

University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

Symposium Proceedings—Coyotes in the
Southwest: A Compendium of Our Knowledge
(1995)

Wildlife Damage Management, Internet Center for

3-31-1995

DISEASE AND COYOTES IN TEXAS

Danny B. Pence

Texas Tech University Health Sciences Center

Follow this and additional works at: <http://digitalcommons.unl.edu/coyotesw>



Part of the [Environmental Health and Protection Commons](#)

Pence, Danny B., "DISEASE AND COYOTES IN TEXAS" (1995). *Symposium Proceedings—Coyotes in the Southwest: A Compendium of Our Knowledge (1995)*. Paper 17.

<http://digitalcommons.unl.edu/coyotesw/17>

This Article is brought to you for free and open access by the Wildlife Damage Management, Internet Center for at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Symposium Proceedings—Coyotes in the Southwest: A Compendium of Our Knowledge (1995) by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

DISEASE AND COYOTES IN TEXAS

DANNY B. PENCE, Department of Pathology, Texas Tech University Health Sciences Center, 3601 4th Street, Lubbock, TX 79413

Abstract. The coyote (*Canis latrans*) population in southern Texas has a recurring group of 3 common helminths and several peripheral species of lesser importance. Although recurrent group analyses have not been applied to other macro- or microparasite communities, there are certain infectious agents with high prevalences that could form recurrent groups, and that are potentially important in terms of impacting host population (i.e., coyote) abundance. While the current rabies epizootic involving coyotes in southern Texas is of public health concern, it probably will not have a major impact on the coyote population. Most likely, the net effect of canine rabies will be compensatory with other mortality factors as occurred in other introduced microparasitic (canine distemper virus, canine parvovirus) and periodically recurring macroparasitic (sarcoptic mange) infections that have caused recent epizootics in this coyote population. In contrast, neonatal mortality from hookworm is conjectured to have a possible regulatory effect on the coyote population in southern Texas, but this remains unproven. The effects of disease on the host population should always be considered prior to initiating management or control strategies for any vertebrate species.

With some notable exceptions, coyotes are infected with most of the diseases occurring in other wild and domestic canid species. The last comprehensive review of diseases of coyotes and other canids in North America was by Pence and Custer (1981). Herein, I have not elected to update that publication for Texas. Rather, I will discuss the impact of several recently studied disease epizootics of a coyote population in southern Texas with particular reference to their effect on the host population.

Relative importance of infectious agents

Radomski and Pence (1993) using data on helminth species collected over 9 years from 329 coyotes in southern Texas found a temporally persistent recurrent group of 3 common helminth species. The importance of this study was that it not only determined which of the co-occurring helminth species were members of an interactive recurrent group, but it also provided insight on which of the many helminth species infecting coyotes could potentially affect the coyote population. Because of problems with quantification (collection and culture procedures) and in determining present versus past experience with infection (serological data), there have been no recurrent group analyses on other macroparasites (arthropods) or microparasites (viruses, bacteria, protozoa), or on the collective community of infectious agents.

However, there are certain of these micro- and macroparasites with high prevalences that have caused recent epizootics in this coyote population. These include rabies, distemper, canine parvovirus, sarcoptic mange and hookworm. Probably, they would be important members of a recurrent group of "all infectious agents", certain species of which could potentially impact the host population.

The actions of parasites as mortality factors on host populations are reviewed by Holmes and Price (1986). The net effect with any infectious agent may be either:

- (1) compensatory with other mortality factors, with individual losses having no net effect on the overall population abundance and composition, or
- (2) additive, where losses affect the abundance of the host population

The additive effects of parasite-induced mortality may be severe in some instances, causing host population levels to drop substantially below the threshold for maximum sustained density. However, such cases are not common and often involve introduced pathogens or invading host species. Additive micro- or macroparasite-induced mortality also may function to regulate the host population, with gains or losses in abundance adjusting the number of individuals in the population at a threshold near equilibrium with maximum

sustainable density for the host species. Proven examples of the latter are rare.

