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HEALTH PROTOCOL FOR TRANSLOCATION OF FREE-RANGING ELK

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ABSTRACT: When considering an elk (*Cervus elaphus*) restoration program, wildlife managers must evaluate the positive and negative elements of translocation. We prepared this protocol to give an overview of health considerations associated with translocation of elk, with an emphasis on movement of free-ranging elk from western North America to the southeastern USA. We evaluated infectious agents and ectoparasites reported in elk from two perspectives. First, we made a qualitative estimate of the ability of the agent to be introduced and to become established. This was done using a selected set of epidemiologic factors. Second, if there was a good possibility that the organism could become established in the release area, the potential pathological consequences for elk and other wildlife, domestic animals, and humans were assessed via examination of the literature and consultation with other animal health specialists. The results of these evaluations were used to classify infectious agents and ectoparasites as low risk ($n = 174$), unknown risk ($n = 10$), and high risk ($n = 9$). We classified *Anaplasma marginale, Anaplasma ovis, Mycobacterium paratuberculosis, Pasteurella multocida* serotype 3, *Elaphostrongylus cervi, Dicrocoelium dendriticum, Fascioloides magna, Echinococcus granulosus, Dermacentor albipictus,* and *Otobius megnini* as unknown risks. High risk infectious agents and ectoparasites were the agent of chronic wasting disease, *Brucella abortus, Mycobacterium bovis, Dermacentor andersoni, Ixodes pacificus,* and *Psoroptes* sp. *Parelaphostrongylus tenuis, Elaeophora schneideri,* and a *Babesia* sp. are parasites endemic in the southeastern USA that may present a "reverse risk" and adversely affect elk if released in some parts of the region. We developed a five-component protocol to reduce the risk of introduction of high risk infectious agents and ectoparasites that included: (1) evaluation of the health status of source populations, (2) quarantines, (3) physical examination and diagnostic testing, (4) restrictions on translocation of animals from certain geographic areas or populations, and (5) prophylactic treatment.

Key words: Cervus elaphus, ectoparasites, elk, importation, infectious agents, restoration, translocation.

INTRODUCTION

Historically, elk (*cervus elaphus*) were indigenous to much of the eastern United States (Bryant and Maser, 1982), and there is considerable public interest in the concept of elk restoration. Game agencies in Arkansas (USA) and Kentucky (USA) made trial elk translocations in 1981 to 1985 and 1997 to present, respectively (M.E. Cartwright, pers. comm.; J. Gassett, pers. comm.), and wildlife managers are in the process of evaluating the positive and negative elements of elk restoration. Translocation is an accepted wildlife management tool, and has been a component in the success of restoration programs involving species such as the wild turkey (*Meleagris gallopavo*) (Lewis, 1987) and white-tailed deer (*Odocoileus virginianus*) (Downing, 1987); however, the potential negative elements of translocation are numerous (see Nielsen and Brown, 1988). Among the negative elements are the potential to cause adverse changes in the composition of plant and animal communities, to create conflicts with humans and agriculture, and to introduce infectious agents and ectoparasites injurious to wildlife, domestic animals, or humans.

Elk are hosts to a variety of infectious agents and ectoparasites and should be viewed as "biological packages" (Davidson and Nettles, 1992). Prior translocations of elk or red deer (also *C. elaphus*) have been attributed as the vehicle for introduction of liver flukes (*Fascioloides magna*) to Italy (Bassi, 1893 *in* Kistner, 1982), the abomasal nematodes *Spiculopteragia spiculoptera, Spiculopteragia asymmetrica,* and

Ostertagia leptospicularis to Argentina, New Zealand, and Texas (USA) (Andrews, 1973; Suarez et al., 1991; Rickard et al., 1993), and *Onchocerca cervipedis* to Poland (Tarezy'nski, 1954). *Elaphostrongylus cervi* was found in quarantined red deer being imported from New Zealand into Canada (Gajadhar et al., 1994).

Although it is impossible to create a "sterile animal" without accompanying organisms (e.g., normal flora of the mucous membranes, skin, and digestive tract), it is feasible to reduce the risks by not moving animals that are infected with known pathogenic agents. We present a health protocol for translocation of elk because the problems created when an infectious agent or ectoparasite enters a free-ranging wildlife population are difficult or impossible to resolve. Additionally, we contend that infectious agent and ectoparasite avoidance is one of the most important tools available to state and federal authorities in regard to wildlife health management. Emphasis in this protocol is on translocation of wild elk from western North America to the southeastern USA based on disease status and information available through 1997.

METHODS

We conducted a search of databases, including AGRICOLA, BIOSIS, and MEDLINE, and reference literature, to determine what infectious agents and ectoparasites had been reported from free-ranging and captive elk or red deer worldwide. Red deer were included because they are conspecific with elk and presumably are susceptible to the same agents. We then evaluated each infectious agent and ectoparasite reported in elk based on two perspectives (Schaffer et al., 1981; Davidson and Nettles, 1992). First, we made a qualitative estimate of the ability of the agent to be introduced and to become established in the southeastern USA. Second, if there was a good probability that the organism could become established in the release area, we assessed the pathological consequences based on the disease potential for elk and other wildlife, domestic animals, and human beings. Because elk from outside of North America are not being considered for translocation, we rated infectious agents and ectoparasite not endemic to North America or not reported from elk in North America as low risk. We also rated infectious agents and ectoparasites endemic in the southeastern USA as low risk regardless of the theoretical possibility that infected elk could intensify an existing problem. For all other agents we considered the following epidemiologic factors important in enhancing or inhibiting establishment in the release area: (1) the organism can be harbored by elk without elk exhibiting illness during transport; (2) the organism is transmitted directly from elk to elk (i.e., no vector or intermediate host required); (3) if required, known or potential vectors or intermediate hosts already are present in the release area; (4) the organism is noncontagious (i.e., elk are dead-end hosts); (5) the organism may not be able to survive the climate of the southeastern USA; and (6) the organism infects animals other than elk.

We made qualitative assessments of the health consequences of introduction for agents determined to be of unknown or high risk for introduction based on the pathological potential for each organism in elk, other wildlife, domestic animals, and humans. These assessments were made via examination of the literature and through consultation with other animal health specialists. We considered the risk of health consequences for each infectious agent or ectoparasite to be low, unknown, or high. The final risk assignment was made based on an evaluation of the two perspectives delineated above (potential for establishment and pathogenicity). An infectious agent or ectoparasite was assigned low risk status if (1) the agent does not occur in North America or the agent has never been reported in elk in North America; (2) the agent is endemic in the southeastern United States; (3) only serologic evidence of exposure to the agent has been reported; (4) only experimental evidence of susceptibility exists; (5) the agent is not known to be pathogenic to elk, other wildlife, domestic animals, or humans; (6) the agent is noncontagious; or (7) the agent is highly pathogenic to elk, and it is likely that infected animals would be detected during the transportation phase of the trip and the shipment would be stopped. Infectious agents and ectoparasites were placed in the unknown risk category when sufficient information was not available, relative to either the potential for establishment in the Southeast or the pathological potential, to make an assignment of low or high risk. High risk infectious agents and ectoparasites were those that were considered to have potential for introduction into the southeastern USA via translocation of wild elk and a high potential for health consequences relative to elk, white-tailed deer,

other wildlife, livestock, and/or humans. Infectious agents and ectoparasites present in the southeastern USA that could impact the health of imported elk populations also were considered and are designated as "reverse risk" agents.

