Drinking Water Quality: Tetrachloroethene (PCE) Contaminant Levels

Type of EPHT Indicator	Hazard, Exposure
Measures	Level of Contaminant in Finished Water 1. Yearly distribution of number of Community Water Systems (CWS) by maximum PCE concentration (cut-points: 0-<1, 1-<2, 2-<5, 5+ µg/L PCE). 2. Yearly distribution of number of CWS by mean PCE concentration (cut-points: 0-<1, 1-<2, 2-<5, 5+ µg/L PCE). 3. Average Concentration of PCE, by Year. Potential Population Exposure to Contaminants in Finished Water 4. Yearly distribution of number of people served by CWS by maximum PCE concentration (cut-points: 0-<1, 1-<2, 2-<5, 5+ µg/L PCE). 5. Yearly distribution of number of people served by CWS by mean PCE concentration (cut-points: 0-<1, 1-<2, 2-<5, 5+ µg/L PCE).
Derivation of Measures	PCE measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	PCE, µg/L
Geographic Scope	State and Community Water System
Geographic Scale	The finest detail will be the approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	2000-Most Recent Year Available
Time Scale	Calendar year
Rationale	**Tetrachloroethene (PCE) and Public Health** Tetrachloroethene (PCE) is a volatile halogenated short-chain hydrocarbon. Tetrachloroethene is used in dry cleaning, metal cleaning, the synthesis of other chemicals, and household products such as water repellants, silicone lubricants, and spot removers. PCE is produced and used in high volumes in the U.S. and has been detected in urban and ambient air and occasionally in soils and drinking water most likely contaminated by industrial discharge (Moran et al., 2007; Rowe et al., 2007). Because of its volatility, this solvent does not persist in the soil or water following the discontinuation of contamination. Inhalation is the most common exposure route for the general population including indoor sources from paints, adhesives, and cleaning solutions. Volatilization from contaminated water (e.g., shower water) as well as the use of household products containing this solvent can result in higher indoor than outdoor air concentrations (ATSDR, 1997; Martin et al.,

	2005). Nearby dry cleaning establishments, industries producing PCE, and contaminated waste disposal sites can also contribute to human exposure (Armstrong and Green, 2004; ATSDR, 1997 and 2000; Schreiber et al., 1993; Wallace et al., 1991). Drinking water may contribute to exposure when underground drinking water supplies have been contaminated. Workers in industries such as dry cleaning, aircraft maintenance, electronics manufacturing, and chemical production may be exposed by inhalation or by dermal contact with PCE. The EPA has established drinking water standards and other environmental standards for PCE, and the FDA regulates PCE and trichloroethene as indirect food additives. Workplace standards have been established by OSHA, and ACGIH has recommended occupational guidelines and biological exposure indices for monitoring workers. Human health effects from PCE at low environmental doses or at biomonitored levels from low environmental exposures are unknown. PCE is well absorbed by ingestion and inhalation, and animal studies have demonstrated that liquid forms can be dermally absorbed. Following absorption, part of the solvent dose is excreted into expired air; for PCE, about 97-99% of the dose is eliminated unmetabolized into expired air, though it has an elimination half-life of several days (ATSDR 1997; Monster, 1986). The retained solvent can undergo hepatic metabolism. PCE is metabolized to trichloroacetic acid and trichloroethanol, which are eliminated in the urine. Accidental or intentional high dose acute exposure by ingestion or inhalation can result in loss of motor coordination, somnolence, and unconsciousness. Inhaling high doses of PCE may also produce cardiac arrhythmias attributed to enhanced sensitivity to catecholamines. High dose acute exposure to PCE has resulted in reversible kidney impairment, and prolonged, low level PCE exposure has been associated with altered renal enzyme excretion and liver enlargement (ATSDR, 1997). Chronic occupational exposure to PCE may be associated with mild degrees of neurological impairments, including reaction times, verbal skills, cognitive ability, and motor function (Armstrong and Green, 2004). Various epidemiologic studies of chronic PCE exposure in dry cleaning workers found increased incidences of esophageal and cervical cancers and non-Hodgkins lymphoma, but confounding exposures (e.g., other solvents and trichloroethene) were likely (IPCS, 2006). In animal studies, PCE-induced kidney and liver tumors and caused leukemia (IARC, 1995). IARC classifies PCE as a probable human carcinogen, and NTP classifies it as reasonably anticipated to be a human carcinogen (IARC, 1995; NTP, 2004). Additional information about these solvents is available from ATSDR at: http://www.atsdr.cdc.gov/toxprofiles/index.asp. In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, PCE was detected in 1,262 systems serving close to 32 million people (EPA, 2009). Concentrations of PCE were greater than the MCL in 241 systems serving close to 15 million people. PCE was the fifth highest occurring regulated volatile organic chemical found based on the percent of detections found from the 6 Year Review data (EPA, 2009). **Biomonitoring Information** Levels of halogenated solvents in blood reflect recent exposure. In the NHANES 2003-2004 subsample, the level of blood PCE for adults at the 75th percentile of the U.S. population appear similar to the levels at the 75th percentile reported for non-smoking adults in a subsample of NHANES 1999-2000 participants (CDC, 2009; Lin et al., 2008) and were similar or slightly less than levels reported in a nonrepresentative subsample of the earlier NHANES III (1988-1994) (Ashley et al., 1994; Churchill et al., 2001). A recent study of low income, urban children in the Midwest reported slightly lower median PCE levels (Sexton et

