INTRODUCTION
**INTRODUCTION**

Celphos, proprietary preparation of 56 per cent aluminium phosphide (AlP) and ammonium carbonate, when exposed to moisture liberates phosphine (PH₃), which is highly toxic [Child and Coates, 1971; Hockenberg, 1972]. This is used as a fumigant for grain and other stored products against rodents and pests worldwide. Isolated cases of fatal exposure to phosphine gas have been reported in literature when aluminium phosphide was used as grain fumigant for bulk shipment of wheat [Wilson et al., 1990]. It is also used as 'doping' agent to treat the silicon crystals in the semiconductor industry. Phosphine gas seems to be emerging as a potential accidental or intentional health hazard in India because of the increasing use of Celphos for grain storage.

Most of the foreign compounds entering the body are converted to chemically inert metabolites that are readily excreted into urine, air or bile. However, some foreign compounds are metabolized to intermediates that result in the formation of chemically reactive substances which in turn react with various cellular and subcellular constituents.
including proteins, lipids, enzymes, DNA and RNA. The formation of such intermediates frequently results in one or more serious toxic effects, such as cancer (Weishberger and Williams, 1975), cellular necrosis (Gillette et al., 1974; Weishberger and Williams, 1975), hypersensitivity reactions, blood dyscrasias and foetotoxicities (Daly et al., 1974; Davie et al., 1974).

Numerous examples are now known where lipid peroxidation in vivo or in vitro is considered to be a primary event initiated by the reactive metabolites of the toxic chemicals or through the generation of any array of reactive oxygen derived metabolites including superoxide anion (O₂⁻), hydrogen peroxide (H₂O₂) and hydroxyl radical (OH), such as ozone (Goldstein et al., 1969), carbon tetrachloride (Slatter, 1968, 1972; Recknagel, 1967), paraquat (Bus et al., 1975) and acetylphenylhydrazine (Lal et al., 1970). Besides drugs and chemicals, photosensitization (Slater and Riley, 1966) has also been shown to produce tissue damage mediated by free radical induced lipid peroxidation.
Lipid peroxidation often brings about the irreversible damage to the membrane systems, which results in the death of the affected cells or the tissue. However, besides lipid peroxidation other damaging reactions do also occur but lipid peroxidation is without doubt the major feature of overall injury initiated by the reactive toxic intermediates of chemicals and activated oxygen species.

Poisoning of insects by phosphine is manifested by respiratory inhibition. However, yet unexplained reasons, the toxicity to insects is oxygen dependent. In the absence of oxygen, phosphine is virtually non-toxic and is not absorbed to any appreciable extent. Phosphine is a strong inhibitor of respiration in mitochondrion in active state (Stage 3), respiration in state 4 is less sensitive. This inhibition could not be relieved by uncouplers, suggesting that it is due to direct effect on electron transport. Phosphine was unable to activate the 'latent' ATPase nor did it have any inhibition of Mg-stimulated ATPase and only high levels (1.1 um)
showed modest inhibitions of uncoupled stimulated ATPase. Phosphine has no effect on ATP-Pi exchange reaction of concentration causing respiratory inhibition (Chefurka et al., 1976). Phosphine is also known to bring about increase in the leakage of SGOT, SGPT, CPK from certain tissues and thereby raising their levels in plasma (Wilson et al., 1983).

Although no direct evidence exists for the involvement of the oxygen radicals and celphose toxicity. The strong influence of oxygen concentration on celphose metabolism and toxicity (Chefurka et al., 1976) suggests that oxygen free radicals may be formed by the interaction of celphose or phosphine with red cell constituents particularly haemoglobin or secondary radicals with oxygen.

From the foregoing discussion it becomes clear that phosphine itself or through the generation of radicals might be damaging the integrity of the cellular membrane. To our knowledge no reference is made in the literature as to the production of
reactive oxygen species by aluminium phosphide
in addition to phosphine (Freeman and Crean, 1982)
under controlled doses in rats. Because of paucity
of experimental work regarding the celphos poisoning
in reference to membrane lipid peroxidation, the
present study was thus planned to investigate the
importance of membrane damage and lipid peroxidation
in celphos poisoning and in vitro effect of
phosphine.