

# Health Consultation

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KOSHKONONG SCHOOL WELL SITE  
KOSHKONONG, OREGON COUNTY, MISSOURI

EPA FACILITY ID: MON000705885

**Prepared by the  
Missouri Department of Health and Senior Services**

SEPTEMBER 25, 2009

Prepared under a Cooperative Agreement with the  
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Agency for Toxic Substances and Disease Registry  
Division of Health Assessment and Consultation  
Atlanta, Georgia 30333

## **Health Consultation: A Note of Explanation**

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

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

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HEALTH CONSULTATION

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Prepared By:

Missouri Department of Health and Senior Services  
Division of Community and Public Health  
Section for Disease Control and Environmental Epidemiology  
Bureau of Environmental Epidemiology  
Under a Cooperative Agreement with the  
U.S. Department of Health and Human Services  
Agency for Toxic Substances and Disease Registry (ATSDR)

**SUMMARY**

**INTRODUCTION**

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A top priority for the Missouri Department of Health and Senior Services (DHSS), in cooperation with the federal Agency for Toxic Substances and Disease Registry (ATSDR) is to protect the health of faculty and students who work and attend the Koshkonong School. This health consultation evaluates the public health impact on students and faculty from drinking well water in the past with a low but increasing level of trichloroethylene (TCE) over a period of years at the Koshkonong School.

The Missouri Department of Natural Resources (MDNR) requested DHSS to follow up on TCE contamination detected in the Koshkonong School Well and their investigation of private wells surrounding the site. MDNR found no TCE contamination in any of the private wells they were able to test. The school is now connected to the Koshkonong public water system.

DHSS evaluated the data reported in the MDNR Site Inspection Report and MDNR’s Drinking Water Branch on-line sample results. DHSS also conducted a site visit to the Koshkonong School and adjoining area to further investigate the situation and possible exposures.

**CONCLUSIONS**

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DHSS/ATSDR reached two conclusions in the health consultation.

Conclusion 1

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DHSS concludes that exposure to trichloroethylene (TCE) contaminated drinking water at the Koshkonong School in the past is not expected to have harmed people’s health. Exposure to TCE contamination at the school in the future is not expected unless the Koshkonong City water system becomes contaminated.

Basis for Decision

Although students and faculty of the Koshkonong School were exposed to low levels of TCE by ingestion, inhalation, and dermal contact over several years, exposure calculations were below levels of health concern for non-cancerous and cancerous health effects.

Conclusion 2

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DHSS concludes that there is the possibility that future exposures to TCE could occur at different well locations in the area.

Basis for Decision

Since the Koshkonong School well is no longer pumping water from the Ozark aquifer, the possibility exists that the TCE could move to other wells in the area.

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Next steps                      DHSS will consider future monitoring of groundwater wells in the area to ensure that the TCE contamination has not moved to other wells and others are not being exposed.

## **STATEMENT OF ISSUES AND BACKGROUND**

### **Statement of Issues**

The Missouri Department of Health and Senior Services (DHSS) prepared this health consultation under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). This health consultation is a follow-up to the Missouri Department of Natural Resources (MDNR) Site Inspection Report for the Koshkonong School Well Site and their referral of the site to DHSS for further evaluation and monitoring. This health consultation will evaluate past exposures to TCE in groundwater for students and faculty and consider future actions to avoid additional exposures in the area.

### **Background**

The Koshkonong School Well Site is located on the Koshkonong School property on the north end of the small town (population 205) at 398 School Street, Koshkonong, Oregon County, Missouri (1). The number of students and faculty at the school is approximately 353 (2). The school used the approximately 400 feet deep well for their source of potable water until October 2006 when the pump stopped working and the school connected to the city water system. The school grounds consist of approximately 12 acres and include a pre-school, elementary school, high school, maintenance buildings, green houses, and special education and Future Farmers of America buildings (See Figure 1) (1).

Levels of trichloroethylene (TCE) had been detected in the school well since at least 1996. The level of TCE in the well has gradually increased in concentration over the years. In February 2006, the detected level was 6.01 parts per billion (ppb), which is above Environmental Protection Agency's (EPA's) Maximum Contaminant Level (MCL) of 5 ppb. TCE levels decreased slightly in the May and August 2006 sampling at 4.25 ppb and 5.32 ppb respectively (3). No other contaminants were detected in the well. When the pump quit working in October 2006, the school decided to connect to the Koshkonong public water system instead of replacing the pump. The Koshkonong city well (1,100 feet deep) is located on the southwest corner of the property and an emergency city well (650 feet deep) is located approximately 0.3 miles south of the Koshkonong School well. The Koshkonong public water system has not shown any TCE contamination (1).

On September 19, 2006, MDNR's Public Drinking Water Program referred the site to the MDNR Hazardous Waste Program (HWP) Superfund Section because of the elevated levels of TCE. The MDNR HWP conducted an investigation of the school's property in June 2007 and found no obvious spills or source areas of TCE contamination. To determine if the TCE contamination found in the school's well may also be present in the area's groundwater, MDNR personnel sampled available domestic wells in the area (1).

