INHALATION OF PHOSPHINE GAS FOLLOWING
A FIRE ASSOCIATED WITH FUMIGATION OF
PROCESSED PISTACHIO NUTS

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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 non-specific systemic symptoms consistent with exposure to phosphine gas. Six of the 10 workers had respiratory distress, indicated by chest pain or shortness of breath, elevated respiratory rate, or decreased oxygen saturation. Recommendations are made for the management of similar illnesses and prevention of similar exposures.

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 accidently 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 (PH$_3$) 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:

$$\text{AlP} + 3\text{H}_2\text{O} \rightarrow \text{Al(OH)}_3 + \text{PH}_3$$

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 for 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 (PH$_3$, heat of formation 5.4 kilojoules/g-mole) to phosphoric acid (H$_3$PO$_4$, heat
of formation -1271.7 kilojoules/g-mole) is an estimated 1277 kilojoules/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 3 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 non-specific symptoms. The California illness data indicate that more than half of the reported cases involve non-specific systemic symptoms (e.g., headache, nausea, diarrhea and dizziness) without accompanying respiratory complaints. Respiratory symptoms (e.g. dyspnea and cough [12]) occur more frequently in conjunction with systemic symptoms than in isolation. Fatal prolonged inhalation exposures in confined spaces[13] may include both systemic effects and pulmonary edema, 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 US EPA[14]. The dose response for inhalation in humans is nevertheless uncertain (see discussion of exposure standards in the section below). Several animal studies examine 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 occur at the LC₅₀ concentration of 0.44 umole/l (calculated as equivalent to 15
In a 2-year chronic inhalation study with phosphine concentrations of 0.3 ppm, 1 ppm, and 3 ppm (at and just above the PEL) no pathological signs of respiratory irritation were present.

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 1980’s and 1990's (Table 2 - 73-KER-88, 79-KER-88) were ascribed to phosphine off-gassing from aluminum phosphide. Eye irritation probably does occur on direct contact with incompletely reacted fumigant dust (1996- 1906, Table 2).

Susceptibility of children

Exposed children may be especially vulnerable to the systemic effects of phosphine, evidenced by differential fatal outcomes in 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 1980’s and 1990’s, many cases of poisonings from ingestion of phosphide fumigants have been reported in India and elsewhere in Asia. Symptoms from these exposures include: fatigue, nausea, headache, dizziness, thirst, cough, shortness of breath, tachycardia, chest tightness, paresthesia, and jaundice. A high mortality rate of 50% to 90% is due to ingestion, and cardiogenic shock is present in severe cases. Pulmonary edema is the most common cause of death.[14] The frequency of pulmonary edema in these cases is far greater than in the occupational cases reported in California.

Ingestions are associated with additional symptoms, including elevation of the ST segments on the 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 from 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]
Zinc levels have been proposed as a marker of exposure to zinc phosphide, based on animal studies. 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, burns near the hairline found at autopsy were noted and suspected to be caused by ignition of phosphine elaborated from aluminum phosphide found in the stomach.[38] A 2011 report by Shadnia, described 2 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 upon the study reported in 1964 by Jones describing phosphine exposures to Australian grain terminal workers.[42] 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, an 8-hour limit of 0.1 ppm was suggested, based upon 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

Fifty ppm is the phosphine IDLH concentration, set by NIOSH[45] based upon reviews by Beliles[46] 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 from 0.25-20 ppm (Tube 121SD), 5-90 ppm (Tube 121SB, Sensidyne), or from 0.05 -2.0 ppm (Tube 121U, Sensidyne).[48] and continuous reading monitors (Draeger and Sensidyne).[48, 49] The OSHA reference method employs a calibrated sampling pump and a glass tube with beaded carbon impregnated with 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 employ 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 a phosphine-generating fumigant. In the 337 cases reported to the California illness registry between 1982-2008 for example, phosphine measurements of workplace air were reported in only 8 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 non-specific, 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 air supplied respirator is acceptable for exposures below 3 ppm. For higher exposures, the NIOSH guide recommends the following:

3ppm - 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. 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 1/2 (or approximately 1/3) 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. 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 U.S. The major uses included stored wheat, peanuts and corn.

