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Authors: Mörner, Torsten, and Petersson, Lars

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SHORT COMMUNICATIONS

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Lead Poisoning in Woodpeckers in Sweden

Torsten Mörner[1,3] **and Lars Petersson**[2] [1] Department of Wildlife, National Veterinary Institute, P.O. Box 7073, S-750 07 Uppsala, Sweden; [2] Department of Chemistry, National Veterinary Institute, P.O. Box 7073, S-750 07 Uppsala, Sweden; [3] Corresponding author (E-mail: Torsten.Morner@sva.se).

ABSTRACT: Lead poisoning was demonstrated in two gray-headed woodpeckers (*Picus canus*) and one white-backed woodpecker (*Dendrocopus leucotos*) in Sweden; they had liver lead levels between 9.4 and 26.2 mg^{-1} wet weight. At necropsy one gray-headed woodpecker showed signs of emaciation and the other one had severe traumatic injuries, caused by a cat. The white-backed woodpecker died in the transportation box during a translocation program. The source of the lead could not be determined, but it was suspected that it may have originated from lead pellets shot into trees and picked out by the woodpeckers during food search.

Key words: Dendrocopus leucotos, gray-headed woodpecker, lead poisoning, *Picus canus,* white-backed woodpecker.

In Sweden mortality in wildlife is continuously monitored in a national program at the National Veterinary Institute (NVI; Uppsala, Sweden) covering a range of different species of mammals and birds (Mörner, 1992). The monitoring program includes a routine post mortem examination including histology, and in many cases are special investigations carried out as bacteriology, virology, parasitology, and/or chemical analysis. In addition are chemical analyses performed randomly without indications of intoxication.

In the early 1990's three woodpeckers, two gray-headed woodpeckers (*Picus canus*) and one white-backed woodpecker (*Dendrocopus leucotos*), were examined at NVI, and on chemical analysis were found to be lead poisoned. The gray-headed woodpeckers were found at two different localities, approximately 100 km apart, in the middle part of Sweden (61°15′N, 17°E, 60°30′N, 15°E), and the white-backed woodpecker originated from Latvia and was intended to be released in the middle part of Sweden. The birds were necropsied and inspected macroscopically. The cause of death in one of the gray-headed woodpeckers was emaciation and, in the second, traumatic injuries caused by a cat. The white-backed woodpecker died in the transportation box during a translocation program and showed signs of acute stress with acute congestion in the liver, spleen, lungs, and kidneys. Necropsy revealed no findings of lead pellets in the gastrointestinal tract. Tissue samples from liver, spleen, heart, kidneys, and brain were taken for microscopic examination and fixed in 10% neutral buffered formalin. Formalin fixed organs were embedded in paraffin, cut in 5μm thick slices, and stained with hematoxylin and eosin (Luna, 1968). Histological examination showed no signs of pathological changes, nor any lesions typical for lead poisoning such as inclusion bodies in the kidneys (Bageley and Locke, 1967).

For chemical analysis, 1 to 5 g of liver and kidney tissues were wet ashed. The samples were placed in ashing tubes of borosilicat glass (Foss-Tecator AB, Höganäs, Sweden). Oxidizing acid (15 ml), containing 65% nitric acid and 70% perchloric acid (7:3 by volume) was added. The ashing procedure was performed automatically overnight (Frank, 1976; Frank and Petersson, 1983), using a electrically heated aluminium block connected to a microprocessor for control of temperature and time (Tecator Digestion System, Model 40, Foss-Tecator AB, Höganäs, Sweden). The digested solution was evaporated under filtered air.

Prior to determination of lead, the res-

idue in the ashing tube was dissolved in 10 ml ionic buffer (2 M HNO_3 and 0.25 M $LiNO_3$) (Frank and Petersson, 1983, 1985). The element was then determined by using atomic emission spectrometry technique (direct-current plasma-DCP-atomic emission spectrometer (model SpectaSpan IIIA, Applied Research Laboratories Inc., Valencia, California, USA) and inductively coupled plasma-ICP-atomic emission spectrometer (model JY 50 P, Instruments S.A., division Jobin Yvon, Longjumeau, France). The limit of detection was 0.02 mg kg^{-1}. All results were expressed as mg kg^{-1} tissue wet weight.