MDNR's database of wells installed after 1987 when the registry of drilled wells started being required shows 12 domestic wells within one mile of the site. Wells drilled before 1987 may not be listed on the database. In their investigation, MDNR was able to sample seven of those registered wells and two additional wells. Since MDNR determined that the expected direction of groundwater movement is to the southeast, the majority of the wells sampled are up gradient (north) of the school well. None of the wells had detectable levels of TCE; although, one well had a detectable level of 1,2,4-trimethylbenzene at 2.51 ppb. Since no MCL exists for 1,2,4-trimethylbenzene, MDNR compared the detected level to the Missouri Risk-based Corrective Action Risk-based Target Level of 7.06 ppb. MDNR reported the water was more turbid (cloudy) than others when tested and reportedly not used for consumption (1).

All of the wells within a four mile radius of the site draw water from the Ozark Aquifer with well depths ranging from 260 feet deep to 1,100 feet deep. The area is also highly karst (an area where erosion has produced fissures, sinkholes, underground streams, and caverns) with numerous sinkholes. MDNR reported that topographic maps for the area illustrate roughly 470 sinkholes within four miles of the site with numerous sinkholes around the school property, in town, and within a mile from the site (1).

DHSS personnel conducted a site visit of the Koshkonong School site on March 11, 2009 as a follow-up of the MDNR site investigation and as requested by MDNR. A visual tour was made of the town and area where numerous sinkholes were noticed. A visual tour was also made of the school area where it was noted that the Burlington Northern railroad track was located adjacent to the school property on the west with a large sinkhole just west of the tracks. DHSS personnel then met with the Koshkonong School maintenance supervisor who showed us the well house and explained the events that lead up to connecting to public water after the well pump failed. Through visual inspection, it was confirmed that the school well was no longer providing water to the school.

## **DISCUSSION**

### **Pathways Analysis**

This section addresses the pathways by which students and faculty of the Koshkonong School may have been exposed to the TCE contaminated groundwater.

When a chemical is released into the environment, the release does not always lead to exposure. Exposure only occurs when a chemical comes into contact with and enters the body. To

determine the extent that students and faculty at the Koshkonong School were exposed to contaminants at this site, DHSS conducted an analysis of exposure pathways. For a chemical to pose a health risk, a completed exposure pathway must exist. An exposure pathway consists of five elements including a source of contamination, transport through an environmental medium, a point of exposure, a route of human exposure, and an exposed population. Completed exposure pathways require that all five of the elements of exposure exist. An exposure pathway can be eliminated if at least one of the five elements is missing and will never be present. Potential exposure pathways, however, have at least one of the five elements missing or uncertain, but could exist. Completed and potential exposure pathways could have occurred in the past, could be occurring presently, or could occur in the future.

### **Completed Exposure Pathways**

#### **Past:**

The five elements of a completed exposure pathway at the Koshkonong School are explained below:

1. **Contaminant source** - location not determined.
2. **Environmental medium and transport** - TCE contaminated groundwater.
3. **Point of exposure** – Koshkonong School well.
4. **Route of exposure** - ingestion, inhalation, and dermal contact.
5. **Exposed population** – Koshkonong students and faculty.

Students and faculty at the Koshkonong School site were exposed to TCE through ingestion, inhalation, and dermal contact. Ingestion exposure would occur by drinking the water or eating food prepared with contaminated water. People can also be exposed to TCE through inhalation and dermal contact while showering, bathing, and washing clothes and dishes as well as other activities. Inhalation exposure would be greater in areas that have limited air circulation.

Completed exposure pathways to TCE contaminated water existed for the users of the Koshkonong School well from the approximate time it was detected in 1996 until the well was disconnected in 2006. The level of TCE contamination increased over time, until it exceeded EPA's MCL in February 2006. The pathway to TCE contaminated water did not end until the school well pump became inoperable and the school connected to public water in October 2006.

#### **Present:**

The point of exposure has been removed so no completed exposure pathways are expected unless the Koshkonong public water system becomes contaminated.

### **Future Potential Exposure Pathways**

None, unless the Koshkonong public water system or other private wells become contaminated.

## TOXICOLOGICAL EVALUATION

### Introduction

This section will discuss the health effects of exposure to specific contaminants. A discussion of non-cancerous health effects and the possibility that TCE exposure might cause cancer is evaluated in this section. ATSDR has developed Comparison Values that are media-specific levels that when exceeded require further evaluation. ATSDR Comparison Values include Minimal Risk Level (MRL) and Environmental Media Evaluation Guides (EMEGs) for contaminants commonly found at hazardous waste sites. The MRL/EMEG is an estimate of daily human exposure. Exposure to a contaminant level below the MRL/EMEG is not expected to cause adverse non-cancer health effects. Levels above an MRL/EMEG do not mean that health effects will definitely occur; rather, it calls for more investigation into whether health effects may or may not occur. MRLs/EMEGs are developed for each route of exposure, such as ingestion and inhalation, and for the length of exposure, such as acute (less than 14 days), intermediate (15 to 364 days), and chronic (greater than 365 days). This toxicological evaluation section will discuss the possible adverse health effects that water users might expect from short and long-term exposure to low levels of TCE contamination in their drinking water. The possibility of TCE causing cancer and the theoretical risk of exposure to TCE causing additional cancers is discussed under the cancer section.

