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Review

Trichloroethylene and tetrachloroethylene contamination: A review of toxicity, analytical methods, occurrence in foods, and risk assessment

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Abstract Polychlorinated hydrocarbons are continuously released into the environment from various industrial processes. Trichloroethylene (TCE) and tetrachloroethylene (perchloroethylene, PCE) are of primary concern because of their large-scale production, wide industrial application, poor biodegradability, and tendency to circulate in the air and water. The common routes of human exposure to these compounds include inhalation, ingestion, and dermal adsorption. Additionally, they have been detected in various plant foods. Prolonged exposure to these contaminants is associated with certain risks. They are carcinogenic and have other toxic effects, including gastrointestinal, developmental, neurological, and hematological toxicity. To analyze these contaminants, they are generally extracted from various matrices, followed by instrumental analysis. Gas chromatography, often in combination with different detectors, is the most widely used analytical method. This review covers the toxicity, analytical methods, occurrence in foods, and risk assessment of these contaminants.

Keywords trichloroethylene, tetrachloroethylene, food, toxicity, analytical methods

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1. Introduction

Polychlorinated organic compounds have since many years significantly been contributing to water and environmental pollution (Bruckner et al., 1989). Specifically, this issue has been linked to the widespread industrial usage of trichloroethylene (TCE) and tetrachloroethylene, also referred to as perchloroethylene (PCE) (Hughes et al., 1994; Jollow et al., 2009). TCE is a colorless, volatile liquid organic compound that smells like chloroform. It is primarily and most widely used in the degreasing of metal parts (ATSDR, 1997). People who work in the metal industries are therefore frequently exposed to TCE (Bakke et al., 2007). TCE is also employed in various other industries, such as the chemical, dry cleaning, textile, food, agricultural, electrical, and leather processing industries (Bakke et al., 2007; Khan et al., 2009). PCE is a highly volatile and lipophilic solvent that is frequently used in textile, metal, and dry-cleaning operations (Guyton et al., 2014).

Humans are frequently exposed to these substances through the ingestion of foods, breast milk, and plants, inhalation, groundwater contamination, occupational exposure, and other means (Doucette et al., 2007; Lan et al., 2010; Moran et al., 2007). Numerous reports regarding the hazardous properties of these substances have been published. For example, soybean oil meal extracted with TCE resulted in an outbreak of refractory and fatal hemorrhagic toxicity in cattle

(Mckinney et al., 1955; Picken et al., 1955). TCE induced metabolic and biochemical transformations in rat tissues (Khan et al., 2009) and reportedly induces Parkinson's disease (Liu et al., 2018). Exposure to PCE can cause reproductive and developmental defects (Bagnell and Ellenberger, 1977; Beliles, 2002; Bove et al., 2002; Kyyronen et al., 1989; Schwetz et al.1975), hematological toxicity (Seidei et al., 1992), acute and chronic toxicity (Richter et al., 1983), neurobehavioral toxicity (Seeber, 1989) and cardiopulmonary toxicity (Kobayashi et al., 1982). Both TCE and PCE are categorized as Group 2A carcinogens by the International Agency for Research on Cancer (IARC), indicating a potential risk for human cancer (IARC, 2014).

Chromatographic techniques (e.g., gas chromatography and high-performance liquid chromatography) are the most widely used to analyze these substances and their metablites in various media. Other methods include capillary electrophoresis (Ahrer and Buchberger, 1999), ion chromatography (Sarzanini et al., 1999), and high-field asymmetric waveform ion mobility spectrometry (Ells et al., 2000). Common detectors used in their analysis include mass spectrometry (MS) (Brashearl et al., 1997), flame ionization (Xu et al., 1996), electron capture (Forkert et al., 2003; Ketcha et al., 1996; Merdink et al., 1998), ultraviolet (Kim et al., 2001; Martınez et al., 1999), and conductivity (Narayanan et al., 1999) detectors. Common techniques to extract these substances and their metabolites from their matrix include liquid-liquid extraction (Ko et al., 2000), solid-phase microextraction (SPME) (Dehon et al., 2000; Xu et al., 1996), solid-phase extraction (Benanou et al., 1998; Calafat et al., 2003), and protein precipitation (Narayanan et al., 1999).

Several reports have documented the existence of TCE and PCE in various food items and food products, albeit at low concentrations. Furthermore, these harmful substances are associated with specific health hazards. This review aims to provide an overview of the relevant literature regarding the toxicity, analytical techniques, occurrence in foods, and risk assessment of these pollutants.

2. Toxicity

There are several publications concerning the harmful effects of TCE and PCE on experimental animals as well as humans. Some of these harmful effects are highlighted in Tables 1 and 2.

