Diseases Diagnosed in Red Foxes from the Southeastern United States

Authors: Little, Susan E., Davidson, William R., Howerth, Elizabeth W., Rakich, Pauline M., and Nettles, Victor F.

Source: Journal of Wildlife Diseases, 34(3) : 620-624

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-34.3.620

Journal of Wildlife Diseases, 34(3), 1998, pp. 620–624

Diseases Diagnosed in Red Foxes from the Southeastern United States

Susan E. Little,[1,5] William R. Davidson,[2] Elizabeth W. Howerth,[3] Pauline M. Rakich,[4] and Victor F. Nettles,[1]
[1] Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602, USA; [2] School of Forest Resources and Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602, USA; [3] Department of Pathology, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602, USA; [4] Athens Diagnostic Laboratory, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602, USA; and [5] Corresponding author (e-mail: slittle@calc.vet.uga.edu)

ABSTRACT: Diagnostic findings on 51 red foxes (*Vulpes vulpes*) from the southeastern United States examined from 1967 to 1995 were reviewed. Etiologic diagnoses included sarcoptic mange (n = 33), traumatic injury and associated complications (n = 5), suspected canine distemper (n = 2), capture myopathy (n = 1), congenital absence of guard hairs (n = 1), intradermal tick infestation (n = 1), otodectic mange (n = 1), and toxicosis (n = 1). The cause of morbidity was not determined for three of the foxes, and three others were classified as normal animals. Sarcoptic mange was diagnosed in 65% of the red foxes, was found in foxes submitted from four of the eight southeastern states represented, and was seen in 19 of 29 yr covered by this study.

Key words: Diseases, mortality factors, red fox, sarcoptic mange, survey, *Vulpes vulpes*.

The red fox (*Vulpes vulpes*) is the most widely distributed carnivore in the world (Voight, 1987). Although outnumbered by gray foxes (*Urocyon cinereoargenteus*) throughout much of the southeastern United States, red foxes are a common inhabitant of this region and are found in a variety of habitats (Sheldon, 1992). Despite this wide geographic distribution, only a small amount of published information is available regarding mortality factors in this species and none of the available reviews cover the southeastern United States.

Necropsy and laboratory records for red foxes found sick or dead and submitted to the Southeastern Cooperative Wildlife Disease Study (SCWDS; College of Veterinary Medicine, The University of Georgia, Athens, Georgia, USA) by personnel of state or federal wildlife agencies for diagnostic examination were reviewed for the period 1 January 1967 through 31 December 1995. Whole animals, usually refrigerated but sometimes frozen, were available for examination in most instances; however, in two cases, only the head or body was submitted. Examinations were sometimes hampered by postmortem decomposition or inadequate preservation techniques; however, such problems hindered reaching a diagnosis in only one of the cases.

Because necropsies were oriented toward determination of the cause of morbidity or mortality, diagnostic procedures varied among cases. For histologic examination, tissues were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 5 μm, stained with hematoxylin and eosin, and examined microscopically. Frozen sections of brain were tested with a fluorescent antibody for rabies virus (Smith, 1991). Fluorescent antibody testing for canine distemper virus was performed on frozen sections of lung, liver, stomach, brain, and/or conjunctival swabs (Appel, 1969).

Data reviewed for each case accession were the number, sex, and age of animals involved, location, date, case history, and major diagnostic findings. An effort was made to characterize diagnostic findings as primary or secondary factors. Diseases that occurred with sufficient frequency were evaluated for patterns of occurrence relative to temporal, geographic, and host sex and age factors. Monthly frequencies of diagnoses were evaluated for temporal differences using a Chi-square test and the runs test (Remington and Schork, 1970). Differences in prevalence among host sex

and age classes were tested by the G-statistic (Sokal and Rohlf, 1981).

Over the 29 yr period, 51 red foxes from eight states were submitted for examination. Foxes were submitted from throughout the southeastern United States (30°15′N to 39°05′N; 79°30′W to 92°09′W). Most of the foxes were from Georgia (n = 40); fewer were from Tennessee (n = 3), West Virginia (n = 2), Alabama (n = 2), Arkansas (n = 1), North Carolina (n = 1), South Carolina (n = 1), and Virginia (n = 1). Male foxes outnumbered females (29 versus 19; 3 not recorded) and adults outnumbered young (31 versus 18; 2 not recorded). In most cases, history disclosed that only a single animal was found; however, in at least three instances, several additional red foxes were reported sick or dead in the same area as the individual submitted.

