

Health Consultation

SCHLAGE LOCK COMPANY

SECURITY, EL PASO COUNTY, COLORADO

**EXPOSURE AND HEALTH EFFECTS EVALUATION OF PCE
CONTAMINATION IN WILLOW SPRINGS PONDS**

EPA FACILITY ID: COD082657420

AUGUST 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**

Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

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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Prepared by:

Colorado Department of Public Health and
Environment

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

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Foreword

The Colorado Department of Public Health and Environment's (CDPHE) Environmental Epidemiology Section has prepared this health consultation in cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR). ATSDR is part of the US Department of Health and Human Services and is the principal federal public health agency responsible for the health issues related to hazardous waste. This health consultation was prepared in accordance with the methodologies and guidelines developed by ATSDR.

The purpose of this health consultation is to identify and prevent harmful health effects resulting from exposure to hazardous substances in the environment. Health consultations focus on health issues associated with specific exposures so that the state or local department of public health can respond quickly to requests from concerned citizens or agencies regarding health information on hazardous substances. The Environmental Epidemiology Section (EES) evaluates sampling data collected from a hazardous waste site, determines whether exposures have occurred or could occur in the future, reports any potential harmful effects, and then recommends actions to protect public health. The findings in this report are relevant to conditions at the site during the time this health consultation was conducted and should not necessarily be relied upon if site conditions or land use changes in the future.

For additional information or questions regarding the contents of this health consultation or the Environmental Epidemiology Section, please contact the health assessor who prepared this document:

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Summary and Statement of Issues

In late 1987, Schlage Lock Company, located in Security, Colorado, discovered that the groundwater beneath their manufacturing facility was contaminated with the chemical solvent tetrachloroethylene or perchloroethylene (PCE). Schlage utilized the solvent during production of door locks and related hardware as a metal cleaner and degreaser from 1977-1992. Improper disposal and/or storage of spent PCE led to contaminated soil beneath the site. PCE then leached through the subsurface soil to an underlying aquifer. The contaminated groundwater beneath the facility eventually migrated into the Widefield Aquifer, which is a major source of drinking water for the surrounding community. PCE was discovered in the first municipal drinking water system in 1990 and an environmental quality investigation began.

To date, Schlage and other stakeholders have collected a large amount of environmental data to determine the extent of contamination and to guide remedial work plans for removing PCE from the environment. At the request of a concerned citizen and the Colorado Department of Public Health and Environment's (CDPHE) Hazardous Waste and Waste Management Division (HWWMD), the Department's Environmental Epidemiology Section (EES) is conducting an evaluation of the public health impact in the area affected by PCE contamination. The purpose of this evaluation is to identify any prior, existing, or potential health impacts from exposure to PCE contamination originating in the Widefield Aquifer.

This evaluation has been divided into specific focus areas to more accurately assess each exposure pathway from a variety of environmental media and conditions. The PCE plume covers a large area and exposure conditions vary dramatically amongst the potentially exposed population. A health consultation has been performed on each of the three public water supply systems that draw water from the Widefield Aquifer including Fountain, Security, and Widefield municipal water districts. Private residential wells, which also draw water from the Widefield Aquifer, will be addressed in a separate health consultation. Indoor Air quality in homes located above the PCE plume will also be evaluated in a health consultation. This particular health consultation was completed to evaluate exposure to PCE contamination in Willow Springs Ponds (WSP), located within Fountain Creek Regional Park.

WSP are located at the distal extent of the PCE plume ([Figure A1](#)). The Widefield Aquifer is the source of water for these spring-fed ponds. The first evidence of PCE contamination in the ponds occurred on August 21, 1996. Additional investigation into the contamination of the ponds revealed that PCE was also present within fish tissue at levels of potential concern.

As a result, the El Paso County Board of Commissioners closed WSP to fishing on September 10, 1997. In an effort to reduce illegal fishing in the ponds, the parking lots adjacent to the ponds were closed from 2000-2005. In 2000, parks equipment such as playgrounds, a fish cleaning station, and picnic tables were removed from the area. The ponds remained closed as of the date of this publication.

Three possible exposure scenarios were identified in this health consultation. Scenario 1 includes fish consumption and swimming/wading in WSP prior to the ponds closure in

September 1997. Scenario 2 covers *trespassing* exposures from swimming/wading and fish consumption. Scenario 3 combines the aforementioned scenarios based on the conservative assumption that individuals were using the ponds prior to closure and continued to trespass onto the property and use the ponds after they were closed to the general public (albeit at a lower rate). These exposure scenarios account for past and current exposures to PCE from WSP. The exposure dose estimations that were performed for these scenarios do not indicate a significant level of health concern and are considered no apparent public health hazards.

Future fish consumption from WSP was evaluated by comparing the latest available fish tissue data from 2004 to conservative health-based Fish Consumption Limit Guidelines under which no adverse health effects are expected. Based on the latest available fish tissue sampling data, an elevated risk for some ingestion rate categories was observed. El Paso County officials have recently been discussing reopening Willow Springs Ponds to the general public. However, it is still unclear when WSP will be reopened and what the fish tissue levels of PCE may be at that time. More fish data is needed to ensure that the ponds are “safe” for fish consumption. Therefore, future exposures to PCE from WSP are considered an indeterminate public health hazard.

Background

The purpose of this health consultation is to evaluate exposure to tetrachloroethylene (PCE) within Willow Springs Ponds, located in Fountain, Colorado. Fish Consumption and dermal exposure to PCE while swimming are evaluated in this document.

Tetrachloroethylene

Tetrachloroethylene is an organic solvent, which consists of carbon and chlorine atoms. Tetrachloroethylene is primarily used as a chemical intermediate in the production of chlorofluorocarbons. It is also known by the names perchloroethylene (PCE), perc, tetrachloroethene, or ethylene tetrachloride. Throughout the remainder of this document, tetrachloroethylene will be referred to as PCE. Other common uses of PCE are in the dry-cleaning and textile production industries, where PCE serves as a cleaner or degreaser. Individuals may be exposed to PCE in a variety of ways including household chemicals, dry-cleaned clothes, occupational exposures, or environmental contaminated media (ATSDR 1997). [Appendix H2](#) contains an ATSDR fact sheet with additional information on PCE.

The main health outcome of concern amongst the Security-Widefield community related to PCE exposure appears to be cancer. At the current time, the International Agency for Cancer Research (IARC) has classified PCE as a Group 2a carcinogen (IARC 1995). This category indicates that the substance is “probably carcinogenic to humans” based on sufficient experimental animal data and a limited amount of human data. The U.S. Environmental Protection Agency (EPA) is currently reviewing toxicity information on PCE and no cancer classification is available in the Integrated Risk Information System (IRIS) (EPA IRIS 1998).

Site History

The Schlage Lock Company (Schlage), located at 3899 Hancock Expressway, Security, Colorado began operations manufacturing door locks and related hardware in August 1977. From late 1977 until mid 1992, Schlage used PCE as a metal cleaner and degreaser. In mid-July 1987, Schlage discovered PCE contamination in the subsurface soil on their property during excavation for plant expansion. A preliminary investigation, conducted in 1987, revealed that the PCE had leached down to groundwater beneath the site. It was later found that the contaminant had migrated into the Widefield Aquifer, the primary source of drinking water for the surrounding communities.

The plume of PCE-contaminated groundwater currently extends from the Schlage Lock facility in a west-southwest direction below the Little Johnson Reservoir, then turns and proceeds south-southeast as it intersects with the Widefield Aquifer, just south of Bradley Road ([Figure A1](#)). The contaminant plume then travels within the Widefield Aquifer towards WSP, which is the distal extent of the PCE plume. The shape of the contaminant plume is constrained by paleo-channels in bedrock and channel deposits in the Widefield Aquifer. The overall length of the plume is approximately four and a half miles.

Following the identification of subsurface PCE contamination, a variety of remedial measures have taken place to remove and control the migration of PCE through the

environment. Soil Vapor Extraction (SVE) systems were installed in two source areas at the Schlage facility in 1989 and in a third source area in 2000. In 1990, Schlage installed an on-site groundwater recovery and treatment system to treat PCE-contaminated groundwater and in 1992, they began operation of an additional system between their property and the former Little Johnson Reservoir. The treated water is discharged under a permit to the Security Water and Sanitation District sanitary sewer.

The levels of PCE in the Widefield Aquifer appear to be decreasing. In 1999, maximum concentrations of PCE within the plume were over 1,000 ppb in the vicinity of Little Johnson Reservoir, less than 100 ppb south of Bradley Road, and less than 50 ppb south of Fontaine Boulevard. Maximum PCE concentrations detected during the 2nd quarter of 2004 were below 800 ppb in the vicinity of Little Johnson Reservoir, less than 50 ppb south of Bradley Road, and less than 10 ppb south of Fontaine Boulevard. An upgraded groundwater remediation system, described as the Bradley Road/Little Johnson Reservoir Groundwater Recovery, Treatment, and Injection System, has been operating since 1999. The system is designed to halt any further movement of contaminated groundwater from the aquifer beneath the facility into the Widefield aquifer.

Willow Springs Ponds

Willow Springs Ponds (WSP) are located within Fountain Creek Regional Park, which lies approximately 0.25 mile southeast of the Interstate 25 and CO Highway 16 intersection in El Paso County, Colorado (ESC 2003). In addition to the fishing ponds, the park consists of tennis, basketball, and volleyball courts; picnic pavilions, trails, and a playground (EPCPD 1999).

Willow Springs Pond 1 is the northernmost and larger of the two ponds ([Figure A2](#)). Pond 1 was constructed approximately 30 years ago as a gravel pit. The pond is unlined and covers an area of approximately 5.6 acres with a maximum depth of 12 feet (ESC 2003). In 1988, Divers Reef Inc. investigated the groundwater flow from the Widefield Aquifer into pond 1. They estimated that groundwater flows into the pond at a rate of 2 cubic feet per second, predominately along the northeast embankment (ESC 2003). Water from Pond 1 will periodically discharge to Fountain Creek during overflow. Fountain Creek is not expected to be significantly impacted by overflow and will not be evaluated further in this assessment.

Willow Springs Pond 2 is located to the south of pond 1 and covers an area of approximately 1.8 acres. Pond 2 has a maximum depth of 5 feet. It was constructed in 1989 and is lined with bentonite. However, the integrity of the liner is unknown. Pond 2 receives water from Pond 1 via a screened connecting pipe located on the south end of Pond 1.

PCE was initially detected in Pond 1 on August 21, 1996 at an approximate concentration of 2.2 ppb. The applicable Colorado Surface Water Standard for this body of water was initially 5 ppb. However in 1999, El Paso County officials petitioned the Water Quality Control Commission to apply a surface water standard to WSP that corresponds to a

PCE-contaminated surface water body, which is used for human water consumption and fish ingestion. The petition was accepted and the new standard for WSP became 0.8 ppb. This standard was lowered to 0.69 ppb effective August 31, 2005. Further environmental sampling indicated that PCE was also accumulating in fish tissue at levels of potential concern. Subsequently, the El Paso County Board of Commissioners closed the ponds to all fishing on September 10, 1997 pending further fish sampling and analysis. WSP remained closed at the time of this publication.

To restore water quality within WSP to an acceptable level, Schlage installed a 25-horsepower mechanical aerator on February 9, 1999. Aeration cycles the pond water from bottom to the top, which allows PCE to vaporize into the atmosphere while also oxygenating the water. Since the original aerator was installed, the PCE concentration in Pond 1 water has been monitored on a monthly basis. The original aerator was replaced on July 9, 2002 with another 25-horsepower aerator and a second 25-hp aerator was added on July 29, 2003. Contaminated groundwater enters on the north side of Pond 1, is treated by aeration, and then exits to Pond 2 on the south by the connecting pipe. Thus, it is expected that PCE concentration in Pond 2 would not exceed the concentration in Pond 1. In addition, the pipe connecting Pond 1 and 2 has been plugged and Pond 2 has recently degraded to a swamp/wetland environment.

Recently, El Paso County Commissioners have been discussing reopening Willow Springs Ponds to the public. The ponds have served as valuable assets to the county in the past, and officials would like to see them returned to service as a functional recreational area.

Demographics

WSP was a popular recreational area before its closure in 1997. The most frequent users are likely those individuals that live within a close proximity to Fountain Creek Regional Park. U.S. Census 2000 data for this area does not possess any striking demographic characteristics that would normally have an effect on this evaluation. However, El Paso County health officials have raised concerns that a substantial Asian population exists in the area that may have used WSP for subsistence fishing before the closure. The overall percentage of Asians within El Paso County is approximately 2.5 percent or 13,099 individuals (U.S. Census 2000, Population of one race, Asian alone). Moreover, some census tracts near WSP were in the highest tier of percent Asian of total tract population for all census tracts in El Paso County. [Figure A3](#) is a geographic information system (GIS) graphic depiction of percent Asian population by census tract in El Paso County.

Subsistence fishing means that the fisher catches and consumes fish on a regular basis to sustain life. Subsistence fishers, therefore, consume a larger amount of potentially contaminated fish more frequently than the general population. Some Asian populations are known to practice subsistence fishing and subsistence fishing will be evaluated in this assessment. Based on the limited amount of information currently available on the historical use of WSP for fish consumption, subsistence ingestion rates and the

subsequent exposure doses may overestimate or underestimate any actual exposures that have occurred or could occur in the future.

Community Concerns

Community health concerns regarding the PCE contamination within the Widefield Aquifer were solicited and documented in the “Community Involvement and Health Issues Communication Plan” (CDPHE 2004). In addition, Schlage Lock and the Hazardous Waste and Waste Management Division (HWWMD) at CDPHE have also conducted public involvement activities in the affected communities. Their findings were documented in the “Community Involvement Plan for the Schlage Lock Company Site” (Schlage 2001). Community concerns from both documents are summarized below.

Previously Identified Community Concerns (Schlage 2001):

- Safety of the drinking water supply,
- Property Values,
- Progress on the Willow Springs Ponds remediation, and
- Testing of pumping wells west of U.S. Highways 85 and 87.

Current Community Concerns (CDPHE 2004):

- The possibility of PCE exposure causing brain cancer, lymphatic cancer, or other types of cancer,
- The possibility of PCE exposure resulting in respiratory problems, and
- The health of domestic dogs that have swam in Willow Springs Ponds.

The primary health concerns within the community from exposure to PCE appear to be cancer and other non-carcinogenic health effects, such as respiratory problems. The intent of this health consultation is to evaluate any potential adverse human health effects, including cancer, from exposure to PCE contamination from WSP. The drinking water quality of all municipal water systems that were affected by PCE contamination within the Widefield Aquifer has been evaluated in separate health consultations. Please see the “[Public Health Action Plan](#)” section of this document for a list of all other health consultations available on this site.

Discussion

Evaluation Process

The process used to reach the conclusions and recommendations contained within this document is summarized here and presented in greater detail in Appendices B-F. The initial steps of the assessment process involve screening the available environmental data for contaminants and then comparing this information to conservative, health-based environmental guidelines. Exposures to contaminated sources below the environmental guidelines are not expected to result in adverse or harmful health effects. If the concentration of a particular contaminant is above the chosen environmental guideline, the contaminant is normally retained for further analysis. However exceeding the screening value does not necessarily mean that the contaminant poses a public health

hazard, only that further evaluation may be necessary. ATSDR and CDPHE's Environmental Epidemiology Section also consider sampling location, data quality, exposure probability, frequency and duration; and community health concerns in determining which contaminants to evaluate further.

If the contaminant is selected for extended evaluation, the next step is to identify pathways of probable exposure that could pose a hazard. Simply having the substance present in the environment does not necessarily mean that people will come into contact with it and subsequently experience adverse health effects. An exposure pathway consists of five elements:

- a source of contamination (Schlage Lock Source Areas 1,2,3),
- a contaminated environmental medium and transport mechanism (e.g., Surface Water, Widefield Aquifer),
- a point of exposure (e.g., Willow Springs Ponds),
- a route of exposure (e.g., Consumption of fish), and
- a receptor population (e.g., People who catch and consume fish from Willow Springs Ponds).

Exposure pathways are classified as either complete, potential, or eliminated. Only complete exposure pathways can be fully evaluated and characterized to determine the public health implications. Potential exposure pathways exist when one or more of the elements of an exposure pathway may not be present, but the available information is insufficient to eliminate or exclude the element. Depending on the amount of available evidence, potential pathways may also be retained for further analysis. Eliminated exposure pathways, where one or more of the elements is absent and no information exists to suggest this element has ever existed, require no further evaluation.

Contaminants with completed or potential exposure pathways are then analyzed by calculating adult and child exposure doses in the contaminated environmental media present on-site. Exposure doses are estimates of the concentration of contaminants that people may come into contact with or be exposed to under specified exposure conditions. These exposure doses are compared to the appropriate health guidelines for the contaminant. Health guideline values are considered acceptable or "safe" doses; that is, health effects are not likely below this level. If the exposure dose for a contaminant is greater than the health guideline, then the exposure is compared to known health effect levels contained within ATSDR's Toxicological Profiles or other scientific literature. If the contaminant is a carcinogen, the cancer risk is also estimated.

Data Used

WSP water and fish tissue data were the two primary data sets utilized for this assessment. Schlage Lock and their environmental contractors have collected water data from the ponds for PCE since 1996 and on a monthly basis since February 1999. Fish tissue data was collected on 13 joint sampling events between 1997-2004 with representatives from Schlage Lock Company, El Paso County, and the Colorado Division of Wildlife (CDOW) in attendance. Both sets of data used in this health consultation are discussed below.

WSP Water Data

Schlage and their environmental contractors collected the water quality data used in this assessment. The data was gathered and organized into spreadsheets for analysis. Water data was then screened for elevated PCE concentrations using the applicable Colorado Surface Water Standard of 0.69 as a comparison value (CV). This standard was initially developed by the EPA as a Lifetime Health Advisory for water quality (Recommended National Water Quality Criteria) and was adopted by the CDPHE Water Quality Control Commission effective 12/31/2005 (CDPHE 2005). The CV selected for the assessment is an ambient water quality standard, which considers the potential human health risks from consuming fish and drinking water from a PCE-contaminated surface water body. WSP have never been used as a drinking water source, and the selection of this value is conservative. Thus, adverse health effects are not expected from exposures to PCE in WSP occurring below this concentration.