2.1. Trichloroethylene

Male Wistar rats exposed to TCE had severe brain, intestinal, and organ (kidneys and liver) damage as well as altered carbohydrate metabolism and decreased antioxidant activity (Khan et al., 2009). Following the administration of 1,000 mg kg^{-1} day^{-1} TCE in corn oil to male Wistar rats for 25 days, urea blood nitrogen, serum creatinine, cholesterol, and alkaline phosphatase levels, which are indicators of liver and kidney toxicity, were increased, whereas serum glucose, inorganic phosphate, and phospholipid levels were decreased

Table 1. Toxic effects of tetrachloroethylene

Subjects	Toxic effects	References
Mice	Sperm head abnormalities	Beliles et al. (1980)
Male California dry-cleaning workers	Sperm abnormalities	Eskenazi et al. (1991)
Pregnant Finnish dry-cleaning women	Spontaneous abortion	Kyyronen et al. (1989)
Teratology rats	Increased resorptions	Schwetz et al. (1975)
Two-generation rats	Litters with dead pups	Tinston (1994)
Two-year-old boy	Death	Garnier et al. (1996)
Rats	Delayed behavioral changes and altered neurotransmitter levels	Nelson et al. (1979)
Female dry-cleaning workers	Neuropsychiatric tests	Ferroni et al. (1992)
6-Year-old male child	Severe depression	Koppel et al. (1985)
Young male mice	Hyperactivity	Fredriksson et al. (1993)
Pregnant rats	Reduced liver pup	Narotsky and Kavlock (1995)

Table 2. Toxic effects of trichloroethylene

Subjects	Toxic effects	References
Male Wistar rats	Brain, intestinal, and organ damage	Khan et al. (2009)
Rats	Central nervous system disturbance	Honma et al. (1980b)
Human	Permanent paresis of the olfactory nerves, gastric disturbance, liver degeneration, lung hemorrhage, death	James (1963)
Human	Cranial nerve palsies	Buxton and Hayward (1967)
Mouse & rat	Genotoxicity and mutagenicity	Miller and Guengerich (1983) Mazzullo et al. (1992) Nelson and Bull (1988) Walles (1986)
Human, rats	Parkinson's disease	Gash et al. (2008) Liu et al. (2010) Guerl et al. (1999)
Human, rats	Gastrointestinal effects	Liotier et al. (2008) Moritz et al. (2000) Vattemi et al. (2005) Byers et al. (1988) Tucker et al. (1982)

(Khan et al., 2009).

Honma et al. (1980b) determined the effects of TCE and PCE exposure on acetylcholine, dopamine, norepinephrine, and serotonin levles in the rat brain. Following exposure at 200, 400, and 800 ppm for 30 days, TCE increased dopamine levels in the striatum, albeit not significantly, whereas PCE had an opposite effect. Both chemicals significantly decreased acetylcholine levels in the striatum in a dose-dependent manner, with a significant effect at 800 ppm ($p<0.05$). TCE and PCE slightly increased norepinephrine levels in the hypothalamus, whereas TCE reduced norepinephrine levels in the cortex and hippocampus. As for serotonin, a non-significant increase was observed in the cortex and hippocampus following exposure to TCE and PCE (Honma et al., 1980b). These findings indicated that prolonged exposure to these chemicals may disturb the cholinergic neurons present in the central nervous system (Honma et al., 1980b). In a similar experiment, Honma et al. determined the free amino acid content in rat brains following exposure to TCE and PCE. They reported concentration-dependent increases in glutamine, threonine, and serine levels following exposure to PCE, whereas exposure to TCE induced significant increases in the levels of these amino acids at 800 ppm. Exposure to PCE decreased the glutamate content, whereas TCE exposure led to a significant decrease in glutamate at 800 ppm. No significant changes were observed in the contents of taurine, aspartate, and alanine (Honma et al., 1980a).

There is a risk of addiction to TCE inhalation following industrial exposure (James, 1963). Autopsy findings of a person with a TCE inhalation addiction disorder revealed that the addiction can result in permanent paresis of the olfactory nerves, gastric disturbance, fatty degeneration of the liver, lung hemorrhages and ultimately death (James, 1963). Buxton and Hayward (1967) reported that TCE may decompose into a toxic, irreversible substance that can cause cranial nerve palsies. They came to this conclusion based on the effects of inadvertent industrial exposure to TCE by four men, two of whom developed severe multiple cranial nerve palsies, leading to the death of one of them after 51 days (Buxton and Hayward, 1967).

