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Captive Ring-necked Pheasant Response to Very High Experimental Doses of Lead

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ABSTRACT Ingestion of spent lead pellets is a well-documented source of lead exposure in free-ranging birds, although the consequence of lead ingestion varies widely among avian guilds. Ring-necked pheasants (*Phasianus colchicus*) appear to be less susceptible to lead poisoning than other game birds. Our objectives were to determine survival, liver lead accumulation, and body mass change of 129 captive-raised pheasants in response to being gavage-fed 5, 10, 20, or 40 lead pellets. All pheasants survived the 21-day experiment. Liver-lead levels were positively correlated with the number of lead pellets retained and negatively correlated with beginning body mass. Change in percent body mass varied by sex and liver-lead concentration. Higher liver-lead levels were associated with higher percent mass loss for males but not females. Our experiment coincided with the breeding season, which may have contributed to the sex-specific responses. Our pheasants survived lead doses and liver-lead accumulation levels associated with acute lead toxicosis and death for a variety of avian guilds.

KEY WORDS acute toxicosis, lead, lead poisoning, *Phasianus colchicus*, ring-necked pheasant, South Dakota

Exposure to lead adversely affects wildlife, and ingestion of spent pellets or bullet fragments is the primary source of lead exposure in free-ranging birds (Pokras and Kneeland 2009, Tranel and Kimmel 2009). Lead ingestion causes reduced body function resulting in anemia, loss in body mass, reduced reproductive parameters, suppressed brain function, lowered blood oxygen capacity, and changes in behavior which can decrease survival (Sanderson and Bellrose 1986, Kendall et al. 1996, Tranel and Kimmel 2009). Although ingested lead has been documented in >130 avian species (Tranel and Kimmel 2009), the risk profile for a particular species depends on a combination of the individual response to lead exposure and overall population exposure rate. 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 severities of lead exposure effects can be influenced by diet (Damron and Wilson 1975, Sanderson and Bellrose 1986), made more severe by stressors such as changes in temperature (Kendall and Scalon 1984), and be less severe in captive-raised versus wild individuals (Jordan and Bellrose 1950).

Among game birds, the issue of lead poisoning has been particularly problematic for waterfowl because hunting deposited concentrated amounts of lead in high-use areas and the individual effects of lead poisoning were severe. Prior to the 1987–1991 phased-in ban on lead ammunition for waterfowl hunting, an estimated 1.6–2.4 million waterfowl died annually from lead poisoning (Friend and Franson 1999). Additionally, an estimated 1.66 million mourning doves (*Zenaida macroura*) may die annually from ingesting lead pellets (Plautz et al. 2011). Both waterfowl and mourning doves are highly susceptible to acute lead toxicosis, which causes reduced survival after ingestion of as few as 1–3 pellets (Jordan and Bellrose 1950, Schulz et al. 2006).

Reported mortality from lead exposure is more common in waterfowl than resident upland game birds (Friend and Franson 1999), and gallinaceous birds in particular seem less susceptible to lead poisoning than most other birds (Franson 1996, Friend and Pain 2011). Nonetheless, isolated cases of acute fatal lead poisoning from ingesting shot have been documented in ring-necked pheasants (*Phasianus colchicus*, hereafter pheasant) (Calvert 1876, Hunter and Rosen 1965). Ingested lead pellets were found in 34% of a small sample of captive-raised pheasants from a shooting preserve in Canada (Kreager et al. 2008). In a large sample of hunter-harvested wild pheasants from throughout South Dakota excluding shooting preserves, only 0.8% had ingested lead shot (Runia and Solem 2016). In the same study, the prevalence rate of ingested lead was 3.9% for pheasants harvested on shooting preserves where heavy lead deposition occurred. In pheasants that ingested lead (≥1 pellet), the mean number of ingested lead pellets was 2.40 (range: 1–11) for non-preserve areas and 2.65 (range: 1–13) for preserve sites. In a mixture of wild and released pheasants harvested on shooting preserves in Great Britain, 3% contained ingested lead shot (Butler et al. 2005).

