

DENSITY, DEMOGRAPHIC PATTERNS, POPULATION STRUCTURE, AND  
PATHOGEN EXPOSURE OF RACCOONS IN THE CHICAGO METROPOLITAN

AREA

A Thesis

Presented in Partial Fulfillment of the Requirements for  
the Degree of Master of Science in the  
Graduate School of The Ohio State University

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2008

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## ABSTRACT

Human populations and resultant urbanization have increased globally, often altering the abundance and distribution of wildlife species. Raccoons (*Procyon lotor*) are a common mesocarnivore, and benefit from urbanization, often attaining their highest densities in urban and suburban areas. Growing evidence suggests that human-induced alterations of the landscape influence the ecology and emergence of wildlife diseases, often with implications for the health and safety of humans and domestic animals. In light of the increasingly close association between wildlife and humans, the need for a better understanding of how human presence and land-use changes affect wildlife populations and wildlife-pathogen dynamics will be vital for the management and conservation of wildlife, and limiting the risk of human exposure to zoonotic diseases.

To better understand how land-use patterns influence raccoon density and demographic patterns, I sampled raccoons at multiple, replicated sites across an urban landscape; the Chicago metropolitan area. I predicted that raccoon density, population structure and demographic patterns (e.g. age structure, sex ratio, reproductive condition, and body condition) would be influenced by land-use. I sampled 18 sites (3 rural open, 8 urban open and 7 urbanized) during April-August, 2005-2006, and captured 530 adult, and 182 juvenile raccoons. Raccoon density varied substantially within land-use types.

Additionally, density differed by land-use type ( $F_{2,17} = 4.66$ ,  $P = 0.027$ ); mean density at urbanized sites was lower than urban open, and did not differ from rural open sites.

Pooled sex ratios did not differ from 1:1 (M:F) in urban open ( $P = 0.887$ ) or urbanized ( $P = 1.00$ ) sites, but did depart from 1:1 ( $P = 0.001$ ) in rural open sites. There was no association between site type and female reproductive condition ( $P = 0.959$ ).

There was no association between spring body condition index and site type for males ( $P = 0.382$ ) or females ( $P = 0.112$ ). I found no association between summer body condition index and site type for females ( $P = 0.201$ ), however there was a significant association for males ( $P = 0.007$ ). The overall age structure of raccoons differed among urban open, rural open, and urbanized sites. The most striking differences were the absence of the oldest age class (V) at urbanized sites and low numbers of age class I individuals at urban open sites.

I found that land-use influences the density of raccoons across this urban landscape, but the abundance of anthropogenic resources and subtle differences in habitat quality may be strongly influencing density at the site level. Furthermore, age structure was influenced by land-use and may have reflected differences in the prevalence and importance of different mortality sources across the landscape.

I tested raccoon sera collected across the Chicago metropolitan area for exposure to *Toxoplasma gondii* (*T. gondii*), *Leptospira spp.*, canine distemper virus (CDV), canine parvovirus (CPV), and pseudorabies virus (PRV). My objectives were to: 1) document patterns of exposure to infectious disease agents of public and animal health in raccoons across an urban landscape, 2) test for differences in the patterns of exposure to selected infectious disease agents for raccoons inhabiting different areas of the landscape (i.e.

urban open sites, rural open sites, and urbanized areas). Sera were collected from 570 raccoons, 302 from urban open sites, 135 from rural open sites and 133 from urbanized sites. Percentages of seropositive raccoons were 39% for CDV, 51% for CPV, 37% for *T. gondii*, 33% for *Leptospira spp.*, and 0% for exposure to PRV. I observed a positive correlation between age and seroprevalence for CDV, CPV, *T. gondii*, and *Leptospira spp.* Raccoons captured at rural open sites were more likely to be seropositive for CDV than raccoons from urban open sites (adjusted odds ratio {OR} = 2.39,  $P < 0.001$ ), and raccoons from urbanized sites were less likely to be seropositive than raccoons in urban open sites (adjusted OR = 0.57,  $P = 0.04$ ). Raccoons from urban open sites were more likely to be seropositive for *T. gondii* than raccoons from urbanized sites (adjusted OR = 2.70,  $P < 0.001$ ), similarly, raccoons from rural open sites were more likely to be seropositive than urbanized raccoons (adjusted OR = 2.81,  $P < 0.001$ ). Raccoons inhabiting all areas of this urban landscape were commonly exposed to the infectious agents we screened for, some of which pose risks to other wildlife species, humans, and to domestic and captive animal populations. Furthermore, the prevalence of exposure to certain infectious agents (CDV and *T. gondii*) differed across the landscape, therefore, the risks to humans and domestic species may be variable in different portions of the landscape. The relationship between changes in risk of exposure to infectious agents and land-use warrants further investigation due to the potential impacts on the health and safety of humans and domestic animals. My results indicate that landscape changes from urbanization can influence the relationships between land-use, host population dynamics, and host-pathogen relationships.

## ACKNOWLEDGEMENTS

I would like to thank my advisor, Stan Gehrt, for providing me the opportunity to work on such interesting projects as a technician and as a graduate student. I appreciate his support and guidance throughout my graduate research. I thank my committee members Paul Rodewald and Robert Gates for their willingness to provide their valuable advice and time. I would also like to thank Suzie Prange for all of her assistance with GIS and for her willingness to provide guidance and suggestions which greatly improved my work. I would like to acknowledge my funding sources: The National Science Foundation, The Max McGraw Wildlife Foundation, and Cook County Department of Animal and Rabies Control. I further wish to acknowledge The Ohio State School of Environment and Natural Resources and the Terrestrial Wildlife Ecology Lab for their support and the use of their resources. I thank Chris Anchor of the Cook County Forest Preserve District and Brad Woodsen of the McHenry County Conservation District for their assistance and access to county property. I am grateful to those who assisted me with field work including Christie Boser and Jason Isabelle. I am especially indebted to Stephanie Hauver for her dedication and hard work in assisting me to access field sites and trap raccoons in a difficult environment, while working on her own graduate project! I am grateful to all of my fellow TWEL graduate students and co-workers at McGraw for their friendship over the last several years. I wish to thank my father and stepmother, Bill

and Andrea for their love and support as I pursued my interests and education. I especially thank my fiancé, Christy, for her love, encouragement, and patience over the last two years. Finally, I would like to dedicate this thesis in memory of my mother, Tina, who knew I would be a wildlife biologist long before I did. Her example of kindness, love and strength has guided me through the ups and downs of graduate school.

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## CHAPTER 1

### INTRODUCTION

The raccoon is a medium sized mammal, and member of family Procyonidae. They are a highly successful and adaptable species whose range and density has increased markedly since the 1930's. The raccoon's range is estimated to have increased from 6.6 million km<sup>2</sup> to 8.8 million km<sup>2</sup> between 1920 and 1987 (Sanderson 1987). Raccoons are highly opportunistic and omnivorous, and they are also habitat generalists. These factors allow them to live in a variety of habitats throughout their range and to exploit resources in a manner that allows them to flourish even in highly altered, human dominated landscapes.

The human population of the United States has increased since the 1940's. Presently, over 5% of the total surface area of the U.S. is covered by urban and other built areas; and approximately 80% of the population lives in and around urban areas (USCB 2001). Urbanization has many impacts on the landscape ranging from conversion of natural habitats, fragmentation, and increased human presence, to altering the abundance and distribution of resources. Artificial resources are typically augmented by urbanization and can often be concentrated (Prange et al. 2004). The ability of the raccoon to survive in urban areas and exploit anthropogenic resources are important

factors allowing them to reach high densities in these areas (e.g., Prange et al. 2003 and Riley et al. 1998). Elevated population levels and concomitant increased levels of intraspecific contact can have important disease transmission implications, particularly when anthropogenic changes alter resource availability and distribution (Wright and Gompper 2005). Collinge et al. (2005) stated that landscape structure may affect infectious disease dynamics by altering the composition of ecological communities, which in turn alters important ecological interactions involved in pathogen transmission pathways. These authors noted that the influence of landscape structure on disease has been studied in relatively few systems.

Wildlife can have enormous impacts on the health and safety of humans and domestic species. Approximately 60% of recognized human pathogens are zoonotic and approximately 75% of emerging infectious diseases over the last 20 years have a wildlife source (Woolhouse 2002). Raccoons harbor numerous parasites and infectious diseases that are potentially hazardous to humans and domestic animals (e.g., rabies, leptospirosis, toxoplasmosis, raccoon roundworm [*Baylisascaris procyonis*], and canine distemper). Considering rabies alone in the U.S., in 1997, some 45,000 humans received post-exposure prophylaxis and approximately \$300,000,000 dollars were spent on control and prevention (Krebs et al. 1998).

Because raccoons are host to many infectious diseases and parasites and thrive in close association with humans, they are an ideal species with which to observe how landscape factors influence population ecology, demographics and exposure to diseases. Elucidating relationships between land-use and raccoon population density and demographics is crucial for guiding management decisions regarding wildlife populations



and public health and safety. Such knowledge would help modelers, managers and policy makers anticipate and manage the spread of diseases such as rabies. Despite these facts, there is a paucity of ecological knowledge regarding raccoons from multiple sites across urbanized systems. Much of the existing knowledge of urban raccoons comes from studies in urban parklands or suburban areas, and rarely are several sites from each habitat type studied across a landscape.

## LITERATURE REVIEW

### Distribution

The raccoon is a medium-sized carnivore and member of family Procyonidae. They are among the most recognizable mammalian species in North America and their distribution is widespread across North America, extending from Mexico into Canada (Gehrt 2003). The range of raccoons is estimated to have increased from 6.6 million km<sup>2</sup> to 8.8 million km<sup>2</sup> between 1920 and 1987, and it has been estimated that there were 15-30 times more raccoons in North America during the 1980's than there were in the 1930's (Sanderson 1987).

### Habitat and Shelter

Raccoons are habitat generalists, and occur in a wide variety of habitat types throughout their range. The most basic requirements of raccoons are a source of food, water, and protected areas for denning (Rosatte 2000). Habitats associated with water are commonly used by raccoons. During the summer raccoons used wetlands heavily in North Dakota (Fritzell 1978a). Greenwood (1982) reported that 53% of nocturnal locations were in wetlands, 19% in farmyards, 11% in cultivated areas, 7% in wooded

areas, 5% in upland grassland, and 5% on roads and trails in North Dakota. Urban (1970) reported that 87% of the average home range was marsh and 73% of all raccoon activity occurred in shallow water areas. Raccoons in this study also used spoil dikes considerably more than expected based on their availability.

Raccoons use woodlots heavily where they are available. Mature pine and hardwood habitats were selected by raccoons at all 3 spatial scales analyzed in central Mississippi, this selection of mature hardwoods was likely a function of quality foraging and denning opportunities, as well as the availability of free water (Chamberlain et al. 2003). Pedlar et al. (1997) observed raccoon habitat use at macrohabitat (1km) and microhabitat (10m) scales in Ontario, Canada, by using tracking stations. There was a positive relationship between raccoons and agricultural edge at the macrohabitat scale. The study also found a positive relationship between raccoon abundance and the amount of corn measured at the large scale. Results indicated that raccoon abundance in high intensity agricultural areas was tied to remnant wood lots.

Raccoons use a variety of types of shelter for their diurnal rest sites. Generally raccoons do not create their own dens; typically they will use structures that are already available (Gehrt 2003). Eighty-nine percent of raccoons used muskrat huts in a managed marsh in Ohio, but tree dens were heavily used in late fall and winter (Urban 1970). Raccoons in a suburban Ohio study site used a variety of den sites including ground dens, tree dens, abandoned buildings, and sewer systems (Hoffmann and Gottschang 1977). Raccoons commonly used tree dens and piles of logging slash for denning in central Mississippi (Chamberlain et al. 2003, Henner et al. 2004). Henner et al. (2004) observed that females used tree dens more than males, especially during young-rearing (spring and

summer) season, and males tended to utilize brush piles and ground dens more during the spring and summer. Excluding the young-rearing season, tree rest sites were used most frequently by both sexes. Henner et al. (2004) also suggested that raccoons selected den sites based on the availability of food and water resources. The variety of den and rest sites used by raccoons reflects their adaptable nature and enhances their ability to adapt to urban landscapes.

### Food Habits

Raccoons are highly omnivorous and opportunistic in their feeding habits, and have been the subject of many dietary studies. In North Dakota, during the waterfowl nesting season, plant foods were found in 84% of all raccoon scats and animal remains were found in 86% of all scats. Birds and bird eggs were found in 34% and 29% of scats, respectively, and mammals, crustaceans, snails and earthworms were also commonly encountered (Greenwood 1981). Yeager and Elder (1945) studied raccoon food habits at an Illinois goose refuge and found that birds (mostly geese) occurred in 20.7 and 87.9%, and plants occurred in 63.5 and 22.5% of scats pre- and post waterfowl hunting, respectively. They also found that insects were very important in the pre-hunting season, with crayfishes, snails, mussels and mammals also commonly being consumed.

Seemingly little research has been done on the food habits of urban raccoons. Fecal samples from raccoons in suburban Ohio contained seeds from 46 species of plants, insects, bird feathers, fur and skeletal remains of small mammals and evidence that raccoons were consuming human refuse (Hoffman and Gottschang 1977). The diversity of food types reported in these studies illustrates the omnivorous and adaptive feeding

habits of the raccoon and their ability to exploit seasonally and locally abundant resources.

### Population Demographics

Raccoon densities are affected by a wide range of factors including habitat quality, disease, predation and harvest by humans. In general, raccoon densities are thought to reflect habitat quality with respect to the distribution and abundance of resources (Gehrt 2003). Density estimates for rural populations vary substantially, ranging from 1.4/ km<sup>2</sup> (Mech et al 1968) to between 13.9 and 17.5/ km<sup>2</sup> (Urban 1970, Moore and Kennedy 1985). The lowest raccoon density was between 0.5 and 1.0/km<sup>2</sup> and occurred in an area of North Dakota site that was dominated by prairie and approached the northern extent of the raccoon's distribution (Fritzell 1978b). The highest densities occur in suburban and urban areas. For example, raccoon density at a suburban site in Ohio was 66.7/ km<sup>2</sup> (Hoffmann and Gottschang 1977), and in Illinois, at urban and suburban sites, densities ranged from 41.1 to 93/ km<sup>2</sup> (Prange et al. 2003). In the Toronto metropolitan area, raccoon densities ranged from 56/km<sup>2</sup> in forests and parks to 4/km<sup>2</sup> in fields (Rosatte et al. 1991). The highest density estimate from mark-recapture data was in an urban park where average raccoon density was 125/km<sup>2</sup> (Riley et al. 1998). Due to differences in methodology and study site characteristics, comparisons of density estimates should be made with caution, but in general raccoons exist at higher densities in urban and suburban areas than their rural counterparts.

Sex ratios of raccoon populations are typically even (Mech et al. 1968, Prange et al. 2003) or slightly male biased (Lotze and Anderson 1979, Moore and Kennedy 1985).

Sex ratios from trapping data are prone to be male biased due to trap bias towards males (Urban 1970, Gehrt and Fritzell 1996a). In south Texas, Gehrt and Fritzell (1996b) found the sex ratio to be female biased, as did Hoffmann and Gottschang (1977) in suburban Ohio. Most populations are likely to be slightly female biased or near parity, which is expected since the mating system of raccoons is on a continuum between polygyny and promiscuity (Gehrt 2003).

Typically, the age structure of raccoon populations are highly biased toward younger ages (<2 years old; Gehrt 2003). Urban (1970) reported a juvenile/adult ratio of 1.28:1 in an Ohio managed marsh, in a suburban Ohio site the ratio of juveniles to non-juveniles was 0.94:1 (Hoffman and Gottschang 1977). Road kill surveys showed a dramatic decrease in the percentage of raccoons that were >2 years old in rural, urban and suburban study sites in Illinois (Prange et al. 2003).

A variety of factors affect raccoon survival including: nutritional condition, disease, predation, vehicle collisions, hunting, sport trapping, and nuisance-related trapping. Raccoon survival can vary widely within and among areas and varies among seasons and years; generally survival during spring is lower than in other seasons.

Survival tended to be lowest in the spring season in urban, suburban and rural areas in Illinois (Prange et al. 2003). Mech et al. (1968) found that March was the most critical month for juvenile survival, during which 5 of 13 individuals perished.

Mortality factors vary in their importance among areas, most notably between urban/suburban and rural areas. Harvest can be a major mortality factor in rural populations. Mean annual survival was 0.74 for adult and yearling raccoons in rural Illinois where harvest mortality accounted for 74% and 48% of all known deaths for non-

radioed and radio-collared raccoons respectively (Mankin et al. 1999). Annual survival of adults ranged from 0.47 to 0.75 in rural Iowa, with harvest accounting for an average of 78% of deaths annually. Vehicle collision caused an average of 10% of deaths annually in the same area, making 88% of all mortality due to human-related factors (Clark et al. 1989). In Mississippi, annual survival for adults was 0.63 and 0.50 for males and females, respectively, with harvest and canine distemper being the primary mortality factors (Chamberlain et al. 1999). Average annual survival for adults was 0.84 in a protected south Texas population (Gehrt and Fritzell 1999). Prange et al. (2003) found that annual survival for adult female raccoons was higher at an urban site than at a rural site in 2 of 3 years; both sites were protected from harvest.

Throughout much of their range mating occurs in February and March, and gestation is approximately 63 days (Kaufmann 1982). Sanderson (1987) determined that the mean parturition date in Illinois was 18 April. The number of males and females in a litter are typically near parity at birth (Stains 1956). Mean litter size was 3.58 for adults and 3.66 for yearling raccoons in rural Illinois based on the placental scars of harvested raccoons; pregnancy rates for adults ( $\geq 95\%$  all years) were higher than the pregnancy rates of yearlings (range: 59-74%) in all years (Fritzell et al. 1985). Similar patterns were observed in Iowa, where the mean pregnancy rate for yearlings (59%) was lower than that of adults (91%), and mean litter size for yearlings was lower (3.1) than that of adults (3.8) (Clark et al. 1989). Mankin et al. (1999) also found evidence of age-specific reproduction in an Illinois population of raccoons with 46% of yearlings and 87% of adults showing evidence of breeding from 1990-1993. In a comparison of urban,

suburban and rural populations in Illinois the percentage of parous females did not differ among sites (Prange et al. 2003).

### Home Range

Many factors influence the size of raccoon home ranges, including habitat quality, the abundance and distribution of resources, and population density. Home range size varies geographically, among individuals and between sexes. In North Dakota, home range size ranged from 670 to 4,946 ha, and averaged 2,560 ha for males (Fritzell 1978a). Prange et al. (2004) observed female home range sizes ranging from 71.2-182.4 ha at the rural study site, whereas they ranged only from 25.2-52.8 ha at the urban study site. Home ranges were observed to be <129.5 ha in metropolitan Toronto (Rosatte 2000).

Difference in home range size between males and females has been commonly observed. Urban (1970) reported an average home range size of 48.4 ha, but noted significant variation among sexes and age classes. Gehrt and Fritzell (1997) found that seasonal home ranges of males ranged from 200-931 ha and females ranged from 14-535 ha, they further observed that median home range size for males was greater than that of females in all seasons each year. In central Mississippi, home range size differed between genders; males maintained larger home ranges ( $244 \pm 11$  ha) than females ( $153 \pm 13$  ha) (Chamberlain et al. 2003).

While some studies have observed seasonal differences in home range sizes, other studies observed no seasonal changes in home range size. Prange et al. (2004) found that at all sites (rural, urban and suburban), home range sizes varied by season, and were generally smallest in spring (although they did not estimate winter home ranges). They

further observed that raccoons on the suburban site exhibited the least pronounced changes in home range sizes. Glueck et al. (1988) observed a reduction in home range size during winter for an Iowa population of raccoons. Chamberlain et al. (2003) observed that home range sizes differed seasonally, with largest home ranges during the breeding season. Some researchers have observed that females maintained smaller home ranges during the young-rearing season, presumably due to movement restrictions associated with rearing young (Fritzell 1978a, Chamberlain et al. 2003). Gehrt and Fritzell (1997) reported that individual males maintained consistent home range size throughout the year; therefore, median size of home ranges for males did not vary seasonally. In the same population, however, individual females exhibited seasonal variation in size of home range, but medians did not change among seasons because fluctuations occurred at different times among individuals.

### Social Organization

Although raccoons have traditionally been considered to be solitary and/or asocial (Kaufmann 1982), the literature shows a spectrum of social organization in raccoons. In a low-density population, adult males maintained large areas relatively exclusive of other adult males, and were seldom located near each other (Fritzell 1978b). Despite extensive spatial overlap, no parous or pregnant female was located with another adult or yearling raccoon during spring and summer. The home range of each parous or pregnant female was generally located in the home range of a single adult male. Territoriality in adult males appeared to be a function of competition for access to females (Fritzell 1978b).



In south Texas, male raccoons frequently formed spatial groups, whose home ranges overlapped little with adjacent groups of males, which suggested territoriality among groups, but sociality among individuals within groups (Gehrt and Fritzell 1998a). Social groups of males were in areas with multiple females, while solitary males occupied areas devoid of females and rarely interacted with social groups. Gehrt and Fritzell (1998b) reported that related females exhibited extensive home range overlap, even as adults.

In central Mississippi, the home ranges and core areas of adult males frequently overlapped, and some males maintained spatial groups that overlapped little with solitary males or adjacent groups (Chamberlain and Leopold 2002). Instances of males that remained solitary and maintained exclusive home ranges were also observed. Adult females maintained exclusive home ranges during the winter. Several females were observed to share home ranges during other seasons, but these females did not den or forage together. Chamberlain and Leopold (2002) suggested that raccoon social behavior may vary within the same portion of the landscape.

The distribution of females appears to reflect the distribution of resources such as food, water, and den sites (Gehrt 2003). Due to the variety of conditions in which raccoons occur, they exhibit considerable plasticity in social structure, because it appears that male social behavior is determined by the spatial orientation of females and their densities (Gehrt 2003).

## Dispersal

Raccoons exhibit male-biased dispersal patterns. In North Dakota, the main dispersal period for yearling males ( $n = 21$ ) was May to June, the mean dispersal date was 5 June (Fritzell 1978a). One yearling male was killed 23.5 km from its last previous radiolocation after dispersing from the study area. Three dispersers from unknown natal locations had a mean distance of 16.4 km between their capture sites and their last known radiolocations. Dispersers likely traveled much farther than the documented distances, as aerial searches of up to 1,600 km<sup>2</sup> within a few days of dispersal were typically unsuccessful (Fritzell 1978a).

In a south Texas study, all radiocollared females ( $n = 14$ ) remained within their natal areas, and 12 of 13 radiocollared males dispersed or died before their second mating season (Gehrt and Fritzell 1998b). Eight of the males were known to have dispersed; median age at dispersal was 13.5 months, and 80% dispersed at 1 year or older. The average linear distance between initial location and final location was 9.7 km, with a range of 2.2 to 33 km (Gehrt and Fritzell 1998b). Three individuals perished during dispersal, and contact was lost with 3 others, therefore dispersal distances may have been underestimated (Gehrt and Fritzell 1998b).

## Raccoons in Urban Environments

For many species urbanization can be detrimental by altering ecosystems and consuming or degrading habitats. However, species that are generalists with flexible

dietary and habitat requirements often respond positively to anthropogenic resources (McKinney 2002). Food availability and distribution are often altered in urbanized systems due to the occurrence of abundant and at times highly concentrated anthropogenic resources (Prange et al. 2004). Prange and Gehrt (2004) studied changes in mesopredator community structure in response to urbanization and found that raccoons exhibited a stronger demographic response to urbanization than striped skunks (*Mephitis mephitis*) or Virginia opossums (*Didelphis virginiana*). The authors suggested that differences in intraspecific tolerance and the role of learning in foraging behaviors are possible reasons for the disparity in anthropogenic resource use.

Residing in urban and suburban areas affects the extent to which raccoons are subjected to various types of mortality factors, as well as other aspects of population demographics. Annual survival was high, except for juveniles, during a rabies epizootic in a Washington, D.C. parkland (Riley et al. 1998). Disease, vehicle collisions, and nuisance removal were the most common mortality factors in this study.

Prange et al. (2003) reported that urban raccoons experienced the fewest mortality sources, while raccoons at the rural site experienced the most. Disease was the dominant mortality factor at the urban site, while vehicle related mortalities were most common at the suburban and rural sites. Adult female survival was highest at the urban site, except for the third year of the study when an unknown disease caused significant mortality in the urban population. Density was greater at urban and suburban sites than rural for all seasons. Increased survival, higher annual recruitment and increased site fidelity were responsible for the high-density raccoon populations in urban areas (Prange et al. 2003).

Raccoons often reach high densities in urban and suburban areas (e.g., Hoffmann and Gottschang 1977, Riley et al. 1998, Prange et al. 2003), and home ranges are typically smaller in urban and suburban areas relative to rural sites (Prange et al. 2004). High densities of individuals that frequent aggregated food sites will likely increase transmission rates of diseases and parasites. Wright and Gompper (2005) experimentally manipulated resource availability in a population of raccoons to increase host contact rate and observed signs of altered parasite assemblages. Endoparasite infra-community richness in the experimental site doubled relative to the control site after resource supplementation. Individual hosts were more likely to have multispecies infections when contact rates increased and the infections involved a greater number of parasite species. Riley et al. (1998) observed that epizootic diseases such as canine distemper and rabies may be more easily transmitted in populations where individuals live in close association and noted that 6 of 8 mark-recapture studies in high-density raccoon populations cite disease as a major factor in population regulation (e.g., Schinner and Cauley 1974, Roscoe 1993). Riley et al. (1998) further speculated that there may be a tendency for disease to replace harvest by humans as a mortality factor in urbanized areas.

