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Three Fatalities Involving Phosphine Gas, Produced as a Result of Methamphetamine Manufacturing

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ABSTRACT: In August of 1996, Sheriff's deputies investigated the deaths of three individuals suspected to have been overcome by phosphine gas. Phosphine is an extremely toxic gas, and is generally seen in the farming industry where it is used as a grain fumigant. It can also be generated as a by-product during the manufacturing of methamphetamine. Chemicals and equipment consistent with the manufacturing of methamphetamine were noted at the location, as well as an apparent reaction mixture heated to near dryness. Dräger tubes detected an atmospheric phosphine concentration in excess of 0.3 parts per million (ppm), the Threshold Limit Value. Deputies had initially assessed the scene with no protective equipment, raising concerns about phosphine toxicity and the effects of exposure.

The objectives of this paper are to describe how phosphine is formed during the manufacture of methamphetamine, to review the toxicity, health effects and symptoms of exposure, to address the safety concerns regarding potential exposure to law enforcement personnel, and to describe the protective equipment available for personnel who respond to clandestine laboratories.

KEYWORDS: forensic science, phosphine gas, clandestine laboratories, methamphetamine manufacturing, hydriodic acid, red phosphorus, ephedrine, phosphorous acid, fumigant, pulmonary edema

Phosphine (hydrogen phosphide) is a poisonous gas that has been widely used in the farming industry. As a grain fumigant, it is generated by the action of water on metallic phosphides (1). Phostoxin® is one commercially available product which contains an aluminum phosphide mixture. The product is applied to the grain or wheat to protect against insects and rodents during storage or shipment. The atmospheric moisture from the grain releases the phosphine gas which can permeate inaccessible areas (1,2). Phosphine is also used in the manufacturing of semi-conductors and can be found as an impurity in the manufacturing of acetylene (3).

Phosphine is a colorless gas, and is odorless when pure (3). The "fishy" or garlic-type odor generally associated with phosphine gas is due to the presence of diphosphine and other impurities (3,4). The characteristic odor is detectable from 0.02 ppm to 3 ppm. However, the strength of the odor gives no indication of the phosphine concentration (3–5). Phosphine is spontaneously flammable in air when diphosphine is present (6–8). The lower explosive limit of phosphine is 1.8% in air, and it reacts violently with oxidizing chemicals. Phosphine can be generated through a variety of chemical reactions. Although literature discussions of phosphine chemistry appear scarce, a likely mechanism that accounts for the generation of phosphine in clandestine laboratory situations can be invoked.

The Production of Phosphine Gas in Clandestine Laboratory Situations

In the Ephedrine/Hydriodic acid/Red phosphorus method of methamphetamine manufacturing, hydriodic acid is frequently generated in-situ by combining iodine and red phosphorus in aqueous media. Phosphorous acid is also formed in this reaction. Above 180°C, this acid is thermally unstable and readily decomposes to form phosphine gas and phosphoric acid (7,9,10, written personal communication, Dr. Harold Goldwhite, Professor of Chemistry, California State University, Los Angeles, August 11, 1997). Phosphine will not form from phosphorous acid if water is present (10). Above 180°C, superheating occurs, driving off the hydriodic acid and aqueous components, and allowing the formation of phosphine gas. Superheating results from the inability of the apparatus to dissipate excess heat applied to the reaction mixture, either by using an inoperable condenser or by conducting a reaction without one. The odor produced during this reaction is thought to be due to diphosphine, which may be formed by the oxidation of phosphine with molecular oxygen (11, Goldwhite, H). If the boiling temperature is maintained under 180°C, phosphine will not be produced. These reactions are shown in Fig. 1.

Symptoms and Health Effects of Phosphine Exposure

Most of the literature references regarding the health effects of exposure to phosphine gas, address the industrial exposure among grain fumigators. The symptoms due to inhalation are well documented. The more common symptoms include headache, fatigue or weakness, thirst, pain or pressure in the diaphragm or chest, dyspnea or shortness of breath, nausea, vomiting, convulsions and coma. Some references also report vertigo, irritation of the lungs, coughing and a feeling of coldness. Symptoms can begin to occur as soon as the odor is obvious and have been reported with a phosphine concentration of 1 ppm (8).

