MAMMALIAN TOXICOLOGY AND TOXICITY TO AQUATIC ORGANISMS OF WHITE PHOSPHORUS AND "PHOSSY WATER" A WATERBORNE MUNITIONS MANUFACTURING WASTE POLLUTANT - A LITERATURE EVALUATION

Dickinson Burrows, et al

Associated Water and Air Resources Engineers, Incorporated

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<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>AAP</td>
<td>Army Ammunition Plant</td>
</tr>
<tr>
<td>ACGIH</td>
<td>American Conference of Government Industrial Hygienists</td>
</tr>
<tr>
<td>ca</td>
<td>circa (about)</td>
</tr>
<tr>
<td>CN</td>
<td>Cyanide</td>
</tr>
<tr>
<td>DOD</td>
<td>Department of Defense</td>
</tr>
<tr>
<td>g</td>
<td>gram</td>
</tr>
<tr>
<td>gpm</td>
<td>gallons per minute</td>
</tr>
<tr>
<td>kg</td>
<td>kilogram</td>
</tr>
<tr>
<td>iv</td>
<td>intravenous</td>
</tr>
<tr>
<td>LC50</td>
<td>Lethal Concentration (kills 50%)</td>
</tr>
<tr>
<td>LD</td>
<td>Lethal Dose</td>
</tr>
<tr>
<td>LD50</td>
<td>Lethal Dose (kils 50%)</td>
</tr>
<tr>
<td>LT</td>
<td>Lethal Time</td>
</tr>
<tr>
<td>LT50</td>
<td>Lethal Time (kils 50%)</td>
</tr>
<tr>
<td>M</td>
<td>Molar</td>
</tr>
<tr>
<td>MAC</td>
<td>Maximum Allowable Concentration</td>
</tr>
<tr>
<td>mg</td>
<td>milligram</td>
</tr>
<tr>
<td>MGD</td>
<td>Million Gallons per Day</td>
</tr>
<tr>
<td>or</td>
<td>oral</td>
</tr>
<tr>
<td>P, P₄</td>
<td>Phosphorus</td>
</tr>
<tr>
<td>³²P</td>
<td>Phosphorus isotope 32</td>
</tr>
<tr>
<td>ppb</td>
<td>parts per billion</td>
</tr>
<tr>
<td>ppm</td>
<td>parts per million</td>
</tr>
<tr>
<td>sc</td>
<td>subcutaneous</td>
</tr>
<tr>
<td>TLV</td>
<td>Threshold Limit Value</td>
</tr>
<tr>
<td>WP</td>
<td>White Phosphorus</td>
</tr>
<tr>
<td>μg</td>
<td>micro-gram</td>
</tr>
</tbody>
</table>
INTRODUCTION

The objective of this work is to provide part of a data base for standards for military-unique product discharges into waters subject to State and Federal regulations.

Background. This report is concerned with the toxicology of elemental "white" phosphorus (also known as "yellow" phosphorus) and its probable aqueous decomposition products. The generation of "phossy water" wastes in loading operations at the Pine Bluff, Arkansas, Army Ammunition Plant and some environmental consequences thereof have been reviewed by Rosenblatt, Small and Barkley (1973)1. White phosphorus (WP) is purchased commercially and unloaded at Pine Bluff Arsenal. It is shipped in rail tank cars and transferred molten, under a warm water blanket, to munitions loading operations.

"Phossy water" is defined as water that has come in direct contact with large quantities of molten phosphorus, including dip-tank overflow, water used to flush white phosphorus from pipes, spray water used for wetting the dip-fill line conveyor and water from periodic cleaning of the dip-fill tanks. The rate of production of this phosphorus-laden water varies greatly, as does the white phosphorus concentration in the water. Elemental phosphorus is continually undergoing reaction with dissolved oxygen (under water), or with atmospheric oxygen to produce oxides of phosphorus that hydrolyze to give phosphorus compounds, perhaps in various oxidation states. Given adequate time, the white phosphorus particulates settle out as a sludge and the dissolved phosphorus is slowly oxidized. However, streams containing white phosphorus remain noticeably polluted over considerable distances.

Acute phosphorus poisoning early in this century was caused by the ingestion of match tips, fireworks, and quack nostrums, and is still encountered with surprising regularity. The literature abounds in cases of human poisonings after the ingestion of rat poisons or roach powders where phosphorus may be present in concentrations up to 4%. The conquest of occupational phosphorus poisoning in lucifer match manufacturing has been described by Oliver (1938)2 and by Hamilton and Hardy (1949)3.

Chronic intoxication by inhalation of phosphorus fumes occasionally occurs in industry. A chronic phosphorus poisoning literature review for the period up to 1945 (and a presentation of recent case reports) summarizes the chronic effects of elemental phosphorus on humans (Heimann, 1945)4.

Almost all knowledge concerning the toxicity of white phosphorus to aquatic species derives from studies initiated in the late 1960's in response to a massive fish kill caused by the wastewater discharged from a factory manufacturing elemental phosphorus in Newfoundland (Idler, 19695; Malin, 19726). Most of the data concern salt water organisms. A similar
catastrophic die-off of freshwater fish in Pickwick Reservoir in the early 1950's is believed to have resulted from an accidental discharge of phossy water from holding ponds at the Muscle Shoals, Alabama, phosphorus manufacturing facility operated by the Tennessee Valley Authority[7,8,9]. However, phosphorus poisoning was never demonstrated conclusively, and very little research resulted from this episode. Because phosphine, phosphite, hypophosphite and hypophosphate are likely products of the decomposition of white phosphorus in water, the relevant chemistry and toxicology of these compounds are covered in the present report.

Approach to Mammalian Toxicology. A survey was conducted of the chemical and biological literature on white phosphorus and on the related compounds. Sources for the literature search are set out in detail in Appendix A. Most of the references were obtained from Chemical Abstracts. Several machine searches were also made, but were not very helpful. In addition to the references obtained from the sources listed, a paper by Fleming, Miller, and Swayne (1942)[10] on "Some recent observations on phosphorus toxicology" noted that their references had been selected from some 1000 reports on phosphorus toxicology as having special bearing on their reported findings; this bibliography (actually only 444 references) was obtained from the technical library of the Tennessee Valley Authority.

The papers obtained were grouped in the following categories for toxicological evaluation.

1. Acute toxicity studies
2. Chronic toxicity studies
3. Clinical studies (as they relate)
4. Biochemical studies
5. Analytical and related chemical studies.