TCE was first detected in the Koshkonong School well in approximately early 1996 and the level has gradually increased over the years (See Table 1). In February 2006, the level of TCE exceeded its MCL at a maximum of 6.01 ppb, then dropped to 4.25 ppb at the next quarterly sampling in May 2006, and then above the MCL again at 5.32 ppb at the next quarterly sampling in August 2006. Exposure to TCE contaminated drinking water would have occurred to the students and faculty of the Koshkonong School during those periods, so calculations were made to determine the health risks during the time the level of TCE exceeded its MCL and during the long-term exposure since TCE was detected.

### Trichloroethylene

TCE is a volatile organic compound that evaporates easily in air, but stays in soil and water without much decomposition. Since it is heavier than water it can pass easily through the soil into the groundwater. It is used mainly as a solvent to remove grease from metal parts, but can also be found in household products such as wood stains, varnishes, lubricants, adhesives, typewriter correction fluids, paint removers, and cleaners. Most people can begin to smell TCE in air at around 100,000 ppb. Once it enters the body it is changed to other chemicals that have been shown to be toxic to animals and probably toxic to humans (4).

The major target of TCE is the nervous system where exposure to large amounts may cause dizziness, sleepiness, headaches and even unconsciousness (TCE was once used as an anesthetic for surgery at around 1,000,000 ppb). At higher levels and possibly long-term low-level exposure, liver and kidney damage and changes in heartbeat can occur (4).

When TCE is inhaled, approximately half of the amount of TCE breathed in is exhaled and the other half enters the bloodstream. After TCE contaminated water is ingested, most of the TCE is absorbed into the bloodstream. Much of the TCE in the bloodstream will also be removed through the lungs by exhalation. Once in the blood, the liver breaks down TCE into other chemicals (dichloroacetic acid, trichloroacetic acid, chloral hydrate, and 2-chloroacetaldehyde). The majority of these chemicals will be excreted in urine within a day (4). For more on the health effects of TCE, see Appendix B, Discussion of Trichloroethylene (TCE) Studies.

### **Exposure (non-cancerous)**

People can be exposed through ingestion by drinking contaminated water or eating food prepared with contaminated water. People can also be exposed to air borne TCE through inhalation. School activities using TCE contaminated water such as showering, washing clothes and dishes, and other similar activities can increase TCE levels in the air. Exposure through dermal contact can take place during showering, or other activities that put the skin in contact with TCE-contaminated water.

### **Ingestion Exposures**

Ingestion exposure doses were calculated using the worse-case exposure scenario (child) for past exposures of the short-term exposure that exceeded the MCL and the long-term exposure. Calculations were made using the maximum level (6.01 ppb) detected for the short-term exposure and the average level (3.6 ppb) of TCE for the long-term exposure.

#### **Calculated Ingestion Doses of TCE for a Child**

- Short-term exposure to maximum TCE level detected = **0.00043 mg/Kg/day**
- Long-term exposure to average TCE level detected = **0.000172 mg/Kg/day**

No Reference Dose is available at this time to calculate a hazard index for the ingestion of TCE contaminated drinking water. ATSDR's Acute (14 days or less) ingestion for TCE = 0.2 mg/Kg/day. ATSDR has not derived an intermediate (15-364 days) or a chronic (365 days or more) ingestion exposure MRL for TCE (MRL is ATSDR's minimal risk level). Studies of animals found a no-observed-adverse-effect-level (NOAEL) for rats and mice at around 1,000 mg/Kg/day with one study on rats having a NOAEL of 250 mg/Kg/day for chronic exposure. These numbers were for adverse effects on the body's different systems. Humans have not shown similar effects from exposure to TCE.

## Inhalation Exposure:

Inhalation exposure doses were calculated using the worse-case exposure scenario (child) for past exposures of the short-term exposure that exceeded the MCL and the long-term exposure. Calculations were made using the maximum level (6.01 ppb) detected for the short-term exposure and the average level (3.6 ppb) of TCE for the long-term exposure.

### Calculated Inhalation Doses of TCE for a Child

- Short-term exposure to maximum TCE level detected = **0.000626 mg/m<sup>3</sup>**
- Long-term exposure to average TCE level detected = **0.00025 mg/m<sup>3</sup>**

ATSDR has not developed a MRL/EMEG for chronic (greater than 364 days) exposure, but have developed a value of 0.50 mg/m<sup>3</sup> for intermediate exposure (15-364 days). Since the short-term exposure falls within the intermediate time period and is 1,000 times lower than the MRL/EMEG level, we do not expect any health concerns. Also, given that the long-term exposure is also 1,000 times lower than the intermediate MRL/EMEG, no health effects are expected.

## Dermal Exposure:

Dermal effects of exposure to TCE are usually the consequence of direct skin contact with concentrated solutions in occupational settings. Exposures to these concentrated solutions usually result in desiccation (drying) of the skin due to the defatting action of the solvent that may lead to dermatitis. Dermal contact with TCE at the levels present in the school well are magnitudes lower (ppb versus the possibility of pure product used in occupational settings), do not pose a health concern, and will not be discussed further.

## Cancer

The American Cancer Society estimates that in the United States, slightly less than half of all men and slightly more than one-third of all women will develop some form of cancer in their lifetime (5). To determine the theoretical cancer risk for adults exposed to hazardous chemicals, EPA has developed cancer unit risk factors (Oral Slope Factor). Cancer risks are calculated over a lifetime, estimated to be 70 years. DHSS calculated the cancer risk for the Koshkonong School Well site and the different exposure pathways associated with the site using the worst case scenario (using the highest level of the cancer slope factor range, the maximum level detected, and the maximum expected exposure time). Because of the conservative nature of these calculations, this approach provides a theoretical estimated risk of cancer. In actuality, the true or actual risk is unknown and could be as low as zero. The cancer calculations can be found in Table 2.

