Uploaded to the VFC Website

2018

This Document has been provided to you courtesy of Veterans-For-Change!

Feel free to pass to any veteran who might be able to use this information!

For thousands more files like this and hundreds of links to useful information, and hundreds of "Frequently Asked Questions, please go to:

Veterans-For-Change

If Veterans don't help Veterans, who will?

Note: VFC is not liable for source information in this document, it is merely provided as a courtesy to our members & subscribers.

Reprinted from JAMA® The Journal of the American Medical Association
August 4, 1989, Volume 262

Tear Gas—Harassing Agent or Toxic Chemical Weapon?

Howard Hu, MD, MPH; Jonathan Fine, MD; Paul Epstein, MD, MPH; Karl Kelsey, MD, MOH; Preston Reynolds, MD, PhD; Bailus Walker, PhD, MPH

Tear gas has gained widespread acceptance as a means of controlling civilian crowds and subduing barricaded criminals. The most widely used forms of tear gas have been *o*-chlorobenzylidenemalononitrile and ω-chloroacetophenone. Proponents of their use claim that, if used correctly, the noxious effects of exposure are transient and of no long-term consequences. The use of tear gas in recent situations of civil unrest, however, demonstrates that exposure to the weapon is difficult to control and indiscriminate, and the weapon is often not used correctly. Severe traumatic injury from exploding tear gas bombs as well as lethal toxic injury have been documented. Moreover, available toxicological data are deficient as to the potential of tear gas agents to cause long-term pulmonary, carcinogenic, and reproductive effects. Published and recent unpublished in vitro tests have shown *o*-chlorobenzylidenemalononitrile to be both clastogenic and mutagenic. Sadly, the nature of its use renders analytic epidemiologic investigation of exposed persons difficult. In 1969, eighty countries voted to include tear gas agents among chemical weapons banned under the Geneva Protocol. There is an ongoing need for investigation into the full toxicological potential of tear gas chemicals and renewed debate on whether their use can be condoned under any circumstances.

(*JAMA*. 1989;262:660-663)

TEAR gas is a weapon that has become familiar to the world. Hardly a week goes by without press reports of tear gas being used in a public setting, typically the dispersal of demonstrators or the subdual of a barricaded criminal. Recent years have seen the use of large amounts of tear gas in several countries, including Chile; Panama; South Korea; and the Gaza Strip and West Bank, Israel.

Tear gas is actually the common term for a family of chemical compounds that have been otherwise referred to as "harassing agents" because of their ability to cause temporary disablement. Some 15 chemicals have been used worldwide as tear gas agents. Four of these—ω-chloroacetophenone (CN), *o*-chlorobenzylidenemalononitrile (CS), 10-chloro-5,10-dihydrophenarsazine, and α-bromo-α-tolunitrile—have been used extensively.[1] In the United States, Britain, and Europe, CN and CS have been employed most widely. *o*-Chlorobenzylidenemalononitrile, in particular, is a weapon that has gained widespread acceptance as a means of controlling civilian populations during disturbances.

The widespread use of tear gas agents naturally raises the question of their safety. Relatively little, however, has appeared in the mainstream medical literature regarding their toxicology. In general, authors of review articles have averred that, if used correctly, the noxious effects of exposure are transient and of no long-term consequence.[2-4] Much emphasis has been given to the findings of the Himsworth Report,[5] the results of an inquiry by a committee appointed by the British Secretary of State for the Home Department following the use of CS in Londonderry, Northern Ireland, in 1969. In addition to investigating the use of CS in Londonderry, the committee reviewed a wide range of scientific data. Its main conclusion was that while exposure to CS can be lethal, most likely in the form of toxic pulmonary damage leading to pulmonary edema, such an occurrence would only be at concentrations that were several hundred times greater than the exposure dosage that produces intolerable symptoms.

Many questions remain, however. Epidemiologic inquiry following the use of tear gas under actual field conditions has been almost completely absent.