Chemical analysis showed lead levels of 20 mg kg^{-1} in the liver and 76 mg kg^{-1} in the kidneys of the gray-headed woodpecker that died from emaciation, 9.4 mg kg^{-1} in the liver and 1.4 mg kg^{-1} in the kidneys of the gray-headed woodpecker killed by the cat, and 26.2 mg kg^{-1} in the liver and <0.02 mg kg^{-1} in the kidneys of the white-backed woodpecker.

Lead is a non-essential and toxic metal that is spread in the environment mainly due to human activities (Asplund, 1979). As a result of lead contamination of the environment wildlife is exposed to varying amounts of dietary lead. The degree of lead exposure may differ between species due to diet and lead content in different food sources (DiGuilio and Scanlon, 1984). Gunshot pellets are the most frequent lead source in lead poisoned anseriform birds as well as in other bird species (Pain, 1996), while lead poisoning in mammals is attributed to other sources of lead such as paint and batteries (Ma, 1996). Lead from petrol, lead cables, and lead-containing paints also have been discussed as possible sources for birds (Borg, 1975) and in a few cases minewastes are known to have caused lead toxicosis (Blus et al., 1991).

Lead levels in birds are normally monitored in liver and/or kidney. Experimental studies with Canada geese (*Branta canadensis*) have shown that a liver lead level of ≥ 5 mg kg^{-1} was indicative of lead toxicosis (Cook and Trainer, 1966). In mallards (*Anas platyrhynchos*) lead levels of 6–20 mg kg^{-1} in kidney and >3 mg kg^{-1} in brain were indicative of acute lead exposure (Danell and Andersson, 1975). Consequently, liver and kidney lead levels of ≥ 5 mg kg^{-1} should be considered diagnostic of lead poisoning in birds.

Lead poisoning in Sweden is most often observed in anseriform birds (swans, geese and ducks) (Frank and Borg, 1979) and has been recognized in North America for more than a century (Wobeser, 1981). Lead as a source of intoxication was first studied as a problem for Anseriformes by Bellrose (1959). Lead poisoning also has been reported in birds from orders Falconiformes, Columbiformes, Galliformes, Charadriiformes, Gruiformes, Ciconiformes, Gaviformes, and Strigiformes (Franson, 1996), not from the order Piciformes. In the present study, we observed lead poisoning in three woodpeckers. No macroscopically visible pellets were found in the gizzards at necropsy, but birds were not examined radiographically and not especially studied for the presence of lead in the gastrointestinal tract.

Both gray-headed and white-backed woodpeckers are birds that normally are found in remote forested areas feeding mainly on insect larvae in trees like aspen (*Populus tremula*) and willow (*Salix* sp.) (Rosenberg, 1953). During winter, gray-headed woodpeckers may be found at bird feeders close to human settlements. It may be that these birds were exposed to lead during their normal behavior of foraging for invertebrates in and on tree trunks. A probable cause of deposition of lead in these trees are lead pellets from hunting with shotguns. When shooting in a forest, pellets will penetrate the bark or deeper layers of trees that are in the line of fire. Holes made in the bark resemble holes made by insects and may signal woodpeckers that insect larvae may reside in these trees, and to increase their foraging there. This behavior might result in wood-

peckers ingesting lead pellets, mistaking them for food items.

Our findings have resulted in further studies on lead exposure and tissue levels in approximately 40 dead woodpeckers of other species that occur in Sweden (*Dendrocopus* spp., *Picus viridis*, *Dryocopus martius*). So far, no new cases with elevated levels of lead or any findings of lead pellets in the gizzards of these birds have been demonstrated (T. Mörner, unpubl. data). Gray-headed and white-backed woodpeckers are both rare species in Scandinavia (Rosenberg, 1953; Aulen and Carlsson, 1990) and our findings of lead poisoning in these species may indicate a special risk for these declining populations of woodpeckers.

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