ATSDR

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Case Studies in Environmental Medicine

24

1,1,1-Trichloroethane

Environmental ALERT . . .

- ***Although 1,1,1-trichloroethane will be phased out of use by 1996, it will remain an environmental concern. Its wide use as a solvent in industry and for consumer products has resulted in large amounts being released to the environment. In addition, about 20% of the approximately 1200 hazardous waste sites on the National Priorities List contain this chemical.***
- ***For the general population, the most likely sources of exposure to 1,1,1-trichloroethane are home consumer products, building products, and contaminated food and water.***
- ***Inhalation abuse of 1,1,1-trichloroethane can result in "sudden sniffing death."***

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. See page 17 for more information about continuing medical education credits and continuing education units.

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U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

ATSDR

How to use this issue...

This issue begins with a composite case study that describes a realistic encounter with a patient. This description is followed by a pretest. The case study is further developed through Challenge questions at the end of each section. To fully benefit from this monograph, readers are urged to answer each question when it is presented. (Answers to the Pretest and Challenge questions are found on pages 15-16.) The monograph ends with a posttest, which can be submitted to the Agency for Toxic Substances and Disease Registry (ATSDR) for continuing medical education (CME) credit or continuing education units (CEU). See page 17 for further instructions on how to receive these credits.

The objectives of this monograph on 1,1,1-trichloroethane are to help you

- ❑ **Explain why 1,1,1-trichloroethane may be an acute and chronic health hazard**
- ❑ **Describe the factors that may contribute to 1,1,1-trichloroethane poisoning**
- ❑ **Identify potential environmental and occupational sources of exposure to 1,1,1-trichloroethane**
- ❑ **Identify evaluation and treatment protocols for persons exposed to 1,1,1-trichloroethane**
- ❑ **List sources of information on 1,1,1-trichloroethane**

Contents

This issue is prepared with the assistance of those who share a common concern for physician education, public health, and the environment, including the following organizations: American Academy of Clinical Toxicology (AACT), American Academy of Family Physicians (AAFP), American Academy of Pediatrics (AAP), American College of Emergency Physicians (ACEP), American College of Occupational and Environmental Medicine (ACOEM), American Medical Association (AMA), Association of State and Territorial Health Officials (ASTHO), and the Society of Teachers of Family Medicine (STFM). Final responsibility for the contents and views expressed in this monograph resides with ATSDR.

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Case Study

A mother who attributes her daughter's congenital heart defect to air pollution

Your practice is located in a valley with a number of computer-related, high-technology, "smokeless" industries. One of your patients, a young mother whose 8-month-old daughter was born with a congenital heart defect, comes to your office to discuss a recent newspaper article that suggests toxic chemicals may cause birth defects. After reading the article, she discovered that two of her neighbors had miscarriages shortly before her daughter's birth, two neighbors had recently given birth to babies who have defects, and one neighbor had a stillbirth. Many of the neighbors spoke of often smelling a sweet, solvent-like odor in the outdoor air.

Your patient is determined to confirm her suspicion that a chemical in the air from the local computer chip manufacturing plant is responsible for these events. She has called the city council and is not satisfied that she will get a prompt hearing. She has organized a small group of concerned citizens. She asks you to be a consultant to the group and provide medical information at the group's first meeting.

Your patient gives you a list of chemicals that she has determined are or have been used at the plant. The list includes arsine, phosphine, trichloroethylene, 1,1,1-trichloroethane, tetrachloroethylene, epoxy resins and curing agents, hydrofluoric acid, and gallium arsenide. You know that arsine, phosphine, and hydrofluoric acid are quite toxic and used in relatively small quantities in the electronics industry. Emissions are unlikely to escape the plant daily. In addition, the odors of these chemicals do not fit the patient's description. The epoxy resins and gallium arsenide are not volatile substances and are not likely to be detected outside the plant. You conclude that the chemicals most likely to be emitted in large quantities are the solvents.

You contact the regional office of the Environmental Protection Agency (EPA) for information on processes used by computer chip manufacturers and the quantities of solvents that might be involved. The environmental specialist searches the Toxic Chemical Release Inventory (TRI) database and informs you that the plant in question is a major emitter of 1,1,1-trichloroethane (TCA); last year, it emitted more than 100,000 pounds of the chemical. The specialist informs you that the plant does not emit significant quantities of the other chemicals on your list. You focus on the cluster of cases and the TCA exposure.

(a) What are the major health effects caused by 1,1,1-trichloroethane?

(b) Is 1,1,1-trichloroethane likely to be responsible for the congenital heart defect of your patient's daughter?

(c) What is the Toxic Chemical Release Inventory database that was used by the specialist?

(d) What sources will you use to prepare for the community group meeting? What will you advise?

Answers to the Pretest questions are on page 15.

Exposure Pathways

- **More than 700 million pounds of TCA are produced in the United States annually.**

1,1,1-Trichloroethane (Cl_3CCH_3) is not known to occur naturally. Originally produced as a safer replacement for carbon tetrachloride and later for trichloroethylene, it has become ubiquitous in the environment. 1,1,1-Trichloroethane is a nonflammable, colorless liquid that evaporates quickly at room temperature. It has a sweet, somewhat sharp odor that can be detected at about 100 parts per million (ppm). (This odor threshold is below the permissible exposure limit [PEL] for the workplace of 350 ppm.)

