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September 23, 2019

Humboldt County Board of Supervisors
825 5th Street, Room 111
Eureka, CA 95501

RE: Formal Withdrawal and Substitution of Appeal of Planning Commission Approval of Applications 13319/SP16-868, 13328/SPl6-870, 13339 /SPl6-871, and 13346/SP16-872 submitted by Michael Brosgart and Arielle Brosgart; APN 516-111-064

To the Humboldt County Board of Supervisors:

On September 19, 2019, this office prepared an appeal letter (proposed appeal) on behalf of the Humboldt Bay Municipal Water District (the District) regarding the September 5, 2019 Planning Commission decision to approve applications 13319/SP16-868, 13328/SPl6-870, 13339 /SPl6-871, and 13346/SP16-872 submitted by Michael Brosgart and Arielle Brosgart for APN 516-111-064. Prior to submission, the District's Board of Directors reviewed the proposed appeal and requested revisions, which were incorporated into a subsequent version (revised appeal). However, due to clerical error, the proposed appeal was submitted instead of the revised appeal.

Pursuant to this letter, the District formally withdraws the proposed appeal, submitted September 19, 2019, and substitutes the revised appeal, attached as Attachment 1.

Respectfully,

Anne Baptiste

cc: Humboldt Bay Municipal Water District
California Department of Toxic Substances Control
California Department of Fish and Wildlife
Humboldt Baykeeper
North Coast Regional Water Quality Control Board

Attachment 1

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September 19, 2019

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825 5th Street, Room 111
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RE: Appeal of Planning Commission Approval of Applications 13319/SP16-868, 13328/SPl6-870, 13339 /SPl6-871, and 13346/SP16-872 submitted by Michael Brosgart and Arielle Brosgart; APN 516-111-064

To the Humboldt County Board of Supervisors:

The Humboldt Bay Municipal Water District (the District) hereby appeals the September 5, 2019 Planning Commission decision to approve applications 13319/SP16-868 (Volatile Manufacturing), 13328/SPl6-870 (Non-Volatile Manufacturing), 13339 /SP16-871 (Distribution), and 13346/SP16-872 (Processing) (collectively, the Project), submitted by Michael and Arielle Brosgart (Applicants) for APN 516-111-064. The District appeals the Planning Commission's decision to approve the Project and adopt a mitigated negative declaration (MND) despite substantial evidence in the record providing a fair argument that the Project will have significant environmental impacts. Because there is a fair argument of significant environmental impacts, the California Environmental Quality Act (CEQA) mandates preparation of an environmental impact report (EIR) for the Project to analyze the full scope of impacts prior to project approval. The District reserves the right to submit further information in support of its appeal of the Planning Commission's actions before the Board of Supervisors.

The Project proposes volatile and non-volatile extracting manufacturing, processing, and distribution on a 1.77-acre site that lies approximately 550 feet from Hall Creek, which drains into the Mad River, and approximately 2,000 feet from the Mad River itself.

The District is a municipal water district, which supplies high quality water to the greater Humboldt Bay Area, including drinking water to 88,000 residents of Humboldt County. It operates intake wells in the Mad River, which are located downstream of both the Project site and the point at which Hall Creek flows into the Mad River.

The MND fails to address the fact that the Project may result in pentachlorophenol- (PCP-) and/or dioxin-contaminated groundwater or soil running offsite into Hall Creek and/or the Mad River. Because the District's intake wells lie downstream, contaminants that flow off of the Project site will

flow into the County's drinking water supplies. This is an environmental impact that must be considered in an EIR.

I. The Project improperly relies on a mitigated negative declaration where there is a fair argument that the Project will result in significant environmental impacts related to contaminated soils and groundwater.

A. A fair argument of significant environmental impacts was presented to the Planning Commission.

A lead agency may not rely on an MND for project approval where substantial evidence supports a fair argument that the project may have a significant impact on the environment. (*Clews Land & Livestock, LLC v. City of San Diego* (2017) 19 Cal.App.5th 161, 183-184.) This standard sets a "low threshold" for preparation of an EIR, such that an EIR must be prepared if there is a "reasonable probability" that the project will result in a significant impact. (*Consolidated Irrig. Dist. v City of Selma* (2012) 204 Cal.App.4th 187, 207; *Sundstrom v County of Mendocino* (1988) 202 Cal.App.3d 296, 309, citing *No Oil, Inc. v. Los Angeles* (1974) 13 Cal.3d 68, 83, fn. 16.)

Here, there is a reasonable probability that the Project may result in PCP and/or dioxins migrating off the Project site into Hall Creek and/or the Mad River, which may result in significant environmental and human health impacts since the Mad River provides drinking water to 88,000 Humboldt residents. A review of the Planning Commission hearing demonstrates that several members of the Planning Commission were concerned about potential significant impacts related to contamination at the site but felt it was unfair to target and burden the Applicants with further environmental testing and analysis. While an EIR may be burdensome, it is clearly mandated when a project may cause significant environmental impacts. Therefore, the Planning Commission abused its discretion in approving the Project without requiring an EIR.

1. Background on PCP and dioxin contamination.

Timber and lumber industries have used PCP as a wood preservative to prevent decay and discoloration from fungal growth and insect damage since 1936.[1] Human populations exposed to PCP indicate potential neurobehavioral impacts, neuropsychological effects, respiratory diseases, and possibly birth defects. Studies on laboratory animals indicate liver toxicity, hormone disruptions,

[1] Exhibit A includes the sources providing the general background information on PCP use in the lumber industry and health risks associated with PCP and its contaminants, including dioxin. Excerpts of the larger documents have been provided along with the specific web address where the full document may be found and downloaded. By providing either full documents or the specific web address where the full document may be found, the District hereby submits the full documents as part of the administrative record. (See *Consolidated Irrigation Dist. v. Superior Court* (2012) 205 Cal.App.4th 697, 724-725 [concluding citation to the specific web page containing a document serves as "submitting" that document to the lead agency for purposes of Public Resources Code section 21167.6, subdivision (e)(7), meaning the document is part of the administrative record].)

potential to impact nervous system development, neurotoxicity, and other effects. The EPA characterizes PCP as "likely to be carcinogenic to humans" by all routes of exposure.[2]

Despite the severity of the potential impacts from PCP contamination, dioxin contamination is likely a greater concern. PCP historically used in the lumber industry was not a pure product, and included highly toxic byproducts, chlorinated dibenzodioxins and chlorinated dibenzofurans (collectively referred to as dioxins). The most toxic of these chemicals is commonly referred to as 2,3,7,8-TCDD. In 1985, the EPA ranked 2,3,7,8-TCDD as the most potent of 55 suspected carcinogens, at 50 million times more potent than trichloroethylene (TCE), a highly toxic solvent used in dry cleaning and degreasing operations.[3] In addition to being carcinogenic, dioxins can cause reproductive and developmental problems, heart disease, diabetes, damage to the immune system, and interfere with the endocrine system. Additionally, studies show dioxins may not only cause birth defects, but may cause reproductive problems and increases in adult onset disease in future generations that were not exposed to dioxins themselves.[4]

The risks related to dioxins are amplified by the fact that dioxins degrade much slower than other contaminants, such that they remain in the environment for a long period of time. Thus, they are classified as persistent organic pollutants. Further, some dioxins create more toxic chemicals as they break down. In short, their mere presence, even at extremely low concentrations, is a human health risk.

Due to the toxicity of PCP and its byproducts, the EPA banned PCP in 1984 for all but a limited number of industrial applications, such as production of utility poles.[5]

2. There is a fair argument that the Project site has been contaminated by PCP releases, and disturbing the soil and groundwater may cause contamination to migrate into the District's drinking water supply.

The Project site is located on land formerly owned by McNamara and Peepe that was used for timber processing for decades.[6] The timber processing activities included the use of PCP, which led to significant levels of contamination beneath and near the "green chain," which was a conveyor system where lumber was moved, sorted, and submersed in solutions containing PCP. Figure 2 in the Phase II shows that the former "green chain" lies approximately 700 feet to the west of the Project site. PCP

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[2] EPA, TOXICOLOGICAL REVIEW OF PENTACHLOROPHENOL (2010) p. 184, available at https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0086tr.pdf.

[3] State Water Resources Control Board, REPORT NO 88-5WQ: CHLORINATED DIBENZO-P-DIOXIN AND DIBENZOFURAN CONTAMINATION IN CALIFORNIA FROM CHLOROPHENOL WOOD PRESERVATIVE USE (1988) p. 39, available at https://www.hbmwd.com/files/76bbcf1de/DioxinContaminationFromPentaReportSWRCB1988.pdf; Agency for Toxic Substances & Disease Registry, TOXIC SUBSTANCES PORTAL - TRICHLOROETHYLENE (TCE), U.S. Department of Health and Human Services, https://www.atsdr.cdc.gov/phs/phs.asp?id=171&tid=30 (as of Sept. 18, 2019).

[4] Bryan Hamel, *Dioxin Exposure Causes Transgenerational Health Effects*, National Institute of Environmental Health Sciences (Nov. 2012), https://factor.niehs.nih.gov/2012/11/science-dioxin/index.htm.

[5] EPA, TOXICOLOGICAL REVIEW OF PENTACHLOROPHENOL (2010) p. 3, available at https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0086tr.pdf.

[6] The District has been monitoring the properties that comprised the McNamara and Peepe Lumber Mill site and related contamination since 1994 and is extremely concerned with the lack of progress in remediation.

was used in, and likely contaminated, other areas of the McNamara and Peepe property as well. PCP was sprayed on lumber in a planer building located approximately 250 feet from the Project site. (See Exhibit B [Second Quarter 2017 Groundwater Monitoring Report], p. 2-2 & Figure 3.) After being sprayed, the wet lumber would be sorted. A sorter building was located next to the planer building, and part of that building or structure appears to have extended onto the Project site. (See Exhibit C [Humboldt County Department of Environmental Health Diagram]; see also Exhibit B, Figure 3; Exhibit D [site photo]). During the time these buildings were in use, "several incidents of improper storage, spills, and leaks" of PCP were documented. (Exhibit B, p. 2-2.)

The MND suggests that the contaminated area near the green chain was remediated under DTSC oversight. In doing so, the MND improperly relies upon the 2003 Phase II and fails to address the fact that remedial measures have failed, such that PCP concentrations have skyrocketed above the maximum contaminant level (MCL) of 1 µg/L at numerous monitoring wells surrounding the former green chain. Grab groundwater samples in 2005 contained PCP and TCP concentrations as high as 16,000 µg/L and 1,500 µg/L, respectively. (Exhibit E [DTSC Decertification Letter, Dec. 28, 2018], p. 3.) DTSC explained that groundwater elevations rose approximately 15 feet since 2002 causing groundwater to come into contact with PCP- and TCP-impacted soil, which has resulted in "mobilizing hazardous substances from soil to groundwater." (*Ibid*.) During the most recent groundwater sampling event of monitoring wells surrounding the former green chain area for which data is available at the time of writing, PCP levels exceeded the MCL in 4 of 8 wells sampled, reaching as high as 570 µg/L, and the levels of PCP in each of those wells had increased since the prior sampling event in 2016. (Exhibit B, p. 4-1, 5-1.) Significantly, PCP levels increased and exceeded the MCL at MW-11—the monitoring well closest to the Project site. (Exhibit B, Figure 3 & Table 2.) **In December 2018, DTSC rescinded the prior Remedial Action Certification finding "soil and groundwater contamination at the Site is not under control and the implemented remedial actions are no longer protective of human health and the environment."** (Exhibit E, p. 1.)

Given DTSC's finding that groundwater contamination is no longer under control and remedial actions are no longer protective of human health and the environment, it is possible that the groundwater under the Project site is contaminated with PCP and has contaminated the soil at the Project site as well. Therefore, it is possible that contaminated groundwater and soil will be encountered during excavation. (As discussed below, the groundwater assessment performed in July, when groundwater levels are low, fails to provide an adequate picture of wet-weather groundwater levels at the site.)

The MND not only fails to recognize the risk of contamination spreading through groundwater, but it also fails to recognize the potential for hazardous releases that occurred onsite. As noted above, part of the sorting operations appear to have been located on the Project site. Figure 2 in the 2003 Phase II shows wood storage occurred on or about the Project site. This means after wood was treated with PCP, it was sorted and stored onsite, where it would have dripped PCP and dioxins into the soil during the drying process.

As the water provider for 88,000 residents of Humboldt County, the District is concerned that the Project may result in PCP and dioxins from contaminated groundwater and soil flowing into Hall Creek to the Mad River and, ultimately, into the District's downstream intake wells. At minimum,

the fact that the PCP plume is migrating and may have contaminated the Project site constitutes substantial evidence to support a fair argument that the Project may result in a significant environmental impact.

B. The DTSC letter and Groundwater Assessment are inadequate to overcome substantial evidence in the record of a fair argument of significant environmental impacts.

As discussed above, the District provided substantial evidence of potential environmental impacts. The Applicants submitted a "comfort letter" from DTSC and a groundwater assessment to further their argument that the Project will not have significant environmental impacts.[7] However, as explained below, these do not overcome the County's obligation to require preparation of an EIR. Moreover, as outlined below, those documents are faulty and incomplete.

1. When the record includes evidence supporting fair argument of a significant environmental impact, it is irrelevant whether the record also contains substantial evidence of no impact.

The fact that the Applicants provided evidence that there may not be environmental impacts does not overcome the obligation to prepare an EIR. "[T]he agency must prepare an EIR whenever substantial evidence in the record supports a fair argument that a proposed project may have a significant effect on the environment. [Citations.] 'If such evidence is found, it cannot be overcome by substantial evidence to the contrary.'" (*Inyo Citizens for Better Planning v. Inyo County Bd. of Supervisors* (2009) 180 Cal.App.4th 1, 7.) Thus, it is inconsequential that the Applicants have provided evidence supporting a finding that the Project may not result in an environmental impact because there is a fair argument that the Project will result in significant impacts to the environment and human health. Further, as discussed in greater detail below, the evidence relied on by the Applicants is either faulty or incomplete.

2. The Planning Commission relied on faulty and incomplete evidence in approving the Project.

The DTSC letter relies on the 2003 Phase II in concluding: "based on our review of the submitted documents, DTSC concludes the Property is not impacted by PCP-contaminated soil or groundwater." However, the 2003 Phase II does not encompass the Project site, APN 516-111-064. (2003 Phase II, p. 1 ["The scope of work includes the review and sampling of parcels Assessor Parcel Number (APN) 516-101-006, -017, -040, -041, -060, -064, -068, 516-111-004, -005, -006, 015, -033, & 516-151-019. It also includes the properties owned by Charles Aalfs APN 516-101-002 -059 & -063." Absent from this list is the Project site, APN 516-111-064].) Because the 2003 Phase II does not encompass the Project site, it does not speak to conditions there.

[7] The DTSC comfort letter and groundwater assessment were not made available to the public until the Staff Report for the Planning Commission hearing was released. Thus, the District was limited in its ability to respond to them prior to and during the hearing.

Further, reliance on the 2003 Phase II was an egregious abuse of discretion because it is severely outdated. Like a Phase I environmental site assessment, a Phase II investigation is a due diligence document that a prospective property purchaser may rely upon in determining site conditions at the time of the investigation and as providing statutory protection against liability. As noted in the 2003 Phase II, its purpose was "to expedite the sale of these parcels." (2003 Phase II, p. 1.) Because site conditions change over time, Phase I site assessments cannot be relied on without a comprehensive update after 6 months and become invalid after one year. (See ASTM E1527-13, § 4.6, Continued Viability of Environmental Site Assessment.) If the 2003 Phase II followed a Phase I site assessment pursuant to ASTM Practice E1527 or Practice E2247, its expiration would mirror that of the Phase I—i.e., no longer than 6 months if not updated, and no longer than 1 year at maximum. (ASTM E 1903-11, § 4.2.4, Data Usability.) If the 2003 Phase II did not follow a Phase I, its expiration is not strictly delineated by the ASTM, but Phase IIs likewise become outdated over time—because conditions change, sometimes significantly. If DTSC were seeking to identify potentially responsible parties (PRPs) to fund a remediation action, it would never accept a 16-year-old Phase II from a PRP as a shield from liability.

In the area surrounding the Project site, conditions have changed significantly. The 2003 Phase II noted there was contamination at the McNamara and Peepe site, but relied on a 2002 comprehensive review that concluded remedial actions were successfully preventing PCP contamination from leaching into the groundwater. (2003 Phase II, pp. 4-5.) However, as discussed above and as all parties know, in 2018, DTSC rescinded the prior Remedial Action Certification finding "soil and groundwater contamination at the Site is not under control and the implemented remedial actions are no longer protective of human health and the environment." (Exhibit E, p. 1.) There is no question that this reflects changed conditions that could not possibly have been captured in the 16-year-old 2003 Phase II. In the same vein, the 2003 Phase II could not have predicted the potential offsite migration of contaminated groundwater from the McNamara and Peepe site to the Project site.

Finally, DTSC's letter in no way guarantees the Project site is not contaminated, and it is not clear that DTSC actually intended to convey that the Project site is uncontaminated. Our office contacted DTSC to inquire whether new groundwater and soil sample data was available for the McNamara and Peepe site and asked how DTSC found the 2003 Phase II data reliable for concluding there is no contamination at the Project site. We were told that DTSC's intention was to provide an ambivalent response because there is no *current* data with respect to the Project site, and DTSC had neither the authority nor intention to require sampling at the Project site. However, we understand the County spoke to DTSC and understood the letter as being unambivalent. Accordingly, we request clarification be sought from DTSC as to whether DTSC concludes the site is unambivalently uncontaminated, or whether the 16-year-old data for the site gives no reason to find the site is contaminated and that DTSC is simply unwilling to comment further.

As discussed above, groundwater rose 15 feet following 2002, coming into contact with PCP contaminated soil, resulting in the migration of these contaminants. Such contamination has been detected above the MCL at a monitoring well near the Project site. This leads to the following concerns with respect to the project site, discussed in more detail below: (a) the Project may encounter contaminated groundwater during construction; (b) installation of the water and sewer lines may independently expedite migration of the contaminant plume; (c) even if construction does not

encounter contaminated groundwater, it may encounter contaminated soil; and (d) no party or agency has undertaken testing to determine the risk posed by dioxins at the Project site.

a. The groundwater assessment fails to reflect seasonal variations in groundwater. Depending on the time of year construction occurs, groundwater may be encountered.

Applicants provided a groundwater assessment, performed July 31, 2019, in claiming groundwater is no higher than 7.86 feet below ground surface at the Project site. However, this does not guarantee groundwater will not be encountered during construction. Groundwater levels fluctuate significantly between different years and seasons. For instance, six years of data from the adjacent site show how variable groundwater levels can be—groundwater levels commonly fluctuated by over 4 feet between May and November, once by over 5 feet. (Exhibit B, Table 1.) And this data does not account for the fact that groundwater levels generally are lower still in the summer (e.g., July) than the spring (e.g., May). The Applicant's groundwater assessment occurred on July 31, a point when groundwater levels would be at or near their lowest point. Depending on when construction occurs, groundwater levels may be significantly higher than 7.86 feet below ground surface. If groundwater levels rise by 3.86 feet, trenching will encounter groundwater. Table 1 also shows a number of sampling events where groundwater was encountered at 4 feet or fewer below ground surface. Thus, there is potential for the Project to encounter groundwater during construction. And as discussed above, this could result in contaminated groundwater running offsite into Hall Creek and/or the Mad River and contaminating the District's water supply.

b. Water and sewer lines have potential to act as preferential pathways that expedite the future migration of contaminated groundwater.

Independent of whether groundwater is encountered during construction, the Board should be aware that the act of installing water and sewer lines in contaminated conditions provides a preferential pathway for contamination, expediting plume migration. Thus, if and when contaminated groundwater levels rise and meet the installed water and sewer lines, contamination will spread faster along the length of those lines than in soil alone.

c. Site development creates the possibility of contaminated stormwater runoff draining into Hall Creek and/or the Mad River and contaminating the District's drinking water supplies.

The MND and Planning Commission failed to address the fact that the soils at the Project site may be contaminated, and this gives rise to issues unrelated to groundwater. There are two routes by which the soil at the site may have become contaminated, which were ignored by the MND and Planning Commission. First, contaminants were likely released directly upon the Project site. As noted above, it was the location for lumber sorting and storage following PCP treatment. Thus, while drying, such lumber would have dripped excess PCP directly into the soil. Additionally, the soil may be contaminated due to contaminated groundwater. Contaminants from the McNamara and Peepe site have spread through groundwater, but some absorbs into the soil. Thus, these contaminants do not fully recede with falling groundwater levels; some remain, impacting the soil.

Presently, the site is paved over. The MND attempts to argue that stormwater runoff issues will be improved because the paved surface increases the amount of stormwater runoff. Under normal circumstances this would be true since the Project will result in *less* stormwater runoff. However, because the MND fails to account for potentially contaminated soils at the site, it misses the mark here. Presently, the paved surface blocks stormwater from coming into contact with potentially contaminated soils, such that it cannot carry potential contamination along with it. If the Project is completed, it will reduce the amount of runoff, but runoff will still occur when the ground at the site is saturated and stormwater capture basins flood during heaving rainfall. And, due to the changed conditions at the site, future stormwater will first interact with the potentially contaminated soils, and thus may carry contaminants offsite to Hall Creek and/or the Mad River, the source of water for 88,000 County residents and habitat for ESA listed aquatic species. Further, as discussed and admitted during the Planning Commission hearing, it is unknown whether the stormwater filtration system has the ability to filter out toxic compounds such as PCP and dioxins. This remains a potentially significant environmental and human health impact.

d. The 2003 Phase II, MND, and DTSC letter fail to address the potential risks of dioxin contamination at the site.

Last, the 2003 Phase II, MND, and DTSC letter do not address risks related to potential for dioxin contamination at the site because it has never been tested for. Therefore, no one knows the potential scope and concentration of dioxin contamination. This likely contamination cannot be ignored given dioxins persist longer than PCP and are more toxic by orders of magnitude. In light of the Project site's historical function and the plume migration from the McNamara and Peepe site, the Project is potentially contaminated with dioxins. Due to their extreme toxicity, this amounts to a potentially significant environmental impact. Thus, approval of the Project with an MND was an abuse of discretion.

II. The County failed to comply with CEQA's notice requirements.

In letters submitted to the County on May 29, 2019 and August 28, 2019, the District raised concerns related to the County's failure to properly notice its intent to adopt the MND pursuant to CEQA sections 21092(b)(3) and 21092.2(a) as well as Guidelines sections 15072(b) & 15073. The District continues to assert, and does not waive, the issue of improper notice raised in those comment letters.

III. The Project site should be treated as if it were located in a designated Critical Municipal Water Supply Area.

The District is in the process of applying to have the Mad River Watershed designated as a Critical Municipal Water Supply Area under the Humboldt County General Plan due to the potential for cumulative impacts from land uses within the area to significantly impact the quality of the District's water supplies. The purpose of designating the Mad River Watershed as a Critical Municipal Water Supply Area is to protect the safety of the County's drinking water. Designation furthers this purpose by requiring development activities to mitigate significant adverse effects. Due to the importance of the Mad River Watershed in providing drinking water supplies, it is in the County's best interest to

ensure potential significant adverse effects are fully mitigated as if the watershed were already designated a Critical Municipal Water Supply Area. The next section identifies and recommends conditions of approval to protect the District's/County's drinking water supplies.

IV. The District has identified conditions of approval that it has determined are necessary to mitigate the impacts of the Project in the absence of an EIR.

As discussed above, there is a fair argument that the Project may have a significant impact on the environment and human health. Because the MND does not account for this significant impact, never mind mitigate its effects, an EIR is required. However, the District proposes two additional conditions of approval, outlined below, which, if adopted, would adequately mitigate the District's concerns.

1. The Applicants shall have an experienced and qualified professional sample groundwater on the Project site for the presence of PCP and dioxins. The sample shall be taken no closer than 100 feet from monitoring well MW-11 for the McNamara and Peepe site. Due to the extreme toxicity and low MCL for dioxins, a detection limit of 0.5 picograms/liter shall be used for dioxins and/or dioxin TEQ, using the 2005 WHO approach as described in a DTSC 2013 Technical Memorandum.[8] If the contaminant concentrations exceed their respective MCLs, the Applicants must submit a remediation plan that is accepted by the County and subsequently implement the remediation plan as part of the Project. If remediation is required, the County shall seek to coordinate with DTSC's remediation of the McNamara and Peepe site.

2. Prior to the issuance of any permits, the Applicants must conduct comprehensive soil sampling by an experienced and qualified professional for PCP and dioxins. Due to the extreme toxicity and low MCL for dioxins, a detection limit of 0.5 picograms/liter shall be used for dioxins and/or dioxin TEQ, using the 2005 WHO approach as described in the DTSC 2013 Technical Memorandum.[9] If the contaminant concentrations exceed their respective MCLs, the Applicants must submit a remediation plan that is accepted by the County and subsequently implement the remediation plan as part of the Project. If remediation is required, the County shall seek to coordinate with DTSC's remediation of the McNamara and Peepe site. It should be noted Planning Director John Ford suggested a similar condition of approval at the Planning Commission hearing.

3. The Applicants shall have an experienced and qualified professional sample stormwater runoff for PCP and dioxins. Due to the extreme toxicity and low MCL for dioxins, a detection limit of 0.5 picograms/liter shall be used for dioxins and/or dioxin TEQ, using the 2005 WHO approach as described in the DTSC 2013 Technical Memorandum.[10] If the contaminant concentrations exceed their respective MCLs, the Applicants must submit a remediation plan that is accepted by the County and subsequently implement the remediation plan as part of the Project. If remediation is required, the County shall seek to coordinate with DTSC's remediation of the McNamara and Peepe site.

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[8] DTSC, TECHNICAL MEMORANDUM: RATIONALE AND SUMMARY FOR USING WORLD HEALTH ORGANIZATION TOXICITY EQUIVALENCY APPROACH, SANTA SUSANA FIELD LABORATORY, VENTURA COUNTY, CALIFORNIA (May 21, 2013), available at https://www.dtsc-ssfl.com/files/lib_cbs/correspondence/66072_TEQ_White_Paper.pdf.

[9] *Ibid*.

[10] *Ibid*.

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In sum, reliance on an MND to approve the Project is improper on procedural and substantive grounds. There is substantial evidence to support a fair argument that the Project may have significant environmental impacts related to contamination groundwater and soils on the Project site. Under these circumstances, CEQA requires an EIR to adequately analyze these impacts and provide mitigation to prevent any potential contamination of the District's drinking water supplies. To the extent the County refuses to require an EIR, the District has identified conditions of approval that are necessary to determine whether the site is contaminated and ensure the safety of the drinking water that over 80,000 Humboldt residents rely upon.

Respectfully,

Anne Baptiste

cc: Humboldt Bay Municipal Water District
California Department of Toxic Substances Control
California Department of Fish and Wildlife
Humboldt Baykeeper
North Coast Regional Water Quality Control Board

Exhibit A

EPA, TOXICOLOGICAL REVIEW OF PENTACHLOROPHENOL (2010), available at https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0086tr.pdf

EPA/635/R-09/004F
www.epa.gov/iris

TOXICOLOGICAL REVIEW

OF

PENTACHLOROPHENOL

(CAS No. 87-86-5)

In Support of Summary Information on the Integrated Risk Information System (IRIS)

September 2010

U.S. Environmental Protection Agency
Washington, DC

2. CHEMICAL AND PHYSICAL INFORMATION

PCP (CASRN 87-86-5) is a chlorinated aromatic compound that appears in a solid crystalline state and ranges in color from colorless to white, tan, or brown. The chemical, also referred to as penta, pentachlorofenol, 2,3,4,5,6-PCP, and chlorophen, has a phenolic odor that is pungent when heated. PCP is nonflammable and noncorrosive, and, although solubility is limited in water, it is readily soluble in alcohol (Budavari et al., 1996; NTP, 1989). The physical/chemical properties of PCP are summarized in Table 2-1.

Table 2-1. Chemical and physical properties of PCP

Chemical formula	C_6HOCl_5
Molecular weight	266.34
Melting point	190–191°C
Boiling point	~309–310°C
Density	1.978 g/mL (at 22°C/4°C)
Vapor density	9.20 (air = 1)
Vapor pressure	0.00011 (at 20°C)
Log K_{ow}	5.01
Log K_{oc}	4.5
Water solubility	80 mg/L (at 20°C), 14 mg/L (at 26.7°C)
Henry's law constant	2.45×10^{-8} (atm × m^3)/mole
Conversion factors	1 ppm = 10.9 mg/m^3; 1 mg/m^3 = 0.09 ppm; 1 ppm = 0.01088 mg/L; 1 mg/L = 99.1 ppm (at 25°C)

Sources: NLM (1999a, b); Budavari et al. (1996); Allan (1994); Royal Society of Chemistry (1991).

PCP has been used as a wood preservative to prevent decay from fungal organisms and insect damage since 1936. The first pesticidal product containing PCP as an active ingredient was registered in the United States in 1950 (U.S. EPA, 2008; Ahlborg and Thunberg, 1980). Historically, PCP was widely used as a biocide and could also be found in ropes, paints, adhesives, canvas, leather, insulation, and brick walls (U.S. EPA, 2008; Proudfoot, 2003; ATSDR, 2001). Indoor applications of PCP were prohibited in 1984; PCP application was limited to industrial areas (e.g., utility poles, cross arms, railroad cross ties, wooden pilings, fence posts, and lumber/timbers for construction). Currently, products containing PCP remain registered for heavy duty wood preservation, predominantly to treat utility poles and cross arms. Pentachlorophenol is a restricted use pesticide available to certified applicators only (U.S. EPA, 2008).

PCP is produced via two pathways, either "by stepwise chlorination of phenols in the presence of catalysts (anhydrous aluminum chloride or ferric chloride) or alkaline hydrolysis of [hexachlorobenzene] HCB" (Proudfoot, 2003). In addition to industrial production of PCP, the

degradation or metabolism of HCB (Rizzardini and Smith, 1982), pentachlorobenzene (Kohli et al., 1976), or pentachloronitrobenzene (Renner and Hopfer, 1990) also yields PCP. Impurities found in PCP are created during the production of the chemical. Technical-grade PCP (tPCP), frequently found under the trade names Dowicide 7, Dowicide EC-7 (EC-7), Dow PCP DP-2 Antimicrobial (DP-2), Duratox, Fungol, Penta-Kil, and Permacide, is composed of approximately 90% PCP and 10% contaminants. The impurities consist of several chlorophenol congeners, chlorinated dibenzo-p-dioxins, and chlorinated dibenzofurans. Of the chlorinated dibenzo-p-dioxin and dibenzofuran contaminants, the higher chlorinated congeners are predominantly found as impurities within tPCP. In addition to the chlorinated dibenzo-p-dioxin and dibenzofuran contaminants, HCB and chlorophenoxy constituents may also be present in tPCP. Use of the analytical grade of PCP (aPCP) first requires a purification process to remove the contaminants that were created during the manufacturing of PCP. The physicochemical properties of these contaminants are listed in Appendix B in Tables B-1 and B-2.

