

EPIDEMIOLOGY OF SUBURBAN DEER

BY

KARMEN M. HOLLIS

DISSERTATION

Submitted in partial fulfillment of the requirements
for the degree of Doctor of Philosophy in Natural Resources and Environmental Sciences
in the Graduate College of the
University of Illinois at Urbana-Champaign, 2011

Urbana, Illinois

Doctoral Committee:

Professor Richard Warner, Chair
Professor Jeffrey Brawn
Professor Patrick Brown, Northern Michigan University
Associate Professor Timothy VanDeelen

ABSTRACT

Urban and suburban development has expanded beyond the traditional core of major cities. Preservation of open spaces in metropolitan landscapes provides aesthetic and recreational values for humans. However, white-tailed deer (Odocoileus virginianus) populations in North America are overabundant in many suburban areas. This trend is directly related to changes to habitat as a result of landscape alteration and fragmentation caused by humans. This close relationship between humans and wildlife increases the risk for zoonotic disease transmission from many vectors, such as mosquitoes and ticks. There is limited research for suburban deer examining the relationship between zoonotic prevalence and ecological factors, such as habitat. Additionally, an extensive search of deer ecology and wildlife disease literature revealed no peer-reviewed publications of zoonoses from suburban white-tailed deer. From 1995-99, I conducted a zoonotic serological survey of live-captured (tagged and radio-collared) and culled deer from Chicago, Illinois forest preserves for Jamestown Canyon (JC) and LaCrosse (LAC) encephalitis viruses, toxoplasmosis, and leptospirosis. The prevalence of sera samples collected from suburban white-tailed deer (live-captured and culled) was 55.9% for Toxoplasma gondii (n=443), 16.2% for Leptospira spp. (n=444), 38.1% for JC virus (n=404), and 6.7% for LAC virus (n=404). Prevalence was higher for adult deer for all pathogens sampled except Leptospira spp. Prevalence also was less likely in males for JC virus. A higher prevalence for T. gondii and JC virus was detected at Des Plaines (DP) and prevalence for Leptospira spp. was less likely at DP. Differences in year the sample was collected were present for T. gondii, Leptospira spp., and JC virus. Variation in month the samples were collected was evident for T. gondii, Leptospira spp., and LAC virus. Main effects variables (age, sex, site, year, and month) for multivariate logistic regression (LR) models were screened using backward

stepwise elimination ($p \leq 0.20$). The final LR model for *T. gondii* contained all main effects variables. *Leptospira* spp. and JC viral models both contained age, site, and year, while the final LAC viral LR model contained age, sex, and month. A subsample of deer ($n=169$ from DP and Palos; radio-collared and culled) analyzed for JC and LAC viral antibodies was used to evaluate habitat characteristics. Radio-collared does selected for forest, savanna, and grassland habitats (available habitat= $p < 0.0001$; home range= $p < 0.001$). Akaike's Information Criterion (AICc) optimal models for JC contained water and wetland habitat parameters while LAC models contained forested habitat. Deer biological parameters for JC viral models included age and month/year the samples were collected. Total home-range or buffer size and month were biological parameters contained in LAC viral models. Blood samples from live-captured and culled deer tested for JC (238 seropositive and 402 seronegative cases) and LAC viruses (51 seropositive and 402 seronegative cases) from 11 forest preserves were used for spatial analysis. Using the Bernoulli model in the space-time scan statistic in SaTScan[®], a significant viral "hot spot" was revealed for JC virus in DP ($p=0.009$). A significant cluster for LAC virus was also found in DP ($p=0.005$). All clusters were independent of each other. Temporal patterns for JC virus spanned samples from December 1, 1995 to March 31, 1998. This research demonstrates deer are positive for several zoonotic diseases in the Chicago area. The environmental and habitat factors required to maintain pathogens and vectors are present at various levels in Cook County Forest Preserves. "Hot spots" are present for JC and LAC viruses and deer serve as an effective wildlife sentinel. The relationship between habitat and spatial data can be used to focus surveillance. This research not only quantifies deer exposures to pathogens, but also provides a more sensitive assessment of risk than human or pet surveillance alone. Wildlife species, such as deer, serve as effective disease sentinels because they are exposed to potential pathogens

everyday in their natural environment when humans and pets are typically random and accidental exposures. Wildlife ecologists have a responsibility to increase public knowledge about the health status of animal populations while protecting humans and domestic animals from zoonotic diseases. This research represents the largest suburban deer zoonotic surveillance project to date in the United States and provides evidence that suburban deer are effective biomonitors for zoonoses. International, federal, and state management agencies collect various harvested wildlife species annually which could be easily accessed for zoonoses sampling. Annual culling of deer continues in Chicago area forest preserves and samples should be collected and analyzed for zoonotic diseases with minimum effort and cost. Results should be communicated to public health officials to assist in focusing vector control and to notify the public of potential exposures.

ACKNOWLEDGEMENTS

This research could not have been conducted or completed without the endless advice, assistance, and support of numerous individuals. I want to thank my advisor, Dr. Richard Warner for his guidance, knowledge, and commitment to me and this research project. Even when I could not see the light at the end of the tunnel, he was there. You have set a life-time example for me as an advisor, and you are the most patient man I know. I want to thank my present and past committee members, Dr. Jeff Brawn, Dr. Patrick Brown, Dr. Phil Mankin, Dr. Randy Peper, Dr. Gary Rolfe, and Dr. Tim Van Deelen for their knowledge, support, scientific, and editorial input while completing my dissertation.

I have some very good friends in the Forest Preserve District of Cook County's Wildlife Field Office. I am indebted to Chris Anchor and Jim Chelsvig. They literally spent years working on this project with me. They opened their doors, loaned equipment and vehicles, and allowed me to work with them side by side. We spent hours in the office discussing strategies and many nights in the field catching, radio-tracking, culling, and processing deer. It is not just the postman who delivers in the wind, rain, sleet or snow, so do Chris and Jim. I know it has been a long time coming. Thanks for trusting me.

In addition, a project of this magnitude cannot be completed without the assistance of many professionals in their field of expertise and knowledge of data analysis. Thank you for the many hours that were sacrificed by Dr. Pat Brown, Dr. Dwayne Etter, and Dr. Richard Warner while reviewing and editing the dissertation. It would not have been completed without you. Additional thanks to Dr. Jeff Brawn, Dr. Dwayne Etter, and Dr. Tim Van Deelen for sharing their statistical expertise. I want to thank Dr. Uriel Kitron for his review and input on geographical information systems and spatial analysis. Thank you to Dr. Leo Chapa for his

extensive knowledge and assistance in running the geographic data. Additional acknowledgements to my publication co-authors Dr. J. P. Dubey, Dr. Paul Grimstad, Dr. Marvin Godsey, and Diane Lovin for performing serological analysis during this research project and to Dr. Laura Hungerford who taught me epidemiology. Thank you to Dr. Sagar Goyal and his staff at the Minnesota Veterinary Diagnostic Laboratory for running all the leptospirosis serology. Hats also go off to the Natural Resources and Environmental Sciences (NRES) Graduate Office staff, especially Karen and Piper, who guided me repeatedly through the department and graduate college processes.

Many thanks to the Cook County Department of Animal Control, Forest Preserve District of Cook County, and University of Illinois for generously funding and supporting this research. I greatly appreciate the assistance with sample collection by the Forest Preserve District of DuPage County. Special thanks goes out to D. Antilitz, T. Anchor, R. Bartman, M. Ermer, B. Green, L. Kammin, R. Keough, N. Komar, S. Lavin, W. Luby, M. Narrish, J. Nevis, D. Parmer, M. F-Peters, J. Phillips, B. Piccolo, C. Pollock, C. Ryndak, S. Strack, R. Trakur, M. VanKast, and T. Weicherding for their outstanding assistance and extensive contributions to this project in the field, office, and political arenas.

Last, but not least, I want to thank my husband, Dr. Dwayne Etter. Not only did we move to Chicago and start our research projects together, we sacrificed a large part of our lives while pursuing our PhD's. He is the perpetual example of an ideal leader, wildlife research biologist, published scientist, and excels with the Michigan Department of Natural Resources. He stood by me through thick and thin while picking me up many times during this journey. It has been a very long road. Thank you for believing in me and loving me like no one else.

TABLE OF CONTENTS

INTRODUCTION.....	1
GENERAL MATERIALS AND METHODS.....	14
SEROLOGICAL SURVEY OF SUBURBAN DEER.....	22
HABITAT MODELING OF ENCEPHALITIDES USING SUBURBAN DEER.....	53
SPATIAL AND TEMPORAL ANALYSIS OF ENCEPHALITIDES IN SUBURBAN DEER.....	73
MANAGEMENT RECOMMENDATIONS.....	88
APPENDIX A: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR BABESIA.....	90
APPENDIX B: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR CACHE VALLEY VIRUS.....	91
APPENDIX C: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR <u>EHRlichia</u> <u>CHAFFEENSIS</u>	93
APPENDIX D: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR EPIZOOTIC HEMORRHAGIC VIRUS.....	94
APPENDIX E: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR <u>ESCHERICHIA</u> <u>COLI</u>	95
APPENDIX F: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR JAMESTOWN CANYON VIRUS.....	96
APPENDIX G: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR LACROSSE VIRUS.....	98
APPENDIX H: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR <u>LEPTOSPIRA</u>	100

APPENDIX I: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR <u>NEOSPORA</u> <u>CANINUM</u>	102
APPENDIX J: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR POTOSI VIRUS.....	104
APPENDIX K: SUBURBAN WHITE-TAILED DEER SEROPOSITIVE FOR <u>TOXOPLASMA</u> <u>GONDII</u>	105
APPENDIX L: MAP OF DUPAGE COUNTY FOREST PRESERVES, CHICAGO, ILLINOIS, USA, 1995-99.....	107
APPENDIX M: FRAGSTATS VARIABLES INCLUDED IN THE ORIGINAL ANALYSIS TO MODEL HABITAT.....	108

INTRODUCTION

Zoonoses are infectious diseases that can be transmitted naturally between humans and wild or domestic animals (Slingenbergh et al., 2004). Zoonoses from wildlife and vector-borne pathogens threaten human health, global economics, and are driving an emerging infectious disease (EID) trends worldwide. More than half of the defined 1,400 EID organisms can cause human infections (Woolhouse and Gowtage-Sequeria, 2005). Greater than 60% of EID events are caused by zoonotic pathogens and more than 70% of these events result from pathogens with a wildlife origin. Additionally, vector-borne diseases are responsible for 23% of EID events and 29% have occurred in the last decade (Jones et al., 2008). Examples of some important EIDs include human immunodeficiency virus (HIV), tuberculosis, cholera, Lyme disease, Parvovirus B19, rotavirus, Escherichia coli (E. coli) 0517-H7, and West Nile virus (WNV). Some diseases like tularemia and plague are established diseases capable of causing mass mortalities in small mammals and are now appearing in new locations. In the United States, more than a dozen food borne diseases have emerged and the Pan American Health Organization reports several food borne zoonoses have increased by 100% in recent years (Friend, 2006).

Presently, there are several anthropogenic changes occurring to natural ecosystems globally that have the potential to influence pathogen dynamics (McCallum, 2008). Climate change facilitates expansion of diseases into new territories. Change in weather patterns and temperature may influence arthropod vectors, their life cycles, and life histories. Such changes may result in altered vector and pathogen distribution and transmission. Climate can affect the way pathogens interact with arthropod vector(s) and human or animal hosts. With increased temperatures and rainfall associated with global warming in some parts of the world, new habitats where vector mosquitoes can breed contributes to a suitable climate for disease

transmission, like dengue fever. In 1970 dengue fever was only documented in nine countries and now is documented in over 100 countries (Hales et al., 2002). Other diseases linked to climate change include Lyme disease, schistosomiasis, tuberculosis, bubonic plague, and cholera (Cunningham and Cunningham, 2009).

Rapid international travel also makes it possible for diseases to spread around the world. In 2008, 992 million people travelled internationally (World Tourism Organization, 2009). West Nile virus was introduced into North America in 1999 by imported birds or mosquitoes. The disease rapidly spread across the United States from New York in less than two years. The virus found a foothold within populations of susceptible birds, hosts, vectors, and mammals that had never had contact with the virus (Enserink, 2000). The abundance of susceptible native avian species and available habitats for avian hosts and vector mosquitoes supported the establishment and rapid expansion of WNV across the United States (McLean, 2008). The virus is now found in all 48 lower states and infects at least 250 bird and 18 mammalian species, including humans (Cunningham and Cunningham, 2009). Travel-associated fungal infections, such as histoplasmosis and coccidioidomycosis, are worldwide illnesses on the rise. The fungus creates diagnostic challenges when human disease may not develop for many years and away from the endemic area where the fungus was contracted (Friend, 2006).

International trade of livestock and wildlife also increases the spread of zoonotic diseases and may have important economic and/or public health impacts. In 2003, humans in the United States became infected with monkey pox from the importation of wild west African rodents (Reed et al., 2004). Severe acute respiratory syndrome (SARS) and highly pathogenic avian influenza (H5N1) are emerging infections that have the potential for massive public health and economic consequences. Both diseases are maintained in wildlife reservoir hosts: H5N1 in wild

fowl and SARS in horseshoe bats (Rhinolophus spp.) in China. The trade in bats has likely brought SARS into contact with susceptible amplifying hosts such as the masked palm civet (Parguma larvata). Dog (Canis familiaris) and fox (Vulpes vulpes) rabies outbreaks reported in the United States during the late 1700's was likely exacerbated by the introduction of canines imported for fox hunting (Winkler, 1975). Translocation of raccoons (Procyon lotor) from Florida to Virginia for hunting led to the emergence of raccoon rabies in the mid-Atlantic states (Jenkins and Winkler, 1987). The threat of animal translocations and invasions into new geographic areas pose risks for native species, endangered species, and species diversity (Lau et al., 2005; Li et al, 2005).

Infectious disease studies in wildlife are crucial because wild animals serve as a reservoir, amplifier, and/or host for many pathogenic agents transmitted to humans and domestic animals (Hudson et al., 2001; Newman, et al, 2005). Pathogen surveillance is an early warning system for disease emergence, potential disease outbreaks, and opportunities for public health education. However, effective management of infectious diseases requires knowledge about the factors influencing infection within a host and transmission dynamics of the pathogen. Laboratory studies of infections assist in understanding disease transmission, but these studies are not sufficient to understand, indicate, or predict transmission dynamics in open populations between hosts and vectors. It is essential to understand the factors that increase transmission between wildlife and humans when developing surveillance programs, wildlife management plans, and predictive approaches to disease emergence.

Wildlife have an intimacy with the environment and provide enhanced sensitivity for disease detection (Friend, 2006). They have direct contact with the soil, water, vegetation, insects, and other food sources. For example, coyotes (Canis latrans) may act as biological

sensors for bovine tuberculosis (TB) by consuming infected host material (VerCauteren et al., 2008). Wildlife have been used as biomonitors for nuclear wastes at Chernobyl and chemical contaminants have been measured in fish and waterfowl (National Research Council, 1991). Captive chickens, pigeons, and dead crows have been used for WNV surveillance (Komar, 2001). White-tailed deer (*Odocoileus virginianus*) have served as biomonitors for encephalitis, anaplasmosis, and *Ehrlichia chaffeensis* (Trainer, 1973; Yabsley, 2003; Dugan, 2006). Disease surveillance in free-ranging wildlife populations provides an early warning system for public health agencies.

Urbanization changes landscape structure by creating fragmented habitats and brings certain wildlife species into closer contact with domestic animals and humans (Power and Mitchell, 2004). Landscape changes can also lead to disease emergence by increasing the abundance and/or distribution of disease vectors like mosquitoes and ticks. Urban/suburban landscapes are ideal locations to study zoonoses among wildlife, vectors, and pathogens that can threaten human health.

The greater Chicago, Illinois region consists of dense human development with interspersed green belts (parks and forest preserves), many associated with riparian corridors. Wetland and riparian areas contain ecological biodiversity, but also serve as habitat for mosquito larvae and disease vectors. Epidemiological models indicate corridors increase contact among various populations and can enhance the transmission of infectious diseases (Hess, 1994; Hess, 1996). Human population density in the Chicago area exceeds 2,100 people per km² (U. S. Census Bureau, 2000). In Chicago, numbers for domestic dogs and cats (*Felis catus*) are estimated at 1.3 and 1.5 million, respectively (Wise et al., 2002). Physical recreation (i.e., biking, canoeing, jogging, fishing, and outdoor sports) and picnicking are very common

activities in parks and forest preserves throughout the Chicago Region. Human recreationists are sometimes accompanied by pets (primarily domestic dogs) and sometimes encounter wildlife either directly (i.e., hand feeding or handling young wildlife) or indirectly (i.e., contacting urine, feces, and disease vectors like mosquitoes and ticks). These encounters increase the risk for contacting zoonoses and potentially expose other family members by bringing disease agents into the home. The interface of dense human development with abundant habitat for disease vectors in the Chicago region provided a unique opportunity to examine potential exposure to zoonoses.

Due to a lack of natural predators and because many jurisdictions do not permit recreational hunting and trapping to control wildlife populations, many common suburban wildlife species exist at high densities (Brown et al., 2000; Conover, 2001). Suburban raccoon densities in Chicago have exceeded 80 raccoons km² while adult coyote populations average 3-6 animals km² (Prange et al., 2003; Gehrt et al., 2009). Recent estimates of white-tailed deer densities in the Chicago region have exceeded 40-100 deer/km² (Witham and Jones, 1990; Etter, 2001). Within the Chicago region, white-tailed deer culling programs were effective at reducing deer densities for management of natural areas (Etter, 2001).

The awareness of infectious diseases and the population, community, and ecosystem level impacts have increased dramatically over the past decade (Daszak et al., 2000; Deem et al., 2001). Deer are widely recognized as the premier big game mammal in North America (Baker, 1984). However, deer are host to numerous bacteria, viruses, and parasites. Mycobacterium bovis is a bacterial pathogen causing bovine tuberculosis and can be transmitted to humans from deer by consuming infected tissues (Clifton-Hadley et al., 2001). This organism is now commonly found in deer in Michigan (Schmitt et al., 1997). Leptospira is a zoonotic bacterium

typically transmitted in contaminated water and deer are known to be reservoir hosts by shedding the organism (Leighton and Kuiken, 2001). Toxoplasma is a parasitic pathogen known to cause human disease when consuming infected venison (Davidson and Nettles, 1997). Many pathogenic parasites maintain transmission cycles on white-tailed deer, such as mosquitoes and ticks. Deer do not directly transmit disease to humans, but contribute to zoonoses such as Lyme disease, Babesiosis, and Ehrlichiosis by harboring the parasites (Thompson et al., 2001).

To be effective sentinels, suburban deer 1) facilitate detection of pathogen presence with a reasonable sampling effort and abundant numbers, 2) can be used to detect several pathogens at a time instead of sampling several different wildlife hosts/reservoirs for the same pathogens, 3) have a direct ecological relationship with the pathogen over time, 4) are easily identified and biological data collected (age and sex), 5) live for a long time in a relatively defined sedentary home range, and 6) interact with other wildlife and domestic species (Trainer, 1973; Beeby, 2001). Therefore, deer are a cost-effective way to infer pathogen prevalence in wildlife populations.

The conditions in Chicago area forest preserves created a unique opportunity to study wildlife pathogens present in a suburban environment. I began by examining zoonotic pathogen seroprevalence for Jamestown Canyon (JC) and LaCrosse (LAC) viral encephalitis, toxoplasmosis, and leptospirosis in white-tailed deer. I further examined the spatial and ecological factors associated with the epidemiology of JC and LAC viruses. Effectively, this project examined the unique approach of using white-tailed deer as a zoonotic surveillance tool. Such an approach can be combined with ongoing deer culling programs in the region to monitor zoonoses. This unique biomonitoring approach between epidemiology and deer ecology will supply wildlife managers and public health officials with valuable information to make decisions

about public health, managing wildlife populations, suburban development, and future emerging and reemerging infectious disease threats.

LITERATURE CITED

- BAKER, R. H. 1984. Origin, classification, and distribution *In* White-tailed deer: Ecology and management, W. R. Davidson, F. A. Hayes, V. F. Nettles, and F. E. Kellog (eds). Stackpole Books, Harrisburg, Pennsylvania, pp. 1-18.
- BEEBY, A. 2001. What do sentinels stand for? *Environmental Pollution* 112:285-298.
- BROWN, T. L., D. J. DECKER, S. J. RILEY, J. W. ENCK, B. LAUBER, P. D. CURTIS, AND G. F. MATTFELD. 2000. The future of hunting as a mechanism to control white-tailed deer populations. *Wildlife Society Bulletin* 28(4):797-807.
- CLIFTON-HADLEY, R. S., I. W. LUGTON, R. JACKSON, P. A. DURR, AND J. W. WILESMITH. 2001. Mycobacterial diseases *In* Infectious diseases of wild mammals, E. S. Williams and I. K. Barker (eds). Iowa State University Press, Ames, Iowa, pp.340-361.
- CONOVER, M. R. 2001. Effect of hunting and trapping on wildlife damage. *Wildlife Society Bulletin* 29(2):521-553.
- CUNNINGHAM, W. P. AND M. A. CUNNINGHAM. 2009. Environmental health and toxicology. *In* Principles of environmental science: Inquiry and applications. McGraw Hill Companies, New York, New York, pp. 175-198.
- DASZAK, P. A., A. CUNNINGHAM, AND A. D. HYATT. 2000. Emerging infectious diseases of wildlife: Threats to biodiversity and human health. *Science* 287:443-449.
- DAVIDSON, W. R. AND V. F. NETTLES. 1997. Field manual of wildlife diseases in the Southeastern United States. Southeastern Cooperative Wildlife Disease Study, Athens, Georgia, 417 pp.
- DEEM, S. L., W. B. KARESH, AND W. WEISMAN. 2001. Putting theory into practice: Wildlife health and conservation. *Conservation Biology* 15:1224-1233.