Pheasants ingest lead shot in the wild, but the individual effects of lead exposure have not been adequately investigated in this species. No mortality or significant mass loss was observed when captive female pheasants were gavage-fed 2, 4, or 6 lead pellets weekly for 10 weeks (Gasparik et al 2012). However, egg mass, fertilization, and hatchability were lower

for treatment groups than the control groups (Gasparik et al. 2012). In a 21-day experiment, Runia and Solem (2017) did not detect mass loss or reduced survival when captive female pheasants were gavage-fed a one-time dose of 1 or 3 lead pellets even though liver-lead levels reached a concentration consistent with lead poisoning in other birds.

Lethal dose measures are often used as a way of comparing relative susceptibility of lead poisoning among bird species or guilds. Because the lethal dose of lead for pheasants remains unknown, we expanded upon past pheasant dose-response studies by increasing the experimental dose of lead to much higher levels. Our objectives were to estimate survival, liver-lead concentration, and change in body mass of captive-raised adult pheasants after being gavage-fed 0, 5, 10, 20, or 40 lead pellets.

STUDY AREA

We studied captive pheasants within the poultry building of the South Dakota State Fairgrounds in Huron, South Dakota, USA (44.3633° N, 98.2143° W). 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 7.1° C during the 47-day acclimation period and 11.4° C during the 21-day post-treatment period (NOAA 2016).

METHODS

We conducted a dose-response feeding trial experiment using captive-raised adult pheasants hatched from captive stock. We randomly assigned 129 pheasants (57 females and 72 males) to each of four treatment groups (n = 119) and a control group (n = 10) (Table 1). After a 47-day acclimation period, we administered the treatments on 26 April 2016 and monitored survival during a 21-day post-treatment period. We used #5 lead shot size as it is one of the more popular shot sizes used for pheasant hunting. The shot was obtained from a commercial shotshell source; therefore, it was assumed there was very little variability in weight/size from pellet to pellet and we did not use any pellets that were obviously damaged or malformed. We gavage-fed 5, 10, 20, or 40, #5 lead pellets one time by inserting a flexible tube (6-mm outside diameter) down each pheasant's esophagus and inserting the pellets into the crop. We replicated this process for birds in the control group without inserting lead pellets. We placed the birds in individual cages (38 cm × 44 cm × 46 cm) within the enclosed building and provided them with unlimited commercial poultry food, water, and gravel grit. Nutritional content of the food was 16.00% crude protein, 0.70% lysine, 0.30% methionine, 2.50% crude fat, 8.00% crude fiber, 3.40−3.90% calcium, 0.45% phosphorus, 0.25−0.65% salt, and 0.15−0.23% sodium.

We weighed 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 21-day post-treatment period. We stored individual livers in plastic freezer bags and kept them frozen prior to testing. We radiographed and necropsied each gizzard to confirm the presence and number of lead pellets. Each liver was tested for lead concentration (parts per million wet weight [ppm]) by atomic absorption spectroscopy by the University of Missouri-Columbia Veterinary Medical Diagnostic Laboratory, Columbia, USA. Animal care guidelines as outlined in Fair et al. (2010) were followed.

Table 1. Number of #5 lead pellets gavage-fed to captive-raised pheasants by treatment group and sex in South Dakota, 2016.

Treatment Group	No. lead pellets	No. females	No. males
Control	0	5	5
Low	5	11	17
Medium	10	13	16
High	20	14	17
Very High	40	14	17

Statistical Analysis

Although we administered lead pellets at specific doses (treatment groups), we expected a retention rate of <100% and anticipated a range of lead exposure among all birds. We tested for a difference in pellet retention rate among treatment groups using analysis of variance (ANOVA). Because the pellet retention rate was similar among groups and there was a range of lead exposure among all birds, we assumed overall lead exposure was best described by the number of lead pellets retained by each bird, not the treatment group. We used linear regression to model post-treatment liver-lead concentration as a function of pre-treatment mass, sex, and lead pellets retained. Data from the control group was not used in the regression models predicting liver-lead concentration. For the acclimation period, we tested for a difference in body mass change among treatment groups using ANOVA and between sexes using a *t*-test. We used linear regression to model post-treatment percent change in body mass as a function of pre-treatment mass, sex, lead pellets retained to end of experiment, and liver-lead concentration.

