# Health Consultation

TENAHA WOOD TREATING

TENAHA, SHELBY COUNTY, TEXAS

EPA FACILITY ID: TXD072691462

SEPTEMBER 30, 2006

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

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In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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# HEALTH CONSULTATION

## TENAHA WOOD TREATING

## TENAHA, SHELBY COUNTY, TEXAS

## EPA FACILITY ID: TXD072691462

Prepared By:

Texas Department of State Health Services
Under Cooperative Agreement with the
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

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# Summary and Statement of Issues

## Background

In September 2004, the Texas Commission on Environmental Quality (TCEQ) asked the Texas Department of State Health Services (DSHS) under cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) to evaluate the potential public health implications of contaminants found on the former Tenaha Wood Treating Site (also formerly known as the Shelby Wood Treating facility). At the time of the request two adults and four children were living on the property; an additional adult male took up residence on the site some time after the request was made.

## Site Description and History

The six-acre property is located at 275 County Road (CR) 4382, approximately one and one-half (1½) miles south of Tenaha, Shelby County, Texas [1]. There is an onsite drainage ditch on the eastern portion of the property; the surrounding properties consist of rural farmland and woodlands. Based on conversations with a previous owner, the subject property began operations as the Shelby Wood Treating facility during the 1950s and was abandoned in 1985 [1, 2]. No buildings or equipment from the former wood treating facility remain on the property with the exception of concrete pillars [3]. The chemicals that were used to preserve wood included copper chromated arsenate (CCA) and pentachlorophenol (PCP).

The United States Environmental Protection Agency (EPA) conducted two limited site assessments in 1984 and 1988. A Site Inspection Prioritization report, prepared by FluorDaniel, Inc., dated September 14, 1994, was submitted to the EPA. The EPA classified the property as a Federal No Further Remedial Action Planned (NFRAP) site, because there was no complete exposure pathway. Because contamination was evident on site (i.e. staining), the site was referred to the Texas Natural Resource Conservation Commission (TNRCC, n/k/a TCEQ) [1].

The current owner purchased the property in 1994: TCEQ began site investigation activities in 1995 [1]. Between 1999 and 2002, the owner had a water well installed and placed two mobile homes on the site. The residential well is within 200 feet of the homes and reportedly supplies water for drinking and other household uses. The terminal depth of the well is 250 feet, based on TCEQ field sampling notes. In July 2004, TCEQ visited the site and noted that the grass cover around the mobile homes was sparse, possibly indicating stressed vegetation from former operations. They also observed children's toys near one of the grass-free areas. As of October 2005, six occupants, including four children, were living on the property. The ages of the children range from six to twelve years [4]. According to the mother, living on the site had not affected the health of her children. A non-intrusive site visit was conducted by TCEQ and DSHS personnel on January 12, 2006. The site was not entered, and observations were made from the roadway. Observations included limited property access, which is controlled by the property owner; and the site is a residential property located in a rural area.

During February 2006, TCEQ began remediation efforts to remove the contaminated soils. The inhabitants of one of the on-site residences, including all four children, have since left the property, and the contaminated portion of the site characterized by stressed vegetation was fenced by TCEQ to prevent access. As of March 2006 one adult male remained on site [5]. The children resided on site for approximately two years.

## Methods

To assess the potential health risks that may be associated with the contaminants found on the site, we compared contaminant concentrations with their media specific health assessment comparison (HAC) values for non-cancer and cancer endpoints. These values are guidelines that specify levels of chemicals in specific environmental media (soil, air, and water) that are considered safe for human contact with respect to identified human endpoints. Non-cancer screening values are generally based on ATSDR's minimal risk levels (MRLs)[1] and EPA's reference doses (RfDs)[2]. Both are based on the assumption that there is an identifiable exposure threshold (both for the individual and for populations) below which there are no observable adverse effects. Thus, MRLs and RfDs are estimates of daily exposures to contaminants that are unlikely to cause adverse non-cancer health effects even if exposure occurs for a lifetime. The HAC values used to evaluate cancer: the cancer risk evaluation guides (CREGs)[3], are based on EPA's chemical-specific cancer slope factors (CSFs)[4] and an estimated excess lifetime risk of developing cancer of one in one million persons exposed for a lifetime. The environmental media evaluation guides (EMEGs) are used as a screening tool to compare site specific soil, water, and/or air concentrations. The EMEGs are derived from the chemical's toxicity and default exposure criteria.

Dioxins are a contaminant of interest at this site. Dioxins consist of several different chemical structures called congeners. The toxicity of individual dioxin congeners varies. As per ATSDR guidelines, the concentrations of individual congeners associated with the site were converted to Toxic Equivalents (TEQs), based on the World Health Organization (WHO) 2005 Toxic Equivalency Factors (TEFs), which were derived from the relationship of the specific dioxin congener's toxicity to the toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD), the most toxic of all congeners [6].

[1] An MRL is a contaminant specific exposure dose below those which might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs generally are based on the most sensitive chemical-induced end point considered to be of relevance to humans.
[2] An RfD is an estimate (with a level of uncertainty from 10 to 1000 times below the level of harmful effects) of a daily exposure to the human population (including sensitive groups) that is likely to be without appreciable risk of deleterious effects during a lifetime.
[3] A CREG is the concentration of a chemical in specific media (air, water or soil) corresponding to an excess estimated lifetime cancer risk of one in one million (1 in 1,000,000) persons exposed for a lifetime.
[4] A CSF is the upper 95th percentile confidence limit of the slope of the dose-response curve and is expressed in unit of measure of $(mg/kg\text{-}day)^{-1}$.

