Effects of Landscape Spatial Heterogeneity on Host-Parasite Ecology

by

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Abstract

Landscape spatial heterogeneity interacts with ecological processes that influence pathogen emergence and infectious disease spread. Modification of landscape composition and configuration is hypothesized to alter host densities, frequency of host-parasite interactions, host community assembly processes, and dispersal processes of host, vector, and parasite populations. Understanding the spatial and environmental dependence on host-parasite interactions is, therefore, critical in predicting the response of disease dynamics to widespread habitat modification. I combine wildlife disease ecology with landscape ecological concepts and varied modeling techniques (i.e., multivariate redundancy analyses, network connectivity models, bipartite mutualistic networks, beta diversity analyses) to test the mediating role of landscape spatial heterogeneity on the spread of a vector-borne disease system (chapter 2), and the effect of urban landscape spatial heterogeneity on host exposure to parasitism (chapter 3), host-parasite interaction structure (chapter 4), and host-parasite beta diversity (chapter 5). My results demonstrate that landscape spatial heterogeneity has a differential influence on tick dispersal than pathogen dispersal by the movement of a host community, most critically mediated by stepping-stone habitat. I also provide evidence that urban landscape spatial heterogeneity affects the exposure likelihood of a single host population, the structure of host-parasite interactions in
multiple host populations, and the spatial contributions of host and parasite beta diversity. My work demonstrates that landscape spatial heterogeneity has a mediating role in multiple disease systems whereby spatial and environmental factors play a critical role in the likelihood of short- and long-distance pathogen invasion, the persistence of environmentally-mediated parasites, and the selection of generalist host and parasite species in urbanized habitat. Overall my research contributes to our theoretical understanding of the multi-scalar ecological interactions between landscape change and disease dynamics and our applied understanding of the role of landscape in the emergence of infectious diseases.
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Permission to publish these manuscripts has been obtained from the co-authors.


Publications related to work conducted at the University of Toronto during my PhD include:


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Chapter 1

1 Introduction

1.1 Spatial dependence in disease ecology

The study of the ecology of disease in natural populations aims at understanding how environmental conditions shape species interactions that drive disease dynamics (Holt 1977, Anderson and May 1978, Price et al. 1986, Poulin 2010). At a minimum, infectious diseases involve interactions among at least two species, the pathogen and the host species it infects (Ostfeld et al. 2008). While some host-parasite relationships have evolved specialist interactions involving only one host and one parasite species, many single pathogen species infect multiple hosts (Woolhouse et al. 2001, Roche et al. 2012, Buhnerkempe et al. 2015) or are mediated by at least one species of vector, such as a mosquito or tick (Keesing et al. 2009, Colwell et al. 2011). Despite the ubiquity of host-parasite interactions in nature (Dobson et al. 2008, Gómez and Nichols 2013), disease patterns are extremely heterogeneous in space and time. In a context of global land-use change, the study of host-parasite relationships has recently caught the attention of a growing number of researchers due to this central perspective in ecology that considers disease processes in a dynamic, spatial context.

The likelihood of transmission by host-host and host-vector interactions is dependent on biotic and abiotic factors embedded within ecological systems that vary spatially in the environment (Mollison 1977, Bradley and Altizer 2007, Plantegenest et al. 2007, Meentemeyer et al. 2012). Hosts, vectors, or infective parasitic stages are more likely to interact if they are closer together in space (Tobler 1970, Mollison 1977, Keeling 1999), and overlapping environmental conditions that support the niche breadth of these species deeply influences host-vector-pathogen spatio-temporal niche overlap (Glass et al. 1995, Schweiger et al. 2005, Estrada-Peña et al. 2014). In nature, most host species are spatially distributed in patches that will influence the way in which disease spreads and how pathogens persist (Hess et al. 2002). Therefore, the spatial dependence of host-parasite ecology is a fundamental property of disease dynamics in natural populations.
Spatial dependence of host-parasite interactions only became explicitly incorporated into disease ecology modeling approaches at the end of the 20th century (May and Anderson 1984, Dwyer and Elkinton 1995). Many standard models of host-parasite interactions do not include space explicitly (Anderson and May 1978, Anderson and May 1979). In many cases, the implicit assumption for both density-dependent transmission (transmission dependent on contacts by density of host population) and frequency-dependent transmission (transmission independent of host densities) is that each individual in the local population has equal probability of encountering every other individual (McCallum et al. 2001, Hess et al. 2002). Therefore, a major tenet of ecology and epidemiology disciplines is to quantify and explain when disease systems deviate from this assumption of spatial homogeneity (McCallum 2008). Mollison (1977) investigated these deviations by quantifying analogous contact and dispersal processes between ecological and epidemic systems, and later extended this work to examine the spatial dependence of invasive spread (Mollison et al. 1986).

