IPCS International Programme on Chemical Safety

Environmental Health Criteria 73

Phosphine and Selected Metal Phosphides

Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organisation, and the World Organization

D HEALTH ORGANIZATION GENEVA 1988

Other titles available in the ENVIRONMENTAL HEALTH CRITERIA series include:

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the United Nations Environment Programme,
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The **International Programme on Chemical Safety (IPCS)** is a joint venture of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization. The main objective of the IPCS is to carry out and disseminate evaluations of the effects of chemicals on human health and the quality of the environment. Supporting activities include the development of epidemiological, experimental laboratory, and risk-assessment methods that could produce internationally comparable results, and the development of manpower in the field of toxicology. Other activities carried out by the IPCS include the development of know-how for coping with chemical accidents, coordination of laboratory testing and epidemiological studies, and promotion of research on the mechanisms of the biological action of chemicals.

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SELECTED METAL PHOSPHIDES

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NOTE TO READERS OF THE CRITERIA DOCUMENTS

Every effort has been made to present information in the criteria documents as accurately as possible without unduly delaying their publication. In the interest of all users of the environmental health criteria documents, readers are kindly requested to communicate any errors that may have occurred to the Manager of the International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, in order that they may be included in corrigenda, which will appear in subsequent volumes.

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A detailed data profile and a legal file can be obtained from the International Register of Potentially Toxic Chemicals, Palais des Nations, 1211 Geneva 10, Switzerland (Telephone no. 988400 - 985850).

ENVIRONMENTAL HEALTH CRITERIA FOR PHOSPHINE AND SELECTED METAL PHOSPHIDES

A WHO Task Group on Environmental Health Criteria for Phosphine and Selected Metal Phosphides met in Geneva on 17-21 November 1986. Dr E.M. Smith opened the meeting on behalf of the Director-General. The Task Group reviewed and revised the draft criteria document and made an evaluation of the risks for human health and the environment from exposure to phosphine and metal phosphides.

The original draft of this document was prepared by DR J.R. JACKSON, Department of Medicine and Health Science, Monsanto Europe, Brussels, Belgium.

The contributions of all who helped in the preparation and finalization of this document are gratefully acknowledged.

1. SUMMARY

1.1 Properties, Analysis, and Occurrence

Phosphine, or hydrogen phosphide, is a colourless gas which is odourless when pure, but the technical product usually has a foul odour, described as "fishy" or "garlicky", because of the presence of substituted phosphines and diphosphine (P_2H_4). Other impurities may be methane, arsine, hydrogen, and nitrogen. For fumigation, it is produced at the site by hydrolysis of a metal phosphide and supplied in cylinders either as pure phosphine or diluted with nitrogen. Aluminium, magnesium (trimagnesium diphosphide), and zinc (trizinc diphosphide) phosphides are the most commonly used metal phosphides for this purpose.

Phosphine is flammable and explosive in air and can autoignite at ambient temperatures. It is slightly soluble in water and soluble in most organic solvents. Metal phosphides are usually powders of various colours, which hydrolyse in acids to yield phosphine and metal salts. Aluminium and magnesium phosphides hydrolyse in water.

Phosphine in air can be detected by the discoloration of silver nitrate or indicator papers impregnated with mercury (II) chloride, and can be measured using indicator tubes or by flame photometry, infrared spectroscopy, mass spectrometry, or gas chromatography. Samples can be taken on solid adsorbents and desorbed for analysis. Various classical analytical techniques are available in which phosphine is trapped by mercury (II) salts.

Phosphine residues in foods can be measured by nitrogen purging and trapping the phosphine. Phosphide residues can be included in the analysis by extraction with silver nitrate or sulfuric acid and measuring the chromophore or the phosphine contents of the head-space gas, respectively.

Metal phosphides are most easily determined after hydrolysis to phosphine. A method has been described in which magnesium phosphide is converted to magnesium pyrophosphate, which is then weighed.

Phosphine may occur naturally in the anaerobic degradation of phosphorus-containing organic matter, such as in the production of marsh gas. Naturally-occurring phosphides are extremely rare. None have been reported in the earth's crust but an iron-nickel phosphide (schreibersite) is found in all iron meteorites.

Phosphine is manufactured by the hydrolysis of metal phosphides, by the electrolysis of phosphorus in the presence of hydrogen, and by a phosphorus-steam reaction. It is produced incidentally by the hydrolysis of impurities in calcium carbide (in the production of ethyne (acetylene)), in ferrosilicon and

spheroidal graphite iron, and in various metallurgical operations. Metal phosphides can be prepared by the reduction of phosphates, by a direct reaction between the metal and phosphorus vapour or amorphous phosphorus, or by an exchange reaction between the metal and another metal phosphide.

Phosphine is used in the synthesis of organophosphines and organic phosphonium derivatives and as a dopant in the manufacture of semiconductors in the electronics industry. Formulations of aluminium or magnesium phosphide are available for fumigation in pest control. Zinc phosphide is used as a rodenticide in the form of a powder or a paste containing 2.5 or 5% of zinc phosphide, which is incorporated in bait at 1 part in 10.

Phosphine in air reacts with HO· radicals and is removed by this mechanism with a half time of 5 - 28 h depending on the conditions. Phosphine in air is slowly absorbed by soil at a rate that is dependent on surface effects and the permeability of the soil matrix, and is slower in wet conditions. Zinc phosphide undergoes negligible hydrolysis in a variety of surface and ocean waters over a period of 11 days, but is oxidized within about five weeks in soils with a 50% or more saturated moisture content. Significant hydrolysis only occurs at a pH value of 4 or less. Aluminium and magnesium phosphides are rapidly hydrolysed in neutral moist conditions. Phosphine and zinc, magnesium, and aluminium phosphides are inherently degradable and non-persistent in the environment. The ultimate fate for the phosphides is inorganic phosphate, water, and metallic compounds.

Phosphine is normally undetectable in air, water, or soil. Residues in fumigated foods depend on the technique of fumigation, but are normally low after aeration, except where a metal phosphide fails to react completely. In general, residues are below the WHO/FAO recommended levels of 0.1 mg/kg (PH_3) for raw cereals and below 0.01 mg/kg (PH_3) for other stored products.

1.2 Effects on Organisms in the Environment

The antimicrobial activity of phosphine varies depending on the microbial species, and the type and moisture content of the product being fumigated. Many microorganisms survive fumigation at exposures (concentration·time) that are effective against arthropods.

The few studies available indicate that phosphine has little effect on growing plants (e.g., sugar cane and lettuce) at effective pesticidal doses. Germination of seeds was unaffected by their fumigation with phosphine or by prior fumigation of the soil in which they were planted, except when the moisture content of the seed exceeded 20%. Lettuce sustained substantially less damage after fumigation with phosphine than with 5 other fumigants.

The few data available on the effects of phosphine and phosphides on aquatic organisms suggest that, despite its low solubility, phosphine in solution can be acutely toxic.

Generally, insects, a principal target, are susceptible, though the susceptibility at different stages of the life-cycle varies and diapausing larvae are particularly tolerant. The threshold for adverse effects of phosphine to *Drosophila melanogaster* is about 1.4 mg/m^3 (1 ppm), which is similar to the threshold for acute inhalation effects in mammals. Resistant strains of insects exist and are sometimes difficult to control. The mechanism of resistance may have a metabolic basis that persists throughout all stages of metamorphosis, and in some cases has been shown to be a process of active exclusion of phosphine by energy-dependent processes. In general, mites are less sensitive than insects.

Wild birds and mammals are similarly susceptible to both phosphine and phosphides. Zinc phosphide formulations vary in acceptability as baits, and the relative efficacy of different commercial preparations may be a function of this. Aversion to zinc phosphide at 0.05% in the diet was demonstrated in Indian gerbils. Census studies have shown that, with appropriate use of bait or the application of aluminium phosphide to the entrance of active burrows, elimination of most or nearly all target species can be achieved with a single application. Ingested zinc phosphide is detectable in the intestine and liver of poisoned animals, but not in the muscle tissue. Poisoned animals are not toxic to carrion eaters.

1.3 Effects on Animals

In mammals, phosphine is readily absorbed by inhalation. Aluminium or magnesium phosphide powder, if inhaled, releases phosphine for absorption on contact with the moist respiratory epithelium. Zinc phosphide would not hydrolyse rapidly in the respiratory tract but might be absorbed as such and hydrolyse in the tissues. The acute dermal LD_{50} for zinc phosphide in rabbits is in the range of 2000 - 5000 mg/kg body weight, suggesting little dermal absorption. Gastrointestinal absorption of phosphine produced by the hydrolysis of ingested phosphide is likely and the absorption of zinc phosphide itself and its transport to the liver, where it can be detected for many hours, has been demonstrated in the rat and in a case of fatal human poisoning. Information regarding the distribution of phosphine in the body has been derived from the clinical syndromes of poisoning, which indicate that it reaches the central nervous system, liver, and kidney.

Absorbed phosphide is hydrolysed to phosphine or oxidized to the salts of the oxyacids of phosphorus. Phosphine is both slowly oxidized to oxyacids and excreted unchanged in the

expired air. Hypophosphite is the principal urinary excretion product. Following an oral dose of zinc phosphide, phosphine in the expired air had disappeared after 12 h and clinical symptoms lasted only a few hours. This suggests that phosphine is eliminated fairly rapidly. On the other hand, the phosphide contents of the liver were higher after daily dosing with zinc phosphide than after a single dose, suggesting that liver phosphide is not completely eliminated within 24 h.

Studies by the inhalation route indicate that both the concentration and duration of exposure are important determinants of acute lethality and that different mammalian species are essentially similar in susceptibility. The 4-h LC_{50} of phosphine in rats is about 15 mg/m^3. The oral LD_{50} value of zinc phosphide in wild Norwegian rats is 40.5 mg/kg body weight.

Results of short-term administration indicate that the effects of phosphine exposure cumulate with daily exposure so that after 6 days pretreatment, the survival time at a concentration of 681 mg/m^3 was reduced to one-third of its value in animals without previous exposure. Clinical features of liver and kidney dysfunction were observed and all the parenchymatous organs were affected by congestion and oedema. Neurohistological changes were seen in rats and less markedly in guinea-pigs and cats. Changes in various serum enzyme levels at very low levels of exposure over a period of 1.5 months have been reported. Short-term feeding studies on female rats administered zinc phosphide resulted in mortality at concentrations of 200 and 500 mg/kg diet, but biological effects, qualitatively similar to those at higher doses, were seen at the lowest dietary concentration of 50 mg/kg.

There are no studies relating to long-term effects, carcinogenicity, or mutagenicity. There is no information regarding factors modifying toxicity in vertebrates or the toxicity of any metabolites. Information on biochemical effects is insufficient to explain the mechanisms of toxicity, in either animals or plants.

1.4 **Effects on Man**

Because the odour of phosphine depends on impurities which may be removed by purification or adsorption, odour cannot be relied on for warning of toxic concentrations.

Ingestion of phosphides may cause nausea, vomiting, diarrhoea, retrosternal and abdominal pain, tightness in the chest and coughing, headache and dizziness. In more severe cases this may progress to cardiovascular collapse, pulmonary oedema, cyanosis and respiratory failure. Pericarditis, renal failure, and hepatic damage including jaundice, may develop later.

Symptoms may be delayed and death may occur up to one week after poisoning. Pathological findings include fatty degeneration and necrosis of the liver and pulmonary hyperaemia and oedema.

Inhalation of phosphine or phosphide may cause severe pulmonary irritation. Mild exposure may cause only mucous membrane irritation, with initial symptoms mimicking an upper respiratory tract infection. Other symptoms may include nausea, vomiting, diarrhoea, headache, fatigue, and coughing, whilst more severe symptoms may include ataxia, paraesthesia, intention tremor, diplopia, and jaundice. Very severe cases may progress to acute pulmonary oedema, cardiac arrhythmias, convulsions, and coma. Renal damage and leukopenia may also occur. Exposure to 1400 mg/m^3 (1000 ppm) for 30 min may be fatal.

Death, which may be sudden, usually occurs within four days but may be delayed for one to two weeks. Post-mortem examinations have revealed focal myocardial infiltration and necrosis, pulmonary oedema and widespread small vessel injury. There is no evidence for cumulative effects from intermittent low-level exposure averaging 14 mg/m^3 (10 ppm) or less.

Chronic poisoning from inhalation or ingestion may cause toothache, swelling of the jaw, necrosis of the mandible (phossy jaw), weight loss, weakness, anaemia, and spontaneous fractures.

Laboratory findings may include abnormal liver function tests, acidosis, increased blood urea and bilirubin, haematuria, and proteinuria. Other diagnostic studies should include electrocardiogram, sputum, and differential white blood cell count.

Occasional cases of accidental exposure of the general population to phosphine have occurred in the region of fumigation operations and on board ships carrying cargoes capable of releasing phosphine. There have been many cases of accidental or suicidal ingestion of phosphide pesticides. Lethal doses vary, but most fatal cases have ingested more than 20 g zinc phosphide, and most of those who recovered had ingested less than 20 g phosphide. Pulmonary oedema and congestion and necrosis of the liver and kidneys are the principal pathological features in fatal cases. There have been occasional cases of fatal occupational exposure to phosphine, some of which have involved repeated exposures.

Various reports of adverse effects of occupational exposure at normal operational levels have been published, but in no case has the description of exposure or the control group been adequate to draw definite conclusions regarding the possibility of the adverse effects of phosphine at the higher current occupational exposure limits.

1.5 Evaluation

Phosphine and metal phosphides are toxic. They have a very limited distribution in the environment. Proper standards and procedures in their use prevent harmful effects. No significant global effects on the environment have resulted from the use of phosphine or phosphides.

2. IDENTITY, PHYSICAL AND CHEMICAL PROPERTIES, ANALYTICAL METHODS

2.1 Identity, Physical and Chemical Properties

2.1.1 Phosphine

2.1.1.1 Identity

Chemical structure:

```
H
|
P-H
|
H
```

Molecular formula: PH_3

Common synonyms: hydrogen phosphide, phosphorus trihydride, phosphoretted hydrogen, phosphane

2.1.1.2 Physical and chemical properties

Pure phosphine is a colourless gas at ambient temperature and pressure. Its relative molecular mass is 34.

The principal physical properties of phosphine are given in Table 1 (Lowe, 1971). The chemistry of phosphine has been extensively reviewed (Fluck, 1973).

Phosphine is odourless when pure, at least up to a concentration of 282 mg/m^3 (200 ppm), which is a highly toxic level. The odour of technical phosphine depends on the presence of odoriferous impurities and their concentrations and the odour threshold is usually in the range 0.14 - 7 mg/m^3 (Netherlands, 1984). The odour factors can easily be removed from phosphine (Dumas & Bond, 1974).

Pure phosphine has an autoignition temperature of 38 °C but, because of the presence of other phosphorus hydrides (particularly diphosphine) as impurities, the technical product often ignites spontaneously at room temperature (ACGIH, 1986). Phosphine forms explosive mixtures with air at concentrations greater than 1.8%.

Phosphine has intense ultraviolet absorption in the 185 - 250 nm (1850 - 2500 Å) region. It dissociates to phosphorus and hydrogen in contact with hot surfaces in the absence of oxygen (Lowe, 1971).

2

Table 1. Physical properties of phosphine and some phosphides[a]

	Phosphine	Trizinc diphosphide	Aluminium phosphide	Magnesium phosphide
Formula	PH_3	Zn_3P_2	AlP	Mg_3P_2
Physical state	gas	solid	solid	solid
Colour	none	grey	grey/yellow	grey
Melting point (°C)	-133.5	sublimes	> 1350[b]	> 750
Boiling point (°C)	-87.4	-	-	-
Spontaneous ignition temperature (°C)	38[d]	-	-	-
Lower explosive limit[c,e]	1.8%	-	-	-
Upper explosive limit[c]	unknown	-	-	-
Vapour density (air = 1)	1.17	-	-	-
Density (temp °C)		4.55 (13)	2.85 (25)	2.1

[a] From: Lowe (1971) and Wilson (1971).
[b] Some authorities give a temperature in excess of 2000 °C.
[c] In air, depending on presence of other gases and ultraviolet irradiation.
[d] The spontaneous ignition behaviour of phosphine is very unpredictable and though this figure is quoted, its validity will depend on the conditions of measurement.
[e] From: Anon. (1936).

Oxidation of phosphine involves a branching chain reaction. In air, the upper and lower explosion limits depend on the temperature, pressure, and proportions of phosphine, oxygen, inert gases and water vapour present, and also on the ultra-violet irradiation. In aqueous solutions, oxidation of phosphine results in the production of hypophosphorous acid.

An important reaction of phosphine is with metals, especially with copper or copper-containing alloys, which causes severe corrosion. The reaction is enhanced in the presence of ammonia (which is given off with phosphine during the decomposition of some fumigation tablets or pellets) and in the presence of moisture and salt. Copper-containing equipment, especially electrical apparatus, may be severely damaged by exposure to phosphine during fumigation.

Bond et al. (1984) reported a systematic study on the corrosion of metals by phosphine using a specially developed method in which the reaction of phosphine was determined by its depletion from an enclosed space. The authors confirmed the positive effects of phosphine concentration, oxygen and relative humidity on the rate of corrosion. The rate of reaction with copper exceeded by a factor of ten or more the rates of reaction with brass, silver, steel, aluminium, galvanized steel (various galvanizing processes), fine gold, nickel, lead solder, platinum, and iron powder. Eighteen carat gold jewellery

reacted at one-eighth of the rate of copper. The corrosion of copper appears to be similar to that produced by phosphoric acid and can be reduced by coating the copper with a saturated solution of sodium carbonate (1/3 rate) or, more effectively, with a saturated solution of potassium dichromate (1/44 rate). The type of polymeric spray used to moisture-proof ignition systems on internal combustion engines was also effective (1/7 rate).

Phosphine dissolves in water to form a neutral solution. Solubility is little affected by the pH. Dissolved phosphine reacts with hydrogen ions to form the phosphonium ion - $(PH_4)^+$. Its solubility in water at different temperatures and in various organic solvents is given in Table 2.

Table 2. Solubility of phosphine in water and organic solvents

	Temperature (°C)	Solubility[a]
Water	17	0.26
	24	0.24
Acetic acid	20	3.19
Acetone	22.4	4.45
Aniline	22	2.8
Benzene	22	7.26
Carbon disulphide	21	10.25
Carbon tetrachloride	20.5	4.19
Cyclohexane	18.5	7.47
Toluene	22.5	7.15
Trifluoroacetic acid	26	15.9

[a] Solubility is measured by the volume of phosphine (measured at the temperature of the study and at 1 atmosphere pressure) dissolved in one volume of solvent under a partial pressure of 1 atmosphere. From: Lowe (1971).

The technical product can contain impurities including higher phosphines (e.g., up to 5% diphosphine, P_2H_4) and substituted phosphines, which are responsible for the characteristic foul odour of phosphine which is often described as "fishy" or "garlicky" (Fluck, 1976) (section 9.1). Depending on the method of manufacture, other impurities can be methane, arsine, hydrogen, and nitrogen. Phosphine is often produced directly, when required, by the hydrolysis of metal phosphides or phosphonium iodide; it may also be supplied compressed in cylinders either pure or in various concentrations in nitrogen.

2.1.1.3 Conversion factors

At 20 °C, 1 ppm - 1.41 mg/m^3; 1 mg/m^3 - 0.71 ppm.
        1 ppm (as P) - 1.1 ppm (as PH$_3$).

2.1.2   *Metal phosphides*

The metal phosphides dealt with in this document are trizinc diphosphide (zinc phosphide), aluminium phosphide, and trimagnesium diphosphide (magnesium phosphide).

The principal physical and chemical properties of zinc, magnesium, and aluminium phosphides are given in Table 1 (Wilson, 1971).

Many metals have one or more phosphides that can be synthesized and have been characterized, but few are of commercial importance. The properties of tricalcium diphosphide (Ca_3P_2) are similar to those of trimagnesium diphosphide. Tricalcium diphosphide also reacts with excess white phosphorus to form calcium monophosphide (CaP) which, in turn, reacts with water to form diphosphine by the reaction $2CaP + 4H_2O = 2Ca(OH)_2 + P_2H_4$. Zinc diphosphide is formed when phosphorus vapour in nitrogen is passed over zinc at 700 °C. Trizinc diphosphide (Zn_3P_2, 1314-84-7, relative molecular mass = 258.1), commonly referred to as zinc phosphide, is used as a rodenticide and fumigant because of its reaction with acid to release phosphine. As a rodenticide, the acid in the stomach causes the hydrolysis. For fumigation, the acid has to be supplied. Since they hydrolyse in neutral moist conditions, aluminium or magnesium phosphide are preferred as fumigants.

Aluminium phosphide (AlP, 20859-73-8, relative molecular mass = 57.96) reacts with water to release phosphine. This reaction may be incomplete, possibly owing to the formation of a protective layer of aluminium hydroxide on the surface. Aluminium phosphide has been used as a rodenticide and a fumigant. It is the only compound of aluminium and phosphorus (Wilson, 1971; Van Wazer, 1982).

Trimagnesium diphosphide (Mg_3P_2, 12057-74-8, relative molecular mass = 134.87), commonly known as magnesium phosphide, is used as a pesticide and fumigant.

Zinc phosphide is available in bulk, typically to a specification of at least 80% Zn_3P_2, and as pastes containing 5% or 2.5% for use as a rodenticide by mixing in bait. Aluminium and magnesium phosphides are available as a number of commercial formulations. Aluminium phosphide formulations usually contain approximately 57% active ingredient and those of magnesium phosphide 34% active ingredient. Some registered trade names and presentations are:

Aluminium phosphide

Alutal
Celphide (tablets)
Celphine (tablets)
Celphos (tablets)

Delicia Gastoxin
Detia Gas-Ex-B (bags)
Detia Gas-Ex-P (pellets)
Detia Gas-Ex-T (tablets)
"L" fume (tablets)
Phosfume (pellets and tablets)
Phostek (pellets and tablets)
Phostoxin (pellets, tablets, "prepacs", rounds, strips)
Quickfos (pellets and tablets)
Zedesa (bags, pellets, tablets)

Magnesium phosphide

Detiaphos (pellets)
Mag-disc (plates)
Magtoxin (pellets, tablets, rounds)

Phostoxin (strip) formulations are designed to produce a controlled release of phosphine to achieve efficient fumigation with low operator risks. Some include other ingredients designed to reduce fire hazards. Depending on the storage conditions, the composition of metal phosphides and their formulations may change over time by hydrolysis and oxidation.

Metal phosphides are analysed by assaying the phosphine liberated by acid hydrolysis. After this preparatory step, the procedure is the same as for gaseous phosphine (Bontoyan, 1981; Terzic, 1981; Worthing & Walker, 1983).

2.2 Analytical Methods

2.2.1 *Gaseous phosphine*

Work-place air monitoring and fumigation control demand a measurement range from about 0.04 $\mu g/m^3$ to more than the lower explosion limit of about 25 000 mg/m^3. Thus, methods covering concentrations differing by six orders of magnitude are required.

Techniques are available that: (a) directly indicate the concentration in a grab sample or time-weighted average sample; (b) adsorb or absorb phosphine from a known volume of air for subsequent analysis directly or by desorption and gas analysis; and (c) give a continuous record of time-dependent concentrations. Some methods reviewed by Verstuyft (1978) are given in Table 3.

