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August 31, 2012

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RE: Comment on Draft Environmental Impact Report for ProLogis Eucalyptus Industrial Park (State Clearinghouse No. 2008021002)

Dear Mr. Bradshaw:

I am writing on behalf of Laborers International Union of North America, Local Union No. 1184 and its members living in Riverside County (collectively "LIUNA Local Union No. 1184" or "LIUNA" or "Commenters") regarding the Draft Environmental Impact Report ("DEIR") prepared for the ProLogis Eucalyptus Industrial Park, State Clearinghouse No. 2008021002 ("Project").

We have reviewed the DEIR with the assistance of:

1. Atmospheric Scientist, Dr. James Clark, Ph.D.
2. Hydrogeologist, Matthew Hagemann, C.Hg., MS.

These experts have prepared written comments that are attached hereto, and which are incorporated in their entirety. The City of Moreno Valley ("City") should respond to the expert comments separately. These experts and our own independent review demonstrate that the DEIR is woefully inadequate and that a new supplemental EIR is required to be prepared and recirculated for public comment. In particular, the EIR suffers from the following significant errors and omissions, among others:

- SEGMENTATION OF PROJECT: The DEIR improperly segments the Project by failing to include the infrastructure (e.g., roads, water, and sewer) as part of the Project.

- LOSS OF FARMLAND: The DEIR acknowledges that the Project's conversion of Prime Farmland is a significant impact, but the DEIR fails to adequately mitigate for the loss of farmland. The conclusion that mitigation measures are infeasible is unsupported.

- HAZARDOUS MATERIALS: The baseline of the physical environmental conditions in the vicinity of the Project is erroneous because the DEIR does not provide any details on the types of pesticides used on the Project site, relies on two outdated Phase I Environmental Site Assessments ("ESAs") that do not cover the entire Project site, and fails to disclose the status of an underground storage tank.

- GREENHOUSE GAS: The DEIR fails to provide support for the conclusion that greenhouse gas emissions after mitigation will be less than significant.

- AIR QUALITY: The DEIR fails to adequately analyze impacts to air quality because: (1) the DEIR underestimates the potential particulate emissions for the construction phase of the Project, (2) fails to accurately compare construction emissions to daily construction significance thresholds, (3) fails to consider health risks from contaminated dust, (4) fails to properly identify and address the Project's operational air quality impacts, (5) fails to disclose impacts to offsite receptors, and (6) fails to adequately analyze cumulative impacts.

Commenters urge the City to revise the EIR to adequately describe, analyze, and mitigate the Project and its impacts.[1] The revised EIR should be recirculated to allow public review and comment.

I. PROJECT DESCRIPTION

The Project site encompasses 122.8 acres of land located within the City of Moreno Valley, south of and adjacent to SR-60, east of Moreno Valley Auto Mall, and adjacent to and west of the Quincy Channel in Riverside County. (DEIR, p. 3-1). Single-family residential uses are located approximately 50 feet southeast of the southeastern corner of the Project site. (DEIR, p. 3-1). The Assessor's Parcel Numbers ("APNs") for this site are 488-330-011, 488-330-012, 488-330-013, 488-330-017, 488-330-018, 488-330-019, 488-330-022, 488-330-023, 488-330-024, and 488-330-025. (DEIR, p. 3-1).

[1] We reserve the right to supplement these comments at later hearings and proceedings for this Project. See, *Galante Vineyards v. Monterey Water Dist.* (1997) 60 Cal. App. 4th 1109.

The Project would include the construction of a warehouse facility comprising six buildings consisting of a total of approximately 2,244,638 square feet. (DEIR, p. 3-2). The Project site is divided into 2 areas: (1) the northern area (north of future Eucalyptus Avenue) would contain approximately 1,030,377 square feet of warehouse uses divided between two buildings and (2) the southern area (south of the future Eucalyptus Avenue) would consist of approximately 1,214,261 square feet of warehouse uses divided among four separate buildings. (DEIR, p. 3-2). The specific uses/users are not known at this time. (DEIR, p. 3-11).

The Project site currently consists of 57 acres used to grow grapefruit, 36 acres used for hay and alfalfa production, as well as portions that are vacant. (DEIR p. 4.2-1). Approximately 82.5 acres of the Project site is designated as Prime Farmland. (DEIR, p. 4.2-6).

The Project would require significant changes to the General Plan and local zoning ordinances including:

- General Plan Amendment. The proposed project includes an amendment to the Land Use Element to change the General Plan designations for a portion of the project site from Residential 15, Residential 5 and Residential 2 to Business Park. (DEIR, p. 1-2). The project also proposes an amendment to the Circulation Element by making changes to the alignment of Encilia Street and the removal of Quincy Street from within the project boundaries. (DEIR, p. 1-2).

- Change of Zone. The proposed project includes a change to the project site zoning from Business Park (BP), Business Park Mixed-use (BPX), Residential Agriculture 2 (RA2), Residential 5 (R5), and Residential 15 (R15) to Light Industrial (LI). (DEIR, p. 1-2).

- Municipal Code Amendment. The project includes a Municipal Code Amendment to establish a minimum clearance of 250 feet between adjacent residential zoning districts and any truck court or primary truck circulation driveway in lieu of the buffer established by the Business Park zone. (DEIR, p. 1-2).

II. Standing

Members of Local Union No. 1184 live, work, and recreate in the immediate vicinity of the Project site. These members will suffer the impacts of a poorly executed or inadequately mitigated Project, just as would the members of any nearby homeowners association, community group, or environmental group. Hundreds of LIUNA Local Union No. 1184 members live and work in areas that will be affected by traffic, air pollution, and water pollution generated by the Project.

In addition, construction workers will suffer many of the most significant impacts from the Project as currently proposed, such as from air pollution emissions from poorly maintained or controlled construction equipment, possible risks related to hazardous materials on the Project site, and other impacts. Therefore, LIUNA Local Union No. 1184 and its members have a direct interest in ensuring that the Project is adequately analyzed and that its environmental and public health impacts are mitigated to the fullest extent feasible.

III. LEGAL STANDARDS

A. EIR

CEQA requires that an agency analyze the potential environmental impacts of its proposed actions in an environmental impact report ("EIR") (except in certain limited circumstances). (See, e.g., Pub. Res. Code § 21100). The EIR is the very heart of CEQA. (*Dunn-Edwards v. BAAQMD* (1992) 9 Cal.App.4th 644, 652). "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." (*Communities for a Better Environment v. Calif. Resources Agency* (2002) 103 Cal. App. 4th 98, 109).

CEQA has two primary purposes. First, CEQA is designed to inform decision makers and the public about the potential, significant environmental effects of a project. (14 Cal. Code Regs. ("CEQA Guidelines") § 15002(a)(1)). "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.'" (*Citizens of Goleta Valley v. Board of Supervisors* (1990) 52 Cal. 3d 553, 564). 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." (*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).

Second, CEQA requires public agencies to avoid or reduce environmental damage when "feasible" by requiring "environmentally superior" alternatives and all feasible mitigation measures. (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). The EIR serves to provide agencies and the public with information about the environmental impacts of a proposed project and to "identify ways that environmental damage can be avoided or significantly reduced." (Guidelines §15002(a)(2)). If the project will have a significant effect on the environment, the agency may approve the project only if it finds 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 overriding concerns." (Pub.Res.Code § 21081; 14 Cal.Code Regs. § 15092(b)(2)(A) & (B)).

While the courts 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.'" (*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)). As the court stated in *Berkeley Jets*, 91 Cal. App. 4th at 1355:

> 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." (*San Joaquin Raptor/Wildlife Rescue Center v. County of Stanislaus* (1994) 27 Cal. App. 4th 713, 722; *Galante Vineyards v. Monterey Peninsula Water Management Dist*. (1997) 60 Cal. App. 4th 1109, 1117; *County of Amador v. El Dorado County Water Agency* (1999) 76 Cal. App. 4th 931, 946).

B. Supplemental EIR

Recirculation of an EIR prior to certification is required "when the new information added to an EIR discloses: (1) a new substantial environmental impact resulting from the project or from a new mitigation measure proposed to be implemented (cf. Guidelines, § 15162, subd. (a)(1), (3)(B)(1)); (2) a substantial increase in the severity of an environmental impact unless mitigation measures are adopted that reduce the impact to a level of insignificance (cf. Guidelines, § 15162, subd. (a)(3)(B)(2)); (3) a feasible project alternative or mitigation measure that clearly would lessen the environmental impacts of the project, but which the project's proponents decline to adopt (cf. Guidelines, § 15162, subd. (a)(3)(B)(3), (4)); or (4) that the draft EIR was so fundamentally and basically inadequate and conclusory in nature that public comment on the draft was in effect meaningless." *Laurel Heights Improvement Assn. v. Regents of University of California* (1993) 6 Cal. 4th 1112, 1130, citing *Mountain Lion Coalition v. Fish & Game Comm'n* (1989) 214 Cal.App.3d 1043.

> Significant new information requiring recirculation can include:
>
> (1) A new significant environmental impact would result from the project or from a new mitigation measure proposed to be implemented.
>
> (2) A substantial increase in the severity of an environmental impact would result unless mitigation measures are adopted that reduce the impact to a level of insignificance.

> (3) A feasible project alternative or mitigation measure considerably different from others previously analyzed would clearly lessen the significant environmental impacts of the project, but the project's proponents decline to adopt it.
>
> (4) The draft EIR was so fundamentally and basically inadequate and conclusory in nature that meaningful public review and comment were precluded.

(14 Cal. Code Regs. § 15088.5(a)).

The DEIR fails to analyze significant environmental impacts pertaining to the Project and to fully consider available mitigation measures to address those impacts. A revised EIR is required to be prepared and recirculated to address these deficiencies.

IV. THE DEIR IMPROPERLY SEGMENTS THE PROJECT

A. Legal Standard

The courts have repeatedly held that "an accurate, stable and finite project description is the *sine qua non* of an informative and legally sufficient [CEQA document]." *County of Inyo v. City of Los Angeles*, 71 Cal.App.3d 185, 193 (1977). Thus, CEQA mandates "that environmental considerations do not become submerged by chopping a large project into many little ones -- each with a minimal potential impact on the environment -- which cumulatively may have disastrous consequences." *Bozung v. LAFCO*, 13 Cal.3d 263, 283-84 (1975); *City of Santee v. County of San Diego*, 214 Cal.App.3d 1438, 1452 (1989). Before undertaking a project, the lead agency must assess the environmental impacts of all reasonably foreseeable phases of a project and a public agency may not segment a large project into two or more smaller projects in order to mask serious environmental consequences. As the Court of Appeal stated:

> The CEQA process is intended to be a careful examination, fully open to the public, of the environmental consequences of a given project, **covering the entire project, from start to finish**…the purpose of CEQA is not to generate paper, but to compel government at all levels to make decisions with environmental consequences in mind.

Natural Resources Defense Council v. City of Los Angeles, 103 Cal.App.4th 268 (2002) (emphasis added).

In *County of Amador v. City of Plymouth*, 149 Cal. App. 4th 1089, 1095 (2007) an Indian tribe intended to build a large gaming development comprised of a hotel, restaurants, and bars, on land located in or adjacent to the city. The Court held that the

construction of public works, including a city road to the casino hotel, constituted a project within the scope of CEQA. *Id*. at 1100. The Court cited to the CEQA Guideline § 15378(a)(1) which states that the following is included in the term "project": "public works construction and related activities, clearing or grading of land [and] improvements to existing public structures…" *Id*. at 1100.

B. The DEIR Improperly Segments the Project By Failing to Include the Infrastructure as Part of the Project

The DEIR states:

> If the proposed project is constructed prior to the West Ridge project, ProLogis will install the infrastructure necessary to serve its project (e.g., roads, water, and sewer) and will be reimbursed by the City from the West Ridge developer at the time that project is constructed. If the West Ridge project is constructed first, ProLogis will contribute an appropriate amount to the City for a reimbursement account to help off-site improvement costs installed by the West Ridge project that serve the ProLogis project. The timing of improvements shall be coordinated by the City in cooperation with ProLogis and the West Ridge.

(DEIR, p. 3-11). Instead of including the roads, water, and sewer lines required to serve the ProLogis Project as part of the Project, the DEIR treats these infrastructure improvements as a separate project included in the cumulative projects list provided in Table 3.C: Cumulative Projects. (DEIR, p. 3-16). The City is improperly chopping the ProLogis Project into different segments, which is prohibited by CEQA because proper analysis of the whole project is thwarted. Like the casino road in *County of Amador v. City of Plymouth*, the roads, water, and sewer lines that will serve the ProLogis Project must be included as part of the Project and properly analyzed as part of the whole Project.

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

An EIR must disclose all potentially significant adverse environmental impacts of a project. (Pub. Res. Code § 21100(b)(1); 14 Cal.Code Regs. § 15126(a); *Berkeley Jets*, 91 Cal. App. 4th 1344, 1354). CEQA requires that an EIR must not only identify the impacts, but must also provide "information about how adverse the impacts will be." (*Santiago County Water Dist. v. County of Orange* (1981) 118 Cal.App.3d 818, 831). The lead agency may deem a particular impact to be insignificant only if it produces rigorous analysis and concrete substantial evidence justifying the finding. (*Kings County Farm Bureau v. City of Hanford* (1990) 221 Cal.App.3d 692).

CEQA requires public agencies to avoid or reduce environmental damage when "feasible" by requiring mitigation measures. (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). The EIR serves to provide agencies and the public with information about the environmental impacts of a proposed project and to "identify ways that environmental damage can be avoided or significantly reduced." (Guidelines §15002(a)(2)). If the project will have a significant effect on the environment, the agency may approve the project only if it finds 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 overriding concerns." (Pub.Res.Code § 21081; 14 Cal.Code Regs. § 15092(b)(2)(A) & (B)).

In general, mitigation measures must be designed to minimize, reduce, or avoid an identified environmental impact or to rectify or compensate for that impact. (CEQA Guidelines § 15370). Where several mitigation measures are available to mitigate an impact, each should be discussed and the basis for selecting a particular measure should be identified. (*Id.* at § 15126.4(a)(1)(B)). A lead agency may not make the required CEQA findings unless the administrative record clearly shows that all uncertainties regarding the mitigation of significant environmental impacts have been resolved.

CEQA requires the lead agency to adopt feasible mitigation measures that will substantially lessen or avoid the Project's potentially significant environmental impacts (Pub. Res. Code §§ 21002, 21081(a)), and describe those mitigation measures in the CEQA document. (Pub. Res. Code § 21100(b)(3); CEQA Guidelines § 15126.4). A public agency may not rely on mitigation measures of uncertain efficacy or feasibility. (*Kings County Farm Bureau v. City of Hanford* (1990) 221 Cal.App.3d 692, 727 (finding groundwater purchase agreement inadequate mitigation measure because no record evidence existed that replacement water was available)). "Feasible" means capable of being accomplished in a successful manner within a reasonable period of time, taking into account economic, environmental, legal, social and technological factors. (CEQA Guidelines § 15364). To demonstrate economic infeasibility, "evidence must show that the additional costs or lost profitability are sufficiently severe as to render it impractical to proceed with the project." (*Citizens of Goleta Valley v. Board of Supervisors* (1988) 197 Cal.App.3d 1167, 1181). The EIR must provide evidence and analysis to show project cannot be economically implemented. (*Kings County, supra*, 221 Cal.App.3d at 734-737). This requires not just cost data, but also data showing insufficient income and profitability. (*See Burger v. County of Mendocino* (1975) 45 Cal.App.3d 322, 327 (infeasibility claim unfounded absent data on income and expenditures showing project unprofitable); *San Franciscans Upholding the Downtown Plan v. City and County of San Francisco* (2002) 102 Cal.App.4th 656, 694 (upholding infeasibility finding based on analysis of costs, projected revenues, and investment requirements)). Mitigation measures must be fully enforceable through permit conditions, agreements, or other legally binding instruments. (*Id.* at § 15126.4(a)(2)).

A lead agency may not conclude that an impact is significant and unavoidable without requiring the implementation of all feasible mitigation measures to reduce the impacts of a project to less than significant levels. (CEQA Guidelines §§ 15126.4, 15091).

A. The DEIR Fails to Adequately Mitigate for the Loss of Farmland

1. Preservation Is an Appropriate Mitigation Measure for the Loss of Agricultural Resources

Preservation can be used as a tool to mitigate impacts of urbanizing land and it is encouraged and supported by legislative pronouncements and case law. For example,

> [s]ee the following legislative pronouncements to the effect that conversion of agricultural land is of significant concern, and that the preservation of agricultural land is significant goal of the state. Gov. Code, § 51220 (Williamson Act findings that agricultural preservation is valuable and necessary); Civ. Code, § 815 (legislative declaration that preservation of agricultural lands "is among the most important environmental assets of California"); Pub. Resources Code, § 10200 *et seq.* (California Farmland Conservancy Program Act (formerly the Agricultural Land Stewardship Program of 1995), promoting the establishment of agricultural easements as a means to preserve agricultural land); Pub. Resources Code, §§ 21031.1, 21061.2, 21095 (CEQA provisions requiring the Resources Agency to take steps it to ensure that the environmental effects of agricultural land conversion are quantitatively and consistently considered in the environmental review process); Stats. 1993, ch. 812, § 1, subd. (d) (declaring a legislative intent that CEQA should play an important role in the preservation of agricultural lands).
>
> In *Mira Mar* [*Mobile Community v. City of Oceanside* (4th Dist. 2004) 119 Cal. App. 4th 477 [14 Cal. Rptr. 3d 176]], the court heard a challenge to the City of Oceanside's approval of a condominium project on 7.5 acres of private property. The project would cause the loss of about .86 acres of coastal sage scrub, which was identified as a significant impact to a sensitive resource. The EIR required the applicant to mitigate for this loss at a ratio of 3 to 1 (or 2.58 acres of mitigation for .86 acres of last habitat). In implementing this mitigation measure, the city required the preservation of .65 acres of undisturbed coastal sage scrub, the restoration and preservation of 2.3 acres of disturbed coastal sage scrub, and the creation of .63 acres of new coastal sage scrub on site. Petitioners argued that this mitigation was inadequate because *preservation* of coastal sage scrub does not mitigate for lost habitat, making the measure "illusory and

> inadequate." 119 Cal. App. 4th 477, 495. The Court of Appeal disagreed, citing CEQA Guildelines section 15370, as well as the opinions of various resource agencies, for the proposition that preservation can be a feasible means of reducing or eliminating the impact of lost habitat.
>
> While the *Mira Mar* case deals specifically with biological and habitat resources, the reasoning of this case seems to have more general applicability to mitigation for lost resources, including agricultural resources.

(Guide to CEQA, Michael H. Remy, et. al., eleventh edition, p. 549-550).

2. The DEIR Fails to Adopt Appropriate Mitigation Measures for the Loss of Farmland

Approximately 82.5 acres of the Project site is designated as Prime Farmland. (DEIR, p. 4.2-6). The DEIR states that "[b]ecause Prime Farmland is a finite resource, its conversion to a non-agricultural use is significant." (DEIR, p. 4.2-6). The DEIR identifies several mitigation measures including mitigation measures discussed in the City General Plan EIR:

- Enrolling productive agricultural land, not presently under contract, under a Williamson Act Contract;
- Providing protection to ongoing agricultural operations from complaints and nuisance complaints from adjacent new development;
- Protecting productive agricultural land subject to conversion through the purchase of or transfer of its development rights;
- Purchasing conservation easements on existing agricultural land to ensure that the land is never converted to urban uses; and
- Donating funds to a regional or statewide program that promotes and implements the use of agricultural land conservation easements.

(DEIR, p. 4.2-7 - 4.2-8). However, the DEIR states that

> [t]he potential mitigation measures identified by the City's General Plan have been deemed infeasible by the property owner under current economic conditions. In addition, supplementary analysis of the project

> site and local economic conditions indicates that continued citrus production and/or the raising of row crops would not be economically feasible on the project site (see Appendix L).

(DEIR, p. 4.2-8) (emphasis added).

The conclusion that the mitigation measures are infeasible is completely unsupported. The DEIR states the City General Plan EIR mitigation measure of enrolling productive land under Williamson Act contracts is infeasible because the "contracts are entered into voluntarily by property owners" and these contracts would "result only in temporary contracts at any time after the ten-year contract period ends." (DEIR, p. 4.2-8). Mitigation measures are designed to minimize significant environmental impacts, not necessarily to eliminate them. (Pub. Res. Code § 21100(b)(3); 14 Cal. Code Regs. § 15126.4(a)(1)). The minimum term for a Williamson Act contract is 10 years, however jurisdictions have the option of making them longer. (*Williamson Act Program - Basic Contract Provisions*, State of California Department of Conservation, available at http://www.conservation.ca.gov/dlrp/lca/basic_contract_provisions/Pages/index.aspx#what is a williamson act contract). Enrolling land into Williamson Act contracts would minimize the environmental impacts of converting Prime Farmland to warehouses.

In evaluating the feasibility of the mitigation measures: (1) purchasing conservation easements and (2) donating funds to a regional or statewide program, the DEIR states

> The purchase or transfer of development rights, purchase of conservation easements, or donation of funds to assist in the conservation of agricultural land would need to be implemented to ensure the preservation of agricultural land. As stated previously, the City anticipates the conversion of agricultural land within the City and does not set aside land for permanent preservation. The City expects that the majority of the land within the City will be converted to urban uses, although some agriculture will continue as interim uses, as allowed by the City's Development Code for all zoning categories.

(DEIR, p. 4.2-8 - 4.2-9). These "reasons" are flawed because the identified mitigation measure was to donate funds to *regional* or *statewide* programs that promote and implement the use of agricultural land conservation easements. The "reasons" do not address why donating funds to regional or statewide programs is infeasible.

A supplemental EIR is required to analyze and require implementation of these feasible mitigation measures to reduce the Project's impacts on agricultural land. The fact that the measures are set forth in the City's own General Plan itself makes a prima facie case that the measures are feasible and should be implemented. If the City

concludes that the measures are infeasible, then it must provide substantial evidence to demonstrate infeasibility. The EIR must provide evidence and analysis to show project cannot be economically implemented. (*Kings County, supra*, 221 Cal.App.3d at 734-737). This requires not just cost data, but also data showing insufficient income and profitability. (*See Burger v. County of Mendocino* (1975) 45 Cal.App.3d 322, 327 (infeasibility claim unfounded absent data on income and expenditures showing project unprofitable); *San Franciscans Upholding the Downtown Plan v. City and County of San Francisco* (2002) 102 Cal.App.4th 656, 694 (upholding infeasibility finding based on analysis of costs, projected revenues, and investment requirements)). The EIR is devoid of any such evidence and is therefore legally inadequate.

B. The DEIR Fails to Adequately Analyze Hazards and Hazardous Materials and Establishes an Erroneous Baseline

1. CEQA Baseline Standard

Every CEQA document must start from a "baseline" assumption. The CEQA "baseline" is the set of environmental conditions against which to compare a project's anticipated impacts. *Communities for a Better Environment v. So Coast Air Qual. Mgmnt. Dist*. (2010) 48 Cal. 4th 310, 321. Section 15125(a) of the CEQA Guidelines (14 C.C.R., § 15125(a)) states in pertinent part that a lead agency's environmental review under CEQA:

> …must include a description of the physical environmental conditions in the vicinity of the project, as they exist at the time [environmental analysis] is commenced, from both a local and regional perspective. This environmental setting will normally constitute the baseline physical conditions by which a Lead Agency determines whether an impact is significant.

(See, *Save Our Peninsula Committee v. County of Monterey* (2001) 87 Cal.App.4th 99, 124-125 ("*Save Our Peninsula*"). As the court of appeal has explained, "the impacts of the project must be measured against the 'real conditions on the ground,'" and not against hypothetical permitted levels. (*Save Our Peninsula*,87 Cal.App.4th 99, 121-123). As the court has explained, using such a skewed baseline "mislead(s) the public" and "draws a red herring across the path of public input." (*San Joaquin Raptor Rescue Center v. County of Merced* (2007) 149 Cal.App.4th 645, 656; *Woodward Park Homeowners v. City of Fresno* (2007) 150 Cal.App.4th 683, 708-711).

2. Residual Pesticides in the Soil May Pose Health Risks to Workers and Nearby Residents

According to the DEIR, 57 acres of the Project site are used to grow grapefruit and 36 acres of the Project site are used for hay and alfalfa production. (DEIR, p. 4.2-1).

The DEIR and supporting documents fail to provide any specific details on the types of pesticides that have been used on the Project site in association with these agricultural operations and therefore the DEIR fails to adequately describe the environmental setting for the Project. According to Mr. Hagemann,

> [o]ur review has shown known and potential pesticide use at the Project site as follows:
>
> - Data available online from the California Department of Pesticide Regulation show that 2,4-D, 2-Ethylhexyl Ester was used on the Project site.[2,3] Occupational exposure to 2,4-D, 2-Ethylhexyl Ester can occur via inhalation or dermal contact and can result in skin irritation, respiratory failure, hyperventilation, and pulmonary enemas.[4]
> - Organochlorine pesticides DDE and DDT were detected in soil samples collected at the Project site[5], indicating past use. Use of organochlorine pesticides in the area is common: review of the Department of Toxic Substances Control's (DTSC) Envirostor database shows that the surrounding lands have been surveyed for organochlorine pesticides, DDE and DDT.[6] These pesticides can persist in soil for hundreds of years despite being banned in the 1970s.[7] Exposure to DDT can result in headaches, nausea, and convulsions.[8] The U.S. EPA identifies DDT and DDE as probable human carcinogens.[9]
> - The EPA states that soils at fruit orchards, such as the grapefruit orchard on the Project site, may contain high levels of arsenic from application as a pesticide.[10] Another chemical used on fruit orchards is lead arsenate, a very persistent pesticide.[11] Arsenic is a known human carcinogen and even short-term inhalation of arsenic

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[2] ftp://cdpr.ca.gov/pub/outgoing/calpip/26814174623515_120824104149.html
[3] ftp://cdpr.ca.gov/pub/outgoing/calpip/26814174623515_120824104217.html
[4] http://toxnet.nlm.nih.gov/cgi-bin/sis/search/a?dbs+hsdb:@term+@DOCNO+7309
[5] Phase I Environmental Site Assessment, 84 acres. Near Intersection of Pittit Street and Highway 60, Moreno Valley, California, p. 9 and Phase I Environmental Site Assessment, 37 acres. Near Intersection of Pittit Street and Highway 60, Moreno Valley, California, p. 8
[6] http://www.envirostor.dtsc.ca.gov/public/profile_report.asp?global_id=60000825 and http://www.envirostor.dtsc.ca.gov/public/profile_report.asp?global_id=60000931
[7] http://www.atsdr.cdc.gov/toxprofiles/tp35.pdf, p. 3
[8] http://www.epa.gov/ttn/atw/hlthef/dde.html
[9] http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=80&tid=20
[10] http://water.epa.gov/drink/info/well/health.cfm
[11] http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1551991/

dust can cause gastrointestinal effects[12] while lead is known to cause neurotoxicological effects.[13]

Pesticide use at the Project site was not disclosed in the DEIR and the detection of pesticide residuals in soil were not described in the Hazards and Hazardous Materials section.

Failure to disclose the presence of pesticide residuals in Project site soils may pose significant health risks to construction workers. Construction of the Project requires grading and the disturbance of subsurface soils and removal of citrus groves (DEIR, p. 4.7-21). During earthmoving activities, construction workers will be exposed, via inhalation of dust and dermal contact, to Project site soils which may contain harmful levels of pesticide residuals associated with agricultural activities on the site. To protect worker safety, Project site soils must be sampled for pesticides. Sampling results should be compared to health-protective regulatory screening levels such as U.S. EPA Regional Screening Levels[14] and California Human Health Screening Levels.[15]

Soil sampling results should also be evaluated for the protection of nearby residents, located 50 feet from the southern boundary and 200 feet from the northern boundary of the Project site (DEIR, p. 4.3-6). Inhalation of pesticides has been linked to asthma in recent research.[16,17] A report prepared by the California Department of Health identifies pesticides as an asthma trigger.[18] Offsite receptors, including any children living in the neighboring residences, may be exposed to pesticide residuals via dust generated during Project construction.

Construction activities, such as grading and excavation of soils, may generate dust that contains pesticides in concentrations that are harmful to the health of workers and nearby residents and which may act as an asthma trigger. Project site soils should be sampled and results should be compared to human health screening levels. A revised DEIR should be prepared to disclose the results of sampling and include any necessary mitigation to reduce impacts to the health of construction workers and nearby residents.

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[12] http://www.epa.gov/ttn/atw/hlthef/arsenic.html
[13] http://www.epa.gov/lead/pubs/learn-about-lead.html#effects
[14] http://www.epa.gov/region9/superfund/prg/
[15] http://www.calepa.ca.gov/brownfields/documents/2005/CHHSLsGuide.pdf
[16] http://extension.psu.edu/ipm/resources/urbanphilly/partnerships/handouts/asthma-pests.pdf
[17] http://www.ncbi.nlm.nih.gov/pubmed/21368619
[18] http://www.cdph.ca.gov/programs/caphi/Documents/AsthmaStrategicPlan.5-5-08.pdf, p. 22

3. The Phase I Environmental Site Assessments Completed for the Project Site are Outdated and Inadequate

According to Mr. Hagemann,

The DEIR relies on the findings from two Phase I Environmental Site Assessments (ESAs) that were completed in October and November 2003, nearly nine years ago. The Phase I ESAs surveyed 121 acres of the 123-acre Project site. The Applicant purchased the Project site more than five years ago.[19] A Phase I ESA, according to the U.S. EPA, must be conducted within one year of the acquisition of the property and on-site visual inspections must be completed within 180 days prior to acquiring ownership of the property.[20]

Because the Phase I ESAs are dated and omit two acres of the Project site, they cannot be used to evaluate conditions that are potentially hazardous to construction workers and future site personnel. Therefore, the DEIR's analysis of the Project site based on these Phase I ESAs is inadequate.

Review of Google Earth images shows that the Project site has been used for ongoing agricultural operations since the Phase I ESAs were completed in 2003. Limited pesticide sampling was conducted during the Phase I ESAs (a total of 8 soil samples for a 123-acre Project site) but because the samples were collected nine years ago and because they do not reflect continued agricultural use, the results are reflective of current site conditions.

The Phase I ESAs cover 121 acres of the 123-acre Project site. We have created a map to show the areas of the Project site surveyed by the two 2003 Phase I ESAs and the boundaries for the current Project site (Attachment A). As the map shows, not all areas of the current Project site were included in the 2003 Phase I ESAs' site assessments.

The DEIR fails to establish an adequate environmental setting for the Project site because it relies on Phase I ESAs that are outdated and do not cover the entire Project site. A revised DEIR is required, including a new Phase I ESA, to evaluate the Project site's current environmental conditions.

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[19] http://www.pe.com/local-news/riverside-county/moreno-valley/moreno-valley-headlines-index/20120726-moreno-valley-officials-seek-comments-on-prologis-project.ece
[20] http://www.epa.gov/brownfields/aai/aaicerclafs.pdf

4. The DEIR Fails to Disclose the Status of an Underground Storage Tank

According to Mr. Hagemann,

> A 13,400 gallon underground storage tank (UST), abandoned in the 1950s, was removed from the Project site in 2004 (Appendix F, p. 3/191). The Phase I ESA recommended an additional investigation to be conducted in the area of the former UST (Appendix F, p. 10/191). Accordingly, a permit for removal of the UST was submitted to the Riverside County's Department of Environmental Health in December 2003 and soil samples around the area of the UST were analyzed in 2004. However, the DEIR and supporting documents did not include any documentation that the UST was properly closed by the Riverside County Department of Environmental Health. If the UST removal was not approved, an Underground Storage Tank Closure Application and Permit[21], per the Riverside County Department of Environmental Health Guidelines,[22] must be submitted.

The DEIR fails to establish an adequate baseline because it does not provide the status of a 13,400 gallon UST. A revised DEIR is required to disclose this important information (i.e., whether closure was granted by the Riverside County Department of Environmental Health).

C. The DEIR Fails to Adequately Analyze Greenhouse Gas Emissions

The DEIR states that the Project's operational greenhouse gas (GHG) emissions, before mitigation, are estimated to be 79,000 metric tons of CO_2e/year (MT CO_2e/yr) which exceed the SCAQMD threshold of 10,000 MT CO_2e/yr and are therefore considered significant. (DEIR, p. 4.13-19). The Project's GHG emissions exceed the SCAQMD threshold by nearly eight times. After mitigation, the DEIR states that GHG emissions will be less than significant. (DEIR, p. 4.13-21). This conclusion is completely unsupported. The DEIR fails calculate what the Project's GHG emissions will be after the mitigation measures are implemented. In fact, the DEIR and supporting documents, including a GHG Study (Appendix B), fail to provide any evidence that the proposed mitigation measures will reduce GHG emissions by a factor of eight.

According to Mr. Hagemann,

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[21] http://www.rivcoeh.org/opencms/system/galleries/download/Environmental-Health/HMM/UST_Closure_App.pdf
[22] http://www.rivcoeh.org/opencms/system/galleries/download/Environmental-Health/HMM/Closure_by_removal_UST.pdf

A revised DEIR should be prepared to show the efficiency of the Project's proposed mitigation measures in reducing greenhouse gases. If these measures do not account for an eight-fold reduction in the Project's estimated GHG levels, additional mitigation measures (listed below) that are routinely considered in other CEQA projects should be implemented:

- Require preparation of a traffic control plan;
- Demonstrate proper inspection and maintenance of construction equipment;
- Implement a carpool program for construction workers;
- Employ a construction site manager to verify that engines are properly maintained and keep a maintenance log;
- Configure construction parking to minimize traffic interference;
- Consolidate truck deliveries when possible;
- Provide dedicated turn lanes for movement of construction trucks and equipment on and off site;
- Suspend use of all construction equipment operations during second stage smog alerts;
- Establish a staging zone for trucks that are waiting to load or unload material at the work zone in a location where diesel emissions from the trucks will have minimum impact on abutters and the general public;
- Locate construction equipment away from sensitive receptors such as fresh air intakes to buildings, air conditioners and operable windows;
- Require all diesel trucks used by construction contractor(s) at the site, or for on-road hauling of construction material, to be post-1996 models; Diesel portable generators less than 50 hp shall not be allowed at the construction site;
- Use of hybrid and fuel efficient construction equipment and support vehicles (e.g., pick-up trucks);
- Use of grid electricity for smaller equipment such as saws, pumps, and welders;[23]
- Reduction in vehicle miles travelled in construction crew commutes through trip carpooling, trip reduction, providing bus service for crews from work sites to carpool parking areas, and in providing incentives to carpool; and
- Use of a Heavy-Duty Off-Road Vehicle Plan to ensure compliances with construction mitigation measures (e.g., hourly meters on equipment, documenting the serial number, horsepower,

[23] http://www.capcoa.org/wp-content/uploads/2010/11/CAPCOA-Quantification-Report-9-14-Final.pdf, p. 47

> manufacture age, fuel, etc. of all onsite equipment and daily logging of the operating hours of the equipment).[24]

A supplemental EIR should be prepared that calculates the Project's GHG emissions after implementation of all feasible mitigation measures. The supplemental EIR should analyze all mitigation measures set forth in the GHG Guidance Document published by the California Attorney General, Addressing Climate Change at the Project Level (see attached exhibit, also available at http://ag.ca.gov/globalwarming/pdf/GW_mitigation_measures.pdf). If GHG impacts remain significant after implementation of all feasible mitigation measures, then the EIR must acknowledge that the impacts are significant an unavoidable, and the City must adopt a statement of overriding considerations.

D. The DEIR Fails to Adequately Analyze Impacts to Air Quality

1. The DEIR Underestimates the Potential Particulate Emissions for the Construction Phase of the Project

Computer modeling (e.g., the California Air Resource Board's ("CARB's") Urban Emission ("URBEMIS") and the California Emissions Estimator Model ("CalEEMod")) is used to estimate emissions of criteria pollutants during construction and operational phases of projects. The South Coast Air Quality Management District ("SCAQMD") permits the use of the outputs from both the URBEMIS and CalEEMOD in air quality analyses. According to Dr. Clark, there are significant differences between these two models that "must be highlighted in the DEIR." In pertinent part, Dr. Clark states:

> The changes in the method used to estimate construction impacts from the proposed project by using the CalEEMod model instead of the URBEMIS model include:
>
> - Failure to account for wind-blown fugitive dust[25]. According to the July, 2011 CalEEMod Technical Paper, wind-blown fugitive dust is not calculated in CalEEMod. For sites as large as the proposed project site, this can result in significant quantities of particulate matter being released.
> - SCAQMD's surveys of construction sites were limited to sites of 35 acres or less. For projects larger than 35 acres the data was extrapolated by increasing the number of construction days but not increasing the number of construction equipment pieces used on a

[24] Ibid., p. 431
[25] CalEEMod. Technical Paper: Methodology Reasoning and Policy Development of the California Emission Estimator Model. July, 2011. Pg 4.

given day. The impact is to reduce the construction PM estimates for the site as compared to URBEMIS[26].

- The acreage to be based upon Walker's Building Estimator's Reference Book. Grading in URBEMIS is based upon 25% of total project acreage in one day. The impact of this change is to decrease PM emissions from grading in the CalEEMod[27].

A revised DEIR should be prepared to highlight the differences between the two models so that the potential impacts are adequately analyzed.

2. The DEIR Fails to Accurately Compare Construction Emissions to Daily Construction Significant Thresholds

According to Dr. Clark, the CalEEMod results were not presented properly. The model shows CEQA significance levels were exceeded as well as South Coast Air Quality Management District Localized Significance Thresholds were exceeded. In pertinent part, Dr. Clark states:

> Unlike the operational emissions from most projects, which are typically more or less continuous, emissions from construction sites are highly variable depending on the type of construction that is being performed. For example, grading results in large quantities of fugitive dust and combustion emissions from diesel-powered equipment. Short-term emissions during the various construction phases can be considerable and may result in degradation of local and regional air quality and severe health effects.
>
> To determine whether short-term emissions may result in degradation of local and regional air quality and severe health effects, it is common practice for lead agencies to compare project emissions to quantitative significance thresholds developed by local air districts as a screening tool for CEQA review. Thresholds of significance for construction emissions are typically expressed on a short-term basis, *i.e.* daily or hourly basis to adequately capture impacts due to the high variability of emissions during different construction stages.
>
> Table 1 presents a summary of short-term emissions thresholds developed by SCAQMD and other air districts for assessing impacts on air quality from construction projects.

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[26] CalEEMod. Technical Paper: Methodology Reasoning and Policy Development of the California Emission Estimator Model. July, 2011. Pg 5.
[27] CalEEMod. Technical Paper: Methodology Reasoning and Policy Development of the California Emission Estimator Model. July, 2011. Pg 5.

Table 1:
CEQA significance thresholds for construction emissions from various air districts

Air district construction thresholds*	**NOx** (lbs/day)	**ROG** (lbs/day)	**PM_{10}** (lbs/day)	**DPM** (lbs/day)	**$PM_{2.5}$** (lbs/day)	**CO** (lbs/day)
SCAQMD	100	75	150		55	550
BAAQMD	54	54	82		54	
EDCAPCD	82	82				
SLOCAPCD				7		
MBUAPCD			82			550
FRAQMD	25	25	80			
SMAQMD	85					
YSAQMD	82	82	150			

SCAQMD = South Coast Air Quality Management District, CEAQ Handbook, 1993;
BAAQMD = Bay Area Air Quality Management District, CEQA Guidelines 2009;
EDCAPCD = El Dorado County Air Pollution Control District, CEQA Guide, February 2002;
SLOCAPCD = San Louis Obispo County Air Pollution Control District, CEQA Air Quality Handbook, December 2009.
MBUAPCD = Monterey Bay Unified Air Pollution Control District, CEQA Air Quality Guidelines, June 2004,
FRAQMD = Feather River Air Quality Management District, http://www.fraqmd.org/CEQA_Thresholds.htm;
SMAQMD Sacramento Metropolitan Air Quality Management District, Guide to Air Quality Assessment, July 2004;
YSAQMD, Yolo-Solano Air Quality Management District, Air Quality Handbook, Guidelines for Determining Air Quality Thresholds of Significance and Mitigation Measures for Proposed Development Projects that Generate Emissions from Motor Vehicles, revised 2002

A review of the CalEEMod analysis for the project shows that the mitigated construction emissions of ROG and PM2.5 exhaust (a surrogate for diesel particulate emissions) are in excess of the CEQA significance thresholds listed above. During Year 2013, ROG and PM2.5 exhaust emissions are estimated to be 368.03 lbs/day and 7.95 lbs/day, respectively.

In addition to the Significant Thresholds above, SCAQMD recommends the use of Localized Significance Thresholds (LSTs) to determine potential impacts to receptors near projects. According to the Air Quality Analysis prepared by the proponent, Table I of the Air Quality Analysis shows that the emissions of the pollutants on the peak day of construction are below the SCAQMD LST. In this table the proponent uses the emission estimates from the grading phase of the construction. The proponent inaccurate asserts that the emission levels will be below the LST values.

Table 2:
Construction LST Impacts from Air Quality Analysis

Emission Sources	Pollutant Emissions, lbs/day			
	NOx	CO	PM_{10}	$PM_{2.5}$
On-Site (grading) Emissions	104	55	8.4	6.3
LST Threshold	270	1,577	13	8
Significant Emissions?	No	No	No	No

A review of the CalEEMod analysis shows that the highest emission values are not associated with the grading phase. In Section 2.0 Emission Summary of the CalEEMod analysis presented in the Air Quality Analysis the construction impacts are listed as:

Table 3:
Construction LST Impacts from CalEEMod Output

Emission Sources	Pollutant Emissions, lbs/day			
	NOx	CO	PM_{10}	$PM_{2.5}$
Mitigated Construction Emissions	139.84	166.77	29.2	8.28
LST Threshold	270	1,577	13	8
Significant Emissions?	No	No	**Yes**	**Yes**

The Proponent's analysis of air quality impacts clearly fails to accurately describe the impacts of the emissions on the receptors closest to the project site. Based on my expert opinion, applicable significance thresholds, and the CalEEMod analysis performed by the proponent, I conclude that the Project will have significant adverse impacts from construction air emissions of fugitive dust, ROG, and diesel emissions. The lead agency must re-evaluate the construction emissions and incorporate a phased approach to estimate the true impacts of construction activities on air quality, and propose all feasible mitigation measures to reduce these significant emissions, in a RDEIR.

3. The DEIR Fails to Consider Health Risks From Contaminated Dust

According to Dr. Clark:

Residual contaminants in soils at the site may be entrained in dust generated during construction activities. The release of residual contamination is a potentially significant impact, given the past use of the site for agricultural production. According to the California Department of Toxic Substances Control August 2002 Interim Guidance for Sampling Agricultural Fields for School Sites (known sensitive receptors), "the most commonly detected pesticides have been DDT and it's derivatives DDD and DDE, toxaphene, dieldrin, and aldrin. Of these pesticides, toxaphene has been the major pesticide driving unacceptable levels of risk requiring remediation by soil removal." Given the volume of soils to be graded at each of the sites it is imperative to understand whether particulate matter generated at the sites will pose a potential health risk to sensitive receptors in the vicinity of each site.

According to DTSC, "the guidance is applicable to agricultural land that is currently under cultivation with row, fiber or food crops, orchards, or pasture. It is also applicable to fallow and former agricultural land that is no longer in production and has not been disturbed beyond normal disking and plowing practices. Each field of the same crop is assumed to have been watered, fertilized, and treated with agricultural chemicals to the same degree across the field. Because of this homogeneous application, contaminant levels are expected to be similar at any given location within the field."

There is no indication of a sampling and analysis plan in the DEIR, or the Project documents provided by the lead agency, a serious deficiency in the documents. Prior to issuing a DEIR for the project, the Proponent should be required to complete a sampling and analysis plan to confirm or rule out the possibility of the presence of residual contaminants at the site. Identifying residual pesticides or other contaminants in soils at the site prior to construction activities will provide an opportunity for the Proponent to remove/mitigate the potential exposure of sensitive receptors within the vicinity of the sites. In the absence of any sampling or analysis, and given the past use of the Project site, I conclude that there is at least a fair argument that the Project may have significant impacts related to residual contaminants at the site.

4. The DEIR Fails to Properly Identify and Address the Project's Operational Air Quality Impacts

The DEIR states, without any evidentiary support, that the project's emissions of criteria pollutants will not result in a considerable net increase of any criteria pollutant for which the project region is in non-attainment under an applicable federal or state ambient air quality standard. According to Dr. Clark,

> During the operational phase of the project the project will have the potential to generate significant quantities of criteria pollutants (NO_x, SO_x, Ozone precursors, PM). According to Table 3-1 of the most recent BAAQMD CEQA guidance, a construction of a 259,000 square foot light industrial or warehouse operation will typically violate NOx construction thresholds and GHG operational thresholds. The proposed Project's 2,000,000 square feet plus of warehouse and manufacturing buildings are nearly 8 times the size of the screening threshold, ensuring a violation of local air quality thresholds. I therefore conclude that the Project will have significant NOx and GHG emissions during Project operations.
>
> The air quality impacts from the traffic associated with a 2,000,000 square foot facility are significant. Typically the impacts are quantified by the number of vehicle trips per day. In the case of the proposed project, the primary concern will be the number of truck trips per day. A truck trip is one round trip (one trip segment to a site and one trip segment away from a site).
>
> According to one source, Bluffstone and Ouderkirk[28], a 500,000 square feet facility on 50 acres, will on average have 350 truck trips per day (or 700 trip segments) associated with its development. This figure is proportionate to estimates for an AMB Property Corporation center in Redlands (1,000 truck trips for a 1.3 million square feet structure); Wal-Mart distribution centers in Pueblo, Colorado (700 truck trips per day for an 880,000 square feet facility), Connecticut, and Delaware (both 1,000 truck trips per day for 1.2 million square feet structures); and a grocery distribution center in New York (Boas, 2002; Gasiewski, 2004; Hernandez, 2005; Pueblo Chieftain, 2004; Sholl, 2004).
>
> Estimates from other sources indicate approximately 1 truck per 1,000 square feet of the building, which means that the proposed project would require 1,000 trucks per day (or 1,000 trip segments per day) for the warehouse segment of the Project. The number of truck trips could be

[28]Bluffstone and Ouderkirk. 2007. Warehouses, trucks, and [PM.sub.2.5]: human health and logistics industry growth in the eastern Inland Empire. Contemporary Economic Policy 25(1):

higher at a new, more efficient facility where more inventory is moved per day. Without proper modeling of the emissions from these additional vehicles the impacts on the environment and the citizens of Moreno Valley is unknown. It is clear that the size of the Project will have significant NOx and GHG emissions during Project operations.

A proper cumulative impact analysis is vital for an environmental analysis 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 the environmental damage often occurs incrementally from a variety of small sources with which they interact. The increase in PM in the region, even for short periods of time, will only exacerbate the already serious air quality issues in the region.

5. The DEIR Fails to Disclose Impacts to Offsite Receptors

The Project is located in the South Coast Air Basin and Riverside County,[29] both of which are designated non-attainment for PM10 and ozone. (DEIR, p. 4.3-6). According to Mr. Hagemann,

> [s]ignificant emissions of PM10 and ozone and contributing factors such as NOx and ROG will lead to a worsening of regional air quality. The Project's estimates of construction emissions need to be properly disclosed and mitigated to ensure that the Project has a less than significant impact on regional air quality.
>
> *Estimates and impacts of project's construction and operational emissions*
> Project construction and operation will result in significant emissions of ROG, NOx, and PM10 even after mitigation (DEIR, pp. 4.3-29, 4.3-34).
>
> Construction emissions
> The DEIR estimates that the Project's construction emissions of NOx and ROG will be significant as they exceed the SCAQMD thresholds of 100 lbs/day and 75 lbs/day, respectively (DEIR, p. 4.3-23) and identifies mitigation measures to reduce the Project's emissions (DEIR, pp. 4.3-23 – 4.3-29). Even with mitigation, the Project's emissions of NOx and ROG will still exceed SCAQMD thresholds and therefore are considered significant (DEIR, p. 4.3-29).
>
> Operational emissions
> The DEIR estimates that the Project's emissions of ROG, NOx, and PM10 from operational activities will be significant as they exceed the SCAQMD

[29] http://www.epa.gov/oaqps001/greenbk/ancl.html

thresholds of 55 lbs/day, 55 lbs/day, and 150 lbs/day, respectively (DEIR, p. 4.3-33). The DEIR proposes mitigation measures to reduce the Project's emissions (DEIR, pp. 4.3-33 – 4.3-34) but, even with mitigation, the Project's emissions will still exceed SCAQMD thresholds and therefore are considered significant (DEIR, p. 4.3-34).

Gaseous particles such as NOx can react in the atmosphere to form PM10.[30,31] Because Riverside County and the South Coast Air Basin are both designated non-attainment for PM10, significant emissions of NOx can lead to a further degradation of regional air quality. NOx emissions can also react to produce ground-level ozone.[32] Exposure to NOx emissions and its products (ozone and PM10) can lead to the airway inflammation and can cause or exacerbate conditions such as emphysema and bronchitis.[33]

ROG can react to form ozone and contributes to smog formation.[34,35] Exposure to ozone can result in coughing, throat irritation, and chest pain, burning, and discomfort.[36] Smog exposure can lead to sneezing, nausea, coughing, headaches, and chest constriction.[37] A study published in the New England Journal of Medicine concluded that the risk of dying from respiratory diseases is three times higher in areas of concentrated ozone.[38]

Exposure to PM10 can cause bronchitis, increase the number and severity of asthma attacks, damage to lung tissue, and even premature death.[39] Research identifies that dust from construction is a major contributor to PM10 and that PM10 exposure is associated with asthma.[40] Inhalation of PM10 can exacerbate asthma especially in children who are susceptible to higher risks from PM10 exposure.[41]

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[30] http://www.fhwa.dot.gov/resourcecenter/teams/airquality/brochure/particulatebrochure.pdf
[31] http://www.epa.gov/captrade/documents/power.pdf
[32] *Ibid.*
[33] http://www.epa.gov/air/nitrogenoxides/health.html
[34] http://www.arb.ca.gov/research/health/fs/fs2/fs2.htm
[35] http://www.arb.ca.gov/html/gloss.htm#smog
[36] http://www.epa.gov/o3healthtraining/population.html
[37] http://are.berkeley.edu/courses/EEP101/spring03/AllThatSmog/extern.html
[38] http://www.nytimes.com/gwire/2009/03/12/12greenwire-study-links-smog-exposure-to-premature-death-10098.html
[39] http://www.epa.gov/airtrends/aqtrnd95/pm10.html and http://www.arb.ca.gov/html/brochure/pm10.htm
[40] http://scerpfiles.org/cont_mgt/doc_files/EH-01-2.pdf
[41] http://www.co.imperial.ca.us/airpollution/attainment%20plans/final%20ic%202009%20pm10%20sip%20document.pdf

The Project will have significant emissions of ROG, NOx, and PM10. Because Riverside County and the South Coast Air Basin are designated non-attainment areas for ozone and PM10, Project construction and operation will further degrade regional air quality. Exposure to ROG, NOx, and PM10 has adverse health effects and can impact offsite receptors, especially children in the nearby residences – a significant and undisclosed public health impact that the DEIR does not consider.

A revised DEIR should be prepared to disclose impacts to offsite receptors from Project construction and operation. Additional mitigation measures must be implemented to ensure that Project emissions of ROG, NOx, and PM10 are reduced to the maximum extent feasible.

6. The DEIR Fails to Adequately Analyze Cumulative Impacts

1. Legal Standard

An EIR must discuss significant cumulative impacts. CEQA Guidelines section 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." "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 section 15355(a). "[I]ndividual effects may be changes resulting from a single project or a number of separate projects." (CEQA Guidelines section 15355(a)).

"The cumulative impact from several projects is the change in the environment which results from the incremental impact of the project 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." (*Communities for a Better Environment v. Cal. Resources Agency ("CBE v. CRA")*, (2002) 103 Cal.App.4th 98, 117). 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)).

As the court stated in *CBE v. CRA,* 103 Cal. App. 4th at 114:

> 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.

(Citations omitted).

In *Kings County Farm Bureau v. City of Hanford,* 221 Cal.App.3d at 718, the court concluded that an EIR inadequately considered an air pollution (ozone) cumulative impact. The court said: "The EIR concludes the project's contributions to ozone levels in the area would be immeasurable and, therefore, insignificant because the [cogeneration] plant would emit relatively minor amounts of [ozone] precursors compared to the total volume of [ozone] precursors emitted in Kings County. The EIR's analysis uses the magnitude of the current ozone problem in the air basin in order to trivialize the project's impact." The court concluded: "[t]he relevant question to be addressed in the EIR is not the relative amount of precursors emitted by the project when compared with preexisting emissions, but whether any additional amount of precursor emissions should be considered significant in light of the serious nature of the ozone problems in this air basin."[42] The *Kings County* case was reaffirmed in *CBE v. CRA,* 103 Cal.App.4th at 116, where the court rejected cases with a narrower construction of "cumulative impacts."

Similarly, in *Friends of Eel River v. Sonoma County Water Agency,* (2003) 108 Cal. App. 4th 859, the court held that the EIR for a project that would divert water from the Eel River had to consider the cumulative impacts of the project together with other past, present and reasonably foreseeable future projects that also divert water from the same river system. The court held that the EIR even had to disclose and analyze projects that were merely proposed, but not yet approved. The court stated, CEQA requires "the Agency to consider 'past, present, and probable future projects producing related or cumulative impacts . . . .'" (Guidelines, § 15130, subd. (b)(1)(A)). The Agency must interpret this requirement in such a way as to 'afford the fullest possible protection of the environment.'" (*Id.*, at 867, 869). The court held that the failure of the EIR to

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[42] *Los Angeles Unified v. City of Los Angeles*, 58 Cal.App.4th at 1024-1026 found an EIR inadequate for concluding that a project's additional increase in noise level of another 2.8 to 3.3 dBA was insignificant given that the existing noise level of 72 dBA already exceeded the regulatory recommended maximum of 70 dBA. The court concluded that this "ratio theory" trivialized the project's noise impact by focusing on individual inputs rather than their collective significance. The relevant issue was not the relative amount of traffic noise resulting from the project when compared to existing traffic noise, but whether any additional amount of traffic noise should be considered significant given the nature of the existing traffic noise problem.

analyze the impacts of the project together with other proposed projects rendered the document invalid. "The absence of this analysis makes the EIR an inadequate informational document." (*Id.,* at 872).

The Court in *Citizens to Preserve the Ojai v. Bd. of Supervisors,* 176 Cal.App.3d 421 (1985), held that an EIR prepared to consider the expansion and modification of an oil refinery was inadequate because it failed to consider the cumulative air quality impacts of other oil refining and extraction activities combined with the project. The court held that the EIR's use of an Air District Air Emissions Inventory did not constitute an adequate cumulative impacts analysis. The court ordered the agency to prepare a new EIR analyzing the combined impacts of the proposed refinery expansion together with the other oil extraction projects.

2. The DEIR Fails to Adequately Analyze Cumulative Construction Impacts

As part of its cumulative impact analysis, the DEIR identifies 13 proposed projects encompassing approximately 7.3 million square feet of space within five miles of the Project site (DEIR, p. 3-16). However, the DEIR does not identify the construction schedule of these projects except to state that "a number of individual projects may be under construction simultaneously with the proposed project." (DEIR, p. 4.3-37). The WestRidge Commerce Center Project (which will be built adjacent to the proposed Project) is scheduled to be constructed in 2012, a schedule similar to the proposed Project. According to Mr. Hagemann,

> [s]imultaneous construction of these projects, along with other potential projects, is likely to result in PM10, NOx, and ROG emissions that will have a cumulatively significant impact. The construction timetables of all projects within the vicinity of the Project site should be identified. Any cumulatively significant emissions should be disclosed and impacts to workers and nearby residents should be addressed in a revised DEIR.

VI. CONCLUSION

For the foregoing reasons, LIUNA Local Union No. 1184 urge the City to continue the matter for future consideration pending completion of a supplemental EIR addressing the Project's significant impacts and mitigation measures. Thank you for your attention to these comments. Please include this letter and all attachments hereto in the record of proceedings for this project.

Sincerely,

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Attorneys for LIUNA Local Union No. 1184

Exhibit

Addressing Climate Change at the Project Level
California Attorney General's Office

Addressing Climate Change at the Project Level
California Attorney General's Office

Under the California Environmental Quality Act (CEQA), local agencies have a very important role to play in California's fight against global warming – one of the most serious environmental effects facing the State today. Local agencies can lead by example in undertaking their own projects, insuring that sustainability is considered at the earliest stages. Moreover, they can help shape private development. Where a project as proposed will have significant global warming related effects, local agencies can require feasible changes or alternatives, and impose enforceable, verifiable, feasible mitigation to substantially lessen those effects. By the sum of their actions and decisions, local agencies will help to move the State away from "business as usual" and toward a low-carbon future.

Included in this document are various measures that may reduce the global warming related impacts at the individual project level. (For more information on actions that local governments can take at the program and general plan level, please visit the Attorney General's webpage, "CEQA, Global Warming, and General Plans" at http://ag.ca.gov/globalwarming/ceqa/generalplans.php.)

As appropriate, the measures can be included as design features of a project, required as changes to the project, or imposed as mitigation (whether undertaken directly by the project proponent or funded by mitigation fees). The measures set forth in this package are examples; the list is not intended to be exhaustive. Moreover, the measures cited may not be appropriate for every project. The decision of whether to approve a project – as proposed or with required changes or mitigation – is for the local agency, exercising its informed judgment in compliance with the law and balancing a variety of public objectives.

Mitigation Measures by Category

Energy Efficiency

Incorporate green building practices and design elements.	The California Department of Housing and Community Development's Green Building & Sustainability Resources handbook provides extensive links to green building resources. The handbook is available at http://www.hcd.ca.gov/hpd/green_build.pdf. The American Institute of Architects (AIA) has compiled fifty readily available strategies for reducing fossil fuel use in buildings by fifty percent. AIA "50 to 50" plan is presented in both guidebook and wiki format at http://wiki.aia.org/Wiki%20Pages/Home.aspx.

Meet recognized green building and energy efficiency benchmarks.	For example, an ENERGY STAR-qualified building uses less energy, is less expensive to operate, and causes fewer greenhouse gas emissions than comparable, conventional buildings. http://www.energystar.gov/index.cfm?c=business.bus_index. California has over 1600 ENERGY STAR-qualified school, commercial and industrial buildings. View U.S. EPA's list of Energy Star non-residential buildings at http://www.energystar.gov/index.cfm?fuseaction=labeled_buildings.locator. Los Angeles and San Francisco top the list of U.S. cities with the most ENERGY STAR non-residential buildings. http://www.energystar.gov/ia/business/downloads/2008_Top_25_cities_chart.pdf. Qualified ENERGY STAR homes must surpass the state's Title 24 energy efficiency building code by at least 15%. Los Angeles, Sacramento, San Diego, and San Francisco-Oakland are among the top 20 markets for ENERGY STAR homes nationwide. http://www.energystar.gov/ia/new_homes/mil_homes/top_20_markets.html. Builders of ENERGY STAR homes can be more competitive in a tight market by providing a higher quality, more desirable product. See http://www.energystar.gov/ia/partners/manuf_res/Horton.pdf. There are a variety of private and non-profit green building certification programs in use in the U.S. See U.S. EPA's Green Building / Frequently Asked Questions website, http://www.epa.gov/greenbuilding/pubs/faqs.htm. Public-Private Partnership for Advancing Housing Technology maintains a list of national and state Green Building Certification Programs for housing. See http://www.pathnet.org/sp.asp?id=20978. These include the national Leadership in Energy and Environmental Design (LEED) program, and, at the state level, Build it Green's GreenPoint Rated system and the California Green Builder program. Other organizations may provide other relevant benchmarks.
Install energy efficient lighting (e.g., light emitting diodes (LEDs)), heating and cooling systems, appliances, equipment, and control systems.	Information about ENERGY STAR-certified products in over 60 categories is available at http://www.energystar.gov/index.cfm?fuseaction=find_a_product. The California Energy Commission maintains a database of all appliances meeting either federal efficiency standards or, where there are no federal efficiency standards, California's appliance efficiency standards. See http://www.appliances.energy.ca.gov/. The Electronic Product Environmental Assessment Tool (EPEAT) ranks computer products based on a set of environmental criteria, including energy efficiency. See http://www.epeat.net/AboutEPEAT.aspx. The nonprofit American Council for an Energy Efficient Economy maintains an Online Guide to Energy Efficient Commercial Equipment, available at http://www.aceee.org/ogeece/ch1_index.htm. Utilities offer many incentives for efficient appliances, lighting, heating and cooling. To search for available residential and commercial incentives, visit Flex Your Power's website at http://www.fypower.org/.

Use passive solar design, e.g., orient buildings and incorporate landscaping to maximize passive solar heating during cool seasons, minimize solar heat gain during hot seasons, and enhance natural ventilation. Design buildings to take advantage of sunlight.	See U.S. Department of Energy, Passive Solar Design (website) http://www.energysavers.gov/your_home/designing_remodeling/index.cfm/mytopic=10250. See also California Energy Commission, Consumer Energy Center, Passive Solar Design (website) http://www.consumerenergycenter.org/home/construction/solardesign/index.html. Lawrence Berkeley National Laboratories' Building Technologies Department is working to develop innovative building construction and design techniques. Information and publications on energy efficient buildings, including lighting, windows, and daylighting strategies, are available at the Department's website at http://btech.lbl.gov.
Install light colored "cool" roofs and cool pavements.	A white or light colored roof can reduce surface temperatures by up to 100 degrees Fahrenheit, which also reduces the heat transferred into the building below. This can reduce the building's cooling costs, save energy and reduce associated greenhouse gas emissions, and extend the life of the roof. Cool roofs can also reduce the temperature of surrounding areas, which can improve local air quality. See California Energy Commission, Consumer Energy Center, Cool Roofs (webpage) at http://www.consumerenergycenter.org/coolroof/. See also Lawrence Berkeley National Laboratories, Heat Island Group (webpage) at http://eetd.lbl.gov/HeatIsland/.
Install efficient lighting, (including LEDs) for traffic, street and other outdoor lighting.	LED lighting is substantially more energy efficient than conventional lighting and can save money. See http://www.energy.ca.gov/efficiency/partnership/case_studies/TechAsstCity.pdf (noting that installing LED traffic signals saved the City of Westlake about $34,000 per year). As of 2005, only about a quarter of California's cities and counties were using 100% LEDs in traffic signals. See California Energy Commission (CEC), Light Emitting Diode Traffic Signal Survey (2005) at p. 15, available at http://www.energy.ca.gov/2005publications/CEC 400 2005 003/CEC 400 2005 003.PDF. The California Energy Commission's Energy Partnership Program can help local governments take advantage of energy saving technology, including, but not limited to, LED traffic signals. See http://www.energy.ca.gov/efficiency/partnership/.
Reduce unnecessary outdoor lighting.	See California Energy Commission, Reduction of Outdoor Lighting (webpage) at http://www.energy.ca.gov/efficiency/lighting/outdoor_reduction.html.

Use automatic covers, efficient pumps and motors, and solar heating for pools and spas.	During the summer, a traditional backyard California pool can use enough energy to power an entire home for three months. Efficiency measures can substantially reduce this waste of energy and money. See California Energy Commission, Consumer Energy Center, Pools and Spas (webpage) at http://www.consumerenergycenter.org/home/outside/pools_spas.html. See also Sacramento Municipal Utilities District, Pool and Spa Efficiency Program (webpage) at http://www.smud.org/en/residential/saving-energy/Pages/poolspa.aspx.
Provide education on energy efficiency to residents, customers and/or tenants.	Many cities and counties provide energy efficiency education. See, for example, the City of Stockton's Energy Efficiency website at http://www.stocktongov.com/energysaving/index.cfm. See also "Green County San Bernardino," http://www.greencountysb.com at pp. 4-6. Businesses and development projects may also provide education. For example, a homeowners' association (HOA) could provide information to residents on energy-efficient mortgages and energy saving measures. See The Villas of Calvera Hills, Easy Energy Saving Tips to Help Save Electricity at http://www.thevillashoa.org/green/energy/. An HOA might also consider providing energy audits to its residents on a regular basis.

Renewable Energy and Energy Storage

Meet "reach" goals for building energy efficiency and renewable energy use.	A "zero net energy" building combines building energy efficiency and renewable energy generation so that, on an annual basis, any purchases of electricity or natural gas are offset by clean, renewable energy generation, either on-site or nearby. Both the California Energy Commission (CEC) and the California Public Utilities Commission (CPUC) have stated that residential buildings should be zero net energy by 2020, and commercial buildings by 2030. See CEC, 2009 Integrated Energy Policy Report (Dec. 2009) at p. 226, available at http://www.energy.ca.gov/2009publications/CEC-100-2009-003/CEC-100-2009-003-CMF.PDF; CPUC, Long Term Energy Efficiency Strategic Plan (Sept. 2008), available at http://www.cpuc.ca.gov/PUC/energy/Energy+Efficiency/eesp/.
Install solar, wind, and geothermal power systems and solar hot water heaters.	The California Public Utilities Commission (CPUC) approved the California Solar Initiative on January 12, 2006. The initiative creates a $3.3 billion, ten-year program to install solar panels on one million roofs in the State. Visit the one-stop GoSolar website at http://www.gosolarcalifornia.org/. As mitigation, a developer could, for example, agree to participate in the New Solar Homes program. See http://www.gosolarcalifornia.org/builders/index.html. The CPUC is in the process of establishing a program to provide solar water heating incentives under the California Solar Initiative. For more information, visit the CPUC's website at http://www.cpuc.ca.gov/puc/energy/solar/swh.htm. To search for available residential and commercial renewable energy incentives, visit Flex Your Power's website at http://www.fypower.org/.

Install solar panels on unused roof and ground space and over carports and parking areas.	In 2008 Southern California Edison (SCE) launched the nation's largest installation of photovoltaic power generation modules. The utility plans to cover 65 million square feet of unused commercial rooftops with 250 megawatts of solar technology – generating enough energy to meet the needs of approximately 162,000 homes. Learn more about SCE's Solar Rooftop Program at http://www.sce.com/solarleadership/solar-rooftop-program/general-faq.htm. In 2009, Walmart announced its commitment to expand the company's solar power program in California. The company plans to add solar panels on 10 to 20 additional Walmart facilities in the near term. These new systems will be in addition to the 18 solar arrays currently installed at Walmart facilities in California. See http://walmartstores.com/FactsNews/NewsRoom/9091.aspx. Alameda County has installed two solar tracking carports, each generating 250 kilowatts. By 2005, the County had installed eight photovoltaic systems totaling over 2.3 megawatts. The County is able to meet 6 percent of its electricity needs through solar power. See http://www.acgov.org/gsa/Alameda%20County%20-%20Solar%20Case%20Study.pdf. In 2007, California State University, Fresno installed at 1.1-megawatt photovoltaic (PV)-paneled parking installation. The University expects to save more than $13 million in avoided utility costs over the project's 30-year lifespan. http://www.fresnostatenews.com/2007/11/solarwrapup2.htm.
Where solar systems cannot feasibly be incorporated into the project at the outset, build "solar ready" structures.	U.S. Department of Energy, A Homebuilder's Guide to Going Solar (brochure) (2008), available at http://www.eere.energy.gov/solar/pdfs/43076.pdf.
Incorporate wind and solar energy systems into agricultural projects where appropriate.	Wind energy can be a valuable crop for farmers and ranchers. Wind turbines can generate energy to be used on-site, reducing electricity bills, or they can yield lease revenues (as much as $4000 per turbine per year). Wind turbines generally are compatible with rural land uses, since crops can be grown and livestock can be grazed up to the base of the turbine. See National Renewable Energy Laboratory, Wind Powering America Fact Sheet Series, Wind Energy Benefits, available at http://www.nrel.gov/docs/fy05osti/37602.pdf. Solar PV is not just for urban rooftops. For example, the Scott Brothers' dairy in San Jacinto, California, has installed a 55-kilowatt solar array on its commodity barn, with plans to do more in the coming years. See http://www.dairyherd.com/directories.asp?pgID=724&ed_id=8409 (additional California examples are included in article.)

Include energy storage where appropriate to optimize renewable energy generation systems and avoid peak energy use.	See National Renewable Energy Laboratory, Energy Storage Basics (webpage) at http://www.nrel.gov/learning/eds_energy_storage.html. California Energy Storage Alliance (webpage) at http://storagealliance.org/about.html. Storage is not just for large, utility scale projects, but can be part of smaller industrial, commercial and residential projects. For example, Ice Storage Air Conditioning (ISAC) systems, designed for residential and nonresidential buildings, produce ice at night and use it during peak periods for cooling. See California Energy Commission, Staff Report, Ice Storage Air Conditioners, Compliance Options Application (May 2006), available at http://www.energy.ca.gov/2006publications/CEC-400-2006-006/CEC-400-2006-006-SF.PDF.
Use on-site generated biogas, including methane, in appropriate applications.	At the Hilarides Dairy in Lindsay, California, an anaerobic-lagoon digester processes the run-off of nearly 10,000 cows, generating 226,000 cubic feet of biogas per day and enough fuel to run two heavy duty trucks. This has reduced the dairy's diesel consumption by 650 gallons a day, saving the dairy money and improving local air quality. See http://www.arb.ca.gov/newsrel/nr021109b.htm; see also Public Interest Energy Research Program, Dairy Power Production Program, Dairy Methane Digester System, 90-Day Evaluation Report, Eden Vale Dairy (Dec. 2006) at http://www.energy.ca.gov/2006publications/CEC 500 2006 083/CEC 500 2006 083.PDF. Landfill gas is a current and potential source of substantial energy in California. See Tom Frankiewicz, Program Manager, U.S. EPA Landfill Methane Outreach Program, Landfill Gas Energy Potential in California, available at http://www.energy.ca.gov/2009_energypolicy/documents/2009-04-21_workshop/presentations/05-SCS_Engineers_Presentation.pdf. There are many current and emerging technologies for converting landfill methane that would otherwise be released as a greenhouse gas into clean energy. See California Integrated Waste Management Board, Emerging Technologies, Landfill Gas-to-Energy (webpage) at http://www.ciwmb.ca.gov/LEACentral/TechServices/EmergingTech/default.htm.

Use combined heat and power (CHP) in appropriate applications.	Many commercial, industrial, and campus-type facilities (such as hospitals, universities and prisons) use fuel to produce steam and heat for their own operations and processes. Unless captured, much of this heat is wasted. CHP captures waste heat and re-uses it, e.g., for residential or commercial space heating or to generate electricity. See U.S. EPA, Catalog of CHP Technologies at http://www.epa.gov/chp/documents/catalog_of_%20chp_tech_entire.pdf and California Energy Commission, Distributed Energy Resource Guide, Combined Heat and Power (webpage) at http://www.energy.ca.gov/distgen/equipment/chp/chp.html. The average efficiency of fossil-fueled power plants in the United States is 33 percent. By using waste heat recovery technology, CHP systems typically achieve total system efficiencies of 60 to 80 percent. CHP can also substantially reduce emissions of carbon dioxide. http://www.epa.gov/chp/basic/efficiency.html. Currently, CHP in California has a capacity of over 9 million kilowatts. See list of California CHP facilities at http://www.eea-inc.com/chpdata/States/CA.html. The Waste Heat and Carbon Emissions Reduction Act (Assembly Bill 1613 (2007), amended by Assembly Bill 2791 (2008)) is designed to encourage the development of new CHP systems in California with a generating capacity of not more than 20 megawatts. Among other things, the Act requires the California Public Utilities Commission to establish (1) a standard tariff allowing CHP generators to sell electricity for delivery to the grid and (2) a "pay as you save" pilot program requiring electricity corporations to finance the installation of qualifying CHP systems by nonprofit and government entities. For more information, see http://www.energy.ca.gov/wasteheat/.

Water Conservation and Efficiency

Incorporate water-reducing features into building and landscape design.	According to the California Energy Commission, water-related energy use – which includes conveyance, storage, treatment, distribution, wastewater collection, treatment, and discharge – consumes about 19 percent of the State's electricity, 30 percent of its natural gas, and 88 billion gallons of diesel fuel every year. See http://www.energy.ca.gov/2007publications/CEC 999 2007 008/CEC 999 2007 008.PDF. Reducing water use and improving water efficiency can help reduce energy use and greenhouse gas emissions.
Create water-efficient landscapes.	The California Department of Water Resources' updated Model Water Efficient Landscape Ordinance (Sept. 2009) is available at http://www.water.ca.gov/wateruseefficiency/landscapeordinance/technical.cfm. A landscape can be designed from the beginning to use little or no water, and to generate little or no waste. See California Integrated Waste Management Board, Xeriscaping (webpage) at http://www.ciwmb.ca.gov/organics/Xeriscaping/.

Install water-efficient irrigation systems and devices, such as soil moisture-based irrigation controls and use water-efficient irrigation methods.	U.S. Department of Energy, Best Management Practice: Water-Efficient Irrigation (webpage) at http://www1.eere.energy.gov/femp/program/waterefficiency_bmp5.html. California Department of Water Resources, Landscape Water Use Efficiency (webpage) at http://www.water.ca.gov/wateruseefficiency/landscape/. Pacific Institute, More with Less: Agricultural Water Conservation and Efficiency in California (2008), available at http://www.pacinst.org/reports/more_with_less_delta/index.htm.
Make effective use of graywater. (Graywater is untreated household waste water from bathtubs, showers, bathroom wash basins, and water from clothes washing machines. Graywater to be used for landscape irrigation.)	California Building Standards Commission, 2008 California Green Building Standards Code, Section 604, pp. 31-32, available at http://www.documents.dgs.ca.gov/bsc/2009/part11_2008_calgreen_code.pdf. California Department of Water Resources, Dual Plumbing Code (webpage) at http://www.water.ca.gov/recycling/DualPlumbingCode/. See also Ahwahnee Water Principles, Principle 6, at http://www.lgc.org/ahwahnee/h2o_principles.html. The Ahwahnee Water Principles have been adopted by City of Willits, Town of Windsor, Menlo Park, Morgan Hill, Palo Alto, Petaluma, Port Hueneme, Richmond, Rohnert Park, Rolling Hills Estates, San Luis Obispo, Santa Paula, Santa Rosa, City of Sunnyvale, City of Ukiah, Ventura, Marin County, Marin Municipal Water District, and Ventura County.
Implement low-impact development practices that maintain the existing hydrology of the site to manage storm water and protect the environment.	Retaining storm water runoff on-site can drastically reduce the need for energy-intensive imported water at the site. See U.S. EPA, Low Impact Development (webpage) at http://www.epa.gov/nps/lid/. Office of Environmental Health Hazard Assessment and the California Water and Land Use Partnership, Low Impact Development at http://www.coastal.ca.gov/nps/lid-factsheet.pdf.
Devise a comprehensive water conservation strategy appropriate for the project and location.	The strategy may include many of the specific items listed above, plus other innovative measures that are appropriate to the specific project.
Design buildings to be water-efficient. Install water-efficient fixtures and appliances.	Department of General Services, Best Practices Manual, Water-Efficient Fixtures and Appliances (website) at http://www.green.ca.gov/EPP/building/SaveH2O.htm. Many ENERGY STAR products have achieved their certification because of water efficiency. See California Energy Commission's database, available at http://www.appliances.energy.ca.gov/.

Offset water demand from new projects so that there is no net increase in water use.	For example, the City of Lompoc has a policy requiring new development to offset new water demand with savings from existing water users. See http://www.cityoflompoc.com/utilities/pdf/2005_uwmp_final.pdf at p. 29.
Provide education about water conservation and available programs and incentives.	See, for example, the City of Santa Cruz, Water Conservation Office at http://www.ci.santa-cruz.ca.us/index.aspx?page=395; Santa Clara Valley Water District, Water Conservation at http://www.valleywater.org/conservation/index.shtm; and Metropolitan Water District and the Family of Southern California Water Agencies, Be Water Wise at http://www.bewaterwise.com. Private projects may provide or fund similar education.

Solid Waste Measures

Reuse and recycle construction and demolition waste (including, but not limited to, soil, vegetation, concrete, lumber, metal, and cardboard).	Construction and demolition materials account for almost 22 percent of the waste stream in California. Reusing and recycling these materials not only conserves natural resources and energy, but can also save money. For a list of best practices and other resources, see California Integrated Waste Management Board, Construction and Demolition Debris Recycling (webpage) at http://www.ciwmb.ca.gov/condemo/.
Integrate reuse and recycling into residential industrial, institutional and commercial projects.	Tips on developing a successful recycling program, and opportunities for cost-effective recycling, are available on the California Integrated Waste Management Board's Zero Waste California website. See http://zerowaste.ca.gov/. The Institute for Local Government's Waste Reduction & Recycling webpage contains examples of "best practices" for reducing greenhouse gas emissions, organized around waste reduction and recycling goals and additional examples and resources. See http://www.ca-ilg.org/wastereduction.
Provide easy and convenient recycling opportunities for residents, the public, and tenant businesses.	Tips on developing a successful recycling program, and opportunities for cost effective recycling, are available on the California Integrated Waste Management Board's Zero Waste California website. See http://zerowaste.ca.gov/.
Provide education and publicity about reducing waste and available recycling services.	Many cities and counties provide information on waste reduction and recycling. See, for example, the Butte County Guide to Recycling at http://www.recyclebutte.net. The California Integrated Waste Management Board's website contains numerous publications on recycling and waste reduction that may be helpful in devising an education project. See http://www.ciwmb.ca.gov/Publications/default.asp?cat=13. Private projects may also provide waste and recycling education directly, or fund education.

Land Use Measures

Ensure consistency with "smart growth" principles – mixed-use, infill, and higher density projects that provide alternatives to individual vehicle travel and promote the efficient delivery of services and goods.	U.S. EPA maintains an extensive Smart Growth webpage with links to examples, literature and technical assistance, and financial resources. See http://www.epa.gov/smartgrowth/index.htm. The National Oceanic and Atmospheric Administration's webpage provides smart growth recommendations for communities located near water. See Coastal & Waterfront Smart Growth (webpage) at http://coastalsmartgrowth.noaa.gov/. The webpage includes case studies from California. The California Energy Commission has recognized the important role that land use can play in meeting our greenhouse gas and energy efficiency goals. The agency's website, Smart Growth & Land Use Planning, contains useful information and links to relevant studies, reports, and other resources. See http://www.energy.ca.gov/landuse/. The Metropolitan Transportation Commission's webpage, Smart Growth / Transportation for Livable Communities, includes resources that may be useful to communities in the San Francisco Bay Area and beyond. See http://www.mtc.ca.gov/planning/smart_growth/. The Sacramento Area Council of Governments (SACOG) has published examples of smart growth in action in its region. See Examples from the Sacramento Region of the Seven Principles of Smart Growth / Better Ways to Grow, available at http://www.sacog.org/regionalfunding/betterways.pdf.
Meet recognized "smart growth" benchmarks.	For example, the LEED for Neighborhood Development (LEED-ND) rating system integrates the principles of smart growth, urbanism and green building into the first national system for neighborhood design. LEED-ND is a collaboration among the U.S. Green Building Council, Congress for the New Urbanism, and the Natural Resources Defense Council. For more information, see http://www.usgbc.org/DisplayPage.aspx?CMSPageID=148.
Educate the public about the many benefits of well-designed, higher density development.	See, for example, U.S. EPA, Growing Smarter, Living Healthier: A Guide to Smart Growth and Active Aging (webpage), discussing how compact, walkable communities can provide benefits to seniors. See http://www.epa.gov/aging/bhc/guide/index.html. U.S. EPA, Environmental Benefits of Smart Growth (webpage) at http://www.epa.gov/dced/topics/eb.htm (noting local air and water quality improvements). Centers for Disease Control and Prevention (CDC), Designing and Building Healthy Places (webpage), at http://www.cdc.gov/healthyplaces/. The CDC's website discusses the links between walkable communities and public health and includes numerous links to educational materials. California Department of Housing and Community Development, Myths and Facts About Affordable and High Density Housing (2002), available at http://www.hcd.ca.gov/hpd/mythsnfacts.pdf.

Incorporate public transit into the project's design.	Federal Transit Administration, Transit-Oriented Development (TOD) (webpage) at http://www.fta.dot.gov/planning/planning_environment_6932.html (describing the benefits of TOD as "social, environmental, and fiscal.") California Department of Transportation (Caltrans), Statewide Transit-Oriented Development Study: Factors for Success in California (2002), available at http://transitorienteddevelopment.dot.ca.gov/miscellaneous/StatewideTOD.htm Caltrans, California Transit-Oriented Development Searchable Database (includes detailed information on numerous TODs), available at http://transitorienteddevelopment.dot.ca.gov/miscellaneous/NewHome.jsp. California Department of Housing and Community Development, Transit Oriented Development (TOD) Resources (Aug. 2009), available at http://www.hcd.ca.gov/hpd/tod.pdf.
Preserve and create open space and parks. Preserve existing trees, and plant replacement trees at a set ratio.	U.S. EPA, Smart Growth and Open Space Conservation (webpage) at http://www.epa.gov/dced/openspace.htm.
Develop "brownfields" and other underused or defunct properties near existing public transportation and jobs.	U.S. EPA, Smart Growth and Brownfields (webpage) at http://www.epa.gov/dced/brownfields.htm. For example, as set forth in the Local Government Commission's case study, the Town of Hercules, California reclaimed a 426-acre brownfield site, transforming it into a transit-friendly, walkable neighborhood. See http://www.lgc.org/freepub/docs/community_design/fact_sheets/er_case_studies.pdf. For financial resources that can assist in brownfield development, see Center for Creative Land Recycling, Financial Resources for California Brownfields (July 2008), available at http://www.cclr.org/media/publications/8-Financial_Resources_2008.pdf.
Include pedestrian and bicycle facilities within projects and ensure that existing non-motorized routes are maintained and enhanced.	See U.S. Department of Transportation, Federal Highway Administration, Bicycle and Pedestrian Program (webpage) at http://www.fhwa.dot.gov/environment/bikeped/. Caltrans, Pedestrian and Bicycle Facilities in California / A Technical Reference and Technology Transfer Synthesis for Caltrans Planners and Engineers (July 2005), available at http://www.dot.ca.gov/hq/traffops/survey/pedestrian/TR_MAY0405.pdf. This reference includes standard and innovative practices for pedestrian facilities and traffic calming.

Transportation and Motor Vehicles

Meet an identified transportation-related benchmark.	A logical benchmark might be related to vehicles miles traveled (VMT), e.g., average VMT per capita, per household, or per employee. As the California Energy Commission has noted, VMT by California residents increased "a rate of more than 3 percent a year between 1975 and 2004, markedly faster than the population growth rate over the same period, which was less than 2 percent. This increase in VMT correlates to an increase in petroleum use and GHG production and has led to the transportation sector being responsible for 41 percent of the state's GHG emissions in 2004." CEC, The Role of Land Use in Meeting California's Energy and Climate Change Goals (Aug. 2007) at p. 9, available at http://www.energy.ca.gov/2007publications/CEC-600-2007-008/CEC-600-2007-008-SF.PDF. Even with regulations designed to increase vehicle efficiency and lower the carbon content of fuel, "reduced VMT growth will be required to meet GHG reductions goals." *Id.* at p. 18.
Adopt a comprehensive parking policy that discourages private vehicle use and encourages the use of alternative transportation.	For example, reduce parking for private vehicles while increasing options for alternative transportation; eliminate minimum parking requirements for new buildings; "unbundle" parking (require that parking is paid for separately and is not included in rent for residential or commercial space); and set appropriate pricing for parking. See U.S. EPA, Parking Spaces / Community Places, Finding the Balance Through Smart Growth Solutions (Jan. 2006), available at http://www.epa.gov/dced/pdf/EPAParkingSpaces06.pdf. Reforming Parking Policies to Support Smart Growth, Metropolitan Transportation Commission (June 2007) at http://www.mtc.ca.gov/planning/smart_growth/parking_seminar/Toolbox_Handbook.pdf. See also the City of Ventura's Downtown Parking and Mobility Plan, available at http://www.cityofventura.net/community_development/resources/mobility_parking_plan.pdf, and Ventura's Downtown Parking Management Program, available at http://www.ci.ventura.ca.us/depts/comm_dev/downtownplan/chapters.asp.
Build or fund a major transit stop within or near the development.	"'Major transit stop' means a site containing an existing rail transit station, a ferry terminal served by either a bus or rail transit service, or the intersection of two or more major bus routes with a frequency of service interval of 15 minutes or less during the morning and afternoon peak commute periods." (Pub. Res. Code, § 21064.3.) Transit Oriented Development (TOD) is a moderate to higher density development located within an easy walk of a major transit stop. http://transitorienteddevelopment.dot.ca.gov/miscellaneous/NewWhatisTOD.htm. By building or funding a major transit stop, an otherwise ordinary development can become a TOD.

Provide public transit incentives such as free or low-cost monthly transit passes to employees, or free ride areas to residents and customers.	See U.S. Department of Transportation and U.S. EPA, Commuter Choice Primer / An Employer's Guide to Implementing Effective Commuter Choice Programs, available at http://www.its.dot.gov/JPODOCS/REPTS_PR/13669.html. The Emery Go Round shuttle is a private transportation service funded by commercial property owners in the citywide transportation business improvement district. The shuttle links a local shopping district to a Bay Area Rapid Transit stop. See http://www.emerygoround.com/. Seattle, Washington maintains a public transportation "ride free" zone in its downtown from 6:00 a.m. to 7:00 p.m. daily. See http://transit.metrokc.gov/tops/accessible/paccessible_map.html#fare.
Promote "least polluting" ways to connect people and goods to their destinations.	Promoting "least polluting" methods of moving people and goods is part of a larger, integrated "sustainable streets" strategy now being explored at U.C. Davis's Sustainable Transportation Center. Resources and links are available at the Center's website, http://stc.ucdavis.edu/outreach/ssp.php.
Incorporate bicycle lanes, routes and facilities into street systems, new subdivisions, and large developments.	Bicycling can have a profound impact on transportation choices and air pollution reduction. The City of Davis has the highest rate of bicycling in the nation. Among its 64,000 residents, 17 percent travel to work by bicycle and 41 percent consider the bicycle their primary mode of transportation. See Air Resources Board, Bicycle Awareness Program, Bicycle Fact Sheet, available at http://www.arb.ca.gov/planning/tsaq/bicycle/factsht.htm. For recommendations on best practices, see the many resources listed at the U.S. Department of Transportation, Federal Highway Administration's Bicycle and Pedestrian website at http://www.fhwa.dot.gov/environment/bikeped/publications.htm. See also Caltrans Division of Research and Innovation, Designing Highway Facilities To Encourage Walking, Biking and Transit (Preliminary Investigation) (March 2009), available at http://www.dot.ca.gov/research/researchreports/preliminary_investigations/docs/pi-design_for_walking_%20biking_and_transit%20final.pdf.
Require amenities for non-motorized transportation, such as secure and convenient bicycle parking.	According to local and national surveys of potential bicycle commuters, secure bicycle parking and workplace changing facilities are important complements to safe and convenient routes of travel. See Air Resources Board, Bicycle Awareness Program, Bicycle Fact Sheet, available at http://www.arb.ca.gov/planning/tsaq/bicycle/factsht.htm.

Ensure that the project enhances, and does not disrupt or create barriers to, non-motorized transportation.	See, e.g., U.S. EPA's list of transit-related "smart growth" publications at http://www.epa.gov/dced/publications.htm#air, including Pedestrian and Transit-Friendly Design: A Primer for Smart Growth (1999), available at www.epa.gov/dced/pdf/ptfd_primer.pdf. See also Toolkit for Improving Walkability in Alameda County, available at http://www.acta2002.com/ped toolkit/ped_toolkit_print.pdf. Pursuant to the California Complete Streets Act of 2008 (AB 1358, Gov. Code, §§ 65040.2 and 65302), commencing January 1, 2011, upon any substantive revision of the circulation element of the general plan, a city or county will be required to modify the circulation element to plan for a balanced, multimodal transportation network that meets the needs of all users.
Connect parks and open space through shared pedestrian/bike paths and trails to encourage walking and bicycling. Create bicycle lanes and walking paths directed to the location of schools, parks and other destination points.	Walk Score ranks the "walkability" of neighborhoods in the largest 40 U.S. cities, including seven California cities. Scores are based on the distance to nearby amenities. Explore Walk Score at http://www.walkscore.com/. In many markets, homes in walkable neighborhoods are worth more than similar properties where walking is more difficult. See Hoak, *Walk appeal / Homes in walkable neighborhoods sell for more: study*, Wall Street Journal (Aug. 18, 2009), available at http://www.marketwatch.com/story/homes-in-walkable-neighborhoods-sell-for-more-2009-08-18. By creating walkable neighborhoods with more transportation choices, Californians could save $31 million and cut greenhouse gas emissions by 34 percent, according to a study released by Transform, a coalition of unions and nonprofits. See Windfall for All / How Connected, Convenient Neighborhoods Can Protect Our Climate and Safeguard California's Economy (Nov. 2009), available at http://transformca.org/windfall-for-all#download-report.
Work with the school districts to improve pedestrian and bike access to schools and to restore or expand school bus service using lower-emitting vehicles.	In some communities, twenty to twenty-five percent of morning traffic is due to parents driving their children to school. Increased traffic congestion around schools in turn prompts even more parents to drive their children to school. Programs to create safe routes to schools can break this harmful cycle. See California Department of Public Health, Safe Routes to School (webpage) and associated links at http://www.cdph.ca.gov/HealthInfo/injviosaf/Pages/SafeRoutestoSchool.aspx. See also U.S. EPA, Smart Growth and Schools (webpage), available at http://www.epa.gov/dced/schools.htm. California Center for Physical Activity, California Walk to School (website) at http://www.cawalktoschool.com Regular school bus service (using lower-emitting buses) for children who cannot bike or walk to school could substantially reduce private vehicle congestion and air pollution around schools. See Air Resources Board, Lower Emissions School Bus Program (webpage) at http://www.arb.ca.gov/msprog/schoolbus/schoolbus.htm.

Institute teleconferencing, telecommute and/or flexible work hour programs to reduce unnecessary employee transportation.	There are numerous sites on the web with resources for employers seeking to establish telework or flexible work programs. These include U.S. EPA's Mobility Management Strategies: Commuter Programs website at http://www.epa.gov/otaq/stateresources/rellinks/mms_commprograms.htm; and Telework, the federal government's telework website, at http://www.telework.gov/. Through a continuing FlexWork Implementation Program, the Traffic Solutions division of the Santa Barbara County Association of Governments sponsors flexwork consulting, training and implementation services to a limited number of Santa Barbara County organizations that want to create or expand flexwork programs for the benefit of their organizations, employees and the community. See http://www.flexworksb.com/read_more_about_the_fSBp.html. Other local government entities provide similar services.
Provide information on alternative transportation options for consumers, residents, tenants and employees to reduce transportation-related emissions.	Many types of projects may provide opportunities for delivering more tailored transportation information. For example, a homeowner's association could provide information on its website, or an employer might create a Transportation Coordinator position as part of a larger Employee Commute Reduction Program. See, e.g., South Coast Air Quality Management District, Transportation Coordinator training, at http://www.aqmd.gov/trans/traing.html.
Educate consumers, residents, tenants and the public about options for reducing motor vehicle-related greenhouse gas emissions. Include information on trip reduction; trip linking; vehicle performance and efficiency (e.g., keeping tires inflated); and low or zero-emission vehicles.	See, for example U.S. EPA, SmartWay Transport Partnership: Innovative Carrier Strategies (webpage) at http://www.epa.gov/smartway/transport/what-smartway/carrier-strategies.htm. This webpage includes recommendations for actions that truck and rail fleets can take to make ground freight more efficient and cleaner. The Air Resources Board's Drive Clean website is a resource for car buyers to find clean and efficient vehicles. The web site is designed to educate Californians that pollution levels range greatly between vehicles. See http://www.driveclean.ca.gov/. The Oregon Department of Transportation and other public and private partners launched the Drive Less/Save More campaign. The comprehensive website contains fact sheets and educational materials to help people drive more efficiently. See http://www.drivelesssavemore.com/.
Purchase, or create incentives for purchasing, low or zero-emission vehicles.	See Air Resources Board, Low-Emission Vehicle Program (webpage) at http://www.arb.ca.gov/msprog/levprog/levprog.htm. Air Resource Board, Zero Emission Vehicle Program (webpage) at http://www.arb.ca.gov/msprog/zevprog/zevprog.htm. All new cars sold in California are now required to display an Environmental Performance (EP) Label, which scores a vehicle's global warming and smog emissions from 1 (dirtiest) to 10 (cleanest). To search and compare vehicle EP Labels, visit www.DriveClean.ca.gov.

