ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

VALMONT TCE SITE:
FORMERLY VALMONT INDUSTRIAL PARK SITE
(A/K/A VALMONT INDUSTRIAL PARK)
WEST HAZELTON, LUZERNE COUNTY, PENNSYLVANIA
EPA FACILITY ID: PAD982363970
APRIL 28, 2006

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
Agency for Toxic Substances and Disease Registry

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 30-day public comment period. Subsequent to the public comment period, ATSDR addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes the public health assessment 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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WEST HAZELTON, LUZERNE COUNTY, PENNSYLVANIA

EPA FACILITY ID: PAD982363970

Prepared by:

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

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Executive Summary

The Valmont Trichloroethylene Site is immediately northwest of the borough of West Hazleton, Pennsylvania. The site presently consists of the Chromatex Plant #2 and surrounding approximately seven acres and is in the Valmont Industrial Park. Chromatex Plant #2 (large manufacturing building on the property) operated since the early 1960s as an industrial site for several enterprises, including the manufacturing of coffins and knitted fabrics. In 1978, Chromatex Plant #2 began using trichloroethylene or TCE in its operations for upholstery fabric manufacturing. Past operations at the site caused chemical contamination (especially TCE) in the ground water under the site and under the closest residential neighborhood to the northeast. EPA Toxics Release Inventory records show that an estimated 7,640 pounds of TCE were released in 1987 to the air in and around the plant during normal manufacturing processes. The Chromatex Plant #2 discontinued the use of TCE in 1988 and subsequently closed its business on March 2001.

The purpose of this public health assessment is to evaluate on-site and off-site contamination, human exposure pathways, public health concerns, and associated public health implications. In preparing this public health assessment (PHA), the Agency for Toxic Substances and Disease Registry (ATSDR) and the Pennsylvania Department of Health (PADOH) reviewed available data from the United States Environmental Protection Agency Region 3 (EPA) and the Pennsylvania Department of Environmental Protection (PADEP), formerly known as the Pennsylvania Department of Environmental Resources. Additionally, since 2001, ATSDR and PADOH staff members have conducted site visits and meetings with governmental officials, community residents, and local physicians to gather more information about the site as well as to identify and discuss community health concerns. Two public availability sessions were held by PADOH and ATSDR. One public availability session was specifically for the public comment period/version of the Valmont TCE PHA. The public comment period for this document was from July 14, 2005 to August 30, 2005 and had been extended into November 2005.

ATSDR initially identified the health concerns of this community through meetings with PADEP, EPA, and the local township, as well as through EPA records of public health concerns from 2001 into 2004 and through PADOH home visits conducted in 2002. Additional community concerns were identified through three major public meetings with the involved agencies and the affected community in the summer and fall of 2002 and February of 2004. However, ATSDR and PADOH also collected community concerns through reviewing information in health survey forms distributed in 2002 by a local community group. ATSDR and PADOH determined that the exposed residential population was too small to complete a reliable review of health outcome data such as cancer registry information.

Currently and for the future, the Valmont TCE site is classified by ATSDR and PADOH as posing *no apparent public health hazard*. In the published initial version Valmont TCE site PHA, it was stated that future exposures posed an indeterminate health hazard. Significant changes have occurred at the site since the initial document publication. Specifically, a large portion of on-site contaminated soil has been remediated, plans were made for ground water treatment, and indoor air carbon filtration units and sump pump covers were installed in selected residences above the plume.

Community members felt that the conclusion regarding a public health threat from past exposures at this site needed further substantiation in the public comment version of this document. ATSDR and PADOH have clarified that language in this Final version of this document. There is some uncertainty about *past* cumulative effects from all of the combined VOC exposures from all pathways at this site. Based on a thorough evaluation, ATSDR and PADOH conclude that exposures in the past to the ground water at the *highest levels* off-site could possibly have yielded adverse health effects (assuming *ten years* of exposure). After public water was installed in the neighborhood in 1988, health risks at this site were significantly decreased.

In this document, ATSDR and PADOH conclude that from a public health assessment perspective, current and future exposures to contaminants from the underground plume in residential indoor air are not likely to cause health effects in residents. However, EPA has determined that eight homes in the site area currently have levels of site-related contaminants in their indoor air above EPA's acceptable risk for increased cancer. As a result, EPA installed carbon filtration units in these homes and plans to install household vapor reduction systems at these homes in the near future. ATSDR and PADOH support these actions as proactive public health measures that will reduce stress and uncertainty for the affected residents. ATSDR and PADOH recommend that installing these systems in any homes affected by the plume as determined by EPA would provide a similar benefit for all of the residents in the affected neighborhood.

In the April 2003 Health Consultation (*Public Health Evaluation of Soil Samples*), ATSDR and PADOH concluded that current exposures to chemicals in off-site soil posed an indeterminate public health hazard. In this document, past, current and future exposures to off-site contaminated soil, after further review, are classified as posing no apparent public health hazard.

PADOH recommends that residents continue to remove any possible sources of VOCs in their homes originating from household products and not the plume. EPA should continue to collect additional private well samples and monitoring well samples to determine if ground water quality is improving now that on-site soil remediation has taken place and after EPA's currently planned future off-site ground water treatment is in place and/or is completed. Furthermore, if household carbon filtration or vapor reduction systems are not installed in all of the homes determined to be affected by the plume at this site, ATSDR and PADOH recommend that the indoor air in these affected homes be retested in the future to ensure that indoor air contaminant levels remain below levels of public health concern.

Background and Statement of the Issues

Introduction

The Pennsylvania Department of Health (PADOH), operating through a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR), was requested by the United States Environmental Protection Agency Region 3 (EPA) to prepare a public health assessment (PHA) concerning the Valmont Trichloroethylene Site (also called the Valmont TCE Site and formerly called the Valmont Industrial Site). The Valmont TCE Site is situated northwest of West Hazleton, Pennsylvania, and consists of the Chromatex Plant #2. The purpose of this public health assessment is to evaluate on-site and off-site contamination, human exposure pathways, public health concerns, and associated public health implications. In this PHA, ***on-site*** refers to areas within the Valmont TCE Site (specifically around the Chromatex Plant # 2) property boundary and ***off-site*** refers to homes, residential wells and monitoring wells not on the industrial property.

Past operations at the site caused chemical contamination (especially trichloroethylene or TCE) to the ground water under the site and under the residential neighborhood northeast of the site, including the residents' drinking water. Apparently, TCE was used for about 10 years in the operations. At various times between 1987 and 2004, the EPA and the Pennsylvania Department of Environmental Protection (PADEP) - formerly known as the Pennsylvania Department of Environmental Resources (PADER) - collected and analyzed various environmental media for organic compounds such as TCE and other contaminants. In addition, the 1987 EPA TRI data shows that an estimated 7640 pounds of TCE were released that year to the air in and around the Chromatex Plant #2 during normal manufacturing processes. In preparing this PHA, ATSDR and PADOH reviewed the EPA and PADEP data. Numerous chemicals of *potential* concern were detected on-site and off-site and were evaluated for this PHA.

In addition, ATSDR and PADOH conducted site visits and met with other government officials and community residents during public meetings to gather more information about the site as well as to identify community health concerns. Some of the health-related concerns that were raised by the community are addressed in the Community Health Concerns section of this document and/or were addressed during public meetings and discussions with the ATSDR and PADOH staff and also with the ATSDR's Regional Medical Toxicologist.

Site Description and History

The Valmont TCE site is an area of contaminated soil and ground water near the intersection of Jaycee Drive and Deer Run Road in Hazle Township and West Hazleton, Luzerne County, Pennsylvania (Appendix A – Figures 1 and 2). Bordering the site, the Valmont Business Park (industrial park) is south and west of the site and extends for about a one-half mile radius. The approximate residential area affected by the contamination is bounded on the east by Route 93 and a small shopping center, and by the Black Creek on the north (Appendix A - Figure 2). Locational coordinates of the Valmont TCE Chromatex Plant #2 and the site on a Geographical Information Systems map are 40.9682 north and 76.0156 west. The EPA identification number for the site is #1387010250785PA and EPA CERCLIS ID is PAD982363970. The initial CERCLIS records for the site may be found on the EPA web site [1].

The history of the site dates back to 1963 when a building shell was constructed on the property by CAN DO Inc., the first known owner of the property. The Chromatex Plant #2 (large manufacturing building on the property) had operated since the early 1960s as an industrial site for several enterprises, including the manufacturing of coffins and knitted fabrics. In 1965, Wallace Metal Products, a coffin manufacturer purchased the property. From 1972-1978, Futura Fabrics operated the facility. Starting in 1978, Chromatex, Incorporated, an upholstery fabric manufacturer operated the facility and started using TCE in its operations. Records show that TCE was used in the Chromatex Plant #2 operations until June or July 1988 [1,2,3]. Operations at the plant ceased on March 2001.

The contaminant plume from the Valmont TCE site presented a complex hydrogeological and human exposure problem for investigators, especially since contaminants were found in deep and shallow ground water and in the indoor air of some of the residents' homes. The site is underlain by fractured pebbly conglomerate and sandstone of the Pennsylvania Pottsville formation. Soil cover is generally thin around the site, and on the topographic high points, outcrops are abundant and readily visible. Ground water table depth is about 18 feet and variably deep aquifer water entry zones depend upon fractures encountered by the well borings [3].

The area of concern covers about one-fifth square mile and includes the Chromatex #2 plant on Jaycee Drive and at least 35 homes about 1000 feet down gradient and to the northeast as shown in Appendix A - Figure 2. For purposes of this PHA, the 65 to 80 affected homes and apartments are considered *off-site*. Previously, the residents all had private wells, but since 1988 the area is served by a public water supply [1].

Remedial and Regulatory History

- In October 1987 a complaint was received by PADEP (called PADER at that time) about an alcohol and/or a xylene spill at another facility located west of the Chromatex Plant #2. During sample analysis of residential wells northeast of Chromatex Plant #2, TCE was detected. The concentrations of the TCE were found as high as 1,400 parts per billion (ppb). EPA initiated further investigation and the TCE levels were confirmed [4]. Immediate action was taken to remove the risk by supplying residents with bottled water. EPA also subsequently provided emergency funding in December 1987 to oversee the installation of public water supply lines into the neighborhood. The public water line installation was completed in February 1988 [4].

- The Chromatex Plant #2 maintained a 10,000-gallon underground storage tank just northwest of the building to contain emergency spillage or overflow of hazardous materials stored in the plant, and to receive floor drain waste. In November 1987, the tank was drained of about 10,000 gallons of TCE-contaminated wastewater with a TCE concentration of 3,500,000 ppb and nine 55-gallon drums of bottom sludge. Pressure testing of the tank at that time revealed no leaks [4]. The tank was disconnected from feeder pipes and ultimately removed from the site in October 1994 [4].

- Following an EPA Administrative Consent Order in March of 1988, Chromatex Incorporated (owner of Chromatex plant #2) installed and sampled 11 monitoring wells at the site to perform an extent of ground water contamination study.

- EPA proposed the site to the National Priority List on June 14, 2001, and it was formally added to the list on September 13, 2001. EPA began coordinating plans for a Remedial Investigation/ Feasibility Study with other agencies, including ATSDR, PADEP, and PADOH, to determine the extent of contamination in ground water, to identify the possible source area(s) for the contamination, and to characterize the local ground water flow regime.

- From April 2004 to August 2004, a soil removal action (mobilization/demobilization period) was conducted for the contaminated on-site soil under the Chromatex Plant #2 parking area and other exterior areas adjacent to the Plant. In general, soil excavations were to a depth of 10 feet with approximately 10 percent of the area to a depth of 3 feet. Approximately 18,000 tons of soil was removed during the 2004 removal action.

- In July 2004, Tetra Tech NUS (TTNUS) prepared the Valmont TCE site Final Remedial Investigation Report for Operable Unit (OU-3). (Tetra Tech NUS is Tetra Tech NUS, Incorporated at 600 Clark Avenue, Suite 3, King of Prussia, Pennsylvania.). Also in July 2004, EPA issued an Action Memo for implementation of a Soil Vapor Extraction (SVE) system to address the soils beneath the plant.

- In November 2004, TTNUS conducted a pilot study to determine the effectiveness of SVE to remediate these soils and establish design parameters. However, results of the pilot study did not convincingly support the use of SVE as a cleanup alternative. As a result, EPA decided to also conduct a field treatability study of in-situ chemical oxidation of the contaminated soils. This study was conducted in February 2005. The results of the in-situ chemical oxidation study for contaminated soils below the building foundation indicated that in-situ chemical oxidation also did not achieve the desired results of reducing contaminant concentrations in the soil. Coincidentally, however, during the treatability study a TCE "hotspot" was encountered in the soils below the building foundation up to two orders of magnitude higher than any previous results. Since neither SVE nor in-situ chemical oxidation appear to be practicable alternatives, EPA decided to perform "limited" excavation within the TCE "hotspot" only to remove contaminated soils with the highest TCE concentrations. EPA contemplates beginning the excavation in the Spring 2006.

- An in-situ chemical oxidation treatability study was also completed for ground water. EPA modified the Scope of Work for the in-situ chemical oxidation treatability study for ground water to include a similar study for contaminated soils beneath the building as discussed above. The injection event for the in-situ chemical oxidation treatability study occurred in February 2005. The results of the in-situ chemical oxidation treatability study did achieve some destruction of TCE in the ground water, but there were some problems with the delivery of the oxidant into the wells and the radius of influence of the oxidant once injected into the ground water. EPA has not made a final determination on the cleanup methodology for ground water. However, in-situ chemical oxidation will be retained as a potential cleanup alternative in addition to a more conventional pump and treat system for ground water. The Final Draft FS is currently under review by EPA and PADEP for the ground water alternatives.

- Indoor air carbon filtration units were given to the eight residents considered at risk by EPA and sump pump covers (some covers were custom built) for homes above the plume with sump pumps and electric accounts were credited monthly for the air filters within the eight homes. In September 2005, EPA decided to install sub slab pressurization units to replace the existing air filters in the eight impacted residences. The installation of these systems began in January 2006.

- In response to citizens' concerns expressed at the public meeting to more thoroughly evaluate indoor air and vapor intrusion, in December 2005 EPA decided to perform sub slab sampling in all residences adjacent to the Valmont Site (excluding the 8 homes that already have air filtration systems). This work began in early 2006 and included sampling for VOCs and radon below the slab and radon only within the breathing space of the basements. In this case, radon was used to help determine if a potential vapor intrusion pathway existed and/or to develop a residence-specific attenuation factor, to be applied to the VOCs.

PADOH and ATSDR Site Visits and Meetings and Other Assessment Activities

As part of the PHA process, ATSDR and PADOH conducted numerous site visits and met with other government officials, physicians and hospital employees, and community residents in 2001 through 2005. The purposes of these visits and meetings were to:

- Identify the residents' site-related community health concerns;
- Discuss the work to be performed by the regulatory agencies (PADEP and EPA), including possible identification of contamination sources both in indoor air and in the ground water and the impacted neighborhoods, and the Remedial Investigation Feasibility Study sampling points;
- Have PADOH become more familiar with the community and the site;
- Explain the results of indoor air samples to the community residents whose homes were tested and to discuss other household sources of indoor air pollutants;
- View actual on-site remediation by the contractor and to update community members about the Agencies' site-related activities;
- Work with EPA and PADEP on discussing and reviewing the Valmont TCE Remedial Investigation /Feasibility Study; and
- Address physicians' concerns about any patients living adjacent to the site
- Most recently, recognize *public comment*/community input for the final version Valmont TCE PHA at the public availability meeting in November 2005.

Description of Visits and Meetings:

- On June 28, 2001, a representative from PADOH visited the site and met with representatives from PADEP, EPA, and the local township. The purpose of this visit was

to: 1) identify the residents' site-related public health concerns; 2) discuss work to be performed by the regulatory agencies, including the identification of possible contamination sources, impacted neighborhoods, and EPA's Remedial Investigation/Feasibility Study sampling points; and 3) become more familiar with the community and the site. In addition to the possible adverse health effects as a result of drinking contaminated water in the past, another community health concern was the potential ongoing exposures from using contaminated residential wells used in watering the yard and washing cars.

- On October 29, 2001, a representative from PADOH visited the site again and observed the PADEP contractor performing a geophysical investigation of a contaminated residential well. In addition, the PADOH and PADEP officials conducted a surficial geological investigation of the area, noting bedrock outcrops and strike and dip of formation bedding in an effort to determine possible preferential ground water flow directions. It is noted that the extent of ground water contamination was still questionable at this time.

- On October 30-31, 2001, another representative from PADOH met with a PADEP official and the PADEP contractors. The PADEP contractors had performed the additional soil gas sampling**,** residential indoor air sampling, and residential and monitoring well water sampling, as well as the geophysical investigation of monitoring wells. It was observed that the area around the old Chromatex Plant #2 is easily accessible to trespassers and that there are currently no workers on site. The nearest homes were observed to be about 1000 feet from the site and the eight residential wells were no longer used for drinking.

- On February 11-13, 2002, representatives from PADOH, ATSDR, EPA, and PADEP visited eight homes as requested by PADEP and as part of community outreach activities performed in support of the PADEP's request to primarily explain the results of the indoor/basement air samples to the community residents. Through these visits, PADOH and ATSDR were also able to discuss household sources of indoor air pollutants, identify community health concerns, and update community members on the agencies' site-related activities.

- A PADOH site visit and meeting with EPA and PADEP was conducted on May 15, 2002 to discuss current community concerns and future sampling plans, and view the early stage of site evaluation and undertaking of EPA's Remedial Investigation Feasibility Study.

- On July 18, 2002, PADOH and ATSDR attended an EPA sponsored public availability meeting and public availability session. At this time, PADOH and ATSDR collected community health concerns for this public health assessment and responded to the health questions of residents living near the site. Residents had questions about their indoor air quality and how past exposure to the site could have impacted their current health.

- In November of 2002, PADOH held a Public Availability Meeting. PADOH representatives again visited the site and talked to community residents to gather additional community health concerns.

- On July 01, 2003, two representatives from PADOH visited the site again and observed the PADEP contractors perform monitoring of an on-site well. In addition, the PADOH and PADEP officials conducted a tour of the Chromatex #2 plant building and discussed EPA's plans for installing a monitoring well inside the building. It is noted that the extent of ground water contamination was still undefined at this time.

- On February 19, 2004, an EPA public meeting was held for the community in West Hazleton, Pennsylvania. Representatives from PADOH (Health Assessors and Health Educators), ATSDR, and PADEP attended and spoke at the meeting as requested by EPA. The purpose of the public meeting was to explain the results of on-site and off-site sampling and remediation plans being considered by EPA at that time. At that time, the community was requested by EPA not to continue using contaminated ground water for yard watering and car washing because of neighbors' concerns, even though EPA had determined the health risk was very low [5,6]. The State Medical Toxicologist (Dr. Keith Burkhart), offered to be available to any resident's physician for future or current health concerns.

- In March and April 2004, a PADOH representative participated in a teleconference and in a meeting in Philadelphia to review and comment on the health-related sections of the draft EPA Remedial Investigation Report [5].

- On June 16, 2004, an informational meeting sponsored by ATSDR and PADOH was held for physicians at the Hazleton General Hospital. This was held during their regular physicians' meeting. Dr. Keith Burkhart, MD (Medical Toxicologist), Barbara Allerton, and Pauline Risser-Clemens from the PADOH attended a General Medical Staff meeting at the Hazleton General Hospital. Lora Werner from ATSDR and John Mellow from PADEP also attended. Dr. Keith Burkhart and Lora Werner presented information to the physicians relating to the chemical contamination at the Valmont TCE (Valmont Trichloroethylene) site and especially concerning possible exposures of potential patients to TCE and 1,1,1-trichloroethane. Dr. Burkhart reiterated his offer of availability to discuss any health problems that might be associated with the Valmont TCE site with the affected resident's physicians.

- On August 8, 2005, PADOH and ATSDR held a Public Availability Meeting specifically for the public comment period/version of the Valmont TCE PHA. ATSDR's Region 3 Medical Toxicologist also represented health information to the community members.

- On November 3, 2005, EPA held a public meeting for the community. Representatives from ATSDR and PADEP participated in this meeting. ATSDR spoke about the public comment version PHA, community health concerns, recommendations, and conclusions. ATSDR also discussed the citizens group VRAP's health survey with the residents.

Previous PADOH and ATSDR Health Consultations and the Initial Version of the PHA

As part of the initial health assessment of the site, PADOH and ATSDR published two health consultations (HCs) on the Valmont TCE site (*Public Health Evaluation of Residential Indoor Air* and *Public Health Evaluation of Soil Samples*) and the initial version of this PHA in June 2002. The two HCs were:

1. In the November 2002 HC, PADOH evaluated residential indoor air data and determined whether the residents were being exposed to harmful levels of volatile organic compounds (VOCs) in the indoor air of their homes. VOCs include a variety of chemicals that volatilize easily into the air. Although the chemical plume in the ground water cannot be ruled out as a possible source, these VOCs can come from a number of sources and are often emitted from products commonly used in the home, office, and school, and in arts, crafts and hobby activities and products as shown in Appendix B - Tables 1 and 2 [5,7, 8, 9,10]. When TCE is found in ground water, harmful breakdown products of TCE (including dichloroethenes, dichloroethanes, and vinyl chloride) can result from the action of naturally occurring bacteria.

2. In the April 2003 HC, PADOH evaluated off-site soil data and determined whether the residents were being exposed to any chemicals detected in the soil around their homes. PADOH considered how occupants came into contact with the chemicals, as well as the frequency of exposure to the chemicals. PADOH also considered whether the contaminants were present at harmful levels [11].

Environmental Sampling History

Since 1987 EPA and PADEP sampled numerous on-site monitoring wells and off-site residential wells. Surface soils and subsurface soils were sampled on-site and off-site. More recently, indoor air samples were taken inside residents' homes. Numerous chemicals, including volatile organic compounds (VOCs), semi-volatile organic compounds, pesticides/polychlorinated biphenyls, and inorganic chemicals, were detected at various levels *on-site* and *off-site* through this sampling from 1988 through 2004. Not all chemicals detected were at concentrations of potential health concern (see Appendix C for the ATSDR screening process).

Off-Site Residential Wells Data

In 1987, 29 residential wells were sampled along Deer Run Road, Bent Pine Road, and Twin Oaks Road in the development immediately northeast of the Chromatex Plant (Appendix A - Figure 2). The levels of VOC contamination – especially TCE and 1,1,1-trichloroethane – found in residential wells at that time in *1987* are summarized in the ***TABLE OF ORIGINAL WELL DATA*** (on the following page). Within four months, EPA funded the installation of public water supply connections, and by February 1988, all affected homes were connected to public water [3,5]. Maximum sample results for 1987 to 2002 are shown in Appendix B - Tables 3 and 4 [4, 8]. In 1988, monitoring wells showed a high contamination by VOCs on-site (Appendix B - Table 5) [8].