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 are “possibly,” “probably” or “definitely” related to application of aluminum phosphide; 75 non-related 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 repored in 9 sporadic cases, all involving application workers. There were 31 isolated cases of eye

* Only licensed applicators may apply them. Use reporting is required.
symptoms, including 6 sporadic cases, and 25 associated with 2) 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 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 of California**
Burgess (2000) briefly described an episode involving failure to properly dispose of aluminum phosphide in Washington.[56] In 2005, Sudakin reported on a manufacturing operation with a history of explosions in hoppers used for formulating phosphide fumigants. The phosphine over exposure 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 (Zn₃P₂ - ZP Rodent Bait®, Dexol Gopher Killer®, et al), used as a bait for rodent control. Zn₃P₂ is more chemically stable than AlP, and only generates PH₃ on contact with stomach acid[60] or a large volume of water.

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 data base.[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 US 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 2). The 9 additional related cases included 3 intentional ingestions (45-SD-01, 21-LA-07, 14-Ven-08, Table 2) and 5 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 (\(\text{Mg}_3\text{P}_2\) - 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 5 formulations registered currently in California, ranging in concentration from 56% to 95% active ingredient.[63] California data showed 7,957 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 US EPA re-registration eligibility document.[43]

Between 1982 and 2008 there were 7 cases were 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 5 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 2 formulations of phosphine mixed with 98% \(\text{CO}_2\) 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”. 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 2 outbreaks associated with accidental exposures to warehouse workers (45-FRE-04, 71-KER-07, Table 2) accounting for all but 3 cases.

Twenty two cases involved isolated systemic symptoms. There were 13 respiratory illnesses, all but 1 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 non-agricultural 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 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 3 hospitals that treated the employees were reviewed. The hospital

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b Regulatory pest control would include use by county departments of agriculture for rodent control and other uses
records 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 California Department of Regulation’s (CDPR) visit to the plant on January 12, 2010 shows bulk bags of pistachios under black plastic films, 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 2,200 pounds (997 kg) and measured 4 ft. x 4 ft. x 5.5 ft. in dimension (88 cubic feet or 2.5 cubic meters). The total volume under fumigation was 200 cubic meters or $2 \times 10^5$ liters.

The bags were elevated off the ground by wooden pallets (Figure 2b), which provided a space beneath for trays of fumigant pellets. Each tray was loaded with 1 pound (453 grams, or approximately 750 pellets) of aluminum phosphide. This corresponds to about 213 pellets per 1,000 cubic feet, which is within the listed “Dosage Range” of the AMF (150 to 450 pellets per 1,000 cubic feet).

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 p.m. and 10:00 p.m. of 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 rain water 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 p.m., 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_\text{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 prior to 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 2 cases were available from information collected by Kern Department of Agriculture staff. Medical records for 7 additional cases were made available to the Department of Pesticide Regulation, Worker Health and Safety 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 3 of the patients seen there, and found abnormalities in each. The 7 patients examined at 2 other hospitals did not have bronchoscopies. There were also minor variations in the use of x-rays and 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.

**A summary of the findings for individual workers is shown below:**

**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, he described additional symptoms including tremor, abdominal pain and sleepiness. Physical examination showed elevated blood pressure (162/93mmHg), rapid heart rate (112 beats per minute), and a rapid respiratory rate (28 cycles per minute). Additional exam findings

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\(^{c}\) Hospital A – Bakersfield Heart Hospital, Hospital B – Mercy Hospital in Bakersfield, Hospital C- Delano Regional Medical Center

\(^{d}\) WHS interview 1/12/2010
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 O$_2$ saturation (97%) and a borderline low level of 
bicarbonate (22 mEq/L). His ECG (electrocardiogram) demonstrated a variable 
heart rate (61-96 beats per minute), with occasional paired premature ventricular 
contractions (PVCs). He had a normal chest x-ray, but the bronchoscopy 
performed prior to 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 4-5 months at the plant. He assisted in putting out the fire. On subsequent 
interview,$^d$ 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 mmHg) and he had a normal chest x-ray.