For dioxins we compared site data to ATSDR's screening level of 50 parts per trillion (ppt) (0.05 µg/kg or 0.00005 mg/kg)[5]. This level is based on the ATSDR MRL of 1 picogram 2,3,7,8-TCDD per kilogram body weight per day (pg/kg/day) [7].

Exceeding either a non-cancer or a cancer screening value does not necessarily mean that the contaminant will cause harm; rather it suggests that potential exposure to the contaminant warrants further consideration.

Estimated exposure doses were calculated from average contaminant concentrations for those contaminants which exceeded their respective HAC values. To account for concentration values below the detection limit for the analytical method used, half of the method detection limit (MDL) was used as a proxy concentration. A six-year default exposure duration was assumed for children (age 1 to 6); a seven-year default exposure duration period was assumed for children (age 6 to 12); and a 30-year exposure duration was assumed for adults. These duration periods are conservative: The children lived on the site for approximately two years.

Sample data were provided by the TCEQ. DSHS did not review the analytical packet to determine laboratory adherence to Quality Assurance/Quality Control (QA/QC) protocols. Rather, DSHS is depending on TCEQ's review of QA/QC protocols to be accurate and committed to good common practice.

## Environmental Sampling

TCEQ staff collected seven surface soil, seven sediment, and five ground water samples in July 2004. The samples were analyzed for volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs) including PCP, chlorinated dibenzo-p-dioxin (dioxins), and metals. The soil samples were collected within 200 feet of the two residential dwellings. The sediment samples were collected from nearby wetland areas associated with an intermittent creek [1]. Soil and sediment sample depths are shown on Tables 1 and 2. Two ground water samples were collected from an onsite drinking water well (one sample and one duplicate), one sample was collected from each of two offsite drinking water wells, and one sample was collected from a well used to water cattle.

### *Surface Soil*

Pentachlorophenol, dioxins, antimony, arsenic, chromium, and copper all exceeded their respective HAC values for soil. PCP and arsenic both exceeded their respective non-cancer and cancer screening values; dioxins exceeded ATSDR's screening level for TCDD and TCDD equivalents; chromium exceeded its non-cancer screening value for both children and adults; and antimony and copper each exceeded their respective non-cancer screening values for children (Table 1). There are no cancer screening values for antimony, chromium, or copper.

[5] TCDD equivalents are based on the relative toxicity of the individual dioxin congeners to that of 2,3,7,8-TCDD the most extensively studied and most toxic dioxin compound.

***Sediment***

Sample results did not exceed the health based screening values for PCP, antimony, chromium or copper. Arsenic data were consistent with the USGS documented background levels for this part of the United States (Table 2) [8]. Dioxin TEQs levels exceeded the ATSDR screening level.

Dioxin TEQ values exceeded the ATSDR screening level of 0.00005 mg/kg in three of the seven samples collected with concentrations of 0.000339 mg/kg (collected from 2-6 inches below grade surface [bgs]), 0.000383 mg/kg (collected from 1-6 inches bgs), and 0.0000688 mg/kg (collected from 0-6 inches bgs). The average TEQ (0.000129 mg/kg) was calculated from the seven sample values.

In the absence of site-specific information pertaining to accessibility, it is feasible that the wetland areas may be accessed for recreational use. Exposure dose estimates were calculated based on the average TEQ value for the seven samples. Calculations were based on excursions into the wetland areas 182 days (one-half) of the year, standard body weight, and standard soil ingestion values. The exposure dose estimates were below the chronic, intermediate, and acute MRLs for children aged one to six years, children aged six to twelve years, and adults.

***Ground Water***

No contaminants were identified above their respective ATSDR HAC values; however, the MDL of 0.082 micrograms per liter (µg/L) for 1,2-dibromoethane is above both the most conservative HAC value of 0.02 µg/L and the MCL (0.05 µg/L). There is no evidence available suggesting that this compound was used on the site.

## Discussion

*Pentachlorophenol (PCP)*

Historically, PCP was used as a wood preservative and biocide. By 1984, PCP was no longer available to the general public, and its use was restricted to certified applicators. Exposure to high concentrations of PCP can cause the body's cells to produce excessive heat. It is also considered a probable human carcinogen (based on inadequate human studies but sufficient animal studies) by the EPA and is associated with increased liver, adrenal gland, and nasal tumors [9]. PCP is not classified with respect to its carcinogenicity by the National Toxicology Program (NTP). At this site the most likely way for PCP to enter a person's body is through the digestive tract after incidental ingestion of soil.

Surface soil sample concentrations ranged from below the analytical detection limit (ND or non-detect) to 2,500 mg/kg and exceeded the non-cancer HAC values for children in four of the seven soil samples and the non-cancer HAC value for adults in one of the seven samples. The non-cancer HAC value for PCP is based on an MRL of 0.001 mg/kg/day. This MRL, based on a multigenerational reproductive study of minks continuously fed PCP of unspecified purity in the diet, was derived by dividing the lowest observable adverse effects level (LOAEL) of 1

mg/kg/day by an uncertainty factor of 1,000. The effects associated with the LOAEL included significantly decreased serum thyroxine concentrations in male minks of the first generation and male and female minks of the second generation, as well as, decreased relative thyroid weight in females of the second generation [9].