#### Diseases and Parasites

Raccoons are host to a wide variety of parasites and diseases. Raccoons have been observed to be host to no less than 10 species of fleas, and 14 species of ticks (Stains 1956). Birch et al. (1994) examined the gastrointestinal tracts of 60 raccoons for helminths and found 6 species, 4 of which were nematodes, including *Baylisascaris procyonis*. Raccoons can act as reservoirs of the spirocheteal agent of Lyme disease

(*Borella burgdorferi*), in southern New York, 55% of raccoons sampled produced spirochete-positive ticks (Fish and Daniels 1990).

In a study of raccoons in the southeastern U.S., Bigler et al. (1975) emphasized that serologic and microbiological studies have found that raccoons were exposed to at least 13 pathogens known to cause disease in humans. Serological surveys of raccoons from other areas have also found a variety of other pathogens, including *Toxoplasma gondii* (Hill et al. 1998, Mitchell et al. 1999), *Leptospira interrogans* (Mitchell et al. 1999), *Trypanosoma cruzi* (Yabsley and Noblet 2002), canine distemper (Mitchell et al. 1999), and pseudorabies (Mitchell et al. 1999).

#### Canine Distemper

Canine distemper virus (CDV) is an infectious, contagious viral disease and is among the most significant infectious disease of domestic and wild carnivores. CDV is a morbillivirus with a wide host-range that includes species in all families in the order Carnivora; however, its epidemiology is poorly understood for all but a few free-ranging species (Williams 2001). Transmission of CDV is primarily by aerosol or contact with oral, respiratory, and ocular fluids and exudates containing the virus (Williams 2001). Close association between affected and susceptible animals is required, as CDV is highly fragile in the environment. Dense populations are required to maintain epidemics of CDV, and epidemiology depends on factors such as the relative susceptibility of hosts, population density of sympatric susceptible hosts, and inter- and intraspecific behavior (Williams 2001).

Canine distemper has been studied in free-ranging raccoons. Roscoe (1993) identified 17 epizootics of CDV between September 1977 and March 1991 in New

Jersey. Epizootics occurred 3 times at 4-year intervals in 3 separate areas. Peak prevalence occurred at the end of the breeding season, with another period of CDV activity with increased activity of young raccoons in September (Roscoe 1993). Epizootics were associated with river drainages and other wetlands. Lethargy was the most commonly reported clinical sign in raccoons (Roscoe 1993). Hoff et al. (1974) described an epizootic of CDV in Florida raccoons and gray foxes (*Urocyon cinereoargenteus*) that persisted for 2 years, CDV antibodies were detected in 54.5% ( $n = 22$ ) of clinically ill and apparently healthy raccoons over this time. In rural Illinois 23% of raccoons from a farmed area and park were seropositive for canine distemper virus (Mitchell et al. 1999). In an urban Missouri population of raccoons 54.1% of raccoons were seropositive for canine distemper virus over a 6 year period (Junge et al. 2007). Schubert et al. (1998) reported similar antibody prevalence (49.2%) for raccoons in Ontario, Canada. In an experiment to test the effects of canine distemper on an urban raccoon population, there was no clear evidence of population effects, raccoon abundance appeared to be unaffected by CDV (Schubert et al. 1998). It was likely that some other factor was responsible for the observed patterns of raccoon abundance and that CDV mortality was only compensatory (Schubert et al. 1998). Canine distemper appears to be more important as a mortality factor and threat to certain highly susceptible species, such as the gray fox, and several threatened and endangered carnivores (Williams 2001).

## Parvovirus

Species from 6 families of the order Carnivora are susceptible to parvoviruses of the feline parvovirus subgroup, among them are Canidae, Felidae, Mustelidae, and Procyonidae (Barker and Parrish 2001). Transmission is thought to be through the fecal-

oral route, most likely through the ingestion of the virus from the environment rather than through direct contact (Reif 1976). Along with rabies and distemper, parvovirus infection is one of the most important infectious diseases of raccoons (Barker and Parrish 2001). Epidemics are likely to be small, dispersed, and will have only a transient local impact that is scattered unpredictably in space and time once parvovirus becomes endemic in a population of a susceptible species (Barker and Parrish 2001). Over a 5-year period in a high-density urban raccoon population in St. Louis 48.7% of raccoons tested positive for feline parvovirus (FPV) with seroprevalence ranging from 25-88.6% (Junge et al. 2007). Seventy-one percent of raccoon sera were positive for antibodies against feline panleukopenia virus (FPL) in 1985 in metropolitan Toronto (Rosatte et al. 1991).

#### Toxoplasmosis

*Toxoplasma gondii* is a protozoan parasite that is widespread in humans and other warm-blooded animals. Felids are the only host species that can excrete the oocysts of *T. gondii*. It is transmitted in 3 ways: congenitally, by ingestion of food and water contaminated with sporulated oocysts from infected cat feces, and by eating uncooked meat that contains tissue cysts (Dubey 1996).

Since raccoons are omnivorous and consume vegetation and carrion, they are good monitors for environmental *T. gondii* contamination; however, they do not shed *T. gondii* oocysts in their feces and are unlikely to serve as a source of infection (Mitchell et al. 1999). In Illinois, Mitchell et al. (1999) found 184 (49%) of 379 raccoons to be seropositive for *T. gondii*, with a significant difference in seroprevalence during spring (73%) compared to autumn (33%). Hill et al. (1998) found serologic evidence of

exposure to *T. gondii* in 111 of 809 raccoons (14%). Both Hill et al. (1998) and Mitchell et al. (1999) found a correlation between increasing age and seroprevalence of *T. gondii*. In Fairfax Virginia, 84.4% of 216 raccoons tested positive for exposure to *T. gondii* over a 2-year study with prevalence being 80.9% and 88.3% in the first and second year respectively (Hancock et al. 2005).

### Leptospirosis

Raccoons are a reservoir for the pathogen *Leptospira interrogans*. Bigler et al. (1975) suggested that raccoons can be used as serologic sentinels for leptospirosis and tularemia. In the southeastern United States, 10% of 333 raccoons were found to be seropositive for leptospirosis (Bigler et al. 1975). Mitchell et al. (1999) found 222 (48%) of 459 raccoons to be seropositive for *Leptospira interrogans*, with a vast majority being the *grippityphosa* serovar. Raccoons captured in the farm area were more likely to be seropositive than those from the park area, 52% and 43%, respectively (Mitchell et al. 1999). In an urban raccoon population inhabiting a zoological park in Missouri, serovars *icterohemorrhagiae* (8.9%) and *grippityphosa* (6.3%) were most commonly detected by serological testing (Junge et al. 2007).

Raccoons often spend time feeding and hunting in and around water, so it is likely that they can contaminate water sources when shedding *L. interrogans*. As a result, humans and pets can become exposed to the spirochete from drinking or recreational use of such waters.

### Pseudorabies

Pseudorabies (also called Aujeszky's disease) is caused by suid herpes virus 1, which is also called pseudorabies virus (PRV). Domestic and wild swine are the



reservoir hosts for PRV, however PRV can infect and cause disease in many wild vertebrate hosts including raccoons (Stallknecht and Howerth 2001). Most raccoons perished following experimental inoculation with pruritis and depression being the most commonly displayed clinical signs (Wright and Thawley 1980). It has been postulated that raccoons could potentially be a source of pseudorabies infection for domestic swine, but their possible role in the maintenance cycle is short-term and limited at best (Kirkpatrick et al. 1980). Although raccoons have been suggested to be involved in outbreaks of pseudorabies, it has never been reported in raccoons without concurrent infection in swine nearby (Thawley and Wright 1982). Mitchell et al. (1999) found 17% of 479 raccoons in their Illinois study area to be seropositive to PRV and they also reported that other studies have had much difficulty in identifying free-ranging raccoons with positive serum neutralizing titers. Furthermore, Mitchell et al. (1999) observed that several PRV seropositive free-ranging raccoons survived greater than 6 months, indicating that it may be possible for raccoons to maintain or spread PRV.

## RESEARCH RELEVANCE

Understanding the natural history of wildlife species and infectious pathogens in urban areas will become increasingly vital as wildlife and human contact increases due to urban expansion, human recreation, wildlife feeding and other anthropogenic effects. The changes in landscape composition and structure that result from urbanization have the potential to alter overall ecological community composition, host behavior and ecology, which could greatly impact pathogen transmission pathways and emergent patterns of infectious disease.

Although raccoon ecology has been studied in urban and suburban areas in the past, significant knowledge gaps still exist. Prior urban raccoon research was typically focused on parklands and suburban areas, with the most urbanized areas such as industrial and high-density residential areas largely being ignored. Additionally, these studies were typically conducted on single study sites. My research was conducted at multiple sites spanning the urban gradient more completely than previous research.

The behavior, food habits and adaptability of raccoons qualify it as a useful environmental monitor for exposure to numerous zoonotic diseases and other infectious agents (Bigler et al. 1975). The serological data from my project will provide useful baseline information on the seroprevalence of several important pathogens across an urbanized landscape. These data will further provide basic insight into the extent to which raccoons in an urban environment are exposed to pathogens of concern to human and animal health.

## THESIS ORGANIZATION

In Chapter 2, I will investigate the relationships between land-use patterns and the population ecology and demographic parameters of raccoons (e.g. density, age structure, sex ratio, and body condition). In Chapter 3, I will investigate the overall patterns of exposure to infectious disease agents of public and animal health significance (e.g. *Toxoplasma gondii*, *Leptospira* spp., canine distemper virus, parvovirus, and pseudorabies virus) in raccoons, as well as the relationships between land-use and pathogen exposure status.

## LITERATURE CITED

- Barker, I. K., and C. R. Parrish. 2001. Parvovirus infections Pp. 131-146 in Infectious diseases of wild mammals (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa.
- Bigler, W. J., J. H. Jenkins, P. M. Cumbie, G. L. Hoff, and E. C. Prather. 1975. Wildlife and environmental health: Raccoons as indicators of zoonoses and pollutants in southeastern United States. *Journal of the American Veterinary Medical Association* 167: 592-597.
- Birch, G. L., G. A. Feldhamer, and W. G. Dyer. 1994. Helminths of the gastrointestinal tract of raccoons in southern Illinois with management implications of *Baylisascaris procyonis* occurrence. *Transactions of the Illinois State Academy of Sciences* 87: 165-170.
- Chamberlain, M. J., K. M. Hodges, B. D. Leopold, and T. S. Wilson. 1999. Survival and cause-specific mortality of adult raccoons in central Mississippi. *Journal of Wildlife Management* 63: 880-888.
- Chamberlain, M. J., and B. D. Leopold. 2002. Spatio-temporal relationships among adult raccoons (*Procyon lotor*) in central Mississippi. *American Midland Naturalist* 148: 297-308.
- Chamberlain, M. J., L. M. Conner, B. D. Leopold, and K. M. Hodges. 2003. Space use and multi-scale habitat selection of adult raccoons in central Mississippi. *Journal of Wildlife Management* 67: 334-340.
- Clark, W. R., J. J. Hasbrouck, J. M. Kienzler, and T. F. Gluek. 1989. Vital statistics and harvest of an Iowa raccoon population. *Journal of Wildlife Management* 53: 982-990.
- Collinge, S. K., W. C. Johnson, C. Ray, R. Matchett, J. Grensten, J. F. Cully Jr., K. L. Gage, M. Y. Kosoy, J. E. Loye, and A. P. Martin. 2005. Landscape structure and plague occurrence in black-tailed prairie dogs on grasslands of the western USA. *Landscape Ecology* 20: 941-955.

- Dubey, J. P. 1996. Strategies to reduce transmission of *Toxoplasma gondii* to animals and humans. *Veterinary Parasitology* 64: 65-70.
- Fish, D., and T. J. Daniels. 1990. The role of medium-sized mammals as reservoirs of *Borrelia burgendorferi* in southern New York. *Journal of Wildlife Diseases* 26: 339-345.
- Fritzell, E. K. 1978a. Habitat use by prairie raccoons during the waterfowl breeding season. *Journal of Wildlife Management* 42: 118-127.
- Fritzell, E. K. 1978b. Aspects of raccoon (*Procyon lotor*) social organization. *Canadian Journal of Zoology* 56: 260-71.
- Fritzell, E. K., G. F. Hubert, B. E. Meyen, and G. C. Sanderson. 1985. Age-specific reproduction in Illinois and Missouri raccoons. *Journal of Wildlife Management* 49: 901-905.
- Gehrt, S. D. 2003. Raccoons and allies. In *Wild mammals of North America: biology, management, and conservation* (G. A. Feldhamer, B. C. Thompson, and J.A. Chapman, eds.). 2nd ed. Johns Hopkins University Press, Baltimore, Maryland, pp. 611-633.
- Gehrt, S. D., and E. K. Fritzell. 1996a. Second estrus and late litters in raccoons. *Journal of Mammalogy* 77: 388-393.
- Gehrt, S. D., and E. K. Fritzell. 1996b. Sex-biased response of raccoons (*Procyon lotor*) to live traps. *The American Midland Naturalist* 135: 23-32.
- Gehrt, S. D., and E. K. Fritzell. 1997. Sexual differences in home ranges of raccoons. *Journal of Mammalogy* 78: 921-931.
- Gehrt, S. D., and E. K. Fritzell. 1998a. Resource distribution, female home range dispersion and male spatial interactions: group structure in a solitary carnivore. *Animal Behavior* 55: 1211-1227.
- Gehrt, S. D., and E. K. Fritzell. 1998b. Duration of familial bonds and dispersal patterns for raccoons in south Texas. *Journal of Mammalogy* 79: 859-872.
- Gehrt, S. D., and E. K. Fritzell. 1999. Survivorship of a nonharvested raccoon population in south Texas. *Journal of Wildlife Management* 63: 889-894.
- Glueck, T. F., W.R. Clark, and R.D. Andrews. 1988. Raccoon movement and habitat use during the fur harvest season. *Wildlife Society Bulletin* 16: 6-11.

- Greenwood, R. J. 1981. Foods of prairie raccoons during the waterfowl nesting season. *Journal of Wildlife Management* 45: 754-760.
- Greenwood, R. J. 1982. Nocturnal activity and foraging of prairie raccoons (*Procyon lotor*) in North Dakota. *The American Midland Naturalist* 107: 238-243.
- Hancock, K., L. A. Thiele, A. M. Zajac, F. Elvinger, and D. S. Lindsay. 2005. Prevalence of antibodies to *Toxoplasma gondi* in raccoons (*Procyon lotor*) from an urban area of Northern Virginia. *Journal of Parasitology* 91: 694-695.
- Henner, C. M., M. J. Chamberlain, B. D. Leopold, and W. Burger Jr. 2004. A multi-resolution assessment of raccoon den selection. *Journal of Wildlife Management* 68: 179-187.
- Hill, R. E., J. E. Zimmerman, R. W. Willis, S. Patton, and W. R. Clark. 1998. Seroprevalence of antibodies against *Toxoplasma gondii* in free ranging mammals in Iowa. *Journal of Wildlife Diseases* 34: 811-815.
- Hoff, G. L., W. J. Bigler, S. J. Proctor, and L. P. Stallings. 1974. Epizootic of canine distemper virus infection among urban raccoons and gray foxes. *Journal of Wildlife Diseases* 10: 423-428.
- Hoffman, C. O., and J. L. Gottschang. 1977. Numbers, distribution, and movements of a raccoon population in a suburban residential community. *Journal of Mammalogy* 58: 623-636.
- Junge, R. E., K. Bauman, M. King, and M. E. Gompper. 2007. A serologic assessment of exposure to viral pathogens and *Leptospira* in an urban raccoon (*Procyon lotor*) population inhabiting a large zoological park. *Journal of Zoo and Wildlife Medicine* 38: 18-26.
- Kaufmann, J. H. 1982. Raccoons and allies. In *Wild Mammals of North America: biology, management, and economics* (J. A. Chapman and G. A. Feldhamer, eds.). Johns Hopkins University Press, Baltimore, Maryland, USA, pp. 567-585.
- Kirkpatrick, C. M., C. L. Kanitz, and S. M. McCrocklin. 1980. Possible role of wild mammals in the transmission of pseudorabies to swine. *Journal of Wildlife Diseases* 16: 601-614.
- Krebs, J. W., S. C. Long-Marin, and J. E. Childs. 1998. Causes, costs, and estimates of rabies postexposure prophylaxis treatment in the United States. *Journal of Public Health Management and Practice* 4: 56-62.
- Lotze, J., and S. Anderson. 1979. *Procyon lotor*. *Mammalian Species* 119: 1-8.

- Mankin, P. C., C. M. Nixon, J. B. Sullivan, T. L. Esker, R. G. Koerkenmeier and L. L. Hungerford. 1999. Raccoon (*Procyon lotor*) survival in west-central Illinois. *Transactions of the Illinois State Academy of Science* 92: 247-256.
- McKinney, M. L. 2002. Urbanization, biodiversity, and conservation. *Bioscience* 52: 883-890.
- Mech, L. D., D. M. Barnes, and J. R. Tester. 1968. Seasonal weight changes, mortality, and population structure of raccoons in Minnesota. *Journal of Mammalogy* 49: 63-73.
- Mitchell, M. A., L. L. Hungerford, C. Nixon, T. Esker, J. Sullivan, R. Koerkenmeier, and J. P. Dubey. 1999. Serologic survey for selected infectious disease agents in raccoons from Illinois. *Journal of Wildlife Diseases* 35: 347-355.
- Moore, D. W., M. L. Kennedy. 1985. Weight changes and population structure of raccoons in western Tennessee. *Journal of Wildlife Management* 49: 906-909.
- Pedlar, J. H., L. Fahrig, and H. G. Merriam. 1997. Raccoon habitat use at 2 spatial scales. *Journal of Wildlife Management* 61: 102-112.
- Prange, S., and S. D. Gehrt. 2004. Changes in mesopredator-community structure in response to urbanization. *Canadian Journal of Zoology* 82: 1804-1817.
- Prange, S., S. D. Gehrt, and E. P. Wiggers. 2003. Demographic factors contributing to high raccoon densities in urban landscapes. *Journal of Wildlife Management* 67: 324-333.
- Prange, S., S. D. Gehrt, and E. P. Wiggers. 2004. Influences of anthropogenic resources on raccoon (*Procyon lotor*) movements and spatial distribution. *Journal of Mammalogy* 85: 483-490.
- Reif, J. S. 1976. Seasonality, natality and herd immunity in feline panleukopenia. *American Journal of Epidemiology* 103: 81-87.
- Riley, S. P. D., J. Hadidian, and D. A. Manski. 1998. Population density, survival, and rabies in raccoons in an urban national park. *Canadian Journal of Zoology* 76: 1153-1164.
- Rosatte, R. C. 2000. Management of raccoons (*Procyon lotor*) in Ontario, Canada: Do human intervention and disease have significant impact on raccoon populations? *Mammalia* 64: 369-390.
- Rosatte, R. C., M. J. Power, and C. D. MacInnes. 1991. Ecology of urban skunks, raccoons and foxes in metropolitan Toronto. In *Wildlife conservation in*

- metropolitan environments. Edited by L. W. Adams and D. L. Leedy. National Institute for Urban Wildlife, Columbia, Maryland, pp. 31-38.
- Roscoe, D. E. 1993. Epizootiology of canine distemper in New Jersey raccoons. *Journal of Wildlife Diseases* 29: 390-395.
- Sanderson, G. C. 1987. Raccoon. In M. Novak, J.A. Baker, M. E. Obbard, and B. Malloch, eds. *Wild furbearer management and conservation in North America*. Ontario Trappers Association, North Bay, Canada, pp.487-499.
- Schinner, J. R., and Cauley, D. L. 1974. The ecology of urban raccoons in Cincinnati, Ohio. In *Wildlife in an urbanizing environment*. Edited by J. H. Noyes and D. R. Progulske. Planning and Resource Development Series No. 28, Holdsworth Natural Resources Center, Amherst, Massachusetts, pp. 125-130.
- Schubert, C. A., I. A. Barker, R. C. Rosatte, C. D. MacInnes, and T. D. Nudds. 1998. Effect of canine distemper on an urban raccoon population: An experiment. *Ecological Applications* 82: 379-387.
- Stains, H. J. 1956. *The raccoon in Kansas, natural history, management, and economic importance*. University of Kansas, Lawrence, Kansas.
- Stallnecht, D. E., and E. W. Howerth. 2001. Pseudorabies (Aujeszky's Disease) Pp. 164-170 in *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa.
- Thawley, D. G., and J. C. Wright. 1982. Pseudorabies virus infection in raccoons: a review. *Journal of Wildlife Diseases* 18: 113-116.
- Urban, D. 1970. Raccoon populations, movement patterns, and predation on a managed waterfowl marsh. *Journal of Wildlife Management* 34: 372-282.
- U.S. Census Bureau. 2001. *Statistical Abstract of the United States: 2001*. One hundred and twelfth edition. U.S. Bureau of the Census, Washington D.C., USA.
- Williams, E. S. 2001. Canine distemper Pp. 50-59 in *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa.
- Woolhouse, M.E. 2002. Population biology of emerging and re-emerging pathogens. *Trends in Microbiology*. 10 (10 Suppl.) S3-7.
- Wright, A. N., and M. E. Gompper. 2005. Altered parasite assemblages in raccoons in response to manipulated resource availability. *Oecologia* 144: 148-156.

Wright, J. C., and D. G. Thawley. 1980. Role of the raccoon in the transmission of psuedorabies: a field and laboratory investigation. *American Journal of Veterinary Research* 41: 581-583.

Yabsley, M. J., and G. P. Noblet. 2002. Seroprevalence of *Trypanosoma cruzi* in raccoons from South Carolina and Georgia. *Journal of Wildlife Diseases* 38: 75-83.

Yeager, L. E., W. H. Elder. 1945. Pre-and post-hunting season foods of raccoons on an Illinois goose refuge. *Journal of Wildlife Management* 9: 48-56.



## CHAPTER 2

### DENSITY, DEMOGRAPHIC PATTERNS AND POPULATION STRUCTURE OF RACCOONS IN AN URBANIZED LANDSCAPE

#### INTRODUCTION

Rising human population growth in the United States and abroad, has resulted in increased levels of urbanization nationally and globally. Urbanization can have a range of effects on the distribution and abundance of wildlife species, with some species becoming overabundant. Human perceptions of wildlife are often dynamic and can be altered by increases in wildlife abundance and subsequent nuisance problems (DeStephano and Deblinger 2005). In light of the increasingly close association between wildlife and humans, the need for a better understanding of how human presence and land-use changes affect wildlife populations will become increasingly vital as wildlife managers strive to balance the needs of humans and wildlife.

There has been a dramatic shift to urban living in the last century; 50% of the global population now resides in urban areas relative to 10% in 1900 (Grimm et al. 2008). Much of the future increase in global population is expected to be in the cities of developing nations, many of which may grow to unprecedented sizes. The U.S. population increased 24% from 1980 to 2000, resulting in a 34% increase in land

occupied by urban and built-up areas (USCB 2001). Urban land-use is projected to increase 79% by 2025, as a result, the percentage of the land base devoted to developed uses will rise from 5.2 to 9.2% (Alig et al. 2004).

Urbanization often results in the permanent loss of habitat and further influences remaining natural areas through increased fragmentation and isolation, and alteration of the matrix in which they are embedded (Wang and Moskovits 2001). Fragmentation and isolation can have a range of effects on the composition of animal communities. The distribution and abundance of mammalian carnivores can be highly influenced by fragmentation and isolation. Some species are highly sensitive to fragmentation (e.g. mountain lions [*Puma concolor*]), certain species can be enhanced (e.g. opossums [*Didelphis virginiana*]) and others appear to be relatively unaffected by fragmentation or isolation (e.g. raccoons [*Procyon lotor*] and striped skunks [*Mephitis mephitis*]; Crooks 2002). By altering the species composition of communities, urbanization can be a major source of biological homogenization on a global scale whereby some “urban-adapted” species become common in cities globally and a subset of native species become locally or regionally abundant (McKinney 2006). Although many species are negatively impacted by urbanization, generalist species with flexible dietary and habitat requirements can respond positively to urbanization and the anthropogenic resources available in urban environments (McKinney 2002).

The raccoon benefits from urbanization. High densities of raccoons are often found in urban (Rosatte et al. 1991, Riley et al. 1998, Prange et al. 2003) and suburban areas (Schinner and Cauley 1974, Hoffman and Gottschang 1977) relative to their rural counterparts (Fritzell 1978, Mech et al. 1968). Raccoons appear to exhibit a stronger

demographic response to urbanization than opossums or skunks, suggesting that they exploit anthropogenic resources more efficiently than other mesocarnivores (Prange and Gehrt 2004). Their efficient use of anthropogenic resources is further evidenced by the fact that urban raccoons appear to shift foraging strategies to focus on anthropogenic resources (Bozek et al. 2007). Increased survival, higher annual recruitment and increased site fidelity are among other factors responsible for high-density raccoon populations in urban areas (Prange et al. 2003). Although high raccoon densities are often reported in urban and suburban sites, raccoon densities generally appear to reflect habitat quality with respect to the distribution and abundance of resources (Gehrt 2003).

