surface Water Habitats

Screening levels for freshwater, marine and estuarine water bodies are presented in Table F. These screening levels consider the same set of environmental concerns as groundwater, with the addition of screening levels for the potential bioaccumulation of chemicals in aquatic organisms and subsequent human consumption of these organisms. Locally, the areas north of the Dumbarton Bridge and west of the Richmond-San Rafael Bridge are considered to be marine. The areas south of the Dumbarton Bridge and east of the Richmond-San Rafael Bridge to the upstream extent of tidal influences are considered to be estuarine. Tidally influenced portions of creeks, rivers and streams flowing into the Bay between these areas should also be considered to be estuarine in screening level assessments.

For the purposes of the Tier 1 lookup tables, it is assumed that impacted or potentially impacted groundwater at all sites could at some time migrate offsite and discharge into a body of surface water. This could occur due to the natural, downgradient migration of groundwater or to human activities such as dewatering of construction sites. For several pesticides and heavy metals, including dieldrin, endrin and endosulfan, aquatic habitat goals are more stringent than drinking water toxicity goals for humans. This is reflected in the final groundwater screening levels (refer also to Appendix 1).

The groundwater screening levels for potential impacts to aquatic habitats do not consider dilution of groundwater upon discharge to a body of surface water. Benthic flora and fauna communities situated below or at the groundwater/surface water interface are assumed to be exposed to the full concentration of chemicals in impacted groundwater. Use of a generic "dilution factor" to adjust the surface water protection screening levels with respect to dilution of groundwater upon discharge to surface water was therefore not considered. Consideration of dilution/attenuation factor and alternative groundwater screening levels for the protection of surface water quality may, however, be appropriate on a site-specific basis.

Consideration of surface water standards for bioaccumulation concerns in groundwater investigations and cleanup actions may be warranted at sites where large plumes of impacted groundwater threaten to cause long-term impacts to important aquatic habitats. The bioaccumulation standards will generally not need to be considered at sites with small, isolated plumes of impacted groundwater located some distance from a body of surface water. Although these plumes could conceivably migrate offsite and discharge into a body of surface water in the distant future, impacts are likely to be short-lived and the plumes are likely to become significantly diluted as they mix with surface water. The need for a more detailed study of potential groundwater impacts on surface water with respect to bioaccumulation of chemicals in aquatic organisms should be evaluated on a site-by-site basis. This may include the need for more stringent soil cleanup levels (to prevent additional leaching) and development of a more comprehensive, ecological risk assessment.

The soil and groundwater screening levels presented in the lookup tables do not directly address the protection of sediment quality. Site-specific concerns could include the accumulation and magnification of concentrations of highly sorptive chemicals in sediment over time due to long-term discharges of impacted groundwater. This may be especially true for groundwater impacted with highly sorptive (lipophyllic) chemicals, including heavy petroleum products.

Potential erosion and runoff of surface soils from impacted sites may also need to be considered, particularly at sites impacted with metals and pesticides that are situated near a sensitive body of surface water. The need for a more detailed, ecological risk assessment of impacts to sediment should be evaluated on a site-by-site basis and discussed with the overseeing regulatory agency.

2.7 Screening For Indoor-Air Impact Concerns

Volatile chemicals can be emitted from contaminated soil or groundwater and intrude overlying buildings, impacting the quality of indoor air. Heating systems, basements, and strong winds can exacerbate this problem by reducing the internal air pressure and creating a "vacuum effect" that enhances the advective flow of vapors out of the underlying soil and into the building. Additional information on subsurface vapor intrusion into buildings is provided in the USEPA document *User's Guide For The Johnson and Ettinger (1991) Model For Subsurface Vapor Intrusion Into Buildings* (USEPA 2000; refer also to Appendix 1).

The direct collection and analysis of indoor air samples would seem to be an easy way to evaluate this concern. Identification of the source of impacts is complicated, however, by the presence of the same chemicals in many household goods (aerosol sprays, dry-cleaned clothing, cleaners, etc.). In addition, plumes of groundwater impacted with volatile chemicals are known to extend over significant areas and comprehensive testing of every structure over the plume is not practical.

As an alternative, the comparison of site groundwater, soil gas and soil data to conservative screening levels for indoor air concerns is recommended. Screening levels incorporated into this document are based on scientific models for vapor intrusion into buildings as well as a growing body of data from actual field investigations. A detailed discussion of the screening levels is presented in Appendix 1. The following three-phase, sequential approach is recommended for initial evaluation of potential indoor-air impact concerns at sites where shallow groundwater has been impacted by volatile chemicals:

1) Compare groundwater data to appropriate screening levels for indoor air concerns (see Table E-1a of Appendix 1).

2) For areas over the plume where groundwater screening levels for indoor-air concerns are approached or exceeded, collect shallow soil gas samples under (preferred) or adjacent to buildings and compare results to soil-gas screening levels for this concern (refer to Table E in this volume or Table E-2 in Appendix 1).

3) At buildings soil-gas screening levels for indoor-air concerns are approached or exceeded, collect indoor-air samples and compare results to indoor-air screening levels (refer to Table E in this volume or Table E-3 in Appendix 1).

For sites where the vapor permeability of shallow soils has not been evaluated, screening levels for groundwater overlain by highly permeable vadose-zone soils should be used. Imported fill material or disturbed native soils should be considered to be highly permeable unless site-specific data indicates otherwise.

Unless inhibited by very high water tables or other obstacles, soil gas samples should be collected immediately beneath the foundations of existing buildings (e.g., "subslab" or in crawl spaces) or three to five feet below ground surface in open areas where buildings may be constructed in the future. Soil gas samples collected from depths less than three feet are currently considered unreliable due to the increased potential to draw in ambient, surface air. If site-specific modeling of vapor flow rates or indoor-air impacts is to be carried out, the collection of additional geotechnical data at the time soil gas samples are collected should be considered (soil grain-size analysis, moisture content, vapor permeability, etc.).

Soil screening levels for potential indoor-air concerns are incorporated into the summary tables of this volume and presented separately in Table E-1b of Appendix 1. At sites where minor releases of volatile chemicals have occurred (e.g., restricted spills around underground tank fill ports), direct comparison of soil screening levels to site data is generally acceptable. If screening levels are exceeded, a similar approach to that outlined above for impacted groundwater is recommended. The restricted size of soil samples and the difficulty in predicting vapor-phase concentrations of chemicals from soil data limits the use of this data as a stand-alone tool for evaluating indoor-air concerns. **At sites where significant releases of volatile chemicals have occurred, the direct use of soil gas data in conjunction with soil data is strongly recommended.**

Guidance on the collection of indoor air and soil gas samples is provided in the following documents, among other sources:

- *Indoor Air Sampling And Evaluation Guide* (2002): Massachusetts Department of Environmental Protection, Office of Research and Standards, WSC Policy #02-430; http://www.state.ma.us/dep/bwsc/finalpol.htm;

- *Soil Gas Advisory* (January 2003): Department of Toxic Substances Control and Los Angeles Regional Water Quality Control Board; http://www.dtsc.ca.gov/PolicyAndProcedures/SiteCleanup/SMBR_ADV_activesoilgasinvst.pdf.

Additional information on the intrusion of subsurface vapors into buildings will be incorporated into this document as available. Individuals are encouraged to provide comments and suggestions to the contacts listed in the front of this document at anytime.

2.8 Substitution of Laboratory Reporting Limits and Ambient Background Concentrations for ESLs

In cases where an ESL for a specific chemical is less than the laboratory method reporting limit for that chemical (as agreed upon by the overseeing regulatory agency), it is generally acceptable to consider the method reporting limit in place of the screening level. Potential examples include the soil health-based ESLs for dioxin (e.g., 0.0000045 mg/kg for residential exposure).

Background concentrations of metals in soils are presented in the summary lookup tables in cases where they exceed screening levels for human health and environmental concerns. This is particularly an issue for arsenic and thallium in Bay area soils. For example, typical mean background concentrations of arsenic in Bay area soils ranges from approximately 5 mg/kg to 20 mg/kg, with some soils containing up to 40+ mg/kg arsenic (LBNL 2002). These concentrations are well above the health-based, direct-exposure goals for arsenic in soil of 0.39 mg/kg (residential exposure) and 1.6 mg/kg (commercial/industrial exposure) presented in the appendices.

For use in this document, an assumed background level of 5.5 mg/kg arsenic was substituted for toxicity-based goals in the lookup table if higher than the later. A background concentration of 58 mg/kg total chromium in soil is also assumed in the lookup tables. Note that background levels of total chromium can be significantly higher (>1,000 mg/kg) in soils developed over mafic and ultramafic rocks in the Bay area. Refer also to Appendix 1, Section 3.2.4 for additional discussion of this issue.

Figure 4 suggests steps that could be taken when evaluating a site for potential arsenic impacts. The natural background concentration of a chemical in soil or groundwater can vary significantly between and even within sites and is most appropriately evaluated by the collection of on-site samples or by reference to local data collected from past studies. Guidance for estimating background concentrations of chemicals in soil and groundwater is provided in the CalEPA document *Supplemental Guidance For Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities* (CalEPA 1996a). Sources of background metal concentration in soils in California include the University of California-Riverside report *Background Concentrations of Trace and Major Elements in California Soils* (UCR 1996) and the Lawrence Berkeley

Laboratory document *Protocol for Determining Background Concentrations if Metals in Soil at Lawrence Berkeley National Laboratory* (LBNL 2002).

A similar approach should be taken for total chromium. Additional review of background total chromium concentrations in soil should be carried out at sites where the screening level of 58 mg/kg is exceeded. If reported levels of total chromium still appear to exceed anticipated site-specific background levels, then soil samples should be tested for Cr VI and Cr III. Data should be compared to screening levels for these specific species of chromium and action taken as needed.

2.9 Implied Land-Use Restrictions Under Tier 1

Allowing the option to tie screening levels or cleanup levels to site-specific land use and exposure conditions can save considerably in investigation and remediation costs. For example, the screening level for polychlorinated biphenyls (PCBs) in surface soils is 0.22 mg/kg in residential areas but up to 7.0 mg/kg (at target risk of 10^{-5}) for commercial/industrial areas. Even higher levels of PCBs could potentially be allowed to remain in place onsite provided that adequate controls to mitigate potential exposure are put into effect (e.g., permanent cap, protection of groundwater, etc.).

The use of final cleanup levels less stringent than those appropriate for unrestricted land use will, however, place restrictions on future use of the property. For example, if a site is remediated using ESLs (or alternative criteria) intended for commercial/industrial land use then the site cannot be used for residential purposes in the future without additional evaluation. In most cases, this will require that a formal covenant to the deed be recorded to restrict future use of the property. As stated in recent provisions in the Porter-Cologne Act (Section 13307.1(c)):

> "...if the state board or the regional board finds that the property is not suitable for unrestricted use...then the state board and regional boards may not issue a closure letter, or make a determination that no further action is required...unless a land restriction is recorded..."

The use of ESLs for deep soils at a site similarly assumes that the impacted soil will remain isolated below the ground surface "for eternity". For single-family, residential areas, future disturbance of soil situated greater than three meters is generally considered to be unlikely (CalEPA 1996a) and use of the ESLs for deep soil below this depth without restrictions may be reasonable (see Section 2.4). During the redevelopment of properties for commercial/industrial or high-density residential use, however, excavation and removal of soils from depths in excess of five or even ten meters could take place (e.g., for underground parking garages, elevator shafts, utilities, etc.). The need to impose enforceable, institutional controls for proper management of deep, impacted soils

at properties where the subsurface ESLs (or alternative cleanup levels) are applied should be discussed with the overseeing regulatory agency on a site-by-site basis.

Land-use restrictions inherent in the selection of ESLs from the Tier 1 lookup tables (or assumptions used in site-specific risk assessments) should be kept as minimal as possible. **Concentrations of chemicals in impacted soils left in place at a commercial/industrial site should always be compared to both commercial/industrial AND residential ESLs (or alternative criteria for unrestricted land use).** If the soils in fact meet ESLs for unrestricted land use after cleanup then this should be clearly stated in the site closure report. Recognizing this point may prove important should the site unexpectedly become desirable for other use in the future (e.g., residential, school day care, health care, etc.). **Assumptions that impacted soil at a property will remain isolated at shallow depths under pavement, buildings or some other type of "cap" should likewise be avoided if at all possible.** Such assumptions place significant and oftentimes unnecessary restrictions on the future use and redevelopment of a site. If done, appropriate covenants to the property deed should be prepared and methods to prevent or manage future disturbance of the soil should be clearly described and ensured. A foresighted approach in the use of Tier 1 ESLs or alternative, site-specific cleanup levels will allow more flexibility in future use of a site, help avoid unexpected complications during site redevelopment and minimize the liability of future land owners.

2.10 Cumulative Risks at Sites With Multiple Chemicals of Concern

Risks posed by direct exposure to multiple chemicals with similar health affects are considered to be additive or "cumulative." For example, the total risk of cancer posed by the presence of two carcinogenic chemicals in soil is the sum of the risk posed by each individual chemical. The same is true for chemicals that cause noncarcingenic health effects. A summary of example target health effects for the chemicals listed in the lookup tables is provided in Appendix 1 (Table L).

Use of ESLs for single chemicals is limited to the extent that the screening levels remain protective of human health should other chemicals with similar health effects also be present. Soil ESLs are considered to be adequate for use at sites where no more three carcinogenic chemicals or five chemicals with similar noncarcinogenic ("systemic") health effects are present. This is based on a combination of conservative exposure assumptions and target risk factors in direct-exposure models. Refer to Appendix 1, Section 1.3, for additional discussion of this subject.

2.11 Framework For a Tier 1 Environmental Risk Assessment

Tier 1 environmental risk assessments should serve as "stand alone" documents that provide a good summary of environment impacts at a site and assess the threats posed to human health and the environment by these impacts. The risk assessment can be prepared as a component of a site investigation or remedial action report or as a separate document. Information on each of the topics listed below should be addressed in report that presents the risk assessment, however (after MADEP 1995). Together, this information is intended to provide a basic "conceptual model" of site conditions. The level of detailed required for each topic will vary depending on site-specific considerations.

1. Summarize Past, Current and Anticipated Future Site Activities and Uses:

- Describe past and current site uses and activities;
- Describe foreseeable future site uses and activities. **(Always include a comparison of site data to ESLs for unrestricted land use to evaluate need for formal covenants to the deed; see Section 2.9).**

2. Summary of Site Investigation:

- Identify all types of impacted media;
- Identify all sources of chemical releases;
- Identify all chemicals of concern;
- Identify magnitude and extent of impacts that exceed ESLs to extent feasible and applicable (include maps of site with isoconcentration contours for soil and groundwater);
- Identify nearby groundwater extraction wells, bodies of surface water and other potentially sensitive ecological habitats;
- Ensure data are representative of site conditions.

3. Summarize Appropriateness of Use of Tier 1 Lookup Tables and ESLs (see Section 1.5):

- Do Tier 1 ESLs exist for all chemicals of concern?
- Does the site have a high public profile and warrant a fully documented, detailed environmental risk assessment?
- Do soil and groundwater conditions at the site differ significantly from those assumed in development of the lookup tables (e.g., low pH at mine sites)?
- Do impacts pose a heightened threat to sensitive ecological habitats (e.g., presence of endangered or protected species)?
- Is the thickness of vadose-zone soils impacted by volatile organic compounds greater than three meters (10 feet, see Section 1.5 and Appendix 1);
- Have more than three carcinogens or five chemicals with similar noncarcinogenic health effects been identified (see Section 2.10)?

- Other issues as applicable to the site.

4. Soil and Groundwater Categorization (see Sections 2.3 and 2.4):

   - State the regulatory beneficial use of impacted or potentially impacted groundwater beneath the site; discuss the actual, likely beneficial use of groundwater based on measured or assumed quality of the groundwater and the hydrogeologic nature of the soil or bedrock containing the groundwater.
   - Characterize the soil type(s) and location of impacted soil as applicable to the lookup tables (e.g., soil stratigraphy, soil texture and permeability, depth to and thickness of impacted soil, etc.).

