

## **Perchloroethylene (PCE) in Dry Cleaning Establishments**

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### **Abstract**

Perchloroethylene (PCE) is a chlorinated solvent used mainly in the dry cleaning industry in the United States. Although production of the chemical is decreasing, its persistence in the environment makes it a significant groundwater and air pollutant. In humans, PCE causes toxic effects in the liver, kidney, and central nervous system. It is suspected to cause harm to reproductive and developmental health, and is a possible carcinogen. The major human exposure route is inhalation of contaminated air. Efforts are underway to reduce the use of PCE in the dry cleaning industry by development of green technologies. Until suitable alternatives to PCE can be found, we must rely on remediation efforts to clean up environmental releases and limit our exposure from point sources as dry cleaning.

## Introduction

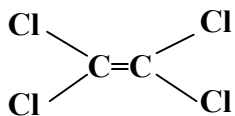


Figure 1. Perchloroethylene.

Perchloroethylene (PCE) is a chlorinated aliphatic solvent (Figure 1). It is also known as *perc*, or by its IUPAC name tetrachloroethene. Its Chemical Abstract Service (CAS) Registry Number is 127-18-4. It exists as a nonflammable, colorless liquid (Budavari, 1989). If one of the chlorine atoms is replaced with hydrogen, the molecule becomes trichloroethylene (TCE), or trichloroethene by IUPAC. TCE's CAS number is 79-01-6. PCE is more stable than TCE due to the total chlorination of the ethene molecule (Watts, 1996). Since the compounds are similar, and through degradation are usually found together, some information on TCE will also be presented.

## Discussion

### A) Sources

Production of PCE in the United States has been steadily decreasing. In 1978 736 million pounds of the chemical were produced (U. S. EPA 1998), in 1991, 310 million pounds were produced. Between 1989 and 1991 there had been a 35% drop in demand for the chemical due to increased solvent recycling and lower production of a derivative of PCE, freon (U. S. EPA, 1994). This decrease was expected to continue, and in fact the annual production of PCE in 1994 was down to 246 million pounds (Schettler, et al, 1999). Three companies produce PCE in the United States. Dow Chemical, PPG Industries, and Vulcan Chemicals operate in Louisiana, and Vulcan also operates in Kansas (U. S. EPA, 1994). The production rates for TCE were between 200

million and 400 million pounds per year as of 1995 (Watts, 1996). PCE is derived from petroleum distillate, specifically through ethane (Crosby, 1998).

Due to its excellent solvent characteristics, 60% of PCE's application is in dry cleaning, where it is used by about 80 % of the industry (U. S. EPA, 1994; Watts, 1996). PCE is attractive as a dry cleaning agent for the following reasons: it is safe on textiles, fibers, and dyes; has no residual odors; is chemically stable under the conditions in which it is used; is noncorrosive to machinery; is easily removed from clothing; and is effective for removing fats, oils, and greases (HSIA, 1999). Other applications for PCE include vapor degreasing, machining, manufacture of chlorofluorocarbons, auto paint, assembly plants, and electroplating. Particular consumer products containing PCE are typewriter correction fluid and shoe polish. Since TCE is also an excellent solvent, its uses are similar: vapor degreasing; textile processing; refrigerant; in stains, finishes, lubricants, adhesives, and rug cleaners; and in the production of vinyl chloride, pharmaceuticals, and insecticides (Schettler, et al, 1999). The major use for TCE, about 80%, is in vapor degreasing (Watts, 1996).

## B) Pathways

If PCE is released to soil, some will evaporate quickly, and some may leach to groundwater. The water solubility of PCE is 275 mg/L, and 1310 mg/L for TCE. The specific gravities of PCE and TCE are 1.623 and 1.464. These water solubilities and high specific gravities make the chemicals dense, nonaqueous phase liquids (DNAPLs). The mean log octanol-water partition coefficients ( $\log K_{ow}$ s) for PCE and TCE are 2.79 and 2.33, predicting moderate hydrophobicity and adsorption to soil (Watts, 1996).

DNAPL pools in the subsurface can be particularly difficult to treat because they can be resistant to pumping, especially if they become trapped in stratigraphic layers. This immobile pool would then become a long-term contamination source for groundwater (Dawson and Roberts, 1997).

