



# TRICHLOROETHYLENE (TCE) TOXICITY

## *Environmental Alert*

- Trichloroethylene (TCE) is a common industrial solvent and contaminant of hazardous waste sites, groundwater, and drinking water.
- TCE is a central nervous system depressant and a suspected hepatotoxin in humans.
- The U.S. Environmental Protection Agency considers TCE an animal carcinogen and a potential cancer hazard to humans.

*This monograph is one in a series of self-instructional publications designed to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients. This course is also available on the ATSDR Web site, [www.atsdr.cdc.gov/HEC/CSEM/](http://www.atsdr.cdc.gov/HEC/CSEM/). See page 3 for more information about continuing medical education credits, continuing nursing education units, and continuing education units.*



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**Disclaimer**

The state of knowledge regarding the treatment of patients potentially exposed to hazardous substances in the environment is constantly evolving and is often uncertain. In this monograph, ATSDR has made diligent effort to ensure the accuracy and currency of the information presented, but makes no claim that the document comprehensively addresses all possible situations related pediatrics and environmental health. This monograph is intended as a resource for pediatricians and other child health care providers in assessing the condition and managing the treatment of patients potentially exposed to hazardous substances. It is not, however, a substitute for the professional judgment of a health care provider. The document must be interpreted in light of specific information regarding the patient and in conjunction with other sources of authority.

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# Case Studies in Environmental Medicine (CSEM): Trichloroethylene (TCE) Toxicity

## Goals and Objectives

The goals of this CSEM are to increase the knowledge of health care providers, especially pediatricians, of the special susceptibilities of children to hazardous substances in the environment and to aid in their evaluation of potentially exposed patients.

After completion of this educational activity, the reader should be able to discuss the major exposure routes for trichloroethylene, describe two potential environmental and occupational sources of exposure to trichloroethylene, state two reasons why trichloroethylene is a health hazard, describe factors contributing to trichloroethylene toxicity, identify evaluation and treatment protocols for persons exposed to trichloroethylene, and list two sources of information on trichloroethylene.

## Instructions

See page 4

## Accreditation

### Continuing Medical Education (CME)

The Centers for Disease Control and Prevention (CDC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. CDC designates this educational activity for a maximum of 1.5 hours in category 1 credit toward the American Medical Association (AMA) Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

### Continuing Nursing Education (CNE)

This activity for 1.6 contact hours is provided by CDC, which is accredited as a provider of continuing education in nursing by the American Nurses Credentialing Center's Commission on Accreditation.

### Continuing Education Units (CEU)

CDC has been approved as an Authorized Provider of continuing education and training programs by the International Association for Continuing Education and Training and awards 0.1 continuing education units (CEUs).

The questionnaire and posttest must be completed and returned electronically, by fax, or by mail for eligibility to receive continuing education credit.

### Instructions for Completing CSEM Online

1. Read this CSEM, *Trichloroethylene (TCE) Toxicity*; all answers are in the text.
2. Link to the MMWR/ATSDR Continuing Education General Information page ([www.cdc.gov/atsdr/index.html](http://www.cdc.gov/atsdr/index.html)).
3. Once you access this page, select the Continuing Education Opportunities link.
4. Once you access the MMWR/ATSDR site online system, select the electronic file and/or register and test for a particular ATSDR course.
  - a. Under the heading “Register and Take Exam,” click on the test type desired.
  - b. If you have registered in this system before, please use the same login and password. This will ensure an accurate transcript.
  - c. If you have not previously registered in this system, please provide the registration information requested. This allows accurate tracking for credit purposes. Please review the CDC Privacy Notice ([www.cdc.gov/privacy.htm](http://www.cdc.gov/privacy.htm)).
  - d. Once you have logged in/registered, select the test and take the posttest.
5. Answer the questions presented. To receive continuing education credit, you must answer all of the questions. Some questions have more than one answer. Questions with more than one answer will instruct you to “indicate all that are true.”
6. Complete the course evaluation and posttest no later than **January 29, 2004**.
7. You will be able to immediately print your continuing education certificate from your personal transcript.

### Instructions for Completing CSEM on Paper

1. Read this CSEM, *Trichloroethylene (TCE) Toxicity*; all answers are in the text.
2. Complete the evaluation questionnaire and posttest, including your name, mailing address, phone number, and e-mail address, if available.
3. Circle your answers to the questions. To receive your continuing education credit, you must answer all of the questions.
4. Sign and date the posttest.
5. Return the evaluation questionnaire and posttest, no later than **December 30, 2003**, to CDC by mail or fax:

<b>Mail</b>	<b>or</b>	<b>Fax</b>
Continuing Education Coordinator		404-498-0061
Division of Health Education and		ATTN: Continuing Education Coordinator
Promotion, ATSDR		
1600 Clifton Road, NE (MS E-33)		
Atlanta, GA 30333		
6. You will receive an award certificate within 90 days of submitting your credit forms. No fees are charged for participating in this continuing education activity.

## Case Study

Your practice is in a suburban community with a number of high-technology industries. A couple for whom you have been the family physician asks for an appointment to discuss their daughter's illnesses and a matter of concern to them.

During the initial consultation, the mother reports that they are living in an area supplied by municipal well water. They recently received a notice from the municipal water district stating that their drinking water contains 100 parts per billion (ppb) trichloroethylene (TCE), and as a precaution, they are being supplied with bottled drinking water until an alternative well can be put into service. The notice indicates that the well water is suitable for bathing and laundering. The father indicates that he is familiar with TCE; it is used in the electronics plant where he works.

The daughter, aged 4, has had a number of ear infections during her first 2 years, culminating in a myringotomy at age 3. Follow-up by an ear, nose, and throat specialist has shown normal hearing. Although there have been no further infections, the mother stresses that her daughter seems to have a greater number of colds than her classmates and "has not seemed as healthy as she should be." However, the daughter's chart does not reflect an unusual number of office visits or calls. The mother also notes that the child's day-care center is next to "some kind of machine shop" where a chemical odor has been noticed recently. Several of the children and one of the teachers have complained of eye and throat irritation in association with the odor.

The mother, who is 33 years old, then reveals that she might be pregnant and that she has had mild nausea for 1 week. It has been 8 weeks since her last menstrual period. Both parents are concerned about the possibility that the TCE in the drinking water might have affected the fetus. Although this pregnancy was planned, they might consider terminating the pregnancy if the baby was likely to be "damaged." They are also concerned that the entire family might suffer from cancer or other diseases in the future.

Before receiving bottled water, the family drank tap water when thirsty and made coffee with tap water. The family also used tap water for cooking and for brushing their teeth, and it is still used for bathing. They have never noticed discoloration or an off-taste to the tap water. They encourage their child to drink water instead of sodas during the summer and estimate that the amount of water each of them consumes is 2 to 3 glasses a day.

You schedule each parent and the child for an individual office visit.

