Health Consultation

LONG LAKE TRICHLOROETHYLENE CONTAMINATION

NEW BRIGHTON, RAMSEY COUNTY, MINNESOTA

AUGUST 29, 2008

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

Health Consultation: A Note of Explanation

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

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HEALTH CONSULTATION

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NEW BRIGHTON, RAMSEY COUNTY, MINNESOTA

Prepared By:

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

FOREWORD

This document summarizes public health concerns related to contamination in a surface water body (lake) in Minnesota. It is based on a formal evaluation prepared by the Minnesota Department of Health (MDH). For a formal site evaluation, a number of steps are necessary:

- *Evaluating exposure:* MDH scientists begin by reviewing available information about environmental conditions at the site. The first task is to find out how much contamination is present, where it is found on the site, and how people might be exposed to it. Usually, MDH does not collect its own environmental sampling data. Rather, MDH relies on information provided by the Minnesota Pollution Control Agency (MPCA), the US Environmental Protection Agency (EPA), and other government agencies, private businesses, and the general public.

- *Evaluating health effects:* If there is evidence that people are being exposed—or could be exposed—to hazardous substances, MDH scientists will take steps to determine whether that exposure could be harmful to human health. MDH's report focuses on public health— that is, the health impact on the community as a whole. The report is based on existing scientific information.

- *Developing recommendations:* In the evaluation report, MDH outlines its conclusions regarding any potential health threat posed by a site and offers recommendations for reducing or eliminating human exposure to pollutants. The role of MDH is primarily advisory. For that reason, the evaluation report will typically recommend actions to be taken by other agencies—including EPA and MPCA. If, however, an immediate health threat exists, MDH will issue a public health advisory to warn people of the danger and will work to resolve the problem.

- *Soliciting community input*: The evaluation process is interactive. MDH starts by soliciting and evaluating information from various government agencies, the individuals or organizations responsible for the site, and community members living near the site. Any conclusions about the site are shared with the individuals, groups, and organizations that provided the information. Once an evaluation report has been prepared, MDH seeks feedback from the public. *If you have questions or comments about this report, we encourage you to contact us.*

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OR call us at: (651) 201-4897 *or* 1-800-657-3908
(toll free call - press "4" on your touch tone phone)

On the web: http://www.health.state.mn.us/divs/eh/hazardous/index.htmls

Summary

The Minnesota Pollution Control Agency (MPCA) requested the Minnesota Department of Health (MDH) review chemical contamination found in Long Lake, New Brighton, Minnesota. Surface water trichloroethylene (TCE) concentrations in the southern lobe of Long Lake have averaged around 1 microgram per liter (µg/L) during the summer months in recent years. Summertime concentrations have decreased from apparent highs in the 1980s around 10 µg/L. Higher concentrations have been measured in samples taken from under the ice in winter. *Cis*-1,2-dichloroethylene (DCE), a TCE dechlorination (degradation) product, has also been found at low concentrations in summer samples near the beach area in the southern lobe and under the ice. In addition, vinyl chloride was found in sediment porewater, demonstrating additional dechlorination of TCE or another chlorinated solvent.

Inhalation of TCE during swimming is likely to be the largest source of exposure to individuals. However, quantitative modeling of exposure is complex. TCE quickly volatilizes from surface water. The volatilization rate is primarily dependent on the temperature of the water, wind speed and water circulation. In addition, while the emission rate increases with increasing wind speed, so does dispersion of the TCE in air, thus simultaneously decreasing potential exposures. Changes in the rate of volatilization and dispersion occur over short time periods.

Even though the concentration of TCE in the lake appears to have been decreasing over the last 20 years, the persistence of TCE in surface water over this period of time suggests that there is a large and continuing source to the lake. This makes it likely that there is an as yet unidentified area, or areas, where TCE concentrations in surface water and/or groundwater are much higher. MDH recommends that the sources of contamination be identified, and that efforts be made to assure that discharges to the lake continue to decrease or are stopped.

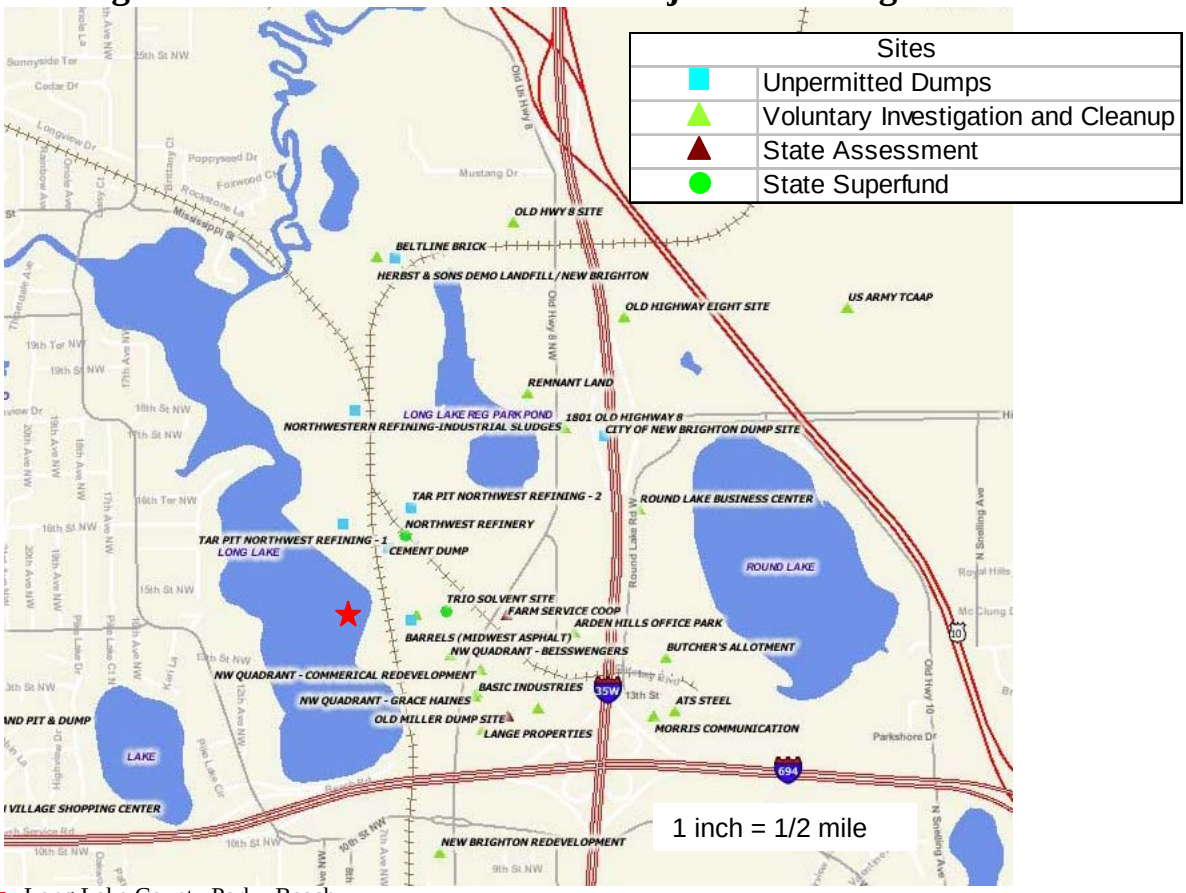
Modeling suggests that exposure to current contamination by inhalation, dermal exposure and ingestion will result in a calculated incremental cancer risk of less than 1 additional cancer in 100,000 individuals who swim in the lake very frequently for their entire life. There is No Apparent Public Health Hazard associated with frequent swimming or recreation in Long Lake. This evaluation applies to areas where surface water samples were taken and other well-mixed areas in the Lake. It is possible that there is not sufficient mixing in the area of the source (or sources) to maintain low TCE concentrations, but exposures in such an area may also be limited.

Introduction

The Minnesota Pollution Control Agency (MPCA) asked the Minnesota Department of Health (MDH) to review data on the presence of volatile organic compounds (VOCs), principally trichloroethylene (TCE), in Long Lake, for potential health risks. Long Lake is located in New Brighton, Ramsey County, Minnesota, an older, inner ring suburb of the Twin Cities. The western and northern shores of Long Lake are mostly populated by single family homes. There is a county park (Long Lake Regional Park) on the eastern

and southern shores of the Lake. Historically there were some industries to the east and northeast of the Lake. These include a refinery, a solvent company and an Army ammunitions plant (see Figure 1).

Figure 1: Hazardous Waste Sites Adjacent to Long Lake



★ - Long Lake County Park - Beach

From MPCA: http://pca-gis04.pca.state.mn.us/website/mes/mesfin/entry.htm

The park has a public beach (see Figure 1) which is used from late May to early September. Long Lake is a popular lake for boating, jet skiing, water skiing and other water sports during the summer months. Individuals swimming or otherwise recreating in a lake can be exposed to VOC contaminants through ingestion, dermal absorption or inhalation. In this Health Consultation, MDH evaluates the potential health risk from these chemicals that may be associated with a high level of activity in the lake for up to 32 years (ages 1 through 32; upper end default exposure period).

Abbreviations of units, variables and constants are defined in Appendix A - Glossary.

Site Background and History

Historically there were a number of industries to the east and northeast of Long Lake. Likely contaminants from these facilities included solvents and petroleum (see Figure 1). In general, groundwater flow is from the northeast. This would allow contaminants in

groundwater from these facilities to infiltrate into the Lake. In addition, ponds to the east of the Lake drain into Long Lake. Rice Creek enters into the Lake in the northeast (northern lobe) and exits the Lake in the northwest. Rice Creek is the major surface water outlet from the Lake. Surface water in the Lake appears to flow from the southern basin (lobe) of the Long Lake into the northern basin.

Among the historic potential industrial sources of contaminants on the east side of the Lake are Midwest Asphalt, Northwestern Refinery, Trio Solvent, Twin Cities Army Ammunition Plant (TCAAP), a number of old dump sites, and a few currently operating businesses (see Figure 1). Contaminated groundwater on the east side of the Lake may discharge into the Lake, but no specific TCE source has been identified. It has not been demonstrated or suggested that groundwater may flow from any other direction into the Lake. This Health Consultation does not review the groundwater contamination or the potential sources of contamination to Long Lake; but reviews available surface water contamination data and assesses potential human health risks.