Further references on the chemistry and mammalian toxicology of white phosphorus, of only peripheral interest, are listed in Appendix B.

Approach to Toxicity to Aquatic Organisms. The principal source of information on the aquatic toxicology of white phosphorus is contained in a collection of reprints from Dr. David Idler of the Memorial University of Newfoundland[7]. Additional information and literature sources were forthcoming in telephone consultations with personnel from Memorial
University11,12, ERCO Industries, Ltd.13,14, Monsanto Company15-18, TVA7-9, Fisheries Research Board of Canada20-22, American Chemical Society23, and Ash Stevens, Inc.24.

The published literature was independently surveyed using Chemical Abstracts. Appropriate subheadings under phosphorus were scanned from 1937 to August 1973. Phosphine, phosphite, hypophosphite, hypophosphate, phosphorous acid, hypophosphorous acid, and hypophosphoric acid were reviewed from 1917 to August 1973.

A machine search for relevant DOD documents was carried out using key words phosphorus, white phosphorus, phossy water, plasticized white phosphorus25. Two documents relating to spray treatment of Pine Bluff phossy water, revealed as a result of this search, had not been received at the time this report was compiled. See also Appendix A.

Current Research on WP Toxicology and "Phossy Water". Apart from a few recent studies on the biochemical effects of white phosphorus, there does not appear to be any current research activity on the toxicity and toxicology of WP and phossy water. Laboratories listed in references 11, 12, 20, 21, and 22 are believed to be still active in research on the effects of elemental phosphorus pollution.

MAMMALIAN TOXICOLOGY OF WHITE PHOSPHORUS AND RELATED MATERIALS

Acute Toxicity in Animals. A summary of acute lethal toxicity of phosphorus in the rabbit, dog, rat, and mouse is given in Table 1. No LD50 values have been recorded for phosphorus. The effects of single toxic doses of phosphorus in olive oil given subcutaneously to male rabbits have been studied by Huruya (1928)26. Changes associated with the production of fatty livers by white phosphorus in rats have been investigated by Seakins and Robinson (1964)27 and by Pani, Gravela, Mazzarino and Burdino (1972)28. Love (1934)29 studied the effect of phosphorus on the normal and on the restored liver following partial hepatectomy in the albino rat.

Acute Toxicity in Man. In acute phosphorus poisoning, the predominant effect is fatty degeneration of the tissues. This occurs essentially in the liver, the damage to which is the cause of death in most cases. As little as 1/8 grain (8.1 mg) has been reported as having caused acute toxicity and death but the average fatal dose taken by mouth is generally considered to be of the order of 1.5 grains (97 mg). Christensen (1972)37 gives the oral LD as 1.4 mg/kg. Acute phosphorus poisoning is peculiar because symptoms appear in two stages. During the first 24 hrs, symptoms
TABLE 1. Summary of Acute Toxicity of White Phosphorus in Animals.

<table>
<thead>
<tr>
<th>Animal Species</th>
<th>Route of administration</th>
<th>Dose* (mg/kg)</th>
<th>Remarks</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rabbit</td>
<td>or 7</td>
<td>LD</td>
<td>LD</td>
<td>Hirz (1913)30</td>
</tr>
<tr>
<td></td>
<td>or 10</td>
<td>LD</td>
<td>LD</td>
<td>Frank and Isaac (1910-1911)31</td>
</tr>
<tr>
<td></td>
<td>sc 12.5</td>
<td>LD</td>
<td>LD</td>
<td>Santesson and Malmgren (1904)32</td>
</tr>
<tr>
<td></td>
<td>sc 30</td>
<td>LD</td>
<td>LD</td>
<td>Santesson and Malmgren (1904)32</td>
</tr>
<tr>
<td></td>
<td>or 20.0</td>
<td>LD</td>
<td>LD</td>
<td>Cameron and Patrick (1966)33</td>
</tr>
<tr>
<td>Dog</td>
<td>sc 2-3</td>
<td>LD</td>
<td>LD</td>
<td>Rubow (1905)34</td>
</tr>
<tr>
<td></td>
<td>sc 12</td>
<td>LD</td>
<td>LD</td>
<td>Welsch (1905)35</td>
</tr>
<tr>
<td>Rat</td>
<td>or &lt;0.1 grain†</td>
<td>LD</td>
<td>LD</td>
<td>Shepard (1951)36</td>
</tr>
<tr>
<td></td>
<td>or 0.5</td>
<td>LD</td>
<td>LD</td>
<td>Cameron and Patrick (1966)33</td>
</tr>
<tr>
<td>Mouse</td>
<td>or 3.5</td>
<td>LD</td>
<td>LD</td>
<td>Cameron and Patrick (1966)33</td>
</tr>
</tbody>
</table>

*All administered in oil.
†For a 400 g rat.
of severe gastrointestinal irritation occur as soon as one-half hour after ingestion. The victim may die of cardiovascular failure within 12 hr. This first stage may be followed by a latent period lasting from a few hrs to a few days depending upon the amount ingested. The systemic stage is characterized by abdominal pain, nausea, vomiting, hematemesis and other hemorrhagic manifestations, jaundice, hepatomegaly, oliguria, toxic psychosis, convulsions, coma, and shock. There may be severe damage to the liver, heart, and kidney, and death may ensue at any time. Cirrhosis of the liver has been reported after recovery from the acute state. The signs and symptoms of acute poisoning in man are also adequately summarized by Gleeson, Gosselin, Hodge and Smith (1969).48

Fourteen papers on acute human phosphorus poisoning (episodes and case studies) are listed in the references (nos. 39-52) at the end of this report.

Chronic Toxicity in Animals. Sollmann (1925) was the first investigator to study the effects of chronic feeding of phosphorus to albino rats. Further similar experiments by Flemming et al. (1942) confirmed the retardation in growth and the effects on bone development.

No significant changes were found in the blood, urine and tissues of dogs given subcutaneous injections of phosphorus in peanut oil (0.4, 0.2, and 0.1 mg P/kg/day) according to Buchanan, Sigal, Robinson, Pope, Ferguson and Thomison (1954). The fate of orally administered choline in dogs with livers damaged through phosphorus poisoning was studied by Sigal, Buchanan and Robinson (1954).

Ferraro, Jervis and English (1938) drew attention to the fact that studies on white phosphorus intoxication in rabbits have been focused generally on liver changes and the metabolism of fatty substances. These workers have described the pathological changes in the brains of adult rabbits (nerve cells, blood vessels and glia) given iv injections of a 1% solution of phosphorus in oil (0.20 to 1.0 ml doses), over periods of 2 days to 15 weeks.