The log  $K_{ow}$  values presented above are not high enough to preclude the chemicals from migrating through groundwater systems. PCE and TCE were two of the most commonly detected organic chemicals in a study of 7000 wells in California from 1984 to 1988 (Watts, 1996). TCE in particular is the most common organic contaminant in groundwater, appearing in one-tenth to one-third of all samples tested in the United States (Schettler, 1999). PCE can also find its way into drinking water by leaching from vinyl liners in water distribution pipelines and during chlorination treatment of water (Paulu, et al, 1999; U. S. EPA, 1998).

The vapor pressures of PCE and TCE are 14 and 58 mm Hg at 20°C, and the Henry's Law constants are 0.0153 and 0.0091 atm-m<sup>3</sup>/mole at 25°C. These values show a high tendency for volatilization (Watts, 1996). TCE is a common indoor air pollutant, due to its use in building materials (Wallace, et al, 1987). The vapor density values of PCE and TCE are 5.8 and 4.5; suggesting that the volatilized compounds will stay close to the ground (Watts, 1996). PCE and TCE in the atmosphere may find their way back to the aqueous phase by washout in rain (U. S. EPA, 1998).

The United States Environmental Protection Agency (EPA) conducted the Total Exposure Assessment Methodology (TEAM) study in the 1980's to attempt to quantify peoples' total daily exposures to several chemicals, including PCE and TCE. Six hundred people in four states were studied for 24 hours with personal exposure

monitoring. The TEAM study found that indoor air concentrations of contaminants exceeded outdoor air concentrations by at least a factor of two. TCE in particular was found in around 85% of the personal air monitoring samples (Schettler, 1999).

Shown in Table 1 are the amounts of PCE and TCE that find their way into different compartments of the environment in the United States each year.

Environmental Compartment	PCE (pounds)	TCE (pounds)
Air	7,861,170	21,272,166
Surface water	1,311	541
Groundwater	13,436	1,291
Land	30,442	23,140
Off-site releases	22,071	76,327
Total releases	7,928,430	21,373,465

Table 1. Annual releases of PCE and TCE (Schettler, 1999).

Inspection of the PCE molecule shows the oxidation state of each carbon atom to be (+II), or oxidized. The oxidation state suggests a tendency to be reduced, and PCE

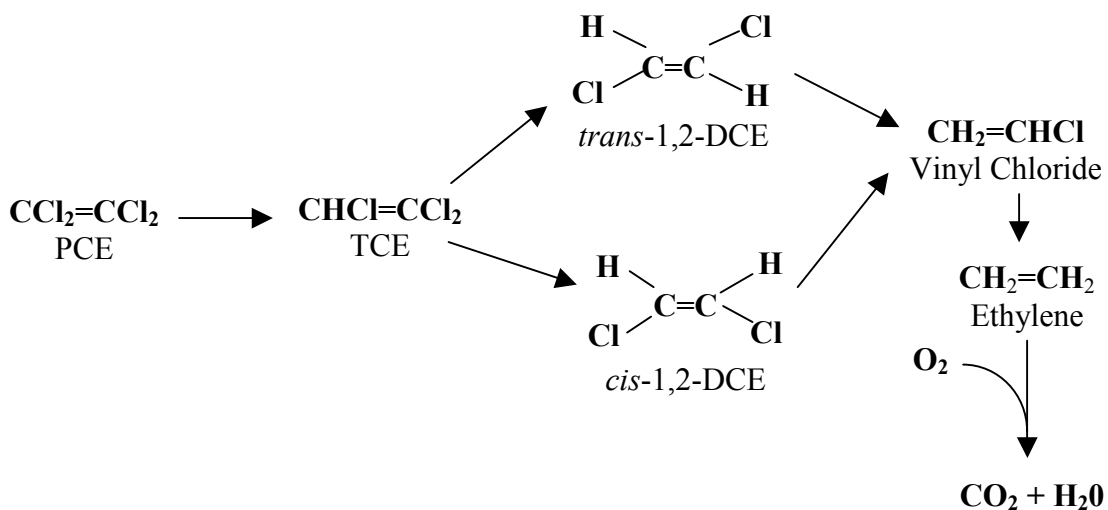


Figure 2. Reductive dehalogenation of PCE (Vogel and McCarty)

is degraded anaerobically by reductive dehalogenation. No organism is known that can degrade PCE aerobically. Reductive dehalogenation does not occur in nature unless the microorganisms capable of halorespiration are present, and then only if electron donors are present. The availability of an electron donor is the rate-limiting step for the reaction. The donor, or cometabolite, is oxidized, with PCE operating as the terminal electron acceptor. By this method, PCE is reduced to TCE, and the microorganism gains energy for growth (McCarty, 1997). The process continues with TCE reducing to DCE, then vinyl chloride, ethylene, and with products of carbon dioxide and water (Figure 2). PCE and TCE can persist in the environment for years due to the lack of available electron donors for this pathway. Reductive dehalogenation is stimulated in remediation efforts by the addition of an electron donor (McCarty, 1997).