1.2 Landscape spatial heterogeneity and disease dynamics

1.2.1 Global change and the emergence and re-emergence of infectious diseases

Landscape spatial heterogeneity is defined as the spatial variability of biotic and abiotic conditions over a specified geographic area (Turner 1989, Pickett and Cadenasso 1995). While most landscapes are naturally heterogeneous (e.g., forest mosaic structure, island landscapes), anthropogenic modification of the environment can amplify the rate at which land cover is altered in terms of composition, proportion, and configuration. Consequentially, landscape spatial heterogeneity caused by human activities can have multi-scalar effects on the biotic and abiotic factors that determine disease persistence and diffusion. Emerging infectious diseases are a key threat in global public health (Daszak et al. 2000), livestock health (Caron et al. 2012), wildlife conservation (McCallum and Dobson 2002), and ecosystem functioning (Murray and Daszak 2013). Habitat fragmentation and loss caused by agricultural intensification, road construction, and urbanization, represent major pressures on these critical epidemiological factors (Patz et al. 2004). The effects of habitat fragmentation have been shown to cascade through communities by trophic interactions between species which can lead to complex changes in host and parasite community structure and function (Lambin et al. 2010, Hicks et al. 2015). In effect, habitat modification can have unpredictable consequences on the spatial variation and dependence of disease risk or incidence in natural populations (Jousimo et al. 2014). We should, therefore, expect that landscape processes occurring at these larger spatial scales play critical roles in disease dynamics at the local scale.

Environmental changes, including changes in climate and landscape characteristics, are predicted to have cascading effects on the extrinsic and intrinsic processes that influence infection heterogeneities in wild host populations (Anderson and May 1978, McCallum and Dobson 2002, McCallum 2008, Lambin et al. 2010) which, in turn, can cause shifts in parasite community composition (Kennedy and Bush 1994, Galli et al. 2001, Power and Mitchell 2004) and contact rates between hosts and vectors (Vander Wal et al. 2012, Jousimo et al. 2014). Generally, heterogeneous environments can have two different effects on disease spread and persistence. First, the rate of movement of hosts or vectors and connectivity varies through a heterogeneous landscape (Hicks et al. 2015). The functional capacity of landscape to affect host or vector dispersal has consequences for the movement of the epidemic front and whether
invasion can occur (Smith et al. 2002, Robinson et al. 2013). Next, population density and survival rates of hosts, vectors, and infective stages vary across a heterogeneous landscape (Brownstein et al. 2005). However, knowledge of interactions among landscape composition, landscape configuration and the local-scale demographic-dispersal processes within and between fragmented host populations remains limited (Holdenrieder et al. 2004, Ostfeld et al. 2005).

1.2.2 Landscape composition and disease processes

Host-pathogen dynamics are usually assumed to be regulated by density-dependent processes where transmission rates increase with host density (Anderson and May 1978, Arneberg et al. 1998, McCallum et al. 2001). Indeed, shifts in landscape structure by habitat fragmentation and loss often results in higher population densities and, theoretically, greater likelihood for transmission between individuals (Roland and Taylor 1997, Mbora and McPeek 2009). Infectious disease transmission requires contact between infectious hosts or infective stages and susceptible individuals, and because landscape affects population density and the movement of hosts, vectors, or transmission stages, landscape change will intuitively affect disease dynamics. A key limitation to studies of how disease emergence is driven by land-use composition is our significant lack of knowledge of the diversity of parasites present in wildlife in a region, of the ecology of these parasites, and their impact on different hosts (including human hosts) (Morse et al. 2012, Murray and Daszak 2013).

Compositional shifts in habitat quality can cause shifts in the structuring processes of both host and parasite communities (McCallum 2008, Lafferty 2009). The niche breadth of hosts, vectors, and parasites is dependent on various environmental factors (i.e., climatic regimes, vegetation types, refugia, etc.) that allow for these species to persist in a particular location (Estrada-Peña et al. 2014). Hence shifts in landscape composition and environmental heterogeneity can alter transmission dynamics within and between populations. In particular, resource provisioning of abundant food resources often results in sustained contact between host species (Wright and Gompper 2005, Hartemink et al. 2014, Becker et al. 2015). Interspecific interactions can have profound effects on parasite transmission and the potential for pathogen spillover (Thompson 2013, Becker and Hall 2014). Urban habitat, for example, provides anthropogenic food sources offering spatially-clustered foraging sites with potential for disease

Compositional shifts in fragmented habitat may cause alterations in competitive abilities of native host species, allowing for the invasion and dominance of non-native hosts and, potentially, the parasites that infect those hosts (Holt 1984, Jules et al. 2014). More complex and fragmented landscapes are associated with more ecotones (i.e., transition areas between two adjacent ecosystems), which increase the likelihood of intra- and inter-specific contacts between host species (Despommier et al. 2006). Opportunistic, multi-host parasites that can infect multiple host species may thrive in habitat conditions where invasive host species dominate. Immunologically-naïve susceptible hosts residing in fragmented habitat may be vulnerable to infection by these invading generalist parasites. Consequentially, shifts in the relative diversity of host and parasite community composition may have demographic and trophic effects on ecosystem services (Lafferty et al. 2008, Dunne et al. 2013).

Similarly, biodiversity losses by habitat fragmentation and loss may alter the host community competence in maintaining parasites (Keesing et al. 2006). Smaller degraded habitat patches may select for smaller-bodied, generalist hosts that persist well and are highly competent in infecting susceptible reservoirs or other hosts (Allan et al. 2003, LoGiudice et al. 2003b, Brownstein et al. 2005). These processes of biotic homogenization, whereby native species are replaced by dominant, non-native generalists, is often caused by landscape-scale modification of habitat (McKinney 2006). Specifically, the proportion of specialists to generalist hosts relative to the proportion of specialist versus generalist parasites will have critical implications on the persistence and amplification of disease reservoirs in natural environments. Depending on the magnitude of environmental disturbance, biotic homogenization processes are therefore hypothesized to have a vital role in the community ecology that underlies infectious disease dynamics in modified landscapes.

