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Serologic Survey for Pathogens Potentially Affecting Pronghorn (*Antilocapra Americana*) Fawn Recruitment in Arizona, USA

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ABSTRACT: During the 1990s, pronghorn (*Antilocapra americana*) populations declined in Arizona, USA. To investigate potential causes of decline, we collected blood samples from hunter-harvested male pronghorn from 2001 to 2003 on four Arizona sites. Sera were tested for antibody to parainfluenza virus type 3 (PI3), bovine viral diarrhea virus, infectious bovine rhinotracheitis virus, bovine respiratory syncytial virus, epizootic hemorrhagic disease virus (EHDV), bluetongue virus (BTV), and *Chlamydia psittaci*. Antibody against PI3 was found in 33% of the samples, whereas antibody against BTV/EHDV was found in 77%. Antibodies to other pathogens were found at low prevalence rates. Although pronghorn decline in Arizona is probably not directly related to disease, potential reproductive effects of BTV/EHDV and PI3 infection on pronghorn in Arizona merit further study.

Key words: *Antilocapra americana*, bluetongue virus, epizootic hemorrhagic disease virus, parainfluenza 3, pronghorn, serologic survey.

During the last 15 yr, pronghorn (*Antilocapra americana*) have declined on numerous sites in Arizona (USA). In 1987, the statewide population of pronghorn was estimated to be 12,000 individuals, but declined to fewer than 8,000 by 2000 (Arizona Game and Fish Department, 2001). Decreased fawn recruitment has been identified as a primary factor in the decline (Arizona Game and Fish Department, 2001), and poor recruitment could be caused by competition with cattle and sheep for adequate forage, inadequate nutrition, predation, and parasites and diseases (Lee et al., 1998).

Over much of their range in Arizona, pronghorn commonly come in contact with other free-ranging ungulates, as well as domestic livestock, and therefore could be exposed to diseases that affect these species. Of particular concern is hemorrhagic disease (HD), caused by bluetongue viruses (BTVs) and epizootic hemorrhagic disease viruses (EHDVs). Hemorrhagic disease has been identified occasionally in free-ranging mule deer (*Odocoileus hemionus*) and white-tailed deer (*Odocoileus virginianus*) (Noon et al., 2002*a*; Dubay et al., 2004), as well as bighorn sheep (*Ovis canadensis*) in Arizona (Noon et al., 2002*b*). Moreover, HD has been implicated in deaths of pronghorn in Wyoming, USA. In 1976, more than 3,200 pronghorn died during a BTV epizootic in eastern Wyoming (Thorne et al., 1988) and in 1984, 288 pronghorn carcasses were recovered; BTV was isolated from necropsied animals during 1984 and an estimated 600 to 1,000 pronghorn died (Thorne et al., 1988). Given that HD epizootics occur in late summer and early fall and coincide with the pronghorn breeding season, BTV or EHDV infections could cause behavioral or physiologic changes or reproductive pathology that decreases breeding success and fawn recruitment. The pathogenicity of these viruses for gestating pronghorn fetuses is uncertain, but infections resulting in reproductive failure have been reported in domestic cattle and sheep, as well as elk (*Cervus elaphus*) and white-tailed deer (Thomas and Trainer, 1970; Bowne, 1971; Hoff et al., 1974; Stott et al., 1982; Thorne et al., 1988; Osborn and MacLachlan, 1990; Sohn and Yuill, 1991; Barker et al., 1992). In addition, BTV or EHDV infections could result in suppressed ovulation in females and reduced sperm motility in males. Thorne et al.

(1988) documented a fawn to female ratio of 47:100 1 yr after a BTV epizootic in 1976, whereas a ratio of 101:100 was calculated outside the area of the epizootic. The objective of this study was to determine if and to what extent pronghorns in Arizona are exposed to common pathogens of ungulates, including BTV/EHDV.

Sites in four grassland regions of Arizona were chosen on the basis of availability of hunter-harvested samples and low fawn recruitment. We identified pronghorn fawn recruitment at several sites from data obtained from the Game Branch, Arizona Game and Fish Department (AZGFD). Mean recruitment from 1990 through 2000 was calculated by game management unit (GMU). Fawn recruitment was calculated with the use of data acquired from fixed-wing aerial surveys during the morning hours in July and August of each year (Jim Heffelfinger, AZGFD, pers. comm.). Two observers and a pilot located and classified all pronghorn while flying at 75–85 knots 30–60 m above ground level. Pilots flew transects approximately 0.5 to 0.7 km apart until the entire grassland area was surveyed. Number of males, females, and fawns was tallied for each area, and the fawn:female ratio was calculated.

