

Inhalation of Phosphine Gas Following a Fire Associated with Fumigation of Processed Pistachio Nuts

Michael O'Malley MD MPH , Harvard Fong CIH , Martha E. Sánchez MA , Rachel Roisman MD MPH , Yvette Nonato MD FARM & Louise Mehler MD PhD

To cite this article: Michael O'Malley MD MPH , Harvard Fong CIH , Martha E. Sánchez MA , Rachel Roisman MD MPH , Yvette Nonato MD FARM & Louise Mehler MD PhD (2013) Inhalation of Phosphine Gas Following a Fire Associated with Fumigation of Processed Pistachio Nuts, Journal of Agromedicine, 18:2, 151-173, DOI: [10.1080/1059924X.2013.768135](https://doi.org/10.1080/1059924X.2013.768135)

To link to this article: <https://doi.org/10.1080/1059924X.2013.768135>



Published online: 29 Mar 2013.



Submit your article to this journal [↗](#)



Article views: 133



View related articles [↗](#)



Citing articles: 3 View citing articles [↗](#)

PRACTICE/CASE HISTORY

Inhalation of Phosphine Gas Following a Fire Associated With Fumigation of Processed Pistachio Nuts

Michael O'Malley, MD, MPH
Harvard Fong, CIH
Martha E. Sánchez, MA
Rachel Roisman, MD, MPH
Yvette Nonato, MD, FPARM
Louise Mehler, MD, PhD

ABSTRACT. On December 10, 2009, a fumigation stack containing aluminum phosphide became soaked with rain water and caught fire at a pistachio processing plant in Kern County, California. Untrained plant personnel responding to the fire had exposure to pyrolysis by-products, particulates, and extinguisher ingredients. Ten workers taken for medical evaluation had respiratory and nonspecific systemic symptoms consistent with exposure to phosphine gas. Six of the 10 workers had respiratory distress, indicated by chest pain, shortness of breath, elevated respiratory rate, or decreased oxygen saturation. Recommendations are made for the management of similar illnesses and prevention of similar exposures.

KEYWORDS. Aluminum phosphide, difficulty of breathing, eye irritation, fumigant, headache, nausea, O₂ saturation, pistachios, respiratory illness, spontaneous oxidation, vomiting

Michael O'Malley is affiliated with the Center for Health and the Environment University of California Davis, Davis, California, USA; and the Worker Health and Safety Branch, Department of Pesticide Regulation, California Environmental Protection Agency, Sacramento, California, USA.

Harvard Fong, Louise Mehler, Martha Sanchez, and Yvette Nonato are affiliated with the Worker Health and Safety Branch, Department of Pesticide Regulation, California Environmental Protection Agency, Sacramento, California, USA.

Rachel Roisman is affiliated with the Occupational Health Branch, California Department of Public Health, Richmond, California, USA.

This work was funded in part under a consulting contract between the California Department of Pesticide Regulation and the University of California, Davis.

Address correspondence to: Michael O'Malley, MD, MPH, University of California Davis, Center for Health and Environment, Building 3792, Room 129, Old Davis Road, Davis CA 95616, USA (E-mail: momalley@ucdavis.edu).

INTRODUCTION

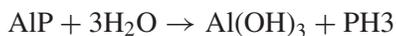
During commodity fumigations and rodent control operations, agricultural laborers handling or working in the vicinity of compounds that generate phosphine gas may develop serious respiratory problems and systemic illness. Phosphine-generating fumigants also present unique safety hazards in both handling and disposal because of the tendency of phosphine gas to spontaneously oxidize.^{1,2}

This report describes the investigation of a December 2009 fire that occurred near Wasco, California, after rainwater accidentally contaminated a stack of pistachios under fumigation with aluminum phosphide. The incident illustrates both the safety problems associated with handling aluminum phosphide and the inhalation toxicity of its phosphine by-product. For those unfamiliar with the toxicity of phosphine and the related safety issues, some background is provided in the section below.

BACKGROUND

Phosphide fumigants liberate phosphine (PH₃) gas on contact with either moisture in the environment or acid in the intestinal tract. Aluminum phosphide products such as Phostoxin and Fumitoxin are used for commodity fumigation and occasionally for rodent control.

Aluminum phosphide reacts with water to form phosphine gas:



Phosphine is odorless in its pure state, but the technical product may have a foul odor associated with impurities. These include substituted phosphines, diphosphine, methane, arsine, hydrogen, and nitrogen (Table 1). The presence of an odor cannot be relied on as a warning of toxic concentrations.¹⁻⁶

Phosphine Chemical Reactions

Phosphine tends to decompose to more stable oxidized forms of phosphorous, generating thermal energy in the process. The energy

released by the oxidation of a gram-mole of phosphine (PH₃; heat of formation 5.4 KJ/g-mole) to phosphoric acid (H₃PO₄; heat of formation -1271.7 KJ/g-mole) is an estimated 1277 KJ/g-mole. This energy release may occur explosively at phosphine concentrations above 1.8%, especially when trace amounts of diphosphine are present to catalyze the reaction.¹⁻⁶

Phosphine Mode of Action and Toxidrome

Once liberated, the phosphine gas generated by all three phosphide fumigants is identical, with the same chemical and physical properties (Table 1) and same expected toxidrome, or commonly associated set of signs and symptoms.

Mode of Action

Oxidized forms of phosphorous have the potential to cause corrosion after combining with water, forming acids of variable potency: hypophosphorous acid (H₃PO₂), phosphorous (phosphonic) acid (H₃PO₃), and phosphoric acid (H₃PO₄). Phosphine is also associated with the formation of hydroxyl radicals and decreased stores of glutathione.⁷

Phosphine can also act as a metabolic poison, disrupting mitochondrial production of energy. It targets cytochrome *c* oxidase, although inhibition of this enzyme is less pronounced in vivo than in vitro. Other biochemical targets studied have included cholinesterase, peroxidase, and catalase.^{8,9}

At the tissue level, the biochemical effects of phosphine cause histopathological effects similar to cellular hypoxia.¹⁰ Myocardium in patients showing cardiac injury, for example, shows vacuoles, cytolysis, and degeneration in muscle cells.¹¹ Degeneration of renal tubules and necrosis of liver cells may also occur.⁹

Toxidrome

Inhalation of phosphine gas tends to cause nonspecific symptoms. The [California illness data indicate that more than half of the reported cases involve nonspecific systemic symptoms (e.g., headache, nausea, diarrhea, and dizziness) without accompanying respiratory complaints.

TABLE 1. Phosphine Physical/Chemical Properties in Comparison to Analog Compounds From Group V of the Periodic Table: Ammonia, Stibine, and Arsine

Property	Ammonia	Phosphine	Arsine	Stibine
Molecular weight, g/mole	17.03	34.04	77.946	124.78
Formula	NH ₃	PH ₃	AsH ₃	SbH ₃
CAS no.	7664-41-7	7803-51-2	7784-42-1	7803-52-3
Melting point, °C	-77.7	-134	-116	-88
Boiling point, °C	-33.35	-87.7	-62.5	-17
Solubility in water, g/100 cc	31 at 25°C	0.036 at 17°C*	0.028 at 20°C	4.1 at 0°C
Density, g/L	.696	1.39	3.18	5.48
Vapor pressure, mm Hg at 25°C	$7.51 \times 10^{+3}$	$2.93 \times 10^{+4}$	11,000 (20°C)	>760 (20°C)
Appearance and odor, odor threshold	Sharp, cloying, repellent, like drying urine; 2.6 ppm average odor threshold	Fish/garlic odor; 0.02–3 ppm; no tests reported with current methodology	Disagreeable garlic odor, 0.5 ppm and above	Disagreeable
Heat of formation, kJ/g-mole	-45.9	5.4	66.4	145.11
Pattern of signs and symptoms (toxicidrome) after airborne exposure	Eye and upper respiratory symptoms ⁶¹	Headache, nausea, diarrhea and dizziness, dyspnea and cough, less irritating than ammonia ^{1,22}	Hemolysis and related renal effects, dyspnea, ⁶² minimal irritation	Hemolysis and related renal effects, dyspnea, ⁶³ minimal irritation

*Calculated from solubility of 26 cc phosphine/100 cc^{40,41}.

Respiratory symptoms (e.g., dyspnea and cough¹²) occur more frequently in conjunction with systemic symptoms than in isolation. Fatal prolonged inhalation exposures in confined spaces¹³ may include systemic effects and pulmonary edema, both thought to be caused by acidic by-products or oxidized phosphine.

Inhalation Exposures and Dose Response

Phosphine has been described as a strong respiratory irritant in a standard reference on pesticides published by the United States Environmental Protection Agency (EPA).¹⁴ The dose response for inhalation in humans is nevertheless uncertain (see discussion of exposure standards in the section below). Several animal studies examining exposures to phosphine air concentrations ranging from below the accepted human exposure limit (0.3 ppm; discussed below) to fatal concentrations (lethal concentrations for 50% of tested animals [LC₅₀]) are available. A 4-hour inhalation study of rodents found that only mild clinical signs of respiratory tract irritation occurred at the LC₅₀

concentration of 0.44 μ mole/L (calculated as equivalent to 15 mg/m³).¹⁵ In a 2-year chronic inhalation study with phosphine concentrations of 0.3, 1, and 3 ppm (at and just above the permissible exposure limit [PEL]), no pathological signs of respiratory irritation were present.¹⁶

Eye and Skin Irritation

Eye and skin irritation is not a feature of most cases of inhalation exposure to phosphine, perhaps because of its limited water solubility (Table 1). Nevertheless, outbreaks of eye irritation in California nut processing operations described in the 1980s and 1990s (Table 3: 73-KER-88, 79-KER-88) were ascribed to phosphine off-gassing from aluminum phosphide.¹⁷ Eye irritation probably does occur on direct contact with incompletely reacted fumigant dust (Table 3: 1996-1906).