RESULTS AND DISCUSSION

Low risk infectious agents and ectoparasites

We found reports of 190 infectious agents and ectoparasites in elk and red deer, and considered 174 of these to be low risk relative to potential health hazards in regard to translocation (Table 1). Some of the agents could have been designated as "low risk" relative to ≥2 of the categories listed in the Methods section. For example, only serological or experimental infection data exist for diseases such as bluetongue (Hoff and Trainer, 1978), infectious bovine rhinotracheitis (Kingscote et al., 1987), parainfluenza virus 3 (Kingscote et al., 1987), tularemia (Merrell and Wright, 1978), most serovars of leptospirosis (Kistner, 1982; Bender and Hall, 1996), several arboviral encephalitis viruses (Eldridge et al., 1987), and vesicular stomatitis (Webb et al., 1987). Proof that wild elk are capable reservoirs of the infectious agents of these diseases has not been established. In these instances, the most compelling reason for a low risk rating (i.e., endemic status in the southeastern USA versus lack of proof by isolation) was selected based on what generally is known about the agent in question. Another example of our risk evaluation process was *Bacillus anthracis,* the causative agent of anthrax, which is endemic in soil in parts of the southeastern USA but would result in a "new" disease in other areas (Choquette and Broughton, 1981; Kistner, 1982). Anthrax is a rapid, fatal disease; however, we believe it is a low risk because infected animals develop clinical signs and die, and infected elk would likely be detected during the transportation phase of the translocation.

Unknown risk infectious agents and ectoparasites

Agents we classified as unknown risk were *Anaplasma marginale, Anaplasma ovis, Mycobacterium paratuberculosis, Pasteurella multocida* serotype 3, *Elaphostrongylus cervi, Dicrocoelium dendriticum, Fascioloides magna, Echinococcus granulosus, Dermacentor albipictus* and *Otobius megnini.* Experimental studies have demonstrated that elk can harbor asymptomatic infections with *A. marginale* and *A. ovis,* the causes of anaplasmosis in cattle and sheep, respectively (Howe et al., 1964; Renshaw et al., 1979; Zaugg et al., 1996). Subclinical infections with *A. marginale* can last for ≥1 yr in experimentally infected elk (Howe et al., 1964; Renshaw et al., 1979). However, efforts to recover *Anaplasma* spp. from free-ranging elk populations have been unsuccessful, suggesting that even though these species are susceptible, they are probably not responsible for maintaining infections or acting as a source of infection for cattle (Howe and Hepworth, 1965; Vaughn et al., 1976; Merrell and Wright, 1978; Kuttler, 1984). Clinical anaplasmosis has not been reported in elk.

Paratuberculosis or Johne's disease is caused by the bacterium *M. paratuberculosis.* Clinical disease has been important in farmed red deer in Europe and New Zealand (Vance, 1961; Smits, 1991; de Lisle et al., 1993), but only one instance of spontaneous infection in wild elk in North America has been reported (Jessup et al., 1981) and this was in an elk herd associated with dairy cattle and known-infected exotic cervids (Riemann et al., 1979). The elk in this herd have remained infected for 13 yr, including 6 yr without observed clinical signs (Cook et al., 1997). Experimental inoculation revealed that elk can harbor the organism for 1 yr without clinical signs (Williams et al., 1983). This disease is endemic in cattle in the southeastern USA but the extent to which elk could serve as an important new reservoir

TABLE 1. Infectious agents and ectoparasites reported from elk and red deer considered as low risk of health consequences from importation of elk for restoration.

Reason/s for low risk	Infectious agents and ectoparasites
Endemic in southeastern USA	Viruses: Bluetongue virus[a], bovine virus diarrhea virus, epizootic hemorrhagic disease virus, infectious bovine rhinotracheitis virus[a], parainfluenza 3 virus (PI3)[a], rabies virus Bacteria and Rickettsia: *Actinobacillus lignieresii, Actinomyces bovis, Actinomyces pyogenes, Clostridium chauvoei, C. hemolyticum, C. novyi, C. perfringens, C. tetani, Francisella tularensis*[a], *Fusobacterium necrophorum, Leptospira interrogans australis*[a], *L. interrogans autumnalis, L. interrogans ballum*[a], *L. interrogans brataslava, L. interrogans grippotyphosa*[a], *L. interrogans hardjo, L. interrogans icterohemorrhagiae*[a], *L. interrogans pomona, Listeria monocytogenes, Mycobacterium avium, Salmonella bovis morbificans, S. cholerasuis, S. dublin, S. typhimurium, Staphylococcus aureus, Yersinia enterocolitica, Y. pseudotuberculosis* Fungi: *Absidia corymbifera, Aspergillus fumigatus* Protozoa: *Cryptosporidia* sp., *Eimeria zurnii, Toxoplasma gondii, Trypanosoma cervi* Helminths: *Moniezia benedeni, M. expansa, Taenia hydatigena, Capillaria bovis* (=*brevipes*), *Dictyocaulus viviparus, Elaeophora schneideri, Haemonchus contortus, Mazamastrongylus odocoilei, Nematodirus filicollis, N. helvetianus, N. spathiger, Oesophagostomum cervi, O. venulosum, Onchocerca cevipedis, Ostertagia ostertagi, Parelaphostrongylus tenuis, Setaria yehi, Trichostrongylus askivali, Trichostrongylus axei, Trichuris ovis* Arthropods: *Damalinia lipeuroides*
Not reported in North America or not reported in North American elk	Viruses: Adenovirus, astrovirus, foot and mouth disease virus, herpesvirus of Cervidae type 1 (HCV-1), louping ill virus, parapoxvirus, rinderpest virus Fungi: "Mycotic pneumonia" Protozoa: *Babesia capreoli, B. divergens, B. ovis, Cryptosporidia* sp., *Eimeria asymmetrica, E. austriaca, E. cervi, E. elaphi, E. gallivalerioi, E. robusta, E. schoenbuchi, E. sordida* Helminths: *Taenia cervi, Acanthospiculum cervipedis, A. flexuose, A. jakutensis, Apteragia quadrispiculata, Bicaulus sagittatus, Chabertia ovina, Cooperia onchophora, C. pectinata, Elaeophora elaphi, Nematodirus oriatianus, N. roscidus, Oesophagostomum sikae, Onchocerca fluxuosa, O. cervipedis, Rinadia mathevossiani, Setaria altaica, S. cervi, Skrjabinagia kolchida, Spiculopteragia schulzi, Trichocephalus ovis, Fasciola hepatica, Fischoederius skrjabina, Paramphistomum* sp. Arthropods: *Bovicola longicornis, B. tibialis, Cephenemyia auribarbis, C. ulrichii, Damalinia meyeri, Demodex acutipes, D. kutzeri, Dermacentor marginatus, D. niveus, D. reticulatus, D. silvarum, Hypoderma actaeon, H. diana, Haemaphysalis concinna, H. inermis, H. japonica, H. longicornis, H. punctata, H. sulcata, Hyalomma anatolicum, H. asiaticum, H. detritum, H. lusitanicum, H. marginatum marginatum, Ixodes persulcatus, I. ricinus, Rhipicephalus bursa, R. leporis, R. pumilio, R. pusillus, R. rossicus, R. schultzei, R. turanicus, Sarcoptes scabiei rupicaprae*
Antibody reported/no agent recovered from North American elk	Viruses: California encephalitis virus, Cache Valley virus, Jamestown Canyon encephalitis virus, snowshoe hare encephalitis virus, vesicular stomatitis virus
Experimental infection/no agent recovered from North American elk	Viruses: Contagious echthyma virus Bacteria and Rickettsia: *Chlamydia psittaci*

TABLE 1. Continued.

