A review of episodes of zinc phosphide toxicosis in wild geese (*Branta* spp.) in Oregon (2004–2011)

Rob J. Bildfell,[1] Wilson K. Rumbeiha, Krysten L. Schuler, Carol U. Meteyer, Peregrine L. Wolff, Colin M. Gillin

Abstract. Epizootic mortality in several geese species, including cackling geese (*Branta hutchinsii*) and Canada geese (*Branta canadensis*), has been recognized in the Willamette Valley of Oregon for over a decade. Birds are generally found dead on a body of water or are occasionally observed displaying neurologic clinical signs such as an inability to raise or control the head prior to death. Investigation of these epizootic mortality events has revealed the etiology to be accidental poisoning with the rodenticide zinc phosphide (Zn_3P_2). Gross and histologic changes are restricted to acute pulmonary congestion and edema, sometimes accompanied by distension of the upper alimentary tract by fresh grass. Geese are unusually susceptible to this pesticide; when combined with an epidemiologic confluence of depredation of specific agricultural crops by rodents and seasonal avian migration pathways, epizootic toxicosis may occur. Diagnosis requires a high index of suspicion, appropriate sample collection and handling, plus specific test calibration for this toxicant. Interagency cooperation, education of farmers regarding pesticide use, and enforcement of regulations has been successful in greatly decreasing these mortality events since 2009.

Key words: *Branta*; epizootic mortality; geese; neurologic disease; pulmonary edema; rodenticide; toxicosis; zinc phosphide.

Epizootic mortality of migrating cackling geese (*Branta hutchinsii* sp.) and other *Branta* species during the winter to early spring has been recognized in the Willamette Valley of Oregon since the late 1990s, but the cause of most of these episodes could not be determined. The current study describes the investigations of multiple epizootics, with mortalities ranging from 5 to over 300 birds, occurring between 2004 and 2008. These epizootics plus at least 2 others, investigated by the Oregon Department of Agriculture (ODA) in August of 2005, were determined to be toxicoses caused by the ingestion of zinc phosphide (Zn_3P_2). Based on time of year, geographic location, and number of birds affected, it is speculated that zinc phosphide was responsible for 3 other epizootic mortality events occurring in the same area between 1999 and 2003. Although various pesticides are capable of killing large numbers of wild birds, it is unusual for a rodenticide to be implicated in large mortality events of waterfowl. The present study describes the clinicopathological findings, toxicological assay results, and the unusual epidemiologic circumstances involved in these episodes of zinc phosphide–induced mortality in wild geese.

The clinical history for these mortality events was generally similar; most geese were simply found dead in and around a body of water over the course of a few days to slightly over 2 weeks depending on the incident. Occasionally, clinical signs were observed. Signs included weakness, inability to raise the head or to control head movements, impaired locomotion in the water, and convulsions. At least 1 bird was noted to have froth in the oral cavity at the time of capture. In 1 instance, birds "crashed" in midflight into a shed located near the lakeshore. Affected live birds generally died within a few hours of capture, and some of these geese were observed to develop a progressive paralysis, moving from legs to head. Details of species of geese, the total number affected, location by county, and the approximate dates of 9 mortality events are included in Table 1. During the November 2004 event, 4 dead herring gulls (*Larus argentatus*) were also collected; for all other outbreaks, only geese mortalities were identified. This pattern was notable because, for most of the events, various species of ducks, grebes, gulls, and other birds were observed in the bodies of water where the geese mortalities occurred. True cackling geese (*Branta hutchinsii minima*) were most frequently affected, and no age or gender predilection was identified in the mortality patterns of any of these events. The herring gulls proved to have severe aspergillosis (confirmed via culture),

From the Veterinary Diagnostic Laboratory, Oregon State University (Bildfell), and Oregon Department of Fish and Wildlife (Gillin), Corvallis, OR; Department of Veterinary Diagnostic and Production Animal Medicine, Iowa State University, Ames, IO (Rumbeiha); Animal Health Diagnostic Center, Cornell University, Ithaca, NY (Schuler); United States Geological Survey National Center, Reston, VA (Meteyer); and Nevada Department of Wildlife, Reno, NV (Wolff).

