Original Article

Effects of vitamin E administration on phostoxin-induced changes in the kidney of adult Wistar rats

Ibegbu A. O, Babatunde A. O, Alatise A. T, Dassah S. J, Umana U. E, Hamman W. O, Musa S. A

Department of Human Anatomy, Faculty of Medicine, Ahmadu Bello University Zaria, Kaduna State, Nigeria

Abstract

Aim: The effect of vitamin E on phostoxin-induced changes in biochemical parameters and the kidney of adult Wistar rats were studied. **Materials and Methods:** Thirty adult Wistar rats of both sexes were randomly separated into six groups of five rats each. Group 1 was the Control and was given normal saline. Group 2 was exposed to phostoxin for 3 hours per day with vitamin E and Group 3 was exposed to phostoxin for 1½ hours with vitamin E. Groups 4 and 5 were treated with phostoxin only on exposure time of 1½ and 3 hours respectively while Group 6 was treated with vitamin E only. The rats were exposed to phostoxin through inhalational method for 7 days and at the end of the exposure period, the rats were sacrificed. The blood and tissues were collected for analysis and were processed for histological studies. **Results:** The results showed significant changes in body weight of the rats ($P < 0.05$) while there was a significant increase in weight of the kidneys in Groups 3 and 6 when compared to the Control ($P < 0.05$). The results of the biochemical parameters Na^+, K^+, Cl^-, urea, creatinine and HCO3 showed a significant difference ($P < 0.05$) between the control and the experimental groups. The results of histological examination of the kidney showed changes in animals exposed to phostoxin when compared to the Control animals. **Conclusion:** The changes in the kidney depend on the duration of the exposure time while vitamin E administration has been shown to have some level of protection on phostoxin-induced toxicity on the kidney tissues and biochemical parameters of the adult Wistar rats.

Key words: Biochemical parameters, kidney, oxidative enzymes, phostoxin, vitamin E, Wistar rats

INTRODUCTION

Aluminium phosphide is an inorganic phosphide used to control insects and rodents in a variety of settings. It is mainly used as an indoor fumigant during crop transport, storage or in processing facilities for both food and non-food crops. It may also be used as outdoor fumigant for burrowing rodent and in mole control or in bait for rodent control in crops (Meister, 1992; O'Malley *et al*., 2013).

Aluminium phosphide is available in pallet and tablet form, and also available in porous blister packs, sachets or as dusts. Phostoxin is one of the market names for aluminium phosphide products which emit a colourless gas and is odourless when pure, but the technical product has a foul smell (Degesch, 2011). Phostoxin is flammable and explosive in air and can auto-ignite at ambient temperature (Sudakin, 2005; Degesch, 2011). It is soluble in water and in most organic solvents and supplied in cylinders either as pure phosphide or diluted with nitrogen (Shaheen, 1996; Sudakin and Power, 2007). Phostoxin may be formulated as 55% active ingredient along with aluminium carbamate and inert ingredients (Gehring *et al*., 1991; Easterwood *et al*., 2010).

Address for correspondence:
Dr. Ibegbu A.O,
Department of Human Anatomy, Faculty of Medicine,
Ahmadu Bello University Zaria, Kaduna State, 81006, Nigeria.
E-mail: aoibegbu@yahoo.com

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The application of phostoxin must be long so as to establish adequate control of insect pests infesting the communities being treated. The fumigation period should be long enough to give room for appropriate reaction of phostoxin with moisture so that minimal or un-reacted aluminium phosphide remains (Easterwood *et al*., 2010). This will minimise water exposure during further storage and also will minimise the hazardous effect during disposal of partially spent aluminium phosphide that is left after fumigation. The fumigator exposure time to phostoxin must not exceed 8-hour time weighted average of 0.3 ppm or 15-minute short-term exposure limit of 1.0 ppm phosphine (USDHHS, 1994; O'Malley *et al*., 2013).