The ability of TCE to cause cancer from ingestion and inhalation exposure is presently under review by the EPA. TCE is classified by the International Agency for Research on Cancer as probably carcinogenic to humans (limited human evidence; sufficient evidence in animals).

EPA presently has a draft document that draws information from 16 new state-of-the-science papers on the health effects of exposure to TCE. After evaluation of these 16 new studies on TCE health risks is complete, some of the uncertainties may be cleared up or further studies may need to be done.

ATSDR has not calculated Cancer Risk Evaluation Guide (CREG) for TCE for ingestion or inhalation. Since no Comparison Value is available for comparison, this health consultation will use EPA's Target Risk Range of  $10^{-4}$  to  $10^{-6}$  as the level of concern.

### Calculated Levels of Cancer Risks

#### Ingestion Exposure:

- Short-term cancer risk from exposure to maximum TCE level detected:  $4 \times 10^{-8}$
- Long-term cancer risk from exposure to average TCE level:  $3.2 \times 10^{-7}$

#### Inhalation Exposure:

- Short-term cancer risk from exposure to maximum TCE level detected:  $8.86 \times 10^{-9}$
- Long-term cancer risk from exposure to average TCE level:  $7.1 \times 10^{-8}$

#### Total Exposure from TCE in Drinking Water (Ingestion and Inhalation Exposure)

Total cancer risk from Ingestion and Inhalation at maximum level (6.01 ppb) of TCE detected:

$$(4 \times 10^{-8}) + (0.886 \times 10^{-8}) = \underline{4.88 \times 10^{-8}}$$

Total cancer risk from Ingestion and Inhalation at average level (3.6 ppb) of TCE detected

$$(3.2 \times 10^{-7}) + (0.71 \times 10^{-7}) = \underline{3.91 \times 10^{-7}}$$

Considering that calculated levels for cancer and the total from ingestion and inhalation are below the lowest range ( $10^{-6}$ ) of EPA Target Risk Range, no elevated levels of cancer risk are expected.

Studies of humans exposed to TCE for chronic periods via the inhalation and dermal route in the workplace apparently do not experience an increased incidence of cancer, as indicated by numerous epidemiological studies. Ingestion exposure to TCE and cancer in humans from short-term low-level exposure is inconclusive, with a number of studies indicating an association and a number of studies not indicating an association.

### **Children and Other Sensitive Populations**

A sensitive population may exhibit a different or enhanced response to hazardous chemicals than will most persons exposed to the same level of hazardous chemicals in the environment. Reasons for sensitivity might include genetic makeup, age, gender, health and nutritional status, and exposure to other toxic substances. In general the elderly, with declining organ function, and the young, with immature and developing organs, are more vulnerable to toxic substances than healthy adults.

A number of studies have suggested or shown associations between TCE exposure and children's health effects, but these studies had limitations that question their validity. Developing fetuses are susceptible to the toxic effects of chemicals that can cross the placental barrier. Also, premature and newborn infants will be more vulnerable to TCE exposure than the general population because of their immature and developing organs. In general, several studies suggest, but do not conclude, that exposure to TCE may cause birth defects (heart defects, respiratory system defects, eye defects, neural tube defects, and oral cleft defects) or childhood leukemia in children who were exposed in utero (as a fetus). In some studies, other chemicals were present besides TCE. Children listed in ATSDR's National Exposure Registry for TCE were reported as having higher rates of hearing and speech impairment, but there are still questions regarding these reports (18). Because it is difficult to predict the amount of exposure that a developing fetus may be exposed to at this site, it is also difficult to predict what birth defects or disease, if any, may result from exposure at this site. However, considering the maximum concentration of TCE (6.01 ppb) that the faculty and students may have been exposed to for a short period of time, it is not expected that developing fetuses and/or children would be affected. Women, who believe they were exposed to TCE during pregnancy and are concerned about possible health impacts to their unborn child, should consult their personal physicians (4).

Other sensitive populations including those that consume alcohol or who are treated with disulfiram (a drug used to treat alcohol dependency) may be at greater risk of TCE poisoning. This occurs because ethanol and disulfiram can both inhibit the metabolism of TCE and cause it to accumulate in the bloodstream, increasing its effects on the nervous system. Also, those with compromised liver and kidney function may be at a higher risk from exposure to TCE or its metabolites. The liver serves as the primary site of TCE metabolism and the kidney as the major excretory organ for TCE metabolites. People who smoke may also increase their risk of toxic effects from TCE (4).

## **CONCLUSIONS**

DHSS/ATSDR reached two conclusions in this health consultation:

DHSS concludes that exposure to trichloroethylene (TCE) contaminated drinking water at the Koshkonong School in the past is not expected to have harmed people's health. Exposure to TCE contamination at the school in the future is not expected unless the Koshkonong City water system becomes contaminated.

DHSS concludes that there is the possibility that future exposures to TCE could occur at different well locations in the area.

## **RECOMMENDATIONS**

Follow up well sampling should be considered to determine if the TCE contamination in the Koshkonong School Well will move to other wells in the area.