Reason/s for low risk	Infectious agents and ectoparasites
Agent not known to be pathogenic	Viruses: Papillomavirus (although no virus isolated) Bacteria and Rickettsia: *Eperythrozoon* Protozoa: *Eimeria wapiti, E. wassilewski, Sappinia diploidea, Sarcocystis miescheriana, S. sybillensis, S. wapiti, Trypanosoma* sp. Helminths: *Capillaria brevipes, Nematodirella longispiculata, Thysanosoma actinoides, Marshallagia marshalli, Ostertagia leptospicularis, Spiculopteragia asymmetrica, S. spiculoptera, Trichostrongylus colubriformis* Arthropods: *Bovicola longicornis, B. concavifrons, Cephenemyia jellisoni, Damalinia parallela, Lipoptena cervi, L. depressa, Neolipoptena ferrisi, Solenopotes ferrisi, Werneckiella equi*
Infection not contagious from elk	Viruses: Malignant catarrhal fever virus Fungi: *Coccidiodes immitis*
Agent highly detectable/survival low	Bacteria and Rickettsia: *Bacillus anthracis*

[a] Denotes serological evidence only.

or disseminator species for *M. paratuberculosis* is unknown.

Pasteurella multocida can cause pneumonia in elk (Cowan, 1951; Murie, 1951; Franson and Smith, 1988; Smits, 1991, 1992; Rhyan et al., 1997); however, the report of septicemic pasteurellosis due to serotype 3 in 48 elk in a large herd at the National Elk Refuge near Jackson (Wyoming; USA) (Franson and Smith, 1988) and on state feed grounds in Wyoming (E. S. Williams, pers. comm.) is of concern. Septicemic pasteurellosis of cattle is an acute infection that may be the result of infection with highly pathogenic strains of *P. multocida* (serotypes B:2 and E:2) that are uncommon in North America but are endemic in parts of Europe, Africa, the Near East, and southern Asia. In the USA, hemorrhagic septicemia due to serotype B:2 was confirmed in bison (*Bison bison*) in 1992; other reports were made in 1912 and 1965 (United States Animal Health Association, 1992).

Elaphostrongylus cervi, a nematode parasite found in red deer in Europe and New Zealand, was found in red deer in a quarantine facility in Canada. These red deer were being imported from New Zealand for use in deer farming (Gajadhar et al., 1994). A closely related nematode, *Elaphostrongylus rangiferi,* likely was introduced into free-ranging caribou (*Rangifer tarandus*) in Newfoundland (Canada) via infected reindeer brought from Norway (Lankester and Fong, 1989). *Elaphostrongylus cervi* is neurotropic and can cause neurologic disturbances in mule deer (*Odocoileus hemionus*), a nondefinitive host (Gajadhar and Tessaro, 1995). This parasite has not been reported in elk in North America, and there appears to be no risk of introducing this parasite via free-ranging elk from the USA. The only potential hazard would be if farmed elk from an infected captive herd were substituted for *bona fide* free-ranging elk.

Dicrocoelium dendriticum, the lancet fluke, is widespread in Europe and Asia and probably was introduced to North America (Mapes, 1951). It occurs in the bile ducts of a wide range of domestic and wild mammals including sheep, cattle, and deer (Mapes, 1951; Davis and Libke, 1971; Pybus, 1990) and can cause progressive clinical illness, particularly in older sheep (Bowman, 1995). The range of this fluke in North America is limited, and there is only one report of its occurrence in elk in North America in the literature. It was reported in mule deer, white-tailed deer, and elk in Alberta (Canada; Pybus,

1990), in white-tailed deer and woodchucks (*Marmota monax*) in New York (USA) (Mapes and Baker, 1950; Mapes, 1951), and possibly in Key deer (*O. virginianus clavium*) in Florida (USA) (Schulte et al., 1976). *Dicrocoelium dendriticum* is not reported in the southeastern USA, thus the importation of infected elk could possibly lead to its spread to wildlife and livestock in the region.

The large liver fluke (*F. magna*) of deer and elk is not highly pathogenic for cervids, but it can cause serious necrotizing hepatitis in domestic sheep and extensive liver tissue damage in cattle. This trematode parasite is present in the southeastern USA, but primarily in the costal areas and river bottoms of the deep south (Pursglove et al., 1977; Malone, 1986); however, the current distribution is patchy and possibly could be explained by past relocations of infected white-tailed deer (Pursglove et al., 1977). An alternative hypothesis is that the parasite's distribution in the region is determined by habitat factors, which in turn influence the abundance of aquatic snails that are required to complete the fluke's life cycle. Introduction of elk infected with *F. magna* may result in its establishment at new locations. A treatment protocol has been developed to reduce infection in elk (Pybus et al., 1991).

Echinococcus granulosus, a cestode for which elk can serve as an intermediate host, appears to exist as host-adapted strains, some strains of which are capable of causing human disease. The cestode strain present in Utah and neighboring states probably is the sheep strain of *E. granulosus,* and has caused human infections in the region. Infection is associated with persons who herd sheep and have infected sheep dogs (Schantz et al., 1995; Thompson, 1995). Cervids are not considered hosts for the sheep strain. The cervid strain of *E. granulosus* is found in North America, primarily in the holarctic zones of the tundra and boreal forests of Canada and Alaska (USA), but also in northern Minnesota (USA) and along the Cascade Mountains into northern California (USA). The cervid strain cycles primarily among wolves (*Canis lupus*), moose (*Alces alces*) and caribou, but coyotes (*Canis latrans*) can substitute as a definitive host, and deer and elk can serve as intermediate hosts. The cervid strain of *E. granulosus* is assumed to have zoonotic potential based on infection among people in northern regions where the sheep strain does not exist. However, human disease has not been linked to cervid strain *E. granulosus* infections except when certain risk factors are present (e.g., presence of uncontrolled dogs, ingestion of ruminant viscera by dogs, and unsanitary living conditions) (Schantz et al., 1995). Human cases of echinococcosis have not been linked to exposure to elk.

The winter tick, *D. albipictus,* can cause severely debilitating alopecia in cervids, including elk (Murie, 1951; Love, 1955; Franson and Smith, 1988; Samuel et al., 1991). Previously, the taxonomy was undecided (Bishopp and Trembley, 1945), but authorities consider *D. albipictus* as synonymous with *D. nigrolineatus,* which is present in the southeastern USA. Nevertheless, debilitating alopecia has not been associated with the southern *D. albipictus* (formerly *D. nigrolineatus*), thus the possibility exists that the northern *D. albipictus* could be a more pathogenic strain.