1,1,1-Trichloroethane is commonly referred to as TCA (used throughout this document). Synonyms for TCA include methyl chloroform, chloroethene, methyltrichloromethane, trichloromethylmethane, alpha-trichloroethane, and alpha-T. TCA should not be confused with TCE, which commonly refers to trichloroethylene ($Cl_2C=CHCl$).

The general population is exposed to TCA through ambient air, contaminated water, or by using household or office products containing the chemical. TCA is found in many building materials, including carpet glues and fabric finishers. The trend toward airtight, highly insulated houses has resulted in higher concentrations indoors than outdoors. In one study, average TCA air levels were about 4 ppb indoors and less than 1 part per billion (ppb) outdoors. Volatilization of TCA from contaminated water during showering, laundering, and other activities can add substantially to indoor exposure.

- **For the general population, exposure occurs most often through the many home products containing TCA.**

In a recent EPA study of household products, nearly 250 consumer items contained TCA. Some of these items include the following: fabric water repellants, spot removers, spray shoe polishes, spray paints, paint thinners and removers, pesticides, lubricants for auto door locks, tape and video recorder head cleaners, electric shaver cleaners, and typewriter correction fluids. Some correction fluids and thinners that have high concentrations of TCA are widely abused through inhalation for their narcotic effects. Deaths have resulted from this exposure route despite the mustard oil added to these products to discourage abuse, and some states have banned TCA use in correction fluids and thinners. TCA is also used as a propellant and solvent carrier in some aerosolized bronchodilators. In the past, TCA was used as an anesthetic. In 1990, 705 million pounds of TCA were produced in the United States.

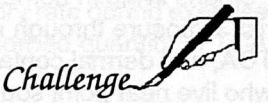
- **Airtight, highly insulated homes enhance accumulation of indoor TCA levels.**

TCA is released to the environment by stack and fugitive emissions and in wastewater from the numerous industries that produce or use this compound. It is used in more than 120 industrial classifications (including the defense and electronics industries), primarily as a solvent, vapor degreaser, cold metal cleaner, and propellant. Releases to surface water evaporate quickly, and spills on land dissipate readily through volatilization and leaching. The half-life of TCA in surface water ranges from hours to a few weeks, depending on wind and water turbulence. Bioaccumulation does not occur to a significant degree.

In soil, TCA undergoes slow degradation, and because it is not strongly adsorbed to sandy or clay soils, it can readily leach to underground aquifers. Amounts of TCA in groundwater vary widely by location. In one study of groundwater in 13 U.S. cities, EPA found 23% of the sources contained TCA (maximum concentration 13 micrograms per liter [µg/L] [13 ppb]). Groundwater samples taken near sources of release have been as high as 11,000 ppb.

TCA released to air is transported long distances and will partially return to earth in rain. In the troposphere, TCA degrades very slowly by photooxidation; the half-life is 6 months to 25 years, depending on the intensity of sunlight. Due to the large input of TCA into the atmosphere and its slow degradation, the amount of TCA in ambient air is increasing by 12% to 17% annually. TCA slowly diffuses to the stratosphere where photodegradation is rapid, and the chlorine free radicals that are generated contribute to the destruction of the ozone layer. In 1992, the 93 signatories to the Montreal Protocol, an international agreement to control ozone-depleting substances, agreed to ban TCA and certain other chlorocarbons by 1996, 4 years earlier than previously agreed.

* Use of trade names is for identification only and does not imply endorsement by the Public Health Service or the U.S. Department of Health and Human Services.



Additional information for the case study: In a discussion with the environmental engineer at the plant, you learn that within the last 6 months the plant has installed control measures to reduce emissions and is switching to aqueous-based degreasing agents and ultrasonic technology in their cleaning and degreasing processes. He predicts very little will be emitted from the plant in the near future.

(1) What are other possible sources of exposure to TCA for your patient and her neighbors?

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Who's at Risk

- **Workers involved in operations using TCA are at greatest risk of exposure.**

According to a study conducted by the National Institute for Occupational Safety and Health (NIOSH), more than 1.5 million workers are potentially exposed to TCA. Workers with the greatest potential for exposure to TCA are in the following industries or operations:

auto repair
computer chip manufacture
degreasing and cleaning of metal parts
dry cleaning
electrical parts manufacture
heating and cooling parts during manufacture
mixing and applying commercial resins
photography
solvent recovery
spray painting and gluing
typesetting
waste disposal

- **Persons who inhale TCA intentionally for its narcotic properties risk "sudden sniffing death."**

The general population is at risk of exposure through the use of home products that contain TCA, but the levels of exposure associated with normal use have not been shown to cause adverse health effects. Intentional inhalation of TCA for its narcotic effects is a problem, however, and is most common among adolescents. Persons who deliberately sniff glue or solvents to achieve euphoria risk adverse health effects, such as "sudden sniffing death" from cardiac dysrhythmias.

Persons living in homes that use a TCA-contaminated water source risk exposure through ingestion of tapwater, inhalation of volatilized TCA, and dermal contact during showering and laundering. Persons who live near point sources of TCA or near waste disposal sites also risk exposure to above-background levels.

Persons who have histories of alcoholism or severe underlying liver disease may be at increased risk of TCA's adverse effects. Alcohol competitively inhibits the metabolism of TCA. Alcohol and severe liver disease could prolong TCA metabolism and its effects on the central nervous system (CNS).