Grades described as analytical or pure are generally ≥98% PCP and the levels of dioxins and furans are low to nondetectable. Purities of technical- and commercial-grade PCP formulations are reported to be somewhat less than the analytical formulations, ranging from 85 to 91%. Hughes et al. (1985) reported that tPCP contains 85–90% PCP, 10–15% trichlorophenol and tetrachlorophenol (TCP), and <1% chlorinated dibenzo-p-dioxins, chlorinated dibenzofurans, and chlorinated diphenyl ethers. The compositions of different grades of PCP, as reported by the National Toxicology Program (NTP) (and similar to values reported in the general literature), are listed in Table 2-2.

6. MAJOR CONCLUSIONS IN THE CHARACTERIZATION OF HAZARD AND DOSE RESPONSE

6.1. HUMAN HAZARD POTENTIAL

6.1.1. Noncancer

PCP is a nonflammable, noncorrosive chemical that was first registered in the United States in 1936 as a wood preservative to prevent decay from fungal organisms and insect damage. It was widely used as a biocide and could also be found in ropes, paints, adhesives, canvas, insulation, and brick walls. After use was restricted in 1984, PCP applications were limited to utilization in industrial areas, including utility poles, cross arms, railroad cross-ties, wooden pilings, fence posts, and lumber/timbers for construction. Currently, products containing PCP remain registered for wood preservation, and utility poles and cross arms represent approximately 92% of all uses for PCP-treated lumber.

During manufacture of PCP, the chemical is contaminated with impurities that consist of several congeners of the chlorophenols, chlorinated dibenzo-p-dioxins, and chlorinated dibenzofurans. Of the chlorinated dibenzo-p-dioxin and dibenzofuran contaminants, the higher chlorinated congeners are predominantly found as impurities within tPCP (approximately 90% purity). Use of the aPCP first requires a purification process to remove the contaminants that are simultaneously created during the manufacturing of PCP.

Instances of PCP poisoning have been documented, indicating the potentially severe consequences of acute, high-dose exposures. Few studies have examined the effects of the lower exposures that occurred in occupational settings or through residential or environmental sources. Many of the available studies are relatively small (<50 participants) (Peper et al., 1999; Triebig et al., 1987; Klemmer et al., 1980; Begley et al., 1977) or may not be representative of the exposed population (Gerhard et al., 1999; Walls et al., 1998). Despite these limitations, there are indications of specific types of neurobehavioral effects seen with chronic exposure to PCP in nonoccupational settings (Peper et al., 1999). A larger study of 293 former sawmill workers in New Zealand also suggests neuropsychological effects and respiratory diseases (McLean et al., 2009b). In addition, the results from a large nested cohort study of reproductive outcomes in offspring of sawmill workers (Dimich-Ward et al., 1996) indicate that specific types of birth defects warrant additional research.

The toxicity of PCP in orally exposed animals was investigated in numerous studies in experimental animals. These studies indicate that PCP is toxic to the liver. In chronic studies in rats and dogs, liver toxicity was characterized primarily by increased incidence of chronic inflammation, cytoplasmic vacuolization, pigmentation, and hepatocellular necrosis as well as changes in liver weight (NTP, 1999; Mecler, 1996; Schwetz et al., 1978). Liver toxicity in mice was exhibited as necrosis, cytomegaly, chronic active inflammation, pigmentation, and bile duct

lesions (NTP, 1989). The increased severity of liver toxicity observed in mice versus rats could be based, in part, on differences in biotransformation of PCP (Lin et al., 1997), but it is also noted that in the mouse studies, the PCP test material contained higher concentrations of chlorinated dibenzo-p-dioxin or dibenzofuran contaminants, which could contribute to the severity of the liver response. Liver toxicity in the dog (Mecler, 1996) was similar to that of the mouse, but the doses inducing toxicity were lower than those in the mouse (i.e., 1.5 mg/kg-day in the dog versus 17–18 mg/kg-day in the mouse). Studies using domestic or farm animals showed that pigs, but not cattle, exhibited similar liver toxicity as that observed in mice. Pigment deposition was also observed in the proximal convoluted tubules in the kidneys of rats (NTP, 1999). Developmental toxicity studies (Welsh et al., 1987; Schwetz et al., 1974a) indicated toxic effects in offspring at dose levels below those producing maternal toxicity. Studies in mink indicate some reproductive effects following exposure to PCP (Cook et al., 1997). The spleen weights of mice (NTP, 1989), rats (Bernard et al., 2002), and cattle (Hughes et al., 1985) were decreased following exposure to PCP.

Disruption of thyroid homeostasis has been observed following the administration of PCP. Several studies have reported decreased serum T_4 and T_3 levels in rats (Jekat et al., 1994) and cattle (Hughes et al., 1985; McConnell et al., 1980). Decreases in serum T_4 have been observed in ram and ewe lambs (Beard et al., 1999a, b), mature ewes (Rawlings et al., 1998), and mink (Beard and Rawlings, 1998) after administration of PCP. TSH was unaffected by treatment with 1 mg/kg-day PCP in calves (Hughes et al., 1985) and sheep (Beard et al., 1999b). However, Jekat et al. (1994) reported a decrease in TSH accompanying the decrease in T_4 levels in rats administered 3 mg/kg-day tPCP and aPCP. Considering that TSH acts on the thyroid to control production of T_4, the concurrent decrease in TSH is in contrast to the expected TSH response to a decrease in T_4 (TSH is generally expected to increase in response to a decrease in T_4), which led Jekat et al. (1994) to suggest that this was due to interference with thyroid hormone regulation at the hypothalamic/pituitary level and possibly increased peripheral thyroid hormone metabolism. However, the available data do not allow for determination of the mechanism involved in the effects on T_3, T_4, and TSH following exposure to PCP. The effect of PCP on thyroid hormone homeostasis has been attributed to PCP and not to contaminants. Changes in thyroid hormones have been associated with effects (i.e., delayed myelination, neuronal proliferation, and synapse formation) on neurons. Considering that thyroid hormones may play a role in neurodevelopmental processes, the disruption of thyroid homeostasis that has been observed with PCP indicates a potential concern for critical period of development of the nervous system (CalEPA, 2006). However, the downstream effects associated with PCP and decreased T_4 levels have not been explored.

Studies examining the immunotoxic effects of PCP showed that the humoral response and complement activity in mice were impaired by tPCP, but not by aPCP, when administered to adult animals (NTP, 1989; Holsapple et al., 1987; Kerkvliet et al., 1985a, b; 1982a). However,

treatment of mice with aPCP from the time of conception to 13 weeks of age resulted in impaired humoral and cell-mediated immunity (Exon and Koller, 1983), suggesting that PCP, and not just the contaminants, induce immunotoxicity. Human studies showed that immune response was impaired in patients who had blood PCP levels >10 μg/L and in particular in those whose levels were >20 μg/L (Daniel et al., 1995; McConnachie and Zahalsky, 1991). Based on the limited available information, immunotoxic effects of PCP may be elicited, in part, through the presence of the dioxin/furan contaminants within PCP.

In vitro neurotoxicity studies showed that PCP causes a dose-dependent irreversible reduction in endplate potential at the neuromuscular junction and interferes with axonal conduction in the sciatic nerve from the toad (Montoya and Quevedo, 1990; Montoya et al., 1988). An NTP (1989) study in mice showed only decreased motor activity in rotarod performance in male rats treated with tPCP for 5 weeks and increases in motor activity and startle response in females receiving purified and tPCP for 26 weeks. Another in vivo study showed that treatment of rats with PCP for up to 14 weeks caused biochemical changes in the rat brain (Savolainen and Pekari, 1979). The most definitive study showed that rats receiving PCP in drinking water for at least 90 days had marked morphological changes in sciatic nerves (Villena et al., 1992).

Elevated blood sugar levels (considered minor by Demidenko, 1969) and increases in organ weights were observed in rats and rabbits exposed to 21–29 mg/m^3 PCP by inhalation for 4 months (Ning et al., 1984; Demidenko, 1969). Additional effects included anemia, leukocytosis, eosinophilia, hyperglycemia, and dystrophic processes in the liver. Minor effects were noted on the liver, cholinesterase activity, and blood sugar effects of animals exposed to 2.97 mg/m^3 (calculated as 0.3 mg/kg-day PCP by Kunde and Böhme [1978]), a dose that is lower than the lowest NOAELs (1 mg/kg-day) observed in animals orally exposed to 28.9 mg/m^3 PCP (Demidenko, 1969). Ning et al. (1984) reported significant increases in organ weights (lung, liver, kidney, and adrenal glands), serum γ-globulin, and blood-glucose levels at 21.4 mg/m^3.

Studies examining the mutagenicity of PCP have shown that in a variety of test systems, PCP is nonmutagenic, with the exception of one study (Gopalaswamy and Nair, 1992) in which PCP exhibited a positive response for mutagenicity in the Ames Salmonella assay. In contrast to data on PCP, data for the TCHQ metabolite of PCP show positive mutagenic effects in CHO cells (Jansson and Jansson, 1991; Carstens et al., 1990; Ehrlich, 1990), an increase in micronuclei using V79 cells (Jansson and Jansson, 1992), covalent binding to DNA (Witte et al., 2000, 1985), and induction of DNA SSBs (Witte et al., 1985).

6.1.2. Cancer

The available epidemiologic studies support an association between PCP exposure and development of specific cancers: non-Hodgkin's lymphoma, multiple myeloma, soft tissue sarcoma, and liver cancer (limited evidence). These studies used PCP-specific exposure

assessment and in some cases, additional assessment of other chlorophenols and potential contaminants. PCP preparations are produced with methods that allow for the formation of contaminants, and degradation products occur naturally in most formulations. However, these contaminants are unlikely to spuriously produce the observed associations seen in the epidemiologic studies, given the difference in the patterns of cancer risk seen in studies of dioxins compared with the studies of PCP, and the relative strengths of the effects of different chemicals (PCP, other chlorophenols, dioxins, and furans) in the studies that examined more than one of these chemicals. It should be noted that in the epidemiological studies examining the cancer risk associated with exposure to PCP, exposures occurred predominantly via the inhalation and dermal routes.

Animal studies with PCP show evidence of adrenal medullary and hepatocellular tumors in male and female mice, hemangiosarcomas and hemangiomas in female mice, and nasal squamous cell carcinomas and mesotheliomas in male rats. Two well-conducted studies provide data for the carcinogenicity of PCP via the oral route in laboratory animals: one study in $B6C3F_1$ mice (NTP, 1989) and another study in F344 rats (NTP, 1999). Two formulations of PCP (tPCP and EC-7) were carcinogenic in the mouse. Hepatocellular adenomas/carcinomas and adrenal medullary pheochromocytomas developed in male mice treated with tPCP or EC-7, and hepatocellular adenomas/carcinomas and hemangiosarcomas developed in female mice treated with tPCP or EC-7 and adrenal medullary pheochromocytomas developed in female mice treated with EC-7.

Under the U.S. EPA's *Guidelines for Carcinogen Risk Assessment* (2005a), PCP is characterized as "likely to be carcinogenic to humans" by all routes of exposure.

6.2. DOSE RESPONSE

6.2.1. Noncancer—Oral Exposure

The most sensitive endpoints identified for effects of PCP by oral exposure relate to liver toxicity in the chronic gelatin capsule study Mecler (1996) in beagle dogs. Mecler (1996) was selected for the derivation of the oral RfD. This study was conducted in accordance with good laboratory practice guidelines valid at that time and included both sexes of beagle dogs, four animals per sex and dose group, and three dose groups plus controls (0, 1.5, 3.5, and 6.5 mg/kg-day). The study reported multiple toxic endpoints, including changes in absolute and relative organ weights, changes in hematological parameters, and histopathologic outcomes. Hepatotoxicity characterized by dose-related increases in incidence and severity of hepatic lesions (including liver pigmentation, cytoplasmic vacuolation, chronic inflammation, and the appearance of dark, discolored livers) accompanied by significant increases in absolute (in females only) and relative liver weight, and serum activity of ALT and ALP in dogs was considered the critical effect. Another target of PCP toxicity following oral exposure considered in the selection of the critical effect was the developing organism. Studies in experimental

State Water Resources Control Board, REPORT NO 88-5WQ: CHLORINATED DIBENZO-P-DIOXIN AND DIBENZOFURAN CONTAMINATION IN CALIFORNIA FROM CHLOROPHENOL WOOD PRESERVATIVE USE (1988), available at https://www.hbmwd.com/files/76bbcf1de/DioxinContaminationFromPentaReportSWRCB1988.pdf.

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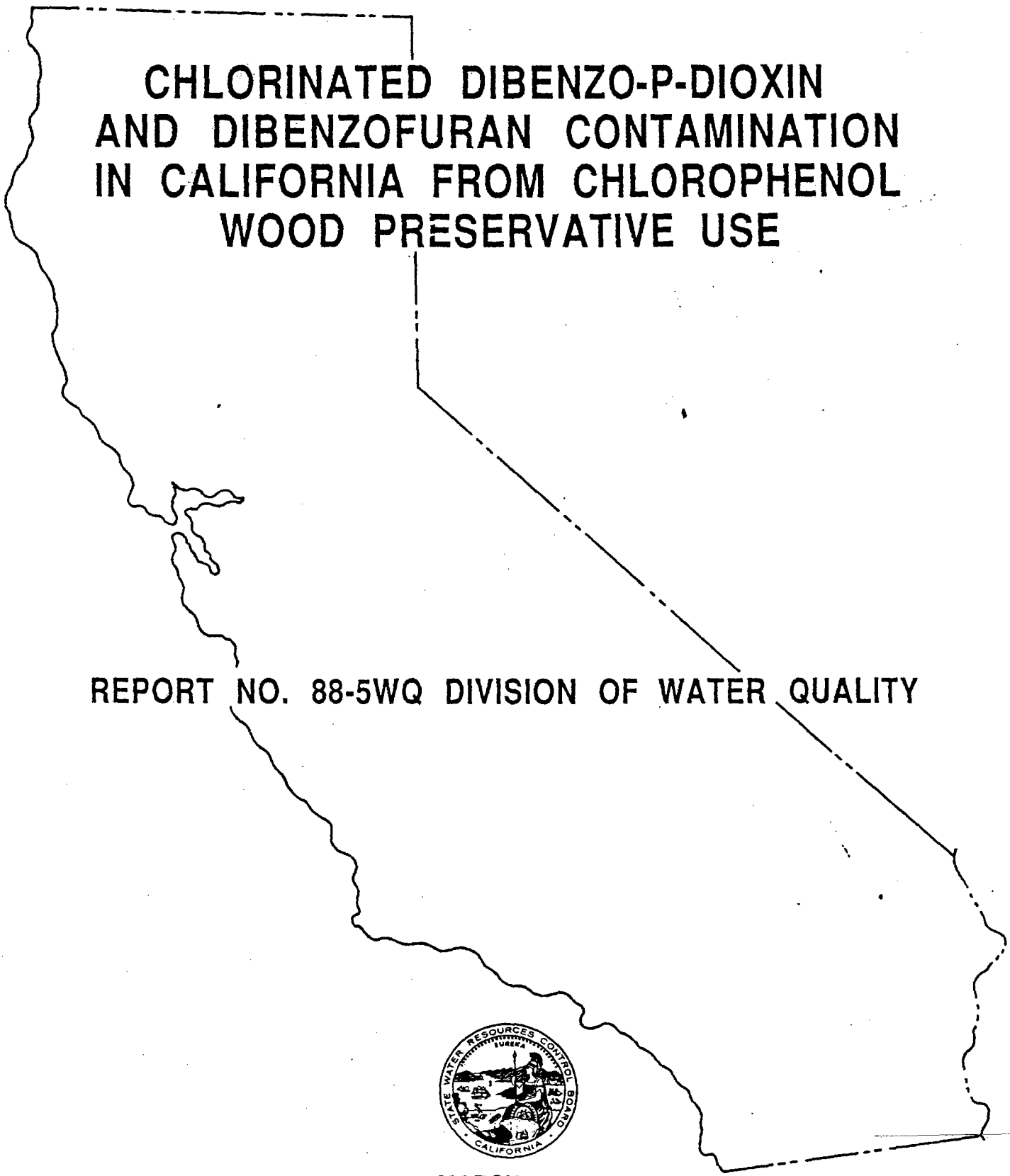
CHLORINATED DIBENZO-P-DIOXIN AND DIBENZOFURAN CONTAMINATION IN CALIFORNIA FROM CHLOROPHENOL WOOD PRESERVATIVE USE



REPORT NO. 88-5WQ DIVISION OF WATER QUALITY

MARCH 1988

STATE WATER RESOURCES CONTROL BOARD

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CHLORINATED DIBENZO-P-DIOXIN AND DIBENZOFURAN CONTAMINATION IN CALIFORNIA FROM CHLOROPHENOL WOOD PRESERVATIVE USE

REPORT NO. 88-5WQ DIVISION OF WATER QUALITY

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MARCH 1988
STATE WATER RESOURCES CONTROL BOARD

PREFACE

This report is one in a series of reports issued by the State Water Resources Control Board on industrial and agricultural chemicals. These reports deal with priority chemicals of concern to water quality and the protection of beneficial uses of water in California. In February 1982, the State Board initiated an Industrial Chemicals program based on the premise that the production and use of chemicals should not occur at the expense of water quality protection.

Chemicals are of inestimable value to society, and most are considered relatively safe under normal conditions of use. There are some chemicals whose environmental and health effects have been proven harmful. The possibility that toxic chemicals in the environment can cause cancer in humans and severely impair the health of wildlife has led to increased action by government to foster the safe use and disposal of these chemicals.

The chronic effects of persistent chemicals (e.g., impaired growth and reproduction) may be more devastating in the long run than immediately apparent effects, such as fish kills. Preventative measures are invariably less costly to society than corrective actions required after toxic chemical pollution has occurred.

Some current chemical use and disposal practices may have an adverse impact on water quality. These activities can usually be modified to minimize adverse environmental effects. Where existing or potential water quality problems have been identified, the State Board will recommend appropriate measures to correct or prevent such adverse impacts.

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EXECUTIVE SUMMARY

1. INTRODUCTION

In 1984, the State Board began a priority chemical investigation of certain chemicals used for wood preservation at California sawmills and wood treatment plants. Pentachlorophenol, one of the most widely used wood preservative fungicides, was given special attention, as it is known to contain highly toxic byproducts produced during its chemical manufacture. These contaminants include chlorinated dibenzodioxins (CDDs) and a related group of chemicals, chlorinated dibenzofurans (CDFs). Chemical identification of these substances is extremely difficult, in part because there are so many of them (75 different CDDs and 135 possible CDFs). Only 15 of these 210 compounds (6 CDDs and 9 CDFs) are considered highly toxic. The most toxic compound is commonly referred to as "dioxin" or 2,3,7,8-tetrachlorodibenzodioxin. As "dioxin" has been studied most extensively, much of what has been estimated about the other CDDs and CDFs is based on knowledge of this compound.

The CDDs and CDFs have never been intentionally manufactured. They are only produced as reference standards which are required for chemical analysis. In addition, CDDs and CDFs are known to occur as byproducts of chemical synthesis, from electrical equipment fires, and from municipal solid waste incinerators. The CDDs and CDFs have received widespread media attention because of several incidents involving human exposures. These events include the use of the herbicide Agent Orange in Vietnam, a chemical plant explosion at Seveso, Italy, CDD-contaminated oil used for dust control in Missouri, and CDF-contaminated rice oil poisoning incidents in Japan and Taiwan.

The State Board study described in this report was designed to determine which, if any, of the 15 most toxic CDDs and CDFs were present at sawmills and wood treatment plants in California. In order to perform the difficult chemical analysis, split samples were sent to three laboratories in the United States and Sweden. Several of the 15 most toxic CDDs and CDFs were detected in samples of soil, sawmill sludges and liquids, commercial pentachlorophenol formulations, and crystals formed during wood pressure treatment.

2. ENVIRONMENTAL FATE

As a group, the CDDs and CDFs share three characteristics that make them long-lived in the environment: very low water solubility, high affinity for soil and sediment and resistance to breakdown. However, as individual compounds, the CDDs and CDFs exhibit wide diversity. For example, the eight chlorine CDD is about 100,000 times less soluble than the CDDs containing four chlorine atoms. The combination of very high toxicity and very low water solubility has made the measurement and modeling of CDDs and CDFs in the environment a difficult task. However, recent work has shed some light on a number of processes that may affect the persistence of these compounds in the environment. These include the following:

a. On soil surfaces, CDDs and CDFs can be both formed and broken down by sunlight. For example, they can be formed from the joining of two pentachlorophenol molecules, while more highly chlorinated compounds can be converted to lower chlorinated ones. Under certain conditions, the lower chlorinated CDDs and CDFs that are formed from such breakdown conversions can be more toxic than more highly chlorinated parent compounds.

b. Naturally occurring micro-organisms will not significantly breakdown CDDs and CDFs.

c. Despite having low vapor pressures, CDDs and CDFs can be transported from water and soil to the air. Detection of these compounds at clean sites is therefore strongly suggestive of atmospheric deposition.

d. CDDs and CDFs can migrate to ground water if organic solvents are also present. In the absence of organic solvents, they are not expected to migrate significantly unless "channels" such as cracks in rocks are present.

e. CDDs and CDFs will bind strongly to suspended matter in water. The major "sinks" for these compounds in water are sediments, particulates, and living organisms.

f. Because of the extremely low water solubility of CDDs and CDFs, water-based leachate tests designed to simulate conditions in a municipal landfill are not likely to detect their presence.

3. AQUATIC TOXICOLOGY

In addition to toxic effects occurring at very low (parts per trillion) concentrations, the most striking aspect about the effect of "dioxin" on aquatic life is that toxic reactions are not observed until 5 to over 100 days after exposure. An amount as low as 5.6 parts per trillion has been shown to be lethal to salmon with other toxic effects observed as low as 0.1 parts per trillion. The CDDs and CDFs also are bioconcentrated to a high degree in aquatic organisms. The highest reported bioconcentration factor is approximately 9,000 for both rainbow trout and mosquito larvae. The most toxic CDDs and CDFs are also most preferentially bioconcentrated.

As this report went to press, the State Board learned of new toxicity and bioconcentration information obtained from a recent chronic study. Published in January 1988, the study examined the effects over a 56-day period of very low levels of the most toxic CDD and most toxic CDF on rainbow trout. Levels as low as 38 parts per quadrillion of the CDD had significant adverse effects on survival and growth. CDF levels as low as 0.9 parts per trillion reduced growth and 4 parts per trillion reduced survival. Bioconcentration factors by rainbow trout also were higher than previously reported: 39,000 for the CDD and 6,000 for the CDF.

4. MAMMALIAN TOXICOLOGY

Both CDDs and CDFs are absorbed and concentrated by humans and laboratory animals. The half-life of the most toxic CDD was over five years in a human volunteer, in contrast to shorter half-lives (10 to 40 days) in laboratory animals.

The most toxic CDD is also extremely variable in lethality, depending on animal species. For example, it takes approximately 5,000 times as strong a dose to kill a hamster as a guinea pig. As with aquatic animals, death in mammals is delayed after a single lethal dose, typically between 5 and 45 days. Death occurs after a period of wasting away.

In addition to lethality, these compounds also produce long term effects. Studies with laboratory animals have shown that the most toxic CDD causes reproductive (teratogenic) and fetal (fetotoxic) defects at very low exposure levels. These effects have not, however, been observed to date after accidental human exposure. Studies of the most toxic CDD and of a mixture of two other toxic CDDs have shown these compounds to be strong animal carcinogens. The U.S. Environmental Protection Agency (EPA) has rated the most

toxic CDD as the most potent animal carcinogen ever tested. However, there is little conclusive evidence from human exposure to date that this compound is linked to human cancer. A recent newspaper account in the New York Times (December 9, 1987) noted that EPA may reduce the estimate of CDD potency by a factor of 16. If this EPA rating system estimate does change, CDD will still be the most toxic carcinogen known. At the new estimate, the "safe" daily dose would be raised to 0.1 parts per quadrillion per day based on body weight (A part per quadrillion is one divided by 10^{15}).

5. CRITERIA AND STANDARDS

Criteria and standards have been developed primarily for the most toxic CDD. For example, the U. S. Food and Drug Administration in 1983 set a safe level of 25 parts per trillion in fish for human consumption as long as fish was not consumed more than twice a month. The U. S. Centers for Disease Control recommended a site specific cleanup level of 1 part per billion in soil. There is considerable debate in the scientific community over whether the 1 part per billion level for soil cleanup is too conservative (too safe) or not safe enough.

The EPA currently considers the most toxic CDD such a strong carcinogen that the one in one million risk level is set below the current chemical detection limit. This water criterion of 0.013 parts per quadrillion is based on a daily intake by a 70 kilogram man of 2 liters of water and 6.5 grams of fish or shellfish.

6. WOOD TREATMENT PRACTICES AND CALIFORNIA SITE CONTAMINATION

Pentachlorophenol and similar compounds have been used routinely for decades at sawmills and wood treatment facilities in California. Wood is typically treated by either dipping it in tanks containing the preservative solution, by spraying, or by forcing the solution under pressure into the wood. The latter method is used at wood treatment plants to provide long lasting protection. In contrast, sawmills use the dipping or spraying methods as a shorter term means to protect the surface from fungal growths that stain the wood and degrade its market value. Typically, the areas where wood is treated have been contaminated by the treatment chemical. Where pentachlorophenol has been used, the contaminants have included CDDs and CDFs. Because of their environmental persistence, these compounds may be present many years after the use of pentachlorophenol has ceased.

This report provides three examples of contamination by pentachlorophenol in California: at Visalia, Selma, and Oroville. At the Visalia site, a plume of organic solvents transported pentachlorophenol, CDDs and CDFs into both the shallow and deep aquifers. Contamination of the deeper aquifer was especially worrisome since the City of Visalia's drinking water wells were located downstream of the site. High levels of CDDs and CDFs were detected in soil samples at Selma while extensive pentachlorophenol contamination of ground water has occurred near Oroville.

7. CALIFORNIA WATER RESOURCES CONTROL BOARD STUDY

Based on a report of high levels of CDFs found in dip tanks at two Swedish sawmills, the State Board investigated wood treatment facilities in California to determine if CDDs and CDFs were also present.

When CDDs and CDFs are found, they usually occur as a complex mixture of different compounds. The degree of difficulty of chemical analysis for CDDs and CDFs depends on the type of analysis performed. The easiest method is group analysis. For example, one group of CDDs contains six chlorine atoms. There are actually ten different CDDs with six chlorines, but, by measuring only the group, the analysis is simplified.

Testing for individual CDD and CDF compounds is much more difficult. For example, of the ten different compounds in the six chlorine group of CDDs, three are highly toxic. The most accurate approach to evaluate their toxicity would be to measure the individual concentrations of these three highly toxic compounds.

The State Board study tested both the simpler group approach as well as individual compound analysis. In the group analysis phase, 13 samples -- soil (4), sludge (4), dip tank liquid (2), and commercial pentachlorophenol (3) -- were examined for presence of CDDs and CDFs. Significant concentrations of these groups of compounds were detected in all samples, with the highest concentrations found in the commercial formulations and dip tank sludges.

In the subsequent individual compound analysis phase, 12 samples from four sites (three sawmills, and one wood pressure treatment plant) were analyzed for the 15 most toxic CDDs and CDFs. Typically, at least three of the six toxic CDDs and seven of the nine CDFs were present in sawmill sludges and commercial mixtures. A noteworthy sample was obtained at a pressure treatment plant, where the

crystals or "bloom" formed on the surface of treated lumber contained five of the most toxic CDDs and eight of the most toxic CDFs. This information indicates that highly toxic CDDs and CDFs can be present, often at significant concentrations, as contaminants at sawmills and wood treatment plants.

8. HAZARD EVALUATION

The approach used in this report is based on an interim method, published in 1986 by the EPA, to evaluate the toxicity of CDD and CDF mixtures. It follows the premise that these different compounds follow similar toxicological pathways and that their toxic effects in mixtures are additive.

Each of the 15 highly toxic CDDs and CDFs has a different estimated toxicity. The EPA approach is to assign the most highly toxic CDD (the "dioxin") a toxicity value of 1.0 units, while the remaining 14 are given values ranging from 0.001 to 0.5 units, based on available toxicity information.

The "total" toxicity of a particular mixture of CDDs and CDFs is then calculated by multiplying the toxicity value of each separate CDD or CDF by its concentration in the sample. This step is performed for each of the highly toxic CDDs and CDFs and the results are added to obtain a total toxicity concentration for the mixture. Using this method, the highest relative toxicity concentration determined in a commercial pentachlorophenol formulation was 290 parts per billion. In sawmill dip tank sludge, the relative toxicity concentration ranged from 27 to 330 parts per billion. In the crystals formed after pressure treatment, the relative toxicity concentration was calculated to be 100 ppb.

Characterization of the "total" toxicity of CDDs and CDFs in mixtures by this method allows for estimation of site specific potential hazards as well as options for remedial action. The report recommends that remedial action assessment be based upon the "Decision Tree" approach developed by the California Department of Health Services. At some sites, moving the material may create more of a hazard than on-site storage of CDD and CDF containing materials isolated from humans and the environment. The latter approach may be the most effective interim measure until acceptable methods of CDD and CDF destruction become available.

RECOMMENDATIONS

1. Sawmill sludges and soils should be analyzed for the presence of CDDs and CDFs prior to disposal.

   The CDDs and CDFs previously concentrated in dip tank sludges will remain until the tanks are cleaned. Before disposal, these sludges should be analyzed for potential presence of CDDs and CDFs. If these compounds are present, sludge disposal by land or low temperature burning should be avoided. These materials should be held in interim storage until an effective means of destruction is identified and is available.

2. Wood treatment plants should improve management practices to isolate crystals of pentachlorophenol formed after treatment.

   Crystals (or "bloom") formed on lumber after pentachlorophenol pressurized treatment contain high levels of toxic CDDs and CDFs. During sampling by State Board staff, it was observed that some of this material falls to the ground during normal operating procedures. Plant operations should be improved to prevent environmental contamination by these crystals.

3. The highest priority should be given to isolating chlorinated dibenzodioxins and dibenzofurans from the environment and destroying them.

   Over 100 million dollars has been expended worldwide for research on the most toxic CDD. Nevertheless, many questions regarding toxicity and environmental fate of CDDs and CDFs still exist. Effective means to safely degrade these compounds, such as high temperature incineration or other methods, must be developed as rapidly as possible.

4. Interim on-site storage of CDD and CDF-containing materials is recommended until effective means of destruction are developed.

   Mobility and availability of CDDs and CDFs are dependent upon site specific soil types and characteristics, annual rainfall, plant and animal populations, and bioavailability. CDDs and CDFs should therefore not be placed in landfills. If, in the future, on-site land treatment is proposed, methods must be specifically designed for each site to avoid human or environmental exposure.

5. The California Site Mitigation Decision Tree Manual (Decision Tree) should be used as guidance for clean-up of CDD and CDF-contaminated sites.

   The Decision Tree process, published by the California Department of Health Services, consists of five elements: (1) preliminary site appraisal; (2) site assessment; (3) risk appraisal; (4) environmental fate and risk determination; and (5) development of site mitigation strategies and selection of remedial action.

6. Estimates of the concentrations of the most highly toxic CDDs and CDFs in contaminated materials should be made by following procedures described in this report.