There are numerous reports on the genotoxicity and mutagenicity of TCE. There is evidence that TCE metabolites can bind to and damage DNA (Mazzullo et al., 1992; Miller and Guengerich, 1983). Miller and Guengerich (1983) compared mice and rats exposed to TCE and found that mouse hepatocytes had significantly higher amounts of DNA adducts than hepatocytes. Mitotic recombination and aneuploidy are examples of mutational damage that can be induced by exposure to TCE (Cantelli-Forti and Bronzetti, 1988; Crebelli et al., 1985; Shahin and Von Borstel, 1977). *In vivo* examination of mouse and rat hepatic and kidney cells showed that TCE or its metabolites can bind to and

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induce single-strand breaks in DNA in these cells (Mazzullo et al., 1992; Nelson and Bull, 1988). The rate of DNA single-strand breaks differs depending on the species; low-level exposure to TCE more readily induced breaks in mice than in rats (Nelson and Bull, 1988; Walles, 1986).

Evidence suggests that exposure to TCE can induce Parkinson's disease (Gash et al., 2008; Liu et al., 2010). When TCE was administered systemically to adult Fischer 344 rats, dopaminergic neurons in the substantia nigra pars compacta were lost in a dose-dependent manner, and the rats displayed abnormal rotarod behavior, a marked decrease in mitochondrial complex I activity, higher oxidative stress marker levels, and activated microglia in the nigral region (Liu et al., 2010). One clinical report linked the onset of Parkinson's disease to occupational exposure to TCE (Guehl et al., 1999). In light of these findings, the authors exposed mice to TCE and measured tyrosine hydroxylase immunoreactivity to assess neuronal death; compared with control mice, those exposed to TCE a significant loss of dopaminergic neurons. (Guehl et al., 1999).

Gastrointestinal effects such as diarrhea, vomiting, and hemorrhagic gastritis can result from exposure to large amounts of TCE (Liotier et al., 2008; Moritz et al., 2000; Vattemi et al., 2005). A 47-year-old woman who intentionally consumed 500 mL of TCE and benzodiazepines suffere a fatal abdominal compartment syndrome, which resulted in multiple organ failure due to abdominal distension (Liotier et al., 2008). Complaints of people exposed to TCE and some other chlorinated hydrocarbons in Woburn, MA, USA included severe nausea, constipation, and diarrhea (Byers et al., 1988). In mice, exposed to 660 mg kg^{-1} day^{-1} TCE in water, gas pockets in the intestinal coating and blood in the intestines were observed (Tucker et al., 1982).

2.2. Tetrachloroethylene

To assess the hematological toxicity of PCE, Seidei et al. (1992) exposed mice to 270 and 135 ppm of PCE for 6 h per, 5 days per week, for 11.5 weeks (270 ppm) and 7.5 weeks (135 ppm), follwed by a 3-week exposure-free period. The experiment showed that, while nearly full regeneration occurred during the exposure-free period, peripheral blood neutrophil and lymphocyte counts decreased during the exposure period, and prolonged exposure led to hematopoietic failure in the myeloid and lymphoid cell lines (Seidel et al., 1992; Van Duuren et al., 1979).

Exposure to PCE during early pregnancy is associated with an increased risk of spontaneous abortion, according to a Finnish study in pregnant workers in the dry-cleaning industry (Kyyronen et al., 1989). Female rats exposed to PCE for two weeks (2 h day^{-1}, 5 days $week^{-1}$, N = 3) fewer fertilized oocytes after mating than control rats, indicating a connection between PCE exposure and fertility (Berger and Horner, 2003). Effects of PCE exposure on human sperm quality have been reported by Eskenazi et al. (1991). The effects included an increased proportion of round sperm, a reduced proportion of narrow sperm, and an increased amplitude of lateral head displacement, resulting in unsteady sperm movement. However, PCE exposure had no effect on the average percentage of motile or abnormally shaped sperm, the volume, number, and concentration of sperm, and did not increase the prevalence of azoospermia or oligospermia (Eskenazi et al., 1991). Some studies have reported decreased birth and fetal body weights as a result of exposure to PCE (Carney et al., 2006; Szakmary et al., 1997; Tinston, 1994).

Kobayashi et al. (1982) reported that PCE exerted cardiopulmonary toxicity in rabbits, cats, and dogs following an intravenous PCE injection. PCE increased the vulnerability of the ventricles to epinephrine-induced extrasystoles, bigeminal rhythms, and tachycardia, with mean threshold dosage levels of 10 mg kg^{-1}, 24 mg kg^{-1}, and 13 mg kg^{-1} in rabbits, cats, and dogs, respectively. Beyond the threshold dosage levels, the cats suffered acute pulmonary edema whereas the dogs experienced decreased left intraventricular $dP/dt_{(max)}$ (Kobayashi et al., 1982).

