Draft Site Investigation Report and Remediation Work Plan

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VRP # 6090502
TRI # 100.05.08

Prepared for:

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EXECUTIVE SUMMARY

This document is an Additional Site Investigation Report and Remediation Work Plan (RWP) for the Former Fame Laundry, now 1352 North Illinois Street, LLP site located at 1352 North Illinois Street in Indianapolis, IN (the Site). Historic operations at the Site have resulted in chemical impacts to soil and groundwater that exceed the Indiana Department of Environmental Management's (IDEM) current regulatory standards. This document, which is being submitted pursuant to the requirements of the IDEM's Voluntary Remediation Program, describes both the nature and extent of contaminant impacts and our proposed strategy to obtain regulatory closure for the Site. This document was prepared in accordance with the IDEM's Risk Integrated System of Closure (RISC) Technical Manual (as amended January 2006).

Analytical results from soil and groundwater testing indicate the presence of chlorinated volatile organic compounds (cVOCs) in concentrations exceeding applicable regulatory standards. Of the constituents identified, tetrachloroethylene (PCE) was found in both vadose zone soil and groundwater at concentrations exceeding the RISC Industrial Default Closure Levels (IDCLs). In addition, trichloroethylene (TCE) is reported in groundwater at concentrations exceeding the RISC Residential Default Closure Levels (RDCLs).

Troy Risk performed a Site-specific Risk Assessment as part of the development of the proposed closure strategy (see Section 5.0). Among the exposure pathways assessed, direct contact with impacted soil, migration of volatile constituents into indoor air (vapor intrusion), and ingestion of impacted groundwater were considered to have the greatest potential to impact human health. Each of these exposure pathways will be addressed through remediation activities, the proposed use of institutional and/or engineering controls, or through the demonstration that completed exposure pathways do not exist.

Potentially, Site workers can be exposed to impacted media during the implementation of the proposed corrective actions. A Site safety plan was prepared to protect workers responsible for completing these activities.

The Site is located in an urban commercial area within Indianapolis; situated at an approximate elevation of 715 feet above mean sea level (MSL). The Site is bounded on the north by a commercial building operated by Shank Simply Self Storage and Rich Furniture; on the west by Tip Top Tavern and Yellow Rose Carriage; on the south by Fastenal Industrial and Construction Supplies; and on the east by the local United Steel Workers union hall (across Illinois Street).

The proposed cleanup criteria include default and non-default closure objectives summarized in Table 5. Default closure objectives were obtained from Table A / Appendix 1 of the RISC Technical Manual, as amended May 2009. Non-default closure objectives are summarized in the Site-specific risk assessment contained in Section 5.0.

Troy Risk proposes to mitigate the risk to human health posed by soil and groundwater impacts utilizing the following approach:

1) A Site-specific risk assessment of cVOC impacts entrained within fine-grained sediments occurring between 0 and approximately 7 feet bgs;
2) The installation and operation of a Soil Vapor Extraction (SVE) system to address impacts in coarse-grained sediments between approximately 7 and 30 feet bgs (implemented March 2009);
3) Demonstration that the dissolved cVOC plume in groundwater is either stable or shrinking through plume stability monitoring, or through attenuation modeling;
4) Installation and operation of a sub-slab depressurization system to eliminate the potential for contaminant exposure through vapor intrusion; and
5) Execution of deed restrictions to prevent the future use of groundwater beneath the Site.

In addition to the above activities, representatives for Michaelis Development, LLP are actively participating in IDEM's 14th Street Corridor Potentially Responsible Party (PRP) Group. This group is comprised of several sites within close proximity of each other, all with cVOC contamination that has impacted regional groundwater resources. The group is in discussions with the Marion County Health Department to create a No Well Zone in the affected area. In meetings with IDEM and Indianapolis Water, IDEM has indicated that they may adopt region-specific closure objectives for cVOC contamination to groundwater. The PRP group is currently evaluating potential closure objectives for IDEM review.

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1.0 INTRODUCTION

On behalf of 1352 North Illinois Street, LLP, Troy Risk, Inc. (Troy Risk) has prepared this report to both summarize Site characterization activities and to propose a remedial approach to gain regulatory closure for cVOC impacts originating from the Former Fame Laundry located at 1352 North Illinois Street in Indianapolis, IN. This report also summarizes the design and implementation of the proposed remedial strategy. This document was prepared in accordance with guidance in the IDEM's RISC Technical Manual (as amended January 2006).

1.1 Site Background and Description

The Site is located at 1352 North Illinois Street, Indianapolis, IN, in Center Township of Marion County; situated at an approximate elevation of 715 feet above mean sea level (MSL). The Site is bounded on the north by a commercial building operated by Shank Simply Self Storage and Rich Furniture; on the west by Tip Top Tavern and Yellow Rose Carriage; on the south by Fastenal Industrial and Construction Supplies; and on the east by the local United Steel Workers union hall (across Illinois Street). A topographic vicinity map and general Site map are provided as Figures 1 and 2, respectively. A property zoning map is included as Figure 3.

The Site property was purchased by Michaelis Development, LLP, in March of 2003, and was subsequently transferred to 1352 North Illinois Street, LLP. The Site is currently awaiting redevelopment as a mixed use (commercial on the first floor, with residential units on the upper floors) or residential property. The Site consists of a rectangular-shaped property (approximately 0.9 acres) with two buildings. The main building, situated on the northern half of the property, consists of a 68,000 ft^2, three-story brick building on a concrete slab. This property was formerly used as a commercial laundry and dry cleaner operated by Fame Laundry between 1940s and the early 1960s. The second building, situated on the southern half of the property, is a 6,700 ft^2 single-story brick building that is reported to have previously been used as a garage for the laundry's delivery vehicles. The second building is also situated on a concrete slab. Together the buildings encompass approximately 60% of the total footprint of the Site. The remainder of the Site is covered with asphalt (~30% ground cover) and a small grass area located between the two buildings (~10% ground cover). There are no known basements, vaults, or sumps onsite. The property is serviced by public utilities including municipal water and sewer. The Site and its immediate surroundings are shown in Figure 2.

1.2 Site Investigations

An initial Site investigation was completed by Patriot Engineering and Environmental, Inc. (Patriot) in 2002. This investigation included the advancement of 7 borings at locations designated as B-1 thru B-7

on Figure 2. Results from this investigation indicated the presence of tetrachloroethylene (PCE) in both soil and groundwater at concentrations exceeding RISC IDCLs. In addition, TCE was found in groundwater at concentrations exceeding RISC RDCLs.

Beginning in 2003, August Mack Environmental (AME) and Troy Risk performed a series of subsurface investigations, both onsite and offsite. These investigations have focused on delineating the extent of cVOC impacts in vadose zone soil, assessing potential human exposure pathways, and collecting geologic and hydrogeologic data necessary to assess Site remediation alternatives. Site characterization performed during these investigations included:

1) The advancement of 52 Geoprobe borings both onsite and offsite;
2) Collection and analysis of soil samples for both VOCs and total organic carbon (TOC);
3) Installation of 10 permanent groundwater monitoring wells;
4) Collection and analysis of groundwater samples for VOCs;
5) Slug testing on monitoring wells MW-1 thru MW-5; and,
6) Pilot-scale testing to evaluate the efficacy of Soil Vapor Extraction (SVE) in addressing vadose zone soil impacts

A compilation of data produced from these investigations are presented within this document. Methods and materials used in the course of performing Troy Risk's investigations are summarized in Appendix A.

2.0 SITE CHARACTERIZATION

2.1 Geology/Hydrogeology

2.1.1 Regional Geology

The geology in the region is comprised of Devonian limestone and dolomite overlain by 50 to 100 feet of unconsolidated, calcareous deposits. The Site lies within the New Castle Till Plains and Drainageways section of the Central Till Plains of Indiana (Gray, 2004). The New Castle Plains are distinguished by relatively flat till-covered uplands occasionally cut by deep, steep-sided glacial valleys that contain thick accumulations of sands and gravels. The Site is situated within one such valley occupied by the White River.

2.1.2 Site Geology

Soil borings indicate a relatively uniform geology over most of the project area. Shallow sediments primarily consist of 0 to 4 feet of sandy loam fill materials overlying 3 to 8 feet of silty clay loam. These surficial sediments are underlain by interbedded sands to a depth of approximately 25 feet bgs, followed by sands and gravels to a depth of at least 30 feet bgs. Geologic cross sections of the project area are provided as Figures 4 and 5. Soil boring logs and monitoring well construction diagrams are included as Appendix B.

2.1.3 Regional Hydrology

The Site is situated approximately 0.8 miles east of Fall Creek and approximately 1.6 miles east of the confluence of Fall Creek and White River. Groundwater in the region occurs in and is available from unconsolidated deposits, primarily sand and gravel from outwash and glacial drift, and from bedrock. Recharge is through precipitation that seeps through overlying permeable outwash, alluvium and kame materials. Water well logs obtained from the Indiana Department of Natural Resources – Division of Water (Appendix C) indicate unconsolidated deposits in the local area are dominated by water-bearing sand and gravel outwash deposits; however, these deposits may in places be divided into two or more distinct units by relatively impermeable clay-rich deposits. Review of available well construction information indicates most wells in the vicinity of the Site are screened in either sand and gravel formations at depths ranging from 41 to 97 feet bgs, or in limestone at depths greater than approximately 100 ft bgs.

2.1.4 Site Hydrology

Troy Risk defined three stratigraphic units (Figure 6) during the course of performing subsurface investigations. Their definition was based upon field observations of their composition, but more importantly their potential effect on the migration of CoCs at the Site. These units were not specifically distinguished based upon depositional history, nor have they been assessed based upon traditional stratigraphic sequence analysis. The stratigraphic units are as follows:

<u>Stratigraphic Unit #1</u>

- This unit consists of 0 to 4 feet of dry to moist sandy loam fill material overlying 3 to 8 feet of moist to wet silty clay loam.
- Depth Interval: 0 to 8 feet bgs.

<u>Stratigraphic Unit #2</u>

- Outwash deposits consisting of interbedded layers of fine to medium sand, sand and gravel and discontinuous loams.
- Depth Interval: 8 to 25 feet bgs.

<u>Stratigraphic Unit #3</u>

- Saturated sand and gravel outwash deposits
- Depth Interval: greater than 25 feet bgs
- Current Monitoring Well Set: MW-1 thru MW-10

Depth to groundwater in this unit has historically ranged between 27.24 and 31.40 feet bgs during past groundwater gauging events (Table 1). Groundwater flow within this unit occurs under unconfined conditions in a southwesterly direction, as illustrated in Figure 7. This groundwater flow direction is in

close agreement to that reported to the IDEM during subsurface investigations related to other contaminant releases within the local area (References).

Slug tests were performed on monitoring wells MW-2 thru MW-5 on June 10, 2008. The purpose of these tests was to estimate the horizontal hydraulic conductivity of soil within the unit. Slug tests were performed using the methodology outlined in EPA Standard Operating Procedure (SOP) #2046, using a pressure transducer in conjunction with an automated data logger. The slug tests were conducted by quickly adding a slug (known volume) of water to a well and then measuring the time required for the water level to return to its static level. The rate at which the well re-established equilibrium (i.e., static water level) enables one to estimate the hydraulic conductivity (K) for the immediate area surrounding the well screen. Estimated K values are summarized in Table 2 (sample calculations are included as Appendix D.

The hydraulic gradient in this unit was calculated using a vector method on monitoring well gauging data collected on January 26, 2006. This analysis indicates groundwater flow at a hydraulic gradient of approximately 0.002 ft/ft. Groundwater discharge velocities were calculated using the range of hydraulic conductivity ($10^{-1.9}$ to $10^{-2.1}$ cm sec^{-1}) observed in the unit assuming a range in effective soil porosity of 0.25 to 0.35. Using this approach groundwater velocities are estimated to range between 47 and 104 ft $year^{-1}$, with a mean velocity of approximately 73 ft $year^{-1}$.

2.2 Contaminants of Concern (CoCs)

Analytical results from soil and groundwater testing indicate the presence of chlorinated volatile organic compounds (cVOCs) in concentrations exceeding applicable regulatory standards. Of the constituents identified, tetrachloroethylene (PCE) was found in both vadose zone soil and groundwater at concentrations exceeding the RISC Industrial Default Closure Levels (IDCLs). In addition, trichloroethylene (TCE) is reported in groundwater at concentrations exceeding the RISC Residential Default Closure Levels (RDCLs). Soil and groundwater analytical results are summarized in Tables 3 and 4, respectively. Laboratory analytical results are attached as Appendix E.

Chlorinated solvents are often released to the environment in a more or less pure form. It is reasonable to assume that the ultimate source of both PCE and TCE impacts was likely a release of nearly pure solvent. Site inspections and review of historical documentation have not indicated the existence of any subsurface process equipment, process piping or underground storage tanks associated with the use of the PCE. The specific source of historical releases associated with PCE impacts to soil and groundwater has not been determined; however, the level of sampling performed at the Site does provides a good understanding of the general distribution of PCE within the vadose zone (Figure 8).

The Site is situated within an area of downtown Indianapolis known to have multiple sources of PCE impacts to groundwater. A review of IDEM files indicated as many as four properties within a two block radius of the Site have been documented to, or are suspected to, be a source of PCE impacts. The sites are: 1) Karstadt Reed Cleaners, 2) Former Greater Diversified Supply, 3) Former Shuron Optics Facility, and 4) Former Stewart Manufacturing. These properties are situated hydraulically upgradient, side gradient and down gradient of the Site; therefore, delineating the full nature and extent of PCE

impacts originating from the Site is complicated by overlapping dissolved cVOC plumes. cVOCs concentrations in groundwater near the Site are shown on Figure 9.

2.2.1 Contaminant Delineation

As shown on Figure 8, cVOC impacts to the vadose zone extend offsite to the north, east, and south in Stratigraphic Unit #2. Soil samples across 14th Street, Illinois Street, and Cora Street have all shown low-level PCE impacts. It appears that cVOC impacts to soil and groundwater were sourced from the northeastern corner of the main Site building. Since groundwater flow is to the southwest at the Site, these impacts are upgradient and side-gradient from the suspected source area. It is likely that the low-level detections of PCE in the vadose zone of these areas are from volatilization of the regional dissolved PCE plume.

cVOC impacts to groundwater also extend offsite. As shown on Figure 9, the northern and southern boundaries of the dissolved PCE plume have been delineated to RDCLs. The southern boundary was defined by MW-10 as of September 2009. Since that time, the concentration of PCE has increased to 6.01 ppb, slightly above the RDCL. Low levels of dissolved PCE appear to cross Illinois Street. However, access is not available to install monitoring wells on the United Steel Workers union hall property, further east of MW-7. In addition, prior to 2009, MW-3 had low-level detections of PCE in groundwater. These, combined with the low-level detections to the east of the Site indicate there may be an upgradient source of PCE in groundwater. Due to the increase in PCE concentration from MW-5 to MW-9 (in the direction of groundwater flow), it is believed that the PCE plume originating from the Site comingles with the regional dissolved plume in the vicinity of MW-9.

2.2.2 Physical and Chemical Properties of cVOCs

The physical and chemical properties of most cVOCs encourage this class of compounds to migrate through soil and groundwater. The following list discusses the characteristics of these compounds as they pertain to fate and transport in subsurface environments:

1) The combination of low absolute solubility and high specific gravity common to most cVOCs suggest that when a significant quantity of these compounds are spilled on the ground surface, liquid solvent will be able to migrate as a DNAPL into the subsurface, potentially accumulating as pools on the tops of low permeability layers. The low solubility will then permit such pools to persist for extended periods of time.
2) The relatively low viscosities of cVOCs allow rapid downward movement into the subsurface. Downward mobility of these constituents increases with increasing density/viscosity ratios.
3) The low interfacial tension between chlorinated solvent DNAPL and water allows the DNAPL to easily enter into small fractures and pore spaces, facilitating deep penetration into the subsurface. Low interfacial tension also contributes to the low retention capacity of soil for the contaminants.
4) The high relative solubility of cVOCs means that a solvent release can cause groundwater contamination at levels that are high relative to cleanup criteria (e.g., the solubility of PCE in water is approximately 30,000 times greater than the RISC RDCL).

5) The low partitioning to soil materials exhibited by cVOCs indicates that the soil will bind these compounds only weakly. Therefore, sorption to soils will not significantly retard the movement of contaminants, and the zone of impact can grow relatively quickly.
6) The low degradability of chlorinated solvents, either by biological means, or by abiotic chemical reactions, suggests that subsurface lifetimes of these chemicals can be very long.

Guidance from the United States Environmental Protection Agency (USEPA, 2002) recommends operating under the assumption that DNAPL is present at Sites where concentrations of solvents in groundwater exceed 1% of their aqueous solubility. This criterion has not been met at the Site. Furthermore, the general levels of PCE impacts observed in vadose zone soil are not indicative of the presence of DNAPLs.

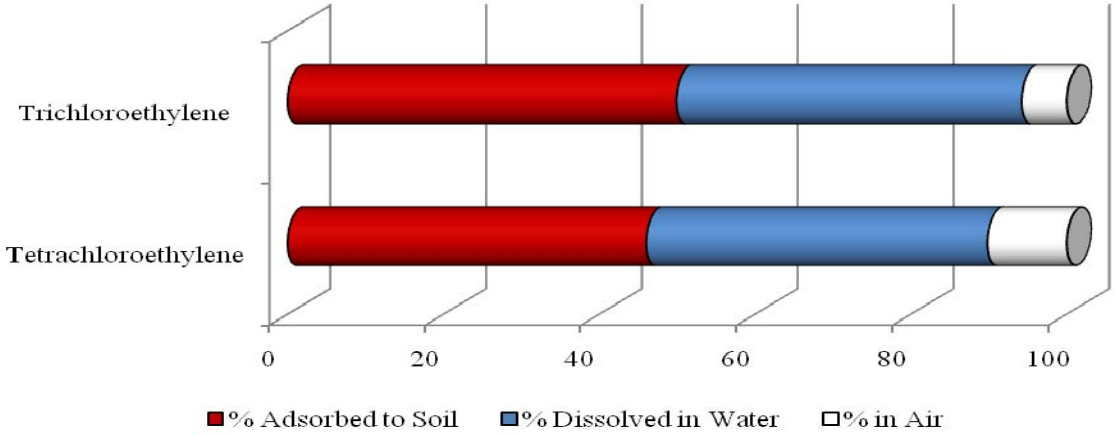
Material Safety Data Sheets (MSDSs) and information from the EPA's Integrated Risk Information System (IRIS) are included for cVOCs found at the Site in Appendix F.

2.2.3 Partitioning of cVOCs

Each contaminant has its own distinct set of physiochemical properties that govern its behavior in subsurface environments. When releases of organic compounds take place, the contaminants may exist in the subsurface as four distinct phases: mobile free product or NAPLs, the adsorbed phase, the dissolved phase, and the vapor phase.

In order to develop a conceptual understanding of the distribution of CoCs among these phases, Troy Risk developed a theoretical model of their partitioning using chemical data and 'default' geologic properties summarized in Appendix 1 of the RISC Technical Manual. As mentioned above, dissolved concentrations suggest that NAPLs are not present at the Site; therefore, this phase was excluded from consideration. The theoretical distribution of Site contaminants in both the vadose zone and the saturated zone is as follows:

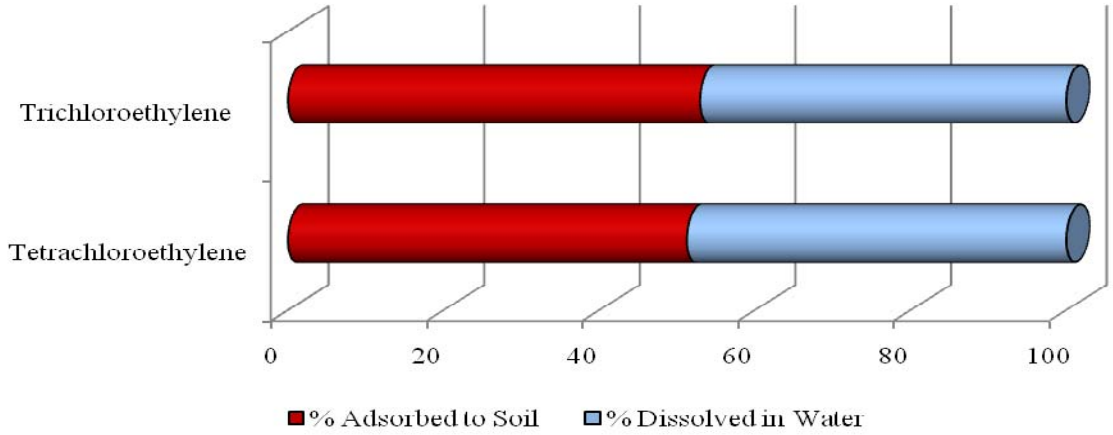
Theoretical Distribution of cVOCs in Vadose Zone Soil



Assumptions:

- Fraction of organic carbon (f_{oc}) = 0.2%
- Air Filled Soil Porosity = 13.4%
- Water Filled Soil Porosity = 30%
- No NAPL Present

Theoretical Distribution of cVOCs in Saturated Soil / Groundwater



Assumptions:

- Fraction of organic carbon (f_{oc}) = 0.2%
- No NAPL Present
- Water Filled Soil Porosity = 43.4%

2.3 Sensitive Receptors

2.3.1 Ecological Receptors

Troy Risk conducted a Baseline Ecological Assessment (BEA) at the Site to determine critical habitats that could potentially be impacted by Site contaminants. This BEA included the following efforts:

1) A field inspection of the Site and surrounding areas for visual evidence of critical habitats;
2) A review of the United States Geological Survey (USGS) Indianapolis West 7.5-minute quadrangle topographic map for features such as parks, preserves, and other special land use areas;
3) A review of National Wetlands Inventory (NWI) maps published by the United States Department of the Interior – Fish and Wildlife Service (1990); and
4) Inquiries regarding endangered, threatened, and rare species, and high quality natural communities to the IDNR – Division of Nature Preserves.

As mentioned previously, the Site is located within the Indianapolis city limits. Areas surrounding the Site have been developed for commercial and/or industrial activities. There are no forested areas, prairies, dunes, sinkholes, water reservoirs, hatcheries, nature preserves or fish and wildlife management areas on or in the vicinity of the Site. The nearest identified is Sherman Park, which is located approximately 3.1-miles east-southeast of the Site. The nearest surface water and wetlands area are associated with the Central Canal approximately 0.35-miles to the southwest (Figure 10). The canal is not a significant habitat for wildlife. Neither of these receptors is considered susceptible to contaminants originating from the Site.

Troy Risk has contacted the Indiana Department of Natural Resources – Division of Nature Preserves and for information regarding the presence of endangered, threatened, or rare (ETR) species, high quality natural communities, or natural area at or in the vicinity of the Site. According to information obtained from the IDNR there are no ETR species, high quality natural communities, or natural areas documented with the project area. Copies of IDNR correspondence are included in Appendix G.

2.3.2 Potentially Susceptible Areas

High-capacity and low-capacity groundwater wells identified in the IDNR-DOW data base are shown in Figures 11 and 12, respectively. The Site is situated just within the eastern-most boundary of the Riverside Wellhead Protection Area within Indianapolis. This designation is based on the Site's proximity to high capacity potable supply wells owned and operated by the Indianapolis Water Company (IWC). The closest wells belonging to the IWC are situated approximately 0.64-miles to the west-northwest (Figure 11), which is hydraulically side-gradient of the Site. Therefore, these wells are not anticipated to be susceptible to cVOCs impacts originating from the Site. Nevertheless, Troy Risk has performed a series of modeling exercises to evaluate potential impacts to these wells assuming groundwater flow was toward the west-northwest. These modeling exercises are summarized in Appendix H, and indicate that even under a highly conservative set of assumptions groundwater impacts originating from Site could not impact the potable supply wells at concentrations exceeding the U.S. EPA's maximum contaminant level (MCL) for PCE in drinking water.

Apart from wells belonging to the IWC, Troy Risk identified two water wells within a quarter mile radius of the Site, including one high capacity well. Eight water wells were identified within a quarter to half-mile down gradient radius, including one high capacity well. Troy Risk has learned of two high-capacity water wells downgradient of the Site (Figure 11) owned by the City of Indianapolis, that have not been identified in the IDNR database . These wells are reported to source the Central Canal. Modeling exercises have been performed to evaluate potential impacts to these wells. These modeling

exercises are summarized in Appendix H, and indicate that even under a highly conservative set of assumptions groundwater impacts originating from Site could not impact the suspected canal supply wells at concentrations exceeding the U.S. EPA's maximum contaminant level (MCL) for PCE in drinking water.

In addition to parks, which were discussed in Section 3.3.1, potentially susceptible areas include hospitals and schools. The nearest identified school is Herron High School approximately 0.3-miles to the northeast of the Site. The nearest identified hospital is Methodist Hospital approximately 0.4-miles to the northwest. Neither of these receptors is considered susceptible to contaminants originating from the Site.

3.0 CLEANUP CRITERIA SELECTION

The proposed cleanup criteria include default and non-default closure objectives summarized in Table 5. Default closure objectives were obtained from Table A / Appendix 1 of the RISC Technical Manual. Non-default closure objectives are summarized in the Site-specific risk assessment contained in Section 5.0.

4.0 STATEMENT OF WORK

4.1 Summary of Activities

The following activities will be performed to mitigate the risk to human health and environmental resources:

1) The installation and operation of a Soil Vapor Extraction system to remove cVOCs entrained in higher permeability sediments between approximately 7 and 30 feet bgs (implemented March 2009);
2) Demonstration that the dissolved cVOC plume in groundwater sourced by the Site is stable or shrinking through plume stability monitoring, or through attenuation modeling;
3) Installation and operation of a sub-slab depressurization system to eliminate the potential for vapor intrusion into the Site building in the future (as necessary); and
4) Execution of deed restrictions to prevent the future use of groundwater beneath the Site.

4.2 Site Safety Plan

A Site Safety Plan is included as Appendix I.

4.3 Quality Assurance Project Plan

A *Quality Assurance Project Plan* (QAPP) is provided in Appendix J. The QAPP was prepared in accordance with the guidance in the *VRP Resource Guide.*

4.4 Community Relations Plan

A *Community Relations Plan* is provided in Appendix K. The plan was prepared in accordance with the guidance in the *VRP Resource Guide.*

5.0 RISK ASSESSMENT / DEFINITION OF CLOSURE OBJECTIVES

Troy Risk evaluated the potential risks to human health and the environment for populations that may now, or at some time in the future, be exposed to chemical impacts defined in Section 3.0. This risk assessment focused on the analysis of exposure pathways associated with environmental media including surface soil, subsurface soil, groundwater and surface water. Exposure routes that were assessed for each media are as follows:

Surface Soil
- Direct contact with skin (dermal adsorption route)
- Inhalation of soil particulates and dust (ingestion and inhalation routes)
- Volatilization from soil to air (inhalation route)

Subsurface Soil
- Migration to groundwater
- Volatilization from soil to air (inhalation route)

Groundwater
- Volatilization from water to air (inhalation route)
- Direct contact with skin (dermal adsorption route)
- Water consumption (ingestion route)

5.1 Surface Soil (0 to 0.5 ft bgs)

The risk to human health associated with surface soil is attributed to direct exposure to contaminated soil. Because the four direct contact routes assessed (skin contact, dust inhalation, volatilization, and soil consumption) often exist simultaneously for any potential receptor, their evaluation is usually performed as one operation. The RISC Technical Manual provides a default assessment of this exposure pathway through the calculation of default 'Direct Contact' closure levels for soil under both residential and non-residential land-use scenarios. For regulatory closure of surface soil, default residential 'Direct Contact' closure levels are proposed for onsite and offsite surface soil. Concentrations of PCE detected in soil are well below the default 'Direct Contact' closure levels of 9.9 and 16 mg kg^{-1} for residential and non-residential land uses, respectively. At present, the Site is nearly entirely paved with asphalt or concrete, or contains a significant structure. Future development plans for the Site include improving and/or maintaining these physical barriers to soils. As such, direct contact and ingestion exposure risks at the Site are greatly reduced, if not completely eliminated.

5.2 Subsurface Soil (0.5 to 30 ft bgs)

The risk to human health associated with impacts in this unit is primarily attributed to migration of contaminants into groundwater, which, in turn, could result in exposure through routes associated with groundwater. In addition, inhalation of volatile constituents liberated from impacted soil is also of concern. The RISC Technical Manual provides a default assessment of the migration to groundwater exposure pathway through the calculation of default 'Migration to Groundwater' closure levels for soil under both residential and non-residential land-use scenarios. For regulatory closure of subsurface soil, default residential 'Migration to Groundwater' closure levels are proposed for onsite and offsite soil. However, if necessary, Troy Risk may in the future propose to obtain closure for groundwater through the non-default plume stability approach described in the RISC Technical Manual. In the event this plume stability approach is proposed, the definition of specific closure levels for subsurface soil, as they relate to 'Migration to Groundwater', will not be necessary. Provided the groundwater plume is stable, migration of contaminants to groundwater will not be considered a significant exposure pathway.

IDEM has published soil screening levels to assess vapor intrusion for both PCE and TCE in their *Draft Vapor Intrusion Pilot Program Guidance* (April 2006). These levels are 5 mg kg^{-1} for each constituent assuming a 30 year residential exposure scenario. Analytical data collected from the Site indicate that soil impacts exceeding these screening levels are isolated to Stratigraphic Unit #2., beneath the northeastern portion of the Site's main building. The operation of the soil vapor extraction system is expected to reduce soil impacts to below these screening levels.

5.3 Groundwater

The risk to human health associated with groundwater impacts is primarily attributed to ingestion of and direct skin contact with groundwater, and the inhalation of volatile constituents liberated from the groundwater. The RISC Technical Guidance provides a default assessment of these exposure routes through the calculation of default groundwater closure levels under both residential and non-residential land-use scenarios. At this time, default residential 'Groundwater' closure levels are proposed for onsite and offsite groundwater. However, Troy Risk anticipates it may ultimately be necessary to propose closure for groundwater impacts through the plume stability approach described in the RISC Technical Manual or through the use of a groundwater use ordinance. Regardless of the closure objectives ultimately utilized for groundwater, the future potential for ingestion of and direct skin contact with cVOCs in groundwater will be eliminated through the execution of a deed restriction to prevent the future use of groundwater at the Site.

As mentioned previously, the full delineation of the nature and extent of groundwater impacts is complicated by the presence of multiple cVOC plumes in the local area. As a result, achieving default residential groundwater closure levels on offsite, downgradient properties will not be possible.

The Site is currently participating in IDEM's 14th Street Corridor Potentially Responsible Party (PRP) Group. This group is comprised of several sites within close proximity of each other, all with cVOC contamination that has impacted regional groundwater resources. The group is in discussions with the Marion County Health Department to create a No Well Zone Ordinance in the affected area. This would eliminate the groundwater exposure scenario. Also, in meetings with IDEM and Indianapolis Water,

IDEM has indicated that they may adopt region-specific closure objectives for cVOC contamination to groundwater. The PRP group is currently evaluating potential closure objectives for IDEM review.

6.0 REMEDIATION PLAN

6.1 Additional Field Investigations

No further investigation is planned at this time.

6.2 Evaluation of Remediation Technologies for Stratigraphic Unit #2

There is often more than one technology available to achieve remediation objectives at any given site. These alternatives are considered and compared as part of the evaluation process leading to the selection of a remedial approach. Site geologic and hydrogeologic characteristics, cleanup objectives, and the contaminants targeted for remediation play a primary role in selecting the appropriate remediation strategy. In addition, secondary screening criteria include estimated remediation costs, the estimated time to achieve regulatory closure, potential interruptions to ongoing site activities and permitting requirements. The following remediation alternatives were evaluated with respect to these criteria:

- No action
- Excavation/Disposal
- Soil Vapor Extraction
- Electrical Resistance Heating
- In-Situ Chemical Oxidation

6.2.1 No Action

Effectiveness
In this option, the toxicity, mobility, and volume of contamination will not be reduced. Because no action will be taken, the vadose zone PCE impact will remain as a source of cVOCs to groundwater. Without removal of the source, groundwater concentrations of cVOCs are likely to increase over time.