THE USE OF TEAR GAS IN SEOUL, SOUTH KOREA

This lack of information became apparent to us during a July 1987 visit to Seoul, South Korea, during the course of which we gathered information on the use and effects of tear gas. Political demonstrations resulting in the use of tear gas had taken place in Seoul, Pusan, Taegu, Kwangju, Taejon, and Inchon—almost every major city in South Korea—during the month of June. By its own account, the government had used 351 200 tear gas canisters and grenades against civilian demonstrators in that month (*New York Times*. July 1, 1987;sect 1:8). We interviewed more than a hundred people, including hospital and medical school staff, medical and other university students, individuals who had been exposed to tear gas, bystanders, religious and community leaders, and officials of the US Embassy in Seoul.

A compilation of our findings, including interviews, results of physical examinations, and a community epidemiology survey, was summarized in monograph form.[6] We were able to ob-

From the Channing Laboratory, Brigham and Women's Hospital (Dr Hu), and Department of Internal Medicine (Dr Epstein), Harvard Medical School, and the Department of Epidemiology (Dr Hu) and Occupational Health Program (Dr Kelsey), Harvard School of Public Health, Boston, Mass; Physicians for Human Rights, Somerville, Mass (Dr Fine); Department of Internal Medicine, Cambridge (Mass) Hospital (Dr Epstein); American Medical Student Association, Preston, Va (Dr Reynolds); Duke University Medical School, Raleigh, NC (Dr Reynolds); and Graduate School of Public Health Sciences, State University of New York—Albany (Dr Walker).

The opinions expressed herein are those of the authors alone.

Reprint requests to Physicians for Human Rights, 58 Day St, Somerville, MA 02144 (Dr Fine).

tain a sample of tear gas chemical that represented the typical compound being used by the South Korean government. Mass spectrometry analysis identified the substance as pure CS. We were struck by the indiscriminate use and effects of tear gas on bystanders and others in proximity to the demonstrators being teargassed. We heard pervasive accounts of police firing canisters and throwing tear gas grenades directly into crowd gatherings and enclosed spaces, such as rooms, motor vehicles, and subway corridors. Persons who were close to the exploding tear gas grenades and canisters commonly sustained penetrating trauma from plastic fragments that was exacerbated by the presence of tear gas chemical. Many individuals sustained blistering skin burns from direct contact with the tear gas powder. There were several accounts of people who were alleged to have experienced more severe toxic injuries requiring hospitalization. Our community survey of small shopkeepers close to university campuses where student demonstrations were common uncovered some symptoms, including cough and shortness of breath, among the interviewees and their children that persisted for weeks up to the time of the survey. Physicians noted that patients with asthma and chronic obstructive lung disease who were exposed to tear gas wafting into hospital wards through open windows experienced deterioration in lung function, some to a serious degree requiring a lengthened hospital stay.

We were especially struck by the lack of information available to the Korean medical community on tear gas. According to Korean scientists we interviewed, the government withheld the chemical composition of agents employed. Local laboratories apparently refused to perform chemical analyses on tear gas substances for fear of government reprisal. No guidelines had been issued to the public or health authorities on methods of treating injuries or toxic effects of tear gas weapons. Hospital authorities would not share with us medical records data, citing fear of persecution. Senior and junior physicians, without exception, confirmed that no one dared to undertake laboratory, clinical, and epidemiologic studies of tear gas effects for fear of serious governmental reprisals.

Similar findings have been reported in inquiries into the use of tear gas in Gaza and the West Bank of Israel. Of particular concern are allegations that exposure to tear gas has been associated with increases in miscarriages and stillbirths.[7,8]

CS AND OTHER TEAR GAS AGENTS

While poisonous gases have been used sporadically in military history as early as 428 BC, when burning wax, pitch, and sulfur were used in wars between the Athenians and Spartans, it took the birth of the modern chemical industry and the circumstances of World War I for the invention of chemical warfare agents to begin in earnest. Agents that could temporarily incapacitate victims were among the first to be developed and were deemed "harassing agents." Of these, chemicals that produce lacrimation and uncontrollable blepharospasm, otherwise known as "tear gas agents," became the most popular.