Lawrence and Huffman (1929) noted that sc injections of phosphorus in olive oil into guinea pigs caused a marked increase in the number of the circulating monocytes in the blood.

Chronic Toxicity in Man (Including Potential for Carcinogenicity, Mutagenicity, and Teratogenicity). Chronic poisoning, seen mostly in factory workers exposed repeatedly to phosphorus vapors, is manifest
by cachexia, anemia, bronchitis, and necrosis of the mandible ("phossy jaw"). The signs and symptoms of chronic poisoning, whether from ingestion or inhalation, have been well documented in the publications of Heimann (1946)4 and Gleeson, Gosselin, Hodge, and Smith (1969)38. For summary of chronic toxicity to humans see Table 2.

The TLV for white phosphorus (ACGIH, 1972)58 is 0.1 mg/m³. The TLV for phosphine58 is 0.3 ppm or 0.4 mg/m³.

Testing for the possible carcinogenic activity of elemental phosphorus in experimental animals has been summarized by Hartwell (1951)59, and Shubik and Hartwell (195760, 196961). No animals with tumors were found following either oral feeding or subcutaneous injections in oil, even in rats receiving the compound for 33 months.

White phosphorus does not appear to have been investigated for possible mutagenic or teratogenic potential in living systems.

A case of opthalmophagia due to phosphorus poisoning has been described by Medea (1905)62. The effect of white phosphorus on growing bones and growing teeth of both rabbits and rats has been studied by Adams and Sarrat (1940)63.

Burns due to Phosphorus. Two recent papers describe observations on the burns resulting from elemental white phosphorus. Curreri, Morris, and Pruitt (1970)64 reviewed the clinical records of 111 patients, mostly with burns due to white phosphorus, who were admitted to the US Army Institute of Surgical Research. They noted the high incidence of residual ocular damage and suggested a number of specialized requirements for successful systemic and local wound care. No biochemical or toxicological data were evaluated.

Bowen, Whelan, and Nelson (1971)65 describe a methodology for the production of a standard white phosphorus burn in rabbits. Although their study offered no suggestions regarding new or improved therapy of the white phosphorus-burned patient, it did elucidate electrolyte aberrations (depression of serum calcium and elevation of serum phosphorus and sometimes a reversal of the calcium/phosphorus ratio), and electrocardiographic changes which may explain early, sudden demise in the seemingly inconsequentially white phosphorus-burned patient.

Toxicity of Phosphine. Because of the possibility that phosphine (PH₃) may be a product of phosphorus metabolism (see p. 15) the toxicology has
MAMMALIAN TOXICOLOGY AND TOXICITY TO AQUATIC ORGANISMS OF WHITE PHOSPHorus AND "PHOSSY WATER", A WATERBORNE MUNITIONS MANUFACTURING WASTE POLLUTANT - A LITERATURE EVALUATION

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Elemental white phosphorus is highly toxic to both experimental animals and man. Ingestion of even small amounts may produce severe gastrointestinal irritation, bloody diarrhea, liver damage, skin eruptions, oliguria, circulatory collapse, coma, convulsions and death. The fatal dose for man is about 1-1.4 mcg/kg. No LD50 values have been determined. Acute effect differ considerably from chronic effects. Chronic poisoning (from ingestion or inhalation) is characterized by such effects on the osseous system as bony necrosis ("phossy jaw"), spontaneous fractures, as well as by anemia and weight loss. White
phosphorus appears to be noncarcinogenic when fed to experimental animals. Testing for mutagenicity and teratogenicity has not been carried out. Many studies of a biochemical nature have been reported and have been summarized in the report. The primary effect of phosphine is neurological, and in this it differs markedly from white phosphorus.

White phosphorus is also highly toxic to aquatic animals. The 96 hr LC50's are less than 50 ppb for all fish studied, and the incipient lethal level is probably less than 1 ppb for most fish. Crustaceans and many molluscs are more tolerant, but still succumb to phosphorus concentrations of 1 ppm or less. Phosphorus poisoning appears to be cumulative and irreversible for fish and lobsters, though the cause of mortality in fish has not been determined. There is no information on the toxicity of phosphine to aquatic biota or experimental animals.

Recommendations for studies on white phosphorus (and phosy water) in experimental animals and in wastewaters are outlined.
TABLE 2. Summary of Chronic Toxicity of White Phosphorus to Humans (Heimann, 1946).

<table>
<thead>
<tr>
<th>System Affected</th>
<th>Characteristic Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>Low in potassium and inorganic P content, high in chlorides, fat content. Morphological changes in circulating blood cells. Anemia of so-called &quot;secondary&quot; type.</td>
</tr>
<tr>
<td>Urinary</td>
<td>General cellular damage. Increase in ammonia N₂ and unoxidized sulfur content.</td>
</tr>
<tr>
<td>Liver</td>
<td>No definite liver diseases described.</td>
</tr>
<tr>
<td>Local Irritant</td>
<td>Skin burns, conjunctivitis, skin rashes.</td>
</tr>
<tr>
<td>Effects</td>
<td></td>
</tr>
<tr>
<td>General Cachexia</td>
<td>Exhaustion; loss of appetite; indefinite digestive complaints; garlic-like breath; slight jaundice, bleeding of the mucous membranes and albumin in the urine.</td>
</tr>
</tbody>
</table>
been summarized herewith (see also Webster, 194666). The toxicology of acute mammalian poisoning by the gas phosphine has not been fully worked out according to Patty, Fassett, and Irish (1963)67. It does not alter the blood and its effects are apparently exerted largely through the central nervous system. Some inflammation of the lungs results from its inhalation. The acute action of phosphine does not resemble that of phosphorus; instead, there is marked dyspnea, purgation, weakness, tremor, and finally violent convulsions and death. Mild cases recover without after-effects. Prolonged exposure to small amounts of phosphine is said to give rise to chronic symptoms similar to those of phosphorus poisoning and may be explained by the decomposition of the compound in the body (Meissner, 192468; Müller, 194069; Loewenthal, 194970). A summary of the physiological responses to various concentrations of phosphine is given in Table 3.

**BIOCHEMICAL STUDIES**

Poisoning due to phosphorus, according to Cutler (1931)72, results in an increase of guanidine in the blood of dogs and accompanying hypoglycemia. The results also indicate that the guanidine increase is probably dependent upon liver damage rather than upon kidney failure.