The Threshold Limit Value (TLV) for phosphine is 0.3 ppm, which is recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) (4,5). The ACGIH also recommends a short-term exposure limit of 1 ppm, for no more than

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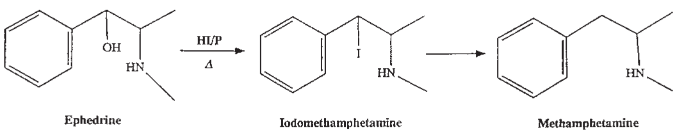
Generation of Methamphetamine

Ephedrine is converted to methamphetamine when refluxed with hydriodic acid and red phosphorus.



Generation of Hydriodic acid

Hydriodic acid can be generated by combining iodine and red phosphorus in aqueous media.

$$3I_2 + 2P \longrightarrow 2PI_3$$
$$2PI_3 + 6H_2O \longrightarrow 6HI + 2H_3PO_3$$

In the reaction between phosphorus triiodide and water, the major product containing phosphorus is phosphorous acid, H_3PO_3.

Generation of Phosphine

Phosphorous acid is thermally unstable and decomposes upon heating to give phosphoric acid and phosphine.

$$4H_3PO_3 \xrightarrow{heat} 3H_3PO_4 + PH_3$$

Generation of Diphosphine

Diphosphine can be produced by the oxidation of phosphine with molecular oxygen.

$$4PH_3 + O_2 \longrightarrow 2H_2O + 2P_2H_4$$

FIG. 1—*Chemical reactions depicting the generation of phosphine gas, produced during the manufacturing of methamphetamine.*

fifteen minutes per exposure and not more than four such exposures per day (4).

The World Health Organization has documented lethal effects at various concentrations. Pulmonary effects can occur after one hour of exposure to 8 ppm. Exposure becomes dangerous after one hour to concentrations up to 400 ppm, and death can occur after 30 min of exposure to levels over 400 ppm.

The available literature documents both occupational exposure to grain fumigators and accidental exposure in areas containing treated foodstuffs. Twenty-nine of thirty-three people on board a cargo ship carrying treated grain became acutely ill when the ship began to take on water. The increased moisture caused an increase in the rate of phosphine production from the hydrolysis of aluminum phosphide, which permeated the ship and caused one death. Levels of 0.5 ppm to 12 ppm were detected in various areas of the ship including the crew quarters (2).

Three individuals living in a house adjoining a granary developed symptoms on the day the grain was treated. Within three days, all three had expired (8). Likewise, two children playing on fumigated wheat expired within hours of exposure (12).

Sixty-seven grain fumigators exposed to levels between 8 and 33 ppm per eight-hour shift were evaluated for symptoms. About one-half of the workers experienced symptoms immediately. The remaining workers experienced a delay in the onset of symptoms from several hours to two days. As in other cases of phosphine exposure, symptoms included vertigo, headache, nausea, vomiting, epigastric and retrosternal pain, tightness in the chest and dyspnea. Although many of the victims were incapacitated for several days, none displayed any signs of exposure upon physical examination. Workers with intermittent exposure to 0.3 ppm reported only headache. There was no evidence of cumulative effects and no evidence of immunity after previous exposure. Symptoms reoccurred after each exposure (8).

Documented symptoms from chronic exposure include gastrointestinal and visual-speech motor disturbances and bronchitis (4,13). In cases where death occurs, autopsy results are nearly identical. Pulmonary edema (fluid in the lungs) appears to be the primary physical evidence of phosphine poisoning (1,14). Congestion of the lungs and other organs is also frequently seen.

Animal studies show similar findings (3). Phosphine is not readily detected in the body by standard testing, and has not been measured in the blood (14). Although the toxic action of phosphine is not fully understood, organs with the greatest oxygen requirement appear to be especially susceptible to damage. These include the brain, kidney, heart and liver, suggesting a wide distribution of phosphine in the tissues (8,15). Inhaled phosphine is freely absorbed by the lungs and a portion may be exhaled unchanged (3,8,14). Some phosphine is oxidized in the body to phosphite and hypophosphite, which are excreted in the urine (3,16).

