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ABSTRACT Lead is toxic to all vertebrate species and ingestion of spent lead pellets from hunting is the primary method of exposure in birds. Both acute and chronic effects occur in response to lead ingestion including death, weight loss, and reduced body function, but the effect is highly variable among species. Most research has focused on lead ingestion impacts on waterfowl, but less is known about the effects of lead ingestion by upland game such as ring-necked pheasants (*Phasianus colchicus*). We gavage-fed zero, one, or three lead pellets to 90 (n = 30/group) captive-raised adult hen pheasants and monitored survival and body mass. We documented no mortalities during the 21-day post-treatment period, and no significant change in body weight was detected among the treatments, although liver lead levels were comparable to diagnostic lead poisoning in waterfowl, mourning doves (*Zenaida macroura*), and chukars (*Alectoris chukar*). We also collected gizzards and livers from hunter-harvested wild pheasants, recorded the presence of ingested lead in the gizzard (n = 335*)* and determined wet weight lead concentration in livers (n = 45). Mean liver lead concentration increased with the number of lead pellets in the gizzard for both samples. Pheasants appear to be less susceptible to the acute effects of lead poisoning when compared to waterfowl, mourning doves, and chukars.

KEY WORDS acute toxicosis, lead, ingestion, *Phasianus colchicus*, ring-necked pheasant.

Lead is a non-specific toxin to all vertebrate species (Eisler 1988, Murray et al. 2004). Ingestion of lead pellets is the most common source of lead exposure in free ranging birds and has been documented in >130 avian species (Tranel and Kimmel 2009). Lead ingestion causes reduced body function resulting in anemia, weight loss, reduced reproductive parameters, brain function, and blood oxygen capacity, and changes in behavior which can decrease survival (Sanderson and Bellrose 1986, Kendall et al. 1996, Tranel and Kimmel 2007). Lead poisoning can be acute and cause death within days of exposure (Schulz et al. 2006) or chronic in which toxicosis symptoms persist during a prolonged period of elevated body lead level (Sanderson and Bellrose 1986, Gasparik et al. 2012).

The consequences of ingesting lead are not universal among bird species (Kendall et al. 1996, Friend and Franson 1999). Body mass of birds can be affected (Sanderson and Bellrose 1986), and may be amplified by external stressors such as temperature (Westermeier 1966, Buerger et al. 1986). Experimental lead ingestion studies have found ingestion of as few as one lead pellet reduces survival of mourning doves (*Zenaida macroura*; Buerger et al. 1986), ducks (Sanderson and Bellrose 1986, Castrale and Oster 1993), and chukars (*Alectoris chukar*; Bingham 2011), but higher doses are needed to cause death in quail and raptors (Kendall et al. 1996).

Lead pellet ingestion and acute fatal poisoning in ring-necked pheasants (*Phasianus colchicus*, hereafter pheasant) were reported as early as 1876 in Great Britain (Calvert 1876). Multiple studies have documented lead pellet ingestion by wild pheasants (Hunter and Rosen 1965, Butler et al. 2005, Runia and Solem 2016), but the acute and chronic effects are largely unknown. Limited research on pheasants suggests they are less susceptible to the acute effects of lead toxicosis (Gasparik et al. 2012). In a sample of 1,301 wild pheasants from throughout South Dakota, 24 had ingested lead and only two birds had ingested ≥3 pellets (Runia and Solem 2016). Our primary objectives were: 1) to estimate survival, change in body mass, liver lead concentration, and lead pellet retention in captive-raised adult hen pheasants 21 days after being gavage-fed zero, one, or three lead pellets; and 2) determine if liver lead concentrations of hunter-harvested male pheasants were influenced by the number of ingested lead pellets present at time of harvest.

STUDY AREA

We conducted the feeding-trial portion of this study in the poultry building of the South Dakota State Fairgrounds in Huron, South Dakota, USA. The enclosed brick building was 48 m by 23 m with a 10 m high ceiling and was not temperature controlled. The mean daily temperature was 11.8° C during a 14-day acclimation period and 4.3° C during the 21-day post-treatment period (National Weather Service Forecast Office 2014).

Our study area for acquiring hunter-harvested wild male pheasants was Lyman County, SD located in the Northwestern Great Plains Eco-region, specifically the Sub-humid Pierre Shale Plains of South Dakota, USA (Bryce et al. 1996). The 425,300 ha county was comprised of 25% cropland, 7% hay land, and 7% enrolled in the Conservation Reserve Program (Farm Service Agency 2015) with much of the remaining landscape dominated by grassland used for cattle grazing (National Agricultural Statistics Service 2016). Most of the cropland acres were winter wheat (45%), sorghum (18%), and corn (18%), with lesser amounts of sunflower

(8%), spring wheat (8%), and soybeans (3%). The remainder of the landscape was composed of small parcels of trees, road rights-of-way, surface water, municipalities, and rural residences.