Initial surface water sampling for VOCs in Long Lake took place in 1986 as part of a Ramsey County assessment of contamination from industries east of the Lake. TCE was found in samples taken from the area of the beach at 7 µg/L and 12 µg/L. Table 1 contains data from all available surface water samples collected by agencies, including the MPCA and Ramsey County. Sample locations for the 2002 – 2005 samples are shown in Figure 2. The latest round of sampling at the beach in 2005 showed TCE and DCE at 0.7 µg/L and 0.2 µg/L, respectively. No other dechlorination products have been found in surface water. However vinyl chloride, a relatively potent dechlorination product, has been found in sediment porewater, and it is likely that additional dechlorination products are present at concentrations below their analytical detection limits.

One sample under the ice (LL 112) showed the presence of some petroleum products. An additional sample showed the presence of 2 petroleum products at concentrations near the reporting limits. These detections suggest that there may be other chemicals entering the lake, but because the detections are infrequent and the contaminant concentrations are low, exposure to these contaminants at this time is not a concern. Therefore, there is no additional discussion of these contaminants in this document.

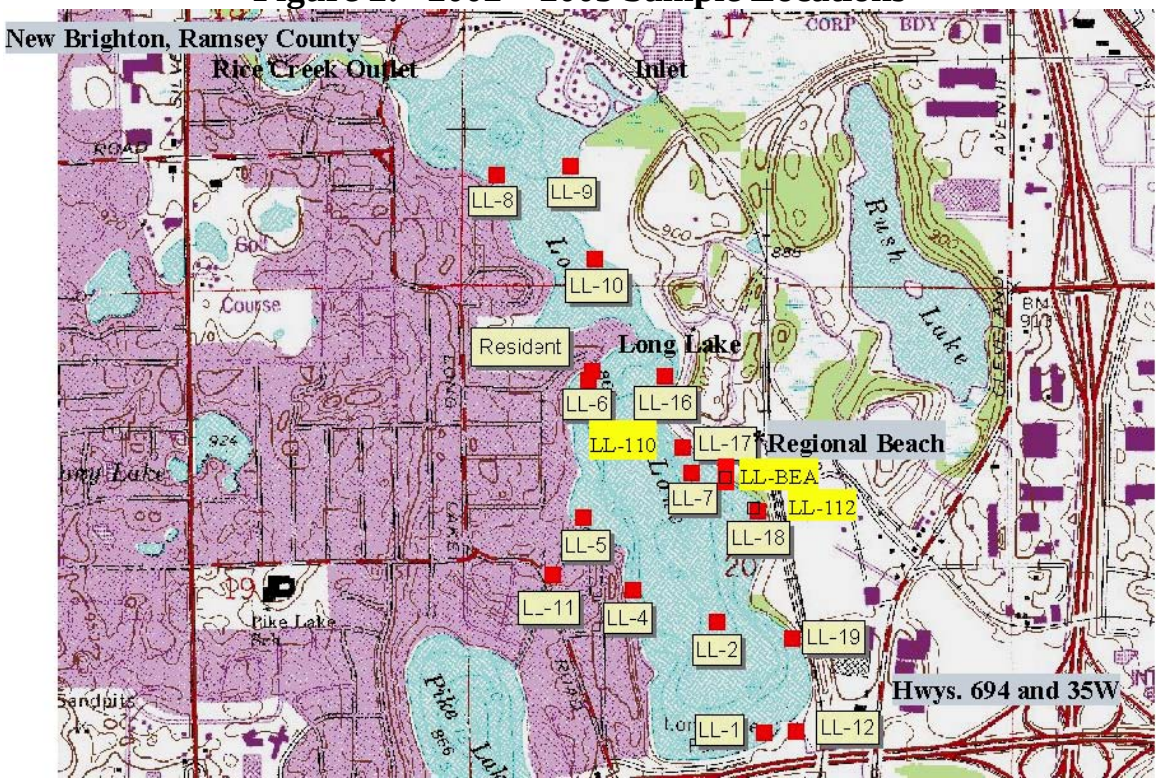
Table 1: VOCs in Surface Water

Date	Location	Trichloroethene µg/L	*cis* -1,2-dichloroethene µg/L	Vinyl Chloride µg/L	Other VOCs µg/L
04-( )-1986	**Beach**	12	0.5		
04-( )-1986	**Beach**	7	1.1		
04-( )-1986	**Slough-West**	<RL 0.4-1	<RL 0.4		
04-( )-1986	**Slough-East**	<RL 0.4-1	<RL 0.4		
05-( )-1986	**Beach**	0.9	NA		
05-( )-1986	Rice Creek	<MDL 0.5	NA		
05-( )-1986	N. Basin	1.2	NA		
05-( )-1986	**S. Basin**	1.5	NA		
06-( )-1986	**Beach**	1.9	NA		
07-( )-1986	**Beach**	3.8	NA		
09-( )-1986	**Beach**	1.6	NA		
02-( )-1987	**Beach**	20	2.5		
05-( )-1987	**Beach**	1.3	NA		
06-( )-1987	**Beach**	0.6	NA		
07-( )-1987	**Beach**	3	NA		
05-( )-1988	**Beach**	3.6	NA		
11-25-2002	**LL 1 - SE**	1.2	0.4	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 2 - S 18"**	1.2	0.4	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 3 - S 20'**	1.2	0.4	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 4 - SW**	1.2	0.4	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 5 - SW**	1.1	0.4	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 6 - SW**	1.3	0.5	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 16 - SE**	1.0	0.4	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 17 - SE**	1.3	0.5	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 18 - Beach**	1.4	0.6	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 18 - Beach (duplicate)**	1.3	0.5	<RL 0.5	<RL 0.1 - 20
11-25-2002	**LL 19 - SE**	1.5	0.5	<RL 0.5	<RL 0.1 - 20
07-11-2003	**LL 7 - Beach**	1.0	Peak<0.2	<RL 0.5	<RL 0.1 - 20
07-11-2003	LL 8 - NW	<RL 0.1	<RL 0.2	<RL 0.5	<RL 0.1 - 20
07-11-2003	LL 9 - NE	<RL 0.1	<RL 0.2	<RL 0.5	Petroleum Hydrocarbons 2 peaks < 0.2 µg/L ea.
07-11-2003	LL 10 - NE	0.4	<RL 0.2	<RL 0.5	<RL 0.1 - 20
07-11-2003	**LL 11 - Inlet Pike LI**	<RL 0.1	<RL 0.2	<RL 0.5	<RL 0.1 - 20
07-11-2003	**LL 12 - Inlet culvert**	<RL 0.1	<RL 0.2	<RL 0.5	<RL 0.1 - 20
09-25-2003	**LL 110 - Beach**	<RL 1.0	<RL 1.0	<RL 1.0	<RL 0.1 - 20
02-13-2004	**LL 112 - SE**	Peak <0.1	Peak<0.2	<RL 0.5	Petroleum Hydrocarbons 29 µg/L + 4 additional peaks
02-13-2004	**LL BEA - Beach**	3.5	1.7	<RL 0.5	<RL 0.1 - 20
06-24-2005	**LL 7-1 - Beach**	0.7	0.2	<RL 0.5	<RL 0.1 - 20
06-24-2005	**LL 7-2 - Beach**	0.7	0.2	<RL 0.5	<RL 0.1 - 20
06-24-2005	**LL 7-3 - Beach**	0.7	Peak<0.2	<RL 0.5	<RL 0.1 - 20

Surface water samples taken under ice

Sample Locations for 2002-2005 samples shown in Figure 3
Bold Locations in southern lobe of Long Lake; unbolded Locations in northern lobe
RL: laboratory reporting limit
MDL: method detection limit

Figure 2: 2002 – 2005 Sample Locations



(from MPCA, 2006)

Chemicals of Interest

Two VOCs, TCE and DCE, were found in surface water samples from Long Lake between 1986 and 2005 (see Table 1). In addition another VOC, vinyl chloride, was found in a single porewater sample (and duplicate) from the sediments of Long Lake at 1.5 µg/L (duplicate, 1.3 µg/L). TCE can be metabolized or chemically degraded to DCE and vinyl chloride in the environment. Therefore, these 3 compounds may have originated from a single TCE contamination source. Table 2 contains some toxicological criteria for these contaminants, developed for protection of public health. Minnesota Health Risk Values (HRVs), US EPA Reference Doses (RfDs), US EPA Reference Concentrations (RfCs), Agency for Toxic Substances and Disease Registry (ATSDR) Minimum Risk Levels (MRLs), and California Public Health Goals (PHGs) are intended to be protective of individuals, including sensitive individuals (e.g. children), who are exposed for a described duration. While each organization may specify different durations for their criteria, generally acute exposures are presumed to be short duration exposures (minutes to hours); intermediate (sub-chronic) exposures are typically exposures that last for a few months; and chronic exposures are exposures that may last up to a lifetime.