Create a ride sharing program. Promote existing ride sharing programs e.g., by designating a certain percentage of parking spaces for ride sharing vehicles, designating adequate passenger loading and unloading for ride sharing vehicles, and providing a web site or message board for coordinating rides.	For example, the 511 Regional Rideshare Program is operated by the Metropolitan Transportation Commission (MTC) and is funded by grants from the Federal Highway Administration, U.S. Department of Transportation, the Metropolitan Transportation Commission, the Bay Area Air Quality Management District and county congestion management agencies. For more information, see http://rideshare.511.org/. As another example, San Bernardino Associated Governments works directly with large and small employers, as well as providing support to commuters who wish to share rides or use alternative forms of transportation. See http://www.sanbag.ca.gov/commuter/rideshare.html. Valleyrides.com is a ridesharing resource available to anyone commuting to and from Fresno and Tulare Counties and surrounding communities. See http://www.valleyrides.com/. There are many other similar websites throughout the state.
Create or accommodate car sharing programs, e.g., provide parking spaces for car share vehicles at convenient locations accessible by public transportation.	There are many existing car sharing companies in California. These include City CarShare (San Francisco Bay Area), see http://www.citycarshare.org/; and Zipcar, see http://www.zipcar.com/. Car sharing programs are being successfully used on many California campuses.
Provide a vanpool for employees.	Many local Transportation Management Agencies can assist in forming vanpools. See, for example, Sacramento Transportation Management Association, Check out Vanpooling (webpage) at http://www.sacramento-tma.org/vanpool.html.
Create local "light vehicle" networks, such as neighborhood electric vehicle systems.	See California Energy Commission, Consumer Energy Center, Urban Options - Neighborhood Electric Vehicles (NEVs) (webpage) at http://www.consumerenergycenter.org/transportation/urban_options/nev.html. The City of Lincoln has an innovative NEV program. See http://www.lincolnev.com/index.html.
Enforce and follow limits idling time for commercial vehicles, including delivery and construction vehicles.	Under existing law, diesel-fueled motor vehicles with a gross vehicle weight rating greater than 10,000 pounds are prohibited from idling for more than 5 minutes at any location. The minimum penalty for an idling violation is now $300 per violation. See http://www.arb.ca.gov/enf/complaints/idling_cv.htm.
Provide the necessary facilities and infrastructure to encourage the use of low or zero-emission vehicles.	For a list of existing alternative fuel stations in California, visit http://www.cleancarmaps.com/. See, e.g., Baker, *Charging-station network built along 101*, S.F. Chron. (9/23/09), available at http://articles.sfgate.com/2009-09-23/news/17207424_1_recharging-solar-array-tesla-motors.

Agriculture and Forestry (additional strategies noted above)

Require best management practices in agriculture and animal operations to reduce emissions, conserve energy and water, and utilize alternative energy sources, including biogas, wind and solar.	Air Resources Board (ARB), Economic Sectors Portal, Agriculture (webpage) at http://www.arb.ca.gov/cc/ghgsectors/ghgsectors.htm. ARB's webpage includes information on emissions from manure management, nitrogen fertilizer, agricultural offroad equipment, and agricultural engines. "A full 90% of an agricultural business' electricity bill is likely associated with water use. In addition, the 8 million acres in California devoted to crops consume 80% of the total water pumped in the state." See Flex Your Power, Agricultural Sector (webpage) at http://www.fypower.org/agri/. Flex Your Power, Best Practice Guide / Food and Beverage Growers and Processors, available at http://www.fypower.org/bpg/index.html?b=food_and_bev. Antle et al., Pew Center on Global Climate Change, Agriculture's Role in Greenhouse Gas Mitigation (2006), available at http://www.pewclimate.org/docUploads/Agriculture's%20Role%20in%20GHG%20Mitigation.pdf.
Preserve forested areas, agricultural lands, wildlife habitat and corridors, wetlands, watersheds, groundwater recharge areas and other open space that provide carbon sequestration benefits.	"There are three general means by which agricultural and forestry practices can reduce greenhouse gases: (1) avoiding emissions by maintaining existing carbon storage in trees and soils; (2) increasing carbon storage by, e.g., tree planting, conversion from conventional to conservation tillage practices on agricultural lands; (3) substituting bio-based fuels and products for fossil fuels, such as coal and oil, and energy-intensive products that generate greater quantities of CO2 when used." U.S. EPA, Carbon Sequestration in Agriculture and Forestry, Frequently Asked Questions (webpage) at http://www.epa.gov/sequestration/faq.html. Air Resources Board, Economic Sectors Portal, Forestry (webpage) at http://www.arb.ca.gov/cc/ghgsectors/ghgsectors.htm.
Protect existing trees and encourage the planting of new trees. Adopt a tree protection and replacement ordinance.	Tree preservation and planting is not just for rural areas of the state; suburban and urban forests can also serve as carbon sinks. See Cal Fire, Urban and Community Forestry (webpage) at http://www.fire.ca.gov/resource_mgt/resource_mgt_urbanforestry.php.

Off-Site Mitigation

If, after analyzing and requiring all reasonable and feasible on-site mitigation measures for avoiding or reducing greenhouse gas-related impacts, the lead agency determines that additional mitigation is required, the agency may consider additional off-site mitigation. The project proponent could, for example, fund off-site mitigation projects that will reduce carbon emissions, conduct an audit of its other existing operations and agree to retrofit, or purchase verifiable carbon "credits" from another entity that will undertake mitigation.

The topic of off-site mitigation can be complicated. A full discussion is outside the scope of this summary document. Issues that the lead agency should consider include:

- The location of the off-site mitigation. (If the off-site mitigation is far from the project, any additional, non-climate related co-benefits of the mitigation may be lost to the local community.)

- Whether the emissions reductions from off-site mitigation can be quantified and verified. (The California Registry has developed a number of protocols for calculating, reporting and verifying greenhouse gas emissions. Currently, industry-specific protocols are available for the cement sector, power/utility sector, forest sector and local government operations. For more information, visit the California Registry's website at http://www.climateregistry.org/.)

- Whether the mitigation ratio should be greater than 1:1 to reflect any uncertainty about the effectiveness of the off-site mitigation.

Offsite mitigation measures that could be funded through mitigation fees include, but are not limited to, the following:

- Energy efficiency audits of existing buildings.

- Energy efficiency upgrades to existing buildings not otherwise required by law, including heating, ventilation, air conditioning, lighting, water heating equipment, insulation and weatherization (perhaps targeted to specific communities, such as low-income or senior residents).

- Programs to encourage the purchase and use of energy efficient vehicles, appliances, equipment and lighting.

- Programs that create incentives to replace or retire polluting vehicles and engines.

- Programs to expand the use of renewable energy and energy storage.

- Preservation and/or enhancement of existing natural areas (e.g., forested areas, agricultural lands, wildlife habitat and corridors, wetlands, watersheds, and groundwater recharge areas) that provide carbon sequestration benefits.

- Improvement and expansion of public transit and low- and zero-carbon transportation alternatives.

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August 30, 2012

Brooke O'Hanley
Lozeau | Drury LLP
410 12th Street, Suite 250
Oakland, CA 94607

Subject: Comments on the Prologis Eucalyptus Industrial Park Project, Riverside County, California

Dear Ms. O'Hanley:

We have reviewed the July 2012 Draft Environmental Impact Report (DEIR) for Prologis Eucalyptus Industrial Park Project ("Project"). The Project would construct six buildings encompassing approximately 2.3 million square feet (or 53 acres) of warehouse space. The Project site would be located on a 123-acre lot in the eastern portion of the City of Moreno Valley in Riverside County, California.

We have reviewed the DEIR for issues associated with hazards and hazardous materials, greenhouse gases, air quality, and cumulative impacts. Project construction will result in potentially significant impacts to construction workers and nearby residents that are not adequately disclosed in the DEIR. A revised DEIR needs to be prepared to fully disclose, evaluate, and mitigate these impacts.

Hazards and Hazardous Materials:

Construction workers and nearby residents may be at risk during construction from failure to disclose baseline soil conditions at the Project site.

Residual pesticides in soil may pose health risks to workers and nearby residents

Currently, 57 acres of the Project site are used to grow grapefruit and 36 acres of the Project site are used for hay and alfalfa production (DEIR, p. 4.2-1). The DEIR and supporting documents do not provide any specific details on the types of pesticides that have been used on the Project site in association with

these agricultural operations. Our review has shown known and potential pesticide use at the Project site as follows:

- Data available online from the California Department of Pesticide Regulation show that 2,4-D, 2-Ethylhexyl Ester was used on the Project site.[1,2] Occupational exposure to 2,4-D, 2-Ethylhexyl Ester can occur via inhalation or dermal contact and can result in skin irritation, respiratory failure, hyperventilation, and pulmonary enemas.[3]
- Organochlorine pesticides DDE and DDT were detected in soil samples collected at the Project site[4], indicating past use. Use of organochlorine pesticides in the area is common: review of the Department of Toxic Substances Control's (DTSC) Envirostor database shows that the surrounding lands have been surveyed for organochlorine pesticides, DDE and DDT.[5] These pesticides can persist in soil for hundreds of years despite being banned in the 1970s.[6] Exposure to DDT can result in headaches, nausea, and convulsions.[7] The U.S. EPA identifies DDT and DDE as probable human carcinogens.[8]
- The EPA states that soils at fruit orchards, such as the grapefruit orchard on the Project site, may contain high levels of arsenic from application as a pesticide.[9] Another chemical used on fruit orchards is lead arsenate, a very persistent pesticide.[10] Arsenic is a known human carcinogen and even short-term inhalation of arsenic dust can cause gastrointestinal effects [11] while lead is known to cause neurotoxicological effects.[12]

Pesticide use at the Project site was not disclosed in the DEIR and the detection of pesticide residuals in soil were not described in the Hazards and Hazardous Materials section.

Failure to disclose the presence of pesticide residuals in Project site soils may pose significant health risks to construction workers. Construction of the Project requires grading and the disturbance of subsurface soils and removal of citrus groves (DEIR, p. 4.7-21). During earthmoving activities, construction workers will be exposed, via inhalation of dust and dermal contact, to Project site soils which may contain harmful levels of pesticide residuals associated with agricultural activities on the site. To protect worker safety, Project site soils must be sampled for pesticides. Sampling results should be

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[1] ftp://cdpr.ca.gov/pub/outgoing/calpip/26814174623515_120824104149.html
[2] ftp://cdpr.ca.gov/pub/outgoing/calpip/26814174623515_120824104217.html
[3] http://toxnet.nlm.nih.gov/cgi-bin/sis/search/a?dbs+hsdb:@term+@DOCNO+7309
[4] Phase I Environmental Site Assessment, 84 acres. Near Intersection of Pittit Street and Highway 60, Moreno Valley, California, p. 9 and Phase I Environmental Site Assessment, 37 acres. Near Intersection of Pittit Street and Highway 60, Moreno Valley, California, p. 8
[5] http://www.envirostor.dtsc.ca.gov/public/profile_report.asp?global_id=60000825 and http://www.envirostor.dtsc.ca.gov/public/profile_report.asp?global_id=60000931
[6] http://www.atsdr.cdc.gov/toxprofiles/tp35.pdf, p. 3
[7] http://www.epa.gov/ttn/atw/hlthef/dde.html
[8] http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=80&tid=20
[9] http://water.epa.gov/drink/info/well/health.cfm
[10] http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1551991/
[11] http://www.epa.gov/ttn/atw/hlthef/arsenic.html
[12] http://www.epa.gov/lead/pubs/learn-about-lead.html#effects

compared to health-protective regulatory screening levels such as U.S. EPA Regional Screening Levels[13] and California Human Health Screening Levels.[14]

Soil sampling results should also be evaluated for the protection of nearby residents, located 50 feet from the southern boundary and 200 feet from the northern boundary of the Project site (DEIR, p. 4.3-6). Inhalation of pesticides has been linked to asthma in recent research.[15,16] A report prepared by the California Department of Health identifies pesticides as an asthma trigger.[17] Offsite receptors, including any children living in the neighboring residences, may be exposed to pesticide residuals via dust generated during Project construction.

Construction activities, such as grading and excavation of soils, may generate dust that contains pesticides in concentrations that are harmful to the health of workers and nearby residents and which may act as an asthma trigger. Project site soils should be sampled and results should be compared to human health screening levels. A revised DEIR should be prepared to disclose the results of sampling and include any necessary mitigation to reduce impacts to the health of construction workers and nearby residents.

Phase I ESAs completed for the Project site are outdated and inadequate

The DEIR relies on the findings from two Phase I Environmental Site Assessments (ESAs) that were completed in October and November 2003, nearly nine years ago. The Phase I ESAs surveyed 121 acres of the 123-acre Project site. The Applicant purchased the Project site more than five years ago.[18] A Phase I ESA, according to the U.S. EPA, must be conducted within one year of the acquisition of the property and on-site visual inspections must be completed within 180 days prior to acquiring ownership of the property.[19]

Because the Phase I ESAs are dated and omit two acres of the Project site, they cannot be used to evaluate conditions that are potentially hazardous to construction workers and future site personnel. Therefore, the DEIR's analysis of the Project site based on these Phase I ESAs is inadequate.

Review of Google Earth images shows that the Project site has been used for ongoing agricultural operations since the Phase I ESAs were completed in 2003. Limited pesticide sampling was conducted during the Phase I ESAs (a total of 8 soil samples for a 123-acre Project site) but because the samples were collected nine years ago and because they do not reflect continued agricultural use, the results are reflective of current site conditions.

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[13] http://www.epa.gov/region9/superfund/prg/
[14] http://www.calepa.ca.gov/brownfields/documents/2005/CHHSLsGuide.pdf
[15] http://extension.psu.edu/ipm/resources/urbanphilly/partnerships/handouts/asthma-pests.pdf
[16] http://www.ncbi.nlm.nih.gov/pubmed/21368619
[17] http://www.cdph.ca.gov/programs/caphi/Documents/AsthmaStrategicPlan.5-5-08.pdf, p. 22
[18] http://www.pe.com/local-news/riverside-county/moreno-valley/moreno-valley-headlines-index/20120726-moreno-valley-officials-seek-comments-on-prologis-project.ece
[19] http://www.epa.gov/brownfields/aai/aaicerclafs.pdf

The Phase I ESAs cover 121 acres of the 123-acre Project site. We have created a map to show the areas of the Project site surveyed by the two 2003 Phase I ESAs and the boundaries for the current Project site (Attachment A). As the map shows, not all areas of the current Project site were included in the 2003 Phase I ESAs' site assessments.

The Phase I ESAs are outdated and do not cover the entire Project site; therefore, they cannot be used to define baseline conditions for the DEIR's Hazards and Hazardous Materials section. A revised DEIR should be prepared to include a new Phase I ESA that evaluates current Project site conditions.

Status of an underground storage tank is uncertain

A 13,400 gallon underground storage tank (UST), abandoned in the 1950s, was removed from the Project site in 2004 (Appendix F, p. 3/191). The Phase I ESA recommended an additional investigation to be conducted in the area of the former UST (Appendix F, p. 10/191). Accordingly, a permit for removal of the UST was submitted to the Riverside County's Department of Environmental Health in December 2003 and soil samples around the area of the UST were analyzed in 2004. However, the DEIR and supporting documents did not include any documentation that that the UST was properly closed by the Riverside County Department of Environmental Health. If the UST removal was not approved, an Underground Storage Tank Closure Application and Permit[20], per the Riverside County Department of Environmental Health Guidelines,[21] must be submitted. A revised DEIR should be prepared to disclose whether closure was granted by the Riverside County Department of Environmental Health.

Greenhouse Gas Emissions:

The Project's operational greenhouse gas (GHG) emissions, before mitigation, are estimated to be 79,000 metric tons of CO_2e/year (MT CO_2e/yr) which exceed the SCAQMD threshold of 10,000 MT CO_2e/yr and are therefore considered significant (DEIR, p. 4.13-19). After mitigation, the DEIR states that GHG emissions will be less than significant (DEIR, p. 4.13-21). However, the DEIR does not calculate what the Project's GHG emissions will be after the mitigation measures are implemented.

The Project's GHG emissions exceed the SCAQMD threshold by nearly eight times. The DEIR and its supporting documents, including a Greenhouse Gas Study attached as Appendix B, do not provide any evidence that the proposed mitigation measures will reduce GHG emissions by a factor of eight.

A revised DEIR should be prepared to show the efficiency of the Project's proposed mitigation measures in reducing greenhouse gases. If these measures do not account for an eight-fold reduction in the Project's estimated GHG levels, additional mitigation measures (listed below) that are routinely considered in other CEQA projects should be implemented:

- Require preparation of a traffic control plan;

[20] http://www.rivcoeh.org/opencms/system/galleries/download/Environmental-Health/HMM/UST_Closure_App.pdf
[21] http://www.rivcoeh.org/opencms/system/galleries/download/Environmental-Health/HMM/Closure_by_removal_UST.pdf

- Demonstrate proper inspection and maintenance of construction equipment;
- Implement a carpool program for construction workers;
- Employ a construction site manager to verify that engines are properly maintained and keep a maintenance log;
- Configure construction parking to minimize traffic interference;
- Consolidate truck deliveries when possible;
- Provide dedicated turn lanes for movement of construction trucks and equipment on and off site;
- Suspend use of all construction equipment operations during second stage smog alerts;
- Establish a staging zone for trucks that are waiting to load or unload material at the work zone in a location where diesel emissions from the trucks will have minimum impact on abutters and the general public;
- Locate construction equipment away from sensitive receptors such as fresh air intakes to buildings, air conditioners and operable windows;
- Require all diesel trucks used by construction contractor(s) at the site, or for on-road hauling of construction material, to be post-1996 models; Diesel portable generators less than 50 hp shall not be allowed at the construction site;
- Use of hybrid and fuel efficient construction equipment and support vehicles (e.g., pick-up trucks);
- Use of grid electricity for smaller equipment such as saws, pumps, and welders;[22]
- Reduction in vehicle miles travelled in construction crew commutes through trip carpooling, trip reduction, providing bus service for crews from work sites to carpool parking areas, and in providing incentives to carpool; and
- Use of a Heavy-Duty Off-Road Vehicle Plan to ensure compliances with construction mitigation measures (e.g., hourly meters on equipment, documenting the serial number, horsepower, manufacture age, fuel, etc. of all onsite equipment and daily logging of the operating hours of the equipment).[23]

Air Quality:

The Project is located in the South Coast Air Basin and Riverside County[24], both of which are designated non-attainment for PM10 and ozone (DEIR, p. 4.3-6). Significant emissions of PM10 and ozone and contributing factors such as NOx and ROG will lead to a worsening of regional air quality. The Project's estimates of construction emissions need to be properly disclosed and mitigated to ensure that the Project has a less than significant impact on regional air quality.

Estimates and impacts of project's construction and operational emissions

[22] http://www.capcoa.org/wp-content/uploads/2010/11/CAPCOA-Quantification-Report-9-14-Final.pdf, p. 47
[23] Ibid., p. 431
[24] http://www.epa.gov/oaqps001/greenbk/ancl.html

Project construction and operation will result in significant emissions of ROG, NOx, and PM10 even after mitigation (DEIR, pp. 4.3-29, 4.3-34).

Construction emissions

The DEIR estimates that the Project's construction emissions of NOx and ROG will be significant as they exceed the SCAQMD thresholds of 100 lbs/day and 75 lbs/day, respectively (DEIR, p. 4.3-23) and identifies mitigation measures to reduce the Project's emissions (DEIR, pp. 4.3-23 – 4.3-29). Even with mitigation, the Project's emissions of NOx and ROG will still exceed SCAQMD thresholds and therefore are considered significant (DEIR, p. 4.3-29).

Operational emissions

The DEIR estimates that the Project's emissions of ROG, NOx, and PM10 from operational activities will be significant as they exceed the SCAQMD thresholds of 55 lbs/day, 55 lbs/day, and 150 lbs/day, respectively (DEIR, p. 4.3-33). The DEIR proposes mitigation measures to reduce the Project's emissions (DEIR, pp. 4.3-33 – 4.3-34) but, even with mitigation, the Project's emissions will still exceed SCAQMD thresholds and therefore are considered significant (DEIR, p. 4.3-34).

Gaseous particles such as NOx can react in the atmosphere to form PM10.[25,26] Because Riverside County and the South Coast Air Basin are both designated non-attainment for PM10, significant emissions of NOx can lead to a further degradation of regional air quality. NOx emissions can also react to produce ground-level ozone.[27] Exposure to NOx emissions and its products (ozone and PM10) can lead to the airway inflammation and can cause or exacerbate conditions such as emphysema and bronchitis.[28]

ROG can react to form ozone and contributes to smog formation.[29,30] Exposure to ozone can result in coughing, throat irritation, and chest pain, burning, and discomfort.[31] Smog exposure can lead to sneezing, nausea, coughing, headaches, and chest constriction.[32] A study published in the New England Journal of Medicine concluded that the risk of dying from respiratory diseases is three times higher in areas of concentrated ozone.[33]

Exposure to PM10 can cause bronchitis, increase the number and severity of asthma attacks, damage to lung tissue, and even premature death.[34] Research identifies that dust from construction is a major

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[25] http://www.fhwa.dot.gov/resourcecenter/teams/airquality/brochure/particulatebrochure.pdf
[26] http://www.epa.gov/captrade/documents/power.pdf
[27] *Ibid.*
[28] http://www.epa.gov/air/nitrogenoxides/health.html
[29] http://www.arb.ca.gov/research/health/fs/fs2/fs2.htm
[30] http://www.arb.ca.gov/html/gloss.htm#smog
[31] http://www.epa.gov/o3healthtraining/population.html
[32] http://are.berkeley.edu/courses/EEP101/spring03/AllThatSmog/extern.html
[33] http://www.nytimes.com/gwire/2009/03/12/12greenwire-study-links-smog-exposure-to-premature-death-10098.html
[34] http://www.epa.gov/airtrends/aqtrnd95/pm10.html and http://www.arb.ca.gov/html/brochure/pm10.htm

contributor to PM10 and that PM10 exposure is associated with asthma.[35] Inhalation of PM10 can exacerbate asthma especially in children who are susceptible to higher risks from PM10 exposure.[36]

The Project will have significant emissions of ROG, NOx, and PM10. Because Riverside County and the South Coast Air Basin are designated non-attainment areas for ozone and PM10, Project construction and operation will further degrade regional air quality. Exposure to ROG, NOx, and PM10 has adverse health effects and can impact offsite receptors, especially children in the nearby residences – a significant and undisclosed public health impact that the DEIR does not consider.

A revised DEIR should be prepared to disclose impacts to offsite receptors from Project construction and operation. Additional mitigation measures must be implemented to ensure that Project emissions of ROG, NOx, and PM10 are reduced to the maximum extent feasible.

Cumulative Impacts:

The DEIR identifies 13 proposed projects encompassing approximately 7.3 million square feet of space within five miles of the Project site (DEIR, p. 3-16). The DEIR does not identify the construction schedule of these projects except to state that "a number of individual projects may be under construction simultaneously with the proposed project" (DEIR, p. 4.3-37). The WestRidge Commerce Center Project (which will be built adjacent to the proposed Project) is scheduled to be constructed in 2012, a schedule similar to the proposed Project. Simultaneous construction of these projects, along with other potential projects, is likely to result in PM10, NOx, and ROG emissions that will have a cumulatively significant impact.

The construction timetables of all projects within the vicinity of the Project site should be identified. Any cumulatively significant emissions should be disclosed and impacts to workers and nearby residents should be addressed in a revised DEIR.

Sincerely,

Uma Bhandaram

Matt Hagemann, P.G., C.Hg.

[35] http://scerpfiles.org/cont_mgt/doc_files/EH-01-2.pdf
[36] http://www.co.imperial.ca.us/airpollution/attainment%20plans/final%20ic%202009%20pm10%20sip%20document.pdf

SWAPE Technical Consultation, Data Analysis and Litigation Support for the Environment

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Matthew F. Hagemann, P.G., C.Hg., QSD, QSP

Geologic and Hydrogeologic Characterization
Industrial Stormwater Compliance
CEQA Review
Investigation and Remediation Strategies
Litigation Support and Testifying Expert

Education:

M.S. Degree, Geology, California State University Los Angeles, Los Angeles, CA, 1984.
B.A. Degree, Geology, Humboldt State University, Arcata, CA, 1982.

Professional Certification:

California Professional Geologist
California Certified Hydrogeologist
Qualified SWPPP Developer and Practitioner

Professional Experience:

Matt has 25 years of experience in environmental policy, assessment and remediation. He spent nine years with the U.S. EPA in the RCRA and Superfund programs and served as EPA's Senior Science Policy Advisor in the Western Regional Office where he identified emerging threats to groundwater from perchlorate and MTBE. While with EPA, Matt also served as a Senior Hydrogeologist in the oversight of the assessment of seven major military facilities undergoing base closure. He led numerous enforcement actions under provisions of the Resource Conservation and Recovery Act (RCRA) while also working with permit holders to improve hydrogeologic characterization and water quality monitoring.

Matt has worked closely with U.S. EPA legal counsel and the technical staff of several states in the application and enforcement of RCRA, Safe Drinking Water Act and Clean Water Act regulations. Matt has trained the technical staff in the States of California, Hawaii, Nevada, Arizona and the Territory of Guam in the conduct of investigations, groundwater fundamentals, and sampling techniques.

Positions Matt has held include:

- Founding Partner, Soil/Water/Air Protection Enterprise (SWAPE) (2003 – present);
- Geology Instructor, Golden West College, 2010 – present;
- Senior Environmental Analyst, Komex H2O Science, Inc (2000 -- 2003);

- Executive Director, Orange Coast Watch (2001 – 2004);
- Senior Science Policy Advisor and Hydrogeologist, U.S. Environmental Protection Agency (1989–1998);
- Hydrogeologist, National Park Service, Water Resources Division (1998 – 2000);
- Adjunct Faculty Member, San Francisco State University, Department of Geosciences (1993 – 1998);
- Instructor, College of Marin, Department of Science (1990 – 1995);
- Geologist, U.S. Forest Service (1986 – 1998); and
- Geologist, Dames & Moore (1984 – 1986).

Partner, SWAPE:

With SWAPE, Matt's responsibilities have included:

- Lead analyst and testifying expert in the review of numerous environmental impact reports under CEQA that identify significant issues with regard to hazardous waste, water resources, water quality, air quality, greenhouse gas emissions and geologic hazards.
- Stormwater analysis, sampling and best management practice evaluation at industrial facilities.
- Lead analyst and testifying expert in the review of environmental issues in license applications for large solar power plants before the California Energy Commission.
- Technical assistance and litigation support for vapor intrusion concerns.
- Manager of a project to evaluate numerous formerly used military sites in the western U.S.
- Manager of a comprehensive evaluation of potential sources of perchlorate contamination in Southern California drinking water wells.
- Manager and designated expert for litigation support under provisions of Proposition 65 in the review of releases of gasoline to sources drinking water at major refineries and hundreds of gas stations throughout California.
- Expert witness on two cases involving MTBE litigation.
- Expert witness and litigation support on the impact of air toxins and hazards at a school.
- Expert witness in litigation at a former plywood plant.

With Komex H2O Science Inc., Matt's duties included the following:

- Senior author of a report on the extent of perchlorate contamination that was used in testimony by the former U.S. EPA Administrator and General Counsel.
- Senior researcher in the development of a comprehensive, electronically interactive chronology of MTBE use, research, and regulation.
- Senior researcher in the development of a comprehensive, electronically interactive chronology of perchlorate use, research, and regulation.
- Senior researcher in a study that estimates nationwide costs for MTBE remediation and drinking water treatment, results of which were published in newspapers nationwide and in testimony against provisions of an energy bill that would limit liability for oil companies.
- Research to support litigation to restore drinking water supplies that have been contaminated by MTBE in California and New York.
- Expert witness testimony in a case of oil production-related contamination in Mississippi.
- Lead author for a multi-volume remedial investigation report for an operating school in Los Angeles that met strict regulatory requirements and rigorous deadlines.
- Development of strategic approaches for cleanup of contaminated sites in consultation with clients and regulators.

Executive Director:

As Executive Director with Orange Coast Watch, Matt led efforts to restore water quality at Orange County beaches from multiple sources of contamination including urban runoff and the discharge of wastewater. In reporting to a Board of Directors that included representatives from leading Orange County universities and businesses, Matt prepared issue papers in the areas of treatment and disinfection of wastewater and control of the dischrge of grease to sewer systems. Matt actively participated in the development of countywide water quality permits for the control of urban runoff and permits for the discharge of wastewater. Matt worked with other nonprofits to protect and restore water quality, including Surfrider, Natural Resources Defense Council and Orange County CoastKeeper as well as with business institutions including the Orange County Business Council.

Hydrogeology:

As a Senior Hydrogeologist with the U.S. Environmental Protection Agency, Matt led investigations to characterize and cleanup closing military bases, including Mare Island Naval Shipyard, Hunters Point Naval Shipyard, Treasure Island Naval Station, Alameda Naval Station, Moffett Field, Mather Army Airfield, and Sacramento Army Depot. Specific activities were as follows:

- Led efforts to model groundwater flow and contaminant transport, ensured adequacy of monitoring networks, and assessed cleanup alternatives for contaminated sediment, soil, and groundwater.
- Initiated a regional program for evaluation of groundwater sampling practices and laboratory analysis at military bases.
- Identified emerging issues, wrote technical guidance, and assisted in policy and regulation development through work on four national U.S. EPA workgroups, including the Superfund Groundwater Technical Forum and the Federal Facilities Forum.

At the request of the State of Hawaii, Matt developed a methodology to determine the vulnerability of groundwater to contamination on the islands of Maui and Oahu. He used analytical models and a GIS to show zones of vulnerability, and the results were adopted and published by the State of Hawaii and County of Maui.

As a hydrogeologist with the EPA Groundwater Protection Section, Matt worked with provisions of the Safe Drinking Water Act and NEPA to prevent drinking water contamination. Specific activities included the following:

- Received an EPA Bronze Medal for his contribution to the development of national guidance for the protection of drinking water.
- Managed the Sole Source Aquifer Program and protected the drinking water of two communities through designation under the Safe Drinking Water Act. He prepared geologic reports, conducted public hearings, and responded to public comments from residents who were very concerned about the impact of designation.

- Reviewed a number of Environmental Impact Statements for planned major developments, including large hazardous and solid waste disposal facilities, mine reclamation, and water transfer.

Matt served as a hydrogeologist with the RCRA Hazardous Waste program. Duties were as follows:

- Supervised the hydrogeologic investigation of hazardous waste sites to determine compliance with Subtitle C requirements.
- Reviewed and wrote "part B" permits for the disposal of hazardous waste.
- Conducted RCRA Corrective Action investigations of waste sites and led inspections that formed the basis for significant enforcement actions that were developed in close coordination with U.S. EPA legal counsel.
- Wrote contract specifications and supervised contractor's investigations of waste sites.

With the National Park Service, Matt directed service-wide investigations of contaminant sources to prevent degradation of water quality, including the following tasks:

- Applied pertinent laws and regulations including CERCLA, RCRA, NEPA, NRDA, and the Clean Water Act to control military, mining, and landfill contaminants.
- Conducted watershed-scale investigations of contaminants at parks, including Yellowstone and Olympic National Park.
- Identified high-levels of perchlorate in soil adjacent to a national park in New Mexico and advised park superintendent on appropriate response actions under CERCLA.
- Served as a Park Service representative on the Interagency Perchlorate Steering Committee, a national workgroup.
- Developed a program to conduct environmental compliance audits of all National Parks while serving on a national workgroup.
- Co-authored two papers on the potential for water contamination from the operation of personal watercraft and snowmobiles, these papers serving as the basis for the development of nation-wide policy on the use of these vehicles in National Parks.
- Contributed to the Federal Multi-Agency Source Water Agreement under the Clean Water Action Plan.

Policy:

Served senior management as the Senior Science Policy Advisor with the U.S. Environmental Protection Agency, Region 9. Activities included the following:

- Advised the Regional Administrator and senior management on emerging issues such as the potential for the gasoline additive MTBE and ammonium perchlorate to contaminate drinking water supplies.
- Shaped EPA's national response to these threats by serving on workgroups and by contributing to guidance, including the Office of Research and Development publication, Oxygenates in Water: Critical Information and Research Needs.
- Improved the technical training of EPA's scientific and engineering staff.
- Earned an EPA Bronze Medal for representing the region's 300 scientists and engineers in negotiations with the Administrator and senior management to better integrate scientific principles into the policy-making process.
- Established national protocol for the peer review of scientific documents.

Geology:

With the U.S. Forest Service, Matt led investigations to determine hillslope stability of areas proposed for timber harvest in the central Oregon Coast Range. Specific activities were as follows:

- Mapped geology in the field, and used aerial photographic interpretation and mathematical models to determine slope stability.
- Coordinated his research with community members who were concerned with natural resource protection.
- Characterized the geology of an aquifer that serves as the sole source of drinking water for the city of Medford, Oregon.

As a consultant with Dames and Moore, Matt led geologic investigations of two contaminated sites (later listed on the Superfund NPL) in the Portland, Oregon, area and a large hazardous waste site in eastern Oregon. Duties included the following:

- Supervised year-long effort for soil and groundwater sampling.
- Conducted aquifer tests.
- Investigated active faults beneath sites proposed for hazardous waste disposal.

Teaching:

From 1990 to 1998, Matt taught at least one course per semester at the community college and university levels:

- At San Francisco State University, held an adjunct faculty position and taught courses in environmental geology, oceanography (lab and lecture), hydrogeology, and groundwater contamination.
- Served as a committee member for graduate and undergraduate students.
- Taught courses in environmental geology and oceanography at the College of Marin.

Matt currently teaches Physical Geology (lecture and lab) to students at Golden West College in Huntington Beach, California.

Invited Testimony, Reports, Papers and Presentations:

Hagemann, M.F., 2008. Disclosure of Hazardous Waste Issues under CEQA. Presentation to the Public Environmental Law Conference, Eugene, Oregon.

Hagemann, M.F., 2008. Disclosure of Hazardous Waste Issues under CEQA. Invited presentation to U.S. EPA Region 9, San Francisco, California.

Hagemann, M.F., 2005. Use of Electronic Databases in Environmental Regulation, Policy Making and Public Participation. Brownfields 2005, Denver, Coloradao.

Hagemann, M.F., 2004. Perchlorate Contamination of the Colorado River and Impacts to Drinking Water in Nevada and the Southwestern U.S. Presentation to a meeting of the American Groundwater Trust, Las Vegas, NV (served on conference organizing committee).

Hagemann, M.F., 2004. Invited testimony to a California Senate committee hearing on air toxins at schools in Southern California, Los Angeles.

Brown, A., Farrow, J., Gray, A. and **Hagemann, M.**, 2004. An Estimate of Costs to Address MTBE Releases from Underground Storage Tanks and the Resulting Impact to Drinking Water Wells. Presentation to the Ground Water and Environmental Law Conference, National Groundwater Association.

Hagemann, M.F., 2004. Perchlorate Contamination of the Colorado River and Impacts to Drinking Water in Arizona and the Southwestern U.S. Presentation to a meeting of the American Groundwater Trust, Phoenix, AZ (served on conference organizing committee).

Hagemann, M.F., 2003. Perchlorate Contamination of the Colorado River and Impacts to Drinking Water in the Southwestern U.S. Invited presentation to a special committee meeting of the National Academy of Sciences, Irvine, CA.

Hagemann, M.F., 2003. Perchlorate Contamination of the Colorado River. Invited presentation to a tribal EPA meeting, Pechanga, CA.

Hagemann, M.F., 2003. Perchlorate Contamination of the Colorado River. Invited presentation to a meeting of tribal repesentatives, Parker, AZ.

Hagemann, M.F., 2003. Impact of Perchlorate on the Colorado River and Associated Drinking Water Supplies. Invited presentation to the Inter-Tribal Meeting, Torres Martinez Tribe.

Hagemann, M.F., 2003. The Emergence of Perchlorate as a Widespread Drinking Water Contaminant. Invited presentation to the U.S. EPA Region 9.

Hagemann, M.F., 2003. A Deductive Approach to the Assessment of Perchlorate Contamination. Invited presentation to the California Assembly Natural Resources Committee.

Hagemann, M.F., 2003. Perchlorate: A Cold War Legacy in Drinking Water. Presentation to a meeting of the National Groundwater Association.

Hagemann, M.F., 2002. From Tank to Tap: A Chronology of MTBE in Groundwater. Presentation to a meeting of the National Groundwater Association.

Hagemann, M.F., 2002. A Chronology of MTBE in Groundwater and an Estimate of Costs to Address Impacts to Groundwater. Presentation to the annual meeting of the Society of Environmental Journalists.

Hagemann, M.F., 2002. An Estimate of the Cost to Address MTBE Contamination in Groundwater (and Who Will Pay). Presentation to a meeting of the National Groundwater Association.

Hagemann, M.F., 2002. An Estimate of Costs to Address MTBE Releases from Underground Storage Tanks and the Resulting Impact to Drinking Water Wells. Presentation to a meeting of the U.S. EPA and State Underground Storage Tank Program managers.

Hagemann, M.F., 2001. From Tank to Tap: A Chronology of MTBE in Groundwater. Unpublished report.

Hagemann, M.F., 2001. Estimated Cleanup Cost for MTBE in Groundwater Used as Drinking Water. Unpublished report.

Hagemann, M.F., 2001. Estimated Costs to Address MTBE Releases from Leaking Underground Storage Tanks. Unpublished report.

Hagemann, M.F., and VanMouwerik, M., 1999. Potential Water Quality Concerns Related to Snowmobile Usage. Water Resources Division, National Park Service, Technical Report.

VanMouwerik, M. and **Hagemann, M.F**. 1999, Water Quality Concerns Related to Personal Watercraft Usage. Water Resources Division, National Park Service, Technical Report.

Hagemann, M.F., 1999, Is Dilution the Solution to Pollution in National Parks? The George Wright Society Biannual Meeting, Asheville, North Carolina.

Hagemann, M.F., 1997, The Potential for MTBE to Contaminate Groundwater. U.S. EPA Superfund Groundwater Technical Forum Annual Meeting, Las Vegas, Nevada.

Hagemann, M.F., and Gill, M., 1996, Impediments to Intrinsic Remediation, Moffett Field Naval Air Station, Conference on Intrinsic Remediation of Chlorinated Hydrocarbons, Salt Lake City.

Hagemann, M.F., Fukunaga, G.L., 1996, The Vulnerability of Groundwater to Anthropogenic Contaminants on the Island of Maui, Hawaii. Hawaii Water Works Association Annual Meeting, Maui, October 1996.

Hagemann, M. F., Fukanaga, G. L., 1996, Ranking Groundwater Vulnerability in Central Oahu, Hawaii. Proceedings, Geographic Information Systems in Environmental Resources Management, Air and Waste Management Association Publication VIP-61.

Hagemann, M.F., 1994. Groundwater Characterization and Cleanup at Closing Military Bases in California. Proceedings, California Groundwater Resources Association Meeting.

Hagemann, M.F. and Sabol, M.A., 1993. Role of the U.S. EPA in the High Plains States Groundwater Recharge Demonstration Program. Proceedings, Sixth Biennial Symposium on the Artificial Recharge of Groundwater.

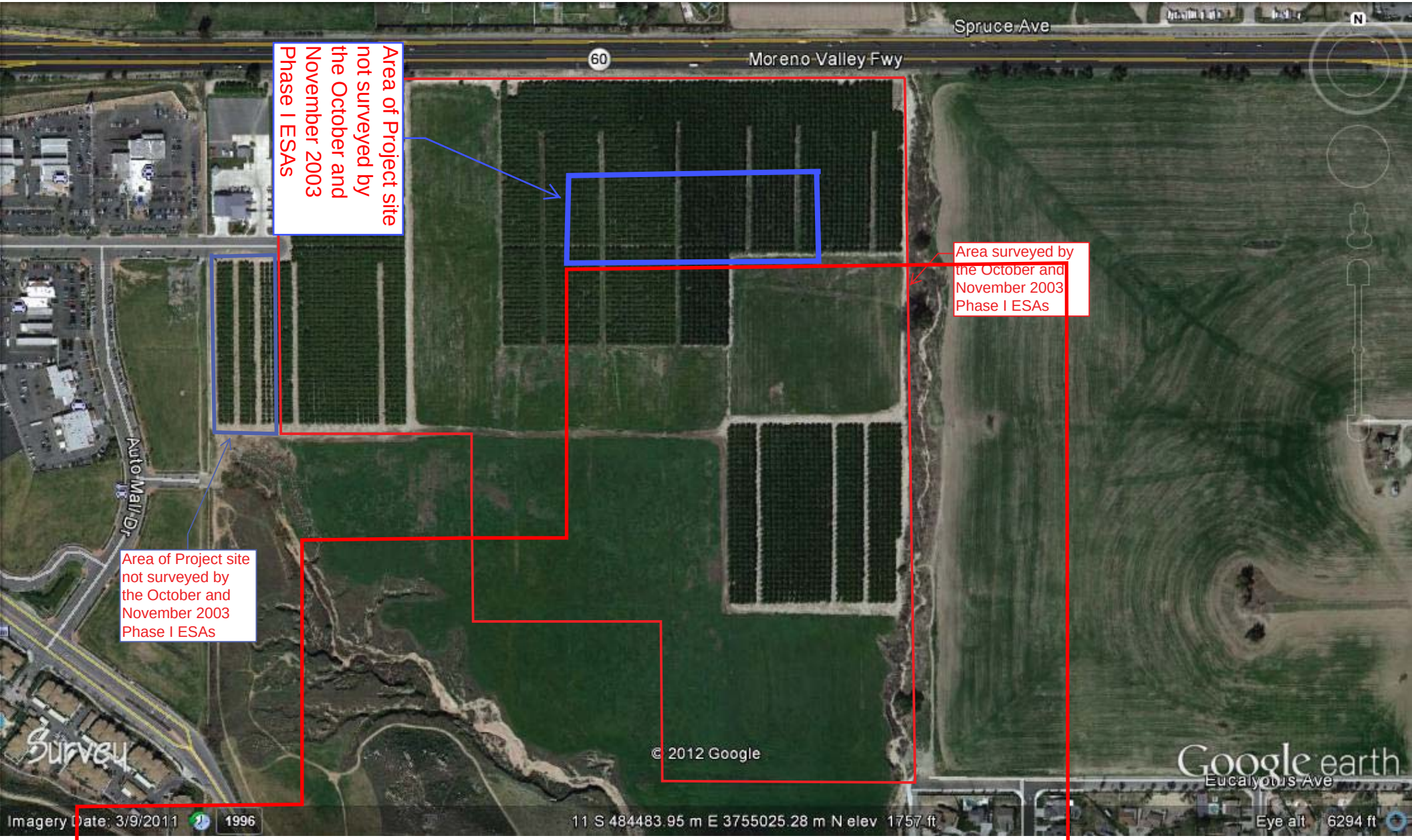
Hagemann, M.F., 1993. U.S. EPA Policy on the Technical Impracticability of the Cleanup of DNAPL-contaminated Groundwater. California Groundwater Resources Association Meeting.

Hagemann, M.F., 1992. Dense Nonaqueous Phase Liquid Contamination of Groundwater: An Ounce of Prevention... Proceedings, Association of Engineering Geologists Annual Meeting, v. 35.

Other Experience:

Selected as subject matter expert for the California Professional Geologist licensing examination, 2009-2011.

ATTACHMENT A


Spruce Ave
60
Moreno Valley Fwy
Area of Project site not surveyed by the October and November 2003 Phase I ESAs
Area surveyed by the October and November 2003 Phase I ESAs
Auto Mall Dr
Area of Project site not surveyed by the October and November 2003 Phase I ESAs
© 2012 Google
Google earth
Eucalyptus Ave
Imagery Date: 3/9/2011
1996
11 S 484483.95 m E 3755025.28 m N elev 1757 ft
Eye alt 6294 ft

Selected References

Query returned the following data:

There are 4 records returned.

YEAR	DATE	COUNTY NAME	COMTRS	SITE NAME	PRODUCT NAME	POUNDS PRODUCT APPLIED	CHEMICAL NAME	POUNDS CHEMICAL APPLIED	AMOUNT TREATED	UNIT TREATED	AERIAL GROUND INDICATOR
2010	20-MAR-10	RIVERSIDE	33S03S03W02	WHEAT, GENERAL	NUFARM WEEDONE LV6 EC BROADLEAF HERBICIDE	56.4474	2,4-D, 2-ETHYLHEXYL ESTER	48.8834484	72	A	A
2010	20-MAR-10	RIVERSIDE	33S03S03W02	WHEAT, GENERAL	NUFARM WEEDONE LV6 EC BROADLEAF HERBICIDE	38.2196	2,4-D, 2-ETHYLHEXYL ESTER	33.0981736	65	A	A
2010	18-MAR-10	RIVERSIDE	33S03S03W02	WHEAT, GENERAL	NUFARM WEEDONE LV6 EC BROADLEAF HERBICIDE	104.6629	2,4-D, 2-ETHYLHEXYL ESTER	90.6380714	133	A	G
2010	20-MAR-10	RIVERSIDE	33S03S03W02	WHEAT, GENERAL	NUFARM WEEDONE LV6 EC BROADLEAF HERBICIDE	29.3997	2,4-D, 2-ETHYLHEXYL ESTER	25.4601402	37	A	A

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Query returned the following data:

There are 1 records returned.

YEAR	DATE	COUNTY NAME	COMTRS	SITE NAME	PRODUCT NAME	POUNDS PRODUCT APPLIED	CHEMICAL NAME	POUNDS CHEMICAL APPLIED	AMOUNT TREATED	UNIT TREATED	AERIAL GROUND INDICATOR
2008	01-MAR-08	RIVERSIDE	33S03S03W02	WHEAT, GENERAL	NUFARM WEEDONE LV6 EC BROADLEAF HERBICIDE	76.4392	2,4-D, 2-ETHYLHEXYL ESTER	66.1963472	65	A	G

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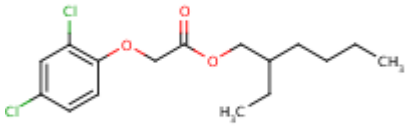
2,4-D 2-ETHYLHEXYL ESTER



CASRN: 1928-43-4

For more information, search the NLM HSDB database.

Human Health Effects:

Probable Routes of Human Exposure:
Occupational exposure to 2,4-D, 2-ethylhexyl ester may occur through inhalation and dermal contact with this compound at workplaces where 2,4-D, 2-ethylhexyl ester is produced or used. (SRC)
PEER REVIEWED

Emergency Medical Treatment:

Emergency Medical Treatment:

EMT Copyright Disclaimer:

Portions of the POISINDEX(R) and MEDITEXT(R) database have been provided here for general reference. THE COMPLETE POISINDEX(R) DATABASE OR MEDITEXT(R) DATABASE SHOULD BE CONSULTED FOR ASSISTANCE IN THE DIAGNOSIS OR TREATMENT OF SPECIFIC CASES. The use of the POISINDEX(R) and MEDITEXT(R) databases is at your sole risk. The POISINDEX(R) and MEDITEXT(R) databases are provided "AS IS" and "as available" for use, without warranties of any kind, either expressed or implied. Micromedex makes no representation or warranty as to the accuracy, reliability, timeliness, usefulness or completeness of any of the information contained in the POISINDEX(R) and MEDITEXT(R) databases. ALL IMPLIED WARRANTIES OF MERCHANTABILITY AND FITNESS FOR A PARTICULAR PURPOSE OR USE ARE HEREBY EXCLUDED. Micromedex does not assume any responsibility or risk for your use of the POISINDEX(R) or MEDITEXT(R) databases. Copyright 1974-2012 Thomson MICROMEDEX. All Rights Reserved. Any duplication, replication, "downloading," sale, redistribution or other use for commercial purposes is a violation of Micromedex' rights and is strictly prohibited.

The following Overview, *** CHLOROPHENOXY COMPOUNDS ***, is relevant for this

HSDB record chemical.

Life Support:

o This overview assumes that basic life support measures have been instituted.

Clinical Effects:

0.2.1 SUMMARY OF EXPOSURE

0.2.1.1 ACUTE EXPOSURE

A) WITH POISONING/EXPOSURE

1) ACUTE INGESTION - Miosis, coma, fever, hypotension, emesis, tachycardia, bradycardia, ECG abnormalities, muscle rigidity, possible respiratory failure, pulmonary edema, and rhabdomyolysis may occur. Deaths have resulted from cardiorespiratory arrest.

a) Concentrated formulations of 2,4-D-esters may contain petroleum solvents, contributing to the overall toxicity. Please refer to the HYDROCARBONS management for further information.

2) PATHOPHYSIOLOGY - These agents are primarily irritants, but one case of degenerative brain cell changes and CNS toxicity has been reported.

0.2.3 VITAL SIGNS

0.2.3.1 ACUTE EXPOSURE

A) WITH POISONING/EXPOSURE

1) Fever of sudden but delayed onset may occur following ingestion.

0.2.4 HEENT

0.2.4.1 ACUTE EXPOSURE

A) WITH POISONING/EXPOSURE

1) Eye, nose, and mouth irritation are possible with direct contact.

0.2.5 CARDIOVASCULAR

0.2.5.1 ACUTE EXPOSURE

A) WITH POISONING/EXPOSURE

1) Tachycardia, bradycardia, ECG abnormalities, asystole, other dysrhythmias, and hypotension have been reported with overdose. Deaths have resulted from cardiorespiratory arrest.

0.2.6 RESPIRATORY

0.2.6.1 ACUTE EXPOSURE

A) WITH POISONING/EXPOSURE

1) Ingestion of large amounts may cause bradypnea, respiratory failure, hyperventilation, or pulmonary edema.

0.2.7 NEUROLOGIC

0.2.7.1 ACUTE EXPOSURE

A) WITH POISONING/EXPOSURE

1) LOW DOSE EXPOSURES - Vertigo, headache, malaise, and paresthesias may occur depending on the specific compound involved.

2) HIGH DOSE EXPOSURES - Muscle twitching, spasms, profound weakness, polyneuritis, and unconsciousness may occur depending on the specific compound involved.

3) IDIOSYNCRATIC REACTIONS - Peripheral neuropathies

0.2.8 GASTROINTESTINAL

0.2.8.1 ACUTE EXPOSURE

A) WITH POISONING/EXPOSURE

1) Nausea, vomiting, and diarrhea have been reported. Necrosis of the gastrointestinal mucosa has been reported.

0.2.9 HEPATIC
0.2.9.1 ACUTE EXPOSURE
A) WITH POISONING/EXPOSURE
1) Elevated LDH, AST (SGOT), and ALT (SGPT) have been reported.
0.2.10 GENITOURINARY
0.2.10.1 ACUTE EXPOSURE
A) WITH POISONING/EXPOSURE
1) Albuminuria and porphyria may occur; renal failure due to rhabdomyolysis is also possible.
0.2.12 FLUID-ELECTROLYTE
0.2.12.1 ACUTE EXPOSURE
A) WITH POISONING/EXPOSURE
1) Ingestion of 2,4-D has produced hypocalcemia, hyperkalemia, and hypophosphatemia.
0.2.13 HEMATOLOGIC
0.2.13.1 ACUTE EXPOSURE
A) WITH POISONING/EXPOSURE
1) Thrombocytopenia is the primary hematologic effect. Leukopenia has also been reported.
0.2.14 DERMATOLOGIC
0.2.14.1 ACUTE EXPOSURE
A) WITH POISONING/EXPOSURE
1) Direct contact may cause skin irritation. Chlorodioxin contamination of products may produce chloracne with heavy exposure.
0.2.15 MUSCULOSKELETAL
0.2.15.1 ACUTE EXPOSURE
A) WITH POISONING/EXPOSURE
1) Muscle cramps, muscle rigidity, elevated creatine kinase, and rhabdomyolysis were reported after ingestion of MCPP. EMG abnormalities, elevated creatine kinase, and proximal muscle weakness have been described following 2,4-D ester exposure.
0.2.16 ENDOCRINE
0.2.16.1 ACUTE EXPOSURE
A) WITH POISONING/EXPOSURE
1) Hypoglycemia has been reported in cases of acute 2,4-D poisoning. Animal studies showed decreased T3 and T4 levels, but this effect has not been reported in humans.
0.2.20 REPRODUCTIVE HAZARDS
A) 2,4-D and 2,4,5-T have caused adverse reproductive effects in experimental animals. Allegations of human birth defects due to these compounds have not been confirmed.
0.2.21 CARCINOGENICITY
0.2.21.1 IARC CATEGORY
A) IARC Carcinogenicity Ratings for CAS94-75-7 (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2006; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2007; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010a; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2008; IARC, 2004):
1) Not Listed
B) IARC Carcinogenicity Ratings for CAS93-76-5 (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2006; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2007; IARC Working

Group on the Evaluation of Carcinogenic Risks to Humans, 2010; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010a; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2008; IARC, 2004):

1) Not Listed

C) IARC Carcinogenicity Ratings for CAS94-74-6 (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2006; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2007; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010a; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2008; IARC, 2004):

1) Not Listed

D) IARC Carcinogenicity Ratings for CAS93-65-2 (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2006; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2007; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010a; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2008; IARC, 2004):

1) Not Listed

0.2.21.2 HUMAN OVERVIEW

A) Human studies show conflicting results. Some studies have suggested a relationship between chlorophenoxy herbicides and both soft tissue sarcoma and non-Hodgkin's lymphoma, while others have not.

0.2.21.3 ANIMAL OVERVIEW

A) Animal studies are limited, but have generally been negative.

0.2.22 GENOTOXICITY

A) The chlorophenoxy herbicides have produced mixed negative and positive responses in various genotoxicity test systems. A recent review found no evidence of genotoxic or mutagenic potential in vitro and in vivo for 2,4-D.

B) One study was conducted to determine whether or not New Zealand Vietnam War veterans showed evidence of genetic disturbances arising as a consequence of their now confirmed exposure to chlorophenoxy herbicides. During 1965 to 1971, more than 76 million liters of phenoxylic herbicides were sprayed over parts of Southern Vietnam and Laos. A sample group of 24 New Zealand Vietnam War veterans and 23 control volunteers were compared using a sister chromatid exchange (SCE) analysis. The results showed a significant difference between the mean of the experimental group and the mean of the control group (11.05 vs 8.18; $p<0.001$). The experimental group also had an extremely elevated proportion of cells with high SCE frequencies (HFCs) above the 95th percentile compared to the controls (11% and 0.07%, respectively) (Rowland et al, 2007).

Laboratory:

A) These herbicides can be measured in the urine, but the values are not clinically useful. Plasma levels also appear to be poorly correlated with clinical effects.

B) Erythrocyte cholinesterase is not affected by these herbicides.
C) Obtain baseline CBC, platelet count, serum electrolytes, and renal/hepatic function tests. Monitor LDH, AST (SGOT), ALT (SGPT), alkaline phosphatase, CPK, arterial pH, and bicarbonate.
D) Monitor urine for pH, protein, RBC's, myoglobin, and urinary output.
E) Monitor the patient for at least 6 to 12 hours as there is a potential for delayed onset of symptoms.

Treatment Overview:

0.4.2 ORAL EXPOSURE

A) Treat ingestions of greater than 40 mg/kg with gastric decontamination if within 4 hours of ingestion.
B) ACTIVATED CHARCOAL: Administer charcoal as a slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old.
C) URINARY ALKALINIZATION: May enhance elimination. Should be considered with severe poisoning.
D) VENTRICULAR DYSRHYTHMIAS/SUMMARY: Institute continuous cardiac monitoring, obtain an ECG, and administer oxygen. Evaluate for hypoxia, acidosis, and electrolyte disorders. Lidocaine and amiodarone are generally first line agents for stable monomorphic ventricular tachycardia, particularly in patients with underlying impaired cardiac function. Amiodarone should be used with caution if a substance that prolongs the QT interval and/or causes torsades de pointes is involved in the overdose. Unstable rhythms require immediate cardioversion.
E) LIDOCAINE: ADULT: LOADING DOSE: 1 to 1.5 mg/kg IV push; for refractory VT/VF may give an additional bolus of 0.5 to 0.75 mg/kg over 3 to 5 min. Do not exceed 3 mg/kg or 200 to 300 mg over one hour. INFUSION: Once circulation restored begin infusion of 1 to 4 mg/min. PEDIATRIC: LOADING DOSE: 1 mg/kg; INFUSION: 20 to 50 mcg/kg/min. Monitor ECG continuously.

0.4.3 INHALATION EXPOSURE

A) INHALATION: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Administer oxygen and assist ventilation as required. Treat bronchospasm with inhaled beta2 agonist and oral or parenteral corticosteroids.
B) ACUTE LUNG INJURY: Maintain ventilation and oxygenation and evaluate with frequent arterial blood gas or pulse oximetry monitoring. Early use of PEEP and mechanical ventilation may be needed.

0.4.4 EYE EXPOSURE

A) DECONTAMINATION: Irrigate exposed eyes with copious amounts of room temperature water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist, the patient should be seen in a health care facility.

0.4.5 DERMAL EXPOSURE

A) OVERVIEW
1) DECONTAMINATION: Remove contaminated clothing and jewelry. Wash the skin, including hair and nails, vigorously; do repeated soap washings. Discard

contaminated clothing.
2) Treat dermal irritation or burns with standard topical therapy. Patients developing dermal hypersensitivity reactions may require treatment with systemic or topical corticosteroids or antihistamines.

Range of Toxicity:

A) Limited data are available.
B) Fatalities have been seen following ingestion of 80 mg/kg.
C) Intravenous injection of 28 mg/kg of 2,4-D was tolerated; 50 mg/kg produced toxicity.

[Rumack BH POISINDEX(R) Information System Micromedex, Inc., Englewood, CO, 2012; CCIS Volume 154, edition expires Nov, 2012. Hall AH & Rumack BH (Eds): TOMES(R) Information System Micromedex, Inc., Englewood, CO, 2012; CCIS Volume 154, edition expires Nov, 2012.] **PEER REVIEWED**

Antidote and Emergency Treatment:

Skin decontamination: Flush contaminating chemicals from eyes with copious amounts of water for 10 to 15 minutes. If irritation persists, an ophthalmological examination should be performed. /Chlorophenoxy Herbicides/
[U.S. Environmental Protection Agency/Office of Prevention, Pesticides, and Toxic Substances. Reigart, J.R., Roberts, J.R. Recognition and Management of Pesticide Poisonings. 5th ed. 1999. EPA Document No. EPA 735-R-98-003, and available in electronic format at: http://www.epa.gov/pesticides/safety/healthcare p. 97] **PEER REVIEWED**

Ingestions of these herbicides are likely to be followed by vomiting and diarrhea due to the irritant properties. ... Activated charcoal is probably effective in limiting irritant effects and reducing absorption of most or all of these herbicides. Aluminum hydroxide antacids may be useful in neutralizing the irritant actions of mose acidic agents. Sorbitol should be given to induce catharsis if bowel sounds are present and if spontaneous diarrhea has not already commenced. Dehydration and electrolyte disturbances may be severe enough to require intravenous fluids. There are no specific antidotes for poisoning by these herbicides. /Other Herbicides/
[U.S. Environmental Protection Agency/Office of Prevention, Pesticides, and Toxic Substances. Reigart, J.R., Roberts, J.R. Recognition and Management of Pesticide Poisonings. 5th ed. 1999. EPA Document No. EPA 735-R-98-003, and available in electronic format at: http://www.epa.gov/pesticides/safety/healthcare p. 123] **PEER REVIEWED**

Administer intravenous fluids to accelerate excretion of the chlorophenoxy compound, and to limit concentration of the toxicant in the kidney. A urine flow of 4-6 mL/minute is desirable. Intravenous saline/dextrose has sufficed to rescue comatose patients who drank 2,4-D and mecoprop several hours before hospital admission. CAUTION: Monitor urine protein, cells. BUN, serum creatine,serum electrolytes, and fluid intake/output carefully to insure that renal function remains unimpaired and that fluid overload does not occur. /Chlorophenoxy Herbicides/
[U.S. Environmental Protection Agency/Office of Prevention, Pesticides, and Toxic Substances. Reigart, J.R., Roberts, J.R. Recognition and Management of Pesticide Poisonings. 5th ed. 1999. EPA Document No. EPA 735-R-98-003, and available in electronic format at: http://www.epa.gov/pesticides/safety/healthcare p. 97] **PEER REVIEWED**

Forced alkaline diuresis has been used successfully in management of suicidal ingestions of chlorophenoxy compounds, especially when initiated early. Alkalinizing the urine by including sodium bicarbonate ... in the intravenous solution accelerates excretion of 2,4-D dramatically and mecoprop excretion substantially. Urine pH should be maintained between 7.6 and 8.8. Include potassium chloride to offset increased potassium losses. ... It is crucial to monitor serum electrolytes carefully, especially potassium and calcium. There may possibly be some hazard to the kidneys when urine concentrations of toxicant are very high, so the integrity of renal function and fluid balance should be monitor carefully as the chlorophenoxy compound is excreted. Renal failure has occured in patients with severe intoxication during alkaline diuresis. /Chlorophenoxy Herbicides/
[U.S. Environmental Protection Agency/Office of Prevention, Pesticides, and Toxic Substances. Reigart, J.R., Roberts, J.R. Recognition and Management of Pesticide Poisonings. 5th ed. 1999. EPA Document No. EPA 735-R-98-003, and available in electronic format at: http://www.epa.gov/pesticides/safety/healthcare p. 97] **PEER REVIEWED**

Hemodialysis is not likely to be of significant benefit in poisonings by chlorophenoxy compounds. It has been used in four patients who survived intoxication. However, given the highly protein-bound nature of these herbicides and lack of any other evidence , hemodialysis is not recommended. /Chlorophenoxy Herbicides/
[U.S. Environmental Protection Agency/Office of Prevention, Pesticides, and Toxic Substances. Reigart, J.R., Roberts, J.R. Recognition and Management of Pesticide Poisonings. 5th ed. 1999. EPA Document No. EPA 735-R-98-003, and available in electronic format at: http://www.epa.gov/pesticides/safety/healthcare p. 97] **PEER REVIEWED**

Animal Toxicity Studies:

Non-Human Toxicity Excerpts:

/LABORATORY ANIMALS: Acute Exposure/ English pointer dogs dosed po with encapsulated 2,4-dichlorophenoxyacetic acid (2,4-D) at 1.3, 8.8, 43.7, 175 or 220 mg/kg body weight failed to exhibit abnormalities in hematologic, serum biochemical, urinalysis, or electrocardiographic parameters. At the 3 lowest doses, no changes were noted in electro-encephalograms (EEGs). In the dog given 175 mg/kg, at 24 h postdosing mild sedation was accompanied by excessive slowing in the EEG with loss of low voltage fast activity. In the dog given 220 mg/kg, nonspecific alterations in the EEG suggestive of irritation and mild seizure activity was detected 7 hr, but the EEG returned to normal by 24 hr. /2,4-D/
[Arnold EK et al; Vet Hum Toxicol 33 (5): 446-9 (1991)] **PEER REVIEWED** PubMed Abstract

/LABORATORY ANIMALS: Acute Exposure/ The acute toxicity of 2,4-dichlorophenoxyacetic acid (2,4-D), a herbicide, was studied in chicks dosed with 100, 300, 500, or 600 mg 2,4-D/kg BW, by the oral route. Clinical, laboratory, and histopathological methods were used as indicators of toxicity. After acute exposure, the herbicide decreased motor activity and induced muscular weakness and motor incoordination; decreased weight gain; increased serum creatine kinase (CK) and alkaline phosphatase (AP) activities and serum uric acid (UA), creatinine (CR), and total proteins (TP) levels; and did not change serum aspartate aminotransferase (AST) or alanine aminotransferase (ALT) activities. These changes were time- and dose-dependent and reversible. The LD50 (lethal dose 50%) calculated for oral 2,4-D in chicks was 420 mg/kg BW (385 to 483). Chromatographic analysis of the serum of the intoxicated chicks showed the presence of the herbicide; the amount found was dose- and time-dependent, increasing from 2 to 8 hr after exposure and decreasing afterwards. Histopathological post-mortem studies conducted on intoxicated chicks showed hepatic (vacuolar degeneration of the hepatocytes), renal (tubular nephrosis), and intestinal (hemorrhagic) lesions. /2,4-D/
[Morgulis MS, et al; Poult Sci 77 (4): 509-515 (1998)] **PEER REVIEWED** PubMed Abstract

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ Forms of 2,4-dichlorophenoxyacetic acid (collectively known as 2,4-D) are herbicides used to control a wide variety of broadleaf and woody plants. Subchronic toxicity studies in rats were conducted on three forms of 2,4-D: the parent form, 2,4-D acid; 2,4-D dimethylamine salt (DMA); and 2,4-D 2-ethylhexyl ester (2-EHE). Doses in the subchronic studies (on an acid equivalent basis) were 0, 1, 15, 100, and 300 mg/kg/day. Major treatment related findings in the three studies included decreases in red cell mass, decreases in T3 and T4 levels, decreases in ovary and testes weights, increases in liver, kidney, and thyroid weights, and cataracts and retinal degeneration (high-dose females). These data demonstrated the comparable toxicities of 2,4-D acid, DMA, and 2-EHE and support a subchronic no-observed-effect level of 15 mg/kg/day for all three forms.
[Charles JM, et al; Fundam Appl Toxicol 33 (2): 161-165 (1996)] **PEER REVIEWED** PubMed Abstract

/LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ The influence of sublethal doses of 2,4-dichlorophenoxyacetic acid (2,4-D) on serum T3 & T4 concns in Hsd Cpb: Wistar rats of both sexes was studied. The trial was performed on 24 males & females respectively, each divided into three groups of 8 animals (control, groups 1 & 2). Aqueous soln of the compound (11 mg/kg bw--group 1 & 110 mg/kg bw--group 2) or clean tap water (control group) was used. Aliquots of 2.4 mL/kg bw were administered with a stomach tube from the 1st-10th day of the experiment. Three days before the first treatment & on the 6th & 13th day of the experiment the serum T3 & T4 concns were determined by commercial radioimmunoassay kits (Byk-Sangtec Diagnostica), validated for rats. A significant decr of serum T4 ($P<0.01$) & T3 ($P<0.001$) was determined in males of groups 1 & 2 during the experiment. On the 6th day of experiment serum T4 & T3 values were significantly lower ($P<0.001$ & 0.01 respectively) in group 2 than in the controls & group 1 of both males & females. During the whole experiment serum T4 levels were lower in females than in males ($P<0.05$). /2,4-D/
[Kobal S, et al; Pflugers Arch 440 (5 Suppl): R171-172 (2000)] **PEER REVIEWED**

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ Groups of 25 male & 25 female 3 wk old Osborne-Mendel rats were fed for 2 yrs on diets containing 0, 5, 25, 125, 625 or 1250 mg/kg of diet 2,4-D. 2,4-D was 96.7% pure & contained no detectable levels of 2,7-dichloro- or 2,3,7,8-tetrachlorodibenzo-p-dioxin ... . Numbers of male & female rats with malignant tumors were 6 in controls & 8, 7, 7, 8 & 14 in the treated groups, respectively. Tumors were randomly distributed & were also found in aging rats of this strain. ... A statistical increase ($p< 0.05$) in number of treated rats with malignant tumors over controls were found only in males receiving ... 1250 mg/kg. /2,4-D/
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). Available at: http://monographs.iarc.fr/index.php p. V15 117 (1977)] **PEER REVIEWED**

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ 6xC3H/Anf) F1 mice & 18 male & 18 female (C57BL/6xAKR)F1 mice received commercial 2,4-D (90%, mp 136-140 deg C) according to the following dose schedule: 46.4 mg/kg body wt in 0.5% gelatin by stomach tube at 7 days of age & the same amount (not adjusted for incr body wt) daily up to 28 days of age; subsequently, the mice were given 149 mg/kg of diet /feed/. ... The experiment was terminated when the mice were about 78 weeks of age ... Tumor incidences were compared with those observed among groups of ... control mice, which had been untreated or had received gelatin only: the incidences were not significantly greater ($p> 0.05$) when any group or combination of groups were considered. Similar results were obtained in groups of mice given 2,4-D isopropyl, butyl, or isooctyl esters (99%, 99%, and 97% pure) at doses of 46.6 mg/kg body wt from 7-28 days of age and, subsequently 111, 149, & 130 mg/kg of diet /feed/ respectively up to 78 weeks of age. /2,4-D/
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). Available at: http://monographs.iarc.fr/index.php p. V15 117 (1977)] **PEER REVIEWED**

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ When 2,4-D was administered at a concentrations of 500 mg/kg of diet during entire pregnancy of a sow, anorexia was noted; newborn piglets were underdeveloped & apathetic & 10/15 died within 24 hr. Continued feeding of 50 mg/kg of diet to survivors until ... 8 months of age caused growth depression, persistent anemia, & moderate degenerative changes of liver & kidneys. /2,4-D/
[IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). Available at: http://monographs.iarc.fr/index.php p. V15 123 (1977)] **PEER REVIEWED**

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ The reproductive toxicity of 2,4-D has been studied at dietary doses of 0, 5, 20, and 80 mg/kg/day in a two generation reproductive study in Fischer 344 rats. The parental Fo group was treated with 2,4-D for 15 weeks prior to mating. No adverse effects on fertility were observed in the 5 and 20 mg/kg daily dose groups, although reduced pup weights were noted in the 20 mg/kg F2a litters. A daily NOAEL of 5 mg/kg for reproductive toxicity was established from this study. In addition to this reproduction study, recent subchronic and chronic studies in rats, mice and dogs produced no evidence of treatment related histopathological changes in the testes at any of the dose levels ... . /2,4-D/
[Bingham, E.; Cohrssen, B.; Powell, C.H.; Patty's Toxicology Volumes 1-9 5th ed. John Wiley & Sons. New York, N.Y. (2001)., p. V4 493] **PEER REVIEWED**

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ The cytogenetic effect of 2,4-dichlorophenoxy acetic acid (2,4-D) & its metabolite 2,4-dichlorophenol (2,4-DCP) was studied in bone-marrow, germ cells & sperm head abnormalities in the treated mice. Swiss mice were treated orally by gavage with 2,4-D at 1.7, 3.3 and 33 mg kg(-1)BW (1/200, 1/100 and 1/10 of LD(50)). 2,4-DCP was intraperitoneally (i.p.) injected at 36, 72 and 180 mg kg(-1)BW (1/10, 1/5, 1/2 of LD(50)). A significant increase in the percentage of chromosome aberrations in bone-marrow and spermatocyte cells was observed after oral administration of 2,4-D at 3.3 mg kg(-1)BW for three and five consecutive days. This percentage increased and reached 10.8+/-0.87 (P<0.01) in bone-marrow and 9.8+/-0.45 (P<0.01) in spermatocyte cells after oral administration of 2,4-D at 33 mg kg(-1)BW for 24 hr. This percentage was, however, lower than that induced in bone-marrow and spermatocyte cells by mitomycin C (positive control). 2,4-D induced a dose-dependent increase in the percentage of sperm head abnormalities. The genotoxic effect of 2,4-DCP is weaker than that of 2,4-D, as indicated by the lower percentage of the induced chromosome aberrations (in bone-marrow and spermatocyte cells) and sperm head abnormalities. /2,4-D/
[Amer SM, Aly FA; Mutat Res 25; 494 (1-2): 1-12 (2001)] **PEER REVIEWED**

/LABORATORY ANIMALS: Neurotoxicity/ The acute effects of 2,4-dichlorophenoxyacetic acid (2,4-D) administered orally to female mongrel dogs in doses of 25, 50, 75, 100 or 125 mg/kg were investigated by means of neurological examinations, electromyography and motor nerve conduction velocity tests carried out at various times following treatment. On day one after treatment with 125 mg/kg, one of four dogs was lethargic but recovered by day three. Also on day one, myotonic dimpling was evident in one dog each in the groups treated with 50, 100, 125 mg/kg. Dogs treated with more than 50 mg/kg had generalized myotonic discharges which increased according to the dose and were resolved by day 14 but not day seven. Treatment failed to affect motor nerve conduction velocity. Pathologic changes in teased nerve fibers involved occasional fiber degeneration, paranodal demyelination and intercalated internodes. Transverse semi-thin sections showed mild focal fiber degeneration and eventual medial plantar nerve depletion in five dogs treated with 25, 100 and 125 mg/kg and in lateral plantar nerve of two dogs treated with 125 mg/kg and one control. A single exposure to sublethal oral doses of 2,4-D is not associated with evidence of polyneuropathy. /2,4-D/
[Steiss JE et al; J Neurol Sci 78 (3): 295-301 (1987)] **PEER REVIEWED** PubMed Abstract

/LABORATORY ANIMALS: Neurotoxicity/ Forms of 2,4-dichlorophenoxyacetic acid ... are herbicides used to control a wide variety of broadleaf and woody plants. Single-dose acute and 1-year chronic neurotoxicity screening studies in male and female Fischer 344 rats (10/sex/dose) were conducted on 2,4-D according to the U.S. EPA 1991 guidelines. The studies emphasized a Functional Observational Battery (which included grip performance and hindlimb splay tests), automated motor activity testing, and comprehensive neurohistopathology of perfused tissues. Dosages were up to 250 mg/kg by gavage for the single-dose study, and up to 150 mg/kg/day in the diet for 52 weeks in the repeated-dose study. In the acute study, gavage with 250 mg/kg test material caused slight transient gait and coordination changes and clearly decreased motor activity at the time of maximal effect on the day of treatment (day 1). Mild locomotor effects occurred in one mid-dose rat (75 mg/kg), on Day 1 only. No gait, coordination, or motor activity effects were noted by day 8. In the chronic study, the only finding of neurotoxicologic significance was retinal degeneration in females in the high-dose group (150 mg/kg/day). Body weights of both sexes were slightly less than controls in the mid-dose group, and 10% less than controls in the high-dose group. /2,4-D/
[Mattsson JL, et al; Fundam Appl Toxicol 40 (1): 111-119 (1997)] **PEER REVIEWED** PubMed Abstract

/GENOTOXICITY/ 2,4-D, 2-Ethylhexyl ester, 98.0% purity, at concentrations of 0 (DMSO), 0.501, 1.00, 2.50, 5.00, 10.0, or 25.0 ug/mL, was assayed with primary rat hepatocytes. The treatment period was 19 hours. 2,4-D, 2-Ethylhexyl ester, did not induce unscheduled DNA synthesis.
[California Environmental Protection Agency/Department of Pesticide Regulation; Toxicology Data Review Summaries. Available from: http://www.cdpr.ca.gov/docs/toxsums/toxsumlist.htm on 2,4-D as of February 1, 2005.] **PEER REVIEWED**

/GENOTOXICITY/ 2,4-D, 2-Ethylhexyl Ester [grouped with 2,4-D free acid as of 7/23/91], purity of 98.0%, at

concentrations of 0 (DMSO), 333, 667, 1000, 3330, 6670, or 10000 ug/plate without and with metabolic activation (Aroclor 1254-induced rat liver) was assayed with Salmonella typhimurium strains TA98, TA100, TA1535, TA1537 and TA1538. Incubation period was for 48 hours. 2,4-D,-2-Ethylhexyl Ester did not increase the number of revertants in either the initial or repeat assay.
[California Environmental Protection Agency/Department of Pesticide Regulation; Toxicology Data Review Summaries. Available from: http://www.cdpr.ca.gov/docs/toxsums/toxsumlist.htm on 2,4-D as of February 1, 2005.] **PEER REVIEWED**

/GENOTOXICITY/ 2,4-D, 2-Ethylhexyl Ester, purity 98.0%, LOT # 04KF54479, was administered as a single dose by gavage at 0 (corn oil), 50, 167, or 500 mg/kg to 5 ICR mice/sex/group. Bone marrow was harvested at 24, 48, and 72 hours after dosing. Polychromatic erythrocytes were scored for micronuclei and the PCE/NCE ratio determined. One thousand PCE's were scored per animal. The test substance did not induce a significant increase in micronuclei in bone marrow polychromatic erythrocytes.
[California Environmental Protection Agency/Department of Pesticide Regulation; Toxicology Data Review Summaries. Available from: http://www.cdpr.ca.gov/docs/toxsums/toxsumlist.htm on 2,4-D as of February 1, 2005.] **PEER REVIEWED**

/GENOTOXICITY/ Using the Curly-Lobe-Plum method in Drosophila melanogaster, this herbicide, manifested a significant mutagenic effect: frequency of the lethal recessive mutations was 6 times higher in the group of flies treated with the herbicide than in the untreated, control group.
[Coman N et al; Studia Universitatis Babes-Bolyai Biologia 37 (1): 65-70 (1992)] **PEER REVIEWED**

/OTHER TOXICITY INFORMATION/ The effects of daily dosing with the 2-ethyl hexyl ester of 2,4-D and its components at 250 mg/kg on blood urea nitrogen and plasma Mg:Ca ratios in cattle and sheep are tabulated. The formulation of the herbicide (emulsifiable concentrate or technical grade) showed no difference in the effects. Treatment with the compound resulted in a decrease in plasma Ca and an increase in plasma Mg significantly changing the ratio in the plasma of two sheep and a yearling heifer that died. In some cases, there was a 50% ratio decrease. Increased blood urea nitrogen (in one case increased from 4 to 40 mg/100 mL) was noted in the herbicide-treated animals. Kidney damage and swollen blood-engorged thyroids were commonly noted during the postmortem examinations.
[Hunt LM et al; Bull Environ Contam Toxicol 5 (1): 54-60 (1970)] **PEER REVIEWED**

Ecotoxicity Excerpts:

/AQUATIC SPECIES/ In studies conducted according to the guidelines of the US Environmental Protection Agency, 2,4-D acid and ethylhexyl ester had no effect on the early life stages, embryo hatch, larval weight, or larval length of the fathead minnow (Pimephales promelas) at concentrations of 12.6-102 mg/L for up to 32 days (acid). The 32-day NOEC for the acid was 63.4 mg/L, comparable to the 33-day NOEC for the diethanolamine salt of 29.1 mg/L. The ethylhexyl ester was more toxic, with a 32-day NOEC of 0.12 mg/L... .
[FAO/WHO; Pesticide Residues in Food: Toxicological and Environmental Evaluations: 2,4-Dichlorophenoxyacetic acid (2,4-D), salts and esters (1997). Available from, as of February 1, 2005: http://www.inchem.org/documents/jmpr/jmpmono/v097pr16.htm **PEER REVIEWED**

/AQUATIC SPECIES/ The esters of 2,4-D are clearly more toxic to invertebrate species such as the tidewater silverside (Menidia beryllina), Atlantic silverside (Menidia menidia), grass shrimp (Palaemonetes puqio), pink shrimp (Panaeus duorarum), and Dungeness crab (Cancer magister) than is the dimethylamine salt or the acid. The same is true for formulated 2-ethylhexyl ester.
[FAO/WHO; Pesticide Residues in Food: Toxicological and Environmental Evaluations: 2,4-Dichlorophenoxyacetic acid (2,4-D), salts and esters (1997). Available from, as of February 2, 2005: http://www.inchem.org/documents/jmpr/jmpmono/v097pr16.htm **PEER REVIEWED**

Non-Human Toxicity Values:

LD50 Rat (male) oral 982 mg/kg
[European Chemicals Bureau; IUCLID Dataset, 2-ethylhexyl 2,4-dichhlorophenoxyacetate (1928-43-4) (2000 CD-ROM edition). Available from, as of January 13, 2005: http://esis.jrc.ec.europa.eu/ **PEER REVIEWED**

LD50 Rat (female) oral 864 mg/kg
[European Chemicals Bureau; IUCLID Dataset, 2-ethylhexyl 2,4-dichhlorophenoxyacetate (1928-43-4) (2000 CD-ROM edition). Available from, as of January 13, 2005: http://esis.jrc.ec.europa.eu/ **PEER REVIEWED**

LD50 Mouse oral 673 mg/kg
[European Chemicals Bureau; IUCLID Dataset, 2-ethylhexyl 2,4-dichhlorophenoxyacetate (1928-43-4) (2000 CD-ROM edition). Available from, as of January 13, 2005: http://esis.jrc.ec.europa.eu/ **PEER REVIEWED**

LD50 Rat oral 896 mg/kg
[Tomlin CDS, ed. 2,4-D-2-ethylhexyl (1928-43-4). In: The e-Pesticide Manual, 13th Edition Version 3.0 (2003-04). Surrey UK, British Crop Protection Council.] **PEER REVIEWED**

LD50 Rabbit dermal >2000 mg/kg
[Tomlin CDS, ed. 2,4-D-2-ethylhexyl (1928-43-4). In: The e-Pesticide Manual, 13th Edition Version 3.0 (2003-04). Surrey UK, British Crop Protection Council.] **PEER REVIEWED**

LC50 Rat inhalation >5.4 mg/L air/4 hr
[Tomlin CDS, ed. 2,4-D-2-ethylhexyl (1928-43-4). In: The e-Pesticide Manual, 13th Edition Version 3.0 (2003-04). Surrey UK, British

Crop Protection Council.] **PEER REVIEWED**

Ecotoxicity Values:

LD50 Anas platyrhynchos (Mallard duck, juvenile) oral 663 mg/kg/14 days
[USEPA, Office of Pesticide Programs; Pesticide Ecotoxicity Database (2000) on 2,4-D, 2-Ethylhexyl ester (1928-43-4). Available from, as of January 26, 2005: http://cfpub.epa.gov/ecotox/quick_query.htm **PEER REVIEWED**

LD50 Anas platyrhynchos (Mallard duck, 14 day old) oral >4640 mg/kg/8 days
[USEPA, Office of Pesticide Programs; Pesticide Ecotoxicity Database (2000) on 2,4-D, 2-Ethylhexyl ester (1928-43-4). Available from, as of January 26, 2005: http://cfpub.epa.gov/ecotox/quick_query.htm **PEER REVIEWED**

LC50 Anas platyrhynchos (Mallard duck, juvenile) dietary >5620 ppm/8 days
[USEPA, Office of Pesticide Programs; Pesticide Ecotoxicity Database (2000) on 2,4-D, 2-Ethylhexyl ester (1928-43-4). Available from, as of January 26, 2005: http://cfpub.epa.gov/ecotox/quick_query.htm **PEER REVIEWED**

LC50 Colinus virginianus (Northern bobwhite, juvenile) dietary 7187 ppm/8 days
[USEPA, Office of Pesticide Programs; Pesticide Ecotoxicity Database (2000) on 2,4-D, 2-Ethylhexyl ester (1928-43-4). Available from, as of January 26, 2005: http://cfpub.epa.gov/ecotox/quick_query.htm **PEER REVIEWED**

EC50 Anabaena flosaquae (Blue-green algae; population abundance) >0.32 ppm/5 days; static /formulated product/
[USEPA, Office of Pesticide Programs; Pesticide Ecotoxicity Database (2000) on 2,4-D, 2-Ethylhexyl ester (1928-43-4). Available from, as of January 26, 2005: http://cfpub.epa.gov/ecotox/quick_query.htm **PEER REVIEWED**

EC50 Selenastrum capricornutum (Green algae; population abundance) >30.0 ppm/5 days; static /formulated product/
[USEPA, Office of Pesticide Programs; Pesticide Ecotoxicity Database (2000) on 2,4-D, 2-Ethylhexyl ester (1928-43-4). Available from, as of January 26, 2005: http://cfpub.epa.gov/ecotox/quick_query.htm **PEER REVIEWED**

EC50 Crassostrea virginica (American oyster; intoxication immobilization) >3.0 ppb/96 hr; flow-through /formulated product/
[USEPA, Office of Pesticide Programs; Pesticide Ecotoxicity Database (2000) on 2,4-D, 2-Ethylhexyl ester (1928-43-4). Available from, as of January 26, 2005: http://cfpub.epa.gov/ecotox/quick_query.htm **PEER REVIEWED**

LC50 Gammarus fasciatus (Scud) 2400 ppb/96 hr (95% confidence interval: 1900-3000 ppb); static /formulated product/
[USEPA, Office of Pesticide Programs; Pesticide Ecotoxicity Database (2000) on 2,4-D, 2-Ethylhexyl ester (1928-43-4). Available from, as of January 26, 2005: http://cfpub.epa.gov/ecotox/quick_query.htm **PEER REVIEWED**

LC50 Oncorhynchus Mykiss (Rainbow trout) 7.2 mg/L/96 hr; flow-through
[European Chemicals Bureau; IUCLID Dataset, 2-ethylhexyl 2,4-dichhlorophenoxyacetate (1928-43-4) (2000 CD-ROM edition). Available from, as of January 13, 2005: http://esis.jrc.ec.europa.eu/ **PEER REVIEWED**

EC50 Navicula pelliculosa (algae) 4.1 mg/L 5 days endpoint: growth rate; NOEC = 0.1875
[European Chemicals Bureau; IUCLID Dataset, 2-ethylhexyl 2,4-dichhlorophenoxyacetate (1928-43-4) (2000 CD-ROM edition). Available from, as of January 13, 2005: http://esis.jrc.ec.europa.eu/ **PEER REVIEWED**

EC50 Skeletonema costatum (Algae; growth inhibition) 0.23 mg/L/5 days; static /from table/
[FAO/WHO; Pesticide Residues in Food: Toxicological and Environmental Evaluations: 2,4-Dichlorophenoxyacetic acid (2,4-D), salts and esters (1997). Available from, as of February 1, 2005: http://www.inchem.org/documents/jmpr/jmpmono/v097pr16.htm **PEER REVIEWED**

LD50 Honeybee (Apis mellifera) oral or contact >100 mg/bee/72 hr
[FAO/WHO; Pesticide Residues in Food: Toxicological and Environmental Evaluations: 2,4-Dichlorophenoxyacetic acid (2,4-D), salts and esters (1997). Available from, as of February 2, 2005: http://www.inchem.org/documents/jmpr/jmpmono/v097pr16.htm **PEER REVIEWED**

Metabolism / Pharmacokinetics:

Metabolism / Metabolites:

The pharmacokinetics of the 2-ethylhexyl ester of 2,4-D were investigated following a single oral administration of 130 mg/kg body weight dose to both male and female Fischer 344 rats. Blood samples were drawn from 24 rats per sex in serial groups of 3 at intervals of 0.25, 0.5, 1, 2, 4, 8, 24, and 72 hours post dosing and urine was collected from the 72 hours group at 12 hour intervals. The most significant finding from this evaluation was the absence of any 2-ethylhexyl ester of 2,4-D in either the blood or urine for either sex evaluated (limit of quantification 10 ppb). Conversely 2,4-D acid was detected in both blood and urine. The present data indicate that the 2-ethylhexyl ester of 2,4-D is converted very rapidly to 2,4-D acid, and that the acid is then excreted into the urine. A similarity exists in interval excretion data with that seen in previous investigations with 2,4-D acid. Indications are that the 2,4-D acid is probably derived via the hydrolysis of the 2-ethylhexyl ester moiety and is eliminated from the body in the same manner as the orally administered 2,4-D acid. It is therefore anticipated from these results that the 2-ethylhexyl ester of 2,4-D should be toxicologically comparable to 2,4-D acid itself.
[European Chemicals Bureau; IUCLID Dataset, 2-ethylhexyl 2,4-dichhlorophenoxyacetate (1928-43-4) (2000 CD-ROM edition). Available from, as of January 13, 2005: http://esis.jrc.ec.europa.eu/ **PEER REVIEWED**

2,4-D 2-ethylhexyl ester is hydrolysed to 2,4-D by esterase enzymes present in the gut wall, in blood plasma, in liver cells and in skin. Any 2,4-D /ethylhexyl ester/ absorbed orally or dermally is hydrolysed to 2,4-D, the acid ionic form.
[European Chemicals Bureau; IUCLID Dataset, 2-ethylhexyl 2,4-dichhlorophenoxyacetate (1928-43-4) (2000 CD-ROM edition). Available from, as of January 13, 2005: http://esis.jrc.ec.europa.eu/ **PEER REVIEWED**

Absorption, Distribution & Excretion:
A maximum 2,4-D concentration in serum of 1075 ppm was detected 5 hr after /English pointer dogs were given a/ po dose of 220 mg/kg. A maximum 2,4-D, concentration in urine of 1792 ppm was detected 2 hr after a po dose of 175 mg/kg, while 25 hr after that dose kidney tissue contained 271 ppm. /2,4-D/
[Arnold EK et al; Vet Hum Toxicol 33 (5): 446-9 (1991)] **PEER REVIEWED** PubMed Abstract

Pharmacology:

Environmental Fate & Exposure:

Environmental Fate/Exposure Summary:
2,4-D, 2-ethylhexyl ester's production may result in its release to the environment through various waste streams; its use as a herbicide will result in its direct release to the environment. If released to air, a vapor pressure of 3.59X10-4 mm Hg at 25 deg C indicates 2,4-D, 2-ethylhexyl ester will exist solely as a vapor in the ambient atmosphere. Vapor-phase 2,4-D, 2-ethylhexyl ester will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 27 hours. If released to soil, 2,4-D, 2-ethylhexyl ester is expected to have no mobility based upon an estimated Koc of 33,000. Volatilization from moist soil surfaces is expected to be an important fate process based upon a Henry's Law constant of 1.8X10-5 atm-cu m/mole. If released into water, 2,4-D, 2-ethylhexyl ester is expected to adsorb to suspended solids and sediment based upon the estimated Koc. 2,4-D, 2-ethylhexyl ester is expected to hydrolyze and form the parent compound 2,4-D acid. The estimated hydrolysis half-lives of this reaction are 35 and 3.5 days at pH values of 7 and 8, respectively. Field studies have resulted in half-lives of 1 to 51 days when applied as a spray and 4-16 days when applied in granule form. These results are similar to those found in the parent compound, 2,4-D acid. Volatilization from water surfaces is expected to be an important fate process based upon this compound's Henry's Law constant. Estimated volatilization half-lives for a model river and model lake are 94 hours and 821 hours, respectively. However, volatilization from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. The estimated volatilization half-life from a model pond is 51 months if adsorption is considered. An estimated BCF of 5,600 suggests the potential for bioconcentration in aquatic organisms is very high. Occupational exposure to 2,4-D, 2-ethylhexyl ester may occur through inhalation and dermal contact with this compound at workplaces where 2,4-D, 2-ethylhexyl ester is produced or used. (SRC)
PEER REVIEWED

Probable Routes of Human Exposure:
Occupational exposure to 2,4-D, 2-ethylhexyl ester may occur through inhalation and dermal contact with this compound at workplaces where 2,4-D, 2-ethylhexyl ester is produced or used. (SRC)
PEER REVIEWED

Artificial Pollution Sources:
2,4-D, 2-ethylhexyl ester production may result in its release to the environment through various waste streams; its use as a herbicide(1) will result in its direct release to the environment(SRC).
[(1) Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Environmental Fate:
TERRESTRIAL FATE: Based on a classification scheme(1), an estimated Koc value of 33,000(SRC), determined from a log Kow of 5.78(2) and a regression-derived equation(3), indicates that 2,4-D, 2-ethylhexyl ester is expected to be immobile in soil(SRC). Volatilization of 2,4-D, 2-ethylhexyl ester from moist soil surfaces is expected to be an important fate process(SRC) given a Henry's Law constant of 1.8X10-5 atm-cu m/mole(2). However, adsorption to soil is expected to attenuate volatilization(SRC). 2,4-D, 2-ethylhexyl ester is not expected to volatilize from dry soil surfaces(SRC) based upon a vapor pressure of 3.59X10-4 mm Hg(2). Field studies have resulted in half-lives of 1 to 51 days when applied as a spray and 4-16 days when applied in granulate form(3).
[(1) Swann RL et al; Res Rev 85: 17-28 (1983) (2) Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003) (3) Wilson RD et al; Environ Tox Chem 16: 1239-1246 (1997)]
PEER REVIEWED