- DUGAN, V. G., M. J. YABSLEY, C. M. TATE, D. G. MEAD, U. G. MUNDERLOH, M. J. HERRON, D. E. STALLKNECHT, S. E. LITTLE, AND W. R. DAVIDSON. 2006. Evaluation of white-tailed deer (Odocoileus virginianus) as natural sentinels for Anaplasma phagocytophilum. *Vector-Borne and Zoonotic Diseases* 6(2):192-207.
- ENSERINK, M. 2000. The enigma of West Nile. *Science* 290(5496):1482-1484.
- ETTER, D. R. 2001. Ecology and management of overabundant white-tailed deer from suburban Chicago, Illinois. Ph.D. Thesis. University of Illinois, Urbana-Champaign, 107 pp.
- FRIEND, M. 2006. Disease emergence and resurgence: The wildlife-human connection. USGA National Wildlife Health Center in cooperation with the U.S. Fish and Wildlife Service, 387 pp.
- GEHRT, S. D., C. ANCHOR, AND L. A. WHITE. 2009. Home range and landscape use of coyotes in a metropolitan landscape: Conflict or coexistence? *Journal of Mammalogy* 90(5):1045-1057.
- HALES, S., N. DE WET, J. MAINDONALD, AND A. WOODWARD. 2002. Potential effect of population and climate changes on global distribution of dengue fever: An empirical model. *Lancet* 360(9336):830-834.
- HESS, G. 1994. Conservation corridors and contagious disease: A cautionary note. *Conservation Biology* 8:256-262.
- _____. 1996. Disease in metapopulation models: Implications for conservation. *Ecology* 77:1617-1632.
- HUDSON, P. J., A. RIZZOLI, B. T. GRENFELL, J. A. P. HEESTERBEEK, AND A. P. DOBSON. 2001. Ecology of wildlife diseases. Oxford University Press, Oxford, 197 pp.

- JONES, K. E., N. G. PATEL, M. A. LEVY, A. STOREYGARD, D. BALK, J. L. GITTLEMAN, AND P. DASZAK. 2008. Global trends in emerging infectious diseases. *Nature* 451:990-993.
- JENKINS, S. R. AND W. G. WINKLER. 1987. Descriptive epidemiology from an epizootic of raccoon rabies in the middle Atlantic States, 1982-82. *American Journal of Epidemiology* 126(3):429-437.
- KOMAR, N. 2001. West Nile virus surveillance using sentinel birds. *Annals New York Academy of Science* 951:58-73.
- LAU, S. K. P., P. C. Y. WOO, K. S. M. LI, Y. HUANG, H. W. TSOI, B. H. L. WONG, S. S. Y. WONG, S. Y. LEUNG, K. H. CHAN, AND K. Y. YUE. 2005. Severe acute respiratory syndrome coronavirus-like virus in Chinese horseshoe bats. *Proceedings of the National Academy of Science USA* 102(39):14040-14045.
- LEIGHTON, F. A. AND T. KUIKEN. 2001. Leptospirosis *In* *Infectious diseases of wild mammals*, E. S. Williams and I. K. Barker (eds). Iowa State University Press, Ames, Iowa, pp. 498-502.
- LI, W., Z. SHI, M. UY, W. RNE, C. SMITH, J. H. EPSTEIN, H. WANG, G. CRAMERI, Z. HU, H. ZHANG, J. ZHANG, J. MCEACHERN, H. FIELD, P. DASZAK, B. T. EATON, S. ZHANG, AND L. F. WANG. 2005. Bats are natural reservoirs of SARS-like Coronaviruses. *Science* 310:676-679.
- MCCALLUM, H. 2008. Landscape structure, disturbance, and disease dynamics. *In* *Infectious disease ecology: The effects of ecosystems on disease and of disease on ecosystems*, R. S. Ostfeld, F. Kessing, and V. T. Eviner (eds). Princeton University Press, Princeton, New Jersey, pp. 100-122.

- MCLEAN, R. G. 2008. The introduction and emergence of wildlife diseases in North America. *In* Wildlife science: Linking ecological theory and management implications, T. E. Fulbright and D. G Hewitt (eds). CRC Press, Boca Raton, Florida, pp. 261-278.
- NATIONAL RESEARCH COUNCIL. 1991. Animals as sentinels of environmental health hazards. National Academy Press, Washington D. C., 160 pp.
- NEWMAN, S. H., J. H. EPSTEIN, AND L. M. SCHLOEGEL. 2005. The nature of emerging zoonotic diseases: Ecology, prediction, and prevention. *Medical Laboratory Observer* 37(7):10-19.
- POWER, A. G. AND C. E. MITCHELL. 2004. Pathogen spillover in disease epidemics. *American Naturalist* 164:S79-S89.
- 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(2):324-333.
- REED, K. D, J. W. MELSKI, M. B. GRAHAM, R. L. REGNERY, M. J. SOTIR, M. V. WEGNER, J. J. KAZMIERCZAK, E. J. STRATMAN, Y. LI, J. A. FAIRLEY, G. R. SWAIN, V. A. OLSON, E. K, SARGENT, S. C. KEHL, M. A. FRACE, R. KLINE, S. L. FOLDY, J. P. DAVIS, AND I. K. DAMON. 2004. The detection of monkeypox in humans in the western hemisphere. *New England Journal of Medicine* 350:342-350.
- SCHMITT, S. M., S. D. FITZGERALD, T. M. COOLEY, C. S. BRUNING-FANN, L. SULLIVAN, D. BERRY, T. CARLSON, R. B. MINNIS, J. B. PAYEUR, AND J. SIKARSKIE. 1997. Bovine tuberculosis in free-ranging white-tailed deer from Michigan. *Journal of Wildlife Diseases* 33:749-758.

- SLINGENBERGH, J., M. GILBERT, K. DE BALOGH, AND W. WINT. 2004. Ecological sources of zoonotic diseases. *Scientific and Technical Review International Office of Epizootics* 23(2):467-484.
- THOMPSON, C., A. SPIELMAN, AND P. J. KRAUSE. 2001. Coinfecting deer-associated zoonoses: Lyme disease, babesiosis, and ehrlichiosis. *Clinical Infectious Diseases* 33(5):676-685.
- TRAINER, D. O. 1973. Wildlife as monitors of disease. *American Journal of Public Health* 63(3):201-203.
- U. S. CENSUS BUREAU. 2000. Annual estimates of the population of metropolitan and micropolitan statistical areas.
- VERCAUTEREN, K. C., T. C. ATWOOD, T. J. DELIBERTO, H. J. SMITH, J. S. STEVENSON, B. V. TOMSEN, T. GIDLEWSKI, AND J. PAYEUR. 2008. Sentinel-based surveillance of coyotes to detect bovine tuberculosis, Michigan. *Emerging Infectious Diseases* 14(12):1862-1869.
- WINKLER, W. G. 1975. Fox rabies. *In* The natural history of rabies, G. M. Baer (ed). Academic Press, New York, pp. 3-22.
- WISE, J. K., B. L. HEATHCOTT, AND M. L. GONZALEZ. 2002. Results of the AVMA survey on companion animal ownership in the US pet-owning households. *Journal of the American Veterinary Medical Association* 221(11):1572-1573.
- WITHAM, J. H. AND J. M. JONES. 1990. White-tailed deer abundance on metropolitan forest preserves during winter in northeastern Illinois. *Wildlife Society Bulletin* 18:13-16.
- WOOLHOUSE, M. E. J. AND S. GOWTAGE-SEQUERIA. 2005. Host range and emerging and reemerging pathogens. *Emerging Infectious Diseases* 11(12):1842-1847.

WORLD TOURISM ORGANIZATION. 2009. Tourism highlights 2009. 12 pp.

YABSLEY, M. J., V. G. DUGAN, D. E. STALLKNECHT, S. E. LITTLE, J. M. LOCKHART, J. E. DAWSON, AND W. R. DAVIDSON. 2003. Evaluation of a Prototype Ehrlichia chaffeensis surveillance system using white-tailed deer (Odocoileus virginianus) as natural sentinels. Vector-Borne and Zoonotic Diseases 3(4):195-207.

GENERAL MATERIALS AND METHODS

Study area

The study areas encompassed >27,499 ha (11% land cover) of forest preserves in suburban Chicago, Illinois (41°85' N, 87°65' W) owned and managed by the Forest Preserve District of Cook County (FPDCC). The forest preserves include over 200 picnic areas, 161 km of bike trails, lakes, rivers, and 323 km of multiuse trails. The study included 11 managed tracts within the FPDCC including Bemis (437 ha), Black Partridge (177 ha), Busse (1508 ha), Camp Sagawau (24 ha), Crab Tree (668 ha), Des Plaines (DP; 1168 ha), Palos (1178 ha), Popular Creek (1204 ha), Somme (201 ha), Swallow Cliff (959 ha), and Zander-Jurgenson (815 ha) preserves (C. Anchor, pers. comm.). Land cover in the region varied including urbanization, urban grassland, forested woodland, cropland, rural grassland, wetland, and open water.

Witham and Jones (1987) estimated deer densities in Busse and DP exceeded 50 deer/km² in 1985-86. Aerial surveys in Cook County conducted from 1985-99 suggested minimum deer estimates of >23 deer/km² (FPDCC, unpublished data). Etter et al. (2000) estimated aerial surveys only detect approximately 2/3 of the deer within suburban forest preserves; therefore deer densities were likely greater. Aerial surveys suggest populations were at high-density indicating a population at carrying capacity (Etter et al. 2000; Piccolo et al., 2010).

Regional climate is temperate, consisting of warm, humid summers and cold winters. The average high daily temperature is 28 °C during the midsummer months and -10.4 °C as the low in January. Mean annual rainfall is 84.9 cm and annual snowfall is 97.3 cm (Mapes, 1979).

Deer capture and culling

Deer were captured with drop-nets (Wildlife Materials Inc., Carbondale, Illinois, USA;

Ramsey, 1968) or remote dart gun (Pneu-Dart Inc., Williamsport, Pennsylvania, USA; Kilpatrick et al., 1997) from December to March (1995-1998) from the two primary study sites DP and Palos (Fig. 1). Netted deer were anesthetized with xylazine hydrochloride (2.0 mg/kg Cervazine[®], Wildlife Pharmaceuticals Inc., Fort Collins, Colorado, USA) and darted deer with tiletamine/zolazepam hydrochloride (4.4 mg/kg Telazol[®], Fort Dodge Laboratories, Fort Dodge, Iowa, USA) and 2.0 mg/kg xylazine hydrochloride. The xylazine hydrochloride was reversed with yohimbine hydrochloride (0.25 mg/kg Antagonil[®], Wildlife Pharmaceuticals Inc., Fort Collins, Colorado, USA; A. DeNicola, White Buffalo Inc., pers. comm.). Additionally, deer were culled from all study sites (Fig. 1) by state certified sharp shooters as part of a management program. Deer were removed strategically from approved bait sites and throughout the forest preserves from accessible trails. Culling sites were selected based upon deer use, sharp shooter accessibility, and human safety. All sites were approved by the Illinois Department of Natural Resources (IDNR). Deer were removed from bait sites and opportunistically from trails when traveling between sites. To maximize population reduction efforts, females were preferred during culling.

All live-captured deer (tagged and radio-collared) were marked with two numbered plastic ear tags for visual identification and with metal ear tags with FPDCC return information. Selected female deer were fitted with radio-collars equipped with an 8-hour, time delayed, mortality switch (Advanced Telemetry Systems, Isanti, Minnesota, USA; Telonics, Mesa, Arizona, USA; Etter et al., 2002) to examine female social behavior and population dynamics. Physiological information (age and sex) was recorded from all live-captured and culled deer. Locations were recorded on topographic images and later transferred to ArcView[®] 3.2 (Environmental Systems Research Institute Inc., Redlands, California, USA; ESRI, 1996)

geographic information system (GIS). Deer age was determined for live-captured deer by tooth replacement and wear as fawn (<1 year old), yearling (1-2 years old), or adult (≥ 2 years old; Severinghaus, 1949). For culled deer, age was determined by cementum annuli (Matson's Laboratory, Milltown, Montana, USA; Gilbert, 1966). Blood was collected (≤ 45 cc) from carcasses of culled deer. Blood was collected from the jugular vein (25-40 cc) from live-captured deer. Blood samples were centrifuged to separate sera from whole blood and stored at -60°C .

Radio-telemetry

All radio-collared females were radio-tracked to examine home ranges and movement patterns. In addition, radio-telemetry data allowed for inferences to culled deer about home range size and habitats use. To describe home range characteristics for radio-tracked deer, radio-locations were attempted 1-3 times per week using two truck mounted, 4-element yagi antennas, aligned in a null configuration (Nixon et al., 1991). All telemetry data were entered into in a spreadsheet and transferred in Locate II[®] software 1.3 (Pacer, Truro, Nova Scotia, Canada; Nams, 1990). To determine deer locations, the maximum likelihood estimator was used from ≥ 3 radio-bearings collected within a 20 minute interval. The 90th percentile of deer location error ellipses (11.5 ha) was used as the upper limit for acceptable error and locations with larger error were deleted (Etter et al., 2002). Ninety-five and 50% minimum convex polygon (MCP) home ranges were generated in software CALHOME[®] (Fortran 5.0, U. S. Forest Service, Pacific Southwest Research Station, California, USA; Kie et al., 1994).

Serology

For detection of Jamestown Canyon (JC) and LaCrosse (LAC) encephalitis zoonotic pathogen antibodies, sera were initially tested by CAL group virus IgG captured enzyme linked

immunosorbent assay (ELISA) (Grimstad et al., 1984; Godsey, 1995-1996 samples, Centers for Disease Control (CDC), Fort Collins, Colorado, USA; Grimstad, 1997-1999 samples, University of Notre Dame, Notre Dame, Indiana, USA). Samples were determined as positive for CAL group antibodies (JC and LAC) when the mean absorbance for the three test wells was ≥ 2 times the mean of the negative control. Samples were retested if positive for both CAL groups using plaque-reduction neutralization assays (PRNA) with serum dilutions of 1:20-1:160 (Pantuwatana et al., 1972; Grimstad et al., 1984). The PRNA titers were compared to differentiate closely related JC and LAC viruses. The higher antibody titer was used to indicate virus specificity when >2 fold difference in antibody titer was detected against closely related viruses (Boromisa and Grimstad, 1987; Grimstad et al., 1987; Neitzel and Grimstad, 1991; Osorio et al., 1996). Samples were classified as non-determined when testing was <2 fold titer difference for a positive result. All serological testing for deer was completed by laboratories with the highest gold standard to reduce false positive and false negative results.

All culled venison was processed and donated to local food depositories (Good Samaritan Act, House Bill 3412, 1991-1992). The University of Illinois Laboratory Animal Care Advisory Committee reviewed and approved all methods (Protocol V5R246 and V5R246/8340). Deer were collected and captured under Illinois Department of Natural Resources (IDNR) and FPDCC ecological study permits.

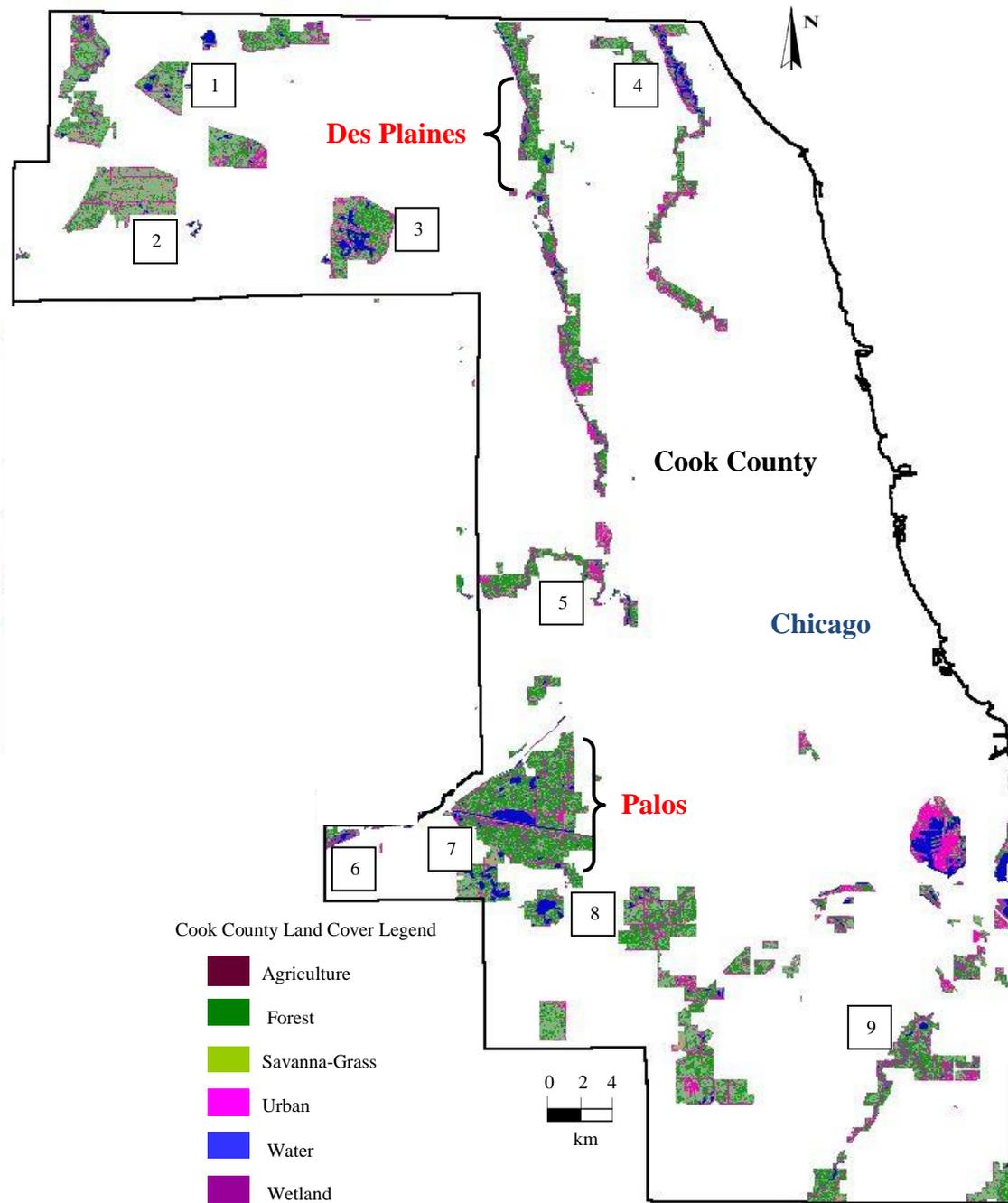


FIGURE 1. Map of Cook County Forest Preserves in Chicago, Illinois, USA, 1995-99. Deer samples were collected from primary study sites Des Plaines and Palos. Additional culled samples were collected from 1=Crab Tree, 2=Poplar Creek, 3=Busse, 4=Somme, 5= Bemis, 6= Black Partridge, 7=Camp Sagawau, 8=Swallow Cliff, and 9=Zander-Jurgenson study sites.

LITERATURE CITED

- BOROMISA, R. D. AND P. R. GRIMSTAD. 1987. Seroconversion rates to Jamestown Canyon virus among six populations of white-tailed deer (Odocoileus virginianus) in Indiana. *Journal of Wildlife Diseases* 23(1):23-33.
- ESRI (ENVIRONMENTAL SYSTEMS RESEARCH INSTITUTE). 1996. ArcView® GIS: The geographic information system for everyone. Redlands, California.
- ETTER, D. R., K. M. HOLLIS, T. R. VAN DEELEN, D. R. LUDWIG, J. E. CHELSVIG, C. L. ANCHOR, AND R. E. WARNER. 2002. Survival and movements of white-tailed deer in suburban Chicago, Illinois. *Journal of Wildlife Management* 66(2):500-510.
- _____, T. R. VAN DEELEN, D. R. LUDWIG, S. N. KOBEL, AND R. E. WARNER. 2000. Management of white-tailed deer in Chicago, Illinois forest preserves. *In Proceedings 19th Vertebrate Pest Conference*, T. P. Salmon and C. Crabb (eds). University of California, Davis, California, pp. 190-196.
- GILBERT, F. F. 1966. Aging white-tailed deer by annuli in the cementum of the first incisor. *Journal of Wildlife Management* 30(1):200-202.
- GRIMSTAD, P. R., C. L. BARRETT., R. L. HUMPHREY, AND M. J. SINSKO. 1984. Serological evidence for wide-spread infection with LaCrosse and St. Louis encephalitis viruses in the Indiana human population. *American Journal of Epidemiology* 119:913-930.
- GRIMSTAD, P. R., D. G. WILLIAMS, AND S. M. SCHMITT. 1987. Infection of white-tailed deer (Odocoileus virginianus) in Michigan with Jamestown Canyon virus (California Serogroup) and the importance of maternal antibody viral maintenance. *Journal of Wildlife Diseases* 23(1):12-22.