The spinous ear tick (*O. megnini*) resides in the external ear canals of dogs, sheep, horses, cattle, and many wild ruminants and occurs mostly in the southwestern USA. Pathogenicity occurs when ticks create inflammation that leads to secondary bacterial infection in the ear. Climatological factors likely would impede establishment of this tick outside of the Southwest (Bishopp and Trembley, 1945); however, *O. megnini* has been reported on elk in Wyoming (E. S. Williams, pers. comm.), horses in Florida (Bishopp and Trembley, 1945), and on white-tailed deer in Arkansas (Southeastern Cooperative

Wildlife Disease Study [SCWDS], unpubl. data).

High risk infectious agents and ectoparasites

High risk infectious agents and ectoparasites were the agent of chronic wasting disease (CWD), *Brucella abortus, Mycobacterium bovis, Dermacentor andersoni, Ixodes pacificus,* and *Psoroptes* sp., the agent of psoroptic mange. Chronic wasting disease belongs to a family of diseases known as transmissible spongiform encephalopathies that are characterized by spongiform changes in the brain, and has been reported in mule deer, white-tailed deer, and elk (Williams and Young, 1982, 1992; Spraker et al., 1997). The known geographic distribution of CWD in free-ranging deer and elk is limited to a relatively small region in north-central Colorado (USA) and southeast Wyoming, where it has been diagnosed in captive and free-ranging mule deer and elk (Williams and Young; 1982, 1992; Spraker et al., 1997) as well as free-ranging white-tailed deer (Spraker et al., 1997). Because of the long incubation period and current lack of diagnostic tests, there is no effective way to identify infected animals while alive. Chronic wasting disease has been diagnosed in captive elk in Saskatchewan, (Canada) and in the USA in Oklahoma, Montana (A. Jenny, pers. comm.), South Dakota, Nebraska (Nettles and Petty, 1998) and Colorado (Williams and Young, 1992). This disease has a history of being moved via relocation of captive animals and can persist in free-ranging deer.

Brucella abortus is present in free-ranging elk in Idaho (USA) and Montana and in elk and bison in Wyoming in the Greater Yellowstone Area. Serosurveys of female elk on feedgrounds in Wyoming revealed that approximately one-third were test-positive (Thorne et al., 1978; Morton et al., 1981); therefore, it is important to avoid translocation of elk with any link to the infected free-ranging elk herds in the Greater Yellowstone Area. Outside of the Greater Yellowstone Area, free-ranging elk do not appear to be infected with brucellosis (Adrian and Keiss, 1977; McCorquodale and DiGiacomo, 1985).

Mycobacterium bovis has been the subject of a long-term national eradication program that has nearly eradicated bovine tuberculosis (TB) among cattle, and most states are accredited TB-free. However, for over a decade, problems have occurred with TB among captive ungulates such as bison, elk, red deer, and fallow deer (*Dama dama*), and twice there have been set-backs in the TB-free status for cattle that were attributed to infection from captive cervids. Between 1991–1996, TB occurred in 31 herds of captive elk and deer in 15 states (VanTiem and Essey, 1996), and in additional herds in several Canadian provinces (Hillman and Thompson, 1996). In general, native cervids in the USA have remained free of TB; however, in 1993 TB was found in a mule deer collected on property adjacent to an infected game ranch in Montana (Rhyan et al., 1995). Furthermore, TB was discovered in a white-tailed deer population in Michigan (USA) in 1994 (Nettles and Petty, 1996; Schmitt et al., 1997). As of 24 February 1998, subsequent surveillance had disclosed 149 infected deer and three infected coyotes in a 5-county area (S. Schmitt, pers. comm.). The concern about TB in free-ranging elk is based on the past history of infection in captive cervids and the serious consequences if TB becomes established in free-ranging ruminants.

Demacentor andersoni and *I. pacificus* are categorized as high risk because they are not endemic in the southeastern USA (Bishopp and Trembley, 1945) and are notable relative to disease transmission. *Dermacentor andersoni* is a vector of *A. marginale* (Strickland et al., 1976; Stiller et al., 1981) and the agents of tularemia and Colorado tick fever (Thorne et al., 1982). *Dermacentor andersoni* also is a primary vector of *Rickettsia rickettsii,* the agent of Rocky Mountain spotted fever in humans and dogs. In addition, *D. andersoni* is a significant cause of tick paralysis (Strick-

land et al., 1976). *Ixodes pacificus* is the vector of the agent of Lyme disease in western North America (Lane et al., 1991) and can cause tick paralysis (Gothe and Neitz, 1991).

Infestations by mites of the genus *Psoroptes* have been severe enough to cause the death of elk (Murie, 1951; Colwell and Dunlap, 1975; Franson and Smith, 1988; Samuel et al., 1991). The taxonomy of this genus is in question; however, *Psoroptes* sp. apparently is a host-specific parasite of elk and possibly bighorn sheep (*Ovis canadensis*) (Hepworth and Thomas, 1962; Worley et al., 1969; Kistner, 1982; Samuel et al., 1991), and would not be beneficial to future elk populations.

"Reverse risk" disease agents

The most notable "reverse risk" disease agent is the neurotropic nematode *Parelaphostrongylus tenuis.* This helminth, known as the meningeal worm, is harbored asymptomatically by white-tailed deer throughout the southeastern USA except in the lower Coastal Plain. Infection in elk can result in serious neurologic damage when the nematodes migrate through the spinal cord and brain (Samuel et al., 1992). Not all elk succumb to infection; however, multiple observations of fatal neurologic disease in elk and red deer have been reported (Carpenter et al., 1973; Woolf et al., 1977; Olsen and Woolf, 1978, 1979). Several confirmed cases have occurred in elk reintroduced into Arkansas (SCWDS, unpubl. data). The ultimate impact of meningeal worm on introduced elk may depend upon the degree to which elk use the habitat occupied by the gastropod intermediate hosts of *P. tenuis* (Raskevitz et al., 1991).

Another nematode pathogenic to elk is the arterial worm *Elaeophora schneideri.* This nematode is well documented in the southwestern USA where mule deer serve as normal, asymptomatic hosts (Hibler, 1981). Elk are not the normal host for *E. schneideri;* however, infection may result in occlusion of arterial vessels and impaired blood supply to the neck and head and subsequent blindness, neurologic deficits, and avascular necrosis of the ears and muzzle (Adcock and Hibler, 1969). White-tailed deer in the southeastern USA can be infected with *E. schneideri*; such infections are not common but appear to be prevalent in Arkansas (USA), Louisiana (USA), and certain areas on the Atlantic and Gulf Coasts (Couvillion et al., 1985; SCWDS, unpubl. data).

There is one report of mortality due to *Babesia* sp. in yearling elk imported into Texas (Holman et al., 1994). The identification of the protozoan parasite was incomplete, but it was noted that the parasite resembled *Babesia odocoilei. Babesia odocoilei* is a parasite of white-tailed deer that probably is endemic to the southeastern USA. Reports of *B. odocoilei* are scattered through a wide area of the USA and include Florida, New Mexico, Oklahoma, South Carolina, Texas, and Virginia (Kingston, 1981; Perry et al., 1985; Waldrup et al., 1989; Forrester, 1992; SCWDS, unpubl. data). Furthermore, studies have revealed that the black-legged tick (*I. scapularis*) is the probable vector (Waldrup et al., 1990, 1992). This tick is widespread on white-tailed deer in the southeastern USA, thus, it is likely that elk will become exposed to *B. odocoilei.* It is not known if *B. odocoilei* could be a threat to elk because the taxonomic status of *Babesia* in cervids is unclear and the isolate from the elk may not have been *B. odocoilei* (Holman et al., 1994).