METHODS

Captive Pheasant Feeding Trial

We conducted a 21-day dose-response experiment using captive-raised adult hen pheasants. We randomly assigned 30 pheasants to each of 2 treatment groups ($n = 60$) and a control group ($n = 30$). We fed treatment groups 1 or 3 lead pellets one time at the beginning of the experiment. We placed birds in individual cages (38 cm × 44 cm × 46 cm) within the enclosed building and provided them with unlimited amounts of commercial poultry food, water, and oyster shell grit. Nutritional content of the food was 16% crude protein, 0.70% lysine, 0.30% methionine, 2.50% crude fat, 8.0% crude fiber, 3.40−3.90% calcium, 0.45% phosphorus, 0.25−0.65% salt, and 0.15−0.23% sodium. After a 14-day acclimation period, we administered the treatments on 16 October 2013, and monitored survival during a 21-day post-treatment period. We gavage-fed one or three, #5 lead pellets by inserting a 6 mm outside diameter flexible tubing down each pheasant's esophagus and inserting the pellets down the tube into the crop. We replicated this process for birds in the control group without inserting the lead pellet(s).

We determined mass of birds at the beginning and end of the acclimation period and at the end of the 21-day post-treatment period to the nearest 5 g using a WeiHeng ® digital hanging scale (model 40KG, Guangzhou Weiheng Electronics Company, Guangzhou, Guangdong, China). We euthanized all birds by cervical dislocation and removed livers and gizzards from each bird at the end of the study. We stored livers individually in plastic freezer bags and kept them frozen prior to testing. We radiographed gizzards for the presence of lead pellets to determine pellet retention rate. We necropsied each gizzard to confirm the number of lead pellets observed in the radiograph. Livers from all 60 treatment birds and 10 control birds were tested for lead concentration by the University of Missouri-Columbia Veterinary Medical Diagnostic Laboratory, Columbia. We did not test all control birds for liver lead level because they were not exposed to lead and were not expected to have elevated liver lead levels.

We reported summary statistics for liver lead parts per million (ppm) in relation to the number of pellets in the gizzard at the end of the experiment. We used a paired *t*-test to compare bird weight between the beginning and end of the acclimation period. Because some birds did not retain the full lead pellet treatment, we analyzed the experiment data in two ways. First, we tested for a difference in % body weight change among the 2 treatment groups and control group for only birds that retained the full treatment. Secondly, we compared % body weight change between the control group and all birds that retained ≥1 lead pellet. We used analysis of covariance for both tests and included bird weight at the beginning of the experiment as a covariate (Huitema 1980). We used R version 3.1.3 (R Core Team, 2015) for all analyses. Animal care guidelines as outlined in Fair et al. (2010) were followed.

Wild Pheasant Collections

We collected gizzards and livers from hunter-harvested wild pheasants from 15 October 2011 – 31 December 2011 from a commercial hunting operation. We radiographed gizzards to determine the presence of metal pellets. We necropsied gizzards that contained pellets to determine if pellets were ingested or lodged, and if pellets were lead or non-toxic (Bingham et al. 2015). We tested livers for lead concentration in the same manner as the captive experiment samples. Only livers without obvious shot damage were submitted to prevent detection of contamination from lead pellet residue. We reported summary statistics for liver lead ppm in relation to number of pellets in the gizzard at time of harvest.

RESULTS

Captive Pheasant Experiment

Two birds from the control group and two birds from the treatment group fed one pellet were excluded from the analysis due to injuries sustained from escaping their cage. All birds survived the duration of the 21-day experiment. Of the 118 gavage-fed lead pellets, 62 (52%; 95% CI, $44 \leq \bar{x} \leq 61\%$) were retained through the end of the study. Twenty-two of the 58 birds administered lead pellets retained ≥1 lead pellet(s) during the 21-day experiment. For the 28 birds administered one pellet, 15 retained one pellet and 13 retained zero pellets. For the 30 birds administered three pellets, 10 retained the full treatment, six retained two pellets, five retained one pellet, and nine did not retain any pellets.

The control bird livers ($n = 10$) did not have detectable amounts of lead. Mean observed liver lead level increased as the number of pellets retained during the experiment increased (Table 1). Six of the 22 birds that retained zero pellets had liver lead levels ≥0.53 ppm (range 0.53–4.06), while remaining birds did not have detectable levels. Three birds that were administered and retained one pellet did not have detectable lead levels in the liver, while only six of the 36 birds that retained ≥1 pellet had liver lead levels ≤0.96 ppm. The highest liver lead level we observed during our pen trial was 7.18 ppm. The highest liver lead level we observed was in a hunter-harvested male pheasant which had a liver lead ppm of 24.61 and had nine ingested lead pellets in its gizzard.