Sarcoptic mange (SM), characterized by severe dermatitis and the presence of numerous *Sarcoptes scabiei* mites, was the most frequent diagnosis and occurred in 33 (65%) foxes (Table 1). Red foxes with SM were submitted from 21 counties in four states. Sarcoptic mange was diagnosed in 19 of the 29 yr, and in every year in which more than one red fox was submitted for examination. The occurrence of mange cases did not differ significantly among months (Chi-square = 13.01; df = 11). Calculation of 3-mo moving averages to smooth the trend of monthly submissions of mange cases produced a seasonal pattern, with more cases of SM submitted in the summer and fall. Runs test on raw data was not significant ($P > 0.05$), but runs test on smoothed data by calculation of 3-mo moving averages approached significance ($P = 0.05$). The prevalence of mange cases did not differ ($P > 0.05$) between sexes (males = 59%, females = 74%) or between young (57%) and adult (68%) red foxes.

Red foxes with SM were invariably emaciated, often severely. Clinical signs reported in foxes with SM included lack of fear, weakness, lethargy, incoordination, and profound depression. Five of the mangy red foxes reportedly had contact with domestic dogs immediately prior to death. The most prominent gross lesion was diffuse, marked hyperkeratosis and alopecia with severe dermal lichenification. Numerous *S. scabiei* mites were found easily in scrapings of lesions. In most cases, dehydration and generalized lymphadenopathy also were noted.

Histologically, profound parakeratotic and orthokeratotic hyperkeratosis containing sarcoptic mites, mite feces, and eggs were present in most sections. Serocellular crusts containing degenerate neutrophils, gram positive bacterial cocci, and occasional yeast overlaid an acanthotic epidermis. Rete ridge formation was prominent and consisted of epidermal pegs extending into the dermis and dermal projections towards the skin surface. Cross sections of adult mites often were found in close apposition to the stratum basale. The superficial dermis had marked mast cell hyperplasia, numerous eosinophils, and occasional neutrophils. The lymph nodes of infected foxes were markedly hyperplastic with numerous large follicles.

The additional mortality findings were less common than SM and are summarized in Table 1. The two foxes presumptively diagnosed with canine distemper had characteristic intranuclear inclusion bodies in epithelial cells, but fluorescent antibody testing of conjunctival swabs was negative for canine distemper virus. Both of these foxes had abnormal neurologic signs prior to death and both were negative on fluorescent antibody testing of brain for rabies virus. One of the two also had systemic toxoplasmosis; *Toxoplasma gondii* tachyzoites, identified presumptively by recognizing characteristic morphology (Gardiner et al., 1988), were found in alveolar macrophages, Kupffer cells, and lymph node histiocytes.

Submission of cases was influenced by many indefinable factors and, as such, these cases form a biased data set that may give an erroneous assessment of the rela-

TABLE 1. Diagnostic findings in 51 sick or dead red foxes from the southeastern United States from 1967 through 1995.

Diagnosis	Number of foxes	(%)	States[a]	Yr
Sarcoptic mange[b]	33	(65)	AL, GA, SC, WV	1967, 1971, 1972, 1976–1981, 1983–1988, 1991, 1992, 1994, 1995
Trauma	5[d]	(10)	AR, GA, TN	1982, 1984
Canine distemper, suspected	2	(4)	GA	1987, 1988
Capture myopathy	1	(2)	GA	1985
Congenital lack of guard hairs	1	(2)	GA	1981
Intradermal tick infestation	1	(2)	GA	1994
Otodectic mange	1[e]	(2)	GA	1969
Strychnine toxicosis[c]	1	(2)	NC	1985
Normal animal	3	(6)	AL, GA, TN	1987, 1991, 1994
Undetermined	3	(6)	GA, VA	1978, 1980, 1987

[a] AL = Alabama, AR = Arkansas, GA = Georgia, NC = North Carolina, SC = South Carolina, TN = Tennessee, VA = Virginia, WV = West Virginia.

[b] Mites identified according to published descriptions (Baker et al., 1956); representative specimens deposited in the U.S. National Parasite Collection, Beltsville, Maryland, USA; Accession Nos. 87063, 87064, and 87065.

[c] Strychnine detected in stomach contents as described by Plantanow et al. (1970) modified for use with a thin layer chromatography plate.

[d] Only body submitted from one animal.