Breathing-in low level of phosphine gas can cause headache and medium level of exposure can cause nausea, dizziness and tightness of the chest. While higher exposure levels can cause diarrhoea, abdominal pain, vomiting, chest pain, pulmonary oedema, irregular heartbeat, shock, convulsions, coma and death (USEPA,1992; Proudfoot, 2009; Easterwood *et al*., 2010). At a concentration of 50 ppm, phosphine is immediately dangerous to life and health (W.H.O., 1998; Shadnia *et al*., 2008). It is used to fumigate bagged, packaged or treated cereals, grasses, sorghum or small legume seeds destined for planting use only. Phostoxin provides an effective alternative to traditional control methods of moles, rabbits and rats (W.H.O., 1988; Shadnia *et al*., 2008).

Antioxidants protect the body cells against the effects of free radicals and free radicals are produced when the body breaks down food or exposed to substances like tobacco smoke and radiation. Free radicals can damage cell and may play a role in heart diseases, cancer and other diseases. Antioxidant substances include vitamin E, vitamin C, vitamin A, lycopene and beta-carotene (NIHODS, 2012). Vitamin E is very important in the treatment of many diseases of the circulatory system, the treatment of heart attacks, angina, arthero-sclerosis, rheumatic fever, acute and chronic rheumatic heart diseases, congenital heart diseases, intermittent claudication, varicose veins, thrombophlebitis, high blood pressure, diabetes and burns (Wilfrid *et al*., 2012). It has been shown that exposure to phosphine gas can be health and life threatening and may invariably result to death (Proudfoot, 2009; Easterwood *et al*., 2010), but the study of the effects on the kidney and biochemical parameters have not been done to understand the cause of death by this chemical agent. The aim of the present study was to evaluate the effects of antioxidant (Vitamin E) on phostoxin-induced changes in the kidney and biochemical parameters in adult Wistar rats.

MATERIALS AND METHODS

Chemicals

Phostoxin tablet used was manufactured by D and D Holdings Inc., USA. The tablets weigh 3 g and releases 1 g of phosphine gas, was purchased from Agro Allied Store, Zaria, Kaduna State, Nigeria. It takes an average of 3 days to completely decompose leaving a gray-white powder of aluminium hydroxide and inert ingredients of the ammonium carbarnate (Degesch, 2011). Vitamin E manufactured by Medizen USA was purchased from Beautiful Gate Pharmaceutical Store, Zaria Kaduna State-Nigeria. Vitamin E used was soft gelatin capsules containing 100 mg of vitamin acetate.

Ethical Approval

Application for ethical approval was made, considered and obtained from the Ahmadu Bello University Ethical Committee.

Experimental Animals

Thirty Wistar rats were obtained and acclimatized for 3 weeks in the animal house of Department of Human Anatomy, Faculty of Medicine, Ahmadu Bello University Zaria. The animals of an average weight of 140 g were randomly separated into six groups of five animals each and were fed with standard pellets and water was provided *ad-bilitum*.

Phostoxin was given through inhalation by using lightly suspended cotton wool in an enclosed box for 1.5 hours and 3 hours respectively for a period of 7 days as shown in Figure 1. Vitamin E was administered at 800 mg/kg body weight, orally by means of insulin syringe for animals in Groups 2, 3 and 6.

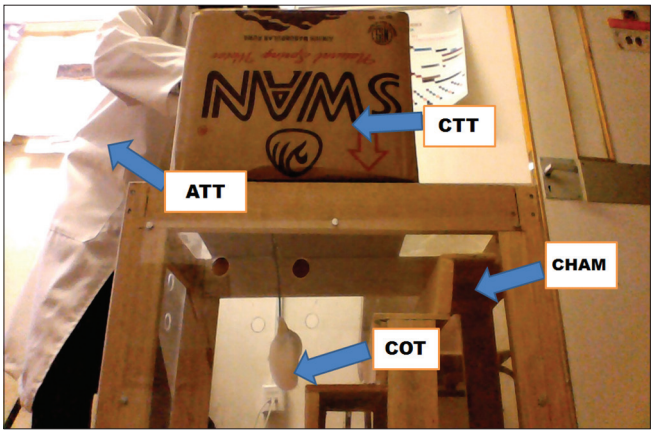

Figure 1: Showing the Laboratory set for the exposure of the animals to phostoxin in a lightly suspended Cotton wool (COT) from an over head carton (CTT) within the exposure Chamber (CHAM) with the Researcher (ATT) during the exposure time