Water Quality data was divided into three groups to characterize the effects of treatment within WSP. The first data group (Phase 1) includes water quality data prior to the initial installation of the mechanical aerator in February 1999. The second data group (Phase 2) included the time period between the initial installation of the first aerator through the installation of the second aerator in July 2003. The third group (Phase 3) included the time period after both aerators were installed and operating (July 2003-present). Water quality data collected from WSP Pond 1 is summarized and presented below in Table 1.

Table 1: Willow Springs Pond 1 Water Data Summary 1996-2005

| Year | Average PCE Concentration | Median | Maximum | Minimum | Colorado Surface Water Standard for WSP | Sample Count |
|----------------|---------------------------|--------|---------|---------|---|--------------|
| Phase 1 | | | | | | |
| 1996 | 2.15 | 2.1 | 3.3 | 1.1 | 5 | 4 |
| 1997 | 2.12 | 1.9 | 3.8 | 1.1 | 5 | 5 |
| Phase 2 | | | | | | |
| 1998 | 2.12 | 2.15 | 2.3 | 1.9 | 5 | 6 |
| 1999 | 0.88 | 0.7 | 2.8 | 0.25 | 0.8 | 70 |
| 2000 | 0.92 | 0.84 | 2 | 0.14 | 0.8 | 37 |
| 2001 | 1.53 | 1 | 6.9 | 0.25 | 0.8 | 33 |
| 2002 | 0.76 | 0.84 | 1.1 | 0.33 | 0.8 | 12 |
| Phase 3 | | | | | | |
| 2003 | 0.64 | 0.59 | 0.93 | 0.34 | 0.8 | 17 |
| 2004 | 0.37 | 0.34 | 0.55 | 0.25 | 0.8 | 12 |
| 2005 | 0.39 | 0.32 | 1.1 | 0.16 | 0.69 | 20 |

*All Concentrations reported in µg/L or parts per billion (ppb)

Source: TRI 1996-2000, ESC 2000-2005

Contaminated groundwater from the Widefield Aquifer enters Pond 1 predominantly along the northeast embankment (ESC 2003). The groundwater then moves through Pond 1 where it is treated by mechanical aeration. Pond 2 receives water from Pond 1 via a screened connecting pipe located on the south end of Pond 1. Therefore, PCE in Pond 2 water is not expected to exceed the concentration of PCE in Pond 1. In recent years the connection to Pond 2 from Pond 1 was closed and surface water no longer flows into Pond 2. Pond 2 has since degraded to a wetland environment that is unsuitable for fishing. Therefore, Pond 2 water data was not extensively reviewed or utilized in this assessment. Water data collected from WSP Pond 2 is summarized and presented in Appendix A, [Table A1](#).

WSP Fish Tissue Data

Fish tissue data was collected by the CDOW on 13 separate sampling events using gill nets as the capture mechanism. The samples were sent to Battelle Laboratories in Duxbury, MA for chemical analysis using EPA Method 8260M. The data was confirmed and inserted into spreadsheets for analyses. The PCE concentration in fish tissue was then screened against a health-conservative environmental guideline, under which no adverse health effects are expected. In this case, a conservative Comparison Value (CV), or screening guideline for the general population, of 5.8 parts per billion (ppb) for PCE in fish tissue was selected. EPA Region 3 developed the CV used in this assessment as a Risk-Based Concentration (RBC) for fish consumption, which accounts for theoretical carcinogenic health effects (EPA 2006). Specifically, the RBC calculation is based on the consumption of 54 grams of fish per day for the average adult (70 kg) over a period of thirty years with a 10^{-6} Risk Level (RL). A RL of 10^{-6} implies that no more than 1 excess theoretical cancer case out of a million people is likely to result from exposure to concentrations below this level (provided estimated exposure conditions represent actual exposure conditions).

For this assessment, fish tissue samples were also roughly organized by trophic levels and species. The major species classifications are Sunfish, Bass, Catfish, Suckers, and Trout. There is an insufficient amount of data in each species class to perform detailed statistical analyses to quantify differences between species. Therefore, only general statements can be made from the observed data in this regard. Fish tissue data by “trophic level”/species is presented below in Table 2.

Table 2: WSP Fish Tissue Data 1997-2004

| Fish Species | Mean Concentration* | Median Concentration | Minimum Concentration | Maximum Concentration | Comparison Value | Number of Samples |
|--------------------------|---------------------|----------------------|-----------------------|-----------------------|------------------|-------------------|
| Sunfish | 33.78 | 14.02 | 0.98 | 308.82 | 5.8 | 27 |
| Bass | 26.90 | 8.38 | 1.37 | 127.26 | 5.8 | 11 |
| Catfish | 122.95 | 85.09 | 2.13 | 435.72 | 5.8 | 18 |
| Sucker | 158.60 | 35.16 | 2.49 | 1153.18 | 5.8 | 39 |
| Trout | 83.66 | 23.28 | 8.3 | 219.4 | 5.8 | 3 |
| Other** | 66.82 | 70.19 | 2.3 | 124.61 | 5.8 | 4 |
| All fish combined | 99.26 | 19.34 | 0.98 | 1153.18 | 5.8 | 102 |

*All Concentrations reported in µg/L or parts per billion (ppb)

**Other includes 2 E. Red and 2 Basefish

Source: TRI 1996-2000, ESC 2000-2005

Generally, edible portions of fish tissue are preferred over whole, homogenized fish tissue samples. Edible fillet portions are considered more representative of the actual exposure point concentration unless site-specific information exists to suggest that some individuals in the site population may consume other portions of fish. This is particularly relevant in the case of PCE as it is a lipophilic compound, which tends to bind to fatty tissues of the fish that are not generally consumed. Some fish contain more fat in the edible portions that are generally consumed resulting in higher concentrations and, subsequently, higher exposures to PCE. The WSP fish tissue data is a combination of whole and edible portions of fish tissue. Therefore, risk estimations based on the probable exposure point concentration may overestimate individual risk.

Exposure Assessment

The guiding purpose of this health consultation is to evaluate whether individuals have been, are being, or may be exposed to PCE within WSP. If excessive exposures are identified, actions are taken to reduce or eliminate these exposures to protect public health. A major step in this process is to determine what types of exposure pathways exist, or how people could come into contact with site-related contamination. This process that characterizes the route, duration, intensity, and frequency of contact with a chemical by a receptor is formally known as the exposure assessment. In this assessment, the primary receptors of interest are individuals that may reside in the vicinity of WSP, and the principal exposure routes of interest are fish ingestion, dermal contact with surface water while swimming/wading, and incidental ingestion of water while swimming/wading. All past, current and future exposure pathways will be discussed in the following section. Generally, the major steps of an exposure assessment are:

- (1) conceptual site model presented in Table 3;
- (2) estimation of exposure point concentration; and
- (3) estimation of exposure dose.

Past Exposures (1990 to 1997)

WSP was a popular recreational area for the surrounding communities prior to its closure to the general public on September 10, 1997. At this time, PCE was present in fish tissues and pond water. Individuals were allowed to catch and take fish for consumption. Swimming and/or wading was not allowed at WSP prior to closure. However, some evidence, gathered through community interviews, indicates that teenagers often times trespassed onto the property during summer months after WSP was closed to swim in the ponds. If people were swimming in the ponds after they were closed to the public, it is reasonable to assume that at least some swimming/wading took place when WSP were actually open. Due to the fact that people do not use the ponds for drinking water, exposure to PCE does not occur for a drinking water pathway. The fish consumption pathway is considered a complete exposure pathway since all 5 elements of the pathway are present. Dermal contact through swimming is a potential exposure pathway because the route of exposure (dermal contact) is still unclear. Due to the possibility that some individuals swam/wade in the ponds prior to the date of closure, the dermal absorption pathway will also be evaluated. Exposure dose calculations will be performed for these pathways for the time period prior to the date of WSP closing. The exposure pathways discussed in this health consultation are summarized below in Table 3.

Table 3: Conceptual Site Model

| Source | Transport Mechanism | Point of Exposure | Affected Environmental Medium | Timeframe of Exposure | Potentially Exposed Population | Route of Exposure |
|------------------------------|---------------------|----------------------|--------------------------------------|--|--------------------------------|---|
| Schlage Source Areas 1,2,& 3 | Widfield Aquifer | Willow Springs Ponds | Surface Water, Sediment ^a | Recreational & subsistence fishing 1990-1997 (before pond closure) | Local Residents | Fish Ingestion |
| Schlage Source Areas 1,2,& 3 | Widfield Aquifer | Willow Springs Ponds | Surface Water, Sediment ^a | Trespasser recreational fishing 1997-2004 (during pond closure) | Local Residents | Fish Ingestion (Potential) |
| Schlage Source Areas 1,2,& 3 | Widfield Aquifer | Willow Springs Ponds | Surface Water, Sediment ^a | Swimming/wading 1990-1997 (before pond closure) | Local Residents | Dermal contact and incidental water ingestion (Potential) |
| Schlage Source Areas 1,2,& 3 | Widfield Aquifer | Willow Springs Ponds | Surface Water, Sediment ^a | Trespasser Swimming/wading 1997-2005 (during pond closure) | Local Residents | Dermal Contact and incidental water ingestion |

^a This pathway is not evaluated in this assessment because no data is available for PCE concentrations in sediments.

NOTE: Inhalation of PCE vapors during swimming/wading is a complete exposure pathway, but is considered insignificant exposure pathway for this assessment and therefore is not quantitatively evaluated

After WSP closed, the only way an individual could be affected by PCE within the ponds is by trespassing onto the property. As previously mentioned, there is evidence to suggest that teenagers have trespassed onto the property to go swimming in the ponds after they were closed. Representatives of the EES at CDPHE conducted 31 community interviews in early 2004 to document community health concerns and gather site-related information from community members and other stakeholders. These concerns and information were published in the Health Issues Communication Plan (CDPHE 2004). This document states that “*we found several teenage children swam in the ponds approximately once a week, every week, throughout the past two summers (2002-2003).*” No one that was interviewed stated that they had consumed fish from WSP after the ponds closed. However, it is possible that some individuals have ignored the posted warnings, trespassed onto the property, and caught and consumed fish from WSP. Therefore, trespassing swimming is a complete exposure pathway and trespassing fish consumption is a potential pathway since one element is uncertain. Both exposure pathways will be analyzed for health risks in this assessment.

Inhalation of PCE and dermal exposures to PCE in sediment are possible exposure pathways that were also identified in this exposure assessment. However, no data exists for either sediment or outdoor air. Mechanical aeration occurring at the ponds would likely increase the PCE in air surrounding WSP while decreasing the PCE concentration of the sediment (aeration cycles water from bottom to top). However, as PCE from the ponds enters the atmosphere it is diluted to a large degree. The low PCE concentration in WSP water is not likely to have an appreciable effect on outdoor air quality and this pathway was excluded from further analysis. As mentioned above, aeration cycles water from bottom to top. Normally, at high concentrations, PCE would sink to the bottom of the pond because it is denser than water. When PCE is near the bottom of the water, it will generally adhere to particles in the sediment and the PCE concentration could increase over time. Aeration modifies this scenario by distributing PCE evenly throughout the body of water. Therefore, the PCE concentration in sediment is not likely to build up to a large degree. The potential sediment pathway was also excluded from further analysis due to a lack of data and the low probability that PCE concentration in sediment will be significant.

Current Exposures (1997-2006)

WSP remained closed to the public at the time this health consultation was performed. Current exposure pathways will be addressed in the same manner as the trespassing exposures mentioned above. That is, exposures that have occurred after WSP was closed to the public are assumed to be representative of the exposures that could be occurring at the present time.

Future Exposures (After re-opening of WSP)

Willow Springs Ponds was once a popular recreational area amongst the local population. As such, it is an important asset to El Paso County and the surrounding community. Recently, El Paso County Commissioners have been discussing reopening WSP. At this time, the reopening date of WSP to fishing is unknown. Low levels of PCE were still

present in WSP water at the time this consultation was conducted. The last available fish tissue samples that were collected in October 2004 also contained PCE. Future exposure conditions will be addressed in greater detail later in this document.

Three major exposure scenarios were identified in this assessment. These scenarios are summarized in the following discussion and presented in greater detail in Appendices [C](#), [D](#), and [E](#).

Exposure Scenario 1 accounts for all complete and potential exposure pathways that occurred before the ponds were closed to the general public. Adult and Child exposure dose calculations will be performed for the time period 1990 (first detection of PCE in any municipal water system) through the date of closure on September 10, 1997. Fish consumption calculations will be based on the fish tissue data that was available before the ponds closed in 1997. Swimming/Wading exposure doses will be calculated with the available water data prior to the date of closure. Scenario 1 swimming/wading was identified as a potential exposure pathway and fish consumption is considered a complete exposure pathway.

Scenario 2 exposures include actual and potential *trespassing* exposures, which occurred after WSP were closed to the public in 1997. Child exposure dose calculations for trespassers will be made in a similar manner as in Scenario 1 with adjustments made for the trespassing scenario. For Scenario 2 exposures, it is assumed that the same exposure pathways exist for trespassers as in Scenario 1, but at a lower frequency or rate. However, in this case trespassing swimming was identified as the complete exposure pathway from community interviews. No data is available to support a trespassing fisher exposure pathway. But if people are trespassing to swim, it is reasonable to assume that some individuals also trespass onto the property to fish. This is considered a potential exposure pathway because it is unclear if trespassing fishing has actually occurred. The timeframe of exposure will be set from 1997-2005 (9 year ED). Exposure Frequency is equal to 100 days per year for both fishing and swimming.

Exposure Scenario 3 addresses the possibility that individuals were swimming/fishing prior to the ponds closure and continued those activities after the closure (at a lower rate). For this scenario, exposure doses from Scenario 1 and Scenario 2 can be added to estimate the dose that individuals could have received under these conditions. All assumptions made in the aforementioned exposures scenarios were adopted for Scenario 3 exposure conditions. It should be noted that Scenario 3 exposure conditions likely overestimate the risk that any one individual could have experienced. Combined, Exposure Scenario 3 suggests that people were fishing and being exposed to PCE in fish tissue from 1990-1997 at a frequency of 240 days per year and also from 1997-2005 at a frequency of 100 days per year. Each of the identified exposure scenarios is summarized in Table 4 below.

Table 4. Summary of Identified Exposure Scenarios

| Scenario | Timeframe of Exposure | Exposure Pathway | Exposure Frequency (days) | Exposure Duration (years) | Exposure Point Concentration | Complete/Potential Pathway |
|----------|------------------------|------------------|---------------------------|---------------------------|------------------------------|----------------------------|
| 1 | 1990-1997 | Fish Consumption | 240 | 8 | 28.32 ppb | Complete |
| 1 | 1990-1997 | Swimming/wading | 100 | 8 | 3.8 ppb | Potential |
| 2 | 1997-2005 | Fish Consumption | 100 | 9 | 220.02 ppb | Potential |
| 2 | 1997-2005 | Swimming/wading | 100 | 9 | 0.68 ppb | Complete |
| 3 | 1990-1997 1997-2005 | Fish Consumption | 240 100 | 8 9 | 28.32 ppb 220.02 ppb | Complete Potential |
| 3 | 1990-1997 1997-2005 | Swimming/wading | 100 100 | 8 9 | 3.8 ppb 0.68 ppb | Potential Complete |

Toxicological Evaluation

The basic objective of a toxicological evaluation is to identify what adverse health effects a chemical causes, and how the appearance of these adverse effects depends on dose. Please see [Appendix H](#) for additional details on the various toxicity values used in this assessment.

Health Assessment: cancer and non-cancer effects

In this section, the calculated exposure doses are compared to health-based non-cancer and cancer guidelines to provide an estimate of the magnitude of the potential health risks from the previously identified scenarios. The exposure dose calculations and results are presented in the Appendices. The strength of the conclusions is also evaluated in terms of the uncertainty in the information used to generate these estimates. Both cancer and non-cancer health effects are evaluated in this assessment.

Risk estimates for exposure scenario 1

The exposure doses calculations for Scenario 1 exposures indicate that it is highly unlikely that any non-carcinogenic harmful health effects could have resulted from consuming fish or swimming for an eight year time period prior to the park's closure. Non-carcinogenic exposures were compared to the EPA's Oral Reference Dose (RfD). The largest estimated exposure dose for non-carcinogenic effects is 0.000079 mg/kg-day ([Table C4](#), Child, non-cancer risks, 99th % ingestion rate). The most conservative health-based value for noncarcinogenic health effects of PCE is 0.01 mg/kg-day (EPA's Oral RfD). This results in a Hazard Quotient (HQ) of 0.0079. HQs greater than 1 indicate a potential for noncarcinogenic adverse health effects. Therefore, non-carcinogenic adverse health effects are not likely during this timeframe of exposure.

To estimate Scenario 1 carcinogenic risk from low-dose, chronic exposures, the exposure dose calculation for carcinogens is multiplied by the cancer slope factor [$0.54 \text{ (mg/kg-day)}^{-1}$]. The highest theoretical cancer Risk Level (RL) observed in this scenario was $3.6 * 10^{-6}$ (Table C4, Child, non-cancer risks, 99th % ingestion rate). This means that no more than 3.6 excess cases of cancer out of one million would be expected from this exposure. To evaluate carcinogenic RL from both swimming and fishing, the RL from each exposure pathway can be summed. In this case, the largest estimated RL would be $4.9 * 10^{-6}$ ($3.6 * 10^{-6} + 1.3 * 10^{-6}$).

It is important to note that the highest theoretical cancer risk estimates presented in Tables C3 and C4 represent conservative exposure assumptions of the subsistence fishing population. In addition, the exposure doses were calculated with 8-year exposure duration, which is likely to overestimate the actual duration of exposure to fish tissue at elevated contamination levels. Therefore, it is concluded that Scenario 1 exposures to fish tissue from WSP prior to closure represent no apparent public health hazard.