Challenge

Additional information for the case study: As a result of your work with the community group, you receive a call from a reporter. A wire story indicates that a consumer group had hired a laboratory to test disposable baby diapers and found TCA at 20 ppb in one brand of diapers. The reporter asks whether he should run the story, warning parents that these diapers might have adverse health effects on their babies and challenging grocers to pull the brand from their shelves.

(2) What is your response to the reporter's question?

Biologic Fate

TCA is absorbed efficiently and rapidly through the lungs and gastrointestinal tract and less rapidly through the skin. Steady-state lung retention of 25% to 30% of inhaled TCA in humans is reached within 1 to 3 hours of continuous exposure. Continuous exposure also results in a gradual rise and plateau of arterial and venous blood levels. Increases in pulmonary ventilation or respiratory rate during exercise are not believed to affect changes in the absorbed quantity of TCA after steady-state levels are reached.

- **TCA is absorbed efficiently by all routes of exposure.**

TCA is lipophilic and accumulates to a small extent in the body fat. The largest amount of TCA is found in brain tissue, although tissue concentrations throughout the body vary widely. Blood levels correlate highly with levels found in alveolar air. A much greater amount of TCA is absorbed during inhalation exposure than during skin contact.

- **Regardless of its route of absorption, most TCA is excreted unchanged through the lungs.**

Regardless of the route of absorption, most TCA (e.g., about 90% of an inhaled dose) is quickly excreted unchanged by the lungs. It has a half-life for elimination from the blood of approximately 53 hours; small amounts of TCA have been detected in the breath several days after exposure has ceased. The remaining 10% of an inhaled dose is excreted unchanged in the urine or metabolized by hepatic cytochrome P-450 mixed-function oxidase enzymes to trichloroethanol and then to trichloroacetic acid. These metabolites can be detected in blood and urine. The half-life of trichloroethanol, which is excreted in urine, is about 9 hours. Because trichloroacetic acid binds to serum albumin, this metabolite persists in the bloodstream with a half-life of about 3 days.

- **A small amount of TCA is metabolized in the liver.**

Challenge

Additional information for the case study: Community concern about TCA prompts another patient to call you. He and his wife think their adolescent son is abusing TCA and want you to run some tests to confirm this.

(3) What is your response?

Physiologic Effects

- **Primarily the central nervous, cardiovascular, and respiratory systems are affected by acute TCA exposure.**

Fatalities following exposure to anesthetic concentrations of TCA have usually been due to CNS depression, resulting in respiratory arrest. Cardiac dysrhythmias may also occur after exposure to high levels, especially in solvent abusers. Ingestion of TCA may cause immediate burning of the mouth, throat, and esophagus; nausea, vomiting, and diarrhea occur after large ingestions. Because of TCA's defatting action, local skin irritation, erythema, vesiculations, and dermatitis can occur after repeated contact. Stabilizing agents added to TCA, such as epichlorohydrin and dioxane, are sensitizers and may be responsible for some of TCA's dermatologic effects. TCA contact with unprotected eyes can produce transient and superficial tissue effects, including chemosis, hyperemia, and conjunctivitis.

Neurologic Effects

- **The principal effect of TCA exposure is CNS depression.**

With increasing concentration of TCA, CNS depression and euphoric narcosis occur in proportion to the amount of TCA absorbed. Mild motor impairment is followed by stupor, coma, and seizures. In acute, high-level exposures, the anesthetic effect occurs rapidly. With chronic exposure, a consistent finding is agitation or lethargy during sedentary periods. Decreased memory and sleep disturbances have also been reported from chronic, low-level exposure.

TCA is lipophilic and accumulates in the central nervous system, resulting in respiratory depression. This depression is mediated, in part, by effects in the brain stem, as well as by generalized CNS narcosis.

Cardiovascular Effects

Acute or chronic exposures to high concentrations of TCA may cause reduced blood pressure and dysrythmias. Sudden death can occur in chronic abusers who inhale TCA. One study suggests that sudden sniffing death from TCA may result from light-plane anesthesia in combination with acute respiratory depression from a medullary chemical lesion. Others suggest that death may be due to a fatal dysrhythmia caused by lowering the myocardial threshold to the effects of epinephrine or other catecholamines.

Tachycardia noted in exposures to low doses and bradycardia observed in exposures to high doses may be controlled by the sympathetic nervous system. If dysrhythmias resolve spontaneously or by treatment, usually no permanent or recurrent problem occurs; however, bouts of premature ventricular contractions, including bigeminy, have been reported to reoccur within 1 to 2 weeks.

- **Cardiac dysrhythmias may result from lowering of the myocardial threshold to the dysrhythmogenic effects of epinephrine.**

Respiratory Effects

TCA has a direct irritating effect on the nasal and respiratory mucous membranes. Although this irritant effect is moderate with pure TCA, it can be more severe with technical grade TCA that is stabilized with dioxane or epichlorohydrin. Shortness of breath and chest pain can occur. Acute, high-level exposures to TCA may result in CNS-mediated respiratory depression.