AQUATIC FATE: Based on a classification scheme(1), an estimated Koc value of 33,000(SRC), determined from a log Kow of 5.78(2) and a regression-derived equation(3), indicates that 2,4-D, 2-ethylhexyl ester is expected to adsorb to suspended solids and sediment(SRC). Volatilization from water surfaces is expected(3) based upon a Henry's Law constant of 1.8X10-5 atm-cu m/mole(2). Using this Henry's Law constant and an estimation method(3), volatilization half-lives for a model river and model lake are 94 hours and 820 hours, respectively(SRC). However, volatilization from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column(SRC). The estimated volatilization half-life from a model pond is 51 months if adsorption is considered(4). According to a classification scheme(5), an estimated BCF of 5,600(SRC), from its log Kow(2) and a regression-derived equation(6), suggests the potential for bioconcentration in aquatic organisms is very high(SRC). Hydrolysis of 2,4-D, 2-ethylhexyl ester is expected to yield the parent compound 2,4-D acid(SRC). A base-catalyzed second-order hydrolysis rate constant of 2.3 L/mole-sec(SRC) was estimated using a structure estimation method(7); this corresponds to half-lives of 35 and 3.5 days at pH values of 7 and 8, respectively(7). Biodegradation data were not available(SRC, 2005).
[(1) Swann RL et al; Res Rev 85: 17-28 (1983) (2) Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003) (3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 4-9, 15-1 to 15-29 (1990) (4) US EPA; EXAMS II Computer Simulation (1987) (5) Franke C et al; Chemosphere 29: 1501-14 (1994) (6) Meylan WM et al; Environ Toxicol Chem 18: 664-72 (1999) (7) Mill T et al; Environmental Fate and Exposure Studies Development of a PC-SAR for Hydrolysis: Esters, Alkyl Halides and Epoxides. EPA Contract No. 68-02-4254. Menlo Park, CA: SRI International (1987)] **PEER REVIEWED**

ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere(1), 2,4-D, 2-ethylhexyl ester, which has a vapor pressure of 3.59X10-4 mm Hg at 25 deg C(2) is expected to exist solely as a vapor in the ambient atmosphere. Vapor-phase 2,4-D, 2-ethylhexyl ester is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals(SRC); the half-life for this reaction in air is estimated to be 27 hrs(SRC), calculated from its rate constant of 15X10-12 cu cm/molecule-sec at 25 deg C(SRC) that was derived using a structure estimation method(3). 2,4-D, 2-ethylhexyl has been reported to be stable to light(2).
[(1) Bidleman TF; Environ Sci Technol 22: 361-367 (1988) (2) Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003) (3) Meylan WM, Howard PH; Chemosphere 26: 2293-99 (1993)] **PEER REVIEWED**

Environmental Abiotic Degradation:
The rate constant for the vapor-phase reaction of 2,4-D, 2-ethylhexyl ester with photochemically-produced hydroxyl radicals has been estimated 15X10-12 cu cm/molecule-sec at 25 deg C(SRC) using a structure estimation method(1). This corresponds to an atmospheric half-life of about 27 hours at an atmospheric concentration of 5X10+5 hydroxyl radicals per cu cm(1). Hydrolysis of 2,4-D, 2-ethylhexyl ester is expected to yield the parent compound 2,4-D acid(SRC). A base-catalyzed second-order hydrolysis rate constant of 2.3 L/mole-sec(SRC) was estimated using a structure estimation method(2); this corresponds to half-lives of 35 and 3.5 days at pH values of 7 and 8, respectively(2). 2,4-D, 2-ethylhexyl has been reported to be stable to light(3).
[(1) Meylan WM, Howard PH; Chemosphere 26: 2293-99 (1993) (2) Mill T et al; Environmental Fate and Exposure Studies Development of a PC-SAR for Hydrolysis: Esters, Alkyl Halides and Epoxides. EPA Contract No. 68-02-4254. Menlo Park, CA: SRI International (1987) (3) Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Environmental Bioconcentration:
An estimated BCF of 5,600 was calculated for 2,4-D, 2-ethylhexyl ester(SRC), using a log Kow of 5.78(1) and a regression-derived equation(2). According to a classification scheme(3), this BCF suggests the potential for bioconcentration in aquatic organisms is very high(SRC), provided the compound is not altered physically or chemically once released into the environment(SRP).
[(1) Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003) (2) Meylan WM et al; Environ Toxicol Chem 18: 664-72 (1999) (3) Franke C et al; Chemosphere 29: 1501-14 (1994)] **PEER REVIEWED**

Soil Adsorption/Mobility:
The Koc of 2,4-D, 2-ethylhexyl ester is estimated as 33,000(SRC), using a log Kow of 5.78(1) and a regression-derived equation(2). According to a classification scheme(3), this estimated Koc value suggests that 2,4-D, 2-ethylhexyl ester is expected to be immobile in soil.
[(1) Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003) (2) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 4-9 (1990) (3) Swann RL et al; Res Rev 85: 17-28 (1983)] **PEER REVIEWED**

Volatilization from Water/Soil:
The Henry's Law constant for 2,4-D, 2-ethylhexyl ester is 1.8X10-5atm-cu m/mole(1). This Henry's Law constant indicates that 2,4-D, 2-ethylhexyl ester is expected to volatilize from water surfaces(2). Based on this Henry's Law constant, the volatilization half-life from a model river (1 m deep, flowing 1 m/sec, wind velocity of 3 m/sec)(2) is estimated as 94.4 hours(SRC). The volatilization half-life from a model lake (1 m deep, flowing 0.05 m/sec, wind velocity of 0.5 m/sec)(2) is

estimated as 34.2 days(SRC). However, volatilization from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. The estimated volatilization half-life from a model pond is 51 months when adsorption is considered(3). Volatilization of 2,4-D, 2-ethylhexyl ester from moist soil surfaces is expected to be an important fate process(SRC) given a Henry's Law constant of 1.8X10-5 atm-cu m/mole(1). However, adsorption to soil is expected to attenuate volatilization(SRC). 2,4-D, 2-ethylhexyl ester is not expected to volatilize from dry soil surfaces(SRC) based upon its vapor pressure of 3.59X10-4 mm Hg(1).
[(1) Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003) (2) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 15-1 to 15-29 (1990) (3) US EPA; EXAMS II Computer Simulation (1987)] **PEER REVIEWED**

Environmental Standards & Regulations:

FIFRA Requirements:
Tolerances are established for residues of 2,4-D at: barley, grain; blueberry; corn, forage; corn, fresh, sweet, kernel plus cob with husk removed; corn, grain; corn, stover; cranberry; fruit, stone; grapes; grass hay; grasses, pasture; grasses, rangeland; millet, forage; millet, grain; millet, straw; nut; oat, forage; oat, grain; pistachio; rice, grain; rice, straw; rye, forage; rye, grain: sorghum, forage; sorghum, grain; sorghum, grain, stover; sugarcane, cane; sugarcane, forage; wheat, forage; and wheat, grain. (Residues on all the above may result from application of 2,4-D in acid form, or in the form of one or more of the following esters: amyl (pentyl), butoxyethoxypropyl, butoxyethyl, butoxypolythylene glycol butyl ether, butoxypropyl, butyl, dipropylene glycol isobutyl ether, ethoxyethoxyethyl, ethoxyethoxypropyl, ethyl, ethoxypropyl, isobutyl, isooctyl (including, but not limited to, 2-ethylhexyl, 2-ethyl-4-methylpentyl, and 2-octyl), isopropyl, methyl, polyethylene glycol 200, polypropoxybutyl, polypropylene glycol, propylene glycol, propylene glycol butyl ether, propylene glycol isobutyl ether, tetrahydrofurfuryl, and tripropylene glycol isobutyl ether.)
[40 CFR 180.142(a)(2); U.S. National Archives and Records Administration's Electronic Code of Federal Regulations. Available from, as of February 1, 2005: http://www.gpoaccess.gov/ecfr **PEER REVIEWED**

As the federal pesticide law FIFRA directs, EPA is conducting a comprehensive review of older pesticides to consider their health and environmental effects and make decisions about their future use. Under this pesticide reregistration program, EPA examines health and safety data for pesticide active ingredients initially registered before November 1, 1984, and determines whether they are eligible for reregistration. In addition, all pesticides must meet the new safety standard of the Food Quality Protection Act of 1996. Isooctyl(2-ethylhexyl) 2,4-dichlorophenoxyacetate is found on List A, which contains most food use pesticides and consists of the 194 chemical cases (or 350 individual active ingredients) for which EPA issued registration standards prior to FIFRA '88. Case No: 0073; Pesticide type: fungicide, herbicide (growth regulator); Registration Standard Date: 9/1/88 PB89-102396; Case Status: OPP is reviewing data from the pesticide's producers regarding its human health and/or environmental effects, or OPP is determining the pesticide's eligibility for reregistration and developing the RED document.; Active ingredient (AI): isooctyl(2-ethylhexyl) 2,4-dichlorophenoxyacetate; Data Call-in (DCI) Date(s): 3/25/94; AI Status: The producers of the pesticide have made commitments to conduct the studies and pay the fees required for reregistration, and are meeting those commitments in a timely manner. /RED scheduled for May 2005/
[United States Environmental Protection Agency/ Prevention, Pesticides and Toxic Substances; Status of Pesticides in Registration, Reregistration, and Special Review. (1998) EPA 738-R-98-002, p. 71] **PEER REVIEWED**

Allowable Tolerances:
Tolerances are established for residues of 2,4-D at: barley, grain: 0.5 ppm; blueberry: 0.1 ppm; corn, forage: 20 ppm; corn, fresh, sweet, kernel plus cob with husk removed: 0.5 ppm; corn, grain: 0.5 ppm; corn, stover: 20 ppm; cranberry: 0.5 ppm; fruit, stone: 0.2 ppm; grapes: 0.5 ppm; grass hay: 300 ppm; grasses, pasture: 1,000 ppm; grasses, rangeland: 1,000 ppm; millet, forage: 20 ppm; millet, grain: 0.5 ppm; millet, straw: 20 ppm; nut: 0.2 ppm; oat, forage: 20 ppm; oat, grain: 0.5 ppm; pistachio: 0.2 ppm; rice, grain: 0.1 ppm; rice, straw: 20 ppm; rye, forage: 20 ppm; rye, grain: 0.5 ppm: sorghum, forage: 20 ppm; sorghum, grain: 0.5 ppm; sorghum, grain, stover: 20 ppm; sugarcane, cane: 2 ppm; sugarcane, forage: 20 ppm; wheat, forage: 20 ppm; and wheat, grain: 0.5 ppm. (Residues on all the above may result from application of 2,4-D in acid form, or in the form of one or more of the following esters: amyl (pentyl), butoxyethoxypropyl, butoxyethyl, butoxypolythylene glycol butyl ether, butoxypropyl, butyl, dipropylene glycol isobutyl ether, ethoxyethoxyethyl, ethoxyethoxypropyl, ethyl, ethoxypropyl, isobutyl, isooctyl (including, but not limited to, 2-ethylhexyl, 2-ethyl-4-methylpentyl, and 2-octyl), isopropyl, methyl, polyethylene glycol 200, polypropoxybutyl, polypropylene glycol, propylene glycol, propylene glycol butyl ether, propylene glycol isobutyl ether, tetrahydrofurfuryl, and tripropylene glycol isobutyl ether.)
[40 CFR 180.142(a)(2); U.S. National Archives and Records Administration's Electronic Code of Federal Regulations. Available from, as of February 1, 2005: http://www.gpoaccess.gov/ecfr **PEER REVIEWED**

Chemical/Physical Properties:

Molecular Formula:

C16-H22-Cl2-O3

[National Library of Medicine, SIS; ChemIDplus Record for 2,4-D-2-ethylhexyl (1928-43-4). Available from, as of March 2, 2005: http://chem.sis.nlm.nih.gov/chemidplus/direct.jsp?regno=1928-43-4 **PEER REVIEWED**

Molecular Weight:

333.28

[Lewis, R.J. Sax's Dangerous Properties of Industrial Materials. 10th ed. Volumes 1-3 New York, NY: John Wiley & Sons Inc., 1999., p. V2: 1103] **PEER REVIEWED**

Color/Form:

Golden yellow, non viscous liquid

[Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Odor:

Sweet slightly pungent odor

[Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Boiling Point:

>300 deg C (decomp)

[Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Melting Point:

<-37 deg C

[Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Density/Specific Gravity:

1.148 at 20 deg C

[Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Octanol/Water Partition Coefficient:

log Kow = 5.78 at 25 deg C

[Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Solubilities:

In water, 0.086 mg/L at 25 deg C

[Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Vapor Pressure:

47.9 mPa /3.59X10-4 mm Hg/ at 25 deg C (Calculated)

[Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Other Chemical/Physical Properties:

In water, 0.0324 mg/L

[Ahrens, W.H. Herbicide Handbook of the Weed Science Society of America. 7th ed. Champaign, IL: Weed Science Society of America, 1994., p. 79] **PEER REVIEWED**

Henry's Law constant = 1.8 Pa cu m/mol (1.8X10-5 atm-cu m/mol)

[Tomlin CDS, ed; The e-Pesticide Manual. 2,4-D-2-ethylhexyl. 13th ed. PC CD-ROM, Version 3.0, 2003-04. Surrey, UK: British Crop Protection Council (2003)] **PEER REVIEWED**

Hydroxyl radical reaction rate constant = 15X10-12 cu cm/molec-sec at 25 deg C /Estimated/

[US EPA; Estimation Programs Interface (EPI). ver. 3.11. U.S. EPA version for Windows. Washington, DC: U.S. EPA (2003). Available from,

as of Dec 15, 2004: http://www.epa.gov/oppt/exposure/pubs/episuitedl.htm **PEER REVIEWED**

Chemical Safety & Handling:

Flash Point:

171 deg C (Cleveland open cup)
[Tomlin CDS, ed. 2,4-D-2-ethylhexyl (1928-43-4). In: The e-Pesticide Manual, 13th Edition Version 3.0 (2003-04). Surrey UK, British Crop Protection Council.] **PEER REVIEWED**

Stability/Shelf Life:

Hydrolysis DT50 <1 hr. Stable to light, DT50 >100 days. Stable at 54 deg C.
[Tomlin CDS, ed. 2,4-D-2-ethylhexyl (1928-43-4). In: The e-Pesticide Manual, 13th Edition Version 3.0 (2003-04). Surrey UK, British Crop Protection Council.] **PEER REVIEWED**

Disposal Methods:

SRP: The most favorable course of action is to use an alternative chemical product with less inherent propensity for occupational exposure or environmental contamination. Recycle any unused portion of the material for its approved use or return it to the manufacturer or supplier. Ultimate disposal of the chemical must consider: the material's impact on air quality; potential migration in soil or water; effects on animal, aquatic, and plant life; and conformance with environmental and public health regulations.
PEER REVIEWED

Occupational Exposure Standards:

Manufacturing/Use Information:

Major Uses:

For 2,4-D, 2-ethylhexyl ester (USEPA/OPP Pesticide Code: 030063) ACTIVE products with label matches. /SRP: Registered for use in the U.S. but approved pesticide uses may change periodically and so federal, state and local authorities must be consulted for currently approved uses./
[U.S. Environmental Protection Agency/Office of Pesticide Program's Chemical Ingredients Database on 2,4-D, 2-Ethylhexyl Ester (1928-43-4). Available from, as of February 1, 2005: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Manufacturers:

Dow Agrosciences LLC, 9330 Zionsville Rd., Indianapolis, IN 46268, (317) 337-3000; Production site: Midland, MI 48667 /2,4-D and esters and salts/
[SRI Consulting. 2004 Directory of Chemical Producers. SRI International, Menlo Park, CA 2004., p. 766] **PEER REVIEWED**

Nufarm, Inc., 1333 Burr Ridge Pkwy., Suite 125A, Burr Ridge, IL 60521-0866, (800) 345-3330; Production site: Burr Ridge, IL 60521-0866 /2,4-D and esters and salts/
[SRI Consulting. 2004 Directory of Chemical Producers. SRI International, Menlo Park, CA 2004., p. 766] **PEER REVIEWED**

Riverdale (a Nufarm Co.), 1333 Burr Ridge Pkwy., Suite 125A, Burr Ridge, IL 60521-0866, (800) 345-3330; Production site: Chicago Heights, IL 60411 /2,4-D and esters and salts/
[SRI Consulting. 2004 Directory of Chemical Producers. SRI International, Menlo Park, CA 2004., p. 766] **PEER REVIEWED**

Agriliance LLC, 64089 St. Paul, MN 55164-0089, 712-234-2853 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Agsco, 13458, Grand Forks, ND 58208-3458, 701-775-532 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Albaugh Inc., 2127, Valdosta, GA 31604-2127, 229-244-3288 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

AMREP Inc., 990 Industrial Dr., Marietta, GA 30062, 770-422-2071 /Registrant/

[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

AMVAC Chemical Corp., 4695 Macarthur Court, Suite 1250, Newport Beach, CA 92660-1706, 949-260-1212; Athea Laboratories Inc., 240014. Milwaukee, WI 53224, 800-743-6417 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Atanor S.A., 2127 Valdosta, GA 31604-2127 229-244-3288 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Bayer Cropscience LP, 2 T.W. Alexander Drive, Research Triangle Park, NC 27709, 919-549-2365 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Helena Chemical Co., 225 Schilling Blvd., Suite 300, Collierville, TN 38017 901-752-4410 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Hill Manufacturing Corp.,1500 Jonesboro Rd., SE Atlanta, GA 30315 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Loveland Products Inc., 1286, Greeley, CO 80632, 970-347-1470 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Lubar Chemical Co., 208 Iron North, Kansas City, MO 64116, 816-472-5515 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Micro-Flo Co., LLC, 530 Oak Court Dr., Memphis TN 38117 901-432-5000 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Nufarm Limited, 2300 Frederick Ave., Suite 208, St. Joseph, MO 64504, 816-676-9000 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

PBI/Gordon Corp., 014090, 1217 West 12th St., Kansas City, MO 64101-0090, 816-460-6292. /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Rockland Chemical Corp., 71 Carolyn Blvd., Farmingdale, NY 11735, 978-887-1424 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Universal Cooperatives Inc., 1300 Corporate Center Curve, Eagan, MN 55121, 651-239-1128 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Value Gardens Supply, 585, St. Joseph, MO 64502, 540-864-8100 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Voluntary Purchasing Group Inc., 1806 Auburn Dr., Carrollton, TX 75007-1451, 972-939-8390 /Registrant/
[US EPA; USEPA/OPP Pesticide Related Database Queries. Chemical Ingredient Database on 2,4-D, 2-ethylhexyl ester. (1928-43-4). Available from, as of Dec 22, 2004: http://ppis.ceris.purdue.edu/htbin/epachem.com **PEER REVIEWED**

Formulations/Preparations:

Selected products: 'Esteron 6E'; 'Esteron 99C'; 'Lentemul'; ...'Barrage'; 'Brush-Rhap'; 'Fivestar'; 'Low Vol 4 Ester'; 'Salvo'; 'Weed Rhap LV-4D'; 'Weedone LV4'; 'Weed-Rhap'. Mixtures: 'Adrenalin' (+ imazamox); 'B-4' (+bromoxynil heptanoate+ bromoxynil octanoate); 'Broadsword' (+dicamba+ triclopyr-butotyl) (dicamba as butotyl ester); 'Oasis' (+imazapic); 'Shotgun' (+atrazine); 'Tiller' (+fenoxaprop-P-ethyl+ MCPA-2-ethylhexyl); 'Weedone 638 Solventless' (+2,4-D).
[Tomlin CDS, ed. 2,4-D-2-ethylhexyl (1928-43-4). In: The e-Pesticide Manual, 13th Edition Version 3.0 (2003-04). Surrey UK, British Crop Protection Council.] **PEER REVIEWED**

Laboratory Methods:

Analytic Laboratory Methods:
Method: 8321A: Procedure: high performace liquid chromatography coupled with either thermospray-mass spectrometry and/or ultraviolet detection; Analyte: 2,4-D, ethylhexyl ester; Matrix: wastewater, ground water, and soil/sediment matrices; Detection Limit: 1.2 ng.
[[U.S. Environmental Protection Agency. Solid Waste Test Methods SW-846 with Update III. CD-ROM (ISO 9660, V381SW8). Solutions Software Corp (1998)]] **PEER REVIEWED**

Special References:

Synonyms and Identifiers:

Related HSDB Records:
202 [2,4-D] (hydrolysis product)

Synonyms:
USEPA/OPP Pesticide Code: 030063
PEER REVIEWED

Isooctyl(2-ethylhexyl) 2,4-dichlorophenoxyacetate
PEER REVIEWED

2,4-D, 2-Ethylhexyl
PEER REVIEWED

2-Ethylhexyl (2,4-dichlorophenoxy)acetate
PEER REVIEWED

Acetic acid, (2,4-dichlorophenoxy)-, 2-ethylhexyl ester
PEER REVIEWED

(2,4-Dichlorophenoxy)acetic acid 2-ethylhexyl ester
PEER REVIEWED

Formulations/Preparations:
Selected products: 'Esteron 6E'; 'Esteron 99C'; 'Lentemul'; ...'Barrage'; 'Brush-Rhap'; 'Fivestar'; 'Low Vol 4 Ester'; 'Salvo'; 'Weed Rhap LV-4D'; 'Weedone LV4'; 'Weed-Rhap'. Mixtures: 'Adrenalin' (+ imazamox); 'B-4' (+bromoxynil heptanoate+ bromoxynil octanoate); 'Broadsword' (+dicamba+ triclopyr-butotyl) (dicamba as butotyl ester); 'Oasis' (+imazapic); 'Shotgun' (+atrazine); 'Tiller' (+fenoxaprop-P-ethyl+ MCPA-2-ethylhexyl); 'Weedone 638 Solventless' (+2,4-D).
[Tomlin CDS, ed. 2,4-D-2-ethylhexyl (1928-43-4). In: The e-Pesticide Manual, 13th Edition Version 3.0 (2003-04). Surrey UK, British Crop Protection Council.] **PEER REVIEWED**

Administrative Information:

Hazardous Substances Databank Number: 7309

Last Revision Date: 20051114

Last Review Date: Reviewed by SRP on 5/5/2005

Update History:
Field Update on 2012-04-07, 1 fields added/edited/deleted
Field Update on 2012-04-07, 1 fields added/edited/deleted
Field Update on 2012-04-07, 1 fields added/edited/deleted
Field Update on 2012-04-07, 1 fields added/edited/deleted
Complete Update on 2005-11-14, 36 fields added/edited/deleted
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DEPARTMENT OF TOXIC SUBSTANCES CONTROL
ENVIROSTOR

MOUNTAIN VIEW MIDDLE SCHOOL EXPANSION (60000825)

SIGN UP FOR EMAIL ALERTS

13130 MORRISON AVENUE
MORENO VALLEY, CA 92555
RIVERSIDE COUNTY
SITE TYPE: SCHOOL

SUPERVISOR: SHAHIR HADDAD
OFFICE: SOUTHERN CALIFORNIA SCHOOLS & BROWNFIELDS OUTREACH
SCHOOL DISTRICT: MORENO VALLEY UNIFIED SCHOOL DISTRICT

Site Information

CLEANUP STATUS
NO FURTHER ACTION AS OF 6/16/2008

SITE TYPE: SCHOOL
NATIONAL PRIORITIES LIST: NO
ACRES: 0.42 ACRES
APN: NONE SPECIFIED
CLEANUP OVERSIGHT AGENCIES:
DTSC - SITE CLEANUP PROGRAM - LEAD

SCHOOL DISTRICT: MORENO VALLEY UNIFIED SCHOOL DISTRICT
ENVIROSTOR ID: 60000825
SITE CODE: 404779
SPECIAL PROGRAM:
FUNDING: SCHOOL DISTRICT
ASSEMBLY DISTRICT: 61
SENATE DISTRICT: 31

Regulatory Profile

PAST USE(S) THAT CAUSED CONTAMINATION
AGRICULTURAL - ROW CROPS, SCHOOL - MIDDLE

POTENTIAL CONTAMINANTS OF CONCERN
ARSENIC
ORGANOCHLORINE PESTICIDES (8081 OCPS)

POTENTIAL MEDIA AFFECTED
SOIL

Site History

The Site comprises approximately 0.42-acres within the existing Mountain View Middle School property. The Site has been historically used for agricultural purposes from approximately 1938 to 1980. The school was constructed in 1980. Surrounding properties consist of Valley View High School to the east, and residential to the north, south, and west. To evaluate the impact from historical operations, the site was investigated for arsenic and organochlorine pesticides. DTSC concurred with the conclusion in the PEA that no further action is necessary for the Site.

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DEPARTMENT OF TOXIC SUBSTANCES CONTROL
ENVIROSTOR

PROPOSED HIGH SCHOOL (60000931)

SIGN UP FOR EMAIL ALERTS

IRONWOOD / QUINCY
MORENO VALLEY, CA 92555
RIVERSIDE COUNTY
SITE TYPE: SCHOOL

SUPERVISOR: SHAHIR HADDAD
OFFICE: SOUTHERN CALIFORNIA SCHOOLS & BROWNFIELDS OUTREACH
SCHOOL DISTRICT: MORENO VALLEY UNIFIED SCHOOL DISTRICT

Site Information

CLEANUP STATUS
NO FURTHER ACTION AS OF 10/23/2008

SITE TYPE: SCHOOL
NATIONAL PRIORITIES LIST: NO
ACRES: 56 ACRES
APN: NONE SPECIFIED
CLEANUP OVERSIGHT AGENCIES:
DTSC - SITE CLEANUP PROGRAM - LEAD

SCHOOL DISTRICT: MORENO VALLEY UNIFIED SCHOOL DISTRICT
ENVIROSTOR ID: 60000931
SITE CODE: 404806
SPECIAL PROGRAM:
FUNDING: SCHOOL DISTRICT
ASSEMBLY DISTRICT: 61
SENATE DISTRICT: 31

Regulatory Profile

PAST USE(S) THAT CAUSED CONTAMINATION
AGRICULTURAL - ROW CROPS

POTENTIAL CONTAMINANTS OF CONCERN
ARSENIC
ORGANOCHLORINE PESTICIDES (8081 OCPS)

POTENTIAL MEDIA AFFECTED
SOIL

Site History

The Site is approximately 55.6-acres and has historically been used for agricultural purposes since 1938. Surrounding properties consist of vacant land to the north, residential properties to the east, residential and agricultural properties to the west (across Quincy Wash), and residential and agricultural properties to the south (across Ironwood Avenue). To evaluate the impact from historical operations, the site was investigated for arsenic, copper and organochlorine pesticides. The PEA concludes that no further action is necessary for the Site. DTSC concurred with a No Further Action determination.

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TOXICOLOGICAL PROFILE FOR
DDT, DDE, and DDD

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

September 2002

Large amounts of DDT were released into the air and on soil or water when it was sprayed on crops and forests to control insects. DDT was also sprayed in the environment to control mosquitos. Although the use of DDT is no longer permitted in the United States, DDT may be released into the atmosphere in other countries where it is still manufactured and used, including Mexico. DDT, DDE and DDD may also enter the air when they evaporate from contaminated water and soil. DDT, DDE, and DDD in the air will then be deposited on land or surface water. This cycle of evaporation and deposition may be repeated many times. As a result, DDT, DDE, and DDD can be carried long distances in the atmosphere. These chemicals have been found in bogs, snow, and animals in the Arctic and Antarctic regions, far from where they were ever used. Some DDT may have entered the soil from waste sites. DDT, DDE, and DDD may occur in the atmosphere as a vapor or be attached to solids in air. Vapor phase DDT, DDE, and DDD may break down in the atmosphere due to reactions caused by the sun. The half-life of these chemicals in the atmosphere as vapors (the time it takes for one-half of the chemical to turn into something else) has been calculated to be approximately 1.5–3 days. However, in reality, this half-life estimate is too short to account for the ability of DDT, DDE, and DDD to be carried long distances in the atmosphere.

DDT, DDE, and DDD last in the soil for a very long time, potentially for hundreds of years. Most DDT breaks down slowly into DDE and DDD, generally by the action of microorganisms. These chemicals may also evaporate into the air and be deposited in other places. They stick strongly to soil, and therefore generally remain in the surface layers of soil. Some soil particles with attached DDT, DDE, or DDD may get into rivers and lakes in runoff. Only a very small amount, if any, will seep into the ground and get into groundwater. The length of time that DDT will last in soil depends on many factors including temperature, type of soil, and whether the soil is wet. DDT lasts for a much shorter time in the tropics where the chemical evaporates faster and where microorganisms degrade it faster. DDT disappears faster when the soil is flooded or wet than when it is dry. DDT disappears faster when it initially enters the soil. Later on, evaporation slows down and some DDT moves into spaces in the soil that are so small that microorganisms cannot reach the DDT to break it down efficiently. In tropical areas, ΣDDT may disappear in much less than a year. In temperate areas, half of the ΣDDT initially present

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http://www.epa.gov/ttn/atw/hlthef/dde.html
Last updated on Tuesday, November 06, 2007

Technology Transfer Network
Air Toxics Web Site

DDE

DDE (1,1-DICHLORO-2,2-BIS(p-CHLOROPHENYL) ETHYLENE) (A)

72-55-9

Hazard Summary-Created in April 1992; Revised in January 2000

1,1-Dichloro-2,2-bis(p-chlorophenyl) ethylene (DDE) is a breakdown product of DDT, which was used in the past as an insecticide. No information is available on the acute (short-term) or chronic (long-term) effects of DDE. Acute, oral exposure to high doses of DDT in humans results in central nervous system (CNS) effects, such as headaches, nausea, and convulsions. The only effect noted in epidemiologic studies of workers exposed to DDT and other pesticides was an increase in activity of liver enzymes. Animal studies have reported effects on the liver, immune system, and CNS from chronic oral exposure to DDT. Human studies are inconclusive regarding DDE and cancer. Animal studies have reported an increased incidence of liver tumors in mice and hamsters, and thyroid tumors in female rats from oral exposure to DDE. EPA has classified DDE as a Group B2, probable human carcinogen.

Please Note: The main source of information for this fact sheet is the Agency for Toxic Substances and Disease Registry's (ATSDR's) *Toxicological Profile for 4,4-DDT, 4,4-DDE, and 4,4-DDD* and EPA's Integrated Risk Information System (IRIS), which contains information on the carcinogenic effects of DDE including the unit cancer risk for oral exposure.

Uses

- DDT was extensively used in the past for the control of malaria, typhus, and other insect-transmitted diseases. It was banned for use in the United States in 1972, except in the case of a public health emergency. (1)
- DDE is a breakdown product of DDT and has no uses. (1)

Sources and Potential Exposure

- DDE is found in the environment as a result of the breakdown of DDT, an insecticide. (1)
- Human exposure to DDE appears to be primarily through food; in the United States in 1981, consumption of DDE in foods was estimated to be 0.001 parts per million per day (ppm/d). However, the levels of DDE in foods have been decreasing and are expected to continue to decrease. (1)
- Levels of DDE in air and water samples are very low. (1)
- DDE has been listed as a pollutant of concern to EPA's Great Waters Program due to its persistence in the environment, potential to bioaccumulate, and toxicity to humans and the environment (2).

Assessing Personal Exposure

- DDE can be detected in fat, blood, urine, semen, and breast milk. (1)

Health Hazard Information

Acute Effects:

- No studies are available on the acute effects of DDE in humans. (1)
- Acute oral exposure to high doses of DDT in humans results in CNS effects, such as headaches, nausea, and convulsions. (1)
- Case reports in humans have noted that doses as high as 285 milligrams DDT per kilogram body weight per day (mg/kg/d) have been ingested accidentally with no fatal results. (1)
- Tests involving acute exposure of rats, guinea pigs, and rabbits have shown DDT to have moderate acute toxicity from oral exposure. (3)

Chronic Effects (Noncancer):

- The only effect noted in epidemiologic studies of workers exposed to DDT and other pesticides was an increase in activity of liver enzymes. No adverse effects on the blood, liver, heart, or CNS were noted. (1)
- Animal studies have reported effects on the liver, immune system, and CNS from chronic oral administration of DDT. (1,4,9)
- EPA has not established a Reference Concentration (RfC) or a Reference Dose (RfD) for DDE. (5)
- EPA has established an RfD of 0.0005 milligrams per kilogram body weight per day (mg/kg/d) for DDT based on liver effects in rats. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous ingestion exposure to the human population (including sensitive subgroups), that is likely to be without appreciable risk of deleterious noncancer effects during a lifetime. It is not a direct estimator of risk but rather a reference point to gauge the potential effects. At exposures increasingly greater than the RfD, the potential for adverse health effects increases. Lifetime exposure above the RfD does not imply that an adverse health effect would necessarily occur. (5)

Reproductive/Developmental Effects:

- No information is available on the reproductive or developmental effects of DDT or DDE in humans via inhalation exposure. (1)
- No studies are available on the developmental effects in humans after oral exposure to DDT or DDE. However, DDT and DDE have been found in human blood, placental tissue, and umbilical cord blood. (1)
- Epidemiologic studies did not find an association between DDT maternal blood levels and miscarriages or premature rupture of fetal membranes in humans. (1)
- Oral animal studies have reported reproductive effects, such as reduced fertility, adverse effects on spermatogenesis, and decreased testicular and ovarian weights from DDT exposure. Developmental effects, such as embryotoxicity and fetotoxicity, but not teratogenicity (birth defects) have also been observed in oral animal studies. (1)
- DDT has been shown to elicit estrogenic activity in rats after oral exposure (1).

Cancer Risk:

- Studies of workers exposed to DDT have yielded conflicting results. Three studies reported that tissue levels of DDT and DDE were higher in cancer victims than in those dying of other diseases. In other studies, no such relationship was seen. (5,9)
- Animal studies have reported an increased incidence of liver tumors in mice and hamsters and thyroid tumors in female rats from oral exposure to DDE. (5)
- EPA has classified DDE as a Group B2, probable human carcinogen. (5)
- EPA uses mathematical models, based on animal studies to estimate the probability of a person developing cancer from ingesting water containing a specified concentration of a chemical. EPA has calculated an oral cancer slope factor of 0.34 $(mg/kg/d)^{-1}$ and a unit risk estimate of 9.7×10^{-6} $(\mu g/L)^{-1}$. EPA estimates that, if an individual were to continuously ingest water containing an average of DDE at 0.1 µg/L over his or her entire lifetime, that person would theoretically have no more than a one-in-a-million increased chance of developing cancer as a direct result of ingesting water containing this chemical. Similarly, EPA estimates that ingesting water containing 1.0 µg/L would result in not greater than a one-in-a-hundred-thousand increased chance of developing cancer, and water containing 10.0 µg/L would result in not greater than a one-in-ten thousand increased chance of developing cancer. For a detailed discussion of confidence in the potency estimates, please see IRIS. (5)

Physical Properties

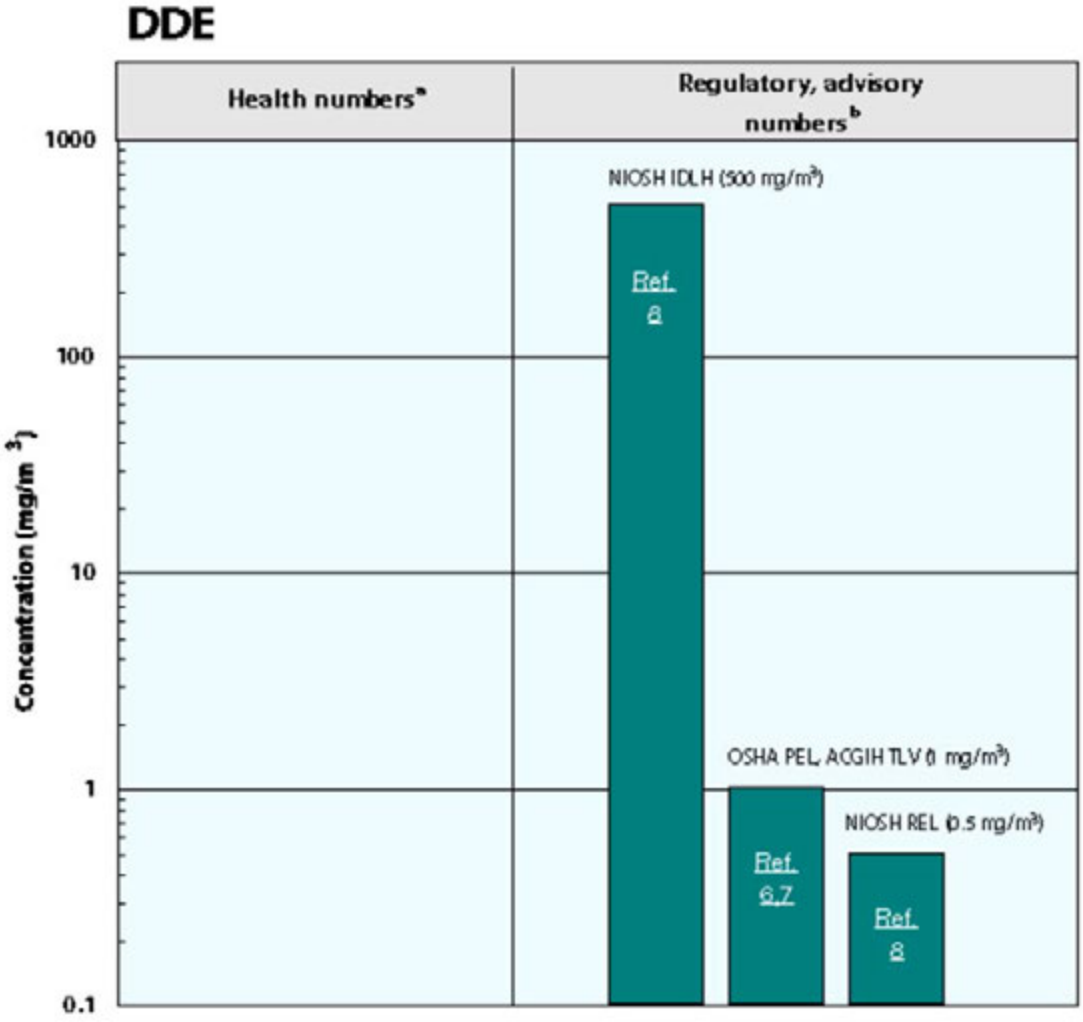
- DDE is also known as 1,1-dichloro-2,2-bis(p-chlorophenyl) ethylene and *p,p*-dichlorodiphenyldichloroethylene.

- DDE is a white crystalline solid. (1)
- The odor threshold for DDE is not available. (1)
- The chemical formula for DDE is $C_{14}H_8Cl_4$, and the molecular weight is 318.03 g/mol. (1)
- The vapor pressure for DDE is 6.5×10^{-6} torr at 20 °C, and it has a log octanol/water partition coefficient (log K_{ow}) of 7.0. (1)

Conversion Factors:

To convert concentrations in air (at 25 °C) from ppm to mg/m^3: mg/m^3 = (ppm) × (molecular weight of the compound)/(24.45). For DDE: 1 ppm = 13.0 mg/m^3; for DDT: 1 ppm = 14.5 mg/m^3.

Health Data from Inhalation Exposure*



ACGIH TLV--American Conference of Governmental and Industrial Hygienists' threshold limit value expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effects.
NIOSH IDLH--National Institute of Occupational Safety and Health's immediately dangerous to life or health limit; NIOSH recommended exposure limit to ensure that a worker can escape from an exposure condition that is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from the environment.
NIOSH REL--NIOSH's recommended exposure limit; NIOSH-recommended exposure limit for an 8- or 10-h time-weighted-average exposure and/or ceiling.

OSHA PEL--Occupational Safety and Health Administration's permissible exposure limit expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effect averaged over a normal 8-h workday or a 40-h workweek.

* All health and regulatory numbers are for DDT.
The health and regulatory values cited in this fact sheet were obtained in December 1999.
[a] Health numbers are toxicological numbers from animal testing or risk assessment values developed by EPA.
[b] Regulatory numbers are values that have been incorporated in Government regulations, while advisory numbers are nonregulatory values provided by the Government or other groups as advice. OSHA numbers are regulatory, whereas NIOSH and ACGIH numbers are advisory.

References

1. Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for 4,4-DDT, 4,4-DDE, and 4,4-DDD*. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA. 1994.
2. U.S. Environmental Protection Agency. *Deposition of Air Pollutants to the Great Waters*. EPA-453/R-93-055. First Report to Congress. Office of Air Quality Planning and Standards, Research Triangle Park, NC. 1994.
3. U.S. Department of Health and Human Services. Registry of Toxic Effects of Chemical Substances (RTECS, online database). National Toxicology Information Program, National Library of Medicine, Bethesda, MD. 1993.
4. U.S. Department of Health and Human Services. Hazardous Substances Data Bank (HSDB, online database). National Toxicology Information Program, National Library of Medicine, Bethesda, MD. 1993.
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A. This fact sheet focuses on the health effects of DDE. However, since DDE is a breakdown product of DDT, in those cases where no information is available on DDE and there is information on DDT, the information on DDT is presented.

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ATSDR Agency for Toxic Substances & Disease Registry

ToxFAQs™ for DDT, DDE, and DDD

(*DDT, DDE y DDD* (/es/toxfaqs/es_tfacts35.html))

September 2002

CAS#: DDT 50-29-3; DDE 72-55-9; DDD 72-54-8

(/tfacts35.pdf) **PDF Version, 55 KB** (/tfacts35.pdf)

This fact sheet answers the most frequently asked health questions about DDT, DDE, and DDD. For more information, you may call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. 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.

Highlights

Exposure to DDT, DDE, and DDD occurs mostly from eating foods containing small amounts of these compounds, particularly meat, fish and poultry. High levels of DDT can affect the nervous system causing excitability, tremors and seizures. In women, DDE can cause a reduction in the duration of lactation and an increased chance of having a premature baby. DDT, DDE, and DDD have been found in at least 441 of the 1,613 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What are DDT, DDE, and DDD?

DDT (dichlorodiphenyltrichloroethane) is a pesticide once widely used to control insects in agriculture and insects that carry diseases such as malaria. DDT is a white, crystalline solid with no odor or taste. Its use in the U.S. was banned in 1972 because of damage to wildlife, but is still used in some countries.

DDE (dichlorodiphenyldichloroethylene) and DDD (dichlorodiphenyldichloroethane) are chemicals similar to DDT that contaminate commercial DDT preparations. DDE has no commercial use. DDD was also used to kill pests, but its use has also been banned. One form of DDD has been used medically to treat cancer of the adrenal gland.

What happens to DDT, DDE, and DDD when they enter the environment?

- DDT entered the environment when it was used as a pesticide; it still enters the environment due to current use in other countries.
- DDE enters the environment as contaminant or breakdown product of DDT; DDD also enters the environment as a breakdown product of DDT.
- DDT, DDE, and DDD in air are rapidly broken down by sunlight. Half of what's in air breaks down within 2 days.
- They stick strongly to soil; most DDT in soil is broken down slowly to DDE and DDD by microorganisms; half the DDT in soil will break down in 2-15 years, depending on the type of soil.
- Only a small amount will go through the soil into groundwater; they do not dissolve easily in

water.
- DDT, and especially DDE, build up in plants and in fatty tissues of fish, birds, and other animals.

How might I be exposed to DDT, DDE, and DDD?

- Eating contaminated foods, such as root and leafy vegetable, fatty meat, fish, and poultry, but levels are very low.
- Eating contaminated imported foods from countries that still allow the use of DDT to control pests.
- Breathing contaminated air or drinking contaminated water near waste sites and landfills that may contain higher levels of these chemicals.
- Infants fed on breast milk from mothers who have been exposed.
- Breathing or swallowing soil particles near waste sites or landfills that contain these chemicals.

How can DDT, DDE, and DDD affect my health?

DDT affects the nervous system. People who accidentally swallowed large amounts of DDT became excitable and had tremors and seizures. These effects went away after the exposure stopped. No effects were seen in people who took small daily doses of DDT by capsule for 18 months.

A study in humans showed that women who had high amounts of a form of DDE in their breast milk were unable to breast feed their babies for as long as women who had little DDE in the breast milk. Another study in humans showed that women who had high amounts of DDE in breast milk had an increased chance of having premature babies.

In animals, short-term exposure to large amounts of DDT in food affected the nervous system, while long-term exposure to smaller amounts affected the liver. Also in animals, short-term oral exposure to small amounts of DDT or its breakdown products may also have harmful effects on reproduction.

How likely are DDT, DDE, and DDD to cause cancer?

Studies in DDT-exposed workers did not show increases in cancer. Studies in animals given DDT with the food have shown that DDT can cause liver cancer.

The Department of Health and Human Services (DHHS) determined that DDT may reasonable be anticipated to be a human carcinogen. The International Agency for Research on Cancer (IARC) determined that DDT may possibly cause cancer in humans. The EPA determined that DDT, DDE, and DDD are probable human carcinogens.

How can DDT, DDE, and DDD affect children?

There are no studies on the health effects of children exposed to DDT, DDE, or DDD. We can assume that children exposed to large amounts of DDT will have health effects similar to the effects seen in adults. However, we do not know whether children differ from adults in their susceptibility to these substances.

There is no evidence that DDT, DDE, or DDD cause birth defects in people. A study showed that teenage boys whose mothers had higher DDE amounts in the blood when they were pregnant were taller than those whose mothers had lower DDE levels. However, a different study found the opposite in preteen girls. The reason for the discrepancy between these studies is unknown.

Studies in rats have shown that DDT and DDE can mimic the action of natural hormones and in this way affect the development of the reproductive and nervous systems. Puberty was delayed in male

rats given high amounts of DDE as juveniles. This could possibly happen in humans. A study in mice showed that exposure to DDT during the first weeks of life may cause neurobehavioral problems later in life.

How can families reduce the risk of exposure to DDT, DDE, and DDD?

- Most families will be exposed to DDT by eating food or drinking liquids contaminated with small amounts of DDT.
- Cooking will reduce the amount of DDT in fish.
- Washing fruit and vegetables will remove most DDT from their surface.
- Follow health advisories that tell you about consumption of fish and wildlife caught in contaminated areas.

Is there a medical test to show whether I've been exposed to DDT, DDE, and DDD?

Laboratory tests can detect DDT, DDE, and DDD in fat, blood, urine, semen, and breast milk. These tests may show low, moderate, or excessive exposure to these compounds, but cannot tell the exact amount you were exposed to, or whether you will experience adverse effects. These tests are not routinely available at the doctor's office because they require special equipment.

Has the federal government made recommendations to protect human health?

The Occupational Safety and Health Administration (OSHA) sets a limit of 1 milligram of DDT per cubic meter of air (1 mg/m^3) in the workplace for an 8-hour shift, 40-hour workweek.

The Food and Drug Administration (FDA) has set limits for DDT, DDE, and DDD in foodstuff at or above which the agency will take legal action to remove the products from the market.

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Where can I get more information?

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

For more information, contact:
Agency for Toxic Substances and Disease Registry
Division of Toxicology and Environmental Medicine
1600 Clifton Road NE, Mailstop F-62
Atlanta, GA 30333
Phone: 1-800-CDC-INFO · 888-232-6348 (TTY)
Fax: 1-770-488-4178
Email: cdcinfo@cdc.gov (mailto:cdcinfo@cdc.gov)

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.

Information line and technical assistance:
Phone: 888-422-8737
FAX: (770)-488-4178

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

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- Page last reviewed: March 3, 2011
- Page last updated: March 3, 2011
- Content source: Agency for Toxic Substances and Disease Registry (http://www.atsdr.cdc.gov/)

Agency for Toxic Substances and Disease Registry, 4770 Buford Hwy NE, Atlanta, GA 30341
Contact CDC: 800-232-4636 / TTY: 888-232-6348

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Water: Private Wells

Human Health

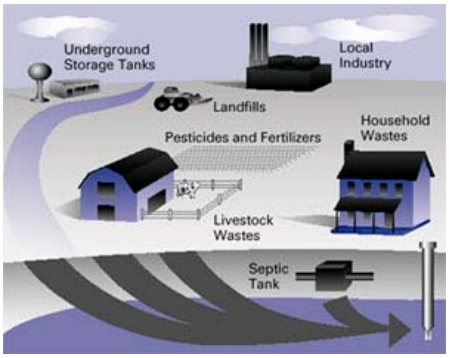
The first step to protect your health and the health of your family is learning about what may pollute your source of drinking water. Potential contamination may occur naturally, or as a result of human activity.

What are Some Naturally Occurring Sources of Pollution?

- **Microorganisms**: Bacteria, viruses, parasites and other microorganisms are sometimes found in water. Shallow wells — those with water close to ground level — are at most risk. Runoff, or water flowing over the land surface, may pick up these pollutants from wildlife and soils. This is often the case after flooding. Some of these organisms can cause a variety of illnesses. Symptoms include nausea and diarrhea. These can occur shortly after drinking contaminated water. The effects could be short-term yet severe (similar to food poisoning) or might recur frequently or develop slowly over a long time.
- **Radionuclides**: Radionuclides are radioactive elements such as uranium and radium. They may be present in underlying rock and ground water
- **Radon**: Radon isa gas that is a natural product of the breakdown of uranium in the soil — can also pose a threat. Radon is most dangerous when inhaled and contributes to lung cancer. Although soil is the primary source, using household water containing Radon contributes to elevated indoor Radon levels. Radon is less dangerous when consumed in water, but remains a risk to health.
- **Nitrates and Nitrites**: Although high nitrate levels are usually due to human activities (see below), they may be found naturally in ground water. They come from the breakdown of nitrogen compounds in the soil. Flowing ground water picks them up from the soil. Drinking large amounts of nitrates and nitrites is particularly threatening to infants (for example, when mixed in formula).
- **Heavy Metals**: Underground rocks and soils may contain arsenic, cadmium, chromium, lead, and selenium. However, these contaminants are not often found in household wells at dangerous levels from natural sources.
- **Fluoride**: Fluoride is helpful in dental health, so many water systems add small amounts to drinking water. However, excessive consumption of naturally occurring fluoride can damage bone tissue. High levels of fluoride occur naturally in some areas. It may discolor teeth, but this is not a health risk.

What Human Activities Can Pollute Ground Water?

- **Bacteria and Nitrates**: These pollutants are found in human and animal wastes. Septic tanks can cause bacterial and nitrate pollution. So can large numbers of farm animals. Both septic systems and animal manures must be carefully managed to prevent pollution. Sanitary landfills and garbage dumps are also sources. Children and some adults are at extra risk when exposed to water-born bacteria. These include the elderly and people whose immune systems are weak due to AIDS or treatments for cancer. Fertilizers can add to nitrate problems. Nitrates cause a health threat in very young infants called “blue baby” syndrome. This condition disrupts oxygen flow in the blood.
- **Concentrated Animal Feeding Operations (CAFOs)**: The number of CAFOs, often called “factory farms,” is growing. On these farms thousands of animals are raised in a small space. The large amounts of animal wastes/manures from these farms can threaten water supplies. Strict and careful manure management is needed to prevent pathogen and nutrient problems. Salts from high levels of manures can also pollute ground water.
- **Heavy Metals**: Activities such as mining and construction can release large amounts of heavy metals into nearby ground water sources. Some older fruit orchards may contain high levels of arsenic, once used as a pesticide. At high levels, these metals pose a health risk.



Septic tanks are designed to have a “leach field” around them — an area where wastewater flows out of the tank. This wastewater can also move into the ground water.

- **Fertilizers and Pesticides**: Farmers use fertilizers and pesticides to promote growth and reduce insect damage. These products are also used on golf courses and suburban lawns and gardens. The chemicals in these products may end up in ground water. Such pollution depends on the types and amounts of chemicals used and how they are applied. Local environmental conditions (soil types, seasonal snow and rainfall) also affect this pollution. Many fertilizers contain forms of nitrogen that can break down into harmful nitrates. This could add to other sources of nitrates mentioned above. Some underground agricultural drainage systems collect fertilizers and pesticides. This polluted water can pose problems to ground water and local streams and rivers. In addition, chemicals used to treat buildings and homes for termites or other pests may also pose a threat. Again, the possibility of problems depends on the amount and kind of chemicals. The types of soil and the amount of water moving through the soil also play a role.
- **Industrial Products and Wastes**: Many harmful chemicals are used widely in local business and industry. These can become drinking water pollutants if not well managed. The most common sources of such problems are:
  - **Local Businesses**: These include nearby factories, industrial plants, and even small businesses such as gas stations and dry cleaners. All handle a variety of hazardous chemicals that need careful management. Spills and improper disposal of these chemicals or of industrial wastes can threaten ground water supplies.
  - **Leaking Underground Tanks & Piping**: Petroleum products, chemicals, and wastes stored in underground storage tanks and pipes may end up in the ground water. Tanks and piping leak if they are constructed or installed improperly. Steel tanks and piping corrode with age. Tanks are often found on farms. The possibility of leaking tanks is great on old, abandoned farm sites. Farm tanks are exempt from the EPA rules for petroleum and chemical tanks.
  - **Landfills and Waste Dumps**: Modern landfills are designed to contain any leaking liquids. But floods can carry them over the barriers. Older dumpsites may have a wide variety of pollutants that can seep into ground water.
- **Household Wastes**: Improper disposal of many common products can pollute ground water. These include cleaning solvents, used motor oil, paints, and paint thinners. Even soaps and detergents can harm drinking water. These are often a problem from faulty septic tanks and septic leaching fields.
- **Lead & Copper**: Household plumbing materials are the most common source of lead and copper in home drinking water. Corrosive water may cause metals in pipes or soldered joints to leach into your tap water. Your water’s acidity or alkalinity (often measured as pH) greatly affects corrosion. Temperature and mineral content also affect how corrosive it is. They are often used in pipes, solder, or plumbing fixtures. Lead can cause serious damage to the brain, kidneys, nervous system, and red blood cells. The age of plumbing materials — in particular, copper pipes soldered with lead — is also important. Even in relatively low amounts these metals can be harmful. EPA rules under the Safe Drinking Water Act limit lead in drinking water to 15 parts per billion. Since 1988 the Act only allows “lead free” pipe, solder, and flux

in drinking water systems. The law covers both new installations and repairs of plumbing.
  - For more information on avoiding lead in drinking water, visit the EPA's Lead in Drinking Water web site.
- **Water Treatment Chemicals**: Improper handling or storage of water-well treatment chemicals (disinfectants, corrosion inhibitors, etc.) close to your well can cause problems.

Last updated on Tuesday, March 06, 2012

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Environ Health Perspect. 2006 August; 114(8): A470–A476. PMCID: PMC1551991
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The Apple Bites Back: Claiming Old Orchards for Residential Development

Ernie Hood

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As the U.S. population continues to grow, increasing demand for housing and related community resources means more land is being converted from agricultural uses to residential applications. According to the revised 1997 National Resources Inventory conducted by the USDA Natural Resources Conservation Service, more than 6 million acres of American farmland were converted to developed uses between 1992 and 1997. That is an annual conversion rate of roughly 1.2 million acres per year—a 51% increase over the average annual rate reported for the preceding decade.

Naturally, many of these areas were routinely treated with pesticides and other chemicals during their agricultural lifetimes. Although this legacy has been problematic in a wide variety of land conversion scenarios, one in particular seems to have attracted the attention and concern of environmental officials and property buyers in several states across the country: the residential development of historic orchard properties. In state after state, these old orchards (which most often produced apples, but also peaches, cherries, pears, and other tree crops) are metamorphosing into highly desirable subdivisions—desirable, that is, until it emerges that the soil beneath the feet of the proud new residents may be contaminated with lead and arsenic. These toxic by-products are left from the days before DDT and before organophosphates, when arsenical pesticides, particularly lead arsenate (LA), were the treatment of choice to prevent the ravages of insect damage.

They Loved LA

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LA was introduced in 1892 in Massachusetts for use against the gypsy moth. Two other arsenical pesticides (copper acetoarsenite, known as "Paris green," and calcium arsenate) also were in use, although LA largely replaced them in the 1930s due to lower cost, greater efficacy, and lower phytotoxicity. Even though arsenic residue was recognized as a problem as early as 1919, LA was the most widely used pesticide in the nation—recommended by the USDA and applied to millions of acres of crops—until the late 1940s, when DDT (considered at the time to be safer and more effective) became available. LA continued to be used in some locations into the 1970s, and was ultimately banned in 1988.

LA was perhaps most commonly applied in apple orchards, due to its excellent control of the codling moth, a major apple pest. Today, apple orchard properties that were in production during the heyday of LA use are the focal point of environmental concerns; given the nature of the pests peculiar to orchard crops, growers tended to apply the chemicals frequently and in high concentrations, often over many years. "In some cases, they dusted the apple trees or peach trees every week, whereas most field crops may have had one or two applications during the growing season," says Kevin Schick, a bureau chief with the Site Remediation and Waste Management Program in the New Jersey Department of Environmental Protection.

LA and the other arsenical pesticides were designed to be persistent, and it is that persistence that is causing environmental contamination problems decades after their use ended. "These chemicals have just tremendously long half-lives in the ground," says North Carolina state toxicologist Ken Rudo. "They bind very tightly to the soil."

Once LA reached the soil through over-spray, spillage, rainfall wash-off, or simply fallen fruit and leaves, the lead arsenate underwent hydrolysis, separating into lead and arsenic bound to organic particles in the soil. The lead, being poorly soluble, was immobilized, typically within the top 12 to 18 inches of topsoil. The fate of the arsenic was similar, but a bit more complicated. "Arsenic, as arsenate, even though somewhat sparingly soluble, *is* soluble, and it will move in water," says Washington State University soil scientist Frank Peryea. "I've seen some sites where almost all of the arsenic is still in the topsoil, in the tillage zone, and I've seen sites where I've measured arsenic movement as deep as a meter or so."

Carl Renshaw, a hydrogeologist at Dartmouth College, published a study in the January/February 2006 issue of the *Journal of Environmental Quality* showing that arsenate in the soil can be remobilized by being disturbed. He compared two fields in the same historic New Hampshire orchard. One field had never been disturbed, whereas the other had been tilled and replanted in the early 1990s. "What we found was that in the field that had been replanted, there was somewhat less arsenic on it than in the undisturbed field," he says.

Given the assumption of virtually identical application rates over the years, the discrepancy apparently arose from a portion of the arsenic in the disturbed field having been mobilized and removed by surface water. Renshaw found arsenic in the sediment of a nearby stream in amounts that very closely matched the arsenic missing from the tilled field.

"The implication from our study," says Renshaw, "is that if you're not really careful about erosion, you're going to end up sending a lot of arsenic down into the stream channel." To date, researchers have seen no evidence of direct health effects in humans, animals, or plants exposed to this stream-bound arsenic. However, more study is needed to fully understand the ramifications—if any—of the mobilization.

How Dangerous?

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The potential danger posed to human health by lead and arsenic contamination in historic orchards is a complex issue, fraught with scientific uncertainties and competing interests. Arsenic is a known human carcinogen. Exposure to lead, especially prenatally and in childhood, can lead to neurological damage. There is no doubt that excessive exposure to either substance can adversely impact health, but in this case any risks are almost exclusively long-term—virtually no instances of acute adverse health effects have been documented in people living on historic orchard properties.

Regulatory agencies such as the EPA and state health and environmental departments determine allowable levels of chemicals in soils and water based upon formulas that take into account criteria such as toxicity, exposure, and naturally occurring background concentrations of the chemicals. For carcinogens such as arsenic, the calculations are based upon the amount of a chemical that is predicted to result in 1 additional cancer case occurring in 1 million people exposed over their lifetimes. But there is some flexibility in the standards based on local conditions and

practical considerations. In New Jersey, for example, where background arsenic concentrations are often high, the criterion for residential soil cleanup is set at 20 ppm—50 times the EPA's level of 0.4 ppm.

In historic orchard properties, cleanup action is often triggered when a so-called "hot spot" is discovered—typically an area where the pesticides had been mixed and loaded or stored, and where repeated spills or disposal of excess materials may have occurred. The contaminant concentrations in those hot spots can be significantly higher than in the tree crop areas. But locating hot spots after many decades can be very difficult.

The ATSDR is often called in to analyze the health risks at contaminated historic orchard properties. "We look at the contaminants, the concentrations, the pathway, how long [residents] are exposed to it—all of the different aspects of an exposure," says Robert Safay, an environmental health scientist with the agency. "For example, when you're looking at lead contamination in the soil, you're primarily concerned about young children playing out in the soil."

In all but the most extreme cases, the health risks of living atop contaminated historic orchard soil are ultimately characterized as very low and manageable. Exposure is the critical element. "The real issue here is direct contact—you want to limit the direct contact," says Lori Bowman, director of the Agrichemical Management Bureau in the Wisconsin Department of Agriculture, Trade, and Consumer Protection. As Safay explains, there must be a completed exposure pathway for there to be even the potential for health effects. Ultimately, the amount of risk depends on the level of contamination and the use of the land.

For the most part, residents are advised to limit their direct exposure to the soil if it's unremediated and to take simple measures such as wearing gardening gloves and wiping their feet before entering the house. Peryea says there is little risk from eating plants grown in this type of soil, but advises that home gardeners rinse off produce before bringing it into the home, then wash it again with a detergent and scrub brush to remove any remaining soil particles, paying particular attention to rough vegetables like broccoli and leafy vegetables like lettuce, which can trap and retain dust. He also advises paring root and tuber crops such as potatoes, carrots, and radishes, and not composting the peelings or other unused plant parts.

The risks involved may be modest and long-term in most cases, but low risk is not the same as no risk, and regulatory agencies across the country are finding themselves in a thorny situation as more and more contaminated historic orchard properties are developed. They are caught between their duty to protect public health and the environment, and the fact that the risks presented by most of these properties pale in comparison to those associated with other, more acute contamination sites, such as lands near smelters or toxic waste dumps. Naturally, budgets are limited, and priorities must be set. Yet the orchard situation cannot be ignored, and several states have been wrestling with how to deal with this issue for several years.

The sheer scope of the phenomenon adds another layer to the challenge of how to most effectively deal with it. "The magnitude of the problem is just staggering," says Peryea. Millions of acres across the nation are involved. In the state of Washington alone, Peryea says, some 188,000 acres are affected. In Wisconsin, 50,000 acres may be affected, and in New Jersey, up to 5% of the state's acreage is estimated to be impacted by the historical use of arsenical pesticides. Both New Jersey and Washington have had multistakeholder task forces examine the problem and issue recommendations and guidelines.

Wisconsin is likely to convene a similar task force later in 2006, according to Bowman. "We want to develop a protective, economical, and practical strategy to address potential residues of lead and arsenic in soils related to historic orchard use," she says. "The charge of the task force would be to evaluate the health and environmental impacts, and [also evaluate] what kind of alternatives and strategies we could put into place to limit exposure and to educate and provide outreach to homeowners and developers as to what types of precautions can be taken at these orchard sites to mitigate any risk."

What Can, Should, or Must Be Done

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Because contamination can be spread over large areas, remediation measures vary widely, depending upon the level of contamination, the current or intended use of the property, and state or local regulations. Each method has its advantages and its drawbacks, and each site has its own unique circumstances that will often dictate how, when, and even if the situation will be dealt with.

Excavation is the quickest and most thorough remediation method. This involves scraping up the contaminated topsoil, hauling it away to an approved landfill, and replacing it with clean dirt. Realistically, says Peryea, removal is the only way to eliminate risk, "but it's very expensive." Such total remediation can cost $1 million per acre or more. And it's a huge undertaking. Peryea does the math for 1 acre: "If you have contamination down to three feet, you're looking at getting rid of three acre-feet of soil—that's twelve million pounds of soil."

Capping, which involves simply putting a 12- to 18-inch layer of clean soil over the contaminated soil, has been used in some locations. However, this requires enormous amounts of clean dirt. Further, capping cannot be considered a permanent solution—plants will grow on the soil caps, their roots will penetrate the contaminated soil, and the vegetation will eventually redistribute the lead and arsenic to the clean soil. Also, it is common for the soil caps to be disturbed by construction activities.

Soil blending is another alternative, and one that is growing in popularity, particularly when contaminant concentrations are only minimally in excess of actionable levels. This involves bringing clean soil to a site and mixing it with the existing topsoil, with the intent of reducing concentrations below levels that require health-protective actions. Although relatively effective, blending can be a hit-or-miss operation. The main reason is that operators can't always achieve 100% blending, and it very much matters where the subsequent samples are taken—even a few inches can make a difference. Sometimes it is necessary to repeat the procedure, which, of course, drives up costs. Also, disturbing the soil in this way could actually mobilize the arsenic, as Renshaw's research showed. Regardless of its shortcomings, however, blending is an option many states have chosen in recent years.

In some instances, a simple solution can be adequate. "What seems to do a good job of reducing exposure in areas where people aren't digging in the soil is just to keep turf on it, or keep it vegetated somehow," says Peryea. At some sites, simply moving the contaminated soil to another location on the site and capping it—for example, by burying it under a roadway—has been acceptable, although this option requires that a deed notice be executed, so that all of the records of the sampling and disposal of the contamination become part of the property's permanent title record.

Thus far, other remediation methods have proven to be ineffective, impractical, or counterproductive on these sites. Researchers such as David Butcher, a professor of analytical chemistry at Western Carolina University in Cullowhee, North Carolina, have explored the possibility of phytoremediation of these properties, in which plants are used to suck the contaminants out of the soil, after which the contaminated biomass is destroyed. But this method, though effective in certain remediation situations, doesn't appear to hold much promise in lead- and arsenic-contaminated orchard soils. Phytoremediation is quite slow, potentially taking decades or longer to effectively remove contaminants.

Butcher also was unable to discover a method of removing the lead from the soil without the addition of other chemicals (such as EDTA) to release the tightly bound element.

One way to release the lead is by adding phosphorus to the soil, but this also mobilizes the arsenic. “That creates an even bigger problem,” Peryea says. “If you get the arsenic moving, and it moves down into the ground-water, cleanup becomes much more difficult than trying to keep it in the topsoil.”

According to Peryea, you can scratch microbial volatization as well. In that method, native soil microorganisms are stimulated to volatilize arsenic. The gaseous arsenic can then be trapped. But for this method to be effective, soils must be kept quite wet. Many of the historic orchard properties are well-drained, sloping sites, where it would be difficult to keep the soil adequately flooded. Plus, of course, as Peryea points out, “if you are evolving arsenic off your soil, and it flows down and contaminates your neighbor’s property, that’s going to create some problems.”

Cleanup and real estate disclosure issues are usually handled at the state and local levels, where approaches vary considerably. As public awareness of the potential contamination of historic orchards increases in the affected areas, state agencies are fielding more and more calls from concerned property owners or prospective buyers. Chuck Warzecha, a risk assessor with the Wisconsin Department of Health and Family Services, fields 10 to 15 such calls a year. He tries to give concerned citizens a balanced message. “My first statement is that it’s not a real scary issue and doesn’t have to be a big problem on their property,” he says. “It’s something that now that they know about it, it’s worth doing something about, but they shouldn’t be concerned that past exposure is going to be a real serious issue for their families.”

If callers haven’t had their soil tested yet, Warzecha recommends that they do so. Then he advises them on how to manage the problem if there is one. If contamination hot spots are identified, cleanup may be required under Wisconsin’s Agricultural Chemical Cleanup Program. In such cases the property owner would pay a 25% deductible, with the rest of the costs covered by the state, according to Bowman.

In Washington, the Model Toxics Control Act requires the reporting, study, and cleanup of sites where hazardous substances are above state-set cleanup levels. In residential developments, the state is working to increase awareness of the potential for contamination on historic orchard lands, particularly among developers. The goal is to get developers to incorporate that consideration at the outset of projects, when there are opportunities to deal with problems more easily than could be done once housing is in place. As in other states, several departments are involved in providing consultation, health assessment, and technical assistance on a case-by-case basis.

Washington has also chosen to be proactive in its cleanup efforts at sites where children are especially likely to be affected. “We have elected to focus on schools, child care facilities, and parks where groups of young children might be present, trying to take steps to reduce exposures for kids,” says Dave Bradley, a toxicologist and risk assessor with the Toxics Cleanup Program in the Washington State Department of Ecology. “We’ve focused on a handful of counties, and have further focused on schools, trying to integrate with existing community processes such as school construction, and then trying to prioritize how we use either our authority or funds out of the state Superfund to actually perform some of the cleanup actions.”

In New Jersey, the recommendations and guidelines put forth in the 1999 report of the Historic Pesticide Contamination Task Force set the agenda. Schick, whose department handles historic orchard contamination cases, says there’s no excuse for ignorance on the part of New Jersey developers at this point, and it should be a standard element of their due diligence.

“It’s common knowledge, the guidance is out there, it already involved the real estate agents, the bankers, the insurers, the farm bureau,” Schick says. “It’s been out there long enough that anyone making any kind of investment in developing farmland should have known about it, and they will be held at fault for not coming to the department or cleaning prior to development.”

Paradise Lost, Paradise Regained?

Go to:

Today, Barber Orchard, a 500-acre subdivision located a few miles west of Waynesville, North Carolina, is “not a place where it looks like there are any problems,” says Butcher. “It’s not a place like where there’s been a lot of mining and it looks like a moonscape. It looks beautiful up there.” It may look beautiful, but that doesn’t change the fact that Barber Orchard has had a troubled history.

Barber Orchard was a commercial apple orchard from 1903 until the mid-1980s, when the operation went bankrupt and the land was parceled off for development. In 1999, a pregnant resident heard rumors of birth defects from neighbors and friends in the area. She contacted Rudo, who, with the county health department, initiated an extensive investigation that included soil and water sampling and a series of public meetings with residents. In late 1999 through mid-2000, the federal EPA conducted a $4 million emergency removal of a foot of topsoil from 28 residents’ yards.

Reflecting the tremendous variation in contamination typical of historic orchard sites, the EPA found only trace amounts of lead and arsenic in some sampling locations, but several others were well in excess of the agency’s cleanup goals of 40 ppm arsenic and 400 ppm lead. Samples came in as high as 400 ppm arsenic and 1,200 ppm lead. The highest levels were detected at spots where trees were still located, or had been cultivated in the past, reflecting the cumulative impact of long years of pesticide applications.

In 2001, the site was placed on the National Priorities List under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), an unusual step for a historic orchard. “CERCLA authority is hobbled when it comes to normal use of pesticides,” says James Bateson, branch head of the Superfund Site Evaluation and Removal Branch of the North Carolina Department of Environment and Natural Resources. “In cases where [a pesticide has] been spilled or dumped in large quantities or misused, that’s when CERCLA can have some authority. At Barber Orchard, the case was made that there was enough spillage associated with the way they handled things up there that it wasn’t normal application of pesticide.”

“The way they handled things” was by distributing the pesticides through a unique underground high-pressure piping system, with aboveground nozzles at the tree sites where sprayers were hooked up. The system left pesticide hot spots at several locations throughout the orchard property. “If there was spillage at a particular location above-ground where that particular distribution pipe was located, or if there was a fracture in the pipe, or a joint in the pipe that got a crack or leak in it, then we may have contamination locally at that one particular site, or along the connections along the way,” explains Haywood County Health Department director Carmine Rocco. According to Bateson, the EPA has in fact found several places where pesticides had leaked into the soil because of poor maintenance of the piping system.

In 2004 the EPA issued a record of decision (a document specifying how the agency planned to clean up the site) for the orchard’s soil, calling for much more removal of contaminated dirt, mainly from vacant lots on the property. “What we’re doing right now is waiting

for funding to implement the cleanup for soil," says Jon Bornholm, the EPA's project manager for the Barber Orchard site. That phase of the cleanup, which should take less than a year, is projected to cost $20 million, and there's no telling when the funds will be released by the EPA for it to take place.

The EPA is expected to render a record of decision for dealing with groundwater contamination on the site before the end of 2006. Bornholm expects that the agency will opt for "monitored natural attenuation"—in other words, let Mother Nature take care of the problem, and hope that contaminant concentrations will decrease over time through natural processes such as biodegradation and dispersion. He guesses that could take 30 to 50 years, with the EPA monitoring the situation continually. Residents have been advised to filter their well water since the problem was uncovered, and city water is now available to the site, although not all of the current homeowners have elected to hook up to the service.

Since the problem arose, the ATSDR has also been involved at Barber Orchard, evaluating the health situation. In April 2002, the agency released its official public health assessment for the site, which concluded that "current exposures to site contaminants are not likely to result in adverse health effects. . . . The exposure pathways for lead and arsenic were disrupted within a relatively short time frame, so past exposures are not likely to lead to health effects at this time."

Meanwhile, Barber Orchard's tax values have increased, and buying and selling of homes in the subdivision has not been hurt by the site's Superfund status. "The heat of the moment has passed, and I think we've gotten over the panic mode," says Ellis Morris, president of the Haywood County Board of Realtors. "Initially, people were tentative about buying in to that particular neighborhood, but that's been resolved, there's a comfort level now, and the real estate there is keeping pace with all of the other areas of Haywood County in terms of days on the market and selling price."

David Miller would agree with that assessment. He and his wife retired to Barber Orchard from Florida in 1997, and his 1.4-acre lot was one of the properties cleaned up by the EPA. He is unconcerned about the contamination at the site and thinks the whole situation has been overblown. "I haven't changed the way I live," he says. "I work in the garden just about every day, I've planted a vegetable garden and eaten the vegetables, I've planted some fruit and eaten the fruit. So it has not affected me or my wife in any way."

So it appears that Barber Orchard was paradise lost for a time, but is now paradise regained. Now, however, some neighbors just down the road may be facing a similar situation. In May 2006 residents of the Tan Woods and Orchard Estates subdivisions, built on what was once Francis Orchard, were notified that soil samples from a vacant lot at the site had tested positive for lead, arsenic, and other pesticides—a mix similar to that found at Barber Orchard. And like Barber Orchard, Francis Orchard was equipped with an underground pesticide piping system.

It's still early in the process, and the results of more thorough sampling and testing are not yet available, so it's too soon to predict whether Francis Orchard may eventually become a Superfund site. But this time around, according to Bateson, both residents and involved officials can benefit from the Barber Orchard experience. At Francis Orchard, he says, "the residents are well schooled after seeing what's gone on at Barber Orchard, and of course the county and state people have been around the block now too."

Questions Remain

Go to:

Despite the large scale scope of the problem, it appears that living on a historic orchard property contaminated by lead and arsenic does not constitute an immediate threat to human health. So it is still an open question whether it's really necessary to spend huge amounts of money, often from tax dollars, to ameliorate these sites.

Peryea thinks that what is needed is a solid epidemiologic study to document whether there really is a problem with people living on these arsenical pesticide–contaminated soils. "If that sort of study was done," he says, "and it was to show that there's no problem, or that the problem is controllable by setting up some sort of engineering controls or behavioral controls, like they do with urban lead nowadays, that would probably take care of a lot of the problem. The response—rather than trying to force a cleanup that would probably be wildly impractical, very expensive, and potentially ruin property values—would be that people would change their behavior a bit and end up minimizing the risk."

Online Resources

Go to:

New Jersey, Washington, and Wisconsin offer detailed advice to residents, developers, and other interested parties about what to do if they suspect or know their land is contaminated. Wisconsin has posted a variety of publications (**http://www.datcp.state.wi.us/arm/agriculture/pestfert/pesticides/accp/lead_arsen_resources.jsp**), including tips for safe gardening in lead- and arsenic-contaminated soil. Washington provides a comprehensive toolbox of resources stemming from its Area-Wide Soil Contamination Project, a task force that addressed not only historical orchard contamination, but also lead and arsenic contamination over widespread areas of the state from smelters and leaded gasoline combustion; see **http://www.ecy.wa.gov/programs/tcp/area_wide/area_wide_hp.html**. New Jersey offers the report of the Historic Pesticide Contamination Task Force (**http://www.state.nj.us/dep/special/hpctf/index.html**) and i-MapNJ, an environmental mapping tool that lets residents obtain detailed contamination information for specific locations (**http://www.state.nj.us/dep/gis/depsplash.htm**).

You spray, you pay?

A blooming problem?

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http://www.epa.gov/ttn/atw/hlthef/arsenic.html
Last updated on Tuesday, November 06, 2007

Technology Transfer Network
Air Toxics Web Site

Arsenic Compounds

ARSENIC COMPOUNDS[(A)]

107-02-8

Hazard Summary-Created in April 1992; Revised in January 2000

Arsenic, a naturally occurring element, is found throughout the environment; for most people, food is the major source of exposure. Acute (short-term) high-level inhalation exposure to arsenic dust or fumes has resulted in gastrointestinal effects (nausea, diarrhea, abdominal pain); central and peripheral nervous system disorders have occurred in workers acutely exposed to inorganic arsenic. Chronic (long-term) inhalation exposure to inorganic arsenic in humans is associated with irritation of the skin and mucous membranes. Chronic oral exposure has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, and liver or kidney damage in humans. Inorganic arsenic exposure in humans, by the inhalation route, has been shown to be strongly associated with lung cancer, while ingestion of inorganic arsenic in humans has been linked to a form of skin cancer and also to bladder, liver, and lung cancer. EPA has classified inorganic arsenic as a Group A, human carcinogen.

Arsine is a gas consisting of arsenic and hydrogen. It is extremely toxic to humans, with headaches, vomiting, and abdominal pains occurring within a few hours of exposure. EPA has not classified arsine for carcinogenicity.

Please Note: The main sources of information for this fact sheet are EPA's Integrated Risk Information System (IRIS), which contains information on inhalation chronic toxicity and the RfC for arsine, oral chronic toxicity and the RfD for inorganic arsenic, and the carcinogenic effects of inorganic arsenic including the unit cancer risk for inhalation exposure, and the Agency for Toxic Substances and Disease Registry's (ATSDR's) *Toxicological Profile for Arsenic*.

Uses

- The major use for inorganic arsenic is in wood preservation; arsine is used in the microelectronics industry and in semiconductor manufacture. (2)
- Until the 1940s, inorganic arsenic solutions were widely used in the treatment of various diseases, such as syphillis and psoriasis. Inorganic arsenic is still used as an antiparasitic agent in veterinary medicine and in homeopathic and folk remedies in the United States and other countries. (2)

Sources and Potential Exposure

- Inorganic arsenic is found throughout the environment; it is released into the air by volcanoes, the weathering of arsenic-containing minerals and ores, and by commercial or industrial processes. (1,2)
- For most people, food is the largest source of arsenic exposure (about 25 to 50 micrograms per day [µg/d]), with lower amounts coming from drinking water and air. Among foods, some of the highest levels are found in fish and shelfish; however, this arsenic exists primarily as organic compounds, which are essentially nontoxic. (1)

- Elevated levels of inorganic arsenic may be present in soil, either from natural mineral deposits or contamination from human activities, which may lead to dermal or ingestion exposure. (1)
- Workers in metal smelters and nearby residents may be exposed to above-average inorganic arsenic levels from arsenic released into the air. (1)
- Other sources of inorganic arsenic exposure include burning plywood treated with an arsenic wood preservative or dermal contact with wood treated with arsenic. (2)
- Most arsenic poisoning incidents in industry have involved the production of arsine, a short-lived, extremely toxic gas. (3)

Assessing Personal Exposure

- Measurement of inorganic arsenic in the urine is the best way to determine recent exposure (within the last 1 to 2 days), while measuring inorganic arsenic in hair or fingernails may be used to detect high-level exposures that occurred over the past 6-12 months. (1)

Health Hazard Information

Acute Effects:

Inorganic Arsenic

- Acute inhalation exposure of workers to high levels of arsenic dusts or fumes has resulted in gastrointestinal effects (nausea, diarrhea, abdominal pain), while acute exposure of workers to inorganic arsenic has also resulted in central and peripheral nervous system disorders. (1)
- Acute oral exposure to inorganic arsenic, at doses of approximately 600 micrograms per kilogram body weight per day (µg/kg/d) or higher in humans, has resulted in death. Oral exposure to lower levels of inorganic arsenic has resulted in effects on the gastrointestinal tract (nausea, vomiting), central nervous system (CNS) (headaches, weakness, delirium), cardiovascular system (hypotension, shock), liver, kidney, and blood (anemia, leukopenia). (1,2)
- Acute animal tests in rats and mice have shown inorganic arsenic to have moderate to high acute toxicity. (5)

Arsine

- Acute inhalation exposure to arsine by humans has resulted in death; it has been reported that a half-hour exposure to 25 to 50 parts per million (ppm) can be lethal. (4)
- The major effects from acute arsine exposure in humans include headaches, vomiting, abdominal pains, hemolytic anemia, hemoglobinuria, and jaundice; these effects can lead to kidney failure. (4,8)
- Arsine has been shown to have extreme acute toxicity from acute animal tests. (5)

Chronic Effects (Noncancer):

Inorganic arsenic

- Chronic inhalation exposure to inorganic arsenic in humans is associated with irritation of the skin and mucous membranes (dermatitis, conjunctivitis, pharyngitis, and rhinitis). (1,2)
- Chronic oral exposure to inorganic arsenic in humans has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, gangrene of the extremities, vascular lesions, and liver or kidney damage. (1,2)
- No chronic inhalation exposure studies have been performed in animals for any inorganic arsenic compound. (1)
- Some studies have suggested that inorganic arsenic is an essential dietary nutrient in goats, chicks, and rats. However, no comparable data are available for humans. EPA has concluded that essentiality, although not rigorously established, is plausible. (1,6)
- EPA has not established a Reference Concentration (RfC) for inorganic arsenic. (6)

- The California Environmental Protection Agency (CalEPA) has established a chronic inhalation reference level of 0.00003 milligrams per cubic meter (mg/m^3) based on developmental effects in mice. The CalEPA reference exposure level is a concentration at or below which adverse health effects are not likely to occur. It is not a direct estimator of risk, but rather a reference point to gauge the potential effects. At lifetime exposures increasingly greater than the reference exposure level, the potential for adverse health effects increases. (7)
- The Reference Dose (RfD) for inorganic arsenic is 0.0003 milligrams per kilogram body weight per day (mg/kg/d) based on hyperpigmentation, keratosis, and possible vascular complications in humans. The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious noncancer effects during a lifetime. (6)
- EPA has medium confidence in the study on which the RfD for inorganic arsenic was based because, although an extremely large number of people were included in the assessment (>40,000), the doses were not well characterized and other contaminants were present. The supporting human toxicity database, while extensive, is somewhat flawed and, consequently, EPA has assigned medium confidence to the RfD. (6)

Arsine

- No information is available on the chronic effects of arsine in humans.
- The RfC for arsine is 0.00005 mg/m^3 based on increased hemolysis, abnormal red blood cell morphology, and increased spleen weight in rats, mice, and hamsters. (4)
- EPA has medium confidence in the RfC based on: (1) high confidence in the studies on which the RfC for arsine was based because the sample sizes were adequate, statistical significance was reported, concentration dose-response relationships were documented, three species were investigated, and both a no-observed-adverse-effect level (NOAEL) and a lowest-observed-adverse-effect level (LOAEL) were identified, and (2) medium confidence in the database because while there were three inhalation animal studies and a developmental/reproductive study, there were no data available on human exposure. (4)

Reproductive/Developmental Effects:

Inorganic arsenic

- Several studies have suggested that women who work in, or live near, metal smelters may have higher than normal spontaneous abortion rates, and their children may exhibit lower than normal birthweights. However, these studies are limited because they were designed to evaluate the effects of smelter pollutants in general, and are not specific for inorganic arsenic. (1)
- Ingested inorganic arsenic can cross the placenta in humans, exposing the fetus to the chemical. (2)
- Oral animal studies have reported inorganic arsenic at very high doses to be fetotoxic and to cause birth defects. (1)

Arsine

- Human studies have indicated higher than expected spontaneous abortion rates in women in the microelectronics industry who were exposed to arsine. However, these studies have several limitations, including small sample size and exposure to other chemicals in addition to arsine. (4)

Cancer Risk:

Inorganic arsenic

- Human, inhalation studies have reported inorganic arsenic exposure to be strongly associated with lung cancer. (1,2,6)
- Ingestion of inorganic arsenic in humans has been associated with an increased risk of nonmelanoma skin cancer and also to an increased risk of bladder, liver, and lung cancer. (1,6)

- Animal studies have not associated inorganic arsenic exposure via the oral route with cancer, and no cancer inhalation studies have been performed in animals for inorganic arsenic. (1)
- EPA has classified inorganic arsenic as a Group A, human carcinogen. (6)
- EPA used a mathematical model, using data from an occupational study of arsenic-exposed copper smelter workers, to estimate the probability of a person developing cancer from continuously breathing air containing a specified concentration of inorganic arsenic. EPA calculated an inhalation unit risk estimate of $4.3 \times 10^{-3}(\mu g/m^3)^{-1}$. EPA estimates that, if an individual were to continuously breathe air containing inorganic arsenic at an average of 0.0002 $\mu g/m^3$ (2×10^{-7} mg/m^3) over his or her entire lifetime, that person would theoretically have no more than a one-in-a-million increased chance of developing cancer as a direct result of breathing air containing this chemical. Similarly, EPA estimates that continuously breathing air containing 0.002 $\mu g/m^3$ (2×10^{-6} mg/m^3) would result in not greater than a one-in-a-hundred thousand increased chance of developing cancer, and air containing 0.02 $\mu g/m^3$ (2×10^{-5} mg/m^3) would result in not greater than a one-in-ten thousand increased chance of developing cancer. For a detailed discussion of confidence in the potency estimates, please see IRIS. (6)
- EPA has calculated an oral cancer slope factor of 1.5 $(mg/kg/d)^{-1}$ for inorganic arsenic. (6)

Arsine

- No cancer inhalation studies in humans or animals are available for arsine. (1)
- EPA has not classified arsine for carcinogenicity. (4)

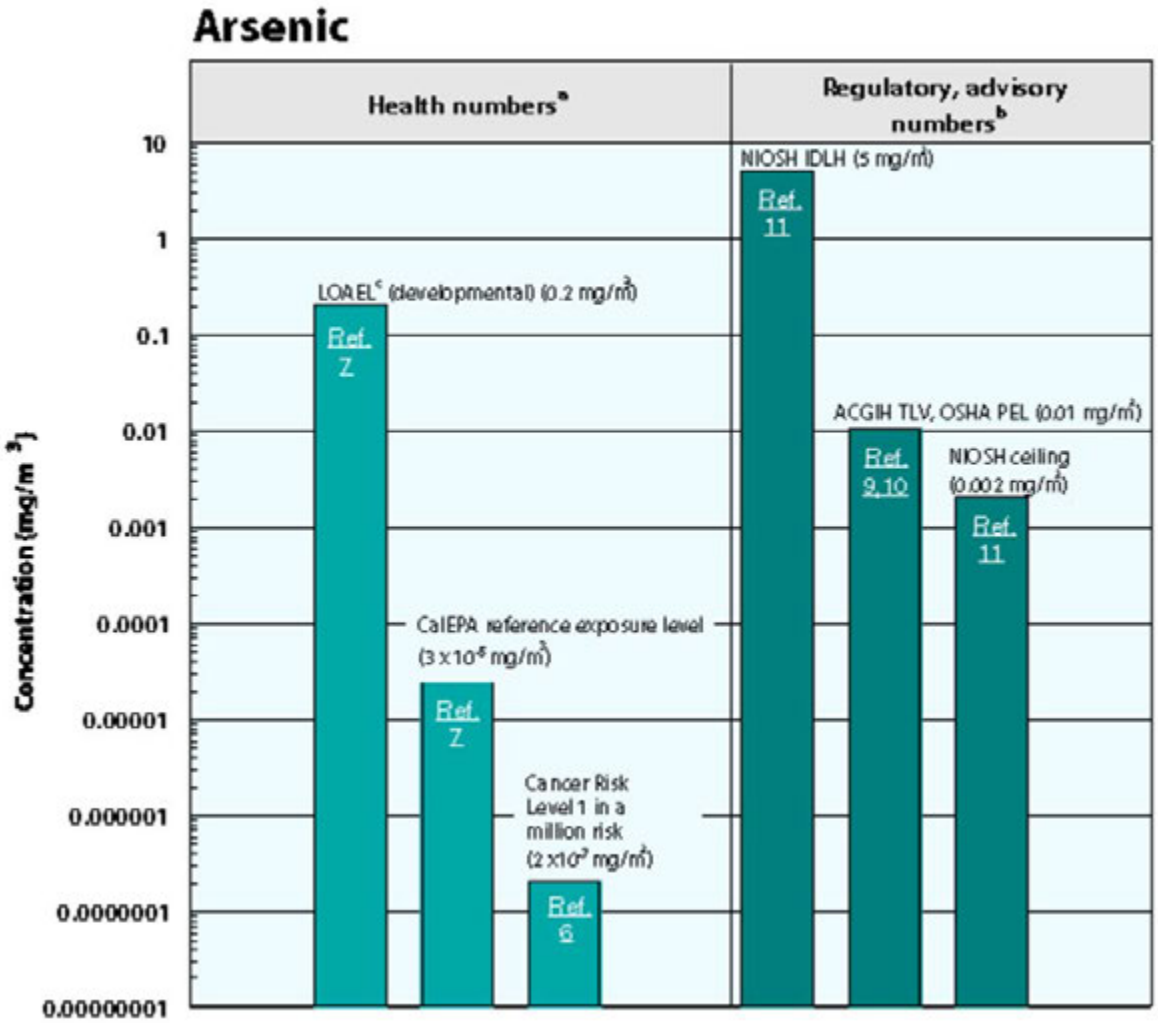
Physical Properties

- Inorganic arsenic is a naturally occurring element in the earth's crust.(1)
- Pure inorganic arsenic is a gray-colored metal, but inorganic arsenic is usually found combined with other elements such as oxygen, chlorine, and sulfur. (1)
- The chemical symbol for inorganic arsenic is As, and it has an atomic weight of 74.92 g/mol. (3)
- The chemical formula for arsine is AsH_3, and it has a molecular weight of 77.95 g/mol. (8)
- Arsine is a colorless gas with a disagreeable garlic odor. (8)
- Arsenic combined with elements such as oxygen, chlorine, and sulfur forms inorganic arsenic; inorganic arsenic compounds include arsenic pentoxide, arsenic trioxide, and arsenic acid. Arsenic combined with carbon and hydrogen forms organic arsenic; organic arsenic compounds include arsanilic acid, arsenobetaine, and dimethylarsinic acid. (1)

Conversion Factors (only for the gaseous form):

To convert concentrations in air (at 25°C) from ppm to mg/m^3: mg/m^3 = (ppm) × (molecular weight of the compound)/(24.45). For inorganic arsenic: 1 ppm = 3.06 mg/m^3. For arsine: 1 ppm = 3.19 mg/m^3. To convert concentrations in air from $\mu g/m^3$ to mg/m^3: mg/m^3 = ($\mu g/m^3$) × (1 mg/1,000 µg).

Health Data from Inhalation Exposure (Inorganic Arsenic)



ACGIH TLV--American Conference of Governmental and Industrial Hygienists' threshold limit value expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effects.
NIOSH IDLH--National Institute of Occupational Safety and Health's immediately dangerous to life or health concentration; NIOSH recommended exposure limit to ensure that a worker can escape from an exposure condition that is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from the environment.
NIOSH REL ceiling value--NIOSH's recommended exposure limit ceiling; the concentration that should not be exceeded at any time.
OSHA PEL--Occupational Safety and Health Administration's permissible exposure limit expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effect averaged over a normal 8-h workday or a 40-h workweek.

The health and regulatory values cited in this factsheet were obtained in December 1999.
[a] Health numbers are toxicological numbers from animal testing or risk assessment values developed by EPA.
[b]Regulatory numbers are values that have been incorporated in Government regulations, while advisory numbers are nonregulatory values provided by the Government or other groups as advice. OSHA numbers are regulatory, whereas NIOSH and ACGIH numbers are advisory.
[c]The LOAEL is from the critical study used as the basis for the CalEPA chronic reference exposure level.

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A. * This fact sheet addresses the toxicity of the inorganic arsenic compounds as well as the toxicity of the gaseous arsenic trihydride: arsine.

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Lead

Learn about Lead

- What is lead?
- Where is lead found?
- How can people be exposed to lead?
- Possible adverse health effects of exposures to lead
- Lead exposure data

What is Lead?

Lead is a highly toxic metal and it is all around us. Lead was used for many years in paints and other products found in and around our homes. Lead-based paint and lead contaminated dust are the main sources of exposure for lead in U.S. children. Lead-based paints were banned for use in housing in 1978. There is a good chance that any home, building, school or day care center built before 1978 contains some lead paint.

One million children are affected by lead poisoning, but when you know what to look for and what to do, lead poisoning is entirely preventable.

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Where is Lead Found?

The most common source of lead is from paint in homes and buildings built before 1978. Lead also can be emitted into the air from industrial sources and leaded aviation gasoline, and lead can enter drinking water through plumbing materials.

It is also used in the production of batteries, ammunition, metal products (solder and pipes), and devices to shield X-rays. Because of health concerns, lead from paints and ceramic products, caulking, and pipe solder has been dramatically reduced in recent years. The use of lead as an additive to automobile gasoline was banned in 1996 in the United States.

Lead is also a naturally occurring element. Natural levels of lead in soil range between 50 parts per million (ppm) and 400 ppm. Mining, smelting, and refining activities have resulted in substantial increases in lead levels in the environment, especially near mining and smelting sites. For example, near some types of industrial and municipal facilities, and adjacent to highways (Chaney et al., 1984; Schacklette et al., 1984) soil lead concentrations have been reported to be more than 11,000 ppm (National Research Council, 1980).

