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Common-Source Community and Industrial Exposure to Trichloroethylene

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ABSTRACT: In July 1979, 1,900 gallons of trichloroethylene (TCE) were released into ground and surface water from a pipe manufacturing plant in Montgomery County, Pennsylvania. To evaluate community and occupational exposure to TCE, we conducted environmental and medical surveys. In well water samples obtained in August 1979 within 1 km of the factory, TCE concentrations ranged to 183,000 parts per billion (ppb); EPA's proposed guideline for TCE in drinking water is 5 ppb. Levels of TCE declined with distance from the plant and decreased in the months following the spill. However, lower level TCE contamination was widespread and persistent, suggesting multiple releases. Within the plant, mean time-weighted occupational exposure to TCE of degreaser operators was 205 mg/m³; the recommended time-weighted exposure limit is 135 mg/m³. Mean short-term exposure was 1,084 mg/m³; the recommended short-term limit is 535 mg/m³. Seven of 9 exposed workers reported drowsiness, dizziness, or mental confusion. In exposed workers, mean urinary excretion of TCE metabolites rose from 298 µg/L pre-shift to 480 µg/L post-shift. On re-evaluation of the factory following improvements in ventilation and work practices, mean time-weighted occupational exposure to TCE had decreased to 84 mg/m³ and short-term exposure to 400 mg/m³; symptom frequency and concentrations of urinary TCE metabolites also were reduced. This episode demonstrates that community and occupational exposure to chemical toxins may share a common origin.

TOXIC CHEMICAL WASTES pose a major threat to public health. Since 1958, the 53 largest chemical manufacturers in the United States have disposed more than 750 million tons of unwanted chemical by-products,¹ including polychlorinated biphenyls (PCBs), chlorophenoxy herbicides, dioxins, pesticide intermediates, heavy metals, solvents, carcinogens, mutagens, and neurotoxins.

Various technologies exist for disposing of toxic wastes. Conceptually, the most attractive is detoxification, recycling, and reuse. This approach is already used widely in Europe,² and is employed in the United States for recovery of scrap metals.³ High-temperature incineration to produce water, carbon dioxide, hydrochloric acid, and other small molecules is a second approach. A third, still largely theoretical technique is bio-

logical degradation using microorganisms genetically modified to metabolize toxic chemicals.⁴ The least attractive, but heretofore most widely used alternative is dumping. Because short-term economics favors this approach, toxic waste chemicals have been discharged into landfills, canals, holding ponds, caves, deep wells, oil pipelines, rivers, lakes, and the oceans. In the United States, there exist an estimated 30,000 to 50,000 waste chemical disposal sites.⁵ They range in size from small operations with a few 55-gallon drums to massive accumulations such as Love Canal.

Two groups within the population—industrial workers and residents of communities adjacent to chemical plants and dumps—are at greatest risk of exposure to chemical toxins. Although these groups may be exposed to the same toxins from the same sources, their exposures differ in intensity, hazard, and amenability to correction. Workers may be exposed to high concentrations, usually by inhalation or dermal absorption. These exposures can produce acute or chronic illness. Community residents, by contrast, are exposed usually at lower levels. Their exposures can result from consumption of contaminated drinking water,^{6,7} inhalation of vapors, fugitive particulates,⁸ and combustion products^{9,10} or direct dermal contact.¹¹ Community exposures only occasionally produce acute illness, but carry the threat of chronic future toxicity.

In this report, we describe an episode of community and occupational exposure to the organic solvent trichloroethylene (TCE). A community population was exposed to TCE through consumption of drinking water contaminated by discharges from a pipe manufacturing plant. Workers within that same factory were exposed to TCE while degreasing metal. We compare and contrast the two aspects of this common-source exposure.

Background

On July 17, 1979, a pipe leading to an outdoor storage tank at a metal pipe manufacturing plant in Montgomery County, Pennsylvania, ruptured and released approximately 1,900 gallons of TCE. Trichloroethylene drained through soil into ground water and flowed over the surface into a nearby creek.

To evaluate the extent of this spill, ground water monitoring was begun by the Pennsylvania Department of Environmental Resources in July, 1979. Samples were obtained from 27 wells, 22 of which were within 0.5 km of the plant and the remaining 5 between 0.5 and 1.0 km. No earlier data were available because analytical methodology sufficiently sensitive to detect TCE in ground water was not available before July, 1979.

