The Dynamics of Sarcoptic Mange in an Urban Coyote (Canis latrans) Population

THESIS

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By

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Abstract

Coyotes (*Canis latrans*) are the top predator in the metropolitan Chicago area, and other urban areas of North America. As such, coyotes play an important role in the dynamics of the urban ecosystem. Coyotes have increasingly come into conflict with humans as both human and coyote populations have increased in the areas surrounding major cities. Sarcoptic mange is an important disease of coyotes throughout their range, and is capable of epizootics with high prevalence and mortality rates. My objectives were to examine the dynamics of sarcoptic mange in an urban coyote population, determine the effects of changes in mange prevalence on coyote population dynamics and determine whether mange infection resulted in altered habitat selection and use.

Coyotes were trapped and radio-collared during 2000 - 2011. Individuals were examined for signs of sarcoptic mange at the time of capture. Visual observations of individuals being radio-tracked meant that animals that had developed signs of sarcoptic mange could be identified based on hair loss patterns after capture.

Three hundred ten coyotes were examined for signs of sarcoptic mange during the course of the study and 49 (16%) were diagnosed with sarcoptic mange at some point. Sarcoptic mange incidence remained relatively steady in the study population throughout the study, implying that mange infection during this time period was enzootic. The majority of mortalities due to

sarcoptic mange occurred during the winter (December – January). There was no evidence that changes in the prevalence of sarcoptic mange had any effect on annual survival rates, nor was there any relationship between annual survival and mange-specific mortality rates. Coyotes with sarcoptic mange showed a significantly higher mean percentage of locations in medium-density urban areas during the period spanning 60 days prior to death.

Sarcoptic mange is currently enzootic in the Chicago metropolitan area. This does not preclude epizootics in the future, as sarcoptic mange has been documented in the literature to remain enzootic in a population and until eventually erupting in an epizootic over a larger area at a later date. I found no evidence that mortality due to mange was additive or compensatory with other forms of mortality in the population. At the current time, sarcoptic mange does not seem the occur in high enough prevalence to have a major impact on coyote populations in this area, although it is an important cause of mortality in the system and may have impacts on coyote abundance at a local scale. Additionally, sarcoptic mange appears to affect the behavior of heavily infected individuals which may contribute to human-coyote conflict. The combination of increased mortality during winter months and increased residential habitat use prior to mortality may be an important factor in human-coyote conflict in the Chicago metropolitan region and other urban areas with enzootic mange and cold winters.

Information on the factors that contribute to coyote mortality in urban systems can increase our knowledge about the factors which regulate coyote populations in urban areas, increasing our ability to effectively manage coyote populations. Understanding reasons why human-coyote conflicts take place can not only inform management decisions on how to handle

conflicts, but educating the public about causes of conflict can prevent conflicts from occurring in the first place.

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Chapter 1

A Review of Sarcoptic Mange in Urban Coyote (Canis latrans) Populations

INTRODUCTION

The coyote (*Canis latrans*) is a medium-sized member of the mammalian family Canidae, native to North America. As a member of the genus Canis, its closest relatives include wolves, jackals and the domestic dog. Coyotes are known to consume a wide variety of prey items including, but not limited to deer, lagomorphs, small mammals, birds, fruit, and insects (Harrison and Harrison 1984, Andelt et al. 1987, Morey et al. 2007). Fossil records indicate that the coyote's pre-European range was primarily the southwest United States, Rocky Mountains and Great Plains regions. However, in recent years, the coyote's range has expanded from its historic range through almost all of the continental United States (Bekoff 1977). While the coyote ranks as one of the most widely studied mammals in North America, large gaps remain in our understanding of its ecology and life history.

The coyote is the largest predator common in metropolitan Chicago. While the exact number of coyotes living in the metropolitan region is unknown, and probably in constant

flux, they appear capable of using most any habitat available to them. Coyotes are found throughout the metropolitan Chicago area including the most populated areas of downtown Chicago. Coyotes in urban and suburban areas often provoke strong reactions among residents. While some individuals enjoy the presence of coyotes in their neighborhood, others view coyotes as a menace and a potential danger to themselves, their children, and their pets. Regardless of an individual's personal feelings toward coyotes, they are here to stay in the region and continued research into the reasons for their success and role in the ecosystem may serve to prevent conflicts between humans and coyotes in this system.

Sarcoptic mange is a cause of mortality for coyotes in the metropolitan Chicago area. Understanding the processes by which mange affects coyote populations can increase our knowledge of coyote population dynamics in this suburban area. As public awareness of coyote presence in this area increases, expanding knowledge of coyote ecology in this system will aid in educating the public as to safety concerns, conflict avoidance and informing management decisions.

COYOTES IN URBAN ENVIRONMENTS

Behavioral Changes in Urban Environments

Populations of animals that live in urbanized areas often display different behavioral characteristics than those of populations in rural areas (Ditchkoff et al. 2006). The coyote's adaptability has served the species well in urban environments, allowing them to colonize areas that are inhospitable for competitors. In Indiana, home range sizes varied along a suburban to rural gradient and were inversely related to urbanization (Atwood et

al. 2004). Several studies in urban areas have observed a shift in periods of activity from crepuscular to nocturnal (McClennen et al. 2001, Atwood et al. 2004, Grubbs and Krausman 2009). Grubbs (2009) hypothesized that urban coyotes switched from crepuscular to nocturnal activity to avoid human contact in Arizona. Urban coyotes in Arizona tended to avoid areas of high human activity, and utilized washes (dry riverbeds) that ran through Tucson for cover and travel corridors. Other studies have agreed that coyotes tend to avoid areas where natural components are absent in urban environments (Gibeau 1998, Grinder and Krausman 2001b). In a survey of coyote diets in the Chicago area, anthropogenic food sources were significantly present in coyote scats in only the most urbanized areas (Morey et al. 2007). Mean nightly movements by coyotes are also greater in suburban areas than have been reported in other study areas (Way et al. 2004). A possible reason for this is the fragmented nature of the ecosystem, in which animals must cross areas of poor habitat to reach natural areas encompassed in their home range (Way et al. 2004).

Impacts on other urban species

The presence of coyotes affects other species living in the urban matrix. Gosselink (2007) reported that 40% of red fox (*Vulpes vulpes*) mortalities in a rural areas in Illinois were due to coyote predation. As both coyotes and red fox appear in urban areas as well, it is reasonable to assume that coyotes have a similar impact on red foxes in urban environments. Brown (2007) observed that coyotes in the Chicago metropolitan area frequently predate Canada goose (*Branta canadensis*) nests and also feed on adult geese. Deer fawns are a frequent prey source for coyotes (Saalfeld and Ditchkoff 2007),

particularly during the pup-rearing season (Kilgo et al. 2010). In fact, fawns may be preferred over other sources of prey by breeding individuals, since it requires less effort to bring large prey items back to the den than small ones (Harrison and Harrison 1984). Costs of the Urban Lifestyle

While coyotes thrive in urban environments, the urban matrix is not without its hazards. Automobile collisions are a major source of mortality (Grinder and Krausman 2001a, Gehrt 2007). In addition, the large highways associated with urban areas act as barriers to dispersal and migration (Riley et al. 2006). Riley et al. (2006) established that freeways could restrict gene flow in wide ranging species based on genetic differentiation among coyotes on opposite sides of the Ventura Freeway in Los Angeles. Their results also implied that individuals who did manage to cross the freeway rarely reproduced. Lack of reproductive success was attributed not only to mortalities caused by vehicles, but by highways acting as an artificial boundary for home ranges, leading to territorial pileup along these boundaries (Riley et al. 2006). A similar effect has been seen among dispersing animals when they come across a river or other body of water (Harrison 1992). Roads can also affect animals' daily movement patterns. Red foxes in Bristol, UK showed a change in expected activity patterns to avoid major roads, that were a major source of mortality in this environment (Baker et al. 2007).

Coyote Social Organization

Pence and Windberg (1994) proposed that the coyote social structure can serve to decrease the severity of epizootics from diseases such as sarcoptic mange. Coyotes are a territorial monogamous species whose base social unit is the mated pair (Andelt 1985,

Hennessey 2007). The mated pair may share its home-range with one or more helper animals, who do not breed, to form a resident group or "pack" (Andelt 1985). Genetic analyses of packs from the Chicago area have shown that coyotes in this system are monogamous with no observed incidents of extra-pair copulations (Hennessey 2007). Pack formation in coyotes is hypothesized to be caused by the delayed dispersal of juvenile individuals (Messier and Barrette 1982). These individuals maintain their home ranges inside their natal range past the time when they could potentially reproduce. A possible advantage to delayed dispersal is that it allows the individual to gain more experience in a safe environment, thereby increasing its chance of survival and success when it does disperse. Adult and yearling coyotes in Yellowstone National Park were observed to have lower rates of attempted captures of small mammals than pups. Though the older animals made fewer attempts to catch small mammals than young ones the number of prey captured per hour was the same (Gese et al. 1996). Increased formation of packs may also be a function of population density. The highest degree of coyote sociality has been observed in wildlife reserves where coyote densities are high (Messier and Barrette 1982). Some individuals may make exploratory movements outside of their natal territory prior to dispersal and return before finally dispersing (Harrison et al. 1991). These movements may be instrumental in the introduction of disease to resident groups.

In addition to resident animals there are also transient individuals, solitary non-breeding animals whose home range may overlap the home ranges of several resident animals, that constitute coyote populations (Gese et al. 1989). Transient animals are most likely individuals who are in the 'vagrant' stage of dispersal. Hence, transient individuals

may occur in higher proportions in areas where available territory space is low. Proportions of these social classes may vary within the population depending on food availability and other environmental factors. Andelt (1985) reported, the population was estimated at 70% resident groups, 17% resident pairs and 13% transient animals in Welder Wildlife Refuge in Texas. Alternatively, a study based in the forests of Quebec estimated that the winter population was 35% resident groups, 28% resident pairs and 37% transient animals (Messier and Barrette 1982). Little information is available about the movements of transient animals due to the difficulty involved in tracking them (Gehrt 2007). However, in the course of some studies, previously transient individuals have established territories and become resident animals. A study in Kansas showed avoidance of grassland areas among transient animals, while at the same time showing selection for grassland areas among resident animals (Kamler and Gipson 2000). The authors proposed that this was due to transients avoiding territorial confrontations with resident animals.

Disease in Coyote Populations

Coyotes can be host to a wide variety of parasites and pathogens capable of causing disease including macroparasites such as ticks, fleas, lice, tapeworms, and roundworms and microparasites such as viruses and bacteria (Gier et al. 1978). Canine distemper virus (CDV) and canine parvovirus (CPV) are enzootic in many coyote populations in North America (Gese et al. 1991, Holzman et al. 1992, Gese et al. 1997, Almberg et al. 2009, Miller et al. 2009). Little information exists on the effects of disease-related mortality on coyote population dynamics. CDV has been shown to be

highly lethal of pups in captivity and is suspected of taking a high toll on coyote pups in the wild (Gier et al. 1978). Almberg (2009) observed that the presence of CPV, CDV and canine adenovirus (CAV) did not appear to have long-term impacts on canid populations in Yellowstone National Park. Nelson (2003) hypothesized that canine heartworm (*Dirofilaria immitis*), while capable of negatively affecting individual coyotes' condition, are only a minor factor in coyote population dynamics in Illinois. Sarcoptic mange has been observed in many coyote populations (Pence and Ueckermann 2002), and is capable of affecting the population dynamics of other canid species (Lindstrom and Morner 1985).

SARCOPTIC MANGE

Sarcoptic mange is widespread in coyote populations. Outbreaks of sarcoptic mange in coyotes have been documented in Wisconsin (Trainer and Hale 1969), Alberta (Todd et al. 1980), south Texas (Pence et al. 1983, Pence and Windberg 1994), Kansas (Kamler and Gipson 2002) and South Dakota (Chronert et al. 2007) and northeastern Illinois (Gehrt et al. 2009).

Sarcoptic mange is caused by the mite *Sarcoptes scabiei* (Acariformes: Sarcoptidae) (Fain 1978). Cases of sarcoptic mange have been reported in at least 10 orders, 27 families and 104 mammalian species (Pence and Ueckermann 2002). A variety of this mite, *Sarcoptes scabiei var. hominis*, causes the disease scabies in humans. While experimental transfers of mites between hosts have indicated some host specificity (Samuel 1981, Arlian et al. 1984b, Arlian et al. 1996a), molecular genetic studies have indicated that the genus *Sarcoptes* is monospecific (Zahler 1999). Consequently, mites

are referred to as variants based on the host from which they were isolated from (i.e. *Sarcoptes scabiei var. canis*) (Bornstein et al. 2001). Andrews (1983) proposed that *S. scabiei* was originally a parasite of primates and was passed from humans to domesticated canids to wild canids.

Sarcoptic mange is capable of causing epizootics over large areas with high prevalence and mortality rates (Trainer and Hale 1969, Todd et al. 1980, Morner 1992, Pence and Windberg 1994, Fernandez-Moran et al. 1997, Skerratt et al. 1998, Soulsbury et al. 2007). Pence and Ueckermann (2002) suggested that mange epizootics occur in some species, including coyotes, on 30-45 year cycles, with an epizootic being prompted by the appearance of a more virulent strain of mite. While it is proposed that mange outbreaks are related to high population density along with reduced prey abundance, several studies of epizootics noted high prey levels during the same period (Pence and Windberg 1994, Chronert et al. 2007).

Transmission

Mites are transmitted among individuals primarily through body contact (Andrews 1983). Mange can also be transferred to individuals living in the dens of mange-infected animals (Bornstein et al. 2001). Sarcoptic mites that become dislodged from hosts can detect odor and heat from a host and seek the source (Arlian et al. 1984c). Andrews (1983) proposed that phoretic transport may also play a role in transmission, as mites have been observed on the ovipositors of flesh flies for up to 24 hours after feeding on an infected carcass.

Mites are capable of surviving off-host for extended periods of time (Arlian et al. 1984a). In laboratory experiments on *Sarcoptes scabiei var. canis* and var. *hominis*, Arlian (1984a) determined that survival time off of hosts depends on ambient temperature and relative humidity, with longer survival times associated with lower temperatures and higher relative humidity. *Sarcoptes* mites were capable of surviving for 24-36 hours off of host at room temperature, and were still capable of infecting a new host (Arlian et al. 1984a).

Pathology

Upon arrival on a new host the mite secretes a solution that dissolves tissue and creates a small depression in the epidermis (Arlian et al. 1984a). The mite burrows into the host's epidermis and feeds on living cells and tissue fluids accumulating in the burrow. The initial signs of mange infection are the appearance of non-pruretic lesions on the skin. After this stage, there are major differences in host reactions based on whether the host is able to mount a hypersensitivity response. This ability is based primarily on whether the host's immune system has encountered the mite previously, or whether previously exposed hosts are immunologically compromised and unable to mount a response (Arlian et al. 1996b, Pence and Ueckermann 2002).

In individuals capable of a hypersensitivity response, the initial lesions become extremely pruritic (itchy), and there is hyperkeratosis (thickening of the *stratus corneum*), alopecia (hair loss) and inflammation of the epidermis (Bornstein et al. 2001). As the disease progresses the skin becomes thickened due to hyperkeratosis, takes on a wrinkled appearance and is usually hairless and discolored. As the reaction progresses mites

become rare to absent in the skin. It has been proposed that the intense pruritis leads to self-inflicted wounds leading to secondary infection (Pence and Ueckermann 2002).

Disease shows a different progression due to the lack of a sensitivity response in immunologically compromised or naïve individuals (Pence and Ueckermann 2002). The level of alopecia is highly variable and often depends on the host. The skin lesions are characterized by "encrusting dermatitis" and are non-pruritic (Pence and Ueckermann 2002). The thickened crusts that form on the skin generally fissure and hemorrhage or form pustules. In these cases, there are often large numbers of mites present in the skin (Pence and Ueckermann 2002).

Walton and Currie (2007) suggested that mange may lead to secondary infection (pyoderma) in the skin, which may contribute to sepsis and death. Both *Streptococci* and *Staphylococci* have both been found in burrows and fecal pellets of *Sarcoptes scabiei var. hominis* on hosts suggesting some role of the mite in introducing bacteria (McCarthy et al. 2004). Pneumonia caused by sepsis was recorded as cause of death for several sarcoptic mange afflicted raccoon dogs (*Nyctereutes procyonoides*) in Japan (Nakagawa et al. 2009).

Physical effects of mange in coyotes

Coyotes with severe infections of sarcoptic mange are often in poor physical condition (Pence et al. 1983). Infected coyotes in south Texas with class III mange had lower body weights than those with milder or no infections (Pence et al. 1983). Greater numbers of uninfected adults also had higher indices of intra-peritoneal fat compared to infected adults (Pence et al. 1983). Weights of xiphoid-process fat globules (an index of

total body fat) of mange-infected coyotes were less than those of non-infected animals of both sexes and juvenile and adult age classes in Alberta (Todd et al. 1980).

Sarcoptic mange causes serological changes in infected individuals. Infected coyotes in south Texas showed lower levels of α -globulin and albumin, and significantly higher levels of γ -globulin (Pence et al. 1983). Domestic dogs (*Canis familiaris*) with mange have been shown to have increased levels of total leukocytes, T-lymphocytes, mast cells, neutrophyles, eosinophiles and α , β and γ globulins (Camkerten et al. 2009). An analysis by Camkerten (2009) on domestic dogs found an association between mange infection and high total oxidant status, lipid hydroperoxide, oxidative stress index and low concentrations of sera sulfhydryls, suggesting a possible relationship between mange infection and imbalance in oxidant/antioxidant levels.

Infection with sarcoptic mange also has an effect on breeding capability. Pence and Windberg (1994) observed that as severity of mange infection increased number of ova and viable fetuses in adult females decreased, additionally females with Class I-II mange had higher resorption of all fetuses than uninfected females. Todd et al. (1980) suspected that entire litters of mange infected females often contract mange. Mange-infected red foxes in Bristol, UK displayed decreased breeding potential shown by lack of breeding success in females and decreased testes size in males (Soulsbury et al. 2007).

Sarcoptic mange is not necessarily a lethal condition in coyotes. As with many diseases, disease outcome depends on the physical and immunological condition of the host. Approximately 1% of individuals in the population showed signs of recovery from mange in the form of re-growth of hair on the edges of lesions during an epizootic in

coyotes in southern Texas (Pence and Windberg 1994). Of coyotes initially infected with sarcoptic mange, 20% recovered from the disease in Wind Cave National Park (Chronert et al. 2007). Todd et al. (1980) also noted the examination of two individuals who were re-growing hair in Alberta. Treatment with ivermectin, an acaricide, is usually successful in halting infections in domestic species (Bornstein et al. 2001).

CONCLUSION

Sarcoptic mange is a common disease that affects many mammal species around the globe. Like many diseases, it can have a myriad of effects on the host species who become infected. In North America, coyotes are one of the most common hosts of sarcoptic mange, and it appears to be present throughout their range. Sarcoptic mange can have important implications for coyote populations particularly when it occurs in epizootics. Sarcoptic mange has also been associated with a variety of behavioral changes, specifically lethargy and a loss of wariness, seen in several species including coyotes. A closer examination of the host-parasite relationship between *Sarcoptes scabiei* and coyotes can provide new and important insight into how disease affects wildlife populations, particularly those in urban areas.

LITERATURE CITED

- Almberg, E. S., L. D. Mech, D. W. Smith, J. W. Sheldon, and R. L. Crabtree. 2009. A serological survey of infectious disease in Yellowstone National Park's canid community. PloS one 4:e7042.
- Andelt, W. F. 1985. Behavioral ecology of coyotes in south Texas. Wildlife Monographs 94:3-45.

- Andelt, W. F., J. G. Kie, F. F. Knowlton, and K. Cardwell. 1987. Variation in coyote diets associated with season and successional changes in vegetation. Journal of Wildlife Management 51:273-277.
- Andrews, J. R. 1983. The origin and evolution of host associations of *Sarcoptes scabiei* and the subfamily *Sarcoptinae Murray*. Acarologia 24:85-94.
- Arlian, L. G., M. S. Morgan, and J. J. Arends. 1996a. Immunologic cross-reactivity among various strains of *Sarcoptes scabiei*. Journal of Parasitology 82:66-72.
- Arlian, L. G., M. S. Morgan, C. M. Rapp, and D. L. Vyszenski-Moher. 1996b. The development of protective immunity in canine scabies. Veterinary Parasitology 62:133-142.
- Arlian, L. G., R. A. Runyan, S. Achar, and S. A. Estes. 1984a. Survival and infectivity of *Sarcoptes scabiei var. canis* and *var. hominis*. Journal of the American Academy of Dermatology 11:210-215.
- Arlian, L. G., R. A. Runyan, and S. A. Estes. 1984b. Cross infestivity of *Sarcoptes scabiei*. Journal of the American Academy of Dermatology 10:979-986.
- Arlian, L. G., R. A. Runyan, L. B. Sorlie, and S. A. Estes. 1984c. Host-seeking behavior of *Sarcoptes scabiei*. Journal of the American Academy of Dermatology 11:594-598.
- Atwood, T. C., H. P. Weeks, and T. M. Gehring. 2004. Spatial ecology of coyotes along a suburban-to-rural gradient. Journal of Wildlife Management 68:1000-1009.
- Baker, P. J., C. B. Dowding, S. B. Molony, C. L. W. Piran, and S. Harris. 2007. Activity patterns of urban red foxes (*Vulpes vulpes*) reduce the risk of traffic-induced mortality. Behavioral Ecology 18:716-724.
- Bekoff, M. 1977. Canis latrans. Mammalian Species 79:1-9.
- Bornstein, S., T. Morner, and W. M. Samuel. 2001. *Sarcoptes scabiei* and sarcoptic mange. Pages 107-119 *in* W. M. Samuel, M. J. Pybus, and K. A.A., editors. Parasitic Diseases of Wild Mammals. Iowa State University Press, Ames, IA.
- Brown, J. 2007. The influence of coyotes on an urban Canada goose population in the Chicago metropolitan area. Thesis, The Ohio State University, Columbus, OH.
- Camkerten, I., T. Sahin, G. Borazan, A. Gokcen, O. Erel, and A. Das. 2009. Evaluation of blood oxidant/antioxidant balance in dogs with sarcoptic mange. Veterinary Parasitology 161:106-109.