Susceptibility of Children

Exposed children may be especially vulnerable to the systemic effects of phosphine, evidenced by differential fatal outcomes in

TABLE 2. Applications of Phosphine-Generating Fumigants Reported in California During 2009*

Active ingredient	Most frequently treated commodities	Commodity pounds	Rodent, mammalian pest control, unspecified	Total
Aluminum phosphide	Dried fruits, pistachios, almonds, walnuts, rice, wheat	49,369	58,395	107,764
Magnesium phosphide	Dried fruits, almonds, walnuts, rice, grains	7630	379	8009
Zinc phosphide	None	0	20,893	20,893
Phosphine/CO ₂ gas	Almonds, pistachios, dried fruit	30,203	0	30,203

*Data reported to California Pesticide Information Portal (CalPIP), available online at: calpip.cdpr.ca.gov.

cases involving both adults and children. These include exposures to phosphine associated with residential storage of aluminum phosphide treated grain,¹⁸ shipboard fumigation of grain,¹⁹ and aluminum phosphide treatment of rodent burrows immediately adjacent to an occupied dwelling.²⁰ From these limited case reports, it is uncertain whether the differential pediatric fatality rate is related to variant pathways for detoxifying phosphine or higher pediatric respiratory rates.

Ingestion of Phosphide Products

Between the 1980s and 1990s, many cases of poisonings from ingestion of phosphide fumigants were reported in India and elsewhere in Asia. Symptoms from these exposures included fatigue, nausea, headache, dizziness, thirst, cough, shortness of breath, tachycardia, chest tightness, paresthesia, and jaundice. A high mortality rate of 50% to 90% was due to ingestion, and cardiogenic shock was present in severe cases. Pulmonary edema was the most common cause of death.¹⁴ The frequency of pulmonary edema in these cases was far greater than in the occupational cases reported in California.

Ingestions are associated with additional symptoms including elevation of the ST segments on the electrocardiogram (ECG), ventricular and atrial arrhythmias, and hypotension unresponsive to pressors.¹² A handful of intentional exposure cases have been reported in California (Table 3), but have been reported most often in countries where access to phosphide fumigants is less restricted.^{9,21–34}

Biomonitoring

Because phosphine breaks down quickly to phosphate after absorption in the lungs, it is not usually possible to confirm poisoning with biological monitoring. In cases of ingestion, serum residues of aluminum may be elevated, but this is not an expected finding after inhalation exposure to phosphine. In the single case where elevated aluminum levels were reported in a case of suspected inhalation, there was limited documentation of the primary exposure.³⁵

Based on animal studies, zinc levels have been proposed as a marker of exposure to zinc phosphide. Using neutron activation analysis, it is possible to measure zinc levels in tissues as well as in serum samples.³⁶

At autopsy, or in cases of ingestion, identification of phosphine gas can confirm exposure.^{28,37} The analysis is typically not performed by commercial laboratories, which can present logistical difficulties. Even if a suitable commercial assay were available, aluminum phosphide samples might prove too unstable to safely ship.

A spot test using silver nitrate is sensitive enough to use in cases of ingestion.²⁹ Otherwise, these may be most easily overcome by measuring phosphine with a direct reading instrument (see industrial hygiene section below). This approach has the additional potential benefit of protecting autopsy staff or hospital personnel caring for patients who have ingested phosphine-generating fumigants.

In some instances, expired air may contain high enough levels of phosphine to spark or spontaneously ignite. In a suicide case described in 2009 by Wahab et al.,³⁸ burns near the hairline

TABLE 3. Selected Safety Problems Associated With Aluminum Phosphide, Zinc Phosphide, Magnesium Phosphide, or Phosphine Fumigations Reported to the California Illness Registry

ID number of index case, and/or priority case number	Description
1983	
1983-1351	A worker put fumigation tarps and packed trays of partially spent dust into a truck for removal after an application at a Bakersfield seed company. A tray of partially spent aluminum phosphide pellets wrapped in a plastic bag exploded, just after it was loaded onto the truck. Shortly afterwards, the worker developed breathing and talking difficulty, and dizziness. He was observed in the hospital for 24 hours, and lost 2 days from work.
1983-2159, 1983-2160	Grain elevator employees collecting samples of barley treated 8 days earlier with aluminum phosphide, noted a suspicious odor. Subsequent testing with a colorimetric tube showed 25 ppm of phosphine. Two affected employees developed headache, nausea, tightness in chest and stomach cramps.
1983-2253	An aluminum phosphide fire occurred at a spaghetti sauce factory in North Hollywood, following two attempts to dispose of unspent material from 62 trays of aluminum phosphide. Initially, 20 trays were placed in a barrel with liquid and a detergent, which resulted in a small explosion. A second explosion occurred when 42 trays were placed in a dry barrel. Four firefighters responding to the call developed nausea and were hospitalized for 40 hours. Plant employees were treated and released at a nearby emergency room. Four neighborhood residents were briefly admitted to the hospital for observation. A total of six cases were reported.
1983-2653, 1983-2671	Aluminum phosphide pellets were improperly disposed in a dumpster. While trying to extinguish the subsequent fire, case 1983-2671 inhaled smoke, causing shortness of breath, flushing in face, and headache. Case 1983-2653, a police officer, reported some irritation of the throat after responding to the fire. Case 1983-2675 was possibly exposed to fumes while covering story for TV news, but the investigation did not indicate whether symptoms occurred.
1984	
1984-2567, 1984-2610, 1984-2571	1984-2567—a worker employed by a Sacramento county almond grower reported not feeling well while working in a warehouse next to fumigation facility. No phosphine was found in the area with colorimetric tubes measurement. A co-worker, case 1984-2610, reported a slight headache. The following day at the same facility, case 1984-2571, a mechanic working in the shop area 200 ft from phosphine burning in an open area, also reported symptoms.
1986	
34-TEH-86, 1986-861	A grower placed aluminum phosphide pellets in a squirrel hole next to his house. The treated burrow apparently communicated with the basement of his home, where the reacting pellets released smoke. The smoke was discovered by his spouse, who called for emergency assistance. While responding to the call, seven fire fighters were exposed to the smoke and were hospitalized for observation.
1987	
1987-89	A grain fumigation was conducted in the fume room with aluminum phosphide. Some illnesses resulted because of improper aeration of fumigant, and failure to measure phosphine levels before entering the room.
1987-2924	A port worker unloading phosphine-treated rice from the bottom of a truck trailer developed nausea and headache. The Draeger tube reading showed phosphine levels in excess of the 0.3 ppm limit.
1988	
73-KER-88, 1988-2574; 79-KER-88, 1988-2911	Workers complained of conjunctivitis in two separate episodes in an almond and pistachio/sorting operation. Fourteen workers complained of eye irritation after a fumigation of the storage building was made the previous weekend and were diagnosed as either corneal abrasion or conjunctivitis. Eleven workers experienced a reoccurrence of eye irritations while working in an almond/pistachio processing plant. They were diagnosed as having "chemical conjunctivitis." ²⁷
1989-474	An unemployed man stowed away in a rice filled rail car that was being fumigated with aluminum phosphide pellets—in transit from Houston, Texas. He was found dead several days later when the train arrived in Colusa, California. ²³

(Continued)

TABLE 3. (Continued)

ID number of index case, and/or priority case number	Description
1990	
17-SJ-90, 1990-534	A rodenticide bait containing diphacinone and zinc phosphide was left under the sink in a San Joaquin county bank. The bait got wet from a leaking pipe, causing an apparent phosphine release. A colorimetric tube employed by the county hazardous materials team registered 5–10 ppm of an unspecified gas, but it was not possible to confirm the presence of phosphine with a specific detector tube. Eighteen ill employees sought medical attention for nausea, headache, and upper respiratory irritation.
27-MAD-90, 1990-842	Firefighters put out a fire in five piles of cotton debris, each measuring approximately 35 ft × 105 ft × 10 ft, used for fuel at a biomass co-generation plant. They were not initially aware that the piles were under fumigation with aluminum phosphide. After the fire, colorimetric tubes measurements taken by county investigators showed no residual phosphine. One plant employee and four firefighters had precautionary medical evaluations, but none reported symptoms.
1990-3004	Approximately 25% of 3200 pellets used remained unreacted after a commodity fumigation in Kern County. Because this possibly seemed too many to dispose of using soapy water, workers placed unused aluminum phosphide pellets in a 4-ft ³ bin and covered it with a tarpaulin. The bin exploded while two workers were checking it to see whether the aluminum phosphide completely reacted. One worker suffered facial burns, hair loss, a broken left leg, and a perforated eardrum. The other worker received facial cuts and burns.
81-BUT-90, 1990-2429	After fumigating a rice mill, employees of a Butte County pest control business disposed of the unspent residue in a dumpster behind their office in an industrial business park, starting a fire. Four firemen and one employee of an adjoining business developed headache and nonspecific gastrointestinal (nausea, stomach cramps, gas, and diarrhea), and constitutional symptoms (aching muscles, weakness, and fatigue). Three of the firemen were hospitalized overnight and four lost 2 days from work. Four additional firefighters received medical evaluation, but had no symptoms.
1991	
1991-330	Two trash men picked up a dumpster at a packing house. The dumpster contained Fumitoxin tablets which produced a chemical reaction and the truck started smoking. Thinking the truck was on fire, they dumped the refuse in an empty lot and called the fire department. Case 1991-330 developed a burning sensation in the nose and chest; his co-worker developed conjunctivitis. Both lost 2 days from work.
1993	
24-SD-93, 1993-1513	A navy applicator put incompletely inactivated aluminum phosphide in a dumpster, where it started a fire, possibly made worse by a worker who tried to extinguish it with water from a garden hose. Two firemen and seven bystanders were exposed, and most reported symptoms, including headache, metallic taste in the mouth, chest tightness, lightheadedness, weakness, and fatigue. A distasteful garlic odor was also noted by several of those exposed.
1993-742	After unloading five carloads of rice, an employee reported feeling ill. Monitoring done before aeration showed low levels of phosphine gas but there were no detectable levels after aeration. Symptoms reported were nausea, dizziness, "hot feeling."
1994	
1994-1468, 1994-1469, 1994-1470 1994-1047	Following a warehouse fumigation of seed garlic, the fumigator removed the aluminum phosphide dust and put it into a barrel and a wooden box. The dust in the box caught fire and three employees inhaled the smoke. A county agricultural inspector entered an unposted chamber that was fumigated 3 days earlier. She was there to do a prefumigation inspection with a pest control operator (PCO). A Draeger tube sample showed 4 ppm of phosphine in the chamber. She was taken to a community hospital for evaluation, complaining of headache, nausea, and vomiting.
1996	
1996-1906	As an employee removed cardboard boxes containing residue of spent aluminum phosphide tablets from raisin bins, a second employee placed new boxes with tablets in the bins. The first employee may have been exposed to the residual dust and developed a rash.

