Phosphine

Analysis of an exposure incident in a commodity warehouse

By Gerald Beaumont and Joan Beaumont

Abstract: *An exposure incident at a commodity warehouse fumigated with phosphine resulted in permanent lung damage, ranging from moderate to severe, in warehouse employees. The exposure resulted from a violation of EPA's pesticide registration during the fumigation. This article examines the limited air monitoring data collected during and after the fumigation and concludes that airborne concentrations exceeded accepted exposure limits for phosphine and for phosphoric acid. The authors call for new preventive measures to address this concern.*

PHOSPHINE IS A STRONG REDUCING AGENT used as a doping agent in the manufacture of semiconductors, as a chemical intermediate in the chemical industry and as a fumigant in the pesticide industry. In the semiconductor and chemical industries, phosphine is used in closed systems with releases into the work area being carefully controlled.

Phosphine also has been used for several years as a fumigant to kill insects and their larvae, and rodents in commodities and food processing facilities. Fumigation requires releasing the substance into the air of the space to be treated. Phosphine diluted in carbon dioxide and some phosphine-generating materials, aluminum and magnesium phosphides (metal phosphides) are registered pesticides regulated by EPA. These pesticides are recommended for the control of a broad spectrum of insects and their larvae, and several small mammals.

Phosphine has been used to treat animal hides and furs, natural fibers or cloth and clothing, wool and hair, leather products, tobacco and tobacco products, wood and wood products, paper and paper products, flowers, grains and flour, nuts, fodder, vegetables, and many prepared food products and food processing and storage facilities. It is a gas and leaves no significant residual when properly aerated.

Humans have died from inadvertent exposure during commodity fumigations and in other situations involving airborne phosphine (Heyndrickx, van Petigham, van den Heede, 1976; Wilson, Lovejoy, Jaeger, et al., 1980). As a pesticide, phosphine and phosphine-releasing materials may only be purchased by licensed pesticide applicators for use in accordance with the product labeling.

The case described here occurred because EPA registration requirements were not followed. Employees were exposed to phosphine and related materials following a fumigation. As a result, they suffered residual lung damage. It was difficult to reconstruct the exposures because of changes made to the warehouse and limited access to the modified building. A graphical presentation of the data collected during and after the fumigation allowed the investigator to estimate the concentration of phosphine. Published data on the environmental fate of phosphine (Frank & Rippen, 1986) provide a basis for using chemical reaction rate calculations to estimate the concentration of phosphorous oxyacids in the warehouse's separate sections.

Toxicity of Materials

Phosphine

In its "Note to Physician," one manufacturer (Degesch America) states:

> Magnesium phosphide fumigant reacts with moisture in air, water, acids and many other liquids to release phosphine gas. Mild inhalation exposure causes malaise (indefinite feeling of sickness), ringing in the ears, fatigue, nausea and pressure in the chest which is relieved by removal to fresh air. Moderate poisoning causes weakness, vomiting, pain just above the stomach, chest pain, diarrhea and dyspnea (difficulty in breathing). Symptoms of severe poisoning may occur within a few hours to several days, resulting in pulmonary edema (fluid in lungs) and may lead to dizziness, cyanosis (blue or purple skin color), unconsciousness and death.
>
> In sufficient quantity, phosphine affects the liver, kidneys, lungs, nervous system and circulatory system. Inhalation can cause lung edema (fluid in lungs) and hyperemia (excess blood in a body part), small perivascular brain hemorrhages and brain edema (fluid in brain). Ingestion can cause lung and brain symptoms but damage to the viscera (body cavity organs) is more common. Phosphine poisoning may result in 1) pulmonary edema, 2) liver elevated serum GOT, LDH and alkaline phosphatase, reduced prothrombin, hemorrhage and jaundice (yellow skin color) and 3) kidney hematuria (blood in urine) and anuria (abnormal or lack of urination). Pathology is characteristic of hypoxia (oxygen deficiency in body tissue). Frequent exposure to concentrations above permissible levels over a period of days or weeks may cause poisoning. Treatment is symptomatic.