Schwetz et al. (1975) exposed Sprague-Dawley rats and Swiss-Webster mice to airborne PCE. The rats showed signs of embryotoxicity due to increased resorption, whereas the mice showed lower fetal body weights, delayed ossification of the skull bone, and increased subcutaneous edema (Schwetz et al., 1975). In another study, increased resorption occurred in pregnant Sprague-Dawley rats exposed to 1,800 ppm of PCE (Nelson et al., 1979).

Bagnell and Ellenberger (1977) reported that exposure to PCE through breast milk resulted in obstructive jaundice and hepatomegaly in a 6-week-old baby whose mother had been exposed to PCE after visiting a dry-cleaning service. After breast feeding was stopped, the baby's health improved rapidly, and liver function normalized within two years following the exposure.

In Summary, both TCE and PCE are toxic to both humans

and experimental animals. Their toxic effects include Parkinson's disease, spontaneous abortion, reproductive and developmental issues, and cardiopulmonary and gastrointestinal disorders.

3. Analytical methods

TCE and PCE are frequently detected in tandem with other volatile organic chemicals or chlorinated hydrocarbons. Before instrumental analysis, these chemicals generally must be extracted from their matrix. TCE and PCE are mainly analyzed using chromatographic techniques. In particular, headspace GC is often used analytical technique. Common detectors used include electron capture, MS, ultraviolet, flame ionization, and conductivity detectors. Some common analytical methods used for TCE and PCE are summarized in Table 3.

3.1. Extraction and pretreatment

Some of the common extraction techniques used for TCE and PCE include liquid-liquid extraction, acid digestion, microextraction, solid-phase extraction and protein precipitation.

Liquid-liquid extraction is very helpful for extracting TCE and PCE from different matrices as they are lipophilic compounds. For the analysis of drinking water, an organic solvent is added to the sample to facilitate the transfer of TCE and PCE into the organic layer (Brown et al., 2003a; Dewulf and Langenhove, 1999; Russo et al., 2003; Song and Ho, 2003). However, this extraction method often requires large amounts of sometimes toxic solvents. A modified liquid-liquid extraction method involves using large a volume of water (10 L) with 10 ml of n-hexane (Zoccolillo et al., 2004) or room-temperature ionic liquids (Pandey, 2006) for extraction.

Solvent microextraction has received widespread interest as it is a more effective procedure and addresses some of the limitations of liquid-liquid extraction (Psillakis and Kalogerakis, 2003). It uses a lower volume of solvent (~1,000× less) than liquid-liquid extraction, and sample extraction, preconcentration,

Table 3. Method of analysis of trichloroethylene & tetrachloroethylene

Sample	Analyte	Sample preparation	LOD/LOQ	Instrumental analysis	References
Food	TCE, PCE	Digestion, 20N H_2SO_4	10-50 ppb	Headspace GC-ECD	Entz and Hollifield (1982)
Food	TCE, PCE	Digestion, 20N H_2SO_4		Headspace GC-ECD, GC-MS	Entz et al. (1982)
Water	TCE, PCE	Microextraction		GC-FID	Ilavský and Barloková (2017)
Food	TCE, PCE	Purge and trap		GC-MS	Fleming-Jones and Smith (2003)
Food	TCE	Solid-phase microextraction	0.035-4.8 ng g^{-1}	GC-MS	Cao et al. (2016)
Water	TCE	Solid-phase microextraction		GC-FID	Xu et al. (1996)
Water	TCE	Liquid-liquid extraction	5 ng mL^{-1}	GC-MS	Brown et al. (2003b)
Blood	TCE	Liquid-liquid extraction	5 ng mL^{-1}	GC-MS	Brown et al. (2003b)
Water	TCE	Purge and trap	5 ng mL^{-1}	GC-MS	Eichelberger et al. (1990)
Water	TCE, PCE	Liquid-liquid extraction	30 ng L^{-1} TCE 25 ng L^{-1} PCE	GC-ECD	Russo et al. (2003)
Blood	TCE	Solid phase microextraction	1 ng mL^{-1}	GC-MS	Dixon et al. (2005)
Olive oil	PCE	Headspace equilibration	1 pg	GC-ECD	Van Rillaer and Beernaert (1989)
Olive oil	PCE	Direct injection	5 ppb	Membrane Inlet mass spectrometer	Kotiaho et al. (1995)
Fatty and non-fatty foods	TCE, PCE	Liquid extraction with isooctane, 20% acetone-5% sodium chloride in H_3PO_4 and isooctane		GC-ECD GC-Hall electrolytic conductivity detection (HECD)	Daft (1988)
Grain and grain-based foods	TCE, PCE	Purge & trap	0.5 ppb TCE 0.4 ppb PCE	GC-ECD GC-HECD	Heikes and Hopper (1986)

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and introduction occur simultaneously (Dong et al., 2006; Tor and Aydin, 2006; Zhao et al., 2004). The two main types of this extraction technique are single-drop microextraction and liquid-phase microextraction. Dispersive liquid-liquid microextraction is a recent solvent microextraction method developed by Rezaee et al. (2006). This method is advantageous in that the extraction time is reduced because of the large surface area between the solvent and the aqueous phase.

