DISEASES AND PARASITES OF RED FOXES, GRAY FOXES, AND COYOTES FROM COMMERCIAL SOURCES SELLING TO FOX-CHASING ENCLOSURES

Authors: Davidson, William R., Appel, Max J., Doster, Gary L., Baker, Osborne E., and Brown, John F.

Source: Journal of Wildlife Diseases, 28(4) : 581-589

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-28.4.581

Journal of Wildlife Diseases, 28(4), 1992, pp. 581–589

DISEASES AND PARASITES OF RED FOXES, GRAY FOXES, AND COYOTES FROM COMMERCIAL SOURCES SELLING TO FOX-CHASING ENCLOSURES

William R. Davidson,[1] Max J. Appel,[2] Gary L. Doster,[3] Osborne E. Baker,[4] and John F. Brown[5]

[1] School of Forest Resources and Southeastern Cooperative Wildlife Disease Study, Department of Parasitology, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602 USA
[2] James A. Baker Institute for Animal Health, New York State College of Veterinary Medicine, Cornell University, Ithaca, New York 14853 USA
[3] Southeastern Cooperative Wildlife Disease Study, Department of Parasitology, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602 USA
[4] South Carolina Wildlife and Marine Resources Department, Post Office Box 167, Columbia, South Carolina 29202 USA
[5] South Carolina Department of Health and Environmental Control, 2600 Bull Street, Columbia, South Carolina 29201 USA

ABSTRACT: Fifty-six red foxes (*Vulpes vulpes*), 18 gray foxes (*Urocyon cinereoargenteus*), and 13 coyotes (*Canis latrans*) obtained by the South Carolina Wildlife and Marine Resources Department during an investigation of suspected illegal wildlife translocation were examined for diseases and parasites. Red foxes and coyotes were confiscated from an animal dealer based in Ohio (USA), and gray foxes were purchased from an animal dealer in Indiana (USA). Emphasis was placed on detection of pathogens representing potential health risks to native wildlife, domestic animals, or humans. All animals were negative for rabies; however, 15 gray foxes were incubating canine distemper at necropsy. Serologic tests disclosed antibodies to canine parvovirus, canine distemper virus, canine adenovirus, canine coronavirus, canine herpesvirus, and canine parainfluenza virus in one or more host species. Twenty-three species of parasites (two protozoans, three trematodes, four cestodes, eleven nematodes, and three arthropods) were found, including species with substantial pathogenic capabilities. *Echinococcus multilocularis*, a recognized human pathogen not enzootic in the southeastern United States, was found in red foxes. Based on this information, we conclude that the increasingly common practice of wild canid translocation for stocking fox-chasing enclosures poses potential health risks to indigenous wildlife, domestic animals, and humans and, therefore, is biologically hazardous.

Key words: Infectious diseases, parasitism, *Echinococcus multilocularis*, red fox, *Vulpes vulpes*, gray fox, *Urocyon cinereoargenteus*, coyotes, *Canis latrans*, host translocation, biological risks.

INTRODUCTION

Recreational pursuit of foxes with hounds, commonly termed fox hunting, has been a popular tradition in many regions of the United States since colonial times. Over time, fox hunting in the United States has evolved into two different forms. One is the formal European "redcoat" style hunt with horse-mounted participants using large packs of hounds. The second is a less formal style of hunting that does not involve horses and is conducted by individuals or small groups with their own hounds. The latter type currently is more widely practiced in the United States and often is done at night. Participants in this style of fox hunting have an organized system of field trial competitions under the auspices of state and national organizations.

Fox hunting of either style has certain requirements for its conduct. Tracts of land that are of adequate size and suitable for both habitation by foxes and pursuit by hounds must be available. With changes in land ownership patterns, such areas have diminished greatly. Fragmentation of properties into smaller ownerships, with the resultant problem of trespass by hounds on posted land, has prompted houndsmen to seek alternatives in order to preserve their sport. A relatively recent, but increasingly frequent, practice is the con-

struction of large enclosures in which foxes may be pursued by hounds. These fox-chasing enclosures, commonly termed "fox pens," have the advantages of facilitating containment and retrieval of hounds, and consequently aid greatly in eliminating conflicts between fox hunters and other landowners.

Within the past decade the number of fox-chasing enclosures in the southeastern United States has increased dramatically. We estimate that there are at least 150 pens in 12 southeastern states. Nearly all enclosure owners periodically stock foxes in their pens. In addition to both red foxes (*Vulpes vulpes*) and gray foxes (*Urocyon cinereoargenteus*), coyotes (*Canis latrans*) are also released in many enclosures. Although fox-chasing enclosures have certain clear benefits, many state wildlife agencies have concerns regarding their operation; a frequent one is that translocation of wild canids is a potential disease risk. Regulations governing the operation of fox-chasing enclosures, including those on sources and species of wild canids stocked and on the amount of health monitoring required, vary among states.

