POTENTIAL SOURCES OF LEAD EXPOSURE FOR BALD EAGLES: A RETROSPECTIVE STUDY

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EXTENDED ABSTRACT.—A 12-year (1996–2008) retrospective study of lead poisoning in Bald Eagles (*Haliaeetus leucocephalus*) was conducted at The Raptor Center at the University of Minnesota. The objectives of this study were to 1) Investigate trends in lead poisoning as a cause of morbidity and mortality in Bald Eagles admitted to The Raptor Center and 2) Examine evidence to determine the potential source(s) of the lead and more specifically to investigate incidence with reference to hunting of White-tailed Deer.

For the former objective, the incidence of lead poisoning from this 12-year period was compared to the incidence of lead poisoning reported on for the preceding 16 years (Kramer and Redig 1997). The latter objective was investigated based on the analysis of four epidemiological parameters: a) temporal/seasonal prevalence and relationship with deer hunting season start dates in Minnesota, Wisconsin and Iowa; b) spatial/geographical data (correlation of the animal recovery location with deer hunting zones); c) lead isotope ratio analysis of blood and metal fragments (found in the gastrointestinal tract) of lead-poisoned Bald Eagles; and, d) comparison of kidney copper concentration from lead-exposed vs. not-exposed eagles.

Our results showed a continuing trend on the incidence of lead poisoning in Bald Eagles admitted at The Raptor Center. No significant difference was seen in the number of cases admitted per year between the current 12-year period and the preceding 16-year period. Similarly, the mean blood lead concentration remained unchanged with a low level chronic exposure predominating.

A temporal-spatial association was found between deer hunting season onset and incidence of eagle poisoning. The majority of cases occurred during late fall and early winter. A significantly higher number of poisoned Bald Eagles were recovered from the deer hunting rifle zone, suggesting a greater bioavailability of lead fragments when compared to the shotgun zone.

The lead isotope ratio analysis yielded the following results. First, most of the paired blood-metal fragments samples have a closely matched isotopic signature and secondly, the isotope ratio of the majority of the blood samples and stomach contents samples (from lead exposed eagles) were within the isotope ratio from ammunition samples reported by Church et al. (2006).

The kidney copper concentration was significantly higher in lead exposed eagles. This implies that copper fragments (from copper-jacketed rifle bullets) are being ingested by eagles along with lead fragments.

Lead poisoning continues to be a cause of morbidity and mortality to Bald Eagles in the Upper Midwest (Minnesota, Wisconsin and Iowa). We conclude that none of the four epidemiological parameters examined here can be used as standalone evidence as to the source of the lead; however, taken together, they significantly reduce the validity of any other possible explanations other than ammunition of lead exposure for Bald Eagles. *Received 31 May 2008, accepted 25 September 2008.*

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