   Considering the complexity and expense of analyzing for 210 individual CDDs and CDFs, analysis should be focused on the eight groups. Then the "total" toxicity of the most toxic CDD and CDF compounds in soil and dip tank samples can be estimated by using the percentage of highly toxic compounds calculated in this report. This will greatly simplify analysis for CDDs and CDFs by identifying only the four, five, six, and seven chlorine groups for each of these two compound classes.

7. Estimation of the toxicity of CDD and CDF mixtures should follow the U.S. EPA "toxicity equivalency factor" approach.

   As an interim approach to estimating the toxicity of samples containing CDD and CDF complex mixtures, the U.S. EPA has recommended a system based on multiplying the concentrations of individual highly toxic CDDs and CDFs by respective potency factors. These factors are based on both carcinogenicity and other toxicity test values of various CDDs and CDFs relative to the most toxic CDD.

8. EPA should develop a national strategy for identifying chemicals (or classes of chemicals) that may cause toxicity beyond the normal 96 hour acute test period.

   For chemicals thus identified, EPA should recommend observation periods for acute aquatic toxicity be extended from the current 96 hour standard bioassay test to at least 30 days beyond the acute test period. These recommendations follow observations of toxic effects induced by CDDs and CDFs up to one month after the initial exposure, when mortality did not occur within the standard 96 hour test period.

9. Interim advisories for highly toxic CDDs.

Advisory limits have been proposed by the U. S. Government, by other states, and by the province of Ontario, Canada, for drinking water, fish flesh, and soil cleanup. Although not the focus of this State Board report, the starred (*) levels listed below can serve as interim guidelines for California until advisories are established by the California Department of Health Services. It should be noted that some of these advisories are at or below the current practical detection limits for these compounds.

a. 2,3,7,8-tetraCDD (the most toxic CDD)

i. Drinking Water (protection of human health)

U.S. EPA (1984)	2.2×10^{-4} ppt*	(0.2 parts per quadrillion)
National Academy of Sciences (1977)	0.7 ppt	
New York State (Ground Water-drinking water supply) (1987)	3.5×10^{-2} ppt	(35 parts per quadrillion)

ii. Fish Flesh

U. S. FDA (1983)	50 ppt*
Province of Ontario (1986)	20 ppt
Michigan (1986)	10 ppt
New York (1987)	10 ppt

iii. Soil Cleanup Level

United States Centers for Disease Control (Atlanta, GA) (1984)	1 ppb	(site-specific for Times Beach, Missouri)

b. hexaCDD (six-chlorine CDD) - Drinking Water

U.S. EPA (1985)	5.5×10^{-3}* ppt	(5.5 parts per quadrillion)

TECHNICAL SUMMARY

1. INTRODUCTION

Highly toxic compounds were found in products and environmental samples at selected California sawmills and wood treatment plants. These were chlorinated dibenzodioxins and chlorinated dibenzofurans ("CDDs" and "CDFs"). These classes of compounds include 2,3,7,8-tetrachlorodibenzodioxin, which is popularly referred to as "dioxin".

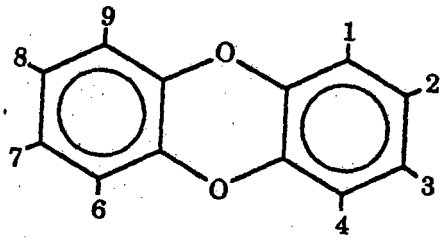
Structures of these compounds are shown in Figure 1. Dioxin has received widespread press coverage because it was a contaminant in Agent Orange, an herbicide used in Vietnam. It was detected in the streets of Times Beach, Missouri, and traced to contaminated oil used for dust control. The town was evacuated and bought out by the U. S. Government after the Centers for Disease Control determined that the 2,3,7,8-tetraCDD concentrations in soil represented an unreasonable risk to humans. Chlorinated dibenzofurans (CDFs) are contaminants in polychlorinated biphenyl (PCB) formulations. Both CDFs and PCBs contributed to significant human health problems in Japan and Taiwan. Rice oil had been accidentally contaminated with high concentrations of both compounds and was consumed by humans.

As shown in Figure 1, the chlorinated dibenzodioxin and dibenzofuran molecules each can contain from one to eight chlorine atoms. Since these can be arranged in a variety of ways, up to 75 CDDs and 135 CDFs are possible (Table 1). A mixture having both CDDs and CDFs theoretically could contain 210 individual compounds. The CDDs and CDFs having four, five, six, or seven chlorine atoms, four of which are in the 2,3,7, and 8 positions, are considered to be significantly toxic to mammals. The number of these is fifteen: six CDDs and nine CDFs (Table 2). The two eight-chlorine containing ("octa-") CDDs and CDFs also have four 2,3,7,8-substituted chlorine atoms. However, the octaCDDs and CDFs are believed to have low toxicity and in this report are not considered in the hazard evaluations of samples containing them.

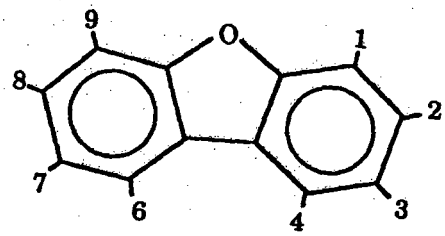
CDDs and CDFs are not produced intentionally, except as reference standards for chemical analysis. They appear, for example, as by-products of chemical synthesis, electrical equipment fires, and municipal incineration of solid wastes. They are contaminants of chlorophenol wood preservatives. In California, approximately 100 sawmills and wood treatment plants have been in operation or exist today. Almost half of these have used chlorophenol wood preservatives. These chemicals and their contaminants are present at an undefined number of sites, regardless of whether or not the plants are still operating.

FIGURE 1

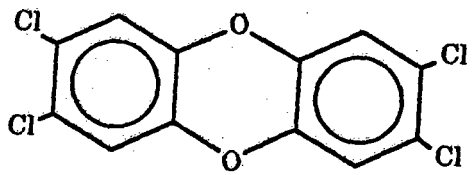
CHEMICAL STRUCTURES



Dibenzo-p-dioxin



Dibenzofuran



2,3,7,8-Tetrachlorodibenzo-p-dioxin
(2,3,7,8-TetraCDD)
("Dioxin")

NUMBERS ON STRUCTURES REFER TO LOCATION WHERE CHLORINE ATOMS CAN BE ATTACHED

TABLE 1

NUMBER OF COMPOUNDS IN CHLORINATED DIBENZODIOXIN AND DIBENZOFURAN ISOMER GROUPS

Isomer Group	Number of Compounds in Isomer Group
CDDs	
1. Monochlorodibenzodioxin (monoCDD)	2
2. Dichlorodibenzodioxin (diCDD)	10
3. Trichlorodibenzodioxin (triCDD)	14
4. Tetrachlorodibenzodioxin (tetraCDD)	22
5. Pentachlorodibenzodioxin (pentaCDD)	14
6. Hexachlorodibenzodioxin (hexaCDD)	10
7. Heptachlorodibenzodioxin (heptaCDD)	2
8. Octachlorodibenzodioxin (octaCDD)	1
TOTAL CDD COMPOUNDS	75
CDFs	
1. Monochlorodibenzofuran (monoCDF)	4
2. Dichlorodibenzofuran (diCDF)	16
3. Trichlorodibenzofuran (triCDF)	28
4. Tetrachlorodibenzofuran (tetraCDF)	38
5. Pentachlorodibenzofuran (pentaCDF)	28
6. Hexachlorodibenzofuran (hexaCDF)	16
7. Heptachlorodibenzofuran (heptaCDF)	4
8. Octachlorodibenzofuran (octaCDF)	1
TOTAL CDF COMPOUNDS	135
CDD AND CDF TOTAL	210

TABLE 2

2,3,7,8-CHLORINE SUBSTITUTED DIBENZODIOXINS AND DIBENZOFURANS

Isomer Group	Total Compounds in Isomer Group	Number of Compounds in Isomer Group with 2,3,7,8 Substitution	Specific Isomers
CDDs:			
Tetra-	22	1	2,3,7,8-tetraCDD
Penta-	14	1	1,2,3,7,8-pentaCDD
Hexa-	10	3	1,2,3,4,7,8-hexaCDD
			1,2,3,6,7,8-hexaCDD
			1,2,3,7,8,9-hexaCDD
Hept-	2	1	1,2,3,4,6,7,8-heptaCDD
Octa-	1	1	1,2,3,4,6,7,8,9-octaCDD
Total tetra through octaCDD compounds	49	7	
CDFs			
Tetra-	38	1	2,3,7,8-tetraCDF
Penta-	28	2	1,2,3,7,8-pentaCDF
			2,3,4,7,8-pentaCDF
Hexa-	16	4	1,2,3,4,7,8-hexaCDF
			1,2,3,6,7,8-hexaCDF
			1,2,3,7,8,9-hexaCDF
			2,3,4,6,7,8-hexaCDF
Hepta-	4	2	1,2,3,4,6,7,8-heptaCDF
			1,2,3,4,7,8,9-heptaCDF
Octa-	1	1	1,2,3,4,6,7,8,9-octaCDF
Total tetra through octaCDF compounds	87	10	

2,3,7,8-TetraCDD is the most potent animal carcinogen ever evaluated in the laboratory. EPA has estimated that this compound is approximately 20 and 50 times more potent than the next two highest-ranked carcinogens (a mixture of two hexaCDDs and Aflatoxin B_1, respectively). It is 50 million times more potent than trichloroethylene (TCE) or vinyl chloride. This 2,3,7,8 four chlorine-containing compound also is highly acutely toxic to certain animal species. A single feeding of one part to one billion parts body weight will kill half of a guinea pig test population.

The findings of dramatic CDD acute toxicity and carcinogenicity in animals contrasts with the lack of comparable findings in humans. Over one hundred million dollars has been spent over the last few decades studying the toxicity and fate of principally one compound, 2,3,7,8-tetraCDD. Large gaps in knowledge still exist. The most prudent approach at this time should be minimizing CDD and CDF entry into the environment. This is an alternative to continuing to spend large sums of money on research that produces as many questions as answers.

The present State Board study detected CDDs and CDFs at sawmills and wood treatment plants in soils and dip tank liquids and sludges. CDDs and CDFs were present where pentachlorophenol had been used for wood preservation. Most of the toxic CDD and CDF compounds listed in Table 2 were detected in all samples. To our knowledge, this is the first study in the United States which has identified the fate of individual 2,3,7,8-substituted CDDs and CDFs in chlorophenol wood preservatives after their use. The analytical chemistry necessary to perform such detailed trace analysis involved three laboratories in the United States and Sweden.

2. ENVIRONMENTAL FATE

The anticipated stability and distribution of CDDs and CDFs depends upon the individual compound, environmental conditions, and the nature of experiments designed to predict its environmental fate. Available data show that CDDs and CDFs can be (1) formed in the environment; (2) degraded; (3) remain unchanged; and (4) migrate through soil to ground water. The most useful predictive information comes from actual field measurements as well as laboratory experiments which have been constructed to simulate field conditions closely. The fairly sizable number of environmental fate experiments, especially in the area of light-related effects is confusing, but a general understanding of this fate is beginning to emerge.

Phototransformation

CDDs and CDFs resist sunlight-induced breakdown when they are present in water and on dry surfaces such as soil, wood, and glass (i.e., solid-phase surfaces). This resistance is increased with increasing number of chlorine atoms in the molecule. When chlorine atoms are lost under solid-phase conditions, those in the most toxic 2,3,7,8-substituted positions appear to be preferentially retained. This relative stability contrasts with the instability demonstrated in laboratory experiments. In these, CDDs and CDFs were dissolved in organic solvents, which enhance breakdown, and were irradiated with ultraviolet light. These conditions promote transformation to less toxic CDDs and CDFs. In the field, if organic solvents are present, they would enhance the transport of CDDs and CDFs through the soil out of range of sunlight effects. This is a current explanation for the finding of CDDs in California and Florida ground water. Until recently, CDDs and CDFs were thought to be immobile in soil, tightly bound to soil particles due to their low water solubility, and therefore not a threat to ground water.

Microbial Degradation

Unlike the sometimes marked degradative effect that microorganisms have on many compounds, CDDs either resist transformation or are only slowly degraded by microorganisms.

Sometimes, transformation compounds cannot be identified and therefore their toxicities cannot be estimated. One fungal species has been shown to degrade 2,3,7,8-tetraCDD in a nitrogen-limited culture. The usefulness of this fungus to transform CDD contaminated soil awaits evaluation. Literature on microorganism effects on CDFs is lacking, but these compounds probably show similar resistance to transformation.

Volatilization

The U.S. EPA has recently noted that volatilization is a likely fate for CDDs in aquatic environments. This contrasts with an earlier conclusion that volatilization probably was not an important process. It is consistent with a 1981 evaluation by the National Research Council of Canada: In simulating the fate of 2,3,7,8-tetraCDD in two model aquatic ecosystems, 100 percent was estimated to be lost through volatilization and none to photolysis or microbial degradation. The Research Council concluded in 1984 that despite a lack of data for CDFs, but by inference from CDD data, volatilization could play a role in environmental distribution for this class of compounds.

Little information is available to predict the stability and extent of distribution of CDDs and CDFs once they have evaporated from water and land surfaces. They can exist in vapor and adsorb to particulate matter in air. The presence of CDDs and CDFs in lake sediments located on a Lake Superior island indicates that these compounds can be atmospherically transported and subsequently redeposited.

Persistence and Movement in Soils and Sediments

As noted above, CDDs and CDFs have been detected in ground water, probably by being transported through soil by organic solvents. CDDs have reached a depth of 30 meters in Florida, and 16 meters in California. In the absence of organic solvents, CDDs and CDFs are not expected to move downward to any great extent.

Migration of these compounds at waste disposal and land treatment sites cannot be predicted accurately by spiking solvent-free soil with CDDs and CDFs, and rinsing the soil with water. A standard soil leachate test specified by RCRA for dioxin-containing wastes requires use of water to leach CDDs from soil. A more accurate test would employ a mixture of water and organic solvents. The more accurate test would increase the amounts of CDD and CDF compounds extracted from soil and thereby their concentration in a leachate test. This in turn would increase the likelihood that CDD and CDF contaminated soil would not be acceptable under RCRA treatment standards. At present, the RCRA treatment standard requires that wastes found to contain any tetra-, penta-, or hexaCDDs or CDFs at concentrations of 1 ppb or more in a standard leachate test be treated before land disposal.

CDDs and CDFs are also expected to adsorb strongly to sediments and suspended particulate matter in water. As a result, and because of their stability, they are expected to be highly persistent in these associations. In aquatic systems, therefore, the major "sinks" for CDDs and CDFs will be sediments, suspended particulates, and biota.

Plant Uptake

Measurements of CDDs and CDFs have shown that these compounds are concentrated in aquatic plant extracts. This can be interpreted to show that CDDs and CDFs are taken up and concentrated by aquatic plants. However, an undetermined amount of this material may be adsorbed onto the plant surface rather than being absorbed by the plants. Bioaccumulation figures for these compounds in aquatic plants should be interpreted with some reservation, especially for unicellular phytoplankton where the surface area is large compared to the internal volume. The distinction is not important to zooplankton or fish consumers of aquatic plants:

adsorbed and absorbed CDDs and CDFs are both consumed with the food. With respect to terrestrial plants, EPA has recently concluded that 2,3,7,8-tetraCDD present in contaminated soil is "not likely" to concentrate in them. If true, plants would not be effective scavengers of CDDs and CDFs in soil, a use which has been suggested for on-site treatment of CDD and CDF-contaminated soil. Reported bioconcentration of CDDs by terrestrial plants may be due to contamination of leaf and plant surfaces by CDDs in dust and soil particles.

3. AQUATIC TOXICOLOGY

Two striking aspects of 2,3,7,8-tetraCDD toxicity to aquatic life are the (1) delayed toxic effects after brief periods of exposure; and (2) low concentrations which cause toxic reactions. Frequently, toxicity is not seen in the standard short-term, 96-hour acute test. Statistically significant adverse effects have been delayed for periods ranging from five to over 100 days after exposure to this chemical. Growth retardation is the most common effect reported for 2,3,7,8-tetraCDD. Other effects include fin necrosis, loss or underdevelopment of caudal fins, edema, liver necrosis, and hemorrhaging.

Toxic effects have been reported at water concentrations as low as 0.1 parts per trillion (ppt) 2,3,7,8-tetraCDD. The lowest acute LC_{50} value of 5.6 ppt for coho salmon is one order of magnitude lower than for two of the most toxic chemicals to aquatic life, endosulfan and toxaphene. (LC_{50} refers to the concentration of a chemical which kills 50 percent of a test population within a specified time period.)

Due to the delayed lethality normally found in 2,3,7,8-tetraCDD bioassays, the expression of LC_{50} for a 96-hour exposure is not a meaningful indicator of 2,3,7,8-tetraCDD toxicity. As a result, the literature concerning 2,3,7,8-tetraCDD describes modified LC_{50}s indicating mortality at some given time after the exposure period. There is no agreement on a standardized post exposure observation period for the calculation of LC_{50}.

Most toxicity studies with CDDs have focused on 2,3,7,8-tetraCDD. They have generally been short-term 96-hour exposures, and have been "static" or "static renewal" bioassays. The water and test chemical were either not renewed for the test period, or were renewed periodically as a batch replacement. Studying the toxicity of highly water insoluble compounds such as CDDs under static testing conditions can present difficulties. For example, a compound will tend to migrate out of the aqueous test solution and adsorb onto solid surfaces such as the test container, test organisms, or particulate debris. The adsorbed test chemical may not be available to the test organisms.

Adverse toxic reactions most likely would have been observed at lower concentrations of 2,3,7,8-tetraCDD than reported, if the bioassays had been the continuous-flow type. Here, both water and toxic chemicals are renewed on a continuing basis. This simulates many natural situations. Effects may be seen at lower water concentrations because of the continuous renewal of water containing the toxicant.

Few CDD chronic studies have been reported. CDF toxicity has been estimated only in studies where CDF-contaminated food was provided to the fish.

CDDs and CDFs accumulate in aquatic organisms. The highest reported bioconcentration factor for 2,3,7,8-tetraCDD is approximately 9,000 for both rainbow trout and mosquito larvae. This is possibly an underestimate of bioconcentration potential due to the static test condition.

One investigator exposed fish to a mixture of CDDs and CDFs containing from four to eight chlorine atoms. With few exceptions, those compounds having chlorines at the 2,3,7, and 8 positions were selectively concentrated by the fish. Others have observed that compounds with chlorine atoms in other positions also were accumulated by fish. In these latter experiments, the 2,3,7, and 8 compounds were not present. The extent to which molecular configuration influences uptake needs clarification.

Studies of elimination of CDDs and CDFs from fish that have been exposed to these compounds in water showed: (1) rate of elimination decreases with increasing chlorination of the compound; and (2) for the same degree of chlorination, CDFs are depurated at a greater rate than CDDs.

Subsequent to completion of this State Board report, data were published that showed higher toxicity and bioconcentration than previously reported. This new study, published in January 1988, described chronic effects of 2,3,7,8-tetraCDD and 2,3,7,8-tetraCDF on rainbow trout. The experiment was a 56-day flow-through test with 28 days of exposure followed by 28 days of depuration. At 38 parts per quadrillion 2,3,7,8-tetraCDD, the lowest concentration tested, significant adverse effects were observed on growth and survival. Because effects were determined at the lowest level, a no observed effect concentration (NOEC) for this CDD could not be derived. At 0.9 parts per trillion (ppt) 2,3,7,8-tetraCDF, reduced growth effects were reported and reduced survival was observed at 4 ppt. NOEC values were 0.4 ppt for growth and 1.8 ppt for survival for this CDF. While the

higher concentrations tested caused mortality within 28 days, the toxic effect of lower concentrations was not manifested until later. During the 28-day depuration period, mortality continued and there was no observed recovery in clean water.

The same study also reported bioconcentration factors of 39,000 for 2,3,7,8-tetraCDD and 6,049 for 2,3,7,8-tetraCDF. This newly published study concluded that 2,3,7,8-tetraCDD is more than 10,000 times as toxic to fish as the insecticides endrin or toxaphene and that 2,3,7,8-tetraCDF is roughly 1,000 times as toxic.

4. MAMMALIAN TOXICOLOGY

Absorption, Tissue Distribution, Metabolism, and Half-Lives

Both CDDs and CDFs are absorbed and concentrated by laboratory animals and humans. Up to 90 percent of the chemicals will be absorbed if they are present in food. Approximately 40 percent can be absorbed after skin application to laboratory animals.

The half-life of 2,3,7,8-tetraCDD in a 42 year old human volunteer was estimated to be 5.8 years. This is longer than the half-life of about one year for the same compound estimated for monkeys. It contrasts with 10 to 40 day half-lives measured in several small laboratory animals. Based on blood sample analyses, a half-life of greater than one year was calculated for 2,3,4,7,8-pentaCDF and 1,2,3,4,7,8-hexaCDF compounds in humans. These people had ingested rice oil contaminated with these and other CDFs in Taiwan. In Japan, following a similar incident, the same pentaCDF could still be detected in human blood 11 years after exposure.

Studies with 2,3,7,8-tetraCDD contaminated soil show that ingested soil can influence toxicity. Soil from a Times Beach, Missouri, area which was contaminated with waste oil containing CDDs and CDFs, produced a variety of adverse effects, including acute toxicity in laboratory studies. In contrast, contaminated soil from a 2,4,5-T and 2,4-D formulation site in New Jersey produced no toxicity in laboratory animals. Bioavailability of the chemicals, including CDDs and CDFs, appears to account for the difference between these two observations. This was estimated to range from 0.5 to 21 percent for the New Jersey soil and 25 to 85 percent for the Times Beach soil. Bioavailability refers to the amount which is expected to be absorbed into the animal's bloodstream and not tightly bound to the soil particles which would be eliminated as waste.

CDDs and CDFs can be expected to be distributed in the body in proportion to the amount of fat content of a particular tissue. In both laboratory animals and humans, highest concentrations are found in adipose tissue and liver.

Laboratory experiments with 2,3,7,8-tetraCDD and a CDF mixture have shown that these chemicals can move through the placenta. One study also showed that CDFs are transferred to the offspring in greater amounts through milk, compared to transport through the placenta.

Laboratory studies have shown that animals can transform absorbed 2,3,7,8-tetraCDD. Unidentified transformation products have been detected principally in the bile and urine. Depending on the compound, metabolites can be either more or less toxic than the parent from which they are derived. EPA has noted that metabolism of 2,3,7,8-tetraCDD appears to be mostly a detoxification process which produces metabolites less toxic than the parent compound.

Acute, Subchronic, and Chronic Toxicity Effects

As noted, one of the most acutely toxic substances known is 2,3,7,8-tetraCDD. However, species sensitivity can differ significantly. The male hamster is approximately 8000 times less sensitive than the male guinea pig in a short-term lethal dose test. When 2,3,7,8-tetraCDD is fed to animals in acutely toxic doses, death is delayed and may take from 5 to 45 days. During this period, weight loss occurs with the animals exhibiting a characteristic "wasting away" appearance. This compound also induces liver damage in most species. The immune system is adversely affected in all species tested. Thymic atrophy is the principal change. The spleen, lymph nodes, and bone marrow may be affected. Susceptibility to bacterial infection is increased, and antibody production decreased.

One experiment focused on the relative effects of technical grade pentachlorophenol (PCP) and its contaminants on immunosuppresion. The contaminants included chlorinated diphenyl ethers, phenoxy phenols, dibenzodioxins and dibenzofurans. Technical grade PCP contained 86 percent pentachlorophenol. This produced a dose-related decrease in antibody response. In contrast, analytical grade PCP, which was greater than 99 percent pure, had no effect. Neither did the chlorinated phenoxy phenol or diphenyl ether components. The experimenters concluded that a significant amount of the immunosuppression was caused by the CDDs and CDFs.

Most human exposures to CDDs and CDFs have occurred either occupationally or accidentally, and concurrently with exposure with other chemicals. In these situations the actual dose

received could not be determined. The most common human effects attributed to 2,3,7,8-tetraCDD exposures include chloracne, liver abnormalities, hematologic disorders, porphyria, and hyperpigmentation disorders. Also reported have been peripheral and central neurological disorders, lethargy and sensory impairment. Chloracne is characterized by comedones and cysts. These may subside within a few months or persist for years, with some cases reported lasting up to 15 years after exposure.

Other human exposure sources to 2,3,7,8-tetraCDD include (1) dirt roads in Missouri sprayed with waste oil containing 2,3,7,8-tetraCDD, and (2) Agent Orange, the herbicide used in Vietnam also contaminated with this compound. The 2,3,7,8-tetraCDD concentrations in the Missouri soil ranged from 39 to 2200 ppb. Persons exposed to this material had lived in the area from one to five years during the period of contamination. Signs of altered liver function included lower serum bilirubin and elevated urinary uroporphyrin concentrations. However, these measurements were considered to be "subclinical"; i.e., not significantly differing from the normal.

The Agent Orange exposure is discussed in the section "Carcinogenicity" below.

The Japanese rice oil contaminated with 1,000 ppm PCBs and 5 ppm CDFs, which included 0.45 ppm 2,3,7,8-tetraCDF, produced the following toxic effects in humans, collectively known as "Yusho": pigmentation disorders, chloracne, eye discharge, swelling of upper eyelids, distinctive hair follicles, and neurological disturbances.

Teratogenicity and Reproduction

2,3,7,8-TetraCDD is a teratogen to laboratory animals. Cleft palate is the most common malformation observed in mice. Kidney defects are also common as well as embryo toxicity. In rats, teratogenic effects include subcutaneous edema, hemorrhage in the gastrointestinal tract, kidney malformation, cleft palate, and vertebral defects. In monkeys there are insufficient data to clearly define a teratogenic response, although fetotoxicity has been observed. Studies of humans exposed to 2,3,7,8-tetraCDD in the chemical industry, during the Vietnam war and in forestry operations, have not been able to show a teratogenic or other adverse effect on reproduction. The animal data conclusively demonstrate that 2,3,7,8-tetraCDD is teratogenic and fetotoxic at low levels of exposure. They indicate a need to determine more carefully the potential for adverse human reproductive effects.

Studies whose purpose has been to determine the mutagenic potential of CDDs and CDFs have produced conflicting results. One of the reasons for this, at least for 2,3,7,8-tetraCDD, is that its high toxicity may preclude demonstration of a mutagenic response.

Carcinogenicity

Both 2,3,7,8-tetraCDD and a mixture of two hexaCDDs are potent animal carcinogens, as noted. At this time, although many people have been regularly exposed to CDD-contaminated formulations, there is little conclusive evidence linking CDD to human cancers. The difference between laboratory and human observations is surprising.

Public Law 96-151, enacted in December 1979, mandated the U. S. Veterans Administration to perform a comprehensive review and analysis of the world literature on Agent Orange and other phenoxy herbicides. Output from the original task has continued as a series of publications with Volumes IX and X being published in May 1987. These latest analyses show some associations between exposure to phenoxy herbicides, which may or may not have contained dioxins, and adverse human health impacts. However, the cited studies are noted to have shortcomings which "limit their usefulness as evidence of a cause-and-effect relationship." These include negative findings in observations made by other researchers and lack of ability to correlate effect with known exposure dose, or even to determine conclusively that all affected persons were exposed to the herbicide. One recent observation that needs further study is a statistically significant excess of non-Hodgkin's lymphoma in U. S. Marine Corps veterans who served in Vietnam compared to those who did not serve in Vietnam.

5. CRITERIA, STANDARDS, AND REGULATIONS

In the United States and Canada, criteria have been developed for certain chlorinated dibenzodioxins but not chlorinated dibenzofurans. The CDDs identified are 2,3,7,8-tetraCDD and "hexaCDD". The only agency to have adopted criteria as legally enforceable standards is the New York State Department of Environmental Conservation. The standards are for 2,3,7,8-tetraCDD: (a) 1 part per quadrillion in ambient water (10^{-6} ug/l); and (b) 35 parts per quadrillion in ground water (3.5×10^{-5} ug/l). The former is lower because of potential for bioaccumulation by aquatic organisms.

EPA has developed several criteria for 2,3,7,8-tetraCDD including those for the following: (1) ambient water for drinking purposes only (0.2 parts per quadrillion); (2) ambient water based on

consumption of fish and shell-fish only (0.014 parts per quadrillion); (3) ambient water based on consumption of water, fish and shellfish (0.013 parts per quadrillion); (4) total intake from all sources for humans (0.006 picograms per kilogram body weight per day); and (5) ambient air (0.03 picograms per cubic meter). Specific criteria are listed which relate to the one increased incidence of cancer per one million population risk level.

Other 2,3,7,8-tetraCDD criteria have been developed by the following agencies: (1) Michigan Department of Public Health (10 ppt in fish); (2) California Air Resources Board and Department of Health Services (30 femtograms per cubic meter in air); (3) U. S. Centers for Disease Control (1 ppb in soil); (4) U. S. Food and Drug Administration (50 ppt in fish); and (5) Ontario Ministry of the Environment, Ontario, Canada (20 ppt total intake from all sources for humans): The U. S. Food and Drug Administration also set an additional advisory level for consumption of fish containing 25 to 49 ppt 2,3,7,8-tetraCDD. Fish with concentrations in this range should not be consumed more than twice per month.

HexaCDD criteria have been developed by the following: (1) EPA (5.5 parts per quadrillion in drinking water; 0.8 picograms per cubic meter in air; and 0.16 picograms per kilogram body weight per day for all sources in humans); (2) California Air Resources Board and Department of Health Services (1 picogram per cubic meter in air); and (3) National Research Council of Canada (13 ppt in ambient water for human consumption of fish; and 20 ppt for fish flesh).

Regulations have been developed for both CDDs and CDFs which relate primarily to treatment methods and disposal. The California Department of Health Services regulates 2,3,7,8-tetraCDD in wastes disposed to land to protect against migration to surface and ground water. EPA has developed CDD and CDF treatment standards and prohibits land disposal of certain wastes containing these compounds unless treatment standards are achieved. The designated wastes include several chemicals with which CDDs and CDFs are associated as contaminants and include tri-, tetra-, and pentachlorophenol; tetra-, penta-, and hexachlorobenzene, and 2,4,5-T. They also include residues resulting from incineration or thermal treatment of soil contaminated with certain EPA-designated hazardous wastes. In addition, EPA regulations require registrants of pentachlorophenol to reduce the concentration of hexaCDD in three phases. By February 2, 1989, the maximum batch hexaCDD concentration allowed will be 4 ppm, with a maximum average of 2 ppm; this is a decrease from the present allowable maximum batch concentration of 15 ppm.

6. WOOD TREATMENT PRACTICES AND CALIFORNIA SITE CONTAMINATION

Chlorophenols such as pentachlorophenol (PCP), tetrachlorophenol (TCP), and their potassium and sodium salts, creosote, coal tars, and copper arsenate compounds have been used routinely at sawmills and wood treatment facilities in California. Wood is typically treated by immersing it in tanks containing the preservative solution, by spraying, or by forcing the solution under pressure into the wood to saturate the cells more fully for a longer lasting protection. Over time there is an accumulation of chemical residuals in sediments and sludge of the treatment systems. Often the treating, sorting, and drying areas become contaminated by the preservative solution.