PCE and TCE can also be oxidized. The second-order rate constants for reactivity with hydroxyl radicals for PCE and TCE are  $2.8 \times 10^9$  and  $4.0 \times 10^9 \text{ M}^{-1}\text{sec}^{-1}$  (Watts, 1996). These values suggest rapid photooxidation for atmospheric PCE, during daylight hours when hydroxyl radicals are formed (Crosby, 1998). An estimated half-life for PCE in the atmosphere is 96 days, and the degradation products are phosgene and chloroacetylchlorides (ATSDR, 1993). These products contribute to the production of photochemical smog (Crosby, 1998). Although the major loss of PCE from surface waters is volatilization, photooxidation of PCE also occurs in surface waters exposed to sunlight, forming trichloroacetic acid (U. S. EPA, 1998; Crosby, 1998).

### C) Receptors

The main exposure route for PCE in humans is by inhalation, the gastrointestinal tract is second, and it is not absorbed well by the skin (Torkelson and Rowe, 1981). If inhaled to the lungs, PCE is rapidly transferred to the blood stream via the alveoli. Humans are exposed from recently dry cleaned clothes or locations near dry cleaners, from manufacturing and repair shops, and from contaminated drinking water. Occupational exposure to PCE is through dry cleaning and facilities that use degreasers. Exposure to TCE is from drinking water, inhalation indoors from building materials, and consumer products. Occupational exposure is from vapor degreasing or other production processes (Schettler, 1999).

Once inhaled or ingested, the blood transports PCE to fatty tissue, the liver, the kidney, or to the lungs to be exhaled. The most significant excretion route for PCE is through exhaled breath; it is exhaled in the form of the parent compound. A small portion is metabolized in the liver by cytochrome P-450. This system converts PCE to the main urinary metabolite, trichloroacetic acid (TCA), and also to trichloroethanol. Levels of TCA detected in urine reach a maximum with further increased dose, suggesting that the urinary excretion pathway can become saturated (ATSDR, 1993).

The behavior of PCE in the body leads to other, more insidious routes of exposure. Since one of the ways that people excrete solvents is through exhaled breath, workers exposed to both PCE and TCE may return home to unknowingly expose their families (Thompson and Evans, 1993). PCE is known to concentrate in breast milk, due to its lipophilic nature and the breast tissue's relative inefficiency in clearing contaminants, thereby exposing infants (Fisher, et al, 1997). There is some

evidence that the fetus may be dosed by PCE crossing the placenta (ATSDR, 1993).

Acute exposures to PCE and TCE affect the liver, kidney, and central nervous system (U. S. EPA, 1998). Animal studies provide much of the information available on toxicity. Inhaled doses of PCE, which caused death within four hours, were 3786 parts per million (ppm) for rats and 2613 ppm for mice (NTP, 1986). The LD50 (the dose producing lethality for 50% of the test subjects) for oral administration of PCE in laboratory rats is 2,629 mg/kg, and for TCE in mice is 22,402 mg/kg (Watts, 1996). For high inhaled or oral doses of PCE, humans experience central nervous system effects such as eye and respiratory irritation, dizziness, loss of coordination, and unconsciousness. The threshold for neurotoxicity effects from acute inhalation is 100 ppm (U. S. EPA, 1994). Exposure to levels of PCE above 1,500 ppm can lead to death from respiratory depression (HSIA, 1999). TCE has also caused death in humans, such as in large doses of occupational exposure with poor ventilation (Ford, 1995).

In chronic exposures, PCE and TCE target the liver and kidney, and can irritate skin. Human liver damage in exposed workers manifests itself as hepatomegaly and jaundice. Kidney damage arrives in the form of altered renal function (ATSDR, 1993). Prolonged skin exposure to PCE causes the skin to defat, leading to irritation, dryness, and dermatitis (Torkelson and Rowe, 1981). Animal studies of chronic PCE exposure support the effects claimed for humans, and have produced a no-observed-adverse-effect-level (NOAEL) of 14 mg/kg/day in rats. Based on this, the EPA established an oral reference dose (RfD) for PCE of 0.01 mg/kg/day. This is the dose that should not produce adverse effects to humans, including sensitive populations (U. S. EPA, 1994).