Studies regarding the long-term health effects of phosphine exposure have been conducted primarily on behalf of workers in the grain industry. Research is directed toward phosphine interaction with heme proteins where it appears to effect the transport of oxygen by hemoglobin and inhibits the mitochondrial oxidative phosphorylation process (17,18). Although the precise nature of interaction is unknown, phosphine was seen to interact with hemoglobin in the presence of oxygen. A slow removal of oxygen with the formation of a hemichrome was seen when red blood cells were exposed to phosphine (16,19). Hemichromes are derivatives of methemoglobin and are associated with the precipitation of hemoglobin. Such precipitation is seen with reactions between phosphine and oxyhemoglobin, and studies have shown that the uptake of phosphine is related to this interaction (16). There is also a dose dependent change in the surface morphology of red blood cells, which become crenated as the concentration of phosphine is increased (16).

Some studies have also associated the formation of Heinz bodies with this increase in phosphine concentration. Heinz body formation was observed when phosphine concentrations were greater than 1.25 ppm (13).

In studies involving the mitochondria, phosphine was shown to interfere with state 3 and state 4 respiratory activity due to the interaction with cytochrome oxidase. This interaction results in the inhibition of electron transport from cytochrome to oxygen (3,17), the final electron acceptor in the oxidative phosphorylation process. The phosphine effects on cytochrome oxidase may involve a conformational change in the heme moiety (8,17).

Additional studies on the health effects of phosphine exposure involve chromosomal damage. Grain fumigators were evaluated for chromosomal aberrations. These, and in-vitro studies, demonstrate that lymphocytes exposed to phosphine showed an increase in chromosomal gaps and deletions three to five times more often than with control groups (18).

Case Report

In August of 1996, Los Angeles County Sheriff's deputies assisted with a "rescue responding" call concerning a possible drug overdose of three individuals in a Carson, California motel room. Deputies and paramedics entered the room, and ascertained that all

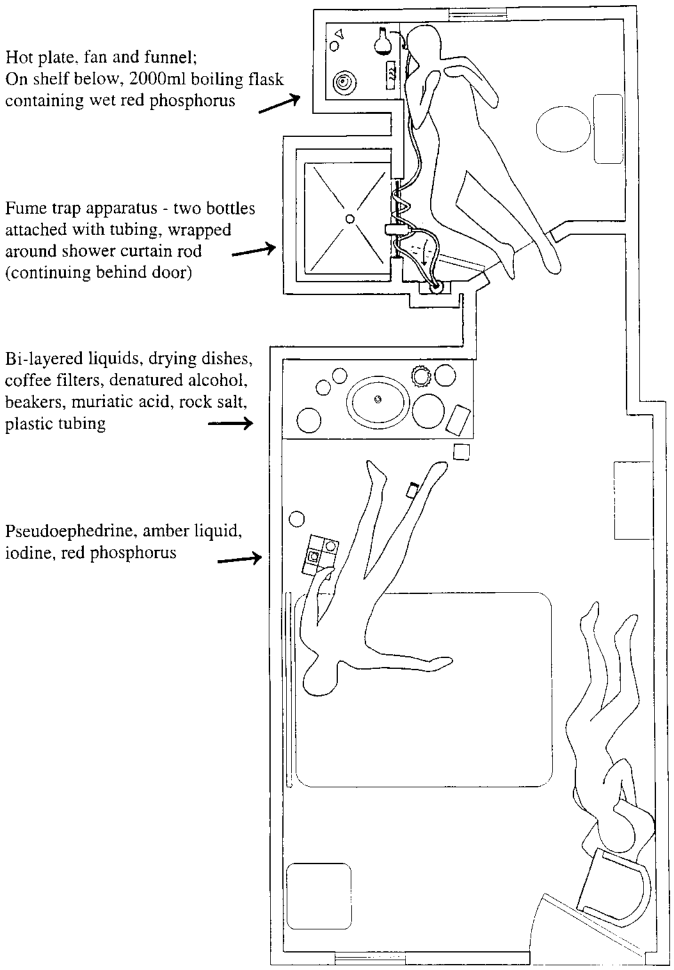


FIG. 2—*Diagram of the Carson, California motel room, showing the locations of victims in relation to chemicals and clandestine laboratory apparatus.*