One study of the toxicity of phosphine in rats concluded that the primary hazard to exposed rats was

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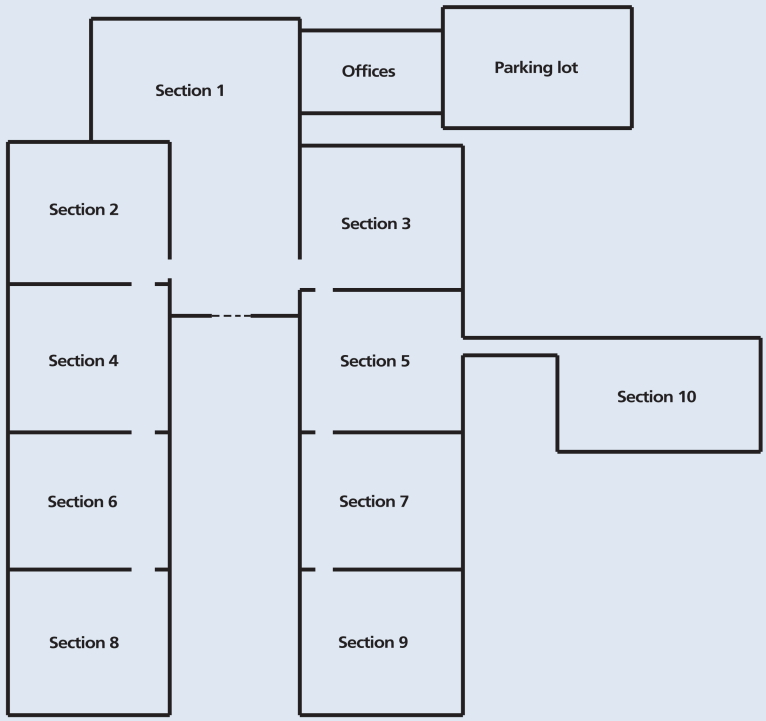
Figure 1

Plan of Tobacco Warehouse



The warehouse was divided into 10 sections. Sections 1, 2 and 3 were chosen for analysis because they are connected in a U-shape and because section 1 was the staging area from which employees received assignments for retrieving commodity.

lethality and that exposure-related sublethal effects were completely reversible after 13 weeks of exposure to 0.3, 1 and 3 ppm phosphine (Newton, Schroder, Sullivan, et al., 1993). Another study found that single, 4-hour exposures to phosphine at 21, 28 and 40 ppm reduced activity in rats, but they fully recovered within a week of exposure (Schafer, Newton, Gruebbel, et al., 1998). That study also concluded that phosphine was not neurotoxic to rats at 0.3, 1 or 3 ppm after 13 weeks of exposure.

A 2-year inhalation study of rats exposed to 0.3, 1 and 3 ppm concluded that no exposure-related changes were suggestive of a toxic or carcinogenic effect (Newton, Hilaski, Banas, et al., 1999). OSHA's permissible exposure limit (PEL) for phosphine and American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV) for phosphine is 0.3 ppm.

Phosphonic Acid

The authors found no reference to phosphonic acid toxicity in the literature. As it is a weak acid, it is likely to have toxicity similar to phosphoric acid.

Phosphoric Acid

Little information in the literature addresses airborne phosphoric acid. Phosphoric acid is a weak acid, so a mist of it is expected to be irritating to eyes, skin and respiratory tract. Inhalation of significant amounts of a weak acid is expected to cause swelling and excess fluid in the lungs and lower serum pH. Ingestion of phosphoric acid is likely to be corrosive to the esophagus and affect serum pH. Partially neutralized phosphoric acid is used in colas to buffer the pH in the range of 2 to 2.5; thus, intake of phosphate does not have substantial toxicity, unless it is in the fully acid form. The PEL and TLV for phosphoric acid is 1 mg/m^3.