[1]Corresponding Author: Rob J. Bildfell, OSU VDL, PO Box 429, Corvallis, OR 97339-0429. Rob.Bildfell@oregonstate.edu

Table 1. Epidemiologic data and ancillary tests performed during investigations of goose mortality events in Oregon, 2004–2008.*

	Outbreak date									
	11/15/2004	12/30/2004	1/20/2005	3/10/2005	3/17/2005	8/5/2007	8/9/2007	11/2/2007	1/10/2008	4/11/2008
Species	*B. hm*	*B. hm, B. ht, B. co*	*B. hm*	*B. hm*	*B. hm*	*B. hm*	*B. cm*	*B. hm, B. cm*	*B. cm*	*B. hm*
Estimated mortalities	50	80	300	60	50	5	10	10	7	67
County	Marion	Marion	Marion	Benton	Marion	Yamhill	Multnomah	Washington	Washington	Marion
Bacterial culture	*Aspergillus* (1/4)	*E. coli* (2/4)	3/3 Neg	3/3 Neg	3/3 Neg	2/2 Neg	3/3 Neg	3/3 Neg	2/2 Neg	3/3 Neg
Botulinum toxin	3/3 Neg	2/2 Neg	NT	3/3 Neg	NT	2/2 Neg	NT	NT	NT	NT
Virus isolation	4/4 Neg	1/1 Neg	4/4 Neg	NT	NT	NT	NT	3/3 Neg	4/4 Neg	NT
AIV PCR	NT	NT	4/4 Neg	3/3 Neg	3/3 Neg	3/3 Neg	1/5 Pos	3/3 Neg	4/4 Neg	3/3 Neg
WNV PCR	Neg	2/2 Neg	NT	NT	3/3 Neg	2/2 Neg	2/2 Neg	NT	NT	NT
Lead	NT	4/4 Neg	3/3 Neg	NT	3/3 Neg	NT	NT	NT	NT	3/3 Neg
Cholinesterase activity	2/2 Norm	4/4 Norm	4/4 Norm	3/3 Norm	3/3 Norm	2/2 Norm	2/2 Norm	NT	NT	3/3 Norm
GC-MS for zinc phosphide	NT	3/3 Pos	2/2 Pos	3/3 Pos	3/3 Pos	1/1 Pos	3/3 Pos	2/2 Pos	2/2 Pos	3/3 Pos
Miscellaneous assays	NT	NT	2/2 Neg†	3 /3 Neg‡	NT	1/1 Neg§	NT	NT	NT	NT

* *B. hm = Branta hutchinsii minima; B. ht = Branta hutchinsii taverneri; B. co = Branta canadensis occidentalis; B. cm = Branta canadensis moffitti;* AIV = avian *Influenza A virus;* PCR = polymerase chain reaction; WNV = *West Nile virus;* GC-MS = gas chromatography–mass spectrophotometry; Neg = negative; Pos = positive; Norm = normal; NT = not tested.
† Gastrointestinal tract and/or liver tested for bromethalin, roquefortine, penitrem A, strychnine, metaldehyde, and mycotoxins.
‡ Liver tested for strychnine and cyanide.
§ Liver tested for anticoagulant rodenticides and strychnine.

and the mortalities were interpreted as unrelated to the process affecting the geese.

Antemortem testing on 2 affected geese included a complete blood cell count (1 bird) performed on whole blood collected in EDTA, a partial serum biochemical profile from both birds, plus whole blood samples (both birds) for botulism testing. Samples were collected via wing vein venipuncture of the 2 moribund birds. Findings included a moderate leukopenia and a marked lymphopenia, but a normal packed cell volume with 1+ polychromasia.[15] When compared to normal ranges published for either Aleutian Canada geese or domestic geese[9,10] (depending on the analyte being assayed), biochemical values were unremarkable for calcium, phosphorus, and uric acid for both geese, but one of the birds had decreased serum protein (3.5 mg/dl; range: 4.1–5.5 mg/dl) and albumin (1.5 mg/dl; range: 1.8–2.3 mg/dl). Both birds had elevated creatine kinase values (1,181 and 471 U/l, respectively; normal range: 165–378 U/l) and aspartate aminotransferase levels (100 and 710 U/l, respectively; normal range: 55–87 U/l). Both birds also had marked hypoglycemia (69 and 32 mg/dl, respectively; normal range: 195–288 mg/dl).