[e] Only head submitted.

tive importance of natural mortality factors in red foxes. Foxes that die of obvious traumatic lesions might not be submitted for diagnostic evaluation, whereas those with remarkable external lesions such as the diffuse hyperkeratotic dermatitis and hair loss seen in severe mange cases might be submitted at a higher rate (Davidson et al., 1992). Proximity to the SCWDS laboratory apparently also influenced submissions; the majority of cases were submitted from Georgia.

Despite the influence of bias on submission of cases, the data indicate SM is a common diagnostic finding in red foxes in the southeastern United States, exceeding all other infectious and non-infectious diseases combined as a diagnosis in dead or moribund red foxes. This finding agrees with reports from other areas of North America. Sixty-seven percent of red foxes submitted to a diagnostic laboratory from New Brunswick and Nova Scotia were diagnosed with SM (Smith, 1978) and outbreaks also have been reported in Alberta (Todd et al., 1981), New York (Stone et al., 1974), Ohio (Olive and Riley, 1948), Pennsylvania (Pryor, 1956), Wisconsin (Trainer and Hale, 1969), and the midwestern United States (Gier et al., 1978).

The regular submission of mange cases over nearly three decades suggests that SM has been consistently present in red foxes in this region for some time. However, the impact of this disease on the survival of red foxes in the wild is not well understood. Only one report documents recovery of affected red foxes in the wild (Storm et al., 1976). More recent work indicates self-cure can occur in coyotes with SM (Pence and Windberg, 1994). Experimental infections of red foxes do not provide any evidence of an ability to recover spontaneously once disease has developed (Stone et al., 1972; Mörner and Christensson, 1984); however, these findings are potentially attributed to the overwhelming infections established experimentally or the stress and subsequent immunocompromise induced by captive experimental conditions.

The significance of SM as a mortality factor in red foxes in the southeastern United States is not possible to definitively determine from this review. The majority of foxes with SM were intentionally killed by humans due to their appearance and compromised condition or by dog attacks or vehicular collisions. This suggests that in the southeastern United States where relatively mild winter weather conditions exist, mange may not kill red foxes directly, but could render foxes more susceptible to traumatic events. In contrast, Scandinavian red fox experienced a marked reduction in population following the introduction of SM (Lindstrom et al., 1994). Overskaug (1994) showed that infested red foxes were less active than healthy ones and suggested that decreased activity could result in both starvation and relaxed vigilance.

Other infectious diseases were virtually absent from our review. Canine distemper was strongly suspected in two cases based on histologic findings, but was not confirmed by fluorescent antibody testing. The apparent insignificance of canine distemper as a mortality factor in red foxes stands in sharp contrast to reviews of gray fox mortality, which show distemper to be the most important disease affecting gray foxes in the eastern United States (Davidson et al., 1992). Red foxes and gray foxes respond quite differently to canine distemper vaccines (Halbrook et al., 1991); simultaneous vaccination of both species produced disease in gray foxes and not in red foxes (Davidson et al., 1992). Interestingly, gray foxes are resistant to infection with *S. scabiei* even under experimental conditions (Stone et al., 1972) and SM is extremely rare in wild gray foxes (Stone et al., 1982).

This study was supported through sponsorship from the fish and wildlife agencies of Alabama, Arkansas, Florida, Georgia, Kansas, Kentucky, Louisiana, Maryland, Mississippi, Missouri, North Carolina, Puerto Rico, South Carolina, Tennessee,

Virginia, and West Virginia. Funds were provided by the Federal Aid to Wildlife Restoration Act (50 Stat. 917) and through Grant Agreement 14-45-0009-94-906, National Biological Service, U.S. Department of the Interior. We thank numerous personnel with the above sponsoring agencies for field assistance.

LITERATURE CITED

APPEL, M. 1969. Pathogenesis of canine distemper. American Journal of Veterinary Research 30: 1167–1182.

BAKER, E. W., T. M. EVANS, D. J. GOULD, W. B. HULL, AND H. L. KEEGAN. 1956. A manual of parasitic mites of medical or economic importance. National Pest Control Association, Inc., New York, New York, 170 pp.

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

GARDINER, C. H., R. FAYER, AND J. P. DUBEY. 1988. An atlas of protozoan parasites in animal tissues. Agriculture Handbook No. 651, U.S. Department of Agriculture, Washington, D.C., 83 pp.

GIER, H. T., S. M. KRUCKENBERG, AND R. J. MARLER. 1978. Parasites and diseases of coyotes. *In* Coyotes: Biology, behavior, and management, M. Bekoff (ed.). Academic Press, Inc., New York, New York, pp. 37–71.