Implementability
There are no technical or material factors that would inhibit implementation of this alternative. However, the location of the Site within a wellhead protection area necessitates the implementation of a strategy that will reduce or eliminate further migration of contamination to groundwater. The Site would not be eligible for closure under this scenario.

Cost
There is no direct cost associated with this option. However, by not reducing the vadose zone source of PCE, cVOCs will continue to migrate downward into groundwater. The cost of groundwater monitoring will likely increase due to the ongoing impact to groundwater.

6.2.2 Excavation / Disposal

Technology Description
In this option, impacted soil is excavated and disposed of at an approved waste landfill. Excavation is a standard construction practice and equipment and construction methods are readily available for the excavation and handling of contaminated material.

Effectiveness
The primary benefit of an excavation and disposal approach is that it provides a certain outcome with respect to soil. The contaminated soil is completely removed from the Site and restored with clean fill. Upon completion of excavation activities, confirmation soil samples would show that cleanup objectives have been met.

Implementability
The aerial extent of contaminant impacts is situated beneath the Site's main building. Special engineering precautions would be necessary to assure the stability of load-bearing walls and columns during excavation activities. The close proximity of these load-bearing structures is anticipated to limit the depth of excavation in most places to approximately 4 to 8 feet bgs, which is not sufficient to remove the majority of impacted soil, thereby necessitating a secondary remedial strategy to address deeper soils.

Cost
Approximately 6,700 tons of impacted vadose zone soil are accessible for excavation. The approximate cost to excavate and dispose of the shallow vadose zone soil and restore the Site is $380,000. However, the majority of impacted soil is located beyond the capabilities of excavation. The deeper impacted soil would require a secondary remedial strategy for treatment.

6.2.3 Soil Vapor Extraction

Technology Description
Soil Vapor Extraction (SVE), also known as soil venting or vacuum extraction, is an in-situ remedial technology that reduces concentrations of volatile contaminants adsorbed to unsaturated soil. Using this technology, a vacuum is applied through a series of extraction wells screened within impacted vadose zone soil. Volatile constituents partition into the gas phase and the vapors are drawn toward the extraction wells. Extracted vapor is then treated as necessary before being released to the atmosphere.

Effectiveness
The highly permeable sands found in Stratigraphic Unit #2 are favorable for the utilization of SVE at the Site Application of SVE will reduce both the mobility and volume of contamination. The applied vacuum will capture PCE sorbed to soil particles, as well as limit the downward migration of PCE into groundwater. A total contaminant removal in excess of 80 percent is expected through soil vapor extraction at the Site.

Implementability
There are no Site-specific factors that prevent the use of this technology to treat contaminant impacts in Stratigraphic Unit #2. Materials and contractors necessary to construct and install the designed SVE system are readily available. Permits are required to place the SVE equipment enclosure at the Site and to discharge any groundwater captured by the system to the sanitary sewer for treatment. Troy Risk estimates that the SVE equipment will require a run time of no more than three years, after which the Site would be eligible for closure.

Cost
The capital cost to implement SVE at the Site is expected to be approximately $176,600. This includes design and permitting ($5,300), construction SVE aboveground equipment ($67,500), installation ($94,200), and initial startup and optimization of the system ($9,600). Yearly operation and maintenance of the system will cost approximately $20,000 and includes electrical service, sewer bills, and maintenance by Troy Risk personnel.

6.2.4 Electrical Resistance Heating

Technology Description
Electrical Resistance Heating (ERH) is an in-situ technology that applies electricity into the ground through electrodes. ERH operates under the principal that electrical current passing through a resistive component, such as soil, will generate heat. As subsurface temperatures increase, contaminants and soil moisture are vaporized into steam. The steam is then withdrawn from the soil by vapor extraction. As steam is withdrawn from the soil, the subsurface begins to dry out. Drying reduces the electrical conductivity of the soil in these areas, causing an increase in soil resistance. As the resistance of the soil increases, other pathways become preferential for current flow, redirecting the heating to untreated areas. This self-regulation provides uniform heating, and therefore treatment, of even heterogeneous lithologies.

Effectiveness
ERH will reduce the volume of contamination by driving sorbed PCE into the vapor phase; PCE vapors are then extracted and discharged aboveground. The inclusion of the vapor extraction component will also reduce the downward mobility of contaminants. ERH is a proven technology for the treatment of contaminants in low permeability soil, and is reported to be capable of obtaining contaminant reduction in excess of 99%, though such a reduction will not be required for closure at the Site.

Implementatability
No Site-specific factors prevent the implementation of this approach. Construction activities associated with the installation of the system would interrupt any activity at the Site for a period of 4 to 6 weeks. However, there are few trained and qualified contractors capable of installing an ERH system at the Site. As such, there may be a significant waiting period before contractors are available to install the infrastructure required for utilization for ERH. For smaller sites, typical implementation of ERH lasts approximately three months.

Cost

Although ERH is applicable to the Site, costs associated with its implementation are significantly greater than other technologies assessed. Troy Risk has obtained cost estimates from each of the major technology vendors (Current Environmental Solutions, LLC and Thermal Remediation Services, Inc.). Full-scale implementation of ERH for treatment of soil in Stratigraphic Unit #2 was estimated to cost between $1,500,000 and $2,000,000. The total capital cost of ERH is expected to be approximately $750,000 and includes design and permitting (approximately $200,000), construction (approximately $200,000), installation (approximately $300,000), and startup and optimization (approximately $50,000). The ERH system is expected to operate for a period of three months and cost approximately $750,000.

6.2.5 In-Situ Chemical Oxidation

Technology Description

In-Situ Chemical Oxidation (ISCO) involves the injection of chemical oxidants into contaminated media in order to convert hazardous contaminants to nonhazardous or less toxic compounds. Oxidants most commonly employed to date include peroxide, ozone and permanganate. Each of these oxidants have demonstrated effectiveness in reducing the concentrations of chlorinated ethenes in soil and groundwater; however, permanganate (MnO_4^-)–based oxidants have several advantages. These advantages include: 1) The reaction efficiency of MnO_4^- is not significantly affected by pH within the range of pH values encountered in subsurface environments; 2) the half-life of MnO_4^- in the subsurface is significantly longer (weeks compared to minutes or hours) than the other oxidants, allowing MnO_4^- to permeate through the subsurface; and 3) MnO_4^- is thermodynamically more stable than peroxide, lessening the safety concerns with its implementation.

Effectiveness

Well designed ISCO treatments have demonstrated a high efficacy in a rapid timeframe. Provided adequate oxidant-contaminant contact time, contaminant reductions on the order of 80% are expected. Due to their established efficacy, ISCO is often used to treat NAPL source areas and contaminant ‘hot spots’.

Implementability

ISCO is generally used to treat cVOC impacts in saturated soils and groundwater. This is because ISCO relies on diffusion and dispersion to spread through the contaminant volume. When used to treat vadose zone soils, the vadose zone must be “flooded”. In order to effectively treat the large vadose zone impact at the Site, the water level must be raised by approximately 25 feet and maintained at that level throughout the ISCO treatment. This flooding of the vadose zone is considered technologically impracticable due to the very conductive outwash material beneath the Site.

Cost

Due to the technological impracticability of implementing ISCO at the Site, a detailed cost analysis has not been performed. However, ISCO is generally considered to be most cost effective when used to treat relatively small volumes of heavily impacted soil and groundwater. The large vadose zone volume targeted for treatment at the Site would be cost prohibitive to treat with ISCO.

6.3 Recommended Technology for Stratigraphic Unit #2

Given the necessity to treat impacted soil beneath the existing building, a remedial strategy which created minimal subsurface disturbance was required for the Site. The necessity to remove the vadose zone impact, highly permeable sands, nature of contaminants found onsite, and moderate cost made soil vapor extraction an ideal remedial strategy. In March 2009, Troy Risk installed, and began operation of an SVE system for the Site.

6.4 Remedial Design and Implementation

The design specifications, installation, and startup of the SVE system are detailed in the *Soil Vapor Extraction System Startup and Optimization Report*, dated May 1, 2009. The layout of SVE extraction wells, process and instrumentation diagram, and extraction well schematic are included as Figures 13, 14, and 15, respectively.

6.4.1 Plume Stability Monitoring

In the event that Plume Stability Monitoring is proposed for the Site, a *Remediation Work Plan Addendum* will be submitted for approval to IDEM.

6.5 Permit Requirements

We do not anticipate permitting will be required for the vapor extraction activities. The proposed remedial strategy includes the treatment of VOC emissions during activities where the potential to emit is greatest. If during the operation of the system, total VOC emission rates exceed permitting requirements, Troy Risk will go through a formal permitting process with the IDEM.

7.0 MONITORING/CONFIRMATION SAMPLING PLAN

This section describes the long term monitoring plan for this Site, including groundwater monitoring, remediation progress monitoring, sample collection methods, and post remedial action confirmation sampling.

7.1 Groundwater Monitoring

The purpose of groundwater monitoring is to evaluate the effectiveness of the remediation strategy and evaluate the progress of attaining the proposed cleanup objectives in groundwater. A Site monitoring schedule, including sample location, monitoring parameters, analysis method and sample collection frequency is included in Table 6. Locations of monitoring wells and system effluent sampling points are shown in Figures 2 and 14, respectively. There are no known private water supply wells onsite or in the immediate vicinity of the Site.

7.2 Vapor Extraction Monitoring

Monitoring of the SVE system will include a minimum of monthly sampling of the vapor effluent. The mass of VOCs removed during long-term monitoring intervals will be calculated using vapor phase concentrations and flow rate measurements taken at the vapor extraction manifold(s). The instantaneous and cumulative mass removal will then be plotted versus time. Monitoring of the system will continue until consistent asymptotic behavior is observed between vapor effluent concentration reduction and cumulative mass removal of VOCs.

7.3 Post Remedial Action Confirmation Sampling

Groundwater analytical results will be reviewed quarterly, together with remediation system operation data, to determine when the SVE system has either met remediation goals, or is no longer significantly influencing contaminant concentrations. A *Sampling and Analysis Plan* will be submitted to the IDEM's review prior to shutting down the SVE system.

8.0 DATA MANAGEMENT

Reports generated during the performance of the work described in this RWP will consist of the following:

1) Quarterly Groundwater / Remediation System Monitoring Reports;
2) Closure Sampling and Analysis Plan; and
3) Remediation Completion Report

All laboratory analytical data collected at the Site will be tabulated, and submitted to the IDEM within the documents listed above. In addition, a complete log of maintenance activities performed on the remediation system will be kept in an Operations Log Book.

A summary of system operation and maintenance will be submitted to the IDEM as part of the Remediation Completion Report.

9.0 OPERATION AND MAINTENANCE PLAN

9.1 Normal O&M

All normal operation and maintenance activities associated with the operation and maintenance of the Site's SVE system will be in accordance with manufacturer's recommendations. Upon request, an operation and maintenance manual will be submitted to the IDEM upon system installation.

9.2 Potential Operating Problems

During operation of the SVE system, problems can occur that will limit the ability of the system to function properly. Problems may result from the methods used to operate the system or from parts failure. Malfunction of remedial system components may develop gradually over time or occur suddenly. Minor problems are those that can typically be corrected quickly by making operation adjustments, or simple repairs that result in little to no system downtime. Significant problems, however, may require termination of the operation of the remedial system until the problem is corrected or equipment is replaced.

9.3 Vapor Extraction Component

The main potential for operation problems with the vapor extraction component of the system is with the blower. Maintenance and repairs of the blower will be made as identified during routine inspections based on manufacturer's recommendations. The primary operational concern regarding the piping system is freezing of condensate, which can limit flow. Having all lines sloped back to the wells minimizes this concern. Caution must also be taken to prevent pipe breakage during other Site construction activities.

9.4 Contingency O&M

If major system components such as the blower, transfer pumps, or control systems fail, repairs and / or replacements will be completed as soon as possible according to manufacturer's specifications, or based on the availability of replacement parts. If operation of any system component fails as a result of freezing liquid, corrective measures must be taken. Measures include the installation of insulation and may include heat trace wire.

10.0 COMPLETION OF REMEDIAL ACTION

10.1 Completion Report

A remediation completion report will be submitted to the IDEM after Site cleanup objectives have been met.

10.2 Future Use of Site

Long-term plans for the Site may include the conversion of the Site's main building into residential condominiums.

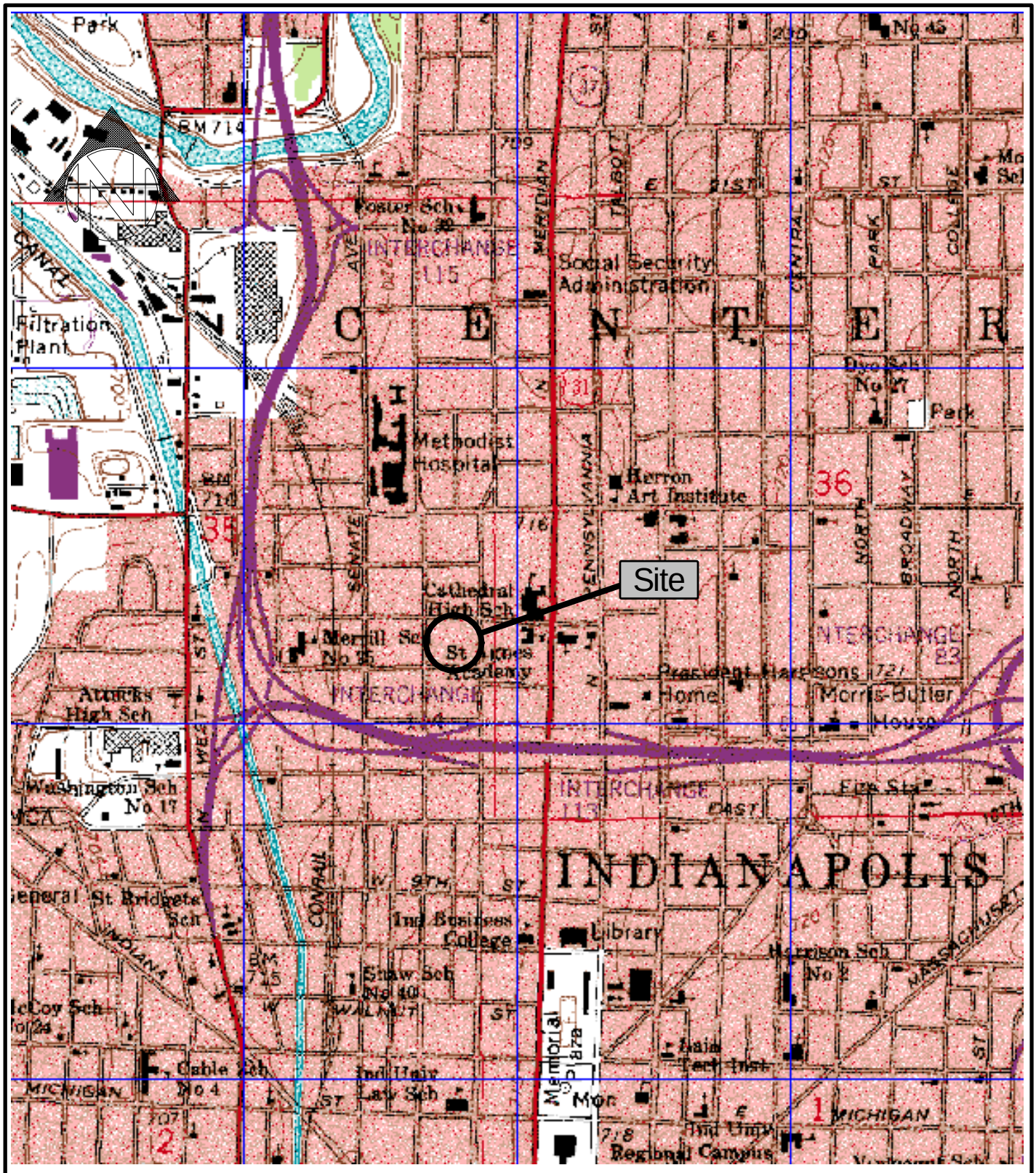
11.0 SCHEDULE

Table 7 summarizes an estimated timetable for implementation of this RWP.

Current Owner:
1352 North Illinois Street, LLP
2601 East 56th Street
Indianapolis, Indiana 46220
c/o: Mr. Richard Michaelis, Michaelis Corporation
317-251-1935

Technical Contact:
Troy Risk, Inc.
7466 Shadeland Station Way
Indianapolis, Indiana 46256
c/o: Mr. Jason B. Flagg, EIT
317-570-6730

FIGURES



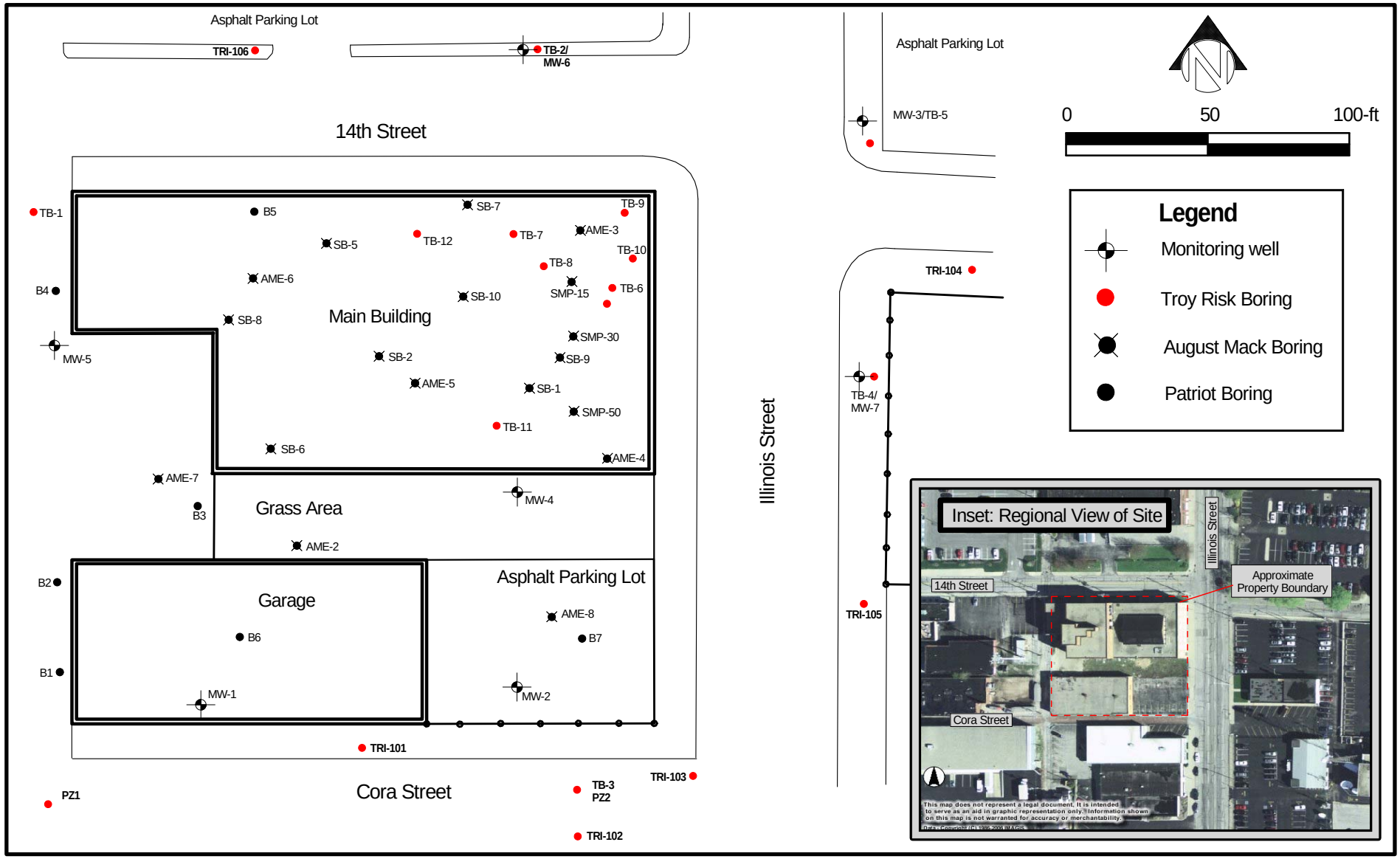
Topographic Vicinity Map

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Topo Map
Date:	December 10, 2009
Scale:	1" = 2000'
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 1



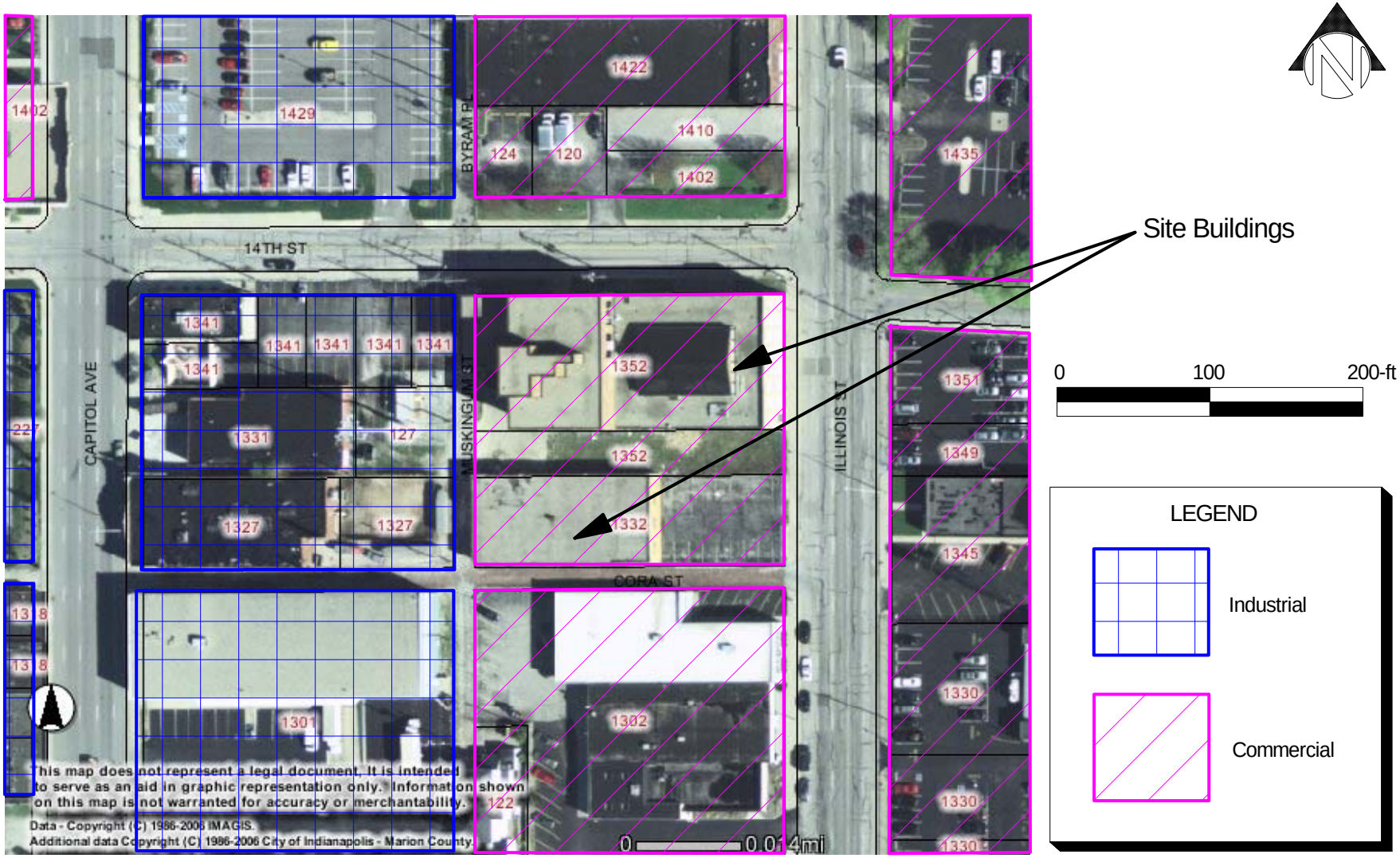
General Site Map

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Site Map
Date:	December 10, 2009
Scale:	1" = 50'
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 2



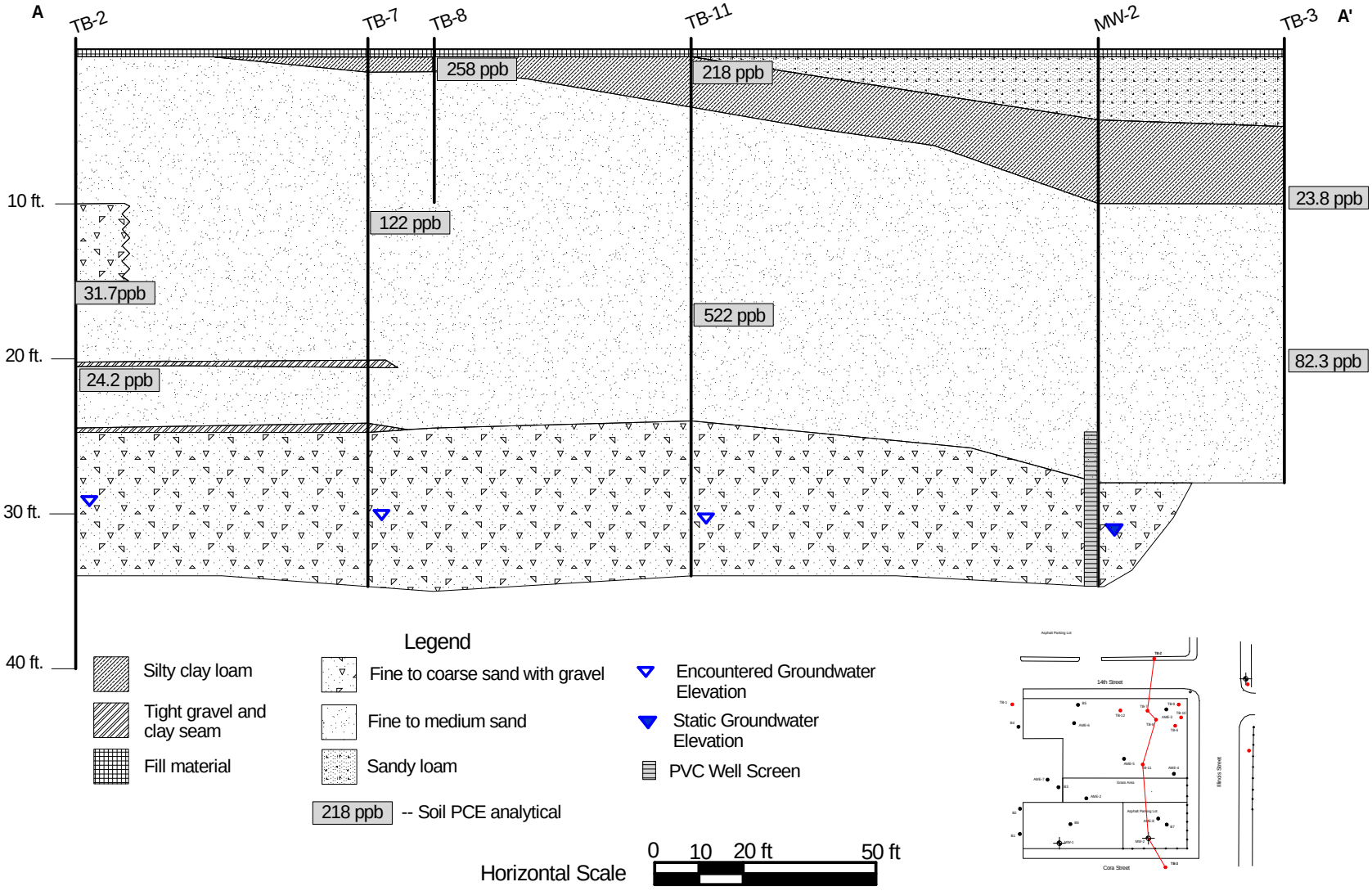
Property Zoning Map

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Zoning
Date:	December 22, 2009
Scale:	1" = 100'
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 3



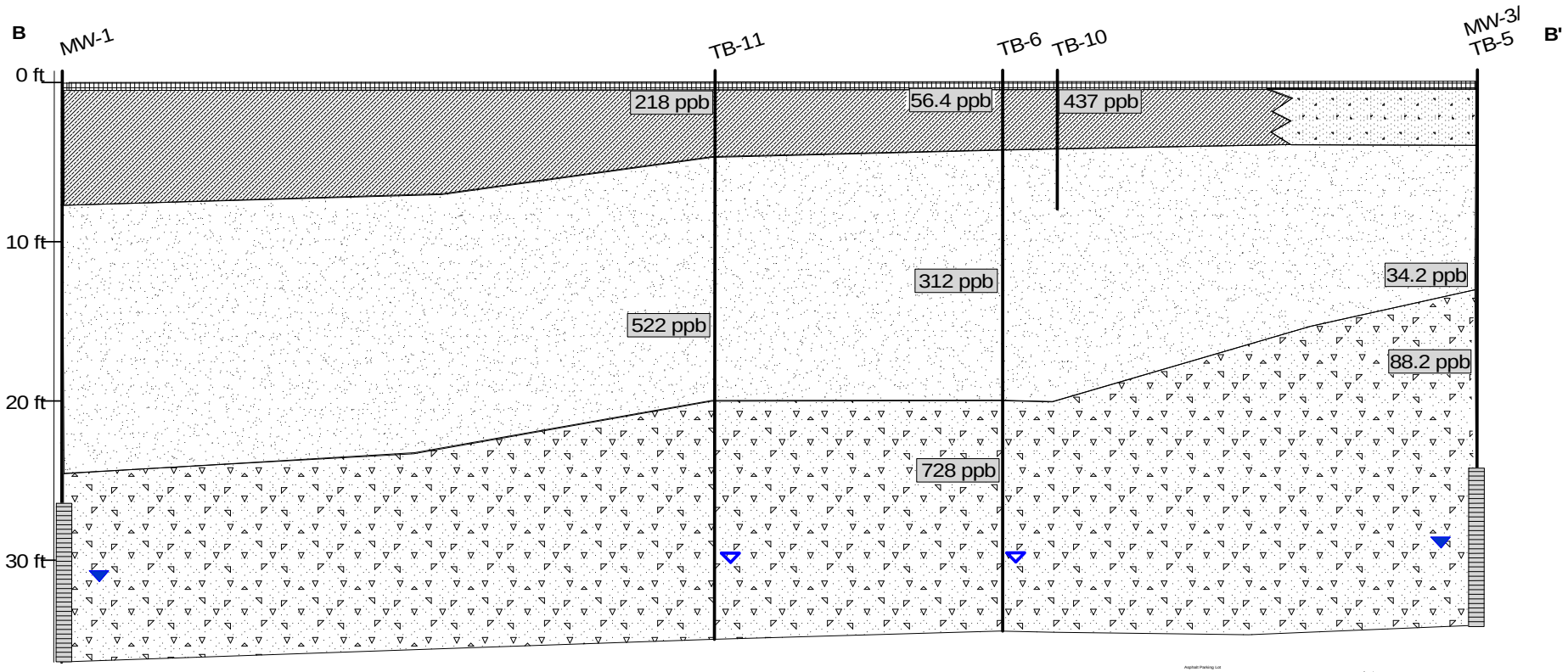
Geologic Fence Diagram A-A'

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Fence Diag A-A'
Date:	December 22, 2009
Scale:	As Shown
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