During the investigation of these outbreaks, representative mortalities were examined at the U.S. Geological Survey, National Wildlife Health Center (NWHC; Madison, Wisconsin) and/or the Oregon State University Veterinary Diagnostic Laboratory (OSUVDL; Corvallis, Oregon). In some instances, the birds had been frozen by the Oregon Department of Fish and Wildlife biologists at the time of collection. Necropsy examinations were generally performed on 3–6 birds per outbreak. Toxicology samples collected from mortalities were tested for various chemicals/agents at the NWHC and the Diagnostic Center for Population and Animal Health (DCPAH; Michigan State University, Lansing, Michigan). Table 1 lists many of the toxicological test results from the various mortality events. A general summary of overall findings plus some details regarding test protocols and results are provided below.

Gross necropsy findings. Most birds were in good body condition and often had fresh grass in the stomachs and/or esophagus (Fig. 1). Grain was observed in the proventriculi in some birds during the January 2008 incident. Gross lesions generally consisted of pulmonary congestion and edema, sometimes with concurrent mild renal pallor. Hepatic congestion was also sometimes observed.

Histopathology. A wide variety of tissues (brain, lung, trachea, liver, kidney, spleen, gonad, heart, skeletal muscle, pancreas, esophagus, proventriculus, ventriculus, large and small intestine, peripheral nerve, and less frequently, spinal cord) were collected into 10% neutral buffered formalin and routinely processed for staining with hematoxylin and eosin. For all but 1 bird, no pathologic changes were detected in the samples of brain, spinal cord, or peripheral nerves examined. Pulmonary congestion and edema was generally present but, in many instances, this histologic diagnosis was confounded

Figure 1. Zinc phosphide toxicosis in a cackling goose (*Branta hutchinsii minima*). The esophagus is filled with fresh rye grass from a field treated with the rodenticide.

by postmortem change and freezing artifact. The cellular response to this intrapulmonary fluid was mild to absent. Renal changes were inconsistently present and were limited to mild tubular nephrosis, sometimes complicated by mild renal coccidiosis, presumed to be *Eimeria truncata*.[9] Several geese had evidence of schistosomiasis with granulomatous lesions, with or without parasites, visible in the enteric proprial tissues and vasculature plus occasionally minor hepatic involvement.[9] Mild ventriculitis associated with *Amidostomum* spp. infection was seen in a few birds. Overall, the parasite-associated lesions were mild and deemed incidental. Potentially significant inflammatory lesions were limited to mild nonsuppurative encephalitis in 1 bird and a multifocal lymphoplasmacytic myocarditis in another.

Bacteriology. Culture of visceral samples (minimum liver for all birds sampled, often intestine, lung and/or spleen, as well) of several birds from multiple outbreaks onto blood and MacConkey agar plates was unrewarding. A single bird with *Aspergillus* was identified, in addition to various isolates of mixed environmental bacteria in some of the other birds. Mouse inoculation assays for detection of botulinum toxin type C in the blood from live clinically ill geese (2005 outbreak) were negative as were tests utilizing heart and clotted blood from carcasses during 3 other outbreaks.[12]

Virology. Testing at the NWHC included attempts at virus isolation utilizing samples collected from 5 of the outbreaks via inoculation of oropharyngeal and cloacal swabs into embryonated chicken eggs. The swabs were also utilized for real-time polymerase chain reaction (real-time PCR) tests for highly pathogenic avian *Influenza A virus* using published procedures and primers.[13] A single positive culture in 2007 proved to be a non-H5, non-H7 avian influenza strain. All real-time PCR testing on multiple birds at NWHC and/or OSUVDL was negative in 6 out of 6 outbreaks. In some instances, testing of oropharyngeal and cloacal swabs for Newcastle disease by real-time PCR and of brain tissue for

West Nile virus[4] was performed; these test results were uniformly negative.

