National Quail Symposium Proceedings

Volume 3 Article 11

1993

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SURVIVAL OF NORTHERN BOBWHITE INFECTED WITH AVIAN POX

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Abstract: Avian pox is an enzootic disease among northern bobwhite (*Colinus virginianus*) in the southeastern United States, but occasionally it occurs as local or regional epizootics. Little information exists concerning survival of wild bobwhite infected with this disease. During the winters of 1985 and 1986, we compared survival of radio-tagged bobwhite with and without pox lesions. Pox lesions were considered "wet" or "dry" depending on field evaluations. The incidence of pox was greater in 1985 ($X^2 = 16.536$, df = 1, $P < 0.005$) than in 1986. Bobwhite with wet pox lesions weighed less than those with dry pox ($t = 2.550$, $P = 0.014$) or no pox ($t = 2.393$, $P = 0.018$). In 1985 6-week survivorship of bobwhite showing signs of wet pox was different compared to those with dry pox ($Z = 1.7498$, $P = 0.0402$) and no pox ($Z = 2.9992$, $P = 0.0014$). Survivorship of birds with dry pox and no pox was not different ($Z = 0.6460$, $P = 0.2611$. Bobwhite with wet pox in 1985 had 45.6 and 53.3% overall lower 6-week survival rates than birds with dry and no pox, respectively. No difference in survivorship existed between bobwhite with dry pox and those with no pox in 1986 ($Z = 1.1727$, $P = 0.1210$). No difference in predatory agents responsible for mortalities between birds with or without pox occurred ($X^2 = 0.8851$, df = 2, $P > 0.05$). All mortality of infected birds appeared to be caused by predation and not the disease itself. Implications of these data for inter- and intraspecific disease transmission are discussed.

Key words: avian pox, *Colinus virginianus*, mortality, northern bobwhite, radio-tagging.

Citation: Mueller, B. S., W. R. Davidson and J. B. Atkinson Jr. 1993. Survival of northern bobwhite infected with avian pox. Pages 79-82 *in* K. E. Church and T. V. Dailey, eds. Quail III: national quail symposium. Kansas Dep. Wildl. and Parks, Pratt.

Northern bobwhite are susceptible to numerous diseases and are hosts to a variety of parasites (Kellog and Doster 1972). Avian pox virus is prevalent worldwide, and a diverse array of birds are susceptible to this disease (Karstad 1971, Cunningham 1978). Several strains of avian pox viruses exist, many of which are host-specific to certain species of birds, while others may infect a variety of species (Davidson et al. 1982).

Avian pox is characterized by discrete proliferative lesions on the skin and/or mucous membranes of the mouth and upper respiratory tract (Karstad 1971), and can occur in 2 forms. Dry pox (or the cutaneous form) is characterized by lesions that develop primarily on unfeathered skin, such as the legs and feet. Wet pox involves lesions on the mucous membranes of the mouth, nasal passages, and upper respiratory tract (Davidson et al. 1982). In some cases both dry and wet pox may occur on the same bird.

Avian pox is spread by direct mechanical transmission of the virus (i.e., pecking at lesions; Cunningham 1978). In addition, the disease can be caused by inhalation of viral particles in dust or by blood-feeding insects, particularly mosquitoes (Davidson et al. 1982).

Reports of avian pox in wild bobwhite are infrequent (Stoddard 1931, Davidson et al. 1982, Hansen 1987). However, this disease is known to exist in pen-raised bobwhite, with occasional severe outbreaks (Shillinger and Morley 1937, Poonacha and Wilson 1981). Avian pox is endemic in southeastern bobwhite populations and normally occurs at low levels (Davidson et al. 1982); however, local or regional epizootics may occur. Davidson et al. (1980) described an outbreak of pox in southwestern Georgia and northcentral Florida that resulted in an estimated 12-fold increase in the incidence of infection among wild bobwhite and a mortality rate between 0.6 and 1.2%.

Survival rates of wild free-ranging bobwhite infected with a disease are difficult to determine due to the rapid removal of dead birds by predators and scavengers and to the species' cryptic coloration and secretive nature (Rosene and Lay 1963). To more accurately assess the effect of

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avian pox on bobwhite, we compared survival of wild radio-tagged birds with and without pox lesions. Implications of these data for inter- and intraspecific disease transmission are discussed. Additionally, effects of an increased use of pen-raised bobwhite and their potential to spread this disease are addressed.