### **The concerns of a young family exposed to trichloroethylene-contaminated drinking water**

#### ***Pretest***

- (a) *What would you include on the mother's and daughter's problem lists?*
- (b) *What additional information would you seek before seeing the family again?*
- (c) *What reassurances might you provide at the end of this initial visit?*

## Who's at Risk

- Workers in metal-fabricating and cleaning operations have the greatest likelihood of exposure to high concentrations of TCE.
- Because TCE inhalation can cause euphoria, deliberate abuse can occur.
- Some people might be predisposed to developing ventricular fibrillation or asystole after exposure to high concentrations of TCE.

Most significant exposures to TCE occur in the workplace. The National Institute for Occupational Safety and Health (NIOSH) has estimated that 3.5 million workers in the United States are exposed to TCE, with the majority of high exposures ascribed to metal-degreasing operations. Deaths have occurred in workers who were accidentally exposed to high levels of TCE, and in solvent abusers deliberately sniffing typewriter correction fluid from plastic bags or in enclosed spaces. Some of these deaths were due to asphyxia, whereas others were attributed to either ventricular fibrillation or asystole. Although no human studies have directly assessed potential dysrhythmogenic effects of TCE, no evidence exists to show that persons exposed to TCE at background environmental concentrations or at allowable workplace levels are at increased risk of developing cardiac dysrhythmias.

Until 1977, when certain uses were banned, TCE was used as an inexpensive, nonflammable, and self-administered obstetrical anesthetic (Tri-lene). It was discovered that alkali in rebreathing systems could lead to the production of dichloroacetylene, which produces cranial nerve injuries. Workers in environments containing this TCE-decomposition product could also be at risk of developing trigeminal, optic, or facial nerve effects.

- Ingestion of alcohol can potentiate the central nervous system depressant effects of TCE.
- TCE crosses the placenta and can accumulate in the fetus.

Alcohol potentiates TCE's effects on the central nervous system (CNS). Concurrent alcohol consumption and exposure to TCE can result in "degreaser's flush," a temporary redness and itching of the back, neck, and face. Theoretically, liver dysfunction or disulfiram (Antabuse) treatment could reduce the metabolism of TCE and thus increase its CNS depressant effects.

TCE is one of the volatile organic contaminants most frequently found in groundwater. The possibility of an association between ingested TCE and long-term effects, including malignancies, has been raised, but scientific evidence proving that these effects are due to TCE exposure is lacking. TCE rapidly crosses the placenta in both humans and animals, and can accumulate in the fetus. To gather information on the health effects of ingesting TCE-contaminated water, the Agency for Toxic Substances and Disease Registry (ATSDR), in cooperation with the states, has established a national registry. This registry is discussed in the Sources of Information section.

### Challenge

- (1) Which members of the family described in the case study are at increased risk for adverse effects from TCE? Explain.

# Exposure Pathways

Trichloroethylene or TCE ( $\text{Cl}_2\text{C}=\text{CHCl}$ ) is a clear, colorless, nonflammable liquid that has a sweet, fruity odor characteristic of chloroform. The odor threshold is approximately 100 parts per million (ppm). For some workers, TCE's odor might not be detectable at concentrations near the American Conference of Governmental Industrial Hygienists (ACGIH) recommended threshold limit value of 50 ppm or the Occupational Safety and Health Administration (OSHA) limit of 100 ppm (both determined by an 8-hour time-weighted average), and so might not provide adequate warning of its presence.

Synonyms for trichloroethylene include TCE, Tri, trichloroethene, acetylene trichloride, and ethylene trichloride. Trade names for this industrial solvent include Benzinol, Circosolve, Flock Flip, Narcogen, Perm-A-Chlor, Triclene, and Vestrol.

TCE does not occur naturally; therefore, its presence indicates manufacture, use, or storage. Estimated use patterns suggest that 80% of TCE is used for vapor degreasing of fabricated metal parts in the automotive and metal industries. Consumer products that contain TCE include typewriter correction fluids, paint removers and strippers, adhesives, spot removers, and cleaning fluids for rugs. Before its ban for certain applications in 1977, TCE was also used as a general (mostly obstetric) anesthetic, grain fumigant, disinfectant, pet food additive, and extractant of spices in foods and caffeine in coffee.

Occupational exposures might occur in chemical industries that manufacture polyvinyl chloride, pentachloroethane, and other polychlorinated aliphatic hydrocarbons, flame-retardant chemicals, and insecticides where TCE is a chemical intermediate. Other potential exposures occur in manufacturing processes of disinfectants, pharmaceuticals, dyes, perfumes, and soaps. Mechanics, oil processors, printers, resin workers, rubber cementers, shoemakers, textile and fabric cleaners, tobacco denicotinizers, varnish workers, and dry cleaners also have increased likelihood of TCE exposure, although most dry cleaners now use tetrachloroethylene (perchloroethylene) or 1,1,1-trichloroethane.

In the workplace, TCE is seldom present as a pure substance. Industrial-grade TCE contains small amounts of stabilizers in the form of antioxidants or acid receptors; total chemical impurities usually do not exceed 0.1% by weight. Decomposition of TCE into dichloroacetylene (a neurotoxic compound) and phosgene (a serious pulmonary irritant) occurs in the presence of alkali at temperatures above 60°C for the unstabilized compound and above 130°C for the stabilized compound.

- The most common sources of nonoccupational exposure to TCE are ambient air and drinking water.



Because of its widespread use, TCE has become a common environmental contaminant. Contamination results from evaporative losses during use; discharge to surface waters and groundwater by industry, commerce, and individual consumers; leaching from hazardous waste landfills into groundwater; and from the incidental addition of TCE during food production.

In the atmosphere, TCE is destroyed by photooxidation, with a half-life of 3–8 days during the summer months and approximately 2 weeks in cold climates during the winter. This relatively short half-life significantly limits the transport of TCE in air; however, the continual volatilization of TCE from emission sources or contaminated surface waters ensures its persistence in air. Examination of arctic air between 1982 and 1983 demonstrated mean TCE levels of 8 parts per trillion (ppt) to 9 ppt. This compares to mean concentrations of 30 ppt TCE in rural or remote areas, 460 ppt in urban and suburban areas, and up to 1,200 ppt in areas nearest emission sources. Indoor air concentrations have ranged from 140 ppt in a school to 5,000 ppt in an office building. Surveys have detected TCE in at least 8,612 of 1,428 hazardous waste sites on the National Priorities List of the U.S. Environmental Protection Agency (EPA), with a maximum level of 12,300 ppt TCE in the ambient air at one New Jersey site.

TCE in drinking water is a result of its rapid leaching from landfills and its discharge from industrial wastewaters. TCE volatilizes quickly from water depending on temperature, water movement, and aeration. The biodegradation of TCE under anaerobic conditions is slow, making TCE relatively persistent in subsurface waters. An EPA national groundwater survey detected TCE in approximately 10% of the wells tested. It is the most frequently detected organic solvent in groundwater supplies, and is estimated to be in up to 34% of the nation's drinking water supplies.