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 without any medications, but on 
subsequent interview,$^d$ 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**, age 20, a 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 slight elevation of 
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 PaCO$_2$ (25 mm Hg), an elevated PaO$_2$(151 mm Hg, while receiving 1.5 L 
O$_2$/minute by nasal cannula). His chest x-ray was normal except for hyper-inflated 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 re-evaluation at the Hospital B ER. At that ER visit, he had slight elevation of blood pressure, but otherwise normal vital signs, normal O₂ saturation, normal physical findings, and a clear chest x-ray.

He had a CT scan December 22, 2009 that showed no evidence of restrictive lung disease or pulmonary fibrosis. 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 3 separate Pulmonary Function Tests (PFTs), on May 14, 2010, October 18, 2010 and November 30, 2010 that did not meet American Thoracic Society (ATS) standards for acceptability and repeatability. The Pulmonary Function Studies showed decreased lung volumes, indicating possible restrictive lung disease.

Case # 2010-296, the 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 x-ray 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 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 interview⁶), nausea and vomiting. Pulse oximetry showed an oxygen saturation of 95%, but he did not have persistent respiratory difficulty.⁷ A subsequent arterial blood gas, on 2 liters/minute of oxygen, showed 99% saturation. His

⁶ A non-calcified left upper lung granule, was noted, but no findings likely related to the exposure. Specifically, there was no CT evidence of interstitial lung disease.

⁷ 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 non-tender. Breath sounds and chest x-ray were normal.
chest x-ray was reported as normal. He was admitted to the hospital for observation overnight and discharged on December 11, 2009. He reported subsequently\(^5\) 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, eyes burning, 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\(^6\). He had a slightly elevated white blood cell count, normal arterial blood gases on room air, and a normal chest x-ray. 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,\(^7\) 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 PaO\(_2\) (76 mm Hg) and an O\(_2\) 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 x-ray was initially interpreted as showing infiltrates by the ER staff, but was reported as normal by radiology.

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 prior to 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\(^8\)). Upon arrival at the hospital, his O\(_2\) 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 x-ray 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.

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\(^5\) The examination in the Medical Unit noted “few rhonchi and no rales.” He was given an “aerosol treatment because of an obvious wheezing”
Although a repeat chest x-ray a few hours after admission showed a right lower lobe infiltrate, his O_2 saturation rose to 100%. He was discharged on December 11, 2009. He was re-evaluated in the Mercy ER on December 12, 2009 because of chest pain, but his O_2 saturation remained at 100% and a chest x-ray 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\(^h\) - all new since the incident. He was evaluated for a possible acute cerebrovascular accident (stroke), which was ruled out on a computed tomography (CT) scan. He was re-evaluated 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 1 worker had transient infiltrate (an indication of possible pneumonia or fluid on the lung) on a chest x-ray. Bronchoscopy carried out in 3 cases seen at Hospital A showed airway inflammation. Three workers (case numbers: 2009-294, 2009-295, 2009-300) reported persistent symptoms 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.

\(h\) Patient complained of tingling in both arms lasting for about 10 minutes, occurring twice weekly. On exam, there were decreases in his upper arm reflexes and slight decrease of sensation from the right shoulder to the right fingertips.
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 a probably rare occurrence but possible in aluminum phosphide ingestion cases.[9] Exposure to phosphine at possibly toxic levels during hospitalization or at post-mortem (see Table 3, 47-RIV-03, 2003-1075) is much more likely.[73, 74] Health care providers and staff performing post-mortems 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 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 Figures 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. Bronchoscopies performed on 3 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% CO$_2$, 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.