- **Respiratory depression associated with TCA exposure is secondary to CNS depression.**

Hepatic and Renal Effects

Hepatic and renal dysfunction can occur 1 to 2 days after a single acute exposure to TCA, as evidenced by abnormal liver function and hepatic lesions. In one study, mild and transient elevation of liver function tests occurred in 33% of workers acutely exposed to high concentrations of TCA. Liver enzymes are often elevated on the first day after exposure, with bilirubin typically rising on the second day. Transient renal damage may also occur as initially evidenced by hematuria and proteinuria. These abnormal test results usually subside within a few days to 1 week, except in persons who have chronic exposures.

- **Hepatic and renal dysfunction may occur within 1 to 2 days after TCA exposure.**

Developmental and Reproductive Effects

No data are available on the reproductive or developmental effects of TCA in humans. A multigeneration reproductive study of rats exposed to TCA in drinking water found no reproductive effects. Only one inhalation study found evidence of minor embryotoxicity in experimental animals. No human studies have been reported.

- **No data exist to assess the reproductive and developmental effects of TCA in humans.**

Carcinogenic Effects

❑ **No evidence has been reported that suggests TCA is carcinogenic in humans or experimental animals.**

TCA does not appear to be carcinogenic in humans or experimental animals, although the data in humans are limited.

Additional information for the case study: The story of your patient and her activities receives national attention. You receive a call from a young woman in another state who has been diagnosed with multiple sclerosis. She had been a secretary for 5 years and used correctional fluids and thinners extensively. She read in the newspaper that these materials contain TCA, as do many household products, and that TCA can cause neurologic effects. She asks you whether TCA could be the cause of her condition.

(4) How will you respond to this question?

Clinical Evaluation

History and Physical Examination

❑ **A thorough work and exposure history is essential to correctly diagnosing TCA toxicity.**

A careful history should first be obtained from the person with suspected exposure, unless life-threatening circumstances exist. Even if serious conditions require immediate treatment, the patient should be questioned while medical care is being given. Family members or friends, as well as emergency medical response personnel, may also be able to provide valuable information regarding exposure.

If the exposure occurred in the workplace, identification of the substance to which the patient was exposed should be verified, if possible, from the container label or a Material Safety Data Sheet (MSDS) or by calling the workplace supervisor. A regional poison control center can help determine ingredients of brand name products and treatment for patients who have been exposed.

Especially in cases of chronic exposure, TCA exposure from all sources must be assessed. These sources include the following: use of TCA-containing household products, nearby industries, a recently remodeled or constructed home, diet, water supply, workplace, hobbies such as furniture refinishing, and recreational activities involving machinery or vehicles. If an airborne exposure is involved, the time the odors were detected and the time of onset of symptoms and their sequence are important. However, the presence of an odor does not necessarily mean that toxic effects will occur because the TCA air level that causes adverse health effects is substantially above the odor threshold. The relative ventilation and amount of TCA vaporized compared to the air volume of the room are important.

After addressing the patient's initial complaints, the physical examination should emphasize the organ systems most likely to be affected by TCA—cardiovascular, pulmonary, central nervous, and gastrointestinal (if ingestion of TCA occurred). Evaluation of the renal and hepatic systems should be assessed through laboratory testing. The patient should be questioned about intercurrent illness and medications, which may compromise the patient's response to toxic exposure.

Signs and Symptoms

Initial symptoms of TCA toxicity include headache, dizziness, fatigue, sleepiness, and nausea. Ataxia, confusion, and stupor can occur with acute exposure. Underlying disorders of the heart and lungs may be exacerbated by TCA exposure. Gastrointestinal signs usually do not proceed beyond nausea in cases of inhalation exposure. Ingestion of TCA may cause vomiting.

- **Initial symptoms of overexposure to TCA are neurologic.**

Chronic exposure to levels greater than 350 ppm may result in mild headaches, short-term memory loss, sleep disturbances, and ataxia. The patient should be questioned about other exposures that could be contributory including alcohol and drug abuse, prescription drugs, and psychiatric disorders, as well as exposure to other chemical neurotoxins.

Laboratory Tests

Direct Biologic Indicators

Most of the TCA absorbed by the body is excreted unchanged by the lungs. About 60% to 80% of an absorbed dose is excreted in 1 week, but TCA can be detected in breath weeks after exposure. TCA has an elimination half-life in blood of about 53 hours. TCA blood and breath tests are complex, however, and usually are not available on an emergency basis.

- **Generally, only reference laboratories perform tests for TCA blood and breath levels.**

Indirect Biologic Indicators

- **TCA metabolites can be measured in blood and urine.**

The metabolites of TCA, trichloroethanol and trichloroacetic acid, can be detected in blood and urine. Trichloroacetic acid binds to serum albumin and will persist in the blood with a half-life of about 3 days. These metabolites can be detected in the urine for a few days after mild exposures and a few weeks after heavy exposures. TCA metabolite measurements are useful if they are markedly elevated compared with normal background levels and if exposure to trichloroethylene, chloroform, and chloral hydrate (which produce the same metabolites) can be excluded.

Mild elevations of liver function tests, such as hepatic transaminases (SGOT or AST, SGPT or ALT), can occur after acute exposures but are usually mild; they most often return to normal after several days. Total bilirubin may also increase. Transient proteinuria and microscopic hematuria are sometimes accompanied by an increase in BUN or serum creatinine.