PCE and TCE may have consequences for reproductive and developmental health in humans. PCE has been implicated in cases of spontaneous abortion and infertility (Schettler, 1999). A few reproductive studies were performed involving women working in the dry cleaning industry. The studies found an increase in menstrual disorders and spontaneous abortions, but could not identify PCE as the sole cause. Animal studies have not found teratogenic effects (ATSDR, 1993). TCE may cause miscarriage and cardiac abnormalities in infants (Schettler, 1999).

Research has not been able to show PCE or TCE to be known human carcinogens, although there is evidence of carcinogenicity in animal studies (U. S. EPA, 1994). In humans, exposure to PCE in drinking water was found to be associated with cancers of the lung and colon-rectum (Paulu, et al, 1999). There is evidence of increased risk of esophageal, laryngeal, and tongue cancers from exposure to PCE in dry cleaning establishments (Vaughan, et al, 1997; Weiss, 1995; Lynge, et al, 1997). In a study of occupational exposure to PCE, Weiss (1995) did not find an increased risk in liver cancer or leukemia, but did find a two-fold increase in rates of esophageal and bladder cancer. Increased risks of liver and biliary tract cancers were found with TCE exposure (Lynge, et al, 1997).

Studies finding evidence for a causal association between cancer and PCE or TCE are disputed noting methodological shortcomings, lack of quantitative evidence of exposure, or the inability to rule out confounding factors (McLaughlin and Blot, 1997; HSIA, 1999). In the study of occupational exposure noted above, Weiss (1995) adds that the increase in esophageal cancer cannot be separated from the possible confounding effect of cigarette smoking and heavy alcohol consumption, and the

increase in bladder cancer could be due to exposure to solvents other than PCE used in dry cleaning. Currently PCE is classified by the EPA as a “probable human carcinogen” (HSIA, 1999).

PCE and TCE may also find receptors in aquatic organisms and terrestrial animals. In aquatic organisms, concentrations producing toxicity range from below 1 to 100 mg/L. LC50 (the concentration producing lethality for 50% of the test subjects) values for 96 hour tests of fathead minnow, bluegill, and rainbow trout were between 5 and 21.4 mg/L. The 48 hour LC50 for daphnia magna was 9.1-18.0 mg/L (U. S. EPA, 1994). Bioconcentration factors (BCF) measured in fish are 39-49. These values suggest that PCE will not bioaccumulate significantly in fish, but that environmental exposure can be detrimental to the health of aquatic organisms (U. S. EPA, 1998). Due to the high volatilization rate of PCE and TCE, they are not expected to be significantly toxic to terrestrial animals at environmental levels (U. S. EPA, 1994).

#### D) Controls

Controls on PCE and TCE exist in the form of several Federal regulations. Different EPA offices produce their own laws which cover PCE and TCE. The Office of Pollution Prevention & Toxics oversees the Toxic Substances Control Act and the Emergency Planning and Community Right-to-Know Act, including the Toxics Release Inventory data. The Office of Air covers the Clean Air Act. The Office of Solid Waste & Emergency Response is responsible for the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA, or Superfund), and for the Resource Conservation and Recovery Act (RCRA). The Office of Water presides over the Clean

Water Act and the Safe Drinking Water Act (U. S. EPA, 1994).

Under the Safe Drinking Water Act (SDWA), Maximum Contaminant Levels (MCLs) are established for regulated contaminants. These values are intended to protect the health of people drinking water from public systems over their lifetime. MCLs are based on a  $1 \times 10^{-6}$  level cancer risk to humans, unless the analytical sensitivity required or the available technologies for water treatment place practical limits on the requirement. The MCL for both PCE and TCE is 5 µg/L (Watts, 1996).

When PCE or TCE waste is generated from a business, it is listed under the Resource Conservation and Recovery Act (RCRA) as F001, one of the “F list”, hazardous wastes from nonspecific sources. As a RCRA hazardous waste, TCE and PCE are subject to cradle-to-grave management practices enforced by the EPA. These practices include the preparation of a Uniform Hazardous Waste Manifest by all generators, which is passed on to transporters of the chemicals, and finally to treatment, storage and disposal facilities. The Manifest ensures proper documentation of the location and fate of the waste. PCE has been implicated at Superfund sites as well, such as Hardeman County in Tennessee (Watts, 1996). The reportable quantity for releases to qualify under Superfund is 100 pounds (HSIA, 1999).