(Continued)

TABLE 3. (Continued)

ID number of index case, and/or priority case number	Description
41-TUL-96, 1996-1775	Nine workers became ill with nausea, headache, and dizziness while sorting almonds not previously treated with aluminum phosphide. An industrial hygienist investigation of the plant showed no detectable phosphine, but high levels of carbon monoxide were found in the plant.
1997	
4-STA-97, 1996-1872	Mill employees noted a noxious odor coming from a silo just filled with wheat fumigated and aerated 19 days earlier by a grain supplier. Monitoring detected 2 ppm of phosphine. Symptoms were present in two of five exposed employees and included tightness in the chest and shortness of breath.
1998	
1998-96	A fire occurred in agricultural warehouse building where spent aluminum phosphide ash was present. Fire investigators suspected that the ash ignited spontaneously—because of heat from a fire that started elsewhere, or ignited on contact with water used to extinguish the fire. A firefighter accidentally inhaled smoke and subsequently developed shortness of breath, dizziness, loss of balance, a fainting spell, nausea, and burning sensation in the chest.
1998-878, 1998-879	Fumigation of raisins took place in a chamber adjacent to a break room. Two employees developed headaches and nausea while in the break area, the following morning. A gas tech reading showed 1.0 ppm of phosphine in the break room air.
1999	
1999-662	A truck driver opened a fumigated rail car and took readings until the fumigant concentration dropped to prescribed levels. For unknown reasons, he remained on top of the car—instead of following procedure and climbing down between readings. He developed dizziness, abdominal pain, diarrhea, coughing, and ringing in the ears.
2000	
38-LA-00, 2000-558	A 46-year-old woman attempted to commit suicide by ingesting an aluminum phosphide tablet. She was hospitalized for 9 days. The investigator obtained the medical records and interviewed the woman's nurses. She previously attempted suicide 2 years ago. Symptoms were altered mental status, hypotension, respiratory failure, vomiting, nausea, slurred speech, lethargy, rales.
2001	
45-SD-01, 2001-708	A 50-year-old man stated he ingested a rodenticide obtained in Mexico and identified on the label as "fosfuro de zinc" [zinc phosphide]. He vomited in the ambulance. The doctor admitted him to the intensive care unit to monitor him for pulmonary edema.
2002	
39-LA-02, 2002-219	In an attempt to commit suicide, a 33-year-old woman supposedly ingested some pellets of zinc phosphide along with ambien and vicodin. In the emergency room, no odor was noted when she vomited. Due to the toxicity of zinc phosphide, it is unlikely she ingested it, since her symptoms were minor.
2003	
24-MAD-03, 2003-468	A farm worker, who had previously used aluminum phosphide at work, intentionally inhaled phosphine in a motel room and was subsequently found dead by housekeeping staff. There was a small amount of spent residue near his body and a plastic bag containing 20 unused aluminum phosphide tablets among his possessions.
30-MAD-03, 2003-515	A farmer intentionally ingested fumitoxin tablets. When he began vomiting, his wife had him transported to the hospital. He informed the medical staff of his ingestion. His condition deteriorated and culminated in his death 9 hours later.
47-RIV-03, 2003-1075	A man found his mother-in-law's body in his parked van, next to an empty container of aluminum phosphide tablets. The container had no US registration number, and was possibly purchased in Mexico. During autopsy, the coroner's staff noticed a garlic odor, confirmed to be phosphine by air sampling—necessitated evacuation of the building. Symptoms reported by two ill coroner's staff members included dry mouth, headache, runny nose, sore throat, dizziness, nausea, and vomiting.

(Continued)

TABLE 3. (Continued)

ID number of index case, and/or priority case number	Description
2004	
45-FRE-04, 2004-937	An almond processing plant worker mistakenly fumigated an extra 22 bins of almonds with phosphine gas. The next morning, this worker helped to open the bins and dumped the almonds onto the sorting belt. Fourteen cases of respiratory and systemic illness occurred. Symptoms included: headache, dizziness, nausea, vomiting, and respiratory irritation. Fumigant levels were not tested.
2005	
33-RIV-05, 2005-1307 to 1310	Three young Mexicans entered the United States clandestinely and rode in a fumigated rail car for 2 or 3 hours. They pried the hatch open with a stick, breaking a 3/16-inch braided aluminum seal. Darkness hid the placarding. Reported symptoms were dizziness, vomiting, difficulty breathing, loss of consciousness, and death. Case 2005-1309 survived; cases 2005-1307 and 2005-1308 did not. Case 2005-1310 involved an emergency room nurse with potential exposure from patient off gassing. Symptoms were itchiness, prickly red welts on the arms, burning sensation around the neck, and shortness of breath.
2006	
45-SBD-06, 2006-613	A warehouse forklift driver experienced symptoms when he inhaled fumes from spent aluminum phosphide tablets that ignited during disposal in a barrel of soapy water. He was taken to a hospital & admitted overnight. His symptoms were: painful eyes, stomach pain, and headache. Several violations were noted.
2008	
71-KER-07, 2007-1229	On 11/21/2007, an almond processing operation applied fumigant gas containing 2% phosphine and 98% carbon dioxide to several work areas in the plant. The structure was aerated until phosphine levels were below 0.1 ppm, measured with a continuous monitoring device. When workers returned on 11/26/2007, there was an odor noted. Twenty-three cases were reported. Symptoms reported were headache, nausea, dizziness, and eye or respiratory irritation in some cases. Air levels of phosphine at 1:30 PM on the day of the incident was recorded as 0.03 ppm.
2007-249	A teenaged girl tried to rid her home of mice by pouring 2% zinc phosphide (sold as gopher bait) into corners of two bedrooms. She said she read the label, but did not understand it. She developed symptoms within hours, including "pain behind the eyes" and chest tightness. She was taken to the emergency for evaluation but did not require treatment beyond removal from treated areas.
2007-713	A 57-year-old woman mixed 2% zinc phosphide bait pellets with water, in violation of the product label directions. As she prepared to pour the mixture into gopher holes in her yard, she tripped. The liquid splashed onto her face, body, and into her mouth. She developed extreme dizziness, blurred vision, foaming at mouth.
21-LA-07	A 20-year-old man intentionally ingested an unknown quantity of rat poison, possibly obtained from Mexico (labeled as "fosfuro de zinc"). He was taken to a hospital where he stayed for 2+ days for observation and treatment. Prothrombin time and International Normalized Ratio (INR) were initially measured because of confusion about the nature of the product ingested.
14-VEN-08, 2008-131	One week after ingesting brodifacoum in a suicide attempt in Ventura, California, a woman drank a slurry of coffee mixed with a gopher killer containing 2% zinc phosphide. She called family members to say goodbye. Her mother called the police who found her at the beach and took her for care. She was combative in the emergency room, requiring sedation and intubation. She developed hypothermia in the ICU (temperature of 93.4°F) that required treatment with external warming, but did not develop pulmonary edema or oxygen desaturation. Upon her discharge 2 weeks later, she was referred for long-term psychiatric care. Hospital staff called poison control because of concerns about secondary exposure from the gastric aspirate and exhaled air from her ventilator, but no cases of secondary illness in staff were reported.

found at autopsy were noted and suspected to be caused by ignition of phosphine elaborated from aluminum phosphide found in the stomach. A 2011 report by Shadnia and Soltaninejad³⁹ described two cases of aluminum phosphide ingestion complicated by facial burns. In both cases, flames were noted by medical personnel during the passage of a nasogastric tube, resulting in burns on the left side of the face in one patient and burns in the hair of the second patient.³⁹

Phosphine 8-Hour Exposure Standards

The current 0.3 ppm threshold limit value (TLV), calculated as an 8-hour time-weighted average, is intended to prevent systemic phosphine poisoning.^{40,41} The short-term exposure limit is 1 ppm. These standards are based primarily on the study reported in 1964 by Jones et al.⁴² describing phosphine exposures to Australian grain terminal workers. Most of the phosphine measurements reported were area samples, so it was difficult to identify the level of exposure associated with individual cases of illness and consequently difficult to identify levels of exposure that were tolerated without symptoms.

In 1998, the re-registration eligibility document (RED) for aluminum phosphide and magnesium phosphide published by the EPA suggested an 8-hour limit of 0.1 ppm, based on risk assessment from animal studies.⁴³ A subsequent paper published in 2004 took issue with the standard suggested in the 1998 RED, arguing that there was little reason to change the long established 0.3 ppm standard.⁴⁴

Immediately Dangerous to Life or Health (IDLH) Concentration

The phosphine IDLH concentration set by the National Institute for Occupational Safety and Health (NIOSH)⁴⁵ is 50 ppm, based on reviews by Beliles⁴⁶ and Henderson and Haggard.⁴⁷ Data for setting the standard were apparently extremely limited.

Environmental Monitoring Techniques

Techniques for monitoring phosphine in the environment include colorimetric tubes

measuring ranges of 0.25–20 ppm (Tube 121SD; Sensidyne), 5–90 ppm (Tube 121SB; Sensidyne), or 0.05–2.0 ppm (Tube 121U; Sensidyne),⁴⁸ and continuous reading monitors (Draeger and Sensidyne).^{48,49} The Occupational Safety and Health Administration (OSHA) reference method employs a calibrated sampling pump and a glass tube with beaded carbon impregnated with potassium hydroxide (KOH). The KOH degrades the PH₃ to oxidized phosphorous that can be conveniently analyzed later.⁵⁰

Regardless of the specific technique employed, air monitoring in the workplace is often the best means of confirming exposure to phosphine. In industrial operations that utilize phosphine and similar toxic gases (e.g., in the semiconductor industry), continuous environmental monitoring is a recommended strategy.⁵¹

In agricultural workplaces, however, air monitoring may often be neglected in responding to a fire or other emergency associated with the use of a phosphine-generating fumigant. In the 337 cases reported to the California illness registry between 1982 and 2008, for example, phosphine measurements of workplace air were reported in only eight episodes (Table 3: 1983-2159, 1984-2567, 1987-2924, 1993-742, 1994-1047, 41-TUL-96 [1996-1755], 4-STA-97 [1996-1872], 1998-878, 1999-662).