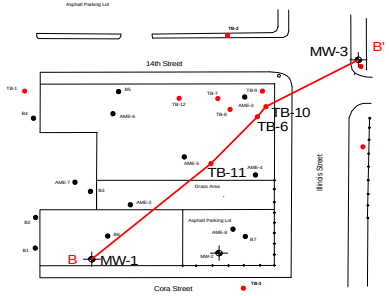
Figure: 4



Legend

- Silty clay loam
- Sandy loam
- Fill material
- Fine to coarse sand with gravel
- Fine to medium sand
- 728 ppb -- Soil PCE analytical data
- Encountered Groundwater Elevation
- Static Groundwater Elevation
- PVC Well Screen

Horizontal Scale 0 10 20 ft 50 ft



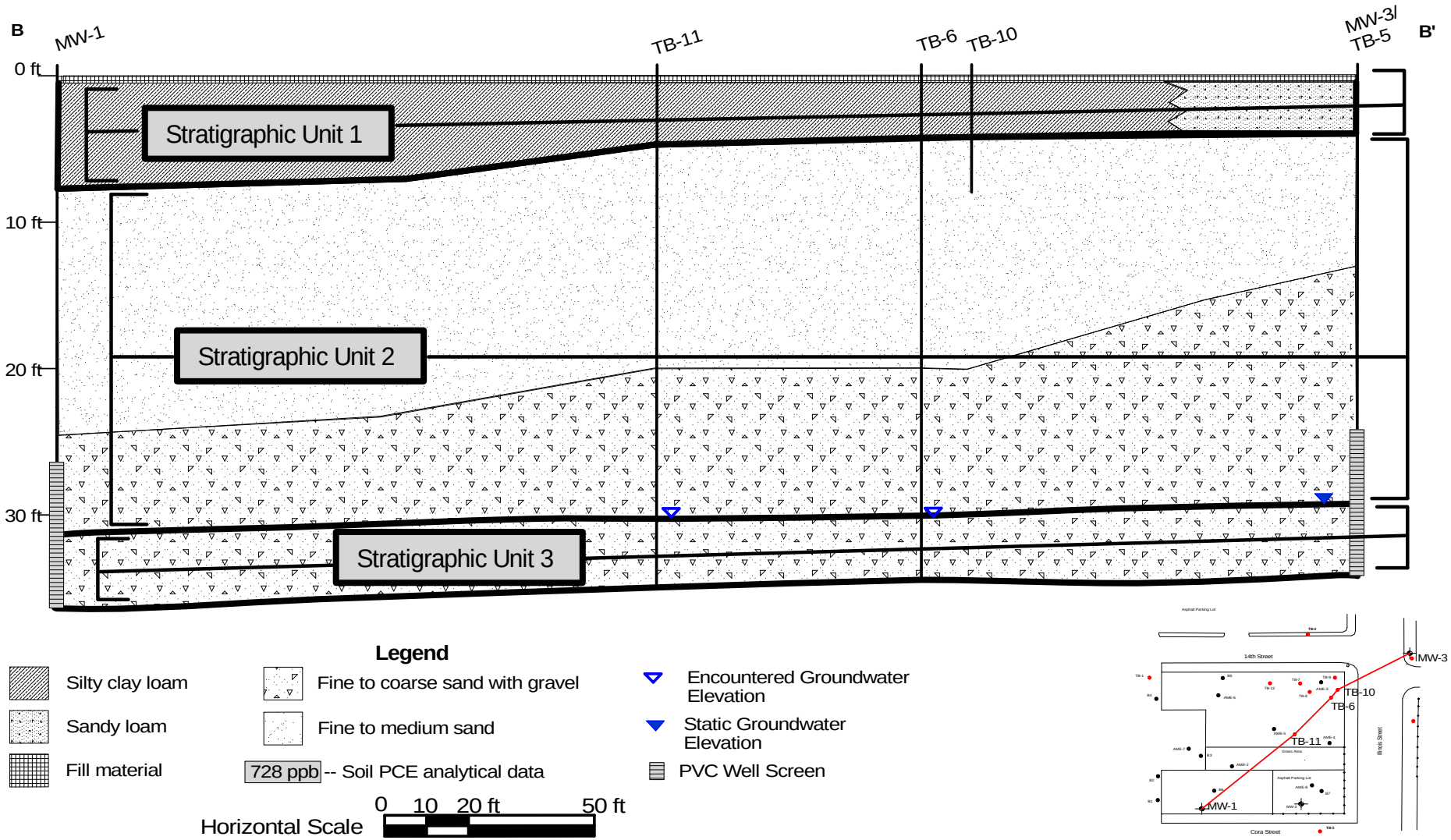
Geologic Fence Diagram B-B'

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Fence Diag B-B'
Date:	December 22, 2009
Scale:	As Shown
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 5



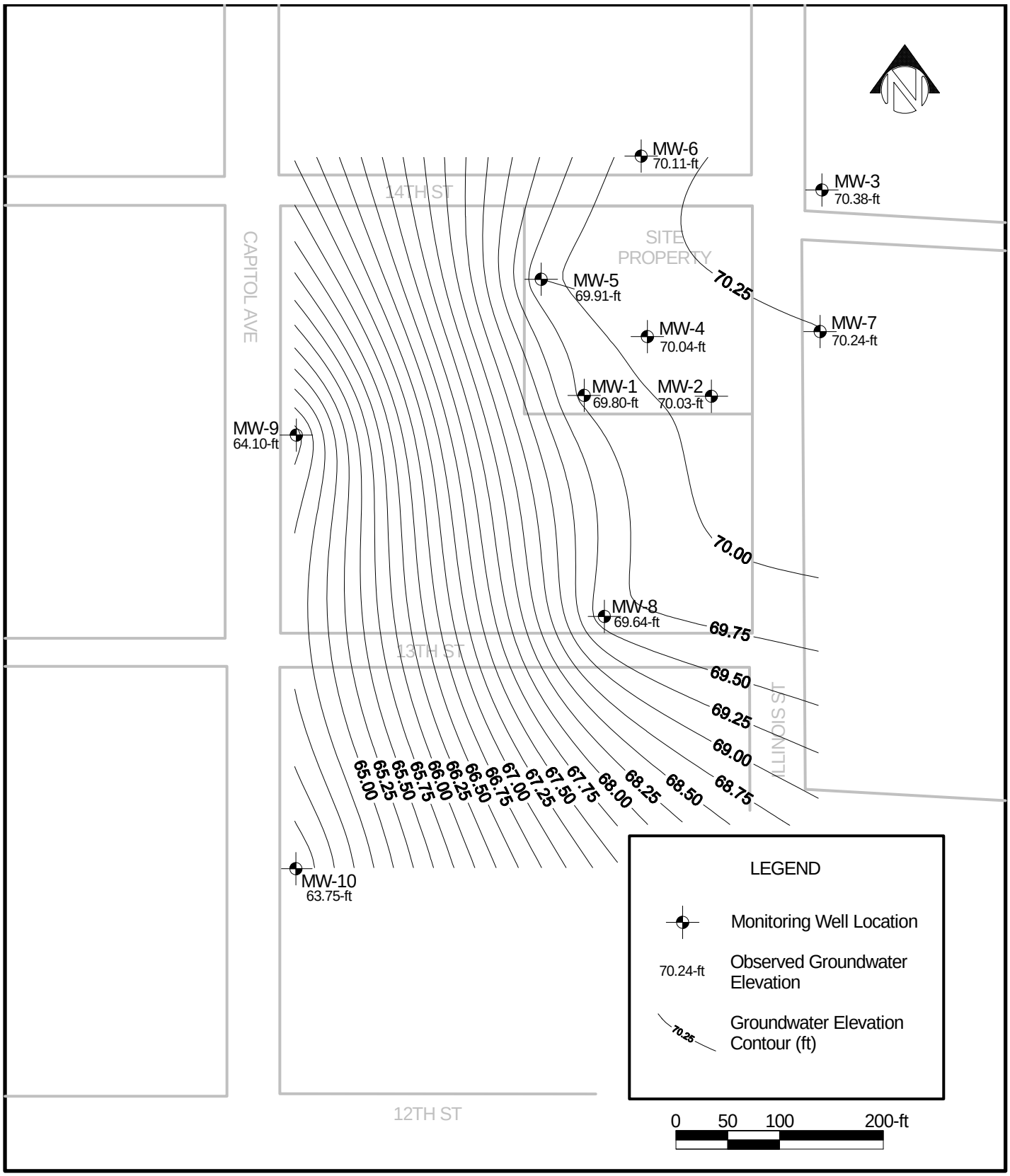
Definition of Stratigraphic Units

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Stratigraphic Units
Date:	December 22, 2009
Scale:	As Shown
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 6



Potentiometric Surface Map
December 7,2009

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Potentiometric
Date:	December 23, 2009
Scale:	As Shown
Drawn By: JF	*Checked by:* PT

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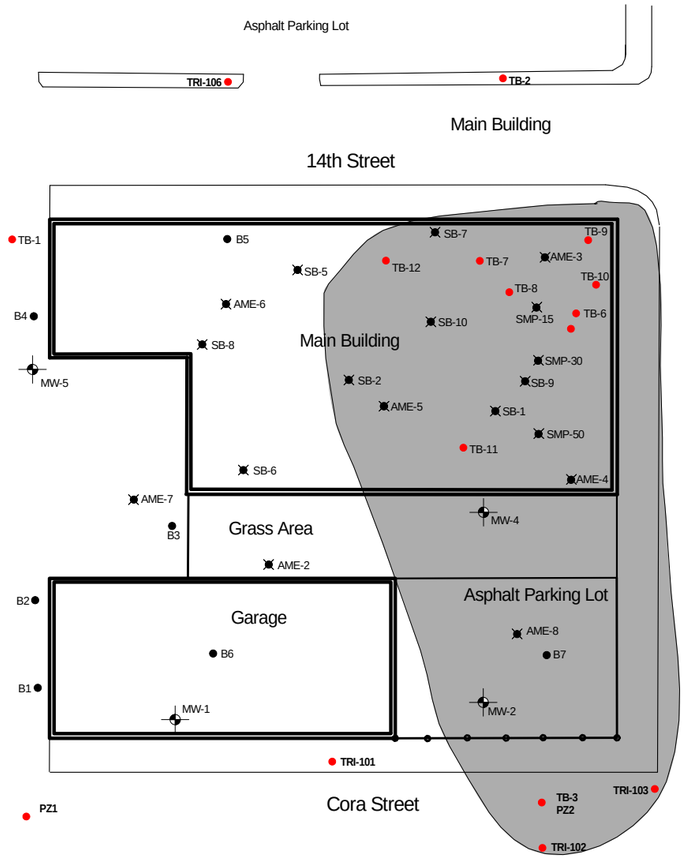
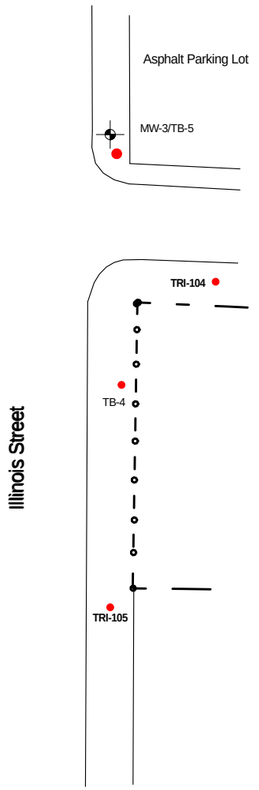
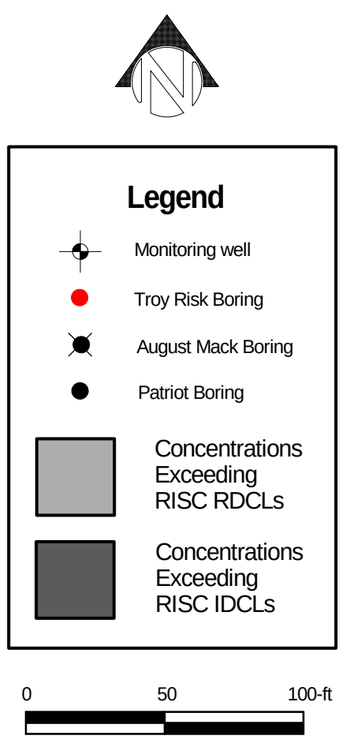
Figure: 7

PCE Impacts in Stratigraphic Unit #1







PCE Impacts in Stratigraphic Unit #2

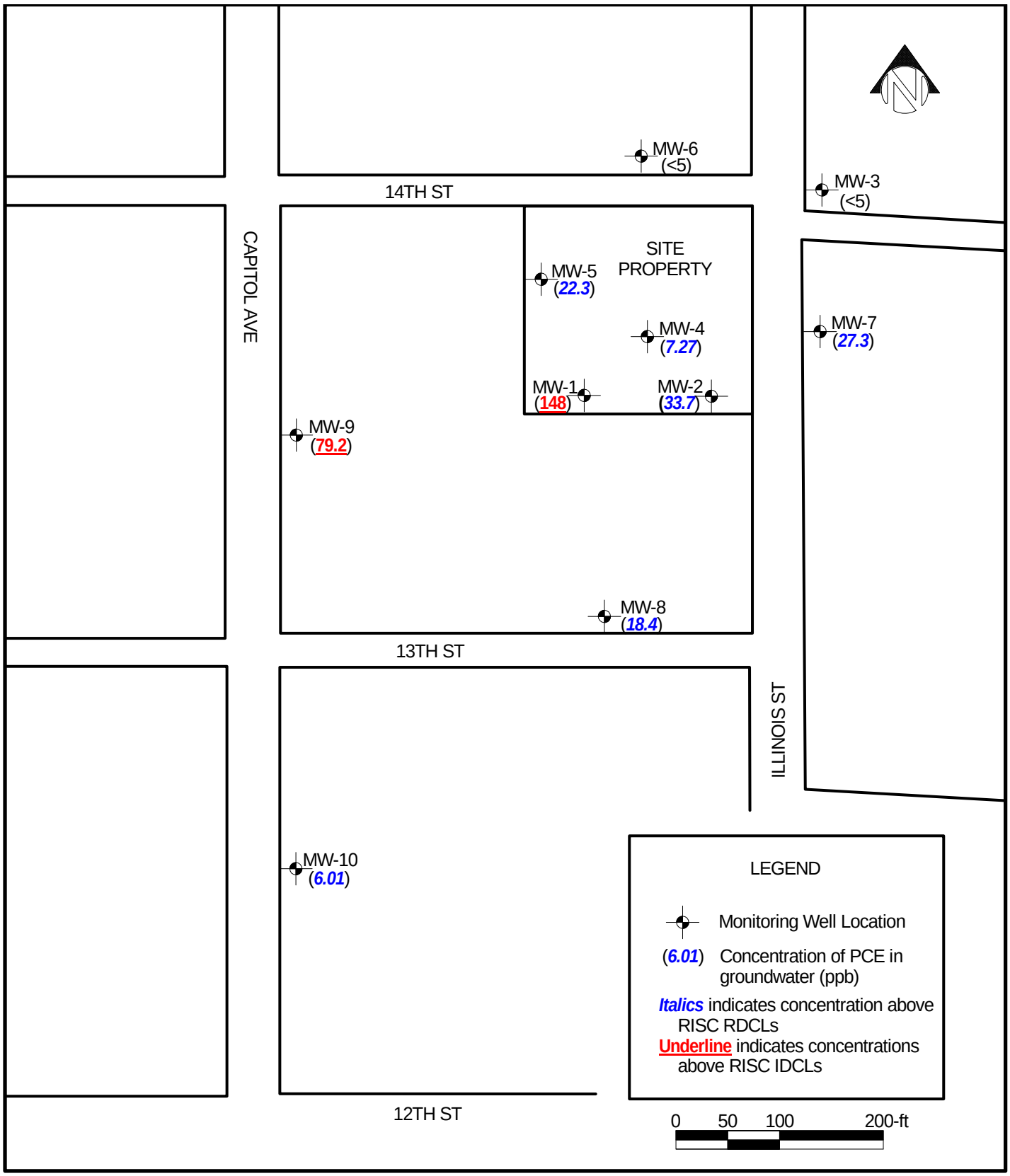
PCE Impacts to Vadose Zone Soil

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	PCE in Vadose
Date:	December 23, 2009
Scale:	As Shown
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 8



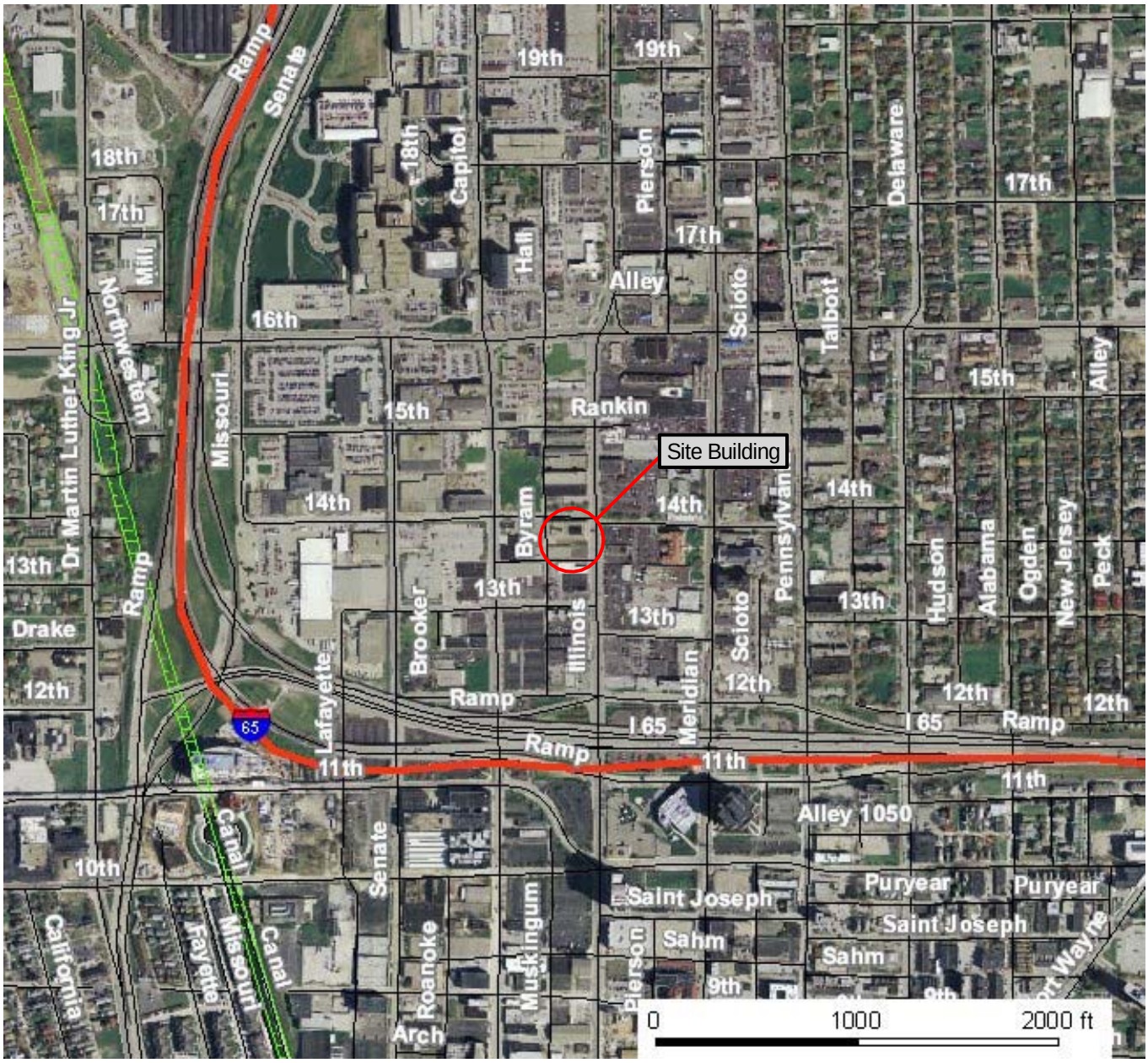
PCE Impacts to Groundwater - December 7,2009

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Groundwater
Date:	December 23, 2009
Scale:	As Shown
Drawn By: JF	Checked by: PT

TROY RISK INC.

Figure: 9



LEGEND

Wetland

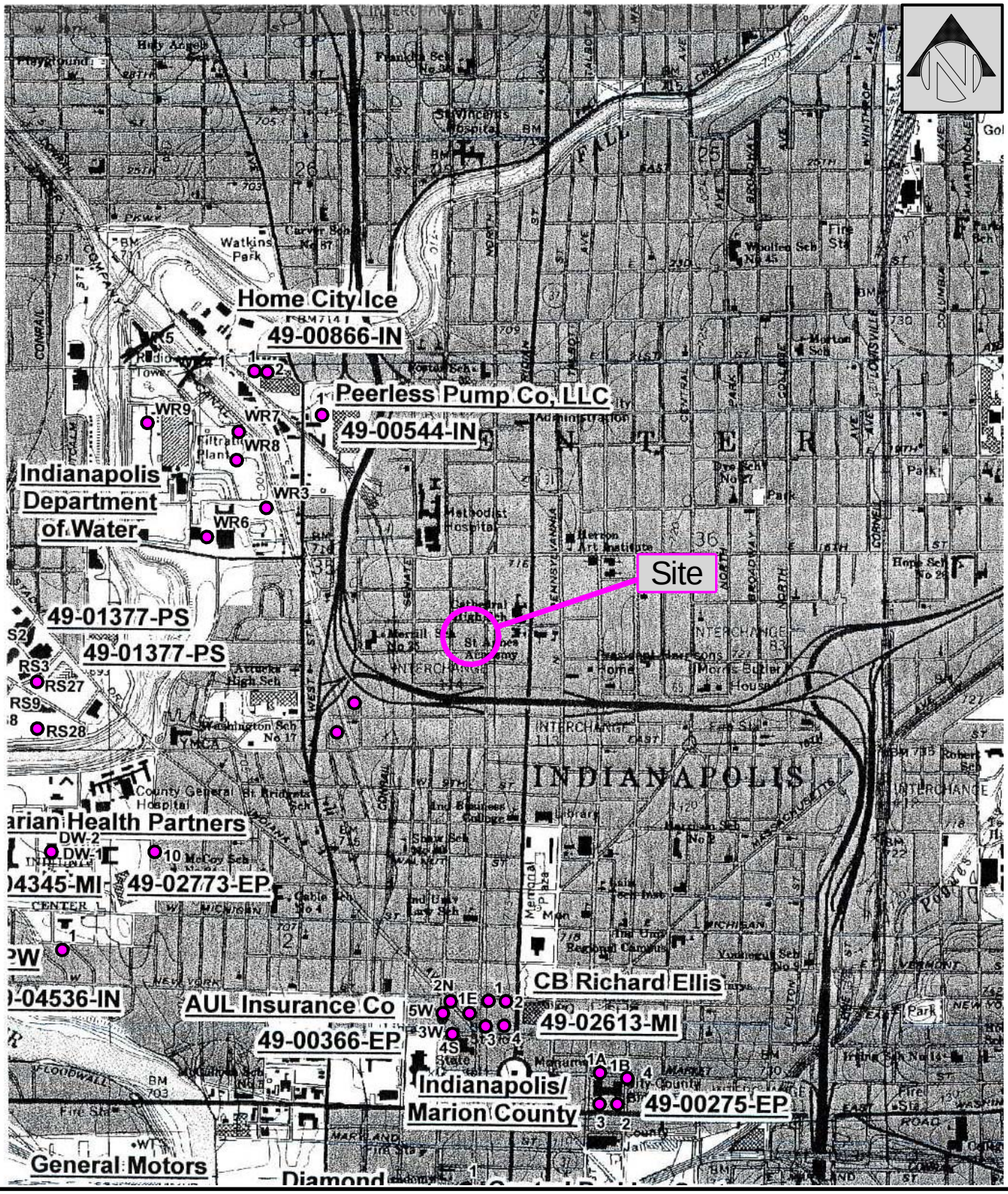
Wetlands Vicinity Map

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Wetlands
Date:	December 10, 2009
Scale:	As Shown
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 10



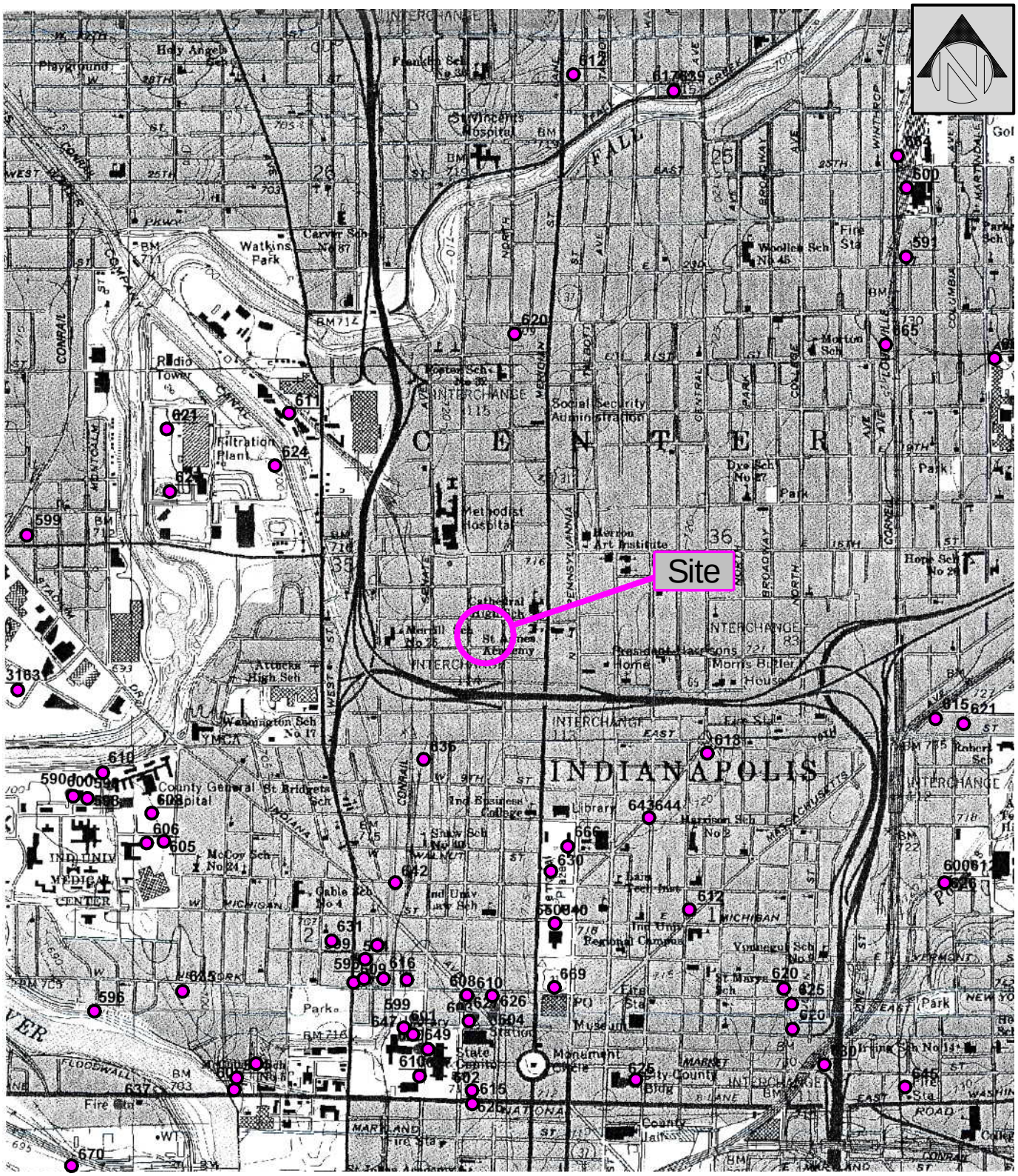
DRAFT High Capacity Water Well Map

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	High Cap Wells
Date:	December 10, 2009
Scale:	1" = 2000'
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 11



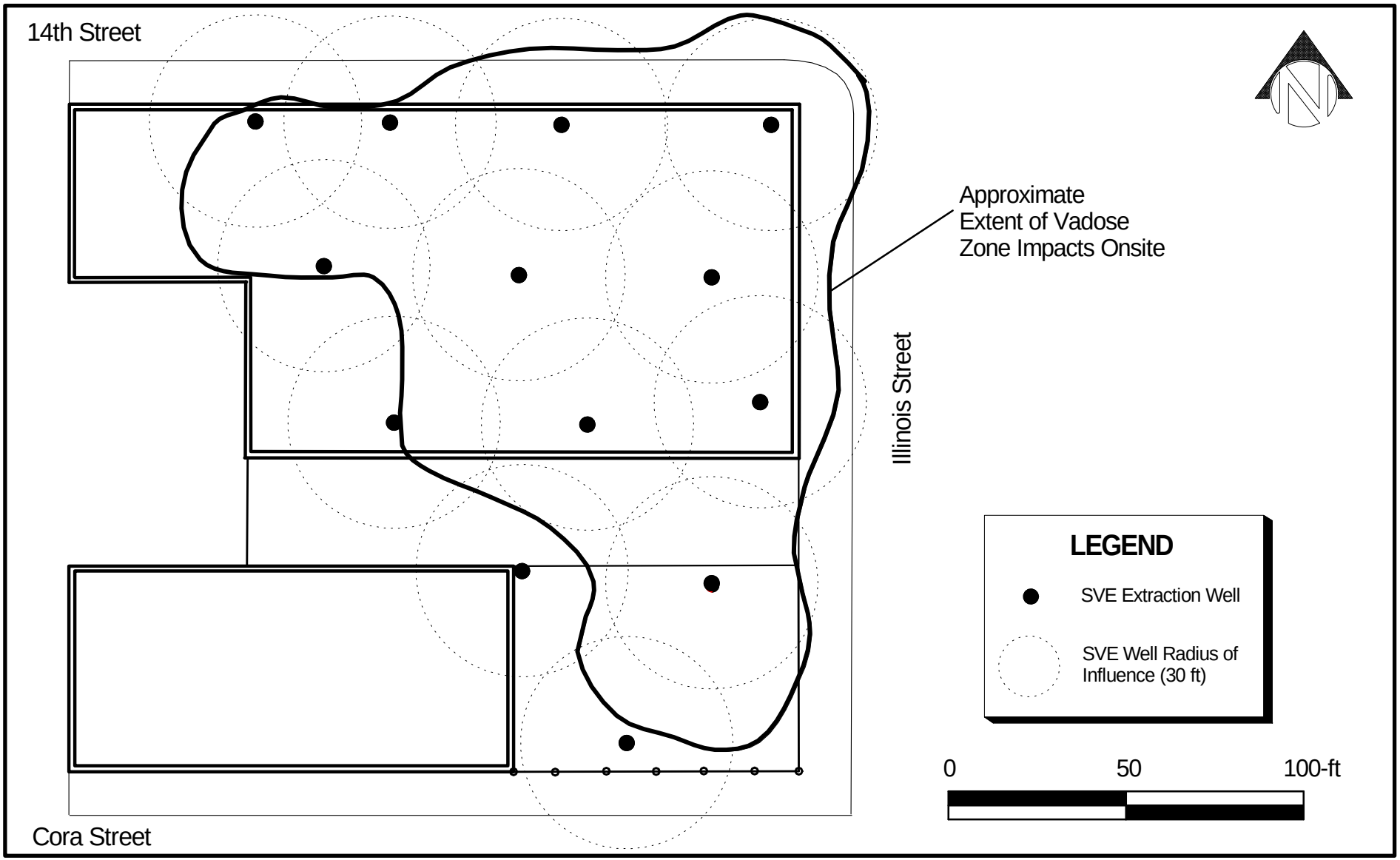
DRAFT Low Capacity Water Well Map

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	Low Cap Wells
Date:	December 10, 2009
Scale:	1" = 2000'
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 12



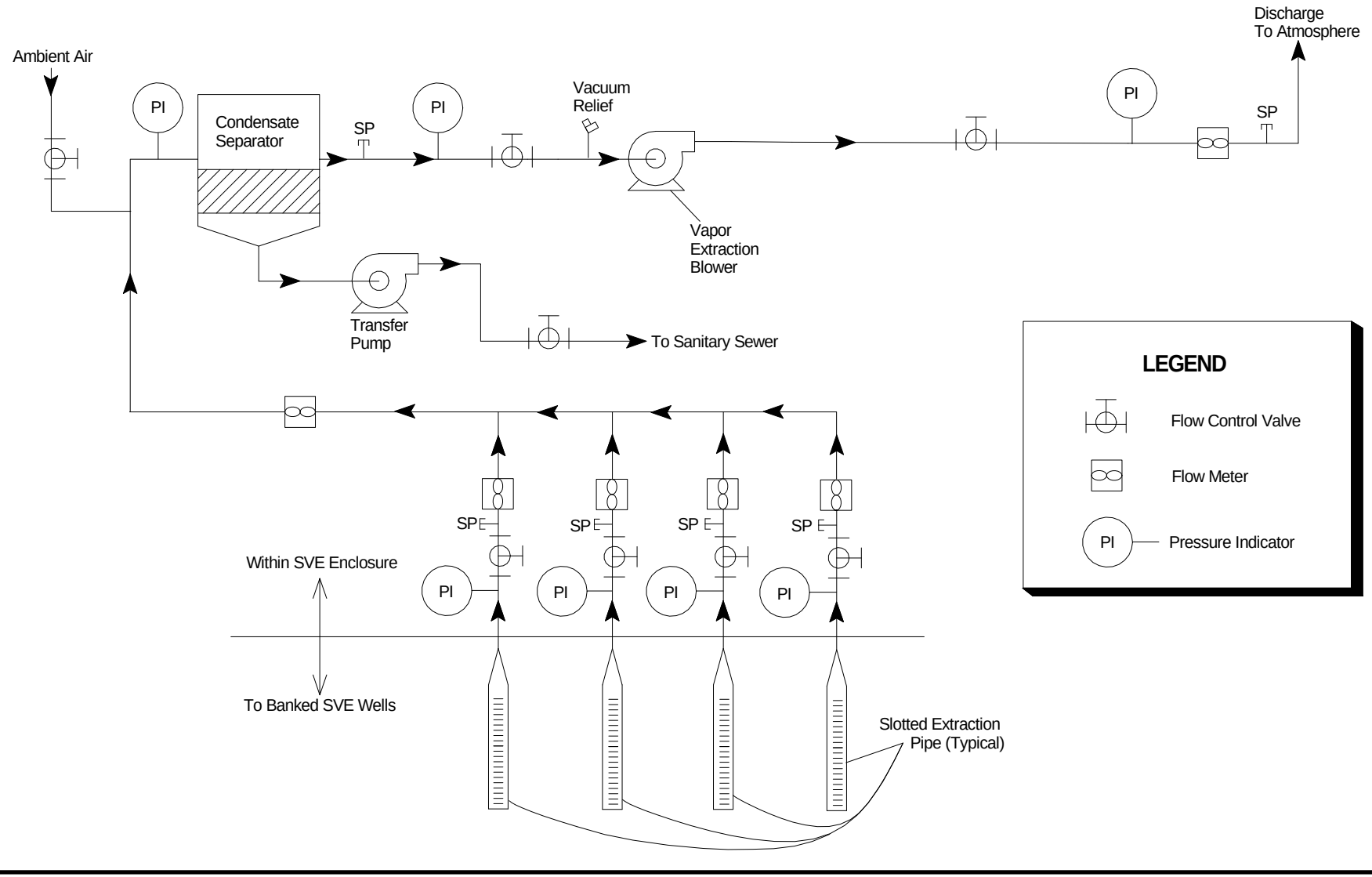
Layout of Soil Vapor Extraction System

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	SVE Layout
Date:	April 14, 2009
Scale:	1" = 40'
Drawn By: JF	*Checked by:* JAB

TROY RISK INC.