In this report, we present the results of a survey for diseases and parasites among red foxes, gray foxes, and coyotes procured by the South Carolina Wildlife and Marine Resources Department (SCWMRD) during a covert investigation of fox-chasing enclosure operations in that state. The data are evaluated with regard to potential disease risks that may be associated with the current form of private sector translocation and release of wild canids in fox-chasing enclosures.

MATERIALS AND METHODS

Animals examined during this study originated from an 18-month covert investigation of suspected illegal possession and/or importation of foxes and coyotes into South Carolina (USA) conducted by the SCWMRD Division of Law Enforcement. The animals included 56 red foxes, 18 gray foxes, and 13 coyotes.

The red foxes and coyotes were confiscated on 2 December 1989, when the supplier and local purchasers were arrested for violations of animal importation and possession regulations. The state of origin of individual animals was uncertain, although the supplier reported obtaining animals in Ohio (USA) and other nearby states. Reportedly, medications and vaccinations had not been administered. The animals were transported from the supplier's facility in Ohio to South Carolina as a single shipment. During transit the animals were kept in approximately 1 m × 1.5 m × 1 m cages with each cage containing two to five animals segregated by species. Eight red foxes that were dead when seized by SCWMRD were frozen and submitted intact to the Southeastern Cooperative Wildlife Disease Study (SCWDS; College of Veterinary Medicine, The University of Georgia, Athens, Georgia, USA) on 7 December 1989. The remaining 48 red foxes and all 13 coyotes were housed at a SCWMRD facility until 20 December 1989 when they were transported alive to SCWDS. Within the SCWMRD facility, all 13 coyotes were housed together and red foxes were divided equally between two pens.

The gray foxes originated from a group of 20 animals purchased covertly in August 1989 from an animal dealer in Indiana (USA). After purchase they were held in an animal-holding facility belonging to a local trapper. Medications and vaccines were not administered. While housed at the trapper's facilities, two animals died but were not retained. The remaining 18 foxes were submitted alive to SCWDS on 15 February 1990. Locally acquired gray foxes and red foxes, including new animals acquired periodically, also were housed at the trapper's facility but were kept in separate pens.

Because of the large number of animals submitted on relatively short notice, necropsy and diagnostic procedures were designed to detect only those diseases or parasites with wildlife, veterinary, or public health significance, or those easily detected. Attempts were not made to precisely quantify parasites or pathogens considered to have limited pathologic consequence.

Live animals were anesthetized with an intramuscular injection of a mixture of 5 mg/kg ketamine hydrochloride (Ketaset, Bristol Laboratories, Syracuse, New York, USA) and 1 mg/kg xylazine (Rompum, Haver-Lockhart Laboratories, Shawnee, Kansas, USA) and then euthanized with an intracardial injection of sodium pentobarbital (Fatal-plus, Vortech Pharmaceuticals, Dearborn, Michigan USA). While animals were anesthetized, 50-ml samples of whole blood were obtained by cardiac puncture and allowed to clot; the serum was removed and frozen for serologic tests. Thin blood films prepared from EDTA-preserved

(Becton-Dickinson Vacutainer Systems, Rutherford, New Jersey, USA) whole blood were air-dried, fixed in 100% methanol, stained with Giesma, and examined microscopically (100–1000×) for hematozoan parasites. The sex, age, weight, and general physical condition of each animal was recorded, and each animal was assigned an individual identification number. Ages were determined as described by Petrides (1950) for red and gray foxes and by Nellis et al. (1978) for coyotes. Physical condition was evaluated based on the estimation of intraperitoneal fat described by Windberg et al. (1991). Pelage was parted with forceps and brushed with a small brush to recover arthropod parasites; the arthropods were preserved in 70% ethanol for subsequent identification. Scrapings of suspected mange lesions and of ear canals were prepared in immersion oil and examined microscopically to confirm infestations of mites. Each animal was examined visually and by palpation for external injuries or wounds.

