

# The Scientific Facts About the Dry-Cleaning Chemical Perc

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## Executive Summary

- Perchloroethylene (PCE, or perc) has been the subject of close government and public scrutiny for more than 20 years. But government agencies in the U.S. and around the world have not agreed about the potential of environmental exposure to PCE to cause adverse health effects, including cancer, in humans. This report summarizes and evaluates the evidence behind these disparate views, and provides a balanced assessment of the possible risks of PCE based on the best available science.
- Inhalation of high levels of PCE, and chemically similar solvents, can cause neurological effects such as nausea, headache and dizziness. High inhaled doses also have been linked to changes in blood chemistry indicating that the liver and kidneys have been affected.
- These effects have been seen almost exclusively in workers, particularly in the dry-cleaning and chemical industries. There also have been claims that reproductive difficulties are associated with occupational exposure to PCE.
- The claim that PCE is a carcinogen has received the most public and governmental attention. Concern has been expressed that environmental exposures to PCE in ambient and indoor air, and in drinking water, can be carcinogenic in humans.
- Carcinogenicity claims are largely based on studies performed in rats and mice in which the rodents were exposed daily over a lifetime to very high air concentrations of PCE. The results of these experiments suggested that PCE exposure was associated with liver cancers in mice, and with kidney tumors in male, but not female, rats.
- In addition, results of some epidemiological studies of dry cleaning and chemical workers exposed to PCE have been interpreted to suggest a relationship between occupational exposure and various types of cancer.

Careful examination of the conduct of these studies reveals serious problems including uncertainties about the amounts of PCE to which people were exposed, lack of consideration of exposures to other chemicals at the same time, and failure to take into account known confounders. Due to these deficiencies, these studies do not support a link between PCE and cancer or other adverse effects.

- Studies on metabolism and mechanism of action have shown that the metabolism of PCE to trichloroacetic acid (TCA) occurs preferentially at high doses and to a much greater extent in rodents, particularly mice, than in humans; and it is likely the TCA that causes the adverse effects on the liver. Other studies have shown that PCE combines with glutathione much more rapidly in rodents than humans and that male rats have the unique ability to accumulate a protein known as alpha-2 globulin in the kidney—findings which likely explain the occurrence of kidney tumors in male rats only.
- The differences between humans and rodents in the metabolism and mechanisms of action of PCE make it unlikely that the carcinogenic effects seen in mice and rats administered high levels of PCE will occur in humans exposed at environmentally relevant levels.
- A number of government agencies have investigated the risks of PCE exposure in humans but their conclusions vary greatly. A careful examination of the science behind such differences reveals that the more data that are considered, the lower the estimated risk. Indeed, the agency that incorporates the most data, Health Canada, concludes that PCE poses little or no risk to the public.
- A careful and balanced evaluation of the possible risks of PCE to the public—based on occupational data, epidemiological studies, laboratory animal experiments, and biochemical research—finds no credible evidence that adverse public health effects, including cancer, are caused by environmental exposure to PCE.

## Introduction

Tetrachloroethylene—also known as perk, perchloroethylene, and PCE—is a nonflammable solvent that has been in use commercially for over 75 years. Current applications include use as a chemical intermediate, particularly in the synthesis of hydrofluorocarbons, as a metal degreaser, and as a dry-cleaning solvent. U.S. production of PCE has declined greatly during the past few decades. (ATSDR, 1997) Contributing to this decline are increased recycling and re-use of PCE and substitution of other compounds for PCE in a number of uses.

While it was used medicinally as a treatment for hookworm in the first half of the 20<sup>th</sup> century, public concern about adverse effects of exposure to PCE came to the forefront as the result of a number of studies and observations during the past few decades. These included observations of liver and neurological effects in workers exposed to prolonged and high levels of PCE in air; laboratory animal studies suggesting that PCE may cause cancer; studies of indoor air suggesting that humans are routinely exposed to PCE in their homes; and the discovery that PCE was a common contaminant at Superfund sites. Adding to the concern were reports of adverse health effects from studies of related compounds, particularly trichloroethylene (TCE). (ATSDR, 1997)

As a result of these concerns, there was increased scientific study of PCE and intensified governmental attention to the risks of PCE. As part of this attention, a number of agencies assessed the risk of PCE, with varying results. For example, the International Agency for Research on Cancer (IARC) labeled PCE a “probable human carcinogen” (IARC, 1995), while the U.S. Environmental Protection Agency decided it was unable to issue a cancer classification for PCE. On the other hand, Health Canada has classified PCE as “unlikely to be a human carcinogen” and, more importantly, has concluded that PCE “is not entering the environment in quantities or under conditions that may constitute a danger . . . to human life or health.” (TERA, 2001; HC, 1993) The bases for these and other assessment disparities will be addressed in the following sections, especially the one on toxicity.