2.2.1.1 *Direct-indicating methods*

Phosphine can be detected by filter papers impregnated with a mixture of silver nitrate and mercury (II) chloride. The

Table 3. Methods of sampling and analysis[a]

Method	Range ppm	mg/m^3	Efficiency	Interference
Sampling				
Silver nitrate (0.1 N) impregnated paper	0.05 - 8.0	9.07 - 11.3	90%	
Ethanolic mercuric chloride	0.05 - 3.0	0.07 - 4.2		NH$_3$
Acidic potassium permanganate (0.1 N) impinger		0.01 - 0.05	100%	H$_3$S
Silver diethyldithiocarbamate (0.5%) bubbler	0.6 - 18	0.85 - 25	54 - 86.2%	H$_2$S,AsH$_3$, SbH$_3$
Mercuric chloride (0.5%) aqueous bubbler	10 - 28	14 - 28		AsH$_3$
Toluene impinger			41.5%	
Mercuric chloride (0.1%) conductance cell	0.05 - 2.5	0.07 - 3.5	88.0%	SO$_2$, H$_2$S, AsH$_3$, SbH$_3$
Silver nitrate impregnated silica gel	0.05 - 4.1	0.07 - 5.8	95%	H$_2$A, AsH$_3$
Auric chloride impregnated silica gel	0.01 - 1000	0.014 - 1 400	100%	AsH$_3$, SbH$_3$
Ethanolic mercuric chloride (0.1%)		0.0006	88 - 100%	AsH$_3$,SO$_2$,HCN, H$_2$S
Mercuric cyanide impregnated silica gel	0.014 - 1.18	0.02 - 1.7	80%	

Table 3 (contd.)

Method[b]	Sensitivity
Analysis	
Bendix phosphorus method/sulfur monitor	ns
HNU photoionization detector	0.1 - 100 ppm
Gas chromatography - FID, 3% TCP	ns
Gas chromatography - Therm. 4.5% QF-1	2 ppt
Gas chromatography - FPD, 3% Carbowax	0.5 ppt
Gas chromatography - Coulson	500 ppt
Gas chromatography - TC, porous beads	10 ppm
Gas chromatography - Therm. 30% Apiezon	50 ppb
Gas chromatography - FID, FPD, beta-ionization Porapak Q	20 ppb
Colorimetric - ammonium molybdate 720 nm	
Colorimetric ammonium molybdate 880 nm	
Colorimetric silver DDC 465 nm	0.05 ppm
Colorimetric silver sulfide 470 nm	0.02 ppm

Sensitivity (right column continued):

	$0.14 - 140$ mg/m^3
	3 pg/m^3
	0.7 pg/m^3
	0.7 µg/m^3
	14 mg/m^3
	70 µg/m^3
	30 µg/m^3
	10 mg/litre
	10 mg/litre
	70 µg/m^3
	30 µg/m^3

a   From: Verstuyft (1978).
b   DDC - diethyldithiocarbamate.
    FID = flame ionization detector.
    FPD = flame photometric detector.
    Therm = thermal detector.

ns = not stated.

method can be made semi-quantitative by appropriate configuration and measurement of stain-length (Brandon, 1983) or colour comparison (Hughes & Jones, 1963). Kashi & Muthu (1975) described a simple and sensitive method for detecting phosphine, using paper strips impregnated with dimethyl yellow, cresol red, and mercury (II) chloride in methanol. It is claimed that these paper strips have a better shelf-life and are more sensitive than paper strips impregnated with dimethyl yellow alone, or cresol red plus mercury (II) chloride. The strips need not be kept in air-tight containers or under controlled conditions of temperature and humidity.

Direct-indicating detector tubes are commercially available for spot sampling (using syringes and grab-samplers) and personal monitoring of occupational exposure (using portable pumps). Leesch (1982) compared the accuracy of various pump (5) and detector tube (7) combinations. Means of measured values for test concentrations varied from 59 to 256% of actual concentrations over the whole range of pump/tube combinations. Pumps and tubes from the same manufacturer gave mean values varying from 85 to 214% of the actual values. Measurements at low levels of phosphine in the range 2.8 - 7 mg/m^3 (2 - 5 ppm) with matched pumps and tubes from the same manufacturers resulted in mean values of 133%, 130%, and 128%. Using tubes from three manufacturer's and the pump from only one manufacturer gave mean values of 120%, 101%, and 90%. In most cases, the techniques over-estimated the concentrations. Clearly, inaccuracies of individual measurements are greater than those of these mean values and care is required in the choice of pumps and detector tubes, in their calibration, and in the interpretation of results obtained with them.

Other direct-indicating tubes of lower sensitivity are available for the measurement of the higher phosphine concentrations used in fumigation. Classical analytical techniques, such as oxidation to phosphate and the formation of the phosphomolybdate complex have been specially applied to fumigants (Kao, 1981). Shaheen et al. (1983) recently described the use of indicator tubes as passive dosimeters to record concentration·time product integrals. The stain-length was closely, but not linearly, related to the concentration·time product in the range 0 - 1400 mg·days/m^3.

2.2.1.2 Absorptive or adsorptive sampling and analysis

Gas chromatography is the most sensitive method for the determination of the phosphine content of air samples. Usually, samples are desorbed from a solid absorbent coated with mercury (II) cyanide, although samples taken in syringes, gas bags, or tonometers can be used. Microcoulometric and thermionic detectors have detection limits of 5000 and 20 pg, respectively.

The limit for flame photometric and argon and helium beta ionization detectors is 5 pg and that for mass spectrometry, 1 ng. Photoionization detection is also commonly used. Flame photometry combines both sensitivity and stability (Verstuyft, 1978).

Using a gas chromatograph equipped with a nitrogen/phosphorus-selective detector, Vinsjansen & Thrane (1978) were able to detect phosphine in the environment at a concentration of 0.04 $\mu g/m^3$.

Other techniques for estimating phosphine in air involve entrapping phosphine by adsorption or reaction with subsequent analysis of the desorbed or reacted sample. In a classical method (Furman, 1962), air containing phosphine is reacted with mercury (II) chloride, followed by the addition of potassium iodide and then excess standard iodine solution. Back-titration of the excess iodine with thiosulfate is used to quantify the phosphine.

Of the solid sorbents, acid-washed charcoal and silica gels coated with silver nitrate and potassium permanganate effectively collect phosphine but quantitative release has not been achieved. Mercury (II) cyanide on silica gel collects phosphine quantitatively and holds 80% of the phosphine over 2 weeks storage. The phosphine is released for gas chromatographic analysis by treatment with alkaline sodium borohydride solution (Barrett & Dillon, 1977) or is oxidized (using a hot acid permanganate solution) to phosphate, which is measured using the phosphomolybdate colorimetric technique (US NIOSH, 1979). It should be noted that many commercially available sorbents are unsuitable (Dumas & Bond, 1981).

Liquid impingers/bubblers containing a variety of solutions can be used to collect and react phosphine for quantification by colorimetry/spectrophotometry, by conductance, or by potentiometric titration. Classical colorimetric techniques are the development of the red-orange complex with silver diethyldithiocarbamate, which can be measured at 465 nm, or the oxidation by permanganate to phosphate which is reacted with a solution of ammonium molybdate in concentrated sulfuric acid, extracted with toluene-isobutanol, reduced with tin (II) chloride and measured as the phosphomolybdate complex at 625 nm (US NIOSH, 1979). The first method suffers from arsine interference and the second also measures any phosphorus species that are oxidized to phosphate by oxalic acid/permanganate treatment.

The quantity of phosphine bubbled through a solution of mercury (II) chloride and reacting:

$$PH_3 + 3HgCl_2 = P(HgCl)_3 + 3HCl$$
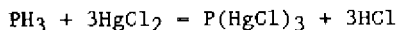
can be measured by the change in electrical conductivity using a conductance cell or by potentiometric titration of the HCl with NaOH (Verstuyft, 1978).

2.2.1.3 Continuous methods

There are directly indicating continuous samplers in which phosphine-containing gas is passed through a paper tape impregnated with a silver nitrate-containing mixture, which develops a colour related to the phosphine concentration. The tape can subsequently be passed through a reader-recorder to produce a concentration versus time plot (McMahon & Fiorese, 1983). Other continuous monitors draw air at a metered rate through detectors using either flame photometry or photo-ionization. The detection limit of infrared spectroscopy is about 0.4 mg/m^3 (0.3 ppm) but this is barely sensitive enough for monitoring occupational exposure (Webley et al., 1981). Continuous direct estimation of phosphine can also be made using the property of chemiluminescence in ozone and measuring the emission at 550 - 560 nm using a photomultiplier tube (Boubal et al., 1981). A portable quadrupole mass spectrometer has been used (Arnold & Robbiano, 1974).

2.2.2 Residues

Fumigated foodstuffs may contain gaseous phosphine (adsorbed and interstitial) and residual aluminium or magnesium phosphide. Different techniques for the determination of residues may measure the phosphine or the phosphide residues.

Interstitial and adsorbed phosphine can be purged by nitrogen and trapped in reagents for classical analysis or on adsorbents for chromatographic analysis (Dumas, 1978; Nowicki, 1978; Saeed & Abu-Tabanja, 1984).

Total phosphine and phosphide is measured by extraction of the fumigated stored product with silver nitrate (Rangaswamy, 1984; Rangaswamy & Muthu, 1985), or with sulfuric acid (Nowicki, 1978; Saeed & Abu-Tabanja, 1984). The silver nitrate forms a chromophore, which is measured spectrophotometrically at 400 nm. Following sulfuric acid extraction, headspace gas can be analysed spectrophotometrically for phosphine.

Saeed & Abu-Tabanja (1984) reported substantial differences between the purge-and-trap method and the sulfuric acid method. Some of these differences may have been due to the presence of powdered aluminium phosphide in the sample, but some may have been due to incomplete desorption during purging. The sulfuric acid method was preferred because it also measures the capacity of the product to release phosphine and this is of more biological significance than the measurement of free phosphine only.

Robinson (1972) reported the measurement by neutron activation analysis of phosphorus-containing residues in filter paper fumigated with Phostoxin[R] and demonstrated a net gain in phosphorus content.

Robison & Hilton (1971) described the estimation of phosphine/phosphide residues in zinc phosphide-treated sugarcane by extraction in sealed flasks into a mixture of aqueous acid and toluene followed by the gas chromatographic analysis of toluene.

2.2.3   *Metal phosphides*

Hydrolysis of metal phosphides with acids yields phosphine, which can be measured by any of the methods already described. Terzic (1981) has described a method in which the evolved phosphine is absorbed in silver nitrate to form silver phosphide, which is oxidized to silver phosphate, precipitated as magnesium ammonium phosphate, and converted by ignition to magnesium pyrophosphate, which is weighed. A single conversion factor applied to this weight gives the quantity of phosphide in the sample.

3. SOURCES OF HUMAN AND ENVIRONMENTAL EXPOSURE

3.1 Natural Occurrence

Phosphine is extremely rare in nature. It occurs transiently
in marsh gas and other sites of anaerobic degradation of
phosphorus-containing matter, and the equilibrium of reactions
in which it participates favour its oxidation (Ciba, 1978).
Although phosphorus could be expected to occur naturally as a
phosphide, the only phosphide in the earth's crust is found in
iron meteorites as the mineral schreibersite $(Fe,Ni)_3P$. in which
cobalt and copper may also be found (Van Wazer, 1961).

3.2 Man-Made Sources

3.2.1   Production levels and processes

Apart from natural sources, atmospheric phosphine results
from emissions and effluents from industrial processes and from
the use of phosphides as rodenticides and fumigants. Unexpected
focal release of phosphine may occur due to the action of water
on phosphides present as impurities in some industrial
materials. Amounts of phosphate arising from atmospheric
phosphine are insignificant in comparison with the amounts of
phosphate added to the environment from sewage, agricultural
run-off, and industrial and urban effluents.

3.2.1.1 World production figures

It is difficult to quantify the production of phosphine,
since much of it is manufactured and used in relation to another
process. Though some phosphine is supplied in cylinders, it is
often produced, as and when required, by hydrolysis of a metal
phosphide. Phosphine is also produced as a by-product or evolved
incidentally in various industrial processes.
A main use of phosphine is as a dopant in the electronics
industry. A total of 42 electronics companies used 6 million
litres of phosphine gas mixtures in various concentrations in
1979, probably equivalent to some 300 000 litres of pure
phosphine (LaDou, 1983). In 1981, 90% of speciality gases for
the electronics industry were produced by six manufacturers
(LaDou, 1983).The annual growth rate for the use of dopants by
the electronics industry has been estimated to be 30% per annum
(SRI, 1982). By contrast, volumes more than twice this may be
produced and used as a chemical intermediate in a single plant,
and even greater amounts are used for fumigation of stored
products. For example, in the Federal Republic of Germany in
1975, 37 000 kg (approximately 28 million litres) of phosphine

were used for fumigation, though it decreased to 10 000 kg
(approximately 7.5 million litres) in 1977 as a result of the
introduction of alternatives (Noack & Reichmuth, 1982a).

3.2.1.2 *Manufacturing processes*

Phosphine is manufactured by the hydrolysis of aluminium
phosphide or magnesium-aluminium phosphide, or by the electro-
lysis of phosphorus in the presence of nascent hydrogen (Boenig
et al., 1982). It is formed as a co-product in the manufacture
of hypophosphites by the reaction of white phosphorus with
alkali where conditions can be established so that the
phosphorus-steam reaction yields phosphine. Phosphine is
produced as a by-product in the manufacture of acetylene,
through the hydrolysis of calcium carbide, if this contains
calcium phosphide as an impurity. It is also evolved from
phosphorus furnaces (Al'zhanov et al., 1983), impurities in
ferrosilicon alloy (Anon., 1956; Lutzmann et al., 1963),
machining of spheroidal graphite iron (Bowker, 1958; Mathew,
1961), steel pickling (Vdovenko et al., 1984), and other
metallurgical operations (Habashi & Ismail, 1975). Zinc
phosphide has been prepared by reducing trizinc phosphate with
hydrogen at 600° C, by passing phosphorus vapour over zinc at
400 °C, or by direct reaction between amorphous phosphorus and
powdered zinc under pressure or heat. Aluminium phosphide and
magnesium phosphide are produced in similar ways or by exchange
reactions between aluminium or magnesium and a heavy-metal
phosphide (Wilson, 1971).

3.2.2 *Uses*

A ternary compound, magnesium aluminium phosphide, in which
aluminium and magnesium are present in the ratio by weight 1:3,
is used in sea flares and as an intermediate for the local
formation of phosphine by hydrolysis.

As a chemical intermediate, phosphine is used in the
synthesis of organophosphines and organic phosphonium
derivatives. Alkyl phosphines may be made by additional
reactions across an olefinic double bond. Using Grignard
reactions, both aryl and alkyl phosphines can be made from the
appropriate alkyl or aryl halide and PCl_3. Organophosphines are
used in oil-additive and pharmaceutical applications. Phos-
phonium compounds are made by the addition of P-H bonds across a
carbonyl function with acid catalysis. For example, the reaction
of phosphine with formaldehyde, in the presence of hydrochloric
or sulfuric acid, forms the corresponding salt of tetrakis-
(hydroxymethyl)-phosphonium which is employed in the manufacture
of polymers used in the flame-retardant treatment of cotton
fabrics.

As a fumigant in pest control, phosphine is invariably produced at the site of fumigation by the hydrolysis of a phosphide, usually of aluminium or magnesium. As a dopant in the electronics industry, phosphine is used in high purity at low concentrations in nitrogen.

Zinc phosphide is used principally in the form of a 2.5% or 5% paste as a rodenticide incorporated in food (10%) as a bait (WHO/FAO, 1976). There are other formulations in which powdered zinc phosphide is incorporated, possibly with a zinc salt to inhibit phosphine formation before use, and histamine, or a histamine releaser, to stimulate acid secretion (and therefore phosphine production) in the stomach of rodents taking the bait. One formulation includes a binder and is encapsulated in a form that enables it to be included with grain as bait (Degesch GmbH, 1978). Aluminium and magnesium phosphides are used in powder form or in formulations as a source of phosphine for the fumigation of storage facilities and stored products, to prevent spoilage by a wide range of pests. In general, for the satisfactory hydrolysis of the phosphide, the material being fumigated should have a moisture content of 10% or more.

4. ENVIRONMENTAL TRANSPORT, DISTRIBUTION, AND TRANSFORMATION

4.1 Transport and Distribution Between Media

4.1.1   Air

Frank & Rippen (1986) studied the disappearance of phosphine from air released following the aeration of fumigated premises. The most important chemical reactions are with the HO· radicals, which are usually present in abundance in the atmosphere due to the reaction of ozone with water, a reaction enhanced by impurities such as NO_x:

$$H_2O + O_3 + 2\ \epsilon^- \rightarrow 2\ HO\cdot\ + O_2$$
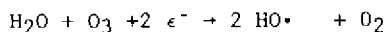
The following reactions of HO· and phosphine may occur:

$$PH_3 + HC\cdot \rightarrow H_2O + PH_2\cdot$$

$$PH_3 + HO\cdot\ \rightarrow\ HOP\cdot H_3$$
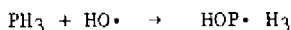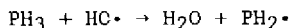
This reaction is dependent on phosphine concentration and is very rapid with a reaction rate constant at room temperature of about 1.5×10^{-11} $cm^3/mol\cdot sec$ (Fritz et al., 1982; Becker et al., 1984). Direct reactions with ozone are quantitatively unimportant, since reaction with HO· occurs before PH_3 can reach the ozone-rich upper atmosphere. With the usual concentrations of HO·, the half-life of phosphine in air is about 28 h. Sunshine increases the HO· concentration and may reduce the half-life to less than 5 h. Direct photolysis cannot be expected with phosphine (Calvert & Pitts, 1966). The eventual oxidation product of phosphine will be phosphorus oxyacids and inorganic phosphate, which will be deposited and contribute to the nutritive environment of soils and surface waters.

Hilton & Robison (1972) studied the disappearance of phosphine from dry tubes sealed by a rubber membrane and from similar tubes containing a small quantity of water. Phosphine completely disappeared from the dry tubes in 40 days, but the presence of water in the others reduced the rate of disappearance. However, possible losses through, or by adsorption on, the membrane were not quantified and no information was given regarding illumination of the tubes.

4.1.2   Soil

Hilton & Robison (1972) introduced phosphine at 1.4 g/m^3 (1000 ppm) (as P) in the headspace of tubes containing 3 types of soil at 5 moisture levels, 0%, 25%, 50%, 75%, and 100%

saturation. It was not stated whether the soils had been
sterilized. Phosphine disappeared within 18 days from all air-
dried soils, whereas up to 40 days was necessary for disappear-
ance from moisture-saturated soils. Quantities of phosphorus
recoverable as phosphate from the soils after incubation for 40
days varied widely with different soil types and reached about
70% of total phosphine in a slightly acidic soil, containing
12% - 15% organic matter content at 25% moisture saturation.
Variation in phosphate recovery probably reflected rates of
diffusion of phosphine into the soil matrix as a function of
moisture content, as well as differences in the efficiency of
different soils with different moisture contents as oxidizing
substrates for phosphine. Clearly, in time, soils are able to
entrap the phosphine in the air in contact with them and oxidize
it to orthophosphate.

Hilton & Robison (1972) studied the fate of zinc phosphide
and phosphine in the soil-water environment. The results of
preliminary experiments showed that phosphine was undetectable
in headspace gases over soil containing zinc phosphide at
1.4 mg/m^3 and 14 mg/m^3 (1 and 10 ppm) (as PH$_3$). Zinc phosphide
was added at 1000 ppm (as P) to each of 3 types of soil at 0%,
25%, 50%, 75%, and 100% water saturation in sealed bottles which
were incubated at 27 - 28 °C for up to 34 days. Headspace gases
were sampled periodically and the appearance and disappearance
of phosphine followed. At the end of the phosphine evolution
period, acetic acid was added to the samples to hydrolyse
phosphide to phosphine with minimum conversion to phosphate.
The headspace was analysed for phosphine after heating for 3 h
and cooling. It was then flushed with nitrogen to remove
phosphine, and sulfuric acid was added to the soil to extract
the phosphate. The amount of phosphine in the headspace
increased with increasing moisture content up to 50%. The data
were sufficient to estimate the extent of hydrolysis of zinc
phosphide to phosphine. After 34 days of incubation, residual
phosphide was detectable only in the dry (about 35% of original)
and 25% saturated (about 10% of original) soils. Over 80% of
the phosphide phosphorus was recovered as phosphate, except in
the case of dry soil samples.

4.1.3   *Aquatic environment*

The hydrolysis of zinc phosphide was negligible in a variety
of surface waters, tap water, and ocean water over periods of up
to 11 days. Only in a buffered solution at pH 4 did significant
hydrolysis take place (Hilton & Robison, 1972). The authors
concluded that zinc phosphide released or carried into streams
or ocean water would not decompose rapidly. Bottom or suspended
sediments would be likely to decompose zinc phosphide with the

formation of phosphine or phosphoric acid under anaerobic and aerobic conditions, respectively.

4.1.4   *Vegetation, wildlife, and entry into the food chain*

Robison & Hilton (1971) studied phosphine residues in sugarcane *in vitro* using $^{32}PH_3$. About 30% of the $^{32}PH_3$ reacted irreversibly to form water-soluble compounds of phosphorus while another 10% remained irreversibly bound to the fibre. It was suggested that the water-soluble compounds were phosphorus oxyacids and a portion of the acids may have formed insoluble iron or aluminium salts in the fibre.

Effects on viable seeds, (section 6.4.3) suggest little effect on plant metabolism.

The only significant exposure of wildlife to phosphine or metal phosphides is through their use as pesticides; domestic animals may also be accidentally exposed in this way. Zinc phosphide baits are known to be highly palatable for rodents. Acceptability of bait by non-target species depends on the presentation (Janda, 1973) and the precise location in relation to natural feeding sites (Chentsova, 1972). Persistence of phosphine or phosphides in the carcasses of poisoned animals is low and their carcasses are not toxic when eaten by other animals (Kozhemyakin et al., 1971; Schitoskey, 1975).

Various studies have been undertaken on the effects of feeding fumigated diet to experimental animals. Kadkol & Jayaraj (1967) reported the effects of feeding phosphine-fumigated rice to albino rats for 12 weeks. The only dose-related effects were slight increases in liver and kidney weights in male rats. Hackenberg (1972) reported a study on the effects on Wistar rats (30 male and 30 female in both test and control groups) of a standard laboratory chow treated for 72 h with Phostoxin[R] uniformly distributed in the food at 48 mg/kg for the first 16 weeks and thereafter at 90 mg/kg (corresponding to 10 times the recommended treatment level). Following treatment, food was mixed and aerated for 1 h before determination of phosphine, which was found to be in the region of 1 mg/kg food, including any residues of pellets. The methods of administration and storage of food were probably responsible for further losses of phosphine after the aeration period. No differences attributable to diet were observed in behaviour, development, body weights, food consumption, or in blood and urine composition (at any stage in the study), and in gross or microscopic pathology between test and control animals.

Cabrol Telle et al. (1985) undertook a similar study of the effects of feeding a phosphine-fumigated test diet to 30 Sprague Dawley rats of both sexes for 2 years, with a control group fed an identical but non-fumigated control diet. The test diet had been fumigated by storage in phosphine at 2820 mg/m^3 (2000 ppm)

for at least 6 months. Just before consumption, it was aerated
for 48 h and supplemented with a balanced vitamin preparation
(also added to the non-fumigated control diet). The average
residue level in the fumigated diet was less than 5 mg/kg.
There were no differences between test and control groups
attributable to the consumption of a fumigated diet in terms of
weight gain, food consumption, plasma chemistry, haematology,
urinalysis, behaviour, growth, survival, organ weights, histo-
pathology, tumour incidence, or carcass analysis.