5. Exposure Point Concentrations (see Section 2.2, Step 7):

   - Identify maximum concentrations of chemicals present in impacted media.
   - Describe how alternative exposure point concentrations were determined (e.g., 95% UCLs), if proposed, and provide supporting data. **For residential land use scenarios, sample data should be averaged over no more than a 1,000 ft^2 area.**
   - Discuss the need to evaluate groundwater data with respect to surface water standards for potential bioaccumulation of chemicals in aquatic organisms ("Elevated threat to surface water body"), due to the size of the plume, the proximity of the plume to a body of surface water and the potential for minimal dilution of groundwater upon discharge to surface water (see Section 2.7).
   - Discuss how background concentrations of chemicals were determined, if considered for use in the risk assessment (see Section 2.8).

6. Selection of Tier 1 ESLs and Comparison to Site Data (see Section 2.2)

   - Summarize how Tier 1 ESLs were selected with respect to the information provided above and additional assumptions as applicable.
   - Compare site data to the selected summary Tier 1 ESLs (presented in Volume 1) and discuss general results.
   - If desired or recommended, compare site data to detailed ESLs for individual environmental concerns (presented in Volume 2, Appendix 1) and discuss specific, potential environmental concerns present at site.

7. Conclusions (see Section 2.9):

   - Describe the extent of soil and groundwater impacts above Tier 1 ESLs, using maps and cross sections as necessary.
   - Discuss if a condition of potential risk to human health and the environment exists at the site.
   - Discuss if a more site-specific risk assessment is warranted at the site.
   - Present a summary of recommended future actions proposed to address environmental concerns ay the site.
   - Discuss the need to impose land-use restrictions and institutional controls at the site based on the results of the Tier 1 assessment (e.g., requirements for caps,

etc.; need for covenant to deed to restrict land use to commercial/industrial purposes only, etc).

The above list is not intended to be exhaustive or representative of an exact outline required for all Tier 1 risk assessments. Requirements for completion of an adequate site investigation and Tier 1 environmental risk assessment should be discussed with the overseeing regulatory agency.

TABLE B: SHALLOW SOIL (≤3M BGS) - WATER IS NOT A CURRENT OR POTENTIAL SOURCE OF DRINKING WATER

Notes:

- Always compare final soil data for commercial/industrial sites to residential ESLs and evaluate need for formal land-use restrictions (see Section 2.9).
- Assumption that groundwater is not a current or potential source of drinking water should be approved by overseeing regulatory agency prior to use of this table (see Section 2.3).

TABLE A. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Shallow Soils (≤3m bgs)
Groundwater IS Current or Potential Source of Drinking Water

	[1]Shallow Soil		
CHEMICAL PARAMETER	[2]Residential Land Use (mg/kg)	Commercial/ Industrial Land Use Only (mg/kg)	[3]Groundwater (ug/L)
ACENAPHTHENE	1.6E+01	1.6E+01	2.0E+01
ACENAPHTHYLENE	1.3E+01	1.3E+01	3.0E+01
ACETONE	2.4E-01	2.4E-01	7.0E+02
ALDRIN	2.9E-02	1.0E-01	2.0E-03
ANTHRACENE	2.8E+00	2.8E+00	7.3E-01
ANTIMONY	6.3E+00	4.0E+01	6.0E+00
ARSENIC	5.5E+00	5.5E+00	3.6E+01
BARIUM	7.5E+02	1.5E+03	1.0E+03
BENZENE	4.4E-02	4.4E-02	1.0E+00
BENZO(a)ANTHRACENE	3.8E-01	1.3E+00	2.7E-02
BENZO(b)FLUORANTHENE	3.8E-01	1.3E+00	2.9E-02
BENZO(k)FLUORANTHENE	3.8E-01	1.3E+00	2.9E-02
BENZO(g,h,i)PERYLENE	2.7E+01	2.7E+01	1.0E-01
BENZO(a)PYRENE	3.8E-02	1.3E-01	1.4E-02
BERYLLIUM	4.0E+00	8.0E+00	2.7E+00
BIPHENYL, 1,1-	6.5E-01	6.5E-01	5.0E-01
BIS(2-CHLOROETHYL)ETHER	1.8E-04	1.8E-04	1.4E-02
BIS(2-CHLOROISOPROPYL)ETHER	5.4E-03	5.4E-03	5.0E-01
BIS(2-ETHYLHEXYL)PHTHALATE	6.6E+01	6.6E+01	4.0E+00
BORON	1.6E+00	2.0E+00	1.6E+00
BROMODICHLOROMETHANE	1.2E-02	3.9E-02	1.0E+02
BROMOFORM	2.2E+00	2.2E+00	1.0E+02
BROMOMETHANE	2.2E-01	3.9E-01	9.8E+00
CADMIUM	1.7E+00	7.4E+00	2.2E+00
CARBON TETRACHLORIDE	1.2E-02	3.5E-02	5.0E-01
CHLORDANE	4.4E-01	1.7E+00	4.0E-03
CHLOROANILINE, p-	5.3E-02	5.3E-02	5.0E+00
CHLOROBENZENE	1.5E+00	1.5E+00	2.5E+01
CHLOROETHANE	6.3E-01	8.5E-01	1.2E+01
CHLOROFORM	9.8E-02	2.7E-01	1.0E+02
CHLOROMETHANE	2.9E-01	4.2E-01	2.7E+00
CHLOROPHENOL, 2-	1.2E-02	1.2E-02	1.8E-01
CHROMIUM (Total)	5.8E+01	5.8E+01	5.0E+01
CHROMIUM III	7.5E+02	7.5E+02	1.8E+02
CHROMIUM VI	1.8E+00	1.8E+00	1.1E+01
CHRYSENE	3.8E+00	1.3E+01	2.9E-01
COBALT	4.0E+01	8.0E+01	3.0E+00
COPPER	2.3E+02	2.3E+02	3.1E+00
CYANIDE (Free)	1.0E+02	5.0E+02	1.0E+00
DIBENZO(a,h)ANTHTRACENE	1.1E-01	3.8E-01	8.5E-03
DIBROMOCHLOROMETHANE	1.9E-02	5.8E-02	1.0E+02
1,2-DIBROMO-3-CHLOROPROPANE	1.1E-03	1.1E-03	2.0E-01
DIBROMOETHANE, 1,2-	3.3E-04	3.3E-04	5.0E-02
DICHLOROBENZENE, 1,2-	1.1E+00	1.1E+00	1.0E+01

TABLE A. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Shallow Soils (≤3m bgs)
Groundwater IS Current or Potential Source of Drinking Water

CHEMICAL PARAMETER	[1]Shallow Soil		[3]Groundwater (ug/L)
	[2]Residential Land Use (mg/kg)	Commercial/ Industrial Land Use Only (mg/kg)	
DICHLOROBENZENE, 1,3-	7.2E-01	7.2E-01	6.3E+00
DICHLOROBENZENE, 1,4-	4.7E-02	1.3E-01	5.0E+00
DICHLOROBENZIDINE, 3,3-	7.7E-03	7.7E-03	2.9E-02
DICHLORODIPHENYLDICHLOROETHANE (DDD)	2.4E+00	1.0E+01	1.0E-03
DICHLORODIPHENYLDICHLOROETHYLENE (DDE)	1.7E+00	4.0E+00	1.0E-03
DICHLORODIPHENYLTRICHLOROETHANE (DDT)	1.7E+00	4.0E+00	1.0E-03
DICHLOROETHANE, 1,1-	2.0E-01	2.0E-01	5.0E+00
DICHLOROETHANE, 1,2-	4.5E-03	4.5E-03	5.0E-01
DICHLOROETHYLENE, 1,1-	1.0E+00	1.0E+00	6.0E+00
DICHLOROETHYLENE, Cis 1,2-	1.9E-01	1.9E-01	6.0E+00
DICHLOROETHYLENE, Trans 1,2-	6.7E-01	6.7E-01	1.0E+01
DICHLOROPHENOL, 2,4-	3.0E-01	3.0E-01	3.0E-01
DICHLOROPROPANE, 1,2-	5.2E-02	1.2E-01	5.0E+00
DICHLOROPROPENE, 1,3-	3.3E-02	5.9E-02	5.0E-01
DIELDRIN	2.3E-03	2.3E-03	1.9E-03
DIETHYLPHTHALATE	3.5E-02	3.5E-02	1.5E+00
DIMETHYLPHTHALATE	3.5E-02	3.5E-02	1.5E+00
DIMETHYLPHENOL, 2,4-	6.7E-01	6.7E-01	1.0E+02
DINITROPHENOL, 2,4-	4.0E-02	4.0E-02	1.4E+01
DINITROTOLUENE, 2,4-	8.5E-04	8.5E-04	1.1E-01
1,4 DIOXANE	1.8E-03	1.8E-03	3.0E+00
DIOXIN (2,3,7,8-TCDD)	4.5E-06	1.8E-05	5.0E-06
ENDOSULFAN	4.6E-03	4.6E-03	8.7E-03
ENDRIN	6.5E-04	6.5E-04	2.3E-03
ETHYLBENZENE	3.3E+00	3.3E+00	3.0E+01
FLUORANTHENE	4.0E+01	4.0E+01	8.0E+00
FLUORENE	8.9E+00	8.9E+00	3.9E+00
HEPTACHLOR	1.4E-02	1.4E-02	3.8E-03
HEPTACHLOR EPOXIDE	1.5E-02	1.5E-02	3.8E-03
HEXACHLOROBENZENE	2.7E-01	9.6E-01	1.0E+00
HEXACHLOROBUTADIENE	1.0E+00	1.0E+00	2.1E-01
HEXACHLOROCYCLOHEXANE (gamma) LINDANE	4.9E-02	4.9E-02	8.0E-02
HEXACHLOROETHANE	2.4E+00	2.4E+00	7.0E-01
INDENO(1,2,3-cd)PYRENE	3.8E-01	1.3E+00	2.9E-02
LEAD	2.0E+02	7.5E+02	2.5E+00
MERCURY	2.5E+00	1.0E+01	1.2E-02
METHOXYCHLOR	1.9E+01	1.9E+01	1.9E-02
METHYLENE CHLORIDE	7.7E-02	7.7E-02	5.0E+00
METHYL ETHYL KETONE	3.9E+00	3.9E+00	4.2E+03
METHYL ISOBUTYL KETONE	2.8E+00	2.8E+00	1.2E+02
METHYL MERCURY	1.2E+00	1.0E+01	3.0E-03
METHYLNAPHTHALENE (total 1- & 2-)	2.5E-01	2.5E-01	2.1E+00
METHYL TERT BUTYL ETHER	2.3E-02	2.3E-02	5.0E+00
MOLYBDENUM	4.0E+01	4.0E+01	3.5E+01

TABLE A. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Shallow Soils (≤3m bgs)
Groundwater IS Current or Potential Source of Drinking Water

	[1]Shallow Soil		
CHEMICAL PARAMETER	[2]Residential Land Use (mg/kg)	Commercial/ Industrial Land Use Only (mg/kg)	[3]Groundwater (ug/L)
NAPHTHALENE	4.2E+00	4.2E+00	2.1E+01
NICKEL	1.5E+02	1.5E+02	8.2E+00
PENTACHLOROPHENOL	4.4E+00	5.0E+00	1.0E+00
PERCHLORATE	7.0E-03	7.0E-03	7.0E-01
PHENANTHRENE	1.1E+01	1.1E+01	4.6E+00
PHENOL	7.6E-02	7.6E-02	5.0E+00
POLYCHLORINATED BIPHENYLS (PCBs)	2.2E-01	7.4E-01	1.4E-02
PYRENE	8.5E+01	8.5E+01	2.0E+00
SELENIUM	1.0E+01	1.0E+01	5.0E+00
SILVER	2.0E+01	4.0E+01	1.9E-01
STYRENE	1.5E+00	1.5E+00	1.0E+01
tert-BUTYL ALCOHOL	7.3E-02	7.3E-02	1.2E+01
TETRACHLOROETHANE, 1,1,1,2-	2.4E-02	2.4E-02	1.3E+00
TETRACHLOROETHANE, 1,1,2,2-	9.0E-03	1.8E-02	1.0E+00
TETRACHLOROETHYLENE	8.8E-02	2.5E-01	5.0E+00
THALLIUM	1.0E+00	1.3E+01	2.0E+00
TOLUENE	2.9E+00	2.9E+00	4.0E+01
TOXAPHENE	4.2E-04	4.2E-04	2.0E-04
TPH (gasolines)	1.0E+02	1.0E+02	1.0E+02
TPH (middle distillates)	1.0E+02	1.0E+02	1.0E+02
TPH (residual fuels)	5.0E+02	1.0E+03	1.0E+02
TRICHLOROBENZENE, 1,2,4-	7.6E+00	7.6E+00	2.5E+01
TRICHLOROETHANE, 1,1,1-	7.8E+00	7.8E+00	6.2E+01
TRICHLOROETHANE, 1,1,2-	3.3E-02	7.0E-02	5.0E+00
TRICHLOROETHYLENE	2.6E-01	4.6E-01	5.0E+00
TRICHLOROPHENOL, 2,4,5-	1.8E-01	1.8E-01	1.1E+01
TRICHLOROPHENOL, 2,4,6-	1.7E-01	1.7E-01	5.0E-01
VANADIUM	1.1E+02	2.0E+02	1.5E+01
VINYL CHLORIDE	6.7E-03	1.9E-02	5.0E-01
XYLENES	1.5E+00	1.5E+00	1.3E+01
ZINC	6.0E+02	6.0E+02	8.1E+01

TABLE A. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Shallow Soils (≤3m bgs)
Groundwater IS Current or Potential Source of Drinking Water

CHEMICAL PARAMETER	[1]Shallow Soil		[3]Groundwater (ug/L)
	[2]Residential Land Use (mg/kg)	Commercial/ Industrial Land Use Only (mg/kg)	
Electrical Conductivity (mS/cm, USEPA Method 120.1 MOD)	2.0	4.0	not applicable
Sodium Adsorption Ratio	5.0	12	not applicable

Notes:

1. Shallow soils defined as soils less than or equal to 3 meters (approximately 10 feet) below ground surface.
2. Category "Residential Land Use" generally considered adequate for other sensitive uses (e.g., day-care centers, hospitals, etc.)
3. Assumes potential discharge of groundwater into a freshwater, marine or estuary surface water system.

Source of soil ESLs: Refer to Appendix 1, Tables A-1 and A-2.

Source of groundwater ESLs: Refer to Appendix 1, Table F-1a.

Soil data should be reported on dry-weight basis (see Appendix 1, Section 6.2).

Soil ESLs intended to address direct-exposure, groundwater protection, ecologic (urban areas) and nuisance concerns under noted land-use scenarios. **Soil gas data should be collected for additional evaluation of potential indoor-air impacts at sites with significant areas of VOC-impacted soil. See Section 2.6 and Table E.**

Groundwater ESLs intended to be address drinking water, surface water, indoor-air and nuisance concerns. **Use in conjunction with soil gas screening levels to more closely evaluate potential impacts to indoor-air if groundwater screening levels for this concern approached or exceeded (refer to Section 2.6 and Appendix 1, Table F-1a).**

Aquatic habitat goals for bioaccumulation concerns not considered in selection of groundwater goals (refer to Section 2.7).

Refer to appendices for summary of ESL components.

TPH -Total Petroleum Hydrocarbons. TPH ESLs must be used in conjunction with ESLs for related chemicals (e.g., BTEX, PAHs, oxidizers, etc.). See Volume 1, Section 2.2 and Appendix 1, Chapter 5.