It is emphasized that recurrent group members vary across geographic localities in helminth communities and probably also across other gradients that could be established for other macro- and microparasitic communities. Thus, the potential importance of a given parasite to its host population may vary dramatically across geographic localities (Pence 1990)

Rabies

Prior to 1988, rabies occurred only sporadically in coyotes, involving just a few individuals (usually fewer than 10) in the more than 10,000 laboratory confirmed cases per year reported in North America (Pence and Custer 1981). However, in the latter months of 1988, there was a mortality event involving coyotes and domestic dogs in the extreme southern counties of Texas and adjacent Republic of Mexico. Between 1988 and June 1995 there have been 2 human and 638 animal cases (laboratory-confirmed) of rabies with 244 and 322 of these in dogs and coyotes, respectively, across 20 counties of southern Texas (Anonymous 1995). The rabies virus involved is known as the "canine/coyote" or "Mexican dog" strain.

Current efforts are directed toward containment and control of the rabies epizootic in southern Texas through utilization of a vaccine/bait aerial delivery program (Anonymous 1995). The first vaccine/bait drop of the South Texas Oral Rabies Vaccination Project for coyotes was undertaken in February 1995, delivering 830,000 vaccine/bait units over much of southern Texas in the largest single oral vaccination deployment ever undertaken in the world (Anonymous 1995). The oral vaccination project was an attempt to stop the northward and eastward movement of rabies in southern Texas. If this project fails, the epizootic will undoubtedly continue to spread throughout Texas. Also, the epizootic will continue to spread if individuals fail to observe the statewide rabies quarantine on movement of unvaccinated wild canids. The strain already has been identified in Alabama, Florida, Montana and The Netherlands (Anonymous 1995).

There are many unanswered questions concerning the current rabies epizootic in southern Texas. Despite the occurrence of very high densities

of coyotes and the concurrent existence of rabies in dogs in southern Texas for many decades, why did it take so long for the virus to become enzootic in the coyote population? Also, regardless of the much publicized present "epizootic" in coyotes, the prevalence of rabies in this coyote population remains lower than that in similar fox, skunk or raccoon rabies epizootics in other geographic regions in North America. Finally, while there have been no definitive studies on abundance or composition, the coyote population in southern Texas does not appear to be declining due to the present rabies epizootic (S. E. Henke, pers. commun.).

In the red fox (*Vulpes vulpes*) population of central Europe, enzootic rabies acts as a form of time-delayed density-dependent regulator of fox population growth. The length of time lag is determined by how long the fox density is below a critical threshold density for transmission of the disease (about 1 fox/400 acres). As a result of this damped oscillatory cycle, epizootics recur every 3 to 5 years in many areas (Anderson 1981, May 1983). Because of its high pathogenicity, rabies persists within this fox population at very low prevalences between epizootic periods.

Once established as an enzootic disease, will the coyote/dog strain of rabies function in a similar capacity as fox rabies in Europe, to regulate population abundance of coyotes from southern Texas? More likely, the rabies-induced mortality simply will be compensatory with other mortality factors in this population, as has occurred in other recently introduced viral pathogens. It should be noted that host population regulation has not been demonstrated for rabies in red fox or other carnivore populations in North America.

Canine distemper

Certainly distemper virus can be highly lethal to coyote pups in captivity (Gier and Ameel 1959). However, after finding 37% of a small sample of coyotes in southern Texas serologically positive for distemper, Trainer and Knowlton (1968) suggested that canine distemper was enzootic and perhaps not an important mortality factor in free-living coyote populations. This was confirmed by Guo et al. (1986) who examined 228 randomly selected coyote serum samples from a serum bank assembled from specimens collected in southern Texas. The

proportion of seropositive coyotes increased from 30% to 86% in the period 1975 to 1984, respectively, reflecting the establishment of enzootic infection (over 60% seropositive rate). The seropositive rate of distemper virus was age-dependent in this coyote population. Antibodies against canine distemper virus were found in 25%, 67% and 91% of coyotes less than 1-year-old, those from 1 to 2 years old, and those over 2 years old, respectively. This increase in seroprevalence with age is not reflective of a disease with high pup mortality. Conversely, it indicates that coyotes may be a reservoir and source of the infection of canine distemper virus for domestic dogs. Thus, distemper-induced mortality losses in the coyote population of southern Texas are regarded as compensatory with other mortality factors.