Habitat-specific demography is seldom documented in free ranging wildlife populations, but has been observed for the pika (*Ochotona princeps*; Kruezer and Huntly 2003) and the black-throated blue warbler (*Dendroica caerulescens*; Holmes et al. 1996). Habitat-specific demographic patterns can be reflected in population structure, and may be indicative of differences in habitat quality and mortality patterns in an urban landscape. The demographic patterns of raccoons in urban landscapes are likely to be influenced by anthropogenic resources and human related mortality sources. It has been postulated that anthropogenic resources lead to better physical condition and consequently higher reproductive rates (Rosatte et al. 1991). Although anthropogenic resources can be beneficial, there are numerous potential mortality sources in urbanized areas including: vehicle collisions, disease, euthanasia by animal control agents, and attacks by dogs (Rosatte et al. 1991, Riley et al. 1998). Due to the greater number of potential mortality sources, urbanized sites may have younger age-structure than protected urban and rural open areas. If mortality rates are extremely high in the most

urbanized areas, altered sex ratios may be present due to the male biased-dispersal patterns of raccoons (Fritzell 1978, Gehrt and Fritzell 1998), which could indicate that these areas represent “sink” populations.

In addition to being abundant in urban and suburban areas, raccoons are known to harbor numerous parasites and diseases of importance to the health and safety of humans and domestic species ranging from raccoon roundworm (*Baylisascaris procyonis*) and rabies to canine distemper and leptospirosis. Raccoon roundworm can cause blindness and death in humans and can be common in free-ranging raccoons (Jacobson et al. 1982). Raccoon rabies is a disease of great importance from a human health, economic and disease management standpoint. Rabies prevention and post-exposure treatment are a costly endeavor with an estimated expense between \$230 million and \$1 billion annually (Fishbein and Robinson 1993). Managers have relied on oral rabies vaccination (ORV) programs across the eastern USA to prevent the westward spread of the mid-Atlantic epizootic of raccoon rabies. Knowledge of host distribution, density and demographic parameters across the landscape is vital to developing useful models of disease dynamics with which to plan and evaluate control methods, as was exemplified by the modeling of fox rabies in Europe (Dobson 2000).

Because increasing urbanization will likely place more humans and domestic animals in close association with raccoon populations, the need for knowledge of raccoon ecology in urban and suburban areas will become increasingly vital. Although certain demographic parameters have been found to differ for raccoons in urban, suburban and rural open areas, these patterns have not been investigated within the most developed areas of the urban matrix. Much of the existing research of urban and suburban raccoons

has been restricted to relatively few study sites and has generally been conducted in remnant fragments of natural habitat (Hoffman and Gottschang 1977, Riley et al. 1998, Prange et al. 2003). Additionally, previous research has been carried out using a variety of methods, making comparisons among studies problematic.

To better understand how raccoon populations respond to urbanization, additional research is needed where sites are sampled across an urban landscape with replication. Therefore, the objective of this study was to gain insight as to how land-use patterns influence raccoon density, demographic patterns and population structure across an urban landscape. I predicted that raccoon density, demographic patterns and population structure would be strongly influenced by land-use. I investigated the following research questions related to my overall objective: 1) Are raccoon densities higher in urban open (i.e. protected urban parklands and undeveloped areas consisting primarily of natural vegetation communities), urbanized (i.e. areas dominated by residential land-uses) or rural open sites (i.e. protected parklands in rural surroundings)? 2) What land-use features best predict raccoon density across an urban landscape? 3) Are there differences in the sex ratio of raccoon subpopulations in different land-use types? 4) Is there a tendency toward increasing male-bias with increasing levels of urban land-use? 5) Is there an association between site type and the reproductive condition of female raccoons? 6) Are raccoons from urban open sites in better physical condition (i.e. higher body condition index) than raccoons from rural open and urbanized sites? 7) Do urban open, urbanized and rural open sites have differing age structures?

## METHODS

### Study Area

#### The Chicago Metropolitan Area

The city of Chicago and its surrounding suburbs, collectively referred to as the Chicago metropolitan area, have grown substantially in recent decades. The Chicago-Naperville-Joliet, IL-IN-WI Metropolitan Statistical Area (MSA) contains 14 counties from southeastern Wisconsin to northwest Indiana and had an estimated population of 9,443,356 in 2005, making it the 3<sup>rd</sup> largest MSA in the United States (USCB 2006a). Within this vast urbanized area exists approximately 810 km<sup>2</sup> of protected natural areas in county preserves, state parks, federal preserves and privately owned areas, known as the Chicago Wilderness (Wang and Moskovits 2001). The Illinois portion of the Chicago metropolitan area is generally considered to consist of the following 6 counties: Cook, DuPage, Kane, Lake, McHenry and Will, which had an estimated population of 8,092,145 in 2000 (USCB 2001). The Northeastern Illinois Planning Commission (NIPC) has forecasted that the population of this area will exceed 10,000,000 by 2030, attaining the status of megacity (NIPC 2006).

Local climate is mainly continental; however, conditions are strongly influenced by Lake Michigan. At the O'Hare International Airport weather station, mean annual temperature for 1971-2000 was 9.5°C, with mean temperatures for February and July

being  $-2.8^{\circ}$  and  $22.9^{\circ}\text{C}$  respectively. Mean precipitation over this period was 92.13cm (NOAA 2002).

### Cook County

Cook County is located in northeastern Illinois, has 2,449.30 km<sup>2</sup> of land area, is home to the city of Chicago, and had an estimated population of 5,288,265 and overall human density of 2159.09/ km<sup>2</sup> in 2006 (USCB 2006b; Figure 2.1). Population change in Cook County was greatest from 1910-1970 when there was a 128% increase; after the 1970 census the population declined slightly (USCB 1995). The Chicago Metropolitan Agency for Planning (CMAP) estimated that approximately 71% of the county was occupied by urban land-uses and 15% of the county was open space in 2001 (CMAP 2006). The Cook County Forest Preserve District (CCFPD) owned and managed 271.14 km<sup>2</sup> of open land, or approximately 11% of the county's land mass as of 2007 (Chris Anchor, CCFPD, pers. comm.).

### McHenry County

McHenry County is located in northeastern Illinois approximately 50 km from the city of Chicago and is 1582 km<sup>2</sup> (Figure 2.1). Although historically a predominantly agricultural county, McHenry County has recently experienced marked urbanization. Between 1960 and 1990 the county's population increased 117.6% (USCB 1995), and there was a 70% increase between 1990 and 2006 when overall population was estimated to be 312,373 and population density was 199.84/km<sup>2</sup> (USCB 2006b). Urbanization is expected to continue, and the population of McHenry County is expected to reach 457,594 by 2030 (NIPC 2006). Urban and built up areas occupied approximately 21% of McHenry County as of 2001, and roughly 50% of the county was in agricultural use

(CMAP 2006). The McHenry County Conservation District owns 87 km<sup>2</sup> of woodlands, wetlands, prairies and savannahs within the county.

### Habitat Data and Site Selection

I used the land cover mapping and classification data compiled by the Illinois Gap Analysis Project (IL-GAP) to determine land cover composition, select sites and perform data analyses. I made layers of Cook County and McHenry County using a geographic information system (GIS) and the IL-GAP coverage data, and overlaid a grid with 4km<sup>2</sup> cells onto Cook County. Land cover classifications were simplified for the purposes of this research by consolidating several classifications used by IL-GAP to form 6 basic land cover classifications: open space (urban or rural parklands and undeveloped areas consisting primarily of natural vegetation communities), open water, high-density urban (impervious surfaces account for 80-100% of total cover, typically industrial and commercial land-uses), medium-density urban (50-80% impervious surfaces, typically single-family housing), low-density urban (20-50% impervious surfaces, typically single-family housing) and agriculture. Percent land cover for each of the classes was calculated for each grid cell using Microsoft Access. Cells were classified as being either urban open space, rural open space, or high, medium or low-density urban land-use, dependent on the primary land-use type in the cell. I selected live-trapping grid cells in Cook County using a stratified random sampling method based on the IL-GAP land cover data. If a site could not be accessed or was adjacent to a previously selected cell, a new cell was randomly selected. Additionally, 3 sites were selected in protected open spaces



in rural areas of McHenry County, due to the paucity of agricultural areas in Cook County.

### Capture and Handling

I live-trapped raccoons using single-door Tomahawk box traps (Tomahawk Live Trap Co., Tomahawk, Wisconsin, USA) baited with commercial cat food. Trapping was conducted during spring and summer (March-August), 2005-2006. During each trapping session 30 traps were placed within a 2.4km<sup>2</sup> grid composed of a 3 X 5 array of 400m X 400m cells, with 2 traps placed in each cell and maintained for 5-10 nights (Gehrt 2002). Captured raccoons were immobilized using an intramuscular injection of Telazol® (Fort Dodge Animal Health, Fort Dodge, Iowa; Gehrt et al. 2001). Immobilized raccoons were weighed, sexed, and placed into age classes (class I: 0-14 months, class II: 15-38 months, class III: 39-57 months, class IV: 58-86 months, class V: >86 months) based on patterns of tooth wear (Grau et al. 1970). Reproductive condition was determined by observing the length and coloration of teats for females and the size of testes in males (Sanderson and Nalbandov 1973). All raccoons and opossums were marked with 2 uniquely numbered ear tags (Monel #3, National Band and Tag Company, Newport, Kentucky, USA). The Ohio State University Animal Care and Use Committee approved the above capture and handling methods (ILACUC#2003R0062).

### Abundance and Density Estimation

I computed abundance estimates using the Huggins closed capture-recapture conditional likelihood modeling procedure (Huggins 1989, 1991) implemented in program MARK (White and Burnham 1999). The Huggins estimator includes

parameters for initial capture probability ( $p$ ) and recapture probability ( $c$ ), however, population size ( $N$ ) is not included in the likelihood function and is calculated as a derived parameter based on the number of unique individuals captured and  $p$  (Finley et al. 2005). The Huggins procedure allows for the incorporation of covariates (e.g. sex, age), which can account for sources of heterogeneity (Huggins 1991). The use of group-specific, time-specific, and individual-specific covariates provides additional information with which to improve estimates of detection probability (White 2005).

I modeled the combined data from all 18 of the grid cells to obtain a parsimonious estimate of  $p$ , however each grid cell was treated as a distinct attribute group for which an estimate of  $N$  was generated (Finley et al. 2005, White 2005). I considered models that assumed the same  $p$  and  $c$  probabilities across grid cells. In this set of models, effects on  $p$  were modeled by the sex and age of the raccoon captured, year of capture, occasion-specific detection probabilities and behavioral response to initial capture (i.e. inclusion of the recapture parameter  $c$  in the model). Additionally, models where  $p$  and  $c$  were invariant across trapping occasion were considered.

I employed an information-theoretic model-selection approach, using a bias corrected version of Akaike's information criterion ( $AIC_c$ ; Hurvich and Tsai 1989, Burnham and Anderson 2002) to rank candidate models, and calculated the weight associated with each model. To account for model selection uncertainty, I calculated model-averaged abundance estimates for each site from the entire model set based on Akaike weights (Burnham and Anderson 2002, Converse et al. 2006).

Density estimates (raccoons/km<sup>2</sup>) were calculated using the model-averaged abundance estimates from each site. Rather than computing a naïve density estimate

based on the 2.4 km<sup>2</sup> trapping grid area, I calculated density based on the overall 4km<sup>2</sup> grid cell, because animals likely moved beyond the artificial boundaries of the trapping grid.

### Data analysis

I evaluated differences in raccoon densities among land-use types using a one-way analysis of variance (ANOVA). I used a Tukey-Kramer multiple comparison test to detect pairwise differences between mean densities for each site type ( $\alpha = 0.05$ ) when ANOVA results were significant. To determine which land-use features best predicted raccoon density, I used multiple linear regression analysis. The land-use composition values for each cell were used as predictor variables. To avoid multicollinearity, I tested for correlation among the predictor variables and did not use predictor variables in the same model if they were highly correlated ( $r \geq 0.70$ ). An information-theoretic approach, utilizing AIC<sub>c</sub> was used for model-selection of a “best model” from an *a priori* set of biologically reasonable candidate models (Burnham and Anderson 2002).

I tested for overall departure from 1:1 (M:F) sex ratio in each dominant land-use type (e.g. urban open, urbanized) using chi-square goodness of fit tests. Additionally, I tested the prediction of increasing male-bias with increasing levels of urban land-use using a linear regression, with the natural log transformed proportion of males at each site as the response variable and percent urban land use as the predictor variable. I determined whether the reproductive condition of females was associated with land-use type using a chi-square test. Females were considered to be reproductive if they were currently lactating, fetuses could be palpated or if juveniles were observed in the trap. I

calculated a body condition index (BCI) by regressing the natural log transformed body mass (kg) of each raccoon against natural log transformed body length (mm), the residuals were used as the BCI (Jakob et al. 1996). A residual above the regression line indicated that a raccoon was in “good” body condition and a residual below the regression line indicated “poor” body condition. Male raccoons are larger than females in body measurements and weight (Gehrt 2003) and seasonal weights can vary substantially (Mech et al. 1968, Moore and Kennedy 1985), therefore I calculated BCI separately for males and females in spring and summer. BCI was only calculated for individuals captured in 2006. To assess association between BCI and site type I used chi-square tests of contingency tables. I compared BCIs of males and females at rural open and urbanized sites during spring, and urban open and urbanized sites during summer.

I utilized a polytomous logistic regression to determine if age-structure differed among the dominant land-use types. I used the age class of each raccoon as the response variable, and the type of site the raccoon was captured in as the predictor variable to determine the individual’s relative risk of being in older age classes relative to younger age classes, while controlling for the sex of the individual. Juveniles were removed from age class I for this analysis because their availability for capture differed substantially, depending on when a site was sampled. If necessary, categories were combined in a biologically reasonable manner to avoid numerical calculation problems caused by zero-cells (Hosmer and Lemeshow 1989). Descriptive and inferential statistics were performed using program Stata 9.0 (StataCorp. 2005 College Station, TX).

## RESULTS

During July-August 2005, 4 urban open sites were sampled, and during April-August 2006, 3 rural open, 4 urban open and 7 urbanized sites were sampled (Table 2.1, Figure 2.2). On 3,476 trapnights, I captured 530 adult raccoons, 182 juvenile raccoons, 105 adult opossums and 80 cats (Table 2.2). To obtain parsimonious estimates of  $p$  and  $N$ , 16 closed-capture models were constructed in program MARK (Table 2.3). The best model was  $p(\text{Year}+\text{Age}) c(\cdot)$ , which had a year and sex effect on  $p$ , and  $p$  and  $c$  were invariant over trapping occasion. Since  $N$  was calculated as a derived parameter, I used model averaged estimates of  $N$  for the calculation of raccoon density at each of the sites.

Density estimates (raccoons/km<sup>2</sup>) ranged from 1.25-10.00 at urbanized sites, 3.0-29.25 at urban open sites, and 13.0-20.25 at rural open sites (Figure 2.3). Raccoon density differed by site type ( $F_{2,17} = 4.66$ ,  $P = 0.027$ ; Figure 2.4), mean density at urbanized sites was lower than urban open, and did not differ from rural open sites. Mean density of urban open and rural open sites were not significantly different. The top-ranked model of raccoon density was (intercept + OPEN + AG) with a weight of 0.754 (Table 2.4). The variable OPEN had a positive regression coefficient, while AG had a negative coefficient. The second-ranked model was (intercept + OPEN + HIGH URB) with a weight of 0.094.

Pooled sex ratios did not differ from 1:1 (M:F) in urban open (1.03:1;  $X^2 = 0.05$ , d.f. = 1,  $P = 0.887$ ) or urbanized (0.98:1;  $X^2 = 0.01$ , d.f. = 1,  $P = 1.00$ ) sites. However, the sex ratio departed from 1:1 (1.78:1;  $X^2 = 10.94$ , d.f. = 1,  $P = 0.001$ ) in rural open sites. There was no significant relationship between the proportion of males captured by site

and percent urban land-use ( $F_{1,17} = 1.35$ ,  $P = 0.263$ ). There was no association between site type and female reproductive condition ( $X^2 = 0.084$ , d.f. = 2,  $P = 0.959$ ; Table 2.5). There was no association between spring body condition index and site type for males ( $X^2 = 0.763$ , d.f. = 1,  $P = 0.382$ ) or females ( $X^2 = 2.498$ , d.f. = 1,  $P = 0.112$ ). I found no association between summer body condition index and site type for females ( $X^2 = 1.637$ , d.f. = 1,  $P = 0.201$ ), however, there was a significant association for males ( $X^2 = 7.199$ , d.f. = 1,  $P = 0.007$ ), which were in poorer condition at urbanized sites.

The median age class for urban open, rural open and urbanized sites was III, II and II respectively. Because no individuals of age class V were captured at urbanized sites (Figure 2.5), I pooled age classes IV and V into a new category, hereafter called IV+. Coefficients were positive when comparing the odds of a raccoon being age class II vs I, III vs I, IV+ vs I and IV+ vs II for an urban open individual relative to an individual from an urbanized site (all  $P$ -values  $\leq 0.04$ ; Table 2.6). In a comparison of individuals from rural open sites relative to individuals from urban open sites, coefficients were negative when comparing the odds of a raccoon being age class II vs I, III vs I, and IV+ vs I (all  $P$ -values  $\leq 0.01$ ; Table 2.6). When comparing individuals from urbanized sites with individuals from rural open sites, coefficients were positive when comparing the odds of a raccoon being age class II vs I and III vs I (all  $P$ -values  $\leq 0.02$ ; Table 2.6).

## DISCUSSION

My research is the first in which a raccoon population was studied using standardized methods at multiple, replicated sites across an urban landscape in the U.S. The use of similar methods and multiple study sites alleviates some of the confounding

factors associated with comparing past urban and rural raccoon research which was carried out using a variety of methods and often lacked replication. It is evident that urbanization and resulting land-use changes alter the density of raccoons across this urban landscape. Furthermore, urbanization was found to shape age structure.

Mean density of raccoons was higher in urban open sites than urbanized sites, but was not significantly different between urban open sites and rural open sites. My estimates of raccoon densities at urban open sites were generally lower than most other published raccoon densities in urban and suburban areas (Table 2.7). Densities varied greatly, even among sites of the same land-use type. The high degree of variation suggested that raccoon subpopulations were influenced by subtle differences in habitat quality that were not reflected in the variables I used, and differences in the availability of anthropogenic resources. The urban open sites with the lowest raccoon densities appeared to be in earlier successional stages and generally had less human activity and decreased access to refuse, relative to sites with higher raccoon densities (Graser personal observation). Given the adaptable feeding habits of the raccoon (Yeager and Elder 1945, Hoffman and Gottschang 1977), their ability to key in on areas of high anthropogenic resources (Prange et al. 2003, Bozek et al. 2007), and the fact that they exist in a wide spectrum of areas across the landscape, variation in density across similar site types could be expected. Similar patterns were observed in metropolitan Toronto, where density varied substantially across the study area, ranging from 56 raccoons/km<sup>2</sup> in forested parklands to 4 raccoons/km<sup>2</sup> in field areas (Rosatte et al. 1991). Substantial variation in raccoon density has also been observed in rural areas, ranging from  $\leq 1/\text{km}^2$  in North

Dakota prairie to 17.5/km<sup>2</sup> in an Ohio marsh, presumably due to differences in habitat quality (Fritzell 1978, Urban 1970).

The observed variation in density among land-use types and across similar site types has important implications for disease control, particularly for rabies. Specifically, with oral rabies vaccination (ORV) the distribution of an adequate amount of bait is crucial to achieving an adequate level of immunity in the target population. Without knowledge of the target population density, standard bait distribution densities may be insufficient to produce a populations-wide immunoprotective response, especially in high-density target populations (Ramey et al. 2008). Accounting for variation of raccoon density across the landscape would increase the effectiveness and cost efficiency of ORV programs by reducing the likelihood of under-baiting high density areas or over-baiting lower density areas. Identifying areas of high raccoon density and close human association would also allow managers to prioritize disease control efforts.

The models of raccoon density and land-use indicated the importance of forested habitat, as the models best supported by the data contained the variable OPEN, which represented the amount of forested parkland or undeveloped forest habitat at sites. The four highest ranked models all contained the variable OPEN and these models received a combined 98.6% of the model weight. These results indicated that forest habitat was the most important factor explaining raccoon density across this urban landscape. Other studies have also reported the importance of forested habitat to raccoons (Twitchell and Dill 1949, Pedlar et al. 1997, Chamberlain et al. 2003). In metropolitan Toronto, habitat appeared to be the primary factor determining raccoon density, which was highest in



forested parks followed by residential areas, commercial areas and fields (Rosatte et al. 1992).

Previously, researchers were limited to estimating population size only for grids where adequate data existed to estimate initial capture probability ( $p$ ) and recapture probability ( $c$ ) for that site. The estimation of nuisance parameters in common across multiple grids allows more precise estimation of detection probability and thus abundance (Finley et al. 2005). Although resulting estimates of population size are not likely to be completely unbiased, they will certainly have less bias than the minimum number known alive (MNKA; White 2005). Model selection results (i.e. the high degree of support for the simplest models) indicated that the length of trapping effort in my study did not provide adequate data to support complex models where  $p$  and  $c$  vary over trapping session. However, the use of a standardized trapping grid, similar trapping effort at most sites, and modeling of detection probability using biologically reasonable covariates served to minimize potential bias. Despite efforts to maintain consistent trapping methods at all sites, trapping effort differed slightly among sites due to logistical and time constraints. Rural open sites were trapped for 10 nights, whereas most urban open and urbanized sites were trapped for 5 nights, it is plausible that density estimates from sites sampled for 5 nights may be biased low. However, I believe that rural open estimates may also be biased low. These sites were sampled in April; the mean parturition date for Illinois raccoons is 18 April (Sanderson 1987) and female raccoons have been observed to exhibit restricted movements around this period (Ellis 1964), which would reduce their probability of being captured. In general my estimates of raccoon density should be similarly biased, meaning the observed trends and study

conclusions should be valid. If extremely accurate measures of raccoon density are desired it is likely that additional research will be required where trapping periods are extended beyond 5 occasions. Additionally, results suggested that habitat analyses that account for fine-scale differences in habitat quality (e.g. early successional vs. mature forest) and quantification of the availability of anthropogenic resources would be required to produce highly accurate estimates of raccoon density across the landscape.

I observed differences in population structure among land-use types. Although there were no clear patterns in sex ratio, reproductive condition or BCI, there were differences in age structure among urban open, rural open and urbanized sites. There appeared to be an overall male-bias in the population at rural open sites, however, this pattern may have reflected the period when the rural open sites were sampled as was previously discussed. There was no overall departure from 1:1 (M:F) sex ratio in urban open or urbanized sites and no relationship between male-biased sex ratio and the level of urban land-use at sites. Therefore, it is unlikely that populations in highly urbanized areas are “sink” populations maintained by primarily by dispersal.

I found no evidence of an association between the reproductive condition of females and site type. Although breeding condition is a relatively crude indicator of productivity, differences in reproduction between yearling females and adult females have been observed (Mankin et al. 1999), as well as differences between urban and rural female raccoons in Ontario using similar methods (Rosatte et al. 1991). Although I did not observe differences in the reproductive condition of females among the land-use types, it is difficult to distinguish whether the older age structure at urban open sites was

due to higher survival or differences in reproductive output without accurate knowledge of litter size.

In general, there did not appear to be an association between BCI and site type. When comparing raccoons captured during spring at rural open and urbanized sites there was no association between site type and body condition for males or females. In a comparison of raccoons captured during summer at urban open and urbanized sites there was no association between site type and body condition for females, however, significantly more males from urbanized sites in poor condition than males from urban open sites. Therefore, in general one site type was not likely providing an overwhelming advantage in terms of food resource availability.

The overall age structure of raccoons differed among urban open, rural open and urbanized sites. The most striking differences were the absence of the oldest age class (V) at urbanized sites and low numbers of age class I at urban open sites. Additionally, the rural open and urbanized sites had younger median age class than the urban open sites. The relatively low numbers of age class I individuals in the urban open sites may be indicative of low survival in fall and over-winter for juveniles. Winter can be a critical time for juveniles, when starvation and parasitism can cause significant mortality (Mech et al. 1968). In metropolitan Toronto, the highest frequency of deaths and the period where most road-kills were observed was in September-November, when juveniles were dispersing (Rosatte et al. 1991). The absence of individuals in age class V (>86 months) at the urbanized sites and the increased likelihood of individuals from urban open sites being in older age classes suggested that survival might be lower at

urbanized sites than at urban open sites. This is feasible given the numerous potential mortality sources at the urbanized sites.

Although much has been learned about urban and suburban raccoons in recent decades, significant knowledge gaps remain. Additional research on raccoon survival in urban areas, especially within the urbanized matrix is needed. Research to determine litter size and juvenile survival in urbanized areas would aid in quantifying annual recruitment and its contribution to population density. The prevalence and distance of dispersal as well as its relationship to habitat quality represent crucial knowledge deficits in the ecology of urban raccoons and could have important disease implications. Finally, raccoon density, population structure and demographic patterns should be explored at multiple sites in an urban environment over an extended time period, using fine-scale habitat variables to more accurately establish the trends and fluctuations in these parameters over time.

My results indicated that raccoon density was influenced by land-use, however, density varied substantially across the landscape, even in areas of the similar land-use type. Furthermore, age structure was influenced by land-use and may have reflected differences in the prevalence and importance of different mortality sources across the landscape. However, further research is required to better understand how adult and juvenile survival, litter size, and dispersal shape the density and demographic patterns of raccoons in urban environments. Understanding these dynamic processes will be crucial for wildlife managers to balance the needs of wildlife and humans in an increasingly urbanized world.