## Exposure

Assessing human exposure to PCE requires knowledge of sources of the chemical, its fate in the environment, and human behaviors related to use of the chemical. PCE is a volatile compound and, not surprisingly, the vast majority of it is released into the air from applications where it is used in the highest volumes—the chemical industry and the dry-cleaning industry. It is estimated that over 80 percent of PCE used in commerce ends up in the atmosphere. (OW, 1982) In addition to current sources, there is public concern about residuals from previous contamination, particularly at hazardous waste sites. (ATSDR, 1997)

Once released to the atmosphere, PCE is fairly stable, with a half-life of about 50 days, depending on temperature and other environmental conditions. As a result, PCE released to the air can move significant distances from the source and has the potential to contaminate a variety of environmental compartments. If PCE is released to soil, it can move readily downward into groundwater where it is very slowly broken down by microbes over many years. The products of PCE biodegradation in soil and groundwater are less chlorinated compounds, including TCE, dichloroethylene, and vinyl chloride. It is thought that the detection of these latter three compounds at hazardous waste sites can be ascribed, at least in part, to this degradation process. Although the mechanism of contamination is not clear, small amounts of PCE are found in a variety of foodstuffs, and so ingestion of food as well as water can be added to inhalation as a possible (though probably not significant) route of exposure. (ATSDR, 1997)

Many studies have been conducted to determine PCE levels in both ambient air and the indoor environment so as to estimate inhalation exposures of the general population. Available surveys of PCE levels in food have been used to provide general estimates of population exposures through this source. In addition, analyses of soil and groundwater at hazardous waste sites have been utilized in assessing exposures of local populations. (ATSDR, 1997)

It has become clear through these studies that indoor air is the greatest contributor to general population exposure to PCE. Although the ratio of PCE levels in indoor to outdoor air varies greatly depending on the properties of the dwelling (e.g., air exchange rate) and the behaviors of the occupants (e.g., frequency and quantity of dry-cleaning), it appears that indoor air levels of PCE can range from about equivalent to outdoor-air levels to about 100 times higher than outdoor-air concentrations. For

example, Health Canada estimates that outdoor air levels range from 0.2 to 5.0 micrograms of PCE per cubic meter of air ( $\mu\text{g}/\text{m}^3$ ) while indoor air levels average about  $5.1 \mu\text{g}/\text{m}^3$ . (HC, 1993) (A microgram is a millionth of a gram or a billionth of a kilogram.)

Assuming that the average person spends 20 hours/day indoors and 4 hours/day outdoors, these levels correspond to an adult indoor PCE intake through inhalation of about 1.4 micrograms of PCE per kilogram of body weight per day ( $\mu\text{g}/\text{kg}/\text{day}$ ) and an outdoor intake through inhalation ranging from 0.01 to  $0.27 \mu\text{g}/\text{kg}/\text{day}$ . By comparison, intake of PCE in water is estimated at 0.002 to  $0.02 \mu\text{g}/\text{kg}/\text{day}$  and in food at  $0.12 \mu\text{g}/\text{kg}/\text{day}$ . Total PCE intake for all age groups is estimated to vary between 1.2 and  $2.7 \mu\text{g}/\text{kg}/\text{day}$ . (HC, 1993) These values are very similar to those estimated by the U.S. Agency for Toxic Substances and Disease Registry (ATSDR). (ATSDR, 1997)

These data suggest that the indoor environment is the most significant contributor to PCE exposure of the general population. It is possible that a few members of the public are exposed to high levels of PCE from other sources—e.g., local industrial sources that emit large amounts of PCE, or heavily contaminated drinking water. While this possibility exists, there are currently no data to quantify either the number of such individuals or the magnitudes of their exposures.