Mean recruitment by GMU ($n=24$) from 1990 to 2000 ranged from 11.7±7.9 (mean±SD) fawns per 100 females to 43.6±14.6 fawns per 100 females, and 10 GMUs had average fawn recruitment of more than 25 fawns per 100 females. The four sites chosen for sample collection had average fawn recruitments of 11.7±7.9 fawns per 100 females in GMU 5B (34°52.5′N, 111°15′W), 15.7±13.4 fawns per 100 females in GMU 2B (34°25′N, 109°12.5′W), 28.4±7.5 fawns per 100 females in GMU 8 (35°05′N, 112° 12.5′W), and 25.9±10.3 fawns per 100 females in GMU 1 (34°0′N, 109°17.5′W; Fig. 1). The fawn:female ratios calculated on our four sites were well below those determined in Wyoming by Thorne et al. (1988).

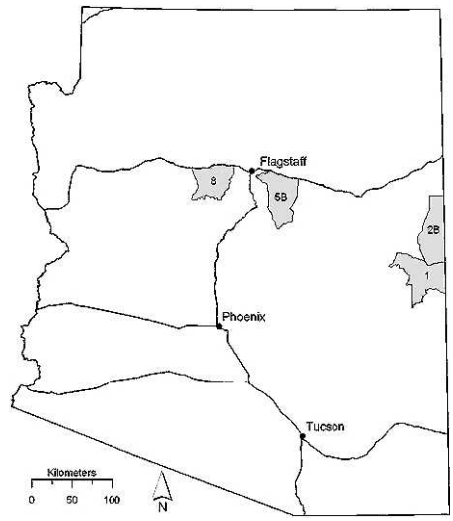

FIGURE 1. Geographic areas in Arizona where hunter-killed male pronghorn were samples for antibodies to PI3, BVD, IBR, BREV, BTV/EHDV, and *chlamydia psittaci* from 2001–2003.

During September 2001–03, 50-ml blood collection tubes (Becton Dickinson Falcon, Franklin Lake, New Jersey, USA) and letters asking for hunter assistance in collecting blood samples were mailed to all pronghorn hunters in GMUs 1, 2B, 5B, and 8 (Fig. 1). Letters requested that hunters collect the freshest and cleanest blood possible (from the heart or chest cavity), keep it cold, and submit the sample to the nearest check station as soon as possible. Check stations were set up in each GMU to collect blood samples. Blood samples were centrifuged at 1,500 × G for 12–15 min, and serum was separated from cells with 1-ml tuberculin syringes without needles (Becton-Dickinson, Beltsville, Maryland, USA). Sera were delivered to the Arizona Veterinary Diagnostic Laboratory (AZVDL; Tucson, Arizona, USA), where samples were tested for antibody to parainfluenza virus type 3 (PI3), bovine viral diarrhea virus (BVD), infectious bovine rhinotracheitis virus (IBR), and bovine respiratory syncytial virus (BRSV) by serum neutralization (OIE Standards Commission, 1996). Antibody to EHDV and BTV was determined

by agar immunodiffusion (AGID) at AZVDL (Pearson and Jochim, 1979). Sera were sent to the Texas Veterinary Medical Diagnostic Laboratory (College Station, Texas, USA) to be tested for complement fixing antibody against *Chlamydia psittaci* (Gustafson and Pearson, 1977). A titer of 16 or higher was considered evidence of exposure to PI3, BVD, IBR, BRSV, and *C. psittaci*. Because of known cross reactions, sera testing positive for antibody on either the BTV or EHDV AGID tests were considered positive for antibodies to BTV, EHDV, or both.

Of the 139 blood samples collected during September 2001, 2002, and 2003, 15 (11%) were negative for antibodies to all pathogens tested. Antibodies to BTV, EHDV, or both were detected in 106 of 138 (77%) samples. Of the 129 samples tested for antibodies against PI3, 43 (33%) were positive, and titers of 256 or more were detected in four animals. Because of insufficient serum, serologic testing for BRSV was conducted in 2002 and 2003 only: six of 84 (7%) samples had antibody to BRSV, and with the exception of one animal, all titers were less than 1:32. Antibodies to BVD were detected in seven of 128 (5%) samples tested and titers of 256 or more were detected in four pronghorn. Two of 92 (2%) animals had antibodies to *C. psittaci*; antibody titers in both animals were 16. Antibodies to IBR were detected in two of 131 (2%) samples tested; both animals were sampled in 2003 and had titers of 4,096 or more.

Antibodies to PI3 and BTV/EHDV were detected in pronghorn sera from all sites. Prevalence of antibodies to BTV/EHDV was 89% in GMU 2B, 65% in GMU 1, 81% in GMU 8, and 73% in GMU 5B. For PI3 antibodies, prevalence was 21% in GMU 2B, 41% in GMU 1, 31% in GMU 8, and 55% in GMU 5B.