Mizuno (1932)73 reported a relatively large increase (3 to 8 times) in the lactic acid content of the urine of rabbits with liver damage due to phosphorus poisoning.

The glucuronic acid produced by surviving liver slices from guinea pigs given a sc injection of yellow phosphorus (0.75 mg/kg body wt) in olive oil was found by Bueding and Ladewig (1939)74 to be considerably reduced. Chloroform poisoning did not affect glucuronic acid production.

Barone, Cittadini, Galeatti, and Terranova (1973)75 reported that fatty liver degeneration in rats caused by white phosphorus had no influence on the level of the microsomal respiratory pigments cytochrome bs and cytochrome P450.

Rabbits with fatty livers caused by white phosphorus poisoning had three times the amounts of cytochrome c over fasted control animals according to Prader (1947)76. Milder poisoning resulted in less cytochrome c and a lag period was also evident.

Glosha5, Porta, and Hartroft (1972)77 measured the liver microsomal glucose-6-phosphatase in the fatty livers of rats given oral doses of
TABLE 3. Summary of the Physiological Responses to Various Concentrations of Phosphine (Henderson and Haggard, 1943).71

<table>
<thead>
<tr>
<th>Response</th>
<th>Phosphene Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAC for prolonged exposure</td>
<td>2ppm 2.6μg/m³</td>
</tr>
<tr>
<td>Slight symptoms after several hrs. exposure</td>
<td>7ppm 10,010μg/m³</td>
</tr>
<tr>
<td>Max. concentration that can be inhaled for 1 hr. without serious consequences</td>
<td>100 - 200ppm 143,000 - 286,000μg/m³</td>
</tr>
<tr>
<td>Dangerous after-exposure for 1/2 - 1 hr.</td>
<td>400 - 600ppm 572,000 - 878,000μg/m³</td>
</tr>
<tr>
<td>Fatal after-exposure for 30 mins.</td>
<td>1,000 - 2,000ppm 1,430,000 - 2,860,000μg/m³</td>
</tr>
</tbody>
</table>
phosphorus (7.5 mg/kg body wt) and found no significant depression at 6 or 24 hours.

Serological studies on the blood of rabbits given yellow phosphorus (0.1, 1.0, 2.0 mg/kg body wt) were reported by Tanaka (1960) with the following results: All groups showed an early increase in non-protein nitrogen, amino acid nitrogen and uric acid, and a slight increase in creatine and creatinine. A decrease in erythrocyte count, hemoglobin and red cell fragility was also recorded.

The changes in the serum proteins after blood and plasma losses have been studied in normal and phosphorus poisoned rabbits by Wenner (1949). He found an increase of the α- and β-globulins after bleeding but no increase of the glucosamine content.

Fleming and Collings (1951) studied the chylomicron count in the blood of rats given sc injections of white phosphorus (1.1 mg/kg body wt) in peanut oil. No significant changes occurred in fasting rats but significant and reproducible changes were noted 4 hours after resuming food intake.

Recent papers by Truhaut et al. (1969, 1971, 1972) describe the effects of both single sc doses and repeated sc doses of white phosphorus in the rat. The effects on the levels of liver phospholipids and triglycerides were reported.

TOXICITIES OF WHITE PHOSPHORUS TO AQUATIC ORGANISMS

Toxicity of Elemental Phosphorus in Water. In the earliest aquatic study of which we are aware, the tolerance of bluegills (Lepomis macrochirus Rafinesque) for white phosphorus was measured in freshwater at 26°C (Isom, 1960). The author concluded that phosphorus was nontoxic to the limit of its solubility (ca. 3 mg/l at 15°C), but paradoxically, that much smaller quantities of colloidal phosphorus were highly toxic. The conclusion concerning colloid-free water is probably invalid, because the author did not have available an analytical procedure sufficiently precise to tell whether elemental phosphorus was present at all; however, the 96 hour median tolerance level of 45 μg/l, colloidal or dissolved, may reasonably be taken as an upper limit (cf. Table 1). Curiously, a Russian report claims that the toxicity of different forms of elemental phosphorus to freshwater fishes decreases in the order emulsion > solution > suspension > colloid (Kresnov, 1970).
The most complete studies on white phosphorus intoxication in a marine environment have been carried out in connection with operation of a plant producing elemental phosphorus in Newfoundland. This plant, constructed by ERCO Industries, Ltd. on Long Harbour, Placentia Bay, began discharging phosy water in December, 1968. Shortly thereafter large numbers of fish, principally cod and herring, were found dead in Long Harbour and adjacent waters, and in May, 1969, the plant was shut down and part of Placentia Bay was closed to fishing. Subsequently, groups associated with the Fisheries Research Board of Canada have studied the toxicity of white phosphorus in seawater to various fish and crustaceans (Zitko et al., 197086; Fletcher et al., 197087; Fletcher and Hoyle, 197286). Their results are summarized in Table 4. For five genera and four families of fish in saltwater, 96 hour LD 50's are remarkably uniform, 3 ± .7 ug/l, although field experience indicates that cod are substantially more tolerant than herring (Fletcher, 197312. Incipient lethal levels are very low, suggesting that phosphorus poisoning is accumulative in these fish. Two separate studies emphasize the importance of the time-dosage relationship (Zitko et al., 197086; Fletcher and Hoyle88); a short exposure to a relatively high concentration may be lethal in the same time period as continuous exposure to a low concentration. For crustaceans the tolerance to phosphorus may be several orders of magnitude higher, though the incipient lethal level for lobsters is still quite low in terms of likely pollution levels. The beach flea has the greatest tolerance for white phosphorus of any animal studied, and is the only one for which reversibility of intoxication has been clearly demonstrated.

Similar investigations of other phyla have not been reported. However, a study of the bottom of Long Harbour in the vicinity of the effluent outlet provided evidence of selective mortality among benthic organisms (Peer, 197291. Thus, e.g., mussels (Modiolus modiolus) and a burrowing sea anemone (Edwardsia sp.) were alive and apparently normal in a region from which scallops and sand dollars (Echinarchium parina) had been nearly exterminated. In a recent study, Black (1973)22 has found no significant variation in annual growth rates for Long Harbour mussels (Mytilus edulis) during 1969, the year of significant phosphorus pollution, and preceding and succeeding years (Black, 1973)92. On the other hand, no periwinkles (Littorina littorea) old enough to have been set before 1970 were found, indicating that all were killed by the 1969 spill.