It is unlikely, therefore, that the use of phosphine or
phosphides results in food residues that are of any toxico-
logical significance.

4.2 Biotransformation

There have been no studies on the biotransformation of
phosphine or metal phosphides. Energy considerations suggest
that phosphine is liable to be oxidized to phosphate in
biological systems (Ciba, 1978). There is no suggestion of
bioaccumulation or biomagnification.

4.3 Ultimate Fate

The ultimate fate of phosphine and the phosphide moiety of
metal phosphides is the formation of phosphate.

Disposal of phosphide-bearing wastes is regulated in many
countries. Effluent phosphine is usually burned and the
resultant gases can be scrubbed to remove phosphorus oxides and
oxy-acids before discharge into the atmosphere. Zinc phosphide
formulations and baits in some countries are regulated as
hazardous wastes, for instance when their concentration exceeds
10% (US EPA, 1983). Deep burial is the preferred method of
disposal.

IRPTC (1985) gives the following recommendations:

Aluminium phosphide

"Allow it to react slowly with moisture out in the open,
taking precautions to see that the poisonous gas (phosphine) is
dissipated"[a]

---

[a] It should be added that precautions should be taken to
ensure that the area is inaccessible to children and ani-
mals. The IRPTC comments "Phosphine gas creates no permanent
environmental hazard because it is eventually converted to
harmless phosphoric acid and water". An alternative is
admixture with an inert dry diluent and burning at a temper-
ature greater than 1000 °C with effluent gas scrubbing.
These recommendations also apply to magnesium phosphide.

Phosphine

"Surplus gas or leaking cylinders can be vented slowly to air in a safe, open area or gas can be burnt off through a suitable burner in a fume cupboard".

5. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

5.1 Environmental Levels

5.1.1 Air, water, and soil

Phosphine and metal phosphides have only been detected in the general environment in relation to the recent use of metal phosphides in pest control and in relation to a number of industrial activities.

Arendt et al. (1979) studied open-air concentrations in the vicinity of a silo at successive stages during fumigation with phosphine and aeration. Within 20 m of the silo wall, concentrations reached a peak of 3 mg/m^3 (2.15 ppm) during the fumigation phase and averaged 0.37 mg/m^3 (0.26 ppm) during the aeration phase, but the maximum value after 3 h of aeration was 0.06 mg/m^3 (0.04 ppm). At distances greater than 20 m from the silo, the concentration was at all times less than 0 05 mg/m^3 (0.035 ppm). Measurements at 7 locations from < 5 to 60 m from the silo, during aeration, showed that the highest concentration 20 - 30 min after the start of aeration was 0.1 mg/m^3 (0.08 ppm), while other values were about half of this or less. Seven hours after the commencement of aeration, concentrations were uniformly less than 0.014 mg/m^3 (0.01 ppm).

Reichmuth et al. (1981) reported similar results. They found that open air concentrations immediately adjacent to the outer walls of buildings under phosphine fumigation reached 280 mg/m^3 (200 ppm (v/v)), but at a distance of > 10 m from the buildings, all concentrations except one were less than 0.14 mg/m^3 (0.1 ppm (v/v)). However, there are isolated reports of persons in the neighbourhood of fumigated stores being affected by phosphine (Gessner, 1937; Hallerman & Primela, 1959; Sayvetz, 1982); in the 2 earlier reports, effects were fatal (section 9.2).

These results indicate that fumigation operations contribute only locally and transiently to atmospheric phosphine exposure. The volumes released in industrial operations (section 3.2.1.2) are much smaller and are therefore of less significance in relation to atmospheric concentrations, except at the immediate work station.

5.1.2 Food and feed

5.1.2.1 Residue values

Because of the techniques of analysis used, residues of phosphine or metal phosphides are usually measured together and reported as total phosphine or phosphorus. The results indicate

that residues in fumigated foods are negligible at 0.01 mg/kg (0.01 ppm) or less (Dieterich et al., 1967). Urga (1983) reported residue levels in a variety of stored grains or pulses (minimum moisture content 7.6%) that had been fumigated by inserting Phostoxin[R] at 3 tablets per tonne under PVC sheeting for 3 - 8 days, followed by a minimum of 2 months' aeration. Phosphine was measured by bromine oxidation and the phosphomolybdate colorimetric method. Average phosphine residues measured as phosphorus in different stored grains or pulses are shown in Table 4.

Table 4.   Average PH$_3$ residues (as phosphorus)[a]

	Number of samples	mg/kg average (range)
Wheat	15	0.05 (0.01-0.08)
Chick-peas	9	0.07 (0.02-0.12)
Barley	3	0.11
Maize	12	0.06 (0.01-0.11)
Sorghum	3	0.11

[a]   From: Urga (1983).

Cabrol Telle et al. (1985) (section 4.1.4) found that residue levels after prolonged fumigation at 2820 mg/m^3 (2000 ppm) were on average 0.005 mg/kg (0.005 ppm), but the phosphine was apparently not produced from mixing a metal phosphide in the diet. Hackenberg (1972) mixed Phostoxin[R] pellets with basic diet and determined residues included hydrolysis of unreacted phosphide to phosphine. Residues were generally under 0.75 mg PH$_3$/kg feed. Since the fumigation was at 10 times the recommended rate, this result is comparable with a residue level of less than 0.1 mg/kg (0.1 ppm) at normal doses. Samples of 2 of the batches of feed contained exceptionally high levels of 1.3 and 7.5 mg PH$_3$/kg feed, respectively, presumably as a result of residual phosphide in the samples selected. Singh et al. (1983) fumigated pulses with Phosfume[R] tablets at the recommended dosage (2 tablets/tonne) and also at 2 and 4 times the recommended dosage. After fumigation for 5 days, phosphine residues decreased exponentially with a half-life that depended on the type of pulse and the fumigation dose; the half-lives varied from 0.66 to 1.33 days. In each case, residues fell below the limit of detection of 0.001 mg/kg

(0.001 ppm) in less than 3 days at normal fumigation rates, and
in less than 6 days at 4-fold fumigation rates.

Breyer (1973) described field studies using magnesium and
aluminium phosphide formulations as the source of phosphine for
the treatment of wheat. After 12 days exposure at 17 °C with a
concentration of 6 g phosphine/tonne, the wheat contained
0.009 mg phosphine/kg in the case of magnesium phosphide and
0.012 mg/kg for aluminium phosphide.

Residues in wheat, millet, milled rice, soya beans, and
azuki beans were studied after exposure to 5640 mg phosphine/m^3
(4000 ppm) at 25 °C (Sato & Suwanai, 1974). After 12 days of
fumigation, the residue levels were 0.46, 1.16, 0.34, 0.18, and
0.24 mg phosphine/kg, respectively.

When wheat was fumigated with phosphine at 5 mg
phosphine/kg, the residue level after 4 days of aeration was
0.2 μg/kg (0.2 ppb); after 220 days of aeration, the level
dropped to 0.004 μg/kg (0.004 ppb) (Dumas, 1986).

Phosphine levels in Iraqi dates fell rapidly within 24 h
after fumigation, but residues persisted for at least 9 days.
Higher residue levels were found with storage at low
temperature. For example, more than 1 mg phosphine/kg (1 ppm)
could still be detected after 10 days in dates stored at 4 °C
(Alomar & Al-Bassomy, 1984).

5.1.2.2 Factors affecting residue levels

The factors affecting phosphine residues in hazel nuts, soya
beans, and wheat that had been fumigated at 20.5 mg
phosphine/litre, for periods of 1 and 14 days, were studied
(Noack et al., 1984a). Fumigated food was then aerated for 5
weeks in a layer about 20 mm deep in a wire basket, open to the
air at 70% relative humidity and at temperatures of -18 °C,
+5 °C, +20 °C, and +35 °C. Residue levels were studied as a
function of fumigation duration and aeration temperature and
duration. Residues decreased approximately exponentially with
time, more rapidly at higher temperatures and with the shorter
fumigation period. Residues were much more persistent in hazel
nuts than in wheat, with intermediate levels in soya beans.
Temperature dependence was much less marked with hazel nuts than
with the other 2 foods. Residue levels immediately at the end
of fumigation were not much higher after 14 days of fumigation
than after 1 day, presumably because equilibrium was achieved
within the shorter fumigation period. Concentrations in wheat
and soya beans after 3 days aeration or more were about 2.5
times higher when fumigated for 14 days than when fumigated for
1 day. For hazel nuts, the corresponding ratio was about 5.
Times for phosphine residues to reach 0.1 mg/kg (0.1 ppm) and
0.01 mg/kg (0.01 ppm) are given in Table 5 (Noack et al., 1983,
1984a).

Table 5. Approximate time necessary for phosphine residues to reach
0.14 mg/kg and 0.01 mg/kg in different stored products at various storage
temperatures and durations of fumigation[a]

Food type	Fumigation (20.5 mg pH₃/ litre) duration (days)	Target residue level mg/kg	Time to reach residue level (days) Temperature (°C)			
			-18	+5	+20	+35
Hazel nuts	1	0.1	35+	35+	35+	20
		0.0	35+	35+	35+	35+
	14	0.1	35+	35+	35+	35+
		0.01	35+	35+	35+	35+
Soya beans	1	0.1	35+	10	4	3
		0.01	35+	35+	9	6
	14	0.1	35+	35+	18	5
		0.01	35+	35+	35+	16
Wheat	1	0.1	20	< 1	< 1	< 1
		0.01	35+	25	4	3
	14	0.1	35+	15	7	2
		0.01	35+	35+	28	8

[a] Adpated from: Noack et al. (1984a).

The shorter the period of fumigation, the lower the dose,
and the higher the storage temperature, the more rapid is the
decomposition of phosphine. Crops with high contents of fat and
protein appear to retain a higher level of phosphine than those
with a high water content. The residue level for hazel nuts
fumigated at 20.5 mg phosphine/litre for 14 days was 15.2 mg/kg,
whereas for wheat similarly fumigated only 0.6 mg phosphine/kg
was detected (Noack et al., 1983).

A further study described in the same report showed that
after fumigation for 10 days (5.6 mg phosphine/litre) and active
ventilation at 8, 50, or 100 litres/h for 12 days during
aeration, residue levels were independent of airflow.

The results indicate that the decrease in phosphine residues
is limited by the rate of diffusion within the food substance,
the rate of desorption from its surface, or chemical decompo-
sition, rather than by interstitial diffusion; the rate-limiting
process is temperature dependent.

Noack et al. (1984a,b) were able to describe the behaviour
of phosphine residues in semi-empirical mathematical models
which were in good agreement with experimental data. In a
further study (Noack & Wohlgemuth, 1985), the effects of non-
constant phosphine concentrations on fumigation residues were
examined. There was a correlation between the final residues
and the concentration of phosphine used but during fumigation

concentrations rose to a peak then declined. Residues in samples of foodstuffs removed from fumigation at a particular concentration in the rising phase decreased more rapidly than in samples removed from fumigation at the same concentration in the declining phase. Residue levels in samples of food fumigated at constant concentration reached a maximum later than the time at which the maximum fumigation concentration had been achieved. These results could be explained on the basis of the sorption and diffusion of phosphine and the authors concluded that residues are minimized by fumigation at low concentrations (a few hundred mg/m^3) over a long period (2 - 3 weeks).

Robison & Hilton (1971) measured phosphine residues in sugarcane harvested from fields treated 4 times with zinc phosphide as a rodenticide at 5.6, 11.2, or 56 kg/ha. Sugarcane was analysed for phosphine after 7 and 110 days using extraction by aqueous acid and toluene and gas chromatographic analysis. In general, residue levels were less than 0.01 mg/kg (0.01 ppm) and in no case did the levels exceed 0.1 mg/kg (0.1 ppm). At harvest, 110 days after application, sugarcane from a wet location did not contain any residues of phosphine, whereas that from a dry location still contained up to 0.032 mg/kg (0.032 ppm) (highest application).

In a study by Robinson & Bond (1970), ^{32}P-labelled phosphine derived from labelled aluminium phosphide was used to investigate the presence of phosphorus residues after treatment of wheat and flour. The radioactive residue in wheat and flour could not be removed by thorough aeration or by heating at baking temperature. It was shown to be largely water soluble and paper chromatography identified the main products as hypophosphite and phosphite. It was concluded that the oxidation of phosphine to the lower oxyacids of phosphorus was mainly a surface phenomenon and that, in the normal course of air oxidation, all residues would eventually appear as ortho-phosphate. Deposition of oxidation products of phosphine also occurred on glass and other surfaces.

Laboratory studies by Disney & Fowler (1972a) showed that wheat exposed to phosphine levels (3 mg/litre) several times higher than those normally used, at 25 °C for 5 days (10% moisture content) and 14 days (19.7% moisture content), contained residue levels varying from 1.7 mg/m^3 (1.2 ppm) in the first case to 25.9 mg/m^3 (18.4 ppm) in the second, demonstrating the important effect of water content as well as exposure period in determining residues. Autoradiography of sections of whole grain showed that most of the radioactivity was present in the outer layers and crease, and that 70% of the residues were extractable with hot water. Tkachuk (1971, 1972) showed that, in wheat, flax, and rapeseed, approximately 50% of the phosphine formed non-phosphine residues, which were distributed in wheat in the bran (85%), endosperm (14%), and germ fractions. Eleven

percent of the residues were water soluble and appeared to be hypophosphite and pyrophosphate. The remainder may have included insoluble aluminium salts. It was suggested that this did not represent the behaviour of non-radioactive phosphine, but further studies confirmed the isotopic studies (Disney & Fowler, 1972b; WHO/FAO, 1972). The adsorbed non-phosphide residues were oxyacids of phosphorus and of no toxicological significance (Robinson, 1972; US EPA, 1986).

Some national and international standards and recommendations for phosphine residues in food and feeds are given in Table 6.

5.1.3   Tobacco and consumer products

Residues of phosphine in fumigated tobacco were reported to be not greater than 8 mg/kg (8 ppm) (Childs et al., 1969; Kuhn et al., 1971). Experimental fumigation of tobacco with ^{32}P-phosphine similarly led to residual ^{32}P-phosphorus of less than 5 mg/kg (5 ppm) (Underwood, 1972). However, this may not qualitatively simulate the residues found in practice since the ^{32}P-phosphorus contained in tobacco was predominantly in the form of phosphate (which is a normal constituent). This contrasts with the results of a similar study (Kuhn et al., 1971) where the majority of residual ^{32}P-phosphorus was as phosphine and approximately 25% was non-volatile. In Underwood's study, fumigated tobacco was made into cigarettes that were smoked in a smoking machine and the ash, mainstream smoke, and butt analysed for ^{32}P. The ^{32}P was found to be confined to the ash as phosphate. Winks (1970) reported that tablet and pellet residues from tobacco fumigated with Phostoxin[R] contained 3.4% and 2.9%, respectively, of unreacted aluminium phosphide. According to subjective evaluation of the fumigated leaf after aeration for 2 days there were no off-odours or the odour of phosphine.

5.1.4   Terrestrial and aquatic organisms

Phosphine or phosphides have only been reported in terrestrial or aquatic organisms due to deliberate or accidental administration of phosphine or phosphides.

Muscle tissue of rabbits and chickens poisoned with zinc phosphide did not contain detectable residues (Kozhemyakin et al., 1971; Bubien et al., 1974). Intestines and liver may contain phosphides (Bubien et al., 1974; Meredith, 1981) but the whole carcass of a kangaroo rat killed by a dose of zinc phosphide was not toxic to the kit fox, despite the fact that the amount ingested by the rat was 3 times the amount expected to be lethal for the fox, on a body weight and LD_{50} of zinc phosphide for the fox (Schitoskey, 1975). Rats, cats, and mice

Table 6. Various national and international standards for phosphine
residues in food

Organization (reference)	Commodity	Level of phosphine ppm	Level of phosphine mg/kg
Joint FAO/WHO Food Standards Programme Codes Alimentarius Commission			
Codex Alimentarius (1986)	Maximum Residue Limits (MRL)		
	Cereal grains		0.1
	Flour and other milled cereal products, dried foods, fruit and vegetables, spices, cocoa beans, nuts, peanuts and breakfast cereals		0.01
Canada cited in US EPA (1986)	Raw cereals, soy beans, processed foods, animal feeds	0.1	0.1
India Government of India: Department of Health (1976)	Whole food grains	0.05	0.05
(cited by Smugh et al. (1983))	Milled food grains	0.01	0.01
USA US EPA (1985a)	Tolerances for residues applicable to either aluminium or magnesium phosphide: almonds, barley, cashews, cocoa beans, coffee beans, corn, cottonseed, dates, filberts, millet, nuts (Brazil, pistachio), oats, peanuts, pecans, popcorn, rice, rye, flower seed, sesame seed, sorghum, soybeans, sunflower seed	0.1	0.1
	Tolerances for residues of aluminium phosphide: vegetables (seed and pod, except soybeans)	0.1	0.1
	Walnuts, wheat	0.01	0.01
	Tolerances for residues of magnesium phosphide: avocados, bananas, Chinese cabbage, egg-plants, endive (escavole) grapefruit, kumquats, lemons, lettuce, limes, mangoes, mushrooms, oranges, papayas, peppers, persimons, pimentos, plantains, salsify tops, tangelos, tangerines, tomatoes	0.01	0.01
USSR Kagan (1985)	Cereals	0.01	0.01

fed for 30 days on meat from the carcasses of rabbits and
chickens killed with zinc phosphide did not show any specific
toxic effects (Kozhemyakin et al., 1971).

5.2 General Population Exposure

5.2.1 *Access to phosphine and phosphides*

Zinc phosphide pastes, though legally restricted in many
countries, are available without restriction for use as
rodenticides in others, and there are many reports of their
ingestion in suicide cases. Magnesium and aluminium phosphide
formulations are also restricted and not normally available to
the general public. In most countries, there is strict control
of fumigation to prevent public exposure to phosphine, and
guidelines for safe fumigation are available. These measures,
combined with negligible ambient air concentrations, effectively
limit exposure.

5.2.2 *Residue exposure*

Residue levels in fumigated foods are generally regulated at
0.1 mg/kg (0.1 ppm) or sometimes 0.01 mg/kg (0.01 ppm). Even
among populations whose diet is mainly derived from stored pro-
ducts, the daily intake would be unlikely to exceed 0.1 mg/day,
even if the phosphine or phosphides survived cooking. Under
most circumstances daily intakes would be several orders of
magnitude lower than this, because of lower residues, the small
fractions of the diet that have been fumigated, and losses of
fumigant in storage and food preparation.

5.2.3 *Subgroups at special risk*

No subgroups of the general population have been identified
to be at special risk from phosphine or phosphides except for
children, who might find and eat bait containing phosphides.

5.3 Occupational Exposure During Manufacture,
Formulation, or Use

Occupational exposure can be divided into 4 general
categories: (a) workers producing phosphine and phosphides; (b)
workers in operations that can release phosphine, e.g., welding,
metallurgy, semi-conductors (c) fumigators and pest-control
operatives; and (d) transport workers, e.g., drivers, seamen.
Both the exposure patterns and the potential for control of
exposure differ from case to case.

Exposure to phosphine and phosphorus oxides, which occurs
during the manufacture of metal phosphides, varies according to

the method of manufacture. High levels of exposure may occur in
the direct methods involving the reaction of red phosphorus with
powdered metal. Freshly produced powdered phosphides evolve
phosphine at fairly high rates initially, producing warehouse
concentrations ranging from 0.4 to 1.6 mg/m^3 (0.3 - 1.13 ppm).
Concentrations of 2.5 and 4 mg/m^3 (1.8 and 2.9 ppm), necessit-
ating the use of personal respiratory protection, have been
reported in production areas (Jackson & Elias, in press).

The use of zinc phosphide in the preparation of poisoned
bait would not be associated with significant phosphine
exposure, because of the stability of zinc phosphide in neutral
media. There is no literature regarding occupational exposure
to phosphine during the formulation of preparations of aluminium
phosphide for use in fumigation. Jones et al. (1964) reported
that atmospheric levels to which operatives were exposed while
adding tablets of formulated aluminium phosphide to wheat were
undetectable. Levels encountered when stores were re-entered for
loading or turning were much higher, ranging from 18 to 35 mg/m^3
(13 - 25 ppm). In the USA, an estimated one million workers are
at risk of inadvertent exposure. Ten thousand of these workers
are engaged in the grain freight trades where accidental
exposure is a considerable hazard (US NIOSH, 1977).

Exposure to phosphine has also been described in the
operation of acetylene generators (Harger & Spolyar, 1958);
exposure to a level of 11 mg/m^3 for up to 2 h per day has been
estimated. Exposure to phosphine can also occur in the
production of phosphorus (Beloskurskaya, 1978), in the
conversion of white phosphorus to red phosphorus where levels of
up to 0.5 mg/m^3 (0.35 ppm) were found (Jackson & Elias, in
press), and also in steel pickling (Vdovenko et al., 1984).

The carriage of ferrosilicon as a badly ventilated cargo,
particularly in barges, can release phosphine accidentally by
the action of water on calcium phosphide, one of the impurities
present. The transport of ferrosilicon requires precautionary
measures to prevent dangerous emissions of phosphine (Hunter,
1978). High ship-board concentrations of 1.4 - 3 mg/m^3 (1 -
2 ppm) in living quarters and about 8.5 mg/m^3 (6 ppm) in the
hold were reported in a vessel transporting ferrosilicon
(Lutzmann, 1963) and a case of fatal poisoning in the same
environment was reported by Ziemer, (1963). Jones et al.
(1964) reported phosphine concentrations of 5 - 13 mg/m^3 (3.7 -
9 ppm) in the holds of ships transporting fumigated wheat.
Phosphine fumigation of agricultural commodities on board ship
during transit is common practice in some parts of the world.
Countries that import grain normally accept fumigation en route
and some nations require such treatment (Davis, 1986). Guide-
lines for safe fumigation practice have been established by the
United Nations International Maritime Organization (IMO, 1980)
and include the regular monitoring of air in the living quarters

to ensure that there is no hazard for crew members from the accumulation of phosphine.

Many metals contain phosphorus in small amounts, and phosphine can be generated in a variety of metallurgical processes. Cole & Bennett (1950) reported the presence of about 4 mg phosphine/m^3 in the immediate vicinity of magnesium powder after its manufacture from bulk metal containing 0.0038 - 0.0093% phosphorus. Ferrosilicon alloys in contact with water form phosphine in amounts that vary with the silicon content (which correlates with the phosphide levels) so that alloys containing 30 - 60% silicon can release 10 - 230 litres phosphine per tonne (Delomenie, 1933). Concentrations of phosphine close to the tool cutting edge in the machining of spheroidal graphite iron were reported by Mathew (1961) to vary between 0.01 and 6.5 mg/m^3 (0.01 and 4.6 ppm). Concentrations in the breathing zone ranged from 0.01 to 1.3 mg/m^3 (0.01 - 0.95 ppm) and the average was 0.9 mg/m^3 (0.65 ppm). Earlier, Bowker (1958) had reported similar results and found that phosphine concentrations fell from 8.5 mg/m^3 (6 ppm) at a distance of 1 cm from the tool to slightly over 1 mg/m^3 (0.8 ppm) at 8 cm from the tool.

Although phosphine is used extensively in semi-conductor manufacture, there are no published figures for occupational exposure in this industry. Since installations are invariably modern and phosphine is used under precise and strictly controlled conditions, frequently diluted with inert gases, exposure of workers is not likely to be high. There are no published data relating to exposure to phosphine in the synthesis of organophosphine or phosphonium derivatives.