SPME is another extraction technique. It requires an SPME fiber to be inserted into the sample headspace, or immersed in the sample (Kataoka et al., 2000). The analyte adsorbs and is concentrated on the fiber coating. Several studies have adopted SPME using polydimethylsiloxane fiber to analyze TCE in biological samples (Dehon et al. 2000; Dixon et al., 2005; Xu et al., 1996).

Solid phase extraction is commonly used as an alternative to liquid-liquid extraction because it requires less solvent. It is mostly utilized for semi-volatile organic compounds (Delinsky et al., 2005; Kot-Wasik et al., 2004; Santos and Galceran, 2002); very few studies have employed this extraction method for volatile organic compounds because of the possibility of analyte loss due to volatility (Delinsky et al., 2005).

3.2. Instrumental analysis

Skender et al. (1993) used headspace GC to identify TCE and PCE in urine and venous blood samples of 39 patients residing in Zagreb, Croatia, and in drinking water collected from a kitchen tap. The limit of detection (LOD) was 0.020 μg L^{-1} for TCE and 0.015 μg L^{-1} for PCE. Entz and Hollifield (1982) developed a similar method involving headspace chromatography and an electron capture detector (ECD) to analyze volatile hydrocarbons, including chloroform, TCE, and PCE in foods. Some samples were digested with 20 N H_2SO_4, shereas others were not pretreated. Using this technique, the authors found residues of TCE, PCE, and other volatile hydrocarbons in eight food samples, including fish, chocolate sauce, mayonnise, ice cream, and other processed foods (Entz et al., 1982). The GC technique involved the use of three different columns at different stages of the analysis; column 3 was used for initial identification and quantification of residues, column 1 was used for provisional GC confirmation, and column 2 was used for GC-MS confirmation.

Ilavský and Barloková (2012) analyzed TCE, PCE and other chlorinated hydrocarbons in water at concentrations of 1-30 μg L^{-1} using a microextraction technique that involved manual mixing of the water with 0.5 mL of n-pentane at 5-7°C for 5 min. The analysis was conducted using capillary GC with a flame ionization detector (FID) and ECD.

PCE is of high concern because it can readily transform into trichloroacetic acid, which is a persistent herbicide and a significant cause of forest decline (Frank and Frank, 1989; Frank et al., 1989). Frank and Frank (1989) determined PCE concentrations in spruce needles in southwest Germany. After hexane extraction and separation using capillary GC, detection was achieved using chemical ionization MS. Frank et al. (1989) analyzed one- and two-carbon halocarbons, including TCE and PCE in forest soil and air using thermodesorption, cryogenic trapping, GC on thick-film capillaries, ECD.

TCE and PCE can transition from the air into foods bcause they are major organic pollutants present in the air (Grob et al., 1990). The concentrations of these contaminants in foods and air have been analyzed using headspace techniques with column effluents being detected using an ECD for small quantities, and a FID for large quantities (Grob et al., 1990). Van Rillaer and Beernaert (1989) developed an analytical method to quantitatively determine PCE residues in olive oil samples and other products using headspace chromatography with an ECD. The authors reported the LOD of PCE to be 1 pg, and the concentration exceeded the proposed maximum level of 1 mg kg^{-1} in only one of the samples analyzed.

Kotiaho et al. (1995) developed a membrane-inlet MS method to rapidly determine styrene and PCE levels in olive oil. A peristatic pump was used to supply a clean stream of olive oil to the membrane inlet at a flow rate of 3.5 mL/min. The injection time was 2 or 3 min, and a wait time of 3-4 min was required after each sample injection, during which the signal returned to the base line level after clean olive oil was pumped through it. The LOD values for styrene and PCE were 100 and 5 ppb, respectively.

Fleming-Jones and Smith (2003) analyzed PCE, TCE, and other volatile organic compounds in 70 food samples using a purge-and-trap procedure in combination with GC-MS. They used a Tekmar 6,000 thermal desorber with 6016 autosampler and a Varian 3,400 GC interfaced with a Saturn II ion trap mas spectrometer for GC-MS.