Accumulation of Phosphorus by Aquatic Organisms. In a 16 hour exposure, cod have been shown to concentrate phosphorus 1000 times in the liver and 10 to 1000 times in muscle tissue from water containing 20 to 80 ug/l (Dyer et al., 1970)93. Concentration factors are roughly proportional to the lipid content of the tissue, and inversely related to the phosphorus content of the water. Other fish have not been studied in such
TABLE 4. Summary of Toxicity of White Phosphorus to Fish Species and Crustaceans.

<table>
<thead>
<tr>
<th>Organism</th>
<th>Incipient Lethal Level mg/l</th>
<th>96 hr Test</th>
<th>Water</th>
<th>Temp. °C</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bluegill (Lepomis macrochirus)</td>
<td>ca. 45 static fresh 26</td>
<td></td>
<td></td>
<td></td>
<td>Isom (1960)83</td>
</tr>
<tr>
<td>Cod (Gadus morhua)</td>
<td>&lt;1.9</td>
<td>2.5 cont.</td>
<td>salt</td>
<td>9-10</td>
<td>Fletcher and Hoyle (1972)88</td>
</tr>
<tr>
<td>Atlantic salmon (Salmo salar)</td>
<td>&lt;.8</td>
<td>2.3 cont.</td>
<td>salt</td>
<td>10-13</td>
<td>Zitko et al. (1970)86</td>
</tr>
<tr>
<td>Herring (Clupea harengus)</td>
<td>&lt;2.5</td>
<td>3.7 static</td>
<td>salt</td>
<td>7-9</td>
<td>Fletcher and Hoyle (1972)88</td>
</tr>
<tr>
<td>Brook trout (Salvelinus fontinalis)</td>
<td>&lt;.5</td>
<td>2.5 cont.</td>
<td>salt</td>
<td>7-9</td>
<td>Fletcher et al. (1970, 1971)87,89</td>
</tr>
<tr>
<td>Smelt (Osmerus mordax)</td>
<td>2.5</td>
<td>3 cont.</td>
<td>salt</td>
<td>7-9</td>
<td>Fletcher et al. (1970, 1971)87,89</td>
</tr>
<tr>
<td>Killifish (Fundulus heteroclitus)</td>
<td>&gt;20 cont.</td>
<td></td>
<td>salt</td>
<td>7-9</td>
<td>Fletcher et al. (1971)89</td>
</tr>
<tr>
<td>Cunner (Tautogolabrus adspersus)</td>
<td>* cont.</td>
<td></td>
<td>salt</td>
<td>7-9</td>
<td>Fletcher et al. (1971)89</td>
</tr>
<tr>
<td>Lobster (Homarus americanus)</td>
<td>20-40</td>
<td>300 static</td>
<td>salt</td>
<td>?</td>
<td>Zitko et al. (1970)86</td>
</tr>
<tr>
<td>Beach flea (Gammarus oceanicus)</td>
<td>3000-4000</td>
<td>static</td>
<td>salt</td>
<td>18-21</td>
<td>Zitko et al. (1970)86</td>
</tr>
<tr>
<td>Atlantic salmon smolts</td>
<td>ca. 10 cont. fresh 11-14</td>
<td></td>
<td></td>
<td></td>
<td>Fletcher and Hoyle (1972)88</td>
</tr>
</tbody>
</table>

8Al 50, 3-4 µg/l, 200-300 hr.
detail, but from a survey of fish collected in Placentia Bay in 1969-1970, and from limited bioassays, it is known that herring and salmon accumulate phosphorus to the 1 ppm level, and that other fish can accumulate at least measurable quantities (Ackman et al., 1970).

Fletcher (1971) has recorded the 48 hour uptake of white phosphorus by several marine invertebrates and seaweed from seawater containing 15 ± 9 μg/l of white phosphorus (cf. Table 5). It may be noted that periwinkles, which are foraging grazers, accumulated greater amounts of phosphorus than the filter feeding molluscs. Fletcher (1971) suggests that the former may ingest phosphorus with the seaweed. Intoxication was not reported for any of these invertebrates, and elemental phosphorus was eliminated from all the organisms of Table 6 within seven days following transfer to uncontaminated seawater.

Toxicity to Waterfowl. Dabbling ducks of the genus Anas feeding in areas of phossy water discharge have been found to ingest lethal quantities of elemental phosphorus (Coburn et al., 1950). In a series of experiments conducted with captive black ducks and mallards, chronic and acute responses varied widely among individuals, but a single dose of 3 mg/kg body weight was sufficient to kill all ducks studied in 6 to 33 hours.

Toxicity of the Total Waste. Analysis of a phossy water sample from Pine Bluff AAR showed it to contain 242 mg/l of phosphate and 208 mg/l of lower phosphorus oxides, as phosphorus (Blumbergs et al., 1973). These might include hypophosphate, phosphite, and hypophosphite, the latter two being known products of nonaqueous oxidation of phosphorus (Van Wazer, 1958). No one of these is reported to be very toxic.

\[
\begin{bmatrix}
  0 & 0 \\
  0-P-P-0 \\
  0 & 0
\end{bmatrix}^{4-} \quad \begin{bmatrix}
  0 \\
  0-P-H \\
  0
\end{bmatrix}^{2-} \quad \begin{bmatrix}
  0 \\
  H-P-H \\
  0
\end{bmatrix}^{2-}
\]

