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Source: Journal of Wildlife Diseases, 28(4) : 669-673

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-28.4.669>

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## White Phosphorus Poisoning of Waterfowl in an Alaskan Salt Marsh

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**ABSTRACT:** The cause of the yearly death of an estimated 1,000 to 2,000 migrating dabbling ducks (*Anas* spp.) and 10 to 50 swans (*Cygnus buccinator* and *C. columbianus*) has remained a mystery for the last ten years in Eagle River Flats (ERF), a 1,000 ha estuarine salt marsh near Anchorage, Alaska, used for artillery training by the U.S. Army. We have gathered evidence that the cause of this mortality is the highly toxic, incendiary munition white phosphorus (P<sub>4</sub>). The symptoms of poisoning we observed in wild ducks included lethargy, repeated drinking, and head shaking and rolling. Death was preceded by convulsions. Farm-reared mallards dosed with white phosphorus showed nearly identical behavioral symptoms to those of wild ducks that became sick in ERF. White phosphorus does not occur in nature but was found in both the sediments where dabbling ducks and swans feed and in the gizzards of all carcasses collected in ERF. We hypothesize that feeding waterfowl are ingesting small particles of the highly toxic, incendiary munition P<sub>4</sub> stored in the bottom anoxic sediments of shallow salt marsh ponds.

**Key words:** White phosphorus, waterfowl, mortality, salt marsh, Alaska, *Anas* spp., *Cygnus* spp.

Since 1980, an estimated 1,000 to 2,000 waterfowl deaths have been observed each year at Eagle River Flats (ERF) (61°19'N, 149°44'W), a 1,000-ha estuarine salt marsh complex on Cook Inlet near Anchorage, Alaska (USA) (Fig. 1). Infectious avian diseases, cholinesterase inhibition by pesticides, predation, trauma, or direct injury from explosions and metal fragments was ruled out as the primary cause of death (Tweten, 1989; Environmental Science and Engineering Incorporated, 1990). Poisoning from lead, other heavy metals, or well-known organic poisons such as the insecticide DDT and polychlorinated biphenyls (PCBs) also were ruled out.

In the spring of 1990, we investigated

the possibility that munitions fired into the salt marsh were the cause of the mortality. Of the various munitions fired into ERF, the smoke-producing incendiary, white or elemental phosphorus (P<sub>4</sub>), became a prime suspect. White phosphorus is known to be highly toxic to waterfowl (Coburn et al., 1950) and other animals including humans (Murphy, 1986).

Eagle River Flats has been used since 1949 as a primary impact area for artillery training by the U.S. Army at Ft. Richardson; it also may be used by ≤5,000 waterfowl on a single day during spring (May to mid-June) and fall (August to September) migrations, the periods when mortality has occurred. Dabbling ducks including pintails (*Anas acuta*), mallards (*A. platyrhynchos*), and green-winged teal (*A. carolinensis*), as well as trumpeter swans (*Cygnus buccinator*) and tundra swans (*C. columbianus*), were the most common species found dead. Since these waterfowl species feed mainly in the bottom sediments, unlike other unaffected species such as snow geese (*Chen caerulescens*) and Canada geese (*Branta canadensis*), we hypothesized that the poison was located in the bottom sediments of the shallow salt marsh ponds.

During the September 1990 migration in ERF, detailed observations of feeding ducks were made from a blind erected in a shallow salt marsh pond where large numbers of duck carcasses had been found in the past. Ducks on the surrounding shallow ponds were watched until one or more individuals became sick. The stricken bird was approached and behavior monitored.

We compared the behavioral symptoms of dying wild ducks in the salt marsh to

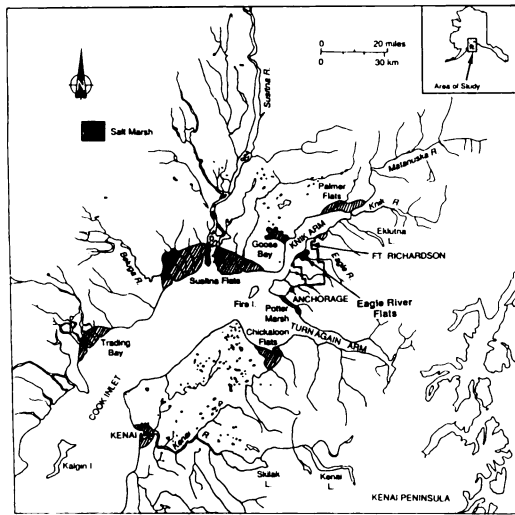


FIGURE 1. Upper Cook Inlet area in southcentral Alaska (inset) showing the location of Eagle River Flats, Ft. Richardson, and other estuarine salt marshes used by migrating waterfowl.