## Toxicity

### *Metabolism and mechanism of action*

To understand the possible health impacts of human exposures to PCE and to extrapolate effects seen in laboratory animals to humans, it is critical to compare the metabolism and fate of PCE in humans with those of experimental animals. Inhaled PCE is rapidly absorbed in humans and reaches equilibrium in the blood within three hours. (Hake and Stewart, 1977) It can accumulate in human tissues with high fat content but concentrations decrease rapidly with a half-life of 2–3 days. (Monster, 1986) The vast majority of absorbed PCE leaves the body unchanged, largely through the lungs, whether inhaled or ingested. (NTP, 1986)

In humans, the major metabolic pathway is conversion to trichloroacetic acid (TCA), although only about 1–2 percent of the PCE that remains in the body can be metabolized to this product, a step that appears to occur only at high levels of inhalation exposure (100–200 ppm). (Ohtsuki et al., 1983) The conversion of PCE to TCA occurs to a

much greater extent in mice than in humans and to a somewhat greater extent in rats as compared to humans. (Ikeda and Ohtsuji, 1972; Odum et al., 1988) In another metabolic reaction, PCE combines with glutathione, a compound that is part of important biochemical pathways in living creatures. This reaction occurs preferentially in male rats; it occurs about twice as fast in male compared to female rats and 30 times faster in male rats than in mice or humans. (Green, 1990)

These differences are important in light of the mechanisms of toxicity for PCE's various effects. While PCE is thought to directly affect the nervous system, metabolism appears to play an important role in its other toxicities. For example, liver toxicity in rodents is thought to be produced by effects of TCA, especially on peroxisome proliferation, a process associated with cancer production. (DeAngelo et al., 1989) In addition, kidney effects in rodents result from reactions following the combination of PCE with glutathione. PCE appears to cause a particular type of effect in the kidney of male rats only, because these males have the unique ability to accumulate a protein, namely alpha-2 globulin, that is critical to the renal effects. (Olson et al., 1990)

In sum, biochemical research has shown that there are significant differences between humans and rodents with respect to the metabolism of PCE. These include greater conversion of PCE to TCA, and preferential reaction with glutathione in rodents. These differences have an important impact on toxicity since many of the adverse effects of PCE exposure are due to metabolic products rather than to PCE itself. In addition, research demonstrating the unique ability of male rats to accumulate alpha-2 globulin is important to consider in extrapolating toxicity data from these animals to humans.

### *Non-cancer effects*

While there have been a number of laboratory-animal and epidemiological studies of the non-cancer effects of both oral and inhalation exposure to PCE, the resulting data are not always easy to interpret. This is partly because the laboratory-animal experiments were designed to detect carcinogenic effects and partly because epidemiological studies provided only limited information on exposure and confounders. There are, however, some conclusions that can be drawn.

Acute exposures to high levels of PCE produce neurotoxicity and liver and kidney dysfunction in humans. (Stewart, 1969; Hake and Stewart, 1977) Chronic exposure to high doses of ingested PCE seems to affect mainly the liver and kidney, while neurotoxicity seems to be the major effect of high inhalation exposure (ATSDR, 1997). In laboratory

animal studies, PCE does not produce teratogenic effects and appears to be fetotoxic and embryotoxic only at levels toxic to the mothers. (Schwetz et al., 1975) Epidemiological investigations of occupationally exposed humans indicate that PCE is not teratogenic. There are conflicting results with respect to reproductive effects in the same populations—overall the evidence does not support a link between PCE and such effects. (HC, 1993)

Using the data collected, and operating under the assumptions that: (1) high-dose results can be extrapolated to low doses, and (2) results in rodents can be extrapolated directly to humans without considering metabolism or mechanism of action, government agencies have calculated the doses (or concentrations) of PCE that humans can be exposed to daily over a lifetime without harm. These are generally known as acceptable daily intakes although they are given different names by different agencies; e.g., Tolerable Daily Intake/Concentration by Health Canada (HC) and Reference Dose/Reference Concentration by the U.S. EPA (EPA).

Based on the laboratory studies selected by each agency and their respective assumptions, the EPA and HC have calculated acceptable lifetime daily ingestion values and acceptable lifetime daily inhalation concentrations for humans. These represent the maximum values that humans could be exposed to safely every day over a lifetime, and they include significant margins of safety. Since the inhalation values are generally based on studies where laboratory animals are exposed to air containing specific concentrations of the chemical under study, the toxicity values are provided in concentrations rather than doses. These values can be translated into doses using information about how much air humans inhale daily and how much they weigh. The table summarizes the EPA and HC values for PCE.