Nitrogen fixation by Azotobacter vinelandii is completely inhibited by 300 mg/l of phosphite (Bulen and Frear, 1957) as P, ca. 10−2M, while some other bacteria (e.g. Escheria coli and certain Pseudomonas, Agotobacter, Rhiz obium and Agrobacterium species) can utilize phosphite for growth at the same concentration (Casida, 1960), and Pseudomonas fluorescens.
<table>
<thead>
<tr>
<th>Organism</th>
<th>White Phosphorus (mg/g)</th>
<th>Conc. Factor Approx.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seaweed</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Fucus vesiculosus</em></td>
<td>.33</td>
<td>22</td>
</tr>
<tr>
<td><em>Fucus distichus</em></td>
<td>.33</td>
<td>22</td>
</tr>
<tr>
<td>Invertebrates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clam (Mya arenaria)</td>
<td>.34</td>
<td>23</td>
</tr>
<tr>
<td>Quahog (Arctica islandica)</td>
<td>.26</td>
<td>17</td>
</tr>
<tr>
<td>Mussel (Mytilus edulis)</td>
<td>.16</td>
<td>11</td>
</tr>
<tr>
<td>Periwinkle (Littorina littorea)</td>
<td>.64</td>
<td>43</td>
</tr>
<tr>
<td>Starfish (Asterias vulgaris)</td>
<td>.40</td>
<td>27</td>
</tr>
</tbody>
</table>
converts phosphite to phosphate in excess of that required for growth. Still other micro-organisms, including some yeasts, are unable to utilize phosphite, but are unaffected by it. Robertson and Boyer (1956)\textsuperscript{100} report that rat weanlings fed inorganic phosphite at 250 mg/kg on alternate days show some restriction of growth, but 125 mg/kg is without effect. Table 6 summarizes enzyme inhibition data for phosphite and hypophosphate at the $10^{-2}$M level. The effect is generally small. Van Wazer (1958)\textsuperscript{101} says that "the phosphites exhibit considerable toxicity, acting on the nerve centers and abdominal glands; he also says "there is no evidence that hypophosphate exerts the slightest effect on mammals, and it passes through the mammalian system unchanged"\textsuperscript{102}, but he presents no data more recent than 1902. There appear to be no toxicity data of any kind for hypophosphate. At higher pH levels, phosphine is formed from elemental phosphorus, and a small amount may be present in phossy water. Phosphine is a toxic gas (MPC 0.3 ppm for humans) with a moderately low water solubility. It is alleged to be toxic to rainbow trout in concentrations exceeding 3.6 ppm (Daudoroff and Katz, 1950)\textsuperscript{106}. A few papers report the generation of phosphine in natural waters by anaerobic sediments (Datsko, 1956\textsuperscript{107}; Luning and Brohm, 1932\textsuperscript{108}, 1934\textsuperscript{109}), and one implies that it is highly toxic to fish\textsuperscript{107}, but otherwise there does not appear to be a literature of aquatic toxicology. It is rapidly degraded by the microorganisms in wet soil (Hilton and Robison, 1972)\textsuperscript{110}.

Polyphosphoric acid, if formed in phossy water, would not be expected to present a problem. Although it is a sequestering agent, like NTA, and might enhance the toxicities of any heavy metals in the water, it is readily biodegradable to orthophosphoric acid (Metcalf, 1973\textsuperscript{113}, Karl-kroupa et al., 1957)\textsuperscript{111}.

The phossy water discharged in the manufacture of phosphorus is a more complex waste than that released in filling operations, such as that at Pine Bluff AAP\textsuperscript{5},15-19. The latter consists only of dissolved and suspended elemental phosphorus and oxidation and hydrolysis products thereof (principally phosphoric acid). The manufacturing wastewater includes as well a large amount of suspended solids, fluosilicic acid and sulfur dioxide, and some ammonia and cyanide. A typical wastewater analysis for the ERCO Industries, Ltd. plant, prior to shutdown in 1969, is given in Table 7. Thus although extensive mortality studies were carried out in the area, it was necessary that early investigators consider that the catastrophic effect of the Long Harbour discharge may have been due in part to waste components other than phosphorus. Bioassays of the various waste components, however, have shown phosphorus to be the most toxic by several orders of magnitude (Fletcher et al., 1971)\textsuperscript{89}. For example, Figure 1 illustrates the comparative toxicities of several substances to brook trout maintained in seawater. The total wastewater
<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Anion</th>
<th>Conc</th>
<th>% Inhibition</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intestinal phosphatase</td>
<td>orthophosphate</td>
<td>$10^{-2}M$</td>
<td>5</td>
<td>Robertson and Boyer (1956)</td>
</tr>
<tr>
<td>3-Phosphoglyceraldehyde</td>
<td>orthophosphate</td>
<td>$2\times10^{-2}M$</td>
<td>salt effect only</td>
<td>Robertson and Boyer (1956)</td>
</tr>
<tr>
<td>dehydrogenase</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>E. coli</em> formic dehydrogenase</td>
<td>hypophosphate</td>
<td>$10^{-2}M$</td>
<td>~20</td>
<td>Crewther (1956)</td>
</tr>
<tr>
<td><em>E. coli</em> formic hydrogenlyase</td>
<td>hypophosphate</td>
<td>$10^{-2}M$</td>
<td>~40</td>
<td>Crewther (1956)</td>
</tr>
<tr>
<td><em>E. coli</em> hydrogenase</td>
<td>hypophosphate</td>
<td>?</td>
<td>0</td>
<td>Crewther (1956)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Gruenberg-Monago et al. (1951)</td>
</tr>
<tr>
<td>Horse serum cholinesterase</td>
<td>hypophosphate</td>
<td>$10^{-2}$</td>
<td>~35</td>
<td>Frommel et al. (1944)</td>
</tr>
<tr>
<td></td>
<td>Value</td>
<td>Concentration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------</td>
<td>------------------------</td>
<td>---------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flow</td>
<td>ca. 8000 gpm</td>
<td>11.5 MGD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fluoride</td>
<td>22800 lb/day</td>
<td>245 mg/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SO₂</td>
<td>6250 lb/day</td>
<td>67 mg/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total P</td>
<td>1630 lb/day</td>
<td>18 mg/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P₄</td>
<td>1250 lb/day</td>
<td>13 mg/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CN</td>
<td>96 lb/day</td>
<td>1 mg/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NH₃</td>
<td>455 lb/day</td>
<td>5 mg/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Solids</td>
<td>38360 lb/day</td>
<td>410 mg/l</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
FIGURE 1: TOXICITIES OF VARIOUS SUBSTANCES TO SEAWATER MAINTAINED BROOK TROUT (Fletcher, et al., 1971)
exhibits no enhanced toxicity to cod beyond that to be predicted on the basis of its white phosphorus content alone (Idler, 1965).5

Other constituents of the waste may affect the distribution and dispersal of phosphorus (Jangaard, 1970).112. Fluosillicic acid reacts with calcium and magnesium in seawater to produce a flocculant precipitate, which can coagulate and thereby immobilize colloidal white phosphorus in the immediate vicinity of the outfall. This greatly reduces the efficacy of tidal dilution, and may result in the creation of permanent toxic zones. In the case of the Long Harbour operation, much of the phosphorus contamination resulted from accidental spills in the course of loading ships with liquid phosphorus.13. Phosy water from this source would not be expected to differ greatly from that from ammunition plants.