Toxicology. Tests for cholinesterase activity in brain tissue were within normal ranges in 6 out of 6 events investigated.[7] Hepatic lead levels were not elevated in the birds tested (4 events) using flame atomic absorption after microwave digestions.[3] Initial gas chromatography–mass spectrophotometry (GC-MS) assays of liver and stomach contents in the 2004 and 2005 outbreaks were negative for toxic compounds. However, subsequent analyses at the DCPAH laboratories using modified techniques designed to detect phosphine implicated the zinc phosphine toxin. The technique modification consisted of acid extraction of the ingesta plus the use of a separation column capable of detecting the phosphine gas that became trapped in toluene solvent.[14] Such a test method was subsequently employed during geese mortality investigations and retrospectively applied to samples from the 2004 outbreak.

Although not performed on the majority of outbreaks, assays for the following poisons were also performed 1 or more times, utilizing liver and/or stomach contents from affected birds: metaldehyde, strychnine, cyanide, and a panel for anticoagulant rodenticides and bromethalin. Results were uniformly negative.

Mineral analyses of the stomach contents of 6 confirmed zinc phosphide–induced mortalities revealed the following zinc values: 3,630; 3,300; 2,760; 1,820; 706; and 539 ppm wet weight, respectively. This contrasts with the zinc content of grasses and cereal foodstuffs normally ingested by geese, which includes an upper range of only 120 ppm wet weight.[6]

Mass mortality events in birds can be due to a wide array of etiologies ranging from infectious agents, such as *Pasteurella multocida* or highly pathogenic avian *Influenza A virus*, to intoxications, such as botulism or the ingestion of avicides such as 4-aminopyridine. The clinical signs observed during many of the mortality events in the current study, including those occurring prior to the study (only partial records available prior to 2003), were interpreted as indicative of neurologic disease. Investigations therefore specifically pursued botulism, lead poisoning, and organophosphate and carbamate toxicity as key differential diagnoses. The observation of convulsions also led investigators to request strychnine assays. Repeated negative findings for these toxicants, as well as for various highly pathogenic infectious agents, emphasize the importance of zinc phosphide as the culprit in this long-standing local problem.

The Willamette Valley and lower Columbia River are part of the Pacific flyway and feature several state and National Wildlife Refuge areas designed to enhance and protect certain populations, including cackling geese and certain subpopulations of *Branta canadensis*. Large amounts of commercial grass seed are produced in Oregon with annual production figures during the past decade (2000–2010) involving nearly 200,000 hectares and associated farm gate revenues of approximately US$200 million (ODA: 2010,

Oregon agriculture: facts and figures. Available at: http://www.oregonexplorer.info/data_files/OE_location/northcoast/documents/NorthCoastPDFs/ff.pdf. Accessed on November 20, 2012). In the Willamette Valley, flocks of migrating geese commonly graze on this valuable crop, resulting in considerable economic loss and a source of conflict between wildlife managers and agriculturalists. Indeed, cackling geese are recognized as voracious grazers in comparison to other geese species. However, the mortality events described herein are not believed to be malicious acts to control marauding geese. Instead, the deaths were mainly due to the application of zinc phosphide for the control of rodents, especially meadow voles (*Microtus* spp.), which also damage the grass crop. Licensed applicators may use these products by ground broadcasting along rows or by depositing it within rodent burrows, provided various restrictions (e.g., quantity, time of year, etc.) are observed.