Harassing agents are capable of a number of immediately perceived effects: intense irritation of the eyes, causing crying or temporary blindness; irritation of the mucous membranes of the nose, trachea, or lungs, causing coughing; irritation of the throat and stomach, with the induction of vomiting and possibly diarrhea; and irritation of the skin. Most harassing agents will cause several or all of these reactions to a greater or lesser extent.

For many years, CN was the most widely used agent by civil and military authorities. It is the active ingredient in Mace and is still used in many parts of the world. Dissatisfaction with its potency and chemical instability, however, led military scientists to search for alternative agents.

In the 1950s, the Chemical Defence Experimental Establishment (Porton, England) developed CS. *o*-Chlorobenzylidenemalononitrile is a white crystalline substance that is usually mixed with a pyrotechnic compound in a grenade or canister for use. Its useful form is intended to be a smoke or fog of suspended particles. Effectiveness in crowd control derives from its properties as an extremely severe skin and mucous membrane irritant and lacrimator, even at minute doses. Instantaneous conjunctivitis with concomitant blepharospasm, burning, and pain are characteristic. These symptoms are exacerbated in hot or humid weather. *o*-Chlorobenzylidenemalononitrile that has been micronized and mixed with an antiagglomerant or treated with a silicone water repellent (formulations known as CS1 and CS2, respectively) can remain active for days to weeks when dusted on the ground.

Since its introduction, CS has virtually replaced CN as the riot control agent of choice in England and the United States. During the Vietnam war, the United States developed an array of delivery vehicles for CS, including small pocket grenades, the "Mighty Mite" (a continuous-spray device used in caves and tunnel systems), and 58-kg cluster bombs dropped from helicopters and planes.

TOXICOLOGY OF CS

Military studies among volunteers have noted that, in most cases, removal from exposure to CS results in fairly rapid recovery with cessation of all symptoms within minutes.[9] Proponents of the use of CS believe that, when used properly, high or prolonged exposure to the substance would be precluded by an individual's natural aversion to remaining in an area where the substance is present (United Kingdom patent specification 967 660; 1960). Its popularity among military and police authorities stems partly from comparisons with the other tear gas agents, which suggests that CS is a more potent lacrimator and seems to cause less long-term injury, particularly with respect to the eye.

Inhalation toxicology studies[10-12] at high levels of CS exposure, however, have demonstrated its ability to cause chemical pneumonitis and fatal pulmonary edema. In situations in which high levels of exposures have occurred, the same effects, as well as heart failure, hepatocellular damage, and death, have been reported in adults.[5p231,8,13] An infant exposed to CS in a house into which police had fired CS canisters to subdue a mentally disturbed adult developed severe pneumonitis requiring therapy with steroids, oxygen, antibiotics, and 29 days of hospitalization.[14]

The respiratory concentration of CS that would be lethal for 50% of healthy adults has been estimated to be 25 000 to 150 000 mg/m^3 per minute, based on animal studies.[15] When detonated outside, a CS grenade generates a cloud 6 to 9 m in diameter, at the center of which a concentration of 2000 to 5000 mg/m^3 can be produced, with concentrations rapidly tapering off at the periphery.[16]

If detonated in an enclosed space or in clusters, however, much higher levels of exposure could be expected. Moreover, chemical weapons have generally been noted to be notoriously uneven in their dispersal.[17]

Oral toxicology studies[18,19] have noted the ability of CS to cause severe gastroenteritis with perforation. Metabolic studies[20,21] indicate that absorbed CS is metabolized to cyanide in peripheral tissues.

The potential for CS exposure at levels seen in the field to result in significant generation of cyanide at the tissue level is controversial.[21,22] Authors who downplay this possibility reason that

one would have to inhale massive quantities that could only occur if the gas were used improperly, and that severe pulmonary injury would overshadow the effects of cyanide generation.[2] However, this argument ignores the ingestion of tear gas chemical that can occur with pharyngeal deposition of incompletely dispersed CS compound and swallowing of respiratory secretions.

Contact burns and the development of skin sensitization with contact dermatitis have been described in a number of experimental and observational studies on animals and humans.[15,23-25] This is in keeping with the many skin burns encountered during our inquiry.