Read more about where lead can be found:

- At home
- At schools and childcare facilities
- In products
- In drinking water
- In outdoor air
- In soil

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How Can People Be Exposed to Lead?

Children

Lead is dangerous to children because babies and young children often put their hands and other objects that can have lead dust on them in their mouths. Also, children's growing bodies absorb more lead than adult bodies do, and their brains and nervous systems are more sensitive to the damaging effects of lead.

Children living at or below the poverty line who live in older housing are at greatest risk. Children of some racial and ethnic groups, and those living in older housing, are disproportionately affected by lead.

Learn more about sources of lead exposure.

Pregnant Women

Pregnant women can be exposed to lead by spending time in areas where lead-based paints are deteriorating into lead dust that they then breathe in. Likewise, eating and drinking from dishes or glasses that contain lead water, or using certain folk remedies to which lead is intentionally added can cause exposures to lead. In addition, working in a job or engaging in hobbies where lead is used, such as making stained glass, can increase exposure.

Adults

Adults are also susceptible to lead exposure. This may be from:

- Breathing in lead dust, especially during renovation or repair work that disturbs painted surfaces in older homes and buildings.
- Putting their hands or other objects covered with lead dust in their mouths.
- Eating or drinking contaminated food or water or using certain folk remedies.

- Working in a job or engaging in hobbies where lead is used.

Learn more about sources of lead exposure.

Lower Your Chances of Exposure to Lead

Simple steps like keeping your home clean and feeding your family a well-balanced diet will go a long way in preventing lead poisoning. You can lower the chances of exposure to lead in your home, both now and in the future, by taking these steps:

- Use only cold water to prepare food and drinks.
- Flush all water outlets used for drinking or food preparation.
- Clean debris out of all outlet screens or aerators on faucets on a regular basis.
- Keep your home clean and dust-free.
- Wipe up any paint chips or visible dust with a wet sponge or rag. Clean dust around areas where there is friction and dust can be generated, such as doors, windows, and drawers.
- Wash children's hands, bottles, pacifiers and toys often.
- Teach children to wipe and remove their shoes and wash hands after playing outdoors.
- Ensure that your family members eat well-balanced meals. Lead interferes with some of the body's basic functions. Our bodies can't tell the difference between lead and calcium, which is a mineral that strengthens bones. Children with healthy diets absorb less lead.
- Make sure your contractor is Lead Safe Certified.

Determine if your family is at risk for lead poisoning with the Lead Poisoning Home Checklist (PDF) (1 pg, 47K, About PDF).

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Possible Adverse Health Effects of Exposures to Lead

Lead exposure affects the nervous system and can cause a range of health effects, from behavioral problems and learning disabilities, to seizures and death. Children six years old and younger are most at risk.

Children

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
- Anemia
- In rare cases of acute lead poisoning from ingestion of lead, seizures, coma and even death.

Pregnant Women

Lead can accumulate in our bodies over time, where it is stores in bones along with calcium. During pregnancy, lead is released from bones as maternal calcium is used to help form the bones of the fetus. This is particularly true if a woman does not have enough dietary calcium. Lead can also be easily circulated from the mother's blood stream through the placenta to the fetus. Mothers with high levels of lead in their bodies can expose their developing fetuses, resulting in serious and developmental problems including:

- Miscarriages,
- Premature births or low birth weight,
- Brain damage, decreased mental abilities and learning difficulties, and/or
- Reduced growth in young children.

Find out more about lead's effects on pregnancy:

- March of Dimes Healthy Pregnancy EXIT Disclaimer
- Effects of Workplace Hazards on Female Reproductive Health, National Institute for Occupational Safety and Health.

Adults

Lead is also harmful to adults. Adults can suffer from:

- Hearing and vision impairment,
- Reproductive problems (in both men and women),
- High blood pressure and hypertension,
- Nerve disorders,
- Memory and concentration problems,
- Poor muscle coordination, and
- Muscle and joint pain.

Read more on the health effects of lead at the Agency for Toxic Substances and Disease Registry (ATSDR).

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Lead Exposure Data

The Centers for Disease Control's National Center for Health Statistics monitors blood lead levels in the United States.

National Center for Health Statistics

Get information on the number of children with elevated blood lead levels, and number and percentage of children tested for lead in your area.

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Last updated on Thursday, May 17, 2012

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Key: I = IRIS; P = PPRTV; A = ATSDR; C = Cal EPA; X = PPRTV Appendix; H = HEAST; J = New Jersey; Y = New York; O = EPA Office of Water; E = Environmental Criteria and Assessment Office; S = see user guide Section 5; L = see user guide on lead; M = mutagen; V = volatile; F = See FAQ; c = cancer; * = where: n SL < 100X c SL; ** = where n SL < 10X c SL; n = noncancer; m = Concentration may exceed ceiling limit (See User Guide); s = Concentration may exceed Csat (See User Guide); SSL values are based on DAF=1

Toxicity and Chemical-specific Information												Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	volc	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
1.8E-02	C	5.1E-06	C	1.5E-01	I					1	0.1		ALAR	1596-84-5	2.7E+01	c	9.6E+01	c	4.8E-01	c	2.4E+00	c	3.7E+00	c		8.2E-04	
8.7E-03	I			4.0E-03	I					1	0.1		Acephate	30560-19-1	5.6E+01	c**	2.0E+02	c*					7.7E+00	c**		1.7E-03	
		2.2E-06	I			9.0E-03	I	V		1		1.1E+05	Acetaldehyde	75-07-0	1.0E+01	c**	5.2E+01	c**	1.1E+00	c**	5.6E+00	c**	2.2E+00	c**		4.5E-04	
				2.0E-02	I					1	0.1		Acetochlor	34256-82-1	1.2E+03	n	1.2E+04	n					2.7E+02	n		2.2E-01	
				9.0E-01	I	3.1E+01	A	V		1		1.1E+05	Acetone	67-64-1	6.1E+04	n	6.3E+05	nms	3.2E+04	n	1.4E+05	n	1.2E+04	n		2.4E+00	
				3.0E-03	P	6.0E-02	P	V		1		1.1E+05	Acetone Cyanohydrin	75-86-5	2.0E+02	n	2.1E+03	n	6.3E+01	n	2.6E+02	n	3.4E+01	n		6.9E-03	
						6.0E-02	I	V		1		1.3E+05	Acetonitrile	75-05-8	8.7E+02	n	3.7E+03	n	6.3E+01	n	2.6E+02	n	1.3E+02	n		2.6E-02	
				1.0E-01	I			V		1		2.5E+03	Acetophenone	98-86-2	7.8E+03	ns	1.0E+05	nms					1.5E+03	n		4.5E-01	
3.8E+00	C	1.3E-03	C							1	0.1		Acetylaminofluorene, 2-	53-96-3	1.3E-01	c	4.5E-01	c	1.9E-03	c	9.4E-03	c	1.4E-02	c		6.5E-05	
				5.0E-04	I	2.0E-05	I	V		1		2.3E+04	Acrolein	107-02-8	1.5E-01	n	6.5E-01	n	2.1E-02	n	8.8E-02	n	4.1E-02	n		8.4E-06	
5.0E-01	I	1.0E-04	I	2.0E-03	I	6.0E-03	I		M	1	0.1		Acrylamide	79-06-1	2.3E-01	c	3.4E+00	c	9.6E-03	c	1.2E-01	c	4.3E-02	c		9.1E-06	
				5.0E-01	I	1.0E-03	I			1	0.1		Acrylic Acid	79-10-7	3.0E+04	n	2.9E+05	nm	1.0E+00	n	4.4E+00	n	7.7E+03	n		1.6E+00	
5.4E-01	I	6.8E-05	I	4.0E-02	A	2.0E-03	I	V		1		1.1E+04	Acrylonitrile	107-13-1	2.4E-01	c*	1.2E+00	c*	3.6E-02	c*	1.8E-01	c*	4.5E-02	c*		9.8E-06	
						6.0E-03	P			1	0.1		Adiponitrile	111-69-3	8.5E+06	nm	3.6E+07	nm	6.3E+00	n	2.6E+01	n					
5.6E-02	C			1.0E-02	I					1	0.1		Alachlor	15972-60-8	8.7E+00	c*	3.1E+01	c					9.1E-01	c	2.0E+00	7.5E-04	1.6E-03
				1.0E-03	I					1	0.1		Aldicarb	116-06-3	6.1E+01	n	6.2E+02	n					1.5E+01	n		3.8E-03	
				1.0E-03	I					1	0.1		Aldicarb Sulfone	1646-88-4	6.1E+01	n	6.2E+02	n					1.6E+01	n		3.4E-03	
1.7E+01	I	4.9E-03	I	3.0E-05	I					1	0.1		Aldrin	309-00-2	2.9E-02	c*	1.0E-01	c	5.0E-04	c	2.5E-03	c	2.1E-04	c		3.4E-05	
				2.5E-01	I					1	0.1		Ally	74223-64-6	1.5E+04	n	1.5E+05	nm					3.8E+03	n		1.5E+00	
				5.0E-03	I	1.0E-04	X			1	0.1		Allyl Alcohol	107-18-6	3.0E+02	n	3.1E+03	n	1.0E-01	n	4.4E-01	n	7.8E+01	n		1.6E-02	
2.1E-02	C	6.0E-06	C			1.0E-03	I	V		1		1.4E+03	Allyl Chloride	107-05-1	6.8E-01	c**	3.4E+00	c**	4.1E-01	c**	2.0E+00	c**	6.3E-01	c**		2.0E-04	
				1.0E+00	P	5.0E-03	P			1			Aluminum	7429-90-5	7.7E+04	n	9.9E+05	nm	5.2E+00	n	2.2E+01	n	1.6E+04	n		2.3E+04	
				4.0E-04	I					1			Aluminum Phosphide	20859-73-8	3.1E+01	n	4.1E+02	n					6.2E+00	n			
				3.0E-04	I					1	0.1		Amdro	67485-29-4	1.8E+01	n	1.8E+02	n					4.7E+00	n		1.7E+03	
				9.0E-03	I					1	0.1		Ametryn	834-12-8	5.5E+02	n	5.5E+03	n					1.2E+02	n		1.2E-01	
2.1E+01	C	6.0E-03	C							1	0.1		Aminobiphenyl, 4-	92-67-1	2.3E-02	c	8.2E-02	c	4.1E-04	c	2.0E-03	c	2.6E-03	c		1.3E-05	
				8.0E-02	P					1	0.1		Aminophenol, m-	591-27-5	4.9E+03	n	4.9E+04	n					1.2E+03	n		4.7E-01	
				2.0E-02	P					1	0.1		Aminophenol, p-	123-30-8	1.2E+03	n	1.2E+04	n					3.1E+02	n		1.2E-01	
				2.5E-03	I					1	0.1		Amitraz	33089-61-1	1.5E+02	n	1.5E+03	n					5.9E+00	n		3.0E+00	
						1.0E-01	I			1			Ammonia	7664-41-7					1.0E+02	n	4.4E+02	n					
				2.0E-01	I					1			Ammonium Sulfamate	7773-06-0	1.6E+04	n	2.0E+05	nm					3.1E+03	n			
5.7E-03	I	1.6E-06	C	7.0E-03	P	1.0E-03	I			1	0.1		Aniline	62-53-3	8.5E+01	c**	3.0E+02	c*	1.0E+00	n	4.4E+00	n	1.2E+01	c**		3.9E-03	
4.0E-02	P			2.0E-03	X					1	0.1		Anthraquinone, 9,10-	84-65-1	1.2E+01	c*	4.3E+01	c*					1.2E+00	c*		1.2E-02	
				4.0E-04	I					0.15			Antimony (metallic)	7440-36-0	3.1E+01	n	4.1E+02	n					6.0E+00	n	6.0E+00	2.7E-01	2.7E-01
				5.0E-04	H					0.15			Antimony Pentoxide	1314-60-9	3.9E+01	n	5.1E+02	n					7.5E+00	n			
				9.0E-04	H					0.15			Antimony Potassium Tartrate	11071-15-1	7.0E+01	n	9.2E+02	n					1.3E+01	n			
				4.0E-04	H					0.15			Antimony Tetroxide	1332-81-6	3.1E+01	n	4.1E+02	n					6.0E+00	n			
						2.0E-04	I			0.15			Antimony Trioxide	1309-64-4	2.8E+05	nm	1.2E+06	nm	2.1E-01	n	8.8E-01	n					
				1.3E-02	I					1	0.1		Apollo	74115-24-5	7.9E+02	n	8.0E+03	n					1.8E+02	n		1.1E+01	
2.5E-02	I	7.1E-06	I	5.0E-02	H					1	0.1		Aramite	140-57-8	1.9E+01	c	6.9E+01	c	3.4E-01	c	1.7E+00	c	2.7E+00	c		3.0E-02	
1.5E+00	I	4.3E-03	I	3.0E-04	I	1.5E-05	C			1	0.03		Arsenic, Inorganic	7440-38-2	3.9E-01	c*	1.6E+00	c	5.7E-04	c*	2.9E-03	c*	4.5E-02	c	1.0E+01	1.3E-03	2.9E-01
				3.5E-06	C	5.0E-05	I			1			Arsine	7784-42-1	2.7E-01	n	3.6E+00	n	5.2E-02	n	2.2E-01	n	5.4E-02	n			
				9.0E-03	I					1	0.1		Assure	76578-14-8	5.5E+02	n	5.5E+03	n					9.3E+01	n		1.4E+00	
				5.0E-02	I					1	0.1		Asulam	3337-71-1	3.1E+03	n	3.1E+04	n					7.8E+02	n		2.0E-01	
2.3E-01	C			3.5E-02	I					1	0.1		Atrazine	1912-24-9	2.1E+00	c	7.5E+00	c					2.6E-01	c	3.0E+00	1.7E-04	1.9E-03
8.8E-01	C	2.5E-04	C							1	0.1		Auramine	492-80-8	5.5E-01	c	2.0E+00	c	9.7E-03	c	4.9E-02	c	6.7E-02	c		6.1E-04	
				4.0E-04	I					1	0.1		Avermectin B1	65195-55-3	2.4E+01	n	2.5E+02	n					6.3E+00	n		1.1E+01	
1.1E-01	I	3.1E-05	I					V		1			Azobenzene	103-33-3	5.1E+00	c	2.3E+01	c	7.8E-02	c	4.0E-01	c	1.0E-01	c		8.0E-04	
				2.0E-01	I	5.0E-04	H			0.07			Barium	7440-39-3	1.5E+04	n	1.9E+05	nm	5.2E-01	n	2.2E+00	n	2.9E+03	n	2.0E+03	1.2E+02	8.2E+01
				4.0E-03	I					1	0.1		Baygon	114-26-1	2.4E+02	n	2.5E+03	n					6.1E+01	n		2.0E-02	
				3.0E-02	I					1	0.1		Bayleton	43121-43-3	1.8E+03	n	1.8E+04	n					4.3E+02	n		3.4E-01	
				2.5E-02	I					1	0.1		Baythroid	68359-37-5	1.5E+03	n	1.5E+04	n					8.7E+01	n		2.3E+01	
				3.0E-01	I					1	0.1		Benefin	1861-40-1	1.8E+04	n	1.8E+05	nm					1.2E+03	n		4.1E+01	
				5.0E-02	I					1	0.1		Benomyl	17804-35-2	3.1E+03	n	3.1E+04	n					7.5E+02	n		6.6E-01	
				3.0E-02	I					1	0.1		Bentazon	25057-89-0	1.8E+03	n	1.8E+04	n					4.4E+02	n		9.6E-02	
				1.0E-01	I			V		1		1.2E+03	Benzaldehyde	100-52-7	7.8E+03	ns	1.0E+05	nms					1.5E+03	n		3.3E-01	
5.5E-02	I	7.8E-06	I	4.0E-03	I	3.0E-02	I	V		1		1.8E+03	Benzene	71-43-2	1.1E+00	c*	5.4E+00	c*	3.1E-01	c	1.6E+00	c*	3.9E-01	c*	5.0E+00	2.0E-04	2.6E-03
				2.0E-04	X					1	0.1		Benzenediamine-2-methyl sulfate, 1,4-	6369-59-1	1.2E+01	n	1.2E+02	n					3.1E+00	n		8.7E-04	
				1.0E-03	P			V		1		1.3E+03	Benzenethiol	108-98-5	7.8E+01	n	1.0E+03	n					1.3E+01	n		8.6E-03	
2.3E+02	I	6.7E-02	I	3.0E-03	I				M	1	0.1		Benzidine	92-87-5	5.0E-04	c	7.5E-03	c	1.4E-05	c	1.8E-04	c	9.2E-05	c		2.4E-07	
				4.0E+00	I					1	0.1		Benzoic Acid	65-85-0	2.4E+05	nm	2.5E+06	nm					5.8E+04	n		1.4E+01	
1.3E+01	I							V		1		3.2E+02	Benzotrichloride	98-07-7	4.9E-02	c	2.2E-01	c					2.6E-03	c		5.6E-06	
				1.0E-01	P					1	0.1		Benzyl Alcohol	100-51-6	6.1E+03	n	6.2E+04	n					1.5E+03	n		3.7E-01	
1.7E-01	I	4.9E-05	C	2.0E-03	P	1.0E-03	P	V		1		1.5E+03	Benzyl Chloride	100-44-7	1.0E+00	c*	4.9E+00	c*	5.0E-02	c*	2.5E-01	c*	7.7E-02	c*		8.4E-05	
		2.4E-03	I	2.0E-03	I	2.0E-05	I			0.007			Beryllium and compounds	7440-41-7	1.6E+02	n	2.0E+03	n	1.0E-03	c*	5.1E-03	c*	1.6E+01	n	4.0E+00	1.3E+01	3.2E+00

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Toxicity and Chemical-specific Information												Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	vol	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
				1.0E-04	I					1	0.1		Bidrin	141-66-2	6.1E+00	n	6.2E+01	n					1.6E+00	n		3.6E-04	
				9.0E-03	P					1	0.1		Bifenox	42576-02-3	5.5E+02	n	5.5E+03	n					7.5E+01	n		5.7E-01	
				1.5E-02	I					1	0.1		Biphenthrin	82657-04-3	9.2E+02	n	9.2E+03	n					2.3E+02	n		1.1E+03	
8.0E-03	X			5.0E-02	I	4.0E-04	X	V		1		2.1E+02	Biphenyl, 1,1'-	92-52-4	5.1E+01	n	2.1E+02	n	4.2E-01	n	1.8E+00	n	8.3E-01	n		8.7E-03	
7.0E-02	H	1.0E-05	H	4.0E-02	I			V		1		1.0E+03	Bis(2-chloro-1-methylethyl) ether	108-60-1	4.6E+00	c	2.2E+01	c	2.4E-01	c	1.2E+00	c	3.1E-01	c		1.1E-04	
				3.0E-03	P					1	0.1		Bis(2-chloroethoxy)methane	111-91-1	1.8E+02	n	1.8E+03	n					4.7E+01	n		1.1E-02	
1.1E+00	I	3.3E-04	I					V		1		5.1E+03	Bis(2-chloroethyl)ether	111-44-4	2.1E-01	c	1.0E+00	c	7.4E-03	c	3.7E-02	c	1.2E-02	c		3.1E-06	
1.4E-02	I	2.4E-06	C	2.0E-02	I					1	0.1		Bis(2-ethylhexyl)phthalate	117-81-7	3.5E+01	c*	1.2E+02	c	1.0E+00	c	5.1E+00	c	7.1E-02	c*	6.0E+00	1.7E-02	1.4E+00
2.2E+02	I	6.2E-02	I					V		1		4.2E+03	Bis(chloromethyl)ether	542-88-1	7.7E-05	c	3.9E-04	c	3.9E-05	c	2.0E-04	c	6.2E-05	c		1.5E-08	
				5.0E-02	I					1	0.1		Bisphenol A	80-05-7	3.1E+03	n	3.1E+04	n					5.8E+02	n		4.4E+01	
				2.0E-01	I	2.0E-02	H			1			Boron And Borates Only	7440-42-8	1.6E+04	n	2.0E+05	nm	2.1E+01	n	8.8E+01	n	3.1E+03	n		9.9E+00	
				4.0E-02	C	1.3E-02	C			1			Boron Trifluoride	7637-07-2	3.1E+03	n	4.1E+04	n	1.4E+01	n	5.7E+01	n	6.2E+02	n			
7.0E-01	I			4.0E-03	I					1			Bromate	15541-45-4	9.1E-01	c	4.1E+00	c					9.6E-02	c	1.0E+01	7.4E-04	7.7E-02
2.0E+00	X	6.0E-04	X					V		1		2.4E+03	Bromo-2-chloroethane, 1-	107-04-0	2.4E-02	c	1.2E-01	c	4.1E-03	c	2.0E-02	c	6.5E-03	c		1.8E-06	
				8.0E-03	I	6.0E-02	I	V		1		6.8E+02	Bromobenzene	108-86-1	3.0E+02	n	1.8E+03	ns	6.3E+01	n	2.6E+02	n	5.4E+01	n		3.6E-02	
						4.0E-02	X	V		1		4.0E+03	Bromochloromethane	74-97-5	1.6E+02	n	6.8E+02	n	4.2E+01	n	1.8E+02	n	8.3E+01	n		2.1E-02	
6.2E-02	I	3.7E-05	C	2.0E-02	I			V		1		9.3E+02	Bromodichloromethane	75-27-4	2.7E-01	c	1.4E+00	c	6.6E-02	c	3.3E-01	c	1.2E-01	c	8.0E+01(F)	3.2E-05	2.2E-02
7.9E-03	I	1.1E-06	I	2.0E-02	I					1	0.1		Bromoform	75-25-2	6.2E+01	c*	2.2E+02	c*	2.2E+00	c	1.1E+01	c	7.9E+00	c*	8.0E+01(F)	2.1E-03	2.1E-02
				1.4E-03	I	5.0E-03	I	V		1		3.6E+03	Bromomethane	74-83-9	7.3E+00	n	3.2E+01	n	5.2E+00	n	2.2E+01	n	7.0E+00	n		1.8E-03	
				5.0E-03	H					1	0.1		Bromophos	2104-96-3	3.1E+02	n	3.1E+03	n					2.6E+01	n		1.1E-01	
				2.0E-02	I					1	0.1		Bromoxynil	1689-84-5	1.2E+03	n	1.2E+04	n					3.1E+02	n		2.7E-01	
				2.0E-02	I					1	0.1		Bromoxynil Octanoate	1689-99-2	1.2E+03	n	1.2E+04	n					1.0E+02	n		8.7E-01	
3.4E+00	C	3.0E-05	I			2.0E-03	I	V		1		6.7E+02	Butadiene, 1,3-	106-99-0	5.4E-02	c*	2.6E-01	c*	8.1E-02	c*	4.1E-01	c*	1.6E-02	c		8.6E-06	
				1.0E-01	I					1	0.1		Butanol, N-	71-36-3	6.1E+03	n	6.2E+04	n					1.5E+03	n		3.2E-01	
1.9E-03	P			2.0E-01	I					1	0.1		Butyl Benzyl Phthlate	85-68-7	2.6E+02	c*	9.1E+02	c					1.4E+01	c*		2.0E-01	
				2.0E+00	P	3.0E+01	P			1	0.1		Butyl alcohol, sec-	78-92-2	1.2E+05	nm	1.2E+06	nm	3.1E+04	n	1.3E+05	n	3.1E+04	n		6.3E+00	
				5.0E-02	I					1	0.1		Butylate	2008-41-5	3.1E+03	n	3.1E+04	n					3.4E+02	n		3.3E-01	
2.0E-04	C	5.7E-08	C							1	0.1		Butylated hydroxyanisole	25013-16-5	2.4E+03	c	8.6E+03	c	4.3E+01	c	2.2E+02	c	3.4E+02	c		6.3E-01	
				5.0E-02	P			V		1		1.1E+02	Butylbenzene, n-	104-51-8	3.9E+03	ns	5.1E+04	ns					7.8E+02	n		2.5E+00	
				1.0E+00	I					1	0.1		Butylphthalyl Butylglycolate	85-70-1	6.1E+04	n	6.2E+05	nm					1.6E+04	n		3.5E+02	
				2.0E-02	A					1	0.1		Cacodylic Acid	75-60-5	1.2E+03	n	1.2E+04	n					3.1E+02	n			
		1.8E-03	I	1.0E-03	I	2.0E-05	C			0.025	0.001		Cadmium (Diet)	7440-43-9	7.0E+01	n	8.0E+02	n									
		1.8E-03	I	5.0E-04	I	2.0E-05	C			0.05	0.001		Cadmium (Water)	7440-43-9					1.4E-03	c*	6.8E-03	c*	6.9E+00	n	5.0E+00	5.2E-01	3.8E-01
				5.0E-01	I					1	0.1		Caprolactam	105-60-2	3.1E+04	n	3.1E+05	nm					7.7E+03	n		1.9E+00	
1.5E-01	C	4.3E-05	C	2.0E-03	I					1	0.1		Captafol	2425-06-1	3.2E+00	c*	1.1E+01	c	5.7E-02	c	2.9E-01	c	3.5E-01	c*		6.1E-04	
2.3E-03	C	6.6E-07	C	1.3E-01	I					1	0.1		Captan	133-06-2	2.1E+02	c*	7.5E+02	c	3.7E+00	c	1.9E+01	c	2.7E+01	c*		1.9E-02	
				1.0E-01	I					1	0.1		Carbaryl	63-25-2	6.1E+03	n	6.2E+04	n					1.4E+03	n		1.3E+00	
				5.0E-03	I					1	0.1		Carbofuran	1563-66-2	3.1E+02	n	3.1E+03	n					7.3E+01	n	4.0E+01	2.8E-02	1.6E-02
				1.0E-01	I	7.0E-01	I	V		1		7.4E+02	Carbon Disulfide	75-15-0	8.2E+02	ns	3.7E+03	ns	7.3E+02	n	3.1E+03	n	7.2E+02	n		2.1E-01	
7.0E-02	I	6.0E-06	I	4.0E-03	I	1.0E-01	I	V		1		4.6E+02	Carbon Tetrachloride	56-23-5	6.1E-01	c	3.0E+00	c	4.1E-01	c	2.0E+00	c	3.9E-01	c	5.0E+00	1.5E-04	1.9E-03
				1.0E-02	I					1	0.1		Carbosulfan	55285-14-8	6.1E+02	n	6.2E+03	n					1.6E+02	n		3.8E+00	
				1.0E-01	I					1	0.1		Carboxin	5234-68-4	6.1E+03	n	6.2E+04	n					1.5E+03	n		8.0E-01	
						9.0E-04	I			1			Ceric oxide	1306-38-3	1.3E+06	nm	5.4E+06	nm	9.4E-01	n	3.9E+00	n					
				1.0E-01	I					1	0.1		Chloral Hydrate	302-17-0	6.1E+03	n	6.2E+04	n					1.5E+03	n		3.1E-01	
				1.5E-02	I					1	0.1		Chloramben	133-90-4	9.2E+02	n	9.2E+03	n					2.3E+02	n		5.7E-02	
4.0E-01	H									1	0.1		Chloranil	118-75-2	1.2E+00	c	4.3E+00	c					1.7E-01	c		1.4E-04	
3.5E-01	I	1.0E-04	I	5.0E-04	I	7.0E-04	I			1	0.04		Chlordane	12789-03-6	1.6E+00	c*	6.5E+00	c*	2.4E-02	c*	1.2E-01	c*	2.7E-02	c*	2.0E+00	1.8E-03	1.4E-01
1.0E+01	I	4.6E-03	C	3.0E-04	I					1	0.1		Chlordecone (Kepone)	143-50-0	4.9E-02	c	1.7E-01	c	5.3E-04	c	2.7E-03	c	3.0E-03	c		1.1E-04	
				7.0E-04	A					1	0.1		Chlorfenvinphos	470-90-6	4.3E+01	n	4.3E+02	n					8.6E+00	n		2.3E-02	
				2.0E-02	I					1	0.1		Chlorimuron, Ethyl-	90982-32-4	1.2E+03	n	1.2E+04	n					3.0E+02	n		1.0E-01	
				1.0E-01	I	1.5E-04	A			1			Chlorine	7782-50-5	7.5E+03	n	9.1E+04	n	1.5E-01	n	6.4E-01	n	1.6E+03	n		7.0E-01	
				3.0E-02	I	2.0E-04	I			1			Chlorine Dioxide	10049-04-4	2.3E+03	n	3.0E+04	n	2.1E-01	n	8.8E-01	n	4.7E+02	n			
				3.0E-02	I					1			Chlorite (Sodium Salt)	7758-19-2	2.3E+03	n	3.1E+04	n					4.7E+02	n	1.0E+03		
						5.0E+01	I	V		1		1.2E+03	Chloro-1,1-difluoroethane, 1-	75-68-3	5.8E+04	ns	2.4E+05	nms	5.2E+04	n	2.2E+05	n	1.0E+05	n		5.2E+01	
		3.0E-04	I	2.0E-02	H	2.0E-02	I	V		1		7.5E+02	Chloro-1,3-butadiene, 2-	126-99-8	9.4E-03	c	4.7E-02	c	8.1E-03	c	4.1E-02	c	1.6E-02	c		8.5E-06	
4.6E-01	H									1	0.1		Chloro-2-methylaniline HCl, 4-	3165-93-3	1.1E+00	c	3.7E+00	c					1.3E-01	c		7.4E-05	
1.0E-01	P	7.7E-05	C	3.0E-03	X					1	0.1		Chloro-2-methylaniline, 4-	95-69-2	4.9E+00	c*	1.7E+01	c	3.2E-02	c	1.6E-01	c	6.7E-01	c*		3.8E-04	
2.7E-01	X							V		1	0.1	2.8E+04	Chloroacetaldehyde, 2-	107-20-0	1.8E+00	c	6.4E+00	c					2.5E-01	c		5.0E-05	
				2.0E-03	H					1	0.1		Chloroacetic Acid	79-11-8	1.2E+02	n	1.2E+03	n					3.1E+01	n	6.0E+01	6.3E-03	1.2E-02
						3.0E-05	I			1	0.1		Chloroacetophenone, 2-	532-27-4	4.3E+04	n	1.8E+05	nm	3.1E-02	n	1.3E-01	n					
2.0E-01	P			4.0E-03	I					1	0.1		Chloroaniline, p-	106-47-8	2.4E+00	c	8.6E+00	c					3.2E-01	c		1.3E-04	
				2.0E-02	I	5.0E-02	P	V		1		7.6E+02	Chlorobenzene	108-90-7	2.9E+02	n	1.4E+03	ns	5.2E+01	n	2.2E+02	n	7.2E+01	n	1.0E+02	4.9E-02	6.8E-02
1.1E-01	C	3.1E-05	C	2.0E-02	I					1	0.1		Chlorobenzilate	510-15-6	4.4E+00	c	1.6E+01	c	7.8E-02	c	4.0E-01	c	2.7E-01	c		8.8E-04	
				3.0E-02	X					1	0.1		Chlorobenzoic Acid, p-	74-11-3	1.8E+03	n	1.8E+04	n					3.9E+02	n		9.9E-02	
				3.0E-03	P	3.0E-01	P	V		1		1.2E+02	Chlorobenzotrifluoride, 4-	98-56-6	2.1E+02	ns	2.3E+03	ns	3.1E+02	n	1.3E+03	n	2.6E+01	n		9.3E-02	

Key: I = IRIS; P = PPRTV; A = ATSDR; C = Cal EPA; X = PPRTV Appendix; H = HEAST; J = New Jersey; Y = New York; O = EPA Office of Water; E = Environmental Criteria and Assessment Office; S = see user guide Section 5; L = see user guide on lead; M = mutagen; V = volatile; F = See FAQ; c = cancer; * = where: n SL < 100X c SL; ** = where n SL < 10X c SL; n = noncancer; m = Concentration may exceed ceiling limit (See User Guide); s = Concentration may exceed Csat (See User Guide); SSL values are based on DAF=1

Toxicity and Chemical-specific Information													Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	voc	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
				4.0E-02	P			V		1		7.3E+02	Chlorobutane, 1-	109-69-3	3.1E+03	ns	4.1E+04	ns					4.8E+02	n		2.0E-01	
						5.0E+01	I	V		1		1.7E+03	Chlorodifluoromethane	75-45-6	5.3E+04	ns	2.2E+05	nms	5.2E+04	n	2.2E+05	n	1.0E+05	n		4.3E+01	
3.1E-02	C	2.3E-05	I	1.0E-02	I	9.8E-02	A	V		1		2.5E+03	Chloroform	67-66-3	2.9E-01	c	1.5E+00	c	1.1E-01	c	5.3E-01	c	1.9E-01	c	8.0E+01(F)	5.3E-05	2.2E-02
						9.0E-02	I	V		1		1.3E+03	Chloromethane	74-87-3	1.2E+02	n	5.0E+02	n	9.4E+01	n	3.9E+02	n	1.9E+02	n		4.9E-02	
2.4E+00	C	6.9E-04	C					V		1		2.6E+04	Chloromethyl Methyl Ether	107-30-2	1.9E-02	c	9.4E-02	c	3.5E-03	c	1.8E-02	c	5.6E-03	c		1.2E-06	
				8.0E-02	I			V		1		1.8E+02	Chloronaphthalene, Beta-	91-58-7	6.3E+03	ns	8.2E+04	ns					5.5E+02	n		2.9E+00	
3.0E-01	P			3.0E-03	P	1.0E-05	X			1	0.1		Chloronitrobenzene, o-	88-73-3	1.6E+00	c	5.7E+00	c	1.0E-02	n	4.4E-02	n	2.0E-01	c		1.9E-04	
6.3E-03	P			1.0E-03	P	6.0E-04	P			1	0.1		Chloronitrobenzene, p-	100-00-5	6.1E+01	n	2.7E+02	c**	6.3E-01	n	2.6E+00	n	9.4E+00	c**		8.7E-03	
				5.0E-03	I			V		1		2.2E+04	Chlorophenol, 2-	95-57-8	3.9E+02	n	5.1E+03	n					7.1E+01	n		5.7E-02	
						4.0E-04	C	V		1		6.2E+02	Chloropicrin	76-06-2	2.1E+00	n	8.8E+00	n	4.2E-01	n	1.8E+00	n	8.3E-01	n		2.5E-04	
3.1E-03	C	8.9E-07	C	1.5E-02	I					1	0.1		Chlorothalonil	1897-45-6	1.6E+02	c**	5.6E+02	c*	2.7E+00	c	1.4E+01	c	1.9E+01	c*		4.3E-02	
				2.0E-02	I			V		1		9.1E+02	Chlorotoluene, o-	95-49-8	1.6E+03	ns	2.0E+04	ns					1.8E+02	n		1.7E-01	
				2.0E-02	X			V		1		2.5E+02	Chlorotoluene, p-	106-43-4	1.6E+03	ns	2.0E+04	ns					1.9E+02	n		1.8E-01	
2.4E+02	C	6.9E-02	C							1	0.1		Chlorozotocin	54749-90-5	2.0E-03	c	7.2E-03	c	3.5E-05	c	1.8E-04	c	2.8E-04	c		6.2E-08	
				2.0E-01	I					1	0.1		Chlorpropham	101-21-3	1.2E+04	n	1.2E+05	nm					2.2E+03	n		1.9E+00	
				1.0E-03	A					1	0.1		Chlorpyrifos	2921-88-2	6.1E+01	n	6.2E+02	n					6.2E+00	n		9.2E-02	
				1.0E-02	H					1	0.1		Chlorpyrifos Methyl	5598-13-0	6.1E+02	n	6.2E+03	n					8.9E+01	n		4.1E-01	
				5.0E-02	I					1	0.1		Chlorsulfuron	64902-72-3	3.1E+03	n	3.1E+04	n					7.7E+02	n		6.5E-01	
				8.0E-04	H					1	0.1		Chlorthiophos	60238-56-4	4.9E+01	n	4.9E+02	n					2.0E+00	n		5.2E-02	
				1.5E+00	I					0.013			Chromium(III), Insoluble Salts	16065-83-1	1.2E+05	nm	1.5E+06	nm					1.6E+04	n		2.8E+07	
5.0E-01	J	8.4E-02	S	3.0E-03	I	1.0E-04	I		M	0.025			Chromium(VI)	18540-29-9	2.9E-01	c	5.6E+00	c	1.1E-05	c	1.5E-04	c	3.1E-02	c		5.9E-04	
										0.013			Chromium, Total	7440-47-3											1.0E+02		1.8E+05
		9.0E-03	P	3.0E-04	P	6.0E-06	P			1			Cobalt	7440-48-4	2.3E+01	n	3.0E+02	n	2.7E-04	c*	1.4E-03	c*	4.7E+00	n		2.1E-01	
		6.2E-04	I						M	1	0.1		Coke Oven Emissions	8007-45-2					1.5E-03	c	2.0E-02	c					
				4.0E-02	H					1			Copper	7440-50-8	3.1E+03	n	4.1E+04	n					6.2E+02	n	1.3E+03	2.2E+01	4.6E+01
				5.0E-02	I	6.0E-01	C			1	0.1		Cresol, m-	108-39-4	3.1E+03	n	3.1E+04	n	6.3E+02	n	2.6E+03	n	7.2E+02	n		5.7E-01	
				5.0E-02	I	6.0E-01	C			1	0.1		Cresol, o-	95-48-7	3.1E+03	n	3.1E+04	n	6.3E+02	n	2.6E+03	n	7.2E+02	n		5.8E-01	
				1.0E-01	A	6.0E-01	C			1	0.1		Cresol, p-	106-44-5	6.1E+03	n	6.2E+04	n	6.3E+02	n	2.6E+03	n	1.4E+03	n		1.1E+00	
				1.0E-01	A					1	0.1		Cresol, p-chloro-m-	59-50-7	6.1E+03	n	6.2E+04	n					1.1E+03	n		1.3E+00	
				1.0E-01	A	6.0E-01	C			1	0.1		Cresols	1319-77-3	6.1E+03	n	6.2E+04	n	6.3E+02	n	2.6E+03	n	1.4E+03	n		1.2E+00	
1.9E+00	H			1.0E-03	P			V		1		1.7E+04	Crotonaldehyde, trans-	123-73-9	3.4E-01	c	1.5E+00	c					3.5E-02	c		7.1E-06	
				1.0E-01	I	4.0E-01	I	V		1		2.7E+02	Cumene	98-82-8	2.1E+03	ns	1.1E+04	ns	4.2E+02	n	1.8E+03	n	3.9E+02	n		6.4E-01	
2.2E-01	C	6.3E-05	C							1	0.1		Cupferron	135-20-6	2.2E+00	c	7.8E+00	c	3.9E-02	c	1.9E-01	c	3.1E-01	c		5.3E-04	
8.4E-01	H			2.0E-03	H					1	0.1		Cyanazine	21725-46-2	5.8E-01	c	2.1E+00	c					7.6E-02	c		3.5E-05	
													Cyanides														
				1.0E-03	I					1			~Calcium Cyanide	592-01-8	7.8E+01	n	1.0E+03	n					1.6E+01	n			
				5.0E-03	I					1			~Copper Cyanide	544-92-3	3.9E+02	n	5.1E+03	n					7.8E+01	n			
				6.0E-04	I			V		1		1.0E+07	~Cyanide (CN-)	57-12-5	4.7E+01	n	6.1E+02	n					9.3E+00	n	2.0E+02	9.4E-02	2.0E+00
				1.0E-03	I			V		1			~Cyanogen	460-19-5	7.8E+01	n	1.0E+03	n					1.6E+01	n			
				9.0E-02	I			V		1			~Cyanogen Bromide	506-68-3	7.0E+03	n	9.2E+04	n					1.4E+03	n			
				5.0E-02	I			V		1			~Cyanogen Chloride	506-77-4	3.9E+03	n	5.1E+04	n					7.8E+02	n			
				6.0E-04	I	8.0E-04	I	V		1			~Hydrogen Cyanide	74-90-8	4.7E+01	n	6.1E+02	n	8.3E-01	n	3.5E+00	n	1.4E+00	n			
				2.0E-03	I					1			~Potassium Cyanide	151-50-8	1.6E+02	n	2.0E+03	n					3.1E+01	n			
				5.0E-03	I					0.04			~Potassium Silver Cyanide	506-61-6	3.9E+02	n	5.1E+03	n					5.9E+01	n			
				1.0E-01	I					0.04			~Silver Cyanide	506-64-9	7.8E+03	n	1.0E+05	nm					1.3E+03	n			
				1.0E-03	I					1			~Sodium Cyanide	143-33-9	7.8E+01	n	1.0E+03	n					1.6E+01	n	2.0E+02		
				2.0E-04	P			V		1		4.6E+03	~Thiocyanate	463-56-9	1.6E+01	n	2.0E+02	n					3.1E+00	n		6.6E-04	
				5.0E-02	I					1			~Zinc Cyanide	557-21-1	3.9E+03	n	5.1E+04	n					7.8E+02	n			
						6.0E+00	I	V		1		1.2E+02	Cyclohexane	110-82-7	7.0E+03	ns	2.9E+04	ns	6.3E+03	n	2.6E+04	n	1.3E+04	n		1.3E+01	
2.3E-02	H									1	0.1		Cyclohexane, 1,2,3,4,5-pentabromo-6-chloro-	87-84-3	2.1E+01	c	7.5E+01	c					2.1E+00	c		1.2E-02	
				5.0E+00	I	7.0E-01	P			1	0.1		Cyclohexanone	108-94-1	3.1E+05	nm	3.1E+06	nm	7.3E+02	n	3.1E+03	n	7.7E+04	n		1.8E+01	
				2.0E-01	I					1	0.1		Cyclohexylamine	108-91-8	1.2E+04	n	1.2E+05	nm					3.0E+03	n		7.9E-01	
				5.0E-03	I					1	0.1		Cyhalothrin/karate	68085-85-8	3.1E+02	n	3.1E+03	n					7.8E+01	n		5.3E+01	
				1.0E-02	I					1	0.1		Cypermethrin	52315-07-8	6.1E+02	n	6.2E+03	n					1.6E+02	n		2.5E+01	
				7.5E-03	I					1	0.1		Cyromazine	66215-27-8	4.6E+02	n	4.6E+03	n					1.2E+02	n		3.0E-02	
2.4E-01	I	6.9E-05	C							1	0.1		DDD	72-54-8	2.0E+00	c	7.2E+00	c	3.5E-02	c	1.8E-01	c	2.8E-01	c		6.6E-02	
3.4E-01	I	9.7E-05	C							1	0.1		DDE, p,p'-	72-55-9	1.4E+00	c	5.1E+00	c	2.5E-02	c	1.3E-01	c	2.0E-01	c		4.6E-02	
3.4E-01	I	9.7E-05	I	5.0E-04	I					1	0.03		DDT	50-29-3	1.7E+00	c*	7.0E+00	c*	2.5E-02	c	1.3E-01	c	2.0E-01	c*		6.7E-02	
				1.0E-02	I					1	0.1		Dacthal	1861-32-1	6.1E+02	n	6.2E+03	n					9.3E+01	n		1.1E-01	
				3.0E-02	I					1	0.1		Dalapon	75-99-0	1.8E+03	n	1.8E+04	n					4.7E+02	n	2.0E+02	9.7E-02	4.1E-02
7.0E-04	I			7.0E-03	I					1	0.1		Decabromodiphenyl ether, 2,2',3,3',4,4',5,5',6,6'- (BDE-209)	1163-19-5	4.3E+02	n	2.5E+03	c**					9.6E+01	c**		5.3E+01	
				4.0E-05	I					1	0.1		Demeton	8065-48-3	2.4E+00	n	2.5E+01	n					5.2E-01	n			
1.2E-03	I			6.0E-01	I					1	0.1		Di(2-ethylhexyl)adipate	103-23-1	4.1E+02	c*	1.4E+03	c					5.6E+01	c	4.0E+02	4.0E+00	2.9E+01
6.1E-02	H									1	0.1		Diallate	2303-16-4	8.0E+00	c	2.8E+01	c					4.6E-01	c		6.8E-04	
				7.0E-04	A					1	0.1		Diazinon	333-41-5	4.3E+01	n	4.3E+02	n					7.9E+00	n		4.9E-02	

Key: I = IRIS; P = PPRTV; A = ATSDR; C = Cal EPA; X = PPRTV Appendix; H = HEAST; J = New Jersey; Y = New York; O = EPA Office of Water; E = Environmental Criteria and Assessment Office; S = see user guide Section 5; L = see user guide on lead; M = mutagen; V = volatile; F = See FAQ; c = cancer; * = where: n SL < 100X c SL; ** = where n SL < 10X c SL; n = noncancer; m = Concentration may exceed ceiling limit (See User Guide); s = Concentration may exceed Csat (See User Guide); SSL values are based on DAF=1

Toxicity and Chemical-specific Information													Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	voc	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
8.0E-01	P	6.0E-03	P	2.0E-04	P	2.0E-04	I	V	M	1		9.8E+02	Dibromo-3-chloropropane, 1,2-	96-12-8	5.4E-03	c	6.9E-02	c	1.6E-04	c	2.0E-03	c	3.2E-04	c	2.0E-01	1.4E-07	8.6E-05
				1.0E-02	I					1	0.1		Dibromobenzene, 1,4-	106-37-6	6.1E+02	n	6.2E+03	n					9.8E+01	n		9.3E-02	
8.4E-02	I	2.7E-05	C	2.0E-02	I			V		1	0.1	8.0E+02	Dibromochloromethane	124-48-1	6.8E-01	c	3.3E+00	c	9.0E-02	c	4.5E-01	c	1.5E-01	c	8.0E+01(F)	3.9E-05	2.1E-02
2.0E+00	I	6.0E-04	I	9.0E-03	I	9.0E-03	I	V		1		1.3E+03	Dibromoethane, 1,2-	106-93-4	3.4E-02	c	1.7E-01	c	4.1E-03	c	2.0E-02	c	6.5E-03	c	5.0E-02	1.8E-06	1.4E-05
				1.0E-02	H	4.0E-03	X	V		1		2.8E+03	Dibromomethane (Methylene Bromide)	74-95-3	2.5E+01	n	1.1E+02	n	4.2E+00	n	1.8E+01	n	7.9E+00	n		1.9E-03	
				1.0E-01	I					1	0.1		Dibutyl Phthalate	84-74-2	6.1E+03	n	6.2E+04	n					6.7E+02	n		1.7E+00	
				3.0E-04	P					1	0.1		Dibutyltin Compounds	NA	1.8E+01	n	1.8E+02	n					4.7E+00	n			
				3.0E-02	I					1	0.1		Dicamba	1918-00-9	1.8E+03	n	1.8E+04	n					4.4E+02	n		1.1E-01	
		4.2E-03	P					V		1		5.2E+02	Dichloro-2-butene, 1,4-	764-41-0	6.9E-03	c	3.5E-02	c	5.8E-04	c	2.9E-03	c	1.2E-03	c		5.4E-07	
		4.2E-03	P					V		1	0.1	5.2E+02	Dichloro-2-butene, cis-1,4-	1476-11-5	6.9E-03	c	3.5E-02	c	5.8E-04	c	2.9E-03	c	1.2E-03	c		5.4E-07	
		4.2E-03	P					V		1	0.1	7.6E+02	Dichloro-2-butene, trans-1,4-	110-57-6	6.9E-03	c	3.5E-02	c	5.8E-04	c	2.9E-03	c	1.2E-03	c		5.4E-07	
5.0E-02	I			4.0E-03	I					1	0.1		Dichloroacetic Acid	79-43-6	9.7E+00	c*	3.4E+01	c*					1.3E+00	c*	6.0E+01	2.7E-04	1.2E-02
				9.0E-02	I	2.0E-01	H	V		1		3.8E+02	Dichlorobenzene, 1,2-	95-50-1	1.9E+03	ns	9.8E+03	ns	2.1E+02	n	8.8E+02	n	2.8E+02	n	6.0E+02	2.7E-01	5.8E-01
5.4E-03	C	1.1E-05	C	7.0E-02	A	8.0E-01	I	V		1			Dichlorobenzene, 1,4-	106-46-7	2.4E+00	c	1.2E+01	c	2.2E-01	c	1.1E+00	c	4.2E-01	c	7.5E+01	4.0E-04	7.2E-02
4.5E-01	I	3.4E-04	C							1	0.1		Dichlorobenzidine, 3,3'-	91-94-1	1.1E+00	c	3.8E+00	c	7.2E-03	c	3.6E-02	c	1.1E-01	c		7.1E-04	
				9.0E-03	X					1	0.1		Dichlorobenzophenone, 4,4'-	90-98-2	5.5E+02	n	5.5E+03	n					1.4E+02	n		8.5E-01	
				2.0E-01	I	1.0E-01	X	V		1		8.5E+02	Dichlorodifluoromethane	75-71-8	9.4E+01	n	4.0E+02	n	1.0E+02	n	4.4E+02	n	1.9E+02	n		3.0E-01	
5.7E-03	C	1.6E-06	C	2.0E-01	P			V		1		1.7E+03	Dichloroethane, 1,1-	75-34-3	3.3E+00	c	1.7E+01	c	1.5E+00	c	7.7E+00	c	2.4E+00	c		6.8E-04	
9.1E-02	I	2.6E-05	I	6.0E-03	X	7.0E-03	P	V		1		3.0E+03	Dichloroethane, 1,2-	107-06-2	4.3E-01	c*	2.2E+00	c*	9.4E-02	c*	4.7E-01	c*	1.5E-01	c*	5.0E+00	4.2E-05	1.4E-03
				5.0E-02	I	2.0E-01	I	V		1		1.2E+03	Dichloroethylene, 1,1-	75-35-4	2.4E+02	n	1.1E+03	n	2.1E+02	n	8.8E+02	n	2.6E+02	n	7.0E+00	9.3E-02	2.5E-03
				9.0E-03	H			V		1		1.3E+03	Dichloroethylene, 1,2- (Mixed Isomers)	540-59-0	7.0E+02	n	9.2E+03	ns					1.3E+02	n		3.7E-02	
				2.0E-03	I			V		1		2.4E+03	Dichloroethylene, 1,2-cis-	156-59-2	1.6E+02	n	2.0E+03	n					2.8E+01	n	7.0E+01	8.2E-03	2.1E-02
				2.0E-02	I	6.0E-02	P	V		1		1.7E+03	Dichloroethylene, 1,2-trans-	156-60-5	1.5E+02	n	6.9E+02	n	6.3E+01	n	2.6E+02	n	8.6E+01	n	1.0E+02	2.5E-02	2.9E-02
				3.0E-03	I					1	0.1		Dichlorophenol, 2,4-	120-83-2	1.8E+02	n	1.8E+03	n					3.5E+01	n		4.1E-02	
				1.0E-02	I					1	0.05		Dichlorophenoxy Acetic Acid, 2,4-	94-75-7	6.9E+02	n	7.7E+03	n					1.3E+02	n	7.0E+01	3.5E-02	1.8E-02
				8.0E-03	I					1	0.1		Dichlorophenoxy)butyric Acid, 4-(2,4-	94-82-6	4.9E+02	n	4.9E+03	n					9.1E+01	n		3.6E-02	
3.6E-02	C	1.0E-05	C	9.0E-02	A	4.0E-03	I	V		1		1.4E+03	Dichloropropane, 1,2-	78-87-5	9.4E-01	c*	4.7E+00	c*	2.4E-01	c*	1.2E+00	c*	3.8E-01	c*	5.0E+00	1.3E-04	1.7E-03
				2.0E-02	P			V		1		1.5E+03	Dichloropropane, 1,3-	142-28-9	1.6E+03	ns	2.0E+04	ns					2.9E+02	n		9.9E-02	
				3.0E-03	I					1	0.1		Dichloropropanol, 2,3-	616-23-9	1.8E+02	n	1.8E+03	n					4.7E+01	n		9.9E-03	
1.0E-01	I	4.0E-06	I	3.0E-02	I	2.0E-02	I	V		1		1.6E+03	Dichloropropene, 1,3-	542-75-6	1.7E+00	c*	8.3E+00	c*	6.1E-01	c*	3.1E+00	c*	4.1E-01	c*		1.5E-04	
2.9E-01	I	8.3E-05	C	5.0E-04	I	5.0E-04	I			1	0.1		Dichlorvos	62-73-7	1.7E+00	c*	5.9E+00	c*	2.9E-02	c*	1.5E-01	c*	2.3E-01	c*		7.0E-05	
				8.0E-03	P	7.0E-03	P	V		1		1.3E+02	Dicyclopentadiene	77-73-6	3.1E+01	n	1.3E+02	ns	7.3E+00	n	3.1E+01	n	1.2E+01	n		4.3E-02	
1.6E+01	I	4.6E-03	I	5.0E-05	I					1	0.1		Dieldrin	60-57-1	3.0E-02	c	1.1E-01	c	5.3E-04	c	2.7E-03	c	1.5E-03	c		6.1E-05	
		3.0E-04	C			5.0E-03	I			1	0.1		Diesel Engine Exhaust	NA					8.1E-03	c	4.1E-02	c					
						3.0E-03	C			1	0.1		Diethanolamine	111-42-2	4.3E+06	nm	1.8E+07	nm	3.1E+00	n	1.3E+01	n					
				8.0E-01	I					1	0.1		Diethyl Phthalate	84-66-2	4.9E+04	n	4.9E+05	nm					1.1E+04	n		4.7E+00	
				3.0E-02	P	1.0E-04	P			1	0.1		Diethylene Glycol Monobutyl Ether	112-34-5	1.8E+03	n	1.8E+04	n	1.0E-01	n	4.4E-01	n	4.7E+02	n		1.0E-01	
				6.0E-02	P	3.0E-04	P			1	0.1		Diethylene Glycol Monoethyl Ether	111-90-0	3.6E+03	n	3.6E+04	n	3.1E-01	n	1.3E+00	n	9.4E+02	n		1.9E-01	
				1.0E-03	P					1	0.1		Diethylformamide	617-84-5	6.1E+01	n	6.2E+02	n					1.6E+01	n		3.2E-03	
3.5E+02	C	1.0E-01	C							1	0.1		Diethylstilbestrol	56-53-1	1.4E-03	c	4.9E-03	c	2.4E-05	c	1.2E-04	c	4.3E-05	c		2.4E-05	
				8.0E-02	I					1	0.1		Difenzoquat	43222-48-6	4.9E+03	n	4.9E+04	n					1.2E+03	n			
				2.0E-02	I					1	0.1		Diflubenzuron	35367-38-5	1.2E+03	n	1.2E+04	n					2.2E+02	n		2.5E-01	
						4.0E+01	I	V		1		1.4E+03	Difluoroethane, 1,1-	75-37-6	5.2E+04	ns	2.2E+05	nms	4.2E+04	n	1.8E+05	n	8.3E+04	n		2.8E+01	
4.4E-02	C	1.3E-05	C					V		1	0.1	1.5E+01	Dihydrosafrole	94-58-6	2.4E-01	c	1.2E+00	c	1.9E-01	c	9.4E-01	c	3.0E-01	c		3.7E-04	
						7.0E-01	P	V		1		2.3E+03	Diisopropyl Ether	108-20-3	2.4E+03	ns	1.0E+04	ns	7.3E+02	n	3.1E+03	n	1.5E+03	n		3.7E-01	
				8.0E-02	I			V		1		5.3E+02	Diisopropyl Methylphosphonate	1445-75-6	6.3E+03	ns	8.2E+04	ns					1.2E+03	n		3.5E-01	
				2.0E-02	I					1	0.1		Dimethipin	55290-64-7	1.2E+03	n	1.2E+04	n					3.1E+02	n		6.9E-02	
				2.0E-04	I					1	0.1		Dimethoate	60-51-5	1.2E+01	n	1.2E+02	n					3.1E+00	n		7.0E-04	
1.4E-02	H									1	0.1		Dimethoxybenzidine, 3,3'-	119-90-4	3.5E+01	c	1.2E+02	c					4.7E+00	c		5.7E-03	
1.7E-03	P			6.0E-02	P					1	0.1		Dimethyl methylphosphonate	756-79-6	2.9E+02	c*	1.0E+03	c*					3.9E+01	c*		8.3E-03	
4.6E+00	C	1.3E-03	C							1	0.1		Dimethylamino azobenzene [p-]	60-11-7	1.1E-01	c	3.7E-01	c	1.9E-03	c	9.4E-03	c	4.3E-03	c		1.8E-05	
5.8E-01	H									1	0.1		Dimethylaniline HCl, 2,4-	21436-96-4	8.4E-01	c	3.0E+00	c					1.1E-01	c		6.2E-05	
2.0E-01	P			2.0E-03	X					1	0.1		Dimethylaniline, 2,4-	95-68-1	2.4E+00	c*	8.6E+00	c					3.2E-01	c*		1.8E-04	
				2.0E-03	I			V		1		8.3E+02	Dimethylaniline, N,N-	121-69-7	1.6E+02	n	2.0E+03	ns					2.7E+01	n		9.8E-03	
1.1E+01	P									1	0.1		Dimethylbenzidine, 3,3'-	119-93-7	4.4E-02	c	1.6E-01	c					5.6E-03	c		3.7E-05	
				1.0E-01	P	3.0E-02	I			1	0.1		Dimethylformamide	68-12-2	6.1E+03	n	6.2E+04	n	3.1E+01	n	1.3E+02	n	1.6E+03	n		3.2E-01	
				1.0E-04	X	2.0E-06	X			1	0.1		Dimethylhydrazine, 1,1-	57-14-7	6.1E+00	n	6.1E+01	n	2.1E-03	n	8.8E-03	n	1.6E+00	n		3.5E-04	
5.5E+02	C	1.6E-01	C							1	0.1		Dimethylhydrazine, 1,2-	540-73-8	8.8E-04	c	3.1E-03	c	1.5E-05	c	7.7E-05	c	1.2E-04	c		2.8E-08	
				2.0E-02	I					1	0.1		Dimethylphenol, 2,4-	105-67-9	1.2E+03	n	1.2E+04	n					2.7E+02	n		3.2E-01	
				6.0E-04	I					1	0.1		Dimethylphenol, 2,6-	576-26-1	3.7E+01	n	3.7E+02	n					8.1E+00	n		9.8E-03	
				1.0E-03	I					1	0.1		Dimethylphenol, 3,4-	95-65-8	6.1E+01	n	6.2E+02	n					1.4E+01	n		1.6E-02	
				1.0E-01	I			V		1		5.5E+00	Dimethylterephthalate	120-61-6	7.8E+03	ns	1.0E+05	nms					1.4E+03	n		3.8E-01	
4.5E-02	C	1.3E-05	C					V		1	0.1	1.1E+03	Dimethylvinylchloride	513-37-1	2.0E-01	c	1.0E+00	c	1.9E-01	c	9.4E-01	c	3.0E-01	c		1.8E-04	
				8.0E-05	X					1	0.1		Dinitro-o-cresol, 4,6-	534-52-1	4.9E+00	n	4.9E+01	n					1.2E+00	n		2.0E-03	
				2.0E-03	I					1	0.1		Dinitro-o-cyclohexyl Phenol, 4,6-	131-89-5	1.2E+02	n	1.2E+03	n					1.7E+01	n		5.7E-01	

Key: I = IRIS; P = PPRTV; A = ATSDR; C = Cal EPA; X = PPRTV Appendix; H = HEAST; J = New Jersey; Y = New York; O = EPA Office of Water; E = Environmental Criteria and Assessment Office; S = see user guide Section 5; L = see user guide on lead; M = mutagen; V = volatile; F = See FAQ; c = cancer; * = where: n SL < 100X c SL; ** = where n SL < 10X c SL; n = noncancer; m = Concentration may exceed ceiling limit (See User Guide); s = Concentration may exceed Csat (See User Guide); SSL values are based on DAF=1

Toxicity and Chemical-specific Information													Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	vo c	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
				1.0E-04	P					1	0.1		Dinitrobenzene, 1,2-	528-29-0	6.1E+00	n	6.2E+01	n					1.5E+00	n		1.4E-03	
				1.0E-04	I					1	0.1		Dinitrobenzene, 1,3-	99-65-0	6.1E+00	n	6.2E+01	n					1.5E+00	n		1.4E-03	
				1.0E-04	P					1	0.1		Dinitrobenzene, 1,4-	100-25-4	6.1E+00	n	6.2E+01	n					1.5E+00	n		1.4E-03	
				2.0E-03	I					1	0.1		Dinitrophenol, 2,4-	51-28-5	1.2E+02	n	1.2E+03	n					3.0E+01	n		3.4E-02	
6.8E-01	I									1	0.1		Dinitrotoluene Mixture, 2,4/2,6-	25321-14-6	7.2E-01	c	2.5E+00	c					9.2E-02	c		1.3E-04	
3.1E-01	C	8.9E-05	C	2.0E-03	I					1	0.102		Dinitrotoluene, 2,4-	121-14-2	1.6E+00	c*	5.5E+00	c	2.7E-02	c	1.4E-01	c	2.0E-01	c		2.8E-04	
				1.0E-03	P					1	0.099		Dinitrotoluene, 2,6-	606-20-2	6.1E+01	n	6.2E+02	n					1.5E+01	n		2.0E-02	
				2.0E-03	S					1	0.006		Dinitrotoluene, 2-Amino-4,6-	35572-78-2	1.5E+02	n	2.0E+03	n					3.0E+01	n		2.3E-02	
				2.0E-03	S					1	0.009		Dinitrotoluene, 4-Amino-2,6-	19406-51-0	1.5E+02	n	1.9E+03	n					3.0E+01	n		2.3E-02	
				1.0E-03	I					1	0.1		Dinoseb	88-85-7	6.1E+01	n	6.2E+02	n					1.1E+01	n	7.0E+00	9.8E-02	6.2E-02
1.0E-01	I	7.7E-06	C	3.0E-02	I	3.0E+00	C			1	0.1		Dioxane, 1,4-	123-91-1	4.9E+00	c	1.7E+01	c	3.2E-01	c	1.6E+00	c	6.7E-01	c		1.4E-04	
													Dioxins														
6.2E+03	I	1.3E+00	I							1	0.03		~Hexachlorodibenzo-p-dioxin, Mixture	NA	9.4E-05	c	3.9E-04	c	1.9E-06	c	9.4E-06	c	1.1E-05	c		1.5E-05	
1.3E+05	C	3.8E+01	C	7.0E-10	I	4.0E-08	C			1	0.03		~TCDD, 2,3,7,8-	1746-01-6	4.5E-06	c*	1.8E-05	c*	6.4E-08	c	3.2E-07	c	5.2E-07	c*	3.0E-05	2.6E-07	1.5E-05
				3.0E-02	I					1	0.1		Diphenamid	957-51-7	1.8E+03	n	1.8E+04	n					4.7E+02	n		4.6E+00	
				8.0E-04	X					1	0.1		Diphenyl Sulfone	127-63-9	4.9E+01	n	4.9E+02	n					1.1E+01	n		2.8E-02	
				2.5E-02	I					1	0.1		Diphenylamine	122-39-4	1.5E+03	n	1.5E+04	n					2.4E+02	n		4.4E-01	
8.0E-01	I	2.2E-04	I							1	0.1		Diphenylhydrazine, 1,2-	122-66-7	6.1E-01	c	2.2E+00	c	1.1E-02	c	5.6E-02	c	6.7E-02	c		2.2E-04	
				2.2E-03	I					1	0.1		Diquat	85-00-7	1.3E+02	n	1.4E+03	n					3.4E+01	n	2.0E+01	6.5E-01	3.7E-01
7.4E+00	C	2.1E-03	C							1	0.1		Direct Black 38	1937-37-7	6.6E-02	c	2.3E-01	c	1.2E-03	c	5.8E-03	c	9.1E-03	c		4.4E+00	
7.4E+00	C	2.1E-03	C							1	0.1		Direct Blue 6	2602-46-2	6.6E-02	c	2.3E-01	c	1.2E-03	c	5.8E-03	c	9.1E-03	c		1.4E+01	
6.7E+00	C	1.9E-03	C							1	0.1		Direct Brown 95	16071-86-6	7.3E-02	c	2.6E-01	c	1.3E-03	c	6.5E-03	c	1.0E-02	c			
				4.0E-05	I					1	0.1		Disulfoton	298-04-4	2.4E+00	n	2.5E+01	n					3.8E-01	n		7.1E-04	
				1.0E-02	I			V		1	0.1	2.9E+03	Dithiane, 1,4-	505-29-3	6.1E+02	n	6.2E+03	ns					1.5E+02	n		7.6E-02	
				2.0E-03	I					1	0.1		Diuron	330-54-1	1.2E+02	n	1.2E+03	n					2.8E+01	n		1.2E-02	
				4.0E-03	I					1	0.1		Dodine	2439-10-3	2.4E+02	n	2.5E+03	n					6.2E+01	n		3.2E-01	
				2.5E-02	I			V		1		4.1E+02	EPTC	759-94-4	2.0E+03	ns	2.6E+04	ns					2.9E+02	n		1.5E-01	
				6.0E-03	I					1	0.1		Endosulfan	115-29-7	3.7E+02	n	3.7E+03	n					7.8E+01	n		1.1E+00	
				2.0E-02	I					1	0.1		Endothall	145-73-3	1.2E+03	n	1.2E+04	n					3.0E+02	n	1.0E+02	7.1E-02	2.4E-02
				3.0E-04	I					1	0.1		Endrin	72-20-8	1.8E+01	n	1.8E+02	n					1.7E+00	n	2.0E+00	6.8E-02	8.1E-02
9.9E-03	I	1.2E-06	I	6.0E-03	P	1.0E-03	I	V		1		1.1E+04	Epichlorohydrin	106-89-8	2.0E+01	n	8.8E+01	n	1.0E+00	n	4.4E+00	n	2.0E+00	n		4.5E-04	
						2.0E-02	I	V		1		1.5E+04	Epoxybutane, 1,2-	106-88-7	1.7E+02	n	7.2E+02	n	2.1E+01	n	8.8E+01	n	4.2E+01	n		9.2E-03	
				5.0E-03	I					1	0.1		Ethephon	16672-87-0	3.1E+02	n	3.1E+03	n					7.8E+01	n		1.6E-02	
				5.0E-04	I					1	0.1		Ethion	563-12-2	3.1E+01	n	3.1E+02	n					3.2E+00	n		6.3E-03	
				1.0E-01	P	6.0E-02	P			1	0.1		Ethoxyethanol Acetate, 2-	111-15-9	6.1E+03	n	6.2E+04	n	6.3E+01	n	2.6E+02	n	1.5E+03	n		3.2E-01	
				4.0E-01	H	2.0E-01	I			1	0.1		Ethoxyethanol, 2-	110-80-5	2.4E+04	n	2.5E+05	nm	2.1E+02	n	8.8E+02	n	6.2E+03	n		1.3E+00	
				9.0E-01	I			V		1		1.1E+04	Ethyl Acetate	141-78-6	7.0E+04	ns	9.2E+05	nms					1.4E+04	n		2.9E+00	
4.8E-02	H							V		1		2.5E+03	Ethyl Acrylate	140-88-5	1.3E+01	c	6.0E+01	c					1.4E+00	c		3.0E-04	
						1.0E+01	I	V		1		2.1E+03	Ethyl Chloride	75-00-3	1.5E+04	ns	6.1E+04	ns	1.0E+04	n	4.4E+04	n	2.1E+04	n		5.9E+00	
				2.0E-01	I			V		1		1.0E+04	Ethyl Ether	60-29-7	1.6E+04	ns	2.0E+05	nms					3.1E+03	n		6.8E-01	
				9.0E-02	H	3.0E-01	P	V		1		1.1E+03	Ethyl Methacrylate	97-63-2	1.5E+03	ns	7.5E+03	ns	3.1E+02	n	1.3E+03	n	4.2E+02	n		9.9E-02	
				1.0E-05	I					1	0.1		Ethyl-p-nitrophenyl Phosphonate	2104-64-5	6.1E-01	n	6.2E+00	n					6.6E-02	n		2.1E-03	
1.1E-02	C	2.5E-06	C	1.0E-01	I	1.0E+00	I	V		1		4.8E+02	Ethylbenzene	100-41-4	5.4E+00	c	2.7E+01	c	9.7E-01	c	4.9E+00	c	1.3E+00	c	7.0E+02	1.5E-03	7.8E-01
				3.0E-02	P					1	0.1		Ethylene Cyanohydrin	109-78-4	1.8E+03	n	1.8E+04	n					4.7E+02	n		9.5E-02	
				9.0E-02	P					1	0.1		Ethylene Diamine	107-15-3	5.5E+03	n	5.5E+04	n					1.4E+03	n		3.2E-01	
				2.0E+00	I	4.0E-01	C			1	0.1		Ethylene Glycol	107-21-1	1.2E+05	nm	1.2E+06	nm	4.2E+02	n	1.8E+03	n	3.1E+04	n		6.3E+00	
				1.0E-01	I	1.6E+00	I			1	0.1		Ethylene Glycol Monobutyl Ether	111-76-2	6.1E+03	n	6.2E+04	n	1.7E+03	n	7.0E+03	n	1.5E+03	n		3.2E-01	
3.1E-01	C	8.8E-05	C			3.0E-02	C	V		1		1.2E+05	Ethylene Oxide	75-21-8	1.7E-01	c	8.3E-01	c	2.8E-02	c	1.4E-01	c	4.4E-02	c		9.1E-06	
4.5E-02	C	1.3E-05	C	8.0E-05	I					1	0.1		Ethylene Thiourea	96-45-7	4.9E+00	n	3.8E+01	c**	1.9E-01	c	9.4E-01	c	1.2E+00	n		2.8E-04	
6.5E+01	C	1.9E-02	C					V		1	0.1	1.5E+05	Ethyleneimine	151-56-4	2.3E-03	c	1.0E-02	c	1.3E-04	c	6.5E-04	c	2.1E-04	c		4.5E-08	
				3.0E+00	I					1	0.1		Ethylphthalyl Ethyl Glycolate	84-72-0	1.8E+05	nm	1.8E+06	nm					4.5E+04	n		1.0E+02	
				8.0E-03	I					1	0.1		Express	101200-48-0	4.9E+02	n	4.9E+03	n					1.3E+02	n		4.9E-02	
				2.5E-04	I					1	0.1		Fenamiphos	22224-92-6	1.5E+01	n	1.5E+02	n					3.4E+00	n		3.3E-03	
				2.5E-02	I					1	0.1		Fenpropathrin	39515-41-8	1.5E+03	n	1.5E+04	n					4.6E+01	n		2.1E+00	
				1.3E-02	I					1	0.1		Fluometuron	2164-17-2	7.9E+02	n	8.0E+03	n					1.9E+02	n		1.4E-01	
				4.0E-02	C	1.3E-02	C			1			Fluoride	16984-48-8	3.1E+03	n	4.1E+04	n	1.4E+01	n	5.7E+01	n	6.2E+02	n		9.3E+01	
				6.0E-02	I	1.3E-02	C			1			Fluorine (Soluble Fluoride)	7782-41-4	4.7E+03	n	6.1E+04	n	1.4E+01	n	5.7E+01	n	9.3E+02	n	4.0E+03	1.4E+02	6.0E+02
				8.0E-02	I					1	0.1		Fluridone	59756-60-4	4.9E+03	n	4.9E+04	n					1.1E+03	n		1.3E+02	
				2.0E-02	I					1	0.1		Flurprimidol	56425-91-3	1.2E+03	n	1.2E+04	n					2.6E+02	n		1.2E+00	
				6.0E-02	I					1	0.1		Flutolanil	66332-96-5	3.7E+03	n	3.7E+04	n					7.2E+02	n		3.9E+00	
				1.0E-02	I					1	0.1		Fluvalinate	69409-94-5	6.1E+02	n	6.2E+03	n					1.6E+02	n		2.3E+02	
3.5E-03	I			1.0E-01	I					1	0.1		Folpet	133-07-3	1.4E+02	c*	4.9E+02	c					1.7E+01	c*		4.1E-03	
1.9E-01	I									1	0.1		Fomesafen	72178-02-0	2.6E+00	c	9.1E+00	c					3.4E-01	c		1.1E-03	
				2.0E-03	I					1	0.1		Fonofos	944-22-9	1.2E+02	n	1.2E+03	n					1.8E+01	n		3.5E-02	
		1.3E-05	I	2.0E-01	I	9.8E-03	A			1	0.1		Formaldehyde	50-00-0	1.2E+04	n	1.2E+05	nm	1.9E-01	c*	9.4E-01	c*	3.1E+03	n		6.2E-01	

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Toxicity and Chemical-specific Information													Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	vo c	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
				9.0E-01	P	3.0E-04	X			1	0.1		Formic Acid	64-18-6	4.9E+04	n	4.2E+05	nm	3.1E-01	n	1.3E+00	n	1.4E+04	n		2.8E+00	
				3.0E+00	I					1	0.1		Fosetyl-AL	39148-24-8	1.8E+05	nm	1.8E+06	nm					4.7E+04	n			
													Furans														
				1.0E-03	X			V		1		1.7E+02	~Dibenzofuran	132-64-9	7.8E+01	n	1.0E+03	ns					5.8E+00	n		1.1E-01	
				1.0E-03	I			V		1		6.2E+03	~Furan	110-00-9	7.8E+01	n	1.0E+03	n					1.5E+01	n		5.7E-03	
				9.0E-01	I	2.0E+00	I	V		1	0.1	1.7E+05	~Tetrahydrofuran	109-99-9	1.8E+04	n	9.5E+04	n	2.1E+03	n	8.8E+03	n	3.2E+03	n		7.1E-01	
3.8E+00	H									1	0.1		Furazolidone	67-45-8	1.3E-01	c	4.5E-01	c					1.8E-02	c		3.4E-05	
				3.0E-03	I	5.0E-02	H			1	0.1		Furfural	98-01-1	1.8E+02	n	1.8E+03	n	5.2E+01	n	2.2E+02	n	4.6E+01	n		9.9E-03	
1.5E+00	C	4.3E-04	C							1	0.1		Furium	531-82-8	3.2E-01	c	1.1E+00	c	5.7E-03	c	2.9E-02	c	4.4E-02	c		5.9E-05	
3.0E-02	I	8.6E-06	C							1	0.1		Furmecyclox	60568-05-0	1.6E+01	c	5.7E+01	c	2.8E-01	c	1.4E+00	c	9.6E-01	c		1.0E-03	
				4.0E-04	I					1	0.1		Glufosinate, Ammonium	77182-82-2	2.4E+01	n	2.5E+02	n					6.3E+00	n		1.4E-03	
						8.0E-05	C			1	0.1		Glutaraldehyde	111-30-8	1.1E+05	nm	4.8E+05	nm	8.3E-02	n	3.5E-01	n					
				4.0E-04	I	1.0E-03	H			1	0.1		Glycidyl	765-34-4	2.4E+01	n	2.5E+02	n	1.0E+00	n	4.4E+00	n	6.3E+00	n		1.3E-03	
				1.0E-01	I					1	0.1		Glyphosate	1071-83-6	6.1E+03	n	6.2E+04	n					1.6E+03	n	7.0E+02	3.2E-01	1.4E-01
				3.0E-03	I					1	0.1		Goal	42874-03-3	1.8E+02	n	1.8E+03	n					2.4E+01	n		1.9E+00	
				3.0E-03	A	1.0E-02	A			1	0.1		Guthion	86-50-0	1.8E+02	n	1.8E+03	n	1.0E+01	n	4.4E+01	n	4.3E+01	n		1.3E-02	
				5.0E-05	I					1	0.1		Haloxyfop, Methyl	69806-40-2	3.1E+00	n	3.1E+01	n					5.8E-01	n		6.4E-03	
				1.3E-02	I					1	0.1		Harmony	79277-27-3	7.9E+02	n	8.0E+03	n					2.0E+02	n		6.1E-02	
4.5E+00	I	1.3E-03	I	5.0E-04	I					1	0.1		Heptachlor	76-44-8	1.1E-01	c	3.8E-01	c	1.9E-03	c	9.4E-03	c	1.8E-03	c	4.0E-01	1.4E-04	3.3E-02
9.1E+00	I	2.6E-03	I	1.3E-05	I					1	0.1		Heptachlor Epoxide	1024-57-3	5.3E-02	c*	1.9E-01	c*	9.4E-04	c	4.7E-03	c	3.3E-03	c*	2.0E-01	6.8E-05	4.1E-03
				2.0E-03	I					1	0.1		Hexabromobenzene	87-82-1	1.2E+02	n	1.2E+03	n					3.1E+01	n		1.8E-01	
				2.0E-04	I					1	0.1		Hexabromodiphenyl ether, 2,2',4,4',5,5'- (BDE-153)	68631-49-2	1.2E+01	n	1.2E+02	n					3.1E+00	n			
1.6E+00	I	4.6E-04	I	8.0E-04	I					1	0.1		Hexachlorobenzene	118-74-1	3.0E-01	c	1.1E+00	c	5.3E-03	c	2.7E-02	c	4.2E-02	c	1.0E+00	5.3E-04	1.3E-02
7.8E-02	I	2.2E-05	I	1.0E-03	P					1	0.1		Hexachlorobutadiene	87-68-3	6.2E+00	c**	2.2E+01	c*	1.1E-01	c	5.6E-01	c	2.6E-01	c*		5.0E-04	
6.3E+00	I	1.8E-03	I	8.0E-03	A					1	0.1		Hexachlorocyclohexane, Alpha-	319-84-6	7.7E-02	c	2.7E-01	c	1.4E-03	c	6.8E-03	c	6.2E-03	c		3.6E-05	
1.8E+00	I	5.3E-04	I							1	0.1		Hexachlorocyclohexane, Beta-	319-85-7	2.7E-01	c	9.6E-01	c	4.6E-03	c	2.3E-02	c	2.2E-02	c		1.3E-04	
1.1E+00	C	3.1E-04	C	3.0E-04	I					1	0.04		Hexachlorocyclohexane, Gamma- (Lindane)	58-89-9	5.2E-01	c*	2.1E+00	c	7.8E-03	c	4.0E-02	c	3.6E-02	c*	2.0E-01	2.1E-04	1.2E-03
1.8E+00	I	5.1E-04	I							1	0.1		Hexachlorocyclohexane, Technical	608-73-1	2.7E-01	c	9.6E-01	c	4.8E-03	c	2.4E-02	c	2.2E-02	c		1.3E-04	
				6.0E-03	I	2.0E-04	I			1	0.1		Hexachlorocyclopentadiene	77-47-4	3.7E+02	n	3.7E+03	n	2.1E-01	n	8.8E-01	n	2.2E+01	n	5.0E+01	7.0E-02	1.6E-01
4.0E-02	I	1.1E-05	C	7.0E-04	I	3.0E-02	I			1	0.1		Hexachloroethane	67-72-1	1.2E+01	c**	4.3E+01	c*	2.2E-01	c	1.1E+00	c	7.9E-01	c**		4.8E-04	
				3.0E-04	I					1	0.1		Hexachlorophene	70-30-4	1.8E+01	n	1.8E+02	n					4.7E+00	n		6.3E+00	
1.1E-01	I			3.0E-03	I					1	0.015		Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)	121-82-4	5.6E+00	c*	2.4E+01	c					6.1E-01	c*		2.3E-04	
						1.0E-05	I	V		1		5.2E+03	Hexamethylene Diisocyanate, 1,6-	822-06-0	3.4E+00	n	1.4E+01	n	1.0E-02	n	4.4E-02	n	2.1E-02	n		2.1E-04	
				4.0E-04	P					1	0.1		Hexamethylphosphoramide	680-31-9	2.4E+01	n	2.5E+02	n					6.2E+00	n		1.4E-03	
				6.0E-02	H	7.0E-01	I	V		1		1.4E+02	Hexane, N-	110-54-3	5.7E+02	ns	2.6E+03	ns	7.3E+02	n	3.1E+03	n	2.5E+02	n		1.8E+00	
				2.0E+00	P					1	0.1		Hexanedioic Acid	124-04-9	1.2E+05	nm	1.2E+06	nm					3.1E+04	n		7.7E+00	
				5.0E-03	I	3.0E-02	I	V		1		3.3E+03	Hexanone, 2-	591-78-6	2.1E+02	n	1.4E+03	n	3.1E+01	n	1.3E+02	n	3.4E+01	n		7.9E-03	
				3.3E-02	I					1	0.1		Hexazinone	51235-04-2	2.0E+03	n	2.0E+04	n					5.0E+02	n		2.3E-01	
3.0E+00	I	4.9E-03	I			3.0E-05	P			1			Hydrazine	302-01-2	2.1E-01	c	9.5E-01	c	5.0E-04	c*	2.5E-03	c*	2.2E-02	c			
3.0E+00	I	4.9E-03	I							1			Hydrazine Sulfate	10034-93-2	2.1E-01	c	9.5E-01	c	5.0E-04	c	2.5E-03	c	2.2E-02	c			
						2.0E-02	I			1			Hydrogen Chloride	7647-01-0	2.8E+07	nm	1.2E+08	nm	2.1E+01	n	8.8E+01	n					
				4.0E-02	C	1.4E-02	C			1			Hydrogen Fluoride	7664-39-3	3.1E+03	n	4.1E+04	n	1.5E+01	n	6.1E+01	n	6.2E+02	n			
						2.0E-03	I			1			Hydrogen Sulfide	7783-06-4	2.8E+06	nm	1.2E+07	nm	2.1E+00	n	8.8E+00	n					
6.0E-02	P			4.0E-02	P					1	0.1		Hydroquinone	123-31-9	8.1E+00	c	2.9E+01	c					1.1E+00	c		7.5E-04	
				1.3E-02	I					1	0.1		Imazalil	35554-44-0	7.9E+02	n	8.0E+03	n					1.4E+02	n		2.5E+00	
				2.5E-01	I					1	0.1		Imazaquin	81335-37-7	1.5E+04	n	1.5E+05	nm					3.8E+03	n		1.9E+01	
				1.0E-02	A					1			Iodine	7553-56-2	7.8E+02	n	1.0E+04	n					1.6E+02	n		9.4E+00	
				4.0E-02	I					1	0.1		Iprodione	36734-19-7	2.4E+03	n	2.5E+04	n					5.7E+02	n		1.7E-01	
				7.0E-01	P					1			Iron	7439-89-6	5.5E+04	n	7.2E+05	nm					1.1E+04	n		2.7E+02	
				3.0E-01	I					1	0.1		Isobutyl Alcohol	78-83-1	1.8E+04	n	1.8E+05	nm					4.6E+03	n		9.5E-01	
9.5E-04	I			2.0E-01	I	2.0E+00	C			1	0.1		Isophorone	78-59-1	5.1E+02	c*	1.8E+03	c*	2.1E+03	n	8.8E+03	n	6.7E+01	c*		2.2E-02	
				1.5E-02	I					1	0.1		Isopropalin	33820-53-0	9.2E+02	n	9.2E+03	n					2.3E+02	n		5.4E+00	
						7.0E+00	C			1	0.1		Isopropanol	67-63-0	9.9E+09	nm	4.2E+10	nm	7.3E+03	n	3.1E+04	n					
				1.0E-01	I					1	0.1		Isopropyl Methyl Phosphonic Acid	1832-54-8	6.1E+03	n	6.2E+04	n					1.6E+03	n		3.4E-01	
				5.0E-02	I					1	0.1		Isoxaben	82558-50-7	3.1E+03	n	3.1E+04	n					5.6E+02	n		1.5E+00	
						3.0E-01	A	V		1			JP-7	NA	4.3E+08	nm	1.8E+09	nm	3.1E+02	n	1.3E+03	n	6.3E+02	n			
				7.5E-02	I					1	0.1		Kerb	23950-58-5	4.6E+03	n	4.6E+04	n					9.0E+02	n		9.1E-01	
				2.0E-03	I					1	0.1		Lactofen	77501-63-4	1.2E+02	n	1.2E+03	n					1.9E+01	n		8.7E-01	
													Lead Compounds														
2.8E-01	C	8.0E-05	C							1	0.1		~Lead acetate	301-04-2	1.7E+00	c	6.2E+00	c	3.0E-02	c	1.5E-01	c	2.4E-01	c			
										1			~Lead and Compounds	7439-92-1	4.0E+02	L	8.0E+02	L	1.5E-01	L			L	L	1.5E+01		1.4E+01
3.8E-02	C	1.1E-05	C							1	0.1		~Lead subacetate	1335-32-6	1.3E+01	c	4.5E+01	c	2.2E-01	c	1.1E+00	c	1.8E+00	c			
				1.0E-07	I					1	0.1		~Tetraethyl Lead	78-00-2	6.1E-03	n	6.2E-02	n					9.9E-04	n		3.5E-06	
				2.0E-03	I					1	0.1		Linuron	330-55-2	1.2E+02	n	1.2E+03	n					2.6E+01	n		2.3E-02	
				2.0E-03	P					1			Lithium	7439-93-2	1.6E+02	n	2.0E+03	n					3.1E+01	n		9.3E+00	

Key: I = IRIS; P = PPRTV; A = ATSDR; C = Cal EPA; X = PPRTV Appendix; H = HEAST; J = New Jersey; Y = New York; O = EPA Office of Water; E = Environmental Criteria and Assessment Office; S = see user guide Section 5; L = see user guide on lead; M = mutagen; V = volatile; F = See FAQ; c = cancer; * = where: n SL < 100X c SL; ** = where n SL < 10X c SL; n = noncancer; m = Concentration may exceed ceiling limit (See User Guide); s = Concentration may exceed Csat (See User Guide); SSL values are based on DAF=1

Toxicity and Chemical-specific Information													Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	volc	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
				2.0E-01	I					1	0.1		Londax	83055-99-6	1.2E+04	n	1.2E+05	nm					3.1E+03	n		7.9E-01	
				5.0E-04	I					1	0.1		MCPA	94-74-6	3.1E+01	n	3.1E+02	n					5.7E+00	n		1.5E-03	
				1.0E-02	I					1	0.1		MCPB	94-81-5	6.1E+02	n	6.2E+03	n					1.6E+02	n		6.2E-02	
				1.0E-03	I					1	0.1		MCPP	93-65-2	6.1E+01	n	6.2E+02	n					1.2E+01	n		3.5E-03	
				2.0E-02	I					1	0.1		Malathion	121-75-5	1.2E+03	n	1.2E+04	n					3.0E+02	n		7.9E-02	
				1.0E-01	I	7.0E-04	C			1	0.1		Maleic Anhydride	108-31-6	6.1E+03	n	6.1E+04	n	7.3E-01	n	3.1E+00	n	1.5E+03	n		3.0E-01	
				5.0E-01	I					1	0.1		Maleic Hydrazide	123-33-1	3.1E+04	n	3.1E+05	nm					7.8E+03	n		1.6E+00	
				1.0E-04	P					1	0.1		Malononitrile	109-77-3	6.1E+00	n	6.2E+01	n					1.6E+00	n		3.2E-04	
				3.0E-02	H					1	0.1		Mancozeb	8018-01-7	1.8E+03	n	1.8E+04	n					4.7E+02	n		6.6E-01	
				5.0E-03	I					1	0.1		Maneb	12427-38-2	3.1E+02	n	3.1E+03	n					7.8E+01	n		1.1E-01	
				1.4E-01	I	5.0E-05	I			1			Manganese (Diet)	7439-96-5													
				2.4E-02	S	5.0E-05	I			0.04			Manganese (Non-diet)	7439-96-5	1.8E+03	n	2.3E+04	n	5.2E-02	n	2.2E-01	n	3.2E+02	n		2.1E+01	
				9.0E-05	H					1	0.1		Mephosfolan	950-10-7	5.5E+00	n	5.5E+01	n					1.4E+00	n		2.1E-03	
				3.0E-02	I					1	0.1		Mepiquat Chloride	24307-26-4	1.8E+03	n	1.8E+04	n					4.7E+02	n		1.6E-01	
													Mercury Compounds														
				3.0E-04	I	3.0E-05	C			0.07			~Mercuric Chloride (and other Mercury salts)	7487-94-7	2.3E+01	n	3.1E+02	n	3.1E-02	n	1.3E-01	n	4.3E+00	n	2.0E+00		
						3.0E-04	I	V		1		3.1E+00	~Mercury (elemental)	7439-97-6	1.0E+01	ns	4.3E+01	ns	3.1E-01	n	1.3E+00	n	6.3E-01	n	2.0E+00	3.3E-02	1.0E-01
				1.0E-04	I					1			~Methyl Mercury	22967-92-6	7.8E+00	n	1.0E+02	n					1.6E+00	n			
				8.0E-05	I					1	0.1		~Phenylmercuric Acetate	62-38-4	4.9E+00	n	4.9E+01	n					1.2E+00	n		3.9E-04	
				3.0E-05	I					1	0.1		Merphos	150-50-5	1.8E+00	n	1.8E+01	n					4.7E-01	n		4.6E-02	
				3.0E-05	I					1	0.1		Merphos Oxide	78-48-8	1.8E+00	n	1.8E+01	n					6.1E-02	n		3.0E-04	
				6.0E-02	I					1	0.1		Metalaxyl	57837-19-1	3.7E+03	n	3.7E+04	n					9.2E+02	n		2.5E-01	
				1.0E-04	I	7.0E-04	H	V		1		4.6E+03	Methacrylonitrile	126-98-7	3.2E+00	n	1.8E+01	n	7.3E-01	n	3.1E+00	n	7.5E-01	n		1.7E-04	
				5.0E-05	I					1	0.1		Methamidophos	10265-92-6	3.1E+00	n	3.1E+01	n					7.8E-01	n		1.6E-04	
				5.0E-01	I	4.0E+00	C			1	0.1		Methanol	67-56-1	3.1E+04	n	3.1E+05	nm	4.2E+03	n	1.8E+04	n	7.8E+03	n		1.6E+00	
				1.0E-03	I					1	0.1		Methidathion	950-37-8	6.1E+01	n	6.2E+02	n					1.5E+01	n		3.7E-03	
				2.5E-02	I					1	0.1		Methomyl	16752-77-5	1.5E+03	n	1.5E+04	n					3.9E+02	n		8.5E-02	
4.9E-02	C	1.4E-05	C							1	0.1		Methoxy-5-nitroaniline, 2-	99-59-2	9.9E+00	c	3.5E+01	c	1.7E-01	c	8.8E-01	c	1.3E+00	c		4.6E-04	
				5.0E-03	I					1	0.1		Methoxychlor	72-43-5	3.1E+02	n	3.1E+03	n					2.7E+01	n	4.0E+01	1.5E+00	2.2E+00
				8.0E-03	P	1.0E-03	P			1	0.1		Methoxyethanol Acetate, 2-	110-49-6	4.9E+02	n	4.9E+03	n	1.0E+00	n	4.4E+00	n	1.3E+02	n		2.6E-02	
				5.0E-03	P	2.0E-02	I			1	0.1		Methoxyethanol, 2-	109-86-4	3.1E+02	n	3.1E+03	n	2.1E+01	n	8.8E+01	n	7.8E+01	n		1.6E-02	
				1.0E+00	X			V		1		2.9E+04	Methyl Acetate	79-20-9	7.8E+04	ns	1.0E+06	nms					1.6E+04	n		3.2E+00	
				3.0E-02	H			V		1		6.8E+03	Methyl Acrylate	96-33-3	2.3E+03	n	3.1E+04	ns					4.6E+02	n		9.8E-02	
				6.0E-01	I	5.0E+00	I	V		1		2.8E+04	Methyl Ethyl Ketone (2-Butanone)	78-93-3	2.8E+04	n	2.0E+05	nms	5.2E+03	n	2.2E+04	n	4.9E+03	n		1.0E+00	
		1.0E-03	X	1.0E-03	P	2.0E-05	X			1	0.1		Methyl Hydrazine	60-34-4	6.1E+01	n	6.1E+02	n	2.4E-03	c**	1.2E-02	c**	1.6E+01	n		3.5E-03	
				8.0E-02	H	3.0E+00	I	V		1		3.4E+03	Methyl Isobutyl Ketone (4-methyl-2-pentanone)	108-10-1	5.3E+03	ns	5.3E+04	ns	3.1E+03	n	1.3E+04	n	1.0E+03	n		2.3E-01	
						1.0E-03	C	V		1	0.1	1.7E+04	Methyl Isocyanate	624-83-9	5.0E+00	n	2.1E+01	n	1.0E+00	n	4.4E+00	n	2.1E+00	n		5.9E-04	
				1.4E+00	I	7.0E-01	I	V		1		2.4E+03	Methyl Methacrylate	80-62-6	4.8E+03	ns	2.1E+04	ns	7.3E+02	n	3.1E+03	n	1.4E+03	n		3.0E-01	
				2.5E-04	I					1	0.1		Methyl Parathion	298-00-0	1.5E+01	n	1.5E+02	n					3.4E+00	n		5.7E-03	
				6.0E-02	X					1	0.1		Methyl Phosphonic Acid	993-13-5	3.7E+03	n	3.7E+04	n					9.4E+02	n		1.9E-01	
				6.0E-03	H	4.0E-02	H	V		1		3.8E+02	Methyl Styrene (Mixed Isomers)	25013-15-4	2.5E+02	n	1.6E+03	ns	4.2E+01	n	1.8E+02	n	3.1E+01	n		5.0E-02	
9.9E-02	C	2.8E-05	C							1	0.1		Methyl methanesulfonate	66-27-3	4.9E+00	c	1.7E+01	c	8.7E-02	c	4.4E-01	c	6.8E-01	c		1.4E-04	
1.8E-03	C	2.6E-07	C			3.0E+00	I	V		1		8.9E+03	Methyl tert-Butyl Ether (MTBE)	1634-04-4	4.3E+01	c	2.2E+02	c	9.4E+00	c	4.7E+01	c	1.2E+01	c		2.8E-03	
				2.0E-04	X					1	0.1		Methyl-1,4-benzenediamine dihydrochloride, 2-	615-45-2	1.2E+01	n	1.2E+02	n					3.1E+00	n		1.9E-03	
9.0E-03	P			2.0E-02	X					1	0.1		Methyl-5-Nitroaniline, 2-	99-55-8	5.4E+01	c*	1.9E+02	c*					7.0E+00	c*		3.9E-03	
8.3E+00	C	2.4E-03	C							1	0.1		Methyl-N-nitro-N-nitrosoguanidine, N-	70-25-7	5.9E-02	c	2.1E-01	c	1.0E-03	c	5.1E-03	c	8.1E-03	c		2.8E-06	
1.3E-01	C	3.7E-05	C							1	0.1		Methylaniline Hydrochloride, 2-	636-21-5	3.7E+00	c	1.3E+01	c	6.6E-02	c	3.3E-01	c	5.0E-01	c		2.1E-04	
				1.0E-02	A					1	0.1		Methylarsonic acid	124-58-3	6.1E+02	n	6.2E+03	n					1.6E+02	n			
				2.0E-04	X					1	0.1		Methylbenzene,1-4-diamine monohydrochloride, 2-	74612-12-7	1.2E+01	n	1.2E+02	n					3.1E+00	n			
				2.0E-04	X					1	0.1		Methylbenzene-1,4-diamine sulfate, 2-	615-50-9	1.2E+01	n	1.2E+02	n					3.1E+00	n			
2.2E+01	C	6.3E-03	C						M	1	0.1		Methylcholanthrene, 3-	56-49-5	5.2E-03	c	7.8E-02	c	1.5E-04	c	1.9E-03	c	9.8E-04	c		1.9E-03	
2.0E-03	I	1.0E-08	I	6.0E-03	I	6.0E-01	I	V	M	1		3.3E+03	Methylene Chloride	75-09-2	5.6E+01	c**	9.6E+02	c**	9.6E+01	c**	1.2E+03	c**	9.9E+00	c**	5.0E+00	2.5E-03	1.3E-03
1.0E-01	P	4.3E-04	C	2.0E-03	P				M	1	0.1		Methylene-bis(2-chloroaniline), 4,4'-	101-14-4	1.2E+00	c	1.7E+01	c*	2.2E-03	c	2.9E-02	c	1.4E-01	c		1.6E-03	
4.6E-02	I	1.3E-05	C							1	0.1		Methylene-bis(N,N-dimethyl) Aniline, 4,4'-	101-61-1	1.1E+01	c	3.7E+01	c	1.9E-01	c	9.4E-01	c	6.0E-01	c		3.3E-03	
1.6E+00	C	4.6E-04	C			2.0E-02	C			1	0.1		Methylenebisbenzenamine, 4,4'-	101-77-9	3.0E-01	c	1.1E+00	c	5.3E-03	c	2.7E-02	c	4.1E-02	c		1.8E-04	
						6.0E-04	I			1	0.1		Methylenediphenyl Diisocyanate	101-68-8	8.5E+05	nm	3.6E+06	nm	6.3E-01	n	2.6E+00	n					
				7.0E-02	H			V		1		5.0E+02	Methylstyrene, Alpha-	98-83-9	5.5E+03	ns	7.2E+04	ns					5.8E+02	n		9.3E-01	
				1.5E-01	I					1	0.1		Metolachlor	51218-45-2	9.2E+03	n	9.2E+04	n					2.1E+03	n		2.5E+00	
				2.5E-02	I					1	0.1		Metribuzin	21087-64-9	1.5E+03	n	1.5E+04	n					3.8E+02	n		1.2E-01	
				3.0E+00	P			V		1	0.1	3.4E-01	Mineral oils	8012-95-1	1.8E+05	nms	1.8E+06	nms					4.7E+04	n		1.9E+03	
1.8E+01	C	5.1E-03	C	2.0E-04	I					1	0.1		Mirex	2385-85-5	2.7E-02	c	9.6E-02	c	4.8E-04	c	2.4E-03	c	3.7E-03	c		2.7E-03	
				2.0E-03	I					1	0.1		Molinate	2212-67-1	1.2E+02	n	1.2E+03	n					2.3E+01	n		1.3E-02	
				5.0E-03	I					1			Molybdenum	7439-98-7	3.9E+02	n	5.1E+03	n					7.8E+01	n		1.6E+00	
				1.0E-01	I					1			Monochloramine	10599-90-3	7.8E+03	n	1.0E+05	nm					1.6E+03	n			
				2.0E-03	P					1	0.1		Monomethylaniline	100-61-8	1.2E+02	n	1.2E+03	n					3.0E+01	n		1.1E-02	