HALBROOKS, R. D., L. J. SWANGO, P. R. SCHNURRENBERGER, F. E. MITCHELL, AND E. P. HILL. 1981. Response of gray foxes to modified live-virus canine distemper vaccines. Journal of the American Veterinary Medical Association 179: 1170–1174.

LINDSTROM, E. R., H. ANDREN, P. ANGELSTAM, G. CEDERLUND, B. HORNFLEDT, L. JADERBERG, P. LEMNELL, B. MARTINSSON, K. SKOLD, AND J. E. SWENSON. 1994. Disease reveals the predator: Sarcoptic mange, red fox predation, and prey populations. Ecology 75: 1042–1049.

MÖRNER, T., AND D. CHRISTENSSON. 1984. Experimental infestation of red foxes (*Vulpes vulpes*) with *Sarcoptes scabiei* var. *vulpes*. Veterinary Parasitology 15: 159–164.

OLIVE, J. R., AND C. V. RILEY. 1948. Sarcoptic mange in the red fox in Ohio. Journal of Mammalogy 29: 73–74.

OVERSKAUG, K. 1994. Behavioural changes in free-ranging red foxes (*Vulpes vulpes*) due to sarcoptic mange. Acta Veterinaria Scandinavica 35: 457–459.

PENCE, D. B., AND L. A. WINDBERG. 1994. Impact of a sarcoptic mange epizootic on a coyote population. The Journal of Wildlife Management 58: 624–633.

PLANTANOW, W., H. S. FUNNELL, AND W. T. OLIVER. 1970. Determination of strychnine in biological materials by gas chromatography. Journal of Forensic Science 15: 443–446.

PRYOR, L. B. 1956. Sarcoptic mange in wild foxes in Pennsylvania. Journal of Mammalogy 37: 90–93.

REMINGTON, R. D., AND M. A. SCHORK. 1970. Statistics with applications to the biological and health sciences. Prentice-Hall, Inc., Englewood Cliffs, New Jersey, 418 pp.

SHELDON, J. W. 1992. Wild dogs: The natural history of the nondomestic Canidae. Academic Press, Inc., San Diego, California, 248 pp.

SMITH, H. J. 1978. Parasites of red foxes in New Brunswick and Nova Scotia. Journal of Wildlife Diseases 14: 366–370.

SMITH, J. S. 1991. Rabies virus. *In* Manual of clinical microbiology, 5th ed. A. Balows, W. J. Hausler, K. L. Herrmann, H. D. Isenberg, and H. J. Shadomy (eds.). American Society for Microbiology, Washington, D.C., pp. 936–942.

SOKAL, R. R., AND F. J. ROHLF. 1981. Biometry: The principles and practice of statistics in biological research, 2nd ed. W. H. Freeman and Company, New York, New York, 859 pp.

STONE, W. B., E. PARKS, B. L. WEBER, AND F. J. PARKS. 1972. Experimental transfer of sarcoptic mange from red foxes and wild canids to captive wildlife and domestic animals. New York Fish and Game Journal 19: 1–11.

———, B. F. TULLAR, J. B. ZEH, AND B. L. WEBER. 1974. Incidence and distribution of mange mites in foxes in New York. New York Fish and Game Journal 21: 163–166.

———, I. F. SALKIN, AND A. MARTEL. 1982. Sarcoptic mange in a gray fox. New York Fish and Game Journal 29: 102–103.

STORM, G. L., R. D. ANDREWS, R. L. PHILLIPS, R. A. BISHOP, D. B. SINIFF, AND J. R. TESTER. 1976. Morphology, reproduction, dispersal, and mortality of midwestern red fox populations. Wildlife Monographs 49: 1–74.

TODD, A. W., J. R. GUNSON, AND W. M. SAMUEL. 1981. Sarcoptic mange: An important disease of coyotes and wolves in Alberta, Canada. *In* Worldwide furbearer conference proceedings, J. A. Chapman and D. Pursley (eds.). R. R. Donneley and Sons, Falls Church, Virginia, pp. 706–729.

TRAINER, D. O., AND J. B. HALE. 1969. Sarcoptic mange in red foxes and coyotes of Wisconsin. Bulletin of the Wildlife Disease Association 5: 387–391.

VOIGHT, D. R. 1987. Red fox. *In* Wild furbearer management and conservation in North America, M. Novak, J. A. Baker, M. E. Obbard, and B. Malloch (eds.). Ontario Ministry of Natural Resource, Toronto, Ontario, pp. 379–392.

Received for publication 27 March 1997.