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MORBIDITY—MORTALITY FACTORS AND SURVIVAL OF AN URBAN COYOTE POPULATION IN ARIZONA

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ABSTRACT: The health of coyotes (*Canis latrans*) in urban areas has not been studied. Our objectives were to assess the health of coyotes in Tucson (Arizona, USA) by determining the prevalence of antibodies to selected pathogens, estimating survival rates, and identifying sources of mortality. We drew blood from 22 coyotes to evaluate the prevalence of heartworm (*Dirofilaria immitis*) antigens, and antibodies to canine distemper virus (CDV), infectious canine hepatitis (ICH), canine parvovirus (CPV), and seven serovars of *Leptospira interrogans*. We trapped and radiocollared 19 coyotes to determine survival rates. We performed necropsies on 19 coyotes to quantify their general health, the presence of internal and external parasites, and causes of mortality. No coyotes tested positive for heartworm antigens. The prevalence of antibody to CDV, ICH, and CPV was 27, 50, and 100%, respectively. Twenty-seven percent of coyotes tested positive for one of five serovars of *L. interrogans*. The diseases for which coyotes in Tucson possessed antibodies appear to be enzootic in the population. The annual survival rate of coyotes was 0.72. Eleven necropsied coyotes were killed by cars, five coyotes were hit by cars, two were killed by a trapper, and the cause of death for one coyote was unknown. Coyotes in Tucson appear to be exposed to the viral, bacterial, and parasitic infections common in many coyote populations, but humans are the major source of mortality.

Key words: Canine distemper, canine parvovirus, *Canis latrans*, heartworm, infectious canine hepatitis, leptospirosis, survival rate.

Coyotes (*Canis latrans*) are one of the most widely-studied canids in North America (Bekoff, 1982). As such, factors related to their survival have been examined in depth. In particular, many studies have examined the survival of coyotes and causes of mortality in different locations (Gese et al., 1989; Windberg et al., 1997). The pervasiveness of sarcoptic mange (Pence and Windberg, 1994) and internal parasites (Radomsky and Pence, 1993) in coyotes have also been topics of research. In the past two decades, studies have also evaluated the prevalence of pathogens in coyotes (Cypher et al., 1998).

In recent years, coyotes have become common in many urban areas (Baker and Timm 1998). No studies, however, have assessed the health and condition of coyotes in urban areas. It is important to document the health of urban coyotes to establish the impact of diseases on populations in these areas. Furthermore, coyotes in urban areas may be reservoirs and sources of infection of common canid diseases for domestic dogs (Guo et al., 1986).

Our objectives were to evaluate the health of coyotes in Tucson (Arizona, USA) by determining the prevalence of pathogens known or suspected of being able to produce mortality in coyotes (Gier et al., 1978), estimating survival rates, and identifying sources of mortality.

MATERIALS AND METHODS

We captured and collared coyotes in Tucson, and a few urbanized areas directly outside of the city limits (32°09′ to 32°22′N, 110°44′ to 111°01′W). Tucson, which is in eastern Pima County, encompasses about 500 km^2 with an estimated human population of 475,000 (Tucson Update, 1999). Tucson is in the Sonoran Desert, with an elevation of about 745 m in midtown. The climate is characterized by low, unevenly distributed rainfall (about 28 cm annually; Sellers and Hill, 1974), low humidity, high air temperatures and periodic strong winds (Hastings and Turner, 1965).

A professional trapper live-trapped coyotes using padded leg-hold traps (i.e., #3 Victor Softcatch Coilspring, Woodstream Corp., Lititz, Pennsylvania). We immobilized each

trapped coyote with a noose rod, muzzle, and nylon stockings to tie its legs (Woolsey, 1985). We then fit coyotes with a uniquely-colored radiocollar (Telonics Inc., Mesa, Arizona).

We weighed each coyote, determined the sex and reproductive condition, and classed coyotes as juveniles <12-mo-old, yearlings 1-to 2 yr-old, or adults >2-yr-old by looking at tooth wear (Gier, 1968) and, for young of the year, by looking at the condition of the coat and tail. We extracted a 10-ml blood sample from a saphenous vein of trapped coyotes. Finally, we evaluated the animal's general health by checking for external parasites (including the mange mite, *Sarcoptes scabiei*), wounds, or other obvious signs of ill heath, and released it at the trapping site.