Challenge

Additional information for the case study: A neighbor of the patient in the case study is an employee at an airline maintenance depot. He has been taking TCA home from work to use as a cleaner for many household chores. His wife, who used it for carpet cleaning, broke out with hives. The condition cleared when her physician prescribed prednisone in tapered doses. The next time she used the TCA, however, the hives returned and became much worse, developing into large confluent splotches. She had had hives several years before when working in a photo lab.

(5) Is TCA the cause of this skin condition? Explain.

Treatment and Management

There are no antidotes for TCA toxicity. Patients should be removed from the contaminated environment as soon as possible, with priority given to ventilatory support, if needed. Supplemental oxygen should be initiated if respiration, pulse, blood pressure, or mental status is compromised. Continuous cardiac monitoring should be maintained until stability is established. Hemodialysis and hemoperfusion have not been proven efficacious.

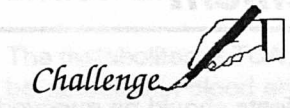
Contaminated clothing and personal items should be removed and double-bagged. Skin should be washed with mild soap or detergent and copious water. Care should be taken to avoid secondary contamination of health care personnel from off-gassing vapors or vomitus.

If TCA is splashed in the eye, the eye should be irrigated with water for 15 minutes. If the splash was more than transient, the corneal surface should be stained with fluorescein and examined using a slit lamp. Applying a protective lubricating antibiotic ointment and eye patch usually will enable the cornea to re-form epithelium within 24 to 48 hours.

If TCA is ingested, emesis is not advised because of possible pulmonary aspiration. Gut decontamination with lavage and activated charcoal is recommended if the procedure can be initiated within 2 to 3 hours after ingestion of more than a single swallow of TCA. Protect the airway to prevent pulmonary aspiration. Electrolyte balance should be maintained, and hepatic and renal dysfunction should be monitored until normal.

Chronic exposure to TCA is treated symptomatically. Hepatic and renal function should be assessed. If the exposure was occupational, periodic workplace air monitoring should be carried out to ensure compliance with regulations of the Occupational Safety and Health Administration (OSHA).

- **Supportive care and removal from the source of exposure are recommended treatment for TCA toxicity.**



Additional information for the case study: Testing of private wells in the vicinity of the plant reveals TCA contamination ranging from 30 to 232 µg/L (ppb). (EPA has a standard of 200 ppb in public drinking water supplies.)

(6) Do you expect these residents to manifest symptoms of exposure? What management and treatment would you recommend for the exposed residents?

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Standards and Regulations

Workplace

Air

OSHA regulations are based on protecting healthy workers from significant exposure to chemical hazards over a 40-hour workweek. The 1989 permissible exposure limit (PEL) for TCA as an 8-hour time-weighted average (TWA) was set at 350 ppm. The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a threshold limit value (TLV) of 350 ppm. ACGIH also recommends a short-term exposure limit (STEL) of 450 ppm. NIOSH recommends that occupational exposures to TCA be controlled so that workers are not exposed to levels greater than 350 ppm (ceiling concentration) as determined by a 15-minute sample (Table 1).

ACGIH has also set guidelines (Biological Exposure Indices [BEI]) for the amount of TCA metabolites in urine of exposed workers. For trichloroethanol, the BEI is 30 milligrams/liter (mg/L) in urine samples obtained at the end of the workweek, and for trichloroacetic acid, the BEI is 10 mg/L.

Table 1. Standards and regulations for 1,1,1-trichloroethane

Agency*	Focus	Level	Comments
ACGIH	**Air-workplace**	350 ppm	Advisory; TLV-TWA†
NIOSH	**Air-workplace**		Advisory; TWA
	Ceiling	350 ppm	
	IDLH §	1000 ppm	
OSHA	**Air-workplace**	350 ppm	Regulation; PEL¶ as TWA
EPA	**Air-environment**	NA**	Under review
	Drinking water	200 ppb	Regulation
	Ambient water	18 ppm	Regulation

*ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration

†TLV-TWA (threshold limit value–time-weighted average) = time-weighted average concentration for a normal workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed without adverse effects.

§IDLH (immediately dangerous to life and health) = represents the maximum concentration from which, in the event of respirator failure, one could escape within 30 minutes without a respirator and without experiencing any escape-impairing (e.g., severe eye irritation) or irreversible health effects.

¶PEL (permissible exposure limit) = level in air to which a worker may be exposed, averaged over an 8-hour workday.

**NA = not available

Environment

Water

EPA has set a standard of 200 µg/L (200 ppb) of TCA for drinking water. To protect fish and wildlife, EPA has set the ambient water level at 18 mg/L (18 ppm).

Air

EPA currently has an ambient air standard under review.

Suggested Reading List

General

Astrand I. Uptake of solvents in the blood and tissues of man: a review. Scand J Work Environ Health 1975;1:199-218.

Baselt RC, Cravey RH. Trichloroethane. In: Disposition of Toxic Drugs and Chemicals in Man. Chicago: Year Book Medical Publishers, 1989:824-7.

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Wallace L, Pellizzari E, Hartwell T. Concentrations of 20 volatile organic compounds in the air and drinking water of 350 residents of New Jersey compared with concentrations in their exhaled breath. J Occup Med 1986;28:603-8.

Specific Health Effects

deNevers N. A fatal fire with "nonflammable" methyl chloroform. Arch Environ Health 1986;41:279-81.