Based on standard assumptions of body weight and soil ingestion, in the past when children lived on the site, they could have been exposed to PCP at doses above the MRL. Currently, only one male adult is living on the site. A 70 kilogram (kg) male ingesting 100 mg of soil everyday containing an average soil concentration of 394 mg/kg would receive an average daily dose of 0.0006 mg/kg/day; a daily dose 1.6 times lower than the MRL and almost 1,700 times lower than the identified LOAEL (Table 3).

PCP also exceeded its CREG in four of the seven samples. Using the standard assumptions (Table 4) we estimate that for adults, daily exposure to the average concentration found in the soil would result in an increased lifetime risk for developing cancer of three in 100,000. For children aged one to six and six to twelve, we estimate the excess lifetime cancer risks to be five in 100,000 and three in 100,000, respectively. Qualitatively, we would interpret the PCP as posing no apparent increased risk for developing cancer.

In the past, PCP in onsite soil could have posed a public health hazard to children, consisting of non-carcinogenic, transient effects. Even though the full extent of contamination has not been determined, the area with the highest measured concentrations has been fenced; thus, based on available information, we have concluded that the PCP in the soil currently poses no apparent public health hazard.

*Dioxins*

Dioxins have never been manufactured for industrial purposes, but they are commonly found as impurities in other chlorinated chemical mixtures, such as PCP. The most common health effect associated with dioxin exposure is chloracne, although exposure also has been associated with skin rashes and discoloration, excessive body hair, weight loss, liver damage, endocrine disruption, immunosuppression, reproductive damage, and birth defects.

The absorption of dioxins and dioxin-like compounds varies, depending on the lipid solubility of the congener and the media on which it is delivered (i.e. soil, food, or oils) [10]. Dioxins accumulate in fatty tissues, and within five to 14 years, the body will rid itself of half of the accumulated dioxin. Because dioxins persist, they can cause health effects long after the exposure period has ended.

The WHO has determined that dioxins (more specifically 2,3,7,8-TCDD) are a human carcinogen [11]. Additionally, the International Agency for Research on Cancer (IARC) and the National Toxicology Program (NTP) have determined that dioxins are a known human carcinogen. The EPA has classified dioxins as a probable human carcinogen with inadequate human, but sufficient animal, data.

Surface soil TEQ values ranged from 0.000002 mg/kg to 0.148457 mg/kg. Surface soil TEQs exceeded the ATSDR screening level of 0.00005 mg/kg, the chronic EMEG for children (0.00005 mg/kg), and the chronic EMEG for adults (0.0007 mg/kg) for 2,3,7,8-TCDD in five of the seven samples collected from the subject property. Additionally, four of the seven samples exceeded the ATSDR action level of 0.01 mg/kg. Based on the ATSDR guidelines, the action level is a threshold at which public health actions are considered.

At this site the most likely way for dioxins to enter a person's body is through the digestive tract after incidental ingestion of soil. Exposure doses were estimated based on standard body weight and soil ingestion for adults and children, as shown on Table 3. The doses ranged from $6.23 \times 10^{-7}$ mg/kg/day in children ages six to twelve to $7.12 \times 10^{-8}$ mg/kg/day in adults. These estimated doses are greater than the oral MRLs for chronic ($1.0 \times 10^{-9}$ mg/kg/day) and intermediate ($2.0 \times 10^{-8}$ mg/kg/day) exposure. The estimated dose for children also exceeds the MRL for acute ($2.0 \times 10^{-7}$ mg/kg/day) exposure, while the estimated dose for adults does not.

The chronic, intermediate, and acute-duration MRLs were derived from studies which observed the following effects: social changes in monkeys observed pre-natal and during lactation; decreased thymus weights in guinea pigs; and altered hemolytic complement activity in the mice studied, respectively [11]. The MRLs were extrapolated to determine a level at which human health effects are unlikely. For example, a 90-fold uncertainty factor was used to derive the MRL, and the chronic MRL is one to two orders of magnitude below levels at which adverse health effects were observed either experimentally or through epidemiological studies [12].

In the past, the dioxins in soil posed a public health hazard. The children may have been exposed to harmful concentrations. The area with the highest measured concentrations has been fenced to reduce exposure. Based on available information, we have concluded that the dioxins in the soil currently pose no apparent public health hazard. However, the off-site soils have not been fully characterized and are classified as an indeterminate public health hazard.

*Antimony*

Antimony is a naturally occurring metal that when mixed with other metals is used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, and pewter. Antimony sorbs to soils, and people may come in contact with it by inhaling dust particles from or eating soil contaminated with antimony. Limited information is available to determine if antimony will cause cancer or birth defects in humans. At this site the most likely way for antimony to enter a person's body is through the digestive tract after incidental ingestion of soil. Ingestion of antimony has been linked to vomiting and diarrhea both in humans and animals. Ingestion of antimony by animals also has resulted in liver damage [13].

Two of the seven onsite soil samples contained detectable levels of antimony (35.4 mg/kg and 44.6 mg/kg). There are no cancer screening values for antimony; however, both sample data exceeded the non-cancer HAC value for children. Based on available information, the fact that the area with the highest measured concentrations has been fenced and that exposure to the average concentration of antimony found in the soil would not be likely to result in adverse

health effects; we have concluded that the antimony in the soil poses no apparent public health hazard.