Necropsy examinations were conducted as described by Nettles (1981), except that the skin was not removed. Internal organs and other body tissues were examined *in situ* for lesions or abnormalities. One-half of the brain obtained by longitudinal bisection was placed in a plastic container, refrigerated, and tested for rabies within 48 hr using direct immunofluorescent methodology (Dean and Abelseth, 1973). The stomach, small intestine, large intestine, and cecum were excised, and helminth parasites were recovered by the methods of Crum et al. (1978). The lungs, esophagus, and heart were excised and examined without magnification for parasites as described by Nettles (1981). Sections of brain, lymph node, lung, heart, liver, kidney, spleen, adrenal gland, pancreas, stomach, duodenum, jejunum, ileum, large intestine, diaphragm, tongue, masseter muscle, urinary bladder, and any gross lesions were excised, preserved in 10% neutral buffered formalin, embedded in paraffin, sectioned at 6 to 9μ, stained with hematoxylin and eosin, and examined microscopically (40 to 1,000×). Sections of brain, kidney, spleen, liver, mesenteric lymph node, retropharyngeal lymph node, and feces were placed in whirl pacs and frozen for subsequent microbiologic studies. Frozen brain, liver, and spleen from selected red and gray foxes were tested for canine distemper virus using direct fluorescent antibody methodology (Appel and Gillespie, 1972). Sheather's sugar flotations (Levine, 1985) were performed on feces to detect helminth eggs and coccidian oocysts. Serum samples were tested for antibodies to canine parvoviruses using the hemagglutination inhibition test described by Carmichael et al. (1980) and for antibodies to canine distemper virus, canine adenovirus, canine coronavirus, canine herpesvirus, and canine parainfluenza virus (SV-5) using serum neutralization tests described by Appel and Robson (1973). Cell lines for virus neutralization tests included secondary dog kidney cells (canine adenovirus, herpesvirus and parainfluenza virus), the Vero cell line (canine distemper virus), and the NLFK cell line (canine coronavirus). A titer of $\geq$1:10 in all tests would indicate a previous infection with the corresponding virus in dogs. The cut-off point in foxes and coyotes is not known.

Because of the large number of red foxes, complete examinations as described above were restricted to 20 animals. Many of the above procedures, however, were performed on additional red foxes, and sample sizes are specified where appropriate. Representative specimens of parasites recovered were deposited in the U.S. National Parasite Collection, Beltsville, Maryland, under accession numbers 81977 to 81990. The potential health risks posed by the diseases and parasites detected were evaluated by criteria described previously (Schaffer, 1979; Schaffer et al., 1981; Schorr et al., 1988).

RESULTS

Determination of general physical condition was made on 28 red foxes and on all coyotes and gray foxes. The general physical condition of red foxes (14 fair, 14 good) was subjectively rated as poorer than that of coyotes (13 good) or gray foxes (1 fair, 17 good). Red foxes had a higher prevalence of external wounds or injuries (71%) than coyotes (46%) or gray foxes (11%). Externally detectable injuries included both recent and long-standing lacerations, ulcerations, abrasions, bite wounds, bone and tooth fractures, soft tissue infections, pyodermas, and areas of hair loss.

Direct immunofluorescent testing of brain tissue for rabies virus was negative for all 56 red foxes, 13 coyotes, and 18 gray foxes. Visual inspection specifically for oral papillomas was negative for all animals, as were visual examinations at necropsy for lesions that might be attributable to other infectious diseases.

Canine distemper virus infection was diagnosed at necropsy in 15 gray foxes based on histopathology. Intracytoplasmic and/or intranuclear eosinophilic inclusion bodies were found in bronchiolar, bile duct,

TABLE 1. Antibody titers to selected viral diseases in red foxes, coyotes, and gray foxes translocated from the midwest to South Carolina.[a]

Antibody titer	Red foxes	Coyotes	Gray foxes
Canine parvovirus			
Negative	0/48	0/13	2/18
1:20 to 1:80	6/48	1/13	7/18
1:160 to 1:320	18/48	2/13	5/18
1:640 to 1:1,280	16/48	3/13	3/18
1:2,560 to ≥1:10,240	8/48	7/13	1/18
Canine distemper virus			
Negative	36/36	1/7	12/12
1:300 to 1:800	0/36	6/7	0/12
Canine adenovirus			
Negative	0/47	0/13	8/18
1:10 to 1:50	1/47	0/13	2/18
1:100 to 1:500	19/47	6/13	1/18
1:800 to 1:25,000	27/47	7/13	7/18
Canine coronavirus			
Negative	46/46	10/13	4/14
1:10 to 1:50	0/46	3/13	8/14
1:100 to 1:160	0/46	0/13	2/14
Canine herpesvirus			
Negative	38/39	6/13	18/18
1:10 to 1:50	0/39	6/13	0/18
1:160 to 1:300	1/39	1/13	0/18
Canine parainfluenza virus			
Negative	11/42	5/12	17/17
1:10 to 1:50	27/42	7/12	0/17
1:100 to 1:160	4/42	0/12	0/17

[a] Variations in sample size within a species (column) are due to serum toxicity within cell culture systems; these results were omitted.

pancreatic duct, urinary bladder, gastric, and intestinal epithelial cells. Inclusions also were present in mononuclear cells in spleen and lymph nodes and in glial and ependymal cells in the brain. Mild multifocal nonsuppurative encephalitis, mild interstitial pneumonia and bronchitis, splenic hemosiderosis, and hydropic degeneration of hepatocytes were noted in infected foxes. Liver, spleen, or brain tissues from each of five gray foxes diagnosed as having canine distemper based on histopathologic findings were positive for viral antigen on fluorescent antibody tests. Liver, spleen, and brain from three red foxes which had mild nonsuppurative encephalitis, but no inclusion bodies, were negative for canine distemper virus antigen on fluorescent antibody tests.