In summary, this section describes the typical analytical processes for TCE and PCE in various food samples. Generally, instrumental analysis is preceded by an extraction process. Common of extraction methods include solvent

microextraction, SPME, and SPE, whereas headspace GC is the most popular instrumental method for the analysis of these compounds.

4. Occurrence in foods

TCE and PCE are widely produced because of their usefulness and application potential in several industries. Because of their toxicity, they have been monitored in water, biological, environmental, and food samples. These chemicals have been detected in breast milk, coffee, yoghurt, margarine, olive oil and other food samples. Table 4 summarizes some studies that have detected these contaminants in various food sources.

4.1. Trichloroethylene

Cao et al. (2016) conducted a total diet stuies on the occurrence of volatile organic compounds in foods in Canada. They analyzed 153 composite food samples, and TCE was detected in 31 samples. The mean TCE concentration in the food samples was 0.53 ng g^{-1}, and the highest TCE concentration of 4 ng g^{-1} was found in potato chips.

In a similar study on the presence of TCE and other volatile organic compounds in foods from markets in Belgium, 377 food samples representing 14 food groups were analyzed. TCE was detected in eight food groups, albeit at low concentrations. Among the total food samples analyzed, the highest percentage of occurrence of TCE (9%) was found for cookies and cakes. The total maximum concentration of TCE in all food categories analyzed was 0.2 μg kg^{-1}, with an occurrence of 2% (Vinci et al., 2015).

Miyahara et al. (1995) detected volatile halogenated organic compounds in 13 food samples obtained from 20 families living in Tokyo. The concentration of TCE ranged from not detectible (ND) to 1.7 μg kg^{-1} in cakes, from ND to 0.6 μg kg^{-1} in juice, from ND to 0.5 μg kg^{-1} in lactic beverage, from ND to 1.3 μg kg^{-1} in ice cream, from ND to 0.6 μg kg^{-1} in plain yoghurt, and from ND to 1 μg kg^{-1} in ice milk. The mean TCE concentrations in cakes, juice, lactic beverage, ice cream, plain yoghurt, and ice milk were 0.8, 0.03, 0.03, 0.16, 0.03, and 0.3 μg kg^{-1}, respectively.

A 3-year monitoring study by Doucette et al. (2007) revealed that TCE in groundwater can migrate into residential communities and be uptaken into vegetables and fruits. In 2001, TCE was detected in 167 fruit (0.4-17.9 μg kg^{-1}) and fruit tree core (0.4-7.5 μg kg^{-1}) samples collected from17 private residential areas. In 2002, TCE was not detected above the method detection limit (MDL) in 300 fruit and vegetables sampled, but it was found in a number of fruit tree cores. In 2003, samples were collected repeatedly from five locations over several months, and trends were the same as in 2002; TCE was not detected above the MDL in fruits, but it was detected in tree cores. TCE concentrations in tree cores during the 3-year study were in the range of 0.6-7.8 μg kg^{-1} in walnut and 10.9-103.6 μg kg^{-1} in apple.

Heikes (1987) detected residues of chlorinated solvents, including TCEc in decaffeinated coffees from nine commercial brands. TCE was detected at concentrations up to 44 μg kg^{-1}

Table 4. Occurrence of trichloroethylene & tetrachloroethylene in food samples

Analyte	Foods	Concentration	References
TCE	Yoghurt	<0.34 μg kg^{-1}	Cao et al. (2016)
	Ice cream	<0.92 μg kg^{-1}	
	Butter	<4.1 μg kg^{-1}	
	Ground beef	<0.61 μg kg^{-1}	
	Fresh pork	<0.42 μg kg^{-1}	
	Lamb	<0.83 μg kg^{-1}	
	Eggs	<0.34 μg kg^{-1}	
	Poultry chicken/turkey	<0.25 μg kg^{-1}	
	Fresh water fish	<0.30 μg kg^{-1}	
	Shellfish	<0.43 μg kg^{-1}	
	Coffee	<0.05 μg kg^{-1}	
	Tea	<0.06 μg kg^{-1}	
	Pizza	0.26 μg kg^{-1}	
TCE	Dairy products	0.3 μg kg^{-1}	Vinci et al. (2015)
	Fats & oils	0.1 μg kg^{-1}	
	Ready-to-eat meals	0.1 μg kg^{-1}	
	Sugar & confectionary	0.1 μg kg^{-1}	

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(continued)