*Arsenic*

Arsenic is part of a common wood preservative, CCA. Wood that is treated with CCA is referred to as "pressure-treated." Health effects associated with ingesting low levels of arsenic include fatigue, abnormal heart rhythm, blood-vessel damage resulting in bruising, and impaired nerve function causing a "pins and needles" feeling. Long term exposure causes darkening of the skin and corns or warts on the hands, torso, and feet.

Swallowing arsenic has also been reported to increase the risk of cancer in the liver, bladder, kidneys, prostate, and lungs. Dermal contact can cause skin irritation and redness. There is some evidence that arsenic can result in lower IQ scores in children [14].

The liver converts arsenic to an organic form, which is less harmful. Organic and inorganic arsenic leaves the body in the urine within several days of exposure, although some may remain in the body for months.

At this site the most likely way for people to have been exposed to arsenic was through the incidental ingestion of soil.

Arsenic concentrations in onsite soils ranged from 1.6 mg/kg to 1,850 mg/kg with five of the seven samples exceeding the noncancer screening value for children. Two of the seven samples exceeded the non-cancer HAC value for adults of 200 mg/kg. Although all of the samples exceeded the cancer HAC value, this HAC value is lower than background soil concentrations for this part of the US [8].

In the past, the average concentration of arsenic to which people could have been exposed exceeded the HAC values for both children and adults. Additionally, the estimated lifetime cancer risk calculated for adults and children is greater than $1 \times 10^{-4}$, indicating a low to moderate increased risk, as shown on Table 4. Thus, assuming that the arsenic is 100% bioavailable, in the past the arsenic in the onsite soil could have posed a public health hazard; however, the body does not store arsenic for an extended period of time (i.e. years). Because the area with the highest measured concentrations has been fenced, we have concluded that currently the arsenic in the soil poses no apparent public health hazard.

*Chromium*

Chromium is a naturally occurring element found in several different forms in rocks, animals, plants, soil, and in volcanic dust and gases. The most common forms are chromium (0), trivalent chromium (III) (trivalent chromium), and chromium (VI) (hexavalent chromium). Trivalent chromium occurs naturally and is an essential nutrient required by the human body, whereas chromium (0) and hexavalent chromium are produced by industrial processes. Trivalent chromium can be converted to hexavalent chromium through natural processes and human activities. In general, chromium (VI) is more toxic than chromium (III). At this site the most

likely way for chromium to enter a person's body is through the digestive tract after the incidental ingestion of soil [15].

Accidental or intentional ingestion of extremely high doses of chromium (VI) compounds by humans has resulted in severe respiratory, cardiovascular, gastrointestinal, hematological, hepatic, renal, and neurological effects. Animals exposed to very high doses of chromium (VI) and chromium (III) compounds have exhibited gastrointestinal, hepatic, renal, immunological, neurological, developmental, and reproductive effects. The doses that resulted in these effects in both humans and animals were orders of magnitude greater than those that theoretically could occur at this site. In general, chromium (VI) compounds are more toxic than chromium (III) compounds and chromium (III) is less well absorbed from the gastrointestinal tract than chromium (VI). After ingestion, chromium (VI) is reduced to chromium (III) in the stomach; this reduces its bioavailability and may account for the relatively low oral toxicity of chromium (VI) compared to other routes of exposure.

Total chromium concentrations identified in the soil samples collected from the site ranged from ND to 3,360 mg/kg. There are no cancer screening values for chromium. However, three samples exceeded the non-cancer HAC value for chromium (VI) for children, and two samples exceeded the non-cancer HAC value for adults. These HAC values are based on EPA's RfD for chromium (VI) of 0.003 mg/kg/day. The RfD was derived from a NOAEL identified from one study where male and female rats were supplied with drinking water containing 0.45-11.2 ppm (0.45-11.2 mg/L) hexavalent chromium for one year and another study where male and female rats where given 25 ppm (25 mg/L) chromium. Comparing the soil data to HAC values for hexavalent chromium is very conservative because it is unlikely that all of the chromium in the soil is hexavalent. None of the soil samples exceeded the HAC values for trivalent chromium. Although chromium (VI) is considered to be a carcinogen via the inhalation route, the oral carcinogenicity of chromium (VI) cannot be determined as data suggesting that chromium (VI) is carcinogenic by the oral route of exposure are not available. Based on available information, the fact that the area with the highest measured concentrations has been fenced and that it is not likely that all the chromium found in the soil is of the hexavalent species, we would not expect exposure to the chromium in the soil to result in adverse health effects. Thus, we have concluded that the chromium in the soil poses no apparent public health hazard.

*Copper*

Copper is a naturally occurring metal that has been used to make plumbing pipe and pennies, and it is present in sheet metal. It also has been used to preserve wood, leather, and fabrics. Low levels are required by the body as an essential element, but ingesting high concentrations can cause nausea, vomiting, and diarrhea [16].

Analytical results indicate copper concentrations range from ND to 1,680 mg/kg with three of the seven data values above the detection limit. Although there are no cancer screening values for copper, two of the sample values exceed ATSDR's intermediate non-cancer HAC for children (500 mg/kg). Based on the average concentrations found on the site, the calculated daily dose that children might receive from the incidental ingestion of soil would not be expected to result in adverse health effects. Based on the fact that the area with the highest measured

concentrations has been fenced and that the estimated exposure dose that children might receive is not likely to result in adverse health effects, we have concluded that the copper in the soil poses no apparent public health hazard.