Risk Estimates for Exposure Scenario 2

Scenario 2 exposure dose estimations also indicate a very low risk of non-carcinogenic adverse health effects. The highest non-carcinogenic exposure dose calculated for Scenario 2 exposures is $0.00018 \text{ mg/kg-day}$ (Table D4, Child non-cancer, 45.63 g/day), which results in a Hazard Quotient of 0.018. The most conservative health-based value for PCE is 0.01 mg/kg-day (EPA's Oral RfD). Therefore, non-carcinogenic adverse health effects are not likely during this timeframe of exposure.

The highest estimated theoretical cancer RL for Scenario 2 fish consumption exposures is $8.1 * 10^{-6}$ (Table D4, Child non-cancer, 45.63 g/day). The highest estimated theoretical carcinogenic risk from Scenario 2 swimming exposures is $2.4 * 10^{-7}$ (Table D5 Child DAD). When both exposure pathways are combined, the total RL becomes $8.3 * 10^{-6}$.

Again, it is important to note that the highest theoretical cancer risk estimates presented here represent conservative exposure assumptions with high ingestion rates that likely overestimate the actual cancer risk. In addition, the exposure doses were calculated with 9-year exposure duration and a 100-day per year frequency. That is, the exposure assumptions made for Scenario 2 exposures account for the potential exposure events occurring 100 days per year for 9 years. This is likely to overestimate the actual duration and frequency of exposure to fish tissue and surface water at elevated contamination levels. Therefore, it is concluded that Scenario 2 exposures to PCE from WSP after the ponds closure represent no apparent public health hazard.

Risk Estimates for Exposure Scenario 3

Non-carcinogenic exposure dose estimations for Scenario 3 exposures also indicate a very low risk of non-carcinogenic adverse health effects. The highest non-carcinogenic exposure dose calculated for Scenario 3 exposures is $0.00024 \text{ mg/kg-day}$ (Table E2,

Child, non-cancer, IR = 45.63 g/day), which results in a Hazard Quotient of 0.024. Therefore, non-carcinogenic adverse health effects are not likely during this timeframe of exposure.

The highest estimated theoretical cancer RL for Scenario 3 fish consumption exposures is $1.1 * 10^{-5}$ (Table E2, Child, Cancer, IR = 45.63 g/day). The highest estimated theoretical carcinogenic risk from Scenario 3 swimming exposures is $1.55 * 10^{-6}$ (Table E3, Child DAD). When both Scenario 3 exposure pathways are combined, the total RL becomes $1.3 * 10^{-5}$.

Scenario 1 and Scenario 2 exposure dose estimations individually incorporate conservative exposure assumptions. When the exposure assumptions from these two scenarios are combined, the conservative assumptions made in the individual dose calculations doubles. Therefore, Scenario 3 exposures likely overestimate the potential risk to any one individual. Even with the conservative assumption that an individual was fishing and swimming in the ponds for the past 17 years, the cancer and non-cancer risks are still not substantial and represent no apparent public health hazard.

Future Exposures

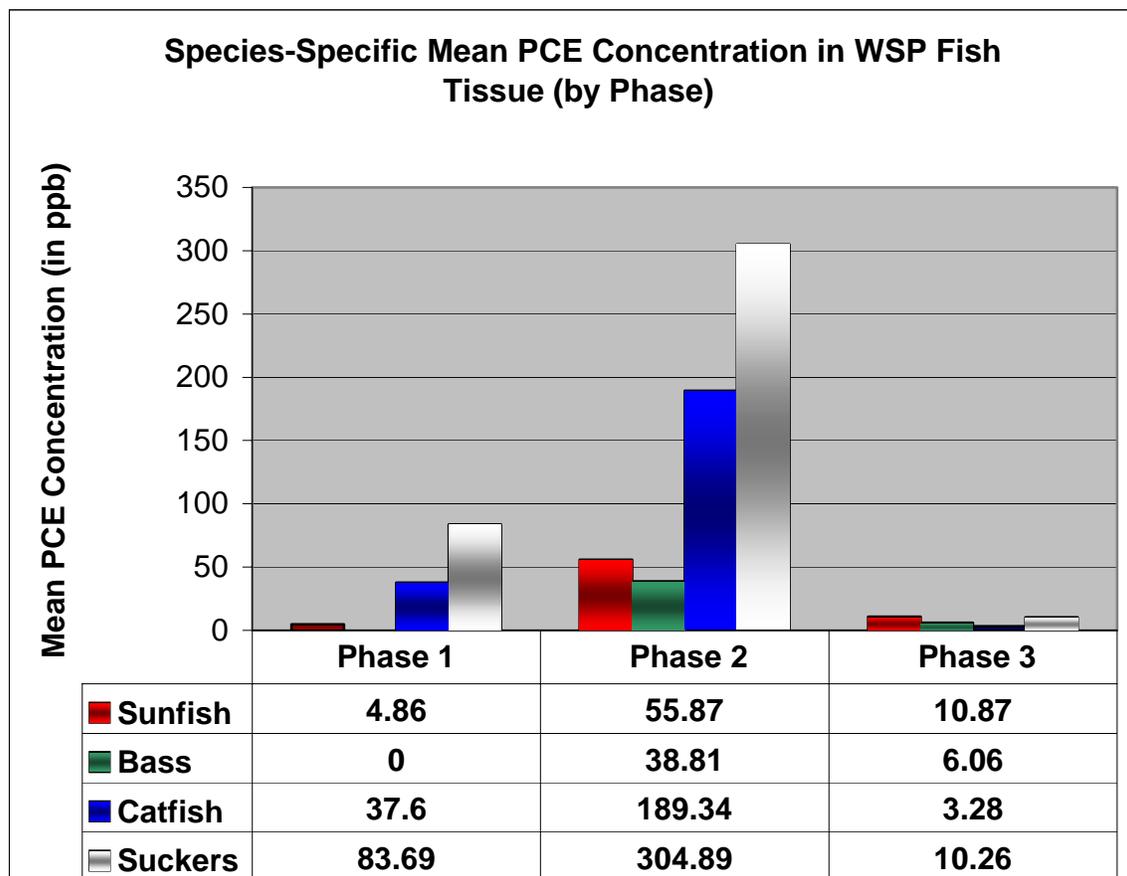
All past, current, and future exposures were evaluated for potential public health hazards in this health consultation. Fish tissue data was divided into 3 phases to evaluate future exposure conditions in this evaluation. Phase 1 occurred before the initial installation of a mechanical aerator in WSP (prior to Feb. 1999). Phase 2 occurred after the installation of the first aerator, but prior to the installation of the 2nd aerator. Phase 3, or the current phase, covers the time period after both aerators were installed and functioning in WSP. Graph 1 below is a depiction of the species-specific average fish tissue concentration by phase. The average PCE concentration in fish tissue appears to be decreasing. Statistically, the difference in PCE concentration between Phases 1 and 2 when compared to Phase 3 is significant.

The last available fish tissue data was collected in 2004 and two sampling events took place during that year. Graph 2 shows the average PCE concentration in fish tissue by species and sampling date. Indeed, it appears that the PCE concentration in fish tissue is decreasing. However, it is unclear if the level of PCE in fish tissue would be a health hazard if the ponds were reopened at this time, based on the last available data. In this section, the data collected in 2004 will be used to predict what potential health hazards may exist if WSP were to be reopened to the general public at the current time. Twenty-six fish tissue samples were available from 2 separate sampling events in 2004. This data was compiled and analyzed. Summary statistics for the 2004 fish tissue data are listed in Table 5.

Table 5. Summary Statistics of 2004 WSP Fish Tissue Data

| <i>2004 Fish Tissue Data</i> | |
|------------------------------|--------|
| Mean PCE Concentration* | 9.00 |
| Median PCE Concentration | 5.169 |
| Minimum PCE Concentration | 0.977 |
| Maximum PCE Concentration | 35.157 |
| Sample Count | 26 |
| 95% Upper Confidence Limit | 12.40 |

* All PCE concentrations denoted in $\mu\text{g/L}$ or ppb

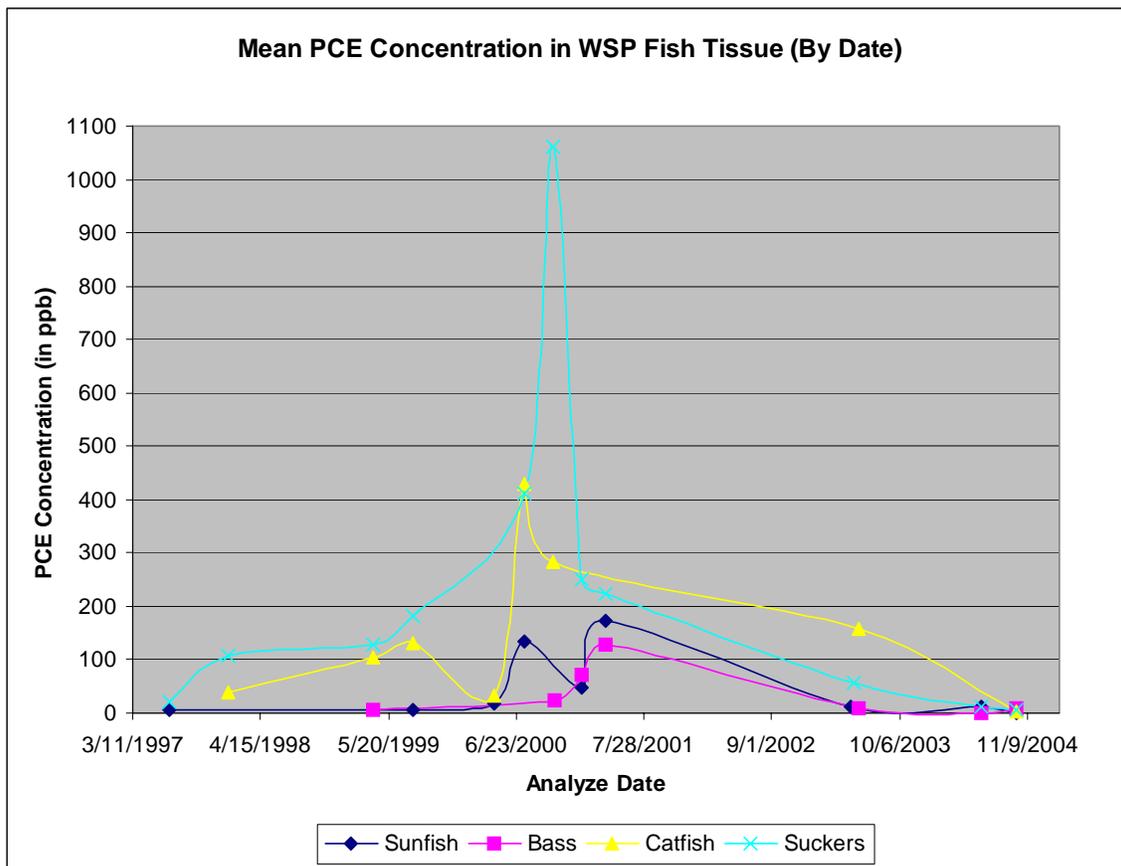
Graph 1: Mean PCE Concentration in Fish Tissue by Phase and Species

Source: TRI 1996-2000, ESC 2000-2005

The PCE concentrations in fish tissues collected in 2004 were then compared to Fish Consumption Limit Guidelines (FCLGs) to determine if there is a potential for public health hazards. FCLG tables were prepared for both carcinogenic and non-carcinogenic health effects of PCE and include a range of ingestion rates, which are broken down by meals per month. At each ingestion rate, a range of “safe” or recommended concentrations of PCE in fish tissue are listed. The FCLGs in this health consultation are based on a 70-year exposure duration.

No adverse health effects are expected to occur from fish consumption of PCE concentrations under the recommended values. For carcinogenic effects, a range of concentrations is listed for each ingestion rate category with the corresponding cancer risk level. The FCLG tables prepared for this consultation are presented in [Appendix A](#). The average 2004 PCE concentration in fish tissues is significantly below the recommended guideline for non-carcinogenic effects in all categories of ingestion rate. However, the average value does exceed the recommended guideline for carcinogenic effects in some categories.

Graph 2: Mean PCE Concentration in Fish Tissue by Analysis Date



Source: TRI 1996-2000, ESC 2000-2005

As previously mentioned, Willow Springs Ponds was once a popular recreational area amongst the local population. As such, it is an important asset to El Paso County. Recently, El Paso County Commissioners have been discussing reopening Willow Springs Ponds. At this time, the circumstances regarding the reopening of Willow Springs Ponds to fishing are still unclear. Due to the fact that the PCE concentration in fish tissues from WSP has been decreasing in recent years, it is possible that the concentration has continued to decrease since 2004 through the time this consultation was performed in 2006. The only way to ensure that the PCE concentration in fish tissue has

dropped to a safe level is to collect more recent fish tissue data. This data can then be compared to the FCLG tables to determine if future public health hazards are likely.

Future public health hazards from consuming fish caught in WSP are classified as an indeterminate public health hazard because it is unknown when WSP will be reopened to the public and what the concentration of PCE in fish tissue will be at that time.

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. For example, infants can be exposed to PCE that has been transferred into breast milk. Additionally, PCE can also cross the placenta. Therefore, the developing fetus and infants should be considered a susceptible population for exposure to PCE. It should be noted that fish also contains high quality proteins and other essential nutrients, and are low in saturated fat and contain omega-3 fatty acids which can contribute to children's proper growth and development (EPA 2004).

Child exposure estimates were calculated in this assessment and compared to health-based guidelines. The estimated exposure doses for children do not indicate any increased risk of non-carcinogenic adverse health effects. No other special public health considerations are indicated for children in this consultation.

Conclusions

The conclusions made in this health consultation are based solely on the combined, available water and fish tissue data collected from Willow Springs Ponds (WSP). Three major exposure scenarios were identified in this assessment of potential health effects from exposure to PCE within WSP. Past, current and future public health hazards were evaluated in this consultation. Scenarios 1-3 describe past and current exposures. The future public health hazard evaluation compares the last available data to FCLG tables located in [Appendix A](#).

CDPHE strives to achieve a target theoretical cancer RL of $1 * 10^{-6}$ or no more than 1 excess theoretical cancer case out of a million people for all exposures. In general, the USEPA considers a risk level of $1 * 10^{-6}$ to $1 * 10^{-4}$ as the acceptable range of risk. In this

health consultation, the major contributor to cancer risk is the fish consumption pathway. None of the exposure scenarios identified in this document estimate a RL greater than $1.3 * 10^{-5}$. This estimated RL corresponds to the conservative exposure scenario 3, which combines RME exposures from fishing and swimming/wading from 1990 to 2005. The fish ingestion pathway largely drives this risk estimate. Overall, it appears while risks from the ingestion of PCE contaminated fish cannot be excluded, the theoretical excess cancer risks are likely to be low; especially, in light of balancing health benefits of fish consumption with low levels of cancer risk.

ATSDR classifies sites as to their public health hazard category. Under ATSDR's classification system, past and current PCE exposures as defined in this consultation (1990 to 2005) are classified as "no apparent public health hazard" and future PCE exposures are an "indeterminate public health hazard". However, there are uncertainties associated with the conclusions made in this health consultation because of inadequate data, particularly, fish tissue PCE levels, usage patterns, and sediment data. These uncertainties have been evaluated in regards to the conservative assumptions made for the exposure dose calculations performed for this assessment and are considered negligible. Additional information regarding ATSDR's public health hazard categories is available in [Appendix G](#) of this document.

Recommendations

Based on these conclusions, the Colorado Department of Public Health and Environment's Environmental Epidemiology Section (EES) suggests the following recommendations:

- At this time, Willow Springs Ponds should remain closed to fishing until the current PCE concentration in fish tissues can be determined.
- Additional fish tissue samples should be collected to determine the current PCE concentration.
- Sediment data should be collected to evaluate the potential health risks of this recreational pathway.
- If Willow Springs Ponds are reopened to the public, the appropriate measures should be taken to reduce the potential of exposures to contaminant levels of concern. Such measures could include:
 - Following a standardized sampling procedure to accurately determine the extent of contamination in fish tissue,
 - Limiting the amount of fish taken from the ponds,
 - Limiting the species and size of fish that can be taken from the ponds, and/or
 - Health education on how to get the positive health benefits from eating fish by minimizing exposure to PCE. For example, explain the

preferential accumulation of PCE in fatty tissue and appropriate methods of fish preparation with special focus on site-specific subsistence fishers.

- El Paso County Officials and the CDPHE should work in conjunction in regards to the reopening of Willow Springs Ponds to assure that fish tissue levels are safe for the public including the site-specific subsistence fisher population.

Public Health Action Plan

The public health action plan describes the actions designed to mitigate or prevent adverse human health effects that might result from exposure to hazardous substances associated with site contamination. The Environmental Epidemiology Section at CDPHE commits to do the following public health actions related to fish consumption from Willow Springs Ponds:

- Review any additional fish tissue data from Willow Springs Ponds at the request of El Paso County officials, other stakeholders, or Security-Widefield residents.
- Evaluate environmental sampling data for other municipal water supplies affected by the PCE groundwater plume and publish the evaluations in future health consultations.
- Evaluate groundwater sampling data for private residential wells affected by the PCE groundwater plume and publish the evaluation in a future health consultation.
- Evaluate the available indoor air data and publish the outcome in a future health consultation.
- Conduct community outreach activities to inform the public of the potential health risks from exposure to PCE within the Widefield Aquifer as well as methods that can be employed to reduce exposures to PCE.