MANAGEMENT IMPLICATIONS

General

We propose a five-component protocol to reduce the risk of introduction of the infectious agents and ectoparasites addressed. This protocol includes (1) evaluation of the health status of the source population, (2) quarantines, (3) physical examination and diagnostic testing, (4) restrictions on translocation of animals from certain geographic areas or populations, and (5) prophylactic treatment. Inclusion

of each of the five components in a specific translocation would depend on what infectious agents or ectoparasites were considered high risk.

Evaluation of the health status of the source population

We commend that the recipient state fish and wildlife agency and state veterinarian obtain and jointly review all available information pertaining to the health of the source elk population. Historical information would include published literature and records from the state fish and wildlife agency, state departments of agriculture, diagnostic laboratories, and colleges of veterinary medicine. Consultation with the appropriate agriculture and/or wildlife agencies, including the state veterinarian's office in the source state, should be sought to determine if any of the agents of concern are known to be endemic in elk, other wildlife, or domestic animals in the area.

Prior to relocation of elk from a source population, it is highly desirable to sample the population by serology and necropsy. Serums should be tested for bovine brucellosis and anaplasmosis. Serologic tests for other diseases (e.g., bluetongue, bovine virus diarrhea, epizootic hemorrhagic disease, infectious bovine rhinotracheitis, leptospirosis, and paratuberculosis) are desirable; however, positive test results may not equate to serious consequences in regard to relocation of the elk. Serologic evaluation of an elk population will be most useful if large numbers of animals are monitored over a prolonged period. Positive serologic results may warrant further investigation for the presence of active infection. Hunter-harvested elk or roadkills could be used if it is not possible to kill elk for necropsy. Necropsies should include examination and/or tests for *M. bovis, M. paratuberculosis, E. granulosus, D. dendriticum, F. magna, D. albipictus, D. andersoni, I. pacificus, O. megnini, Psoroptes* sp., and CWD.

Quarantines

Guidelines for the proposed quarantines are linked to testing requirements for brucellosis and bovine tuberculosis (United States Department of Agriculture, 1997, 1998). Fulfillment of these guidelines, which were written for farm- or ranch-raised cervidae, require that animals with no prior testing history be held in the state of origin in isolation from all other members of the herd for at least 93 days. This minimum figure accounts for the required 90 days between two tuberculosis tests and an additional three days for reading of the second test. Test requirements for brucellosis, which require a negative test within 30 days prior to shipment, could be satisfied within the same time frame. An additional negative brucellosis test made after 90 days post-entry is strongly recommended. Thus, strict complete compliance to both brucellosis and tuberculosis program standards would require holding captured elk for 93 days before shipping and ≥90 days post-entry in the recipient state. Such measures will require holding enclosures.

We recommend that effort be directed toward testing of source elk populations such that they can be afforded a status equivalent to a monitored herd as described in the Cervidae program standards (see Physical Examination and Diagnostic Testing below). This would eliminate the need for quarantines and allow the elk to move with only one test. However, if these measures are not taken, we recommend a 93-day quarantine period, either pre- or post-entry. This will allow time for the animals to be tested twice for brucellosis and tuberculosis and observed for any evidence of disease.

Physical examination and diagnostic testing

Each group of elk should be inspected by a licensed and accredited veterinarian in the state of origin for visible conditions including abnormal swellings, areas of hair loss, inappetence, emaciation, diarrhea, lack of awareness, excess salivation, poly-

dipsia, polyuria, lameness, and traumatic injuries. All shipments should be accompanied by a Certificate of Veterinary Inspection signed by the accredited veterinarian. Each animal should be permanently identified.

Diagnostic tests are not available for CWD in live Cervidae, thus the importing agency must rely on observations of the clinical signs of the disease during quarantine, avoidance of the known distribution of CWD in Cervidae, and data from source population surveys. If tests for live animals become available, they should be incorporated into the protocol.

The current test standards for brucellosis and TB are the Brucellosis in Cervidae: Uniform Methods and Rules (United States Department of Agriculture, 1998) and the Draft Tuberculosis Eradication in Cervidae Uniform Methods and Rules (United States Department of Agriculture, 1997). The aforementioned testing and quarantine guidelines for brucellosis and TB were developed for captive Cervidae thus we propose two options for free-ranging elk.

Option 1: Reasonable assurance of freedom from brucellosis and TB could be obtained by the following protocol: (1) all sexually intact elk six months of age or older must test negative for brucellosis and TB within 30 days prior to shipment; (2) none of the elk in the group can be shipped if any test-positive or test-suspects for either disease are found: (3) elk are held in isolation in a fenced area for at least 90 days; and (4) elk are retested negative for brucellosis and TB after 90 days.

Option 2: As an alternative, we recommend developing a quasi-monitored herd status whereby source elk herds are chosen for their geographic isolation and portions of the herd are tested for brucellosis and TB as per the above guidelines (United States Department of Agriculture, 1997, 1998). Sample sizes would be based on the estimated elk population and be sufficient to detect a 2% prevalence in the herd at a 95% confidence level (United States Department of Agriculture, 1997). Brucellosis testing could be by serology and culture. Tuberculosis testing could be by skin testing, but it would be more practical to use necropsy with appropriate follow-through of lesions (histopathology and, if histologically compatible, culture). Results would have to be negative. Elk from such herds could be moved with one negative test for brucellosis and TB from the animals to be moved (United States Department of Agriculture, 1997, 1998).

Restrictions on origin

Elk should not be translocated from geographic areas or populations currently known to harbor elk with CWD, *B. abortus, M. bovis, M. paratuberculosis, E. cervi,* or a septicemic form of *P. multocida.* Designation of such areas and their size will depend on the status of elk in the area at the time such movement is proposed and can be determined through monitoring the health status of source populations as described above and through consultation between the state veterinarians and state fish and game agencies of the affected states.

Prophylactic treatment

A method to eliminate the risk of parasite introduction is to remove the agents with parasiticides. Unfortunately, with rare exceptions, information on use of drugs in elk is not available, and the administration of pesticides and anthelminthics to elk must be based on extrapolation from their use on other ruminant species for comparable parasite infestations and infections. We would strongly encourage the use of parasiticides in elk prior to translocation because information in the literature suggests that certain drugs should be efficacious. When animals are held in quarantine, multiple treatments are desirable. However, due to limited first-hand experience with these products in elk, we cannot provide recommendations for drugs or dosages. There are no parasiticides approved for use in elk per se, but

under the provisions of the Animal Medical Drug Use Clarification Act (AMDUCA), veterinarians have the latitude to prescribe off-label treatments with licensed products.

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LITERATURE CITED

ADCOCK, J. L., AND C. P. HIBLER. 1969. Vascular and neuro-ophthalmic pathology of elaeophorosis in elk. Veterinary Pathology 6: 185–213.

ADRIAN, W. J., AND R. E. KEISS. 1977. Survey of Colorado's wild ruminants for serologic titers to brucellosis and leptospirosis. Journal of Wildlife Diseases 13: 429–431.

ANDREWS, J. R. H. 1973. A host-parasite checklist of helminths of wild ruminants in New Zealand. New Zealand Veterinary Journal 21: 43–47.