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Toxicity and Chemical-specific Information													Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	vo c	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
				3.0E-04	X					1	0.1		N,N'-Diphenyl-1,4-benzenediamine	74-31-7	1.8E+01	n	1.8E+02	n					2.7E+00	n		2.8E-01	
				2.0E-03	I					1	0.1		Naled	300-76-5	1.2E+02	n	1.2E+03	n					3.1E+01	n		1.4E-02	
				3.0E-02	X	1.0E-01	P	V		1			Naphtha, High Flash Aromatic (HFAN)	64724-95-6	2.3E+03	n	3.1E+04	n	1.0E+02	n	4.4E+02	n	1.4E+02	n			
1.8E+00	C	0.0E+00	C							1	0.1		Naphthylamine, 2-	91-59-8	2.7E-01	c	9.6E-01	c					3.3E-02	c		1.7E-04	
				1.0E-01	I					1	0.1		Napropamide	15299-99-7	6.1E+03	n	6.2E+04	n					1.3E+03	n		8.3E+00	
				5.0E-02	C	5.0E-05	C			0.04			Nickel Carbonyl	13463-39-3	3.7E+03	n	4.4E+04	n	5.2E-02	n	2.2E-01	n	6.7E+02	n			
				5.0E-02	C	1.0E-04	C			1			Nickel Oxide	1313-99-1	3.8E+03	n	4.7E+04	n	1.0E-01	n	4.4E-01	n	7.8E+02	n			
		2.4E-04	I	5.0E-02	C	5.0E-05	C			0.04			Nickel Refinery Dust	NA	3.7E+03	n	4.4E+04	n	1.0E-02	c**	5.1E-02	c**	7.6E+02	n		1.1E+02	
		2.6E-04	C	2.0E-02	I	9.0E-05	A			0.04			Nickel Soluble Salts	7440-02-0	1.5E+03	n	2.0E+04	n	9.4E-03	c*	4.7E-02	c**	3.0E+02	n		2.0E+01	
1.7E+00	C	4.8E-04	I	5.0E-02	C	5.0E-05	C			0.04			Nickel Subsulfide	12035-72-2	3.8E-01	c	1.7E+00	c	5.1E-03	c*	2.6E-02	c**	3.9E-02	c			
				1.6E+00	I					1			Nitrate	14797-55-8	1.3E+05	nm	1.6E+06	nm					2.5E+04	n	1.0E+04		
				1.0E-01	I					1			Nitrite	14797-65-0	7.8E+03	n	1.0E+05	nm					1.6E+03	n	1.0E+03		
				1.0E-02	X	5.0E-05	X			1	0.1		Nitroaniline, 2-	88-74-4	6.1E+02	n	6.0E+03	n	5.2E-02	n	2.2E-01	n	1.5E+02	n		6.2E-02	
2.0E-02	P			4.0E-03	P	6.0E-03	P			1	0.1		Nitroaniline, 4-	100-01-6	2.4E+01	c*	8.6E+01	c*	6.3E+00	n	2.6E+01	n	3.3E+00	c*		1.4E-03	
		4.0E-05	I	2.0E-03	I	9.0E-03	I	V		1		3.1E+03	Nitrobenzene	98-95-3	4.8E+00	c*	2.4E+01	c*	6.1E-02	c	3.1E-01	c	1.2E-01	c*		7.9E-05	
				3.0E+03	P					1	0.1		Nitrocellulose	9004-70-0	1.8E+08	nm	1.8E+09	nm					4.7E+07	n		1.0E+04	
				7.0E-02	H					1	0.1		Nitrofurantoin	67-20-9	4.3E+03	n	4.3E+04	n					1.1E+03	n		4.7E-01	
1.3E+00	C	3.7E-04	C							1	0.1		Nitrofurazone	59-87-0	3.7E-01	c	1.3E+00	c	6.6E-03	c	3.3E-02	c	5.2E-02	c		4.6E-05	
1.7E-02	P			1.0E-04	P					1	0.1		Nitroglycerin	55-63-0	6.1E+00	n	6.2E+01	n					1.5E+00	n		6.6E-04	
				1.0E-01	I					1	0.1		Nitroguanidine	556-88-7	6.1E+03	n	6.2E+04	n					1.6E+03	n		3.8E-01	
		9.0E-06	P			2.0E-02	P	V		1		1.8E+04	Nitromethane	75-52-5	4.9E+00	c*	2.5E+01	c*	2.7E-01	c*	1.4E+00	c*	5.4E-01	c*		1.2E-04	
		2.7E-03	H			2.0E-02	I	V		1		4.9E+03	Nitropropane, 2-	79-46-9	1.3E-02	c	6.4E-02	c	9.0E-04	c	4.5E-03	c	1.8E-03	c		4.7E-07	
2.7E+01	C	7.7E-03	C						M	1	0.1		Nitroso-N-ethylurea, N-	759-73-9	4.3E-03	c	6.4E-02	c	1.2E-04	c	1.6E-03	c	7.9E-04	c		1.9E-07	
1.2E+02	C	3.4E-02	C						M	1	0.1		Nitroso-N-methylurea, N-	684-93-5	9.6E-04	c	1.4E-02	c	2.8E-05	c	3.6E-04	c	1.8E-04	c		4.0E-08	
5.4E+00	I	1.6E-03	I					V		1		7.1E+03	Nitroso-di-N-butylamine, N-	924-16-3	8.7E-02	c	4.0E-01	c	1.5E-03	c	7.7E-03	c	2.4E-03	c		4.8E-06	
7.0E+00	I	2.0E-03	C							1	0.1		Nitroso-di-N-propylamine, N-	621-64-7	6.9E-02	c	2.5E-01	c	1.2E-03	c	6.1E-03	c	9.3E-03	c		7.0E-06	
2.8E+00	I	8.0E-04	C							1	0.1		Nitrosodiethanolamine, N-	1116-54-7	1.7E-01	c	6.2E-01	c	3.0E-03	c	1.5E-02	c	2.4E-02	c		4.8E-06	
1.5E+02	I	4.3E-02	I						M	1	0.1		Nitrosodiethylamine, N-	55-18-5	7.7E-04	c	1.1E-02	c	2.2E-05	c	2.9E-04	c	1.4E-04	c		5.2E-08	
5.1E+01	I	1.4E-02	I	8.0E-06	P	4.0E-05	X		M	1	0.1		Nitrosodimethylamine, N-	62-75-9	2.3E-03	c	3.4E-02	c	6.9E-05	c	8.8E-04	c	4.2E-04	c		1.0E-07	
4.9E-03	I	2.6E-06	C							1	0.1		Nitrosodiphenylamine, N-	86-30-6	9.9E+01	c	3.5E+02	c	9.4E-01	c	4.7E+00	c	1.0E+01	c		5.7E-02	
2.2E+01	I	6.3E-03	C							1	0.1		Nitrosomethylethylamine, N-	10595-95-6	2.2E-02	c	7.8E-02	c	3.9E-04	c	1.9E-03	c	3.0E-03	c		8.7E-07	
6.7E+00	C	1.9E-03	C							1	0.1		Nitrosomorpholine [N-]	59-89-2	7.3E-02	c	2.6E-01	c	1.3E-03	c	6.5E-03	c	1.0E-02	c		2.5E-06	
9.4E+00	C	2.7E-03	C							1	0.1		Nitrosopiperidine [N-]	100-75-4	5.2E-02	c	1.8E-01	c	9.0E-04	c	4.5E-03	c	7.1E-03	c		3.8E-06	
2.1E+00	I	6.1E-04	I							1	0.1		Nitrosopyrrolidine, N-	930-55-2	2.3E-01	c	8.2E-01	c	4.0E-03	c	2.0E-02	c	3.2E-02	c		1.2E-05	
				1.0E-04	X					1	0.1		Nitrotoluene, m-	99-08-1	6.1E+00	n	6.2E+01	n					1.3E+00	n		1.2E-03	
2.2E-01	P			9.0E-04	P			V		1		1.5E+03	Nitrotoluene, o-	88-72-2	2.9E+00	c*	1.3E+01	c*					2.7E-01	c*		2.5E-04	
1.6E-02	P			4.0E-03	P					1	0.1		Nitrotoluene, p-	99-99-0	3.0E+01	c**	1.1E+02	c*					3.7E+00	c*		3.4E-03	
				3.0E-04	X	2.0E-01	P	V		1		6.9E+00	Nonane, n-	111-84-2	2.1E+01	ns	2.3E+02	ns	2.1E+02	n	8.8E+02	n	4.6E+00	n		6.6E-02	
				4.0E-02	I					1	0.1		Norflurazon	27314-13-2	2.4E+03	n	2.5E+04	n					6.0E+02	n		3.9E+00	
				7.0E-04	I					1	0.1		Nustar	85509-19-9	4.3E+01	n	4.3E+02	n					8.3E+00	n		1.4E+00	
				3.0E-03	I					1	0.1		Octabromodiphenyl Ether	32536-52-0	1.8E+02	n	1.8E+03	n					4.7E+01	n		9.3E+00	
				5.0E-02	I					1	0.006		Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetra (HMX)	2691-41-0	3.8E+03	n	4.9E+04	n					7.8E+02	n		9.9E-01	
				2.0E-03	H					1	0.1		Octamethylpyrophosphoramide	152-16-9	1.2E+02	n	1.2E+03	n					3.1E+01	n		7.5E-03	
				5.0E-02	I					1	0.1		Oryzalin	19044-88-3	3.1E+03	n	3.1E+04	n					6.2E+02	n		1.1E+00	
				5.0E-03	I					1	0.1		Oxadiazon	19666-30-9	3.1E+02	n	3.1E+03	n					3.5E+01	n		3.6E-01	
				2.5E-02	I					1	0.1		Oxamyl	23135-22-0	1.5E+03	n	1.5E+04	n					3.9E+02	n	2.0E+02	8.6E-02	4.4E-02
				1.3E-02	I					1	0.1		Paclobutrazol	76738-62-0	7.9E+02	n	8.0E+03	n					1.7E+02	n		3.6E-01	
				4.5E-03	I					1	0.1		Paraquat Dichloride	1910-42-5	2.7E+02	n	2.8E+03	n					7.0E+01	n		9.7E-01	
				6.0E-03	H					1	0.1		Parathion	56-38-2	3.7E+02	n	3.7E+03	n					6.5E+01	n		3.3E-01	
				5.0E-02	H					1	0.1		Pebulate	1114-71-2	3.1E+03	n	3.1E+04	n					4.2E+02	n		3.3E-01	
				4.0E-02	I					1	0.1		Pendimethalin	40487-42-1	2.4E+03	n	2.5E+04	n					1.3E+02	n		1.5E+00	
				2.0E-03	I					1	0.1		Pentabromodiphenyl Ether	32534-81-9	1.2E+02	n	1.2E+03	n					3.1E+01	n		1.4E+00	
				1.0E-04	I					1	0.1		Pentabromodiphenyl ether, 2,2',4,4',5- (BDE-99)	60348-60-9	6.1E+00	n	6.2E+01	n					1.6E+00	n		6.8E-02	
				8.0E-04	I					1	0.1		Pentachlorobenzene	608-93-5	4.9E+01	n	4.9E+02	n					2.3E+00	n		1.7E-02	
9.0E-02	P									1	0.1		Pentachloroethane	76-01-7	5.4E+00	c	1.9E+01	c					5.6E-01	c		2.7E-04	
2.6E-01	H			3.0E-03	I					1	0.1		Pentachloronitrobenzene	82-68-8	1.9E+00	c*	6.6E+00	c					1.0E-01	c		1.3E-03	
4.0E-01	I	5.1E-06	C	5.0E-03	I					1	0.25		Pentachlorophenol	87-86-5	8.9E-01	c	2.7E+00	c	4.8E-01	c	2.4E+00	c	1.7E-01	c	1.0E+00	1.7E-03	1.0E-02
4.0E-03	X			2.0E-03	P					1	0.1		Pentaerythritol tetranitrate (PETN)	78-11-5	1.2E+02	c**	4.3E+02	c**					1.6E+01	c**		2.4E-02	
						1.0E+00	P	V		1		3.9E+02	Pentane, n-	109-66-0	8.7E+02	ns	3.7E+03	ns	1.0E+03	n	4.4E+03	n	2.1E+03	n		1.0E+01	
													Perchlorates														
				7.0E-04	I					1			~Ammonium Perchlorate	7790-98-9	5.5E+01	n	7.2E+02	n					1.1E+01	n			
				7.0E-04	I					1			~Lithium Perchlorate	7791-03-9	5.5E+01	n	7.2E+02	n					1.1E+01	n			
				7.0E-04	I					1			~Perchlorate and Perchlorate Salts	14797-73-0	5.5E+01	n	7.2E+02	n					1.1E+01	n	1.5E+01(F)		
				7.0E-04	I					1			~Potassium Perchlorate	7778-74-7	5.5E+01	n	7.2E+02	n					1.1E+01	n			
				7.0E-04	I					1			~Sodium Perchlorate	7601-89-0	5.5E+01	n	7.2E+02	n					1.1E+01	n			

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Toxicity and Chemical-specific Information													Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	voc	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
				5.0E-02	I					1	0.1		Permethrin	52645-53-1	3.1E+03	n	3.1E+04	n					7.8E+02	n		1.9E+02	
2.2E-03	C	6.3E-07	C							1	0.1		Phenacetin	62-44-2	2.2E+02	c	7.8E+02	c	3.9E+00	c	1.9E+01	c	3.0E+01	c		8.3E-03	
				2.5E-01	I					1	0.1		Phenmedipham	13684-63-4	1.5E+04	n	1.5E+05	nm					3.0E+03	n		1.6E+01	
				3.0E-01	I	2.0E-01	C			1	0.1		Phenol	108-95-2	1.8E+04	n	1.8E+05	nm	2.1E+02	n	8.8E+02	n	4.5E+03	n		2.6E+00	
				5.0E-04	X					1	0.1		Phenothiazine	92-84-2	3.1E+01	n	3.1E+02	n					3.2E+00	n		1.0E-02	
				6.0E-03	I					1	0.1		Phenylenediamine, m-	108-45-2	3.7E+02	n	3.7E+03	n					9.4E+01	n		2.5E-02	
4.7E-02	H									1	0.1		Phenylenediamine, o-	95-54-5	1.0E+01	c	3.7E+01	c					1.4E+00	c		3.8E-04	
				1.9E-01	H					1	0.1		Phenylenediamine, p-	106-50-3	1.2E+04	n	1.2E+05	nm					3.0E+03	n		7.9E-01	
1.9E-03	H									1	0.1		Phenylphenol, 2-	90-43-7	2.5E+02	c	8.9E+02	c					2.6E+01	c		3.5E-01	
				2.0E-04	H					1	0.1		Phorate	298-02-2	1.2E+01	n	1.2E+02	n					2.3E+00	n		2.6E-03	
						3.0E-04	I	V		1		1.6E+03	Phosgene	75-44-5	3.3E-01	n	1.4E+00	n	3.1E-01	n	1.3E+00	n					
				2.0E-02	I					1	0.1		Phosmet	732-11-6	1.2E+03	n	1.2E+04	n					2.9E+02	n		6.4E-02	
													Phosphates, Inorganic														
				4.9E+01	P					1			~Aluminum metaphosphate	13776-88-0	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Ammonium polyphosphate	68333-79-9	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Calcium pyrophosphate	7790-76-3	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Diammonium phosphate	7783-28-0	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Dicalcium phosphate	7757-93-9	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Dimagnesium phosphate	7782-75-4	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Dipotassium phosphate	7758-11-4	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Disodium phosphate	7558-79-4	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Monoaluminum phosphate	13530-50-2	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Monoammonium phosphate	7722-76-1	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Monocalcium phosphate	7758-23-8	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Monomagnesium phosphate	7757-86-0	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Monopotassium phosphate	7778-77-0	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Monosodium phosphate	7558-80-7	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Polyphosphoric acid	8017-16-1	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Potassium tripolyphosphate	13845-36-8	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Sodium acid pyrophosphate	7758-16-9	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Sodium aluminum phosphate (acidic)	7785-88-8	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Sodium aluminum phosphate (anhydrous)	10279-59-1	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Sodium aluminum phosphate (tetrahydrate)	10305-76-7	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Sodium hexametaphosphate	10124-56-8	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Sodium polyphosphate	68915-31-1	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Sodium trimetaphosphate	7785-84-4	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Sodium tripolyphosphate	7758-29-4	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Tetrapotassium phosphate	7320-34-5	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Tetrasodium pyrophosphate	7722-88-5	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Trialuminum sodium tetra decahydrogenoctaorthophosphate (dihydrate)	15136-87-5	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Tricalcium phosphate	7758-87-4	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Trimagnesium phosphate	7757-87-1	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Tripotassium phosphate	7778-53-2	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				4.9E+01	P					1			~Trisodium phosphate	7601-54-9	3.8E+06	nm	5.0E+07	nm					7.6E+05	n			
				3.0E-04	I	3.0E-04	I			1			Phosphine	7803-51-2	2.3E+01	n	3.1E+02	n	3.1E-01	n	1.3E+00	n	4.7E+00	n			
				4.9E+01	P	1.0E-02	I			1			Phosphoric Acid	7664-38-2	3.0E+06	nm	2.7E+07	nm	1.0E+01	n	4.4E+01	n	7.6E+05	n			
				2.0E-05	I					1			Phosphorus, White	7723-14-0	1.6E+00	n	2.0E+01	n					3.1E-01	n		1.1E-03	
				1.0E+00	H					1	0.1		Phthalic Acid, P-	100-21-0	6.1E+04	n	6.2E+05	nm					1.5E+04	n		5.3E+00	
				2.0E+00	I	2.0E-02	C			1	0.1		Phthalic Anhydride	85-44-9	1.2E+05	nm	1.2E+06	nm	2.1E+01	n	8.8E+01	n	3.0E+04	n		6.6E+00	
				7.0E-02	I					1	0.1		Picloram	1918-02-1	4.3E+03	n	4.3E+04	n					1.1E+03	n	5.0E+02	2.9E-01	1.4E-01
				1.0E-04	X					1	0.1		Picramic Acid (2-Amino-4,6-dinitrophenol)	96-91-3	6.1E+00	n	6.2E+01	n					1.5E+00	n		1.0E-03	
				1.0E-02	I					1	0.1		Pirimiphos, Methyl	29232-93-7	6.1E+02	n	6.2E+03	n					9.1E+01	n		8.7E-02	
3.0E+01	C	8.6E-03	C	7.0E-06	H					1	0.1		Polybrominated Biphenyls	59536-65-1	1.6E-02	c*	5.7E-02	c*	2.8E-04	c	1.4E-03	c	2.2E-03	c*			
													Polychlorinated Biphenyls (PCBs)														
7.0E-02	S	2.0E-05	S	7.0E-05	I					1	0.14		~Aroclor 1016	12674-11-2	3.9E+00	n	2.1E+01	c**	1.2E-01	c	6.1E-01	c	9.6E-01	c**		9.2E-02	
2.0E+00	S	5.7E-04	S					V		1	0.14	7.6E+02	~Aroclor 1221	11104-28-2	1.4E-01	c	5.4E-01	c	4.3E-03	c	2.1E-02	c	4.3E-03	c		7.4E-05	
2.0E+00	S	5.7E-04	S					V		1	0.14	7.3E+01	~Aroclor 1232	11141-16-5	1.4E-01	c	5.4E-01	c	4.3E-03	c	2.1E-02	c	4.3E-03	c		7.4E-05	
2.0E+00	S	5.7E-04	S							1	0.14		~Aroclor 1242	53469-21-9	2.2E-01	c	7.4E-01	c	4.3E-03	c	2.1E-02	c	3.4E-02	c		5.3E-03	
2.0E+00	S	5.7E-04	S							1	0.14		~Aroclor 1248	12672-29-6	2.2E-01	c	7.4E-01	c	4.3E-03	c	2.1E-02	c	3.4E-02	c		5.2E-03	
2.0E+00	S	5.7E-04	S	2.0E-05	I					1	0.14		~Aroclor 1254	11097-69-1	2.2E-01	c**	7.4E-01	c*	4.3E-03	c	2.1E-02	c	3.4E-02	c**		8.8E-03	
2.0E+00	S	5.7E-04	S							1	0.14		~Aroclor 1260	11096-82-5	2.2E-01	c	7.4E-01	c	4.3E-03	c	2.1E-02	c	3.4E-02	c		2.4E-02	
3.9E+00	E	1.1E-03	E	3.3E-05	E	1.3E-03	E			1	0.14		~Heptachlorobiphenyl, 2,3,3',4,4',5,5'- (PCB 189)	39635-31-9	1.1E-01	c*	3.8E-01	c*	2.1E-03	c	1.1E-02	c	1.7E-02	c*		1.2E-02	
3.9E+00	E	1.1E-03	E	3.3E-05	E	1.3E-03	E			1	0.14		~Hexachlorobiphenyl, 2,3',4,4',5,5'- (PCB 167)	52663-72-6	1.1E-01	c*	3.8E-01	c*	2.1E-03	c	1.1E-02	c	1.7E-02	c*		7.2E-03	
3.9E+00	E	1.1E-03	E	3.3E-05	E	1.3E-03	E			1	0.14		~Hexachlorobiphenyl, 2,3,3',4,4',5'- (PCB 157)	69782-90-7	1.1E-01	c*	3.8E-01	c*	2.1E-03	c	1.1E-02	c	1.7E-02	c*		7.4E-03	
3.9E+00	E	1.1E-03	E	3.3E-05	E	1.3E-03	E			1	0.14		~Hexachlorobiphenyl, 2,3,3',4,4',5- (PCB 156)	38380-08-4	1.1E-01	c*	3.8E-01	c*	2.1E-03	c	1.1E-02	c	1.7E-02	c*		7.4E-03	

Key: I = IRIS; P = PPRTV; A = ATSDR; C = Cal EPA; X = PPRTV Appendix; H = HEAST; J = New Jersey; Y = New York; O = EPA Office of Water; E = Environmental Criteria and Assessment Office; S = see user guide Section 5; L = see user guide on lead; M = mutagen; V = volatile; F = See FAQ; c = cancer; * = where: n SL < 100X c SL; ** = where n SL < 10X c SL; n = noncancer; m = Concentration may exceed ceiling limit (See User Guide); s = Concentration may exceed Csat (See User Guide); SSL values are based on DAF=1

Toxicity and Chemical-specific Information												Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	voc	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
3.9E+03	E	1.1E+00	E	3.3E-08	E	1.3E-06	E			1	0.14		~Hexachlorobiphenyl, 3,3',4,4',5,5'- (PCB 169)	32774-16-6	1.1E-04	c*	3.8E-04	c*	2.1E-06	c	1.1E-05	c	1.7E-05	c*		7.2E-06	
3.9E+00	E	1.1E-03	E	3.3E-05	E	1.3E-03	E			1	0.14		~Pentachlorobiphenyl, 2',3,4,4',5- (PCB 123)	65510-44-3	1.1E-01	c*	3.8E-01	c*	2.1E-03	c	1.1E-02	c	1.7E-02	c*		4.5E-03	
3.9E+00	E	1.1E-03	E	3.3E-05	E	1.3E-03	E			1	0.14		~Pentachlorobiphenyl, 2,3',4,4',5- (PCB 118)	31508-00-6	1.1E-01	c*	3.8E-01	c*	2.1E-03	c	1.1E-02	c	1.7E-02	c*		4.4E-03	
3.9E+00	E	1.1E-03	E	3.3E-05	E	1.3E-03	E			1	0.14		~Pentachlorobiphenyl, 2,3,3',4,4'- (PCB 105)	32598-14-4	1.1E-01	c*	3.8E-01	c*	2.1E-03	c	1.1E-02	c	1.7E-02	c*		4.5E-03	
3.9E+00	E	1.1E-03	E	3.3E-05	E	1.3E-03	E			1	0.14		~Pentachlorobiphenyl, 2,3,4,4',5- (PCB 114)	74472-37-0	1.1E-01	c*	3.8E-01	c*	2.1E-03	c	1.1E-02	c	1.7E-02	c*		4.5E-03	
1.3E+04	E	3.8E+00	E	1.0E-08	E	4.0E-07	E			1	0.14		~Pentachlorobiphenyl, 3,3',4,4',5- (PCB 126)	57465-28-8	3.4E-05	c*	1.1E-04	c*	6.4E-07	c	3.2E-06	c	5.2E-06	c*		1.3E-06	
2.0E+00	I	5.7E-04	I							1	0.14		~Polychlorinated Biphenyls (high risk)	1336-36-3	2.2E-01	c	7.4E-01	c	4.3E-03	c	2.1E-02	c					
4.0E-01	I	1.0E-04	I							1	0.14		~Polychlorinated Biphenyls (low risk)	1336-36-3					2.4E-02	c	1.2E-01	c	1.7E-01	c	5.0E-01	2.6E-02	7.8E-02
7.0E-02	I	2.0E-05	I							1	0.14		~Polychlorinated Biphenyls (lowest risk)	1336-36-3					1.2E-01	c	6.1E-01	c					
1.3E+01	E	3.8E-03	E	1.0E-05	E	4.0E-04	E			1	0.14		~Tetrachlorobiphenyl, 3,3',4,4'- (PCB 77)	32598-13-3	3.4E-02	c*	1.1E-01	c*	6.4E-04	c	3.2E-03	c	5.2E-03	c*		8.1E-04	
3.9E+01	E	1.1E-02	E	3.3E-06	E	1.3E-04	E			1	0.14		~Tetrachlorobiphenyl, 3,4,4',5- (PCB 81)	70362-50-4	1.1E-02	c*	3.8E-02	c*	2.1E-04	c	1.1E-03	c	1.7E-03	c*		2.7E-04	
						6.0E-04	I			1	0.1		Polymeric Methylene Diphenyl Diisocyanate (PMDI)	9016-87-9	8.5E+05	nm	3.6E+06	nm	6.3E-01	n	2.6E+00	n					
													Polynuclear Aromatic Hydrocarbons (PAHs)														
				6.0E-02	I			V		1	0.13		~Acenaphthene	83-32-9	3.4E+03	n	3.3E+04	n					4.0E+02	n		4.1E+00	
				3.0E-01	I			V		1	0.13		~Anthracene	120-12-7	1.7E+04	n	1.7E+05	nm					1.3E+03	n		4.2E+01	
7.3E-01	E	1.1E-04	C						M	1	0.13		~Benz[a]anthracene	56-55-3	1.5E-01	c	2.1E+00	c	8.7E-03	c	1.1E-01	c	2.9E-02	c		1.0E-02	
1.2E+00	C	1.1E-04	C							1	0.13		~Benzo(j)fluoranthene	205-82-3	3.8E-01	c	1.3E+00	c	2.2E-02	c	1.1E-01	c	5.6E-02	c		6.7E-02	
7.3E+00	I	1.1E-03	C						M	1	0.13		~Benzo[a]pyrene	50-32-8	1.5E-02	c	2.1E-01	c	8.7E-04	c	1.1E-02	c	2.9E-03	c	2.0E-01	3.5E-03	2.4E-01
7.3E-01	E	1.1E-04	C						M	1	0.13		~Benzo[b]fluoranthene	205-99-2	1.5E-01	c	2.1E+00	c	8.7E-03	c	1.1E-01	c	2.9E-02	c		3.5E-02	
7.3E-02	E	1.1E-04	C						M	1	0.13		~Benzo[k]fluoranthene	207-08-9	1.5E+00	c	2.1E+01	c	8.7E-03	c	1.1E-01	c	2.9E-01	c		3.5E-01	
7.3E-03	E	1.1E-05	C						M	1	0.13		~Chrysene	218-01-9	1.5E+01	c	2.1E+02	c	8.7E-02	c	1.1E+00	c	2.9E+00	c		1.1E+00	
7.3E+00	E	1.2E-03	C						M	1	0.13		~Dibenz[a,h]anthracene	53-70-3	1.5E-02	c	2.1E-01	c	8.0E-04	c	1.0E-02	c	2.9E-03	c		1.1E-02	
1.2E+01	C	1.1E-03	C							1	0.13		~Dibenzo(a,e)pyrene	192-65-4	3.8E-02	c	1.3E-01	c	2.2E-03	c	1.1E-02	c	5.6E-03	c		7.3E-02	
2.5E+02	C	7.1E-02	C						M	1	0.13		~Dimethylbenz(a)anthracene, 7,12-	57-97-6	4.3E-04	c	6.2E-03	c	1.4E-05	c	1.7E-04	c	8.6E-05	c		8.5E-05	
				4.0E-02	I					1	0.13		~Fluoranthene	206-44-0	2.3E+03	n	2.2E+04	n					6.3E+02	n		7.0E+01	
				4.0E-02	I			V		1	0.13		~Fluorene	86-73-7	2.3E+03	n	2.2E+04	n					2.2E+02	n		4.0E+00	
7.3E-01	E	1.1E-04	C						M	1	0.13		~Indeno[1,2,3-cd]pyrene	193-39-5	1.5E-01	c	2.1E+00	c	8.7E-03	c	1.1E-01	c	2.9E-02	c		1.2E-01	
2.9E-02	P			7.0E-02	A			V		1	0.13	3.9E+02	~Methylnaphthalene, 1-	90-12-0	1.6E+01	c	5.3E+01	c					9.7E-01	c		5.1E-03	
				4.0E-03	I			V		1	0.13	3.7E+02	~Methylnaphthalene, 2-	91-57-6	2.3E+02	n	2.2E+03	ns					2.7E+01	n		1.4E-01	
		3.4E-05	C	2.0E-02	I	3.0E-03	I	V		1	0.13		~Naphthalene	91-20-3	3.6E+00	c*	1.8E+01	c*	7.2E-02	c*	3.6E-01	c*	1.4E-01	c*		4.7E-04	
1.2E+00	C	1.1E-04	C							1	0.13		~Nitropyrene, 4-	57835-92-4	3.8E-01	c	1.3E+00	c	2.2E-02	c	1.1E-01	c	1.6E-02	c		2.8E-03	
				3.0E-02	I			V		1	0.13		~Pyrene	129-00-0	1.7E+03	n	1.7E+04	n					8.7E+01	n		9.5E+00	
1.5E-01	I			9.0E-03	I					1	0.1		Prochloraz	67747-09-5	3.2E+00	c	1.1E+01	c					3.2E-01	c		1.6E-03	
				6.0E-03	H					1	0.1		Profluralin	26399-36-0	3.7E+02	n	3.7E+03	n					1.9E+01	n		1.2E+00	
				1.5E-02	I					1	0.1		Prometon	1610-18-0	9.2E+02	n	9.2E+03	n					1.9E+02	n		9.2E-02	
				4.0E-03	I					1	0.1		Prometryn	7287-19-6	2.4E+02	n	2.5E+03	n					4.5E+01	n		6.9E-02	
				1.3E-02	I					1	0.1		Propachlor	1918-16-7	7.9E+02	n	8.0E+03	n					1.9E+02	n		1.2E-01	
				5.0E-03	I					1	0.1		Propanil	709-98-8	3.1E+02	n	3.1E+03	n					6.3E+01	n		3.5E-02	
				2.0E-02	I					1	0.1		Propargite	2312-35-8	1.2E+03	n	1.2E+04	n					1.2E+02	n		8.8E+00	
				2.0E-03	I					1	0.1		Propargyl Alcohol	107-19-7	1.2E+02	n	1.2E+03	n					3.1E+01	n		6.4E-03	
				2.0E-02	I					1	0.1		Propazine	139-40-2	1.2E+03	n	1.2E+04	n					2.6E+02	n		2.3E-01	
				2.0E-02	I					1	0.1		Propham	122-42-9	1.2E+03	n	1.2E+04	n					2.7E+02	n		1.7E-01	
				1.3E-02	I					1	0.1		Propiconazole	60207-90-1	7.9E+02	n	8.0E+03	n					1.6E+02	n		5.3E-01	
						8.0E-03	I	V		1		3.3E+04	Propionaldehyde	123-38-6	8.0E+01	n	3.4E+02	n	8.3E+00	n	3.5E+01	n	1.7E+01	n		3.4E-03	
				1.0E-01	X	1.0E+00	X	V		1	0.1	2.6E+02	Propyl benzene	103-65-1	3.4E+03	ns	2.1E+04	ns	1.0E+03	n	4.4E+03	n	5.3E+02	n		9.9E-01	
						3.0E+00	C	V		1	0.1	3.5E+02	Propylene	115-07-1	2.4E+03	ns	1.0E+04	ns	3.1E+03	n	1.3E+04	n	6.3E+03	n		6.0E+00	
				2.0E+01	P					1	0.1		Propylene Glycol	57-55-6	1.2E+06	nm	1.2E+07	nm					3.1E+05	n		6.3E+01	
						2.7E-04	A			1	0.1		Propylene Glycol Dinitrate	6423-43-4	3.9E+05	nm	1.6E+06	nm	2.8E-01	n	1.2E+00	n					
				7.0E-01	H					1	0.1		Propylene Glycol Monoethyl Ether	1569-02-4	4.3E+04	n	4.3E+05	nm					1.1E+04	n		2.2E+00	
				7.0E-01	H	2.0E+00	I			1	0.1		Propylene Glycol Monomethyl Ether	107-98-2	4.3E+04	n	4.3E+05	nm	2.1E+03	n	8.8E+03	n	1.1E+04	n		2.2E+00	
2.4E-01	I	3.7E-06	I			3.0E-02	I	V		1		7.8E+04	Propylene Oxide	75-56-9	2.0E+00	c	9.0E+00	c	6.6E-01	c*	3.3E+00	c*	2.3E-01	c		4.8E-05	
				2.5E-01	I					1	0.1		Pursuit	81335-77-5	1.5E+04	n	1.5E+05	nm					3.9E+03	n		3.4E+00	
				2.5E-02	I					1	0.1		Pydrin	51630-58-1	1.5E+03	n	1.5E+04	n					3.9E+02	n		2.5E+02	
				1.0E-03	I			V		1		5.3E+05	Pyridine	110-86-1	7.8E+01	n	1.0E+03	n					1.5E+01	n		5.3E-03	
				5.0E-04	I					1	0.1		Quinalphos	13593-03-8	3.1E+01	n	3.1E+02	n					3.8E+00	n		3.2E-02	
3.0E+00	I									1	0.1		Quinoline	91-22-5	1.6E-01	c	5.7E-01	c					2.1E-02	c		6.8E-05	
						3.0E-02	A			1			Refractory Ceramic Fibers	NA	4.3E+07	nm	1.8E+08	nm	3.1E+01	n	1.3E+02	n					
				3.0E-02	I					1	0.1		Resmethrin	10453-86-8	1.8E+03	n	1.8E+04	n					4.8E+01	n		3.0E+01	
				5.0E-02	H					1	0.1		Ronnel	299-84-3	3.1E+03	n	3.1E+04	n					3.0E+02	n		2.7E+00	
				4.0E-03	I					1	0.1		Rotenone	83-79-4	2.4E+02	n	2.5E+03	n					4.7E+01	n		2.4E+01	
2.2E-01	C	6.3E-05	C						M	1	0.1		Safrole	94-59-7	5.2E-01	c	7.8E+00	c	1.5E-02	c	1.9E-01	c	6.2E-02	c		3.8E-05	
				2.5E-02	I					1	0.1		Savey	78587-05-0	1.5E+03	n	1.5E+04	n					8.1E+01	n		3.6E-01	
				5.0E-03	I					1			Selenious Acid	7783-00-8	3.9E+02	n	5.1E+03	n					7.8E+01	n			
				5.0E-03	I	2.0E-02	C			1			Selenium	7782-49-2	3.9E+02	n	5.1E+03	n	2.1E+01	n	8.8E+01	n	7.8E+01	n	5.0E+01	4.0E-01	2.6E-01
				5.0E-03	C	2.0E-02	C			1			Selenium Sulfide	7446-34-6	3.9E+02	n	5.1E+03	n	2.1E+01	n	8.8E+01	n	7.8E+01	n			

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Toxicity and Chemical-specific Information													Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	voc	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
				9.0E-02	I					1	0.1		Sethoxydim	74051-80-2	5.5E+03	n	5.5E+04	n					7.8E+02	n		6.9E+00	
						3.0E-03	C			1			Silica (crystalline, respirable)	7631-86-9	4.3E+06	nm	1.8E+07	nm	3.1E+00	n	1.3E+01	n					
				5.0E-03	I					0.04			Silver	7440-22-4	3.9E+02	n	5.1E+03	n					7.1E+01	n		6.0E-01	
1.2E-01	H			5.0E-03	I					1	0.1		Simazine	122-34-9	4.1E+00	c*	1.4E+01	c					5.2E-01	c	4.0E+00	2.6E-04	2.0E-03
				1.3E-02	I					1	0.1		Sodium Acifluorfen	62476-59-9	7.9E+02	n	8.0E+03	n					2.0E+02	n		1.6E+00	
				4.0E-03	I					1			Sodium Azide	26628-22-8	3.1E+02	n	4.1E+03	n					6.2E+01	n			
2.7E-01	H			3.0E-02	I					1	0.1		Sodium Diethyldithiocarbamate	148-18-5	1.8E+00	c	6.4E+00	c					2.5E-01	c			
				5.0E-02	A	1.3E-02	C			1			Sodium Fluoride	7681-49-4	3.9E+03	n	5.1E+04	n	1.4E+01	n	5.7E+01	n	7.8E+02	n			
				2.0E-05	I					1	0.1		Sodium Fluoroacetate	62-74-8	1.2E+00	n	1.2E+01	n					3.1E-01	n		6.3E-05	
				1.0E-03	H					1			Sodium Metavanadate	13718-26-8	7.8E+01	n	1.0E+03	n					1.6E+01	n			
2.4E-02	H			3.0E-02	I					1	0.1		Stirofos (Tetrachlorovinphos)	961-11-5	2.0E+01	c*	7.2E+01	c					2.4E+00	c		7.0E-03	
				6.0E-01	I					1			Strontium, Stable	7440-24-6	4.7E+04	n	6.1E+05	nm					9.3E+03	n		3.3E+02	
				3.0E-04	I					1	0.1		Strychnine	57-24-9	1.8E+01	n	1.8E+02	n					4.6E+00	n		5.1E-02	
				2.0E-01	I	1.0E+00	I	V		1		8.7E+02	Styrene	100-42-5	6.3E+03	ns	3.6E+04	ns	1.0E+03	n	4.4E+03	n	1.1E+03	n	1.0E+02	1.2E+00	1.1E-01
				1.0E-03	P	2.0E-03	P			1	0.1		Sulfolane	126-33-0	6.1E+01	n	6.2E+02	n	2.1E+00	n	8.8E+00	n	1.6E+01	n		3.4E-03	
				8.0E-04	P					1	0.1		Sulfonylbis(4-chlorobenzene), 1,1'-	80-07-9	4.9E+01	n	4.9E+02	n					1.3E+01	n		7.4E-02	
						1.0E-03	C			1			Sulfuric Acid	7664-93-9	1.4E+06	nm	6.0E+06	nm	1.0E+00	n	4.4E+00	n					
				2.5E-02	I					1	0.1		Systhane	88671-89-0	1.5E+03	n	1.5E+04	n					3.5E+02	n		4.3E+00	
				3.0E-02	H					1	0.1		TCMTB	21564-17-0	1.8E+03	n	1.8E+04	n					3.7E+02	n		2.6E+00	
				7.0E-02	I					1	0.1		Tebuthiuron	34014-18-1	4.3E+03	n	4.3E+04	n					1.1E+03	n		3.0E-01	
				2.0E-02	H					1	0.1		Temephos	3383-96-8	1.2E+03	n	1.2E+04	n					3.1E+02	n		6.0E+01	
				1.3E-02	I					1	0.1		Terbacil	5902-51-2	7.9E+02	n	8.0E+03	n					2.0E+02	n		5.9E-02	
				2.5E-05	H					1	0.1		Terbufos	13071-79-9	1.5E+00	n	1.5E+01	n					1.8E-01	n		3.9E-04	
				1.0E-03	I					1	0.1		Terbutryn	886-50-0	6.1E+01	n	6.2E+02	n					1.0E+01	n		1.4E-02	
				1.0E-04	I					1	0.1		Tetrabromodiphenyl ether, 2,2',4,4'- (BDE-47)	5436-43-1	6.1E+00	n	6.2E+01	n					1.6E+00	n		4.2E-02	
				3.0E-04	I					1	0.1		Tetrachlorobenzene, 1,2,4,5-	95-94-3	1.8E+01	n	1.8E+02	n					1.2E+00	n		5.8E-03	
2.6E-02	I	7.4E-06	I	3.0E-02	I			V		1		6.8E+02	Tetrachloroethane, 1,1,1,2-	630-20-6	1.9E+00	c	9.3E+00	c	3.3E-01	c	1.7E+00	c	5.0E-01	c		1.9E-04	
2.0E-01	I	5.8E-05	C	2.0E-02	I			V		1		1.9E+03	Tetrachloroethane, 1,1,2,2-	79-34-5	5.6E-01	c	2.8E+00	c	4.2E-02	c	2.1E-01	c	6.6E-02	c		2.6E-05	
2.1E-03	I	2.6E-07	I	6.0E-03	I	4.0E-02	I	V		1		1.7E+02	Tetrachloroethylene	127-18-4	2.2E+01	c**	1.1E+02	c**	9.4E+00	c**	4.7E+01	c**	9.7E+00	c**	5.0E+00	4.4E-03	2.3E-03
				3.0E-02	I					1	0.1		Tetrachlorophenol, 2,3,4,6-	58-90-2	1.8E+03	n	1.8E+04	n					1.7E+02	n		1.1E+00	
2.0E+01	H									1	0.1		Tetrachlorotoluene, p- alpha, alpha, alpha-	5216-25-1	2.4E-02	c	8.6E-02	c					3.4E-03	c		1.1E-05	
				5.0E-04	I					1	0.1		Tetraethyl Dithiopyrophosphate	3689-24-5	3.1E+01	n	3.1E+02	n					5.3E+00	n		3.9E-03	
						8.0E+01	I	V		1		1.1E+03	Tetrafluoroethane, 1,1,1,2-	811-97-2	1.1E+05	nms	4.6E+05	nms	8.3E+04	n	3.5E+05	n	1.7E+05	n		9.3E+01	
				4.0E-03	P					1	0.1		Tetryl (Trinitrophenylmethylnitramine)	479-45-8	2.4E+02	n	2.5E+03	n					6.3E+01	n		5.9E-01	
				1.0E-05	X					1			Thallium (Soluble Salts)	7440-28-0	7.8E-01	n	1.0E+01	n					1.6E-01	n	2.0E+00	1.1E-02	1.4E-01
				1.0E-02	I					1	0.1		Thiobencarb	28249-77-6	6.1E+02	n	6.2E+03	n					1.2E+02	n		4.2E-01	
				7.0E-02	X					1	0.008		Thiodiglycol	111-48-8	5.4E+03	n	6.8E+04	n					1.1E+03	n		2.2E-01	
				3.0E-04	H					1	0.1		Thiofanox	39196-18-4	1.8E+01	n	1.8E+02	n					4.1E+00	n		1.4E-03	
				8.0E-02	I					1	0.1		Thiophanate, Methyl	23564-05-8	4.9E+03	n	4.9E+04	n					1.2E+03	n		1.1E+00	
				5.0E-03	I					1	0.1		Thiram	137-26-8	3.1E+02	n	3.1E+03	n					7.6E+01	n		1.1E-01	
				6.0E-01	H					1			Tin	7440-31-5	4.7E+04	n	6.1E+05	nm					9.3E+03	n		2.3E+03	
						1.0E-04	A			1			Titanium Tetrachloride	7550-45-0	1.4E+05	nm	6.0E+05	nm	1.0E-01	n	4.4E-01	n					
				8.0E-02	I	5.0E+00	I	V		1		8.2E+02	Toluene	108-88-3	5.0E+03	ns	4.5E+04	ns	5.2E+03	n	2.2E+04	n	8.6E+02	n	1.0E+03	5.9E-01	6.9E-01
1.8E-01	X			1.0E-04	X					1	0.1		Toluene-2,5-diamine	95-70-5	2.7E+00	c**	9.6E+00	c**					3.7E-01	c**		1.2E-04	
1.9E-01	H									1	0.1		Toluidine, p-	106-49-0	2.6E+00	c	9.1E+00	c					3.4E-01	c		1.4E-04	
1.1E+00	I	3.2E-04	I							1	0.1		Toxaphene	8001-35-2	4.4E-01	c	1.6E+00	c	7.6E-03	c	3.8E-02	c	1.3E-02	c	3.0E+00	2.1E-03	4.6E-01
				7.5E-03	I					1	0.1		Tralomethrin	66841-25-6	4.6E+02	n	4.6E+03	n					1.2E+02	n		4.5E+01	
				3.0E-04	A					1	0.1		Tri-n-butyltin	688-73-3	1.8E+01	n	1.8E+02	n					4.7E+00	n		1.0E-01	
				1.3E-02	I					1	0.1		Triallate	2303-17-5	7.9E+02	n	8.0E+03	n					8.7E+01	n		1.9E-01	
				1.0E-02	I					1	0.1		Triasulfuron	82097-50-5	6.1E+02	n	6.2E+03	n					1.6E+02	n		1.6E-01	
				5.0E-03	I					1	0.1		Tribromobenzene, 1,2,4-	615-54-3	3.1E+02	n	3.1E+03	n					7.8E+01	n		1.1E-01	
9.0E-03	P			1.0E-02	P					1	0.1		Tributyl Phosphate	126-73-8	5.4E+01	c*	1.9E+02	c*					4.5E+00	c*		2.2E-02	
				3.0E-04	P					1	0.1		Tributyltin Compounds	NA	1.8E+01	n	1.8E+02	n					4.7E+00	n			
				3.0E-04	I					1	0.1		Tributyltin Oxide	56-35-9	1.8E+01	n	1.8E+02	n					4.4E+00	n		2.3E+02	
				3.0E+01	I	3.0E+01	H	V		1		9.1E+02	Trichloro-1,2,2-trifluoroethane, 1,1,2-	76-13-1	4.3E+04	ns	1.8E+05	nms	3.1E+04	n	1.3E+05	n	5.3E+04	n		1.3E+02	
7.0E-02	I			2.0E-02	I					1	0.1		Trichloroacetic Acid	76-03-9	6.9E+00	c	2.5E+01	c					9.4E-01	c	6.0E+01	1.9E-04	1.2E-02
2.9E-02	H									1	0.1		Trichloroaniline HCl, 2,4,6-	33663-50-2	1.7E+01	c	5.9E+01	c					2.3E+00	c		6.4E-03	
7.0E-03	X			3.0E-05	X					1	0.1		Trichloroaniline, 2,4,6-	634-93-5	1.8E+00	n	1.8E+01	n					3.0E-01	n		2.7E-03	
				8.0E-04	X			V		1	0.1	1.5E+02	Trichlorobenzene, 1,2,3-	87-61-6	4.9E+01	n	4.9E+02	ns					5.2E+00	n		1.5E-02	
2.9E-02	P			1.0E-02	I	2.0E-03	P	V		1		4.0E+02	Trichlorobenzene, 1,2,4-	120-82-1	2.2E+01	c**	9.9E+01	c**	2.1E+00	n	8.8E+00	n	9.9E-01	c**	7.0E+01	2.9E-03	2.0E-01
				2.0E+00	I	5.0E+00	I	V		1		6.4E+02	Trichloroethane, 1,1,1-	71-55-6	8.7E+03	ns	3.8E+04	ns	5.2E+03	n	2.2E+04	n	7.5E+03	n	2.0E+02	2.6E+00	7.0E-02
5.7E-02	I	1.6E-05	I	4.0E-03	I	2.0E-04	X	V		1		2.2E+03	Trichloroethane, 1,1,2-	79-00-5	1.1E+00	c**	5.3E+00	c**	1.5E-01	c**	7.7E-01	c**	2.4E-01	c**	5.0E+00	7.7E-05	1.6E-03
4.6E-02	I	4.1E-06	I	5.0E-04	I	2.0E-03	I	V	M	1		6.9E+02	Trichloroethylene	79-01-6	9.1E-01	c**	6.4E+00	c**	4.3E-01	c**	3.0E+00	c**	4.4E-01	c**	5.0E+00	1.6E-04	1.8E-03
				3.0E-01	I	7.0E-01	H	V		1		1.2E+03	Trichlorofluoromethane	75-69-4	7.9E+02	n	3.4E+03	ns	7.3E+02	n	3.1E+03	n	1.1E+03	n		6.9E-01	
				1.0E-01	I					1	0.1		Trichlorophenol, 2,4,5-	95-95-4	6.1E+03	n	6.2E+04	n					8.9E+02	n		3.3E+00	

Key: I = IRIS; P = PPRTV; A = ATSDR; C = Cal EPA; X = PPRTV Appendix; H = HEAST; J = New Jersey; Y = New York; O = EPA Office of Water; E = Environmental Criteria and Assessment Office; S = see user guide Section 5; L = see user guide on lead; M = mutagen; V = volatile; F = See FAQ; c = cancer; * = where: n SL < 100X c SL; ** = where n SL < 10X c SL; n = noncancer; m = Concentration may exceed ceiling limit (See User Guide); s = Concentration may exceed Csat (See User Guide); SSL values are based on DAF=1

Toxicity and Chemical-specific Information													Contaminant		Screening Levels											Protection of Ground Water SSLs	
SFO (mg/kg-day)$^{-1}$	key	IUR (ug/m^3)$^{-1}$	key	RfD_o (mg/kg-day)	key	RfC_i (mg/m^3)	key	vol	muta-gen	GIABS	ABS	C_{sat} (mg/kg)	Analyte	CAS No.	Resident Soil (mg/kg)	key	Industrial Soil (mg/kg)	key	Resident Air (ug/m^3)	key	Industrial Air (ug/m^3)	key	Tapwater (ug/L)	key	MCL (ug/L)	Risk-based SSL (mg/kg)	MCL-based SSL (mg/kg)
1.1E-02	I	3.1E-06	I	1.0E-03	P					1	0.1		Trichlorophenol, 2,4,6-	88-06-2	4.4E+01	c**	1.6E+02	c**	7.8E-01	c	4.0E+00	c	3.5E+00	c**		1.3E-02	
				1.0E-02	I					1	0.1		Trichlorophenoxyacetic Acid, 2,4,5-	93-76-5	6.1E+02	n	6.2E+03	n					1.2E+02	n		5.2E-02	
				8.0E-03	I					1	0.1		Trichlorophenoxypropionic acid, -2,4,5	93-72-1	4.9E+02	n	4.9E+03	n					8.4E+01	n	5.0E+01	4.6E-02	2.8E-02
				5.0E-03	I			V		1		1.3E+03	Trichloropropane, 1,1,2-	598-77-6	3.9E+02	n	5.1E+03	ns					7.8E+01	n		3.1E-02	
3.0E+01	I			4.0E-03	I	3.0E-04	I	V	M	1		1.4E+03	Trichloropropane, 1,2,3-	96-18-4	5.0E-03	c	9.5E-02	c	3.1E-01	n	1.3E+00	n	6.5E-04	c		2.8E-07	
				3.0E-03	X	3.0E-04	P	V		1		4.5E+02	Trichloropropene, 1,2,3-	96-19-5	7.8E-01	n	3.3E+00	n	3.1E-01	n	1.3E+00	n	6.2E-01	n		3.1E-04	
				3.0E-03	I					1	0.1		Tridiphane	58138-08-2	1.8E+02	n	1.8E+03	n					4.7E+01	n		3.3E-01	
						7.0E-03	I	V		1		2.8E+04	Triethylamine	121-44-8	1.2E+02	n	5.2E+02	n	7.3E+00	n	3.1E+01	n	1.5E+01	n		4.4E-03	
7.7E-03	I			7.5E-03	I					1	0.1		Trifluralin	1582-09-8	6.3E+01	c**	2.2E+02	c*					2.2E+00	c*		7.2E-02	
2.0E-02	P			1.0E-02	P					1	0.1		Trimethyl Phosphate	512-56-1	2.4E+01	c*	8.6E+01	c*					3.4E+00	c*		7.4E-04	
						5.0E-03	P	V		1		2.9E+02	Trimethylbenzene, 1,2,3-	526-73-8	5.3E+01	n	2.2E+02	n	5.2E+00	n	2.2E+01	n	1.0E+01	n		1.5E-02	
						7.0E-03	P	V		1		2.2E+02	Trimethylbenzene, 1,2,4-	95-63-6	6.2E+01	n	2.6E+02	ns	7.3E+00	n	3.1E+01	n	1.5E+01	n		2.1E-02	
				1.0E-02	X			V		1		1.8E+02	Trimethylbenzene, 1,3,5-	108-67-8	7.8E+02	ns	1.0E+04	ns					8.7E+01	n		1.2E-01	
				3.0E-02	I					1	0.019		Trinitrobenzene, 1,3,5-	99-35-4	2.2E+03	n	2.7E+04	n					4.6E+02	n		1.7E+00	
3.0E-02	I			5.0E-04	I					1	0.032		Trinitrotoluene, 2,4,6-	118-96-7	1.9E+01	c**	7.9E+01	c**					2.2E+00	c**		1.3E-02	
				2.0E-02	P					1	0.1		Triphenylphosphine Oxide	791-28-6	1.2E+03	n	1.2E+04	n					2.8E+02	n		1.2E+00	
2.0E-02	P			7.0E-03	P					1	0.1		Tris(2-chloroethyl)phosphate	115-96-8	2.4E+01	c*	8.6E+01	c*					3.3E+00	c*		3.2E-03	
3.2E-03	P			1.0E-01	P					1	0.1		Tris(2-ethylhexyl)phosphate	78-42-2	1.5E+02	c*	5.4E+02	c					2.1E+01	c*		1.0E+02	
				3.0E-03	I					1			Uranium (Soluble Salts)	NA	2.3E+02	n	3.1E+03	n					4.7E+01	n	3.0E+01	2.1E+01	1.4E+01
1.0E+00	C	2.9E-04	C						M	1	0.1		Urethane	51-79-6	1.2E-01	c	1.7E+00	c	3.3E-03	c	4.2E-02	c	2.1E-02	c		4.8E-06	
		8.3E-03	P	9.0E-03	I	7.0E-06	P			0.026			Vanadium Pentoxide	1314-62-1	4.0E+02	c**	2.0E+03	c**	2.9E-04	c*	1.5E-03	c*	1.1E+02	n			
				5.0E-03	S					1			Vanadium and Compounds	NA	3.9E+02	n	5.2E+03	n					7.8E+01	n		7.8E+01	
				1.0E-03	I					1	0.1		Vernolate	1929-77-7	6.1E+01	n	6.2E+02	n					8.3E+00	n		6.6E-03	
				2.5E-02	I					1	0.1		Vinclozolin	50471-44-8	1.5E+03	n	1.5E+04	n					3.4E+02	n		2.6E-01	
				1.0E+00	H	2.0E-01	I	V		1		2.8E+03	Vinyl Acetate	108-05-4	9.7E+02	n	4.1E+03	ns	2.1E+02	n	8.8E+02	n	4.1E+02	n		8.7E-02	
		3.2E-05	H			3.0E-03	I	V		1		0.0E+00	Vinyl Bromide	593-60-2	1.1E-01	c*s	5.6E-01	c*s	7.6E-02	c*	3.8E-01	c*	1.5E-01	c*		4.4E-05	
7.2E-01	I	4.4E-06	I	3.0E-03	I	1.0E-01	I	V	M	1		3.9E+03	Vinyl Chloride	75-01-4	6.0E-02	c	1.7E+00	c	1.6E-01	c	2.8E+00	c	1.5E-02	c	2.0E+00	5.3E-06	6.9E-04
				3.0E-04	I					1	0.1		Warfarin	81-81-2	1.8E+01	n	1.8E+02	n					4.4E+00	n		4.6E-03	
				2.0E-01	S	1.0E-01	S	V		1		3.9E+02	Xylene, P-	106-42-3	6.0E+02	ns	2.6E+03	ns	1.0E+02	n	4.4E+02	n	1.9E+02	n		1.8E-01	
				2.0E-01	S	1.0E-01	S	V		1		3.9E+02	Xylene, m-	108-38-3	5.9E+02	ns	2.5E+03	ns	1.0E+02	n	4.4E+02	n	1.9E+02	n		1.8E-01	
				2.0E-01	S	1.0E-01	S	V		1		4.3E+02	Xylene, o-	95-47-6	6.9E+02	ns	3.0E+03	ns	1.0E+02	n	4.4E+02	n	1.9E+02	n		1.9E-01	
				2.0E-01	I	1.0E-01	I	V		1		2.6E+02	Xylenes	1330-20-7	6.3E+02	ns	2.7E+03	ns	1.0E+02	n	4.4E+02	n	1.9E+02	n	1.0E+04	1.9E-01	9.8E+00
				3.0E-04	I					1			Zinc Phosphide	1314-84-7	2.3E+01	n	3.1E+02	n					4.7E+00	n			
				3.0E-01	I					1			Zinc and Compounds	7440-66-6	2.3E+04	n	3.1E+05	nm					4.7E+03	n		2.9E+02	
				5.0E-02	I					1	0.1		Zineb	12122-67-7	3.1E+03	n	3.1E+04	n					7.8E+02	n		2.3E+00	

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Use of California Human Health Screening Levels (CHHSLs) in Evaluation of Contaminated Properties

January 2005

California Environmental Protection Agency

DISCLAIMER

Use of California Human Health Screening Levels in Evaluation of Contaminated Properties has been prepared by the California Environmental Protection Agency (Cal/EPA). This document is not intended to establish policy or regulation. The Human Health Screening Levels presented here are not to serve as: 1) a stand-alone decision making tool, 2) a substitute for guidance for the preparation of baseline human health risk assessments, 3) a rule to determine if a waste is hazardous under the state or federal regulations, 4) a rule to determine when the release of hazardous chemicals must be reported to the overseeing regulatory agency, 5) set of final cleanup or action levels to be applied at contaminated sites or 6) a guarantee that an oversight regulatory agency will determine that a project is adequately studied or agree with the conclusions of the site investigation and risk assessment report.

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The CHHSLs should NOT be used to determine when impacts at a site should be reported to a regulatory agency. The list of CHHSLs is also not a comprehensive list of all potential chemicals of concern that may be found at a property. All releases of hazardous substances to the environment should be reported to the appropriate regulatory agency in accordance with governing regulations. Staff overseeing work at a specific site should be contacted prior to use of the information in this document to ensure that the document is applicable to the site and that the user has the most up-to-date version available.

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1 HUMAN-EXPOSURE-BASED SCREENING NUMBERS DEVELOPED TO AID ESTIMATION OF CLEANUP COSTS FOR CONTAMINATED SOIL, NOVEMBER 2004, JANUARY 2005 REVISION

Overview

What are the CHHSLs?

The California Human Health Screening Levels (CHHSLs or "Chisels") are concentrations of 54 hazardous chemicals in soil or soil gas that the California Environmental Protection Agency (Cal/EPA) considers to be below thresholds of concern for risks to human health. The CHHSLs were developed by the Office of Environmental Health Hazard Assessment (OEHHA) on behalf of Cal/EPA, and are contained in their report entitled "Human-Exposure-Based Screening Numbers Developed to Aid Estimation of Cleanup Costs for Contaminated Soil" (Appendix 1). The thresholds of concern used to develop the CHHSLs are an excess lifetime cancer risk of one-in-a-million (10^{-6}) and a hazard quotient of 1.0 for noncancer health effects. The CHHSLs were developed using standard exposure assumptions and chemical toxicity values published by the U.S. Environmental Protection Agency (USEPA) and Cal/EPA.

How can the CHHSLs help facilitate restoration of contaminated properties?

The CHHSLs can be used to screen sites for potential human health concerns where releases of hazardous chemicals to soils have occurred. Under most circumstances, and within the limitations described in this document, the presence of a chemical in soil, soil gas or indoor air at concentrations below the corresponding CHHSLs can be assumed to not pose a significant health risk to people who may live (residential CHHSLs) or work (commercial/industrial CHHSLs) at the site. As discussed below, however, evaluation of other potential environmental concerns must also be addressed.

The presence of a chemical at concentrations in excess of a CHHSL does <u>not</u> indicate that adverse impacts to human health are occurring or will occur but suggests that further evaluation of potential human health concerns is warranted. Residential CHHSLs may be used in conjunction with the human health screening evaluation described in the Department of Toxic Substances Control (DTSC) Preliminary Endangerment Assessment (PEA) Guidance Manual to assist the risk manager in deciding whether further site characterization, risk assessment, or remediation is necessary (Cal/EPA 1994b). Further evaluation may include additional sampling at the site, consideration of ambient levels in the environment, or a reassessment of the assumptions used to calculate the CHHSLs

or PEA estimates. This stepwise approach expedites judgments about the degree of effort that may be necessary to remediate contaminated properties and restore the properties to productive use.

How do the CHHSLs differ from cleanup standards?

The CHHSLs presented in the lookup tables are NOT regulatory "cleanup standards". Use of the CHHSLs and this document is voluntary on the part of those who choose to use them. At sites where cleanup of contaminated soils to levels at or below the CHHSLs would be costly, the time and effort to develop more site-specific cleanup may be desired. At sites where the extent of contaminated soil is limited or the timeframe available to carry out cleanup actions is very short, use of the CHHSLs as final soil cleanup standards may be cost-beneficial. However, this would require the concurrence of both the responsible party and the overseeing regulatory agency and can only be done after a full evaluation of site conditions and other potential environmental concerns. Regulatory agencies cannot be compelled to use the CHHSLs as final cleanup standards for a contaminated property.

If contaminant concentrations are below the CHHSLs am I finished?

As discussed above, the CHHSLs cannot be used as a stand-alone tool for final cleanup and closure decisions. In addition, using only the CHHSLs may not be protective of groundwater resources or address other potential environmental concerns. Therefore, a thorough investigation of site conditions must also be performed to ensure that: 1) all potential human exposure pathways and exposure scenarios at the site are fully accounted for; 2) groundwater resources are protected; 3) terrestrial and aquatic habitats are protected, including the erosion of contaminated soils and subsequent runoff into a nearby wetland, stream or other aquatic habitat; and 4) that nuisance (e.g., odors and staining) and gross contamination concerns are addressed. These and other issues related to environmental contamination that are identified at the site must be evaluated separately. If a formal regulatory decision or determination is desired, additional assessment or cleanup of contaminated soils to address these concerns may ultimately be required.

How should the CHHSLs be integrated into the DTSC PEA process?

The human health screening evaluation presented in the DTSC Preliminary Endangerment Assessment (PEA) document is intended to provide a preliminary evaluation of potential risk and hazard to human health. The PEA process uses models and exposure assumptions similar to those used to develop the residential CHHSLs but does not provide actual risk-based screening levels based on these models. The PEA screening evaluation assumes that the land use of the site will be residential, regardless of the current use and zoning for the site. Therefore, residential CHHSLs for specific chemicals may be utilized in a PEA. Chemicals that do not have CHHSLs should be evaluated using the DTSC PEA methodology for their potential to pose human health risks. Chemicals found at a site should be evaluated separately for other potential environmental concerns, using the PEA guidance and other references as appropriate. The user should consult DTSC for additional information about use of the CHHSLs in the PEA process.

How are the CHHSLs related to the USEPA Preliminary Remediation Goals (PRGs) and to the San Francisco Bay Regional Water Quality Control Board Environmental Screening Levels (ESLs)?

The soil and soil gas CHHSLs are modeled after the USEPA Region IX "Preliminary Remediation Goals (PRGs)" for these media (http://www.epa.gov/region09/waste/sfund/prg/index.htm). The primary difference between the CHHSLs and the PRGs is the use of Cal/EPA-specific "toxicity factors" (estimates of a chemical's toxicity to humans) in development of the CHHSLs, when available, rather than toxicity factors published by the USEPA. For volatile chemicals, soil gas CHHSLs were developed to evaluate the potential intrusion of subsurface vapors (soil gas) into buildings and subsequent impacts to indoor air quality.

The San Francisco Bay Regional Water Quality Control Board (SFBRWQCB) Environmental Screening Levels (ESLs) are a compilation of screening levels for not only risk to human health but also a number of other environmental concerns. The ESLs are intended for use only at sites overseen by that agency. These ESLs may be found at the SFRWQCB web site at http://www.waterboards.ca.gov/sanfranciscobay/esl.htm. The SFBRWQCB refers to the comprehensive evaluation of all potential environmental concerns as an "Environmental Risk Assessment," as opposed to a more focused "Human Health Risk Assessment" reflected in development of the CHHSLs and this

document in general. The soil, soil gas and indoor air ESLs and CHHSLs for human health concerns were developed using similar methodology and are essentially identical. In addition, the SFBRWQCB document provides soil screening levels for leaching of contaminants into groundwater, toxicity to flora and fauna and nuisance or gross contamination concerns. These concerns are not addressed by the CHHSLs and must be evaluated separately.

Because many different sets of screening levels are now available, the overseeing regulatory agency should be consulted before using any screening levels in a human health screening evaluation. The regulatory agency may have specific recommendations with respect to which screening levels it prefers to use at sites under their jurisdiction.

If I am in the jurisdiction of the San Francisco Bay Regional Water Quality Control Board, can I continue to use that office's Environmental Screening Levels (ESLs) document?

At sites in the jurisdiction of and overseen by the SFBRWQCB, the reader should consult the SFBRWQCB regarding continued use of the ESLs versus use of the CHHSLs.

How often are the CHHSLs updated?

The CHHSLs will be updated as needed to incorporate new toxicity information of referenced chemicals as well as new information regarding the exposure or potential exposure of humans to potentially hazardous chemicals in soils. CHHSLs for additional chemicals will also be included as they become available.

Who can I contact for more information?

Refer to the CHHSL link posted on the Cal/EPA website (www.calepa.ca.gov) for further information and local contacts. The document will also be posted on the OEHHA web site (www.oehha.ca.gov), the DTSC web site (www.dtsc.ca.gov), the SWRCB web site (www.waterboards.ca.gov) and at the SFBRWQCB web site (www.waterboards.ca.gov/sanfranciscobay/), as well as other Regional Boards' web sites.

1 Introduction

1.1 Purpose and Development

The California Human Health Screening Levels (CHHSLs) were developed as a tool to assist in the evaluation of contaminated sites for potential adverse threats to human health. Residential and commercial/industrial land use screening levels for soil, soil gas and indoor air are provided in Tables 1 and 2. The screening levels in Table 1 pertain to direct exposure of humans to contaminants in soil via incidental soil ingestion, dermal contact and inhalation of vapors or dust in outdoor air. The soil gas and indoor air screening levels in Table 2 pertain to the emission of volatile chemicals from contaminated soil or groundwater and their potential intrusion into overlying buildings.

Preparation of the CHHSLs by the California Environmental Protection Agency (Cal/EPA) was required under the California Land Environmental Restoration and Reuse Act of 2001 (CLERRA 2001). CLERRA also required that a guidance document be prepared to explain how the CHHSLS may be used in California to aid in making judgments about the degree of effort (or costs) that might be necessary to remediate contaminated properties, facilitate the restoration and revitalization of contaminated properties, and assist local-level remediation programs in making more efficient and effective decisions.

Appendix 1 is the Office of Environmental Health Hazard Assessment's (OEHHA) report entitled "Human-Exposure-Based Screening Numbers Developed to Aid Estimation of Cleanup Costs for Contaminated Soil" which contains the CHHSLs, and describes the approach used to develop the human-health-risk-based screening levels, the comments received regarding the draft document and OEHHA's response to those comments. The approach reflected in OEHHA's report is based on the USEPA *Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A)* (USEPA 1989) and is essentially equivalent to the approach used by USEPA Region IX in developing their *Preliminary Remediation Goals* (USEPA 2004), the San Francisco Bay Area Regional Water Quality Control Board (SFRWQCB) in developing their Environmental Screening Levels for human health (SFRWQCB 2003), and the Department of Toxic Substances Control (DTSC) in their Preliminary Endangerment Assessment (PEA) guidance (Cal/EPA 1994b).

Soil and soil gas data collected at a site can be directly compared to CHHSLs for each chemical of concern. Under most circumstances, and within the limitations described, the presence of a chemical in soil or soil gas at concentrations below the corresponding CHHSLs can be assumed to not pose a significant health risk to people who may live or work at the site. The presence of a chemical at concentrations in excess of a CHHSL does not necessarily indicate that adverse impacts to human health are occurring but indicates that a potential for adverse risk may exist and that additional evaluation is warranted.

Residential CHHSLs are appropriate for other types of sensitive property use, including hospitals, day care centers and schools. In order to assess the maximum, future beneficial use of a property, data collected at commercial or industrial sites should be compared to both residential and commercial sets of screening levels. A formal restriction to the deed may be required for sites that meet requirements for commercial/industrial use but not residential use. Regulatory agency oversight would be needed in this circumstance.

The scope of the CHHSLs is limited to human health concerns. For this reason, the CHHSLs cannot be used as a stand-alone tool to determine the extent of remedial actions needed at sites with contaminated soils. Depending on site conditions and the chemicals present, additional cleanup of contaminated soils may be required to protect groundwater resources, prevent toxicity to flora and fauna, address uptake in edible plants, and address nuisance and aesthetic concerns posed by odors and staining. A brief summary of these concerns and a list of references for evaluating these issues are provided at the end of the text.

1.2 Tiered Approach to Environmental Risk Assessments

Human health risk assessments for regulatory purposes are usually carried out using a step-wise or "tiered" approach. Comparison of site data to residential soil or soil gas CHHSLs (e.g., in a screening health risk evaluation performed using the DTSC PEA guidance) usually represents "Tier 1". If multiple chemicals with similar health effects are present at a site then "forward mode," cumulative health risks may also need to be calculated and compared to target Tier 1 goals before an evaluation of potential human health concerns can be completed (refer to Section 2.8).

If the results of the Tier 1 assessment indicate that further evaluation of human health risks is warranted, site-specific exposure assumptions, target risks, etc., can

be substituted for default parameter values used to develop the Tier 1 CHHSLs and alternative screening levels developed under a Tier 2 assessment. This assessment can be incorporated into the guidelines presented in the DTSC PEA document. Prior to modifying the Tier 1 default assumptions, concurrence from the appropriate regulatory agency should be obtained. Site data can then be compared to the revised screening levels. This provides an intermediate but still relatively rapid and cost-effective option for preparing more site-specific screening or cleanup levels. Cumulative health risks or hazards should also be presented under a Tier 2 assessment, as described in Section 2.8.

If exposure pathways of concern and conditions at the site do not match those taken into account by the CHHSL framework or PEA methodology, a Tier 3, baseline human health and ecological risk assessment should be performed. In a baseline human health and ecological risk assessment, alternative models and site-specific assumptions are used to quantify the risk/hazard posed to human and/or ecological receptors by the impacted media in the "forward" mode. After a baseline health risk assessment is accepted by the regulatory agency, the assessment may be used in the "backward' model to develop site-specific screening or cleanup levels. An understanding of the methodologies used to develop the CHHSLs is important to ensure consistency between all tiers of assessments and to expedite their preparation and review.

1.3 Chemicals Not Listed In CHHSL Lookup Tables

The lookup tables list 54 chemicals, including many that are commonly found at sites where releases of hazardous chemicals have occurred. Cal/EPA will incorporate CHHSLs for additional chemicals in future updates of this document as needed and practical. Prior to that time, the PEA methodology should be used to evaluate those chemicals for which CHHSLs do not exist. Toxicity factors published by Cal/EPA should be utilized in the PEA when available, unless otherwise instructed by the overseeing regulatory agency.

1.4 Limitations

The CHHSLs presented in this document are NOT regulatory "cleanup standards." Use of the CHHSLs as final cleanup levels to address human health concerns should be discussed with the overseeing regulatory agency and evaluated in terms of the cost/benefit of developing more site-specific cleanup levels through a risk assessment.

The CHHSLs presented in this document are NOT adequate to evaluate ALL environmental conditions at ALL contaminated sites. Other environmental concerns posed by the presence of contamination at a site may include:

- Leaching of contaminants from soil to groundwater and subsequent impacts to groundwater quality;
- Intrusion of subsurface vapors into basements or buildings with substandard ventilation systems and subsequent impacts to indoor air;
- Uptake of contaminants in edible fruit and vegetables and subsequent intake by humans;
- Exposure of children and teachers at school sites;
- Toxicity to terrestrial flora and fauna;
- Gross contamination, including nuisance (odors, etc.) and aesthetic concerns.

A summary of potential environmental concerns that may also be relevant at a site for a particular chemical is also provided in Table 1.

The CHHSLs specifically do not address contamination in groundwater, surface water or sediment or the erosion of contaminated soils and subsequent runoff into a nearby wetland, stream or other aquatic habitat. Contamination identified in these media or that may threaten these media must be considered separately. References for evaluation of contaminants in these media are provided in Chapter 4.

The soil gas CHHSLs for the intrusion of vapors into buildings may not be adequately conservative for estimating impacts to indoor air in poorly ventilated basements or buildings with substandard ventilation systems in general. Additional guidance on this subject is provided in Section 2.5.2.

The CHHSLs for direct-exposure to soils concerns are calculated assuming that specific exposure pathways are complete for the human receptor: incidental soil ingestion, dermal absorption of chemicals in soil, and inhalation of vapors or particulate matter in ambient (outdoor) air. For volatile chemicals, the soil gas CHHSLs are calculated assuming that the exposure pathway of inhalation of

indoor air contaminated with vapors intruding from the subsurface is complete. If these pathways are not congruent with site conditions, the CHHSLs should not be used. The PEA guidance should then be followed.

The CHHSLS for inorganic chemicals (metals) are based on human health risks. However, metals are naturally occurring in the soil. Therefore, metals concentrations should be compared to local background levels as discussed in Section 2.7.

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2 CHHSL Lookup Tables

2.1 Organization of Lookup Tables

CHHSLS for soil, soil gas and indoor air are presented in Tables 1 and 2. Soil CHHSLs address the potential direct exposure of residents and workers to contaminants in soil. Indoor air and soil gas screening levels address the potential intrusion of subsurface vapors into buildings and subsequent impacts to indoor air quality (and resulting potential exposure of residents and workers in those buildings).

Separate CHHSLs are presented for residential and commercial/industrial land uses. A summary of models and exposure assumptions used for each land use is in Appendix 1. The category "Residential Land Use" applies to sites where unrestricted land use is desired. This includes use for residences, hospitals, day-care centers and other sensitive purposes (Cal/EPA 2002). Residential CHHSLs incorporate conservative assumptions regarding the long-term, frequent exposure of children and adults to contaminated soils in a residential setting. In contrast, "Commercial/Industrial Use Only" assumes that only working age adults will be present at the site on a regular basis. Exposure assumptions incorporated into these CHHSLs are less conservative than assumptions used in the residential land-use scenario.

In a DTSC PEA, the land use of the site under a Tier 1 assessment is assumed to be residential, regardless of the current use and zoning for the site. Other regulatory agencies may evaluate land use 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 evaluation.

If chemicals at a site exceed residential CHHSLs but are below CHHSLs for commercial/industrial land-use, restrictions on the use of affected property will likely be necessary (refer to Section 2.10). The need for such restrictions should be weighed against the cost-benefit of remediating the property to meet the CHHSLs for unrestricted land use.

Although schools may also be a sensitive land use, proposed school sites must be evaluated using the OEHHA Guidance for Assessing Exposures and Health Risks at Existing and Proposed School Sites (Cal/EPA 2004a) rather than the CHHSLs. Refer to Section 2.9 for a discussion of school-specific risk evaluations. 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.

2.2 Developing a Conceptual Site Model

The primary condition for use of CHHSLs is that exposure pathways of concern and conditions at the site match those taken into account in the development of the CHHSLs. Thus, it is always necessary to develop a conceptual site model (CSM) to identify likely contaminant source areas, exposure pathways, and potential receptors to determine the applicability of CHHSLs at the site and the need for additional information. The conceptual site model summarizes information about site conditions in a schematic presentation in terms of: 1) primary sources (e.g., leaking tanks); 2) secondary sources (e.g., contaminated soil); 3) contaminant transport mechanisms (e.g., volatilization and intrusion into buildings); 4) contaminated exposure media (e.g., indoor air); and 5) potentially complete exposure pathways.

The CSM can be used to provide a rationale for additional site investigation, as a basis for a more detailed CSM, and/or to select screening levels or cleanup levels for specific environmental concerns. An example model is shown in Figure 2-1. The example model represents a hypothetical release of petroleum-based fuels and pesticides to soil and groundwater at a large housing redevelopment project with open spaces accessible to residents (direct exposure), enclosed buildings (vapor intrusion), wetlands (ecotoxicity) and communal garden areas where fruits and vegetables are grown (uptake in edible plants). Potential environmental concerns at the hypothetical site are identified by a check mark in the appropriate column. In addition, xylene and other compounds in petroleum often cause odor and aesthetic concerns (nuisances). Cleanup to address these and other gross contamination concerns may be required even after all other potential concerns have been adequately addressed.

If completed exposure pathways at a site match those pathways considered in the development of the CHHSLs, the appropriate soil and soil gas data can be directly compared to the CHHSLs to determine if the magnitude of exposure may pose a potential threat to human health. If the exposure pathways at a site do not match those pathways used in the development of the CHHSLs, these screening levels <u>may not</u> be used, and a site-specific human health risk evaluation should be performed.

Other potential environmental concerns must be evaluated separately, either through use of a comparable set of screening levels or through a more detailed, site-specific environmental risk assessment. Additional information regarding the preparation of conceptual site models is provided in the DTSC *Preliminary Endangerment Assessment Manual* (Cal/EPA 1994b), the USEPA Region IX *Preliminary Remediation Goals* document (USEPA 2004), the USEPA *Guidance for Conducting Remedial Investigations and Feasibility Studies Under CERCLA*, Interim Final Document (USEPA 1988) and the Region 2 Environmental Screening Levels document (SFBRWQCB 2003).

2.3 Using the Lookup Tables

A step-by-step approach for using the CHHSLs is summarized below.

Step 1 – Check for CHHSL Updates and Applicability

Check with the overseeing regulatory agency to determine if the CHHSLs can be applied to the subject site. Ensure that the most up-to-date CHHSLs are being used.

Step 2 - Prepare a Conceptual Site Model

The purpose of the conceptual site model is to present information about site conditions and potential impacts to receptors. All potential environmental concerns at the site (e.g., contaminant sources, pathways, exposure routes and receptors) should be clearly identified in a conceptual site model (Section 2.2 and Chapter 4). Identification of these concerns helps to provide the rationale for the type and location for site sampling. The level of detail required in a conceptual site model will vary from site to site. The presentation and scope of the model should be discussed with the overseeing regulatory agency. The conceptual site model should be continually updated as additional data for the site is obtained.

Step 3 – Collect Data

An environmental risk assessment is based on the results of a thorough site investigation, where all chemicals of potential concern have been identified. The scope and type of site investigation will vary depending on the site specific history and the nature of the actual or suspected chemical release. Sampling objectives should be defined in advance of field activities. For example, the objective may be to document whether a release has occurred; to identify hot spots that may require an expedited removal action; to provide sufficient data to determine whether site remediation is necessary; or to evaluate whether site conditions would be consistent with proposed or potential land uses.

Steps 4 - Determine the Desired Land Use

Screening levels for residential land use are generally appropriate for other sensitive uses of the property (e.g., day-care centers, hospitals, etc.). If preparing a DTSC PEA, residential land use CHHSLs should be used. **For evaluation of commercial/industrial properties, it is highly recommended that site data be compared to CHHSLs for both unrestricted/residential and commercial/industrial land use.** Commercial/industrial CHHSLs should be used only under the oversight of a regulatory agency, as that agency will likely require a land use covenant that restricts use of the property to these purposes.

Steps 5 - Select CHHSLs

Based on the actual or proposed land use, select the appropriate soil and/or soil gas CHHSLs. Replace CHHSLs with naturally occurring, background concentrations of chemicals of concern (e.g., arsenic) or laboratory method reporting levels if appropriate (see Sections 2.6 and 2.7).

Step 6 - Compare Site Data To CHHSLs; calculate cumulative risks as necessary

Compare site data to CHHSLs to identify areas where concentrations of contaminants pose potential human health concerns. For sites where sample data are limited and/or if preparing a DTSC PEA, compare the maximum-detected concentrations of chemicals of concern to the CHHSLs.

For sites where an adequate number of data points are available, statistical methods can be used to estimate site-specific exposure point concentrations. The exposure point concentration is the lesser of the maximum-detected concentration and the 95% upper confidence limit (UCL) of the arithmetic mean of sample data (Cal/EPA 1996a). The USEPA guidance document *Calculating Upper Confidence Limits for Exposure Point Concentrations at Hazardous Waste Sites* recommends evaluating the distribution of the data and choosing the best UCL estimate for the data set (USEPA 2002). Guidance for the estimation of exposure point concentrations, use of "non-detect" data, and other issues is also provided in the Cal/EPA documents *Preliminary Endangerment Assessment Guidance Manual* (Cal/EPA 1994b), *Supplemental Guidance For Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities* (Cal/EPA 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, soil sample data should be averaged over no more than a 1,000 ft^2 area (assumed area of a typical, urban area back yard and footprint area of typical residence). For commercial/industrial properties, soil sample data can be averaged within affected areas of open spaces.

Use the maximum soil gas concentration over an area of the footprint of existing or assumed future buildings to compensate for potentially isolated rooms within a building and the uncertainties in soil gas collection.

If multiple chemicals with similar heath effects are present at a site, the cumulative excess cancer risk and/or noncancer hazard index should be calculated before final consideration of the site for closure. This will be of particular concern at sites where residual concentrations of chemicals with similar noncancer health effects may approach CHHSLs following the proposed, final cleanup of contaminated soil. Calculation of cumulative risks and hazard indices is discussed in Section 2.8. The need to include calculation of cumulative health risks in final closure reports should be discussed with the overseeing regulatory agency.

Steps 7 - Evaluate the Need for Additional Investigation or Actions to Address Human Health Concerns

Based on a comparison of available site data to the CHHSLs, the objectives identified in Step 3 should be evaluated. For example, comparison to CHHSLs may show that a site does not pose an unacceptable health risk to residential users, or it may show that additional investigation is warranted. Summarize the results of this evaluation in the Tier 1 Human Health Risk Assessment report (or preliminary endangerment assessment), and include recommendations for additional investigations or remediation as needed. Decisions for or against additional actions should always be made in coordination with the overseeing regulatory agency.

Step 8 - Evaluate Other Potential Environmental Concerns

The soil CHHSLs presented in Table 1 are limited to human health concerns associated with direct exposure to contaminated soil. In many instances, the presence of a potential hazardous chemical in soil may pose other environmental concerns that outweigh the risk to human health through direct exposure (see Sections 1.4 and 2.2, Chapter 4 and Table 1). The purpose of the Conceptual Site Model (Step 2) is to assist the user in identifying these concerns early in the process. For example, many metals and pesticides are significantly more toxic to flora and fauna than they are to humans (e.g., copper and nickel). Chemicals that easily leach from soils (e.g., MTBE) may pose a threat to shallow groundwater

resources even though direct exposure to the soils does not pose a significant health risk. Since the CHHSLs do not address impacts to groundwater, surface water or sediment, these and other potential environmental concerns should be addressed as part of a comprehensive environmental risk assessment.

2.4 Screening For Soil Direct-Exposure Concerns

The soil screening levels presented in Table 1 address potential exposure of humans to contaminants in soil through incidental soil ingestion, dermal absorption and inhalation of dust or vapors in outdoor air. These soil screening levels are given in milligrams (mg) of chemical per kilogram (kg) of dry soil. Therefore, the analytical laboratory must be instructed to report their results accordingly. Models and assumptions used to develop the soil CHHSLs are summarized in Appendix 1. The CHHSLs represent a combination of standard assumptions regarding exposure of residents and workers to contaminants in soil and outdoor air and toxicity factors for each of the specific chemicals listed. CHHSLs for chemicals that are known or suspected carcinogens were calculated using a target excess lifetime cancer risk of one-in-one-million (10^{-6}). A target hazard quotient of 1.0 was used to calculate CHHSLS for noncancer health effects.

The presence of a chemical in soil at concentrations below its corresponding CHHSL can be assumed to not pose a significant health risk to people who may live or work at the site. Since sites usually have multiple contaminants, the cumulative, or total risk and hazards posed by all the hazardous chemicals a site should also be estimated using the approach described in Section 2.8.

Residential and commercial/industrial soil CHHSLs are applicable to soils that are at the ground surface or could be brought to the ground surface at some time in the future, with subsequent potential exposure by human receptors. A depth of more than three meters (approximately 10 feet) is generally used to delineate "deep" soils that are likely to remain isolated in the subsurface versus "shallow" soils that may be exposed during future redevelopment activities (Cal/EPA 1996a). Exposure of workers to deeper soils could still occur during periodic construction and utility maintenance work. Even if deep soil contamination does not present a human health risk, the overseeing regulatory agency may require preparation of a formal land-use covenant in order to allow such contamination to remain on site.

2.4.1 Evaluating Lead

In Table 1, the Commercial/Industrial Soil CHHSL for lead is listed as 3,500 mg/kg. This number was calculated using the methods described in Appendix 1. It should be noted, however, that this screening number is above the Total Threshold Limit Concentration for lead (1,000 mg/kg) as defined in Title 22 of the California Code of Regulations. It is also above the USEPA Region IX Preliminary Remediation Goal (PRG) of 800 mg/kg for commercial land use.

OEHHA is evaluating the method it used to derive its health-based screening number for a commercial/industrial scenario. Until this evaluation is complete, the commercial/industrial Soil CHHSL for lead in Table 1 should be considered an interim value, and the overseeing regulatory agency should be consulted on the appropriate screening number to be used at a site under investigation.

2.5 Screening of Volatile Organic Chemicals

2.5.1 Soil Screening Levels for Direct Exposure Concerns

Screening levels for direct exposure to volatile organic compounds (VOCs) in soil were not developed by OEHHA and are not included in this edition of the CHHSLs document. Direct-exposure models such as those used by USEPA Region IX do not take into account the total amount (mass) of a volatile chemical that might be present at a site (refer to Appendix 2). This is important, since the direct-exposure models assume a continuous off-gassing of vapors throughout a 30-year exposure period. In addition, the models assume exposure both via inhalation of vapors emitted to outdoor air and via incidental ingestion of volatile chemicals in soil. These assumptions may be overly conservative for highly volatile chemicals that are not expected to remain at significant concentrations in the soil over time following off-gassing to the outdoor air.

Bulk soil screening levels (i.e. concentrations measured in soil) for volatile chemicals are not presented in this document. The restricted size of soil samples limits the ability to use soil data to evaluate vapor intrusion concerns except at sites with very minor releases. At sites where significant releases of volatile chemicals have occurred, the collection of soil gas data in conjunction with bulk soil data is strongly recommended. For sites characterized by only minor releases of volatile chemicals and limited impacts to soil (e.g., minor spills around the fill ports of underground storage tanks), cleanup of soils to meet direct-exposure

concerns should generally be adequate to address vapor intrusion concerns (see also Table 1).