TABLE OF ORIGINAL WELL DATA - October 1987, VOC Detection Results Off-site and Listed by Street Name (The samples were taken before Public Water was installed).
All units are in parts per billion (ppb).

Location by Street	*TCE*	*1,1,1-trichloroethane*	*1,1-dichloroethane*	*1,1-dichloroethene*	*cis -1,1-dichloroethene*
Bent Pine Road	100.0	18.0	-	-	-
	300.0	40.0	-	2.0	2.0
	1,000.0	130.0	0.7	2.0	-
	1,000.0	170.0	2.0	2.0	-
	200.0	50.0	<1.0	<1.0	2.0
	800.0	450.0	2.0	2.4	10.0
	800.0	450.0	2.0	2.4	10.0
	1,400.0	430.0	3.4	5.4	30.0
	1,200.0	300.0	2.6	3.0	-
	550.0	100.0	-	1.8	8.0
	220.0	40.0	-	-	-
	500.0	54.0	-	< 1.0	< 0.3
	550.0	40.0	-	-	2.7
Deer Run Road	1.0	-	-	-	-
	1.0	5.0	-	-	-
	150.0	25.0	-	-	< 1.0
	18.0	8.0	-	-	-
	16.0	7.0	-	-	-
	18.0	7.0	-	-	-
	21.0	3.5	-	-	-
	15.0	6.6	-	-	-
	22.0	5.6	-	-	-
	12.0	5.0	-	-	-
Route 93	4.6	2.3	-	-	-
Twin Oaks Road	48.0	6.7	-	-	-

Off-Site Soil Data

In May 1987, samples were taken from five residential yards; background samples were also taken at that time. These samples were analyzed for VOCs and all were non-detects [11]. Off-site sampling continued periodically into 2004. In 2004, EPA completed a home-by-home evaluation based on the sample results [5].

Indoor Air Data

In May 2001, the EPA contractor collected indoor air samples from 22 homes in an attempt to generally cover the residential area known to be underlain by the plume. The sampling included homes of people who responded to EPA's request for sampling or who volunteered for their homes to be sampled. In October through November 2001, PADEP collected basement air samples from eight homes primarily based on the May 2001 findings and because of concerns that the underground plume of TCE and other VOCs could possibly threaten the health of those residents [8]. Sample results dated up to and including February 2002, that levels are greater than the health based ATSDR screening levels for each specific chemical, are listed in Appendix B - Table 6 [7,12]. TCE was detected but levels were below the ATSDR screening levels [7].

EPA and PADEP completed more indoor air sampling through November 2003. Overall four rounds of sampling were done and a total of 89 indoor air samples were collected [5]. EPA identified homes that were considered to have vapor intrusion from the underground chemical plume and those are shown in Appendix B - Table 7 of this document. Some homes had openings in their basement concrete floors for purposes such as for sump pumps. In those affected homes, sampling was conducted directly at these openings with a mobile testing unit in addition to the stationary summa canisters used for previous sampling [5].

On-Site Monitoring Wells

In 1988, a maximum TCE concentration of 17,000 ug/L was detected on-site strongly suggesting that residual pools or pockets of liquid product may have been present in the subsurface. EPA and PADEP sampled more monitoring wells from 1993 into 2003 as shown in Appendix B - Tables 4 and 5 [4, 8]. In December 2000, the contaminants were similar to previous sampling in chemistry and concentration, except for the detection of vinyl chloride (a known human carcinogen) in one well, indicating that additional breakdown products of TCE could have been present in the ground water plume [4, 8]. Later sampling did not show vinyl chloride. Two other industrial sites - Polyclean dry cleaners in the shopping center northeast of the site and an ink company on Unico Drive Site northwest of the site - were identified by PADEP as possibly contributing to the contamination in the area of the Valmont TCE site. (Currently, PADEP is continuing to conduct further evaluation/investigation of the Polyclean site.)

On-Site Soil Data

A summary and summary table of past on-site soil sampling data is presented in the Valmont TCE Health Consultation, Evaluation of Soil Samples published April 30, 2003 [11]. On May 5, 1988, seven soil samples were obtained by an EPA subcontractor and analyzed for VOCs.

Contaminants, especially TCE, were consistently detected at the maximum concentrations in the soil below the plant's parking lot, but VOCs were detected at other locations on-site. In September 1993, surface soil samples and subsurface soil samples were collected. More on-site sampling occurred through 2004 and VOCs were detected in many of these later soil samples [5].

Other Data

The EPA Toxics Release Inventory (TRI) reporting from the Chromatex Plant #2 estimated that a total of 7640 pounds of TCE was released to the air in 1987 in and around the plant during normal manufacturing processes that year [2]. Of this amount, 7390 pounds of TCE were fugitive chemical releases (or releases from no specific point) to the air in and around the plant. The amount estimated by Chromatex to be released through the plant's stack was 250 pounds per year. Chromatex reported that an additional amount of TCE per year was released in water to the publicly owned sewage treatment plant for treatment before that water was released to surface waters. These release amounts are shown in the **TABLE OF TRI DATA (**on the following page) and are from the EPA's 1987-1996 records for TRI [2]. The reported TCE TRI data for Chromatex in 1987 was reviewed by PADOH and ATSDR (1987 was the only year reported by the facility) [2]. TRI quantities are self-reported. Though these releases were "allowed" in 1987, the significance is past residential exposures in addition to the contaminated ground water exposures.

Quality Assurance and Quality Control

In preparing this public health assessment (PHA), ATSDR and PADOH relied on the information provided in the referenced documents. We assumed adequate quality assurance and quality control measures were followed regarding data gathering, chain-of-custody, laboratory procedures, and data reporting. We expect that to ensure high quality data, extreme care was taken during all aspects of sample collection. We expect that the laboratory only used certified, clean-sample collection devices. Once samples were collected, we expect they were stored according to the method protocol and were delivered to the analytical laboratory as soon as possible. Finally, we expect that laboratory Standard Operating Procedures and other procedures and guidance for sample analysis, reporting, and chains of custody were followed.

Discussion

Pathway Analysis

In this section, ATSDR and PADOH evaluated whether the community has been, is, or could be exposed to harmful levels of contaminants in the environment. ATSDR and PADOH considered how individuals might come into contact with contaminated media, as well as the duration and frequency of any exposures.

To determine whether people have been exposed to contaminants migrating from the site, the PADOH evaluates the environmental and human components that lead to human exposure. The PADOH and the ATSDR identify exposure pathways as completed, potential, or eliminated

TABLE OF TRI DATA - Toxics Release Inventory (TRI) Records for Chromatex Plant #2, Reported Releases of TCE at Chromatex Plant #2 (Source: EPA 1987-1996 Toxics Release Inventory, Landview III, Light Edition CD-ROM Quick Reference Guide)

FACILITY ID INFORMATION	*CHEMICAL & PROCESS ID*
EPA Submission ID (DCN): 1387010250785PA TRI Facility ID: 18201CHRMTJAYCE Reporting Year: 1987 EPA Region: 3 Amount of Facility Covered: A--AN ENTIRE COVERED FACILITY Type of Facility: COMMERCIAL Facility Name: CHROMATEX INC. PLANT # 2 Facility Street: JAYCEE DR. VALMONT INDUSTRIAL PARK Facility City: WEST HAZLETON Facility County: LUZERNE Facility State: PA Facility Zip Code: 182011194 Public Contact: STEVE ENGELMYER Public Contact Phone: 215-851-8419 Reported Latitude (deg/min/sec): 0405719 Reported Longitude (deg/min/sec): 0760000 Underground Injection Code ID 1: NA Parent Company Name: ROSSVILLE IND. INC. Parent Company D&B Number: NA Submission Type: FORM R Preferred Latitude (decimal degrees): 40.955278 Preferred Longitude (decimal degrees): 76.000000	CAS Registry Number: 79-01-6 Chemical Name: TRICHLOROETHYLENE Mixture/Component Name: NA Chemical is Trade Secret: N Maximum Amount Onsite (lbs): 10,000 TO 99,999 Carcinogen: Y Pesticide: N Developmental Toxin: N Processing Use: AS AN ARTICLE COMPONENT State and County FIPS Code: 42079 Facility D&B Number 1: NA NPDES Permit Number 1: NA Facility's RCRA ID 1: PAD000779942 Primary SIC Code: 2262 Primary SIC Name: FINISHING PLANTS, MAN-MADE SIC Code 2: 2258 SIC Name 2: LACE AND WARP KNIT FABRIC MILLS

TABLE OF TRI DATA - TRI Records for Chromatex Plant #2, Chromatex's Reported Releases of TCE (Source: EPA 1987-1996 Toxics Release Inventory, Landview III, Light Edition CD-ROM Quick Reference Guide) – continued.

RELEASES/TRANSFERS

Air Releases Submission Total (lbs): 7640
Water Releases Total (lbs): 0
Land Releases Total (lbs): 0
All Releases Submission Total (lbs): 7640
POTW Transfers Submission Total (lbs): 750
Offsite Transfer Total (lbs): 0
POTW and Transfers Total (lbs): 750
Releases and Transfers Total (lbs): 8390

INDIVIDUAL RELEASES AND TRANSFERS

Storm Water Percent: 0000.00
POTW/Offsite State: PA

Release/Transfer Type: 1--NON-POINT AIR RELEASE
Fugitive/non-point Air Release (lbs): 7390
Stack/point Air Release (lbs): 250
POTW Transfer (lbs): 750
POTW/Offsite Name: GREATER HAZLETON JOINT SEWAR AUTHORITY
POTW/Offsite City: WEST HAZLETON
POTW/Offsite County: LUZERNE
POTW/Offsite Zip Code: 182011194
Range Estimate: ESTIMATE
Basis for Estimate: MASS BALANCE CALCULATIONS

Release/Transfer Type: 2--POINT AIR RELEASE
Storm Water Percent: 0000.00
Range Estimate: MIDPOINT OF RANGE
Basis for Estimate: PUBLISHED EMISSION FACTORS

Release/Transfer Type: 6--POTW TRANSFER
Storm Water Percent: 0000.00
Range Estimate: MIDPOINT OF RANGE

Basis for Estimate: OTHER APPROACHES

POLLUTION PREVENTION AND WASTE

Onsite Treatment Efficiency: 0095.00
Based on Operating Data: NO

Onsite Treatment Method 1:
SOLVENTS/ORGANICS RECOVERY -- OTHER
Influent Concentrate: 100 PARTS PER MILLION (0.01 PERCENT) TO 1 PERCENT (10,000 PARTS PER MILLION)
Onsite Waste Stream Type: GASEOUS (INCLUDING GASES, VAPORS, AIRBORNE PARTICULATES)

as shown in Table 3 - *Summary of Site Related Exposure Pathway Elements* (on the following page). In completed exposure pathways, the five elements exist, and so exposure has occurred, is occurring, or will occur. The five elements are: (a) a source of contamination; (b) a fate or way of transport; (c) an environmental medium in which the contaminants may be present or may migrate; (d) a human exposure point (such as by drinking water, having skin contact, or by inhalation); and (e) a receptor population. In potential exposure pathways, however, at least one of the five elements is or may have been present, and exposure to a contaminant might have occurred in the past, or may possibly occur in the future. An exposure pathway may be eliminated if at least one of the five elements is missing and never will be present.

Off-site *Completed* Exposure Pathways Associated with Contaminated Ground Water

Past exposure pathways associated with ground water are described in the ***TABLE OF PATHWAYS*** **(**on the following page) and occurred through the use of contaminated ground water in residential wells. Contaminants from drinking water were ingested by some residents, possibly absorbed through direct skin contact, and especially for VOCs, inhaled during bathing, cooking or other water uses. Assuming that residential wells became contaminated after 1978, residents in 65 to 80 homes and apartments could have been exposed from ingesting the ground water for up to ten years. **Current** or **future** exposure pathways via ground water have been greatly reduced or eliminated since the public water supply was brought on-line in 1988, though vapor intrusion is still a pathway of exposure by inhalation in some of the homes [5]. Vapor intrusion is the migration of volatile chemical vapors from the subsurface (at this site from the contaminated ground water of the underlying aquifers) into overlying buildings (in this case, the residential homes) [13].

Off-site *Potential* Exposure Pathways Associated with Ambient Air

Past exposures to residents may have occurred through inhalation of chemicals, especially TCE, during manufacturing at the Chromatex plant. It is fairly certain that some TCE was released to the outdoors as "fugitive" or non-point releases and also some up the stack of the Chromatex plant during manufacturing processes [2]. It is unknown how much TCE may have traveled toward the affected residences, but the winds in the area generally prevail from the Southwest (generally speaking, Pennsylvania is in the Southwest trade winds and wind travels to the Northeast in the direction of these homes). Other than the EPA TRI data for Chromatex Plant #2, little information about possible past exposures from ambient air is available. Even with the TRI data for 1987, it would be difficult to determine the ambient air TCE concentrations at which the residents might have been exposed during that year. The use of TCE at the plant was discontinued in 1988.

Off-site *Potential* Exposure Pathways Associated with Soil

Chemicals spilled to the on-site soil may have been carried by surface water to soil in residential areas. In addition, there may be other unknown and uncertain sources of chemicals in the soil of the residential areas. During analysis of soil samples off-site, EPA and PADEP detected some contaminants [5]. **Past** and **current** exposures may have occurred to children and adults through exposures to contaminated soil through skin contact, accidental ingestion, and inhalation of contaminated soil particles. For chemical contaminations such as VOCs, exposures would

TABLE OF PATHWAYS – Summary of Site Related Exposure Pathway Elements

Site Related Exposure Pathway Elements					*Pathway Status and Time Frame*
Source	***Environmental Media***	***Point of Exposure***	***Route of Exposure***	***Exposed Population***	
VOCs (especially TCE) in ground water under the site	On-site Ground Water	Using and drinking the contaminated ground water	Inhalation (assumed), Ingestion (assumed), Dermal contact (assumed)	On-site workers, Visitors	**Completed** Past (actual ingestion and water use is unknown)
Chemicals (especially TCE) in ground water under the site and under the affected residents' homes	Off-site Ground Water	Using and drinking the contaminated ground water; indoor air vapor intrusion	Ingestion (drinking water and cooking); Dermal contact (bathing); and Inhalation (bathing, cooking, and other water uses)	Nearby Residents	**Completed** Past, Current (After 1988, only inhalation through outdoor uses of private wells and via vapor intrusion to indoor air remained)
Fugitive chemicals (especially TCE)[1]	On-site Ambient Air	Ambient Air	Inhalation	On-site workers, Visitors, Trespassers	**Completed**[2] Past
Fugitive chemicals (especially TCE) plus TCE out the plant stack[1]	Off-site Ambient Air	Ambient Air	Inhalation	Nearby residents, Pedestrians	**Potential** Past
Chemicals (especially TCE and other VOCs) spilled to soil	On-site Surface Soil	Soil on-site	(Assumed) Ingestion, Dermal contact, Inhalation	On-site workers, trespassers	**Potential**[2] Past
Chemicals (especially TCE and other VOCs) spilled to soil and carried by surface water runoff; Uncertain sources	Off-site Surface Soil	Soil off-site	Ingestion, Dermal contact, Inhalation (possible)	Nearby Residents	**Potential** Past, Current Future

1 - Based on EPA Toxics Release Inventory records.
2 –(Refers to before 2001) EPA completed an assessment prior to remediation on-site by workers in 2003; this assessment may be found in the RI Section 6 [5].

primarily have been through inhalation of soil gas. Future exposures to children and adults could also take place in the residential areas where contamination was detected, but concentrations of VOCs are so low that health effects are unlikely.

On-site *Completed* Exposure Pathways Associated with Contaminated Ground Water

Little information is available regarding the past pathways of exposure for the workers, though inhalation and ingestion are assumed at the Chromatex Plant #2. Since we can only theorize about the possible impact of exposure to workers, this document should not be construed as a complete assessment of worker exposures in the past. **Past** exposures to workers may have occurred through the use of contaminated ground water for drinking water, washing purposes, and any other use of ground water in the manufacturing processes in the Chromatex Plant #2. The plant was connected to public water in 1988.

On-site *Completed* Exposure Pathways Associated with Ambient Air
Past exposures to workers (before 2001) may have occurred by inhalation of chemicals, especially TCE, through the use of chemicals during manufacturing processes. Other than the EPA TRI data, little information about possible **past** exposures is available [2].

On-site *Potential* Exposure Pathways Associated with Soil

Past exposures may have occurred through exposures to soil at the Chromatex Plant #2. Especially for VOCs, exposures would have been primarily through inhalation or accidental ingestion of contaminated soil particles and through inhalation of the VOC itself, though dermal contact may have been a route of exposure. All is assumed and no information exists to determine the extent of exposures from contaminated soils.

Toxicological and Data Evaluation

PADOH and ATSDR Toxicological Evaluation Process

The primary public health issues that need to be evaluated are the past on-site and off-site exposures to chemicals, especially TCE, through the contaminated ground water, residential indoor air vapor intrusion from the plume beneath the homes, surface water runoff, fugitive and stack releases in outdoor air coming from the direction of the Chromatex Plant #2, and soil. The ATSDR has developed health-based comparison values (CVs) that are chemical-specific concentrations, which help to determine which environmental contaminants are of possible health concern and need further evaluation [12]. If a chemical concentration is found in the environment at levels below the CV, it is not likely to cause an adverse health effects, though chemicals that exceed CVs do not necessarily produce adverse health effects. If a contaminant exceeds its corresponding CV, PADOH examines the other health-based guideline values of the contaminant. For a more detailed explanation of this process, please refer to Appendix C.

Some Assumptions and Scenarios Used in the Evaluation Process

ATSDR and PADOH considered various exposure scenarios in this evaluation: 1) past exposure of community residents to off-site ground water though drinking water (ingestion, skin contact,

and inhalation), bathing, cooking, and other water uses, and through indoor air vapor intrusion (the worst case exposure period of 10 years was assumed for direct exposure to the contaminated ground water); and 2) past exposure of workers on-site through the ground water (the worst case exposure to the ground water of 10 years was assumed) and to on-site soil samples (the worst case exposure of 23 years was assumed). These assumed exposure durations are very conservative since it is expected that exposures were most likely for a shorter duration.

On-site, there were too many unknown variables to determine actual past inhalation and dermal contact to TCE during the normal manufacturing process though some estimates for releases of fugitive TCE exist at 7390 pounds of TCE for 1987 in the TRI data as shown in Table 2 - TRI Records for Chromatex Plant #2. Assuming the workers worked 250 days per year, it could be assumed that about 30 pounds (2.5 gallons) of TCE were released in and around the plant on the average per day. Off-site, it could be assumed that some of this TCE was carried by wind into the residential areas in unknown amounts for additive inhalation exposures during the 10 years that TCE was used in the manufacturing operations. EPA soil gas sampling in the residential areas may indicate that this - or possibly another source of air contaminants together – may have occurred in the past as discussed in the RI Section 4 *Summary and Conclusions* [5]. The significance is that there may have been additional, but not quantifiable exposures by this route.

Assumptions Used to Evaluate Off-site Exposures to Contaminated Ground Water and Off-Site Exposures to Contaminated Soil

For residential wells, PADOH discusses health effects that could result from site-related contaminant exposures assuming they occurred for 10 years between 1978 and 1988, when the public water supply became available to the community residents. Moreover, PADOH estimated exposures from three routes of exposure (inhalation, ingestion, dermal). In evaluating VOCs, we assumed that the ingestion (drinking water) and inhalation pathways (bathing, cooking, vapor intrusion, fugitive TCE from the Chromatex plant blowing into the residential areas) are the pathways of greatest exposure with very small contributions from other pathways such as a dermal dose during bathing. Vapor intrusion was assumed to be from the plume beneath the home, especially in homes with openings in the flooring for sump pumps and other routes for vapor intrusion infiltrations, such as cracks and plumbing. Vapor intrusion was actually found by EPA in some homes with sump pumps and around the openings for the sump pumps.

The assumption is that adults drink 2 liters of water and that children drink 1 liter of water where the highest levels of the contaminants detected were consumed daily [12]. Body weights used in the drinking water exposure assumptions were 10 kg (for children) and 70 kg (for adults) [12]. For vapor intrusion and indoor air calculations, residents were assumed to be exposed to contaminants for 24 hours per day for 350 days per year. Children were assumed to inhale 12 cubic meters of the indoor air per day (m^3/day) and be 0 to 7 years of age [5]. Adults were assumed to inhale 20 m^3/day and to weigh 70 kilograms [5].

For final estimates using indoor air measures taken in the basements, using an assumed 24-hour exposure, in most cases, yields a very conservative and “biased” estimate of risk as stated in the RI Section 6 [5]. This estimate was used as the upper bound exposure for households and where *unacceptable risk* was found more exact exposure times and frequencies were used to determine refined exposures based on the uses of the basement by that particular household [5]. EPA

defines the carcinogenic potential risk in excess of 1 in 10,000 and hazard indices in excess of 1 as unacceptable or an unacceptable risk. A *Public Health Hazard* is defined by ATSDR as an evaluation of available relevant information and 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. In site-specific exposure evaluations, PADOH and ATSDR may consider other criteria such as the determination of an unacceptable risk or another agency or scientific-based value to determine whether there is a public health hazard.

In evaluating the off-site soil exposure, PADOH considered the worst-case scenarios for residents through accidental ingestion of soil for total exposure duration of 10 years at the maximum levels of the contaminants detected. Dermal exposure and inhalation of fugitive dust particles are not likely to contribute significantly to the hazard represented by ingestion of soil. To evaluate for the health effects, PADOH assumed that the child's age was up to 7 years and that an adult weighed 70 kg. Furthermore, it was assumed that the soil ingestion rate for children is 200 mg/day and for adults is 100 mg/day.

Assumptions Used to Evaluate On-site Exposures to Contaminated Ground Water, On-site Exposures to Fugitive TCE in Ambient Air, and On-site Exposures to Contaminated Soil

For on-site wells, PADOH assumed that an employee worked for about 8 hours a day for 5 days a week and drank 1 liter per day of water at the maximum levels of contaminants. PADOH assumed that, for ground water exposures, ingestion of drinking water was one of the major pathways of exposure to TCE. Additionally, because TCE is very volatile, inhalation is also a major pathway even from drinking contaminated water. PADOH assumed that site-related contaminant exposures might have occurred for 23 years (1978 – 2001).