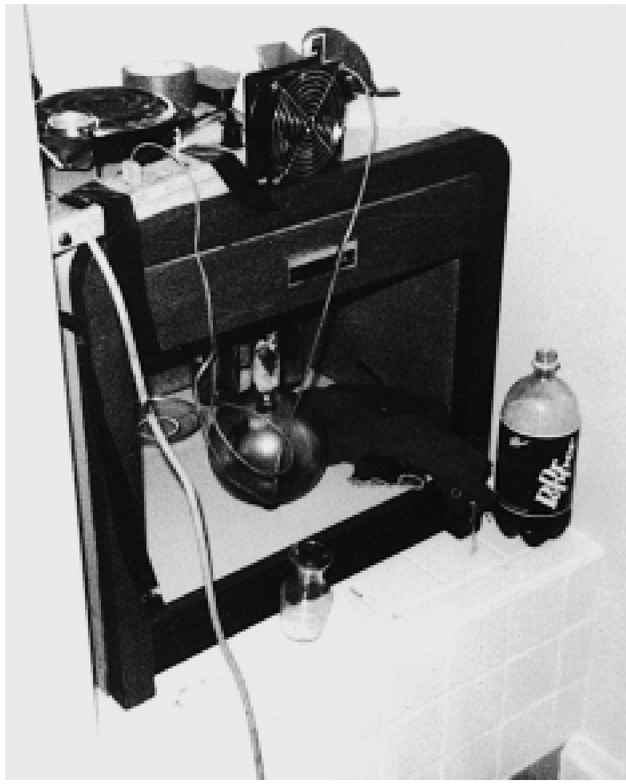


FIG. 3—*Polaroid photograph of the phosphine-generating flask as it was found in the motel room.*

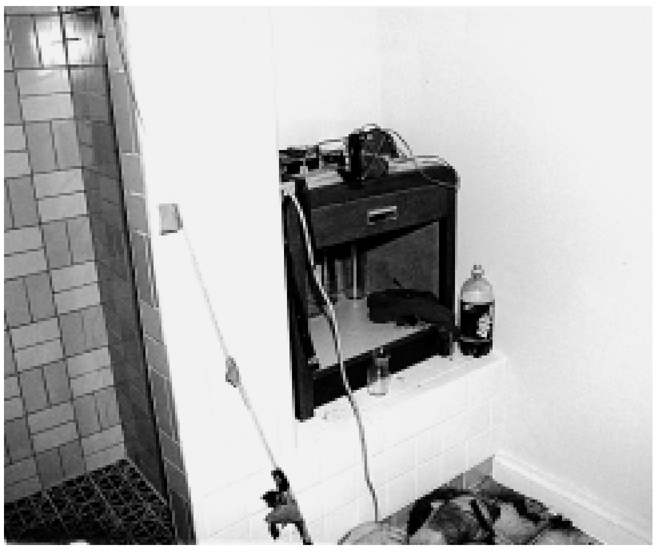


FIG. 4—*The original location of the flask showing the proximity of the nearest victim.*

three victims were deceased. None displayed any signs of injury. One victim lay on the bathroom floor while the two others lay in the adjoining room; one on the bed and one on the floor behind the door. A diagram is depicted in Fig. 2. The deputies noted a collection of typical drug lab chemicals and apparatus, including a 2000 mL flask in the bathroom. One deputy later reported a "heavy chemical odor." The air conditioner was on and a small bathroom window was slightly open. Bed sheets surrounded the perimeter of the door, as if the occupants were attempting to keep fumes from escaping. The deputies exited the location and notified the Bureau of Narcotics Enforcement Clandestine Laboratory Task Force. Due to the nature of the scene, poisoning by phosphine gas was suspected. Members of the Task Force and representatives from County Health Hazardous Materials assessed the scene utilizing the self-contained breathing apparatus. Dräger tubes were used to test for phosphine, which was detected at levels in excess of 0.3 ppm near the flask. (The scale for the tube used was 0.01–0.3 ppm, and the tube was completely discolored after one pump of the bellows.)

The first concern was the safe removal of the phosphine-generating flask. The flask contained a damp red sludge, and it was located on a shelf in the bathroom, near an electrical hot-plate (Fig. 3). The flask was stoppered and a small hole in the rubber stopper appeared to have accommodated a vent tube which lead to a fume-trap in the shower area. The end of this same tubing was found on the floor under the victim's head (Fig. 4). The flask was removed from the room, packed in ice and a sample was collected for later analysis. The odor trap apparatus presented another safety concern. It consisted of two bottles joined by tubing which was wrapped around a fixed shower curtain rod. One bottle contained a packed solid material while the second container enclosed a clear liquid.