If a coyote had sarcoptic mange, we assigned it to 1 of 3 categories, depending upon the severity of the mange infectation (Pence et al., 1983). Prior to performing blood tests, each blood sample was placed into a glass serum tube (Vacutainer, Becton Dickinson, Rutherford, New Jersey, USA) and centrifuged for 30 min; the serum was then harvested and stored at −20 C. The serum samples were analyzed for the presence of heartworm (*Dirofilaria immitis*) antigens, and antibodies to canine distemper virus (CDV), and seven serovars of *Leptospira interrogans* at the University of Arizona Veterinary Diagnostic Laboratory (Tucson, Arizona). Serum samples were sent to the Texas Medical Diagnostic Laboratory (College Station, Texas, USA), to be analyzed for antibodies to infectious canine hepatitis (ICH) and canine parvovirus (CPV). We tested for the presence of heartworm antigens using an enzyme immunoassay (Snap®, IDEXX Laboratories, Inc., Westbrook, Maine, USA). The presence of CDV and ICH antibodies was determined by the serum virus neutralization test described by Appel and Robson (1973). A titer of ≥1:16 was considered positive for antibodies against CDV (Gese et al., 1997). A titer level of ≥1:10 was considered positive for antibodies against ICH (Gese et al., 1997). We used the hemagglutination inhibition test following the procedures outlined by Carmichael et al. (1980) to detect antibodies against CPV. A titer of ≥1:100 was considered positive for CPV antibodies (Gese et al., 1997). A CPV titer level of ≥1:1,280 was considered evidence for a recent infection (Carmichael et al., 1980). Antibodies for seven serovars of the bacterium *L. interrogans* (*bratislava, canicola, icterohemorrhagiae, pomona, hardjo, szwajizak*, and *grippotyphosa*) were detected using the microscopic agglutination test (Office International Des Epizooties, 1996). A titer of ≥1:100 was considered evidence of exposure to leptospires (Gese et al., 1997).

To determine survivorship, we relocated coyotes ≥ two times/week by homing with handheld Yagi antennas (White and Garrott, 1990: 42). We estimated the date of death as the midpoint between an animal's last recorded location and the date of discovery, unless carcass condition or observations from the public provided a basis for a more accurate date of death. We estimated overall survival estimates and obtained separate estimates for males, females, adults, and a combined yearling-and-juvenile (non-adult) class by Pollack et al.'s (1989) modification of Kaplan and Meier's (1958) nonparametric technique. We converted Kaplan-Meier estimates of survival (S) from 11 October 1996 through 28 February 1999 (870 days) to annual survival rates (S_{an}) to enable us to make comparisons with other studies, by the formula: $S = (S_{an})^{365/870}$ (Holzman et al., 1992). We used Cox and Oakes' (1984) method of approximating variance, which we then used to construct 95% confidence intervals (Pollack et al. 1989). We tested differences in survival rates between sexes and age classes using a normal approximation *z*-test (Pollack et al., 1989).

To determine sources of mortality for coyotes in Tucson, we performed necropsies on collared animals that died during the study and uncollared animals reported to us by The Arizona Game and Fish Department and Pima Animal Control Officers. Animals had usually been dead >24 hr when we collected them; we froze them immediately upon receipt. We thawed animals for 24–48 hr before performing necropsies. Animals were not frozen immediately upon death, therefore we were only able to make general determinations about the presence of disease (i.e., occurrence of helminths, broken bones, damage to internal organs). As a crude index of health, we subjectively rated the amount of fat on the kidneys and mesentaries as absent (poor health), light (fair health), or moderate-to-heavy (good health) (Pence et al., 1983).

Most of these coyotes were in good health when trapped; three had mange, two of which had severe cases with over half of their bodies affected. We trapped and radiocollared 14 coyotes from October 1996 through March 1997, and five coyotes from December 1997 through January 1998. We trapped three additional animals in July and August 1996 but did not collar them because they were sick or young.