Studies have not adequately examined the possibility that CS at less than high concentrations can cause lasting pulmonary effects. One study[11] of CS exposure on volunteers showed no increase in airway resistance following several exposures. However, only seven healthy military recruits were examined and volunteers with a history of asthma were excluded. Previous studies have shown that single exposures to high levels of respiratory irritants similar to CS have been associated with the development of reactive airways disease syndrome in some individuals.[26] The symptoms of prolonged cough and shortness of breath that were reported in our community survey suggest that such an effect may have occurred as a result of CS exposure in South Korea.

Only one study[27] has assessed the effect of CS on pregnancy in animals and it found no significant effect. The Himsworth committee[5] found no significant increase in abortions, stillbirths, or congenital abnormalities in geographic districts of tear gas use, comparing a 9-month period of heavy tear gas exposure to a previous 9-month period. More sophisticated epidemiologic studies do not exist.

POTENTIAL FOR GENOTOXICITY

The agent CS can alkylate sulfhydryl groups and, possibly, DNA.[28-30] As such, it is potentially genotoxic. The agent has not, however, been well studied for its genetic effects in vitro or in vivo. Some researchers have shown CS to be mutagenic in both Ames *Salmonella* assays[30] and in the L5178Y tk+/tk− mouse lymphoma forward mutation assay.[31] Zeiger et al[32] reported CS to be questionably mutagenic in the Ames assay, testing lower doses than Von Daniken et al.[30] When Von Daniken et al accounted for the toxicity of CS, its mutagenic effects increased by a factor of 2. Thus, the toxicity of this agent can make it difficult to study in vitro. Cytogenetic testing done by the National Toxicology Program (unpublished data, 1988) and the National Institute of Environmental Health Sciences has shown CS to be clastogenic in Chinese hamster ovary cells and to induce sister chromatid exchanges in these same mammalian cells. Other researchers[33,34] have reported negative results in testing CS for mutagenicity on the Ames test. A single study[27] of animal embryos did not reveal any teratogenic effects of CS.

The agent CS has been found to suppress nonspecific esterase activity in mouse skin sebaceous gland.[35,36] This property has been suggested for use as a screening test for the carcinogenic potential of suspected chemicals.[36] A study[37] of the carcinogenicity of CS in A/J strain mice and Sprague-Dawley-Wistar rats done at the Edgewood Arsenal reported CS to induce more pulmonary tumors in exposed animals after 4-week inhalation experiments, conducted at 0, 50, and 500 mg/m^3 per minute. The increase, however, was not strictly dose related and of borderline statistical significance. This report concluded that CS was not significantly tumorigenic in these animals, but observed that chronic exposure to very low concentrations of CS is of greater concern and should be further studied. In addition, Marrs et al[38] studied the inhalation toxicity of CS in rodents. Owing to the limited number of animals studied, they were also unable to draw a firm conclusion concerning the tumorigenicity of CS.

TOXICITY OF CN

Although CS has been the most widely used and well studied of the tear gas agents, other agents are still available. Of particular importance is CN, which is still being produced in the United States[39] and was reported to have been used in the West Bank and Gaza Strip (*Jerusalem Post*. May 6, 1988:1).[8,40] ω-Chloroacetophenone is generally acknowledged to be of greater toxicity than CS, being more likely to cause permanent corneal damage on contact with the eye[19] and primary and allergic contact dermatitis.[41,42] The maximum safe inhaled dose has been estimated to be several times lower than that of CS[3] and at least five deaths have been reported following the use of CN grenades in confined spaces.[2,43,44] Little is known regarding its potential for chronic pulmonary or genotoxic effects or for potential effects on reproduction.

TREATMENT

Most exposures to CS and CN typically cause immediate and severe irritation of the eyes and respiratory tract, accompanied by blepharospasm, lacrimation, coughing, sneezing, and rhinorrhea, followed rapidly by a burning sensation of exposed skin surfaces and the mouth. Some persons also experience nausea and vomiting, photophobia, and headache. These symptoms usually disappear within a few hours after removal from exposure.