FIG. 5—*The fume-trap and associated tubing wrapped around the shower curtain rod.*

The odor trap is depicted in Fig. 5. Due to the flammable nature of phosphine/diphosphine, the tubing was crimped off in sections before being cut from the shower curtain rod. The fume-trap was removed to allow for victim processing by the Department of the Coroner's Investigator.

Several samples were collected for analysis. The flask had contained red phosphorus, residual hydriodic acid and methamphetamine. Other chemicals at the location included iodine, muriatic acid, pseudoephedrine, bi-layered liquids containing methamphetamine and various solvents.

The Coroner's report listed, "Phosphine gas toxicity by inhalation" as a cause of death for all three victims. The autopsy report for the victim closest to the flask listed pulmonary edema as the primary result. The pathologist's conclusion stated, "the inhalation of phosphine is consistent with the pulmonary edema found at autopsy" (20). It should be noted that autopsy results were based upon anatomical findings only and the consideration that phosphine was detected at the location. Few testing procedures are noted in the available literature and those that are, generally involve the ingestion of metallic phosphides (21,22). There is no method for phosphine testing currently in practice at the Los Angeles County Department of the Coroner.

Additional Encounters with Phosphine Gas at Clandestine Laboratory Sites

A similar situation to the one just described was encountered around 1985 (14), by the Los Angeles Police Department. This case also involved the death of three individuals and the cause of death was presumed to be phosphine inhalation, based upon positive colorimetric test results for phosphine at the location. According to the responding chemist, a piece of tubing connecting a water source to the condenser became disconnected. This allowed the reaction mixture to overheat and boil up through the condenser and into the attached fume-trap tubing. Two flash fires occurred during disassembly of the apparatus indicating the presence of phosphine/diphosphine still present in the closed tube system.

While the detection of phosphine remains largely infrequent, the August 1996, incident promoted concern and increased awareness of this potential hazard. Since then, two more clandestine labs have been encountered where the odor was recognized and phosphine was detected. In March of 1997, a damp red phosphorus sample with a strong garlic odor was submitted. A Dräger test measured just over 1 ppm phosphine. Bi-layered liquids from the same case also emitted the odor, although not quite as strong. Phosphine, and presumably diphosphine, are slightly soluble in water and soluble in most organic solvents (3), explaining why the odor would transfer after an extraction. This solubility property has been noted by this laboratory with samples emitting strong garlic odors. A fume-trap tube containing residual liquid was washed with water, basified and extracted into pentane. The garlic odor was still detectable in the extract and was in fact quite strong. At the lab site in March, at least two deputies from the entry team complained of symptoms. One experienced headache only, lasting one hour. The second deputy experienced light-headedness, headache, fatigue and shortness of breath, lasting into the next day. This incident was similar to fumigator studies which showed that several people with similar exposure experienced different susceptibility (8). In addition, the subject at the location was semi-conscious upon arrival of the deputies and had to be pulled from the enclosed garage by the entry team.

Two months following this incident, criminalists responded to a clandestine lab involving a reaction mixture that had exploded. Phosphine was detected in several containers in concentrations from 0.1 ppm to over 4.0 ppm. The higher concentration was detected in the plastic tubing leading to a water fume-trap.

Discussion

The recent experiences with phosphine gas has led to increased concern for the safety of responding personnel. The fact that deputies have experienced symptoms of phosphine exposure demonstrates that more communication and training are needed. A close working relationship between narcotics officers and criminalists should be encouraged. Many entry teams do not wear personal protective equipment and should alternatively be trained on the recognition of specific hazards. It should be reiterated that offi-

FIG. 6—*Dräger Accuro® Pump and colorimetric tubes for the detection of phosphine gas.*