## LITERATURE CITED

- Alig, R. J., J. D. Kline, M. Lichtenstein. 2004. Urbanization on the US landscape: looking ahead in the 21<sup>st</sup> century. *Landscape and Urban Planning* 69: 219-234.
- Bozek, C. K., S. Prange, and S. D. Gehrt. 2007. The influence of anthropogenic resources on multi-scale habitat selection by raccoons. *Urban Ecosystems* 10: 413-425.
- Burnham, K. P., Anderson, D.R. 2002. Model selection and multi-model inference: a practical information-theoretic approach. 2<sup>nd</sup> ed. Springer-Verlag, New York.
- Chamberlain, M. J., L. M. Conner, B. D. Leopold, and K. M. Hodges. 2003. Space use and multi-scale habitat selection of adult raccoons in central Mississippi. *Journal of Wildlife Management* 67: 334-340.
- Chicago Metropolitan Agency for Planning. 2006. Data Bulletin: 2001 Land-use inventory for northeastern Illinois. Chicago Metropolitan Agency for Planning, Chicago, Illinois.
- Converse, S. J., W. M. Block, and G. C. White. 2006. Small mammal population and habitat responses to forest management and prescribed fire. *Forest Ecology and Management* 228: 263-273.
- Crooks, K. R. 2002. Relative sensitivities of mammalian carnivores to habitat fragmentation. *Conservation Biology* 16: 488-502.
- DeStephano, S., and R. D. Deblinger. 2005. Wildlife as valuable natural resources vs. intolerable pests: a suburban wildlife management model. *Urban Ecosystems* 8: 179-190.
- Dobson, A. 2000. Raccoon rabies in space and time. *Proceedings of the National Academy of Sciences* 97: 14041-14043.
- Ellis, R. J. 1964. Tracking raccoons by radio. *Journal of Wildlife Management* 28: 363-368.

- Fishbein, D. B., and L. E. Robinson. 1993. Rabies. *New England Journal of Medicine* 329: 1632-1638.
- Finley, D. J., G. C. White, and J. P. Fitzgerald. 2005. Estimation of swift fox population size and occupancy rates in eastern Colorado. *Journal of Wildlife Management* 69: 861-873.
- Fritzell, E. K. 1978. Aspects of raccoon (*Procyon lotor*) social organization. *Canadian Journal of Zoology* 56: 260-271.
- Gehrt, S. D. 2002. Evaluation of spotlight and road-kill surveys as indicators of local raccoon abundance. *Wildlife Society Bulletin* 30: 449-456.
- Gehrt, S. D. 2003. Raccoons and allies. In *Wild mammals of North America: biology, management, and conservation* (G. A. Feldhamer, B. C. Thompson, and J.A. Chapman, eds.). 2nd ed. Johns Hopkins University Press, Baltimore, Maryland, pp. 611-633.
- Gehrt, S. D., and E. K. Fritzell. 1998. Duration of familial bonds and dispersal patterns for raccoons in south Texas. *Journal of Mammalogy* 79: 859-872.
- Gehrt, S. D., L. L. Hungerford and S. Hatten. 2001. Drug effects on recaptures of raccoons. *Wildlife Society Bulletin* 29: 833-837.
- Grau, G. A., G. C. Sanderson, and J. P. Rogers. 1970. Age determination of raccoons. *Journal of Wildlife Management* 34: 364-372.
- Grimm, N. B., S. H. Faeth, N. E. Golubiewski, C. L. Redman, J. Wu, X. Bai, and J. M. Briggs. 2008. Global change and the ecology of cities. *Science* 319: 756-760.
- Hoffman, C. O., and J. L. Gottschang. 1977. Numbers, distribution, and movements of a raccoon population in a suburban residential community. *Journal of Mammalogy* 58: 623-636.
- Holmes, R. T., P. P., Marra, and T. W. Sherry. 1996. Habitat-specific demography of breeding black-throated blue warblers (*Dendroica caerulescens*): implications for population dynamics. *Journal of Animal Ecology* 65: 183-195.
- Hosmer, D. W., and S. Lemeshow. 1989. *Applied logistic regression*. John Wiley & Sons, New York.
- Huggins, R. M. 1989. On the statistical analysis of capture experiments. *Biometrika* 76: 133-140.

- Huggins, R. M. 1991. Some practical aspects of a conditional likelihood approach to capture experiments. *Biometrics* 47: 725-732.
- Hurvich, C. M., and C. L. Tsai. 1989. Regression and time series model selection in small sample sizes. *Biometrika* 76: 297-307.
- Jacobson, J. E., K. R. Kazacos, and F. H. Montague. 1982. Prevalence of eggs of *Baylisascaris procyonis* (Nematoda:Ascaroidea) in raccoon scats from an urban and rural community. *Journal of Wildlife Disease* 18: 461-464.
- Jakob, E. M., S. D. Marshall, and G. W. Uetz. 1996. Estimating fitness: a comparison of body condition indices. *Oikos* 77: 61-67.
- Kruezer, M. P., N. J. Huntly. 2003. Habitat-specific demography: evidence for source-sink population structure in a mammal, the pika. *Oecologia* 134: 343-349.
- Mankin, P. C., C. M. Nixon, J. B. Sullivan, T. L. Esker, R. G. Koerkenmeier and L. L. Hungerford. 1999. Raccoon (*Procyon lotor*) survival in west-central Illinois. *Transactions of the Illinois State Academy of Science* 92: 247-256.
- McKinney, M. L. 2002. Urbanization, biodiversity, and conservation. *Bioscience* 52: 883-890.
- McKinney, M. L. 2006. Urbanization as a major cause of biotic homogenization. *Biological Conservation* 127: 247-260.
- Mech, L. D., D. M. Barnes, and J. R. Tester. 1968. Seasonal weight changes, mortality, and population structure of raccoons in Minnesota. *Journal of Mammalogy* 49: 63-73.
- Moore, D. W., M. L. Kennedy. 1985. Weight changes and population structure of raccoons in western Tennessee. *Journal of Wildlife Management* 49: 906-909.
- National Oceanic and Atmospheric Administration (NOAA). 2002. Climatography of the United States No. 81: Monthly normals of temperature, precipitation, and heating and cooling degree days 1971-2000. National Climatic Data Center/NESDIS/NOAA, Asheville, North Carolina.
- Northeastern Illinois Planning Commission. 2006. 2030 forecasts of population, households and employment by county and municipality. Northeastern Illinois Planning Commission, Chicago, Illinois.
- Pedlar, J. H., L. Fahrig, and H. G. Merriam. 1997. Raccoon habitat use at 2 spatial scales. *Journal of Wildlife Management* 61: 102-112.

- Prange, S., S. D. Gehrt, and E. P. Wiggers. 2003. Demographic factors contributing to high raccoon densities in urban landscapes. *Journal of Wildlife Management* 67: 324-333.
- Prange, S., and S. D. Gehrt. 2004. Changes in mesopredator-community structure in response to urbanization. *Canadian Journal of Zoology* 82: 1804-1817.
- Ramey, P. C., B. F. Blackwell, R. J. Gates, and R. D. Slemons. 2008. Oral rabies vaccination of a northern Ohio raccoon population: relevance of population density and prebait serology. *Journal of Wildlife Disease* 44: 553-568.
- Riley, S. P. D., J. Hadidian, and D. A. Manski. 1998. Population density, survival, and rabies in raccoons in an urban national park. *Canadian Journal of Zoology* 76: 1153-1164.
- Rosatte, R. C., M. J. Power, and C. D. MacInnes. 1991. Ecology of urban skunks, raccoons and foxes in metropolitan Toronto. In *Wildlife conservation in metropolitan environments*. Edited by L. W. Adams and D. L. Leedy. National Institute for Urban Wildlife, Columbia, Maryland, pp. 31-38.
- Rosatte, R. C., M. J. Power, and C. D. MacInnes. 1992. Density, dispersion, movements, and habitat of skunks (*Mephitis mephitis*) and raccoons (*Procyon lotor*) in metropolitan Toronto. In *Wildlife 2001: populations*. Edited by D. R. McCullough and R. H. Barrett. Elsevier Science, Barking, Essex, UK. pp. 932-942.
- Sanderson, G. C. 1987. Raccoon. In M. Novak, J.A. Baker, M. E. Obbard, and B. Malloch, eds. *Wild furbearer management and conservation in North America*. Ontario Trappers Association, North Bay, Canada, pp. 487-499.
- Sanderson, G. C., and A. V. Nalbandov. 1973. The reproductive cycle of the raccoon in Illinois. *Illinois Natural History Survey Bulletin* 31: 25-85.
- Schinner, J. R., and Cauley, D. L. 1974. The ecology of urban raccoons in Cincinnati, Ohio. In *Wildlife in an urbanizing environment*. Edited by J. H. Noyes and D. R. Progulsk. Planning and Resource Development Series No. 28, Holdsworth Natural Resources Center, Amherst, Massachusetts, pp. 125-130.
- StataCorp. 2005. *Stata statistical software: release 9*. College Station, TX: StataCorp LP.
- Twichell, A. R., and H. H. Dill. 1949. One hundred raccoons from one hundred and two acres. *Journal of Mammalogy* 30: 130-133.
- Urban, D. 1970. Raccoon populations, movement patterns, and predation on a managed waterfowl marsh. *Journal of Wildlife Management* 34: 372-282.



- U.S. Census Bureau. 1995. Population of counties by census: 1900 to 1990. Population Division, U.S. Bureau of the Census, Washington D.C., USA.
- U.S. Census Bureau. 2001. Statistical abstract of the United States: 2001, 112<sup>th</sup> edition. U.S. Bureau of the Census, Washington D.C., USA.
- U.S. Census Bureau. 2006a. Statistical abstract of the United States: 2006, 125<sup>th</sup> edition. U.S. Bureau of the Census, Washington D.C., USA.
- U.S. Census Bureau. 2006b. State and county QuickFacts. U.S. Bureau of the Census, Washington D.C., USA.
- 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. 2005. Correcting wildlife counts using capture probabilities. *Wildlife Research* 32: 211-216.
- White, G. C., and K. P. Burnham. 1999. Program MARK: survival estimation from populations of marked animals. *Bird Study* 46: 120-138.
- Yeager, L. E., W. H. Elder. 1945. Pre-and post-hunting season foods of raccoons on an Illinois goose refuge. *Journal of Wildlife Management* 9: 48-56.

Site	Dominant Land-Use	% Agriculture	% High Density Urban	% Low Density Urban	% Medium Density Urban	% Open Space	% Surface Water	% Barren
18-28 <sup>b</sup>	Urbanized	0.00	19.29	10.11	61.96	10.03	0.13	0.00
19-22 <sup>b</sup>	Urbanized	0.00	18.98	12.12	63.45	5.78	0.08	0.00
26-17 <sup>b</sup>	Urbanized	0.00	21.44	1.21	54.15	23.74	0.00	0.00
20-25 <sup>b</sup>	Urbanized	0.00	14.28	2.59	76.54	6.91	0.00	0.00
24-16 <sup>b</sup>	Urbanized	0.00	16.51	6.32	70.94	6.61	0.18	0.00
27-14 <sup>b</sup>	Urbanized	0.00	23.94	6.21	56.20	14.26	0.00	0.00
15-33 <sup>b</sup>	Urbanized	0.07	66.74	1.37	21.90	10.03	0.00	0.00
20-30 <sup>b</sup>	Urban Open	0.18	15.76	2.63	26.61	52.79	2.21	0.00
19-15 <sup>b</sup>	Urban Open	15.07	0.05	0.90	2.34	71.35	10.78	0.11
28-3 <sup>b</sup>	Urban Open	0.06	1.55	4.99	24.90	68.61	0.80	0.00
19-19 <sup>b</sup>	Urban Open	0.05	18.92	2.33	15.66	63.11	0.26	0.16
30-8 <sup>a</sup>	Urban Open	0.16	2.79	2.41	16.24	71.07	2.00	6.11
23-11 <sup>a</sup>	Urban Open	3.67	2.17	5.29	32.10	57.22	0.25	0.00
17-13 <sup>a</sup>	Urban Open	30.55	0.00	0.67	0.52	53.83	15.07	0.00
23-6 <sup>a</sup>	Urban Open	21.10	1.42	3.31	10.65	62.33	1.20	0.83
Rush Creek <sup>b</sup>	Rural Open	1.63	0.00	1.94	0.15	95.10	0.94	0.18
Marengo Ridge <sup>b</sup>	Rural Open	27.30	0.00	0.84	0.84	71.60	0.22	0.00
Coral Woods <sup>b</sup>	Rural Open	21.10	0.00	2.24	2.24	75.00	0.31	0.00

<sup>a</sup> site sampled in 2005.

<sup>b</sup> site sampled in 2006.

Table 2.1. The percent coverage of land-use types for sampled grid cells in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006. High Density Urban = impervious surfaces account for 80-100% of the total cover (e.g. row houses, and commercial and industrial areas). Low Density Urban = impervious surfaces account for 20-50% of the total cover (e.g. single family housing units). Medium Density Urban = impervious surfaces account for 50-80% of the total land cover (e.g. single family housing units). Open Space = parklands and remnant habitat fragments (e.g. forested and grassland areas).

Site	Dominant Land-Use	Trapnights	Adult Raccoons	Juvenile Raccoons	Adult Opossums	Cats
18-28	Urbanized	150	3	9	7	6
19-22	Urbanized	148	12	7	1	9
26-17	Urbanized	300	29	0	15	5
20-25	Urbanized	300	10	0	23	28
24-16	Urbanized	146	3	24	4	2
27-14	Urbanized	141	10	6	5	2
15-33	Urbanized	133	22	18	3	9
	Total	1318	89	64	58	61
20-30	Urban Open	150	41	17	9	12
19-15	Urban Open	300	69	8	4	1
28-3	Urban Open	150	67	25	1	0
19-19	Urban Open	150	43	20	5	1
30-8	Urban Open	150	45	18	4	2
23-11	Urban Open	110	14	4	3	0
17-13	Urban Open	108	16	3	1	0
23-6	Urban Open	140	8	5	2	0
	Total	1258	303	100	29	15
Rush Creek	Rural Open	300	60	7	7	1
Marengo Ridge	Rural Open	300	41	6	6	0
Coral Woods	Rural Open	300	37	5	5	3
	Total	900	138	18	18	4
	All Sites	3476	530	182	105	80

Table 2.2. Raccoon trapping results, including trapping effort, and the number of adult and juvenile raccoons, opossums and cats captured at sites in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006.

Model <sup>a</sup>	<i>K</i>	AICc	ΔAICc	ω <sub>i</sub>	Model likelihood	Deviance
{ <i>p</i> (Year+Age) <i>c</i> (.)}	4	2884.557	0	0.46439	1	2876.549
{ <i>p</i> (.) <i>c</i> (.)}	2	2886.199	1.641	0.20439	0.4401	2882.197
{ <i>p</i> (Year+Sex+Age) <i>c</i> (.)}	5	2886.468	1.911	0.17865	0.3847	2876.457
{ <i>p</i> (Year) <i>c</i> (.)}	3	2887.673	3.115	0.09782	0.2106	2881.668
{ <i>p</i> (Year+Sex) <i>c</i> (.)}	4	2889.504	4.947	0.03915	0.0843	2881.497
{ <i>p</i> ( <i>t</i> )= <i>c</i> ( <i>t</i> )+const}	11	2891.428	6.871	0.01496	0.0322	2869.378
{ <i>p</i> (Year+Sex+Age)= <i>c</i> (.)}	4	2899.760	15.202	0.00023	0.0005	2891.752
{ <i>p</i> (Sex+Age)= <i>c</i> (.)}	3	2900.293	15.735	0.00018	0.0004	2894.288
{ <i>p</i> ( <i>t</i> +Year+Sex+Age)= <i>c</i> ( <i>t</i> )}	13	2901.528	16.971	0.00010	0.0002	2875.459
{ <i>p</i> (Year+Sex)= <i>c</i> (.)}	3	2903.043	18.485	0.00004	0.0001	2897.038
{ <i>p</i> (Year+Age)= <i>c</i> (.)}	3	2903.403	18.846	0.00004	0.0001	2897.399
{ <i>p</i> ( <i>t</i> +Year+Sex)= <i>c</i> ( <i>t</i> )}	12	2904.210	19.653	0.00003	0.0001	2880.151
{ <i>p</i> ( <i>t</i> +Year+Age)= <i>c</i> ( <i>t</i> )}	12	2905.264	20.707	0.00001	0	2881.205
{ <i>p</i> (.)= <i>c</i> (.)}	1	2906.395	21.838	0.00001	0	2904.395
{ <i>p</i> ( <i>t</i> +Year)= <i>c</i> ( <i>t</i> )}	11	2907.706	23.148	0	0	2885.655
{ <i>p</i> ( <i>t</i> )= <i>c</i> ( <i>t</i> )}	10	2908.393	23.836	0	0	2888.351

<sup>a</sup> Variable definitions: *p* = initial capture probability, *c* = recapture probability, *t* = trapping occasion specific estimates, Year = year trapping took place, Age = age of the raccoon (yearling or adult), Sex = raccoon's sex, const = additive effect on recaptures from initial captures, *K* = number of parameters estimated.

Table 2.3. Model selection results of the 16 models constructed in program MARK to estimate raccoon abundance at 18 sites in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006. Akaike weight (ω<sub>i</sub>) and number of parameters (*K*) are also reported for each model.

Model <sup>a</sup>	<i>K</i>	AICc	ΔAICc	Likelihood	ω <sub>i</sub>	<i>R</i> <sup>2</sup> adj.
OPEN + AG	3	114.1	0	1	0.754	0.61
OPEN + HIGH URB	3	118.2	4.1	0.125243	0.094	0.51
OPEN	2	118.2	4.1	0.124636	0.093	0.46
OPEN + WATER	3	121.0	6.9	0.031304	0.023	0.43
OPEN + BARREN	3	121.1	7.0	0.029086	0.022	0.42
MEDIUM URB	2	123.2	9.1	0.010654	0.008	0.29
MEDIUM URB + WATER	3	125.5	11.4	0.003258	0.002	0.27
MEDIUM URB + HIGH	3	125.9	11.8	0.002576	0.002	0.25

<sup>a</sup> OPEN = urban and rural open space; AG = agriculture; HIGH URB = high-density urban; MEDIUM URB = medium-density urban; WATER = surface water and non-forested wetland; BARREN = barren land.

Table 2.4. Candidate models explaining variation in raccoon density (raccoons/km<sup>2</sup>) at sites sampled in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006. Models are ranked by small sample-corrected Akaike's Information Criterion (AICc). Also included are the number of parameters (*K*), model weight (ω<sub>i</sub>), and proportion of variance accounted for (*R*<sup>2</sup> adj).

Site type	<i>n</i>	% Reproductive
Urban Open	45	60.0
Urbanized	133	59.4
Rural Open	42	61.9

Table 2.5. The percentage of females exhibiting current reproductive activity (lactating, pregnant, young present in trap) by site type in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006.

LAND USE														
Urban Open vs Urbanized					Rural Open vs Urban Open					Urbanized vs Rural Open				
Age Class	B	P-value	$e^{\beta^a}$	$e^{\beta Stdx}$	Age Class	$\beta$	P-value	$e^{\beta}$	$e^{\beta Stdx}$	Age Class	$\beta$	P-value	$e^{\beta}$	$e^{\beta Stdx}$
II vs I	1.15	0.01	3.17	1.77	II vs I	-2.15	0.00	0.12	0.39	II vs I	1.00	0.01	2.71	1.46
III vs I	1.50	0.00	4.47	2.10	III vs I	-2.49	0.00	0.08	0.33	III vs I	0.99	0.02	2.69	1.45
IV+ vs I	2.01	0.00	7.44	2.71	IV+ vs I	-2.28	0.00	0.10	0.37	IV+ vs I	0.27	0.61	1.31	1.11
III vs II	0.34	0.23	1.41	1.19	III vs II	-0.34	0.22	0.72	0.86	III vs II	-0.01	0.98	0.99	1.00
IV+ vs III	0.51	0.25	1.66	1.29	IV+ vs III	0.21	0.55	1.23	1.10	IV+ vs III	-0.72	0.16	0.49	0.76
IV+ vs II	0.85	0.04	2.35	1.53	IV+ vs II	-0.12	0.71	0.88	0.95	IV+ vs II	-0.73	0.13	0.48	0.76

<sup>a</sup>  $e^{\beta}$  = factor change in odds for a 1 unit change in Land-use type.

Table 2.6. Resulting coefficients ( $\beta$ ) and odds ratios ( $e^{\beta}$ ) from 3 polytomous logistic regression equations with the age class (class I [0-14 months], class II [15-38 months], class III [39-57 months], class IV [58-86 months], class V [>86months]) of an individual raccoon as the response variable and land-use type as the predictor variable, while controlling for the sex of the individual. The odds ratios represent the odds of a raccoon being in one age class versus another when comparing one land-use type to another.

Location	Density <sup>a</sup>	Urban/Suburban	Reference
Washington, D.C	125	Urban	Riley et al. 1998
Cincinnati, OH	111	Urban	Schinner and Cauley 1974
Glendale, OH	66	Suburban	Hoffman and Gottschang 1977
Sandusky, OH	91	Suburban/Rural	Ramey et al. 2008
Northeastern, IL	40-70	Urban	Prange et al. 2003
Scarborough, ON	7-12	Mixed	Rosatte et al. 1991

<sup>a</sup>Raccoons/km<sup>2</sup>

Table 2.7. Estimates of raccoon density from published mark-recapture studies conducted in urban and suburban areas.

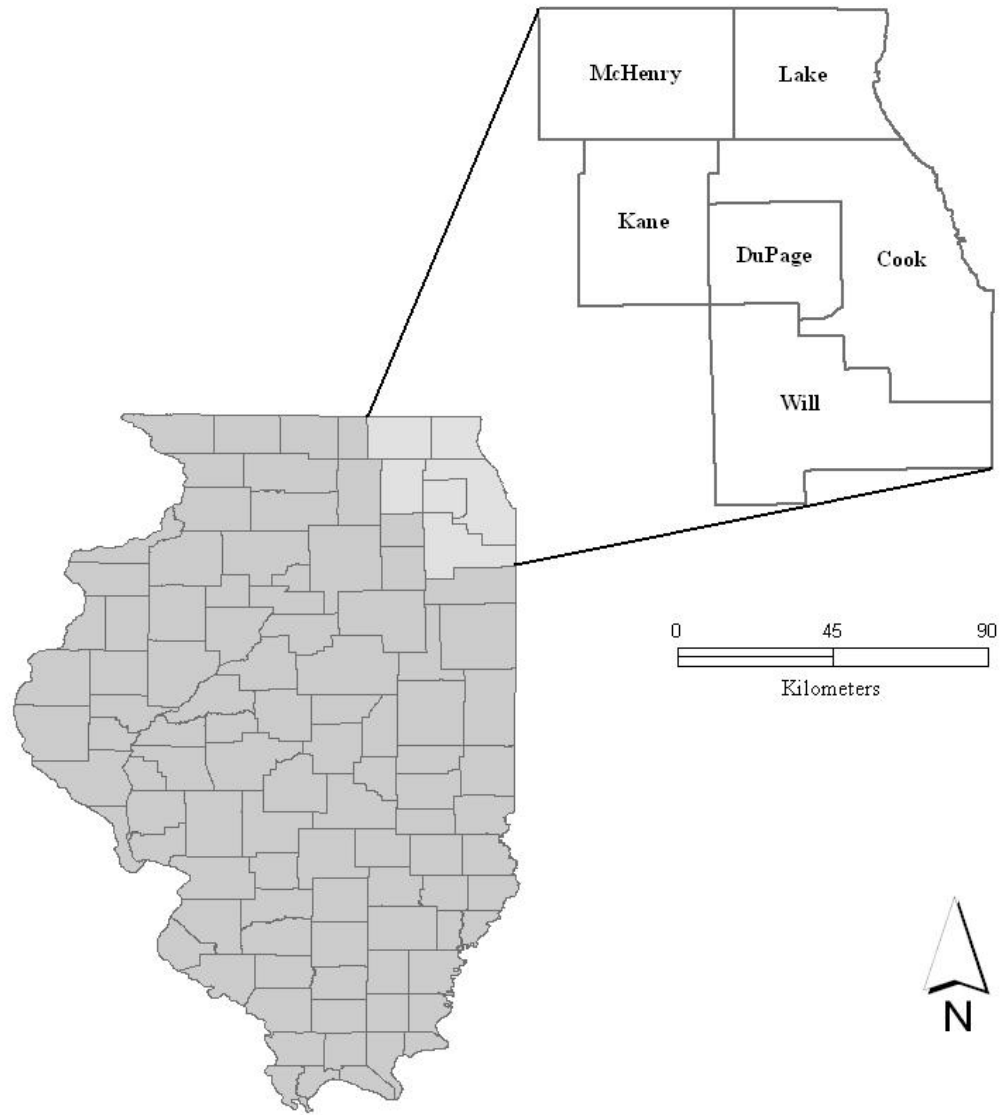


Figure 2.1. The state of Illinois with emphasis on the 6-county Chicago metropolitan area including Cook, DuPage, Kane, Lake, McHenry and Will Counties.