We collected blood from 22 coyotes (Table 1). No coyotes tested positive for heartworm antigens. Antibodies to leptospirosis, CDV, CPV, and ICH were detected (Table 1). The weights of 19 trapped animals ranged from 7.3

TABLE 1. Weight (kg), health, and serological titers to selected pathogens of coyotes captured in Tucson, Arizona, July 1996 through January 1998.

Age/sex	*n*	Mean weight	Serologic results[a]							
			Leptospira interrogans bratislava	*L. interrogans canicola*	*L. interrogans icterohemorragiae*	*L. interrogans pomona*	*L. interrogans grippotyphosa*	CDV[b]	CPV[c]	ICH[d]
Adult males	3	13.3 (11.8–15.5)	0	0	0	0	0	2 (16–128)	3 (640–5,120)	3 (64–256)
Adult females	7	10.2 (8.6–11.8)	1 (100)	0	0	0	1 (100)	3 (32–256)	7 (320–2,560)	4 (32–128)
Yearling males	3	10.5 (8.2–12.7)	1 (100)	0	0	1 (100)	2 (100–400)	0	3 (640–2,560)	0
Juvenile male	7	9.6 (7.3–12.3)	3 (100)	3 (100)	3 (100)	3 (100)	3 (100–6,400)	1 (256)	7 (640–2,560)	3 (128–1,024)
Juvenile female	2	9.6 (9.1–10.0)	0	0	0	0	0	0	2 (2,560–5,120)	1 (1,024)

[a] Number of positive (range of observed antibody titers).
[b] Canine distemper virus.
[c] Canine paravirus type 2.
[d] Infectious canine hepatitis.

TABLE 2. Kaplan-Meier annual survival probabilities for coyotes in Tucson, Arizona, October 1996–February 1999.

Group	*n*[a]	Annual survival rate[b]	95% confidence interval
All animals	19	0.7249	0.5032 to 0.9466
Males	13	0.8571	0.6170 to 1.0972
Females	6	0.6307	0.3228 to 0.9386
Adults	11	0.6542	0.4000 to 0.9084
Yearling-juveniles	8	0.8389	0.5538 to 1.1240

[a] Sample size of coyotes.
[b] Transformed from an estimated survival rate of coyotes, 11 October 1996–28 February 1999.

kg for a juvenile male to 15.5 kg for an adult male (Table 1).

Overall annual survival was 0.72 and there was no difference in the survival rates of males versus females ($z = 1.14$, $P = 0.13$) or adults versus non-adults ($z = 0.94$, $P = 0.17$) (Table 2). Of the 19 animals radiocollared, we determined the date of death and fate of nine. All collared animals were in good or fair health when collared; of the four whose condition was known at the time of death, three were in good health, and one was in poor health. Seven coyotes were hit by cars, one was euthanized by an Arizona Game and Fish Department officer, and one was killed by a trapper.

We performed necropsies on 19 animals (Grinder, 2000). Most coyotes were in good health when they died but one had a severe case of mange (Mange III).

Canine heartworm disease was not an important cause of mortality for coyotes in Tucson during our study. By contrast, animals in Georgia (Holzman et al., 1992) and other southeastern states (Custer and Pence, 1981) tested positive for canine heartworm. Weinmann and Garcia (1980) demonstrated that 45% of coyotes from central California had heartworms and postulated that coyotes were a good potential reservoir for disease transmission to domestic canids.

The prevalance of 27% CDV seroprevalence in coyotes in Tucson is among the lowest reported. Other studies have found prevalences as high as 76% (Gese et al., 1997) in Yellowstone National Park, Wyoming, 57% in Colorado (Gese et al., 1991), 56% and 37% in Texas (Trainer and Knowlton, 1968), 50% in other parts of Wyoming (Williams et al., 1988), and 37% in California (Cypher et al., 1998). Holzman et al. (1992), however, found no antibodies to CDV in coyotes in Georgia.

Five of 6 coyotes that tested positive for

CDV antibodies were adults; canine distemper virus may be fatal to young coyotes not protected by antibodies (Gier et al., 1978), which would minimize the chances of capturing juvenile or yearling animals with antibodies to the disease. Other studies have found an increase in prevalence of CDV antibodies with increasing age (Guo et al., 1986; Gese et al., 1997).