Many parasite species have adapted developmental stages off the host, either as infective stages developing in the free-living environment or by persisting in vertebrate intermediate hosts such as rodents or amphibians (Bush et al. 1997, Pietrock and Marcogliese 2003). As these developmental strategies are so tightly linked with particular environmental conditions, numerous studies have aimed to understand associations between land cover and disease risk,
focusing on critical habitat types for vectors or reservoir hosts to maintain parasites in particular environments (Glass et al. 1995, Lambin et al. 2010, Messier et al. 2015). For example, feline tularemia was observed at higher prevalence in grassland habitat adjacent to residential areas than those with low-grassland cover (Raghavan et al. 2013) and habitat defined by rocky outcrops and high leaf litter were linked to greater likelihood of malaria infection in fence lizards (Eisen and Wright 2001). Also, rodent reservoirs of the zoonotic Junin virus in Argentina were more likely to live in vegetation growing beside roads, perhaps indirectly causing an increase in the geographic range of the disease (Mills et al. 1992). Therefore, the sustained transmission and spread of environmentally-mediated parasites is likely governed by landscape composition factors that affect the density and dispersal of parasites, disease vectors, and intermediate hosts (Park 2012).

1.2.3 Landscape configuration and disease processes

Whether a pathogen can invade a population of fully susceptible hosts is a central question in epidemiology yet animal movements across the landscape play a critical role in the ecology of wildlife infectious diseases (Fortin et al. 2010, Robinson et al. 2013). Thus, the spatial distribution of hosts and parasites, and the connectivity between host populations, can have significant effects on pathogen transmission and disease prevalence (Ostfeld et al. 2005). Functional connectivity, defined as the ability of the landscape to impede or facilitate organismal movement (Bélisle 2005), is likely altered by landscape-scale modification of habitat. Indeed, connectivity may be beneficial for parasites whereby access to a greater number of subpopulations of hosts ensures survival and reproduction of parasitic species. Small or isolated habitat patches may influence extinction probability of a parasite if susceptible host populations are too distant or few to maintain infection (Heard et al. 2015). Host availability for parasites increases with increasing density of host networks, so short- and long-distance dispersal events are critical in the gradual or rapid spread of parasites across landscapes, respectively. Therefore, the ability of animal hosts to traverse at short or long distances between spatially-heterogeneous landscapes will depend on the degree of spatial separation between a pair of patches, the resistance of the inter-patch environment, and the biological ability of the host to disperse a sufficient distance to arrive successfully in a destination patch. Indirectly, these biotic and abiotic factors are also expected to impact the invasion potential of disease vectors or parasites residing in or on moving hosts.
Landscapes consist of heterogeneous patches or variably hostile habitats which must be navigated by dispersing hosts or vectors for pathogens to persist (Reisen 2010). Abrupt transitions in landscape such as highly-resistant surfaces, mountain features or rivers may delimit or facilitate both host and, indirectly, vector or pathogen dispersal potential. For example, while rivers likely impeded the spread of rabies in raccoon population in Connecticut, disease spread may actually be faster alongside rivers in other areas where hosts use riparian habitat for movement (Smith et al. 2002). Previous epidemiological studies have suggested that increasing habitat connectivity and host movements in metapopulations usually facilitate disease transmission, allowing a pathogen to successfully invade a metapopulation (Huang et al. 2015) and increase the prevalence and incidence of endemic diseases in metapopulations. Conversely, spatially-clustered habitat favouring frequent migration could indirectly facilitate transmission and, in effect, create increased risks for pathogen extinction within highly connected metapopulations. Despite increasing evidence of the role of landscape configuration on host, pathogen, and vector spread dynamics, there remains uncertainty about the role of connectivity on the structuring processes that underlie host-parasite interactions and disease spread processes.

1.2.4 Urbanization and disease processes

Understanding how cities affect disease ecology is a current frontier in disease ecology. Landscape spatial heterogeneity caused by urbanization is hypothesized to influence the ecology of infectious disease dynamics within and between natural populations (Bradley and Altizer 2007, Brearley et al. 2013, Giraudeau et al. 2014, Mackenstedt et al. 2015). Rapid urbanization over the last few hundred years has dramatically altered natural habitat structure, ecosystem functioning, and biodiversity (McKinney 2006, Luck and Smallbone 2011, Aronson et al. 2014). Accordingly, there has been much interest to understand finer-scale effects of cities on the ecology, behaviour, and, more recently, the ecology of infectious disease of natural communities.

How cities structure the ecology of host-parasite interactions remains an elusive yet critical problem in contemporary ecology. Recently, studies have shown that the invasion and persistence of resilient, synanthropic species causes shifts in the densities and interactions between hosts, potentially advancing the frequency of inter-specific transmission of parasitic infections and the likelihood of intra-specific, cross-species transmission (Wright and Gompper 2005, Lehrer et al. 2010). Host traits that allow host-species persistence in competitive, degraded
environments may persist due to adapted or humoral immunity responses (Schmid-Hempel 2008, Rohr et al. 2010). Consequentially, urban wildlife populations may act as disease reservoirs by maintaining and transmitting pathogens to conspecific wildlife populations or potentially to human populations (Haydon et al. 2002, Havel et al. 2005, Viana et al. 2014). Similarly, multi-host generalist parasites may be indirectly favoured in more-urbanized environments where inter- and intra-species contact rates are expected to be more frequent than rural or pristine environments.