Table 2: Toxicity Criteria

Chemical	Health-based Standards (ingestion, dermal) mg/(kg·d)				Health-based Standards (inhalation) mg/m^3				Cancer Slope Factor (CSF) $(mg/(kg·d))^{-1}$		Cancer Unit Risk (UR) $(\mu g/m^3)^{-1}$	
	Chronic	Intermediate	Acute	Reference	Chronic	Intermediate	Acute	Reference		Reference		Reference
Trichloroethylene (TCE)	0.5 (kidney)			CA PHG	0.04 (CNS, liver, endocrine)			EPA, 2001	0.021 (liver)	MDH (site-specific)	3.3×10^{-6} (liver, lung)	MDH, 2007
			0.2 (developmental)	ATSDR MRL		0.5 (neurological)	11 (neurological)	ATSDR MRL				
cis -1,2-Dichloroethylene (DCE)		0.3 (hematopoietic)	1 (hematopoietic)	ATSDR MRL								
Vinyl Chloride	0.003 (liver)			EPA IRIS RfD	0.1 (liver)			EPA IRIS RfC	1.4 (liver)	EPA IRIS	8.8×10^{-6} (liver)	MDH HRV
	0.003 (liver)			ATSDR MRL		0.078 (liver)	1.3 (developmental)	ATSDR MRL				

EPA, 2001: http://oaspub.epa.gov/eims/eimscomm.getfile?p_download_id=4580
MDH, 2007: http://www.health.state.mn.us/divs/eh/risk/guidance/tcememo.html
ATSDR MRL (Minimum Risk Level): http://www.atsdr.cdc.gov/mrls/
CA PHG (Public Health Goal): http://www.oehha.ca.gov/water/phg/pdf/tce_f.pdf
EPA IRIS RfD (Reference Dose) and RfC (Reference Concentration): http://www.epa.gov/ncea/iris/subst/1001.htm
MDH HRV (Health Risk Value): http://www.health.state.mn.us/divs/eh/air/hrvtable.htm

In order for chemicals to impact human health there must be exposure. For chemicals that are not carcinogens, a threshold below which no health effects will occur is determined. Cancer risk is evaluated by MDH such that exposure to chemicals from a site are not considered for analyses or regulation unless there is a calculated incremental increase of greater than 1 in 100,000 individuals exposed for a lifetime (more than $1x10^{-5}$ risk).

Trichloroethylene (TCE)

The weight of evidence provides support for classifying TCE as a carcinogen (MDH 2007; Appendix B). In December 2007 MDH provided a Health-Based Value (HBV) for TCE inhalation based on the cancer unit risk developed by the California EPA's Office of Environmental Health Hazard Assessment (OEHHA) (CA OEHHA 2005). This unit risk was adjusted for age utilizing the US EPA method of adjusting cancer potency estimates for early life exposure (USEPA 2005). The resulting Age Adjusted Unit Risk (AAUR) was $3.3 \times 10^{-6}(\mu g/m^3)^{-1}$. The MDH HBV (lifetime exposure level) calculated for an additional lifetime risk of 1×10^{-5} is 3 $\mu g/m^3$. Additional information on this HBV can be found in the MDH Memo Trichloroethylene: Chronic Health-Based Value for Air (2007; Appendix B).

OEHHA has developed an oral cancer slope factor for TCE $(0.007\ (mg/kg/d)^{-1})$ based on the same studies used to develop their inhalation unit risk (CA OEHHA 2005). California also has used a cancer slope factor of $0.013\ (mg/kg/d)^{-1}$ to develop a drinking water Public Health Goal (CA OEHHA 1999). Furthermore, in 2001 the EPA published a draft assessment of TCE (USEPA 2001) that suggested a range of cancer slope factors from $0.02\ (mg/kg/d)^{-1}$ to $0.4\ (mg/kg/d)^{-1}$. This draft received substantial comments from the National Academy of Sciences (NRC 2006) and is currently under revision. At this time, MDH recommends using the EPA Maximum Contaminant Level (MCL) for TCE when evaluating drinking water (http://www.epa.gov/safewater/consumer/pdf/mcl.pdf). However, recreational exposures to TCE contaminated surface waters are quite different

than exposures through drinking water, and calculating cancer risk from dermal exposure and incidental ingestion requires a cancer slope factor. For the modeling described throughout this Health Consultation and the development of the Long Lake site-specific screening concentrations, the California cancer slope factor of 0.013 $(mg/kg/d)^{-1}$ was used. For the purposes of calculating site-specific screening criteria, the California oral slope factor was adjusted similarly to the adjustment used in the MDH TCE Memo (MDH 2007) from the published value of 0.013 $(mg/kg/d)^{-1}$ to 0.021 $(mg/kg/d)^{-1}$. The adjustment is based on the EPA method of adjusting cancer potency estimates for early life exposure.

This assessment also uses 0.04 mg/m^3, from the EPA Draft Trichloroethylene Risk Assessment (USEPA 2001), as an inhalation screening criterion for non-cancer exposure to TCE. ATSDR has non-cancer intermediate and acute MRLs for inhalation of TCE, as well as an acute MRL for the ingestion of TCE (ATSDR MRL 2007).

The Cancer Unit Risk ($3.3 \times 10^{-6} (\mu g/m^3)^{-1}$) and the Cancer Slope Factor ($0.021\ (mg/kg/d)^{-1}$) were the most restrictive criteria for evaluating TCE contamination in Long Lake and will be a focus of the risk evaluation in this document.

cis-1,2-dichloroethylene (DCE)

Limited toxicological data are available for DCE (U.S. National Library of Medicine 2008). MDH is not aware of any human toxicity characterizations of DCE for inhalation exposure nor for DCE as a carcinogen (see Table 2). However, DCE appears to generally have similar or less toxicity than TCE. ATSDR has published an intermediate MRL for DCE (0.3 mg/kg/d) and an acute MRL for DCE (1 mg/kg/d) for ingestion (ATSDR MRL 2007). These values were used to evaluate DCE ingestion and dermal exposure from surface water. Further direct evaluation of DCE was not possible. For screening purposes only, TCE was used as a conservative surrogate for chronic inhalation (non-cancer) exposure to DCE.

Vinyl Chloride

Vinyl chloride is considered by the US EPA to be a known carcinogen (USEPA IRIS 2000). The US EPA Cancer Slope Factor and the Cancer Unit Risk used to develop the MDH HRV are shown in Table 2. The vinyl chloride cancer slope factor and unit risk both include early life adjustments. In addition the US EPA has evaluated the non-cancer toxicity of vinyl chloride and published a chronic Reference Concentration (RfC) and a chronic Reference Dose (RfD) for vinyl chloride as well. ATSDR MRLs for chronic ingestion and intermediate and acute inhalation (ATSDR MRL 2007) are noted in Table 2.

The Cancer Slope Factor and Cancer Unit Risk are the most restrictive criteria for evaluating vinyl chloride exposures in Long Lake and will be a focus of the risk evaluation in this document.

Discussion

Previously, MDH developed a Sediment Screening Model (SSM) for evaluating exposure to chemicals in sediments (MDH 2005). The SSM evaluated calculated sediment

screening concentrations that are protective for exposure to ingestion, dermal exposure, inhalation and fish consumption based on partitioning of contaminants and standard exposure parameters to environmental media. For this assessment, the SSM was modified to evaluate the inhalation, ingestion and dermal exposure pathways to contaminants in water. A description of the assessment and equations used to calculate the site-specific screening values can be found in Appendix C.

Potential Exposures

Table 3 contains a reasonable maximum schedule of water-related activities that were used to evaluate reasonable maximal exposure (RME) to contaminants in Long Lake. It is assumed that this activity is maintained from ages 1 through 32 years old for the highly exposed individual. Reasonable maximums from EPA, used to estimate the frequency and duration that an individual may swim are similar to but somewhat higher than MDH defaults. EPA suggests that an individual with a high exposure may swim 1 hour per event, 1 event per day and 150 days per year (USEPA 2002; Table 9-16). MDH defaults are more suitable for outdoor swimming in the Minnesota climate.

Table 3: Assumed Maximum Water-related Activity

Age (yr)	Wading Events (0.5 hr duration)					
	May, September		June, July, August		Totals	
	8.6 weeks/year		12.9 weeks/year		events/year	days/year
	events/day	days/week	events/day	days/week		
1 - 6	1	3	0	0	25.8	25.8
7 - 17	1	3	0	0	25.8	25.8
18 - 33	1	3	0	0	25.8	25.8
1 - 33	1.0	3	0	0	25.8	25.8
Age (yr)	Swimming Events (0.5 hr duration)					
	May, September		June, July, August		Totals	
	8.6 weeks/year		12.9 weeks/year		events/year	days/year
	events/day	days/week	events/day	days/week		
1 - 6	0	0	2	6	155	77.4
7 - 17	0	0	2	6	155	77.4
18 - 33	0	0	1	6	77.4	77.4
1 - 33	0	0	1.5	6	117	77.4

The Superfund Exposure Assessment Manual (USEPA 1988) states that 50 milliliters per hour (mL/hr) swimming is a reasonable estimate for incidental ingestion by an adult. Because children playing in water ingest considerably more water than adults, it is conservatively assumed that children and adolescents ingest five times the adult ingestion. There are no published data on water ingestion during wading. This analysis assumes that a reasonable adult ingestion for screening purposes is 1/100th of the adult swimming ingestion and, because the difference between wading and swimming for children is not discrete, ingestion by 1-17 year-olds is 1/10th of their swimming ingestion. Uptake of ingested chemicals in this assessment was assumed to be 100%.

Dermal exposure to surface water occurs during any wading or swimming event. Dermal exposure to a chemical in water is based on the fraction of that chemical in water non-actively transferred through the skin and into the body. Exposure only occurs while the event is taking place. Therefore, more than one event during a single day results in more than one exposure. The percent of the total body surface area that is exposed to surface water during wading and swimming was assumed to be 20% and 90%, respectively. Dermal uptake of the chemicals in this assessment was calculated using surface water dermal permeability coefficients (K_p) and guidance published by the US EPA in Risk Assessment Guidance for Superfund (RAGS), Part E (2004).

Inhalation exposure is limited to the fraction of the time that the exposed individual is swimming or wading. The inhalation rate for an individual that is in the water is expected to be above the 24 hour average inhalation rate. Therefore, as described in Appendix C, the inhalation rate is adjusted, proportionally ($Inh_{adj} = 1.5$), so that it is similar to an occupational inhalation rate (USEPA 1994).

Toxicity of Early Life Exposures

As noted above, non-cancer toxicity criteria are derived to be protective of sensitive individuals including children. In addition exposure modeling in this document includes early-life exposures. Therefore the non-cancer screening criteria developed in this Health Consultation are protective of early life exposures.

Early life exposures to TCE and vinyl chloride are expected to result in more cancer risk than exposures later in life (see previous section on Chemicals of Interest). This early life susceptibility is considered in the cancer slope factors (and unit risks) used in this Health Consultation by averaging 3 different age-dependent slope factors. However, this assumes that exposures during these 3 periods of life (ages 0-2, 3-15, 16-70) are similar (USEPA 2005).