Halevy J, Pitlik S, Rosenfeld J. 1,1,1-Trichloroethane intoxication: a case report with transient liver and renal damage, review of the literature. Clin Toxicol 1980;16:467-72.

Kurt TL, Gallagher JS. Neonatal exposure to methyl chloroform in tape remover. Vet Human Toxicol 1990;32:43-5.

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

McLeod AA, Margot R, Monaghan MJ. Chronic cardiac toxicity after inhalation of 1,1,1-trichloroethane. Brit Med J 1987;294:727-9.

Perry GF, Jr. Trichloroethane and connective tissue disorders. J Occup Med 1992;35:5-6.

Related Government Publications

Agency for Toxic Substances and Disease Registry. Toxicological Profile for 1,1,1-Trichloroethane (Draft), Atlanta: US Public Health Service, Agency for Toxic Substances and Disease Registry, 1990. Draft report TP-90-27.

Environmental Protection Agency. Household solvent products: a national usage survey. Washington, DC: US Environmental Protection Agency, Office of Toxic Substances,1987. Report No. EPA-OTS 560/5-87-005.

Environmental Protection Agency. Household solvent products: a "shelf" survey with laboratory analysis. Washington, DC: US Environmental Protection Agency, Office of Toxic Substances,1987. Report No 560/5-87-006.

Sources of Information

More information on the adverse effects of 1,1,1-trichloroethane and treating cases of exposure to TCA can be obtained from ATSDR, your state and local health departments, and university medical centers. *Case Studies in Environmental Medicine: 1,1,1-Trichloroethane Toxicity* is one of a series. To obtain other publications in this series, please use the order form on the inside back cover. For clinical inquiries, contact ATSDR, Division of Health Education, Office of the Director, at (404) 639-6204.

Answers to Pretest and Challenge Questions

Pretest questions are on page 1. Challenge questions begin on page 3.

Pretest

(a) Chronic, low-level TCA exposure produces primarily neurologic effects including agitation, lethargy, decreased memory, and sleep disturbances. Chronic abuse of TCA may cause cardiac dysrhythmias and central nervous system depression leading to respiratory arrest.

(b) There is no evidence in humans or animals that TCA causes congenital heart defects at any air level.

(c) The Toxic Chemical Release Inventory (TRI) is mandated by the federal government as part of community right-to-know legislation. Industries must report annually the chemicals they discharge to the environment and an estimate of the amounts. This information is incorporated into a database, which is organized by geographic location (state and county) and by chemical. Citizens may access TRI to determine the major sources of environmental release in their vicinity and the chemicals causing that pollution.

(d) Sources that will assist you in preparing for the group meeting are standard toxicology books, on-line databases, and journal articles. In addition, you could talk to persons at ATSDR, the state health department, EPA, the regional poison control center, the trade association for the electronics industry, and health and safety personnel at the plant.

At the meeting, you could discuss your findings, then tell the audience about the effects of TCA, pointing out areas where health data are incomplete or unavailable. You could explain that different levels of exposure to TCA can occur and include deliberate inhalation abuse (air concentrations in thousands of ppm), legal workplace levels (air concentrations to 350 ppm), and environmental levels (typically lower than workplace levels). Because some of the people could smell the solvent outdoors, one may assume the concentration was above 100 ppm, the odor threshold for TCA. However, EPA currently has no ambient air standard for TCA, so it is difficult to assess the risk. You could also explain to the audience that events such as birth defects or cancer could occur in random clusters.

There is no evidence that TCA causes heart defects. However, exposures to any source of TCA should be minimized, and you might discuss how exposures could occur. Your advice might be that the group express their concerns to officials at the plant and that they contact the local or state health department to request investigation of the problem through air and water measurements, air and water modeling, and further epidemiologic investigations.

Challenge

Challenge questions begin on page 3.

(1) TCA is in many household products as a degreaser, solvent, or propellant. Commonly used products are fabric water repellants, spot removers, spray shoe polishes, spray paints, and paint thinners and removers. Household and office exposures can also occur from off-gassing of carpet and fabric drapes if the building is newly built or recently remodeled. TCA is in many aerosolized medications, such as bronchodilators. It is important to have adequate ventilation indoors, especially if any of these products are used in significant amounts or are spilled.

It would not be unusual for the computer chip manufacturing plant to store TCA in underground storage tanks. Leaking storage tanks at electronics plants have been reported to contaminate groundwater in several areas. Testing the community's water supply could help to determine whether leaking underground tanks are contributing to TCA exposure.

(2) You tell the reporter that he should not run the story and falsely alarm parents. You then attempt to put the information in perspective for the media representative. Under EPA rules, drinking water may contain up to 200 µg/L (200 ppb); bathing water may contain up to 18,000 ppb. You are not aware of a TCA standard for disposable diapers. While it is true that children are more susceptible to many toxicants and often exhibit signs of poisoning at lower levels than adults (e.g., lead or organophosphate pesticide exposures), the TCA level of 20 ppb is not cause for alarm. You further explain that TCA is one of the less hazardous solvents in terms of health effects. The level of TCA found is also not likely to cause diaper rashes, which occur in more than 20% of infants in the first 6 months of life.

