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INFECTIOUS DISEASE SURVEY OF GEMSBOK IN NEW MEXICO

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ABSTRACT: Exotic wildlife can introduce new diseases or act as reservoirs of endemic diseases. On White Sands Missile Range, New Mexico (USA), significant declines in populations of native ungulates generally correspond to increases in range and population density of the exotic gemsbok (*Oryx gazella gazella*), introduced beginning in 1969. We surveyed gemsbok in 2001 for exposure to a variety of diseases potentially important for native ungulates. High seroprevalence was found for malignant catarrhal fever virus (49 [98%] of 50 sera; 43 [96%] of 45 plasma samples), bluetongue virus (48 [96%] of 50), bovine respiratory syncytial virus (33 [66%] of 50), and parainfluenza-3 virus (10 [20%] of 50). Low numbers of *Nematodirus* spp. eggs in a few individuals were the only parasites detected in gemsbok. Exposure to the above diseases in gemsbok is of interest to managers because of potential implications for recovery of desert bighorn sheep (*Ovis canadensis mexicana*) and desert mule deer (*Odocoileus hemionus crooki*) in the White Sands area because each has been implicated in mortality in these species either in the White Sands area or elsewhere in the western/southwestern United States.

Key words: Bluetongue, bovine respiratory syncytial virus, desert bighorn sheep, gemsbok, malignant catarrhal fever, mule deer, New Mexico, parainfluenza-3, pasteurellosis.

INTRODUCTION

Gemsbok (*Oryx gazella gazella*) were introduced onto White Sands Missile Range (WSMR, New Mexico, USA) by the New Mexico Department of Game and Fish (NMDGF) to provide a huntable ungulate in an area of the state providing limited big game hunting opportunities (Wood et al., 1970). Native to arid habitats throughout southern Africa, 93 gemsbok obtained from captive-bred stock were released from 1969–77 and have since greatly increased their range and population density throughout WSMR and surrounding areas (Burkett et al., 2002). Gemsbok population estimates currently range from 3,000–6,000 (L. Bender, unpubl. data), and are still increasing despite hunting harvests in excess of 600–700 annually (Burkett et al., 2002). The current extent of gemsbok range is unknown, but is in excess of 15,000 km^2 in southern New Mexico. The significant increases in range, population size, and population density illustrate how well gemsbok have adapted to habitat conditions of the Chihuahuan Desert, which are markedly similar to their native range in southern Africa (Wood et al., 1970). In the ~30 yr since their introduction, gemsbok have become the most numerous ungulate in the Chihuahuan Desert system of New Mexico (Burkett et al., 2002).

Concurrent with expansion of gemsbok was a decline in native ungulates, particularly desert mule deer (*Odocoileus hemionus crooki*) and the state-designated endangered desert bighorn sheep (*Ovis canadensis mexicana*) (Burkett et al., 2002). Neither proximate nor ultimate causes of decline of these species have been identified, although speculation for desert bighorn has focused on disease, particularly scabies, despite the absence of documented mortalities (Lange et al., 1980; Hoban, 1990). Anecdotal evidence suggests that dramatic population declines coincided with large increases in gemsbok population densities and expansion of gemsbok range. Gemsbok possess a number of characteristics that make them superior com-

petitors to mule deer and desert bighorn in the arid relatively unproductive habitats of the Chihuahuan Desert. Among these are a broader diet breadth (Smith et al., 1998), impressive physiologic and behavioral specializations to conserve water (Taylor, 1969; Hamilton et al., 1977; Burkett et al., 2002), and aggressive behavior (Walther, 1980). Gemsbok are also less vulnerable to native Chihuahuan Desert predators (Burkett et al., 2002).

Gemsbok are an introduced exotic in the Chihuahuan Desert. As well as competing with native species, introduced species have the potential to introduce new diseases, as well as to serve as a new reservoir for existing diseases (Clark et al., 1970; Miller, 2001; Rupprecht et al., 2001). A recent example of the former include introduced reindeer (*Rangifer tarandus tarandus*) that brought the parasite *Elaphostrongylus rangiferi* from Norway to Newfoundland (Canada); this exotic parasite has caused epizootics of cerebrospinal elaphostrongylosis in native caribou (*R. tarandus caribou*) (Ball et al., 2001). Similarly, movement of ranched elk (*Cervus elaphus*) has been implicated in spread of chronic wasting disease (CWD) among game farms (Williams et al., 2001).