Chlorophenols are recognized to contain CDDs and CDFs. The used preservative solution, including accumulated sediment and sludge, and contaminated soil, also contain CDDs and CDFs. Currently these wastes must be either stored on-site or disposed of outside of California because CDD and CDF-containing wastes are no longer accepted at California landfills. A nationwide ban on land-filling of dioxin-containing wastes goes into effect November 9, 1988.

On-site methods of disposal have been attempted; none are effective. These include burning in a teepee burner which, because of relatively low temperature burning, not only does not destroy CDDs and CDFs, but also produces them from precursor chlorophenol compounds. In addition, this procedure releases them to the environment adsorbed to the soot. Burial of wastes on-site also has been a common practice. As a temporary measure, on-site storage and containment of these materials in drums has been recommended as an interim disposal practice, but a long-term solution is still needed.

Three examples of California contamination occurring as a result of wood treatment operations are described. Each of these is in a different stage of the evaluation and cleanup process. They are representative of several additional sites in the state which are awaiting further investigation.

Oroville Wood Treatment Site: A 200-acre wood treatment facility near Oroville, Butte County, has been associated with the lumber industry since about 1920. Both PCP and creosote have been found in soil and ground water, both on and off-site. PCP in concentrations of up to 15,000 ppb has been detected in ground water below the site. (The California Department of Health Services Drinking Water Action Level for PCP is 30 ppb.) The depth to water is approximately 30 feet. A plume of PCP in concentrations up to 2000 ppb has been detected at least two miles south of the site. The depth to water in this area is

90 to 120 feet. Approximately 30 domestic wells have been found contaminated with PCP. No CDDs or CDFs have been detected. Residents have complained of various adverse health effects. A comprehensive study is underway to define the extent of PCP contamination.

Selma Wood Treatment Site: An 18-acre wood treatment facility has been in operation since approximately 1936 near Selma, Fresno County. As with similar facilities, a number of preservative chemicals have been used here including chromated copper arsenate and pentachlorophenol dissolved in a variety of solvents. Wastes were discharged into dry wells, into an unlined pond, as runoff into drainage ditches, to open ground, and into a sludge pit. PCP has been detected on-site in surface and ground water and in soil. Surface water concentrations have ranged from 0.24 to 2.3 ppm. The PCP ground water concentration was determined to be 2 ppb. The depth to water is approximately 30 feet. All tetra-through-octaCDD and CDF isomer groups, except tetraCDD, were detected in soil.

Off-site migration may have occurred since the vertical and horizontal extent of soil and ground water contamination has not been defined. EPA is currently conducting a sampling program to clarify this uncertainty.

Visalia Wood Treatment Site: This facility, in Tulare County, had used PCP for electrical pole treatment from 1968 to 1980, when operations ceased. Ground water contamination was detected in 1973 and has been followed since then. Hexa-, hepta-, and octaCDDs and CDFs, and PCP have been detected in shallow and deep aquifers. These and pentaCDFs also were detected in soil. There were no pentaCDDs detected in soil. PCP was detected in monitoring wells 600 feet to the south of the site at concentrations ranging up to 37 ppm and 1600 feet to the southwest at concentrations up to 2 ppm. Creosote was found in these samples. Additional monitoring wells were constructed in 1984 and soil cores taken during this work were analyzed to provide information on the vertical distribution of PCP, creosote, CDDs, and CDFs.

Ground water has been pumped from the shallow aquifer to the City of Visalia wastewater treatment plant since 1975. The purpose has been to reduce contaminant concentrations and prevent further migration away from the site. Additionally, a bentonite-cement slurry wall has been built below the surface to inhibit down-gradient movement of the contaminants. The barrier surrounds the shallow aquifer beneath the site and extends from the surface to its lower boundary. PCP, creosote, CDDs, and CDFs were detected in both aquifers whose waters were discharged to the treatment plant. All of these compounds were detected also in plant

influent, effluent, and sludge with one exception: CDDs and CDFs were not detected in the plant effluent. Water from the deep aquifer is used by the City of Visalia for drinking water. Sludge from the treatment plant has been used as a soil amendment by farms and residents. CDFs have been detected in soil. In 1985 a pretreatment system was installed at the site to remove ground water contaminants before water transfer to the treatment plant.

7. CALIFORNIA WATER RESOURCES CONTROL BOARD STUDY

The study reported here originally was based on potential pentachlorophenol contamination of the environment. The focus was on its use by sawmills and wood treatment plants. Samples taken for analysis included aquatic invertebrates and fish, treatment site runoff, ground water, and soil. At that time, State Board staff considered that environmental contamination by chlorinated dibenzodioxins and dibenzofurans might be of equal significance. (They were known to be contaminants of chlorophenol formulations.) To test this hypothesis, 13 samples were taken from five sawmills and one wood treatment plant (Table 3, Section A). Sample types and numbers were as follows: soil (4), sludge (4), dip tank liquid (2), and commercial chlorophenol formulations (3). Analyses detected significant CDD and CDF concentrations. A decision was made to base the study on CDD and CDF presence in areas of sawmills and wood treatment plants. Chlorophenols would become the subject of another survey.

This initial work showed that tetra-, penta-, hexa-, hepta-, and octaCDDs and CDFs were present in all 13 samples, with one exception; tetraCDDs were detected only in wet and dry sludge samples from one sawmill and in one pentachlorophenate commercial product. The commercial chlorophenol and chlorophenate products were found to contain both tetra-chlorophenol and pentachlorophenol.

Analyses at this stage identified CDDs and CDFs in terms of "isomer groups", e.g., "tetraCDD", "heptaCDF". The analyses did not identify specific CDDs and CDFs, e.g., 2,3,7,8-tetraCDD. Determination of the exact position of the chlorine atoms requires a rigorous analytical procedure. As noted earlier, a total of 210 individual CDDs and CDFs possibly can occur.

After CDD and CDF presence was firmly established in the 13 samples, a decision was made to concentrate future work on the 15 CDDs and CDFs that were toxicologically most significant, i.e., the tetra, penta, hexa, and hepta-chlorinated compounds

TABLE 3

CALIFORNIA WATER RESOURCES CONTROL BOARD
CHLORINATED DIBENZODIOXIN AND DIBENZOFURAN STUDY

A. Preliminary Screening: Isomer Group Analyses

1. 5 Sawmills and 1 Wood Treatment Plant: 13 samples as indicated:

   a. Soil (4)
   b. Sludge (4)
   c. Dip tank liquid (2)
   d. Commercial formulations (3)

B. Phase I: Compound Specific Analyses

1. Sawmill A (Trinity County): 2 samples

   a. Commercial sodium pentachlorophenate
   b. Dip tank sludge

2. Sawmill B (Glenn County): 2 samples

   a. Wet dip tank sludge
   b. Dry mix tank sludge

C. Phase II: Compound Specific Analyses

1. Sawmill C (Humboldt County): 4 samples

   a. Commercial potassium tetrachlorophenate
   b. Dip tank liquid
   c. Dip tank sludge (2 samples)

2. Wood Treatment Plant (San Joaquin County): 4 samples

   a. "Bloom"
   b. "Commercial"--recycled treatment material
   c. Soil at retort
   d. Sump liquid

which have four of the chlorine atoms located in the 2,3,7, and 8 positions (Table 2). Three of the target compounds are potent carcinogens to laboratory animals: 2,3,7,8-tetraCDD and a mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-hexaCDD.

In order to estimate the concentration of these 15 compounds in complex mixtures, which also include many non-2,3,7,8-substituted CDDs and CDFs, three methods can be followed. The first two are fairly straightforward. They require little more time than that to determine isomer group concentrations. One of these methods assumes that all tetra through heptaCDDs and CDFs are chlorinated at positions 2,3,7, and 8. This procedure could greatly overestimate the significance of the 2,3,7,8-substituted compounds since there may be a far greater number of non-2,3,7,8-substituted compounds present. The second method assumes that all compounds within an isomer group are present in equal numbers; e.g., 2,3,7,8-tetraCDD is one of 22 possible compounds in the tetraCDD isomer group, and its concentration would be 1/22 of the total tetraCDD concentration detected. While simple in concept, this procedure could significantly underestimate or overestimate the toxicity of a CDD mixture, depending whether or not 2,3,7,8-tetraCDD was present. The third method identifies each 2,3,7,8 CDD and CDF in a potential mixture of 210 CDDs and CDFs and numerous other interferences. This approach represents state-of-the-art analytical chemistry for CDDs and CDFs. It was the course chosen for the study reported here.

The work proceeded in two phases. Phase 1 was directed at analyzing some of the previously collected samples which were shown to contain high concentrations of CDD and CDF isomer groups. Phase 2 was initiated with additional samples once 2,3,7,8-substituted CDDs and CDFs were identified in Phase 1. All samples analyzed are described in Table 3.

A brief summary of the analytical results follows. All data are described in detail in the accompanying report appendices.

CDDs: 2,3,7,8-Chlorinated compounds from all four target isomer groups (tetra through hepta) were detected in all 12 samples analyzed, with the following exceptions: 2,3,7,8-tetraCDD was detected in only one sample and 2,3,7,8-pentaCDDs were detected in five of 12 samples.

CDFs: 2,3,7,8-Chlorinated compounds from all target isomer groups were detected in the 12 samples analyzed, with the following exceptions: tetraCDFs were found in 9 of 12 samples, with pentaCDFs and hexaCDFs in 10 of 12.

The concentration of the 2,3,7,8-substituted compounds was calculated also as a percentage of the total CDD or CDF concentration for each isomer group. Depending on the sample and the isomer group, the proportion of the 2,3,7,8 compounds ranged from a few percent to greater than 80 percent of the total concentration of the respective isomer group. This finding was based on analysis of the environmental samples and the two commercial chlorophenate products.

All samples except for one dip tank solution contained at least 1,000 ppb total tetra-through-hepta 2,3,7,8-chlorinated CDDs and CDFs (Table 4; Total 2,3,7,8 CDDs and CDFs). The total concentration of tetra-through-hepta 2,3,7,8-chlorinated CDDs and CDFs ranged between 44 and 41,000 ppb. The concentrations of 2,3,7,8-tetraCDD and CDF are given separately because of the high toxicity of the former, and of the latter by analogy. The presence of 2,3,7,8-tetraCDF is particularly significant because of its close structural resemblance to 2,3,7,8-tetraCDD. The table shows that 2,3,7,8-tetraCDF was present in all 8 samples taken at sawmills, up to concentrations of 200 ppb.

The study data also show that the following 2,3,7,8-chlorinated CDD and CDF compounds are most likely to be found as a result of tetrachlorophenol and pentachlorophenol use at sawmills and wood treatment plants.

1,2,3,6,7,8-hexaCDD
2,3,7,8-tetraCDF
1,2,3,7,8-pentaCDF
2,3,4,7,8-pentaCDF
1,2,3,6,7,8-hexaCDF

Although the data are complex, a brief overview of analyses of these 12 samples indicates the following: 2,3,7,8-chlorinated CDDs and CDFs are present as contaminants at sawmills and wood treatment plants, often at significant concentrations.

8. HAZARD EVALUATION

Compound Detection

As noted, a major assumption was made that most of the toxicity in CDD and CDF mixtures is contributed by the 2,3,7,8-chlorinated compounds. Laboratories in the United States and Sweden participating in the State Board Study obtained analytical standards for the 15 most toxic 2,3,7,8-CDDs and CDFs. Often these had to be synthesized since they were not commercially available. Analytical procedures were developed and refined for their detection. When detected in a sample, a concentration for each 2,3,7,8-chlorinated CDD and CDF was determined for each of

TABLE 4

SUMMARY OF 2,3,7,8-SUBSTITUTED CDD AND CDF CONCENTRATIONS IN TWELVE COMPOUND-SPECIFIC ANALYSES (TETRA, PENTA, HEXA, AND HEPTA ISOMER GROUPS) - ppb[1/]

Sample	2,3,7,8-Tetra-CDD	2,3,7,8-Tetra-CDF	All 2,3,7,8-Chlorinated CDDs[2/]	All 2,3,7,8-Chlorinated CDFs[2/]	Total 2,3,7,8-CDDs & CDFs[2/]
Commercial Na-PCP, Sawmill A	0	201	34,751	6,540	41,291
Commercial K-TetraCP, Sawmill C	0	200	1,197	1,148	2,345
Sawmill Dip Tanks					
Sawmill A sludge	0	15	25,305	3,333	28,638
Sawmill B wet sludge	0	17	2,332	485	2,817
Sawmill B dry sludge	9.7	95	15,411	2,177	17,588
Sawmill C center sludge	0	54	1,092	560	1,652
Sawmill C corner sludge	0[3/]	65	1,161	574	1,735
Sawmill C liquid	0	2.0	18	26	44
Wood Treatment Plant-					
PCP "Bloom"	0	4.4	24,183	10,712	34,895
Recycled "Commercial"	0	0	6,715	726	7,441
Soil at Retort Mouth	0	0	1,618	169	1,887
Sump Liquid	0	0	8,684	69	8,753

1/ Average of samples split between two laboratories.

2/ Does not include octaCDD and octaCDF.

3/ Reported at 6.8 ppb by one laboratory but not confirmed by second.

the 12 samples. For each of the samples, the relative proportion of the 2,3,7,8-chlorinated CDDs and CDFs in each isomer group also was calculated.

Toxicity Evaluation

Toxicity information was available for only a few of the 2,3,7,8-chlorinated compounds. In order to overcome this deficiency, three methods were considered to determine total sample toxicity based on toxicity of the individual 2,3,7,8-compounds.

1. The simplest approach is to assign the same "toxic equivalency factor" to each 2,3,7,8-chlorinated tetra-, penta-, hexa-, and heptaCDD and CDF, i.e., assume they are all equally toxic. The toxic equivalency factor is multiplied by the concentration of each compound detected to yield a "relative toxicity concentration." All products are added together to estimate a "total relative toxicity concentration" for all CDDs and CDFs in each sample.

   This approach does not take into consideration the different toxicities of individual compounds. It can be justified on the basis of limited toxicity information for most of the 2,3,7,8-substituted compounds, taking into account that toxicity generally was high where it has been measured.

2. The California Department of Health Services currently favors an approach which is based solely on data provided by carcinogenicity bioassays. Only two toxic equivalency factors can be estimated with this scenario because only 2,3,7,8-tetraCDD and a mixture of two 2,3,7,8-chlorinated hexaCDD compounds have been tested for carcinogenicity. With this method, all other CDDs and CDFs are assigned one or the other of the two factors. As with the first approach just described, each factor is multiplied by the appropriate compound concentration to estimate a relative toxicity concentration for each compound. The products also are added to estimate a total relative toxicity concentration for all CDDs and CDFs.

3. The U.S. Environmental Protection Agency has developed toxic equivalency factors for the 2,3,7,8-chlorinated CDDs and CDFs by taking into consideration both carcinogenicity information and other toxic effects data, such as those relating to reproductive effects. These equivalency factors are listed in Table 5. EPA also considers toxicity of non-2,3,7,8-chlorinated CDDs and CDFs and assigns them factors. These are one to three orders of magnitude less than those for the respective chlorinated compounds. Relative toxicity concentrations and total toxicities are estimated using the same steps described for the first two approaches.

TABLE 5

TOXIC EQUIVALENCY FACTORS FOR 2,3,7,8-CHLORINATED DIBENZODIOXINS AND DIBENZOFURANS (SOURCE: BELLIN AND BARNES, 1986)

	Compound	Toxic Equivalency Factor[2/]
CDD		
	2,3,7,8-tetraCDD	1.0
	1,2,3,7,8-pentaCDD	0.5
	1,2,3,4,7,8-hexaCDD	0.04
	1,2,3,6,7,8-hexaCDD	0.04
	1,2,3,7,8,9-hexaCDD	0.04
	1,2,3,4,6,7,8-heptaCDD	0.001
CDF		
	2,3,7,8-tetraCDF	0.1
	1,2,3,7,8-pentaCDF	0.1
	2,3,4,7,8-pentaCDF	0.1
	1,2,3,4,7,8-hexaCDF	0.01
	1,2,3,6,7,8-hexaCDF	0.01
	1,2,3,7,8,9-hexaCDF	0.01
	2,3,4,6,7,8-hexaCDF	0.01
	1,2,3,4,6,7,8-heptaCDF	0.001
	1,2,3,4,7,8,9-heptaCDF	0.001

1/ Bellin, J., and D. Barnes, 1986. Interim Procedures for Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzodioxins and Diobenzofurans (CDDs and CDFs). Risk Assessment Forum, U. S. Environmental Protection Agency EPA/625/3-87/012. Washington, DC.

2/ Toxic Equivalency Factors are based on carcinogenicity and other toxicity data relative to that for 2,3,7,8-tetrachlorodibenzodioxin (2,3,7,8-tetraCDD).

A comparison of the total relative toxicity concentrations estimated by the three methods for each of the 12 samples, shows a difference of three orders of magnitude between them (Table 6). The most conservative, i.e. highest, concentrations are based on the sum of all tetra through hepta 2,3,7,8-chlorinated CDDs and CDFs which have been given the same toxic equivalency factor (Method 1). These concentrations were 5 to 30 times greater than those calculated by the California Department of Health Services procedure (Method 2). The latter, in turn, were 3 to 30 times higher than those calculated by the EPA approach (Method 3). For example, the total relative toxicity concentrations calculated by the three methods for the "Sawmill B" dry-sludge sample were 17,588 ppb, 1094 ppb, and 330 ppb, respectively.

The authors of this report recommend that, until more 2,3,7,8-chlorinated compound-specific toxicity information is available, the EPA procedure be used to estimate the total relative toxicity concentrations of CDD and CDF mixtures. This method, unlike the previous two, takes into account all available toxicity information for the various CDD and CDF compounds.

Comparisons of relative toxicity concentrations also were made between CDD and CDF "isomer groups" in each sample for the 2,3,7,8-substituted compounds. In one sawmill dip-tank sludge sample, the compounds contributing the most relative toxicity, based on the EPA method, were the pentaCDFs (38 percent); the hexaCDDs (32 percent); and 2,3,7,8-tetraCDF (23 percent). These figures are based on a total sample relative toxicity concentration for CDDs and CDFs of 27.9 ppb (Sawmill C, Table 6).

The relative toxicity concentration of all 2,3,7,8-substituted CDFs in the same sample was approximately twice that of the CDDs. The estimated relative toxicity concentration for these CDFs was 18 ppb and for CDDs, 9.9 ppb.

Future Sample Toxicity Evaluation

The authors recommend a simplified approach to estimating total CDD and CDF toxicity of similar samples in future analyses. It is based on (1) performing isomer group analyses; and (2) using the ratios of the 2,3,7,8-chlorinated compounds identified in this study, relative to isomer group concentrations. These ratios can be used to estimate relative toxicity concentrations for similar sample types, when only isomer group analyses are performed. The current data bases (12 samples) can be increased by additional compound-specific analyses by other specialist laboratories. CDD and CDF isomer group analyses can be performed by many commercial laboratories. Only a few laboratories in the United States are capable of doing the more definitive analyses on a reasonable schedule.

TABLE 6

TOTAL RELATIVE TOXICITY CONCENTRATIONS (ppb) OF 2,3,7,8-CHLORINATED DIBENZODIOXINS AND DIBENZOFURANS: A COMPARISON BASED ON THREE METHODS[1/]

	Method 1	Method 2	Method 3
SAMPLE	TEF=1[2/]	CDHS 1986[3/]	Bellin and Barnes, EPA 1986[4/]
Commercial Na-PCP			
Sawmill A	41,291	2,055	289.5
Commercial K-tetraCP			
Sawmill C	2,345	463	72.8
Sawmill Dip Tanks			
Sawmill A sludge	28,638	1,184	139.1
Sawmill B wet sludge	2,817	173	32.0
Sawmill B dry sludge	17,588	1,094	329.6
Sawmill C center of tank sludge	1,652	216	27.0
Sawmill C corner of tank sludge	1,735	218	27.9
Sawmill C liquid	44	8.4	0.8
Wood Treatment Plant-PCP			
"Bloom"	34,895	1,120	100.5
Recycled "Commercial"	7,441	223	11.3
Soil at Retort Mouth	1,887	64	5.6
Sump Liquid	8,753	274	9.8

1/ OctaCDD and octaCDF were not considered in the calculations due to estimated low toxicity.

2/ Toxic Equivalency Factor = 1 for each 2,3,7,8-chlorinated CDD and CDF.

3/ California Department of Health Services, 1986; Relative potency of 2,3,7,8-tetra- and pentaCDDs and CDFs = 2,3,7,8-tetraCDD; and 2,3,7,8-hexa- and heptaCDDs and CDFs = 2,3,7,8-hexaCDD (or 0.03 2,3,7,8-tetraCDD).

4/ Bellin, J. and D. Barnes. 1986. Interim Procedures for Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzodioxins and Dibenzofurans (CDDs and CDFs). Risk Assessment Forum, U. S. Environmental Protection Agency EPA/625/3-87/012. Washington, DC.

The only site-specific cleanup level that has been established for CDDs or CDFs in the United States has been 1 ppb for 2,3,7,8-tetraCDD in Times Beach, Missouri. Total relative toxicity concentrations calculated for the 12 samples in this study -- using the EPA method -- showed that 11 exceeded 1 ppb. These ranged from 5.6 to 329.6 ppb (Table 6).

The concentration for the twelfth sample was 0.8 ppb. All 12 exceeded the 1 ppb level based on the California Department of Health Services' method of calculation.

Setting a Clean Up Level. Contamination by CDD and CDF mixtures associated with chlorophenol products used at sawmills and wood treatment plants should be cleaned up following a site-specific procedure. The present study concludes that the DHS California Site Mitigation Decision Tree Manual, although complex, should be followed. The Decision Tree includes five components:

1. Preliminary risk appraisal;
2. Site assessment;
3. Risk appraisal;
4. Environmental fate and risk determination; and
5. Determination of mitigation strategy and remedial action plan selection.

The risk appraisal phase uses applied action levels (AAL) for specific media of exposure such as air, soil, water, and biota. These have been set to protect specific biological "receptors". The AALs also take into account the amount of a substance taken in by inhalation, ingestion, and adsorption, as well as other toxicological factors such as absorption, metabolism, distribution, and elimination characteristics of the medium.

The California Department of Health Services is currently reviewing a consultant's report containing proposed air and water AALs for CDDs. A CDHS report describes a strategy for developing AALs related to soil contact. Numerical AALs for CDDs in soil will be proposed by CDHS in 1988.

Characterization of CDD and CDF mixtures in samples by calculating total relative toxicity concentrations will allow an estimate of potential hazard. The options for remedial action can then be identified. At some sites, moving the material may create more of a hazard than encapsulation and on-site storage. On-site storage with material isolated from humans and the environment may be the most effective interim measure until acceptable methods of CDD and CDF destruction are available.

CHAPTER 1: INTRODUCTION

Why are polychlorinated dibenzo-p-dioxins (CDDs) and dibenzofurans (CDFs) important? The best known and most studied of the CDDs is the chemical 2,3,7,8-tetrachlorodibenzo-p-dioxin, commonly called "dioxin". In ranking the potency of 55 suspected human carcinogens, the U.S. EPA (1985b) listed "dioxin" as the most potent -- 50 million times more potent than trichlorethylene (TCE) or vinyl chloride. This CDD compound also is highly toxic in a single dose to certain animal species. In a single feeding of one part "dioxin" to one billion parts body weight, half of the guinea pigs dosed will die. However, unlike a lethal dose from many other highly toxic chemicals, death is delayed from 5 to 45 days after exposure occurs. In addition to 2,3,7,8-tetrachlorodibenzo-p-dioxin, several other CDDs and CDFs are of probable toxicological concern.

BACKGROUND

The California Water Resources Control Board's (State Board) investigation of chlorinated dibenzo-p-dioxins and dibenzofurans originated with a study of pentachlorophenol (PCP). This compound is a major industrial chemical and biocide used worldwide. In California, PCP has been used extensively for wood preservation at lumber mills and wood treatment plants. Typically, a water soluble form of PCP is used at sawmills for surface protection against fungal staining of lumber. In contrast, wood treatment plants inject insoluble PCP under pressure for long-term protection of materials such as poles and posts. Most of these facilities are located in two areas of the state, the northwest and the central valley. Investigations by the Regional Water Quality Control Boards and other agencies have documented a number of effects on California's environment. These include fish kills; contaminated soil, surface water and ground water; accumulation in marine sediments and organisms; and incidents of worker exposure. A few California studies have also detected CDDs and CDFs in both commercial PCP and PCP-contaminated soil.

In the past five years, conditions at sawmills have noticeably improved. Some mills have converted to systems that completely contain and recycle wood preservative chemicals on site, preventing environmental contamination. In particular, the "unit dip" tank has been successful. In a unit dip operation, a below ground rectangular tank is filled with a wood preservation solution (typically the soluble form of pentachlorophenol is diluted 1:100 parts water). Sawn lumber is bundled together, immersed in the tank, then allowed to dry in a covered building sloped such that drippage drains back into the dip tank. A side

effect of this "best management practice" is accumulation of sawdust and dirt on the tank bottom that forms a sludge. Eventually, the sludge becomes deep enough to interfere with dip operations and must be removed. An examination of two Swedish sawmills that used chlorinated phenols as wood preservatives noted that these sludges became "remarkedly enriched" in chlorinated dibenzofurans (Levin et al., 1976). Levels of total CDFs as high as 700 ppm were detected. Upon learning of the findings in Sweden, the State Board's priority chemical study of pentachlorophenol was expanded to include monitoring for CDDs and CDFs in dip tanks and other locations at sawmills and to investigate contaminant levels at wood treatment plants.

NOMENCLATURE OF CDDs AND CDFs

Although the term "dioxin" has become synonymous with 2,3,7,8-tetrachlorodibenzo-p-dioxin, "dioxin" is not used elsewhere in this document because the subject of this report is not one but a number of different CDDs. The nomenclature of CDDs and CDFs is important because there are enormous differences in toxicity between compounds. Those compounds chlorinated at the 2,3,7, and 8 positions and containing from four to seven chlorine atoms are believed to be most toxic. In this report, these CDDs and CDFs are referred to as 2,3,7,8 chlorine-substituted compounds or, more simply, as 2,3,7,8 congeners.

The basic skeleton of all the CDDs is dibenzo-p-dioxin, a molecule containing two benzene rings joined by two oxygen atoms (Figure 1.1). The dibenzo-p-dioxin molecule is chlorinated if a chlorine atom is attached to any of the positions numbered 1 through 4 and 6 through 9. The dibenzo-p-dioxin skeleton can accommodate up to eight chlorine atoms. 2,3,7,8-Tetrachlorodibenzo-p-dioxin contains four chlorine atoms, one each at the 2,3,7, and 8 positions (Figure 1.1). For purposes of simplification, 2,3,7,8-tetrachlorobdibenzo-p-dioxin is abbreviated as 2,3,7,8-tetraCDD in this document. Numbers indicate location of chlorine atoms on the molecule and tetra refers to four chlorines. Other four chlorine dibenzo-p-dioxins can also occur, for example 1,4,6,9-tetraCDD. In fact, there are 22 different ways that four chlorines can be arranged on the molecule; in chemical terminology there are 22 different "isomers".

There are three terms of chemical nomenclature that are used in this document to characterize CDDs: isomer group, isomer, and congener. CDDs can be divided into eight groups called isomer groups (also called homologues), with each isomer group containing the same number of chlorine atoms. For example, tetraCDD is the four chlorine isomer group of CDDs. An isomer is defined by the arrangement of chlorine atoms within an isomer

matter in the air. Summarizing other work, the CARB and CDHS (1986) stated that CDDs and CDFs appear stable when adsorbed to particulate matter, can migrate over great distances in the air, and are probably highly persistent in the atmosphere. Czucwa et al. (1984) found CDDs and CDFs in sediments from a lake located on Isle Royale in Lake Superior and concluded that their presence could only be explained by atmospheric deposition. Thus, while volatilization may remove CDDs and CDFs from aquatic and terrestial compartments, these compounds may be atmospherically transported and subsequently redeposited.

Persistence and Movement in Soil and Sediments

CDDs and CDFs are believed to adsorb strongly to soils, sediments, and biota. Sediments and suspended particulates will serve as sinks for these compounds in aquatic systems (NRCC, 1981); because of strong sorption, they will be highly persistent in the environment. As demonstrated at a site in Visalia, California, these compounds can travel considerable distances downward in soil if organic solvents are present (see Chapter 6: Monitoring). In the absence of organic solvents, CDDs and CDFs are not expected to migrate downward to any great extent. Recent evidence suggests that measurement of CDD movement in soil, based on spiking clean soil with CDDs, does not accurately portray migration at waste disposal and land treatment sites (Nkedi-Kizza et al., 1985; Enfield, 1985; Jackson et al., 1985).

Because these compounds will bind very tightly to organic material in soils and contaminated materials, CDDs and CDFs may escape detection in standard water leachate tests. Use of aqueous leachate tests for these compounds as a screening device prior to land disposal is inappropriate. Rather, a leachate mixture composed of water and organic solvents should be developed and used to determine levels of CDDs and CDFs (Nkedi-Kizza et al., 1985; Jackson et al., 1985). Thus, use of standard leachates required under RCRA for dioxin-containing wastes (U.S. EPA, 1986b) is probably inappropriate because it will underestimate concentrations of CDDs and CDFs in contaminated soils. The treatment standard requires that waste found to contain any tetra-, penta-, or hexaCDD or CDF at levels of 1 ppb or higher in a standard leachate test be treated before land disposal. Young (1981) noted that when soil has been contaminated for several years, the extraction of 2,3,7,8-tetraCDD and subsequent chemical analysis is difficult. The aqueous leachates referenced in the RCRA regulations may not desorb CDDs and CDFs that are highly adsorbed to organic material. In order to extract CDDs and CDFs from soils for chemical analysis, organic solvents are required (see discussion

of chemical analysis in Appendix E). Similar extraction compounds should be used in leachate tests for detection of CDDs and CDFs.

Plant Uptake

There is consensus that plants in aqueous systems take up and concentrate CDDs and CDFs (U.S. EPA 1984a; U.S. EPA 1985b), although Kenaga and Norris (1983) have noted that these compounds may be adsorbed onto external surfaces of aquatic plants rather than actually taken up into plant cells. However, the data for uptake by terrestial plants are less clear. Studies of 2,3,7,8-tetraCDD concentrations in crops grown near the site of the Seveso, Italy chemical accident are contradictory: the presence of this congener in plants is variously attributed to plant uptake and translocation, contaminated dust, and volatilization from soil. After reviewing the literature, the U.S. EPA (1985b) concluded that 2,3,7,8-tetraCDD is "not likely to concentrate in plants grown on contaminated soils."

It should be noted that concentrations of 2,3,7,8-tetraCDD in contaminated soils where plant uptake was examined were on the order of one ppb. Levels of 2,3,7,8-chlorinated CDDs and CDFs encountered at California wood treatment plants and sawmills were much greater (in the high ppb to low ppm range as described in Chapter 5). An important research project would be to determine kinetics of plant uptake where high levels of CDDs and CDFs are present in soils. As Young (1981) has observed, animals foraging on CDD contaminated plants can potentially relocate these compounds off-site.