There were no air measurements reported for episodes involving aluminum phosphide fires. The presence of phosphine in these episodes can be inferred because of phosphine-compatible illnesses in bystanders or emergency responders. However, because the phosphine toxidrome is nonspecific, with headache, nausea, and dyspnea among the most frequently reported symptoms, it is not always possible to distinguish individuals with serious exposures from those with anxiety or unrelated medical conditions.

Respiratory Protection

NIOSH does not recommend use of respirators as a primary means of protecting workers. Work practices and air sampling should be used to ensure that exposures remain below the 0.3 ppm exposure limit. No specific gas/

vapor cartridge respirator is recommended in the NIOSH pocket guide section dealing with phosphine.⁵² Any supplied-air respirator is acceptable for exposures below 3 ppm. For higher exposures, the NIOSH guide recommends the following:

- 3–7.5 ppm: The supplied air respirator should be operated in the continuous-flow mode.
- 7.5–15 ppm: Any supplied-air respirator operated in a continuous-flow mode, or any air-purifying, full-facepiece respirator (gas mask) with a chin-style, front- or back-mounted canister providing protection against the compound of concern (acid-gas) or any self-contained breathing apparatus with a full facepiece or any supplied-air respirator with a full facepiece.
- 15–50 ppm: Any supplied-air respirator operated in a pressure-demand or other positive-pressure mode, including emergency or planned entry into unknown concentrations or IDLH conditions:
 - Any self-contained breathing apparatus (SCBA) that has a full facepiece and is operated in a pressure-demand or other positive-pressure mode.
 - Any supplied-air respirator that has a full facepiece and is operated in a pressure-demand or other positive-pressure mode in combination with an auxiliary self-contained positive-pressure breathing apparatus.

Registered Pesticide Products, Use and Illness Data

Aluminum Phosphide

California registers 20 products that contain 55% to 78% aluminum phosphide, all restricted use materials.⁵³ⁱ There were 107,764 pounds of aluminum phosphide use reported in California in 2009. Comparable levels were used in the years between 2000 and 2009. In 2009, slightly more than half (or approximately one third) of aluminum phosphide was used for

rodent control, and the remainder for commodity fumigation. The most frequently fumigated commodities included dried fruits, pistachios, almonds, walnuts, rice, and wheat.⁵⁴ Use data for aluminum phosphide and other phosphine-generating fumigants are summarized in Table 2. The most recently available national data, published in the 1998 aluminum phosphide and magnesium phosphide re-registration eligibility document, indicated 1.6 million pounds are used annually in the United States. The major uses included stored wheat, peanuts, and corn.⁴³

There were 310 cases of aluminum phosphide-associated illness reported to the California Pesticide Illness Surveillance Program (PISP) between 1982 and 2009. These included 235 cases that were “possibly,” “probably,” or “definitely” related to application of aluminum phosphide; 75 nonrelated cases evaluated as “unlikely,” “unrelated,” “exposed without symptoms,” and cases with incomplete information. Exposures occurred during rodent control applications, commodity fumigations in enclosed spaces, accidental exposures to commodities fumigated in transit, and cases of deliberate ingestion or deliberate inhalation.

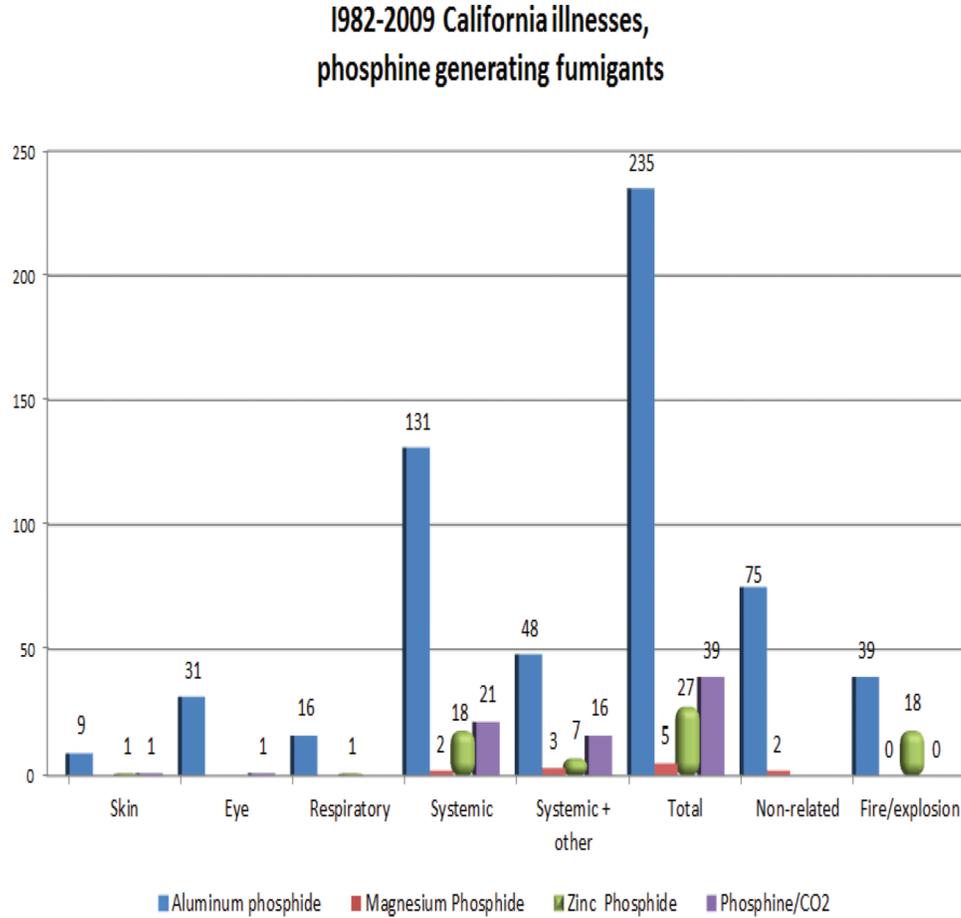
Illness Patterns

Cases with only systemic symptoms accounted for more than half of all cases. Respiratory symptoms were present in a total of 57 cases; 41 in cases involving mixed systemic and respiratory symptoms; and 16 with no accompanying systemic illness (see Figure 1 and accompanying note). Isolated cases of skin symptoms were reported in nine sporadic cases, all involving application workers. There were 31 isolated cases of eye symptoms, including 6 sporadic cases, and 25 associated with two separate clusters in the same nut processing operation.

California Safety Issues

There were 12 episodes involving aluminum phosphide fires or explosions, accounting for 39 total cases. The most recent recorded episode was in 2006 (2006-613 [45-SBD-06]; Table 3). Many occurred because of problems arising from the disposal of unspent

Figure 1. California cases related to phosphine or phosphide fumigants, 1982–2009. *Respiratory* category includes isolated respiratory cases, and cases with respiratory symptoms + eye or skin involvement. *Systemic + other* category includes cases with systemic symptoms + respiratory, eye, or skin symptoms (color figure available online).



residue (1983- 2253, 1983-2653, 1990-2429 [81-BUT-90], 1990-3004 [17-SJ-90], 1991-330, 1993-1513 [24-SD-93], 1994-1468, 1996-1906, 1998-96, 2006-613 [45-SBD-06]), but fires also occurred during a rodent control treatment (1986-861 [34-TEH-86]), and a commodity fumigation (1990-842 [27-MAD-90]). One episode involved stored zinc phosphide accidentally contaminated with water (1990-534, [17-SJ-90]). The remaining cases involved aluminum phosphide. Details of individual episodes are given in Table 3.

An episode reported in a newspaper, but not reported to the California surveillance program, involved a 2007 raisin warehouse fire in Madera. No illnesses were described, but the fire was

reported to result in \$2.5 million in destroyed commodity.⁵⁵

Aluminum Phosphide Safety Issues Outside California

Burgess et al.⁵⁶ briefly described an episode involving failure to properly dispose of aluminum phosphide in Washington. In 2005, Sudakin⁵⁷ reported on a manufacturing operation with a history of explosions in hoppers used for formulating phosphide fumigants. The phosphine overexposure described in the article, however, was apparently related to poor workplace hygiene and ventilation rather than a fire or explosion.⁵⁷

Episodes reported in the press that were not reported to public health surveillance programs included a 2001 grain silo fire in Oxfordshire, England,⁵⁸ and a 2002 fire in Tybee, Georgia, caused by aluminum phosphide tablets inappropriately discarded in a trash can.⁵⁹

Zinc Phosphide

Other phosphine-generating pesticides include zinc phosphide (Zn_3P_2 ; ZP Rodent Bait, Dexol Gopher Killer, etc.), used as a bait for rodent control. Zn_3P_2 is more chemically stable than AIP and only generates PH_3 on contact with stomach acid or a large volume of water.⁶⁰

There are currently 42 products registered in California containing 2% zinc phosphide. However, there are several concentrated formulations with 63% or 80% zinc phosphide. Some 2% formulations are allowed for use outdoors near residential structures, either by homeowners or professional applicators, according to the California registration database.⁶¹

California pesticide use data for 2009 showed 20,893 pounds of zinc phosphide use reported for 2009, almost exclusively for rodent control (Table 2).⁵⁴ The EPA registration document for zinc phosphide, published in 1998, indicates that most national use is agricultural, with 40% on farm structures, 10% on rangeland, 10% on landscape (golf courses and other turf), 10% on grain crops (wheat, barley, and oats), and 10% on sugar beets. No specific estimate of national use was made.⁶²

Between 1982 and 2009 there were 34 cases associated with zinc phosphide reported to the California Pesticide Illness Program, with 27 cases classified as related to the reported exposures. These included 18 illnesses associated with a 1990 San Joaquin episode resulting from a leaking pipe that dripped water onto a container of zinc phosphide (17-SJ-90; Table 3). The nine additional related cases included three intentional ingestions (45-SD-01, 21-LA-07, 14-Ven-08; Table 3) and five cases associated with mishandling of zinc phosphide during transport, storage, or application. The remaining case involved potential exposure to pyrethrins, a synthetic pyrethroid, and zinc phosphide.