Mean body weight of birds increased ($t_{85} = -15.85$, $P \leq 0.001$) during the 14-day acclimation period from 785.3 ±

Table 1. Summary statistics of pheasant liver lead concentration (parts per million wet weight [ppm]) by number of lead pellets in gizzard at time of death, South Dakota, USA, 2011−2013. Feeding trial pheasants were females and hunter-harvested pheasants were males.

Source[a]	Pellets[b]	*n*	Mean Pb ppm	SE	Range
Feeding trial	0	22	0.38	0.19	0.00−4.06
Feeding trial	1	20	1.39	0.22	0.00−3.58
Feeding trial	2	6	2.35	0.13	1.87−2.86
Feeding trial	3	10	3.90	0.63	1.20−7.18
Hunter-harvest	0	33	0.22	0.09	0.00−2.86
Hunter-harvest	1	6	1.32	0.49	0.06−2.68
Hunter-harvest	2	4	2.48	0.48	1.38−3.58
Hunter-harvest[c]	3	1	6.95		
Hunter-harvest[c]	9	1	24.61		

[a] All 10 birds from the control group for the feeding trial did not have detectable levels of lead in the liver.
[b] Lead pellets retained in gizzard after 21-day post-treatment period for feeding trial and number of lead pellets in gizzard at time of harvest for hunter-harvested birds.
[c]Blank cells represent no data.

8.5 g to 860.1 ± 8.3 g. Percent change in body weight during the 21-day post-treatment period did not differ among the control group and groups of birds that retained the full treatment ($F_{2,49}$ = 0.099, P = 0.906). The weight at beginning of the experiment was not a significant covariate as an additive ($F_{1,49}$ = 1.143, P = 0.290) or interaction ($F_{2,47}$ = 1.278, P = 0.288) term. Percent change in body weight during the 21-day post-treatment period did not differ between the control group and birds that retained ≥ 1 pellet ($F_{1,61}$ = 0.223, P = 0.639). The weight at beginning of the experiment was not a significant covariate as an additive ($F_{1,61}$ = 0.599, P = 0.442) or interaction ($F_{1,60}$ = 2.341, P = 0.131) term. All birds combined averaged 4.3 ± 5.9% body weight change during the 21-day post-treatment period.

Wild Pheasant Collections

We collected 336 gizzards and livers from hunter-harvested pheasants, 12 of which had ingested lead in the gizzard. We tested for liver lead levels for all birds that had ingested lead and an additional 33 livers in which no ingested lead was found. Mean observed liver lead ppm increased as the number of lead pellets in the gizzard at time of harvest increased (Table 1). One hunter-harvested bird with zero lead pellets in the gizzard had a liver lead liver level of 2.86 ppm while all others had liver lead levels of ≤0.47 ppm. Excluding the bird with the elevated lead level, the mean liver lead ppm was reduced to 0.14 ± 0.03 for birds without ingested lead. Nine of the 12 birds with ingested lead had liver lead levels ≥1.38 ppm, while three had levels ≤0.60 ppm.

DISCUSSION

Unlike most other experimental lead ingestion studies, we did not observe any mortality or weight loss during our 21-day post-treatment period, although liver lead levels consistent with diagnostic lead poisoning were observed (Friend 1985, Franson et al. 2009). A similar study found no mortality or weight loss when captive pheasants were gavage-fed two, four, or six lead pellets weekly for 10 weeks. Waterfowl exhibit weight loss and reduced survival for at least 3 weeks after ingestion of 1−2 lead pellets (Jordan and Bellrose 1950, Sanderson and Bellrose 1986). Ingestion of a single pellet can cause weight loss and death in captive chukars within 15 days (Bingham 2011). Buerger et al. (1986) found 24% mortality within 11 days for captive mourning doves dosed with one gavage-fed lead pellet. Similarly, Schulz et al. (2006) observed 50% survival of a group of captive mourning doves with two or fewer gavage-fed pellets during a 21-day experiment.

Our captive-raised hen pheasants ($\bar{x}$ = 860 g) were larger than mourning doves (112–127 g; Schulz et al. 2006) or chukars (~200–500 g; Bingham 2011). Hazard of death decreased 2.4–3.9% for every 1 g increase in pretreatment body

weight for mourning doves, which suggests the larger size of pheasants may have reduced the effects of acute lead toxicosis when administered similar lead doses (Schulz et al. 2006). But northern bobwhite (*Colinus virginianus*), which are similar in size to chukars, tolerated doses of five lead pellets per week for four weeks without mortality (Damron and Wilson 1975). Geese are generally able to survive low dosages of lead (1−2 pellets) while ducks, which are smaller, generally die within several weeks (Sanderson and Bellrose 1986).