Land Treatment

In-situ land treatment has been proposed as a potential cleanup method for sites contaminated by CDDs, CDFs, and chlorinated phenols. This option has the potential advantage of being a relatively inexpensive method to clean large volumes of contaminated soil when compared to costs of thermal destruction or removal to distant landfills. Although the inherent assumption is that land treatment will be accomplished by photolysis and perhaps microbial degradation, Young (1981) has observed that reductions in CDD and CDF levels may involve off-site transport, including wind and water movement of contaminated particles, volatilization, and biomass removal. What is needed is a careful study of land treatment, which will examine a number of uncertainties including those listed below.

CHAPTER 6: WOOD TREATMENT PRACTICES AND CALIFORNIA SITE CONTAMINATION

This chapter presents a brief overview of wood treatment practices. Three California wood treatment facilities are described to provide examples of chlorophenol-related CDD and CDF contamination.

WOOD TREATMENT PRACTICES

Chlorophenols, such as pentachlorophenol (PCP), tetrachlorophenol and their potassium and sodium salts, creosote, coal tars and copper arsenate compounds have been used routinely at sawmills and wood treatment facilities in California. At sawmills, salts of PCP are used to treat freshly cut wood to prevent sap stains caused by the action of fungi and molds, an example being "blue stain" which leaves a dark discoloration on unprotected wood. Unlike pentachlorophenol, which is highly insoluble in water, the sodium and potassium salts (pentachlorophenates) are very soluble.

PCP is commonly sold as a solid containing 95 percent PCP, and is applied to wood products as a 5 percent solution after being dissolved in a petroleum solvent (Baker and Matheson, 1981). It is used also in the form of sodium and potassium chlorophenate salts, usually as aqueous solutions of approximately 0.15 percent pentachlorophenate. Most chlorophenate products are mixtures of chlorophenols with one compound normally present in greater amounts than others. For example, one such product has approximately 14 percent pentachlorophenol, 8 percent tetrachlorophenol, 6 percent other chlorophenols and the remainder composed of inert ingredients. Such a solution would then be diluted with water for surficial wood treatment purposes.

At sawmills where surficial treatment is used to prevent fungal damage, rough sawn lumber may be treated by either dipping it into large tanks containing the preservative solution, or by spraying the solution on the wood after sawing. The wood is then set aside and allowed to dry. Provisions may or may not be made to recover the excess treatment solution, and in many cases it is lost to the soil in the sorting or drying areas. A recent improvement employed by some operations is the construction of treating and drying facilities with sloping floors and sumps, which allow the excess treatment solution to be collected and either recycled or disposed of.

Other wood preservation methods may use pressure treatment methods, usually a pressurized retort, to more fully saturate the cells of the wood for more complete and longer lasting protection; utility poles are commonly treated in this manner. Several carriers have been used in pressure retort operations; for example, PCP may be dissolved in oil, liquified petroleum gas

and isopropyl ether mixture, mineral spirits or methylene chloride (Morgan, 1986). In some cases the PCP may be dissolved in a solvent such as diesel oil, and wood products may be soaked in the solution without the use of a pressure system.

Over time there is an accumulation of treating process residuals in the form of sediments and sludge in most systems. In sawmill dip tank operations, these residuals consist of sawdust and other debris which collect at the bottom of the treatment tank and must be periodically removed. Disposal of these wastes is currently a problem, since they contain CDDs and CDFs at much higher levels than found in the treatment solution. Since CDD and CDF containing wastes are no longer accepted at California landfills due to potential liability problems (see Chapter 7; Criteria, Standards, and Regulations), this waste must be either temporarily stored on-site or disposed of outside of California. (The U.S. Hazardous and Solid Waste Amendments of 1984 specifically banned landfilling of "dioxin-containing wastes" effective November 8, 1986. However, the Amendments allowed issuance of a national variance on the ban for up to 2 years. Citing the lack of disposal and treatment options, the U.S. EPA issued a variance that will expire on November 8, 1988.)

One available means of disposal has been to burn the sludge on-site by various low-temperature methods, such as in a tepee burner. Burning under such conditions not only does not reliably destroy CDDs and CDFs, but also produces them from precursor chlorophenol compounds. In addition, it releases them to the environment adsorbed to the soot (Tiernan et al., 1983). Burial of these wastes on-site has also been a common practice. Recently, as a temporary measure, on-site storage and containment of these materials in drums has been recommended as an interim measure, but a long-term solution is still needed.

CASE STUDIES OF CONTAMINATED SITES IN CALIFORNIA

Annual production of PCP in the United States is estimated by U.S. EPA (Esposito et al., 1980) to be about 53 million pounds annually. In California, over 2 million pounds of PCP were sold in 1983 (CARB and CDHS, 1986). Approximately 90 percent of this amount was used in wood treatment facilities employing pressure treatment methods (pentachlorophenol), and 10 percent was used in sawmill operations (pentachlorophenate salts).

Three examples of contamination occurring as a result of wood treatment operations are described, with each in a different stage of the evaluation and cleanup process. They are also fairly representative of several additional sites in the State which are awaiting further investigation. California currently has approximately 10 wood treatment facilities and 86 sawmills in operation (CARB and CDHS, 1986), along with a number of facilities which are no longer functional. A recent consultant's

report to the CARB contains an inventory of California sawmills and wood treatment plants using chlorinated phenols as of December 1986 (Chinkin et al., 1987). According to the report, five pressure treatment plants account for 98 percent of current chlorophenol use in wood treatment. The remaining two percent reflects tetrachlorophenate use at four sawmills. This represents a decline in chlorophenol use at sawmills since 1983.

Oroville Wood Treatment Site

A wood treatment facility near Oroville, California is currently being evaluated for soil and ground water contamination resulting from long-term wood preservative use. This 200 acre site has been associated with the lumber industry since about 1920. Both PCP and creosote have been found in soil and ground water both on and off-site (U.S. EPA, 1986a).

The investigation, still in its preliminary stages, has determined levels of PCP in the soil of at least 10 ppm with creosote also present. The depth to water is about 30 feet, and levels of PCP in the ground water below the site range up to 15,000 ppb. Ground water flows in a south-southwest direction, and private wells adjacent to and downgradient from the site have levels of PCP ranging up to 4000 ppb. To the south of the site a plume of contamination extending at least two miles and containing levels of PCP up to 2000 ppb has been detected. The depth to water in this second area is 90 to 120 feet (U.S. EPA, 1986a).

While approximately 30 domestic wells in this rural area have been found to be contaminated with PCP, no CDDs or CDFs have been detected in ground water. Residents have complained of adverse health effects they believe are related to the contamination, such as diarrhea and skin disorders, and an alternate domestic water supply has been provided. Recovery wells have been constructed in the area to the southwest of the site in an effort to reduce contaminant levels (U.S. EPA, 1986a).

In compliance with a U.S. EPA work plan, surface water, ground water, sediment and soil core studies will be performed along with other hydrogeological testing. The first phase of soil and ground water study is expected to better define the extent of contamination (U.S. EPA, 1986a).

Selma Wood Treatment Site

A facility near Selma in Fresno County has also been associated with soil and water contamination resulting from wood preserving operations. This 18 acre facility has been in operation since about 1936, and is bordered by residential, agricultural and industrial areas, some located as close as one-fourth of a mile. (U.S. EPA, 1986d).

A variety of wood preserving chemicals have been used at this site during its history, with those used since 1965 including chromated copper arsenate (CCA), copper-8-quinolinolate, and pentachlorophenol dissolved in ketone solvents, diesel fuel or mineral spirits. During 1982 it was estimated that, using pressure treating methods, about 1,000 gallons of 5 percent PCP solutions and 3,000 to 4,000 gallons of a 1.5 percent CCA solution were used daily to treat lumber products, including utility poles, grape stakes, and fence posts (CVRWQCB, 1982).

During its operation the disposal of treatment related wastes was accomplished by discharge into dry wells, into an unlined pond, runoff into drainage ditches, to the open ground and into a sludge pit. Relatively recent improvements include disposal of drummed waste, such as sludge off-site, and containment of contaminated surface runoff from the treatment area (CVRWQCB, 1982).

The Central Valley Regional Water Quality Control Board first sampled the site in 1971, with the Department of Health Services becoming involved in 1983, and the EPA assuming enforcement responsibility in 1984. The results of this sampling are summarized in Table 6.1.

The aquifers and aquitards in the area are composed of continuous and discontinuous layers of unconsolidated gravel, sand, silt and clay, with the depth to water approximately 30 feet. To the west of the site is found the Corcoran Clay layer, which divides the ground water into a confined and unconfined aquifer system. Because the facility is located on the eastern side of the Central Valley in what may be a recharge zone for those aquifers to the west (ground water flows to the southwest from the site), there is concern about off-site migration since the vertical and horizontal extent of soil and ground water contamination has not been completely defined (U.S. EPA, 1986d). Currently, the U.S. EPA is conducting a sampling program as part of its investigation to better define the vertical and lateral extent of contamination both on and off site.

The CDD and CDF results for 2 of the 25 soil samples and for both pressurized retort effluent samples taken in April 1986 are shown in Table 6.2. The CDD and CDF levels in the soil samples are similar to those of the retort effluent samples; however, some tetra- and pentaCDFs and pentaCDDs were detected in soil samples which were not present in the retort effluent samples.

Visalia Wood Treatment Site

Ground water contamination resulting from the use of PCP and creosote at a facility where electrical power poles were treated has been followed and documented since 1973. This site is located at Visalia, California, where a dip tank containing PCP

TABLE 6.1

SELMA PRESSURE TREATMENT PLANT CONTAMINATION SUMMARY
(adapted from U. S. EPA 1986c)

	PCP (ppm)[1/]
Drinking Water Standard	1.0[2/]
Surface Water Sampling Results Range	0.24-2.3
Soil Sampling Range	
Surface to 2 ft. depth	0.06-4,500
2 ft. to 5 ft.	<0.8 -3,100
5 ft. to 10 ft.	0.1 - 600
10 ft. to 20 ft.	2.6 - 41
Greater than 20 ft.	<0.5 - 1.2
Ground Water Sampling Results Range	0.002

1/ Parts per million
2/ State of California Action Level

TABLE 6.2

CDD AND CDF CONCENTRATIONS (WET WEIGHT)
IN SOIL AND RETORT EFFLUENT AT SELMA
PRESSURE TREATMENT SITE
(Compiled from U. S. EPA, 1986c)

	Soil Samples		Retort Effluent Samples	
	A	B	A	B
CDD (ppb):				
tetra	ND[a/]	ND	ND	ND
penta	5.8*	ND	ND	ND
hexa	324*	383*	380*	275*
hepta	3,970*	5,100*	18,500*	19,400*
octa	13,300	14,900	10,500	110,000
CDF (ppb):				
tetra	2.3*	3.1*	ND	ND
penta	72.5*	80*	ND*	ND
hexa	601*	711*	917*	767*
hepta	1,410*	1,660*	11,300*	10,500*
octa	2,990	6,200*	43,700	49,200

[a/] Not detected
* Approximate values

dissolved in number 2 diesel oil was discovered leaking in late 1972. The tank was replaced and an investigation initiated to determine the extent of soil and ground water contamination. The plant used PCP for pole treatment from 1968 to 1980, when operations came to an end.

In this area ground water is contained in two saturated zones separated by an aquitard, a feature characteristic of the San Joaquin ground water basin (Figure 6.1; DWR, 1982). At the site, depth to water in the shallow unconfined aquifer is approximately 30 feet, with the aquitard varying between 10 and 20 feet in thickness encountered at about 65 feet confining the deeper aquifer (SCE, 1985d).

The unsaturated and unconfined layers, considered moderately permeable, are composed of alluvial silts and fine sands near the surface, progressing through medium and coarse grained sands to pebble gravel at the upper boundary of the aquitard (SCE, 1983). The aquitard is composed of silts and clays of low hydraulic conductivity and is considered a leaky, saturated confining layer for the deep aquifer, which consists of coarser grained more permeable alluvial deposits. Ground water in the shallow aquifer flows in a generally south-southwest direction, following a gradient of about 17 feet per mile. Flow in the deep aquifer is in a generally west-southwest direction, following a gradient of about 15 feet per mile (SCE, 1984). The deep aquifer is confined at its base by relatively impermeable beds, and is widely used as a source of drinking water by many in the area, including the City of Visalia.

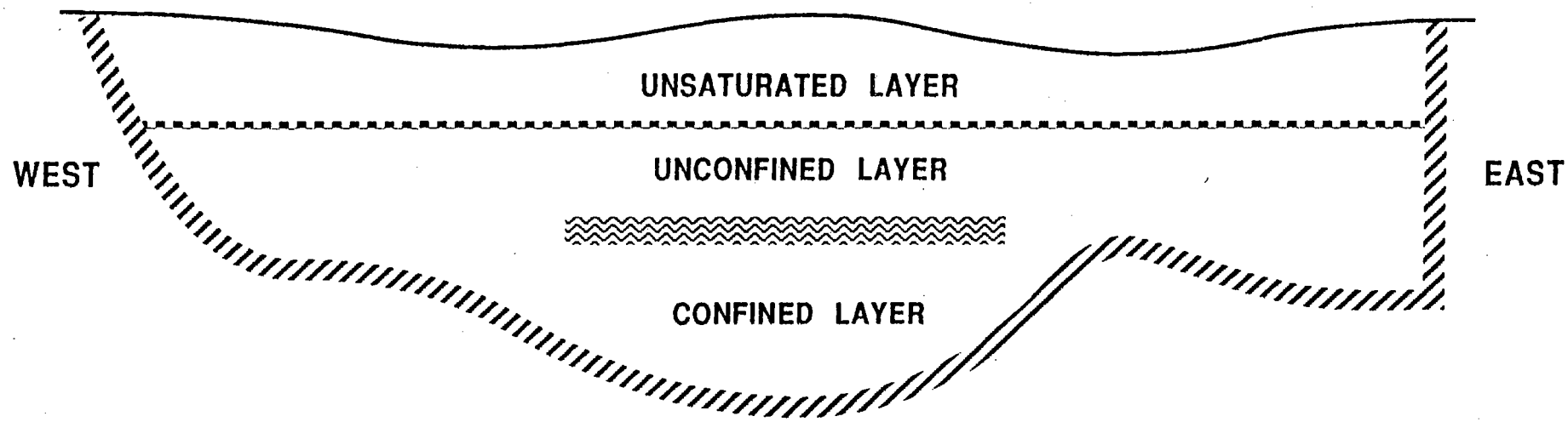
Since the leak was discovered, a series of monitoring and recovery wells have been installed, as both the shallow and deep aquifers have been contaminated with PCP, creosote, CDDs and CDFs. To inhibit downgradient movement of the contaminants off-site, a bentonite-cement slurry wall has been built below the surface. This barrier surrounds the shallow aquifer beneath the site, and extends from the surface to its lower boundary.

Contaminant levels in ground water have fluctuated significantly since the investigation began in 1973, with the highest levels reached in 1977, (Table 6.3). During this same period PCP was detected in monitoring wells 600 feet to the south of the site at levels of 0.3 to 37 ppm, and also 1600 feet to the soutwest at levels of 0.007 to 2 ppm, with creosote also present in both cases (SCE, 1983).

To reduce contaminant levels and prevent further migration away from the site, ground water has been pumped from the shallow aquifer since 1975, and from the deep aquifer since 1976. Over time, additional monitoring and recovery wells have been added. The water has been discharged to the City of Visalia Water Conservation Plant. CDDs and CDFs were found in the ground water

FIGURE 6.1

TYPICAL CROSS SECTION OF THE
SAN JOAQUIN VALLEY GROUNDWATER BASIN
(ADAPTED FROM DWR, 1982)



LEGEND

Aquitard (Confining Clay Layer)

Water Table

No Flow Boundary

TABLE 6.3

CONCENTRATIONS OF PCP, CDDs AND CDFs IN SOIL AND WATER AT VISALIA POLE TREATMENT SITE*

Contaminant	Shallow Unconfined Aquifer 1977	1984	1985	Deep Confined Aquifer 1977	1984	1985	Soil A 1985		Soil B 1985		Sewage Sludge 1985
							Lab 1	Lab 2	Lab 1	Lab 2	
PCP (ppm)	44,000	17.0	16.7	6.3	4.5	1.5	-		-		-
Creosote (ppm)	73,000	6.2	6.5	270	47.0	21.2	-		-		-
CDD (ppb):											
Tetra	-[a/]	<0.001	<0.00054	-	<0.001	<0.0007	<0.2	<0.07	<0.1	<0.05	<0.03
Penta	-	<0.001	<0.0013	-	<0.001	<0.00023	<0.2	<0.23	0.1	<0.17	<0.17
Hexa	-	<0.001	<0.00064	-	0.0025	0.0027	21	12	125	240	6.5
Hepta	-	<0.001	<0.0011	-	0.113	0.210	260	490	1730	500	110
Octa	-	0.049	0.0069	-	1.140	0.870	1810	2300	2388	1700	86
CDFs (ppb):											
Tetra	-	<0.001	<0.00044	-	<0.001	<0.00034	<0.2	<0.05	<0.1	<0.05	<0.04
Penta	-	<0.005	<0.00063	-	<0.01	<0.00051	0.4	2.3	3.8	9.3	<0.07
Hexa	-	<0.001	<0.0013	-	0.030	0.014	48	89	366	180	3.7
Hepta	-	<0.001	<0.0023	-	0.15	0.210	141	670	1047	600	12.0
Octa	-	<0.001	<0.0044	-	0.15	0.220	76	540	331	190	6.7

a/ Not Analyzed

* Compiled from SCE 1983, 1984, 1985a, 1985b, 1985c, 1985d

of both shallow and deep aquifers in 1984, and as shown in Table 6.3, the levels increased in 1985. Further investigation of soils from the site both at the surface and to a depth of eight inches detected significant levels of CDDs and CDFs, mostly hexa-, hepta-, and octa- isomer groups, with pentaCDFs also present.

Soil cores from the construction of additional monitoring wells in 1984 have provided data on the vertical distribution of PCP, creosote, CDDs and CDFs in the shallow aquifer (Table 6.4). The hexa-, hepta-, and octa- isomer groups again predominate. While no tetraCDDs or pentaCDDs were detected at any depth, tetraCDFs and pentaCDFs were present. The soil corings were taken from the location of the leaking tank, and began at a depth of 30.5 feet, as the contaminated soil above previously had been removed and replaced with clean fill. The aquitard material separating the two aquifers was also sampled at several locations at the site to determine if contaminants were able to penetrate this barrier. PCP, creosote, and low levels of hexa- through octaCDDs and penta- through octaCDFs (Table 6.4) were found within the aquitard under a location where treated poles had been stored.

Water recovered from both aquifers containing PCP, creosote, CDDs and CDFs has been discharged to the Visalia Water Conservation Plant since pumping began. PCP and creosote have been detected in the plant influent, effluent and sludge. CDDs and CDFs have also been detected in the influent, with the highest levels found in the sludge shown on Table 6.3. CDDs and CDFs have not been detected in the plant effluent.

Sludge from this plant is used as a soil amendment by farms and residents in the area, and a recent study (SCE, 1986) determined that sludge stockpiles at the distribution point contained CDDs and CDFs at levels similar to sludge from the water conservation plant. In this study sludge application rates ranged from 2.3 tons per acre to 259 tons per acre, and levels of CDDs and CDFs in the soils in these areas appeared to correlate with the application rate. While no tetraCDDs were found in the soil samples, tetraCDFs were present. Approximately 20 percent of the total tetraCDFs present were estimated to be the 2,3,7,8-tetraCDF isomer.

In 1985 a pretreatment system was installed at the site to remove contaminants from the extracted ground water before being received by the water conservation plant. The water is first passed through filters designed to trap CDD and CDF containing particulates, and then through carbon beds to remove PCP along with other organics. The system is designed to allow ground water to eventually be pumped directly into a nearby creek after treatment, bypassing the water conservation plant. Only trace levels of PCP (0.15 ppb) have been found after such treatment, with creosote, CDDs and CDFs not detected. A request based on this system's performance is before the Regional Water Quality Control Board to allow such a discharge (SCE, 1985c, 1986).

TABLE 6.4

VERTICAL DISTRIBUTION OF SOIL AND AQUIFER CONTAMINANTS AT VISALIA SITE[a]

Depth (ft)	PCP (ppm)	Creosote (ppm)	CDD (ppb)					CDF (ppb)				
			Tetra	Penta	Hexa	Hepta	Octa	Tetra	Penta	Hexa	Hepta	Octa
Soil Core Samples												
30.5	61	3700	<0.01	<0.08	1.2	68	460	1.3	<0.38	36	100	180
35.5	48	1500	<0.02	<0.05	0.3	30	320	<0.02	<0.04	13	36	60
40.5	120	ND[b]	<0.02	<0.06	7.0	340	1700	<0.02	0.33	49	455	200
40.5	5.9	ND	<0.02	<0.05	1.6	41	370	<0.01	<0.02	4.2	32	26
46.5	-[c]	-	<0.02	<0.06	4.7	100	720	<0.01	0.19	100	100	41
54.5	14	160	<0.12	<0.3	<0.33	2.8	63	<0.18	<0.27	<0.42	2.8	<2.2
Aquitard Profile Samples												
45.0	0.16	620	-	-	-	-	-	-	-	-	-	-
50.0	0.27	250	-	-	-	-	-	-	-	-	-	-
52.0	1.3	900	-	-	-	-	-	-	-	-	-	-
54.0	0.82	110	-	-	-	-	-	-	-	-	-	-
56.0	3.6	1100	-	-	-	-	-	-	-	-	-	-
58.0	8.0	2000	<0.0092	<0.036	2.4	216	327	<0.010	0.27	9.5	197	167

[a] Data from SCE, 1984, 1985a
[b] Not Detected
[c] Not Analyzed

Progress in removing contaminants from both aquifers has generally been good; in most cases a greater than 90 percent reduction of peak levels has been seen. Since about 1980, levels have been erratic from one analysis to the next, and the level of improvement somewhat uncertain, particularly for wells on the site. Contaminant levels in wells located further away from the site in the path of the plume have been more consistent, and do indicate a downward trend. The proposed level of clean up for ground water at the extraction wells before treatment is 1 ppm total phenols, 30 ppb for PCP, and below detection limits for creosote, CDDs and CDFs.

Bryan Hamel, *Dioxin Exposure Causes Transgenerational Health Effects*, National Institute of Environmental Health Sciences (Nov. 2012), https://factor.niehs.nih.gov/2012/11/science-dioxin/index.htm.

National Institute of Environmental Health Sciences

Environmental Factor, November 2012

Dioxin exposure causes transgenerational health effects

By Brant Hamel

A new study, funded in part by NIEHS, found that dioxin affects not only the health of an exposed rat, but also unexposed descendants through a mechanism of epigenetic transgenerational inheritance.

The study was conducted in the laboratory of Michael Skinner, Ph.D., a professor in the Center for Reproductive Biology in the Department of Biological Sciences at Washington State University (WSU) who designed the study. Co-authors included assistant research professor Mohan Manikkam, Ph.D., research technician Rebecca Tracey, and postdoctoral researcher Carlos Guerrero-Bosagna, Ph.D.

"Although not designed for risk assessment, these results have implications for the human populations that are exposed to dioxin and are experiencing declines in fertility and increases in adult onset disease, with a potential to transmit them to later generations," the authors concluded.

In a WSU press release, Skinner said of his latest findings, "It is not just the individuals exposed, but potentially the great-grandchildren that may experience increased adult-onset disease susceptibility." (Photo courtesy of Steve McCaw)

Dangers of dioxin last for decades after initial exposure

Dioxin, 2,3,7,8-tetrachlorodibenzo[p]dioxin (TCDD), is a chemical compound that constitutes part of the Agent Orange herbicide used as a defoliant in the Vietnam War. According to research cited in the study, exposure is estimated to have caused 400,000 deaths and 500,000 birth defects. Dioxin has also been released from industrial accidents, leading to human exposures. Due to its extremely long half-life of up to 10 years in humans, dioxin may still affect pregnancies occurring even 20 years after exposure.

In the Skinner group's experiments, exposure to dioxin caused changes in the DNA methylation patterns of sperm that were transmitted across generations, in an imprinted-like manner, to affect the health of multiple generations of descendents. The grandchildren of exposed rats showed dioxin-induced effects ranging from polycystic ovarian disease to kidney disease. The work raises the serious concern that even if toxic chemicals, such as dioxin, were completely removed from the environment, they could continue to cause disease for multiple generations.

Health effects of dioxin include early onset of puberty in females

Skinner's group used low *in vivo* doses of dioxin, so that toxic effects were not expected. Female rats were exposed while pregnant, and both their direct progeny and descendants two generations removed were examined.

Although the most prominent phenotypes were kidney disease in males and polycystic ovarian disease in females, a number of other effects including abscesses, colon impaction, lung abnormalities, and missing testes were also observed in animals from the dioxin-treated lineage. Additionally, females from the dioxin-exposed lineage experienced the early onset of puberty. Conversely, males showed delayed puberty, suggesting sex-specific effects of exposure. Early puberty in humans has increased over recent decades and is believed to have an environmental link.

Dioxin alters methylation patterns in germ line DNA across generations

The researchers were able to identify 50 specific regions of DNA that were differentially methylated in the dioxin-treated animals. These regions were permanently reprogrammed and protected from DNA methylation, in a manner that allowed them to be passed down across generations. In the future, these regions may serve as biomarkers that would allow early detection of exposure and risk for disease.

Other chemical compounds, including bisphenol A, phthalates, the insecticide DEET, and the jet fuel JP8 have all been shown to promote disease across generations, through a similar mechanism of epigenetic transgenerational inheritance (see story). This pathway of disease propagation exists not only in rats, but also in humans, mice, worms, flies, and even plants. Thus, future research will be needed to see if other environmental compounds may also lead to health effects across generations.

In addition to NIEHS, NIH and the U.S. Department of Defense provided support for the study.

Citation: Manikkam M, Tracey R, Guerrero-Bosagna C, Skinner MK. 2012. Dioxin (TCDD) induces epigenetic transgenerational inheritance of adult onset disease and sperm epimutations. PLoS One 7(9):e46249.

(Brant Hamel, Ph.D., is an Intramural Research Training Award fellow in the NIEHS Laboratory of Signal Transduction.)

Agency for Toxic Substances & Disease Registry, *Toxic Substances Portal - Trichloroethylene (TCE)*, U.S. Department of Health and Human Services, https://www.atsdr.cdc.gov/phs/phs.asp?id=171&tid=30 (as of Sept. 18, 2019).

ATSDR Agency for Toxic Substances & Disease Registry

Public Health Statement for Trichloroethylene

CAS#: 79-01-6

(/ToxProfiles/tp19-c1.pdf) **PDF Version, 63 KB** (/ToxProfiles/tp19-c1.pdf)

This Public Health Statement is the summary chapter from the Toxicological Profile for trichloroethylene (/ToxProfiles/TP.asp?id=173&tid=30). It is one in a series of Public Health Statements about hazardous substances and their health effects. A shorter version, the ToxFAQs™ (/toxfaqs/TF.asp?id=172&tid=30), is also available. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present. For more information, call the ATSDR Information Center at 1-800-232-4636.

Overview

This Public Health Statement summarizes the Agency for Toxic Substances and Disease Registry's (ATSDR) findings on trichloroethylene, including chemical characteristics, exposure risks, possible health effects from exposure, and ways to limit exposure.

The U.S. Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are sites targeted for long-term federal clean-up activities. The EPA has found trichloroethylene in at least 1,051 of the 1,854 current or former NPL sites. The total number of NPL sites evaluated for trichloroethylene is not known. But the possibility remains that as more sites are evaluated, the sites where trichloroethylene is found may increase. This information is important because these future sites may be sources of exposure, and exposure to trichloroethylene may be harmful.

If you are exposed to trichloroethylene, many factors determine whether you'll be harmed. These include how much you are exposed to (dose), how long you are exposed (duration), how often you are exposed (frequency), and how you are exposed (route of exposure). You must also consider the other chemicals you are exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

What is trichloroethylene?

Trichloroethylene is a colorless, volatile liquid. Liquid trichloroethylene evaporates quickly into the air. It is nonflammable and has a sweet odor.

The two major uses of trichloroethylene are as a solvent to remove grease from metal parts and as a chemical that is used to make other chemicals, especially the refrigerant, HFC-134a. Trichloroethylene has also been used as an extraction solvent for greases, oils, fats, waxes, and tars; by the textile processing industry to scour cotton, wool, and other fabrics; in dry cleaning operations; and as a component of adhesives, lubricants, paints, varnishes, paint strippers, pesticides, and cold metal cleaners.

What happens to trichloroethylene when it enters the enviornment?

Most of the trichloroethylene used in the United States is released into the atmosphere by evaporation, primarily from degreasing operations. Once in the atmosphere, the dominant trichloroethylene degradation process is reaction with hydroxyl radicals; the estimated half-life for this process is about 3–7 days. This relatively short half-life indicates that trichloroethylene is not a persistent atmospheric compound. Most trichloroethylene in surface waters or on soil surfaces evaporates into the atmosphere, although its high mobility in soil may result in it moving into groundwater below the soil surface. In these subsurface environments, trichloroethylene is only slowly degraded and may be relatively persistent. Trichloroethylene and other volatile organic chemicals may diffuse from contaminated groundwater and soil and migrate into air spaces beneath buildings to enter the indoor air, a process termed vapor intrusion.

How might I be exposed to trichloroethylene?

You may be exposed to trichloroethylene from trichloroethylene-contaminated air, water, food, or soil, or direct skin contact. You are most likely to be exposed to trichloroethylene by drinking trichloroethylene-contaminated water; you may also be exposed by breathing trichloroethylene released to the air from trichloroethylene-contaminated water. If you work in the degreasing industry or another industry where trichloroethylene is produced or used, you may be exposed by

breathing in trichloroethylene-contaminated air or by contacting the chemical with your skin. Some trichloroethylene is released to the air by its evaporation from products such as adhesives, paints, and coatings; and through its evaporation from trichloroethylene-contaminated soil at landfills.

You may also be exposed to trichloroethylene by consumption of trichloroethylene-contaminated foods, by contact with consumer products containing trichloroethylene, and by direct contact with trichloroethylene-contaminated soil.

How can trichloroethylene enter and leave my body?

Trichloroethylene can enter your body from trichloroethylene-contaminated air, water, food, or soil.

Trichloroethylene in air can easily enter your body when you breathe. Most of the trichloroethylene that you breathe in will go into your bloodstream and into other organs. A small amount of trichloroethylene in the air can also move through your skin and into your bloodstream.

When trichloroethylene is found in water, it can enter your body when you drink or touch the water or when you breathe in steam from the water. Most of the trichloroethylene that you breathe in or drink will move from your stomach or lungs into your bloodstream. When you touch water containing trichloroethylene (such as showering or bathing with trichloroethylene-contaminated water), some of it can get through your skin into your body. Also, you can be exposed when trichloroethylene in groundwater evaporates and migrates into air spaces beneath buildings to enter the indoor air, a process termed vapor intrusion, and you breathe that contaminated indoor air.

Trichloroethylene has been detected in table-ready foods at concentrations generally in the range of 2–100 ppb.

You can be exposed to trichloroethylene in soil when small amounts of soil are transferred to your mouth accidentally, when your skin touches the soil, or when you breathe air or dust coming from the soil. You can also be exposed when trichloroethylene in soil evaporates and migrates into air spaces beneath buildings to enter the indoor air, a process termed vapor intrusion, and you breathe that contaminated indoor air.