STUDY AREA

We studied 2 sites on Dekle Plantation in Grady County, Georgia. Site A encompassed approximately 190 ha forested primarily with mature longleaf (*Pinus palustris*), with loblolly (*P. taeda*), and shortleaf pine (*P. echinata*) interspersed in old-field areas. Dominant understory plants were bracken fern (*Pteridum aquilinum*) and wiregrass (*Aristida stricta*). Agricultural fields (primarily corn), ranging from 0.5 to 7.0 ha, occupied about 15% of this site. Area B contained about 100 ha and had an overstory of naturally regenerated loblolly and shortleaf pine and a grass-forb understory characteristic of southeastern old-field communities. Small (0.5-3.0 ha) corn fields comprised 30% of the area.

METHODS

Bobwhite were captured with baited funnel traps (Stoddard 1931) and with nets at roost sites. Trapping periods were 3-16 January 1985 and 28 December 1985-5 January 1986. Individuals from 25 different coveys were trapped, and radio-transmitters were distributed based on the number of captured bobwhite in a given covey. Number of radio-tagged individuals within a given covey ranged from 2 to 11, ($\bar{x}$ = 6). Birds were aged (Rosene 1969), banded, sexed, weighed, radio-tagged (Shields et al. 1982), and checked for lesions of pox.

We monitored 73 radio-tagged bobwhite in 1985 and 76 in 1986. The 6-week monitoring periods were 17 January-27 February 1985 and 6 January-16 February 1986. Bobwhite were monitored daily and attempts were made to confirm mortality within 24 hours.

We determined depredation from field signs, postpredation condition of the transmitter, direct observations, and remains in hawk nests. The predatory agents were categorized as mammal, avian, or unknown.

Survival was estimated with the staggered entry design (Pollock et al. 1989). A *Z*-test was used for comparing survival curves (Pollock et al. 1989). A 6-week survival time frame was used because it approximates the average length of a pox occurrence (Karstad 1971, Cunningham 1978). Because of our short trapping periods, the survival time frame began immediately after the first capture and ended 6 weeks after the last bird was trapped.

Laboratory confirmation of pox could not be made on location and utilize the radio-tagged bird in the field; therefore, field determination of pox was accomplished by visual inspection using 2 trained observers. Additionally, 5 cases of pox within the total capture sample were confirmed by laboratory analysis consisting of histopathologic examination conducted by the Southeastern Cooperative Wildlife Disease Study.

RESULTS

One hundred and forty-nine wild northern bobwhite were captured, examined for pox infection, radio-tagged, and monitored during the winters of 1985 and 1986. Of this total, 103 (69.1%) had no evidence of avian pox infection, whereas 46 (30.9%) had pox-like lesions. Of the 46 suspected cases of pox, 27 (58.7%) were represented by lesions on the legs or around the nares ("dry pox"), and 19 (41.3%) had lesions on the eyelids, in the mouth, or inside the nasal cavity ("wet pox").

Survivorship

A difference existed in the prevalence of pox between 1985 and 1986 (X^2 = 8.815, df = 1, P = 0.003) and in the survivorship of birds showing pox-like lesions ($\hat{S}_{1985}$ = 0.4514, 95% confidence interval [CI] = 0.2822-0.6206; $\hat{S}_{1986}$ = 0.8264, 95% CI = 0.6015-1.0514; Z = 2.1845, P = 0.0146). Therefore, survivorship data were analyzed by year. No differences in survivorship (Z = 0.4610, P = 0.3228) or prevalence of pox (X^2 = 0.905, df = 1, P = 0.342) were noted between sites A ($\hat{S}$ = 0.7968, 95% CI = 0.7166-0.8770), and B ($\hat{S}$ = 0.7642, 95% CI = 0.6519-0.8764); therefore study sites were combined for analysis of data.

1985.---Of the 73 bobwhite monitored, 39 were free of pox lesions, and 34 had lesions. Of the 34 birds, 18 had lesions typical of wet pox and 16 showed signs of dry pox. Bobwhite with wet pox had a lower survival ($\hat{S}$ = 0.3277, 95% CI = 0.1287-0.5268) than birds with dry pox, ($\hat{S}$ = 0.6027, 95% CI = 0.3394-0.8695; Z = 1.7498, P = 0.0402) or those with no pox, ($\hat{S}$ = 0.7011, 95% CI = 0.5591-0.8437; Z = 2.9992, P = 0.0014). No difference existed between bobwhite with dry pox and those

with no pox ($Z = 0.6460$, $P = 0.2611$). Bobwhite with wet pox had a 45.6 and 53.3% overall lower 6-week survival rate than dry and no-pox birds, respectively.

1986.—Of 76 bobwhite monitored, 64 were free of pox. Of the infected birds, 1 had lesions typical of wet pox and 11 showed signs of dry pox. The 1 bird with wet pox died 2 weeks after capture; with only 1 wet-pox bird in 1986, no significance can be placed on this survivorship. No difference existed between survivorship of bobwhite with dry pox ($\hat{S} = 0.9091$, 95% CI = 0.7300-1.0882) and those without pox ($\hat{S} = 0.7828$, 95% CI = 0.6698-0.8959; $Z = 1.1727$, $P = 0.1210$).