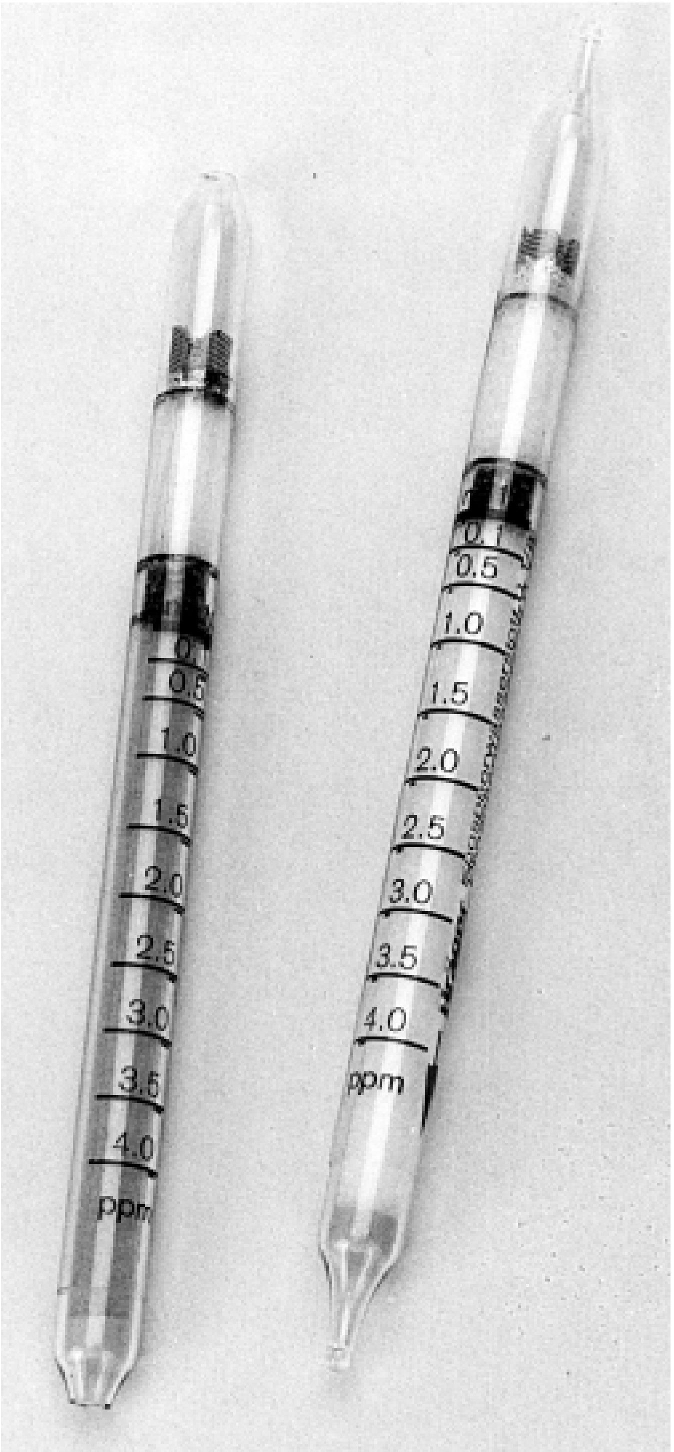


FIG. 7—*Phosphine exposed Dräger tube (darker colored tube) and an unexposed tube. (Upon exposure, the color change is white to violet).*

FIG. 8—*MSA phosphine-specific gas mask.*

cers need to exit the location and wait for specifically-trained personnel, when strong chemical odors are present. The deputies who initially assessed the Carson motel room with no protective equipment fortunately experienced no ill effects. The situation however, might have been more serious had they remained inside the location for more than a few minutes.

Due to these encounters, the potential hazard has been addressed in part by the purchase of a colorimetric air monitoring device (Fig. 6). A bellows pump draws air through a glass tube containing chemically treated inert material. The indicating layer changes color in the presence of phosphine gas (Fig. 7). The approximate concentration is determined by the length of discoloration in the tube. (Various manufacturers and measuring ranges for these colorimetric devices are available.) Atmospheres containing greater than 0.3 ppm (the TLV) require level B protection. A self-contained breathing apparatus (SCBA) or equivalent protection must be worn for exposure, as the standard air-purifying respirator (APR) does not protect against phosphine gas. A less expensive alternative to using the SCBA for initial air-monitoring is the use of a phosphine-specific gas mask. A mask of this type is shown in Fig. 8. These are similar to an APR, but contain a larger canister that is worn in a body harness. These masks are NIOSH approved for phosphine exposure up to concentrations of 30 ppm during routine use and up to 1500 ppm for escape. An indicator window on the canister changes color when the canister is spent. It should be noted that the phosphine-specific gas mask is not intended for oxygen-deficient atmospheres. When the oxygen level has fallen below 19.5%, an SCBA must be used regardless of the phosphine concentration present. The air-monitoring system and gas mask can be purchased through lab safety supply companies.

In addition to the phosphine hazards discussed herein, two other immediate environmental hazards should be considered and addressed. The previously mentioned oxygen-deficient atmosphere as well as the lower explosive limits of solvents present are of equal concern. Monitoring of these levels should occur along with the monitoring of phosphine during scene assessment.