those of ducks given  $P_4$  (Aldrich Chemical Company, Milwaukee, Wisconsin, USA). Six adult farm-reared mallards were each gavaged with 12 mg/kg body weight of  $P_4$  dissolved in 5 ml tricaprylin oil (Sigma Chemical Company, St. Louis, Missouri, USA). Tricaprylin was used as a carrier for  $P_4$  so that the dosage could be accurately measured and orally administered. The dosage was based on an earlier study of  $P_4$  toxicity in ducks (Coburn et al., 1950) and was known to be lethal within a few hours. Each duck was dosed separately and returned to a room containing a shallow pool, food and other untreated ducks. All laboratory procedures followed the guidelines of our institutional animal care and use committee.

Tissues were collected from both the wild and the experimentally-treated ducks. Skin samples were cut from the breast using scissors and fat depots in the region of the cloaca were dissected free with a tweezers. The entire liver was excised with a scalpel. Gizzard contents were collected from the wild birds to determine if  $P_4$  was ingested. The liver and gizzard contents from five wild birds (four green-winged

teal and one pintail) that we observed die in ERF were stored in small glass vials and frozen on dry ice. In addition, carcasses of six ducks (four mallards and two pintails) and eight tundra swans found dead in ERF were collected, frozen and shipped to the laboratory for tissue and gizzard contents analyses. In September, 1990, five green-winged teal were collected in Susitna Flats (67°94'N, 150°00'W), another Cook Inlet salt marsh located 40 km from ERF (Fig. 1). The fat and gizzard contents samples from these ducks were used as controls.

In the laboratory, tissue samples were cut into small pieces and blended with degassed water under a nitrogen atmosphere. The tissue homogenate was extracted with isooctane (Aldrich Chemical Company) by shaking for 12 hr and centrifuged for 1 hr to separate the organic phase from the aqueous phase. Gizzard contents were scrapped into vials containing isooctane and shaken for 12 hr.

To confirm the presence of  $P_4$ , extracts from the gizzard contents of three ducks (a green winged teal, a pintail and a mallard), and one tundra swan were analyzed by gas chromatography-mass spectrometry (GCMS). The gas chromatograph was a 5890 Series II (Hewlett-Packard, Avondale, Pennsylvania, USA). A 1- $\mu$ l splitless injection was made onto a 5% Phenyl Methyl Silicone fused silica capillary column (25 m, 0.2 mm id) that was temperature-programmed from 75 to 100 C at 20 C/min after a 2 min hold. The Hewlett-Packard 5970 mass spectrometer was operated in scan mode over the mass range of 29 to 300 atomic mass units (amu). The mass spectrum of  $P_4$  has a base peak at mass/charge 124 amu corresponding to the molecular ion. White phosphorus also fragments into  $P^+$ ,  $P_2^+$  and  $P_3^+$ , which have peaks at mass/charge (relative abundance) 31 amu (12%), 62 amu (20%), and 93 amu (10%), respectively.

Quantitative measurements of  $P_4$  in tissue and gizzard contents were made on all samples using a Sigma 2 Gas Chromatograph (Perkin Elmer, Norwalk, Connect-

icut, USA) equipped with a flame photometric detector (GC-FPD), based on the procedures of Addison and Ackman (1970). A 1- $\mu$ l aliquot of each extract was injected onto a 15-m (0.53-mm-inner diameter) 1% methyl silicone fused silica capillary column maintained at 75 C.

Samples of salt marsh sediments were collected from the bottom of shallow ponds where wild ducks were observed to feed and die. Redox measurements were made at each site using a One Portable Meter (in millivolt mode) (Hach, Loveland, Colorado, USA) equipped with a Hach Oxidation-Reduction (ORP) electrode. Wet sediment samples (10 to 20 g) were placed in isooctane to extract the  $P_4$  and were shaken for about 12 hr and analyzed by both GCMS and GC-FPD as described.

To identify particulate  $P_4$  in the salt marsh sediments, samples positive for  $P_4$  by GC-FPD were placed in a dispersing agent (40 g/l of hexametaphosphate (J. T. Baker Chemical Company, Phillipsburg, New Jersey, USA) and washed through a 0.150-mm mesh sieve. The material left in the sieve was placed in water and examined under a stereo microscope for particulate  $P_4$ .