Once in your blood, your liver changes much of the trichloroethylene into other chemicals. When the body absorbs more trichloroethylene than it can break down quickly, some of the trichloroethylene or its breakdown products can be stored in body fat for a brief period. However, once absorption ceases, trichloroethylene and its breakdown products quickly leave the fat.

You will quickly breathe out much of the trichloroethylene that reaches your bloodstream; most of the trichloroethylene breakdown products leave your body in the urine within a day.

How can trichloroethylene affect my health?

The health effects of trichloroethylene depend on how much trichloroethylene you are exposed to and the length of that exposure. Environmental monitoring data suggest that trichloroethylene levels the public might encounter by direct contact or through air, water, food, or soil, are generally much lower than the levels at which adverse effects are elicited in animal studies. However, some drinking water sources and working environments have been found to contain levels of trichloroethylene that may cause health problems.

Trichloroethylene was once used as an anesthetic for surgery. People who are overexposed to moderate amounts of trichloroethylene may experience headaches, dizziness, and sleepiness; large amounts of trichloroethylene may cause coma and even death. Some people who breathe high levels of trichloroethylene may develop damage to some of the nerves in the face. Other effects seen in people exposed to high levels of trichloroethylene include evidence of nervous system effects related to hearing, seeing, and balance, changes in the rhythm of the heartbeat, liver damage, and evidence of kidney damage. Some people who get concentrated solutions of trichloroethylene on their skin develop rashes.

Relatively short-term exposure of animals to trichloroethylene resulted in harmful effects on the nervous system, liver, respiratory system, kidneys, blood, immune system, heart, and body weight.

Exposure to trichloroethylene in the workplace may cause scleroderma (a systemic autoimmune disease) in some people. Some men occupationally-exposed to trichloroethylene and other chemicals showed decreases in sex drive, sperm quality, and reproductive hormone levels.

Long-term exposure studies in animals have mainly focused on carcinogenicity and relatively insensitive noncancer end points following oral exposure; these studies are not helpful in defining noncancer end points in humans following long-term exposure. However, depressed body weight and evidence of effects on the thymus were reported in one recent study of mice exposed to trichloroethylene via their mothers during gestation and lactation and via the drinking water for up to 12 months thereafter.

There is strong evidence that trichloroethylene can cause kidney cancer in people and some evidence that it causes liver cancer and malignant lymphoma (a blood cancer). Lifetime exposure to trichloroethylene resulted in increased liver cancer in mice and increased kidney cancer in rats at relatively high exposure levels. There is some evidence for trichloroethylene-induced testicular cancer and leukemia in rats and lymphomas and lung tumors in mice.

The Department of Human Health Services (HHS) has classified trichloroethylene as "*known to be a human carcinogen*" based on sufficient evidence of carcinogenicity from humans. Similarly, the International Agency for Research on Cancer (IARC) has classified it as "carcinogenic to humans" and EPA has characterized it as "carcinogenic in humans by all routes of exposure." These agencies concluded that there were sufficient evidence from human studies that trichloroethylene exposure can cause kidney cancer in humans. There is also some evidence of an association between trichloroethylene exposure and non-Hodgkin's lymphoma in humans.

How can trichloroethylene affect children?

This section discusses potential health effects of trichloroethylene exposure in humans from when they're first conceived to 18 years of age.

Trichloroethylene is expected to affect children in the same manner as adults. It is not known whether children are more susceptible than adults to the effects of trichloroethylene.

Some human studies indicate that trichloroethylene may cause developmental effects such as spontaneous abortion, congenital heart defects, central nervous system defects, and small birth weight. However, these people were exposed to other chemicals as well. In some animal studies, exposure to trichloroethylene during development may have caused effects such as decreased body weight, increased incidences of heart defects, functional or structural changes in the developing nervous system, and effects on the immune system.

How can families reduce the risk of exposure to trichloroethylene?

If your doctor finds that you have been exposed to significant amounts of trichloroethylene, ask whether your children might also be exposed. Your doctor might need to ask your state health department to investigate. You may also contact the state or local health department with health concerns.

Exposure to contaminated drinking water should be limited. Trichloroethylene has been detected in some drinking water supplies. For bottled water, consumers should contact the bottler with specific questions on potential contaminants.

If you live near an industrial site where trichloroethylene is produced or is a byproduct or you live near a hazardous waste site where it has been discarded, there may be high levels of trichloroethylene in the water and soil. If you find your home water supply and/or soil to be contaminated with trichloroethylene, consider using a cleaner source of water and limiting contact with soil (for example, through use of a dense ground cover or thick lawn) to reduce exposure to trichloroethylene. By paying careful attention to dust and dirt control in the home (air filters, frequent cleaning), you can reduce family exposure to contaminated dirt. Some children eat a lot of dirt. You should prevent your children from eating dirt. You should discourage your children from putting objects in their mouths. Make sure that they wash their hands frequently and before eating. Discourage your children from putting their hands in their mouths or from other hand-to-mouth activity.

Trichloroethylene is widely used as a solvent for extraction, waterless drying, and finishing, and as a general purpose solvent in adhesives, lubricants, paints, varnishes, paint strippers, pesticides, and cold metal cleaners. Follow instructions on product labels to minimize exposure to trichloroethylene.

Are there medical tests to determine whether I have been exposed to trichloroethylene?

Trichloroethylene and its breakdown products (metabolites) can be measured in blood and urine. Because trichloroethylene and its metabolites leave the body fairly rapidly, the tests need to be conducted within a few hours after exposure. Tests for trichloroethylene and its metabolites in the blood or urine require special analytical equipment not readily available at medical facilities. Some metabolites of trichloroethylene can be formed from chemicals other than trichloroethylene, so detection of these metabolites in blood or urine does not guarantee that one has been exposed to trichloroethylene.

For more information on the different substances formed by trichloroethylene breakdown and on tests to detect these substances in the body, see Chapters 3 and 7.

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

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the Environmental Protection Agency (EPA),

the Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA). Recommendations provide valuable guidelines to protect public health but are not enforceable by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Institute for Occupational Safety and Health (NIOSH).

Regulations and recommendations can be expressed as "not-to-exceed" levels; that is, levels of a toxic substance in air, water, soil, or food that do not exceed a critical value usually based on levels that affect animals; levels are then adjusted to help protect humans. Sometimes these not-to-exceed levels differ among federal organizations. Different organizations use different exposure times (e.g., an 8-hour workday or a 24-hour day), different animal studies, or emphasize some factors over others, depending on their mission.

Recommendations and regulations are also updated periodically as more information becomes available. For the most current information, check with the federal agency or organization that issued the regulation or recommendation.

EPA set a maximum contaminant level goal (MCLG) of zero as a national primary drinking standard for trichloroethylene; EPA noted liver problems and increased risk of cancer as potential health effects from long-term exposure above the maximum contaminant level (MCL) of 0.005 milligrams per liter (mg/L; 5 ppb).

OSHA set a permissible exposure limit (PEL) of 100 ppm for trichloroethylene in air averaged over an 8-hour work day, an acceptable ceiling concentration of 200 ppm provided the 8-hour PEL is not exceeded, and an acceptable maximum peak of 300 ppm for a maximum duration of 5 minutes in any 2 hours.

NIOSH considers trichloroethylene to be a potential occupational carcinogen and established a recommended exposure limit (REL) of 2 ppm (as a 60-minute ceiling) during the usage of trichloroethylene as an anesthetic agent and 25 ppm (as a 10-hour time-weighted average [TWA]) during all other exposures.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2019 Toxicological Profile for Trichloroethylene (/ToxProfiles/TP.asp?id=173&tid=30). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

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 Human Health Sciences
1600 Clifton Road NE, Mailstop S102-1
Atlanta, GA 30333
Phone: 1-800-CDC-INFO · 888-232-6348 (TTY)
Email: Contact CDC-INFO (http://www.cdc.gov/cdc-info/requestform.html)

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.

- Page last reviewed: January 21, 2015
- Page last updated: January 21, 2015
- Content source: Agency for Toxic Substances and Disease Registry (http://www.atsdr.cdc.gov/)

EPA, *Learn About Dioxin*, https://www.epa.gov/dioxin/learn-about-dioxin (as of Sept. 18, 2019).

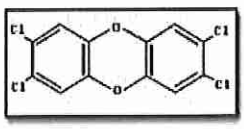
EPA United States Environmental Protection Agency

Learn about Dioxin

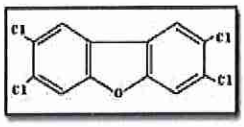
Dioxin Key Facts

- Dioxins are called persistent organic pollutants (POPs), meaning they take a long time to break down once they are in the environment.
- Dioxins are highly toxic and can cause cancer, reproductive and developmental problems, damage to the immune system, and can interfere with hormones.
- Dioxins are found throughout the world in the environment and they accumulate in the food chain, mainly in the fatty tissue of animals
- More than 90% of human exposure is through food, mainly meat and dairy products, fish and shellfish.

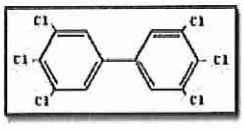
Figure 1: Dioxin chemical structures



2,3,7,8-Tetrachlorodibenzo-p-dioxin



2,3,7,8-Tetrachlorodibenzofuran



3,3',4,4',5,5'-Hexachlorobiphenyl

What is Dioxin?

Dioxins refers to a group of toxic chemical compounds that share certain chemical structures and biological characteristics (see figure 1). Several hundred of these chemicals exist and are members of three closely related families:

- Chlorinated dibenzo-p-dioxins (CDDs),
- Chlorinated dibenzofurans (CDFs) and
- Certain polychlorinated biphenyls (PCBs).

CDDs and CDFs are not created intentionally, but are produced as a result of human activities like the backyard burning of trash. Natural processes like forest fires also produce CDDs and CDFs. PCBs are manufactured products, but they are no longer produced in the United States.

Most Toxic Dioxin

The most studied and most toxic of all dioxins is **2,3,7,8-Tetrachlorodibenzo-p-dioxin.** In 2012, EPA released an updated IRIS assessment on this chemical.

What does dioxin look like?

Dioxin looks like white crystalline needles.

Where does dioxin come from?

Industrial activities: Dioxin is not produced or used commercially in the United States. It is a contaminant formed in the production of some chlorinated organic compounds, including a few herbicides such as silvex. Over the past decade, EPA and industry have been working together to dramatically reduce the production of dioxin in the environment.

However it should be noted that though levels have decreased in the last 30 years, dioxins are extremely persistent compounds and break down very slowly. In fact, a large part of the current exposures to dioxins in the US is due to releases that occurred decades ago.

Even if all human-generated dioxins were eliminated, low levels of naturally produced dioxins would remain. EPA is working with other parts of the government to look for ways to further reduce dioxin levels entering the environment and to reduce human exposure to them.

Other ways dioxins are produced:

Burning: Dioxins are formed as a result of combustion processes such as waste incineration (commercial or municipal) or from burning fuels (like wood, coal or oil).

> Dioxins are formed as a result of combustion processes such as ... burning fuels like wood, coal or oil.

EPA's 2006 Dioxin Inventory of Sources Report summarizes that man-made emissions dominate current releases in the US, but acknowledges the need for more data on natural sources. You may not be aware that dioxins can also be formed when household trash is burned or from events like forest fires.

- Dioxins produced by backyard burning

Bleaching: Chlorine bleaching of pulp and paper, based on certain types of chemical manufacturing and processing, and other industrial processes this can create small quantities of dioxins in the environment.

Smoking: Cigarette smoke also contains small amounts of dioxins.

Drinking Water: Dioxin can get into drinking water from:

- emissions from waste incineration and other combustion that get deposited into bodies of water; and
- discharges into water from chemical factories.

Learn more about dioxin in drinking water from the table of regulated drinking water contaminants.
EPA's private drinking water wells

How can dioxin affect my health?

Dioxins are highly toxic and can cause cancer, reproductive and developmental problems, damage to the immune system, and can interfere with hormones.

Related Resources

- More information about common sources of exposure
- FDA's Chemical Contaminants: Dioxin
- Questions and answers about dioxin and food safety - Dioxin Related Activities (Feb 2012) (Joint FDA and EPA)

LAST UPDATED ON JULY 15, 2019

National Institute of Environmental Health Sciences, *Dioxin*, https://www.niehs.nih.gov/health/topics/agents/dioxins/ (as of Sept. 18, 2019).

National Institute of Environmental Health Sciences
(https://www.niehs.nih.gov)

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Dioxins

Introduction

Dioxins are mainly byproducts of industrial practices. They are produced through a variety of incineration processes, including improper municipal waste incineration and burning of trash, and can be released into the air during natural processes, such as forest fires and volcanoes. Almost every living creature has been exposed to dioxins or dioxin-like compounds (DLCs).

Strict regulatory controls on major industrial sources of dioxin have reduced emissions into the air by 90 percent, compared to levels in 1987.

Today people are exposed to dioxins primarily by eating food, in particular animal products, contaminated by these chemicals. Dioxins are absorbed and stored in fat tissue and, therefore, accumulate in the food chain. More than 90 percent of human exposure is through food.

Before safeguards and regulations were introduced, dioxin releases were a major problem in the United States. The Polychlorinated Biphenyls (PCBs) (https://www.epa.gov/pcbs/learn-about-polychlorinated-biphenyls-pcbs) worked with industry to ban products containing dioxin and to curb dioxin emissions. In 1979, the EPA banned the manufacture of products containing Polychlorinated Biphenyls (PCBs) (https://www.epa.gov/pcbs/learn-about-polychlorinated-biphenyls-pcbs) some of which are included under the term dioxin.

Consumers should eat a balanced diet and follow the 2010 Dietary Guidelines for Americans. Each food group provides important nutrients needed for health.

The following steps can reduce the potential for exposure to dioxin:

- Remove skin from fish and chicken
- Select cuts of meat that are naturally lean, or trim visible fat
- When catching your own fish, check local fishing advisories, as there may be consumption limits for particular kinds of fish, in particular bodies of water where local contamination has occurred
- Use fat-free or low-fat milk and use butter in moderation

However, dioxins break down very slowly and emissions released long ago remain in the environment. Some dioxins endure a long time, are extremely resistant to environmental degradation, and therefore are classified as persistent organic pollutants (POPs). Dioxin contamination is an increasing problem in some developing countries, particularly with uncontrolled burning, and dismantling and recycling of electronic products, such as computers.

Health Effects

The dioxin TCDD, or Mutagen: Talking Glossary of Genetic Terms *(https://www.genome.gov/genetics-glossary/Mutagen)*, is a known cancer-causing agent, and other DLCs are known to cause cancer in laboratory animals. Additionally, dioxin exposure has been linked to a number of other diseases, including type 2 diabetes, ischemic heart disease, and an acne-like skin disease called chloracne, a hallmark of dioxin exposure.

Dioxins can cause developmental problems in children, lead to reproductive and infertility problems in adults, result in miscarriages, damage the immune system, and interfere with hormones.

Exposure to dioxins has widespread effects in nearly every vertebrate species, at nearly every stage of development, including in the womb.

The Science of Dioxins

Dioxins are a family of compounds that share distinct chemical structures and characteristics. Numerous dioxin-like compounds have been identified that are considered to have significant toxicity and can cause disease. The singular term dioxin refers to the most toxic compound, TCDD.

NIEHS researchers continue to explore the detailed chemical pathway through which dioxin damages the body, but scientists are now confident that the first step takes place when dioxin binds to an intracellular protein known as the aryl hydrocarbon receptor (AhR). When that happens, the AhR can alter the expression, or function, of certain genes. The resulting cellular imbalance leads to a disruption in normal cell function and ultimately adverse health effects.

In addition to TCDD, many other chemicals bind to AhR. About 400 compounds in the environment act on the body through the AhR receptor. Public health officials around the world are concerned about the combined effects of multiple chemicals that activate the AhR, and are developing health standards that take into account the fact that people are exposed to mixtures of DLCs, not just one at a time.

Dioxins' Impact

The public health threats posed by dioxins were highlighted dramatically in the public consciousness in the late 1970s and early 1980s. Newspapers and television broadcasts were full of stories about ailing veterans who had been exposed to dioxins through Agent Orange, an herbicide and defoliant used in the Vietnam War.

Concerns about Agent Orange and other DLCs continue today. Research supported by NIEHS and many others, examining the link between dioxin and serious illnesses, has helped lead the U.S. Department of Veterans Affairs (VA) *(https://www.va.gov/)* to recognize certain cancers and other health problems as presumptive diseases associated with exposure to Agent Orange or other herbicides during military service. Presumptive diseases are certain diseases that the VA assumes can be related to a Veteran's qualifying military service.

Dioxins were also brought to light in 1982, when the town of Times Beach, Mo., was declared off-limits, because of dioxin contamination. This incident in Missouri, as well as others, helped spark passage of legislation that created Superfund, the environmental program established to address abandoned hazardous waste sites.

In addition to funding work in labs across the nation, NIEHS administers the Superfund Research Program (SRP). SRP involves a network of university grants that are designed to seek solutions to the complex health and environmental issues associated with the nation's hazardous waste sites.

The research conducted by the SRP is a coordinated effort with the EPA, the federal entity charged with cleaning up the worst hazardous waste sites in the country, including those contaminated with dioxins.

Today, the hazards posed by dioxins have faded from public view. And, in fact, the extent of the hazard has diminished in the U.S., as environmental controls significantly reduced the introduction of new industrial sources of dioxin.

However, the problem has not vanished, and the scientific community has continued its work to reduce exposures and treat diseases that arise from them.

Further Reading

Stories from the *Environmental Factor* (NIEHS Newsletter)

- Solving a Mystery *(https://factor.niehs.nih.gov/2014/4/science-mystery/index.htm)* (April 2014)
- Birnbaum Talks Science and Strategy at Dioxin 2012 *(https://factor.niehs.nih.gov/2012/10/science-dioxin/index.htm)* (October 2012)
- Dioxin Exposure Cause Transgenerational Health Effects *(https://factor.niehs.nih.gov/2012/11/science-dioxin/index.htm)* (November 2012)
- Seminar Addresses Developmental Origins of Immune Disease *(https://factor.niehs.nih.gov/2012/6/science-immune/index.htm)* (June 2012)

Additional Resources

- Dioxin Mixtures Research *(https://ntp.niehs.nih.gov/ntp/htdocs/LT_rpts/tr531.pdf)* - by the National Toxicology Program (NTP) at NIEHS
- Applying 21st Century Toxicology to Green Chemical and Material Design *(http://nas-sites.org/emergingscience/workshops/green-chemistry/workshop-presentations-green-chemistry/)* - Workshop Presentations from the National Academies Standing Committee on Use of Emerging Science for Environmental Health Decisions

Content courtesy of the US Centers for Disease Control and Prevention (CDC)

Dioxins, Furans, PCBs (contain phenyl rings of carbon atoms)

Dioxins, furans, and polychlorinated biphenyls (PCBs) are a class of similar chlorinated aromatic organic compounds. Dioxins have two phenyl rings connected by two oxygen atoms. Furans have one or two phenyl rings connected to a furan ring. PCBs have two phenyl rings attached at one point. One or more chlorine atoms can attach to any available carbon atom, allowing for 100 - 200 forms of each. Dioxins and dioxin-like furans have no known commercial or natural use. They are produced primarily during the incineration or burning of waste; the bleaching processes used in pulp and paper mills; and the chemical syntheses of trichlorophenoxyacetic acid, hexachlorophene, vinyl chloride, trichlorophenol, and pentachlorophenol. PCBs were once synthesized for use as heat-exchanger, transformer, and hydraulic fluids, and also used as additives to paints, oils, window caulking, and floor tiles. Production of PCBs peaked in the early 1970s and was banned in the United States after 1979.

Substances Listing

- Chlorinated Dibenzo-p-dioxins (CDDs)
- Chlorodibenzofurans (CDFs)
- Polybrominated Biphenyls (PBBs)
- Polybrominated Diphenyl Ethers (PBDEs)
- Polychlorinated Biphenyls (PCBs)

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Related Health Topics

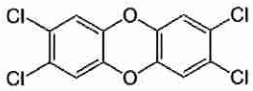
- Cancer
- Toxicology

File Assistance: Downloads for viewing files

Adobe Reader *(https://get.adobe.com/reader/)*

California Office of Environmental Health Hazard Assessment, *2,3,7,8-Tetrachlorodibenzo-p-dioxin and Related Compounds*, https://oehha.ca.gov/chemicals/2378-tetrachlorodibenzo-p-dioxin-and-related-compounds (as of Sept. 18, 2019).

2,3,7,8-Tetrachlorodibenzo-p-dioxin and related compounds



CAS Number

1746-01-6

Synonym(s)

Dioxin, TCDD

Occurence(s)/Use(s)

Byproduct of manufacture of chlorophenols, hexachlorophene, and herbicides; contaminant of the Agent Orange, PCBs, and pentachlorophenol; component of combustion, including waste incineration and tobacco smoke.

Soil Screening

Last CHHSL revision:	1/2005
CHHSL Comments:	For chemical-specific screening levels for use in assessing contaminated sites, please see HHRA Note 3 (DTSC HERO). https://www.dtsc.ca.gov/assessingrisk/humanrisk2.cfm

Cancer Potency Information

Inhalation Unit Risk (µg/cubic meter)-1:	3.8 E+1
Inhalation Slope Factor (mg/kg-day)-1:	1.3 E+5
Oral Slope Factor (mg/kg-day)-1:	1.3 E+5
Comments/References:	OEHHA 2009. Air Toxics Hot Spots Program Technical Support Document for Cancer Potencies. Appendix B. Chemical-specific summaries of the information used to derive unit risk and cancer potency values. Updated 2011.

Air

Acute RELs

Last Acute REL Revision:	12/01/2008

Hot Spots Unit Risk and Cancer Potency values:
Unspeciated mixtures treated as 2,3,7,8-tetrachlorodibenzo-p-dioxin (1746-01-6)

Chronic REL

Chronic Inhalation REL (µg/m3):	0.00004 (inhalation); 0.00001 µg/kg BW-day (oral)
Chronic Target Organs:	Inhalation and oral: Alimentary system (liver); reproductive system; development; endocrine system; respiratory system; hematologic system
Human Data:	No
Last Chronic REL Revision:	12/01/2008
Chronic Reference Exposure Levels Comments:	OEHHA 2008. Technical Supporting Document for Noncancer RELs, Appendix D3

Proposition 65

Chemical Status

Cancer:	Currently listed
Reproductive Toxicity:	Currently listed

Cancer

Listed as causing:	Cancer
Date of Listing:	01/01/1988
Basis for Listing:	SQE

Reproductive Toxicity

Listed as causing:	Developmental Toxicity

Developmental Toxicity

Date of Listing:	04/01/1991
Basis for Listing:	AB-US EPA

Safe Harbor Levels

Cancer

No Significant Risk Level (NSRL):	0.000005 µg/day

Documents, Presentations, and Publications

Cancer:	OEHHA 2013. Proposition 65 NSRLs and MADLs Aug 15, 2013

Public notices related to this chemical:

- 2012 Priority List for the Development of Proposition 65 NSRLs for Carcinogens and MADLs for Chemicals Causing Reproductive Toxicity

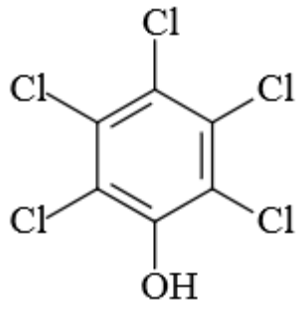
Water

California Public Health Goals Data

Health Risk Category:	Carcinogenicity
Public Health Goal (mg/L):	0.05 picograms/L (pg/L)
Public Health Goal - Download:	Public Health Goal for TCDD (Dioxin) in Drinking Water
Cancer Risk at PHG:	0.000001
MCL value (mg/L):	0.00000003
Cancer Risk at MCL:	0.0006
Last PHG Revision :	2010
California PHG Comments:	The Public Health Goal for TCDD (Doxin) is 0.00005 nanograms/liter (ng/L) or 0.05 picograms/liter (pg/L).

California Office of Environmental Health Hazard Assessment, *Pentachlorophenol*, https://oehha.ca.gov/chemicals/ pentachlorophenol (as of Sept. 18, 2019).

Pentachlorophenol



CAS Number

87-86-5

Synonym(s)

AI3-00134; Caswell No. 641; Chlorophen; 1-hydroxypentachlorobenzene; NCI-C5933; NCI-C55378; NCI-C56655; PCP; Pentachlorofenol; Pentachlorol; Pentachlorophenate; 2,3,4,5,6-pentachlorophenol; Pentanol; EPA Pesticide Code: 063001

Occurence(s)/Use(s)

Wood preservative in specified outdoor applications, such as utility poles. No longer registered as a pesticide in California.

Child-Specific Reference Dose

Child-specific refence dose (chRD) (mg/kg-day, unless noted):	1.0 E-3
Last chRD revision:	06/2006
chRD Comments:	OEHHA 2006. Child-Specific Reference Doses (chRDs) for School Site Risk Assessment: Manganese and Pentachlorophenol.

Soil Screening

Last CHHSL revision:	1/2005
CHHSL Comments:	For chemical-specific screening levels for use in assessing contaminated sites, please see HHRA Note 3 (DTSC HERO). https://www.dtsc.ca.gov/assessingrisk/humanrisk2.cfm

Cancer Potency Information

Latest Criteria:	California Public Health Goals
Inhalation Unit Risk (µg/cubic meter)-1:	5.1 E-6
Inhalation Slope Factor (mg/kg-day)-1:	1.8 E-2
Oral Slope Factor (mg/kg-day)-1:	8.1 E-2

Comments/References:	OEHHA 2009. Air Toxics Hot Spots Program Technical Support Document for Cancer Potencies. Appendix B. Chemical-specific summaries of the information used to derive unit risk and cancer potency values. Updated 2011. OEHHA 2009. Public Health Goal for Pentachlorophenol in Drinking Water.

Air

Proposition 65

Chemical Status

Cancer:	Currently listed

Cancer

Listed as causing:	Cancer
Date of Listing:	01/01/1990
Basis for Listing:	AB-NTP AB-US EPA

Safe Harbor Levels

Cancer	
No Significant Risk Level (NSRL):	40 µg/day

Documents, Presentations, and Publications

Cancer:	OEHHA 2013. Proposition 65 NSRLs and MADLs Aug 15, 2013

Public notices related to this chemical:

- December 18, 2001 Meeting of the Science Advisory Board's Carcinogen Identification Committee (CIC)

Water

California Public Health Goals Data

Health Risk Category:	Carcinogenicity
Public Health Goal (mg/L):	0.0003
Public Health Goal - Download:	Public Health Goal for Pentachlorophenol in Drinking Water (2009)
Cancer Risk at PHG:	0.000001
MCL value (mg/L):	0.001
Cancer Risk at MCL:	0.000003

Lori A. Verbrugge et al., *Pentachlorophenol, Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzo Furans in Surface Soil Surrounding Pentachlorophenol-treated Utility Poles on the Kenai National Wildlife Refuge, Alaska USA*, 25,19 Envtl. Science and Pollution Res. Int'l (2018), pp. 19187-19195, available at https://www.ncbi.nlm.nih.gov/ pmc/articles/PMC6061508/.

Springer
Environmental Science and Pollution Research International
springer.com

Environ Sci Pollut Res Int. 2018; 25(19): 19187–19195.
Published online 2018 Jun 1. doi: 10.1007/s11356-018-2269-7
PMCID: PMC6061508
PMID: 29858999

Pentachlorophenol, polychlorinated dibenzo-p-dioxins and polychlorinated dibenzo furans in surface soil surrounding pentachlorophenol-treated utility poles on the Kenai National Wildlife Refuge, Alaska USA

Lori A. Verbrugge,[1] Lynnda Kahn,[2] and John M. Morton[2]

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Responsible editor: Zhihong Xu

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Abstract

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Introduction

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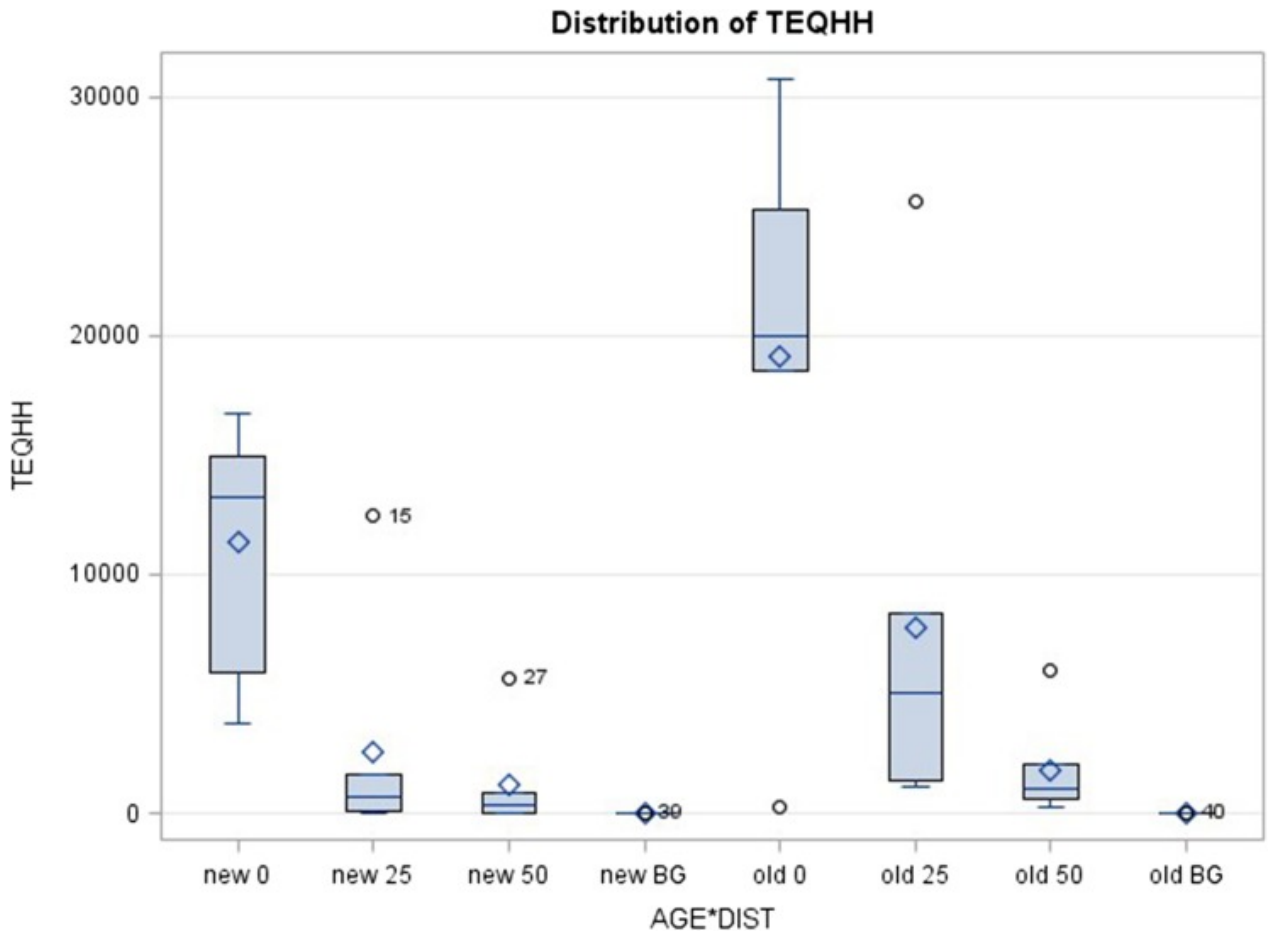
Methods



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Discussion

Conclusions and recommendations

Acknowledgements

Compliance with ethical standards

Disclaimer

References

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Exhibit B

Full Report available at
https://www.envirostor.dtsc.ca.gov/public/deliverable_documents/8077635049/Groundwater%20Monitoring%20Report%2C%20May%202017%20%5BSGI%207-19-17%5D.pdf

SECOND QUARTER 2017
GROUNDWATER MONITORING REPORT

McNamara and Peepe Lumber Mill
1619 and 1678 Glendale Drive
Arcata, California

01-DTSC-006

Prepared For:

California Department of Toxic Substances Control
700 Heinz Avenue
Berkeley, California 94710

Contract No. 14-T3913

Prepared By:

The Source Group, Inc., A division of Apex Companies, LLC.
3478 Buskirk Avenue, Suite 100
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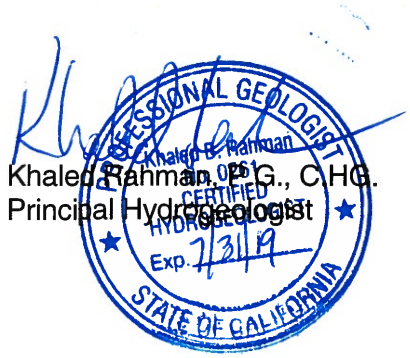
July 19, 2017



Khaled Rahman, P.G., C.HG.
Principal Hydrogeologist

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1.0 INTRODUCTION

The Source Group, Inc., a division of Apex Companies, LLC. (SGI-Apex), has prepared this *Second Quarter 2017 Groundwater Monitoring Report* (Report) for the McNamara and Peepe Lumber Mill located at 1619 and 1678 Glendale Drive in Arcata, California (hereinafter the Site, Figure 1). This Report and the scope of work presented herein were conducted for the California Environmental Protection Agency (Cal/EPA), Department of Toxic Substances Control (DTSC) under Contract No. 14-T3913.