It is important to note that we could not accurately assess workers' exposures that may have occurred from the on-site manufacturing processes, though inhalation was most likely a major pathway of exposure, with dermal contact with contaminated products, during the manufacturing processes most likely the third pathway of exposure. Logically, TCE inhalation exposures were cumulative from the manufacturing processes and from drinking and other uses of the contaminated ground water.

In evaluating the on-site soil exposure, PADOH made assumptions for the workers as described in the *Public Health Evaluation of Soil Samples* Health Consultation for the Valmont TCE site, published April 2003. It was concluded that past exposures to on-site soil contaminants posed no apparent public health hazard to workers, residents, and trespassers (children and adults) [11]. PADOH only considered children as having been on-site very temporarily (visiting or trespassing), since this was an occupational setting.

General Contaminant Evaluation

PADOH and ATSDR identified chemicals of *possible* concern in one or more media (water, air, and soil) at this site by comparing levels to the ATSDR Comparison Values [12]. If no ATSDR CVs were available, then comparison was completed using other agency standards and/or by researching related medical literature. Most of the VOCs found in the indoor air of homes were

not associated with the Valmont TCE site chemical plume (see Appendix B – Tables 1 and 2) [5]. The semi-volatile organic chemicals and inorganic chemicals detected in one or more media were also evaluated for this PHA.

For the purposes of this PHA, ***on-site*** refers to areas within the Valmont TCE Site (specifically around the Chromatex Plant # 2) property boundary and ***off-site*** refers to homes, residential wells and monitoring wells not on the industrial property.

Specific On-site and Off-site Contaminant Evaluation

Evaluation of Data from Off-Site Wells

All residences were connected to the public water supply so exposure to and risk of health effects from ground water have greatly diminished. Additional residential well samples were collected from 1993-2003. Analysis of ground water up to 2001 showed that off-site the detected chemicals of *potential* concern included carbon tetrachloride; 1,1-dichloroethane; 1,1-dichloroethene; 1,1,1-trichloroethane; TCE; and vinyl chloride [5]. The *past* maximum concentrations detected of the selected chemicals of *potential* concern in the years up to and including 2002 for residential wells are summarized in Appendix B - Table 3. Appendix B - Table 4 contains the more recent 2003 ground water sample data for both on-site and off-site and notes whether the sample was taken from a shallow or deep well. In 2003, the detected chemicals of *potential* concern *off-site* included 1,1-dichloroethane; cis-1, 2-dichloroethene; 1,4-dioxane; and TCE [8].

EPA determined that water use for other purposes such as yard watering and car washing had very little associated risk according to the EPA RI Report, Section 6 [5]. ATSDR and PADOH agree with this conclusion. Some residential wells had been continuously used for yard work, car washing and landscape irrigation until 2004, even after the public water had been installed. In 2003, EPA completed an assessment and determined this to be "at least a 100 times below a level of concern" which may be found in the EPA RI Report Section 6.6.4.4 and EPA RI Appendix G-4 [5]. Even though the water was not determined to be a health risk, EPA Region 3 asked the residents to discontinue using water from their wells for any purpose at the February 2004 EPA public meeting in West Hazleton, Pennsylvania due to community perceptions.

TCE

Off-site, children and adults may have been exposed to TCE in the residential well water at the ***maximum*** *concentration of 1,400 ppb* as shown in Appendix B – Table 3. For residents who may have been exposed to TCE for 10 years at the maximum level of 1,400 ppb, ATSDR and PADOH evaluated the potential for noncancerous health effects at the levels found during this investigation. Assuming *maximum* exposures to the ground water via ingestion, inhalation and skin contact, the estimated total past *maximum* daily dose for children and adults would have been 0.42 and 0.12 mg/kg/day, respectively. The estimated doses are about 400 to 1000 times greater than EPA's reference dose or RfD (this RfD of 0.0003 mg/kg/day is currently provisional under EPA review) and the estimated doses are about two (2) to three (3) orders of magnitude - meaning 10 to 100 times - less than the no observable adverse effect levels (NOAEL) in animal studies of 250 mg/kg/day [9,12]. Some recent reviews show that a lower NOAEL or LOAEL

such as a lowest observed adverse effect level (LOAEL) at 50 mg/kg day might be considered, therefore, the estimated dose ***for a child*** might only be one (1) to two (2) orders of magnitude less than the LOAEL. *It is possible that there could have been non-cancerous health effects related to these exposures at the* ***highest*** *levels of TCE in the ground water.*

In order to evaluate the possible cancer risks to residents who may have ingested contaminated ground water at the maximum TCE concentration (1,400 ppb), the TCE cancer slope factor (CSF) of 0.02 to 0.4 $(mg/kg/day)^{-1}$ was used (note – this cancer risk association is currently under review by EPA). Assuming residents were exposed to the ground water as described above, the expected cancer risk range would be *between* ***six (6) excess cancers per 1,000 people and three (3) per 10,000 people***. This is an extremely conservative estimate since it takes into account the worst-case scenario including exposures by ingestion, inhalation, and skin contact and extrapolates a cancer risk meant for a person's lifetime evaluation [12]. In 2003, the maximum concentration of TCE off-site was 510 ppb, but since the population was already using public water for drinking water, there would have been a significant reduction in cancer risk. For community residents, assuming that they were exposed for *ten years* to contaminated ground water and at the ***highest*** *levels detected in 1988, it is possible that these exposures will contribute to carcinogenic health effects over their lifetime.*

1,1,1-Trichloroethane and 1,1-Dichloroethane (1,1-DCA)

Also found in the wells was 1,1,1-trichloroethane at a maximum concentration of 450 ppb as shown in the Appendix B - Table 3. Based on the assumptions previously discussed, the estimated exposure dose by ingestion would be about 0.013 mg/kg/day for adults and about 0.045 mg/kg/day for children. Even including additional exposures via routes of inhalation and skin, this estimated oral exposure dose is about four (4) to five (5) orders of magnitude less than the level at which no observed adverse health effects were observed in animals [16]. Assuming *maximum* exposures to the ground water via ingestion, inhalation and skin contact, the estimated total past *maximum* daily dose of 1,1,1-trichlorethane for children and adults would have been well below levels of health concern.

Children and adults may have been exposed to 1,1-DCA in the residential well water at the maximum concentration of 3.4 ppb (Appendix B - Table 3). Based on the assumptions previously discussed, the estimated exposure dose by ingestion would be about 0.0001 mg/kg/day for adults and about 0.00034 mg/kg/day for children. ATSDR does not have a Minimal Risk Level (MRL) for 1,1-DCA, nor does EPA have **a** RfD [16]. The estimated oral exposure doses are about six orders of magnitude less than the level at which no observed adverse health effects were observed in animals [19]. Assuming *maximum* exposures to the ground water via ingestion, inhalation and skin contact, the estimated total past *maximum* daily dose of 1,1-DCA for children and adults would have been well below levels of health concern.

Therefore, it is *very unlikely that noncancerous health effects* would have occurred in people exposed to 1,1,1-trichloroethane or 1,1-DCA in the contaminated ground water offsite. In 2003, the 1,1,1-trichloroethane and 1,1-DCA were even lower and well below the ATSDR CV and health effect levels, even if people were still to ingest the water [12].

The International Agency for Research on Cancer (IARC) has determined that 1,1,1-trichloroethane is not classifiable as to its carcinogenicity in humans. The EPA has also determined that 1,1,1-trichloroethane is not classifiable as to its human carcinogenicity [12,14,16,17].

Carbon Tetrachloride

The residents may have been exposed to carbon tetrachloride in the drinking water at the maximum concentration of 9 ppb (see Appendix B – Table 3). Based on the assumptions previously discussed, the estimated oral exposure dose for children would be 0.00026 mg/kg/day and adults would be 0.0009 mg/kg/day - slightly more than EPA's chronic oral RfD of 0.0007 mg/kg/day [12]. Even including additional exposures via routes of inhalation and skin, the estimated oral exposure dose is about three (3) orders of magnitude less than the level at which no observed adverse health effects were observed in animals [24]. Carbon tetrachloride was not confirmed at this level nor was it consistently detected off-site and was not detected above the ATSDR CV in 2003. It is *unlikely that noncancerous health effects* would have occurred to residents due to ingesting the carbon tetrachloride in the contaminated ground water.
EPA has established a CSF of 0.13 $(mg/kg/day)^{-1}$ for carbon tetrachloride [12]. PADOH used the CSF for carbon tetrachloride to evaluate an increased cancer risk. Based on the theoretical cancer risk estimation for past exposures for 10 years and the assumptions enumerated previously, the predicted cancer occurrence would be about *one (1) additional cancers per one million people*. It is our opinion that past exposure to carbon tetrachloride into the ground water *would have been insignificant* in regard to an increased cancer risks.

Evaluation of Data from Off-Site Soil Samples

In May 1987, samples were taken from five residential yards; background samples were also taken at that time. These samples were non-detects for VOCs [11]. In June and August 2002, 53 surface soil samples (including background samples) were collected as described in the EPA RI Report, Sections 2 and 4 [5]. In October 2002 and May 2003, 30 more surface soil samples were collected including samples from 0 to 3 inches in depth at key residences. No organic chemicals were found above levels of health concern. Several inorganic chemicals were detected, but none were above the ATSDR CV [11].

In Fall 2003, EPA completed a home-by-home evaluation on soil samples that contained concentrations of chemicals of *potential* concern. This evaluation is discussed under *Review of Residential Soil Samples for Valmont TCE Site* Section 6 and also the EPA RI Appendix G-2 [5]. No TCE was found in any off-site soil samples [5]. PCE was found in three places - one close to the former dry cleaning dump - but all sample results were below health levels [5]. Chemicals of *potential* concern included: aluminum; arsenic; benzo[a]pyrene; dibenz[a,h]anthracene; iron; and manganese, but only manganese and aluminum were determined to pose any risk according to the EPA RI Section 6 [5]. Excess cancer risks associated with any carcinogens found in the soil samples were within EPA's acceptable ranges [5]. It appears that the inorganic chemicals are at background levels (especially, manganese and aluminum). PADOH and ATSDR are in agreement with EPA's conclusions [5]. The conclusions are that the soil itself did not appear to pose an unacceptable risk and these contaminants pose no apparent increase in human health risk [5,11].

Evaluation of Indoor Residential Indoor Air Sample Results

Due to concerns that the TCE (and other chlorinated VOCs) plume could be causing vapor intrusion, EPA collected indoor air samples from 22 homes on May 2001. A general attempt was made to include the residential area known or thought at the time to be underlain by the chemical plume. The sampling included people who responded to EPA's request for sampling or who volunteered for their homes to be sampled. Due to the May 2001 findings and because of further concerns in late 2001, PADEP collected more basement air samples from eight homes in 2002. Residential indoor air data for 2001 - 2002 for the selected chemicals of *potential* concern and three rounds of air sampling are summarized in Appendix B - Table 6 [7]. Reviews of the data by EPA in 2003 indicated that eight residences had plume related VOCs above EPA's acceptable risk or above the cancer risk of 1 additional cancer per 10,000 people for prolonged exposure [5].

At the time of the 2001 indoor air sampling, certain detected VOCs were of potential concern and are shown in Appendix B - Table 6 in this PHA [7]. At the time of the PADOH and ATSDR 2002 Indoor Air HC publishing, even though *TCE was the key contaminant of concern* at this site, TCE and 1,1,1-trichloroethane were not listed in the *Summary of Data for Selected VOCs From Indoor Air Samples* table from the 2002 HC (see Appendix B - Table 6) since the sample results were all below the ATSDR CV. PADOH's list of *chemicals of potential concern* for the first three round of indoor air sampling is similar to EPA's list of "chemicals of potential concern" in the EPA RI Appendix E [5].

More indoor air sampling was completed for the residences in 2003. A summary of the occurrence and distribution of organic chemicals found in the indoor air samples for 2001 through 2003 is shown in Appendix B - Table 7 [5]. Background data sets for various chemicals, the ranges of chemicals detected including the maximum results, the frequency of detection of chemicals, and the mean for the sample results for all detected chemicals are listed in Appendix B - Table 7. *TCE* (ranging from 0.24 to 16.4 ug/m^3) was *detected in 10 homes out of 42 sampled* and PCE was detected in 13 out of 42 homes [5]. Out of 42 homes sampled *1,1,1-trichloroethane* (ranging from 0.25 to 1,500 ug/m^3) *was found in 17 homes*. This contaminant was more widely distributed and even found in homes not likely to be affected by the chemical plume [5].

Indoor air is generally not pristine. There are numerous sources of both man-made and natural chemicals in indoor air as shown in Appendix B -Table 1 and its addendum. At room temperature, VOCs may be emitted as gases from certain solids or liquids. Often indoor air pollution comes from sources inside the building. For example, some of the main indoor sources of VOCs are carpeting, glues and adhesives, moth repellents, pesticides, upholstery, manufactured wood products and wood preservatives, furniture polish, copying machines and agents, cleaning agents, perfumes, hair sprays, air fresheners, and many other products used in the house and office. Tobacco smoke from cigarettes, pipes, and cigars is also a very common contributor to high levels of VOCs in the indoor environmental [33]. ATSDR's Toxicological Facts (ToxFAQs) contains information listed by chemical [9,16]. More *Health & Safety Information on Household Products* may be found on the National Institutes of Health and the National Library of Medicine web page, which may be found on-line at

http://householdproducts.nlm.nih.gov/products.htm or write to the U.S. National Library of Medicine, 8600 Rockville Pike, Bethesda, MD 20894 for this information. A number of studies have been published in recent years dealing specifically with chlorinated VOCs in background indoor air. In general, all of these studies have found 1,1,1-trichloroethane, TCE, PCE, benzene, dichloromethane and chloroform to be commonly present in residential indoor air, even at rural locations far removed from industrial sources [33]. However, very few published studies have had adequately low detection limits to detect the presence of 1,2-DCA or vinyl chloride in background indoor air. A few studies identified the presence of 1,1-DCE, but with some uncertainty. More recently, the local community has been concerned about 1,3-butadiene, which had showed up in the soil gas samples and one indoor air sample at one residence. It is a common indoor air contaminant, and currently may or may not be site-related. Indoor air sources of VOCs were determined to contribute to the findings of VOCs in the indoor air of some homes near the Valmont TCE site.

Evaluation of Data from On-Site Monitoring Wells

On-site contaminants found in the monitoring wells are evaluated below and in Appendix B - Table 5 based on the 1988 data. (The contaminants were detected in the monitoring wells and the assumption was made that workers *might* have been drinking the same ground water at some point.)

TCE

In 1988, a maximum TCE concentration of 17,000 ug/L was detected in a monitoring well on-site and strongly suggested that residual pools or pockets of liquid product may have been present in the subsurface (see Appendix B - Table 5). Based on the assumptions previously discussed, the estimated exposure dose by ingestion of the ground water for adults would be calculated at about 0.174 mg/kg/day (a child's dose was not calculated since it is not expected that children were on-site). This estimated dose is 580 times greater than EPA's RfD (the RfD is currently under review by EPA), but *this level is about one (1) to two (2) orders of magnitude less than the NOAELs in chronic exposure animal studies* [9,14]. Other additive TCE exposures in addition to the ground water exposures, such as inhalation during the manufacturing process are not taken into account in this estimated reference dose [2]. Therefore, it is *possible that noncancerous health effects could have occurred to workers if they were consistently exposed* to TCE contaminated ground water on-site through drinking water.

In order to evaluate the possible cancer risks to workers who may have consumed TCE contaminated ground water at the maximum TCE concentration (17,000 ppb), the TCE CSF of 0.02 to 0.4 $(mg/kg/day)^{-1}$ was used (note – the cancer risk association is under review by EPA). Assuming on-site workers were exposed for 10 year to this maximum contamination in drinking water, the cancer risk at this level would be *one (1) excess cancer per 100 people to five (5) excess cancers per 10,000 people* and may have posed a moderate to high increase in the risk of cancer [9,12]. This is a conservative estimate for the drinking water since it takes into account the worst-case scenario and extrapolates a cancer risk actually meant for a person's lifetime evaluation, but it should be noted that it does not take into account additive exposures from the manufacturing process, such as inhalation [15]. Therefore, it is *possible* that carcinogenic health effects could have occurred to workers *if* they were *consistently exposed* to contaminated ground water on-site through ingesting water at the ***highest*** *levels* detected and it is possible that these

exposures will *contribute to carcinogenic health effects over their lifetime*. Since there is not a determined TCE threshold exposure dose for cancer, possibly even chronic exposures to lower concentrations of TCE could theoretically slightly increase a person's risk of developing cancer over their lifetime.

1,1,1-Trichloroethane

The past workers may have been exposed to in the plant's well water at the maximum concentration of 13,000 ppb (see Table 5). Based on this concentration, the estimated exposure dose by ingestion for adults was 0.133 mg/kg/day. EPA withdrew its RfD in 1991. The estimated dose was three orders of magnitude less than the NOAEL in animals [14,16]. Therefore, it is *unlikely* that *noncancerous health effects* would have occurred in workers from the 1,1,1-trichloroethane in the contaminated ground water on-site.

As previously noted, 1,1,1-trichloroethane is not classifiable as to its carcinogenicity in humans and EPA has also determined that 1,1,1-trichloroethane is not classifiable as to its human carcinogenicity [12,14,16,17].

1,1-DCA

Past workers may have been exposed to 1,1-DCA in the plant's well water at the maximum concentration of 370 ppb (Table 5). Based on the assumptions previously discussed, the estimated exposure dose by ingestion for adults would be about 0.0038 mg/kg/day. ATSDR does not have MRLs for 1,1-DCA, nor does EPA have RfDs [16]. The estimated oral exposure dose is about five (5) orders of magnitude less than the level at which the NOAEL was observed in animals [18,19,20]. Therefore, it is *very unlikely* that *noncancerous* health effects would have occurred to workers from the 1,1-DCA contaminated in ground water on-site.

EPA has classified 1,1-DCA as a possible human carcinogen based limited evidence of carcinogenicity in rats and mice, but does not have a CSF for 1,1-DCA in drinking water [12].

1,1-Dichloroethene (1,1-DCE)

The past workers may have been exposed to 1,1-DCE in the plant's tap water at the maximum concentration of 280 ppb. Based on the assumptions previously discussed, the estimated exposure dose by ingestion for adults would be about 0.0029 mg/kg/day. Even though this concentration is 40 to 50 times higher than the EPA's lifetime health concentration, it is below EPA's oral RfD of 0.05 mg/kg/da**y,** and the oral dose is also three (3) orders of magnitude less than the level at which no observed adverse health effects were observed in animals [12,21]. Therefore, it is *unlikely* that *any noncancerous* health effects would have occurred in workers exposed from 1,1-DCE in the ground water.

EPA has withdrawn its CSF and is re-evaluating the cancer risk association to 1,1-DCE [19]. Previously the EPA had established a cancer risk number for 1,1-DCE and based on this theoretical cancer risk estimation for past exposures for 10 years and the assumptions enumerated previously, the predicted cancer occurrence would be about *three (3) additional cancers per 10,000 people* [14]. It is our opinion that past exposure to 1,1-DCE *might* have

posed *a low increased cancer risk* for workers drinking the contaminated ground water, but this is also *uncertain depending on EPA's pending determination* of any associated cancer risk.

Cis-1,2-dichloroethene

Past workers may have been exposed to 1,2-dichloroethene in the plant's well water at the maximum concentration of 1,030 ppb (Table 5). Based on the assumptions previously discussed, the estimated exposure dose by ingestion for adults would be about 0.011 mg/kg/day. This estimated oral dose is at the EPA's chronic oral RfD of 0.01 mg/kg/day, but is also three (3) orders of magnitude less than the NOAEL [14]. Therefore, it is *unlikely* that *noncancerous health effects* could have occurred in workers exposed to 1,2-dichloroethene contaminated ground water onsite over the ten years through ingesting contaminated ground water.

The EPA and IARC have determined that cis-1, 2-dichloroethene is not classified as a human or animal carcinogen [14,22,23].

Carbon Tetrachloride

The past workers may have been exposed to carbon tetrachloride in the plant's tap water at the maximum concentration of 5.8 ppb. Based on the assumptions previously discussed, the estimated oral exposure dose by ingestion for adults would be about 0.00063 mg/kg/day and this is slightly at or less than the EPA's chronic oral RfD of 0.0007 mg/kg/day [12]. The estimated oral exposure dose is about three (3) orders of magnitude less than the level at which no observed adverse health effects were observed in animals [24]. It is *unlikely* that *noncancerous health effects* would have occurred in workers due to ingesting the carbon tetrachloride in the contaminated ground water.

As stated previously under the section for residential wells, EPA has established a CSF for carbon tetrachloride and PADOH used this CSF to evaluate any increased cancer risk. Based on the theoretical cancer risk estimation for past exposures and the assumptions enumerated previously, the predicted cancer occurrence would have been *insignificant* in regard to an increased cancer risk for onsite workers.

Tetrachloroethylene (PCE)

Past workers may have been exposed to PCE in the plant's well water at the maximum concentration of 35 ppb. Based on the assumptions previously discussed, the estimated exposure dose by ingestion for adults would be about 0.00036 mg/kg/day and is much lower than the EPA's RfD of 0.01 mg/kg/day [14]. The estimated oral exposure dose is about six (6) orders of magnitude less than the level at which no observed adverse health effects were observed in animals [25]. Therefore, it is *very unlikely* that *noncancerous* health effects would have occurred to those workers from PCE through drinking the ground water.

PCE has not been shown to cause cancer in people, though it may be an animal carcinogen. A CSF had previously been proposed, but PCE is currently under review by EPA and ATSDR [14].

Evaluation of Data from On-Site Soil Samples

A summary and evaluation of past on-site soil sampling data up to and including June 2002 is shown in the PADOH HC of April 2003 [11]. On May 5, 1988, soil samples revealed TCE and 1,1,1-trichloroethane as the chemicals of *potential* concern [8]. The maximum concentration of the detected contaminants was consistently detected in the soil sample taken below the plant's parking area [8]. In September 1993 and December 2000, more surface soil samples and subsurface soil samples were collected, and VOCs were detected in the on-site soil samples. The chemicals of *potential* concern at that time included 1,1,1-trichloroethane; 1,1-DCA; and TCE [8]. In 2002, more than 100 soil samples were analyzed for organics (VOCs, semi-volatiles, pesticides/polychlorinated biphenyls) and metals. These sample results were below the PADEP Act 2 Cleanup Standards [11]. Past exposures of workers to these contaminants on-site cannot be determined for certain, but appear to have posed no apparent public health hazard [11]. Further evaluation was done on these samples by EPA for purposes of site remediation and worker protection and may be found in the EPA RI Report [5].