Concentrations of TCE in ground water within 1 km of the pipe plant ranged to 183,000 parts per billion (ppb); the U.S. Environmental Protection Agency's (EPA) Proposed Maximum Contaminant Level for TCE in drinking water is 5 ppb. Peak concentrations occurred in August, 1979 and then declined over the next 6 mo to below 10,000 ppb (Table 1). Levels of 5,000 to 10,000 ppb were found, however, to persist throughout the period from 1979 to 1985. Highest concentrations of TCE were observed within 0.5 km of the plant,

Table 1.—Mean Trichloroethylene (TCE) Concentrations in Well Water*, Collegeville, Pennsylvania (1979–1985)

Time period	No. samples	Mean TCE level (ppbt)
1979		
July	1	74,300
August	7	130,000
September	4	104,000
October	6	20,000
November	8	5,400
December	4	5,780
1980		
January	5	6,960
February	4	7,350
March	3	7,400
April	3	8,630
May	3	6,350
1984		
Annual mean	2	9,300
1985		
Annual mean	2	9,700

*Well located 0.2 km north of pipe plant.
†ppb = parts per billion.

particularly to the north. Levels decreased with distance (Fig. 1).

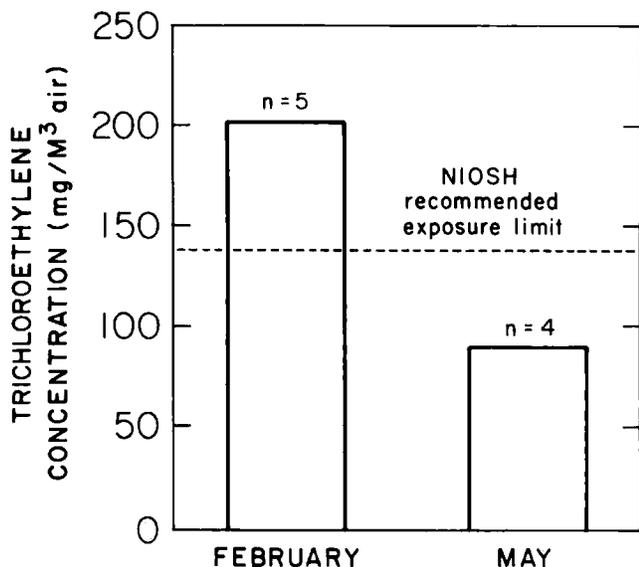
On further, more extensive evaluation, lower level TCE contamination of ground water was found to be widespread in Montgomery County. This pattern suggests the occurrence of multiple smaller releases from the pipe manufacturing plant as well as releases from other industrial establishments in the area, such as automobile repair shops and machine tool operations.

In residential drinking water wells, concentrations of TCE ranged to 1,000 ppb. Community exposure to TCE-contaminated water occurred through bathing, showering, and drinking. EPA estimated that more than 500 residents of communities near the pipe manufacturing plant were exposed to drinking water containing more than 5 ppb of TCE.

Methods

Community evaluation. In November, 1979, to evaluate community exposure to TCE, we screened 13 residents of Montgomery County who were potentially at high risk of exposure. Invited to participate were persons found in environmental testing to have highest concentrations of TCE in their well water; the survey was not population-based.

A questionnaire was administered to all survey participants. It sought information on TCE exposures, on other chemical exposures, and on occurrence of signs and symptoms, such as headache, liver dysfunction, or neuropathy, potentially related to TCE exposure. First morning urine specimens were collected from all survey participants. They were preserved with crystalline thymol, refrigerated, and analyzed colorimetrically for total trichloro-compounds (trichloroacetic acid and trichloroethanol) by the method of Tanaka and Ikeda¹³ in



*Time-weighted average

Fig. 2. Mean 8-hr time-weighted average breathing-zone exposures to trichloroethylene at the liquid-vapor degreaser, Collegeville, Pennsylvania (February and May 1980).