Figure: 13



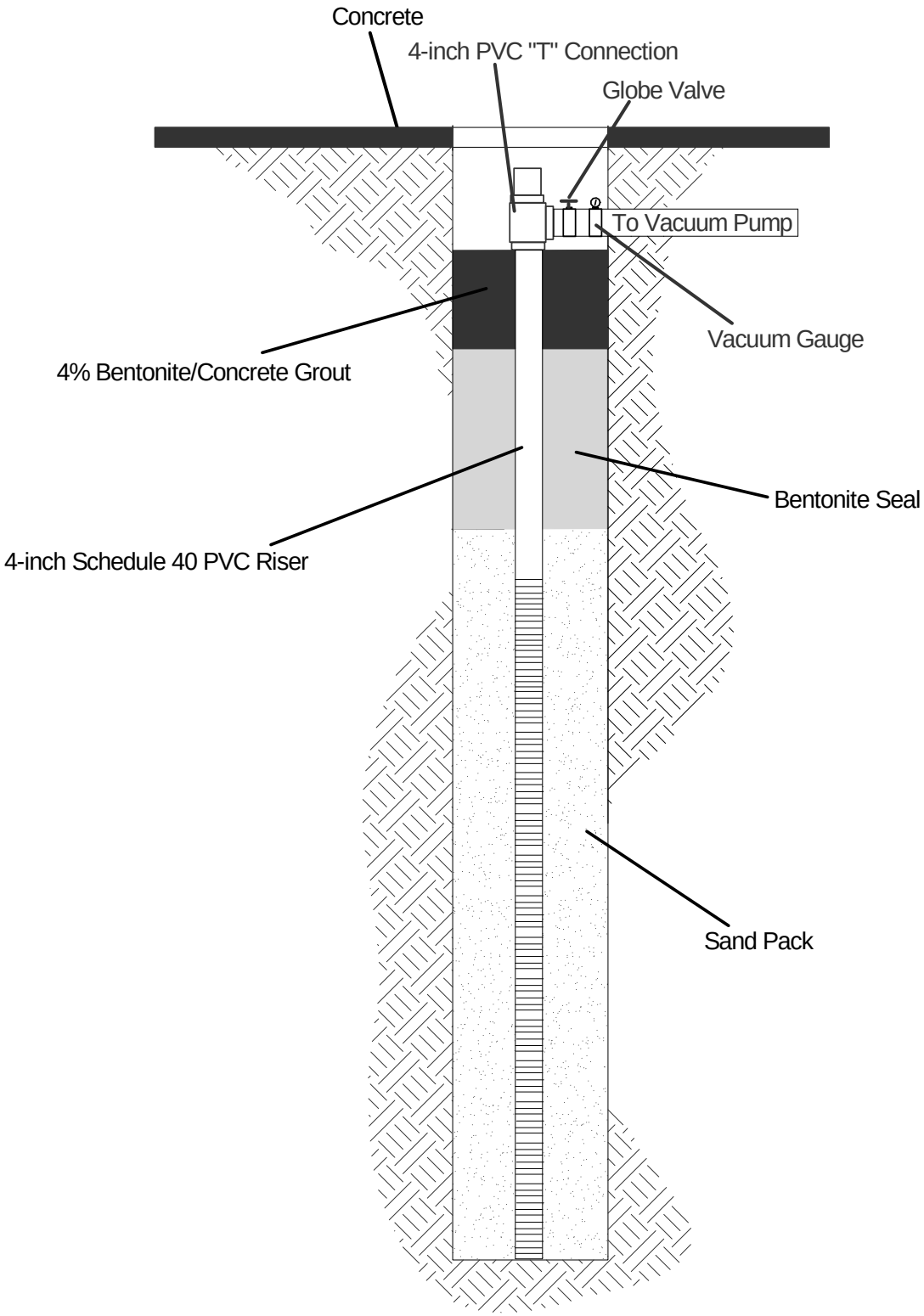
Process and Instrumentation Diagram of SVE System

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	P&ID
Date:	April 14, 2009
Scale:	Not to Scale
Drawn By: JF	*Checked by:* JAB

TROY RISK INC.

Figure: 14



General SVE Well Construction Diagram

1352 North Illinois Street, LLP
1352 North Illinois Street
Indianapolis, Indiana

Project Number:	100.05.08
Drawing File:	SVE Well Const
Date:	December 11, 2009
Scale:	Not to Scale
Drawn By: JF	*Checked by:* PT

TROY RISK INC.

Figure: 15

TABLES

Table 1
Groundwater Elevation Data
1352 North Illinois Street, LLP
1352 North Illinois Street, Indianapolis, IN
TRI Project No. 100.05.08

Well ID	Top of Casing (ft)	Gauging Date	Depth to Water (ft)	Water Level (ft)
MW-1	101.01	1/27/2007	29.35	71.66
		4/10/2009	31.10	69.91
		6/9/2009	29.45	71.56
		9/8/2009	30.32	70.69
		12/7/2009	31.21	69.80
		12/8/2009	31.21	69.80
MW-2	100.67	1/27/2007	28.81	71.86
		4/10/2009	30.55	70.12
		6/9/2009	28.86	71.81
		9/8/2009	29.73	70.94
		12/7/2009	30.63	70.04
		12/8/2009	30.64	70.03
MW-3	100.03	1/27/2007	27.75	72.28
		4/10/2009	29.57	70.46
		6/9/2009	27.82	72.21
		9/8/2009	28.70	71.33
		12/7/2009	29.64	70.39
		12/8/2009	29.65	70.38
MW-4	101.44	1/27/2007	29.51	71.93
		4/10/2009	31.28	70.16
		6/9/2009	29.62	71.82
		9/8/2009	30.42	71.02
		12/7/2009	31.40	70.04
		12/8/2009	31.40	70.04
MW-5	100.56	1/27/2007	28.72	71.84
		4/10/2009	30.51	70.05
		6/9/2009	28.80	71.76
		9/8/2009	29.76	70.80
		12/7/2009	30.65	69.91
		12/8/2009	30.65	69.91
MW-6	99.84	5/29/2009	28.05	71.79
		6/9/2009	27.90	71.94
		9/8/2009	28.81	71.03
		12/7/2009	29.73	70.11
		12/8/2009	29.73	70.11
MW-7	99.25	5/29/2009	27.39	71.86
		6/9/2009	27.24	72.01
		9/8/2009	28.08	71.17
		12/7/2009	29.01	70.24
		12/8/2009	29.01	70.24
MW-8	99.12	5/29/2009	27.91	71.21
		6/9/2009	27.73	71.39
		9/8/2009	28.53	70.59
		12/7/2009	29.50	69.62
		12/8/2009	29.48	69.64
MW-9	94.46	5/29/2009	28.67	65.79
		6/9/2009	28.54	65.92
		9/8/2009	29.54	64.92
		12/7/2009	30.36	64.10
		12/8/2009	30.36	64.10
MW-10	93.82	5/29/2009	28.47	65.35
		6/9/2009	28.37	65.45
		9/8/2009	29.26	64.56
		12/7/2009	30.09	63.73
		12/8/2009	30.07	63.75

Table 2
Aquifer Permeability Estimates
1352 North Illinois Street, LLP
1352 North Illinois Street, Indianapolis, IN
TRI Project No. 100.05.08

Monitoring Well ID	Hydraulic Conductivity ($cm\ sec^{-1}$)[1]	Intrinsic Permeability (cm^2)
MW-2	2.66×10^{-2}	2.71×10^{-7}
MW-3	3.07×10^{-2}	3.13×10^{-7}
MW-4	3.15×10^{-2}	3.21×10^{-7}
MW-5	5.72×10^{-2}	5.83×10^{-7}

Intrinsic Permeability = Hydraulic Conductivitiy ($cm\ sec^{-1}$) X 1.02×10^{-5} cm sec

1 - Calculated using Bouwer and Rice method under partial penetrating conditions

Table 3
Detections of Chemicals of Concern in Soil
1352 North Illinois Street, LLP
1352 North Illinois Street, Indianapolis, IN
TRI Project No. 100.05.08

Sample ID	Depth (ft)	Date	Tetrachloroethylene
B-1	21'	4/18/2002	ND
B-2	12'	4/19/2002	ND
B-3	12'	4/20/2002	ND
B-4	11'	4/21/2002	ND
B-5	12'	4/22/2002	**0.014**
B-6	12'	4/23/2002	**0.0067**
B-7	15'	4/24/2002	***0.058***
AME-1	12-14'	7/22/2004	**0.034**
	24-26'		**0.034**
AME-2	10-12'	7/22/2004	**0.0082**
	24-26'		***0.07***
AME-3	8-10'	7/22/2004	**0.86**
	20-22'		**1.3**
(Duplicate)	20-22'		**5.5**
AME-4	4-6'	7/22/2004	**0.056**
	20-22'		***0.16***
AME-5	4-6'	7/23/2004	***0.55***
	10-12'		***0.16***
AME-6	2-4'	7/23/2004	**0.04**
	14-16'		***0.52***
AME-7	12-14'	7/23/2004	**0.013**
	18-20'		**0.035**
AME-8	6-8'	7/23/2004	***0.14***
	14-16'		**0.055**
TB-1	8-10'	10/25/2005	**0.00998**
	18-20'		**0.0314**
TB-2	12.5-15'	10/25/2005	**0.0317**
	20-22.5'		**0.0242**
TB-3	10-12'	10/27/2005	**0.0238**
	20-22'		***0.0823***
TB-4	10-12'	10/27/2005	**0.0324**
	17-18'		**0.0438**
TB-5	10-12'	10/27/2005	**0.0342**
	17-19'		***0.0882***
TB-6	0-1'	11/9/2005	**0.0564**
	12-14'		***0.312***
	24.5-25.5		***0.728***
TB-7	12-14'	11/9/2005	***0.122***
TB-8	4"-1'	11/9/2005	***0.258***
TB-9	4"-1'	11/9/2005	***0.1084***
	4-5'		**0.0973**
TB-10	4"-1'	11/9/2005	***0.437***
TB-11	4"-6"	11/9/2005	***0.218***
	16-18'		***0.522***
TB-12	4"-1'	11/9/2005	**1.144**
	24-26'		**0.0115**
SB-1	2-4'	5/26/2006	***0.198***
SB-2	2-4'	5/26/2006	<0.006
SB-3	2-4'	5/26/2006	**0.048**
SB-4	2-4'	5/26/2006	***0.38***
SB-5	2-4'	5/26/2006	**0.022**
SMP-15	14-16'	5/26/2006	**0.36**
SMP-30	12-14'	5/26/2006	**1.12**
SMP-50	2-4'	5/26/2006	**0.007**
	14-16'		**0.975**
TRI-101	2-4'	1/6/2009	**0.023**
	18-20'		***0.216***
TRI-102	4-6'	1/6/2009	***0.101***
	16-17'		***0.257***
TRI-103	4-5.5'	1/6/2009	***0.140***
	20-21.5'		***0.299***
TRI-104	4-5'	1/7/2009	**0.052**
	22-24'		***0.082***
TRI-105	4-6'	1/7/2009	**0.011**
	14-16'		***0.085***
TRI-106	2-4'	1/7/2009	**0.006**
	14-16'		***0.089***
MW-6	25-27'	5/27/2009	<0.005
MW-7	26-28'	5/27/2009	**7.917**
MW-8	12-14'	5/28/2009	<0.005
MW-9	24-26'	5/28/2009	***0.0728***
MW-10	18-20'	5/28/2009	<0.005
RISC Residential Default Closure Level			***0.058***
RISC Industrial Default Closure Level			**0.64**

Bold - Detection above Laboratory method detection limit
Italicized - Exceed RISC Residential Default Closure Level
Underline - Exceed RISC Commercial Default Closure Level
ND - Not Detected
Analytical data given in mg kg^{-1}

Table 4
Detections of Chemicals of Concern in Groundwater
1352 North Illinois Street, LLP
1352 North Illinois Street, Indianapolis, IN
TRI Project No. 100.05.08

Sample ID	Date	Tetrachloroethylene	Trichloroethylene	Chloroform
B-1	4/18/2002	**69**	<5	<5
B-5	4/18/2002	**73**	<5	<5
B-7	4/18/2002	**69**	<5	**10**
AME MW-1	7/26/2004	**190**	<1	**2.8**
AME MW-2	7/26/2004	**180**	**1.2**	**7.7**
AME-2	7/23/2004	**130**	<1	**2.7**
AME-3	7/23/2004	**190**	**2.1**	**2.7**
AME-4	7/23/2004	***39***	<1	**3.5**
TB-1W	10/25/2005	***15.5***	***18.9***	<5
TB-2W	10/27/2005	<5	<5	<5
TB-3W	10/25/2005	***12.7***	<5	<5
TB-4W	10/25/2005	***21.7***	<5	<5
TB-5W	10/27/2005	***6.75***	<5	<5
TB-6W	11/9/2005	**531**	<5	<5
(Duplicate)		**474**	<5	<5
TB-7W	11/9/2005	**219**	<5	<5
TB-11W	11/9/2005	**525**	<5	<5
TB-12W	11/9/2005	**66.6**	<5	<5
PZ-1	1/26/2007	**170**	<5	<5
PZ-2	1/26/2007	**257**	<5	<5
PZ-3	1/26/2007	**55.2**	<5	<5
TRI-13 50'	12/14/2007	***13.2***	<5	<5
TRI-14 35'	12/14/2007	**64.4**	<5	<5
TRI-14 50'	12/14/2007	***32.4***	<5	<5
TRI-15 35'	12/14/2007	**122**	<5	<5
TRI-15 50'	12/14/2007	***7.83***	<5	<5
TRI-16 35'	12/14/2007	<5	<5	**12.4**
TRI-16 50'	12/14/2007	<5	<5	<5
TRI-17 35'	12/14/2007	<5	<5	<5
TRI-17 50'	12/14/2007	<5	<5	<5
MW-1	11/9/2005	**143**	<5	<5
	1/26/2007	**187**	<5	<5
	4/10/2009	**203**	<5	<5
	9/9/2009	**171**	<5	<5
	12/7/2009	**148**	<5	<5
(Duplicate)		**137**	<5	<5
MW-2	11/9/2005	**138**	<5	<5
	1/26/2007	**148**	<5	<5
	4/10/2009	**75.3**	<5	<5
	9/9/2009	**31.2**	<5	<5
	12/7/2009	**33.7**	<5	<5
MW-3	10/31/2005	***9.53***	<5	<5
(Duplicate)		***7.11***	<5	<5
	1/26/2007	***11.6***	<5	<5
	4/10/2009	<5	<5	<5
	9/9/2009	<5	<5	<5
	12/7/2009	<5	<5	<5
MW-4	1/26/2007	**145**	<5	<5
	4/10/2009	***33.2***	<5	<5
	9/9/2009	<5	<5	<5
	12/7/2009	***7.27***	<5	<5
MW-5	1/26/2007	***47.3***	<5	<5
	4/10/2009	***40.4***	<5	<5
	9/9/2009	***11.7***	<5	<5
	12/7/2009	***22.3***	<5	<5
MW-6	6/9/2009	***15.0***	<5	<5
	9/9/2009	<5	<5	<5
	12/7/2009	<5	<5	<5
MW-7	6/9/2009	***23.5***	<5	<5
	9/9/2009	***22.7***	<5	<5
	12/7/2009	***27.3***	<5	<5
MW-8	6/9/2009	***18.7***	<5	<5
	9/9/2009	***14.9***	<5	<5
	12/7/2009	***18.4***	<5	<5
MW-9	6/9/2009	**76.2**	<5	<5
	9/9/2009	**99.1**	<5	<5
	12/7/2009	**79.2**	<5	<5
MW-10	6/9/2009	***12.5***	<5	<5
	9/9/2009	<5	<5	<5
	12/7/2009	***6.01***	<5	<5
RISC Residental Default Closure Levels		***5***	***5***	***80***
RISC Industrial Default Closure Levels		**55**	**31**	**1,000**

Bold - Detection above Laboratory method detection limit
Italicized - Exceed RISC Residential Default Closure Level
Underline - Exceed RISC Commercial Default Closure Level
Analytical data given in ug L^{-1}

Table 5
DRAFT (12/23/2009) Proposed Closure Objectives
1352 North Illinois Street, LLP
1352 North Illinois Street, Indianapolis, IN
TRI Project No. 100.05.08

Primary Cleanup Objectives

Media	Location	Tetrachloroethylene	Trichloroethylene	Chloroform
Surface Soil (0-0.5 ft bgs)	On-Site (mg kg^{-1})	9.9	4.9	3
(Direct Contact Exposure)	Off-Site (mg kg^{-1})	9.9	4.9	3
Subsurface Soil (0.5 - 6 ft bgs)	On-Site (mg kg^{-1})	0.058	0.057	0.47
(Migration to Groundwater Exposure)	Off-Site (mg kg^{-1})	0.058	0.057	0.47
Groundwater	On-Site (mg L^{-1})	Plume Stability Monitoring		
(Groundwater Exposure)	Off-Site (mg L^{-1})	Plume Stability Monitoring		

Residential closure levels are proposed onsite and offsite properties
Closure levels selected for identified exposure scenario from RISC Technical Manual, revised 5/1/2009
Closure objectives are proposed for all VOCs; however, only CoCs found to exceed method detection limits are listed
Execution of a deed restriction to prevent the future use of groundwater at the Site will be required for closure with Plume Stability Monitoring

Secondary (Contingency) Cleanup Objectives

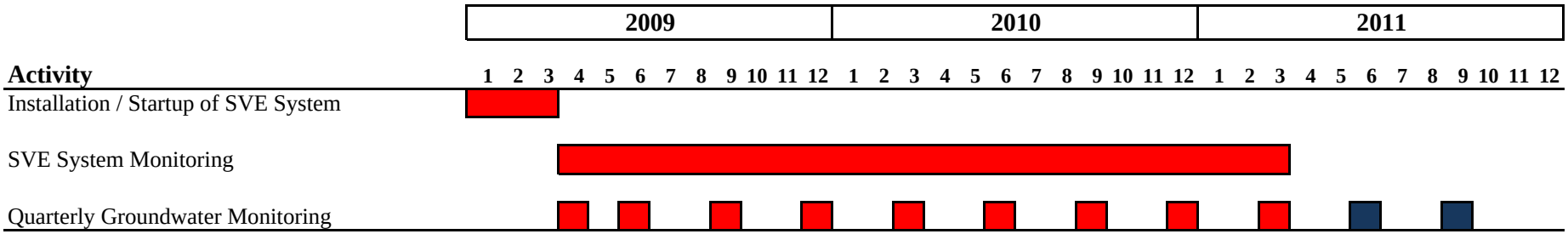
Media	Location	Tetrachloroethylene	Trichloroethylene	Chloroform
Surface Soil (0-0.5 ft bgs)	On-Site (mg kg^{-1})	9.9	4.9	3
(Direct Contact Exposure)	Off-Site (mg kg^{-1})	9.9	4.9	3
Subsurface Soil (0.5 - 6 ft bgs)	On-Site (mg kg^{-1})	Plume Stability Monitoring		
	Off-Site (mg kg^{-1})	Plume Stability Monitoring		
Groundwater	On-Site (mg L^{-1})	Plume Stability Monitoring		
	Off-Site (mg L^{-1})	Plume Stability Monitoring		

Table 6
Site Monitoring Schedule
1352 North Illinois Street, LLP
1352 North Illinois Street, Indianapolis, IN
TRI Project No. 100.05.08

Sample Location	Quarterly Groundwater Monitoring	Parameters	MPE System Monitoring (biweekly to monthly)	Parameters
MW-1	X	1,2		
MW-2	X	1,2		
MW-3	X	1,2		
MW-4	X	1,2		
MW-5	X	1,2		
MW-6	X	1,2		
MW-7	X	1,2		
MW-8	X	1,2		
MW-9	X	1,2		
MW-10	X	1,2		
SVE System Vapor Influent/Effluent	X	3,4,5	X	3,4,5,6

X - Sampled
1 - Dissolved VOCs by SW 846 Method 8260B
2 - Water Level
3 - System Vacuum
4 - VOC Concentration in Vapor Phase (Photoionization Detector)
5 - System Vapor Flow Rate (Dedicated Flow Meter)
6 - VOC Concentration in Vapor Phase (Method TO-15; analysis performed periodically)

Table 7
Timetable for RWP Implementation
1352 North Illinois Street, LLP
1352 North Illinois Street, Indianapolis, IN
TRI Project No. 100.05.08



- Proposed scope of work

- Additional work that may be required based upon observed contaminant removal efficiencies

APPENDIX A

METHODS AND MATERIALS

Troy Risk, Inc.

1.0 GEOPROBE INVESTIGATIONS

Soil borings were completed using either a hand auger or a Geoprobe drill rig equipped with both macro-core and a duel tube sampling systems. This sampling device consists of a 4-ft long, 1.25-inch diameter, stainless steel tube that is hydraulically driven into the subsurface. Soil samples were collected inside an acetate liner, which was removed after retrieval of the sampler at the surface.

A field scientist inspected each soil core in the field for physical evidence of contamination such as staining, odors, free product, etc. In addition, each soil core was split into two-foot intervals (0.0 to 2.0 ft, 2.0 to 4.0 ft, etc.). Each two-foot interval was divided into two aliquots. The first aliquot was used to determine the emission of total photoionizable vapors (TPVs) using a MiniRae 2000 Photoionization Air Monitor equipped with an 10.2 eV lamp, which measures TPVs in parts per million (ppm). The second aliquot was immediately placed in an appropriate laboratory-grade container, and stored in a cooler with ice. The second aliquot of selected soil samples was submitted to an environmental analytical laboratory (Envision Laboratories, Inc., 1439 Sadlier Circle West Drive, Indianapolis, IN, 46239) for analysis. All soil samples collected after May 1, 2007 were collected using 5035 sampling protocols when volatile constituents were intended for analysis. All drilling and sampling equipment that entered a borehole was cleaned prior to advancing the subsequent boring. The equipment was washed in a non-phosphate detergent solution and rinsed with tap water.

2.0 MONITORING WELLS INSTALLATION / SAMPLING

Monitoring wells at the installed at the Site were installed into Stratigraphic Unit #2 at locations designated on Figure 9.

2.1 Well Installation

The monitoring wells consisted of 2-inch diameter schedule 40 PVC pre-pack monitoring wells, and were installed using a track-mounted Geoprobe drill rig, equipped with a 4.25-inch hollow-stem auger. A sand filter pack was placed in the borehole annulus to a depth of 2-feet above the well screen. The wells were completed to the surface with concrete grout. The top of the wells have a flush manhole with a bolt-down lid and locking gripper plug. After installation, the monitoring wells were developed to remove fine silt and borehole smearing effects. Approximately 3 well volumes of water were developed from each well. Development water was drummed and appropriately disposed of off-Site.

2.2 Monitoring Well Gauging and Sampling

The top of each monitoring well's casing elevation was surveyed relative to an arbitrary 100 ft value. After installation, the monitoring wells were allowed to stabilize for at least 72 hours. After stabilization, the static water level was gauged to a precision of 0.01 inches using an electronic water gauge. The depth to water readings were then combined with surveyed casing elevations to determine groundwater flow direction.

After the monitoring wells were gauged, the entire monitoring well network was sampled in accordance with IDEM guidance. Groundwater samples were collected in approved containers and were submitted to an analytical laboratory following chain-of-custody protocols.

3.0 SLUG TESTING

Slug tests were performed by Troy Risk using the methodology outlined in EPA SOP#2046, using a pressure transducer in conjunction with an automated data logger. Briefly, the slug test is conducted by quickly adding a slug (known volume) of water to a well and then measuring the time required for the water level to return to its static level. The rate at which the well re-establishes equilibrium (i.e., static water levels) enables one to calculate the hydraulic conductivity (K) for the immediate area surrounding the well screen. Hydraulic conductivity is a coefficient of proportionality describing the rate at which water can move through a permeable medium, given a known head pressure.

APPENDIX B

(Not Included in this Draft)

APPENDIX C

(Not Included in this Draft)

APPENDIX D

Appendix D

Groundwater Velocity Calculation

Using the estimated hydraulic conductivities, hydraulic gradients, and porosity on site, groundwater velocities were calculated based on the equation:

$$v = \frac{K\left(\frac{cm}{s}\right) * \Delta\left(\frac{ft}{ft}\right) * \frac{ft}{30.48\ cm}}{\eta}$$

where v = groundwater velocity (ft/s)
K = Hydraulic conductivity
Δ = Hydraulic gradient
η = porosity (unitless)

This calculation yielded groundwater velocities in feet per second. These velocities were then converted to feet per year.

The following example shows the calculation of the groundwater velocity for MW-3.
K = 1.26×10^{-2} to 7.94×10^{-3} cm/s, Δ = 0.002 ft/ft, η = 0.25 to 0.35

$$v = \frac{1.26 \times 10^{-2}\frac{cm}{s} * 0.002\ \frac{ft}{ft} * \frac{ft}{30.48\ cm}}{0.25} = 3.30 \times 10^{-6}\frac{ft}{s}$$

$$v = 3.30 \times 10^{-6}\frac{ft}{s} * \frac{60\ s}{min} * \frac{60\ min}{hr} * \frac{24\ hr}{day} * \frac{365\ day}{yr} = \mathbf{104.20}\frac{\boldsymbol{ft}}{\boldsymbol{yr}}$$

APPENDIX E

(Not Included in this Draft)

APPENDIX F

Material Safety Data Sheet
Tetrachloroethylene

ACC# 22900

Section 1 - Chemical Product and Company Identification

MSDS Name: Tetrachloroethylene
Catalog Numbers: C182 20, C182 4, C182-20, C182-4, C18220, C1824, O4586 4, O4586-4, O45864
Synonyms: Ethylene tetrachloride; Tetrachlorethylene; Perchloroethylene; Perchlorethylene
Company Identification:

Fisher Scientific
1 Reagent Lane
Fair Lawn, NJ 07410

For information, call: 201-796-7100
Emergency Number: 201-796-7100
For CHEMTREC assistance, call: 800-424-9300
For International CHEMTREC assistance, call: 703-527-3887

Section 2 - Composition, Information on Ingredients

CAS#	Chemical Name	Percent	EINECS/ELINCS
127-18-4	Tetrachloroethylene	99.0+	204-825-9

Hazard Symbols: XN N
Risk Phrases: 40 51/53

Section 3 - Hazards Identification

EMERGENCY OVERVIEW

Appearance: clear, colorless liquid. Irritant. May cause severe eye and skin irritation with possible burns. May cause central nervous system depression. May cause liver and kidney damage. May cause reproductive and fetal effects. May cause cancer based on animal studies. **Caution!** May cause respiratory tract irritation.
Target Organs: Kidneys, central nervous system, liver.

Potential Health Effects
Eye: Contact with eyes may cause severe irritation, and possible eye burns.
Skin: May cause severe irritation and possible burns.
Ingestion: May cause central nervous system depression, kidney damage, and liver damage. Symptoms may include: headache, excitement, fatigue, nausea, vomiting, stupor, and coma. May cause gastrointestinal irritation with nausea, vomiting and diarrhea.
Inhalation: Inhalation of vapor may cause respiratory tract irritation. May cause central nervous system effects including vertigo, anxiety, depression, muscle incoordination, and emotional instability.
Chronic: Possible cancer hazard based on tests with laboratory animals. Prolonged or repeated skin contact may cause defatting and dermatitis. May cause respiratory tract cancer. May cause

adverse nervous system effects including muscle tremors and incoordination. May cause liver and kidney damage. May cause reproductive and fetal effects.

Section 4 - First Aid Measures

Eyes: Flush eyes with plenty of water for at least 15 minutes, occasionally lifting the upper and lower eyelids. Get medical aid.
Skin: Get medical aid if irritation develops or persists. Wash clothing before reuse. Flush skin with plenty of soap and water.
Ingestion: If victim is conscious and alert, give 2-4 cupfuls of milk or water. Never give anything by mouth to an unconscious person. Get medical aid.
Inhalation: Remove from exposure and move to fresh air immediately. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Get medical aid.
Notes to Physician: Treat symptomatically and supportively.