During 49 hours of field observations, we saw eight green-wing teal and one pintail duck violently convulse and subsequently die, each 4 to 6 hr after their arrival at the pond. The first obvious signs of poisoning in each duck were rapid head shaking and repeated drinking. These behaviors alternated with periods of lethargy during which the eyes were closed. These ducks also sought shelter in tall vegetation and could be readily approached. In the next stage of poisoning, the ducks were observed to arch their necks back, sway their heads, and swim in very tight circles. Finally, each duck convulsed, with its wings fully extended and its head arched backwards and tail up so that the head and tail nearly touched over the back. Most of the convulsing ducks would repeatedly somersault in the water and become entangled in vegetation. There was consid-

erable variation in the time of the observed signs of intoxication, but 2 to 6 hr from the observation of head shaking and drinking to convulsions and death was common.

The symptomatic behavior of mallards dosed with  $P_4$  was similar to the behavior of wild ducks dying in ERF. Following  $P_4$  administration, normal activities were observed, including wing flapping, preening, drinking, bathing and frequent movement from the pool to the floor. Within 1 to 2 hr, violent head shakes with an open bill occurred, followed by more normal behavior with mild head shakes. Four to 5 hr after  $P_4$  administration each duck showed uncontrollable head-shaking with an open bill and constant drinking followed by lethargy, and, with eyes closed, the duck sought isolation and dark areas in the cage. Finally, convulsions of varying magnitude occurred; these involved extension of the wings and arching of the head and neck over the back. Upon observation of convulsions, each moribund duck was anesthetized with a 0.4 ml intramuscular injection of 45 mg of ketamine (Aveco Company, Incorporated, Fort Dodge, Iowa, USA) and rapidly killed by exsanguination. The ketamine was used to assure sufficient analgesia. The dose of ketamine used was greater than four times that used for anesthesia.

White phosphorus was confirmed by GCMS in the gizzard contents of three wild ducks (a green winged teal, a pintail and a mallard), and one tundra swan found dead in ERF. White phosphorus was positively identified by GCMS on a limited number of samples; thereafter, quantitative measurements using GC-FPD were made on all the sample extracts.

The gizzard contents of all 19 waterfowl carcasses (8 swans and 11 ducks) collected in ERF contained  $P_4$  in widely varying amounts (Table 1). The mass of  $P_4$  in the gizzard contents varied from 0.01  $\mu$ g in a green-winged teal to 3 mg in one mallard and 11 mg in a tundra swan. Because  $P_4$  is insoluble in water and because sediment-feeding waterfowl were poisoned, we as-

TABLE 1. White phosphorus concentrations in tissues and gizzard contents from wild duck and swan carcasses collected in the Eagle River Flats salt marsh and in tissues from adult mallards dosed with 12 mg P<sub>4</sub> per kg body weight in the laboratory.

Sample	White phosphorus concentration (μg/g)		
	Wild swans	Wild ducks	Experimentally-treated mallards
Gizzard contents	52 (0.02 to 207); 8 <sup>a</sup>	304 (0.08 to 3,140); 11	NA <sup>b</sup>
Fat	0.67 (0.10 to 2.90); 7	0.21 (<d <sup>c</sup> to 0.43); 5	1.98 (0.39 to 3.52); 6
Skin	0.06 (0.01 to 0.14); 3	0.07 (0.03 to 0.13); 4	1.29 (0.59 to 2.23); 5
Liver	NA	0.05 (<d to 0.14); 5	0.25 (0.01 to 0.68); 6

<sup>a</sup> Mean (range); sample size.

<sup>b</sup> NA, Not analyzed.

<sup>c</sup> <d, less than detection limit of 0.01 μg/g.

sumed that P<sub>4</sub> was ingested as a particulate in a manner similar to that of lead shot (Roscoe et al., 1989). In addition, the fat, skin and livers of these birds contained detectable concentrations of P<sub>4</sub> (Table 1) indicating that it had been absorbed from the digestive tract. Of these tissues, fat contained the highest P<sub>4</sub> concentrations as would be expected of a lipid-soluble chemical. Since P<sub>4</sub> concentrations were highest in the fat, we analyzed fat samples from five green-winged teal from the Susitna Flats to serve as controls. We analyzed gizzard contents as well since P<sub>4</sub> was detected in the gizzard contents of all nineteen waterfowl carcasses from ERF. None of the ducks from Susitna Flats contained P<sub>4</sub> in either the gizzard contents or their body fat where the highest concentrations were found in ERF carcasses.

All farm-reared mallards that were dosed orally with P<sub>4</sub> contained P<sub>4</sub> in their fat, skin and livers (Table 1). Gizzard contents were not analyzed. The highest concentrations of P<sub>4</sub> were detected in body fat and generally were higher than the concentrations in fat from wild birds. The mechanism by which P<sub>4</sub> kills waterfowl is not known.