Background & Overview of the Exposure

The exposure incident occurred at a warehouse that stored bales of tobacco. To prepare the facility for fumigation, all electronic equipment and metal objects that would readily be damaged by the fumigant were removed. Objects of this nature, including coins and the radio repeater antenna, had suffered significant corrosion during prior fumigation. To seal the warehouse, vents were covered with plastic sheeting, and cracks around building openings and the sheeting were taped.

Employees went home on Friday afternoon and were away from the warehouse during the week of fumigation. Placards were placed on building entrances, and packets of information about phosphine releases were placed in all 10 sections of the commodity warehouse.

Measurements were taken and recorded inside the warehouse and from inside the baled commodity at time intervals during fumigation. The exact locations of the sampling were not available to the author. The fumigator used a self-contained breathing apparatus to protect against the airborne phosphine, since no cartridge is known to protect against phosphine, and its warning properties are considered inadequate for use of a cartridge respirator.

At about 101 hours from the start of fumigation, the sealing materials were removed and the warehouse doors were opened to allow airflow to remove the fumigant. Phosphine in the warehouse and inside of the commodity was again measured and documented. The phosphine rapidly cleared from the warehouse atmosphere, but some remained inside the commodity. The placards were removed and the warehouse doors were closed on Friday afternoon, about 151 hours after fumigation began.

Employees returned to work on Monday morning and experienced respiratory problems. Two employees who went beyond section 1 became nauseous and vomited. Sensing something was wrong, management sent the sick employees home after about 2 hours of exposure.

Before leaving the warehouse, the manager and another employee opened all the doors to air out the building, at approximately 215 hours after fumigation. The fumigator returned to the warehouse about 3 hours later, after driving from his home base. He measured the concentration of phosphine in the ambient air and in some bales of tobacco. The pesticide manufacturer filed a voluntary report to EPA some months later.

Employees' symptoms worsened and they visited a physician the next day. One employee developed serious respiratory problems about 3 days after the exposure. Physicians treated the employees sympto-

matically, as recommended in the manufacturer's note. These employees have mild to severe residual lung impairment from this exposure incident.

Measurement Methods

The fumigator measured phosphine using a Portasens (Analytical Technology Inc., Oaks, PA) and a Draeger bellows pump with unspecified detector tubes for phosphine. The electrochemical, direct-reading instrument has a detection limit of approximately 10 ppm when fitted with the high-range phosphine sensor, and has a detection limit of about 0.02 ppm when fitted with the PPB sensor for phosphine. The detector tubes in the low range have detection limits of about 0.1 ppm.

Employee exposure monitoring for time-weighted-average (TWA) and short-term exposures during routine use can be accomplished using OSHA method 1003 or NIOSH method 6002. The OSHA method uses a mercuric-chloride-treated polyester filter to collect phosphine. Air flow through the cassette for TWA measurements is 1 liter per minute (Lpm) for up to 4 hours, and is 2 Lpm for the 15-minute measurement. The filter is then digested in sulfuric acid and the resulting solution analyzed using an inductively coupled plasma-atomic emission spectrometer (ICP-AES).

The NIOSH method uses a silica gel tube, 300/150 mg, treated with mercuric cyanide. The recommended air flow rate through the tube ranges from 0.01 to 0.2 Lpm, with a minimum air volume of 1 L and a maximum of 16 L. The silica gel tube is desorbed with hot, acidic permanganate solution and the resulting solution analyzed using an ultraviolet-visible spectrophotometer.

These methods require calibrated sampling pumps and yield similar exposure results. The NIOSH method utilizes a glass sampling tube, which may be broken during sampling, while the OSHA method uses a plastic cassette. Each method requires moderately expensive analytical equipment and preparation of reagents, so analysis is commonly completed by an accredited analytical laboratory.