Limits values have been established for workers who may be exposed to chemicals in their workplaces. To avoid chronic toxicity, caused by long-term, low-level exposure to airborne contaminants, Threshold Limit Values (TLVs) are set and continually updated by the American Conference of Governmental Industrial Hygienists (ACGIH). The 8 hour TLV for PCE is 25 ppm. This value is a time weighted average (TWA) for a 40 hour workweek. The 15 minute Short Term Exposure Limit (STEL) is

100 ppm. The Occupational Safety and Health Administration (OSHA) also publishes permissible exposure limits (PELs) for PCE given a variety of exposures. Currently the 8-hour TWA value is 100 ppm, the ceiling limit is 200 ppm, and the peak limit is 300 ppm (HSIA, 1999).

Occupational controls on exposure to PCE include personal protection equipment (PPE) and provisions for ventilation in the workplace. Ventilation should include local exhaust or a process enclosure ventilation system. PPE includes splash resistant safety goggles and chemically resistant clothing and gloves. Also required are an emergency eye wash fountain and shower in the immediate area (MSDS).

Remediation efforts and treatment technologies are a form of control for PCE. The Best Available Technologies (BATs) named by the EPA are granular activated charcoal and packed tower aeration (U. S. EPA, 1998). Incineration is another technology that has been used. Unfortunately in the case of groundwater contamination, all of these procedures require that PCE be pumped to the surface, which can be costly if possible at all (Talley and Sleeper, 1997). In 1998, the State Coalition for Remediation of Drycleaners (SCRD) was established with support from the EPA. The goals of the organization include providing a forum for the exchange of ideas with other states and supporting innovative technologies in drycleaner remediation (SCRD, 2000).

An alternative to pump-and-treat for PCE and TCE is permeable reactive barriers (PRBs). PRBs are a passive technology, remaining in the ground and treating contaminants *in situ*. Since PCE and TCE can persist in aquifers for many years PRBs may be a useful and cost effective technology (Gavaskar, 1999). One example of a

successful PRB pilot study used iron foam reactive media placed in bedrock using high pressure hydraulic fracturing equipment. Monitoring of treatment effectiveness through adjacent observation wells showed concentrations of chlorinated volatile organic compounds, including PCE and TCE, to decrease as much as 98% (Marcus and Bonds, 1999).

Other *in situ* remediation methods are available. Stimulation of reductive dehalogenation of PCE and TCE by adding electron donors has already been mentioned. Combined chemical and biological treatments have been found to increase the extent of PCE mineralization over stand-alone reactions. One example is using modified Fenton's reagent with a bacteria, *Xanthobacter flavus* (Buyuksonmez, et al, 1999). Another innovative technology named the "Lasagna" process uses buried electrodes to supply current to contaminated soil. The current generates heat, increasing the mobility of TCE. The chemical moves through treatment zones containing kaoline clay and iron filings, which remove the TCE before it reaches groundwater aquifers (Canning, 2000).

The best form of control for PCE may be to reduce its use by replacing it with green chemistry. A surfactant has been developed (Micell Technologies, Inc., Raleigh, N.C.) which binds to liquid carbon dioxide at one side and to oils and dirt on the other. The waste from this process is recyclable and harmless. Although the machines needed to use the alternative dry cleaning agent cost twice as much as those that use PCE, they might still be attractive to the industry. Not only would they allow businesses to avoid environmental issues, but dry cleaners could clean a broader range of fabrics and provide services such as addition of stain resistant coatings or water repellency

with the same machine, and eliminate the possibility of heat damage to clothing (Ward, 1997).

### **Conclusions**

High production rates and usage patterns of PCE lead to significant environmental releases. These releases contaminate air and surface waters, and the chemical finds its way into groundwater. Even though PCE is degraded relatively quickly as an atmospheric contaminant, humans are subject to significant exposures from point sources. As a groundwater contaminant, PCE is persistent due to its behavior as a DNAPL, lack of potential for photooxidation, and limiting factors in biodegradation.

Through inhalation or consumption of contaminated water, humans are receptors for PCE, with both acute and chronic effects. The main target organs are the liver, kidneys, and central nervous system. There are some adverse health effects involving reproductive and developmental health. Although contested, evidence exists for the possibility of carcinogenicity.

For people wishing to control their own exposure to PCE, the most effective way would be to avoid having your clothing dry cleaned (or choose a dry cleaner with a green technology), to avoid living near a dry cleaning establishment that uses PCE, and to avoid consumer products that contain PCE. Those who risk occupational exposure must adhere to proper handling and protective equipment procedures.

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