Behavioral Effects. The most obvious symptom of phosy water poisoning, other than death of the victim, is the red color acquired by the belly and head of certain fish, particularly herring. All fish eventually become lethargic, and on the approach of death may swim in circles (Zitko et al., 1970).86. Lobsters exposed to lethal levels of white phosphorus "lose muscle tone, become lethargic, move the pereiopods in a jerky, uncoordinated and aimless manner, and eventually lose all neuromuscular response."86. Beach fleas become paralyzed when subjected to lethal doses, but may recover when transferred to clean water.86.

Among waterfowl, acute poisoning results in depression, followed by leg weakness and finally violent convulsions and death. Birds suffering from chronic poisoning steadily lose weight and eventually show signs of paralysis. Field cases, usually suffering from acute poisoning, are described as having "ungainly flight" (Coburn et al., 1950).95.

Pathology. White phosphorus can be expected to cause extensive tissue damage wherever it accumulates. Tissues of the gills, kidneys, liver and spleen may undergo substantial disintegration (Odense et al., 1972).113. However, the symptoms of phosphorus intoxication, including death, are not necessarily related to tissue destruction.

The cause of fish mortality from phosy water has yet to be determined. White phosphorus levels low enough to permit survival of the fish for more than a few hours may cause extensive hemolysis of erythrocytes for some species, viz. herring (Zitko et al., 1970; Fletcher et al., 1970), Atlantic salmon (Zitko et al., 1970; Fletcher and Hoyle, 1972), and brook trout (Fletcher et al., 1970). Some of the hemoglobin released may then leave the circulatory system and pass into the interstitial areas, temporarily imparting a bright red color to the head and belly. Hematocrits
may drop to a very low value, and in later stages the gills may turn pale, but loss of red cells is apparently not the cause of death. Cod and smelt exhibit no evidence of hemolysis at any level of phosphorus exposure, and trout and salmon expire without hemolysis if the dosage is great enough (Fletcher et al., 1970a, 1972b). Recent Russian studies suggest that the central nervous system may be involved (Krasnov, 1970b; Mazmaridi, 1970c). Fletcher (1973) has found that brook trout fed cod poisoned by elemental phosphorus develop the same symptoms as trout exposed to phosphorus in water. Whatever the cause, phosphorus intoxication of fish appears not to be reversible.

In lobsters, phosphorus intoxication brings about degeneration of the hepatopancreas and antennal gland tissue (Aiken and Byard, 1972). Although this damage should eventually kill the lobster, death is probably due to asphyxiation resulting from blood coagulation. Toxic effects are cumulative and irreversible or very slowly reversible.

For waterfowl, the histopathology of phosphorus intoxication is inconsistent. The kidneys are usually congested, the liver commonly shows signs of damage, and duodenal ulcers may be present, but it is not certain that any of these disorders are related to the early death of the bird (Coburn et al., 1950).

Summary and Conclusions. White phosphorus is highly toxic to fish. The 96 hour LC 50's are less than 50 ppb for all fish studied, and the incipient lethal level is probably less than 1 ppb for most fish. Phosphorus poisoning appears to be accumulative and irreversible, though the cause of mortality has not been determined. While phosphorus is readily taken up by fish and other aquatic organisms directly from the water, fish may also acquire lethal quantities of elemental phosphorus through the food chain, since the few macroinvertebrates studied have a much higher tolerance for white phosphorus than fish. The symptoms of phosphorus intoxication are passed onto brook trout when they are fed muscle tissue from phosphorus-poisoned cod (Fletcher, 1973). Furthermore, elemental phosphorus can be passed on to humans, since a considerable portion, 25% or more, remains in the muscle of the fish after processing, storage, and cooking (Dyer et al., 1972).

So toxic is white phosphorus to some fish that it is difficult to set maximum safe levels, although there are indications that fish acquire a tolerance after exposure (Fletcher, 1973). If it is assumed that the minimum incipient lethal level is 0.1 µg/l (1/5 the lowest measured), then it would seem advisable to set a maximum of 0.01 µg/l in the mixing zone of any outfall. Zero discharge of phosphorus manufacturing process water has been recommended (Anon., 1973).
Other components of phossy water are probably inconsequential in terms of their likely concentrations, but there is uncertainty concerning phosphine.

CHEMISTRY OF WHITE PHOSPHORUS AND DERIVED SUBSTANCES

The Fate of Elemental Phosphorus in Water. Although there are no definitive published reports on the kinetics of oxidation of elemental phosphorus in water, it appears that the rate is highly dependent on the degree of dispersion. At concentrations (ca. 10 \( \mu \text{g/l} \)) well below the accepted solubility limit of 3 mg/l, with the dissolved oxygen content unspecified, elemental phosphorus disappears by a first order process with a half-life of 2 hours at ca. 10°C (Zitko et al., 1970). At concentrations (50 to 100 mg/l) well above the solubility limit, with a dissolved oxygen content of 6 to 7 mg/l, the same reaction has a half-life of 80 hours at 30°C and 240 hours at 0°C (Bullock and Newlands, 1969). The relatively small temperature effect combined with the large inverse concentration effect is consistent with a diffusion controlled process. The oxidation of colloidal phosphorus in seawater is reported to be measurably slower than in fresh water, suggesting that the high salt content brings about agglomeration of the phosphorus particles. Thus, rapidly moving fresh water should lose elemental phosphorus faster than quiescent seawater.

Addison has presented evidence indicating that 1 ppm aqueous elemental phosphorus is oxidized to phosphate in a single step. Likely intermediates, though stable under the reaction conditions, could not be isolated. Blumbergs et al., 1973 report that phossy water from Pine Bluff AAP contains as much phosphorus in intermediate oxidation states as orthophosphate. A synthetic mixture prepared with several hundred ppm white phosphorus gave the same results. It is possible that the products of oxidation of suspended phosphorus are different from those of dissolved phosphorus.

The rapid reaction of white phosphorus with chlorine in water to form phosphorus acid via phosphorus trichloride is well documented (Blumbergs et al., 1973; Anon, 1952; Barber, 1969).