Clinically, signs of exposure consist of blepharospasm, conjunctival injection, palpebral edema, and lacrimation. Management is conservative, beginning with aeration and the disposal of all contaminated clothing in plastic bags. Skin should be washed, although contact with water can briefly exacerbate skin symptoms from CS exposure, and a mild alkaline solution (6% sodium bicarbonate, 3% sodium carbonate, and 1% benzalkonium chloride) has been recommended to hasten decontamination of CS.[16] Persistent eye irritation can be relieved with application of a local anesthetic preparation and a patch. Contact dermatitis may respond to corticosteroid creams and antipruritics.

Exposure to high concentrations of tear gas by inhalation or ingestion, as may occur in an enclosed space or in proximity to an exploding tear gas device, should be treated cautiously. Pulmonary injury with edema can be delayed and the patient should be kept under observation for several days. Initial treatment may begin with humidified oxygen; bronchodilators and ventilator therapy may be necessary. Prophylactic antibiotics have been suggested.[2] We believe a thiocyanate assay should be considered in cases of ingestion or extremely high exposure.

Persons with preexisting lung disease such as asthma or emphysema should be observed carefully for exacerbation of their condition.

COMMENT

From a toxicological perspective, there is a great need for epidemiologic and more laboratory research that would illuminate the full health consequences of exposure to tear gas compounds such as CS. The possibility of long-term health consequences such as tumor formation, reproductive effects, and pulmonary disease is especially disturbing in view of the multiple exposures sustained by demonstrators and nondemonstrators alike in some areas of civilian unrest. The development of tolerance to CS, a phenomenon that has been confirmed in studies of human volunteers,[45] has likely increased the length and intensity of exposure sustained by some individuals. Unfortunately, the same social conditions that accompany political unrest and the use

of tear gas make epidemiologic research difficult, if not impossible.

We also believe, however, that the evidence already assembled regarding the pattern of use of tear gas, as well as its toxicology, raises the question of whether its further use can be condoned under any conditions. Fact-finding missions to areas of civil unrest in addition to South Korea have frequently observed security forces using tear gas against peaceful demonstrators and not uncommonly against civilians in no way involved in protests.[7,46]

We recognize it is not adequate for health professionals simply to study and reject as "medically unacceptable" every modality of riot control. As with many hazards—for example, asbestos, industrial toxic emissions, or radiation—there is an important role for the independent professional: to study, document, analyze, and report on such hazards and to advise government on what does and does not carry an acceptable risk. If a weapon is found to present too serious a risk, it is then the responsibility of those in charge of public safety to decide on alternatives. In doing so, active consultations should be sought with medical and public health specialists who are independent of law enforcement agencies and, ideally, drawn from both governmental and nongovernmental agencies and institutions. In the United States, for example, health specialists might be recruited from medical school faculties, state and local health departments, the Public Health Service, and the Centers for Disease Control.

At a time when the world has recently seen the recurrence of the use of mustard gas, this time in the Middle East, it is also worthy to note that in 1969, at the United Nations General Assembly, 80 countries voted to ban the use of *any* chemical in war, including tear gas, under the Geneva Protocol.[47]

Finally, we have been persuaded that in many instances in which harassing agents have been used, dialogue and negotiation could have been pursued. Often, public order might be better served if riot police are not called immediately to duty. It is the hallmark of repressive regimes to equate the voicing of dissent with disorder and to deny opponents the freedom of assembly and speech, rights guaranteed universally among signatories to the Universal Declaration of Human Rights.[48]

This study was supported by a grant from Physicians for Human Rights.

Many thanks to Patti Goldman of Health Research Group, Washington, DC, for assistance in requesting information through the Freedom of Information Act.