Because of TCE's volatility, household activities such as bathing, laundering, and cooking with contaminated water can produce TCE air concentrations above ambient levels. Both natural and processed foods can contain TCE because of direct uptake through the environment, contamination of water used in food processing, and contamination by solvents used in cleaning food processing equipment. Most processed foods examined contain levels of a few parts per billion. Studies indicate that TCE has a low tendency to bioaccumulate in the food chain.

### ***Challenge***

- (2) *What are the possible sources of exposure to TCE for the family described in the case study?*



## Biologic Fate

At 100 to 200 ppm TCE, the lungs of human volunteers absorbed approximately 50% of an inhaled dose during the first 30 minutes of exposure. Case reports of human poisoning after ingestion of TCE indicate that gastrointestinal absorption is also substantial.

Once absorbed, TCE is rapidly cleared from the blood. Because of its lipid solubility, TCE accumulation occurs in organs containing high levels of adipose tissue. Data from human studies indicate that body fat and the liver accumulate the greatest portion of absorbed TCE.

In humans, TCE is metabolized primarily in the liver by the mixed-function oxidase system that probably converts TCE to an oxide (epoxide). Subsequently, this reactive intermediate might rearrange to trichloroacetaldehyde and then to chloral hydrate, the latter forming the trichloroethanol and trichloroacetic acid metabolites excreted in the urine after TCE exposure. At levels of 54 to 140 ppm TCE, human volunteers metabolized approximately 90% of an inhaled dose. No studies have provided evidence of saturation of TCE metabolism in humans, at least for short-term inhalation exposure to high concentrations (i.e., about 300 ppm). Kinetic modeling shows a plateau or steady state in metabolism with repeated exposure.

A relatively small amount of absorbed TCE is exhaled unchanged; most of an absorbed dose is metabolized and excreted in the urine. After exposure to air concentrations of between 100 and 200 ppm, approximately 30% to 50% of an absorbed dose appears in urine as trichloroethanol, and about 10% to 30% appears as trichloroacetic acid. The time between TCE inhalation and urinary excretion of trichloroethanol is relatively short (biologic half-life approximately 10 hours) compared to the urinary excretion of trichloroacetic acid (biologic half-life approximately 52 hours). Trichloroacetic acid is theoretically detectable in urine for at least a week after TCE exposure.

Species differences in TCE metabolism might explain observed differences in susceptibility to specific TCE-related diseases. Liver cancer, for example, occurs mainly in strains of mice that generate high levels of trichloroacetic and dichloroacetic acids as TCE metabolites in liver cells. By contrast, rats that metabolize more TCE via glutathione conjugation are prone to renal cancer. Because of such species-specific effects, caution must be used when extrapolating adverse effects from experimental animals to humans.

- Pulmonary and gastrointestinal absorption of TCE is rapid.
- TCE metabolism occurs mainly in the liver.
- Because of species differences in TCE metabolism, caution should be used in extrapolating adverse effects to humans.

### Challenge

- (3) *On the next visit to your office, the mother states that some families in their neighborhood are being seen by another practitioner, who has sent specimens to a laboratory for measurement of indicators of TCE exposure. What biologic indicators of TCE exposure are likely being measured?*
- (4) *If biologic measurements are performed, what considerations should be taken into account to properly interpret the results?*

## Physiologic Effects

Some occupational studies have shown that TCE produces CNS effects; mucous membrane, skin, and gastrointestinal irritation; decreased appetite; and headache. Hepatotoxicity has been associated primarily with TCE inhalation and ingestion of very large amounts. Renal failure has been reported in concert with confirmed hepatic damage. Cardiac dysrhythmias may be induced by heavy TCE exposure in susceptible persons.

### Central Nervous System Effects

- CNS depression is the most prominent effect of acute TCE exposure.

TCE-induced CNS symptoms depend on both concentration and exposure duration. In one study of human volunteers, exposure to TCE air levels of 27 ppm for 1 to 4 hours caused drowsiness and mucous membrane irritation; at 81 ppm it caused headaches. In another study, drowsiness, lethargy, and nausea were noted within 5 minutes at anesthetic concentrations of 2,000 ppm. TCE presumably causes anesthesia by affecting cell membranes and altering neuronal transmission. Symptoms due to short-term exposures typically resolve within a few hours of exposure.

In a study of 50 workers employed from 1 month to 15 years in various industrial cleaning and degreasing operations using TCE, complaints due to chronic exposure included decreased appetite, sleep disturbances, ataxia, vertigo, headache, short-term memory loss, and a reduced number of word associations. Greater frequency of symptoms was noted in workers exposed to higher (85 ppm) than lower (14 ppm) mean TCE concentrations.

- Chronic occupational TCE exposure has been associated with neurologic abnormalities.

Some of the observed neurologic effects from long-term exposure to TCE indicate impaired trigeminal nerve function (e.g., blink reflex and masseter reflex). In the brains of animals chronically exposed to high concentrations of TCE (1,000 to 3,000 ppm), histologic changes have been demonstrated. Persons who have deliberately abused volatile chlorocarbon solvents have developed cerebellar damage and ataxia.

## Cardiovascular Effects

Mortality studies of TCE-exposed workers do not indicate an increased risk of cardiovascular death. A few susceptible persons who are exposed to near-anesthetic levels during vigorous activity might have increased risk of cardiac dysrhythmia. However, no evidence shows that exposure at high TCE levels causes a predisposition to cardiac toxicity at lower levels. When TCE was administered as an anesthetic agent, serious ventricular dysrhythmias and cardiac arrests were rare and were nearly always associated with hypoxia. Significant ventricular ectopy would not be expected from TCE exposure at background environmental levels or those currently allowed in the workplace.

- Death due to cardiac dysrhythmia related to sensitization of the myocardium to catecholamines in TCE-exposed workers has been associated with high doses in conjunction with vigorous physical activity.

## Gastrointestinal and Renal Effects

When swallowed, TCE causes gastrointestinal (GI) irritation, with possible inflammation of the GI tract, manifested as nausea, vomiting, diarrhea, and abdominal pain. Hepatotoxicity has been associated primarily with intentional TCE inhalation abuse. In these cases, hepatic histologic examination has revealed centrilobular necrosis with fatty infiltration. Chronic TCE exposures at concentrations currently permissible in the workplace or at those expected in ambient air are not likely to cause liver damage.

- Case reports associate liver damage with inhalation of high doses of TCE.
- Renal toxicity has been described in the literature but would not be expected under ambient air exposure conditions.

TCE-induced renal failure in humans has been reported, albeit infrequently, and usually in concert with confirmed hepatic damage. One case involved a long-time metal degreaser who developed acute tubular necrosis (confirmed by biopsy), which led to renal failure. Another case involved a worker who was exposed to TCE for 8 hours and who developed allergic interstitial nephritis with secondary tubular necrosis. Animals demonstrate little nephrotoxicity after single, high-dose exposures.