We followed information-theoretic methods to evaluate our candidate model sets for the most parsimonious model(s) (Burnham and Anderson 2002, Arnold 2010). We inspected our model set for uninformative variables by identifying nested models where the addition of one parameter only improved model fit by trivial amounts of deviance (e.g., 1–2). We also evaluated parameter estimate 85% confidence limits (CLs) relative to zero (Arnold 2010). We report estimates and 85% CLs for the most parsimonious model while holding all other continuous variables at their mean. We used the program R (R Version 3.1.3, www.r-project.org, accessed 9 March 2015) for all statistical analyses.

RESULTS

All pheasants survived the acclimation and post-treatment periods. Of the 2,290 gavage-fed pellets, 873 (38%, range: 0–35/bird) were retained to the end of the experiment. Pellet retention rate did not vary by treatment group ($F_{3,118} = 0.651$, $P = 0.584$). Of the 119 pheasants that were administered lead pellets, 112 (94%) retained ≥1 lead pellet. Mass increased 3.57% (SE = 0.99, $n = 129$) during the acclimation period and did not differ between sexes ($F_{1,127} = 1.575$, $P = 0.212$) or among groups ($F_{4,124} = 0.398$, $P = 0.810$).

Our top-ranked model for predicting liver-lead concentration included mass at beginning of treatment period and number of pellets retained through the post-treatment period (Table 2). The second-ranked model was identical to the top-ranked model except for the addition of the variable for sex, which had 85% CLs that overlapped zero. Because the second ranked model contained an uninformative variable, we considered it to be uncompetitive with the top model. Additionally, no other models were within 4 ΔAIC_c of the top model, so we only used the top-ranked model for parameter estimation. Liver-lead concentration was positively correlated with the number of pellets retained through the treatment period and negatively correlated with beginning pheasant mass (Fig. 1).

Our top-ranked model for body mass change during the post-treatment period included liver-lead concentration, sex, and an interaction between liver-lead concentration and sex (Table 2). The second-ranked model was identical to the top-ranked model except beginning mass was included. Because beginning mass was not informative (85% CL overlapped zero), the second-ranked model was not considered competitive. The third-ranked model differed from the top model by the variables mass and an interaction between mass and sex, both of which were uninformative variables (85% CLs overlapped zero). Therefore, we selected the top-ranked model as most parsimonious. Body mass loss was highly correlated with increasing amounts of liver-lead concentration for males but had very little influence for females (Fig. 2). Females lost more body mass than males during the post-treatment period when liver-lead concentration was ≤15 ppm.

DISCUSSION

Gallinaceous birds are known to be resistant to the effects of lead toxicosis, and pheasants appear the least affected within the Galliformes. Our pheasants survived the largest single experimental dose of lead administered to the species to date and one of the largest doses administered to any species. We are only aware of one study that administered a larger one-time dose to any species. Cook and Trainer (1966) experimentally fed 25–100 lead pellets to Canada geese (*Branta canadensis*); all died in ≤10 days. Pheasants also survived the more chronic but still large dose by Gasparik et al. (2012) (2–6 pellets weekly for 10 weeks) and the lower dose of 1–3 pellets by Runia and Solem (2017). Northern bobwhites (*Colinus virginianus*) exhibited 95% survival to 56 days after being gavage-fed up to 3 spent lead pellets (Tannenbaum 2014). Most (90%) northern bobwhites survived a 5-lead pellet/week dose for six weeks, but mortality was 92% when the dose was increased to 10 lead pellets three times per week for four weeks (Damron and Wilson 1975). In the same study, mortality was over 67% when the dose was 10 lead pellets per week for four weeks. In willow ptarmigan (*Lagopus lagopus*), an experimental dose of three or six lead pellets caused increased mortality (Fimreite 1984).

Comparatively, a single lead pellet causes reduced survival in captive chukars (*Alectoris chukar*; Bingham 2011), mallards (*Anas platyrhynchos*; Jordan and Bellrose 1950), and mourning doves (Buerger et al. 1986). Wetmore (1919) found six pellets were always lethal to captive mallards while Jordan and Bellrose (1950) observed 60–70%

Table 2. Linear regression model selection results for predicting liver-lead concentration (parts per million wet weight [ppm]) and body-mass change (%) of lead-dosed captive-raised pheasants after a 21-day post-treatment period, South Dakota, 2016. Only models with differences in Akaike's Information Criterion corrected for small sample sizes (ΔAIC_c) ≤4.0 of the first-ranked model are presented.