Some recommended occupational exposure limits for phosphine in various countries are given in Table 7.

Table 7. Occupational exposure limits for phosphine in various countries

Country	Legal	mg/m^3	Comments	Source
Australia	Rec[a]	0.4	TLV TWA[c]	Approved occupational health guide threshold limit values (1983)
Belgium	Rec[b]	0.4	TLV	Threshold limit values (1978)
Bulgaria	Reg	0.1	MPC[c]	Official journal, 88 (1971)
Czechoslovakia	Reg	0.1 0.2	MAC TWA MAC Ceiling value	Hygienicke predpisy Ministerstva zdravotnictvi CSR/Hygienic regulations of Ministry of Health of CSR, 58 (1985) Uprava No. 7/1985 Vest. Mz SSR Reg. V Ciast.24/1985 ZB/ Regulation of Ministry of Health of SSR No. 7/1985 Reg. In Section 25/1985 ZB
Finland	Reg	0.1	MPC TWA	Luftfoereningar paa Arbets- platsen (Air pollutants at the workplace) (1982)
German Democratic Republic	Reg	0.1 0.3	TWA STEL[c]	Maximale zulässige Konzentrationen Gesundheits- gefärdender Stoffe in der Luft am Arbeitsplatz (Maximum allowable concentrations of noxious substances in the Atmosphere (1983)
Germany, Federal Republic of	Rec	0.15 0.3	8-h TWA 5-min STEL	Deutsche Forschungsgemein- schaft, "Maximale Arbeits- platzkonzentrationen", xxi, 16, (1985) German Association for Research, "Maximum Workplace Concentrations", xxi, 16, (1985)
Italy	Rec	0.4	8-h TWA	Valori limitati ponderati (Threshold limit values) (1978)
Hungary		0.1		ILD (1980)
Netherlands	Rec	0.4 1.5	TWA STEL	Nationale MAC-Lijst (National MAC-List) (1985)
Poland	Reg	0.1	Ceiling value	Ordinance of the Minister of Labour, Wages & Social Affairs 22 Dec. (1982)

Table 7 (contd).

Country	Legal	mg/m^3	Comments	Source
Romania	Reg	0.2 0.5	TWA Ceiling value	Ordinance of the Ministry of Health, 60 (1975)
Sweden	Reg	0.4	1-day TWA	Arbetarskyddsstyrelsens Foerfattiningssamling, (1984, 1985) 10 (1984)
Switzerland	Reg	0.15	TWA	Zulässige werte in Arbeitsplatz (permitted values in the workplace (1984))
United Kingdom	Rec	0.4 1.0	8-h TWA 10-min TWA	Health and Safety Executive EH40/85 (1985)
USA	Rec	0.4 1.0	TWA STEL	ACGIH (1986) ACGIH (1986) US CFR (1981)
USSR	Reg	0.1	Ceiling value	Gosndarstrennyi Standart SSSR/State Standard of USSR, 12.1.005 (1976)
Yugoslavia	Reg	0.1	MAC TWA	Ordinance, 24-3698/1 (1971)

[a] Rec. = Recommendation.
[b] Reg. = Registered regulatory requirement.
[c] TLV = Threshold limit value.
    TWA = Time-weighted average.
    MPC = Maximum permitted concentration.
    MAC = Maximum allowable concentration.
    STEL = Short-term exposure limit.

6. KINETICS AND METABOLISM

6.1 Insects

Uptake of phosphine by insects is rapid in the presence of oxygen, but little absorption occurs in low or zero oxygen atmospheres, and the insecticide potential is thus reduced. Over 100 mg phosphine/kg body weight may be absorbed by insects at high dosage rates, and some insects continue to absorb phosphine for long periods, even after knock down (Bond et al., 1969). Phosphine taken up by insects is not removed by ventilation of volatile phosphine derivatives or phosphine itself but is apparently excreted slowly (Price et al., 1983) Most of the ^{32}P, derived from $^{32}PH_3$, taken up by insects is found in the soluble fraction of the cells (Robinson & Bond, 1970); in deproteinized tissue extracts, the radiolabel is present mainly as hypophosphite and orthophosphate (Price et al., 1982).

6.2 Mammals

There have been no formal studies of the toxicokinetics of phosphine and metal phosphides in mammals.

6.2.1 Absorption

6.2.1.1 Inhalation

Because systemic toxic effects are detectable after short exposures to very low atmospheric concentrations of phosphine, inhaled phosphine is generally considered to be readily absorbed through the lungs. Hydrolysis suggests that aluminium or magnesium phosphides deposited on the moist surfaces of the respiratory tract would release absorbable phosphine but zinc phosphide, which hydrolyses significantly only under acid conditions, would be stable for some time. However, the transfer of a proportion of inhaled zinc phosphide to the intestinal tract by the lung particulate clearance mechanisms would permit hydrolysis to phosphine by gastric acid as well as absorption of the zinc phosphide. The lungs also absorb particulates and, as it is known that zinc phosphide is absorbed intact from the gut (Meredith, 1981), inhaled zinc phosphide dust might be absorbed directly via the respiratory tract and then hydrolysed in the tissues.

6.2.1.2 Dermal

An acute dermal LD_{50} for zinc phosphide in rabbits (2000 - 5000 mg/kg body weight) has been reported, but no details are

available. Hydrolysis of aluminium and magnesium phosphides on the skin would lead to the evolution of gaseous phosphine, which could then be absorbed by inhalation, but this is unlikely with zinc phosphide, and the LD_{50} result suggests toxicity by dermal absorption (US EPA, 1983). In general, dermal absorption of phosphine and metal phosphides is insignificant.

6.2.1.3 Oral

The oral route is not relevant to the absorption of gaseous phosphine. Ingestion of zinc phosphide results in detectable amounts of acid-hydrolysable phosphide in the liver of rats (Curry et al., 1959; Meredith, 1981). Human ingestion of tablets containing aluminium phosphide yielded evidence of acid-hydrolysable phosphide in blood and liver (Chan et al., 1983). These results indicate that metal phosphides can be absorbed directly. Meredith (1981) showed that when zinc phosphide was administered to rats by gavage in corn oil, the fraction recovered as phosphine from the air of the metabolic chamber increased with the dose administered up to about 25% at a dose of 4 mg/rat (approximately 20 mg/kg). The possibility that this phosphine derived directly from the alimentary canal and not via systemic absorption and subsequent exhalation could not be excluded. For comparison, *in vitro* hydrolysis of zinc phosphide for 12 h yielded 7.1% as phosphine at pH 4 and 38.8% at pH 2 and, unless there is some special enzymic mechanism, acidic gastric conditions afford the only opportunity for such a level of hydrolysis. The efficacy of zinc phosphide as a rodenticide depends on the absorption of phosphide or phosphine after oral administration. Meredith (1981) also showed that about 4 times as much phosphide was recoverable from the liver when zinc phosphide was administered in corn oil rather than in water, suggesting greater absorption of unhydrolysed material.

McGirr (1953) speculated that commercially available zinc phosphide might consist of 2 fractions that are attacked at different rates in the gastrointestinal tract. This was based on the fact that phosphine is produced rapidly and yet zinc phosphide is recoverable from the liver of poisoned animals. However, this did not consider the possibility that storage in fat might lead to a fraction of the zinc phosphide being more slowly hydrolysed and thus more readily absorbed unchanged (Meredith, 1981).

6.3 Distribution

Inhaled phosphine produces neurological and hepatic symptoms suggesting that it reaches the nervous system and liver (Childs & Coates, 1971). Ingested phosphides have been shown to reach the liver and blood in rats and human beings (Curry et al.,

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1959; Meredith, 1981; Chan et al., 1983). On the other hand, muscle tissue of animals poisoned with supralethal doses of zinc phosphide did not contain detectable levels of phosphine or phosphide and did not produce toxic effects when fed to test animals (Kozhemyakin et al., 1971; Bubien et al., 1974). Curry et al. (1959) reported the presence of acid-hydrolysable phosphide in the kidney as well as in the liver of a fatal case of zinc phosphide poisoning.

6.4   Metabolic Transformation

Metal phosphides are hydrolysed to phosphine and the corresponding metal cation (Van Wazer, 1982). In rats, phosphine that is not excreted in the expired air is oxidized and appears in the urine, chiefly as hypophosphite and phosphite (Curry et al., 1959; Meredith, 1981). Meredith (1981) also reported an unidentified metabolite, detectable by paper chromatography and distinct from pyrophosphate and metaphosphate. The fact that (a) phosphine is incompletely oxidized; and (b) the proportion of an administered dose that is eliminated as expired phosphine increases with the dose suggests that the oxidative pathway is slow.

6.5   Elimination and Excretion

Meredith (1981) administered zinc phosphide suspended in corn oil, by gavage, to Wistar rats (body weight approximately 200 g) and measured the phosphine concentrations in a metabolic chamber over the following 12 h. After doses of 0.5, 1, 2, 3, and 4 mg, the proportions of the administered doses as phosphine in air were 1.5%, 1.7%, 3.2%, 15.6%, and 23.5%, respectively, but some or much of this could have been derived from faeces or intestinal gas rather than by absorption and exhalation. Paper chromatographic estimation of hypophosphite and phosphite in the urine of rats dosed with zinc phosphide showed gradients positively correlated with dose, but the proportion of the administered dose excreted was not quantified. Hypophosphite is the principal urinary excretion product (Curry et al., 1959).

6.6   Retention and Turnover

Virtually all the phosphine in the air of metabolic chambers housing Wistar rats, dosed orally with zinc phosphide, had disappeared after 12 h (Meredith, 1981), and this period corresponds with the duration of symptoms following sub-lethal doses of zinc phosphide. The same author determined the acid-hydrolysable phosphide in the liver of single rats given a diet containing 15 mg zinc phosphide/kg diet. The liver of a rat fed for 15 days contained nearly twice as much as that of a rat fed

for 7 days. However, this limited study cannot be considered to
provide evidence for accumulation of metal phosphides.

6.7 Reaction with Body Components

Phosphine reacts with some haem- and copper-containing
proteins *in vitro*. Insect cytochrome c oxidase is reduced and
not reoxidizable in air (Rajak, 1971). Mammalian haemoglobin
does not react with phosphine in the absence of oxygen, but
oxyhaemoglobin is converted through Fe^{3+}-containing compounds to
a verdichromogen-like material (Trimborn & Klimmer, 1962). The
nature of the reaction between phosphine and these proteins is
uncertain, but oxyhaemoglobin is denatured and a variety of
enzymes are inhibited by reaction with phosphine.

7. EFFECTS ON ORGANISMS IN THE ENVIRONMENT

7.1 **Microorganisms**

Ruschel & Da Costa (1966) treated seeds of French bean
(*Phaseolum vulgaris* L.) with aluminium phosphide, using 3
Phostoxin[R] tablets/m[3]. Treated and untreated control seeds,
inoculated with a pure culture of the nitrogen-fixing bacterium
Rhizobium phaseoli, were sown in pots containing sandy soil
(pH 4.5) and maintained under greenhouse conditions. Plants,
sampled during the flowering period, showed no differences in
the numbers of nodules formed by the bacteria.

Natarajan & Bagyaraj (1984) reported laboratory studies on
the effects of phosphine fumigation (100 mg/litre for 15 days at
an unspecified temperature) on the microbial load (fungi,
bacteria, and actinomycetes) of blackgram and fieldbean seeds
with different moisture contents. Total microbial populations
were estimated using the dilution plate technique. Results are
given in Table 8. It was concluded that phosphine was effective
on actinomycetes. The effects of phosphine on each type of
microorganism depended on the seed type and the moisture
content.

Table 8 Microbial load[a] per g seeds[b]

	Moisture level (%)	Fungi (x100)	Bacteria (x1000)	Actinomycetes (x100)
Blackgram				
Control	8	11.54	25.86	0.41
	12.5	51.96	267.20	0.30
	15	61.92	358.80	0.35
Phosphine-fumigated	8	.95	13.89	0.33
	12.5	1.16	34.23	0.06
	15	.88	69.71	0.09
Fieldbean				
Control	8.6	1.80	21.86	0.82
	12.5	3.79	35.77	0.38
	15	5.29	67.68	0.27
Phosphine-fumigated	8.6	1.32	6.17	0.22
	12.5	1.02	12.13	0.29
	15	.60	18.10	0.20

[a] Microbial counts after 15 days fumigation at 100 mg phosphine/litre.
[b] From: Natarajan & Bagyaraj (1984).

Lück et al. (1984) reported the effects of phosphine fumigation over 6 days with variable concentrations equivalent to at least 85 mg·h/litre on microorganisms in a variety of dairy products. There were no differences in bacterial, yeast, or mould counts between fumigated products and non-fumigated control products handled similarly but without fumigation.

The data of Rolulich & Menser (1970) (section 6.4.3), indicate that phosphine damages and inhibits the respiration and growth of microflora in moist stored wheat grains.

7.2 Aquatic Organisms

An LC_{50} for phosphine for the frog from a 30-min exposure was reported to be 0.56 mg/litre. The LC_{50} for a 15-min exposure was 0.84 mg/litre (WHO/FAO, 1980). The aluminium phosphide formulation known as Detia Pellets[R] is reported to be highly toxic for the bluegill sunfish, with a 96-h LC_{50} of 0.178 mg/m^3 (0.126 ppm) (Freyberg, 1979).

7.3 Terrestrial Organisms

7.3.1 Insects and mites

Insects are a major target organism of phosphine as a pesticide.

In practice, fumigation is carried out by the distributed insertion of formulated aluminium phosphide tablets into the bulk of the stored product, so that the phosphine evolved by hydrolysis with the moisture content of the product percolates the bulk and is retained by the gas-tight design of the store or the use of an impermeable covering. Typically, the phosphine concentration rises to a maximum and then decays over a longer period. The phosphine dose is often described in terms of the duration of exposure and estimate of concentrations or by the concentration·time product. This is calculated from the area under the curve of concentration versus time $\int C_t \cdot dt$ or, approximately, $\Sigma (C_t \times I_t)$, where C_t is the concentration at time t and I_t is the interval over which C_t is a sufficiently accurate measure of the actual concentration.

Because the susceptibility to phosphine of different developmental stages varies, it is possible that some individuals will be at a less susceptible stage at the time of the peak phosphine concentration and may survive fumigation. Moreover, acclimatization may occur with rising concentrations. The dose or concentration that is lethal for a particular species is therefore a complicated function of the concentration and its time-course (Reichmuth, 1985). Laboratory fumigation at constant concentrations is not a sound basis for the calculation of doses for practical fumigation (Reichmuth, 1986). Moreover,

the effects of any concentration•time product will be influenced by the temperature and other aspects of the gaseous environment, such as the partial pressure of oxygen which may fall to a low level in stores of metabolically active pulses. It has been shown that insects are tolerant to phosphine in a nitrogen- or oxygen-deficient atmosphere (Kashi, 1981a,b). By influencing the rate of hydrolysis of the phosphide, the relative humidity or moisture content affects the concentration•time course and thus independently influences the toxicity for pests.

In spite of the complexity of these relationships, the concentration•time product is frequently calculated as an index of the dose. However, though the concentration•time product is related to mortality with some insecticides, there is a deviation with phosphine. Longer exposures are much more effective in achieving control than shorter ones with the equivalent concentration•time product (Hole et al., 1976; Kashi, 1982; Winks, 1984, 1985). Winks (1985) suggested that part of the explanation of the relative tolerance to high dosages was narcosis, which reduced absorption to sub-lethal doses, but his experimental data neither confirmed nor refuted his hypothesis.

In addition to these uncertainties in determining the effectiveness of fumigation, there is considerable variation in susceptibility to phosphine among target organisms. Grain mites are tolerant (Bowley & Bell, 1981) to concentration•time products 2 or 3 orders of magnitude greater than those effective for many insect species. Hole (1981) reported that 3 strains of *Sitophilus granarius* and *Rhyzopertha dominica* were relatively tolerant compared with 3 other species of *Coleoptera*. The data also indicated considerable variation between strains within a species (Table 9).

Adu & Muthu (1985) reported a 6-fold variation in the LC95 values for different life cycle stages of *Callosobruchus chinensis* L. with the susceptibility of developmental stages decreasing in the order: larva > adult > pupa > egg. However, Bell et al. (1984) found that the eggs of *Trogoderma granarium* were much more susceptible than the larva and Winks (1981) found that the early pupae of *Tribolium castaneum* were substantially more tolerant than the larvae.

Larvae of many species can enter a state of suppressed development known as diapause, so that the insects can survive under unfavourable conditions. Cox et al. (1984) found that diapause increased tolerance to phosphine in the larvae of *Ephestia kühniella*. Diapausing larvae of *Trogoderma granarium* survived a 5-day exposure to a concentration•time product of 164 mg•h/litre, while laboratory stock larvae were killed by a 4-day exposure to 120 mg•h/litre (Bell et al., 1984).

Table 9. Concentration·time products of phosphine for 100% mortality and maximum survived doses (MSD) for various numbers of strains of 7 species of stored-product Coleoptera[a]

Species	Number	Stage	Temper-ature (°C)	RH (%)	LD_{hi}(mort %) (mg·h/ litre)	MSD[b] (mg·h/ litre)
Sitophilus granarius	8	all	25	70	46 (100)	22
	3	all	25	70	> 46 (100)	46
Sitophilus zeamais	5	all	25	70	46 (100)	22
	5	all	25	70	> 46 (100)	46
Sitophilus oryzae	1	all	25	70	22 (100)	9
	6	all	25	70	46 (100)	22
	4	all	25	70	> 46 (100)	46
Rhyzopertha dominica	5	all	25	70	9 (100)	< 9
	4	all	25	70	22 (100)	9
	1	all	25	70	46 (100)	22
Oryzaephilus surinamensis	7	all	25	70	> 4 (100)	> 4
Tribolium castaneum	17	all	25	70	9 (100)	4
	9	all	25	70	> 9 (100)	9
Tribolium confusum	3	all	25	70	> 22 (100)	> 22
	1	all	25	70	> 9 (100)	> 9

[a] From: Hole (1981).
[b] Maximum phosphine concentrations were about 0.28 mg/litre and the dose varied with the duration of the exposure period. In many cases, 100% lethality was not achieved at the highest dose used.

A further variant in the response of insects to phosphine has been the development of resistant strains (Champ & Dyte, 1976). Attia (1984) found that phosphine resistance in *Tribolium castaneum* and *Rhyzopertha dominica* was sometimes coincident with, but not otherwise related to, resistance to 6 other insecticides. The phosphine-resistant strains had resistance factors of about 5 and 10, respectively. Nakakita & Winks (1981) reported that ratios of LC_{50} and $LC_{99.9}$ values for various stages of resistant and susceptible strains of *Tribolium castaneum* varied from 0.1 to about 6. In general, all stages of a resistant species have a higher tolerance than the equivalent stage of the susceptible strain, suggesting a metabolic difference persisting through metamorphosis. Price (1984) reported that adult insects of a resistant strain absorbed much less radioactive phosphine than their susceptible counterparts and retained normal respiratory activity. Living resistant

insects absorbed less phosphine than dead insects, and the author concluded that the active exclusion of phosphine as a possible mechanism for resistance was supported by the data.

Noack & Reichmuth (1981) determined a toxic threshold value for phosphine in *Drosophila melanogaster* of about 1.4 mg/m^3 (1 ppm) in air, using the criterion of a statistically just-significant increase in mortality rate. The value obtained is comparable to the threshold for toxicity in human beings and other mammals.

Lethal doses of phosphine for a variety of stored-product pests are given in Tables 9, 10, and 11. Childs (1972) reported that all stages of the cigarette beetle were killed by fumigation with phosphine at concentrations that never exceeded 500 ppm (< 33.6 mg·h/litre) for 48 h, in field tests using transport containers at an unspecified temperature and humidity.

Winks (1970) inoculated tobacco bales with the tobacco beetle *Lasioderma serricorne* and studied the mortality after phosphine fumigation (20 tablets Phostoxin^R/28 m^3). Temperatures ranged from 11 to 25 °C and the relative humidity from 65 to 82%. Peak concentrations were about 300 mg/m^3. Mortality was assessed after a 7-day recovery period, and the number of progeny from eggs laid during fumigation was assessed 8 weeks after fumigation. Mortality was 100% and the number of progeny was 0.2% compared with unfumigated controls.

7.3.2 *Birds*

Ikeda (1971) studied the lethality of zinc phosphide for the quail and reported an oral LD$_{50}$ of 35 mg/kg body weight; there was a reduction in egg laying at 3.5 mg/kg. Shivanandappa (1979) found that the acute oral LD$_{50}$ and LD$_{90}$ of zinc phosphide in poultry were 25 and 31 mg/kg body weight, respectively. Treatment of chickens with encapsulated doses of 14, 21, 31.5, or 47.2 mg/kg body weight, daily for 4 weeks, resulted in deaths at all doses. Mortality was 12% at the lowest dose and 100% at the highest dose, where death occurred within 6 - 18 h of administration of the first dose. Hill et al. (1975) studied the effects of zinc phosphide administered in the diet for 5 days to mallard ducks. The dosing period was followed by 3 days of untreated feed. The zinc phosphide concentration of 1285 mg/kg diet was calculated to produce 50% mortality. Though the acute oral LD$_{50}$ for most avian species is generally in the range of 20 - 100 mg zinc phosphide/kg body weight, it has been reported that chickens fed 12 - 16 mg/kg body weight displayed toxic symptoms, including reduced red-cell counts, reduced haemoglobin concentration, and leukocytosis, 1 - 1.5 h after dosing (Kozhemyakin et al., 1971). Baxland & Gordon (1945) administered oral doses of zinc phosphide ranging from 15 to

Table 10. Concentration·time products for high and 50% lethality for different stages of common insect pests under specified conditions

Species	Stage	Temperature (°C)	Relative humidity (%)	Time (h)	LDhi (mort %) (mg·h/litre)	LD50 (mg·h/litre)	Reference
Trogoderma granarium	DP[a] larva	20	60	120	75.0 (100)	-	Bell et al. (1984)
	DP larva	20	60	96	118.5 (100)[b]	-	Bell et al. (1984)
	egg	20	60	72	50.0 (100)	-	Bell et al. (1984)
	egg	20	60	48	22.6 (100)	-	Bell et al. (1984)
Callosobruchus chinensis L.	egg	25-27	70-75	24	3.127 (95)	0.0708	Adu & Muthu (1985)
	larva	25-27	70-75	24	0.556 (95)	0.1622	Adu & Muthu (1985)
	pupa	25-27	70-75	24	2.542 (95)	0.684	Adu & Muthu (1985)
	adult	25-27	70-75	24	0.823 (95)	0.240	Adu & Muthu (1985)
Rhyzopertha dominica	adult	27	65	20	0.5 (99.9)	0.126	Attia & Greening (1981)
Tribolium castaneum	adult	27	65	20	0.32 (99.9)	0.166	Attia & Greening (1981)
Tribolium confusum	adult	27	65	20	0.58 (99.9)	0.26	Attia & Greening (1981)
Ephestia kuhniella	DP larva	10	70	< 20	79.6[b] (100)	29-42	Cox et al. (1984)
	larvae	10	70	< 20	79.6[b] (100)	21[b]	Cox et al. (1984)

Table 10 (contd).