- KIE, J. G., J. A. BALDWIN, AND C. J. EVANS. 1994. CALHOME[®]: Home range analysis program. Electronic user's manual. United States Forest Service, Fresno, California.
- KILPATRICK H. J., S. M. SPOHR, AND A. J. DENICOLA. 1997. Darting urban deer: Techniques and technology. *Wildlife Society Bulletin* 25(2):542-546.
- MAPES, D. R. 1979. Soil survey of DuPage and part of Cook counties, Illinois. Illinois Agricultural Experiment Station Report 108, Urbana, Illinois.
- NAMS, V. O. 1990. Locate II[®] user's guide. Pacer, Truro, Nova Scotia, 82 pp.
- NEITZEL, D. F. AND P. R. GRIMSTAD. 1991. Serological evidence of California group and Cache Valley virus infection in Minnesota white-tailed deer. *Journal of Wildlife Diseases* 27(2):230-237.
- NIXON, C. M., L. P. HANSEN, P. A. BREWER, AND J. E. CHELSVIG. 1991. Ecology of white-tailed deer in an intensively farmed region of Illinois. *Wildlife Monographs* 118, 77 pp.
- OSORIO, J. E., M. S. GODSEY, G. R., DEFOLIART, AND T. M. YUILL. 1996. LaCrosse viremias in white-tailed deer and chipmunks exposed by injection or mosquito bite. *American Journal of Tropical Medicine and Hygiene* 54(4):338-342.
- PANTUWATANA, S., W. H. THOMPSON, D. M. WATTS, AND R. P. HANSON. 1972. Experimental infection of chipmunks and squirrels with LaCrosse and Trivittatus viruses and biological transmission of LaCrosse virus by Aedes triseriatus. *American Journal of Tropical Medicine and Hygiene* 21(4):476-481.
- PICCOLO, B.P., T. R. VANDEELEN, K. HOLLIS-ETTER, D. R. ETTER, R. E. WARNER, AND C. ANCHOR. 2010. Behavior and survival of white-tailed deer neonates in 2 suburban forest preserves. *Canadian Journal of Zoology* 88:487-495.
- RAMSEY, C. W. 1968. A drop-net deer trap. *Journal of Wildlife Management* 32(1):187-190.

SEVERINGHAUS, C. A. 1949. Tooth development and wear as criteria of age in white-tailed deer. *Journal of Wildlife Management* 13:195-216.

WITHAM, J. H. AND J. M. JONES. 1987. Chicago Urban Deer Study. Illinois Natural History Survey Report No. 265, Champaign, Illinois.

CHAPTER 1

SEROLOGICAL SURVEY OF SUBURBAN DEER

ABSTRACT: The presence and abundance of vertebrates influences the circulation of zoonotic diseases. White-tailed deer (Odocoileus virginianus) are widely distributed in North America and deer densities are frequently high in un hunted areas, including most major metropolitan regions. This study investigated the seroprevalence of four disease agents from live-captured and culled deer sampled in two suburban forest preserves around Chicago, Illinois from 1995-99. Seroprevalence for Toxoplasma gondii was 55.9% (n=443), Leptospira spp. 16.2% (n=444), Jamestown Canyon (JC) virus 38.1% (n=404), and LaCrosse (LAC) virus 6.7% (n=404). Prevalence was higher for adult deer for all pathogens sampled except Leptospira spp. Prevalence was less likely in males for JC virus. A higher prevalence for T. gondii and JC virus was detected at Des Plaines (DP) and prevalence for Leptospira spp. was less likely at DP. Differences in year the sample was collected were present for T. gondii, Leptospira spp., and JC virus. Variation in month the samples were collected was evident for T. gondii, Leptospira spp., and LAC virus. Multivariate logistic regression (LR) screened main effects variables (age, sex, site, year, and month) by backward stepwise elimination ($p \leq 0.20$). The final LR model for T. gondii contained all main effects variables. Leptospira spp. and JC viral models both contained age, site, and year, while the final LAC viral LR model contained age, sex, and month. Deer are an appropriate wildlife species for this investigation because they inhabit the same environment as disease vectors and humans who recreate in suburban parks. Chicago area deer are indicators for zoonoses as evidenced by the prevalence rates in this study. This information will serve as baseline data for prevalence of four pathogens providing information to public health officials.

INTRODUCTION

Wild mammals can serve as hosts, amplifiers, or reservoirs for various zoonotic diseases (Day et al., 1996). Serological surveys of white-tailed deer (Odocoileus virginianus) have examined the presence of antibodies against many zoonotic agents (Thomas and Trainer, 1970; Hoff et al., 1973; Adrian and Keiss, 1977; Acha and Szyfres, 1994; Dawson et al., 1994). Deer populations can be pathogenic hosts, reservoirs, or amplifiers and have been considered a link in zoonotic disease emergence (Daszak et al., 2001). Open deer populations have direct contact with the environment and are considered a sentinel pathogen species because of their wide distribution, abundance, and sedentary behavior (Trainer and Hanson, 1969).

Toxoplasmosis is a zoonotic infection caused by the protozoan parasite Toxoplasma gondii. It is an obligate intracellular protozoan parasite that may cause abortion in domestic animals and wildlife. The oocyst readily infects humans, including approximately 30% of the population depending on age and environment, but clinical illness is relatively uncommon. Those at risk of developing clinical illness include pregnant women, as the parasite can pose a serious threat to the unborn child and individuals who are immunosuppressed. The two most likely sources of human infection are ingestion of raw or rare cooked meat containing live T. gondii tissue cysts from intermediate hosts like deer or exposure to oocysts derived from cat (Felis catus) feces found in gardens, litter boxes, and children's sand pits (Dubey, 2010).

Leptospirosis is a contagious disease caused by the bacterial spirochete Leptospira. The organism is transmitted through contaminated urine from animals to humans in water, food, or soil. The common route of entry in humans is oral (food and/or water) but may also occur through the skin (Acha and Szyfres, 1994; Benenson, 1995). Many different animals serve as a host for the bacterium and may be asymptomatic or sick. Leptospira organisms have been found

in cattle, pigs, horses, dogs, rodents, and wild animals.

Arboviruses (arthropod-borne viruses), like Jamestown Canyon (JC) and LaCrosse (LAC) viral encephalitis, are spread by Aedes mosquitoes in the eastern United States (Grimstad, 1989). Jamestown Canyon virus persists in cycles of infection among wild ungulates, especially deer as the primary reservoir host (Andreadis et al., 2008). Jamestown Canyon virus is maintained in a variety of different ecological habitats where ungulates are present and in several mosquito spp. with a wide range of habitats and geographical regions. Grimstad et al. (1984) reported humans' seropositive for JC in Michigan. LaCrosse virus circulates between small mammalian hosts and specifically tree-hole mosquitoes, A. triseriatus. The mosquito breeds in forested areas or containers conducive for reproduction. Deer are amplifiers for LAC viral transmission once it has been established (Clark et al., 1983; Osorio et al., 1996). LaCrosse virus has been reported to produce central nervous system infections in humans (Grimstad et al. 1984).

Long-term monitoring for zoonoses can indicate transmission risks for humans, endemic patterns in a population, and/or disease presence. Deer seroprevalence can serve to inform resource managers and public health officials about the risk of zoonoses in a targeted surveillance area. I investigated seroprevalence patterns for T. gondii, Leptospira spp., JC virus, and LAC virus in white-tailed deer (live-captured and culled) in suburban Des Plaines (DP) and Palos forest preserves in Cook County, Illinois from 1995-99. This information will serve as baseline data for prevalence of four pathogens providing information to public health officials.

MATERIALS AND METHODS

General materials and methods are specified in pages 14-21.

Samples were collected from a more extensive network of forest preserves throughout the Chicago region. However, DP and Palos were the preferred sites for this chapter analysis due to

samples from live-captured deer and large samples sizes from culled deer. Additional samples and sites collected by pathogen are included in Appendix A-L.

Samples were tested for T. gondii by Dr. J. P. Dubey at the United States Department of Agricultural (USDA) Research Center in Beltsville, Maryland, USA. Serum agglutination tests for T. gondii were examined at dilutions of 1:25, 1:50, 1:500, and ≥ 500 (Dubey and Desmonts, 1987). Samples were classified as positive if the titer was $\geq 1:25$.

Samples for six L. interrogans serovars (bratislava, canicola, grippotyphosa, hardjo, icterohaemorrhagiae, and pomona) were analyzed by the Minnesota Veterinary Diagnostic Laboratory at the University of Minnesota, St. Paul, Minnesota, USA. Microscopic serum agglutination (MAT) to six common leptospire strains were tested and a four-fold rise in antibody titers determined diagnostic positive animals (Cole et al, 1979; 1983). Samples were tested at dilutions of 1:100, 1:200, 1:400, and 1:800. Samples were classified as positive if the titer was $\geq 1:100$.

Univariate analyses were used to detect differences in prevalence by serological outcome for age, sex, site, and collection period (year, month; Crosstabs in SPSS[®] 17.0, Chicago, Illinois, USA; Rothman, 1986). Pearson chi-square statistics ($p \leq 0.05$) and Cochran-Mantel-Haenszel (CMH) odds ratios (OR) were used to examine significant relationships (Fleiss, 1981). Biological parameters were stratified (controlled) by age and sex. Multivariate logistic regression (LR) was used to evaluate deer parameters for each serological outcome (Nash et al., 1995). The initial models included age, sex, site, and collection period (year, month). The variables were screened by backward stepwise elimination to remove main effect variables ($p \leq 0.20$) which did not significantly affect the likelihood statistics or the magnitude of remaining variables (Hosmer and Lemeshow, 1989; Kleinbaum, 1994). Hosmer and Lemeshow goodness

of fit (GOF) statistic was used to determine model fit (Hosmer and Lemeshow, 1989). Biologically relevant interactions of main effects variables were also evaluated. Receiver operating characteristic (ROC) curves were used to plot the sensitivity values (true positive fraction) versus the equivalent 1-specificity values (false positive fraction) for final logistic regression model variables (Hanley and McNeil, 1982). Area under the curve (AUC) was used as a measure of model accuracy and is evaluated from 0-1 with 1 indicating perfect discrimination (Viña et al., 2010).

RESULTS

Overall seroprevalence for T. gondii was 55.9% (n=443), Leptospira spp. 16.2% (n=444), Jamestown Canyon (JC) virus 38.1% (n=404), and LaCrosse (LAC) virus 6.7% (n=404). Field data and/or sera analyses were incomplete for some deer and could not be included for all seroprevalence analyses. The R^2 and AUC values indicate minor variation in the models. However, the results and discussion also consider the biological parameters for data typically contained in open populations of large game species and noisy ecological systems.

Prevalence for T. gondii increased with age ($\chi^2=40.86$, 2 df, $p<0.001$; Table 1). There was a higher prevalence by site for DP (OR=2.97, CI=2.02-4.39, $\chi^2=30.97$, $p<0.001$). Seroprevalence differed by year ($\chi^2=11.26$, 3 df, $p=0.01$) favoring 1996-97 and 1997-98 and month ($\chi^2=11.52$, 3 df, $p=0.009$) being higher in December and January. The final LR model included age (beta= -0.830, SE=0.134), sex (beta=0.455, SE=0.238), site (beta=0.70, SE=0.014), year (beta=-0.201, SE=0.105), and month (beta=-0.080, SE=0.037; $R^2=0.214$, GOF=0.544, 5df, $p<0.001$). Area under the curve values for the final LR model were ≥ 0.50 except study site=0.37 (Table 2). Interaction terms did not contribute any biological significance beyond the final LR model.

Leptospira spp. seroprevalence varied by site (OR=0.59, CI=0.35-0.98, $\chi^2=4.20$, $p=0.04$; Table 3) and was less likely in DP. Seroprevalence also differed by year ($\chi^2=47.73$, 3 df, $p<0.001$) being higher in 1998-99 and month ($\chi^2=7.84$, 3 df, $p=0.05$) was higher in March. The most prevalent serovar found in deer from the two study sites was L. interrogans grippotyphosa (14.6%). The final LR model included age (beta= -0.448, SE=0.171), site (beta=-0.041, SE=0.018), and year (beta=-0.791, SE=0.147; $R^2=0.152$, GOF=0.037, 3 df, $p<0.001$). Area under the curve values for the final LR model were ≥ 0.50 (Table 2). Interactions of main effects variables did not contribute to any significant multivariate models.

Prevalence for JC virus was higher for adult deer ($\chi^2=75.47$, 2 df, $p<0.001$) and less likely for males (OR=0.56, CI=0.36-0.86, $\chi^2=6.59$, $p=0.01$; Table 4). There was also a difference by site favoring DP (OR=1.76, CI=1.17-2.65, $\chi^2=7.47$, $p=0.006$). Seroprevalence varied among years ($\chi^2=28.17$, 3 df, $p<0.001$) and was higher in 1996-97 and 1997-98. The final LR model included age (beta= -1.237, SE=0.162), site (beta=0.029, SE=0.015), and year (beta=-0.150, SE=0.116; $R^2=0.258$, GOF=0.001, 3 df, $p<0.001$). Area under the curve values for the final LR model were ≥ 0.50 except for study site=0.43 (Table 2). None of the interaction terms were significant.

LAC viral prevalence increased by age ($\chi^2=8.52$, 2 df, $p=0.01$; Table 5) and by month ($\chi^2=9.16$, 3 df, $p=0.03$) with higher prevalence detected in January and February. The final LR model included age (beta= -0.724, SE=0.319), sex (beta=-0.678, SE=0.522), and month (beta=0.253, SE=0.148; $R^2=0.094$, GOF=0.202, 3 df, $p=0.002$). Area under the curve values for the final LR model were ≥ 0.50 except for month=0.35 (Table 2). There were no significant interaction terms.

DISCUSSION

The advantage of any wildlife zoonoses monitoring program is early detection of new and emerging diseases, some with serious disease and economic implications. Wild animals, like deer, inhabit natural environments where they have direct contact with vectors and may be exposed to zoonoses. I took advantage of an existing deer culling program and samples from collared deer to investigate prevalence for zoonoses in a suburban environment. Deer were an appropriate wildlife species for this investigation because they inhabit the same environments as disease vectors and humans who recreate in suburban parks and forest preserves. Chicago area deer are indicators for zoonotic agents as evidenced by prevalence rates for T. gondii, Leptopira spp., JC virus, and LAC virus in this study.

White-tailed deer are herbivores and may become exposed and/or infected with toxoplasmosis by ingesting plants, soil, or contaminated water containing the oocysts (Humphreys et al., 1995). Toxoplasma oocysts have been recovered from oak-hickory woodland habitats in Missouri and prevalence is higher in warm, humid climates (Acha and Szyfres, 1994; Smith and Frenkel, 1995). Smith and Frenkel (1995) reported 11% (n=117) of the herbivores were positive for the parasite from Missouri and Kansas during a mammal study. The prevalence for T. gondii was higher (60.2%) in hunter killed deer in 1991 in Pennsylvania compared to this study (55.9%; Humphreys et al., 1995).

The trend of increasing T. gondii with age (Table 1) is expected due to continual exposure of oocysts by long-lived animals, like suburban deer. Increased prevalence in adult deer indicates pathogen presence, long-term, and/or repeated exposures. It is likely that increased prevalence in yearlings is due to new annual exposures to the oocyte. Prevalence in black-tailed deer (Odocoileus hemionus) from northern California ranged from 11% in fawns,

40% in yearlings, and 30.6% in adults (Franti et al., 1975). Ferreira et al. (1997) found the prevalence for the organism in marsh (Blastocercus dichotomus) and pampas (Ozotocercus bezoarticus) deer was 13% in fawns, 32% in yearlings, and 27% in adults. The results from this study indicate a higher prevalence in all age groups for suburban deer compared to elsewhere, likely because of high domestic cat populations, increased deer densities, and persistence of the parasite over time. Felines (i.e., domestic and feral cats) are the definitive host while actively shedding the parasite. In Chicago, domestic cat populations are estimated at 1.5 million or more (Wise et al., 2002). Feral cat populations also pose a potential health risk for infecting people with T. gondii (Jessup, 2004). In a study from California, outdoor fecal deposition by free-roaming domestic cats was estimated at 69.31 metric tons per year and an additional 26.76 metric tons per year by feral cats (Dabritz et al., 2006). Many feral cats were observed in the forests preserves during this study (C. Anchor, J. Chelsvig, and D. Etter, pers. comm.). The environmental contamination from cat feces is a concern for human health (Jessup, 2004). Witham and Jones (1987) stated deer densities in Busse and DP forest preserves exceeded 50 deer/km² or higher. Many other species of warm-blooded mammals (i.e., raccoons, Procyon lotor; coyotes, Canis latrans) found in Chicago can also serve as intermediate hosts for the oocysts. The parasites also have the ability to remain infective in water or moist soil while surviving various environmental conditions, including heat, freezing, and humidity for up to 18 months (Frenkel et al., 1975). All these conditions contributed to the increased prevalence for the parasite by age in suburban white-tailed deer.

Difference in prevalence by site (Table 1) may be due to differences in abundance of the definitive host for toxoplasmosis, domestic or feral cats. Toxoplasmosis antibodies have been found in more than 63% of non-domestic mammals in the presence of cats (Dubey et al, 1995).

The DP forest preserve is surrounded by residential homes and commercial development while Palos has very little residential development directly adjacent to the preserve.

Hunter killed deer from Minnesota serologically examined for Toxoplasma did not reveal any difference by year (Vanek et al., 1996). However, temporal trends (year and month) were revealed in this study (Table 1) and can be used as indicators for oocyst exposure(s). Change in prevalence among years will provide information relevant to increases or decreases in annual parasitic presence. Annual variation in human serology for toxoplasmosis from Ontario, Canada was not detected (Tizard et al., 1977) due to human's constant exposure to domestic cats. Month the sample was collected indicates that deer are more likely to have measureable positive titers in early winter months rather than later (Table 1). Seasonal transmission (spring and summer) was identified in skunks (Mephitis mephitis) from Canada by a predominance of high titer levels collected from animals in April-September (Schowalter et al., 1980). Experimental exposures for cattle and elk (Cervus canadensis) indicate measurable antibody levels within 28 days or less (Beverley et al., 1977; Costa et al., 1977; Dubey et al., 1980). However, research on experimental infection dynamics in white-tailed deer is limited.

The multivariate model for T. gondii also included sex. Differences in sex may be attributed to differences in home range size between the sexes (Nixon et al., 1991). Female deer from the Chicago region maintained smaller home ranges (Etter et al., 2002); therefore increasing potential repeated exposures to foci in contaminated soil, vegetation, and water sources. Additionally, sampling for deer (live-captured or culled) in this study favored females due to population control and social behavior study goals.

Leptospire cannot survive in acidic urine, therefore herbivores and animals with alkaline urine are more important carriers of the organism (Faine, 1994). Overall prevalence for

Leptospira spp. (Table 3) in deer from Chicago forest preserves (16.4%) was similar to previously reported rates. The prevalence for antibody titers to leptospire in deer populations in North America varies between 7-27% (Wedman and Driver, 1957; Ferris et al., 1961; Shotts and Hayes, 1970; Fournier et al.1986). In this study, the common leptospire grippotyphosa was identified in deer samples. Domestic dogs (Canis familiaris) and cats have predominant serovars for canicola and icterohaemorrhagiae (Acha and Szyfres, 1994). Raccoons and skunks have been documented as a natural reservoir for this common pathogenic leptospire and 48% (n=459) of raccoons sampled in rural Illinois were positive for grippotyphosa (Shotts et al., 1975; Mitchell et al., 1999). Raccoon densities are high in the Chicago region, documented as 8-12 times higher than rural areas (Gehrt, 2005). Skunks examined from Busse forest preserve in Chicago, Illinois had low prevalence for L. interrogans (17%) and are unlikely to be a primary reservoir for the spirochete, even though skunk populations are high (Gehrt et al., 2010). Other rural deer studies have reported various serovars including canicola, grippotyphosa, hardjo, and pomona (Clark et al., 1961; Goyal et al., 1992; New et al., 1993; Leighton and Kuiken, 2001). Deer positive for this strain of leptospirosis is evidence for environmental exposure and interspecies transmission within preserves commonly used by humans.

Site specific differences in prevalence for Leptospira spp. (Table 3) may be due in part to differences in water sources between preserves. The risk of infection rates increases with high rainfall and areas along river corridors and flood plains with poor drainage (Heath and Johnson, 1994). Raccoons frequent areas with standing water while hunting for aquatic foods and are known to actively shed the bacterial spirochete in urine (Mitchell et al., 1999). Even though DP encompasses a river corridor, Palos contained numerous lakes and wetlands without constant free-flowing water. These standing water bodies were more likely to be contaminated with

grippotyphosa and create an environment where the organism may be more persistent and focal for deer in Palos compared to DP. Additionally, deer from Palos selected for wetland habitats more than DP when habitat use was compared to habitat availability (Hollis, 2011). Humans and pets are at higher risk of infection from leptospirosis during warmer weather and after heavy rains with standing water (Acha and Szyfres, 1994).

Prevalence by year and month (Table 3) for leptospire indicated sampling from deer in the winter can be used to identify positive animals. Experimental infection of deer with Leptospira indicated antibody seroconversion in blood samples in ~5 days with persistence for ~15 weeks, while deer may shed the organism in the urine for approximately 35 days post exposure (Trainer et al., 1961; Reilly et al., 1962). High prevalence of seropositive animals has been correlated to high mean annual temperatures in domestic cattle sampled during the summer, fall, and winter (Miller et al., 1991). In domestic dogs infected with leptospirosis, a seasonal distribution of late summer to fall and rainfall recordings three months prior to diagnosis were significant in the disease model (Ward et al., 2002). Prevalence for leptospirosis increased significantly from deer sampled in 1998-99. Further investigation of the data revealed 57% of the deer sampled from Palos were seropositive while 27% were positive from DP. Both rates from each site are higher than previously sampled years. I hypothesize that water supplies and/or other reservoir species (i.e., raccoons) influenced these results, however, additional data would need to be collected for analysis.

Age was included in the final logistic regression model. The older the animal is, the more likely it has experienced past and/or a repeated exposure, resulting in positive deer. Deer examined in Tennessee and Wisconsin also had a higher prevalence for adults than subadults (Trainer and Hanson, 1960; New et al., 1993).