## Reproductive and Developmental Effects

No increased incidence of congenital malformations has been detected in babies born to mothers occupationally exposed to TCE. One retrospective occupational study suggested an increased risk of spontaneous abortion in women exposed to TCE, but the result was not statistically significant, and the effect disappeared when odds ratios were adjusted for potential confounders. A small cross-sectional study of degreasing workers showed no effect of TCE exposure on male germ cells. Data from animal studies reveal no adverse effects on reproductive system histology, fertility, or other reproductive performance parameters.

- Limited studies in workers have not detected significant reproductive or developmental abnormalities due to TCE exposure.

TCE crosses the placenta in animals and has been found in human newborns after maternal TCE anesthesia during childbirth. Human congenital defects were attributed to the ingestion of TCE-contaminated water in two studies, but the significance of these findings is questionable because of mixed chemical exposures and methodologic inadequacies of the studies. In

animals, some abnormalities (decreased fetal body weight, ossification anomalies, and cardiac defects) have been reported infrequently. Evidence from animal studies, together with the limited information from human studies, suggests that developmental effects might be of concern.

## Carcinogenic Effects

- Epidemiologic studies of TCE-exposed persons to determine cancer risk are suggestive but inconclusive.

Several epidemiologic studies have evaluated carcinogenic effects from TCE exposure. Some have found significant increases in certain types of cancer (stomach, liver, prostate, kidney, and non-Hodgkin lymphoma), whereas other studies did not find any evidence that TCE is carcinogenic to humans. All of these studies have limitations, and firm conclusions on the carcinogenic risk from TCE are not possible.

Inhalation or oral exposure to high doses of TCE produces liver and lung tumors in mice, and renal adenocarcinomas, testicular tumors, and possibly leukemia in rats. The presence of TCE stabilizers, such as epichlorohydrin, may also confound some of these results. These studies indicate that mice are more susceptible than rats to TCE carcinogenicity.

Most epidemiologic studies of workplace exposures to TCE have not demonstrated a significant increase in the incidence of cancer. A 1997 follow-up study of workers found excesses of some cancers, but there was no control for potential confounding factors, and those workers with higher exposure levels generally did not have higher cancer rates than those with lower exposures, which argues against a dose-response effect. The significance of this study has yet to be confirmed. Some inconsistencies between results of animal and human studies might be due to metabolic saturation and the formation of reactive intermediates that occur in animals exposed to high TCE levels but not in humans after low-level exposure.

The cancer classification of TCE is under review by EPA. The International Agency for Research on Cancer has concluded that limited evidence exists in humans for the carcinogenicity of TCE, but sufficient evidence in experimental animals.

## Other Effects

- TCE is a mild respiratory tract irritant and can produce contact dermatitis.

TCE produces minimal irritation of the respiratory tract except at concentrations that exceed current workplace standards. Use of TCE in anesthetic concentrations does not damage the pulmonary system. TCE is not a sensitizing agent, and bronchospasm is unlikely to occur except in highly susceptible persons after exposure to high concentrations. There are only a few reports of patch tests being positive.

Like other organic solvents, TCE may produce contact dermatitis, rashes, and burns. The defatting dermatitis resulting from prolonged contact may reduce resistance to skin infections. An irritant reaction resembling an

exfoliative dermatitis or scarlatiniform reaction can occur from dermal contact with contaminated clothing.

A syndrome called degreaser's flush has been associated with the interaction of ingested ethanol and inhaled TCE. Typically, erythema resulting from vasodilation develops around the face, back, and shoulders within 30 minutes and resolves within an hour of appearance.

No deleterious effects on the immune system have been noted in persons exposed to TCE through environmental sources.

### **Challenge**

- (5) *The father says that he has felt increasingly tired and easily fatigued for the past few months. Results of his physical examination are entirely within normal limits. What tests, if any, would you order?*
- (6) *The mother's obstetrician calls 1 month later. Examination, including sonogram, is normal for her stage of pregnancy. The obstetrician asks you about the potential fetotoxicity of TCE and whether a more invasive evaluation (amniocentesis or chorionic villus biopsy) is indicated. What is your response?*

## **Clinical Evaluation**

### **History and Physical Examination**

When considering the human health effects of TCE, it is important to make a distinction between occupational exposures to relatively high levels by inhalation and general environmental exposures to low levels in drinking water and ambient air. Very rarely has low level exposure been associated with clinical problems. It is of concern only because of the potential for long-term delayed effects such as cancer and possibly as a very weak mutagen.

No unique pattern of symptoms characterizes TCE-induced illness. An occupational history should be routinely obtained and should include items such as company name and location, job title, description of chemical processes encountered, known toxic agents used, workplace investigations, and complaints of co-workers. An environmental history should also be obtained, including location and duration of residence, proximity to industry, diet, daily activities, type of water supply, and use of consumer products that contain TCE.

The patient's complaints should be identified in terms of onset, duration, and intensity. Complaints should be investigated by focusing first on major organ systems that are likely to be affected by exposure to TCE (CNS, hepatic,

- TCE exposure produces no unique clinical clues.

integumentary, cardiovascular, and renal), and then on systems unlikely to be affected (respiratory, gastrointestinal, endocrine, and skeletal). Patients should receive a complete neurologic examination, including a mental status exam and an evaluation of the cranial nerves, to detect either peripheral or CNS involvement. Cranial neuropathies in patients with a history of TCE exposure are uncommon. Presence or absence of an irregular pulse or abnormal cardiac auscultation should be noted. The patient's abdomen should be palpated for hepatomegaly and right upper quadrant tenderness.

## Signs and Symptoms

### Acute Exposure

- Respiratory depression can result from acute, high-dose TCE exposure.

With inhalation of high concentrations, TCE causes initial CNS excitation followed by CNS depression. Depending on the duration and intensity of exposure, symptoms can include drowsiness, dizziness, visual disturbances, light-headedness, fatigue, headache, lethargy, confusion, ataxia, and stupor. Coma and respiratory depression may occur with prolonged, high-level exposure (i.e., above 2,000 ppm). Serious ventricular dysrhythmias can develop up to 24 hours after large TCE ingestions.

After any type of acute exposure, the clinician should carefully assess the adequacy of ventilation, because respiratory depression is the most common serious sequela of acute TCE exposure. Because of possible dysrhythmias, patients with preexisting cardiovascular disease should be monitored by continuous electrocardiogram and frequent evaluation of vital signs. Because hepatic injury may occur, liver function tests should be performed.

### Chronic Exposure

- At permissible workplace levels, CNS symptoms of TCE exposure are usually nonspecific and transient.