## Child Health Considerations

In communities faced with air, water, or food contamination, the many physical differences between children and adults demand special emphasis. Children could be at greater risk than are adults from certain kinds of exposure to hazardous substances. Children play outdoors and sometimes engage in hand-to-mouth behaviors that increase their exposure potential. Children are shorter than are adults; this means they breathe dust, soil, and vapors close to the ground. A child's lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. If toxic exposure levels are high enough during critical growth stages, the developing body systems of children can sustain permanent damage. Finally, children are dependent on adults for access to housing, for access to medical care, and for risk identification. Thus adults need as much information as possible to make informed decisions regarding their children's health. In estimating the potential public health hazards associated with this site, we considered children when estimating potential exposure.

### Public Health Implications

In the past, the children who lived on the site could have been exposed to contaminants at levels above health based-screening values. Whether actual exposures would or could have resulted in adverse health effects is not known. The children lived on site for approximately two years. Based on conversations with the mother of the children she did not believe that living on the site had affected her children's health [4]. The children received regular check-ups with their physician and health problems (attributable to onsite contaminants) were never reported.

Based on available information we have classified this site and the off-site wetland area as currently posing no apparent public health hazard. This is based on the fact that the property is privately owned, only one person (an adult male) is living on the site, and access to the known contaminants in the soil is restricted by fencing. It is unlikely that the public will be exposed to onsite soil or offsite sediment contamination at levels that would cause adverse health effects. There are uncertainties associated with this conclusion; the greatest uncertainty lies in the fact that the full nature and extent of contamination has not been determined.

## Conclusions

- Based on current conditions and the limited access to the site, the known levels of contaminants on the subject property pose no apparent public health hazard either to the general public or to the one adult who continues to live on the property.

- Sediment contamination was identified in adjacent wetland areas. Estimated exposure doses indicate that the dioxin contamination identified in these areas poses no apparent public health hazard.

- Soil contamination on the subject property has not been fully characterized or delineated, and no off-site soil sample data are available. Based on this information, the exposure to potential contaminants in off-site soils poses an indeterminate public health hazard.

- PCP concentrations on site may have posed a past public health hazard to resident children. However, the non-carcinogenic effects of PCP exposure are transient, and no long term effects are anticipated.

- Calculated TEQs, based on onsite dioxin concentrations may have posed a past public health hazard to resident children and adults. There is no EPA cancer slope factor to estimate increased cancer risk. The actual exposure and potential health effects are indeterminate.

- Based on estimated lifetime cancer risk calculations, the arsenic in onsite soils posed a past public health hazard. The estimated cancer risk for the past exposures to arsenic indicate that the increased lifetime risk of cancer from arsenic exposure is low to moderate. The exposure pathway to arsenic has been eliminated by the fencing of the highly impacted area.

## Recommendations

Texas DSHS recommends that the TCEQ continue investigation and remediation efforts, as currently planned.

# Public Health Action Plan

## Actions Completed

In February 2006, DSHS sent letters to educate the residents about the health effects of the onsite contaminants and advising residents to prevent exposure to the contaminated soils. Subsequent to that letter, TCEQ began remediation efforts by fencing the portion of the property characterized by stressed vegetation and elevated soil contamination. The children no longer live on site, and the potential for exposure to the known contaminated area has been eliminated.

## Actions Planned

- TCEQ has initiated remediation efforts on the site and DSHS will continue to review environmental sampling data and/or other pertinent information as it becomes available.
- Current and former residents should inform their personal physician of the potential exposure to dioxins and arsenic. DSHS will provide additional information to the physicians upon request.
- This Health Consultation will be provided to the public and to local, state, and federal health and environmental agencies.

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## References

1. Texas Commission on Environmental Quality. HRS Documentation Record for Tenaha Wood Treating, Tenaha, Texas: Austin, TX, December 2004.
2. Texas Commission on Environmental Quality. Public Meeting, Tenaha City Hall. January 12, 2006.
3. Texas Department of State Health Services. Personal Communication Susan Prosperie to Gary Hazelwood, Texas Commission on Environmental Quality, Tyler Regional Office. Regarding current site conditions Tenaha Wood Treating. September 2, 2004.
4. Texas Commission on Environmental Quality. Personal Communication Mike Aplin to Michelle Bost Texas Department of State Health Services. Tenaha occupant information. January 27, 2006
5. Texas Commission on Environmental Quality. Personal Communication John Sedberry to Michelle Bost Texas Department of State Health Services. Site update. February 28, 2006.
6. Van den Berg, Martin, Linda S. Birnbaum, Michael Denison, Mike De Vito, William Farland, Mark Feeley, Heidelore Fiedler, Helen Hakansson, Annika Hanberg, Laurie Haws, Martin Rose, Stephen Safe, Dieter Schrenk, Chiharu Tohyama, Angelika Tritscher, Jouko Tuomisto, Mats Tysklind, Nigel Walker, and Richard E. Peterson. The 2005 World Health Organization Re-evaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-like Compounds. ToxSci Advanced Access, July 7, 2006.
7. De Rosa, Christopher T., David Brown, Rosaline Dhara, Woodrow Garrett, Hugh Hansen, James Holler, Dennis Jones, Denise Jordan-Izaguirre, Ralph O'Connor, Hana Pohl, and Charles Xintaras. Dioxin and Dioxin-like Compounds in Soil, Part I: ATSDR Interim Policy Guideline. Toxicology and Industrial Health: 13(6), 1997.
8. Agency for Toxic Substances and Disease Registry. Public Health Assessment Guidance Manual. Atlanta: ATSDR, January 2005.
9. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Pentachlorophenol. Atlanta: ATSDR, September 2001.
10. De Rosa, Christopher T., David Brown, Rosaline Dhara, Woodrow Garrett, Hugh Hansen, James Holler, Dennis Jones, Denise Jordan-Izaguirre, Ralph O'Connor, Hanna Phol, and Charles Xintaras, Dioxin and Dioxin-like Compounds in Soil, Part II: Technical Support Document for ATSDR Interim Policy Guideline. Toxicology and Industrial Health: 13(6), 1997.
11. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Chlorinated Dibenzo-p-dioxins (CDDs). Atlanta: ATSDR, December 1998.
12. HR Pohl, HE Hicks, DE Jones, H Hansen, and CT DeRosa. Public Health Perspectives on Dioxin Risks: Two Decades of Evaluations. Human and Ecological Risk Assessment: 8(2), 2002.

13. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Antimony. Atlanta: ATSDR, December 1992.

14. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. Atlanta: ATSDR, September 2000.

15. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Chromium. Atlanta: ATSDR, September 2000.

16. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Copper. Atlanta: ATSDR, September 2004.

## Certification

This public health consultation was prepared by the Texas Department of State Health Services (DSHS) under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the public health consultation was initiated. Editorial review was completed by the Cooperative Agreement partner.

Technical Project Officer, CAT, CAPEB, DHAC, ATSDR

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

Team Lead, CAT, CAPEB, DHAC, ATSDR

# Appendices

**Appendix A: Acronyms and Abbreviations**

**Appendix B: Tables**

**Appendix A – Acronyms and Abbreviations**

## Acronyms and Abbreviations

| | |
|---|---|
| ATSDR | Agency for Toxic Substances and Disease Registry |
| CCA | Copper chromated arsenate |
| CREG | Carcinogenic Risk Evaluation Guide |
| CSF | Cancer Slope Factor |
| DSHS | Department of State Health Services |
| EMEG | Environmental Media Evaluation Guide |
| EPA | Environmental Protection Agency |
| HAC Value | Health Assessment Comparison Value |
| HRS | Hazard Ranking System |
| LOAEL | Lowest Observable Adverse Effects Level |
| MCL | Maximum Contaminant Level |
| mg/kg | milligrams per kilogram |
| mg/L | milligrams per liter |
| MRL | Minimal Risk Level |
| ND | Non-detect or not detected |
| NFRAP | No Further Remedial Action Planned |
| NOAEL | No Observable Adverse Effects Level |
| NPL | National Priorities List |
| NTP | National Toxicology Program |
| PCP | Pentachlorophenol |
| QA/QC | Quality Assurance/Quality Control |
| RfD | Reference Dose |
| RMEG | Reference Dose Media Evaluation Guide |
| SVOCs | Semi-Volatile Organic Compounds |
| TCDD | 2,3,7,8-tetrachlorodibenzo-p-dioxin |
| TCEQ | Texas Commission on Environmental Quality |
| TDH | Texas Department of Health |
| TEF | Toxic Equivalency Factors |
| TEQ | Toxic Equivalents |
| TNRCC | Texas Natural Resource Conservation Commission |
| VOCs | Volatile Organic Compounds |

# Tables

**Table 1: Tenaha Surface Soil Sample Results and Respective Health-based Screening Values (mg/kg)**

| **Sample Identifier**<br>**Sample Depth (inches)** | **SO-01**<br>1-5 | **SO-02**<br>1-6 | **SO-03**<br>2-6 | **SO-04**<br>2-7 | **SO-05**<br>2-7 | **SO-06**<br>1-6 | **SO-07**<br>3-5 | **Average** | **HAC Values** |
|---|---|---|---|---|---|---|---|---|---|
| Pentachlorophenol | <0.72 | <0.69 | **86** | **89** | **82** | **2,500** | <3.6 | **394.23** | 6 CREG 50/700 chrEMEG |
| Total TCDD TEQ | 0.000002 | 0.000003 | **0.042854** | **0.091057** | **0.081306** | **0.148457** | **0.000394** | **0.052010** | 0.00005 ATSDR Screening Level for TCDD & TCDD Equivalents<br>0.00005/0.0007 chrEMEG c/a |
| Antimony | <0.21 | <0.049 | 0.038J | **44.6** | **35.4** | 1.2 | 0.37J | 11.73 | 20/300 RMEGc/a |
| Arsenic | **2.5** | **1.6** | **106** | **1770** | **1850** | **188** | **20.7** | **562.7** | 0.5 CREG<br>20/200 chrEMEGc/a |
| Chromium | 11.1J | 7.0J | 90.8J | **2850J** | **3360J** | **259J** | 42.2J | **945.7** | $Cr^{6+}$ 200/2000 RMEGc/a<br>$Cr^{3+}$ 80000/1000000 RMEGc/a |
| Copper | 11.6J | 0.92J | 32.0J | **1300J** | **1680J** | **75.6J** | 12.0J | **516.7** | 20/500/7000 intEMEGc/a |