The exposure model applied to Long Lake assumes higher exposures from age 1 through adolescence. Therefore, cancer slope factors (and unit risks) for exposure at this site were adjusted to account for a 1.59 times increase in lifetime cancer risk due to the increased amount of early life recreational exposure. See Appendix D for calculation of this site-specific adjustment factor.

Relative Hazard/Risk Evaluation for Different Routes of Exposure

As noted in the Chemical of Interest section above, data on the inhalation hazard and the cancer risk of DCE are not available, but DCE is presumed to be less toxic than TCE. Therefore, the DCE inhalation screening value is calculated using a non-cancer inhalation toxicity criterion for TCE. This provides a conservative value that may be used to screen DCE exposure in Long Lake.

People swimming or otherwise contacting surface water can be exposed to contaminants in many ways. They can ingest the contaminant with water; they can be exposed through the skin (dermally); they can inhale contaminant that has evaporated from the water, and; they can eat the contaminant that has accumulated in fish that they consume.

Given the physical / chemical characteristics of TCE, DCE and vinyl chloride (and essentially all VOCs), these chemicals do not accumulate in fish so this pathway has not been evaluated.

Evaluating ingestion, dermal and inhalation exposures requires estimating an exposure time and exposure concentration for each route of exposure over an averaging length of time (i.e. an hour, day, season, year, lifetime). Reasonable maximal exposure (RME) times are shown in Table 3. Ingestion and dermal exposure concentrations can be estimated from the contaminant concentration in surface water; and inhalation exposure concentrations can be estimated from the contaminant concentration in air.

Long Lake surface water contaminant concentrations are available, but air concentrations are not available. Measurement of inhalation exposure concentrations, especially in the layer of air close to the surface of a lake is difficult and, unfortunately, such data are not available. The concentration of a VOC in air above a lake is related to the concentration of the VOC in the lake. Therefore, there should be a relationship between the inhalation exposure and the ingestion and dermal exposures.

Appendix C contains the equations used to calculate the screening values and determine the contribution of different routes of exposure. Appendix E contains a discussion of the results of the modeling: the relationship between Long Lake water and overlying air concentrations; the relative importance of ingestion, dermal and inhalation exposures to contaminants in Long Lake; and their relationship to the total VOC exposure.

Results from the screening model suggests that surface water concentrations at which MDH would recommend additional evaluation of potential exposures at Long Lake would be 40-50 µg/L, 700 - 1000 µg/L and 2-2.5 µg/L for TCE, DCE and vinyl chloride, respectively. RME exposure to each contaminant through ingestion, dermal and inhalation exposures for 32 years at these concentrations, could convey an additional lifetime incremental cancer risk approaching 10^{-5}. Clearly, contaminant concentrations found in Long Lake have not approached these concentrations.

Contamination Source

Size of the source

TCE and its breakdown products, DCE and vinyl chloride, are very volatile compounds. Therefore, when they are mixed into water they will partition out of the water and into air rather quickly. In fact, at 19.8° C (Long Lake summer average temperature), it is expected that the amount of TCE that is in the top 3 centimeters (cm) of the entire lake will volatilize every hour (water-side mass transfer coefficient from MPCA 2006). That means the amount of TCE found in the Lake to 10 feet depth evaporates from the Lake every 4 days. In order to maintain concentrations from season to season, the contaminant sources have to replace all of the TCE that is evaporating. Even though contaminant concentrations are higher in some areas of the Lake than others, maintaining measurable concentrations of TCE and DCE in surface water for more than 20 years requires a large and constant source.

While the highest measured contamination has been found in the area of the beach, no source discharge area has been identified. Attempts to find areas of seepage or areas of surface water infiltration have not located the place, or places, where contamination enters the Lake. If the source discharge is in an area of the Lake where there is significant mixing, concentrations of VOCs and exposures to people using the lake are likely to be limited. However, if the source discharge is in a confined area like a small inlet, exposures may be quite high. Given the resources expended trying to locate the source, the relatively small size of the Lake, and the large amount of activity on and around the Lake, it is likely that the discharge is not in a confined area. It is more likely that the discharge area is off-shore in the body of the Lake.

Contamination in the northern lobe

There were a limited number (5) of samples taken from the northern lobe of the Lake (Table 1: Rice Creek, North Basin, LL-8, LL-9, LL-10). Two of these samples showed TCE above detection/reporting limits. As noted above, the surface water appears to flow from the southern lobe of the Lake to the northern lobe. Therefore, the presence of contaminants in the southern lobe suggests that there is a source that discharges into the southern lobe. On the other hand, the presence of contamination in the northern lobe may be the result of contamination of the southern lobe and northern migration of the contamination in surface water, or there may be an additional source(s) discharging contamination into the northern lobe. If the surface water flows from south to north slowly enough, TCE will evaporate before it reaches the northern lobe. This may suggest the presence of an additional source in the northern lobe.

Additional Potential Exposures

Groundwater contaminated with volatile organic compounds (VOCs), such as TCE and its dechlorination products, will release vapors into soil. These vapors will rise through the soil and escape into the atmosphere. If a building overlays a contaminated groundwater plume, the vapors often penetrate the building, and significant concentrations can accumulate in the basement of the building. This phenomenon is called vapor intrusion. From data reviewed it is apparent that there is a large, unidentified source(s) of TCE and TCE dechlorination products entering Long Lake. The source or sources are likely to be contaminated groundwater plumes. The only way to be certain that vapor intrusion is not now and never will be a problem in buildings near the Lake is to characterize the contaminated plumes in the vicinity of Long Lake.

Conclusion

MDH has reviewed data from surface water at Long Lake and concluded that identified exposures to VOC contaminants in the Lake are currently at levels below health concern. The amount of contaminants in surface water and the length of time that measurable concentrations have persisted suggests that there is a large source that is continuing to infiltrate into the Lake. If the source discharges into a confined area of the Lake where contaminant concentrations may become high, exposures could be significant. However, it is more likely that the source(s) are into areas of the Lake where there is good mixing. Finding the source(s) and the

location(s) where the contamination enters the Lake would assure that there are not unknown exposures occurring in other areas of the Lake. In addition, characterizing contaminated groundwater plumes in the vicinity of the Lake would prevent unknown exposures to contaminants by vapor intrusion should buildings be located above the groundwater plume.

Contaminant concentrations in Long Lake are below levels of concern even for people who have a high level of activity in open areas of the Lake. Surface water concentrations at which MDH would suggest additional evaluation of potential exposures at this specific site (Long Lake) would be 40-50 µg/L, 700 - 1000 µg/L and 2-2.5 µg/L for TCE, DCE and vinyl chloride, respectively. In addition, if the concentration of any of these chemicals approaches these levels, evaluation of potential impacts should include cumulative impacts from all chemicals of interest.

The potential exposures modeled in this evaluation are reasonable maximum exposures. However it is possible that there may be some individuals who swim in the lake even more often than the model assumes. It is unlikely that these individuals would incur any health risk from contaminants in the lake because surface water concentrations are considerably below concentrations of concern. If the contaminant concentrations increase, the potential exposures should be reassessed.

The estimates of VOC concentrations in air (inhalation exposure concentrations) at this site are uncertain. Collection of VOC concentrations in air above Long Lake under different weather conditions or site-specific inhalation exposure data would provide additional assurance that exposures are below levels of concern.

Consumption of fish from Long Lake should not be limited due to concerns about VOC contamination. However, fish consumption advice due to state-wide concerns about mercury accumulation in large predatory fish, still apply to Long Lake (http://www.health.state.mn.us/divs/eh/fish/).

The detection of petroleum products in 2 samples suggest that there may be other chemicals entering the lake. Exposure to these contaminants at this time is not a concern.

There is No Apparent Public Health Hazard associated with frequent swimming or activity in Long Lake as defined by the Agency For Toxic Substances and Disease Registry (http://www.atsdr.cdc.gov/COM/hazcat.html).

Recommendations

- Potential exposures should be further characterized if:
  - surfacewater concentrations in Long Lake approach 40-50 µg/L, 700 - 1000 µg/L and 2-2.5 µg/L for TCE, DCE and vinyl chloride, respectively;
  - the assumed exposure scenarios in this document underestimate actual exposures that occur on Long Lake.

- The source(s) of VOC contamination to Long Lake should be located and, if the source is in a confined area, potential localized exposures should be evaluated.
- Chemical analysis of samples for contaminants in addition to VOCs should continue.
- Analysis of contamination source(s) should include analysis of the potential for significant exposures by vapor intrusion.

Public Health Action Plan

MDH will provide assistance to the MPCA and other governmental agencies in evaluating potential health risk from contamination at this site. In addition MDH will, on invitation, work with the community to understand the health impacts that may be associated with exposure to contaminants.

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Appendix A: Glossary

Unit abbreviations

°K = degrees Kelvin
% = percent
atm = atmospheres
cm = centimeters
d = days
g = grams
hr = hours
kg = kilograms
L = liters
m = meters
mg = milligrams
mL = milliliters
mol = moles
µg = micrograms
wk = weeks
yr = years

Variable abbreviations

ABS_{GI} = chemical specific constant – fraction of applied dose absorbed in primary (RfD) study (unitless)

ABS_{SW} ((mg/(cm^2·event))/(mg/cm^3)) general term representing the dermally absorbed dose from a chemical concentration in water: dependent on event duration and chemical specific factors.