References
41. Wykes, S.L. "FOUR HURT IN SUNNYVALE GAS EXPLOSION HUNDREDS EVACUATED FROM AREA SURROUNDING MICROCHIP FIRM". Jose Mercury News. 1988: San Jose, California.


65. Witt, J. "*EXPLOSION IN GARBAGE HURTS 10.*" *Richmond Times-Dispatch*. 1986: Richmond, VA.
Lloyd-McDonald, H. "EMERGENCY CREWS QUELL TOXIC BLAZE." Herald Sun (Melbourne, Australia). 2005: Melbourne, Australia.


Staff. "THREE INJURED AS GAS BLAST RIPS THROUGH GUANXI PESTICIDE FACTORY." South China Morning Post (Hong Kong). 2011.


Rowlands, L. "PILLS TURN BODY INTO TOXIC PERIL / 20 FALL SICK AT HOSPITAL." Daily Telegraph (Sydney, Australia.)1998.


# Table 1- Phosphine physical/chemical properties in comparison to analog compounds from group V of the periodic table: Ammonia, Stibine and Arsine

<table>
<thead>
<tr>
<th>Property</th>
<th>Ammonia</th>
<th>Phosphine</th>
<th>Arsine</th>
<th>Stibine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular weight grams/mole</td>
<td>17.03</td>
<td>34.04</td>
<td>77.946</td>
<td>124.78</td>
</tr>
<tr>
<td>Formula</td>
<td>NH₃</td>
<td>PH₃</td>
<td>AsH₃</td>
<td>SbH₃</td>
</tr>
<tr>
<td>CAS #:</td>
<td>7664-41-7</td>
<td>7803-51-2</td>
<td>7784-42-1</td>
<td>7803-52-3</td>
</tr>
<tr>
<td>Melting point °C:</td>
<td>-77.7</td>
<td>-134</td>
<td>-116</td>
<td>-88 °C</td>
</tr>
<tr>
<td>Boiling point: °C</td>
<td>-33.35</td>
<td>-87.7</td>
<td>-62.5</td>
<td>-17 °C</td>
</tr>
<tr>
<td>Solubility in water g/100 cc</td>
<td>31 g in 100 cc 25 degrees</td>
<td>0.36 g in 100 cc</td>
<td>28 mg/100 ml @ 20 °C</td>
<td>.41 g/100 c at 0°C</td>
</tr>
<tr>
<td>Density g/ L</td>
<td>.696</td>
<td>1.39</td>
<td>3.18</td>
<td>5.48</td>
</tr>
<tr>
<td>Vapor pressure mmHg at 25 °C</td>
<td>7.51X10+3</td>
<td>2.93X10+4</td>
<td>11,000 (20 °C)</td>
<td>&gt;760 (20 °C)</td>
</tr>
<tr>
<td>Appearance and odor, odor threshold</td>
<td>Sharp, cloying, repellent, like drying urine; 2.6 ppm average odor threshold</td>
<td>fish/garlic odor; 0.02 - 3 ppm; no tests reported with current methodology</td>
<td>Disagree-able garlic odor, 0.5 ppm and above</td>
<td>Disagreeable</td>
</tr>
<tr>
<td>Heat of formation kJ/g-mole</td>
<td>-45.9</td>
<td>5.4</td>
<td>66.4</td>
<td>145.11</td>
</tr>
<tr>
<td>Pattern of signs and symptoms (Toxidrome) after airborne exposure</td>
<td>Eye and upper respiratory symptoms[61]</td>
<td>Headache, nausea, diarrhea and dizziness, dyspnea and cough, less irritating than ammonia[1,22]</td>
<td>Hemolysis and related renal effects, dyspnea, [62] minimal irritation</td>
<td>Hemolysis and related renal effects, dyspnea, [63] minimal irritation</td>
</tr>
</tbody>
</table>
Table 2 -- Applications of phosphine-generating fumigants reported in California during 2009*