## **PUBLIC HEALTH STATEMENT**

This Public Health Action Plan (PHAP) for the Koshkonong School Well site contains a description of actions to be taken by the Missouri Department of Health and Senior Services (DHSS), the Agency for Toxic Substances and Disease Registry (ATSDR) and other stakeholders. The purpose of the PHAP is to ensure that this public health assessment not only identifies public health hazards, but provides an action plan to mitigate and prevent adverse human health effects resulting from past, present, and future exposures to hazardous substances at or near the site. Below is a list of commitments of public health actions to be implemented by DHSS, ATSDR, or other stakeholders at the site:

- DHSS/ATSDR will consider follow up sampling of wells sampled and determine if additional wells can be sampled to monitor the TCE contamination in the local groundwater.
- DHSS/ATSDR will address community health concerns and questions as they arise.

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### Appendix 1:

Figure 1:	Koshkonong School Well Site Location Map
Table 1:	Koshkonong R-III School Well Results
Table 2:	Calculations of Trichloroethylene Exposure

### Appendix 2: Discussion of Trichloroethylene (TCE) Studies

## CERTIFICATION

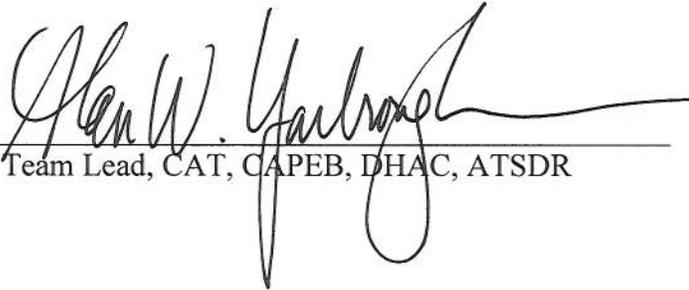
This Koshkonong School Well Site Health Consultation was prepared by the Missouri Department of Health and Senior Services under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It was completed in accordance with approved methodologies and procedures existing at the time the health consultation was initiated. Editorial review was completed by the Cooperative Agreement partner.



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Technical Project Officer, CAT, CAPEB, DHAC

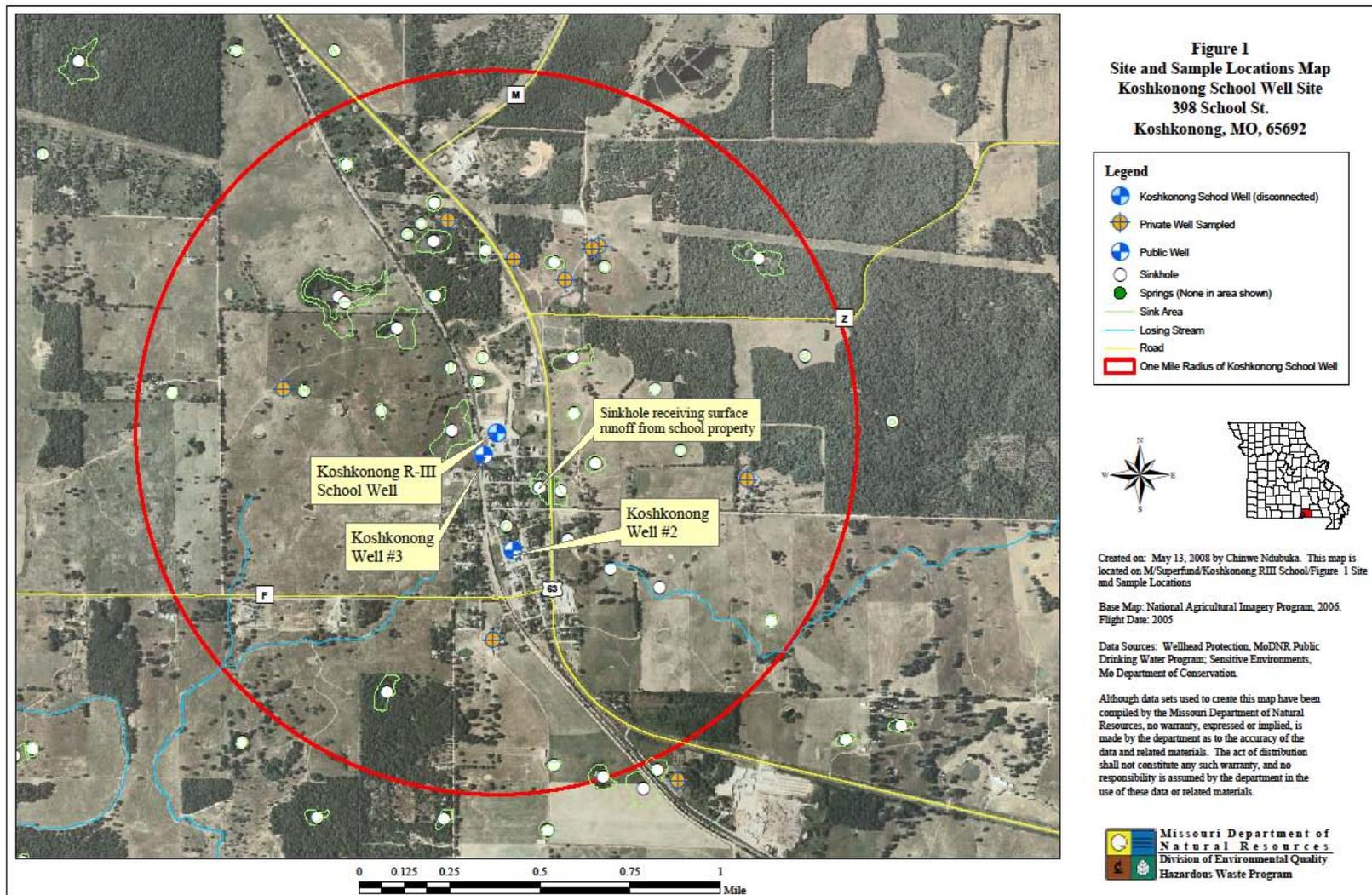
The Division of Health Assessment and Consultation (DHAC), has reviewed this health consultation and concurs with its findings.