Other documents currently available on Schlage Lock:

- Assessment of Drinking Water Quality, Widefield Water and Sanitation District
- Assessment of Drinking Water Quality, Fountain Municipal Water District
- Assessment of Drinking Water Quality, Security Municipal Water District

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Appendices

Appendix A: Additional Tables and Figures
Table A1: Willow Springs Pond 2 Water Data Summary 1996-2005

| Year | Average PCE Concentration | Median | Maximum | Minimum | Colorado Surface Water Standard for WSP | Sample Count |
|----------------|---------------------------|---------|---------|---------|---|--------------|
| Phase 1 | | | | | | |
| 1996 | 2 | 2 | 2.5 | 1.5 | 0.8 | 2 |
| 1997 | 1.8 | 1.7 | 2.3 | 1.4 | 0.8 | 5 |
| Phase 2 | | | | | | |
| 1998 | No Data | No Data | No Data | No Data | 0.8 | 0 |
| 1999 | 0.77 | 0.7 | 2.7 | 0.25 | 0.8 | 39 |
| 2000 | No Data | No Data | No Data | No Data | 0.8 | 0 |
| 2001 | 1.46 | 0.95 | 6.4 | 0.25 | 0.8 | 33 |
| 2002 | 0.70 | 0.68 | 1 | 0.34 | 0.8 | 12 |
| Phase 3 | | | | | | |
| 2003 | 0.56 | 0.52 | 1 | 0.22 | 0.8 | 16 |
| 2004 | 0.26 | 0.26 | 0.41 | 0.14 | 0.8 | 12 |
| 2005 | 0.34 | 0.31 | 0.92 | 0.15 | 0.69 | 20 |

*All Concentrations reported in µg/L or parts per billion (ppb)

Source: TRI 1996-2000, ESC 2000-2005

Table A2: Willow Springs Ponds Fish Tissue Data

| Sample Number | Species | Phase | Analysis Date | Fish PCE Conc. (ppb) |
|---------------|------------------------|-------|---------------|----------------------|
| Z6342 | Green Sunfish-4 | 1 | 7/3/1997 | 5.58 |
| Z6343 | Green Sunfish-5 | 1 | 7/3/1997 | 4.14 |
| Z6339 | Western White Sucker-1 | 1 | 7/3/1997 | 28.32 |
| Z6340 | Western White Sucker-2 | 1 | 7/3/1997 | 13.02 |
| Z6341 | Western White Sucker-3 | 1 | 7/3/1997 | 18.11 |
| Y0088-E | Channel Catfish | 1 | 1/5/1998 | 4.7 |
| Y0088-W | Channel Catfish | 1 | 1/6/1998 | 70.5 |
| Y0117-E | Rainbow Trout | 1 | 1/6/1998 | 8.3 |
| Y0117-W | Rainbow Trout | 1 | 1/6/1998 | 219.4 |
| Y0090-E-1 | Western White Sucker | 1 | 1/6/1998 | 10.3 |
| Y0091-E-1 | Western White Sucker | 1 | 1/6/1998 | 16.3 |
| Y0092-E | Western White Sucker | 1 | 1/6/1998 | 6.5 |
| Y0093-E | Western White Sucker | 1 | 1/6/1998 | 7.4 |
| Y0090-W | Western White Sucker | 1 | 1/6/1998 | 51.3 |
| Y0091-W | Western White Sucker | 1 | 1/6/1998 | 82.7 |
| Y0092-W | Western White Sucker | 1 | 1/6/1998 | 223.8 |
| Y0093-W | Western White Sucker | 1 | 1/6/1998 | 462.8 |
| Y7026 | Channel Catfish | 2 | 3/31/1999 | 103.63 |
| Y7025 | Bass | 2 | 3/31/1999 | 5.74 |
| Y7023 | Trout | 2 | 3/31/1999 | 23.28 |
| Y7024 | Western White Sucker | 2 | 3/31/1999 | 128.3 |
| X0584 | Basefish A | 2 | 8/3/1999 | 19.44 |
| X0762 | Basefish B | 2 | 8/3/1999 | 2.3 |
| X5461-1 | Hybrid Blue Gill | 2 | 8/3/1999 | 6.48 |
| X0582 | Channel Catfish-A | 2 | 8/3/1999 | 128.85 |
| X0758 | Channel Catfish-B | 2 | 8/3/1999 | 138.69 |
| X0759 | Channel Catfish-C | 2 | 8/3/1999 | 125.69 |
| X0583 | Western White Sucker-A | 2 | 8/3/1999 | 293.18 |
| X0760 | Western White Sucker-B | 2 | 8/3/1999 | 108.99 |
| X0761 | Western White Sucker-C | 2 | 8/3/1999 | 146.34 |
| W0342-VOC | Crappy #1 | 2 | 4/14/2000 | 11.2 |
| W0343-VOC | Crappy #2 | 2 | 4/14/2000 | 22.66 |
| X5462-1 | Channel Catfish-3 | 2 | 4/14/2000 | 51.03 |
| X5463-1 | Channel Catfish-4 | 2 | 4/14/2000 | 34.22 |
| X5464-1 | Channel Catfish-5 | 2 | 4/14/2000 | 11.14 |
| X5460-2 | Blue Gill-4 | 2 | 7/20/2000 | 308.82 |
| X6916 | Green Sunfish-3 | 2 | 7/20/2000 | 47.31 |
| X6914 | Channel Catfish-1 | 2 | 7/20/2000 | 435.72 |
| X6920 | Channel Catfish-7 | 2 | 7/20/2000 | 424.94 |
| X6915 | Western White Sucker-2 | 2 | 7/20/2000 | 226.71 |
| X6919 | Western White Sucker-6 | 2 | 7/20/2000 | 593.42 |
| X9025 | Channel Catfish-1 | 2 | 10/16/2000 | 99.69 |

| | | | | |
|-----------|------------------------|---|------------|---------|
| X9026-2 | Channel Catfish-2 | 2 | 10/16/2000 | 374.95 |
| X9028 | Bass #2 | 2 | 10/16/2000 | 25.57 |
| X9029-2 | Western White Sucker-1 | 2 | 10/17/2000 | 1153.18 |
| X9030-1 | Western White Sucker-2 | 2 | 10/17/2000 | 969.4 |
| X9027-1 | Bass #1 | 2 | 10/20/2000 | 25.05 |
| T1892-V | E-Red (Fish) | 2 | 1/12/2001 | 120.94 |
| T1893-V | E-Red (Fish) | 2 | 1/12/2001 | 124.61 |
| W0344-VOC | Green Sunfish | 2 | 1/12/2001 | 46.73 |
| W0341-VOC | Bass | 2 | 1/12/2001 | 72.57 |
| W0345-VOC | Western White Sucker | 2 | 1/12/2001 | 383.23 |
| W0426-VOC | Western White Sucker | 2 | 1/12/2001 | 116.88 |
| W1757-V | Sunfish-1 | 2 | 3/28/2001 | 173.6 |
| W1758-V | Sunfish-2 | 2 | 3/28/2001 | 80.75 |
| W1759-V | Sunfish-3 | 2 | 3/28/2001 | 17.81 |
| W1755-V | Western White Sucker-1 | 2 | 3/28/2001 | 364.95 |
| W1756-V | Western White Sucker-2 | 2 | 3/28/2001 | 432.83 |
| W1760-V | Bass #1 | 2 | 3/29/2001 | 127.26 |
| X0585 | Blue Gill-3 | 2 | 5/6/2003 | 5.56 |
| T1894-V | Blue Gill-1 | 2 | 5/7/2003 | 8.61 |
| X6917 | Blue Gill-2 | 2 | 5/7/2003 | 9.55 |
| T1895-V | European Rudd-1 | 2 | 5/7/2003 | 14.5 |
| T1896-V | European Rudd-2 | 2 | 5/7/2003 | 15.45 |
| T1897-V | Western White Sucker-1 | 2 | 5/7/2003 | 44.84 |
| T2684-V | Lg. Mouth Bass-2 | 2 | 5/29/2003 | 8.38 |
| T2681-V | Western White Sucker-1 | 2 | 5/29/2003 | 33.32 |
| T2687-V | European Rudd-1 | 2 | 5/30/2003 | 13.02 |
| T2688-V | European Rudd-2 | 2 | 5/30/2003 | 14.02 |
| T2689-V | European Rudd-3 | 2 | 5/30/2003 | 19.24 |
| T2685-V | Channel Catfish-1 | 2 | 5/30/2003 | 157.95 |
| T2686-V | Channel Catfish-2 | 2 | 5/30/2003 | 41.58 |
| T2683-V | Lg. Mouth Bass-1 | 2 | 5/30/2003 | 7.11 |
| T2682-V-1 | Western White Sucker-2 | 2 | 5/30/2003 | 88.96 |
| T2690-V | Western White Sucker-3 | 2 | 5/30/2003 | 54.23 |
| T2691-V | White Amur-1 | 2 | 5/30/2003 | 12.96 |
| S2852-V | Lg. Mouth Bass | 3 | 6/16/2004 | 1.437 |
| S2838-V | Western White Sucker | 3 | 6/16/2004 | 5.279 |
| S2839-V | Western White Sucker | 3 | 6/16/2004 | 35.157 |
| S2845-V | European Rudd | 3 | 6/17/2004 | 16.629 |
| S2846-V | European Rudd | 3 | 6/17/2004 | 6.918 |
| S2847-V | European Rudd | 3 | 6/17/2004 | 16.213 |
| S2848-V | European Rudd | 3 | 6/17/2004 | 13.109 |
| S2849-V | European Rudd | 3 | 6/17/2004 | 10.72 |
| S2850-V | European Rudd | 3 | 6/17/2004 | 3.656 |
| S2851-V | European Rudd | 3 | 6/17/2004 | 18.77 |

| | | | | |
|---------|----------------------|---|-----------|--------|
| S2840-V | Western White Sucker | 3 | 6/17/2004 | 8.52 |
| S2841-V | Western White Sucker | 3 | 6/17/2004 | 12.071 |
| S2842-V | Western White Sucker | 3 | 6/17/2004 | 25.539 |
| S2843-V | Western White Sucker | 3 | 6/17/2004 | 2.485 |
| S2844-V | Western White Sucker | 3 | 6/17/2004 | 2.693 |
| S5236-V | Green Sunfish | 3 | 10/4/2004 | 0.977 |
| S5233-V | Lg. Mouth Bass | 3 | 10/4/2004 | 1.368 |
| S5234-V | Lg. Mouth Bass | 3 | 10/4/2004 | 3.999 |
| S5235-V | Lg. Mouth Bass | 3 | 10/4/2004 | 17.44 |
| S5226-V | Western White Sucker | 3 | 10/4/2004 | 3.004 |
| S5230-V | Channel Catfish | 3 | 10/5/2004 | 3.331 |
| S5227-V | Western White Sucker | 3 | 10/5/2004 | 5.059 |
| S5228-V | Western White Sucker | 3 | 10/5/2004 | 9.428 |
| S5229-V | Western White Sucker | 3 | 10/5/2004 | 3.608 |
| S5231-V | Channel Catfish | 3 | 10/7/2004 | 2.13 |
| S5232-V | Channel Catfish | 3 | 10/7/2004 | 4.373 |

Whole Fish Tissue Samples**Fish Fillet Tissue Samples**

Source: TRI 1996-2000, ESC 2000-2005

Table A3: Health-Based Fish Consumption Limit Guidelines (FCLG) for PCE based on Chronic Noncarcinogenic Health Effects**General Population Ages 18 and over**

| Frequency of Fish Meals ^a | Daily Fish Intake Rates for General Population (Age 18 and Older) | Fish Tissue Levels (PPB) |
|---|--|---------------------------------|
| 24 meals/month (6 meals/week) | 179.0 g/day | 0 – 3,910 |
| 20 meals/month (5 meals/week) | 149.1 g/day ^b | >3,910 – 4,700 |
| 16 meals/month (4 meal/week) | 119.3 g/day | >4,700 – 5,870 |
| 12 meals/month (3 meals/week) | 89.5 g/day ^c | >5,870 – 7,820 |
| 8 meals/month (2 meals/week) | 59.7 g/day | >7,820 – 11,730 |
| 4 meals/month (1 meal/week) | 29.8 g/day | >11,730 – 23,500 |
| 3 meals/month | 22.4 g/day | >23,500 – 31,250 |
| 2 meals/month | 14.9 g/day | >31,250 – 46,980 |
| 1 meal/month | 7.5 g/day ^d | >46,980 – 93,330 |
| NO CONSUMPTION RECOMMENDED | NO CONSUMPTION RECOMMENDED | >93,330 |

^a The assumed meal size for a person weighing 70 kg is a default value of 227g (8 oz portion of uncooked fish). (EPA, 2000; *Guidance for risk assessment and fish consumption limits, Volume 2*).

^b This value is within the 99th percentile upper bound interval range of 125.27 – 156.84 g/day for the general population group (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4 - Freshwater/Estuarine Fish, p. 5-6*).

^c This value is similar to EPA's value of 87.12g/day that represents the 90th percentile upper limit on the mean for "Consumers Only" (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4-Freshwater/Estuarine Fish, p. 5-43*).

^d This value represents EPA's mean value for the general population group (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4 - Freshwater/Estuarine Fish, p. 5-6*).

Notes:

1. The FCLG is a concentration of tetrachlorethylene (PCE) in fish tissue that is likely to be without appreciable risk of noncarcinogenic human health effects. It is assumed that no other contaminated fish is being eaten. The total fish consumption should not exceed the amount of fish given in the above table. The FCLG for the general population (18 and over) is based on:
 - EPA recommended body weight values of 70 kg (for general population).
 - Reference dose (RfD) of 0.01 mg/kg/day.
 - RSC of fish to total exposure = 1. This means that other sources of PCE to the total body burden are not taken into account in deriving FCLGs.
2. Fish Tissue Level (in PPM) = RfD (mg/kg/day) x Body weight (kg) x Relative Source Contribution (RSC) / Fish intake rate (kg/day).
3. To convert from fish tissue levels in ppm-ppb, multiply the concentration in ppm by one thousand. E.g. 3.26 ppm * 1000 = 3,260 ppb
4. Fish Intake Rate (g/day) = Monthly frequency of meals (Number of meals per month) x Population-specific Meal size (227 g) / 30.44 days.
5. Monthly limits are based on the total allowable dose over a 1-month period (30 days) based on the RfD. When monthly limit is consumed in a few large meals (bolus dose), in less than a month, the daily dose may exceed the RfD (i.e., EPA's allowable/acceptable daily dose). Therefore bolus doses should be avoided.
6. It is important to adjust meal size to body weight. Meal sizes can be adjusted by using a general guide of 0.114 oz per kg body weight. For example, a meal size for a person weighing 88 kg is a ten-ounce serving (uncooked fish weight).

Table A4: Health-Based Fish Consumption Limit Guidelines (FCLG) for PCE based on Chronic Noncarcinogenic Health Effects

Children Ages 6 and under

| Frequency of Fish Meals^a | Daily Fish Intake Rates | Fish Tissue Levels (PPB) for Children (under the age of 6) |
|--|--------------------------------|---|
| 24 meals/month (6 meals/week) | 67.0 g/day ^b | 0 – 2,160 |
| 20 meals/month (5 meals/week) | 55.8 g/day ^c | >2,160 – 2,600 |
| 16 meals/month (4 meals/week) | 44.7 g/day | >2,600 – 3,240 |
| 12 meals/month (3 meals /week) | 33.5 g/day | >3,240 – 4,330 |
| 8 meals/month (2 meals/week) | 22.3 g/day | >4,330 – 6,500 |
| 4 meals/month (1 meal/week) | 11.2 g/day ^d | >6,500 – 12,950 |
| 3 meals/month | 8.4 g/day ^e | >12,950 – 17,260 |
| 2 meals/month | 5.6 g/day | >17,260 – 25,890 |
| 1 meal/month | 2.8 g/day ^f | >25,890 – 51,790 |
| NO CONSUMPTION RECOMMENDED | NO CONSUMPTION RECOMMENDED | >51,790 |

^a The assumed meal size is a default value of 85 g (3oz portion of uncooked fish) for children younger than 4 years old (EPA, 2000; *Guidance for risk assessment and fish consumption limits, Volume 2*).

^b This value is within the 90th percentile upper bound interval range of 63.77 – 110.53 g/day on the 99th percentile for children ages 6 to 10 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-7).

^c This value is within the 90th percentile upper bound interval range of 45.57 – 61.53 g/day on the 99th percentile for children ages 3 to 5 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-7).

^d This value is within the 90th percentile upper bound interval range of 10.26 – 14.05 g/day on the 95th percentile for children ages 3 to 5 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-7).

^e This value is within the 90th percentile upper bound interval range of 4.77 – 20.11 g/day on the 95th percentile for children ages 6 to 10 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-7).

^f This value is similar to the 90th percentile upper bound interval value of 2.58 g/day on the mean for children ages 3 to 5 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 5-Freshwater/Estuarine Fish*, p. 5-7).