Reported neurologic effects associated with chronic workplace exposure to TCE have included nonspecific symptoms such as headache, ataxia, decreased appetite, sleep disturbances, fatigue, weakness, dizziness, memory loss, emotional instability, or impaired judgment. However, study design defects (e.g., exposure data that do not allow for differentiation of acute and chronic effects, failure to analyze confounding variables, lack of controls, and observer bias) limit the conclusion that chronic TCE exposure may cause these effects.

Although some CNS symptoms can disappear within several weeks after cessation of exposure, other CNS adverse health effects such as memory loss and mood swings may persist in persons who have been exposed to TCE for long periods. Persistent neurologic symptoms should also prompt a search for exposure to other potential neurotoxicants, such as drugs of abuse, including alcohol, or psychiatric disorders.



# Laboratory Evaluation

## Direct Biologic Indicators

Data are limited for interpreting TCE levels in plasma. Detectable plasma levels of TCE in persons without occupational exposure are approximately 0.01 to 0.13 micrograms per deciliter ( $\mu\text{g/dL}$ ). Although TCE disappears rapidly from the blood, metabolites (e.g., trichloroacetic acid) can persist in the blood for several weeks and in urine for up to 3 weeks after heavy exposure. The presence of TCE metabolites should be interpreted with caution because some medications (chloral hydrate and disulfiram) and other chlorinated hydrocarbons (1,1,1-trichloroethane and tetrachloroethylene) are also metabolized to trichloroacetic acid and excreted in the urine.

- TCE can be detected in the breath and urine up to 16 hours after exposure; metabolites can persist for a week or more.
- Urinary metabolites are trichloroethanol and trichloroacetic acid.

## Indirect Biologic Indicators

Biochemical abnormalities are uncommon after acute TCE exposures. Rarely have elevations of serum hepatic transaminases (serum glutamic-oxaloacetic transaminase [SGOT] or aspartate aminotransferase [AST], serum glutamic-pyruvic transaminase [SGPT] or alanine aminotransferase [ALT]), bilirubin, and creatinine resulted from acute TCE exposure; nevertheless, liver and kidney function and serum creatinine tests should be performed to establish baselines. Electrocardiogram and continuous cardiac monitoring should be considered for heavily exposed persons. Ingestion of large amounts of TCE, which can cause profuse diarrhea, can produce an electrolyte imbalance. Because the trigeminal, optic, and facial nerves can be impaired by exposure to dichloroacetylene, changes in the visual fields and trigeminal nerve potentials can be noted.

- Liver function tests, a serum creatinine test, and continuous cardiac monitoring should be considered for persons acutely exposed to high levels of TCE.

## Challenge

- (7) *You evaluate the 4-year-old child. A review of her history reveals three to four episodes of otitis media, which were treated with ampicillin, in each of the last 3 years. The child was placed on continuous prophylactic antibiotics during the last two cold seasons. Last year, the child developed additional infections despite the antibiotic regimen, and you referred her to an otolaryngologist, who performed a myringotomy and tympanostomy without incident. The mother estimates the child has had four episodes of coryza or mild influenza last year, with about 7 days of illness that merited staying home from day care. Does this pattern reflect compromise of the child's immune system?*
- (8) *The mother asks about immune system tests. A health care practitioner evaluating other families has performed such tests. Is the assessment of immunocompetence appropriate in this case?*



# Treatment and Management

## Acute Exposure

- Removal from the source and supportive care is the recommended treatment for acute TCE exposure.

In the case of dermal contact with liquid TCE, contaminated clothes should be removed and the affected areas washed with copious amounts of soap and water. Direct eye splashes require irrigation for at least 15 minutes. Corneal epithelium damage usually resolves spontaneously after irrigation.

Patients should be removed from the contaminated environment as soon as possible; begin artificial ventilation, if needed. Those with altered mental status or apparent respiratory insufficiency should receive supplemental oxygen. If the patient's pulse is absent, cardiopulmonary resuscitation should be initiated.

Gut decontamination (emesis, lavage, or saline cathartic) is recommended if it can be initiated within 2 to 3 hours after the ingestion of more than a swallow of TCE. However, the effects of these measures have not been clinically evaluated. If emesis is considered, administer the emetic only to patients who are fully conscious and have an intact gag reflex. Activated charcoal has not been proven to absorb TCE, but in general, it effectively decreases absorption of most ingested toxic agents. No data are available on the ability of hemodialysis or hemoperfusion to increase TCE elimination. No specific antidotes exist.

Patients with serious TCE toxicity should be monitored for the possible development of dysrhythmias. When diarrhea is present, monitor for the development of electrolyte abnormalities and screen for the possible development of hepatorenal dysfunction. Sequelae are unusual in acute exposures.

## Chronic Exposure

- Symptomatic treatment is recommended for chronic TCE exposure.

No known treatment for chronic exposure to TCE exists. Potentially involved organ systems should be independently evaluated, and supportive measures should be initiated.

# Standards and Regulations

## Workplace

### Air

- OSHA's current permissible exposure limit is 100 ppm.

The OSHA permissible exposure limit (PEL) is a time-weighted average (TWA) of 100 ppm, with 300 ppm TCE as a 5-minute maximum peak allowable in any 2-hour period. The National Institute for Occupational Safety and Health considers TCE a potential occupational carcinogen and recommends an exposure limit of 2 ppm (as a 60-minute ceiling) during the

usage of TCE as an anesthetic agent (TCE is no longer used as an anesthetic agent) and 25 ppm as a 10-hour TWA during all other exposures. ACGIH recommends an 8-hour TWA of 50 ppm and a short-term exposure limit (STEL) of 100 ppm. The TWA concentration for a normal 8-hour workday and 40-hour work week is set at a level at which nearly all workers may be repeatedly exposed without adverse effects. The STEL for TCE is a concentration at which workers can be exposed continuously for a short period of time (usually 15 minutes) without suffering irritation, chronic irreversible tissue damage, or narcosis. Table 1 summarizes current standards and regulations for TCE exposure.

Biologic exposure indices are recommended by ACGIH and might involve either direct or indirect measures of individual worker exposure. Free trichloroethanol in the blood can be measured, but a number of other compounds affect the level of trichloroethanol and must be considered as alternate explanations for elevated levels.

Alternatively, a concentration of 100 milligrams (mg) of trichloroacetic acid per gram of creatinine in urine at the end of the work week reflects the upper biologic limit for TCE exposure. Urinary trichloroacetic acid levels can be increased by the same compounds that affect blood trichloroethanol levels. Because of large individual variations, a urinary trichloroacetic acid level of 100 mg per gram of creatinine should be used only as a “warning” level or mean for a group of workers.

**Table 1. Standards and Regulations for Trichloroethylene**

Agency	Focus	Level*	Comments
American Conference of Governmental Industrial Hygienists	Air: workplace	50 ppm 100 ppm	Advisory; TLV/TWA <sup>†</sup> Advisory; STEL <sup>‡</sup>
National Institute for Occupational Safety and Health	Air: workplace	25 ppm	Recommendation; 10-hour TWA <sup>§</sup> ; potential carcinogen
Occupational Safety and Health Administration	Air: workplace	100 ppm 300 ppm	Regulation; PEL <sup>¶</sup> over 8-hour workday Regulation; 5-minute maximum peak in any 2-hour period
U.S. Environmental Protection Agency	Air: environment Drinking water	Not available 5 ppb	Not available Regulation

\* ppm: parts per million; ppb: parts per billion.