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Team Lead, CAT, CAPEB, DHAC, ATSDR

**Figure 1**  
**Koshkonong School Well Site Location Map**  
 Koshkonong, Oregon County, Missouri



Source: Missouri Department of Natural Resources

**Table 1****KOSHKONONG R-III SCHOOL WELL RESULTS  
Koshkonong, Oregon County, Missouri****Sample Results 1996 through 2006**

Compiled February 11, 2009

<b>Date Sampled</b>	<b>Chemical</b>	<b>Level µg/L (ppb)</b>	<b>EPA's MCL µg/L (ppb)</b>
08/28/2006	Trichloroethylene	5.32	5
05/23/2006	Trichloroethylene	4.25	5
02/23/2006	Trichloroethylene	6.01	5
11/17/2005	Trichloroethylene	3.78	5
08/30/2005	Trichloroethylene	2.47	5
06/01/2005	Trichloroethylene	3.47	5
02/24/2005	Trichloroethylene	4.22	5
01/24/2005	Trichloroethylene	3.1	5
11/22/2004	Trichloroethylene	4.33	5
08/20/2004	Trichloroethylene	3.61	5
05/17/2004	Trichloroethylene	4.32	5
02/19/2004	Trichloroethylene	3.83	5
08/21/2003	Trichloroethylene	3.73	5
02/20/2003	Trichloroethylene	2.89	5
06/03/2002	Trichloroethylene	4.2	5
11/14/2001	Trichloroethylene	3.2	5
08/28/2001	Trichloroethylene	3.8	5
02/26/2001	Trichloroethylene	3.8	5
08/02/2000	Trichloroethylene	3.8	5
02/22/2000	Trichloroethylene	3.1	5
02/02/1999	Trichloroethylene	2.9	5
1998 - 1997	Trichloroethylene	Not Sampled/Reported	5
03/06/1996	Trichloroethylene	0.8	5
01/15/1996	Trichloroethylene	2.0	5
1996 - 2006	Pesticides	OK	
1996 -2006	Metals	OK	
1996 – 2006	Nitrates	OK	

**Source:** Missouri Department of Natural Resources, Drinking Water Branch – Non-TCR Results 1996 – 2006.

µg/L = micrograms per Liter = ppb = parts per billion

TCR = Total Coliform Rule

EPA's MCL = Environmental Protection Agency's Maximum Contaminant Level

**Table 2**

**Calculation of Trichloroethylene Exposure**

During the final time period that the Koshkonong School Well was in operation, levels of trichloroethylene (TCE) briefly exceeded the Environmental Protection Agency's Maximum Contaminant Level (MCL). Calculations have been made to determine if the exposure to TCE at the Koshkonong School will have a detrimental effect on students and faculty. Because of the range in ages of those exposed (children to adults), a worse-case exposure scenario will be used in the calculations. Worse-case exposure scenario will use the maximum detected and the average detected over the years and the most sensitive population exposed (children). Calculations were made using EPA's Risk Assessment Guidance for Superfund (RAGS).

**Past Exposures**

**Non-Cancerous**

**Ingestion Exposure for TCE in drinking water:**

$$\text{Ingestion Exposure Dose} = \frac{C \times IR \times EF \times ED}{BW \times AT}$$

Where:

C = contaminant concentration in mg/L (example – 6.01 ppb = 0.00601 ppm or mg/L)

IR = ingestion rate

EF = exposure factor (calculations consider complete absorption of contaminants)

ED = exposure duration in years

BW = body weight in Kilograms (example – 10 Kg is used for a child)

AT = averaging time in days

Dose is in milligrams/Kilograms/day or mg/Kg/day

**Child drinking water for Koshkonong School Well at maximum level of 0.00601 mg/L**

$$\text{Child Ingestion Exposure Dose} = \frac{0.00601 \text{ TCE mg/L} \times 1\text{L/day} \times 130 \text{ days/year} \times 1 \text{ year}}{10 \text{ Kg} \times 182 \text{ days}}$$

$$\text{Child Ingestion Exposure Dose} = \underline{\underline{0.00043 \text{ or } 4.3 \times 10^{-4} \text{ mg/Kg/day}}}$$

**Child drinking water for Koshkonong School Well at averaged level since TCE detected = 3.6 ppb or 0.0036 mg/L.**

$$\text{Child Ingestion Exposure Dose} = \frac{0.0036 \text{ TCE mg/L} \times 1\text{L/day} \times 174 \text{ days/year} \times 10 \text{ years}}{10 \text{ Kg} \times 3650 \text{ days}}$$

$$\text{Child Ingestion Exposure Dose} = \underline{\underline{0.000172 \text{ or } 1.72 \times 10^{-4} \text{ mg/Kg/day}}}$$

No Reference Dose is available at this time to calculate a hazard index for the ingestion of TCE contaminated drinking water. ATSDR's Acute (14 days or less) ingestion for TCE = 0.2 mg/Kg/day. ATSDR has not derived an intermediate (15-364 days) or a chronic (365 days or more) ingestion exposure MRL for TCE (MRL is ATSDR's minimal risk level). Studies of animals found a no-observed-adverse-effect-level (NOAEL) for rats and mice at around 1,000 mg/Kg/day with one study on rats having a NOAEL of 250 mg/Kg/day for chronic exposure. These numbers were for adverse effects on the body's different systems. Humans have not shown similar effects from exposure to TCE.