(3) The best test for confirmation of exposure is the measurement of TCA in the blood. If TCA is detected in the blood, the adolescent most likely inhaled it within the last 5 to 7 days. If TCA metabolites (trichloroethanol or trichloroacetic acid) are also detected in the same blood sample, he may be a chronic abuser. TCA is quickly exhaled from the lungs; therefore, TCA metabolites are not likely to be present after a one-time use.

An approach that requires biologic testing is a bit adversarial, however. Rather, both parents should educate their son about the hazards of inhalant abuse. They should get involved in closely monitoring their child's activities. If school performance is deteriorating or they have other reasons to suspect their son's activities, enrolling him in a community substance abuse program and enlisting the help of a mental health professional to explore why their son is taking such risks may be appropriate. Inhalation abuse of solvents is most common in adolescents, and many states have mandated restrictions on selling spray paints and solvents to juveniles.

(4) TCA exposure has not been associated with multiple sclerosis (MS). MS is the most common chronic disease of the central nervous system in young adults in North America. It is characterized by the occurrence of discrete areas of demyelination or plaques in the brain or spinal cord with symptoms including some degree of paralysis, nystagmus, and disturbances of speech, depending on the site of the lesions.

(5) Although degreasing or defatting skin of endogenous oils is the most common skin problem with TCA, contact dermatitis, hives, and urticaria have been reported, usually associated with a history of these conditions. These allergic conditions are associated with use of TCA in a stabilized form, which contains dioxane or epichlorohydrin. Both of these stabilizers are not only stronger irritants than TCA, but also skin sensitizers. (See *Case Studies in Environmental Medicine: Skin Lesions and Environmental Exposures.*)

(6) The appearance of symptoms will depend upon dose, duration, and route of exposure. The routes of exposure include ingestion of the contaminated water and inhalation and dermal exposure during showering, laundering, or cleaning. The highest level found is not significantly above the EPA regulation. The mean daily intake of TCA from all sources (air, food, and water) has been determined to be between 50 and 1000 µg/day; therefore, one would not expect symptoms in most people. Because children and elderly persons are generally more susceptible to toxic agents, households with these occupants could consider changing their source of water.

Posttest and Credits

Continuing education credit is available to health professionals who use this monograph and complete the posttest. The criterion for awarding continuing medical education (CME) credits and continuing education units (CEU) is a posttest score of 70% or better.

The Centers for Disease Control and Prevention (CDC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians, and by the International Association for Continuing Education and Training (IACET) to sponsor continuing education units for other health professionals.

The Agency for Toxic Substances and Disease Registry, in joint sponsorship with CDC, is offering 1 hour of CME credit in Category 1 of the Physician's Recognition Award of the American Medical Association and 0.1 hour of CEU for other health professionals upon completion of this monograph.

In addition, the series *Case Studies in Environmental Medicine* has been reviewed and is acceptable for credit by the following organizations:

The **American Academy of Family Physicians (AAFP)**. This program has been reviewed and is acceptable for 1 prescribed hour by the American Academy of Family Physicians. (Term of Approval: beginning January 1992.) For specific information, please consult the AAFP Office of Continuing Medical Education.

The **American College of Emergency Physicians (ACEP)**. Approved by the American College of Emergency Physicians for one hour per issue of ACEP Category I credit.

The **American Osteopathic Association (AOA)**. AOA has approved this issue for 1 credit hour of Category 2-B credit.

The **American Association of Occupational Health Nurses (AAOHN)**. AAOHN has approved this program for 1.0 contact hours. Applicant will receive the assigned code number in the award letter.

The **American Board of Industrial Hygiene (ABIH)**. ABIH has approved this program for 0.5 certification maintenance (CM) point per three Case Studies. The CM approval number is 2817.

To receive continuing education credit (CME or CEU), complete the Posttest on page 18 in the manner shown in the sample question below. **Circle all correct answers.**

Which of the following is known to precipitate migraine headaches?

(a) fatigue
(b) alcohol
c. grapefruit
(d) sunlight
e. sleep

After you have finished the Posttest, please transfer your answers to the answer sheet on page 19 and complete the evaluation on the lower half of that page. Fold, staple, and mail the back cover to Continuing Education Coordinator, Agency for Toxic Substances and Disease Registry, Division of Health Education, E33, 1600 Clifton Road, Atlanta, GA 30333. Your confidential test score will be returned with an indication of where the correct answers can be found in the text. Validation of earned CME credit and CEU will also be forwarded to participants, and their names, if requested, will be placed on the mailing list to receive other issues in the *Case Studies in Environmental Medicine* series.

POSTTEST: 1,1,1 - TRICHLOROETHANE

Circle **all** correct answers. Record your answers on page 19.