<table>
<thead>
<tr>
<th>Active Ingredient</th>
<th>Most frequently treated commodities</th>
<th>Commodity pounds</th>
<th>Rodent, mammalian pest control, unspecified</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aluminum phosphide</td>
<td>Dried fruits, pistachios, almonds, walnuts, rice, wheat</td>
<td>49,369</td>
<td>58,395</td>
<td>107,764</td>
</tr>
<tr>
<td>Magnesium phosphide</td>
<td>Dried fruits, almonds, walnuts, rice, grains</td>
<td>7,630</td>
<td>379</td>
<td>8009</td>
</tr>
<tr>
<td>Zinc Phosphide</td>
<td>None</td>
<td>0</td>
<td>20,893</td>
<td>20,893</td>
</tr>
<tr>
<td>Phosphine/CO2 gas</td>
<td>Almonds, pistachios, dried fruit</td>
<td>30,203</td>
<td>0</td>
<td>30203</td>
</tr>
</tbody>
</table>

Data reported to California Pesticide Information Portal (CalPIP), available online at: calpip.cdpr.ca.gov
Table 3 – Selected safety problems associated with Aluminum Phosphide, Zinc Phosphide, Magnesium Phosphide, or Phosphine fumigations reported to the California illness registry*

<table>
<thead>
<tr>
<th>ID number of index case, and / or priority case number</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1983</strong> 1983-1351</td>
<td>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.</td>
</tr>
<tr>
<td><strong>1983-2159, 1983-2160</strong></td>
<td>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.</td>
</tr>
<tr>
<td><strong>1983-2253</strong></td>
<td>An aluminum phosphide fire occurred at a spaghetti sauce factory in North Hollywood, following 2 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 -- this 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 (ER). Four neighborhood residents were briefly admitted to the hospital for observation. A total of 6 cases were reported.</td>
</tr>
<tr>
<td><strong>1983-2653, 1983-2671</strong></td>
<td>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.</td>
</tr>
<tr>
<td><strong>1984</strong> 1984-2567; 1984-2610; 1984-2571</td>
<td>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 feet from phosphine burning in an open area, also reported symptoms.</td>
</tr>
<tr>
<td><strong>1986</strong> 34-TEH-86, 1986-861</td>
<td>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, 7 fire fighters were exposed to the smoke and were hospitalized for observation.</td>
</tr>
<tr>
<td><strong>1987</strong> 1987-89</td>
<td>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.</td>
</tr>
<tr>
<td><strong>1987-2924</strong></td>
<td>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.</td>
</tr>
<tr>
<td><strong>1988</strong> 73-KER-88, 1988-2574; 79-KER-88, 1988-2911</td>
<td>Workers complained of conjunctivitis -- in 2 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 re-occurrence of eye irritations while working in an almond/pistachio processing plant. They were diagnosed as having &quot;chemical conjunctivitis&quot;.[27]</td>
</tr>
</tbody>
</table>