Infection dynamics can change across a gradient of habitat in some cases leading to increased prevalence in urban or suburban environments (Daszak et al. 2000, Meentemeyer et al. 2012). Yet, there is a paucity of research investigating the mechanisms driving host-parasite interactions in urbanized environments despite the critical importance of urban wildlife parasitism on biodiversity patterns, infectious disease ecology, and potentially for public health concerns (Knudsen and Slooff 1992, Frank et al. 1998, Bradley and Altizer 2007, Mackenstedt et al. 2015). Further, disease ecology has few studies investigating the role of human-modified landscapes in the structuring processes of multi-host multi-parasite communities (Caron et al. 2012, Brearley et al. 2013). Understanding how cities affect disease ecology is a critical frontier in disease ecology. Landscape spatial heterogeneity caused by urbanization is hypothesized to influence the ecology of infectious disease dynamics within and between natural populations (Bradley and Altizer 2007, Brearley et al. 2013, Giraudeau et al. 2014, Mackenstedt et al. 2015). Rapid urbanization over the last few hundred years has dramatically altered natural habitat structure, ecosystem functioning, and biodiversity (McKinney 2006, Luck and Smallbone 2011, Aronson et al. 2014). Accordingly, there has been much interest to understand finer-scale effects of cities on the ecology, behaviour, and, more recently, the ecology of infectious disease of natural communities (Bradley and Altizer 2007).

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Rohr et al. 2010). Consequentially, synanthropic urban wildlife populations may persist as disease reservoirs by maintaining and transmitting parasites to conspecific wildlife populations or potentially to human populations (Haydon et al. 2002, Havel et al. 2005, Viana et al. 2014). Similarly, multi-host generalist parasites may be indirectly favoured in more-urbanized environments where inter- and intra-species contact rates are expected to be more frequent than rural or pristine environments.

1.2.5 Landscape epidemiology: a burgeoning discipline

Despite a growing interest in the relationship between landscape and disease ecology, the mechanisms that enable long-term disease persistence and likelihood of spread within and between wild communities remain elusive. Landscape epidemiology represents a nascent, contemporary research domain to provide insights into these complex linkages between large-scale environmental change and local-scale disease transmission systems. Landscape epidemiology is the study of these dynamic interactions between landscape composition and configuration with population-level epidemiological processes in natural populations (Meade 1988, Plantegenest et al. 2007, Reisen 2010, Meentemeyer et al. 2012). Its origins lie in medical geographer Evgeny N. Pavlovsky who proposed the concept of disease foci (nidality) of infections in relation to geographic or landscape features (Pavlovsky et al. 1966). The field is inherently ecological as modeling and empirical approaches aim to quantify the biotic and abiotic factors influencing spatial interactions between susceptible and infected individuals over multiple spatial and temporal scales (Lambin et al. 2010). With significant technological advances in remote sensing, geospatial analysis, and computational modeling, we can now address questions that were limited in 20th century disease ecology: how does landscape structure affect biogeographic patterns of disease? What ecological mechanisms of disease transmission are most affected by habitat fragmentation? How does landscape spatial heterogeneity affect host and parasite community composition? Landscape epidemiology overlaps in important ways with many ideas in community ecology and biogeography in that most ecological communities are connected by dispersal to regional pools of species including hosts, parasites, and pathogens (Poulin 2003, Polis et al. 2004).
1.2.6 Knowledge gaps and research needs

The dependence of infectious disease processes on their specific spatial and ecological context has been receiving considerable recent attention. Yet, studies focusing on the ways in which geographical and environmental factors affect host-parasite dynamics are still limited. While a significant body of the literature has focused on single-host single-parasite systems under controlled conditions, there is a need to understand the ecological factors related to widespread multi-host pathogens and systems involving multiple host-parasite interactions. Generalist, multi-host parasites represent significant drivers of pathogen spillover to other wildlife or domestic species and therefore represent public health threats.

Another major hypothesis of ecology is the argument that there is no single natural scale at which ecological phenomena should be studied (Keitt et al. 1997). Mechanisms of ecological processes occur on very different scales of space, time, and ecological organization (Levin 1992, Kennedy and Bush 1994). In plant and forest disease systems, there have been many recent advances in understanding at which scales particular demographic or dispersal processes occur to affect transmissibility of parasites within and between populations (Power and Mitchell 2004, Plantegenest et al. 2007, Meentemeyer et al. 2011, Seabloom et al. 2015). Yet, for vertebrate systems, empirical investigations are lacking. Despite significant headway in our understanding of essential properties of natural disease transmission systems, there is much room to explore these ecological interactions in an epidemiological context.