Canine parvovirus

In 1978 a previously unknown parvovirus caused an extensive epizootic of hemorrhagic enteritis and myocarditis in domestic dogs in North America. Canine parvovirus infection was characterized by high morbidity and mortality (10% to 50%) in young domestic dogs. Thomas et al. (1984) examined the seroprevalence of canine parvovirus in serum samples collected from coyote populations in southern Texas, Utah and Idaho between 1972 and 1983.

The onset of canine parvovirus seroprevalence in coyotes began in 1979, coinciding with the domestic canine epizootic. The seroprevalence rapidly increased to more than 70% by 1982 indicating enzootic establishment of the infection. Prevalence ultimately reached 90% to 100% in all sites. These high antibody prevalence rates are reflective of a highly contagious infection with low mortality rates. In 1980-81 just following introduction of canine parvovirus, the southern Texas coyote population experienced a decrease in population abundance. The decline resulted from increased pup mortality as reflected by lower juvenile/adult ratios (Pence et al. 1983). However, in the following years, coyote population abundance and juvenile recruitment subsequently returned to previous levels once canine parvovirus became enzootic.

Thus, in addition to distemper virus, the establishment of canine parvovirus as another new and highly contagious pathogen capable of causing

high juvenile mortality in a naive population failed to ultimately affect the abundance or composition of this coyote population.

Sarcoptic mange

Pence et al. (1983) and Pence and Windberg (1994) documented the effects of an epizootic of sarcoptic mange caused by the mite *Sarcoptes scabiei* in the coyote population of southern Texas from 1971-91. Although sporadic cases were reported previously, during the initial phase of the epizootic (1975-1978) mange prevalence increased from 14 to 24% in this coyote population. From spring 1979 to spring 1982 the mange prevalence peaked at 69% during the stationary period of the epizootic. The fall of 1982 marked the beginning of the decline phase of the epizootic with prevalences slowly decreasing to 0% by spring 1991. Subsequently, only sporadic cases have been reported.

From its point of origin in Webb County in 1975, the mange epizootic expanded centrifugally to encompass most of southern Texas during 1982-89, plus an unmeasured area in the adjacent Republic of Mexico. The high prevalences of mange, reaching nearly 70% at the peak of the epizootic with only about 1% of these animals recovering. Coupled with the decreased reproductive rates in mature territorial females infected with mange, the epizootic increased disease-induced mortality and natality rates in this coyote population.

Despite such mortality, the abundance and juvenile/adult ratios remained stable at levels consistent with a high-density population over the 21 year period of study (Pence and Windberg 1994). Thus, mange-induced mortality was regarded as compensatory with other mortality factors in this coyote population.

Hookworm

Radomski and Pence (1993) found that of 8 common species, there was temporal persistence of a small recurrent group of 3 dominant, unrelated species. This group dominates the intestinal helminth community in the coyote population of southern Texas. The dog hookworm (*Ancylostoma caninum*) was the most important pathogen of these 3 species. Further, it was the most abundant helminth, with prevalences always over 95% in all

host subpopulations over the 9-year study period.

Of all the species of helminths in this coyote population, hookworm is the only macroparasite that has the long-standing host-parasite relationship with an aggregated distribution that could effect the degree of density-dependent pathogenesis in juveniles (Anderson 1978, May 1983) necessary to regulate the host population. This effect would manifest itself by decreasing the number of juveniles available for recruitment. Hookworm disease-induced mortality results from a complex interaction of parasite density-, host age-, and nutritional-dependent factors in coyote neonates and juveniles (Radomski 1989)

Pence et al (1988) demonstrated that coyote pups were infected naturally at a very young age by transmammary transmission. Radomski (1989) showed that a threshold dose of about 300 infective hookworm larvae were sufficient to account for over 50% mortality in coyote neonates experimentally infected with hookworm in the first few weeks of life. Extrapolated to a free-ranging population, this indicates that juvenile mortality can be expected in populations with high hookworm abundances

In the coyote population of southern Texas, fall-collected juvenile (6 to 7 months old) coyotes still had very heavy infections (Pence and Windberg, 1984). There were 78%, 63%, 42%, and 24% of these juveniles with more than 150, 200, 250 and 300 hookworms, respectively (D. B. Pence and L. A. Windberg, unpublished data). These were juveniles which had survived the initial effects of hookworm disease due to heavy transcolostrally-acquired infections as neonates.