The prevalence of antibodies to ICH was 50% in coyotes in Tucson, similar to the levels reported by Trainer and Knowlton (1968) in Texas (51%) and by Holzman et al. (1992) in Georgia (41%). By contrast, Gese et al. (1997) found >80% seroprevalence in coyotes in Yellowstone National Park although the population was healthy and had normal natality rates (Gese, 1995).

The prevalence of CPV in coyotes in Tucson is high compared to many other studies. We found 100% exposure to CPV; Gese et al. (1997) also reported 100% exposure to CPV in all coyotes in Yellowstone National Park, Wyoming, except among pups ≤3 months old. In Texas, Utah, Idaho, and Colorado, >70% of the coyotes had antibodies to CPV (Thomas et al., 1984; Gese et al., 1991). In Georgia (Holzman et al., 1992) and California (Cypher et al., 1998) 65% of coyotes had antibodies to CPV. Thomas et al. (1984) considered CPV antibody prevalence >50% to be "high." The CPV virus is extremely resistant to heat and desiccation, so many coyotes are probably exposed to contamination. Although CPV is most likely widespread throughout coyotes populations, only one report has been made of a coyote dying from CPV enteritis (Holzman et al., 1992).

Close to one third of the coyotes in our study tested positive for antibodies to at least one serovar of *L. interrogans.* Other studies have found much lower evidence of exposure to leptospirosis (Trainer and Knowlton, 1968; Holzman et al., 1992; Gese et al., 1997). Drewek et al. (1981), however, reported that 44% of coyotes in another Arizona study tested positive for leptospirosis.

Some researchers have suggested that CDV, ICH, CPV, and other viral and bacterial diseases have the capacity to exist in an enzootic state within coyote populations (Thomas et al., 1984; Pence, 1995) and may only cause significant mortality during stressful conditions such as high density, food scarcity, or parasitism (Trainer and Knowlton, 1968; Pence and Custer, 1981).

The annual survival rate of coyotes in Tucson (0.72) is similar to that reported by other studies in North America, which range from 0.68 to 0.90 (Andelt, 1985; Harrison, 1992). Some studies, however, have found survival rates to be in the range of 0.38 to 0.50 (Roy and Dorrance, 1985; Holzman et al., 1992).

All collared coyotes and most coyotes (95%) on which we performed necropsies were killed by humans; most of these were hit by cars. Mortality due to shooting, trapping, poisoning, or road fatalities is high in many coyote populations (Pence et al., 1983; Gese et al., 1989) and reported percentages of coyotes killed by humans ranges from 22% in Georgia (Holzman et al., 1992) to 95% in Utah (Mills and Knowlton, 1991). Gese et al. (1989) found that the mortalities of all dispersers and transients were human-related. In general, the survival of adult coyotes tends to be lower in areas with greater human exploitation (Windberg et al., 1985) and in Tucson, adult coyotes appeared to be as susceptible to being hit by cars as non-adults.

Three of the 22 coyotes (14%) from which we drew blood showed signs of mange, and one of the necropsied animals appeared to have a severe case of mange. Three of these animals were males. Pence et al. (1983) suggested that adult males may have greater contact with other coyotes and may therefore be more likely to contract and transmit mange. Pence et al. (1983) and Pence and Windberg (1994) documented the effects of a mange epizootic in a coyote population in southern Texas from 1971 to 1991 and found that coyotes with mange had lower reproductive rates and higher mortality rates due to other diseases than did non-infected coyotes. Despite such population effects, mange-induced mortality was considered compensatory with other mortality factors in the population. To the urban public, easily visible diseases such as mange may appear devastating to a species but a more thorough examination of the disease-host relationship may reveal an insignificant effect at the host population level (Pence and Windberg, 1994).

Coyotes in Tucson appear to have exposure to viral, bacterial and parasitic infections that are common in many coyote populations. Indications are that their body condition is similar to those of coyotes in more rural areas. As in less-urbanized locations, man constitutes the major source of mortality for coyotes. Coyotes in urban areas are exposed to the same factors that confront coyotes elsewhere. The potential exists for urban coyotes to be sources of disease for domestic canids in these areas, but it is not clear how important coyotes may be in this capacity.

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