The use of zinc phosphide has been popular in the United States for decades, with the first product registered as a pesticide in 1947 (U.S. Environmental Protection Agency: 1998, R.E.D. facts: zinc phosphide. Prevention, pesticides and toxic substances (7508W). EPA-738-F-98-003. Available at: http://www.epa.gov/oppsrrd1/REDs/factsheets/0026fact.pdf. Accessed on November 20, 2012). Granular, powdered, pelleted, and grain-baited forms are available. Related products utilize aluminum or magnesium as the phosphide carrier. The mechanism of action of such compounds involves the liberation of phosphine gas and free radicals when the parent compound is exposed to an acidic fluid. Phosphine can block cytochrome oxidases, preventing mitochondrial metabolic processes and energy production.[1,5] The neurologic signs described herein likely reflect the blockade of oxidative phosphorylation in the brain, a tissue highly dependent on this metabolic pathway.[1] Zinc phosphide–induced suppression of acetylcholine esterase has also been proposed but the results of cholinesterase assays in these geese do not support this hypothesis.[5] Hypoglycemia, as seen in the 2 birds sampled in the present study, has been observed in human beings and has been speculated to be a consequence of impairment of hepatic glycogenolysis and gluconeogenesis.[2] Hypoglycemia is not commonly reported in zinc phosphide toxicosis cases involving animal species. The various reactive oxygen species generated compound the cellular damage and can contribute to hepatic, renal, muscular, and myocardial damage.[1] The serum enzyme elevations in the 2 birds tested herein could be a combination of exertional muscle damage and/or zinc phosphide–induced cellular damage to muscle and liver.

Zinc phosphide has an unusual odor described variably as acetylene to rotting fishlike (U.S. Environmental Protection Agency: 1998, R.E.D. facts: zinc phosphide). The compound is registered in Toxicity Category I (highest of 4 categories) due to the potential for lethal acute effects via oral or inhalation routes of exposure. Zinc phosphide poisoning of human beings is rare, provided handling instructions are observed. Although primary mortalities in nontarget species, especially

birds, have been reported on numerous occasions, there are several factors that reduce the threat of accidental poisonings. Secondary toxicity problems due to consumption of targeted rodent species by predators and scavengers is less of a concern than with many other pesticides because zinc phosphide does not accumulate in muscle and decomposes readily in the digestive tract. Other factors reducing both primary and secondary toxicity problems in nontarget species include an emetic effect (useful since most rodent species are less capable of vomition), a lower LD_{50} (median lethal dose) for most target species in comparison to most predator species, and a tendency for predators to avoid digestive tracts containing this pesticide (Johnson GD, Fagerstone KA: 1994, Primary and secondary hazards of zinc phosphide to nontarget wildlife—a review of the literature. U.S. Department of Agriculture, Animal and Plant Health, Denver Wildlife Research Center Research Report no. 11-55-005. Available at: http://www.aphis.usda.gov/wildlife_damage/nwrc/publications/znp_primary_secondary.pdf. Accessed on November 20, 2012).

Reports of accidental zinc phosphide poisoning of avian species include peafowl, chickens, wild turkeys, and geese (Mohr J: 1959, The Oregon meadow vole eruption of 1957–1958: influences of the poisoning program on wildlife. Federal Cooperative Extension Service, pp. 27–34).[8,11,14] One of the earliest documented zinc phosphide–induced mortality events in North America involved the control of voles in the Klamath Basin of Oregon when over 3,000 geese were thought to have been poisoned (Mohr J: 1959). The report cited the observation of geese "dropping out of flight in an erratic manner." Application methods for the rodenticide at that time tended to be less discriminate and included aerial broadcasting, yet ducks and upland game birds were minimally affected. A review of the literature considering LD_{50} values for target rodent species gives figures generally ranging from 18 to 40 mg/kg with even higher values for most carnivore and ruminant species. Geese are unusually susceptible, with LD_{50} of 12.0 mg/kg for Canada geese, and values of 7.5 and 8.8 mg/kg for white-fronted geese and snow geese, respectively (Johnson GD, Fagerstone KA: 1994, Primary and secondary hazards of zinc phosphide). Such dosages are generally lower than those identified for other avian species. A 1962 California Fish and Game study indicated that snow geese consuming grain treated with 1% Zn_3P_2 could ingest several lethal dosages per feeding (Johnson GD, Fagerstone KA: 1994). Most products currently licensed for use in Oregon contain 2% Zn_3P_2.