All three host species had high (>89%) prevalences of antibodies to canine parvovirus and canine adenovirus (Table 1). Coyotes also had high (≥54%) prevalences of antibodies to canine distemper virus, canine herpesvirus, and canine parainfluenza virus. Most (71%) gray foxes had antibodies to canine coronavirus, and most (74%) red foxes had antibodies to canine parainfluenza virus. Antibodies to canine coronavirus in coyotes (23%) and to canine herpesvirus in red foxes (3%) were less frequent. The antibodies detected are indicative of prior exposure to the specific viruses but do not necessarily indicate infection at the time of testing.

Red foxes harbored 17 species of parasites, coyotes had seven species, and gray foxes had 12 species. (Table 2). Parasites detected included *Echinococcus multilocularis* which is not indigenous in the southeastern United States. Values for parasitism prevalence and intensity should be considered minimum estimates because in some instances portions of organs were used for other purposes and in other instances examination methods were of limited sensitivity. Examinations for hematotropic parasites were negative for 20 red foxes and for all coyotes and gray foxes.

Lesions were associated with infections of *Paragonimus kellicotti, Capillaria plica,* and *Spirocerca lupi* and with infestations of *Otodectes cynotis.* A 1-cm subpleural granuloma within a zone of congestion and atelectasis surrounded a pair of *P. kellicotti* in lung parenchyma of a red fox. Diffuse congestion and focal hemorrhage within urinary bladder epithelium were associated with infection by *C. plica* in a red fox. Multiple granulomatous nodules and aortic aneurysms occurred in all four coyotes harboring *S. lupi.* Gray foxes infested with large numbers of *O. cynotis* had excessive cerumen and detritus in ear canals. The remaining parasites were not associated with significant

TABLE 2. Parasites found in red foxes, coyotes, and gray foxes translocated from the midwest to South Carolina.

	Red foxes	Coyotes	Gray foxes
	Protozoans		
Isospora spp.	32/44 (NA) (NA)[a]	0/13	12/18 (NA) (NA)
Sarcocystis spp.	5/28 (NA) (NA)	2/13 (NA) (NA)	2/18 (NA) (NA)
	Helminths		
Alaria americana	2/44 (6.5) (12)	5/13 (25.8) (61)	0/18
Alaria arisaemoides	5/44 (6.0) (13)	0/13	0/18
Alaria spp.	5/44 (1.8) (4)	0/13	0/18
Paragonimus kellicotti	1/44 (2) (2)	0/13	0/18
Echinococcus multilocularis	3/44 (1,121) (3,320)	0/13	0/18
Mesocestoides spp.	0/44	5/13 (17.4) (47)	0/18
Taenia crassiceps	21/44 (13.0) (118)	2/13 (21.5) (39)	1/18 (5) (5)
Taenia pisiformis	4/44 (5.0) (15)	7/13 (43.0) (91)	7/18 (4.7) (19)
Ancylostoma caninum	0/44	0/13	12/18 (6.3) (28)
Capillaria plica	2/28 (NA) (NA)	0/13	0/18
Capillaria sp.	0/44	0/13	1/18 (1) (1)
Dirofilaria immitis	6/28 (5.5) (10)	0/13	0/18
Molineus sp.	0/44	0/13	5/18 (2.0) (3)
Physaloptera spp.	8/44 (1.3) (2)	3/13 (2.0) (3)	10/18 (1.3) (2)
Spirocerca lupi	0/28	4/13 (NA) (NA)	0/18
Toxascaris sp.	2/44 (8.0) (15)	0/13	1/18 (2) (2)
Toxocara sp.	11/44 (1.5) (3)	0/13	2/18 (1.5) (2)
Trichinella spiralis	2/28 (NA) (NA)	0/13	0/18
Trichuris spp.	4/44 (1.3) (2)	0/13	0/18
Uncinaria stenocephala	4/44 (1.0) (1)	0/13	7/18 (2.9) (5)
	Arthropods		
Cediopsylla simplex	7/28 (1.7) (4)	0/13	0/18
Pulex simulans	0/28	0/13	5/18 (1.2) (2)
Otodectes cynotis	0/28	0/13	18/18 (166.4) (600)

[a] Number infected/number examined (mean intensity) (maximum intensity); NA, data not available.

lesions; however, we did not conduct the careful inspection of gastrointestinal mucosa which would be required to detect often subtle tissue damage typical of many of the gastrointestinal parasites.