- Chronert, J. M., J. A. Jenks, D. E. Roddy, M. A. Wild, and J. G. Powers. 2007. Effects of sarcoptic mange on coyotes at Wind Cave National Park. Journal of Wildlife Management 71:1987-1992.
- Ditchkoff, S. S., S. T. Saalfeld, and C. J. Gibson. 2006. Animal behavior in urban ecosystems: Modifications due to human-induced stress. Urban Ecosystems 9:5-12.
- Fain, A. 1978. Epidemiological problems of scabies. International Journal of Dermatology 17:20-30.
- Fernandez-Moran, J., S. Gomez, F. Ballesteros, P. Quiros, J. L. Benito, C. Feliu, and J. M. Nieto. 1997. Epizootiology of sarcoptic mange in a population of cantabrian chamois (*Rupicapra pyrenaica parva*) in northwestern Spain. Veterinary Parasitology 73:163-171.
- Gehrt, S. D. 2007. Ecology of coyotes in urban landscapes. Pages 303-311 in Proceedings of 12th Wildlife Damage Management Conference. The Wildlife Society, 9 12 April 2007, Corpus Christi, TX, USA.
- Gehrt, S. D., C. Anchor, and L. A. White. 2009. Home range and landscape use of coyotes in a metropolitan landscape: conflict or coexistence? Journal of Mammalogy 90:1045-1057.
- Gese, E. M., O. J. Rongstad, and W. R. Mytton. 1989. Population dynamics of coyotes in southeastern Colorado. Journal of Wildlife Management 53:174-181.
- Gese, E. M., R. L. Ruff, and R. L. Crabtree. 1996. Intrinsic and extrinsic factors influencing coyote predation of small mammals in Yellowstone National Park. Canadian Journal of Zoology 74:784-797.
- Gese, E. M., R. D. Schultz, M. R. Johnson, E. S. Williams, R. L. Crabtree, and R. L. Ruff. 1997. Serological survey for diseases in free-ranging coyotes (*Canis latrans*) in Yellowstone National Park, Wyoming. Journal of Wildlife Diseases 33:47-56.
- Gese, E. M., R. D. Schultz, O. J. Rongstad, and D. E. Andersen. 1991. Prevalence of antibodies against canine parvovirus and canine distemper virus in wild coyotes in southeastern Colorado. Journal of Wildlife Diseases 27:320-323.
- Gibeau, M. L. 1998. Use of urban habitats by coyotes in the vicinity of Banff Alberta. Urban Ecosystems 2:129-139.

- Gier, H. T., S. M. Kruckenberg, and R. J. Marler. 1978. Parasites and Diseases of Coyotes. Pages 37-71 *in* M. Bekoff, editor. Coyotes: Biology, Behavior and Management. The Blackburn Press, Caldwell, NJ.
- Gosselink, T. E., T. R. Van Deelen, R. E. Warner, and P. C. Mankin. 2007. Survival and cause-specific mortality of red foxes in agricultural and urban areas of Illinois. Journal of Wildlife Management 71:1862-1873.
- Grinder, M., and P. R. Krausman. 2001a. Morbidity-mortality factors and survival of an urban coyote population in Arizona. Journal of Wildlife Diseases 37:312-317.
- Grinder, M. I., and P. R. Krausman. 2001b. Home range, habitat use, and nocturnal activity of coyotes in an urban environment. The Journal of Wildlife Management 65:887-898.
- Grubbs, S. E., and P. R. Krausman. 2009. Use of urban landscape by coyotes. Southwestern Naturalist 54:1-12.
- Harrison, D. J. 1992. Dispersal characteristics of juvenile coyotes in Maine. Journal of Wildlife Management 56:128-138.
- Harrison, D. J., and J. A. Harrison. 1984. Foods of adult Maine coyotes and their knownaged pups. The Journal of Wildlife Management 48:922-926.
- Harrison, D. J., J. A. Harrison, and M. O'Donoghue. 1991. Predispersal movements of coyote (*Canis latrans*) pups in eastern Maine. Journal of Mammalogy 72:756-763.
- Hennessey, C. A. 2007. Mating strategies and pack structure of coyotes in an urban landscape: a genetic investigation. Thesis, The Ohio State University, Columbus, OH.
- Holzman, S., M. J. Conroy, and W. R. Davidson. 1992. Diseases, parasites and survival of coyotes in south-central Georgia. Journal of Wildlife Diseases 28:572-580.
- Kamler, J. F., and P. S. Gipson. 2000. Space and habitat use by resident and transient coyotes. Canadian Journal of Zoology 78:2106-2111.
- _____. 2002. Sarcoptic mange on coyotes in northeastern Kansas. Prairie Naturalist 34:143-144.
- Kilgo, J. C., H. S. Ray, C. Ruth, and K. V. Miller. 2010. Can coyotes affect deer populations in southeastern North America? Journal of Wildlife Management 74:929-933.

- Lindstrom, E., and T. Morner. 1985. The spreading of sarcoptic mange among Swedish red foxes *Vulpes-vulpes* in relation to fox population dynamics. Revue d'Ecologie la Terre et la Vie 40:211-216.
- McCarthy, J. S., D. J. Kemp, S. F. Walton, and B. J. Currie. 2004. Scabies: more than just an irritation. Postgraduate Medical Journal 80:382-387.
- McClennen, N., R. R. Wigglesworth, S. H. Anderson, and D. G. Wachob. 2001. The effect of suburban and agricultural development on the activity patterns of coyotes (*Canis latrans*). American Midland Naturalist 146:27-36.
- Messier, F., and C. Barrette. 1982. The social system of the coyote (*Canis latrans*) in a forested habitat. Canadian Journal of Zoology 60:1743-1753.
- Miller, D. L., J. Schrecengost, A. Merrill, J. C. Kilgo, H. S. Ray, K. V. Miller, and C. A. Baldwin. 2009. Hematology, parasitology, and serology of free-ranging coyotes (*Canis latrans*) from South Carolina. Journal of Wildlife Diseases 45:863-869.
- Morey, P. S., E. M. Gese, and S. Gehrt. 2007. Spatial and temporal variation in the diet of coyotes in the Chicago Metropolitan Area. American Midland Naturalist 158:147-161.
- Morner, T. 1992. Sarcoptic mange in Swedish wildlife. Revue Scientifique et Technique (International Office of Epizootics) 11:1115-1121.
- Nakagawa, T., Y. Takai, M. Kubo, H. Sakai, T. Masegi, and T. Yanai. 2009. A pathological study of sepsis associated with sarcoptic mange in raccoon dogs (*Nyctereutes procyonoides*) in Japan. Journal of Comparative Pathology 141:177-181.
- Nelson, T. A., D. G. Gregory, and Laursen. 2003. Canine heartworms in coyotes in Illinois. Journal of Wildlife Diseases 39:593-599.
- Pence, D. B., and E. Ueckermann. 2002. Sarcoptic mange in wildlife. Revue Scientifique et Technique (International Office of Epizootics) 21:385-398.
- Pence, D. B., and L. A. Windberg. 1994. Impact of a sarcoptic mange epizootic on a coyote population. Journal of Wildlife Management 58:624-633.
- Pence, D. B., L. A. Windberg, B. C. Pence, and R. Sprowls. 1983. The epizootiology and pathology of sarcoptic mange in coyotes, *Canis latrans*, from south Texas. The Journal of Parasitology 69:1100-1115.
- Riley, S. P. D., J. P. Pollinger, R. M. Sauvajot, E. C. York, C. Bromley, T. K. Fuller, and R. K. Wayne. 2006. A southern California freeway is a physical and social barrier to gene flow in carnivores. Molecular Ecology 15:1733-1741.

- Saalfeld, S. T., and S. S. Ditchkoff. 2007. Survival of neonatal white-tailed deer in an exurban population. Journal of Wildlife Management 71:940-944.
- Samuel, W. M. 1981. Attempted experimental transfer of sarcoptic mange (*Sarcoptes scabiei, Acarina: Sarcoptidae*) among red fox, coyote, wolf and dog. Journal of Wildlife Diseases 17:343-347.
- Skerratt, L. F., R. Martin, and K. Handasyde. 1998. Sarcoptic mange in wombats. Australian Veterinary Journal 76:408-410.
- Soulsbury, C. D., G. Iossa, P. J. Baker, N. C. Cole, S. M. Funk, and S. Harris. 2007. The impact of sarcoptic mange *Sarcoptes scabiei* on the British fox *Vulpes vulpes* population. Mammal Review 37:278-296.
- Todd, A. W., J. R. Gunson, and W. M. Samuel. Sarcoptic Mange: An important disease of coyotes and wolves of Alberta, Canada. Pages 706-729 in Worldwide Furbearer Conference Proceedings, 3-11 August, 1980, Frostburg, Maryland, USA.
- 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.
- Walton, S. F., and B. J. Currie. 2007. Problems in diagnosing scabies, a global disease in human and animal populations. Clinical Microbiology Reviews 20:268-279.
- Way, J. G., I. M. Ortega, and E. G. Strauss. 2004. Movement and activity patterns of eastern coyotes in a coastal, suburban environment. Northeastern Naturalist 11:237-254.
- Zahler, M. 1999. Molecular analyses suggest monospecificity of the genus *Sarcoptes* (Acari : Sarcoptidae). International Journal for Parasitology 29:759-766.

Chapter 2

Mortality and Morbidity Due to Sarcoptic Mange in an Urban Coyote Population

INTRODUCTION

Urbanization is increasing globally at an unprecedented rate. By 2050, 67.2% of the world's population will live in urbanized areas, and in the United States urban areas will contain 88.9% of the population (United Nations World Population Prospects 2011). This increase of urbanized areas has important implications for ecosystems that are replaced by the outward expansion of metropolitan areas. Large areas of the landscape are covered with impervious surfaces, which disrupt the natural hydrological cycle (Gehrt 2010). Natural habitats are destroyed and fragmented into smaller separated areas by roadways and urban development (McKinney 2002). Exotic species are introduced to the remaining fragments of the ecosystem, competing with and displacing native species (McKinney 2006). Urbanization alters the biological composition of natural ecosystems leading to decreased biodiversity (McKinney 2002). A negative relationship has been shown in urban areas between human population size and species richness (Olden et al. 2006).

The dynamics of host-parasite relationships can be altered by urbanization as well. Many urban-adapted species occur at higher densities in urbanized environments compared to rural areas (Bradley and Altizer 2007). High population densities can increase contact rates among hosts and increase the transmission of parasites spread through direct contact of individuals (Prange et al. 2003). Supplemental feeding can create areas where large numbers of individuals congregate, also leading to increased contact rates. Alternatively, supplemental feeding can prevent malnourishment, which suppresses an individual's immune system making it more susceptible to parasitism (Ezenwa 2004). Stress from increased inter-specific competition in urban areas (Ruiz et al. 2002) and increased heavy metal and pesticide pollution found in urban areas (Bernier et al. 1995, Krzystyniak et al. 1995) can be detrimental to immune system response and lead to increases in infection and mortality (Bernier et al. 1995, Krzystyniak et al. 1995, Padgett and Glaser 2003). Since a variety of factors can lead to altered host-parasite dynamics in urbanized systems, management decisions in urban areas should be based on information gained through studies in urban areas as opposed to relying on studies that take place in rural areas.

Coyotes have recently become established in many North American cities, including Chicago, Illinois. The emergence of coyotes in metropolitan areas has direct effects on a variety of other species, including Canada goose (*Branta canadensis*) (Brown 2007), red fox (*Vulpes vulpes*) (Gosselink et al. 2003), gray fox (*Urocyon cinereoargenteus*) (Willingham 2008) and white-tailed deer (*Odocoileus virginianus*) (Gehrt and Riley 2010) that reside inside the urban matrix. As the top predator in this

system, coyotes are free from predation or competition from larger predators such as gray wolves (*Canis lupus*) or mountain lions (*Puma concolor*). While harvest and hunting pressure is a major factor affecting survival and population densities in rural areas, this pressure is largely absent inside urban areas (Gehrt and Riley 2010).

As some of the factors that affect coyote populations in rural areas are absent from urban areas, other factors must limit coyote populations in urban environments. Coyote population densities are typically higher in urban areas than rural. When coyote densities were calculated using genotyping of scat and compared between several sites of varying urbanization in southern California, it was determined that coyote densities are highest in those areas closest to urban areas (Fedriani et al. 2001). Adult and juvenile survival rates are higher in urban areas compared to rural coyotes (Gehrt and Riley 2010).

Parasites can have important impacts on host population dynamics. Parasitic infections in wildlife populations occur in two forms: epizootic infections, characterized by rapid spread and increases in prevalence and virulence, and enzootic infections, characterized by a lack of wide fluctuations through time in a defined location (Tompkins et al. 2002). Theoretical approaches to disease dynamics have established that parasites can regulate populations of a host in a density dependent manner (Anderson and May 1978, May and Anderson 1978). Regulation occurs when a population decreases in size when it is above a threshold level, but increases in size when it is below that level (Begon et al. 1996). Therefore, any process that impacts birth and death rates or immigration and emigration rates in a density dependent manner has the potential to regulate a population (Tompkins et al. 2002). However, regulation of a wildlife population by a parasite can

only be definitively shown by the perturbing of a system, and subsequent examination of changes in the abundance of hosts and parasites (Scott and Dobson 1989). Even if parasitism is shown to diminish survival or fecundity in a population, this is not sufficient evidence that regulation is occurring, as reductions may be occurring in a compensatory manner (Tompkins and Begon 1999). There is a need for further research into the relationships between host and parasite population dynamics, particularly long-term studies (Tompkins and Begon 1999, Tompkins et al. 2011).

Sarcoptic mange is a well-documented parasite of wild canids and is known to affect population dynamics of host species (Lindstrom and Morner 1985, Gortazar et al. 1998). Documented cases among canids in North America have been reported in red fox (Trainer and Hale 1969, Gosselink et al. 2007), gray wolves (*Canis lupus*) (Shelley and Gehring 2002, Wydeven et al. 2003, Jimenez et al. 2010) and coyotes (Todd et al. 1980, Pence et al. 1983, Pence and Windberg 1994, Kamler and Gipson 2002, Chronert et al. 2007). Sarcoptic mange is a disease caused by infection with the parasitic mite *Sarcoptes scabiei* (Acariformes:Sarcoptidae) (Fain 1978). Sarcoptic mange can occur as epizootics resulting in high mortality (Pence et al. 1983, Pence and Windberg 1994, Chronert et al. 2007), but also can be enzootic in some wildlife populations (Todd et al. 1980, Gortazar et al. 1998).

Sarcoptic mange can be transmitted either directly through physical contact or indirectly through the use of shared dens (Bornstein et al. 2001). The mite burrows into the epidermis of an infected individual, causing a hypersensitive reaction and intense pruritis (Bornstein et al. 2001). Common signs of sarcoptic mange include alopecia,

hyperkeratosis of the epidermis and crusted lesions (Pence and Ueckermann 2002). Canids severely infected with sarcoptic mange typically have reduced body mass and fat reserves (Pence et al. 1983, Gortazar et al. 1998). Sarcoptic mange is frequently fatal for the infected individual, but mortality rates vary with host species and geographic location if they are known at all (Pence and Ueckermann 2002).

The effects of sarcoptic mange on host population dynamics can vary greatly by species, geographic location and the nature of the infection in the population (i.e. enzootic or epizootic). Epizootics can have short-term effects on red fox populations, but minimal long-term consequences. An epizootic of sarcoptic mange in red fox in Sweden in the early 1980's was responsible for the death of ~50% of the fox population, however the population was recovering by the late 1980's (Lindstrom 1994). Local extinction of red fox on the island of Bornholm in Denmark was caused by the same mange epizootic that impacted Swedish fox populations (Bornstein et al. 2001). In a study of an enzootic infection in the Ebro Valley in Spain, overall prevalence in the study area was low (~3%), but in some local areas prevalence reached as high as 23% (Gortazar et al. 1998). These areas of high prevalence experienced local declines in fox abundance in subsequent years, with an average decline of 38% (Gortazar et al. 1998).

There is differing evidence as to short-term effects of sarcoptic mange on coyote populations, and the long-term effects are poorly documented. Pence et al. (1983) reported no additive mortality due to epizootic sarcoptic mange in a coyote population in south Texas, even though prevalence reached 69% at the epizootic's peak mange. In contrast, Chronert et al. (2007) observed a decrease in coyote abundance during an

epizootic in Wind Cave National Park, South Dakota. While both the southern Texas and Wind Cave studies observed epizootics of sarcoptic mange, Todd et al (1980) reported an enzootic infection of sarcoptic mange in both wolves and coyotes in Alberta. Mange prevalence during winter in Alberta was inversely related to the proportion of pups in the population, suggesting an impact on recruitment (Todd et al. 1980). All of these previous studies of sarcoptic mange in coyotes took place in rural environments.

While no studies have to date examined the relationship between coyotes and *Sarcoptes scabiei* in an urban setting, a study of an epizootic in Bristol, UK red fox population does provide a basis for comparison of the dynamics of sarcoptic mange in an urban setting. Sarcoptic mange first appeared in the Bristol fox population in 1993, reached epizootic levels between 1994 and 1996 and then continued at enzootic levels between 1996 and 2004 (Soulsbury et al. 2007). By the end of 1996, fox populations in Bristol had declined to less than 10% of pre-epizootic numbers (Newman et al. 2002). During the epizootic phase, mortality from sarcoptic mange occurred equally in both juvenile and adult age classes, though there were higher levels of deaths due to sarcoptic mange in the juvenile age class during the enzootic phase (Soulsbury et al. 2007).

Knowledge about a species' population dynamics is essential for informed management decisions. In particular, changes in incidence or mortality over time, whether mortality and morbidity rates vary with demographics within the population, whether survival rates are affected by parasitism and how mortalities caused by parasitism interact with other causes of mortality within the population are important factors that contribute to a species population dynamics. My overall goal was to examine

how sarcoptic mange affects coyote population dynamics in an urban system. Under this overarching goal, my objectives were to: 1) describe the mortality and morbidity rates of sarcoptic mange in the Chicago coyote population, 2) determine if coyote survival is affected by the incidence of sarcoptic mange in the population, and 3) determine if there is evidence that sarcoptic mange is compensatory with other forms of mortality found in this population.

STUDY SITE

Field work was conducted in the Chicago Metropolitan Area, which contains the third most populated city in the United States and comprises Cook, DuPage, Kane, Lake and McHenry counties of Illinois. Over five million residents resided in Cook County alone in 2010 (2010 U.S. census data). Land cover in the Chicago area was estimated to be 30% urban, 16% natural areas, 33% agricultural and 21 % unassociated vegetation in 1997 (Wang and Moskovits 2001). One of the notable features of this metropolitan area is the system of county forest preserves which are protected from urban development. These forest preserves provide large areas of unfragmented habitats, where a wide variety of species use as refugia within the urban matrix. While these preserves are inhabited by wildlife, they are also heavily used by human residents, and are a location where humans and wildlife are frequently in close proximity. Most of the field work took place in the northwestern part of the metropolitan area (Figure 2.1). Radio-collared individuals frequently dispersed beyond county boundaries and I continued to track their movements even though they had left the main study area. Therefore, instead of establishing strict

boundaries for the study area I allowed for the movements of individual coyotes to determine the boundaries of the study area.

METHODS

Field Methods

Capture.-Adult coyotes were captured opportunistically during March 2000 – April 2011 using a combination of cable restraint devices and padded MB-650 foothold traps (Minnnesota Trapline Products, Pennock, MN). Captured individuals were immobilized using an injection of Telazol (Fort Dodge Animal Health, Fort Dodge, IA). Sedated individuals were weighed, marked with individually numbered ear tags (NASCO Farm & Ranch, Fort Atkinson, WI), morphometric measurements were taken, and each individual was fitted with a very-high-frequency radio collar (Advanced Telemetry Systems, Isanti, MN). Individuals were classified into age classes based on tooth wear, physical condition and breeding status. Individuals under 1 year were classified as juveniles, and all individuals older than 1 year were classified as adults, following a precedent set in previous literature of coyote survival and cause-specific mortality (Chamberlain and Leopold 2001, Heisey and Patterson 2006).

I examined each coyote for physical signs of sarcoptic mange, specifically crusted lesions and hairless patches on the coyote's epidermis. If these physical signs were present, a skin scraping was taken by scraping a scalpel blade along the edge of the lesion. The resulting sample was examined under a microscope for evidence of sarcoptic mites. Unfortunately, this particular test tends to be inconclusive because of the lack of sarcoptic mites on the skin during later stages of disease (Bornstein et al. 2001, Pence and

Ueckermann 2002). As such, my diagnosis of infection by sarcoptic mange was based primarily on the physical signs. These diagnoses are supported by the fact that during twenty years of disease surveillance by the Cook County Forest Preserve District and the pathology lab at Brookfield Zoo, skin biopsies routinely confirmed the presence of *Sarcoptes scabiei* mites in the skin of individuals diagnosed with sarcoptic mange based on physical signs (C. Anchor, Cook County Forest Preserve District, unpublished data).