Species		Stage	Temper-ature (°C)	Relative humidity (%)	Time (h)	LD$_{hi}$(mort %) (mg·h/litre)	LD$_{50}$ (mg·h/litre)	Reference.
Tribolium	S[c]	15-day larva	25	ns	6	0.52 (99.9)	0.17	Nakakita & Winks (1981)
castaneum	R[d]	15-day larva	25	ns	6	2.57 (99.9)	0.34	Nakakita & Winks (1981)
	S	20-day larva	25	ns	6	0.29 (99.9)	0.13	Nakakita & Winks (1981)
	R	20-day larva	25	ns	6	0.74 (99.9)	0.18	Nakakita & Winks (1981)
	S	pre-pupa	25	ns	6	0.74 (99.9)	0.17	Nakakita & Winks (1981)
	R	pre-pupa	25	ns	6	4.22 (99.9)	0.33	Nakakita & Winks (1981)
	S	early pupa	25	ns	6	11.94 (99.9)	4.2	Nakakita & Winks (1981)
	R	early pupa	25	ns	6	-	-	Nakakita & Winks (1981)
	S	mid-pupa	25	ns	6	-	0.69	Nakakita & Winks (1981)
	R	mid-pupa	25	ns	6	-	21.0	Nakakita & Winks (1981)
	S	late pupa	25	ns	6	1.12 (99.9)	0.09	Nakakita & Winks (1981)
	R	late pupa	25	ns	6	0.80 (99.9)	0.438	Nakakita & Winks (1981)
Trichoplusia		egg	24	ns	2	0.56 (100)	-	Leesch (1984)
ni Hübner		larva	24	ns	0.67	0.28 (100)	-	Leesch (1984)
		pupa	24	ns	2	0.56 (100)	-	Leesch (1984)

a Diapausing.
b Approximate values.
c Sensitive strain.
d Resistant strain.

ns = not stated.

Table 11. Concentration·time products for 100% mortality and maximum
survived doses (MSD) for three species of grain-infesting mite[a]

Species	Stage	Temper- ature (°C)	Relative humidity (%)	Time (days)	LD_{hi}(mort %) (mg·h/ litre)	MSD (mg·h/ litre)
Tyrophagus longior	adult	10	60-70	21	450 (100)	190
Acurus siro	adult	10	60-70	14	310 (100)	150
Glycyphagus destructor	adult	10	60-70	14	310 (100)	150

[a]   From: Bowley & Bell (1981).

400 mg/kg body weight to single domestic hens of 2 species. All
the birds receiving more than 30 mg/kg body weight died while
those receiving 20 mg/kg or less survived.

Klimmer (1969) exposed 3 turkeys to phosphine at a concen-
tration of 211 mg/m^3 and 6 hens at 224 mg/m^3 in an acute
inhalation study. The turkeys exhibited apathy, restlessness,
dyspnoea, and tonic-clonic convulsions, and died after 68, 74,
and 80 min, respectively. When examined, organs were congested
with oxygenated blood. Hens exhibited tonic-clonic convulsions
and died after an average of 59 min (range, 50 - 64 min). Their
organs were also congested with oxygenated blood.

7.3.3   *Mammals*

7.3.3.1 *Non-target species*

The acute oral LD_{50} of zinc phosphide in the kit fox was
calculated to be 93 mg/kg body weight (Schitoskey, 1975). Dogs
(which may also be a target species) were reported to be killed
by a dose of 100 mg zinc phosphide/kg body weight when fasted
for 24 h, but not when fed (Aminzhanov, 1972).

The acute toxicity of zinc phosphide for large domestic
animals was reported by Fitzpatrick et al. (1955). Single doses
were administered by capsule, stomach tube, or mixed with the
feed.   Deaths occurred as shown in Table 12.

McGirr (1953) quoted unpublished work by Fitzpatrick
indicating that the dose of zinc phosphide producing toxic
effects in cats and dogs lies between 20 and 40 mg/kg body
weight.

Table 12. Mortality of domestic mammals given single oral doses of zinc
phosphide by capsule, stomach tube, or mixed with the feed

Dose (mg/kg body weight)	Mortality				
	Cow	Goat	Sheep	Pig	Total
20	0/1	1/2	.	0/3	1/6
30	-	-	0/1	0/3	1/4
40	1/1	0/1	1/2	1/2	1/6
50	-	.	-	0/1	1/1
60	-	1/1	1/1	1/2	1/4

7.3.3.2 Rodents

The results of acceptance studies of 4 commercially avail-
able wax baits containing zinc phosphide on Norwegian rats and
house mice were reported by Marshall (1981). Each animal was
offered the bait and a palatable non-toxic alternative. While
one bait achieved an acceptance of 48.8% with 100% mortality in
rats and 38.5% acceptance with 100% mortality in mice, the other
baits achieved less than 6.5% acceptance and mortality rates did
not exceed 50%. Gill & Redfern (1983) reported 1 day "no-
choice" feeding tests, using standard procedures, which studied
the effects of zinc phosphide administered to Shaw's gerbil
(*Meriones shawi*) at 10, 20, 30, 40, or 50 g/kg diet (EPPO,
1975). There was very little reduction in average food consump-
tion at 40 and 50 g/kg diet and none at 10, 20, and 30 g/kg
diet. Zinc phosphide at 10 g/kg resulted in 20% mortality but
dietary concentrations of 20 - 50 g/kg resulted in 70 - 100%
mortality. In palatability tests, consumption of both poisoned
(zinc phosphide at 20 or 50 g/kg) and plain baits was very low,
presumably because the poison acted rapidly and interfered with
feeding. Survival of 8/10 animals given zinc phosphide bait
(20 g/kg) and 2/10 given 50 g/kg may indicate that these animals
could detect the poison and avoided consuming a lethal dose.
Similar results were reported at doses of 40 and 50 g/kg for the
golden hamster (*Mesocricetus auratus* Waterhouse) (Bradfield &
Gill, 1984). Sridhara (1983), in a study on the Indian gerbil
(*Tatera indica cuvieri* Waterhouse), reported that phosphide
incorporated in ragi (*Eleusine coracana*), a preferred food, at
0.5 g/kg induced aversion to this and enhanced the consumption
of maize (*Zea mais*) offered as an alternative. Administra-
tion of sub-lethally baited food on the fifth day only of the
study also induced aversion to the same food baited with a
different poison, and to a new food substituted for the
preferred food. However, it was not reported how long the
animals that had been given the poisoned preferred food on day 5

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exhibited subsequent aversion to the same unpoisoned food. This makes it difficult to evaluate the study.

Field census studies have been carried out to assess the effects of rodenticide use. Traps are set and the rodent population assessed by the number of each species trapped per 100 traps per 24 h. The rodenticide is administered and the traps again set and the numbers captured are compared with those of the earlier study to yield a percentage figure for rodent control. Advani (1983) reported 90 - 95% overall control both in winter (wheat, vegetable, and oil-seed crops) and summer (millet crop) with zinc phosphide administered, after pre-baiting, at 20 g/kg for one day at the entrance to active burrows and the administration of aluminium phosphide at 1.5 g/burrow followed by sealing with moist earth. Chopra (1984) undertook a similar study on aluminium phosphide in rice and wheat fields with 3 species in which the following percentage control rates were found (Table 13).

Table 13. Control rates (%) for 3 species in 2 crops achieved by aluminium phosphide[a]

Species	Rice fields	Wheat fields
Rattus meltada	85.88	75
Bandicota bengalensis	63.33	50
Mus species	100	91.8

[a] From: Chopra (1983).

Zinc phosphide dose-response data were reported for Shaw's gerbil (*Meriones shawi*) (Gill & Redfern, 1983) and the golden hamster (*Mesocricetus auratus* Waterhouse) (Bradfield & Gill, 1984). Zinc phosphide was administered in no-choice feeding tests at 10, 20, 30, 40, or 50 g/kg diet according to a standard protocol (EPPO, 1975). Their mortality data showed an irregular relationship for the gerbil (100% mortality at 20 g/kg diet but 90% at 50 g/kg diet), probably as a result of small study numbers. For the hamster, there was a dose-related mortality with 100% at 50 g/kg diet.

An LD_{50} of zinc phosphide for the black-tail prairie dog has been reported to be 18 mg/kg body weight (Tietjen, 1976).

7.4 Plants

The threshold concentration of phosphine in air for a harmful effect on growing lettuce (chosen as a representative,

highly sensitive species) was determined by Noack & Reichmuth
(1982b) to be between 3 and 8 mg/m^3. There were no adverse
effects on the germination of watercress seeds in soil that had
been treated for 3 days with air containing either 20 or 1400 mg
phosphine/m^3; in fact, the high phosphine concentration
stimulated the growth of watercress plants.

7.4.1   Harvested plants

Leesch (1984) studied the damage to harvested fresh iceberg
lettuce 14 days after fumigation with various fumigants,
including phosphine, at 4.4 °C without air circulation and at
24 °C both with and without air circulation. Three lettuce
heads were exposed to phosphine at each of 3 concentrations
(0.28, 0.56, and 0.83 mg/litre) for each of 3 periods (16, 24,
and 48 h). Lettuce heads were then stored at 4.4 °C in a sealed
bag made of 1 mm polythene. An ordinal 5-point scale was used
to rate damage to the lettuce heads and an index calculated by
dividing the sum of the scores for each of the 3 lettuces by the
sum of the scores of 3 unfumigated control lettuces. There was
no clear dose-effect relationship between the concentra-
tion·time product and the damage index. Damage was slight to
moderate after fumigation at 24 °C with circulation, but without
circulation (both at 4.4 and 24 °C) damage was generally rated
as "none", though one excursion into the "slight" category
occurred. This level of damage was small compared with the
damage resulting from 5 other fumigants. Only acetaldehyde
produced similar slight damage. However, the comparisons are
not clear because of the arithmetic manipulation of ordinal
scales.

7.4.2   Viable seeds and grain

Natarajan & Bagyaraj (1984) studied the effects of phosphine
on the viability of blackgram and fieldbean seeds and found that
it did not have any effects on seed germination.

Effects on viable seeds of leguminous plants were studied by
Singh et al. (1983) who reported the effects on stored pulses of
phosphine fumigation for 5 days. Phosphine residues at normal
fumigation levels (2 tablets of Phosfume[R] per tonne) decreased
to below the detection limit of 1.5 μg/kg after 3 or 4 days
aeration. Standard germination tests carried out immediately at
the end of fumigation revealed no impairment of germination,
even with pulses fumigated at 4 times the recommended dose.
These results suggest that stored pulses are not adversely
affected by exposure to phosphine or formulated aluminium
phosphide.

Ahmad (1976) studied seeds of 11 edible legumes and measured
the moisture content and the germination rate of control seeds

and of seeds fumigated for 7 days at 21 ± 5 °C using Phostoxin[R] at a rate equivalent to approximately 3 tablets/m^3, about 4 times the recommended dosage. After fumigation, test seeds were aerated for 3 days. No differences in germination rates were observed.

Zutshi (1966) cited earlier work on the effects of phosphine fumigation on seed germination and reported the results of germination tests on paddy, wheat, maize, bhindi, brinjal, tomato, and onion seeds. Three lots of each type of seed were fumigated with Phostoxin[R] at doses equivalent to 12 or 18 mg/kg for 7 days followed by 24 h aeration. After 30 days air-tight storage, one lot was given a second fumigation for 7 days, and a second lot was stored for 90 days in air-tight jars. In no case was there any reduction in germination rate.

Kamel et al. (1973, 1974) reported that one variety of wheat (Giza 155) was susceptible to phosphine fumigation (Phostoxin[R], 2.5 tablets/m^3) over 72 h with a germination rate that fell rapidly with the number of fumigations.

The effects of phosphine on the biochemical reactions in wheat grain of different moisture contents, which had been fumigated with 10 mg phosphine/litre at 20 °C for 5 days, were studied (Rohrlich & Menser, 1970). Germination of fumigated grain with a 30% moisture content was totally inhibited, it was reduced with a 25% moisture content, and was unaltered with a 20% content. Phosphine reduced the respiration of wheat partly by damaging the microflora present. Activity of glutamate decarboxylase was reduced by phosphine, when the moisture content was 18% or more. The activity of glutamate-oxaloacetate transaminase was not significantly changed compared with that in untreated grain. The enzymes hexokinase, aldolase, glyceraldehyde-3-phosphate dehydrogenase, and pyruvate kinase had nearly constant activity throughout all the studies. Alcohol dehydrogenase activity was reduced to zero within 7 days as a result of phosphine exposure of the grain at a moisture content of more than 24%. Catalase activity in wheat (moisture content about 15%) was reduced by about 20% after a 2-week exposure to phosphine fumigation. Phosphine treatment markedly inhibited respiration and growth of microorganisms in wheat with a moisture content up to 20%, thereby protecting the grain from microbial damage. In grain with a moisture content of between 18 and 27%, the amount of adenosine triphosphate (ATP) was reduced by phosphine fumigation, but adenosine diphosphate (ADP) was not, indicating that the respiratory activity in treated grain was markedly reduced.

8. EFFECTS ON EXPERIMENTAL ANIMALS AND *IN VITRO* TEST SYSTEMS

This section deals with studies of effects on experimental animals. Studies related to the field use of phosphine and phosphides as a fumigant or rodenticide are discussed in section 6.

8.1 Single Exposures

8.1.1 *Inhalation studies on phosphine*

The effects of phosphine on a kitten, a puppy, rabbits and a guinea-pig exposed to concentrations that proved fatal in a matter of hours were described in 1890 (Schulz, 1890). Meissner (1924) reported observations on rabbits in which exposure to 70 mg phosphine/m^3 (50 ppm) for 10 min did not produce any symptoms, but exposure to 140 mg/m^3 (100 ppm) was fatal in 2.5 - 3 h, and 700 mg/m^3 (500 ppm) was fatal in 25 - 30 min. Rats survived exposure to 80 and 800 mg/m^3 for 4 and 1 h, respectively; cats survived exposure to 240 mg/m^3 for 2 h; guinea-pigs did not survive 2 h exposure to 400 mg/m^3 (Rebmann, 1933).

Klimmer (1969) investigated the effects of single inhalation exposures on cats, rabbits, rats, and guinea-pigs. The concentrations varied from 35 to 564 mg/m^3. The symptoms described were similar to those described in section 8.2.1 for short-term studies. Death was attributed to respiratory paralysis followed by cardiac arrest.

The Pesticide Registration Standards for both aluminium phosphide and magnesium phosphide (US EPA, 1981, 1982) tabulate the survival times for all these studies and there is a clear relationship, independent of species, between concentration and survival time. This is illustrated in Fig 1. The relationship approximates to $C \times t^{1.43} = 200\ 000$, where C is the concentration of phosphine measured in mg/m^3 and t is the time to death in minutes.

Waritz & Brown (1975) determined a 4-h LC_{50} (95% confidence limits) for phosphine in male Charles River-CD rats of 15 (11 - 21) mg/m^3 (11 (8.1 - 15) ppm). Using CFT-Wistar female rats, Muthu et al. (1980) calculated LC_{50} and LC_{95} values for phosphine, produced by hydrolysis of aluminium phosphide, on the basis of concentration•time products. The LC_{50} (95% confidence limits) was 220 (180 - 270) mg•h/m^3, corresponding to a 4-h LC_{50} of 55 mg/m^3. However, the exposure durations were longer than 4 h, and there is evidence that the concentration•time product is not a good index of lethality in insects. The LC_{95} was 420 (260 - 670) mg•h/m^3. These values were obtained at 27 ± 2 °C, whereas an LC_{50} value of 360 mg•h/m^3 and an LC_{95} of 490 mg•h/m^3 were obtained at about 26 °C.
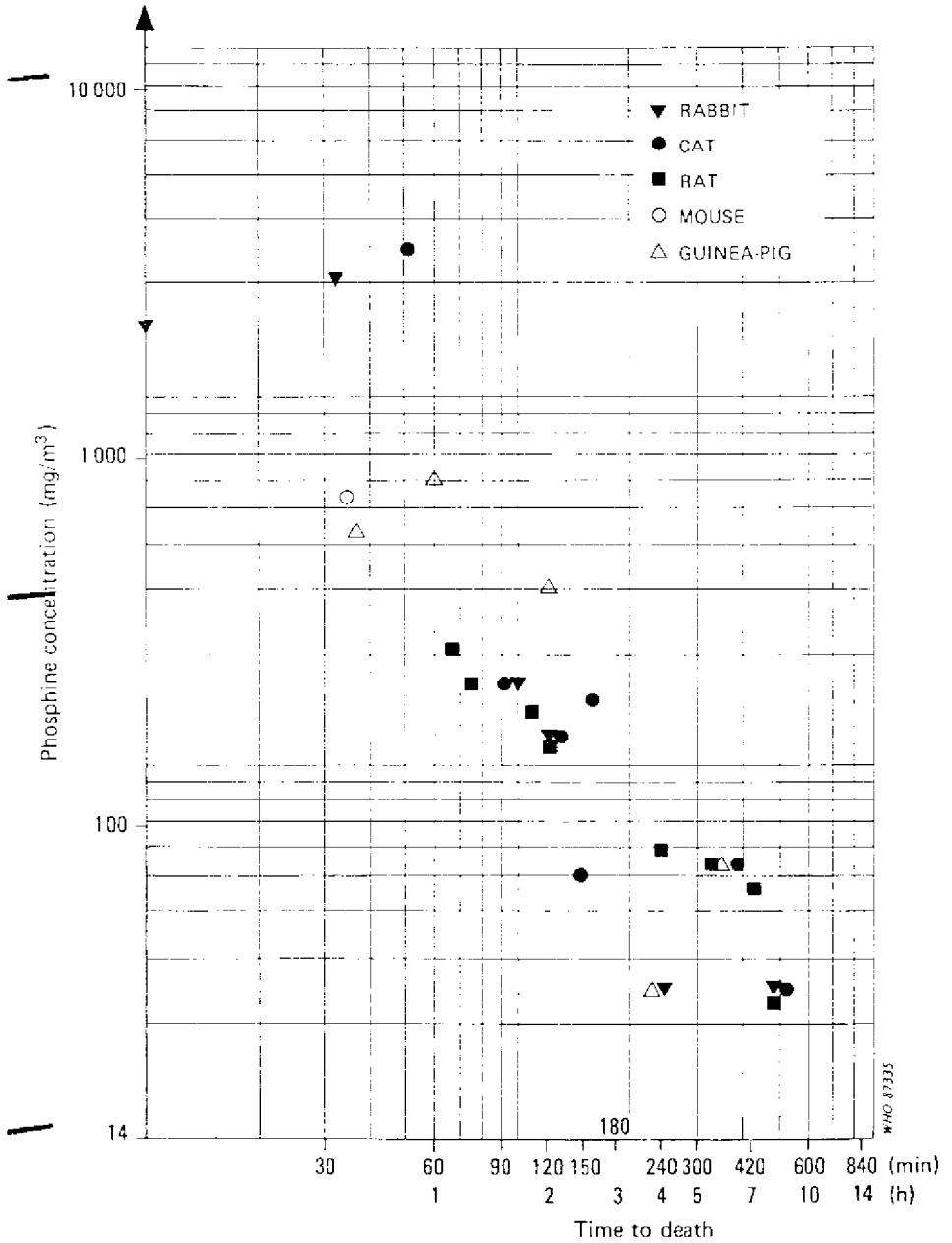
Fig. 1.   Survival time for different animal species in relation to phosphine concentration.

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Neubert & Hoffmeister (1960) investigated the effects of short-term exposures ranging from 460 to 2150 mg phosphine/m^3 on cellular respiration in rats. The oxidation of alpha-keto-glutarate in liver cells was inhibited, but not oxidative phosphorylation. Alpha-ketoglutarate oxidation was also reduced in heart muscle, and phosphorylation was disrupted. Survival of groups of 8 rats at various concentrations of diphosphine-free phosphine was studied with the following results (Table 14).

Table 14. Survival times of 8 rats in different concentrations of phosphine. Concentration·time products calculated from the mid-point of the range of survival times[a]

Concentration (mg/m^3)	Survival (min)	Concentration·time product (mg·min/m^3)
4640	16 - 20	83 520
1000	50 - 70	60 000
320	150 - 200	56 000
100	400 - 600	50 000

[a]   From: Neubert & Hoffmeister (1960).

If the use of the mid-point of the range of survival times is reasonable, these results indicate that the relationship between concentration and survival time is not simply reciprocal, and that the index of t is greater than 1, but less than the value of 1.43 derived from the data quoted by US EPA (1981, 1982).

Klimmer (1969) determined lethal concentration·time products for rats and calculated, for example, 19 000 mg·min/m^3 for a concentration of 287 mg/m^3, but these results do not agree with those calculated from the data of US EPA (1981, 1982) nor with those of Neubert & Hoffmeister (1960). The value of the concentration·time product, as a measure of phosphine dose in mammals, is therefore open to question, as it is in insects.

8.1.2 Inhalation studies on zinc phosphide

The US National Pest Control Association submitted a value of 19.6 mg/litre for an inhalation LC$_{50}$ of 10% zinc phosphide powder in rats. It was not stated whether the value was for pure zinc phosphide or the 10% dilution and there was no indication of exposure time (US EPA, 1983).

8.1.3 Oral studies on metal phosphides

In a study on 35 rats of both sexes administered doses of 20, 40, 50, or 80 mg/kg, Dieke & Richter (1946) reported an LD_{50} for zinc phosphide of 40.5 ± 2.9 mg/kg body weight for wild Norway rats. Schitoskey (1975) reported an LD_{50} for the kit fox of 93 mg zinc phosphide/kg body weight. Aminzhanov (1972) reported that 100 mg/kg body weight of zinc phosphide was fatal for starved dogs but not for dogs that were fed. Ikimanova (1977) reported increased blood- and urinary-porphyrin concentrations in rats dosed with zinc phosphide. Other authorities refer to 27 mg/kg body weight for an acute oral LD_{50} value for 94% pure zinc phosphide in rats (US EPA, 1983).

8.1.4 Dermal and other studies on metal phosphides

An acute dermal LD_{50} of 2 000 - 5 000 mg/kg body weight for zinc phosphide (94% Zn_3P_2) in rabbits was submitted by the US National Pest Control Association (US EPA, 1983).

8.2   Short-term Exposures

8.2.1   Inhalation exposure to phosphine

Jokote (1904) determined cumulative survival times in intermittent phosphine exposure as shown in Table 15.

Table 15. Survival time for rabbits, cats, and guinea-pigs at 3 concentrations of phosphine[a]

	Concentration of phosphine (mg/m^3)		
	14	35	140
Survival time (h) (rabbits, cats, and guinea-pigs)	16 - 30	8.5 - 12	2.5 - 3.5

[a]   From: Jokote (1904).

Miller (1940) exposed rabbits and guinea-pigs for 4 h per day to various concentrations of phosphine. At 28 mg/m^3 (20 ppm), both rabbits and guinea-pigs died during or after the second exposure. At 14 mg/m^3 (10 ppm), rabbits survived 7 - 14 successive exposures, but at 12 mg/m^3 (8.3 ppm), only 4 or 5 exposures. In another study in the series, 2 rabbits that had had five 4-h exposures to phosphine at 7 mg/m^3 were accidentally exposed to 20 mg/m^3 (14 ppm) on the sixth day. Both rabbits died during this exposure. The authors concluded that

pretreatment with sub-lethal concentrations of phosphine reduced resistance to near-lethal concentrations. At low concentrations (up to 14 mg/m^3), animals displayed no signs until about 1/2 h before death when they exhibited diminished reactivity, became stuporose with shallow respiration, and died in coma. Occasionally, animals died following exposure, with symptoms of pulmonary oedema. At 28 mg/m^3 or more, all animals exhibited signs of respiratory irritation and died of pulmonary oedema. Pathological examination of the lungs revealed bronchiolitis and atelectasis of the lungs. There was no evidence of haemolysis, but all organs were hyperaemic. The liver showed fatty infiltration and there was cloudy swelling of kidney tubular cells. After prolonged exposure, lungs frequently contained a brown pigment that did not stain with Prussian Blue and therefore was not iron-containing.