Because most hookworm infections of coyotes in southern Texas probably result from transmammary transmission (Pence and Windberg 1984, Pence et al 1988), and 78% of the 6 to 7 month old juveniles harbored over 150 hookworms, neonates which had slightly higher abundances of hookworms probably were lost from the population. About 25% of the 6 to 7 month old coyote neonates had over 300 hookworms, the LD₅₀ threshold of Radomski (1989) in experimentally-infected neonates

There was an associated hemorrhagic enteritis and ancylostomiasis in these juveniles which was complicated by high intensities of other intestinal helminths. Despite this, these animals appeared to be in reasonably good condition at the end of the

warm season and prior to the fall dispersion from the family group.

Based on overwinter juvenile mortality from fall-to-spring (Windberg et al. 1985), it is estimated that perhaps one-third of the coyote pups whelped in southern Texas die between birth and 6 months of age, with another one-third of these survivors dying during the first overwinter period (L. A. Windberg, pers. commun.).

The following may occur in at least some of the juvenile coyotes that survived the initial consequences of prenatal-colostrum hookworm infections, but maintained moderate-to-heavy hookworm infections through the summer and into early fall.

Food supplies in southern Texas are most abundant following whelping (Brown 1977), and neonates should be able to maintain the highest level of nutrition when they are part of a family group living in a territorial range. Dispersal of juveniles from parental territories occurs during the fall and early winter (Andelt 1985). Although fall food supplies appear adequate in most years, this is a period of dietary transition when diets shift from fruits as a major component to greater use of rodents and lagomorphs (Brown 1977). Therefore, heavy hookworm infections may compound an already nutritionally-, behaviorally-, and socially-stressed juvenile coyote. Thus, ancylostomiasis could have an effect on the growth rate and survival of juvenile coyotes during the fall and the subsequent overwinter period

Knowlton and Stoddart (1978) concluded that explanations regarding regulation of coyote populations were speculative. However, evidence at that time suggested that social intolerance, as mediated by abundances and availability of food, were the primary determinants of coyote densities. Behavioral characteristics are linked with survivorship. Although available evidence indicates that hookworm-induced juvenile mortality may provide a mechanism for regulation of this coyote population, this remains to be verified through further field studies

Conclusions

Coyote populations, such as the 1 in southern Texas that have been studied extensively, can suffer

what appear to be frequent and severe disease epizootics. The casual observer witnessing morbidity or episodes of mass mortality may interpret the effects of these epizootics as devastating to the population (Pence and Windberg 1994). However, the disease-induced mortality from distemper, canine parvovirus and mange that have recently caused epizootics in the coyote population of southern Texas was compensatory with other mortality factors. Probably the same effect will be observed in the present rabies epizootic, once the virus becomes enzootic. Though unproven, it is conjectured that the abundant and pathogenic dog hookworm represents the only macroparasitic infection that may effect regulation by reducing juvenile recruitment in this coyote population.

As emphasized by Pence and Windberg (1994) in their study of sarcoptic mange in the coyote population from southern Texas, more critical examination of host-disease ecological relationships may reveal an insignificant effect at the host population level. Alternatively, certain diseases could be very important to a host population if the effects of mortality were additive and contributed to the regulation of the population abundance at the threshold of its maximum sustainable density, as is suspected in hookworm infection. Thus, it is of importance to understand the actual effect of the common diseases on the specific host population in question prior to implementation of any intervention or control procedures for those diseases. Further diseases and parasites should be considered when developing an overall management or control strategy for any given host population.