Experimental Protocol

The animals in Group 1 were used as the Control and given distil water, Group 2 was exposed to phostoxin for 1.5 hours and administered vitamin E at a concentration of 112 mg/kg body weight, Group 3 was exposed to phostoxin for 3 hours and administered with vitamin E at concentration of 112 mg/kg body weight, Group 4 was exposed to phostoxin only for 3 hours, Group 5 was exposed to phostoxin only for 1.5 hours and Group 6 was administered with vitamin E only at the concentration of 112 mg/kg body weight. The administration lasted for 7 days and on the ninth day, the rats were humanely sacrificed after anesthetizing them with chloroform and the blood was collected in properly labelled EDTA bottles for biochemical analysis. The kidney tissues were harvested and weighed using a digital weighing balance. The tissues were fixed in 10% buffered formalin, processed and stained for histological analysis using haematoxylin and eosin (H and E) for general tissue architecture, and Masson Trichrome special stain for detailed study of the tissues.

Biochemical Assay

The levels of biochemical parameters were determined by the use of an auto analyzer made by Roche-Hittachi, Japan, using commercial assay kits manufactured by Roche, Basel, Switzerland.

Estimation of Plasma Proteins

The levels of plasma proteins in the blood was estimated using Okutucu *et al*. (2007) method of which the level of plasma protein could be altered in kidney diseases. The estimation of uric acid, urea and creatinine values in the blood was done according to the methods of Sembulingam and Sembulingam (2010). The blood level of these substances has been shown to increase in renal diseases and renal failure (Sembulingam and Sembulingam, 2010).

Estimation of Oxidative Parameters

Oxidative stress markers such as catalase activity, superoxide dismutase (SOD) activity, assessment of lipid peroxidation and glutathione (GSH) concentration were studied using the respective assay methods according to the instructions of the manufacturers. Catalase activity was determined using the method described by Sinha (1992). The absorbance was read at 570 nm and Standard cure was made using the absorbance obtained at various levels. SOD activity was determined by the method described by Fridovich (1989) and the absorbance was measured every 30 s up to 150 s at 480 nm. Lipid peroxidation as evidenced by the formation of TBARS was measured by the modified method of Niehaus and Samuelson (1968) as described by Adhikari *et al*. (2009). The absorbance of the pink supernatant was measured against a reference blank using spectrophotometer at 535 nm. Reduced glutathione (GSH) concentration was measured according to the methods by Ellman (1959) as modified by Rajagopalan *et al*. (2004) and Seiler *et al*. (2008). The absorbance was read at 412 nm.

Statistical Analysis

The data were expressed as means ± standard deviation (SD). Differences between group means were estimated using Students' T-test and one-way analysis of variance (ANOVA) followed by *post hoc* Turkey's test using SPSS 12.0 for windows. A $P \leq 0.05$ was considered to be significant.

RESULTS

Physical Observation

The result of the physical observation of the rats showed a statistical significant increase in the body weight of the animals in Groups 2, 3, 4, 5 and 6 when compared to the Control, while there was a significant increase in the kidney weight in Groups 3 and 6 when compared to the Control group. The results showed that the Group exposed to low dose of phostoxin alone had a significant gain in weight than the Group exposed to low dose phostoxin with vitamin E. The result also showed that the Group exposed to high dose of phostoxin alone had a significant gain in weight than the Group exposed to high dose of phostoxin with vitamin E as shown in Table 1.

Biochemical Parameters

The result of the biochemical analysis following phostoxin and vitamin E administration showed an increase and a decrease in some parameters as shown in Table 2. The mean value of sodium ion showed a significant increase in Group 3 when compared to the Control ($P \leq 0.05$).

Table 1: The change in body weight and kidney weight between the control and experimental groups of Wistar rats

Groups	Administration (mg/kg bwt)	Kidney weight (g)	Initial body weight (g)	Final body weight (g)	Change in body weight (g)
1	Control (Distil water)	1.06±0.17	121.4±14.64	136.2±20.26	14.80*
2	3 g of phostoxin tablet (1½ hrs/day) + 800 mg/kg bwt of vit E	1.06±0.23	136.6±29.95	148.2±32.87*	11.60
3	3 g of phostoxin tablet (3 hrs/day) + 800 mg/kg bwt of vit E	1.44±0.30*	134.0±31.62	144.0±29.84*	10.00
4	3 g of phostoxin tablet (3 hrs/day)	1.26±0.31	136.4±34.62	149.4±31.35*	13.00*
5	3 g of phostoxin tablet (1½ hrs/day)	1.10±0.26	137.2±31.46	152.2±35.69*	15.00*
6	800 mg/kg bwt of vit E	2.36±1.83*	127.4±28.27	151.2±28.96*	23.80*