I recorded the amount of hair loss and percentage of affected body area in order to classify the level of infection based on criteria of Pence et al. (1983). Class I individuals had an initial infection involving fore and hind limbs, ischium and ears; class II individuals had a more advanced case with up to 50% of the body's surface area affected; class III individuals had infections affecting > 50% of the body's surface area, and class IV individuals exhibited signs of mange recovery indicated by the re-growth of hair around the edges of lesions.

In addition to capturing adults, during the pup-rearing season I found and examined the dens of known breeding females. I recorded the number of pups in each den site, the numbers of males and females, weighed individuals, and checked for signs of disease. Since mange is spread through bodily contact, it is likely that if one individual in a litter becomes infected the rest of the litter will become infected. Previous literature has hypothesized that if a breeding female is infected with sarcoptic mange, the litter will subsequently become infected and all of the pups will die. Hence, I present the number of litters infected in addition to the number of individuals.

Captured animals were released at the capture site upon recovery from the sedative. Trapping and handling protocols were approved by the Ohio State University's Institutional Animal Care and Use Committee (IACUC 2003R0061) and conformed to protocols set forth by the American Society of Mammalogists (Sikes et al. 2011).

Radio telemetry.-Locations were obtained for radio-collared coyotes using a truck-mounted yagi system. Locations were obtained via triangulation, and calculated with Locate III (Truro Computing, Nova Scotia, Canada) allowing for a maximum error ellipse of 50,000 m². Mean error (± standard deviation) for triangulations was calculated using test transmitters as 108±87 m (Morey 2004). Diurnal locations were obtained a minimum of once per week, and nocturnal locations were obtained sequentially with a minimum of one hour between each subsequent location. There was a minimum goal of ten night locations per animal per month.

Visual observations during radio telemetry were used to assess the status of sarcoptic mange in an individual post-capture. Upon sighting an individual, myself or a technician would attempt to determine if mange was present based upon any signs of hair loss on the individual. If hair loss was noted then the observer estimated the percentage of hair loss on the individual in order to classify the status of the infection according to Pence et al. (1983).

Mortalities.- Radio-collared individuals who died during the study were recovered as quickly as possible following mortality. Carcasses were necropsied at the Brookfield Zoo's pathology laboratory to determine cause of death and to record any underlying conditions (i.e. parasite burden, previous injuries) in the animal. Causes of

mortality were designated as either vehicle trauma, hunting/trapping, mange-related, unknown or other. Vehicle trauma consisted primarily of individuals struck by cars, although it also included incidences where coyotes were struck by trains or in one instance, a plane. Gunshot was defined as mortality caused by individuals shot by landowners or hunters and mortalities due to individuals trapped by fur or nuisance trappers. Mange-related mortality was defined as mortality judged to be directly due to an individual being infected with sarcoptic mange, specific causes included exposure, starvation, secondary infection or euthanization. Animals whose cause of death was recorded as unknown were censored from subsequent analysis. Unknown deaths were generally a result of an individual being recovered in an advanced state of decomposition, and necropsy was unable to definitively determine cause of death. Other was defined as any cause of death identified through necropsy that did not specifically fall into one of the previous categories.

In animals with suspected cases of sarcoptic mange, skin biopsies were examined under a microscope by a trained pathologist. In some cases, *Sarcoptes scabiei* mites were present in the skin sample (Figure 2.2). These mites were identified as *Sarcoptes scabiei* based on the placement of the channels in the epidermis and the location of the mite within the stratus corneum (C. Anchor, CCFPD, pers. communication). *Analysis*

Changes in sarcoptic mange during the study period. I chose to characterize temporal changes in sarcoptic mange by calculating the annual incidence rate for sarcoptic mange in the sample radio-collared population during that year. To more

accurately reflect the population fluctuations that accompany breeding seasons I defined a year as extending from 1 May through 30 April of the subsequent year. Only animals captured as adults were used for this portion of the analysis. Pups were hand caught during pup-rearing season and if they were >3.0 kg marked with a foam-padded radio collar. To avoid biased results based on age, individuals collared as pups were not included in the tallies for the year of their capture, but they were included if they were still radio-collared and actively tracked in subsequent years.

The incidence rates of sarcoptic mange are calculated as the number of new cases of a disease during a specified time period, in this case a year, divided by the number of susceptible individuals in the sample population (Oleckno 2002). Additionally, I calculated annual prevalence of sarcoptic mange in captured individuals. Prevalence was calculated as the number of coyotes captured in each year diagnosed with sarcoptic mange divided by the total number captured (Oleckno 2002).

I also calculated the percentage of animals that died from sarcoptic mange. I looked at this in two different ways: between year differences and within year differences. For within year differences I again used a year starting on 1 May as opposed to a calendar year. I only included individuals in our sample who were radio-collared at the time of death, as including individuals found opportunistically may have biased our sample since most of these individuals were found as the result of vehicle collisions. I determined the percentage of animals that died of mange by dividing the number of animals who died of mange-related causes by the total number of individuals who died

and whose carcasses were recovered. Animals whose cause of death was unknown were included in this sample as well.

To calculate within year changes in mange-related mortality I used the same sample of animals as for the between year analysis. The percentage of individuals who died from sarcoptic mange related causes in each calendar month was calculated by dividing the number of individuals who died of mange-related causes by the number of total mortalities that occurred during that month.

Recovery and time to mortality.- To calculate the percentage of individuals who recovered from sarcoptic mange I classified all individuals throughout the study who were infected with sarcoptic mange into three groups: 1) died of mange, 2) lost radio contact, or 3) the individual recovered from sarcoptic mange. Recovery was determined when an individual was visually observed showing no signs of hair loss, or when the individual was recovered dead it showed no signs of hair loss. I censored all individuals who lost radio-contact since there was no way of determining whether they recovered or died from the disease. Recovery rate was determined by dividing the number of animals who recovered from sarcoptic mange by the number of individuals who were infected with sarcoptic mange with known fate.

In order to determine an estimate for the time it takes an individual to succumb to sarcoptic mange it was important to determine date of infection. I estimated date of infection based on the records for visual observations, taking the halfway point between the last time the animal was seen healthy and the first time it was observed showing signs of sarcoptic mange. Since individuals who were diagnosed with sarcoptic mange at

capture had no date for the last time it was healthy, those individuals were censored from this analysis. Similarly, some individuals who died of sarcoptic mange had dispersed from the study area, and were recovered based on a public report. With these individuals it would have been imprecise to base an estimated date of infection on the last time they were seen. Since these animals were not being regularly tracked, there was no opportunity for visuals and no opportunity to diagnose the infection. For that reason, I censored all individuals who did not have six months of continuous tracking prior to the date of their death. I calculated the number of days between estimated date of infection and the date the individual recovered for individuals who met this qualification,.

Predictors of sarcoptic mange.- I also wanted to determine if there were covariates that increased the probability of infection for sarcoptic mange. Based on previous literature and knowledge of the system, I chose an a priori set of covariates that I hypothesized may play a role in sarcoptic mange infection. The covariates chosen were sex, residency status, and location of capture. All covariates were analyzed using simple logistic regression.

Sex has been proposed as a potential factor that may contribute to sarcoptic mange infection, as it has been proposed that young males make more extra territorial movements and therefore have a higher likelihood of contacting another infected individual (Todd et al. 1980, Pence et al. 1983). Since sex is a covariate that does not change throughout the study period, I was able to evaluate whether males have a higher probability of contracting mange at any point in their lifetime. All individuals older than

five months when captured or radio-tracked were included in this analysis. Individuals were identified as male or female at the time of capture.

It has been hypothesized that transient animals in a population may facilitate the spread of disease due to the fact they traverse a much larger area, and can come in contact with more individuals. Since individuals frequently switch from transient to resident and vice-versa, I only looked at an individual's residency status and disease status at the time of capture. Residency status was determined based on a home range estimated by constructing a 95% minimum convex polygon (MCP) using the locations from the period two months post-capture. Resident coyotes have smaller home ranges, and do not overlap territories of other resident coyote groups. Transient coyotes have larger home ranges than residents and often overlap the territories of several different groups.

Individuals who disappeared within the two-month period were assumed to be transients. I assumed that movements of the individual in the two-month period post-capture are similar to the individuals movements prior to capture. Individuals who did not survive through the two-month period were censored from the analysis, since there was not enough information to make an accurate determination.

In order to determine if there was a spatial component to where sarcoptic mange occurred, it was important to determine whether capture location affected the probability of that individual being infected with sarcoptic mange. Disease status was based on the individual's status at the time of capture. While trapping occurred opportunistically, there were several areas trapped consistently from year-to-year. The capture location categories were MMWF, Poplar Creek, Crabtree, Highland Woods, Busse, Schaumburg

and other. The other category included any site where <5 individuals were captured.

Busse was coded as the dummy variable. All individuals captured as adults were used for this analysis.

Survival.- I used the known-fate models in Program MARK to estimate the effects of sarcoptic mange on coyote survival (White and Burnham 1999). Known-fate models rely on a binomial model to estimate the maximum likelihood estimates for survival for competing models (Murray 2006). Competing models can then be compared by using Akaike's Information Criterion (AIC) to determine the most appropriate model.

Encounter histories for 306 radio-collared individuals were constructed using a period of one month as a sampling period using a staggered entry design (Pollock et al. 1989). The first sampling period occurred in March 2000 and continued until the final sampling period of September 2011, resulting in a total of 139 sampling periods.

Sampling periods were standardized to a 30-day month inside MARK by adjusting the time intervals. The period from 10 March to 31 April of 2000 was censored due to the difference in length between this period and all other years.

Radio-collared pups were tracked similarly to adults, but it is unlikely that survival of individuals of that age group was similar to that of adults. Most studies of coyote survival have shown differences in survival between adults and juveniles (Chamberlain and Leopold 2001, Nelson and Lloyd 2005). We therefore censored these radio-collared pups until September of their birth year, since that is the earliest possible time when they could have been captured via our usual methods.

I analyzed survival data in two stages. First, I established the temporal scale for survival by constructing models in the design matrix of MARK for several different time scales. The temporal models included in the temporal analysis were (MONTH), (SEASON), (YEAR), and (MONTH+YEAR). All models contained an intercept term in addition to the appropriate time scale terms. The (MONTH) model contained variation among months but no variation among study years. The (SEASON) model contained variation based on four-month seasons based on biological events: breeding (1 January – 30 April), pup-rearing (1 May – 31 August) and dispersal (1 September – 31 December) (Gehrt et al. 2009). The (YEAR) model contained variation between study years but no variation among months. The (MONTH+YEAR) model variation between each month of each year of the study. I used an information theoretic approach to compare support among the temporal models, based on an AIC value adjusted for small sample size (AIC_c). I considered models with a Δ AIC_c \leq 2.0 to have equivalent support (Burnham and Anderson 1998). I calculated annual survival rates based on the top temporal model.

Next, since the primary interest was to determine how sarcoptic mange affects survival, I took the top temporal model and added a covariate measuring the monthly prevalence of sarcoptic mange in the population. Prevalence was calculated by dividing the number of individuals that were either trapped or located during each month by the number of individuals known to be infected with sarcoptic mange during that month. Significance of the covariate was determined by examining the β value and the corresponding confidence interval.

Cause-specific mortality.- To determine whether there was evidence that mange-related mortalities were additive or compensatory with other forms of mortality I calculated a cumulative incidence function (CIF) (Heisey and Patterson 2006) to estimate the cause-specific mortality for each of the major causes of mortality in our study: vehicle trauma, hunting/trapping, mange-related mortality, other, and unknown. Encounter histories were constructed for all radio-collared individuals based on the design proposed by Heisey and Patterson (2006), for calculation of CIF's, using a staggered design (Pollock et al. 1989). A year was based on a calendar running from 1 May through 30 April of the subsequent calendar year.

A CIF is used to estimate the probability that an individual will die from a specific cause during a specified time period (Heisey and Patterson 2006). CIF's were calculated using the program csm in the R package wild1 (Sargeant 2011). I calculated separate CIF's for each year (1 May to 30 April) in order to estimate cause-specific mortality rates for each cause of death in each year of the study. This way we have cause specific mortality rates for vehicular trauma, being shot, mange-related causes, other and a fifth category "not mange" which included all other known causes of mortality besides mange-related.

I performed a linear regression of mange-related mortality rates against the annual survival rates for all causes of death generated when the CIF was calculated. A negative relationship between annual survival rates and mange-related mortality rates would provide evidence that mange-related mortality is additive in this population (Sandercock et al. 2011). Additionally, annual cause specific mortality rates for each cause of

mortality were used to compare mange-related annual mortality rates to the annual mortality rates of the other categories of mortality using linear regression. The mortality types other or unknown were not included in the comparison analyses, since other contained only a small number of individuals and the unknown category potentially contained some individuals who died of sarcoptic mange but were not identified as such due to decomposition. A negative linear relationship between annual mange-related mortality rates and the annual mortality rates of another cause would provide strong evidence that mange-related mortality is compensatory in the population.

RESULTS

During March 2000 – May 2011, 292 coyotes (141 F, 151 M, 215 adults, 73 juveniles and 4 of unknown age class) were captured and radio-collared as adults. Of 292 coyotes, 22 (7%) of them were diagnosed with sarcoptic mange at the time of capture. Another 18 coyotes (8 females, 10 males) were collared as pups, but were later monitored for signs of sarcoptic mange via radio-tracking. Of these 310 individuals, 49 (16%) were diagnosed with sarcoptic mange at some point during the study (Table 2.1). Of the 22 animals diagnosed with sarcoptic mange at the time of capture, 5 individuals (23%) were diagnosed with Class I mange, 13 (59%) with Class II mange, 2 (9%) with Class III mange and 2 individuals whose mange classification could not be determined due to missing data (Table 2.2).

During 2000 - 2011, 57 litters were examined for signs of sarcoptic mange in juveniles. These 57 litters yielded 274 pups. Only 3 litters (5%) showed signs of sarcoptic mange, 7 (3%) pups from these three litters showed signs of sarcoptic mange.

In the litters affected with sarcoptic mange, all littermates were infected with sarcoptic mange without exception.

Temporal Dynamics of Sarcoptic Mange

Radio-marked coyotes were monitored for a total of 573 coyote years. Annual incidence rate of sarcoptic mange in this population varied from 0% in 2000-2001 to 16% in 2010-2011 (Figure 2.3). There was no evidence of a linear trend ($F_{1,9}$ =2.50 P=0.15) across years. Annual prevalence rates from captures varied between 0% in the year spanning 2000 and 2001 to a maximum of 25% in the year spanning 2010 and 2011 (Figure 2.4).

Mortalities

The carcasses of 174 radio-collared coyotes were recovered during March 2000 – May 2011. Cause of mortality was determined for 147 of those individuals. The leading cause of mortality in the study area was from vehicle collisions with 86 of the 147 (59%) mortalities of known cause (Table 2.3). The second leading cause of mortality in the study system was gunshot which accounted for 36 (24%) of the 147 mortalities. Mangerelated mortality comprised 22 (15%) of the known causes of mortality (Table 2.3).

The percentage of mortalities attributable to sarcoptic mange varied among study years. I did not detect mange-related mortality in some years, but the mange-related mortality rate reached a maximum of 23% (n=30) of all known mortalities in 2010-2011 (Figure 2.5). There was variation between months within years in mange-related mortalities. Mange was the most common cause of mortalities during December, with 35% (n=28) and January with 23% (n=26) (Figure 2.6). In addition to having the highest

proportion of total known mortalities, the majority of mange-related deaths occurred during winter months, with the majority of mange related deaths occurring in December (46%) and January (27%) (n=22).

Recovery rate & rate of disease progression

Of 49 individuals diagnosed with sarcoptic mange, nine individuals dispersed from the study area or were lost from radio contact, and were censored from these analyses. Of the 40 remaining animals, 22 individuals (55%) died of sarcoptic mange. Another 13 individuals (33%) died from causes other than sarcoptic mange, but were infected at the time of death. Thus, we could not determine the full progression of the disease for these individuals. Five infected individuals (13%); eventually recovered; these individuals were observed to no longer show physical signs of sarcoptic mange or were free from signs of mange at the time of their death.

The elapsed time for an individual to progress from initial infection until death due to mange was highly variable. Of the 22 individuals that died of mange, 10 individuals were either already infected with sarcoptic mange at the time of initial capture or were not being consistently tracked during the six-month period prior to death and were censored. The mean time between estimated infection date and death for the remaining 12 individuals was 131±41.6 days. The shortest time period from estimated infection to death was 11.5 days and the longest time period was 441 days.

Predictors of sarcoptic mange

For the regression analysis using sex as a covariate all 310 coyotes captured and radio-collared were included in the analysis. There was no evidence that sex had any

bearing on the probability that it would contract sarcoptic mange (χ_1^2 =2.07, P=0.15) (Table 2.4). For the analysis using residency status as a covariate, only the 292 individuals captured as adults were included in analysis. Twenty individuals were censored from the analysis because not enough locations were collected to determine residency status. There was no evidence that being a transient had any effect on an individual's probability of being infected with sarcoptic mange (χ_1^2 =0.15, P=0.70) (Table 2.4). Only the 292 adults captured were included in the regression analysis for capture location. There were no significant differences between any of the study sites used in this analysis (χ_6^2 =8.55, P=0.20) (Table 2.4).

Survival

The top model from the temporal model set was (MONTH) (Table 2.5). None of the other models had a ΔAIC_c value less than 2.0. Annual survival was estimated to be 0.696 (95% CI: 0.652, 0.743). All other temporal models showed a ΔAIC_c value of > 7.0, including the (NULL) model, which was the second highest ranked model. The addition of the mange prevalence covariate had no significant effects on the (MONTH) model, and the confidence interval for $\beta_{PREVALENCE}$ contained zero (β =-4.34, 95% CI: -9.94, 1.26).

Cause-specific Mortality

The annual CIF for mange-related mortality throughout the study was estimated to be 0.048 (95% CI: 0.025, 0.071). There was no relationship between mange-related mortality and all other categories of mortality in the study system pooled together $(F_{1,9}<0.01, P=0.94)$ (Figure 2.7.A). There was no relationship between mange-related

mortality and mortality from vehicle collisions ($F_{1,9}$ =0.06, P=0.81) (Figure 2.7.B). There was no relationship between mange-related mortality and mortality from gunshot ($F_{1,9}$ =1.78, P=0.22) (Figure 2.7.C). There was no relationship between the annual mortality rate for mange-related mortality and annual survival ($F_{1,9}$ =2.12, P=0.18) (Figure 2.7.D).

DISCUSSION

Temporal Dynamics of Sarcoptic Mange

Sarcoptic mange was enzootic in the Chicago metropolitan area during this study as indicated by the relatively low and consistent incidence and prevalence rates of sarcoptic mange compared to known epizootics in coyotes (Pence and Windberg 1994, Chronert et al. 2007) and fox (Morner 1992, Soulsbury et al. 2007). The highest recorded prevalence rate for captured animals in the Chicago area was 25%, compared to the epizootic of sarcoptic mange that occurred in southern Texas from 1979-1987 where prevalence of sarcoptic mange in captured animals peaked at 67% (Pence and Windberg 1994) and the epizootic in Wind Cave National Park in 2003-2004 which peaked at 56% (Chronert et al. 2007) (Table 2.6). During a three year study in Alberta, Canada, where mange has been enzootic since the early 1900's mange prevalence based on hunter/trapper kills remained relatively constant during the years of study and peaked at 24% (Todd et al. 1980). The prevalence and incidence rates found in the Chicago metropolitan area most closely resemble those found in the enzootic infection in Alberta, Canada (Table 2.6). Although sarcoptic mange was enzootic during my study, this does not preclude the possibility of future epizootics in the Chicago region. Pence and

Windberg (1994) presented anecdotal evidence that sarcoptic mange had existed in the south Texas population at low levels for at least a decade prior to the epizootic of the early 1980's, and hypothesized that the epizootic was caused by a new strain of *Sarcoptes* mite (Pence and Windberg 1994). Moreover, Soulsbury (2007) reported that while sarcoptic mange was absent from Bristol, UK prior to the epizootic in red fox there, mange had been enzootic in areas surrounding London since the 1940's. Populations such as the Chicago area, where sarcoptic mange is enzootic, may act as a source of future outbreaks in other areas through dispersal of infected individuals. Dispersing individuals have been previously cited as a potential vector for the spread of sarcoptic mange (Pence and Windberg 1994).

While incidence and prevalence of mange were low throughout the project, it is noteworthy that mange incidence and prevalence were at their highest levels during the final year of the study. Considering that mange was absent the first year of the study, there is a suggestion that prevalence of mange may be increasing in this population, albeit slowly. While the changes in coyote abundance are unknown during this study, it is suspected that coyote densities have continued to increase throughout the study area (S. Gehrt, Ohio State University, pers. comm.) and this may be contributing to increased mange prevalence, although there is no conclusive evidence.

While annual incidence rates of sarcoptic mange remained relatively constant throughout the study, there was a great deal of within-year variation particularly relating to incidences of mange-related mortality. The majority of mange-related mortalities occurred during winter months (Figure 2.5). While mortality in coyotes was higher in

general during these months, mange-related mortality accounts for a disproportionate amount of mortality compared to other months (Figure 2.6). High winter mortality of infected individuals is presumably caused by the physical signs of sarcoptic mange infection. The loss of insulation due to alopecia undoubtedly hastens death for individuals who enter the winter months infected with sarcoptic mange. High mortality of mange-infected coyotes has been observed frequently in coyote and wolf populations in areas that experience severe winters and results in decreased prevalence in spring months (Gier et al. 1978, Todd et al. 1980). Alternatively, in areas such as south Texas where winters are mild, overwinter prevalence of sarcoptic mange increased in some age classes (Pence and Windberg 1994).