DISCUSSION

The animals examined during this study were obtained fortuitously. Because they were not selected according to a planned study design, they may not be representative of all animals available to fox-chasing enclosure operators. Nevertheless, they provide the only insight currently available on the health status of wild canids being translocated to supply fox-chasing enclosures. Furthermore, based on records obtained during the covert investigation, thousands of animals from the same source as the red foxes and coyotes had been supplied to fox-chasing enclosures in 17 states, suggesting that our data on these two species may be reasonably representative of a considerable portion of the animals being translocated for this purpose.

In addition, data from these animals must be interpreted with caution. Isolation of viruses and recovery of parasites confirm current infections among the animals, whereas antibody titers provide only indirect evidence of infection. Based on the antibody titers to canine viruses found in this study, we infer probable previous infections but not necessarily current infections. However, the presence of antibodies does not exclude the possibility of current infection and virus transmission. In dogs, an antibody titer of $\geq$1:10 in any of the

reported tests is considered positive. the cut-off point in foxes or coyotes is not known but is assumed to be similar.

Criteria for evaluation of the health risks associated with the translocation of other species of wildlife have been outlined previously (Schaffer, 1979; Schaffer et al., 1981; Schorr et al., 1988), and these criteria are applicable to disease risks associated with the translocation of foxes and coyotes. The criteria are composed of two separate, but related, factors. One factor is the pathogenicity of the agents in question. Understandably, this aspect includes not only pathogenicity for the hosts being studied, but also pathogenicity for other species of wildlife, domestic animals, and humans. The second factor is the probability that the agents in question, especially those which are exotic to release sites, will persist under the ecologic conditions present at release sites. Under these criteria, potential risks include both the introduction of exotic pathogens in release areas and artificial intensification of enzootic pathogens.

Most diseases and parasites detected or indicated by serologic tests during this study have not been associated with epizootic mortality among wild populations of these canids, although canine distemper among gray foxes is a notable exception (Helmboldt and Jungherr, 1955; Hoff et al., 1974; Monson and Stone, 1976; Nicholson and Hill, 1984; Davidson et al., 1992). Many of these diseases and parasites, however, have caused sporadic morbidity or mortality among these hosts. In addition to canine distemper, other agents pathogenic for wild foxes or coyotes include canine parvovirus (Evermann et al., 1980; Holzman et al., 1992), canine adenovirus (Cabasso, 1981), *Alaria* spp. (Schmidt and Roberts, 1989), *Paragonimus kellicotti* (Davis and Libke, 1971; Davidson et al., 1992), *Ancylostoma caninum* (Radomski, 1989), *Dirofilaria immitis* (Crowell et al., 1977; Custer and Pence, 1981), *Spirocerca lupi*, *Uncinaria stenocephala*, and *Otodectes cynotis* (Soulsby, 1968).

As noted, a complete assessment of the potential health risks also should include pathogenicity for other species of wildlife, domestic animals, and humans. In this regard, the occurrence of *Echinococcus multilocularis* in red foxes is particularly significant because of its implications for public health. Alveolar hydatid disease, caused by *E. multilocularis*, is a devastating disease in humans which often is difficult to diagnose and to treat and which typically has a high case fatality rate regardless of treatment (Rausch, 1975; Schantz et al., 1982). Additional agents found with significant pathogenic capabilities in other wildlife, domestic animals, or humans include: canine parvovirus, canine distemper virus, canine adenovirus, canine coronavirus, canine herpesvirus, canine parainfluenza virus (Fenner et al., 1987; Budd, 1981; Cabasso, 1981), *Isospora* spp. (Levine, 1985; Soulsby, 1968), *Alaria* spp., *P. kellicotti*, *A. caninum*, *Physaloptera* spp., *D. immitis*, *S. lupi*, *Trichinella spiralis*, *U. stenocephala*, and *O. cynotis* (Davis and Libke, 1971; Schmidt and Roberts, 1989; Soulsby, 1968). Some of the remaining agents also may cross species boundaries but have more limited pathologic potentials.

Except for *E. multilocularis*, all of the diseases and parasites detected are known to be enzootic within the southeastern United States and, according to the rationale delineated for evaluating health risks (Schaffer, 1979; Schaffer et al., 1981; Schorr et al., 1988), would have an excellent probability of persisting within release sites. Based on Gemmell and Lawson (1986), eggs of *E. multilocularis* would not be vulnerable to environmental extremes in the Southeast; thus, any eggs shed would be expected to successfully develop to the infective stage. Genera of rodents (*Microtus*, *Peromyscus*, *Mus*) important as intermediate hosts for *E. multilocularis* (Leiby, 1965; Leiby and Nickel, 1968; Leiby and Kritsky, 1972; Leiby et al., 1970) are widely distributed in the southeastern United States (Hall, 1981). In addition, the

abundant and widely distributed cotton rat (*Sigmodon hispidus*) (Hall, 1981) experimentally was found to be a suitable intermediate host for *E. multilocularis* (Rau and Tanner, 1973; Baron et al., 1974). Ample numbers of definitive hosts, including red foxes, coyotes, and domestic dogs and cats or their feral counterparts, are present throughout the region. Thus, we conclude that *E. multilocularis* could become established in the southeastern United States. This conclusion is supported by the fact that *E. multilocularis* apparently was introduced within this century into the upper great plains where it now occurs in at least nine states and three Canadian provinces (Leiby et al., 1970; Kritsky et al., 1977; Wilson and Rausch, 1980; Ballard and Vande Vusse, 1983; Ballard, 1984).