Table showing body weight change and kidney weight of experimental rats *$P \leq 0.05$

The mean value of urea showed a significant increase in Groups 5 and 6, when compared to the Control group ($P \leq 0.05$).The results showed that the mean potassium ion value showed a significant increase in Group 3 when compared to the Control ($P \leq 0.05$), while Groups 2, 5 and 6 showed a non-significant increase when compared to the Control except Group 4 which showed a non-significant decrease when compared to the Control. The mean value of chloride ion showed a significant increase in Groups 2 and 5 when compared to the Control ($P \leq 0.05$). Creatinine mean value showed a significant increase in Groups 4 and 6 when compared to the Control ($P \leq 0.05$). There was a significant decrease in the bicarbonate mean value between the Control and all other Groups ($P \leq 0.05$), except for Group 5 of which the decrease was not significant as shown in Table 2.

Oxidative Parameters

The oxidative stress markers namely the catalase, SOD, glutathione (GSH), and lipid peroxidase (LPO) were accessed in the blood of the experimental animals. The result showed an increase or a decrease in the levels of these parameters in the kidney of the rats depending on the type of the treatment. The results showed a significant decrease in Groups 4 and 5 ($P \leq 0.05$), while Groups 2 and 3 showed a non-significant decrease in the levels of catalase when compared to the Control. The levels of LPO showed a non-significant increase in all the Groups when compared to the. The levels of SOD, showed a significant decrease in Groups 4 and 5 ($P \leq 0.05$), and a non-significant decrease in Groups 2, 3 and 6. However levels of GSH showed a significant decrease in Group 4 and a non-significant decrease in Groups 2, 3, 4, 5 and 6 when compared to the Control as shown in Table 3.

Histological Observations

The result of histological observations from the transverse section of the kidney of the experimental animals showed normal histology of the kidney with normal glomerular architecture and cellular organisation in the Control Group and Group 6 that received vitamin E only, while the Groups that were exposed to phostoxin alone and phostoxin with vitamin E showed different levels of changes in the kidney structures and cellular changes. The results showed dose-dependent changes in the kidney tissues, while the Groups that received high exposure of phostoxin showed more severe pathological changes such as cellular degeneration and altered kidney structure when compared to the low exposed Groups. Also the changes that manifested in the Group that was exposed to phostoxin only were more severe cellular degeneration and glomerular changes than those that occur in Groups that received vitamin E as a supplement.

The results show a transverse section of kidneys in Group 1 animals (Control) showing normal cyto-architecture of the glomerulus, proximal convoluted and distal convoluted tubules as in Plate 1, while Group 2 animals show interlobular vessel damage in their kidney as shown in Plate 2. Plate 3 shows kidney section of Group 3 animals with degenerated glomerular tissues, degenerated tubules and interlobular vessel damage, when compared to the Control Group. Plate 4 shows degeneration of glomerular tissues, degenerative tubules, interlobular vessel damage and necrotic glomerular cells in the kidney of Group 4 animals. Group 5 animals show few degenerated tubules and few interlobular vessel damage in the kidney section as shown in Plate 5, while Group 6 show normal appearance of glomerular tissues, proximal convoluted and distal convoluted tubules as shown in Plate 6.

Table 2: The values of biochemical parameters in Mean±SD of Wistar rats exposed to phostoxin and vitamin E

Groups	Na^+	K^+	Urea	Cl^-	HCO^3	Creatinine
1	144.3±2.5	8.53±0.57	2.90±0.70	112.7±5.0	18.0±2.0	59.0±12.0
2	145.0±3.6	10.50±0.46	2.67±0.40	128.7±4.7*	10.7±2.1	55.0±6.93
3	218.7±116.42*	14.43±7.04	3.13±0.40	107.3±11.6	11.7±3.5	63.0±6.93
4	152.3±3.5	7.40±0.66	6.67±1.12**	118.3±5.1	14.7±1.5	106.0±12.0**
5	150.0±12.1	10.20±3.37	2.57±0.29	123.3±7.5*	17.0±8.5	55.0±6.9
6	150.3±4.5	9.17±1.11	7.17±0.75**	119.0±3.0	12.3±2.1	109.3±7.6**