Magnesium Phosphide

Magnesium phosphide (Mg_3P_2 ; Magtoxin) is less stable than aluminum phosphide. It is used for commodity applications when short fumigation times are required and very occasionally for rodent control.⁶⁰ There are five formulations registered currently in California, ranging in concentration from 56% to 95% active ingredient.⁶³ California data showed 7957 pounds of magnesium phosphide use reported for 2009. Commodities treated with magnesium phosphide were principally dried fruit, almonds, walnuts, rice, and grains.⁵⁴ National use estimates were not included in the EPA re-registration eligibility document.⁴³

Between 1982 and 2008 there were seven cases associated with exposure to magnesium phosphide reported to the California Pesticide Illness Surveillance Program. One case was asymptomatic, and a second involved a skin reaction evaluated as unrelated to the reported exposure. The five related cases involved systemic symptoms, or combined systemic and respiratory symptoms, following phosphine gas inhalation by employees working near indoor fumigation operations or near incompletely aerated commodities. These included two packing/processing workers, an electrician working near a fumigated silo, a dock worker exposed to a previously fumigated shipping container, and a carpenter exposed to phosphine while constructing a treatment chamber put into premature use.

No fires or explosions associated with the use magnesium phosphide were reported to the California illness registry. However, a newspaper report from 1984 described a Sacramento almond warehouse fire that occurred during a magnesium phosphide fumigation. It was not associated with any reported illnesses.⁶⁴

Press reports of episodes outside of California included a 1986 fire in Richmond, Virginia, resulting in 10 reported illnesses associated with improper disposal of magnesium phosphide.⁶⁵ A 2005 Melbourne, Australia fire was associated with magnesium phosphide stored in drums at a chemical factory.⁶⁶

Mixtures of Phosphine Gas and Carbon Dioxide

There are two formulations of phosphine mixed with 98% carbon dioxide (CO₂) that have been registered in California since 2001. The formulation is designed to address safety issues associated with the use of phosphides. A formulation with 100% phosphine is also available and designed to be used with a CO₂ dispensing tank to deliver an end-use concentration of 2% phosphine.⁶⁷ According to the California data, 30,203 lbs were reported used in 2009, with approximately 50% listed as “regulatory pest control.”ⁱⁱ The most frequently treated commodities included almonds, pistachios, walnuts, and dried fruit.⁸

Forty-one cases associated with the new phosphine formulations were reported between 2004 and 2008, with two outbreaks associated with accidental exposures to warehouse workers (45-FRE-04, 71-KER-07; Table 3) accounting for all but three cases.

Twenty-two cases involved isolated systemic symptoms. There were 13 respiratory illnesses, all but one involving simultaneous systemic symptoms.

Safety Issues

No fires have been reported in association with use of the 98% CO₂, 2% phosphine formulation. Fires related to the nonagricultural storage or transport of phosphine gas were reported in a Nashville, Tennessee, chemical factory in 2000,⁶⁸ in an electronics operation in 2001 in Allentown, Pennsylvania,⁶⁹ and in a 2005 truck fire in Brisbane, Australia.⁷⁰ Episodes reported in 2011 included an explosion in a Guanxi, China, pesticide factory,⁷¹ and a boatyard fire in Plymouth, England.

METHODS

The cases were reported on the day of exposure to both the California Poison Control System (CPCS) and the Kern County Department of Agriculture–County Agricultural Commissioner’s Office (Kern CAC). CPCS provided medical management advice to the

three local treating hospitals (although all of the patients were scheduled for discharge from the emergency room, CPCS suggested overnight observation because of the risk of delayed pulmonary symptoms).

Kern CAC interviewed affected workers at local hospitals and conducted a worksite visit to assess violations of proper aluminum phosphide handling procedures. A worksite evaluation was also conducted at the Kern County pistachio processing operation by California Department of Regulation’s (CDPR) staff on January 12, 2010. Information regarding the plant operation was obtained from the sanitation/fumigation supervisor and by direct observation. The plant Fumigation Management Plan (FMP) and the plant copy of the *Applicator’s Manual for Fumitoxin*[®] (AMF) were reviewed.

Information on the illnesses experienced by affected employees were obtained from interviews with them by the staff members of the Worker Health and Safety (WHS) branch and staff members of the Kern Department of Agriculture. Likewise, medical records from the three hospitals that treated the employees were reviewed. The hospital record review was conducted in conjunction with the California Department of Public Health, Occupational Health Branch.

RESULTS

Fumigation of stacked bags of pistachios on December 8, 2009, took place on the sidewalk outside of a warehouse building. On Figure 2a, a photo taken during CDPR visit to the plant on January 12, 2010, shows bulk bags of pistachios under black plastic film, ground-sealed with sand snakes. The pile under fumigation on December 10, 2009, was 4 bags wide and 20 bags long, for a total of 80 bags under fumigation. Each bulk bag weighed 2200 pounds (997 kg) and measured 4 ft × 4 ft × 5.5 ft in dimension (88 ft³ or 2.5 m³). The total volume under fumigation was 200 m³ or 2 × 10⁵ L.

The bags were elevated off the ground by wooden pallets (Figure 2b), which provided a space beneath for trays of fumigant pellets. Each

Figure 2. (a) Pistachio bags covered by black film; (b) wooden pallets under the stacks of pistachio bags; (c) downspout from an adjacent warehouse; (d) bottom of burned pistachio sacks (color figure available online).



tray was loaded with 1 lb (453 g, or approximately 750 pellets) of aluminum phosphide. This corresponds to about 213 pellets per 1000 ft³, which is within the listed “Dosage Range” of the AMF (150 to 450 pellets per 1000 ft³).

During the fumigation, a rainstorm arrived in the area of the plant. The Belridge-Lost Hills weather station, 15 miles west and slightly

south of the plant, recorded 0.46 inches of rain between 4:00 PM and 10:00 PM on December 10, 2009. The fumigation was located next to the downspout from the roof of an adjacent warehouse (Figure 2c). The soil and angle iron shown in the photo had apparently been placed there to divert water from the downspout away from the fumigation pile. On December 10, however, the increased

volume of rainwater allowed pooling around the fumigation pile and for water to seep under the tarp. This seepage was directly in line with the trays of aluminum phosphide under the end pallet. Photos of the burned stacks taken during the January 12, 2010, visit showed burns on the bottoms of the pistachio sacks (Figure 2d) as well as on the cardboard on which the lowest sacks of pistachios rested.

At approximately 4:00 PM, the stack of pistachios under fumigation caught fire. Workers near the pile noted smoke. Although untrained in the proper response, they attempted to douse the fire with water.

The Fumitoxin label states: "*Classified by UL, Inc. as to fire hazard only when used specifically as directed in the instructions on this container, and supplemental labeling. FUMITOXIN® is noncombustible, but exposure to moist air or water releases flammable and toxic phosphine gas. Spontaneous ignition may result if contacted by water, acids or chemicals.*"

When the workers realized that water did not put out the fire, some used chemical fire extinguishers. Others cut up the tarp to direct the extinguisher directly on the flames. Workers in the vicinity were likely exposed, not only to phosphine but also to phosphorus oxides (PO_x), and combustion products from the burning films, bags, and cardboards. Exposure to extinguisher agent was also likely. Workers may also have come in contact with both unreacted aluminum phosphide and spent materials (primarily aluminum hydroxides).

Medical Findings

The Kern County Department of Agriculture investigation indicated that 10 employees of a pistachio processing plant had medical evaluations following the December 10, 2009, accident; 9 were available for interview by Worker Health and Safety (WHS) staff on Tuesday January 12, 2010. The remaining employee left employment with the plant before the WHS visit and could not be reached. Information on this employee was available from the initial interview by Kern County Department of Agriculture staff. Medical records on two cases were available from

information collected by Kern Department of Agriculture staff. Medical records for seven additional cases were made available to the Department of Pesticide Regulation WHS staff by the California Department of Public Health, Occupational Health Branch.

The extent of medical evaluation varied according to the judgment of individual treating providers. One Bakersfield hospital performed bronchoscopy on all three of the patients seen there, and found abnormalities in each. The seven patients examined at two other hospitals did not have bronchoscopies.ⁱⁱⁱ There were also minor variations in the use of radiography, pulse oximetry, and the supportive treatment administered to individual patients.

The case summaries below integrate information from emergency room and hospital admission records, interviews conducted by Kern County Department of Agriculture staff on December 10 and December 11, 2009, and interviews by WHS staff on January 12, 2010.

Summary of the Findings for Individual Workers

Case 2010-291, a 25-year-old forklift driver assisted in trying to extinguish the fire. When evaluated in the emergency room (ER) at Hospital A, he reported symptoms of nausea, vomiting, chest tightness, wheezing, and shortness of breath exacerbated by coughing. At a subsequent interview,^{iv} he described additional symptoms including tremor, abdominal pain, and sleepiness. Physical examination showed elevated blood pressure (162/93 mm Hg), rapid heart rate (112 beats per minute), and a rapid respiratory rate (28 cycles per minute). Additional findings included diaphoresis (sweating), pharyngeal erythema (redness in the throat), decreased breath sounds at the bases of the lungs, and mild rhonchi (abnormal breath sounds).

Blood gases showed a normal oxygen (O_2) saturation (97%) and a borderline low level of bicarbonate (22 mEq/L). His ECG demonstrated a variable heart rate (61–96 beats per minute), with occasional paired premature ventricular contractions (PVCs). He had a normal

chest radiograph, but the bronchoscopy performed before discharge showed acute airway inflammation, with no evidence of pulmonary edema.