Literature Cited

- Andelt, W. F. 1974. Behavioral ecology of coyotes in south Texas. Wildl. Monogr. 94. 45pp.
- Anderson, R. M. 1978. The regulation of host population growth by parasitic species. Parasitol 76: 119-157.
- Anderson, R. M. 1981. Fox rabies. Pages 242-261 in R. M. Anderson (Ed.) Population dynamics of infectious diseases. Chapman and Hall, London.
- Anonymous. 1995. (Unpublished) Monthly rabies reports. Tex. Dept Health, Publ. Health Region II, Harlingen.
- Brown, K. L. 1977. Coyote food habits in relation to a fluctuating prey base in south Texas. M.S. Thesis, Tex A&M Univ., College Station. 58pp.
- Gier, H. T., and D. J. Ameel. 1959. Parasites and diseases of Kansas coyotes. Kansas St. Univ. Agric. Exp. Sta. Bull. 91. 34pp.
- Guo, W., J. F. Evermann, W. F. Foreyt, F. F. Knowlton, and L. A. Windberg. 1986. Canine distemper virus in coyotes: A serologic survey. J. Am. Vet. Med. Assoc. 189. 1099-1100.
- Holmes, J. C., and P. W. Price. 1986. Communities of parasites. Pages 187-213 in D. J. Anderson and J. Kikpawa, (Eds.) Community ecology: Pattern and processes. Blackwell. Oxford.
- Knowlton, F. F., and L. C. Stoddart. 1978. Coyote population mechanics. Another look. Pages 93-111 in F. L. Bunnell, D. S. Eastman, and J. M. Peak, (Eds.) Symp. on natural regulation of wildlife populations. Forest, Wildl. Range Exper. Stat., Univ. Idaho. Moscow.
- May, R. M. 1983. Parasite infections as regulators of animal populations. Amer. Sci. 71: 36-45.
- Pence, D. B. 1990. Helminth community of mammalian hosts: concepts at the infracommunity, component and compound community levels. Pages 233-260 in G. W. Esch, A. O. Bush, and J. M. Aho, (Eds.) Parasite communities, patterns and processes. Chapman and Hall. N.Y.
- _____, and J. W. Custer. 1981. Host-parasite relationships in the wild Canidae of North America. II. Pathology of infectious diseases in the genus *Canis*. Pages 760-845 in J. A. Chapman and D. Pursley, (Eds.) Proc. Worldwide Furbearer Conference. R. R. Donnelley and Sons, Falls Church.
- _____, F. F. Knowlton, and L. A. Windberg. 1988. Transmission of *Ancylostoma caninum* and *Alaria marcianae* in coyotes (*Canis latrans*). J. Wildl. Dis. 24: 560-563.

- Pence, D. B., and L. A. Windberg. Population dynamics across selected variables of the helminth community in coyotes, *Canis latrans*, from south Texas. *J. Parasitol.* 70: 735-746.
- _____, and _____. 1994. Impact of a sarcoptic mange epizootic on a coyote population. *J. Wildl. Manage.* 58: 624-633
- _____, _____, and R. Sprowls. 1983. The epizootology and pathology of sarcoptic mange in coyotes, *Canis latrans*, from south Texas. *J. Parasitol.* 69: 1100-1115.
- Radomski, A. A. 1989. Host-parasite relationships of helminths in a coyote population from southern Texas with particular reference to dog hookworm. M.S, Thesis Texas Tech Univ., Lubbock 132pp
- _____, and D. B. Pence. 1993. Persistence of a recurrent group of intestinal helminth species in a coyote population from southern Texas. *J. Parasitol.* 79 371-378.
- Thomas, N. J., W. J. Foreyt, J. F. Evermann, L. A. Windberg, and F. F. Knowlton. 1984. Seroprevalence of canine parvovirus in wild coyotes from Texas, Utah, and Idaho (1972 to 1983). *J. Am. Vet. Med. Assoc.* 185: 1283-1287.
- Trainer, D. O., and F. F. Knowlton. 1968. Serologic evidence of diseases in Texas coyotes. *J. Wildl. Manage.* 32: 981-983.
- Windberg, L. A., H. L. Anderson, and R. M. Engeman. 1985. Survival of coyotes in southern Texas. *J. Wildl. Manage.* 49: 301-307.