Despite the potential of introduced diseases accompanying introduced wildlife, disease surveillance has been sporadic for gemsbok in New Mexico, considering their widespread distribution and large population size. Previous work identified exposure of gemsbok to viral pathogens including bluetongue virus (BTV), epizootic hemorrhagic disease virus (EHDV), bovine viral diarrhea (BVD) virus, parainfluenza-3 (PI3) virus, bovine respiratory syncytial virus (BRSV), equine viral rhinopneumonitis virus, western equine encephalitis virus (WEEV), vesicular stomatitis virus, infectious bovine rhinotracheitis (IBR) virus, and Malpai Springs virus (MSV) (Clark et al., 1988; White Sands Missile Range, 1991; Calisher and Taylor, 1993). Calisher and Taylor (1993) speculated on the role of gemsbok as a reservoir or an amplifying host of viral diseases or viruses (BTV, EHDV, WEEV, MSV) on WSMR based upon long-term high seroprevalence. However, most of this preliminary work focused on longitudinal studies of select diseases, not potential implications to wild ungulate populations, because gemsbok numbers and distribution were still relatively limited during these early studies (600–800; Clark et al., 1988) and population declines of native ungulates were not yet widespread.

Since the initial disease work in the 1980s, gemsbok numbers and distribution have increased dramatically, and populations of native ungulates have declined significantly and show no signs of recovery. Thus, our goal was to survey free-ranging gemsbok for exposure to selected diseases and parasites, emphasizing those associated with exotic ungulates or previously identified with gemsbok in New Mexico which could pose potential threats to native ungulates (desert mule deer, desert bighorn sheep, pronghorn [*Antilocapra americana*]) in New Mexico.

MATERIALS AND METHODS

White Sands Missile Range (approximately 32°50′N, 106°30′W) encompasses about 8,800 km^2 (approximately 165 km north to south and 64 km east to west) and includes portions of the Jornada del Muerto, the Tularosa Basin, and the San Andres and several other mountain ranges of southcentral New Mexico. Major vegetation communities on WSMR include semidesert grassland, Chihuahuan desertscrub, and Great Basin conifer woodland (Burkett et al., 2002). Physical, climatic, and vegetation characteristics of WSMR and surrounding areas are detailed in Burkett et al. (2002).

We collected blood and fecal samples from 101 gemsbok captured by aerial darting on WSMR incidental to a large-scale capture operation during February and April 2001. Gemsbok were captured over approximately 700,000 ha and included all sex and age classes except those <6 mo old. We collected blood samples by jugular puncture and fecal samples from the rectum. Blood samples were divided into serum and ethylenediaminetetraacetic acid tubes and placed in a cooler immediately after collection. Serum tubes were spun (3,200 × G; 8–10 min) and separated the evening following

TABLE 1. Results of serology for evidence of exposure to *Leptospira interrogans* (Leptospira; serovars bratislava, canicola, grippotyphosa, hardjo, icterohemorrhagiae, and pomona), *Brucella abortus, Brucella ovis, Mycobacterium avium* subsp. *paratuberculosis, Anaplasma* spp., bovine viral diarrhea (BVD) virus, infectious bovine rhinotracheitis virus (bovine herpesvirus-1; IBR), bluetonge virus (BRV), bovine respiratory syncytial virus (BRSV), parainfluenza-3 (PI-3) virus, and malignant catarrhal fever (MCF) virus in free-ranging gemsbok from White Sands Missile Range, New Mexico.

Agent	Number tested/number positive	Test (threshold) for positive reaction)[a]	Reference
Leptospira	1/50	MAT (1:100)	Gochenour et al., 1958
Brucella abortus	0/50	Card[b]	Alton et al., 1985
Brucella ovis	1/50	ELISA[c]	Lee et al., 1985
Mycobacterium avium subsp. *paratuberculosis*	0/50	ELISA[d]	Shulaw et al., 1986
Anaplasma spp.	0/50	CF (1:5)	Hidalgo and Dimopoullos, 1967
BVD virus	2/50	VN (1:4)	Carbrey et al., 1971
IBR virus	0/50	VN (1:4)	Carbrey et al., 1971
BTV	48/50	cCELISA[e]	Pearson et al., 1992
BRSV	33/50	VN (1:4)	Westenbrink et al., 1985
PI-3 virus	10/50	VN (1:4)	Carbrey et al., 1971
MCF virus (serum)	49/50	CI-ELISA[f]	Li et al., 1994, 2001
MCF virus (plasma)	43/45	CI-ELISA[f]	Li et al., 1994, 2001