<sup>†</sup> TLV/TWA (threshold limit value/time-weighted average): time-weighted average concentration for a normal 8-hour workday or 40-hour work week to which nearly all workers may be repeatedly exposed.

<sup>‡</sup> STEL (short-term exposure limit): maximum level allowed in any 15-minute sampling period.

<sup>§</sup> TWA (time-weighted average): concentration for up to a 10-hour workday during a 40-hour work week.

<sup>¶</sup> PEL (permissible exposure limit): highest level averaged over an 8-hour workday to which a worker may be exposed. Note: A PEL of 50 ppm was enacted by the Occupational Safety and Health Administration in 1989, but that level, along with 375 others, was vacated for procedural reasons by the 11th Circuit Federal Court in 1993.

## Environment

Environmental exposures to TCE are generally low and are decreasing because limitations have been imposed on its use as an anesthetic, solvent extractant, fumigant, and dry-cleaning agent. TCE has a short atmospheric half-life (less than 7 days) and is not likely to bioaccumulate in the food chain. The World Health Organization recommended drinking water limit is 30 µg TCE/liter (L) of water (30 ppb); EPA has set a maximum contaminant level of 5 µg/L (5 ppb) in drinking water. Although there are no known human health effects associated with exposures to environmental levels of TCE, there are limited and controversial data suggesting possible human health effects. More comprehensive information is necessary for a final assessment.

### *Challenge*

- (9) *TCE has been identified as the irritant at the day-care center. The mother described in the case study is concerned and wishes to take action to get the level reduced. What can you recommend to her?*

## Suggested Reading List

### General

American College of Physicians. 1989. Clinical ecology. *Ann Intern Med* 2:168–78.

Bell DM, Gleiber DW, Mercer AA, Phifer R, Guintier RH, Cohen AJ, et al. 1989. Illness associated with child day care: a study of incidence and cost. *Am J Public Health* 79:479–84.

Fan AM. 1988. Trichloroethylene: water contamination and health risk assessment. *Rev Environ Contam Toxicol* 101:55–92.

Feldman RG, White RF, Currie JN, Travers PH, Lessell S. 1985. Long-term follow-up after single toxic exposure to trichloroethylene. *Am J Ind Med* 8:119–26.

Goeptar AR, Commandeur JNM, van Ommen B, van Bladeren PJ, Vermeulen NP. 1995. Metabolism and kinetics of trichloroethylene in relation to toxicity and carcinogenicity. Relevance of the mercapturic acid pathway. *Chem Res Toxicol* 8:3–21.

International Agency for Research on Cancer. 1995. Dry cleaning, some chlorinated solvents, and other industrial chemicals. *IARC Monogr Eval Carcinog Risk Chem Hum* 63.

International Programme on Chemical Safety. 1985. Trichloroethylene. Geneva: World Health Organization. Environmental Health Criteria 50.

Kahn E, Letz G. 1989. Clinical ecology: environmental medicine or unsubstantiated theory? *Ann Intern Med* 2:104–6.

Kimbrough RD, Mitchell FL, Houk VN. 1985. Trichloroethylene: an update. *J Toxicol Environ Health* 15:369–83.

Shindell S, Ulrich S. 1985. A cohort study of employees of a manufacturing plant using trichloroethylene. *J Occup Med* 27:577–9.

Smith GF. 1966. Trichloroethylene: a review. *Br J Ind Med* 23:249–62.

Tola S, Vilhunen R, Jarvinen E, Korkala ML. 1980. A cohort study on workers exposed to trichloroethylene. *J Occup Med* 22:737–40.

## Specific Health Effects

Adams RM. 1976. Degreaser's flush. *West J Med* 125:487.

Forkert PG, Sylvestre PL, Poland JS. 1985. Lung injury induced by trichloroethylene. *Toxicology* 35:143–60.

King GS, Smialek JE, Troutman WG. 1985. Sudden death in adolescents resulting from the inhalation of typewriter correction fluid. *JAMA* 253:1604–6.

Lawrence WH, Partyka EK. 1981. Chronic dysphagia and trigeminal anesthesia after trichloroethylene exposure. *Ann Intern Med* 95:710.

## Related Government Publications

Agency for Toxic Substances and Disease Registry. 1995. Toxicological profile for trichloroethylene (updated, draft for public comment). Atlanta: US Department of Health and Human Services.

US Environmental Protection Agency. 1985. Health assessment document for trichloroethylene. Final report. Washington: US Environmental Protection Agency. NTIS Report No. PB/85-249696.

US Environmental Protection Agency. 1997. Addendum to the health assessment document for trichloroethylene: update carcinogenicity assessment for trichloroethylene. Review draft. Washington: US Environmental Protection Agency. Report No. EPA/600/8-82/006FA.

# Answers to Pretest and Challenge Questions

## Pretest

- (a) The mother's problem list includes pregnancy and anxiety; the child's list includes frequent otitis media (status postmyringotomy and tympanostomy tube placement) and frequent upper respiratory infections.
- (b) You will need information on TCE toxicity, including reproductive and developmental effects; information on TCE contamination of the family's drinking water, including duration and level of contamination; copies of information provided to the family by the municipal water company; and responses, if any, from local and state health agencies.
- (c) None of the symptoms described in the case indicate serious illness. However, you should reassure the family that you will perform complete physical examinations with appropriate testing at the next visit. In response to concern about the child's infections, you should indicate that you will collect information about possible TCE effects on the immune system. Explain to the parents that tests of immune function are often difficult to interpret and might not be appropriate. You might indicate that you will consult sources of information on TCE's effects on pregnancy. It is important to maintain a balance between reassurance that the unborn child is probably not affected by the water contamination and concern for the possible risk to the fetus. Reassurance should not, however, appear to trivialize the family's fears. It would also be appropriate to discuss that no evaluation, however thorough, can totally exclude the possibility that a person might develop an illness, including cancer.