Analyte	Foods	Concentration	References
PCE	Non-alcoholic drinks	0.2 μg kg^{-1}	Vinci et al. (2015)
	Sauces	3.4 μg kg^{-1}	
	Dairy products	0.5 μg kg^{-1}	
	Fruits & vegetables	1.8 μg kg^{-1}	
	Meat & meat products	0.2 μg kg^{-1}	
	Fish & fish products	1.7 μg kg^{-1}	
	Cereal products	0.1 μg kg^{-1}	
	Cookies and cakes	2.3 μg kg^{-1}	
	Fats & oils	1.5 μg kg^{-1}	
	Ready-to-eat meals	1.7 μg kg^{-1}	
	Sugar & confectionary	6.0 μg kg^{-1}	
	Egg	0.7 μg kg^{-1}	
TCE	Cakes	0.8 μg kg^{-1}	Miyahara et al. (1995)
	Juice	0.03 μg kg^{-1}	
	Lactic beverage	0.03 μg kg^{-1}	
	Ice cream	0.16 μg kg^{-1}	
	Plain yoghurt	0.03 μg kg^{-1}	
	Ice milk	0.3 μg kg^{-1}	
TCE	Walnut	0.06-7.8 μg kg^{-1}	Doucette et al. (2007)
	Apple	10.9-103.6 μg kg^{-1}	
TCE	Ground decaffeinated coffee	41-44 μg kg^{-1}	Heikes (1987)
	Brew and grounds from decaffeinated ground coffee	13 μg kg^{-1}	
PCE	Breast milk	13-75 μg kg^{-1}	Schreiber et al. (2002)
	Breast milk	31 μg L^{-1} (after 1 month) 2.2 μg L^{-1} (after 4 months)	
PCE	Breast milk	1.0 mg dL^{-1} after 24 hr 0.3 mg dL^{-1} after 44 hr	Bagnell and Ellenberger (1977)
PCE	Wheat and wheat products	0-2.6 μg kg^{-1}	Heikes and Hopper (1986)
	Corn and corn products	0-1.8 μg kg^{-1}	
PCE	Dairy products	5-102 μg kg^{-1}	Fleming-Jones and Smith (2003)
	Meat	2-60 μg kg^{-1}	
	Nuts	7-54 μg kg^{-1}	
	Fruit & vegetables	5-12 μg kg^{-1}	
	Margarine, oils & fats	3-42 μg kg^{-1}	
	Baked goods	2-52 μg kg^{-1}	
PCE	Margarine	1-5 μg kg^{-1}	Entz and Diachenko (1988)
PCE	Cereals	0-108 μg kg^{-1}	Daft (1988)
	Oils	0-21 μg kg^{-1}	
	Nuts	0-120 μg kg^{-1}	
	Fruit & vegetables	0-14 μg kg^{-1}	
	Baked goods	0-48 μg kg^{-1}	
	Meat	0-124 μg kg^{-1}	
	Dairy products	0-30 μg kg^{-1}	

in ground decaffeinated coffee and up to 13 μg kg^{-1} in brews and grounds of decaffeinated ground coffee.

4.2. Tetrachloroethylene

Schreiber et al. (2002) detected PCE in breast milk samples of two nursing mothers who participated in a study to assess PCE exposure levels in residents of an apartment where dry-cleaning services were operated in the USA. Mean PCE concentration in the breast milk of the first mother who had been lactating for 6 weeks before sample collection were in the range of 13-75 μg L^{-1}. The second mother was exposed to PCE throughout her pregnancy but breast milk samples were analyzed 1 and 4 months postpartum, after the dry-cleaning services were discontinued. The PCE concentrations in

her breast milk samples were 31 μg L^{-1} after 1 month and 2.2 μg L^{-1} after 4 months. Similarly, Bagnell and Ellenberg (1977) detected PCE in the breast milk of a nursing mother 24 h after she was briefly exposed to PCE. The concentration after 24 h was 1.0 mg dL^{-1}, but it decreased to 0.3 mg dL^{-1} after 48 h, suggesting a selective concentration of chlorinated hydrocarbons in milk.

Vinci et al. (2015) detected PCE alongside other volatile organic compounds in 377 food samples, representative of 14 food groups, from Belgian markets. PCE was detected at higher levels than TCE in 13 out of 14 groups. The only group in which PCE was not detected were alcoholic drinks. The highest maximum TCE concentration (6.0 μg kg^{-1}) was found in the sugar and confectionary group, and the total maximum TCE concentration in all food categories analyzed was also 6.0 μg kg^{-1}, with an occurrence of 24%.

Heikes and Hopper (1986) detected PCE and other compounds used as fumigants in whole grains, milled grain products, and intermediate grain-based foods. The PCE concentration was in the range of 0-2.6 μg kg^{-1} in wheat and wheat products and 0-1.8 μg kg^{-1} in corn and corn products.