Neubert & Hoffmeister (1960) exposed female Wistar and E-B (inbred strain) rats in a 121-litre chamber repeatedly for 6 days to 681 mg/m^3 phosphine for 10 - 20 min per day. On the seventh day, they were exposed until they died (22 - 35 min). The concentration·time product for lethality after 6 days pretreatment (about 20 mg·min/litre) was about one third of that for a first exposure (48 - 87 mg·min/litre) and the authors concluded that the effects of exposure were cumulative, rather than that the phosphine accumulated in the body.

Klimmer (1969) exposed cats, guinea-pigs and rats to phosphine concentrations of 1.4 and 3.5 mg/m^3 (1 and 2.5 ppm) for 4 - 6 h per day, 6 days a week, for a total of more than 800 h over a 24-week period without any clinical, laboratory, or pathological evidence of effects. The liver function in the cats, measured by bromosulphthalein (BSP) excretion, was normal. In cats (3), guinea-pigs (3), and rats (10) exposed to phosphine at 7 mg/m^3 (5 ppm) for 6 - 8 h per day, the cats became apathetic, showing anorexia after exposure, but exhibiting thirst. Later, they developed unsteadiness, vomiting, agitation, dyspnoea, and apnoea, before cardiac arrest after a total of 35.5 - 45.5 h of exposure. The haemoglobin concentration and erythrocyte count were reduced by 10 - 20% compared with initial values, there was proteinuria and delayed BSP excretion. Post-mortem examination revealed congestion of all organs, with oedema and focal emphysema and red pigmentation of the lungs. The guinea-pigs exhibited signs that were similar to, but more marked than, those in the cats, and 2 animals had asphyxial convulsions. Blood tests showed no deviations from normal values, but the blood contained a brown coloration that was not due to methaemoglobin, since spectroscopy revealed the absorption bands of oxyhaemoglobin only. The guinea-pigs died on the sixth day after 24 h - 32 h cumulative exposure, and post-mortem examination revealed pulmonary oedema and red discolouration and congestion of other organs. The rats died

after 27 - 36 h cumulative exposure. All exhibited congestion of organs, 3 had pulmonary oedema and 7 had proteinuria. A second series of exposures under slightly changed exposure conditions at a different time of year produced qualitatively similar results. In both series, neurohistological studies in rats showed widening of the perivascular spaces, vacuolation of the nuclei of ganglion cells, a reduction in the Purkinje cells, and a glial reaction. Similar, but less marked, changes without glial reaction were seen in the cats and guinea-pigs. There was no control group in these studies, and certain observations in exposed animals were dismissed on the grounds that they had previously been seen in unexposed animals.

Waritz & Brown (1975) exposed Charles River-CD rats to phosphine for 4 h per day, for 12 days, at a concentration of 5.5 mg/m^3 (0.163 μmol/litre). The rate of weight gain was reduced during the exposure period. Although the authors stated that the rate returned to normal during a 14-day post-exposure observation period, their data indicate a uniformly reduced rate of weight gain throughout the exposure and recovery phases. Post-mortem examination of rats, sacrificed both at the end of the exposure period and at the end of the recovery period, revealed no gross or microscopic abnormalities.

The effects of a 1.5-month exposure of white rats to phosphine at concentrations of 0.05, 0.2, 1.5, and 8 mg/m^3 were reported by Pazynich et al. (1984). There were changes in blood cholinesterase, peroxidase, and catalase activity and in phagocyte behaviour. The magnitude of the changes was large, but not generally dose-related. On the basis of their results, the authors recommended mean exposure limits for urban air for exposure durations of 24 h, one month, and one year of 0.004, 0.0015, and 0.001 mg/m^3, respectively, with a ceiling value of 0.01 mg/m^3, and this recommendation has been adopted in the USSR.

In a study of the effects of phosphine on rats by inhalation at 0.1 mg/m^3 and 0.05 mg/m^3, Atchabarov et al. (1984) found a reduction in total plasma protein without a change in the relative proportions of the various fractions, a modest but significant reduction in the glycoprotein A fraction with some changes in the relative proportions of the sub-fractions, increased plasma bile acids, and a marked increase in seromucoids. Liver glycogen, lipids, and cytochrome oxidase levels were reduced. Biochemical changes were similar to those in a control group treated with hydrogen fluoride at a known toxic level.

8.2.2 Oral exposure to metal phosphides

Bai et al. (1980) reported a 13 week feeding study in female weanling albino rats. Zinc phosphide was mixed with the diet at

0 (control), 50 mg/kg (50 ppm), 100 mg/kg (100 ppm), 200 mg/kg (200 ppm), and 500 mg/kg (500 ppm) w/w. Deaths occurred at the 2 higher dosage regimes in 1/12 and 10/12 animals, respectively. Food intake and weight increase were reduced and dose-dependent depilation occurred at all dosages. The relative weights of liver, heart, brain, and thyroid were increased at 200 and 500 mg/m^3 and the serum zinc and liver alkaline phosphatase levels were increased at the highest dose. There was a dose-dependent reduction in haemoglobin concentration, red cell count, and haematocrit.

The WHO/FAO Data Sheet on Pesticides (No. 24) which deals with zinc phosphide (WHO/FAO, 1976) cites a study in which 300 mg zinc phosphide/kg administered to rats produced 6/6 deaths in the second week while 200 mg/kg produced reduced weight gain and 2/6 deaths. Histopathological studies revealed liver damage in the peripheral and central lobular areas and the lungs were congested with haemorrhage or exudate in the alveolar spaces.

8.2.3 Dermal and other exposures

No reports are available on short-term studies involving the dermal or other routes.

8.3 Skin and Eye Irritation; Sensitization

Zinc phosphide powder moistened with saline produced mild skin irritation in rabbits (Draize method) (Rao, 1986).[a]

8.4 Long-Term Exposure

No long-term studies on phosphine or metal phosphides exposure have been reported.

8.5 Reproductive, Mutagenicity, and Carcinogenicity

There have been no long-term reproductive, mutagenicity, or carcinogenicity studies on phosphine or metal phosphides.

8.6 Factors Modifying Toxicity; Toxicity of Metabolites

Aminzhanov (1972) reported that 100 mg zinc phosphide/kg body weight was lethal for dogs starved for 24 h beforehand but not for dogs that were fed.

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[a]   Personal communication to the IPCS Task Group on Phosphine and Metal Phosphides.

Phosphine is not a powerful reducing agent and its toxic effects are unlikely to be associated with chemical reduction. The recent discovery that phosphites and phosphorous acid have a fungicidal activity (Bompeix & Saindreman, 1984) suggests that metabolites of phosphine have biological effects and therefore might contribute to its toxicity. However, this contribution can only be small, since phosphine is only slowly oxidized to hypophosphites and phosphites and a maximum of about 40% of the phosphide dose is recovered as these metabolites, even with small doses. Moreover, the toxicity of phosphites is much less than that of phosphides (Schulz, 1887). The contribution of toxicity of contaminants of phosphine such as diphosphides is uncertain.

8.7 Mechanisms of Toxicity - Mode of Action

Studies on isolated rat liver showed that mitochondrial oxygen uptake was inhibited by phosphine (Nakakita et al., 1971) and that this effect was due to the reaction of phosphine with cytochrome c and cytochrome c oxidase (Kashi & Chefurka, 1976). Although this inhibitory *in vitro* effect was also shown in insects, it was found that insects severely poisoned with phosphine did not suffer any inhibition of their cytochrome system (Price & Dance, 1983). In the same report, phosphine was found to inhibit insect catalase though this appeared to be an indirect effect and might have been a result of phosphine toxicity rather than a cause.

There have not been any systematic studies on the mechanism of phosphine toxicity. Various effects on intermediary metabolism have been described. Ikimanova (1977) reported increased blood- and urinary-porphyrin concentrations, which were related to the dose of zinc phosphide and the duration of treatment. In a study on rabbits, Minchev & Dimitrov (1970) reported changes in serum glutamic-pyruvic and glutamic-oxaloacetic transaminases, leucine aminopeptidase, aldolase, alkaline phosphatase and albumin in the first 24 h of zinc phosphide poisoning. Dysfunction of hepatic fat metabolism was also observed. Loss of cell viability and cell membrane integrity accounted for the raised hepatic enzymes, the bronchiolitic effect, the cloudy swelling of renal tubular cells and the occasionally reported haemorrhagic lesions in the myocardium. There is no adequate explanation for the fact that phosphine does not cause the haemolysis characteristic of arsine.

9. EFFECTS ON MAN

9.1 Organoleptic Effects

The odour of phosphine depends on the impurities it contains; phosphine of high purity has no odour, even at 280 mg/m^3 (Dumas & Bond, 1974; Fluck, 1976). Phosphine prepared conventionally, without purification, has a fishy or garlic-like odour attributed to impurities. These may be adsorbed by stored products during fumigation with resultant loss of odour, even though phosphine remains at toxic concentrations (Bond & Dumas, 1967). Amoore & Hautala (1983) reviewed the literature on odour thresholds and stated that the geometric mean of all the 6 reported values for phosphine was 0.71 mg/m^3 (0.51 ppm). Fluck (1976) reported that thresholds for phosphine at which 50% or more of 10 persons could positively identify the odour associated with phosphine differed according to the source of the phosphine (Table 16).

Table 16. Odour thresholds for phosphine[a]

Source	Odour thresholds for PH$_3$ (mg/m^3 converted from ppm)
Technical aluminium phosphide + H$_2$SO$_4$	0.14 - 0.28
Phosphorium iodide + aqueous hydroxide	2.8
Phostoxin + H$_2$SO$_4$	0.014 - 0.028
(5 Å molecular seived)	1.4 - 2.3
Phostoxin + H$_2$SO$_4$	

[a]   From: Fluck (1976)

Amoore & Hautala (1983) classified phosphine in safety class D in their classification because 20 - 50% of attentive persons can detect the threshold limit value (TLV) (0.42 mg/m^3) by smell. However, the smell of phosphine cannot be relied on to warn of toxic concentrations.

9.2 General Population Exposure

9.2.1 Phosphine

There is negligible exposure of the general population to phosphine. Gessner (1937) described an incident in which the 12

inhabitants of an apartment house developed nausea and one died
when phosphine was emitted from an adjacent warehouse containing
bags of aluminium phosphide, which became damp. Some passengers
on ships and barges carrying cargoes of ferrosilicon or grain
under fumigation have also been poisoned by phosphine (Harger &
Spolyar, 1958; Netherlands, 1984). Effects were similar to
acute occupational poisoning.

Hallerman & Ribilla (1959) reported the deaths of 2 adults
and a child living in a dwelling with a party wall to a granary
being fumigated. Symptoms were non-specific and insidious
initially and illustrate the risks of sustained exposures to
relatively low concentrations (a few mg phosphine/m^3); it was
estimated that the concentration in the bedroom reached
1.2 mg/m^3. At autopsy, there was congestion of all organs.
Pulmonary oedema and focal emphysema were found in the lungs and
there was vacuolation in the liver.

9.2.2 Metal phosphides

Zinc phosphide baits and formulated aluminium phosphide
pellets are widely used. Occasional accidental, or more usually
suicidal, exposure to metal phosphides may be encountered.
Ingestion, the only easily toxic route, has almost always been
suicidal and the effects acute (Stephenson, 1967).

Stephenson (1967) reviewed 20 cases of acute zinc phosphide
poisoning by ingestion (including one treated by himself) in
which the approximate dose was recorded. Ten cases were fatal
and the doses ranged from 4.5 to 180 g; 6 cases had ingested
20 g or more. In the 10 non-fatal cases, the doses ranged from
0.5 to 50 g with 7/10 ingesting less than 20 g. In the case
treated by the author, clinical features included metabolic
acidosis, hypocalcaemic tetany, methaemalbuminaemia, and reduced
blood coagulation (thrombotest 28% of normal). Post-mortem
findings included blood in all the serous cavities, pulmonary
congestion and oedema, haemorrhagic changes in the intestinal
epithelium, centrilobular congestion and necrosis and yellow
discoloration of the liver, and patchy necrosis of the proximal
convoluted tubules of the kidneys.

An unsuccessful suicidal attempt was described by Zipf
(1967). A 25-year-old man ingested 6 tablets of Phostoxin[R] in
water (- 6 g phosphine). Immediate symptoms were severe retro-
sternal pain, a generalized burning sensation, and vomiting.
There was circulatory collapse necessitating resuscitation and
he was also treated by gastric lavage with potassium perman-
ganate and magnesium sulfate. Subsequently, symptoms of cardiac,
cerebral, and hepatic dysfunction appeared and there was severe
renal failure requiring haemodialysis.

9.3 Occupational Exposure

Cases of acute phosphine poisoning reported in the litera-
ture were reviewed by Harger & Spolyar (1958). Fifty-nine cases
with 26 deaths had been recorded since 1900 (including Gessner's
(1937) non-occupational cases). In 6 out of 11 papers, cargoes
of ferrosilicon were cited as the source of phosphine and, in
these cases, the victims were passengers or crew members of the
ships or barges concerned. Other cases involved the exposure of
welders to calcium carbide and raw acetylene and of submariners
to sodium phosphide. The most common autopsy finding was
congestion of the lungs with marked oedema. The authors added a
case of their own in which a 16-year-old youth operating
acetylene generators died, probably as a result of phosphine
exposure at a level of about 11 mg/m^3 (8 ppm) for 1 - 2 h daily
over a period of 6 weeks. Arsine and hydrogen sulfide exposures
were considered to have been too low to have accounted for the
death. Autopsy revealed acute pulmonary oedema. Other cases
have been described, all of which exhibited similar features
(Ziemer, 1963; Furuno et al., 1976; & Netherlands, 1984). Three
groups of symptoms of acute phosphine poisoning were described
by Childs & Coates (1971): (a) nervous symptoms including
headache, vertigo, tremors, and unsteady gait, progressing in
severe cases to convulsions, coma, and death; (b) gastro-
intestinal symptoms include loss of appetite, thirst, nausea and
vomiting, diarrhoea, and severe epigastric pain; and (c) respir-
atory symptoms including a feeling of pressure and pain in the
chest as well as shortness of breath. In addition, a sharp fall
in blood pressure was described as a characteristic, but seldom-
mentioned symptom. Chronic effects include anaemia, bronchitis,
and gastrointestinal, speech, and motor disturbances, but these
are by no means general.

The toxic effects of phosphine are summarized in Table 17.

The IDLH (Immediately Dangerous to Life or Health) level is
282 mg/m^3 (200 ppm) (US EPA, 1985b).

Verga & Belloni (1958) described a case of purpura ascribed
to poisoning by phosphine. The platelet count was reduced to
60 000/mm^3; the red-cell count was also low at 3.1 x 10^6/mm^3 and
the haemoglobin concentration (Sahli) was 55%. With recovery,
both the red-cell and thrombocyte counts increased to 4.8 x 10^6
and 210 000/mm^3, respectively, at the end of the observation
period. The electrolysis of phosphate solutions being applied
to metals as a corrosion inhibitor was considered to have
produced phosphine, but there was no measurement of the level of
exposure and it is possible that the exposure also included
arsine, which would account for the anaemia with a hyperplastic
erythroid series in the marrow biopsy. While haemolysis is
recognized as a complication of arsine poisoning, purpura and
thrombocytopenia are not widely recognized features of

Table 17. Toxic effects of phosphine[a]

Effect	Concentration	
	mg/m^3	ppm
Rapidly fatal	2800	2000
Death after 1/2 - 1 h	560 - 840	400 - 600
Dangerous to life after 1/2 - 1 h	400 - 600	290 - 430
Serious effect after 1/2 - 1 h	140 - 260	100 - 190
No serious effects after 1/2 - 1 h	10	7

[a] From: Childs & Coates (1971).

phosphine poisoning, though Borodin et al. (1983) described changes related to haemolysis (prolongation of clotting time and reduced plasminogen levels) in workers exposed to a variety of biocides and disinfectants, including zinc phosphide. However, these changes could not be related specifically to exposure to phosphine.

In 1978, Beloskurskaya et al. published the findings of a study on 206 phosphorus workers, which revealed 57 cases of toxic hepatitis. In 49 of these cases, there was stated to be no other apparent cause than occupational exposure to phosphorus, phosphine, or phosphorus oxides. No measurements of exposure were reported. Diagnostic criteria included a history of hypochondrial pain, clinical hepatomegaly, and abnormal liver function tests and ^{131}I-Rose Bengal scanning and clearance studies. Forty-six of the 57 cases had additional symptoms, such as the astheno-vegetative syndrome, chronic gastritis, impotence, toxic encephalopathy, toxic cardiomyopathy, and respiratory disease. There was no control group and no information on alcohol consumption was provided.

In a later study, Wilson et al. (1980) described the symptoms of acute poisoning by phosphine as headache, fatigue, nausea, vomiting, jaundice, paraethesia, ataxia, tremor, diplopia, myocardial infiltration with necrosis, pulmonary oedema, and myocardial and peripheral muscle damage. Laboratory results indicated that there had been urinary tract involvement (occult blood) and levels of several liver enzymes were elevated. Furthermore, Roaldsnes (1982) reported that metal-workers at a large shipyard in Norway, drilling deep holes in spheroidal graphite iron, became ill during work. The symptoms were mostly nausea, dizziness, chest tightness, dyspepsia, and disturbances of smell and taste. Measurement of the phosphine concentration in the workers' breathing zone (with Dräger tubes)

showed a phosphine concentration of about 1.4 mg/m^3 (1 ppm).
After installing local exhaust ventilation on the drilling
machines, there were no longer any measurable amounts of
phosphine, and there were no complaints from the workers. When
the local exhaust ventilation was removed for technical reasons
5 years later, illness among the workers recurred. Measurement
of phosphine levels just above the machines, showed concentra-
tions of up to 56 - 70 mg/m^3 (40 - 50 ppm). When the local
exhaust ventilation was re-installed, the phosphine concentra-
tions dropped to unmeasurable amounts, and no further cases of
illness were reported. Addition of copper sulfate solution to
the cutting oil effectively removed the phosphine gas, but
resulted in problems of corrosion.

Jackson & Elias (in press) studied liver function and
gastrointestinal symptoms in: (a) workers exposed to white
phosphorus at up to 5 times the occupational exposure limit and
also to some phosphine (0 - 0.5 mg/m^3); (b) workers exposed to
phosphine alone (at up to 2.8 mg/m^3, depending on the efficacy
and use of air-line breathing apparatus); and (c) gastro-
intestinal symptoms in a group of workers who were not exposed
to either compound, but who worked the same shift pattern.
Complaints to the factory medical department of gastrointestinal
symptoms in the phosphine-exposed group, in the phosphorus
exposed-group, and in the unexposed group were 1.51, 0.61, and
0.24 per man per year, respectively. However, the alcohol
consumption in the phosphine- and phosphorus-exposed groups
differed (289 and 154 ml per week, respectively). The alcohol
consumption was not estimated in the control group. None of the
conventional liver function tests reflected the differences in
occupational exposure or alcohol intake. The phosphine-exposed
group did have a significantly raised post-prandial bile acid
concentration, but whether this reflected a difference in phos-
phine exposure or alcohol consumption could not be determined.

10. EVALUATION OF HUMAN HEALTH RISKS AND EFFECTS ON THE ENVIRONMENT

10.1 Evaluation of Human Health Risks

Phosphine and metal phosphides are only focally distributed in the environment and exposure of large numbers of the general population is unlikely. The sources and uses of phosphine and metal phosphides are sufficiently well established for safety techniques to be generally known, though it is disturbing that shipboard poisonings are still occurring when the first case was reported over half a century ago. The extensive world-wide use of metal phosphides as fumigant sources and as rodenticides creates the hazard of accidental poisoning in children and others and provides a means of suicide, unless the supply and use of these materials is appropriately regulated. There is no evidence of danger to the population from residues of phosphine in fumigated foods or from residues of phosphides, if adequate fumigation and aeration techniques are used. Permitted limits for residues are in the range of 0.01 - 0.1 mg total phosphine/ kg. There do not appear to be any deleterious effects on food quality as a result of adequately undertaken fumigation with phosphine.

Published data on occupational exposure to phosphine in most industries are not sufficient to evaluate the success of control measures in relation to occupational exposure limits. There is also some disagreement regarding these limits and some data suggest that an occupational exposure limit of 0.4 mg phosphine/m^3 is above the threshold for biological effects with long-term exposure. However, there are no studies adequate to settle this question definitively.

Major accidental release of stored phosphine presents a serious explosion/fire hazard and an acute toxic hazard for man and animals.

10.2 Evaluation of Effects on the Environment

Only very small amounts of phosphine and phosphides occur in the environment as a result of natural processes and those resulting from human activity are not persistent.

Phosphine and phosphides are introduced into the environment for the control of pests, for which they are particularly suitable, because of their efficacy, lack of persistence, and harmless decomposition products.

Careful positioning of bait and the low toxicity of carcasses for scavengers of poisoned animal targets minimize the effects of rodenticidal metal phosphides on non-target species. Phosphine and phosphides are oxidized in the environment and the

final product, phosphate, is normally widely present in the natural environment. Phosphine and phosphides are only used where the natural environment is already modified by human activity and their environmental impact must be viewed in this context.

Since resistance to phosphine in insects does not appear to reverse in the absence of selective pressures, it is particularly important to achieve effective doses of phosphine by using adequate treatment rates and good practices. In practice, the development of resistance is more of a problem than the effects on non-target species. Major accidental release of stored phosphine would not result in long-term environmental consequences.

10.3 Conclusions

In general, there is no risk to the public from the use of phosphine or phosphides, from emissions from fumigated products or spaces, from alloys, industrial processes, or residues in food, provided that proper fumigation, transport, and industrial practices are used.

While phosphine and metal phosphides are toxic, their extensive use in occupational settings has resulted in the establishment of good practice guidelines. If these are followed, there is a low order of risk for human health. However, there is some doubt about the completeness of protection afforded by the higher occupational exposure limit (0.4 mg phosphine/m^3).

There is no risk of important environmental effects from phosphine or metal phosphides.

11.   RECOMMENDATIONS

11.1 Gaps in Knowledge

1.   The principal area related to human health where information
is inadequate is concerned with occupational exposure levels and
their safety. There is a need for an adequate study of well-
being and organ function in exposed and control populations,
with a complete description of phosphine exposure, concurrent
exposures to other chemicals (which should be low), alcohol
consumption, and smoking habits. The establishment of exposure
levels that are free from any detectable effects would help to
determine agreed occupational exposure limits.

2.   It would be of assistance, in this regard, to undertake
laboratory studies to identify biological markers that are most
indicative of phosphine effects and reflect the suggested short-
term cumulative effects of repeated phosphine exposure.

3.   Further research on the mechanisms of action of phosphine
and phosphides will assist in the design of good fumigation
practice and fumigation schedules, and minimize the occurrence
of resistance in target organisms and associated problems.

4.   It has been suggested that zinc phosphide may be relatively
persistent in aquatic sediments and there is a general lack of
aquatic toxicological data. There is a need for research on the
levels and persistence of zinc phosphide in sediments and for
further studies of its effects on aquatic organisms.

11.2 Preventive Measures

11.2.1 Management

The most important factor in the safe handling of phosphine
and metal phosphides, and in their formulation, is proper work
practices.
Management should identify these, provide training for the
operatives, and ensure that the practices are carried out.
Personal protection measures recommended to reduce the
likelihood of absorption of phosphide preparations include the
wearing of:

(a)   synthetic rubber gloves;
(b)   rubber boots;
(c)   lightweight impervious overalls; and
(d)   suitable eye protection.