“California group” (CAL) viruses include pathogenic agents to man such as JC and LAC (Grimstad, 1989; Acha and Szyfres, 1994). Transmission of these viruses often occurs in an endemic pattern among animals within a limited geographic region (Neitzel and Grimstad, 1991). Etter et al. (2002) determined that survivorship was high and dispersal low for deer from the Chicago region suggesting that populations within individual preserves were insular. High densities of deer may create a potential for increased activity of the viruses by increasing mosquito larval stages during the spring and late summer emergence and breeding cycles (Matschke et al., 1984; Grimstad et al., 1987). Deer densities were high on both areas during this study. Minimum winter aerial counts indicated 28.4-32.3 deer/km² at DP and 14.9-27.5 deer/km² at Palos (Forest Preserve District of Cook County, unpublished data); however, aerial counts typically detect <35% of deer present (Ludwig, 1981; Beringer et al., 1998) suggesting that densities were higher at both sites.

The prevalence rate for JC virus (38.1%) in this study falls between ranges previously reported. Prevalence for JC was 21% in hunter killed deer from Connecticut, 91% in Minnesota, and 56% in Wisconsin (Murphy, 1989; Neitzel and Grimstad, 1991; Zamparo et al., 1997). Additional studies reported on prevalence rates for LAC in deer from Wisconsin (16%) and Minnesota (6.9%; Issel et al., 1972b; Neitzel and Grimstad, 1991).

Jamestown Canyon and LAC viral seroprevalence was higher in adults (Tables 4 and 5). It is likely deer are maintaining measurable titers from regular exposure(s) to infected Aedes spp. for JC virus and A. triseriatus for LAC virus. Grimstad et al. (1987) reported 47% of adult does in Michigan were positive for JC while fawns had lower prevalence for antibodies. Only 2% of the adult deer sampled in Wisconsin in 1972 were positive for LAC (Issel et al., 1972b).

Prevalence was less likely to be found in males for JC virus (Table 4) and sex was

included in the final LR model for LAC virus (Table 5). Few male deer were collared and home ranges for males were not determined in this study, but rural male deer home ranges significantly exceed those of females (Nixon et al., 1991). Larger male home range sizes may reduce repeated exposures to focal areas of infected mosquitoes over time. The LAC tree-hole mosquito vector A. triseriatus does not venture far from a tree hole (<30m) and not above the forest canopy (Thompson, 1984).

Site differences for JC and LAC (Table 4 and 5) indicated habitat use and/or availability by deer are variable (Hollis, 2011), therefore creating variable habitats for vectors. Mosquito population fluctuations, time of vector emergence, and deer density may influence prevalence by site. Additionally, the DP site is along a riparian corridor which is known to fluctuate regularly during storm events. During dryer periods the water recedes forming pools and ideal breeding habitat for mosquitoes. Boromisa and Grimstad (1987) found differences in JC prevalence between northern and southern deer populations in Indiana. The differences were attributed to seasonal water pools and increased vector presence. Palos contains many lakes and wetlands, however water levels, even with seasonal fluctuations, create common mosquito breeding areas.

The difference in seroprevalence for JC virus by year (Table 4) and LAC virus by month (Table 5) confirms deer are antibody positive and can be tested in the winter for the pathogen. Grimstad et al. (1987) reported annual seroconversion of deer for JC overlapped with the spring emergence of aedine mosquitoes. Deer experimentally infected with JC had peak titers by day 5 and detectable antibody titers beyond 180 days (Issel et al., 1972a). Even human infections for LAC peak seasonally from July to September in central Illinois, reflective of infected mosquito activity (Kitron et al., 1997). Additionally, the role of LAC viral amplification by deer is not clear, because viremic levels are very short and not sufficient for infecting mosquitoes.

However, seasonal breeding cycles by mosquitoes are triggered by blood meals and deer can amplify LAC virus by providing this food resource (Clark et al., 1983; Osorio et al., 1996). Sampling deer over time could indicate a variation in mosquito presence, vertebrate exposure, and/or pathogen existence/cycles.

Deer are important monitors for examining zoonoses because they can be hosts, ecological amplifiers, or reservoirs (Day et al., 1996) for pathogen existence and emergence. Results from this study indicate deer are useful biomonitors while providing baseline information for pathogen exposure. An advantage of using deer is that samples are easily obtained from culled or hunted animals globally (Tataruch and Kierdorf, 2003). Deer as sentinels can provide information for emerging pathogen detection and continual screening for changes in zoonoses that are present. For example, fawn and/or yearling deer are indicators for immediate exposures to disease agents while adult deer can provide information about long-term, repeated exposures. Caution is advised when examining fawn exposures because of the exchange of maternal antibodies, which may alter interpretation of samples. Issel (1974) reported JC maternal antibodies in fawns from 8-23 weeks.

Stratifying a subsample from various deer populations in a region would provide an effective biomonitoring tool for specific pathogens. For example, deer could be sampled from specific locations for JC and LAC virus monitoring while considering specific mosquito species and mosquito habitat(s). This surveillance technique would provide specific data for potential disease reemergence relative to seasonal or annual variation in weather patterns and mosquito abundance. As mosquito species continue to be introduced into the United States, so are potential vectors. Furthermore, additional mosquito species (Anopheles, Culiseta, and Ochlerotatus) are being identified as positive for JC virus and increasing possible viral

transmitters in specific geographic locations (Andreadis et al. 2008). However it remains to be examined if all mosquito species use deer as a primary blood source and actively transmit JC virus to humans. Grimstad et al. (1986) reported only 22% of encephalitis cases were of known etiologies in 1986. If diagnostic laboratories continue to only use the LAC viral antigen, JC viral infections will rarely be diagnosed and reported only as “California encephalitis.”

Penned deer studies replicating natural conditions are needed to examine pathogen exposures, seroconversion rates, and detailed maternal antibody transmission for all organisms. Additional research explaining the likelihood of encephalitis viral transmission directly between deer and vector(s) should be conducted. However, environmental factors such as soil, water, vectors, other wildlife species, and climate have potential to influence interpretation of pathogen data and these studies should attempt to duplicate and/or control for true environmental conditions.

Overlapping site specific human and pet disease cases may result in a more accurate risk evaluation map for the Chicago region. A survey of residents from communities surrounding DP and Palos forest preserves should be conducted to assist in risk assessment. The survey should address human and pet population dynamics such as: number of pets, type of pet(s), feces disposal location, if pets are allowed to roam freely, how often people and pets use the recreational areas, and what activities are pursued. Additionally, research investigating the relationship between accurately estimated wildlife densities and disease prevalence should be explored. Additional studies should include extensive radio-telemetry work on suburban male deer because of the differences in pathogen prevalence between the sexes revealed in this study and a lack of male ecology information in published urban/suburban deer literature.

Information about zoonoses and parasites in white-tailed deer will become increasingly

important as deer populations continue to rise and habitat decreases (Gaydos, 2005). Emerging and reemerging infectious diseases continue to increase worldwide and it is important to monitor for zoonotic pathogens that persist in the environment before outbreaks become endemic.

Understanding the health status of wildlife populations provides an investigative tool and surveillance for wildlife zoonoses. Using sentinel animals can be a cost-effective means to infer prevalence in host or reservoir populations when direct estimation is difficult (VerCauteren et al, 2008). This is an essential component in managing free-ranging wildlife and protecting public health in urban/suburban environments.

TABLE 1. Prevalence for *Toxoplasma gondii* in suburban white-tailed deer (n=443) from Chicago, Illinois, USA, 1995-99.

Variables	Positive deer	Prevalence (%)
Age		
Fawn	36	32.4
Yearling	42	49.4
Adult	168	68.0
Sex		
Female	171	56.3
Male	75	54.0
Study site		
Des Plaines	159	68.0
Palos	87	41.6
Year		
1995-96	48	45.7
1996-97	76	61.8
1997-98	84	62.3
1998-99	38	47.0
Month		
December	36	73.5
January	61	62.2
February	56	49.1
March	93	51.1

Table 2. Area under the curve (AUC) values by pathogen for the final logistic regression models for suburban white-tailed deer from Chicago, Illinois, USA, 1995-99.

Pathogen	Deer variables	AUC value
<u>Toxoplasma gondii</u>	Age	0.66
	Sex	0.51
	Study site	0.37
	Year	0.52
	Month	0.50
<u>Leptospira</u> spp.	Age	0.56
	Study site	0.57
	Year	0.69
Jamestown Canyon virus	Age	0.72
	Study site	0.43
	Year	0.51
LaCrosse virus	Age	0.65
	Month	0.35

TABLE 3. Prevalence for Leptospira spp. in suburban white-tailed deer (n=444) from Chicago, Illinois, USA, 1995-99.

Variables	Positive deer	Prevalence (%)
Age		
Fawn	13	11.7
Yearling	13	15.1
Adult	46	18.6
Sex		
Female	51	15.2
Male	21	16.7
Study site		
Des Plaines	30	12.8
Palos	42	20.0
Year		
1995-96	9	8.5
1996-97	13	10.6
1997-98	16	12.0
1998-99	34	41.5
Month		
December	3	6.1
January	14	14.3
February	16	14.0
March	39	21.3

TABLE 4. Prevalence for Jamestown Canyon virus in suburban white-tailed deer (n=404) from Chicago, Illinois, USA, 1995-99.

Variables	Positive deer	Prevalence (%)
Age		
Fawn	5	4.5
Yearling	31	41.9
Adult	118	53.6
Sex		
Female	114	42.5
Male	40	29.4
Study site		
Des Plaines	93	44.5
Palos	61	31.3
Year		
1995-96	17	20.5
1996-97	58	49.2
1997-98	62	47.3
1998-99	17	23.6
Month		
December	18	45.0
January	32	35.6
February	35	36.1
March	69	39.0

TABLE 5. Prevalence for LaCrosse virus in suburban white-tailed deer (n=404) from Chicago, Illinois, USA, 1995-99.

Variables	Positive deer	Prevalence (%)
Age		
Fawn	3	2.7
Yearling	2	2.7
Adult	22	10.0
Sex		
Female	22	8.2
Male	5	3.7
Study site		
Des Plaines	14	6.7
Palos	13	6.7
Year		
1995-96	7	8.4
1996-97	3	2.5
1997-98	10	7.6
1998-99	7	9.7
Month		
December	1	2.5
January	9	10.0
February	11	11.3
March	6	3.4

LITERATURE CITED

- ACHA, P. N. AND B. SZYFRES. 1994. California encephalitis. *In* Zoonoses and communicable diseases common to man and animals, 2nd Edition, Pan American Health Organization, Washington, D.C., pp. 304-308.
- ADRIAN, W. J. AND R. E. KEISS. 1977. Survey of Colorado's wild ruminants for serologic titers to brucellosis and leptospirosis. *Journal of Wildlife Diseases* 13:429-431.
- ANDREADIS, T. G., J. F. ANDERSON, P. M. ARMSTRONG, AND A. J. MAIN. 2008. Isolations of Jamestown Canyon virus (Bunyaviridae: Orthobunyavirus) from field-collected mosquitoes (Diptera: Culicidae) in Connecticut, USA: A ten-year analysis, 1997-2006. *Vector-Borne and Zoonotic Diseases* 8(2):175-188.
- BENENSON, A. S. 1995. Control of communicable diseases manual. American Public Health Association, Washington, D. C., pp. 267-270.
- BERINGER, J., L. P. HANSEN, AND O. SEXTON. 1998. Detection rates of white-tailed deer with a helicopter over snow. *Wildlife Society Bulletin* 26:24-28.
- BEVERLEY, J. K. A., L. HENRY, D. HUNTER, AND M. E. BROWN. 1977. Experimental toxoplasmosis in calves. *Research Veterinary Science* 23(33):33-37.
- BOROMISA, R. D. AND P. R. GRIMSTAD. 1987. Seroconversion rates to Jamestown Canyon virus among six populations of white-tailed deer (Odocoileus virginianus) in Indiana. *Journal of Wildlife Diseases* 23(1):23-33.
- CLARK, L. G., J. I. KRESSE, A. CARBREY, R. R. MARSHAK, AND C. J. HOLLISTER. 1961. Leptospirosis in cattle and wildlife on a Pennsylvania farm. *Journal of the American Veterinary Medical Association* 139(8):889-891.

- CLARK, G. C., H. L. PRETULA, C. W. LANGKOP, R. J. MARTIN, AND C. H. CALISHER. 1983. Persistence of LaCrosse virus (California encephalitis serogroup) in North-Central Illinois. *American Journal of Tropical Medicine and Hygiene* 32:175-184.
- COLE, J. R., H. C. ELLINGHAUSEN, AND H. L. RUBIN. 1979. Laboratory diagnosis of leptospirosis of domestic animals. *Proceedings of U. S. Animal Health Association* 83:189-195.
- COLE, J. R., C. R. SULZER, AND A. R. PURSEL. 1983. Improved microtechnique for the leptospiral microscopic agglutination test. *Applied Microbiology* 25(6):976-980.
- COSTA, A. J., F. G. ARAUJO, J. O. COSTA, J. D. LIMA, AND E. NASCIMENTO. 1977. Experimental infection of bovines with oocysts of Toxoplasma gondii. *Journal of Parasitology* 63(2):212-218.
- DABRITZ, H. A., E. R. ATWILL, I. A. GARDNER, M. A. MILLER, AND P. A. CONRAD. 2006. Outdoor fecal deposition by free-roaming cats and attitudes of cat owners and nonowners toward stray pets, wildlife, and water pollution. *Journal of the American Veterinary Medical Association* 229:74-81.
- DASZAK, P., A. A. CUNNINGHAM, AND A. D. HYATT. 2001. Anthropogenic environmental change and the emergence of infectious diseases in wildlife. *ACTA Tropica* 78:103-116.
- DAWSON, J. E., J. E. CHILDS, K. L. BIGGIE, C. MOORE, D. STALLKNECHT, J. SHADDOCK, J. BOUSEMAN, E. HOFMEISTER, AND J. G. OLSON. 1994. White-tailed deer as a potential reservoir of Ehrlichia spp. *Journal of Wildlife Diseases* 30(2):162-168.
- DAY, J. F., L. M. STARK, J. ZHANG, A. M. RAMSEY, AND T. W. SCOTT. 1996. Antibodies to arthropod-borne encephalitis viruses in small mammals from southern Florida. *Journal of Wildlife Diseases* 32(3):431-436.

DUBEY, J. P. 2010. Toxoplasmosis of animals and humans. 2nd Edition, CRC Press, Boca Raton, Florida.

DUBEY, J. P. AND G. DESMONTS. 1987. Serological responses of equids fed Toxoplasma gondii oocysts. Equine Veterinary Journal 19:337-339.

_____, E. T. THORNE, AND S. P. SHARMA. 1980. Experimental toxoplasmosis in elk (Cervus canadensis). American Journal of Veterinary Research 41(5):792-793.

_____, R. M. WEIGEL, A. M. SIEGEL, P. THULLIEZ, U. D. KITRON, M. A. MITCHEL, A. MANNELLI, N. E. MATEUS-PINILLA, S. K. SHEN, O. C. H. KWOK., AND K. S. TODD. 1995. Sources and reservoirs of Toxoplasma gondii infection on 47 swine farms in Illinois. Journal of Parasitology 81(5):723-729.

ETTER, D. R., K. M. HOLLIS, T. R. VAN DEELEN, D. R. LUDWIG, J. E. CHELSVIG, C. L. ANCHOR, AND R. E. WARNER. 2002. Survival and movements of white-tailed deer in suburban Chicago, Illinois. Journal of Wildlife Management 66(2):500-510.

FAINE, S. 1994. Leptospira and leptospirosis. CRC Press, Boca Raton, Florida, 353 pp.

FERREIRA, R. A., J. R. MINEO, J. M. DUARTE, D. A. O. SILVA, AND J. H. PATARROYO. 1997. Toxoplasmosis in naturally infected deer from Brazil. Journal of Wildlife Diseases 33(4):896-899.

FERRIS, D. H., L. E. HANSON, H. E. RHOADES, AND J. O. ALBERTS. 1961.

Bacteriological and serological investigations of brucellosis and leptospirosis in Illinois deer. Journal of the American Veterinary Medical Association 139:892-896.

FLEISS, J. 1981. Statistical methods for rates and proportions. 2nd Edition, John Wiley and Sons, New York, New York.

- FOURNIER, J. S., J. C. GORDON, AND C. R. DORN. 1986. Comparison of antibodies to Leptospira in white-tailed deer (Odocoileus virginianus) and cattle in Ohio. *Journal of Wildlife Diseases* 22:335-359.
- FRANTI, C. E., G. E. CONNOLLY, H. P. RIEMANN, D. E. BEHYMER, R. RUPPANNER, C. M. WILLADSEN, AND W. LONGHURST. 1975. A survey of Toxoplasma gondii antibodies in deer and other wildlife on a sheep range. *Journal of the American Veterinary Medical Association* 167:565-568.
- FRENKEL, J. F., A. RUIZ, AND M. CHINCHILLA. 1975. Soil survival of Toxoplasma oocysts in Kansas and Costa Rica. *American Journal of Tropical Medicine and Hygiene* 24(3):439-443.
- GAYDOS, J. K. 2005. Diseases of white-tailed deer. *In* *Wildlife diseases: Landscape epidemiology, spatial distribution, and utilization of remote sensing technology*, S. K. Majumdar, J. E. Huffman, F. J. Brenner, and A. I. Panah (eds). The Pennsylvania Academy of Science, Easton, Pennsylvania, pp. 259-271.
- GEHRT, S. D. 2005. Urban raccoon study provides clues to improve rabies prevention. Ohio State University Extension.
- GEHRT, S. D., M. J. KINSEL, AND C. ANCHOR. 2010. Pathogen dynamics and morbidity of striped skunks in the absence of rabies. *Journal of Wildlife Diseases* 46(2):335-347.
- GOYAL, S. M., L. D. MECH, AND M. E. NELSON. 1992. Prevalence of antibody titers to Leptospira spp. in Minnesota white-tailed deer. *Journal of Wildlife Diseases* 28(3):445-448.
- GRIMSTAD, P. R. 1989. California group virus disease. *In* *The arboviruses: Epidemiology and ecology*, T. P. Monath (ed). CRC Press, Boca Raton, Florida, pp. 99-136.

- _____. C. L. BARRETT, R. L. HUMPHREY, AND M. J. SINSKO. 1984. Serological evidence for wide-spread infection with LaCrosse and St. Louis encephalitis viruses in the Indiana human population. *American Journal of Epidemiology* 119:913-930.
- _____, C. H. CALISHER, R. N. HARROFF, AND B. B. WENTWORTH. 1986. Jamestown Canyon virus (California Serogroup) is the etiological agent of widespread infection in Michigan humans. *American Society of Tropical Medicine and Hygiene* 35(2):376-386.
- _____, P. R., D. G. WILLIAMS, AND S. M. SCHMITT. 1987. Infection of white-tailed deer (*Odocoileus virginianus*) in Michigan with Jamestown Canyon virus (California Serogroup) and the importance of maternal antibody viral maintenance. *Journal of Wildlife Diseases* 23(1):12-22.
- HANLEY, J. A. AND B. J. MCNEIL. 1982. The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology* 143:29-36.
- HEATH, S. E. AND R. JOHNSON. 1994. Leptospirosis. *Journal of the American Veterinary Medical Association* 205(11):1518-1523.
- HOFF, G. L., C. J. ISSEL, D. O. TRAINER, AND S. H. RICHARDS. 1973. Arbovirus serology in North Dakota mule and white-tailed deer. *Journal of Wildlife Diseases* 9(4):291-295.
- HOLLIS, K. M. 2011. Epidemiology of suburban deer. Ph.D. Thesis. University of Illinois, Urbana-Champaign.
- HOSMER, D. W. AND S. LEMESHOW. 1989. Applied logistic regression. John Wiley and Sons, New York, New York.

HUMPHREYS, J. G., R. L. STEWART, AND J. P. DUBEY. 1995. Prevalence of Toxoplasma gondii antibodies in sera of hunter-killed white-tailed deer in Pennsylvania. *American Journal of Veterinary Research* 56(2):172-173.

ISSEL, C. J. 1974. Maternal antibody to Jamestown Canyon virus in white-tailed deer. *The American Journal of Tropical Medicine and Hygiene* 23(2):242-245.

_____, D. O. TRAINER, AND W. H. THOMPSON. 1972a. Experimental studies with white-tailed deer and four California group arboviruses (LaCrosse, Trivittatus, Snowshoe Hare, and Jamestown Canyon). *American Journal of Tropical Medicine and Hygiene* 21(6):979-984.

_____, _____, AND _____. 1972b. Serologic evidence of infections of white-tailed deer in Wisconsin with three California group arboviruses (La Crosse, Trivittatus, and Jamestown Canyon). *American Journal of Tropical Medicine and Hygiene* 21(6):985-988.

JESSUP, D. A. 2004. The welfare of feral cats and wildlife. *Journal of the American Veterinary Medical Association* 225:1377-1383.

KITRON, U., J. MICHAEL, J. SWANSON, AND L. HARAMIS. 1997. Spatial analysis of the distribution of LaCrosse encephalitis in Illinois, using a geographic information system and local and global spatial statistics. *American Journal of Tropical Medicine and Hygiene* 57(4):469-475.

KLEINBAUM, D. G. 1994. *Logistic regression: A self-learning text*. Springer-Verlag, New York, New York, 282 pp.

LEIGHTON, F. A. AND T. KUIKEN. 2001. Leptospirosis *In* *Infectious diseases of wild mammals*, E. S. Williams and I. K. Barker (eds). Iowa State University Press, Ames, Iowa, pp. 498-502.

LUDWIG, J. 1981. Proportion of deer seen in aerial counts. *Minnesota Wildlife Resources Quarterly* 41:11-19.