Sarcoptic mange appears to have little effect on recruitment in the Chicago metropolitan area. I found few litters in which the pups showed signs of sarcoptic mange. It is possible that signs developed later on, after the period when we were actively checking dens. In addition to infecting and killing pups, sarcoptic mange can also affect the reproductive potential of adults. In severely infected red fox, males fail to undergo spermatogenesis and females do not breed (Soulsbury et al. 2007). Additionally, mange-infected coyote females have a higher probability of reabsorbing their all their fetuses than healthy individuals (Pence and Windberg 1994). Reduction in fecundity would not be apparent just through checking dens, and effects such as decreased litter size or the reabsorption of fetuses in infected individuals are unaccounted for. However, since sarcoptic mange prevalence in the Chicago metropolitan area is relatively low,

particularly during the breeding and pup-rearing seasons, it is not likely that sarcoptic mange has a major impact on fecundity in the coyote population.

Recovery and Time Elapsed until Death

The percentage of coyotes who recover from sarcoptic mange varies greatly among different studies. Pence et al. (1983) reported that only 3% of captured coyotes in south Texas showed signs of hair re-growth on the edge of lesions indicating recovery. In Wind Cave National Park, 20% of the coyotes infected with sarcoptic mange had recovered by the end of the study (Chronert et al. 2007). I found that 13% of the animals diagnosed with sarcoptic mange recovered in the Chicago metropolitan area. Similar to other studies (Pence et al. 1983), some individuals who seemed to recover from sarcoptic mange based on re-growth of hair relapsed and were observed later with patterns of hairloss indicating sarcoptic mange infection. It is likely that the actual recovery rate from sarcoptic mange is closer to our own estimate and the Wind Cave study (Chronert et al. 2007) opposed to the south Texas study. This is due mainly to the fact that the former two studies used radio-collars and tracking individuals post-capture to monitor infection and the south Texas study relied primarily on capture data (Pence and Windberg 1994).

My estimate of 13% recovery is probably conservative. While I was able to accurately assess mange infection in all captured animals, it was more difficult to assess mange condition visually. Observations usually only lasted a few seconds and occurred at night. While we were able to identify many cases via visual observations based on patterns of hair loss, the ability to identify light infections of mange of the Class I type is

questionable. It is probable that additional animals had light Class I infections that went undetected and then recovered.

The time elapsed between initial infection and death varied greatly between coyotes, particularly in comparison to experimental studies involving the inoculation of coyotes with *Sarcoptes scabiei* mites (Samuel 1981). During experimental studies, three treatment coyotes died 75, 73 and 52 days post-infection (Samuel 1981). Some of the variation is probably attributable to inaccuracy in estimating infection date. While every effort was made to observe individuals frequently to assess condition, occasionally several months would pass between visual observations of an individual. Furthermore, often these observations only lasted a few seconds, and particularly in poorly lit areas, it was sometimes difficult to accurately assess an individual's condition. Hence, the estimates for initial date of infection may not always be accurate. Variation in time between infection and death, probably depends on a variety of factors, most likely the status of the host's immune system at time of infection and whether the host has any natural immunity to infection (Samuel 1981).

None of the covariates tested proved to be a significant factor associated with contracting sarcoptic mange. While being a transient individual does not necessarily predispose an individual to contracting sarcoptic mange, Lindstrom and Morner (1985) and Pence and Windberg (1994) suggested that transient individuals may facilitate the spread of the disease via long range movements. Similarly, while capture location was not a significant predictor of an individual being infected with sarcoptic mange, this does not preclude a spatial component to sarcoptic mange infection. Capture location

documented only where the animal was caught, and did not take into account other life history factors such as whether an animal was a resident or a transient, or whether it lived in a forest preserve or urban area. Small sample sizes precluded me from comparing all of these life-history traits in the same model. Furthermore, during the study many animals dispersed from the areas in which they were initially captured and established their own territories elsewhere or were killed as transients. Transient animals may explain why there was little spatial variation in this system. Transient coyotes have home ranges much larger than resident coyotes (Gehrt et al. 2009), and the movements of transient animals across the landscape can easily spread disease across the study area. Further investigation as to factors that influence an individual's probability of contracting sarcoptic mange is required.

Survival

I found no evidence that annual survival rates in the Chicago coyote population are affected by annual changes in prevalence of sarcoptic mange. The annual survival estimate (0.696) for coyotes in the study area was similar to values estimated for other lightly exploited populations (Windberg et al. 1985, Gese et al. 1989, Grinder and Krausman 2001), and was higher than values estimated for rural coyote populations in Illinois (0.53) (Van Deelen and Gosselink 2006). Even in epizootic infections it is unclear how much of an effect sarcoptic mange prevalence has on survival. Though prevalence rates in a coyote population reached 69% in southern Texas during an epizootic, annual survival remained constant throughout the epizootic (Pence and Windberg 1994). This study may be an aberration, as numerous studies of epizootic and

enzootic infections in red fox have shown decreases in abundance, in some cases as high as 90% decreases associated with sarcoptic mange infection (Lindstrom and Morner 1985, Gortazar et al. 1998, Soulsbury et al. 2007). Epizootics of coyotes have also shown decreases in coyote abundance associated with sarcoptic mange infection (Chronert et al. 2007). Changes in the prevalence rates of sarcoptic mange in the Chicago area during the study period were not reflected by changes in survival. Based on these comparisons of annual survival of coyotes, it is likely that coyote survival rates are more likely to be affected by factors other than the prevalence of sarcoptic mange.

Cause-specific mortality

Based on the lack of a relationship between annual survival and rates of mangerelated mortality there is no evidence that mange-related mortality is additive in the
population. However, since there was also no relationship between rates of mangerelated mortality and rates of other forms of mortality there is no evidence that mangerelated mortality is compensatory. The existence of compensatory or additive forms of
mortality is difficult to provide evidence for outside of an experimental design where
observers can control mortality rates for the cause of mortality in question (Sandercock et
al. 2011). Both annual survival rates and rates of sarcoptic mange-related mortality were
relatively constant throughout the study. Hence, without a perturbation of the system it
would be difficult to establish either compensatory or additive mortality.

Differences in causes of mortality for coyotes are an important distinction between rural and urban systems. While hunting and trapping mortality is a major cause of mortality in many rural systems, hunting and trapping are largely absent from urban ecosystems (Gehrt and Riley 2010) and this can potentially have effects on sarcoptic mange prevalence. In some rural locations hunting and trapping can account for > 50% of all mortalities (Chamberlain and Leopold 2001, Van Deelen and Gosselink 2006). The absence of hunting and trapping means that urban areas typically have higher survival rates than rural areas (Windberg et al. 1985). Increased survival means coyotes live longer and the population density increases. Increased densities and increased contact rates likely contribute to the presence of sarcoptic mange in this region. Hence, sarcoptic mange may be a disease that primarily affects urban coyotes in this region, and likely occurs at higher prevalence in areas of light to no human exploitation. A similar pattern was seen in red foxes in Illinois (Gosselink et al. 2007). Among foxes in urban areas mange was the highest cause of mortality, while in rural systems coyotes and vehicles accounted for the largest number of mortalities (Gosselink et al. 2007).

Previous studies have reported epizootics of sarcoptic mange that occurred concurrently with outbreaks of other disease. An epizootic of canine parvovirus (CPV) occurred around the peak of the mange epizootic of in southern Texas (Pence and Windberg 1994). Outbreaks of sarcoptic mange in wolf populations in the greater Yellowstone area have occurred simultaneously with outbreaks of canine distemper virus (CDV) and CPV (Almberg et al. 2009, Jimenez et al. 2010). CPV and CDV are both present in the Chicago coyote population (S. Gehrt, unpublished data). While mortality from these diseases is extremely low, serology has shown that many coyotes were exposed to these viruses (S. Gehrt, unpublished data). Exposure to multiple pathogens increases the impact on a host's fitness (Tompkins et al. 2011), and it is possible the

presence of these pathogens combined with high densities in an urban environment contribute to sarcoptic mange infection in the urban coyote population. Further research into the relationships between sarcoptic mange and other common diseases of coyotes in urban area is required to determine how interactions of these diseases can affect coyote populations in urban areas.

MANAGEMENT IMPLICATIONS

Sarcoptic mange is the leading cause of disease-related mortality for coyotes of the Chicago metropolitan area. While there is little inter-year variation in the incidence of mange, there is a great deal of intra-year variation particularly with respect to mangerelated mortalities. High densities and absence of other mortality factors, specifically hunting and trapping, likely contribute to enzootic sarcoptic mange infection in the Chicago coyote population. Managers in urban areas should be able to recognize the physical signs of sarcoptic mange, and the possibility that mange can be present in urban systems in an enzootic state. The appearance of coyotes suffering from sarcoptic mange is likely to draw attention and concern from observers, potentially leading to an increase in nuisance reports. It is possible for coyotes to transmit mange to domestic dogs (Pence and Ueckermann 2002), and this is a likely concern for dog owners who view mangeinfected coyotes in their neighborhoods. Soulsbury (2007) observed an increase in mange in domestic dog populations in Bristol concurrent with the epizootic in red fox. However, the contact rates between domestic dogs and coyotes in this system are unknown, as is the role that consistent application of acaricides as tick and flea prevention can have on preventing mange. Experimental inoculation with mange mites

from an infected coyote does not always result in disease for domestic dogs (Samuel 1981). While mange appears to be currently enzootic in the Chicago area population of coyotes, this does not preclude future epizootics of sarcoptic mange in this region.

Continued monitoring of sarcoptic mange within this population will further increase our knowledge of the role of sarcoptic mange in coyote population dynamics.

LITERATURE CITED

- Almberg, E. S., L. D. Mech, D. W. Smith, J. W. Sheldon, and R. L. Crabtree. 2009. A serological survey of infectious disease in Yellowstone National Park's canid community. PloS one 4:e7042.
- Anderson, R. M., and R. M. May. 1978. Regulation and stability of host-parasite population interactions: I. Regulatory processes. Journal of Animal Ecology 47:219-247.
- Begon, M., J. L. Harper, and C. R. Townsend. 1996. Ecology:Individuals, Populations and Communities. Blackwell Scientific Publications, Oxford.
- Bernier, J., P. Brousseau, K. Krzystyniak, H. Tryphonas, and M. Fournier. 1995. Immunotoxicity of heavy metals in relation to Great Lakes. Environmental Health Perspectives 103:23-34.
- Bornstein, S., T. Morner, and W. M. Samuel. 2001. *Sarcoptes scabiei* and sarcoptic mange. Pages 107-119 *in* W. M. Samuel, M. J. Pybus, and K. A.A., editors. Parasitic Diseases of Wild Mammals. Iowa State University Press, Ames, IA.
- Bradley, C. A., and S. Altizer. 2007. Urbanization and the ecology of wildlife diseases. Trends in Ecology & Evolution 22:95-102.
- Brown, J. 2007. The influence of coyotes on an urban Canada goose population in the Chicago metropolitan area. Thesis, The Ohio State University, Columbus, OH.
- Burnham, K. P., and D. R. Anderson. 1998. Model Selection and Inference: A Practical Information-Theoretic Approach. Springer-Verlag, New York, NY.

- Chamberlain, M. J., and B. D. Leopold. 2001. Survival and cause-specific mortality of adult coyotes (*Canis latrans*) in central Mississippi. American Midland Naturalist 145:414-418.
- Chronert, J. M., J. A. Jenks, D. E. Roddy, M. A. Wild, and J. G. Powers. 2007. Effects of sarcoptic mange on coyotes at Wind Cave National Park. Journal of Wildlife Management 71:1987-1992.
- Ezenwa, V. O. 2004. Interactions among host diet, nutritional status and gastrointestinal parasite infection in wild bovids. International Journal for Parasitology 34:535-542.
- Fain, A. 1978. Epidemiological problems of scabies. International Journal of Dermatology 17:20-30.
- Fedriani, J. M., T. K. Fuller, and R. M. Sauvajot. 2001. Does availability of anthropogenic food enhance densities of omnivorous mammals? An example with coyotes in southern California. Ecography 24:325-331.
- Gehrt, S. D. 2010. The Urban Ecosystem. *in* S. D. Gehrt, B. L. Cypher, and S. P. D. Riley, editors. Urban Carnivores. The Johns Hopkins University Press, Baltimore, MD.
- Gehrt, S. D., C. Anchor, and L. A. White. 2009. Home range and landscape use of coyotes in a metropolitan landscape: conflict or coexistence? Journal of Mammalogy 90:1045-1057.
- Gehrt, S. D., and S. P. D. Riley. 2010. Coyotes (*Canis latrans*). Pages 79-95 *in* S. D. Gehrt, S. P. D. Riley, and B. L. Cypher, editors. Urban Carnivores. The Johns Hopkins University Press, Baltimore, MD.
- Gese, E. M., O. J. Rongstad, and W. R. Mytton. 1989. Population dynamics of coyotes in southeastern Colorado. Journal of Wildlife Management 53:174-181.
- Gier, H. T., S. M. Kruckenberg, and R. J. Marler. 1978. Parasites and Diseases of Coyotes. Pages 37-71 *in* M. Bekoff, editor. Coyotes: Biology, Behavior and Management. The Blackburn Press, Caldwell, NJ.
- Gortazar, C., R. Villafuerte, J. C. Blanco, and D. Fernandez-De-Luco. 1998. Enzootic sarcoptic mange in red foxes in Spain. Zeitschrift fuer Jagdwissenschaft 44:251-256.
- Gosselink, T. E., T. R. Van Deelen, R. E. Warner, and M. G. Joselyn. 2003. Temporal habitat partitioning and spatial use of coyotes and red foxes in east-central Illinois. Journal of Wildlife Management 67:90-103.

- Gosselink, T. E., T. R. Van Deelen, R. E. Warner, and P. C. Mankin. 2007. Survival and cause-specific mortality of red foxes in agricultural and urban areas of Illinois. Journal of Wildlife Management 71:1862-1873.
- Grinder, M., and P. R. Krausman. 2001. Morbidity-mortality factors and survival of an urban coyote population in Arizona. Journal of Wildlife Diseases 37:312-317.
- Heisey, D. M., and B. R. Patterson. 2006. A review of methods to estimate cause-specific mortality in presence of competing risks. The Journal of Wildlife Management 70:1544-1555.
- Jimenez, M. D., E. E. Bangs, C. Sime, and V. J. Asher. 2010. Sarcoptic mange found in wolves in the rocky mountains in western United States. Journal of Wildlife Diseases 46:1120-1125.
- Kamler, J. F., and P. S. Gipson. 2002. Sarcoptic mange on coyotes in northeastern Kansas. Prairie Naturalist 34:143-144.
- Krzystyniak, K., H. Tryphonas, and M. Fournier. 1995. Approaches to the evaluation of chemical-induced immunotoxicity. Environmental Health Perspectives 103:17-22.
- Lindstrom, E., and T. Morner. 1985. The spreading of sarcoptic mange among Swedish red foxes *Vulpes-vulpes* in relation to fox population dynamics. Revue d'Ecologie la Terre et la Vie 40:211-216.
- Lindstrom, E. R. 1994. Disease reveals the predator- sarcoptic mange, red fox predation, and prey populations. Ecology 75:1042-1049.
- May, R. M., and R. M. Anderson. 1978. Regulation and stability of host-parasite population interactions: II. destabilizing processes. Journal of Animal Ecology 47:249-267.
- McKinney, M. L. 2002. Urbanization, biodiversity, and conservation. BioScience 52:883-890.
- _____. 2006. Urbanization as a major cause of biotic homogenization. Biological Conservation 127:247-260.
- Morey, P. S. 2004. Landscape use and diet of coyotes, *Canis latrans*, in the Chicago metropolitan area. Thesis, Utah State University, Logan, UT, USA.
- Morner, T. 1992. Sarcoptic mange in Swedish wildlife. Revue Scientifique et Technique (International Office of Epizootics) 11:1115-1121.
- Murray, D. L. 2006. On improving telemetry-based survival estimation. The Journal of Wildlife Management 70:1530-1543.

- Nelson, T. A., and D. M. Lloyd. 2005. Demographics and condition of coyotes in Illinois. American Midland Naturalist 153:418-427.
- Newman, T. J., P. J. Baker, and S. Harris. 2002. Nutritional condition and survival of red foxes with sarcoptic mange. Canadian Journal of Zoology 80:154-161.
- Olden, J. D., N. L. Poff, and M. L. McKinney. 2006. Forecasting faunal and floral homogenization associated with human population geography in North America. Biological Conservation 127:261-271.
- Oleckno, W. A. 2002. Essential Epidemiology: Principles and Applications. Waveland Press, Long Grove, IL.
- Padgett, D. A., and R. Glaser. 2003. How stress influences the immune response. Trends in Immunology 24:444-448.
- Pence, D. B., and E. Ueckermann. 2002. Sarcoptic mange in wildlife. Revue Scientifique et Technique (International Office of Epizootics) 21:385-398.
- Pence, D. B., and L. A. Windberg. 1994. Impact of a sarcoptic mange epizootic on a coyote population. Journal of Wildlife Management 58:624-633.
- Pence, D. B., L. A. Windberg, B. C. Pence, and R. Sprowls. 1983. The epizootiology and pathology of sarcoptic mange in coyotes, *Canis latrans*, from south Texas. The Journal of Parasitology 69:1100-1115.
- Pollock, K. H., S. R. Winterstein, C. M. Bunck, and P. D. Curtis. 1989. Survival analysis in telemetry studies: the staggered entry design. The Journal of Wildlife Management 53:7-15.
- Prange, S., S. D. Gehrt, and E. P. Wiggers. 2003. Demographic factors contributing to high raccoon densities in urban landscapes. The Journal of Wildlife Management 67:324-333.
- Ruiz, G., M. Rosenmann, F. F. Novoa, and P. Sabat. 2002. Hematological parameters and stress index in rufous-collared sparrows dwelling in urban environments. The Condor 104:162-166.
- Samuel, W. M. 1981. Attempted experimental transfer of sarcoptic mange (*Sarcoptes scabiei, Acarina: Sarcoptidae*) among red fox, coyote, wolf and dog. Journal of Wildlife Diseases 17:343-347.
- Sandercock, B. K., E. B. Nilsen, H. Broseth, and H. C. Pedersen. 2011. Is hunting mortality additive or compensatory to natural mortality? Effects of experimental harvest on the survival and cause-specific mortality of willow ptarmigan. Journal of Animal Ecology 80:244-258.

- Sargeant, G. B. 2011. Wild1-R Tools for Wildlife Research and Management. http://cran.r-project.org/web/packages/wild1 Accessed 3 November 2011.
- Scott, M. E., and A. Dobson. 1989. The role of parasites in regulating host abundance. Parasitology Today 5:176-183.
- Shelley, D. P., and T. M. Gehring. 2002. Behavioral modification of gray wolves, *Canis lupus*, suffering from sarcoptic mange: Importance of sequential monitoring. Canadian Field-Naturalist 116:648-650.
- Sikes, R. S., W. L. Gannon, D. S. Carroll, B. J. Danielson, J. W. Dragoo, M. R. Gannon, W. L. Gannon, D. W. Hale, C. McCain, D. K. Odell, L. E. Olson, S. Ressing, R. M. Timm, S. A. Trewhitt, and J. E. Whaley. 2011. Guidelines of the American Society of Mammalogists for the use of wild mammals in research. Journal of Mammalogy 92:235-253.
- Soulsbury, C. D., G. Iossa, P. J. Baker, N. C. Cole, S. M. Funk, and S. Harris. 2007. The impact of sarcoptic mange *Sarcoptes scabiei* on the British fox *Vulpes vulpes* population. Mammal Review 37:278-296.
- Todd, A. W., J. R. Gunson, and W. M. Samuel. Sarcoptic Mange: An important disease of coyotes and wolves of Alberta, Canada. Pages 706-729 in First Worldwide Furbearer Conference Proceedings, 3-11 August, 1980, Frostburg, Maryland, USA.
- Tompkins, D. M., and M. Begon. 1999. Parasites can regulate wildlife populations. Parasitology Today 15:311-313.
- Tompkins, D. M., A. P. Dobson, P. Arneberg, M. E. Begon, I. M. Cattadori, J. V. Greenman, J. A. P. Heesterbeek, P. J. Hudson, D. Newborn, A. Pugliese, A. P. Rizzoli, R. Rosa, F. Rosso, and K. Wilson. 2002. Parasites and host population dynamics. Pages 45-62 *in* P. J. Hudson, A. Rizzoli, B. T. Greenfell, H. Heesterbeek, and A. P. Dobson, editors. The Ecology of Wildlife Diseases. Oxford University Press, New York.
- Tompkins, D. M., A. M. Dunn, M. J. Smith, and S. Telfer. 2011. Wildlife diseases: from individuals to ecosystems. Journal of Animal Ecology 80:19-38.
- 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.
- United Nations Population Division. 2012. World Population Prospects: The 2011 Revision, CD-ROM Edition. Department of Economic and Social Affairs. New York, New York, USA.

- United States Census Bureau. 2010. < http://2010.census.gov/2010census/>. Accessed 11 December 2011.
- Van Deelen, T. R., and T. E. Gosselink. 2006. Coyote survival in a row-crop agricultural landscape. Canadian Journal of Zoology 84:1630-1636.
- Wang, Y., and D. K. Moskovits. 2001. Tracking fragmentation of natural communities and changes in land cover: applications of landsat data for conservation in an urban landscape (Chicago Wilderness). Conservation Biology 15:835-843.
- White, G. C., and K. Burnham. 1999. Program MARK: survival estimation from populations of marked animals. Bird Study 46:S120-S139.
- Willingham, A. N. 2008. Emerging factors associated with the decline of a gray fox population and multi-scale land cover associations of mesopredators in the Chicago metropolitan area. Thesis, Ohio State University, Columbus, Ohio.
- Windberg, L. A., H. L. Anderson, and R. M. Engeman. 1985. Survival of coyotes in southern Texas. The Journal of Wildlife Management 49:301-307.
- Wydeven, A. P., S. R. Boles, R. N. Schultz, and C. J. T. Doolittle. 2003. Death of gray wolves, *Canis lupus*, in porcupine, *Erethizon dorsatum*, dens in Wisconsin. Canadian Field-Naturalist 117:469-471.