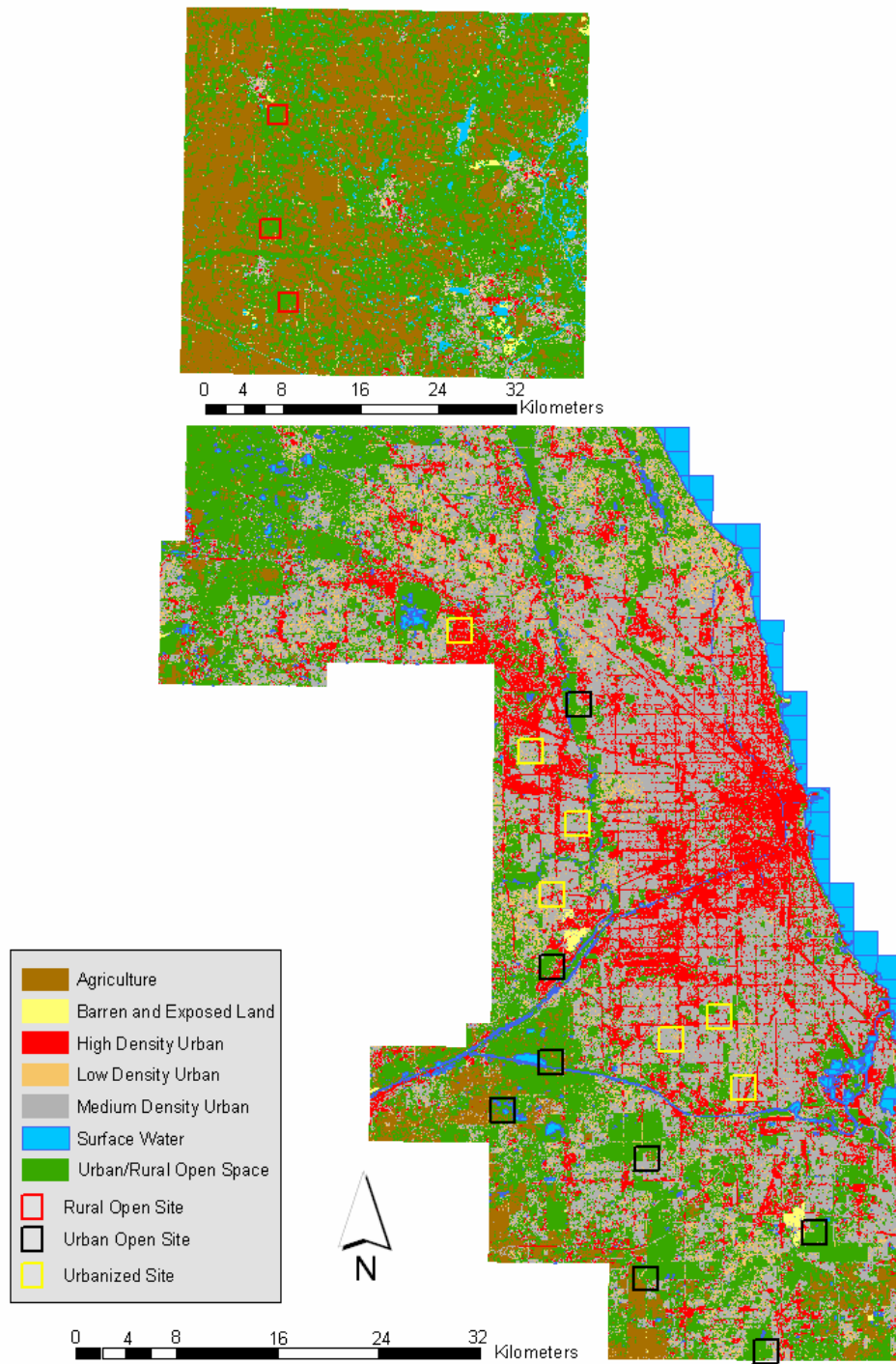


Figure 2.2. Sites sampled in Cook County (lower) and McHenry County (upper), northeastern Illinois, USA, 2005-2006.

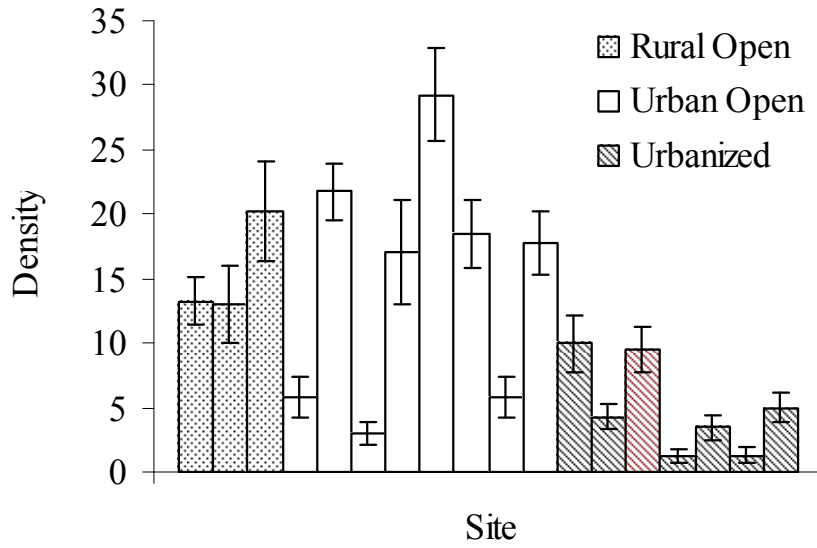


Figure 2.3. Model-averaged raccoon density estimates (raccoons/km<sup>2</sup>) and unconditional standard error (SE) at rural open, urban open, and urbanized sites in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006.

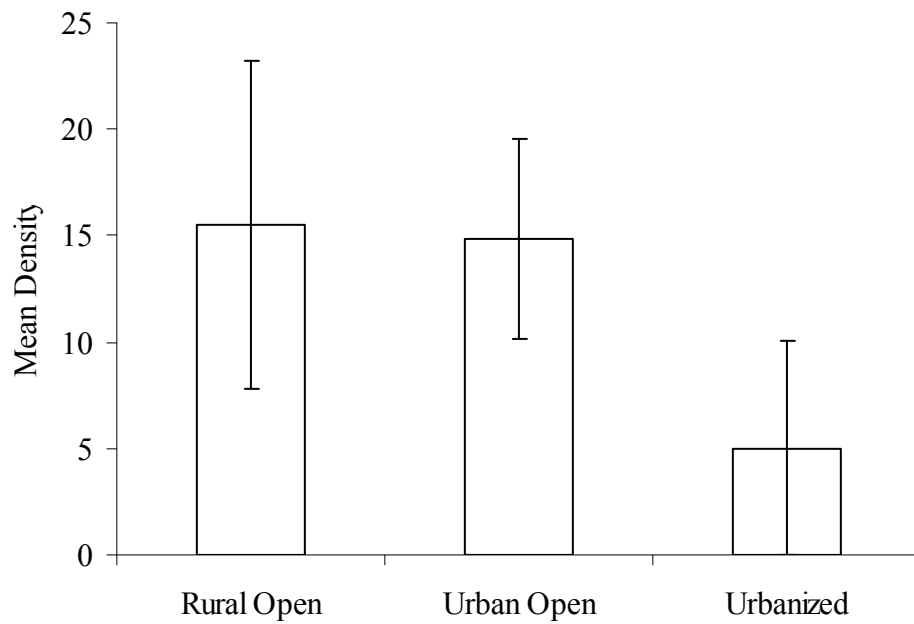


Figure 2.4. Mean raccoon density (raccoons/km<sup>2</sup>) with 95% confidence interval for each site type as determined by dominant land-use, Cook County and McHenry County, northeastern, Illinois, USA, 2005-2006.

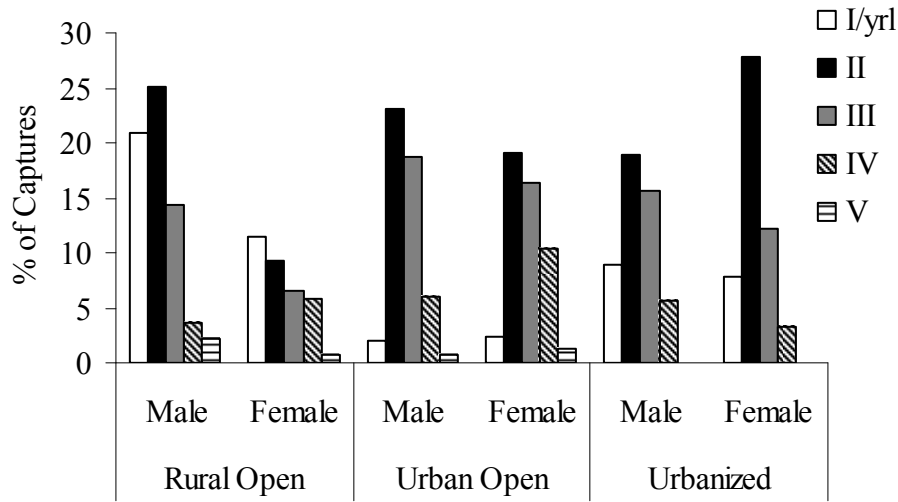


Figure 2.5. The percentage of captures in each age class (Grau et al. 1970), excluding juveniles, by site type and sex in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006. Age classes: class I/yr1 (0-14 months), class II (15-38 months), class III (39-57 months), class IV (58-86 months), class V (>86months). Juveniles were not included in age class I due to disparity in their availability for capture over the study period.

## CHAPTER 3

### URBANIZATION AND PATHOGEN EXPOSURE: A SEROLOGICAL SURVEY FOR EXPOSURE TO SELECTED INFECTIOUS DISEASE AGENTS IN RACCOONS OF THE CHICAGO METROPOLITAN AREA

#### INTRODUCTION

Rising human population growth in the United States and abroad, has resulted in increased levels of urbanization nationally and globally. Urbanization can impact overall wildlife community composition and can have a range of effects on the distribution and abundance of wildlife species. There is increasing evidence suggesting that urbanization and resultant land-use changes contribute to the emergence of wildlife diseases through multiple mechanisms (Bradley and Altizer 2006), with consequences for human health and wildlife conservation. In light of the increasingly close association between wildlife and humans, the need for a better understanding of how human presence and land-use changes affect wildlife-pathogen dynamics will be vital for the management and conservation of wildlife and limiting the risk of human exposure to zoonotic diseases (Bradley and Altizer 2006).

Urbanization often results in the permanent loss of habitat and further influences remaining natural areas through increased fragmentation and isolation, and alteration of the matrix in which they are embedded (Wang and Moskovits 2001). Fragmentation and

isolation can have a range of effects on the composition of animal communities. The distribution and abundance of mammalian carnivores can be highly influenced by fragmentation (Crooks 2002). While many species are negatively impacted by urbanization, generalist species with flexible dietary and habitat requirements can respond positively to urbanization (McKinney 2002). Urban-adapted species can benefit from access to anthropogenic resources in urbanized environments. The spatial distribution of resources can influence the movement patterns of a species as well as patterns of intra and inter-specific contact rates, with important implications for disease transmission.

Disease is of great importance to the health and safety of humans, domestic animals and imperiled wildlife species. Fifty-eight percent of recognized human pathogens are zoonotic and 73% of emerging and reemerging pathogens are known to be zoonotic (Woolhouse and Gowtage-Sequeria 2005). Land-use changes such as deforestation, fragmentation, human settlement and the movement of humans and domestic animals can be important drivers of infectious disease emergence (Patz et al. 2004). For example recent research has linked urban sprawl, forest fragmentation and loss of biodiversity to increased risk for Lyme disease in the northeastern United States (Schmidt and Ostfeld 2001). Imperiled wildlife can be adversely affected by diseases and parasites (e.g. Allegheny woodrat [*Neotoma magister*]; LoGiudice 2003). There is also concern that diseases in urban wildlife represent a potential risk to zoological animal collections (Junge et al. 2007).

The raccoon is a common medium-sized carnivore with a distribution that extends over much of North America (Gehrt 2003). Raccoons exist in a variety of environments

and high densities of raccoons are often found in urban (Riley et al. 1998, Rosatte et al. 1991, Prange et al. 2003) and suburban areas (Schinner and Cauley 1974, Hoffman and Gottschang 1977) relative to their rural counterparts (Fritzell 1978, Mech et al. 1968). Raccoons respond to seasonally and locally abundant food sources (Yeager and Elder 1945) and their foraging behavior can be heavily influenced by the abundance and distribution of anthropogenic resources (Bozek et al. 2007). Raccoons appear to be more efficient at exploiting anthropogenic resources than other mesocarnivores (Prange and Gehrt 2004). Resource distribution may have an important role in disease transmission, as experimentally increasing the contact rate of raccoons by manipulating resource availability resulted in altered parasite assemblages (Wright and Gompper 2005).

Raccoons are the definitive host of *Baylisascaris procyonis*, which is considered to be an emerging helminthic zoonosis (Sorvillo et al. 2002), and prevalence of infection can be high (Page et al. 2008). Canine distemper virus (CDV) is an infectious, contagious viral disease and is among the most significant infectious diseases of domestic and wild carnivores. Transmission is primarily by aerosol or contact with oral, respiratory, and ocular fluids and exudates containing the virus. Close association between affected and susceptible animals is required, as CDV is highly fragile in the environment (Williams 2001). CDV is cited as an important mortality source in many raccoon populations (Roscoe 1993, Schinner and Cauley 1974, Mech et al. 1968) and prevalence has been observed to be high in serological surveys (Junge et al. 2007, Mitchell et al. 1999, Schubert et al. 1998, Hoff et al. 1974). Canine distemper can be important as a mortality factor and threat to certain highly susceptible species, such as the gray fox (*Urocyon cinereoargenteus*), and several threatened and endangered carnivores

(Williams 2001). Along with distemper, parvovirus infection is one of the most important infectious diseases of raccoons (Barker and Parrish 2001). Transmission is thought to be through the fecal-oral route, most likely through the ingestion of the virus from the environment rather than through direct contact (Reif 1976). Exposure to parvovirus is commonly detected in raccoons (Rosatte et al. 1991, Junge et al. 2007) which may pose a risk to pets and captive animal collections. *Toxoplasma gondii* is a protozoan parasite that is widespread in humans and other warm-blooded animals. Exposure in raccoons is common and seroprevalence can be quite high, with seroprevalence as high as 88% being observed in free-ranging raccoons (Hancock et al. 2005). Due to their omnivorous food habits raccoons can be exposed to *T. gondii* by ingesting tissue cysts from infected prey or oocysts from the environment. Raccoons are a reservoir for the pathogen *Leptospira spp.*, most commonly serovar *grippotyphosa* and have been suggested for use as serologic sentinels for leptospirosis and other diseases (Bigler et al. 1975). Pseudorabies is caused by swine herpes virus 1, which is also called pseudorabies virus (PRV). Domestic and wild swine are the reservoir hosts for PRV, however PRV can infect and cause disease in many wild vertebrate hosts including raccoons (Stallknecht and Howerth 2001). The potential role of raccoons in the transmission of pseudorabies virus is unclear.

Because raccoons utilize a variety of terrestrial and aquatic habitats, and are highly omnivorous, they represent an effective indicator of many diseases and environmental contaminants (Bigler et al 1975). Urbanization can have profound effects on the density and demographic patterns of raccoons often placing high density raccoon populations in close association with humans and domestic animals. The distribution and



abundance of resources can influence patterns of intraspecific contact, potentially impacting disease transmission (Wright and Gompper 2005). These factors may contribute to different patterns of pathogen exposure across the landscape and therefore varying levels of risk to the health and safety of humans, domestic animals and other wildlife. The objectives of my research were to: 1) document patterns of exposure to infectious disease agents of public and animal health significance (e.g. *Toxoplasma gondii*, *Leptospira spp.*, canine distemper virus, parvovirus, and pseudorabies virus) in raccoons across an urban landscape, 2) test for differences in the patterns of exposure to selected infectious disease agents for raccoons inhabiting different areas of the landscape (i.e. urban open, rural open, and urbanized areas).

## METHODS

### Study Area

#### The Chicago Metropolitan Area

The city of Chicago and its surrounding suburbs, collectively referred to as the Chicago metropolitan area, have grown substantially in recent decades. The Chicago-Naperville-Joliet, IL-IN-WI Metropolitan Statistical Area (MSA) contains 14 counties from southeastern Wisconsin to northwest Indiana and had an estimated population of 9,443,356 in 2005, making it the 3<sup>rd</sup> largest MSA in the United States (USCB 2006a). Within this vast urbanized area exists approximately 810 km<sup>2</sup> of protected natural areas in county preserves, state parks, federal preserves and privately owned areas, known as the Chicago Wilderness (Wang and Moskovits 2001). The Illinois portion of the Chicago metropolitan area is generally considered to consist of the following 6 counties: Cook, DuPage, Kane, Lake, McHenry and Will, which had an estimated population of

8,092,145 in 2000 (USCB 2001). The Northeastern Illinois Planning Commission (NIPC) has forecasted that the population of this area will exceed 10,000,000 by 2030, attaining the status of megacity (NIPC 2006).

Local climate is mainly continental however conditions can be strongly influenced by Lake Michigan. At the O'Hare International Airport weather station, mean annual temperature for 1971-2000 was 9.5°C, with mean temperatures for February and July being -2.8° and 22.9°C respectively. Mean precipitation over this period was 92.13cm (NOAA 2002).

#### Cook County

Cook County is located in northeastern Illinois, has 2,449.30 km<sup>2</sup> of land area, is home to the city of Chicago, and had an estimated population of 5,288,265 and overall human density of 2159.09/ km<sup>2</sup> in 2006 (USCB 2006b; Figure 2.1). Population change in Cook County was greatest from 1910-1970 when there was a 128% increase; after the 1970 census the population declined slightly (USCB 1995). The Chicago Metropolitan Agency for Planning (CMAP) estimated that approximately 71% of the county was occupied by urban land-uses and 15% of the county was open space in 2001 (CMAP 2006). The Cook County Forest Preserve District (CCFPD) owned and managed 271.14 km<sup>2</sup> of open land, or approximately 11% of the county's land mass as of 2007 (Chris Anchor, CCFPD, pers. comm.).

#### McHenry County

McHenry County is located in northeastern Illinois approximately 50 km from the city of Chicago and is 1582 km<sup>2</sup> (Figure 2.1). Although historically a predominantly agricultural county, McHenry County has recently experienced marked urbanization.

Between 1960 and 1990 the county's population increased 117.6% (USCB 1995), and there was a 70% increase between 1990 and 2006 when overall population was estimated to be 312,373 and population density was 199.84/km<sup>2</sup> (USCB 2006b). Urbanization is expected to continue, and the population of McHenry County is expected to reach 457,594 by 2030 (NIPC 2006). Urban and built up areas occupied approximately 21% of McHenry County as of 2001, and roughly 50% of the county was in agricultural use (CMAP 2006). The McHenry County Conservation District owns 87 km<sup>2</sup> of woodlands, wetlands, prairies and savannahs within the county.

#### Habitat data and site selection

I used the land cover mapping and classification data compiled by the Illinois Gap Analysis Project (IL-GAP) to determine land cover composition, select sites and perform data analyses. I created layers of Cook County and McHenry County using a geographic information system (GIS) and the IL-GAP coverage data, and overlaid a grid with 4km<sup>2</sup> cells onto Cook County. Land cover classifications were simplified for the purposes of this research by consolidating several classifications used by IL-GAP to form 6 basic land cover classifications: open space (urban or rural parklands and undeveloped areas consisting primarily of natural vegetation communities), open water, high-density urban (impervious surfaces account for 80-100% of total cover, typically industrial and commercial land-uses), medium-density urban (50-80% impervious surfaces, typically single-family housing), low-density urban (20-50% impervious surfaces, typically single-family housing) and agriculture. Percent land cover for each of the classes was calculated for each grid cell using Microsoft Access. Cells were classified as being either

urban open space, rural open space, or high, medium or low-density urban land-use, dependent on the primary land-use type within the cell. In Cook County I selected live-trapping grid cells using a stratified random sampling method based on the IL-GAP land cover data. If a site could not be accessed or was adjacent to a previously selected cell, a new cell was randomly selected. Additionally, 3 sites were selected in protected open spaces in rural areas of McHenry County, due to the paucity of agricultural areas in Cook County.

### Capture and Handling

I live-trapped raccoons using single-door Tomahawk box traps (Tomahawk Live Trap Co., Tomahawk, Wisconsin, USA) baited with commercial cat food. Trapping was conducted during spring and summer (March-August), 2005-2006. During each trapping session 30 traps were placed within a 2.4km<sup>2</sup> grid composed of a 3 X 5 array of 400m X 400m cells, with 2 traps placed in each cell and maintained for 5-10 nights (Gehrt 2002). Captured raccoons were immobilized using an intramuscular injection of Telazol® (Fort Dodge Animal Health, Fort Dodge, Iowa; Gehrt et al. 2001). Immobilized raccoons were weighed, sexed, and placed into age classes (class I: 0-14 months, class II: 15-38 months, class III: 39-57 months, class IV: 58-86 months, class V: > 86months) based on patterns of tooth wear (Grau et al. 1970). Reproductive condition was determined by observing the length and coloration of teats for females and the size of testes in males (Sanderson and Nalbandov 1973). If adequate sedation was achieved a blood sample (ca. 5-10 ml) was collected from each raccoon via femoral or cephalic venipuncture. All raccoons and opossums were marked with 2 uniquely numbered ear tags (Monel #3, National Band and

Tag Company, Newport, Kentucky, USA). The Ohio State University Animal Care and Use Committee approved the above capture and handling methods (ILACUC#2003R0062).

### Sample Handling and Serological Testing

I placed blood samples into separator tubes, allowed them to clot, and then centrifuged and separated the samples. The serum was then removed from each sample and transferred to microcentrifuge tubes and frozen at -70°C. Serologic testing was performed at the University of Illinois Veterinary Diagnostic Laboratory (Champaign, Illinois, USA).

Canine distemper virus (CDV) and canine parvovirus (CPV) testing was performed using the TiterCHEK<sup>®</sup> CDV/CPV antibody test kit (Synbiotics, San Diego, California, USA). TiterCHEK<sup>®</sup> CDV/CPV is an enzyme-linked immunosorbent assay (ELISA) based test. Any reaction greater than the positive control or slightly weaker than the positive control was considered positive for exposure to CPV or CDV for each of the respective tests. Weak positive reactions are still considered a positive reaction for this test on raccoon sera (Dr. Federico Zuckermann, personal communication).

*Toxoplasma gondii* testing was performed using the indirect immuno-fluorescence (IFA) slide test, using ImmunoFA product No. 1207 toxoplasma slides (GenBio, San Diego, California, USA) and affinity purified fluorescein-labeled, goat anti-Raccoon IgG antibodies (Kirkegard & Perry Laboratories, Gaithersburg, Maryland, USA). A titer of  $\geq$  1:40 was considered positive for exposure to *T. gondii*.

Sera were tested for *Leptospira spp.* using the microscopic agglutination microtiter test (MAT) versus 7 serovars of *Leptospira*; *L. interrogans* serovars *autumnalis*, *bratislava*, *canicola*, *icterohaemorrhagiae*, *pomona*, and *L. kirschneri* serovar *grippotryphosa*, and *L. borgpeterseni* serovar *hardjo*. A titer  $\leq 1:50$  was considered negative, while a titer  $\geq 1:100$  was considered positive for exposure to *Leptospira*. Serial two-fold dilutions to 1:3200 were reported. The antigens were provided by the National Veterinary Service Laboratory in Ames, Iowa, USA.

Testing for pseudorabies virus was performed using a pseudorabies virus antibody test kit (Viral Antigens Inc., Memphis, Tennessee, USA), which is a latex agglutination test. A titer  $\geq 1:4$  was considered positive for exposure to pseudorabies.

Prevalence of exposure to each of the respective pathogens was calculated as the percent of individuals who were confirmed to be seropositive. I used logistic regression analysis to assess the association between risk factors and exposure to each of the pathogens. I considered the following risk factors: age (juvenile/yearling or adult), sex (male or female), year (2005 or 2006), land-use (urban open space, rural open space or urbanized). The urbanized land-use category contains cells that were either medium or high-density urban. Models were constructed using purposeful selection, a process based on biological importance and statistical significance (Hosmer and Lemeshow 2000). The steps I used for purposeful selection were as follows: 1) tested for univariate significance of each covariate ( $P < 0.15$ ), 2) fit a multivariate model containing all significant variables from the univariate analysis, 3) removed variables based on Wald statistic ( $P > 0.05$ ) and assessed the significance of the variable removed using a likelihood ratio test, 4) confounding was assessed for removed variables, if the coefficients of any remaining

risk factors changed >10%, the removed variable was considered to be a confounder and was retained in the model, and 5) tested for effect modification and included interaction terms that were both statistically significant ( $P < 0.05$ ) and biologically plausible.

## RESULTS

During July-August 2005, 4 urban open sites were sampled, and during April-August of 2006, 3 rural open, 4 urban open and 7 urbanized sites were sampled (Table 2.1, Figure 2.2). On 3,476 trap-nights, we captured 530 adult raccoons, 182 juvenile raccoons, 105 adult opossums and 80 cats (Table 2.2). Individuals with missing information (e.g. sex or age) were not included in analyses ( $n = 6$ ). Sera were collected from 570 raccoons, of which 302 were from urban open sites, 135 were from rural open sites and 133 were from urbanized sites. There were 130 age class I (juveniles/yearlings) individuals (64, 43 and 23 individuals from urbanized, rural open and urban open sites respectively), and 440 adults (279 from urban open sites, 92 from rural open sites, and 69 from urbanized sites). There were 304 males (152 from urban open sites, 88 from rural open sites, and 64 from urbanized sites), and 266 females (150 from urban open sites, 69 from urbanized sites, and 47 from rural open sites). Of the 570 sera collected, 98.6% of samples were tested for exposure to all 5 infectious agents (Table 3.1). Of the individuals not tested ( $n = 114$ ), 87.7% were juveniles, which were often too small to immobilize and collect adequate blood sample volumes. Seventy-nine percent of raccoons showed evidence of exposure to  $\geq 1$  of the infectious agents, and 52% were positive for  $\geq 2$  infectious agents. None of the samples were seropositive for exposure to pseudorabies virus.