- MATSCHKE, G. H., K. A. FAGERSTONE, R. F. HARLOW, F. A. HAYES, V. F. NETTLES, W. PARKER, AND D. O. TRAINER. 1984. Population influences. *In* White-tailed deer ecology and management, L. K. Halls (ed). Stackpole, Harrisburg, Pennsylvania, pp. 169-188.
- MILLER, D. A., M. A. WILSON, AND G. W. BERAN. 1991. Survey to estimate prevalence of Leptospira interrogans infection in mature cattle in the United States. *American Journal of Veterinary Research* 52(1111):1761-1765.
- MITCHELL, M. A., L. L. HUNGERFORD, C. NIXON, T. ESKER, J. SULLIVAN, R. KOERKENMEIER, AND J. P. DUBEY. 1999. Serological survey for selected infectious disease agents in raccoons from Illinois. *Journal of Wildlife Diseases* 35(2):347-355.
- MURPHY, R. K. 1989. Serologic evidence of arboviral infections in white-tailed deer from Central Wisconsin. *Journal of Wildlife Diseases* 25(2):300-301.
- NASH, M. L., L. L. HUNGERFORD, T. G. NASH, AND G. M. ZINN. 1995. Epidemiology and economics of clinical listeriosis in a sheep flock. *Preventive Veterinary Medicine* 24:147-156.
- NEITZEL, D. F. AND P. R. GRIMSTAD. 1991. Serological evidence of California group and cache valley virus infection in Minnesota white-tailed deer. *Journal of Wildlife Diseases* 27(2):230-237.
- NEW, J. C., W. G. WATHEN, AND S. DLUTKOWSKI. 1993. Prevalence of Leptospira antibodies in white-tailed deer, Cades Cove, Great Smoky Mountains National Park, Tennessee, USA. *Journal of Wildlife Diseases* 29(4):561-567.
- NIXON, C. M., L. P. HANSEN, P. A. BREWER, AND J. E. CHELSVIG. 1991. Ecology of white-tailed deer in an intensively farmed region of Illinois. *Wildlife Monographs* 118, 77 pp.

OSORIO, J. E., M. S. GODSEY, G. R., DEFOLIART, AND T. M. YUILL. 1996. LaCrosse viremias in white-tailed deer and chipmunks exposed by injection or mosquito bite. *American Journal of Tropical Medicine and Hygiene* 54(4):338-342.

REILLY, J. R., T. F. MURASCHI, AND D. J. DEAN. 1962. Experimental Leptospira pomona infection in white-tailed deer, Odocoileus virginianus, and in cattle. *Journal of the American Veterinary Medical Association* 140(1):53-57.

ROTHMAN, K. J. 1986. *Modern epidemiology*. Little, Brown and Company, Boston, Massachusetts.

SCHOWALTER, D. B., J. O. IVERSEN, L. C. CORNER, AND J. R. GUNSON. 1980. Prevalence of antibodies from Toxoplasma gondii in striped skunks from Saskatchewan and Alberta. *Journal of Wildlife Diseases* 16(2):189-194.

SHOTTS, E. B., C. L. ANDREWS, AND T. W. HARVEY. 1975. Leptospirosis in selected wild mammals of the Florida panhandle and southwestern Georgia. *Journal of the American Veterinary Medical Association* 167:587-589.

_____ AND F. A. HAYES. 1970. Leptospiral antibodies in white-tailed deer of the southeastern United States. *Journal of Wildlife Diseases* 6:295-298.

SMITH, D. D. AND J. K. FRENKEL. 1995. Prevalence of antibodies to Toxoplasma gondii in wild mammals of Missouri and east central Kansas: biologic and ecologic considerations of transmission. *Journal of Wildlife Diseases* 31(1):15-21.

TATARUCH, F. AND H. KIERDORF. 2003. Mammals as biomonitors. *In* *Bioindicators and biomonitors: Principles, concepts, and applications*, B. Markert, A. Breure, and H. Zechmeister (eds). Elsevier Science Limited, Kidlington, Oxford.

- THOMAS, F. C. AND D. O. TRAINER. 1970. Bluetongue virus in white-tailed deer. *American Journal of Veterinary Research* 31(2):271-278.
- TIZARD, I. R., S. S. CHAUHAN, AND CH. H. LAI. 1977. The prevalence and epidemiology of toxoplasmosis in Ontario. *Journal of Hygiene* 78:275-282.
- TRAINER D. O. AND R. P. HANSEN. 1960. Leptospirosis and brucellosis serological reactors in Wisconsin deer, 1957-58. *Journal of Wildlife Management* 24(1):44-52.
- _____ AND _____. 1969. Serologic evidence of arbovirus infections in wild ruminants. *American Journal of Epidemiology* 90(4):354-358.
- _____, L. KARSTAD, AND R. P. HANSEN. 1961. Experimental leptospirosis in white-tailed deer. *The Journal of Infectious Diseases* 108:278-286.
- VANEK, J. A., J. P. DUBEY, P. THULLIEZ, M. R. RIGGS, AND B. E. STROMBERG. 1996. Prevalence of Toxoplasma gondii antibodies in hunter-killed white-tailed deer (Odocoileus virginianus) in four regions of Minnesota. *Journal of Parasitology* 82(1):41-44.
- VERCAUTEREN, K. C., T. C. ATWOOD, T. J. DELIBERTO, H. J. SMITH, J. S. STEVENSON, B. V. THOMSEN, T. GIDLEWSKI, AND J. PAYEUR. 2008. Sentinel-based surveillance of coyotes to detect bovine tuberculosis, Michigan. *Emerging Infectious Diseases* 14(12):1862-1869.
- VIÑA, A., M-N. TUANMU, W. XU, Y. LI, Z. OUYANG, R. DEFRIES, AND J. LIU. 2010. Range-wide analysis of wildlife habitat: Implications for conservation. *Biological Conservation* 143:1960-1969.
- WARD, M. P., L. T. GLICKMAN, AND L. F. GUPTILL. 2002. Prevalence of and risk factors for leptospirosis among dogs in the United States and Canada: 677 cases (1970-1998). *Journal of the American Veterinary Medical Association* 220(1):53-58.

WEDMAN, E. E. AND F. C. DRIVER. 1957. Leptospirosis and brucellosis titers in deer blood. *Journal of the American Veterinary Medical Association* 130:513-514.

WISE, J. K., B. L. HEATHCOTT, AND M. L. GONZALEZ. 2002. Results of the AVMA survey on companion animal ownership in the US pet-owning households. *Journal of the American Veterinary Medical Association* 221(11):1572-1573.

WITHAM, J. H. AND J. M. JONES. 1987. Chicago Urban Deer Study. Illinois Natural History Survey Report No. 265, Champaign, Illinois.

ZAMPARO, J. M., T. G. ANDREADIS, R. E. SHOPE, AND S. J. TIRRELL. 1997. Serologic evidence of Jamestown Canyon virus infection in white-tailed deer populations from Connecticut. *Journal of Wildlife Diseases*, 33(3):623-627.

CHAPTER 2

HABITAT MODELING OF ENCEPHALITIDES USING SUBURBAN DEER

ABSTRACT: Wildlife disease ecology has gained much attention because of the emergence and re-emergence of pathogens causing human disease. The study of zoonoses in wildlife populations requires an understanding of relationships between organisms and their environment. I modeled habitat use for 169 radio-collared and culled suburban white-tailed deer (Odocoileus virginianus) to understand viral seroprevalence for Jamestown Canyon (JC) and LaCrosse (LAC) encephalitis in two suburban Chicago forest preserves (DesPlaines and Palos) from 1995-99. Radio-collared does selected for forest, savanna, and grassland habitats (total available habitat= $p < 0.0001$; home range= $p < 0.001$). Akaike's Information Criterion (AICc) optimal models for JC contained water and wetland habitat parameters while LAC models contained forested habitat. Deer biological parameters in JC viral models included age and month/year samples were collected. Total home-range or buffer size and month were biological parameters contained in LAC viral models. Deer are sufficient biomonitors for zoonotic pathogens and a better understanding of the relationship between deer and habitat use will assist in focusing future research and surveillance for JC and LAC viruses.

INTRODUCTION

Alterations in land use and habitat availability can alter contact between humans and pathogens. Urbanization creates a shift in disease host(s) geographic ranges and densities, interspecific interactions, and pathogen contamination (Bradley and Altizer, 2006). During the past quarter-century the emergence of new diseases and the re-emergence of previous "conquered" diseases have become an international focus for concern and action (McLean, 2001). It is becoming increasingly important to understand the relationship among wildlife,

zoonoses, and pathogen ecology.

Jamestown Canyon (JC) and LaCrosse (LAC) viruses belong to the California encephalitis viral group and are widely distributed throughout North America (Grimstad et al., 1986; Acha and Szyfres, 1994). Jamestown Canyon virus is transmitted vertically to Aedes spp. mosquito offspring and horizontally to vertebrates, especially deer (Odocoileus virginianus; Acha and Szyfres, 1994). Jamestown Canyon vectors breed in ground pools of semi-permanent to permanent waters (Horsfall, 1972). After a 1984 (Mokry et al.) retrospective study in New York, 62% of patients with central nervous system (CNS) infections had been infected with JC. Grimstad et al. (1984) found widespread JC endemic human infection in Michigan.

LaCrosse virus is transmitted by the primary mosquito vector, A. triseriatus. LAC mosquitoes breed in tree-holes or man-made containers confining water (Kitron et al., 1997). The virus is transmitted by the vector transovarially and to various squirrel species (Tamias striatus, Sciurus carolinensis, and S. niger) in oak forests (Acha and Szyfres, 1994). Additional mammal species serve to maintain the virus through vector mosquitoes in the summer months. LaCrosse antibodies were reported in 16% of southwestern Wisconsin deer (Issel et al., 1974). LaCrosse virus has been documented as an important California serogroup producing CNS illness in humans, however there is a lack of serological detection and differentiation among the California viruses in many clinical cases (Kappus et al., 1983; Grimstad et al., 1984).

Deer are useful indicators for the presence of arboviral pathogens because of their large population, ubiquitous distribution, non-migratory behavior, and opportunity for large sera samples (Trainer, 1973). In addition, deer have distinct habitat use patterns based upon landscape characteristics associated with food and cover requirements (Nixon et al., 1994). Many infectious agents have necessary optimal habitat types, especially arboviral zoonoses like

JC and LAC viruses, requiring mosquito vectors (Anderson and Gutzwiller, 1996; Wilson, 2000). Transient water sources for mosquitoes include temporary water pools created by flooding, melting snow, or near homes in man-made debris such as tires and buckets. Tree-holes found in common maple, oak, or mixed hardwood forests generally hold water useful for mosquito breeding. Jamestown Canyon virus was isolated from Aedes mosquitoes collected from transient water pools in Canada (Iverson et al., 1969). During a LAC encephalitis study in Illinois, 14 out of 50 tree-hole pools were positive for infected mosquito larvae (Clark et al., 1983). Dynamic interactions among wildlife species, vector(s), disease agent, and the environment allow modeling of complex ecological and environmental data.

I modeled habitat use in white-tailed deer to understand viral seroprevalence (JC and LAC) in two suburban Chicago forest preserves (Des Plaines and Palos) from 1995-99. The goal was to determine if specific habitat characteristics within deer home ranges and culled deer buffers was useful in determining pathogen presence. This research will provide insight about the relationship between habitat used by deer and required by mosquito vectors as it relates to pathogens.

MATERIALS AND METHODS

General materials and methods are specified in pages 14-21.

ANOVA was used to compare deer home range size (95% and 50%) by JC and LAC viruses ($\alpha \leq 0.05$; JMP[®] 4.02, SAS Institute Inc., Cary, North Carolina, USA; SAS Institute, 2000; Sokal and Rohlf, 1995). The software ArcView[®] 3.2 (Environmental Systems Research Institute Inc., Redlands, California, USA; ESRI, 1996) was used to create a geographic information system (GIS) from 1999 thematic mapper land cover data generated by the Illinois Natural History Survey (INHS). Every 5th culled deer from Des Plaines (DP) and Palos were

systematically selected to reduce potential overlap among family groups of deer removed from the same site. Data points were imported into a GIS for culled deer and 95% minimum convex polygon (MCP) home ranges for radio-collared deer from Des Plaines (DP) and Palos. It was assumed individual locations of culled deer were the center of each deer's home range. Culled deer locations were buffered with a 95% MCP (DP = 83 ha, Palos = 45 ha) and the buffered area was representative of the average live female deer home ranges for each respective site. Male deer were not radio-tracked so buffered areas for culled male deer were underestimated because male home ranges average larger than females (Nixon et al., 1991; 1994).

Patterns of habitat selection were evaluated by compositional analysis for radio-collared does from DP and Palos. Compositional analysis treats each animal as a sampling unit, thereby avoiding autocorrelation and pseudoreplication problems associated with use of telemetry observations as sampling units (Hurlbert, 1984; Aebischer et al., 1993; Otis and White, 1999). Resource Selection software (RSW[®] 1.0, University of Idaho, Pocatello, Idaho, USA; Leban, 1998) was used to test for departure from habitat use among radio-collared deer by site considering 1) home range and 2) total available habitat (Hurlbet, 1984; Aebisher et al., 1993; Sokal and Rohlf, 1995; Otis and White, 1999). Analysis of habitat use allows for large scale analysis (home range selection). Log ratios were derived in RSW to identify selection indexes and detect differences in habitat use and availability within sites (Aebisher et al., 1993).

Land cover variables from satellite images were reduced from 23 land cover categories to six based on habitat use patterns for Midwest deer, while also considering important habitats for vector mosquitoes (Table 6). From previous deer studies and the compositional analysis, deer favored agriculture, forest, savanna, wetland, grasslands, and urban sites as homes supply lawns

and enhanced landscapes as a food resource (Murphy et al., 1985; Nixon et al., 1991; Swihart et al., 1995).

Fragstats[®] 2.0 (Fragstats, Oregon State University, Corvallis, Oregon, USA; McGarigal and Marks, 1993) in ArcInfo[®] 8.0 software (Environmental Systems Research Institute Inc., Redlands, California, USA; ESRI, 1995) was used to estimate habitat metrics. Habitat parameters were calculated at the class level for deer (Appendix M). Variable selection continued based upon deer habitat and JC/LAC viral studies while considering available Fragstats metrics (Halls, 1984; Grimstad, 1988; Nixon et al., 1991; 1994; McShea et al., 1997; Yuill and Seymour, 2001). Many Fragstats variables provided duplicate or redundant information and were deleted. Highly correlated Fragstat habitat class variables (Table 7) were reduced using multivariate pair wise comparisons by JC or LAC viral prevalence ($\% \leq 0.25$; JMP[®] 4.02, SAS Institute Inc., Cary, North Carolina, USA; SAS, 2000; Sokal and Rohlf, 1995). Fragstat variables were reduced further by viral prevalence using t-tests ($\% \leq 0.10$).

A priori I posited logistic regression models based upon deer ecology, mosquito habitat, and JC/LAC viruses (Table 8). I selected models best supported by the data using Akaike's Information Criterion for small sample sizes (AICc). Models were selected based upon empirical support by evaluating the delta AIC (ΔAIC) values ~ 2.0 (Burnham and Anderson, 2002).

RESULTS

Since there was no difference in viral prevalence between 50% or 95% MCP (JC $p=0.65$; LAC $p=0.60$), 95% MCP was selected for home range analyses. Female deer maintained sedentary home ranges during the study (Etter et al., 2002). The original Fragstats analysis

included 32 radio-collared and 137 culled deer (total n=169). Radio-collared does positively selected for forested, savanna, and grassland habitats while negatively selecting for urban, agriculture, wetland, and open water land cover classifications (available habitat ($p < 0.0001$) and home range ($p < 0.001$); Fig. 2).

Four models had support for JC virus based on ΔAIC values ranging from 0-2.13 (Table 9). All four models contained month the deer sample was collected (month) and deer age. Year the deer sample was collected (year) was also present in three of the four top models. Models two and three contained habitat variables water IJI and wet %Land.

Three models contained supportive ΔAIC values for LAC virus ranging from 0-2.76 (Table 9). Habitat variables ft NNCV, ft PSCV, and ft AWMSI appeared in the top two models. Deer parameters month and total home range or buffer size (total) also appeared in the top model.

DISCUSSION

White-tailed deer have distinct patterns for habitat requirements (Nixon et al., 1991; Grund et al., 2002) and many infectious agents require specific habitat types (Anderson and Gutzwiller, 1996; Wilson, 2000). Deer use various habitat types for cover and as a transitional shift when food supplies are limited (Nixon et al., 1991; Grund et al., 2002). A behavioral response by deer to humans may also play a role in habitat use. Deer in highly fragmented agricultural landscapes search out maximum cover from intruding human activities (Storm et al., 2006; Nixon et al., 2007). However, deer studied in suburban environments did not appear to avoid human activities and were even observed being directly fed by humans on several occasions (C. Anchor, D. Etter, pers. comm.). It is probable that the more humans and wildlife interact, the more likely for zoonoses transmission.

JC viral models 2 and 3 (Table 9) included habitat variables related to standing water, a prerequisite for mosquitoes. Deer from both sites had a negative selection for water and a strong negative (DP) to slightly positive (Palos) selection for wetlands. Mosquitoes and biting insects averaged 15 times more abundant in bottom-lands compared to upland habitats during a study of rural deer in Illinois (Nixon et al., 1991). This suggests deer with lowland habitats or wet areas in their home ranges would be at greater risk of being bitten by infected mosquitoes. The distribution, proximity, and patch relationship of the water and wetland resources increases the likelihood that deer will be exposed to JC virus carrying mosquitoes in these specific habitats.

The top two models for LAC virus (LAC viral models 1 and 2; Table 9) contained forested parameters and metrics supporting the likely exposure of deer to tree-hole mosquitoes, the primary vector. A. triseriatus breed inside tree-holes commonly found in deciduous oak forests and other places where water collects, including tires near homes (Acha and Szyfres, 2001). The relationship among forested habitat, size of the habitat patches, and the percent of landscape covered by savanna-grass (Table 9, LAC model 3) supports deer using a greater number of habitat types which may increase the risk of exposure to vectors and pathogenic organisms. Increased rainfall can have a positive effect on vectors by providing adequate microenvironments for breeding mosquitoes, even in urban areas (Pherez, 2007).

GIS data layers, while they are easily used and offer powerful data manipulation and measurement capabilities, have limitations when evaluating large regions. Large-scale GIS databases may not contain the data layers required for examining the specific ecological question(s) of interest (Pendleton et al., 1998). For example, the GIS databases used did not contain specific mosquito breeding habitat. Land cover classification, errors in classification images, and the reduction of land cover categories into six habitat types may have also

influenced results. Land cover images from data collected in 1999 were used because they overlapped with the study time frame. However, the accuracy of the images (forests=75.6% accurate; INHS, Land Cover Classification Accuracy, 1999-2000) and the lack of fine scale details could affect results. For example, water was an important component in JC viral models, but deer negatively selected for open water habitats. In addition, land cover data is influenced by when it is collected and landscapes change annually due to development, vegetative growth, and seasonal weather patterns. I noted error in the classification of agriculture and grasslands in specific areas of both preserves. However, I suspect that because deer and pathogen vectors like mosquitoes occur at different habitat scales, it may be difficult to elucidate fine-scale pathogen-vector-host relationships without detailed ecological data from both deer and mosquitoes.

Top models for JC and LAC (Table 9) viruses also included repeated parameters related to basic deer biology (month, age, and year). As deer get older, they increase the risk of pathogen exposure over time and maintain antibodies (Grimstad, 1988; Grimstad et al., 1987). Many infectious diseases have seasonal and annual cycles (Wilson, 2000), explaining the importance of month and year in our models. Most human infections occur during occupational or recreational activities from July to September (Acha and Szyfres, 2001). Therefore, deer sampled in the winter will provide indicators for annual exposure(s) to encephalitis. Age of deer sampled for JC and LAC viruses should also be considered because maternal antibodies and seroconversion rates may influence serological results. Yearlings are useful in determining new exposures and adults for long-term information. Prevalence analysis including specific deer parameters and conclusions was detailed in Hollis (2011).

White-tailed deer were a useful sentinel for determining the absence or presence of JC and LAC viruses in a fragmented suburban environment. Deer are a generalist species and use

various ecotones within forested and savanna cover (Nixon, et al., 1991). Deer have larger home ranges compared to many sedentary mammals in the Chicago region, use multiple habitat types, and therefore increase exposures to various pathogens. However, a specialist species may serve as a better sentinel to intensely survey specific focal areas, especially for LAC. A fine-scale analysis involving both generalist and specialist species could serve to identify habitat differences and create more detailed risk profiles at different scales for host, vectors, and disease. I only had data available for two sites in Cook County where deer were radio-collared and culled. Increasing sample sizes, number of study sites, and focusing on seasonal home ranges overlapping with vector emergence and abundance may also alter the results because habitat availability and use by deer varied between the two study sites.

This research revealed that deer serve as a sufficient biomonitor for locating pathogens and create a potential for human exposure to disease (Hollis, 2011). The habitat, zoonotic pathogens, and vectors are present in Cook County Forest Preserves. This research further allows scientists to finely focus future projects. Occasional sampling for pathogen antibodies among deer populations may be beneficial for documenting viral changes, habitat differences, landscape alterations, and human encroachment into wildlife habitat (Arjo et al., 2003). Proactive surveillance can identify gaps in zoonotic information by locating areas where human outbreaks have not occurred but where pathogens are present. Investigative strategies analyzing the relationship between mammals, habitat use, zoonotic organisms, and vectors will serve to advance our understanding of ecological and pathogen relationships.