$ABS_{SW\text{-}met}$ = dermal absorption of metals from water (mg/(cm^2·event))/(mg/cm^3) (*Equation #C-10*)

$ABS_{SW\text{-}org}$ = dermal absorption of organics from water (mg/(cm^2·event))/(mg/cm^3) (*Equation #C-11*)

$AccptRsk_c$ = acceptable risk - cancer (1:100,000 (unitless))

AF_{ED} = event duration-dependent adjustment factors (hr/event) (*Equation #C-18, #19*)

$AT_{(c)}$ = cancer averaging period (70 yrs; EPA convention (EPA 1989))

β = ratio of stratum corneum and epidermis permiabilities (unitless) (*Equation #C-13*)

BW, bw = body weight (kg)

$CF_{d/y}$ = conversion factor (365 d/yr)

$CF_{hr/d}$ = conversion factor (24 hr/d)

$CF_{cm3/L}$ = conversion factor (1,000 cm^3/L)

$CF_{L/m3}$ = conversion factor (1,000 L/m^3)

$CF_{L/mL}$ = conversion factor (0.001 L/mL)

$CF_{µg/mg}$ = conversion factor (1,000 µg/mg)

CSF_{adj} = site, exposure-specific - CSF and UR adjustment factor (1.59 (unitless); Appendix D)

CSF_c = oral cancer slope factor ((mg/(kg$_{bw}$·d))$^{-1}$)

Dil_{air} = air dilution adjustment factor (equilibrium = 1) (unitless)

ED = event duration (hr/event)

EF_{swm} = event frequency - swimming (event/yr)

EF_{wad} = event frequency - wading (event/yr)

$EP_{(c)}$ = exposure period - cancer (32 years)

FA = chemical specific constant – fraction absorbed from water (unitless)
i = from 1 – 32 (yrs)
Ing_{swm} = surface water ingested per hour swimming (mL/hr)
Ing_{wad} = surface water ingested per hour wading (mL/hr)
Inh_{adj} = inhalation rate adjustment above mean rate (1.5 (unitless))
Inh_{frac} = fraction of time onsite (unitless) (*Equation #C-5*)
Inh_{frac-c} = time onsite - lifetime average (unitless) (*Equation #C-6*)
K_H = chemical specific constant – Henry's Law (@ 298.13ºK) (atm-m^3/mol)
K_p = chemical specific constant – permeability coefficient (cm/hr)
MW = chemical specific constant – molecular weight (g/mol)
R = ideal gas constant (@ 1 atm) (0.082057 L/ (mol · ºK))
RfC = chemical specific constant – reference concentration - chronic criteria for general public (air) (mg/m^3)
RfD = chemical specific constant – reference dose - chronic criteria for general public (ingestion, dermal) (mg/(kg·d))
SA_{ttl} = total surface area (cm^2)
$SA_{\%swm}$ = percent of body exposed swimming (%)
$SA_{\%wad}$ = percent of body exposed wading (%)
SC_{Inh} = route-specific air screening concentration (chronic) - inhalation (µg/m^3) (*Equation #C-23*)
SC_{Inh-c} = route-specific air screening concentration (cancer) - inhalation (µg/m^3) (*Equation #C-24*)
$SWC_{\%x}$ = % contribution by individual routes of exposure to SSV_{ttl} (%) (*Equation #C-27*)
SWC_{Derm} = route-specific surface water screening concentration (chronic) - surface water dermal (mg/L) (*Equations #C-9, #C-9a*)
SWC_{Derm-c} = route-specific surface water screening concentration (cancer) - surface water dermal (mg/L) (*Equation #C-20*)
SWC_{Ing} = route-specific surface water screening concentration (chronic) - water ingestion (mg/L) (*Equation #C-7*)
SWC_{Ing-c} = route-specific surface water screening concentration (cancer) - water ingestion (mg/L) (*Equation #C-8*)
SWC_{Inh} = route-specific surface water screening concentration (chronic) - inhalation (mg/L) (*Equation #C-21*)
SWC_{Inh-c} = route-specific surface water screening concentration (cancer) - inhalation (mg/L) (*Equation #C-22*)
SWC_{ttl} = surface water screening concentration - chronic (mg/L) (*Equation #C-25*)
SWC_{ttl-c} = surface water screening concentration - cancer (mg/L) (*Equation #C-26*)
SW_{Derm} = surface area exposed to surface water (cm^2·event/(kg_{bw}·d)) (*Equation #C-3*)
SW_{Derm-c} = surface area exposed to surface water - lifetime average (cm^2·event/(kg_{bw}·d)) (*Equation #C-4*)
SW_{Ing} = amount of water ingested (L/(kg_{bw}·d)) (*Equation #C-1*)
SW_{Ing-c} = amount of water ingested - lifetime average (L/(kg_{bw}·d)) (*Equation #C-2*)
τ = lag time per event (hr/event) (*Equation #C-12*)
T = temperature (293.13ºK default)
t^* = chemical specific constant – time to steady state (hr) (*Equation #C-14, #C-15*)
UR_c = chemical specific constant – unit risk - cancer ((µg/m^3)$^{-1}$)

Appendix B: Memo

The following guidance was developed by the Health Risk Assessment Unit (HRA) of the Minnesota Department of Health (MDH) at the request of the Site Assessment and Consultation Unit of MDH and the Minnesota Pollution Control Agency.

Trichloroethylene: Chronic Health-Based Value for Air
December 28, 2007

Chemical: Trichloroethylene (TCE)
CAS Number: 79-01-6
Endpoint: Cancer
Chronic Value: 3µg/m^3
Sources: 2001 U.S. EPA draft guidance for TCE; California EPA's OEHHA, 2005; and NRC, 2006

The MDH has developed a chronic Health-Based Value (HBV) of 3 µg/m^3 for inhalation exposures to TCE. A description of the techniques, assumptions and caveats used in developing this number follows.

There is a large and still growing body of experimental and epidemiological information concerning the toxicity of TCE. Rather than increasing the confidence in the ability of risk assessors to develop a protective number for TCE, the accumulated research has resulted in a great deal of controversy regarding the level of exposure required to produce effects in humans. Research, and the evaluation of this research, is continuing and it is likely that as new information and EPA's reevaluation become available the HBV that has been developed will need to be reevaluated.

At this point it is clear that TCE is capable of inducing several types of tumors in different organs in experimental animals. Experimental information also indicates that TCE likely operates by multiple modes of action, including genotoxicity, to induce cancers.

Following a review of the available literature, including the 2001 U.S. EPA draft guidance for TCE (U.S. EPA, 2001) and the National Academy of Sciences report generated in response to EPA's draft guidance (NRC, 2006), HRA has developed an HBV for TCE. Given the increased weight of evidence (discussed in the NAS report) indicating the potential for carcinogenicity in humans it is prudent to develop an HBV for TCE using cancer as an endpoint.

As pointed out by the NAS panel, epidemiological evidence accumulated to date indicates that TCE is likely to be carcinogenic in humans. In addition, epidemiological studies have associated TCE exposure to the induction of multiple types of cancer in humans. However, these data are insufficient to support quantitative dose response modeling for TCE and cancer. The committee recommended the toxicologic data be used

to fit the primary dose response models and that the available epidemiological data be used only for validation.

Based on these comments HRA has elected to use an analysis of rodent cancer data posted on the California EPA's OEHHA website (http://www.oehha.ca.gov/air/hot_spots/pdf/May2005Hotspots.pdf ***)*** as the basis for the HBV (California EPA, 2005). The California Department of Health Services used data from four studies in male and female mice to generate independent estimates of unit risk. For this analysis they used:

- PBPK modeling to adjust the applied dose for metabolism
- Surface area scaling to account for interspecies variation
- Linearized multistage modeling for low dose risk assessment

A best estimate of unit risk was obtained by taking the geometric mean of the independent unit risks from the four studies.

This approach is consistent with recommendations from the NRC report which suggested that, because the available information is insufficient to determine the best dose response curve model for TCE, a linear extrapolation between zero and the modeled point of departure is acceptable and consistent with current techniques suggested in EPA's 2005 cancer guidelines (U.S. EPA, 2005a).

California EPA's analysis produced a unit risk of 2.0×10^{-6} $(\mu g/m^3)^{-1}$. Because it is likely that at least some types of cancer induced by TCE are produced via a mutagenic mode of action, HRA has utilized the U.S. EPA method of adjusting cancer potency estimates for early life exposure (U.S. EPA, 2005b). Age specific adjustments for exposure are currently unavailable. The equation used follows:

Age Adjusted Unit Risk (AAUR) = $2/70\ [(2 \times 10^{-6}) \times 10] + 13/70\ [(2 \times 10^{-6}) \times 3] + 55/70\ [(2 \times 10^{-6} \times 1] = 3.3 \times 10^{-6}\ (\mu g/m^3)^{-1}$

Consistent with MDH policy an additional lifetime risk level of 1×10^{-5} was used with the AAUR of 3.3×10^{-6} $(\mu g/m^3)^{-1}$ to calculate an exposure level.

$$\frac{1 \times 10^{-5}}{3.3 \times 10^{-6}} = 3.03 \text{ (rounded to 3 } \mu g/m^3)$$

TCE is toxic to a number of organs and several other non-cancer endpoints have been used to develop health-based numbers. The U.S. EPA's draft guidance for TCE developed a reference concentration for TCE of 40 $\mu g/m^3$ based on CNS, liver and endocrine system toxicity (U.S. EPA, 2001). The California EPA's OEHHA has developed a chronic reference exposure level (REL) of 600 $\mu g/m^3$ based on CNS effects in workers (California EPA, 2005). The HBV developed for cancer would be adequately protective of these non-cancer endpoints.

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Appendix C: Sediment Screening Model adapted for developing site-specific surface water screening concentrations.

Abbreviations of units, variables and constants are defined in Appendix A - Glossary.

The MDH Sediment Screening Model (MDH 2005) was developed to calculate sediment concentrations of contaminants that would not exceed human health exposure criteria when 6 possible routes of exposure are considered. The model was adapted to calculate surface water concentrations and air concentrations of contaminants that would not result in exceedance of exposure criteria for individuals that frequently wade and swim in Long Lake. This model assumes that exposure to sediments and fish consumption do not contribute to contaminant exposures to individuals. The potential for exposure to these chemicals from additional sources or outside of the modeled exposure scenarios were not evaluated. Furthermore, each chemical was evaluated separately and endpoint similarities between chemicals were not considered.

