

## Occupational Fatalities Associated With 2,4-Dichlorophenol (2,4-DCP) Exposure, 1980–1998

2,4-Dichlorophenol (2,4-DCP) is a feedstock chemical primarily used to produce the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D). In October 1998, the U.S. Environmental Protection Agency (EPA) was notified of the death of a worker acutely exposed to 2,4-DCP. Follow-up investigation by EPA, the Occupational Safety and Health Administration (OSHA), and CDC's National Institute for Occupational Safety and Health (NIOSH) identified four earlier deaths associated with acute 2,4-DCP exposure, which occurred during 1980–1992. All of these incidents resulted in rapid death after dermal exposure to the heated liquid form of the chemical. This report describes the five deaths associated with 2,4-DCP exposure (presented in the order in which they were identified) and provides recommendations for preventing additional deaths.

### Case Reports

**Case 1.** On October 12, 1998, a 29-year-old man employed at a Michigan chemical company producing 2,4-D was sprayed with 2,4-DCP from a leak in tubing while he was using steam to clear a blocked pump. The worker bypassed the nearest safety shower and used a locker room shower, where he became unconscious. Resuscitation attempts were unsuccessful, and the worker was pronounced dead at a hospital 1 hour after exposure. Skin surfaces exposed to 2,4-DCP included his forearms, right knee, right thigh, and face. Except for chemical burns on his face and extremities and pulmonary edema, the autopsy findings were unremarkable. 2,4-DCP was found in his blood (7.2 mg/L free 2,4-DCP, 13.1 mg/L total 2,4-DCP) and urine (4.8 mg/L free 2,4-DCP, 6.2 mg/L total 2,4-DCP). Death was attributed to acute dichlorophenol intoxication.

**Case 2.** In 1991, a 33-year-old man working at a factory in France was splattered over portions of his right thigh and arm with pure liquid 2,4-DCP while disposing of industrial waste (1). He walked away from the scene and washed himself with water without undressing. He experienced a seizure, collapsed within 20 minutes of exposure, and died after unsuccessful attempts at resuscitation. 2,4-DCP was found in his blood (24.3 mg/L), urine (5.3 mg/L), bile (18.7 mg/L), and stomach (1.2 mg/L).

**Case 3.** In September 1980, a 45-year-old man working at the same facility as the decedent in case 1 sustained skin and upper-airway exposure after being sprayed by steam containing 2,4-DCP. The worker bypassed the nearest safety shower, started decontamination using an unalarmed shower in a dressing area, and then moved to an alarmed shower, which automatically notified emergency personnel and summoned an ambulance. He sustained thermal burns to his skin, mouth, and upper airway, lost consciousness, and died despite resuscitation attempts. An autopsy revealed cutaneous burns on his neck, upper chest, back, and thighs; pulmonary congestion with alveolar hemorrhage; and moderately severe hepatocellular fatty change. His larynx was congested in a manner consistent with a steam/chemical burn, but the trachea was unremarkable, suggesting only upper airway exposure to the steam and 2,4-DCP. No reliable data on 2,4-DCP concentration in biologic fluids were available.\* The final pathologic diagnosis was "acute steam and dichlorophenol exposure."

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\*Analytic methods used to measure 2,4-DCP in biologic fluids were developed after 1980.

*2,4-DCP Exposure — Continued*

**Case 4.** In April 1992, a 64-year-old man at a chemical facility in England was using steam to unblock a clogged pump carrying 2,4-DCP (2,3). A pump seal failure allowed steam and 2,4-DCP to spurt onto his face and neck. Death occurred 20 minutes after exposure.

**Case 5.** In April 1985, a 33-year-old man working at an Arkansas manufacturing facility was splashed with a solution containing 51% 2,4-DCP<sup>†</sup> while moving a hose used to transfer the material. The solution covered 60%–65% of his body surface area (head, chest, neck, abdomen, arms, and thighs). When paramedics arrived, he was unconscious and convulsing on the shower room floor. He was transported to a hospital and pronounced dead approximately 90 minutes after exposure. An autopsy revealed first-degree chemical burns on exposed skin surfaces; swollen, red, sloughed mucosa of the larynx, trachea, and bronchi; focal hemorrhage and considerable hemorrhagic frothy fluid in the lungs (with fluid extruding through his mouth and nostrils); blue/tan swollen esophageal mucosa; and reddened mucosa and turbid hemorrhagic fluid in the stomach. Microsections of the brain revealed intense congestion and petechial hemorrhages. Serum total dichlorophenol concentration at postmortem was 67 mg/L. The final pathologic diagnosis was “acute chlorinated phenolic exposure and 60% chemical burns.”

*Reported by: Office of Pollution Prevention and Toxics, US Environmental Protection Agency, Occupational Safety and Health Administration, Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.*

**Editorial Note:** 2,4-DCP is a white solid at room temperature, but liquifies at 111 F–116 F (43 C–45 C). The liquid is rapidly absorbed through the skin. 2,4-DCP is not believed to be used outside the chemical industry, although small amounts may be present in drinking water when chlorination converts other phenolic compounds into this chemical (4). An estimated 200 U.S. workers are potentially exposed to 2,4-DCP. As of 1998, at least eight U.S. facilities were known to use or handle 2,4-DCP. Annual worldwide production is estimated at 88 million pounds (5). No OSHA, NIOSH, or American Conference of Governmental Industrial Hygienists exposure limits exist for 2,4-DCP.