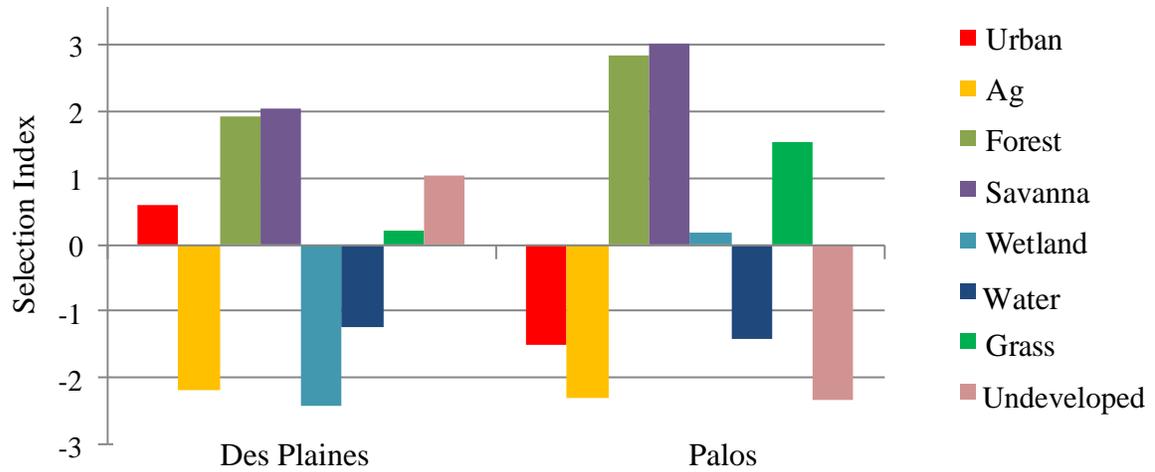


FIGURE 2. Suburban white-tailed deer habitat selection index from compositional analysis by study site Chicago,, Illinois, USA, 1995-99 ($\alpha < 0.05$).

TABLE 6. Reduction of 23 original land cover types to 6 habitat categories from Landsat 1999 TM images for suburban white-tailed deer from Chicago, Illinois, USA, 1995-99.

1999 Land cover types	Habitat category
High density urban Medium density urban Low density urban Undifferentiated urban mix	Urban
Row crop-rural grass Agriculture-mixed uses Agriculture-corn Agriculture-small grains Agriculture-soybeans Agriculture-winter wheat Rural grasses	Agriculture
Urban forest Urban grasses Dry savanna	Savanna-Grass
Mesic forest Savanna/mesic-dry mesic forest Mesic/dry-mesic forest Dry-mesic forest	Forest
Shallow marsh emergent wetland Deep marsh emergent wetland Non-swamp forested wetland Mixed wetlands	Wetland
Open water	Water

TABLE 7. Final Fragstat variables used to model suburban white-tailed deer habitat by Jamestown Canyon (JC) and LaCrosse (LAC) viruses from Chicago, Illinois, USA, 1995-99.

Habitat type	Description	Acronym
JC		
Agriculture	Largest patch index	Ag LPI
Agriculture	Percent of total landscape	Ag %Land
Wetland	Percent of total landscape	Wet %Land
Open water	Interspersion-juxtaposition index	Water IJI
LAC		
Savanna-Grass	Percent of total landscape	SG % Land
Savanna-Grass	Largest patch index	SG LPI
Savanna-Grass	Mean nearest neighborhood distance	SG MNN
Forest	Nearest neighborhood coefficient of variation	Ft NNCV
Forest	Patch size coefficient of variation	Ft PSCV
Forest	Area weighted mean shape index	Ft AWMSI

Table 8. Data considered when creating logistic regression models for Akaike's Information Criterion (AICc) to infer Jamestown Canyon (JC) and LaCrosse (LAC) viruses and habitat relationships for suburban white-tailed deer, Chicago, Illinois, USA, 1995-99.

Parameters	Interpretation	References
Deer ecology	Deer habitat, home-range	Nixon et al., 1991, Grund et al., 2002, Horsefall, 1972
Mosquitoes	Habitat use	Grimstad et al., 1987
JC/LAC viruses	Disease exposure	Grimstad et al., 1987, 1988

TABLE 9. Logistic regression models used for Akaike's Information Criterion (AICc) to evaluate Jamestown Canyon (JC) and LaCrosse (LAC) viruses and habitat relationships for suburban white-tailed deer, Chicago, Illinois, USA, 1995-99

Rank	Model ^{a, b}	K	R ²	AICc	ΔAIC	w _i ^c
JC viral models						
1	Month, age, year	5	0.197	202.65	0.00	0.375
2	Month, age, year, water IJI	6	0.203	203.42	0.77	0.255
3	Month, age, total, wet %Land, water IJI	7	0.208	204.66	2.01	0.137
4	Month, age, year, habitats	6	0.197	204.78	2.13	0.129
LAC viral models						
1	Month, total, ft NNCV	5	0.050	83.01	0.00	0.505
2	Ft NNCV, ft PSCV, ft AWMSI	5	0.036	85.59	2.58	0.139
3	Month, sg LPI	4	0.023	85.76	2.76	0.127

^a See Table 7 for description of Fragstats model variables.

^b Month=month sample collected, age=fawn (<6 months old), yearling (1.5 years old), or adult (>2.5 years old), year=year sample collected, total=home range or buffer size, and habitats= number of habitat types per home range or buffer size.

^c w_i=Akaike weight; interpreted as the probability a model is the best model supported by the data, given a set of alternative models (Burnham and Anderson, 2002).

LITERATURE CITED

- ACHA, P. N. AND B. SZYFRES. 1994. California encephalitis. *In* Zoonoses and communicable diseases common to man and animals, 2nd Edition, Pan American Health Organization, Washington, D.C., pp. 304-308.
- ____ AND _____. 2001. California encephalitis. *In* Zoonoses and communicable diseases common to man and animals: Volume II Chlamydioses, Rickettsioses, and Viruses, 3rd Edition, Pan American Health Organization, Washington, D.C., pp. 68-72.
- AEBISCHER, N. J., P. A. ROBERTSON, AND R. E. KENWOOD. 1993. Compositional analysis of habitat use from animal radio-tracking data. *Ecology* 74:1313-1325.
- ANDERSON, S. H. AND K. J. GUTZWILLER. 1996. Habitat evaluation methods. *In* Research and management techniques for wildlife and habitats, T. A. Bookhout (ed). The Wildlife Society, Bethesda, Maryland.
- ARJO, W. M., E. M. GESE, C. BROMLEY, A. KOZLOWSKI, AND E. S. WILLIAMS. 2003. Serological survey for diseases in free-ranging coyotes (*Canis latrans*) from two ecological distinct areas of Utah. *Journal of Wildlife Diseases* 39(2):449-455.
- BRADLEY, C. A. AND S. ALTIZER. 2006. Urbanization and the ecology of wildlife disease. *Trends in Ecology and Evolution* 22(2):95-122.
- BURNHAM, K. P. AND D. R. ANDERSON. 2002. Model selection and inference: A practical information-theoretic approach. Springer-Verlag, New York, New York.
- CLARK, G. C., H. L. PRETULA, C. W. LANGKOP, R. J. MARTIN, AND C. H. CALISHER. 1983. Persistence of LaCrosse virus (California encephalitis serogroup) in North-Central Illinois. *American Journal of Tropical Medicine and Hygiene* 32:175-184.

- ESRI (ENVIRONMENTAL SYSTEMS RESEARCH INSTITUTE). 1995. Understanding GIS: the ARC/INFO method: self study workbook. GeoInformation International, Cambridge, United Kingdom. _____. 1996. ArcView[®] GIS: The geographic information system for everyone. Redlands, California.
- ETTER, D. R., K. M. HOLLIS, T. R. VAN DEELEN, D. R. LUDWIG, J. E. CHELSVIG, C. L. ANCHOR, AND R. E. WARNER. 2002. Survival and movements of white-tailed deer in suburban Chicago, Illinois. *Journal of Wildlife Management* 66(2):500-510.
- GRIMSTAD, P. R. 1988. California group virus disease. *In* The arboviruses: epidemiology and ecology, T.P. Monath (ed.). CRC Press, Boca Raton, Florida, pp. 99-136.
- _____. C. L. BARRETT, R. L. HUMPHREY, AND M. J. SINSKO. 1984. Serological evidence for wide-spread infection with LaCrosse and St. Louis encephalitis viruses in the Indiana human population. *American Journal of Epidemiology* 119:913-930.
- _____, S. M. SCHMITT, AND D. G. WILLIAMS. 1986. Prevalence of neutralizing antibody to Jamestown Canyon virus (California group) in populations of elk and moose in northern Michigan and Ontario, Canada. *Journal of Wildlife Diseases* 22(4):453-458.
- _____, D. G. WILLIAMS, AND S. M. SCHMITT. 1987. Infection of white-tailed deer (*Odocoileus virginianus*) in Michigan with Jamestown Canyon virus (California serogroup) and the importance of maternal antibody viral maintenance. *Journal of Wildlife Diseases* 23:12-22.
- GRUND, M. D., J. B. MCANINCH, AND E. P. WIGGERS. 2002. Seasonal movements and habitat use of female white-tailed deer associated with an urban park. *Journal of Wildlife Management* 66(1):123-130.
- HALLS, L. K. (ed). 1984. White-tailed deer: Ecology and management. The Wildlife Management Institute, Washington D. C.

- HOLLIS, K. M. 2011. Epidemiology of suburban deer. Ph.D. Thesis. University of Illinois, Urbana-Champaign.
- HORSFALL, W. R. 1972. Mosquitoes: Their bionomics and relation to disease. Hafner Press, New York.
- HURLBERT, S. H. 1984. Pseudoreplication and the design of ecological field experiments. *Ecological Monographs* 54:187-211.
- ISSEL, C. J. 1974. Maternal antibody to Jamestown Canyon virus in white-tailed deer. *American Journal of Tropical Medicine and Hygiene* 23(2):242-245.
- IVERSON, J., R. P. HANSON, O. PAPADOPULOS, C. V. MORRIS, AND G. R. DEFOLIART. 1969. Isolation of viruses of the California encephalitis virus group from boreal *Aedes* mosquitoes. *American Journal of Tropical Medicine and Hygiene* 18:735-742.
- KAPPUS, K. D., T. P. MONATH, R. M. KAMINSKI, AND C. H. CALISHER. 1983. Reported encephalitis associated with California serogroup virus infections in the United States, 1963 to 1981. *In California serogroup viruses*, C. H. Calisher and W. H. Thompson (eds). Alan R. Liss, New York.
- KITRON, U., J. MICHAEL, J. SWANSON, AND L. HARAMIS. 1997. Spatial analysis of the distribution of LaCrosse encephalitis in Illinois, using a geographic information system and local and global spatial statistics. *Journal of Tropical Medicine and Hygiene* 57(4):469-475.
- LEBAN, F. 1998. Resource selection for windows. University of Idaho, Pocatello, Idaho.
- MARCHINTON, R. L. AND D. H. HIRTH. 1984. Behavior *In White-tailed deer: Ecology and management*, L. K. Halls (ed). The Wildlife Management Institute, Washington D. C., pp. 129-168.

MCGARIGAL, K. AND B. J. MARKS. 1993. FRAGSTATS[®]: Spatial pattern analysis program for quantifying landscape structure (Version 1.0). Forest Science Department, Oregon State University, Corvallis, Oregon.

MCLEAN, R. G. 2001. Changing patterns of wildlife diseases. *Transactions of the North American Wildlife and Natural Resource Conference* 66:320-326.

MCSHEA, W. J., H. B. UNDERWOOD, AND J. H. RAPPOLE (eds). 1997. *The science of overabundance: Deer ecology and population management*. Smithsonian Institution Press, Washington D. C., 402 pp.

MOKRY, J., H. ARTSOB, AND R. BUTLER. 1984. Studies on California serogroup virus activity in Newfoundland, Canada, 1980-83. *Mosquito News* 44:310-314.

MURPHY, R. K., N. F. PAYNE, AND R. K. ANDERSON. 1985. White-tailed deer use of an irrigated agriculture-grassland complex in Central Wisconsin. *Journal of Wildlife Management* 49(1):125-128.

NIXON, C. M., L. P. HANSEN, P. A. BREWER, AND J. E. CHELSVIG. 1991. Ecology of white-tailed deer in an intensively farmed region of Illinois. *Wildlife Monographs* 118, 77 pp.

_____, _____, _____, _____, J. B. SULLIVAN, T. ESKER, R. K. KOERKENMEIER, D. R.

ETTER, J. CLINE, AND J. A. THOMAS. 1994. Behavior, dispersal, and survival of male white-tailed deer in Illinois. *Biological Note* 139, Illinois Natural History Survey, Urbana, Illinois.

_____, P. C. MANKIN, D. R. ETTER, L. P. HANSEN, P. A. BREWER, J. E. CHELSVIG, T. E.

ESKER, AND J. B. SULLIVAN. 2007. White-tailed deer dispersal behavior in an agricultural environment. *American Midland Naturalist* 157:212-220.

- OTIS, D. L. AND G. C. WHITE. 1999. Autocorrelation of location estimates and the analysis of radio-tracking data. *Journal of Wildlife Management* 63:1039-1044.
- PENDLETON, G. W., K. TITUS, E. DEGAYNER, C. J. FLATTEN, AND R. E. LOWELL. 1998. Compositional analysis and GIS for study of habitat selection by goshawks in southeast Alaska. *Journal of Agricultural, Biological, and Environmental Statistics* 3(3):280-295.
- PHEREZ, F. M. 2007. Factors affecting the emergence and prevalence of vector borne infections (VBI) and the role of vertical transmission (VT). *Journal of Vector Borne Diseases* 44:157-163.
- SAS INSTITUTE. 2000. JMP[®] statistics and graphics guide, Version 3. SAS Institute, Inc., Cary, North Carolina.
- SOKAL, R. R. AND F. J. ROHLF. 1995. *Biometry*. W. H. Freeman and Company, New York, New York.
- STORM, D. J., C. K. NIELSEN, E. M. SCHAUBER, AND A. WOOLF. 2006. Space use and survival of white-tailed deer in an exurban landscape. *Journal of Wildlife Management* 71:1170-1176.
- SWIHART, R. K., P. M. PICONE, A. J. DENICOLA, G. S. KANIA, AND L. CORNICELLI. 1995. Ecology of urban and suburban white-tailed deer. *In* *Urban deer: A manageable resource?*, J. B. McAninch (ed). Proceedings of the 1993 Symposium of the North Central Section, The Wildlife Society, pp. 35-44.
- TRAINER, D. O. 1973. Wildlife as monitors of disease. *American Journal of Public Health* 63(3):201-203.
- WILSON, M. E. 2000. Environmental change and infectious disease. *Ecosystem Health* 6(1):7-12.

YUILL, T. M. AND C. SEYMOUR. 2001. Arbovirus infectious. *In* Infectious Diseases of Wildlife Mammals, E. S. Williams and I. K. Barker (eds). Iowa State University Press, Ames, Iowa, pp. 98-118.

CHAPTER 3
SPATIAL AND TEMPORAL ANALYSIS OF ENCEPHALITIDES
IN SUBURBAN DEER

ABSTRACT: Spatial and temporal epidemiology examines patterns of disease providing insight into vector/host distribution and potential for disease transmission. Blood samples from live-captured and culled suburban white-tailed deer (*Odocoileus virginianus*) from 11 Chicago, Illinois forest preserves were tested for Jamestown Canyon (JC; n=238 seropositive cases, 402 seronegative cases) and LaCrosse (LAC; n=51 seropositive cases, 402 seronegative cases) encephalitis viral antibodies from 1995-99. Using the Bernoulli model in the space-time scan statistic in SaTScan[®], a significant viral “hot spot” was revealed for JC in Des Plaines forest preserve (DP; p=0.009). A significant cluster for LAC virus was also found in DP (p=0.005). All clusters were independent of each other. Temporal patterns for JC virus spanned samples collected from December 1, 1995 to March 31, 1998. Clustering suggests interactions between deer, vectors, and JC and LAC viruses. Examination of the distribution and spatial relationships for each virus allows for identification of potential focal locations. These results indicate deer can serve as sentinels for JC and LAC viruses while providing information about spatial distributions on important diseases to management and policy makers.

INTRODUCTION

Organisms are distributed neither uniformly nor randomly (Legendre and Fortin, 1989) and zoonoses also exhibit clustering in space and time (Ward and Carpenter, 2000). By examining landscape patterns of pathogens, scientists may be able to link risk factors and predict where outbreaks would commonly occur. Analysis for geographic clustering of zoonotic events can provide clues to the components of a disease process because common risk factors may be

responsible for observed events. Various disease models and geographic risk maps have been created to describe spatial and/or temporal patterns to interpret disease processes (Siegel et al., 1991; Kitron et al., 1992; Lockhart et al., 1996; Joly et al., 2003).

All arboviral encephalitides are zoonotic, have a global distribution, and are maintained through complex life cycles between vertebrate hosts and arthropod vectors (Centers for Disease Control, 2005). Jamestown Canyon (JC) virus occurs throughout much of temperate North America. White-tailed deer (*Odocoileus virginianus*) are the primary reservoir host and boreal *Aedes* mosquitoes are the primary vectors (Andreadis et al., 2008). Perennial exposures of Jamestown Canyon virus in deer occur as early as February through October with 100% of captive animals seroconverting within 2-10 weeks of exposure (Grimstad et al., 1987). Jamestown Canyon's temporal transmission occurs from months to years and most human cases are reported between April and October (Grimstad, 1988).

LaCrosse encephalitis (LAC) has a focal distribution in the north-central/eastern United States and is the leading cause of pediatric encephalitis (Elmore et al., 2000). In the Midwest, LAC produces continuous seasonal endemic infections (Grimstad et al., 1994). Incidences of LAC are highly seasonal, depending on the vector (*A. triseriatus*), weather, viral amplification, and host presence (Clark et al., 1983; Acha and Szyfres, 1994).

The objective of this study was to examine spatial and temporal foci for JC and LAC viral antibodies in live-captured and culled white-tailed deer from 11 Chicago, Illinois forest preserves from 1995-99. Analyzing spatial and temporal patterns for JC and LAC can identify focal areas for potential human exposure. Disease clustering provides compelling evidence to help improve control and prevention programs (Ward and Carpenter, 2000).

MATERIALS AND METHODS

General materials and methods are specified in pages 14-21.

ArcView[®] 3.2 (Environmental Systems Research Institute Inc., Redlands, California, USA; ESRI, 1996) software was used to create data layers of plotted locations for live tagged and culled deer, home ranges for radio-collared deer, calculated point of central tendency for radio-collared deer, and land cover generated by the Illinois Natural History Survey (INHS) from thematic mapper (TM) 1999 images.

Data recorded for each deer included sera samples classified as seropositive (case) or seronegative (control) for JC or LAC viruses, x-y coordinates generated in ArcView, date, age, and sex. Coordinates were based upon point removal locations for all sites, tagged deer capture location, or point of central tendency for radio-collared deer from Des Plaines (DP) and Palos. Data were analyzed using the Bernoulli model in SaTScan[®] 4.0.3 with retrospective space-time scan statistics creating log likelihood ratios (LLR) and Monte Carlo simulations generating p-values (SaTScan, National Cancer Institute, Bethesda, Maryland, USA; Kulldorff and Nagarwalla, 1995; Kulldorff et al., 1997; Kulldorff, 2002). LLR indicates how much the positive odds for disease increases in a specific location and/or time when compared to non-diseased individuals (Pfeiffer, 2002). The LLR statistic was calculated for each random replication as well as for the true data set. If the true data set was among the highest 5%, then the test was significant at the $p \leq 0.05$ level (Viel et al., 2000). Relative risk (RR) was calculated as the estimated risk within the cluster divided by the estimated risk outside the cluster.

A space-time statistic generates a cylindrical window. The base is defined by the infinite number of distinct geographic circles encompassing seropositive versus seronegative candidates with overlapping potential candidate clusters. The height reflects the time period of each

potential cluster. The cluster windows are then moved in space and time so that each possible location overlaps each time period creating an infinite number of overlapping potential clusters. The maximum possible spatial cluster size was set at 50% of the total population at risk. P-values were obtained by repeating the same analytical functions on a large number of random replications of the data (Kulldorff and Nagarwalla, 1995; Kulldorff et al., 1998; Smith et al., 2000). Output files from SaTScan were imported into ArcView to create maps.

RESULTS

Jamestown Canyon virus samples included 238 seropositive and 402 seronegative cases. LaCrosse virus samples included 51 seropositive and 402 seronegative cases. Spatial analysis resulted in two clusters for JC virus. The DP cluster contained 62 cases including a radio-collared doe from Beck Lake in DP ($p=0.009$; Figs. 3, 4). Relative risk was 1.60 (or 1.6 times more likely to be positive than random data replications), indicating an increased risk of pathogen exposure within this region. Temporal patterns included deer sampled from December 1, 1995 to March 31, 1998. Space and time clustering indicated $LLR=12.77$ representing an independence of clustering.

Spatial analysis for LAC virus contained only one cluster for exposure at Dam 1 in DP ($p=0.005$; Fig. 5). Six clusters in Palos were identified as secondary but were not statistically significant. The DP cluster included eight LAC viral cases with increased risk for exposure in the northern DP region ($RR=6.88$). Space and time analysis resulted in $LLR=12.99$ representing an independence of clustering.

DISCUSSION

It is important to define the distribution of a disease within its overall geographic range (Wobeser, 2007). In this study of suburban white-tailed deer, there was a northern cluster for JC

virus in the areas sampled from Chicago, Illinois. In Indiana white-tailed deer, Boromisa and Grimstad (1987) reported a difference in JC antibody prevalence between north and south zones. This pattern indicates a vector mosquito (Aedes) and deer association likely mediated by open water and riparian corridors. JC vectors require transient to permanent ground pools for breeding and egg survival. Radio-collared doe #98 was positive for JC virus and her home-range overlaps with the identified JC virus positive cluster at Beck Lake in DP (Fig. 4). Her home range also overlapped open water and riparian corridors including Beck Lake and the DesPlaines river. Aedes mosquitoes are present and documented by various mosquito abatement districts in the Chicago region, including the DP forest preserve (Northwest Mosquito Abatement District, pers. comm.). JC vectors require transient to permanent ground pools for breeding and egg survival.