Case 2010-292, a 24-year-old forklift driver, employed by a labor contractor, had worked 4 to 5 months at the plant. He assisted in putting out the fire. On subsequent interview,^v he mentioned that there was a strong “ugly” odor present at the time. In the ER, he reported that he had breathed fire extinguishing agent as well as fumes from the fire.

In the ER at Hospital B, he complained of headache, sore throat, intractable nausea, vomiting, mild shortness of breath, and body aches. His blood pressure was slightly elevated (141/69 mm Hg) and he had a normal chest radiograph.

He was treated with morphine and ketorolac for pain, as well metoclopramide for nausea. He was admitted for observation because of concern about his exposure to phosphine. He was discharged on December 11, 2009, with no medications, but on subsequent interview, it was noted that he had a headache for 4 days afterwards.

Case 2010-293 provided no information on job title, age, or details of exposure. When Kern County Agricultural Commissioner (CAC) investigators interviewed him at an ER on December 10, 2009, he reported dizziness and nausea. He signed himself out of the ER against medical advice, and was no longer employed at the pistachio processing company when WHS staff came to interview workers on January 12, 2010.

Case 2010-294, a 20-year-old forklift driver, worked at the processing plant 6 months before the fire and was employed by a labor contractor. He saw sparks and smoke coming from the covered tarp, accompanied by a smell that resembled “maiz” (corn), and used the extinguisher to put the fire out. He estimated that his total exposure time was about 15 or 20 minutes.

In the ER at Hospital C, he reported having nausea, neck pain, chest pain, shortness of breath, and irritation of the eyes and upper respiratory tract. He had a slightly elevated temperature (100.2°F), slightly elevated blood pressure (146/91 mm Hg), and conjunctival congestion.

His arterial blood gases showed a borderline elevation of pH (7.43), lower than expected PaCO₂ (25 mm Hg), and an elevated PaO₂ (151 mm Hg, while receiving 1.5 L O₂/min by nasal cannula). His chest radiograph was normal except for hyperinflated lungs. He was observed for 24 hours, and then released from the hospital. At the time of the January 12, 2010, follow-up interview, he reported that he felt some symptoms intermittently since being released from the hospital.

Case 2010-295, a 38-year-old maintenance mechanic, helped remove the tarp from the burning pallets and also used an extinguisher to help put out the fire. In the ER at Hospital B, he reported sore throat, headache, nausea, and vomiting. In a separate interview the same day with Kern CAC staff, he reported additional symptoms including chest pain and eye irritation. CPCS advised extended observation and he was held overnight.

He was discharged on December 11, 2009, but had chest pain the following day that required reevaluation at the Hospital B ER. At that ER visit, he had slightly elevated blood pressure, but otherwise normal vital signs, normal O₂ saturation, normal physical findings, and a clear chest radiograph.

He had a computed tomography scan on December 22, 2009, that showed no evidence of restrictive lung disease or pulmonary fibrosis.^{vi} However, he reported still having mild chest pain and a burning sensation in his chest at the January 12, 2010, interview.

He sought follow-up care for physical and anxiety-related symptoms that caused him to miss work intermittently through December 2010. He had three separate pulmonary function tests (PFTs), on May 14, 2010, October 18, 2010, and November 30, 2010, that did not meet American Thoracic Society standards for acceptability and repeatability. The PFTs showed decreased lung volumes, indicating possible restrictive lung disease.

Case 2010-296, this 20-year-old was the initial responder to the fire. He reported sore throat, a runny nose, chest tightness, and shortness of breath exacerbated by coughing, as well as nausea and vomiting when evaluated in the ER at Hospital A. He had a low-grade

fever (99.5°F), slightly elevated blood pressure (151/83 mm Hg), and was observed to be anxious, pale, and diaphoretic. Examination showed conjunctival and pharyngeal erythema (eye and throat redness), nasal swelling, and rhonchi (abnormal breath sounds) in the lungs. Arterial blood gases were normal on room air. His chest radiograph was normal. His ECG showed some minor abnormalities, probably unrelated to the exposure. On the advice of CPCS, he was observed overnight in the hospital and received supportive treatment for nasal congestion and nausea. Intravenous steroids, inhaled steroids, bronchodilators, and antibiotics were prescribed for his respiratory problems. He had a bronchoscopy on December 11, 2009, that showed marked erythema in the left upper lobe airways, with evidence of tracheobronchitis (airway inflammation).

Case 2010-297, a 34-year-old maintenance worker, initially learned about the fire over a plant radio. He reported that he tried to calm down the other plant workers who were attempting to extinguish the fire, and said that he observed a cloud of “smog” at the scene of the fire. In the ER at Hospital C, he reported burning of the eyes (described as blurry vision in a subsequent interview^{vii}), nausea, and vomiting. Pulse oximetry showed an oxygen saturation of 95%, but he did not have persistent respiratory difficulty.^{viii} A subsequent arterial blood gas, on 2 L/min of oxygen, showed 99% saturation. His chest radiograph was reported as normal. He was admitted to the hospital for observation overnight and discharged on December 11, 2009. He reported subsequently that he felt fine when he got out of the hospital.

Case 2010-298, a 20-year-old maintenance worker and assistant mechanic also learned about the fire over a plant radio. He cut the tarp covering a burning pallet and then used a forklift to move the pallet. In the ER at Hospital C, he reported difficulty breathing, headache, burning eyes, nausea, vomiting, feeling weak, chest congestion, burning lungs, and sore throat. (In a subsequent interview, he also reported experiencing dizziness.)

He had an elevated respiratory rate (22 cycles per minute), conjunctival congestion, and erythema. Examination of the lungs

demonstrated rhonchi (abnormal breath sounds) and wheezing.^{ix} He had a slightly elevated white blood cell count, normal arterial blood gases on room air, and a normal chest radiography. He was admitted for overnight observation and received supportive treatment that included supplemental oxygen and “aerosol treatment.”

Case 2010-299, a 51-year-old maintenance worker who had worked at the plant for 10 years, learned about the fire on the plant radio and decided to help put it out. In the ER at Hospital A, he reported shortness of breath, nausea, vomiting, chest pain and discomfort, and sore throat. (At a subsequent interview,^x he reported that he smelled the gas from fire, but did not have symptoms, and he went to hospital for evaluation only). He had a slightly elevated blood pressure (153/98 mm Hg), and a slightly rapid respiratory rate (22 cycles per minute). Examination revealed diaphoresis (sweating), pharyngeal erythema (redness from apparent irritation of the throat), decreased breath sounds at the lung bases, and rhonchi (abnormal breath sounds). The white blood cell count was slightly elevated and the arterial blood gases showed slightly low PaO₂ (76 mm Hg) and an O₂ saturation of 96%. Minor abnormalities of the ECG were identified, but it was unclear whether or not these were related to his exposure. His chest radiograph was initially interpreted as showing infiltrates by the ER staff, but was reported as normal by radiology.

The ER staff felt his symptoms may have been related to a preexisting upper respiratory infection, but nonetheless admitted him for observation, after receiving information about his exposure from CPCS. He was treated for “inhalation injury” with systemic steroids, and medications for nausea and pain. He had a bronchoscopy before discharge that showed tracheobronchitis with marked erythema and easy friability. A biopsy done during bronchoscopy showed benign pulmonary tissue with occasional anthracotic pigment deposits with no evidence of granuloma or malignancy.

Case 2010-300, a 28-year-old supervisor, with 7 years of experience at the plant, used an extinguisher to try to put out the fire. In the ER at Hospital B, he complained of nausea, headache, vomiting, and sore throat. (He complained of

dryness in the chest and throat to Kern CAC interviewers. He also reported blurry vision in the follow-up interview.^{xi)} Upon arrival at the hospital, his O₂ saturation was 94%, despite being on supplemental oxygen. His blood pressure was slightly elevated (141/92 mm Hg), as was his white blood cell count. His chest radiograph in the ER was clear. Although he appeared stable clinically, he was admitted for observation because of concerns about his exposure raised by CPCS staff.

Although a repeat chest radiograph a few hours after admission showed a right lower lobe infiltrate, his O₂ saturation rose to 100%. He was discharged on December 11, 2009. He was reevaluated in the Mercy ER on December 12, 2009, because of chest pain, but his O₂ saturation remained at 100% and a chest radiograph was clear.

On January 12, 2010, he reported that he still intermittently experienced burning in the chest and anxiety. He was subsequently seen in an occupational clinic on February 6, 2010, for headaches, visual changes, and paresthesias,^{xiii} all new since the incident. He was evaluated for a possible acute cerebrovascular accident (stroke), which was ruled out on a computed tomography scan. He was reevaluated February 7, 2010, in the ER and treated for a vascular (migraine) headache. CPCS was contacted and felt that his symptoms were not related to his prior exposure.

DISCUSSION

The medical findings were consistent with exposure to phosphine gas, although it is possible some symptoms were attributable to other factors, including possible exposure to fire extinguisher chemicals or combustion by-products. Six of the 10 workers had mild respiratory compromise, indicated by chest pain or shortness of breath, an elevated respiratory rate, or oxygen saturation below 96% (case numbers: 2010-291, 2010-296, 2010-297, 2010-298, 2010-299, 2010-300). Two additional cases had short-term respiratory symptoms (case numbers: 2010-292, 2010-294). None had oxygen saturation below 90%, the recommended threshold

for use of supplemental oxygen in treatment of acute asthma.⁷²

No confirmed cases of pulmonary edema were identified, but one worker had transient infiltrate (an indication of possible pneumonia or fluid on the lung) on a chest radiograph. Bronchoscopy carried out in three cases seen at Hospital A showed airway inflammation. Three workers (case numbers: 2009-294, 2009-295, 2009-300) reported persistent symptoms that they associated with the December 10, 2009, fire, more than a month after the exposure.

Medical Management of Exposure to Phosphine From Combustion Accidents

Standard medical management of inhalation exposures to phosphine includes an immediate evaluation by any trained health care provider to support or restore effective oxygenation, ventilation, and circulation,⁶⁵ and overnight hospital observation to evaluate possible delayed pulmonary effects. Because few cases of delayed pulmonary edema have been reported in California following typical occupational exposures, this recommendation might seem excessive. Hospital records related to the December 2009 Kern County episode demonstrated evidence of oxygen desaturation in some workers; transient pulmonary infiltrates and significant airway inflammation in the three workers who had bronchoscopy. In the absence of information from on-site air monitoring at the time of exposure, the existing recommendations for hospital observation appear justified.