This Report presents the results of the groundwater monitoring and sampling event conducted at the Site on May 8, 2017. The field activities were conducted in general accordance with the *Soil and Groundwater Investigation Work Plan* (Work Plan; URS Corporation [URS], 2011). This Report summarizes the monitoring and sampling field activities, laboratory analytical results for pentachlorophenol (PCP) and 2,3,4,6-tetrachlorophenol (TCP), water quality parameters, and quality assurance protocols. In response to a DTSC request, the concrete slab at the "new" dip tank building (Dip Tank Building), which is located at 1678 Glendale Drive, was also inspected during the May 2017 event.

2.0 SITE BACKGROUND

The Site is a former lumber mill located in an unincorporated area of Humboldt County, approximately one mile southeast of McKinleyville, California and five miles northeast of Arcata, California. The Site operated as a lumber mill under multiple owners from the 1940s until 2002 (URS Corporation [URS], 2011). A detailed summary of background information for the Site is presented in a *Five-Year Comprehensive Review* prepared by the DTSC (DTSC, 2014). The following section provides a brief overview of the Site.

2.1 Site and Vicinity Description

As shown on Figure 2, the Site totals approximately 21 acres located north and south of Glendale Drive (DTSC, 2014). The northern portion of the Site is located at 1619 Glendale Drive (Assessor Parcel Numbers [APNs]: 516-111-062 and 516-111-063) and consisted of the former Green Chain area, Saw Mill, Planer Chain, and a groundwater production well (URS, 2011). The 1619 Glendale Drive portion of the Site is currently leased to Royal Gold for storage and distribution of potting soil and compost. The southern portion of the Site is located at 1678 Glendale Drive (APNs: 516-151- 003 and 516-151-004) and is the location of the Dip Tank Building. Based on the findings of our Site walk (see below), the 1678 Glendale Drive portion of the Site is currently owned by Gary Johnson and is used for equipment and vehicle maintenance storage.

The Site is surrounded by residential and commercial/light industrial properties to the west, north, east and south. The Mad River is located approximately 0.25 miles south of the Site.

2.2 Geological and Hydrogeological Setting

The Site is located in the Dows Prairie Subbasin, which is the northern portion of the Mad River Groundwater Basin (California Department of Water Resources [DWR], 2004). The Hookton Unit is the primary water-bearing unit in the Dows Prairie Subbasin and underlain by the Franciscan Formation (DWR, 2004). The Hookton Unit consists of fine-grained (clay) and coarse-grained (sand and gravel) intervals that are approximately 150-200 feet in depth (DWR, 2004).

Previous investigations conducted at the Site indicate that the shallow subsurface consists of alluvial and terrace deposits composed of fine-grained silts and clays, and coarse-grained sands and gravels. Based on previous investigations, groundwater was measured at depths of approximately 8.0 feet below ground surface (bgs) to 30 feet bgs and generally flows to the south-southwest toward the Mad River (URS, 2011).

2.3 Historic Land Use

McNamara and Peepe operated the lumber mill from 1969 until they filed for bankruptcy in 1985 (DTSC, 2014). Chemical fungicides containing PCP and TCP were applied to processed lumber at the Site in dip tanks or with spray applications from 1967 to 1984 (URS, 2011). Dip tanks were present near the Green Chain area on the 1619 Glendale Drive portion of the Site (Figure 3), and in

the Dip Tank Building on the 1678 Glendale Drive portion of the Site (Figure 2). Spray applications were conducted at the Planer Chain building (Figure 2). During this period, several incidents of improper storage, spills, and leaks are documented (DTSC, 2014). Blue Lake Forest Products leased and operated the mill without the use of PCP and TCP from 1986 until lumber mill operations ceased at the Site in 2002 (DTSC, 2014).

2.4 Regulatory Oversight

Regulatory oversight of the Site was conducted by the North Coast Regional Water Quality Control Board (NCRWQCB) from 1968 to 1984 and included establishment of waste discharge requirements (WDRs) for the Site (URS, 2011). In 1982, NCRWQCB adopted WDRs, issued a Cease-and Desist Order (Order No. 82-3; the Order), required the lumber mill operator to cease discharge of fungicide wastes, determine the source of the discharge, prepare a plan for eliminating discharges, and implement the plan according to the schedule outlined in the Order (URS, 2011). DTSC became the lead oversight agency for the Site in 1984 and issued a Remedial Action Order (RAO; No. 88/89-023), which was amended in 1996 (No. 95/96-072). In 2008, DTSC issued an Imminent and Substantial Endangerment Determination (ISED No. 07/08-009; DTSC, 2008).

2.5 Remedial Activities

DTSC approved a *Remedial Action Plan* (RAP) for the Site in 1994 (DTSC, 2014). The former Green Chain area and former Saw Mill building were identified as the source area for PCP and TCP in soil and groundwater (Figure 3). A concrete cap over the Green Chain area was selected as a remedy for the Site and was constructed in 1998 (DTSC, 2014). A land use covenant (LUC) was issued in 1998 to restrict use in two areas of the Site: the “Cap Restricted Area” on the former lumber mill property located at 1619 Glendale Drive and the “Concrete Slab Restricted Area” located in the Dip Tank Building located at 1678 Glendale Drive. Routine assessments of the concrete cap indicate the condition of the cap was excellent. Since construction of the concrete cap, the former Saw Mill building has been demolished.

Elevated PCP concentrations (>1,100 micrograms per liter [µg/L]) in groundwater were detected in Site monitoring wells during the 2003 annual monitoring event. A remedial investigation (RI) was conducted in 2005 to evaluate the source of the elevated PCP concentrations in groundwater (DTSC, 2014). The RI concluded that dissolution of PCP and TCP from soil into groundwater was due to a rise in groundwater elevations of up to 15 feet across the Site since 2001. The rise in groundwater elevations was attributed to cessation of groundwater extraction from production well PW-1 in the northern portion of the Site in 2002 (DTSC, 2014).

2.6 Groundwater Monitoring Well Network

The groundwater monitoring well network consists of wells MW-1, MW-5, MW-7, MW-8, MW-9, MW-11, and MW-12, which are located at 1619 Glendale Avenue, and well MW-10 offsite on Glendale Avenue (Figure 3). As summarized on the table below, well construction details indicate that the monitoring wells are screened to maximum depths of 25 feet bgs, except for well MW-7,

which is screened from 22 feet bgs to 37 feet bgs. Readily available groundwater monitoring well logs are included in Appendix A.

Well Name	**TOC** (feet amsl)	**Screened Interval** (feet btoc)
MW-1	90.92	19-23
MW-5	93.25	18-23
MW-7	98.90	22-37
MW-8	96.04	8.5-24
MW-9	99.65	21-25
MW-10	95.65	9-24
MW-11	91.70	9.5-24.5
MW-12	91.73	10-20

Notes:
TOC = top of casing
amsl = above mean seal level
btoc = below top of casing

2.7 Recent and Planned Activities

Groundwater monitoring events conducted in December 2016 were documented in the *Fourth Quarter 2016 Groundwater Monitoring Report*, which included supplemental analytical results collected to support an evaluation of remedial alternatives (SGI-Apex, 2017). A remedial alternative evaluation for PCP and TCP in groundwater is in preparation.

3.0 GROUNDWATER MONITORING ACTIVITIES

3.1 Groundwater Monitoring Wells

On May 8, 2017, eight (8) groundwater monitoring wells (MW-1, MW-5, MW-7, MW-8, MW-9, MW-10, MW-11, and MW-12) were gauged and sampled. Field data forms are included in Appendix B.

3.2 Groundwater Sampling Activities

Groundwater sampling activities were completed in accordance with the Groundwater Monitoring Well Low Flow Sampling Standard Operating Procedure (SOP-005) included in Appendix D of the Work Plan (URS, 2011). No deviations from the SOP were noted. Sampling activities consisted of the following:

- Depth to groundwater and total depth were gauged in each monitoring well to the nearest 0.01 foot using an electronic water level indicator;
- Low-flow sampling methods were used to collect samples from groundwater monitoring wells. Well purging and water quality parameters (pH, temperature, specific conductance, dissolved oxygen [DO], and oxidation-reduction potential [ORP], turbidity, and total dissolved solids [TDS]) using a water quality meter were recorded on groundwater sampling forms (Appendix B);
- One duplicate sample was collected from well MW-1 for quality assurance/quality control (QA/QC) purposes;
- Sample containers provided by the analytical laboratory were labeled with a unique sample identification number consistent with previous sampling events (e.g., MW-1), date and time of sample collection, sampler, preservation, and analytical method; and
- Samples were submitted to North Coast Laboratories of Arcata, California, a California State Environmental Laboratory Accreditation Branch (CA ELAP)-certified laboratory under standard chain-of-custody protocols.

3.3 Laboratory Analysis

Groundwater samples were analyzed for:

- PCP and TCP by Canadian Pulp Method (Chlorinated Phenols) National Council for Air and Stream Improvement (NCASI) 86.07.

Laboratory analytical reports are presented in Appendix C.

3.4 Investigation-Derived Waste Disposal

Purgewater and decontamination water produced during sampling activities were stored onsite in a Department of Transportation (DOT)-approved 55-gallon drum. The drum was transported to the

Woodward Drilling Company, Inc. wastewater treatment facility, in Rio Vista, California on May 9, 2017 (Appendix D).

3.5 Site Walk of 1678 Glendale Drive Dip Tank Building

A reconnaissance of the concrete slab floor of the Dip Tank Building located at 1678 Glendale Drive was completed in response to an April 21, 2017 DTSC email request. Prior to the monitoring event, contact information for the owner of this property was not readily available. During the monitoring event, an onsite facility representative indicated that Gary Johnson was the property owner. During a subsequent discussion, Mr. Johnson verbally approved access to the property for inspection.

The condition of the concrete slab floor of the Dip Tank Building appeared similar to DTSC's 2007 observations documented in the Annual Inspection Report (DTSC, 2007). The building is largely used to store vehicles and maintenance equipment. Localized oil staining and surface deterioration (e.g., chatter marks) were observed. No signs of cracking or settling were observed in the readily accessible areas. Photographs of the concrete slab floor of the Dip Tank Building are provided in Appendix E.

4.0 GROUNDWATER MONITORING RESULTS

4.1 Groundwater Elevations

During the May 2017 gauging event, depth to groundwater measurements ranged from 5.00 feet below top of casing (btoc) in well MW-1 to 11.38 feet btoc in well MW-7. The water levels are approximately 0.9 feet to 2.0 feet deeper than observed during the December 2016 monitoring event. Note that the depth to water in well MW-10, which was considered anomalous in December 2016, was more consistent with historic levels in May 2017.

Groundwater elevations ranged from 84.71 feet above mean sea level (msl) in well MW-10 to 90.66 feet above msl in well MW-9. Based on the groundwater elevation data collected during the May 2017 gauging event, horizontal hydraulic gradients are generally to the south-southwest. The May 2017 groundwater elevation data and contours are presented on Figure 4. Groundwater level measurements and elevation calculations are presented in Table 1.

4.2 Water Quality Parameter Data Summary

The water quality parameters measured in the field during the May 2017 monitoring event is summarized on Table 1. General findings for May 2017 water quality parameters are described below.

- DO concentrations ranged from 0.17 milligrams per liter (mg/L) to 1.47 mg/L. DO concentrations below 1 mg/L were measured in wells MW-1, MW-5, MW-8, MW-10, and MW-11;
- ORP levels ranged from 14.4 millivolts (mV) to 465.7 mV;
- pH ranged from 5.08 to 6.00. The prevalence of pH values below 7.0 indicates slightly acidic groundwater conditions beneath the Site;
- Conductivity measurements ranged from 0.094 millisiemens per centimeter (mS/cm) to 0.546 mS/cm; and
- TDS levels ranged from 62 mg/L to 355 mg/L.

4.3 PCP and TCP Groundwater Analytical Results

The PCP and TCP analytical results for groundwater samples collected during the May 2017 monitoring event are summarized on Table 2. Laboratory analytical reports are presented in Appendix C. General findings for PCP and TCP in groundwater are described below.

- PCP was detected above laboratory reporting limits in four of the eight monitoring wells sampled. Detected concentrations were reported at up to 570 µg/L in well MW-1, 81 µg/L in well MW-12, 46 µg/L in well MW-5, and 1.9 µg/L in well MW-11.
- TCP was detected above laboratory reporting limits in two of the eight monitoring wells sampled at a concentration of up to 8.4 µg/L in well MW-1 and 2.3 µg/L in well MW-5.

The May 2017 distribution of PCP and TCP in shallow water-bearing zone are depicted on Figure 4.

4.4 Groundwater Monitoring Quality Assurance/Quality Control

The groundwater analytical data collected during the May 2017 monitoring event were evaluated to ensure that the data quality objectives identified in the *Quality Assurance Project Plan* were met (URS, 2011). The results were reviewed for precision, accuracy, representativeness, completeness, comparability, and method detection limits. The laboratory reports were reviewed for data completeness, chain-of-custody, holding times, blanks, surrogates, and laboratory control samples and duplicates. In addition, QA/QC samples (field duplicate samples) were collected during the 2017 monitoring event. QA/QC analyses included the following:

- Method blank;
- Laboratory control spike (LCS)/laboratory control spike duplicate (LCSD);
- Surrogate recoveries; and
- Field duplicate samples for similarity.

The QA/QC findings indicate the following:

- No detections in the method blanks were noted;
- LCS/LCSD and surrogate recoveries were within control limits; and
- Field duplicates results were sufficiently similar (RPD < 30%) in PCP and TCP concentrations (Table 3).

Based on these findings, the overall data quality is considered acceptable.

5.0 FINDINGS

5.1 Water Levels

Findings of the water level data for May 2017 indicate:

- Groundwater elevations were approximately 1.0 to 2.0 feet lower than during the December 2016 monitoring event; and
- Horizontal hydraulic gradients to the south, in general, were consistent with historic observations.

5.2 PCP and TCP Distribution

PCP and TCP concentrations were detected in monitoring wells in the central area of the Site near the former Green Chain area and former Saw Mill building (Figure 4). For screening level purposes, the California maximum contaminant level (CA MCL) for PCP of 1 µg/L was used. There is no CA MCL for TCP. A summary of the May 2017 findings indicates:

- PCP concentrations exceed the CA MCL of 1 µg/L in wells MW-1, MW-5, MW-11, and MW-12, which are located hydraulically downgradient and south of the former Green Chain area and former Saw Mill building. The December 2016 and May 2017 data indicate PCP concentrations increased in each of these four wells. The PCP concentration in well MW-1 increased from up to 1.2 µg/L in December 2016 to 570 µg/L in May 2017. Concentration increases may be attributed to a dissolution of mass associated with observed higher groundwater elevations in the fourth quarter of 2016 across the Site;
- TCP was detected above laboratory reporting limits in wells MW-1 and MW-5. The December 2016 and May 2017 data indicate that the TCP concentration in well MW-1 increased but was similar to May 2016 concentration. TCP concentrations in well MW-5 were similar to previous results since 2002; and
- The May 2017 PCP and TCP distributions are similar and consistent with the historical distribution. As depicted on Figure 4, the absence of TCP in well MW-12, suggests PCP has a slightly larger distribution than TCP. The presence of a low concentration of PCP in well MW-11 was similar to intermittent low detections since 2010.

6.0 LIMITATIONS

This document was prepared for the exclusive use of the DTSC for the express purpose of complying with a client- or regulatory directive for environmental investigation or restoration. SGI-Apex and DTSC must approve any re-use of this work product in whole or in part for a different purpose or by others in writing. If any such unauthorized use occurs, it shall be at the user's sole risk without liability to SGI-Apex or DTSC. To the extent that this document is based on information provided to SGI-Apex by third parties, including DTSC, their direct contractors, previous workers, and other stakeholders, SGI-Apex cannot guarantee the completeness or accuracy of this information, even where efforts were made to verify third-party information. SGI-Apex has exercised professional judgment to collect and present findings and opinions of a scientific and technical nature. The opinions expressed are based on the conditions of the Site existing at the time of the field investigation, current regulatory requirements, and any specified assumptions. The presented findings and recommendations in this document are intended to be taken in their entirety to assist DTSC in applying their own professional judgment in making decisions related to the property. SGI-Apex cannot provide conclusions on environmental conditions outside the completed scope of work. SGI-Apex cannot guarantee that future conditions will not change and affect the validity of the presented conclusions and recommended work. No warranty or guarantee, whether expressed or implied, is made with respect to the data or the reported findings, observations, conclusions, and recommendations.

7.0 REFERENCES

California Department of Toxics Substances Control (DTSC). 2007. Annual Inspection Report, Former McNamara and Peepe Lumber Mill. July 11.

DTSC. 2008. McNamara and Peepe Lumber Mill, Docket Number I&/SE 07/08-009, Imminent and Substantial Endangerment Determination. April 22.

DTSC. 2014. Five-Year Comprehensive Review, McNamara and Peepe Lumber Mill, 1619 Glendale Drive, McKinleyville, California. November.

California Department of Water Resources (DWR). 2004. California's Groundwater Bulletin 118. Updated February 27.

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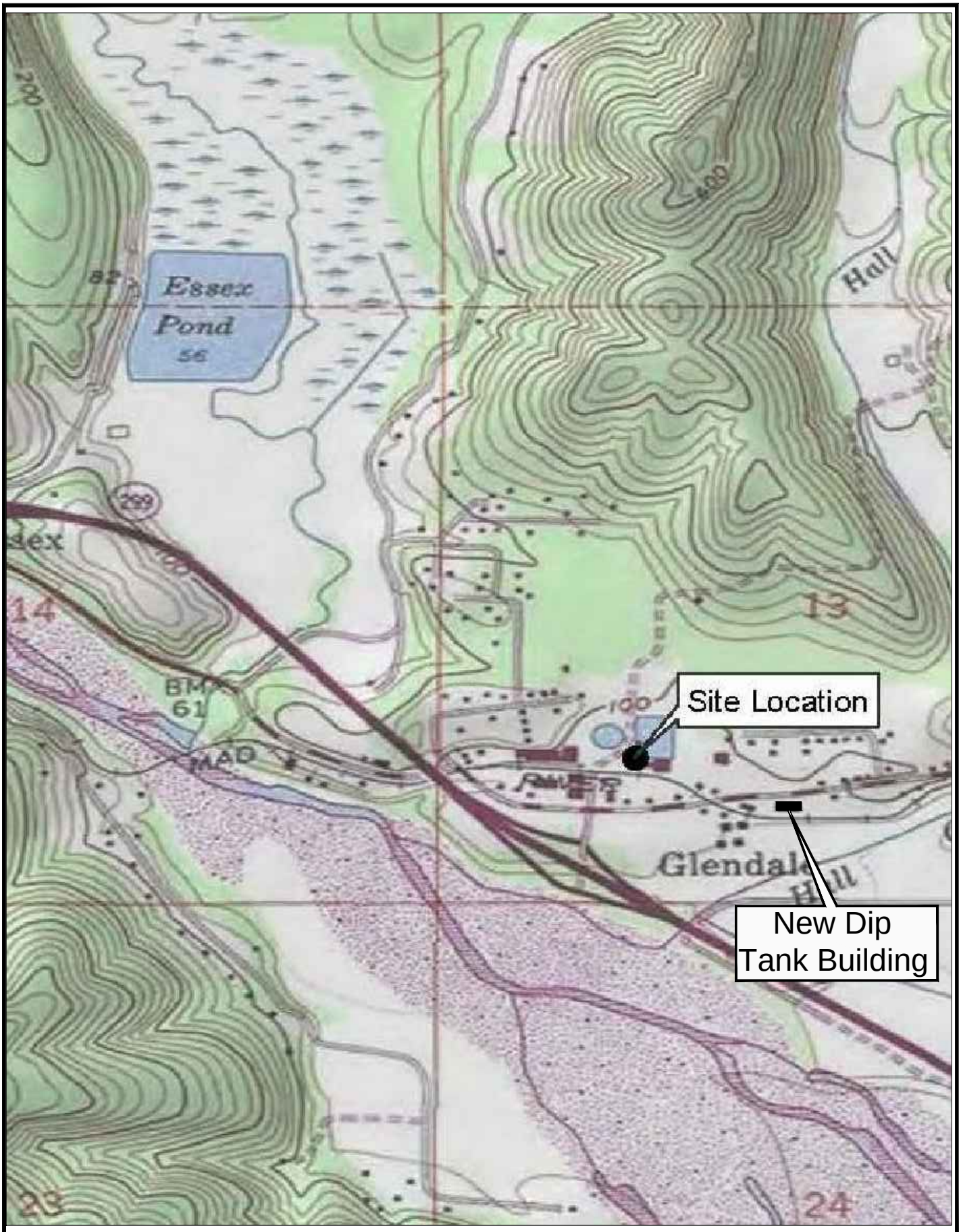
URS Corporation (URS). 2011. Soil and Groundwater Investigation Work Plan, McNamara & Peepe Lumber Mill, 1589 Glendale Drive, Arcata, California. October 24.

FIGURES



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SGI environmental | APEX

3478 BUSKIRK AVENUE, SUITE 100
PLEASANT HILL, CA 94523

McNAMARA AND PEEPE
LUMBER MILL
ARCATA, CALIFORNIA

SITE LOCATION MAP

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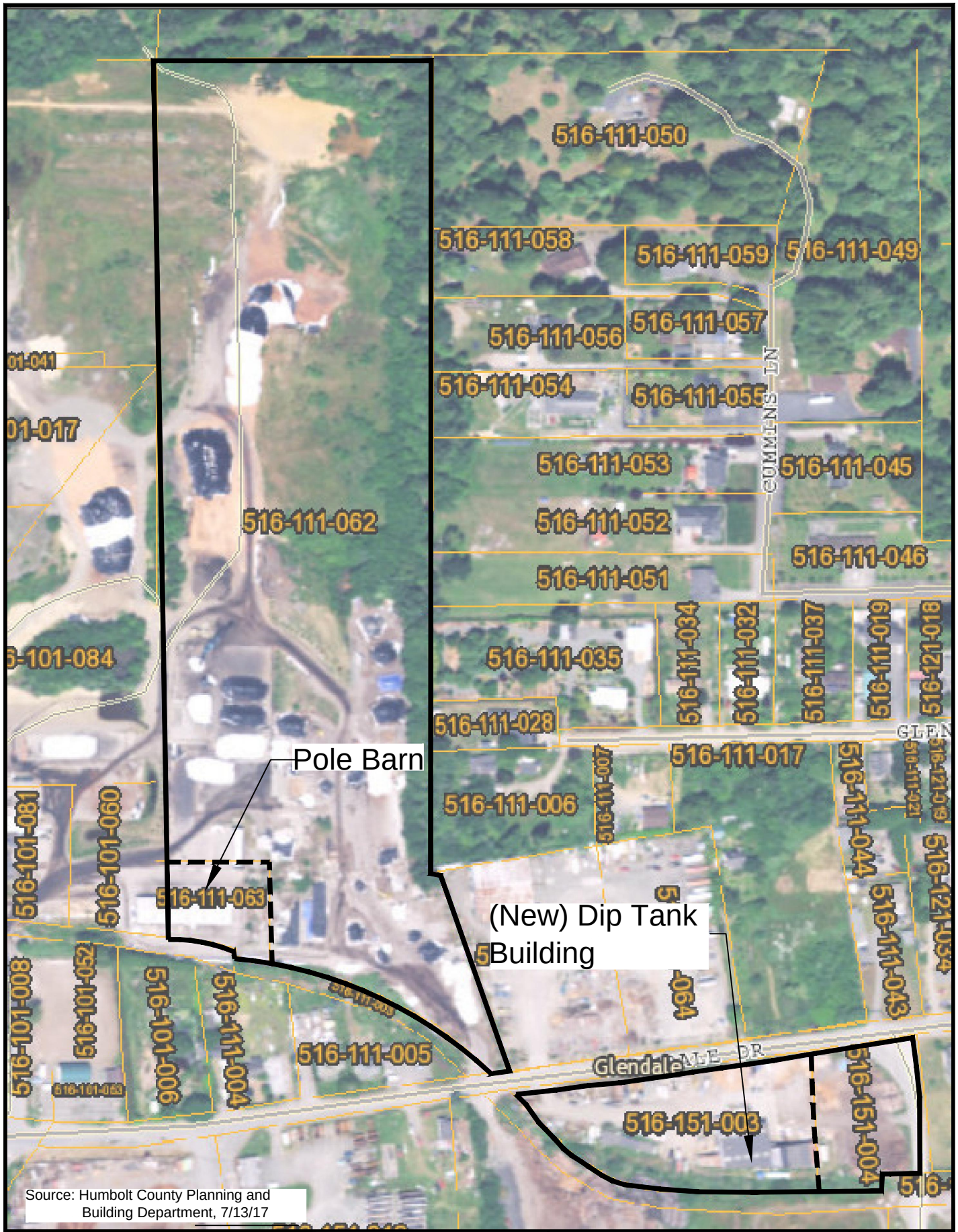
PROJECT NO.	DATE	DR.BY:	APP. BY:
01-DTSC-006	07/19/17	ZA	KR

FIGURE 1



Source: Humbolt County Planning and Building Department, 7/13/17

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SGI environmental | APEX

3478 BUSKIRK AVENUE, SUITE 100
PLEASANT HILL, CA 94523

McNAMARA AND PEEPE
LUMBER MILL
ARCATA, CALIFORNIA

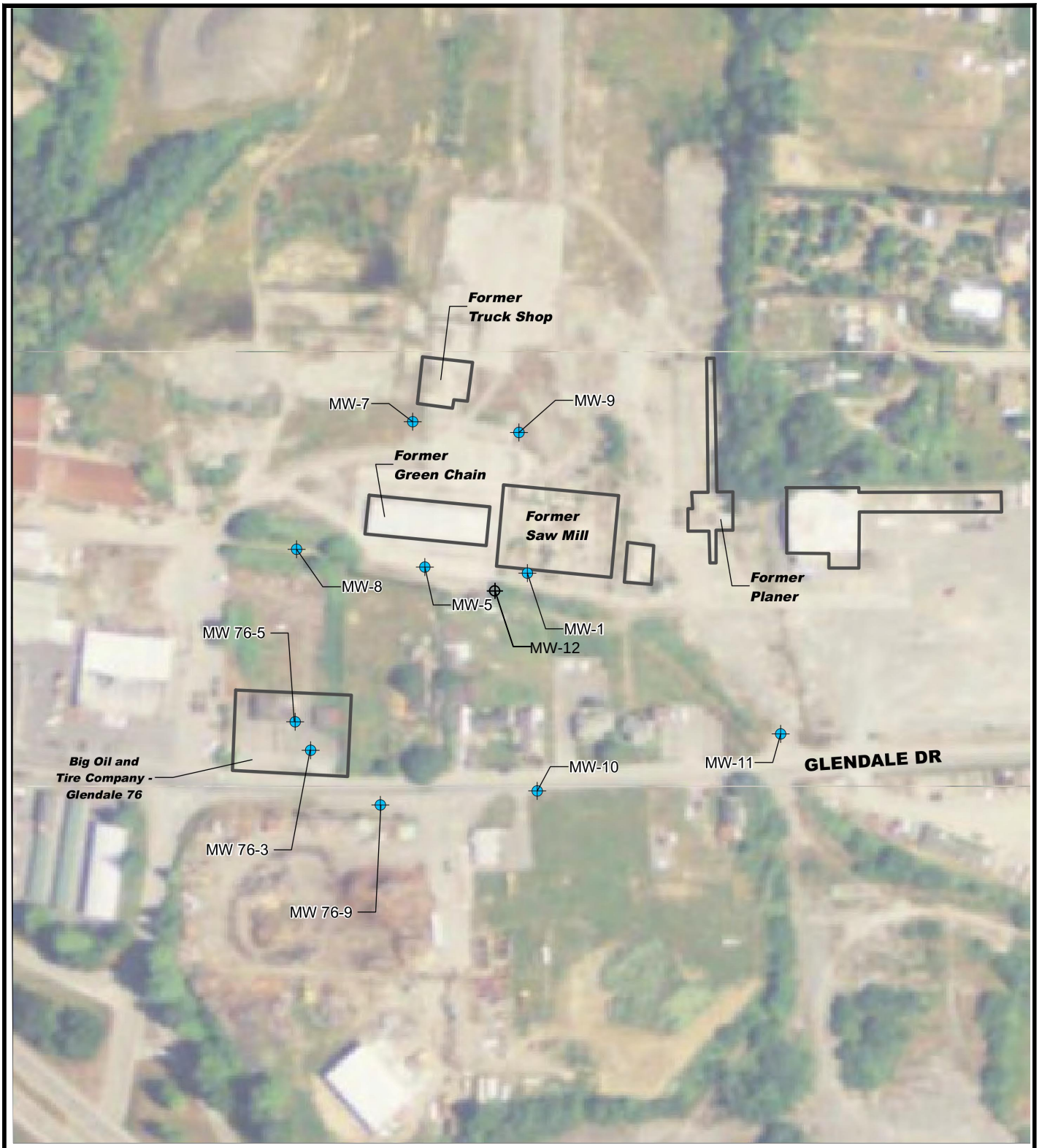
SITE LAYOUT

N

PROJECT NO.	DATE	DR.BY:	APP. BY:
01-DTSC-006	07/19/17	ZA	KR

0 300 600
HORIZONTAL SCALE IN FEET

FIGURE 2



LEGEND

Monitoring well location

Building/former building

Source: URS, 2011 Soil and Groundwater Investigation Work Plan, October.