	al., 2005; Sexton et al., 2006) than the NHANES III levels (Ashley et al., 1994; Churchill et al., 2001). Comparatively higher blood levels of PCE and trichloroethene have been noted for urban and industrial residential settings than for rural settings (Barkley et al., 1980; Begerow et al., 1996; Brugnone et al., 1994). Residing near dry-cleaning facilities or storing recently dry-cleaned clothes at home can contribute to increased blood PCE levels (Begerow et al., 1996; Popp et al., 1992). In contrast, PCE blood levels in occupationally exposed workers have been reported to be many thousand times higher than the general population (Begerow et al., 1996; Furuki et al., 2000; Monster et al., 1983). The occupational biological exposure index associated with an 8-hour exposure of 25 ppm is 500 µg/L PCE in blood (ACGIH, 2007). Non-occupational exposures are usually well below this level. Finding a measurable amount of any of these solvents in blood does not mean that the level of the solvent causes an adverse health effect. Biomonitoring studies of blood halogenated solvents can provide physicians and public health officials with reference values so that they can determine whether or not people have been exposed to higher levels of halogenated solvents than levels found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects. **Sources of PCE** The major source of PCE in drinking water is discharge from factories and dry cleaners. A federal law called the Emergency Planning and Community Right to Know Act requires facilities in certain industries, which manufacture, process, or use significant amounts of toxic chemicals, to report annually on their releases of these chemicals. For more information on the uses and releases of chemicals in your state, contact the Community Right-to-Know Hotline: (800) 424-9346 (EPA, 2010). **PCE Regulation and Monitoring** The EPA limits the amount of PCE that may be present in drinking water to 5 parts of PCE per billion parts of water (5 ppb), or 5 ug/L.
Use of Measure	These measures can assist by addressing the following surveillance functions: • Distribution measures provide information on the number of CWS and the number of people potentially exposed to PCE at different concentrations. • Maximum concentrations provide information on the peak potential exposure to PCE at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	The current measures are derived for CWS only. Private wells may be another source of population exposure to PCE. Transient non-community water systems, which are regulated by EPA, also may be an important source of PCE exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	Iowa Department of Natural Resources
Limitations of Data Sources	Ground water systems may have multiple wells with different PCE concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the

	distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the PCE concentration of people served by wells with higher PCE concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different PCE levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use
References	1. ACGIH. TLVs and BEIs Based on the documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. 2007. Signature Publications. Cincinnati OH. p.104. 2. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for tetrachloroethylene update. 1997 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=265&tid=48. 4/22/09 3. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for Tetrachloroethylene update. 2000 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=234&tid=42. 4/22/09 4. Armstrong SR, Green LC. Chlorinated hydrocarbon solvents. Clin Occup Environ Med 2004;4(3):481-496. 5. Ashley DL, Bonin MA, Cardinali FL, McCraw JM, Wooten JV. Blood concentrations of volatile organic compounds in a nonoccupationally exposed US population and in groups with suspected exposure. Clin Chem 1994;40(7 Pt 2):1401-1404. 6. Barkley J, Bunch J, Bursey JT, Castillo N, Cooper SD, Davis JM, et al. Gas chromatography mass spectrometry computer analysis of volatile halogenated hydrocarbons in man and his environment—a multimedia environmental study. Biomed Mass Spectrom 1980;7(4):139-147. 7. Begerow J, Jermann E, Keles T, Freier I, Ranft U, Dunemann L. Internal and external tetrachloroethene exposure of persons living in differently polluted areas of Northrhine-Westphalia (Germany). Zentralbl Hyg Umweltmed. 1996;198(5):394-406. 8. Brugnone F, Perbellini L, Guiliari C, Cerpelloni M, Soave M. Blood and urine concentrations of chemical pollutants in the general population. Med Lav 1994; 8(5):370-389. 9. Centers for Disease Control and Prevention (CDC). Fourth National Report on Human Exposure to Environmental Chemicals, 2009. Available at: http://www.cdc.gov/exposurereport/pdf/FourthReport.pdf 10. Churchill JA, Ashley DL, Kaye WE. Recent chemical exposures and blood volatile organic compound levels in a large population- based sample. Arch Environ Health 2001; 56(2):157-166. 11. Furuki K, Ukai H, Okamoto S, Takada S, Kawai T, Miyama Y, Mitsuyoshi K, et al. Monitoring of occupational exposure to tetrachloroethene by analysis for unmetabolized tetrachloroethene in blood and urine in comparison with urinalysis for trichloroacetic acid. Int Arch Occup Environ Health. 2000; 73(4):221-227. 12. International Agency for Research in Cancer (IARC). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals Vol. 63, 1995. Available at: http://monographs.iarc.fr/ENG/Monographs/vol63/mono63.pdf 13. International Programme on Chemical Safety (IPCS). Concise International Chemical Assessment Document 68-Tetrachloroethene. 2006 [online]. Available at URL: http:// www.inchem.org/documents/cicads/cicads/cicad68.htm. 4/22/09 14. Lin YS, Egeghy PP, Rappaport SM. Relationships between levels of volatile organic compounds in air and blood from the general population. J Exp Sci Environ Epidemiol 2008; 18:421-429. 15. Martin SA, Simmons MB, Ortiz-Serrano M, Kendrick C, Gallo A, Campbell J, et al. Environmental exposure of a community to airborne trichloroethylene. Arch Environ

Occup Health 2005; 60(6):341-316.

16. Monster AC. Biological monitoring of chlorinated hydrocarbon solvents. J Occup Med 1986; 28:583-588.
17. Monster AC, Regouin-Peeters W, Van Schijndel A, van der Tuin J. Biological monitoring of occupational exposure to tetrachloroethene. Scand J Work Environ Health 1983; 9:273-281.
18. Moran MJ, Zogorski JS, Squillace PJ. Chlorinated solvents in groundwater of the United States. Environ Sci Technol 2007; 41:74-81.
19. National Toxicology Program (NTP). Report on Carcinogens, 12th ed. 2010. [online]. Available at URL: http://ntp.niehs.nih.gov/ntp/roc/twelfth/ListedSubstancesReasonablyAnticipated.pdf 5/9/2013.
20. Popp W, Muller G, Baltes-Schmitz B, Wehner B, Vahrenholz C, Schmieding W, et al. concentrations of tetrachloroethene in blood and trichloroacetic acid in urine in workers and neighbours of dry-cleaning shops. Int Arch Occup Environ Health 1992; 63:393- 395.
21. Rowe BL, Toccalino PL, Moran MJ, Zogorski JS, Price CV. Occurrence and potential human-health relevance of volatile organic compounds in drinking water from domestic wells in the United States. Environ Health Perspect 2007; 115(11):1539-1546.
22. Schreiber JS, House S, Prohonic E, Smead G, Hudson C, Styk M, et al. An investigation of indoor air contamination in residences above dry cleaners. Risk Anal 1993; 13(3):335-344. Sexton K, Adgate JL, Church TR, Ashley DL, Needham LL, Ramachandran, et al. Children's exposure to volatile organic compounds as determined by longitudinal measurements in blood. Environ Health Perspect 2005; 113(3):342-349.
23. Sexton K, Adgate JL, Fredrickson AL, Ryan AD, Needham LL, Ashley DL. Using biologic markers in blood to assess exposure to multiple environmental chemicals for inner-city children 3-6 years of age. Environ Health Perspect 2006; 114(3):453-459.
24. U.S. Environmental Protection Agency (U.S. EPA). Basic Information about Tetrachloroethylene in Drinking Water, 2010. Available at:
25. http://water.epa.gov/drink/contaminants/basicinformation/tetrachloroethylene.cfm
26. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.
27. Wallace L, Nelson W, Ziegenfus R, Pellizzari E, Michael L, Whitmore R, et al. The Los Angeles TEAM Study: Personal exposures, indoor-outdoor air concentrations, and breath concentrations of 25 volatile organic compounds. J Exp Anal Environ Epidemiol 1991; 1(2):157-192.