Table showing the values of biochemical parameters *P<0.05, HCO^3 - Bicarbonate, Cl^- - Chloride ion, SD - Standard deviation, **P value = 0.01 (P≤0.01)

Table 3: Blood serum levels of oxidative stress markers

Groups	Administration	Catalase (µmol/mg)	LPO (nmol/mg)	SOD (U/mg)	GSH (U/mg)
1	Control (Distil water)	12.90±4.82	42.52±4.85	346.60±16.41	26.75±3.11
2	Phostoxin 1½ hrs/day+800 mg/kg bwt of vit E	9.46±1.86	50.37±6.10	287.25±82.38	19.32±5.78
3	Phostoxin 3 hrs/day+800 mg/kg bwt of vit E	10.37±8.87	46.21±18.55	254.75±154.5	18.87±12.34
4	Phostoxin 3 hrs/day	7.59±0.45*	55.39±0.29*	140.95±6.44*	14.77±2.83*
5	Phostoxin 1½ hrs/day	7.71±2.341*	53.11±17.32*	174.50±4.95*	19.00±11.5
6	800 mg/kg bwt of vit E	13.09±3.15	42.00±1.67	306.50±44.55	25.00±10.10

Table showing the levels of oxidative markers in experimental and control groups *P≤0.05, LPO - Lipid peroxidase, SOD - Superoxide dismutase, GSH - Glutathione

DISCUSSION

The present study has shown that the administration of phostoxin has effects on the body weight across all the experimental animals. During the period of exposure, there was observed an increase in food consumption in all the animals in the exposed groups though the result is not shown here. This could be due to the effects of phosphine, a major component of phostoxin, having appetite-boosting effects which could have resulted in the increase in body weight that was observed following exposure to phostoxin (Lall *et al*., 2000). The result from the present study contradicted the study by Newton, *et al*. (1993), who reported the effects of phosphine gas on male and female rats maintained at levels of 0.5, 1.5 and 4.5 mg/m^3 for 6 hours per day over 13 weeks period. The results of studies by other researchers showed that the higher exposed Group showed dose-dependent effects of which there was reduction in the body weight and decreased food consumption (Newton, *et al*., 1993; Sinha *et al*., 2005; Türkez and Toğar, 2013). Warits and Brown (1975) had reported experiment with Charles River rats exposed to phosphine for 4 hours per day for 12 days at a concentration of 5.5 mg/m^3 and the result showed there was loss in weight during the exposure period. The increase in weight in the experimental groups of rats could be the result of weight increment due to the natural growth of the rats as they were young growing rats (Lall *et al*., 2000).

The results of biochemical parameters in this study as in the case of serum levels of urea, creatinine, sodium ion, potassium ion, chloride ion and bicarbonate showed a significant difference in some experimental Groups when compared to the Control which were in agreement with other studies (Lall *et al*., 2000; Shadnia *et al*., 2011). This implied that the kidneys were experiencing some pathological changes in the Experimental groups as shown by the changes in the biochemical parameters (Cullen, 2000; Valmas and Ebert, 2006). This was similar to the work of Newton *et al*. (1993) that reported dose-dependent changes in blood urea, nitrogen and other clinical parameters that were seen across the exposed groups. In the Control group and group that was given vitamin E only, the blood serum level of these biochemical parameters showed normal levels, while the groups that was exposed to phostoxin and vitamin E showed variation in blood serum level of the parameters depending on the exposure time of the phostoxin and vitamin E administered (Saleki *et al*., 2007).

The histological changes in the present study show that the administration of phostoxin alone caused some changes in the kidney compared with the control rats. The kidney sections from the Control and vitamin E-treated Groups showed normal glomeruli and tubular interstitial cells. In contrast, the kidney of phostoxin-treated rats showed marked deleterious histological changes (Arora *et al*., 1995; Shadnia *et al*., 2009). The kidney section showed significant glomerular and tubular degeneration varying from glomerular basement thickening, interstitial inflammation, interlobular vessel disorder, tubular cell swelling, medullary vascular congestion and moderate to severe necrosis (Saleki *et al*., 2007). The vitamin E-treated Group showed normal morphology and architecture of the kidney.