2.5.2 Soil Gas Screening Levels for Vapor Intrusion Concerns

The indoor air and soil gas screening levels presented in Table 2 address the potential emission of volatile chemicals from contaminated soil or groundwater and subsequent intrusion into the indoor air of overlying buildings. A full discussion of the development of the soil gas screening levels, and the models and assumptions used, is discussed in Appendix 1.

The soil gas CHHSLs for the intrusion of vapors into buildings were developed assuming that buildings have a "slab on grade" construction. The screening levels are also considered to be adequately conservative for buildings with crawl space or underground parking construction. These reflect the most common type of building designs in California. The soil gas screening levels may not be adequately conservative for estimating impacts to indoor air in structures with basements, however, or buildings with substandard ventilation systems in general. Field data suggest that attenuation of vapors in such scenarios may be an order of magnitude below that expected in rooms or buildings with normal ventilation systems. Therefore, at sites where significant vapor intrusion concerns may exist, the collection and evaluation of samples from both basement areas and overlying living spaces may be warranted.

Additional information on subsurface vapor intrusion into buildings is provided the USEPA document *User's Guide for the Johnson and Ettinger (1991) Model for Subsurface Vapor Intrusion into Buildings* (USEPA 2003) and in the following section.

2.5.3 Evaluating Vapor Intrusion Concerns

If the concentration of a volatile chemical in soil gas at a site exceeds its CHHSL, the exposure pathway of soil vapor intrusion into indoor air should be further evaluated using the Cal/EPA *Guidance for the Evaluation and Mitigation of Subsurface Vapor Intrusion to Indoor Air* (Cal/EPA 2004b). The investigation of this pathway can be complex. The identification of sources of indoor air contaminants is often complicated by the presence of the same or similar chemicals products found and used in many households and industrial buildings (e.g., aerosol sprays, dry-cleaned clothing, cleaners, and tobacco smoke). Elevated levels of the same chemicals in ambient, outdoor air also pose a

problem. Plumes of groundwater contaminated with volatile chemicals can also serve as the source of volatile chemicals found in soil gas and extend over significant areas. If there is strong evidence that the intrusion of vapors into buildings may exceed levels of potential concern, the collection and analysis of indoor air samples may be necessary. The inevitable effect of indoor air studies on the personal lives of residents and building workers will further require that risk issues be carefully communicated.

Guidance on the collection of soil gas and indoor air samples is provided in the following documents, among other sources:

- *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.

- *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.

Properly collected indoor air sample data may be compared to the indoor air screening levels. Averaging of indoor air data within a single building may not be appropriate beyond the specific room being tested. Screening levels for indoor air (Table 2) are based on standard exposure models for long-term inhalation of contaminants in air at a target excess cancer risk of 10^{-6} and a target hazard quotient of 1.0. The indoor air CHHSLs do not account for potential cumulative effects posed by the presence of multiple contaminants in air (see Section 2.8).

2.6 Substitution of Laboratory Reporting Limits for CHHSLs

The overseeing regulatory agency should review and agree to the analytical methods used to quantify chemicals in soil samples to make sure that the methods are sensitive enough to detect low concentrations of chemicals of potential concern. The attainment of detection limits that are at or below the screening levels should be part of the Data Quality Objectives. If all agreed-upon methods have been used, the overseeing regulatory agency may allow the use of the method reporting limit in place of the screening level in cases where a CHHSL for a specific chemical is less than its laboratory method reporting limit. Potential

examples include the soil direct-exposure CHHSL for dioxin (e.g., 0.0000046 mg/kg for residential exposure).

2.7 Substitution of Naturally Occurring Concentrations for CHHSLs

Naturally occurring background concentrations of arsenic, beryllium, cadmium, chromium and other metals in soils may exceed their respective soil CHHSLs. Cal/EPA generally does not require cleanup of soil to below background levels. This issue is frequently encountered with arsenic. Natural background concentrations of arsenic in California are often well above the health-based, direct-exposure goals in soil of 0.07 mg/kg for residential land use and 0.24 mg/kg for commercial/industrial land use (e.g., Bradford et. al, 1996; LBNL 2002). Background concentration of arsenic or other metals of potential concern at a site should be determined from analysis of site-specific samples in uncontaminated areas using guidance published by Cal/EPA and/or reference to published data for nearby sites (Cal/EPA 1997). However, background data for nearby sites may only be used as a surrogate for uncontaminated site data if those data are obtained from soil of the same lithology as that found on-site.

2.8 Cumulative Risks at Sites with Multiple Contaminants

Risks posed by exposure to multiple chemicals with similar health affects are considered to be additive or "cumulative." For example, the total excess lifetime risk of cancer posed by the presence of several carcinogenic chemicals in all exposure media is the sum of the risk posed by each individual chemical. The same is true for chemicals that cause noncarcingenic health effects.

A stepwise approach for screening of sites with multiple contaminants is suggested (after USEPA 2004):

Step 1: Identify potential chemicals of concern.

Step 2: Record CHHSLs for each chemical separated by media type (soil, soil gas and/or indoor air). Include CHHSLs for both cancer and noncancer effects, if available (refer to Appendix 1). If CHHSLs are not available for specific chemicals, evaluate those chemicals using the approaches discussed in Appendix 1 and in the PEA manual.

Step 3: Calculate cumulative cancer risk estimates by taking the assumed exposure point concentration for each chemical (maximum or approved 95% UCL) and divide by the respective CHHSL concentration designated for cancer evaluation. Multiply the ratio by 10^{-6} (the target risk used to develop the CHHSLs) to calculate the estimated cancer risk for that specific chemical for a reasonable maximum exposure (RME).

$$Risk = [\left(\frac{conc_x}{CHHSL_x}\right) + \left(\frac{conc_y}{CHHSL_y}\right) + \left(\frac{conc_z}{CHHSL_z}\right)] \times 10E-06$$

For multiple chemicals, simply add the risks for individual chemicals or sum individual ratios and multiply the total by a factor of 10^{-6}:

Step 4: Calculate cumulative noncancer hazard estimates by taking the assumed exposure point concentration for each chemical (maximum or approved 95% UCL) and divide by the respective CHHSL concentration designated for noncancer effects. This generates an individual Hazard Quotient for that chemical. Calculate a cumulative Hazard Index by adding the individual Hazard Quotients. A Hazard Index of one or less is generally considered "safe". A ratio that is greater than one suggests that further evaluation is necessary. (Note that carcinogens may have CHHSLs for both cancer effects as well as noncancer effects. Refer to Appendix 1).

For more information, refer to the USEPA Preliminary Remediation Goals

$$Hazard\,Index = [\left(\frac{conc_x}{CHHSL_x}\right) + \left(\frac{conc_y}{CHHSL_y}\right) + \left(\frac{conc_z}{CHHSL_z}\right)]$$

document (USEPA 2002). OEHHA has also developed a spread sheet tool for calculating cumulative risk. This spread sheet is available on Cal/EPA's, DTSC's, the State Board's and OEHHA's web pages.

2.9 Evaluation of School Sites

DTSC's Schools Property Evaluation and Cleanup Division is the lead agency for the environmental assessment of potential contamination at new, expanding, or existing schools. Since January 2000, school districts have been required to conduct an environmental assessment under the oversight and approval of DTSC prior to the construction of new schools. By law, DTSC uses specific guidance and protocols for school projects. Because of this, the CHHSLs may not be applicable for these sites. Contact DTSC for further information and direction for

the evaluation of potential contamination on school properties and the application of the CHHSLs.

2.10 Use of CHHSLs as Cleanup Levels and Land Use Restrictions

As stated earlier in this guidance, these CHHSLs are not stand-alone decision making tools, a set of final cleanup or action levels to be applied at contaminated sites or a guarantee that an oversight regulatory agency will determine that a project is adequately studied or agree with the conclusions of the site investigation and risk assessment report. Cleanup decisions are at the discretion of the overseeing regulatory agency and can only be made after a full evaluation of site conditions and potential human health and environmental concerns.

While regulatory agencies cannot be compelled to use the CHHSLs as final cleanup standards for a contaminated property, there may be circumstances where the residential CHHSLS would be sufficiently protective and considered as appropriate cleanup levels with the following caveats.

- The overseeing regulatory agency has determined that the site has been adequately characterized and agrees that the use of CHHSLs is appropriate.

- The potentially complete exposure pathways at the site match the exposure pathways used to develop the CHHSLs and no additional completed exposure pathways or receptors were identified.

- All other environmental concerns have been addressed to the satisfaction of the overseeing regulatory agency (refer to Section 1.4 and Table 1).

In a similar manner, there may be circumstances where the Commercial/Industrial CHHSLS would be sufficiently protective and considered as appropriate cleanup goals under regulatory agency oversight. Their use at a site in this context must also be coupled with the understanding that such a use of these CHHSLs may be subject to existing regulations and land-use covenants. In addition, the following should also be considered:

- **Concentrations of chemicals in soils left in place at a commercial/industrial site should always be compared to both commercial/industrial AND residential CHHSLs.** If the soils meet

CHHSLs for residential land use after cleanup then this should be clearly stated in the site closure report. This point may prove important should the site unexpectedly become desirable for other uses in the future (e.g., residential, day care, health care, etc.).

- Sites cleaned up to commercial CHHSLs only are not suitable for unrestricted land use without further evaluation. The appropriate regulatory agency should be consulted to determine actions necessary to remove land-use restrictions.

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3 Conditions Warranting Site Specific Human Health Risk Assessments

3.1 Site Considerations

Use of the CHHSLs is optional and a standard human health risk assessment may be undertaken for any site. Site conditions may prevent the full use of the CHHSLs and require preparation of a more site-specific, health risk evaluation or baseline risk assessment (refer to Section 1.2). Examples of site conditions that may warrant site-specific or detailed human health risk assessment include:

- Sites that have a high public profile and need a detailed, fully documented human health risk assessment for public review;
- Sites where multiple contaminants with similar health effects are present and cumulative health risks (or hazards) must be calculated;
- Sites with contaminants for which CHHSLs have not been developed.
- Sites where alternative target risk levels or chemical-specific toxicity factors may be acceptable to the regulatory agency (Appendix 1);
- Sites where direct-exposure concerns for residents and workers may not need to be considered (Section 2.4);
- Sites where site conditions may be engineered to eliminate or reduce specific exposure pathways;
- Sites where field observations or site conditions indicate that the CHHSLs may not be adequately protective or may be excessively conservative.

Additional considerations should be evaluated on a site-by-site basis and discussed with the overseeing regulatory agency.

3.2 Tier 2 Human Health Risk Assessments

3.2.1 Purpose

The Tier 1 CHHSLs were developed with default or generic assumptions that are not specific to any particular site condition. If site soil concentrations exceed CHHSLs, site-specific exposure assumptions may be used in the standard risk models described in Appendix 1 or the PEA guidance to estimate risk and/or develop site-specific CHHSLs. Using alternative exposure assumptions in these standard risk models could reduce the time and cost incurred by both the regulated business and the overseeing responsible party in finalizing the risk assessment. Modifications to the default assumptions must be described and justified in the text of the report, presented with the revised set of screening or cleanup levels, and agreed to beforehand with the regulatory agency.

3.2.2 Examples of Site-Specific Adjustments

Potential site-specific modifications include:

- Use of alternative target risk levels, and/or alternative exposure assumptions;
- Elimination of direct-exposure concerns through imposition of institutional controls;
- Inclusion of potential exposure of construction and trench workers to contaminated soil not likely to be exposed at the ground surface in the future (e.g., capped soils or soils isolated at depth);
- Consideration of method reporting limits or natural background or ambient concentrations of a chemical in place of the CHHSL.

After incorporating site-specific parameter values into the Tier 1 direct-exposure models, alternative human-health-based screening levels can be calculated and re-compared to site data.

3.3 Tier 3 (Baseline) Human Health Risk Assessments

3.3.1 Purpose

In a site-specific baseline human health risk assessment, alternative models and assumptions are used and fully justified to develop a detailed, comprehensive

human health risk assessment. Portions of the models and assumptions used to develop the CHHSLs may still be retained for some components of the risk assessment. Any baseline human health risk assessment should be carried out under the oversight of the regulatory agency.

Detailed guidance on the preparation of and information for use in site-specific baseline environmental risk assessments is provided in the following references:

Human Health Risk Assessment:

- *Risk Assessment Guidance for Superfund. Volume I, Human Health Evaluation Manual* (Part A) (USEPA 1989a);
- *Soil Screening Guidance: Technical Background Document* (USEPA 1996);
- *CalTOX, A Multimedia Total Exposure Model For Hazardous-Waste Sites* (Cal/EPA 1994a);
- *Preliminary Endangerment Assessment Guidance Manual* (Cal/EPA 1994b);
- *Supplemental Guidance For Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities* (Cal/EPA 1996a);
- *Exposure Factors Handbook* (USEPA 1997a); and
- *Assessing the Significance of Subsurface Contaminant Vapor Migration to Enclosed Spaces* (Johnson et. al, 1998).

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4 Evaluation of Other Potential Environmental Concerns

The importance of identifying all environmental concerns at sites where releases of hazardous chemicals have occurred is discussed in Sections 1.4 and 2.2. The CHHSLs provided in Tables 1 and 2 specifically address risks to human health posed by exposure to contaminated soil and indoor air. At sites affected by highly toxic but relatively immobile chemicals (e.g., PCBs, DDT, arsenic, etc.), cleanup of contaminated soils to address human health concerns will generally be sufficient to address other potential environmental concerns provided that sensitive ecological habitats are not threatened. In other cases or for other chemicals, additional environmental concerns may still be present even after impacted soils have been remediated to levels sufficient to address risks to human health. This could include leaching of contaminants from soil and subsequent impacts on groundwater resources, toxicity to terrestrial biota, uptake of contaminants in edible fruits or vegetables and nuisance or gross contamination concerns.

A summary of other environmental concerns potentially posed by contaminants in soil is incorporated into Table 1. This summary compares the CHHSLs to the SFBRWQCB's ESLs for leaching, ecotoxicity and nuisance concerns. The ESLs can be found at http://www.waterboards.ca.gov/sanfranciscobay/esl.htm.

For example, the residential CHHSL for endrin in soil (21 mg/kg) is much higher than the corresponding ESL for ecotoxicity concerns (0.06 mg/kg). This means that ecotoxicity concerns may outweigh human health concerns at sites where potentially sensitive habitats are present (designated by an "X" in the Table 1). This is not surprising, since endrin, a pesticide, was specifically formulated to be highly toxic to terrestrial biota.

Additional evaluation should be carried out at sites where the basic conceptual site model indicates that the presence of contaminated soils may pose other environmental concerns or where potential impacts to groundwater, surface water or sediment are identified. It is beyond the scope of this document to present guidance on the proper evaluation of these additional concerns. However, useful references are provided in Figure 4-1. Additional risk assessment guidance should be consulted as needed.

5 References

Bradford, G. R., et. al, 1996, *Background Concentrations of Trace and Major Elements in California Soils*: University of California (Riverside), Division of Agriculture and Natural Resources, March 1996.

Cal/EPA, 1994a, *CalTOX, A Multimedia Total Exposure Model For Hazardous-Waste Sites*: California Environmental Protection Agency, Department of Toxics Substances Control, Version 1.5 (and updates), www.dtsc.ca.gov/ScienceTechnology/index.html.

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FIGURES

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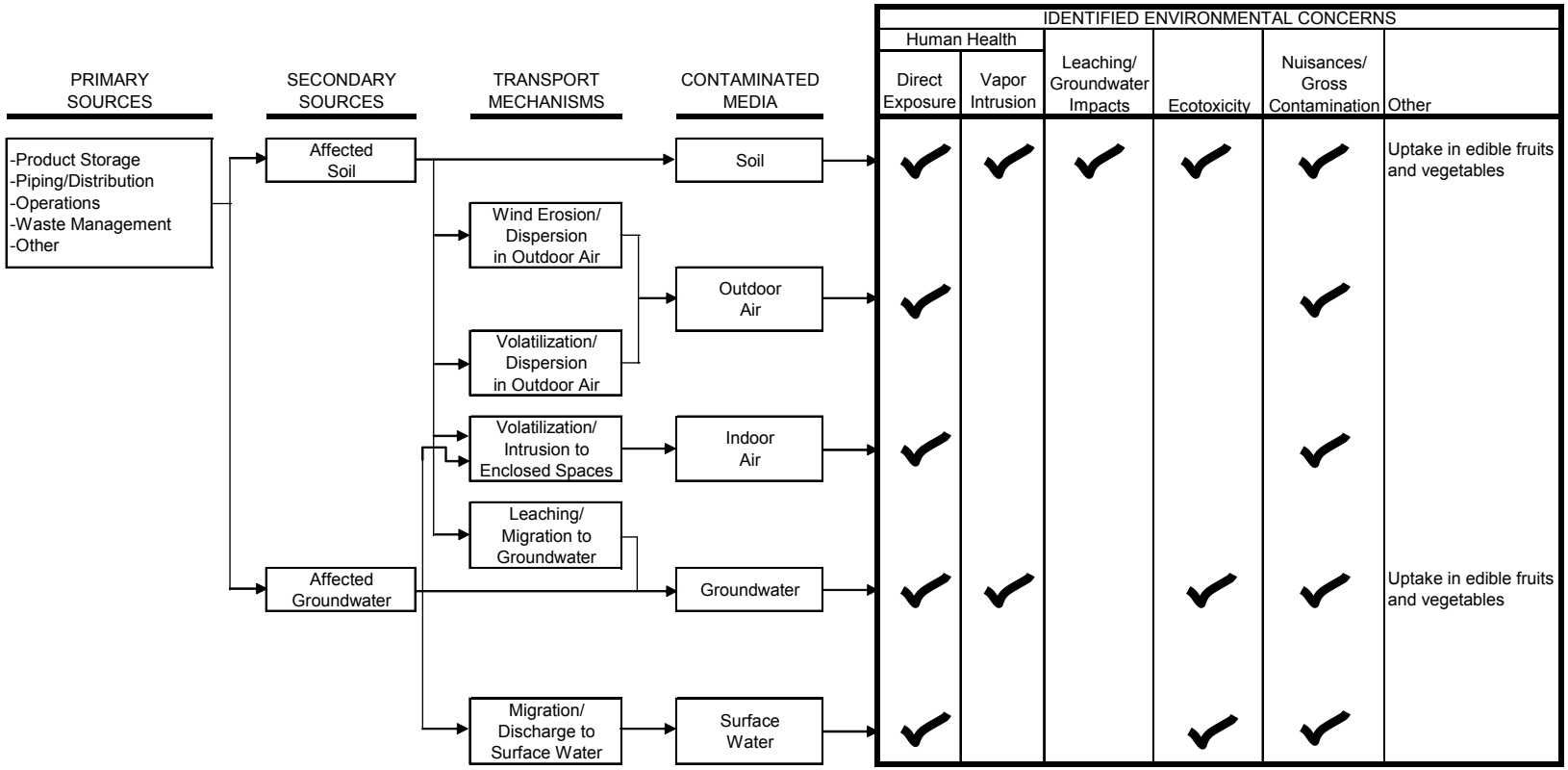

Figure 2-1. Example conceptual site model depicting environmental concerns identified at a site where hazardous chemicals were released to soil and groundwater. See Section 2.2.

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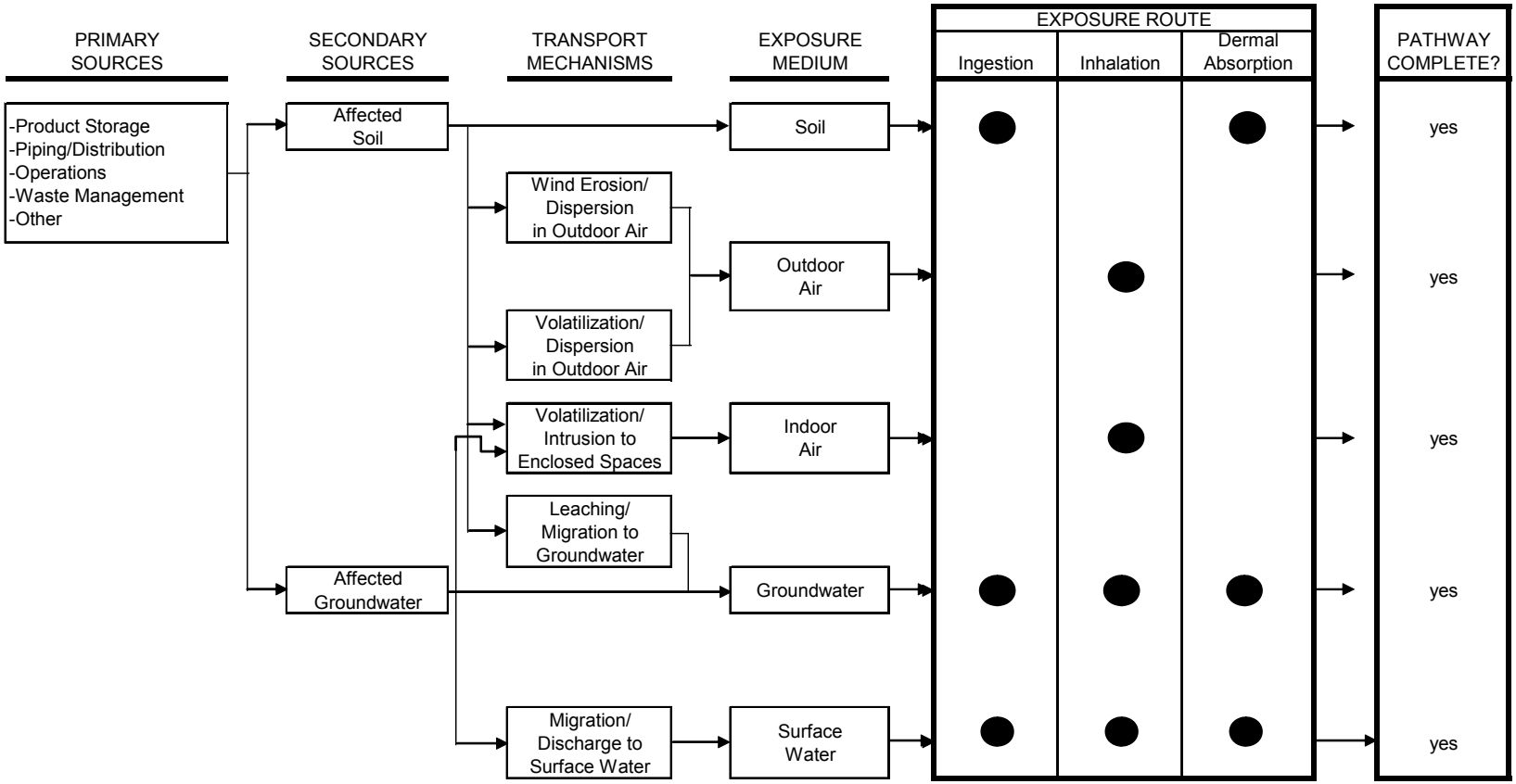

Figure 2-2. Example focused conceptual site model of human health concerns identified at a site where hazardous chemicals were released to soil and groundwater. See Section 2.2.

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Environmental Concern	Reference/Website
Leaching and migration of contaminants to groundwater	USEPA Soil Screening Guidance (USEPA 1996): http://www.epa.gov/superfund/resources/soil/index.htm SFBRWQCB ESL Document (SFBRWQCB 2003): http://www.waterboards.ca.gov/sanfranciscobay/esl.htm. USEPA Synthetic Precipitation Leaching Procedure (USEPA 1994): http://www.epa.gov/epaoswer/hazwaste/test/main.htm. Commonly Used Models: SESOIL, VLEACH
Ecotoxicity	USEPA Ecological Soil Screening Guidance (USEPA 1996): http://www.epa.gov/superfund/programs/risk/ecorisk/ecossl.htm Risk Assessment Guidance for Superfund: Volume II Environmental Evaluation Manual (USEPA 1989b); Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments (USEPA 1997b) Guidance for Ecological Risk Assessments at Hazardous Waste Sites and Permitted Facilities (CalEPA 1996a,b) Ontario MOEE Rational for the Development and Application of Generic Soil, Groundwater and Sediment Criteria for Use at Contaminated Sites in Ontario (MOEE 1996): http://www.ene.gov.on.ca/ SFBRWQCB ESL Document (SFBRWQCB 2003): http://www.waterboards.ca.gov/sanfranciscobay/esl.htm NOAA Sediment Screening Table (NOAA 1999): http://response.restoration.noaa.gov/cpr/sediment/squirt/squirt.html
Ingestion via plant uptake	USEPA Soil Screening Guidance (USEPA 1996): http://www.epa.gov/superfund/resources/soil/index.htm USEPA Fertilizer Risk Assessment (USEPA 1999): http://www.epa.gov/epaoswer/hazwaste/recycle/fertiliz/risk/ CalEPA CALTOX model (CalEPA 1994a): http://www.dtsc.ca.gov/ Massachusetts DEP Guidance for Disposal Site Risk Characterization (MADEP 1995): http://www.state.ma.us/dep/ors/orspubs.htm
Nuisance/Gross Contamination	Massachuestts DEP Background Documentation for the Development of the MCP Numerical Standards (MADEP 1994): http://www.state.ma.us/dep/ors/orspubs.htm SFBRWQCB ESL Document (SFBRWQCB 2003): http://www.waterboards.ca.gov/sanfranciscobay/esl.htm

Figure 4-1. Suggested references for evaluation of environmental concerns not currently addressed by the CalEPA CHHSLs.

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TABLE 1: California Human Health Screening Levels for Soil and Comparison to Other Potential Environmental Concerns

Notes:
Always compare soil data for commercial/industrial sites to residential CHHSLs and evaluate need for formal land-use restrictions (see Section 2.10).

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Table 1. California Human Health Screening Levels for Soil And Comparison To Other Potential Environmental Concerns

Chemical	[1]Soil Human Health Screening Levels (mg/kg of dry soil)		[2]Other Potential Environmental Concerns Posed By Contaminated Soil			
	Residential Land Use	Commercial/ Industrial Land Use Only	[3]Leaching	[4]Ecotoxicity	[5]Nuisance/ Aesthetic Concerns	[6]Other
Organic Acidic Chemicals						
2,4-D	6.9E+02	7.7E+03	X	X	o	
2,4,5-T	5.5E+02	6.1E+03	X	X	o	
Pentachlorophenol	4.4E+00	1.3E+01	X	X	o	
Organic Neutral Chemicals						
Aldrin	3.3E-02	1.3E-01	o	X	o	
Benzo(a)pyrene	3.8E-02	1.3E-01	o	X	o	TPH
Chlordane	4.3E-01	1.7E+00	o	X	o	
DDD	2.3E+00	9.0E+00	o	X	o	
DDE	1.6E+00	6.3E+00	o	X	o	
DDT	1.6E+00	6.3E+00	o	X	o	
Dieldrin	3.5E-02	1.3E-01	X	X	o	
1,4 Dioxane	1.8E+01	6.4E+01	X	o	o	
Dioxin (2,3,7,8-TCDD)	4.6E-06	1.9E-05	o	o	o	
Endrin	2.1E+01	2.3E+02	X	X	o	
Heptachlor	1.3E-01	5.2E-01	X	X	o	
Lindane	5.0E-01	2.0E+00	X	X	o	
Kepone	3.5E-02	1.3E-01	X	o	o	
Methoxychlor	3.4E+02	3.8E+03	o	X	o	
Mirex	3.1E-02	1.2E-01	X	X	o	
PCBs	8.9E-02	3.0E-01	o	X	o	
Toxaphene	4.6E-01	1.8E+00	X	X	o	

Table 1. California Human Health Screening Levels for Soil And Comparison To Other Potential Environmental Concerns

Chemical	[1]Soil Human Health Screening Levels (mg/kg of dry soil)		[2]Other Potential Environmental Concerns Posed By Contaminated Soil			
	Residential Land Use	Commercial/ Industrial Land Use Only	[3]Leaching	[4]Ecotoxicity	[5]Nuisance/ Aesthetic Concerns	[6]Other
Inorganic Chemicals						
Antimony and compounds	3.0E+01	3.8E+02	site specific	o	o	
Arsenic	7.0E-02	2.4E-01	site specific	X	o	Ambient background
Barium and compounds	5.2E+03	6.3E+04	site specific	X	o	Construction workers
Beryllium and compounds	1.5E+02	1.7E+03	site specific	X	o	
Beryllium oxide[7]	9.1E-02	4.1E-01	o	o	o	Construction workers
Beryllium sulfate[7]	2.1E-04	9.5E-04	o	o	o	
Cadmium and compounds	1.7E+00	7.5E+00	site specific	X	o	Ambient background
Chromium III	1.0E+05	1.0E+05	site specific	X	X	
Chromium VI	1.7E+01	3.7E+01	site specific	X	o	Construction workers
Cobalt	6.6E+02	3.2E+03	site specific	X	o	Construction workers
Copper and compounds	3.0E+03	3.8E+04	site specific	X	X	
Fluoride	4.6E+03	5.7E+04	site specific	o	o	
Lead and lead compounds	1.5E+02	3.5E+03[9]	site specific	X	o	Uptake in fruits and vegetables
Lead acetate[7]	2.3E+00	1.0E+01	X	o	o	
Mercury and compounds	1.8E+01	1.8E+02	site specific	X	o	
Molybdenum	3.8E+02	4.8E+03	site specific	X	X	
Nickel and compounds	1.6E+03	1.6E+04	site specific	X	X	Construction workers
Nickel subsulfide[7]	3.8E-01	1.1E+04	site specific	o	o	
Perchlorate[8]	pp[8]	pp[8]	X	o	o	
Selenium	3.8E+02	4.8E+03	site specific	X	X	
Silver and compounds	3.8E+02	4.8E+03	site specific	X	X	
Thallium and compounds	5.0E+00	6.3E+01	site specific	o	o	Ambient background
Vanadium and compounds	5.3E+02	6.7E+03	site specific	X	X	

Table 1. California Human Health Screening Levels for Soil And Comparison To Other Potential Environmental Concerns

Chemical	[1]Soil Human Health Screening Levels (mg/kg of dry soil)		[2]Other Potential Environmental Concerns Posed By Contaminated Soil			
	Residential Land Use	Commercial/ Industrial Land Use Only	[3]Leaching	[4]Ecotoxicity	[5]Nuisance/ Aesthetic Concerns	[6]Other
Zinc	2.3E+04	1.0E+05	site specific	X	X	

Notes:

1. Direct-exposure screening levels address human exposure to chemicals in soil via incidental ingestion, dermal absorption and inhalation of vapors and particulates emitted to outdoor air (refer to Appendix 1). Assumes impacted soil is situated at or near the ground surface or could be at some time in the future. Volatile chemicals not included at this time (refer to Section 2.5).
   "Residential Land Use" screening levels generally considered appropriate for other sensitive uses (e.g., day-care centers, hospitals, etc.).
   Commercial/industrial properties should be evaluated using both residential and commercial/industrial CHHSLs. A deed restriction that prohibits use of the property for sensitive purposes may be required at sites that are evaluated and/or remediated under a commercial/industrial land use scenario only.
   Carcinogens: CHHSLs based on target cancer risk of 10^{-6}. Cal/EPA cancer slope factors used when available.
   Noncarcinogens: CHHSLs based on target hazard quotient of 1.0.
   Calculation of cumulative risk may be required at sites where multiple contaminants with similar health effects are present (see Section 2.8).
   Residential and C/I soil CHHSLs for arsenic below background for most sites in California (0.07 mg/kg and 0.24 mg/kg, respectively - see Appendix 1). Use identified or anticipated background as screening level (see Section 2.7).
2. Environmental concerns in addition to direct exposure that may need to be considered in evaluation of contaminated soil. Based on a comparison of soil CHHSLs to soil screening levels for noted concerns compiled by the San Francisco Bay Regional Water Quality Control Board (SFBRWQCB 2003). The need to address other environmental concerns must be evaluated separately in coordination with the lead regulatory agency (See Sections 1.4, 2.2 and Chapter 4).
   "X": Noted concern may outweigh direct-exposure risks at many sites and drive decisions for cleanup actions.
   "o": Potential concern but generally will be addressed if cleanup of contaminated soils to meet direct-exposure CHHSLs is carried out.
   "site specific": Potential concern, but evaluation as to whether this factor is a potential concern must be done on a site specific basis.
3. Leaching of chemicals from soil and subsequent impacts to groundwater. Soil ESLs consider of impacts to drinking water resources, re-emission of volatile chemicals from groundwater into overlying buildings and discharges of contaminated groundwater to surface water. Leaching of metals from soil should be evaluated on a site-specific basis, depending on the potential mobility of the metal species present. Laboratory-based leaching studies are generally preferred over model-derived screening levels.
4. Toxicity to terrestrial flora and fauna. Need to consider ecotoxicity concerns generally determined on a site-by-site basis.
5. Nuisance and gross contamination concerns address odors and aesthetic concerns as well as general resource degradation and presence of potentially mobile free product.
6. Other pertinent environmental concerns and considerations as determined on a site-specific basis.
   Health risk to construction workers may outweigh risk to residents or commercial/industrial workers for chemicals that are carcinogenic due to increased exposure to airborne dust particles and incidental ingestion of soil. Uptake of chemicals in edible fruits and vegetables from soil may need to be considered in some cases for noted chemicals.
7. These metal salts are significantly (greater than 10-fold) more toxic than the values for the metals in general. If it is known that this chemical was used at the site, the screening number for this chemical should be used instead of the screening number for the metal and its compounds.
8. Calculation of a screening number for the chemical has been postponed (pp) until the toxicity criterion currently being developed by OEHHA is published as a final document.
9. This screening number is above the Total Threshold Limit Concentration for lead of 1000 mg/kg, as defined in Title 22, California Code of Regulations. It is also above the US EPA Region IX PRG of 800 mg/kg.

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TABLE 2: California Human Health Screening Levels for Indoor Air and Soil Gas

Notes:
Always compare soil data for commercial/industrial sites to residential CHHSLs and evaluate need for formal land-use restrictions (see Section 2.10).

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Table 2. California Human Health Screening Levels for Indoor Air and Soil Gas

Chemical	[1]Indoor Air Human Health Screening Levels ($\mu g/m^3$)		[2]Shallow Soil Gas Human Health Screening Levels (Vapor Intrusion) ($\mu g/m^3$)	
	Residential Land Use	Commercial/ Industrial Land Use Only	Residential Land Use	Commercial/ Industrial Land Use Only
Benzene	8.40 E-02	1.41 E-01	3.62 E+01	1.22 E+02
Carbon Tetrachloride	5.79 E-02	9.73 E-02	2.51 E+01	8.46 E+01
1,2-Dichloroethane	1.16 E-01	1.95 E-01	4.96 E+01	1.67 E+02
cis-1,2-Dichloroethylene	3.65 E+01	5.11 E+01	1.59 E+04	4.44 E+04
trans-1,2-Dichloroethylene	7.30 E+01	1.02 E+02	3.19 E+04	8.87 E+04
Ethylbenzene	Postponed[3]	Postponed[3]	Postponed[3]	Postponed[3]
Mercury, elemental	9.40 E-02	1.31 E-01	4.45 E+01	1.25 E+02
Methyl tert-Butyl Ether	9.35 E+00	1.57 E+01	4.00 E+03	1.34 E+04
Naphthalene	7.20 E-02	1.20 E-01	3.19 E+01	1.06 E+02
Tetrachloroethylene	4.12 E-01	6.93 E-01	1.80 E+02	6.03 E+02
Tetraethyl Lead	3.65 E-04	5.11 E-04	2.06 E-01	5.78 E-01
Toluene	3.13 E+02	4.38 E+02	1.35 E+05	3.78 E+05
1,1,1-Trichloroethane	2.29 E+03	3.21 E+03	9.91 E+05	2.79 E+06
Trichloroethylene	1.22 E+00	2.04 E+00	5.28 E+02	1.77 E+03
Vinyl Chloride	3.11 E-02	5.24 E-02	1.33 E+01	4.48 E+01
m-Xylene	7.30 E+02	1.02 E+03	3.19 E+05	8.87 E+05
o-Xylene	7.30 E+02	1.02 E+03	3.15 E+05[4]	8.79 E+05[4]
p-Xylene	7.30 E+02	1.02 E+03	3.17 E+05	8.87 E+05

Reference: Appendix 1, OEHHA Target Indoor Air Concentrations and Soil-Gas Screening Numbers for Existing Buildings under Residential and Industrial/Commercial land uses.

Notes:

1. "Residential Land Use" screening levels generally considered adequate for other sensitive uses (e.g., day-care centers, hospitals, etc.). Commercial/industrial properties should be evaluated using both residential and commercial/industrial CHHSLs. A deed restriction that prohibits use of the property for sensitive purposes may be required at sites that are evaluated and/or remediated under a commercial/industrial land use scenario only.
Calculation of cumulative risk may be required at sites where multiple contaminants with similar health effects are present.
Carcinogens: CHHSLS based on target cancer risk of 10-6. Cal/EPA cancer slope factors used when available.
Noncarcinogens: CHHSLS based on target hazard quotient of 1.0.
2. Soil Gas: Screening levels based on soil gas data collected <1.5 meters (five feet) below a building foundation or the ground surface. Intended for evaluation of potential vapor intrusion into buildings and subsequent impacts to indoor-air. Soil gas data should be collected and evaluated at all sites with significant areas of VOC-impacted soil. Screening levels also apply to sites that overlie plumes of VOC-impacted groundwater.
3. Calculation of a screening number for the chemical has been postponed (pp) until the toxicity criterion currently being developed by OEHHA is published as a final document.
4. Representative Screening Numbers for mixed xylenes. The representative value for mixed xylenes is based on the calculated lowest one amongst the three isomers.

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Appendix 1: Human-Exposure-Based Screening Numbers Developed To Aid Estimation of Cleanup Costs for Contaminated Soil

OEHHA (November 2004)

(Revised January 2005)

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APPENDIX 2: Comparison of CHHSLs to Existing Screening Levels and Standards

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Comparison of CHHSLs to Existing Screening Levels and Standards

The U.S. Environmental Protection Agency Region IX office in San Francisco publishes "Preliminary Remediation Goals (PRGs)" for soil, drinking water and ambient air with a focus on risks to human health (USEPA 2004). The San Francisco Bay Area Regional Water Quality Control Board (SFBRWQCB) publishes Environmental Screening Levels (ESLs) for soil, groundwater, surface water and air that provide screening levels for other common environmental concerns as well (SFBRWQCB 2003).

Methods used by the USEPA and the SFBRWQCB to assess potential human exposure to contaminants in soil and air are very similar. The resulting screening levels are therefore almost identical. Similarities and differences between the CHHSLs and these suites of screening levels are summarized below. In addition, federal and state agencies publish screening levels or regulatory standards for hazardous waste that are sometimes confused with environmental screening levels. The applicability of these criteria to contaminated sites is also briefly described.

USEPA Region IX PRGs

The USEPA Region IX "Preliminary Remediation Goals" or "PRGs" address the direct exposure of residents and commercial workers to contaminants found in soil, drinking water and air (USEPA 2004). These PRGs may be found at http://www.epa.gov/region09/waste/sfund/prg/index.htm. Equations and assumptions used to develop the PRGs are consistent with the human health risk assessment guidance prepared by Cal/EPA, including the CalTOX model (Cal/EPA 1994a) and the *Preliminary Endangerment Assessment Guidance Manual* (Cal/EPA 1994b) and *Supplemental Guidance for Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities* (Cal/EPA 1996a).

The USEPA approach for developing the PRGs was adopted to develop the CHHSLs with minor modifications. The CHHSLs are an adjustment of soil and ambient air PRGs by using Cal/EPA-specific toxicity factors. For the majority of the chemicals listed, Cal/EPA toxicity factors are slightly more stringent or equal to those used by the USEPA to develop the PRGs. Some CHHSLs are significantly more restrictive.

A detailed discussion of the USEPA Region IX PRGs models is provided in Appendix 1. 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" and cannot be used

as a stand-alone tool for the evaluation of contaminated sites (USEPA 2004). The same is true for the CHHSLs.

USEPA Soil Screening Levels

The USEPA Office of Emergency and Remedial Response document *Soil Screening Guidance: Technical Background Document* presents methodologies and related soil screening levels for evaluation of direct-exposure concerns, leaching of contaminants from soil and subsequent impacts to groundwater, uptake of contaminants into plants and the intrusion of volatile chemicals into buildings (USEPA 1996). Although subsequent guidance documents on specific topics have since been prepared by USEPA and other agencies (USEPA PRGs, USEPA vapor intrusion guidance document, etc.), the Soil Screening Guidance nonetheless provides a valuable resource for evaluation of these environmental concerns.

Soil screening levels for direct exposure concerns are based on USEPA toxicity factors and similar exposure models used to develop the USEPA Region IX PRGs and the Cal/EPA CHHSLs. Screening levels are presented for specific pathways (e.g., ingestion, inhalation of outdoor air, etc.), rather than for combined exposure routes as now presented in the PRGs and the CHHSLs. Dermal absorption was not considered in calculation of the direct-exposure screening levels. This pathway was included in calculation of the PRGs and CHHSLs, however. The ultimate difference in screening levels is in most cases minimal.

Soil screening levels for leaching concerns are based on a simplistic contaminant equilibrium partitioning model. The model uses USEPA maximum contaminant levels (MCLs) for drinking water as target groundwater impact goals. Generic dilution factors of "1" and "20" are presented for mixing of leachate in groundwater and subsequent dilution of contaminant concentrations. The leaching based soil screening levels are presented in the USEPA Region IX PRG document.

The Soil Screening Guidance model does not take into account fate and transport of leachate in the vadose zone and can be excessively conservative for highly volatile or highly sorptive chemicals or for use at sites where groundwater is greater than ten meters or more below the base of contaminated soil. The document also presents leaching based screening levels for inorganic (contaminants, primarily metals). Leaching of metals from soil is highly dependent on the actual specifies of the metal present and site-specific soil factors. Laboratory-based studies are generally preferable over model-based approaches for evaluation of leaching of metals and other inorganic chemicals from soil.

The uptake of contaminants in edible plants is briefly discussed in the Soil Screening Guidance document. Screening levels are presented for a limited number of inorganic contaminants. The report concludes that uptake of contaminants into plants may be of particular concern for arsenic and cadmium. With the exception of these compounds, the report notes that inorganic contaminants in soil are likely to be toxic to the plants themselves at levels far lower than would be of concern for uptake and consumption of the plants by humans. (DTSC also considers the uptake of lead in edible plants. Refer to Table 1 of the main document).

A brief discussion of the Johnson and Ettinger model for vapor intrusion from contaminated soils into buildings is provided in the Soil Screening Guidance document. Soil screening levels for this concern are not presented, however, due to concerns that the soil model significantly overestimates potential impacts to indoor air. The document instead recommends that soil gas data be used to evaluate this concern, although screening levels are likewise not provided. Soil gas CHHSLs presented in Table 2 of this document reflect more up-to-date USEPA methods for evaluation of vapor intrusion concerns (see Appendix 1). The USEPA is currently developing additional guidance on this subject.

SFBRWQCB Environmental Screening Levels (ESLs)

The SFBRWQCB ESLs are a compilation of screening levels specific for use at sites overseen by that agency in the San Francisco bay area for a number of different environmental concerns, including risk to human health. The July 2003 edition (updated February 2004) of the SFBRWQCB ESLs includes screening levels for the following exposure pathways and/or environmental concerns:

Soil:

- 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) biota;
- Protection against nuisance concerns (odors, etc.) and general resource degradation;

Indoor Air:

- Protection of human health;

Shallow Soil Gas:

- Emission of subsurface vapors to building indoor air.

Similar ESLs are also provided for the environmental media of groundwater and surface water. In the ESL document, soil screening levels for individual environmental concerns are compared and the lowest of these levels (i.e., the concentration of the chemical at which all other environmental concerns would likewise be addressed) is presented in the ESL summary lookup tables.

By comparison, the CHHSLs reflect a subset of the screening levels considered in the ESL document specific to human health concerns. CHHSLs were developed for the follow concerns only:

Soil:

- Direct/indirect exposure to impacted soil (nonvolatile chemicals only - ingestion, dermal absorption, inhalation of vapors and dust in outdoor air);

Indoor Air:

- Protection of human health;

Shallow Soil Gas:

- Emission of subsurface vapors to building indoor air.

For comparative purposes, the most current ESLs may be found at http://www.waterboards.ca.gov/sanfranciscobay/esl.htm. The soil direct exposure CHHSLs and ESLs for nonvolatile chemicals and soil gas CHHSLs and ESLs for volatile chemicals are essentially identical. Soil and indoor air ESLs for human health concerns were developed by incorporating Cal/EPA toxicity factors into the USEPA PRG models for direct exposure to contaminated soil and USEPA models for the intrusion of soil gas into buildings. Since this mimics the approach used to develop the CHHSLs, the resulting screening levels are very similar.

The primary difference is the assumption in the ESL soil and indoor air screening levels for human health that up to five chemicals with similar noncancer health effects may be present at a given site. This allows potential cumulative health risks to be conservatively taken into account at most sites without requiring that the screening levels be adjusted on a site-by-site basis (see Section 2.8). This was done by simply dividing the initial screening level based on a hazard quotient of 1.0 by a factor of five (adjusting the target Hazard Quotient to 0.2). Future editions of the ESL document will directly incorporate the Cal/EPA CHHSLs for soil and indoor air as part of that document, again adjusted to address cumulative risk concerns at a Tier 1 level.

Hazardous Waste Regulations

California Total Threshold Limit Concentrations (TTLC) criteria for solids and Soluble Threshold Limit Concentration (STLC) are used to determine whether a waste is a hazardous waste (Title 22, California Code of Regulations, section 66261.24(a)(2)(A) and (B)). If a waste is determined to be a hazardous waste, specific regulations and statues regarding the management, storage, transportation and disposal must be met.

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. The TTLC for combined DDT/DDE/DDD is 1.0 mg/kg while the residential, direct-exposure soil screening for each compound ranges from 1.6 mg/kg to 2.3 mg/kg, for a sum of 5.5 mg/kg (see Table 1).

In practice, the extent of soil contaminated above 1.0 mg/kg versus 5.5 mg/kg total DDT/DDE/DDD may not be significant in the field following cleanup to the risk-based CHHSLs. However, it may be prudent to use TTLCs as final cleanup values for residential sites where the TTLC is less than cleanup values that were based on actual risk to human health and the environment. This may help to avoid potential future problems with soil management and disposal.

TSCA Cleanup Levels for PCBs

The treatment, storage and disposal of polychlorinated biphenyls (PCBs) are regulated under the federal Toxics Substance Control Act (TSCA), as described in 40 CFR Part 761 (revised 7/1/99), which is administered by the USEPA Toxics Section. If PCBs are found at a site, the regulation should be consulted to determine its applicability and to ensure that the appropriate notifications are provided to and approvals are obtained from USEPA (refer also to *Guidance on remedial Actions for Superfund Sites with PCB Contamination,* USEPA 1990). To obtain more information regarding regulations and guidance, the USEPA's PCB web page can be accessed at: http://www.epa.gov/opptintr/pcb/

Within each USEPA Region, the Regional Administrator has designated Regional PCB Coordinators to oversee the development of PCB efforts. The staff of the Region IX PCB Program is available to members of the regulated community and others who have questions concerning the manufacture, processing, distribution in commerce, use, cleanup, storage and disposal of PCBs and PCB articles. The Region IX PCB web page can be accessed at:
http://www.epa.gov/region09/toxic/pcb/index.html

USEPA Region IX staff can be contacted at:

U.S. EPA Region 9
Mail Code CMD-4-2
75 Hawthorne Street
San Francisco, CA 94105

Max Weintraub	415-947-4163	weintraub.max@epa.gov
Christopher Rollins	415-947-4166	rollins.christopher@epa.gov

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The Pennsylvania Integrated Pest Management Program /
Philadelphia School & Community IPM Partnership

Asthma, Pests, and Pesticides

Asthma
Asthma is a long-term condition causing inflammation of the lung's airways. Symptoms of asthma include wheezing, coughing, feeling of tightness in the chest, difficulty breathing, and itching neck, throat and ears. While the causes of asthma are not fully understood, a combination of genetic susceptibility and environmental factors are involved. Although we cannot control our genetic make-up, we *can* help prevent asthma attacks by paying attention to the environmental conditions that irritate lungs and set off an attack.

Why be Concerned?
Approximately 20 million Americans have asthma and it is the most common chronic childhood disease – afflicting over 6 million children nationally and over 100,000 children in Southeastern Pennsylvania. In Philadelphia, the asthma rates among school-aged children are more than twice the rates for Pennsylvania and the nation as a whole. Asthma is the leading cause of school absences. Parents, in turn, must miss work to stay home with their sick children. In Philadelphia, 16,000 children visit emergency rooms each year. African-American and Hispanic/Latino children have asthma rates 2-3 times that of white children. A bad asthma attack can be fatal.

Asthma Triggers
Asthma attacks are usually started by exposure to certain substances called ***triggers***. Triggers are either allergens or lung irritants. Airborne allergens are substances such as pollen, animal dander, cigarette smoke, aerosols, or mold that cause an allergic reaction. Chemical lung irritants include pesticides, perfumes, air fresheners and household and industrial cleaning products. Repeated exposure to allergens or irritants, such as cockroach and/or mouse allergens, can "sensitize" people - making them more likely to experience allergic reactions. Awareness of asthma triggers can help you take steps to reduce them, and thereby preventing asthma symptoms or attacks.

Pests Trigger Asthma
Pests are unwanted creatures that invade our homes. Once they have gotten inside, some of these pests, notably, mice, rats and cockroaches, can contribute to an asthma attack. In fact, research is going on to determine whether or not these pests can actually cause asthma to develop.

The **single major factor** contributing to asthma in urban-dwelling children in the Northeastern US has been found to be **exposure to cockroach allergens**. Cockroaches shed skins, leave behind feces, and when cockroaches are dead, their bodies turn into dust – all things that can trigger an asthma attack. To make matters worse, when pesticide sprays or "bug bombs" are used to combat roaches, they can also irritate lungs and potentially cause an attack. Rodents, such as

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rats and mice, can trigger asthma as well. These rodents shed dead skin cells and produce waste products that can trigger attacks if someone with asthma breathes them in.

Pesticides and Human Health

Pesticides are substances designed to kill, control or repel pests, including insects, rodents, weeds, and molds. The US Environmental Protection Agency lists pesticides as one of four environmental pollutants that may influence the induction and exacerbation of asthma symptoms. Pesticides do this by irritating the lungs as they are breathed in. In laboratory tests with animals, commonly used pesticides have been linked to cancer, birth defects, reproductive disorders, and neurological, kidney and liver damage. To be safe, it is important to limit children's exposures to toxins of all kinds, including pesticides.

What Can You Do to Safely Control Pests?

Integrated Pest Management (IPM) is an approach to pest control that focuses on *eliminating the root causes of pest problems* and *using the safest, most effective methods available* to get rid of active infestations. IPM prevents pest by using a combination of physical and chemical methods. Because **IPM focuses on prevention**, it is more effective than reactive, spray-based approach to pest control and reduces the need to use pesticides.

Pest Prevention

These methods are at the heart of an IPM program:

- **Keeping watch:** Certain areas of the house are more susceptible to pests such as the kitchen, basement or bathroom. Small sticky traps or glue boards can be used in these areas as an "early warning" system. The goal is to quickly find any pests and how they are getting in, *before* they become a big problem.
- **Prevent pest access:** Caulk the cracks and crevices pests may use to move or hide in. For larger holes, use stainless steel or copper mesh to plug the holes, and then use a silicone caulk to seal it. Pay special attention to areas where pipes and wires come in through the wall. Make sure to use window screens and that they are in good repair.
- **Prevent harborage:** Reduce clutter – get rid of the things you do not need such as old clothes, newspapers, magazines and cardboard boxes where pests can easily hide.
- **Prevent food sources:** Store food in plastic or glass containers with tight-fitting lids to prevent pests from eating it. Keep dirty dishes in soapy water so that pests cannot eat the scraps. Clean thoroughly, with particular attention to the floor under the refrigerator, stove/oven and other places where food crumbs and spills may be collecting. Remove and store pet foods in pest-proof containers at night. Use a trash can with a tight-fitting lid and empty regularly.
- **Prevent water sources:** Fix any water leaks, wipe up spills and remove pet's water dishes at night.

Physical Controls

Sticky traps for insects and snap-traps for rodents are safe and good tools for catching the occasional invader. Be sure they are placed correctly for maximum benefit. Roaches and rodents run along the wall in concealed spaces, so make sure the traps are flush with the wall. Snap traps should snap toward the wall.

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Chemical Controls: Less-Risky Pesticides
After using all of the above methods, you may need to consider using a pesticide. Try to select products that limit human exposures to the product. Aerosols, liquid sprays, mothballs or "bug bombs" all pose *more* risk of chemical exposure and cause lung irritation. Instead, look for pesticides in tamper-resistant bait stations or a "gel" formula. Boric acid dust can be used, if *carefully* puffed gently and in small amounts behind wall voids and socket covers to eliminate insects hiding behind these areas. Avoid spreading any kind of pesticidal dust in and around the rooms of the home.

Safety First!
ALWAYS read the entire label on any pesticide product before you buy and use them in your home. Ask yourself: does this product control the pest I have? Can I use this product without exposing myself and/or my family to the pesticide? If pesticides are stored in the home, store in a **locked** cabinet at least 4 feet up and out of the reach of children.
NEVER buy pesticides in unmarked containers or that do not have an EPA registration number on the container. These products are **illegal** and potentially very dangerous to your family.

Eliminating pests *safely* will help reduce the number one asthma trigger in the home!

For more information and assistance, contact:
www.paipm.org
The Pennsylvania Integrated Pest Management Program
Phone: (814) 863-8884
Philadelphia School & Community IPM Partnership
Phone: 215-471-2200 Ext. 109
Email: pscip@psu.edu

SAFER PEST CONTROL PROJECT This fact sheet adapted from the original by Safer Pest Control Project
www.spcpweb.org

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Curr Opin Allergy Clin Immunol. 2011 Apr;11(2):90-6.

Pesticides and asthma.

Hernández AF, Parrón T, Alarcón R.

University of Granada School of Medicine, Granada, Spain. ajerez@ugr.es

Abstract

PURPOSE OF REVIEW: Several clinical and epidemiological studies have reported an association between exposure to pesticides, bronchial hyper-reactivity and asthma symptoms. This article reviews the mechanistic evidence lending support to the concept that either acute or chronic low-level inhalation of pesticides may trigger asthma attacks, exacerbate asthma or increase the risk of developing asthma.

RECENT FINDINGS: Pesticide aerosols or gases, like other respiratory irritants, can lead to asthma through interaction with functional irritant receptors in the airway and promoting neurogenic inflammation. Cross-talk between airway nerves and inflammatory cells helps to maintain chronic inflammation that eventually damages the bronchial epithelium. Certain organophosphorus insecticides cause airway hyper-reactivity via a common mechanism of disrupting negative feedback control of cholinergic regulation in the lungs. These pesticides may interact synergistically with allergen sensitization rendering individuals more susceptible for developing asthma.

SUMMARY: Many pesticides are sensitizers or irritants capable of directly damaging the bronchial mucosa, thus making the airway very sensitive to allergens or other stimuli. However, most pesticides are weakly immunogenic so that their potential to sensitize airways in exposed populations is limited. Pesticides may increase the risk of developing asthma, exacerbate a previous asthmatic condition or even trigger asthma attacks by increasing bronchial hyper-responsiveness.

PMID: 21368619 [PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms, Substances

LinkOut - more resources

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Strategic Plan for Asthma in California

2008–2012

February 2008

CDPH
California Department of
PublicHealth

d. Asthma management strategies that lead to a reduction in asthma morbidity and mortality.

e. Identification, translation, and implementation of evidence-based best practices in health care service delivery, at the levels of the individual practitioner, group practice and insurance plan.

2.4.2. CDPH will convene an asthma research symposium every two years to summarize recent important research findings, to assess their implications and to address current interests, and research questions as suggested by stakeholders. The symposium will provide an opportunity to track etiologic research and foster communication among researchers to increase the chances of crosscutting research (Figure 5. Possible Research Areas for Future Research Symposia).

Sample Performance Indicator

An asthma research symposium is convened every two years starting in 2008.

2.5. Policy regarding asthma in California will be informed by analysis and interpretation of data.

2.5.1. The determination of priority data to be collected will be guided by both availability and the need for developing and evaluating specific policies and interventions.

2.5.2. Data analysis, reports, and key findings will be disseminated to policy makers, health care providers, employers, community based organizations and the public.

2.5.3. Data will be identified, analyzed, and interpreted to support policy development for goals 1–5 of this *Plan*.

2.5.4. When data is limited or unavailable, expert opinion and the best available evidence will be used to assess policy proposals and to guide policy development.

Sample Performance Indicator

Data is considered in policy decisions and policy is considered in setting data priorities.

Figure 4. Potential Indoor and Outdoor Research Areas

- Research related to air pollution (e.g., traffic and industrial facilities); link data from the Air Resources Board and the Air Quality Management Districts.
- Research on the pathways, drift patterns, and exposure levels of second hand smoke and the health effects associated with this trigger in multi-unit housing settings.
- Research on the connections between global warming, air pollution, and asthma.
- Research on specific asthma triggers, sensitizers, and irritants such as cleaning chemicals, pesticides, pollens, landscaping practices, and fragrances.

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MORENO VALLEY: Officials seek comments on ProLogis project

BY LORA HINES

STAFF WRITER

lhines@pe.com

Published: 26 July 2012 07:45 PM

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Public comments are being accepted on a draft environmental impact report for a proposed 2.2 million square-foot warehouse project in Moreno Valley that officials began discussing about five years ago.

City planning officials recently released the report for the proposed ProLogis Eucalyptus Industrial Park Project, which would consist of six warehouses south of Highway 60 and east of the Moreno Valley Auto Mall. Residents, state and local agencies and community and environmental groups have until Sept. 4 to submit comments on the report.

ProLogis, a San Francisco-based international warehouse developer, bought more than 125 acres in the 28000 block of Eucalyptus Avenue more than five years ago. Almost all of it will be used for the project, which will require amendments to the city's general plan and zoning requirements.

When it was initially proposed, ProLogis officials estimated the project could cost as much as $150 million to develop and would create between 1,000 and 1,500 jobs. No one from the corporation could be reached Thursday, July 26, to offer a cost or job update or comment on the project.

According to the draft environmental impact report, the poor economy in 2008 stalled the project. ProLogis recently decided to pursue the process, the report states.

City planning official John Terell said there is nothing unusual about the project or its potential impacts that have delayed it.

In March 2008, city planning officials received 25 responses from state and local agencies, residents and environmental groups about concerns with the proposed project, including increased traffic, pollution and its proximity to schools.

The report states the project could affect areas such as air and water quality, animal habitat, Native American prehistoric sites, drainage and traffic.

Resident Marti Orth was among those who submitted comment about the proposed project in 2008. She said she is as opposed to it now as she was then, but she believes her opinion will have little effect on the City Council, which will decide whether to approve the project later this year.

"I think it's a forgone conclusion," said Orth, a resident of more than 40 years. "First, decisions are made. Then they ask for opinions."

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MORENO VALLEY: Prison time, payback for Sutton

MORENO VALLEY: Home invasion slaying has four defendants

MORENO VALLEY: Proposed Harbor Freight project moves forward

MORENO VALLEY: City spending nearly $2 million for surveillance cameras

Today's Poll

What's this?

Beaumont is reducing the number of cats residents are allowed to have at home without getting a cattery license, from nine to four. How many cats do you have at home?

- Zero
- One
- Two
- 3-4
- 5-9
- 10 or more
- No comment

Submit

Photos

INLAND EMPIRE: Rain cells hit inland counties

NORCO: Mounted Posse/Professional Rodeo

ANAHEIM: Harvest Crusades

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Moreno Valley storm cleanup
Published: Friday, August 31, 2012
Category: News & Politics

Rains block Moreno Valley road
Published: Thursday, August 30, 2012
Category: News & Politics

Star scores
Published: Thursday, August 30, 2012
Category: Education

CRIME BLOTTER: $10,000 reward offered in shooting death of Ashanti Hassan
Published: Thursday, August 30, 2012
Category: News & Politics

Community College Students Fighting for Classes
Published: Thursday, August 30, 2012
Category: News & Politics

On Wednesday, July 25, city manager Henry Garcia told hundreds of Inland area officials and business owners that warehouse development and health care will be Moreno Valley's job growth focus areas because they have the most potential to employ the city's primarily blue-collar workforce.

Orth said residents have little reason to believe that the proposed project will bring as many jobs as officials claimed because the Skechers warehouse didn't.

"I don't know why (ProLogis) would be any better," she said.

Skechers had employed about 1,000 people in five smaller warehouses in Ontario before consolidating and moving to Moreno Valley. Moreno Valley officials and project supporters promised that Skechers warehouse would bring more than 1,000 jobs. It employs about 600 people.

City officials have said they expect the number of employees to increase as the economy improves.

Comments about the ProLogis project are to be sent to associate city planner Jeff Bradshaw, Moreno Valley Planning Division, 14177 Frederick St., Moreno Valley 92553 or send e-mail to jeffreyb@moval.org.

Comments

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EPA Brownfields Grants, CERCLA Liability, and All Appropriate Inquiries

To be eligible for an EPA brownfields grant to address contamination at brownfields properties, eligible entities must demonstrate that they are not liable under CERCLA for the contamination at the site. Accordingly, eligible entities who may be considered "potentially responsible parties" under CERCLA must demonstrate they meet one of the liability protections or defenses set forth in CERCLA by establishing that they are (1) an innocent landowner, (2) a contiguous property owner, (3) a bona fide prospective purchaser, or (4) a government entity that acquired the property involuntarily through bankruptcy, tax delinquency, or abandonment, or by exercising its power of eminent domain.

To claim protection from liability as an innocent landowner, contiguous property owner, or bona fide prospective purchaser, property owners, including state and local governments, must conduct all appropriate inquiries prior to acquiring the property.

What is CERCLA?

The Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), also known as "Superfund," was established to address abandoned hazardous waste sites. Among other things, CERCLA establishes a liability scheme for determining who can be held accountable for releases of hazardous substances. CERCLA also establishes the authority for EPA's Brownfields Program and sets forth which entities and properties are eligible for brownfields grants.

Can state and local governments be found liable for contamination at brownfields?

Yes. Under CERCLA, persons (including state and local governments) can be liable by virtue of property ownership, or by virtue of their actions with respect to a particular site. For sites from which there is a release or threatened release of hazardous substances, the categories of "potentially responsible parties" include any person or party who:

- Currently owns or operates the property, or owned or operated the property at the time of disposal of hazardous substances;
- Arranged for hazardous substances to be disposed of or transported to the site for disposal; or
- Transported hazardous substances to the site.

Applicants should note that CERCLA employs a "strict liability" scheme—that means it is without regard to fault. Accordingly, a person who owns a property from which there is a release of hazardous substances can be held liable just by virtue of ownership.

If I am applying for a brownfields grant, do I have to worry about CERCLA liability?

Yes. Brownfields grantees are prohibited from using grant money to pay response costs at a brownfield site for which the grantee is potentially liable under CERCLA.

Therefore, all brownfields grantees who may be potentially liable at the site for which they are seeking funds must demonstrate that they are not liable for the contamination that will be addressed by the grant, subgrant, or loan. Applicants who own or operate the property for which they are seeking funding, or who may have owned or operated the property at the time of disposal of hazardous substances, must demonstrate they fall within one of the liability protections.

Cleanup grant applicants in particular should take note of this prohibition. Because cleanup grantees are required to own a site to receive brownfields funding—and because owners of contaminated property are liable under CERCLA—cleanup grant applicants ***must*** demonstrate they meet one of the liability protections described above. Some grant applicants who do not own the property for which they are seeking funding, or who are not seeking site-specific grant funds, may not fall within one of the categories of "potentially responsible parties," and thus may not have to demonstrate they meet a liability protection.

Please contact your Regional Brownfields representative if you are not sure whether you will need to demonstrate a liability protection to be eligible for a grant.

Who may be protected from liability under CERCLA?

The CERCLA statute provides protection from liability for certain parties, provided they comply with specific criteria outlined in the statute. Parties provided protection from CERCLA liability include:

- Innocent landowners (CERCLA §101(35)(A))
- Contiguous property owners (CERCLA §107(q))
- Bona fide prospective purchasers (CERCLA §§101(40) and 107(r))
- Units of state or local government that acquire ownership or control involuntarily through bankruptcy, tax delinquency, or abandonment (CERCLA §101(20)(D))

Government entities that acquire property by eminent domain (CERCLA §101(35)(A)(ii))

- Not be affiliated with any liable party through any familial relationship or any contractual, corporate or financial relationship (other than a relationship created by the instrument by which title to the property is conveyed or financed).

NOTE: Property acquisition includes properties acquired by gifts and zero price transactions.

How can a state or local government demonstrate that it is not liable for contamination at a brownfield?

All state and local governments that may be potentially liable at a site for which they are applying for funding (including site-specific assessment grants, cleanup grants, or subgrants or loans from revolving loan funds), ***must*** demonstrate that they qualify for one of the CERCLA liability protections. All non-profit entities applying for brownfields cleanup grants also must make this demonstration.

Eastern Manufacturer Brewer, Maine, prior to cleanup (above) and after (right)

What are the conditions for attaining liability protection under CERCLA?

To be eligible for liability protection under CERCLA as an innocent landowner, contiguous property owner or bona fide prospective purchaser, prospective property owners must:

- Conduct All Appropriate Inquiries in compliance with 40 CFR Part 312, prior to acquiring the property;
- Comply with all Continuing Obligations after acquiring the property. (CERCLA §§101(40)(C – G) and §§107(q)(A)(iii – viii)); and

To demonstrate that it qualifies as an innocent landowner, contiguous landowner, or bona fide prospective purchaser, the applicant must:

- Conduct All Appropriate Inquires prior to acquiring the property, ***and***
- Comply with all Continuing Obligations after acquiring the property.

State and local governments that acquired a property involuntarily through bankruptcy, tax delinquency, or abandonment, or by exercising their power of eminent domain, do not have

to conduct all appropriate inquiries prior to acquiring the property, but must exercise "due care" after acquiring the property (CERCLA §101(35)(A) and §§107(b)(3)(a – b)). [Note: One threshold criteria for applicants seeking cleanup grant funding is that a Phase I must be conducted prior to application submission. Accordingly, although state and local governments that acquired property involuntarily are not required to conduct all appropriate inquiries for purposes of establishing a liability protection, they may have to conduct all appropriate inquiries anyway to be eligible for a cleanup grant.]

What is "All Appropriate Inquiries"?

"All Appropriate Inquiries," or AAI is the process of conducting due diligence or a Phase I Environmental Site Assessment to determine prior uses and ownership of a property and assess conditions at the property that may be indicative of releases or threatened releases of hazardous substances at, on, in, or to the property.

The standards and practices established as comprising "All Appropriate Inquiries" are set forth in regulations promulgated at 40 CFR Part 312.

EPA recognizes two ASTM International Standards as compliant with the AAI requirements: ASTM E1527-05 "Standard Practice for Environmental Site Assessments: Phase I Environmental Site Assessment Process" and E2247-08 "Standard Practice for Environmental Site Assessments: Phase I Environmental Site Assessment Process for Forestland or Rural Property."

When must All Appropriate Inquiries be conducted?

- All Appropriate Inquiries must be conducted or updated within one year ***prior to acquiring ownership of a property.***
- Certain aspects or provisions of All Appropriate Inquiries (i.e., interviews of current and past owners, the review of government records, the on-site visual inspection, and searches for environmental cleanup liens) must be conducted or updated ***within 180 days prior to acquiring ownership of a property.***

Who can perform All Appropriate Inquiries?

The individual who supervises or oversees the conduct of the AAI investigation and signs the final report required in the AAI regulation must meet the definition of an "Environmental Professional" provided in the AAI Final Rule (40 CFR §312.10).

A person that does not qualify as an "Environmental Professional" as defined in 40 CFR §312.10, may assist in the conduct of the investigation if he or she is under the responsible charge of a person meeting the definition.

What are "Continuing Obligations?"

After acquiring a property, to maintain the liability protections, landowners must comply with "continuing obligations" during their property ownership. The continuing obligations include:

1. Provide all legally required notices with respect to the discovery or release of a hazardous substance;
2. Exercise appropriate care with respect to the hazardous substances by taking reasonable steps to stop or prevent continuing or threatened future releases and exposures, and prevent or limit human and environmental exposure to previous releases;
3. Provide full cooperation, assistance, and access to persons authorized to conduct response actions or natural resource restoration;
4. Comply with land use restrictions and not impede the effectiveness of institutional controls; and
5. Comply with information requests and subpoenas.

Where can I get additional information?

For general information, see the EPA Brownfields website at: **www.epa.gov/brownfields**

For more information on the AAI requirements, see: **http://www.epa.gov/brownfields/regneg.htm**

For more information on continuing obligations, see:
http://www.epa.gov/compliance/resources/policies/cleanup/superfund/common-elem-guide.pdf

Contact Patricia Overmeyer at: **Overmeyer.patricia@epa.gov**

Brownfields Fact Sheet
EPA Brownfields Grants,
CERCLA Liability,
and All Appropriate Inquiries

Solid Waste
and Emergency
Response (5105)

EPA 560-F-09-026
April 2009
www.epa.gov/brownfields

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COUNTY OF RIVERSIDE COMMUNITY HEALTH AGENCY
DEPARTMENT OF ENVIRONMENTAL HEALTH

Underground Storage Tank Closure Application and Permit

A permit will be issued for closure or abandonment in place of UST when a work plan is submitted. In addition to this permit, all applicable permits required by the local fire department, building department, and the Air Quality Management District must be obtained and should be available for review at the closure site. **A WORK PLAN MUST BE SUBMITTED TO OBTAIN A PERMIT**. All tank closures must, at a minimum, comply with the California Underground Storage Tank Regulations and the appropriate section of the California Health and Safety Code.

_____ _____
FACILITY NUMBER **PLAN CHECK NUMBER**

NAME OF FACILITY	ADDRESS OF FACILITY	CITY	ZIP	PHONE NUMBER
NAME OF OWNER/OPERATOR	ADDRESS OWNER/OPERATOR	CITY	ZIP	PHONE NUMBER
NAME OF CONTRACTOR/APPLICANT	ADDRESS CONTRACTOR/APPLICANT	CITY	ZIP	PHONE NUMBER

CONTRACTOR'S LICENSE TYPE AND NUMBER (Including Hazardous Materials Certification)

ANSWER THE FOLLOWING QUESTIONS DESCRIBING THE TANK(S) TO BE CLOSED OR ABANDONED. IF YOU HAVE MORE THAN FOUR (4) TANKS, PROVIDE INFORMATION ON AN ADDITIONAL FORM.

TANK INFORMATION:	TANK 1		TANK 2		TANK 3		TANK 4	
SINGLE/DOUBLE WALLED TANK/AGE								
SIZE OF TANK/TANK MATERIAL								
SUBSTANCE STORED/ SUSPECTED OF LEAKING								

CIRCLE THE METHOD OF CLOSURE: **REMOVAL** **ABANDONMENT IN PLACE** **TEMPORARY CLOSURE**

UNDERGROUND TANK CLOSURE INSPECTIONS MUST BE SCHEDULED AT LEAST FIVE (5) BUSINESS DAYS IN ADVANCE.

RIVERSIDE (951) 358-5055 INDIO (760) 863-8976 HEMET (951) 766-6524

CONTRACTOR/APPLICANT SIGNATURE:_____ DATE: _____

PERMIT APPROVED BY **(Ensure Workplan is Attached)** :_____ DATE: _____

Please Make Your Check Payable To The County Of Riverside

AMOUNT ATTACHED $_____ TRANSACTION/OCR NO._____ CHECK NO._____

WORK PLAN SUBMITTED_____

****THIS PERMIT FOR CLOSURE IS VALID FOR 90 DAYS FROM THE DATE OF ISSUE.**

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UNDERGROUND STORAGE TANK GUIDELINES TO CLOSURE BY REMOVAL

NOTE: This Division strongly urges applicants to contact the local Fire Department Jurisdiction prior to the removal of any Underground Storage Tank (UST) as local fire restrictions may be more stringent.

A. General Information

1. A completed permit application must be submitted to the Division. Permit fees for UST closure are required.

2. The State Contractors License Board requires contractors who install or remove USTs and piping to have the **Hazardous Substance Certification** and one of the following licenses: General Engineering "A"; Limited Specialty C-61/D-40 for UST's and pipelines; Pipeline Contractor C-34 for pipelines only; or General Building "B" (limited).

3. It is the responsibility of the UST owner or duly authorized representative, to notify other governmental agencies that may have applicable permit requirements. This includes, but is not limited to, the following: Local Fire Agency; Local Building Department; and Air Quality Management District (AQMD).

4. Between cessation of use/storage and the actual closure, monitoring shall be continued as required by the operating permit.

5. **The permitted (i.e. UST owner, contractor) shall be responsible for site safety.**

B. Closure Requirements

1. A completed UST closure application and four (4) copies of a UST removal work plan must be submitted and applicable closure fees paid. A closure permit, valid for ninety (90) days, will be issued upon **RECEIPT** of the work plan. If closure is not completed within ninety (90) day, the closure permit shall expire. Additional fees will be assessed for a new closure permit.

2. A UST closure inspection must be scheduled with the Division at least **FIVE (5) WORKING DAYS IN ADVANCE** of the proposed closure.

3. All liquids, solids, and sludge shall be removed and handled according to the provisions of Chapter 6.5, Division 20 of the Health and Safety Code and Title 22, Chapter 32, Section 67383.1 of the California Code of Regulations. The UST shall be properly cleaned, which usually requires the pressure washing/rinsing of the UST and removal of the contents via a vacuum type pump system that is designed to safely handle flammable liquids. The Division can provide a list of licensed hazardous waste haulers/tank rinsing companies.

4. Flammable vapors must be purged from the UST and the UST must be inerted to prevent an explosion or fire. The Division must verify LEL is < 10% prior to the inerting of the UST with 22.2 lbs. of dry ice per 1,000 gallons of UST capacity. The UST must then promptly be removed and transported to its final destination accompanied by the UST Closure Certification Form. The local fire and AQMD regulations may be more restrictive.

5. All associated piping must be removed. Product or residue spillage must be prevented.

6. Proper UST disposal documentation, in accordance with the requirements of Chapter 6.5, Division 20 of the Health and Safety Code, shall be provided to the Division.

7. Applicant must demonstrate to the satisfaction of the Division whether or not an unauthorized release has occurred. Demonstration will be based upon results of soil/water samples obtained during UST closure activities.

8. The sample analysis must be performed by a California state certified laboratory. The sample analysis, along with the Division Sample Receipt form and a chain of custody must be received by the Division within thirty (30) days.

9. Soil samples shall be taken below the UST/piping system at the time of UST removal. At a minimum, samples are required 2' (feet) and 6' (feet) below the fill end of the tank, with a separate 2' sample taken at the opposite end of the tank. A separate sample for each 20 lineal feet of piping and at each dispenser shall be taken. (It is strongly recommended that 6' samples be taken at each piping and dispenser sampling location.) Division personnel may require additional sampling.

10. The soil samples shall be analyzed for all constituents of the previously stored hazardous substances and their breakdown constituents or transformation products according to the Table titled "Laboratory Analysis for Samples Collected at UST Sites".

11. The Division will evaluate all sample results and determine if any further corrective action is required.

12. The detection limit, in accordance with the table titled "Laboratory Analysis for Samples Collected at UST Sites", shall be reported to the Division in accordance with Article 5 of the California Underground Storage Tank Regulations, Title 23, Division 3, Chapter 16, California Code of Regulations.

C. Work Plan Guidelines

1. A work plan must be submitted (with permit application) to the Division prior to UST removal.

2. The work plan should include the following information:
   - A. Site Description: the physical address along with a site plot plan.
   - B. On-Site Security: indicate who will be on site (what agencies, contractors, etc.), and how site security will be maintained.
   - C. Contacts: Indicate the responsible party's name and phone number, contractor's name and phone number.
   - D. Treatment of USTs prior to removal--indicate the following:
     1) How the USTs will be cleaned. Indicate name and credential of certified UST cleaner, as well as final destination of rinsate.
     2) How you will inert the UST. Indicate the quantity of dry ice to be used, and that it will not be placed into the UST until the Division representative is on site.
     3) If the USTs are to be saw cut. If so, this needs to be detailed.
     4) Destination of UST—indicate where the USTs are going and how they will be transported. All openings in the UST shall be plugged, except for a 1/8" inch vent hole.
     5) Air/vapor monitoring—type of monitoring equipment to be used and date of last calibration.

E. Depth to groundwater: region specific. If tank is in ground water, indicate safety precautions that will be taken.

F. Equipment to be used on site:
   1) Heavy equipment: indicate the type of equipment to be used to physically remove the USTs from the excavation. Ensure the equipment is rated to handle the weight of the UST.
   2) Sampling: indicate the type of equipment to be used to gather the soil/water samples. Ensure equipment is able to reach at least 6' below the bottom of the UST, piping, and dispensers. Indicate the type of container that will be used to hold the samples. Demonstrate how contamination of samples is to be avoided. Provide the name of the California certified lab that will be analyzing the samples. Indicate when the samples will be analyzed and how you will hold the samples in the interim. Ensure a chain of custody accompanies the samples to the lab.

G. Excavation status: indicate the disposition of the excavation upon removal of the tank (i.e. open and fenced, backfilled with new and excavated soil, etc.)

H. Safety—indicate the following:
   1) The type of personal protective equipment to be required for all persons on site.
   2) The safety items that will be available on site (fire extinguisher, first aid, etc.).
   3) The nearest emergency medical facility to be used in the event of an accident or emergency.
   4) That all tools to be used to clean the exterior of the tank will be non-sparking. Give examples and be specific.
   5) Whether shoring is necessary/required.
   6) The person who will be responsible for safety (Safety Officer).
   7) The presence of any overhead hazards (electrical lines, etc.). Indicate how the hazard will be addressed/mitigated.
   8) The presence of any underground hazards (gas pipes, sewer lines, water mains, etc.) and how the hazard will be addressed/mitigated.

NOTE: THIS DOCUMENT IS FOR GUIDANCE ONLY AND IS NOT INTENDED TO SUPERSEDE ANY SAFETY OR OTHER LEGAL REQUIREMENTS. OWNER / CONTRACTOR RETAINS ALL RESPONSIBILITY ASSOCIATED WITH ACTIVITIES SURROUNDING THE SAFE AND LEGAL REMOVAL OF THE TANK(S).

Revised 8/06

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CAPCOA

CALIFORNIA AIR POLLUTION CONTROL OFFICERS ASSOCIATION

Quantifying Greenhouse Gas Mitigation Measures

A Resource for Local Government to Assess Emission Reductions from Greenhouse Gas Mitigation Measures

August, 2010

Quantifying Greenhouse Gas Mitigation Measures

A Resource for Local Government to Assess Emission Reductions from Greenhouse Gas Mitigation Measures

August, 2010

California Air Pollution Control Officers Association

with

Northeast States for Coordinated Air Use Management

National Association of Clean Air Agencies

Environ

Fehr & Peers

the land types. A third way to increase sequestration is by planting new trees on either developed or undeveloped land.

The increase in carbon sequestration capacity is determined by calculating the total sequestration capacity of converted land, new vegetated land and trees; and then subtracting the combined capacity of vegetated land or trees that are removed. Carbon sequestration capacities for different land types (e.g. cropland, forest land) and for different tree species classes are available from IPCC guidelines, and summarized in Table E-2, in Appendix E.

Construction Equipment

Construction equipment typically uses diesel fuel and releases emissions based on the amount of fuel combusted and emission factor of the equipment. Emissions can be reduced by using equipment that emits fewer pollutants for the same amount of work.

This is typically equipment powered through grid electricity or hybrid technology. The exclusive use of grid electricity eliminates the diesel emissions at the site but would increase indirect electricity emissions. However, grid-based emissions are typically small compared to the emissions from the diesel-fueled equipment (depending on the source of grid power). Hybrid-powered equipment would decrease but not completely eliminate fuel use. The electricity for hybrid equipment is self-generated unless the equipment has plug-in capability, so it would not increase grid-based electrical generation and the associated emissions there.

The emissions reductions in this category are determined by finding the difference between the estimated mitigation emissions and the baseline emissions for construction equipment. Emissions for the mitigated scenario may consist of direct emissions from combustion fuel use, and/or indirect emissions from grid electricity. These would be calculated using resources described previously, such as the OFFROAD database and literature-based methodologies and values.

Transportation

Transportation emissions can be reduced by improving the emissions profile of the vehicle fleet that travels the roads, or by reducing the vehicle miles traveled by the fleet. The majority of the measures quantified for this report focus on the reduction of VMT. This can be accomplished by optimizing the location and types of land uses in the project and its immediate vicinity, and by site enhancements to roads, and to bike and pedestrian networks to encourage the use of alternative modes of transportation. Mode shifts are also encouraged by implementing parking policies, transit system improvements, and trip reduction coordination or incentive programs.

8.1.4 Institute a Heavy-Duty Off-Road Vehicle Plan

Range of Effectiveness:

Not applicable on its own. This measure ensures compliances with other mitigation measures.

Measure Description:

The Project Applicant should provide a detailed plan that discusses a construction vehicle inventory tracking system to ensure compliances with construction mitigation measures. The system should include strategies such as requiring hour meters on equipment, documenting the serial number, horsepower, manufacture age, fuel, etc. of all onsite equipment and daily logging of the operating hours of the equipment.

Measure Applicability:

- This measure ensures compliances with other mitigation measures.
- Construction vehicles.

Preferred Literature:

None

Alternative Literature:

None

Literature References:

None

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http://www.epa.gov/oaqps001/greenbk/ancl.html
Last updated on Friday, July 20, 2012

Green Book

You are here: EPA Home Green Book Currently Designated Nonattainment Areas for All Criteria Pollutants

Currently Designated Nonattainment Areas for All Criteria Pollutants

As of July 20, 2012
Listed by State, County then Pollutant
View Notes

State, County, Pollutant, * Part County NAA, NAA Area Name - Classification Standard

ALABAMA

Jackson Co
PM-2.5 1997 * Chattanooga, AL-TN-GA - (Nonattainment)
Jefferson Co
PM-2.5 1997 Birmingham, AL - (Nonattainment)
PM-2.5 2006 Birmingham, AL - (Nonattainment)
Pike Co
Lead 2008 * Troy, AL - (Nonattainment)
Shelby Co
PM-2.5 1997 Birmingham, AL - (Nonattainment)
PM-2.5 2006 Birmingham, AL - (Nonattainment)
Walker Co
PM-2.5 1997 * Birmingham, AL - (Nonattainment)
PM-2.5 2006 * Birmingham, AL - (Nonattainment)

State, County, Pollutant, * Part County NAA, NAA Area Name - Classification Standard

ALASKA

Anchorage Municipality
PM-10 * Eagle River, AK - (Moderate)
Fairbanks North Star Borough
PM-2.5 2006 * Fairbanks, AK - (Nonattainment)
Juneau City and Borough
PM-10 * Juneau, AK - (Moderate)

State, County, Pollutant, * Part County NAA, NAA Area Name - Classification Standard

ARIZONA

Cochise Co
PM-10 * Paul Spur/Douglas (Cochise County), AZ - (Moderate)
Gila Co
PM-10 * Hayden AZ - (Moderate)
PM-10 * Miami, AZ - (Moderate)
Maricopa Co
8-Hr Ozone 1997 * Phoenix-Mesa, AZ - (Marginal)
PM-10 * Phoenix, AZ - (Serious)

8-Hr Ozone 2008	* Phoenix-Mesa, AZ - (Marginal)
Pima Co	
PM-10	* Ajo (Pima County), AZ - (Moderate)
PM-10	* Rillito, AZ - (Moderate)
Pinal Co	
8-Hr Ozone 1997	* Phoenix-Mesa, AZ - (Marginal)
PM-10	* Hayden AZ - (Moderate)
PM-10	* Phoenix, AZ - (Serious)
PM-10	* West Pinal, AZ - (Moderate)
PM-2.5 2006	* West Central Pinal, AZ - (Nonattainment)
SO2	* Hayden (Pinal County), AZ - (Primary)
8-Hr Ozone 2008	* Phoenix-Mesa, AZ - (Marginal)
Santa Cruz Co	
PM-10	* Nogales, AZ - (Moderate)
PM-2.5 2006	* Nogales, AZ - (Nonattainment)
Yuma Co	
PM-10	* Yuma, AZ - (Moderate)

State, County, Pollutant, * Part County NAA, NAA Area Name - Classification Standard

ARKANSAS

Crittenden Co	
8-Hr Ozone 2008	Memphis, TN-MS-AR - (Marginal)

State, County, Pollutant, * Part County NAA, NAA Area Name - Classification Standard

CALIFORNIA

Alameda Co	
8-Hr Ozone 1997	San Francisco Bay Area, CA - (Marginal)
PM-2.5 2006	San Francisco Bay Area, CA - (Nonattainment)
8-Hr Ozone 2008	San Francisco Bay Area, CA - (Marginal)
Amador Co	
8-Hr Ozone 1997	Amador and Calaveras Cos (Central Mtn), CA - (Moderate)
Areas of Indian Country	
8-Hr Ozone 2008	Morongo Band of Mission Indians - (Serious)
8-Hr Ozone 2008	Pechanga Band of Luiseno Mission Indians of the Pechanga Reservation - (Moderate)
Butte Co	
8-Hr Ozone 1997	Chico, CA - (Marginal)
PM-2.5 2006	* Chico, CA - (Nonattainment)
8-Hr Ozone 2008	Chico (Butte County), CA - (Marginal)
Calaveras Co	
8-Hr Ozone 1997	Amador and Calaveras Cos (Central Mtn), CA - (Moderate)

8-Hr Ozone 2008	Calaveras County, CA - (Marginal)
Contra Costa Co	
8-Hr Ozone 1997	San Francisco Bay Area, CA - (Marginal)
PM-2.5 2006	San Francisco Bay Area, CA - (Nonattainment)
8-Hr Ozone 2008	San Francisco Bay Area, CA - (Marginal)
El Dorado Co	
8-Hr Ozone 1997	* Sacramento Metro, CA - (Severe 15)
PM-2.5 2006	* Sacramento, CA - (Nonattainment)
8-Hr Ozone 2008	* Sacramento Metro, CA - (Severe 15)
Fresno Co	
8-Hr Ozone 1997	San Joaquin Valley, CA - (Extreme)
PM-2.5 1997	San Joaquin Valley, CA - (Nonattainment)
PM-2.5 2006	San Joaquin Valley, CA - (Nonattainment)
8-Hr Ozone 2008	San Joaquin Valley, CA - (Extreme)
Imperial Co	
8-Hr Ozone 1997	Imperial Co, CA - (Moderate)
PM-10	* Imperial Valley, CA - (Serious)
PM-2.5 2006	* Imperial Co, CA - (Nonattainment)
8-Hr Ozone 2008	Imperial County, CA - (Marginal)
Inyo Co	
PM-10	* Owens Valley, CA - (Serious)
Kern Co	
8-Hr Ozone 1997	* Kern Co (Eastern Kern), CA - (Moderate)
8-Hr Ozone 1997	* San Joaquin Valley, CA - (Extreme)
PM-10	* East Kern Co, CA - (Serious)
PM-2.5 1997	* San Joaquin Valley, CA - (Nonattainment)
PM-2.5 2006	* San Joaquin Valley, CA - (Nonattainment)
8-Hr Ozone 2008	* Kern Co (Eastern Kern), CA - (Marginal)
8-Hr Ozone 2008	* San Joaquin Valley, CA - (Extreme)
Kings Co	
8-Hr Ozone 1997	San Joaquin Valley, CA - (Extreme)
PM-2.5 1997	San Joaquin Valley, CA - (Nonattainment)
PM-2.5 2006	San Joaquin Valley, CA - (Nonattainment)
8-Hr Ozone 2008	San Joaquin Valley, CA - (Extreme)
Los Angeles Co	
8-Hr Ozone 1997	* Los Angeles South Coast Air Basin, CA - (Extreme)
8-Hr Ozone 1997	* Los Angeles-San Bernardino Cos. (W Mojave Desert), CA - (Severe 15)

Lead 2008	* Los Angeles County-South Coast Air Basin, CA - (Nonattainment)
PM-10	* Los Angeles South Coast Air Basin, CA - (Serious)
PM-2.5 1997	* Los Angeles-South Coast Air Basin, CA - (Nonattainment)
PM-2.5 2006	* Los Angeles-South Coast Air Basin, CA - (Nonattainment)
8-Hr Ozone 2008	* Los Angeles-San Bernardino Counties (West Mojave Desert), CA - (Severe 15)
8-Hr Ozone 2008	* Los Angeles-South Coast Air Basin, CA - (Extreme)
Madera Co	
8-Hr Ozone 1997	San Joaquin Valley, CA - (Extreme)
PM-2.5 1997	San Joaquin Valley, CA - (Nonattainment)
PM-2.5 2006	San Joaquin Valley, CA - (Nonattainment)
8-Hr Ozone 2008	San Joaquin Valley, CA - (Extreme)
Marin Co	
8-Hr Ozone 1997	San Francisco Bay Area, CA - (Marginal)
PM-2.5 2006	San Francisco Bay Area, CA - (Nonattainment)
8-Hr Ozone 2008	San Francisco Bay Area, CA - (Marginal)
Mariposa Co	
8-Hr Ozone 1997	Mariposa and Tuolumne Cos (Southern Mtn), CA - (Moderate)
8-Hr Ozone 2008	Mariposa County, CA - (Marginal)
Merced Co	
8-Hr Ozone 1997	San Joaquin Valley, CA - (Extreme)
PM-2.5 1997	San Joaquin Valley, CA - (Nonattainment)
PM-2.5 2006	San Joaquin Valley, CA - (Nonattainment)
8-Hr Ozone 2008	San Joaquin Valley, CA - (Extreme)
Mono Co	
PM-10	* Mammoth Lake, CA - (Moderate)
PM-10	* Mono Basin, CA - (Moderate)
Napa Co	
8-Hr Ozone 1997	San Francisco Bay Area, CA - (Marginal)
PM-2.5 2006	San Francisco Bay Area, CA - (Nonattainment)
8-Hr Ozone 2008	San Francisco Bay Area, CA - (Marginal)
Nevada Co	
8-Hr Ozone 1997	* Nevada Co. (Western Part), CA - (Moderate)
8-Hr Ozone 2008	* Nevada Co. (Western Part), CA - (Marginal)
Orange Co	
8-Hr Ozone 1997	Los Angeles South Coast Air Basin, CA - (Extreme)
PM-10	Los Angeles South Coast Air Basin, CA - (Serious)
PM-2.5 1997	Los Angeles-South Coast Air Basin, CA - (Nonattainment)
PM-2.5 2006	Los Angeles-South Coast Air Basin, CA - (Nonattainment)

8-Hr Ozone 2008	Los Angeles-South Coast Air Basin, CA - (Extreme)
Placer Co	
8-Hr Ozone 1997	* Sacramento Metro, CA - (Severe 15)
PM-2.5 2006	* Sacramento, CA - (Nonattainment)
8-Hr Ozone 2008	* Sacramento Metro, CA - (Severe 15)
Riverside Co	
8-Hr Ozone 1997	* Los Angeles South Coast Air Basin, CA - (Extreme)
8-Hr Ozone 1997	* Riverside Co, (Coachella Valley), CA - (Severe 15)
PM-10	* Coachella Valley, CA - (Serious)
PM-10	* Los Angeles South Coast Air Basin, CA - (Serious)
PM-2.5 1997	* Los Angeles-South Coast Air Basin, CA - (Nonattainment)
PM-2.5 2006	* Los Angeles-South Coast Air Basin, CA - (Nonattainment)
8-Hr Ozone 2008	* Los Angeles-South Coast Air Basin, CA - (Extreme)
8-Hr Ozone 2008	* Riverside Co, (Coachella Valley), CA - (Severe 15)
Sacramento Co	
8-Hr Ozone 1997	Sacramento Metro, CA - (Severe 15)
PM-10	Sacramento Co, CA - (Moderate)
PM-2.5 2006	Sacramento, CA - (Nonattainment)
8-Hr Ozone 2008	Sacramento Metro, CA - (Severe 15)
San Bernardino Co	
8-Hr Ozone 1997	* Los Angeles South Coast Air Basin, CA - (Extreme)
8-Hr Ozone 1997	* Los Angeles-San Bernardino Cos. (W Mojave Desert), CA - (Severe 15)
PM-10	* Los Angeles South Coast Air Basin, CA - (Serious)
PM-10	* San Bernardino Co, CA - (Moderate)
PM-10	* Trona, CA - (Moderate)
PM-2.5 1997	* Los Angeles-South Coast Air Basin, CA - (Nonattainment)
PM-2.5 2006	* Los Angeles-South Coast Air Basin, CA - (Nonattainment)
8-Hr Ozone 2008	* Los Angeles-San Bernardino Counties (West Mojave Desert), CA - (Severe 15)
8-Hr Ozone 2008	* Los Angeles-South Coast Air Basin, CA - (Extreme)
San Diego Co	
8-Hr Ozone 1997	* San Diego, CA - (Moderate)
8-Hr Ozone 2008	San Diego County, CA - (Marginal)
San Francisco Co	
8-Hr Ozone 1997	San Francisco Bay Area, CA - (Marginal)
PM-2.5 2006	San Francisco Bay Area, CA - (Nonattainment)
8-Hr Ozone 2008	San Francisco Bay Area, CA - (Marginal)
San Joaquin Co	

8-Hr Ozone 1997	San Joaquin Valley, CA - (Extreme)
PM-2.5 1997	San Joaquin Valley, CA - (Nonattainment)
PM-2.5 2006	San Joaquin Valley, CA - (Nonattainment)
8-Hr Ozone 2008	San Joaquin Valley, CA - (Extreme)
San Luis Obispo Co	
8-Hr Ozone 2008	* San Luis Obispo (Eastern San Luis Obispo), CA - (Marginal)
San Mateo Co	
8-Hr Ozone 1997	San Francisco Bay Area, CA - (Marginal)
PM-2.5 2006	San Francisco Bay Area, CA - (Nonattainment)
8-Hr Ozone 2008	San Francisco Bay Area, CA - (Marginal)
Santa Clara Co	
8-Hr Ozone 1997	San Francisco Bay Area, CA - (Marginal)
PM-2.5 2006	San Francisco Bay Area, CA - (Nonattainment)
8-Hr Ozone 2008	San Francisco Bay Area, CA - (Marginal)
Solano Co	
8-Hr Ozone 1997	* Sacramento Metro, CA - (Severe 15)
8-Hr Ozone 1997	* San Francisco Bay Area, CA - (Marginal)
PM-2.5 2006	* Sacramento, CA - (Nonattainment)
PM-2.5 2006	* San Francisco Bay Area, CA - (Nonattainment)
8-Hr Ozone 2008	* Sacramento Metro, CA - (Severe 15)
8-Hr Ozone 2008	* San Francisco Bay Area, CA - (Marginal)
Sonoma Co	
8-Hr Ozone 1997	* San Francisco Bay Area, CA - (Marginal)
PM-2.5 2006	* San Francisco Bay Area, CA - (Nonattainment)
8-Hr Ozone 2008	* San Francisco Bay Area, CA - (Marginal)
Stanislaus Co	
8-Hr Ozone 1997	San Joaquin Valley, CA - (Extreme)
PM-2.5 1997	San Joaquin Valley, CA - (Nonattainment)
PM-2.5 2006	San Joaquin Valley, CA - (Nonattainment)
8-Hr Ozone 2008	San Joaquin Valley, CA - (Extreme)
Sutter Co	
8-Hr Ozone 1997	* Sacramento Metro, CA - (Severe 15)
8-Hr Ozone 1997	* Sutter Co (Sutter Buttes), CA - (Marginal)
PM-2.5 2006	Yuba City-Marysville, CA - (Nonattainment)
8-Hr Ozone 2008	* Sacramento Metro, CA - (Severe 15)
Tehama Co	

8-Hr Ozone 2008	* Tuscan Buttes, CA - (Marginal)
Tulare Co	
8-Hr Ozone 1997	San Joaquin Valley, CA - (Extreme)
PM-2.5 1997	San Joaquin Valley, CA - (Nonattainment)
PM-2.5 2006	San Joaquin Valley, CA - (Nonattainment)
8-Hr Ozone 2008	San Joaquin Valley, CA - (Extreme)
Tuolumne Co	
8-Hr Ozone 1997	Mariposa and Tuolumne Cos (Southern Mtn), CA - (Moderate)
Ventura Co	
8-Hr Ozone 1997	* Ventura Co, CA - (Serious)
8-Hr Ozone 2008	* Ventura County, CA - (Serious)
Yolo Co	
8-Hr Ozone 1997	Sacramento Metro, CA - (Severe 15)
PM-2.5 2006	* Sacramento, CA - (Nonattainment)
8-Hr Ozone 2008	Sacramento Metro, CA - (Severe 15)
Yuba Co	
PM-2.5 2006	* Yuba City-Marysville, CA - (Nonattainment)

State, County, Pollutant, * Part County NAA, NAA Area Name - Classification Standard

COLORADO

Adams Co	
8-Hr Ozone 1997	Denver-Boulder-Greeley-Ft Collins-Loveland, CO - (Marginal)
8-Hr Ozone 2008	Denver-Boulder-Greeley-Ft. Collins-Loveland, CO - (Marginal)
Arapahoe Co	
8-Hr Ozone 1997	Denver-Boulder-Greeley-Ft Collins-Loveland, CO - (Marginal)
8-Hr Ozone 2008	Denver-Boulder-Greeley-Ft. Collins-Loveland, CO - (Marginal)
Boulder Co	
8-Hr Ozone 1997	Denver-Boulder-Greeley-Ft Collins-Loveland, CO - (Marginal)
8-Hr Ozone 2008	Denver-Boulder-Greeley-Ft. Collins-Loveland, CO - (Marginal)
Broomfield Co	
8-Hr Ozone 1997	Denver-Boulder-Greeley-Ft Collins-Loveland, CO - (Marginal)
8-Hr Ozone 2008	Denver-Boulder-Greeley-Ft. Collins-Loveland, CO - (Marginal)
Denver Co	
8-Hr Ozone 1997	Denver-Boulder-Greeley-Ft Collins-Loveland, CO - (Marginal)
8-Hr Ozone 2008	Denver-Boulder-Greeley-Ft. Collins-Loveland, CO - (Marginal)
Douglas Co	

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Human Health and Environmental Effects of Emissions from Power Generation

CLEAN AIR MARKET PROGRAMS

Power generation is a significant source of pollutants that can impair human health and the environment, including sulfur dioxide (SO_2), nitrogen oxide (NOx), and mercury. The Clean Air Act has been successful in reducing these emissions, but power generation still contributes approximately 70% of SO_2, 20% of NOx, and 40% of mercury emissions into the environment. These emissions from power generation contribute to a range of human health and environmental problems, and interstate and long range transport of emissions continue to play significant roles in these problems. Cap and trade programs benefit human health and the environment and address transport by significantly reducing emissions over large geographic areas.