BASSI, R. 1893. *Distomum magnum* (Bassi) in Italia ed in America. Il Moderno Zooiatro 4: 269–270.

BENDER, L. C., AND P. B. HALL. 1996. *Leptospira interrogans* exposure in free-ranging elk in Washington. Journal of Wildlife Diseases 32: 121–124.

BISHOPP, F. C., AND H. L. TREMBLEY. 1945. Distribution and hosts of certain North American ticks. The Journal of Parasitology 31: 1–54.

BOWMAN, D. D. 1995. Georgis' parasitology for veterinarians, 6th Edition. W. B. Saunders Company, Philadelphia, Pennsylvania, 430 pp.

BRYANT, L. D. , AND C. MASER. 1982. Classification and distribution. *In* Elk of North America, J. W. Thomas and D. E. Toweill (eds.). Stackpole Books, Harrisburg, Pennsylvania, pp. 1–59.

CARPENTER, J. W., H. E. JORDON AND B. C. WARD. 1973. Neurologic disease in wapiti naturally infected with meningeal worms. Journal of Wildlife Diseases 9: 148–153.

CHOQUETTE, L. P. E., AND E. BROUGHTON. 1981. Anthrax. *In* Infectious diseases of wild mammals, 2nd Edition, J. W. Davis, L. H. Karstad, and D. O. Trainer (eds.). The Iowa State University Press, Ames, Iowa, pp. 288–296.

COLWELL, D. A., AND J. S. DUNLAP. 1975. Psoroptic mange in a wapiti. Journal of Wildlife Diseases 11: 66–67.

COOK, W. E., T. E. CORNISH, S. SHIDELER, B. LASLEY, AND M. T. COLLINS. 1997. Radiometric culture of *Mycobacterium avium paratuberculosis* from the feces of tule elk. Journal of Wildlife Diseases 33: 635–637.

COUVILLION, C. E., W. R. DAVIDSON, AND V. F. NETTLES. 1985. Distribution of *Elaephora schneideri* in white-tailed deer in the southeastern United States, 1962–1983. Journal of Wildlife Diseases 21: 451–453.

COWAN, I. M. 1951. The diseases and parasites of big game mammals of western Canada. British Columbia Game Convention 5: 37–64.

DAVIDSON, W. R., AND V. F. NETTLES. 1992. Relocation of wildlife: Identifying and evaluating disease risks. Transactions of the 57th North American Wildlife and Natural Resources Conference 57: 466–473.

DAVIS, J. W., AND K. G. LIBKE. 1971. Trematodes. *In* Parasitic diseases of wild mammals, J. W. Davis and R. C. Anderson (eds.). The Iowa State University Press, Ames, Iowa, pp. 235–257.

DE LISLE, G. W., G. F. YATES, AND D. M. COLLINS. 1993. Paratuberculosis in farmed deer: case reports and DNA characterization of isolates of *Mycobacterium paratuberculosis.* Journal of Veterinary Diagnostic Investigation 5: 567–571.

DOWNING, R. L. 1987. Success story: White-tailed deer. *In* Restoring America's wildlife, H. Kallman, C. P. Agee, W. R. Goforth, and J. P. Linduska (eds.). U. S. Department of the Interior, Fish and Wildlife Service, Washington, D. C., pp. 45–57.

ELDRIDGE, B. F., C. H. CALISHER, J. L. FRYER, L. BRIGHT, AND D. J. HOBBS. 1987. Serological evidence of California serogroup virus activity in Oregon. Journal of Wildlife Diseases 23: 199–204.

FORRESTER, D. J. 1992. Parasites and diseases of wild mammals in Florida. University Press of Florida, Gainesville, Florida, 459 pp.

FRANSON, J. C., AND B. L. SMITH. 1988. Septicemic pasteurellosis in elk (*Cervus elaphus*) on the United States National Elk Refuge, Wyoming. Journal of Wildlife Diseases 24: 715–717.

GAJADHAR, A., AND S. V. TESSARO. 1995. Susceptibility of mule deer (*Odocoileus hemionus*) and two species of North American molluscs to *Elaphostrongylus cervi* (Nematoda: Metastrongyloidea). The Journal of Parasitology 81: 593–596.

———, ———, AND W. D. YATES. 1994. Diagnosis of *Elaphostrongylus cervi* infection in New Zealand red deer (*Cervus elaphus*) quarantined in Canada, and experimental determination of a new extended prepatent period. Canadian Veterinary Journal 35: 433–437.

GOTHE, R., AND A. W. J. NEITZ. 1991. Tick paralyses: Pathogenesis and etiology. *In* Advances in disease vector research, Vol. 8, K. F. Harris (ed.). Springer-Verlag, New York, New York, pp. 177–204.

HEPWORTH, W. G., AND G. M. THOMAS. 1962. Attempts to transfer psoroptic mites from elk to cattle and sheep. Journal of the American Veterinary Medical Association 140: 689–690.

HIBLER, C. P. 1981. Diseases. *In* Mule and blacktailed deer of North America, O. C. Wallmo (ed.). University of Nebraska Press, Lincoln, Nebraska, pp. 129–155.

HILLMAN, B. R., AND D. L. THOMPSON. 1996. Report of the committee on tuberculosis. Proceedings of the one-hundredth annual meeting of the United States Animal Health Association 100: 616–624.

HOFF, G. L., AND D. O. TRAINER. 1978. Bluetongue and epizootic hemorrhagic disease viruses: Their relationship to wildlife species. Advances in Veterinary Science and Comparative Medicine 22: 111–132.

HOLMAN, P. J., T. M. CRAIG, D. L. DOAN CRIDER, K. R. PETRINI, AND G. G. WAGNER. 1994. Culture and isolation and partial characterization of a *Babesia* sp. from a North American elk (*Cervus elaphus*). Journal of Wildlife Diseases 30: 460–465.

HOWE, D. L., AND W. G. HEPWORTH. 1965. Anaplasmosis in big game animals: Tests on wild populations in Wyoming. American Journal of Veterinary Research 26: 1114–1120.

———, ———, F. M. BLUNT, AND G. M. THOMAS. 1964. Anaplasmosis in big game animals: Experimental infection and evaluation of serologic tests. American Journal of Veterinary Research 25: 1271–1275.

JESSUP, D. A., B. ABBAS, AND D. BEHYMER. 1981. Paratuberculosis in tule elk in California. Journal of the American Veterinary Medical Association 179: 1252–1254.

KINGSCOTE, B. F., W. D. G. YATES, AND G. B. TIFFIN. 1987. Diseases of wapiti utilizing cattle range in southwestern Alberta. Journal of Wildlife Diseases 23: 86–91.

KINGSTON, N. 1981. Protozoan parasites. *In* Diseases and parasites of white-tailed deer, W. R. Davidson, F. A. Hayes, V. F. Nettles, and F. E. Kellogg, (eds.) Tall Timbers Research Station Miscellaneous Publication Number 7, Tallahassee, Florida, pp. 193–236.

KISTNER, T. P. 1982. Diseases and parasites. *In* Elk of North America, J. W. Thomas and D. E. Toweill, (eds.). Stackpole Books, Harrisburg, Pennsylvania, pp. 181–217.

KUTTLER, K. L. 1984. *Anaplasma* infections in wild and domestic ruminants: A review. Journal of Wildlife Diseases 20: 12–20.