FIGURES AND TABLES

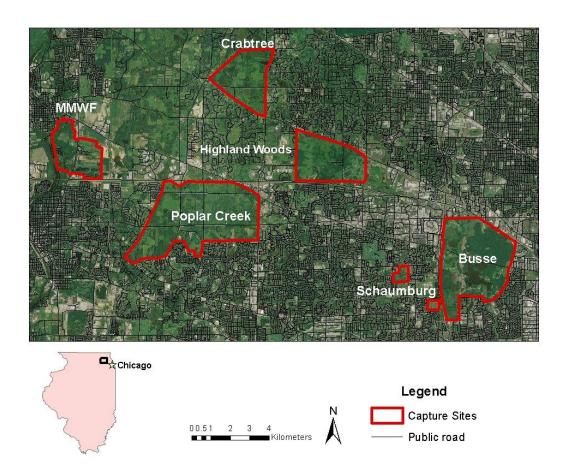


Figure 2.1. Aerial photo of the northwestern suburbs in the Chicago metropolitan area showing road densities and capture locations of coyotes.

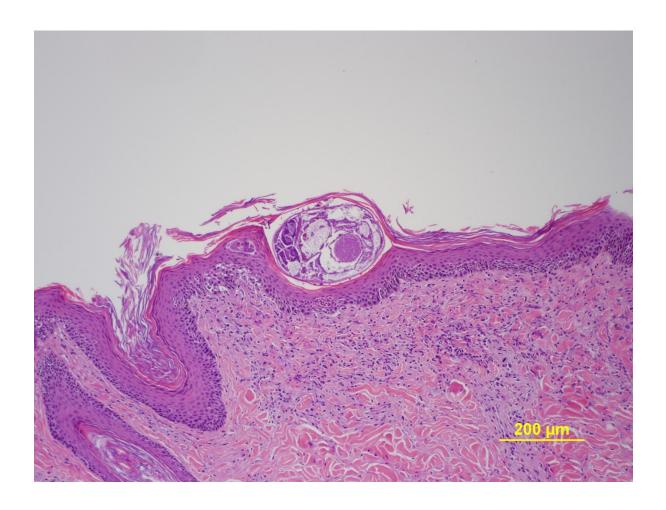


Figure 2.2. Photograph of magnified cross-section of skin from a mange-infected coyote. The round object in the center is the cross-section of a *Sarcoptes scabiei* mite. Courtesy of Cook County Forest Preserve District.

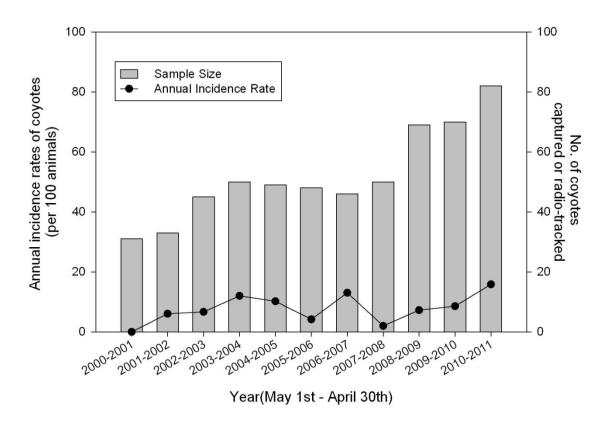


Figure 2.3. Annual variation in the incidence of sarcoptic mange infection among radio collared and captured coyotes in the Chicago metropolitan area between 2000 and 2011.

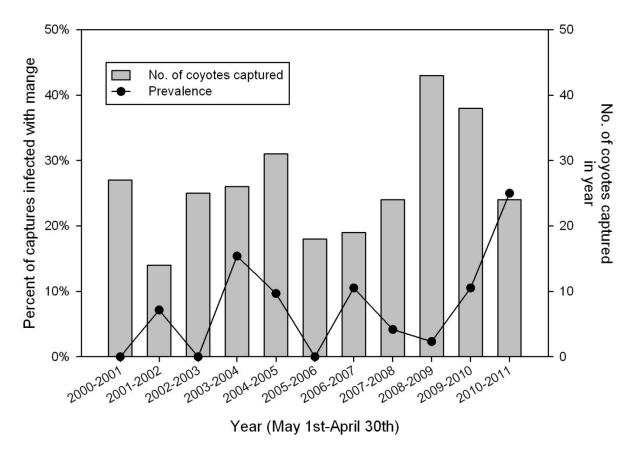


Figure 2.4. Annual variation in the prevalence of sarcoptic mange in adult coyotes captured in the Chicago metropolitan area between 2000 and 2011.

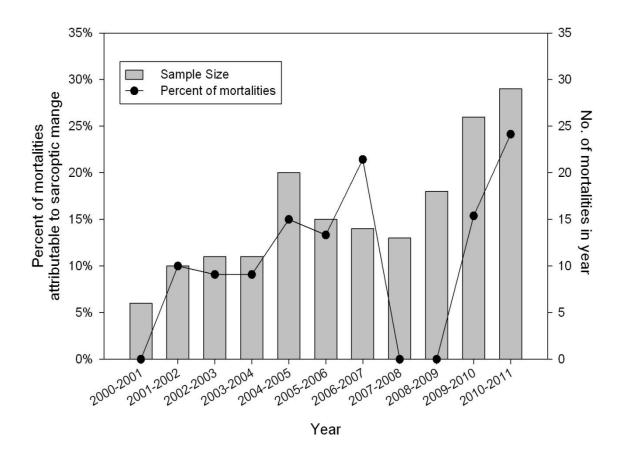


Figure 2.5. Annual variation in the percentage of mortalities attributable to sarcoptic mange for coyotes in the Chicago metropolitan area between 2000 and 2011.

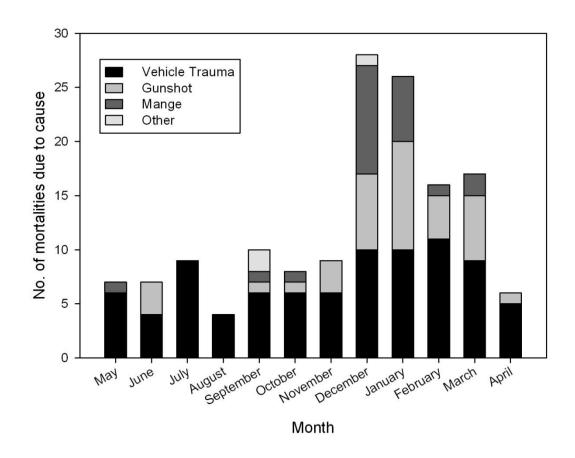


Figure 2.6. Causes of mortality in coyotes in the Chicago metropolitan area between 2000 and 2011.

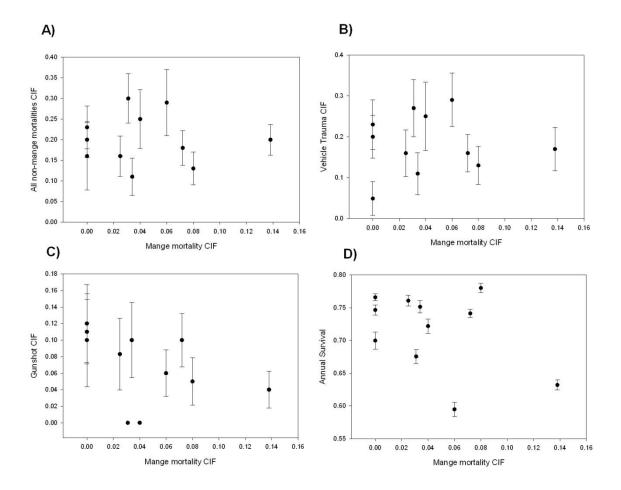


Figure 2.7. Sarcoptic mange-specific mortality rates for coyotes in the Chicago Metropolitan Area compared to A) cause-specific mortality rates for all other forms of mortality B) annual cause-specific mortality rates for vehicle collisions C) annual cause-specific mortality rates for hunting/trapping for coyotes and D) annual survival rates between 2000 and 2011. Error bars indicate standard errors.

Sample Method	No. Infected	Sample Size	Percentage Infected
Capture only	22	292	7%
Mortalities	33	182	18%
All Adults	49	310	16%

Table 2.1. Frequency of sarcoptic mange infection among coyotes in the Chicago metropolitan area between 2000 and 2011 infected with sarcoptic mange at three different opportunities for diagnosis: initial capture, recovery of carcass at death and all adults monitored via capture or radio-tracking. Individuals can be included in more than one sample method.

Sex	Age	Mange Class				
		0	1	Ш	Ш	Unknown
Female	Adult	92	1	4	0	1
	Juvenile	39	0	1	0	1
	Unknown	2	0	0	0	0
	Subtotal	133	1	5	0	2
Male	Adult	105	3	8	1	0
	Juvenile	31	1	0	1	0
	Unknown	1	0	0	0	0
	Subtotal	137	4	8	2	0
	Grand Total	270	5	13	2	2

Table 2.2. Variation in severity of sarcoptic mange between sexes and age classes for coyotes captured in the Chicago metropolitan area between 2000 and 2011. See methods for descriptions of age class and mange infection classes.

Cause of Mortality	N	Percentage
Vehicular Trauma	86	59%
Gunshot	36	24%
Mange-related	22	15%
Other	3	2%
Total	147	100%

Table 2.3. Causes of mortalities of radio-collared coyotes in the Chicago metropolitan area between 2000 and 2011.

Covariate	_	Infected with Sarcoptic Mange				
		Yes	No	Total		
Sex						
	Male	30	131	261		
	Female	19	130	49		
	Total	49	161	310		
Residency Status	5					
	Transient	9	108	117		
	Resident	10	145	155		
	Total	19	253	272		
Capture Location	า					
	Busse	8	46	54		
	Crabtree	1	18	19		
	Highland Woods	1	32	32		
	MMWF	2	51	53		
	Other	0	15	15		
	Poplar Creek	8	81	87		
	Schaumburg	2	27	29		
	Total	22	270	292		

Table 2.4. Frequency of coyotes infected with sarcoptic mange in the Chicago metropolitan area between 2000 and 2011 partitioned by covariates of interest.

Model ^a	k^b	AIC _c ^b	ΔAIC_c^b	$oldsymbol{w_i}^{b}$
Month	12	1076.19	0	0.9698
Null	1	1083.57	7.382	0.0242
Season	3	1086.38	10.19	0.00594
Year	11	1096.41	20.21	0
Month + Year	132	1172.98	96.79	0

^a Month=calendar month; Null=no variation in survival; Season=Breeding (1 Jan. - 30 April), Pup-rearing (1 May - 31 Aug.), Dispersal (1 Sept. – 31 Dec.); Year=1 May – 30 April.

Table 2.5. Known-fate models of survival comparing support for temporal variation in survival using AICc among coyotes in the Chicago metropolitan area between 2000 and 2011.

^b k=no. of parameters in the model; AIC_c=AIC corrected for small sample size; Δ AIC_c= difference between the AICc value of model compared to the AICc value of the top model; w_i = Akaike weight.

Study Location	n	Range of Prevalence	Study Duration	Infection Type	Authors
Alberta, Canada	379	11%-24%	3 yrs.	Enzootic	Todd et al. 1980
Southern Texas, US	1,349	0%-69%	17 yrs.	Epizootic	Pence et al. 1983 Pence and Windberg 1994
South Dakota, US	26	12%-56%	2 yrs.	Epizootic	Chronert et al. 2002
Chicago, IL	310	0%-25%	11 yrs.	Enzootic	Current Study

Table 2.6. Sample size, prevalence ranges, study duration and infection type from previous studies of sarcoptic mange in coyotes in North America.

Chapter 3

Habitat Selection and Use by Coyotes Infected with Sarcoptic Mange in a Suburban Environment

INTRODUCTION

In the past 200 years the geographic range of the coyote (*Canis latrans*) has expanded from its historical range in the southwest and rocky mountain regions of North America, to today, where its geographic range now encompasses nearly the entire continent (Gompper 2002). This range expansion has allowed coyotes to colonize areas previously unavailable to them, and in fact, thrive in these ecosystems. One of the recently colonized ecosystems that has garnered a great deal of attention and interest is the urban ecosystem, particularly throughout the Midwest and eastern states where coyotes are relatively new residents. Established populations of coyotes have been documented in major metropolitan areas such as Los Angeles (Shargo 1988), Tucson (Grinder and Krausman 2001a, Grinder and Krausman 2001b, Grubbs and Krausman 2009), Seattle (Quinn 1995;1997b;a), and Chicago (Gehrt et al. 2009).

With coyotes establishing themselves in major metropolitan areas comes an increasing amount of contact between the coyotes and humans living in these areas.

News articles in the Chicago metropolitan area about human-coyote conflicts have increased dramatically since the mid-1980's (White and Gehrt 2009), and previous reviews have suggested that nationwide, coyote attacks on humans have increased in frequency as well (Baker and Timm 1998, Timm et al. 2004). While most conflict between humans and coyotes does not take the form of attacks on humans, the presence of coyotes in close proximity to human residences can be a danger to small pets (i.e. outdoor cats and small dogs). Furthermore, oftentimes the sight of a coyote in the yard of a homeowner or on a trail can be enough to cause concern for the homeowner, and trigger a nuisance complaint to management officials (Lukasik and Alexander 2011). Many of these potential conflicts are prevented by the behavior of the urban coyote itself.

Coyotes that live in the urban matrix display several behavioral differences when compared with their rural counterparts. While most coyotes in rural and natural areas are most active during crepuscular time periods, coyotes that live in urban ecosystems throughout North America are consistently nocturnal (Grinder and Krausman 2001b, McClennen et al. 2001, Atwood et al. 2004, Grubbs and Krausman 2009). Grubbs and Krausman (2009) hypothesized that this behavioral change allows coyotes to avoid the highest periods of human activity. Furthermore, coyotes living in the urban matrix have repeatedly shown avoidance of human-dominated habitats, instead selecting for natural or undeveloped areas within the landscape (Gibeau 1998, Gehrt et al. 2009, Grubbs and Krausman 2009).

While anthropogenic food sources may lead to increased densities of some species in urban areas, anthropogenic food does not comprise a large part of the urban

coyote's diet. Morey et al. (2007) observed that anthropogenic food was present in <2% of examined scat in the Chicago metropolitan area. There is typically spatial and seasonal variation in the occurrence of anthropogenic food in coyote scat, with the most urbanized areas having the highest occurrence of anthropogenic items (Fedriani et al. 2001, Morey et al. 2007). However, even in the most urbanized areas, occurrence of trash items in scat was < 11% (Fedriani et al. 2001, Morey et al. 2007). While anthropogenic food sources do not compose major portions of the urban coyotes diet, coyote densities are increased when large amounts of anthropogenic food sources are present (Fedriani et al. 2001). Nuisance coyote complaints can frequently be traced back to an individual in the neighborhood who is either actively feeding the animals or putting food outside for cats and other animals and homeowners are urged to avoid this activity (Timm et al. 2004, White and Gehrt 2009).

However, most of the time the causes as to why a coyote becomes a "nuisance" animal are unclear. While some nuisance complaints are the result of an individual coyote becoming accustomed to easy access to anthropogenic food and displaying bold behavior, these circumstances are not true in all cases. A previous analysis of radio-collared coyotes in the Chicago metropolitan region determined that of seven radio-collared individuals who were defined by the investigators as "nuisance" animals, three of the individuals were infected with sarcoptic mange during the time they were exhibiting these behaviors (Gehrt et al. 2009).

Sarcoptic mange is a condition caused by infection of the parasitic mite,

Sarcoptes scabiei (Acariformes:Sarcoptidae) (Fain 1978), and is a major disease of

coyotes throughout their range. Sarcoptic mange can be transmitted either directly through physical contact or indirectly through the use of shared dens (Bornstein et al. 2001). The mite burrows into the epidermis of an infected individual, causing a hypersensitive reaction and intense pruritis (Bornstein et al. 2001). Common signs of sarcoptic mange include alopecia, hyperkeratosis of the epidermis and crusted lesions (Pence and Ueckermann 2002). Canids severely infected with sarcoptic mange typically have reduced body mass and fat reserves (Pence et al. 1983, Gortazar et al. 1998). Sarcoptic mange is frequently fatal for the infected individual, but mortality rates vary by host species (Pence and Ueckermann 2002).

Infection by disease or parasites can be a factor contributing to abnormal behavior in individuals. Numerous examples of parasitism affecting the behavior of mammals can be found in the literature, including examples such as chronic wasting disease making deer more likely to be depredated (Krumm et al. 2009) or hit by a vehicle (Krumm et al. 2005) or rabies increasing aggressive behavior in raccoons (Rosatte et al. 2006). Infection with sarcoptic mange could affect the behavior of coyotes and cause them to exhibit behavior that makes them more likely to be considered a "nuisance" animal by the public, particularly in an urban landscape.

Sarcoptic mange infection can also influence space use by a wildlife population. In an epizootic of red fox in Bristol, UK, Soulsbury et al. (2007) estimated that 90% of foxes living in urban areas died during the peak of the phase. Over the years after the epizootic, as red fox populations slowly started to increase, average territory size was still 10 times the size of pre-epizootic territories, presumably due to the lack of competition

from neighbors due to mortality (Newman et al. 2002). Additionally, while researchers observed litters of mixed-parentage during the pre-mange period, there was no evidence of mixed-parentage litters after the mange epizootic began (Iossa et al. 2008). Groups of fox, living in areas of low-population densities left by the epizootic of mange, also showed a tendency toward female biased social groups and female philopatry (Iossa et al. 2009).

The overarching objective for my study was to determine if sarcoptic mange alters the behavior of urban coyotes. Specifically, I sought to answer the following questions regarding habitat use of sarcoptic mange-infected coyotes. 1) Do sarcoptic mange-infected coyotes select for residential habitat types compared to uninfected individuals? I predicted that individuals with sarcoptic mange would spend more time in residential areas than uninfected coyotes. 2) Does the amount of residential habitat used by mange-infected coyotes increase as the severity of mange infection increases? In this case, I predicted that as the disease progressed and the individual's physical condition worsened, that individual would spend more time in residential areas.

STUDY SITE

Field work was conducted in the Chicago metropolitan area, which comprises Cook, DuPage, Kane, Lake, Will and McHenry counties, and contains the third most populated city in the United States. Cook County contains over five million residents (2010 U.S. census data). Land cover in this area was estimated to be 30% urban, 16% natural areas, 33% agricultural and 21% unassociated vegetation in 1997 (Wang and Moskovits 2001). One of the notable features of this metropolitan area is the system of

county forest preserves, which are protected from urban development and provide habitat for a wide variety of species within the urban environment. Most of the captures and radio-telemetry took place in the northwestern part of the metropolitan area (Figure 3.1). Radio-collared individuals frequently dispersed beyond county boundaries and I continued to track their movements even though they had left the main study area. Therefore, instead of establishing strict boundaries for the study area I allowed for the movements of individual coyotes to determine the boundaries of the study area.

METHODS

Field Methods

Capture.- Adult coyotes were captured opportunistically between 2000 and 2011 using a combination of cable restraint devices and padded MB-650 foothold traps (Minnnesota Trapline Products, Pennock, MN). Upon capture, individuals were immobilized using an injection of Telazol (Fort Dodge Animal Health, Fort Dodge, IA). Once sedated, individuals were weighed, marked with individually numbered ear tags (NASCO Farm & Ranch, Fort Atkinson, WI), morphometric measurements were taken, and each individual was fitted with a very-high-frequency collar (Advanced Telemetry Systems, Isanti, MN).

While the animal was sedated, we examined each coyote for the physical signs of sarcoptic mange, specifically crusted lesions and hairless patches on the coyote's epidermis. If these physical signs were present, a skin scraping was taken by scraping a scalpel blade along the edge of the lesion. The resulting sample was examined under a microscope for evidence of sarcoptic mites. Unfortunately, this particular test tends to be

inconclusive because of the lack of sarcoptic mites on the skin in the later stages of disease (Bornstein et al. 2001, Pence and Ueckermann 2002). As such, my diagnosis of infection by sarcoptic mange was based primarily on the physical signs. These diagnoses are supported by the fact that during twenty years of disease surveillance by the Cook County Forest Preserve District and the pathology lab at Brookfield Zoo, skin biopsies routinely confirmed the presence of *Sarcoptes scabiei* mites in the skin of individuals diagnosed with sarcoptic mange based on physical signs (Figure 3.2) (C. Anchor, Cook County Forest Preserve District, unpublished data).

Upon recovery from the sedative the animal was released at the capture site.

Trapping and handling protocols were approved by the Ohio State University's

Institutional Animal Care and Use Committee (IACUC 2003R0061) and conformed to protocols set forth by the American Society of Mammalogists (Sikes et al. 2011).

Radio telemetry.- Locations were obtained on radio-collared coyotes using a truck-mounted yagi system. Locations were obtained via triangulation, and calculated with Locate III (Truro Computing, Nova Scotia, Canada) allowing for a maximum error ellipse of 50,000 m². Mean error (± standard deviation) for triangulations was calculated using test transmitters as 108±87 m (Morey 2004). Day locations were obtained a minimum of once per week, and night locations were obtained sequentially with a minimum of one hour between each subsequent location. During the course of radio-tracking the opportunity often arose to obtain a visual of a radio collared individual. This gave me the opportunity to diagnose an individual as being infected with sarcoptic mange when signs of the disease developed during the time period post-capture. Upon sighting

an individual, myself or a technician would attempt to determine if mange was present based upon any signs of alopecia on the individual. If alopecia was noted then the observer estimated the percentage of surface area affected on the individual in order to classify the progression of infection.