Demographics

The Valmont TCE site is located at the junction of West Hazleton borough and Hazle Township, Pennsylvania. According to the year 2000 census records, West Hazleton borough has a total population of 3,542 persons. In this census, about 48 percent of the population is male and 52 percent is female. Twenty-six percent of the population is children and about 5 percent are under the age of 5. About twenty-three percent of the population is 65 years or over; the median age is 42 years. The percentage of owner-occupied housing is about 60, and the percent of renter-occupied housing is about 40 percent. According to year 2000 census records, Hazle Township has a total population of 9,000 persons. About 47 percent of the population is male and about 53 percent is female. Twenty-seven percent of the population is children and about 5 percent are under the age of 5. About twenty-three percent of the population is 65 years or over; the median age is 45 years. The percentage of owner-occupied housing is about 80, and the percent of renter-occupied housing is about 20. More information may be found on the Pennsylvania Data Center Web Census 2000 site http://www.pasdc.hbg.psu.edu/pasdc/census_2000/ [34].

Child Health Considerations

Because children generally receive higher doses of contaminants than adults under similar circumstances, PADOH uses the higher doses in forming its conclusions about the health effects of exposures to site-related contaminants when children are known or thought to be involved. Additionally, ATSDR and PADOH recognize that children are especially sensitive when exposed to many contaminants. This sensitivity may be a result of the following factors: (1) children are more likely to be exposed to certain media (e.g., soil, sediment, air, surface water or water from springs) because they play outdoors and have more of a tendency to put their fingers and objects in their mouths than adults; (2) children are shorter than adults, which means they can breath dust, soil, and vapors close to the ground; and (3) children are smaller, therefore childhood exposure results in higher doses of chemicals per body weight than adults. Other factors that must be considered in relationship to VOCs – especially TCE – is that the percent of body fat of infants and young children is higher than adults and there is evidence that TCE may

be stored in the fat. This is also an important consideration for a child that is receiving breast milk. Children can sustain permanent damage if these factors lead to toxic exposure during critical growth stages. ATSDR is committed to evaluating sites such as the Valmont TCE Site using child health considerations.

ATSDR and PADOH evaluated the likelihood that children living near the site may have been or may be exposed to contaminants at levels of health concern. Overall past exposures from the drinking water posed *no public health hazard*, but there is some uncertainty *if* any individuals drank the water for **ten years** at the **highest levels** of TCE. *A*fter reviewing the information for each of the completed and potential exposure pathways, ATSDR and PADOH conclude that past chronic TCE exposures at the **highest levels** of TCE found in the residential drinking water, *if* it was ingested for **ten years**, *might possibly and may have posed a public health hazard due to combined pathways of ingestion, inhalation, and skin contact.* See the Section in this PHA on *Toxicological and Data Evaluation - Specific On-site and Off-site Contamination* and the estimated exposure doses for children from drinking water. ATSDR, PADEP, PADOH, and EPA addressed indoor air public health hazards by the installation of air carbon filter units and the sump pump covers and/or by suggestions on removal of items, which are the source of the indoor air contamination. Current and future exposures pose no apparent public health hazard. As extra caution, EPA will be replacing the carbon filter units with the more permanent sub slab pressurization removal type systems in the near future.

Community Health Concerns

ATSDR identified the initial community health concerns of this West Hazleton community surrounding the site through meetings with PADEP, EPA, and the local township and EPA records we collected of public health concerns from 2001 into 2004 and through PADOH home visits conducted in 2002 and review of survey forms developed and distributed by a community group. Additional community concerns were identified through three major public meetings between the involved agencies and the affected community in the summer and fall of 2002 and February of 2004. The concerns identified included:

1. *Concerns about living near the chemical spill since the residents felt future health problems could occur in residents as a result of movement of the underground VOC plume toward residences.*

   RESPONSE:
   After a thorough evaluation, ATSDR and PADOH found that current off-site exposures to contaminated ground water pose no apparent public health hazard and that current exposures to contaminants (by way of vapor intrusion) from the ground water plume of contamination into residential indoor air are not expected to cause adverse health effects and are classified as posing no apparent public health hazard to the community residents. Air carbon filters and sump pump covers were installed in 2004 in the residences determined to be at risk. Even though the contamination still exists in the ground water at this time, the majority of contaminated soil has been remediated on-site and studies are underway to evaluate options to remediate the ground water. Future off-site exposures to contaminated ground water pose no public health hazard. Future exposures to contaminants from the plume in residential indoor

air are not expected to cause adverse health effects and (relating to the plume only) are classified as posing no apparent public health hazard to the community residents.

2. *Concerns that the levels of TCE and of other chemicals in private wells on Bent Pine Road were never actually monitored on a regular basis after public water had been installed and while the wells were being used for watering yards and washing cars. Concerns that some of the residential wells were still be used for watering yards and washing cars into 2004 and that these wells had not been capped by the environmental agencies*

   RESPONSE:
   EPA determined that water use for other purposes such as yard watering and car washing had very little health risk. ATSDR and PADOH agree with this conclusion. In 2003, EPA completed an assessment and determined this to be "at least a 100 times below a level of concern" which may be found in the EPA RI Report Section 6.6.4.4 and Appendix G-4 [5]. Some private wells had been used continuously for yard and landscape watering and car washing after the public water had been installed and even into 2004.

3. *Concerns that the ground water, specifically the aquifer under the homes, could take more than ten to twenty years to cleanup and could impact the residents' health over that time period.*

   RESPONSE:
   Based on the indoor air sample results, EPA has taken steps to assure that residents are currently safe (they provided carbon air filtration units or recommended sump pump covers, if needed, and more recently is installing sub slab pressurized systems to affected homes). EPA is still working on the cleanup of the aquifer under the affected residences. This ground water treatment is expected to begin in 2006/early 2007, but the complete length of time that will be needed for an adequate groundwater cleanup action is unknown. The aquifer will be monitored during the clean up and levels should decrease over time. Remediation of soil on-site took place in 2004 and contamination of the groundwater and soil under the Chromatex Plant #2 building has been addressed by EPA. These activities should help restore the quality of the ground water under the residences.

4. *Concerns about the health impact of indoor air, especially the indoor air around the sump pumps. A few homes had increased levels of TCE in the air near where the sump pump is located. These levels did not make other areas of the house unsafe for habitation but as an extra precaution to further reduce indoor air contaminants, EPA recommended installing covers on sump pumps.*

   RESPONSE:
   EPA completed a house-by-house assessment of the indoor air sample results. EPA recommended sump pump cover installations in the homes located over the plume and with sump pumps. In some cases, custom sump pump covers were/need to be constructed.

5. *Concerns about indoor air exposures from chemicals not identified as site related. Some residents expressed concerns specifically about the contaminant, 1,3-butadiene that was detected in some of the soil gas samples and was detected in one home.*

   RESPONSE:
   Several homes had low levels of chemicals not associated with the Valmont TCE site. This is not unusual as many common household products contain chemicals that can contribute to indoor air contamination. PADOH and EPA spoke with residents in 2001 and offered advice on how to reduce contributions from household products. The residents should be advised to remove any possible sources of these chemicals from their homes. It has not been determined for certain whether the very low levels of 1,3-butadiene detected were site related or not. This chemical is very commonly used in adhesives and rubber products (see the section on *Evaluation of Indoor Residential Indoor Air Sample Results* in this document or the National Institutes of Health and the National Library of Medicine web page, which may be found on-line at http://householdproducts.nlm.nih.gov/products.htm). Appendix B - Table 1 in this document contain lists of common sources for chemicals in the indoor air. PADOH is available if you have any questions.

6. *Concerns that specific health problems might be related to the site contamination including:*

   - Headaches, hypersensitivity reactions and skin disorders (especially rashes), sinusitis, and chronic cough
   - Enlarged liver
   - Asthma
   - Multiple birth defects and Down's syndrome
   - Lung cancer,
   - Non-Hodgkin's lymphoma
   - Ovarian cancer
   - Bone cancer
   - Stomach cancer
   - Prostate cancer
   - Brain tumor
   - Macular degeneration
   - Infertility
   - Intestinal disorders
   - Cysts/Ovarian
   - *(Note - The community group VRAP prepared and distributed their own health questionnaire form. Some of the health concerns on the form are already addressed in the previous bullets, but the remainder is included in the following list. Details of the results of the questionnaire may be found on the following page.)*
     - Eye irritations, bronchitis, pneumonia, nausea, hay fever, wheezing, shortness of breath, vertigo, drowsiness, and dry throat
     - Jaundice, abnormal liver functions, hepatitis

- Lung problems, emphysema
- Digestive tract problems, ulcers, colitis, Crone's Disease
- Light sensitivity
- Hair Loss
- Anemia (cancer related), cancer myeloma
- Central nervous system depression
- Chronic fatigue
- Diabetes
- Kidney dysfunction
- Congenital Defects, Chromosomal Abnormalities, Premature Birth, Developmental Delays, Learning Disabilities
- Eye/Ear Abnormalities (Hearing Loss)

RESPONSE: (Note - The following information is very general. Any individual with health problems should contact their personal physician for an evaluation) In 1988, the primary compound of *potential* concern at this site was recognized as TCE and, secondarily was 1,1,1-trichloroethane. In the U.S., about 400,000 workers are routinely exposed to TCE [15]. In a recent monitoring study in the U.S., it was found that average levels of TCE in surface water range from 0.1 ppb to 1 ppb of water and in groundwater the average is 7 ppb. The chemical can also get into the air or water in many ways, for example, at waste treatment facilities; from paints, paint removers, glues, and other products; or by release from factories where it is made or used. People living near hazardous waste sites may be exposed to it in the air or in their drinking water, or in the water used for bathing or cooking. Other products that may contain TCE are type-correction fluids, spot removers, rug cleaning fluids, and metal cleaners [9,15].

The chemical 1,1,1-trichloroethane has been commonly found in rivers and lakes (up to 10 ppb), in soil (up to 120 mg/kg soil), in finished drinking water (up to 3.5 ppb), and in drinking water from ground water wells (up to 5,400 ppb). Releases during manufacture and transportation and during industrial or household use can cause these high levels, but the levels vary substantially from one location to another. Certain foods you eat and water you drink or bathe in may be contaminated with 1,1,1-trichloroethane. However, most people are exposed to 1,1,1-trichloroethane primarily by drinking contaminated water and eating contaminated food [17].

In 2003, the citizens' group (VRAP) asked the community at the Valmont TCE site to fill out a health-related questionnaire. Thirty-six forms were completed in a community of about 300 persons at the time. Tallies of the health concerns from these forms are as follows: For adults - Excessive Nausea/Vomiting (4/36); Lung Problems (5/36); Hay Fever/Wheezing, Shortness of Breath (18/36); COPD/Chronic bronchitis (5/35); Emphysema (1/35); Pneumonia (2/35); Digestive Tract Problems (10/36); Ulcers (7/34); Jaundice/Hepatitis (2/34); Colitis (1/34); Crohn's Disease (1/34); Other (2/23); Eye Irritations (11/35); Headaches (21/37); Vertigo (2/35); Drowsiness (7/35); Dry Throat (9/36); Light Sensitivity (6/35); Unusual Hair Loss (Alpecia) (5/34); Allergies (20/35); Any Neurological Problems (2/34); Stroke (0/31); Central Nervous System (CNS) Depression (3/35); Hematological Problems (2/34); Anemia (7/35);

Blood Disorders (1/32); Non-Hodgkin's Lymphoma (2/36); Liver Cancer (0/36); Unusual Tumors (6/36); Abnormal Liver functions (2/36); Kidney Dysfunction (2/37); Enlarged Liver (1/36); and other conditions since moving to the site (15/28). For children – Premature Birth (1/16); Oxygen required (1/16); Developmental Delays (3/15); Learning Disabilities (3/18); Congenital Defects (1/14); Oral Cleft (1/16); and Eye/Ear Abnormalities – Hearing Loss (1/16). (Readers' note - This information was not collected by PADOH or ATSDR and cannot be used statistically, but is good information in presenting a picture of the community health concerns.)

Information gathered by the ATSDR's National Exposure Registry, TCE subregistry provides further evidence that exposure to TCE and other chemicals might be associated with the higher rates of adverse health outcomes reported by registrants [35]. This registry contains information from a cohort of sites, but currently is closed and does not include information from the Valmont TCE site. ATSDR found that, in general, registrants reported some health conditions at a higher rate than the general population. Certain age groups reported some health conditions more frequently, and some had higher rates for only men or only women. Health conditions reported in excess at one or more of the interview time periods included: *Anemia, Diabetes, Hearing Impairment, Hypertension, Kidney Disease, Liver Problems, Skin Rashes, Speech Impairment, Stroke, and Urinary Disorder*. However, other factors--such as other chemical exposures at work or at home, personal lifestyle choices (smoking or drinking alcohol), and complications related to other health conditions-- might have caused the higher rates. The National Exposure Registry is collecting more detailed information on some of the health conditions to better evaluate these results [35].

- ✓ ***Headaches, hypersensitivity reactions and skin disorders (especially rashes), sinusitis, and chronic cough***

  Headaches - At this time, there is no known link between TCE exposure at the levels found off-site at Valmont, and this health effect. TCE was once used as an anesthetic for surgery. People who are exposed to parts per million levels (one part per million equals **one thousand times** a ppb concentration) of TCE can become dizzy or sleepy and may become unconscious at very **high** parts per million levels. People who breathe **moderate** parts per million levels of TCE may have headaches or dizziness [9, 15]. It is possible that some people who breathe **high** parts per million levels of TCE may develop damage to some of the nerves in the face. People have reported health effects when exposed to level of TCE at which its odor is noticeable [15]. The levels of TCE in the indoor air at the Valmont site are thousands of times lower than the ones discussed as associated with the health effects above, so *we would not expect headaches at these levels*.

  Allergy to TCE and Skin disorders (especially rashes) –There is no known link between TCE exposure at the levels found off-site at Valmont, and this health effect, at this time. Some people who have worked with TCE for long periods of time (**high** parts per million or thousands of parts per billion concentrations) may develop or have developed an allergy to TCE or become particularly sensitive to its effects on the skin. People who were experimentally exposed to 200,000 ppb

(or 1,074,000 ug/m3) of TCE vapor for 7 hours experienced dry throats (40% of the subjects), beginning after 30 minutes [9]. The subjects experiencing these symptoms did not experience them when exposed in the same manner on five other consecutive days. These effects are presumed to be due to direct contact with the vapor [9]. These levels are about five (5) orders of magnitude above (or about 100,000 times) the levels seen in the indoor air at this site**,** so we do not expect to see these symptoms at this site.

Skin irritation and rashes have resulted from occupational exposure to TCE [2]. Skin rashes are also one of the most frequently reported health conditions in interviews conducted by the National Registry for TCE Exposures [35]. The dermal effects are usually the consequence of direct skin contact with concentrated solutions, but occupational exposure also involves vapor contact. Adverse effects have not been reported from exposure to dilute aqueous solutions. Stevens-Johnson syndrome, a serious and potentially fatal skin condition, was seen in five people occupationally exposed to TCE acutely at high levels. The study authors suggested that the disease was caused by a hypersensitivity reaction to TCE. An exfoliative dermatitis and scleroderma - also thought to have an immune component - have been reported in persons occupationally exposed to TCE. Histopathological changes in the skin were not observed in experimental animals exposed to 600,000 ppb (or 3,222,000 ug/m3) TCE, 7 hours per day and 5 days per week for 104 weeks [9]. These levels are about five (5) orders of magnitude above (about 100,000 times) the levels seen in the indoor air at the site, so we do not expect to see these symptoms and especially rashes, at this site.

Sinusitis - Acute sinusitis is an infection of limited duration in the sinus cavities. Inflammation that occurs with allergies may block sinus drainage and increase susceptibility to sinusitis. Chronic sinusitis is a prolonged infection or inflammation of the sinus cavities. Inflammation that occurs with allergies may block sinus drainage and increase susceptibility to sinusitis [36]. There is no known link between TCE and sinusitis.

Chronic cough – Currently, there is no known link between TCE exposure and this health effect at the levels found off-site at Valmont. People who smoke may increase their risk of toxic effects from TCE at **high** concentrations [35]. In one documented case, a worker developed labored breathing and respiratory problems after welding steel that had been washed with TCE; the worker was also a cigarette smoker [9]. At **high** levels (thousands of parts per billion levels) of TCE, inhalation changes have been seen in cells in the lungs of animals [9].

- ✓ ***Enlarged liver*** – If a person breathes the chemical TCE, about half the amount will get into the bloodstream and organs [9,15]. The person will exhale the rest. If a person drink water with TCE in it, most of it will be absorbed into their bloodstream [9,15]. If TCE comes in contact with a person's skin, some of it can enter their body, although not as easily as when it is breathed or swallowed [9,15]. Once in the blood, the liver changes much of the TCE into other chemicals. The majority of these breakdown products leave the body in the urine within a day.

The adverse health effects reported at **high** levels include liver and kidney damage and changes in heartbeat. The levels at which these effects occur in humans are not well characterized. Animals that were exposed to moderate levels of TCE had enlarged livers, and high-level exposure caused liver and kidney damage [15]. Presently, there is no known link between this health effect and TCE exposure at the low levels found off-site at the Valmont site.

People who consume alcohol or who are treated with drugs for alcoholism may be at greater risk of TCE poisoning. Ethanol and disulfiram (Antabuse) can both inhibit the metabolism of TCE and can cause it to accumulate in the bloodstream, affecting the nervous system. Compromised hepatic and renal function may place one at higher risk upon exposure to TCE or its metabolites since the liver serves as the primary site of TCE metabolism and the kidney as the major excretory organ for TCE metabolites [9].

- ✓ ***Asthma*** –Asthma is a chronic condition that occurs when the main air passages of the lungs, the bronchial tubes, become inflamed. A person is more likely to develop asthma if they have an inherited predisposition to the condition and are sensitive to allergens or irritants in they environment. The inflammation that causes asthma makes airways overly sensitive to a wide range of environmental triggers, including air pollutants and irritants [36]. Asthma is the most common chronic illness of childhood. Researchers have identified a number of factors that may increase the chances of developing asthma. These asthma-associated factors include living in a large urban area, exposure to secondhand smoke, exposure to occupational triggers, having at least one parent with asthma, having respiratory infections in childhood including the Respiratory Syncytial virus, having Gastroesophageal Reflux Disease, having a low birth weight, and being obese [36].

  One study suggested increased respiratory disorders including asthma in children with chronic exposure to municipal wells contaminated with several solvents including TCE at 267 ppb and PCE at 21 ppb [9]. Additional research is needed to confirm these findings. EPA is revaluating the chronic oral RfD for TCE, but the estimated exposure dose of TCE expected in children in the case above is below ATSDR's acute MRL [12,14]. At this time, there is no confirmed link between this health effect and TCE exposure at the levels found off-site at Valmont.

- ✓ ***Multiple birth defects and Down's syndrome*** –A woman's chances of giving birth to a child with Down's syndrome increases with age. Because a woman's eggs age, there's a greater inclination for chromosomes to divide improperly [36]. More and more studies suggest that more birth defects may occur when mothers consistently drink water contaminated with TCE, but it is still uncertain whether people who breathe air or drink water contaminated with TCE are at higher risk of having reproductive effects [9,15]. Research on the genotoxicity of TCE suggests it is a very weak, indirect mutagen (the chemical is able to mutate DNA, but it is not necessarily a carcinogen). A mechanism for carcinogensis and the potential

for heritable gene mutations are not known. In some studies of workers who were occupationally exposed to TCE, mutations were found, but in other studies no mutations were found [9].

In one study, the authors suggested that smoking and TCE exposure may act together to produce increased sister chromatid exchange frequency (the crossover and breakage and recombining of chromosomes). In the same study in a general comparison between smokers and nonsmokers, the authors showed no significant differences in the rate of sister chromatid exchange. This study was limited by a relatively small sample size. Other researchers have found no significant increase in the rate of sister chromatid exchange among either smoking or nonsmoking workers *and exposure to TCE* [9].

In one study, one community that used water with **high** (thousands of parts per billion) levels of TCE for several years may have had a higher incidence of childhood leukemia than other people, though these findings are not conclusive. In another study of TCE exposure from well water, increased numbers of children were reported to be born with heart defects; this might be supported by data from some animal studies showing developmental effects of TCE on the heart. *However, other chemicals* were also in the water from this well and may have contributed to the heart defects. One study reported a higher number of children with a rare defect in respiratory system and eye defects. Another study reported that the risk for neural tube defects and oral cleft palates was higher among mothers with TCE in their water during pregnancy [9]. Children listed in the National Exposure Subregistry of persons exposed to TCE were reported to have higher rates of hearing and speech impairment [15]. See Appendix D in this PHA for more information. In conclusion, we know that as a mother's age increases at childbirth, the chances of giving birth to a child with Down's syndrome increases. The evidence with TCE exposure and Down's is inconclusive. A possible association with birth defects has been suggested, although Down's has not been specifically linked to exposures to this chemical.

✓ ***Cancers: Lung cancer, non-Hodgkin's lymphoma, ovarian cancer, bone cancer, stomach cancer, and prostate cancer*** –

A number of epidemiological studies have been conducted to investigate human exposure to TCE and subsequent tumor development related to the workplace. These investigators did not find significant increases in incidence of cancer, but some studies were limited by relatively small numbers of subjects, lack of lengthy follow-up periods, and multiple chemical exposure [9]. In addition, several retrospective cohort studies of workers exposed to TCE have been conducted. All of these studies have limitations that restrict their usefulness for evaluating the carcinogenicity of TCE. None has shown clear, unequivocal, evidence that TCE exposure is linked to an increased cancer risk. *It is uncertain whether people who breathe air or drink water contaminated with TCE are at higher risk of cancer* [15].

In six out of eight Swiss epidemiological studies, workers chronically exposed to paints and solvents including TCE for five years or more seemed to have a correlation with the development of multiple myelomas [38]. Multiple myeloma is a cancer of the plasma cell and is an incurable but treatable disease. All workers manifesting this disease had **high** paint and/or solvent exposures [36]. In studies using **high** (thousands of parts per billion) doses of TCE in rats and mice, tumors in the lungs, liver, and testes were found. This provides some evidence that **high** (thousands of parts per billion) doses of TCE can cause cancer in experimental animals. Based on the limited data in humans regarding TCE exposure and cancer, and evidence that high doses of TCE can cause cancer in animals, the IARC has determined that TCE is probably carcinogenic to humans [15].

A survey of Finnish workers exposed to TCE found an association between exposure and incidence of stomach, liver, prostate, and lymphohematopoietic cancers (cancers affecting lymph and blood). However, the study did not reliably separate the effects of individual chemicals. In other studies, associations between liver cancer and TCE exposure have not been observed. A significant association between workplace exposure to TCE and kidney cancer was found in a retrospective cohort study of German workers, but chemical exposure levels were not provided in this study. Thus, the human studies that did show increases in cancer are limited by uncertainties in the exposure data, small sample sizes, and likely exposure to other chemicals [9]. No absolute link was determined between TCE exposures at the levels found off-site at Valmont and human cancers.