Spatial distribution of disease factors must always be considered in conjunction with time and changes in distribution over time (Wobeser, 2007). Timing of JC transmission was associated with spring or summer Aedes mosquito populations for the years sampled. Temporal analysis for JC viral positive deer collected between December, 1995 and March, 1998 indicated repeated viral exposures and annual pathogen presence. The incidence of many pathogens and parasites are well documented as seasonal (Altizer et al., 2006). Annual patterns in host dynamics and biology can generate incidences around the same time each year. The timing for host exposure in wildlife populations can be used to estimate the timing and severity of viral presence. In the upper Midwest, JC virus is transmitted to deer in mid-to late spring with the emergence of the first over wintering Aedes mosquitoes (Grimstad, 1988; Heard et al., 1990) with annual reinfection occurring (Neitzel and Grimstad, 1991). Seasonal variation in rainfall and temperature can limit or enhance the abundance of mosquito vectors.

LaCrosse virus spatial clusters located at Dam 1 in DP (Fig. 5) indicates the presence of the specific mosquito vector, A. triseriatus (Clark et al., 1983; Osorio et al., 1996). Risk factors for LAC virus include residence in or near forested areas with water, presence of tree-holes, or discarded tires (Kitron et al., 1997). The mosquitoes require tree-holes or artificial containers of water for egg laying and vertebrates for blood, preferring white-tailed deer (Burkot and Defoliart, 1982; Nasci, 1985; and Boromisa and Grimstad 1986). Once the vector is established in a focal woodlot, white-tailed deer can amplify the LAC virus. The virus can persist for years and be annually isolated from the same mosquito tree-hole breeding site (Clark et al., 1983). LaCrosse viral infections in humans are associated with the extension of residential areas into forested sites and considered an emerging disease in the early 1960's when large developmental impacts began (Kitron et al., 1997).

Spatial clusters for JC and LAC pathogens were detected in suburban forest preserves surrounding Chicago, Illinois using white-tailed deer as a sentinel. Infectious diseases, including zoonoses, exhibit classic space and time clustering because of natural processes. Emerging infectious disease investigations depend on a temporal or spatial point of origination to determine the "event's" starting point. Globally, emerging infectious diseases are on the rise and over 70% of the pathogens originate from a wildlife source (Jones et al., 2008). It is predicted that 75% of human populations will live in urban/suburban environments in the next quarter century. A vast majority of the growth will occur in less-developed countries with poor infrastructures and resources (Cunningham and Cunningham, 2009a). Growing populations also push people into remote areas where humans encounter disease that may have existed for a long time, but now humans and other animal species are exposed (Cunningham and Cunningham, 2009b). Thus, scientists must continue to explore wildlife surveillance options to protect human

health because urban/suburban environments are continuing to expand. Public health organizations have focused on large incidences of disease mortality in the past and this fails to capture the impacts of disease emergence over time (Cunningham and Cunningham, 2009b). Monitoring for zoonoses will identify potential endemic and emerging or epidemic conditions. Creating more detailed risk assessment maps with spatial epidemiology will allow for interpretation of biotic and abiotic factors identified through surveillance programs and contribute to understanding pathogens and vectors over space and time.

Knowledge about host wildlife populations and disease processes can improve our understanding of pathogen patterns. Deer presence can exert a powerful influence on vector-borne zoonoses, not as a reservoir, but by affecting the distribution and abundance of the pathogenic vector (Wilson and Childs, 1997). Spatial “hot spots” and time relationships for JC and LAC viruses were found in suburban white-tailed deer from Cook County, Illinois forest preserves. Suitable habitat is present in Cook County forest preserves for specific pathogen vectors (Hollis, 2011) and spatial analysis identified focal locations for JC and LAC. The cluster locations can be examined further for specific mosquito species that may transmit encephalitis viruses. In addition, vector species in these study sites could be specifically monitored for spring and fall emergence patterns and changes in potential species transmitting the viruses; both influencing transmission potential and duration of disease. The results quantify deer prevalence data and can be used as a baseline reference for any future JC or LAC virus reemerging disease events in the Chicago region. Spatial statistics and knowledge of pathogen patterns provides a means to assess clustering of seropositive cases and can assist in pathogen surveillance and virus detection.

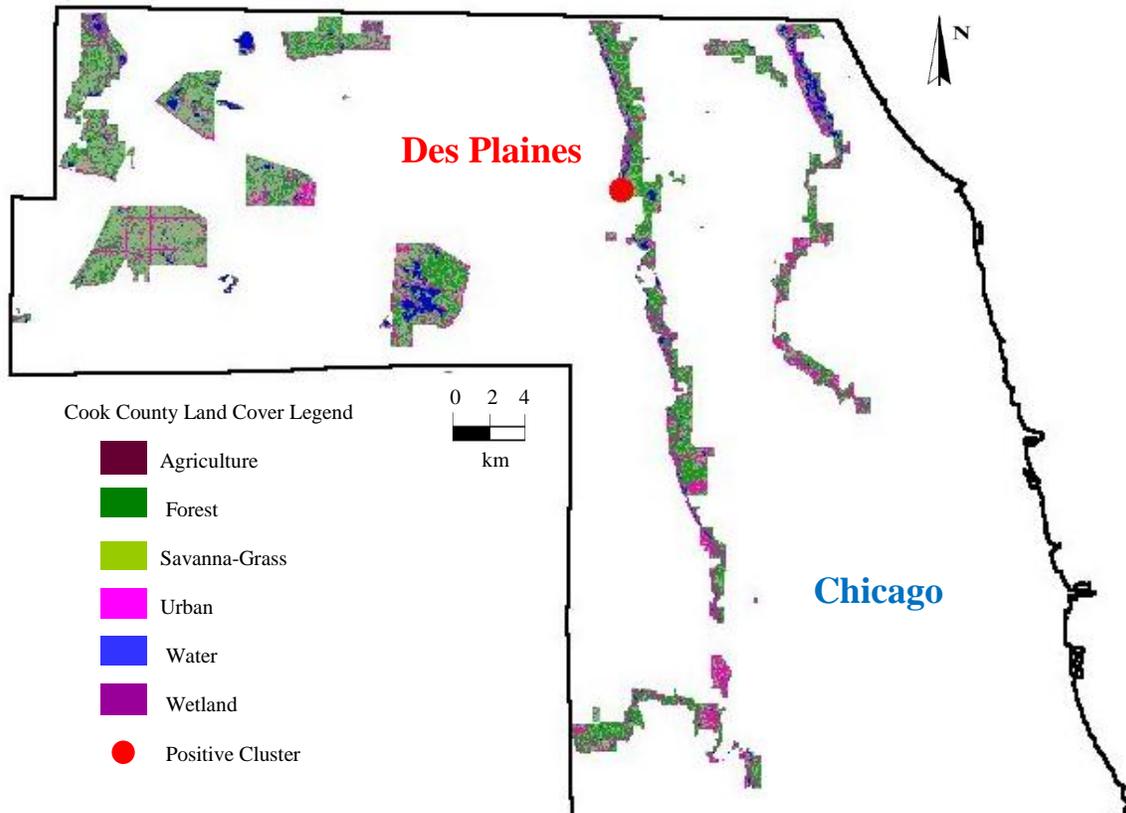


FIGURE 3. Location of Jamestown Canyon (JC) viral cluster in suburban white-tailed deer from DesPlaines area of the Cook County Forest Preserve, Chicago, Illinois, USA, 1995-99 ($p=0.009$).

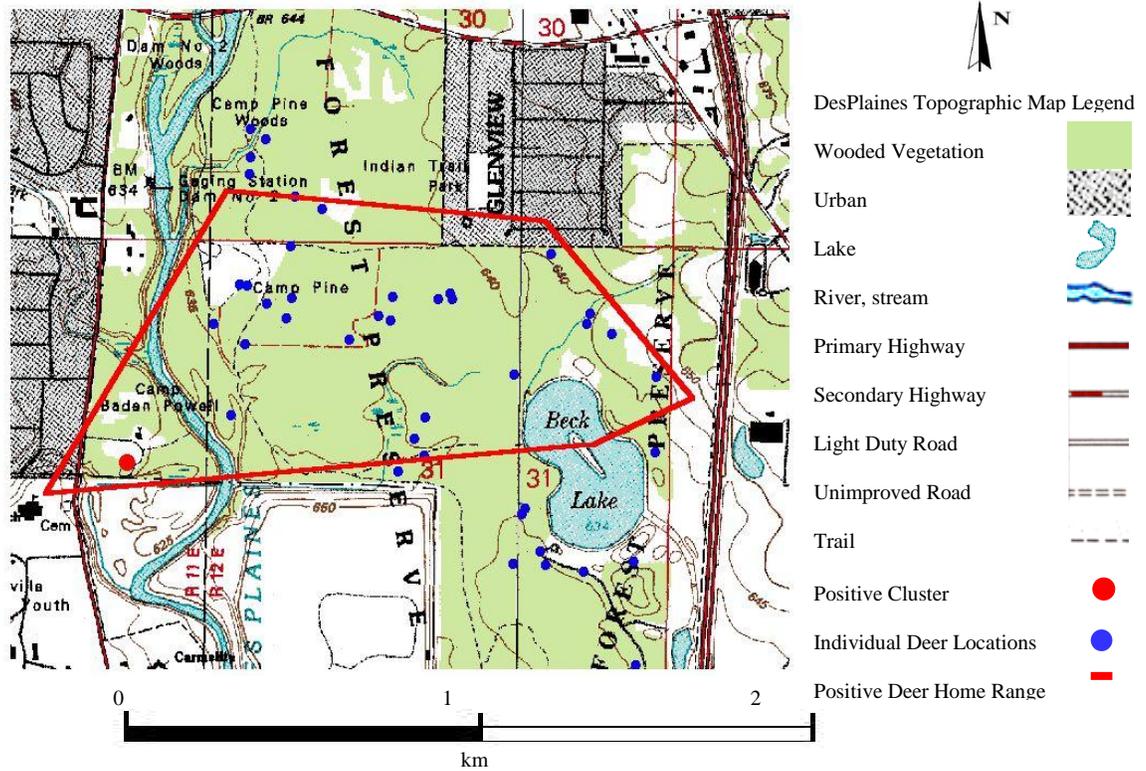


FIGURE 4. Representation of Beck Lake in Des Plaines Cook County Forest Preserve. The calculated seropositive cluster for deer (red dot) in this location and locations of all individual seropositive white-tailed deer (blue dots) for Jamestown Canyon (JC) virus from Chicago, Illinois, USA, 1995-99 ($p=0.009$). Home range of seropositive radio-collared doe #98 is included in this cluster.

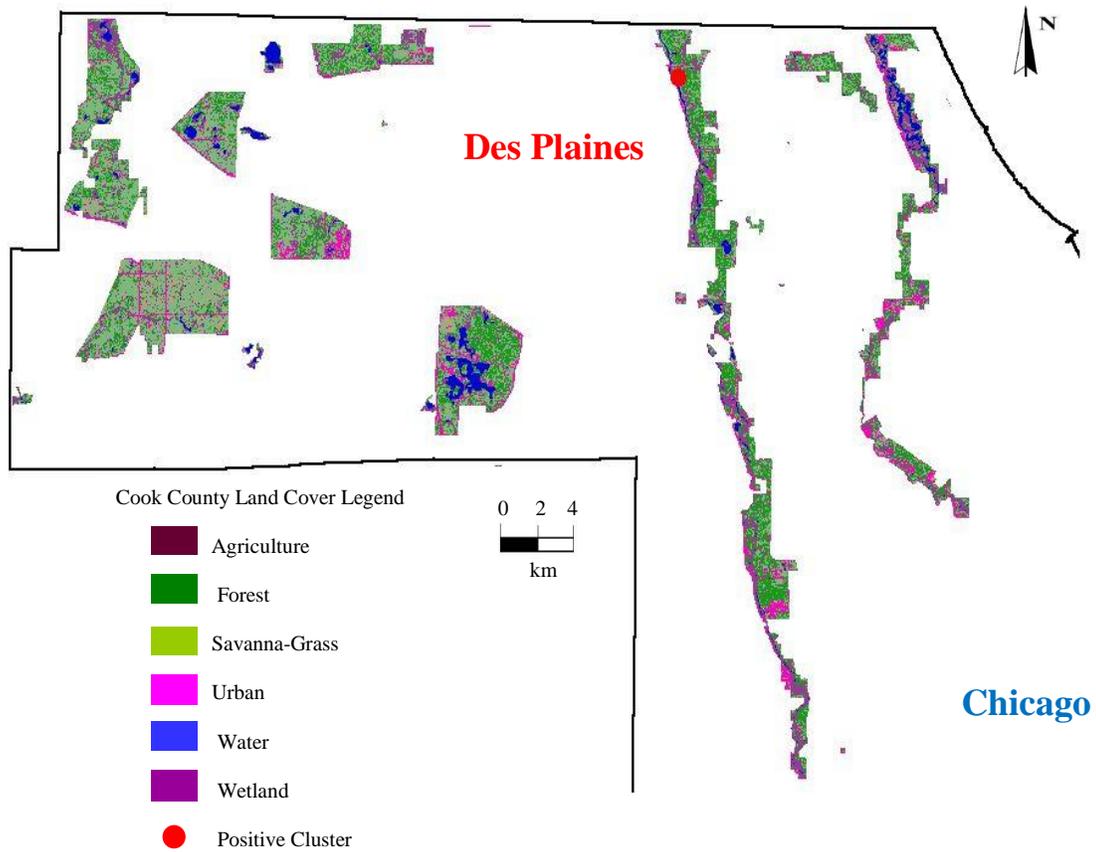


FIGURE 5. Location of LaCrosse (LAC) virus cluster in suburban white-tailed deer at Dam 1 in Des Plaines area of the Cook County Forest Preserve, Chicago, Illinois, USA, 1995-99 ($p=0.005$).

LITERATURE CITED

- ACHA, P. N. AND B. SZYFRES. 1994. California encephalitis. *In* Zoonoses and communicable diseases common to man and animals, 2nd Edition, Pan American Health Organization, Washington, D.C., pp. 304-308.
- ALTIZER, S., A. DOBSON, P. HOSSEINI, P. HUDSON, M. PASCUAL, AND P. ROHANI. 2006. Seasonality and the dynamics of infectious disease. *Ecology Letters* 9:467-484.
- ANDREADIS, T. G., J. F. ANDERSON, P. M. ARMSTRONG, AND A. J. MAIN. 2008. Isolations of Jamestown Canyon virus (Bunyaviridae: Orthobunyavirus) from field-collected mosquitoes (Diptera: Culicidae) in Connecticut, USA: A ten-year analysis, 1997-2006. *Vector-Borne and Zoonotic Diseases* 8(2):175-188.
- BOROMISA, R. D. AND P. R. GRIMSTAD. 1986. Virus-vector- host relationships of Aedes stimulans and Jamestown Canyon virus in a Northern Indiana enzootic focus. *American Journal of Tropical Medicine and Hygiene* 35(6):1285-1295.
- _____ AND _____. 1987. Seroconversion rates to Jamestown Canyon virus among six populations of white-tailed deer (Odocoileus virginianus) in Indiana. *Journal of Wildlife Diseases* 23(1):23-33.
- BURKOT, T. R. AND G. R. DEFOLIART. 1982. Bloodmeal sources of Aedes triseriatus and Aedes vexans in a southern Wisconsin forest endemic for LaCrosse encephalitis virus. *American Journal of Tropical Medicine and Hygiene* 31(2):376-381.
- CENTERS FOR DISEASE CONTROL. 2005. Information on arboviral encephalitides. Division of Vector-Borne Infectious Diseases, Fort Collins, Colorado.

- CLARK, G. C., H. L. PRETULA, C. W. LANGKOP, R. J. MARTIN, AND C. H. CALISHER. 1983. Persistence of LaCrosse virus (California encephalitis serogroup) in North-Central Illinois. *American Journal of Tropical Medicine and Hygiene* 32:175-184.
- CUNNINGHAM, W. P. AND M. A. CUNNINGHAM. 2009a. Economics and urbanization. *In Principles of environmental science: Inquiry and applications*. McGraw Hill Companies, New York, New York, pp. 328-351.
- ____ AND _____. 2009b. Environmental health and toxicology. *In Principles of environmental science: Inquiry and applications*. McGraw Hill Companies, New York, New York, pp. 175-198.
- ELMORE, S. K., M. J. LANCASTER, J. ANDERSON, J. SWANSON, M. CRANDELL, L. HARAMIS, P. GRIMSTAD, C. JONES, AND U. KITRON. 2000. LaCrosse encephalitis infection patterns in chipmunks and squirrels in Peoria, Illinois. *American Journal of Tropical Medicine and Hygiene* 62(3):374.
- ESRI (ENVIRONMENTAL SYSTEMS RESEARCH INSTITUTE). 1996. ArcView® GIS: The geographic information system for everyone. Redlands, California.
- GRIMSTAD, P. R. 1988. California group virus disease. *In The arboviruses: Epidemiology and ecology*, Volume 2, T. P. Monath (ed). CRC Press, Boca Raton, Florida, pp. 99-136.
- _____. 1994. California group viral infections. *In Handbook of zoonoses*, Section B viral, G. W. Beran (ed). CRC Press, Boca Raton, Florida, pp. 71-79.
- _____, D. G. WILLIAMS, AND S. M. SCHMITT. 1987. Infection of white-tailed deer (*Odocoileus virginianus*) in Michigan with Jamestown Canyon virus (California Serogroup) and the importance of maternal antibody viral maintenance. *Journal of Wildlife Diseases* 23(1):12-22.

HEARD, P. B., M. ZHANG, AND P. R. GRIMSTAD. 1990. Isolations of Jamestown Canyon Virus (California Serogroup) from Aedes mosquitoes in an enzootic focus in Michigan. *Journal of the American Mosquito Control Association* 6:461-468.

HOLLIS, K. M. 2011. Epidemiology of suburban deer. Ph.D. Thesis. University of Illinois, Urbana-Champaign.

JOLY, D. O., C. A. RIBIC, J. A. LANGENBERG, K. BEHELER, C. A. BATHA, B. J. DHUEY, R. E. ROLLEY, G. BARTELT, T. R. VAN DEELEN, AND M. D. SAMUEL. 2003. Chronic wasting disease in free-ranging Wisconsin white-tailed deer. *Emerging Infectious Diseases* 9(5):599-601.

JONES, K. E., N. G. PATEL, M. A. LEVY, A. STOREYGARD, D. BALK, J. L. GITTLEMAN, AND P. DASZAK. 2008. Global trends in emerging infectious diseases. *Nature* 451:990-993.

KITRON, U., C. J. JONES, J. K. BOUSEMAN, J. A. NELSON, AND D. L. BAUMGARTNER. 1992. Spatial analysis of the distribution of *Ixodes dammini* (Acari: Ixodidae) on white-tailed deer in Ogle County, Illinois. *Journal of Medical Entomology* 29:259-266.

_____, J. MICHAEL, J. SWANSON, AND L. HARAMIS. 1997. Spatial analysis of the distribution of LaCrosse encephalitis in Illinois, using a geographic information system and local and global spatial statistics. *Journal of Tropical Medicine and Hygiene* 57(4):469-475.

KULLDORFF, M. 2002. SaTScan[®] user guide for version 3.0. National Cancer Institute, Bethesda, Maryland, 59 pp.

_____, W. F. ATHAS, E. J. FEUER, B. A. MILLER, AND C. R. KEY. 1998. Evaluating cluster alarms: A space-time statistic and brain cancer in Los Alamos. *American Journal of Public Health* 88:1377-1380.

- _____, E. J. FEUER, B. A. MILLER, AND L. S. FREEDMAN. 1997. Breast cancer clusters in the northeastern United States: A geographic analysis. *American Journal of Epidemiology* 146:161-170.
- _____ AND N. NAGARWALLA. 1995. Spatial disease clusters: Detection and inference. *Statistics in Medicine* 14:799-810.
- LEDGENDRE, P. AND M. J. FORTIN. 1989. Spatial pattern and ecological analysis. *Vegetation* 80(2):101-138.
- LOCKHART, J. M., W. R. DAVIDSON, D. E. STALLKNECHT, AND J. E. DAWSON. 1996. Site-specific geographic association between Amblyomma americanum (Acari: Ixodidae) infestations and Ehrlichia chaffeensis-reactive (Rickettsiales: Ehrlichieae) antibodies in white-tailed deer. *Journal of Medical Entomology* 33:153-158.
- NASCI, R. S. 1985. Local variation in blood feeding by Aedes triseriatus and Aedes hendersoni (Diptera: Culicidae). *Journal of Medical Entomology* 22:619-623.
- NEITZEL, D. F. AND P. R. GRIMSTAD. 1991. Serological evidence of California group and cache valley virus infection in Minnesota white-tailed deer. *Journal of Wildlife Diseases* 27(2):230-237.
- OSORIO, J. E., M. S. GODSEY, G. R., DEFOLIART, AND T. M. YUILL. 1996. LaCrosse viremias in white-tailed deer and chipmunks exposed by injection or mosquito bite. *American Journal of Tropical Medicine and Hygiene* 54(4):338-342.
- PFEIFFER, D. U. 2002. *Veterinary epidemiology – An introduction*. University of London, 62 pp.

SIEGEL, J. P., U. KITRON, AND J. K. BOUSEMAN. 1991. Spatial and temporal distribution of Ixodes dammini (Acari: Ixodidae) in a northwestern Illinois state park. *Journal of Medical Entomology* 28:101-104.

SMITH, K. L., V. DEVOS, H. BRYDEN, L. B. PRICE, M. E. HUGH-JONES, AND P. KEIM. 2000. Bacillus anthracis diversity in Kruger National Park. *Journal of Clinical Microbiology* 38:3780-3784.

VIEL, J-F., P. ARVEUX, J. BAVEREL, AND J.-Y. CAHN. 2000. Soft-tissue sarcoma and non-Hodgkin's lymphoma clusters around a municipal solid waste incinerator with high dioxin emission levels. *American Journal of Epidemiology* 152:13-19.

WARD, M. P. AND T. E. CARPENTER. 2000. Analysis of space-time clustering in veterinary epidemiology. *Preventive Veterinary Medicine* 43:225-237.