Mortalities.- Radio-collared individuals who died during the project were recovered as quickly as possible following mortality. Individuals were taken to Brookfield Zoo's pathology laboratory for a full necropsy in order to determine cause of death and to record any underlying conditions (i.e. parasite burden, previous injuries) in the animal. Causes of mortality were classified as either vehicle trauma, shot, mangerelated, unknown and other. In animals with suspected cases of sarcoptic mange, the laboratory took skin biopsies and examined under a microscope by a trained pathologist. In some cases, Sarcoptes scabiei mites were present in the skin sample. These mites were identified as Sarcoptes scabiei based on the placement of the channels in the epidermis and the location of the mite within the stratus corneum (C. Anchor, CCFPD, pers. communication).

Analysis

Compositional Analysis.- My first objective was to determine which habitat types were selected for and avoided by mange infected coyotes and uninfected individuals, and determine if there were differences between the two groups. Habitat selection can occur at three spatial scales. First order is the geographic range of a species, second order involves the selection of a home range within a geographic range, and finally third order involves selection of habitat within an animal's home range (Johnson 1980). Analysis

was restricted to third order selection, since I was interested in the habitats that animals selected within their home ranges.

While a variety of methods exist to measure habitat selection, compositional analysis is a method for determining habitat selection using the individual as the unit of measurement and is ideal in studies that utilize radio-collared individuals (Aebischer 1993). Compositional analysis generates a selection ratio (w_i) based on the proportion of habitat used over the proportion available to that individual

$$w_i = \frac{o_i}{\pi_i}$$

where o=used habitat, π = available habitat and i= habitat type. The differences in the natural log of these selection ratios (d_i) between each combination of habitat types for each individual are then calculated

$$d_i = \ln(w_i) - \ln(w_j)$$

where i and j= two different habitat types. An overall test for selection is performed using a Wilk's λ test. If the overall test is significant (P< 0.05), paired t-tests are then used to compare resource types (Manly et al. 2010).

Since my objective was to determine whether mange-infected individuals select habitat differently than uninfected individuals I performed a compositional analysis on two separate groups of animals. The first group, hereafter referred to as the case group, contained study animals diagnosed with sarcoptic mange. The second group, hereafter

referred to as the control group, contained an equal number of study animals that were mange free.

The case group consisted of all mange infected animals with over 30 locations taken during the time period when the individual was showing signs of sarcoptic mange. I used the visual observations recorded during radio telemetry to calculate the estimated infection date. I used the date midpoint between the last date the animal was seen healthy and when the animal was first seen showing signs of mange infection as the estimated infection date. Similarly, I calculated a recovery date in individuals who recovered from mange by calculating the midpoint between when the animal was last seen showing signs and when it was seen without signs. The control group consisted of individuals randomly selected from a pool of radio-collared animals who were being radio-tracked during the same time period as the case interval, and had the same sex and residency status as the case animal (Table 3.2).

Habitat was classified based on the land cover classifications from the Illinois Gap
Analysis Project (Illinois Natural History Survey, Champaign, IL). The original 27 land
cover categories were combined into three categories; Altered, Residential and Natural.
While it may have been preferable to take a more detailed look at habitat types by
including more land cover categories as separate groups, compositional analysis requires
that habitat types be available to all animals in order to compare log-ratios (Aebischer
1993). I therefore combined several habitat classes into more general habitat classes to
eliminate categories in which individuals had zero availability (Table 3.2). One caveat of
compositional analysis is that it is prone to type I errors when zero values are contained

in the estimates for used habitat. To reduce this error I replaced zero values with a value of 0.07% (Bingham and Brennan 2004).

Available habitat was defined by the percentage habitat types within a 95% minimum convex polygon (MCP), calculated using the mcp function in the package adehabitatHR in R (Calenge 2006). Habitat use is defined as the percentage of points within each habitat type. Both available and used habitat types were estimated using ARCMap 10 (ESRI, Redlands, CA). I used the compana function in the R package adehabitat (Calenge 2006) to calculate the rankings of habitat selection and perform an overall test of whether habitat selection was evident. I also analyzed the selection ratios and habitat use data using separate Multivariate Analysis of Variance's (MANOVA) to detect any differences between the two groups in either the mean selection ratio per habitat type or the mean percentage of habitat use per habitat type. When the overall MANOVA test was significant I performed individual Analysis of Variance (ANOVA) tests to determine which habitat types differed between the groups. Selection ratios were transformed using a square root transformation to improve normality. Habitat use was transformed using an arcsin(square root) transformation to improve homoscedasticity.

Changes in Habitat Use.- My second objective was to determine whether use of residential areas increased as sarcoptic mange infection progressed to a terminal stage. This portion of analysis restricted the time period examined to 180 days before death and after, and only included animals that died of mange-related causes. I chose to restrict the analysis to animals that died of mange-related causes, because it was certain that these animals had progressed through all three classes of sarcoptic mange infection. Each 180

day period was partitioned into three smaller periods of 60 days (Figure 3.4). Only animals that died of causes related to sarcoptic mange and had greater than 10 locations in each 60-day period were included in this analysis. Once again, I used the habitat classifications provided by the Illinois Gap Analysis Survey (Illinois Natural History Survey, Champaign, IL). As I was primarily interested in how use of residential areas changed, I only looked at the residential habitat categories, high-density residential, medium-density residential and low-density residential. I used ArcMap 10 (ESRI, Redlands, CA) to determine the percentage of locations per time period in each habitat type. I analyzed the percentages of habitat use with a MANOVA to determine if the mean percentage of locations in each residential habitat type differed between time periods. When the overall MANOVA showed a significant difference, I performed individual ANOVA tests on each residential habitat type to determine which habitat types differed between time periods.

RESULTS

Habitat Selection

During March 2000 through April 2011, 310 coyotes (149 F, 161 M) were captured and radio-collared. Of 310 coyotes, 49 individuals were diagnosed with sarcoptic mange. Of those 49 animals, 16 had more than 30 locations recorded after they were estimated to have been infected with sarcoptic mange. These 16 animals comprised the case group, with another 16 comprising the control group (table 3.2). The case group had a mean home-range area of 6.28±1.16 km² (mean ±SE), while the case group had a

mean home range size of $4.49\pm0.85 \text{ km}^2$. There was no significant difference in mean home range size between the two groups (t_{15} =1.25 P=0.22).

The overall tests for habitat selection for both the case (Wilks λ =0.30, P=0.01) and control (Wilks λ =0.59, P<0.001) groups were significant. In both groups, natural and altered habitats were preferred over residential habitats, although there was not a difference between preference of altered and natural habitats (Table 3.3).

There were no observed differences between the mean selection ratio for habitat types between case and control groups (MANOVA, $F_{3,28}$ =0.66 P=0.588) (Figure 3.5). However, there was a difference between the mean use of habitat types between case and control groups (MANOVA, $F_{3,28}$ =3.42 P=0.031) (Figure 3.6). Specifically, the mean percentage of altered habitat used increased from 41±4 % for the case group to 61±5 % for the control group (ANOVA, $F_{1,30}$ =10.90 P=0.002). The natural habitat type also showed a significant difference (ANOVA, $F_{1,30}$ =7.02 P=0.013) between the two groups with a mean use of 30±5% for the control group and a mean use of 46±5 % for the case group (Figure 3.6).

Shifts in Habitat Use

Of 49 coyotes diagnosed with sarcoptic mange there were 12 animals that died of mange-related causes who had a sufficient number of locations in each period of the 180-day time span prior to death to be included in this analysis. There was a weak significant difference in residential habitat use between the three time periods (MANOVA, $F_{2,33}$ =1.89 P=0.096). There was no difference in the mean habitat used of high density residential (ANOVA, $F_{2,33}$ =1.32 P=0.280) or low density residential (ANOVA,

 $F_{2,33}$ =1.26 P=0.297), but there was a significant difference in the mean percentage of medium density residential habitat used (ANOVA, $F_{2,33}$ =4.10 P=0.026) (Figure 3.7). The percentage of locations recorded in medium-density residential habitat was steady at the two periods farthest from death, with mean values of 3±1% for the period 121-180 days prior to death and 4±1% for the period 61-120 days prior to death. However, during the period 1-60 days prior to death there was an increase to a mean percentage of medium density residential habitat used to 9±2%. There were no respective increases or decreases for either low density residential habitat or high density residential habitat.

DISCUSSION

While human/coyote conflict can take many forms, understanding why these conflicts occur is important in both preventing them from occurring and responding to them once they have transpired. Most coyotes that live in the urban environment show avoidance of humans and habitat types with heavy human use, and this is a key component in preventing many of the potential conflicts that would arise from having humans and coyotes in such close proximity (Gibeau 1998, Grinder and Krausman 2001b, Gehrt et al. 2009). Understanding why some animals deviate from these typical behaviors can help us understand why conflicts occur. Disease, specifically sarcoptic mange, is a potential reason for some individuals, to ignore their wariness of humans, and behave in a manner that makes them become a "nuisance" animal.

Selection of Habitat

I found no evidence that individuals infected with sarcoptic mange selected habitats differently than uninfected individuals during the time period from initial

infection to resolution of disease (i.e. death or recovery). It is important to note, however, that this analysis included all coyotes infected with sarcoptic mange regardless of the degree of infection. Individuals initially diagnosed with a relatively minor infection, in the class I category of infection, were included in the case group. Based on the minimal physical signs it is likely these individuals had a low parasite burden, possibly too low to prompt a behavioral response to the infection. The results from this analysis confirm what has been reported urban coyote literature, which is that urban coyotes avoid human dominated habitats (Gibeau 1998, Gehrt et al. 2009, Grubbs and Krausman 2009). Of the habitat classes used in this study, the residential class contained the areas of highest human activity and both groups showed an avoidance of this habitat type by using it less than its availability within their home range.

While there was a difference between the mean use of both natural and altered habitats (Figure 3.5), there was no difference between the mean selection ratios between the two groups (Figure 3.4). This implies that the significant differences in mean use may be more an indication of differences in the availability of habitat types between the two groups. Considering that neither case nor control groups showed preference for natural or altered habitats from the compositional analysis, it seems reasonable that these groups would use these landscape types in the proportions available to them. At the third order scale of selection there is no evidence of a difference in how these two groups of coyotes use residential habitat within their territories. The evidence supports the idea that both groups avoid the residential areas within their territories and use altered and natural habitat types proportionally to their availability within their territory.

An unknown variable related to the composition of habitat types that make up the home range may play a role in an individual's likelihood of contracting sarcoptic mange. Mange-infected coyotes tended to have more natural habitat available to them than uninfected coyotes did, and uninfected coyotes tended to have more altered habitat available to them than mange-infected coyotes. The reasons for this difference are unclear and it is possible that the differences shown are an artifact of small-sample sizes in the two groups.

The fact that I was unable to show any evidence of differences in habitat selection between infected and uninfected individuals over the entire period of infection does not necessarily mean that sarcoptic mange has no have an effect on how individuals use territory. When locations from all periods of infection are pooled together it may mask any shifts in habitat use that occur in the later stages of infection. Pence et.al. (1994) noted that while intraperitoneal fat indices did not differ between individuals with class II infections (5-50% body coverage) and uninfected individuals, there was a difference between individuals with class III infections (>50% body coverage) and uninfected individuals. It would not be unusual for changes in behavior to reflect the condition of the individual, and only manifest once level of infection reaches a threshold level. *Shifts in Habitat Use*

There are several possible reasons for changes in habitat usage due to mange infection to occur: infected individuals may be seeking food sources around these areas, such as garbage or food left outdoors for pets, they may be using residential areas as a

refuge from other coyotes, or they may be seeking shelter provided by residential buildings

Several previous studies have looked at the diets of coyotes living in urban environments. A previous study in this study area determined that anthropogenic food is not a major part of the urban coyote's diet, based on the examination of scat. In fact, anthropogenic food appeared in less than 2% of all scats collected in the Chicago metropolitan region (Morey et al. 2007). It has been shown that mange-infected coyotes in some areas feed on carrion more frequently than uninfected individuals (Todd et al. 1980), implying that there is a diminished capacity to forage and catch their own food. Frequently, individuals who died of sarcoptic mange were extremely emaciated, and upon necropsy were found to have empty stomachs. It is possible that coyotes, severely debilitated from mange, may become hungry enough to venture into residential areas more frequently looking for food. However, the fact that many coyotes recovered dead with severe sarcoptic mange inside or near residential areas were extremely emaciated implies that these individuals were unsuccessful at finding food in these areas.

A second possibility is mange-infected coyotes are spending more time in medium-density residential areas due to the decreased likelihood of encountering a conspecific. Coyotes are territorial animals and spend a great deal of time monitoring and defending that territory from the intrusion of other coyotes. In Yellowstone National Park, five neighboring packs of coyotes were observed defending their territories an average of once every 22.4 hours (Gese 2001). Debilitation from sarcoptic mange infection could cause a resident animal to lose control of its territory. Even if an

individual is not forced entirely out of its territory, it may be forced into the lower quality habitats (i.e. residential areas). Similar behaviors to those of individuals severely infected with sarcoptic mange, specifically an increase in the use of decks and patios as resting areas, have been observed in urban coyotes of advanced age who have lost their status as alpha members of a pack (Way and Timm 2008).

A final potential reason for mange-infected coyotes to spend more time in residential areas is that they are seeking out the shelter that residential buildings provide. The majority of deaths from sarcoptic mange in this area occur in winter, specifically in December and January (Chapter 2). It is possible that the combination of poor physical condition and cold winter temperatures common to the area during these months, are causing these individuals to seek shelter near residential homes. It was common during this study to find individuals who had expired from mange-related causes curled up next to residential buildings. Several transient individuals who had dispersed out of the study area and died of mange-related causes later, were recovered due only to the fact that they died in close proximity to residential buildings, and the residents alerted us by calling the phone number on the collars. Several other individuals who died of mange-related causes were recovered curled up inside ground dens or tree cavities. Previous studies of mange in other wildlife species have also noted that often individuals will seek out shelter, such as barns, haystacks or ground dens, in the late stages of infection and will often die in these spots (Shelley and Gehring 2002, Wydeven et al. 2003).

The majority of the time an individual is infected with sarcoptic mange its use of habitat is unaltered from that of a healthy coyote. However, during the later stages of the

disease individuals do begin to alter their habitat use in the form of spending more time in residential patches. Similar patterns were observed in coyotes affected with canine heartworm (*Diroflaria immitis*). Activity of heart-worm infected coyotes decreased over time, while uninfected coyotes' activity patterns remained steady, and significant decreases in activity correlated to heart worm burden were only observed during the last two months of life (Sacks and Blejwas 2000). Additionally, some of the infected individuals were frequently observed in residential areas during the day light hours, which is extremely unusual for urban coyotes and suggests another significant change in behavior. This does not imply that coyotes go out and seek residential areas outside of their home range, as we observed no trends of habitats shifting, but more that coyotes will spend more time in the residential patches already contained within their home ranges, as opposed to quickly moving through them enroute to more natural habitat patches. The trend of coyotes increased use of medium-density residential areas might have been stronger and variance in the sample decreases if all individuals had those habitat patches available to them, however several individuals had home ranges entirely within forest preserves and only had small patches of medium-density residential habitat available to them.

While I have focused primarily on the effects of sarcoptic mange on the individual coyote, sarcoptic mange infection on individuals may have important implications in regard to coyote social dynamics. Prevalence of mange is not typically evenly distributed across landscape over time in an enzootic infection. Local areas may experience periods of higher prevalence, while other areas have none or low levels of

sarcoptic mange (Gortazar et al. 1998). While the infection in the Chicago area has not seen widespread mortality similar to epizootic infections found in the foxes of Bristol (Soulsbury et al. 2007), local infections may cause high levels of mortality in localized areas. High localized mortality can have important impacts on social dynamics in specific geographic areas, including localized decreases in abundance and the expansion of surviving pack's territories similar to the effects seen in the Bristol fox population (Newman et al. 2002).

The results from this study showed patterns of behavior similar to those seen in other studies of animals infected with sarcoptic mange. In coyotes and other species, included other members of the canid family, severely infected individuals are frequently described as having lost their sense of wariness and are often easily approached (Todd et al. 1980, Overskaug 1994, Skerratt et al. 2004). This case differs from previous studies though, since it occurs in an urban environment and because of the high potential for conflict in these urban and suburban regions. While the sight of a mangy coyote in rural settings may not raise many concerns, the presence of a diseased animal in a metropolitan subdivision in close proximity to residences has the potential to cause a difficult political situation where homeowners request management officials take some sort of action, particularly if an animal is seen regularly during the daytime. However, while these individuals may be spending more time in residential areas than a healthy coyote would, that does not mean they are inherently dangerous. Most reports on severely mangeinfected coyotes from rural areas report severely infected coyotes being listless and unwary of humans (Trainer and Hale 1969).

While this study has looked at two time scales at which mange infected coyote's behavior could be altered, there is potential for more research to be done on this topic. All of the radio-tracking on this project took place using VHF collars, which limits the number of locations that it is possible to obtain. New technology, such as GPS collars has made the acquisition of hundreds of points with higher precision in a relatively small time frame possible. Using methods such as these, the limiting factor of being able to obtain enough points to perform a detailed analysis would be removed, and there is potential to gain an even clearer picture of how sarcoptic mange affects the habitat use of the urban coyote.

MANAGEMENT IMPLICATIONS

There is evidence that sarcoptic mange could play a role in whether a coyote becomes a "nuisance" animal or not. By increasing the amount of time these individuals are spending in residential areas, they are more likely to be observed by residents, which can lead to nuisance reports. Land managers should be aware of the role mange can play in nuisance reports, and inquire about the animal's physical appearance when interviewing those who report nuisance animals. By increasing our knowledge as to what factors in the environment can increase the probability of human coyote conflict, managers may be able to make more informed decisions about how to prevent and respond to such incidents.

LITERATURE CITED

- Aebischer, N. J. 1993. Compositional analysis of habitat use from animal radio-tracking data. Ecology 74:1313-1325.
- Atwood, T. C., H. P. Weeks, and T. M. Gehring. 2004. Spatial ecology of coyotes along a suburban-to-rural gradient. Journal of Wildlife Management 68:1000-1009.
- Baker, R. O., and R. M. Timm. Management of conflicts between urban coyotes and humans in southern California. Proceedings of Vertebrate Pest Conference. 18:299-312.
- Bingham, R. L., and L. A. Brennan. 2004. Comparison of type I error rates for statistical analyses of resource selection. Journal of Wildlife Management 68:206-212.
- Bornstein, S., T. Morner, and W. M. Samuel. 2001. *Sarcoptes scabiei* and sarcoptic mange. Pages 107-119 *in* W. M. Samuel, M. J. Pybus, and K. A.A., editors. Parasitic Diseases of Wild Mammals. Iowa State University Press, Ames, IA.
- Calenge, C. 2006. The package "adehabitat" for the R software: A tool for the analysis of space and habitat use by animals. Ecological Modelling 197:516-519.
- Fain, A. 1978. Epidemiological problems of scabies. International Journal of Dermatology 17:20-30.
- Fedriani, J. M., T. K. Fuller, and R. M. Sauvajot. 2001. Does availability of anthropogenic food enhance densities of omnivorous mammals? An example with coyotes in southern California. Ecography 24:325-331.
- Gehrt, S. D., C. Anchor, and L. A. White. 2009. Home range and landscape use of coyotes in a metropolitan landscape: conflict or coexistence? Journal of Mammalogy 90:1045-1057.
- Gese, E. M. 2001. Territorial defense by coyotes (*Canis latrans*) in Yellowstone National Park, Wyoming: Who, how, where, when and why. Canadian Journal of Zoology 79:980-987.
- Gibeau, M. L. 1998. Use of urban habitats by coyotes in the vicinity of Banff Alberta. Urban Ecosystems 2:129-139.
- Gompper, M. E. 2002. Top carnivores in the suburbs? Ecological and conservation issues raised by colonization of northeastern North America by coyotes. BioScience 52:185-190.
- Gortazar, C., R. Villafuerte, J. C. Blanco, and D. Fernandez-De-Luco. 1998. Enzootic sarcoptic mange in red foxes in Spain. Zeitschrift fuer Jagdwissenschaft 44:251-256.