In addition to the criteria of pathogenicity and likelihood of establishment, other epizootiologic factors merit consideration. The conditions of husbandry and shipment of the animals were conducive to the transfer of infectious diseases and monoxenous life cycle parasites which are notorious for producing disease among crowded, stressed hosts (Fenner et al., 1987; Schmidt and Roberts, 1989). The occurrence of canine distemper virus infections and severe ear mite infestations in the majority of the gray foxes are examples of this potential. Further, based on the typically rapid course and high mortality of canine distemper in gray foxes (Budd, 1981), we believe the gray foxes were infected at the trapper's facility, probably only days prior to submission to SCWDS. This fact emphasizes the dynamic state of diseases among translocated wildlife, with the potential for either appearance or disappearance of pathogens as noted by Schorr et al. (1988). Finally, although none of these animals were rabid, the frequent occurrence of bite wounds suggests excellent potential for transmission of rabies virus, as noted among raccoons translocated under similar conditions (Nettles and Martin, 1979; Nettles et al., 1979).

Several of the agents detected meet the pathologic and epizootiologic criteria for being significant health risks to wildlife, domestic animals, or humans. *Echinococcus multilocularis* is of greatest concern because of its serious public health implications and because it is currently exotic to the region. Canine distemper virus also merits special consideration because of its importance as a mortality factor among many species of native wildlife and domestic dogs.

We conclude that private-sector translocation of wild canids for the purpose of stocking fox-chasing enclosures, as it is often practiced currently, is biologically hazardous. This conclusion is not without precedent. Several workers found that raccoons translocated within the private sector had infections by rabies virus, parvovirus, and pathogenic parasites, and because of these findings, raccoon translocations also were considered biologically hazardous (Nettles and Martin, 1979; Nettles et al., 1979, 1980; Schaffer, 1979; Schaffer et al., 1979, 1981). The current mid-Atlantic raccoon rabies epizootic, which began coincident with the discovery of rabid translocated raccoons, is due to the same strain of rabies virus enzootic in raccoons in the southeastern United States, providing circumstantial evidence that it was initiated by raccoon translocation (Smith, et al., 1984; Jenkins and Winkler, 1987). Based on the many similarities between raccoon and wild canid translocation, we suggest that the spectrum of health risks posed by wild canid translocation is comparable to that well demonstrated by raccoon translocation.

ACKNOWLEDGMENTS

This study was supported through sponsorship by the fish and wildlife agencies of Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Maryland, Missouri, Mississippi, North Carolina, Puerto Rico, South Carolina, Tennessee, Virginia, and West Virginia, and by the Fish and Wildlife Service, U.S. Department of the Interior. Funds were administered and research coordinated under the Federal Aid in Wildlife Restoration Act (50 Stat. 917) and through Contract Numbers 14-16-0004-89-912, and 14-16-

0004-90-916 Fish and Wildlife Service, U.S. Department of the Interior. We thank Drs. J. W. Mertins and J. L. Schlater, National Veterinary Services Laboratories for identification of arthropods. Dr. P. Schantz, Centers for Disease Control, and Dr. R. L. Rausch, University of Washington, confirmed the identification of *Echinococcus multilocularis*. The Athens Diagnostic Assistance Laboratory, College of Veterinary Medicine, The University of Georgia conducted canine distemper fluorescent antibody tests. We also acknowledge the SCWDS staff for excellent technical support.

LITERATURE CITED

APPEL, M., AND J. H. GILLESPIE. 1972. Canine distemper virus. Virology Monographs 11: 1–96.

———, AND D. S. ROBSON. 1973. Microneutralization test for canine distemper virus. American Journal of Veterinary Research 34: 1459–1463.

BALLARD, N. B. 1984. *Echinococcus multilocularis* in Wisconsin. The Journal of Parasitology 70: 844.

———, AND F. J. VANDE VUSSE. 1983. *Echinococcus multilocularis* in Illinois and Nebraska. The Journal of Parasitology 69: 790–791.

BARON, R. W., M. E. RAU, AND C. E. TANNER. 1974. Growth of secondary *Echinococcus multilocularis* in experimentally infected hosts. Canadian Journal of Zoology 52: 587–589.

BUDD, J. 1981. Distemper. *In* Infectious diseases of wild mammals, 2nd ed. J. W. Davis, L. H. Karstad, and D. O. Triner (eds.). The Iowa State University Press, Ames, Iowa, pp. 31–44.