With either method, the collection media has a relatively short shelf life. One supplier reports a shelf life of 30 days for the NIOSH method media, and 90 days for the OSHA method media. (Always check with suppliers for specifics on the shelf life of their sampling supplies for phosphine.)

Phosphoric Acid

NIOSH method 7903 and OSHA method 165SG use a washed silica gel tube, 400/200 mg, to collect phosphoric acid, which is desorbed with a carbonate buffer solution, and analyzed by ion chromatography (IC). The air sampling rate for these methods is 0.2 Lpm. The silica gel tube is made of glass and may be subject to breakage. Analysis is typically performed by an accredited analytical laboratory.

OSHA method 111 used a mixed cellulose ester filter to collect phosphoric acid at an air flow rate of 2 Lpm, a carbonate buffer solution for desorption, and analysis by IC. This method has been superseded by OSHA method 165SG.

Graphing Methods

Phosphine concentration versus time was plotted manually on a large pad of paper with quarter inch grids. Data points were connected with a smooth line using a pen and a ships curve, a drafting tool with varying curvature for creating a smooth line through nonlinear data points. One section of the graph used an enlarged phosphine scale, to be able to see the changes in the plotted data at lower concentrations.

Projections of data for the period after the warehouse was opened were calculated using slopes equivalent to the rate of concentration decline just

On the day of the incident, employees spent most of their exposure time in section 1. The fire doors were open between sections during the fumigation, so some diffusion occurred between sections 1, 2 and 3.

Table 1

Phosphine Concentrations Measured at Various Times During Fumigation

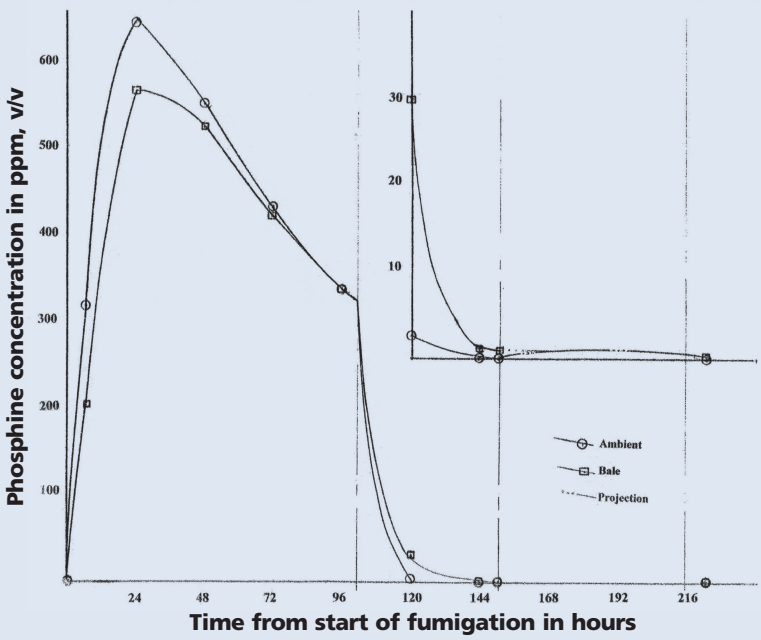
	Time from start of fumigation (hours)	Concentration of phosphine in air in this section (ppm)	Concentration of phosphine in commodity in this section (ppm)
Section 1	6	320	205
	24	650	570
	48	555	530
	72	435	425
	96	340	340
	120	2.6	30
	144	ND	1.2
	151	ND	0.9
	223	ND	0.2
Section 2	6	330	210
	24	645	520
	48	555	530
	72	435	410
	96	340	340
	120	3.2	40
	144	ND	3.1
	151	ND	0.9
	223	ND	0.7
Section 3	6	345	210
	24	600	470
	48	500	475
	72	405	395
	96	320	305
	120	1.9	20
	144	ND	3.9
	151	ND	3.1
	223	ND	0.6

Note. *ND = material was not detected*

Phosphine concentration versus time was plotted manually. Data points were connected with a smooth line using a pen and a ships curve (a drafting tool with varying curvature for creating a smooth line through nonlinear data points). One section of the graph used an enlarged phosphine scale, to be able to see the changes in the plotted data at lower concentrations.