Adequate washing facilities should be available at all times during handling. Eating, drinking, and smoking should be prohibited during handling, and before washing after handling. The means to measure concentrations of phosphine in air should be available and used to check atmospheric concentrations. When necessary, respiratory protective equipment should be worn. In fumigation, each operator or other person liable to be exposed to the gas must be provided with an efficient means of respiratory protection. Persons exposed to magnesium phosphide or aluminium phosphide powders (or other readily hydrolysed phosphides), which may give rise to airborne dust should be protected by respiratory protective equipment, effective against gaseous phosphine, since hydrolysis of dust in the filter of a dust mask or respirator may give rise to high phosphine exposure.

11.2.2   Treatment of poisoning

11.2.2.1 Inhalation of phosphine

(a)   First Aid

Remove from exposure, keep at rest. Rescuers should follow full safety procedures.
If the patient is unconscious, place in semi-prone recovery position or otherwise maintain the airway.
If the patient is conscious but has difficulty in breathing, treat in a seated position and give oxygen if available. Otherwise, allow the patient to recline with the legs slightly elevated.
If breathing stops, immediately ventilate the patient artificially (mouth-to-mouth/nose or mechanically with oxygen, if available).
If the heart stops, begin cardiopulmonary resuscitation (CPR).

(b)   Medical Treatment

1.   Give oxygen by mask if required; perform baseline chest X-ray and examine chest. Treat shock conventionally.

2.   All patients should be observed for 48 - 72 h, as onset of pulmonary oedema may be delayed.

3.   If respiratory distress is severe, give steroids (methyl-prednisolone 30 mg/kg, or equivalent, intramuscularly), preferably within 4 h of exposure.

4.   If pulmonary oedema occurs, treat by positive and expiratory pressure ventilation. Antibiotics should only be given if a secondary infection is present.

5.   Treat any fits conventionally; give general supportive care with particular attention to fluid balance.

6.   Some sources of exposure to phosphine are likely to lead also to exposure to arsine, which has haemolytic effects. Monitor for haemoglobinaemia, haemoglobinuria, unconjugated hyperbilirubinaemia, and renal failure.

In general, recovery is rapid following removal from exposure, but renal damage and leukopenia may occur after several days.

11.2.2.2 Ingestion of metal phosphides

(a) <u>First aid</u>

Do not give milk, fats, or saline emetics by mouth.
Give oxygen if there is respiratory distress.
If first aiders are medically authorized to do so, and the patient is conscious, induce vomiting.
After 20 min (or after vomiting), administer activated charcoal (50 g in water by mouth) if available.
Obtain medical attention as soon as possible: preferably send immediately to hospital.

(b) <u>Medical treatment</u>

1.   Consider tracheal intubation and gastric lavage with 2% sodium bicarbonate solution (to limit hydrolysis of zinc phosphide).

2.   Activated charcoal or medicinal liquid paraffin may limit absorption of phosphine and zinc phosphide, respectively, and may be administered by mouth or stomach tube.

3.   Monitor and support vital functions, particularly hepatic and renal function. Treat shock conventionally.

5.   Hepatic and renal failure should be treated in specialist centres.

11.2.3   Leaks, spillages, residues, and empty containers

11.2.3.1 Phosphine

Small leaks and residues of compressed gas can be discharged slowly to the atmosphere in the open air. Larger quantities should be burned using an appropriate burner.

11.2.3.2 Aluminium and magnesium phosphides and their formulated preparations

Spillages and residues in containers will evolve phosphine for several days by reaction with atmospheric moisture. Respiratory protective equipment will be required by those dealing with them. Even if atmospheric concentrations of phosphine are initially low, they may rise as residues are disturbed. Moderate quantities should be collected in a secure place to protect the public and wildlife, and left in the open air and kept moist until hydrolysis is complete.

Larger amounts should be removed to a deep pit, at an approved site, until phosphine evolution is complete. Then they can be buried.

Residues at the site of spillage should be washed away in a large quantity of water and the area kept secure and aerated until checked for zero gas concentration.

Combustible packages can be incinerated at high temperature (> 1000 °C) using proper facilities. Containers should NOT be cleaned for re-use, but should be disposed of by deep burial, at an approved site, well away from habitation and where there is no danger of contamination of water sources.

11.2.3.3 Zinc phosphide and preparations

Zinc phosphide hydrolyses only slowly. Nevertheless, phosphine may be evolved and respiratory protective equipment should be available.

Spillages and residues in containers should be incinerated at high temperatures (> 1000 °C) or crushed and buried below the topsoil at an appropriate site. Contaminated surface material should be treated similarly or else the area should be secured from public access until the gas concentration is zero.

12. PREVIOUS EVALUATIONS BY INTERNATIONAL BODIES

Phosphine was evaluated in 1971 by WHO and FAO, as a pesticide residue in food (WHO/FAO, 1972). A tolerance of 0.1 mg/kg (0.1 ppm) in raw cereals was confirmed. It was recommended that the tolerance of 0.01 mg/kg (0.01 ppm) in flour, other milled cereal products, breakfast cereals, dried vegetables, and spices be confirmed and extended to include nuts, groundnuts, dried fruit, cocoa beans, and other similar foods, known to be fumigated with phosphine.

The use of zinc phosphide as a rodenticide in public health was reviewed by WHO in 1972. It was concluded that it was a generally effective compound and, while highly toxic to domestic fowl, its safety record was good. The use of zinc phosphide was endorsed (WHO, 1973).

REFERENCES

ACGIH (1986) *Threshold limit values and biological exposure indices for 1986-1987*, Cincinnati, Ohio, American Conference of Governmental Industrial Hygienists.

ADU, O.O. & MUTHU, M. (1985) The relative toxicity of seven fumigants to life cycle stages of *Callosobruchus chinensis* L. *Insect Sci. Appl.*, 6(1): 75-78.

ADVANI, R. (1983) A comparative trial of zinc phosphide, aluminium phosphide and RH-787 against field mixed population of rodents in crop ecosystem of the Indian desert. *Ztg angew. Zool.*, 70(2): 153-168.

AHMAD, M. (1976) Effect of phosphine fumigation on the germination of edible legume seeds. *J. stored Prod. Res.*, 12: 211-212.

AL-OMAR, M.A. & AL-BASSOMY, M. (1984) Persistence of phosphine gas in fumigated Irafir dates. *J. food Saf.*, 6: 253-260.

AL'ZHANOV, S., MOLDABEKOV, Sh.M., ERSHOV, V.A., & POPOV, A.P. (1983) [*Use of copper and iron sulfates in cleaning furnace gas from phosphorus production,*] Moscow, Academy of Sciences of the USSR, Institute of Scientific Information (VINITI) (Document 4802-83) (in Russian).

AMINZHANOV, M. (1972) [Use of zinc phosphide in controlling stray dogs.] In: Azimov, Sh.A., Shevchenko, N.Kh., & Nurmatov, R.Sh., ed. [*Proceedings of the 7th Conference of Young Uzbek Agricultural Scientists,*] Tashkent, Uzbeksii Nauchno-Issled. Veterinary Institute (in Russian).

AMOORE, J.E. & HAUTALA, E. (1983) Odour as an aid to chemical safety: odour thresholds compared with threshold limit values and volatilities for 214 industrial chemicals in air and water dilution. *J. appl. Toxicol.*, 3(6): 272.

ANON. (1936) [Trials to establish whether the Delicia grain weevil fumigation procedure is safe to apply and free from the risk of fire and explosion.] *Gutachten chim. tech. Reichsanst.*, 8(4): 5 (in German).

ANON. (1956) Ferro-silicon explosion risks. *Chem. Trade J. Chem. Eng.*, 16 Nov.: 1180.

ARENDT, G., FARWIK, W., HAAG, F., KAUFMANN, G., KOHL, E.-G., PRUGGMAYER, D., & WAGNER, W. (1979) [Environmental aspects of

pest control. Fumigation of grain and food stores.] *Lebensmit-teltechnik*, 5: 2-8 (in German).

ARNOLD, J.T. & ROBBIANO, P. (1974) *Portable vapour surveil-lance system*, Springfield, Virginia, National Technical Information Service (NTIS Report No. AD-782 844).

ATCHABAROV, B.A., AITBAYEV, T.KH., & AITBEMBETOV, B.N. (1984) [Comparison of the effects of hydrogen fluoride and phosphine on the organism with various exposure levels.] In:[*Health care in Kazakhstan. Part 1,*] pp. 28-31 (in Russian).

ATTIA, F.I. (1984) Multiple cross-resistance characteristics in phosphine-resistant strains of *Rhizopertha dominica* and *Tribolium castaneum*. In: Ripp, B., ed. *Controlled atmosphere and fumigation in grain storages*, Amsterdam, Oxford, New York, Elsevier Science Publishers, pp. 49-53 (Developments in Agricultural Engineering No. 5).

ATTIA, F.I. & GREENING, H.G. (1981) Survey of resistance to phosphine in coleopterous pests of grain and stored products in New South Wales. *Gen. appl. Entomol.*, 13: 93-97.

BAI, K.M., KRISHNAKUMARI, M.K., RAMESH, H.P., SHIVANANDAPPA, T., & MAJUMDER, S.K. (1980) Short-term toxicity study of zinc phosphide in Albino rats (*Rattus norvegicus*). *Indian J. exp. Biol.*, **18**(8): 854-857.

BARRETT, W.J. & DILLON, H.K. (1978) *Development of methods for the determination of elemental phosphorus and phosphine in air*, Cincinnati, Ohio, National Institute of Occupational Safety and Health, 102 pp (DHEW (NIOSH) Publication No. 78-177).

BECKER, K.H., BIEHL, H.M., BRUCKMANN, P., FINK, E.H., FUHR, F., KLOPFER, W., ZELLNER, R., & ZETSCH, C. (1984) *Methods for the ecotoxicological evaluation of chemicals, photochemical degra-dation in the gas phase. VI. OH· reaction rate constants and tropospheric lifetimes of selected environmental chemicals*, Jülich, Kernforschungsanlage (Projektträgerschaft Umweltchemi-kalien Report 1980-83, No. 279).

BELL, C.H., WILSON, S.M., & BANKS, H.J. (1984) Studies on the toxicity of phosphine to tolerant stages of *Trogoderma granarium everts* (Coleoptera: Dermestidae). *J. stored Prod. Res.*, **20**(2): 111-117.

BELOSKURSKAYA, G.I., PARASKEVOPULOS, YA.G., & SHLYGINA, O.E. (1978) [On the clinical and functional condition of the liver in patients with chronic phosphorus poisoning.] *Tr. Inst. Kraev. Patol. Akad. Nauk Kazakhskoi SSR*, **36**: 25-30 (in Russian).

BLAXLAND, J.D. & GORDON, R.F. (1945) Zinc phosphide poisoning in poultry. *Vet. J.*, **101**: 108-110.

BOENIG, I.A., CRUTCHFIELD, M.M., & HEITSCH, C.W. (1982) Phosphorus compounds. In: Kirk-Othmer, ed. *Encyclopaedia of chemical technology*, 3rd ed., New York, Wiley Interscience, **Vol. 17**, pp. 490-539.

BOMPEIX, G. & SAINDRENAN, P. (1984) *In vitro* antifungal activity of Fosetyl Al and phosphorous acid on Phytophthera species. *Fruits*, **39**(12): 777-786.

BOND, E.J. & DUMAS, T. (1967) Loss of warning odour from phosphine. *J. stored Prod. Res.*, **3**: 389-392.

BOND, E.J., ROBINSON, J.R., & BUCKLAND, C.T. (1969) The toxic action of phosphine. Absorption and symptoms of poisoning in insects. *J. stored Prod. Res.*, **5**: 289-298.

BOND, E.J., DUMAS, T., & HOBBS, S. (1984) Corrosion of metals by the fumigant phosphine. *J. stored Prod. Res.*, **20**(2): 57-63.

BONTOYAN, W.R. (1981) Report on pesticide formulations: herbicides, fungicides and miscellaneous. *J. Assoc. Off. Anal. Chem.*, **64**(2): 355.

BORODIN, A., KLIMEK, M., & ZAKL, K. (1983) [Blood clotting and fibrinolysis systems in workers of a disinfection, disinfestation and deratization employment (sic).] *Med. Pr.*, **34**(4): 313 (in Polish).

BOUBAL, M., LOUISE, J., BELOT, D., & POLITI, J.M. (1981) *Analyse directe et continue de traces d'hydrures gazeux à très basses teneurs*, Paris, Institut National de la Propriété Industrielle, Sociéte d'Air Liquide (Document No. 2 506 459) (Demande de Brevet d'Invention No. 81 10316).

BOWKER, J.R. (1958) The liberation of phosphine in the machining of spheroidal graphite iron. In: *Proceedings of the British Cast Iron Research Association, 8 November*, p. 50.

BOWLEY, C.R. & BELL, C.H. (1981) The toxicity of twelve fumigants to three species of mites infesting grain. *J. stored Prod. Res.*, **17**: 83-87.

BRADFIELD, A.A.G. & GILL, J.E. (1984) Laboratory trials of five rodenticides for the control of *Mesocricetus auratus* Waterhouse. *J. Hyg. (Lond.)*, **93**: 389-394.

BRANDON, R.W. (1983) The use of chemically impregnated paper tapes for toxic gas detection and monitoring. *Anal. Chem. Symp. Ser.*, **17**: 726-31.

BREYER, D. (1973) [Studies on hydrogen phosphide residues in grain after treatment with fumigation preparations in powder form.] *Mühle Mischfuttertech.*, **110**: 699-700 in German).

BUBIEN, Z., WARTENBERG, L., & ROSZYK, E. (1974) [Content of zinc in hen carcasses poisoned with zinc phosphide.] *Zesz. Nauk. Akad. Roln Wroclawiu Weter.*, **31**: 173-180 (in Polish).

CABROL TELLE, A.M. & DE SAINT BLANQUAT, G. (1984) *In vitro* digestibility of starch, casein and triolein after fumigation with phosphine. *Sci. Aliments*, **4**(Hors Ser. 3): 287-291.

CABROL TELLE, A.M., DE SAINT BLANQUAT, G., DERACHE, R, HOLLANDS, E., PERIQUET, B., & THOUVENOT, J.-P. (1985) Nutritional and toxicological effects of long-term ingestion of phosphine-fumigated diet by the rat. *Food Chem. Toxicol.*, **23**(11): 1001-1009.

CALVERT, J.G. & PITTS, J.N., Jr (1966) *Photochemistry*, New York, John Wiley and Sons.

CHAMP, B.R. & DYTE, C.E. (1976) *Report of the FAO global survey of pesticide susceptibility of stored grain pests*, Rome, Food and Agriculture Organization of the United Nations, pp. 1-257 (FAO Plant Production and Protection Paper No. 5).

CHAN, L.T.F., CROWLEY, R.J., DELLIOU, D., & GEYER, R. (1983) Phosphine analysis in post-mortem specimens following ingestion of aluminium phosphide. *J. anal. Toxicol.*, **7**: 165-167.

CHENTSOVA, N.YU. (1972) [Danger to poultry during scattering of zinc phosphide-poisoned grain to control rodents in forest plantings.] In: Voronova, L.D., ed. [*Effects of pesticides on wild animals.*] Moscow, pp. 90-102 (in Russian).

CHILDS, A.F. & COATES, H. (1971) The toxicity of phosphorus compounds. In: *Mellor's comprehensive treatise on inorganic and theoretical chemistry*, White Plains, New York, Longman, **Vol. 8**, Suppl. 3, pp. 1437-1440.

CHILDS, D.P. (1972) Tobacco consignment fumigated in containers while in transit. *Container News*, **October**.

CHILDS, D.P., OVERBY, J.E., & NIFFENEGGER, D. (1969) Phosphine fumigation of flue-cured tobacco warehouses for the control of the cigarette beetle. *Tobacco*, **168**(21): 20-25.

CHOPRA, G. (1984) Field efficacy of aluminium phosphide against rodents. *Indian J. agric. Sci.*, 54(3): 204-205.

CIBA (1978) *Phosphorus in the environment: its chemistry and biochemistry*, Amsterdam, Oxford, New York, Elsevier North Holland (Ciba Foundation Symposium 57 - new series).

CODEX ALIMENTARIUS (1986) *Codex maximum limits for pesticide residues*, 2nd ed., Rome, Food and Agriculture Organization of the United Nations (CAC/Vol. XIII-Ed.2).

COLE, R.J. & BENNETT, W.H. (1950) An investigation of the possibility of the occurrence of phosphine and acetylene in the atmosphere of factories for the powdering of magnesium. *J. Soc. Chem. Ind.*, 69: 29-30.

CORBRIDGE, D.E.C. (1974) *The structural chemistry of phosphorus*, Amsterdam, Oxford, New York, Elsevier Science Publishers, pp. 268-269.

COX, P.D., BELL, C.H., PEARSON, J., & BEIREN, M.A., (1984) The effect of diapause on the tolerance of larvae of *Ephestia küehniella* to methyl bromide and phosphine. *J. stored Prod. Res.*, 20(4): 215-219.

CURRY, A.S., PRICE, D.E., & TRYHORN, F.G. (1959) Absorption of zinc phosphide particles. *Nature (Lond.)*, 184(4686): 642-643.

DAVIS, R. (1986) Fumigation of ships. In: *Proceedings of the GASGA Seminar on Fumigation Technology in Developing Countries, Slough, United Kingdom, 18-21 March*, London, Foreign and Commonwealth Office, Overseas Development Administration, Tropical Development and Research Institute, pp. 24-34.

DELOMENIE, H. (1933) Gases evolved from ferrosilicons under the influence of water. *J. Pharm. Chim.*, 18: 289-292, 532

DIEKE, S.H. & RICHTER, C.P. (1946) Comparative assays of rodenticides on wild Norway rats. *Public Health Rep.*, 61: 672-679.

DIETERICH, W.H., MAYR, G., HILD, K., SULLIVAN, J.B., & MURPHY, J. (1967) Hydrogen phosphide as a fumigant for foods, feeds and processed food products. *Residue Rev.*, 19: 135-149.

DISNEY, R.W. & FOWLER, K.S. (1972a) Residues in cereals exposed to hydrogen phosphide. In: *Radiotracer studies on chemical residues in food and agriculture. Proceedings of a Combined Panel and Research Coordination Meeting, Vienna,*

Austria, 25-29 October 1971, Vienna, International Atomic Energy Agency, pp. 99-101.

DISNEY, R.W. & FOWLER, K.S. (1972b) The possibility of isotope exchange or other interfering reactions occurring during the determination of residues from fumigation with ^{32}P-labelled phosphine. In: *Radiotracer studies on chemical residues in food and agriculture. Proceedings of a Combined Panel and Research Coordination Meeting, Vienna, Austria, 25-29 October 1971,* Vienna, International Atomic Energy Agency, pp. 87-88.

DUMAS, T. (1964) Determination of phosphine in air by gas chromatography. *J. agric. food Chem.,* **12**(3): 257-258.

DUMAS, T. (1978) Modified gas chromatographic determination of phosphine. *J. Assoc. Off. Anal. Chem.,* **61**(1): 5-7.

DUMAS, T. (1980) Phosphine sorption and desorption by stored wheat and corn. *J. agric. food Chem.,* **28**: 337-339.

DUMAS, T. & BOND, E.J. (1974) Separation of phosphine from odour-producing impurities. *J. stored Prod. Res.,* **10**: 67-68.

DUMAS, T. & BOND, E.J. (1981) Method of trapping low levels of phosphine at ambient temperature for gas-chromatographic analysis. *J. Chromatogr.,* **206**(2): 384-386.

EPPO (1975) *Guidelines for the development and biological evaluation of rodenticides,* Paris, European and Mediterranean Plant Protection Organisation (Bulletin No. 5(1)).

FITZPATRICK, R.J., MCGIRR, J.L., & PAPWORTH, D.S. (1955) The toxicity of rodenticides. *Vet. Rec.,* **67**: 142-145.

FLUCK, E. (1973) The chemistry of phosphine. *Fortschr. chim Forsch.,* **35**: 1-64.

FLUCK, E. (1976) The odor threshold of phosphine. *J. Air Pollut. Control Assoc.,* **26**(8): 795.

FRANK, R. & RIPPEN, G. (1986) [*Content of phosphine in the atmosphere,*] Frankfurt am Main, Batelle-Institut e.v. (in German).

FREYBERG, W. (1979) *Acute toxicity of Detia^R pellets to bluegill (Lepomis macrochirus),* Washington DC, US Environmental Protection Agency (Unpublished report).

FRITZ, B., LORENZ, K., STEINERT, W., & ZELLNER, R. (1982) Laboratory kinetic investigations of the tropospheric oxidation of selected industrial emissions. In: Versino, B. & Ott, H., ed.

Physico-chemical behaviour of atmospheric pollutants, Dordrecht, Reidel, pp. 192-202.

FURMAN, N.H., ed. (1962) *Standard methods of chemical analysis*, 6th ed., Princeton, New Jersey, D. van Nostrand Company Inc., **Vol.** 1, pp. 821-823.

FURUNO, J., SUGAWARA, N., & IKEDA, H. (1976) A fatal case poisoned by hydrogen phosphide and the simultaneous separation of inorganic gases by gas chromatography. *Bull. Yamaguchi med. Sch.*, **23**(1-2): 49-58.

GESSNER, O. (1937) Fatal phosphine poisoning from "Delicia" grain weevil fumigant (aluminium phosphide). *Samml. Vergiftungs-fällen*, **8**: B-79.

GILL, J.E. & REDFERN, R. (1983) Laboratory tests of seven rodenticides for the control of *Meriones shawi*. *J. Hyg. (Lond.)*, **91**: 351-357.

HABASHI, F. & ISMAIL, M.I. (1975) Health hazards and pollution in the metallurgical industry due to phosphine and arsine. *CIM Bull.*, **August**: 99-103.

HACKENBERG, U. (1972) Chronic ingestion by rats of standard diet treated with aluminium phosphide. *Toxicol. appl. Pharmacol.*, **23**: 147-158.

HALLERMANN, W. & PRIBILLA, O. (1959) [Fatal poisoning with phosphine.] *Arch. Toxikol.*, **17**(4): 219-242 (in German).

HARGER, R.N. & SPOLYAR, L.W. (1958) Toxicity of phosphine with a possible fatality from this poison. *Am. Med. Assoc. Arch. Ind. Health*, **18**: 497-504.

HILL, E.F., HEATH, R.G., SPANN, J.W., & WILLIAMS, J.D. (1975) Lethal dietary toxicities of environmental pollutants to birds. *Wildlife*, **191**: 1-61.

HILTON, H.W. & ROBISON, W.H. (1972) Fate of zinc phosphide and phosphine in the soil-water environment. *J. agric. food Chem.*, **20**(6): 1209-1213.

HOLE, B.D. (1981) Variation in tolerance of seven species of stored product Coleoptera to methyl bromide and phosphine in strains from twenty-nine countries. *Bull. entomol. Res.*, **71**(2): 299-306.

HOLE, B.D., BELL, C.H., MILLS, K.A., & GOODSHIP, G. (1976) The toxicity of phosphine to all developmental stages of thirteen species of stored product beetles. *J. stored Prod. Res.*, **12**: 235-244.

HUGHES, J.G. & JONES, A.T. (1963) Estimation of phosphine in air. *Ind. Hyg. J.*, **March-April**: 164-167

HUNTER, D. (1978)   *The diseases of occupations*, 6th ed., London, Hodder and Stoughton.