WILSON, M. L. AND J. E. CHILDS. 1997. Vertebrate abundance and the epidemiology of zoonotic diseases. *In* The science of overabundance: Deer ecology and population management, W. J. McShea, H. B. Underwood, and J. H. Rappole (eds). Smithsonian Institution Press, Washington D. C., pp. 224-248.

WOBESER, G. A. 2007. Disease in wild animals: Investigation and management, 2nd edition. Springer-Verlag, Heidelberg, Germany, 393 pp.

MANAGEMENT RECOMMENDATIONS

Management of zoonoses and disease in wild mammals should be sustainable, based on sound science, and balance the requirements for preserving biodiversity while protecting the health of humans, domestic pets, and livestock. The use of wildlife as sentinels is a valuable approach to surveillance for emerging infectious diseases. This study of suburban white-tailed deer suggests a proactive approach for zoonotic surveillance and validates deer as an effective biomonitor. Results will provide baseline information to state and county agencies while bringing worldwide awareness to suburban zoonoses in the United States. Understanding the seroprevalence of pathogens is necessary to make informed decisions in management of wildlife-human-pet interactions. This information will allow the appropriate agencies to implement control and management plans while circulating public health information.

Results from this study indicate the presence and prevalence for pathogens in Cook County Forest Preserves that may cause Jamestown Canyon (JC) and LaCrosse (LAC) encephalitis, toxoplasmosis, and leptospirosis. Recommendations for wildlife management and surveillance include, 1) Continue culling programs to maintain deer populations at low densities. This will reduce animal to animal contact and pathogen transmission, potential human exposures, and hosts for parasites and vectors. 2) Continue county-wide surveillance of wildlife populations including deer for zoonoses and emerging infectious diseases. This will allow scientists and managers to distinguish between endemic and emerging or epidemic conditions. 3) Yearling deer should be sampled for new annual exposures and adults for long-term repeated exposures. Sampling for fawns may not indicate immediate exposures for all zoonoses. 4) Use identified “hot spots” for specific pathogen monitoring while continuing to identify new locations from surveillance and as human cases occur. 5) Implement and focus monitoring and control

programs for vectors like mosquitoes while considering breeding habitats. 6) Reduce human-wildlife-pet interactions to decrease possible zoonotic exposures, especially wildlife feeding. 7) Conduct fine-scale habitat analysis to examine relationships between vectors and pathogens. 8) Sampling should increase after floods and during abnormal seasonal weather to monitor for changes in vectors and zoonoses. 9) Deer are the primary vertebrate host for JC virus and likely amplifiers for LAC virus transmission. Additional research is needed to better understand the ecological relationships among vectors, deer, and pathogens.

Additional recommendations for the public, public health agencies, and veterinarians include, 1) Proper hand washing for all people visiting forest preserves to decrease the risk of infection through ingestion of pathogens. 2) Public education about common forms of zoonotic exposures in forest preserves and the common signs/symptoms of human infection. 3) Inform the public to reduce exposure to mosquitoes by remaining on forest preserve trails and direct or indirect use of chemical repellents. 4) Public education about the exposure to zoonoses from natural water resources like ponds, lakes and rivers. 5) Keep all pets on a leash to reduce possible exposure to zoonotic pathogens and vectors, including bringing vectors and parasites into the home. 6) Deer meat provided by the county to food depositories should be frozen in small packages (~2.2-4.6 kg) immediately after processing and cooked thoroughly before serving. 7) Clear brush and debris along trails to reduce habitat available for mosquito vectors. 8) Implement surveillance programs for human and pet zoonoses. Overlapping this information with deer surveillance will facilitate better decision making.

Appendix A. Suburban white-tailed deer (n=245) seropositive for Babesia spp. in Cook and DuPage Counties, Chicago, Illinois, USA, 1996-97.

Variables	N	Positive deer
Age		
Fawn	53	0
Yearling	71	0
Adult	121	1
Sex		
Female	164	1
Male	81	0
Cook County site		
Beck Lake	26	0
Bemis	14	0
Busse (North and South)	31	0
Camp Pine	22	0
Camp Sagawau	5	0
Crab Tree Nature Center	12	0
Dam 1	18	0
Palos	52	1
River Trail Nature Center	5	0
Somme	5	0
Swallow Cliff	15	0
Zander-Jurgenson	7	0
DuPage County site		
Blackwell	4	0
Greene Valley	8	0
Herrick Lake	1	0
Timber Ridge	1	0
Waterfall Glen	19	0
Year		
1996-97	245	1
Month		
December	33	0
January	62	0
February	50	0
March	99	1
August ^a	1	0

^a Radio-collared animal found dead in August and sampled.

Appendix B. Suburban white-tailed deer (n=1033) seropositive for Cache Valley virus in Cook and DuPage Counties, Chicago, Illinois, USA, 1995-99.

Variables	N ^a	Positive deer
Age		
Fawn	313	97
Yearling	230	92
Adult	490	172
Sex		
Female	739	272
Male	294	89
Cook County site		
Beck Lake	107	41
Bemis	57	25
Black Partridge	3	0
Busse (North and South)	12	49
Camp Pine	83	35
Camp Sagawau	19	8
Crab Tree Nature Center	47	15
Dam 1	50	13
Palos	211	51
Popular Creek	1	0
River Trail Nature Center	15	7
Somme	19	4
Swallow Cliff	68	19
Zander-Jurgenson	50	19
DuPage County site		
Blackwell	8	3
Greene Valley	34	8
Herrick Lake	17	7
Pratt's Wayne Woods	5	3
Timber Ridge	11	5
West DuPage Woods	6	3
Waterfall Glen	100	41
Winfield Mounds	3	2
Wood Ridge	6	3
Year		
1995-96	178	35
1996-97	244	58
1997-98	397	150
1998-99	214	118

Appendix B. Continued.

Variables	N ^a	Positive deer
Month		
December	122	48
January	276	102
February	316	110
March	318	101
August ^b	1	0

^aIncludes non-determined samples.

^bRadio-collared animal found dead in August and sampled.

Appendix C. Suburban white-tailed deer (n=49) seropositive for Ehrlichia chaffeensis in Cook County, Chicago, Illinois, USA, 1995-96.

Variables	N	Positive deer
Age		
Fawn	13	1
Yearling	8	0
Adult	28	0
Sex		
Female	38	0
Male	11	1
Cook County site		
Beck Lake	11	0
Bemis	3	0
Busse (North and South)	6	0
Camp Pine	3	0
Camp Sagawau	1	0
Crab Tree Nature Center	2	0
Palos	12	0
Somme	4	1
Swallow Cliff	2	0
Zander	5	0
Year		
1995-96	49	1
Month		
December	5	0
January	14	0
February	16	0
March	14	1

Appendix D. Suburban white-tailed deer (n=172)^a seropositive for Epizootic Hemorrhagic virus in Cook County, Chicago, Illinois, USA, 1995-96.

Variables	N	Positive deer
Age		
Fawn	48	1
Yearling	41	0
Adult	83	0
Sex		
Female	123	0
Male	49	1
Cook County site		
Beck Lake	30	0
Bemis	5	0
Black Partridge	3	1
Busse (North and South)	12	0
Camp Pine	18	0
Camp Sagawau	4	0
Crab Tree Nature Center	6	0
Dam 1	9	0
Palos	42	0
Poplar Creek	1	0
River Trail Nature Center	3	0
Somme	13	0
Swallow Cliff	13	0
Zander	13	0
Year		
1995-96	172	1
Month		
December	27	0
January	34	0
February	71	1
March	40	0

^aAll deer tested negative for bluetongue virus.

Appendix E. Suburban white-tailed deer (n=88) seropositive for Escherichia coli spp. in Cook County, Chicago, Illinois, USA, 1996-97.

Variables	N	Positive deer
Age		
Fawn	22	13
Yearling	20	18
Adult	46	41
Sex		
Female	67	55
Male	21	17
Cook County site		
Beck Lake	1	1
Bemis	6	5
Busse (North and South)	14	11
Camp Pine	8	6
Camp Sagawau	1	1
Crab Tree Nature Center	3	3
Dam 1	10	8
Palos	25	20
Somme	1	1
Swallow Cliff	14	12
Zander-Jurgenson	5	4
Year		
1996-97	88	72
Month		
January	7	6
February	30	25
March	51	41

Appendix F. Suburban white-tailed deer (n=1034) seropositive for Jamestown Canyon virus in Cook and DuPage Counties, Chicago, Illinois, USA, 1995-99.

Variables	N ^a	Positive deer
Age		
Fawn	314	14
Yearling	230	85
Adult	490	214
Sex		
Female	740	233
Male	294	80
Cook County site		
Beck Lake	107	42
Bemis	57	14
Black Partridge	3	0
Busse (North and South)	113	26
Camp Pine	83	38
Camp Sagawau	19	5
Crab Tree Nature Center	47	19
Dam 1	51	22
Palos	211	63
Popular Creek	1	0
River Trail Nature Center	15	5
Somme	19	5
Swallow Cliff	68	17
Zander-Jurgenson	50	13
DuPage County site		
Blackwell	8	3
Greene Valley	34	5
Herrick Lake	17	5
Pratt's Wayne Woods	5	2
Timber Ridge	11	4
West DuPage Woods	6	1
Waterfall Glen	100	23
Winfield Mounds	3	0
Wood Ridge	6	1
Year		
1995-96	178	28
1996-97	244	97
1997-98	398	142
1998-99	214	46

Appendix F. Continued.

Variables	N ^a	Positive deer
Month		
December	122	37
January	276	70
February	316	95
March	319	111
August ^b	1	0

^aIncludes non-determined samples.

^bRadio-collared animal found dead in August and sampled.

Appendix G. Suburban white-tailed deer (n=1034) seropositive for LaCrosse virus in Cook and DuPage Counties, Chicago, Illinois, USA, 1995-99.

Variables	N ^a	Positive deer
Age		
Fawn	314	14
Yearling	230	18
Adult	490	48
Sex		
Female	740	70
Male	294	10
Cook County site		
Beck Lake	107	10
Bemis	57	3
Black Partridge	3	0
Busse (North and South)	113	11
Camp Pine	83	2
Camp Sagawau	19	0
Crab Tree Nature Center	47	1
Dam 1	51	2
Palos	211	15
Popular Creek	1	0
River Trail Nature Center	15	0
Somme	19	1
Swallow Cliff	68	7
Zander-Jurgenson	50	5
DuPage County site		
Blackwell	8	0
Greene Valley	34	3
Herrick Lake	17	2
Pratt's Wayne Woods	5	0
Timber Ridge	11	1
West DuPage Woods	6	1
Waterfall Glen	100	14
Winfield Mounds	3	0
Wood Ridge	6	2
Year		
1995-96	178	9
1996-97	244	5
1997-98	398	41
1998-99	214	25

Appendix G. Continued.

Variables	N ^a	Positive deer
Month		
December	122	9
January	276	27
February	316	32
March	319	12
August ^b	1	0

^aIncludes non-determined samples.

^bRadio-collared animal found dead in August and sampled.

Appendix H. Suburban white-tailed deer (n=1032) seropositive for Leptospira spp. in Cook and DuPage Counties, Chicago, Illinois, USA, 1995-99.

Variables	N	Positive deer
Age		
Fawn	311	25
Yearling	231	42
Adult	490	90
Sex		
Female	739	110
Male	293	47
Cook County site		
Beck Lake	107	16
Bemis	57	7
Black Partridge	3	0
Busse (North and South)	113	18
Camp Pine	83	11
Camp Sagawau	19	2
Crab Tree Nature Center	47	7
Dam 1	51	4
Palos	210	41
Popular Creek	1	0
River Trail Nature Center	15	4
Somme	19	2
Swallow Cliff	68	6
Zander-Jurgenson	50	10
DuPage County site		
Blackwell	8	2
Greene Valley	34	4
Herrick Lake	17	1
Pratt's Wayne Woods	5	0
Timber Ridge	11	3
West DuPage Woods	6	1
Waterfall Glen	99	16
Winfield Mounds	3	0
Wood Ridge	6	2
Year		
1995-96	178	12
1996-97	245	29
1997-98	395	55
1998-99	214	61

Appendix H. Continued.

Variables	N	Positive deer
Month		
December	121	18
January	275	29
February	315	43
March	320	67
August ^a	1	0

^aRadio-collared animal found dead in August and sampled.

Appendix I. Suburban white-tailed deer (n=405)^a seropositive for Neospora caninum in Cook and DuPage Counties, Chicago, Illinois, USA, 1995-98.

Variables	N	Positive deer
Age		
Fawn	148	54
Yearling	66	31
Adult	191	81
Sex		
Female	298	118
Male	107	48
Cook County site		
Beck Lake	26	11
Bemis	18	9
Busse (North and South)	37	6
Camp Pine	29	13
Camp Sagawau	5	2
Crab Tree Nature Center	17	4
Dam 1	18	5
Palos	75	41
River Trail Nature Center	4	3
Somme	1	0
Swallow Cliff	21	14
Zander-Jurgenson	10	3
DuPage County site		
Blackwell	5	3
Greene Valley	20	8
Herrick Lake	14	5
Pratt's Wayne Woods	5	3
Timber Ridge	10	1
West DuPage Woods	6	5
Waterfall Glen	75	29
Winfield Mounds	3	1
Wood Ridge	6	0
Year		
1995-96	1	0
1996-97	33	16
1997-98	371	150

Appendix I. Continued.

Variables	N	Positive deer
Month		
December	49	17
January	105	43
February	118	48
March	132	58
August ^b	1	0

^aData published by J. P. DUBEY, K. HOLLIS, S. ROMAND, P. THULLIEZ, O. C. H. KWOK, L. HUNGERFORD, C. ANCHOR, AND D. ETTER. 1999. High prevalence of antibodies to *Neospora caninum* in white-tailed deer (*Odocoileus virginianus*). *International Journal of Parasitology* 29:1709-1711.

^bRadio-collared animal found dead in August and sampled.

Appendix J. Suburban white-tailed deer (n=178) seropositive for Potosi virus in Cook and DuPage Counties, Chicago, Illinois, USA, 1996-97.

Variables	N ^a	Positive deer
Age		
Fawn	50	28
Yearling	41	16
Adult	87	19
Sex		
Female	128	38
Male	50	25
Cook County site		
Beck Lake	30	12
Bemis	5	2
Black Partridge	3	3
Busse (North and South)	12	3
Camp Pine	18	4
Camp Sagawau	4	1
Crab Tree Nature Center	6	3
Dam 1	9	5
Palos	42	11
Popular Creek	1	0
River Trail Nature Center	3	1
Somme	13	6
Swallow Cliff	13	3
Zander-Jurgenson	13	7
DuPage County site		
Greene Valley	6	2
Year		
1996-97	178	63
Month		
December	27	14
January	36	13
February	75	23
March	40	13

^aIncludes non-determined samples.

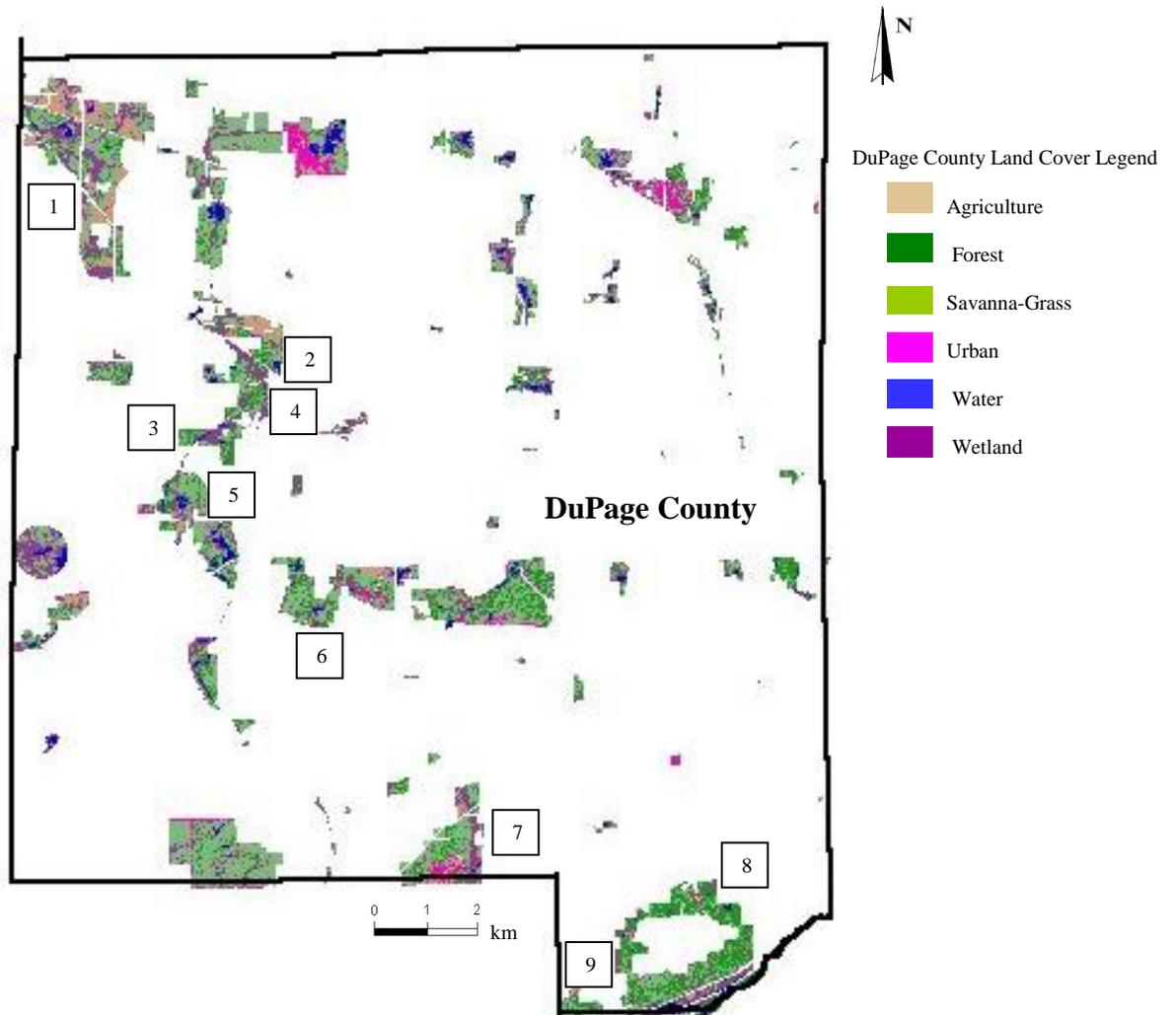
Appendix K. Suburban white-tailed deer (n=1024) seropositive for Toxoplasma gondii in Cook and DuPage Counties, Chicago, Illinois, USA, 1995-99.

Variables	N	Positive deer
Age		
Fawn	310	85
Yearling	229	117
Adult	485	296
Sex		
Female	734	366
Male	290	132
Cook County site		
Beck Lake	107	65
Bemis	57	39
Black Partridge	3	2
Busse (North and South)	112	47
Camp Pine	83	54
Camp Sagawau	18	12
Crab Tree Nature Center	47	16
Dam 1	51	41
Palos	210	83
Popular Creek	1	1
River Trail Nature Center	15	11
Somme	17	8
Swallow Cliff	68	16
Zander-Jurgenson	49	22
DuPage County site		
Blackwell	8	4
Greene Valley	28	12
Herrick Lake	17	5
Pratt's Wayne Woods	5	1
Timber Ridge	11	7
West DuPage Woods	6	4
Waterfall Glen	102	42
Winfield Mounds	3	1
Wood Ridge	6	5
Year		
1995-96	168	69
1996-97	245	134
1997-98	399	204
1998-99	212	91

Appendix K. Continued.

Variables	N	Positive deer
Month		
December	123	77
January	272	143
February	311	142
March	317	136
August ^a	1	0

^aRadio-collared animal found dead in August and sampled.



Appendix L. Map of DuPage County Forest Preserves, Chicago, Illinois, USA, 1995-99. White-tailed deer were sampled from the following study sites: 1=Pratt's Wayne Woods, 2=Timber Ridge, 3=West DuPage Woods, 4=Winfield Mounds, 5=Blackwell, 6=Herrick Lake, 7=Greene Valley, 8= Waterfall Glen, and 9=Wood Ridge.

Appendix M. Fragstats variables included in the original analysis to model habitat (forest, agriculture, savanna-grass, urban, water, wetland) for radio-collared and culled suburban white-tailed deer from Chicago, Illinois, USA, 1995-99.

Variable (measurement)	Abbreviation
Class area (ha)	CA
Total landscape area (ha)	TA
Percent of landscape	%Land
Largest patch index	LPI
Number of patches	NP
Patch density (#/100 ha)	PD
Mean patch size (ha)	MPS
Patch size standard deviation (ha)	PSSD
Patch size coefficient of variation (%)	PSCV
Total edge (m)	TE
Edge density (m/ha)	ED
Landscape shape index	LSI
Mean shape index	MSI
Area weighted mean shape index	AWMSI
Double log fractal dimension	DLFD
Mean patch fractal dimension	MPFD
Area weighted mean patch fractal dimension	AWMPFD
Core area percent of landscape	C%Land
Total core area (ha)	TCA
Number of core areas	NCA
Core area density (#/100 ha)	CAD
Mean core area per patch (ha)	MCA1
Patch core area standard deviation (ha)	CASD1
Patch core area coefficient of variation (%)	CACV1
Mean area per disjunct core (ha)	MCA2
Disjunct core area standard deviation (ha)	CASD2
Disjunct core area coefficient of variation (%)	CACV2
Total core area index (%)	TCAI
Mean core area index (%)	MCAI
Mean nearest neighborhood distance (m)	MNN
Nearest neighborhood standard deviation (m)	NNSD
Nearest neighborhood coefficient of variation (%)	NNCV
Interspersion and juxtaposition index (%)	IJI