Antibody to livestock pathogens has been reported from pronghorn (Dunbar et al., 1999), but potential population impacts have not been evaluated (Lance and Pojar, 1984). Pronghorn populations on Hart Mountain National Antelope Refuge in Oregon (USA) have shown declines similar to those observed in Arizona, and a low fawn to female ratio (1:100 females) was identified as a contributing factor (Dunbar et al., 1999). Antibodies to PI3, BTV, and EHDV were detected in 67, 35, and 30% of tested adult female pronghorn, respectively, and it was concluded that EHDV and BTV circulated sporadically in this herd and that PI3 was not likely contributing to the overall pronghorn decline (Dunbar et al., 1999). Johnson et al. (1986) tested 233 adult pronghorn from Nebraska (USA) and reported antibodies to EHDV, BTV, and BRSV in 30, 27, and 55% of tested animals, respectively; antibody prevalence was similar to the prevalence observed in livestock, suggesting that these viruses were enzootic in Nebraska.

Throughout Arizona, pronghorn are exposed to EHDV and BTV, but it is unknown if these infections affect these populations. Antibodies to BTV previously were reported in 79% of 288 hunter-harvested pronghorn sampled from numerous sites in Arizona (Heffelfinger et al., 1999), and these results are consistent with our more recent data. Although mortality has been reported with BTV in pronghorn in experimental studies (Hoff and Trainer, 1972; Thorne, 1982), HD has not been diagnosed in pronghorn in Arizona in the last decade, suggesting that it is unlikely that HD is a contributor to the pronghorn decline in Arizona. This inconsistency could relate to poor detection of cases in widely dispersed and inconsistently observed pronghorn populations in Arizona, or it might be related to herd immunity. In pronghorn, protective immunity to BTV through prior exposure has been demonstrated (Hoff and Trainer, 1972). A similar situation with HD, involving high BTV/EHDV antibody prevalence with a low incidence of clinical disease, has been reported with other wild cervid populations in both Arizona (Dubay et al., 2004) and Texas (Stallknecht et al.,

1996), suggesting enzootic stability. In addition to acquired immunity, innate immunity, as demonstrated with white-tailed deer from Texas, USA (Gaydos et al., 2002), also could explain the lack of reported clinical HD in Arizona pronghorn populations.

Potential reproductive effects associated with BTV and EHDV in pronghorn in Arizona are unknown but have been suggested with BTV (Sohn and Yuill, 1991). Given that seasonal peaks in BTV and EHDV transmission can coincide with pronghorn breeding season, reproductive effects of BTV/EHDV infection on pronghorn in Arizona merit further study.

Little is known about PI3 infection and antibody response in pronghorn. To date, PI3 infection has not been implicated as a cause of epizootic disease among pronghorn, but infection could increase susceptibility to other infectious agents or cause behavioral changes in affected individuals (Dunbar et al., 1999). Thorsen et al. (1977) isolated PI3 from nasal swabs from four of 50 free-ranging pronghorn sampled in Alberta, but clinical disease associated with PI3 infection in pronghorn has not been reported (Lance and Pojar, 1984). Although exposure to PI3 was documented in all of our study sites, clinical cases have never been reported, and at this time, we cannot link PI3 infections in Arizona pronghorn with disease.

Pronghorn are commonly exposed to the HD viruses (BTV/EHDV) and PI3 in Arizona, but we do not think that these infections are causing large-scale mortality. Potential effects on fawn recruitment deserve further study. Fawn:female ratios for pronghorn in Arizona are lower than those observed elsewhere in their range and these low recruitment rates could represent the combined effects of disease, poor nutrition from drought conditions, lack of adequate fawn hiding cover, and abundance of predators. Research currently is being conducted to investigate these issues.

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LITERATURE CITED

Arizona Game and Fish Department. 2001. Wildlife 2006. Arizona Game and Fish Department, Phoenix, Arizona, 91 pp.

Barker, I. K., A. A. Van Dreumel, and N. Palmer. 1992. Bluetongue and related diseases. *In* Pathology of domestic animals, Vol. 2, 4th Edition, K. V. F. Jubb, P. C. Kennedy and N. Palmer (eds.). Academic Press, San Diego, California, pp. 173–176.

Bowne, J. G. 1971. Bluetongue disease. *In* Advances in veterinary science and comparative medicine, Vol. 15, C. A. Brandley and C. C. E. Cornelius (eds.). Academic Press, New York, New York, pp. 1–46.

Dubay, S. A., J. C. Devos, Jr., T. H. Noon, and S. Boe. 2004. Epizootic of hemorrhagic disease in mule deer near Prescott, Arizona. Journal of Wildlife Diseases 40: 119–124.

Dunbar, M. R., R. Velarde, M. A. Gregg, and M. Bray. 1999. Health evaluation of a pronghorn antelope population in Oregon. Journal of Wildlife Diseases 35: 496–510.