References

1. *Health Aspects of Chemical and Biological Weapons*. Geneva, Switzerland: World Health Organization; 1970.
2. Ballantyne B. Riot control agents. *Med Annu*. 1977/1978:7-41.
3. Beswick FW. Chemical agents used in riot control and warfare. *Hum Toxicol*. 1983;2:247-256.
4. Danto BL. Medical problems and criteria regarding the use of tear gas by police. *Am J Forensic Med Pathol*. 1987;8:317-322.
5. Himsworth H. *Report of the Enquiry Into the Medical and Toxicological Aspects of CS (Orthochlorobenzylidene Malononitrile), II: Enquiry Into Toxicological Aspects of CS and Its Use for Civil Purposes*. London, England: Her Majesty's Stationery Office; 1971.
6. *The Use of Tear Gas in the Republic of Korea: A Report by Health Professionals*. Somerville, Mass: Physicians for Human Rights; 1987.
7. *The Casualties of Conflict: Medical Care and Human Rights in the West Bank and Gaza Strip*. Somerville, Mass: Physicians for Human Rights; 1988.
8. *Report on the Status of Palestinian Children: Uprising in the Occupied Territories (9 Dec 1987–9 Dec 1988)*. East Jerusalem, Israel: Save the Children. In press.
9. Punte CL, Owens E, Gutentag PJ. Exposures to ortho-chlorobenzylidene malononitrile. *Arch Environ Health*. 1963;6:366-374.
10. Ballantyne B, Callaway S. Inhalation toxicology and pathology of animals exposed to *o*-chlorobenzylidene malononitrile (CS). *Med Sci Law*. 1972;12:43-65.
11. Chapman AJ, White C. Case report: death resulting from lacrimatory agents. *J Forensic Sci*. 1978;23:527-530.
12. Kaczmarek B, Gaszynski W. Ultrastructure of the rabbit's lung tissue after administration of the CS preparation. *Acta Med Pol*. 1977;18:327-328.
13. Krapf R, Thalmann H. Akute Exposition durch CS-Rauchgas und linische Beobachtungen. *Schweiz Med Wochenschr*. 1981;11:2056-2060.
14. Park S, Giammona ST. Toxic effects of tear gas on an infant following prolonged exposure. *AJDC*. 1972;123:245-246.
15. Sanford JP. Medical aspects of riot control (harassing) agents. *Annu Rev Med*. 1976;27:412-429.
16. Wiegand DA. Cutaneous reactions to the riot control agent CS. *Milit Med*. 1969;134:437-440.
17. Sidel VW, Goldwya RM. Chemical and biologic weapons: a primer. *N Engl J Med*. 1966;274:21-27.
18. Ballantyne B, Swanston DW. The comparative acute mammalian toxicity of 1-chloroacetophenone (CN) and 2-chlorobenzylidene malononitrile (CS). *Arch Toxicol*. 1978;40:75-95.
19. Gaskins JR, Hehir RM, McCaulley DR, Ligon EW Jr. Lacrimating agents (CS and CN) in rats and rabbits. *Arch Environ Health*. 1972;24:449-454.
20. Cucinell SA, Swentzel KC, Biskup R, et al. Biochemical interactions and metabolic fate of riot control agents. *FASEB J*. 1971;30:86-91.
21. Jones GRN, Israel MS. Mechanism of toxicity of injected CS gas. *Nature*. 1979;228:1315-1316.
22. Jones GRN. Verdict on CS. *Br Med J*. Oct 16, 1971:170.
23. Chung CW, Giles AL Jr. Sensitization of guinea pigs to alpha-chloroacetophenone (CN) and ortho-chlorobenzylidene malono-nitrile (CS), tear gas chemicals. *J Immunol*. 1972;109:284-293.
24. Holland P, White RG. The cutaneous reactions produced by CS and CN when applied directly to the skin of human subjects. *Br J Dermatol*. 1972;86:150-155.
25. Schmunes E. Industrial contact dermatitis: effect of the riot control agent ortho-chlorobenzylidene malonitrile. *Arch Dermatol*. 1973;107:212-215.
26. Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome (RADS). *Chest*. 1985;88:376-384.