**Table: Acceptable Human Lifetime Daily Ingestion and Inhalation Values for PCE**

Country/Agency	Acceptable lifetime daily ingestion dose	Acceptable lifetime daily inhalation concentration/dose
United States/EPA	10 µg/kg/day (IRIS, 2001)	0.600 mg/m <sup>3</sup> (provisional)/ 170 µg/kg/day (RAIS, 1993)
Canada/HC	14 µg/kg/day (TERA, 2001)	0.36 mg/m <sup>3</sup> / 100 µg/kg/day (TERA, 2001)

The disparities in values between the two countries/agencies reflect two factors: (1) differences in judgement as to the study or studies most appropriate for use in developing the acceptable exposure value, and (2) differences in judgement concerning how to extrapolate the results of laboratory-animal studies to human populations. It should be remembered that none of these toxicity values incorporate mechanistic considerations. As indicated, there are convincing data that suggest that metabolism of PCE at high doses is (a) different than at low doses and (b) occurs differently in rodents from humans. In both cases, the data strongly suggest that the toxicity values calculated by government agencies greatly overstate the non-cancer toxicity of PCE.

### *Carcinogenicity*

As indicated earlier, epidemiological studies suggesting a linkage between PCE (and related compounds) and cancer in humans have been the driving force behind both the risk assessment and risk management of PCE. In the aftermath of highly visible contamination incidents—such as the incident in Woburn, Massachusetts (DiPerna, 1984), which involved both TCE and PCE—the public conviction that PCE is carcinogenic in humans was much stronger than it is now. Indeed, in the late 1980s, the EPA determined that PCE was a probable human carcinogen and calculated its potency. But this conclusion and the calculations have been withdrawn, and the EPA has yet to publish a new determination of PCE's carcinogen status. (RAIS, 1993; IRIS, 2001)

The laboratory animal evidence for carcinogenicity of inhaled PCE derives largely from a National Toxicology Program study of rats and mice exposed to high levels of PCE (up to about 2,000 milligrams of PCE per cubic meter of air ( $\text{mg}/\text{m}^3$ ), 6 hours/day, 5 days/week over two years). There was a small, statistically insignificant increase in kidney tumors in the male rats, but not in the females. While other cancers were also detected in the rats, they were at levels comparable to those found in unexposed animals. Both male and female mice exhibited an increase in liver tumors. (NTP, 1986) An increase in liver tumors was also found in another mouse study in which PCE was administered orally, although the conduct of the study does not meet current scientific standards for validity. (NTP, 1977)

There have been a number of epidemiological studies of workers exposed to PCE by inhalation, both dry-cleaning workers and those employed in industries utilizing PCE. While some have suggested increases in the incidences of various types of cancers, others have not. In addition, the validity of almost all of these studies is very limited because of poor (or non-existent) characterization of exposure, failure to take into

account known confounders, exposures to multiple compounds, or some combination of these factors. These same problems adversely affected the validity of studies of the relationship of environmental exposures, such as through drinking water, to PCE toxicity. As a result, the epidemiological evidence is inadequate to contribute to the assessment of the carcinogenicity of PCE to humans. (HC, 1993)

As noted above, the EPA has withdrawn its classification of the carcinogenicity of PCE. Other government bodies have, however, published their determinations. The U.S. National Toxicology Program (NTP) and the International Agency for Research on Cancer (IARC) have classified PCE similarly, as “reasonably anticipated to be a human carcinogen” and “probably carcinogenic to humans,” respectively. (NTP, 1999; IARC, 1995) Health Canada, however, has classified it as “unlikely to be carcinogenic to humans.” (TERA, 2001) It should be noted that IARC originally classified PCE as a possible human carcinogen but, on the basis of newer epidemiological findings, upgraded this to “probable” in 1995. The United Kingdom Department of Health carefully evaluated the totality of the epidemiological literature, including the studies IARC cited, and concluded in 1998 that “available data were inadequate to draw any definite conclusions between exposure to tetrachloroethylene and cancer in humans.” (DOH, 1998)

The main reason the various governmental bodies classified PCE as a probable human carcinogen is that it caused cancer in laboratory animals after a lifetime of exposures to high doses. As with most government evaluations of non-cancer effects, these conclusions do not take into consideration metabolic and mechanistic considerations. For example, the occurrence of kidney cancers in male rats but not female rats or mice of either sex can be explained by the much more rapid reaction of PCE with glutathione in male rats compared to females, mice, or humans. Similarly, the occurrence of liver tumors in mice can be attributed to the much greater degree of metabolism of PCE to TCA in mice than in rats or humans, and to the data suggesting that TCA can cause liver cancer in mice.