*ID numbers have been redacted for privacy. |
<table>
<thead>
<tr>
<th>Year</th>
<th>Incident Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>1989-474</td>
<td>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.</td>
</tr>
<tr>
<td>1990</td>
<td>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.</td>
</tr>
<tr>
<td>1990-17-SJ-90, 1990-534</td>
<td>Firefighters put out a fire in 5 piles of cotton debris, each measuring approximately 35 feet x 105 feet x 10 feet, 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 4 firefighters had precautionary medical evaluations, but none reported symptoms.</td>
</tr>
<tr>
<td>1990-3004</td>
<td>Approximately 25% of 3,200 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 cubic foot bin and covered it with a tarpaulin. The bin exploded while 2 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 ear drum. The other worker received facial cuts and burns.</td>
</tr>
<tr>
<td>1990-842</td>
<td>Firefighters put out a fire in 5 piles of cotton debris, each measuring approximately 35 feet x 105 feet x 10 feet, 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 4 firefighters had precautionary medical evaluations, but none reported symptoms.</td>
</tr>
<tr>
<td>1991</td>
<td>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.</td>
</tr>
<tr>
<td>1991-330</td>
<td>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 1 employee of an adjoining business developed headache and non-specific gastrointestinal (nausea, stomach cramps, gas, and diarrhea), and constitutional symptoms (aching muscles, weakness, and fatigue). Three of the firemen were hospitalized overnight and 4 lost 2 days from work. Four additional firefighters received medical evaluation, but had no symptoms.</td>
</tr>
<tr>
<td>1993</td>
<td>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 7 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.</td>
</tr>
<tr>
<td>1993-24-SD-93, 1993-1513</td>
<td>After unloading 5 car loads 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.”</td>
</tr>
<tr>
<td>1993-742</td>
<td>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 3 employees inhaled the smoke.</td>
</tr>
<tr>
<td>1994</td>
<td>A county agricultural inspector entered an unposted chamber that was fumigated 3 days earlier. She was there to do a pre-fumigation inspection with a 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.</td>
</tr>
<tr>
<td>Year</td>
<td>Case Number</td>
</tr>
<tr>
<td>------</td>
<td>-------------</td>
</tr>
<tr>
<td>1996</td>
<td>1996-1906</td>
</tr>
<tr>
<td>1996-1977</td>
<td>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.</td>
</tr>
<tr>
<td>1997</td>
<td>4-ST-A-97, 1996-1872</td>
</tr>
<tr>
<td>1998</td>
<td>1998-96</td>
</tr>
<tr>
<td>1998-878, 1998-879</td>
<td>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.</td>
</tr>
<tr>
<td>1999</td>
<td>1999-662</td>
</tr>
<tr>
<td>2000</td>
<td>38-LA-00, 2000-558</td>
</tr>
<tr>
<td>2001</td>
<td>45-SD-01, 2001-708</td>
</tr>
<tr>
<td>2002</td>
<td>39-LA-02, 2002-219</td>
</tr>
<tr>
<td>2003</td>
<td>24-MAD-03, 2003-468</td>
</tr>
<tr>
<td>2003-515</td>
<td>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.</td>
</tr>
<tr>
<td>47-RIV-03, 2003-1075</td>
<td>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 U.S. 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 2 ill coroner's staff members included dry mouth, headache, runny nose, sore throat, dizziness, nausea and vomiting.</td>
</tr>
<tr>
<td>2004</td>
<td>45-FRE-04, 2004-937</td>
</tr>
<tr>
<td>Year</td>
<td>Event Code</td>
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<tr>
<td>------</td>
<td>------------</td>
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<tr>
<td>2005</td>
<td>33-RIV-05, 2005-1307 to 1310</td>
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<tr>
<td>2006</td>
<td>45-SBD-06, 2006-613</td>
</tr>
<tr>
<td>2007</td>
<td>71-KER-07, 2007-1229</td>
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<td>2007-249</td>
<td></td>
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<tr>
<td>2007-713</td>
<td></td>
</tr>
<tr>
<td>21-LA-07</td>
<td></td>
</tr>
<tr>
<td>2008</td>
<td>14-VEN-08, 2008-131</td>
</tr>
</tbody>
</table>
Figure 1 — California cases related to phosphine or phosphide fumigants, 1982-2009

Note: 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.
Figure 2a. Pistachio Bags covered by Black Film
Figure 2b. Wooden pallets under the stacks of pistachio bags
Figure 2c. Downspout from an adjacent warehouse
Figure 2d. Bottom of burned pistachio sacks
Figure 3a. SAMPLE WARNING POSTER FOR ALUMINUM PHOSPHIDE

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  
Fosfuro de Aluminio puede producir un gas tóxico-Fosfina

ALERT SUPERVISOR       Pida ayuda de su supervisor
DANGER FUMIGATION
¡PELIGRO FUMIGACIÓN!

DO NOT ENTER
NO ENTRE

Fumigant: Aluminum Phosphide
Fumigant: Fosfuro 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