Model[a]	AIC_c	ΔAIC_c	K[b]	ωi[c]
Liver lead concentration (ppm) models				
Pellets + mass	674.9	0.0	2	0.41
Pellets + mass + sex	675.8	0.9	3	0.26
Pellets × sex + mass	676.3	1.4	4	0.21
Body mass change (%) models				
Lead ppm × sex	860.1	0.0	3	0.46
Lead ppm × sex + mass	861.2	1.1	4	0.26
Lead ppm × sex + mass × sex	863.4	3.3	5	0.09

[a] Predictor variables included in each candidate model. Lead ppm–liver-lead concentration in parts per million at end of post-treatment period, sex–male or female, mass–pheasant mass (g) on day of treatment, pellets–number of lead pellets in gizzard after 21-day experiment.
[b] Number of parameters used in each candidate model.
[c] Akaike model weight.

mortality for wild mallards after ingestion of a single lead pellet. Nearly all captive mallards died after a dose of eight lead pellets (Irby et al. 1967). Ingestion of as few as two lead pellets caused 50% mortality (20 days) in mourning doves (Schulz et al. 2006).

As expected, at the conclusion of the experiment, liver-lead concentration levels were positively correlated with the number of lead pellets remaining in the gizzard. Varying liver-lead concentration interpretations have been suggested for poisoning thresholds. Our liver-lead level model predictions for birds with 0 pellets retained were still about 6 ppm, which is the lower suggested threshold for clinical poisoning in Galliformes (Franson 1996). For birds that retained ≥21 pellets, the predicted liver-lead level (15 ppm) was consistent with severe clinical poisoning and death in Galliformes (Franson1996). We are unaware of suggested liver-lead toxicity thresholds specific to pheasants, but Friend and Franson (1999) suggested 5 ppm was toxic and 16 ppm was fatal for grouse. Our highest observed liver-lead level was 30 ppm for a female pheasant that retained 35 lead pellets, and in previous work we observed a hunter-harvested male pheasant with nine ingested lead pellets and a liver-lead level of 25 ppm (Runia and Solem 2017).

Based on this study, Gasparik et al. (2012), and Runia and Solem (2017), accumulation of lead in the liver of pheasants exhibits an approximate linear relationship (~1–2 ppm liver-lead level per pellet administered/retained). However, lead accumulation has been shown to be highly variable in other species, which might explain why lower survival has been observed in other species given similar lead doses as pheasants. With mourning doves administered one, two, or four lead pellets, liver-lead levels were 1.0, 6.8, and 1.5 ppm respectively for birds surviving to 35 days, but 26.8, 29.8, and 31.1 ppm respectively for birds that died during the 35-day experiment (Buerger et al. 1986). Liver-lead levels ranged from 32 to 83 ppm for captive mallards that died after ingesting six lead pellets (Longcore et al. 1974), whereas the predicted liver-lead ppm for our pheasants that retained six pellets to day 21 of the experiment was only 7.7 ppm (85% CI = 7.2–8.3).

Although liver-lead accumulation did not vary by sex, trends in body-mass loss were influenced by sex and liver-lead levels. Our experiment occurred during the breeding season and we routinely observed our roosters cackling and