1. Acute TCA exposure can adversely affect all of the following except
   a. CNS
   b. skin
   c. pancreas
   d. kidneys
   e. liver

2. Chronic exposure to TCA may
   a. cause headaches
   b. mildly alter liver function
   c. cause short-term memory deficits
   d. cause Alzheimer's disease
   e. cause early onset of symptoms from human immunodeficiency virus (HIV)

3. Laboratory tests to confirm TCA exposure might include
   a. blood analysis for trichloroethanol
   b. breath analysis for trichloracetic acid
   c. urinary creatinine
   d. cardiac isoenzymes
   e. blood analysis for TCA

4. Clinical effects associated with acute exposure to pure TCA at concentrations greater than 1000 ppm include
   a. unconsciousness or coma
   b. cardiac arrhythmias
   c. hematuria or proteinuria
   d. mild liver dysfunction
   e. stroke

5. Drinking water contaminated with low levels of TCA has been associated with
   a. oral mucous membrane irritation
   b. hepatorenal syndrome
   c. children with cleft palates born to exposed women
   d. no specific pathology
   e. cancer

6. Cardiac toxicity due to TCA exposure
   a. may lead to dysrhythmias
   b. is caused by the metabolism of TCA to carbon dioxide
   c. causes increased mortality among metal degreasers
   d. is associated with inhalation abuse of TCA
   e. is a frequent cause of death among dry-cleaning workers

7. Treatment for acute inhalation of TCA may include
   a. symptomatic support
   b. lavage and activated charcoal
   c. hemodialysis
   d. exchange transfusion
   e. ethanol drip

8. The likelihood of TCA exposure exists for
   a. dry cleaners
   b. auto mechanics
   c. silicon chip manufacturers
   d. bus drivers
   e. judges

CASE STUDIES IN ENVIRONMENTAL MEDICINE: 1,1,1 - TRICHLOROETHANE TOXICITY

If you wish CME credits or CEU, please indicate your answers to the Posttest questions on page 18 by circling the letters below for the correct answers. Complete the evaluation questionnaire and fill in the information requested on the reverse side. Tear off this last page, fold, staple, and mail to Continuing Education Coordinator, Agency for Toxic Substances and Disease Registry, Division of Health Education, E33, 1600 Clifton Road, Atlanta, GA 30333.

1. a b c d e
2. a b c d e
3. a b c d e
4. a b c d e
5. a b c d e
6. a b c d e
7. a b c d e
8. a b c d e

Evaluation Questionnaire

Please complete the following evaluation by putting a check in the appropriate box.

	Yes	No	Undecided
1. As a result of completing this monograph, I will be able to:			
Explain why 1,1,1-trichloroethane may be an acute and chronic health hazard.	☐	☐	☐
Describe the factors contributing to 1,1,1-trichloroethane toxicity.	☐	☐	☐
Identify potential environmental and occupational sources of exposure to 1,1,1-trichloroethane.	☐	☐	☐
Identify evaluation and treatment protocols for persons exposed to 1,1,1-trichloroethane.	☐	☐	☐
List sources of information on 1,1,1-trichloroethane.	☐	☐	☐
2. I am more likely to ask patients questions regarding possible environmental exposures as a result of reading this issue.	☐	☐	☐
3. I would recommend this issue to my colleagues.	☐	☐	☐
4. I will keep this issue as a reference.	☐	☐	☐

Comments:_____

To obtain credit, please provide the requested information below.

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☐ CEU ☐ Contact Hours - AAOHN ☐ CM - ABIH

Specialty _____

To be placed on mailing list, check here. ☐

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1600 Clifton Road, NE
Atlanta, GA 30333

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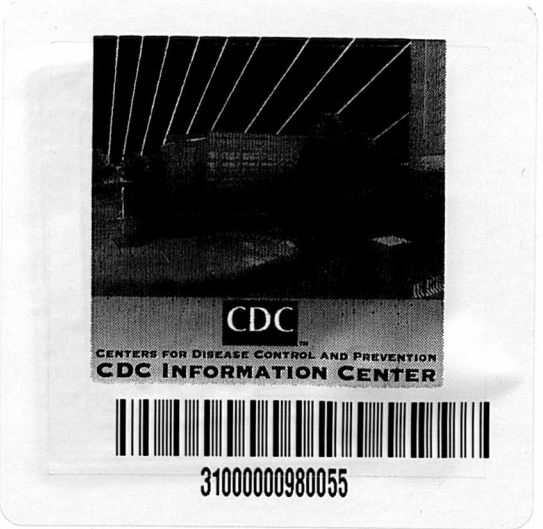
Please send me the following *Case Studies in Environmental Medicine.*

- ☐ Arsenic
- ☐ Asbestos
- ☐ Benzene
- ☐ Beryllium
- ☐ Cadmium
- ☐ Carbon Tetrachloride
- ☐ Chlordane
- ☐ Cholinesterase Inhibitors
- ☐ Chromium
- ☐ Cyanide
- ☐ Dioxins
- ☐ Ethylene/Propylene Glycols
- ☐ Exposure History
- ☐ Gasoline
- ☐ Jet Fuel
- ☐ Lead
- ☐ Mercury
- ☐ Methanol
- ☐ Methylene Chloride
- ☐ Nitrates/Nitrites
- ☐ Pentachlorophenol
- ☐ Polyaromatic Hydrocarbons (PAHs)
- ☐ Polychlorinated Biphenyls (PCBs)
- ☐ Radiation
- ☐ Radon
- ☐ Risk Communication
- ☐ Reproductive and Developmental Hazards
- ☐ Skin Lesions
- ☐ Stoddard Solvent
- ☐ Tetrachloroethylene
- ☐ 1,1,1-Trichloroethane
- ☐ Trichloroethylene
- ☐ Toluene
- ☐ Vinyl Chloride

ML0993

staple or tape



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, the Agency for Toxic Substances and Disease Registry (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 to this substance. This monograph is intended as an additional resource for physicians and other health professionals 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 and must be interpreted in light of specific information regarding the patient available to such a professional and in conjunction with other sources of authority.

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