When emitted into the atmosphere, SO_2 and NOx react with water and other compounds to form various acidic compounds, fine particles, and ozone. These pollutants can remain in the air for days or even years. Prevailing winds can transport them hundreds of miles, often across state and national borders. The pollutants then fall to the earth in either a wet form (rain, snow, and fog) or a dry form (gases and particles). Impacts include impaired air quality; damage to public health; degradation of visibility; acidification of lakes and streams; harm to sensitive forest and coastal ecosystems; and accelerated decay of materials, paints, and cultural artifacts such as buildings, statues, and sculptures nationwide.

Emissions from power generation contribute to a range of human health and environmental concerns.

Mercury, a product of coal-burning, can be deposited locally or it can be transported through the atmosphere for days to years before being deposited into water bodies. Once mercury reaches lakes, rivers and oceans, it can be transformed into methylmercury and bioaccumulate in the food chain. This results in predatory fish and fish-eating birds and mammals accumulating mercury concentrations millions of times higher than what is found in the water or air.

How Do Power Plant Emissions Impact Human Health?

SO_2 and NOx emissions form fine particles in the atmosphere. Particulate matter is the term used for a mixture of solid particles and liquid droplets found in the air; fine particles ($PM_{2.5}$) are smaller than 2.5 microns (millionths of a meter) in diameter. Power plants emit particles directly into the air, but their major contribution to particulate matter air pollution is emissions of SO_2 and NOx, which are converted into sulfate and nitrate particles in the atmosphere. These particles make up a large proportion of the fine particle pollution in most parts of the country. A substantial body of published scientific literature recognizes a correlation between elevated fine particulate matter and increased incidence of illness and premature mortality. The health effects of $PM_{2.5}$ include:

- Increased incidence of premature death, primarily in the elderly and those with heart or lung disease;
- Aggravation of respiratory and cardiovascular illness, leading to hospitalizations and emergency room visits for children and individuals with heart or lung disease;
- Decreased lung function and symptomatic effects, including acute bronchitis, particularly in children and asthmatics;
- New cases of chronic bronchitis;
- Increased work loss days, school absences, and emergency room visits.

NOx emissions react in the atmosphere to form ozone. NOx and volatile organic compounds react in the atmosphere in the presence of sunlight to form ground-level ozone. Ground-level ozone is a major component of smog in our cities and in many rural areas as well. Though naturally occurring ozone in the stratosphere provides a protective layer high above the earth, the ozone that we breathe at ground level has been linked to respiratory illness and other health problems, including:

- Decreases in lung function, resulting in difficulty breathing, shortness of breath, and other symptoms;
- Respiratory symptoms, including bronchitis, aggravated coughing, and chest pain;
- Increased incidence/severity of respiratory problems (e.g. aggravation of asthma, susceptibility to respiratory infection) resulting in more hospital admissions and emergency room visits;
- Chronic inflammation and irreversible structural changes in the lungs, that, with repeated exposure, can lead to premature aging of the lungs and other respiratory illness.

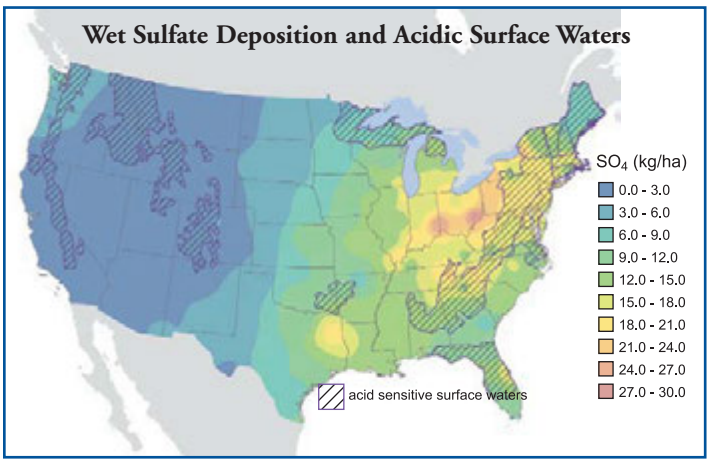
Mercury emissions are deposited in watersheds and transformed into methylmercury, which contaminates fish. In the U.S., human exposure to mercury is primarily the result of consumption of fish contaminated with methylmercury. Other fish-eating

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

mammals and birds are also exposed in this manner. The primary symptoms of mercury exposure are neurological, including brain damage, lack of motor skills, impaired cognitive skills, and difficulty speaking and hearing. These effects are most pronounced on those exposed during the development of the nervous system, such as fetuses and young children. Forty-four states have advisories warning the public to restrict eating fish from their lakes, rivers, streams, and/or coastal waters due to methylmercury. EPA estimates that 12 million acres of lakes and 475,000 miles of rivers, as well as the coastal waters of 11 states, are impaired by mercury.

How Do Power Plant Emissions Impact the Environment?

SO_2 and NOx emissions react in the atmosphere to form acidic compounds that harm lakes and streams. When the acidic compounds that are formed as a result of SO_2 and NOx emissions are deposited to the earth's surface, they can acidify lakes and streams. Acidification (low pH) and the chemical changes that result, including higher aluminum levels, make it difficult for some fish and other aquatic species to survive, grow, and reproduce. In the 1980s, acid rain was found to be the dominant cause of acidification in 75% of acidic lakes and 50% of acidic streams. Areas especially sensitive to acidification include portions of the Northeast (particularly the Adirondack and Catskill Mountains, portions of New England, and streams in the mid-Appalachian highlands) and Southeastern streams. Today in the Adirondack Mountains, Appalachian plateau, and upper Midwest regions, there are 25-30% fewer chronically acidic lakes and streams than in the early 1990s, although these waterbodies remain sensitive to acid rain. Lakes and streams in New England and the Southeast showed little decrease in acidification throughout the 1990s.



Wet Sulfate deposition is highest in many acid sensitive regions.
Source: National Atmospheric Deposition Program.

Acid deposition harms forests and trees. Acid rain can harm forest ecosystems by directly damaging plant tissues. One of the best examples of direct damage involves the leaching of nutrients from the needles of red spruce, which reduces the ability of the trees to tolerate cold winter temperatures and has contributed to the decline of red spruce forests throughout the mountains of the eastern U.S. In other cases, acid rain can combine with other pollutants, such as ozone, to weaken trees and make them vulnerable to threats such as pests, which cause mortality. Acid deposition can also affect forest ecosystems indirectly by changing the chemistry of forest soils, including the leaching of plant nutrients from soils. It can also elevate levels of aluminum in soil water, which impairs the ability of trees to use soil nutrients and can be directly toxic to plant roots.

Nitrogen deposition contributes to impaired coastal water quality. Nitrogen deposited from the atmosphere is a substantial source of nitrogen in many estuaries and coastal waters. Large amounts of nitrogen in estuaries and coastal waters can have significant ecological impacts, including massive die-offs of estuarine and marine plants and animals, loss of biological diversity, and degradation of essential coastal ecosystem habitat such as seagrass beds. For many species of fish and shellfish, these seagrass beds are essential nurseries and places to escape from predators. Excessive amounts of nitrogen in coastal waters from atmospheric deposition are thought to be a contributor to harmful algal blooms, such as red tides, that kill millions of fish each year and can be toxic to humans as well.

Fine particles impair visibility and increase regional haze. Fine particles formed in the atmosphere by the conversion of SO_2 and NOx emissions scatter light and create hazy conditions, decreasing visibility and contributing to regional haze. Visibility impairment spoils scenic vistas across broad regions of the country, including in many National Parks and wilderness areas. Regional haze is also responsible for impaired urban vistas nationwide. In the western U.S., the level of visibility impairment for the worst days remained unchanged through the 1990s. Visibility in the eastern U.S. improved in some areas during the 1990s, but remains significantly impaired overall.

Acid deposition and particles damage materials and cultural resources. A significant number of properties of aesthetic and historical value in the United States, including monuments, buildings, and statues, are potentially at risk for damage from air pollution. Structures made of limestone and marble are particularly sensitive to acid deposition. Acid particles and deposition increase the rate of weathering for these materials, eventually resulting in aesthetic and/or structural damage.

Modeled visibility conditions on the National Mall, Washington, D.C.
Left image: poor visibility, 5 mile visual range. Right image: clear day, 90 mile visual range.

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http://www.epa.gov/air/nitrogenoxides/health.html

Nitrogen Dioxide
Health

Current scientific evidence links short-term NO_2 exposures, ranging from 30 minutes to 24 hours, with adverse respiratory effects including airway inflammation in healthy people and increased respiratory symptoms in people with asthma.

Also, studies show a connection between breathing elevated short-term NO_2 concentrations, and increased visits to emergency departments and hospital admissions for respiratory issues, especially asthma.

NO_2 concentrations in vehicles and near roadways are appreciably higher than those measured at monitors in the current network. In fact, in-vehicle concentrations can be 2-3 times higher than measured at nearby area-wide monitors. Near-roadway (within about 50 meters) concentrations of NO_2 have been measured to be approximately 30 to 100% higher than concentrations away from roadways.

Individuals who spend time on or near major roadways can experience short-term NO_2 exposures considerably higher than measured by the current network. Approximately 16% of U.S housing units are located within 300 ft of a major highway, railroad, or airport (approximately 48 million people). This population likely includes a higher proportion of non-white and economically-disadvantaged people.

NO_2 exposure concentrations near roadways are of particular concern for susceptible individuals, including people with asthma asthmatics, children, and the elderly

The sum of nitric oxide (NO) and NO_2 is commonly called nitrogen oxides or NOx. Other oxides of nitrogen including nitrous acid and nitric acid are part of the nitrogen oxide family. While EPA's National Ambient Air Quality Standard (NAAQS) covers this entire family, NO_2 is the component of greatest interest and the indicator for the larger group of nitrogen oxides.

NOx react with ammonia, moisture, and other compounds to form small particles. These small particles penetrate deeply into sensitive parts of the lungs and can cause or worsen respiratory disease, such as emphysema and bronchitis, and can aggravate existing heart disease, leading to increased hospital admissions and premature death.

Ozone is formed when NOx and volatile organic compounds react in the presence of heat and sunlight. Children, the elderly, people with lung diseases such as asthma, and people who work or exercise outside are at risk for adverse effects from ozone. These include reduction in lung function and increased respiratory symptoms as well as respiratory-related emergency department visits, hospital admissions, and possibly premature deaths.

Emissions that lead to the formation of NO_2 generally also lead to the formation of other NOx. Emissions control measures leading to reductions in NO_2 can generally be expected to reduce population exposures to all gaseous NOx. This may have the important co-benefit of reducing the formation of ozone and fine particles both of which pose significant public health threats.

Last updated on Thursday, March 22, 2012

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ARB Fact Sheet: Air Pollution Sources, Effects and Control

This page reviewed December 2, 2009.

Where does air pollution come from? How does it effect people and the environment? How can we control, or better yet, prevent it? The following table summarizes the sources, effects and prevention and control methods for ten of the most important air pollutants in California.

Pollutant	Sources	Effects	Prevention and Control
Ozone (O3)	Formed when reactive organic gases (ROG) and nitrogen oxides react in the presence of sunlight. ROG sources include any source that burns fuels, (e.g., gasoline, natural gas, wood, oil) solvents, petroleum processing and storage and pesticides.	Breathing Difficulties, Lung Tissue Damage, Damage to Rubber and Some Plastics	Reduce motor vehicle reactive organic gas (ROG) and nitrogen oxide emissions through emissions standards, reformulated fuels, inspections programs and reduced vehicle use. Limit ROG emissions from commercial operations and consumer products. Limit ROG and NOx emissions from industrial sources such as power plants and refineries. Conserve energy.
Respirable Particulate	Road Dust, Windblown	Increased Respiratory	Control Dust Sources,

Matter (PM10)	Dust (Agriculture) and Construction (Fireplaces) Also formed from other pollutants (acid rain, NOx, SOx, organics). Incomplete combustion of any fuel.	Disease, Lung Damage, Cancer, Premature Death, Reduced Visibility, Surface Soiling	Industrial Particulate Emissions, Wood Burning Stoves and Fireplaces Reduce secondary pollutants which react to form PM10. Conserve energy.
Fine Particulate Matter (PM2.5)	Fuel Combustion in Motor Vehicles, Equipment and Industrial Sources, Residential and Agricultural Burning. Also formed from reaction of other pollutants (acid rain, NOx, SOx, organics).	Increases Respiratory Disease, Lung Damage, Cancer, Premature Death, Reduced Visibility, Surface Soiling	Reduces Combustion Emissions from Motor Vehicles, Equipment, Industries and Agriculture and Residential Burning. Precursor controls, like those for ozone, reduce fine particle formation in the atmosphere.
Carbon Monoxide (CO)	Any source that burns fuel such as automobiles, trucks, heavy construction equipment, farming equipment and residential heating.	Chest Pain in Heart Patients, Headaches, Reduced Mental Alertness	Control motor vehicle and industrial emissions. Use oxygenated gasoline during winter months. Conserve energy.
Nitrogen Dioxide (NO2)	See Carbon Monoxide	Lung Irritation and Damage. Reacts in the atmosphere to form ozone and acid rain	Controls motor vehicle and industrial combustion emissions. Conserve energy.
Lead	Metal Smelters, Resource Recovery, Leaded Gasoline,	Learning Disabilities, Brain and Kidney Damage	Control metal smelters, no lead in gasoline. Replace leaded paint with non-lead

	Deterioration of Lead Paint		substitutes.
Sulfur Dioxide (SO2)	Coal or Oil Burning Power Plants and Industries, Refineries, Diesel Engines	Increases lung disease and breathing problems for asthmatics. Reacts in the atmosphere to form acid rain.	Reduces the use of high sulfer fuels (e.g., use low sulfer reformulated diesel or natural gas). Conserve energy.
Visibility Reducing Particles	See PM2.5	Reduces visibility (e.g., obscures mountains and other scenery), reduced airport safety, lower real estate value, discourages tourism.	See PM2.5
Sulfates	Produced by the reaction in the air of SO2 (see SO2 sources), a component of acid rain.	Breathing Difficulties, Aggravates Asthma, Reduced Visibility	See SO2
Hydrogen Sulfide	Geothermal Power Plants, Petroleum Production and Refining, Sewer Gas	Nuisance Odor (Rotten Egg Smell), Headache and Breathing Difficulties (Higher Concentrations)	Control emissions from geothermal power plants, petroleum production and refining, sewers, sewage treatment plants.

If you have questions or comments regarding this web page, please contact Barbara Weller at (916) 445-1324 or via email at blweller@arb.ca.gov.

ARB Fact Sheet

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Decisions Pending and Opportunities for Public Participation
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Cal/EPA | ARB | DPR | DTSC | OEHHA | SWRCB

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Have you ever wondered what a baghouse is or what NMOG stands for? That cold ironing is not a new way to get wrinkles out of a shirt or that a SIP isn't a beverage taste-test? You're not alone. ARB has updated its glossary of air pollution terms and lists of acronyms to help.

Keep in mind that we are not trying to create an exhaustive list, nor are we giving legal terminology. This glossary is simply a resource for the general public.

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

A

AB 1807 (Tanner)

A California state law (Health and Safety Code section 39650 et seq.) that became effective in January of 1984 and established the framework for California's toxic air contaminant identification and control program. For more information, please see our toxics summary.

AB 998

Assembly Bill 998 established the Non-Toxic Dry Cleaning Incentive Program to provide the dry cleaning industry with $10,000 grant funds to switch from systems using perchloroethylene (Perc), an identified toxic air contaminant and potential human carcinogen, to non-toxic and non-smog forming alternatives. The legislation also requires ARB to establish a demonstration program to showcase these non-toxic and non-smog forming technologies.

AB 2588 (Connelly) Air Toxics "Hot Spots" Information and Assessment Program

A California program (Health and Safety Code Section 44300 et seq.) that requires certain stationary sources to report the type and quantity of specific toxic substances they routinely release into the air. The program identifies high priority facilities and requires facilities posing significant risks to notify all exposed individuals. For more information, visit our AB 2588 website.

AB 2766 (Sher) Motor Vehicle Fee Program

A program that permits air districts and local governments to allocate vehicle registration surcharge fees to projects that reduce motor vehicle emissions such as zero-emission vehicles, bike lanes and trip reduction programs.

AB 32(The Global Warming Solutions Act of 2006)

The Legislature passed and Governor Schwarzenegger signed AB 32, which set the 2020 greenhouse gas emissions reduction goal into law. It directed ARB to develop discrete early actions to reduce greenhouse gases while also preparing a scoping plan to identify how best to reach the 2020 limit on greenhouse gas emissions.

Abatement

The reduction or elimination of pollution.

Acceptable Daily Intake (ADI)

The highest daily amount of a substance that may be consumed over a lifetime without adverse effects.

Acid Deposition

A workshop held by a public agency for the purpose of informing the public and obtaining its input on the development of a regulatory action or control measure by that agency.

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R

Radon

A colorless, naturally occurring, radioactive, inert gaseous element formed by radioactive decay of radium atoms in soil or rocks.

Reactive Organic Gas (ROG)

A photochemically reactive chemical gas, composed of non-methane hydrocarbons, that may contribute to the formation of smog. Also sometimes referred to as Non-Methane Organic Gases (NMOGs). (See also Volatile Organic Compounds and Hydrocarbons.)

Reactivity (or Hydrocarbon Photochemical Reactivity)

A term used in the context of air quality management to describe a hydrocarbon's ability to react (participate in photochemical reactions) to form ozone in the atmosphere. Different hydrocarbons react at different rates. The more reactive a hydrocarbon, the greater potential it has to form ozone.

Reasonably Available Control Measures (RACM)

A broadly defined term referring to technologies and other measures that can be used to control pollution. They include Reasonably Available Control Technology and other measures. In the case of PM10, RACM refers to approaches for controlling small or dispersed source categories such as road dust, woodstoves and open burning.

Reasonably Available Control Technology (RACT)

Control techniques defined in U.S. EPA guidelines for limiting emissions from existing sources in nonattainment areas. RACTs are adopted and implemented by states. For more information, visit our RACT website.

Reasonably Available Retrofit Control Technology (RARCT)

(See also Best Available Control Technology.)

Reciprocating Internal Combustion Engine

An engine in which air and fuel are introduced into cylinders, compressed by pistons and ignited by a spark plug or by compression. Combustion in the cylinders pushes the pistons sequentially, transferring energy to the crankshaft, causing it to rotate.

Reference Dose (RfD)

An estimate delivered by the U.S. EPA (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population, (including sensitive subpopulations) that is likely to be without deleterious effects during a lifetime. The RfD is reported in units of mg of substance/kg body weight/day for oral exposures.

Reference Exposure Concentration (RfC)

An estimate, derived by the U.S. EPA with an uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population, (including sensitive subgroups) that is likely to be without

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http://www.epa.gov/o3healthtraining/population.html
Last updated on Friday, February 03, 2012

Ozone and Your Patients' Health
Training for Health Care Providers

Health Effects of Ozone in the General Population

Review Key Points

- Introduction
- How are people exposed to ozone?
- How does ozone react in the respiratory tract?
- What are ozone's acute physiological and symptom effects?
- What effects does ozone have at the cellular level?
- How does response vary among individuals?
- What are the effects of ozone on mortality?
- What are other potential effects of short-term ozone exposure?
- At what exposure levels are effects observed?
- What are the effects of recurrent or long-term exposure to ozone?

Introduction

Breathing ground-level ozone can result in a number of health effects that are observed in broad segments of the population. Some of these effects include:

- Induction of respiratory symptoms
- Decrements in lung function
- Inflammation of airways

Respiratory symptoms can include:

- Coughing
- Throat irritation
- Pain, burning, or discomfort in the chest when taking a deep breath
- Chest tightness, wheezing, or shortness of breath

In addition to these effects, evidence from observational studies strongly indicates that higher daily ozone concentrations are associated with increased asthma attacks, increased hospital admissions, increased daily mortality, and other markers of morbidity. The consistency and coherence of the evidence for effects upon asthmatics suggests that ozone can make asthma symptoms worse and can increase sensitivity to asthma triggers.

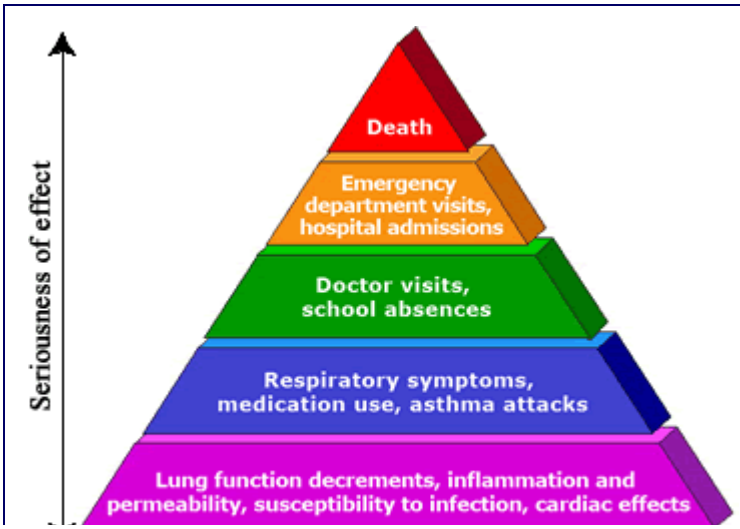
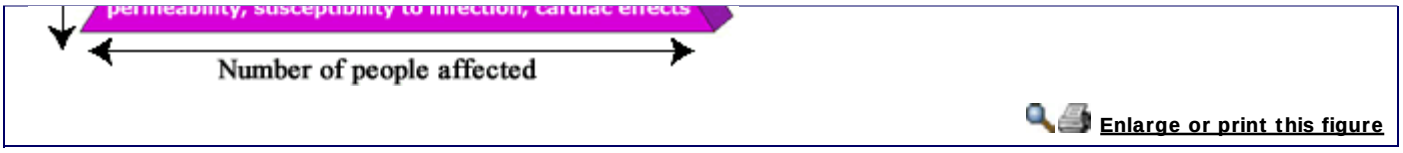


Figure 2: Pyramid of effects caused by ozone
The relationship between the severity of the effect and the proportion of the population experiencing the effect can be presented as a pyramid. Many individuals experience the least serious, most common effects shown at the bottom of the pyramid. Fewer individuals experience the more severe effects such as hospitalization or death.



This section of the course addresses exposure and health effects issues common to all people. The next section of the course, Health Effects in Patients with Asthma and Other Chronic Respiratory Disease, addresses those issues specific to people with asthma and other chronic lung disease.

How are people exposed to ozone?

Primary exposure occurs when people breathe ambient air containing ozone. The rate of exposure for a given individual is related to the concentration of ozone in the surrounding air and the amount of air the individual is breathing per minute (minute ventilation). The cumulative amount of exposure is a function of both the rate and duration of exposure.

Although ozone concentrations in the outside (ambient) air are generally similar across many locations in a particular airshed, a number of factors can affect ozone concentration in "microenvironments" within the larger airshed (e.g., inside a residence, inside a vehicle, along a roadway). Ozone concentrations indoors typically vary between 20% and 80% of outdoor levels depending upon whether windows are open or closed, air conditioning is used, or other factors such as indoor sources. People with the greatest cumulative exposure are those heavily exercising outdoors for long periods of time when ozone concentrations are high. In addition, during exercise people breathe more deeply, and ozone uptake may shift from the upper airways to deeper areas of the respiratory tract, increasing the possibility of adverse health effects. People with the lowest cumulative exposure are those resting for most of the day in an air-conditioned building with little air turnover.

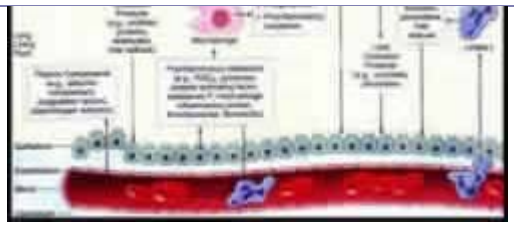
Ozone levels may also affect indoor levels of some aldehydes formed as reaction products of ozone with indoor substances (Apte et al 2008). This provides a potential pathway for people indoors to experience respiratory effects mediated by ozone reaction products. Further research is needed to test the importance of these exposures on health effects.

How does ozone react in the respiratory tract?

Because ozone has limited solubility in water, the upper respiratory tract is not as effective in scrubbing ozone from inhaled air as it is for more water soluble pollutants such as sulfur dioxide (SO_2) or chlorine gas (Cl_2). Consequently, the majority of inhaled ozone reaches the lower respiratory tract and dissolves in the thin layer of epithelial lining fluid (ELF) throughout the conducting airways of the lung.

In the lungs, ozone reacts rapidly with a number of biomolecules, particularly those containing thiol or amine groups or unsaturated carbon-carbon bonds. These reactions and their products are poorly characterized, but it is thought that the ultimate effects of ozone exposure are mediated by free radicals and other oxidant species in the ELF that then react with underlying epithelial cells, with immune cells, and with neural receptors in the airway wall. In some cases, ozone itself may react directly with these structures. Several effects with distinct mechanisms occur simultaneously following a short-term ozone exposure and will be described below.

Figure 3: Ozone is highly reactive in the respiratory tract
When breathed into the airways, ozone



interacts with proteins and lipids on the surface of cells or present in the lung lining fluid, which decreases in depth from 10 µm in the large airways to 0.2 µm in the alveolar region. Epithelial cells lining the respiratory tract are the main target of ozone and its products. These cells become injured and leak intracellular enzymes such as lactate dehydrogenase into the airway lumen, as well as plasma components. Epithelial cells also release a variety of inflammatory mediators that can attract polymorphonuclear leukocytes (PMNs) into the lung, activate alveolar macrophages, and initiate a train of events leading to lung inflammation. Antioxidants present in cells and lining fluid may protect the epithelial barrier against damage by ozone or its reaction products.
Source: Devlin et al., (1997)

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What are ozone's acute physiological and symptom effects?

The predominant physiological effect of short-term ozone exposure is being unable to inhale to total lung capacity. Controlled human exposure studies have demonstrated that short-term exposure - up to 8 hours - causes lung function decrements such as reductions in forced expiratory volume in one second (FEV1), and the following respiratory symptoms:

- Cough
- Throat irritation
- Pain, burning, or discomfort in the chest when taking a deep breath
- Chest tightness, wheezing, or shortness of breath

The effects are reversible, with improvement and recovery to baseline varying from a few hours to 48 hours after an elevated ozone exposure.

Current thinking is that changes in symptoms and lung function are due to stimulation of airway neural receptors (probably airway C-fibers) and transmission to the central nervous system via afferent vagal nerve pathways. Although ozone exposure results in some airway narrowing, neural inhibition of inhalation effort at high lung volumes is believed to be the primary cause of being unable to inhale to total lung capacity.

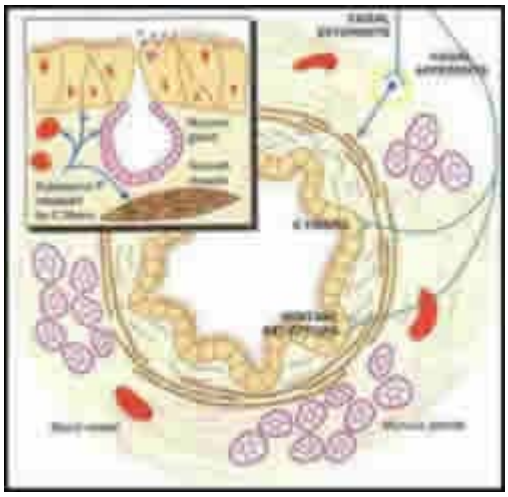

Figure 4: Ozone induces neurally mediated responses in the bronchial airways
Stimulation of nociceptive interepithelial nerve fibers by ozone leads to reflex cough and a decrease in maximal inspiration that is relieved by opioid agonists, which block sensory pathways. Two possible mechanisms are involved: (1) stimulation of irritant receptors contributes to cough and induces a vagally mediated reflex that increases airway resistance, probably via airway smooth muscle contraction that is blocked by atropine; (2) C fiber stimulation releases neurokinins such as substance P that dilate nearby capillaries, activate mucous glands, and contract airway smooth muscle via neurokinin receptors. Prostaglandin E2 released by epithelial cells exposed to ozone or to ozone reaction products also sensitizes C fibers.
Source: Devlin et al. (1997)

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The overall effect is thus primarily restrictive in nature with a smaller obstructive component that reflects itself in decreases in forced vital capacity (FVC), FEV1 and other spirometric measures that require a full inspiration. It is likely that these lung function changes and respiratory symptoms are responsible for observations that short-term ozone exposure limits maximal exercise capability.

Ozone-induced changes in breathing pattern to more rapid shallow breathing may also be a manifestation of C-fiber stimulation and may be a protective response to limit penetration of ozone deep into the respiratory tract. Such effects may also contribute to changes in deposition pattern and retention of other inhaled substances such as allergens and particle pollution (also called particulate matter).

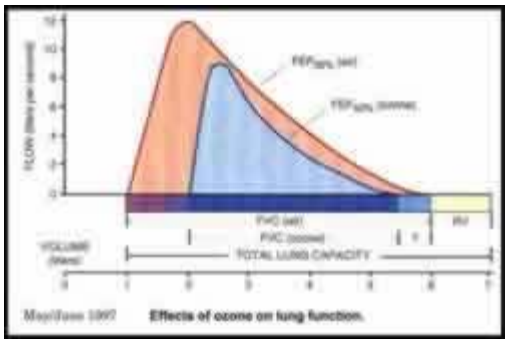

Figure 5: Effects of ozone on lung function
Ozone reduces the maximal inspiratory position (at the left of the curves) and may slightly increase the residual volume (at the right). Reduction in maximum inspiration reduces forced vital capacity (FVC), and this causes a reduction in expiratory flow measurements, such as flow at 50% of FVC expired (FEF50%). Because ozone causes only a small change in resistance, the relationship between flow and volume is not changed to a large extent.
Source: Devlin et al. (1997)

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What effects does ozone have at the cellular level?

As a result of short-term exposure, ozone and/or its reactive intermediates cause injury to airway epithelial cells followed by a cascade of other effects. These effects can be measured by a technique known as bronchoalveolar lavage (BAL), in which samples of epithelial lining fluid (ELF) are collected during bronchoscopy on volunteers experimentally exposed to ozone. Cells and biochemical markers in the lavage fluid and in the blood can be analyzed to provide insight into the effects of exposure.

Evidence for airway inflammation following ozone exposure includes visible redness of the airway seen during bronchoscopy as well as an increase in the numbers of neutrophils in the lavage fluid. Cellular injury is suggested by an increase in the concentration of lactate dehydrogenase (LDH), an enzyme released from the cytoplasm of injured epithelial cells, in the ELF. Mediators (e.g., cytokines, prostaglandins, leukotrienes) that are released by injured cells include a number that attract inflammatory cells resulting in a neutrophilic inflammatory response in the airway. In addition, ozone reaction products as well as some mediators produced in the lung can be detected in the blood providing a possible mechanism for extrapulmonary effects of ozone exposure.

Figure 6: Effects of ozone on lung function
These photos show a healthy lung airway (left) and an inflamed lung airway (right). *Photos courtesy of PENTAX Medical Company.*

Enlarge or print this figure

Other documented ozone-induced effects that may be related to the underlying injury and

inflammatory response are:

- An increase in small airway obstruction
- A decrease in the integrity of the airway epithelium
- An increase in nonspecific airway reactivity
- A decrease in phagocytic activity of alveolar macrophages

The decrease in epithelial integrity can be measured by an increase in the concentration of plasma proteins appearing in the ELF following exposure and by more rapid clearance of inhaled radio-labeled markers from the lung to the blood. This has the potential for allowing increased movement of inhaled substances (e.g. allergens or particulate air pollution) from the airway to the interstitium or the blood and could modify the known effects of inhaled allergen on asthma and particulate matter on mortality.

Although the significance of increased nonspecific airway reactivity to substances such as methacholine or histamine is not understood in healthy individuals, it is clearly of concern for people with asthma, as increased airway reactivity is a predictor for asthma exacerbations. (See section entitled How does ozone affect people with asthma?).

A decrease in macrophage function has the potential to interfere with host defense. Over a period of several days following a single short-term exposure, inflammation, small airway obstruction, and increased epithelial permeability resolve; damaged ciliated airway epithelial cells are replaced by underlying cells; and damaged type I alveolar epithelial cells are replaced by more ozone-resistant type II cells. Over a period of weeks, the type II cells differentiate into type I cells, and following this single exposure, the airway appears to return to the pre-exposure state.

How does response vary among individuals?

One striking characteristic of the acute responses to short-term ozone exposure is the large amount of variability that exists among individuals. For example, for a 2-hour exposure to 40 ppb ozone (note: 40 ppb is equal to .04 ppm) that includes 1 hour of heavy exercise, the least responsive individual may experience no symptom or lung function changes while the most responsive individual may experience a 50% decrement in FEV1 and have severe coughing, shortness of breath, or pain on deep inspiration. A similar range of response is evident for a 6.6-hour exposure to 80 ppb with 5 hours of moderate activity. Other individual responses fall into what appears to be a unimodal distribution between these two extremes. Those with large responses following exposure on one day also tend to have large responses upon re-exposure. Similarly, those with small responses following exposure on one day tend to have small responses upon re-exposure. A small fraction of the observed variability in lung function and symptom responsiveness can be explained by differences in age and in body mass index (BMI) with young adults (teens to thirties) and those with high BMI being much more responsive than older adults (fifties to eighties) and those with low BMI. Results similar to those in Figure 8 are also seen with longer duration exposures to concentrations more relevant to ambient levels (e.g. over a range of 60 to 120 ppb).

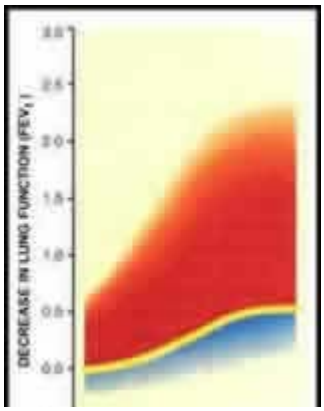


Figure 7: Variability of response to ozone exposure
Source: Devlin et al. (1997)

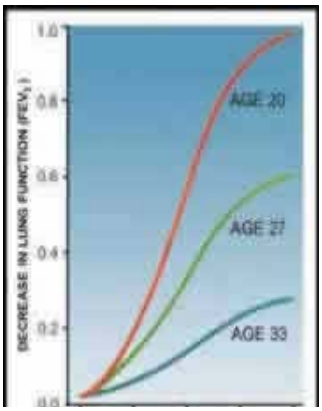
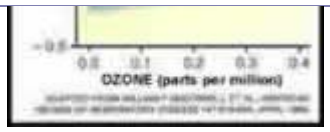
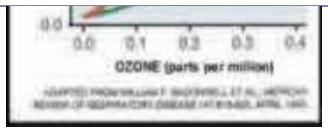


Figure 8: Sensitivity to ozone exposure is age related
Source: Devlin et al. (1997)





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Individual differences in the intensity of the inflammatory response also exist, and it appears that these differences in response are also stable over time. The magnitude of the neurally-mediated lung function response, however, is not related to the degree of cell injury and inflammation for a given individual suggesting that these two effects are the result of different mechanisms of action. Further evidence for multiple mechanisms of action is provided by drug intervention studies. There is some evidence that Vitamin C and E supplements may slightly reduce the lung function effects of ozone but not the inflammatory or symptom responses. Pre-treatment with non-steroidal anti-inflammatory drugs (NSAID) reduces lung function and symptom responses but not the inflammatory responses in non-asthmatics. In asthmatic volunteers NSAID pretreatment did not block the restrictive lung function changes seen in nonasthmatics, but did blunt some of the changes due to airway obstruction. Pre-treatment with high doses of inhaled steroids has been shown to reduce the neutrophil influx following ozone exposure in people with asthma, but not in those without asthma.

True differences in individual responsiveness to ozone can be the result of either environmental or genetic factors. Research has demonstrated that genetic differences among strains of mice can explain the large range of inflammatory responses seen. Some preliminary evidence suggests that genetic polymorphisms for antioxidant enzymes and for genes regulating the inflammatory response may modulate the effect of ozone exposure on pulmonary function and airway inflammation.

What are the effects of ozone on mortality?

Studies show:

- Ozone is associated with increased mortality
- The absolute effect of ozone on mortality is considerably higher in older adults
- The ozone-mortality relationship is most prominent during the warm season

Recent epidemiologic research has clearly demonstrated that both short-term and longer-term exposures to low concentrations of particle pollution, a common air pollutant, are associated with increased mortality. Re-examination of the data upon which those findings are based as well as new studies indicate that short-term exposure to ozone is also associated with increased daily mortality.

The study most representative of the U.S. population (Bell et al 2004) evaluated the relationships between daily mortality counts and ambient ozone concentration for 95 large U.S. communities over the period of 1987-2000. Although there was considerable heterogeneity in the magnitude of effect among the various communities, a 0.5 % overall excess risk in non-accidental daily mortality was observed for each 20 ppb increase in the 24-hour average ozone concentration (approximately equal to a 30 ppb increase in the 8-hour average) on the same day. There was evidence that the effect was greatest on the day of exposure with smaller residual effects being evident for several days. A cumulative 1.04% excess risk was observed for each 20 ppb increase in the 24-hour average concentration during the previous week. The ozone-mortality relationship was robust even after controlling for possible effects of particulate matter and other air pollutants.

Although ozone mortality risk estimates tend to be only slightly higher for the older population compared to the younger population (based predominantly on Medicare studies of people 65 and older), the absolute effect of ozone on mortality is considerably higher in older adults due to their

higher baseline death rates. Even for older adults, however, the risk of dying on any given day as a result of ozone exposure is quite small. However, because of the large number of individuals at risk across the country, an effect of this magnitude has meaningful public health implications.

A preponderance of other time series studies supports the existence of an ozone-mortality relationship although with a wider range of effect estimates primarily due to the smaller sizes of the studies. An independent review of this literature by the National Research Council concludes that short-term ozone is likely to be associated with premature mortality.

Other observations made in these studies include the finding that the ozone-mortality relationship is most prominent during the warm season, with few or smaller effects in the winter. It also appears that the ozone-mortality association persists when deaths are limited to those caused by either cardiac or pulmonary disease or to those caused by cardiovascular disease alone. Risk estimates for other causes of death are generally inconsistent across studies probably reflecting the lower statistical power associated with smaller daily death rates. In the Bell study of 95 cities, the observed city-specific effect rates varied widely. The degree to which this variability reflects different ozone-mortality relationships in the different cities is not clear, but it does raise the question as to whether a single average 0.5% increase in daily mortality rates should be applied to all cities. Other unanswered questions pertain to the lowest concentrations at which these effects occur and the possible mechanisms of action responsible for increased mortality among many who spend much of their time indoors where ozone levels are generally quite low. Bell et al. divided days into those with a 24-hour average ozone concentration above and below 60 ppb and found that the relationship was similar for both subsets suggesting that the relationship is present at even very low levels of ozone. Biological mechanisms responsible for the ozone-mortality relationship are largely unknown although effects of ozone on the autonomic control of the cardiovascular system, on coagulation mechanisms, and on vasoactive substances in the blood are being actively investigated.

What are the other potential effects of short-term ozone exposure?

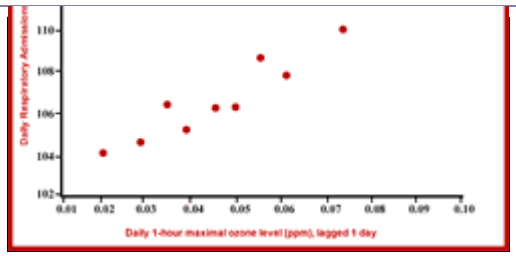
Other potential effects of short-term ozone exposure include:

- hospital admissions and emergency room visits for respiratory causes
- school absences

There is consistent epidemiologic evidence that ambient ozone levels are associated with other markers of respiratory morbidity, particularly during the warm season. In general, studies have reported positive relationships between short-term ozone concentrations and hospital admissions and emergency room visits for respiratory causes. Although not all studies have found significant effects, risk estimates for the majority of studies are positive. It is likely that those most at risk of serious respiratory morbidity are those with underlying respiratory disease. The evidence indicates that some of the increase in hospital visits for respiratory morbidity is due to exacerbations of asthma and possibly chronic obstructive pulmonary disease (COPD). Because of the small numbers of daily hospital admissions, the effects of ozone on other subcategories of respiratory disease are not clear.

A relationship has also been observed between ozone and school absences in two studies. However, in one case the absences were related to a measure of longer-term exposure, and in the other case absences were not limited to those due to illness. Although these latter results are consistent with increased infections secondary to impaired host defense, more research needs to be done before reaching any conclusion regarding any effect of ozone exposure on respiratory infection.

Figure 9: The number of emergency or urgent daily respiratory admissions to acute care hospitals is related to



estimated ozone exposure
Respiratory admission rates to 168 hospitals in Ontario, Canada during the period 1983 through 1988 are plotted against the distribution (deciles) of the daily 1-hour maximum ozone concentration, lagged by 1 day. Admission rates were adjusted for seasonal patterns, day-of-week effects, and hospital effects. Ozone displayed a positive and statistically significant association with respiratory admissions for 91% of the hospitals during the Spring through Fall seasons, but not during the Winter months of December to March when ozone levels were low. *Source: Burnett et al., 1994; U.S. EPA, 1996*

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Ozone has been associated with daily hospitalizations for cardiovascular disease in some studies but it is not a consistent finding. A number of studies have explored the relationships between ozone and various other aspects of cardiovascular pathophysiology including heart rate variability, acute myocardial infarction, and tachyarrhythmias in those with implanted cardiac devices. Although some data are suggestive of a relationship, the results at this time do not fully substantiate a relationship between ozone exposure and adverse cardiovascular events.

At what exposure levels are effects observed?

The concentration of ozone at which effects are first observed depends upon the level of sensitivity of the individual as well as the dose delivered to the respiratory tract. The dose, in turn, is a function of the ambient concentration, the minute ventilation, and the duration of exposure. This can be expressed as a rough formula:

Dose = Ambient concentration X Level of exertion (minute ventilation) X Duration of exposure.

Thus individuals performing strenuous activity (higher minute ventilation) for several hours are likely to respond to lower concentrations than when exposed at rest (lower minute ventilation) for a shorter time. The following examples illustrate this point:

- An average young adult playing an active sport such as soccer or full court basketball outdoors for 2 hours would be expected to experience small to moderate lung function and symptom effects as well as lung injury and inflammation following exposure to 120 ppb ozone.
- If the same average young adult is at rest outdoors for the two hours, such effects would not be expected until exposures reach 300-400 ppb.
- An average outdoor laborer doing intermittent work might experience similar small to moderate lung function and symptom effects as well as lung injury and inflammation following an 8-hour exposure to 60 to 70 ppb ozone.

More sensitive individuals will experience such effects at lower concentrations while less sensitive individuals will experience these effects only at higher concentrations.

Children without asthma experience lung function decrements similar to those of young adults. But children often do not report respiratory symptoms at the lowest ozone concentrations. It is not clear whether this is the result of reduced sensitivity with regard to symptoms or whether children are less likely to recognize and report symptoms.

There are chamber studies and field studies that look at the ozone exposure level at which effects are first observed. It is not surprising that field studies show effects at much lower levels than chamber studies. This is because field studies can look at sensitive populations (including children), include exposure to all oxidant species of pollution, and may include longer exposure times. For

example, field studies of agricultural workers and hikers suggest that lung function changes may be associated with prolonged ozone exposures at lower levels than those observed in chamber studies. Below are findings from key field and observational studies.

Although the results vary somewhat, several field studies suggest that the lung function of highly active asthmatic and ozone sensitive children and the exercise performance of endurance athletes may be affected on days when the 8-hour maximum ozone concentration is less than 80 ppb ozone.

Emergency room data from one study indicate that asthma attacks in the most sensitive population (e.g., children with asthma or reactive airway disease) increase following days on which the 1-hour maximum ozone concentrations exceeded 110 ppb (approximately equivalent to an 8-hour average of 82 ppb). (White et al., 1994) Another study observed increased emergency room visits for asthma on days following those when 7-hour averages exceeded 60 ppb compared to those with lower ozone concentrations. (Weisel et. al., 1995).

For effects measured in some other types of observational studies, the lowest levels at which effects are expected to occur are more difficult to identify for a number of reasons. Effects of ozone on daily mortality have been detected even when study days are restricted to those with a 24-hour average ozone concentration below 60 ppb (approximately equivalent to an 8-hour average below 90 ppb). In one study, hospital admissions for respiratory causes appear to follow a linear relationship down to background levels. (Figure 9). Limited exposure-response modeling suggests that if a population threshold for these ozone effects exists, it is likely near the lower limit of ambient ozone concentrations in the United States.

What are the effects of recurrent or long-term exposure to ozone?

One of the major unanswered questions about the health effects of ozone is whether repeated episodes of damage, inflammation, and repair induced by years of recurrent short-term ozone exposures result in adverse health effects beyond the acute effects themselves.

Daily ozone exposure for a period of 4 days results in an attenuation of some of the acute, neurally-mediated effects (e.g., lung function changes and symptoms) for subsequent exposures occurring within 1 to 2 weeks. Some health experts have, therefore, suggested that individuals living in high ozone areas may be protected from any harmful effects of long-term ozone exposure. Others suggest, however, that the attenuation of the ozone-induced tendency to take rapid and shallow breaths may blunt a protective mechanism, resulting in greater delivery and deposition of ozone deeper in the respiratory tract and other airway responses described below.

Studies including bronchoalveolar lavage and bronchial mucosal biopsies indicate that, unlike the neurally-mediated lung function changes, the processes of airway injury, inflammation, and repair continue to occur during repeated exposure. After either 4 or 5 days of exposure, markers of cell injury and increased epithelial permeability remain elevated, and an increase in airway mucosal PMN, which was not present following a single exposure, has been noted. Also, unlike the neurally-mediated effects, small airway function has been observed to remain depressed over the course of exposures and is thought to be related to the ongoing inflammation.

Studies of laboratory animals have consistently demonstrated that long-term exposure to ozone concentrations above ambient levels results in persistent morphological changes that could be a marker of chronic respiratory disease. Exposed animals experience mucous cell metaplasia and epithelial cell hyperplasia in the upper airway as well as structural changes in the lower airway including an increase in fibrous tissue in the basement membrane area and a remodeling of the distal conducting airways. In addition to airway remodeling and basement membrane changes, concurrent long-term exposure of very young primates to ozone and house dust mite allergen has been observed to result in changes in the innervation of the airways as well as an accumulation of

eosinophils in the distal airways suggesting induction of an allergic phenotype. Other studies indicate that sensitization of animals to antigen occurs more easily during ongoing ozone exposures. Based on traditional measures, there is little evidence that long-term exposure in animals results in substantial changes in airway function. However, these morphological findings suggest that long-term ozone exposure might play a role in the development or progression of chronic lung disease and/or asthma.

The epidemiologic evidence is inconclusive with regard to whether long-term exposure of humans is related to chronic respiratory health effects in humans. Several cross-sectional studies have found that young adults who spent their childhoods in locales with high ozone concentrations had lower measures of lung function than those from locales with lower ozone. Similar results have not been observed, however, in a recent well-conducted longitudinal study of lung function in children or in other cross-sectional studies. Two longitudinal studies have observed associations between development of asthma and long-term ozone concentrations in subgroups of the population. These findings have not been confirmed in other longitudinal or cross-sectional studies, but they are consistent with the animal toxicological literature. Part of the difficulty in evaluating such associations has been the small number of longitudinal epidemiologic studies specifically designed to evaluate respiratory health in samples with differing ozone exposures. The mobility of the population as well as the inability to precisely estimate exposure to ozone and other potential confounders over a period of many years degrades the power of, and leads to bias in, both longitudinal and cross-sectional studies.

In spite of the inconclusive nature of the epidemiologic literature, the repeated cycles of damage, inflammation, and repair in humans and the morphological findings from the animal toxicological studies suggest that it would be prudent to avoid repeated short-term exposures, particularly in young children, until more is known about the effects of long-term ozone exposure.

Review Key Points

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We often do not drive a car thinking of all the hazardous emissions we would release. Nor do we turn on the light with thought as to how that energy was generated. Yet, when we do all these activities subconsciously, we indirectly contribute to the growing amounts of hazardous air pollutants in our atmosphere that are responsible for many adversities.

Smog as a Negative Externality

Depending upon various factors, including location, season, and source of generation, the price an entity pays for a given amount of energy can vary. Typically, the price would account for all costs incurred within the value chain-research and development, design, production, marketing, distribution, and customer services-plus a markup. Unfortunately, this price, the cost charged to consumers, is what is known as the market price, and therefore may not accurately reflect the total costs inflicted upon society as a whole (Baird). In such a case as where the actions of one party directly affecting another are not accounted for, an externality arises.

In the case of energy, both productive and consumptive activities result in smog, a negative externality imposed on the environment and the welfare of society. The production of an output of energy through the process of burning coal or other fossil fuels, for example, releases two main air pollutants: sulfur dioxide and nitrogen oxides. Similarly, the consumption of energy-either for self-consumptive or other productive purposes-releases primary pollutants VOCs and nitrogen dioxides, which in turn can undergo chemical reactions to yield secondary pollutants such as ground level ozone and PAN. All these air pollutants are responsible for adverse effects in both humans and plants and on materials and aesthetics, as well as the negative impacts on the environment, namely acid rain. In whichever case, the stated price of either energy or an intermediate form of energy, such as gas, or a finished output that uses energy as an input, rarely reflects the complete burden placed upon society. This neglect of externalities, in turn, often results in an over-production or over-consumption of energy and other related goods. Here, we take a closer look at these externalities as to see what costs to society the market fails to account for.

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Smog and its Effects on Human Health

We, as humans, can live a few days without food and water, but can only live a few minutes without air. The fact that an active adult inhales 10,000 to 20,000 liters of air each day, or 7 to 14 liters every minute, highlights a critical point in the fight for clean air (Elsom 30).

While the effects of smog vary according to factors such as age, state of health, time of exposure, and dosage, the general symptoms include coughing, sneezing, headaches, tiredness, irritation, nausea, and hoarseness of the throat, nose, and eyes, and constrictions of the chest (Lewis 37; Elsom 31).

Additionally, nitrogen dioxide and ground-level ozone were found to cause reductions in the immune system's ability to fight bacteria and viruses in the respiratory system (Nebel and Wright 530; EPA, "Smog-Who Does it Hurt?" 3). These effects are all considered to be short-term in that once exposure ceases, the symptoms are no longer present. However, in most cases, it is the long-term effects of air pollutants that bring the greatest concerns, since these effects are often the most severe.

Unsurprisingly, most acute effects of smog are related to the respiratory system. Some components of smog such as nitrogen dioxide, sulfur dioxide, and ground-level ozone are found to have caused damages to the mucociliary system responsible for cleaning the air tracts (Elsom 56). As a result, the lung's ability to resist disease is reduced, and illnesses, such as bronchitis and emphysema, can be aggravated (Gow and Pidwirny; Elsom 56). Likewise, while some VOCs were found to be carcinogenic, the main problem with VOCs was its role in the formation of ground-level ozone. Present in ambient concentrations, ground-level ozone can cause inflammation and fibrosis to the lungs, resulting in permanent morphological changes to the lungs (Nebel and Wright 530; EPA, "Smog-Who Does it Hurt?" 3). Consequently, these air pollutants can not only decrease lung function, elasticity, and capacity by as much as 5%, but can also lead to the premature aging of the lungs (Elsom 33, 63; "Smog").

While continuous research is being made as to link the long-term effects of smog to human health, scientists in general have agreed on several findings. By and large, children, asthmatics, people with chronic respiratory or pulmonary and heart disease, and the elderly are the most susceptible to air pollutants (Nebel and Wright 530). Because the lungs of children are not yet fully developed and because children inhale more air per unit of body weight than adults, they are prone to greater health effects as well as long-term damage to the lungs (Elsom 42). Similarly, because asthmatics and those suffering from chronic diseases are already in a weakened state, smog adds stress to their bodies (Nebel and Wright 532). For the elderly, smog increases their susceptibility to viral and bacterial attacks, as both lung and immune system functions decrease with age (Elsom 42). Healthy adults who work actively outdoors or who have higher levels of exposure to air pollutant are also considered, by the EPA, to be in a "sensitive group" (EPA, "Smog-Who Does it Hurt?" 3).

In all these cases, it is important to note that contrary to popular belief, death as a result of a smog siege is often not a result of air pollutant poisoning, but rather, a result of increasing susceptibility to diseases. Equally important, however, is the fact that a great level of uncertainty exists in identifying a cause-and-effect relationship between smog and smog-related illnesses. At most, we can often only say that pollutants are contributing factors to related illnesses. Consequently, this makes the exact measurements of externalities difficult, if not impossible.

Estimates have been made, however, to provide a monetary value of the costs and benefits of smog. In several studies conducted by the American Lung Association, the costs of premature deaths, hospital stays and emergency room visits, productivity loss as a result of missing work or school, and other air pollutant related health effects were an indication of inefficiency within the economy ("Air"). The reports went so far as to argue that economic growth was correlated with environmental protection by demonstrating that human health benefits of cleaner air outweighed the costs industries would have to incur as a result of higher standards (ibid). It was estimated that enforcement of all parts of the Clean Air Act between 1970 and 1990 would result in minimum benefits of $23 trillion over the twenty years, an average of over $1 trillion annually (ibid).

In a similar study conducted by the EPA for United States Congress in 1999, it was estimated that if the Clean Air Act Amendments were enforced in the 48 contingent states for the twenty-year period between 1990 and 2010, the total human health benefits in 2000 would be $68 billion and $118 billion in 2010 (EPA, "The Benefits and Costs" H-27). These benefits represent underestimates, since, in the words of the EPA itself, "there is insufficient information from both the medical and the economic sciences to satisfactorily resolve these issues from a theoretically/analytical standpoint" (ibid. H-36).

Apparently, smog is a costly externality from a human health perspective alone.

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Smog and its Effects on Agriculture and Forests

The adverse effects of smog are not limited to humans alone. As it turns out, plants are perhaps more sensitive to air pollutants than humans. In particular, acid rain has left areas barren or with severely damaged vegetation (Nebel and Wright 533). Yet, perhaps the greatest damage has been from ground-level ozone and PAN. Entering leaves of plants from the stomata during normal gas exchange, both ground-level ozone and PAN can cause discoloration, damage, and loss of leaves-reducing photosynthesis by as much as 50% (Munster; Gow and Pidwirny). Plants also become more vulnerable to attacks by pests, disease, and other environmental disasters (Shaw). Consequently, the plant's ability to store food, grow, and reproduce is hindered (ibid).

In numerical terms, ground-level ozone, alone, has been estimated to cause 10% to 40% growth loss, premature aging, and a decrease in pollen lifespan resulting in an estimable cost in agriculture of $2 billion to $6 billion per year (Nebel and Wright 533; "Smog"). Losses in crop yields were estimated to be 20% to 30% between 1989 and 1992 (Nebel and Wright 533). In Ontario alone, smog was attributed to reduce crop yields equivalent to $70 million per year ("Smog"). In a study conducted by the EPA to Congress, continuous implementation of a Clean Air Act Amendments over the period 1990 to 2010 would accumulate a minimum 1999 net present value of agricultural benefits of at least $4 billion (EPA, "The Benefits and Costs" F-8). Along with the fact that 60% of the world's food is produced in countries that also produce 60% of the world's air pollution, the significance of clean air is clearly seen (Nebel and Wright 533).

In a forestry aspect, smog incurs a cost on the existence value of trees and wild plants. In Los Angeles, smog was attributed to the deaths of 50% of trees in nearby areas (ibid.). Similarly, ground-level ozone from the Central Valley and San Francisco-Oakland metropolitan areas was responsible for increasing stress and vulnerability on the ponderosa and Jeffrey pines in the Sierra Nevada (ibid.). An attack by western pine beetles subsequently diminished the number of these trees.

As it perhaps can be predicted, the monetary costs of the loss of forests are difficult to measure, if measurable at all. Yet, it may still be worthwhile to keep in mind the option value benefits, non-consumptive use benefits, and existence value of forests, when making a balance sheet of costs and benefits of reducing smog. In another aspect, the damage to trees can have direct economic costs-as Canada discovered when it was found that ground-level ozone was the cause of damage to its sugar maple trees and other trees in its forestry industry ("Smog").

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Smog and its Effects on Materials and Aesthetics

It is said that cleaning is just as destructive as it is costly. Perhaps this is even more so when considering the material and aesthetic aspects of smog. Besides the fact that most people derive a psychological benefit of seeing a clear sky and a clean surrounding, the costs of smog can be millions of dollars.

The most visible characteristic of cities smothered by smog is perhaps the black and soot-covered windows, walls, drapes and curtains, and other exposed surfaces. Yet, other damages can be seen. Sulfur dioxide corrodes metal and stone-damaging machinery and industrial instruments, as well as destroying buildings, statues, and monuments (Lewis 33; EPA, "The Plain English"). Ground-level ozone, destroying synthetic materials, can cause leather to become brittle and rubber to lose its elasticity, resulting in

cracks (Lewis 33). Moreover, ground-level ozone has been found to damage cotton, acetate, nylon, polyester, and other textiles, while bleaching dyes, paints, and coatings ("Smog").

While it is uncertain as to how much is exactly spent on the cleaning or replacement of materials, a couple of million dollars is considered to be a reasonable estimate. Canada, alone, estimates that the increase in ground-level ozone from the United States has cost it up to one billion dollars in material damages ("Smog"). Considering that cleaning and replacement costs do not include materials that are irreplaceable and the observation that people have actually spent more to move further away from cities, these costs of pollution most likely will be underestimates (Nebel and Wright 534).

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Smog and its Effects on Ecological Systems

Sulfur dioxide and nitrogen oxides are largely responsible for the sources of acid precipitation. Because it results in acid rain with a pH of 5.5 or less, smog can have serious widespread ecological impacts on aquatic systems, forests, and on humans far away from its point of origin.

As a basic biology course will explain, slight deviations from pH values in the environment can be critical to the proper functioning of enzymes, hormones, and other proteins. In aquatic systems with a normal pH of 6 to 8, a slight deviation in most cases will pose no threat, as organisms adapt (Nebel and Wright 541). However, an organism's ability to successfully reproduce may be hindered, and in more extreme cases, a population of an organism may actually become extinct (ibid.). In forests, acid precipitation not only damages trees and plants, but also affects soil contents, which can thwart growth towards acid-tolerant species (ibid. 542). For humans, the effects of acid rain may vary from aesthetic values to the issue of clean water and air. In all of these cases, no exact monetary value can be assigned.

The fact that everyone and everything in the environment is interlinked in a chain demonstrates the difficulty in measuring an externality such as smog. Yet the simple recognition that such externalities exist can work wonders in policies attempting to ensure a more sustainable and healthier future.

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The New York Times

WATCH TRAILER

March 12, 2009

Study links smog exposure to premature death

By ROBIN BRAVENDER, Greenwire

Long-term exposure to concentrated smog significantly raises the risk of dying from lung disease, a new study shows.

The **study (pdf)**, published today in the *New England Journal of Medicine*, found that the risk of dying from respiratory disease is more than three times higher in metropolitan areas with the most concentrated ozone -- a precursor of smog -- than in those with the lowest ozone concentrations.

The report is the first nationwide study to evaluate the effects of long-term impacts of ozone on human health and the first to separate the effects of ozone pollution from those of fine particle pollution, or soot, according to a statement from New York University's Langone Medical Center.

"Many studies have shown that a high ozone day leads to an increase in risk of acute health effects the next day, for example, asthma attacks and heart attacks," said co-author George Thurston, a professor at NYU's Department of Environmental Medicine, in the statement.

"What this study says is that to protect the public's health, we can't just reduce the peaks, we must also reduce long-term, cumulative exposure."

The study was co-authored by scientists from Health Canada, Brigham Young University, New York University's School of Medicine, the University of Ottawa, the American Cancer Society and the University of California, Berkeley.

Ozone is formed by a chemical reaction between nitrogen oxides and volatile organic compounds in the presence of sunlight. It is considered beneficial in the earth's stratosphere, where it forms a shield that blocks the sun's harmful rays. But ground-level ozone -- which can come from tailpipes, coal-fired utilities and other industries -- can trigger health problems including chest pain, coughing, throat irritation and congestion, according to U.S. EPA. It can also damage vegetation and ecosystems.

'Substantial risk' under EPA limits

Thurston said the study shows that EPA's current standards for airborne ozone -- measured over eight-hour periods -- do not protect against the long-term effects of ozone exposure.

"It seems clear that even in cities that are approaching meeting the existing standard, you still have a substantial risk from the cumulative long-term exposure that's not addressed by the acute standard," he said.

New York City's air, for example, is nearly in compliance with EPA's short-term ozone standard of 75 parts

per billion, he said. Still, New Yorkers face a 25 percent increased risk of respiratory death as a result of their ozone exposures, he said.

Yesterday, the Obama administration asked a federal appeals court to stall a pending court case over EPA's current smog standards to give the agency more time to consider whether to revise the controversial Bush-era air quality standards (*E&ENews PM*, March 11). Environmental groups have blasted the Bush-era standard for being too weak, while industry groups have argued that the current standard is too stringent.

Frank O'Donnell, president of the advocacy group Clean Air Watch, said the study adds fuel to clean air advocates' argument that the federal standards should be stricter.

"There's certainly a great weight of evidence to document that tougher ozone standards are needed," O'Donnell said.

Click here (pdf) to read the report.

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http://www.epa.gov/airtrends/aqtrnd95/pm10.html
Last updated on Thursday, January 05, 2012

AIRTrends 1995 Summary

You are here: EPA Home Air & Radiation Air Trends Reports 1995 Summary Particulate Matter

Particulate Matter (PM-10)

Note: EPA no longer updates this information, but it may be useful as a reference or resource.

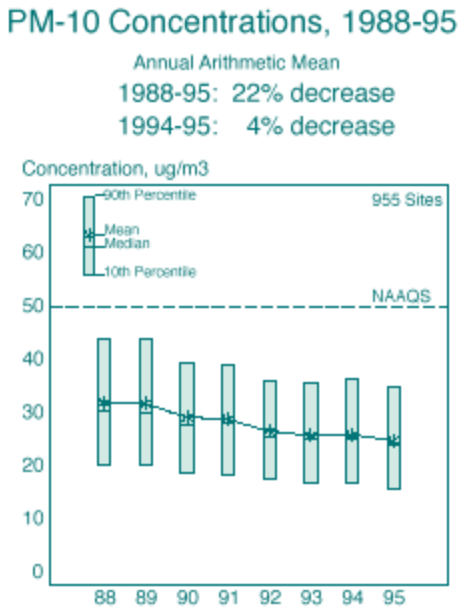
Please see www.epa.gov/airtrends for the latest information on Air Quality Trends.

Nature and Sources of the Pollutant: Particulate matter is the term for solid or liquid particles found in the air. Some particles are large or dark enough to be seen as soot or smoke. Others are so small they can be detected only with an electron microscope. Because particles originate from a variety of mobile and stationary sources (diesel trucks, woodstoves, power plants, etc.), their chemical and physical compositions vary widely. Particulate matter can be directly emitted or can be formed in the atmosphere when gaseous pollutants such as SO_2 and NOx react to form fine particles.

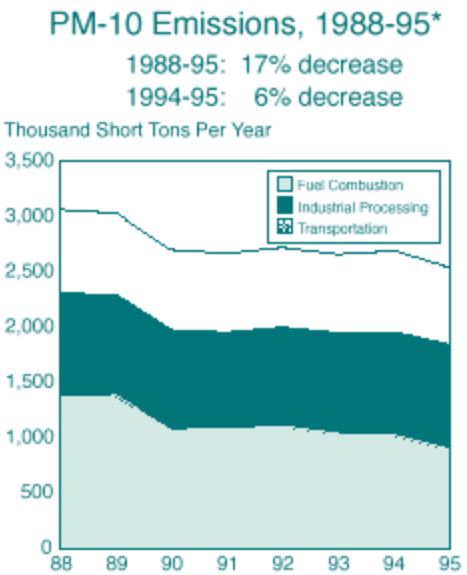
Health and Environmental Effects: In 1987, EPA replaced the earlier Total Suspended Particulate (TSP) air quality standard with a PM-10 standard. The new standard focuses on smaller particles that are likely responsible for adverse health effects because of their ability to reach the lower regions of the respiratory tract. The PM-10 standard includes particles with a diameter of 10 micrometers or less (0.0004 inches or one-seventh the width of a human hair). EPA's health-based national air quality standard for PM-10 is 50 $\mu g/m^3$ (measured as an annual mean) and 150 $\mu g/m^3$ (measured as a daily concentration). Major concerns for human health from exposure to PM-10 include: effects on breathing and respiratory systems, damage to lung tissue, cancer, and premature death. The elderly, children, and people with chronic lung disease, influenza, or asthma, are especially sensitive to the effects of particulate matter. Acidic PM-10 can also damage human-made materials and is a major cause of reduced visibility in many parts of the U.S. New scientific studies suggest that fine particles (smaller than 2.5 micrometers in diameter) may cause serious adverse health effects. As a result, EPA is considering setting a new standard for PM-2.5. In addition, EPA is reviewing whether revisions to the current PM-10 standards are warranted.

Trends in PM-10 Levels: Air monitoring networks were changed in 1987 to measure PM-10 (replacing the earlier TSP monitors).

Between 1988 and 1995, average PM-10 concentrations decreased 22 percent. Short-term trends between 1994 and 1995 showed a decrease of 4 percent in monitored PM-10 concentration levels.



Emissions of PM-10 shown in the chart are based on estimates from fuel combustion sources, industrial processes, and transportation sources, which account for only 6 percent of the total PM-10 emissions nationwide. Between 1988 and 1995, PM-10 emissions for these sources decreased 17 percent. Short-term emissions trends between 1994 and 1995 showed a 6 percent decrease.



*Omits natural and miscellaneous sources.

The emissions estimates presented below do not include emissions from natural and miscellaneous sources which are fugitive dust (unpaved and paved roads), agricultural and forestry activities, wind erosion, wildfires and managed burning. These emissions estimates also do not account for particulate matter that is secondarily formed in the atmosphere from gaseous pollutants (e.g., SO_2 and NOx).

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Air Pollution - Particulate Matter Brochure

This page last reviewed May 6, 2009

What is Particulate Matter (PM10)?

Particulate matter (PM10) pollution consists of very small liquid and solid particles floating in the air. Of greatest concern to public health are the particles small enough to be inhaled into the deepest parts of the lung. These particles are less than 10 microns in diameter - about 1/7th the thickness of the a human hair - and are known as PM10. This includes fine particulate matter known as PM2.5.

PM10 is a major component of air pollution that threatens both our health and our environment.

Where does PM10 come from?

In the western United States, there are sources of PM10 in both urban and rural are as, major sources include:

1. Motor vehicles.
2. Wood burning stoves and fireplaces.
3. Dust from construction, landfills, and agriculture.
4. Wildfires and brush/waste burning.
5. Industrial sources.
6. Windblown dust from open lands.

PM10 is a mixture of materials that can include smoke, soot, dust, salt, acids, and metals. Particulate matter also forms when gases emitted from motor vehicles and industry undergo chemical reactions in the atmosphere.

How does PM10 affect our health?

PM10 is among the most harmful of all air pollutants. When inhaled these particles evade the respiratory system's natural defenses and lodge deep in the lungs.

Health problems begin as the body reacts to these foreign particles. PM10 can increase the number and severity of asthma attacks, cause or aggravate bronchitis and other lung diseases, and reduce the body's ability to fight infections.

Although particulate matter can cause health problems for everyone, certain people are especially vulnerable to PM10's adverse health effects. These "sensitive populations" include children, the elderly, exercising adults, and those suffering from asthma or bronchitis.

Of greatest concern are recent studies that link PM10 exposure to the premature death of people who already have heart and lung disease, especially the elderly.

Does PM10 affect our view?

PM10 is often responsible for much of the haze that we think of as smog. This is a problem in our cities, rural areas and pristine areas - such as national parks and forests.

What is being done to reduce PM10 pollution?

The United States Environmental Protection Agency has set air quality standards for PM10. Based on health research, these identify acceptable levels of PM10. Currently, these standards are violated in many parts of the western United States.

Air quality agencies in several states have developed, or are now developing, air quality plans to bring PM10 concentrations down to healthful levels. These plans include a variety of programs to reduce emissions, including:

1. Dust control for roads, construction, and landfills.
2. Landscaping, barrier, and fencing to reduce windblown dust.
3. Programs to reduce emission from wood stoves and fireplaces.
4. Cleaner - burning gasoline and diesel fuels.
5. Emission control devices for motor vehicles.
6. Controls for industrial facilities.

What can you do?

Here are a few things individuals, business, and other organizations can do immediately to reduce the threat of PM10:

1. Reduce travel on days with poor air quality.
2. Avoid vigorous physical activity on days that have poor air quality.
3. Avoid using your wood stove and fireplace on days that have poor air quality.
4. Avoid using leaf blowers and other dust - producing equipment.
5. Drive slowly on unpaved roads and other dirt surfaces.
6. Get involved with air quality improvement programs in your community.
7. If you own or operate an industrial source of PM10, comply with local rules that apply to your operation. Work with local agencies to develop strategies that will further reduce PM10 emissions.

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2009 IMPERIAL COUNTY STATE IMPLEMENTATION PLAN FOR PARTICULATE MATTER LESS THAN 10 MICRONS IN AERODYNAMIC DIAMETER

FINAL

Prepared for

Imperial County Air Pollution Control District
150 South 9th Street
El Centro, CA. 92243-2801

Prepared by

ENVIRON International Corporation
707 Wilshire Blvd., Suite 4950
Los Angeles, CA 90017

August 11, 2009

PM_{10} is respirable, with fine and ultrafine particles reaching the alveoli deep in the lungs, and larger particles depositing principally in the nose and throat area. PM_{10} deposition in the lungs results in irritation that triggers a range of inflammation responses, such as mucus secretion and bronchoconstriction, and exacerbates pulmonary dysfunctions, such as asthma, emphysema, and chronic bronchitis. Sufficiently small particles may penetrate into the bloodstream and impact functions such as blood coagulation, cardiac autonomic control, and mobilization of inflammatory cells from the bone marrow. Individuals susceptible to higher health risks from exposure to PM_{10} airborne pollution include children, the elderly, smokers, and people of all ages with low pulmonary/cardiovascular function. For these individuals in particular, adverse health effects of PM_{10} pollution include coughing, wheezing, shortness of breath, phlegm, bronchitis, and aggravation of lung or heart disease, leading for example to increased risks of hospitalization and mortality from asthma attacks and heart attacks.[1]

1.2 Imperial County

1.2.1 Geography, Population, and Land Use

Imperial County extends over 4,597 square miles[2] in the southeastern portion of California, bordering Mexico to the south, Riverside County to the north, San Diego County to the west, and the State of Arizona to the east. The Imperial Valley runs approximately north-to-south through the center of the county and extends into Mexico. The terrain elevation varies from as low as 230 feet below sea level at the Salton Sea to the north to more than 2,800 feet above sea level at the mountain summits to the east.

Imperial County's population is about 173,000 people,[3] and its principal industries are farming and retail trade. Most of the population, farming, and retail trade exist in a band of land that, on average, comprises less than one-fourth the width of the county, stretching from the south shore of the Salton Sea to the Mexican border. The road network is densest within this strip, as shown in Figure 1.1. The rest of Imperial County is the Salton Sea and mostly dry, barren desert area with little or no human population. Imperial County's population distribution and population growth in recent years are reported in Appendix V.

Imperial County's agricultural industry[4] grew to $1.37 billion in 2007, led by cattle farming at $334 million. More than 40 types of crops and commodities are grown in the county, ranking Imperial County 11th among California counties.[5] The total acreage of famed land has remained fairly constant at ~500,000 acres over the last decade, and nearly 25% of the county's labor force works in the Agricultural Sector during the high season.

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1 Additional details regarding the adverse health effects of PM can be found in the San Joaquin Valley 2006 PM_{10} Plan (Chapter 1, Section 1.5), available at http://www.valleyair.org/Air_Quality_Plans/06PM10.htm.

2 Official website of Imperial County, http://www.co.imperial.ca.us/.

3 Southern California Association of Governments, http://www.scag.ca.gov/publications/pdf/2007/SOTR07/SOTR07_Population.pdf

4 Imperial County Agricultural Commissioners Office, *Imperial County 2007 Agricultural Crop and Livestock Report*; available at http://imperialcounty.net/ag/Crop%20&%20Livestock%20Reports/Crop%20&%20Livestock%20Report%202007%20Color.pdf

5 California Farm Bureau Federation, http://www.cfbf.com/counties/index.cfm?id=13

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August 31, 2012

Lozeau | Drury LLP
410 12th Street, Suite 250
Oakland, CA 94607

Attn: Mr. Michael Lozeau

Subject: Comment Letter on the Draft Environmental Impact Report for the Prologis Eucalyptus Industrial Park, SCH No. 2008021002.

Dear Mr. Lozeau:

At the request of Lozeau | Drury LLP **(**Lozeau Drury), Clark and Associates (Clark) has reviewed materials related to the above referenced project, including the Draft Environmental Impact Report[1] (DEIR) for the Prologis Eucalyptus Industrial Park (hereafter called the Project), SCH No. 2008021002 and its appendices. The proposed project site is located in the eastern portion of the City of Moreno Valley, in Riverside County, California. The 122.8-acre project site is located south of State Route 60 (SR-60) east of the Moreno Valley Auto Mall, and adjacent to and west of the Quincy Channel. According to the DEIR the proposed project would result in the construction and operation of a warehouse facility, consisting of approximately 2,244,638 square feet (sq ft).

Currently the site is used undeveloped for commercial uses and has two citrus groves in the northeastern and northwestern portions of the site, while the central and southern portions are vacant and support mainly weedy vegetation. According to a March, 2012 Memo from LSA

[1] LSA. 2012. Draft Environmental Impact Report, State Clearinghouse No. 2008021002, Prologis Eucalyptus Industrial Park (formerly Prologis Park Moreno Valley Eucalyptus Project), City of Moreno Valley, Riverside County, California. LSA Associates, Inc. 1500 Iowa Ave, Suite 200, Riverside, CA LAS Project No. PLO1101. Prepared July 2012 pg. 1-2

Associates[2], the project site contains 57-acres of citrus (Grapefruit) trees with the rest of the site vacant. The surrounding area has been dry-farmed in the past, and the eastern end of the City has historically supported a variety of crops, including citrus, melon, potatoes, etc[3]. There are three small natural drainage features on site, two ephemeral channels in the southwestern portion of the site and the larger Quincy Channel along the eastern edge of the property. According to the DEIR[4], there is some minor amount of refuse is present in the southwest and southeast corners of the site from unauthorized dumping.

Land adjacent to the project site includes vacant land east and south of the proposed project site, SR- 60 to the north, and the Moreno Valley Auto Mall and the City of Moreno Valley Fire Station No. 58 northwest of the project site. Existing single-family residential uses are located approximately 50 feet southeast of the southeastern corner of the project site[5].

The proposed project has had and will have significant impacts on the community prior to the approval of the DEIR. The proposed project will require significant changes in the local zoning ordinances (General Plan for the City of Moreno Valley) including:

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[2] LSA. 2012. Project Memorandum: Agricultural Use of ProLogis Industrial Property. Memorandum from Kent Norton, LSA Riverside Office to Jeff Bradshaw, City of Moreno Valley. Dated March 21, 2012.

[3] LSA. 2012. Project Memorandum: Agricultural Use of ProLogis Industrial Property. Memorandum from Kent Norton, LSA Riverside Office to Jeff Bradshaw, City of Moreno Valley. Dated March 21, 2012.

[4] LSA. 2012. Draft Environmental Impact Report, State Clearinghouse No. 2008021002, Prologis Eucalyptus Industrial Park (formerly Prologis Park Moreno Valley Eucalyptus Project), City of Moreno Valley, Riverside County, California. LSA Associates, Inc. 1500 Iowa Ave, Suite 200, Riverside, CA LAS Project No. PLO1101. Prepared July 2012 pg 3-1

[5] LSA. 2012. Draft Environmental Impact Report, State Clearinghouse No. 2008021002, Prologis Eucalyptus Industrial Park (formerly Prologis Park Moreno Valley Eucalyptus Project), City of Moreno Valley, Riverside County, California. LSA Associates, Inc. 1500 Iowa Ave, Suite 200, Riverside, CA LAS Project No. PLO1101. Prepared July 2012 pg 3-1

- Approval of a General Plan Amendment to change the land use designation of 71.3 acres of the project site from Residential (R15, R5, and R2) to Business Park (BP) so the entire site would then be designated Business Park (BP).
- Approval of a Zone Change of the entire 122.8 acres from its current zoning designations of Business Park (BP), Business Park/Mixed Use (BPX), Residential 15 District (R15), Residential 5 District (R5), and Residential Agriculture 2 (RA-2) to all Light Industrial (LI).
- Zone Change will also be used to redraw the boundary of the Primary Animal Keeping Overlay (PAKO) district.
- Approval of an amendment to the City's Master Plan of Trails to relocate the Eucalyptus Avenue Trail to the north side of future Eucalyptus Avenue and eliminate the planned trail segment on Quincy Avenue from SR-60 to Fir Avenue (future Eucalyptus Avenue), based on discussion with the City Trails Commission.
- Approval of an amendment to the Circulation Element of the General Plan. These changes include the following:
    - Eliminate the undeveloped Quincy Street from Eucalyptus Avenue south to Encilia Avenue;
    - Realign Encilia Avenue from its current alignment such that its westerly terminus is located at Moreno Beach Drive instead of the current General Plan westerly terminus at Eucalyptus Avenue; and
    - The segment between Quincy Channel and Moreno Beach Drive would be classified as a Collector.

In addition, to the changes above, the proposal of the project has forced the Moreno Valley Unified School District (MVUSD) to abandon plans to locate an elementary school (MVUSD Elementary School #24), a middle school (MVUSD Middle School #7), and a high school (MVUSD

High School #5) in the vicinity of Redlands Boulevard and future Eucalyptus Avenue, in close proximity to the proposed. After the Notice of Preparation (NOP) for the proposed project was released, MVUSD decided to abandon plans for these school sites and relocate the future school facilities in a different area of the City[6]. Students who live in the area to be serviced by the proposed schools will now have to travel farther to attend schools.

The DEIR for the Project, determined that the proposed project's construction and operational phases would have impacts on air quality that would be less than significant with mitigation incorporated. These conclusions are premature and based upon a flawed analysis of the potential emissions at the site. The proponents should re-evaluate the impacts of the project and present them in a revised draft environmental impact report (RDEIR).

Clark's review of the materials in no way constitutes a validation of the conclusions or materials contained within the plan. If we do not comment on a specific item this does not constitute acceptance of the item.

This DEIR was issued prematurely without considering the serious flaws in the Proponent's analysis of the project. The flaws include:

1. The proponent's use of the CalEEMod ensures an underestimation of the potential particulate emission for the construction phase of the proposed project.
2. Failure of the proponent to compare construction emissions to daily construction significance thresholds;
3. Failure to consider health risks from contaminated dust; and
4. Failure to properly identify and address the Project's operational air quality impacts.

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[6] LSA. 2012. Draft Environmental Impact Report, State Clearinghouse No. 2008021002, Prologis Eucalyptus Industrial Park (formerly Prologis Park Moreno Valley Eucalyptus Project), City of Moreno Valley, Riverside County, California. LSA Associates, Inc. 1500 Iowa Ave, Suite 200, Riverside, CA LAS Project No. PLO1101. Prepared July 2012 pg 4.3-6.

COMMENTS

1. The Proponent's Use Of The CalEEMod Ensures An Underestimation Of The Potential Particulate Emission For The Construction Phase Of The Proposed Project.

The California Air Resource Board's (CARB's) Urban Emission (URBEMIS) model and the California Emissions Estimator Model (CalEEMod) are computer models designed to estimate emissions of criteria pollutants during construction and operational phases of projects. Currently, South Coast Air Quality Management District (SCAQMD) accepts the outputs from both models in their air quality analyses. Significant differences in the models must be highlighted in the DEIR. The changes in the method used to estimate construction impacts from the proposed project by using the CalEEMod model instead of the URBEMIS model include:

- Failure to account for wind-blown fugitive dust[7]. According to the July, 2011 CalEEMod Technical Paper, wind-blown fugitive dust is not calculated in CalEEMod. For sites as large as the proposed project site, this can result in significant quantities of particulate matter being released.
- SCAQMD's surveys of construction sites were limited to sites of 35 acres or less. For projects larger than 35-acres the data was extrapolated by increasing the number of construction days but not increasing the number of construction equipment pieces used on a given day. The impact is to reduce the construction PM estimates for the site as compared to URBEMIS[8].

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[7] CalEEMod. Technical Paper: Methodology Reasoning and Policy Development of the California Emission Estimator Model. July, 2011. Pg 4.

[8] CalEEMod. Technical Paper: Methodology Reasoning and Policy Development of the California Emission Estimator Model. July, 2011. Pg 5.

- Grading in URBEMIS is based upon 25% of total project acreage in one day. Grading in CalEEMod is based upon Walker's Building Estimator's Reference Book. The impact of this change is to decrease PM emissions from grading in the CalEEMod[9] by tying the emissions to the number of pieces of equipment present at the site.

The proponent's must include an analysis of these impacts in a revised DEIR (RDEIR) to ensure that an accurate analysis of the potential impacts from the proposed project are presented as required by CEQA.

2. Failure To Accurately Compare Construction Emissions To Daily Construction Significance Thresholds.

Unlike the operational emissions from most projects, which are typically more or less continuous, emissions from construction sites are highly variable depending on the type of construction that is being performed. For example, grading results in large quantities of fugitive dust and combustion emissions from diesel-powered equipment. Short-term emissions during the various construction phases can be considerable and may result in degradation of local and regional air quality and severe health effects.

To determine whether short-term emissions may result in degradation of local and regional air quality and severe health effects, it is common practice for lead agencies to compare project emissions to quantitative significance thresholds developed by local air districts as a screening tool for CEQA review. Thresholds of significance for construction emissions are typically expressed on a short-term basis, *i.e.* daily or hourly basis to adequately capture impacts due to the high variability of emissions during different construction stages.

[9] CalEEMod. Technical Paper: Methodology Reasoning and Policy Development of the California Emission Estimator Model. July, 2011. Pg 5.

Table 1 presents a summary of short-term emissions thresholds developed by SCAQMD and other air districts for assessing impacts on air quality from construction projects.

Table 1:
CEQA significance thresholds for construction emissions from various air districts

Air district construction thresholds*	**NOx** (lbs/day)	**ROG** (lbs/day)	**PM_{10}** (lbs/day)	**DPM** (lbs/day)	**$PM_{2.5}$** (lbs/day)	**CO** (lbs/day)
SCAQMD	100	75	150		55	550
BAAQMD	54	54	82		54	
EDCAPCD	82	82				
SLOCAPCD				7		
MBUAPCD			82			550
FRAQMD	25	25	80			
SMAQMD	85					
YSAQMD	82	82	150			

SCAQMD = South Coast Air Quality Management District, CEAQ Handbook, 1993;
BAAQMD = Bay Area Air Quality Management District, CEQA Guidelines 2009;
EDCAPCD = El Dorado County Air Pollution Control District, CEQA Guide, February 2002;
SLOCAPCD = San Louis Obispo County Air Pollution Control District, CEQA Air Quality Handbook, December 2009.
MBUAPCD = Monterey Bay Unified Air Pollution Control District, CEQA Air Quality Guidelines, June 2004,
FRAQMD = Feather River Air Quality Management District, http://www.fraqmd.org/CEQA_Thresholds.htm;
SMAQMD Sacramento Metropolitan Air Quality Management District, Guide to Air Quality Assessment, July 2004;
YSAQMD, Yolo-Solano Air Quality Management District, Air Quality Handbook, Guidelines for Determining Air Quality Thresholds of Significance and Mitigation Measures for Proposed Development Projects that Generate Emissions from Motor Vehicles, revised 2002

According to the DEIR[10], "criteria pollutant emissions during project construction would exceed the SCAQMD emission thresholds for oxides of nitrogen (NO_X) and reactive organic gases (ROG). Compliance with SCAQMD Rules and Regulations during construction will minimize construction-related air quality impacts from fugitive dust emissions and construction equipment emissions. Mitigation is required. The proposed

[10] LSA. 2012. Draft Environmental Impact Report, State Clearinghouse No. 2008021002, Prologis Eucalyptus Industrial Park (formerly Prologis Park Moreno Valley Eucalyptus Project), City of Moreno Valley, Riverside County, California. LSA Associates, Inc. 1500 Iowa Ave, Suite 200, Riverside, CA LAS Project No. PLO1101. Prepared July 2012 pg 1.

project would not exceed any of the localized significance thresholds (LSTs) during construction periods." This statement is incorrect and misleading.

A review of the CalEEMod analysis for the project shows that the mitigated construction emissions of ROG and particulate matter less than 2.5 microns ($PM_{2.5}$) exhaust (a surrogate for diesel particulate emissions) are in excess of the CEQA significance thresholds listed above. During Year 2013, ROG and $PM_{2.5}$ exhaust emissions are estimated to be 368.03 lbs/day and 7.95 lbs/day, respectively.

In addition to the Significant Thresholds above, SCAQMD recommends the use of LSTs to determine potential impacts to receptors near projects. According to the Air Quality Analysis prepared by the proponent, Table I of the Air Quality Analysis (Table 2 below) shows that the emissions of the pollutants on the peak day of construction are below the SCAQMD LST. In this table the proponent uses the emission estimates from the grading phase of the construction. The proponent inaccurate asserts that the emission levels will be below the LST values.