**Inhalation Exposure for TCE volatilization into air.**

$$\text{Child Inhalation Exposure Dose} = \frac{C_w \times K \times (ET/24 \text{ hours/day}) \times EF \times ED}{\text{Non-carcinogenic averaging time}}$$

Where:

C<sub>w</sub> = chemical concentration in water in mg/L (example 6.01 ppb = 0.00601 ppm or mg/L)

K = volatilization constant

ET = exposure time

EF = exposure factor (calculations consider complete absorption of contaminants)

ED = exposure duration in years

mg/m<sup>3</sup> = milligram per cubic meter of air

**Child Inhalation Exposure from Koshkonong School Well at Maximum level of 6.01 ppb**

$$\text{Child Inhalation Exposure Dose} = \frac{0.00601 \text{ mg/L} \times 0.5 \text{ L/m}^3 \times (7 \text{ hours/day}/24 \text{ hours day}) \times 1 \text{ year} \times 130 \text{ days/year}}{182 \text{ days}}$$

**Child Inhalation Exposure Dose = 0.000626 or 6.26 x 10<sup>-4</sup> mg/m<sup>3</sup>**

**Child Inhalation Exposure from Koshkonong School Well at Average level of 3.6 ppb**

$$\text{Child Inhalation Exposure Dose} = \frac{0.0036 \text{ mg/L} \times 0.5 \text{ L/m}^3 \times (7 \text{ hours}/24 \text{ hours day}) \times 174 \text{ days/year} \times 10 \text{ years}}{3650 \text{ days}}$$

**Child Inhalation Exposure Dose = 0.00025 or 2.5 x 10<sup>-4</sup> mg/m<sup>3</sup>**

## Cancer Calculations

Considering that trichloroethylene (TCE) is considered a probable human carcinogen or is under review as a carcinogen, we calculated the possible cancer risk from ingestion and inhalation exposure at the maximum level detected and the average level that was present in the Koshkonong School well since it was detected. The calculations assume a child's exposure since this would be the worse-case of exposure and is calculated for a 70 year period, considered to be a lifetime.

### Past Exposures Cancerous

#### Ingestion Exposure for TCE in drinking water:

$$\text{Ingestion Exposure Dose} = \frac{C \times IR \times EF \times ED}{BW \times AT}$$

Where:

C = contaminant concentration in mg/L (example – 6.01 ppb = 0.00601 ppm or mg/L)

IR = ingestion rate

EF = exposure factor (calculations consider complete absorption of contaminants)

ED = exposure duration in years

BW = body weight in Kilograms (example – 10 Kg is used for a child)

AT = averaging time in days

Dose is in milligrams/Kilograms/day or mg/Kg/day

#### Child drinking water for Koshkonong School Well at maximum level of 0.00601 mg/L

$$\begin{aligned} \text{Child Cancer Risk Ingestion Exposure Intake} = \\ \frac{0.00601 \text{ TCE mg/L} \times 1\text{L/day} \times 130 \text{ days/year} \times 1 \text{ year}}{10 \text{ Kg} \times 25550 \text{ days}} \end{aligned}$$

$$\text{Child Cancer Risk Ingestion Exposure Intake} = 0.00000306 \text{ mg/Kg/day}$$

#### Cancer Risk Factor from Ingestion of TCE in drinking water:

$$\text{Cancer Risk factor} = \text{Intake in mg/Kg/day} \times \text{slope factor (mg/Kg/day)}^{-1}$$

Cancer Risk at maximum detected level:

$$\text{Cancer Risk factor} = 0.00000306 \text{ mg/Kg/day} \times 1.3 \times 10^{-2} \text{ (mg/Kg/day)}^{-1} =$$

$$\text{Cancer Risk factor} = \underline{\underline{0.00000004 \text{ or } 4 \times 10^{-8}}}$$

#### Child drinking water for Koshkonong School Well at average level of 0.0036 mg/L

$$\begin{aligned} \text{Child Cancer Risk Ingestion Exposure Intake} = \\ \frac{0.0036 \text{ TCE mg/L} \times 1\text{L/day} \times 174 \text{ days/year} \times 10 \text{ year}}{10 \text{ Kg} \times 25550 \text{ days}} \end{aligned}$$

$$\text{Child Cancer Risk Ingestion Exposure Intake} = 0.0000245 \text{ mg/Kg/day}$$

Cancer Risk at average detected level:

$$\text{Cancer Risk factor} = 0.0000245 \text{ mg/Kg/day} \times 1.3 \times 10^{-2} (\text{mg/Kg/day})^{-1} =$$

$$\text{Cancer Risk factor} = \underline{\mathbf{0.00000032 \text{ or } 3.2 \times 10^{-7}}}$$