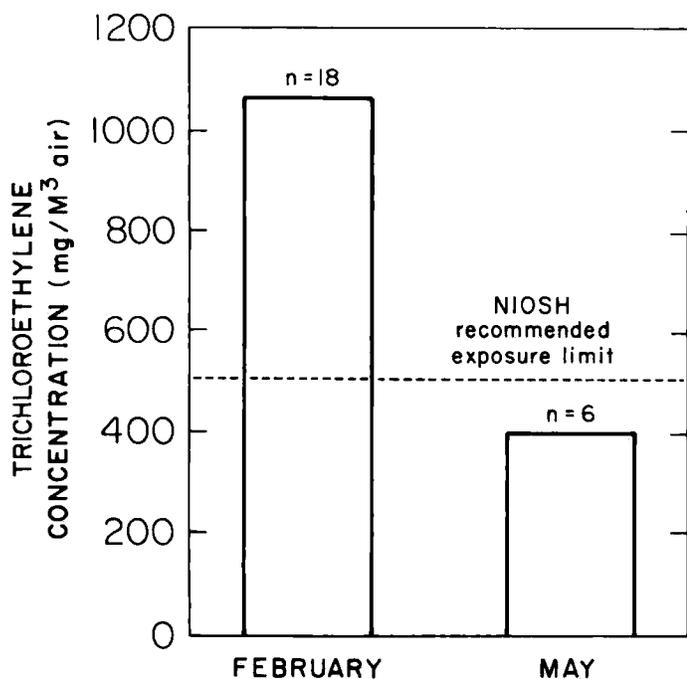


Fig. 3. Mean short-term breathing-zone exposures to trichloroethylene at the liquid-vapor degreaser, Collegeville, Pennsylvania (February and May 1980).

As a result of these findings, we made the following recommendations:¹⁵⁻¹⁷ (a) reduce hoist speed during loading and unloading degreasers to minimize vapor drag-out, (b) alter position of viewing windows to permit visual access to degreasers without removing covers, (c) calibrate performance of the freeboard chiller to

control height of vapor level,²⁴ (d) avoid unnecessary lifting of degreaser covers, and (e) avoid standing over the degreaser when cover is unavoidably open.

Re-evaluation in May, 1980, after implementation of most of these recommendations, showed that 8-hr TWA personal breathing-zone exposures to TCE had decreased to between 37 and 144 mg/m³; all were within the recommended exposure limit (Fig. 2). Concentrations of TCE in 4 of 18 short-term samples still, however, exceeded the recommended short-term exposure limit of 535 mg/m³ (Fig. 3). These residual exposures were attributed to improper work practices, such as unnecessarily removing degreaser covers and standing over open degreasers.

Medical evaluation in February, 1980, included 9 (75%) of 12 exposed workers and 9 unexposed comparison subjects. Seven of the exposed group were male. Mean ages of the groups were 42.7 and 46.4 yr, and mean durations of employment 4.4 and 9.4 yr. Examiners were not blind in regard to workers' exposure status. Seven (77.8%) exposed workers reported acute symptoms consistent with TCE exposure; no control workers reported such symptoms (Table 2). Liquid-vapor degreaser operators noted an average of 4.25 symptoms, cold degreaser operators 2.0 symptoms, and crane operators 1.5 symptoms (Fig. 4). In pre-shift urine samples, mean concentration of total TCE metabolites in exposed workers was 297.5 mg/L. Mean post-shift excretion was 479.9 mg/L (Fig. 5). For trichloroethanol, concentrations rose from 97.9 mg/L pre-shift to 155.2 mg/L post-shift. For trichloroacetic acid, no increases were noted over the work shift: from 29.5 mg/L pre-shift to 29.4 mg/L post-shift. No clustering of chronic disease was apparent in this small group.

In follow-up medical examination in May, 1980, 11 (92%) of the 12 exposed workers (including the 9 exposed workers evaluated in February) and the same 9 comparison subjects were evaluated. Numbers of workers reporting symptoms in the two groups were 7 (63.6%) and 1 (11.1%) (Table 2). Compared to February, significant reduction was noted in mean number of symptoms reported per worker (Fig. 4) to 1.5 for liquid-vapor degreaser operators, to 1.0 for cold degreaser operators, and to 0.75 for crane operators. Significant reduction was noted also in intrashift increase in excretion of TCE metabolites (Fig. 5). No abnormalities were noted in kidney or liver function test results.

Discussion

The two population groups in the United States principally at risk of exposure to toxic chemicals are (1) residents of communities adjacent to chemical plants and waste disposal sites and (2) industrial workers. The episode described in this report compares and contrasts the different, but interrelated exposures of these two groups. It illustrates that their exposures may share a common origin.

