

Bullet Fragments in Deer Remains: Implications for Lead Exposure in Avian Scavengers

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Abstract

Bullet fragments in rifle-killed deer (*Odocoileus* spp.) carrion have been implicated as agents of lead intoxication and death in bald eagles (*Haliaeetus leucocephalus*), golden eagles (*Aquila chrysaetos*), California condors (*Gymnogyps californianus*), and other avian scavengers. Deer offal piles are present and available to scavengers in autumn, and the degree of exposure depends upon incidence, abundance, and distribution of fragments per offal pile and carcass lost to wounding. In radiographs of selected portions of the remains of 38 deer supplied by cooperating, licensed hunters in 2002–2004, we found metal fragments broadly distributed along wound channels. Ninety-four percent of samples of deer killed with lead-based bullets contained fragments, and 90% of 20 offal piles showed fragments: 5 with 0–9 fragments, 5 with 10–100, 5 with 100–199, and 5 showing >200 fragments. In contrast, we counted a total of only 6 fragments in 4 whole deer killed with copper expanding bullets. These findings suggest a high potential for scavenger exposure to lead. (WILDLIFE SOCIETY BULLETIN 34(1):167–170; 2006)

Key words

bullet fragmentation, lead, lead poisoning, raptors, scavengers.

Avian predators and scavengers are susceptible to lead poisoning when they ingest pellets or fragments in the tissues of animals wounded or killed by lead-based bullets (Franson 1996, Locke and Thomas 1996, Wayland and Bollinger 1999). Toxic effects of ingested lead include neural degeneration, modification of kidney structure and bone, and inhibition of blood formation and nerve transmission (Eisler 1988, Kendall et al. 1996). Shotgun pellets experimentally fed to 5 bald eagles (*Haliaeetus leucocephalus*) killed 4 of them, and severe clinical signs prompted euthanization of the fifth (Hoffman et al. 1981, Pattee et al. 1981). Residual weights of recovered pellets showed that the 5 eagles dissolved (mobilized) totals of 19, 38, 42, 129, and 184 mg of lead, each less in mass than a single #4 pellet of 209 mg.

Harmata and Restani (1995) found lead in the blood of 97% of 37 bald eagles and 85% of 86 golden eagles (*Aquila chrysaetos*) captured as spring migrants in Montana during 1985–1993. Pattee et al. (1990) reported that 36% of 162 free-ranging golden eagles captured during 1985–1986 in southern California had been exposed to lead, and 9% had blood lead levels >0.6 ppm. Six of 9 dead or moribund eagles (*Haliaeetus* spp.) in Japan died of lead poisoning; 5 had lead bullet fragments in their stomachs, and evidence implicated hunter-killed deer as the primary vector (Iwata et al. 2000). Lead ingestion was a principal cause of recorded death in wild California condors (*Gymnogyps californianus*) prior to the mid-1980s when the population was brought into captivity (Wiemeyer et al. 1988), and in subsequently reintroduced, captive-bred condors tracked with radiotelemetry in Arizona (Cade et al. 2004). Kramer and Redig (1997) found a reduction in blood lead concentrations in bald and golden eagles after a 1987 ban on lead shot for waterfowl hunting in Minnesota

and Wisconsin; however, they found no change in prevalence of lead poisoning, a finding the authors attributed in part to offal piles from hunter-killed deer.

The availability of ungulate offal piles can be high in some regions. For example, the 10-year mean (1992–2001) of 676,739 white-tailed deer (*Odocoileus virginianus*) annually harvested by rifle hunters in Wisconsin would have produced an average density of about 5 offal piles per km² for the area of the entire state (Dhuey 2004). An unknown number of additional whole carcasses lost to wounding are present in the landscape during and after hunting seasons, possibly on the order of 10% or more (Nixon et al. 2001). The extent to which avian scavengers encounter lead in deer carrion is, therefore, not so much a question of carrion availability, but rather one of lead incidence, abundance, and distribution per offal pile or carcass. Our examination of these 3 factors using radiographic data strengthens the body of evidence that deer killed by rifle bullets are a potentially important pathway of lead contamination to scavenger food webs.

Methods

We obtained whole or partial remains of 38 deer (*Odocoileus virginianus* and *O. hemionus*) killed with standard, center-fire, breach-loading rifles by participating, licensed hunters engaged in normal hunting practices in Wyoming and California during 2002–2004. Thirty-four (89%) of the deer were killed by single shots to the thorax as determined by carcass examination and hunter interviews. The samples consisted of 15 offal piles discarded by hunters in the field, 10 deer carcasses in which tissues and viscera anterior to the diaphragm were left in place (abdominal viscera removed), 4 eviscerated carcasses, and 9 whole deer carcasses; the latter were eviscerated on polyethylene sheets to sequester offal for radiography.