Finally, a prominent criticism of landscape-scale analyses is that observed patterns do not necessarily elucidate mechanistic processes. In disease systems, we are also faced with fundamental methodological issues: pattern-matching offers little intrinsic understanding of the processes that generated the observed patterns; and these methods cannot provide us measures of changing vector, pathogen, or parasite abundance. Therefore, there is a need to incorporate dynamic models to incorporate observed, empirical data with quantified shifts in landscape composition and configuration towards more biologically-realistic evaluation of dynamic disease transmission processes in spatially heterogeneous landscapes.

In community ecology, a central unsolved problem is the extent to which communities exhibit organized patterns and predictable structure (Chase 2003). In a context of parasite ecology, the field of disease ecology is faced with a similar dearth studies considering the spatial
and ecological contexts of pathogen persistence and invasion, leading to critical, unexplored questions (Kennedy and Bush 1994, McCallum 2008, Seabloom et al. 2015):

(a) What is the relative effect of landscape composition versus spatial configuration on the likelihood of host-parasite interactions?

(b) To what extent does the invasion of vectors or parasites depend on the functional connectivity of their hosts?

(c) How does heterogeneity in landscape composition influence the likelihood of parasitic exposure to host populations?

(d) Do landscapes affect community assembly processes of hosts differently than parasites?

(e) Can landscape be used as a proxy for host-parasite interaction processes for optimal disease management and early detection of pathogen invasion fronts?

As more than 75% of human diseases are of zoonotic (animal) origin (Taylor et al. 2001), the mechanisms by which cities may pose disease risks to domestic-animal or human populations is of significant ecological and epidemiological importance. As human-animal contacts are common, human zoonotic health risks are likely to increase due to climate and land-use changes (Daszak et al. 2000, Brearley et al. 2013, Murray and Daszak 2013), and with rapid urbanization trending on global scales, human-wildlife contacts are expected to increase in the future posing unpredictable public health risks. There is therefore a critical need to understand generalized ecological factors linking landscape spatial heterogeneity to the spatially-dependent factors affecting host-parasite community structure (generalists vs. specialists) and spread potential (short- versus long-distance parasite dispersal).

1.3 Thesis outline

The objective of this thesis is to better understand to what extent landscape spatial heterogeneity, defined by variation in landscape composition and spatial configuration, influences the ecology of host-parasite interactions. Many studies document observed patterns of spatial clustering of biological entities, but what structural landscape factors influence clumped dispersion patterns in
both hosts and parasites? So far, there have been very few studies investigating these effects in natural systems with observed, empirical data, and general patterns of response are not yet clear (Hicks et al. 2015). I approach my broad research objective in two perspectives: (a) from the perspective of a focal disease (Lyme disease), and (b) from the perspective of a focal landscape (urban environments). In Chapter 2, I investigate the mediating effects of landscape spatial heterogeneity on the ecology of Lyme disease, a vector-borne disease system, in an island region in southeastern Ontario, Canada. In Chapter 3, I examine a single-host, multi-parasite system to quantify the relationship between land cover (urban landscape spatial heterogeneity), dietary behaviour, and the likelihood of exposure to parasitism in a coyote population along an urbanization gradient in Calgary, Alberta, Canada. For Chapters 4 and 5, I examine the interactions between multiple wildlife hosts and parasite species by quantifying network interaction structure (chapter 4) and beta diversity patterns (chapter 5) over an urbanization gradient in southwestern Ontario, Canada.

1.3.1 Synthesis of chapters

In Chapter 2, I explore the mediating role of landscape spatial heterogeneity on the demographic-dispersal processes that influence vector-borne disease persistence and spread. Using Lyme disease as a focal vector-borne disease system, I seek to elucidate the relative dependence of host movement landscape features on Lyme disease spread, quantified by functional connectivity. For the spread of the disease, movement of the pathogen (Borrelia burgdorferi) is directly linked to the dispersal of its tick vector (Ixodes scapularis) and to the spatio-temporal dynamics of the wildlife host community upon which both parasites depend (Killilea et al. 2008). Yet, we still lack a synthetic empirical understanding of the functional interactions between landscape spatial heterogeneity and the ecological processes that determine Lyme disease expansion (Finch et al. 2014) such that in this chapter, I estimate, using a generalized demographic-dispersal connectivity model, the likelihood of tick and pathogen spread in a heterogeneous landscape using a simplified vertebrate host community: White-footed Mice, American Robins and White-tailed Deer (Madhav et al. 2004). The outcomes of this work provide evidence that (a) landscape spatial heterogeneity mediates tick spread differently than pathogen spread, relative to host dispersal processes; (b) intermediate stepping-stone habitat is critical for short- and long-distance tick invasion; and (c) pathogen invasion capacity is generally limited to within-island movements by mouse and robin dispersal. My theoretical contributions and novel network modeling tools
can be extended to various disease systems with applications to anticipating the impact of land-use changes on the epidemiology of vector-borne diseases.