Exposure to CDV was identified in 39.4% of all raccoons ( $n = 564$ ; Table 3.2). Overall, at urban open sites 40.8% of raccoons ( $n = 299$ ) were seropositive for CDV and seroprevalence at sites ranged from 18.2-76.9% (Table 3.2). Overall, 21.2% of raccoons ( $n = 132$ ) from urbanized sites were seropositive and seroprevalence at sites ranged from 3.6-84.6% (Table 3.2). Fifty-four percent of raccoons ( $n = 133$ ) from rural open sites were seropositive, and seroprevalence at individual sites ranged from 48.2-65.0% (Table 3.2).

The final regression equation for CDV was  $\text{Seropositive} = -1180.61 + 0.87 (\text{Rural Open}) - 0.55 (\text{Urbanized}) + 1.84 (\text{Adult}) + 0.58 (2006)$ ;  $G = 83.31$ , d.f. = 4,  $P < 0.0001$ . There were no significant interaction terms. In the final regression model for CDV, raccoons from rural open sites were more likely to be seropositive compared to raccoons from urban open sites (adjusted odds ratio (OR) = 2.39, 95% CI = 1.47-3.88,  $P < 0.001$ ), and raccoons from urbanized sites were less likely to be seropositive than urban open raccoons (OR = 0.57, 95% CI = 0.34-0.97,  $P = 0.04$ ). Adult raccoons were more likely to be seropositive than juveniles and yearlings (OR = 6.28, 95% CI = 3.50-11.27,  $P < 0.001$ ). Raccoons from urban open sites captured in 2006 were more likely to be seropositive than raccoons captured at urban open sites in 2005 (OR = 1.78, 95% = 1.04-3.07,  $P = 0.036$ ).

Fifty-one percent of raccoons ( $n = 564$ ) were seropositive for CPV. Overall, at urban open sites 53.8% of raccoons ( $n = 299$ ) were seropositive for CPV and seroprevalence at sites ranged from 9.1-69.2% (Table 3.3). Overall, 45.5% of raccoons ( $n = 132$ ) from urbanized sites were seropositive and seroprevalence at sites ranged from 24.0-60.0% (Table 3.3). Fifty percent of raccoons ( $n = 133$ ) from rural open sites were



seropositive, and seroprevalence at individual sites ranged from 39.3-62.5% (Table 3.3). The final regression equation for CPV was Seropositive = -1.10 + 1.45 (Adult);  $G = 46.55$ , d.f. = 1,  $P < 0.0001$ . In the final regression model for CPV, adult raccoons were more likely seropositive than juveniles and yearlings (OR = 4.27, 95% CI = 2.74-6.65,  $P < 0.001$ ).

Overall, 37.4% of raccoons ( $n = 565$ ) were seropositive for *T. gondii*. At urban open sites 44.7% of raccoons ( $n = 299$ ) were seropositive for *T. gondii* and seroprevalence at sites ranged from 20.0-68.2% (Table 3.4). Overall, 17.7% of raccoons ( $n = 130$ ) from urbanized sites were seropositive and seroprevalence at sites ranged from 0.0-33.3% (Table 3.4). Forty percent of raccoons ( $n = 135$ ) from rural open sites were seropositive, and seroprevalence at individual sites ranged from 35.1-46.0% (Table 3.4). The final regression equation for *T. gondii* was Seropositive = -2.18 + 0.99 (Urban Open) + 1.03 (Rural Open) + 1.04 (Adult);  $G = 47.45$ , d.f. = 3,  $P < 0.0001$ . In the final regression model for *T. gondii*, raccoons from urban open sites were more likely to be seropositive compared to raccoons from urbanized sites (adjusted odds ratio (OR) = 2.70, 95% CI = 1.59-4.58,  $P < 0.001$ ), similarly, raccoons from rural open sites were more likely to be seropositive than urbanized raccoons (OR = 2.81, 95% CI = 1.57-5.01,  $P < 0.001$ ). Adult raccoons were more likely to be seropositive than juveniles and yearlings (OR = 2.83, 95% CI = 1.66-4.81,  $P < 0.001$ ). There were no significant interaction terms.

Overall, 33% of raccoons ( $n = 569$ ) were seropositive for *Leptospira spp.* At urban open sites 37% of raccoons ( $n = 303$ ) were seropositive for *Leptospira spp.* and seroprevalence at sites ranged from 20-69% (Table 3.5). Overall, 27% of raccoons ( $n =$

131) from urbanized sites were seropositive and seroprevalence at sites ranged from 4-45% (Table 3.5). Thirty percent of raccoons ( $n = 135$ ) from rural open sites were seropositive, and seroprevalence at individual sites ranged from 25-35% (Table 3.5). The final regression equation for *Leptospira spp.* was Seropositive =  $-1.80 + 1.33$  (Adult);  $G = 29.37$ , d.f. = 1,  $P < 0.0001$ . In the final regression model for *Leptospira spp.*, adult raccoons were more likely seropositive than juveniles and yearlings (OR = 3.78, 95% CI = 2.22-6.46,  $P < 0.001$ ). *Grippotyphosa* was the most common reactive serovar (8 sites), followed by serovar *autumnalis* (3 sites), and *bratislava* (1 site); at the remaining sites, multiple serovars were equally prevalent in reactive samples (Table 3.6).

## DISCUSSION

Raccoons inhabiting all areas of this urban landscape were commonly exposed to the infectious agents I screened for, and exposure to multiple pathogens was also common. Therefore, humans, domestic animals and captive animal collections may be at risk for exposure to these pathogens via spillover from wildlife or environmental contamination. Although the presence of antibodies to a pathogen does not necessarily indicate past or present clinical disease, serological data is commonly used to quantify to quantify the exposure of individuals and populations to pathogens (Bigler et al. 1975, Mitchell et al. 1999, Junge et al. 2007).

CDV occurs worldwide and has a wide host range, with many canids, mustelids and procyonids being susceptible (Williams 2001). Mortality due to CDV is commonly observed in raccoon populations (Robinson et al. 1957, Hoff et al. 1974, Schinner and Cauley 1974, Roscoe 1993), however, its effects on raccoon populations are unclear

(Schubert et al. 1998). CDV can have significant impacts on species that are highly susceptible (e.g. gray fox [*Urocyon cinereoargenteus*]; Hoff et al. 1974) and has been implicated in the death of captive felids (Appel et al. 1994) and the near extirpation of the black-footed ferret (*Mustela nigripes*; Thorne and Williams 1988). Raccoons in urban areas may represent pathogen spillover risks to captive animal collections in zoological parks and unvaccinated domestic dogs (Junge et al. 2007).

Exposure to CDV appeared to be common, with an overall seroprevalence of 39.4%, however, there appeared to be differences in patterns of seroprevalence among sites and land-use types. Seroprevalence detected at my sites were within the range of previously published serologic surveys (23% to 54%; Mitchell et al. 1999, Junge et al. 2007). Positive titers to CDV were detected in all age classes in all land-use types (Figure 3.1) and adults were more likely to be exposed than juveniles and yearlings. These results were consistent with patterns observed in rural Illinois and suggested that there was frequent exposure to CDV in these areas (Mitchell et al. 1999). The higher seroprevalence at rural open and urban open sites suggested that raccoons in these areas have a higher risk of exposure to CDV than raccoons within the urbanized matrix. This may have been due to the lower mean raccoon density in urbanized sites relative to urban and rural open sites (Figure 2.4). The higher risk of exposure rural open sites relative to urban open sites was unexpected because rural open sites generally lacked the aggregated anthropogenic food resources that were prevalent at urban open sites (Graser personal observation). The differences in seroprevalence may have been a result of variation in host social behavior and contact rate. Alternatively, the lower seroprevalence in the urbanized and urban open sites could indicate that the strain of CDV present at urban

sites was a more virulent strain of CDV than at the rural sites. Genetically differing lineages of CDV have been observed in a population of raccoons in Cook County, Illinois, and evidence suggested differing levels of virulence (Lednicky et al. 2004).

The recent development of proximity detecting radiocollars has offered new insights into the social organization and contact rates of raccoons at an urban park (Prange et al. 2006). Deployment of these devices in raccoon populations within rural areas and the highly developed urban matrix could yield valuable data regarding differences in contact rate and sociality, with obvious implications for diseases such as canine distemper and rabies, which require close contact for transmission. Although long-term serological data would provide additional insight, my results represent a comprehensive baseline serological dataset to be expanded upon and suggest differences in the epizootiology of CDV across an urban landscape.

Species from 4 families in order Carnivora (Felidae, Canidae, Procyonidae, and Mustelidae) are susceptible to parvoviruses of the feline parvovirus subgroup, and parvovirus infection is one of the most important infectious diseases of raccoons (Barker and Parrish 2001). Although samples were tested for CPV, significant antigenic cross-reactions occur among the parvovirus agents, therefore, antibodies to a particular parvovirus may not reflect exposure to that specific virus (Barker and Parrish 2001).

In this study, exposure to CPV and possibly other parvoviruses, was common in raccoons across land-use types (> 50%), and seroprevalence was high with few exceptions (Table 3.3). Seroprevalence did not differ by land-use, however, adult raccoons were more likely seropositive than juveniles and yearlings, and this pattern generally remained consistent across adult age classes (Figure 3.2). In an urban Missouri

raccoon population seroprevalence of feline parvovirus was 49.7%, with juveniles tending to have higher prevalence ( $n = 159$ ) over a 5-year period (Junge et al. 2007). However, seroprevalence was 1.7% in a rural Tennessee population ( $n = 117$ ; Rabinowitz and Potgieter 1984). Transmission of parvovirus is thought to be via the fecal-oral route, most likely through the ingestion of the virus from the environment rather than through direct contact (Reif 1976). Because the virus may survive long periods in the environment, wild carnivores can be exposed at marking sites, latrines and other areas contaminated by the feces of virus-shedding individuals (Barker and Parrish 2001). Once parvovirus is endemic in a metapopulation of susceptible species, epidemics are likely to have only transient local impacts, because naïve populations will likely be small and scattered (Barker and Parrish 2001). My results indicated that CPV is likely enzootic in raccoon subpopulations in this study area, and that raccoons are frequently exposed. The high prevalence of exposure to CPV suggested that raccoons may represent a significant hazard to captive carnivores and potentially unvaccinated pets in urbanized and rural areas.

*T. gondii* is a parasitic protozoan, and is the causative agent of toxoplasmosis, an important zoonotic disease. In immunocompetent individuals, *T. gondii* infections are generally asymptomatic; however, results can be devastating in congenitally infected children and individuals with suppressed immune systems (Hill and Dubey 2002). Although numerous species can be infected with *T. gondii*, only felids are known to shed the environmentally resistant oocysts of *T. gondii* (Dubey et al. 1993). Raccoons are omnivores, and can become infected with *T. gondii* by ingesting tissue cysts in prey or by ingesting oocysts from the environment; therefore, they are good indicators of the

prevalence *T. gondii* in the environment (Hill and Dubey 2002, Hancock et al. 2005).

The presence of antibodies to *T. gondii* in a serum sample can only be considered evidence that a host has been infected at some point in the past (Hill and Dubey 2002).

The seroprevalence of *T. gondii* observed in this study is within the lower range of reported values for raccoon populations (15%; Hill et al., 49%; Mitchell et al., 84%; Hancock et al. 2005). Adult raccoons were more likely to have been exposed to *T. gondii* than juveniles and yearlings, similar patterns were observed in rural Illinois (Mitchell et al. 1999). Seroprevalence was expected to be higher at urbanized sites, where 76% of all cats were captured during the study (Table 2.2), however, raccoons captured at urban and rural open sites were more likely to be seropositive for *T. gondii* than raccoons captured at urbanized sites. The higher exposure of raccoons in urban and rural open sites relative to urbanized sites may have reflected differences in the health and hygiene of the cats present in the respective areas and differences in foraging behavior of raccoons in these areas. Cats present in the urban and rural open sites were most likely feral; and consumption of natural foods (e.g. birds and rodents) would increase their likelihood of infection by consuming the tissues of intermediate hosts (Hill and Dubey 2002). The lack of access to litter boxes would mean individuals that shed oocysts would contaminate the environment with feces, further increasing the likelihood that prey items would become infected. Additionally, raccoons in rural and urban open sites are more likely to consume natural prey items than raccoons in urbanized residential areas, which would increase the likelihood of consuming infected intermediate hosts.

The possibility of increased risk of exposure to *T. gondii* in urban and rural open spaces has implications for humans residing near these areas. Cat owners should keep

their pets confined to their home to limit their consumption of potentially infected prey items, especially if the owner is immunocompromised or pregnant. Additionally, individuals with outdoor gardens should wash vegetables thoroughly because of potential contamination with cat feces (Hill and Dubey 2002).

*Leptospira spp.* are the causative agent of leptospirosis, a potentially zoonotic disease. *Leptospira spp.* occur globally in numerous mammalian hosts. Generally, “maintenance hosts” are highly susceptible to infection, but typically do not develop severe clinical disease; whereas “accidental hosts” are less susceptible to infection, but can suffer severe clinical disease when infected (Leighton and Kuiken 2001).

Leptospirosis is re-emerging as an important disease in domestic dogs, with recent evidence suggesting that different serovars are becoming more prevalent than in the past (McDonough 2001). Human infections can result from contacting water contaminated with *Leptospira spp.*, at times resulting in severe disease (CDC 1998). Raccoons can be a reservoir species for *Leptospira spp.*, and are considered to be the maintenance host for serovar *grippityphosa* (Leighton and Kuiken 2001). Due to immunologic cross-reactivity among serovars, serological results alone are best interpreted as a qualitative index of exposure to some form of *Leptospira spp.* (Leighton and Kuiken 2001).

In this study exposure to *Leptospira spp.* was common, with 33% of samples reacting serologically; however, there was substantial variation in seroprevalence at individual sites. Adults were more likely to be exposed than juveniles and yearlings. Similar results have been reported in serological surveys of other raccoon populations. Seroprevalence was 48% ( $n = 459$ ) and adult raccoons were more likely to be exposed than juveniles in a rural Illinois population (Mitchell et al. 1999). In suburban

Connecticut, 36% of raccoons tested showed evidence of exposure (Richardson and Gauthier 2003). At sites in this study where the predominant reacting serovar could be determined, serovar *grippotyphosa* was most common. In rural Illinois, the primary serovar detected was *grippotyphosa*, and this was also the case in an urban Missouri raccoon population (Mitchell et al. 1999, Junge et al. 2007).

The evidence that exposure to *Leptospira spp.* was common in raccoons inhabiting rural open, urban open and urbanized sites indicated that there is a potential risk of spillover from raccoons into domestic pets, humans and other wildlife species. Contaminated water is thought to be the most prevalent source of exposure for dogs (McDonough 2001). Dogs can be a potential source of infection for humans; therefore, pet owners should prevent dogs from drinking any outdoor water source that may be contaminated with raccoon urine, including outdoor water dishes. Humans should also limit their exposure to water sources that may be contaminated with the spirochete and limit their contact with potentially infected wildlife. It would be valuable to conduct future research on the prevalence of *Leptospira spp.* in urban parklands and residential areas that consider other peridomestic species in addition to the raccoon. Furthermore, it would be useful to collect and culture urine samples to isolate and identify the infecting serovars.

The absence of raccoons testing positive for exposure to pseudorabies virus (PRV) was consistent with existing knowledge of the epidemiology of this disease. In a rural Illinois raccoon population, 17% ( $n = 479$ ) were seropositive for PRV, however there was a PRV positive swine farm near their study area (Mitchell et al. 1999). Most evidence suggests that the raccoon is a “dead-end” host with regards to PRV, as such



PRV in raccoons has never been reported without a concurrent infection in swine nearby (Thawley and Wright 1982). My findings were consistent with the fact that there are no known swine farms in Cook County, and while there are active swine operations in McHenry County, I did not observe any in the proximity of my study sites (USDA 2004, Graser personal observation).

My findings indicated that raccoons inhabiting all areas of this urban landscape were commonly exposed to the infectious agents I screened for, some of which may pose risks to other wildlife species, humans, and to domestic and captive animal populations. Furthermore, the prevalence of exposure to certain infectious agents (CDV and *T. gondii*) differed across the landscape, therefore, the risks to humans and domestic species may be variable in different portions of the landscape. The relationship between differences of exposure risk and land-use differed among the pathogens. These relationships warrant further investigation due to the potential impacts on the health and safety of humans and domestic animals. There also exists a need for a better understanding of the role that other wildlife species, and feral and domestic animals play in ecology and dynamics of wildlife diseases. With increasing human presence and urbanization, understanding how human-induced changes to the landscape alter the distribution and abundance of species, resource distribution and disease ecology will be critical to managing wildlife and mitigating disease related threats.

## LITERATURE CITED

- Alig, R. J., J. D. Kline, M. Lichtenstein. 2004. Urbanization on the US landscape: looking ahead in the 21<sup>st</sup> century. *Landscape and Urban Planning* 69: 219-234.
- Appel, M. J., R. A. Yates, G. L. Foley, J. J. Bernstein, S. Santinelli, L. H. Spelman, L. D. Miller, L. H. Arp, M. Anderson, M. Barr, S. Pearce-Kelling, and B. A. Summers. 1994. Canine distemper epizootic in lions, tigers, and leopards in North America. *Journal of Veterinary Diagnostic Investigation* 6: 277-288.
- Barker, I. K., and C. R. Parrish. 2001. Parvovirus infections Pp. 131-146 in *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa.
- Bigler, W. J., J. H. Jenkins, P. M. Cumbie, G. L. Hoff, and E. C. Prather. 1975. Wildlife and environmental health: Raccoons as indicators of zoonoses and pollutants in southeastern United States. *Journal of the American Veterinary Medical Association* 167: 592-597.
- Bozek, C. K., S. Prange, and S. D. Gehrt. 2007. The influence of anthropogenic resources on multi-scale habitat selection by raccoons. *Urban Ecosystems* 10: 413-425.
- Bradley, C. A., and S. Altizer. 2006. Urbanization and the ecology of wildlife diseases. *Trends in Ecology and Evolution* 22: 95-102.
- Center for Disease Control. 1998. Update: leptospirosis and unexplained acute febrile illness among athletes participating in triathlons-Illinois and Wisconsin. *MMWR* 47: 673-676.
- Chicago Metropolitan Agency for Planning. 2006. Data Bulletin: 2001 Land-use inventory for northeastern Illinois. Chicago Metropolitan Agency for Planning, Chicago, Illinois.
- Crooks, K. R. 2002. Relative sensitivities of mammalian carnivores to habitat fragmentation. *Conservation Biology* 16: 488-502.

- Dubey, J. P., A. N. Hamir, S. K. Shen, P. Thulliez, and C. E. Rupprecht. 1993. Experimental *Toxoplasma gondii* infection in raccoons (*Procyon lotor*). *Journal of Parasitology* 79: 548-552.
- Fritzell, E. K. 1978. Aspects of raccoon (*Procyon lotor*) social organization. *Canadian Journal of Zoology* 56: 260-71.
- Gehrt, S. D., L. L. Hungerford and S. Hatten. 2001. Drug effects on recaptures of raccoons. *Wildlife Society Bulletin* 29: 833-837.
- Gehrt, S. D. 2002. Evaluation of spotlight and road-kill surveys as indicators of local raccoon abundance. *Wildlife Society Bulletin* 30: 449-456.
- Gehrt, S. D. 2003. Raccoons and allies. In *Wild mammals of North America: biology, management, and conservation* (G. A. Feldhamer, B. C. Thompson, and J.A. Chapman, eds.). 2nd ed. Johns Hopkins University Press, Baltimore, Maryland, pp. 611-633 .
- Grau, G. A., G. C. Sanderson, and J. P. Rogers. 1970. Age determination of raccoons. *Journal of Wildlife Management* 34: 364-372.
- Grimm, N. B., S. H. Faeth, N. E. Golubiewski, C. L. Redman, J. Wu, X. Bai, and J. M. Briggs. 2008. Global change and the ecology of cities. *Science* 319: 756-760.
- Hancock, K., L. A. Thiele, A. M. Zajac, F. Elvinger, and D. S. Lindsay. 2005. Prevalence of antibodies to *Toxoplasma gondii* in raccoons (*Procyon lotor*) from an urban area of Northern Virginia. *Journal of Parasitology* 91: 694-695.
- Hill, D., and J. P. Dubey. 2002. *Toxoplasma gondii*: transmission, diagnosis and prevention. *Clinical Microbiology and Infection* 8: 634-640.
- Hill, R. E., J. E. Zimmerman, R. W. Willis, S. Patton, and W. R. Clark. 1998. Seroprevalence of antibodies against *Toxoplasma gondii* in free ranging mammals in Iowa. *Journal of Wildlife Diseases* 34: 811-815.
- Hoff, G. L., W. J. Bigler, S. J. Proctor, and L. P. Stallings. 1974. Epizootic of canine distemper virus infection among urban raccoons and gray foxes. *Journal of Wildlife Diseases* 10: 423-428.
- Hoffman, C. O., and J. L. Gottschang. 1977. Numbers, distribution, and movements of a raccoon population in a suburban residential community. *Journal of Mammalogy* 58: 623-636.
- Hosmer, D. W., and S. Lemeshow. 2000. *Applied logistic regression*. John Wiley & Sons, New York.

- Jacobson, J. E., K. R. Kazacos, and F. H. Montague. 1982. Prevalence of eggs of *Baylisascaris procyonis* (Nematoda:Ascarioidea) in raccoon scats from and urban and rural community. *Journal of Wildlife Disease* 18: 461-464.
- Junge, R. E., K. Bauman, M. King, and M. E. Gompper. 2007. A serologic assessment of exposure to viral pathogens and *Leptospira* in an urban raccoon (*Procyon lotor*) population inhabiting a large zoological park. *Journal of Zoo and Wildlife Medicine* 38: 18-26.
- Kaufman, J. H. 1982. Raccoons and allies. In *Wild Mammals of North America: biology, management, and economics* (J. A. Chapman and G. A. Feldhamer, eds.). Johns Hopkins University Press, Baltimore, Maryland, pp. 567-585.
- Lednicky, J. A., J. Dubach, M. J. Kinsel, T. P. Meehan, M. Bocchetta, L. L. Hungerford, N. A. Sarich, K. E. Witecki, M. D. Braid, C. Pedrak, and C. M. Houde. 2004. Genetically distant American canine distemper virus lineages have recently caused epizootics with somewhat different characteristics in raccoons living around a large suburban zoo in the USA. *Virology Journal* 1: 1-14.
- Leighton, F. A., and T. Kuiken. 2001. Leptospirosis. In *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa, pp. 498-502.
- LoGiudice, K. 2003. Trophically transmitted parasites and the conservation of small populations: raccoon roundworm and the imperiled Allegheny woodrat. *Conservation Biology* 17: 258-266.
- McDonough, P. L. 2001. Leptospirosis in dogs-current status. In: Carmichael, L. ed. *Recent Advances in Canine Infectious Diseases*. International Veterinary Information Service, Ithaca, New York.
- McKinney, M. L. 2002. Urbanization, biodiversity, and conservation. *Bioscience* 52: 883-890.
- McKinney, M. L. 2006. Urbanization as a major cause of biotic homogenization. *Biological Conservation* 127: 247-260.
- Mech, L. D., D. M. Barnes, and J. R. Tester. 1968. Seasonal weight changes, mortality, and population structure of raccoons in Minnesota. *Journal of Mammalogy* 49: 63-73.
- Mitchell, M. A., L. L. Hungerford, C. Nixon, T. Esker, J. Sullivan, R. Koerkenmeier, and J. P. Dubey. 1999. Serologic survey for selected infectious disease agents in raccoons from Illinois. *Journal of Wildlife Diseases* 35: 347-355.

- National Oceanic and Atmospheric Administration (NOAA). 2002. Climatography of the United States No. 81: Monthly normals of temperature, precipitation, and heating and cooling degree days 1971-2000. National Climatic Data Center/NESDIS/NOAA, Asheville, North Carolina.
- Northeastern Illinois Planning Commission. 2006. 2030 forecasts of population, households and employment by county and municipality. Northeastern Illinois Planning Commission, Chicago, Illinois.
- Page, L. K., S. D. Gehrt, and N. P. Robinson. 2008. Land-use effects on prevalence of raccoon roundworm (*Baylisascaris procyonis*). *Journal of Wildlife Diseases* 44: 594-599.
- Patz, J. A., P. Daszak, G. M. Tabor, A. A. Aquirre, M. Pearl, J. Epstein, N. D. Wolfe, A. M. Kilpatrick, J. Fofopoulos, D. Molyneux, D. J. Bradley et al. 2004. Unhealthy landscapes: policy recommendations on land use change and infectious disease emergence. *Environmental Health Perspectives* 112: 1092-1098.
- Prange, S., T. Jordan, C. Hunter, and S.D. Gehrt. 2006. New radiocollars for the detection of proximity among individuals. *Wildlife Society Bulletin* 34: 1333-1344.
- Prange, S., S. D. Gehrt, and E. P. Wiggers. 2003. Demographic factors contributing to high raccoon densities in urban landscapes. *Journal of Wildlife Management* 67: 324-333.
- Prange, S., and S. D. Gehrt. 2004. Changes in mesopredator-community structure in response to urbanization. *Canadian Journal of Zoology* 82: 1804-1817.
- Rabinowitz, A. R., and L. N. D. Potgieter. 1984. Serologic survey for selected viruses in a population of raccoons, *Procyon lotor* (L.), in the Great Smoky Mountains. *Journal of Wildlife Diseases* 20: 146-148.
- Reif, J. S. 1976. Seasonality, natality and herd immunity in feline panleukopenia. *American Journal of Epidemiology* 103: 81-87.
- Richardson, D. J., and J. L. Gauthier. 2003. A serosurvey of leptospirosis in Connecticut peridomestic wildlife. *Vector-Borne and Zoonotic Diseases* 3: 187-193.
- Riley, S. P. D., J. Hadidian, and D. A. Manski. 1998. Population density, survival, and rabies in raccoons in an urban national park. *Canadian Journal of Zoology* 76: 1153-1164.