## Challenge

- (1) From the information about the family thus far, none of the family members fit the profile of a person at increased risk from the effects of TCE exposure. That is, there is no indication that any family member has liver dysfunction or cardiac disorders, abuses TCE, or consumes large amounts of alcohol.
- (2) Possible sources of the family's TCE exposure include home drinking water (ingestion and dermal and inhalation exposure during bathing), the father's workplace (inhalation), and the daughter's day-care center (inhalation). Other sources might be the use of TCE-containing consumer products such as correction fluid, spot removers, and so forth (inhalation).
- (3) The most convenient biologic indicators of TCE exposure are the urinary metabolites, trichloroethanol and trichloroacetic acid. These metabolites are not specific to TCE, however, because they are also metabolites of tetrachloroethylene (perchloroethylene); 1,1,1-trichloroethane (methyl chloroform); and certain medications. TCE itself can be measured directly in blood or exhaled air, but because of the difficulty of obtaining samples, such measurements are not indicated here.
- (4) To properly interpret any of the tests mentioned in answer 3, knowledge of the time lapse between exposure and collection is necessary. To prevent contamination or sample loss (evaporation or adsorption), the proper collection, handling, storage, and transportation procedures must be followed. It is unlikely that any member of this family would have levels of TCE or its metabolites significantly above background levels. Furthermore, there are no appropriate reference values currently available for a health risk assessment.
- (5) No further studies are indicated for TCE exposure. A workup for fatigue can indicate additional tests.

(6) Based on limited evidence from animal studies, researchers believe that teratogenicity does not occur at environmental TCE levels. Invasive procedures are not justified on the basis of the drinking water contamination.

(7) No. A recent survey of infections in children under 3 years of age over a September-to-March period found an average of 2.5 total infections and more than one episode of otitis media per child (1.4 episodes per child for those in day care). More than 3% of the children in day care were hospitalized for tympanostomies. The child described in the case study appears to have an above-average rate of infections, but they are not frequent enough to suggest immunologic impairment.

(8) No. Immunocompetence tests are not appropriate because no evidence of immune function abnormalities has been found in similar situations. Nevertheless, physicians might be asked to explain further why they are not performing the tests on their patients. Two studies that may be of value are Kahn and Letz (1989) and American College of Physicians (1989).

If it had been indicated, laboratory evaluation of immunologic host-defense defects would consist of three phases. The preliminary screening is a complete blood count with differential smear and quantitative immunoglobulin levels. These tests, together with history and physical examination, will identify more than 95% of patients with primary immunodeficiencies. The second testing phase consists of readily available studies including B-cell function (such as antibodies and response to immunization), T-cell function (skin tests and contact sensitization), and complement levels. The first two phases combined will detect most immunodeficiencies amenable to conventional treatment with gamma globulin or plasma. The third phase (in-depth investigation) consists of testing induction of B-lymphocyte differentiation in vitro, stimulated by pokeweed mitogen and histologic and immunofluorescent examination of biopsy specimens; T-cell surface markers; assays of T-cell helper or killer cell functions; and functional assays using appropriate target cells. It is inappropriate to perform the latter tests on environmentally exposed patients except for epidemiologic research.

Primary immunodeficiency is suspected in an infant who has repeated upper respiratory tract or other infections. It is also suspected if repeated infection occurs in a child who has had little exposure to infectious agents, or any child with unusual infections, incomplete clearing of infections, growth failure, hepatosplenomegaly, or features associated with specific immunodeficiency disorders, such as ataxia or telangiectasia. The child described in the case study has none of these indications.

(9) EPA has not issued an emission standard for TCE. Assuming discussions with the owner or operator of the shop adjacent to the day-care center have not been effective in reducing the level of ambient TCE, the community's air pollution control center should be notified. States might allow this control under the jurisdiction of local air pollution control districts, county health departments, or other local agencies. The agency responsible for enforcement of air standards should be contacted to investigate possible release of TCE onto the day-care center property.

## Additional Sources of Information

In addition to other resources, ATSDR has created a National Exposure Registry for TCE. This registry is the first in a series of registries mandated by the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA). ATSDR, in cooperation with the states, establish and maintain national registries of (a) persons exposed to substances and (b) persons with serious illness or diseases possibly due to exposure. The registries collect information on the effects of low-level exposures of long duration (the exposures typically found in populations surrounding hazardous waste sites) and the health outcomes for populations receiving a one-time, high-level environmental exposure (such as those experienced at chemical spill sites). The registries will facilitate the identification and subsequent tracking of persons exposed to a defined substance at selected sites and will coordinate the clinical and research activities involving the registrants. For further information on the TCE registry, please contact ATSDR at 1-888-42-ATSDR (1-888-422-8737).

More information on the adverse effects of TCE and the treatment and management of persons exposed to TCE can be obtained from ATSDR, your state and local health departments, and university medical centers. *Case Studies in Environmental Medicine: Trichloroethylene Toxicity* is one in a series. For other publications in this series or for clinical inquiries, contact ATSDR, Division of Health Education and Promotion, Office of the Director, at 404-498-0101.

## Notes

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*Case Studies in Environmental Medicine:*

# Trichloroethylene (TCE) Toxicity

## Evaluation Questionnaire and Posttest, Course Number SS3056

**Course Goal:** To increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients.

### Objectives

- Discuss the major exposure routes for trichloroethylene.
- Describe two potential environmental and occupational sources of exposure to trichloroethylene.
- State two reasons why trichloroethylene is a health hazard.
- Describe factors contributing to trichloroethylene toxicity.
- Identify evaluation and treatment protocols for persons exposed to trichloroethylene.
- List two sources of information on trichloroethylene.

### Tell Us About Yourself

**Please carefully read the questions. Provide answers on the answer sheet (page 29). Your credit will be awarded based on the type of credit you select.**

**1. What type of continuing education credit do you wish to receive?**

**\*\*Nurses should request CNE, not CEU. See note on page 28.**

- A. CME (for physicians)
- B. CME (for non-attending)
- C. CNE (continuing nursing education)
- D. CEU (continuing education units)
- E. [Not used]
- F. [Not used]
- G. [Not used]
- H. None of the above

**2. Are you a...**

- A. Nurse
- B. Pharmacist
- C. Physician
- D. Veterinarian
- E. None of the above

**3. What is your highest level of education?**

- A. High school or equivalent
- B. Associate, 2-year degree
- C. Bachelor's degree
- D. Master's degree
- E. Doctorate
- F. Other

- 4. Each year, approximately how many patients with trichloroethylene toxicity do you see?**
- A. None
  - B. 1–5
  - C. 6–10
  - D. 11–15
  - E. More than 15
- 5. Which of the following best describes your current occupation?**
- A. Environmental Health Professional
  - B. Epidemiologist
  - C. Health Educator
  - D. Laboratorian
  - E. Physician Assistant
  - F. Industrial Hygienist
  - G. Sanitarian
  - H. Toxicologist
  - I. Other patient care provider
  - J. Student
  - K. None of the above
- 6. Which of the following best describes your current work setting?**
- A. Academic (public and private)
  - B. Private health care organization
  - C. Public health organization
  - D. Environmental health organization
  - E. Non-profit organization
  - F. Other work setting
- 7. Which of the following best describes the organization in which you work?**
- A. Federal government
  - B. State government
  - C. County government
  - D. Local government
  - E. Non-governmental agency
  - F. Other type of organization

## **Tell Us About the Course**

- 8. How did you obtain this course?**
- A. Downloaded or printed from Web site
  - B. Shared materials with colleague(s)
  - C. By mail from ATSDR
  - D. Not applicable