SGI environmental | APEX

3478 BUSKIRK AVENUE, SUITE 100
PLEASANT HILL, CA 94523

McNAMARA AND PEEPE
LUMBER MILL
ARCATA, CALIFORNIA

SITE FEATURES

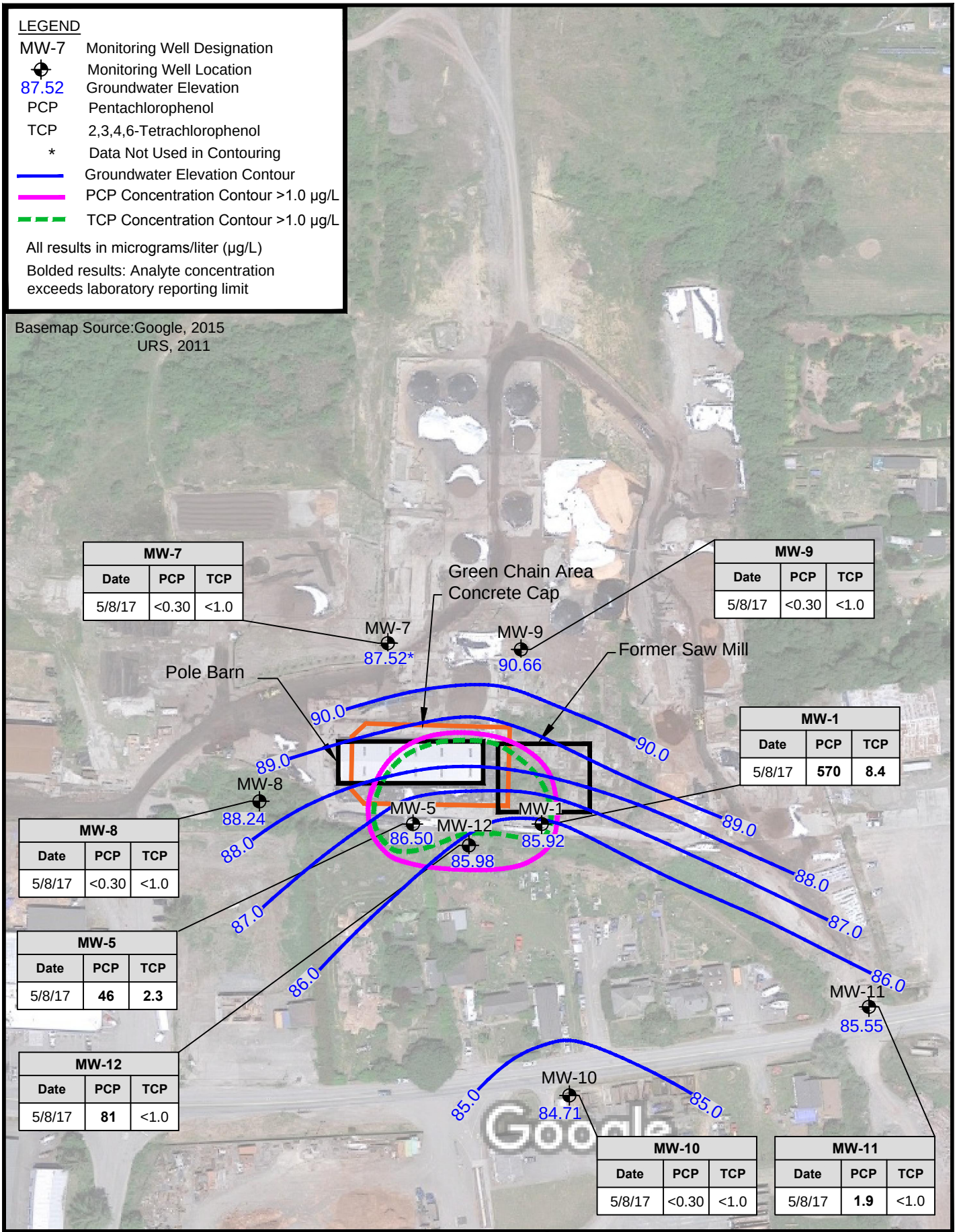
N

PROJECT NO.	DATE	DR.BY:	APP. BY:
01-DTSC-006	07/19/17	ZA	KR

0 200 400
HORIZONTAL SCALE IN FEET

FIGURE 3

S:\Clients A - F\DTSC_McNamara_Peepe Corp - DTSC-006\Report - May 2017\Fig.3-Site Features.dwg, 7/19/2017 12:47:31 PM



S:\Clients A - F\DTSC_McNamara_Peepe Corp - DTSC-006\Report - May 2017\Fig.4-GW Elevation Contours, PCP & TCP Concentration in GW (from Server5).dwg, 7/19/2017 12:49:59 PM

SGI environmental | APEX

3478 BUSKIRK AVENUE, SUITE 100
PLEASANT HILL, CA 94523

McNAMARA AND PEEPE
LUMBER MILL
ARCATA, CALIFORNIA

GROUNDWATER ELEVATION CONTOURS, PCP AND TCP CONCENTRATIONS IN GROUNDWATER MAY 2017

PROJECT NO.	DATE	DR.BY:	APP. BY:
01-DTSC-006	07/19/17	ZA	KR

0 100 200
HORIZONTAL SCALE IN FEET

N

FIGURE 4

TABLES

Table 1
Groundwater Elevation and Field Parameters
McNamara and Peepe Lumber Mill
Arcata, California

Well	Screened Interval	Date	Depth to Water	TOC Elevation	Groundwater Elevation	Temperature	pH	DO	ORP	Conductivity	Turbidity	TDS
	(feet btoc)		(feet btoc)	(feet msl)	(feet msl)	(degrees C)	--	(mg/L)	(mV)	(mS/cm)	(NTU)	(mg/L)
MW-1	19-23	11/2011	3.19	90.92	87.73	14.39	5.80	0.95	134	NR[1]	57.6	NR
		5/13/2015	7.32		83.60	15.51	5.42	0.75	70.7	0.279	3.9	NR
		11/10/2015	11.15		79.77	18.39	5.61	1.27	121.3	0.281	-3.8[2]	NR
		5/23/2016	6.87		84.05	16.37	6.25	0.80	-15.1	0.479	3.1	372
		12/14/2016	3.00		87.92	13.20	6.69	2.89	150.1	0.491	3.6	319
		5/8/2017	5.00		85.92	15.50	6.00	0.21	102.7	0.546	46.8	355
MW-5	18-23	11/2011	5.21	93.25	88.04	14.37	5.88	0.99	-22	NR[1]	121	NR
		5/13/2015	9.40		83.85	14.65	5.15	0.87	183.7	0.243	1.1	NR
		11/10/2015	12.15		81.10	16.62	5.13	1.32	170.1	0.205	1.1[2]	NR
		5/23/2016	8.90		84.35	15.68	5.44	0.54	22.7	0.250	48.5	200
		12/14/2016	5.20		88.05	16.20	5.28	0.05	176.9	0.275	3.3	178
		5/8/2017	6.75		86.50	15.30	5.17	0.17	155.8	0.302	68.3	197
MW-7	22-37	11/2011	9.67	98.90	89.23	15.17	5.55	1.67	119	0.062	104	NR
		5/13/2015	13.63		85.27	16.86	5.28	1.55	151.3	0.095	1.4	NR
		11/10/2015	17.90		81.00	15.33	5.50	1.43	223.7	0.089	-2.8[2]	NR
		5/23/2016	13.33		85.57	18.15	5.70	2.01	17.3	0.130	5.9	96
		12/14/2016	9.82		89.08	16.80	5.60	2.34	237.2	0.108	15.1	NR
		5/8/2017	11.38		87.52	14.80	5.31	1.32	264.8	0.111	50.3	72
MW-8	8.5-24	5/13/2015	8.48	96.04	87.56	15.55	5.96	0.70	26.6	0.476	2.0	NR
		11/10/2015	11.40		84.64	18.03	5.40	1.80	190.5	0.712	3.5[2]	NR
		5/23/2016	8.72		87.32	16.12	6.22	0.82	-137.4	0.392	6.7	302
		12/14/2016	5.90		90.14	14.10	6.16	0.71	103.1	0.321	7.1	NR
		5/8/2017	7.80		88.24	13.60	5.96	0.68	14.4	0.495	48.3	321

Table 1
Groundwater Elevation and Field Parameters
McNamara and Peepe Lumber Mill
Arcata, California

Well	Screened Interval	Date	Depth to Water	TOC Elevation	Groundwater Elevation	Temperature	pH	DO	ORP	Conductivity	Turbidity	TDS
	(feet btoc)		(feet btoc)	(feet msl)	(feet msl)	(degrees C)	--	(mg/L)	(mV)	(mS/cm)	(NTU)	(mg/L)
MW-9	21-25	11/2011	6.27	99.65	93.38	14.26	5.64	1.18	408	NR(1)	33.6	NR
		5/13/2015	11.17		88.48	17.08	5.83	1.65	164.7	0.251	1.5	NR
		11/10/2015	14.29		85.36	17.30	5.70	1.79	465.7	0.178	-4.2(2)	NR
		5/23/2016	10.97		88.68	16.72	6.01	1.09	18.5	0.290	49.1	224
		12/14/2016	8.09		91.56	16.60	6.00	4.82	241.2	0.207	3.4	NR
		5/8/2017	8.99		90.66	14.40	5.69	1.47	465.7	0.301	70.3	196
MW-10	9-24	11/2011	9.74	95.65	85.91	12.12	5.22	7.14	207	0.013	68.1	NR
		5/13/2015	13.44		82.21	15.85	5.03	1.29	179.7	0.118	48.2	NR
		11/10/2015	16.15		79.50	16.93	5.32	1.79	180.5	0.039	36.4(2)	NR
		5/23/2016	13.36		82.29	15.28	5.37	2.91	57.1	0.094	48.3	74
		12/14/2016	5.70		89.95	11.70	5.80	9.28	217.0	0.020	24.1	NR
		5/8/2017	10.94		84.71	13.60	5.93	0.69	117.1	0.094	50.3	62
MW-11	9.5-24.5	11/2011	5.20	91.70	86.50	14.00	5.12	1.37	155	0.048	29.8	NR
		5/13/2015	7.80		83.90	16.88	5.04	0.78	202.8	0.086	1.5	NR
		11/10/2015	9.97		81.73	17.28	5.07	1.40	252.7	0.079	-4.4(2)	NR
		5/23/2016	7.25		84.45	16.42	5.16	1.74	64.8	0.145	0.3	111
		12/14/2016	4.24		87.46	16.90	5.01	0.57	214.9	0.260	4.2	NR
		5/8/2017	6.15		85.55	14.70	5.08	0.47	194.1	0.281	65.7	183

Table 1
Groundwater Elevation and Field Parameters
McNamara and Peepe Lumber Mill
Arcata, California

Well	Screened Interval	Date	Depth to Water	TOC Elevation	Groundwater Elevation	Temperature	pH	DO	ORP	Conductivity	Turbidity	TDS
	(feet btoc)		(feet btoc)	(feet msl)	(feet msl)	(degrees C)	--	(mg/L)	(mV)	(mS/cm)	(NTU)	(mg/L)
MW-12	10-20	11/2011	3.92	91.73	87.81	14.14	5.67	0.91	11	NR[1]	41.6	NR
		5/13/2015	8.20		83.53	14.69	5.28	0.81	167.3	0.189	31.7	NR
		11/10/2015	12.05		79.68	16.09	5.38	1.24	77.9	0.196	-1.1[2]	NR
		5/23/2016	7.75		83.98	15.19	5.55	1.01	10.1	0.230	4.1	184
		12/14/2016	3.80		87.93	14.40	5.42	0.52	240.2	0.228	4.3	NR
		5/8/2017	5.75		85.98	15.70	5.32	1.07	180.4	0.221	43.7	139

Notes:
Data prior to 2015 from URS (2011).
TOC = Top of casing
bgs = Below ground surface
btoc = Below top of casing
C = Celsius
DO = Dissolved oxygen
TDS - total dissolved solids
msl = mean sea level
mS/cm = Millisiemens per centimeter
mg/L = Milligrams per liter
mV = Millivolts
NTU = Nephelometric Turbidity Units
ORP = Oxidation Reduction Potential
NR = Not Recorded
[1] Conductivity not recorded due to equipment errors.
[2] Negative turbidity readings during November 2015 considered suspect due to equipment errors.

Table 2
Groundwater Analytical Results - PCP and TCP
McNamara and Peepe Lumber Mill
Arcata, California

Well Name	Date	PCP	TCP
	CA MCL	***1.0***	***NV***
	Analytical Method	***Canadian Pulp Method***	
MW-1	7/31/1997	<0.30	<1.0
	1/12/1998	<0.30	<1.0
	4/8/1998	<0.30	<1.0
	7/8/1998	<0.30	<1.0
	10/10/1998	--	--
	1/26/1999	<0.30	<1.0
	7/14/1999	<0.30	<1.0
	4/13/2000	<0.30	<1.0
	10/19/2000	<0.30	<1.0
	6/7/2001	**0.49**	<1.0
	12/26/2002	<0.30	<1.0
	12/12/2003	**1,100**	**19**
	12/24/2003	**720**	**11**
	3/15/2004	**1,100**	**15**
	6/10/2004	**900**	**19.8**
	6/28/2005	**890**	**11**
	8/4/2005	**890**	**14**
	06/2010	**0.34**	<1.0
	10/2010	**2,200**	**36**
	11/2011	**1,300**	**25**
	4/2012	**1,300**	**24**
	5/13/2015	**690**	**14**
	5/13/2015 (FD)	**560**	**12**
	11/11/2015	**610**	**120**
	11/11/2015 (FD)	**670**	**120**
	5/23/2016	**830**	**7.1**
	5/23/2016 (FD)	**1,100**	**8.0**
	12/14/2016	**1.2**	<1.0
	12/14/2016 (FD)	**1.2**	<1.0
	5/8/2017	**570**	**8.4**
	5/8/2017 (FD)	**530**	**7.9**
MW-5	7/31/1997	<0.30	<1.0
	1/12/1998	<0.30	<1.0
	4/8/1998	<0.30	<1.0
	7/8/1998	<0.30	<1.0
	7/8/1998 (FD)	<0.30	<1.0
	10/10/1998	--	--
	1/26/1999	<0.30	<1.0
	7/14/1999	<0.30	<1.0

Table 2
Groundwater Analytical Results - PCP and TCP
McNamara and Peepe Lumber Mill
Arcata, California

Well Name	Date	PCP	TCP
	CA MCL	***1.0***	***NV***
MW-5 (Cont.)	4/13/2000	<0.30	<1.0
	10/19/2000	<0.30	<1.0
	10/19/2000 (FD)	<0.30	<1.0
	6/7/2001	<0.30	<1.0
	6/7/2001 (FD)	**0.68**	<1.0
	12/26/2002	<0.30	<1.0
	12/26/2002 (FD)	<0.30	<1.0
	12/12/2003	<0.30	<1.0
	12/12/2003 (FD)	<0.30	<1.0
	1/28/2005	<0.30	<1.0
	1/28/2005 (FD)	<0.30	<1.0
	8/4/2005	<0.30	<1.0
	06/2010	**1.7**	<1.0
	10/2010	**1.6**	<1.0
	11/2011	**5.1**	<1.0
	4/2012	**54**	**2.2**
	5/13/2015	**35**	**4.3**
	11/11/2015	**65**	**3.3**
	5/23/2016	**56**	**1.6**
	12/14/2016	**39**	**2.3**
	5/8/2017	**46**	**2.3**
MW-6	7/31/1997	<0.30	<1.0
MW-7	1/12/1998	<0.30	<1.0
	4/8/1998	<0.30	<1.0
	4/8/1998	<0.30	<1.0
	7/8/1998	<0.30	<1.0
	10/10/1998	<0.30	<1.0
	1/26/1999	<0.30	<1.0
	1/26/1999	<0.30	<1.0
	7/14/1999	<0.30	<1.0
	4/13/2000	<0.30	<1.0
	4/13/2000	<0.30	<1.0
	10/19/2000	<0.30	<1.0
	6/7/2001	**0.36**	<1.0
	12/26/2002	<0.30	<1.0
	12/12/2003	<0.30	<1.0
	1/28/2005	<0.30	<1.0
	8/4/2005	<0.30	<1.0
	8/4/2005 (FD)	<0.30	<1.0

Table 2
Groundwater Analytical Results - PCP and TCP
McNamara and Peepe Lumber Mill
Arcata, California

Well Name	Date	PCP	TCP
	CA MCL	***1.0***	***NV***
MW-7 (Cont.)	06/2010	<0.30	<1.0
	10/2010	<0.30	<1.0
	11/2011	<0.30	<1.0
	4/2012	<0.30	<1.0
	5/13/2015	**0.39**	<1.0
	11/11/2015	<0.30	<1.0
	5/23/2016	<0.30	<1.0
	12/14/2016	<0.30	<1.0
	5/8/2017	<0.30	<1.0
MW-8	1/12/1998	<0.30	<1.0
	4/8/1998	**1.3**	<1.0
	4/27/1998	<0.30	<1.0
	7/8/1998	<0.30	<1.0
	10/10/1998	--	--
	1/26/1999	<0.30	<1.0
	7/14/1999	<0.30	<1.0
	4/13/2000	<0.30	<1.0
	10/19/2000	<0.30	<1.0
	6/7/2001	<0.30	<1.0
	12/26/2002	<0.30	<1.0
	8/4/2005	<0.30	<1.0
	5/13/2015	<0.30	<1.0
	11/11/2015	<0.30	<1.0
	5/23/2016	<0.30	<1.0
	12/14/2016	<0.30	<1.0
	5/8/2017	<0.30	<1.0
MW-9	1/12/1998	<0.30	<1.0
	4/8/1998	<0.30	<1.0
	7/8/1998	<0.30	<1.0
	10/10/1998	<0.30	<1.0
	10/10/1998	<0.30	<1.0
	1/26/1999	<0.30	<1.0
	7/14/1999	<0.30	<1.0
	7/14/1999 (FD)	<0.30	<1.0
	4/13/2000	<0.30	<1.0
	10/19/2000	<0.30	<1.0
	6/7/2001	<0.30	<1.0
	12/26/2002	<0.30	<1.0
	8/3/2005	<0.30	<1.0

Table 2
Groundwater Analytical Results - PCP and TCP
McNamara and Peepe Lumber Mill
Arcata, California

Well Name	Date	PCP	TCP
	CA MCL	***1.0***	***NV***
MW-9 (Cont.)	06/2010	<0.30	<1.0
	10/2010	<0.30	<1.0
	11/2011	<0.30	<1.0
	4/2012	<0.30	<1.0
	5/13/2015	<0.30	<1.0
	11/11/2015	<0.30	<1.0
	5/23/2016	<0.30	<1.0
	12/14/2016	<0.30	<1.0
	5/8/2017	<0.30	<1.0
MW-10	06/2010	<0.30	<1.0
	10/2010	<0.30	<1.0
	11/2011	<0.30	<1.0
	4/2012	<0.30	<1.0
	5/13/2015	<0.30	<1.0
	11/11/2015	<0.60	<2.0
	5/23/2016	<0.30	<1.0
	12/14/2016	<0.30	<1.0
	5/8/2017	<0.30	<1.0
MW-11	10/2010	**0.84**	<1.0
	11/2011	<0.30	<1.0
	4/2012	**1.6**	<1.0
	5/13/2015	<0.30	<1.0
	11/11/2015	**0.67**	<1.0
	5/23/2016	<0.30	<1.0
	12/14/2016	<0.30	<1.0
	5/8/2017	**1.9**	<1.0
MW-12	11/2011	**24**	<1.0
	04/2012	**53**	<1.0
	5/13/2015	**52**	<1.0
	11/11/2015	**51**	<1.0
	5/23/2016	**120**	<1.0
	12/14/2016	**46**	<1.0
	5/8/2017	**81**	<1.0

Notes:
Data prior to 2015 from URS (2011).
All results in micrograms per liter
CA MCL = California Maximum Contaminant Levels
PCP = Pentachlorophenol
TCP = 2,3,4,6-Tetrachlorophenol

Table 2
Groundwater Analytical Results - PCP and TCP
McNamara and Peepe Lumber Mill
Arcata, California

Well Name	Date	PCP	TCP
	CA MCL	***1.0***	***NV***

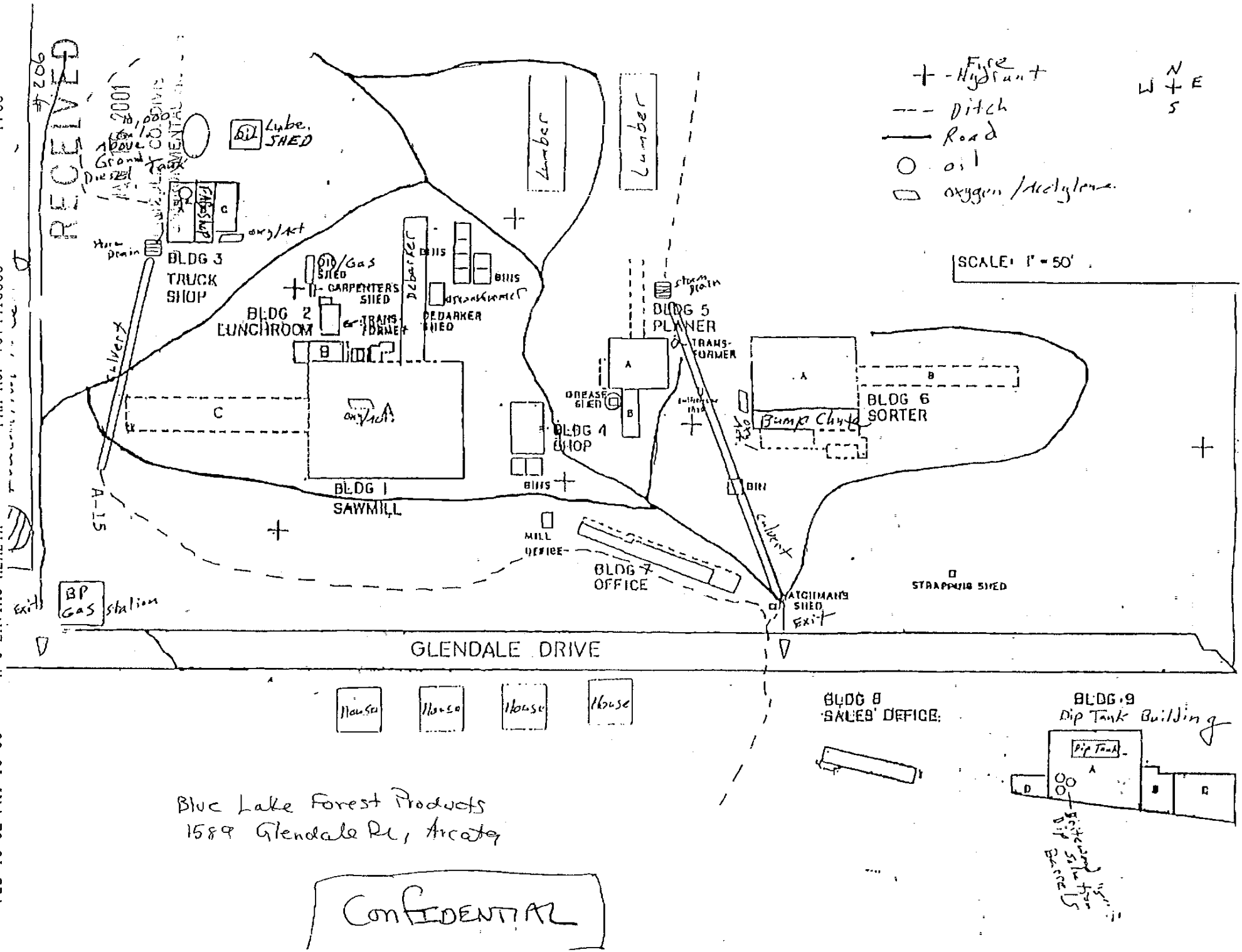
Embolden values: Analyte concentration exceeds laboratory reporting limit
Shaded values: Analyte concentration exceeds MCL
< = indicates value is below the noted laboratory reporting limit
NV = No established value
FD = Field duplicate

Table 3
Comparison of Primary and Duplicate Sample Analytical Results
McNamara and Peepe Lumber Mill
Arcata, California

Well Name	Date	PCP	TCP
MW-1	5/8/2017	570	8.4
	5/8/2017 (FD)	530	7.9
	RPD	**7%**	**6%**

Notes:
Analytical results in micrograms per liter
PCP = Pentachlorophenol
TCP = 2,3,4,6-Tetrachlorophenol
RPD = relative percent difference
FD = Field duplicate
NA = Not applicable

EXHIBIT C



CONFIDENTIAL

EXHIBIT D

11/25/2003

N

Glenwood Ln

Glendale Dr

Image © 2019 DigitalGlobe

Google Earth

2003

Imagery Date: 11/25/2003 40°54'01.70" N 124°01'03.10" W elev 0 ft eye alt 1048 ft

Exhibit E

Available at
https://www.envirostor.dtsc.ca.gov/public/deliverable_documents/8383564591/Decertification%20%5BDTSC%2012-28-18%5D.pdf

Department of Toxic Substances Control

Matthew Rodriquez
Secretary for
Environmental Protection

Barbara A. Lee, Director
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Berkeley, California 94710-2721

Edmund G. Brown Jr.
Governor

December 28, 2018

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Ms. Jennifer Finch and Mr. Robert Schultz
P.O. Box 146
Arcata, California 95518
magnaws@gmail.com

DECERTIFICATION, MCNAMARA AND PEEPE LUMBER MILL, GENDALE, HUMBOLDT COUNTY, CALIFORNIA

Dear Mr. Aalfs, Ms. Finch, and Mr. Schultz:

The Department of Toxic Substances Control (DTSC) issued a Remedial Action Certification on March 9, 1998 for McNamara and Peepe Lumber Mill (Site) upon implementation of the remedial actions pursuant to the December 4, 1994 Remedial Action Plan. However, subsequent soil and groundwater investigations have revealed that soil and groundwater contamination at the Site is not under control and the implemented remedial actions are no longer protective of human health and the environment. Therefore, DTSC rescinds the March 9, 1998 Remedial Action Certification and issues this Decertification based on the following findings:

Site Identification and Landowners: The Site is located in Glendale, an unincorporated community in Humboldt County, approximately 0.9 miles southeast of the City of McKinleyville and approximately 1.2 miles northwest of the City of Blue Lake, Humboldt County, California. The Site occupies approximately 26 acres with nine Assessor's Parcel Numbers (APNs). The current landowners of the Site are (a) Blue Lake Forest Products, Inc. and (b) Jennifer Finch and Robert Schultz.

- Blue Lake Forest Products, Inc. owns seven parcels with APNs 516-091-020, 516-101-040, 516-101-060, 516-111-062, 516-111-063, 516-111-064, and 516-111-066 located on 1619 Glendale Drive.

- Jennifer Finch and Robert Schultz own two parcels with APNs 516-151-003 and 516-151-004 located on 1678 Glendale Drive.

1998 Remedial Action Certification: On December 5, 1994, DTSC approved the Remedial Action Plan with the following remedies for the Site:

- Consolidation of pentachlorophenol (PCP) and tetrachlorophenol (TCP) contaminated soils at the Green Chain area and installation of a new cap over such contaminated soils at areas encompassing APNs 516-101-060 and 516-111-063;

- Surface water and groundwater monitoring; and

- A land use covenant prohibiting any site activities which may compromise the integrity of the cap located at areas within APNs 516-101-060 and 516-111-063 and concrete slab located at an area within APN 516-151-003, as well as prohibiting development of these areas for uses for a residence, long-term care hospital, day-care facility, and school.

On March 9, 1998, DTSC issued the Remedial Action Certification stating that (a) all appropriate remedial actions have been completed, (b) a deed restriction was recorded the County's Recorder Office, and (c) long-term surface water and groundwater monitoring are necessary at the Site.

Subsequent Investigations and Contamination: During groundwater monitoring events conducted from 1997 through 2002, PCP concentrations were predominately below the cleanup goal of 1 µg/L and TCP concentrations were all below the laboratory reporting limit of 1 µg/L. In April 2002, Blue Lake Forest Products, Inc. declared bankruptcy and ceased groundwater pumping from an onsite lumber mill production well PW-1, which caused a rise of the groundwater elevation to approximately 15 feet higher than the previous groundwater elevation measured while the production well was operational. Since April 2002, groundwater has been in contact with the PCP- and TCP-impacted soil beneath the cap, thereby mobilizing hazardous substances from soil to groundwater.

Grab groundwater samples collected in May 2005 at various Site locations contained PCP and TCP concentrations as high as 16,000 µg/L and 1,500 µg/L, respectively. From December 2003 through May 2017, PCP and TCP have been detected in groundwater monitoring wells at concentrations up to 2,200 µg/L and 120 µg/L, respectively.

On April 22, 2008, DTSC issued an Imminent and Substantial Endangerment Determination, Docket No. I&SED 07/08-009 for this Site, because there has been a release or a threatened release of hazardous substances at the Site.

The former saw mill area, located within APNs 516-111-062 and 516-111-063, is partially unpaved and located adjacent to the cap at the Green Chain area encompassing APNs 516-101-060 and 516-111-063. The former saw mill building at the former saw mill area was demolished in 2006. Portions of the building foundation, in poor condition, remain at the former saw mill area. In 2010 and 2011, DTSC conducted investigation at the former saw mill area and found PCP concentrations in soil ranging from 1.8 mg/kg to 40 mg/kg, above the PCP cleanup goal of 1.75 mg/kg established in the 1994 Remedial Action Plan.

Therefore, the remedy selected in the 1994 Remedial Action Plan is no longer protective because (a) rising groundwater level have mobilized PCP/TCP in soil beneath the Green Chain area cap due to cessation of production well pumping in 2002; (b) surface water can percolate through PCP/TCP-impacted soil present below the former saw mill area as this area is partially unpaved and/or covered with a building foundation in poor condition; and (c) PCP/TCP can migrate offsite in groundwater or surface water runoff across the former saw mill area. Since the former saw mill area is partially unpaved and the pavement is in poor condition, people also run the risk of coming into direct contact with the contaminants. Therefore, additional remedial action is necessary to prevent potential exposures and rainwater infiltration at the former saw mill area.

Remedial Action Plan Amendment: To address the contaminated soil and groundwater, DTSC plans to prepare a Remedial Action Plan Amendment and select the appropriate remedy or remedies necessary to mitigate the impact of hazardous substances at the Site. The Remedial Action Plan Amendment will evaluate a range of the alternatives including capping of the former saw mill area, enhanced biodegradation of chemicals in groundwater, long-term groundwater monitoring, and amending the land use covenant.

If you have any questions, please contact Henry Wong of my staff at (510) 540-3770 or henry.wong@dtsc.ca.gov.

Sincerely,

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