J – Estimated
HAC – Health Assessment Comparison Value
mg/kg – milligrams per kilogram
CREG – Cancer Risk Evaluation Guide
chrEMEGc/a – Chronic Environmental Media Evaluation Guide for a child/for an adult
RMEGc/a – Reference Dose based Media Evaluation Guide for a child/for an adult
intEMEGc/a – Intermediate Environmental Media Evaluation for a child/for an adult
Average background metals levels in soil east of the 96$^{th}$ meridian from the US Geological Survey (Arsenic 4.8 mg/kg; Chromium 33 mg/kg; Copper 13 mg/kg)
**Bold** values were detected above the most conservative screening value

**Table 2: Tenaha Sediment Sample Results and Respective Health-based Screening Values (mg/kg)**

| **Sample Identifier**<br>**Sample Depth (inches)** | **SE-01**<br>2-6 | **SE-02**<br>2-5 | **SE-03**<br>1-6 | **SE-04**<br>0-4 | **SE-05**<br>1-6 | **SE-06**<br>2-6 | **SE-07**<br>2-6 | **Average** | **HAC Values** |
|---|---|---|---|---|---|---|---|---|---|
| Pentachlorophenol | <0.91 | <0.81 | <0.94 | <0.93 | <0.89 | <0.98 | <0.91 | 0.46 | 6 CREG 50/700 chrEMEGc/a |
| Total TCDD TEQ | **0.000339** | 0.000013 | **0.000384** | **0.00007** | 0.000032 | 0.000036 | 0.000032 | **0.00013** | 0.00005 ATSDR Screening Level for TCDD & TCDD Equivalents<br>0.00005/0.0007 chrEMEG c/a |
| Antimony | <0.25 | <0.2 | <0.23 | <0.19 | <0.17 | <0.18 | <0.21 | 0.10 | 20/300 RMEGc/a |
| Arsenic* | **4.9**** | **2.3** | **8.5** | **4.6** | **4.9** | **3.3** | **2.8** | **4.5** | 0.5 CREG<br>20/200 chrEMEGc/a |
| Chromium* | 13.9 | 7.8 | 20.9 | 9.7 | 14.7 | 12.1 | 9.5 | 12.7 | $Cr^{6+}$ 200/2000 RMEGc/a<br>$Cr^{3+}$ 80000/1000000 RMEGc/a |
| Copper* | 16.2 | 15.8 | 9.3 | 5.1 | 5.6 | 4.9 | 4.1 | 8.7 | 20/500/7000 intEMEGc/a |

HAC – Health Assessment Comparison Value
mg/kg – milligrams per kilogram
CREG – Cancer Risk Evaluation Guide
chrEMEGc/a – Chronic Environmental Media Evaluation Guide for a child/for an adult
RMEGc/a – Reference Dose based Media Evaluation Guide for a child/for an adult
intEMEGc/a – Intermediate Environmental Media Evaluation Guide for a child/for an adult
*Average background metals levels in soil east of the 96th meridian from the US Geological Survey (Arsenic 4.8 mg/kg; Chromium 33 mg/kg; Copper 13 mg/kg)
****Bold** values were detected above the most conservative screening value

## Table 3: Estimated Doses for Resident Children at the Tenaha Site, Based on Average Chemical Concentrations

**Estimated Doses for Children, Age 1-6**

| | PCP | Arsenic | Copper | Dioxins | Chromium | Antimony |
|---|---|---|---|---|---|---|
| dose=C*IR*CF*AF*EF/BW (mg/kg-d) | 4.73E-03 | 6.74E-03 | 6.19E-03 | 6.23E-07 | 1.13E-02 | 1.40E-04 |
| C=contaminant concentration (mg/kg) | 394.23 | 562.7 | 516.7 | 0.052010 | 945.7 | 11.73 |
| IR=intake rate of soil (mg/day) | 200 | 200 | 200 | 200 | 200 | 200 |
| CF=conversion factor from kg to mg soil | 0.000001 | 0.000001 | 0.000001 | 0.000001 | 0.000001 | 0.000001 |
| AF=bioavailability factor (%, assumed 100%) | 1 | 1 | 1 | 1 | 1 | 1 |
| EF=exposure factor (unitless) | 0.95890411 | 0.95890411 | 0.9589041 | 0.9589041 | 0.9589041 | 0.95890411 |
| EF=(F*ED)/AT | | | | | | |
| F=frequency (d/year) | 350 | 350 | 350 | 350 | 350 | 350 |
| ED=exposure duration (years) | 6 | 6 | 6 | 6 | 6 | 6 |
| AT=averaging time (ED*365d/year) | 2190 | 2190 | 2190 | 2190 | 2190 | 2190 |
| BW=body weight (kg) | 16 | 16 | 16 | 16 | 16 | 16 |