LANE, R. S., J. PIESMAN, AND W. BURGDORFER. 1991. Lyme borreliosis: Relation of its causative agent to its vectors and hosts in North America and Europe. Annual Review of Entomology 36: 587–609.

LANKESTER, M. W., AND D. FONG. 1989. Distribution of Elaphostrongyline nematodes (Metastrongyloidea: Protostrongylidae) in Cervidae and possible effects of moving *Rangifer* spp. into and within North America. Alces 25: 133–145.

LEWIS, J. B. 1987. Success story: Wild turkey. *In* Restoring America's Wildlife, H. Kallman, C. P. Agee, W. R. Goforth, and J. P. Linduska, (eds.). U. S. Department of the Interior, Fish and Wildlife Service, Washington, D. C., pp. 31–43.

LOVE, B. I. 1955. Personal observation in the care and management of an elk herd (wapiti) at Elk Island National Park, Alberta, Canada. Canadian Journal of Comparative Medicine 19: 184–192.

MALONE, J. B. 1986. Fascioliasis and cestodiasis in cattle. Veterinary clinics of North America: Food Animal Practice 2: 261–275.

MAPES, C. R. 1951. Studies on the biology of *Dicrocoelium dendriticum* (Rudolphi, 1819) Looss, 1899 (Trematoda: Dicrocoeliidae), including its relation to the intermediate host, *Cionella lubrica* (Muller). I. A study of *Dicrocoelium dendriticum* and *Dicrocoelium* infection. Cornell Veterinarian 41: 382–432.

———, AND D. W. BAKER. 1950. The white-tailed deer, a new host of *Dicrocoelium dendriticum* (Rudolphi, 1819) Looss, 1899 (Trematoda: Dicrocoeliidae). Cornell Veterinarian 40: 211–212.

MCCORQUODALE, S. M., AND R. F. DIGIACOMO. 1985. The role of wild North American ungulates in the epidemiology of bovine brucellosis: A review. Journal of Wildlife Diseases 21: 351–357.

MERRELL, C. L., AND D. N. WRIGHT. 1978. A serologic survey of mule deer and elk in Utah. Journal of Wildlife Diseases 14: 471–478.

MORTON, J. K., E. T. THORNE, AND G. M. THOMAS. 1981. Brucellosis in elk III. Serologic evaluation. Journal of Wildlife Diseases 17: 23–31.

MURIE, O. J. 1951. The elk of North America. Stackpole Company, Harrisburg, Pennsylvania, 376 pp.

NETTLES, V. F., AND S. PETTY. 1996. Report of the Committee on Wildlife Diseases. Proceedings of the annual meeting of the United States Animal Health Association 100: 660–667.

———, AND ———. 1998. Report of the Committee on Wildlife Diseases. Proceedings of the annual meeting of the United States Animal Health Association 102: 728–738.

NIELSEN, L., AND R. D. BROWN. 1988. Translocation

of wild animals. Wisconsin Humane Society, Inc., Milwaukee, Wisconsin and Caesar Kleberg Wildlife Research Institute, Kingsville, Texas, 333 pp.

OLSEN, A., AND A WOOLF. 1978. The development of clinical signs and the population significance of neurologic disease in a captive wapiti herd. Journal of Wildlife Diseases 14: 263–268.

———, AND ———. 1979. A summary of the prevalence of *Parelaphostrongylus tenuis* in a captive wapiti population. Journal of Wildlife Diseases 15: 33–35.

PERRY, B. D., D. K. NICHOLS, AND E. S. CULLOM. 1985. *Babesia odocoilei* Emerson and Wright, 1970 in white-tailed deer, *Odocoileus virginianus* (Zimmermann), in Virginia. Journal of Wildlife Diseases 21: 149–152.

PURSGLOVE, S. R., A. K. PRESTWOOD, T. R. RIDGEWAY, AND F. A. HAYES, 1977. *Fascioloides magna* infection in white-tailed deer of southeastern United States. Journal of the American Veterinary Medical Association 171: 936–938.

PYBUS, M. J. 1990. Survey of hepatic and publmonary helminths of wild cervids in Alberta, Canada. Journal of Wildlife Diseases 26: 453–459.

———, D. K. ONDERKA, AND N. COOL. 1991. Efficacy of triclabendazole against natural infections of *Fascioloides magna* in wapiti. Journal of Wildlife Diseases 27: 599–605.

RASKEVITZ, R. F., A. A. KOCAN, AND J. H. SHAW. 1991. Gastropod availability and habitat utilization by wapiti and white-tailed deer sympatric on range enzootic for meningeal worm. Journal of Wildlife Diseases 27: 92–101.

RENSHAW, H. W., R. A. MAGONIGLE, AND H. W. VAUGHN. 1979. Evaluation of the anaplasmosis rapid card agglutination test for detecting experimentally-infected elk. Journal of Wildlife Diseases 15: 379–386.

RHYAN, J. C., K. AUNE, D. R. EWALT, J. MARQUARDT, J. W. MERTINS, J. B. PAYEUR, D. A. SAARI, P. SCHLADWEILER, E. J. SHEEHAN, AND D. WORLEY. 1997. Survey of free-ranging elk from Wyoming and Montana for selected pathogens. Journal of Wildlife Diseases 33: 290–298.

———, ———, B. HOOD, R. CLARKE, J. PAYEUR, J. JARNAGIN, AND L. STACKHOUSE. 1995. Bovine tuberculosis in a free ranging mule deer (*Odocoileus hemionus*) from Montana. Journal of Wildlife Diseases 31: 432–435.

RICKARD, L. G., E. P. HOBERG, N. M. ALLEN, G. L. ZIMMERMAN, AND T. M. CRAIG. 1993. *Spiculopteragia spiculoptera* and *S. assymmetrica* (Nematoda: Trichostrongylidae) from red deer (*Cervus elaphus*) in Texas. Journal of Wildlife Diseases 29: 512–515.

RIEMANN, H., M. R. ZAMAN, R. RUPPANNER, O. AALUND, J. B. JORGENSEN, H. WORSAAE, AND D. BEHYMER. 1979. Paratuberculosis in cattle and free-living exotic deer. Journal of the American Veterinary Medical Association 174: 814–843.

SAMUEL, W. M., D. A. WELCH, AND B. L. SMITH. 1991. Ectoparasites from elk (*cervus elaphus nelsoni*) from Wyoming. Journal of Wildlife Diseases 27: 446–451.

———, M. J. PYBUS, D. A. WELCH, AND C. J. WILKE. 1992. Elk as a potential host for meningeal worm: Implications for translocation. The Journal of Wildlife Management 56: 629–639.

SCHAFFER, G. D., W. R. DAVIDSON, V. F. NETTLES, AND E. A. ROLLOR. 1981. Helminth parasites of translocated raccoons (*Procyon lotor*) in the southeastern United States. Journal of Wildlife Diseases 17: 217–227.

SCHANTZ, P. M., J. CHAI, P. S. CRAIG, J. ECKERT, D. J. JENKINS, C. N. L. MACPHERSON, AND A. THAKUR. 1995. Epidemiology and control of hydatid disease. *In Echinococcus* and hydatid disease, R. C. A. Thompson and A. J. Lymbery (eds.). CAB International, Wallington, United Kingdom, pp. 233–331.