Notes:

1. FCLG is a concentration of tetrachlorethylene (PCE) in fish that is likely to be without appreciable risk of noncarcinogenic human health effects. The FCLG for children ages 6 and under is based on:
 - EPA recommended body weight value of 14.5 kg (EPA, 2000, Table 2-2).
 - Reference dose (RfD) of 0.01 mg/kg/day.
2. Monthly limits are based on the total allowable dose over a 1-month period (30 days) based on the RfD. When monthly limit is consumed in a few large meals (bolus dose), in less than a month, the daily dose may exceed the RfD (i.e., EPA's allowable/acceptable daily dose). Therefore bolus doses should be avoided.
3. Fish Tissue Level (in ppm) = RfD (mg/kg/day) x Body weight (kg) x Relative Source Contribution (RSC)/ Fish intake rate (kg/day).
4. To convert from fish tissue levels in ppm-ppb, multiply the concentration in ppm by one thousand. E.g. 3.26 ppm * 1000 = 3,260 ppb
5. RSC of fish to total exposure = 1. This means that other sources of PCE to the total body burden are not taken into account in deriving FCLGs
6. Fish Intake Rate (g/day) = Monthly frequency of meals (Number of meals per month) x Meal size (85 g) / 30.44 days.
7. **It is important to adjust meal size to body weight.** Meal sizes can be adjusted by using a general guide of 0.20 oz per kg body weight for children. For example, a meal size for a child weighing 10 kg is a two-ounce serving (uncooked fish weight).
8. **The above FCLGs may also be used as a general guide for children age 6 or older.**

Table A5: Health-Based Fish Consumption Limit Guidelines (FCLG) for PCE based on chronic, low-dose carcinogenic effects**General Population**

| Frequency of Fish Meals^a | Daily Fish Intake Rates for General Population | Fish Tissue Levels At 10⁻⁴ Risk Level (in PPB) | Fish Tissue Levels At 10⁻⁵ Risk Level (in PPB) | Fish Tissue Levels At 10⁻⁶ Risk Level (in PPB) |
|--|---|--|--|--|
| 24 meals/month (6 meals/week) | 179.0 g/day | 0 – 72 | 0 – 7.2 | 0 – 0.72 |
| 20 meals/month (5 meals/week) | 149.1 g/day ^b | >72 – 87 | >7.2 – 8.7 | >0.72 – 0.87 |
| 16 meals/month (4 meal/week) | 119.3 g/day | >87 – 109 | >8.7 – 10.9 | >0.87 – 1.09 |
| 12 meals/month (3 meals/week) | 89.5 g/day ^c | >109 – 145 | >10.9 – 14.5 | >1.09 – 1.45 |
| 8 meals/month (2 meals/week) | 59.7 g/day | >145 – 217 | >14.5 – 21.7 | >1.45 – 2.17 |
| 4 meals/month (1 meal/week) | 29.8 g/day | >217 – 435 | >21.7 – 43.5 | >2.17 – 4.35 |
| 3 meals/month | 22.4 g/day | >435 – 579 | >43.5 – 57.9 | >4.35 – 5.79 |
| 2 meals/month | 14.9 g/day | >579 – 870 | >57.9 – 87.0 | >5.79 – 8.70 |
| 1 meal/month | 7.5 g/day ^d | >870 – 1,728 | >87.0 – 172.8 | >8.70 – 17.28 |
| NO CONSUMPTION RECOMMENDED | NO CONSUMPTION RECOMMENDED | >1,728 | >172.8 | >17.28 |

^a The assumed meal size for a person weighing 70 kg is a default value of 227g (8 oz portion of uncooked fish). (EPA, 2000; *Guidance for risk assessment and fish consumption limits, Volume 2*).

^b This value is within the 99th percentile upper bound interval range of 125.27 – 156.84 g/day for the general population group (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4 - Freshwater/Estuarine Fish, p. 5-6*).

^c This value is similar to EPA's value of 87.12g/day that represents the 90th percentile upper bound interval on the mean for "Consumers Only" (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4-Freshwater/Estuarine Fish, p. 5-43*).

^d This value represents EPA's mean value for the general population group (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4 - Freshwater/Estuarine Fish*, p. 5-6).

Notes:

1. The FCLG is a concentration of tetrachlorethylene (PCE) in fish that is likely to be without appreciable risk of carcinogenic human health effects. The FCLG developed for carcinogenic health effects in the general population is based on:
 - EPA recommended body weight value of 70 kg (EPA, 2000, Table 2-2)
 - California EPA Oral Cancer Slope Factor (CSF) of $0.54 \text{ mg/kg}\cdot\text{day}^{-1}$
 - Lifetime Exposure Duration of 70 yrs.
2. Monthly limits are based on the total allowable dose over a 1-month period (30 days) based on the RfD. When monthly limit is consumed in a few large meals (bolus dose), in less than a month, the daily dose may exceed the recommended carcinogenic risk levels described above. Therefore bolus doses should be avoided.
3. Fish Tissue Level = $[(\text{RL} / \text{CSF}) * \text{BW}] / \text{Mean Fish Consumption Rate averaged over 70 yrs (kg/day)}$ (EPA 2000, Vol. 1 Ch. 5, p. 5-4).
4. To convert from fish tissue levels in ppm-ppb, multiply the concentration in ppm by one thousand. E.g. $3.26 \text{ ppm} * 1000 = 3,260 \text{ ppb}$
5. The low-dose extrapolation procedure for carcinogenic health effects used in this assessment provides an upper 95 percent bound risk estimate. This is considered by some to be a conservative estimate of cancer risk (EPA 2000, Vol. 1 Ch. 5, p. 5-3).
6. Fish Intake Rate (g/day) = Monthly frequency of meals (Number of meals per month) x Meal size (227 g) / 30.44 days.

Figure A1: Approximation of PCE plume (2002 data)

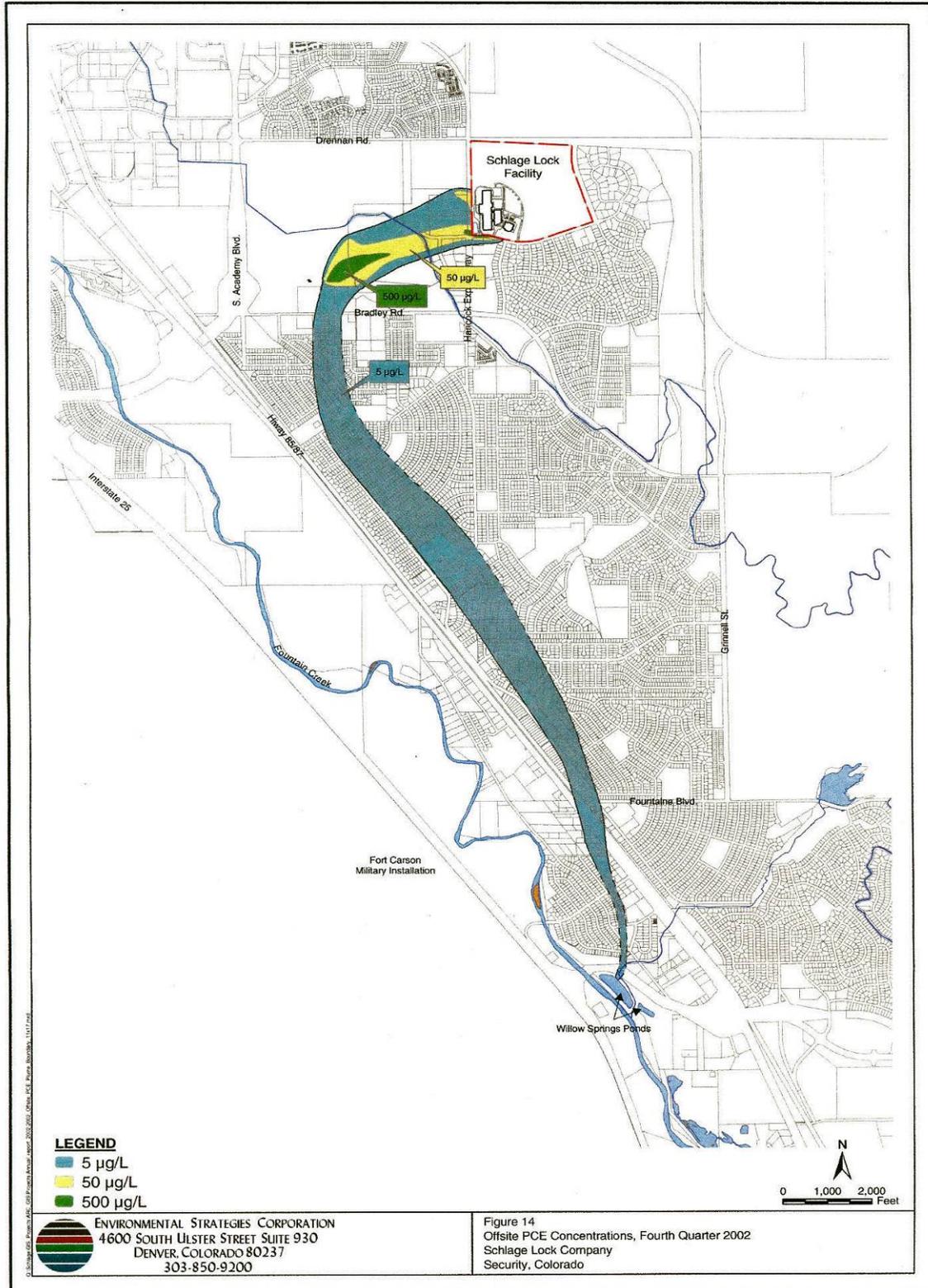


Figure A2: Willow Springs Ponds

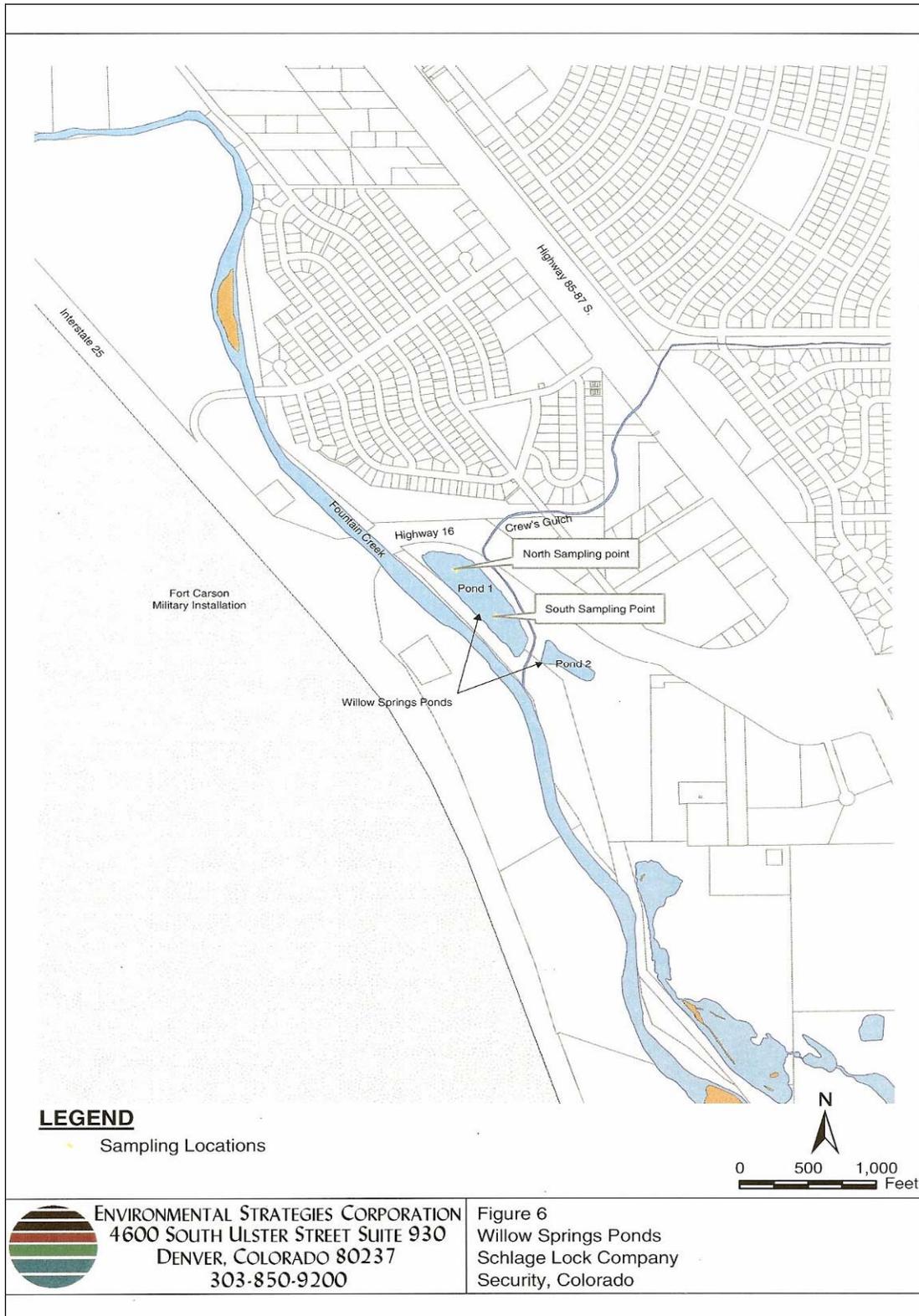
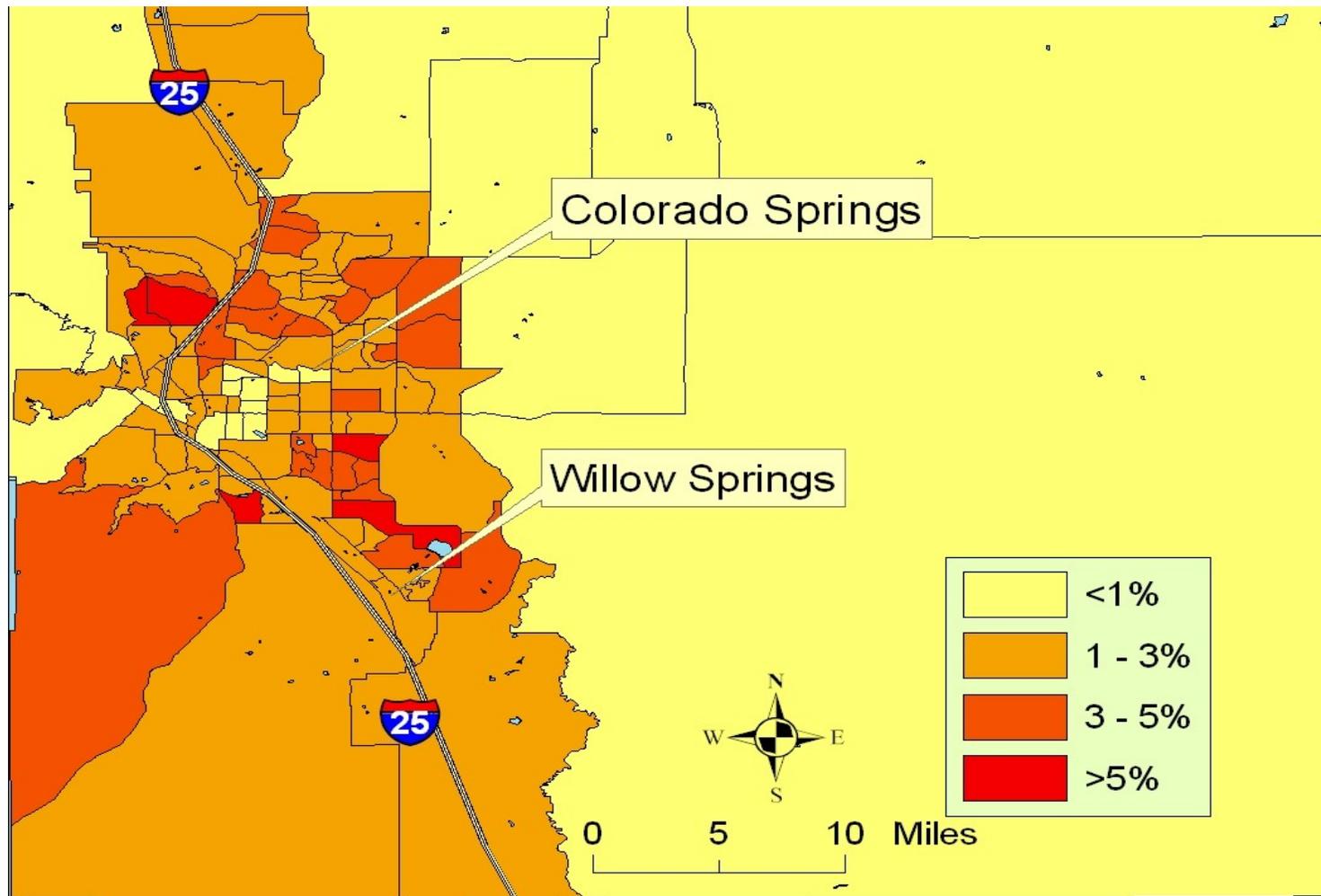


Figure A3. Percent Asian American vs. Total Population by Census Tract in El Paso County (Census 2000)



Appendix B: Supplemental Information on Calculating Exposure Doses

Estimation of Exposure Point Concentration

When people are exposed to a chemical in a medium such as surface water and fish, the point of exposure is usually called the exposure point. The location and size of the exposure point depends in part on human activity patterns and in part on the length of time that is required for a chemical to cause adverse effects.

The concentration term used to assess risk from exposure is the arithmetic mean concentration of a contaminant, averaged over the location where exposure is presumed to occur during a specified time interval. Because the true mean concentration of a chemical within an Exposure Area cannot be calculated with certainty from a limited set of measurements, the USEPA recommends that the upper 95th confidence limit (UCL) of the arithmetic mean concentration be used as the Exposure Point Concentration (EPC) in calculating exposure and risk. If the calculated UCL is higher than the highest measured value, then the maximum value is used as the EPC instead of the UCL.

Exposure Dose Estimation

The amount of a chemical that is ingested, or taken up across the skin is referred to as “dose” or “intake” and is typically calculated using a general equation noted below:

Dose = Chemical concentration x intake rate x exposure duration x exposure frequency / body weight x length of time over which dose is averaged (or averaging time)

Some adjustments to the standard procedure for calculating exposure doses had to be made for the exposure dose estimations in this health consultation. The exposure dose calculation is a mathematical estimate based on variables such as: Body Weight, Ingestion Rate, Exposure Duration, and Averaging Time. The objective of calculating exposure doses is to conservatively estimate the actual dose of contaminant to the population based on the best available information. Generally, there is a wide variation in exposure parameters (e.g., intake rate, body weight, and exposure frequencies) between different members of an exposed population. Therefore, for most dose calculations, standard procedures and default values established by the EPA are used. The intent of the standard procedures and default parameters is to estimate conservative exposures that are still within a range of possible exposures. In addition, for parameters such as the fish ingestion rate difference amongst the general population, attention is focused on different parts of the exposure distribution (e.g., average, Reasonable Maximum, and subsistence population). Generally, attention is given to two different parts of the exposure distribution:

Average or central tendency exposure (CTE) which represents either the arithmetic mean or the median exposure.

Reasonable Maximum Exposure (RME) represents the highest exposure that is reasonably expected to occur. The intent of the RME is to estimate a conservative exposure dose that is still within the range of possible exposures. Conceptually, the RME describes the exposures above the 90th percentile of the population exposure distribution.