TABLE B. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Shallow Soils (≤3m bgs)
Groundwater IS NOT a Current or Potential Source of Drinking Water

	[1]Shallow Soil		
CHEMICAL PARAMETER	[2]Residential Land Use (mg/kg)	Commercial/ Industrial Land Use Only (mg/kg)	[3]Groundwater (ug/L)
ACENAPHTHENE	1.9E+01	1.9E+01	2.3E+01
ACENAPHTHYLENE	1.3E+01	1.3E+01	3.0E+01
ACETONE	5.0E-01	5.0E-01	1.5E+03
ALDRIN	2.9E-02	1.0E-01	1.3E-01
ANTHRACENE	2.8E+00	2.8E+00	7.3E-01
ANTIMONY	6.3E+00	4.0E+01	3.0E+01
ARSENIC	5.5E+00	5.5E+00	3.6E+01
BARIUM	7.5E+02	1.5E+03	1.0E+03
BENZENE	1.8E-01	3.8E-01	4.6E+01
BENZO(a)ANTHRACENE	3.8E-01	1.3E+00	2.7E-02
BENZO(b)FLUORANTHENE	3.8E-01	1.3E+00	2.9E-02
BENZO(k)FLUORANTHENE	3.8E-01	1.3E+00	4.0E-01
BENZO(g,h,i)PERYLENE	2.7E+01	2.7E+01	1.0E-01
BENZO(a)PYRENE	3.8E-02	1.3E-01	1.4E-02
BERYLLIUM	4.0E+00	8.0E+00	2.7E+00
BIPHENYL, 1,1-	6.5E+00	6.5E+00	5.0E+00
BIS(2-CHLOROETHYL)ETHER	4.0E-03	1.3E-02	6.1E+01
BIS(2-CHLOROISOPROPYL)ETHER	6.6E-01	6.6E-01	6.1E+01
BIS(2-ETHYLHEXYL)PHTHALATE	1.6E+02	5.3E+02	3.2E+01
BORON	1.6E+00	2.0E+00	1.6E+00
BROMODICHLOROMETHANE	1.2E-02	3.9E-02	1.6E+02
BROMOFORM	6.1E+01	6.9E+01	3.2E+03
BROMOMETHANE	2.2E-01	5.1E-01	1.6E+02
CADMIUM	1.7E+00	7.4E+00	2.2E+00
CARBON TETRACHLORIDE	1.2E-02	3.5E-02	9.5E+00
CHLORDANE	4.4E-01	1.7E+00	4.0E-03
CHLOROANILINE, p-	5.3E-02	5.3E-02	5.0E+00
CHLOROBENZENE	1.5E+00	1.5E+00	2.5E+01
CHLOROETHANE	6.3E-01	8.5E-01	1.2E+01
CHLOROFORM	9.8E-02	2.7E-01	3.4E+02
CHLOROMETHANE	2.9E-01	8.1E-01	1.7E+02
CHLOROPHENOL, 2-	1.2E-01	1.2E-01	1.8E+00
CHROMIUM (Total)	5.8E+01	5.8E+01	1.8E+02
CHROMIUM III	7.5E+02	7.5E+02	1.8E+02
CHROMIUM VI	1.8E+00	1.8E+00	1.1E+01
CHRYSENE	3.8E+00	1.3E+01	3.5E-01
COBALT	4.0E+01	8.0E+01	3.0E+00
COPPER	2.3E+02	2.3E+02	3.1E+00
CYANIDE (Free)	1.0E+02	5.0E+02	1.0E+00
DIBENZO(a,h)ANTHTRACENE	1.1E-01	3.8E-01	2.5E-01
DIBROMOCHLOROMETHANE	1.9E-02	5.8E-02	1.8E+02

TABLE B. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Shallow Soils (≤3m bgs)
Groundwater IS NOT a Current or Potential Source of Drinking Water

	[1]Shallow Soil		
CHEMICAL PARAMETER	[2]Residential Land Use (mg/kg)	Commercial/ Industrial Land Use Only (mg/kg)	[3]Groundwater (ug/L)
1,2-DIBROMO-3-CHLOROPROPANE	1.1E-03	1.1E-03	2.0E-01
DIBROMOETHANE, 1,2-	7.3E-03	2.1E-02	1.6E+02
DICHLOROBENZENE, 1,2-	1.6E+00	1.6E+00	1.4E+01
DICHLOROBENZENE, 1,3-	3.2E+00	7.4E+00	6.5E+01
DICHLOROBENZENE, 1,4-	4.7E-02	1.3E-01	1.5E+01
DICHLOROBENZIDINE, 3,3-	4.0E-01	1.4E+00	2.5E+02
DICHLORODIPHENYLDICHLOROETHANE (DDD)	2.4E+00	1.0E+01	1.0E-03
DICHLORODIPHENYLDICHLOROETHYLENE (DDE)	1.7E+00	4.0E+00	1.0E-03
DICHLORODIPHENYLTRICHLOROETHANE (DDT)	1.7E+00	4.0E+00	1.0E-03
DICHLOROETHANE, 1,1-	3.3E-01	9.1E-01	4.7E+01
DICHLOROETHANE, 1,2-	2.5E-02	6.9E-02	2.0E+02
DICHLOROETHYLENE, 1,1-	4.3E+00	4.3E+00	2.5E+01
DICHLOROETHYLENE, Cis 1,2-	1.6E+00	3.6E+00	5.9E+02
DICHLOROETHYLENE, Trans 1,2-	3.1E+00	7.3E+00	5.9E+02
DICHLOROPHENOL, 2,4-	3.0E+00	3.0E+00	3.0E+00
DICHLOROPROPANE, 1,2-	5.2E-02	1.5E-01	1.0E+02
DICHLOROPROPENE, 1,3-	3.3E-02	9.1E-02	4.9E+01
DIELDRIN	2.3E-03	2.3E-03	1.9E-03
DIETHYLPHTHALATE	3.5E-02	3.5E-02	1.5E+00
DIMETHYLPHTHALATE	3.5E-02	3.5E-02	1.5E+00
DIMETHYLPHENOL, 2,4-	7.4E-01	7.4E-01	1.1E+02
DINITROPHENOL, 2,4-	2.1E-01	2.1E-01	7.5E+01
DINITROTOLUENE, 2,4-	8.6E-01	8.6E-01	1.2E+02
1,4 DIOXANE	1.8E+01	3.0E+01	5.0E+04
DIOXIN (2,3,7,8-TCDD)	4.5E-06	1.8E-05	5.0E-06
ENDOSULFAN	4.6E-03	4.6E-03	8.7E-03
ENDRIN	6.5E-04	6.5E-04	2.3E-03
ETHYLBENZENE	4.7E+00	1.3E+01	2.9E+02
FLUORANTHENE	4.0E+01	4.0E+01	8.0E+00
FLUORENE	8.9E+00	8.9E+00	3.9E+00
HEPTACHLOR	1.4E-02	1.4E-02	3.8E-03
HEPTACHLOR EPOXIDE	1.5E-02	1.5E-02	3.8E-03
HEXACHLOROBENZENE	2.7E-01	9.6E-01	3.7E+00
HEXACHLOROBUTADIENE	3.7E+00	2.2E+01	4.7E+00
HEXACHLOROCYCLOHEXANE (gamma) LINDANE	4.9E-02	4.9E-02	8.0E-02
HEXACHLOROETHANE	1.2E+01	4.1E+01	1.2E+01
INDENO(1,2,3-cd)PYRENE	3.8E-01	1.3E+00	2.9E-02
LEAD	2.0E+02	7.5E+02	2.5E+00
MERCURY	2.5E+00	1.0E+01	1.2E-02
METHOXYCHLOR	1.9E+01	1.9E+01	1.9E-02
METHYLENE CHLORIDE	5.2E-01	1.5E+00	2.2E+03

TABLE B. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Shallow Soils (≤3m bgs)
Groundwater IS NOT a Current or Potential Source of Drinking Water

	[1]Shallow Soil		
CHEMICAL PARAMETER	[2]Residential Land Use (mg/kg)	Commercial/ Industrial Land Use Only (mg/kg)	[3]Groundwater (ug/L)
METHYL ETHYL KETONE	1.3E+01	1.3E+01	1.4E+04
METHYL ISOBUTYL KETONE	3.9E+00	3.9E+00	1.7E+02
METHYL MERCURY	1.2E+00	1.0E+01	3.0E-03
METHYLNAPHTHALENE (total 1- & 2-)	2.5E-01	2.5E-01	2.1E+00
METHYL TERT BUTYL ETHER	2.0E+00	5.6E+00	1.8E+03
MOLYBDENUM	4.0E+01	4.0E+01	2.4E+02
NAPHTHALENE	4.5E+00	4.8E+00	2.4E+01
NICKEL	1.5E+02	1.5E+02	8.2E+00
PENTACHLOROPHENOL	4.4E+00	5.0E+00	7.9E+00
PERCHLORATE	1.2E+00	1.2E+00	6.0E+02
PHENANTHRENE	1.1E+01	1.1E+01	4.6E+00
PHENOL	1.9E+01	1.9E+01	1.3E+03
POLYCHLORINATED BIPHENYLS (PCBs)	2.2E-01	7.4E-01	1.4E-02
PYRENE	8.5E+01	8.5E+01	2.0E+00
SELENIUM	1.0E+01	1.0E+01	5.0E+00
SILVER	2.0E+01	4.0E+01	1.9E-01
STYRENE	1.5E+01	1.5E+01	1.0E+02
tert-BUTYL ALCOHOL	1.0E+02	1.1E+02	1.8E+04
TETRACHLOROETHANE, 1,1,1,2-	3.1E+00	7.2E+00	9.3E+02
TETRACHLOROETHANE, 1,1,2,2-	9.0E-03	2.5E-02	1.9E+02
TETRACHLOROETHYLENE	8.8E-02	2.5E-01	1.2E+02
THALLIUM	1.0E+00	1.3E+01	2.0E+01
TOLUENE	9.3E+00	9.3E+00	1.3E+02
TOXAPHENE	4.2E-04	4.2E-04	2.0E-04
TPH (gasolines)	1.0E+02	4.0E+02	5.0E+02
TPH (middle distillates)	5.0E+02	5.0E+02	6.4E+02
TPH (residual fuels)	5.0E+02	1.0E+03	6.4E+02
TRICHLOROBENZENE, 1,2,4-	7.6E+00	7.6E+00	2.5E+01
TRICHLOROETHANE, 1,1,1-	7.8E+00	7.8E+00	6.2E+01
TRICHLOROETHANE, 1,1,2-	3.3E-02	9.1E-02	3.5E+02
TRICHLOROETHYLENE	2.6E-01	7.3E-01	3.6E+02
TRICHLOROPHENOL, 2,4,5-	1.8E-01	1.8E-01	1.1E+01
TRICHLOROPHENOL, 2,4,6-	6.9E+00	1.0E+01	4.9E+02
VANADIUM	1.1E+02	2.0E+02	1.9E+01

TABLE B. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Shallow Soils (≤3m bgs)
Groundwater IS NOT a Current or Potential Source of Drinking Water

	[1]Shallow Soil		
CHEMICAL PARAMETER	[2]Residential Land Use (mg/kg)	Commercial/ Industrial Land Use Only (mg/kg)	[3]Groundwater (ug/L)
VINYL CHLORIDE	6.7E-03	1.9E-02	4.0E+00
XYLENES	1.5E+00	1.5E+00	1.3E+01
ZINC	6.0E+02	6.0E+02	8.1E+01
Electrical Conductivity (mS/cm, USEPA Method 120.1 MOD)	2.0	4.0	not applicable
Sodium Adsorption Ratio	5.0	12	not applicable

Notes:

1. Shallow soils defined as soils less than or equal to 3 meters (approximately 10 feet) below ground surface.
2. Category "Residential Land Use" generally considered adequate for other sensitive uses (e.g., day-care centers, hospitals, etc.)
3. Assumes potential discharge of groundwater into marine or estuary surface water system.

Source of soil ESLs: Refer to Appendix 1, Tables A-1 and A-2.

Source of groundwater ESLs: Refer to Appendix 1, Table F-1b.

Soil data should be reported on dry-weight basis (see Appendix 1, Section 6.2).

Soil ESLs intended to address direct-exposure, groundwater protection, ecologic (urban areas) and nuisance concerns under noted land-use scenarios. **Soil gas data should be collected for additional evaluation of potential indoor-air impacts at at sites with significant areas of VOC-impacted soil. See Section 2.6 and Table E.**

Groundwater ESLs intended to address surface water, indoor-air and nuisance concerns. **Use in conjunction with soil gas screening levels to more closely evaluate potential impacts to indoor-air if groundwater screening levels for this concern approached or exceeded (refer to Section 2.6 and Appendix 1, Table F-1a).**

Aquatic habitat goals for bioaccumulation concerns not considered in selection of groundwater goals (refer to Section 2.7).

Refer to appendices for summary of ESL components.

TPH -Total Petroleum Hydrocarbons. TPH ESLs must be used in conjunction with ESLs for related chemicals (e.g., BTEX, PAHs, oxidizers, etc.). See Volume 1, Section 2.2 and Appendix 1, Chapter 5.

TABLE F: SURFACE WATER

TABLE F. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Surface Water Bodies

	SURFACE WATER SCREENING LEVELS		
CHEMICAL PARAMETER	[1]Freshwater (ug/L)	[2]Marine (ug/L)	[3]Estuarine (ug/L)
ACENAPHTHENE	2.0E+01	2.0E+01	2.0E+01
ACENAPHTHYLENE	3.0E+01	3.0E+01	3.0E+01
ACETONE	7.0E+02	1.5E+03	1.5E+03
ALDRIN	1.4E-04	1.4E-04	1.4E-04
ANTHRACENE	7.3E-01	7.3E-01	7.3E-01
ANTIMONY	6.0E+00	5.0E+02	5.0E+02
ARSENIC	1.4E-01	1.4E-01	1.4E-01
BARIUM	1.0E+03	1.0E+03	1.0E+03
BENZENE	1.0E+00	7.1E+01	7.1E+01
BENZO(a)ANTHRACENE	2.7E-02	2.7E-02	2.7E-02
BENZO(b)FLUORANTHENE	2.9E-02	2.9E-02	2.9E-02
BENZO(k)FLUORANTHENE	2.9E-02	4.9E-02	4.9E-02
BENZO(g,h,i)PERYLENE	1.0E-01	1.0E-01	1.0E-01
BENZO(a)PYRENE	1.4E-02	1.4E-02	1.4E-02
BERYLLIUM	2.7E+00	2.7E+00	2.7E+00
BIPHENYL, 1,1-	5.0E-01	5.0E-01	5.0E-01
BIS(2-CHLOROETHYL)ETHER	1.4E-02	1.4E+00	1.4E+00
BIS(2-CHLOROISOPROPYL)ETHER	5.0E-01	6.1E+01	6.1E+01
BIS(2-ETHYLHEXYL)PHTHALATE	4.0E+00	5.9E+00	5.9E+00
BORON	1.6E+00	1.6E+00	1.6E+00
BROMODICHLOROMETHANE	1.0E+02	3.2E+03	3.2E+03
BROMOFORM	1.0E+02	3.6E+02	3.6E+02
BROMOMETHANE	9.8E+00	3.2E+03	3.2E+03
CADMIUM	2.2E+00	9.3E+00	9.3E+00
CARBON TETRACHLORIDE	5.0E-01	4.4E+00	4.4E+00
CHLORDANE	5.9E-04	5.9E-04	5.9E-04
CHLOROANILINE, p-	5.0E+00	5.0E+00	5.0E+00
CHLOROBENZENE	2.5E+01	5.0E+01	5.0E+01
CHLOROETHANE	1.2E+01	1.2E+01	1.2E+01
CHLOROFORM	1.0E+02	4.7E+02	4.7E+02
CHLOROMETHANE	2.7E+00	3.2E+03	3.2E+03
CHLOROPHENOL, 2-	1.8E-01	1.8E-01	1.8E-01
CHROMIUM (Total)	5.0E+01	1.8E+02	1.8E+02
CHROMIUM III	1.8E+02	1.8E+02	1.8E+02
CHROMIUM VI	1.1E+01	5.0E+01	5.0E+01
CHRYSENE	4.9E-02	4.9E-02	4.9E-02
COBALT	3.0E+00	3.0E+00	3.0E+00
COPPER	9.0E+00	3.1E+00	3.1E+00
CYANIDE (Free)	5.2E+00	1.0E+00	1.0E+00
DIBENZO(a,h)ANTHTRACENE	8.5E-03	4.9E-02	4.9E-02
DIBROMOCHLOROMETHANE	4.6E+01	4.6E+01	4.6E+01
1,2-DIBROMO-3-CHLOROPROPANE	2.0E-01	2.0E-01	2.0E-01
DIBROMOETHANE, 1,2-	5.0E-02	1.4E+03	1.4E+03
DICHLOROBENZENE, 1,2-	1.0E+01	1.0E+01	1.0E+01
DICHLOROBENZENE, 1,3-	6.3E+00	6.5E+01	6.5E+01