Thus, differences in assessments of the carcinogenicity of PCE can be traced to whether or not all of the scientific data are used in the evaluation. Simply counting cancers at each dose can lead to one conclusion, but taking the differences in PCE metabolism and mechanism of action at the various doses and in various species into consideration leads to another. A full evaluation strongly suggests that the high dose animal data are not applicable to humans, and certainly not to low-level human exposures. Considering the strength of the evidence on PCE and the weakness

of the evidence to support its carcinogenicity, it is not surprising that after Health Canada conducted a more complete evaluation of PCE, it concluded that PCE is unlikely to be a human carcinogen.

## Risk Characterization

It is important to recognize that while a substance may pose a hazard (i.e., may have the potential to cause harm), adverse effects occur only when exposure is high enough for this potential to be expressed. That is, hazard is not equivalent to risk. To evaluate risk in any population, the minimum exposure levels estimated to cause adverse effects must be compared to measured human exposure levels. Maximum acceptable exposure levels have been presented in the toxicity section and the actual human exposure levels in the exposure section. Based on these, the risk to the general population and uniquely exposed populations can be evaluated.

First, with respect to the general population, Health Canada has estimated that the total daily PCE intake for adults is between 1.2 and 2.7  $\mu\text{g}/\text{kg}/\text{day}$ —of which most— 1.2–2.3  $\mu\text{g}/\text{kg}/\text{day}$ —are from inhalation exposure. These intakes are approximately one hundred times less than government-generated acceptable daily intakes for inhalation exposures, based on non-cancer effects, of about 100–200  $\mu\text{g}/\text{kg}/\text{day}$ . Health Canada estimates that members of the general population ingest 0–0.4  $\mu\text{g}/\text{kg}/\text{day}$  of PCE. Such exposure levels are at least 30 times lower than the oral acceptable intake values of 14–20  $\mu\text{g}/\text{kg}/\text{day}$  set by the EPA and HC. Considering that these acceptable daily intakes include generous margins of safety and do not take mechanistic considerations into account, there appears to be very little risk to the general population of non-cancer effects from exposure to PCE.

The disparities in government-agency assessments make it difficult to evaluate the cancer risk quantitatively. Health Canada indicates that PCE is unlikely to be a human carcinogen—which is equivalent to an essentially zero risk of cancer. Although the NTP suggests that PCE is likely to cause cancer in humans, it provides no estimate of potency. The U.S. EPA has published no current cancer classification for PCE, nor any estimate of its potency, and so it provides no clear guidance.

One could perform an evaluation of cancer risk using previously published (and now withdrawn) EPA potency values for both oral and inhalation exposure to PCE. (RAIS, 1993) Based on these numbers, the total cancer risk can be calculated by combining the inhalation and oral

cancer risks using HC estimates of daily inhalation and oral intakes. The resulting risk value of about 1 in 10,000 is at the high end of the range the U.S. EPA considers acceptable at Superfund sites and for drinking-water exposure. It must be remembered, however, that these values ignore any considerations of mechanism of action and assume that PCE affects organisms by interacting directly with DNA. This assumption is contrary to available evidence. If these factors were included, it is very likely that even if PCE were a human carcinogen, the general population risk would be very small and well within EPA acceptable limits.

Persons with the highest and most prolonged exposures to PCE are workers in the dry-cleaning industry and in certain sectors of the chemical industry. Although good quantitative data have not been collected for most occupational exposures, it seems likely that daily exposures of workers are orders of magnitude higher than those of the general population. In some instances, prolonged, high level occupational exposures have resulted in adverse non-cancer effects. However, studies of workers exposed to such levels in the past, including the most recent one (Ruder et al., 2001) do not provide any convincing evidence of a relationship between these exposures and cancer—supporting the conclusion that the EPA potency values represent overestimates of possible risk.

Other populations possibly at risk are those living near industries utilizing PCE who may be exposed through emissions from these facilities, and those living near hazardous waste sites contaminated with high levels of PCE. Except in very rare cases, their exposures are likely to be so much lower than those of workers that adverse effects are not expected. This is true even taking into consideration the existence of sensitive subpopulations, such as those with serious ailments.

## Risk Management

From the above analysis, it seems clear that occupational exposures are almost always higher than environmental ones. It seems very unlikely that the general population and potentially at-risk subpopulations will be exposed to PCE at levels high enough to produce adverse effects. In almost all cases, the highest environmental and occupational exposures are due to inhalation of indoor air in the home and the workplace, respectively. A question that follows is how well current U.S. governmental risk management approaches reflect these conclusions.