“Spontaneous ignition” of expired phosphine gas is probably a rare occurrence, but possible in aluminum phosphide ingestion cases.⁹ Exposure to phosphine at possibly toxic levels during hospitalization or at postmortem (see Table 3, 47-RIV-03, 2003-1075) is much more likely.^{73,74} Health care providers and staff performing postmortem examinations should be protected by air monitoring and provision of appropriate respiratory protection if needed.⁹

Prevention of Phosphine-Related Illnesses

The fire and subsequent worker exposures that occurred in this case were entirely

Figure 3. (a) Sample warning poster for aluminum phosphide; (b) fumigation warning poster (color figure available online).

B

DANGER FUMIGATION
¡PELIGRO FUMIGACIÓN!



DO NOT ENTER
NO ENTRE

Fumigant: Aluminum Phosphide
Fumigant: Fosforo de Aluminio

Date/Time of Fumigation: December X, 20XX, 8:00 a.m.
Fecha/Hora de Fumigación: Diciembre X, 20XX, 8:00 a.m.

Permittee-Operator/Permisionario-Operador: Juan de la Cruz
123 Main Street, Merced, CA 95348
Tel: (209) 123-4567

A

IN CASE OF FIRE **EN CASO DE INCENDIO**






DO NOT use water



NO USE agua



Phosphine gas can react violently to air and water



Puede reaccionar con agua o aire con violencia



Aluminum Phosphide produces **toxic gas**



Fosforo de Aluminio puede producir un **gas tóxico**-Fosfina



ALERT SUPERVISOR



Pída ayuda de su supervisor

preventable. Although the safety hazards of phosphine-generating fumigants have been known for a considerable time period, several potential problems were ignored in the lead-up to the fire that occurred on December 10, 2009:

1. The outdoor location of the fumigation stack was adjacent to a building downspout.
2. Once the fire began, initial responders included untrained plant personnel who attempted to put out the fire with H₂O-based extinguishers and temporarily made the fire worse.
3. There were no warning signs posted. Title 3 CCR 6782(c) requires posting for fumigations in enclosed spaces visibly posted 24 hours before the actual application. Bilingual warning signs are required.⁷⁵ Although not technically required for commodities fumigated outdoors, warning signs would be a helpful adjunct to increased training. A suggested sign with the simple message stating that phosphine fumigants react adversely with water is shown in Figure 3a. Figure 3b is a sample fumigation “DANGER-NO ENTRY” poster.

It is likely that no workers would have become ill if they had refrained from trying to extinguish the fire and simply called for assistance. Workers developed short-term respiratory distress that required emergency evaluation. Most received only supportive care and had uncomplicated courses of hospital care, although bronchoscopies performed on three workers showed significant airway inflammation.

Other preventive measures that could have been exercised to prevent the likelihood of such incident include

1. Fumigation should only be conducted by personnel trained in the proper, label-directed application methods of aluminum phosphide that meet regulatory requirements.
2. All workers involved in the processing of produce should be adequately trained

how to handle emergencies involving the fumigation process.

3. Fumigation of produce should be conducted in a manner that allows protection from sudden changes in weather and environmental elements.
4. All employees shall be notified of an upcoming application, ahead of schedule.

Alternative control methods that could be used prior to the drying stage of the processing operations include:⁷⁶

1. Use of the 98% CO₂, 2% phosphine formulation. This is also a promising means of preventing fires associated with the use of phosphide fumigants. Nevertheless, precaution is necessary in the use of the new formulation illustrated by the 41 cases associated with its use in California since 2004 (see discussion above).
2. Use of sealed chambers. Filling the chamber with nitrogen or carbon dioxide to deplete oxygen.
3. Storing commodities in refrigerated storage, but this may not be economically feasible for nuts and other dried commodities.
4. Use of less toxic chemical such as hydroprene, methoprene, pyriproxyfen, pyrethrin, and synthetic pyrethroid insecticides, for application with industrial fogging equipment.
5. Treating appropriate warehouse spaces with the relatively volatile organophosphate compound, DDVP. The 7.8% formulation used for this purpose is labeled as a Category I pesticide, much more toxic than foggers containing insect growth regulators and pyrethrin/pyrethroid insecticides.

NOTES

- i. Only licensed applicators may apply them. Use reporting is required.
- ii. Regulatory pest control would include use by county departments of agriculture for rodent control and other uses.

- iii. Hospital A, Bakersfield Heart Hospital; Hospital B, Mercy Hospital in Bakersfield; Hospital C, Delano Regional Medical Center.
- iv. WHS interview January 12, 2010.
- v. WHS interview January 12, 2010.
- vi. A noncalcified left upper lung granule, was noted, but no findings likely related to the exposure. Specifically, there was no computed tomographic (CT) evidence of interstitial lung disease.
- vii. WHS interview January 12, 2010.
- viii. While in triage, O₂ saturation ranged from O₂ sat 95% to 97%. Respiration was listed as 16–17 cpm; 2–4 L/min of oxygen was given. Chest was nontender. Breath sounds and chest x-ray were normal.
- ix. The examination in the Medical Unit noted “few rhonchi and no rales.” He was given an “aerosol treatment because of an obvious wheezing.”
- x. WHS interview January 12, 2010.
- xi. WHS interview January 12, 2010.
- xii. Patient complained of tingling in both arms lasting for about 10 minutes, occurring twice weekly. On examination, there were decreases in his upper arm reflexes and slight decrease of sensation from the right shoulder to the right fingertips.

REFERENCES

1. O'Malley M. *Illnesses and Injuries Associated with Exposures to Phosphine and Phosphine Decomposition Products*. Sacramento, CA: California Environmental Protection Agency Department of Pesticide Regulation Worker Health and Safety Branch; 1998.
2. O'Malley M, Kullman G, Cox-Ganser J. *NIOSH Alert—Preventing Phosphine Poisoning and Explosions During Fumigation*. Cincinnati, OH: NIOSH; 1999.
3. Cotton F, Wilkinson G. The Group V Elements: P, As, Sb, Bi. In: *Advanced Inorganic Chemistry*. 3rd ed. New York: Interscience Publishers; 1972:367–402.
4. National Library of Medicine. *Hazard Substances Database (HSDB)*. Washington, DC: National Library of Medicine; 2011.
5. Smeets M, Bulsing PJ, Van Rooden S, et al. Odor and irritation thresholds for ammonia: a comparison between static and dynamic olfactometry. *Chem Senses*. 2007;32:11–20.
6. Agency for Toxic Substances and Disease Registry (ATSDR). *Medical Management Guidelines for Arsine*. Atlanta, GA: ATSDR; 2011.
7. Hsu C, Han B, Liu M, et al. Phosphine-induced oxidative damage in rats: attenuation by melatonin. *Free Radic Biol Med*. 2000;28:636–642.
8. Proudfoot AT. Aluminium and zinc phosphide poisoning. *Clin Toxicol (Phila)*. 2009;47:89–100.
9. Gurjar M, Baronia AK, Azim A, Sharma K. Managing aluminum phosphide poisonings. *J Emerg Trauma Shock*. 2011;4:378–384.
10. Arora B, Punia RS, Kalra R, et al. Histopathological changes in aluminium phosphide poisoning. *J Indian Med Assoc*. 1995;93:380–381.
11. Shah V, Baxi S, Vyas T. Severe myocardial depression in a patient with aluminium phosphide poisoning: a clinical, electrocardiographical and histopathological correlation. *Indian J Crit Care Med*. 2009;13:41–43.
12. Khasigian P. Phosphine and phosphides. In: Olson KR, ed. *Poisoning and Drug Overdose*. 6th ed. New York: McGraw-Hill Medical; 2012:331–332.
13. Centers for Disease Control and Prevention (CDC). Deaths associated with exposure to fumigants in railroad cars—United States. *MMWR Morb Mortal Wkly Rep*. 1994;43:489–491.
14. Reigart J, Roberts J. Fumigants. In: *Recognition and Management of Pesticide Poisoning*. 5th ed. Washington, DC: United States Environmental Protection Agency; 1999:160.
15. Fisher E. Fire marshal: gas leak not serious. *Bucks County Courier Times*. Levittown, PA; December 31, 2003:1C.
16. Staff writers. Mill fire exposes workers to gas. *St. Petersburg Times*. St. Petersburg, FL; January 1, 1990:3;3.
17. Ames RG. Multiple-episode conjunctivitis outbreak among workers at a nut-processing facility. *J Occup Med*. 1991;33:505–509.
18. Shadnia S, Mehrpour O, Abdollahi M. Unintentional poisoning by phosphine released from aluminum phosphide. *Hum Exp Toxicol*. 2008;27:87–89.
19. Wilson R, Lovejoy FH, Jaeger RJ, Landrigan PL. Acute phosphine poisoning aboard a grain freighter. Epidemiologic, clinical, and pathological findings. *JAMA*. 1980;244:148–150.
20. Lemoine TJ, Schoolman K, Jackman G, Vernon DD. Unintentional fatal phosphine gas poisoning of a family. *Pediatr Emerg Care*. 2011;27:869–871.
21. Gualé FG, Stair EL, Johnson BW, et al. Laboratory diagnosis of zinc phosphide poisoning. *Vet Hum Toxicol*. 1994;36:517–519.
22. Gupta MS, Malik A, Sharma VK. Cardiovascular manifestations in aluminium phosphide poisoning with special reference to echocardiographic changes. *J Assoc Physicians India*. 1995;43:773–774, 779–780.
23. Gupta S, Ahlawat SK. Aluminum phosphide poisoning—a review. *J Toxicol Clin Toxicol*. 1995;33:19–24.
24. Chen T, Shi R, Yang XZ, et al. [Clinical analysis of seven acute phosphine poisoning]. *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi*. 2005;23:223–225.
25. Chugh S, Singhat HR, Girdhar NI, et al. Aluminum phosphide poisoning: analysis of 228 cases. *J Assoc Physicians India*. 1989;37:28–29.