Community residents may be exposed to waste chemicals through ingestion of contaminated ground or surface water,^{6,7,18} or through inhalation of airborne mate-

Table 2.—Reported Prevalence of Symptoms in Workers Exposed and Unexposed to TCE, Collegetville, Pennsylvania, 1980

Symptoms	February		May	
	Exposed (N = 9)	Unexposed (N = 9)	Exposed (N = 9)	Unexposed (N = 9)
Fatigue	7	—	4	—
Light-headedness	4	—	1	—
Sleepiness	4	—	6	—
Eye irritation	4	—	3	—
Cough	1	—	2	—
Shortness of breath	4	—	1	—
Dyspnea on exertion	3	—	—	—
Skin irritation	2	—	1	—
Palpitations	—	—	1	1
Nausea	3	—	2	—
Headache	1	—	—	—
Prevalence of one or more symptoms	7(77.8%)	0(0.0%)	6(63.6%)	1(11.1%)

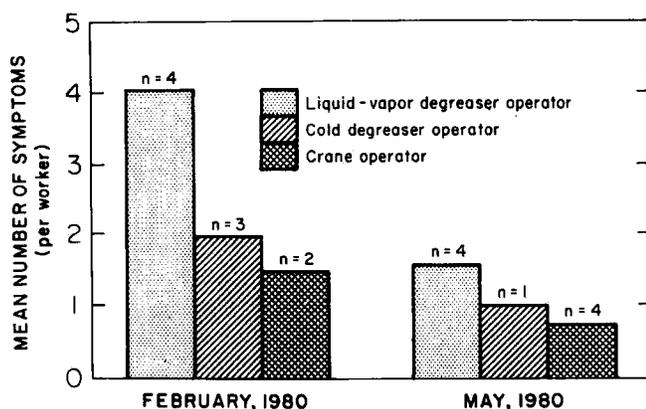


Fig. 4. Mean number of symptoms per worker exposed to trichloroethylene, Collegetville, Pennsylvania, 1980.

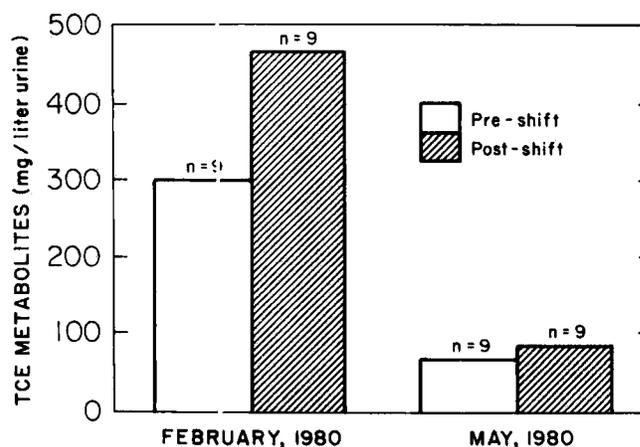


Fig. 5. Urinary excretion of trichloroethylene metabolites, Collegetville, Pennsylvania, 1980.

rials.¹⁹ Families living near the Love Canal, for example, were exposed to lindane and organic solvents which overflowed the canal. Factory workers adjacent to the Hyde Park Landfill in Niagara Falls were exposed to airborne lindane, mirex, and dioxins which had settled in interior dust.⁸ Assessment of health effects of community exposures to chemical wastes may be very difficult because these exposures are usually of lower intensity than those of workers, and adverse health effects may, therefore, be long delayed in their onset. Epidemiologic evaluation of such exposures must be targeted carefully and linked to results of environmental studies.²⁰ Sensitive and specific indicators are needed to identify possible early manifestations of neuropathy, renal, and hepatic dysfunction, and reproductive impairment.²¹

Workers at waste disposal sites may be exposed to toxic waste chemicals in the routine handling and disposal of industrial toxins, or during emergencies when wastes burn, explode, or are acutely released. In routine operations, workers may be splashed with acids, exposed to solvent and pesticide fumes, or coated with

sludges. Those risks are magnified for workers who must investigate or decontaminate abandoned dump sites or participate in emergency clean-ups. A particular hazard to emergency workers is exposure to toxic products formed during waste combustion: overheated polyvinyl chloride (PVC) can evolve hydrochloric acid,²² acrylonitrile can produce hydrogen cyanide,²³ polychlorinated biphenyls (PCBs) can form tetrachlorodibenzofurans,²⁴ and chlorinated benzene can yield tetrachlorodibenzodioxins.²⁵ Emergency workers, particularly in rural areas, are seldom prepared to deal safely with such fires, and episodes of toxic inhalation have been reported among firefighters exposed to burning chemical wastes.^{9,10}

Despite their differences, it is important to recognize that community and occupational exposures to toxic waste chemicals are not infrequently of common origin. Innovative techniques^{2,4} for primary prevention of occupational exposures and for the recycling of hazardous wastes can simultaneously protect against both occupational and community exposures.

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