Table 2:

Construction LST Impacts from Air Quality Analysis

Emission Sources	Pollutant Emissions, lbs/day			
	NOx	CO	PM_{10}	$PM_{2.5}$
On-Site (grading) Emissions	104	55	8.4	6.3
LST Threshold	270	1,577	13	8
Significant Emissions?	No	No	No	No

A review of the CalEEMod analysis shows that the highest emission values are not associated with the grading phase. In Section 2.0 Emission Summary of the CalEEMod analysis presented in the Air Quality Analysis the construction impacts are listed as:

Table 3:

Construction LST Impacts from CalEEMod Output

Emission Sources	Pollutant Emissions, lbs/day			
	NOx	CO	PM_{10}	$PM_{2.5}$
Mitigated Construction Emissions	139.84	166.77	29.2	8.28
LST Threshold	270	1,577	13	8
Significant Emissions?	No	No	**Yes**	**Yes**

The Proponent's analysis of air quality impacts clearly fails to accurately describe the impacts of the emissions on the receptors closest to the project site (homes within 50 feet of the site boundary and the fire station immediately adjacent to the site boundary). Emissions of $PM_{2.5}$ (surrogate for diesel exhaust) and PM_{10} from the construction site may have lasting impacts on the receptors nearby.

Diesel exhaust contains nearly 40 toxic substances including toxic air contaminants (TACs) and may pose a serious public health risk for residents in the vicinity of the facility. TACs are airborne substances that are capable of causing short-term (acute) and/or long-term (chronic or carcinogenic, i.e., cancer causing) adverse human health effects (i.e., injury or illness). TACs include both organic and inorganic chemical substances. The current California list of TACs[11] includes approximately 200 compounds, including particulate emissions from diesel-fueled engines.

DPM and TAC emissions may affect numerous sensitive receptors in the region including onsite construction workers, fire personnel at the station adjacent to the site and the single-family residences located near the site. Evidence exists that clouds of soot emitted by heavy-duty

[11] URS. 2012. Impacts to Air Quality from the Construction and Operation of the Brannon Solar, LLC Solar Energy Generation Project. Dated Febraury 7, 2012. Table A-7

construction equipment can travel downwind for miles, then drift into heavily populated areas. For example, health impact studies from the SCAQMD[12] have documented that diesel emissions travel miles from the sources impacting residents.

Diesel exhaust has been linked to a range of serious health problems including an increase in respiratory disease, lung damage, cancer, and premature death[13,14,15]. Fine diesel particles are deposited deep in the lungs in the smallest airways and can result in increased respiratory symptoms and disease; decreased lung function, particularly in children and individuals with asthma; alterations in lung tissue and respiratory tract defense mechanisms; and premature death.[16] Exposure to diesel exhaust increases the risk of lung cancer. It also causes non-cancer effects including chronic bronchitis, inflammation of lung tissue, thickening of the alveolar walls, immunological allergic reactions, and airway constriction.[17]

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[12] SCAQMD MATES I, II, and III have documented the impacts for DPM in the SCAB.

[13] California Air Resources Board, Initial Statement of Reasons for Rulemaking, Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant, Staff Report, June 1998.

[14] U.S. EPA, Health Assessment Document for Diesel Engine Exhaust, Report EPA/600/8-90/057F, May 2002.

[15] Environmental Defense Fund, Cleaner Diesel Handbook, Bring Cleaner Fuel and Diesel Retrofits into Your Neighborhood, April 2005; http://www.edf.org/documents/4941_cleanerdieselhandbook.pdf, accessed March 27, 2008.

[16] California Air Resources Board , Initial Statement of Reasons for Rulemaking, Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant, Staff Report, June 1998.

[17] Findings of the Scientific Review Panel on The Report on Diesel Exhaust as adopted at the Panel's April 22, 1998 Meeting.

A recent analysis found that air pollution from diesel construction equipment is already taking a heavy toll on the health and economic well-being of Californians[18,19].

PM_{10} emissions from the construction phase of the proposed project may be extremely troublesome for receptors near the site (i.e., homes near the site boundary and the fire station next to the site) since they will act as carriers for residual pesticides/herbicides from the site (see comment below). The project site currently contains 57-acres of citrus (Grapefruit) trees and the surrounding area has been dry-farmed in the past, and the eastern end of the City of Moreno Valley historically supported a variety of other crops[20]. Given the proximity of receptors to the site and the estimated emission rates of particulate matter from the site after mitigation, it is clear that construction activities at the project site will adversely impact the previously identified receptors.

Based on my expert opinion, applicable significance thresholds, and the CalEEMod analysis performed by the proponent, I conclude that the Project will have significant adverse impacts from construction air emissions of fugitive dust, ROG, and diesel emissions. The lead agency must re-evaluate the construction emissions and incorporate a phased approach to estimate the true impacts of construction activities on air quality, and propose all feasible mitigation measures to reduce these significant emissions, in a RDEIR.

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[18] These estimates are conservative because they do not include emissions from a large number of small construction projects (residential and commercial and projects smaller than 1 acre in size). Further, John Hakel, vice president of the Associated General Contractors, which represents construction equipment fleet owners and general contractors, indicated that the report appeared to underestimate the sheer volume of construction equipment.

[19] Union of Concerned Scientists, Digging up Trouble: Construction Pollution in the Bay Area; http://www.ucsusa.org/assets/documents/clean_vehicles/Bay-Area-Fact-Sheet.pdf, accessed March 27, 2008.

[20] LSA. 2012. Project Memorandum: Agricultural Use of ProLogis Industrial Property. Memorandum from Kent Norton, LSA Riverside Office to Jeff Bradshaw, City of Moreno Valley. Dated March 21, 2012.

3. Failure To Consider Health Risks From Contaminated Dust.

Residual contaminants in soils at the site may be entrained in dust generated during construction activities. The release of residual contamination is a potentially significant impact, given the past use of the site for agricultural production. According to the California Department of Toxic Substances Control August 2002 Interim Guidance for Sampling Agricultural Fields for School Sites (known sensitive receptors), "the most commonly detected pesticides have been DDT and it's derivatives DDD and DDE, toxaphene, dieldrin, and aldrin. Of these pesticides, toxaphene has been the major pesticide driving unacceptable levels of risk requiring remediation by soil removal." Given the volume of soils to be graded at each of the sites it is imperative to understand whether particulate matter generated at the sites will pose a potential health risk to sensitive receptors in the vicinity of each site.

According to DTSC, "the guidance is applicable to agricultural land that is currently under cultivation with row, fiber or food crops, orchards, or pasture. It is also applicable to fallow and former agricultural land that is no longer in production and has not been disturbed beyond normal disking and plowing practices. Each field of the same crop is assumed to have been watered, fertilized, and treated with agricultural chemicals to the same degree across the field. Because of this homogeneous application, contaminant levels are expected to be similar at any given location within the field."

There is no indication of a sampling and analysis plan in the DEIR, or the Project documents provided by the lead agency, which is a serious deficiency. Prior to issuing a DEIR for the project, the Proponent should be required to complete a sampling and analysis plan to confirm or rule out the possibility of the presence of residual contaminants at the site. Identifying residual pesticides or other contaminants in soils at the site

prior to construction activities will provide an opportunity for the Proponent to remove/mitigate the potential exposure of sensitive receptors within the vicinity of the sites. In the absence of any sampling or analysis, and given the past use of the Project site, I conclude that there is at least a fair argument that the Project may have significant impacts related to residual contaminants at the site.

4. Failure To Properly Identify and Address the Project's Operational Air Quality Impacts.

The DEIR asserts with no analysis whatsoever that the project's emissions of criteria pollutants will not result in a considerable net increase of any criteria pollutant for which the project region is in no-attainment under an applicable federal or state ambient air quality standard.

During the operational phase of the project the project will have the potential to generate significant quantities of criteria pollutants (NO_x, SO_x, Ozone precursors, PM). According to Table 3-1 of the most recent BAAQMD CEQA guidance, a construction of a 259,000 square foot light industrial or warehouse operation will typically violate NOx construction thresholds and GHG operational thresholds. The proposed Project's 2,000,000 square feet plus of warehouse and manufacturing buildings are nearly 8 times the size of the screening threshold, ensuring a violation of local air quality thresholds. I therefore conclude that the Project will have significant NOx and GHG emissions during Project operations.

The air quality impacts from the traffic associated with a 2,000,000 square foot facility are significant. Typically the impacts are quantified by the number of vehicle trips per day. In the case of the proposed project, the primary concern will be the number of truck trips per day. A truck trip is one round trip (one trip segment to a site and one trip segment away from a site).

According to one source, Bluffstone and Ouderkirk[21], a 500,000 square feet facility on 50 acres, will on average have 350 truck trips per day (or 700 trip segments) associated with its development. This figure is proportionate to estimates for an AMB Property Corporation center in Redlands (1,000 truck trips for a 1.3 million square feet structure); Wal-Mart distribution centers in Pueblo, Colorado (700 truck trips per day for an 880,000 square feet facility), Connecticut, and Delaware (both 1,000 truck trips per day for 1.2 million square feet structures); and a grocery distribution center in New York (Boas, 2002; Gasiewski, 2004; Hernandez, 2005; Pueblo Chieftain, 2004; Sholl, 2004).

Estimates from other sources indicate approximately 1 truck per 1,000 square feet of the building, which means that the proposed project would require 1,000 trucks per day (or 1,000 trip segments per day) for the warehouse segment of the Project. The number of truck trips could be higher at a new, more efficient facility where more inventory is moved per day. Without proper modeling of the emissions from these additional vehicles the impacts on the environment and the citizens of Moreno Valley is unknown. It is clear that the size of the Project will have significant NOx and GHG emissions during Project operations.

A proper operational impact analysis is vital for an environmental analysis 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 the environmental damage often occurs incrementally from a variety of small sources with which they interact. The increase in PM in the region, even for short periods of time, will only exacerbate the already serious air quality issues in the region.

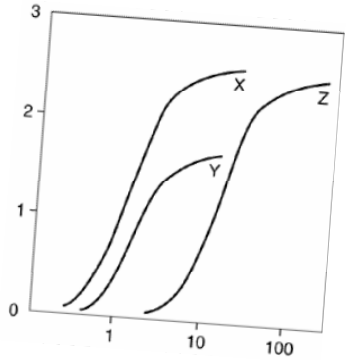
[21]Bluffstone and Ouderkirk. 2007. Warehouses, trucks, and [PM.sub.2.5]: human health and logistics industry growth in the eastern Inland Empire. Contemporary Economic Policy 25(1):

Conclusion

The facts identified and referenced in this comment letter lead me to reasonably conclude that the Project will result in significant adverse impacts that were not identified in the DEIR and that are not adequately mitigated. Many of the DEIR's conclusions that environmental impacts are not significant or less than significant with mitigation are unsupported or contradicted by the evidence. As a result, several analyses presented in the DEIR, including impacts on air quality fail to identify or disclose the magnitude of significant adverse impacts. To protect air quality and public health the Proponent must prepare a RDEIR for the Project.

Sincerely,

James Clark, Ph.D.



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James J. J. Clark, Ph.D.

Principal Toxicologist

Toxicology/Exposure Assessment Modeling

Risk Assessment/Analysis/Dispersion Modeling

Education:

Ph.D., Environmental Health Science, University of California, 1995

M.S., Environmental Health Science, University of California, 1993

B.S., Biophysical and Biochemical Sciences, University of Houston, 1987

Professional Experience:

Dr. Clark is a well recognized toxicologist, air modeler, and health scientist. He has 20 years of experience in researching the effects of environmental contaminants on human health including environmental fate and transport modeling (SCREEN3, AEROMOD, ISCST3, Johnson-Ettinger Vapor Intrusion Modeling); exposure assessment modeling (partitioning of contaminants in the environment as well as PBPK modeling); conducting and managing human health risk assessments for regulatory compliance and risk-based clean-up levels; and toxicological and medical literature research.

Significant projects performed by Dr. Clark include the following:

LITIGATION SUPPORT

Case: James Harold Caygle, et al, v. Drummond Company, Inc. Circuit Court for the Tenth Judicial Circuit, Jefferson County, Alabama. Civil Action. CV-2009

Client: Environmental Litgation Group, Birmingham, Alabama

Dr. Clark performed an air quality assessment of emissions from a coke factory located in Tarrant, Alabama. The assessment reviewed include a comprehensive review of air quality standards, measured concentrations of pollutants from factory, an inspection of the facility and detailed assessment of the impacts on the community. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Settlement in favor of plaintiff.

Case: Rose Roper V. Nissan North America, et al. Superior Court of the State Of California for the County Of Los Angeles – Central Civil West. Civil Action. NC041739

Client: Rose, Klein, Marias, LLP, Long Beach, California

Dr. Clark performed a toxicological assessment of an individual occupationally exposed to multiple chemicals, including benzene, who later developed a respiratory distress. A review of the individual's medical and occupational history was performed to prepare an exposure assessment. The exposure assessment was evaluated against the known outcomes in published literature to exposure to respiratory irritants. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Settlement in favor of plaintiff.

Case: O'Neil V. Sherwin Williams, et al. United States District Court Central District of California

Client: Rose, Klein, Marias, LLP, Long Beach, California

Dr. Clark performed a toxicological assessment of an individual occupationally exposed to petroleum distillates who later developed a bladder cancer. A review of the individual's medical and occupational history was performed to prepare a quantitative exposure assessment. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Summary judgment for defendants.

Case: Moore V., Shell Oil Company, et al. Superior Court of the State Of California for the County Of Los Angeles

Client: Rose, Klein, Marias, LLP, Long Beach, California

Dr. Clark performed a toxicological assessment of an individual occupationally exposed to chemicals while benzene who later developed a leukogenic disease. A review of the individual's medical and occupational history was performed to prepare a quantitative exposure assessment. The exposure assessment was evaluated against the known outcomes in published literature to exposure to refined petroleum hydrocarbons. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Settlement in favor of plaintiff.

Case: Raymond Saltonstall V. Fuller O'Brien, KILZ, and Zinsser, et al. United States District Court Central District of California

Client: Rose, Klein, Marias, LLP, Long Beach, California

Dr. Clark performed a toxicological assessment of an individual occupationally exposed to benzene who later developed a leukogenic disease. A review of the individual's medical and occupational history was performed to prepare a quantitative exposure assessment. The exposure assessment was evaluated against the known outcomes in published literature to exposure to refined petroleum hydrocarbons. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Settlement in favor of plaintiff.

Case: Richard Boyer and Elizabeth Boyer, husband and wife, V. DESCO Corporation, et al. Circuit Court of Brooke County, West Virginia. Civil Action Number 04-C-7G.

Client: Frankovitch, Anetakis, Colantonio & Simon, Morgantown, West Virginia.

Dr. Clark performed a toxicological assessment of a family exposed to chlorinated solvents released from the defendant's facility into local drinking water supplies. A review of the individual's medical and occupational history was performed to prepare a qualitative exposure assessment. The exposure assessment was evaluated against the known outcomes in published literature to exposure to chlorinated solvents. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Settlement in favor of plaintiff.

Case: JoAnne R. Cook, V. DESCO Corporation, et al. Circuit Court of Brooke County, West Virginia. Civil Action Number 04-C-9R

Client: Frankovitch, Anetakis, Colantonio & Simon, Morgantown, West Virginia.

Dr. Clark performed a toxicological assessment of an individual exposed to chlorinated solvents released from the defendant's facility into local drinking water supplies. A review of the individual's medical and occupational history was performed to prepare a qualitative exposure assessment. The exposure assessment was evaluated against the known outcomes in published literature to exposure to chlorinated solvents. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Settlement in favor of plaintiff.

Case: Patrick Allen And Susan Allen, husband and wife, and Andrew Allen, a minor, V. DESCO Corporation, et al. Circuit Court of Brooke County, West Virginia. Civil Action Number 04-C-W

Client: Frankovitch, Anetakis, Colantonio & Simon, Morgantown, West Virginia.

Dr. Clark performed a toxicological assessment of a family exposed to chlorinated solvents released from the defendant's facility into local drinking water supplies. A review of the individual's medical and occupational history was performed to prepare a qualitative exposure assessment. The exposure assessment was evaluated against the known outcomes in published literature to exposure to chlorinated solvents. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Settlement in favor of plaintiff.

Case: Michael Fahey, Susan Fahey V. Atlantic Richfield Company, et al. United States District Court Central District of California Civil Action Number CV-06 7109 JCL.

Client: Rose, Klein, Marias, LLP, Long Beach, California

Dr. Clark performed a toxicological assessment of an individual occupationally exposed to refined petroleum hydrocarbons who later developed a leukogenic disease. A review of the individual's medical and occupational history was performed to prepare a qualitative exposure assessment. The exposure assessment was evaluated against the known outcomes in published literature to exposure to refined petroleum hydrocarbons. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Settlement in favor of plaintiff.

Case: Constance Acevedo, et al., V. California Spray-Chemical Company, et al., Superior Court of the State Of California, County Of Santa Cruz. Case No. CV 146344

Dr. Clark performed a comprehensive exposure assessment of community members exposed to toxic metals from a former lead arsenate manufacturing facility. The former manufacturing site had undergone a DTSC mandated removal action/remediation for the presence of the toxic metals at the site. Opinions were presented regarding the elevated levels of arsenic and lead (in attic dust and soils) found throughout the community and the potential for harm to the plaintiffs in question.

Case Result: Settlement in favor of defendant.

Case: Michael Nawrocki V. The Coastal Corporation, Kurk Fuel Company, Pautler Oil Service, State of New York Supreme Court, County of Erie, Index Number I2001-11247

Client: Richard G. Berger Attorney At Law, Buffalo, New York

Dr. Clark performed a toxicological assessment of an individual occupationally exposed to refined petroleum hydrocarbons who later developed a leukogenic disease. A review of the individual's medical and occupational history was performed to prepare a qualitative exposure assessment. The exposure assessment was evaluated against the

known outcomes in published literature to exposure to refined petroleum hydrocarbons. The results of the assessment and literature have been provided in a declaration to the court.

Case Result: Judgement in favor of defendant.

SELECTED AIR MODELING RESEARCH/PROJECTS

Client – Confidential

Dr. Clark performed a comprehensive evaluation of criteria pollutants, air toxins, and particulate matter emissions from a carbon black production facility to determine the impacts on the surrounding communities. The results of the dispersion model will be used to estimate acute and chronic exposure concentrations to multiple contaminants and will be incorporated into a comprehensive risk evaluation.

Client – Confidential

Dr. Clark performed a comprehensive evaluation of air toxins and particulate matter emissions from a railroad tie manufacturing facility to determine the impacts on the surrounding communities. The results of the dispersion model have been used to estimate acute and chronic exposure concentrations to multiple contaminants and have been incorporated into a comprehensive risk evaluation.

Client – Los Angeles Alliance for a New Economy (LAANE), Los Angeles, California

Dr. Clark is advising the LAANE on air quality issues related to current flight operations at the Los Angeles International Airport (LAX) operated by the Los Angeles World Airport (LAWA) Authority. He is working with the LAANE and LAX staff to develop a comprehensive strategy for meeting local community concerns over emissions from flight operations and to engage federal agencies on the issue of local impacts of community airports.

Client – City of Santa Monica, Santa Monica, California

Dr. Clark is advising the City of Santa Monica on air quality issues related to current flight operations at the facility. He is working with the City staff to develop a comprehensive strategy for meeting local community concerns over emissions from flight operations and to engage federal agencies on the issue of local impacts of community airports.

Client: Omnitrans, San Bernardino, California

Dr. Clark managed a public health survey of three communities near transit fueling facilities in San Bernardino and Montclair California in compliance with California Senate Bill 1927. The survey included an epidemiological survey of the effected communities, emission surveys of local businesses, dispersion modeling to determine potential emission concentrations within the communities, and a comprehensive risk assessment of each community. The results of the study were presented to the Governor as mandated by Senate Bill 1927.

Client: Confidential, San Francisco, California

Summarized cancer types associated with exposure to metals and smoking. Researched the specific types of cancers associated with exposure to metals and smoking. Provided causation analysis of the association between cancer types and exposure for use by non-public health professionals.

Client: Confidential, Minneapolis, Minnesota

Prepared human health risk assessment of workers exposed to VOCs from neighboring petroleum storage/transport facility. Reviewed the systems in place for distribution of petroleum hydrocarbons to identify chemicals of concern (COCs), prepared comprehensive toxicological summaries of COCs, and quantified potential risks from carcinogens and non-carcinogens to receptors at or adjacent to site. This evaluation was used in the support of litigation.

Client – United Kingdom Environmental Agency

Dr. Clark is part of team that performed comprehensive evaluation of soil vapor intrusion of VOCs from former landfill adjacent residences for the United Kingdom's Environment

Agency. The evaluation included collection of liquid and soil vapor samples at site, modeling of vapor migration using the Johnson Ettinger Vapor Intrusion model, and calculation of site-specific health based vapor thresholds for chlorinated solvents, aromatic hydrocarbons, and semi-volatile organic compounds. The evaluation also included a detailed evaluation of the use, chemical characteristics, fate and transport, and toxicology of chemicals of concern (COC). The results of the evaluation have been used as a briefing tool for public health professionals.

EMERGING/PERSISTENT CONTAMINANT RESEARCH/PROJECTS

Client: Ameren Services, St. Louis, Missouri

Managed the preparation of a comprehensive human health risk assessment of workers and residents at or near an NPL site in Missouri. The former operations at the Property included the servicing and repair of electrical transformers, which resulted in soils and groundwater beneath the Property and adjacent land becoming impacted with PCB and chlorinated solvent compounds. The results were submitted to U.S. EPA for evaluation and will be used in the final ROD.

Client: City of Santa Clarita, Santa Clarita, California

Dr. Clark is managing the oversight of the characterization, remediation and development activities of a former 1,000 acre munitions manufacturing facility for the City of Santa Clarita. The site is impacted with a number of contaminants including perchlorate, unexploded ordinance, and volatile organic compounds (VOCs). The site is currently under a number of regulatory consent orders, including an Immanent and Substantial Endangerment Order. Dr. Clark is assisting the impacted municipality with the development of remediation strategies, interaction with the responsible parties and stakeholders, as well as interfacing with the regulatory agency responsible for oversight of the site cleanup.

Client: Confidential, Los Angeles, California

Prepared comprehensive evaluation of perchlorate in environment. Dr. Clark evaluated the production, use, chemical characteristics, fate and transport, toxicology, and remediation of perchlorate. Perchlorates form the basis of solid rocket fuels and have recently been detected in water supplies in the United States. The results of this research

were presented to the USEPA, National GroundWater, and ultimately published in a recent book entitled *Perchlorate in the Environment.*

Client – Confidential, Los Angeles, California

Dr. Clark is performing a comprehensive review of the potential for pharmaceuticals and their by-products to impact groundwater and surface water supplies. This evaluation will include a review if available data on the history of pharmaceutical production in the United States; the chemical characteristics of various pharmaceuticals; environmental fate and transport; uptake by xenobiotics; the potential effects of pharmaceuticals on water treatment systems; and the potential threat to public health. The results of the evaluation may be used as a briefing tool for non-public health professionals.

PUBLIC HEALTH/TOXICOLOGY

Client: Brayton Purcell, Novato, California

Dr. Clark performed a toxicological assessment of residents exposed to methyl-tertiary butyl ether (MTBE) from leaking underground storage tanks (LUSTs) adjacent to the subject property. The symptomology of residents and guests of the subject property were evaluated against the known outcomes in published literature to exposure to MTBE. The study found that residents had been exposed to MTBE in their drinking water; that concentrations of MTBE detected at the site were above regulatory guidelines; and, that the symptoms and outcomes expressed by residents and guests were consistent with symptoms and outcomes documented in published literature.

Client: Confidential, San Francisco, California

Identified and analyzed fifty years of epidemiological literature on workplace exposures to heavy metals. This research resulted in a summary of the types of cancer and non-cancer diseases associated with occupational exposure to chromium as well as the mortality and morbidity rates.

Client: Confidential, San Francisco, California

Summarized major public health research in United States. Identified major public health research efforts within United States over last twenty years. Results were used as a briefing tool for non-public health professionals.

Client: Confidential, San Francisco, California

Quantified the potential multi-pathway dose received by humans from a pesticide applied indoors. Part of team that developed exposure model and evaluated exposure concentrations in a comprehensive report on the plausible range of doses received by a specific person. This evaluation was used in the support of litigation.

Client: Covanta Energy, Westwood, California

Evaluated health risk from metals in biosolids applied as soil amendment on agricultural lands. The biosolids were created at a forest waste cogeneration facility using 96% whole tree wood chips and 4 percent green waste. Mass loading calculations were used to estimate Cr(VI) concentrations in agricultural soils based on a maximum loading rate of 40 tons of biomass per acre of agricultural soil. The results of the study were used by the Regulatory agency to determine that the application of biosolids did not constitute a health risk to workers applying the biosolids or to residences near the agricultural lands.

Client – United Kingdom Environmental Agency

Oversaw a comprehensive toxicological evaluation of methyl-*tertiary* butyl ether (M*t*BE) for the United Kingdom's Environment Agency. The evaluation included available data on the production, use, chemical characteristics, fate and transport, toxicology, and remediation of M*t*BE. The results of the evaluation have been used as a briefing tool for public health professionals.

Client – Confidential, Los Angeles, California

Prepared comprehensive evaluation of *tertiary* butyl alcohol (TBA) in municipal drinking water system. TBA is the primary breakdown product of M*t*BE, and is suspected to be the primary cause of M*t*BE toxicity. This evaluation will include available information on the production, use, chemical characteristics, fate and transport in the environment, absorption, distribution, routes of detoxification, metabolites, carcinogenic potential, and remediation of TBA. The results of the evaluation were used as a briefing tool for non-public health professionals.

Client – Confidential, Los Angeles, California

Prepared comprehensive evaluation of methyl *tertiary* butyl ether (MTBE) in municipal drinking water system. MTBE is a chemical added to gasoline to increase the octane

rating and to meet Federally mandated emission criteria. The evaluation included available data on the production, use, chemical characteristics, fate and transport, toxicology, and remediation of MTBE. The results of the evaluation have been were used as a briefing tool for non-public health professionals.

Client – Ministry of Environment, Lands & Parks, British Columbia

Dr. Clark assisted in the development of water quality guidelines for methyl tertiary-butyl ether (MTBE) to protect water uses in British Columbia (BC). The water uses to be considered includes freshwater and marine life, wildlife, industrial, and agricultural (e.g., irrigation and livestock watering) water uses. Guidelines from other jurisdictions for the protection of drinking water, recreation and aesthetics were to be identified.

Client: Confidential, Los Angeles, California

Prepared physiologically based pharmacokinetic (PBPK) assessment of lead risk of receptors at middle school built over former industrial facility. This evaluation is being used to determine cleanup goals and will be basis for regulatory closure of site.

Client: Kaiser Venture Incorporated, Fontana, California

Prepared PBPK assessment of lead risk of receptors at a 1,100-acre former steel mill. This evaluation was used as the basis for granting closure of the site by lead regulatory agency.

RISK ASSESSMENTS/REMEDIAL INVESTIGATIONS

Client: Confidential, Atlanta, Georgia

Researched potential exposure and health risks to community members potentially exposed to creosote, polycyclic aromatic hydrocarbons, pentachlorophenol, and dioxin compounds used at a former wood treatment facility. Prepared a comprehensive toxicological summary of the chemicals of concern, including the chemical characteristics, absorption, distribution, and carcinogenic potential. Prepared risk characterization of the carcinogenic and non-carcinogenic chemicals based on the exposure assessment to quantify the potential risk to members of the surrounding community. This evaluation was used to help settle class-action tort.

Client: Confidential, Escondido, California

Prepared comprehensive Preliminary Endangerment Assessment (PEA) of dense non-aqueous liquid phase hydrocarbon (chlorinated solvents) contamination at a former printed circuit board manufacturing facility. This evaluation was used for litigation support and may be used as the basis for reaching closure of the site with the lead regulatory agency.

Client: Confidential, San Francisco, California

Summarized epidemiological evidence for connective tissue and autoimmune diseases for product liability litigation. Identified epidemiological research efforts on the health effects of medical prostheses. This research was used in a meta-analysis of the health effects and as a briefing tool for non-public health professionals.

Client: Confidential, Bogotá, Columbia

Prepared comprehensive evaluation of the potential health risks associated with the redevelopment of a 13.7 hectares plastic manufacturing facility in Bogotá, Colombia The risk assessment was used as the basis for the remedial goals and closure of the site.

Client: Confidential, Los Angeles, California

Prepared comprehensive human health risk assessment of students, staff, and residents potentially exposed to heavy metals (principally cadmium) and VOCs from soil and soil vapor at 12-acre former crude oilfield and municipal landfill. The site is currently used as a middle school housing approximately 3,000 children. The evaluation determined that the site was safe for the current and future uses and was used as the basis for regulatory closure of site.

Client: Confidential, Los Angeles, California

Managed remedial investigation (RI) of heavy metals and volatile organic chemicals (VOCs) for a 15-acre former manufacturing facility. The RI investigation of the site included over 800 different sampling locations and the collection of soil, soil gas, and groundwater samples. The site is currently used as a year round school housing approximately 3,000 children. The Remedial Investigation was performed in a manner

that did not interrupt school activities and met the time restrictions placed on the project by the overseeing regulatory agency. The RI Report identified the off-site source of metals that impacted groundwater beneath the site and the sources of VOCs in soil gas and groundwater. The RI included a numerical model of vapor intrusion into the buildings at the site from the vadose zone to determine exposure concentrations and an air dispersion model of VOCs from the proposed soil vapor treatment system. The Feasibility Study for the Site is currently being drafted and may be used as the basis for granting closure of the site by DTSC.

Client: Confidential, Los Angeles, California

Prepared comprehensive human health risk assessment of students, staff, and residents potentially exposed to heavy metals (principally lead), VOCs, SVOCs, and PCBs from soil, soil vapor, and groundwater at 15-acre former manufacturing facility. The site is currently used as a year round school housing approximately 3,000 children. The evaluation determined that the site was safe for the current and future uses and will be basis for regulatory closure of site.

Client: Confidential, Los Angeles, California

Prepared comprehensive evaluation of VOC vapor intrusion into classrooms of middle school that was former 15-acre industrial facility. Using the Johnson-Ettinger Vapor Intrusion model, the evaluation determined acceptable soil gas concentrations at the site that did not pose health threat to students, staff, and residents. This evaluation is being used to determine cleanup goals and will be basis for regulatory closure of site.

Client –Dominguez Energy, Carson, California

Prepared comprehensive evaluation of the potential health risks associated with the redevelopment of 6-acre portion of a 500-acre oil and natural gas production facility in Carson, California. The risk assessment was used as the basis for closure of the site.

Kaiser Ventures Incorporated, Fontana, California

Prepared health risk assessment of semi-volatile organic chemicals and metals for a fifty-year old wastewater treatment facility used at a 1,100-acre former steel mill. This evaluation was used as the basis for granting closure of the site by lead regulatory agency.

ANR Freight - Los Angeles, California

Prepared a comprehensive Preliminary Endangerment Assessment (PEA) of petroleum hydrocarbon and metal contamination of a former freight depot. This evaluation was as the basis for reaching closure of the site with lead regulatory agency.

Kaiser Ventures Incorporated, Fontana, California

Prepared comprehensive health risk assessment of semi-volatile organic chemicals and metals for 23-acre parcel of a 1,100-acre former steel mill. The health risk assessment was used to determine clean up goals and as the basis for granting closure of the site by lead regulatory agency. Air dispersion modeling using ISCST3 was performed to determine downwind exposure point concentrations at sensitive receptors within a 1 kilometer radius of the site. The results of the health risk assessment were presented at a public meeting sponsored by the Department of Toxic Substances Control (DTSC) in the community potentially affected by the site.

Unocal Corporation - Los Angeles, California

Prepared comprehensive assessment of petroleum hydrocarbons and metals for a former petroleum service station located next to sensitive population center (elementary school). The assessment used a probabilistic approach to estimate risks to the community and was used as the basis for granting closure of the site by lead regulatory agency.

Client: Confidential, Los Angeles, California

Managed oversight of remedial investigation most contaminated heavy metal site in California. Lead concentrations in soil excess of 68,000,000 parts per billion (ppb) have been measured at the site. This State Superfund Site was a former hard chrome plating operation that operated for approximately 40-years.

Client: Confidential, San Francisco, California

Coordinator of regional monitoring program to determine background concentrations of metals in air. Acted as liaison with SCAQMD and CARB to perform co-location sampling and comparison of accepted regulatory method with ASTM methodology.

Client: Confidential, San Francisco, California

Analyzed historical air monitoring data for South Coast Air Basin in Southern California and potential health risks related to ambient concentrations of carcinogenic metals and volatile organic compounds. Identified and reviewed the available literature and calculated risks from toxins in South Coast Air Basin.

IT Corporation, North Carolina

Prepared comprehensive evaluation of potential exposure of workers to air-borne VOCs at hazardous waste storage facility under SUPERFUND cleanup decree. Assessment used in developing health based clean-up levels.

Professional Associations

American Public Health Association (APHA)
Association for Environmental Health and Sciences (AEHS)
American Chemical Society (ACS)
California Redevelopment Association (CRA)
International Society of Environmental Forensics (ISEF)
Society of Environmental Toxicology and Chemistry (SETAC)

Publications and Presentations:

Books and Book Chapters

Sullivan, P., **J.J. J. Clark,** F.J. Agardy, and P.E. Rosenfeld. (2007). *Synthetic Toxins In The Food, Water and Air of American Cities*. Elsevier, Inc. Burlington, MA.

Sullivan, P. and **J.J. J. Clark**. 2006. *Choosing Safer Foods, A Guide To Minimizing Synthetic Chemicals In Your Diet*. Elsevier, Inc. Burlington, MA.

Sullivan, P., Agardy, F.J., and **J.J.J. Clark**. 2005. *The Environmental Science of Drinking Water*. Elsevier, Inc. Burlington, MA.

Sullivan, P.J., Agardy, F.J., **Clark, J.J.J.** 2002. *America's Threatened Drinking Water: Hazards and Solutions*. Trafford Publishing, Victoria B.C.

Clark, J.J.J. 2001. "TBA: Chemical Properties, Production & Use, Fate and Transport, Toxicology, Detection in Groundwater, and Regulatory Standards" in *Oxygenates in the Environment.* Art Diaz, Ed.. Oxford University Press: New York.

Clark, J.J.J. 2000. "Toxicology of Perchlorate" in *Perchlorate in the Environment.* Edward Urbansky, Ed. Kluwer/Plenum: New York.

Clark, J.J.J. 1995. Probabilistic Forecasting of Volatile Organic Compound Concentrations At The Soil Surface From Contaminated Groundwater. UMI.

Baker, J.; **Clark, J.J.J.**; Stanford, J.T. 1994. Ex Situ Remediation of Diesel Contaminated Railroad Sand by Soil Washing. Principles and Practices for Diesel Contaminated Soils, Volume III. P.T. Kostecki, E.J. Calabrese, and C.P.L. Barkan, eds. Amherst Scientific Publishers, Amherst, MA. pp 89-96.

Journal and Proceeding Articles

Tam L. K.., Wu C. D., Clark J. J. and **Rosenfeld, P.E.** (2008) A Statistical Analysis Of Attic Dust And Blood Lipid Concentrations Of Tetrachloro-p-Dibenzodioxin (TCDD) Toxicity Equialency Quotients (TEQ) In Two Populations Near Wood Treatment Facilities. Organohalogen Compounds, Volume 70 (2008) page 002254.

Tam L. K.., Wu C. D., Clark J. J. and **Rosenfeld, P.E.** (2008) Methods For Collect Samples For Assessing Dioxins And Other Environmental Contaminants In Attic Dust: A Review. Organohalogen Compounds, Volume 70 (2008) page 000527

Hensley A.R., Scott, A., Rosenfeld P.E., **Clark, J.J.J.** (2007). "Attic Dust And Human Blood Samples Collected Near A Former Wood Treatment Facility." *Environmental Research.* 105:194-199.

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Selected References

CalEEMod™

Technical Paper

Methodology Reasoning and Policy Development of the California Emission Estimator Model

July 2011

South Coast Air Quality Management District
Bay Area Air Quality Management District
Sacramento Metropolitan Air Quality Management District
San Joaquin Valley Air Pollution Control District
Santa Barbara County Air Pollution Control District
San Luis Obispo Air Pollution Control District

The following sections discuss the three primary emission source sectors (construction, area, and operational), the factors and methodology used in CalEEMod that were different from other models such as URBEMIS, and the justification if different from the URBEMIS model, which has been widely used it the past for calculating criteria pollutant emissions from land use development projects.

Construction

A construction schedule is critical in determining the appropriate CARB OFFROAD emission factors for construction equipment because the emission factors changes each year. In addition, the peak daily emissions will be different if the schedule between construction phases (e.g., site preparation, grading, building construction, etc.) overlaps. CalEEMod was developed using a construction survey to determine the construction profile (equipment type, number of equipment, hours of activity, etc.) for each construction phase. When changing the construction schedule, the model does not automatically change the default construction equipment type. The equipment type dictates construction phase activity, such as acres graded per day. Fugitive dust is generated when material (e.g., from demolition objects) and soil (e.g., from site preparation and grading) are transported to and from the site.

For non-residential land uses, the default lot acreage value corresponds to the building footprint. The lot acreage is used to calculate grading values. Therefore, any additional graded area must be entered separately as "other paved surfaces" or other land use to ensure an accurate grading calculation. For residential land uses, the default lot acreage value is greater than the default square footage value because the values are derived from different sources. The default lot acreage per residential dwelling unit is from the ITE Trip Generation and the square footage per dwelling unit is from the California Energy Commission's Residential Appliance Saturation Survey (RASS). Thus, the lot acreage includes building footprint, paved areas and undeveloped areas, so no additional grading area need to be entered separately.

Wind-blown fugitive dust is not calculated in CalEEMod because of the number of input parameters required such as soil type, moisture content, wind speed, etc. This limitation could result in underestimated fugitive dust emissions if high wind and loose soil are substantial characteristics for a given land use/construction scenario.

Construction activity also involves on-road mobile source emissions from vehicles driven to and from the construction site by workers, vendors (e.g., water trucks, product deliveries, etc.), and haulers. In addition, fugitive dust is generated by these vehicles.

Finally, volatile organic compound (VOC) emissions are generated when the interior and exterior surface walls of the structures are painted.

Differences in methodology between CalEEMod and URBEMIS for the construction emissions sector are summarized in the following table.

Table 1 – Updated/New Features in CalEEMod during Construction Phase

CalEEMod Updated/New Feature	Justification for Change in Methodology	General Trends in CalEEMod as compared to URBEMS
Uses a construction profile (equipment type, hours of activity) based on SCAQMD construction survey	Uses documented data (URBEMIS survey data is not well documented). During the development of its localized significance thresholds, SCAQMD staff worked with construction and building industries to conduct a construction site survey gathering accurate information to better estimate emissions from construction equipment based on their typical operations. The SCAQMD hired a consultant to conduct construction site surveys throughout the South Coast Air Basin. The consultant surveyed approximately 50 construction sites and compiled information on the various construction phases including demolition, site preparation, construction of structures, etc. The survey was limited to 35 acres or less. For those projects sized larger, the data was extrapolated by increasing the number of construction phase days but not increasing the number of construction equipment on a given day.	• Increase in construction ROG, NOx, CO and SO2 • Decrease in construction PM (see grading activity)
Revises amount of acres graded	Acreage graded based on construction equipment ability (i.e., maximum acres a piece of equipment can pass over land in an 8-hr day) from Walker's Building Estimator's Reference Book. Grading in URBEMIS is based on 25% of total project acreage in one day.	• Decrease in PM emissions from grading
Modifies calculation methodology from material hauling	Provides a more specific calculation based on actual construction equipment and amount of material hauled. Although the user inputs the amount of material hauled, the model calculates exhaust and fugitive dust emissions based on 16 cubic yards per truck (an industry average). The model credits "phased" trips (i.e., the truck enters and leaves with a load, thus reducing the total number of trips in half).	• PM emissions increase or decrease depending upon user input

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California Environmental Protection Agency

Air Resources Board

INITIAL STATEMENT OF REASONS FOR RULEMAKING

STAFF REPORT

PROPOSED IDENTIFICATION OF DIESEL EXHAUST AS A TOXIC AIR CONTAMINANT

Prepared by the Staff of
the Air Resources Board and
the Office of Environmental Health Hazard Assessment

June 1998

specific compounds emitted from a variety of engine technologies, operating cycles, and fuel to characterize better any differences between old and new fuels and technologies and the potential impact on the toxicity of diesel exhaust.

HEALTH EFFECTS OF DIESEL EXHAUST EXPOSURE

The OEHHA reviewed and evaluated the potential for diesel exhaust to affect human health, and the associated scientific uncertainties. The OEHHA considered acute and chronic noncancer health impacts, and potential cancer health impacts. The SRP approved the OEHHA's health assessment at its April 22, 1998, meeting.

A number of adverse short-term (acute) health effects have been associated with exposures to diesel exhaust. Occupational exposures to diesel exhaust particles have been associated with significant cross-shift decreases in lung function. Increased cough, labored breathing, chest tightness, and wheezing have been associated with exposure to diesel exhaust in bus garage workers. A significant increase in airway resistance and increases in eye and nasal irritation were observed in human volunteers following one-hour chamber exposure to diesel exhaust. In acute and subchronic animal studies, exposure to diesel exhaust particles induced inflammatory airway changes, lung function changes, and increased the animals' susceptibility to infection.

A number of adverse long-term (chronic) noncancer effects have been associated with exposures to diesel exhaust. Occupational studies have shown that there may be a greater incidence of cough, phlegm and chronic bronchitis among those exposed to diesel exhaust than among those not exposed. Histopathological changes in the lung of diesel-exposed test animals reflect inflammation of the lung tissue. Reduced pulmonary function was noted in monkeys during long-term exposure. Reductions in pulmonary function have also been reported following occupational exposures in chronic studies.

Diesel exhaust particles can induce immunological allergic reactions and localized inflammatory responses in humans, as well as acting as an adjuvant for pollen allergy. Intranasal challenge with diesel exhaust particles in human volunteers resulted in an immunological response. Co-exposure to diesel exhaust particles and ragweed pollen resulted in an immune response greater than that following pollen or diesel exhaust particles alone. Effects of intratracheal, intranasal, and inhalation exposures of laboratory animals are supportive of the findings in humans. These effects include allergic reactions and inflammation, increased mucus secretion and respiratory resistance, and airway constriction.

The World Health Organization and the OEHHA have conducted further analyses of the dose-response relationships for several of the non-cancer, adverse effects of chronic exposures to diesel exhaust on the rat lung. These analyses gave a range of health risk guidance values of 2 to 21 $\mu g/m^3$ and support the adoption of 5 $\mu g/m^3$ which is also the 1993 U.S. EPA Reference Concentration. A U.S. EPA Reference Concentration or California Reference Exposure Level (REL) of a chemical is an estimate, with uncertainty spanning perhaps an order of magnitude, of the air concentration below which no noncancer adverse health effects are likely to occur from

lifetime exposure. This estimate takes into consideration persons who may be more sensitive than others to the effects of a chemical. The OEHHA concurs with the U.S. EPA in recommending 5 $\mu g/m^3$ as the chronic REL for diesel exhaust.

Diesel exhaust contains genotoxic compounds in both the vapor phase and the particle phase. Diesel exhaust particles or extracts of diesel exhaust particles are mutagenic in bacteria and in mammalian cell systems, and can induce adverse chromosomal changes. DNA adducts (representing genotoxins bound chemically to DNA) have been shown to increase following inhalation exposure of rodents and monkeys to whole diesel exhaust and have been found in mammalian cells following treatment with diesel exhaust particle extract. Elevated levels of DNA adducts have been associated with occupational exposure to diesel exhaust.

Over 30 human epidemiological studies have investigated the potential carcinogenicity of diesel exhaust. These epidemiological studies provide evidence consistent with a causal relationship between occupational diesel exhaust exposure and lung cancer. These studies, on average, found that long-term occupational exposures to diesel exhaust were associated with a 40 percent increase in the relative risk of lung cancer. The OEHHA analyzed the lung cancer findings for consistency and found that the association was unlikely to be due to bias or chance. Results of inhalation bioassays in the rat, and with less certainty in mice, have demonstrated the carcinogenic potential of diesel exhaust in animals, although the mechanisms by which diesel exhaust induces lung tumors in animals remain uncertain.

Other agencies or scientific bodies have studied the health effects of diesel exhaust. The National Institute of Occupational Safety and Health first recommended that whole diesel exhaust be regarded as a potential occupational carcinogen based upon animal and human evidence in 1988. The IARC concluded that diesel engine exhaust is probably carcinogenic to humans (Group 2A). Based upon the IARC findings, in 1990, the State of California under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) identified diesel exhaust as a chemical "known to the State to cause cancer." (Title 22, California Code of Regulations, section 12000.) The 1998 draft U.S. EPA document (Health Assessment Document for Diesel Emissions, Review Draft, February 1998) similarly concluded that diesel exhaust be considered a "probable" human carcinogen (category B1). This conclusion evolves from positive yet "limited" evidence in the human studies, a "sufficient" level of evidence in bioassays, and consideration of the supporting information from mutagenicity and genotoxicity data.

Risk assessments can use carcinogenicity data from either animal or human studies. For diesel exhaust, there are data from human epidemiological studies of occupationally exposed populations which are useful for quantitative risk assessment. On balance, the OEHHA concluded that available human data lend more confidence in the prediction of human risks than the data from the available animal studies because of the uncertainties in the animal studies and of extrapolating from animals to humans. Thus, the OEHHA preferred to derive the range of human risk estimates based only upon the epidemiological findings and not the animal data. Using data from a case-control study and a cohort study, the OEHHA estimated the risk (95 percent upper confidence limit) of lung cancer in the general population due to diesel exhaust. Because of

uncertainties in the actual workplace exposures, the OEHHA developed a variety of exposure scenarios to bracket the exposures that were plausible. Based on these exposure estimates, presented in Table 1-1, the range of resulting estimates of cancer unit risk is 1.3×10^{-4} to 2.4×10^{-3} $(\mu g/m^3)^{-1}$. The unit risk represents the 95 percent upper confidence limit of cancer risk per million people exposed per microgram of diesel exhaust particulate in a cubic meter of air over a 70-year lifetime. The SRP approved the range of risk estimated by the OEHHA. In addition, the SRP concluded that a value of 3×10^{-4} $(\mu g/m^3)^{-1}$ is a reasonable estimate of unit risk expressed in terms of diesel particulate (see Appendix II).

The OEHHA and ARB staffs recognize that the limited exposure information available contributes to the uncertainty of the dose response risk assessment based on the human studies. However, the overall magnitude of uncertainty is not atypical of the types of uncertainty encountered when the Board identified other TACs. The greater than usual uncertainty in the exposure estimates is substantially offset by the much smaller than usual range of extrapolation from the occupational exposures to the ambient air concentrations. Interspecies extrapolation uncertainty is not an issue in this diesel exhaust risk assessment. In addition, there are more than 30 human studies of more than one occupation that show overall an increase in lung cancer from diesel exhaust exposure.

Based on available scientific evidence, a level of diesel exhaust exposure below which no carcinogenic effects are anticipated has not been identified. This finding was approved by the SRP at its meeting on April 22, 1998.

As with other substances evaluated by the SRP and after reviewing the field of published peer reviewed research studies on diesel exhaust, the SRP indicated that additional research is appropriate to further clarify the health effects of diesel exhaust. The OEHHA and ARB staffs recognize that diesel exhaust health studies will continue. For example, the HEI, which is jointly funded by industry and the U.S. EPA, has started a five-year study to review key epidemiologic studies and make recommendations for the design of new studies. The OEHHA and ARB staffs will follow these efforts closely, and will provide support to the extent resources are available. If the outcome of this, or other future health studies, ultimately reduces uncertainties or improves the scientific basis for estimating diesel exhaust risk, the OEHHA and ARB staffs would consider such information. When research results become available, the TAC program has a process in place for further evaluation of new scientific evidence pertaining to a previously completed TAC risk assessment. The process specifically addresses the evaluation and response to submittals of new scientific information as evidence for review of a TAC risk assessment.

Table 1-1. Summary of Cancer Unit Risks According to Study, Exposure Assumptions, and Modeling Approaches.

		95% UCL Cancer Unit Risk ($\mu g/m^3)^{-1}$	95% Upper Confidence Limit of Cancer Risk per Million per Microgram of Diesel Exhaust Particulate in a Cubic Meter of Air Exposure Over a 70-year Lifetime
Garshick *et al*. (1987a) Case Control[1]			
Scenario[2]			
	A	2.4×10^{-3}	2400
	B	1.8×10^{-3}	1800
	C	1.0×10^{-3}	1000
	D	6.6×10^{-4}	660
	E	3.6×10^{-4}	360
Garshick *et al.* (1988) Cohort Study (Chapter 7)[3]			
Scenario			
	A	1.8×10^{-3}	1800
	B	1.4×10^{-3}	1400
	C	8.2×10^{-4}	820
	D	5.1×10^{-4}	510
	E	2.8×10^{-4}	280
Garshick *et al.* (1988) Cohort Study (Appendix D)[4]			
Scenario A			
	general multiplicative model	1.9×10^{-3}	1900
	biologically based [5]	3.8×10^{-4}	380
Scenario C			
	general multiplicative model	7.2×10^{-4}	720
	biologically based [5]	1.3×10^{-4}	130
	biologically based [6]	1.5×10^{-4}	150

EVALUATION OF NEED AND APPROPRIATE DEGREE OF CONTROL FOR DIESEL

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[1] ***Using published slope coefficient for hazard on years to diesel exhaust as described in Appendix III (Part B, Section 7.3.3).***

[2] ***A Ramp pattern of exposure plateauing in 1959 at the 1980 exposure level of 50 $\mu g/m^3$***

B Roof pattern of exposure peaking in 1959 at twice the 1980 exposure level of 40 $\mu g/m^3$

C Roof pattern of exposure peaking in 1959 at 3-fold the 1980 exposure level of 50 $\mu g/m^3$

D Roof pattern of exposure peaking in 1959 at 3-fold the 1980 exposure level of 80 $\mu g/m^3$

E Roof pattern of exposure peaking in 1959 at 10-fold the 1980 exposure level of 50 $\mu g/m^3$

[3] ***Using individual data to obtain a slope for hazard on years of exposure to diesel exhaust as described in Appendix III (Part B, Section 7.3.4)***.

[4] ***Applying time varying concentrations to individual data to obtain a slope of hazard on exposure as described in Appendix III (Part B, Appendix D***).

[5] ***6th/7 stage model.***

[6] ***7th/7 stage model.***

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United States
Environmental Protection
Agency

Health Assessment Document For Diesel Engine Exhaust

EPA/600/8-90/057F
May 2002

Health Assessment Document for Diesel Engine Exhaust

National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Washington, DC

atmosphere. It is not clear what the overall toxicological consequences of DE's transformations are because some compounds in the DE mixture are altered to more toxic forms while others are made less toxic.

1.5. EXPOSURE TO DIESEL EXHAUST

DPM mass (expressed as μg DPM/m^3) has historically been used as a surrogate measure of exposure for whole DE. Although uncertainty exists as to whether DPM is the most appropriate parameter to correlate with human health effects, it is considered a reasonable choice until more definitive information about the mechanisms of toxicity or mode(s) of action of DE becomes available. In the ambient environment, human exposure to DE comes from both on-road and nonroad engine exhaust. A large percentage of the U.S. population also is exposed to ambient $PM_{2.5}$, of which DPM is typically a significant constituent. Although this document does not provide an exposure assessment, DE exposure information is included to provide a context for the health effects information. Exposure estimates for the early to mid-1990s suggest that national annual average DE exposure from on-road engines alone was in the range of about 0.5 to 0.8 μg DPM/m^3 of inhaled air in many rural and urban areas, respectively. Exposures could be higher if there is a nonroad DE source that adds to the exposure from on-road vehicles. For example, preliminary estimates show that, on a national average basis, accounting for nonroad DE emissions adds another twofold to the on-road exposure. For localized urban areas where people spend a large portion of their time outdoors, the exposures are higher and, for example, may range up to 4.0 μg DPM/m^3 of inhaled air.

1.6. HEALTH EFFECTS OF DIESEL EXHAUST

Available evidence indicates that there are human health hazards associated with exposure to DE. The hazards include acute exposure-related symptoms, chronic exposure-related noncancer respiratory effects, and lung cancer. The health hazard conclusions are based on exhaust emissions from diesel engines built prior to the mid-1990s. With current engine use including some new and many more older engines (engines typically stay in service for a long time), the health hazard conclusions, in general, are applicable to engines currently in use. As new and cleaner diesel engines, together with different diesel fuels, replace a substantial number of existing engines, the general applicability of the health hazard conclusions will need to be re-evaluated. With new engine and fuel technology expected to produce significantly cleaner engine exhaust by 2007 (e.g., in response to new federal heavy duty engine regulations), significant reductions in public health hazards are expected for those engine uses affected by the regulations.

1.6.1. Acute (Short-Term Exposure) Effects

Information is limited for characterizing the potential health effects associated with acute or short-term exposure. However, on the basis of available human and animal evidence, it is concluded that acute or short-term (e.g., episodic) exposure to DE can cause acute irritation (e.g., eye, throat, bronchial), neurophysiological symptoms (e.g., lightheadedness, nausea), and respiratory symptoms (cough, phlegm). There also is evidence for an immunologic effect–the exacerbation of allergenic responses to known allergens and asthma-like symptoms. The lack of adequate exposure-response information in the acute health effect studies precludes the development of recommendations about levels of exposure that would be presumed safe for these effects.

1.6.2. Chronic (Long-Term Exposure) Noncancer Respiratory Effects

Information from the available human studies is inadequate for a definitive evaluation of possible noncancer health effects from chronic exposure to DE. However, on the basis of extensive animal evidence, DE is judged to pose a chronic respiratory hazard to humans. Chronic-exposure, animal inhalation studies show a spectrum of dose-dependent inflammation and histopathological changes in the lung in several animal species including rats, mice, hamsters, and monkeys.

This assessment provides an estimate of inhalation exposure of DE (as measured by DPM) to which humans may be exposed throughout their lifetime without being likely to experience adverse noncancer respiratory effects. This exposure level, known as the reference concentration (RfC) for DE of 5 $\mu g/m^3$ of DPM was derived on the basis of dose-response data on inflammatory and histopathological changes in the lung from rat inhalation studies. In recognition of the presence of DPM in ambient $PM_{2.5}$, it also is appropriate to consider the wealth of $PM_{2.5}$ human health effects data. In this regard, the 1997 National Ambient Air Quality Standard for $PM_{2.5}$ of 15 $\mu g/m^3$ (annual average concentration) also would be expected to provide a measure of protection from DPM, reflecting DPM's current approximate proportion to $PM_{2.5}$.

1.6.3. Chronic (Long-Term Exposure) Carcinogenic Effects

This assessment concludes that DE is "likely to be carcinogenic to humans by inhalation" and that this hazard applies to environmental exposures. This conclusion is based on the totality of evidence from human, animal, and other supporting studies. There is considerable evidence demonstrating an association between DE exposure and increased lung cancer risk among workers in varied occupations where diesel engines historically have been used. The human evidence from occupational studies is considered strongly supportive of a finding that DE

exposure is causally associated with lung cancer, though the evidence is less than that needed to definitively conclude that DE is carcinogenic to humans. There is some uncertainty about the degree to which confounders are having an influence on the observed cancer risk in the occupational studies, and there is uncertainty evolving from the lack of actual DE exposure data for the workers. In addition to the human evidence, there is supporting evidence of DPM's carcinogenicity and associated DPM organic compound extracts in rats and mice by noninhalation routes of exposure. Other supporting evidence includes the demonstrated mutagenic and chromosomal effects of DE and its organic constituents, and the suggestive evidence for bioavailability of the DPM organics in humans and animals. Although high-exposure chronic rat inhalation studies show a significant lung cancer response, this is not thought predictive of a human hazard at lower environmental exposures. The rat response is considered to result from an overload of particles in the lung resulting from the high exposure, and such an overload is not expected to occur in humans at environmental exposures.

Although the available human evidence shows a lung cancer hazard to be present at occupational exposures that are generally higher than environmental levels, it is reasonable to presume that the hazard extends to environmental exposure levels. While there is an incomplete understanding of the mode of action for DE-induced lung cancer that may occur in humans, there is the potential for a nonthreshold mutagenic mode of action stemming from the organics in the DE mixture. A case for an environmental hazard also is shown by the simple observation that the estimated higher environmental exposure levels are close to, if not overlapping, the lower range of occupational exposures for which lung cancer increases are reported. These considerations taken together support the prudent public health choice of presuming a cancer hazard for DE at environmental levels of exposure. Overall, the evidence for a potential cancer hazard to humans resulting from chronic inhalation exposure to DE is persuasive, even though assumptions and uncertainties are involved. While the hazard evidence is persuasive, this does not lead to similar confidence in understanding the exposure/dose-response relationship.

Given a carcinogenicity hazard, EPA typically performs a dose-response assessment of the human or animal data to develop a cancer unit risk estimate that can be used with exposure information to characterize the potential cancer disease impact on an exposed population. The DE human exposure-response data are considered too uncertain to derive a confident quantitative estimate of cancer unit risk, and with the chronic rat inhalation studies not being predictive for environmental levels of exposure, EPA has not developed a quantitative estimate of cancer unit risk.

In the absence of a cancer unit risk, simple exploratory analyses were used to provide a perspective of the range of possible lung cancer risk from environmental exposure to DE. The analyses make use of reported lung cancer risk increases in occupational epidemiologic studies,

and the differences between occupational and environmental exposure. The purpose of having a risk perspective is to illustrate and have a sense of the possible significance of the lung cancer hazard from environmental exposure. The risk perspective cannot be viewed as a definitive quantitative characterization of cancer risk nor is it suitable for estimation of exposure-specific population risks.

1.7. SOURCES OF UNCERTAINTY

Even though the overall evidence for potential human health effects of DE is persuasive, many uncertainties exist because of the use of assumptions to bridge data and knowledge gaps about human exposures to DE and the general lack of understanding about underlying mechanisms by which DE causes observed toxicities in humans and animals. A notable uncertainty of this assessment is whether the health hazards identified from studies using emissions from older engines can be applied to present-day environmental emissions and related exposures, as some physical and chemical characteristics of the emissions from certain sources have changed over time. Available data are not sufficient to provide definitive answers to this question because changes in DE composition over time cannot be confidently quantified, and the relationship between the DE components and the mode(s) of action for DE toxicity is/are unclear. While recognizing the uncertainty, for this assessment a judgment is made that prior-year toxicologic and epidemiologic findings can be applied to more current exposures, both of which use DPM mass in air as the measure of DE exposure.

Other uncertainties include the assumptions that health effects observed at high doses may be applicable to low doses, and that toxicologic findings in laboratory animals generally are predictive of human responses. In the absence of a more complete understanding of how DE may cause adverse health effects in humans and laboratory animals, related assumptions (i.e., the presence of a biological threshold for chronic respiratory effects based on cumulative dosage and absence of a threshold for lung cancer stemming from subtle and irreversible effects) are considered reasonable and prudent.

Although parts of this assessment, particularly the noncancer RfC estimate, have been derived with a generic consideration of sensitive subgroups within the population, the actual spectrum of the population that may have a greater susceptibility to DE is unknown and cannot be better characterized until more information is available regarding the adverse effects of DPM in humans. Increased susceptibility, for example, could result from above-average increases in DE deposition and retention in the respiratory system or intrinsic differences in respiratory system tissue sensitivity. There is no DE-specific information that provides direct insight to the question of differential human susceptibility. Given the nature of DE's noncancer effects on the respiratory system it would be reasonable, for example, to consider possible vulnerable

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The Report on Diesel Exhaust

This page last reviewed July 29, 2008

Findings of the Scientific Review Panel On

The Report on Diesel Exhaust

As Adopted at the Panel's April 22, 1998 Meeting

Pursuant to Health and Safety Code section 39661, the Scientific Review Panel (SRP / Panel) has reviewed the report *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant* by the staffs of the California Air Resources Board (ARB or Board) and the Office of Environmental Health Hazard Assessment (OEHHA) describing the public exposure to, and health effects of, diesel exhaust. The Panel members also reviewed the public comments received on this report.

Panel members participated in workshops devoted to discussion of the exposure and health issues associated with diesel exhaust in September 1994, January 1996, July 1997, and March 1998. The SRP reviewed the issues at its meetings in October 1997 and April 1998. A special meeting of the SRP was held on March 11, 1998, to hear testimony on health issues including the quantitative risk assessment from highly respected scientists invited by the Panel. Based on these reviews and information provided at scientific workshops and meetings, the SRP makes the following findings pursuant to Health and Safety Code section 39661:

Exposure Related Conclusions:

1. Diesel exhaust is a complex mixture of gases and fine particles emitted by a diesel-fueled internal combustion engine.
2. The gaseous fraction is composed of typical combustion gases such as nitrogen, oxygen, carbon dioxide, and water vapor. However, as a result of incomplete combustion, the gaseous fraction also contains air pollutants such as carbon monoxide, sulfur oxides, nitrogen oxides, volatile organics, alkenes, aromatic hydrocarbons, and aldehydes, such as formaldehyde and 1,3-butadiene and low-molecular weight polycyclic aromatic hydrocarbons (PAH) and PAH-derivatives.
3. One of the main characteristics of diesel exhaust is the release of particles at a markedly greater rate than from gasoline-fueled vehicles, on an equivalent fuel energy basis. The particles are mainly aggregates of spherical carbon particles coated with inorganic and organic substances. The inorganic fraction primarily consists of small solid carbon (or elemental carbon) particles ranging from 0.01 to 0.08 microns in diameter. The organic fraction consists of soluble organic compounds such as aldehydes, alkanes and alkenes, and high-molecular weight PAH and PAH-derivatives, such as nitro-PAHs. Many of these PAHs and PAH-derivatives, especially nitro-PAHs, have been found to be potent mutagens and carcinogens. Nitro-PAH compounds can also be formed during transport through the

atmosphere by reactions of adsorbed PAH with nitric acid and by gas-phase radical-initiated reactions in the presence of oxides of nitrogen.

4. Diesel exhaust includes over 40 substances that are listed by the United States Environmental Protection Agency (U.S. EPA) as hazardous air pollutants and by the ARB as toxic air contaminants. Fifteen of these substances are listed by the International Agency for Research on Cancer (IARC) as carcinogenic to humans, or as a probable or possible human carcinogen. Some of these substances are: acetaldehyde; antimony compounds; arsenic; benzene; beryllium compounds; bis(2-ethylhexyl)phthalate; dioxins and dibenzofurans; formaldehyde; inorganic lead; mercury compounds; nickel; POM (including PAHs); and styrene.
5. Almost all of the diesel particle mass is in the fine particle range of 10 microns or less in diameter (PM10). Approximately 94 percent of the mass of these particles are less than 2.5 microns in diameter. Because of their small size, these particles can be inhaled and a portion will eventually become trapped within the small airways and alveolar regions of the lung.
6. The estimated population-weighted average outdoor diesel exhaust PM10 concentration in California for 1995 is 2.2 microgram per cubic meter ($\mu g/m^3$). Several independent studies have reported similar outdoor air diesel exhaust PM10 concentrations. The 1995 estimated average indoor exposure concentration is approximately 1.5 $\mu g/m^3$.
7. The population time-weighted average total air exposure to diesel exhaust particle concentrations across all environments (including outdoors) is estimated to be 1.5 $\mu g/m^3$ in 1995. This total exposure estimate may underestimate many Californians' actual total exposure because it excludes elevated exposures near roadways, railroad tracks, and inside vehicles. Near-source exposures to diesel exhaust may be as much as five times higher than the 1995 population time-weighted average total air exposure. It also excludes other routes of exposure to diesel exhaust, such as ingestion and dermal absorption.
8. Diesel engine exhaust contains small carbonaceous particles and a large number of chemicals that are adsorbed onto these particles or present as vapors. These particles have been the subject of many studies because of their adverse effects on human health and the environment. A recent study conducted for the Health Effects Institute showed that, despite a substantial reduction in the weight of the total particulate matter, the total number of particles from a 1991-model engine was 15 to 35 times greater than the number of particles from a 1988 engine when both engines were operated without emission control devices. This suggests that more fine particles, a potential health concern, could be formed as a result of new technologies. Further study is needed since the extent of these findings only measured exhaust from two engines and engine technologies.
9. The major sources of diesel exhaust in ambient outdoor air are estimated to emit approximately 27,000 tons per year in 1995. On-road mobile sources (heavy-duty trucks, buses, light-duty cars and trucks) contribute the majority of total diesel exhaust PM10 emissions in California. Other mobile sources (mobile equipment, ships, trains, and boats) and stationary sources contribute the remaining emissions.
10. Significant progress has been made as a result of federal and state regulations that have addressed particulate matter levels from diesel engines. Emissions of on-road mobile source diesel exhaust PM10 in California are expected to decline by approximately 85 percent from 1990 to 2010 as a result of mobile source regulations already adopted by the ARB.
11. The results of a study funded by the ARB at the University of California, Riverside, indicate that the diesel exhaust from the new fuel tested contained the same toxic air contaminants as the old fuel, although their concentrations and other components may differ. Further research would be helpful to quantify the amounts of specific compounds emitted from a variety of

engine technologies, operating cycles, and fuel to characterize better any differences between old and new fuels and technologies.

Health Effects Associated with Diesel Exhaust:

12. A number of adverse short-term health effects have been associated with exposures to diesel exhaust. Occupational exposures to diesel exhaust particles have been associated with significant cross-shift decreases in lung function. Increased cough, labored breathing, chest tightness, and wheezing have been associated with exposure to diesel exhaust in bus garage workers. A significant increase in airway resistance and increases in eye and nasal irritation were observed in human volunteers following one-hour chamber exposure to diesel exhaust. In acute or subchronic animal studies, exposure to diesel exhaust particles induced inflammatory airway changes, lung function changes, and increased the animals' susceptibility to infection.
13. A number of adverse long-term noncancer effects have been associated with exposure to diesel exhaust. Occupational studies have shown that there may be a greater incidence of cough, phlegm and chronic bronchitis among those exposed to diesel exhaust than among those not exposed. Reductions in pulmonary function have also been reported following occupational exposures in chronic studies. Reduced pulmonary function was noted in monkeys during long-term exposure. Histopathological changes in the lung of diesel-exposed test animals reflect inflammation of the lung tissue. These changes include dose-dependent proliferations of Type II epithelial cells, marked infiltration of macrophages, plasma cells and fibroblasts into the alveolar septa, thickening of the alveolar walls, alveolar proteinosis, and focal fibrosis.
14. Studies have shown that diesel exhaust particles can induce immunological reactions and localized inflammatory responses in humans, as well as acting as an adjuvant for pollen allergy. Intranasal challenge with diesel exhaust particles in human volunteers resulted in increased nasal IgE antibody production and a significant increase in mRNA for pro-inflammatory cytokines. Co-exposure to diesel exhaust particles and ragweed pollen resulted in a nasal IgE response greater than that following pollen or diesel exhaust particles alone. Effects of intratracheal, intranasal, and inhalation exposures of laboratory animals are supportive of the findings in humans. These effects include eosinophilic infiltration into bronchi and bronchioles, elevated IgE response, increased mucus secretion and respiratory resistance, and airway constriction.
15. Based on the animal studies, the U.S. EPA determined a chronic inhalation Reference Concentration value of 5 $\mu g/m^3$ for noncancer effects of diesel exhaust. This estimate takes into consideration persons who may be more sensitive than others to the effects of diesel exhaust. The report supports the recommendation of 5 $\mu g/m^3$ as the California Reference Exposure Level (REL) (Table 1). It should be noted that this REL may need to be lowered further as more data emerge on potential adverse noncancer effects from diesel exhaust.
16. Diesel exhaust contains genotoxic compounds in both the vapor phase and the particle phase. Diesel exhaust particles or extracts of diesel exhaust particles are mutagenic in bacteria and in mammalian cell systems, and can induce chromosomal aberrations, aneuploidy, and sister chromatid exchange in rodents and in human cells *in vitro*. Diesel exhaust particles induced unscheduled DNA synthesis *in vitro* in mammalian cells. DNA adducts have been isolated from calf thymus DNA *in vitro* following treatment with diesel exhaust particle extracts. DNA adducts have been shown to increase following inhalation exposure of rodents and monkeys to whole diesel exhaust. Elevated levels of DNA adducts have been associated with occupational exposure to diesel exhaust. Results of inhalation bioassays in the rat, and with lesser certainty in mice, have demonstrated the carcinogenicity of diesel exhaust in test animals, although the mechanisms by which diesel exhaust induces lung tumors in animals

remains uncertain.

17. Over 30 human epidemiological studies have investigated the potential carcinogenicity of diesel exhaust. These studies, on average, found that long-term occupational exposures to diesel exhaust were associated with a 40 percent increase in the relative risk of lung cancer. The lung cancer findings are consistent and the association is unlikely to be due to chance. These epidemiological studies strongly suggest a causal relationship between occupational diesel exhaust exposure and lung cancer.
18. Other agencies or scientific bodies have evaluated the health effects of diesel exhaust. The National Institute of Occupational Safety and Health first recommended in 1988 that whole diesel exhaust be regarded as a potential occupational carcinogen based upon animal and human evidence. The International Agency for Research on Cancer (IARC) concluded that diesel engine exhaust is probably carcinogenic to humans and classified diesel exhaust in Group 2A. Based upon the IARC findings, in 1990, the State of California under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) identified diesel exhaust as a chemical "known to the State to cause cancer." The U.S. EPA has proposed a conclusion similar to IARC in their draft documents. The 1998 draft U.S. EPA document concluded similarly that there was sufficient animal evidence of carcinogenicity and that the human evidence was limited.
19. There are data from human epidemiological studies of occupationally exposed populations which are useful for quantitative risk assessment. The estimated range of lung cancer risk (upper 95% confidence interval) based on human epidemiological data is 1.3×10^{-4} to 2.4×10^{-3} $(\mu g/m^3)^{-1}$ (Table 2). After considering the results of the meta-analysis of human studies, as well as the detailed analysis of railroad workers, the SRP concludes that 3×10^{-4} $(\mu g/m^3)^{-1}$ is a reasonable estimate of unit risk expressed in terms of diesel particulate. Thus this unit risk value was derived from two separate approaches which yield similar results. A comparison of estimates of risk can be found in Table 3.
20. Based on available scientific information, a level of diesel exhaust exposure below which no carcinogenic effects are anticipated has not been identified.
21. Based on available scientific evidence, as well as the results of the risk assessment, we conclude that diesel exhaust be identified as a Toxic Air Contaminant.
22. As with other substances evaluated by this Panel and after reviewing the field of published peer reviewed research studies on diesel exhaust, additional research is appropriate to clarify further the health effects of diesel exhaust. This research may have significance for estimating the unit risk value.
23. The Panel, after careful review of the February 1998 draft SRP version of the ARB report, *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant*, as well as the scientific procedures and methods used to support the data, the data itself, and the conclusions and assessments on which the Report is based, finds this report with the changes specified during our October 16, 1997, meeting and as a result of comments made at the March 11, 1998, meeting, is based upon sound scientific knowledge, methods, and practices and represents a complete and balanced assessment of our current scientific understanding.

For these reasons, we agree with the science presented in Part A by ARB and Part B by OEHHA in the report on diesel exhaust and the ARB staff recommendation to its Board that diesel exhaust be listed by the ARB as a Toxic Air Contaminant.

I certify that the above is a true and correct copy of the findings adopted by the Scientific Review

Panel on April 22, 1998.

Sincerely,
/s/
John R. Froines, Ph.D.
Acting Chairman
Scientific Review Panel

TABLE 1

NONCANCER HEALTH VALUES APPROVED BY THE

SCIENTIFIC REVIEW PANEL

1998

Compound	Health Value	End Point
Acetaldehyde	9 $\mu g/m^3$	Respiratory System
Diesel Exhaust	**5 $\mu g/m^3$**	**Respiratory System**
Inorganic Lead	4.6×10^{-4} ($\mu g/m^3)^{-1}$	Cardiovascular Mortality
Perchloroethylene	35 $\mu g/m^3$	Alimentary System (Liver)
$\mu g/m^3$: Microgram Per Cubic Meter		

TABLE 2

CANCER POTENCIES APPROVED BY THE SCIENTIFIC REVIEW PANEL

FROM 1984 TO 1998

(In Order of Cancer Potency)

Compound	Unit Risk $(\mu g/m^3)^{-1}$	Range $(\mu g/m^3)^{-1}$

TABLE 3

COMPARISON OF OTHER ORGANIZATIONS' ESTIMATED 95 PERCENT UPPER CONFIDENCE LIMITS OF LIFETIME RISK PER $\mu g/m^3$ DIESEL PARTICULATE MATTER FROM RISK ASSESSMENTS BASED ON EPIDEMIOLOGIC DATA WITH OEHHA ESTIMATES

Method	Unit Risk / Range	Basis of Assessment	Reference
Epidemiologic Analysis	3×10^{-4}	Based on Smoking-Adjusted Pooled RR	Smith, 1998
Epidemiologic Analysis[b]	**3.6×10^{-4} to 2.4×10^{-3}**	**Case-Control Study of Garshick et al., 1987**	**OEHHA, Part B, Section 7.3.3**
Epidemiologic Analysis	**2.8×10^{-4} to 1.8×10^{-3}**	**Cohort Study of Garshick et al., 1988**	**OEHHA, Part B, Section 7.3.4**
Epidemiologic Analysis	**1.3 to 7.2×10^{-4}**	**Cohort Study, Time Varying Conc., Roof (3,50) Pattern**	**OEHHA, Part B, Appendix D**
Epidemiologic Analysis	**3.8×10^{-4} to 1.9×10^{-3}**	**Cohort Study, Time Varying Conc., Ramp (1,50) Pattern**	**OEHHA, Part B, Appendix D**
Epidemiologic Analysis	1.4×10^{-3}	London Transport Study[c]	Harris, 1983
Epidemiologic Analysis	2×10^{-3}	Epidemiologic Data of Garshick (Top End of U.S. EPA's Range)	U.S. EPA, 1998
Epidemiologic Analysis	1.3×10^{-4} to 1.3×10^{-2}	Using Smoking-Adjusted RR and Exposures of 5 or 500 $\mu g/m^3$	OEHHA, Part B, Section 7.3; Bracketed Risk Bounds

a) Bolded values are included in OEHHA's range of risk.

b) Obtained by applying Harris' slope of 5×10^{-4} ($\mu g/m^3 \times yr)^{-1}$ to California life table.

Air Toxics Program
Particulate Emissions from Diesel-Fueled Engines as a TAC
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Interim Guidance for Sampling Agricultural Fields for School Sites (Second Revision)

California Department of Toxic Substances Control
California Environmental Protection Agency

August 26, 2002

Preface

Effective January 1, 2000, new California Department of Education statutes require the Department of Toxic Substances Control (DTSC) of the California Environmental Protection Agency (CalEPA) to review environmental assessments for proposed new school sites and/or new construction school expansion projects. Some of these sites are situated on agriculture land where residual agricultural chemicals may remain in the soil. In June 2000, DTSC issued "Interim Guidance for Sampling Agricultural Soils" to provide a uniform approach for evaluating former agricultural properties where pesticides have been applied. Since this guidance was issued, over 75 agricultural sites have been evaluated across California with the majority in the Sacramento-San Joaquin Valley, Oxnard Plains, and Imperial Valley. The most commonly detected pesticides have been DDT and it's derivatives DDD and DDE, toxaphene, dieldrin, and aldrin. Of these pesticides, toxaphene has been the major pesticide driving unacceptable levels of risk requiring remediation by soil removal. These results and the experience of working with the guidance has allowed for refinement of the original guidance. The revised guidance contained in this document reflects these refinements.

This guidance is intended to supplement the DTSC Preliminary Endangerment Assessment (PEA) Guidance Manual (Manual), CalEPA 1994

(Second Printing, June 1999). Data obtained from the investigations should be evaluated for potential health risks according the PEA Manual. This guidance is not intended to diminish the need to take focused, authoritative samples at site locations commonly associated with hazardous substances releases nor replace guidance provided by the PEA Guidance Manual. This guidance in not applicable to areas where pesticides were mixed, stored, disposed, or areas where pesticides may have accumulated, such as ponds and drainage ditches.

The scope of this document is limited to evaluating only agricultural fields during a PEA or other initial sampling investigation related to proposed new and/or expanded school sites. These are properties (or portions of properties) where pesticides were uniformly applied for agricultural purposes consistent with normal application practices, and where other non-agriculturally related activities have been absent. The data obtained from the sampling analyses will be incorporated into the PEA Report, including performing a risk analysis in accordance with the guidance in the PEA Manual.

This guidance does not apply to disturbed land, such as, land that has been graded in preparation for construction, areas where imported soil has been brought in, or any other activity that would redistribute or impact the soil, other than normal disking and plowing.

This guidance is an on-going effort to streamline the characterization of agricultural sites. As additional knowledge and experience is obtained, DTSC may modify this guidance, as appropriate.

1.0 PURPOSE

This guidance was prepared for use in evaluating soil at proposed new school sites and/or new school construction expansion projects that are currently, or were previously used for certain types of agricultural activities where residual agricultural chemicals may pose a threat to human health and the environment. This guidance is intended to supplement the DTSC Preliminary Endangerment Assessment (PEA) Guidance Manual (Manual), CalEPA 1994 (Second Printing, June 1999), and provide a uniform and streamlined approach for evaluating agricultural soils. It is intended to assist environmental assessors in designing initial investigations or developing Preliminary Endangerment Assessment (PEA) Work Plans for sites with certain historical agricultural uses. The analytical data obtained are to be incorporated into a risk analysis and PEA Report performed in accordance with the guidance in the PEA Manual.

2.0 IDENTIFYING ELIGIBLE AGRICULTURAL SITES

2.1 Eligible Sites

This guidance is specific to agricultural lands where pesticides and/or fertilizers were presumably applied, more or less uniformly, for agricultural purposes consistent with normal application practices. It is applicable to agricultural land that is currently under cultivation with row, fiber or food crops, orchards, or pasture. It is also applicable to fallow and former agricultural land that is no longer in production and has not been disturbed beyond normal disking and plowing practices. Each field of the same crop is assumed to have been watered, fertilized and treated with agricultural chemicals to the same degree across the field. Because of this homogeneous application, contaminant levels are expected to be similar at any given location within the field. This is the underlying premise of the guidance, and one that must be verified at the scoping stage of the PEA process.

2.2 Sites To Which The Guidance Does Not Apply

This guidance is not applicable to agricultural land under or adjacent to structures such as residences, barns, or other outbuildings. Pesticide mixing/loading areas, fence lines, ditches, canals, berms, and other areas that may have been treated differently from an agricultural field are not considered in this guidance. Also excluded are animal facilities such as cattle and poultry barns, settling ponds, and manure piles. This guidance does not apply to former agricultural land that has been graded for construction or other purposes, that has received fill, or has had parking lots or structures placed on it following active use as an agricultural field. An urban residential area that was agricultural land in the past does not qualify for this guidance since the construction of the residences would have resulted in the disturbance and redistribution of potential agricultural contaminants in the soil. These excluded areas require biased, discrete sampling as opposed to the sampling for agricultural fields discussed in this document.

3.0 SAMPLING STRATEGIES

3.1 Sampling Frequency

Sampling frequency may vary depending on the size of the site and conditions found. When the site has been uniformly used for a single agricultural crop, the presumption is that agricultural chemicals were applied equally to the site in any given year and that their distribution will be relatively uniform. **When differing agricultural crops were produced on different areas of the site, each area should be**

addressed separately and the sampling rate should be sufficient to characterize each area.

The sampling pattern should be sufficient to characterize the site. Recommended numbers of sampling locations are provided in Table 1. For sites two acres or less, discrete samples should be collected on ¼ acre centers. For sites between two and four acres, a total of eight locations, evenly spaced across the site, should be sampled. For sites greater than four acres and up to 20 acres, discrete samples should be collected on ½ acre centers, and for sites between 21 and 100 acres, on 1-acre centers. For sites greater than 100 acres, DTSC should be consulted for the appropriate number of sampling locations. Compositing of samples is discussed in Section 4.5.

Table 1: Recommended Number of Sampling Locations

Land Size	Suggested Minimum Sampling Locations
One (1) to two (2) acres	Discrete samples taken on ¼ acre centers
Greater than two (2) up to four (4) acres	Discrete samples taken from eight (8) locations evenly spaced across the site
Greater than four (4) up to twenty (20) acres	Eight (8) composite samples from discrete samples taken on half-acre centers.
Twenty-one (21) to sixty (60) acres	Fifteen (15) composite samples from discrete samples taken on one (1) acre centers.
Sixty-one (61) to one hundred (100) acres	Twenty five (25) composite samples from discrete samples taken on one (1) acre centers
Greater than one hundred (100) acres	Consult with DTSC

3.2 Sampling Depth

Each location should be sampled to include one surface sample (0 to 6 inches) and one subsurface sample (2 to 3 foot range). [Note: 0 inches means first encountered soil. Thick mats of vegetable material, roots, and other extraneous material should not be sampled.]

3.3 Sample Collection

Sampling both the furrows and beds of existing rows will detect the greatest variability in the residuals. Some methods of pesticide application will favor residuals in the beds while others favor the furrows. In fields where rows remain, roughly half of the samples should be gathered from the furrows and half from the beds in an alternating pattern. Orchards should have the sampling locations placed at the current drip line for the trees, under the canopy, between the tree rows, and between the trees within a row. For sites with slopes, swales, or other uneven topography, sampling from centers should be modified to include samples from those areas where surface water would be expected to flow and accumulate.

3.4 Offsite Background Samples

A minimum of four offsite locations must be sampled at the surface (0 to 6 inches) to determine background or ambient levels of heavy metals in the area. The samples must be collected near the site, preferably one from each of the four sides. The soil type of the offsite samples should be the same as the site samples, and if possible, the offsite samples should be collected from areas that have not been impacted by agricultural or industrial chemicals. If other properties in the area have gone through the PEA process, it may be possible to use data from these sites for establishing background metal concentrations providing that soil types are compatible. This may only be done in consultation with the DTSC Project Manager.

4.0 <u>ANALYSES</u>

4.1 Identifying Agricultural Chemicals Used on the Site

When the land is under active agricultural production, the grower should be interviewed to determine the types and amounts of pesticides historically used at the site. The County Agricultural Commissioner should also be consulted to verify pesticide usage on the property. The Agricultural Commissioner is required to maintain this information for three years, but often will have extensive knowledge of the farming practices over many years. A local or specialized farm advisor such as the University of California Cooperative Extension Agent is another source of information for farming practices in the area. These consultations should occur during the scoping phase of the investigation. For those sites that have not been actively farmed in the past three years, obtaining accurate information is more difficult. Information from surrounding or neighboring agricultural operations on the types of crops grown in the area during the time of active farming can provide clues on what chemicals may have been applied.

4.2 Chemicals of Potential Concern (COPC): Pesticides

The chemicals of greatest concern are those that persist in the environment. For the majority of newer pesticides persistence is limited to a few days; however, organochlorine pesticides (OCPs) can still persist in soil at levels of health concern for many years following application. Unless it can be documented that OCPs were not used on the property, they must be considered COPC. Paraquat also has a relatively long persistence in the soil. Paraquat should also be considered a COPC if there is a history of its use on the property. Under certain conditions, such as in rice growing fields, near surface conditions exist that establish anaerobic soil over an extended time. For these situations, anaerobically stable pesticides such as ametryn, cryomazine, and thiabendazole should also be considered as COPC. The selection of COPCs should be done in consultation with the DTSC project manager and toxicologist assigned to the project.

4.3 Chemicals of Potential Concern (COPC): Metals (Inorganic Elements)

Heavy metals have been applied to agricultural fields, both as pesticides and fertilizers. To ensure that the concentrations of these metals in site soils do not pose a potential heath risk or hazard, the CAM 17 metals must be considered as COPC. Heavy metals are also evaluated to detect natural mineral deposits that may pose an unacceptable risk.

4.4 Discrete Samples

For sites four acres or less, each of the surface discrete samples must be analyzed for OCPs and CAM 17 metals. Analysis for other pesticides may be necessary, depending on the history of agricultural activities at the site. Offsite background samples should be analyzed for CAM 17 metals only. Subsurface samples should be frozen and held for analysis pending the outcome of the surface sampling results. No deterioration is expected during the time period required to complete the PEA.

4.5 Composite Samples

While the analysis of discrete samples is preferred, it is recognized that for large sites this may not be practical. Since this guidance assumes a relatively even distribution of chemicals across the site, compositing of discrete samples may be considered when the area to be sampled is greater than four acres.

4.5.1 Number of Composite Samples

The minimum number of composite samples analyzed is dependent on the size of the site (see Table 1). Compositing is not applicable for sites four acres or less. For sites greater than four acres and up to 20 acres, a minimum of eight composite samples is required. For sites 21 to 60 acres, a minimum of 15 composite samples is required. For sites between 61 and 100 acres, the minimum number of composite samples is 25. For sites over 100 acres, DTSC should be consulted for the appropriate number of composite samples.

4.5.2 Makeup of Composite Samples

Composite surface samples may be made up of a maximum of four discrete surface samples. The discrete samples must be from adjacent sampling locations. In cases where two crops were grown on the site, only discrete samples from within the same crop area may be composited.

4.5.3 Preparation of Composite Samples

The discrete samples should be individually mixed and uniformly split by the laboratory or trained field staff prior to compositing. Mixing and compositing should be performed under uniform, controlled conditions. The unused portion of each discrete sample should be frozen and archived in case additional analysis is warranted from the composite results. The samples may be discarded when the PEA process has been completed and approved by the DTSC.

4.6 Laboratory Analyses

4.6.1 Methods

The analytes of primary concern are OCPs and some of the CAM 17 metals. Depending on the site history, analysis of other types of pesticides may be required. OCPs should be analyzed using U.S. EPA 8081A or equivalent. Metals must be analyzed using the U.S. EPA 6000/7000 series. If the site history indicates other classes of persistent pesticides should be evaluated, DTSC should be consulted for the acceptable method of analysis and appropriate detection limits.

4.6.2 Detection Limits

The actual detection limits obtained will vary depending on the particular analyte. For OCPs, the analytes typically causing detection limit concerns in agricultural fields

are aldrin, dieldrin, and toxaphene. The detection limits should be 0.005 mg/kg for aldrin, dieldrin, and 0.100 mg/kg for toxaphene. Table 3 lists the detection limits for several OCPs and paraquat.

In samples with elevated DDT, the detected concentration may be above the range of calibration. This can result in the analytical laboratory diluting the sample for reanalysis, and then reporting only the final result. In these cases, the reported detection limits for aldrin, dieldrin, and toxaphene may exceed the detection limits needed for determining potential health effects. Ideally the laboratory should be asked to report if those three analytes were detected in the first analysis prior to dilution. Multiple analyses of the same samples may be required to obtain the data necessary for risk assessment purposes.

Table 2. Analytical Methods and Detection Limits for Selected OCPs and Paraquat

Pesticide	Methods	CAS No.[1]	DL[2] mg/kg
ALDRIN	8081A, 8270C	309-00-2	0.005
CHLORDANE	8081A	57-74-9	0.10
CHLORONEB	8081A (R)	2675-77-6	100
DBCP	8081A	96-12-8	0.01
DDD	8081A	72-54-8	0.10
DDE	8081A	72-55-9	0.10
DDT	8081A	50-29-3	0.10
DIELDRIN	8081A	60-57-1	0.005
HEPTACHLOR	8081A, 8270C	76-44-8	0.10
HEXACHLOROBENZENE	8081A, 8121, 8270C, 8275, 8410	118-74-1	0.30
LINDANE	8081A	58-89-9	0.10
METHOXYCHLOR	8081A	72-43-5	0.40
MIREX	8081A(R), 8270C	2385-85-5	0.10
PARAQUAT_DICHLORIDE	Zeneca SOP RAM 272/01; Chevron RM 8-10; 549.1*	4685-14-7	270
TOXAPHENE	8081A, 8270C	8001-35-2	0.1
TRIFLURALIN	8091, 8081A(R), 8270C	1582-09-8	63

*Water and Wastewater Methods. Soil must be extracted and the method validated by the laboratory for a soil matrix.
(R) = must be requested for inclusion in the method
CAS No[1] = Chemical Abstract Service registry number
DL[2] = Detection Limit recommended for risk assessment purposes

4.6.3 Pesticide Analyses

Each of the surface samples, discrete or composite, must be analyzed for OCPs. Analysis for other classes of persistent pesticides may be required as indicated by the agricultural history of the site. When using composites, each discrete sample associated with the composite sample having the highest detected concentration of OCPs must be analyzed.

4.6.4 Metal Analyses (Inorganic Elements)

Each of the background and a minimum of four (4) on-site surface samples must be analyzed for the CAM 17 metals. In addition, each of the on-site discrete surface samples must be analyzed for arsenic. When samples are composited, one (1) discreet sample from each composite must be analyzed for arsenic. The number of discrete samples analyzed for arsenic does not need to be greater than the number of total composite samples used for OCP analysis. The subsurface samples need only be analyzed for CAM 17 metals and arsenic if the concentration of an element detected is above the background concentration for that element. Analysis of additional subsurface samples may be requested by DTSC.

4.6.5 Quality Control

Quality control procedures specified in SW-846 must be followed. A matrix spike/matrix spike duplicate on one soil sample per batch of samples must be performed to demonstrate that the targeted pesticide(s) can be recovered from the soil investigated. Highly organic topsoil may interfere with proper extraction of pesticides. The laboratory data package must include a summary of the quality control sample results: blanks, matrix spike/matrix spike duplicate, surrogate recoveries, laboratory control samples, etc., as specified by the method. The laboratory should provide a signed narrative stating whether the QC was met and listing any discrepancies.

5.0 REPORTING

5.1 Format

The results of the sampling effort are to be reported in a Preliminary Endangerment Assessment (PEA) as described in the DTSC Preliminary Endangerment Assessment (PEA) Guidance Manual (Manual), CalEPA 1994 (Second Printing, June 1999).

5.2 Evaluating Metals (Inorganic Elements) Data

Using a robust statistical procedure to determine if on-site metal concentrations are indicative of background conditions or the result of site-related activities can be problematic because of the limited number of background samples collected at any one site. DTSC is in the process of establishing background metals concentrations for specific school districts. If the site is in a school district for which DTSC background levels have been established, those values should be used. If DTSC background levels are not available, then a defensible procedure for comparing on-site with background metals should be used. The Staff Toxicologist assigned to the project should be consulted on the most appropriate method of comparison.

5.3 Data Interpretation

All detected pesticides, and any onsite metals above background must be evaluated in a risk assessment as described in the DTSC PEA Guidance Manual. In the initial screening analysis, the highest concentration of each detected pesticide and metal above background must be used as the exposure point concentration in the risk assessment. If the maximum concentrations detected on site pose an unacceptable risk or hazard, a spatial analysis should be conducted to determine if the elevated levels represent a “hot spot”, or are representative of concentrations across the site. In those cases where the elevated concentrations are determined to be one or more “hot spots”, risk or concentration isopleths should be constructed to differentiate between those areas of the site in need of further action, and those where no further action is required. Any deviations from these analyses must be approved by the Staff Toxicologist assigned to the project. For sites with elevated levels of chlordane, it may be necessary to determine if the concentrations detected would pose an unacceptable risk from indoor air exposures, as evaluated with the Johnson and Ettinger Indoor Air Model. The DTSC Staff Toxicologist assigned to the project should be consulted for further guidance if necessary.

6.0 ADDITIONAL SOURCES OF INFORMATION

6.1 Pesticide Physical Properties and Half-Lives

http://ace.orst.edu/info/extoxnet/pips/ghindex.html
http://www.arsusda.gov/rsml/ppdb1.html

6.2 Active Pesticide Ingredient by Brand Name

http://www.cdpr.ca.gov/docs/label/prodnam.htm
http://www.cdpr.ca.gov/ - see databases
Farm Chemicals Handbook, current edition, Meister Publishing Company, Willoughby, Ohio.

6.3 Maximum Application Rates

http://ace.orst.edu/info/extoxnet/
Agricultural Chemicals – Thomas Publications, Fresno, CA

6.4 Pesticide Usage by Year, County, and Crop

http://www.ipm.ucdavis.edu/PUSE/puse1.html
http://www.cdpr.ca.gov/ - see databases

6.5 Test Methods

http://www.epa.gov/epaoswer/hazwaste/test/
SW-846: USEPA, *Test Methods for Evaluating Solid Waste, Physical/Chemical Methods, Third Edition, Current Revision*

6.6 Pesticide Toxicology Information

http://ace.orst.edu/info/extoxnet/ghindex.html
http://www.state.nj.us/health/eoh/rtkweb/rtkhsfs.htm