ANALYTICAL METHODOLOGY

Elemental Phosphorus. Until recently, the usual method for determining elemental phosphorus involved extraction of the sample with an organic
solvent such as benzena, oxidation of the extracted phosphorus to orthophosphate, and estimation of the latter by the molybdenum blue or other colorimetric method. Under ideal conditions, a detection limit of about 0.01 ppm of phosphorus in water is attainable by this technique, but of course it is not possible to distinguish $P_4$ from benzene-soluble phosphorus derivatives, phosphine in particular (McGilvery, 1973).14

Addison and Ackman have devised a gas-liquid chromatographic procedure for elemental phosphorus capable of detecting as little as $10^{-12}$g (Addison, 1971; Ackman and Addison, 1969; Addison and Ackman, 1970). Benzene extracts of mud, water, or biological samples are injected onto a gas-liquid chromatograph equipped with a flame photometric detector. Techniques have been contrived to permit detection of 0.01 µg/l of elemental phosphorus in water. (The solubility of $P_4$ in benzene is $10^6$ times its solubility in water). Some interference from organics has been noted (Addison, 1971); for natural water samples, ± 0.1 µg/l is probably the limit of precision, and for mud and biological samples, ± 0.1-1 mg/kg; depending on the nature of the sample. Using their methods, the authors and associates have analyzed for phosphorus in water (Addison et al., 1971, 1972), mud (Ackman et al., 1972), and fish (Ackman et al., 1970a, b) samples from Long Harbour.

More recently, Bohl and Kaelble (1972) of Monsanto have devised a method for the determination of white phosphorus in air, also employing flame photometric gas chromatography. They cite a detection limit of $10^{-11}$g for $P_4$ dissolved in xylene. Gorzyn (1972) has reviewed methods for elemental phosphorus through early 1970. It appears at this time that flame photometric gas chromatography is the method of choice for environmental samples.

Phosphine. Methods for phosphine have been reviewed by Fetchin and Grayson (1972). Because phosphine is usually encountered as a gas, analytical procedures are based on air sampling. A typical procedure utilizes formation of a colored complex with silver diethylidithiocarbamate (Dechant et al., 1966), the sensitivity corresponding to a level of ± 50 ppm in water. Berck et al., 1970 employing flame photometric gas chromatography, are able to detect quantities of gaseous phosphine in the picogram ($10^{-12}$g) range. Extraction and gas chromatography should be applicable to water samples containing phosphine.

Oxyacids of Phosphorus. Van Wazer (1958) and Ohashi (1972) have reviewed analytical methods for the oxyacids of phosphorus. The preferred method is separation by paper on thin layer (Seiler, 1965) chromatography and estimation of the developed spots. More recently, Addison
has employed a gas chromatographic technique based on formation of tri-
methylsilyl derivatives and utilizing the flame ionization detector
(Addison, 1971; Butts, 1970). Sensitivities in the 0.1 ppm range
are implied.

RECOMMENDATIONS FOR FUTURE EXPERIMENTAL
AND FIELD STUDIES ON WHITE PHOSPHORUS

1. The determination of the LD50 for white phosphorus should be undertaken
in two animal species.

2. Because there is no information on the long-term effects of phosphorus,
both chronic 90-day studies of low dose levels of phosphorus in 2 species
of animals as well as a 2 yr long-term feeding study of very low dose levels
of phosphorus in animal species should be undertaken, with special attention
being paid to bone development.

3. The metabolism of phosphorus using radio active $^{32}$P in animal species
should be studied especially to determine retention in bone and possibly
in other organs. The form of urinary and fecal excretion of phosphorus
should be investigated.

4. Teratogenic (especially for possible interference with bone development)
and mutagenic studies of white phosphorus might be desirable.

5. An investigation of phosphine (PH$_3$) production from elemental phosphorus
by microorganisms in both field and laboratory studies should be undertaken.

6. The recommendation made by Rosenblatt, Small, and Barkley (1973) in
their report (p. 25) viz. "Procedures for monitoring white phosphorus in
water should continue to be developed. The toxicity and analysis of lower
oxides of phosphorus and polyphosphates should be investigated", is fully
supported in this report.

7. Because elemental phosphorus is by far the most toxic constituent, the
primary data required for the assessment of phossey water pollution is the
rate of oxidation of dissolved phosphorus in the concentration range of
1-1000 mg/l. A definitive kinetic study should be initiated to determine
the effects of phosphorus concentration, dissolved oxygen content, tempera-
ture, pH and dissolved solids. From such data the time required to achieve
safe phosphorus levels could be estimated for any particular body of water.
8. Pine Bluff AAP phossy water should be analyzed for the presence of intermediate oxyacids of phosphorus and for phosphine. Acute toxicities, based on LD 50's, should be measured for those present at the mg/l level, using appropriate freshwater target species, such as fish and crayfish. For any substance found to be toxic in terms of the quantities present in the waste microbiological studies should be initiated to measure inhibition and utilization of phosphorus.

9. Although toxicity data for elemental phosphorus is available for numerous species of sea-water fish and a few lower animals, further bioassay studies on phossy water using freshwater fish, lower animals and algae is desirable. Chronic toxicity data for white phosphorus levels below 0.1 ppb would be useful for establishing maximum safe concentrations, but the rapid oxidation of dissolved phosphorus at these levels and uncertainties in the present best analytical method would appear to present awesome experimental complications.
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12. Fletcher, Dr. Garth L., Marine Science Laboratory, Memorial University of Newfoundland, St. Johns, Newfoundland, Canada (709) 726-6681. Personal communication, July, 1973.
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LITERATURE CITED (Cont)


LITERATURE CITED (Cont)


LITERATURE CITED (Cont)


LITERATURE CITED (Cont)


APPENDIX A

SOURCES CONSULTED

A. Books:


   Supplement 1 (Shubik and Hartwell), 1957.
   Supplement 2 (Shubik and Hartwell), 1969.


APPENDIX A (Cont)


B. Journals Searched for Phosphorus:

1. Chemical Abstracts, from Vol. 1, 1907 to data.

Includes 1st decennial index, 1-10 (1907-1916)
2nd " " 11-20 (1917-1926)
3rd " " 21-30 (1927-1936)
4th " " 31-40 (1937-1946)
5th " " 41-50 (1947-1956)
6th collective " 51-55 (1957-1961)
7th " " 56-65 (1962-1966)
8th " " 66-75 (1967-1971)
Vols. 76, 77 (1972), and 78 (1973) and 79 (1973) to date.

[Key words: phosphorus, white phosphorus]


[Key words: phosphorus, white]


[Key words: phosphorus, white]
APPENDIX A (Cont)

C. Computer Searches of Literature for Phosphorus:


[Key words: phosphorus, white phosphorus, phossy water, plasticized white phosphorus].
(See Search Control No. 002787)


[Key words: phosphorus, white]
APPENDIX B

UNCITED REFERENCES

A. Relevant References not Quoted in Text


B. References Not Considered Relevant

APPENDIX B (Cont)


C. References Requiring Translation


APPENDIX B (Cont)


APPENDIX B (Cont)