Figure 2

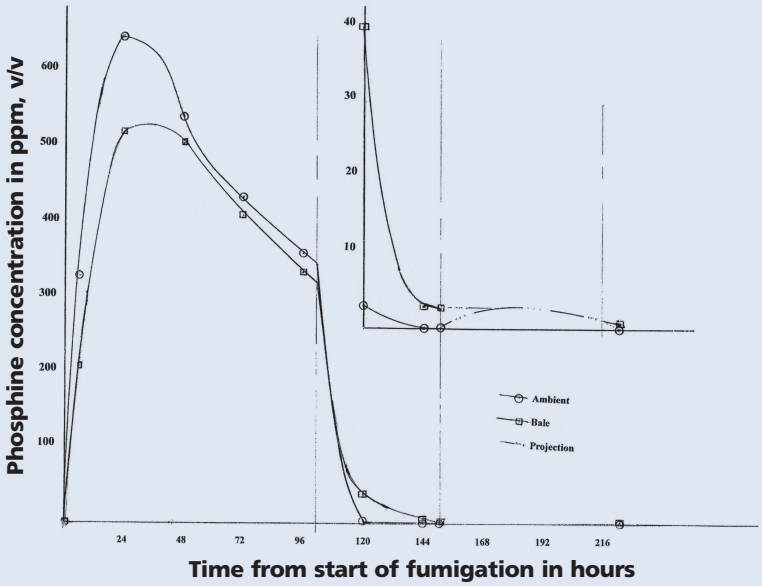
Phosphine Concentration Over Time, Section 1



Note. *Phosphine concentration versus time from start of fumigation in warehouse section 1.*

Figure 3

Phosphine Concentration Over Time, Section 2



Note. *Phosphine concentration versus time from start of fumigation in warehouse section 2.*

before the warehouse was closed and the concentration measured in the commodity on the afternoon of the exposure.

The projection of the ambient concentration was calculated using curves estimating a concentration based on natural diffusion. Vertical lines on the graphs at hour 101 reflect the change in ventilation from sealed to wide open; at hour 151, the lines reflect closure of all doors; and at hour 215 the lines indicate the opening of the doors.

Discussion

The warehouse was divided into 10 sections (Figure 1, p. 34), separated by fire walls and fire rated doors that close after a fusible link melts. Most sections were filled with stacked bales of tobacco—up to 75% of the section's volume. Section 1 was filled to about 50% of the volume, because its staging area contained no commodity.

Sections 1, 2 and 3 were chosen because they are connected in a U-shape with section 1 in the middle, and because it was the staging area from which employees received assignments for retrieving commodity.

On the day of the incident, employees spent most of their exposure time in this area. The fire doors were open between sections during the fumigation, so some diffusion occurred between sections 1, 2 and 3. Table 1 (p. 35) presents data collected by the fumigator for three of the warehouse sections. Figures 2, 3 and 4 are graphical presentations of the data and projections.

Phosphine oxidizes in air to phosphorous oxyacids and the published half-life is 28 hours in the absence of ultraviolet light (Frank & Rippen, 1986). This oxidation of phosphine at ppm concentrations is a first order chemical reaction, which is dependent on the concentration of hydroxyl radical (OH). (Phosphine is known to catch fire without an ignition source when released into air at higher concentrations, because it is

a strong reducing agent.) Stoichiometrically, the following reactions are likely:

$$PH_3 + 6\ OH \longrightarrow H_3PO_3 + 3\ H_2O$$

$$PH_3 + 8\ OH \longrightarrow H_3PO_4 + 4\ H_2O$$

For a batch reaction, the following equation can be used to determine the amount of phosphine converted to phosphorous oxyacids (Barrow, 1961):

$$\ln C_0 - \ln C = kt$$

where:

C_0 = concentration of phosphine at start of reaction at t_0

C = concentration of phosphine at time t

t = time elapsed during reaction

k = rate constant = 0.693/half-life

To simplify the calculations, the phosphorous oxyacid product was considered to be either phosphonic acid or phosphoric acid, when, realistically some combination of the two is most likely. Since phosphine continued to diffuse out of the commodity during the time that ventilation was limited by door closure (hours 151 to 215 after fumigation started) oxidation of the phosphine was not truly a batch process.

A good approximation can be achieved by considering several batch reactions starting at time intervals throughout that period. As the graphs were created using 6-hour grids, those times were used. The sum of these batches is the resulting concentration of phosphorous oxyacid, presented as phosphonic or phosphoric acid. Table 2 (p. 38) shows the starting concentrations for each 6-hour period and the results of the multiple batch calculations in warehouse sections 1, 2 and 3.

Although no PEL or TLV exists for phosphonic acid for comparison, the calculated concentration of airborne phosphoric acid in the staging area of section 1 was more than 15 times the PEL and TLV of 1 mg/m^3 for phosphoric acid, and was probably much higher due to diffusion from sections 2 and 3. The concentrations, as phosphoric acid, in sections 2 and 3, were more than 45 and 47 times the PEL and TLV, respectively. The concentration of phosphine projected for section 1 was double the PEL and TLV of 0.3 mg/m^3 when employees entered, and was 5 to 6 times the PEL and TLV for sections 2 and 3.

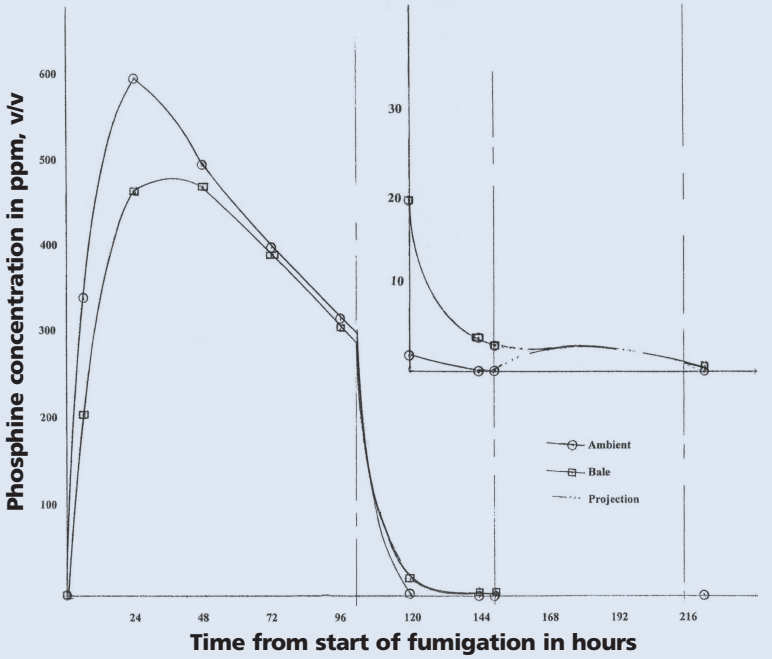
The particles of acid were generated one molecule at a time, so those particles were very small and, thus, were respirable, so they would penetrate deep into the lung and affect small airways. The irritation caused by these weak acids would be similar to that caused by elevated carbon dioxide (the limiting factor in holding one's breath) and would cause increased depth and rate of respiration, increasing the total doses of acid and phosphine to each exposed employee.

The lung edema seen in the overexposed employees was likely the result of severe irritation from inhaling these acids. Residual damage, as seen in this case, may have been caused by the edema isolating sections of deep lung tissue, with an extended period of acid attack on lung tissue resulting from increased expulsion of carbon dioxide (CO_2) to the lung to adjust serum pH, after the rapid influx of substantial acid from the air.