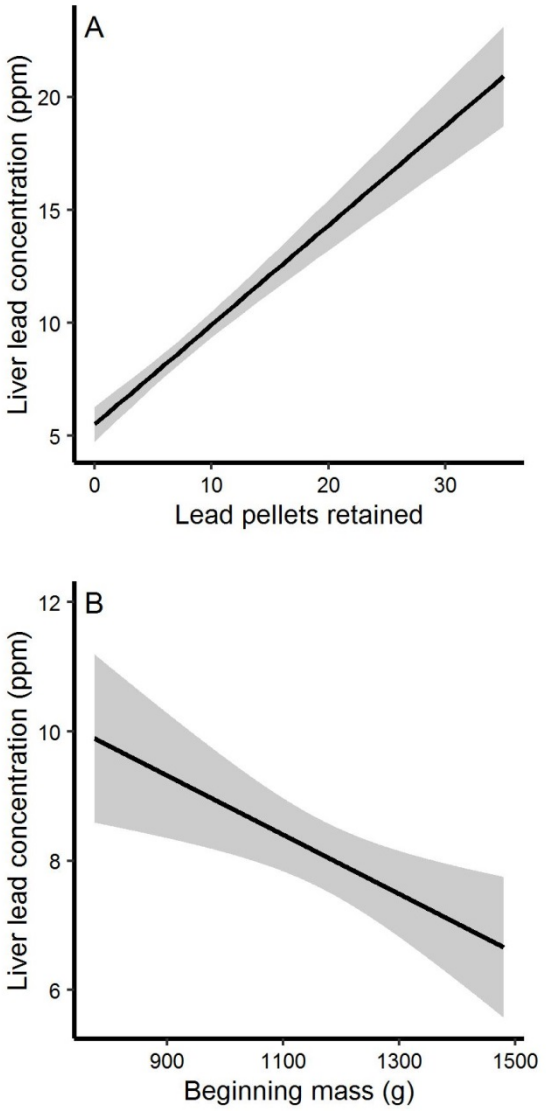

Figure 1. Predicted liver-lead concentration (parts per million wet weight [ppm]) of lead-dosed captive-raised pheasants in response to (A) lead pellets retained and (B) beginning body mass (g) after a 21-day experiment in South Dakota, 2016. Shaded areas represent 85% confidence intervals. All other continuous variables were set to their observed mean.

carrying out territorial wing-flapping behavior. Our females were actively laying eggs, but the facility was not designed to contain eggs within individual cages, so we were not able to estimate egg production for individual birds. Similar mass loss in both male and female wild pheasants has been observed during the breeding season (Edwards et al. 1964). When liver-lead concentration was low, females lost more body mass than males, which suggests the benchmark reproductive energy demand was higher for females than males. Our captive males may have lost less body mass when compared to females because some of the reproductive energy demand in wild males is behavioral in nature (gather harem, protect territories) and thus there is less energy expended in a captive setting.

Body mass loss of approximately 20–40% has been associated with chronic effects of lead exposure in Canada geese (Sileo et al. 1973), ducks (Irwin 1977, Beyer et al. 1988), northern bobwhite (Damron and Wilson 1975, Beyer et al. 1988), and chukars (Bingham 2011). However, lead exposure has not been linked to significant body mass loss in captive female pheasants (Gasparik et al. 2012, Runia and Solem 2017). We have no obvious explanation to why our female

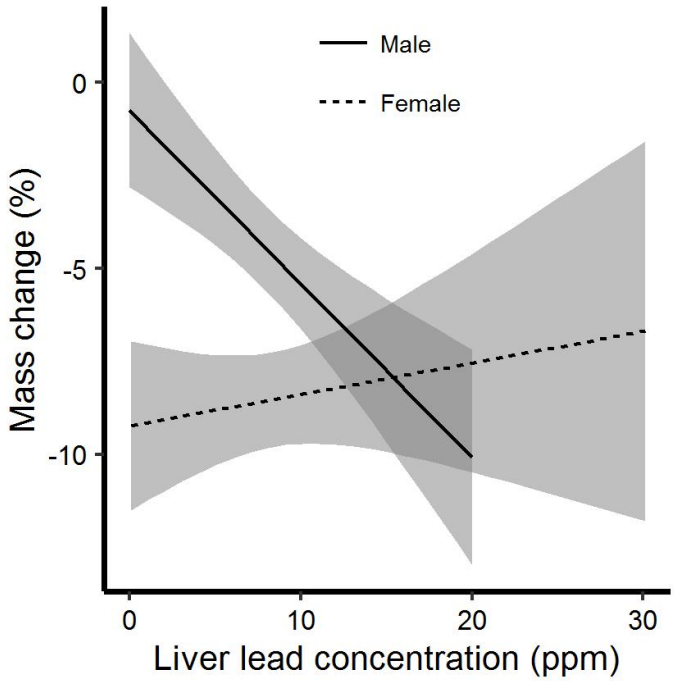

Figure 2. Predicted mass change (% of body mass) of lead-dosed, captive-raised pheasants in response to observed liver-lead concentration (parts per million wet weight [ppm]) after a 21-day experiment in South Dakota, 2016. Shaded areas represent 85% confidence intervals.