Whenever possible, site-specific data are also used. For example, Ingestion rate, or the amount of fish consumed (in kg/day), is dependent upon both the availability of suitable fisheries as well as cultural or lifestyle traits. For instance, individuals living along coastal or large fresh bodies of water consume a large amount of fish. Similarly, the primary source of food for many island communities is fish. For some, fish are deeply ingrained in their culture and lifestyle. Increased rates of fish consumption are likely to continue throughout those individuals life regardless of their physical location. On the other hand, the average fish consumption rate for the average adult is many times less that of a subsistence fishing population. Therefore, it is difficult to accurately estimate doses for the entire population with such a large range in a critical variable.

Assessment of the human health risk from ingestion of contaminated food requires information on the quantities of contaminated foodstuffs consumed and the extent of contamination present in foodstuffs. The most reliable method of assessing the extent of human exposure to contaminants in food is direct measurement of concentrations in foodstuffs. PCE concentrations in fish tissues caught from Willow Springs Ponds were used in the exposure dose calculations. To estimate cancer risk for children, the calculated exposure dose is multiplied by the Oral Cancer Slope Factor. In this case the Oral Slope Factor for PCE that was used in this assessment is the EPA Region 3 Provisional value of 0.54 mg/kg-day⁻¹.

Estimation of exposure dose through food chains requires knowledge of the consumption rate of specific food items in the human diet. EPA's Exposure Factors Handbook (1997) provides intake rates for a variety of foodstuffs. Consumption rates of the population in the vicinity of a hazardous waste site may differ considerably from national average consumption rates. For example, regional consumption rates of beef may vary widely from national averages. Consumption rates of subpopulations within the contaminated area may also vary significantly from the national averages. For example, people such as American Indian or Alaska Natives who subsist on fish from a primary source would likely have an increased consumption rate. When local consumption patterns are available and are different from national averages, they should be used in the calculations to determine exposure doses.

As a conservative estimate, this assessment does not consider contaminant reduction due to cooking. Cooking fish prior to eating can reduce the levels of tetrachloroethylene. You can review scientific literature to identify how cooking may affect the substance under evaluation. For example, studies have shown a 20-70% reduction of some lipophilic substances (e.g., polychlorinated biphenyls [PCBs]) in fish as a result of cooking (Sherer and Price 1993; Wilson et al. 1998).

Appendix C: Scenario 1 Exposure Assessment Details

Exposure Scenario 1: *Fishing and Swimming/Wading prior to the WSP closure on September 10, 1997*

The first fish tissue samples from the ponds were analyzed in July of 1997. This sampling event logged 5 fish tissue samples (2 sunfish and 3 suckers) and is presented below in Table C1. No other fish tissue data was available for the time period prior to the ponds closure in September 1997. Fish tissue PCE concentrations in sunfish were less than that found in suckers. Suckers normally live in the bottom portion of the pond (PCE sinks); they grow to a larger size, consume organisms associated with the sediment, and have a larger percentage of fatty tissue for which PCE can associate with. All of these factors contribute to elevated concentrations of PCE in sucker fish tissue. Of the five samples taken, the maximum concentration of PCE in fish tissue was 28.32 ppb while the minimum concentration was 4.14 ppb. The average concentration for the entire sample set was 13.83 ppb. The species-specific means were 4.86 ppb for sunfish and 19.82 ppb for suckers.

Table C1: Fish Tissue samples collected from WSP prior to closure (Sept. 1997)

| Sample Number | Species | Analysis Date | PCE in Fish Tissue | Comparison Value ¹ |
|---------------|---------|---------------|--------------------|-------------------------------|
| Z6342-TF | Sunfish | 7/3/1997 | 5.58 | 5.8 |
| Z6343-TF | Sunfish | 7/3/1997 | 4.14 | 5.8 |
| Z6339-TF | Sucker | 7/3/1997 | 28.32 | 5.8 |
| Z6340-TF | Sucker | 7/3/1997 | 13.02 | 5.8 |
| Z6341-TF | Sucker | 7/3/1997 | 18.11 | 5.8 |

*All Concentrations reported in µg/L or parts per billion (ppb)

¹ EPA Region 3 Risk-Based Concentration for PCE in fish tissue (EPA 2006)

Source: TRI 1996-2000

All available water data that was collected prior to the ponds closure was also analyzed and is presented in Table C2 below. WSP actually closed on September 10, 1997. The water data from a sampling event that occurred on September 11, 1997 was also included in the data set to evaluate Scenario 1 exposures.

Table C2: WSP Pond 1 water prior to the date of closure

| WSPN-01 | | | | |
|-----------|---------|--------|------|-------|
| Date | Analyte | Result | Unit | Depth |
| 8/21/1996 | PCE | 1.1 | ug/L | 1 |
| 8/21/1996 | PCE | 3.3 | ug/L | 9 |
| 9/13/1996 | PCE | 1.7 | ug/L | 0 |
| 9/13/1996 | PCE | 2.5 | ug/L | 0 |
| 9/11/1997 | PCE | 1.9 | ug/L | 0 |
| 9/11/1997 | PCE | 1.8 | ug/L | 2 |
| 9/11/1997 | PCE | 2 | ug/L | 5 |
| 9/11/1997 | PCE | 3.8 | ug/L | 10 |

Source: TRI 1996-2000

The maximum concentration of PCE detected in WSP water prior to the date of closure was 3.8 ppb. This sample was collected at a depth of 10 feet below the surface of the water. It is unlikely that individuals would be exposed to this concentration at this depth on a regular basis. Thus, the selection of this EPC is a conservative assumption.

Scenario 1 Exposure Point Estimation for Fishing

Scenario 1 exposures include all exposures that occurred before WSP was closed to the public in 1997. Fish consumption prior to 1997 was identified as a completed exposure pathway that includes all 5 pathway elements. Swimming and/or wading is considered a potential pathway since it is unknown if swimming actually occurred at WSP prior to the date of closure. However, this pathway was retained for further analysis because of the possibility that some swimming took place regardless of the fact that it was not permitted.

To estimate fish consumption exposure doses prior to the WSP closure, three different ingestion rates were used to calculate three distinct exposure doses. The ingestion rates are based on the amount of fish consumed by the general adult population, the upper 90th percentile limit of the mean general population (consumers only), and an EPA default rate for subsistence populations. The three doses are intended to account for the dose range over the entire site population without over estimating risk for some individuals.

Exposure point estimations were also based on the highest concentration of PCE in fish tissue detected prior to the closure in September 1997 (28.32 ppb). Only a limited amount of fish tissue data exists before the ponds were closed. It is possible, although unlikely, that the PCE concentration in fish tissue was higher before the first fish samples were collected. To account for this possibility, the highest PCE concentration in fish tissue collected before the ponds closed was used for the exposure dose calculations.

PCE was detected in the first municipal water system served by the Widefield Aquifer in June 1990. The municipal drinking wells are monitored for water quality on a regular basis and they would likely be one of the first receptors to detect PCE in the groundwater (excluding Schlage). Moreover, the municipal wells are located up gradient of WSP and it is highly unlikely that PCE was present in WSP before it was detected in the municipal

wells. WSP was initially sampled for PCE in August 1996. At this time, PCE was already present in the ponds. When PCE first entered WSP is unknown, but it can be assumed that it was not present in 1990. Therefore, an 8-year exposure duration (ED) was used, which is a conservative exposure assumption because it is unlikely that PCE was present in the ponds in 1990.

Scenario 1 Exposure Point Estimation for Swimming

Swimming was identified as a potential Scenario 1 exposure pathway. El Paso County has never allowed swimming in WSP. However, it is possible that some individuals swam in WSP prior to closure based on the fact that teenagers have trespassed onto the property to swim after WSP was closed. Therefore, exposure doses were calculated for this potential pathway. The maximum concentration of PCE found in WSP water prior to the date of closure was 3.8 ppb. This sample was collected at a depth of 10 ft. Therefore, the use of 3.8 ppb as the exposure point concentration is a conservative assumption because dermal exposures occurring at 10 feet below the water surface are not likely to occur for a prolonged period. However, this value was selected as the EPC because of a limited amount of water data from WSP prior to the date of closure. An ED of 8 years was also used for adult swimming/wading exposures.

Scenario 1 Exposure Dose Estimations

In this section, the exposure doses for the complete and potential Scenario 1 exposure pathways identified above will be estimated.

All exposure dose parameters for each scenario were outlined above under the appropriate subsection. The results of adult Scenario 1 exposure dose calculations for fish consumption for both carcinogenic and non-carcinogenic health effects are listed below in Tables C3. Carcinogenic exposure dose calculations are averaged over a 70-year time period, whereas non-carcinogenic exposure dose calculations were averaged over the 8-year exposure duration. This factor is the cause of difference between carcinogenic and non-carcinogenic exposure dose results.

Child exposure dose calculations for Scenario 1 fish consumption are presented below in Table C4. Children have lower body weights and lower fish consumption rates. Therefore, adjustments in the exposure dose calculations must be made for these variables.

Table C3: Potential non-cancer and theoretical cancer risks for adult fish consumption exposures for Exposure Scenario 1 (prior to WSP closure in September 1997)

| Description of Applicable Population | Ingestion Rate (grams/day) | Exposure Dose for Carcinogenic Effects (mg/kg-day) | Estimated Excess Cancer Risk | Exposure Dose for Noncarcinogenic Effects (mg/kg-day) | EPA's Oral Reference Dose (mg/kg-day) | Hazard Quotient (Noncancer Dose / Reference Dose) |
|---|----------------------------|--|--------------------------------------|---|---------------------------------------|---|
| Represents the mean fish consumption of individuals 18 and older living in the United States. | 7.5 ^a | $2.3 * 10^{-7}$ mg/kg-day | $1.2 * 10^{-7}$ 1.2 / 10,000,000 | $2.0 * 10^{-6}$ mg/kg-day | 0.01 mg/kg-day | $2.0 * 10^{-4}$ |
| Represents the upper limit of the 90 % estimate on the mean for "Consumers Only" individuals 18 and over living in the U.S. | 87.12 ^b | $2.6 * 10^{-6}$ mg/kg-day | $1.4 * 10^{-6}$ (1.4 / 1,000,000) | $2.3 * 10^{-5}$ mg/kg-day | 0.01 mg/kg-day | $2.3 * 10^{-3}$ |
| Represents the default value for a subsistence population living in the U.S. | 142.4 ^c | $4.3 * 10^{-6}$ mg/kg-day | $2.3 * 10^{-6}$ (2.3 / 1,000,000) | $3.8 * 10^{-5}$ mg/kg-day | 0.01 mg/kg-day | $3.8 * 10^{-3}$ |

^a This value represents EPA's mean value for the general population group (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4 - Freshwater/Estuarine Fish, p. 5-6*).

^b This value represents the EPA's value of 87.12g/day that represents the 90th percentile upper bound interval on the mean for "Consumers Only" (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4-Freshwater/Estuarine Fish, p. 5-43*).

^c This value is within the 99th percentile upper bound interval range of 125.27 – 156.84 g/day for the general population group (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4 - Freshwater/Estuarine Fish, p. 5-6*).

Table C4: Potential non-cancer and theoretical cancer risk from Child fish consumption exposures for Exposure Scenario 1 (prior to WSP closure in September 1997)

| Description of Applicable Population | Ingestion Rate (grams/day) | Exposure Dose for Carcinogenic Effects (mg/kg-day) | Estimated Excess Cancer Risk | Exposure Dose for Noncarcinogenic Effects (mg/kg-day) | EPA's Oral Reference Dose (mg/kg-day) | Hazard Quotient (Noncancer Dose / Reference Dose) |
|---|----------------------------|--|---------------------------------------|---|---------------------------------------|---|
| Represents mean fish consumption rate of the general U.S. population (Ages 3-5) | 2.8 ^a | $3.1 * 10^{-7}$ mg/kg-day | $1.7 * 10^{-7}$ or $1.7 / 10,000,000$ | $3.6 * 10^{-6}$ mg/kg-day | 0.01 mg/kg-day | $3.6 * 10^{-4}$ |
| Represents the upper confidence limit on the 90% estimate of the mean fish consumption for Consumers Only (Ages 3-5) | 45.63 ^b | $5.0 * 10^{-6}$ mg/kg-day | $2.7 * 10^{-6}$ or $2.7 / 1,000,000$ | $5.9 * 10^{-5}$ mg/kg-day | 0.01 mg/kg-day | $5.9 * 10^{-3}$ |
| Represents the upper confidence limit on the 90% estimate of the 99 th percentile of fish consumption for the general U.S. population (ages 3-5) | 61.53 ^c | $6.7 * 10^{-6}$ mg/kg-day | $3.6 * 10^{-6}$ or $3.6 / 1,000,000$ | $7.9 * 10^{-5}$ mg/kg-day | 0.01 mg/kg-day | $7.9 * 10^{-3}$ |

^a This value is similar to the 90th percentile upper bound interval value of 2.58 g/day on the mean for children ages 3 to 5 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-7).

^b This value represents the 90% UCL on the mean fish consumption rate for children ages 3-5, "Consumers Only" (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-44).

^c This value represents the 90% UCL on the 99th percentile for children ages 3 to 5 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-7).

Swimming and/or wading was identified as a potential exposure pathway prior to WSP closure. Some chemicals have the capability of passing through the protective layers of the skin where they are then absorbed into the bloodstream. Dermal exposures are generally considered less important contributors to health risk than other routes of exposure such as ingestion or inhalation. Many metallic based compounds are not generally taken up by the body through the skin at a high rate, whereas many organic compounds do have this ability.

Exposure dose calculations for swimming/wading exposures that occurred prior to the WSP date of closure were performed using standard EPA default values for swimming. In terms of exposure duration, the same assumption was used for both swimming and fishing (ED = 8 yrs.). The event frequency (EF) was set to 100 days of swimming/wading per year. Scenario 1 estimated exposure dose results for swimming/wading are presented below in Table C5. Only carcinogenic risk was calculated for swimming/wading exposures as it is expected to be the most conservative estimate of adverse health effects for PCE. In addition, the dose received via incidental ingestion while swimming/wading was evaluate and found to be an insignificant contributor to overall risk. Incidental ingestion doses were not evaluated further.

Table C5: Theoretical Carcinogenic risks from swimming/wading in surface water for Scenario 1 exposures (prior to WSP closure in September 1997)

| Dose Parameter | Exposure Dose for Carcinogenic Health Effects | Estimated Cancer Risk |
|-----------------------|--|---------------------------------------|
| Scenario 1 Adult DAD | $1.9 * 10^{-6}$ mg/kg-day | $1.0 * 10^{-6}$ or 1.0 / 1,000,000 |
| Scenario 1 Child DAD | $2.5 * 10^{-6}$ mg/kg-day | $1.3 * 10^{-6}$ or 1.3 / 1,000,000 |

Appendix D. Scenario 2 Exposure Assessment Details

*** Exposure Scenario 2: Trespassing Fishers/ Trespassing Swimmers (September 10, 1997- 2004/2005 based on the last sampling event for fish and water)**

Water data collected in the spring and summer months (May-August) was used to assess Scenario 2 swimming exposures. This data was selected for the evaluation of swimming because it is unlikely that individuals would be swimming in the fall and winter months of the year. In addition, the aeration technique used to restore water quality to WSP is more effective in warmer temperatures due to the increased rate of vaporization of PCE at higher temperatures. The net result is lower concentrations of PCE in WSP during the summer months because of the increased rate of vaporization to the atmosphere. The water data used for Scenario 2 exposures is presented below in Table D1. Seventy water samples were available during this timeframe. No water samples during the spring and summer months were available for 1997-1998. Thus, the sample set used for Scenario 2 exposures includes only spring and summer water data from 1999-2005.

Table D1. WSP Pond 1 Water data, May-August, 1999-2005

| Year | Average PCE Concentration* | Median | Maximum | Minimum | Colorado Surface Water Standard for WSP | Sample Count |
|------------------------|----------------------------|--------|---------|---------|---|--------------|
| Phase 1 1997 | 2.12** | 1.9 | 3.8 | 1.1 | 5 | 5 |
| Phase 2 1998 | 2.12** | 2.15 | 2.3 | 1.9 | 5 | 6 |
| 1999 | 0.46 | 0.5 | 0.72 | 0.25 | 0.8 | 22 |
| 2000 | 0.8 | 0.77 | 0.97 | 0.68 | 0.8 | 12 |
| 2001 | 0.93 | 0.84 | 1.7 | 0.25 | 0.8 | 12 |
| 2002 | 0.54 | 0.52 | 0.77 | 0.33 | 0.8 | 4 |
| Phase 3 2003 | 0.63 | 0.59 | 0.89 | 0.34 | 0.8 | 8 |
| 2004 | 0.27 | 0.25 | 0.34 | 0.25 | 0.8 | 4 |
| 2005 | 0.46 | 0.36 | 1.1 | 0.23 | 0.69 | 8 |

*All Concentrations reported in µg/L or parts per billion (ppb)

**Seasonal data was unavailable for the years 1997-1998. The average concentration of data collected during these years was used in the EPC calculation.

Source: TRI 1996-2000, ESC 2000-2005

All available fish tissue data was utilized for the Scenario 2 exposure assessment. Due to the limited amount of fish tissue data available, the fish samples collected before September 10, 1997 were also included in the data set for Scenario 2. Summary Statistics for all of the available fish tissue data collected for WSP is presented below in Table D2. In [Table 2](#) the fish tissue data is separated by species.

Table D2. Summary Statistics for All Available WSP Fish Tissue Data

| <i>All Fish</i> | |
|-------------------------------|---------|
| Mean | 99.26 |
| Median | 19.34 |
| Minimum | 0.98 |
| Maximum | 1153.18 |
| Count | 102 |
| 97.5% Upper Confidence Limit* | 220.02 |

* As recommended by the EPA's ProUCL statistical software

Scenario 2 Exposure Point Estimation for Swimming

Dermal absorbed dose for children and adults from Scenario 2 exposures were evaluated in this health consultation as a complete exposure pathway based on information gained through community interviews (CDPHE 2004). It states that “*we found several teenage children swam in the ponds approximately once a week, every week, throughout the past two summers (2002-2003).*” Scenario 2 exposure doses were calculated in the same manner as in Scenario 1. However, two adjustments to the Exposure Point Concentration (EPC) and Exposure Duration (ED) had to be made to account for the exposure differences in the trespassing swimmer pathway.