Lung cancer - Lung cancer is the leading cause of cancer deaths in the U.S., among both men and women. Smoking accounts for about 85 to 90 percent of lung cancer cases. Women smokers are at greater risk of lung cancer than are men. Daily exposure to secondhand smoke may increase the chances of developing lung cancer by as much as 30 percent. Some other leading causes of lung cancer are exposures to radon, asbestos, and some other industrial cancer-causing agents [36]. In experimental animals (mice), lung cancer might be associated at **high** (thousands of parts per billion) TCE levels, especially where there is also a body burden of 1,1,1-trichloroethane [9].

Non-Hodgkin's Lymphoma – Non-Hodgkin's lymphomas (NHL) is one of two common types of cancers of the lymphatic system. Hodgkin's disease, the other type, is far less common than NHL. In 2003, there were about 7,600 new cases of Hodgkin's disease compared with 53,400 new cases of NHL in the U.S. Doctors do not know exactly what causes NHL, but researchers believe that the activation of certain abnormal genes may be involved in the development of all cancers, including lymphomas [36].

Although the data are not entirely consistent, occupations dealing with chemicals and agriculture appear to be associated with NHL in some studies [37]. In a study of Swedish workers, a statistically significant increase in NHL was observed [9].

This study found an increased risk of NHL associated with occupational exposure to TCE though these workers were exposed to other solvents in addition [9].

Ovarian cancer - Ovarian cancer is the fifth most common cancer in women. It's diagnosed in more than 25,000 women in the United States each year. The causes of ovarian cancer remain unknown [36]. Some researchers believe it has to do with the tissue-repair process that follows the monthly release of an egg through a tiny tear in an ovarian follicle (ovulation) during a woman's reproductive years. The formation and division of new cells at the rupture site may set up a situation in which genetic errors can occur. Others propose that the increased hormone levels before and during ovulation may stimulate the growth of abnormal cells [36]. There is no known connection between ovarian cancer and TCE exposures.

Bone cancers - Sarcomas (one type of bone cancer) are cancers that originate either in the bones or in the soft tissues of a person's body. Cancer doctors recognize more than two dozen types of sarcoma [36]. Soft tissues connect, support and surround other body structures. They include muscle, fat, blood vessels, nerves, tendons and the lining of your joints (synovial tissues). A large variety of cancers can occur in the bones and in numerous soft tissues. Primary bone cancer is rare, with only about 2,400 new cases a year in the United States, and primary bone cancer usually affects more children than adults. In general, no one knows for sure what causes most bone or soft tissue sarcomas. In a few cases, sarcomas may be hereditary, such as in Li-Fraumeni syndrome or in families with neurofibromatosis [36]. Li-Fraumeni syndrome is a condition of early breast cancer associated with soft tissue sarcomas and other tumors. Neurofibromatosis results in developmental changes in the nervous system, resulting in nerve tumors. Other abnormalities associated with neurofibromatosis include skin changes and bone deformities. Some environmental factors that are thought maybe to lead to an increased risk of sarcoma might possibly include exposures to high doses of certain herbicides, as well as large doses of radiation.

Stomach cancer – Stomach cancer is twice as common in men as it is in women. Diets high in foods preserved by smoking, salting, or pickling increases your risk of stomach cancer. Consistently eating foods that contain nitrites and nitrates, such as bacon, ham, and processed meats also increase your risk of developing stomach cancer. Regularly, eating large amounts of barbecued or well-done red meat also increases your risk. Drinking excess alcohol can cause a number of problems, including irritation of the stomach and esophagus that may lead to cancer. Cigarette smoking has also been implicated in stomach cancer. Other risk factors may include your country of origin (especially Japan, Korea, parts of eastern Europe, and Latin America), having previous stomach surgery, being obese, having stomach polyps, or having a family history of stomach cancer. Certain workplace contaminants, such as coal dust, asbestos and nickel, have been linked to an increased risk of stomach cancer. Studies show that having both *H. pylori* and a form of a gene that causes low stomach acid greatly increases your risk of stomach cancer [36].

Prostate cancer - Prostate cancer is the most common cancer, excluding skin cancers, in American men. As men age, the risk of prostate cancer increases [36]. It's estimated that by age 50, about one-third of all men have some cancerous cells in the prostate gland. By 80, this increases to about three-quarters. If a close family member has prostate cancer, your risk of the disease is greater than that of the average American man. The average age at diagnosis in the United States is 70. Prostate cancer is the second-leading cause of cancer deaths in American men [36].

- ✓ ***Brain tumor*** – At this time, there is no known link between TCE exposure at the levels found off-site at Valmont, and brain tumors. Primary brain tumors originate in the brain and can be noncancerous (benign) or cancerous (malignant). It's not known what causes these brain tumors. Currently, studies are being done to determine whether heredity, viruses, environmental factors, or other factors play a role in their development. Secondary brain tumors result from cancer that has started elsewhere and spread (metastasized) to the brain. Primary brain tumors are less common than secondary brain tumors [36]. Because doctors do not know exactly what causes primary brain tumors, it's difficult to pinpoint risk factors. Brain tumors sometimes strike several members of a family, suggesting heredity may be a risk factor. Heredity appears to account for a small minority of brain tumors. Overall, males and whites are more likely to develop a brain tumor. Although brain tumors can occur at any age, they're most common in people older than 65 [36].

  Some types of brain tumors appear to occur more frequently in people who are exposed to radiation or certain chemicals, such as those who work in oil refining, rubber manufacturing, and chemical and nuclear industries. But a definite link between exposure to chemicals and brain tumors has not been proved. Similarly, electromagnetic fields and the use of cell phones have been studied as causes of primary brain tumors, but no definitive medical evidence indicates that either causes brain tumors [36].

- ✓ ***Macular degeneration*** – The macula is at the back of the eye in the center of the retina. The macula is made up of densely packed light-sensitive cells called cones and rods. The cones are essential for central vision and a healthy macula is needed for normal central vision acuity. As a person ages, the Retinal Pigment Epithelium (RPE) may deteriorate, lose its pigment, and become thin (a process known as atrophy) [36]. The RPE is a critical passageway for nutrients from the choroid - an underlying layer of blood vessels that nourishes the cones and rods of the retina - to the retina and helps remove waste products from the retina to the choroids. Two types of macular degeneration may occur (Dry and Wet form), but both are related to deterioration of the RPE, brought on by aging, and a breakdown in the waste removal system may be involved in both forms [36].

  No evidence of VOC exposure was found to be related to the occurrence of macular degeneration, but some possible risk factors for the disease include: cigarette smoking, cardiovascular disease, obesity, lighter-colored eyes, exposure

to excess ultraviolet light (or sunlight), and diets lacking certain nutrients such as zinc and antioxidants. A family history of macular degeneration can increase a person's risk of getting the disease [36].

- ✓ ***Infertility*** - Infertility differs from sterility. Infertility simply means that becoming pregnant may be a challenge rather than impossibility. More than 6 million American couples are affected by infertility, with the male partner being either the sole or a contributing cause in approximately 40 percent of infertile couples. Problems with female fertility are present about one-half to two-thirds of the time. In both men and women, multiple factors can account for difficulty with fertility [36].

  Endocrine disrupting chemicals have been implicated in contributing to animal and human infertility, though not proven in humans, especially at low levels. *TCE is not considered an "endocrine disruptor"* or hormone-disrupting chemical [38]. Endocrine disruption occurs when a chemical interferes with the function of natural hormones in the body, for example by mimicking a hormone, blocking its effects, or stimulating or inhibiting the endocrine system [38]. The endocrine system consists of various organs known as endocrine glands, including the ovaries, the testes, and the adrenal, thyroid and pituitary glands. These glands release hormones, such as estrogen, testosterone or adrenaline, into the bloodstream. Hormones travel through the bloodstream in small concentrations, bringing chemical messages to distant cells, to regulate diverse functions including reproduction, development, and metabolism.

- ✓ ***Intestinal disorders*** – Irritable bowel syndrome (IBS) is one of the most common disorders that physicians see. Up to one in five American adults has irritable bowel syndrome. The disorder accounts for more than one out of every ten doctor visits [36]. Abdominal pain or cramping and changes in bowel function including bloating, gas, diarrhea and constipation characterize IBS. For many years IBS had been considered a psychological rather than a physical problem. No one knows exactly what causes IBS. Some researchers believe IBS is caused by changes in the nerves that control sensation or muscle contractions in the bowel. Others believe the central nervous system may affect the colon [36]. There is no known link between TCE exposures and this health effect.

- ✓ ***Cysts/Ovarian*** - At this time, there is no known link between TCE exposures and this health effect. Many women have ovarian cysts at some time during their lives. Most cysts present little or no discomfort and are harmless. The majority of cysts disappear without treatment within a few months. Sometimes a normal monthly follicle just keeps growing (*Folicular cyst*). When that happens, it becomes known as a functional cyst. A follicular cyst begins when luteinizing hormone (hormone that causes release of the egg) surge doesn't occur. The result is a follicle that doesn't rupture or release its egg. Instead it grows and turns into a cyst. Follicular cysts are usually harmless, rarely cause pain and often disappear on their own [36]. Sometimes, however, the egg's escape opening seals off and fluid accumulates inside the follicle, causing the corpus luteum to expand into a

cyst (called a C*orpus Luteum cyst*). Although this cyst usually disappears on its own in a few weeks, it can grow to almost 4 inches in diameter and has the potential to bleed into itself or twist the ovary, causing pelvic or abdominal pain. If it fills with blood, the cyst may rupture, causing internal bleeding and sudden, sharp pain [36]. Although there's no definite way to prevent the growth of ovarian cysts, regular pelvic examinations are a way to help ensure that changes in ovaries are diagnosed as early as possible [36].

- ✓ Eye irritations, bronchitis, pneumonia, nausea, hay fever, wheezing, shortness of breath, vertigo, drowsiness, and dry throat
  Eye irritations, hay fever, dry throat – see the previous discussion on hypersensitivity reactions. Eye irritations and dry throat may come from many health problems, as well as from allergy [36]. Usually common bacteria cause bronchitis and pneumonia, often secondary to another microbial infection. Predispositions (e.g., such as chronic illnesses or impaired immune systems) may increase the likelihood of infection. Also see the previous section on chronic cough. Wheezing, shortness of breath, vertigo, and drowsiness may be the results of various health problems that need to be evaluated by a physician [36]. We do not expect these health effects at the levels of TCE contamination at this site.

- ✓ Jaundice, abnormal liver functions, hepatitis
  Jaundice, abnormal liver functions – see the previous section on enlarged liver. Death associated with liver damage has also been reported in persons occupationally exposed to TCE for intermediate and chronic durations, followed by a high acute-duration exposure [9]. Hepatitis is caused by infections of the hepatitis A, B, C, or E viruses, alcohol abuse or autoimmune disease [36]. Presently there is no known link between these health effects and the TCE levels found at this site.

- ✓ Lung problems, emphysema - see the previous section on chronic cough on pages 30-31.

- ✓ Crohn's Disease, digestive tract problems, ulcers, and colitis - An estimated 500,000 Americans have Crohn's disease, an inflammatory bowel disease (IBD) that causes chronic inflammation of the intestinal tract. Crohn's disease and ulcerative colitis are similar. Like ulcerative colitis, another common IBD, Crohn's disease can be both painful and debilitating and sometimes may lead to life-threatening complications [36]. There is no known link between TCE exposures and this illness.

- ✓ Light sensitivity - Light-sensitive eyes can be a result of many diseases from Sjogren's syndrome, an autoimmune disease, to corneal ulcers [36]. You should discuss this problem with your physician.

- ✓ Hair Loss - Hair loss may result from heredity, medications or underlying medical conditions [36]. There is no known link between TCE exposures and this problem.

- ✓ Anemia (cancer-related), multiple (cancer) myeloma

  Anemia (cancer-related) - Cancer survivors can experience side effects long after their cancer treatment ends [36]. Multiple myeloma - Although the exact cause isn't known, doctors do know that multiple myeloma begins with one abnormal plasma cell in your bone marrow. This abnormal cell multiplies and doesn't mature or die as normal cells so they accumulate, eventually overwhelming the production of healthy cells. Healthy bone marrow consists of a small percentage of plasma cells, less than 5 percent, but in people with multiple myeloma, the percentage of plasma cells often increases to more than 10 percent. Uncontrolled plasma cell growth can damage bones and surrounding tissue. It can also interfere with your immune system's ability to fight infections by inhibiting your body's production of normal antibodies [36]. There is no evidence that TCE exposures are associated with these conditions and a physician should be seen for these conditions.

- ✓ Central nervous system depression and heart problems – Inhalation and ingestion are the primary exposure routes, and the liver, heart, and central nervous system are the primary targets for both routes [9]. In humans and laboratory animals, at very high TCE exposure levels, death was often caused by the central nervous system depression. Humans have died from breathing very high concentrations of TCE fumes. Most of the reported deaths have been associated with accidental breathing of unusually high levels of TCE vapors in the workplace, often during its use in degreasing operations or dry-cleaning operations. Deaths have also resulted from the early use of TCE as an anesthetic as well as the intentional inhalation of concentrated fumes from TCE containing typewriter correction fluid and cleaning fluids. None of these cases provided adequate exposure level or duration data to define with accuracy the levels of inhalation exposure that cause human deaths. A study that examined the interaction between exposure concentration and time of exposure on nervous system function found that concentration, rather than time of exposure, was more important in determining effects [9]. Presently there is no known link between these health effects and the TCE levels found at this site.

- ✓ Chronic fatigue - Chronic fatigue syndrome is one of the most mysterious syndromes. It is a flu-like condition that can drain your energy and sometimes last for years. People previously healthy and full of energy may experience a variety of symptoms, including extreme fatigue, weakness and headaches as well as difficulty concentrating and painful joints, muscles and lymph nodes. It can start during or shortly after a period of high stress or come on gradually without any clear starting point or any obvious cause. Unlike infections, it has no clear cause an unlike conditions such as diabetes or anemia, there's essentially nothing to measure. Doctors don't know the cause of chronic fatigue syndrome. Several possible causes have been proposed, including: iron deficiency anemia; low blood sugar (hypoglycemia); history of allergies; virus infection, such as Epstein-Barr virus or human herpesvirus 6 D; dysfunction in the immune system; changes in the levels of hormones produced in the hypothalamus, pituitary glands or adrenal

glands; and mild, chronic low blood pressure (hypotension). The cause of chronic fatigue syndrome may be an inflammation of the pathways of the nervous system as a response to an autoimmune process, but with nothing measurable in the blood like in other autoimmune diseases, such as rheumatoid arthritis or lupus. Chronic fatigue syndrome may also occur when a viral illness is complicated by a dysfunctional immune system. Some people with chronic fatigue syndrome may have a low blood pressure disorder that triggers the fainting reflex. In many cases, however, no serious underlying infection or disease is proved to specifically cause chronic fatigue syndrome. Lack of medical knowledge and understanding of chronic fatigue syndrome has made determining and describing the characteristics of the condition difficult [36].

- ✓ Diabetes - In diabetes, the body doesn't make or properly use insulin. Insulin is the hormone that helps your body convert sugar, starches and other foods into energy. The exact cause of diabetes isn't known, but genetics, excess weight and inactivity play a role [36]. There is some evidence that TCE metabolism creates adducts that interfere with insulin activity, but this is short lived and the concentrations needed remain in question [39]. Another study, in an occupational setting (higher TCE levels), shows steroid and insulin interference from TCE exposures [40]. These studies may suggest areas for further research.

- ✓ Kidney dysfunction - Once in the blood, the liver changes much of the TCE into other chemicals. The majority of these breakdown products leave the body in the urine within a day. The adverse health effects reported at **high** levels include liver and kidney damage and changes in heartbeat. The levels at which these effects occur in humans are not well characterized. Animals that were exposed to moderate levels of TCE had enlarged livers, and high-level exposure caused liver and kidney damage [15]. Presently, there is no known link between this health effect and TCE exposure at the low levels found off-site at the Valmont site.

- ✓ Congenital Defects, Chromosomal Abnormalities, Premature Birth, Developmental Delays, and Learning Disabilities – see the previous discussion on birth defects and TCE on page 32.

- ✓ Hearing Loss (child) - Among persons in the ATSDR exposure subregistry, a statistically significant impairment in hearing was reported in children age 9 years or younger. Because the time of onset for hearing loss is not available, it is not known if this effect may be a result of in utero exposure or exposure after birth. The study authors cautioned that their study does not identify a causal relationship between TCE and effects but does suggest areas for further research [9].

7. *Concerns about cumulative past exposures and risks due to exposures, via indoor and outdoor air and ground water, and especially in 1986 and 1987.*

RESPONSE:
Public drinking water has been provided to residents impacted by the site since 1988. Monitoring wells have defined the groundwater plume of contamination and we will

continue to evaluate any groundwater contamination through these wells to ensure the safety of residents. The levels of contamination in the groundwater will decrease with the future off-site ground water cleanup and the completed excavation of the on-site soil contamination. Additionally, the on-site groundwater and soil under the Chromatex Plant #2 building will be treated by in-situ chemical oxidation and by SVE.

As discussed earlier in this document, the percent of body fat of infants and young children is higher than adults and there is evidence that TCE may be stored for a while in fat. The toxicology of TCE is challenging to evaluate since many pathways and many metabolites may be involved in the body and there are still many gaps in our knowledge of the health effects related to TCE exposures. The estimated doses are about two (2) to three (3) orders of magnitude less than the NOAEL in animal studies as discussed in the *Toxicological and Data Evaluation* section in this document. The Science Advisory Committee has suggested a lower NOAEL or LOAEL to be used by EPA [41]. Therefore, the estimated dose ***for a child*** might only be one (1) to two (2) orders of magnitude less than the LOAEL and it is *possible that there could have been non-cancerous health effects related to these exposures at the **highest** levels of TCE in the ground water*.

Specifically, it is *uncertain* whether people who drank water contaminated with *TCE at the higher levels and consistently over the ten-year period are at increased risk of cancer or other diseases associated with TCE exposures.* Since a threshold exposure dose has not been determined, possibly even chronic exposures to lower levels of TCE could theoretically slightly increase a person's risk of developing cancer over their lifetime. For the residents, there is also a small amount of uncertainty about past cumulative effects from all of the combined VOC exposures and from all combined pathways off-site and on-site. See Appendix B –Table 8 for the *Past and Current Cumulative Exposures and Summary for Residential Exposures to TCE and 1,1,1-Trichloroethane.*

8. *Questions whether there will be a local epidemiological study.*

RESPONSE:
The residential population exposed is too small to justify a local epidemiological study. A study based on too small a population is not statistically valid.

Health Outcome Data Evaluation

The Commonwealth of Pennsylvania maintains health outcome databases including vital statistics and the cancer registry. These databases provide information on total mortality, cancer morbidity and birth defects. The residential population exposed around the Valmont TCE site is too small for a meaningful review of health outcome data.

Conclusions

Based on a thorough evaluation, ATSDR and PADOH conclude that overall current, future, and past off-site exposures to contaminated ground water and soil posed *no apparent public health hazard.* In general, though there is some uncertainty about past cumulative effects of all of the combined VOC exposures, from all combined exposure pathways off-site and on-site.

1. Current off-site exposures to contaminated ground water pose *no apparent public health hazard*. Future off-site exposures to contaminated ground water pose *no public health hazard*. Past exposures to the ***highest*** VOC concentrations (especially to TCE) in the residential well water for ***ten years*** poses some uncertainty relating to expected adverse health effects. Most of the residential well water tested lower for VOC concentrations in 1988 and, therefore, are concluded by PADOH and ATSADR to have posed *no apparent public health hazard* in the past. However, exposures in the past to the ground water at the ***highest levels*** off-site might possibly and may have yielded adverse health effects (assuming ***ten year***s of exposure).

2. Current and future exposures to contaminants (by way of vapor intrusion) from the ground water plume of contamination into residential indoor air are not expected to cause adverse health effects for the community residents and pose no apparent public health hazard. Air carbon filters and sump pump covers were installed in 2004 in the residences determined to be above EPA risk thresholds. Even though the contamination still exists in the ground water at this time, the majority of the contaminated soil has been remediated on-site and plans are underway to treat/remediate the ground water under the residences.

3. Past, current and future exposures to off-site contaminated soil are not expected to cause harmful health effects and are classified as posing *no apparent public health hazard*. The off-site levels are below levels of health concern. Occasional contact with surface soil contaminants, even at the highest off-site levels reported, is not expected to pose a health concern for adults or children.

4. Past exposures on-site may have included workers drinking water from the plant's wells and, in that case, could have caused health problems, but details are unknown. Past, cumulative exposures by inhalation were likely on-site, but actual amounts are unknown and therefore, pose an *indeterminate public health hazard*. Past exposure of workers and trespassers to on-site soil is also unknown. Therefore, past on-site exposures are classified as *an indeterminate public health hazard*.

Recommendations

1. Sump pump covers were/are recommended for homes (above the plume) with sump pumps whether plume-related contaminants were detected or not as a precautionary measure. Sump pump covers have been or may need to be specially built for homes with sump pumps to help minimize vapor intrusion.

2. The residents should continue to remove any possible sources of VOCs in their homes not originating from the chemical plume. The Appendix B - Table 1 (and its addendum) contains lists of common sources of VOCs in indoor air. Also, the residents might wish to visit the National Institutes of Health and the National Library of Medicine web page for ***Health & Safety Information on Household Products.*** This web page/database may be found on-line at http://householdproducts.nlm.nih.gov/products.htm or information may be obtained by writing to the U.S. National Library of Medicine, 8600 Rockville Pike, Bethesda, MD 20894.

3. EPA should continue to collect additional private well samples and on-site monitoring well samples to determine if ground water quality is improving now that on-site soil remediation has taken place and after EPA's currently planned future ground water treatment system is implemented.

4. ATSDR and PADOH recommend that installing household carbon filtration or vapor reduction systems in all of the homes affected by the plume (as determined by EPA) would reduce stress and uncertainty for all of the residents in the affected neighborhood. If household carbon filtration or vapor reduction systems are not installed in all of the homes determined to be affected by the plume at this site, ATSDR and PADOH recommend that the indoor air in the affected homes be retested in the future to ensure that indoor air contaminant levels remain below levels of public health concern.

Public Health Action Plan

The public health action plan (PHAP) contains a description of actions to be taken (or that have been taken) by ATSDR and/or other government agencies at and in the vicinity of the site subsequent or prior to the completion of this public health assessment. The purpose of the PHAP is to ensure that this public health assessment not only identifies public health hazards, but also provides a plan of action designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment.