SCHMITT, S. M., S. D. FITZGERALD, T. M. COOLEY, C. S. BRUNING-FANN, L. SULLIVAN, D. BERRY, T. CARLSON, R. B. MINNIS, J. B. PAYEUR, AND J. SIKARSKIE. 1997. Bovine tuberculosis in free-ranging white-tailed deer from Michigan. Journal of Wildlife Diseases 33: 749–758.

SCHULTE, J. W., W. D. KLIMSTRA, AND W. G. DYER, 1976. Protozoan and helminth parasites of Key deer. The Journal of Wildlife Management 40: 579–581.

SMITS, J. E. G. 1991. A brief review of infectious and parasitic diseases of wapiti, with emphasis on western Canada and the northwestern United States. Canadian Veterinary Journal 32: 471–479.

———, 1992. Elk disease survey in Western Canada and the Northwestern United States. *In* The biology of deer, R. D. Brown (ed.). Proceedings of the second international symposium on the biology of deer. Springer-Verlag, New York, New York, pp. 101–106.

SPRAKER, T. R., M. W. MILLER, E. S. WILLIAMS, D. M. GETZY, W. J. ADRIAN, G. G. SCHOONVELD, R. A. SPOWART, K. I. O'ROURKE, J. M. MILLER, AND P. A. MERZ. 1997. Spongiform encephalopathy in free-ranging mule deer (*Odocoileus hemionus*), white-tailed deer (*Odocoileus virginianus*) and Rocky Mountain elk (*Cervus elaphus nelsoni*) in northcentral Colorado. Journal of Wildlife Diseases 33: 1–6.

STILLER, D., G. LEATCH, AND K. L. KUTTLER. 1981. *Dermacentor albipictus* (Packard): An experimental vector of bovine anaplasmosis. Proceedings of the annual meeting of the United States Animal Health Association 85: 65–73.

STRICKLAND, R. K., R. R. GERRISH, J. L. HOURRIGAN, AND G. O. SCHUBERT. 1976. Ticks of veterinary importance. United States Department of Agriculture, Animal and Plant Health Inspection Service, Agricultural handbook Number 485, Washington, D. C., 122 pp.

SUAREZ, V. H., M. R. BUSETTI, M. C. FORT, AND D. O. BEDOTTI. 1991. *Spiculopteragia spiculoptera, S. assymetrica* and *Ostertagia leptospicularis* from *Cervus elaphus* in La Pampa, Argentina. Veterinary Parasitology 40: 165–168.

TAREZY'NSKI, S. 1954. *Wehrdikmansia cervipedis* (Wehr et Dikmans, 1935) Caballero, 1945, pasozytem jelenia *Cervus elaphus* L. Acta Parasitologica Polonica 2: 209–222. (English and Russian summaries.)

THOMPSON, R. C. A. 1995. Biology and systematics of *Echinococcus. In Echinococcus* and hydatid disease, R. C. A. Thompson and A. J. Lymbery, (eds.). CAB International, Wallington, United Kingdom, pp. 1–37.

THORNE, E. T., J. K. MORTON, AND G. M. THOMAS. 1978. Brucellosis in elk I. Serologic and bacteriologic survey in Wyoming. Journal of Wildlife Diseases 14: 78–81.

———, N. KINGSTON, W. R. JOLLEY, AND R. C. BERGSTROM. 1982. Diseases of wildlife in Wyoming, 2nd Edition. Wyoming Game and Fish Department, Cheyenne, Wyoming, 353 pp.

UNITED STATES ANIMAL HEALTH ASSOCIATION. 1992. Hemorrhagic septicemia. *In* Foreign animal diseases. United States Animal Health Association, Richmond, Virginia, pp. 229–235.

UNITED STATES DEPARTMENT OF AGRICULTURE. 1997. Draft tuberculosis eradication in Cervidae uniform methods and rules. Animal and Plant Health Inspection Service, Washington, D. C., 22 pp.

———. 1998. Brucellosis in Cervidae: Uniform methods and rules. Animal and Plant Health Inspection Service, Washington, D. C., 23 pp.

VANCE, H. N. 1961. Johne's disease in a European red deer. Canadian Veterinary Journal 8: 305–307.

VANTIEM, J. S., AND M. A. ESSEY. 1996. Status of the State-Federal Tuberculosis Eradication Program: Fiscal year 1996. Proceedings of the annual meeting of the United States Animal Health Association 100: 637–652.

VAUGHN, H. W., H. W. RENSHAW, AND F. W. FRANK. 1976. Survey of anaplasmosis in elk of the Clearwater National Forest (Idaho). American Journal of Veterinary Research 37: 615–617.

WALDRUP, K. A., A. A. KOCAN, R. W. BARKER, AND G. G. WAGNER. 1990. Transmission of *Babesia odocoilei* in white-tailed deer (*Odocoileus virginianus*) by *Ixodes scapularis* (Acari: Ixodidae). Journal of Wildlife Diseases 26: 390–391.

———, A. A. KOCAN, T. QURESHI, D. S. DAVIS, D. BAGGETT, AND G. G. WAGNER. 1989. Serological prevalence and isolation of *Babesia odocoilei* among white-tailed deer (*Odocoileus virginianus*) in Texas and Oklahoma. Journal of Wildlife Diseases 25: 195–201.

———, J. MORITZ, D. BAGGETT, S. MAGYAR, AND G. G. WAGNER. 1992. Monthly incidence of *Theileria cervi* and seroconversion to *Babesia odocoilei* in white-tailed deer (*Odocoileus virginianus*) in Texas. Journal of Wildlife Diseases 28: 457–459.

WEBB, P. A., R. G. MCLEAN, G. C. SMITH, J. H. ELLENBERGER, D. B. FRANCY, T. E. WALTON, AND T. P. MONATH. 1987. Epizootic vesicular stomatitis in Colorado, 1982: Some observations on the possible role of wildlife populations in an enzootic maintenance cycle. Journal of Wildlife Diseases 23: 192–198.

WILLIAMS, E. S., S. P. SNYDER, AND K. L. MARTIN. 1983. Experimental infection of some North American wild ruminants and domestic sheep with *Mycobacterium paratuberculosis*: Clinical and bacteriological findings. Journal of Wildlife Diseases 19: 185–191.

———, AND S. YOUNG. 1982. Spongiform encephalopathy of Rocky Mountain elk. Journal of Wildlife Diseases 18: 465–471.

———, AND ———. 1992. Spongiform encephalopathies in Cervidae. Revue Scientifique et Technique Office International des Epizooties 11: 551–567.

WOOLF, A., C. A. MASON, AND D. KRADEL. 1977. Prevalence and effects of *Parelaphostrongylus tenuis* in a captive wapiti population. Journal of Wildlife Diseases 13: 149–154.

WORLEY, D. E., R. E. BARRETT, P. J. A. PRESIDENTE, AND R. H. JACOBSON. 1969. The Rocky Mountain elk as a reservoir host for parasites of domestic animals in western Montana. Bulletin of the Wildlife Disease Association 5: 348–350.

ZAUGG, J. L., W. L. GOFF, W. FOREYT, AND D. L. HUNTER. 1996. Susceptibility of elk (*Cervus elaphus*) to experimental infection with *Anaplasma marginale* and *A. ovis.* Journal of Wildlife diseases 32: 62–66.

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