The zinc phosphide family of rodenticides is a popular tool in agriculture, in large part due to the decreased secondary and nontarget species effects already mentioned. An investigation of a 2008 goose mortality event documented 151 purchases of this restricted use pesticide from licensed dealers in Oregon, totaling over 26,000 kg, during the 6-week period preceding the event (F500 Report of ODA investigation #084204, Pesticide Analytical Response Center, August

12, 2008). The Pesticides division of the ODA is responsible for regulating the use of this chemical in the Oregon agriculture industry and considers input from the industry, as was illustrated in 2005. The vole population that year was perceived to be rapidly increasing, prompting a temporary mild easing of regulations of the Special Local Need (SLN) permits for above-ground use of zinc phosphide for the seed grass industry. Unfortunately, in addition to the epizootics listed for 2005 in Table 1, there were at least 2 other zinc phosphide–related goose mortality events in 2005 (documented by ODA but not included in Table 1). These epizootics may or may not have been the result of regulation changes but did generate considerable press due to concern in public and political arenas. This is an example of how regulatory agencies balance the promotion of profitable and productive agriculture while maintaining the agency mandate to protect the interests of the public and the environment. This balancing act is often more difficult when the public perceives wildlife to be victimized or has a poor understanding of the demands of production-oriented agricultural practices. The goose mortality events have prompted the ODA to launch a vigorous outreach effort aimed at better educating the agricultural industry employing zinc phosphide products (http://www.oregon.gov/ODA/PEST/docs/pdf/zp_geese_outreach.pdf). Methods include brochures, items in quarterly newsletters, and educational presentations on the proper use of both zinc phosphide and alternative non-chemical methods of vole control. In 2008, advisory bulletins were sent out quarterly to licensed applicators reminding them of the regulations. Labels on SLN products emphasize the potential threat to birds, specifically mentioning the threat to geese: products should not be applied if geese have been observed on a field within the last 7 days, and geese should be hazed off fields treated with zinc phosphide during the previous week. Penalties have also been emphasized in press releases, posters, and on product labels, citing up to US$10,000 in fines for failing to comply with the state pesticide regulation act (ORS 634) or the Migratory Bird Treaty Act. In 2008–2009, penalties totaling US$8,405 were issued to 5 producers found to be in violation of ORS 634.

In addition to education and regulation efforts, interagency cooperation has led to modifications of the use of this pesticide in areas frequented by migrating geese. In 2009, the ODA, in consultation with the Oregon Department of Fish and Wildlife and the U.S. Fish and Wildlife Service, delayed the start date for above-ground application of zinc phosphide because cackling geese had not yet migrated out of the area. These various measures appear to have been largely effective as, to the authors' knowledge, no zinc phosphide–associated goose mortality events were documented in Oregon between January 2009 and May 2012.

The diagnosis of zinc phosphide toxicosis is problematic for several reasons. If observed, clinical signs in affected birds tend to be vague, ranging from ruffled feathers and