The first adjustment to Scenario 2 calculations is for the PCE concentration used as the (EPC). A large amount water data exists for Scenario 2 exposures that range from 1997-2005. The data set was modified to include only spring and summer water data as described above. No seasonal data was available for the years 1997 and 1998. Water data for these years was averaged and included in the final Scenario 2 data set. The mean, median, maximum, and minimum values for the entire data set are 0.65 ppb, 0.6 ppb, 0.23 ppb, and 2.12 ppb, respectively. The water data was entered into EPA's ProUCL statistical software and the 95% Upper Confidence Limit (UCL) was generated. The recommended 95% UCL on the mean assuming gamma distribution of the data was 0.68 ppb. This concentration was selected as the EPC for Scenario 2 exposures.

The other adjustment to the exposure dose calculations that had to be made was for the Exposure Duration (ED) or time period over which Scenario 2 exposures occurred. In Scenario 1, the adult ED was set to 8 years (1990-1997). The Scenario 2 ED was set to 9 years (1997-2005). The ED for children remained 6 years.

Scenario 2 Exposure Point Estimation for Fish

Scenario 2 fish consumption is a potential exposure pathway based on trespassing fishers from 1997-2005. The last available fish tissue samples were collected in October 2004. However, to remain consistent with the trespassing swimming exposures mentioned above the ED was set to 9 yrs. The Exposure Frequency (EF) was also set to 100 days per

year for trespassing fisher exposures. In addition, adjustments to the EPC and the ingestion rate were necessary to estimate fish consumption in Scenario 2.

Of the available fish data outlined above in Table D2, the mean fish tissue PCE concentration is equal to 99.26 ppb. The median value is 19.34, which indicates an unnormal statistical distribution of the data. The minimum fish tissue PCE concentration is 0.98 ppb and the maximum value is 1153.18 ppb. Statistically, some of the high end samples are considered data outliers as they lay more than 3 Standard Deviations from the mean. However, all data was retained in the final Scenario 2 fish consumption pathway data set. The ProUCL software, due to a non-parametric distribution of the data, recommended the Chebyshev 97.5% Upper Confidence Limit on the mean of 220.02 ppb. This concentration was selected as the EPC for the Scenario 2 fish consumption pathway.

The last adjustment to the Scenario 2 fish consumption estimates was to the ingestion rate (IR), or amount of fish consumed per day. Under the Scenario 2 exposure conditions, it is unlikely that a subsistence fisher could successfully trespass onto the property frequently enough to maintain a subsistence ingestion rate of fish from WSP. Therefore, only 2 ingestion rates were used to calculate exposure doses for trespassing fishers. The two ingestion rates that were used are mean fish consumption rate amongst the general population and the 90% UCL for “Consumers only.” It should be noted that the 90% UCL for “Consumers only” is a conservative estimate and likely overestimates the actual risk to a trespassing fisher.

Scenario 2 Exposure Dose Estimations

Scenario 2 exposure dose tables for the complete and potential exposure pathways identified in scenario are presented below in Tables D3-D5. Trespassing swimming was identified as a complete exposure pathway and trespassing fishing is considered a potential exposure pathway. As in Scenario 1, only carcinogenic risk was estimated for the trespassing swimmer pathway.

Table D3: Potential and non-cancer theoretical cancer risks for adult fish consumption exposures for Exposure Scenario 2 (Trespassing Adult Fishers 1997-2005)

| Description of Applicable Population | Ingestion Rate (grams/day) | Exposure Dose for Carcinogenic Effects (mg/kg-day) | Estimated Excess Cancer Risk | Exposure Dose for Noncarcinogenic Effects (mg/kg-day) | EPA's Oral Reference Dose (mg/kg-day) | Hazard Quotient (Noncancer Dose / Reference Dose) |
|---|----------------------------|--|---------------------------------------|---|---------------------------------------|---|
| Represents the mean fish consumption of individuals 18 and older living in the United States. | 7.5 ^a | $8.0 * 10^{-7}$ mg/kg-day | $4.3 * 10^{-7}$ (4.3 / 10,000,000) | $6.2 * 10^{-6}$ mg/kg-day | 0.01 mg/kg-day | $6.2 * 10^{-4}$ |
| Represents the upper limit of the 90 % estimate on the mean for "Consumers Only" individuals 18 and over living in the U.S. | 87.12 ^b | $9.3 * 10^{-6}$ mg/kg-day | $5.0 * 10^{-6}$ (5.0 / 1,000,000) | $7.2 * 10^{-5}$ mg/kg-day | 0.01 mg/kg-day | $7.2 * 10^{-3}$ |

^a This value represents EPA's mean value for the general population group (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4 - Freshwater/Estuarine Fish, p. 5-6*).

^b This value represents the EPA's value of 87.12g/day that represents the 90th percentile upper bound interval on the mean for "Consumers Only" (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4-Freshwater/Estuarine Fish, p. 5-43*).

Table D4: Potential non-cancer and theoretical cancer risk from Child fish consumption exposures for Exposure Scenario 2 (Trespassing child fishers 1997-2005)

| Description of Applicable Population | Ingestion Rate (grams/day) | Exposure Dose for Carcinogenic Effects (mg/kg-day) | Estimated Excess Cancer Risk | Exposure Dose for Noncarcinogenic Effects (mg/kg-day) | EPA's Oral Reference Dose (mg/kg-day) | Hazard Quotient (Noncancer Dose / Reference Dose) |
|--|----------------------------|--|---|---|---------------------------------------|---|
| Represents mean fish consumption rate of the general U.S. population (Ages 3-5) | 2.8 ^a | $9.5 * 10^{-7}$ mg/kg-day | $5.1 * 10^{-7}$ or 5.1 / 10,000,000 | $1.1 * 10^{-5}$ mg/kg-day | 0.01 mg/kg-day | $1.1 * 10^{-3}$ |
| Represents the upper confidence limit on the 90% estimate of the mean fish consumption for Consumers Only (Ages 3-5) | 45.63 ^b | $1.5 * 10^{-5}$ mg/kg-day | $8.1 * 10^{-6}$ or 8.1 / 1,000,000 | $1.8 * 10^{-4}$ mg/kg-day | 0.01 mg/kg-day | $1.8 * 10^{-2}$ |

^a This value is similar to the 90th percentile upper bound interval value of 2.58 g/day on the mean for children ages 3 to 5 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-7).

^b This value represents the 90% UCL on the mean fish consumption rate for children ages 3-5, "Consumers Only" (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-44).

Table D5: Theoretical Carcinogenic risks from swimming/wading in surface water for Scenario 2 exposures (Trespassing Swimmers 1997-2005)

| Dose Parameter | Exposure Dose for Carcinogenic Health Effects | Estimated Cancer Risk |
|-----------------------|--|---|
| Scenario 2 Adult DAD | $3.8 * 10^{-7}$ mg/kg-day | $2.0 * 10^{-7}$ or 2.0 / 10,000,000 |
| Scenario 2 Child DAD | $4.5 * 10^{-7}$ mg/kg-day | $2.4 * 10^{-7}$ or 2.4 / 10,000,000 |

Appendix E. Scenario 3 Exposure Assessment Details

* **Exposure Scenario 3:** *Exposures that occurred prior to WSP closure combined with trespassing exposures (Scenario 1 and Scenario 2 combined)*

Table E1: Potential non-cancer and theoretical cancer risks for adult fish consumption for Exposure Scenario 3 (Adult fish consumption for Scenarios 1 and 2 combined)

| Description of Applicable Population | Ingestion Rate (grams/day) | Exposure Dose for Carcinogenic Effects (mg/kg-day) | Estimated Excess Cancer Risk | Exposure Dose for Noncarcinogenic Effects (mg/kg-day) | EPA's Oral Reference Dose (mg/kg-day) | Hazard Quotient (Noncancer Dose / Reference Dose) |
|---|----------------------------|--|--|---|---------------------------------------|---|
| Represents the mean fish consumption of individuals 18 and older living in the United States. | 7.5 ^a | 1.0 * 10 ⁻⁶ mg/kg-day | 5.4 * 10 ⁻⁷ (5.4 / 10,000,000) | 8.2 * 10 ⁻⁶ mg/kg-day | 0.01 mg/kg-day | 8.2 * 10 ⁻⁴ |
| Represents the upper limit of the 90 % estimate on the mean for "Consumers Only" individuals 18 and over living in the U.S. | 87.12 ^b | 1.2 * 10 ⁻⁵ mg/kg-day | 6.4 * 10 ⁻⁶ (6.4 / 1,000,000) | 9.5 * 10 ⁻⁵ mg/kg-day | 0.01 mg/kg-day | 9.5 * 10 ⁻³ |
| Represents the default value for a subsistence population living in the U.S. | 142.4 ^c | 4.3 * 10 ⁻⁶ mg/kg-day | 2.3 * 10 ⁻⁶ (2.3 / 1,000,000) | 3.8 * 10 ⁻⁵ mg/kg-day | 0.01 mg/kg-day | 3.8 * 10 ⁻³ |

^a This value represents EPA's mean value for the general population group (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4 - Freshwater/Estuarine Fish, p. 5-6*).

^b This value represents the EPA's value of 87.12g/day that represents the 90th percentile upper bound interval on the mean for "Consumers Only" (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4-Freshwater/Estuarine Fish, p. 5-43*).

^c Scenario 2 exposure doses were not calculated at this ingestion rate. Therefore, the information listed here for 142.4 g/day is identical to Scenario 1 adult fish consumption. This value is within the 99th percentile upper bound interval range of 125.27 – 156.84 g/day for the general population group (Age 18 and Older) (EPA, 2002, *Per Capita Fish Ingestion Rate, Table 4 - Freshwater/Estuarine Fish, p. 5-6*).

Table E2: Potential non-cancer and theoretical cancer risk from Child fish consumption for Exposure Scenario 3 (Child fish consumption for Scenarios 1 and 2 combined)

| Description of Applicable Population | Ingestion Rate (grams/day) | Exposure Dose for Carcinogenic Effects (mg/kg-day) | Estimated Excess Cancer Risk | Exposure Dose for Noncarcinogenic Effects (mg/kg-day) | EPA's Oral Reference Dose (mg/kg-day) | Hazard Quotient (Noncancer Dose / Reference Dose) |
|---|----------------------------|--|-------------------------------------|---|---------------------------------------|---|
| Represents mean fish consumption rate of the general U.S. population (Ages 3-5) | 2.8 ^a | $1.3 * 10^{-6}$ mg/kg-day | $6.8 * 10^{-7}$ or 6.8 / 10,000,000 | $1.5 * 10^{-5}$ mg/kg-day | 0.01 mg/kg-day | $1.5 * 10^{-3}$ |
| Represents the upper confidence limit on the 90% estimate of the mean fish consumption for Consumers Only (Ages 3-5) | 45.63 ^b | $2.0 * 10^{-5}$ mg/kg-day | $1.1 * 10^{-5}$ or 1.1 / 100,000 | $2.4 * 10^{-4}$ mg/kg-day | 0.01 mg/kg-day | $2.4 * 10^{-2}$ |
| Represents the upper confidence limit on the 90% estimate of the 99 th percentile of fish consumption for the general U.S. population (ages 3-5) | 61.53 ^c | $6.7 * 10^{-6}$ mg/kg-day | $3.6 * 10^{-6}$ or 3.6 / 1,000,000 | $7.9 * 10^{-5}$ mg/kg-day | 0.01 mg/kg-day | $7.9 * 10^{-3}$ |

^a This value is similar to the 90th percentile upper bound interval value of 2.58 g/day on the mean for children ages 3 to 5 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-7).

^b This value represents the 90% UCL on the mean fish consumption rate for children ages 3-5, "Consumers Only" (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-44).

^c Scenario 2 exposure doses were not calculated at this ingestion rate. Therefore, the information listed here for 61.53 g/day is identical to Scenario 1 child fish consumption. This value represents the 90% UCL on the 99th percentile for children ages 3 to 5 (EPA, 2002, *Per Capita Fish Ingestion Rate, Table5-Freshwater/Estuarine Fish*, p. 5-7).

Table E3: Theoretical carcinogenic risks from swimming/wading in surface water for Scenario 3 exposures (Swimming Scenarios 1 and 2 combined)

| Dose Parameter | Exposure Dose for Carcinogenic Health Effects | Estimated Cancer Risk |
|-----------------------|--|--|
| Scenario 3 Adult DAD | $2.3 * 10^{-6}$ mg/kg-day | $1.2 * 10^{-6}$ or 1.2 / 1,000,000 |
| Scenario 3 Child DAD | $3.0 * 10^{-6}$ mg/kg-day | $1.55 * 10^{-6}$ or 1.55 / 1,000,000 |

Appendix F: Additional Information on Calculating Exposure Doses

F1. Fish Ingestion Exposure Dose Calculations (ATSDR 2005)

Fish Ingestion exposure doses were calculated in the following manner:

$$\text{Dose (mg/kg or ppm)} = \frac{\mathbf{C * IR * AF * EF}}{\mathbf{BW}}$$

$$\text{Where EF} = \frac{\mathbf{F * ED}}{\mathbf{AT}}$$

| Variable | Units | Description | Adult | Child |
|--------------------|--------------|--|--------------|--------------|
| C ₁ | mg/kg | Scenario 1 EPC | 0.02832 | 0.02832 |
| C ₂ | mg/kg | Scenario 2 EPC | 0.22002 | 0.22002 |
| IR ₁₋₁ | kg/day | Scenario 1, Ingestion Rate #1 | 0.0075 | 0.0028 |
| IR ₁₋₂ | kg/day | Scenario 1, Ingestion Rate #2 | 0.08712 | 0.04563 |
| IR ₁₋₃ | kg/day | Scenario 1, Ingestion Rate #3 | 0.1424 | 0.06153 |
| IR ₂₋₁ | kg/day | Scenario 2, Ingestion Rate #1 | 0.0075 | 0.0028 |
| IR ₂₋₂ | kg/day | Scenario 2, Ingestion Rate #2 | 0.08712 | 0.04563 |
| AF | unitless | Bioavailability Factor | 1 | 1 |
| F ₁ | days/year | Scenario 1 Frequency of Fish Consumption | 240 | 240 |
| F ₂ | days/year | Scenario 2 Frequency of Fish Consumption | 100 | 100 |
| ED ₁ | years | Scenario 1 Exposure Duration | 8 | 6 |
| ED ₂ | years | Scenario 2 Exposure Duration | 9 | 6 |
| AT _c | days | Averaging Time for Cancer | 25,550 | 25,550 |
| AT _{nc-1} | days | Scenario 1 Averaging Time for Non-cancer | 2,920 | 2,190 |
| AT _{nc-2} | days | Scenario 2 Averaging Time for Non-cancer | 3,285 | 2,190 |
| BW | kg | Body Weight | 70 | 14.5 |

F2. Dermal Absorbed Dose Calculation for Organic Compounds-Water Contact (EPA RAGS Part E EPA 2004)

DAD (mg/cm²-event) is calculated for Organic Compounds as follows:

$$\text{DA event (DAev)} = 2 \text{ FA} * \text{K}_p * \text{C}_w * \text{CF} \sqrt{(6\tau_{ev} * t_{ev} \div \pi)} \quad (\text{EPA 2004, Equation 3-2})$$

$$\text{DAD (mg/cm}^2\text{-event)} = \frac{\text{DAev} * \text{ED} * \text{EF} * \text{SA}}{\text{BW} * \text{AT}} \quad (\text{EPA 2004, Equation 3-1})$$

| Variable | Units | Description | Adult | Child |
|--------------------------------|-------------------|---|------------------------|------------------------|
| DAD | mg/kg-day | Dermally absorbed dose | CS | CS (Chemical Specific) |
| FA | dimensionless | Fraction absorbed water for PCE | 1 | 1 |
| K _p | cm/hour | Dermal permeability coefficient of PCE | 3.3 * 10 ⁻² | 3.3 * 10 ⁻² |
| C _w | mg/L | Concentration of chemical in water | SS | SS (Scenario Specific) |
| CF | L/cm ³ | Conversion factor | 1.0 * 10 ⁻³ | 1.0 * 10 ⁻³ |
| τ _{ev} | hours | Lag Time per event | 0.91 | 0.91 |
| t _{ev} | hour/event | Event Duration | 0.5 | 0.5 |
| ED | years | Exposure duration | SS | SS |
| ET | hours/day | Exposure time | 0.5 | 0.5 |
| EF | days/year | Exposure frequency | SS | SS |
| SA | cm ² | Skin surface area available for contact | 18,000 | 6,600 |
| BW _{adult} | kg | Adult Body weight | 70 | NA (Not Applicable) |
| BW _{child} | kg | Child Body weight | NA | 14.5 |
| AT _{adult non-cancer} | days | Averaging time | SS | NA |
| AT _{adult cancer} | days | Averaging time | 25,550 | NA |
| AT _{child non-cancer} | days | Averaging time | NA | 2,190 |
| AT _{child cancer} | days | Averaging time | NA | 25,550 |

| Parameter | Conc. (mg/cm ²) | EF | ED | AT (non-cancer) | CSFo 1/(mg/kg-day) | RfDo (mg/kg-day) | Carcinogenic Risks | | Noncarcinogenic Risk | |
|--|--------------------------------|-----|----|--------------------|-----------------------|---------------------|------------------------|------------------------|------------------------|------------------------|
| | | | | | | | DAD (mg/kg-day) | Cancer Risk | DAD (mg/kd-day) | Hazard Quotient |
| Scenario 1 Child (Ages 6 & under) | 3.8 * 10 ⁻⁶ | 100 | 6 | 2,190 | 0.54 | 0.01 | 2.5 * 10 ⁻⁶ | 1.3 * 10 ⁻⁶ | 2.9 * 10 ⁻⁵ | 2.9 * 10 ⁻⁵ |
| Scenario 2 Child (Ages 6 & under) | 6.8 * 10 ⁻⁷ | 100 | 6 | 2,190 | 0.54 | 0.01 | 4.9 * 10 ⁻⁷ | 2.6 * 10 ⁻⁷ | 5.2 * 10 ⁻⁶ | 5.2 * 10 ⁻⁶ |
| Scenario 3 Child (Ages 6 & under) | NA | NA | NA | NA | 0.54 | 0.01 | 3.0 * 10 ⁻⁶ | 1.6 * 10 ⁻⁶ | 3.4 * 10 ⁻⁵ | 3.4 * 10 ⁻⁵ |
| Scenario 1 Adult | 3.8 * 10 ⁻⁶ | 100 | 8 | 2,920 | 0.54 | 0.01 | 1.9 * 10 ⁻⁶ | 1.0 * 10 ⁻⁶ | 1.6 * 10 ⁻⁵ | 1.6 * 10 ⁻⁵ |
| Scenario 2 Adult | 6.8 * 10 ⁻⁷ | 100 | 9 | 3,285 | 0.54 | 0.01 | 4.2 * 10 ⁻⁷ | 2.3 * 10 ⁻⁷ | 3.0 * 10 ⁻⁶ | 3.0 * 10 ⁻⁶ |
| Scenario 3 Adult | NA | NA | NA | NA | 0.54 | 0.01 | 3.4 * 10 ⁻⁶ | 1.8 * 10 ⁻⁶ | 3.7 * 10 ⁻⁵ | 3.7 * 10 ⁻⁵ |