Gaydos, J. K., W. R. Davidson, F. Elvinger, D. G. Mead, E. W. Howerth, and D. E. Stallknecht. 2002. Innate resistance to epizootic hemorrhagic disease in white-tailed deer. Journal of Wildlife Diseases 38: 713–719.

Gustafson, G. A., and J. E. Pearson. 1977. Modified direct bluetongue complement fixation test. National Veterinary Services Protocol, Ames, Iowa.

Heffelfinger, J. R., R. J. Olding, T. H. Noon, M. R. Shupe, and D. P. Betzer. 1999. Copper/selenium levels and occurrence of bluetongue virus in Arizona pronghorn. Proceedings of the Pronghorn Antelope Workshop 18: 32–42.

Hoff, G. L., and D. O. Trainer. 1972. Bluetongue virus in pronghorn antelope. American Journal of Veterinary Research 33: 1013–1016.

———, ———, and M. M. Jochim. 1974. Bluetongue virus and white-tailed deer in an enzootic area of Texas. Journal of Wildlife Diseases 10: 158–163.

Johnson, J. L., T. L. Barber, M. L. Frey, and G. Nason. 1986. Serosurvey for selected pathogens in hunter-killed pronghorns in western

Nebraska. Journal of Wildlife Diseases 22: 87–90.

LANCE, W. R., AND T. M. POJAR. 1984. Diseases and parasites of pronghorn: A review. Colorado Division of Wildlife Special Report 57, Denver, Colorado, 14 pp.

LEE, R. M., J. D. Yoakum, B. W. O'GARA, T. M. POJAR, and R. A. OCKENFELS (eds.). 1998. Pronghorn management guides. Supplement to the 18th Pronghorn Antelope Workshop. Arizona Antelope Foundation, Prescott, Arizona, 110 pp.

NOON, T. H., S. L. WESCHE, D. CAGLE, D. G. MEAD, E. J. BICKNELL, G. A. BRADLEY, S. RIPLOG-PETERSON, D. EDSALL, AND C. REGGIARDO. 2002b. Hemorrhagic disease in bighorn sheep in Arizona. Journal of Wildlife Diseases 38: 172–176.

———, ———, J. HEFFELFINGER, A. FULLER, G. A. BRADLEY, AND C. REGGIARDO. 2002a. Hemorrhagic disease in deer in Arizona. Journal of Wildlife Diseases 38: 177–181.

OIE STANDARDS COMMISSION. 1996. Office International des Epizooties, manual of standards for diagnostic tests and vaccines, 3rd Edition. OIE Press, Paris, France, 763 pp.

OSBORN, B. I., AND N. J. MACLACHLAN. 1990. Congenital disease caused by bluetongue. *In* Laboratory diagnosis of livestock abortion, 3rd Edition, C. A. Kirkbride (ed.). Iowa State University Press, Ames, Iowa, pp. 86–90.

PEARSON, J. E., AND M. M. JOCHIM. 1979. Protocol for the immunodiffusion test for bluetongue. Proceedings of the American Association of Veterinary Diagnosticians 22: 463–475.

SOHN, R., AND T. M. YUILL. 1991. Bluetongue and epizootic hemorrhagic disease in wild ruminants. Bulletin of the Society for Vector Ecology 16: 17–24.

STALLKNECHT, D. E., M. P. LUTTRELL, K. E. SMITH, AND V. F. NETTLES. 1996. Hemorrhagic disease in white-tailed deer in Texas: A case for enzootic stability. Journal of Wildlife Diseases 32: 695–700.

STOTT, J. L., L. H. LAUERMAN, AND A. J. LUEDKE. 1982. Bluetongue virus in pregnant elk and their calves. American Journal of Veterinary Research 43: 423–428.

THOMAS, F. C., AND D. O. TRAINER. 1970. Bluetongue virus: (1) In pregnant white-tailed deer, and, (2) a plaque reduction neutralization test. Journal of Wildlife Diseases 6: 384–388.

THORNE, E. T. 1982. Bluetongue. *In* Diseases of Wildlife in Wyoming, E. T. Thorne, N. Kingston, W. R. Jolley and R. C. Bergstrom (eds.). Wyoming Game and Fish Department, Cheyenne, Wyoming, pp. 5–9.

———, E. S. WILLIAMS, T. R. SPRAKER, W. HELMS, AND T. SEGERSTROM. 1988. Bluetongue in free-ranging pronghorn antelope (*Antilocapra americana*) in Wyoming: 1976 and 1984. Journal of Wildlife Diseases 24: 113–119.

THORSEN, J., L. KARSTAD, W. M. BARRETT, AND G. A. CHALMERS. 1977. Viruses isolated from captive and free-ranging wild ruminants in Alberta. Journal of Wildlife Diseases 13: 74–79.

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