Completed Actions

1. Data and information obtained from EPA and PADEP have been evaluated by the PADOH and ATSDR to determine the public health implications of human exposure pathways via all media.

2. Health Education Activities were completed as planned. These activities completed to date included:

   - Two (2) Public Meetings
   - Two (2) Public Availability Sessions
   - Home visits to discuss indoor air results
   - Four (4) Fact Sheets

3. Other completed activities are listed under the section *PADOH and ATSDR Site Visits and Meetings and Other Assessment Activities, Description of Visits and Meetings* on

page 4 in this PHA. Included is the June 2004 informational meeting sponsored by ATSDR and PADOH and held during a physicians' staff meeting at the Hazleton General Hospital. Dr. Keith Burkhart, M.D. and Lora Werner presented information to the physicians relating to the chemical contamination at the Valmont TCE (Valmont Trichloroethylene) site and especially concerning any possible exposures of potential patients to TCE and 1,1,1-trichloroethane. Additionally, Dr. Burkhart is consulting with a resident(s)'s physician(s) and reviewing health records for any possible association with exposures from the Valmont TCE site.

4. PADOH, ATSDR, PADEP, and EPA have been working together to help the citizens of Western Hazleton learn about the meaning of all sample results and the remediation work on-site and off-site. EPA has completed a Remedial Investigation through Tetra Tech NUS, Inc. Much more environmental sampling has been completed since the writing of the initial version PHA in June 2002. Major remediation of contaminated soil took place on-site by EPA's contractor as a first removal action and evaluations of a possible soil vapor extraction and in-situ chemical oxidation of soils under the Chromatex building began. EPA is currently developing a Feasibility Study for the on-site and off-site ground water. This ground water treatment is expected to begin in 2006/early 2007, but the complete length of time that will be needed for an adequate groundwater cleanup action is unknown. The aquifer will be monitored during the clean up and levels should decrease over time.

Ongoing or Planned Actions

1. PADOH and ATSDR will collaboratively evaluate future site data with PADEP and EPA, if EPA continues environmental sampling and investigation of the site. Should any new data or information reveal that site conditions are worse than previously thought or have adversely changed over time, PADOH and the ATSDR may re-evaluate the conclusions and recommendations stated in this PHA.

2. PADOH and ATSDR will: a) make the final version of the Valmont TCE Site PHA available to the community (the public comment period was from July 15, 2005 to August 30, 2005 and had been extended into November 2005); b) respond to individual requests for health information; c) ATSDR's Toxicologists will remain available for consultation with physicians whose patients may have been exposed to contamination from the site; and d) provide any further needed health education to the community and health professionals.

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Acknowledgment: PADOH thanks Dr. Kandiah Sivarajah, Ph.D., State Toxicologist and Dr. Keith Burkhart, MD, ATSDR Region Medical Toxicologist for their input regarding toxicological and medical evaluations and discussions in this document.

Certification

This public health assessment for the Valmont TCE site was prepared by the PADOH under a cooperative agreement with ATSDR. It is in accordance with approved methodology and procedures existing at the time the health consultation were initiated. Editorial review was completed by the cooperative agreement partner.

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

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APPENDIX A. Figures

Figure 1 – Valmont TCE Site Location in Pennsylvania

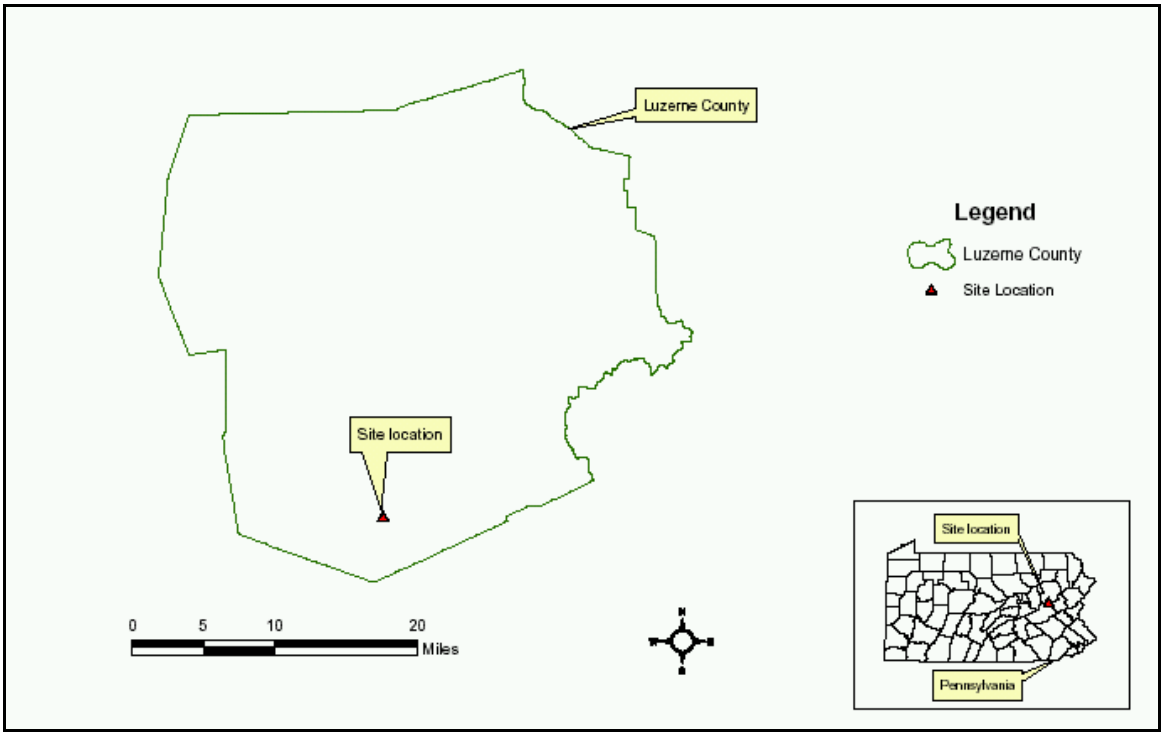
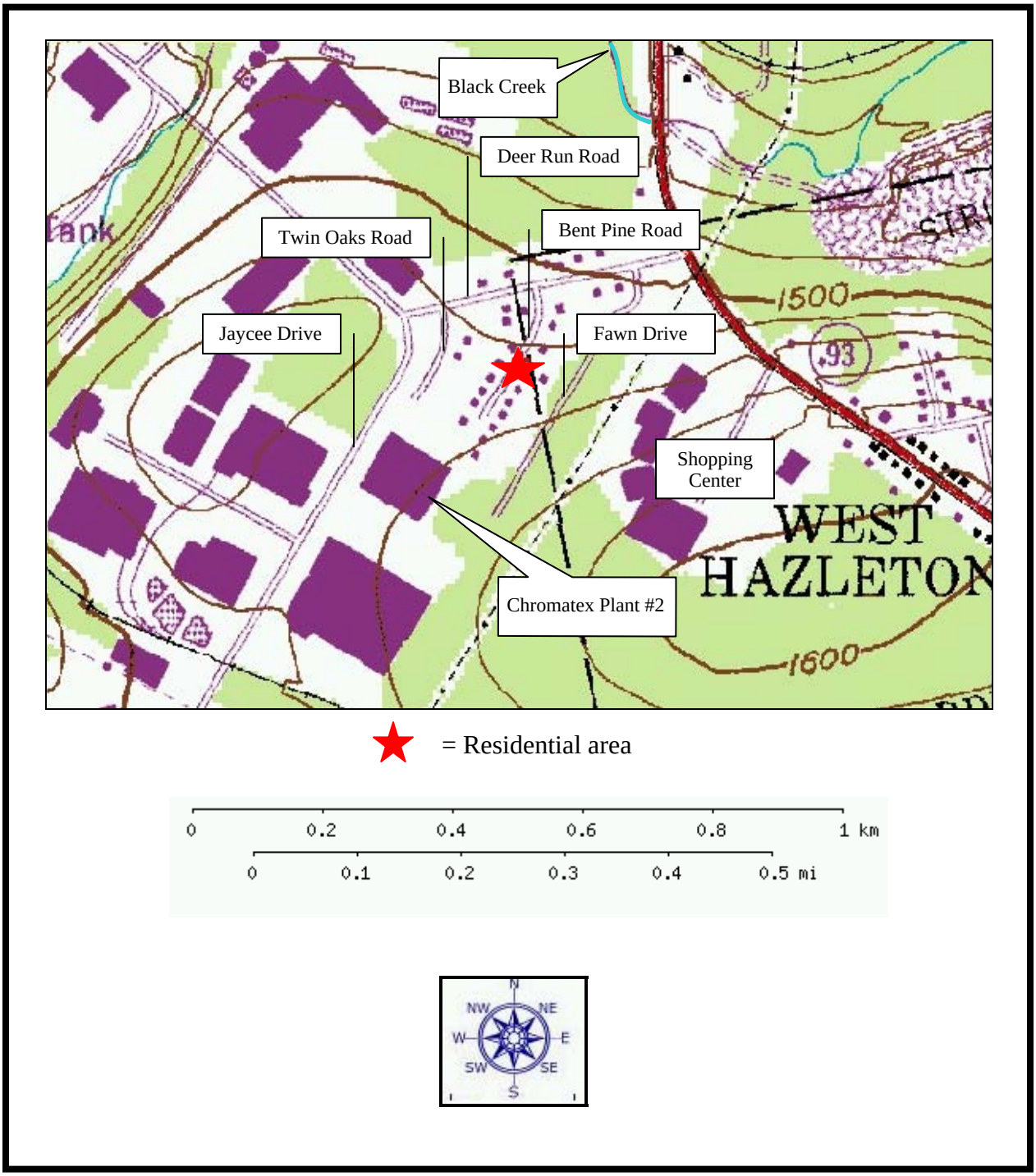


Figure 2 – Valmont TCE Site Location Map and Residential Area



APPENDIX B. Tables

Table 1. Table from the Residential Indoor Air Health Consultation of November 18, 2002, *"Common uses/sources of selected VOCs detected at the Valmont TCE Site"*

CHEMICAL	*COMMON USES/SOURCES*
2-Hexanone	Used in the past in paint and paint thinner, to make other chemical substances, and to dissolve oils and waxes.
4-Ethyltoluene	Used as a solvent; kerosene; light oil vapor
1,1,1-Trichloroethane	Dry-cleaned clothes,pesticides,some household cleaners,spray can propellants.
1,1,2,2-Tetrachloroethane	Used in large amounts to produce other chemicals and as a solvent, to clean and degrease metals, and in paints and pesticides. Presently is used only as a chemical intermediate in the production of other chemicals.
1,2-dichloro-1,1,2,2-tetrafluoroethane (Freon 114)	Used in nonflammable aerosol propellants; as a refrigerant in industrial cooling and air conditioning systems. Used as a solvent, diluent, and degreaser in the electronics and chemical industries; as a blowing agent in manufacture of cellular polymers; and as an extractant for volatile substances. Used in the manufacture of explosives, as a component of dielectric fluid, and as a foaming agent in fire extinguishers.
1,1,2-Trichloro-1,2,2-trifluoroethane (Freon 113)	Generally used as a refrigerant, a dry cleaning solvent and an intermediate.
1,2,4-Trichlorobenzene	Used as a dye carrier, a herbicide intermediate, a heat-transfer medium, a dielectric fluid in transformers, a degreaser, a lubricant, in synthetic transformer oils, and as a solvent in chemical manufacturing. Formerly used as an insecticide against termites.
1,2,4-Trimethylbenzene	Used to make dyes and drugs. Gasoline or certain paints and cleaners.
1,1-Dichloroethane	In the past, used as a surgical anesthetic. Today it is used primarily to make other chemicals, to dissolve substances such as paint, varnish, and finish removers, and to remove grease.
1,1-Dichloroethene	1,1-Dichloroethene is used to make certain plastics, such as flexible films like food wrap, and in packaging materials. It is also used to make flame retardant coatings for fiber and carpet backings, and in piping, coating for steel pipes, and in adhesive applications.
1,2-Dichlorobenzene	Used as an industrial solvent and chemical intermedite. Used in pesticides, insecticide/fumigant,solvent for waxes, gums, resins, tars, rubbers, oils and asphalts, and degreaser for metals (engines), etc.
1,3-Butadiene	A chemical made from the processing of petroleum. Used to make synthetic rubber and plastics including acrylics. Small amounts are found in gasoline.
1,3-Dichlorobenzene	Environmental contamination results from emissions to air and water during the manufacture and use of the chlorinated benzenes and from the disposal of wastes from a number of processes
1,4-Dichlorobenzene	In moth repellent products and in toilet deodorizer blocks.
1,4-Dioxane	Used as a solvent in paints, varnishes, lacquers, paint and varnish removers, cosmetics, and deodorants. A solvent in the pulping of wood, fats, oils, waxes and natural and synthetic resins, and as a degreasing agent. Used as a stabilizer for chlorinated solvents such as 111-TCA.
1,3,5-Trimethylbenzene	Found in gas treatments, valve cleaners, mark/spot remover, floor wax, varnishes, paints, and pesticides. Diesel exhaust component

Table 1. Table from the Residential Indoor Air Health Consultation of November 18, 2002, *"Common uses/sources of selected VOCs detected at the Valmont TCE Site"- continued.*	
Benzene	Used to make other chemicals, which are used to make plastics, resins, and nylon and synthetic fibers. Also used to make some types of rubbers, lubricants, dyes, detergents, drugs, and pesticides. Natural sources of benzene include volcanoes and forest fires. Benzene is also a natural part of crude oil, gasoline, and cigarette smoke.
Carbon Tetrachloride	Used in the production of refrigeration fluid and propellants for aerosol cans, as a pesticide, as a cleaning fluid and degreasing agent, in fire extinguishers, and in spot removers.
Chloroform	Used to make other chemicals and can also be formed in small amounts when chlorine is added to water.
Chlorotoluene	This compound is used as a solvent and a chemical intermediate in the manufacture of pesticides, dyes, and pharmaceuticals
cis-1,2-DCE	It is used to produce solvents and in chemical mixtures.
Cyclohexane	Found naturally to some extent in petroleum but is prepared commercially by catalytic hydrogenation of benzene. Widely used as a solvent and in making certain compounds used in the preparation of nylon.
Dichlorodifluoromethane (Freon 12)	Used as refrigerants, aerosol propellants, and solvents.
Heptane	Used when less volatile solvent is desired, as in the manufacture of certain adhesives and lacquers, and in extraction of edible and commercial oils.
Hexacholorobutadiene	It is mainly used to make rubber compounds. It is also used as a solvent, and to make lubricants, in gyroscopes, as a heat transfer liquid, and as a hydraulic fluid.
m,p-Xylene	Used as a solvent and in the printing, rubber, and leather industries. Also used as a cleaning agent, a thinner for paint, and in paints and varnishes. Found in small amounts in airplane fuel and gasoline.
Methylene Chloride	Used as an industrial solvent and as a paint stripper. It may also be found in some aerosol and pesticide products and is used in the manufacture of photographic film.
o-Xylene	Used as a solvent and in the printing, rubber, and leather industries. Also used as a cleaning agent, a thinner for paint, and in paints and varnishes. Found in small amounts in airplane fuel and gasoline.
Tetrachloroethene or perchloroethylene (PCE)	Used in dry cleaning solutions and metal degreasers.
Tetrahydrofuran	Used in the fabrication of articles for packaging, transporting, and storing of foods; as a solvent for dyes and lacquers; and as a chemical intermediate in polymerization solvent for fat oils, unvulcanized rubber, resins, and plastics. Also an indirect food additive when it is in the contact surface of articles intended for use in food processing.
Trichloroethylene	Used in home and auto cleanres, adhesives, tape, spot removers, cosmetics, insulation, photographic equipment, opaquing fluid, and typewriter correction fluid.
Trichlorofluoromethane (Freon 11)	Used as refrigerants, aerosol propellants, and solvents.
Vinyl Chloride	Used to make polyvinyl chloride (PVC). PVC is used to make a variety of plastic products, including pipes, wire and cable coatings, and the furniture and automobile upholstery. Also results from the breakdown of other substances, such as trichloroethane, trichloroethylene, and tetrachloroethylene.

Addendum to Table 1 above and to the table from the Residential Indoor Air Health Consultation of November 18, 2002, *"Common uses/sources of selected VOCs detected at the Valmont TCE Site".*	
CHEMICAL	*COMMON USES / SOURCES*
1,2-Dichloroethane	Used as a solvent and in the production of vinyl chloride, which is used to make a variety of plastic and vinyl products including polyvinyl chloride pipes, furniture and automobile upholstery, wall coverings, housewares, and automobile parts. It is also is added to leaded gasoline to remove lead.
2-Butanone or Methyl Ethyl Ketone (MEK)	Found in paints, coatings, glues, cleaning agents, and cigarette smoke. It occurs naturally in some fruit and trees.
4-Methyl-2-pentanone	Used as a solvent. Also known as Isopropylacetone.
Acetone	Used as a common solvent, especially in laboratories.
Carbon Disulfide	Used in the manufacturing of rayon, in soil disinfectants, and in solvents.
Chloromethane	Byproduct of burning grasses, wood, cigarettes, charcoal, or plastic. Found in styrofoam insulation, aerosol propellants, and chlorinated swimming pools.
Ethylbenzene	Used as a common solvent, and found in gasoline, inks, insecticides, and paints. Also found in cigarette smoke.
Hexane	Found in petroleum products, is often mixed with other solvents, and is used as a filling for thermometers.
Isopropyl Alcohol	Found in household isopropyl alcohol, cleaners, hair coloring, pesticides, odor removers, sealants and adhesives.
Methyl Tertiary-butyl Ether (MTBE)	Used as an oxidant/additive in unleaded gasoline.
Propene	It is a flammable gas obtained from petroleum. It is found in specific brands of household dusting sheets. Also called poypropylene.
Styrene	Used in wood filler and putty, adhesives, foams, lubricants, and plastics.
Toluene	Used as degreasers and cleaners. Found in "liquid nails", enamels, varnishes, fingernail polishes, sprays, paints, glues, contact cement, adhesives, and paint removers.
Vinyl Acetate	Used in glues, chalks, and wall spackling, sealants, adhesives, and paint primers.

Table 2. Table from the *Remedial Investigation Report for Operable Unit Valmont TCE Site,* Volume II Appendixes, Appendix E, EPA Work Assignment Number 044-RICO-031M, Tetra Tech NUS Project No. 4192, RAC 3 Program Contract Number 68-S6-3003, July 2004.

Chemical	*PADEP Site History*	*Ground Water Plume**	*Indoor Air*	*Ambient Air*	*Soil Gas*	*Sewer*	*Likelihood*
Acetone			X	X	X	X	d
Benzene			X	X	X		c
1,3-Butadiene	X		X		X		b
2-Butanone			X	X	X	X	d
Carbon disulfide			X		X	X	d
Carbon tetrachloride	X	X	X		X		b
Chloroform	X	X	X		X	X	b
Chloromethane	X		X	X	X		c
Chlorotoluene			X				d
Cyclohexane			X				d
1,2-Dichlorobenzene			X				d
1,3-Dichlorobenzene			X				d
1,4-Dichlorobenzene			X	X		X	d
Dichlorodifluoromethane (freon 12)			X	X	X		d
1,1-Dichloroethane	X	X	X				a
1,2-Dichloroethane					X		b^
1,1-Dichloroethene	X	X	X	X	X		a
1,2-Dichloroethene	X	X	X		X		a
1,2-Dichloro-1,1,2,2-tetrafluoroethane			X				d
1,4-Dioxane			X		X		d
Ethanol	X		X	X	X	X	b
Ethylbenzene		X	X	X	X		b
4-Ethyltoluene			X	X			d
Heptane			X		X		d
Hexachlorobutadiene			X	X			d
Hexane			X		X		d
2-Hexanone			X				d
Methyl t-butyl ether			X		X		d

Table 2. Table from the Remedial Investigation Report, Appendix E, July 2004 – *continued.*							
Chemical	*PADEP Site History*	*Ground Water Plume**	*Indoor Air*	*Ambient Air*	*Soil Gas*	*Sewer*	*Likelihood*
Methylene chloride	X		X	X	X	X	c
2-Propanol			X	X	X		d
Styrene	X		X		X		c
1,1,2,2-Tetrachloroethane			X				b^
Tetrachloroethene (PCE)	X	X	X		X		a
Tetrahydrofuran			X	X			d
Toluene		X	X	X	X	X	b
1,2,4-Trichlorobenzene			X	X			d
1,1,1-Trichloroethane	X	X	X	X	X		**a**
Trichloroethylene (TCE)	X	X	X		X		**a**
Trichlorofluoromethane (Freon 11)		X	X	X	X		b
1,1,2-Trichloro-1,2,2-trifluoroethane	X	X	X				c
Trimethylbenzenes			X	X	X		c
Vinyl chloride	X	X	X		X		a
Xylenes		X	X	X	X		b

*List is minimal, representing several available rounds of data, and does not include every sampling event. Therefore, a chemical's absence from the list does not necessarily means that it is absent from ground water.

a = Chlorinated ethenes and ethanes, present in both site history and ground water, usually absent in air; considered most likely to be connected with vapor intrusion.

b = Chemicals with a possible connection to site history and/or ground water, but which have low frequency and / or concentration in ground water, and which often have other possible sources. These chemicals are considered probably unconnected with vapor intrusion from the ground water plume, but there is considerable uncertainty associated with this tentative conclusion.

c = Chemicals considered unlikely to be related to vapor intrusion, although a potential connection cannot be rule out.
d = Chemicals without a current obvious connection to site history or ground water, which are suspected to be unrelated to the ground water plume. However, some of these chemicals may have been detected in soil gas or sewers.

^ - Although 1,2-dichloroethane and 1,1,2,2-tetraethane are chlorinated ethanes, they were detected rarely and are therefore rated "b" rather than "a".

Table 3 – Past Maximum Concentrations Detected - Summary of Off-Site (Residential Wells) Ground water Data up to 2002 for Chemicals of Potential Concern (Source: Initial Version of the Valmont TCE PHA and PADEP's 2002 laboratory data for the residential wells).