In Chapter 3, I focus on urban landscapes and examine the relationships between urbanization, habitat type, and dietary behaviour with Coyote (*Canis latrans*) parasitism structure. In this chapter, I ask: what land-cover, dietary, and spatial factors explain variation in Coyote gastrointestinal parasitism? While Coyote parasitism surveys have been performed in the region (Catalano et al. 2012, Liccioli et al. 2012) and throughout North America (Holmes and Podesta 1968, Samuel et al. 1978), few have predicted parasite structure using urbanization factors and dietary behaviour (Atwood et al. 2004). However, as a facultative carnivore, understanding how landscape spatial heterogeneity affects exposure to parasitism may shed light on the cascading effects of cities on carnivore disease dynamics and associated zoonotic risks (Thompson 2013). Multivariate analyses are utilized whereby variation in parasite and diet components collected from fecal samples in Calgary (Alberta) are predicted using multiple land-cover, connectivity, and dietary predictors. My results demonstrate that developed habitat, grassland cover, and dietary choice interact to possibly influence the exposure of Coyote hosts to enteric parasites. This work is novel as it includes the use of both parasite and dietary data of a single host species to elucidate relationships between landscape and host-parasite interactions and pioneers future investigation of disease ecology for natural populations in anthropogenic landscapes.

For Chapter 4, I specifically explore the role of urban modified-habitat on patterns of host-parasite interactions. In this work, I examine how urban landscapes shape the structure of host-parasite communities. The role of urbanized environments on the structuring processes of natural communities and species interactions is a critical question for ecologists yet largely unknown (Shochat et al. 2006, Brearley et al. 2013). Despite widespread interest in the role of host and parasite community interactions on population dynamics and ecosystem functioning (Lafferty et al. 2008), no study has addressed the effect of urban land cover to influence host-and parasite community assembly processes. I use bipartite network models to evaluate the role of urbanization on the architecture of host-parasite interactions in southwestern Ontario (Canada). My work demonstrates significant differences in asymmetry between urban and natural gradients, and between mammalian and avian functional groups. Abundant hosts such as pigeons and doves as well as small rodents and moles maintained the greatest parasitism in
suburban and urban sites. I provide evidence that urban-modified landscapes can shape host-parasite interaction structure, likely caused by the selection of generalist reservoir host species in highly-urbanized habitat. My work underscores the role of cities in altering the ecology of hosts, parasites, and infectious disease dynamics. These novel results contribute to our knowledge of urbanization and parasite community assembly in wildlife populations but carries possible consequences for zoonotic risks posed to human dwellers.

Finally, in Chapter 5, I investigate how urban landscape spatial heterogeneity mediates the beta diversity of host and parasite communities across an urbanization gradient. Extending from Chapter 4, urban environments are expected to cause shifts in the composition of natural communities of species, often observed as biotic homogenization (McKinney 2006, Devictor et al. 2008). Similar shifts in biodiversity are expected for host and parasite species, but this question has only been addressed empirically through single-host, single-parasite systems (Evans et al. 2009, Martin and Boruta 2013, Giraudeau et al. 2014). Thus, beta diversity can be used to describe turnover in host and parasite species composition across a biogeographic gradient (Legendre and Cáceres 2013) to elucidate relationships between urban land-cover variation and host-parasite community composition. In this work, I quantify the relative contribution of individual host and parasite species to beta diversity (SCBD) as compared to local, spatially-explicit site contributions to beta diversity of host-parasite systems (LCBD). I demonstrate that three host functional groups (pigeons and doves, small rodents and moles, and small carnivores) contributed to beta diversity well above the mean of the total 12 wildlife functional groups. For parasites, viral and ectoparasitic species had the highest SCBD index. My results demonstrate that dominant, generalist host species are more related to overall community diversity among sites; whereas directly-transmitted parasites affiliated with high density wildlife population conditions. This work supports preexisting hypotheses that urban development affects community assembly processes of host-parasite interactions and provides implications for ecological mechanisms that control disease dynamics in cities.

I synthesize my chapters with a concluding Chapter 6 by embedding and contextualizing my PhD thesis work in the history, current state, and future trajectory of the field of disease ecology and landscape epidemiology.
Chapter 2

2 Landscape Spatial Heterogeneity Mediates the Potential Spread of Lyme disease

2.1 Abstract

Dispersal processes are central to the epidemic spread of pathogens in natural populations and habitat structure is hypothesized to influence the likelihood of host, vector, and pathogen movement. In the case of Lyme disease, movement of the pathogen (*Borrelia burgdorferi*) is directly linked to the dispersal of its tick vector (*Ixodes scapularis*), as well as to the spatio-temporal dynamics of the wildlife host community upon which both parasites depend. Yet, we still lack a synthetic empirical understanding of the functional interactions between landscape spatial heterogeneity and the ecological processes that determine Lyme disease expansion. Using a generalized network connectivity model, I estimate the probability of *I. scapularis* and *B. burgdorferi* spread in a heterogeneous landscape using a simplified vertebrate host community: White-footed Mice, American Robins and White-tailed Deer. I parameterized the connectivity model using observed *B. burgdorferi* prevalence in tick and host populations sampled from a Lyme-endemic island landscape in Thousand Islands National Park (Ontario, Canada) where dispersal costs differ for mammalian vs. avian hosts (swimming versus flying, respectively). I provide evidence that (a) landscape spatial heterogeneity mediates tick spread differently than pathogen spread, relative to host dispersal processes; (b) intermediate stepping-stone habitat is critical for short- and long-distance tick invasion; and (c) pathogen invasion capacity is generally limited to within-island movements by mouse and robin dispersal. My theoretical contributions and use of novel network-modeling tools can be extended to various disease systems with applications to anticipating the impact of land-use changes on the epidemiology of vector-borne diseases.