In a 5-year study volatile organic compounds in 70 food samples, Fleming-Jones and Smith (2003) detected PCE at 5-102 μg kg^{-1} in dairy products, at 2-60 μg kg^{-1} in meat, at 7-54 μg kg^{-1} in nuts, at 5-12 μg kg^{-1} in fruit and vegetables, at 3-42 μg kg^{-1} in margarine, oils and fats, and at 2-52 μg kg^{-1} in baked goods.

Entz and Diachenko (1988) detected residues of PCE and other volatile halocarbons in 70 samples of stick, soft, and diet soft margarines in Washington, DC, USA, using headspace chromatography with ECD. The PCE concentration was in the range of 4-5,000 μg kg^{-1} in margarines from a store located next to a dry cleaner and 5- 50 μg kg^{-1} in margarines obtained directly from the producer. The highest concentration of PCE (1-5 μg kg^{-1}) was detected in margarine obtained from a supermarket located next to a dry cleaner.

Daft (1988) analyzed residues of fumigants and industrial chemicals in food samples. In total 10 residues, including PCE, were analyzed in 213 food samples. The concentration range of PCE was in the range of 0-108 μg kg^{-1} in cereals, 0-21 μg kg^{-1} in oils, 0-120 μg kg^{-1} in nuts, 0-14 μg kg^{-1} in fruits and vegetables, 0-48 μg kg^{-1} in baked goods, 0-124 μg kg^{-1} in meat, and 0-30 μg kg^{-1} in dairy products.

Because of the wide production and use of TCE and PCE, these chemicals have been monitored and detected in numerous food samples. Grains, fruits, vegetables, breast milk, cakes, and dairy products have all been found to contain TCE and PCE at various concentration.

5. Risk assessment

Fan et al. (2009) assessed the risk from exposure to 14 volatile organic compounds in groundwater in Taiwan. Using the multimedia environmental pollutant assessment system, they calculated the specific cancer and non-cancer risks at a 1 μg L^{-1} exposure level for each of the compounds investigated. The investigators reported that PCE, along with other two compounds, was associated with the highest non-cancer risk, whereas the specific cancer risk of TCE did not exceed the general guidance value of 10^{-6}. Water ingestion, indoor breathing, and skin absorption during bathing contributed the most to exposure risk, whereas with other absorption routes posed insignificant risks.

Metabolites of TCE and of other parent compounds that produce similar metabolites can exert certain health effects similar to those of TCE. Wu and Schaum (2000) reported that the common exposure route of the general population in the USA to TCE is water ingestion or direct inhalation. Average daily intake was estimated to be 2-20 μg day^{-1} via ingestion and 11-33 μg day^{-1} via inhalation.

Iritas et al. (2021) used methylated arginine biomarkers to assess the risk of cardiovascular diseases from exposure to TCE in 100 exposed and 98 control subjects. They found a strong correlation ($r=0.453$, $p<0.01$) between trichloroacetic acid, a urinary metabolite of TCE, and asymmetric dimethyl arginine, which is a classical risk factor and marker of cardiovascular diseases when present at increased levels. The author concluded that chronic exposure to TCE poses a risk of cardiovascular and other heart diseases.

Drinking water is a common route of exposure to PCE, and certain toxic effects in adults have been reported. Aschengrau et al. (2015) assessed the possible health risks in adults who had been exposed to PCE during gestation or early childhood. The study revealed a 1.8-fold increase in the incidence of cancer, particularly cervical cancer, and a 1.5-fold increase in the incidence of epilepsy in individuals exposed to PCE through contaminated drinking water early in life.

Although results from pharmacokinetic analyses can be inconsistent (lower resultant risks and higher permissible

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exposure), they have been used in calculations for risk assessment, and to assess the risk of PCE carcinogenicity in mice, rats, and humans (Bois et al., 1990). The median cancer risk estimate for humans exposed consistently to 1 ng L^{-1} of PCE in air was found to be 1.6 per million, and 0, 0.04, 2.8, and 6.8 per million for the 5th, 25th, 75th, and 95th percentiles, respectively, when considering the uncertainty in the model parameters (Bois et al., 1990).

6. Conclusions

TCE and PCE are polychlorinated volatile organic compounds that have wide industrial applications. As a result, they are common contaminants found in the environment particularly in the air, water, and foods. They have been categorized as carcinogenic, and have a wide range of other toxic effects. Following extraction from their matrix, chromatography is the most common method used to detect of detecting these contaminants. Humans typically come into contact with these toxins via direct inhalation or drinking water, both of which pose certain health risks. In this review, we assessed the toxicity and analytical methods of TCE and PCE, as well as their occurrence in foods and risk assessment.

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Conflict of interests

The authors declare no potential conflicts of interest.

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