Section 5 - Fire Fighting Measures

General Information: As in any fire, wear a self-contained breathing apparatus in pressure-demand, MSHA/NIOSH (approved or equivalent), and full protective gear. Containers may explode in the heat of a fire. Vapors may be heavier than air. They can spread along the ground and collect in low or confined areas.
Extinguishing Media: Substance is noncombustible; use agent most appropriate to extinguish surrounding fire. For small fires, use dry chemical, carbon dioxide, or water spray. For large fires, use dry chemical, carbon dioxide, alcohol-resistant foam, or water spray. Cool containers with flooding quantities of water until well after fire is out.
Flash Point: Not applicable.
Autoignition Temperature: Not applicable.
Explosion Limits, Lower: Not available.
Upper: Not available.
NFPA Rating: (estimated) Health: 2; Flammability: 0; Instability: 0

Section 6 - Accidental Release Measures

General Information: Use proper personal protective equipment as indicated in Section 8.
Spills/Leaks: Absorb spill with inert material (e.g. vermiculite, sand or earth), then place in suitable container. Avoid runoff into storm sewers and ditches which lead to waterways. Clean up spills immediately, observing precautions in the Protective Equipment section. Flush down the spill with a large amount of water. Remove all sources of ignition. Use a spark-proof tool. Provide ventilation.

Section 7 - Handling and Storage

Handling: Wash thoroughly after handling. Remove contaminated clothing and wash before reuse. Use with adequate ventilation. Do not reuse this container. Avoid breathing vapors from heated material. Avoid contact with skin and eyes. Keep container tightly closed. Keep away from flames

and other sources of high temperatures that may cause material to form vapors or mists.
Storage: Keep away from heat and flame. Store in a cool, dry place. Keep containers tightly closed.

Section 8 - Exposure Controls, Personal Protection

Engineering Controls: Use process enclosure, local exhaust ventilation, or other engineering controls to control airborne levels below recommended exposure limits.
Exposure Limits

Chemical Name	ACGIH	NIOSH	OSHA - Final PELs
Tetrachloroethylene	25 ppm TWA; 100 ppm STEL	150 ppm IDLH	100 ppm TWA; 200 ppm Ceiling

OSHA Vacated PELs: Tetrachloroethylene: 25 ppm TWA; 170 mg/m3 TWA
Personal Protective Equipment
Eyes: Wear appropriate protective eyeglasses or chemical safety goggles as described by OSHA's eye and face protection regulations in 29 CFR 1910.133 or European Standard EN166.
Skin: Wear appropriate protective gloves to prevent skin exposure.
Clothing: Wear appropriate protective clothing to prevent skin exposure.
Respirators: A respiratory protection program that meets OSHA's 29 CFR 1910.134 and ANSI Z88.2 requirements or European Standard EN 149 must be followed whenever workplace conditions warrant a respirator's use.

Section 9 - Physical and Chemical Properties

Physical State: Liquid
Appearance: clear, colorless
Odor: sweetish odor
pH: Not available.
Vapor Pressure: 15.8 mm Hg
Vapor Density: 5.2
Evaporation Rate:9 (ether=100)
Viscosity: 0.89 mPa s 20 deg C
Boiling Point: 121 deg C
Freezing/Melting Point:-22.3 deg C
Decomposition Temperature:150 deg C
Solubility: Nearly insoluble in water.
Specific Gravity/Density:1.623
Molecular Formula:C2Cl4
Molecular Weight:165.812

Section 10 - Stability and Reactivity

Chemical Stability: Stable under normal temperatures and pressures.
Conditions to Avoid: Incompatible materials, excess heat.
Incompatibilities with Other Materials: Strong bases, metals, liquid oxygen, dinitrogen tetroxide.

Hazardous Decomposition Products: Hydrogen chloride, phosgene, carbon monoxide, carbon dioxide.
Hazardous Polymerization: Will not occur.

Section 11 - Toxicological Information

RTECS#:
CAS# 127-18-4: KX3850000
LD50/LC50:
CAS# 127-18-4:
Draize test, rabbit, eye: 162 mg Mild;
Draize test, rabbit, eye: 500 mg/24H Mild;
Draize test, rabbit, skin: 810 mg/24H Severe;
Draize test, rabbit, skin: 500 mg/24H Mild;
Inhalation, mouse: LC50 = 5200 ppm/4H;
Inhalation, rat: LC50 = 34200 mg/m3/8H;
Oral, mouse: LD50 = 8100 mg/kg;
Oral, rat: LD50 = 2629 mg/kg;
Carcinogenicity:
CAS# 127-18-4:
ACGIH: A3 - Animal Carcinogen
California: carcinogen; initial date 4/1/88
NIOSH: potential occupational carcinogen
NTP: Suspect carcinogen
OSHA: Possible Select carcinogen
IARC: Group 2A carcinogen
Epidemiology: Epidemiologic studies have given inconsistent results. Studi es have shown that tetrachloroethylene has not caused canc er in exposed workers. The studies have serious weakne sses such as mixed exposures. In tests with rats and mice, i t appeared that tissue destruction or peroxisome prolifera tion rather than genetic mechanisms were the cause of the observed increases in normally occurring cancers. The oral mouse TDLo that was tumorigenic was 195 gm/kg/50W-I.
Teratogenicity: Has caused musculoskeletal abnormalities. Has caused morphological transformation at a dose of 97mol/L in a study using rat embryos.
Reproductive Effects: Has caused behavioral, biochemical, and metabolic effects on newborn rats when the mother was exposed to the TCLo of 900 ppm/7H at 7-13 days after conception. A dose of 300 ppm/7H 6-15 days after conception caused post-implantation mortality.
Neurotoxicity: No information available.
Mutagenicity: Not mutagenic in Escherichia coli. No mutagenic effects were seen in rat liver after exposure at 200 ppm for 10 weeks. No chromosome changes were seen in the bone marrow cells of exposed mice.
Other Studies: A case of 'obstructive jaundice' in a 6-week old infant has been attributed to tetrachloroethylene in breast milk.

Section 12 - Ecological Information

Ecotoxicity: Fish: Rainbow trout: LC50 = 5.28 mg/L; 96 Hr.; Static Condition, 12 degrees C Fathead Minnow: LC50 = 18.4 mg/L; 96 Hr.; Flow-through condition Bluegill/Sunfish: LC50 = 12.9 mg/L; 96 Hr.; Static Condition ria: Phytobacterium phosphoreum: EC50 = 120.0 mg/L; 30 minutes; Microtox test No data available.

Environmental: In soil, substance will rapidly evaporate. In water, it will evaporate. In air, it can be expected to exist in the vapor phase.
Physical: No information available.
Other: No information available.

Section 13 - Disposal Considerations

Chemical waste generators must determine whether a discarded chemical is classified as a hazardous waste. US EPA guidelines for the classification determination are listed in 40 CFR Parts 261.3. Additionally, waste generators must consult state and local hazardous waste regulations to ensure complete and accurate classification.
RCRA P-Series: None listed.
RCRA U-Series: CAS# 127-18-4: waste number U210.

Section 14 - Transport Information

	US DOT	IATA	RID/ADR	IMO	Canada TDG
Shipping Name:	TETRACHLOROETHYLENE				TETRACHLOROETHYLENE
Hazard Class:	6.1				6.1
UN Number:	UN1897				UN1897
Packing Group:	III				III

Section 15 - Regulatory Information

US FEDERAL

TSCA
CAS# 127-18-4 is listed on the TSCA inventory.
Health & Safety Reporting List
CAS# 127-18-4: Effective Date: 6/1/87; Sunset Date: 6/1/97
Chemical Test Rules
None of the chemicals in this product are under a Chemical Test Rule.
Section 12b
None of the chemicals are listed under TSCA Section 12b.
TSCA Significant New Use Rule
None of the chemicals in this material have a SNUR under TSCA.
SARA

CERCLA Hazardous Substances and corresponding RQs
CAS# 127-18-4: 100 lb final RQ; 45.4 kg final RQ
SARA Section 302 Extremely Hazardous Substances
None of the chemicals in this product have a TPQ.
SARA Codes
CAS # 127-18-4: acute.
Section 313
This material contains Tetrachloroethylene (CAS# 127-18-4, 99 0%),which is subject to the reporting requirements of Section 313 of SARA Title III and 40 CFR Part 373.

Clean Air Act:
CAS# 127-18-4 is listed as a hazardous air pollutant (HAP). This material does not contain any Class 1 Ozone depletors. This material does not contain any Class 2 Ozone depletors.
Clean Water Act:
None of the chemicals in this product are listed as Hazardous Substances under the CWA. CAS# 127-18-4 is listed as a Priority Pollutant under the Clean Water Act. CAS# 127-18-4 is listed as a Toxic Pollutant under the Clean Water Act.
OSHA:
None of the chemicals in this product are considered highly hazardous by OSHA.
STATE
CAS# 127-18-4 can be found on the following state right to know lists: California, New Jersey, Pennsylvania, Minnesota, Massachusetts.
The following statement(s) is(are) made in order to comply with the California Safe Drinking Water Act: WARNING: This product contains Tetrachloroethylene, a chemical known to the state of California to cause cancer. California No Significant Risk Level: CAS# 127-18-4: 14 ug/day NSRL

European/International Regulations

European Labeling in Accordance with EC Directives
Hazard Symbols:
XN N
Risk Phrases:
R 40 Limited evidence of a carcinogenic effect.
R 51/53 Toxic to aquatic organisms; may cause long-term adverse effects in the aquatic environment.

Safety Phrases:
S 23 Do not inhale gas/fumes/vapour/spray.
S 36/37 Wear suitable protective clothing and gloves.
S 61 Avoid release to the environment. Refer to special instructions/Safety data sheets.

WGK (Water Danger/Protection)
CAS# 127-18-4: 3
Canada - DSL/NDSL
CAS# 127-18-4 is listed on Canada's DSL List.
Canada - WHMIS
This product has a WHMIS classification of D1B, D2A.
Canadian Ingredient Disclosure List
CAS# 127-18-4 is listed on the Canadian Ingredient Disclosure List.
Exposure Limits
CAS# 127-18-4: OEL-ARAB Republic of Egypt:TWA 5 ppm (35 mg/m3);Skin OEL-AUSTRALIA:TWA 50 ppm (335 mg/m3);STEL 150 ppm;CAR OEL-BELGIUM:TWA 50 ppm (339 mg/m3);STEL 200 ppm (1368 mg/m3) OEL-CZECHOSLOVAKIA:TWA 250 mg/m3;STEL 1250 mg/m3 OEL-DENMARK:TWA 30 ppm (200 mg/m3);Skin OEL-FINLAND:TWA 50 ppm (335 mg/m3);STEL 75 ppm (520 mg/m3);Skin OEL-FRANCE:TWA 50 ppm (335 mg/m3) OEL-GERMANY:TWA 50 ppm (345 mg/m3);Carcinogen OEL-HUNGARY:STEL 50 mg/m3;Skin;Carcinogen OEL-JAPAN:TWA 50 ppm (340 mg/m3) OEL-THE NETHERLANDS:TWA 35 ppm (240 mg/m3);Skin OEL-THE PHILIPPINES:TWA 100 ppm (670 mg/m3) OEL-POLAND:TWA 60 mg/m3 OEL-RUSSIA:TWA 50 ppm;STEL 10 mg/m3 OEL-SWEDEN:TWA 10 ppm (70 mg/m3);STEL 25 ppm (170 mg/m3) OEL-SWITZERLAND:TWA 50 ppm (345 mg/m3);STEL 100 ppm;Skin OEL-THAILAND:TWA 100 ppm;STEL 200 ppm OEL-UNITED KINGDOM:TWA 50 ppm (335 mg/m3);STEL 15 ppm OEL IN BULGARIA, COLOMBIA, JORDAN, KOREA

check ACGIH TLV OEL IN NEW ZEALAND, SINGAPORE, VIETNAM check ACGI TLV

Section 16 - Additional Information

MSDS Creation Date: 6/17/1999
Revision #3 Date: 3/18/2003

The information above is believed to be accurate and represents the best information currently available to us. However, we make no warranty of merchantability or any other warranty, express or implied, with respect to such information, and we assume no liability resulting from its use. Users should make their own investigations to determine the suitability of the information for their particular purposes. In no event shall Fisher be liable for any claims, losses, or damages of any third party or for lost profits or any special, indirect, incidental, consequential or exemplary damages, howsoever arising, even if Fisher has been advised of the possibility of such damages.

http://www.epa.gov/IRIS/subst/0106.htm
Last updated on Thursday, January 25th, 2007.

Integrated Risk Information System

Tetrachloroethylene (CASRN 127-18-4)

view QuickView

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Reference Dose for Chronic Oral Exposure (RfD)

0106

Tetrachloroethylene; CASRN 127-18-4

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Tetrachloroethylene

File First On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	03/01/1988
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	

_I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

_I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Tetrachloroethylene
CASRN — 127-18-4
Last Revised — 03/01/1988

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without

an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. Oral RfD Summary

Critical Effect	Experimental Doses*	UF	MF	RfD
		1000	1	1E-2 mg/kg/day
Hepatotoxicity in mice, weight gain in rats	NOAEL: 20 mg/kg/day (converted to 14 mg/kg/day)			
6-Week Mouse Gavage Study Buben and O'Flaherty, 1985	LOAEL: 100 mg/kg/day (converted to 71 mg/kg/day)			

*Conversion Factors: Doses have been adjusted for treatment schedule (5 days/week)

I.A.2. Principal and Supporting Studies (Oral RfD)

Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: a dose- effect study. Toxicol. Appl. Pharmacol. 78: 105-122.

Buben and O'Flaherty (1985) exposed Swiss-Cox mice to tetrachloroethylene in corn oil by gavage at doses of 0, 20, 100, 200, 500, 1500, and 2000 mg/kg, 5 days/ week for 6 weeks. Liver toxicity was evaluated by several parameters including liver weight/body weight ratio, hepatic triglyceride concentration, DNA content, histopathological evaluation, and serum enzyme levels. Increased liver triglycerides were first observed in mice treated with 100 mg/kg. Liver weight/body weight ratios were significantly higher than controls for animals treated with 100 mg/kg. At higher doses, hepatotoxic effects included decreased DNA content, increased SGPT, decreased levels of G6P and hepatocellular necrosis, degeneration and polyploidy.

A NOEL of 14 mg/kg/day was established in a second study, as well (Hayes et al., 1986). Groups of 20 Sprague-Dawley rats of both sexes were administered doses of 14, 400, or 1400 mg/kg/day in drinking water. Males in the high-dose group and females in the two highest groups exhibited depressed body weights. Equivocal evidence of hepatotoxicity (increased liver and kidney weight/body weight ratios) were also observed at the higher doses.

I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF — The uncertainty factor of 1000 results from multiplying factors of 10 to account for intraspecies variability, interspecies variability and extrapolation of a subchronic effect level to its chronic equivalent.

MF — None

__I.A.4. Additional Studies/Comments (Oral RfD)

Other data support the findings of the principal studies. Exposure of mice and rats to tetrachloroethylene by gavage for 11 days caused hepatotoxicity (centrilobular swelling) at doses as low as 100 mg/kg/day in mice (Schumann et al., 1980). Mice were more sensitive to the effects of tetrachloroethylene exposure than rats. Increased liver weight was observed in mice at 250 mg/kg, while rats did not exhibit these effects until doses of 1000 mg/kg/day were reached. Relative sensitivity to man cannot be readily established but the RfD of 1E-2 mg/kg/day is protective of the most mild effects observed in humans [diminished odor perception/modified Romberg test scores in volunteers exposed to 100 ppm for 7 hours; roughly equivalent to 20 mg/kg/day (Stewart et al., 1961)].

The principal studies are of short duration. Inhalation studies have been performed which indicate that the uncertainty factor of 10 is sufficient for extrapolation of the subchronic effect to its chronic equivalent. Liver enlargement and vacuolation of hepatocytes were found to be reversible lesions for mice exposed to low concentrations of tetrachloroethylene (Kjellstrand et al., 1984). In addition, elevated liver weight/body weight ratios observed in animals exposed to tetrachloroethylene for 30 days were similar to those in animals exposed for 120 days. Several chronic inhalation studies have also been performed (Carpenter, 1937; NTP, 1985; Rowe et al., 1952). None are inconsistent with a NOAEL of 14 mg/kg/day for tetrachloroethylene observed by Buben and O'Flaherty (1985) and Hayes et al. (1986).

__I.A.5. Confidence in the Oral RfD

Study — Low
Database — Medium
RfD — Medium

No one study combines the features desired for deriving an RfD: oral exposure, large number of animals, multiple dose groups, testing in both sexes and chronic exposure. Confidence in the principal studies is low mainly because of the lack of complete histopathological examination at the NOAEL in the mouse study. The database is relatively complete but lacks studies of reproductive and teratology endpoints subsequent to oral exposure; thus, it receives a medium confidence rating. Medium confidence in the RfD follows.

__I.A.6. EPA Documentation and Review of the Oral RfD

U.S. EPA. 1985. Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA 600/8-82/005F.

U.S. EPA. 1987. Quantification of Toxicological Effects for Tetrachloroethylene. Prepared from the Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Office of Drinking Water, Washington, DC.

Agency Work Group Review — 05/20/1985, 08/05/1986, 09/17/1987

Verification Date — 09/17/1987

__I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov(internet address).

_I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Tetrachloroethylene
CASRN — 127-18-4

Not available at this time.

_II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Tetrachloroethylene
CASRN — 127-18-4

Not available at this time.

_VI. Bibliography

Substance Name — Tetrachloroethylene
CASRN — 127-18-4
Last Revised — 07/01/1989

_VI.A. Oral RfD References

Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose- effect study. Toxicol. Appl. Pharmacol. 78: 105-122.

Carpenter, C.P. 1937. The chronic toxicity of tetrachloroethylene. J. Ind. Hyg. Toxicol. 19(7): 323-336.

Hayes, J.R., L.W. Condie, Jr. and J.F. Borzelleca. 1986. The subchronic toxicity of tetrachloroethylene (perchloroethylene) administered in the drinking water of rats. Fund. Appl. Toxicol. 7: 119-125.

Kjellstrand, P., B. Holmquist, M. Kanje, et al. 1984. Perchloroethylene: Effects on body and organ weights and plasma butyrylcholinesterase activity in mice. Acta Pharmacol. Toxicol. 54 (5): 414-424.

NTP (National Toxicology Program). 1985. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Tetrachloroethylene (perchloroethylene). U.S. Dept. Health and Human Services, NIH Publ. No. 85- 2567.

Rowe, V.K., D.D. McCollister, H.C. Spencer, E.M. Adams and D.D. Irish. 1952. Vapor toxicity of tetrachloroethylene for laboratory animals and human subjects. Arch. Ind. Hyg. Occup. Med. 5: 566-579.

Schumann, A.M., J.F. Quast and P.G. Watanabe. 1980. The pharmacokinetics and macromolecular interaction of perchloroethylene in mice and rats as related to oncogenicity. Toxicol. Appl. Pharmacol. 55: 207-219.

Stewart, R.D., H.H. Gay, D.S. Erley, C.L. Hake and A.W. Schaffer. 1961. Human exposure to tetrachloroethylene vapor. Arch. Environ. Health. 2: 40-46.

U.S. EPA. 1985. Health Assessment Document for Tetrachloroethylene (perchloroethylene). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA 600/8-82-005F. Office of Drinking Water, Washington, DC.

U.S. EPA. 1987. Quantification of Toxicological Effects for Tetrachloroethylene. Prepared from the Health Assessment Document for Tetrachloroethylene (perchloroethylene). Office of Drinking Water, Washington, DC.

_VI.B. Inhalation RfC References

None

_VI.C. Carcinogenicity Assessment References

None

_VII. Revision History

Substance Name — Tetrachloroethylene
CASRN — 127-18-4

Date	Section	Description
12/23/1987	I.A.	RfD withdrawn pending further review
03/01/1988	I.A.	Revised Oral RfD sumary added - RfD changed
03/01/1988	III.A.	Health Advisory added
07/01/1989	VI.	Bibliography on-line
05/01/1990	II.	Carcinogen assessment now under review
06/01/1990	IV.A.1.	Area code for EPA contact corrected

06/01/1990	IV.F.1.	EPA contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.	Regulatory action section withdrawn
08/01/1995	II.	EPA's RfD/RfC and CRAVE workgroups were discontinued in May, 1995. Chemical substance reviews that were not completed by September 1995 were taken out of IRIS review. The IRIS Pilot Program replaced the workgroup functions beginning in September, 1995.
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
01/02/1998	I., II.	This chemical is being reassessed under the IRIS Program.

_VIII. Synonyms

Substance Name — Tetrachloroethylene
CASRN — 127-18-4
Last Revised — 01/31/1987

- 127-18-4
- Ankilostin
- Antisal 1
- Antisol 1
- Carbon bichloride
- Carbon dichloride
- Czterochloroetylen
- Dee-Solv
- Didakene
- Didokene
- Dowclene EC
- Dow-Per
- ENT 1,860
- Ethene, tetrachloro-
- Ethylene tetrachloride
- Ethylene, tetrachloro-
- Fedal-Un
- NCI-C04580
- Nema
- PCE
- PER
- Perawin
- PERC
- Perchloorethyleen, per
- Perchlor
- Perchloraethylen, per
- Perchlorethylene
- Perchlorethylene, per
- Perchloroethylene
- Perclene
- Percloroetilene

- Percosolv
- Percosolve
- PERK
- Perklone
- Persec
- Tetlen
- Tetracap
- Tetrachlooretheen
- Tetrachloraethen
- Tetrachlorethylene
- Tetrachloroethene
- Tetrachloroethylene
- 1,1,2,2-Tetrachloroethylene.
- Tetracloroetene
- Tetraguer
- Tetraleno
- Tetralex
- Tetravec
- Tetroguer
- Tetropil
- WLN: GYGUYGG

IRIS Home

Chronic Health Hazards for Non-Carcinogenic Effects

Reference Dose for Chronic Oral Exposure (RfD)

- Oral RfD Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Oral RfD
- EPA Documentation and Review

Reference Concentration for Chronic Inhalation Exposure (RfC)

- Inhalation RfC Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Inhalation RfC
- EPA Documentation

Material Safety Data Sheet

Trichloroethylene, stabilized

ACC# 23850

Section 1 - Chemical Product and Company Identification

MSDS Name: Trichloroethylene, stabilized
Catalog Numbers: AC158310000, AC158310010, AC158310025, AC421520000, AC421520040, AC421520200, AC421525000, S80327ACS-1, S80327ACS-2, NC9494591, T340-4, T341-20, T341-4, T341-500, T341J4, T403-4
Synonyms: Ethylene trichloride; Trichloroethene; 1,1,2-Trichloroethylene; TCE.
Company Identification:

Fisher Scientific
1 Reagent Lane
Fair Lawn, NJ 07410

For information, call: 201-796-7100
Emergency Number: 201-796-7100
For CHEMTREC assistance, call: 800-424-9300
For International CHEMTREC assistance, call: 703-527-3887

Section 2 - Composition, Information on Ingredients

CAS#	Chemical Name	Percent	EINECS/ELINCS
79-01-6	Trichloroethylene	>99	201-167-4

Section 3 - Hazards Identification

EMERGENCY OVERVIEW

Appearance: clear, colorless liquid.
Warning! Breathing vapors may cause drowsiness and dizziness. Causes eye and skin irritation. Aspiration hazard if swallowed. Can enter lungs and cause damage. May cause cancer based on animal studies. May cause liver damage.
Target Organs: Central nervous system, liver, eyes, skin.

Potential Health Effects
Eye: Causes moderate eye irritation. May result in corneal injury. Contact produces irritation, tearing, and burning pain. Contact with trichloroethylene causes pain but no permanent injury to the eyes. (Doc of TLV)
Skin: Causes mild skin irritation. Prolonged and/or repeated contact may cause defatting of the skin and dermatitis. May cause peripheral nervous system function impairment including persistent neuritis, and temporary loss of touch. Damage to the liver and other organs has been observed in workers who have been overexposed.
Ingestion: May cause irritation of the digestive tract. Aspiration of material into the lungs may cause chemical pneumonitis, which may be fatal.
Inhalation: May cause respiratory tract irritation. May cause liver abnormalities. May cause cardiac abnormalities. May cause peripheral nervous system effects. Inhalation overexposure may lead to central nervous system depression, producing effects such as dizziness, headache,

confusion, incoordination, nausea, weakness, and loss of consciousness. Extreme exposures may cause other CNS effects including death. The chief symptoms of TCE exposure were found to be abnormal fatigue, irritability, headache, gastric disturbances, and intolerance to alcohol. (Doc to TLV)
Chronic: Possible cancer hazard based on tests with laboratory animals. Chronic inhalation may cause effects similar to those of acute inhalation. Prolonged or repeated skin contact may cause defatting and dermatitis. May cause peripheral nervous system function impairment including persistent neuritis, and temporary loss of touch. Damage to the liver and other organs has been observed in workers who have been overexposed.

Section 4 - First Aid Measures

Eyes: Immediately flush eyes with plenty of water for at least 15 minutes, occasionally lifting the upper and lower eyelids. Get medical aid imme diately.
Skin: Get medical aid if irritation develops or persists. Flush skin with plenty of soap and water.
Ingestion: If victim is conscious and alert, give 2-4 cupfuls of milk or water. Never give anything by mouth to an unconscious person. Possible aspiration hazard. Get medical aid immediately.
Inhalation: Get medical aid immediately. Remove from exposure and move to fresh air immediately. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Do NOT use mouth-to-mouth resuscitation.
Notes to Physician: Treat symptomatically and supportively.

Section 5 - Fire Fighting Measures

General Information: As in any fire, wear a self-contained breathing apparatus in pressure-demand, MSHA/NIOSH (approved or equivalent), and full protective gear. During a fire, irritating and highly toxic gases may be generated by thermal decomposition or combustion. Use water spray to keep fire-exposed containers cool.
Extinguishing Media: Use extinguishing media most appropriate for the surrounding fire.
Flash Point: None
Autoignition Temperature: 420 deg C (788.00 deg F)
Explosion Limits, Lower:8
Upper: 10.5
NFPA Rating: (estimated) Health: 2; Flammability: 1; Instability: 0

Section 6 - Accidental Release Measures

General Information: Use proper personal protective equipment as indicated in Section 8.
Spills/Leaks: Absorb spill with inert material (e.g. vermiculite, sand or earth), then place in suitable container. Provide ventilation. Approach spill from upwind. Control runoff and isolate discharged material for proper disposal.

Section 7 - Handling and Storage

Handling: Wash thoroughly after handling. Remove contaminated clothing and wash before reuse.

Use with adequate ventilation. Avoid contact with eyes, skin, and clothing. Avoid breathing vapor.
Storage: Store in a tightly closed container. Store in a cool, dry, well-ventilated area away from incompatible substances.

Section 8 - Exposure Controls, Personal Protection

Engineering Controls: Facilities storing or utilizing this material should be equipped with an eyewash facility and a safety shower. Use adequate general or local exhaust ventilation to keep airborne concentrations below the permissible exposure limits.

Exposure Limits

Chemical Name	ACGIH	NIOSH	OSHA - Final PELs
Trichloroethylene	50 ppm TWA; 100 ppm STEL	1000 ppm IDLH	100 ppm TWA; 200 ppm Ceiling

OSHA Vacated PELs: Trichloroethylene: 50 ppm TWA; 270 mg/m3 TWA
Personal Protective Equipment
Eyes: Wear chemical splash goggles.
Skin: Wear appropriate protective gloves to prevent skin exposure.
Clothing: Wear appropriate protective clothing to prevent skin exposure.
Respirators: Follow the OSHA respirator regulations found in 29 CFR 1910.134 or European Standard EN 149. Use a NIOSH/MSHA or European Standard EN 149 approved respirator if exposure limits are exceeded or if irritation or other symptoms are experienced.

Section 9 - Physical and Chemical Properties

Physical State: Liquid
Appearance: clear, colorless
Odor: chloroform-like
pH: Not available.
Vapor Pressure: 58 mm Hg @ 20 deg C
Vapor Density: 4.5 (air=1)
Evaporation Rate:0.69 (CCl4=1)
Viscosity: 0.0055 poise
Boiling Point: 87 deg C
Freezing/Melting Point:-86 deg C
Decomposition Temperature: Not available.
Solubility: Slightly soluble.
Specific Gravity/Density:1.46
Molecular Formula:C2HCl3
Molecular Weight:131.39

Section 10 - Stability and Reactivity

Chemical Stability: Stable under normal temperatures and pressures.
Conditions to Avoid: Light, confined spaces.
Incompatibilities with Other Materials: Active metals.
Hazardous Decomposition Products: Hydrogen chloride, phosgene, carbon monoxide, carbon

dioxide.
Hazardous Polymerization: May occur.

Section 11 - Toxicological Information

RTECS#:
CAS# 79-01-6: KX4550000
LD50/LC50:
CAS# 79-01-6:
Draize test, rabbit, eye: 20 mg/24H Moderate;
Draize test, rabbit, skin: 2 mg/24H Severe;
Inhalation, mouse: LC50 = 8450 ppm/4H;
Inhalation, mouse: LC50 = 220000 mg/m3/20M;
Inhalation, mouse: LC50 = 262000 mg/m3/30M;
Inhalation, mouse: LC50 = 40000 mg/m3/4H;
Inhalation, rat: LC50 = 140700 mg/m3/1H;
Oral, mouse: LD50 = 2402 mg/kg;
Oral, mouse: LD50 = 2400 mg/kg;
Oral, rat: LD50 = 4920 mg/kg;
Skin, rabbit: LD50 = >20 gm/kg;
Skin, rabbit: LD50 = 20 mL/kg;

Carcinogenicity:
CAS# 79-01-6:

- **ACGIH:** Not listed.
- **California:** carcinogen, initial date 4/1/88
- **NTP:** Suspect carcinogen
- **IARC:** Group 2A carcinogen

Epidemiology: In six epidemiological studies completed, there was no evidence to suggest that trichloroethylene has increased the incidence of cancer in humans. (Documentation of the TLV, 7th edition)
Teratogenicity: No information available.
Reproductive Effects: Experimental reproductive effects have been observed.
Mutagenicity: Human mutation data has been reported. IARC and the National Toxicology Program (NTP) stated that variability in the mutagencity test results with trichloroethylene may be due to the presence of various stabilizers used in TCEwhich are mutagens (e.g.epoxybutane, epichlorohydrin).See actual entry in RTECS for complete infomation.R68 Mutagen Category 3 (CHIP 2002, UK).
Neurotoxicity: No information available.
Other Studies:

Section 12 - Ecological Information

Ecotoxicity: Fish: Fathead Minnow: 41-67 mg/L; 96 hrs.; LC50Daphnia: Daphnia: 2.2-100 mg/L; 48 hrs.; LC50Mollusk Shrimp: 2 mg/L; 96 hrs.; LC50 Bluegill sunfish, LD50= 44,700 ug/L/96Hr. Fathead minnow, LC50=40.7 mg/L/96Hr.

Environmental: In air, substance is photooxidized and is reported to form phosgene, dichloroacetyl chloride, and formyl chloride. In water, it evaporates rapidly. Potential for mobility in soil is high.
Physical: No information available.
Other: Bioconcentration potential is low (BCF less than 100).

Section 13 - Disposal Considerations

Chemical waste generators must determine whether a discarded chemical is classified as a hazardous waste. US EPA guidelines for the classification determination are listed in 40 CFR Parts 261.3. Additionally, waste generators must consult state and local hazardous waste regulations to ensure complete and accurate classification.
RCRA P-Series: None listed.
RCRA U-Series:
CAS# 79-01-6: waste number U228.