The recent cases involving phosphine gas have demonstrated the need to better prepare ourselves for future encounters. There is a tendency to become complacent when we routinely encounter the small and simple clandestine laboratory. The Carson motel lab was not complex or elaborate, but a typical clandestine laboratory with an atypical potential for disaster. The best defense against injury and illness is through increased awareness and communication between criminalists and sworn field personnel. Narcotics officers should receive practical training on the specific hazards associated with clandestine laboratory investigations. Patrol officers and other first responders should be trained to recognize potentially lethal odors, and safety equipment should be purchased and utilized by responding personnel whenever possible. Injury and illness can be avoided by preparing personnel to expect future encounters with phosphine gas.

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References

1. Goodman LS, Gilman A, editors. Goodman and Gilman's the pharmacological basis of therapeutics. 8th rev. ed. New York: Pergamon Press, 1990.
2. Amdur MO, Doull J, Klaassen CD, editors. Cassarett and Doull's toxicology-the basic science of poisons. 4th rev. ed. New York: Pergamon Press, 1991.
3. World Health Organization. Phosphine and selected metal phosphides. Geneva: Environmental Health Criteria 1988;73:1–100.
4. Zaebst DD, Blade LM, Burroughs GE, Morrelli-Schroth P, Woodfin WJ. Phosphine exposures in gain elevators during fumigation with aluminum phosphide. Appl Ind Hyg 1988;3(5):146.
5. Patty FA, editor. Industrial hygiene and toxicology. Vol. II. New York: John Wiley & Sons, 1962.
6. Fluck E. The chemistry of phosphine. Topics in Current Chemistry. 1973;35:1–64.
7. Budavari S, editor. The Merck index. 12th rev. ed. Whitehouse Station: Merck Research Laboratories, 1996.
8. Hayes WJ. Pesticides studied in man. Baltimore: Williams & Wilkins, 1982.
9. Dillard CR, Goldberg DE. Chemistry-reactions, structures and properties. New York: The Macmillan Company, 1971.
10. Van Wazer JR. Phosphorus and it's compounds. New York: Interscience Publishers Ltd, 1958.
11. Robinson JR, Bond EJ. The toxic action of phosphine-studies with $^{32}PH_3$; terminal residues in biological materials. J Stored Prod Res 1970;6:133.
12. Heyndrickx A, Van Peteghem C, Van Den Heede M, Lauwaert R. A double fatality with children due to fumigated wheat. Eur J Toxicol 1976;9(2):113.
13. Potter WT, Rong S, Griffith J, White J, Garry VF. Phosphine-mediated Heinz body formation and hemoglobin oxidation in human erythrocytes. Toxicol Lett 1991;57(1):37.
14. Baselt RC, Cravey RH. Disposition of toxic drugs and chemicals in man. 3rd rev. ed. Chicago: Year Book Medical Publishers, 1989.
15. Poisondex toxicologic management. Topic: Phosphine. Micromedex Inc., 1974;1997;92.
16. Chin KL, Mai X, Meaklim J, Scollary GR, Leaver DD. The interaction of phosphine with haemoglobin and erythrocytes. Xenobiotica 1992; 22(5):599.
17. Chefurka W, Kashi KP, Bond EJ. The effect of phosphine on electron transport in mitochondria. Pestic Biochem and Physiol 1976;6:65.
18. Garry VF, Griffith J, Danzl TJ, Nelson RL, Whorton EB, Krueger LA, Cervenka J. Human genotoxicity: pesticide applicators and phosphine. Science 1989;246:251.
19. Chaudhry MQ, Price NR. A spectral study of the biochemical reactions of phosphine with various haemproteins. Pestic Biochem Physiol 1990;36(1):14.
20. Whiteman DB. Autopsy report. Los Angeles (CA): Department of the Coroner; 1996, August. Report No.: 96-06048.
21. Chan LTF, Crowley RJ, Delliou D, Geyer R. Phosphine analysis in postmortem specimens following ingestion of aluminum phosphide. J Analytic Toxicol 1983;7(4):165.
22. Guale FG, Stair EL, Johnson BW, Edwards WC. Laboratory diagnosis of zinc phosphide poisoning. Vet Hum Toxicol 1994;36(6):517.

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