TABLE F. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Surface Water Bodies

CHEMICAL PARAMETER	SURFACE WATER SCREENING LEVELS		
	[1]Freshwater (ug/L)	[2]Marine (ug/L)	[3]Estuarine (ug/L)
DICHLOROBENZENE, 1,4-	5.0E+00	1.1E+01	1.1E+01
DICHLOROBENZIDINE, 3,3-	2.9E-02	7.7E-02	7.7E-02
DICHLORODIPHENYLDICHLOROETHANE (DDD)	8.4E-04	8.4E-04	8.4E-04
DICHLORODIPHENYLDICHLOROETHYLENE (DDE)	5.9E-04	5.9E-04	5.9E-04
DICHLORODIPHENYLTRICHLOROETHANE (DDT)	5.9E-04	5.9E-04	5.9E-04
DICHLOROETHANE, 1,1-	5.0E+00	4.7E+01	4.7E+01
DICHLOROETHANE, 1,2-	5.0E-01	9.9E+01	9.9E+01
DICHLOROETHYLENE, 1,1-	3.2E+00	3.2E+00	3.2E+00
DICHLOROETHYLENE, Cis 1,2-	6.0E+00	5.9E+02	5.9E+02
DICHLOROETHYLENE, Trans 1,2-	1.0E+01	2.6E+02	2.6E+02
DICHLOROPHENOL, 2,4-	3.0E-01	3.0E-01	3.0E-01
DICHLOROPROPANE, 1,2-	5.0E+00	1.0E+01	1.0E+01
DICHLOROPROPENE, 1,3-	5.0E-01	1.2E+02	1.2E+02
DIELDRIN	2.2E-03	1.9E-03	1.9E-03
DIETHYLPHTHALATE	1.5E+00	1.7E+00	1.7E+00
DIMETHYLPHTHALATE	1.5E+00	1.7E+00	1.7E+00
DIMETHYLPHENOL, 2,4-	1.0E+02	1.1E+02	1.1E+02
DINITROPHENOL, 2,4-	1.4E+01	7.5E+01	7.5E+01
DINITROTOLUENE, 2,4-	1.1E-01	9.1E+00	9.1E+00
1,4 DIOXANE	3.0E+00	5.0E+04	5.0E+04
DIOXIN (2,3,7,8-TCDD)	1.4E-08	1.4E-08	1.4E-08
ENDOSULFAN	5.6E-02	8.7E-03	8.7E-03
ENDRIN	3.6E-02	2.3E-03	2.3E-03
ETHYLBENZENE	3.0E+01	3.0E+01	3.0E+01
FLUORANTHENE	8.1E+00	8.0E+00	8.0E+00
FLUORENE	3.9E+00	3.9E+00	3.9E+00
HEPTACHLOR	2.1E-04	2.1E-04	2.1E-04
HEPTACHLOR EPOXIDE	1.1E-04	1.1E-04	1.1E-04
HEXACHLOROBENZENE	7.7E-04	7.7E-04	7.7E-04
HEXACHLOROBUTADIENE	2.1E-01	4.7E+00	4.7E+00
HEXACHLOROCYCLOHEXANE (gamma) LINDANE	6.3E-02	6.3E-02	6.3E-02
HEXACHLOROETHANE	7.0E-01	8.9E+00	8.9E+00
INDENO(1,2,3-cd)PYRENE	2.9E-02	2.9E-02	2.9E-02
LEAD	2.5E+00	8.1E+00	8.1E+00
MERCURY	5.1E-02	2.5E-02	2.5E-02
METHOXYCHLOR	1.9E-02	1.9E-02	1.9E-02
METHYLENE CHLORIDE	5.0E+00	1.6E+03	1.6E+03
METHYL ETHYL KETONE	4.2E+03	8.4E+03	8.4E+03
METHYL ISOBUTYL KETONE	1.2E+02	1.7E+02	1.7E+02
METHYL MERCURY	3.0E-03	3.0E-03	3.0E-03
METHYLNAPHTHALENE (total 1- & 2-)	2.1E+00	2.1E+00	2.1E+00
METHYL TERT BUTYL ETHER	5.0E+00	1.8E+02	1.8E+02
MOLYBDENUM	3.5E+01	2.4E+02	2.4E+02
NAPHTHALENE	2.1E+01	2.1E+01	2.1E+01
NICKEL	5.2E+01	8.2E+00	8.2E+00

TABLE F. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Surface Water Bodies

CHEMICAL PARAMETER	SURFACE WATER SCREENING LEVELS		
	[1]Freshwater (ug/L)	[2]Marine (ug/L)	[3]Estuarine (ug/L)
PENTACHLOROPHENOL	1.0E+00	7.9E+00	7.9E+00
PERCHLORATE	7.0E-01	6.0E+02	6.0E+02
PHENANTHRENE	6.3E+00	4.6E+00	4.6E+00
PHENOL	5.0E+00	1.3E+03	1.3E+03
POLYCHLORINATED BIPHENYLS (PCBs)	1.7E-04	1.7E-04	1.7E-04
PYRENE	2.0E+00	2.0E+00	2.0E+00
SELENIUM	5.0E+00	7.1E+01	7.1E+01
SILVER	3.4E-01	1.9E-01	1.9E-01
STYRENE	1.0E+01	1.1E+01	1.1E+01
tert-BUTYL ALCOHOL	1.2E+01	1.8E+04	1.8E+04
TETRACHLOROETHANE, 1,1,1,2-	1.3E+00	9.3E+02	9.3E+02
TETRACHLOROETHANE, 1,1,2,2-	1.0E+00	1.1E+01	1.1E+01
TETRACHLOROETHYLENE	5.0E+00	8.9E+00	8.9E+00
THALLIUM	2.0E+00	6.3E+00	6.3E+00
TOLUENE	4.0E+01	4.0E+01	4.0E+01
TOXAPHENE	2.0E-04	2.0E-04	2.0E-04
TPH (gasolines)	1.0E+02	3.7E+03	3.7E+03
TPH (middle distillates)	1.0E+02	6.4E+02	6.4E+02
TPH (residual fuels)	1.0E+02	6.4E+02	6.4E+02
TRICHLOROBENZENE, 1,2,4-	2.5E+01	6.5E+01	6.5E+01
TRICHLOROETHANE, 1,1,1-	6.2E+01	6.2E+01	6.2E+01
TRICHLOROETHANE, 1,1,2-	5.0E+00	4.2E+01	4.2E+01
TRICHLOROETHYLENE	5.0E+00	8.1E+01	8.1E+01
TRICHLOROPHENOL, 2,4,5-	6.3E+01	1.1E+01	1.1E+01
TRICHLOROPHENOL, 2,4,6-	5.0E-01	6.5E+00	6.5E+00
VANADIUM	1.5E+01	1.9E+01	1.9E+01
VINYL CHLORIDE	5.0E-01	5.3E+02	5.3E+02
XYLENES	1.3E+01	1.3E+01	1.3E+01
ZINC	1.2E+02	8.1E+01	8.1E+01

TABLE F. ENVIRONMENTAL SCREENING LEVELS (ESLs)
Surface Water Bodies

CHEMICAL PARAMETER	SURFACE WATER SCREENING LEVELS		
	[1]Freshwater (ug/L)	[2]Marine (ug/L)	[3]Estuarine (ug/L)
Electrical Conductivity (mS/cm, USEPA Method 120.1 MOD)	not applicable	not applicable	not applicable
Sodium Adsorption Ratio	not applicable	not applicable	not applicable

Notes:

1. Source of Freshwater ESLs: Refer to Appendix 1, Table F-2a
2. Source of Marine ESLs: Refer to Appendix 1, Table F-2b.
3. Source of Estuarine ESLs: Refer to Appendix 1, Table F-2c.

Surface water screening levels lowest of drinking water goal (freshwater only), chronic aquatic habitat goal, goal to address bioaccumulation in aquatic organisms and subsequent consumption by humans, and general nuisance goal (odors, etc.). Refer to Section 2.7 of text for discussion.

Estuarine screening levels lowest of freshwater and marine screening levels.

TPH -Total Petroleum Hydrocarbons. TPH ESLs must be used in conjunction with ESLs for related chemicals (e.g., BTEX, PAHs, oxidizers, etc.). See Section 2.2 and Appendix 1, Chapter 5.

April 2004 SCH No. 2003031044

PARAMOUNT REFINERY

CLEAN FUELS PROJECT

FINAL ENVIRONMENTAL IMPACT REPORT

Volume I: Final Environmental Impact Report

Executive Officer
Barry Wallerstein, D. Env.

Deputy Executive Officer
Planning, Rules, and Area Sources
Elaine Chang, DrPH

Assistant Deputy Executive Officer
Planning, Rule Development and Area Sources
Laki Tisopulos, Ph.D., P.E.

Planning and Rules Manager
CEQA and Socioeconomic Analyses
Susan Nakamura

Submitted to:
SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT

Prepared by:
ENVIRONMENTAL AUDIT, INC.

Reviewed by: James Koizumi - Air Quality Specialist
Steve Smith, Ph.D. - Program Supervisor
Barbara Baird - District Counsel

SOUTH COAST AIR QUALITY MANAGEMENT DISTRICT GOVERNING BOARD

Chairman: WILLIAM A. BURKE, Ed.D.
Speaker of the Assembly Representative

Vice Chairman: S. ROY WILSON, Ed.D.
Supervisor, Fourth District
Riverside County Representative

MEMBERS

MICHAEL D. ANTONOVICH
Supervisor, Fifth District
Los Angeles County Representative

JANE CARNEY
Senate Rules Committee Appointee

BEATRICE J. S. LAPISTO – KIRTLEY
Councilmember, City of Bradbury
Cities Representative, Los Angeles County, Eastern Region

RONALD O. LOVERIDGE
Mayor, City of Riverside
Cities Representative, Riverside County

BILL POSTMUS
Supervisor, First District
San Bernardino County Representative

JAMES W. SILVA
Supervisor, Second District
Orange County Representative

CYNTHIA VERDUGO-PERALTA
Governor's Appointee

JAN PERRY
Cities Representative, Los Angeles County, Western Region

DENNIS YATES
Councilmember, City of Chino
Cities Representative, San Bernardino County

EXECUTIVE OFFICER

BARRY WALLERSTEIN, D. Env.

TABLE OF CONTENTS
PARAMOUNT REFINERY CLEAN FUELS PROJECT
DRAFT ENVIRONMENTAL IMPACT REPORT

VOLUME I Page No.

VOLUME I

Page No.

VOLUME I

VOLUME I Page No.

APPENDICES:

FIGURES:

VOLUME I Page No.

FIGURES: (cont.)

TABLES:

VOLUME I Page No.

VOLUME II

Health Risk Assessment

M:\DBS\2150 Paramount\Electronic EIR\Paramount CFP EIR 0.doc

CHAPTER 5
CUMULATIVE IMPACTS

INTRODUCTION
LOCAL REFINERIES
OTHER NEARBY PROJECTS
AIR QUALITY
HAZARDS AND HAZARDOUS MATERIALS
TRANSPORTATION/TRAFFIC

CHAPTER 5.0

CUMULATIVE IMPACTS

A. INTRODUCTION

CEQA Guidelines §15130(a) requires an EIR to discuss cumulative impacts of a project when the project's incremental effect is cumulative considerable, as defined in §15065(c). There are a number of projects proposed for development in the Paramount area that may contribute cumulative regional impacts to those generated by the Paramount Refinery's proposed project. These include reformulated fuels modifications planned by other petroleum refineries in Basin as well as other local projects. Figure 5-1 shows the locations of the six major southern California refineries. The reformulated fuels modifications are to be completed in order to supply reformulated gasoline as required by Executive Order D-5-99 and the resulting CARB RFG Phase 3 requirements. The discussion below lists projects which are reasonably expected to proceed in the foreseeable future, i.e., project information has been submitted to a public agency. Cumulative construction impacts were evaluated herein if the major portion of construction is expected to occur during the same construction period as Paramount's Clean Fuels project.

Public agencies were contacted to obtain information on projects in the Paramount area. Figure 5-2 identifies by number the location of each of the projects discussed below. The number is used to identify the related projects throughout the discussion of cumulative impacts. Localized impacts were assumed to include projects which would occur within the same timeframe as the Paramount's Clean Fuels project and which are in the Paramount area. These projects generally include the RFG Phase 3 project at the British Petroleum (formerly ARCO) refinery; the RFG Phase 3 project at the Conoco-Phillips (formerly Tosco) refinery; the RFG Phase 3 project at the Shell (formerly Equilon) refinery. Regional impacts were assumed to include projects throughout the Basin, e.g., all refineries.

Some of the impacts of the proposed Paramount project would primarily occur during the construction phase, e.g., traffic. Other impacts would primarily occur during the operational phase, e.g., hazards. Other impacts would occur during both phases, e.g., air quality.

B. LOCAL REFINERIES

1) Conoco-Phillips

The Conoco-Phillips Refinery (formerly Tosco and Unocal) is approximately 18 miles southwest of the Paramount Refinery. It consists of facilities at two locations (Wilmington and Carson) approximately three miles apart. The two integrated sites transfer raw, intermediate, and finished materials primarily by pipelines. Finished

products are transferred from the Wilmington location via the Torrance Tank Farm pipeline to distribution terminals in the southern California area or to interstate pipelines. The RFG Phase 3 project will involve physical changes only to the Conoco-Phillips Wilmington Plant, located at 1660 W. Anaheim Street, Wilmington, California, 90745.

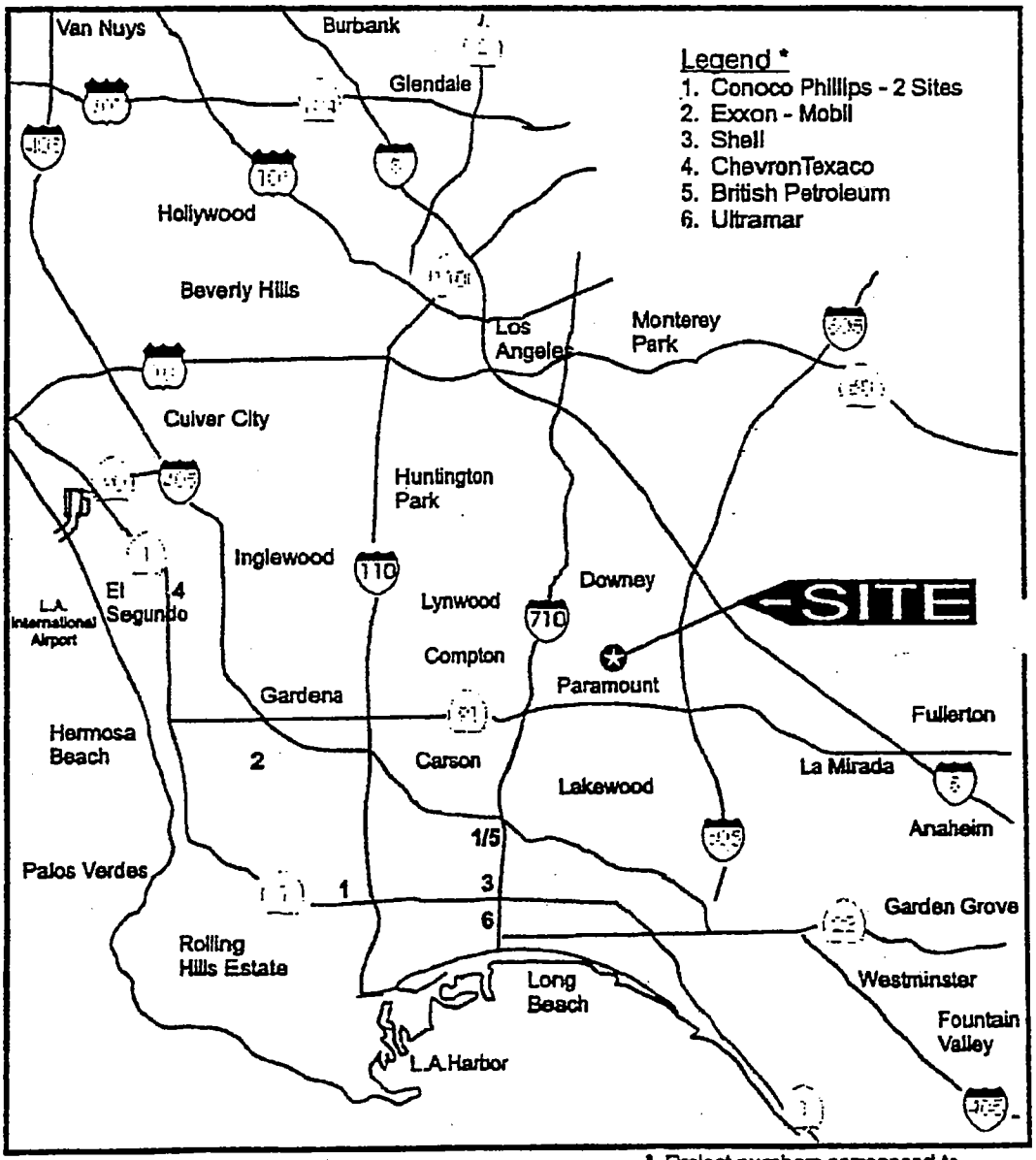
Conoco-Phillips proposed to modify existing process units at the Wilmington Plant in order to produce gasoline in compliance with CARB's Phase 3 requirements (SCAQMD, 2001). No new process units were proposed at the Refinery.