Evidence suggests that various enzymatic and non-enzymatic systems have been developed by mammalian cells to cope with ROS and other free radical. ROS affects the antioxidants defence mechanisms, by reducing the intracellular concentration of GSH and decreases the activity of SOD, CAT and GSH-Px (Seiler *et al*., 2008; Tehrani *et al*., 2013). It was also observed to decrease the detoxification system produced by GSH. The decrease in the level of SOD, CAT and GSH in the kidney of phostoxin-treated rats may be due to the enhanced lipid peroxidation or inactivation of the anti-oxidative enzymes (Arora *et al*., 1995; Sinha *et al*., 2005). The administration of vitamin E prior to phostoxin intoxication protected the antioxidant machineries of the kidney as revealed from enhanced levels of SOD, CAT and GSH activities, increased GSH content and decreased lipid peroxidation. The level of the enzymes, namely SOD, CAT and GSH, was decreased in phostoxin-treated rats which may be as a result of its effect on the antioxidative enzymes (Seiler *et al*., 2008; Turkez and Togar, 2012).

CONCLUSION

This study has demonstrated that phostoxin induced toxic effects and has hazardous effects on the kidney and the study has shown that vitamin E through its marked antioxidant activity coupled with anti-inflammatory effects havehas ameliorative effects on the phostoxin-induced toxicity in the kidney of adult Wistar rats.

REFERENCES

1. Adhikari N.D., Orler R., Chory J., Froehlich J.E., Larkin R.M. (2009). Porphyrins promote the association of Genomes Uncoupled 4 and a Mg-chelatase subunit with chloroplast membranes. J Biol Chem 284(37):24783-96.
2. Arora B., Punia R.S., Kalra R., Chugh S.N., Arora D.R. (1995). Histopathological changes in aluminium phosphide poisoning. J Indian Med Assoc 93(10):380-1.
3. Cullen G. (2000). Pesticide poisoning:Rodenticides. Emerg Nurse 8(7):16-21.
4. Degesch, America Inc. (2011). Phostoxin Tablet and Pellet application manual. Degesch 2011.
5. Easterwood L., Chaffin M.K., Marsh P.S., Porter B., Barr C. (2010). Phosphine intoxication following oral exposure of horses to aluminum phosphide-treated feed. J Am Vet Med Assoc 236 (4):446-50.
6. Ellman G.L. (1959). Tissue's sulthydry groups. Arch Biochem Biophys 82:70-7.