The above analysis implies that government risk management

should focus on the workplace, since that is where the exposure potential is highest, and on the rare environmental situations where exposures may be unusually high. In the U.S., occupational exposure is regulated by the Occupational Safety and Health Administration which has set the standard for occupational exposure to PCE, known as the Permissible Exposure Limit (PEL), at 100 parts of PCE per million parts of air (ppm) (corresponding to about 700 mg/m<sup>3</sup>). (OSHA, 2001)

Assuming a worker inhales 10 m<sup>3</sup> per day, five days a week, and weighs 70 kg (about 155 pounds), this corresponds to a daily intake of about 70 mg/kg/day (70,000 µg/kg/day). It should be noted that a non-governmental organization, the American Council of Governmental Industrial Hygienists (ACGIH) recommends a limit of 25 ppm, corresponding to a daily intake of about 20 mg/kg/day (20,000 µg/kg/day). (ACGIH, 2001) Even this latter value is orders of magnitude higher than the acceptable daily intakes for the general population of 100–200 µg/kg/day promulgated by the EPA.

The U.S. EPA has spent enormous sums of money during the past two decades addressing residual pollution at Superfund sites, mainly in soil and groundwater. Since the major concern regarding exposure from such waste sites is exposure from groundwater used in the home, it is appropriate to examine cleanup standards for groundwater. These standards are generally set to correspond with established drinking water standards, known as Maximum Contaminant Levels (MCL). The MCL for PCE is 5 micrograms of PCE per liter (about a quart) of water (µg/L). (OW, 2001) Using standard EPA assumptions: i.e., consumption of two liters of water/day and a weight of 70 kg, this MCL would correspond to a daily intake of 0.14 µg/kg/day. This represents about one percent of the acceptable oral daily intake of 14–20 µg/kg/day based on non-cancer effects. Even if inhalation exposures due to volatilization of PCE from the water and skin exposures while bathing are included, the total water contribution to exposure is still only about two percent of the acceptable daily intake.

It should be noted that the 5 µg/L value is not based on non-cancer effects but rather on PCE being classified as a probable human carcinogen, a classification the EPA has not issued. In addition, it is not a toxicologically based value; it assumes that the drinking water goal should be zero and sets the MCL on the basis of the lowest PCE levels measurable by available analytical techniques.

Health Canada has also promulgated a drinking-water standard, known as the Maximum Acceptable Concentration (MAC), for PCE. The HC value is 30 µg/L and, using the HC assumptions of consumption of 1.5 Liters/day and a body weight of 70 kg, corresponds to an intake of

about 0.65 µg/kg/day. (HC, 1995) The HC value is based on the non-cancer Total Daily Intake (TDI) value and represents about 5 percent of the HC acceptable daily intake value. Incidentally, the World Health Organization [WHO] has issued a guideline standard for PCE in drinking water of 40 µg/Liter, slightly higher than the HC value and also based on the non-cancer effects. (WHO, 1996)

Considering that the acceptable daily intake numbers include significant margins of safety and do not include consideration of mechanisms of action, and that other government entities, which also utilize significant margins of safety, have promulgated much higher acceptable levels, it seems clear that the established EPA clean up goals for PCE are not likely to decrease any risk that might exist to any appreciable extent, if at all.

Although indoor air is the largest contributor to PCE exposures of the general population, there are no standards for PCE in indoor air set by any level of government, or for PCE in outdoor air at the U.S. federal level. Various U.S. states, however, have established acceptable ambient air concentrations for PCE. (ATSDR, 1997) These levels vary tremendously but in many cases correspond to extremely low exposures considering they must take into account that humans generally spend little time outdoors. Further, they do not spend it in the same place hour after hour and day after day, and maximum levels of PCE often occur at times when people are not exposed. Such extremely low exposures do not contribute significantly to overall PCE intake. Thus, it is very unlikely that enforcement of these standards will appreciably reduce human risk from PCE.

## Conclusion

Overall, the available evidence strongly suggests that the general population is not at risk from exposure to PCE. Those who are potentially at greatest risk are workers who may be exposed to PCE at high levels for prolonged periods of time. Unfortunately, the application of risk management resources to PCE does not seem to reflect these scientific conclusions. These resources have been focused on protecting the general population rather than workers, and on PCE rather than other well-documented public health threats of much greater concern.

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