The equations below describe the calculations performed to develop protective site-specific screening values. The equations have been divided into 5 categories:

- Exposure calculations
- Route-specific surface water screening value calculations
- Activity-exposure only air screening concentration calculations
- Surface water screening concentration calculations
- Route-of-exposure contribution (percent) calculations

Non-cancer exposures were calculated using reasonable maximum exposure estimates for a 1 through 6 year old child. Cancer exposures are assumed to occur from age 1 through age 32.

Table C-1 contains the physical chemical properties of the chemicals of interest.

Table C-1: Physical / Chemical Properties

	Molecular weight (MW; g/mol)	**Henry's Law constant*** (K_H; atm-m^3/mol)	**Dermal permiability coefficient**§ (K_p; cm/hr)	**Fraction absorbed**§ (FA; unitless)
Trichloroethylene	131.39	0.00985	0.012	1
cis -1,2-dichloroethylene	96.94	0.00408	0.012	1
Vinyl chloride	62.5	0.0278	0.0056	1

* (Syracuse Research Corporation 2008)

§ (USEPA 2004)

Exposure calculations

*Average daily incidental surface water **ingestion** rate calculation **(non-cancer)***

SW_{Ing} { $L/(kg_{bw} \cdot d)$ } = $(Ing_{wad} * ED_{wad} * EF_{wad} + Ing_{swm} * ED_{swm} * EF_{swm}) / (BW * CF_{d/y}) * CF_{L/mL}$

Equation #C-1.

*Lifetime average daily incidental surface water **ingestion** rate calculation **(cancer)***

$$SW_{Ing\text{-}c} \{ L/(kg_{bw} \cdot d) \} = (\sum_{i=1}^{33} SW_{Ing(i)}) / AT_{(c)}$$

Equation #C-2.

Where:

$SW_{Ing(i)}$ = (SW_{Ing} at age i) * 1 yr

*Average daily **dermal** surface water contact rate calculation **(non-cancer)***

Since exposure to chemicals in water only occurs during an event, the event frequency for this route-of-exposure is in events per year (event/yr).

Annual daily average surface area exposed to surface water during wading and swimming is:

SW_{Derm} { $cm^2 \cdot event/(kg_{bw} \cdot d)$ } = $SA_{ttl} * (SA_{\%wad} * EF_{wad} + SA_{\%swm} * EF_{swm}) / (BW * CF_{d/y})$

Equation #C-3.

*Lifetime average daily **dermal** surface water contact rate calculation **(cancer)***

The lifetime average surface area exposed to surface water during wading and swimming is:

$$SW_{Derm\text{-}c} \{ cm^2 \cdot event/(kg_{bw} \cdot d) \} = (\sum_{i=1}^{32} SW_{Derm(i)}) / AT_{(c)}$$

Equation #C-4.

*Average activity-related **inhalation** fraction calculation **(non-cancer)***

For screening assessment, the inhalation rate during wading and swimming is adjusted proportionally, so that it is similar to an expected inhalation rate for occupational exposures (USEPA 1994). The fraction of time (annual average) that air overlying contaminated surface water is breathed during recreation is calculated with:

Inh_{frac} { unitless} = $Inh_{adj} * (EF_{wad} * ED_{wad} + EF_{swm} * ED_{swm}) / (CF_{d/y} * CF_{hr/d})$

Equation #C-5.

*Lifetime average activity-related **inhalation** fraction calculation **(cancer)***

The fraction of lifetime inhalation associated with wading and swimming on the site may be calculated from:

$$Inh_{frac\text{-}c}\ \{\ unitless\} = (\sum_{i=1}^{32} Inh_{frac(i)}) / AT_{(c)} \qquad \textit{Equation \#C-6.}$$

Route-specific surface water screening value calculations

*Calculating protective surface water concentrations for water **ingestion (non-cancer)***
A protective surface water concentration for a chemical can be calculated from:

$$SWC_{Ing}\ \{\ mg/L\ \} = RfD /\ SW_{Ing} \qquad \textit{Equation \#C-7.}$$

*Calculating protective surface water concentrations for water **ingestion (cancer)***
A protective surface water concentration for cancer endpoints and the water ingestion pathway can be calculated from:

$$SWC_{Ing\text{-}c}\ \{\ mg/L\ \} = (AccptRsk_c / (CSF_{adj} * CSF_c)) / SW_{Ing\text{-}c} \qquad \textit{Equation \#C-8.}$$

*Calculating protective surface water concentrations for **dermal** exposure **(non-cancer)***
Dermal uptake of contaminants directly from water is a function of contaminant concentration in the sediment, equilibrium partitioning into water, the duration of any single activity, and the permiability of the skin to the chemical. Water values developed for non-cancer effects of water-dermal exposure alone will be:

$$SWC_{Derm}\ \{\ mg/L\ \} = RfD * ABS_{GI} / (ABS_{SW} * SW_{Derm}) * CF_{cm3/L} \qquad \textit{Equation \#C-9.}$$

Note that this is a simplified equation because the model assumes all event durations (swimming and wading) are the same. Site-specific application may require the use of different event durations. If event durations are different, ABS_{SW} (below) and SW_{Derm} should be calculated for each assumed exposure duration (ABS_{SW1}, ABS_{SW2}, ..., ABS_{swn}; SW_{Derm1}, SW_{Derm2}, ..., SW_{Dermn}) and *Equation #C-9* becomes:

$$SWC_{Derm}\ \{\ mg/L\ \} = RfD * ABS_{GI} / (ABS_{SW1} * SW_{Derm1} + ABS_{SW2} * SW_{Derm2} +... + ABS_{swn} * SW_{Dermn}\ ) * CF_{cm3/L} \qquad \textit{Equation \#C-9a}$$

Dermal absorption from water is dependent on the event duration and individual chemical characteristics that effect chemical transfer and diffusion through the skin. Equations used to derive dermal exposure relationships are adapted from the EPA Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) (EPA 2001).

The absorbed dose from exposure to dissolved metals would be predicted by:

$$ABS_{SW\text{-}met}\ \{\ mg/(cm^2{\cdot}event))/(mg/cm^3\ \} =\ K_p * ED \qquad \textit{Equation \#C-10.}$$

For organics, dermal absorption from water is adjusted by additional factors to account for the time to equilibrium between chemical dissolved in water and in skin as well as chemical loss due to desquamation. An estimate of the dermally available dose from exposure to organics dissolved in water is calculated from the following equations:

$$ABS_{SW\text{-}org}\ \{\ mg/(cm^2{\cdot}event))/(mg/cm^3\ \} =\ K_p * AF_{ED} \qquad \textit{Equation \#C-11.}$$

Calculations of event duration-dependent factors are chemical specific:

τ { hr/event } = $0.105*10^{(0.0056 * MW)}$ *Equation #C-12.*

β { unitless } = $K_p * \sqrt{MW} / 2.6$ *Equation #C-13.*

If: $\beta \leq 0.6$

then: t^* { hr } = $2.4 * \tau$ *Equation #C-14.*

If: $\beta > 0.6$

then: t^* { hr } = $6 * \tau (b - \sqrt{(b^2 - c^2)})$ *Equation #C-15.*

Where:

$b = 2 * (1 + \beta)^2 / \pi - c$ *Equation #C-16.*

$c = (1 + 3 * \beta + 3 * \beta^2) / (3 * (1 + \beta))$ *Equation #C-17.*

If: $ED \leq t^*$

then: AF_{ED} { hr/event } = $2 * FA * \sqrt{(6 * \tau * ED / \pi)}$ *Equation #C-18.*

If: $ED > t^*$

then: AF_{ED} { hr/event } = $FA* (ED / (1 + \beta) + 2 * \tau * (1 + 3 * \beta + 3 * \beta^2) / (1 + \beta)^2)$

Equation #C-19.

*Calculating protective surface water concentrations for **dermal** exposure **(cancer)***
The screening values developed for the cancer effects of dermal-water exposure alone are:

SWC_{Derm-c} { mg/L } = $(AccptRsk_c / (CSF_{adj} * CSF_c)) * ABS_{GI} / (ABS_{SW} * SW_{Derm-c}) * CF_{cm3/L}$

Equation #C-20.

Note: if site-specific evaluations require multiple event durations, *Equation #C-20* should be adjusted as *Equation #C-9* above.

*Calculating protective surface water concentrations for **inhalation** exposure*
Using inhalation estimates and chemical specific toxicity criteria and partitioning data, the following equation estimates a surface water screening concentration for inhalation of volatile contaminants. This calculation assumes that the concentrations of chemical in surface water and air are at equilibrium: i.e. the amount of chemical volatilizing into air is equal to the amount returning to the surfacewater from air.

SWC_{Inh} { mg/L } = $Dil_{air} * RfC * R * T / (Inh_{frac} * K_H * CF_{L/m3} * CF_{L/m3})$ *Equation #C-21.*

This surface water concentration will be an unobtainable maximum, because the contaminant concentration in air will certainly be diluted with fresh air, and equilibrium between water and air concentrations will not be maintained any distance above the water surface. As a result, much higher surface water contaminant concentrations will be needed to maintain inhalation concentrations approaching inhalation criteria. The

amount will depend on variables such as wind speed, water (and wind) turbulence, presence or absence of white caps, wind direction in relation to the reach of the lake, and the height above the lake surface where one is breathing.

Equation for carcinogens:

$$SWC_{Inh\text{-}c}\ \{\ mg/L\ \} = Dil_{air} * (AccptRsk_c / (CSF_{adj} * UR_c)) * R * T / (Inh_{frac\text{-}c} * K_H * CF_{L/m3} * CF_{L/m3} * CF_{\mu g/mg})$$ *Equation #C-22.*

Activity-exposure only air screening concentration calculations

*Calculating protective air concentrations for **inhalation** exposure **(non-cancer)***

As noted above, using surface water screening concentrations will over estimate risk from inhalation exposure. However, if the surface water screening concentration is exceeded by a large amount, the air concentration may be measured. Air screening concentrations can be calculated using the following equations.