- 9. How did you first learn about this course?**
- A. State publication (or other state-sponsored communication)
  - B. *MMWR*
  - C. ATSDR Internet site or homepage
  - D. PHTN source (PHTN Web site, e-mail announcement)
  - E. Colleague
  - F. Other
- 10. What was the most important factor in your decision to obtain this course?**
- A. Content
  - B. Continuing education credit
  - C. Supervisor recommended
  - D. Previous participation in ATSDR training
  - E. Previous participation in CDC and PHTN training
  - F. Ability to take the course at my convenience
  - G. Other
- 11. How much time did you spend completing the course, evaluation, and posttest?**
- A. 1 to 1.5 hours
  - B. More than 1.5 hours but less than 2 hours
  - C. 2 to 2.5 hours
  - D. More than 2.5 hours but less than 3 hours
  - E. 3 hours or more
- 12. Please rate your level of knowledge before completing this course.**
- A. Great deal of knowledge about the content
  - B. Fair amount of knowledge about the content
  - C. Limited knowledge about the content
  - D. No prior knowledge about the content
  - E. No opinion
- 13. Please estimate your knowledge gain after completing this course.**
- A. Gained a great deal of knowledge about the content
  - B. Gained a fair amount of knowledge about the content
  - C. Gained a limited amount of knowledge about the content
  - D. Did not gain any knowledge about the content
  - E. No opinion

**Please use the scale below to rate your level of agreement with the following statements (questions 14–25) about this course.**

- A. Agree
- B. No opinion
- C. Disagree
- D. Not applicable

- 14. The objectives are relevant to the goal.**
- 15. The tables and figures are an effective learning resource.**
- 16. The content in this course was appropriate for my training needs.**
- 17. Participation in this course enhanced my professional effectiveness.**
- 18. I will recommend this course to my colleagues.**
- 19. Overall, this course enhanced my ability to understand the content.**
- 20. I am confident I can discuss the major exposure routes for trichloroethylene.**
- 21. I am confident I can describe two potential environmental and occupational sources of exposure to trichloroethylene.**
- 22. I am confident I can state two reasons why trichloroethylene is a health hazard.**
- 23. I am confident I can describe factors contributing to trichloroethylene toxicity.**
- 24. I am confident I can identify evaluation and treatment protocols for persons exposed to trichloroethylene.**
- 25. I am confident I can list two sources of information on trichloroethylene.**

## Posttest

If you wish to receive continuing education credit for this program, you must complete this posttest. Each question below contains five suggested answers, of which one or more is correct. **Circle all correct answers on the answer sheet.**

**26. Acute TCE exposure can adversely affect all of the following except**

- A. CNS
- B. skin
- C. pancreas
- D. mucous membranes
- E. joints.

**27. Chronic exposure to TCE might**

- A. cause headaches or drowsiness
- B. mildly alter liver function
- C. cause short-term memory deficits
- D. cause Alzheimer disease
- E. result in Gilbert syndrome.

**28. Laboratory tests to confirm TCE exposure include**

- A. blood analysis for trichloroethanol
- B. breath analysis for trichloroacetic acid
- C. urinary creatinine
- D. cardiac isoenzymes
- E. urinary trichloroacetic acid.

**29. Common clinical effects associated with acute exposure to pure TCE at concentrations >2,000 ppm include**

- A. trigeminal neuralgia
- B. peripheral neuropathy
- C. CNS depression
- D. nausea
- E. upper respiratory tract and eye irritation.

**30. Drinking water contaminated with low levels of TCE has been conclusively associated with**

- A. trigeminal neuralgia
- B. cardiac ventricular dysrhythmias
- C. hepatorenal syndrome
- D. no specific pathology
- E. cleft palate in children born to exposed pregnant women.

**31. Cardiac toxicity due to TCE exposure**

- A. is a frequent cause of death among dry-cleaning workers
- B. has not caused increased mortality among metal degreasers
- C. is caused by the metabolism of TCE to carbon monoxide
- D. might lead to dysrhythmias
- E. might be due to arteriosclerosis.

**32. Treatment for acute inhalation of TCE might include**

- A. symptomatic support
- B. inducing emesis
- C. hemodialysis
- D. oxygen
- E. milk of magnesia.

**33. The likelihood of TCE exposure exists for**

- A. accountants
- B. automobile parts manufacturers
- C. judges
- D. electronics workers
- E. race-car drivers.

## Note to Nurses

CDC is accredited by the American Nurses Credentialing Center's (ANCC) Commission on Accreditation. ANCC credit is accepted by most State Boards of Nursing.

California nurses should write in "ANCC - Self-Study" for this course when applying for relicensure. A provider number is **not** needed.

Iowa nurses must be granted special approval from the Iowa Board of Nursing. Call 515-281-4823 or e-mail [marmago@bon.state.ia.us](mailto:marmago@bon.state.ia.us) to obtain the necessary application.

*Case Studies in Environmental Medicine:*

# Trichloroethylene Toxicity

## Answer Sheet, Course Number SS3056

**Instructions for submitting hard-copy answer sheet:** Circle your answers. To receive your certificate, you must answer **all** questions. Mail or fax your completed answer sheet to

**Fax:** 404-498-0061, ATTN: Continuing Education Coordinator

**Mail:** Continuing Education Coordinator  
Agency for Toxic Substances and Disease Registry  
Division of Health Education and Promotion  
1600 Clifton Road, NE (MS E-33)  
Atlanta, GA 30333

**Remember, you can access the case studies online at [www.atsdr.cdc.gov/HEC/CSEM/](http://www.atsdr.cdc.gov/HEC/CSEM/) and complete the evaluation questionnaire and posttest online at [www2.cdc.gov/atsdrce/](http://www2.cdc.gov/atsdrce/).**

**Online access allows you to receive your certificate as soon as you complete the posttest.**

**Be sure to fill in your name and address on the back of this form.**

1. A B C D E F G H

2. A B C D E

3. A B C D E F

4. A B C D E

5. A B C D E F G H I J K

6. A B C D E F

7. A B C D E F

8. A B C D

9. A B C D E F

10. A B C D E F G

11. A B C D E

12. A B C D E

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22. A B C D

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24. A B C D

25. A B C D

26. A B C D E

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28. A B C D E

29. A B C D E

30. A B C D E

31. A B C D E

32. A B C D E

33. A B C D E



Name:

E-mail (not required):

Address:

Zip code:

- ☐ Check here to be placed on the list to pilot test new case studies

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**Atlanta, GA 30333**

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**Access the case studies online at [www.atsdr.cdc.gov/HEC/CSEM/](http://www.atsdr.cdc.gov/HEC/CSEM/) and complete the evaluation questionnaire and posttest online at [www2.cdc.gov/atsdrce/](http://www2.cdc.gov/atsdrce/).**

**Online access allows you to receive your certificate as soon as you complete the posttest.**



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