2.2 Introduction

A fundamental prediction in disease ecology is that pathogen invasion depends on the dispersal ability of infected hosts or vectors (May and Anderson 1984, Dwyer and Elkinton 1995, Wang 2004, Altizer et al. 2006). The abundance of migrants capable of dispersing, the proportion of infected migrants, and the dispersal success of infected migrants in naïve habitat (Mollison 1977,
Beasley and Rhodes 2010) are shaped by the ecological conditions within and between host habitat. Yet, how landscape composition and configuration mediates the connectivity of hosts, and the corresponding invasive probability of pathogens and their vectors throughout a given landscape, remains complex and unclear (Reisen 2010). Quantifying the mediating role of the landscape on motile hosts and corresponding vector-borne pathogen spread (Estrada-Peña 2003, Tack et al. 2014) is critical in predicting the epidemic spread potential of vector-borne diseases in current and future human-modified environments. Here, I investigate the role of landscape spatial heterogeneity on the spread of the most common tick vector of Lyme disease in eastern North America, *Ixodes scapularis*, and of the disease-causative agent of Lyme disease, *Borrelia burgdorferi*, using a novel generalized connectivity model that considers both demography and dispersal ability of the host, tick, and pathogen.

Processes facilitating or preventing biological invasion act at different stages of the invasion process, beginning with the introduction of the organism to naïve habitat, the establishment of the organism within the habitat, and the eventual dispersal by successive generations of invasive propagules (Mollison et al. 1986, With 2002). Short-distance and long-distance dispersal of propagules are critical processes to invasion success and rate of disease spread (Mollison et al. 1986, Lavorel et al. 1995). With vector-borne diseases, the introduction, establishment, and eventual dispersal of the pathogen are expected to be linked to the population dynamics and dispersal ability of the vector and hosts (Ostfeld et al. 2006, Hartemink et al. 2009), presenting complex modeling challenges. These complexities are exemplified in the case of Lyme disease where the introduction of the spirochete pathogen, *Borrelia burgdorferi*, depends on the dispersal success of an infected host or tick vector (e.g. *Ixodes scapularis*). Moreover, the dispersal and establishment of the tick in a habitat patch is contingent on the dispersal of vertebrate hosts on which they attach during blood-feeding events. Establishment of ticks cannot occur in the absence of the vertebrate host; and the pathogen cannot persist in the absence of either organism. The dispersal capabilities of the vertebrates that are parasitized by ticks differ considerably (Watts et al. 2009), and the range of short- and long-distance dispersal capabilities of burdened hosts are expected to be mediated by landscape spatial heterogeneity (Lavorel et al. 1995, Estrada-Peña et al. 2014). It is therefore of interest to model the spread potential of both the *Ixodes* tick vector and the *Borrelia* spirochete pathogen as interactions.
between landscape and host-population dynamics may affect the diffusion of the vector and pathogen differently.

My goal was to investigate the mediating role of landscape spatial heterogeneity on Lyme disease dispersal processes. I compared the effect of host functional connectivity, i.e., the ability of landscape spatial heterogeneity to impede or facilitate organismal movement (Bélisle 2005, Baguette and Van Dyck 2007), on the spread of (a) ticks, versus (b) the pathogen. My study is defined by two objectives: (1) quantifying potential extent of tick versus pathogen spread by host dispersal; and (2) quantifying landscape structural characteristics that contribute to relative potential tick/pathogen spread. For objective (1), I calculated the percentage of the total amount of habitat in the patch network that could be potentially reached by a source tick population located randomly in a single patch (hereafter amount of reachable habitat, ARH). For objective (2), I quantified landscape structural characteristics therefore most influential on tick versus pathogen spread, as quantified by patch connectivity metrics.

I predicted that (a) small-bodied mammalian hosts limit the dispersal capacity of ticks and the pathogen because small mammals, though highly-competent reservoirs, are often dispersal-limited species; (b) that avian hosts facilitate short-distance dispersal of ticks and the pathogen because they are not limited by hostile inter-patch matrix; and (c) large-bodied mammalian hosts facilitated long-distance dispersal capacity of ticks but not the pathogen because large mammals can disperse large distances but have low reservoir competence. I bridged demography with large-scale network dispersal accounting for both direct and indirect pathways using the generalized connectivity model. This integration helps to elucidate the interrelated consequences of stepping-stone habitat for host movement and disease establishment, which have not yet been investigated. This hybridized demographic-dispersal modeling approach could be applied to a variety of directly-transmitted or vector-borne disease systems in plant and animal communities for critical theoretical and public health priorities.

2.3 Methods

2.3.1 Study Region

I modeled the relative spread potential of the Lyme disease tick vector, *Ixodes scapularis* (referred to herein as ticks) and the Lyme disease-causative agent, *Borrelia burgdorferi*, in a
spatially heterogeneous patch network of the St. Lawrence River in Thousand Islands National Park (Canada) (44.45320°N, 75.86085°W) (Fig. 2.8.1) composed of discrete, forested islands (referred to herein as habitat patches) separated by water. This study landscape comprised 12 sampled and 446 unsampled patches between the Canadian and United States riverbanks of the river. Sampling design and field methods for observed data are described in detail in Werden et al. (2014) but briefly explained in Appendix 2.9.1.