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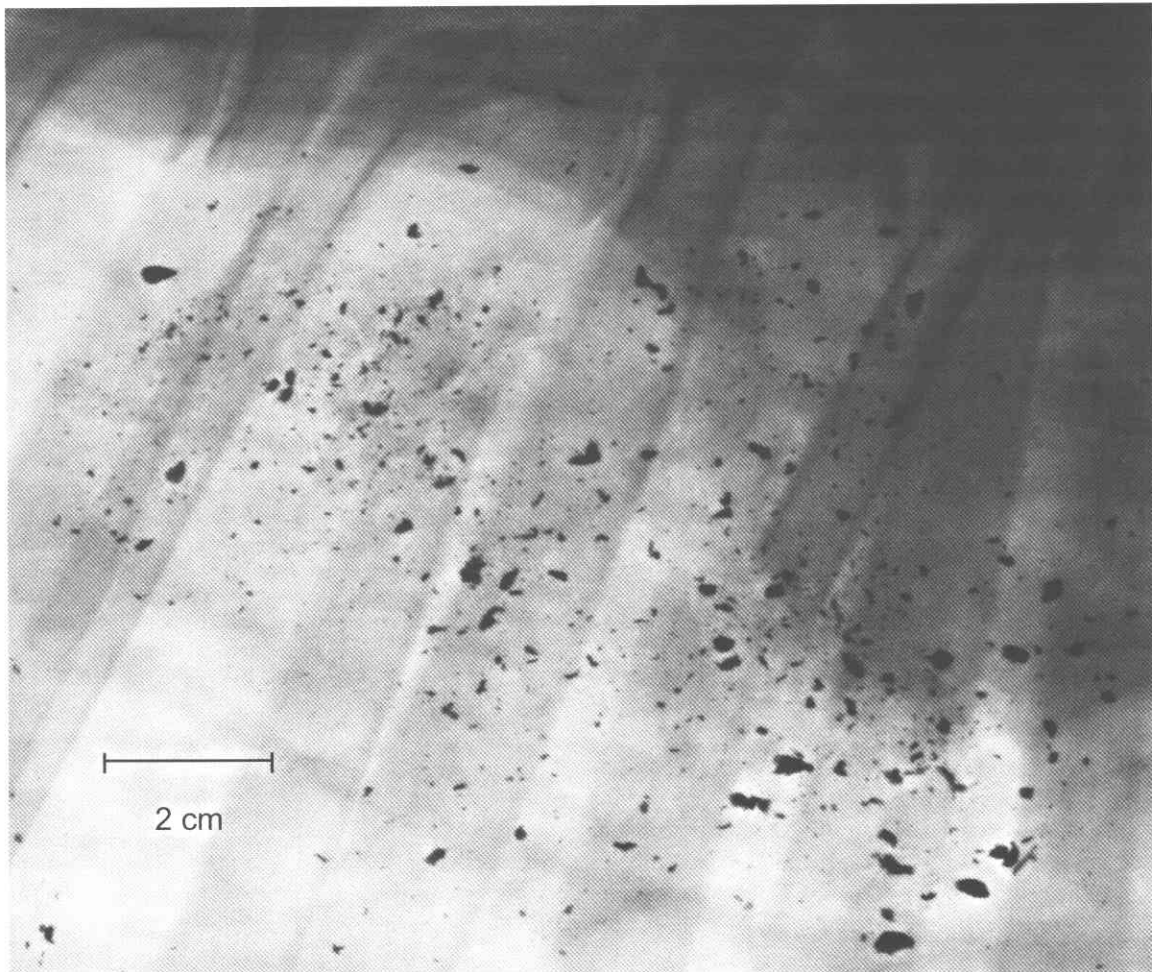


Figure 1. Lateral-view radiograph of the mid-thorax of an adult female white-tailed deer killed by a standard copper-jacketed, lead-core, soft-point hunting bullet in northern Wyoming in 2004. The fragment array surrounding the bullet path was approximately 12 cm in diameter, excluding outliers.

Hunters chose rifles, bullets, and bullet weights. Hunters used 7 standard deer rifle calibers, and the mean weight of 37 bullets was 145 grains (SD = 18, range 100–180). Thirty-four were standard copper-jacketed, lead core bullets, and 4 were monolithic copper expanding “X-bullets.” Seventeen of the former were of lead-tipped configuration (5 brands), 12 were polymer-tipped (5 brands), 2 were hollow points (1 brand), and 3 were of unrecorded structure. Shot distances varied from 37 to >200 m (mean of 12 ranged distances = 158 m, SD = 77).

Local veterinarians radiographed areas of bullet transition of all carcasses and offal dorsoventrally and laterally; and adjusted exposures to maximize contrast (e.g., 56–70 kvp, 100 mAS, 0.3 sec). We placed a 2.5-cm grid transparency on selected radiographs and, using a hand (reading) lens for clarity, we counted all unambiguous metal fragments (opaque to radiation) in each cell and summed the counts. We verified the presence of metal particles in one sample by dissection. We estimated the width of the fragment arrays (excluding outliers) in 5 samples by extrapolation from the width of a 9-mm-diameter carbon-fiber tube inserted through the wound channel and aligned perpendicular to the x-ray beam. We did not attempt to distinguish between copper and lead in fragment counts. Copper, which is less

frangible than lead, accounted for 30% of the mass in one standard (.308 caliber, 150-grain) hunting bullet we analyzed.

Results

Most radiographs showed a profusion of small (<2 mm) metal fragments broadly distributed along wound channels. In deer killed by lead-based bullets, radiographs showed fragments in 18 of 20 offal piles (range = 2–521 fragments, mean = 160, SD = 157). Five showed 0–9 fragments, 5 had 10–99, 5 had 100–199, and 5 showed > 200 fragments. We counted 416–783 fragments (mean = 551, SD = 139) in the 5 whole deer carcasses (Fig. 1), and 25–472 (mean = 213, SD = 172) in 10 carcasses containing thoracic organs but no abdominal viscera. Nine eviscerated carcasses showed fragments (range = 38–544, mean = 181, SD = 153). Fragment clusters in 5 samples radiated as far as 15 cm from wound channels; the average of 30 measurements of the most far-reaching clusters in 11 radiographs was 7 cm (SD = 3). Magnification of one sample of excised tissue showed that fragments ranged in size from a few of >5 mm to tiny ones beyond the limit of unaided vision, estimated to be about 0.5 mm. Copper bullets resisted fragmentation: we counted a total of only 6 fragments in 4 (whole) deer killed with these bullets, and only one in the offal piles (Table 1).