Chronic (non-cancer) screening concentration:

$$SC_{Inh}\ \{\mu g/m^3\} = RfC / Inh_{frac}$$ *Equation #C-23.*

Carcinogen screening concentration:

$$SC_{Inh\text{-}c}\ \{\mu g/m^3\} = (AccptRsk_c / (CSF_{adj} * UR_c)) / Inh_{frac\text{-}c}$$ *Equation #C-24.*

Criteria developed using these equations should be applied judiciously because they will not account for exposures outside of the modeled recreational activity.

Surface water screening concentration calculations

Combined chronic surface water screening values for all routes-of-exposure analyzed in this HC were determined using the following equation calculation.

$$SWC_{ttl}\ \{\ mg/L\ \} = (1/SWC_{Ing} + 1/SWC_{Derm} + 1/SWC_{Inh})^{-1}$$ *Equation #C-25.*

Similarly, surface water screening values for cancer endpoints were calculated with:

$$SWC_{ttl\text{-}c}\ \{\ mg/L\ \} = (1/SWC_{Ing\text{-}c} + 1/SWC_{Derm\text{-}c} + 1/SWC_{Inh\text{-}c})^{-1}$$ *Equation #C-26.*

As noted above, the inhalation route-specific water screening number is extremely conservative because it relies on the maintenance of equilibrium between water and air. Therefore, it may be useful to split the screening water concentration into 2 parts (ingestion+ dermal and inhalation) for evaluation.

Route-of-exposure contribution (percent) calculations

The percent an individual route-of-exposure contributes to the surface water screening value for each chemical for both chronic and cancer endpoints were determined by:

$$SWC_{\%x}\ \{\ \%\ \} = (1/SWC_x) / (1/SWC_{ttl}) * 100$$ *Equation #C-27.*

Appendix D: Cancer Slope Factor, Unit Risk Adjustment for Early Life Exposure

Abbreviations of units, variables and constants are defined in Appendix A - Glossary.

Cancer slope factors (CSFs), as well as Unit Risks (URs) for some carcinogens are adjusted to account for increased potency that is exhibited following early life exposures. The final, published $CSF_{adj\text{-}age}$ (or $UR_{adj\text{-}age}$) is a single CSF (or UR) that reflects a uniform lifetime exposure, but sensitivity that varies with age. The Long Lake site-specific model uses age-adjusted CSFs and URs for both trichloroethylene and vinyl chloride. In addition, the model also assumes that exposures are more frequent during childhood than during adulthood. Therefore the model incorporates an additional adjustment into the CSFs and URs that is described below.

MDH and EPA use cancer risk adjustments to account for the higher risk from exposure to TCE and vinyl chloride in early life. These adjustments are shown in Table D-1. The CSF adjusted for early life sensitivity ($CSF_{adj\text{-}age}$) is calculated from the adult (animal or human) CSF_{study} using the following equation:

$CSF_{adj\text{-}age}$ { $(mg/(kg_{bw}\cdot d)^{-1}$ } = CSF_{study} * (3 (yrs) * 10 {age 0-2 adj} + 13 (yrs) * 3 {age 3-15 adj} + 54 (yrs) * 1 {age 16-70 adj}) / 70 (yrs)

$CSF_{adj\text{-}age}$ / CSF_{study} { unitless } = (3 (yrs) * 10 + 13 (yrs) * 3 + 54 (yrs) * 1) / 70 (yrs)

$CSF_{adj\text{-}age}$ / CSF_{study} { unitless } = 1.757

This expression assumes a similar exposure throughout a lifetime. If site-specific (Long Lake) swimming exposures are averaged out over a 70 year lifetime, as they are using the EPA age-adjustment cancer model, it is assumed that every year there are 56.4 exposures (Table D-1). However, the lifetime exposure scenario for Long Lake suggests 155 swimming events per year from 1 through 17 years old; 77.4 swimming events per year from 18 through 32 years old; and no exposures from 33 years old on (Table D-1). Therefore to compensate for greater exposure during the periods of increased cancer risk, the following equation was used for this site-specific screening assessment:

$CSF_{adj\text{-}event}$ { $(mg/(kg_{bw}\cdot d)^{-1}$ } = CSF_{study} * ($events_{0\text{-}2}$ * 10 + $events_{3\text{-}15}$ * 3 + $events_{16\text{-}70}$ * 1) / $events_{ttl}$

$CSF_{adj\text{-}event}$ / CSF_{study} { unitless } = 2.797

and

$CSF_{adj\text{-}event}$ { unitless } = $CSF_{adj\text{-}age}$ * 2.797 / 1.757 = $CSF_{adj\text{-}age}$ * 1.592

Table D-1: CSF and UR Adjustment

	Ages (years-old)	Years	Early Life Exposures Multiplier (US EPA, 2005)	Site Specific Exposures		
				Exposure (Years)	Events per year	Age-Adjusted Events
	0-2	3	10	2	155	3100
	3-15	13	3	13	155	6045
	16-17	2	1	2	155	310
	18-32	15	1	15	77.4	1161
	33-70	37	1			
Totals	**0-70**	70		32	56.4 (70 years)	10616

Note: The Long Lake CSF/UR exposure adjustment was only calculated using swimming event exposures. Wading events accounted for only about 2%, 2% and 16% of the modeled chemical exposures from ingestion, dermal and inhalation, respectively.

Appendix E: Discussion of the relationship between inhalation, ingestion and dermal exposures in Long Lake

Abbreviations of units, variables and constants are defined in Appendix A - Glossary.

It is not possible, with surface water concentration data alone, to accurately assess the potential health impact from a volatile organic compound (VOC) in surface water. This is because it is difficult to characterize the inhalation exposure to VOCs that evaporate from the water. Unlike exposure to non-volatile contaminants, inhalation is an important exposure pathway for most VOCs.

The concentration of a VOC in air overlying surface water can fluctuate rapidly and can vary over many orders of magnitude. Therefore, it is very difficult to estimate air concentrations over a lake or inhalation exposure to an individual swimming in the lake. This Appendix discusses the impact of different air concentrations on an exposure model, and demonstrates the effects of contaminant dilution by air that is moving over the water surface.

The Minnesota Department of Health previously developed a Sediment Screening Model (SSM). The SSM was developed to calculate Sediment Screening Values for the US Steel site in Duluth, MN and is described in the Sediment Screening Value Memo (MDH 2005). The SSM generally uses relationships and equations established in EPA Risk Assessment Guidance for Superfund (RAGS: USEPA 1989; 1994; 2004). For site-specific reasonable maximum wading, swimming and fish consumption exposures to sediments, the SSM calculates protective criteria for chemical exposures based on the predicted chemical/physical partitioning of the contaminants, contaminant bioaccumulation in fish and contaminant toxicity.

For the assessment in this Health Consultation, the SSM was adopted to model of the relationship between inhalation, ingestion and dermal exposures to contaminants in Long Lake. The modified SSM calculated protective surface water and inhalation criteria for site-specific exposures to trichloroethylene (TCE), *cis*-1,2-dichloroethylene (DCE), and vinyl chloride. Equations used in this model are shown in Appendix C.

The site-specific application of this model does not account for the potential exposure to these chemicals outside of the identified scenarios. In addition, the site-specific application does not provide for exposure to different contaminants with similar endpoints. At highly contaminated sites where the overall risk or hazard approaches levels of concern, a hazard quotient evaluation and/or the inclusion of a relative source contribution may be useful.

Toxicity criteria

Different routes of exposure may result in different sensitivity to toxicants, different internal doses, as well as different toxic endpoints. Toxicity criteria for inhalation, ingestion, and dermal exposures may be found in Table 2 of the main text.

Inhalation exposure criteria

Using the SSM, calculated inhalation (air) screening concentrations for TCE, DCE and vinyl chloride (using chemical-specific toxicity data from Health Consultation, Table 2; site-specific exposure data from Health Consultation, text and Table 3) are 330 $\mu g/m^3$, 2600 $\mu g/m^3$, and 120 $\mu g/m^3$, respectively. TCE and vinyl chloride screening concentrations are based on a calculated incremental upper-bound estimate of lifetime cancer risk of 1×10^{-5}, and the DCE screening concentration (chronic exposure) is based on non-cancer hazard evaluation, using TCE inhalation toxicity as a conservative surrogate for DCE inhalation toxicity. Equations C-5, C-6 and C-24 (Appendix C) were used to calculate the air screening concentrations for TCE and vinyl chloride; Equations C-5 and C-23 (Appendix C) were used to calculate the air screening concentration for DCE. For exposure to TCE or vinyl chloride to be an inhalation concern for someone exposed at a reasonable maximal exposure (RME; for this site defined in Health Consultation, Table 3) for many years, concentrations in air (exposure concentrations) would need to be consistently greater than 330 $\mu g/m^3$ and 120 $\mu g/m^3$, respectively.

To achieve these concentrations in an enclosed space or in still air, the Henry's Law equilibrium constants for TCE, DCE and vinyl chloride (used to calculate equilibrium air concentrations from water concentrations in the SSM) suggest that concentrations of these chemicals in water can be fairly low. But to reach these air concentrations when there is a wind, when dilution is relatively high, the contaminant concentration in water needs to be much higher. This presents a problem for the model: determining realistic air exposures given the uncertain but large dilution that takes place in the outdoor environment.

The Henry's Law partitioning constant was used in the SSM to predict the equilibrium concentration of a contaminant in water from a concentration of the contaminant in air because the model is simple (equations C-21 or C-22). A fugacity model has also been used to estimate concentrations in air and water. (A fugacity model is based on the chemical potential in different model compartments and the flux between compartments, and can be used to calculate stable, steady-state conditions under different environmental conditions.) The results of the fugacity analyses under calm conditions (i.e. diffusive transport predominating) were similar to the equilibrium evaluation conducted here (MPCA 2006). These models (without refined data on relationships between concentration ¼ - 1 foot above the lake surface; windspeed at, typically, 10 meters above the lake; and water turbulence, including the presence of waves and white caps) will always overestimate air concentrations. This is because the air compartment is essentially infinite in size and mixing within this compartment will be determined by wind speed which is highly variable. In addition, even in very calm conditions the unmixed layer of air (layer in which equilibrium occurs) overlying the lake will be very small.