diarrhea seen in some gallinaceous species (Johnson GD, Fagerstone KA: 1994, Primary and secondary hazards of zinc phosphide) to the neurologic signs seen in some of the geese described herein. Gross lesions are generally confined to pulmonary congestion, which is a common nonspecific change in avian mortalities. Microscopic findings of pulmonary congestion and edema are also often equivocal, especially if the birds have been frozen or are moderately autolytic. Depending on the dose ingested, the compound may not result in death until 3–18 hr later. Therefore, carcasses of poisoned geese can be discovered miles away from the site of toxin ingestion. Nevertheless, this is a labile compound, hydrolyzing rapidly to phosphine gas under damp acidic conditions. Samples should be kept frozen in an airtight container during shipping; this also helps protect against human exposure to this dangerous gas. Veterinarians and diagnosticians are at risk when handling biologic materials containing these compounds and the American Veterinary Medical Association has issued a warning to this effect (https://www.avma.org/KB/Resources/Reference/Pages/Phosphine-product-precautions.aspx). Finally, as the authors of the current study discovered, without an acid extraction step, the phosphine gas will not be released and a GC-MS result will be negative. If strongly suspicious of this toxicosis, then a nonspecific but rapid qualitative test that has been suggested is the exposure of silver nitrate filter paper to fresh stomach contents (or vomitus); this should generate the formation of a dark gray color on the paper as the phosphine gas reacts to form silver phosphide (Balali-Mood M: 1991, Phosphine. International Programme on Chemical Safety Poisons Information Monograph 865. Available at: http://www.inchem.org/documents/pims/chemical/pim865.htm. Accessed 2011). A positive result for this test should be confirmed via GC-MS. If postmortem decomposition is advanced, prohibiting the detection of phosphine, then analysis of ingesta for zinc content may be useful; values can be elevated as seen herein and as was reported for poisoned wild turkeys.[8] However, false negatives can occur in avian species, such as geese, because the ingestion of small amounts of zinc phosphide can prove lethal without significantly altering the amount of total metal detected in the ingesta.[14] The definitive diagnosis of zinc phosphide toxicosis requires a high index of suspicion and appropriate handling of this labile and potentially hazardous toxin.

Acknowledgements

References

1. Albretsen JC: 2004, Zinc phosphide. *In*: Clinical veterinary toxicology, ed. Plumlee KH, pp. 456–458. Mosby, St. Louis, MO.
2. Frangides CY, Pneumatikos IA: 2002, Persistent severe hypoglycemia in acute zinc phosphide poisoning. Intensive Care Med 28:223.
3. Franson JC, Smith MR: 1999, Poisoning of wild birds from exposure to anticholinesterase compounds and lead: diagnostic methods and selected cases. Semin Avian Exotic Pet Med 8:3–11.
4. Lanciotti RS, Kerst AJ, Nasci RS, et al.: 2000, Rapid detection of West Nile virus from human clinical specimens, field-collected mosquitoes, and avian samples by a TaqMan reverse transcriptase-PCR assay. J Clin Microbiol 38:4066–4071.
5. Nath NS, Bhattacharya I, Tuck AG, et al.: 2011, Mechanisms of phosphine toxicity. J Toxicol 2011:494168.
6. National Research Council: 1994, Composition of feedstuffs used in poultry diets. *In*: Nutrient requirements of poultry, 9th revised ed., pp. 61–79. National Academy Press, Washington, DC.
7. Nostrandt AC, Duncan JA, Padilla S: 1993, A modified spectrophotometric method appropriate for measuring cholinesterase activity in tissue from carbaryl-treated animals. Fundam Appl Toxicol 21:196–203.
8. Poppenga RH, Ziegler AF, Habecker PL, et al.: 2005, Zinc phosphide intoxication of wild turkeys (*Meleagris gallopavo*). J Wild Dis 41:218–223.
9. Routh A, Sanderson S: 2009, Waterfowl. *In*: Handbook of avian medicine, ed. Tully TN, Dorrestein GM, Jones AK, 2nd ed., pp. 286–308. Saunders Elsevier, New York, NY.
10. Samour J: 2008, Appendix 3. *In*: Avian medicine, 2nd ed., p. 449. Mosby Elsevier, New York, NY.
11. Shivaprasad HL, Galey F: 2001, Diphacinone and zinc phosphide toxicity in a flock of Peafowl. Avian Pathol 30:599–603.
12. Smith, GR: 1980, Concentrations of toxin in the serum of waterfowl with botulism. Vet Rec 107:513.
13. Spackman E, Senne DA, Myers TJ, et al.: 2002, Development of a real-time reverse transcriptase PCR assay for type A influenza virus and the avian H5 and H7 hemagglutinin subtypes. J Clin Microbiol 40:3256–3260.
14. Tiwary AK, Puschner B, Charlton BR, Filigenzi MS: 2005, Diagnosis of zinc phosphide poisoning in chickens using a new analytical approach. Avian Dis 49:288–291.
15. Williams JI, Trainer DO: 1971, A hematological study of snow, blue and Canada geese. J Wild Dis 7:258–265.