Modifications to the following units are proposed:

- Alkylation Unit (fractionation equipment, refrigeration compressor system, pumps, heaters and exchangers)
- Acid Plant (vapor recovery system)
- Butamer Unit (pumps)
- Catalytic Light Ends Fractionation Unit (fractionation equipment, pumps and piping)
- Rail Car Offloading Facilities
- Butane Storage Tank System
- Storage Tank System
- Utilities (the nitrogen, steam, water, condensate, electrical, hydrocarbon relief, and fresh/spent acid systems).

Associated modifications and additions to storage facilities, pipelines and support facilities are also expected (SCAQMD, 2001). The original CARB Phase 3 project was evaluated in the Final EIR (SCAQMD, SCH No. 2000091056, certified April 5, 2001). An Addendum to the April 5, 2001 Final EIR was prepared to include modifications to the Los Angeles Terminal including expansion of rail service at the terminal to include the unloading of ethanol (SCAQMD 2003b).

In addition to the CARB Phase 3 project, Conoco-Phillips has been issued permits for an Ethanol Import and Distribution Project. In order to produce gasoline without MTBE as required by the Governor's Executive Order and to remain compliant with state and federal reformulated fuel standards, Conoco-Phillips will replace MTBE with ethanol. This project is comprised of modifying existing facilities to permit ethanol to be received into the Marine Terminal for transshipment through the Wilmington Plant for ultimate blending into gasoline at existing, offsite marketing terminals. A Negative Declaration has been completed (SCAQMD, 2000b) and approved for this project. Because this project was found not to have any significant effect on the environment, no cumulative impacts are expected. The ConocoPhillips Refinery is located approximately fifteen miles from the Paramount Refinery so cumulative localized impacts are not expected to occur.



Environmental Audit, Inc.

REGIONAL MAP SHOWING REFINERIES

Figure 5.

Project No: 2150
N:\2150\Regional

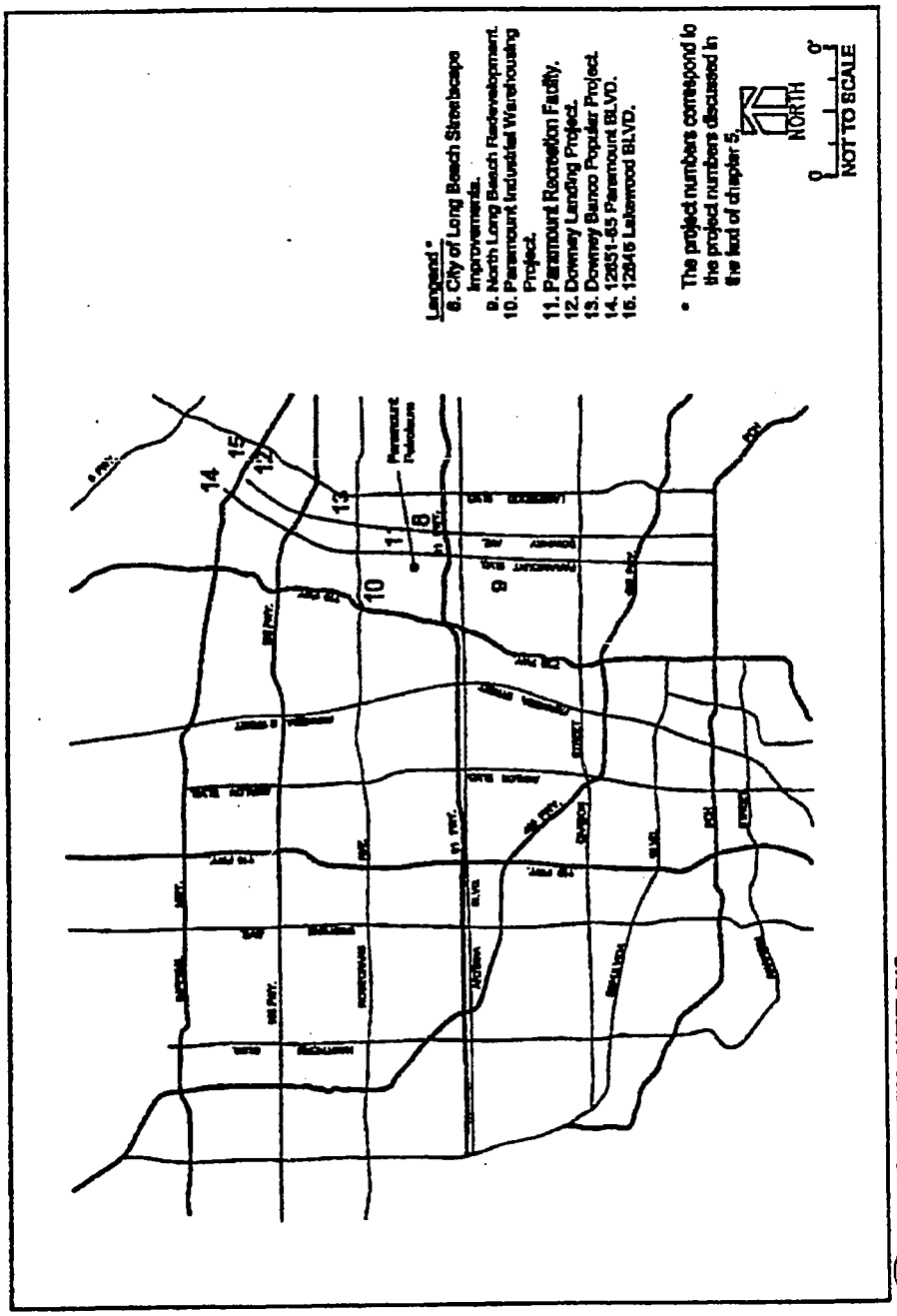


Figure 5-2

2) Exxon-Mobil

The Exxon-Mobil refinery is located at 3700 W. 190th Street in Torrance, about fourteen miles southwest of the Paramount Refinery. The RFG Phase 3 project includes modifications and/or additions to the following equipment:

- Light FCCU – Unsaturated Gas Plant Debutanizer
- Light HDC – Stabilizer, Gasoline Component Isolation Piping
- Deisobutanizer Tower – Butane Handling, KOH Tower
- Alky Feed – Hydrotreating
- Liquefied Petroleum Rail Facilities – Vessels, Loading and Additional Track
- Fuel Ethanol Storage – Tanks, Rail and Off-loading Facilities
- Gasoline Storage – Tanks
- FCC – Hydrotreater Reactors and Heater Modifications
- Alkylate – Additive Water Wash System and Merox System
- Sulfur Contamination Elimination – Overhead Compressor Modifications
- Light FCC Gasoline – Splitter Modifications
- Torrance Loading Rack (add fuel ethanol off-loading rack; modify vapor recovery unit, piping, and manifolds)
- Vernon Terminal (add rail car off-loading system, two truck off-loading areas, gasoline tank, lighting area and drainage system; modify rail spur, loading rack, vapor recovery unit, vapor destruction unit, and two storage tanks)
- Anaheim (Atwood) Terminal (add two truck off-loading areas, storage tank, lighting area and drainage system; modify truck rack)
- One new pentane sphere

Associated modifications and additions to storage facilities, pipelines and support facilities are also expected (SCAQMD, 2001a and SCAQMD 2003c). The Torrance refinery and loading rack, and the Vernon and Anaheim distribution terminals are located at least 10-15 miles from the Paramount Refinery so cumulative localized impacts are not expected to occur.

3) Shell

The Shell refinery (formerly Equilon and Texaco) is located at 2101 East Pacific Coast Highway, Wilmington and is sixteen miles south of the Paramount refinery. Shell's Wilmington Terminal is located adjacent to the southwestern portion of its Refinery at 1926 East Pacific Coast Highway, and the marine terminal is located on Mormon Island at Berths 167-169 within the Port of Los Angeles. The proposed project will also require changes to Shell's other southern California area distribution terminals located in Signal Hill, Carson, Van Nuys, and Colton/Rialto. The RFG Phase 3 project includes the following proposed modifications:

- Alkylation Unit (Contactor and Settler, refrigeration unit, exchangers/pumps, and effluent treating vessels)

- C4 Isomerization Unit (vessels, exchangers, pumps, piping, stabilizer, gas scrubber, and drier)
- Hydrotreater Unit No. 2 (Olefins Saturation Reactor, pretreatment reactor, charge pumps, heat exchangers, trays, stripper reboiler, and control valves)
- Hydrotreater Unit No. 4 (diesel side stripper, feed steam preheater, and heat exchangers)
- Hydrotreater Unit No. 1
- Catalytic Reforming Unit No. 2 (sulfur guard reactor)
- Fractionator Changes (HCU Main Fractionator, FCCU Debutanizer, Feed Prep Tower, Depentanizer, Alky Deisobutanizer, Alky Debutanizer and C4 Isomerization Deisobutanizer, and HCU Depropanizer)
- Refinery Storage Tank modifications
- Storage Tanks (at Wilmington, Carson, Signal Hill, Van Nuys, and Colton/Rialto Terminals)
- Pentane Sphere
- No. 2 (debutanizer tower)
- Flare
- Vapor Recovery Systems
- Carson Terminal (includes storage tanks modifications and a new truck loading rack)
- Lomita Terminal (includes an ethanol railcar unloading facility)
- Signal Hill Terminal (includes storage tank and truck loading rack modifications)
- Colton/Rialto Terminal (includes storage tank and truck loading rack modifications)
- Van Nuys Terminal (includes storage tank and truck loading rack modifications)
- Marine Terminal (includes storage tank modifications)
- Wilmington Terminal (includes storage tank and truck loading rack modifications)

Associated modifications and additions to storage facilities, pipelines and support facilities also are expected (SCAQMD, 2001b and SCAQMD 2002). The Shell refinery is located 16 miles south of the Paramount refinery. The Shell terminal in Signal Hill, is located at least eight miles from the Paramount Refinery and the Van Nuys and Colton/Rialto Terminals are located over 30 miles from the Paramount refinery. Localized cumulative impacts are not anticipated for any of these facilities because of the distance from the Paramount refinery.

4) ChevronTexaco

The ChevronTexaco refinery (formerly Chevron) is located at 324 West El Segundo Boulevard in El Segundo, California, about 18 miles west of the Paramount refinery, which is a sufficient distance away to avoid cumulative localized impacts with the Paramount refinery. The ChevronTexaco refinery has proposed to make changes to the reconfiguration of the Refinery by modifying existing process operating units, constructing and installing new equipment, and providing additional ancillary facilities in order to produce the RFG Phase 3 reformulated gasolines (SCAQMD, 2001c). The proposed new refinery units include:

- Isomax Complex (distillation column, steam reboilers and overhead condensers)
- TAME Plant (steam reboilers and overhead condensers)

- Pentane Storage Sphere
- Pentane Sales (rail loading facilities and railcar storage area)
- TAME Unit (distillation column, reflux pumps, steam reboilers and overhead condensers)
- No. 1 Naphtha hydrotreater (under Option A: one furnace, compressors, exchangers, and pumps. Under Option B: compressors, exchangers, and pumps).
- FCCU Depropanizer
- FCCU Debutanizer
- FCCU Deethanizer (vessels, pumps and exchangers)
- FCCU Propylene Caustic Treating Facilities
- FCCU Butene Caustic Treating Facilities
- FCCU Amine Absorber
- FCCU Relief System (headers)
- FCCU Wet Gas Compressor Insterstage System Upgrades (two exchangers and one vessel)
- Alkylation Plant (two contactors and an acid settler)
- Cooling Tower
- Trim coolers for existing Distillation Columns
- Iso-octene Plant (pressure vessels, exchangers and pumps)
- Two floating roof gasoline component storage tanks

Modifications to existing refinery units are proposed for the following:

- TAME Unit (Depentanizer column)
- No. 1 Naphtha hydrotreater (under Option A: modify one furnace; under Option B: modify two furnaces)
- Deethanizer (column)
- Relief Systems (vapor recovery facilities and flare)
- Main air blower rotor replacement
- Wet Gas Compressor
- Rotor and Gearbox Upgrade
- Recommission Existing Out-of-Service Deisobutanizer
- Retraying Distillation Columns
- MTBE storage tank

The proposed project also includes modifications to the ChevronTexaco Montebello Terminal (storage tank and loading rack modifications and a new ethanol railcar unloading facility), the Van Nuys Terminal (storage tank and loading rack modifications), and the Huntington Beach Terminal (storage tank and loading rack modifications).

Due to the distance separating the ChevronTexaco refinery and terminals from the Paramount refinery, no cumulative impacts are expected during the construction or operation of the proposed project.

5) British Petroleum

The British Petroleum (BP) Refinery (formerly ARCO), located at 1801 E. Sepulveda Boulevard in Carson, is approximately eleven miles south of the Paramount refinery. The BP Carson terminal is located at 2149 E. Sepulveda Boulevard; the Marine Terminal 2 is located at 1300 Pier B Street within the Port of Long Beach. The proposed RFG Phase 3 project will also require changes to BP's other southern California area distribution terminals located in South Gate, Rialto, Long Beach and Signal Hill. The BP refinery has proposed to make changes to the Refinery by modifying existing process operating units, constructing and installing new equipment, and providing additional ancillary facilities in order to produce the RFG Phase 3 reformulated gasolines (SCAQMD, 2001d). The proposed new refinery units include:

- FCCU Gasoline Fractionation (Option #1) – rerun bottoms splitter (splitter tower, heat exchangers, etc.)

Modifications to existing refinery units are proposed for the following:

- Light Hydro Unit (modify heat exchangers; new exchangers, piping pumps and control systems)
- Isomerization Sieve (convert unit to hydrotreater; modifications to heat exchangers, piping and control systems; new reactor, exchangers, pumps and control systems)
- No. 3 Reformer Fractionator and Overhead Condenser (piping and control systems; new pumps)
- Gasoline Fractionation Area (retraying, piping and control systems)
- FCCU Gasoline Fractionation (Option #2) – convert gasoline fractionation area depentanizer to a FCCU bottoms splitter (retraying; new exchangers, flash drum, and product cooling)
- North hydrogen plant (new feed drum, pump and vaporizer)
- MTBE Unit (Option #1) – convert into ISO Octene Unit (modify heat exchangers, piping and control systems; new reactive, steam heater and heat exchangers)
- MTBE Unit (Option #2) – convert into Selective Hydrogenation Unit (modify stripper, reboiler, piping and control systems; new heat exchangers)
- Cat Poly Unit – modify to a Dimerization Unit Hydrotreater reactor system (modify piping and control systems; new pumps, heat exchangers, vessels, piping and control systems)
- Mid-Barrel Unit – modify to a Gasoline Hydrotreater (modify feed and product piping, hydrogen supply system and heat exchanger, controls systems)
- Tank Farm – piping modifications
- Pentane railcar loading facility – modify for pentane off-loading (new repressurizing vaporizer system and two railcar spots)
- Propylene railcar loading facility – modify for butane off- loading.