Sections of the lung prevented from exhaling CO_2 due to swelled airways would remain acidic, as CO_2 was expelled from the blood, until those airways opened or pH balance was restored, likely causing lung damage. The nausea and vomiting that some exposed employees experienced likely resulted from the initial pH imbalance from inhaling larger amounts of airborne acid.

Figure 4

Phosphine Concentration Over Time, Section 3



Note. Phosphine concentration versus time from start of fumigation in warehouse section 3.

Conclusions

EPA regulations require that registered pesticides be used in accordance with labeling, which includes the applicator's manual for the pesticide. The manual for the pesticide used at this site required that warning placards remain in place until the fumigated commodity contained 0.3 ppm or less of phosphine. Placards were removed with the phosphine concentration in the tobacco in considerable excess of 0.3 ppm, which violates EPA regulations. This review of the data indicates that this incident would not have occurred had those regulations been followed.

Employees at this warehouse were exposed in considerable excess of the PEL and TLV for both phosphine and phosphorous oxyacids, when expressed as phosphoric acid, and suffered permanent

The concentration of phosphine projected for section 1 was double the PEL and TLV of 0.3 mg/m^3 when employees entered, and was 5 to 6 times the PEL and TLV for sections 2 and 3.

lung impairment. This combined exposure is probably why phosphine fumigation exposures result in more damage than would be projected from the published studies of rats exposed to phosphine.

Those animal studies used phosphine that was mixed with nitrogen to dilute it, which prevented it from oxidizing prior to being released into the air stream leading into the exposure chamber, and from starting on fire upon release. Several air changes occurred per hour in the chambers, so the time phosphine spent in each chamber was limited to minutes, and, thus, oxidation was limited. Animal exposures were to phosphine with minimal or no phosphorous oxyacids.

Recommendations for Further Action

Pesticide applicators must follow the requirements set forth in the registration of each pesticide being used in order to prevent incidents such as the one described here. Physicians must prepare possible treatment methods to moderate the effects of overexposure to the combination of phosphine and phosphorous oxyacids, with focus on moderating the affects of overexposure to acid. Those treatments should be widely published, and presented to management at each facility to be fumigated prior to that activity.

Phosphine is an effective fumigant and will continue to be used. Therefore, additional exposure incidents are likely, so a sound treatment plan is needed to reduce injury and death among those overexposed. ■

Table 2

Phosphine Concentrations After Building Closure & Resulting Phosphoric Acid Concentration in Warehouse Sections 1-3

	Time from start of fumigation (hours)	Phosphine concentration (ppm)	Conversion time (hrs)	Concentration of phosphoric acid generated in time period batch (mg/m^3)
Section 1	156	0.4	58	1.22
	162	0.8	52	2.32
	168	0.9	46	2.45
	174	0.9	40	2.26
	180	0.9	34	2.05
	186	0.9	28	1.35
	192	0.8	22	1.35
	198	0.8	16	1.05
	204	0.7	10	0.616
	210	0.6	4	0.227
	214	0.6	0	0
				Total 15.3
Section 2	156	1.4	58	4.28
	162	2.2	52	6.39
	168	2.6	46	7.09
	174	2.7	40	6.80
	180	2.7	34	6.16
	186	2.6	28	5.21
	192	2.4	22	4.04
	198	2.3	16	3.01
	204	1.7	10	1.76
	210	1.7	4	0.64
	214	1.1	0	0
				Total 45.4
Section 3	156	1.4	58	4.28
	162	2.1	52	6.10
	168	2.6	46	7.09
	174	2.8	40	7.25
	180	2.9	34	6.63
	186	2.8	28	5.61
	192	2.7	22	4.52
	198	2.4	16	3.15
	204	2.0	10	1.76
	210	1.6	4	0.60
	214	0.9	0	0
				Total 47.0

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