Contaminant	*Sample event/year*	*Maximum Concentration Detected (ppb)*	*CV*	*Source*	*EPA Cancer Classification*	*Other Comparison*
1,1,1-Trichloroethane	1987	450	200	MCLG	D	The MCL for public drinking water is 200.
1,1-Dichloroethane (DCA)	1987	3.4	N/A	N/A	N/A	The estimated oral exposure dose is about six (6) orders of magnitude less than the NOAEL in animal studies.
	Dec 2000	2 J				
	May 2001	1.0 L				
	Nov 2002	2				
Carbon Tetrachloride	Oct–Nov 2001	9.0 J	0.03	CREG	B2	The MCL for public drinking water is 5 ppb.
Trichloroethylene (TCE)	1987	1,400	0 / Listed by ATSDR as under EPA review*	MCLG / CREG	B2	The MCL for public drinking water is 5 ppb.
	Oct 1993	592				
	Dec 2000	370 L				
	May 2001	440 L				
	Oct–Nov 2001	510				
	Nov 2002	420 E				
Vinyl chloride	Nov 2002	7	0 / 0.03	MCLG / CREG	A	The MCL for public drinking water is 2 ppb.

Note: CVs are used for "screening" chemicals for further review. A concentration that exceeds the CV does not necessarily mean that there is a health problem associated with exposures at that level (see Appendix C in this document for more information).

* Proposed CSF (based on EPA's cancer risk number) is 0.02 to 0.4 $(mg/kg/day)^{-1}$. Range of 10^{-6} C.R. is about 0.01 to 0.2 ppb.

Table 4 - Ground water Data for 2003 - Off-site and On-site maximum VOC and BNA Concentrations Detected. Only sample concentrations above the ATSDR CV are on this table. All units are in parts per billion (ppb).

Chemical	*Maximum Concentration Detected (Location)*	*CV*	*Source*	*EPA Cancer Classification*	*Other Comparison*
TCE	3700 (On-Site shallow well) 510 (Off-site intermediate-deep well)	0 / Listed by ATSDR as under EPA under review) *	MCLG / CREG *	B2	The MCL for public drinking water is 5.
1,1,1-Trichloroethane	290 (On-Site shallow well)	200	MCLG / LTHA	D	The MCL for public drinking water is 200.
cis -1, 2-DCE	110 (On-Site shallow well) 72 (Off-site intermediate-deep well)	70	MCLG / LTHA	C	The MCL for public drinking water is 70.
1,1-Dichloroethane	8.6 (On-Site) 2.6 (Off-Site)	N/A	N/A	N/A	The estimated oral exposure dose is about six (6) orders of magnitude less magnitude less than the NOAEL in animal studies.
1,1-Dichloroethene	13 (On-Site shallow well)	6	LTHA	C	The MCL for public drinking water is 200.
1,4 -Dioxane	16 (On-Site deep well) 2.5 (Off-site shallow well)	3	GREG	B2	There is no MCL for public drinking water, but there is an Action Level in Pennsylvania of 20 from the EPA Health Advisory (taste and odor).
Benzene	0.80 (On-Site intermediate / deep well)	0.6	CREG	A	The MCL for public drinking water is 5.

Note - If no off-site levels are listed then these chemicals were not detected or were below health levels.

* Proposed CSF range listed (based on EPA's cancer risk number) is 0.02 to 0.4 (mg/kg/day)-1. Range of 10^{-6} C.R. is about 0.01 to 0.2 ppb.

Table 5. On-Site Ground water (Monitoring Wells) Data Summary for Chemicals of *Potential* Concern (1988).
All units are in parts per billion (ppb). (Source: Table 1 modified from the Initial Version of the Valmont TCE PHA)

Chemical	*Maximum Concentration Detected*	*ATSDR Comparison Values*	*Source*	*EPA Cancer Classification*	*Other Comparison*
1,1,1-Trichloroethane	13,000	200	LTHA	D	The MCL for public drinking water is 200.
1,1-Dichloroethane (DCA)	370	N/A	N/A	N/A	The estimated oral exposure dose is about six (6) orders of magnitude less magnitude less than the NOAEL in animal studies.
1,1-Dichloroethene (DCE)	280	90 / 6	EMEG (child) / LTHA	C	The MCL for public drinking water is 7 ppb.
Carbon Tetrachloride	5.8	0.03	CREG	B2	The MCL for public drinking water is 5 ppb.
Cis-1, 2-Dichloroethene	1030	70	LTHA / MCL = 70	C	The MCL for public drinking water is 70 ppb.
Tetrachloroethylene (PCE)	35	5	MCL	Under EPA review	The MCL for public drinking water is 5 ppb.
Trichloroethylene	17,000	0 / Listed by ATSDR as under EPA review*	MCLG / CREG	B2	The MCL for public drinking water is 5 ppb.

* Proposed CSF (based on EPA's cancer risk number) is 0.02 to 0.4 $(mg/kg/day)^{-1}$. Range of 10^{-6} C.R. is about 0.01 to 0.2 ppb.

Table 6. Indoor Air Sample Results. This table was taken from the Health Consultation; Evaluation of Residential Indoor Air Samples published November 18, 2002.
Summary of Data for Selected VOCs From Indoor Air Samples (Basement or first floor samples).
Valmont TCE Site, May 2001 – February 2002

Contaminant	*Sampling Event*	*Frequency of Detection (homes)*	*Concentrations Detected in ppb (ug/m3)*	*ATSDR Comparison Values*	
				Value	*Source*
1,1,2,2-Tetrachloroethane	May- June 2001	0/24	ND	0.02 ug/m3	CREG
	Oct–Nov 2001	1/8	0.26 (1.78)	400 ppb	MRL (I)
	Feb 2002	0/28	ND		
1,1-Dichloroethane	May- June 2001	0/24	ND	n/a	n/a
	Oct–Nov 2001	1/8	0.053 (0.21)	n/a	n/a
	Feb 2002	0/28	ND	n/a	n/a
1,1-Dichloroethene	May- June 2001	0/24	ND	0.02 ug/m3*	CREG
	Oct–Nov 2001	1/8	1.9 (7.53)	20 ppb	MRL (I)
	Feb 2002	0/28	ND	200 ug/m3	RFC
1,2-Dichlorobenzene	May- June 2001	0/24	ND	n/a	n/a
	Oct–Nov 2001	1/8	0.37 (2.2)	n/a	n/a
	Feb 2002	0/28	ND	n/a	n/a
1,2,4-Trichlorobenzene	May- June 2001	0/24	ND	n/a	n/a
	Oct–Nov 2001	2/8	0.27-0.8 (2.0 - 5.9)	n/a	n/a
	Feb 2002	1/28	0.38 (2.82)	n/a	n/a

* Currently, the ATSDR CV (EPA CREG) for 1,1–dichloroethene of 0.02 ug/m3 has been withdrawn and is under review; EPA considers the chemical to be a possible human carcinogen while ATSDR is considering only suggestive evidence for carcinogenicity via human or animal data.

Table 6 - continued

1,2,4-Trimethylbenzene	May- June 2001	0/24	NT	n/a	n/a
	Oct–Nov 2001	6/8	0.77-4.1(3.79-20.16)	n/a	n/a
	Feb 2002	21/28	0.3-14 (1.47-71.00)	n/a	n/a
1,1,2-Trichloro-1, 2,2-trifluoroethane (Freon 113)	May- June 2001	6/24	0.6-5.1 (4.6-39.09)	n/a	n/a
	Oct–Nov 2001	0/8	ND	n/a	n/a
	Feb 2002	0/28	ND	n/a	n/a
1,2-dichloro-1, 1,2,2-tetrafluoroethane (Freon 114)	May-June 2001	1/24	2.2 (15.38)	n/a	n/a
	Oct–Nov 2001	1/8	5.2-5.3 (36.35-37.05)	n/a	n/a
	Feb 2002	1/28	2.4-2.9 (16.78-20.27)	n/a	n/a
1,3,5-Trimethylbenzene	May-June 2001	0/24	NT	n/a	n/a
	Oct–Nov 2001	5/8	0.26-0.91 (1.31-4.6)	n/a	n/a
	Feb 2002	8/28	0.4-10 (1.97-49.16)	n/a	n/a
1,3-Butadiene	May-June 2001	0/24	NT	0.004 ug/m3[†]	CREG
	Oct–Nov 2001	1/8	1.5 (3.32)		
	Feb 2002	0/28	NT		
1,3-Dichlorobenzene	May-June 2001	0/24	ND	n/a	n/a
	Oct–Nov 2001	1/8	0.27 (1.62)	n/a	n/a
	Feb 2002	0/28	ND	n/a	n/a
1,4-Dichlorobenzene	May-June 2001	4/24	1.20-34.00 (7.21-204.42)	100 ppb	MRL (C)
	Oct–Nov 2001	8/8	0.05–240.00 (0.28-1,443.04)	200 ppb	MRL (I)
	Feb 2002	5/28	0.44-170.00 (2.65-1,022.16)	800 ppb	RFC

[†] Currently, the ATSDR CV (CREG) for 1,3-butadiene of 0.004 ug/m3 was withdrawn and has been replaced by 0.03 ug/m3.

Table 6 - continued

1,4-Dioxane	May-June 2001	0/24	NT	n/a	n/a
	Oct–Nov 2001	2/8	5.60-6.80 (20.18-24.50)	n/a	n/a
	Feb 2002	0/28	NT	n/a	n/a
2-Hexanone	May-June 2001	0/24	ND	n/a	n/a
	Oct–Nov 2001	1/8	2.80 (1.20)	n/a	n/a
	Feb 2002	0/28	ND	n/a	n/a
2-Propanol	May-Jun 2001	0/24	NT	n/a	n/a
	Oct–Nov 2001	8/8	4.60-24.00 (11.31-58.99)	n/a	n/a
	Feb 2002	0/28	NT	n/a	n/a
4-Ethyltoluene	May-June 2001	0/24	NT	n/a	n/a
	Oct–Nov 2001	4/8	1.30-3.20 (6.39-15.73)	n/a	n/a
	Feb 2002	9/28	0.90-12.00 (4.42-58.90)	n/a	n/a
Benzene	May 2001	0/24	NT	0.1 ug/m3	CREG
	Oct–Nov 2001	7/8	0.12–3.20 (0.38-10.22)	4 ppb	MRL (I)
	Feb 2002	27/28	0.28-10.00 (0.89-31.95)		
Carbon Tetrachloride	May 2001	1/24	1.60 J (10.07)	0.07 ug/m3	CREG
	Oct–Nov 2001	5/8	0.05–0.17 (0.31-1.07)	50 ppb‡	MRL (I) ‡
	Feb 2002	0/28	ND		
Chloroform	May 2001	2/24	1.00 J–1.20 (4.88-5.86) J	0.04 ug/m3	CREG
	Oct–Nov 2001	5/8	0.22–0.75 (1.07-3.66)	20 ppb	MRL (C)
	Feb 2002	6/28	0.27-0.84 (1.32-4.10)		

‡ Currently, the ATSDR CV (MRL-intermediate) for carbon tetrachloride of 50 ppb was withdrawn and has been replaced by 30 ppb (MRL- chronic).

Table 6 - continued

Chlorotoluene	May 2001	0/24	NT	n/a	n/a
	Oct–Nov 2001	1/8	0.34 (1.79)	n/a	n/a
	Feb 2002	0/28	NT	n/a	n/a
cis-1,2-DCE	May 2001	1/24	4.70 (18.63) J	n/a	n/a
	Oct–Nov 2001	0/8	ND		
	Feb 2002	0/28	ND		
Cyclohexane	May 2001	0/24	NT	n/a[§]	n/a[§]
	Oct–Nov 2001	1/8	2.20 (7.70)	n/a	n/a
	Feb 2002	0/28	NT	n/a	n/a
Dichlorodifluoromethane (Freon 12)	May-Jun 2001	10/24	0.50-15.00 (2.47-74.19)	n/a	n/a
	Oct–Nov 2001	8/8	0.45-23.00 (2.23-113.76)	n/a	n/a
	Feb 2002	18/28	0.35-9.70 (1.73-47.98)	n/a	n/a
Ethanol	May-June 2001	0/24	NT	n/a	n/a
	Oct–Nov 2001	8/8	19.00-2,400.00 (92.33-4,522.21)	n/a	n/a
	Feb 2002	0/28	NT	n/a	n/a
Heptane	May-Jun 2001	0/24	NT	n/a	n/a
	Oct–Nov 2001	3/8	1.60-2.20 (6.56-9.02)	n/a	n/a
	Feb 2002	0/28	NT	n/a	n/a
Hexacholorobutadiene	May-June 2001	0/24	ND	0.05	CREG
	Oct–Nov 2001	1/8	0.20 (2.17)		
	Feb 2002	0/28	ND		

[§] Currently, the RFC listed in the ATSDR CVs for cyclohexane is 6000 ug/m3.

Table 6 - continued

m,p-Xylene	May-Jun 2001	0/24	NT	n/a□	n/a□
	Oct–Nov 2001	8/8	0.14-13.00 (0.61-56.45)	n/a	n/a
	Feb 2002	26/28	0.56-51.00 (2.43-220.00)	n/a	n/a
Methylene Chloride	May-Jun 2001	19/24	1.00–31.00 (3.47-107.69)	3 ug/m3	CREG
	Oct–Nov 2001	4/8	0.21–5.70 (0.73-19.80)	300 ppb	MRL (C)
	Feb 2002	19/28	0.24 - 9.00 (0.83-31.26)	300 ppb	MRL (I)
o-Xylene	May-Jun 2001	0/24	NT	n/a□	n/a□
	Oct–Nov 2001	6/8	0.54-4.20 (2.34-18.24)	n/a	n/a
	Feb 2002	18/28	0.47-14.00 (2.04-59.00)	n/a	n/a
Tetrahydrofuran	May-June 2001	0/24	NT	n/a	n/a
	Oct–Nov 2001	3/8	1.50-7.80 (4.42-23.00)	n/a	n/a
	Feb 2002	0/28	NT	n/a	n/a
Trichlorofluoromethane (Freon 11)	May-Jun 2001	13/24	0.50-13.00 (2.81-73.04)	n/a	n/a
	Oct–Nov 2001	7/8	0.40-6.50 (2.25-36.52)	n/a	n/a
	Feb 2002	20/28	0.28-24.00 (1.57-134.85)	n/a	n/a
Vinyl Chloride	May-June 2001	1/24	1.00 (2.56)	0.1	CREG
	Oct–Nov 2001	0/8	ND		
	Feb 2002	0/28	ND		

□ Currently, the ATSDR CV (MRL-chronic EMEG) for ***total*** xylenes is 100 ppb.

KEY FOR TABLES 1 – 6:
ppb = parts per billion
ppm = parts per million or milligrams per kilogram
J = Analyte present. Reported value may not be accurate or precise
N/A = not available
L= Analyte present. Reported value might be biased low. Actual value is expected to be higher.
E = Compound was above the calibration range
MCLG = maximum contaminant level goal for public drinking water systems (EPA).
MCL = maximum contaminant level for public drinking water systems (EPA).
MRL (C)= Chronic Minimal Risk Level
MRL (I)= Intermediate Minimal Risk Level
CREG = Cancer Risk Evaluation Guide
RFC = Risk Factor Concentration
ND = Non-Detect; not detected
NT = Samples not tested for the chemical in question
EMEG (I) = Environmental Media Evaluation Guide, Intermediate (15-365 days of exposure)
EMEG (C) Child = Environmental Media Evaluation Guide, Chronic (> 365 days of exposure)
RMEG = Reference Dose Media Evaluation Guide

TABLE 7. SUMMARY CANCER AND NON-CANCER RISKS - INDOOR AIR, VALMONT TCE SITE, W. HAZLETON, PENNSYLVANIA.
Source: Remedial Investigation Report for Operable Unit Valmont TCE Site, Volume I Section 6, EPA Work Assignment Number 044-RICO-031M, Tetra Tech NUS Project No. 4192, RAC 3 Program Contract Number 68-S6-3003, July 2004.
Shaded residences were provided air filtration units and/or sump covers by EPA.

Residence	*Adult Resident*		*Child Resident*		*Lifetime Resident*	*Comments*
	Carcinogenic	*Noncancerous*	*Carcinogenic*	*Noncancerous*	*Carcinogenic*	
1	3.80E-04	1 28E+01	2.65E-04	3.86E+01	6.45E-04	RME ICR > E- 04; RME HI > 1
2	1.64E-04	1.01E+01	1.14E-04	2.84E+01	2.78E-04	*RME ICR > E-04; RME HI > 1*
3	1.89E-05	2.87E-01	1.27E-05	8.03E-01	3.18E-05	
4	NA	2.40E+00	NA	6.60E+00	5.00E-05	*RME HI > 1*
6	NA	3.00E-01	NA	3.00E-01	2.00E-05	
8	NA	2.00E+00	NA	2.00E+00	2.00E-04	RME ICR > E-04
9	4.20E-04	5.85E+00	3.00E-04	1.68E+01	7.20E-04	RME ICR > E-04; RME HI > 1
10	NA	4.00E+00	NA	7.40E+00	9.00E-05	*RME HI > 1*
13	7.50E-06	3.80E-02	5.30E-06	1.06E-01	1.28E-05	
14	NA	1.00E+00	NA	1.00E+00	3.00E-04	RME ICR > E-04
15	NA	5.00E+01	NA	5.00E+01	9.00E-04	RME ICR > E-04; RME HI > I
16	NA	2.00E+00	NA	2.00E+00	3.00E-05	The residential child HI was not >1 when target organs were considered.
17	NA	8.00E+00	NA	8.00E+00	2.00E-04	*RME ICR > E-04*
21	NA	9.00E+00	NA	2.50E+01	4.20E-05	*RME HI > 1*
22	3.14E-03	5.70E+00	2.23E-03	1.60E+01	5.36E-03	*RME ICR > E-04; RME HI > 1*
23	NA	3.00E-01	NA	3.00E-01	4.00E-04	RME ICR > E-04
24	NA	2.80E+00	NA	8.20E+00	2.00E-04	RME HI > 1
25	NA	1.70E+00	NA	4.80E+00	3.20E-05	*RME H1 > 1*
26	NA	2.00E+00	NA	2.00E+00	1.00E-04	*RME ICR > E-04; RME HI > 1*
27	NA	4.00E-01	NA	4.00E-01	7.00E-04	RME ICR > E-04
28	1.64E-04	2.35E+0l	1.17E-04	6.58E+01	2.81E-04	RME ICR >E-04; RME HI > 1
34	NA	3.60E+00	NA	1.05E+01	8.00E-05	*RME HI >1*
35	NA	3.00E-01	NA	3.00E-01	5.00E-05	
36	3.78E-05	1.46E-01	2.69E-05	4.09E+01	6.47E-05	*RME HI > 1*
37+	6.38E-04	1.19E+01	4.32E-04	3.30E+01	1.07E-03	RME ICR > E-04; RME > 1
38	NA	3.00E+00	NA	1.07E+01	5.00E-05	*RME HI > 1*
39	NA	8.90E+00	NA	2.48E+01	4.40E-05	*RME HI > 1*

TABLE 7. SUMMARY CANCER AND NON-CANCER RISKS - INDOOR AIR, VALMONT TCE SITE, W. HAZLETON, PENNSYLVANIA -continued

Residence	*Adult Resident*		*Child Resident*		*Lifetime Resident*	*Comments*
	Carcinogenic	*Noncancerous*	*Carcinogenic*	*Noncancerous*	*Carcinogenic*	
40	NA	1.60E+00	NA	4.60E+00	2.00E-05	*RME HI > 1*
41	9.40E-05	2.50E+01	6.50E-05	7.0SE+01	1 .80E-04	*RME ICR > E-04; RME HI > 1*
43	1.70E-06	9.69E-03	1.20E-06	2.71E-02	2.90E-06	
45	NA	1.00E+01	NA	1.80E+01	5.00E-03	*RME ICR > E-04; RME HI > 1*
48	NA	2.00E+00	NA	6.00E+00	7.00E-05	*RME HI > 1*
49	NA	6.00E+00	NA	6.00E+00	3.00E-04	*RME ICR > E-04; RME HI > 1*
51	7.33E-05	1.87E+01	5.07E-05	5.24E+01	1.24E-04	*RME ICR > E-04; RME HI > 1*
52	NA	9.00E-01	NA	9.00E-01	1.00E-04	*RME ICR > E-04*
53	9.00E-05	1.12E1-01	6.00E-05	3.14E1-0l	1.50E-04	*RME ICR > E-04; RME HI > 1*
54	NA	5.60E+00	NA	1.60E+01	9.00E-05	*RME HI > 1*
55	NA	2.45E+01	NA	7.00E+01	2.00E-04	*RME ICR > E-04; RME HI > 1*
56	NA	9.00E+00	NA	9.00E+00	4.00E-04	RME ICR > E-04; RME HI > I
59	NA	6.00E-01	NA	6.00E-0l	8.00E-05	
60	NA	2.21E-02	NA	6.18E-02	NA	
70	8.60E-06	1.46E-01	5.80E-08	4.09E-01	1.48E-05	
90	5.30E-06	7.96E-02	3.40E-05	2.23E-01	4.30E-05	Background

Notes:
NA - Not applicable for this receptor, or not calculated.
Based on maximum indoor air detections during any one round, regardless of location (first floor or basement).
RME = Reasonable Maximum Exposure.
ICR = Incremental Cancer Risk.
HI = Hazard Index.
* - Indicates that the risks and hazards for these residences are not believed to be site-related.

+ - A final determination as to whether this is site-related or from another source or both has not been made.

Table 8 – Past and Current Cumulative Exposures and Summary for Residential Exposures to TCE and 1,1,1-Trichlorethane

Major Exposure Media / Pathway	***Evaluations Completed: Past (before 1987 and pre-bottled water and public water)***	***Evaluations Completed: October 1987 to 2004***	***Evaluations Completed: Current Situation***
Ground water:			
Drinking water	At the maximum levels for ten years exposure time, there would have been a *possible increase in noncarcinogenic health effects* and a likely *low to moderate increase in lifetime cancer risk*. This cancer risk was insignificant after public water was installed. (See the Section on Toxicological and Data Evaluation.) It was determined that there were no past *noncancerous* health concerns were for lower TCE levels and less exposure times.	The ground water posed no public health hazard for ingestion due to installation of public water.	The ground water posed no public health hazard for ingestion due to installation of public water.
Outdoor water use	Most likely there was a low to insignificant increased carcinogenic and noncancerous health risks	Insignificant increased cancer risk (TCE) or no public health hazard	Insignificant increased cancer risk (TCE) or no public health hazard
Bathing (especially showering), Cooking	In the past, most likely was cumulative to the drinking water health risks and was originally included in the drinking water cancer risk (TCE) calculation*. No public health concern.	No public health hazard due to public water installation	No public health hazard due to public water installation
Soil (soil gases)	Past off-site exposures posed no apparent public health hazards to residents (See the Section on Toxicological and Data Evaluation, Specific On-site and Off-site Contamination)	Past off-site exposures posed no apparent public health hazards to residents (See the Section on Toxicological and Data Evaluation, Specific On-site and Off-site Contamination)	Past off-site exposures posed no apparent public health hazards to residents (See the Section on Toxicological and Data Evaluation, Specific On-site and Off-site Contamination)

* - 1987 EPA Health Advisory for Trichloroethylene.