At equilibrium with the inhalation concentrations of concern shown previously (TCE, 330 µg/m^3; DCE, 2600 µg/m^3; vinyl chloride, 120 µg/m^3), water concentrations for TCE, DCE and vinyl chloride would be about 0.80 µg/L, 15 µg/L and 0.11 µg/L, respectively (equations C-21 and C22). Table E-1 shows the relationship between air concentration and water concentration for TCE, DCE and vinyl chloride with different air-dilution factors. (Dil_{air} = 1, 50, 500, 5000.)

Table E-1: Examples of possible media concentrations at different dilutions

Chemical (VOC)	Air Dilution (n-fold)	Constant air concentration		Constant water concentration	
		Air	Water	Air	Water
		ug/m3	ug/L	ug/m3	ug/L
TCE	Equilibrium	330	0.8	330	0.8
	50		40	6.6	
	500		400	0.66	
	5000		4000	0.066	
DCE	Equilibrium	2600	15.0	2600	15.0
	50		750	52	
	500		7500	5.2	
	5000		75000	0.52	
Vinyl Chloride	Equilibrium	120	0.11	120	0.11
	50		5.5	2.4	
	500		55	0.24	
	5000		550	0.024	

Values were calculated using the MDH Sediment Screening Model (MDH 2005).

When exposures to trichloroethylene (TCE), *cis*-1,2-dichloroethylene (DCE) and vinyl chloride are evaluated assuming equilibrium concentrations between water and air, exposure to the contaminants by inhalation is the primary route of exposure. However in the real environment, for instance at Long Lake, equilibrium conditions would only be approached in extremely calm conditions, and very close to the water surface (e.g. swimming). Otherwise, dispersion and mixing of the contaminants with air would rapidly decrease the contaminant concentration. Nevertheless, consideration of equilibrium conditions is valuable for conceptualizing an upper limit.

Estimating a contaminant's concentration in the air above the surface of the Lake is difficult, and MDH is not aware of studies that may have characterized similar conditions. Although it is easy to calculate equilibrium concentrations, equilibrium conditions (chemical equilibrium between chemical in water and chemical in air) rarely, if ever, occur over a lake. Small disturbances or a slight breeze will cause considerable mixing of air above a lake. As a result, typical air concentrations over a lake will be well below those expected to occur at equilibrium.

Estimating relative ingestion, dermal and inhalation exposure

Parameters used to evaluate ingestion, dermal and inhalation exposures are described in the main text.

Note, from Table E-1, that to maintain the inhalation concentrations of concern (TCE 330 µg/m^3; DCE 2600 µg/m^3; vinyl chloride 120 µg/m^3) when the air is being diluted 500-fold, water concentrations for TCE, DCE and vinyl chloride would need to be maintained at 400 µg/L, 7500 µg/L and 55 µg/L, respectively. Under these conditions, ingestion and dermal exposure would present a serious health hazard (equations C-1 through C-4 and C-7 through C-20). Table E-2 shows the contribution of different routes of exposure (equation C-27) at equilibrium (exposure conditions that considerably overestimate most, if not all, actual exposure conditions), and at different amounts of dilution of air up to 5000-fold. These conditions would likely be reasonable for different wind conditions. Note that the inhalation contribution decreases with dilution, and the ingestion and dermal contribution increase.

Table E-2: Relative contribution by route-of-exposure for different air-mixing (dilution) conditions

Air Dilution (n-fold)	Route of Exposure Contribution to Cancer Risk			Route of Exposure Contribution to Health Hazard		
	Ingestion	Dermal	Inhalation	Ingestion	Dermal	Inhalation
Equilibrium	0.13%	0.21%	**100%**	0.00%	0.00%	**100%**
50	**5.6%**	**9.1%**	**85%**	**0.2%**	**0.2%**	**100%**
100	**9.7%**	**16%**	**75%**	0.4%	0.4%	**99%**
500	**24%**	**39%**	**37%**	**2.0%**	**1.7%**	**96%**
1000	**29%**	**48%**	**23%**	**3.8%**	**3.3%**	**93%**
5000	**36%**	**59%**	**6%**	**15%**	**13%**	**72%**
Equilibrium				0.1%	0.04%	**100%**
50				**3.0%**	**2.1%**	**95%**
100				**5.8%**	4.0%	**90%**
500				**21%**	**15%**	**65%**
1000				**31%**	**22%**	**48%**
5000				**50%**	**35%**	**16%**
Equilibrium	1.1%	0.56%	**98%**	0.12%	0.03%	**100%**
50	**31%**	**15%**	**54%**	**5.5%**	**1.5%**	**93%**
100	**42%**	**21%**	**37%**	**10%**	2.7%	**87%**
500	**60%**	**29%**	**10%**	**34%**	**8.9%**	**57%**
1000	**63%**	**31%**	**5.5%**	**48%**	**13%**	**40%**
5000	**66%**	**33%**	1.2%	**70%**	**18%**	**12%**

Greater than 5% exposure contribution

Values were calculated using equations in Appendix C with exposures at the site-specific RME (see Health Consultation).

Table E-2 shows the relative importance of different routes of exposure for determining cancer risk and non-cancer hazard under different air mixing conditions (air dilution). Note that for cancer risk, ingestion becomes the most important route of exposure for vinyl chloride when there is about 100-fold air dilution; and for TCE, dermal exposure exceeds inhalation at about 500-fold air dilution.

If the VOC concentration in water is low (e.g. Long Lake TCE water concentrations), then the ingestion and dermal exposure contribution to the overall risk will be minimal and can be ignored. At low enough water concentrations, it is unlikely that air concentrations will reach levels of concern because equilibrium conditions would need to be maintained. As the water concentration increases, both the inhalation exposures and the ingestion and dermal exposures will increase. In addition, at a higher water concentration the importance of the ingestion and dermal exposure pathways increases, regardless of the air concentrations. It is likely that the air concentration above a lake will typically be diluted 100-fold or more in air above a lake (personal conversation with G. Pratt, MPCA). Therefore, it may be reasonable to evaluate surface water exposures using exposures that reflect dilution factors around 100. For vinyl chloride, evaluating at this dilution will mean that inhalation is responsible for about 40% of the total exposure, and ingestion and dermal exposure become the most important routes of exposure. However, for TCE inhalation appears to be important at dilutions up to 500-fold.

Table E-3 shows estimated air and water screening concentrations for cancer risk and non-cancer hazard evaluations assuming different air dilutions. These values were calculated using the modified SSM (equations C-23 through C-26).

Table E-3: Air and Water Screening Concentrations for different air dilutions

		Cancer Risk Evaluation		Non-Cancer Hazard Evaluation	
Chemical (VOC)	Air Dilution (n-fold)	Screening Air Concentration	Screening Water Concentration	Screening Air Concentration	Screening Water Concentration
		ug/m3	ug/L	ug/m3	ug/L
TCE	Equilibrium	330	0.8	2600	6.3
	50	6.6	34	52	310
	100	3.3	60	26	630
	500	0.66	150	5.2	3000
	1000	0.33	180	2.6	5900
DCE *	Equilibrium			2600	15.0
	50			52	720
	100			26	1400
	500			5.2	4900
	1000			2.6	7300
Vinyl Chloride	Equilibrium	120	0.11	1300	1.10
	50	2.5	2.9	25	51
	100	1.2	3.9	13	96
	500	0.25	5.6	2.5	310
	1000	0.12	5.9	1.3	440

* TCE non-cancer inhalation toxicity criterion used as conservative surrogate for DCE inhalation criterion
Values were calculated using the MDH Sediment Screening Model (MDH 2005) with exposures at the site-specific RME (see Health Consultation). A relative source contribution factor of 0.2 was used for calculating non-cancer screening concentrations.

Conclusion

As dilution of the contaminant concentration in air increases, the importance of the ingestion and dermal routes of exposure increase. A health assessment of the ingestion

and dermal routes of exposure to the VOCs found in Long Lake can be conducted with the available water concentration data. However, it is not possible to accurately estimate the inhalation exposure from water concentration data. If typical air concentration data were available, inhalation risk could be calculated as well. But there are no site-specific air data from near the surface of the Lake. In addition, reliable models estimating the mixing or dilution of air near a swimmer under different meteorological conditions are not available.

Inhalation exposure may be the biggest contaminant exposure occurring when someone swims in Long Lake. However, it is clear that considerable mixing or dilution of VOCs evaporating from the Lake will keep the overlying air concentrations well below equilibrium with water concentrations.

If the TCE concentration in Long Lake is below 0.80 µg/L, the lifetime incremental cancer risk from exposure (ingestion, dermal and inhalation) will be below 10^{-5} regardless of the concentration in air or the inhalation exposure. If the TCE concentration in Long Lake is below 34 µg/L and the inhaled air for 32 years of exposure at the RME is diluted at least 50-fold from equilibrium, the lifetime incremental cancer risk from exposure will be below 10^{-5}. The inhalation exposure to TCE does not become unimportant until the water concentration is greater than 150 µg/L and the calculated risk from ingestion and dermal exposure approaches 10^{-5}. (If the largest oral cancer slope factor from the draft EPA assessment is used in the model, TCE concentration in Long Lake averages below 9.4 µg/L, and dilution is 50-fold or greater, the lifetime incremental cancer risk from exposure will be below 10^{-5}.)

Similarly, if the vinyl chloride concentration in Long Lake is below 0.11 µg/L, the lifetime incremental cancer risk from exposure (ingestion, dermal and inhalation) will be below 10^{-5} regardless of the concentration in air or the inhalation exposure. If the vinyl chloride concentration in Long Lake is below 2.9 µg/L and the inhaled air for 32 years of exposure at the RME is diluted at least 50-fold from equilibrium, the lifetime incremental cancer risk from exposure will be below 10^{-5}. The inhalation exposure to vinyl chloride does not become unimportant until the water concentration approaches 4-5 µg/L and the calculated risk from ingestion and dermal exposure approaches 10^{-5}.

CERTIFICATION

This Long Lake Trichloroethylene Contamination Health Consultation was prepared by the Minnesota Department of Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the health consultation was begun. Editorial review was completed by the Cooperative Agreement partner.

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

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