Section 14 - Transport Information

	US DOT	Canada TDG
Shipping Name:	TRICHLOROETHYLENE	TRICHLOROETHYLENE
Hazard Class:	6.1	6.1
UN Number:	UN1710	UN1710
Packing Group:	III	III

Section 15 - Regulatory Information

US FEDERAL

TSCA
CAS# 79-01-6 is listed on the TSCA inventory.
Health & Safety Reporting List
None of the chemicals are on the Health & Safety Reporting List.
Chemical Test Rules
None of the chemicals in this product are under a Chemical Test Rule.
Section 12b
None of the chemicals are listed under TSCA Section 12b.
TSCA Significant New Use Rule
None of the chemicals in this material have a SNUR under TSCA.
CERCLA Hazardous Substances and corresponding RQs
CAS# 79-01-6: 100 lb final RQ; 45.4 kg final RQ
SARA Section 302 Extremely Hazardous Substances
None of the chemicals in this product have a TPQ.
SARA Codes
CAS # 79-01-6: immediate, delayed, reactive.
Section 313
This material contains Trichloroethylene (CAS# 79-01-6, >99%),which is subject to the reporting requirements of Section 313 of SARA Title III and 40 CFR
Clean Air Act:
CAS# 79-01-6 is listed as a hazardous air pollutant (HAP).
This material does not contain any Class 1 Ozone depletors.

This material does not contain any Class 2 Ozone depletors.

Clean Water Act:

CAS# 79-01-6 is listed as a Hazardous Substance under the CWA. CAS# 79-01-6 is listed as a Priority Pollutant under the Clean Water Act. CAS# 79-01-6 is listed as a Toxic Pollutant under the Clean Water Act.

OSHA:

None of the chemicals in this product are considered highly hazardous by OSHA.

STATE

CAS# 79-01-6 can be found on the following state right to know lists: California, New Jersey, Pennsylvania, Minnesota, Massachusetts.

California Prop 65

The following statement(s) is(are) made in order to comply with the California Safe Drinking Water Act:

WARNING: This product contains Trichloroethylene, a chemical known to the state of California to cause cancer.

California No Significant Risk Level: CAS# 79-01-6: 50 æg/day NSRL (oral); 80 æg/day NSRL (inhalation)

European/International Regulations

European Labeling in Accordance with EC Directives

Hazard Symbols:

T

Risk Phrases:

R 36/38 Irritating to eyes and skin.
R 45 May cause cancer.
R 52/53 Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
R 67 Vapours may cause drowsiness and dizziness.

Safety Phrases:

S 45 In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible).
S 53 Avoid exposure - obtain special instructions before use.
S 61 Avoid release to the environment. Refer to special instructions /safety data sheets.

WGK (Water Danger/Protection)

CAS# 79-01-6: 3

Canada - DSL/NDSL

CAS# 79-01-6 is listed on Canada's DSL List.

Canada - WHMIS

This product has a WHMIS classification of D1B, D2B.

This product has been classified in accordance with the hazard criteria of the Controlled Products Regulations and the MSDS contains all of the information required by those regulations.

Canadian Ingredient Disclosure List

CAS# 79-01-6 is listed on the Canadian Ingredient Disclosure List.

Section 16 - Additional Information

MSDS Creation Date: 2/01/1999
Revision #7 Date: 12/27/2006

The information above is believed to be accurate and represents the best information currently available to us. However, we make

no warranty of merchantability or any other warranty, express or implied, with respect to such information, and we assume no liability resulting from its use. Users should make their own investigations to determine the suitability of the information for their particular purposes. In no event shall Fisher be liable for any claims, losses, or damages of any third party or for lost profits or any special, indirect, incidental, consequential or exemplary damages, howsoever arising, even if Fisher has been advised of the possibility of such damages.

http://www.epa.gov/IRIS/subst/0199.htm
Last updated on Thursday, January 25th, 2007.

Integrated Risk Information System

Trichloroethylene (CASRN 79-01-6)

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Reference Dose for Chronic Oral Exposure (RfD)

0199

Trichloroethylene; CASRN 79-01-6

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Trichloroethylene

File First On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	08/01/1992
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	withdrawn	07/01/1989

_I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

_I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name —Trichloroethylene
CASRN — 79-01-6

Not available at this time.

_I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name —Trichloroethylene
CASRN — 79-01-6

Not available at this time.

_II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name —Trichloroethylene
CASRN — 79-01-6

The carcinogen assessment summary for this substance has been withdrawn following further review.

Agency Work Group Review — 12/04/1986, 04/06/1989, 05/30/1989, 09/22/1993, 06/09/1994

EPA Contacts:

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

_III. [reserved]
_IV. [reserved]
_V. [reserved]

_VI. Bibliography

Substance Name —Trichloroethylene
CASRN — 79-01-6

Not available at this time.

_VII. Revision History

Substance Name —Trichloroethylene
CASRN — 79-01-6

Date	Section	Description

03/01/1988	II.B.3.	Text revised
03/01/1988	II.B.4.	Confidence statement revised
03/01/1988	II.C.2.	Text added
03/01/1988	II.C.4.	Confidence statement revised
03/01/1988	II.D.4.	Documentation corrected
05/01/1989	II.	Carcinogen assessment summary noted as pending change
06/01/1989	II.D.3.	Primary contact changed
07/01/1989	II.	Withdrawn; new assessment verified (in preparation)
12/01/1989	I.B.	Inhalation RfD now under review
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
07/01/1992	II.	EPA contact changed; work group review dates added
08/01/1992	I.A.	Oral RfD now under review
11/01/1993	II.	Work group review date added
07/01/1994	II.	Work group review date added
08/01/1995	I.A., I.B., II.	EPA's RfD/RfC and CRAVE workgroups were discontinued in May, 1995. Chemical substance reviews that were not completed by September 1995 were taken out of IRIS review. The IRIS Pilot Program replaced the workgroup functions beginning in September, 1995.
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
01/02/1998	I., II.	This chemical is being reassessed under the IRIS Program.
06/05/2003	Status of Data	Correction of administrative error concerning the date the carcinogenicity assessment (II.) was withdrawn from IRIS.
06/07/2004	VIII	Text revised.

_VIII. Synonyms

Substance Name —Trichloroethylene
CASRN — 79-01-6
Last Revised — 03/31/1987

79-01-6
ACETYLENE TRICHLORIDE

ALGYLEN
ANAMENTH
BENZINOL
BLACOSOLV
BLANCOSOLV
CECOLENE
CHLORILEN
1-CHLORO-2,2-DICHLOROETHYLENE
CHLORYLEA
CHLORYLEN
CHORYLEN
CIRCOSOLV
CRAWHASPOL
DENSINFLUAT
1,1-DICHLORO-2-CHLOROETHYLENE
DOW-TRI
DUKERON
ETHINYL TRICHLORIDE
ETHYLENE TRICHLORIDE
ETHYLENE, TRICHLORO-
FLECK-FLIP
FLOCK FLIP
FLUATE
GEMALGENE
GERMALGENE
LANADIN
LETHURIN
NARCOGEN
NARKOGEN
NARKOSOID
NCI-C04546
NIALK
PERM-A-CHLOR
PERM-A-CLOR
PETZINOL
PHILEX
RCRA WASTE NUMBER U228
TCE
THRETHYLEN
THRETHYLENE
TRETHYLENE
TRI
TRIAD
TRIAL
TRIASOL
TRICHLOORETHEEN
TRICHLOORETHYLEEN, TRI
TRICHLORAETHEN
TRICHLORAETHYLEN, TRI
TRICHLORAN
TRICHLOREN
TRICHLORETHENE
TRICHLORETHYLENE

TRICHLORETHYLENE, TRI
TRICHLOROETHENE
Trichloroethylene
1,1,2-TRICHLOROETHYLENE
1,2,2-TRICHLOROETHYLENE
TRI-CLENE
TRICLORETENE
TRICLOROETILENE
TRIELENE
TRIELIN
TRIELINA
TRIKLONE ™, "Triklone" is a trade mark of INEOS Chlor Limited.
TRILEN
TRILENE
TRILINE
TRIMAR
TRIOL
TRI-PLUS
TRI-PLUS M
UN 1710
VESTROL
VITRAN
WESTROSOL

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Chronic Health Hazards for Non-Carcinogenic Effects

Reference Dose for Chronic Oral Exposure (RfD)

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* Principal and Supporting Studies
* Uncertainty and Modifying Factors
* Additional Studies/Comments
* Confidence in the Oral RfD
* EPA Documentation and Review

Reference Concentration for Chronic Inhalation Exposure (RfC)

* Inhalation RfC Summary
* Principal and Supporting Studies
* Uncertainty and Modifying Factors
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http://www.epa.gov/ncea/iris/subst/0025.htm
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Reference Dose for Chronic Oral Exposure (RfD)

You will need Adobe Reader to view some of the files on this page. See EPA's PDF page to learn more.

Note: A TOXICOLOGICAL REVIEW is available for this chemical in Adobe PDF Format (112 Pages, 760 Kbytes). Similar documents can be found in the List of Available IRIS Toxicological Reviews.

Quantitative Dose-Response Modeling, which accompanies the toxicological review, is available in the attached electronic Zip file (193 K). Click on this link, and then click on "Save File."

Links to specific pages in the toxicological review are available throughout this summary. To utilize this feature, your Web browser and Adobe program must be configured properly so the PDF displays within the browser window. If your browser and Adobe program need configuration, please go to EPA's PDF page for instructions.

0025

Chloroform; CASRN 67-66-3; 10/19/01

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Chloroform

File First On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	10/19/01
Inhalation RfC Assessment (I.B.)	not available	
Carcinogenicity Assessment (II.)	on-line	10/19/01

_I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

_I.A. Reference Dose for Chronic Oral Exposure (RfD)

Chloroform
CASRN — 67-66-3
Last Revised — 10/19/01

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg/day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

__I.A.1. Oral RfD Summary

Traditional Approach

For comparison purposes, an RfD was developed using the traditional NOAEL/LOAEL approach. The results of this method are provided below. This is the same approach and RfD result reported on IRIS (01/13/87).

Critical Effect	Experimental Doses*	UF	MF	RfD
		1,000	1	0.01 (mg/kg/day)
Moderate/marked fatty cyst formation in the liver and elevated SGPT Dog, chronic oral bioassay Heywood et al., 1979	NOAEL: none LOAEL: 15 mg/kg/day (converted to 12.9 mg/kg/day)			

*Conversion Factors and Assumptions — 15 mg/kg/day × 6 days/7 days = 12.9 mg/kg/day.

__I.A.2. Principal and Supporting Studies (Oral RfD)

Heywood, R; Sortwell, RJ; Noel, PRB; et al. (1979) Safety evaluation of toothpaste containing chloroform: III. Long-term study in beagle dogs. J Environ Pathol Toxicol 2:835-851.

Heywood et al. (1979) exposed groups of eight male and eight female beagle dogs to doses of 15 or 30 mg chloroform/kg/day. The chemical was given orally in a toothpaste base in gelatin capsules, 6 days/week for 7.5 years. This was followed by a 20- to 24-week recovery period. Eight dogs of each sex served as an untreated group and a final group of 16 dogs (8/sex) received an alternative nonchloroform toothpaste (vehicle control). Four male dogs (one each from the low- and high-dose chloroform groups, the vehicle control group, and the untreated

control group) and seven female dogs (four from the vehicle control group and three from the untreated control group) died during the study. In the low-dose group, levels of serum glutamate-pyruvate transaminase (SGPT, also known as alanine aminotransferase) were increased by an average of about 40% compared with control, with the effects being statistically significant from week 130 through week 364. In the high-dose group, SGPT levels tended to average about twice those in the control group, and the differences were statistically significant from week 6 throughout treatment. After 14 weeks of recovery, SGPT levels remained significantly increased in the high-dose group, but not in the low-dose group, when compared with the controls. After 19 weeks of recovery, SGPT levels were not significantly increased in either treated group when compared with the controls. The authors concluded that the increases in SGPT levels were likely the result of minimal liver damage. Serum alkaline phosphatase (SAP) and SGPT levels were also moderately increased (not statistically significant) in the treated dogs at the end of the treatment period when compared with the controls. Microscopic examinations were conducted on the major organs. The most prominent microscopic effect observed in the liver was the presence of "fatty cysts," which were described as aggregations of vacuolated histiocytes. The fatty cysts were observed in the control and treated dogs, but were larger and more numerous (i.e., higher incidence of cysts rated as "moderate or marked," as opposed to "occasional or minimal") in the treated dogs than in the control dogs at both doses. The prevalence of moderate or marked fatty cysts was 1/27 in control animals, 9/15 in low dose animals, and 13/15 in high dose animals. Nodules of altered hepatocytes were observed in both treated and control animals, and therefore were not considered related to treatment. No other treatment-related nonneoplastic or neoplastic lesions were reported for the liver, gall bladder, cardiovascular system, reproductive system, or urinary system. A NOAEL was not identified in this study. However, a LOAEL of 15 mg/kg/day was identified, based on elevated SGPT levels and increased incidence and severity of fatty cysts (U.S. EPA, 1998a).

Benchmark Dose (BMD) Approach

Selection of Data Sets for Modeling

The following data sets were selected for BMD modeling:

- Incidence of fatty cysts in liver and SGPT levels of dogs (Heywood et al., 1979)
- Histological evidence of renal cytotoxicity in male rats exposed via drinking water (Hard et al., 2000)
- Increased labeling index in kidney of female mice exposed via drinking water (Larson et al., 1994b)
- Increased labeling index in liver of female rats exposed via gavage in corn oil (Larson et al., 1995b)

These studies were chosen because they all provide quantitative dose-response data for sensitive indicators of chloroform toxicity.

BMD Modeling of Selected Data Sets

The detailed results of the BMD model fitting are presented in Appendix B of the Toxicological Review of Chloroform. Within a data set, the preferred model was selected based on the quality of the model fit to the data.

As seen, the kidney LI data set from Larson et al. (1994b) could not be adequately described by any of the continuous models. This is because even though the response was statistically

significant, the magnitude of the response was small in comparison to normal variability, and the data did not form a smooth dose-response relationship (tending to first increase and then decrease as dose increased). The liver and kidney LI data sets from Larson et al. (1995b) were reasonably well fit by the Hill equation, with BMD values of 64-75 mg/kg/day. However, the software was not able to estimate a benchmark dose limit (BMDL) value in either case. The data sets from the studies by Hard et al. (2000) and by Heywood et al. (1979) were adequately fit by one or more of the dichotomous models, with the best fit being given by the log-logistic and the quantal-linear models, respectively. The preferred BMD of 70 mg/kg/day based on the renal cytotoxicity data of Hard et al. (2000) is similar to the BMD values derived for the LI data from Larson et al (1995b), but is significantly higher than the preferred BMD based on the incidence of fatty cysts in dogs (1.7 mg/kg/day) reported by Heywood et al. (1979). The basis for this marked difference in BMD between studies is not known, but the data suggest that liver toxicity in the dog is a more sensitive endpoint of chloroform toxicity than renal or liver cytotoxicity in rodents.

Calculation of the BMD-Based RfD

Critical Effect	Experimental Doses*	UF	MF	RfD
		100	1	0.01 (mg/kg/day)
Moderate/marked fatty cyst formation in the liver and elevated SGPT Dog, chronic oral bioassay Heywood et al., 1979	$BMDL_{10}$: 1.2 mg/kg/day (converted to 1.0 mg/kg/day)			

The $BMDL_{10}$ provided in the table represents the 95% confidence lower bound on the dose associated with a 10% extra risk based on the prevalence of animals with moderate to marked fatty cysts in liver and elevated SGPT. The value of the $BMDL_{10}$ was calculated from the data of Heywood et al. (1979) using EPA's BMDS software Version 1.2. The value derived from the BMD modeling (1.2 mg/kg/day) was adjusted by a factor of 6/7 to account for exposure 6 days per week.

__I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF = 100

In the benchmark dose approach, an uncertainty factor (UF) of 10 was used to account for interspecies extrapolation, and a UF of 10 was used to protect sensitive subpopulations. In the NOAEL/LOAEL approach, an additional factor of 10 was used to account for extrapolation from a LOAEL to a NOAEL (total UF = 1,000). No additional factors were required to account for extrapolation from short term to long term (the study duration was 7.5 years) or to account for limitations in the database.

MF = 1

No additional modifying factors (MFs) were considered necessary because there are no substantial concerns or limitations in the derivation of the RfD that are not accounted for in the UFs described above.

I.A.4. Additional Studies/Comments (Oral RfD)

In general, the NOAEL/LOAEL approach for derivation of an RfD is subject to a number of limitations, most of which are addressed by use of the BMD approach (U.S. EPA, 1995). Thus, the RfD based on the BMD approach is generally preferred, unless there are insufficient dose-response data to support derivation of a reliable BMD.

In this case, the dose-response data set from the critical study (Heywood et al., 1979) is composed of only two doses plus a control group. This is considered to be a limitation, as the shape of the dose-response curve is difficult to define with only three values, especially when the lowest dose yields a response that is well above the benchmark response. Nevertheless, the data do yield curve fits of adequate quality, so the results of the BMD approach are considered preferable to the NOAEL/LOAEL approach.

Note that, in this particular case, the two approaches (NOAEL/LOAEL and benchmark) yield equal RfD values. This is consistent, albeit coincidental, with the results from the default LOAEL/NOAEL method.

Many other studies in animals support the conclusion that the liver and/or the kidney are the key target organs for chloroform-induced toxicity. Most of these studies have been performed in rats and mice, and most yield LOAEL values that are substantially higher than those observed in dogs.

In a study conducted by Palmer et al. (1979), in which rats were administered daily oral doses of 60 mg chloroform/kg/day in a toothpaste vehicle, treatment-related effects included a decrease in plasma but not erythrocyte, cholinesterase in females, a decrease in liver weight in females, and a marginal but consistent and progressive retardation in weight gain in both sexes. The authors stated that although minor histological changes in the liver were noted, there was no evidence of severe fatty infiltration, fibrosis, or bile duct abnormalities in the livers of treated animals. The authors concluded that there was no evidence of treatment-related toxic effects in the liver. However, the "minor histopathological" changes in the liver were not described and the presence of any fatty infiltration that would be designated as less than severe was not reported. Therefore, these results could not be compared to those reported in the dog study. The LOAEL for this study was 60 mg/kg/day.

A slight (2%-3% vs. 7%-8%) increase in moderate to severe fatty degeneration of the liver was seen in ICI mice given 60 mg but not 17 mg chloroform/kg/day in a toothpaste vehicle for 80 weeks (Roe et al., 1979). However, no effects were evident when the incidences of fatty and nonfatty liver degeneration were combined in the ICI or three other mice strains. No other noncancer effects attributable to chloroform were noted. A NOAEL of 17 mg/kg/day and a LOAEL of 60 mg/kg/day were identified from this study.

No treatment-related noncancer effects were noted in rats administered chloroform in drinking water for 23 months at time-weighted average doses up to 160 mg/kg/day (Jorgenson et al., 1982, 1985). However, subsequent review of the histopathology slides from this study revealed evidence that chloroform produced a moderate to low level of renal proximal tubule injury associated with cell turnover indicative of cytotoxicity (Hard et al., 2000). These changes were noted in the high-dose (160 mg/kg) group males as early as 12 months but were increased in grade by 18 months. Similar changes were found in the mid-dose males (81 mg/kg), although at a lower grade, in the 18-month and 2-year dose groups. These changes were not seen in controls or the low-dose group. Therefore, the identified NOAEL for noncancer effects for this study is 38 mg chloroform/kg/day, with the LOAEL at 81 mg/kg/day.

In mice exposed to chloroform in drinking water, mortality within the first 3 weeks was significantly increased in the two highest dose groups, 130 and 263 mg/kg/day, but was comparable with controls after that time (Jorgenson et al., 1982). Early mortality and behavioral effects (e.g., lassitude, lack of vigor) were apparently related to reduced water consumption among some treated mice in the two highest dose groups. A significant increase in liver fat in mice was noted at doses of 65 mg/kg/day and higher at 3 months, but only at doses of 130 and 263 mg/kg/day by 6 months. Liver fat content was not reported for any later time points or at terminal sacrifice; therefore, the relevance of this observation as an adverse effect rather than an adaptive response could not be assessed. No increased incidence of liver tumors was reported, and the presence or absence of nonneoplastic histopathological alterations was not described. These data indicate that doses of 130 to 263 mg/kg/day may produce adverse effects in mice; however, these effects may be secondary to decreased water consumption.

Reproductive/developmental toxicity studies were also considered in the selection of the critical study/effect for the reference dose in the event the fetus represented a more sensitive population. These included studies in rats (Thompson et al., 1974), in rabbits (Thompson et al., 1974), and in mice (NTP, 1988). In the developmental studies in rabbits and rats, no treatment-related effects were noted when chloroform was administered by gavage in corn oil during gestation at doses of 50 mg/kg/day or less (Thompson et al., 1974). In the rabbit study, a clear dose-response was absent and the effects noted in offspring of dams administered chloroform at doses up to 50 mg/kg/day (the highest dose tested) on days 6 to 18 of gestation were not considered to be treatment-related (Thompson et al., 1974). In rats, the only effect noted was a significant reduction in fetal weight found only in offspring of dams given chloroform at the highest dose tested, 126 mg/kg/day, on days 6 to 15 of gestation (Thompson et al., 1974). No fetal effects attributed to chloroform treatment were noted in this rat study for the lower dose groups (up to 50 mg/kg/day during gestation). A NOAEL of 50 mg/kg/day was identified for both studies.

In a two-generation reproductive study in mice, no significant effects were seen in any reproductive parameter assessed in either the parental or the F_1 generations at doses up to 41 mg/kg/day administered by gavage in corn oil (NTP, 1988). Systemic toxicity was not evaluated in the parental generation. However, increased liver weights and liver lesions, described as mild to moderate degeneration of centrilobular hepatocytes accompanied by single-cell necrosis, were noted in F_1 females, but not males, exposed both in utero and postnatally at a dose of 41 mg/kg/day. Postnatal exposure in the F_1 generation began at postnatal day 22 and continued until the birth of the F_2 generation (mice were mated at 64 to 84 days of age). The F_1 offspring in the two lower dose groups, 6.6 and 16 mg/kg/day, were not evaluated histopathologically; therefore, no NOAEL or LOAEL could be definitively established for this study. A dose of 41 mg/kg/day may represent the LOAEL; however, the amount of in utero exposure was not estimated, nor was the contribution of in utero exposure to liver toxicity assessed. Because quantitative data were available only for the control and high-dose groups, the study was not selected for benchmark modeling.

In the reproductive/developmental studies, both maternal toxicity and effects on the fetus or offspring occurred at doses higher than those that produced evidence of liver toxicity in the dog study. Therefore, these were not used as the critical study for derivation of the RfD. ***For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).***

I.A.5. Confidence in the Oral RfD

Study — Medium

Database — Medium
RfD — Medium

The overall confidence in this RfD assessment is medium. The database on noncancer effects in animals is extensive, and data are adequate to derive reliable dose-response curves for key endpoints. Confidence is not rated higher because data in humans are limited, and extrapolation from animals to humans (with an attendant uncertainty factor of 10) is required.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

__I.A.6. EPA Documentation and Review of the Oral RfD

Source Document — U.S. EPA, 2001

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS Summary. A record of these comments is included as an appendix to U.S. EPA (2001). ***To review this appendix, exit to the toxicological review, Appendix A, External Peer Review -- Summary of Comments and Disposition (PDF).***

Other EPA Documentation — U.S. EPA, 1994, 1997, 1998a-c, 2001

Agency Consensus Date — 7/27/2001

__I.A.7. EPA Contacts (Oral RfD)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

_I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

(Not available. To be developed)

_II. Carcinogenicity Assessment for Lifetime Exposure

Chloroform
CASRN — 67-66-3
Last Revised — 10/19/01

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per µg/L drinking water or risk per µg/cu.m air breathed. The third form in which risk is presented is a concentration of the chemical in drinking water or air associated with cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000. The rationale and methods used to develop the

carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

_II.A. Evidence for Human Carcinogenicity

__II.A.1. Weight-of-Evidence Characterization

Under the 1986 U.S. EPA Guidelines for Carcinogen Risk Assessment, chloroform has been classified as Group B2, *probable human carcinogen*, based on "sufficient evidence" of carcinogenicity in animals (U.S. EPA, 1998a).

Under the Proposed Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1996; U.S. EPA, 1999), chloroform is *likely to be carcinogenic to humans by all routes of exposure* under high-exposure conditions that lead to cytotoxicity and regenerative hyperplasia in susceptible tissues (U.S. EPA, 1998a,b). Chloroform is *not likely to be carcinogenic to humans by any route of exposure* under exposure conditions that do not cause cytotoxicity and cell regeneration. This weight-of-evidence conclusion is based on: 1) observations in animals exposed by both oral and inhalation pathways which indicate that sustained or repeated cytotoxicity with secondary regenerative hyperplasia precedes, and is probably required for, hepatic and renal neoplasia; 2) there are no epidemiological data specific to chloroform and, at most, equivocal epidemiological data related to drinking water exposures that cannot necessarily be attributed to chloroform amongst multiple other disinfection byproducts; and 3) genotoxicity data on chloroform are essentially negative, although there are some scattered positive results that generally have limitations such as excessively high dose or with confounding factors. Thus, the weight-of-evidence of the genotoxicity data on chloroform supports a conclusion that chloroform is not strongly mutagenic, and that genotoxicity is not likely to be the predominant mode of action underlying the carcinogenic potential of chloroform. Although no cancer data exist for exposures via the dermal pathway, the weight-of-evidence conclusion is considered to be applicable to this pathway as well, because chloroform absorbed through the skin and into the blood is expected to be metabolized and to cause toxicity in much the same way as chloroform absorbed by other exposure routes.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

__II.A.2. Human Carcinogenicity Data

Inadequate. There are no epidemiological data attributing cancer to exposure to chloroform *per se*. Although there are some equivocal epidemiological data relating a weak association of drinking water exposures to bladder, rectal and colon cancer (Morris et al. 1992 ; McGeehin et al., 1993; Vena et al. 1993; Morris, 1995; King and Marrett, 1996; Doyle et al., 1997; Freedman et al., 1997; Cantor et al, 1998; Hildesheim et al., 1998), these studies can not attribute to chloroform among multiple other disinfection byproducts (DBPs) (SAB, 2000, ATSDR, 1997; IPCS, 2000). Morris et al. (1992) did a meta-analysis that pooled the relative risks from ten cancer epidemiology studies in which there was a presumed exposure to chlorinated water and its byproducts and estimated that approximately 10,000 cases of rectal

and bladder cancer cases per year could be associated with exposure to DBPs in chlorinated water in the United States. Later, Poole (1997) reviewed the studies available to Morris et al. (1992) plus three additional studies (McGeehin et al., 1993; Vena et al., 1993; and King and Marrett, 1996). Poole (1997) observed that there was considerable heterogeneity among the data and that there was evidence of publication bias within the body of literature. In addition, Poole found that the aggregate estimates reported by Morris et al. were sensitive to small changes in the analysis (e.g., addition or deletion of a single study). Based on the observations, Poole recommended that the cancer epidemiology data considered in the Morris evaluation should not be combined into a single summary estimate and that the data had limited utility for risk assessment purposes. Based on the available cancer epidemiology database, bladder cancer studies provide the strongest evidence for an association between exposure to chlorinated water and cancer. Based on the studies of Cantor et al. (1985), McGeehin et al. (1993), King and Marrett (1996), Freedman et al. (1997), and Cantor et al. (1998), EPA calculated that the population attributable risk (the fraction of a disease that could be eliminated if the exposure of concern were eliminated) for bladder cancer ranged from 2% to 17% (U.S. EPA, 1998c). However, these calculations are based on a number of assumptions, including the assumption that there is a cause-effect relationship between exposure to chlorinated drinking water and increased risk of bladder cancer. This assumption is subject to considerable uncertainty, especially because findings are not consistent within or between studies. Evaluation of these studies by application of standard criteria for establishing causality from epidemiological observations (strength of association, consistency of findings, specificity of association, temporal sequence, dose-response relation, biological plausibility) has led EPA to conclude that the current data are insufficient to establish a causal relationship between exposure to chloroform and increased risk of cancer (U.S. EPA, 1998a). Moreover, if, in the future, the weight-of-evidence does reach a point where a causal link is established between exposure to chlorinated water and increased risk of bladder or other types of cancer, it could not be concluded from epidemiological studies of this type that chloroform per se is carcinogenic in humans, as chlorinated water contains numerous disinfection byproducts besides chloroform that are potentially carcinogenic (U.S. EPA, 1998a).

II.A.3. Animal Carcinogenicity Data

Adequate. At high doses, chloroform has been reported to be carcinogenic in several chronic animal bioassays, with significant increases in the incidence of liver tumors in male and female mice and significant increases in the incidence of kidney tumors in male rats and mice (U.S. EPA, 1994, 1998c). When examining the biology of the tumor production, the occurrence of tumors is demonstrably species-, strain-, and gender-specific, and has only been observed under dose conditions that caused cytotoxicity and regenerative cell proliferation in the target organ.

In a gavage bioassay (NCI, 1976), Osborne-Mendel rats and B6C3F1 mice were treated with chloroform in corn oil 5 times/week for 78 weeks (50 animals per sex per dose group). Male rats received 90 or 125 mg/kg/day; females initially were treated with 125 or 250 mg/kg/day for 22 weeks and 90 or 180 mg/kg/day thereafter. A decrease in survival rate and weight gain was evident for all treated rats. A significant increase in kidney epithelial tumors was observed in male rats (0% in controls, 8% in the low dose and 24% in the high dose groups). Male mice received 100 or 200 mg/kg/day, raised to 150 or 300 mg/kg/day at 18 weeks; females were dosed with 200 or 400 mg/kg/day, raised to 250 or 500 mg/kg/day. Survival rates and weight gains were comparable for all groups except high dose female mice which had a decreased survival. In mice, highly significant increases in hepatocellular carcinomas were observed in both sexes (98% and 95% for males and females at the high dose; 36% and 80% for males and females at the low dose as compared with 6% of both matched and colony control males , 0% in matched control females and 1% in colony control females). Nodular hyperplasia of the

liver was observed in many low dose male mice that had not developed hepatocellular carcinoma. Hepatomas have also developed in female strain A mice and NLC mice gavaged with chloroform (Eschenbrenner and Miller, 1945; Rudali, 1967).

Jorgenson et al. (1985) administered chloroform (pesticide quality and distilled) in drinking water to male Osborne-Mendel rats and female B6C3F1 mice at concentrations of 200, 400, 900, and 1,800 mg/L for 104 weeks. These concentrations were reported by the author to correspond to 19, 38, 81, and 160 mg/kg/day for rats and 34, 65, 130, and 263 mg/kg/day for mice. The combined benign and malignant renal tumor incidence in male rats was 2%, 2%, 2%, 5%, 6% and 14% for the control, matched control, 19, 38, 81, and 160 mg/kg/day groups, respectively. A significant increase in renal tumors (14%) in rats was observed in the highest dose group (160 mg/kg/day). A reevaluation of the histopathology of the slides (Hard et al., 2000), found evidence of persistent cytotoxicity and regenerative hyperplasia in all rats of the highest dose group. Similar changes were also observed in rats at 81 mg/kg/day, but at a much lower incidence and grade. Thus, the histopathology reexamination provides evidence supporting chronic renal tubule injury as the mode of action underlying the renal tumor response. The liver tumor incidence in female mice was not significantly increased.

Chloroform administered in toothpaste was not carcinogenic to male C57B1, CBA, CF-1, or female ICI mice or to beagle dogs. Male ICI mice administered 60 mg/kg/day were found to have an increased incidence of kidney epithelial tumors (Roe et al., 1979; Heywood et al., 1979). A pulmonary tumor bioassay in strain A/St mice was negative, as was one in which newborn C57X DBA2/F1 mice were treated s.c. on days 1 to 8 of life (Theiss et al., 1977; Roe et al., 1968).