White phosphorus was determined by GC-FPD in six of 20 sediment samples obtained from one pond. Concentrations varied from 0.0025 to 10.2 μg/g wet weight. The presence of P<sub>4</sub> was confirmed by GCMS in the sample with 10.2 μg/g P<sub>4</sub>.

On examination, we found particulate P<sub>4</sub> in two of five sediment samples. The P<sub>4</sub> particles were waxy, transparent yellow and very irregular in shape with rough surfaces; they smoked when cut and exposed to air. Particle sizes ranged from 0.3 to 1.2 mm in the longest dimension. These sand-sized particles probably were easily distinguished and selected by sediment-feeding waterbirds from the silt and clay-sized (0.0002 to 0.005 mm) particles that made up the salt marsh sediments. White phosphorus particles also were isolated from the gizzard of a mallard carcass from ERF in which over 3 mg of P<sub>4</sub> was measured by GC-FPD. Because P<sub>4</sub> particles probably are not present in all the waterfowl feeding ponds in ERF, not all of the dabbling ducks and swans that feed in this salt marsh ingest P<sub>4</sub> particles and subsequently become sick and die.

We hypothesize that the incendiary and smoke-producing P<sub>4</sub>, as a particulate in the sediments, is responsible for the death of waterfowl in ERF for several reasons. White phosphorus is highly toxic to waterfowl at ingestion levels of a few milligrams per duck. Farm-reared adult mallards dosed with P<sub>4</sub> showed almost identical behavioral symptoms to those of wild ducks observed to die in ERF. White phosphorus was detected by GC in the gizzard contents and fat of all 11 dabbling ducks and 8 tundra swans collected in ERF but in none of five healthy teal collected in a nearby salt marsh. White phosphorus similarly was

detected in several sediment samples from the bottom of the pond in which ducks feed and were observed to die. Sand-sized particles (0.015–1.0 mm) of  $P_4$  were isolated from some of these sediment samples as well as from the gizzard of one duck. Although  $P_4$  does not occur naturally and it spontaneously oxidizes when exposed to air, the flooded and anaerobic salt marsh sediments (measured redox potential or Eh = –200 to –400 mv) apparently prevented oxidation and preserved  $P_4$  particles introduced by artillery firing.

As a result of this case of  $P_4$  poisoning, the Army has prohibited the firing of  $P_4$  munitions in the proximity of all Army wetlands nation-wide. Whether similar  $P_4$  waterfowl-induced mortality is occurring at other military bases is under investigation. Removal of  $P_4$  from sediments by dredging in ERF is not feasible because of unexploded ordnance and wetland destruction. However, other remedial options are being investigated, including oxidation, pond drainage, capping or covering of  $P_4$ -containing sediments and using avian repellents to discourage waterfowl from using areas contaminated with  $P_4$ .

The authors thank William Gossweiler (Ft. Richardson, Alaska), Captain Steve Bird (USATHAMA), Dr. Tom Jenkins, and Susan Taylor (CRREL). This work was

supported by the U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Maryland. It reflects the personal views of the authors and does not suggest or reflect the policy, practices, programs, or doctrine of the U.S. Army or Government of the United States.

#### LITERATURE CITED

- ADDISON, R. F., AND R. G. ACKMAN. 1970. Direct determination of elemental phosphorus by gas-liquid chromatography. *Journal of Chromatography* 47: 421–426.
- COBURN, D. R., J. D. DEWITT, J. V. DERBY, AND E. EDIGER. 1950. Phosphorus poisoning in waterfowl. *Journal of the American Pharmaceutical Association* 39: 151–158.
- ENVIRONMENTAL SCIENCE AND ENGINEERING INCORPORATED (ESE). 1990. Eagle River Flats expanded site investigation, Fort Richardson, Alaska, U.S. Army Toxic and Hazardous Materials Agency, Aberdeen, Maryland, 190 pp.
- MURPHY, S. D. 1986. Toxic effects of pesticides. In Casarett and Doull's toxicology, C. D. Klassen, M. O. Amdur and J. Doull (eds.). Macmillan Publishing Company, New York, New York, pp. 519–581.
- ROSCOE, D., L. WIDJESKOG, AND W. STANSLEY. 1989. Lead poisoning of northern pintail ducks feeding in a tidal meadow contaminated with shot from a trap and skeet range. *Bulletin of Environmental Contamination and Toxicology* 42: 226–233.
- TWETEN, R. 1989. Eagle River Flats waterfowl mortality investigation progress report. U.S. Fish and Wildlife Service, Anchorage, Alaska, 37 pp.

*Received for publication 23 September 1991.*