- Grinder, M., and P. R. Krausman. 2001a. Morbidity-mortality factors and survival of an urban coyote population in Arizona. Journal of Wildlife Diseases 37:312-317.
- Grinder, M. I., and P. R. Krausman. 2001b. Home range, habitat use, and nocturnal activity of coyotes in an urban environment. The Journal of Wildlife Management 65:887-898.
- Grubbs, S. E., and P. R. Krausman. 2009. Use of urban landscape by coyotes. Southwestern Naturalist 54:1-12.
- Iossa, G., C. D. Soulsbury, P. J. Baker, K. J. Edwards, and S. Harris. 2009. Behavioral changes associated with a population density decline in the facultatively social red fox. Behavioral Ecology 20:385-395.
- Iossa, G., C. D. Soulsbury, P. J. Baker, and S. Harris. 2008. Body mass, territory size, and life-history tactics in a socially monogamous canid, the red fox *Vulpes vulpes*. Journal of Mammalogy 89:1481-1490.
- Johnson, D. H. 1980. The comparison of usage and availability measurements for evaluating resource preference. Ecology 61:65-71.
- Krumm, C. E., M. M. Conner, N. T. Hobbs, D. O. Hunter, and M. W. Miller. 2009. Mountain lions prey selectively on prion-infected mule deer. Biology Letters 6:209-211.
- Krumm, C. E., M. M. Conner, and M. W. Miller. 2005. Relative vulnerability of chronic wasting disease infected mule deer to vehicle collisions. Journal of Wildlife Diseases 41:503-511.
- Lukasik, V. M., and S. M. Alexander. 2011. Human-coyote intereactions in Calgary, Alberta. Human Dimensions of Wildlife 16:114-127.
- Manly, B. F. J., L. L. McDonald, D. L. Thomas, T. L. McDonald, and W. P. Erickson. 2010. Resource Selection by Animals. 2nd edition. Kluwer Academic Publishers, Dordrecht.
- McClennen, N., R. R. Wigglesworth, S. H. Anderson, and D. G. Wachob. 2001. The effect of suburban and agricultural development on the activity patterns of coyotes (*Canis latrans*). American Midland Naturalist 146:27-36.
- Morey, P. S. 2004. Landscape use and diet of coyotes, *Canis latrans*, in the Chicago metropolitan area. Thesis, Utah State University, United States -- Utah.
- Morey, P. S., E. M. Gese, and S. Gehrt. 2007. Spatial and temporal variation in the diet of coyotes in the Chicago Metropolitan Area American Midland Naturalist 158:147-161.

- Newman, T. J., P. J. Baker, and S. Harris. 2002. Nutritional condition and survival of red foxes with sarcoptic mange. Canadian Journal of Zoology 80:154-161.
- 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 E. Ueckermann. 2002. Sarcoptic mange in wildlife. Revue Scientifique et Technique (International Office of Epizootics) 21:385-398.
- Pence, D. B., and L. A. Windberg. 1994. Impact of a sarcoptic mange epizootic on a coyote population. Journal of Wildlife Management 58:624-633.
- Pence, D. B., L. A. Windberg, B. C. Pence, and R. Sprowls. 1983. The epizootiology and pathology of sarcoptic mange in coyotes, *Canis latrans*, from south Texas. The Journal of Parasitology 69:1100-1115.
- Quinn, T. 1995. Using public sighting information to investigate coyote use of urban habitat. Journal of Wildlife Management 59:238-245.
- _____. 1997a. Coyote (*Canis latrans*) food habits in three urban habitat types of western Washington. Northwest Science 71:1-5.
- _____. 1997b. Coyote (*Canis latrans*) habitat selection in urban areas of western Washington via analysis of routine movements. Northwest Science 71:289-297.
- Rosatte, R., K. Sobey, D. Donovan, L. Bruce, M. Allan, A. Silver, K. Bennett, M. Gibson, H. Simpson, C. Davies, A. Wandeler, and F. Muldoon. 2006. Behavior, movements, and demographics of rabid raccoons in Ontario, Canada: management implications. Journal of Wildlife Diseases 42:589-605.
- Sacks, B. N., and K. M. Blejwas. 2000. Effects of canine heartworm (*Dirofilaria immitis*) on body condition and activity of free-ranging coyotes (*Canis latrans*). Canadian Journal of Zoology 78:1042-1051.
- Shargo, E. S. 1988. Home range, movements, and activity patterns of coyotes (*Canis latrans*) in Los Angeles suburbs. Dissertation, University of California, Los Angeles, Los Angeles, CA.
- Shelley, D. P., and T. M. Gehring. 2002. Behavioral modification of gray wolves, *Canis lupus*, suffering from sarcoptic mange: Importance of sequential monitoring. Canadian Field-Naturalist 116:648-650.
- Sikes, R. S., W. L. Gannon, D. S. Carroll, B. J. Danielson, J. W. Dragoo, M. R. Gannon, W. L. Gannon, D. W. Hale, C. McCain, D. K. Odell, L. E. Olson, S. Ressing, R. M. Timm, S. A. Trewhitt, and J. E. Whaley. 2011. Guidelines of the American

- Society of Mammalogists for the use of wild mammals in research. Journal of Mammalogy 92:235-253.
- Skerratt, L. F., J. H. L. Skerratt, R. Martin, and K. Handasyde. 2004. The effects of sarcoptic mange on the behaviour of wild common wombats (*Vombatus ursinus*). Australian Journal of Zoology 52:331-339.
- Soulsbury, C. D., G. Iossa, P. J. Baker, N. C. Cole, S. M. Funk, and S. Harris. 2007. The impact of sarcoptic mange *Sarcoptes scabiei* on the British fox *Vulpes vulpes* population. Mammal Review 37:278-296.
- Timm, R. M., R. O. Baker, J. R. Bennett, and C. C. Coolahan. Coyote attacks: An increasing suburban problem. Proceedings of Vertebrate Pest Conference 21:67-88.
- Todd, A. W., J. R. Gunson, and W. M. Samuel. Sarcoptic Mange: An important disease of coyotes and wolves of Alberta, Canada. Pages 706-729 in First Worldwide Furbearer Conference Proceedings, 3-11 August, 1980, Frostburg, Maryland, USA.
- 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.
- United States Census Bureau. 2010. < http://2010.census.gov/2010census/>. Accessed 11 December 2011.
- Wang, Y., and D. K. Moskovits. 2001. Tracking fragmentation of natural communities and changes in land cover: applications of landsat data for conservation in an urban landscape (Chicago Wilderness). Conservation Biology 15:835-843.
- Way, J. G., and B. C. Timm. 2008. Nomadic behavior of an old and formerly territorial eastern coyote (*Canis latrans*). Canadian Field-Naturalist 122:316-322.
- White, L. A., and S. D. Gehrt. 2009. Coyote attacks on humans in the United States and Canada. Human Dimensions of Wildlife 14:419-432.
- Wydeven, A. P., S. R. Boles, R. N. Schultz, and C. J. T. Doolittle. 2003. Death of gray wolves, *Canis lupus*, in porcupine, *Erethizon dorsatum*, dens in Wisconsin. Canadian Field-Naturalist 117:469-471.

FIGURES AND TABLES

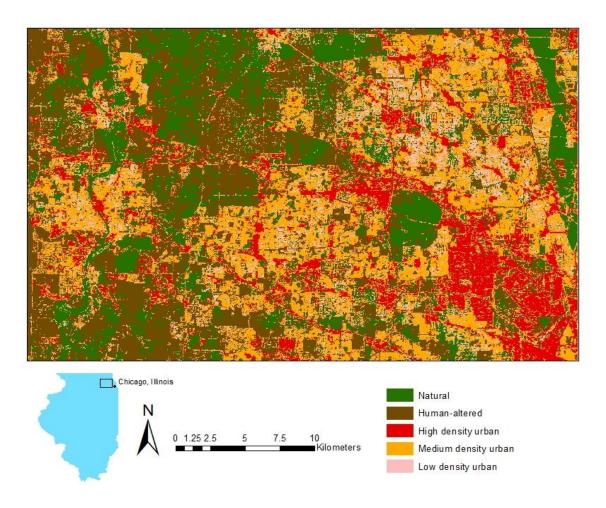


Figure 3.1. Aerial photo of the northwestern suburbs in the Chicago metropolitan area showing the habitat classifications used in the compositional analysis of habitat selection by coyotes.

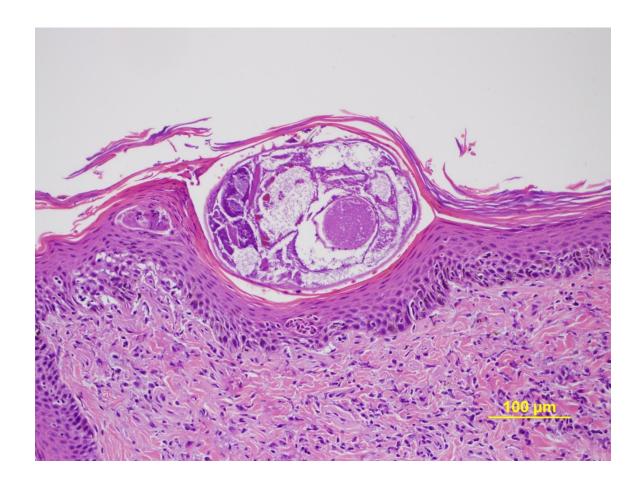


Figure 3.2. Photograph of magnified cross-section of skin from a mange-infected coyote from the Chicago Metropolitan Area. The round object in the center is the cross-section of a *Sarcoptes scabiei* mite. Picture courtesy of Cook County Forest Preserve District.

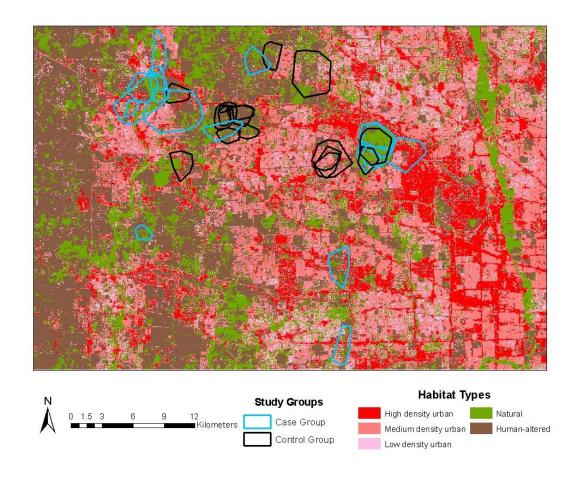


Figure 3.3. 95% MCP home ranges of mange infected (Case Group) and uninfected (Control Group) coyotes in the Chicago metropolitan area between 2000 and 2011.

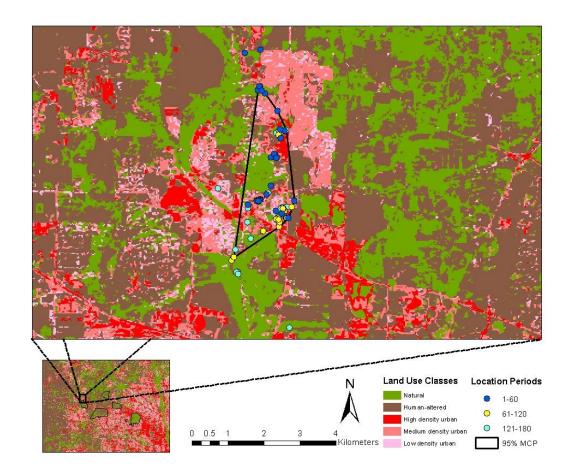


Figure 3.4. Radio-telemetry locations, in intervals of 1-60, 61-120 and 121-180 days before death from sarcoptic mange, of coyote #432 during the time this individual was infected with sarcoptic mange.

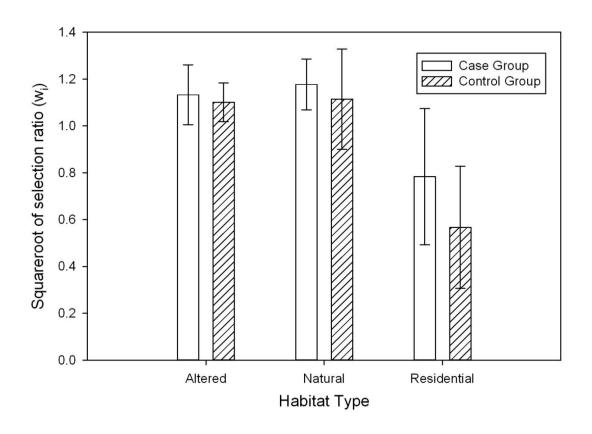


Figure 3.5. Differences in the squareroot of mean selection ratios (w_i) of habitat preference by mange infected (case) and uninfected (control) coyotes in the Chicago Metropolitan Area between 2000 and 2011. Error bars indicate 95% confidence intervals.

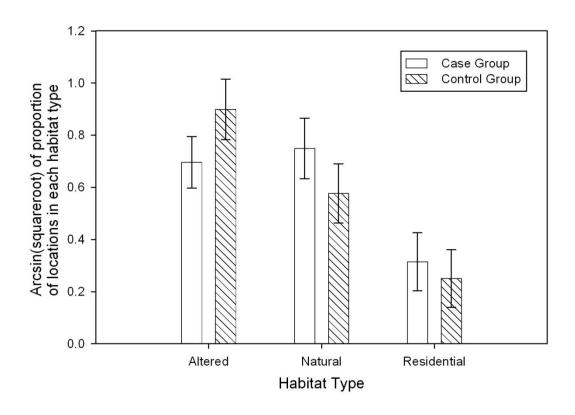


Figure 3.6. Differences in the arcsin squareroot of mean habitat use by mange infected (case) and uninfected (control) coyotes in the Chicago Metropolitan Area between 2000 and 2011. Error bars indicate 95% confidence intervals.

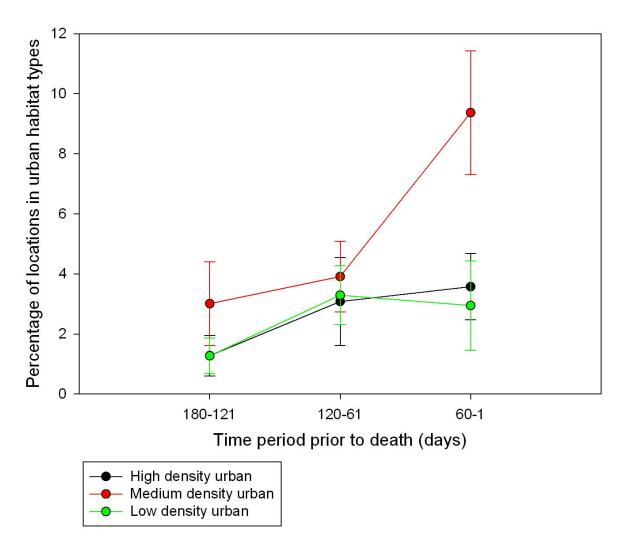


Figure 3.7. Changes in the mean percentage of urban habitat types used by coyotes diagnosed with sarcoptic mange during the time period 180 days prior to death between 2000 and 2011 in the Chicago Metropolitan Area. Error bars represent the standard error of the mean.

Case Group (n=16)						
		Habitat Type				
Habitat Type	Altered	Residential	Natural	Rank		
Altered		+++	+	1		
Residential				0		
Natural	-	+++		2		
Control Group (n=16))					
		Habitat Type				
Habitat Type	Altered	Residential	Natural	Rank		
Altered		+++	+	2		
Residential				0		
Natural	-	+++		1		

Table 3.1. Ranking of habitat preference by coyotes in the Chicago Metropolitan Area between 2000 and 2011. A plus (+) indicates the habitat type in the row heading is used more than the habitat type in the column heading, a minus (-) indicates the habitat type in the row heading is used less than the habitat type in the column heading, statistically significant (p<0.05) results are indicated by a tripling of the appropriate symbol (i.e. +++ or ---). Rank indicates the preference of the habitat type with the highest number being the most preferred.

IL-GAP Habitat Classification	Land Cover Type	Habitat Type
Corn	Agriculture	Altered
Soybeans	Agriculture	Altered
Other Small Grains and Hay	Agriculture	Altered
Winter Wheat/Soybeans	Agriculture	Altered
Other Agriculture	Agriculture	Altered
Rural Grassland	Agriculture	Altered
Dry Upland	Forested	Natural
Dry-Mesic Upland	Forested	Natural
Mesic Upland	Forested	Natural
Partial Canopy/Savannah Upland	Forested	Natural
Coniferous	Forested	Natural
High Density Urban	Urban Land	Residential
Medium Density Urban	Urban Land	Residential
Low Density Urban	Urban Land	Residential
Urban Open Space	Urban Land	Altered
Shallow Marsh/Wet Meadow	Wetland	Natural
Deep Marsh	Wetland	Natural
Seasonally/Temporarily Flooded	Wetland	Natural
Mesic Floodplain Forest	Wetland	Natural
Wet-Mesic Floodplain Forest	Wetland	Natural
Wet Floodplain Forest	Wetland	Natural
Swamp	Wetland	Natural
Shallow Water	Wetland	Natural
Surface Water	Other	Natural
Barren and Exposed Land	Other	Altered

Table 3.2. Table displaying the reclassification of habitat classifications from the IL-Gap Analysis classification system into the habitat type used to determine habitat selection and use in mange-infected coyotes in the Chicago Metropolitan Area between 2000 and 2011.

				Start of Infection	End of Infection
Pair ID	Sex	Case Ind. ID	Control Ind. ID	Period	Period
1	F	50	38	3/28/2002	8/19/2002
2	F	119	125	1/8/2009	12/22/2009
3	M	120	35	4/6/2004	12/1/2004
4	F	155	259	12/29/2008	1/4/2010
5	M	181 ^a	173 ^a	3/17/2005	10/20/2009
6	M	275 ^b	115 ^b	12/23/2006	7/25/2008
7	F	278	167	9/22/2008	6/14/2010
8	F	313	177	2/17/2011	9/2/2011
9	M	373	446	3/23/2010	11/19/2010
10	F	432	356	5/3/2010	9/24/2010
11	F	444	359	7/2/2010	11/23/2010
12	M	454	369	4/20/2010	1/22/2011
13	F	500	358	4/7/2011	6/4/2011
14	F	110	107	2/3/2004	3/10/2004
15	F	232	248	6/14/2006	1/29/2007
16	M	340	357	8/9/2009	5/27/2010

^aCoyote 181 was on two separate occasions observed healthy and then observed at a later date showing signs of mange, hence locations during the healthy periods, 4/16/2006-5/12/2008 and 12/1/2008-2/19/2009, are removed from analysis.

Table 3.3. Designation of coyotes into case (mange infected) and control (uninfected) groups used to compare habitat selection by coyotes in the Chicago Metropolitan Area between 2000 and 2011. Case and control individuals of the same pair were matched based upon sex, residency status and were both being tracked during the same time periods.

^b Coyote 275 was observed healthy and then observed at a later date showing signs of mange, hence locations during the healthy period, 2/11/2008-3/10/2008, are removed from analysis.

BIBLIOGRAPHY

- Aebischer, N. J. 1993. Compositional analysis of habitat use from animal radio-tracking data. Ecology 74:1313-1325.
- Almberg, E. S., L. D. Mech, D. W. Smith, J. W. Sheldon, and R. L. Crabtree. 2009. A serological survey of infectious disease in Yellowstone National Park's canid community. PloS one 4:e7042.
- Andelt, W. F. 1985. Behavioral ecology of coyotes in south Texas. Wildlife Monographs 94:3-45.
- Andelt, W. F., J. G. Kie, F. F. Knowlton, and K. Cardwell. 1987. Variation in coyote diets associated with season and successional changes in vegetation. Journal of Wildlife Management 51:273-277.
- Anderson, R. M., and R. M. May. 1978. Regulation and stability of host-parasite population interactions: I. Regulatory processes. Journal of Animal Ecology 47:219-247.
- Andrews, J. R. 1983. The origin and evolution of host associations of *Sarcoptes scabiei* and the subfamily *Sarcoptinae Murray*. Acarologia 24:85-94.
- Arlian, L. G., M. S. Morgan, and J. J. Arends. 1996a. Immunologic cross-reactivity among various strains of *Sarcoptes scabiei*. Journal of Parasitology 82:66-72.
- Arlian, L. G., M. S. Morgan, C. M. Rapp, and D. L. Vyszenski-Moher. 1996b. The development of protective immunity in canine scabies. Veterinary Parasitology 62:133-142.
- Arlian, L. G., R. A. Runyan, S. Achar, and S. A. Estes. 1984a. Survival and infectivity of *Sarcoptes scabiei var. canis* and *var. hominis*. Journal of the American Academy of Dermatology 11:210-215.

- Arlian, L. G., R. A. Runyan, and S. A. Estes. 1984b. Cross infestivity of *Sarcoptes scabiei*. Journal of the American Academy of Dermatology 10:979-986.
- Arlian, L. G., R. A. Runyan, L. B. Sorlie, and S. A. Estes. 1984c. Host-seeking behavior of *Sarcoptes scabiei*. Journal of the American Academy of Dermatology 11:594-598.
- Atwood, T. C., H. P. Weeks, and T. M. Gehring. 2004. Spatial ecology of coyotes along a suburban-to-rural gradient. Journal of Wildlife Management 68:1000-1009.
- Baker, P. J., C. B. Dowding, S. B. Molony, C. L. W. Piran, and S. Harris. 2007. Activity patterns of urban red foxes (*Vulpes vulpes*) reduce the risk of traffic-induced mortality. Behavioral Ecology 18:716-724.
- Baker, R. O., and R. M. Timm. 1998. Management of conflicts between urban coyotes and humans in southern California. Proceedings of Vertebrate Pest Conference. 18:299-312.
- Begon, M., J. L. Harper, and C. R. Townsend. 1996. Ecology:Individuals, Populations and Communities. Blackwell Scientific Publications, Oxford.
- Bekoff, M. 1977. Canis latrans. Mammalian Species 79:1-9.
- Bernier, J., P. Brousseau, K. Krzystyniak, H. Tryphonas, and M. Fournier. 1995. Immunotoxicity of heavy metals in relation to Great Lakes. Environmental Health Perspectives 103:23-34.
- Bingham, R. L., and L. A. Brennan. 2004. Comparison of type I error rates for statistical analyses of resource selection. Journal of Wildlife Management 68:206-212.
- Bornstein, S., T. Morner, and W. M. Samuel. 2001. *Sarcoptes scabiei* and sarcoptic mange. Pages 107-119 *in* W. M. Samuel, M. J. Pybus, and K. A.A., editors. Parasitic Diseases of Wild Mammals. Iowa State University Press, Ames, IA.
- Bradley, C. A., and S. Altizer. 2007. Urbanization and the ecology of wildlife diseases. Trends in Ecology & Evolution 22:95-102.
- Brown, J. 2007. The influence of coyotes on an urban Canada goose population in the Chicago metropolitan area. Thesis, The Ohio State University, Columbus, OH.
- Burnham, K. P., and D. R. Anderson. 1998. Model Selection and Inference: A Practical Information-Theoretic Approach. Springer-Verlag, New York, NY.
- Calenge, C. 2006. The package "adehabitat" for the R software: A tool for the analysis of space and habitat use by animals. Ecological Modelling 197:516-519.