Weights

Body weight of bobwhite did not differ between study sites ($t = 1.667$, SE = 2.162, $P = 0.097$) or between years ($t = 0.689$, SE = 2.151, $P = 0.492$). Therefore, study sites and years were combined for analysis of weight data.

Bobwhite with wet pox weighed less ($\bar{x} = 151.3$ g, SE = 8.872) than birds with dry pox ($\bar{x} = 162.5$ g, SE = 16.663; $t = 2.550$, SE = 4.399, $P = 0.014$) or no pox ($t = 2.393$, SE = 3.057, $P = 0.018$). There was no difference in body weight between birds with dry pox and those with no pox ($\bar{x} = 158.6$ g, SE = 12.069; $t = 1.381$, SE = 2.830, $P = 0.170$).

Predation

Of the 59 mortalities that occurred over the 2 years, we were able to determine the predatory agent responsible for 40 deaths. Twenty-nine were caused by avian predators and 11 by mammals. The remaining 19 deaths could not be assigned to a specific group with confidence; therefore, the deaths were listed as caused by an unknown predator. No difference existed among the predatory agent responsible for a given kill and the disease condition of the bird (dry, wet, or no pox) ($X^2 = 0.8851$, df = 2, $P > 0.05$).

DISCUSSION

While region-wide outbreaks of avian pox are known to occur (Davidson et al. 1980), most epizootics of this disease are probably localized (Davidson et al. 1982). A variety of factors can contribute to the large variations in year-to-year incidence of avian pox (Karstad 1971, Davidson et al. 1980). The incidence of pox we observed (30.9%) falls within the range of prevalence for occurrence in localized areas (Davidson et al. 1980).

Low mortality of bobwhite infected with dry pox in our study agrees with observations of other researchers (Davidson et al. 1982, L. J. Landers, L. P. Simoneaux and C. D. Sisson, pers. commun., Tall Timbers, Inc. and Southeastern Cooperative Wildlife Disease Study, Tallahassee, FL.). Wet pox, however, is a virulent disease that appeared to greatly increase the probability of mortality, albeit through increased vulnerability to predation. Domesticated birds infected with wet pox usually die of starvation or suffocation due to the proliferative nature of this virus in the moist portions of the esophagus or respiratory tract (Cunningham 1978). However, our data suggest the major cause of death for wild bobwhite infected with wet pox is an increased susceptibility to predation caused by an overall weakened condition.

We attribute differences in body weights between wet-pox and dry- or no-pox birds to reduced food intake. This is reported to be caused by impairment of vision, respiration, or swallowing (esophageal occlusion; Cunningham 1978). In domestic fowl infected with avian pox, weight loss is principally an economic consideration (Cunningham 1978); however, among wild bobwhite this apparent loss of fitness has lethal consequences.

Wet-pox birds suffered higher predation, and consequently lower survival. The ratio of avian to mammalian kills in our project appears to be similar to previous studies (Curtis et al. 1989), suggesting that wet pox infection increased vulnerability to both avian and mammalian predators approximately equally.

MANAGEMENT IMPLICATIONS

Avian pox is an endemic disease, with an historically low prevalence in the southeastern U.S. (Stoddard 1931, Davidson et al. 1980, Hansen 1987, Landers et al., pers. commun.). Background levels of avian pox are normally not a management consideration; however, during pox outbreaks a tremendous potential for intraspecific transmission of this disease can occur. This transmission can be mechanical (by pecking of lesions) or through arthropod vectors. The potential for interspecific disease transmission of pox viruses infecting bobwhite is less well known. Currently there are no known methods to prevent or control epizootics originating in the wild.

The potential for released pen-raised bobwhite to elevate the incidence of pox in wild bobwhite populations also is of concern. Pen-raised birds

are often produced at very high densities (1 bird/0.09 m^2 of pen) and avian pox can spread quickly through an entire flock (Shillinger and Morley 1937, Poonacha and Wilson 1981). Further, avian pox is not uncommon among pen-raised bobwhite (Landers et al., pers. commun.).

While very little is known about pen-raised and wild bird interactions, the 2 groups have been documented to mix in the field (Mueller 1985, DeVos, unpubl. data). This close interaction in the field could substantially increase the chances for avian pox transmission.

Ways to reduce the chances that pen-raised bobwhite could contribute to avian pox outbreaks among wild bobwhite have been detailed in Landers et al. (pers. commun.). Adherence to these recommendations can greatly reduce the potential for transmission of avian pox from pen-raised to wild bobwhite.

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