Associated modifications and additions to distribution storage facilities, pipelines and support facilities also are expected (SCAQMD, 2001d). The BP Arco Refinery is located

about 11 miles from the Paramount Refinery, so cumulative localized impacts are not expected.

6) Ultramar Inc, Valero Refinery

The Ultramar refinery is located at 2042 East Anaheim Street in the Wilmington district of the City of Los Angeles. The Ultramar refinery is about 15 miles south of the Paramount Refinery. In order to produce the RFG Phase 3 project gasoline Ultramar has proposed both new and modified refinery units (SCAQMD, 2000c). The Ultramar's RFG Phase 3 project would include the following new refinery equipment:

- Merox Treater
- Sour Water Stripper – (storage tank, stripper and vapor recovery system)
- Storage Tanks
- Boiler
- Flare
- Cooling Tower

Modifications to the following refinery units were proposed:

- Fluid Catalytic Cracking Unit (FCCU) – (new Gas Concentration Unit Debutanizer, new primary absorber and stripper, new accumulators, pumps, reboiler, distillation columns, vessels and heat exchangers)
- Fluid Catalytic Cracking Unit Liquefied Gas Merox Unit – (new liquefied petroleum gas (LPG) dryer and Selective Hydrogenation Unit, convert existing dryer column to depropanizer)
- Light Ends Recovery Unit – (new debutanizer and depentanizer, convert existing depropanizer to recover butane in Butamer Unit; new vessels, pumps and fin-fans)
- Naphtha Hydrotreater Unit – (modify compressor, new heat exchangers and pumps)
- Olefin Treater – (convert to hydrotreater; new reactor, new stripper, new compressor, changes to piping and new catalyst)
- Gas Oil Hydrotreater – (new pumps, new compressors and modify heater)
- Platformer – (new compressor and depropanizer)
- Butamer Unit – (new column, new heat exchangers, vessels and pumps)
- Storage Tanks
- Flare System

Associated modifications and additions to storage facilities, pipelines and support facilities are also expected (SCAQMD, 2000c). The project also includes modification to existing storage tanks and new storage tanks at the Ultramar Marine Tank Farm, Olympic Tank Farm, and Marine Terminal. The Ultramar Refinery is located about 15 miles from the Paramount Refinery, so no localized cumulative impacts are expected.

7) Third Party Terminals

A number of petroleum companies use third party terminals to distribute their fuel to gasoline stations. The terminals include the Kinder Morgan Orange Terminal, and the Kinder Morgan Colton Terminal. The modifications to the Kinder Morgan Orange and Colton Terminals included the conversion of an existing fixed roof tank to an internal floating roof tank and a change in service of the tank from diesel to ethanol. In addition, new truck unloading racks were added to both the Orange and Colton Terminals.

C. OTHER NEARBY PROJECTS

Other proposed projects within the general vicinity of the Paramount Refinery are described below.

City of Long Beach

8) Street Construction

As part of the ongoing effort by the City of Long Beach to revitalize certain areas, a number of streetscape improvements have been proposed over the next three years. Streetscaping involves landscaping, widening of streets, sidewalk construction and repair, installation of lighting and signage, and construction of medians on streets. Several of these streetscaping activities are currently ongoing or will be conducted in the future within the vicinity of the Paramount Refinery, including the following:

- Atlantic Avenue to Artesia Blvd.
- Artesia Blvd. – Downey Ave. to Obispo Ave.
- Paramount Boulevard – 70th Street and Artesia Blvd.
- Downey Avenue – 70th Street and Artesia Blvd.

(Personal communication, Lee Mayfield, May 2003).

9) North Long Beach Redevelopment Project Area

North Long Beach covers an area of 7,540 acres of land. The majority of the land is within the Redevelopment project area and is located north of I-405 freeway. The area is bordered by the cities of Compton, Paramount and Lakewood. Many of the existing commercial properties in the area are in varying stages of physical deterioration and were built with substandard design and lack adequate parking.

The redevelopment of North Long Beach is already underway and is scheduled to be completed in approximately 2026. Part of the revitalization plan for the area includes converting declining commercial land uses to residential housing or other alternatives, and initiating streetscape improvements (Long Beach, City of, 2002).

City of Paramount

10) Industrial Warehousing Project

An industrial warehousing project located at the intersection of Garfield Avenue and Rosecrans Boulevard is projected to begin construction in approximately August 2004. This project will add 78,605 square feet of warehouse space and is scheduled to be completed within approximately six to eight weeks from commencement (Personal Communication, John Caver, May 2003 and November 2003).

11) Recreation Facility

The City of Paramount plans to build a new recreation center at Progress Park. Progress Park is located at 15500 Downey Ave. The 4,000-square-foot recreation center will replace a 1,400-square-foot preschool that was originally a house built in the 1940s. The new facility will be home to the City's preschool, the Park Pals after-school program, youth and adult recreation classes, the local girls softball league, as well as meetings and counseling sessions for GRIP (Gang Resistance in Paramount) and Neighborhood Watch. In addition, a plaza will be created and there will be extensive landscape and hardscape improvements to the park in the center's vicinity. Construction is scheduled to begin approximately, in April 2004. (Paramount, City of, Press Release, October 2002, Linda Benedetti-Leal and David Johnson, Paramount, City of, Recreation Department, November 2003).

City of Downey

12) Downey Landing

A mixed-use commercial and industrial complex is being proposed in the City of Downey which is located five miles north of the Paramount refinery. The site is bounded by Stewart and Gray roads on the north, Lakewood Boulevard and Clark Avenue on the west, Imperial Highway on the south, and Bellflower Boulevard on the east. The Downey Landing's proposal included multiple uses for 117 acres of the 160 acre site, including a 28-acre retail center that will occupy the northern portion, a movie/TV production studio complex for the central portion, and a business/technology park on the eastern portion. Kaiser Permanente plans a new hospital/medical office complex for 30 acres on the southern portion of the property. The proposed Kaiser Permanente project will include a six-story hospital and a four-story medical office building. The remaining 13 acres of the 160 acres will be reserved for a school/park/learning center.

The final Environmental Impact Report (EIR) (City of Downey, 2002) discusses the impact of the Specific Plan, and contains recommended mitigation measures designed to lessen the extent of identified impacts (City of Downery, 2002).

13) Banco Popular Project

The Banco Project is proposed for the northwest corner of the Rosecrans Avenue/Lakewood Boulevard intersection (13451 Lakewood Boulevard). The project site contains 15,577 square feet and; development will consist of one building containing a 1,200 square foot restaurant and a 2,013 square foot bank. A grading permit has been issued by the City of Downey for the project (Personal Communication Mark Selheim, May 2003).

14) 12651-65 Paramount Boulevard

A residential tract consisting of eight single-family residences is under construction at 12651-65 Paramount Boulevard (Personal Communication Mark Selheim, May 2003).

15) 12645 Lakewood Boulevard

A residential tract consisting of eight single-family residences is proposed for 12645 Lakewood Boulevard (Personal Communication Mark Selheim, May 2003).

City of Bellflower

16) 91 Freeway Ramp Beautification

Landscaping and decorative painting is being performed on the 91 Freeway on/off ramps at Bellflower Boulevard. (City of Bellflower, 2003).

17) Town Center Plaza Project

The Town Center Plaza project is part of the redevelopment plan to revitalize the downtown area of Bellflower. This project will span five acres and feature an outdoor stage, businesses and a train station that would connect to the Metrolink transit system. Environmental clearance is being sought for a two and one half mile bicycle path and walkway on what is currently a railroad track that is scheduled to be removed in the near future. This project is scheduled to begin construction approximately at the end of 2003. (City of Bellflower, 2003).

D. AIR QUALITY

CONSTRUCTION IMPACTS

Construction activities associated with CARB RFG Phase 3 projects at other refineries have or will be essentially completed prior to the commencement of construction activities at the Paramount Refinery. December 31, 2003 is the date when MTBE must be phased out of gasoline sold in California so most of the construction activities at other refineries and terminals have been or will be completed prior to construction of the

Paramount Clean Fuels project. No cumulative construction impacts are expected from other refinery projects.

Air quality impacts due to construction at the Paramount Refinery are considered to be less than significant. It is expected that construction activities associated with several other local projects will occur during the same timeframe as the proposed project including the Industrial Warehousing Project (No. 10), the Recreational Facility (No. 11), the Banco Popular Project (No. 13), and two residential developments (No. 14 and 15). Potential construction emissions have been estimated using the URBEMIS2002 Model. The default assumptions in the URBEMIS2002 Model (Yolo-Solano AQMD, 2003) were used since little information is available regarding these projects (see Appendix B for additional information).

TABLE 5-1

CUMULATIVE PROJECT
PEAK DAY CONSTRUCTION EMISSIONS[1]
(lbs/day)

ACTIVITY	CO	VOC	NOx	SOx	PM10
Paramount Clean Fuels Project	308	32	76	6	118
Industrial Warehouse Project (No. 10)	11	133	1	<1	<1
Recreational Center Project (No. 11)	1	<1	<1	<1	<1
Banco Popular Project (No. 13)	<1	5	<1	<1	<1
Residential Development (No. 14 and 15)	2	66	4	0	<1
Cumulative Emissions	322	236	81	6	118
SCAQMD Thresholds	550	75	100	150	150
Cumulatively Significant (?)	NO	YES	NO	NO	NO

Table 5-1 summarizes the construction emissions of the related projects (projects within approximately one mile of the Refinery) with construction schedules that might coincide with construction of the Paramount Clean Fuels Project. On a cumulative basis, construction emissions would exceed the CEQA thresholds established by the SCAQMD for VOC, assuming the construction projects occur at the same time. Therefore, the cumulative air quality construction impacts are considered significant for VOC emissions. The cumulative air quality construction impacts are less than significant for CO, NOx, SOx and PM10.

OPERATIONAL IMPACTS - CRITERIA POLLUTANTS

The RFG Phase 3 projects at all of the local refineries will increase the criteria pollutants emitted from the refineries. Direct stationary emission sources are generally subject to regulation. The emissions associated with the cumulative CARB Phase 3 projects are shown in Table 5-2. The operation of the CARB Phase 3 projects are expected to exceed

SCAQMD thresholds for CO, VOC, NOx, SOx and PM10, so air quality impacts are significant. No localized increases in air emissions are expected because the refineries and terminals are located a sufficient distances from the Paramount Refinery (see Figure 5-1).

Cumulative impacts associated with other local projects could also occur during the operational phase. Operational emissions from projects other than Paramount are expected to be largely due to mobile source emissions. The operational emissions have been estimated in Table 5-2.

TABLE 5-2

CUMULATIVE PROJECT
PEAK DAY OPERATIONAL EMISSIONS[1]
(Pounds per day)

SOURCE	CO	VOC	NOx	SOx	PM10
Ultramar CARB Phase 3 Project	514	156	2,164	2,678	287
ConocoPhillips Ethanol Import & Dist. Project	9	-54[1]	10	--	1
ConocoPhillips CARB RFG Phase 3	136	22	514	402	43
BP ARCO CARB Phase 3 Project	42	86	49	0	57
Shell CARB Phase 3 Project	2,213	482	2030	71	57
ExxonMobil CARB Phase 3 Project	29	288	138	12	103
ChevronTexaco CARB Phase 3 Project	393	347	3,103	2,498	843
Third Party Terminals	-	4	-	-	-
Paramount Clean Fuels Project	104	66	52	1	69
Industrial Warehouse Project (No. 10)[2]	76	7	10	<1	5
Recreational Center Project (No. 11)[2]	39	3	5	<1	3
Banco Popular Project (No. 13)[1]	109	9	14	<1	8
Residential Development (No. 14 and 15)[2]	80	25	5	<1	10
Cumulative Emissions	**3,744**	**1,441**	**8,094**	**5,662**	**1,486**
SCAQMD Thresholds	550	55	55	150	150
Significant (?)	YES	YES	YES	YES	YES

(1) Negative numbers represent emission reductions.
(2) Based on URBEMIS2002 Model, using default assumptions.

On a regional basis, RFG Phase 3 fuels produced by the refineries are expected to result in a reduction in emissions from mobile sources that utilize the reformulated fuels. Table 5-3 summarizes the expected statewide emission decreases from the mobile sources, which use the reformulated fuels. As a conservative approach, the statewide mobile source emissions reductions are not credited toward mitigation of cumulative impacts.

TABLE 5-3

CARB PHASE 3 EXPECTED STATEWIDE EMISSION CHANGES
(Pounds per Day)

POLLUTANT	1998 Average In-Use Fuel		Future Representative In-Use Fuel Based on Flat Limits		Difference
	2005	2010	2005	2010	2005
NOx	4,200	3,400	-33,200	-27,200	-37,400
Exhaust Hydrocarbons	-16.0 -32,000	-9.3 -18,600	-16.5 -33,000	-9.6 -19,200	-0.5 -1,000
Evaporative Hydrocarbons	-28,800	-22,600	-28,800	-22,600	0
Total Hydrocarbons	-60,800	-41,200	-61,800	-41,800	-1,000

Negative numbers indicate emission reductions. Source: CARB, 1999

Air quality impacts associated with operation of the six RFG Phase 3 projects are considered significant since SCAQMD mass emissions thresholds are expected to be exceeded. Although operations will exceed the significance thresholds, there will be large regional benefits from the use of the reformulated fuels by mobile sources. Emissions of mobile sources will be reduced for NOx and VOCs counteracting the emissions being produced by the refineries and providing an environmental benefit. The emission reductions are expected to be far greater than the direct cumulative emissions from the refineries. In addition, the RFG Phase 3 compliant fuels are expected to result in a 7.2 percent reduction in potency-weighted emissions of toxic air contaminants from mobile sources using the fuel providing additional emissions benefits. Further, the diesel sulfur limit of 15 ppmw will help generate significant air quality benefits by enabling the effective performance of advanced diesel exhaust emissions control technologies that reduce emissions of ozone precursors (NOx and VOCs) and diesel particulate matter.

The cumulative operational emissions associated with projects in the Paramount area are expected to exceed SCAQMD thresholds for CO, VOC, NOx, SOx and PM10. Therefore, cumulative air quality impacts are significant.

OPERATIONAL IMPACTS - TOXIC AIR CONTAMINANTS

In order to determine the cumulative impacts of toxic air contaminants, the emissions from the implementation of the proposed project were analyzed. This is referred to as the post-project scenario and includes all the existing emission sources at the Paramount Refinery, plus the proposed modified emission sources associated with the revised reformulated fuels program. In addition, the potential cumulative impacts associated with the overlap of emissions from other refineries were addressed in the analysis provided below. The other cumulative projects (Projects 8-17) are not expected to emit toxic air contaminants during operations and, therefore, were not included in this analysis.

A comprehensive air dispersion modeling analysis and a Health Risk Assessment (HRA) were performed for the projected refinery emissions following completion of the proposed project. This section discusses the results of the air dispersion modeling and health risk assessment. The procedures used to complete the projected HRA are the same as those used to complete the baseline HRA (see Chapter 3, Air Quality). The HRA is contained in Volume II, which should be consulted for further details.

Hazard Identification

The list of TACs evaluated in the post-project scenario is the same as those identified in the baseline assessment (see Table 3-6).

Emission Estimations and Sources

The estimated mass emissions of toxic air contaminants were based on a combination of the baseline emissions and engineering estimates that reflect operation of the proposed project. For further details on the emission estimates see Chapter 4, Air Quality and Volume II.