Matsushima (1994) exposed F344 rats (50/sex/group) and BDF1 mice (50/sex/group) to chloroform vapor 6 hours/day, 5 days/week for 104 weeks. Rats were exposed to concentrations of 0, 10, 30, or 90 ppm, and mice were exposed to 0, 5, 30, or 90 ppm. In order to avoid short- term lethality, mice in the two highest groups (30 and 90 ppm) were initially exposed to a lower levels for 2-6 weeks before the long-term exposure. The time-weighted average (TWA) for the 30 ppm group was 29.1 ppm and for the 90 ppm group was 85.7 ppm (U.S. EPA, 1998a). Statistically significant increases in the incidence of overall renal cell adenoma and renal cell carcinoma were observed in male mice in the 30 (7/50) and 90 (12/48) ppm groups, when compared to controls (0/50). The overall incidence rates of renal cell carcinoma were statistically significantly increased in males in the 90-ppm group (11/48) when compared to controls (0/50). There were no statistically significant findings reported for female mice in any exposure groups.

II.A.4. Supporting Data for Carcinogenicity

Mutagenicity

Many studies have investigated the mutagenic potential of chloroform. However, there are several reasons these studies must be reviewed carefully and interpreted cautiously. For example, chloroform is relatively volatile, so test systems not designed to prevent chloroform escape to the air may yield unreliable results. Earlier studies in which appropriate P450-based metabolic activation systems were absent are also likely to be unreliable. Further, some older studies that used ethanol as a solvent or preservative for chloroform may be confounded by formation of ethyl or diethyl carbonate, which are potent alkylating agents. Another important issue is that studies that focused on clastogenicity endpoints using excessively high doses may be confounded by severe cytotoxicity, causing lysosomal or other releases (Brusick, 1986).

In Vitro Studies

Two investigators reported DNA binding in studies with calf thymus DNA in the presence of exogenous activation (DiRenzo et al., 1982; Colacci et al., 1991). The study by DiRenzo et al. (1982) used ethanol as a solvent, suggesting that ethyl carbonate formation might be a problem. In the study by Colacci et al. (1991), addition of SKF-525A inhibited DNA binding, suggesting that binding was mediated by a cytochrome P-450 mediated pathway, as would be expected for chloroform. In interpreting these studies, it is important to remember that cell-free systems may not always be a good model for intact cellular processes.

Gene mutation studies in *Salmonella typhimurium* and *E. coli* (Ames assay), including tests done under conditions designed to reduce evaporation, are mostly negative, with or without activation with microsomes from liver or kidney of rats or mice (Rapson et al., 1980; San Agustin and Lim-Sylianco, 1978; Van Abbe et al., 1982; Uehleke et al., 1977; Gocke et al., 1981; Roland-Arjona et al., 1991; Le Curieux et al., 1995; Kirkland et al., 1981; Simmon et al., 1977). However, four studies have showed positive results in bacteria. Varma et al. (1988) reported that chloroform caused mutagenicity in five strains of *S. typhimurium*, but the response was noted only at the lowest dose tested, and all higher doses were not different from control. This unusual pattern casts some doubt on these results. San Agustin and Lim-Sylianco (1997) reported that chloroform caused DNA damage in *Bacillus subtilis*, and Wecher and Scher (1982) reported that chloroform caused mutations in *Photobacterium phosphoreum*. However, neither study reported the exposure concentrations that caused these effects, so the relevance of these reports is uncertain. In addition, the studies by Varma et al. (1988) and Wecher and Scher (1982) each used ethanol as a diluent, raising the possibility that the positive effect might be related to ethyl carbonate formation rather than to chloroform. The majority of results reported for *S. typhimurium* and *E. coli* exposed to the vapor phase were also negative (Van Abbe et al., 1982; Pegram et al., 1997; Simmon, 1977; Sasaki et al., 1998). Pegram et al. (1997) reported that chloroform was weakly positive at vapor concentrations greater than 19,200 ppm (about 770 mg/L in the aqueous phase). Employing physiologically based pharmacokinetic models, the authors estimated the oral doses needed to produce the effect would exceed 2,000 mg/kg (approximately twice the LD50).

Tests of genotoxicity are also mainly negative in fungi (Gualandi, 1984; Mehta and von Borstel, 1981; Kassinova et al., 1981; Jagannath et al., 1981). However, chloroform was shown to induce intrachromosomal recombination in *Saccharomyces cerevisiae* at concentrations of 6,400 mg/L (Callen et al., 1980) or 750 mg/L (Brennan and Schiestl, 1998). In the Brennan and Schiestl study, addition of *N*-acetylcysteine reduced chloroform-induced toxicity and recombination, suggesting a free radical may have been involved. Chromosome malsegregation was also reported in *Aspergillus nidulans* (Crebelli et al., 1988), but only at concentrations above 1,600 mg/L. In all three of these positive studies, doses that caused positive results also caused cell death, indicating that exposures were directly toxic to the test cells.

Studies in intact mammalian cells are mainly negative (Larson et al., 1994a; Perocco and Prodi, 1981; Butterworth et al., 1989; Kirkland et al., 1981; White et al., 1979; Sturrock, 1977), although positive results have been reported in a few systems. Increased sister chromatid exchange was reported in human lymphocytes at a concentration of about 1,200 mg/L without exogenous activation (Morimoto and Koizumi, 1983), and at a lower concentration (12 mg/L) with exogenous activation (Sobti, 1984). In the study by Sobti, the increase was quite small (less than 50%), and there was an increase in the number of cells that did not exclude dye. This suggests that the exposure levels that caused the mutagenic effect may have been directly toxic to the cells. In addition, ethanol was used as a dose vehicle. Mitchell et al. (1988) did not detect an increase in mutation in mouse lymphoma cells at an exposure level of 2,100 mg/L in the absence of exogenous activation, but did detect an effect at a concentration of 59 mg/L with exogenous activation.

In Vivo Studies

A number of different endpoints of chloroform genotoxicity have been measured in intact animals exposed to chloroform either orally or by inhalation. In studies of DNA binding in liver and kidney of mice and rats, negative results have been reported at doses of 742 mg/kg, 119 mg/kg, and 48 mg/kg (Diaz-Gomez and Castro, 1980; Reitz et al., 1982; Pereira et al., 1982). However, positive results have been reported at doses as low as 2.9 mg/kg (Colacci et al., 1991). But, in the study by Colacci et al. (1991), no significant difference in binding was noted between multiple tissues (liver, kidney, lung, and stomach), and there was no increase in binding with phenobarital pretreatment. This suggests the binding may not have been related to chloroform metabolism.

Studies based on signs of DNA damage or repair have been uniformly negative (Larson et al., 1994a; Potter et al., 1996; Reitz et al., 1982; Mirsalis et al., 1982). However, studies based on various signs of chromosomal abnormalities have been mixed, with some studies reporting negative findings at doses of 371 mg/kg and 800 mg/kg (Shelby and Witt, 1995; Topham, 1980), while other studies report positive results at doses as low as 1.2 mg/kg (Fujie et al., 1990). However, the positive result at low dose in the study by Fujie et al. (1990) was observed following intraperitoneal exposure, and positive results following oral exposure were not observed until a dose level of 119 mg/kg. Morimoto and Koizumi (1983) observed an increase in the frequency of sister chromatid exchange in bone marrow cells at a dose of 50 mg/kg/day, but at 200 mg/kg/day, all of the mice died. As discussed before, mutagenicity results observed following highly toxic doses may have been confounded by cytotoxic responses and should be viewed as being of uncertain relevance.

Several studies have reported negative findings for the micronucleus test in rats and mice (Gocke et al., 1981; Salamone et al., 1981; Le Curieux, 1995), but several other studies have detected positive results, mainly at exposure levels of 400-600 mg/kg (San Agustin and Lim-Sylianco, 1982; Robbiano et al., 1998; Sasaki et al., 1998; Shelby and Witt, 1995). This suggests that chloroform may be clastogenic, but it is important to note that these doses are well above the level that causes cytotoxicity in liver and kidney in most oral exposure studies in rodents.

Butterworth et al. (1998) did not detect an increase in mutation frequency in male mice exposed by inhalation at an exposure level of 90 ppm, even though this exposure did cause an increase in tumors in the study by Nagano et al. (1998). Increased incidence of sperm head abnormalities was reported in mice exposed at 400 ppm (Land et al., 1981), but was not observed in mice exposed to 371 mg/kg intraperitoneally (Topham, 1980).

In *Drosophila melanogaster* larvae exposed to chloroform vapor, gene mutation (Gocke et al., 1981) and mitotic recombination tests (Vogel and Nivard, 1993) were both negative. Grasshopper embryos (*Melanoplus sanguinipes*) did not display mitotic arrest at vapor concentrations of 30,000 ppm, but an effect was seen at 150,000 ppm (Liang et al., 1983). San Agustin and Lim-Syllianco (1981) reported a single positive and negative result for host-mediated mutagenicity in *Salmonella typhimurium*, but exposure levels were not reported in either case.

On the basis of the in vitro and in vivo studies reviewed above, even though a role of mutagenicity cannot be completely ruled out, the majority of available studies are negative, and many of the positive studies have limitations (excessive doses or other confounding factors). Thus, the weight-of-evidence supports the conclusion that chloroform is not strongly mutagenic, and that genotoxicity is not likely to be the predominant mode of action underlying the carcinogenic potential of chloroform. This conclusion is supported by a number of other

groups who have reviewed and evaluated the available data on chloroform genotoxicity, including the International Commission for Protection against Environmental Mutagens and Carcinogens (Lohman et al., 1992), ILSI (1997), Health Canada (2000), and WHO (1998).

Mode of Action

1. Summary of Postulated Mode of Action

Studies in animals reveal that chloroform can cause an increased incidence of kidney tumors in male rats and an increased incidence of liver tumors in male and female mice. Available data suggest that tumors are produced only at dose levels that result in cytotoxicity. These induced tumor responses are postulated to be secondary to sustained or repeated cytotoxicity and secondary regenerative hyperplasia. Chloroform's carcinogenic effects in rodent liver and kidney are attributed to oxidative metabolism-mediated cytotoxicity in the target organs. Although chloroform undergoes both oxidative and reductive cytochrome P450-mediated metabolism, it is the oxidative (CYP2E1) metabolic pathway that predominates at low chloroform exposures. This oxidative pathway produces highly tissue-reactive metabolites (in particular phosgene) that lead to tissue injury and cell death. It is likely that the electrophilic metabolite phosgene causes cellular toxicity by reaction with tissue proteins and cellular macromolecules as well as phospholipids, glutathione, free cysteine, histidine, methionine, and tyrosine. The liver and kidney tumors induced by chloroform depend on persistent cytotoxic and regenerative cell proliferation responses. The persistent cell proliferation presumably would lead to higher probabilities of cell mutation and subsequent cancer. The weight of the evidence indicates that a mutagenic mode of action via DNA reactivity is not a significant component of the chloroform carcinogenic process.

2. Identification of key events

There are essentially three key steps in the sequence of events that lead to chloroform-induced tumorigenesis in the liver and kidneys of rodents. The first step is oxidative metabolism of chloroform in the target organs, kidney and liver. Numerous binding and metabolism studies (as described in ILSI, 1997, and U.S. EPA, 1998a) provide support that chloroform is metabolized by the oxidative cytochrome P450 (CYP2E1) pathway. This conclusion is supported by the study of Constan et al. (1999) in Sv/129 wild type, Sv/129 CYP2E1 null, and B6C3F1 mice. In the wild type of each strain, exposure to 90 ppm chloroform for 6 hours per day for 4 consecutive days resulted in severe hepatic and renal lesions along with increased cell proliferation. With the same exposure, neither the cytotoxicity nor cell proliferation occurred in the CYP2E1 null mouse or in the wild type of either strains treated with the P450 inhibitor ABT.

Available evidence indicates that metabolism by CYP2E1 predominates at low exposures and is rate-limiting to chloroform's carcinogenic potential. Reductive metabolism, if it occurs, can lead to free radicals and tissue damage, but this pathway is absent or minor under normal physiological conditions. The next key step is the resultant cytotoxicity and cell death caused by the oxidative metabolites (with phosgene as the significant toxic intermediate). Regenerative cell proliferation follows the hepatotoxicity and nephrotoxicity as measured by labeling index in mouse kidney and liver and rat kidney from chloroform-treated animals. This increase in cell division is responsible for the increased probability of cancer.

3. Strength, consistency, specificity of association

There are numerous cases where exposure to chloroform causes an increase in cytotoxicity (as evidenced by histopathological evaluation and/or increased labeling index) without any observable increase in cancer incidence. These data indicate that chloroform exposures that are

adequate to cause cytotoxicity and regenerative cell proliferation do not always lead to cancer. However, there are no cases where a tumorogenic response has been observed in which evidence of cell regeneration is not also observed at the same or lower dose as that which caused an increase in tumors. This consistency of evidence (i.e., cell regeneration is detected in all cases of tumorigenicity) is strong evidence supporting the conclusion that cell regeneration is a mandatory precursor for tumorigenicity.

Evidence for a link between sustained cytotoxicity/regenerative hyperplasia and cancer is strongest in the kidney. In male Osborne-Mendel rats exposed to chloroform in water for 2 years (Jorgenson et al., 1985), a statistically significant increase in renal tumors was observed at a concentration of 1,800 ppm (160 mg/kg/day). A re-analysis of the histopathological slides from this study (Hard et al., 2000) revealed evidence for sustained cytotoxicity and cell proliferation in the kidney at exposures of 900 ppm (81 mg/kg/day) or higher. Likewise, in BDF_1 mice exposed to chloroform by inhalation at 5, 30, or 90 ppm for 6 hours/day, 5 days/week (Nagano et al., 1998), increased incidence of renal tumors was observed in male mice at the two higher doses, whereas females showed no significant tumor response. Templin et al. (1998) duplicated this exposure regimen in order to study whether the treatment caused cytotoxicity and regenerative hyperplasia. These authors observed cytotoxicity and hyperplasia in the kidneys of male mice exposed to 30 or 90 ppm throughout a 90-day exposure period, but not in females. This observation is consistent with the hypothesis that sustained cytotoxicity and regenerative hyperplasia are key events in the neoplastic response of the kidney to chloroform.

Available data also indicate that cytotoxicity and regenerative hyperplasia are required for liver cancer, although the strength of this conclusion is somewhat limited because most of the observations are based on short-term rather than long-term histological or labeling index measurements. For example, in the B6C3F1 mouse, corn oil gavage (bolus dosing) at the same doses that resulted in liver tumors in the study by NCI (1976) also caused hepatic cytolethality and a cell proliferative response at both 4 days and 3 weeks (Larson et al., 1994b,c). Similarly, exposure of female B6C3F1 mice to chloroform in drinking water at levels that did not induce liver tumors (Jorgenson et al., 1985) also did not induce hepatic cytolethality or cell proliferation at 4 days or 3 weeks (Larson et al., 1994b). This consistency of the data (i.e., evidence of cytolethality and/or regenerative hyperplasia is always observed in cases of increased liver tumors) supports the conclusion that this liver cancer also occurs via a mode of action involving regenerative hyperplasia.

4. Dose-response relationship

Chloroform-induced liver tumors in mice are only seen after bolus corn oil dosing. Mouse liver tumors are not found following administration by other routes (drinking water and inhalation). Rat liver tumors are not induced by chloroform following either drinking water or corn oil gavage administration. Kidney tumors are found in mice exposed to chloroform via inhalation or toothpaste preparations, and in rats when exposed via drinking water or corn oil gavage. Kidney and liver tumors develop only at doses that cause persistent cytotoxicity and regenerative proliferation, regardless of route of exposure or dosing regime. The overall dose-response for the cytotoxicity and cell proliferation responses is nonlinear. All key events and tumor effects depend on the dose-rate as shown by the difference in oil gavage versus drinking water administration (ILSI, 1997; U.S. EPA, 1998a).

5. Temporal relationship

As noted above, there is very strong evidence from short-term and long-term histological and labeling index studies in mice and rats that cytotoxicity and cell proliferation always precede

the occurrence of increased kidney or liver tumor effects in long-term bioassays. For example, a re-evaluation of serial sacrifice data from the chloroform 2-year drinking water bioassay in Osborne-Mendel rats revealed a linkage between toxicity in the renal tubules and tumor development and showed that renal toxicity preceded tumor development (Hard and Wolf, 1999; Hard et al., 2000).

6. Biological plausibility and coherence

The theory that sustained cell proliferation to replace cells killed by toxicity, viral, or other insults such as physical abrasion of tissues can be a significant risk factor for cancer is plausible and generally accepted (Correa, 1996). It is logical to deduce that sustained cytotoxicity and regenerative cell proliferation may result in a greater likelihood of mutations being perpetuated with the possibility of more of these resulting in uncontrolled growth. It may also be that continuous stimulus of proliferation by growth factors involved in inflammatory responses increases the probability that damaged cells may slip through cell cycle check points carrying DNA alterations that would otherwise be repaired. Current views of cancer processes support both these possibilities. There are no data on chloroform that allow the events that occur during cell proliferation to be directly observed. A high proliferation rate alone is not assumed to cause cancer; tissues with naturally high rates of turnover do not necessarily have high rates of cancer and tissue toxicity in animal studies does not invariably lead to cancer. Nevertheless, regenerative proliferation associated with persistent cytotoxicity appears to be a risk factor of consequence.

7. Role of genotoxicity

As noted above, the question whether chloroform or a metabolite is mutagenic has been tested extensively across different phylogenetic orders (i.e., bacterial, eukaryotic, and mammalian systems). Predominately negative results are reported in all test systems, with no pattern of mutagenicity seen in any one system considered to be a competent predictor. Positive results appear sporadically in the database, but they generally have problems with high dose or other confounding issues. ILSI (1997) considered results from 40 tests by the quantitative weight-of-evidence method for heterogeneous genetic toxicology databases from the International Commission for Protection against Environmental Mutagens and Carcinogens (ICEMC) (Lohman et al., 1992). This method scores relative DNA reactivity, with a maximum positive score being +100 and maximum negative -100. The maximum positive score obtained among 100 chemical databases has been +49.7 (triazaquone) and the maximum negative has been -27.7. The score for chloroform was -14.3.

Testing of chloroform in the p53 heterozygous knockout mouse shows no tumor effect (Gollapudi et al., 1999). Heterozygous p53 males were dosed up to 140 mg/kg and females up to 240 mg/kg via corn oil gavage for 13 weeks. This model is known to respond most effectively to mutagenic carcinogens.

Products of oxidative and reductive metabolism of chloroform are highly reactive. Such species are unstable and will likely react with cytoplasmic molecules before reaching nuclear DNA. Such reactive species (e.g., phosgene) have not been evaluated separately for genetic toxicity, and because of reactivity, would not be amenable to study and would not likely be able to transport from the cellular site of production to the nucleus.

Comparative examination of both oxidative and reductive metabolism for structural analogues and chloroform has revealed that carbon tetrachloride, which is largely metabolized to a free radical via the reductive pathway, results in cell toxicity, not mutagenicity. Moreover, chloroform and carbon tetrachloride show very different patterns of liver toxicity (i.e., carbon

tetrachloride's toxicity is more consistent with free radical production and chloroform's is not). For methylene chloride, glutathione conjugation results in mutagenic metabolites. When rat glutathione transferase gene copies are introduced into *Salmonella*, bromodichloromethane produces mutagenic metabolites; the fact that chloroform in this system did so only marginally and only at high toxic doses (Pegram et al., 1997) supports a conclusion that the reductive pathway does not contribute to chloroform's toxicity or carcinogenicity.

In initiation-promotion studies, chloroform at the highest test dose of the drinking water bioassay does not promote development of hepatic lesions in rats or two strains of mice, nor does it initiate or act as a cocarcinogen. Administered in oil, chloroform was a promoter in the rat liver in initiation-promotion protocols. These results are more consistent with the postulated mode of action than with any mutagenic potential.

8. Effects on children

The central questions asked in a mode of action analysis are, 1) whether the standard assumption that a mode of action observed in animals is relevant to humans holds true in a particular case, and 2) what the nature of the mode of action implies about the shape of the dose response relationship. In the case of chloroform the conclusions have been that the rodent mode of action can be assumed to be relevant to humans and that a nonlinear approach is most appropriate. The next question is whether the data lead one to anticipate similarities or differences in response by sex or age.

Ideally, one would have adequate data to compare each of the key events of chloroform toxicity and subsequent carcinogenicity in tissues of adults with those of the developing fetus and young. This kind of information is currently not to be found. In the absence of data on the fetus and young specific to chloroform, an evaluation is made as to whether a cogent biological rationale exists for determining that the postulated mode of action is applicable to children (EPA, 1999). There is no suggestion from available studies of chloroform to indicate that children or fetuses would be qualitatively more sensitive to its effects than adults. The developing organism would not be expected to be particularly sensitive to cytotoxic agents at minimally toxic levels because cell division is proceeding rapidly and repair capacity at the molecular and cellular level is high. This is reflected by the relatively low incidence of spontaneous tumors in developing and young organisms. Moreover, the reproductive and developmental studies available, while they have limitations, show that fetal effects are seen only at doses at which maternal toxicity is evident. Research would be needed to further explore whether there are circumstances in which this relationship does not hold. Research would also be needed to discover whether there is some other mode of action, not seen in rodents, that might be possible. Presently, there are no clues from in vivo or in vitro studies as to what alternative mode of action might be considered. In keeping with traditional toxicologic evaluations, chloroform has been tested in lifetime studies with high level doses to provide maximal opportunities for toxicologic effects to manifest themselves in multiple tissues and organs through multiple mechanisms. In the absence of data to the contrary, this approach is considered to provide evidence for lack of potential for significant response, other than those noted, even for sensitive individuals and life stages.

The mode of action analyzed as well as all other potential modes of action identified required that chloroform be metabolized by cytochrome P450 (CYP2E1) (SAB (2000), p.2). When this is considered along with the comparison of this enzyme activity between adults and the young there is confidence in assuming similarity in response among life stages. Further research on the processes of cell injury, death and regeneration would increase this confidence by addressing any uncertainty about potential quantitative similarity. The literature does not reveal any such quantitative data at present.

Given the above, it is reasonable to assume that: 1) The reactive metabolite inside the cell should have similar effects by reacting with and disrupting macromolecules in the cells of fetuses, children and adults, 2) Cell necrosis and reparative replication are not likely to be qualitatively different in various stages in life, 3) Cancer risk to the fetus or children would be a function of cytotoxic injury, like in adults, and protecting these life stages from sufficient cytotoxicity to elicit this response should protect against cancer risks. Further research would be needed to assess whether there are significant quantitative differences between life stages which have not yet been elucidated.

It can be noted that if data indicated that it were appropriate to apply a linear approach to part of a lifetime, such as the first 3 years of life, the resulting risk would be represented by a small increment of the total dose per body weight over a lifetime since most of a 70 year life is at an adult body weight. When this total is divided by 70 years to derive the lifetime average daily dose, the small increment of early dose does not significantly increase risk.

9. Conclusion regarding cancer mode of action

The weight of the evidence supports the conclusion that chloroform-induced tumors in liver and kidney are produced only at dose levels that result in repeated or sustained cytotoxicity and regenerative cell proliferation. A wide range of evidence across different species, sexes, and routes of exposure implicates oxidative CYP2E1 metabolism leading to persistent cytotoxicity and regenerative cell proliferation as events that precede and are associated with tumor formation. The cytochrome P450 oxidative metabolism that leads to oxidative damage and ensuing cell growth, involving basic tissue responses to cellular toxicity and death, is common to humans and rodents. No data exist indicating that the mode of action observed in rodents is not also likely to apply to humans.

Available data on the mutagenic potential of chloroform are mixed, but the majority of tests are negative, and some of the positive results are observed only at extreme exposure conditions. Thus, the weight of the evidence indicates that chloroform is not a strong mutagen and that neither chloroform nor its metabolites readily bind to DNA. On the basis of these results and the results of studies that evaluated other endpoints of mutagenicity, it seems likely that even though a role for mutagenicity cannot be excluded with certainty, chloroform does not produce carcinogenic effects primarily by a specific genotoxic mechanism.

The proposed dose-response relationship for chloroform tumorigenesis by the cytotoxicity-regenerative hyperplasia mode of action will be nonlinear, as it is dependent on biochemical and histopathological events that are nonlinear. The dose-response assessment would ideally be based on use of phosgene dosimetry because it marks the rate-limiting step of oxidative metabolism. The toxicokinetic modeling to support this phosgene approach is not currently available, so the dose-response assessment is based on the tumor precursor event of cytotoxicity to project a level of exposure that will be protective against the key event of regenerative hyperplasia.

_II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

In accord with proposed EPA guidelines for cancer risk assessment (U.S. EPA, 1996), the method used to characterize and quantify cancer risk from a chemical depends on what is known about the mode of action of carcinogenicity and the shape of the cancer dose-response curve for that chemical. A default assumption of linearity is appropriate when evidence supports a mode of action of gene mutation due to DNA reactivity, or another mode of action that is anticipated to be linear. The linear approach is used as a matter of policy if the mode of

action of carcinogenicity is not understood. Alternatively, an assumption of nonlinearity is appropriate when there is no evidence for linearity and sufficient evidence to support an assumption of nonlinearity. In this case, the carcinogenicity may be a secondary effect of toxicity that itself is a threshold phenomenon (U.S. EPA, 1996).

In the case of chloroform, the mode of action of carcinogenicity is reasonably well understood. Available data indicate that chloroform is not strongly mutagenic and chloroform is not expected to produce rodent tumors via a mutagenic mode of action (ILSI, 1997). Rather, there is good evidence that carcinogenic responses observed in animals are associated with regenerative hyperplasia that occurs in response to cytolethality (ILSI, 1997; U.S. EPA, 1998a,b). Because cytolethality occurs only at exposure levels above some critical dose level, a nonlinear approach is considered the most appropriate method for characterizing the cancer risk from chloroform.

The Proposed Guidelines for Carcinogenic Risk Assessment (U.S. EPA, 1996) state that when the mode-of-action analysis based on available data indicates that "the carcinogenic response is secondary to another toxicity that has a threshold, the margin-of-exposure analysis performed for toxicity is the same as is done for a noncancer endpoint, and an RfD for that toxicity may be considered in the cancer assessment." For chloroform, available evidence indicates that chloroform-induced carcinogenicity is secondary to cytotoxicity and regenerative hyperplasia; hence, the Agency relies on a nonlinear dose-response approach and the use of a margin-of-exposure analysis for cancer risk. The Agency has also chosen not to rely on a mathematical model to estimate a point of departure for cancer risk estimate, because the mode of action indicates that cytotoxicity is the critical effect and the reference dose value is considered protective for this effect.

RfD and Margin of Exposure

For more discussion of margin of exposure (MOE), see the Toxicological Review for Chloroform. Based on the kidney tumor of the drinking water study (Jorgenson et al., 1985), a point of departure (Pdp or LED_{10}) of 23 mg/kg/day can be calculated using quantitative modeling of tumor dose-response data. Comparing the Pdp to the RfD of 0.01 mg/kg/day leads to a MOE of 2,000, which is considered large. Thus, in this case, the RfD for noncancer effect is also considered adequately protective of public health for cancer effects by the oral route, on the basis of the nonlinear dose response for chloroform and the mode of action for both cancer and noncancer effects having a common link through cytotoxicity.

As discussed above, the RfD for noncancer effects is derived from the most sensitive endpoint in the most sensitive species. The RfD is based on fatty cysts formation (fat accumulation) in the liver and elevation of SGPT in dogs (Heywood et al., 1979). Hepatic fat accumulation and elevated SGPT are considered early signs of impaired liver function resulting from chloroform-induced cytotoxicity. This effect occurs at doses at or below those that cause increased labeling index, morphological changes, or cellular necrosis, so protection against this effect is believed to protect against cytolethality and regenerative hyperplasia. Accordingly, the RfD of 0.01 mg/kg/day presented in Section I.A.1 can be considered protective against increased risk of cancer.

__II.B.1. Summary of Risk Estimates

A dose of 0.01 mg/kg/day (equal to the RfD) can be considered protective against cancer risk

____II.B.1.1. Oral Slope Factor — Not applicable (see text).

_____II.B.1.2. Drinking Water Unit Risk — Not applicable (see text).

__II.B.2. Dose-Response Data (Carcinogenicity, Oral Exposure)

Dose-response data used to derive the RfD for chloroform are presented in Section I.A.2.

__II.B.3. Additional Comments (Carcinogenicity, Oral Exposure)

Because chloroform is a volatile chemical, exposure to chloroform in drinking water may occur not only via direct ingestion, but also by inhalation of chloroform released from household uses of water (showering, cooking, washing, etc.) into indoor air. Therefore, assessments of cancer and noncancer health effects from chloroform in water should account for exposures by all pathways, including oral, inhalation, and dermal.

__II.B.4. Discussion of Confidence (Carcinogenicity, Oral Exposure)

Confidence in the cancer assessment for chloroform is rated as medium. This is based on a strong database in animals that supports the conclusion that cancer does not occur without antecedent cytotoxicity and regenerative hyperplasia, leading in turn to the conclusion that cancer risk is negligible at doses that do not result in cytotoxicity. Confidence in this conclusion is tempered by absence of direct studies in humans and by the finding that there are some positive results in studies on the mutagenicity of chloroform, even though the weight-of-evidence indicates that chloroform is not a strong mutagen and that a mutagenic mode of action is not likely to account for the cancer responses observed in animals.

EPA is currently revising its guidelines for cancer risk assessment. Among other issues, EPA is looking closely at how to assess whether a postulated mode of action in adults is applicable to children. When the guidelines are final, EPA will consider their impact on existing health assessments on IRIS.

_II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

NOTE: The following evaluation of cancer risk from chloroform inhalation was developed in 1987 and does not incorporate newer data or the 1996 or 1999 draft cancer assessment guidelines. EPA is currently working to revise the assessment for inhalation exposure.

__II.C.1. Summary of Risk Estimates

_____II.C.1.1. Inhalation Unit Risk — 2.3E-5 per (ug/m3).

_____II.C.1.2. Extrapolation Method — Linearized multistage procedure, extra risk.

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E+0 µg/m3
E-5 (1 in 100,000)	4E-1 µg/m3
E-6 (1 in 1,000,000)	4E-2 µg/m3

__II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure

Tumor Type — hepatocellular carcinoma
Test Animals — mouse, B6C3F1, female
Route — oral, gavage
Reference — NCI, 1976

Dose		
Administered (mg/kg/day)	Human Equivalent (mg/kg/day)	Tumor Incidence
Female		
0	0	0/20
238	9.9	36/45
477	19.9	39/41
Male		
0	0	1/18
138	6.2	18/50
277	12.5	44/45

__II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)

This inhalation quantitative risk estimate is based on data from a gavage study. Above doses are TWA; body weights at the end of the assay were 35 g for males and 28 g for females. Vehicle control animals were run concurrently and housed with test animals. All treated animals experienced decreased body weight gain. Survival was reduced in high-dose males and in all treated females. Experimental data for this compound support complete absorption of orally administered chloroform under conditions of this assay. There are no apparent species differences in this regard. Extrapolation of metabolism-dependent carcinogenic responses from mice to humans on the basis of body surface area is supported by experimental data. The incidence data for both male and female mice were used to derive slope factors of 3.3E-2 and 2.0E-1 per (mg/kg)/day, respectively. The unit risk was prepared by taking a geometric mean of the slope factor and assuming 100% for low doses of chloroform in air. The unit risk should not be used if the air concentration exceeds 400 $\mu g/m^3$, because above this concentration the unit risk may not be appropriate.

__II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure)

Adequate numbers of animals were treated and observed. Risk estimates derived from male rat kidney tumor data (2.4E-2) (NCI, 1976) and studies by Roe et al. (1979) (1.0E-1) are generally supportive of the risk estimate.