CABASSO, V. J. 1981. Infectious canine hepatitis. *In* Infectious diseases of wild mammals, 2nd ed. J. W. Davis, L. H. Karstad, and D. O. Trainer (eds.). The Iowa State University Press, Ames, Iowa, pp. 191–195.

CARMICHAEL, L. E., J. C. JOUBERT, AND R. V. H. POLLOCK. 1980. Hemagglutination by canine parvovirus: Serologic studies and diagnostic applications. American Journal of Veterinary Research 40: 784–791.

CROWELL, W. A., T. R. KLEI, D. I. HALL, N. K. SMITH, AND J. D. NEWSOM. 1977. Occurrence of *Dirofilaria immitis* and associated pathology in coyotes and foxes from Louisiana. *In* Proceedings of the Heartworm Symposium 1977, G. F. Otto (ed.). American Heartworm Society, Washington, D.C. pp. 10–13.

CRUM, J. M., V. F. NETTLES, AND W. R. DAVIDSON. 1978. Studies on endoparasites of the black bear (*Ursus americanus*) in the southeastern United States. Journal of Wildlife Diseases 14: 178–186.

CUSTER, J. W., AND D. B. PENCE. 1981. Dirofilariasis in wild canids from the gulf coastal prairies of Texas and Louisiana, USA. Veterinary Parasitology 8: 71–82.

DAVIDSON, W. R., V. F. NETTLES, L. E. HAYES, E. W. HOWERTH, AND C. E. COUVILLION. 1992. Diseases diagnosed in gray foxes (*Urocyon cinereoargenteus*) from the southeastern United States. Journal of Wildlife Diseases 28: 28–33.

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.

DEAN, D. J., AND M. K. ABELSETH. 1973. The fluorescent antibody test. *In* Laboratory techniques in rabies, 3rd ed. M. M. Kaplan and H. Koprowski (eds.). World Health Organization, Geneva, Switzerland, pp. 73–84.

EVERMANN, J. F., W. J. FOREYT, L. MAAG-MILLER, C. W. LEATHERS, A. J. MCKEIRNAN, AND B. LEAMASTER. 1980. Acute hemorrhagic enteritis associated with canine coronavirus and parvovirus infections in a captive coyote population. Journal of the American Veterinary Medical Association 177: 784–786.

FENNER, F., P. A. BACHMAN, E. P. J. GIBBS, F. A. MURPHY, M. J. STUDDERT, AND D. O. WHITE. 1987. Veterinary virology. Academic Press, Inc., Harcourt Brace Jovanovich, Publishers, Orlando, Florida, 660 pp.

GEMMELL, M. A., AND J. R. LAWSON. 1986. Epidemiology and control of hydatid disease. *In* The biology of *Echinococcus* and hydatid disease, R. C. A. Thompson (ed.). George Allen and Unwin (Publishers) Ltd., London, England, pp. 189–216.

HALL, E. R. 1981. The mammals of North America (Volume II), 2nd ed. John Wiley and Sons, New York, New York, 1181 pp.

HELMBOLDT, C. F., AND E. L. JUNGHERR. 1955. Distemper complex in wild carnivores simulating rabies. American Journal of Veterinary Research 16: 463–469.

HOFF, G. L., W. J. BIGLER, S. J. PROCTOR, AND L. P. STALLINGS. 1974. Epizootic of canine distemper virus infection among urban raccoons and gray foxes. Journal of Wildlife Diseases 10: 423–428.

HOLZMAN, S., M. J. CONROY, AND W. R. DAVIDSON. 1992. Diseases, parasites and survival of coyotes in south-central Georgia. Journal of Wildlife Diseases 28: In press.

JENKINS, S. R., AND W. G. WINKLER. 1987. Descriptive epidemiology from an epizootic of raccoon rabies in the middle Atlantic states, 1982–1983. American Journal of Epidemiology 126: 429–437.

KRITSKY, D. C., P. D. LEIBY, AND G. E. MILLER. 1977. The natural occurrence of *Echinococcus multilocularis* in the bushy-tailed woodrat *Neotoma cinerea rupicola* in Wyoming. American Journal of Tropical Medicine and Hygiene 26: 1046–1047.

LEIBY, P. D. 1965. Cestode in North Dakota: *Echinococcus* in field mice. Science 150: 763.

———, AND D. C. KRITSKY. 1972. *Echinococcus multilocularis*: A possible domestic life cycle in central North America and its public health implications. The Journal of Parasitology 58: 1213–1215.

———, AND M. P. NICKEL. 1968. Studies on sylvatic echinococcosis. I. Ground beetle transmission of *Echinococcus* multilocularis Leuckart, 1863, to deer mice, *Peromyscus maniculatus* (Wagner). The Journal of Parasitology 54: 536–537.