The mechanism by which 2,4-DCP causes death is uncertain, but this and other chlorinated phenols are known to uncouple oxidative phosphorylation (6). Most production of adenosine triphosphate, the carrier of free energy in cells, occurs through oxidative phosphorylation. Uncoupling oxidative phosphorylation at the mitochondrial level leads to profound disturbance of energy production and may have caused the rapid deaths described in this report. A characteristic sequence of signs in animals given lethal doses of solid 2,4-DCP is consistent with the clinical progression noted in these cases and includes tremors, muscle weakness, loss of coordination, clonic convulsions, dyspnea, coma, and respiratory arrest (4). Although three of the decedents in this report also were exposed to steam, the reported symptoms and autopsy findings suggest that steam exposure did not play a substantial role in these deaths. Finally, postmortem drug screens were negative in all five cases, which excludes interaction with a drug or medication as a potential explanation for the deaths.

Potentially exposed workers, their supervisors, and health and safety staff should be aware of the hazards associated with exposure to 2,4-DCP, especially when the chemical is in the liquid state. In an April 1999 letter and a February 2000 chemical advisory (7),

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<sup>†</sup>This solution also contained (in order of diminishing proportion) parachlorophenol, orthochlorophenol, monochloroacetic acid, 2,6-dichlorophenol, phenol, and 2,4,6-trichlorophenol.

*2,4-DCP Exposure — Continued*

EPA and OSHA notified facilities believed to use 2,4-DCP of these fatalities and provided recommendations to prevent additional morbidity and mortality. Standard safe work procedures should be developed and disseminated to workers involved in tasks having potential 2,4-DCP exposure. Engineering controls and source reduction methods should be adopted to eliminate the potential for exposure. Detailed recommendations for appropriate protective clothing for dermal protection and respirators for inhalation protection were specified in the EPA/OSHA chemical advisory (7). Health and safety staff decontaminating exposed workers should wear appropriate personal protective equipment and should participate in drills to ensure proficiency while wearing this gear.

Any skin contact with liquid 2,4-DCP should be considered a life-threatening medical emergency. Safety showers should be located in the immediate vicinity of work areas having potential for 2,4-DCP exposure. These showers should be alarmed so that assistance is summoned promptly. Exposed skin should be flushed for at least 15 minutes, and contaminated clothing must be removed. Because 2,4-DCP is lipophilic and has relatively low water solubility (7), the use of water for skin flushing may lead to a protracted decontamination process. Additional research is needed to identify more effective agents for skin decontamination. Treatment for 2,4-DCP intoxication is supportive, and there is no known antidote.

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**Erratum: Vol. 49, No. 17**

In the article, “Morbidity and Mortality Associated With Hurricane Floyd—North Carolina, September–October 1999,” on page 371, a name was misspelled in the “Reported by” section: J Dolzinger, MD, Pitt Memorial Hospital, Greenville, North Carolina, should be J Dolezal. Also, a credit was missing: S Lynn, North Carolina Dept of Health and Human Svcs.

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**Suspected Brucellosis Case Prompts Investigation  
of Possible Bioterrorism-Related Activity —  
New Hampshire and Massachusetts, 1999**

*Brucella* species, particularly *B. melitensis* and *B. suis*, are potential agents of biological terrorism (1,2). This report describes the public health and law enforcement assessment of a suspected case of brucellosis in a woman, in which the atypical clinical presentation and suspicious circumstances surrounding the case raised the possibility of biological terrorism. Although the investigation did not identify evidence of biological terrorism, the safe resolution of the case illustrates the value of integrated clinical, public health, and law enforcement biological terrorism preparedness and response.

On March 25, 1999, a 38-year-old woman who resided in New Hampshire was admitted to hospital A in New Hampshire with fever, myalgia, and weakness, which progressed over 3 days to respiratory failure requiring mechanical ventilation. On day 22, after 3 weeks of intensive care, the patient was transferred to hospital B in Boston, Massachusetts. Paired serum specimens obtained on day 4 and day 22 showed a 16-fold rise in titer (from 1:20 to 1:320) for *Brucella* antibodies by slide agglutination testing at hospital B. Cultures of blood were negative for *Brucella* species.

Hospital personnel interviewed family members who reported no history of traditional risk factors for *Brucella* exposure (e.g., relevant food, infected animal contact, or travel history). Although the rapid respiratory decompensation was not typical for brucellosis infection, the serologic findings met the surveillance case definition for brucellosis (3). As a result, hospital B made a routine case report of brucellosis to the Boston Public Health Commission (BPHC) on day 23.

On day 24, the patient's family reported to hospital personnel that the patient's illness might have been caused by exposure to "laboratory flasks" and "cultures" kept in her apartment by her boyfriend. He was described as a foreign national studying marine biology who was formerly affiliated with a local university but recently had returned to his country of citizenship. On day 25, the patient's family brought laboratory flasks, petri dishes, and culture media to hospital B from the patient's apartment. Several contained an unidentified clear liquid, and some were marked with dates from the 1980s. Infection-control staff at hospital B were notified of the laboratory-like materials on day 27. The positive *Brucella* antibody serology in association with the unusual laboratory-like equipment in the patient's residence and the acknowledged potential for *Brucella* species to be used as a bioterrorist agents raised concerns among the infection-control staff that this case might be associated with a bioterrorist event or unintentional exposure to