- Camkerten, I., T. Sahin, G. Borazan, A. Gokcen, O. Erel, and A. Das. 2009. Evaluation of blood oxidant/antioxidant balance in dogs with sarcoptic mange. Veterinary Parasitology 161:106-109.
- Chamberlain, M. J., and B. D. Leopold. 2001. Survival and cause-specific mortality of adult coyotes (*Canis latrans*) in central Mississippi. American Midland Naturalist 145:414-418.
- Chronert, J. M., J. A. Jenks, D. E. Roddy, M. A. Wild, and J. G. Powers. 2007. Effects of sarcoptic mange on coyotes at Wind Cave National Park. Journal of Wildlife Management 71:1987-1992.
- Ditchkoff, S. S., S. T. Saalfeld, and C. J. Gibson. 2006. Animal behavior in urban ecosystems: Modifications due to human-induced stress. Urban Ecosystems 9:5-12.
- Ezenwa, V. O. 2004. Interactions among host diet, nutritional status and gastrointestinal parasite infection in wild bovids. International Journal for Parasitology 34:535-542.
- Fain, A. 1978. Epidemiological problems of scabies. International Journal of Dermatology 17:20-30.
- Fedriani, J. M., T. K. Fuller, and R. M. Sauvajot. 2001. Does availability of anthropogenic food enhance densities of omnivorous mammals? An example with coyotes in southern California. Ecography 24:325-331.
- Fernandez-Moran, J., S. Gomez, F. Ballesteros, P. Quiros, J. L. Benito, C. Feliu, and J. M. Nieto. 1997. Epizootiology of sarcoptic mange in a population of cantabrian chamois (*Rupicapra pyrenaica parva*) in northwestern Spain. Veterinary Parasitology 73:163-171.
- Gehrt, S. D. 2007. Ecology of coyotes in urban landscapes. Pages 303-311 in Proceedings of 12th Wildlife Damage Management Conference. The Wildlife Society, 9 12 April 2007, Corpus Christi, TX, USA.
- _____. 2010. The Urban Ecosystem. *in* S. D. Gehrt, B. L. Cypher, and S. P. D. Riley, editors. Urban Carnivores. The Johns Hopkins University Press, Baltimore, MD.
- Gehrt, S. D., C. Anchor, and L. A. White. 2009. Home range and landscape use of coyotes in a metropolitan landscape: conflict or coexistence? Journal of Mammalogy 90:1045-1057.

- Gehrt, S. D., and S. P. D. Riley. 2010. Coyotes (*Canis latrans*). Pages 79-95 in S. D. Gehrt, S. P. D. Riley, and B. L. Cypher, editors. Urban Carnivores. The Johns Hopkins University Press, Baltimore, MD.
- Gese, E. M. 2001. Territorial defense by coyotes (*Canis latrans*) in Yellowstone National Park, Wyoming: Who, how, where, when and why. Canadian Journal of Zoology 79:980-987.
- Gese, E. M., O. J. Rongstad, and W. R. Mytton. 1989. Population dynamics of coyotes in southeastern Colorado. Journal of Wildlife Management 53:174-181.
- Gese, E. M., R. L. Ruff, and R. L. Crabtree. 1996. Intrinsic and extrinsic factors influencing coyote predation of small mammals in Yellowstone National Park. Canadian Journal of Zoology 74:784-797.
- Gese, E. M., R. D. Schultz, M. R. Johnson, E. S. Williams, R. L. Crabtree, and R. L. Ruff. 1997. Serological survey for diseases in free-ranging coyotes (*Canis latrans*) in Yellowstone National Park, Wyoming. Journal of Wildlife Diseases 33:47-56.
- Gese, E. M., R. D. Schultz, O. J. Rongstad, and D. E. Andersen. 1991. Prevalence of antibodies against canine parvovirus and canine distemper virus in wild coyotes in southeastern Colorado. Journal of Wildlife Diseases 27:320-323.
- Gibeau, M. L. 1998. Use of urban habitats by coyotes in the vicinity of Banff Alberta. Urban Ecosystems 2:129-139.
- Gier, H. T., S. M. Kruckenberg, and R. J. Marler. 1978. Parasites and Diseases of Coyotes. Pages 37-71 *in* M. Bekoff, editor. Coyotes: Biology, Behavior and Management. The Blackburn Press, Caldwell, NJ.
- Gompper, M. E. 2002. Top carnivores in the suburbs? Ecological and conservation issues raised by colonization of northeastern North America by coyotes. BioScience 52:185-190.
- Gortazar, C., R. Villafuerte, J. C. Blanco, and D. Fernandez-De-Luco. 1998. Enzootic sarcoptic mange in red foxes in Spain. Zeitschrift fuer Jagdwissenschaft 44:251-256.
- Gosselink, T. E., T. R. Van Deelen, R. E. Warner, and M. G. Joselyn. 2003. Temporal habitat partitioning and spatial use of coyotes and red foxes in east-central Illinois. Journal of Wildlife Management 67:90-103.

- Gosselink, T. E., T. R. Van Deelen, R. E. Warner, and P. C. Mankin. 2007. Survival and cause-specific mortality of red foxes in agricultural and urban areas of Illinois. Journal of Wildlife Management 71:1862-1873.
- Grinder, M., and P. R. Krausman. 2001a. Morbidity-mortality factors and survival of an urban coyote population in Arizona. Journal of Wildlife Diseases 37:312-317.
- Grinder, M. I., and P. R. Krausman. 2001b. Home range, habitat use, and nocturnal activity of coyotes in an urban environment. The Journal of Wildlife Management 65:887-898.
- Grubbs, S. E., and P. R. Krausman. 2009. Use of urban landscape by coyotes. Southwestern Naturalist 54:1-12.
- Harrison, D. J. 1992. Dispersal characteristics of juvenile coyotes in Maine. Journal of Wildlife Management 56:128-138.
- Harrison, D. J., and J. A. Harrison. 1984. Foods of adult Maine coyotes and their knownaged pups. The Journal of Wildlife Management 48:922-926.
- Harrison, D. J., J. A. Harrison, and M. O'Donoghue. 1991. Predispersal movements of coyote (*Canis latrans*) pups in eastern Maine. Journal of Mammalogy 72:756-763.
- Heisey, D. M., and B. R. Patterson. 2006. A review of methods to estimate cause-specific mortality in presence of competing risks. The Journal of Wildlife Management 70:1544-1555.
- Hennessey, C. A. 2007. Mating strategies and pack structure of coyotes in an urban landscape: a genetic investigation. Thesis, The Ohio State University, Columbus, OH.
- Holzman, S., M. J. Conroy, and W. R. Davidson. 1992. Diseases, parasites and survival of coyotes in south-central Georgia. Journal of Wildlife Diseases 28:572-580.
- Iossa, G., C. D. Soulsbury, P. J. Baker, K. J. Edwards, and S. Harris. 2009. Behavioral changes associated with a population density decline in the facultatively social red fox. Behavioral Ecology 20:385-395.
- Iossa, G., C. D. Soulsbury, P. J. Baker, and S. Harris. 2008. Body mass, territory size, and life-history tactics in a socially monogamous canid, the red fox *Vulpes vulpes*. Journal of Mammalogy 89:1481-1490.
- Jimenez, M. D., E. E. Bangs, C. Sime, and V. J. Asher. 2010. Sarcoptic mange found in wolves in the rocky mountains in western United States. Journal of Wildlife Diseases 46:1120-1125.

- Johnson, D. H. 1980. The comparison of usage and availability measurements for evaluating resource preference. Ecology 61:65-71.
- Kamler, J. F., and P. S. Gipson. 2000. Space and habitat use by resident and transient coyotes. Canadian Journal of Zoology 78:2106-2111.
- _____. 2002. Sarcoptic mange on coyotes in northeastern Kansas. Prairie Naturalist 34:143-144.
- Kilgo, J. C., H. S. Ray, C. Ruth, and K. V. Miller. 2010. Can coyotes affect deer populations in southeastern North America? Journal of Wildlife Management 74:929-933.
- Krumm, C. E., M. M. Conner, N. T. Hobbs, D. O. Hunter, and M. W. Miller. 2009. Mountain lions prey selectively on prion-infected mule deer. Biology Letters 6:209-211.
- Krumm, C. E., M. M. Conner, and M. W. Miller. 2005. Relative vulnerability of chronic wasting disease infected mule deer to vehicle collisions. Journal of Wildlife Diseases 41:503-511.
- Krzystyniak, K., H. Tryphonas, and M. Fournier. 1995. Approaches to the evaluation of chemical-induced immunotoxicity. Environmental Health Perspectives 103:17-22.
- Lindstrom, E., and T. Morner. 1985. The spreading of sarcoptic mange among Swedish red foxes *Vulpes-vulpes* in relation to fox population dynamics. Revue d'Ecologie la Terre et la Vie 40:211-216.
- Lindstrom, E. R. 1994. Disease reveals the predator- sarcoptic mange, red fox predation, and prey populations. Ecology 75:1042-1049.
- Lukasik, V. M., and S. M. Alexander. 2011. Human-coyote intereactions in Calgary, Alberta. Human Dimensions of Wildlife 16:114-127.
- Manly, B. F. J., L. L. McDonald, D. L. Thomas, T. L. McDonald, and W. P. Erickson. 2010. Resource Selection by Animals. 2nd edition. Kluwer Academic Publishers, Dordrecht.
- May, R. M., and R. M. Anderson. 1978. Regulation and stability of host-parasite population interactions: II. destabilizing processes. Journal of Animal Ecology 47:249-267.
- McCarthy, J. S., D. J. Kemp, S. F. Walton, and B. J. Currie. 2004. Scabies: more than just an irritation. Postgraduate Medical Journal 80:382-387.

- McClennen, N., R. R. Wigglesworth, S. H. Anderson, and D. G. Wachob. 2001. The effect of suburban and agricultural development on the activity patterns of coyotes (*Canis latrans*). American Midland Naturalist 146:27-36.
- McKinney, M. L. 2002. Urbanization, biodiversity, and conservation. BioScience 52:883-890.
- _____. 2006. Urbanization as a major cause of biotic homogenization. Biological Conservation 127:247-260.
- Messier, F., and C. Barrette. 1982. The social system of the coyote (*Canis latrans*) in a forested habitat. Canadian Journal of Zoology 60:1743-1753.
- Miller, D. L., J. Schrecengost, A. Merrill, J. C. Kilgo, H. S. Ray, K. V. Miller, and C. A. Baldwin. 2009. Hematology, parasitology, and serology of free-ranging coyotes (*Canis latrans*) from South Carolina. Journal of Wildlife Diseases 45:863-869.
- Morey, P. S. 2004. Landscape use and diet of coyotes, *Canis latrans*, in the Chicago metropolitan area. Thesis, Utah State University, United States -- Utah.
- Morey, P. S., E. M. Gese, and S. Gehrt. 2007. Spatial and temporal variation in the diet of coyotes in the Chicago Metropolitan Area American Midland Naturalist 158:147-161.
- Morner, T. 1992. Sarcoptic mange in Swedish wildlife. Revue Scientifique et Technique (International Office of Epizootics) 11:1115-1121.
- Murray, D. L. 2006. On improving telemetry-based survival estimation. The Journal of Wildlife Management 70:1530-1543.
- Nakagawa, T., Y. Takai, M. Kubo, H. Sakai, T. Masegi, and T. Yanai. 2009. A pathological study of sepsis associated with sarcoptic mange in raccoon dogs (*Nyctereutes procyonoides*) in Japan. Journal of Comparative Pathology 141:177-181.
- Nelson, T. A., D. G. Gregory, and Laursen. 2003. Canine heartworms in coyotes in Illinois. Journal of Wildlife Diseases 39:593-599.
- Nelson, T. A., and D. M. Lloyd. 2005. Demographics and condition of coyotes in Illinois. American Midland Naturalist 153:418-427.
- Newman, T. J., P. J. Baker, and S. Harris. 2002. Nutritional condition and survival of red foxes with sarcoptic mange. Canadian Journal of Zoology 80:154-161.

- Olden, J. D., N. L. Poff, and M. L. McKinney. 2006. Forecasting faunal and floral homogenization associated with human population geography in North America. Biological Conservation 127:261-271.
- Oleckno, W. A. 2002. Essential Epidemiology: Principles and Applications. Waveland Press, Long Grove, IL.
- Overskaug, K. 1994. Behavioural changes in free-ranging red foxes (*Vulpes vulpes*) due to sarcoptic mange. Acta Veterinaria Scandinavica 35:457-459.
- Padgett, D. A., and R. Glaser. 2003. How stress influences the immune response. Trends in Immunology 24:444-448.
- Pence, D. B., and E. Ueckermann. 2002. Sarcoptic mange in wildlife. Revue Scientifique et Technique (International Office of Epizootics) 21:385-398.
- Pence, D. B., and L. A. Windberg. 1994. Impact of a sarcoptic mange epizootic on a coyote population. Journal of Wildlife Management 58:624-633.
- Pence, D. B., L. A. Windberg, B. C. Pence, and R. Sprowls. 1983. The epizootiology and pathology of sarcoptic mange in coyotes, *Canis latrans*, from south Texas. The Journal of Parasitology 69:1100-1115.
- Pollock, K. H., S. R. Winterstein, C. M. Bunck, and P. D. Curtis. 1989. Survival analysis in telemetry studies: the staggered entry design. The Journal of Wildlife Management 53:7-15.
- Prange, S., S. D. Gehrt, and E. P. Wiggers. 2003. Demographic factors contributing to high raccoon densities in urban landscapes. The Journal of Wildlife Management 67:324-333.
- Quinn, T. 1995. Using public sighting information to investigate coyote use of urban habitat. Journal of Wildlife Management 59:238-245.
- _____. 1997a. Coyote (*Canis latrans*) food habits in three urban habitat types of western Washington. Northwest Science 71:1-5.
- _____. 1997b. Coyote (*Canis latrans*) habitat selection in urban areas of western Washington via analysis of routine movements. Northwest Science 71:289-297.
- Riley, S. P. D., J. P. Pollinger, R. M. Sauvajot, E. C. York, C. Bromley, T. K. Fuller, and R. K. Wayne. 2006. A southern California freeway is a physical and social barrier to gene flow in carnivores. Molecular Ecology 15:1733-1741.
- Rosatte, R., K. Sobey, D. Donovan, L. Bruce, M. Allan, A. Silver, K. Bennett, M. Gibson, H. Simpson, C. Davies, A. Wandeler, and F. Muldoon. 2006. Behavior,

- movements, and demographics of rabid raccoons in Ontario, Canada: management implications. Journal of Wildlife Diseases 42:589-605.
- Ruiz, G., M. Rosenmann, F. F. Novoa, and P. Sabat. 2002. Hematological parameters and stress index in rufous-collared sparrows dwelling in urban environments. The Condor 104:162-166.
- Saalfeld, S. T., and S. S. Ditchkoff. 2007. Survival of neonatal white-tailed deer in an exurban population. Journal of Wildlife Management 71:940-944.
- Sacks, B. N., and K. M. Blejwas. 2000. Effects of canine heartworm (*Dirofilaria immitis*) on body condition and activity of free-ranging coyotes (*Canis latrans*). Canadian Journal of Zoology 78:1042-1051.
- Samuel, W. M. 1981. Attempted experimental transfer of sarcoptic mange (*Sarcoptes scabiei, Acarina: Sarcoptidae*) among red fox, coyote, wolf and dog. Journal of Wildlife Diseases 17:343-347.
- Sandercock, B. K., E. B. Nilsen, H. Broseth, and H. C. Pedersen. 2011. Is hunting mortality additive or compensatory to natural mortality? Effects of experimental harvest on the survival and cause-specific mortality of willow ptarmigan. Journal of Animal Ecology 80:244-258.
- Sargeant, G. B. 2011. Wild1-R Tools for Wildlife Research and Management. http://cran.r-project.org/web/packages/wild1 Accessed 3 November 2011.
- Scott, M. E., and A. Dobson. 1989. The role of parasites in regulating host abundance. Parasitology Today 5:176-183.
- Shargo, E. S. 1988. Home range, movements, and activity patterns of coyotes (*Canis latrans*) in Los Angeles suburbs. Dissertation, University of California, Los Angeles, Los Angeles, CA.
- Shelley, D. P., and T. M. Gehring. 2002. Behavioral modification of gray wolves, *Canis lupus*, suffering from sarcoptic mange: Importance of sequential monitoring. Canadian Field-Naturalist 116:648-650.
- Sikes, R. S., W. L. Gannon, D. S. Carroll, B. J. Danielson, J. W. Dragoo, M. R. Gannon, W. L. Gannon, D. W. Hale, C. McCain, D. K. Odell, L. E. Olson, S. Ressing, R. M. Timm, S. A. Trewhitt, and J. E. Whaley. 2011. Guidelines of the American Society of Mammalogists for the use of wild mammals in research. Journal of Mammalogy 92:235-253.
- Skerratt, L. F., R. Martin, and K. Handasyde. 1998. Sarcoptic mange in wombats. Australian Veterinary Journal 76:408-410.

- Skerratt, L. F., J. H. L. Skerratt, R. Martin, and K. Handasyde. 2004. The effects of sarcoptic mange on the behaviour of wild common wombats (*Vombatus ursinus*). Australian Journal of Zoology 52:331-339.
- Soulsbury, C. D., G. Iossa, P. J. Baker, N. C. Cole, S. M. Funk, and S. Harris. 2007. The impact of sarcoptic mange *Sarcoptes scabiei* on the British fox *Vulpes vulpes* population. Mammal Review 37:278-296.
- Timm, R. M., R. O. Baker, J. R. Bennett, and C. C. Coolahan. 2004. Coyote attacks: An increasing suburban problem. Proceedings of Vertebrate Pest Conference 21:67-88
- Todd, A. W., J. R. Gunson, and W. M. Samuel. 1980. Sarcoptic Mange: An important disease of coyotes and wolves of Alberta, Canada. Pages 706-729 in First Worldwide Furbearer Conference Proceedings, 3-11 August, 1980, Frostburg, Maryland, USA.
- Tompkins, D. M., and M. Begon. 1999. Parasites can regulate wildlife populations. Parasitology Today 15:311-313.
- Tompkins, D. M., A. P. Dobson, P. Arneberg, M. E. Begon, I. M. Cattadori, J. V.
 Greenman, J. A. P. Heesterbeek, P. J. Hudson, D. Newborn, A. Pugliese, A. P.
 Rizzoli, R. Rosa, F. Rosso, and K. Wilson. 2002. Parasites and host population dynamics. Pages 45-62 in P. J. Hudson, A. Rizzoli, B. T. Greenfell, H.
 Heesterbeek, and A. P. Dobson, editors. The Ecology of Wildlife Diseases. Oxford University Press, New York.
- Tompkins, D. M., A. M. Dunn, M. J. Smith, and S. Telfer. 2011. Wildlife diseases: from individuals to ecosystems. Journal of Animal Ecology 80:19-38.
- 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.
- United Nations Population Division. 2012. World Population Prospects: The 2011 Revision, CD-ROM Edition. Department of Economic and Social Affairs. New York, New York, USA.
- United States Census Bureau. 2010. < http://2010.census.gov/2010census/>. Accessed 11 December 2011.
- Van Deelen, T. R., and T. E. Gosselink. 2006. Coyote survival in a row-crop agricultural landscape. Canadian Journal of Zoology 84:1630-1636.
- Walton, S. F., and B. J. Currie. 2007. Problems in diagnosing scabies, a global disease in human and animal populations. Clinical Microbiology Reviews 20:268-279.

- Wang, Y., and D. K. Moskovits. 2001. Tracking fragmentation of natural communities and changes in land cover: applications of landsat data for conservation in an urban landscape (Chicago Wilderness). Conservation Biology 15:835-843.
- Way, J. G., I. M. Ortega, and E. G. Strauss. 2004. Movement and activity patterns of eastern coyotes in a coastal, suburban environment. Northeastern Naturalist 11:237-254.
- Way, J. G., and B. C. Timm. 2008. Nomadic behavior of an old and formerly territorial eastern coyote (*Canis latrans*). Canadian Field-Naturalist 122:316-322.
- White, G. C., and K. Burnham. 1999. Program MARK: survival estimation from populations of marked animals. Bird Study 46:S120-S139.
- White, L. A., and S. D. Gehrt. 2009. Coyote attacks on humans in the United States and Canada. Human Dimensions of Wildlife 14:419-432.
- Willingham, A. N. 2008. Emerging factors associated with the decline of a gray fox population and multi-scale land cover associations of mesopredators in the Chicago metropolitan area. Thesis, Ohio State University, Columbus, Ohio.
- Windberg, L. A., H. L. Anderson, and R. M. Engeman. 1985. Survival of coyotes in southern Texas. The Journal of Wildlife Management 49:301-307.
- Wydeven, A. P., S. R. Boles, R. N. Schultz, and C. J. T. Doolittle. 2003. Death of gray wolves, *Canis lupus*, in porcupine, *Erethizon dorsatum*, dens in Wisconsin. Canadian Field-Naturalist 117:469-471.
- Zahler, M. 1999. Molecular analyses suggest monospecificity of the genus *Sarcoptes* (Acari: Sarcoptidae). International Journal for Parasitology 29:759-766.