IKEDA, S. (1971) [Effect of zinc phosphide on *Coturnix coturnix japonica.*] *Ringyo Shikenjo Kenkyu Hokuko*, **238**: 141-148 (in Japanese).

IKIMANOVA, G.K. (1977) [Porphyrin metabolism in experimental phosphorus poisoning.] *Zdravookhr. Kaz.*, 1: 39-42 (in Russian).

IMO (1980)   *Recommendations on the safe use of pesticides in ships*, London, International Maritime Organization (UN IMO Circular No. 298, revised).

IRPTC (1985)   *Treatment and disposal methods for waste chemicals*, Geneva, International Register of Potentially Toxic Chemicals, United Nations Environment Programme, 303 pp (Data Profile Series No. 5).

JACKSON, J.R. & ELIAS, E. (1984) Hepatic function and exposure to phosphine and white phosphorus. In: *Proceedings of the 12th International Congress on Occupational Health in the Chemical Industry, Dublin, Ireland, 9-14 September 1984*, pp. 219-228.

JANDA, J. (1973) Danger of rodenticides containing zinc phosphide to wild life. *Agrochemia*, 13(3): 85-86.

JOKOTE, Ch. (1904) [Studies on hydrogen phosphine.] *Arch. Hyg. Bakteriol.*, **49**: 275-306 (in German).

JONES, A.T., JONES, R.C., & LONGLEY, E.O. (1964) Environmental and clinical aspects of bulk wheat fumigation with aluminium phosphide. *Am. Ind. Hyg. Assoc. J.*, 25: 376-379.

KADKOL, S.B. & JAYARAJ, P. (1967) Effect of phosphine-fumigated rice on the growth of albino rats. *Rep. Cent. Food Technol. Res. Inst. (Mysore)*, 2: 6.

KAGAN, YU.S. (1985)   *Principles of pesticide toxicology*, Moscow, Centre of International Projects, GKNT, 176 pp.

KAMEL, A.H., FAM, E.Z., MAHDY, M.T., & SHELTAWY, E.M. (1973) The phytotoxic effect of repeated fumigation of certain cereal crops. *Bull. Entomol. Soc. Egypt (Econ. Ser.)*, 7: 57-62.

KAMEL, A.H., FAM, E.Z., MAHDY, M.T., & SHELTAWY, E.M. (1974) The phytotoxic effect of carbon bisulphide, methyl bromide and

hydrogen phosphide on the germination of seeds of certain cereal field crops. *Bull. Entomol. Soc. Egypt (Econ. Ser.)*, **8**: 75-80.

KAO, C. (1981) Determination of aluminium phosphide in fumigants. *J. food Sci.*, **46**: 1632-1634.

KASHI, K.P. (1975) A mixed indicator strip for phosphine detection. *Pestic. Sci.*, **6**: 511-514.

KASHI, K.P. (1981a) Toxicity of phosphine to five species of stored-product insects in atmospheres of air and nitrogen. *Pestic. Sci.*, **12**: 116-122.

KASHI, K.P. (1981b) Response of five species of stored-product insects to phosphine in oxygen-deficient atmospheres. *Pestic. Sci.*, **12**: 111-115.

KASHI, K.P. (1982) Dose-mortality responses of five species of stored-products insects to phosphine. *Int. Pest Control*, **March/April**: 46-49.

KASHI, K.P. & CHEFURKA, W. (1976) The effect of phosphine of absorption and circular dichroic spectra of cytochrome-c and cytochrome-c oxidase. *Pestic. Biochem. Physiol.*, **6**: 350-362.

KASHI, K.P. & MUTHU, M. (1975) A mixed indicator strip for phosphine detection. *Pestic. Sci.*, **6**: 511-514.

KIM, H.H., KIM, K.H., & HONG, S.S. (1977) Effect of pesticides on exocrine pancreas and liver. *Yonse Uidea Nonmunjip*, **10**(2): 170-180.

KLIMMER, O.R. (1969) [Contribution on poisoning by phosphine. On the question of sub-chronic phosphine intoxication.] *Arch. Toxikol.*, **24**: 164 (in German).

KOZHEMYAKIN, N.G., BUTYAGIN, V.N., BASHMURIN, A.F., & POTAPENKO, M.I. (1971) [Veterinary-sanitary evaluation of the carcasses and organs of animals which had to be killed in relation to their contamination with zinc phosphide.] *Sb. Rab. Leningrad Vet. Inst.*, **32**: 231-236 (in Russian).

KUHN, H., MAREK, J., & REIF, H. (1971) [Fumigation of tobacco with phosphine.] *Fachliche Mitt. Austria Tabakwerke AG*, **12**: 191-203 (in German).

LADOU, J. (1983) Potential occupational health hazards in the microelectronics industry. *Scand. J. Work. environ. Health*, **9**: 42-46.

LEESCH, J.G. (1982) Accuracy of different sampling pumps and detector tube combinations to determine phosphine concentrations. *J. econ. Entomol.*, **75**: 899-905.

LEESCH, J.G. (1984) Fumigation of lettuce: efficacy and phyto-toxicity. *J. econ. Entomol.*, **77**(1): 142-150.

LOWE, E.J. (1971) Phosphorus hydrides and phosphonium compounds. In: *Mellor's comprehensive treatise on inorganic and theoretical chemistry*, White Plains, New York, Longman, **Vol. 8**, Suppl. 3.

LUCK, H. & ALLEMANN, A. (1984) Control of insect infestation during storage of dairy products. Fumigation with hydrogen phosphide. *S. Afr. J. dairy Technol.*, **16**(3): 103-107.

LUTZMANN, L., KEINITZ, M., & KLOSTERKOTTER, W. (1963) [Emission of phosphine during transport of ferrosilicon.] *Med. Welt*, **58**: 114-116 (in German).

MCGIRR, J.L. (1953) Poisoning of livestock by the newer rodenticides, insecticides and weed killers. In: *Proceedings of the XVth International Veterinary Congress, Stockholm, 9-15 August, Part 1*, **Vol. 1**, pp. 479-480.

MCMAHON, R. & FORESE, F.F. (1983) *Hydride gas detecting tape*, Glenview, Illinois, MDA Scientific Inc. (UK Patent Application GB 2 108 662A).

MARSHALL, E.F. (1981) Efficacy variations of rodent baits with the same active ingredient. *Pest Control*, **January**: 22-23.

MATHEW, G.C. (1961) The production of phosphine while machining spheroidal graphite iron. *Ann. occup. Hyg.*, **4**: 19-35.

MEISSNER, R. (1924) [The toxicology of hydrogen phosphide.] *Z. gesamte Med.*, **42**: 267 (in German).

MEREDITH, C. (1981) *Toxicological studies on zinc phosphide*, Birmingham, University of Birmingham, Department of Bio-chemistry (M.Sc. Thesis).

MINCHEV, T. & DIMITROV, D. (1970) [Early changes in the serum of rabbits poisoned with zinc phosphide.] In: Vasilev, R.A., ed. [*Nauch. Tr. Sud. Med. Deontol.*,] Sofia, Meditsina i Fizkultura, pp. 33-38 (in Bulgarian).

MUELLER, W. (1940) [Toxicity of phosphine (animal experiments). Part 1. Acute and subacute toxicity.] *Arch. exp. Pathol. Pharmakol.*, **195**: 184-193 (in German).

MUTHU, M., KRISHNAKUMARI, M.K., MURALIDHARA, V., & MAJUMDER, S.K. (1980) A study on the acute inhalation toxicity of phosphine to albino rats. *Bull. environ. Contam. Toxicol.*, **24**: 404-410.

NAKAKITA, H. & WINKS, R.G. (1981) Phosphine resistance in immature stages of a laboratory selected strain of *Tribolium castaneum* Herbst (Coleoptera: Tenebrionidae). *J. stored Prod. Res.*, **17**: 43-52.

NAKAKITA, H., KATSUMATA, Y., & OZAWA, T. (1971) The effect of phosphine on the respiration of rat liver mitochondria. *J. Biochem.*, **69**: 589-593.

NATARAJAN, T. & BAGYARAJ, D.J. (1984) Fumigation effect on microflora and viability of blackgram and fieldbean seeds. *Pesticides*, **18**(4): 40-42.

NAUS, A. (1982) [Olfactory thresholds of some industrial compounds.] *Prac. Lek.*, **34**(6-7): 217-218 (in Polish).

NETHERLANDS, WERKGROEP VAN DESKUNDIGEN VAN DE NATIONALE MAC-COMMISSIE. (1984) [*Report on the TLV of phosphine*,] 2nd ed., Voorburg, Working Group of the National TLV Committee, 27 pp (Report No. 1/80) (in Dutch).

NEUBERT, D. & HOFFMEISTER, I. (1960) Changes in intermediary metabolism following exposure to hydrogen phosphide. *Naunyn-Schmiedeberg's Arch. exp. Pathol. Pharmakol.*, **239**: 219-233.

NOACK, S., VON & REICHMUTH, C. (1981) [Determination of limits for injuring animals and plants by phosphine and methyl bromide. I. Studies on *Drosophila melanogaster*. *Anz. schädlingskd. Pflanzenschutz Umweltschutz*, **54**: 23-27 (in German).

NOACK, S., VON & REICHMUTH, C. (1982a) [Quantities of phosphine, methyl bromide and hydrocyanic acid used in stored products protection in the FRG from 1975-1977.] *Nachrichtenbl. Dtsch. Pflanzenschutzd.*, **34**(2)S: 17-21 (in German).

NOACK, S., VON & REICHMUTH, C. (1982b) [Determination of limits for injuring animals and plants by phosphine or methyl bromide. II. Studies on water cress and lettuce *Lactuca sativa capitata*.] *Anz. schädlingskd. Pflanzenschutz. Umweltschutz*, **55**: 57-59 (in German).

NOACK, S., VON & WOHLGEMUTH, R. (1985) [Phosphine residues in hazelnuts, soybeans and wheat after fumigation with non-constant concentrations.] *Z. Lebensm. Unters. Forsch.*, **180**: 101-108 (in German).

NOACK, S., VON, REICHMUTH, C., & WOHLGEMUTH, R. (1983) [Relationship of PH₃ residues after fumigation to concentration, time of exposure and length of storage.] *Z. Lebensm. Unters. Forsch.*, **177**: 87-93 (in German).

NOACK, S., VON, REICHMUTH, C., & WOHLGEMUTH, R. (1984a) [Decomposition of phosphine in treated foods related to storage temperature and aeration.] *Z. Lebensm. Unters. Forsch.*, **178**: 31-37 (in German).

NOACK, S., VON, REICHMUTH, C., & WOHLGEMUTH, R. (1984b) [A half-empirical mathematical model for calculation of PH₃-residues in hazelnuts fumigated with phosphine.] *Z. Lebensm. Unters. Forsch.*, **178**: 97-103 (in German).

NOWICKI, T.W. (1978) Gas-liquid chromatography and flame photometric detection of phosphine in wheat. *J. Assoc. Off. Anal. Chem.*, **61**(4): 829-836.

PAZYNICH, V.M., MAZUR, I.A., & PODLOZNYI, A.V. (1984) [Experimental substantiation and prediction of maximum atmospheric phosphine concentrations differentiated by time.] *Gig. i Sanit.*, **1984**(1): 13-15 (in Russian).

PRICE, N.R. (1984) Active exclusion of phosphine as a mechanism of resistance in *Rhyzopertha dominica* F (Coleoptera: Bostrychidae). *J. stored Prod. Res.*, **20**(3): 163-168.

PRICE, N.R. & DANCE, S.J. (1983) Some biochemical aspects of phosphine action and resistance in three species of stored product beetles. *Comp. Biochem. Physiol.*, **76C**(2): 277-281.

PRICE, N.R., MILLS, K.A., & HUMPHRIES, L.A. (1982) Phosphine toxicity and catalase activity in susceptible and resistant strains of the lesser grain borer (*Rhyzopertha dominica*). *Comp. Biochem. Physiol.*, **73C**: 411-413.

RANGASWAMY, J.R. (1984) Simple spectrophotometric method for determination of phosphine residues in wheat. *J. Assoc. Off. Anal. Chem.*, **67**(1): 117-122.

RANGASWAMY, J.R. & MUTHU, M. (1985) Spectrophotometric determination of phosphine residues in rice. *J. Assoc. Off. Anal. Chem.*, **68**(2): 205-208.

REBMANN, Z. (1933) [On a new pesticide (Fosfolon).] *Gesundheitstech. Städtehyg. ver. Städtische Tiefbau*, **5**: 280-284 (in German).

REICHMUTH, C., ed. (1985) [Response of the granary weevil *Sitophilus granarius* L. (Coleoptera: Curculionidae) to gradually increasing and decreasing phosphine concentrations.] *Anz. schädlingskd. Pflanzenschutz Umweltschutz*, **58**(1): 10-16 (in German).

REICHMUTH, C., ed. (1986) The significance of changing concentrations in toxicity of phosphine. In: *Proceedings of the GASGA Seminar on Fumigation Technology in Developing Countries, Slough, United Kingdom, 8-21 March*, London, Overseas Development Administration, Tropical Development and Research Institute. pp. 88-98.

REICHMUTH, C., NOACK, S., & WREDE, A. (1981) [Emission of phosphine caused by fumigation for stored products protection.] *Nachrichtenbl. Dtsch. Pflanzenschutzd.*, **33**(9): 132-136 (in German).

ROALDSNES, J. (1982) [Phosphine poisoning due to machining of ductile iron.] *Norsk. bedv. h. tj.*, **3**: 12-16 (in Norwegian).

ROBINSON, J.R. (1972) Residues containing phosphorus following phosphine treatment: measurement by neutron activation. *J. stored Prod. Res.*, **8**: 19-26

ROBINSON, J.R. & BOND, E.J. (1970) The toxic action of phosphine. Studies with ^{32}PH$_3$: terminal residues in biological materials. *J. stored Prod. Res.*, **6**: 133-146.

ROBISON, W.H. & HILTON, H.W. (1971) Gas chromatography of phosphine derived from zinc phosphide in sugarcane. *J. agric. food Chem.*, **19**(5): 875-878.

ROHRLICH, M. & MEUSER, F. (1970) [Studies on grain fumigated with hydrogen phosphide. III. Communication: biochemical aspects of phosphine fumigation.] *Getreide Mehl*, **20**(1): 1-8 (in German).

RUSCHEL, A.P. & DA COSTA, W.F. (1966) [Symbiotic fixing of atmospheric nitrogen in the french bean (*Phaseolus vulgaris* L). III. Effect of some insecticides and fungicides.] *Pesqui. Agropecu. Bras.*, **1**: 147-149 (in Portugese).

SAEED, T. & ABU-TABANJA, R. (1984) Determination of phosphine: comparison of rates of desorption by purge-and-trap method and by sulfuric acid treatment. *J. Assoc. Off. Anal. Chem.*, **68**(1): 61-64.

SATO, K. & SUWANAI, M. (1974) Absorption of hydrogen phosphide to cereal products. *Appl. Entomol. Zool.*, **9**(3): 127-132.

SAYVETZ, T.A. (1982) *Possible phosphine inhalation related to warehouse fumigation*, Richmond, Virginia, Virginia State Health Department, Office of Health Protection and Environmental Management, 6 p.

SCHITOSKEY, F. (1975) Primary and secondary hazards of three rodenticides to kit fox. *J. wildl. Manage.*, **39**(2): 416-418.

SCHULZ, H. (1887) [Correction concerning the toxicity of phosphorus compounds containing oxygen.] *Arch. exp. Pathol.*, **23**: 150-151 (in German).

SCHULZ, H. (1890) [Concerning phosphine.] *Arch. exp. Pathol. Pharmakol.*, **27**: 314-335 (in German).

SHAHEEN, D.G., BRAGG, L.A., STANOVICK, R.P., & SULLIVAN, J.B. (1983) Phosphine detector tubes as passive dosimeters for use with metal phosphide fumigants. *Tob. Int.*, **185**(20): 36-39.

SHIVANANDAPPA, T., RAMESH, H.P., & KRISHNAKUMARI, M.K. (1979) Rodenticidal poisoning of non-target animals: acute oral toxicity of zinc phosphide to poultry. *Bull. environ. Contam. Toxicol.*, **23**(4-5): 452-455.

SINGH, K.N., SRIVASTAVA, B.P., & NATH, G. (1983) Phosphine residues and its effect on the germination of stored pulses. *Indian J. Entomol.*, **45**(1): 71-80.

SRI (1982) *Electronic chemicals*, Menlo Park, California, Standford Research International, pp. 40-41.

SRIDHARA, S. (1983) Rodenticide induced bait aversion and neophobia in *Tatera indica cuvieri*. *Z. angew. Zool.*, **70**(4): 429-440.

STEPHENSON, J.B.P. (1967) Zinc phosphide poisoning. *Arch. environ. Health*, **15**: 83-88.

TERZIC, Lj. (1981) Quantitative method for the determination of zinc phosphide in technical products. *Acta vet. (Beograd)*, **31**(5-6): 279-288.

TIETJEN, H.P. (1976) *Zinc phosphide: its development as a control agent for black-tailed prairie dogs*, Washington DC, US Department of the Interior, Fish and Wildlife Research Centre (Special Scientific Report Wildlife No. 195).

TKACHUK, R. (1971) Phosphine fumigation of wheat - residue formation. *Cereal Sci. Today*, **16**: 307.

TKACHUK, R. (1972) Phosphorus residues in wheat due to phosphine fumigation. *Cereal Chem.*, **49**: 258-267.

TRIMBORN, H. & KLIMMER, O. (1962) [Experimental studies on chemical changes in blood pigments caused by hydrogen phosphide *in vitro.*] *Arch. int. Pharmacodyn.*, **137**(3-4): 331-347 (in German).

UNDERWOOD, J.G. (1972) The radiochemical analysis of phosphine residues in fumigated tobaccos. *Tob. Sci.*, **October 13**: 47-49.

URGA, K. (1983) Methyl bromide and phosphine residues in cereal grains and pulses. *Ethiop. med. J.*, **21**: 79-83.

US CFR (1981) Code of federal regulations, 29(1910): 673.

US EPA (1981) *Pesticide registration standard: aluminium phosphide*, Washington DC, US Environmental Protection Agency (PB 82-137514).

US EPA (1982) *Pesticide registration standard: magnesium phosphide*, Washington DC, US Environmental Protection Agency (PB 82-195777).

US EPA (1983) Hazardous waste management system: identification and listing of hazardous waste. *Fed. Reg.*, **48**(37): 7714-7716.

US EPA (1984) Hazardous waste management system: identification and listing of hazardous waste. *Fed. Reg.*, **49**(92): 19922-19923.

US EPA (1985a) Aluminium phosphide/magnesium phosphide: tolerances for residues. *Code Fed. Reg.*, **40**: 180, 225, 291-292, 333, 375.

US EPA (1985b) *EPA chemical profile: phosphine*, Washington DC, US Environmental Protection Agency.

US EPA (1986) *Guidance for the re-registration of pesticide products containing aluminium phosphide, February 1986. An amendment to the registration standard issued in October 1981*, Washington DC, US Environmental Protection Agency, Office of Pesticides and Toxic Substances (PB 84-241405).

US NIOSH (1977) *National occupational hazard survey*, Cincinnati, Ohio, National Institute of Occupational Safety and Health, **Vol.** 3 (Publication No. 78-114).

US NIOSH (1979) *Ten NIOSH analytical methods: phosphine*, Cincinnati, Ohio, National Institute for Occupational Safety

and Health, Division of Physical Sciences and Engineering, 42 pp.

VAN WAZER, J.R., ed. (1961) *Phosphorus and its compounds: technology, biological functions and applications,* New York, Interscience Publishers Inc., **Vol. II,** p. 957.

VAN WAZER, J.R. (1982) Phosphorus and the phosphides. In: Kirk-Othmer, ed. *Encyclopaedia of chemical technology,* 3rd ed., New York, John Wiley and Sons, **Vol. 17,** pp. 473-489.

VDOVENKO, I.D., VAKULENKO, L.I., YAKOVLEVA, L.A., SHEDENKA, L.I., BERESLAVSKAYA, A.N., & YATSENKO, V.S. (1984) [Effect of surfactants on phosphine formation in steel pickling.] *Ukr. Khim. Zh.,* **50**(4): 418-421 (in Russian).

VERGA, L. & BELLONI, F. (1958) [Over a case of purpura due to empoisonment of phosphine (a haemato-clinical and histochemical study.] *Rass. Med. ind. Ig. Lav.,* **27**(3): 230-241 (in Italian).

VERSTUYFT, A.W. (1978) Sampling and analytical methods for phosphine - a review. *Am. Ind. Hyg. Assoc. J.,* **39**: 431-437.

VINSJANSEN, A. & THRANE, K.E. (1978) Gas-chromatographic determination of phosphine in ambient air. *Analyst,* **103**: 1195-1198.

WARITZ, R.S. & BROWN, R.M. (1975) Acute and subacute inhalation toxicities of phosphine, phenylphosphine and triphenylphosphine. *Am. Ind. Hyg. Assoc. J.,* **36**: 452-458.

WEBLEY, D.J., HARRIS, A.H., & KILMINSTER, K. (1981) The use of a portable gas analyser to measure phosphine concentrations in experimental fumigations in the tropics. *Int. pest Control,* **March/April:** 47-50.

WILSON, A. (1971) The metal phosphides. In: *Mellor's comprehensive treatise on inorganic and theoretical chemistry,* White Plains, New York, Longman, **Vol. VIII,** Suppl. III.

WILSON, R., LOVEJOY, F.H., JAEGER, R.J., & LANDRIGAN, P.L. (1980) Acute phosphine poisoning aboard a grain freighter. Epidemiologic, clinical and pathological findings. *J. Am. Med. Assoc.,* **244**(2): 148-150.

WINKS, R.G. (1970) Phosphine fumigation of farm-stored tobacco. *Queensland agric. J.,* **November**.

WINKS, R.G. (1984) The toxicity of phosphine to adults of *Tribolium castaneum* (Herbst): time as a dosage factor. *J. stored Prod. Res.*, **20**(1): 45-56.

WINKS, R.G. (1985) The toxicity of phosphine to adults of *Tribolium castaneum* (Herbst): phosphine-induced narcosis. *J. stored Prod. Res.*, **21**(1): 25-29.

WHO (1973) WHO Technical Report Series, No. 513 (Twentieth Report of the Expert Committee on Insecticides), Geneva, World Health Organization, p. 39.

WHO/FAO (1972) *1971 Evaluations of some pesticide residues in food*, Geneva, World Health Organization, pp. 289-295 (WHO Pesticide Residue Series, No. 1).

WHO/FAO (1976) *Data sheet on pesticides No. 24: zinc phosphide*, Geneva, World Health Organization (VBC/DS/77.24).

WHO/FAO (1980) *Data sheet on pesticides No. 46: phosphine*, Geneva, World Health Organization (VBC/DS/80.46).

WORTHING, C.R. & WALKER, S.B., ed. (1983) *The pesticide manual: a world compendium*, 7th ed., London, British Crop Protection Council.

ZIEMER, U. (1963) [Fatal hydrogen phosphide poisoning on a freighter.] *Zbl. Arbeitsmed.*, **13**: 38-39 (in German).

ZIPF, K.E., ARNDT, TH., & HEINTZ, R. (1967) [Clinical observation of a case of Phostoxin poisoning.] *Arch. Toxikol.*, **22**(4): 209-222 (in German).

ZUTSHI, M.K. (1966) Effect of aluminium phosphide (Phostoxin) on the germination of certain cereal and vegetable seeds. *Bull. grain Technol.*, **4**(4): 197-199.

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