- Robinson, V. B., J. W. Newberne, and D. M. Brooks. 1957. Distemper in the American raccoon (*Procyon lotor*). *Journal of the American Veterinary Medical Association* 131: 276-278.
- Rosatte, R. C., M. J. Power, and C. D. MacInnes. 1991. Ecology of urban skunks, raccoons and foxes in metropolitan Toronto. In *Wildlife conservation in metropolitan environments*. Edited by L. W. Adams and D. L. Leedy. National Institute for Urban Wildlife, Columbia, Maryland, pp. 31-38.
- Roscoe, D. E. 1993. Epizootiology of canine distemper in New Jersey raccoons. *Journal of Wildlife Diseases* 29: 390-395.
- Sanderson, G. C. 1987. Raccoon. Pages 487-99 in M. Novak, J.A. Baker, M. E. Obbard, and B. Malloch, eds. *Wild furbearer management and conservation in North America*. Ontario Trappers Association, North Bay, Canada.
- Schinner, J. R., and Cauley, D. L. 1974. The ecology of urban raccoons in Cincinnati, Ohio. In *Wildlife in an urbanizing environment*. Edited by J. H. Noyes and D. R. Progulske. Planning and Resource Development Series No. 28, Holdsworth Natural Resources Center, Amherst, Massachusetts, pp. 125-130.
- Schmidt, K. A., and R. S. Ostfeld. 2001. Biodiversity and the dilution effect in disease ecology. *Ecology* 82: 609-619.
- Schubert, C. A., I. A. Barker, R. C. Rosatte, C. D. MacInnes, and T. D. Nudds. 1998. Effect of canine distemper on an urban raccoon population: An experiment. *Ecological Applications* 8: 379-387.
- Sorvillo, F., L. R. Ash, O. G. W. Berlin, J. Yatabe, C. Degiorgio, and S. A. Morse. 2002. *Baylisascaris procyonis*: An emerging helminthic zoonosis. *Emerging Infectious Diseases* 8: 355-359.
- Stallnecht, D. E., and E. W. Howerth. 2001. Pseudorabies (Aujeszky's Disease) In *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa, pp. 164-170.
- StataCorp. 2005. *Stata statistical software: release 9*. College Station, TX: StataCorp LP.
- Thawley, D. G., and J. C. Wright. 1982. Pseudorabies virus infection in raccoons: a review. *Journal of Wildlife Diseases* 18: 113-116.
- Thorne, E. T., and E. S. Williams. 1988. Disease and endangered species: the black-footed ferret as a recent example. *Conservation Biology* 2: 66-74.

- Twichell, A. R., and H. H. Dill. 1949. One hundred raccoons from one hundred and two acres. *Journal of Mammalogy* 30: 130-133.
- U.S. Census Bureau. 1995. Population of counties by census: 1900 to 1990. Population Division, U.S. Bureau of the Census, Washington D.C., USA.
- U.S. Census Bureau. 2001. Statistical abstract of the United States: 2001, 112<sup>th</sup> edition. U.S. Bureau of the Census, Washington D.C., USA.
- U.S. Census Bureau. 2006a. Statistical abstract of the United States: 2006, 125<sup>th</sup> edition. U.S. Bureau of the Census, Washington D.C., USA.
- U.S. Census Bureau. 2006b. State and county QuickFacts. U.S. Bureau of the Census, Washington D.C., USA.
- USDA. 2004. Illinois Agricultural Statistics: County Highlights. [accessed 8/20/08 [www.agstats.state.il.us/annual/2004/](http://www.agstats.state.il.us/annual/2004/)].
- 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.
- Williams, E. S. 2001. Canine distemper. In *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa, pp. 50-59.
- Woolhouse, M. E., and S. Gowtage-Sequeria. 2005. Host range and emerging and reemerging pathogens. *Emerging Infectious Diseases* 11: 1842-1847.
- Wright, A. N., and M. E. Gompper. 2005. Altered parasite assemblages in raccoons in response to manipulated resource availability. *Oecologia* 144: 148-156.
- Yeager, L. E., W. H. Elder. 1945. Pre-and post-hunting season foods of raccoons on an Illinois goose refuge. *Journal of Wildlife Management* 9: 48-56.

Age Class	<u>Number of Serological Tests</u>				
	0	2	3	4	5
Juv/I	103	1	1	1	56
II	5	0	1	1	206
III	6	0	0	3	150
IV	0	0	0	0	70
V	0	0	0	0	9
Total	114	1	2	5	562

Table 3.1. The number of serological tests performed on sera collected from raccoons in Cook County and McHenry County, northeastern, Illinois, USA, 2005-2006.



Site	Land use	n	Percent seropositive (number tested) for CDV				
			Juv/Yr1	Adult	Male	Female	Total
17-13 <sup>a</sup>	Urban Open	13	0 (0)	76.9 (13)	80.0 (5)	75.0 (8)	76.9
23-6 <sup>a</sup>	Urban Open	11	0 (3)	50.0 (8)	60.0 (5)	16.7 (6)	36.4
23-11 <sup>a</sup>	Urban Open	15	0 (0)	26.7 (15)	30.0 (10)	20.0 (5)	26.7
30-8 <sup>a</sup>	Urban Open	44	0 (3)	19.5 (41)	18.2 (22)	18.2 (22)	18.2
19-15 <sup>b</sup>	Urban Open	67	0 (9)	31.0 (58)	37.0 (27)	20.0 (40)	26.9
19-19 <sup>b</sup>	Urban Open	43	0 (2)	29.3 (41)	26.1 (23)	30.0 (20)	27.9
20-30 <sup>b</sup>	Urban Open	42	75.0 (4)	71.0 (38)	69.6 (23)	73.7 (19)	71.4
28-3 <sup>b</sup>	Urban Open	64	100 (1)	55.6 (63)	51.4 (35)	62.1 (29)	56.3
	Total	299	18.2 (22)	42.6 (277)	42.7 (150)	38.9 (149)	40.8
15-33 <sup>b</sup>	Urbanized	38	0 (18)	25.0 (20)	15.8 (19)	10.6 (19)	13.2
18-28 <sup>b</sup>	Urbanized	8	0 (6)	50.0 (2)	0 (4)	25.0 (4)	12.5
19-22 <sup>b</sup>	Urbanized	13	0 (2)	90.9 (11)	100 (6)	71.4 (7)	84.6
20-25 <sup>b</sup>	Urbanized	10	0 (4)	33.3 (6)	22.2 (9)	0 (1)	20.0
24-16 <sup>b</sup>	Urbanized	25	9.1 (22)	100 (3)	22.2 (9)	18.8 (16)	20.0
26-17 <sup>b</sup>	Urbanized	28	0 (11)	5.9 (17)	0 (12)	6.3 (16)	3.6
27-14 <sup>b</sup>	Urbanized	10	0 (0)	30.0 (10)	75.0 (4)	0 (6)	30.0
	Total	132	4.8 (63)	36.2 (69)	25.4 (63)	17.4 (69)	21.2
Coral Woods <sup>b</sup>	Rural Open	37	31.3 (16)	66.7 (21)	68.0 (25)	16.7 (12)	51.4
Marengo Ridge <sup>b</sup>	Rural Open	40	37.5 (8)	71.9 (32)	69.0 (29)	54.6 (11)	65.0
Rush Creek <sup>b</sup>	Rural Open	56	15.8 (19)	64.9 (37)	46.9 (32)	42.9 (24)	48.2
	Total	133	25.6 (43)	67.8 (90)	60.5 (86)	42.6 (47)	54.1
	All Sites	564	14.1 (128)	46.8 (436)	44.2 (299)	34.0 (265)	39.4

<sup>a</sup> sampled in 2005

<sup>b</sup> sampled in 2006

Table 3.2. Seroprevalence of antibodies against canine distemper virus (CDV) among raccoons sampled at 18 urban open, rural open, and urbanized sites in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006. The total seroprevalence column represents the entire sample from each site, site type and overall sample, while other columns give the seroprevalence for particular classes of individuals.

Site	Land use	n	Percent seropositive (number tested) for CPV				Total
			Juv/Yrl	Adult	Male	Female	
17-13 <sup>a</sup>	Urban Open	13	0 (0)	69.2 (13)	80.0 (4)	62.5 (5)	69.2
23-6 <sup>a</sup>	Urban Open	11	0 (3)	12.5 (8)	0 (0)	16.7 (1)	9.1
23-11 <sup>a</sup>	Urban Open	15	0 (0)	46.7 (15)	40.0 (10)	60.0 (5)	46.7
30-8 <sup>a</sup>	Urban Open	44	0 (3)	73.2 (41)	77.3 (22)	59.1 (22)	68.2
19-15 <sup>b</sup>	Urban Open	67	44.4 (9)	51.7 (58)	40.7 (27)	57.5 (40)	50.7
19-19 <sup>b</sup>	Urban Open	43	50.0 (2)	68.3 (41)	69.6 (23)	65.0 (40)	67.4
20-30 <sup>b</sup>	Urban Open	42	0 (4)	60.5 (38)	60.9 (23)	44.4 (20)	54.8
28-3 <sup>b</sup>	Urban Open	64	0 (1)	44.4 (63)	34.3 (35)	55.2 (29)	43.8
	Total	299	22.7 (22)	56.3 (277)	52.7 (150)	55.0 (149)	53.8
15-33 <sup>b</sup>	Urbanized	38	0 (18)	85.0 (20)	42.1 (19)	47.4 (19)	44.7
18-28 <sup>b</sup>	Urbanized	8	33.3 (6)	50.0 (2)	50 (4)	25.0 (4)	37.5
19-22 <sup>b</sup>	Urbanized	13	100 (2)	45.5 (11)	16.6 (6)	85.7 (7)	53.9
20-25 <sup>b</sup>	Urbanized	10	75.0 (4)	50.0 (6)	55.6 (9)	100 (1)	60.0
24-16 <sup>b</sup>	Urbanized	25	22.7 (22)	33.3 (3)	22.2 (9)	25.0 (16)	24.0
26-17 <sup>b</sup>	Urbanized	28	54.5 (11)	52.9 (17)	41.7 (12)	62.5 (16)	53.6
27-14 <sup>b</sup>	Urbanized	10	0 (0)	60.0 (10)	50.0 (4)	66.7 (6)	60.0
	Total	132	28.6 (63)	60.9 (69)	39.7 (63)	50.7 (69)	45.5
Coral Woods <sup>b</sup>	Rural Open	37	18.8 (16)	81.0 (21)	52.0 (25)	58.3 (12)	54.1
Marengo Ridge <sup>b</sup>	Rural Open	40	62.5 (8)	62.5 (32)	65.5 (29)	54.5 (11)	62.5
Rush Creek <sup>b</sup>	Rural Open	56	5.3 (19)	56.8 (37)	40.6 (32)	37.5 (24)	39.3
	Total	133	20.9 (43)	64.4 (90)	52.3 (86)	46.8 (47)	50.4
	All Sites	564	25.0 (128)	58.7 (436)	49.8 (299)	52.5 (265)	51.1

<sup>a</sup> sampled in 2005

<sup>b</sup> sampled in 2006

Table 3.3. Seroprevalence of antibodies against canine parvovirus (CPV) among raccoons sampled at 18 urban open, rural open, and urbanized sites in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006. The total seroprevalence column represents the entire sample from each site, site type and overall sample, while other columns give the seroprevalence for particular classes of individuals.

Site	Land use	n	Percent seropositive (number tested) for <i>T. gondii</i>				Total
			Juv/Yrl	Adult	Male	Female	
17-13 <sup>a</sup>	Urban Open	12	0 (0)	25.0 (12)	0 (5)	42.9 (7)	25.0
23-6 <sup>a</sup>	Urban Open	11	0 (3)	37.5 (8)	40.0 (5)	16.7 (6)	23.3
23-11 <sup>a</sup>	Urban Open	15	0 (0)	20.0 (15)	20.0 (10)	20 (5)	20.0
30-8 <sup>a</sup>	Urban Open	44	33.3 (3)	70.7 (41)	72.7 (22)	63.6 (22)	68.2
19-15 <sup>b</sup>	Urban Open	68	10.0 (10)	39.7 (58)	50.0 (28)	25.0 (40)	35.3
19-19 <sup>b</sup>	Urban Open	43	0 (2)	43.9 (41)	34.8 (23)	50.0 (20)	41.9
20-30 <sup>b</sup>	Urban Open	42	50.0 (4)	48.7 (39)	39.1 (23)	60.0 (20)	48.8
28-3 <sup>b</sup>	Urban Open	64	0 (1)	50.8 (63)	41.7 (36)	62.1 (29)	50.8
	Total	300	17.4 (23)	46.9 (277)	43.0 (151)	46.3 (149)	44.7
15-33 <sup>b</sup>	Urbanized	36	12.5 (16)	35.0 (20)	16.7 (18)	33.3 (18)	25.0
18-28 <sup>b</sup>	Urbanized	8	0 (6)	50.0 (2)	0 (4)	25.0 (4)	12.5
19-22 <sup>b</sup>	Urbanized	13	0 (2)	18.2 (11)	16.7 (6)	14.3 (7)	15.4
20-25 <sup>b</sup>	Urbanized	10	0 (4)	16.7 (6)	11.1 (9)	0 (1)	10.0
24-16 <sup>b</sup>	Urbanized	25	9.1 (22)	33.3 (3)	10.0 (10)	13.3 (15)	12.0
26-17 <sup>b</sup>	Urbanized	28	0 (11)	11.8 (17)	8.3 (12)	6.3 (16)	7.1
27-14 <sup>b</sup>	Urbanized	10	0 (0)	50.0 (10)	75.0 (4)	33.3 (6)	50.0
	Total	130	6.6 (61)	27.5 (69)	15.9 (63)	19.4 (67)	17.7
Coral Woods <sup>b</sup>	Rural Open	37	43.7 (16)	47.6 (21)	64.0 (25)	8.3 (12)	46.0
Marengo Ridge <sup>b</sup>	Rural Open	41	12.5 (8)	48.5 (33)	40.0 (30)	45.5 (11)	41.5
Rush Creek <sup>b</sup>	Rural Open	57	31.6 (19)	66.7 (38)	42.4 (33)	25.0 (24)	35.1
	Total	135	32.6 (43)	43.5 (92)	47.7 (88)	25.5 (47)	40.0
	All Sites	565	17.3 (127)	43.6 (438)	38.7 (302)	35.7 (263)	37.4

<sup>a</sup> sampled in 2005

<sup>b</sup> sampled in 2006

Table 3.4. Seroprevalence of antibodies against *Toxoplasma gondii* (*T. gondii*) among raccoons sampled at 18 urban open, rural open, and urbanized sites in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006. The total seroprevalence column represents the entire sample from each site, site type and overall sample, while other columns give the seroprevalence for particular classes of individuals.

Site	Land use	n	Percent Seropositive (number tested) for <i>Leptospira spp.</i>				
			Juv/Yrl	Adult	Male	Female	Total
17-13 <sup>a</sup>	Urban Open	13	0 (0)	69.2 (13)	80.0 (5)	62.5 (8)	69.2
23-6 <sup>a</sup>	Urban Open	11	33.3 (3)	50.0 (8)	60.0 (5)	33.3 (6)	45.5
23-11 <sup>a</sup>	Urban Open	15	0 (0)	40.0 (15)	40.0 (10)	40.0 (5)	40.0
30-8 <sup>a</sup>	Urban Open	44	33.3 (3)	39.0 (41)	45.6 (22)	31.8 (22)	38.6
19-15 <sup>b</sup>	Urban Open	68	33.3 (9)	30.5 (59)	17.9 (28)	40.0 (40)	30.9
19-19 <sup>b</sup>	Urban Open	43	0 (2)	29.3 (41)	26.1 (23)	30.0 (20)	27.9
20-30 <sup>b</sup>	Urban Open	44	0 (4)	22.5 (40)	26.1 (23)	14.3 (21)	20.5
28-3 <sup>b</sup>	Urban Open	64	100 (1)	48.4 (64)	50.0 (36)	48.3 (29)	49.2
	Total	303	27.3 (22)	37.4 (281)	36.8 (152)	36.4 (151)	36.6
15-33 <sup>b</sup>	Urbanized	37	0 (17)	85.0 (20)	44.4 (18)	47.4 (19)	44.7
18-28 <sup>b</sup>	Urbanized	8	0 (6)	50.0 (2)	0 (4)	25.0 (4)	12.5
19-22 <sup>b</sup>	Urbanized	13	0 (2)	18.2 (11)	0 (6)	28.6 (7)	15.4
20-25 <sup>b</sup>	Urbanized	10	25.0 (4)	33.3 (6)	33.3 (9)	0 (1)	30.0
24-16 <sup>b</sup>	Urbanized	25	0 (22)	33.3 (3)	0 (10)	6.7 (15)	4.0
26-17 <sup>b</sup>	Urbanized	28	18.2 (11)	41.2 (17)	33.3 (12)	31.3 (16)	32.1
27-14 <sup>b</sup>	Urbanized	10	0 (0)	30.0 (10)	0 (4)	50.0 (6)	30.0
	Total	131	4.8 (62)	47.8 (69)	23.8 (63)	30.9 (68)	27.5
Coral Woods <sup>b</sup>	Rural Open	37	31.3 (16)	38.1 (21)	32.0 (25)	41.7 (12)	35.1
Marengo Ridge <sup>b</sup>	Rural Open	41	12.5 (8)	39.4 (33)	40.0 (30)	18.2 (11)	34.2
Rush Creek <sup>b</sup>	Rural Open	57	15.8 (19)	28.9 (38)	21.1 (33)	29.2 (24)	24.6
	Total	135	20.9 (43)	34.8 (92)	30.7 (88)	29.8 (47)	30.4
	All Sites	569	14.2 (127)	38.5 (442)	32.4 (303)	33.8 (266)	33.0

<sup>a</sup> sampled in 2005

<sup>b</sup> sampled in 2006

Table 3.5. Seroprevalence of antibodies against *Leptospira spp.* among raccoons sampled at 18 urban open, rural open, and urbanized sites in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006. The total seroprevalence column represents the entire sample from each site, site type and overall sample, while other columns give the seroprevalence for particular classes of individuals.

Land-use	No. tested	No. positive	<i>L. aut.*</i>	<i>L. brat.*</i>	<i>L. can.*</i>	<i>L. grippo.*</i>	<i>L. hard.*</i>	<i>L. ictero.*</i>	<i>L. pomona*</i>	Ties
Urbanized	38	17	<b>29.4 (5)</b>	5.9 (1)	0	23.5 (4)	11.7 (2)	0	0	29.4 (5)
Urbanized	8	1	0	<b>100 (1)</b>	0	0	0	0	0	0
Urbanized	13	2	0	<b>50 (1)</b>	0	<b>50 (1)</b>	0	0	0	0
Urbanized	10	3	0	<b>33.3 (1)</b>		<b>33.3 (1)</b>			<b>33.3 (1)</b>	0
Urbanized	25	1	0	0	0	0	0	0	0	100 (1)
Urbanized	28	9	0	11.1 (1)	0	<b>88.9 (8)</b>	0	0	0	0
Urbanized	10	3	0	<b>33.3 (1)</b>	0	<b>33.3 (1)</b>	0	0	0	<b>33.3 (1)</b>
Urban open	68	21	4.8 (1)	19.1 (4)	0	<b>33.3 (7)</b>	19.1 (4)	0	4.8 (1)	19.1 (4)
Urban open	43	12	<b>33.3 (4)</b>	16.7 (2)	0	<b>33.3 (4)</b>	0	0	0	16.7 (2)
Urban open	44	9	<b>22.2 (2)</b>	<b>22.2 (2)</b>	0	11.1 (1)	0	0	0	44.4 (4)
Urban open	15	6	16.7 (1)	0	0	<b>50 (3)</b>	33.3 (2)	0	0	0
Urban open	11	5	<b>80 (4)</b>	0	0	20 (1)	0	0	0	0
Urban open	13	9	0	0	0	<b>77.8 (7)</b>	0	0	22.2 (2)	0
Urban open	65	32	0	9.4 (3)	0	<b>50 (16)</b>	3.1 (1)	0	3.1 (1)	34.4 (11)
Urban open	44	17	5.9 (1)	5.9 (1)	0	<b>47.1 (8)</b>	0	0	5.9 (1)	35.3 (6)
Urban open	37	13	15.4 (2)	7.7 (1)	0	<b>76.9 (10)</b>	0	0	0	0
Rural open	41	14	14.3 (2)	21.4 (3)	0	<b>57.1 (8)</b>	0	0	0	7.1 (1)
Rural open	57	14	<b>57.1 (8)</b>	0	0	21.4 (3)	7.1 (1)	0	0	14.3 (2)
Rural open	38	17	<b>29.4 (5)</b>	5.9 (1)	0	23.5 (4)	11.7 (2)	0	0	29.4 (5)

\* *L. aut.* = *L. interrogans autumnalis*, *L. brat.* = *L. interrogans bratislava*, *L. can.* = *L. interrogans canicola*, *L. grippo* = *L. kirschneri grippotyphosa*, *L. hard.* = *L. borgpeterseni hardjo*, *L. ictero* = *L. interrogans icterohaemorrhagiae*, and *L. pomona*, = *L. interrogans pomona*.

Table 3.6. Seroprevalence of antibodies against *Leptospira spp.* among raccoons sampled at 18 urban open, rural open, and urbanized sites in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006. Percentage of seropositive samples by site for which a single serovar of *Leptospira spp.* presented the highest titer, ties represent samples in which two or more serovars were detected at equal titers. Bold text represents the predominant reactive serovar(s) at each site.

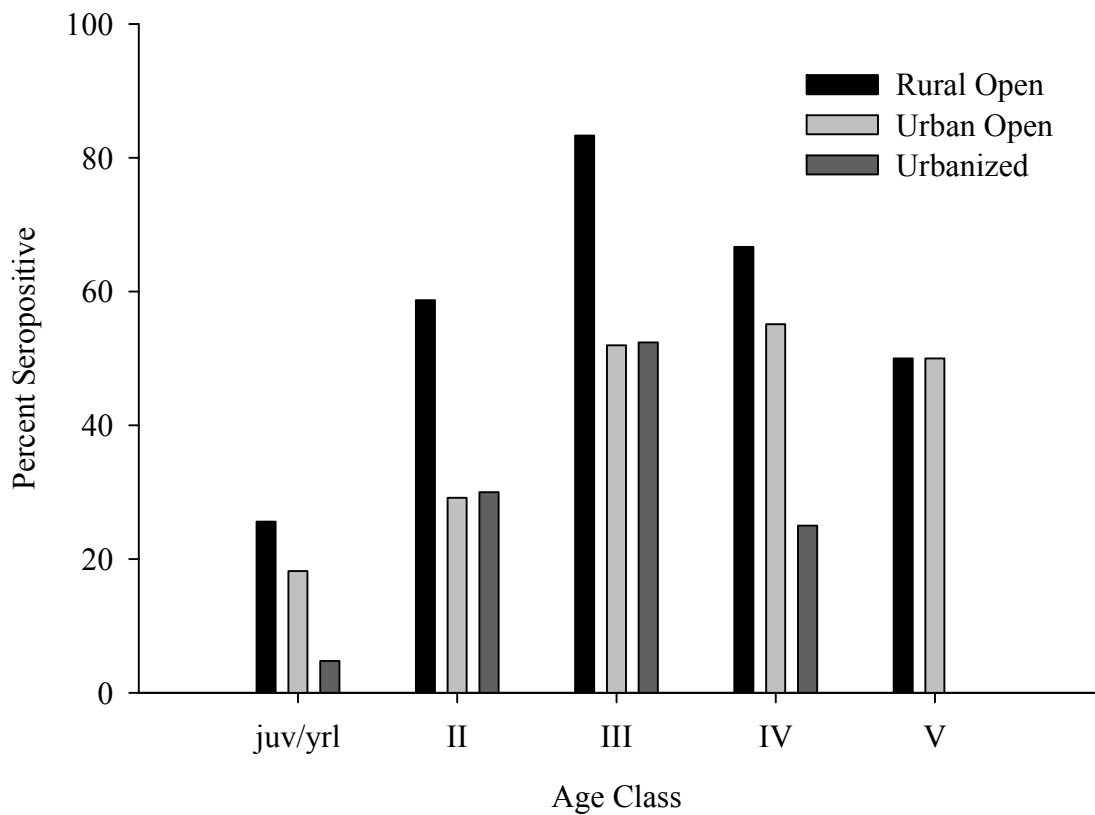


Figure 3.1. Percent of raccoons seropositive for antibodies against canine distemper virus (CDV) at rural open sites ( $n = 133$  raccoons), urban open sites ( $n = 299$ ), and urbanized sites ( $n = 132$ ) in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006.