Major Exposure Media / Pathway	*Evaluations: Past (before 1987 and pre- bottled water and public water)*	*Evaluations: October 1987 to 2004*			*Evaluations: Current Situation*
Air:					
Outdoor air (while the Chromatex plant was operating)	Possible, but not likely and indeterminate past public health concerns – see the EPA Toxics Release Inventory data	No public health hazard for outdoor air inhalation due to the site (Chromatex Plant #2 was no longer using TCE; the manufacturing stopped operating in 2001)			No public health hazard (Chromatex Plant #2 was no longer using TCE; the manufacturing at this plant stopped operations in 2001)
		1987 to 2001	***2001 and 2002***	***2003***	
Residential Indoor Air (specifically, exposure resulting from the plume). Indoor air in the basement and other areas, especially around sump pumps	Unknown or indeterminate risk (no indoor air monitoring was performed before 2001)	Unknown or indeterminate public health hazard (no indoor air monitoring before 2001)	"No Adverse Health Effects" was concluded in ATSDR and PADOH's 2002 HC; EPA calculated "unacceptable risks" in some homes	EPA calculated that at least 5 homes had "unacceptable risks" due to the ground water plume	No public Health hazard due to vapor intrusion; Indoor air filters were installed in and sump pump covers were recommended by EPA and installed
Residential Indoor Air (specifically, non-plume sources of indoor air contaminants) in homes	Unknown or indeterminate risk (no indoor air monitoring was performed before 2001)	Unknown or indeterminate risk (no indoor air monitoring before 2001)	Several homes had low levels of chemicals not associated with the Valmont TCE site; this is not uncommon since household products may contain chemicals that can contribute to indoor air contamination	Several homes had low levels of chemicals not associated with the Valmont TCE site; this is not uncommon since household products may contain chemicals that can contribute to indoor air contamination	PADOH and EPA spoke with residents in 2001 and offered advice on how to reduce contributions from household products. Residents may want to review Table 1 and theTable 1 addendum in this PHA

APPENDIX C. Health Effects Evaluation Process Used by PADOH and ATSDR

ATSDR has developed a toxicological evaluation process for chemicals and exposure pathways in question at Superfund sites. This evaluation consists of two processes: a screening analysis and, at some sites, based on the results of the initial screening analysis, a weight-of-evidence analysis. The screening analysis, however, involves more than a simple comparison of one number against another. Site information is reviewed to select the substance concentrations and comparison values (CVs) that best represent site and exposure conditions. Typically, selecting the maximum detected substances concentrations and the lowest available CVs is used to screen the data. However, an evaluation may also be refined so that the analysis reflects more realistic exposure scenarios. During this selection process, an assessor should be mindful of community concerns, health outcomes of interest, the characteristics of potentially exposed populations, and possible exposures to multiple chemicals and/or pathways.

CVs are concentrations or doses that are conservatively derived (i.e., with many uncertainty or safety factors applied) based on the health effects literature and are below the levels associated with adverse health effects. CVs are used to assess voluminous data sets in an efficient and consistent manner during the screening analysis. They enable identification of substances that are not expected to result in adverse health effects (i.e., substances detected below CVs) and substances requiring further evaluation (i.e., substances detected above CVs). CVs should not be used to predict adverse health effects or to set cleanup levels at a site. These values serve only as guidelines to provide an initial screen of human exposure to substances. ATSDR has developed two types of CVS: health guidelines and environmental guidelines.

Health guidelines generally represent doses of a substance, usually expressed as milligrams of a substance per kilogram of body weight per day (mg/kg/day). For air exposures, the health guidelines are expressed as exposure concentrations (usually in parts per billion [ppb] or micrograms per cubic meter [ug/m3]). Health guidelines are protective of human health and are developed for both noncarcinogenic and carcinogenic effects. Health guidelines for noncarcinogenic effects are derived from human or experimental animal data and modified, as necessary, by a series of "uncertainty" factors (also known as safety factors) that ensure that guidelines are set at levels safely below those that could result in adverse health effects. Health guidelines for cancer are derived by the EPA and represent hypothetical estimates of cancer risk at low levels of exposure. Health guidelines are available for specific routes of exposure, such as ingestion and inhalation. No CVs have been established for dermal contact exposures.

ATSDR and EPA have developed health-driven CVs for noncarcinogenic effects resulting from substance exposures. Minimal Risk Levels (MRLs) are the health guidelines derived by ATSDR. Reference doses (RfDs) and reference concentrations (RfCs) are the health guidelines derived by EPA. In addition, EPA has derived factors to measure the relative potency of various carcinogens (known as cancer slope factors or CSFs and unit risk values for oral and inhalation exposures, respectively).

ATSDR and others (e.g., EPA, state governments, the World Health Organization) derive CVs for substances for which adequate data regarding time periods of exposure and routes of exposure are available. CVs are generally available for three specified exposure periods: acute (14 days or less), intermediate (15 to 365 days), and chronic (more than 365 days). CVs are also generally available for two

exposure routes: ingestion (soil and water) and inhalation. Usually CVs are available for many, but not always all substances found at a site. When CVs are available for a substance, the screening analysis is used. When no CVs are available, the data for the contaminant is generally retained for further evaluation. Exceptions exist, however. For example, essential nutrients (e.g., calcium, iron, magnesium) might only be harmful at very high concentrations or doses and would not necessarily be retained for further analysis. During the assessment it may be helpful to compare these and other naturally occurring elements to background concentrations. In selecting environmental guidelines for screening, the assessor should also consider several issues beyond which value is lowest. Consideration should also be given to e*xposure duration, site-specific conditions, and toxicological equivalency of specific chemicals.*

ATSDR has developed environmental guidelines for substances in drinking water, soil, and air. These guidelines include environmental media evaluation guidelines (EMEGs), cancer risk evaluation guidelines (CREGs), and reference dose media evaluation guidelines (RMEGs). ATSDR sometimes uses these EPA-generated CSFs to derive CREGs. CREGs are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million (10^{-6}) persons exposed during their lifetime (70 years). ATSDR's CREGs are calculated from EPA's cancer slope factors (CSFs) for oral exposures or unit risk values for inhalation exposures. These values are based on EPA evaluations and assumptions about hypothetical cancer risks at low levels of exposure.

To meet their unique mandates, other government agencies, such as EPA, the Food and Drug Administration, and state and tribal environmental and health departments, have developed their own CVs. These other CVs may address hazardous substances in water, soil, air, fish, or other biota. Because the mandates of other agencies may not always be strictly health-driven or consistent with the concerns of Superfund sites, fully understanding the derivation, uncertainties, and possible limitations of a comparison value is key to determining its appropriateness for use in the public health assessment process. Understanding the derivation of a particular comparison value is more important during the weight-of-evidence analysis when evaluating the possible public health significance of exceeding that value.

When RfDs and MRLs are not available, and to estimate chronic health guideline doses below which no adverse health effects (noncancerous) are expected, no observed adverse effect levels (NOAELs) and lowest observed adverse effect levels (LOAELs) are often used where there are recognized studies. Greatest weight is put on human or primate chronic exposure studies, if available. One approach is the use of margin of safety (MOS) analysis based on LOAELs. In general, when the MOS is greater than 1000, harmful effects are not expected. When the MOS ranges from approximately 100 to 1000, further toxicological evaluation is needed. If the MOS is less than 10, harmful effects might be possible, but further toxicological evaluation might still be advisable.

APPENDIX D. EPA's Research on Down's Syndrome

By Jennifer Hubbard, EPA Region 3 Toxicologist – December 2000

Background: Down's syndrome

Down's syndrome (also known as "Down syndrome") is most commonly associated with trisomy-21 (three copies of chromosome 21 instead of two). This occurs most often when chromosome pairs fail to separate ("nondisjunction") during the cell division known as meiosis.

Meiosis occurs as part of the developmental process for egg and sperm cells. Meiosis produces cells with one from each pair of chromosomes (resulting in half the total number), so that both parents' chromosomes are needed (as when the egg is fertilized) for the full set of paired chromosomes. In this way, meiosis differs from the regular cell division known as mitosis, which directly produces daughter cells that have the full number of chromosomes.

DNA analyses reported within the last decade show that trisomy-21 can originate from either the mother's or father's cells, and during meiotic or mitotic cell division, although most of the nondisjunctions occur during maternal meiosis division I.[1] "Mosaicism" can occur when a fertilized egg, with the usual number of chromosomes, yields some cells with trisomy due to nondisjunction during mitosis. In mosaicism, the individual will have some cells with the usual number of chromosomes and some with trisomy-21.

Although Down's syndrome has therefore been linked with chromosomal nondisjunction, it is not clear what causes the nondisjunction. Down's syndrome is reported to occur in about 1 of every 700 live births.[2]

Possible Causes of Down's Syndrome

Aside from a strong association between maternal age and trisomy-21, which has been known for years, specific causes of trisomy-21 are not known. Demonstrating that environmental chemicals can cause unusual numbers of chromosomes in people has been elusive.[3] It must be stated that even if a particular chemical showed an association with trisomy-21, it would be difficult to determine whether an individual case was caused by that chemical. For this reason, scientists usually speak in terms of associations and likelihoods rather than definite direct causes.

So far, the only chemical that has been allegedly linked to Down's syndrome in people is an insecticide called trichlorfon. In 1989-1990, a registry indicated an unusual incidence of Down's syndrome in a small Hungarian village. The use of trichlorfon at nearby fish farms (which produced fish eaten by the mothers) was suspected. After use of trichlorfon was stopped, no more cases of Down's syndrome were reported.[4] Mouse egg cells also reportedly showed disturbance of meiosis after exposure to trichlorfon in the laboratory.[5]

The family of chemicals of interest at the Valmont TCE site consists mainly of chlorinated hydrocarbons, especially trichloroethene and its related and breakdown products (including tetrachloroethene, dichloroethenes, vinyl chloride, trichloroethanes, dichloroethanes, and chloroethane, some of which have been found in the neighborhood ground water). Some of these chemicals have been tested for their association with chromosomal nondisjunction in the laboratory, and have been found to interfere to varying degrees with regular cell division in fungi.[6,7]

Of course, it is difficult to go from identifying chromosomal effects in fungi during mitosis in the laboratory, to answering the question of whether these chemicals can affect meiosis in people. As stated in a basic toxicology text, "Although these fungal assays yield valuable information and are relatively simple to perform, the importance of examining mammalian cells should be emphasized because the mechanisms of nondisjunction, and therefore the response to a given chemical, may differ between fungi and humans."[8] There are also differences between mitosis and meiosis.[9] Furthermore, nondisjunction may be chromosome-specific; a factor that influences nondisjunction of one particular chromosome may not necessarily influence nondisjunction of another.[10]

There are fewer available studies in mammals. One study found that mice, injected with chloral hydrate (a metabolite of trichloroethene), exhibited chromosomal nondisjunction during the development of sperm cells.[11] However, the demonstration of this effect in sperm cells does not necessarily mean that it also occurs in egg cells, where the majority of Down's syndrome cases are believed to originate.

Summary

In short, the question of whether chlorinated hydrocarbons like trichloroethene can cause Down's syndrome in people cannot be answered with a simple "yes" or "no." Scientists are really only beginning to look at possible connections between environmental chemicals and Down's syndrome.

References for Appendix D

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APPENDIX E. ATSDR Plain Language Glossary of Environmental Health Terms

Absorption: How a chemical enters a person's blood after the chemical has been swallowed, has come into contact with the skin, or has been breathed in.

Acute Exposure: Contact with a chemical that happens once or only for a limited period of time. ATSDR defines acute exposures as those that might last up to 14 days.

Adverse Health Effect: A change in body function or the structures of cells that can lead to disease or health problems.

ATSDR: The Agency for Toxic Substances and Disease Registry. ATSDR is a federal health agency in Atlanta that deals with hazardous substance and waste site issues. ATSDR gives people information about harmful chemicals in their environment and tells people how to protect themselves from coming into contact with those chemicals.

Background Level: An average or expected amount of a chemical in a specific environment; or, amounts of chemicals that occur naturally in a specific environment.

Cancer: A group of diseases that occur when cells in the body become abnormal and grow, or multiply, out of control.

Cancer Slope Factor: An upper bound, approximating a 95% confidence limit, on the increased cancer risk from a lifetime exposure to an agent. This estimate, usually expressed in units of proportion (of a population) affected per mg/kg/day, is generally reserved for use in the low-dose region of the dose-response relationship, that is, for exposures corresponding to risks less than 1 in 100.

Carcinogen: Any substance shown to cause tumors or cancer.

Carcinogenicity: Ability of a substance to cause cancer.

CERCLA: Comprehensive Environmental Response, Compensation, and Liability Act, also known as Superfund.

CERCLIS: Comprehensive Environmental Response, Compensation, and Liability Information System, CERCLA files.

Chronic Exposure: A contact with a substance or chemical that happens over a long period of time. ATSDR considers exposures of more than 1 year to be chronic.

Completed Exposure Pathway: (See Exposure Pathway) In completed exposure pathways, the five elements exist, and so exposure has occurred, is occurring, or will occur.

Comparison Value: (CV) Concentrations or the amount of substances in air, water, food, and soil that are unlikely, upon exposure, to cause adverse health effects. Comparison values are used by health assessors to select which substances and environmental media (air, water, food, and soil) need additional evaluation while health concerns or effects are investigated.

Concern: A belief or worry that chemicals in the environment might cause harm to people.

Concentration: How much or the amount of a substance present in a certain amount of soil, water, air, or food.

Dermal Contact: A chemical getting onto your skin.

Dose: The amount of a substance to which a person might be exposed, usually on a daily basis. Dose is often explained as (amount of substances(s) per body weight per day.(

Dose/Response: The relationship between the amount of exposure (dose) and the change in body function or health that results.

Duration: The amount of time (days, months, and years) that a person is exposed to a chemical.

Eliminated Exposure Pathway: (See Exposure Pathway) An exposure pathway can be eliminated if at least one of the five elements is missing and will never be present.

Environmental Contaminant: A substance (chemical) that gets into a system (person, animal, or the environment) in amounts higher than that found in Background Level.

Environmental Media: Usually refers to the air, water, and soil in which chemicals of interest are found. Sometimes refers to the plants and animals that are eaten by humans. Environmental Media is the second part of an Exposure Pathway.

U. S. Environmental Protection Agency (EPA): The federal agency that develops and enforces environmental laws to protect the environment and the public's health.

Exposure: Coming into contact with a chemical substance. For the three ways people can come into contact with substances, see Route of Exposure.

Exposure Pathway: A description of the way that a chemical moves from its source (where it began) to where and how people can come into contact with (or get exposed to) the chemical.

ATSDR defines an exposure pathway as having five parts:
1. Source of contamination;
2. Environmental media and transport mechanism;
3. Point of exposure;
4. Route of exposure, and;
5. Receptor population.

When all five parts of an exposure pathway are present, it is called a Completed Exposure Pathway Each of these five terms is defined in this Glossary.

Frequency: How often a person is exposed to a chemical over time; for example, every day, once a week, twice a month.

Hazardous Waste: By-products of society that can pose a substantial or potential hazard to human health or the environment when improperly managed. Possesses at least one of four characteristics (ignitability, corrosivity, reactivity or toxicity), or appears on special EPA lists.

Health Effect: See definition in this Glossary for Adverse Health Effects.

Indeterminate Public Health Hazard: This category is used for sites when a professional judgment on the level of health hazard cannot be made because information critical to such a decision is lacking.

Ingestion: Swallowing something, as in eating or drinking. It is a way a chemical can enter your body.

Inhalation: Breathing. It is a way a chemical can enter your body. (See Route of Exposure.)

LOAEL: Lowest Observed Adverse Effect Level. The lowest dose of a chemical in a study, or group of studies, that has caused harmful health effects in people or animals.

MRL: Minimal Risk Level. An estimate of daily human exposure-by a specified route and length of time-to a dose of chemical that is likely to be without a measurable risk of adverse, noncancerous effects. An MRL should not be used as a predictor of adverse health effects.

NOAEL: No Observed Adverse Effect Level. The highest dose of a chemical in a study, or group of studies, that did not cause harmful health effects in people or animals.

No Apparent Public Health Hazard: This category is used for sites where human exposure to contaminated media might be occurring, might have occurred in the past, and/or might occur in the future, but the exposure is not expected to cause any adverse health effects.

No Public Health Hazard: This category is used for sites that, because of the absence of exposure, do NOT pose a public health hazard.

NPL: The National Priorities List (part of Superfund). A list kept by the U.S. Environmental Protection Agency (EPA) of the most serious, uncontrolled, or abandoned hazardous waste sites in the country. An NPL site may need to be cleaned-up-or is being looked at to see if people can be exposed to chemicals from the site.

Plume: A volume of air or water containing chemicals that has moved and might continue to move from the source to areas further away. A plume can be a column or clouds of smoke from a chimney, contaminated underground water, or contaminated surface water (such as lakes, ponds, and streams). A plume that has stabilized (boundaries unchanging with time) is said to be in “steady state.”

Point of Exposure (exposure point): This is the specific location where people might come into contact with a contaminated medium.

Population: A group of people living in a certain area; or the number of people in a certain area.

Potential exposure pathways: (See Exposure Pathway.) Those pathways where at least one of the five elements is missing, and exposure to a contaminant could have occurred in the past or could occur in the future.

Public Health Hazard: The category used in Public Health Assessments (PHAs) for sites that have certain physical features or evidence of chronic, site-related chemical exposure that could result in adverse health effects. 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.

Public Health Hazard Criteria: PHA categories given to a site that tell whether people could be harmed by conditions present at the site. Each are defined in the Glossary. The categories are:

. Urgent Public Health Hazard,
. Public Health Hazard,
. Indeterminate Public Health Hazard,
. No Apparent Public Health Hazard, and
. No Public Health Hazard.

Reference Dose (RfD): An estimate, with safety factors (see safety factor) built in, of the daily, lifetime exposure of human populations to a possible hazard that is not likely to cause harm to the person.

Receptor Population: Potentially exposed population or population that might come or might have come in contact with contaminants.

Route of Exposure: The way a chemical can get into a person's body. There are three exposure routes:
. breathing (also called inhalation);
. eating or drinking (also called ingestion); and,
. getting something on the skin (also called dermal contact).

Safety Factor: Also called Uncertainty Factor. When scientists do not have enough information to decide if an exposure will cause harm to people, they use safety factors and formulas in place of the information that is not known. These factors and formulas can help determine the amount of a chemical that is not likely to cause harm to people.

Source (of Contamination): The place where a chemical comes from, such as a landfill, pond, creek, incinerator, tank, or drum. Contaminant source is the first part of an Exposure Pathway.

Toxic: Harmful. Any substance or chemical can be toxic at a certain dose (amount). The dose is what determines the potential harm of a chemical and whether it would cause someone to get sick.

Tumor: Abnormal growth of tissue or cells that have formed a lump or mass.

Unacceptable Risk: EPA has defined carcinogenic potential risk in excess of 1 in 10,000 and hazard indices in excess of 1.0 as unacceptable.

Urgent Public Health Hazard: This category is used for sites where short-term exposures (<1 year) to hazardous substances or conditions could result in adverse health effects that require rapid intervention.

APPENDIX F. Public Comments and Issues

A. The following are the questions/comments submitted by the citizens' group and consultant group for the Public Comment version of this PHA. The public comments are reproduced verbatim in italics below, followed by PADOH and ATSDR's responses.

1. *ATSDR states that EPA's acceptable risk level is 1/10,000 (p.20).*

   EPA's Superfund program uses an acceptable risk range of 1/10,000 to 1/1,000,000. In this PHA, we are saying the levels are …"above the EPA's acceptable risk [which is 1/10,000 to 1/1,000,000], or above the cancer risk of 1 additional cancer per 10,000 persons for prolonged exposure."

2. *Define "no apparent health hazard" – when risk levels are in the 1/1000 range it seems that there could be an apparent hazard.*

   The commenter appears to be referring to the 1/1,000 additional cancer risk range that can be calculated for indoor air exposures at this site.

   After reviewing the available weight of evidence, PADOH and ATSDR agree with EPA that the indoor air data from the homes at this site does support evidence of vapor intrusion from the contaminated groundwater at this site. However, we do not find that the levels detected in the indoor air of these homes would be high enough to produce actual health effects, thus our finding of no apparent health hazard for the current indoor air exposure pathway.

   PADOH and ATSDR's public health evaluation of these indoor air data entails a different, more qualitative approach from EPA's quantitative risk assessment methodology. Using EPA's risk assessment methodology, we concur that an "unacceptable risk" based on the EPA thresholds can be calculated for these data. The end use of these two approaches is different. EPA performs their calculations in order to determine the need for removal or remedial action at a contaminated site. ATSDR and PADOH perform our assessment in order to provide information to community members and inform regulatory partners about the likelihood of actual public health effects. Based on the available environmental data, we agree that it is an important precautionary step to mitigate the potential for continued residential vapor intrusion at this site.

3. *Explain why ATSDR finds "No Adverse Health Effects" in indoor air; but EPA calculated "unacceptable risks" (p. 67)*

   Please see the response to Comment #2 above.

4. *Reference to 10-year exposures to groundwater – what is risk for lesser time periods?*

   Please see the response to Comment #2 above. There are uncertainties in making this

kind of estimation, especially for the various combinations of shorter time periods of exposure. This is particularly the case in evaluating the possibility of an increased cancer risk, which typically involves an estimate of lifetime or 30 year residential exposures. PADOH and ATSDR evaluated the most reasonable worst case scenario for this site of ten years, based on chemical use data and public water availability. PADOH and ATSDR believe that results for evaluating risk for lesser time periods at this site would be less conservative and less reliable than a ten year period.

5. *ATSDR does not know:*

   a. *Past ambient air exposures*
   b. *Pre-2001 indoor air exposures*
   c. *Pre-1998 groundwater levels and exposures*
   d. *Other chemical exposures from Chromatex*

With these data gaps there appears to be significant uncertainty and exposures could be underestimated.

We acknowledge throughout this document that there are important data gaps and uncertainty inherent in our assessment. For that reason, we are conservative in our calculations, conclusions, and recommendations.

6. *Would there be an "apparent health hazard" absent remedial measures?*

   We would find that there would be a public health hazard at this site if no remedial measures had been implemented at this site (e.g., no public water line, no removal of highly contaminated soils, no mitigation of vapor intrusion).

B. Community's Health Questionnaire Forms - The community group VRAP prepared and distributed their own health questionnaire form. Summaries were completed by ATSDR and PADOH. Summary and comment on the questionnaire results may be found in the PHA *Community Health Concerns* section.