**Estimated Doses for Children, Age 6-12**

| | PCP | Arsenic | Copper | Dioxins | Chromium | Antimony |
|---|---|---|---|---|---|---|
| dose=C*IR*CF*AF*EF/BW (mg/kg-d) | 2.29E-03 | 3.27E-03 | 3.00E-03 | 3.02E-07 | 5.50E-03 | 6.80E-05 |
| C=contaminant concentration (mg/kg) | 394.23 | 562.7 | 516.7 | 0.052010 | 945.7 | 11.73 |
| IR=intake rate of soil (mg/day) | 200 | 200 | 200 | 200 | 200 | 200 |
| CF=conversion factor from kg to mg soil | 0.000001 | 0.000001 | 0.000001 | 0.000001 | 0.000001 | 0.000001 |
| AF=bioavailability factor (%, assumed 100%) | 1 | 1 | 1 | 1 | 1 | 1 |
| EF=exposure factor (unitless) | 0.95890411 | 0.95890411 | 0.9589041 | 0.9589041 | 0.9589041 | 0.95890411 |
| EF=(F*ED)/AT | | | | | | |
| F=frequency (d/year) | 350 | 350 | 350 | 350 | 350 | 350 |
| ED=exposure duration (years) | 7 | 7 | 7 | 7 | 7 | 7 |
| AT=averaging time (ED*365d/year) | 2555 | 2555 | 2555 | 2555 | 2555 | 2555 |
| BW=body weight (kg) | 33 | 33 | 33 | 33 | 33 | 33 |

**Estimated Doses for Adults**

| | PCP | Arsenic | Copper | Dioxins | Chromium | Antimony |
|---|---|---|---|---|---|---|
| dose=C*IR*CF*AF*EF/BW (mg/kg-d) | 5.40E-04 | 7.71E-04 | 7.08E-04 | 7.12E-08 | 1.30E-03 | 1.60E-05 |
| C=contaminant concentration (mg/kg) | 394.23 | 562.7 | 516.7 | 0.052010 | 945.7 | 11.73 |
| IR=intake rate of soil (mg/day) | 100 | 100 | 100 | 100 | 100 | 100 |
| CF=conversion factor from kg to mg soil | 0.000001 | 0.000001 | 0.000001 | 0.000001 | 0.000001 | 0.000001 |
| AF=bioavailability factor (%, assumed 100%) | 1 | 1 | 1 | 1 | 1 | 1 |
| EF=exposure factor (unitless) | 0.95890411 | 0.95890411 | 0.9589041 | 0.9589041 | 0.9589041 | 0.95890411 |
| EF=(F*ED)/AT | | | | | | |
| F=frequency (d/year) | 350 | 350 | 350 | 350 | 350 | 350 |
| ED=exposure duration (years) | 30 | 30 | 30 | 30 | 30 | 30 |
| AT=averaging time (ED*365d/year) | 10950 | 10950 | 10950 | 10950 | 10950 | 10950 |
| BW=body weight (kg) | 70 | 70 | 70 | 70 | 70 | 70 |

## Table 4: Calculated Estimated Cancer Risk for Resident Adults and Children at the Tenaha Site, Based on Average Chemical Concentrations

**Cancer Risk Associated with PCP Exposure at the Tenaha Site**

| | Children, ages 1-6 | Children, ages 6-12 | Adult |
|---|---|---|---|
| ER=CSF*dose, estimated theoretical risk (unitless) | 4.86E-05 | 2.75E-05 | 2.78E-05 |
| dose=C*IR*CF*AF*EF/BW | 4.05E-04 | 2.29E-04 | 2.31E-04 |
| C=contaminant concentration (mg/kg) | 394.23 | 394.23 | 394.23 |
| IR=intake rate of soil (mg/day) | 200 | 200 | 100 |
| CF=conversion factor from kg to mg soil | 0.000001 | 0.000001 | 0.000001 |
| AF=bioavailability factor (%, assumed 100% or 1) | 1 | 1 | 1 |
| EF=exposure factor (unitless) | 0.082191781 | 0.095890411 | 0.410958904 |
| EF=(F*ED)/AT | | | |
| F=frequency (d/year) | 350 | 350 | 350 |
| ED=exposure duration (years) | 6 | 7 | 30 |
| AT=averaging time (70y*365d/year) | 25550 | 25550 | 25550 |
| BW=body weight (kg) | 16 | 33 | 70 |
| CSF=cancer slope factor (mg/kg/d)-1 | 0.12 | 0.12 | 0.12 |

**Cancer Risk Associated with Arsenic Exposure at the Tenaha Site**

| | Children, ages 1-6 | Children, ages 6-12 | Adult |
|---|---|---|---|
| ER=CSF*dose, estimated theoretical risk (unitless) | 8.67E-04 | 4.91E-04 | 4.96E-04 |
| dose=C*IR*CF*AF*EF/BW | 5.78E-04 | 3.27E-04 | 3.30E-04 |
| C=contaminant concentration (mg/kg) | 562.7 | 562.7 | 562.7 |
| IR=intake rate of soil (mg/day) | 200 | 200 | 100 |
| CF=conversion factor from kg to mg soil | 0.000001 | 0.000001 | 0.000001 |
| AF=bioavailability factor (%, assumed 100% or 1) | 1 | 1 | 1 |
| EF=exposure factor (unitless) | 0.082191781 | 0.095890411 | 0.410958904 |
| EF=(F*ED)/AT | | | |
| F=frequency (d/year) | 350 | 350 | 350 |
| ED=exposure duration (years) | 6 | 7 | 30 |
| AT=averaging time (70y*365d/year) | 25550 | 25550 | 25550 |
| BW=body weight (kg) | 16 | 33 | 70 |
| CSF=cancer slope factor (mg/kg/d)-1 | 1.5 | 1.5 | 1.5 |