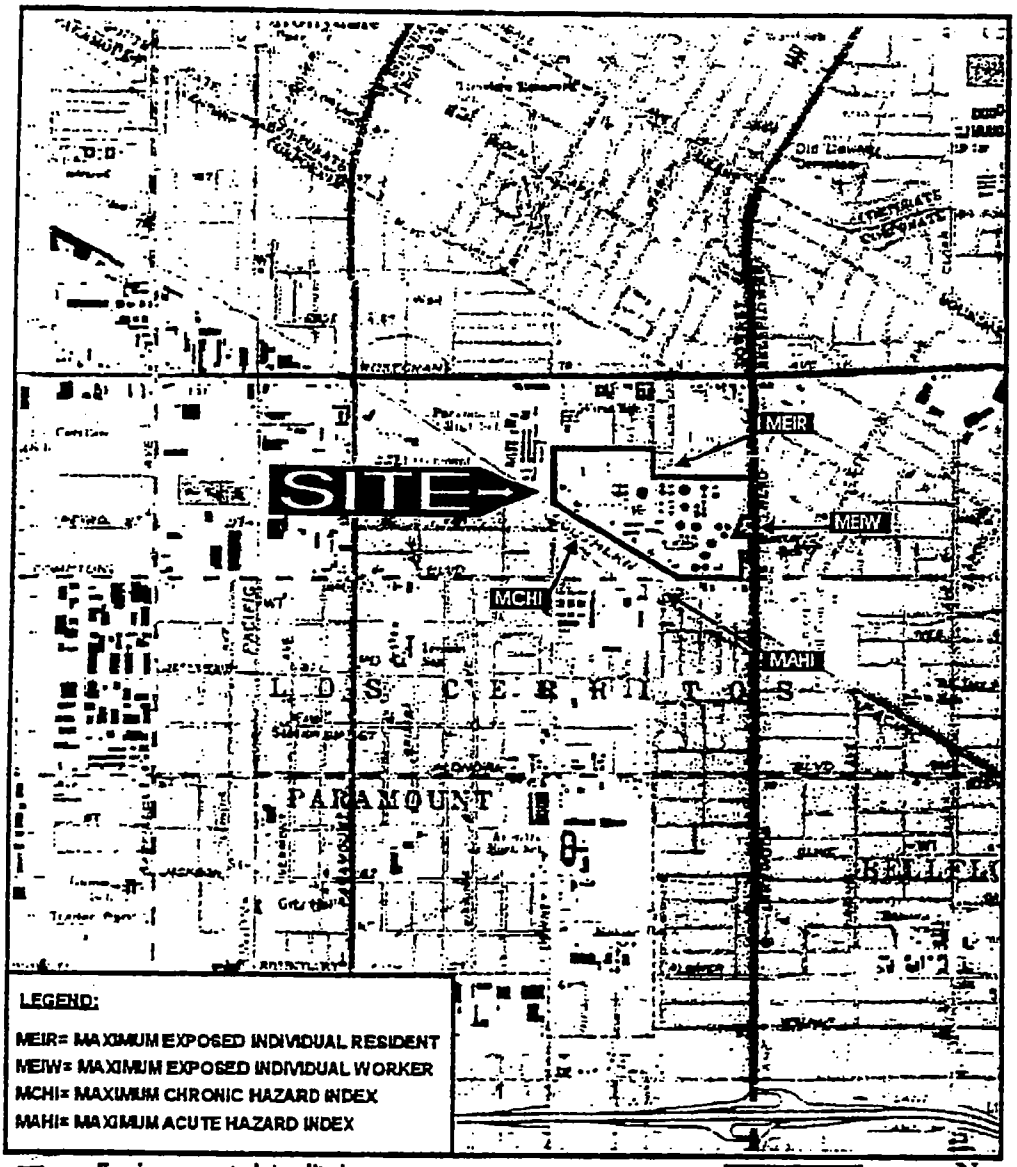
HRA Methodology

The source parameters for the post-project scenario were used as input to the ISCST3 model to determine unitized ground-level concentrations. The output from the ISCST3 model was combined with estimated emissions for each TAC in the ACE2588 model. The ACE2588 model calculated the health risks associated with the post-project scenario. The ISCST3 model used the same assumptions as the baseline model for receptor grids, meteorological data, and so forth. The ACE2588 model used the same assumptions for the post-project scenario as the baseline model for multi-pathway analysis, pathways to exposures, and default exposure assumptions. The model was used to identify the MEIW and MEIR for the post-project scenario. The ACE2588 model calculated both carcinogenic and non-carcinogenic health impacts.

Post-Project HRA Results - Carcinogenic Health Impacts

Maximum Exposed Individual Worker

The predicted maximum cancer risk at the MEIW area due to exposure to projected post-project emissions was calculated to be 2.15E-06 or two per million. The location of the MEIW is the same as that for the baseline scenario and is shown in Figure 5-3. Table 5-4 shows major source contributions to the MEIW. Emissions from Fugitives – Northeast Tank Farms account for about 45 percent of the MEIW cancer risk. Emissions of benzene are responsible for about 75 percent of the MEIW risk (see Table 5-5). The cancer risk at the MEIW does not exceed the cancer risk significance threshold in Table 4-1 and is less than significant.



Environmental Audit, Inc.

PROJECT MAXIMUM IMPACT LOCATIONS
14700 Downey Avenue
Paramount, California

Figure 4-1

Project No. 2150

TABLE 5-4

EMISSION SOURCE CONTRIBUTION TO CANCER RISK FOR POST-PROJECT SCENARIO MEIW

Source No.	Source Name	Percent Contribution
100	Fugitives for Northeast Tank Farm	45.0
111	Heaters H303-306	9.2
130	Fugitives for HDS Units	6.3
89	Fugitives for Crude Unit 1	4.2
92	Fugitives for Jet Fuel Area	3.6
90	Fugitives for Crude Unit 2	3.6
101	Fugitives for Northwest Tank Farm	3.0
116	Fugitives for New BenSat/Isom Unit	2.9
114	COGEN	1.7
102	Fugitives for North-Central Tank Farm	1.1
41	Tank 12502	1.0
19	Flare	1.0

TABLE 5-5

TAC CONTRIBUTION TO CANCER RISK FOR POST-PROJECT SCENARIO MEIW

Toxic Air Contaminant	Cancer Risk	Percent Contribution
Acetaldehyde	4.42E-10	<0.1
Arsenic	1.22E-08	0.6
Benzene	1.58E-06	74.8
1,3-Butadiene	3.81E-09	0.2
Cadmium	1.65E-08	0.8
Carbon Tetrachloride	2.14E-12	<0.1
Chloroform	2.10E-13	<0.1
Chromium (Hex)	1.16E-07	5.5
Ethylene Dibromide	4.36E-12	<0.1
Ethylene Dichloride	7.22E-13	<0.1
Formaldehyde	1.20E-08	0.6
Lead	2.66E-11	<0.1
Methylene Chloride	2.77E-14	<0.1
Nickel	8.75E-10	<0.1
Perchloroethylene	1.26E-09	0.4
PAHs	3.61E-07	17.10
Propylene Oxide	2.03E-16	<0.1
Styrene	4.20E-13	<0.1
Vinyl Chloride	1.61E-12	<0.1
Total	**2.10E-06**	

Maximum Exposed Individual Resident

The predicted maximum cancer risk at the MEIR area due to exposure to projected post-project emissions was calculated to be 9.81E-06 or about ten per million. The location of the MEIR is east of the Refinery and is shown in Figure 5-3. Table 5-6 shows major source contributions to the MEIR. Emissions from Fugitives - HDS Unit account for about 21 percent of the MEIR risk (see Table 5-6). Emissions of benzene are responsible for about 60 percent of the MEIR risk (see Table 5-7).

TABLE 5-6

EMISSION SOURCE CONTRIBUTION TO CANCER RISK FOR POST-PROJECT SCENARIO MEIR

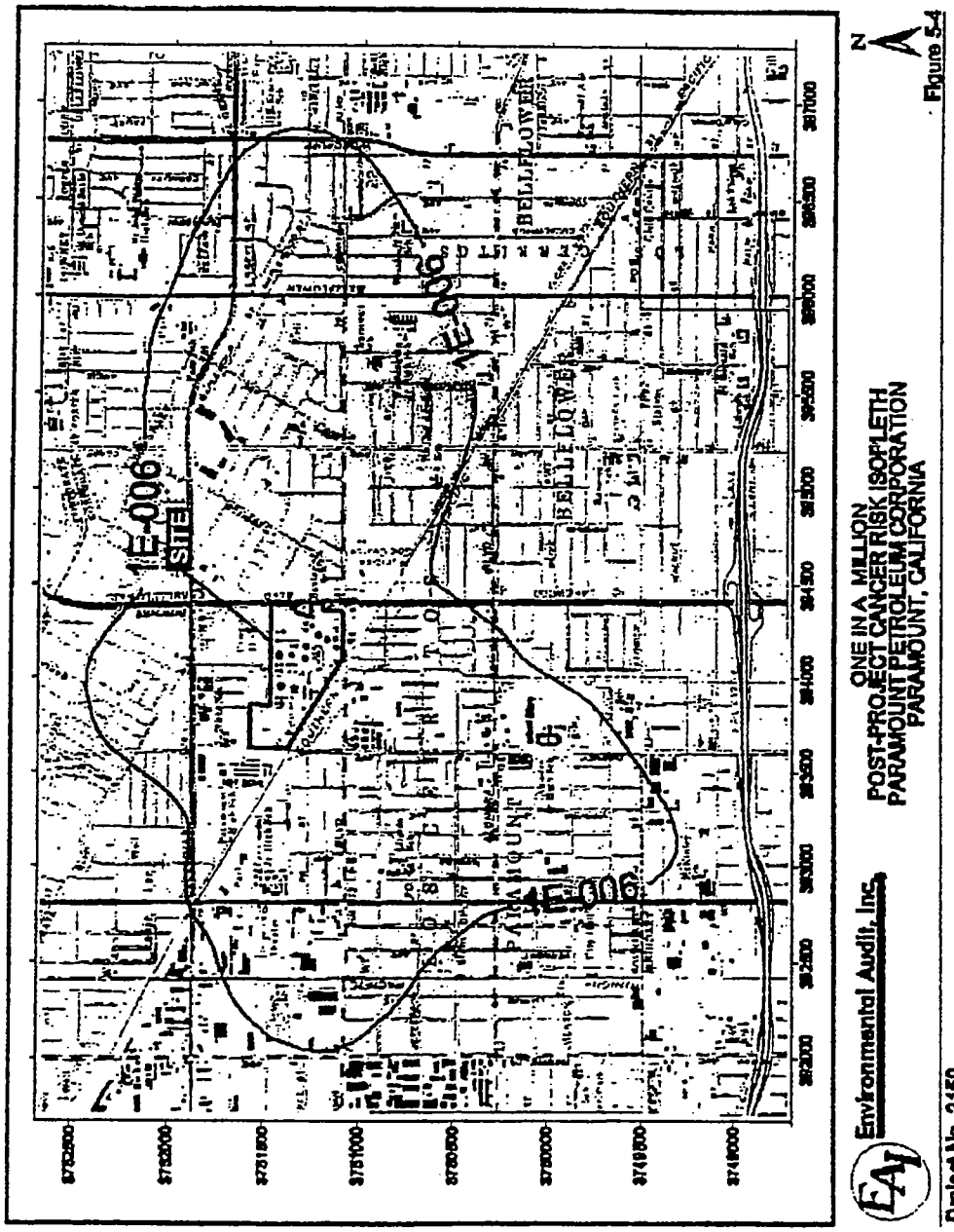
Source No.	Source Name	Percent Contribution
100	Fugitives for Northeast Tank Farm	21.0
89	Fugitives for Crude Unit 1	11.1
111	Heaters H303-306	10.0
90	Fugitives for Crude Unit 2	7.9
130	Fugitives for HDS Units	5.7
92	Fugitives for Jet Fuel Area	4.6
114	COGEN	2.9
101	Fugitives for Northwest Tank Farm	2.8
5	Heater H-601	2.6
116	Fugitives for New BenSat/Isom Unit	2.3
1	Heater H-801	2.2
2	Heater H-802	2.1
4	Heater H-860	1.6
6	Heater H-602	1.4
104	Fugitives for North-Central Tank Farm	1.3
112	Heater H501	1.2
19	Flare	1.2
18	Heater H-907	1.1

TABLE 5-7

TAC CONTRIBUTION TO CANCER RISK FOR POST-PROJECT SCENARIO MEIR

Toxic Air Contaminant	Cancer Risk	Percent Contribution
Acetaldehyde	3.35E-09	<0.1
Arsenic	8.92E-08	0.9
Benzene	5.88E-06	59.9
1,3-Butadiene	2.89E-08	0.3
Cadmium	1.20E-07	1.2
Carbon Tetrachloride	1.62E-11	<0.1
Chloroform	1.59E-12	<0.1
Chromium (Hex)	8.50E-07	8.7
Ethylene Dibromide	3.30E-11	<0.1
Ethylene Dichloride	5.46E-12	<0.1
Formaldehyde	9.04E-08	0.9
Lead	2.01E-10	<0.1
Methylene Chloride	2.10E-13	<0.1
Nickel	6.41E-09	0.1
Perechloroethylene	4.63E-08	0.5
PAHs	2.70E-06	27.5
Propylene Oxide	1.59E-15	<0.1
Styrene	3.18E-12	<0.1
Vinyl Chloride	1.22E-11	<0.1
Total	9.81E-06	

The one per million-cancer risk isopleth for the post-project scenario is shown in Figure 5-3. This isopleth was calculated based on the same assumptions used to calculate the residential cancer risk including a 70-year exposure and multi-pathway assumption. The cancer risk at the MEIR does not exceed the cancer risk significance threshold in Table 4-1 of ten per million and is less than significant. The post project cancer risk is reduced as a result of the project. The reduction is due to the reduced benzene content in products and process streams in order to meet CARB Phase 3 requirements, and the overall reduction of benzene at the facility by the addition of the benzene saturation and isomerization unit, which converts benzene to less toxic components.


SITE
E-006
E-006
E-006
BELLFLOWER
PARAMOUNT
ONE IN A MILLION
POST-PROJECT CANCER RISK ISOPLETH
PARAMOUNT PETROLEUM CORPORATION
PARAMOUNT, CALIFORNIA
Environmental Audit, Inc.
Figure 5-4
Project No. 2150

Cancer Burden

The cancer burden for the area surrounding the Paramount Refinery was calculated using the same assumptions as the baseline cancer burden calculations. The total excess cancer burden within the area of influence was predicted to be 0.122 and 0.0054 for the residential and occupational populations, respectively. (See Volume II for further details.) The combined excess cancer risk was predicted to be 0.127. The cancer burden does not exceed the cancer risk significance threshold in Table 4-1 and is less than significant.

Sensitive Receptors

The maximum cancer risk to a sensitive receptor was estimated to be 7.64E-06 or approximately eight per million at the Baxter Elementary School. This risk estimate is conservative as it is based on a 70-year continuous exposure period. The cancer risk at the sensitive receptors does not exceed the cancer risk significance threshold in Table 4-1 and is less than significant.

Post-Project HRA Results - Non-Carcinogenic Health Impacts

Acute Hazard Index

The highest total acute hazard index for any single toxicological endpoint was estimated to be 0.014, at an occupational receptor, for the respiratory system, primarily due to exposure to hydrogen sulfide (44 percent). The acute hazard index does not exceed the significance threshold in Table 4-1 and is less than significant.

Chronic Hazard Index

The highest chronic hazard index for any single toxicological endpoint was estimated to be 0.031, at an occupational receptor, for the respiratory system, primarily due to exposure to benzene (39 percent) and formaldehyde (23 percent). The chronic hazard index does not exceed the significance threshold in Table 4-1 and is less than significant.

The cumulative impacts associated with the post-project scenario would be below the significance criteria for cancer risk at the MEIW and the MEIR for the chronic and acute hazard indices. Further, the proposed project would reduce emissions of some toxic air contaminants, e.g. benzene, thus reducing the overall health risks associated with exposure to Refinery emissions. Therefore, adverse cumulative impacts associated with toxic air contaminants are not expected from the Paramount Clean Fuels Project.

TAC Impacts from Other Cumulative Projects

Based on the available data, the cumulative impacts associated with other proposed Clean Fuels projects (Project Nos. 1 through 7) are not expected to result in significant TAC impacts since the projects are disbursed throughout the southern California area so TAC emissions would not be expected to overlap. The other cumulative projects (Project Nos. 8 through 17) are not expected to generate significant quantities of toxic air contaminants.

MITIGATION MEASURES

Mitigation measures for construction activities have been imposed on the various individual projects. There are no additional feasible mitigation measures to further control construction emissions.