Appendix G. ATSDR Public Health Hazard Categories

| Category / Definition | Data Sufficiency | Criteria |
|--|---|--|
| <p>A. Urgent Public Health Hazard</p> <p>This category is used for sites where short-term exposures (< 1 yr) to hazardous substances or conditions could result in adverse health effects that require rapid intervention.</p> | <p>This determination represents a professional judgment based on critical data which ATSDR has judged sufficient to support a decision. This does not necessarily imply that the available data are complete; in some cases additional data may be required to confirm or further support the decision made.</p> | <p>Evaluation of available relevant information* indicates that site-specific conditions or likely exposures have had, are having, or are likely to have in the future, an adverse impact on human health that requires immediate action or intervention. Such site-specific conditions or exposures may include the presence of serious physical or safety hazards.</p> |
| <p>B. Public Health Hazard</p> <p>This category is used for sites that pose a public health hazard due to the existence of long-term exposures (> 1 yr) to hazardous substance or conditions that could result in adverse health effects.</p> | <p>This determination represents a professional judgment based on critical data which ATSDR has judged sufficient to support a decision. This does not necessarily imply that the available data are complete; in some cases additional data may be required to confirm or further support the decision made.</p> | <p>Evaluation of available relevant information* suggests that, under site-specific conditions of exposure, long-term exposures to site-specific contaminants (including radionuclides) have had, are having, or are likely to have in the future, an adverse impact on human health that requires one or more public health interventions. Such site-specific exposures may include the presence of serious physical or safety hazards.</p> |
| <p>C. Indeterminate Public Health Hazard</p> <p>This category is used for sites in which “critical” data are insufficient with regard to extent of exposure and/or toxicologic properties at estimated exposure levels.</p> | <p>This determination represents a professional judgment that critical data are missing and ATSDR has judged the data are insufficient to support a decision. This does not necessarily imply all data are incomplete; but that some additional data are required to support a decision.</p> | <p>The health assessor must determine, using professional judgment, the “criticality” of such data and the likelihood that the data can be obtained and will be obtained in a timely manner. Where some data are available, even limited data, the health assessor is encouraged to the extent possible to select other hazard categories and to support their decision with clear narrative that explains the limits of the data and the rationale for the decision.</p> |
| <p>D. No Apparent Public Health Hazard</p> <p>This category is used for sites where human exposure to contaminated media may be occurring, may have occurred in the past, and/or may occur in the future, but the exposure is not expected to cause any adverse health effects.</p> | <p>This determination represents a professional judgment based on critical data which ATSDR considers sufficient to support a decision. This does not necessarily imply that the available data are complete; in some cases additional data may be required to confirm or further support the decision made.</p> | <p>Evaluation of available relevant information* indicates that, under site-specific conditions of exposure, exposures to site-specific contaminants in the past, present, or future are not likely to result in any adverse impact on human health.</p> |
| <p>E: No Public Health Hazard</p> <p>This category is used for sites that, because of the absence of exposure, do NOT pose a public health hazard.</p> | <p>Sufficient evidence indicates that no human exposures to contaminated media have occurred, none are now occurring, and none are likely to occur in the future</p> | |

Appendix H: Additional Information on Tetrachloroethylene

H1. Toxicological Evaluation

Due to widespread use of PCE in the dry cleaning, household products, and other industries, PCE is a common environmental contaminant. Environmental exposures to contaminated groundwater and surface water can occur as a result of PCE environmental releases from industrial wastes, leakage from underground storage tanks, and on-site spills.

The basic objective of a toxicological evaluation is to identify what adverse health effects a chemical causes, and how the appearance of these adverse effects depends on dose. In addition, the toxic effects of a chemical frequently depend on the route of exposure (oral, inhalation, dermal) and the duration of exposure (acute, subchronic, chronic or lifetime). In general, acute and chronic neurological changes, and liver and kidney toxicity, have been observed in humans and animals exposed to PCE (See Appendix... for PCE health effect fact sheet). It is important to note that estimates of human health risks may be based on evidence of health effects in humans and/or animals depending upon the availability of data.

The toxicity assessment process is usually divided into two parts: the cancer effects and the non-cancer effects of the chemical. This two-part approach is employed because there are typically major differences in the time-course of action and the shape of the dose-response curve for cancer and non-cancer effects.

The USEPA IRIS (EPA, 1988) has established an oral reference dose (RfD) of 0.01 mg/kg/day for non-cancer effects. The RfD is based on liver toxicity in mice and weight gain in rats. An RfD is the daily dose in humans (with uncertainty spanning perhaps an order of magnitude), including sensitive subpopulations, that is likely to be without an appreciable risk of noncancer adverse health effects during a lifetime exposure.

The USEPA has not established in the EPA IRIS an inhalation reference concentration as well as a carcinogenicity assessment for lifetime exposures to PCE. However, in the absence of relevant values in the EPA IRIS, the USEPA Office of Solid Waste and Emergency Response (OSWER) recommends using the Cal EPA oral slope factor of 0.54 per mg/kg/day for PCE (EPA, 2003, OSWER Directive No. 9285.7-75). The Cal EPA classifies PCE to be an animal carcinogen and a possible human carcinogen. This classification is based on the observed increased incidence of hepatocellular carcinoma in male and female mice exposed orally to PCE. Additionally, human epidemiological studies suggest that PCE is possibly carcinogenic in humans. The most consistent tumor sites in humans are the esophagus and lymphatic system, but the available information is insufficient to quantify cancer risks. Therefore, quantitative estimates of the potential of PCE to induce human cancer are inferred from animal data. Additionally, estimating the cancer slope factor is often complicated by the fact that observable increases in cancer incidence usually occur only at relatively high doses. Therefore, it is necessary to use mathematical models to extrapolate from the observed high dose data to the desired slope at low dose. In order to account for the uncertainty in this extrapolation process, EPA typically chooses to employ the upper 95th confidence limit of the slope as the Slope

Factor. That is, there is a 95% probability that the true cancer potency is lower than the value chosen for the Slope Factor.

ATSDR has derived an acute-duration oral minimal risk levels (MRLs) for PCE of 0.05 mg/kg/day. The acute MRL is based on an increase in total spontaneous activity (locomotion and rearing) in mice. An MRL is the dose of a compound that is an estimate of daily human exposure that is likely to be without an appreciable risk of adverse noncancerous effects of a specified duration of exposure. The acute MRL addresses short-term exposures of 14 days or less. ATSDR has not established intermediate- and chronic-duration oral MRLs for PCE.

H2. ATSDR Public Health Statement for Tetrachlorethylene

Public Health Statement for Tetrachloroethylene

CAS# 127-18-4

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

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

The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup. Tetrachloroethylene has been found in at least 771 of the 1,430 current or former NPL sites. However, it's unknown how many NPL sites have been evaluated for this substance. As more sites are evaluated, the sites with tetrachloroethylene may increase. This is important because exposure to this substance may harm you and because these sites may be sources of exposure.

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

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

What is tetrachloroethylene?

Tetrachloroethylene is a synthetic chemical that is widely used for dry cleaning of fabrics and for metal-degreasing operations. It is also used as a starting material (building block) for making other chemicals and is used in some consumer products. Other names for tetrachloroethylene include perchloroethylene, PCE, perc, tetrachloroethene, perclene,

and perchlor. It is a nonflammable liquid at room temperature. It evaporates easily into the air and has a sharp, sweet odor. Most people can smell tetrachloroethylene when it is present in the air at a level of 1 part in 1 million parts of air (ppm) or more. In an experiment, some people could smell tetrachloroethylene in water at a level of 0.3 ppm.

What happens to tetrachloroethylene when it enters the environment?

Tetrachloroethylene enters the environment mostly by evaporating into the air during use. It can also get into water supplies and the soil during disposal of sewage sludge and factory waste and when leaking from underground storage tanks. Tetrachloroethylene may also get into the air, soil, or water by leaking or evaporating from storage and waste sites. It can stay in the air for several months before it is broken down into other chemicals or is brought back down to the soil and water by rain.

Much of the tetrachloroethylene that gets into water and soil will evaporate into the air. However, because tetrachloroethylene can travel through soils quite easily, it can get into underground drinking water supplies. If it gets into underground water, it may stay there for many months without being broken down. If conditions are right, bacteria will break down some of it and some of the chemicals formed may also be harmful. Under some conditions, tetrachloroethylene may stick to the soil and stay there. It does not seem to build up in animals that live in water, such as fish, clams, and oysters. We do not know if it builds up in plants grown on land.

How might I be exposed to tetrachloroethylene?

People can be exposed to tetrachloroethylene from environmental and occupational sources and from consumer products. Common environmental levels of tetrachloroethylene (called background levels) are several thousand times lower than levels found in some workplaces. Background levels are found in the air we breathe, in the water we drink, and in the food we eat. The chemical is found most frequently in air and, less often, in water. Tetrachloroethylene gets into air by evaporation from industrial or dry cleaning operations. It is also released from areas where chemical wastes containing it are stored. It is frequently found in water. For example, tetrachloroethylene was found in 38% of 9,232 surface water sampling sites throughout the United States. There is no similar information on how often the chemical is found in air samples, but we know it is widespread. We do not know how often it is found in soil, but in one study, it was found in 5% of 359 sediment samples.

In general, tetrachloroethylene levels in air are higher in cities or industrial areas where it is in use more than in more rural or remote areas. You can smell it at levels of 1 ppm in air. However, the background level of tetrachloroethylene in air is usually less than 1 part in 1 billion parts of air (ppb). The air close to dry cleaning shops and chemical waste sites has levels of tetrachloroethylene higher than background levels. These levels are usually less than 1 ppm, the level at which you can smell it. Water, both above and below ground, may contain tetrachloroethylene. Levels in water are also usually less than 1 ppb. Levels in contaminated water near disposal sites are higher than levels in water far away from those sites. Water polluted with this chemical may have levels greater than 1 ppm. In soil, background levels are probably 100–1,000 times lower than

1 ppm.

You can also be exposed to tetrachloroethylene by using certain consumer products. Products that may contain it include water repellents, silicone lubricants, fabric finishers, spot removers, adhesives, and wood cleaners. Although uncommon, small amounts of tetrachloroethylene have been found in food, especially food prepared near a dry cleaning shop. When you bring clothes home from the dry cleaners, the clothes may release small amounts of tetrachloroethylene into the air. The full significance to human health of these exposures to small amounts of tetrachloroethylene is unknown, but to date, they appear to be relatively harmless. Tetrachloroethylene can also be found in the breast milk of mothers who have been exposed to the chemical.

The people with the greatest chance of exposure to tetrachloroethylene are those who work with it. According to estimates from a survey conducted by the National Institute for Occupational Safety and Health (NIOSH), more than 650,000 U.S. workers may be exposed.

For the general population, the estimated amount that a person might breathe per day ranges from 0.08 to 0.2 milligrams. The estimated amount that most people might drink in water ranges from 0.0001 to 0.002 milligrams per day. These are very small amounts.

How can tetrachloroethylene enter and leave my body?

Tetrachloroethylene can enter your body when you breathe air containing it. How much enters your body in this way depends on how much of the chemical is in the air, how fast and deeply you are breathing, and how long you are exposed to it. Tetrachloroethylene may also enter your body when you drink water or eat food containing the chemical. How much enters your body in this way depends on how much of the chemical you drink or eat. These two exposure routes are the most likely ways people will take in tetrachloroethylene. These are also the most likely ways that people living near areas polluted with the chemical, such as hazardous waste sites, might be exposed to it. If tetrachloroethylene is trapped against your skin, a small amount of it can pass through into your body. Very little tetrachloroethylene in the air can pass through your skin into your body.

Most tetrachloroethylene leaves your body from your lungs when you breathe out. This is true whether you take in the chemical by breathing, drinking, eating, or touching it. A small amount of the tetrachloroethylene is changed by your body (especially your liver) into other chemicals that are removed from your body in urine. Most of the changed tetrachloroethylene leaves your body in a few days. Some of it that you take in is found in your blood and other tissues, especially body fat. Part of the tetrachloroethylene that is stored in fat may stay in your body for several days or weeks before it is eliminated.

How can tetrachloroethylene affect my health?

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

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

Tetrachloroethylene has been used safely as a general anesthetic agent, so at high concentrations, it is known to produce loss of consciousness. When concentrations in air are high—particularly in closed, poorly ventilated areas—single exposures can cause dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, unconsciousness, and death. Irritation may result from repeated or extended skin contact with the chemical. As you might expect, these symptoms occur almost entirely in work (or hobby) environments when individuals have been accidentally exposed to high concentrations or have intentionally abused tetrachloroethylene to get a "high." In industry, most workers are exposed to levels lower than those causing dizziness, sleepiness, and other nervous system effects. The health effects of breathing in air or drinking water with low levels of tetrachloroethylene are not definitely known. However, at levels found in the ambient air or drinking water, risk of adverse health effects is minimal. The effects of exposing babies to tetrachloroethylene through breast milk are unknown. Results from some studies suggest that women who work in dry cleaning industries where exposures to tetrachloroethylene can be quite high may have more menstrual problems and spontaneous abortions than women who are not exposed. However, it is not known for sure if tetrachloroethylene was responsible for these problems because other possible causes were not considered.

Results of animal studies, conducted with amounts much higher than those that most people are exposed to, show that tetrachloroethylene can cause liver and kidney damage and liver and kidney cancers even though the relevance to people is unclear. Although it has not been shown to cause cancer in people, the U.S. Department of Health and Human Services has determined that tetrachloroethylene may reasonably be anticipated to be a human carcinogen. The International Agency for Research on Cancer (IARC) has determined that tetrachloroethylene is probably carcinogenic to humans. Exposure to very high levels of tetrachloroethylene can be toxic to the unborn pups of pregnant rats and mice. Changes in behavior were observed in the offspring of rats that breathed high levels of the chemical while they were pregnant. Rats that were given oral doses of tetrachloroethylene when they were very young, when their brains were still developing, were hyperactive when they became adults. How tetrachloroethylene may affect the developing brain in human babies is not known.

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

One way of testing for tetrachloroethylene exposure is to measure the amount of the chemical in the breath, much the same way breath alcohol measurements are used to

determine the amount of alcohol in the blood. This test has been used to measure levels of the chemical in people living in areas where the air is contaminated with tetrachloroethylene or those exposed to the chemical through their work. Because it is stored in the body's fat and is slowly released into the bloodstream, it can be detected in the breath for weeks following a heavy exposure. Tetrachloroethylene can be detected in the blood. Also, breakdown products of the chemical can be detected in the blood and urine of people exposed to tetrachloroethylene. Trichloroacetic acid (TCA), a breakdown product of tetrachloroethylene can be detected for several days after exposure. These tests are relatively simple to perform. The breath, blood, or urine must be collected in special containers and then sent to a laboratory for testing. Because exposure to other chemicals can produce the same breakdown products in the urine and blood, the tests for breakdown products cannot determine if you have been exposed only to tetrachloroethylene.

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

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

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

The EPA maximum contaminant level for the amount of tetrachloroethylene that can be in drinking water is 0.005 milligrams tetrachloroethylene per liter of water (mg/L) (0.005 ppm).

EPA has established regulations and procedures for dealing with tetrachloroethylene, which it considers a hazardous waste. Many regulations govern its disposal. If amounts greater than 100 pounds are released to the environment, the National Response Center of the federal government must be told immediately.

OSHA limits the amount of tetrachloroethylene that can be present in workroom air. This amount is limited to 100 ppm for an 8-hour workday over a 40-hour workweek.

NIOSH recommends that tetrachloroethylene be handled as a chemical that might potentially cause cancer and states that levels of the chemical in workplace air should be as low as possible.

1.8 Where can I get more information?

For additional information on tetrachloroethylene, refer to the ATSDR Toxicological Profile at: <http://www.atsdr.cdc.gov/toxprofiles/tp18.html>

References

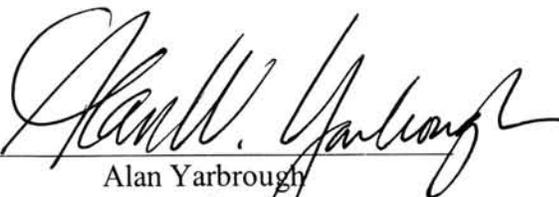
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CERTIFICATION

This Willow Springs Ponds Health Consultation was prepared by the Colorado Department of Public Health and Environment 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 health consultation was begun. Editorial review was completed by the Cooperative Agreement partner.


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


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