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## Toxicity and Hazard of Vanadium to Ducks and Geese

In the winter of 2003, a report of dead and dying Canada geese (*Branta canadensis*) at a petroleum refinery was received by the State of Delaware. Apparently geese were attracted to the 4-hectare fly ash pond at the refinery. Forensic investigation by staff of the U.S. Geological Survey National Wildlife Health Center and the U.S. Fish and Wildlife Service National Fish and Wildlife Forensics Laboratory suggested that ingestion of vanadium (V) from the pond might have been the cause of the mortality event. Notably, liver and kidney samples from dead geese contained high concentrations of the metal vanadium; values of other toxic metals (e.g., arsenic, cadmium, chromium, lead, mercury, nickel, selenium and thallium) were well below levels causing adverse effects in wildlife.



Because of the paucity of V toxicity data for wild birds, controlled exposure studies were undertaken in waterfowl. In 7-day single oral dose trials with mallards (*Anas platyrhynchos*), both vanadium pentoxide and sodium metavanadate were found to be moderately toxic (median lethal dose of 113 and 75.5 mg/kg body weight). In a controlled exposure study with Canada geese, sodium metavanadate was found to be even more toxic than to mallards. Dosed birds were found to have intestinal tissue lesions. Vanadium accumulation in liver and kidney was proportional to the administered dose, and predictive analyses indicate that V concentrations of 10 µg/g dw in liver and 25 µg/g dw in kidney are associated with mortality. In multi-week chronic dietary feeding trials, sodium metavanadate resulted in modest V accumulation in liver and kidney, mild intestinal hemorrhage, some blood chemistry changes and evidence of hepatic oxidative stress in mallards.

### Management Implications

Concern for V toxicity to wild birds originally stemmed from its high concentration in petroleum crude oil and the potential for adverse effects that might accompany crude oil spills. Vanadium mobilization in the environment occurs naturally, and from man's activities including mining activities, steel production, processing of oil and fuel combustion. As

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with most metals, the principal exposure route in wildlife is ingestion of food, sediment and water. Based upon the present study, it seems unlikely that chronic low-level dietary exposure to V poses a direct lethal hazard to wildlife. However, point sources, such as the V laden fly ash impoundment encountered by geese that died in Delaware, certainly do pose a hazard to wildlife. Unfortunately another goose die-off occurred at the very same refinery fly ash pond in 2005. Whether or not the V-linked goose die-offs are isolated events is currently under investigation by the Federal Interagency Testing Committee of the Toxic Substances Control Act. Data from the U.S. Environmental Protection Agency Toxics Release Inventory indicates that 18 million

pounds of V was released into surface impoundments at 172 facilities in 2002, and several of these sites are located in key migratory waterbird flyways. It is certainly possible that some of these impoundments might pose a hazard to migratory birds.

For a more detailed description of this work and its findings, see:

**Rattner, B.A., M.A. McKernan, K.M. Eisenreich, W. Link, G. Olsen, D.J. Hoffman, K.A. Knowles and P.C. McGowan. 2006. Toxicity and hazard of vanadium to mallard ducks (*Anas platyrhynchos*) and Canada geese (*Branta canadensis*). Journal of Toxicology and Environmental Health. Part A, 69:331-351.**