7. Fridovich I. (1986). Biological effects of the superoxide radical. Arch Biochem Biophys 247(1):1-11.
8. Gehring P. J., Nolan R. J., Watanabe P.G., Schumann A.M. (1991). Solvent, fumigants and related compounds, In:Hayes W.J., Laws E.R., Jr, editors. Handbook of Pesticide Toxicology, Academic Press, New York, NY. p.28-45.
9. Lall S.B., Peshin S.S., Mitra S. (2000). Methemoglobinemia in aluminium phosphide poisoning in rats. Indian J Exp Biol 38(1):95-7.
10. Meister R.T., editor. (1992). Farm Chemicals Handbook ‹92. Meister Publishing Company. Willoughby, OH. p.6-13.
11. Newton P.E. (1993). Inhalation toxicity of phosphine in the rat, acute sub-chronic and developmental intal. Toxicology 5(2):223-39.
12. Niehaus W.G., Samuelson B. (1986). Formation of malondialadetiyated from phosholpidararchi donate during micorsornal lipid pervxiodation. Eur J Biochem 6:126-30.
13. NIHODS (2012). National Institute forHealth Office of Dietary Supplements Research Practicum. p.1-5.
14. Okutucu B., Dinçer A., Habib O., Zihnioglu F. (2007). Comparison of five methods for determination of total plasma protein concentration. J Biochem Biophys Methods 70(5):709-11.
15. O›Malley M., Fong H., Sánchez M.E., Roisman R., Nonato Y., Mehler L. (2013). Inhalation of phosphine gas following a fire associated with fumigation of processed pistachio nuts. J. Agromedicine 18(2):151-73.
16. Rajagopalan H., Jallepalli P.V., Rago C., Velculescu V.E., Kinzler K.W., Vogelstein B., *et al.* (2004). Inactivation of hCDC4 can cause chromosomal instability. Nature 428(6978):77-81.
17. Saleki S., Ardalan F.A., Javidan-Nejad A. (2007). Liver histopathology of fatal phosphine poisoning. Forensic Sci Int 166(2-3):190-3.
18. Seiler A., Schneider M., Forester H., Roth S., Wirth E.K., Culmsee C., *et al.* (2008). Glutathione peroxidase 4 senses and translates oxidative stress into 12/15-lipoxygenase dependent- and AIF-mediated cell death. Cell Metab 8(3):237-48.
19. Segbulingam K., Sembulingam P. (2010). Essential of Medical Physiology, 5th ed. Jaypee Brother Medical Publisher Ltd.; New Delhi, India. p.319-21.
20. Shadnia S., Mehrpour O., Abdollahi M. (2008). Unintentional poisoning by phosphine released from aluminum phosphide. Hum Exp Toxicol 27(1):87-9.
21. Shadnia S., Sasanian G., Allami P., Hosseini A., Ranjbar A., Amini-Shirazi N., *et al.* (2009). A retrospective 7-years study of aluminum phosphide poisoning in Tehran:Opportunities for prevention. Hum Exp Toxicol 28(4):8-15.
22. Shadnia S., Soltaninejad K., Hassanian-Moghadam H., Sadeghi A., Rahimzadeh H., Zamani N., *et al.* (2011). Methemoglobinemia in aluminium phosphide poisoning. Hum Exp Toxicol 30(3):250-3.
23. Saheen D. (1996). Technical Vice President Degesch America Inc., Weyers Cave, USA via Oral Communication. p.5-7.
24. Sinha K. (1992). Colonimetric assay of catalase. Anal Biochem 47:389-94.
25. Sinha U.S., Kapoor A.K., Singh A.K., Gupta A., Mehrotra R. (2005). Histopathological changes in cases of aluminium phosphide poisoning. Indian J Pathol Microbiol 48(2):177-80.
26. Sudakin D.L. (2005). Occupational exposure to aluminium phosphide and phosphine gas? A suspected case report and review of the literature. Hum Exp Toxicol 24(1):27-33.
27. Sudakin D.L., Power L.E. (2007). Organophosphate exposures in the United States:A longitudinal analysis of incidents reported to poison centers. J Toxicol Environ Health 70(2):141-7.
28. Tehrani H., Halvaie Z., Shadnia S., Soltaninejad K., Abdollahi M. (2013). Protective effects of N-acetylcysteine on aluminum phosphide-induced oxidative stress in acute human poisoning. Clin Toxicol 51(1):23-8.
29. Turkez H., Togar B. (2012). Aluminum phosphide-induced genetic and oxidative damages in rats:Attenuation by Laurusnobilis leaf extract. Toxicol Ind Health 29(7):579-83.
30. Türkez H., Toğar B. (2013). Aluminium phosphide-induced genetic and oxidative damages *in vitro*:Attenuation by Laurusnobilis L. leaf extract. Indian J Pharmacol 45(1):71-5.
31. United State Department of Health and Human Services (1994). Aluminum Phoshide:Hazarsous Substance Data Base (HSDB). USDHHS, Washington DC. p.10-25.
32. US Environment Protection Agency (1992). Office of Pesticides and Toxic Substances, Fact Sheet Number 118.
33. Valmas N., Ebert P.R. (2006). Comparative toxicity of fumigants and a phosphine synergist using a novel containment chamber for the safe generation of concentrated phosphine gas. PLoS One 1:e130.
34. Waritz R.S., Brown R.M. (1975). Acute, subacute inhalation toxicities of phosphine, phenylphosphine and triphenyl phosphine. Am Ind Hyg Assoc J 36(6):452-8.
35. Wilfrid S., Evan S. (2012). The Therapeutic Uses of Vitamin E. p.20-6.
36. World Health Organization (WHO). (1998). Environmental health criteria 73:Phosphine and selected metal Phosphides.

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