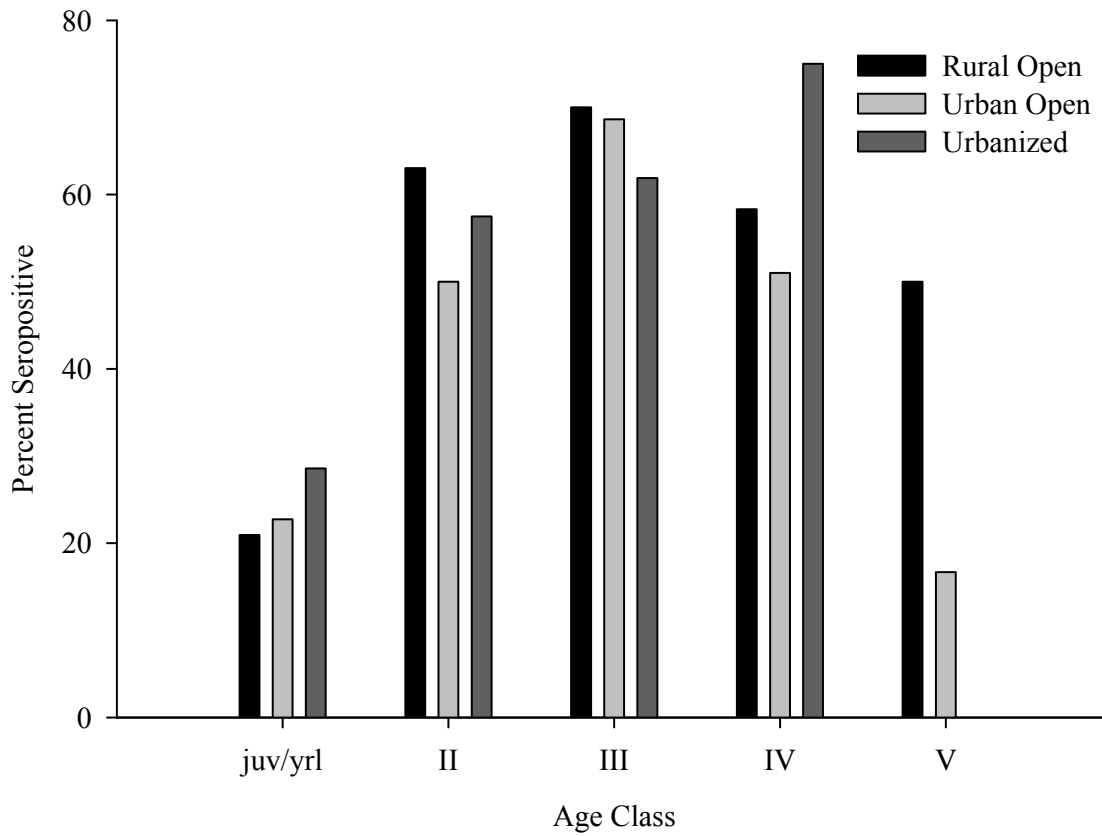


Figure 3.2. Percent of raccoons seropositive for antibodies against canine parvovirus (CPV) at rural open sites ( $n = 133$  raccoons), urban open sites ( $n = 299$ ), and urbanized sites ( $n = 132$ ) in Cook County and McHenry County, northeastern Illinois, USA, 2005-2006.

## BIBLIOGRAPHY

- Alig, R. J., J. D. Kline, M. Lichtenstein. 2004. Urbanization on the US landscape: looking ahead in the 21<sup>st</sup> century. *Landscape and Urban Planning* 69: 219-234.
- Barker, I. K., and C. R. Parrish. 2001. Parvovirus infections. In *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa, pp. 131-146.
- Bigler, W. J., J. H. Jenkins, P. M. Cumbie, G. L. Hoff, and E. C. Prather. 1975. Wildlife and environmental health: Raccoons as indicators of zoonoses and pollutants in southeastern United States. *Journal of the American Veterinary Medical Association* 167: 592-597.
- Birch, G. L., G. A. Feldhamer, and W. G. Dyer. 1994. Helminths of the gastrointestinal tract of raccoons in southern Illinois with management implications of *Baylisascaris procyonis* occurrence. *Transactions of the Illinois State Academy of Sciences* 87: 165-170.
- Bozek, C. K., S. Prange, and S. D. Gehrt. 2007. The influence of anthropogenic resources on multi-scale habitat selection by raccoons. *Urban Ecosystems* 10: 413-425.
- Bradley, C. A., and S. Altizer. 2006. Urbanization and the ecology of wildlife diseases. *Trends in Ecology and Evolution* 22: 95-102.
- Burnham, K. P., Anderson, D.R. 2002. *Model selection and multi-model inference: a practical information-theoretic approach*. 2<sup>nd</sup> ed. Springer-Verlag, New York.
- Center for Disease Control. 1998. Update: leptospirosis and unexplained acute febrile illness among athletes participating in triathlons-Illinois and Wisconsin. *MMWR* 47: 673-676.
- Chamberlain, M. J., K. M. Hodges, B. D. Leopold, and T. S. Wilson. 1999. Survival and cause-specific mortality of adult raccoons in central Mississippi. *Journal of Wildlife Management* 63: 880-888.



- Chamberlain, M. J., and B. D. Leopold. 2002. Spatio-temporal relationships among adult raccoons (*Procyon lotor*) in central Mississippi. *American Midland Naturalist* 148: 297-308.
- Chamberlain, M. J., L. M. Conner, B. D. Leopold, and K. M. Hodges. 2003. Space use and multi-scale habitat selection of adult raccoons in central Mississippi. *Journal of Wildlife Management* 67: 334-340.
- Chicago Metropolitan Agency for Planning. 2006. Data Bulletin: 2001 Land-use inventory for northeastern Illinois. Chicago Metropolitan Agency for Planning, Chicago, Illinois. Chicago Metropolitan Agency for Planning. 2006. Data Bulletin: 2001 Land-use inventory for northeastern Illinois. Chicago Metropolitan Agency for Planning, Chicago, Illinois.
- Clark, W. R., J. J. Hasbrouck, J. M. Kienzler, and T. F. Gluek. 1989. Vital statistics and harvest of an Iowa raccoon population. *Journal of Wildlife Management* 53: 982-990.
- Collinge, S. K., W. C. Johnson, C. Ray, R. Matchett, J. Grensten, J. F. Cully Jr., K. L. Gage, M. Y. Kosoy, J. E. Loye, and A. P. Martin. 2005. Landscape structure and plague occurrence in black-tailed prairie dogs on grasslands of the western USA. *Landscape Ecology* 20: 941-955.
- Crooks, K. R. 2002. Relative sensitivities of mammalian carnivores to habitat fragmentation. *Conservation Biology* 16: 488-502.
- DeStephano, S., and R. D. Deblinger. 2005. Wildlife as valuable natural resources vs. intolerable pests: a suburban wildlife management model. *Urban Ecosystems* 8: 179-190.
- Dobson, A. 2000. Raccoon rabies in space and time. *Proceedings of the National Academy of Sciences* 97: 14041-14043.
- Dubey, J. P. 1996. Strategies to reduce transmission of *Toxoplasma gondii* to animals and humans. *Veterinary Parasitology* 64: 65-70.
- Ellis, R. J. 1964. Tracking raccoons by radio. *Journal of Wildlife Management* 28: 363-368.
- Fishbein, D. B., and L. E. Robinson. 1993. Rabies. *New England Journal of Medicine* 329: 1632-1638.
- Fish, D., and T. J. Daniels. 1990. The role of medium-sized mammals as reservoirs of *Borrelia burgendorferi* in southern New York. *Journal of Wildlife Diseases* 26: 339-345.

- Finley, D. J., G. C. White, and J. P. Fitzgerald. 2005. Estimation of swift fox population size and occupancy rates in eastern Colorado. *Journal of Wildlife Management* 69: 861-873.
- Fritzell, E. K. 1978a. Habitat use by prairie raccoons during the waterfowl breeding season. *Journal of Wildlife Management* 42: 118-127.
- Fritzell, E. K. 1978b. Aspects of raccoon (*Procyon lotor*) social organization. *Canadian Journal of Zoology* 56: 260-71.
- Fritzell, E. K., G. F. Hubert, B. E. Meyen, and G. C. Sanderson. 1985. Age-specific reproduction in Illinois and Missouri raccoons. *Journal of Wildlife Management* 49: 901-905.
- Gehrt, S. D. 2002. Evaluation of spotlight and road-kill surveys as indicators of local raccoon abundance. *Wildlife Society Bulletin* 30: 449-456.
- Gehrt, S. D. 2003. Raccoons and allies. In *Wild mammals of North America: biology, management, and conservation* (G. A. Feldhamer, B. C. Thompson, and J.A. Chapman, eds.). 2nd ed. Johns Hopkins University Press, Baltimore, Maryland, pp. 611-633.
- Gehrt, S. D., and E. K. Fritzell. 1996a. Second estrus and late litters in raccoons. *Journal of Mammology* 77: 388-393.
- Gehrt, S. D., and E. K. Fritzell. 1996b. Sex-biased response of raccoons (*Procyon lotor*) to live traps. *The American Midland Naturalist* 135: 23-32.
- Gehrt, S. D., and E. K. Fritzell. 1997. Sexual differences in home ranges of raccoons. *Journal of Mammalogy* 78: 921-931.
- Gehrt, S. D., and E. K. Fritzell. 1998a. Resource distribution, female home range dispersion and male spatial interactions: group structure in a solitary carnivore. *Animal Behavior* 55: 1211-1227.
- Gehrt, S. D., and E. K. Fritzell. 1998b. Duration of familial bonds and dispersal patterns for raccoons in south Texas. *Journal of Mammalogy* 79: 859-872.
- Gehrt, S. D., and E. K. Fritzell. 1999. Survivorship of a nonharvested raccoon population in south Texas. *Journal of Wildlife Management* 63: 889-894.
- Gehrt, S. D., L. L. Hungerford and S. Hatten. 2001. Drug effects on recaptures of raccoons. *Wildlife Society Bulletin* 29: 833-837.
- Glueck, T. F., W.R. Clark, and R.D. Andrews. 1988. Raccoon movement and habitat use during the fur harvest season. *Wildlife Society Bulletin* 16: 6-11.

- Grau, G. A., G. C. Sanderson, and J. P. Rogers. 1970. Age determination of raccoons. *Journal of Wildlife Management* 34: 364-372.
- Greenwood, R. J. 1981. Foods of prairie raccoons during the waterfowl nesting season. *Journal of Wildlife Management* 45: 754-760.
- Greenwood, R. J. 1982. Nocturnal activity and foraging of prairie raccoons (*Procyon lotor*) in North Dakota. *The American Midland Naturalist* 107: 238-243.
- Grimm, N. B., S. H. Faeth, N. E. Golubiewski, C. L. Redman, J. Wu, X. Bai, and J. M. Briggs. 2008. Global change and the ecology of cities. *Science* 319: 756-760.
- Hancock, K., L. A. Thiele, A. M. Zajac, F. Elvinger, and D. S. Lindsay. 2005. Prevalence of antibodies to *Toxoplasma gondii* in raccoons (*Procyon lotor*) from an urban area of Northern Virginia. *Journal of Parasitology* 91: 694-695.
- Henner, C. M., M. J. Chamberlain, B. D. Leopold, and W. Burger Jr. 2004. A multi-resolution assessment of raccoon den selection. *Journal of Wildlife Management* 68: 179-187.
- Hill, D., and J. P. Dubey. 2002. *Toxoplasma gondii*: transmission, diagnosis and prevention. *Clinical Microbiology and Infection* 8: 634-640.
- Hill, R. E., J. E. Zimmerman, R. W. Willis, S. Patton, and W. R. Clark. 1998. Seroprevalence of antibodies against *Toxoplasma gondii* in free ranging mammals in Iowa. *Journal of Wildlife Diseases* 34: 811-815.
- Hoff, G. L., W. J. Bigler, S. J. Proctor, and L. P. Stallings. 1974. Epizootic of canine distemper virus infection among urban raccoons and gray foxes. *Journal of Wildlife Diseases* 10: 423-428.
- Hoffman, C. O., and J. L. Gottschang. 1977. Numbers, distribution, and movements of a raccoon population in a suburban residential community. *Journal of Mammalogy* 58: 623-636.
- Holmes, R. T., P. P., Marra, and T. W. Sherry. 1996. Habitat-specific demography of breeding black-throated blue warblers (*Dendroica caerulescens*): implications for population dynamics. *Journal of Animal Ecology* 65: 183-195.
- Hosmer, D. W., and S. Lemeshow. 1989. Applied logistic regression. John Wiley & Sons, New York.
- Huggins, R. M. 1989. On the statistical analysis of capture experiments. *Biometrika* 76: 133-140.

- Huggins, R. M. 1991. Some practical aspects of a conditional likelihood approach to capture experiments. *Biometrics* 47: 725-732.
- Hurvich, C. M., and C. L. Tsai. 1989. Regression and time series model selection in small sample sizes. *Biometrika* 76: 297-307.
- Jacobson, J. E., K. R. Kazacos, and F. H. Montague. 1982. Prevalence of eggs of *Baylisascaris procyonis* (Nematoda:Ascaroidea) in raccoon scats from an urban and rural community. *Journal of Wildlife Disease* 18: 461-464.
- Jakob, E. M., S. D. Marshall, and G. W. Uetz. 1996. Estimating fitness: a comparison of body condition indices. *Oikos* 77: 61-67.
- Junge, R. E., K. Bauman, M. King, and M. E. Gompper. 2007. A serologic assessment of exposure to viral pathogens and *Leptospira* in an urban raccoon (*Procyon lotor*) population inhabiting a large zoological park. *Journal of Zoo and Wildlife Medicine* 38: 18-26.
- Kaufmann, J. H. 1982. Raccoons and allies. In *Wild Mammals of North America: biology, management, and economics* (J. A. Chapman and G. A. Feldhamer, eds.). Johns Hopkins University Press, Baltimore, Maryland, USA, pp. 567-585.
- Kirkpatrick, C. M., C. L. Kanitz, and S. M. McCrocklin. 1980. Possible role of wild mammals in the transmission of pseudorabies to swine. *Journal of Wildlife Diseases* 16: 601-614.
- Krebs, J. W., S. C. Long-Marin, and J. E. Childs. 1998. Causes, costs, and estimates of rabies postexposure prophylaxis treatment in the United States. *Journal of Public Health Management and Practice* 4: 56-62.
- Kruezer, M. P., N. J. Huntly. 2003. Habitat-specific demography: evidence for source-sink population structure in a mammal, the pika. *Oecologia* 134: 343-349.
- Lednicky, J. A., J. Dubach, M. J. Kinsel, T. P. Meehan, M. Bocchetta, L. L. Hungerford, N. A. Sarich, K. E. Witecki, M. D. Braid, C. Pedrak, and C. M. Houde. 2004. Genetically distant American canine distemper virus lineages have recently caused epizootics with somewhat different characteristics in raccoons living around a large suburban zoo in the USA. *Virology Journal* 1: 1-14.
- Leighton, F. A., and T. Kuiken. 2001. Leptospirosis. In *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa, pp. 498-502.
- LoGiudice, K. 2003. Trophically transmitted parasites and the conservation of small populations: raccoon roundworm and the imperiled Allegheny woodrat. *Conservation Biology* 17: 258-266.

- Lotze, J., and S. Anderson. 1979. *Procyon lotor*. Mammalian Species 119: 1-8.
- Mankin, P. C., C. M. Nixon, J. B. Sullivan, T. L. Esker, R. G. Koerkenmeier and L. L. Hungerford. 1999. Raccoon (*Procyon lotor*) survival in west-central Illinois. Transactions of the Illinois State Academy of Science 92: 247-256.
- McDonough, P. L. 2001. Leptospirosis in dogs-current status. In: Carmichael, L. ed. Recent Advances in Canine Infectious Diseases. International Veterinary Information Service, Ithaca, New York.
- McKinney, M. L. 2002. Urbanization, biodiversity, and conservation. Bioscience 52: 883-890.
- McKinney, M. L. 2006. Urbanization as a major cause of biotic homogenization. Biological Conservation 127: 247-260.
- Mech, L. D., D. M. Barnes, and J. R. Tester. 1968. Seasonal weight changes, mortality, and population structure of raccoons in Minnesota. Journal of Mammalogy 49: 63-73.
- Mitchell, M. A., L. L. Hungerford, C. Nixon, T. Esker, J. Sullivan, R. Koerkenmeier, and J. P. Dubey. 1999. Serologic survey for selected infectious disease agents in raccoons from Illinois. Journal of Wildlife Diseases 35: 347-355.
- Moore, D. W., M. L. Kennedy. 1985. Weight changes and population structure of raccoons in western Tennessee. Journal of Wildlife Management 49: 906-909.
- National Oceanic and Atmospheric Administration (NOAA). 2002. Climatography of the United States No. 81: Monthly normals of temperature, precipitation, and heating and cooling degree days 1971-2000. National Climatic Data Center/NESDIS/NOAA, Asheville, North Carolina.
- Northeastern Illinois Planning Commission. 2006. 2030 forecasts of population, households and employment by county and municipality. Northeastern Illinois Planning Commission, Chicago, Illinois.
- Page, L. K., S. D. Gehrt, and N. P. Robinson. 2008. Land-use effects on prevalence of raccoon roundworm (*Baylisascaris procyonis*). Journal of Wildlife Diseases 44: 594-599.
- Patz, J. A., P. Daszak, G. M. Tabor, A. A. Aquirre, M. Pearl, J. Epstein, N. D. Wolfe, A. M. Kilpatrick, J. Fofopoulos, D. Molyneux, D. J. Bradley et al. 2004. Unhealthy landscapes: policy recommendations on land use change and infectious disease emergence. Environmental Health Perspectives 112: 1092-1098.

- Pedlar, J. H., L. Fahrig, and H. G. Merriam. 1997. Raccoon habitat use at 2 spatial scales. *Journal of Wildlife Management* 61: 102-112.
- Prange, S., T. Jordan, C. Hunter, and S.D. Gehrt. 2006. New radiocollars for the detection of proximity among individuals. *Wildlife Society Bulletin* 34: 1333-1344.
- Prange, S., and S. D. Gehrt. 2004. Changes in mesopredator-community structure in response to urbanization. *Canadian Journal of Zoology* 82: 1804-1817.
- Prange, S., S. D. Gehrt, and E. P. Wiggers. 2003. Demographic factors contributing to high raccoon densities in urban landscapes. *Journal of Wildlife Management* 67: 324-333.
- Prange, S., S. D. Gehrt, and E. P. Wiggers. 2004. Influences of anthropogenic resources on raccoon (*Procyon lotor*) movements and spatial distribution. *Journal of Mammalogy* 85: 483-490.
- Rabinowitz, A. R., and L. N. D. Potgieter. 1984. Serologic survey for selected viruses in a population of raccoons, *Procyon lotor* (L.), in the Great Smoky Mountains. *Journal of Wildlife Diseases* 20: 146-148.
- Ramey, P. C., B. F. Blackwell, R. J. Gates, and R. D. Slemons. 2008. Oral rabies vaccination of a northern Ohio raccoon population: relevance of population density and prebait serology. *Journal of Wildlife Disease* 44: 553-568.
- Reif, J. S. 1976. Seasonality, natality and herd immunity in feline panleukopenia. *American Journal of Epidemiology* 103: 81-87.
- Richardson, D. J., and J. L. Gauthier. 2003. A serosurvey of leptospirosis in Connecticut peridomestic wildlife. *Vector-Borne and Zoonotic Diseases* 3: 187-193.
- Riley, S. P. D., J. Hadidian, and D. A. Manski. 1998. Population density, survival, and rabies in raccoons in an urban national park. *Canadian Journal of Zoology* 76: 1153-1164.
- Robinson, V. B., J. W. Newberne, and D. M. Brooks. 1957. Distemper in the American raccoon (*Procyon lotor*). *Journal of the American Veterinary Medical Association* 131: 276-278.
- Rosatte, R. C. 2000. Management of raccoons (*Procyon lotor*) in Ontario, Canada: Do human intervention and disease have significant impact on raccoon populations? *Mammalia* 64: 369-390.

- Rosatte, R. C., M. J. Power, and C. D. MacInnes. 1991. Ecology of urban skunks, raccoons and foxes in metropolitan Toronto. In *Wildlife conservation in metropolitan environments*. Edited by L. W. Adams and D. L. Leedy. National Institute for Urban Wildlife, Columbia, Maryland, pp. 31-38.
- Rosatte, R. C., M. J. Power, and C. D. MacInnes. 1992. Density, dispersion, movements, and habitat of skunks (*Mephitis mephitis*) and raccoons (*Procyon lotor*) in metropolitan Toronto. In *Wildlife 2001: populations*. Edited by D. R. McCullough and R. H. Barrett. Elsevier Science, Barking, Essex, UK. pp. 932-942.
- Roscoe, D. E. 1993. Epizootiology of canine distemper in New Jersey raccoons. *Journal of Wildlife Diseases* 29: 390-395.
- Sanderson, G. C. 1987. Raccoon. In M. Novak, J.A. Baker, M. E. Obbard, and B. Malloch, eds. *Wild furbearer management and conservation in North America*. Ontario Trappers Association, North Bay, Canada, pp. 487-499.
- Sanderson, G. C., and A. V. Nalbandov. 1973. The reproductive cycle of the raccoon in Illinois. *Illinois Natural History Survey Bulletin* 31: 25-85.
- Schinner, J. R., and Cauley, D. L. 1974. The ecology of urban raccoons in Cincinnati, Ohio. In *Wildlife in an urbanizing environment*. Edited by J. H. Noyes and D. R. Progulske. *Planning and Resource Development Series No. 28*, Holdsworth Natural Resources Center, Amherst, Massachusetts, pp. 125-130.
- Schmidt, K. A., and R. S. Ostfeld. 2001. Biodiversity and the dilution effect in disease ecology. *Ecology* 82: 609-619.
- Schubert, C. A., I. A. Barker, R. C. Rosatte, C. D. MacInnes, and T. D. Nudds. 1998. Effect of canine distemper on an urban raccoon population: An experiment. *Ecological Applications* 8: 379-387.
- Sorvillo, F., L. R. Ash, O. G. W. Berlin, J. Yatabe, C. Degiorgio, and S. A. Morse. 2002. *Baylisascaris procyonis*: An emerging helminthic zoonosis. *Emerging Infectious Diseases* 8: 355-359.
- Stains, H. J. 1956. *The raccoon in Kansas, natural history, management, and economic importance*. University of Kansas, Lawrence, Kansas.
- Stallnecht, D. E., and E. W. Howerth. 2001. Pseudorabies (Aujeszky's Disease). In *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa, pp. 164-170.
- StataCorp. 2005. *Stata statistical software: release 9*. College Station, TX: StataCorp LP.

- Thawley, D. G., and J. C. Wright. 1982. Pseudorabies virus infection in raccoons: a review. *Journal of Wildlife Diseases* 18: 113-116.
- Twichell, A. R., and H. H. Dill. 1949. One hundred raccoons from one hundred and two acres. *Journal of Mammalogy* 30: 130-133.
- Urban, D. 1970. Raccoon populations, movement patterns, and predation on a managed waterfowl marsh. *Journal of Wildlife Management* 34: 372-282.
- U.S. Census Bureau. 1995. Population of counties by census: 1900 to 1990. Population Division, U.S. Bureau of the Census, Washington D.C., USA.
- U.S. Census Bureau. 2001. Statistical abstract of the United States: 2001, 112<sup>th</sup> edition. U.S. Bureau of the Census, Washington D.C., USA.
- U.S. Census Bureau. 2006a. Statistical abstract of the United States: 2006, 125<sup>th</sup> edition. U.S. Bureau of the Census, Washington D.C., USA.
- U.S. Census Bureau. 2006b. State and county QuickFacts. U.S. Bureau of the Census, Washington D.C., USA.
- USDA. 2004. Illinois Agricultural Statistics: County Highlights. [accessed 8/20/08 [www.agstats.state.il.us/annual/2004/](http://www.agstats.state.il.us/annual/2004/)].
- 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. 2005. Correcting wildlife counts using capture probabilities. *Wildlife Research* 32: 211-216.
- White, G. C., and K. P. Burnham. 1999. Program MARK: survival estimation from populations of marked animals. *Bird Study* 46: 120-138.
- Williams, E. S. 2001. Canine distemper. In *Infectious diseases of wild mammals* (E. S. Williams and I. K. Barker eds.). 3<sup>rd</sup> ed. Iowa State University Press, Ames, Iowa, pp. 50-59.
- Woolhouse, M.E. 2002. Population biology of emerging and re-emerging pathogens. *Trends in Microbiology*. 10 (10 Suppl.) S3-7.
- Woolhouse, M. E., and S. Gowtage-Sequeria. 2005. Host range and emerging and reemerging pathogens. *Emerging Infectious Diseases* 11: 1842-1847.



- Wright, A. N., and M. E. Gompper. 2005. Altered parasite assemblages in raccoons in response to manipulated resource availability. *Oecologia* 144: 148-156.
- Wright, J. C., and D. G. Thawley. 1980. Role of the raccoon in the transmission of psuedorabies: a field and laboratory investigation. *American Journal of Veterinary Research* 41: 581-583.
- Yabsley, M. J., and G. P. Noblet. 2002. Seroprevalence of *Trypanosoma cruzi* in raccoons from South Carolina and Georgia. *Journal of Wildlife Diseases* 38: 75-83.
- Yeager, L. E., W. H. Elder. 1945. Pre-and post-hunting season foods of raccoons on an Illinois goose refuge. *Journal of Wildlife Management* 9: 48-56.