The mitigation measures to minimize emissions associated with operation of the related projects include the use of BACT for all new emission sources and modifications to existing sources. The use of BACT would control localized emissions. A BACT review will be completed during the SCAQMD permit approval process for all new/modified sources. In addition, the related refinery projects would provide regional emission benefits by reducing emissions from mobile sources that use the reformulated fuels.

LEVEL OF SIGNIFICANCE AFTER MITIGATION

The cumulative air quality impacts due to construction and operation of the RFG Phase 3 projects exceed the SCAQMD significance thresholds in spite of implementing all feasible mitigation measures. The cumulative impacts of TACs for cancer risk at the MEIR as less than significant. The cumulative impacts associated with the post-project scenario would be below the significance criteria for cancer risk at the MEIW, MEIR, and for the chronic and acute hazard index.

E. HAZARDS AND HAZARDOUS MATERIALS

PROJECT IMPACTS

The cumulative impacts from and between the onsite operation of the refineries' RFG Phase 3 projects (Project Nos. 1-7) are not expected to be significant because of the distance between Paramount and the other facilities. The closest refinery with a clean fuels project to the Paramount Refinery is the BP ARCO Refinery located about 11 miles south of the Paramount Refinery. The impacts associated with the Paramount Refinery proposed project are expected to travel less than 1,000 feet, which would not reach the other local refineries or any of the other cumulative projects. Projects Nos. 8 through 17 are not expected to involve hazardous materials or generate significant hazard impacts. Therefore, no significant cumulative hazard impacts are expected with the other related projects.

MITIGATION MEASURES

The proposed project impacts on hazards are considered significant. However, these impacts will not combine with the impacts of related projects due to the distance between the facilities. A number of existing rules and regulations apply to the Paramount Refinery and other proposed projects. Compliance with these rules and regulations is expected to minimize refinery-related hazards. Compliance with these rules and regulations should also minimize the hazards at other refineries.

LEVEL OF SIGNIFICANCE AFTER MITIGATION

The impacts of the various projects on hazards are not expected to be cumulatively considerable as hazards at or within one project area are not expected to impact or lead to hazards at other facilities or to combine in the same location.

F. TRANSPORTATION/TRAFFIC

For the proposed project, the project's contribution to cumulative transportation/traffic impacts is not significant because the traffic conditions would essentially be the same whether or not the proposed project is implemented, because the proposed project has such minimal effects on traffic conditions as explained below.

Cumulative traffic impacts have been analyzed using the traffic counts taken in 2003 and assuming general growth in the area. Table 5-8 shows the baseline and the cumulative LOS analysis and volume to capacity ratios due to general growth in the area. These ratios were calculated assuming a projected traffic growth of one percent per year and no changes in existing intersection geometrics. Cumulative impacts are not expected to result in significant changes in LOS.

The cumulative traffic analysis for the morning peak hour indicates that there would be no change in the LOS for all but one intersection in the Paramount area. The Lakewood Blvd./Somerset Blvd. intersection is expected to change from LOS A to B, which is not considered significant since traffic flow would not be significantly adversely impacted. Therefore, cumulative impacts on traffic during the morning are less than significant.

The cumulative traffic analysis for the evening peak hour indicates that there would be no change in the LOS for all but one intersection in the Paramount area. The Downey Avenue/Alondra Boulevard intersection is expect to change from LOS C to D. LOS D typically is the level for which a metropolitan area street system is designed. The growth in traffic is less than two percent of the overall traffic at the intersection and is considered less than significant. Therefore, cumulative impacts on traffic during the p.m. operations are less than significant.

On-and-Off Ramp Freeway Traffic During Operations

Two freeways bordering the proposed project were analyzed for traffic impacts during operations. The Century Freeway (I-105) is located approximately six miles north of the proposed project and the Artesia Freeway (SR-91) is approximately 14 miles south. The cumulative traffic analysis included the intersections of Downey Avenue and SR-91, Lakewood Boulevard and SR-91, both of which are south of the Paramount Refinery, and the intersection of Lakewood Boulevard and the I-105, which is north of the Refinery. The analysis indicates that the LOS at these intersections is not expected to change. Therefore, the cumulative impacts at these intersections are expected to be less than significant.

TABLE 5-8

CUMULATIVE TRAFFIC IMPACTS
LEVEL OF SERVICE ANALYSIS AND VOLUME-TO-CAPACITY RATIOS
OPERATIONAL

	BASELINE				CUMULATIVE IMPACTS					
	AM PEAK		PM PEAK		AM PEAK			PM PEAK		
INTERSECTION	LOS	Volume to Capacity Ratio	LOS	Volume to Capacity Ratio	LOS	Volume to Capacity Ratio	Volume to Capacity Ratio Increase	LOS	Volume to Capacity Ratio	Volume to Capacity Ratio Increase
Downey Ave. & Rosecrans Ave.	B	0.662	C	0.761	B	0.674	0.000	C	0.777	0.003
Downey Ave. & Somerset Blvd.	D	0.854	B	0.687	D	0.871	0.001	B	0.701	0.001
Downey Ave. & Alondra Blvd.	B	0.637	C	0.793	B	0.649	0.000	D	0.808	0.000
Downey Ave. & SR91 WB offramp/ SR91 WB on & EB offramps.	C	0.780	B	0.625	C	0.795	0.000	B	0.637	0.000
Downey Ave. & SR91 EB onramp/ SR91 EB offramp.	B	0.661	B	0.622	B	0.673	0.000	B	0.633	0.000
Lakewood Blvd. & I105 EB offramp/ I105 WB offramp.	A	0.560	C	0.749	A	0.573	0.000	C	0.766	0.001
Lakewood Blvd. & Rosecrans Ave.	A	0.562	C	0.745	A	0.577	0.000	C	0.764	0.000
Lakewood Blvd. & Somerset Blvd.	A	0.598	B	0.671	B	0.621	0.000	B	0.685	0.000
Lakewood Blvd.& Alondra Blvd.	A	0.540	C	0.750	A	0.551	0.000	C	0.765	0.000
Lakewood Blvd & SR91 WB on/off ramps SR91 WB onramp	A	0.418	A	0.586	A	0.427	0.000	A	0.598	0.000
Lakewood Blvd & SR91 EB onramp SR91 EB on/off ramps.	A	0.520	B	0.691	A	0 529	0.000	B	0.704	0.000

LEVEL OF SIGNIFICANCE

All intersections near the Paramount Refinery are considered to have less than significant cumulative impacts, since free-flowing traffic would continue and is not expected to change. Therefore, the cumulative impacts on traffic during the a.m and p.m. would be considered less than significant.

MITIGATION MEASURES

No significant cumulative impacts have been identified so no mitigation measures are required.

LEVEL OF SIGNIFICANCE AFTER MITIGATION

The traffic impacts associated with the proposed project and other related projects are not expected to be significant or result in adverse traffic impacts that would contribute to the cumulative traffic impacts.

DA:WORD:2150ER5

CITY OF LONG BEACH

DEPARTMENT OF PLANNING AND BUILDING

333 West Ocean Boulevard, 5th Floor Long Beach, CA 90802 FAX (562) 570-6610

COMMUNITY & ENVIRONMENTAL PLANNING

April 20, 2005

Elizabeth Campbell
4803 Lorelei Avenue
Long Beach, CA 90808

Dear Ms. Campbell:

The City is in receipt of your letter dated April 15, 2005. In conformance with the California Environmental Quality Act (CEQA), the comments received to the Draft Environmental Impact Report (DEIR) for the Long Beach Memorial Medical Center Expansion project will be made available to the public, along with the response to comments, ten (10) days prior to the scheduled public hearing.

The Long Beach Planning Commission is scheduled to review the Final Environmental Impact Report (FEIR) at the May 5, 2005 hearing. The hearing is scheduled at 1:30 p.m. in the City Hall Council Chambers. The FEIR will be available for a ten-day review period beginning April 25, 2005. A copy of the FEIR will be sent to all parties that submitted comments on the Draft EIR, and will also be available at the Long Beach Central Library, Burnett Branch Library and Dana Branch Library, as well at City Hall, Department of Planning & Building, 7th Floor reception desk and Sapphos Environmental, Inc., 133 Martin Alley, Pasadena, Ca. The City will also make the Final EIR available for public review on the City of Long Beach Web site at: www.longbeach.gov/plan/pb/epd/er.asp

If you have any questions, please contact me at (562) 570-6193.

Sincerely,

Anita Garcia

Anita Garcia
Project Manager

CO NTY OF LOS ANGELES
FIRE DEPARTMENT
1320 NORTH EASTERN AVENUE
LOS ANGELES, CALIFORNIA 90063-3294
(323) 890-4330

P. MICHAEL FREEMAN
FIRE CHIEF
FORESTER & FIRE WARDEN

April 6, 2005

Ms. Anita Garcia
Department of Planning and Building
City of Long Beach
City Hall, 5th Floor
333 West Ocean Blvd.
Long Beach, CA 90802

Dear Ms. Garcia:

DRAFT ENVIRONMENTAL IMPACT REPORT FOR THE LONG BEACH MEMORIAL CENTER EXPANSION PROJECT, "CITY OF LONG BEACH" (EIR #2225/2005)

The Draft Environmental Impact Report for the aforementioned proposed project has been reviewed by the Planning Division, Land Development Unit, and Forestry Division of the County of Los Angeles Fire Department. The following are their responses:

LAND DEVELOPMENT UNIT:

This project is located entirely in the City of Long Beach. Therefore, the City of Long Beach Fire Department has jurisdiction concerning this project and will be setting conditions.

This project is located in close proximity to the jurisdictional area of the County of Los Angeles Fire Department. However, this project is unlikely to have an impact that necessitates a comment concerning general requirements from the Land Development Unit of the County of Los Angeles Fire Department.

Should any questions arise please contact the Land Development Unit, EIR Specialist at (323) 890-4243.

SERVING THE UNINCORPORATED AREAS OF LOS ANGELES COUNTY AND THE CITIES OF:

AGOURA HILLS	BRADBURY	CUDAHY	HAWTHORNE	LA MIRADA	MALIBU	POMONA	SIGNAL HILL
ARTESIA	CALABASAS	DIAMOND BAR	HIDDEN HILLS	LA PUENTE	MAYWOOD	RANCHO PALOS VERDES	SOUTH EL MONTE
AZUSA	CARSON	DUARTE	HUNTINGTON PARK	LAKEWOOD	NORWALK	ROLLING HILLS	SOUTH GATE
BALDWIN PARK	CERRITOS	EL MONTE	INDUSTRY	LANCASTER	PALMDALE	ROLLING HILLS ESTATES	TEMPLE CITY
BELL	CLAREMONT	GARDENA	INGLEWOOD	LAWNDALE	PALOS VERDES ESTATES	ROSEMEAD	WALNUT
BELL GARDENS	COMMERCE	GLENDORA	IRWINDALE	LOMITA	PARAMOUNT	SAN DIMAS	WEST HOLLYWOOD
BELLFLOWER	COVINA	HAWAIIAN GARDENS	LA CANADA FLINTRIDGE	LYNWOOD	PICO RIVERA	SANTA CLARITA	WESTLAKE VILLAGE
							WHITTIER

FORESTRY DIVISION:
The statutory responsibilities of the County of Los Angeles Fire Department, Forestry Division include erosion control, watershed management, rare and endangered species, vegetation, fuel modification for Very High Fire Hazard Severity Zones or Fire Zone 4, archeological and cultural resources, and the County Oak Tree Ordinance. The areas germane to these statutory responsibilities have been addressed.

If you have any additional questions, please contact this office at (323) 890-4330.

Very truly yours,

DAVID R. LEININGER, CHIEF, FORESTRY DIVISION
PREVENTION BUREAU

DRL:lc

LAURA RICHARDSON

CITY OF LONG BEACH
COUNCILWOMAN - SIXTH DISTRICT

City Hall: (562) 570-6816
District Office: 570-4420
FAX: 570-7135
TDD: 570-6629

May 5, 2005

Long Beach City Planning Commission
333 W. Ocean Blvd.
Long Beach, CA 90802

Good afternoon.

I am writing this letter today to be read into the record regarding Agenda Item 2: Long Beach Memorial Medical Center

As Councilwoman of the Sixth District, both Memorial Hospital and Miller Children's Hospitals are in my District. Over the last year, I have participated in many discussions regarding the proposed expansion being considered today.

Given the recent hospital closures throughout our region and the state, I strongly support efforts to provide quality health care for our growing Long Beach population. However, it has been brought to my attention that a few issues need to be addressed that I fully support resolution of.

First, regarding public participation. I believe that it is necessary to have one more meeting so all residents can have the opportunity to participate and further it is imperative that notice for this meeting be sent in tri-lingual format (English/Spanish/Khmer). It is my understanding that bilingual notices were not sent to the public and in such a diverse city with potential impacts on minority communities it is important that information be provided in all languages. Further, it will also be necessary that translation be available at that meeting. I am willing in conjunction with city and hospital staff to organize this meeting, which should take place prior to this item coming before the City Council in June.

Secondly, I understand that there is a concern for the 50 plus residents who reside in the residential building considered part of the expansion proposal. Prior to the above-motioned community meeting, I would like the following:

1. Verification of Memorial Hospital's ownership of that building;
2. Clarification of city limitations on addressing private property issues;
3. Long Beach Housing Corporation's determination of whether or not this property is deemed affordable housing;

Civic Center Plaza, 14th Floor, 333 West Ocean Boulevard, Long Beach, CA 90802
District Office, 1133 Rhea Street, Long Beach, CA 90806
(562) 570-6816 – City Hall (562) 570-7135 – FAX
Email Address:district6@longbeach.gov

4. If the property is deemed affordable housing, Memorial/Miller would agree to the relocation benefits normally applied in such circumstances; and
5. The City would commit to working with the residents over the next 5-10 years t make sure that residents are aware of affordable housing units in the City as they become available.

Thirdly, regarding the worker and labor access issues, I have spoken with Dr. Marks and SEIU and I am more than willing to participate in discussions to insure that quality work and future worker concerns are considered and addressed.

In conclusion, I completely support the staff recommendation to certify the Environmental Impact Report and I respectfully request your assistance in resolving these last remaining issues prior to City Council review so that we can obtain expedient support from the Council and begin commencement on this much needed project. Thank you for your time this afternoon.

Councilwoman Laura Richardson
Sixth District

CLR/TL

planningcommMemexp

LULAC of Greater Long Beach, Council 3088
One World Trade Center
P.O. Box 32364
Long Beach, CA 90832

May 5, 2005

City of Long Beach
Planning Commission
333 W. Ocean Blvd.
Long Beach, CA 90802

Dear Planning Commissioners:

The Long Beach League of United Latin American Citizens, Council 3088, supports the projected expansion of services to the community by Memorial Care Health Systems. The increase of services for children is good for the community. We only ask that certain actions be undertaken in order to minimize the impact of this expansion on the already overburdened neighborhoods surrounding the hospital.

On the top of our list is our concern that this project will destroy at least 50 units of affordable housing without replacing them. LULAC urges a reconsideration of this proposal. We believe that affordable housing should be the top priority of the Planning Commission and rather then destroy these units more should be built utilizing modern construction methods.

LULAC also urges the Commission to explore a covenant that requires the hospital to hire local residents on its construction project. We ask that the hospital hire local workers and that their pay and benefit standards adhere to industry guidelines.

LULAC requests that information and notifications surrounding this proposed expansion project be translated into languages that are spoken by the surrounding community.

LULAC requests that all information concerning this expansion project be released to the community and press, well in advance of

any action, proposed or otherwise, and that there be adequate time for public review and response. This includes the release of technical documents describing the project and any impacts, including the Environmental Impact Report.

LULAC strongly urges you to take the above actions, in the interest of the public good. We know that were Long Beach Memorial Hospital located in your neighborhood you would also be concerned about the impact of this expansion on your quality of life, notwithstanding the overall benefit the project would bring. Should you have any questions, please feel free to contact us at (562) 397-8118.

Sincerely Yours,

Thomas R. Gonzales

Thomas R. Gonzales
President, Great Long Beach
LULAC Council 3088

cc: Angel Luevano, Director, State LULAC
LULAC Council 3088 Board