———, W. P. CARNEY, AND C. E. WOODS. 1970. Studies on sylvatic echinococcosis. III. Host occurrence and geographic distribution of *Echinococcus multilocularis* in the north central United States. The Journal of Parasitology 56: 1141–1150.

LEVINE, N. D. 1985. Veterinary protozoology. The Iowa State University Press, Ames, Iowa, 414 pp.

MONSON, R. A., AND W. B. STONE. 1976. Canine distemper in wild carnivores in New York. New York Fish and Game Journal 23: 149–154.

NELLIS, C. H., S. P. WETMORE, AND L. B. KEITH. 1978. Age-related characteristics of coyote canines. The Journal of Wildlife Management 42: 680–683.

NETTLES, V. F. 1981. Necropsy procedures. *In* Diseases and parasites of white-tailed deer, W. R. Davidson, F. A. Hayes, V. F. Nettles, and F. E. Kellogg (eds.). Miscellaneous Publication Number 7, Tall Timbers Research Station, Tallahassee, Florida, pp. 6–16.

———, AND W. M. MARTIN. 1979. General physical parameters and health characteristics of translocated raccoons. Proceedings of the Annual Conference of the Southeastern Association of Game and Fish Commissioners 32: 71–74.

———, J. H. SHADDOCK, R. K. SIKES, AND C. R. REYES. 1979. Rabies in translocated raccoons. American Journal of Public Health 69: 601–602.

———, J. E. PEARSON, G. A. GUSTAFSON, AND J. L. BLUE. 1980. Parvovirus infection in translocated raccoons. Journal of the American Veterinary Medical Association 177: 787–789.

NICHOLSON, W. S., AND E. P. HILL. 1984. Mortality in gray foxes from east-central Alabama. The Journal of Wildlife Management 48: 1429–1432.

PETRIDES, G. A. 1950. The determination of sex and age ratios in fur animals. American Midland Naturalist 43: 355–382.

RADOMSKI, A. A. 1989. Host-parasite relationships of helminths in a coyote population from southern Texas, with particular reference to the dog hookworm. M.S. Thesis. Texas Tech University, Lubbock, Texas, 132 pp.

RAU, M. E., AND C. E. TANNER. 1973. *Echinococcus multilocularis* in the cotton rat: The effect of preexisting subcutaneous cysts on the development of a subsequent intraperitoneal inoculum of protoscolices. Canadian Journal of Zoology 51: 55–59.

RAUSCH, R. L. 1975. Taeniidae. *In* Diseases transmitted from animals to man, 6th ed. W. T. Hubbert. W. F. McCulloch, and P. R. Schnurrenberger (eds.). Charles C. Thomas Publisher, Springfield, Illinois, pp. 678–707.

SCHAFFER, G. D. 1979. Helminth parasites of translocated raccoons (*Procyon lotor*) in the southeastern United States. M.S. Thesis. The University of Georgia, Athens, Georgia, 93 pp.

———, W. L. HANSON, W. R. DAVIDSON, AND V. F. NETTLES. 1979. Hematotropic parasites of translocated raccoons in the Southeast. Journal of the American Veterinary Medical Association 173: 1148–1151.

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

SCHANTZ, P. M., H. V. VAN DEN BOSSCHE, AND J. ECKERT. 1982. Chemotherapy for larval *Echinococcus* in animals and humans: Report of a workshop. Zeitschrift für Parasitenkunde 67: 5–26.

SCHMIDT, G. D., AND L. S. ROBERTS. 1989. Foundations of parasitology. Times Mirror/Mosby College Publishing, St. Louis, Missouri, 750 pp.

SCHORR, L. F., W. R. DAVIDSON, V. F. NETTLES, J. E. KENNAMER, P. VILLEGAS, AND H. W. YODER. 1988. A survey of parasites and diseases of penraised wild turkeys. Proceedings of the Annual Conference of the Southeastern Association of Fish and Wildlife Agencies 42: 315–328.

SMITH, J. S., J. W. SUMMER, L. F. ROUMILLAT, G. M. BAER, AND W. G. WINKLER. 1984. Antigenic characteristics of isolates associated with a new epizootic of raccoon rabies in the U.S. The Journal of Infectious Disease 149: 769–774.

SOULSBY, E. J. L. 1968. Helminths, arthropods and protozoa of domesticated animals, 6th ed. The Williams and Wilkins Company, Baltimore, Maryland. 824 pp.

WILSON, J. F., AND R. L. RAUSCH. 1980. A review of clinical features of 33 indigenous cases of *Echinococcus multilocularis* infection in Alaskan Eskimos. American Journal of Tropical Medicine and Hygiene 29: 1340–1355.

WINDBERG, L. A., R. M. ENGEMAN, AND J. F. BROMAGHIN. 1991. Body size and condition of coyotes in southern Texas. Journal of Wildlife Diseases 27: 47–52.

Received for publication 26 August 1991.