_II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

__II.D.1. EPA Documentation

Source Document — U.S. EPA, 2001 (oral carcinogenicity assessment); U.S. EPA, 1985, 1987 (inhalation carcinogenicity assessment)

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS Summary. A record of comments on the oral carcinogenicity assessment is included in an appendix to U.S. EPA (2001). ***To review this appendix, exit to the toxicological review, Appendix A, External Peer Review -- Summary of Comments and Disposition (PDF)***.

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Consensus Date (oral carcinogenicity assessment) -- 7/27/2001
Verification Date (inhalation carcinogenicity assessment) - 8/26/1987

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Chloroform
CASRN — 67-66-3
Last Revised — 10/19/01

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_VII. Revision History

Chloroform
CASRN — 67-66-3

Date	Section	Description
03/01/1988	I.A.1.	Dose conversion clarified
03/01/1988	I.A.2.	LOAEL and RfD in text corrected
03/01/1988	I.A.4.	Text revised
03/01/1988	I.A.5.	Text revised
06/30/1988	II.	Carcinogen summary on-line
06/30/1988	I.A.7.	Primary contact changed
10/01/1989	I.B.	Inhalation RfD now under review
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)
02/01/1991	II.C.3.	Information on extrapolation process included
02/01/1991	II.C.4.	Text edited
03/01/1991	II.D.3.	Primary contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
07/01/1992	I.A.	Clarify Schwetz citation
07/01/1992	VI.C.	Oral RfD references on-line
07/01/1992	VI.C.	Carcinogenicity assessment references on-line
09/01/1992	I.A.7.	Primary contact changed
08/01/1995	I.B.	EPA's RfD/RfC and CRAVE workgroups were discontinued in May, 1995. Chemical substance reviews that were not completed by September 1995 were taken out of IRIS review. The IRIS Pilot Program replaced the workgroup functions beginning in September, 1995.
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.
12/10/1998	I.B.	This chemical is being reassessed under the IRIS Program.
10/19/2001	I.A.,VI	Oral RfD and references updated

10/19/2001	II.B.,VI	Oral carcinogenicty assessment and references updated
01/08/2002	II.C.1.	Corrected typographical error in units in inhalation unit risk.
03/26/2002	Tox. Review	Corrected list of external peer reviewers.

_VIII. Synonyms

Chloroform
CASRN — 67-66-3
Last Revised — 10/19/01

- 67-66-3
- Chloroform
- Formyl Trichloride
- Freon 20
- Methane Trichloride
- Methane, Trichloro-
- Methenyl Chloride
- Methenyl Trichloride
- Methyl Trichloride
- NCI-C02686
- R-20
- TCM
- Trichloroform
- Trichloromethane

IRIS Home

Chronic Health Hazards for Non-Carcinogenic Effects

Reference Dose for Chronic Oral Exposure (RfD)

- Oral RfD Summary
- Principal and Supporting Studies
- Uncertainty and Modifying Factors
- Additional Studies/Comments
- Confidence in the Oral RfD
- EPA Documentation and Review

Reference Concentration for Chronic Inhalation Exposure (RfC)

- Inhalation RfC Summary
- Principal and Supporting

Material Safety Data Sheet
Chloroform

ACC# 04770

Section 1 - Chemical Product and Company Identification

MSDS Name: Chloroform
Catalog Numbers: AC95232184, S79960, S79960-1, S79960HPLC-2, S79960SPEC-1, S79960SPEC-2, C2974LC, C297POP19, C297POP200, C297POP50, C297RS115, C297RS200, C297RS28, C297RS50, C297SS115, C297SS19, C297SS200, C297SS28, C297SS50, C29820LC, C298FB115, C298FB19, C298FB200, C298FB50, C298J1, C298POP19, C298POP200, C298POP50, C298POPB19, C298POPB200, C298POPB50, C298RB115, C298RB19, C298RB200, C298RB50, C298RB500, C298RS115, C298RS19, C298RS200, C298RS28, C298RS50, C298SS-11, C298SS19, C298SS28, C605-1, C605-4, C606POP19, C606POP200, C606POP50, C606RS115, C606RS200, C606RS28, C606RS50, C606SS115, C606SS19, C606SS200, C606SS28, C606SS50
Synonyms: Formyl Trichloride; Methane Trichloride; Methenyl Trichloride; Methyl Trichloride; Trichlormethan; Trichloroform; Trichloromethane.
Company Identification:

Fisher Scientific
1 Reagent Lane
Fair Lawn, NJ 07410

For information, call: 201-796-7100
Emergency Number: 201-796-7100
For CHEMTREC assistance, call: 800-424-9300
For International CHEMTREC assistance, call: 703-527-3887

Section 2 - Composition, Information on Ingredients

CAS#	Chemical Name	Percent	EINECS/ELINCS
67-66-3	Chloroform	100	200-663-8
25377-72-4	Amylene	<1.0	246-916-6

Hazard Symbols: XN
Risk Phrases: 22 38 40 48/20/22

Section 3 - Hazards Identification

EMERGENCY OVERVIEW

Appearance: clear, colorless liquid. May cause central nervous system depression. May cause cardiac disturbances. May cause cancer based on animal studies. This substance has caused adverse reproductive and fetal effects in animals. May be harmful if swallowed. **Caution!** Causes eye and skin irritation. Causes digestive and respiratory tract irritation. Light sensitive.
Target Organs: Blood, kidneys, heart, central nervous system, liver, cardiovascular system, excretory system, reproductive system.

Potential Health Effects
Eye: Causes moderate eye irritation. Contact with liquid causes immediate burning pain, tearing, and reddening of the conjunctiva.
Skin: Causes mild skin irritation. Prolonged or repeated contact may dry/defat the skin and cause irritation. Absorption of liquid through intact skin is possible and may cause sys temic poisoning if contact with liquid is prolonged.
Ingestion: Causes gastrointestinal irritation with nausea, vomiting and diarrhea. May cause liver damage. May cause cardiac disturbances. Aspiration of material into the lungs may cause chemical pneumonitis, which may be fatal. Possible aspiration hazard. May cause hallucinations and distorted perceptions.
Inhalation: Inhalation of high concentrations may cause central nervous system effects characterized by

nausea, headache, dizziness, unconsciousness and coma. May cause cardiac sensitization and possible failure. Inhalation of large amounts may cause respiratory stimulation, followed by respiratory depression, convulsions and possible death due to respiratory paralysis. May be absorbed through the lungs. Causes irritation of the mucous membrane and upper respiratory tract.
Chronic: Possible cancer hazard based on tests with laboratory animals. Prolonged or repeated skin contact may cause dermatitis. May cause reproductive and fetal effects. Effects may be delayed. Laboratory experiments have resulted in mutagenic effects. Toxicity may be increased by exposure to alcohol, steroids, and ketones. Prolonged exposure may cause liver, kidney, and heart damage.

Section 4 - First Aid Measures

Eyes: Immediately flush eyes with plenty of water for at least 15 minutes, occasionally lifting the upper and lower eyelids. Get medical aid.
Skin: Get medical aid. Flush skin with plenty of water for at least 15 minutes while removing contaminated clothing and shoes. Wash clothing before reuse.
Ingestion: Do NOT induce vomiting. If victim is conscious and alert, give 2-4 cupfuls of milk or water. Never give anything by mouth to an unconscious person. Get medical aid immediately.
Inhalation: Get medical aid immediately. Remove from exposure and move to fresh air immediately. If not breathing, give artificial respiration. If breathing is difficult, give oxygen.
Notes to Physician: Causes cardiac sensitization to endogenous catelcholamines which may lead to cardiac arrhythmias. Do NOT use adrenergic agents such as epinephrine or pseudoepinephrine. Persons with liver, kidney, or central nervous system diseases may be at increased risk from exposure to this product. Alcoholic beverage consumption may enhance the toxic effects of this substance. Effects may be delayed.

Section 5 - Fire Fighting Measures

General Information: As in any fire, wear a self-contained breathing apparatus in pressure-demand, MSHA/NIOSH (approved or equivalent), and full protective gear. During a fire, irritating and highly toxic gases may be generated by thermal decomposition or combustion. Use water spray to keep fire-exposed containers cool. Substance is nonflammable. Vapors may be heavier than air. They can spread along the ground and collect in low or confined areas. Containers may explode when heated.
Extinguishing Media: Use extinguishing media most appropriate for the surrounding fire. Do NOT get water inside containers. Do NOT use straight streams of water. For small fires, use dry chemical, carbon dioxide, or water spray. For large fires, use water spray, fog or regular foam. Cool containers with flooding quantities of water until well after fire is out.
Flash Point: Not available.
Autoignition Temperature: Not available.
Explosion Limits, Lower: Not available.
Upper: Not available.
NFPA Rating: (estimated) Health: 2; Flammability: 0; Instability: 0

Section 6 - Accidental Release Measures

General Information: Use proper personal protective equipment as indicated in Section 8.
Spills/Leaks: Absorb spill with inert material (e.g. vermiculite, sand or earth), then place in suitable container. Avoid runoff into storm sewers and ditches which lead to waterways. Clean up spills immediately, observing precautions in the Protective Equipment section. Provide ventilation. Approach spill from upwind.

Section 7 - Handling and Storage

Handling: Wash thoroughly after handling. Remove contaminated clothing and wash before reuse. Use only in a well-ventilated area. Avoid contact with eyes, skin, and clothing. Do not breathe dust, vapor, mist, or gas. Do not ingest or inhale. Store protected from light.

Storage: Do not store in direct sunlight. Store in a cool, dry, well-ventilated area away from incompatible substances. Do not store near alkaline substances. Separate from strong mineral acids.

Section 8 - Exposure Controls, Personal Protection

Engineering Controls: Facilities storing or utilizing this material should be equipped with an eyewash facility and a safety shower. Use adequate general or local exhaust ventilation to keep airborne concentrations below the permissible exposure limits.

Exposure Limits

Chemical Name	ACGIH	NIOSH	OSHA - Final PELs
Chloroform	10 ppm TWA	500 ppm IDLH	50 ppm Ceiling; 240 mg/m3 Ceiling
Amylene	none listed	none listed	none listed

OSHA Vacated PELs: Chloroform: 2 ppm TWA; 9.78 mg/m3 TWA Amylene: No OSHA Vacated PELs are listed for this chemical.

Personal Protective Equipment

Eyes: Wear appropriate protective eyeglasses or chemical safety goggles as described by OSHA's eye and face protection regulations in 29 CFR 1910.133 or European Standard EN166.

Skin: Wear appropriate protective gloves to prevent skin exposure.

Clothing: Wear appropriate protective clothing to prevent skin exposure.

Respirators: A respiratory protection program that meets OSHA's 29 CFR 1910.134 and ANSI Z88.2 requirements or European Standard EN 149 must be followed whenever workplace conditions warrant a respirator's use.

Section 9 - Physical and Chemical Properties

Physical State: Liquid
Appearance: clear, colorless
Odor: sweet, fruity odor - ethereal odor
pH: Not available.
Vapor Pressure: 160 mm Hg @ 20 deg C
Vapor Density: 4.12 (Air=1)
Evaporation Rate:11.6 (Butyl acetate=1)
Viscosity: 0.58 cps @ 20 deg C
Boiling Point: 60.5-61.5 deg C
Freezing/Melting Point:-63 deg C
Decomposition Temperature: Not available.
Solubility: Slightly soluble.
Specific Gravity/Density:1.492 (Water=1)
Molecular Formula:CHCl3
Molecular Weight:119.366

Section 10 - Stability and Reactivity

Chemical Stability: Stable at room temperature in closed containers under normal storage and handling conditions. Light sensitive.

Conditions to Avoid: High temperatures, incompatible materials, light.

Incompatibilities with Other Materials: Strong oxidizing agents, aluminum, fluorine, magnesium, sodium potassium, lithium, caustics (e.g. ammonia, ammonium hydroxide, calcium hydroxide, potassium hydroxide, sodium hydroxide), dinitrogen tetraoxide, sodium + methanol, potassium-tert-butoxide, chemically active metals, Attacks some forms of plastics, rubbers, and coatings., nitrogen tetroxide, acetone + alkali, disilane, perchloric acid + phosphorus pentoxide, sodium methylate, triisopropylphosphine, sodium methoxide + methanol.

Hazardous Decomposition Products: Hydrogen chloride, carbon monoxide, carbon dioxide, chlorine, phosgene gas.

Hazardous Polymerization: Will not occur.

Section 11 - Toxicological Information

RTECS#:
CAS# 67-66-3: FS9100000
CAS# 25377-72-4 unlisted.
LD50/LC50:
CAS# 67-66-3:
Draize test, rabbit, eye: 148 mg;
Draize test, rabbit, eye: 20 mg/24H Moderate;
Draize test, rabbit, skin: 500 mg/24H Mild;
Inhalation, rat: LC50 = 47702 mg/m3/4H;
Oral, mouse: LD50 = 36 mg/kg;
Oral, rat: LD50 = 695 mg/kg;
Skin, rabbit: LD50 = >20 gm/kg;

CAS# 25377-72-4:

Carcinogenicity:
CAS# 67-66-3:
ACGIH: A3 - Animal Carcinogen
California: carcinogen; initial date 10/1/87
NIOSH: potential occupational carcinogen
NTP: Suspect carcinogen
OSHA: Possible Select carcinogen
IARC: Group 2B carcinogen CAS# 25377-72-4: Not listed by ACGIH, IARC, NIOSH, NTP, or OSHA.
Epidemiology: Oral, rat: TDLo = 13832 mg/kg/2Y-C (Tumorigenic - Carcinogen ic by RTECS criteria - Blood - leukemia).; Oral, mouse: TDLo = 127 gm/kg/92W-I (Tumorigenic - Carcinogenic by RTECS criteria - Liver - tumors).; Oral, rat: TD = 98 gm/kg/78W-I (Tumorigenic - neoplastic by RTECS criteria - Kidney, Ureter, Bladder - Kidney tumors and Endocrine - thyroid tumors).; Oral, mouse: TD = 18 gm/kg/17W-I (Tumorigenic - neoplastic by RTECS criteria - Liver - tumor s).;
Teratogenicity: Oral, rat: TDL0 = 1260 mg/kg (female 6-15 day(s) after conception) Effects on Embryo or Fetus - fetotoxicity (except death, e.g., stunted fetus) Specific Developmental Abnormalities - musculoskeletal system.; Inhalation, rat: TCLo = 100 ppm/7H (female 6-15 day(s) after conception) Specific Developmental Abnormalities - gastrointestinal system and homeostasis.; Inhalation, mouse: TCLo = 100 ppm/7H (female 8-15 day(s) after conception) Specific Developmental Abnormalities - craniofacial (including nose and tongue).
Reproductive Effects: Inhalation, rat: TCLo = 30 ppm/7H (female 6-15 day(s) after conception) Fertility - other measures of fertility.; Inhalation, rat: TCLo = 300 ppm/7H (female 6-15 day(s) after conception) Fertility - female fertility index (e.g. # females pregnant per # sperm positive females; # females pregnant per # females mated) and post-implantation mortality (e.g. dead and/or resorbed implants per total number of implants).
Neurotoxicity: No information available.
Mutagenicity: DNA Inhibition: Human, HeLa cell = 19 mmol/L.; Sister Chromatid Exchange: Human, Lymphocyte = 10 mmol/L.; Micronucleus Test: Oral, rat = 4 mmol/kg.; Unscheduled DNA Synthesis: Oral, rat = 1 gm/kg.; Sister Chromatid Exchange: Hamster, Embryo = 100 umol/L.
Other Studies: Open irritation test: Administration onto the skin (rabbit) 10 mg/24H (Mild). Standard Draize Test: Administratio n onto the skin (rabbit) = 500 mg/24H (Mild). Standard D raize Test: Administration into the eye (rabbit) = 20 mg /24H (Moderate).

Section 12 - Ecological Information

Ecotoxicity: Fish: Channel catfish: LC50 = 75 ppm; 96 Hr; Unspecified Rainbow trout: LC50 = 43.8 mg/L; 96 Hr; Static bioassay Fathead Minnow: LC50 = 129.0 mg/L; 96 Hr; Static bioassay (pH = 7.6-8.3) Bluegill/Sunfish: LC50 = 100.0 mg/L; 96 Hr; Static bioassay flea Daphnia: EC50 = 28.9 mg/L; 48 Hr; Static bioassay The majority of the environmental releases from industrial uses are to the atmosphere; releases to water and land will be primarily lost by evaporation and will end up in the atmosphere. Release to the atmosphere may be transported long distances and will photodegrade with a half-life of a few months. Spills and other releases on land will also leach into the groundwater where it will reside for long periods of time.

Environmental: Chloroform will not be expected to bioconcentrate into the food chain but contamination of food is likely due to its use as an extractant and its presence in drinking water.
Physical: No information available.
Other: No information available.

Section 13 - Disposal Considerations

Chemical waste generators must determine whether a discarded chemical is classified as a hazardous waste. US EPA guidelines for the classification determination are listed in 40 CFR Parts 261.3. Additionally, waste generators must consult state and local hazardous waste regulations to ensure complete and accurate classification.
RCRA P-Series: None listed.
RCRA U-Series: CAS# 67-66-3: waste number U044.

Section 14 - Transport Information

	US DOT	IATA	RID/ADR	IMO	Canada TDG
Shipping Name:	CHLOROFORM				CHLOROFORM
Hazard Class:	6.1				6.1(9.2)
UN Number:	UN1888				UN1888
Packing Group:	III				II

Section 15 - Regulatory Information

US FEDERAL

TSCA
CAS# 67-66-3 is listed on the TSCA inventory.
CAS# 25377-72-4 is listed on the TSCA inventory.
Health & Safety Reporting List
CAS# 67-66-3: Effective Date: 6/1/87; Sunset Date: 6/1/97
Chemical Test Rules
None of the chemicals in this product are under a Chemical Test Rule.
Section 12b
None of the chemicals are listed under TSCA Section 12b.
TSCA Significant New Use Rule
None of the chemicals in this material have a SNUR under TSCA.
SARA

CERCLA Hazardous Substances and corresponding RQs
CAS# 67-66-3: 10 lb final RQ; 4.54 kg final RQ
SARA Section 302 Extremely Hazardous Substances
CAS# 67-66-3: 10,000 lb TPQ
SARA Codes
CAS # 67-66-3: acute, chronic. CAS # 25377-72-4: acute, flammable.
Section 313
This material contains Chloroform (CAS# 67-66-3, 100%),which is subject to the reporting requirements of Section 313 of SARA Title III and 40 CFR Part 373.
Clean Air Act:
CAS# 67-66-3 is listed as a hazardous air pollutant (HAP). This material does not contain any Class 1 Ozone depletors. This material does not contain any Class 2 Ozone depletors.
Clean Water Act:
CAS# 67-66-3 is listed as a Hazardous Substance under the CWA. CAS# 67-66-3 is listed as a Priority Pollutant under the Clean Water Act. CAS# 67-66-3 is listed as a Toxic Pollutant under the Clean Water Act.
OSHA:
None of the chemicals in this product are considered highly hazardous by OSHA.
STATE

CAS# 67-66-3 can be found on the following state right to know lists: California, New Jersey, Pennsylvania, Minnesota, Massachusetts.
CAS# 25377-72-4 can be found on the following state right to know lists: New Jersey.
The following statement(s) is(are) made in order to comply with the California Safe Drinking Water Act: WARNING: This product contains Chloroform, a chemical known to the state of California to cause cancer. California No Significant Risk Level: CAS# 67-66-3: 20 ug/day NSRL (oral); 40 ug/day NSRL (inhalation)

European/International Regulations

European Labeling in Accordance with EC Directives
Hazard Symbols:
XN
Risk Phrases:
R 22 Harmful if swallowed.
R 38 Irritating to skin.
R 40 Limited evidence of a carcinogenic effect.
R 48/20/22 Harmful : danger of serious damage to health by prolonged exposure through inhalation and if swallowed.

Safety Phrases:
S 36/37 Wear suitable protective clothing and gloves.

WGK (Water Danger/Protection)
CAS# 67-66-3: 3
CAS# 25377-72-4: No information available.
Canada - DSL/NDSL
CAS# 67-66-3 is listed on Canada's DSL List.
CAS# 25377-72-4 is listed on Canada's DSL List.
Canada - WHMIS
This product has a WHMIS classification of D2A, D1B.
Canadian Ingredient Disclosure List
CAS# 67-66-3 is listed on the Canadian Ingredient Disclosure List.
Exposure Limits
CAS# 67-66-3: OEL-ARAB Republic of Egypt:TWA 10 ppm (50 mg/m3) OEL-AUSTRALIA:TWA 10 ppm (50 mg/m3);Carcinogen OEL-AUSTRIA:TWA 10 ppm (50 mg/m3) OEL-BELGIUM:TWA 10 ppm (49 mg/m3);Carcinogen JAN9 OEL-CZECHO SLOVAKIA:TWA 10 mg/m3;STEL 20 mg/m3 OEL-DENMARK:TWA 2 ppm (10 mg/m3); Carcinogen OEL-FINLAND:TWA 10 ppm (50 mg/m3);STEL 20 ppm;Skin;CAR OE L-FRANCE:TWA 5 ppm (25 mg/m3);STEL 50 ppm (250 mg/m3);CAR OEL-GERMANY :TWA 10 ppm (50 mg/m3);Carcinogen JAN9 OEL-HUNGARY:STEL 10 mg/m3 OEL -INDIA:TWA 10 ppm (50 mg/m3);Carcinogen OEL-JAPAN:TWA 50 ppm (240 mg/ m3);Carcinogen OEL-THE NETHERLANDS:TWA 10 ppm (50 mg/m3) OEL-THE PHI LIPPINES:TWA 50 ppm (240 mg/m3) OEL-POLAND:TWA 50 mg/m3 OEL-RUSSIA:T WA 50 ppm OEL-SWEDEN:TWA 2 ppm (10 mg/m3);STEL 5 ppm (25 mg/m3);CAR OEL-SWITZERLAND:TWA 10 ppm (50 mg/m3);STEL 20 ppm (100 mg/m3) OEL-THA ILAND:TWA 50 ppm (240 mg/m3) OEL-TURKEY:TWA 50 ppm (240 mg/m3) OEL-U NITED KINGDOM:TWA 2 ppm (9.9 mg/m3);Skin OEL IN BULGARIA, COLOMBIA, J ORDAN, KOREA check ACGIH TLV OEL IN NEW ZEALAND, SINGAPORE, VIETNAM c heck ACGIH
TLV[illegible]

Section 16 - Additional Information

MSDS Creation Date: 6/09/1999
Revision #7 Date: 9/11/2002

The information above is believed to be accurate and represents the best information currently available to us. However, we make no warranty of merchantability or any other warranty, express or implied, with respect to such information, and we assume no liability resulting from its use. Users should make their own investigations to determine the suitability of the information for their particular purposes. In no event shall Fisher be liable for any claims, losses, or damages of any third party or for lost profits or any special, indirect, incidental, consequential or exemplary damages, howsoever

arising, even if Fisher has been advised of the possibility of such damages.

APPENDIX G

(Not Included in this Draft)

APPENDIX H

(Not Included in this Draft)

APPENDIX I

(Not Included in this Draft)

APPENDIX J

(Not Included in this Draft)

APPENDIX K

Voluntary Remediation Program Community Relations Plan
1352 North Illinois Street, LLP
1352 North Illinois Street, Indianapolis, IN
VRP # 6090502

In accordance with VRP guidance (as stated in IC 13-25-5-7) a Community Relations Plan has been prepared for the above referenced project. The following summarizes the basic components requested by the IDEM in the non-rule policy document "*Voluntary Remediation Program Community Relations Plan*", adopted April 20, 2001.

I. Identify all property owners and property occupants, which include property owners or occupants affected or likely to be affected by the contamination that is the subject of the proposed Voluntary Remediation Project and all owners or occupants of adjacent or closely proximate land.

Address	Property Owner	Identified Occupants
1301 N. Capitol Ave.	South Central Leasing	South Central Company
1327 N. Capitol Ave.	Kathryn Cue & Michael Lennington	Yellow Rose Carriages
1331 N. Capitol Ave.	Wesley & Helen Kidwell	None Identified
1341 N. Capitol Ave.	Circle City Land, LLC	Tip Top Tavern
1302 N. Illinois St.	Mahrdt Properties, Inc.	Fastenal
1352 N. Illinois St.	1352 North Illinois Street, LLP	None Identified

II. Identify all known or registered neighborhood organizations serving the location of the Voluntary Remediation Project, if any.

Historic Landmarks Foundation of Indiana
Indianapolis Downtown, Inc.
Indianapolis Neighborhood Resource Center
Marion County Alliance of Neighborhood Associations
Midtown Economic Development and Industrial Corporation
Near North Community Development Corporation

III. Identify all known or reasonably apparent sensitive community institutions within two (2) miles, including, but not limited to schools, health care facilities, child care facilities, senior citizen residential or care facilities and the administrative office or owner of parks and playgrounds.

Community Institution	Mailing Address	Distance from Site (miles)
Abc Preschool	602 E. Michigan St.	1.0
Angela's Infantcare Daycare	3047 N. Capitol Ave.	1.8
Angelic Child Care Home	3030 N. Capitol Ave.	1.8
Arsenal Technical High School 716	1500 E. Michigan St.	1.6
Charity Dye School 27	545 E. 19th St.	0.7

Community Institution	Mailing Address	Distance from Site (miles)
Child Care Answers	615 N. Alabama St. # 430	0.8
Clarian Health Partners	1701 N. Senate Blvd.	0.4
Crispus Attucks Magnet School	1140 Dr. Martin Luther King St.	0.5
Day Nursery	855 N. East St.	0.7
Day Nursery	575 N. Pennsylvania St. # 170	0.7
Day Nursery	2140 Boulevard Pl.	0.8
Day Nursery	615 N. Alabama St. # 300	0.8
Day Nursery	100 N. Senate Ave. # N150	1.1
Edmondson Latasha	2018 Koehne St.	1.7
Elder W. Diggs School 42	1002 W. 25th St.	1.6
Family Development Svc	1531 Indiana Ave.	0.9
Frances W. Parker School 356	2353 Columbia Ave.	1.6
Fuzzie Bear Child Care	2413 N. Meridian St. # L	1.1
Fuzzie Bear Child Care	168 W. 9th St.	0.4
George W. Carver School 87	2411 Indianapolis Ave.	1.2
God's Little Wonders Childcare	2951 N. Talbott St.	1.7
H. L. Harshman Middle School 501	1501 E. 10th St.	1.4
Herron High School	110 E. 16th St.	0.3
Indiana University Hospital	550 University Blvd.	1.1
Indianapolis Department of Parks and Recreation	200 E. Washington St. #2301	1.3
Indianapolis Metropolitan High School	1635 W. Michigan St.	1.8
Indianapolis Public School 63	1163 N. Belmont Ave.	2.0
Indianapolis Special Education	120 E. Walnut St.	0.6
Interplay Connecting Child Cr	1644 Dr. Andrew J. Brown Ave.	1.3
John H Boner Community Center	2236 E. 10th St.	1.9
Kathy's Loving Care	3045 N. Pennsylvania St.	1.8
Kid Zone	1531 E. Ohio St.	1.8
Kindred Hospital Indianapolis	1700 W. 10th St.	1.8
Little Dove Day Care	2327 E. 10th St.	2.0
Little Red Schoolhouse	2131 E. 10th St.	1.9
Lolliepop Daycare	1418 E. 10th St.	1.3
Luises's Love Child Care Center	1030 W. 16th St.	1.1
Mainstreet Senior LLC	2926 N. Capitol Ave.	1.6
Nette Daycare	2150 Sugar Grove Ave.	1.6
New Beginnings High School	1840 N. Meridian St.	0.4
Pacers Academy School 495	39 Jackson Pl. # 500	1.5
Riley Hospital for Children	702 Barnhill Dr.	1.1
Riverside School 44	2033 Sugar Grove Ave.	1.5
Roudebush V.A. Medical Center	1481 W. 10th St.	1.6
Senior Citizens Center	708 E. Michigan St.	1.1
Shalom Day Care Center	401 N. Delaware St.	0.9
Sonia's Daycare	2021 N. Harding St.	1.6
Sunrise Christian Academy Day	948 W. 30th St.	2.0
Theodore Potter School 74	1601 E. 10th St.	1.5
Washington Irving School 14	1250 E. Market St.	1.7
White River State Park	801 W. Washington St.	1.5
Woodruff Place Child Care	1739 E. Michigan St.	1.7

IV. Include a sample of a written notice to be sent to the property owners and property occupants, neighborhood organizations, and sensitive community institutions.

This notice is being provided to inform you of the presence of a site in your neighborhood that has been accepted into IDEM's Voluntary Remediation Program. This notice is a requirement of a Community Relations Plan, which has been developed by the Applicant and is a component of the Remediation Work Plan that is available for review at the repository listed below. The Community Relations Plan includes provisions for notifying all neighborhood property owners and occupants, neighborhood organizations and other local entities. In addition, the Community Relations Plan may require the applicant to post an informational sign at the subject property. For additional information about the Community Relations Plan and the Remediation Work Plan please review the documents in the repository or contact Ms. Carmen Anderson at (317) 234-5344 or (800) 451-6027.

Remediation activities to be performed at the site include the operation of a soil vapor extraction system to treat impacted soil.

A public comment period concerning site remediation activities is scheduled to occur. Concerned parties are encouraged to contact either Ms. Carmen Anderson or Mr. Jason Flagg with comments.

Public Repositories
Indianapolis Central Library
40 East Saint Clair Street
Indianapolis, IN 46204

Project Managers
Jason B. Flagg, EIT
Troy Risk, Inc.
7466 Shadeland Station Way
Indianapolis, IN 46256
jflagg@troyrisk.com
(317) 570-6730

Ms. Carmen Anderson
Indiana Department of Environmental Management
Indiana Government Center-North
100 N. Senate Avenue
Indianapolis, IN 46204
(317) 234-5344
(800) 451-6027 – toll free

V. Provide the name(s) and mailing address(es) of all affected local governmental units with jurisdiction within one (1) mile of the property affected by the proposed Remediation Work Plan.

Indianapolis Police Department
25 West 9th Street
Indianapolis, IN 46204
(317) 327-6500

Indianapolis Fire Department
555 North New Jersey Street
Indianapolis, IN 46204
(317) 327-6053 – Office

VI. Provide the name(s) and mailing address(es) of the newspaper(s) or other appropriate circulars in which notice of the public comment period will be published.

The Indianapolis Star
P.O. Box 145
Indianapolis, IN 46206-0145

VII. Identify the location of the public library and other public repositories in which a copy of the proposed Remediation Work Plan will be placed. The proposed Remediation Work Plan must be placed in the public library closest to the site and in the county or counties affected by the project. If more than one repository is selected, the participant shall provide one additional copy of the proposed Voluntary Remediation Work Plan for each additional repository.

Indianapolis Central Library
40 East Saint Clair Street
Indianapolis, IN 46204

VIII. In, addition, VRP Participants shall post a sign that:

- *a. identifies the location as a VRP cleanup site;*
- *b. gives the IDEM VRP site number, the VRP phone number and the VRP web site address;*
- *c. shall meet the following criteria;*
  - *i. be visible/readable from 20 feet;*
  - *ii. be in English and the language predominantly used in the neighborhood if other than English; and*
  - *iii. place one sign per site access point; and*
- *d. shall be posted starting with the end of the public comment period for the Remediation Work Plan, before any work begins and remain posted until the Covenant Not To Sue has been issued.*

Signage Text

The property at 1352 North Illinois Street is currently undergoing cleanup under the supervision of the IDEM's Voluntary Remediation Program (VRP # 6090502). Information concerning the Voluntary Remediation Program can be obtained at www.in.gov/idem/4127.htm/ or by calling (317) 234-5344 or (800) 451-6027.

APPENDIX L

(Not Included in this Draft)