[a] Tests were: MAT=microscopic agglutination test; CF=complement fixation; ELISA=enzyme-linked immunoabsorbent assay (c=competitive, CI=competitive inhibition); VN=virus neutralization. Titer in parentheses is threshold value for positive test.

[b] No agglutination on BAPA card=negative; reactors on BAPA then cross-checked by BALA (latex agglutination).

[c] Any optical density greater than the National Veterinary Services Laboratory (Ames, Iowa) standard positive at 1:10 by complement fixation.

[d] Ratio of antibody detected in serum sample compared to a high positive standard serum (S/P)>0.25=positive; S/P 0.200–0.249=suspect.

[e] >50% inhibition of negative controls=positive.

[f] >25% inhibition based on a panel of MCF virus-free sheep sera.

collection. Fecal samples were refrigerated within 30–60 min after collection until analysis.

We randomly selected 50 individual blood samples for disease screening. We selected health tests (Table 1) based on potential significance to gemsbok or other ungulates in the Chihuahuan Desert system. We tested for malignant catarrhal fever (MCF) virus antibodies using the competitive inhibition enzyme-linked immunosorbent assay (Li et al., 1994, 2001) on serum and an additional 45 plasma samples following positive results from serum (not all serum and plasma samples were from the same individuals). Parasites were identified and quantified using either fecal flotation (Foreyt, 2001) or the modified Baermann test for lungworms (Forrester and Lankester, 1997).

RESULTS

Exposure of gemsbok to a variety of disease agents was shown, of which three were of immediate relevance for native ungulates on WSMR (Table 1). First, 98% of serum samples and 96% of plasma samples from gemsbok were positive for MCF virus antibody. Secondly, 66% of gemsbok were positive for BRSV antibodies (titers 1:4–1:128) and 20% for PI3 virus (titers 1:4–1:128) antibodies. Lastly, 96% of gemsbok were positive for BTV antibody. Small numbers of animals were positive for BVD virus antibodies and antibodies against *Brucella ovis* and *Leptospira interrogans* subspecies bratislava (titer 1:100). Antibodies were not detected for other agents (Table 1). The only parasite found in fecal floatations was *Nematodirus* spp. (2–5 eggs/g) in three (6%) of 50 gemsbok.

DISCUSSION

Malignant catarrhal fever is a sporadic, infectious disease of domestic and wild ruminants (Heuschele and Reid, 2001). It is often fatal in aberrant hosts, with deer in particular being especially vulnerable.

Classic MCF was first attributed to either alcelaphine herpesvirus-1 (AlHV-1) the virus first identified in wildebeest (*Connochaetes taurinus*), or ovine herpesvirus-2 (OvHV-2), the natural host of which is domestic sheep (Heuschele and Reid, 2001). Since identification of these viruses, at least four additional herpesviruses in the MCF group have been recognized (Li et al., 2000, 2001; Heuschele and Reid, 2001). Antibody cross-reactions to AlHV-1 in a variety of other African ungulates suggested that other unidentified MCF-group herpesviruses exist (Plowright, 1981). Although transmission of MCF is not completely understood, transmission of AlHV-1 to other species usually occurs from newborns, who shed virus in tears and nasal secretions until ~3–4 mo old, when they develop adequate immunity and cease shedding virus (Heuschele and Reid, 2001).

Polymerase chain reaction sequencing identified two new herpesviruses from gemsbok on WSMR: one within the MCF virus group that is closely related to AlHV-1 and another within the non-MCF lymphotropic herpesvirus group (Li et al., 2003). Currently, no data are available as to whether these viruses are pathogenic to other species. However, presence of an MCF-group herpesvirus similar to AlHV-1 warrants further investigation due to the possibility that this virus may cause MCF in desert mule deer and other North American ungulates.