pheasants did not lose body mass as liver-lead concentration increased while males did. However, sex-specific responses to lead accumulation have been noted with inconsistent responses. In northern bobwhite, Kerr et al. (2011) found feed consumption, body mass gain, packed cell volume, and plasma protein concentration were adversely affected in lead-dosed males but not females. In domestic chickens (*Gallus gallus domesticus*), Mazliah et al. (1989) found no body mass loss in lead-dosed females, which laid more eggs than controls. However, lead-dosed female Japanese quail (*Coturnix japonica*) lost 21% of their body mass whereas that of males was stable (Edens et al. 1976). Our study is yet another example of the high variability in response to lead exposure between sexes as males lost body mass with increasing liver-lead ppm whereas females did not.

A higher resistance to lead poisoning during the breeding season by females has been demonstrated in mallards and is thought to be related to a high metabolic rate and mobilization of energy sources for egg laying (Finley and Dieter 1978). Spring is also the only season in which food intake is higher for female ducks than male ducks (Jordan and Bellrose 1950).

Diet is probably the single most important factor that influences lead absorption into the body and toxicity once absorbed. This makes it difficult to predict the consequences of lead ingestion in wild birds based on captive studies or directly compare results of captive studies when the food provided was not similar. Diets high in protein and calcium have been found to consistently reduce the negative impacts of lead exposure (Sanderson and Bellrose 1986). Diets high in carbohydrates such as grain and a variety of weed seeds have been associated with increased symptoms of lead toxicosis (Sanderson and Bellrose 1986). Among captive bird experiments evaluating lead poisoning, feed and available grit has been highly variable. Of the lead-poisoning pheasant studies, Gasparik et al. (2012) did not report the specific feed or grit, Runia and Solem (2017) fed high-protein poultry food and oyster shell grit, and this study fed high-protein commercial poultry food and gravel as grit. It is possible that the high-protein feed in our study mitigated lead absorption and the deleterious impacts of lead poisoning.

Northern bobwhite have shown resilience to the effects of lead poisoning when on a primarily corn/soybean meal diet (Damron and Wilson 1975) and seed-based diet (Tannenbaum 2014), but both diets included limestone or calcium supplements. In captive chukars, a dose of one or five lead pellets never caused mortality in birds on commercial feed, but five out of 16 died when on a mixed seed diet (Bingham 2011). Waste grain, especially corn and wheat, constitutes ≥70% of wild pheasants' diet (Trautman

1982). Corn is only 9% protein (USDA 2018) compared to 16% in the commercial feed fed to our birds. Calcium intake further confounds the potential differences in the effects of lead poisoning between wild and captive pheasants. In wild pheasants, calcium is consumed through calcareous grit or crustaceans (Trautman 1982). About 20% of a pheasant's spring diet is mineral matter and animal matter, both of which contain calcium, but the exact percentage of calcium in the diet is difficult to quantify and compare to commercial poultry feed (Trautman 1982). Calcium intake peaks in spring particularly in females that need increased calcium for egg production (Trautman 1982). Future research should investigate pheasant response to lead exposure when provided food that would resemble the season- and sex-specific diets of wild birds.

MANAGEMENT IMPLICATIONS

Translating results of captive studies to wild situations is challenging, but pheasants appear to be less susceptible to lead poisoning than other birds. Our pheasants survived and exhibited minimal body-mass loss in response to lead doses far higher than observed in wild pheasants. Dosing wild birds with lead and estimating survival and reproduction may ultimately be the most reliable way of estimating the effects of lead exposure on wild pheasants. However, pheasants are a widespread, popular game bird and shooting occurs in a variety of habitats, including wetlands. Lead deposition from pheasant hunting and its possible impacts to other susceptible birds such as waterfowl may be a more relevant management consideration than the direct impacts to pheasants.

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