26. Chugh SN, Aggarwal HK, Mahajan SK. Zinc phosphide intoxication symptoms: analysis of 20 cases. *Int J Clin Pharmacol Ther.* 1998;36:406–407.
27. Chugh SN, Arora V, Sharma A, Chugh K. Free radical scavengers and lipid peroxidation in acute aluminium phosphide poisoning. *Indian J Med Res.* 1996;104:190–193.
28. Chugh SN, Pal R, Singh V, Seth S. Serial blood phosphine levels in acute aluminium phosphide poisoning. *J Assoc Physicians India.* 1996;44:184–185.
29. Chugh SN, Ram S, Chugh K, Malhotra KC. Spot diagnosis of aluminium phosphide ingestion: an application of a simple test. *J Assoc Physicians India.* 1989;37:219–220.
30. Chugh SN, Ram S, Mehta LK, et al. Adult respiratory distress syndrome following aluminium phosphide ingestion. report of 4 cases. *J Assoc Physicians India.* 1989;37:271–272.
31. Chugh SN, Ram S, Sharma A, et al. Adrenocortical involvement in aluminium phosphide poisoning. *Indian J Med Res.* 1989;90:289–294.
32. Mehrpour O, Alfred S, Shadnia S, et al. Hyperglycemia in acute aluminium phosphide poisoning as a potential prognostic factor. *Hum Exp Toxicol.* 2008;27:591–595.
33. Mehrpour O, Keyler D, Shadnia S. Comment on aluminum and zinc phosphide poisoning. *Clin Toxicol (Phila).* 2009;47:838–839; author reply 839.
34. Misra UK, Tripathi AK, Pandey R, Bhargwa B. Acute phosphine poisoning following ingestion of aluminium phosphide. *Hum Toxicol.* 1988;7:343–345.
35. Garry VF, Good PF, Manivel JC, Perl DP. Investigation of a fatality from nonoccupational aluminum phosphide exposure: measurement of aluminum in tissue and body fluids as a marker of exposure. *J Lab Clin Med.* 1993;122:739–747.
36. Bhadkambekar CA, Swain KK, Mukherjee T, et al. Zinc as a marker in viscera of suspected metal phosphide poisoning: a study by neutron activation analysis. *J Anal Toxicol.* 2008;32:760–762.
37. Chan LT, Crowley RJ, Delliou D, Geyer R. Phosphine analysis in post-mortem specimens following ingestion of aluminium phosphide. *J Anal Toxicol.* 1983;7:165–167.
38. Wahab A, Rabbani MU, Wahab S, Khan RA. Spontaneous self-ignition in a case of acute aluminium phosphide poisoning. *Am J Emerg Med.* 2009;27:752.e5–752.e6.
39. Shadnia S, Soltaninejad K. Spontaneous ignition due to intentional acute aluminum phosphide poisoning. *J Emerg Med.* 2011;40:179–181.
40. American Conference of Governmental Industrial Hygienists. *Documentation of the Threshold Limit Values and Biological Exposure Indices.* 5th ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists; 1986.
41. American Conference of Governmental Industrial Hygienists. *Documentation of the Threshold Limit Values and Biological Exposure Indices.* 7th ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists; 2001.
42. Jones AT, Jones RC, Longley EO. Environmental and clinical aspects of bulk wheat fumigation with aluminum phosphide. *Am Ind Hyg Assoc J.* 1964;25:375–379.
43. United States Environmental Protection Agency. *Prevention, Pesticides and Toxic Substances (7508C). Re-registration Eligibility Decision (RED) Aluminum & Magnesium Phosphide.* EPA 738-R-98-017. Washington, DC: United States Environmental Protection Agency; 1998.
44. Silberman S, Dribben M. 1,500 Evacuees return home no further threat seen at NJ chemical plant. *The Record.* Hackensack, NJ; March 20, 1988:a03.
45. Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health. *Documentation for Immediately Dangerous to Life or Health Concentrations (IDLHs), Phosphine.* 2011. Available at: <http://www.cdc.gov/niosh/idlh/7803512.html>. Accessed December 11, 2012.
46. Beliles R. Phosphine. In: Patty F, ed. *Industrial Hygiene and Toxicology.* 3rd ed. New York: Interscience Publishers; 1981.
47. Henderson Y, Haggard H. *Noxious Gases.* New York: Reinhold; 1943.
48. Sensidyne Industrial Hygiene & Safety Instrumentation. Colorimetric tubes for phosphine. 2011. Available at: http://www.sensidyne.com/colorimetric-gas-detector-tubes/detector-tubes/sensidyne_gas_detector_tube_number-121SC_PHOSPHINE20-1400ppm.php. Accessed December 11, 2012.
49. Thomas News Net. Phosphine-Monitor-Provides-Maintenance-Free-Operation. 2003. Available at: <http://news.thomasnet.com/fullstory/Phosphine-Monitor-provides-maintenance-free-operation-27217>. Accessed December 11, 2012.
50. Occupational Safety and Health Administration. Phosphine in work place atmospheres. 2011. Available at: <http://www.osha.gov/dts/sltc/methods/inorganic/id180/id180.html>. Accessed December 11, 2012.
51. Fluer L, Urmson J, Yakubek P. Continuous monitoring to detect episodic releases of toxic gases, in hazard assessment and control technology. In: American Conference of Governmental Industrial Hygienists, ed. *Semiconductor Manufacturing.* Chelsea, MI: CRC Press; 1989:135–148.
52. National Institute for Occupational Safety and Health. *Pocket Guide to Chemical Hazards: Phosphine.* 2011. Available at: <http://www.cdc.gov/niosh/npg/npgd0505.html>. Accessed December 11, 2012.
53. California Department of Pesticide Regulation. *Pesticide Label Database, Currently Registered Aluminum Phosphide Formulations.* 2011. Available at: http://apps.cdpr.ca.gov/cgi-bin/label/labq.pl?p_chem=484&activeonly=on. Accessed December 11, 2012.

54. California Department of Pesticide Regulation. *Summary of Pesticide Use Report (PUR) data, Indexed by Chemical*. Sacramento, CA: Department of Pesticide Regulation, California Environmental Protection Agency; 2009.
55. Becker D. Growers lose \$2.5M in fire. *The Madera Tribune*. Madera, CA; April 24, 2007.
56. Burgess JL, Morrissey B, Keifer MC, Robertson WO. Fumigant-related illnesses: Washington State's five-year experience. *J Toxicol Clin Toxicol*. 2000;38:7-14.
57. Sudakin DL. Occupational exposure to aluminium phosphide and phosphine gas? A suspected case report and review of the literature. *Hum Exp Toxicol*. 2005;24:27-33.
58. Hanson N. Media has stoked up chemical blaze story. *Oxfordshire County Publications*. Oxfordshire, UK; June 18, 2001.
59. Rossiter E. Chemical causes small explosion At Tybee. *Savannah Morning News*. Savannah, GA; March 23, 2002:B03.
60. Tomlin C. Phosphine, insecticide, rodenticide. In: *The Pesticide Manual*. 14th ed. Alton, Hampshire: British Crop Protection Council; 2006:831-834.
61. California Department of Pesticide Regulation. Pesticide Label Database, Currently Registered Zinc Phosphide Formulations. 2011. Available at: http://apps.cdpr.ca.gov/cgi-bin/mon/bycode.pl?p_chemcode=626. Accessed December 11, 2012.
62. United States Environmental Protection Agency. *Prevention, Pesticides and Toxic Substances (7508W). Re-registration Eligibility Decision (RED) Zinc Phosphide*. Washington, DC: United States Environmental Protection Agency; 1998.
63. California Department of Pesticide Regulation. Pesticide Label Database, Currently Registered Magnesium Phosphide Formulations. 2011. Available at: http://apps.cdpr.ca.gov/cgi-bin/mon/bycode.pl?p_chemcode=2085. Accessed December 11, 2012.
64. Bell T. Fast action, luck saves downtown from deadly gas. *Sacramento Bee*. Sacramento, CA; December 15, 1984:B01.
65. Witt J. Explosion in garbage hurts 10. *Richmond Times-Dispatch*. Richmond, VA; March 6, 1986: A-1
66. Lloyd-McDonald H. Emergency crews quell toxic blaze. *Herald Sun* Melbourne, Australia; August 27, 2005:011.
67. California Department of Pesticide Regulation. Pesticide Label Database, Currently Registered Phosphine Gas Formulations. 2011. Available at: http://apps.cdpr.ca.gov/cgi-bin/mon/bycode.pl?p_chemcode=3541. Accessed December 11, 2012.
68. Press A. No injuries reported in chemical blast. *The Commercial Appeal*. Nashville, TN; May 3, 2000:B4.
69. Lauer-Williams K. Small blast reported at tech facility. **Employees in Bethlehem evacuated briefly to check air after chemical reaction fire. *The Morning Call*. Allentown, PA; December 15, 2001:B20.
70. Limited NNP. Toxic gas fire scare. *Northern Territory News*. Darwin, Australia; August 27, 2005:013.
71. Staff Writers. Three injured as gas blast rips through Guanxi pesticide factory. *South China Morning Post*. Hong Kong; April 26, 2011:06.
72. Siwik JP, Nowak RM, Zoratti EM. The evaluation and management of acute, severe asthma. *Med Clin North Am*. 2002;86:1049-1071.
73. Rowlands L. Pills turn body into toxic peril / 20 fall sick at hospital. *Daily Telegraph*. Sydney, Australia; November 6, 1998:003.
74. Asbury J. Man Who drank poison becomes chemical risk homet: hazmat closes off some rooms and the streets around the hospital because of a potential reaction. *Press-Enterprise*. Riverside, CA; July 16, 2009: C03.
75. California Department of Pesticide Regulation. California Code of Regulations. Title 3. Food and Agriculture, Division 6. Pesticides and Pest Control Operations. 2011. Available at: <http://www.cdpr.ca.gov/docs/legbills/calcode/030304.htm>. Accessed December 11, 2012.
76. International Press. Two say EPA botched dumping. *The Dallas Morning News*. Dallas, TX; August 29, 1984:6f.