27. Upshall DG. Effects of *o*-chlorobenzylidene malononitrile (CS) and the stress of aerosol inhalation upon rat and rabbit embryonic development. *Toxicol Appl Pharmacol*. 1973;24:45-59.
28. Jones GRN. CS in the balance. *New Scientist*. 1971;50:690-692.
29. Toxicity of CS. *Lancet*. 1971;2:698. Editorial.
30. Von Daniken A, Friederich U, Lutz WK, Schlatter C. Tests for mutagenicity in *Salmonella* and covalent binding to DNA and protein in the rat of the riot control agent *o*-chlorobenzylidene malononitrile (CS). *Arch Toxicol*. 1981;49:15-27.
31. McGregor DB, Brown A, Cattanach P, Edwards I, McBride D, Caspary WJ. Responses of the L5178Y tk+/tk− mouse lymphoma cell forward mutation assay, II: 18 coded chemicals. *Environ Mol Mutagen*. 1988;11:91-118.
32. Zeiger E, Anderson B, Haworth S, Lawlor T, Mortelmans K, Speck W. *Salmonella* mutagenicity tests, III: results from the testing of 225 chemicals. *Environ Mol Mutagen*. 1987;9(suppl 9):1-109.
33. Reitveld EC, Delbressine LPC, Waegemaekers THJM, Seutter-Berlage F. 2-chlorobenzylmercapturic acid, a metabolite of the riot control agent 2-chlorobenzylidene malononitrile (CS) in the rat. *Arch Toxicol*. 1983;54:139-144.
34. Wild D, Eckhardt K, Harnasch D, King M-T. Genotoxicity study of CS (ortho-chlorobenzylidene malononitrile) in *Salmonella*, *Drosophila*, and mice. *Arch Toxicol*. 1983;54:167-170.
35. Barry DH, Chasseaud LF, Hunter B, Robinson WE. The suppression of non-specific esterase activity in mouse skin sebaceous gland by CS gas. *Nature*. 1972;240:560-561.
36. Chasseaud LF, Bunter B, Robinson WE, Barry DH. Suppression of sebaceous gland non-specific esterase activity by electrophilic α β-unsaturated compounds. *Experientia*. 1975;31:1196-1197.
37. McNamara BP, Renne RA, Rozmiarek H, Ford DF, Owens EJ. *CS: A Study of Carcinogenicity*. Edgewood Arsenal, Md: National Technical Information Service; 1973. Publication FB-TR-73027.
38. Marrs TC, Colgrave HV, Cross NL, Gazzard MF, Brown RFR. A repeated dose study of the toxicity of inhaled 2-chlorobenzylidene malononitrile (CS) aerosol in three species of laboratory animals. *Arch Toxicol*. 1983;52:183-198.
39. *Riot Control Manual*. Saltsburg, Pa: Federal Laboratories; 1988.
40. *Israel and the Occupied Territories: The Misuse of Tear Gas by Israeli Army Personnel in the Israeli Occupied Territories*. London, England: Amnesty International; 1988.
41. Holland P, White RG. The cutaneous reactions produced by *o*-chlorobenzylidene malononitrile and ω-chloroacetophenone when applied directly to the skin of human subjects. *Br J Dermatol*. 1972;86:150-154.
42. Penneys NS, Israel RM, Indgin SM. Contact dermatitis due to 1-chloroacetophenone and chemical mace. *N Engl J Med*. 1969;281:413-415.
43. Gonzales TA, Vance M, Helpern M, Umberger CJ. *Legal Medicine, Pathology and Toxicology*. East Norwalk, Conn: Appleton-Century-Crofts; 1957.
44. Stein AA, Kirwan WE. Chloracetophenone (tear gas) poisoning: a clinico-pathologic report. *J Forensic Sci*. 1964;9:374-382.
45. Beswick FW, Holland P, Kemp KH. Acute effects of exposure to orthochloro-benzylidene malononitrile (CS) and the development of tolerance. *Br J Ind Med*. 1972;29:298-306.
46. *Panama 1987: Health Consequences of Police and Military Actions*. Somerville, Mass: Physicians for Human Rights; 1988.
47. Bazell RJ. CBW ban: Nixon would exclude tear gas and herbicides. *Science*. 1971;172:246-248.
48. *International Bill of Human Rights: Universal Declaration of Human Rights*. New York, NY: United Nations; 1978. Articles 19 and 20.

Printed and Published in the United States of America