As expected, liver lead levels at the end of our 21-day post treatment period increased with the number of pellets retained. The mean liver lead concentration for pheasants that retained two pellets was similar (2.98 ppm) to pheasants given two pellets per week for 10 weeks (Gasparik et al. 2012). An increase in mean liver lead concentration as the number of pellets administered increased also was noted by Gasparik et al. (2012). For pheasants that retained all three pellets they were given, mean liver lead concentration (3.90 ppm) was at a level consistent with subclinical poisoning in waterfowl, although some individual samples had levels ≥6 ppm which is indicative of clinical poisoning (Beyer et al. 1996). Lead-dosed mourning doves that survived a 21-day treatment period had liver lead levels of 3.44 ppm while mourning doves that died during the treatment period had a mean level of 49.20 ppm (Schulz et al. 2006). Buerger et al. (1986) found mourning doves that died after being gavage-fed a single lead pellet had liver lead levels of 23–238 ppm, a level much higher than in our pheasants that retained one pellet.

Either death or weight loss is a common result of acute lead toxicosis; however, experimental studies have not documented these results in pheasants. Our captive pheasant study suggests that pheasants are less susceptible to the acute effects of lead poisoning than mourning doves, chukars, or waterfowl. It is difficult to determine the overall effect of lead poisoning from ingested lead on the wild pheasant population. Although results of our captive experiment did not reveal any mortality from the ingestion of 1−3 pellet(s), the most common number of pellets found in hunter-harvested birds, detrimental effects could still impact wild birds. For instance, multiple studies have shown that the effects of acute lead toxicosis can be more severe when birds are exposed to cold temperatures. For a dose-response study conducted on mourning doves outdoors, 90% of the mortalities occurred when temperatures dropped to 0° C (Buerger et al. 1986). Another outdoor experiment found mortality rates were 5.5 times higher for mourning doves dosed during the winter compared to those dosed during the summer (McConnell 1968). This suggests that results of captive studies conducted in controlled settings may not fully reveal the expected effects on wild birds, although our study was conducted in a non-temperature controlled building. Because exposure to lead affects the nervous system, lead poisoning could cause behavioral changes in birds, making them more susceptible to predation even if direct mortality from acute toxicosis does not occur (Bellrose 1955). Future studies should evaluate the consequences of ingesting lead by experimentally dosing wild birds and monitoring survival and reproduction in natural habitats.

Our pen trial supported a threshold level between background and elevated liver lead level between 0.53–0.96 ppm, which is similar to the results for our wild bird sample and the 0.70–1.15 ppm level suggested by Bingham (2015) for chukars in Utah. Similar to Bingham et al. (2015), liver lead concentrations from our sample of hunter-harvested pheasants exhibited a clear distinction between background and elevated levels. Our samples from wild birds without ingested lead suggest 0.47 ppm liver lead level as the upper limit for background lead exposure. Most (9 of 12) of the birds harvested with lead in the gizzard had liver lead levels of ≥1.38 ppm, which suggests a threshold level of 0.47–1.38 ppm for elevated lead exposure from our samples of wild birds. Threshold levels are relative measures because background concentrations can vary among species and populations (Pain et al. 2009). Pheasants in South Dakota and chukars in Utah appear to have threshold levels lower than the most commonly used threshold of 2 ppm (Friend 1985) developed for waterfowl.

Both our study and Bingham et al. (2015) documented hunter-harvested birds with ingested lead pellets without elevated liver lead levels and vice versa. This suggests a delay may occur between lead ingestion and elevated liver lead levels, and elevated liver lead levels may persist after ingested lead pellets are voided or fully eroded. Our pen study and Schulz et al. (2007) documented elevated liver lead concentration in birds which had recently been exposed to ingested lead, although no pellets were present at time of necropsy. Interestingly, three birds in our captive study retained a single lead pellet for the entire 21-day post-treatment period, yet did not have detectable levels of lead in the liver. Analyzing both livers for lead concentration and gizzards for ingested lead in combination is expected to reduce bias when estimating overall lead exposure from bird samples.

MANAGEMENT IMPLICATIONS

Until the consequences of lead ingestion are determined for free ranging pheasants in natural habitats, the potential impacts to wild populations will be difficult to estimate. Lead deposition from pheasant hunting may be more of a concern for other sympatric species that are more susceptible to the acute effects of lead poisoning. Pheasant hunting occurs in a wide variety of upland and wetland habitats and could impact species vulnerable to lead poisoning such as waterfowl. Wildlife managers and policy makers should consider the risks of lead poisoning to all birds when making management decisions related to the use of lead shot for hunting.

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