Antibodies against two bovine respiratory viruses implicated as predisposing agents to pasteurellosis, BRSV and PI3 virus (Miller, 2001), were found in gemsbok on WSMR. Pneumonic pasteurellosis can cause high mortality in bighorn sheep populations (Miller, 2001). Heavy infestation with lungworms (i.e., *Protostrongylus* spp., *Muellerius* spp.) may also predispose bighorn sheep to pneumonic pasteurellosis (Spraker and Hibler, 1982; Miller, 2001). It has been hypothesized that abundance and distribution of bighorn sheep populations is limited by recurrent pasteurellosis epidemics (Hobbs and Miller, 1992).

Gemsbok occur throughout desert bighorn range adjacent to WSMR, and because the San Andres range is a priority desert bighorn recovery area in New Mexico, the potential for disease to limit success of future transplant efforts needs to be evaluated. This is especially true because desert bighorn apparently declined from epizootics of unknown origin several times in the past in this area (Hoban, 1990). Gemsbok should be sampled to determine whether BRSV and PI3 virus are being maintained in the population, or whether antibodies detected were due to exposure to bighorn sheep and/or livestock with these viruses. This is particularly relevant given that recent surveillance resulted in isolation of *Pasteurella trehalosi* from gemsbok on San Andres National Wildlife Refuge (SANWR), adjacent to WSMR (Bender and Weisenberger, unpubl. data). Thus, if BRSV and/or PI3 virus are common in gemsbok there could be an impact on successful desert bighorn sheep recovery in areas occupied by gemsbok.

Bluetongue is an arthropod-vectored infectious but noncontagious disease of wild and domestic ruminants (Ramsay et al., 1985; Howerth et al., 2001). Among ruminants present in the gemsbok range, bluetongue has caused clinical disease in mule deer, elk, bison (*Bison bison*), pronghorn, and desert bighorn sheep (Howerth et al., 2001). Bluetongue can cause extensive wildlife mortalities over large areas (Thorne, 1982; Howerth et al., 2001), low-level annual mortality (Hoff et al., 1974), and limit reproduction following outbreaks due to fetal resorption, abortion, and/or congenital physical or nervous system deformities without causing clinical disease in adults (Thorne, 1982; Ramsay et al., 1985). The presence of extensive outbreaks of bluetongue in domestic sheep and subsequent bluetongue in desert bighorn sheep was implicated in elimination of desert bighorn from Texas (Robinson et al., 1967). Mortality from bluetongue also

was documented in desert bighorn from SANWR in New Mexico (Lange et al., 1980; Hoban, 1990).

Perinatal infection with BTV has been reported in an aborted gemsbok calf (Ramsay et al., 1985), though no cases of clinical disease in adults have been reported (Howerth et al., 2001). However, based on high levels of exposure to BTV detected during long-term serologic surveillance, gemsbok may serve as amplifying hosts of BTV in the WSMR vicinity (Calisher and Taylor, 1993). If so, presence of BTV in gemsbok has implications for native ungulates in gemsbok range, particularly mule deer and desert bighorn sheep, both of which are susceptible to bluetongue and present in extremely low numbers.

Management implications

Behavioral, physiologic, and physical characteristics make gemsbok a superior competitor to native ungulates in the Chihuahuan Desert system, and thus they potentially limit population size of native ungulates (Burkett et al., 2002). Gemsbok also may have introduced new disease agents (i.e., MCF) and/or serve as a reservoir for existing disease agents to which they are resistant but native ungulates susceptible. Combined impacts of competition and disease may make restoration of viable populations of native species such as desert bighorn sheep difficult or impossible in areas dominated by gemsbok. Further, popular game species such as desert mule deer also may remain depressed.

In New Mexico, gemsbok are highly prized by the hunting public as a trophy of great value; gemsbok hunts on WSMR are once in a lifetime licenses, with >4,000 hunters applying for >800 licenses annually. Any effort to eliminate or limit gemsbok numbers because of perceived detriment to desert bighorn sheep and desert mule deer would have significant recreational and economic impacts. Greater investigation is therefore needed on how disease and/or competition may impact populations of native ungulates. Without an understanding of the exact nature of disease relationships between gemsbok and native ungulates, it is unlikely that there can be successful management or recovery of gemsbok or native ungulates in the Chihuahuan Desert system in New Mexico.

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