### **Inhalation Exposure from TCE in drinking water:**

Calculation at maximum TCE concentration (6.01 ppb or 0.00601 mg/L)

$$\text{Intake in mg/m}^3 = \frac{C_w \times K \times (\text{ET}/24 \text{ hours/day}) \times \text{EF} \times \text{ED}}{\text{Carcinogenic averaging time}}$$

Child Inhalation Exposure Dose =

$$\frac{0.00601 \text{ mg/L} \times 0.5 \text{ L/m}^3 \times (7 \text{ hours/day}/24 \text{ hours day}) \times 1 \text{ year} \times 130 \text{ days/year}}{25550 \text{ days}} =$$

$$\text{Child Inhalation Exposure Intake} = \mathbf{0.00000443 \text{ mg/m}^3}$$

**Child Cancer Risk = Intake x inhalation Unit Risk**

$$\text{Child Cancer Risk} = 0.00000443 \text{ mg/m}^3 \times 0.002 (\text{mg/m}^3)^{-1} =$$

$$\text{Child Cancer Risk} = \underline{\mathbf{0.0000000886 \text{ or } 8.86 \times 10^{-9}}}$$

**Cancer calculation at average TCE concentration of 0.0036 mg/L**

Child Inhalation Exposure Dose =

$$\frac{0.0036 \text{ mg/L} \times 0.5 \text{ L/m}^3 \times (7 \text{ hours}/24 \text{ hours day}) \times 174 \text{ days/year} \times 10 \text{ years}}{25550 \text{ days}} =$$

$$\text{Child Inhalation Exposure Dose} = 0.0000355 \text{ mg/m}^3$$

**Child Cancer Risk = Intake x inhalation Unit Risk**

$$\text{Child Cancer Risk} = 0.0000355 \text{ mg/m}^3 \times 0.002 (\text{mg/m}^3)^{-1} =$$

$$\text{Child Cancer Risk} = \underline{\mathbf{0.000000071 \text{ or } 7.1 \times 10^{-8}}}$$

### **Total Exposure from TCE in Drinking Water (Ingestion and Inhalation Exposure)**

Total cancer risk from Ingestion and Inhalation at maximum level of TCE detected:

$$(4 \times 10^{-8}) + (0.886 \times 10^{-8}) = \underline{\mathbf{4.88 \times 10^{-8}}}$$

Total cancer risk from Ingestion and Inhalation at average level of TCE detected

$$(3.2 \times 10^{-7}) + (0.71 \times 10^{-7}) = \underline{\mathbf{3.91 \times 10^{-7}}}$$

## **Discussion of Trichloroethylene (TCE) Studies**

In studies of humans who were exposed to TCE contaminated drinking water, varying conclusions have been reached. ATSDR has maintained a TCE Subregistry Baseline data file on approximately 5,000 persons with documented environmental exposure to TCE (along with other chemicals) through private wells. For the TCE Subregistry, ATSDR compared health conditions reported by the TCE Subregistry registrants with health conditions reported in a nation-wide survey of the non-exposed general population. Certain age groups reported some health conditions more frequently, while some had higher rates for only men or only women. This study did not confirm the health conditions (they were self-reported) and the study did not completely identify the exposure level. Persons in the study were exposed from 6.5 to 18 years to concentrations varying from less than 1 ppb to 19,380 ppb (determined from limited sampling data, usually one to two sampling events) (6,7). Findings of the latest follow-up of the study indicate that subregistry participants had a reporting rate above the national norms in various age groups for speech impairment and hearing impairment for children under 10 years of age (only on the baseline study), anemia and other blood disorders, stroke, urinary tract disorders, liver problems, kidney problems, diabetes rates, and skin rashes. Although the findings of ATSDR TCE Subregistry report do not identify a cause and effect relationship between TCE exposure and adverse health effects, they do reinforce the need to continue ongoing follow-up of the participants (7).

Of the reported health problems listed above, only the rate of strokes was reported to increase with increasing concentration of TCE. For the other health problems, their occurrence did not increase with higher exposure levels. If the health problems were related with the exposure to TCE, we would expect the number of people with a specific health problem to increase with higher levels. Therefore, it is unlikely that the reported health problems (anemia and other blood disorders, urinary tract disorders, liver problems, kidney problems, diabetes rates, and skin rashes) are associated with the exposure to TCE in private wells. Of those reporting strokes, a good portion also reported having other health problems including hypertension, diabetes, and being smokers, all of which contribute to the incidence of stroke (6).

Information on humans exposed to TCE for chronic periods of time (greater than 1 year) by the inhalation route consist mostly of studies of workers exposed to much higher levels than those detected. These studies indicate that the nervous system may be the most sensitive target, such as in a study of 50 workers employed for various lengths of time (1 month to 15 years) in different industrial cleaning and degreasing operations using TCE. Complaints due to chronic exposure included decreased appetite, sleep disturbances, ataxia, vertigo, headaches, short-term memory loss, and fewer word associations. A greater frequency of symptoms was noted in workers exposed to higher (85,000 ppb) than lower (14,000 ppb) mean TCE concentrations. Other studies of workers occupationally exposed for chronic periods of time indicate that the liver and kidney are targets of TCE (4).