This binary landscape (patch – water) provided a unique opportunity to isolate host dispersal-related demographic processes from relatively unknown effects of spatial arrangement of landscape quality between patches that may facilitate or impede host movement to different degrees (Zeller et al. 2012). Water serves as the prominent barrier to movement between patches and should have a different effect on the dispersal of mammalian hosts versus avian hosts. Southeastern Canadian regions represent a northern range front of *Borrelia* expansion (Leighton et al. 2012), and have been followed by a recent onset of Canadian Lyme disease cases.

### 2.3.2 Model host community

I focused on a host community of three critical species: White-footed Mice (*Peromyscus leucopus*), American Robin (*Turdus migratorius*), and White-tailed Deer (*Odocoileus virginianus*) (LoGiudice et al. 2003b, Madhav et al. 2004, Schwanz et al. 2011) (Table 2.7.1). Modeling efforts on Lyme disease dynamics have utilized this simplified vertebrate host community (Madhav et al. 2004) and thus different hosts are expected to have differential roles in facilitating *B. burgdorferi* spread in heterogeneous landscapes. White-footed Mice are a vital host in maintaining tick populations (Allan et al. 2003) and are a critical reservoir host for *Borrelia burgdorferi* acting to re-infect unexposed tick populations (Goodwin et al. 2001). American Robins are exemplary ground-dwelling host species that feed a large number of immature ticks (Anderson and Magnarelli 1984, Richter et al. 2000). American Robins may have a role in maintaining the *Borrelia* pathogen (Richter et al. 2000), and have some capacity to infect feeding tick populations (Ogden et al. 2008, Newman et al. 2015). Additionally, infected or uninfected robins may have an important role in both tick and pathogen spread because robins could simply fly between patches where mammals require swimming. White-tailed Deer have long been identified as a critical, definitive host of *I. scapularis* and support the maintenance
(Ostfeld et al. 2006, Werden et al. 2014) and regional dispersal (Bouchard et al. 2013) of tick populations.

Mice and deer have been observed swimming among islands (Schemnitz 1975, Sullivan 1977) due to natal dispersal behaviour and density-dependent foraging behaviour, respectively, whereas robins simply fly across island gaps (Marzluff et al. 2007). Mice are expected to be less capable of traversing large distances of water between island patches than deer. I assumed White-tailed Deer alone cannot spread *Borrelia* (Stafford III et al. 2003) as they are incompetent hosts in re-infecting tick populations with *Borrelia* spp. (Telford 3rd et al. 1988, Kurtenbach et al. 2002), they can clear *Borrelia* infection in some infected ticks (Lacombe et al. 1993), and the spread of infected adult female ticks by deer only propagates the tick population as I assumed no transovarial transmission (Rollend et al. 2013).

### 2.3.3 Generalized network connectivity model

I applied a generalized connectivity model (Saura et al. 2014) to estimate tick versus pathogen spread. This model accounts for (a) $N$: the number of ticks (infected or uninfected) that are available for dispersal (by attaching to hosts) in an occupied source patch; (b) $k$: the number of ticks (infected or uninfected) required to reach a vacant destination patch in order to establish a tick population in that patch; (c) the relative likelihood of long-distance dispersal events controlled by a parameter ($\beta$) in the dispersal kernel; and (d) the potential role of stepping stones to enhance spread via species dispersal and reproduction in intermediate patches (Saura et al. 2014). The generalized model results provided (1) probabilities of establishment of individuals through a single direct-dispersal event from source patch $i$ to destination patch $j$, without using other intermediate patches ($p_{ij}$); and (2) maximum product probabilities, accounting for stepping-stone intermediate habitat that may facilitate dispersal success between patches $i$ and $j$ ($p^*_{ij}$). I applied this model to compare the landscape- and patch-level probabilities of vertebrate host dispersal on the potential spread of tick vectors versus the pathogen.

Both $N$ and $k$ values were parameterized by a combination of observed host, tick, and pathogen demographic data and published estimates from the literature (Table 2.7.2), a simple method to capture the dynamics of the inter-dependent role of host dispersal on tick versus pathogen spread in a spatially-explicit model. For *Ixodes* spread, $N$ values related to the number of uninfected dispersing ticks that can be transported by hosts from a source patch to a
destination patch. These values were limited by (1) the relative abundance of ticks and hosts and (2) the average burden of ticks per host. I calculated $N$ values per host (Table 2.7.2) dependent on these demographic factors: for a given host, $N$ was equal to the minimum value of (1) the number of hosts in a patch (because there cannot be more dispersing hosts than that number); and (2) the ratio between the potential number of ticks (uninfected for the tick models, $t_i$; or infected, for the pathogen models, $t_{\phi \cdot inf}$) and the average number of uninfected or infected ticks that burden a given host ($B$ or $B_{inf}$, respectively). For *Ixodes* spread, the number of dispersal events was expected to be limited by the number of ticks or by the number of available hosts. $N$ values were calculated accordingly per host for all patches and used in consecutive model runs given constant per host $k$ values. In sum, tick-spread was modeled in three scenarios: dispersal of burdened mice, robins, and deer.

Considering pathogen spread, $N$ values depended on the prevalence of *B. burgdorferi* in the respective tick or host populations. I therefore parameterized additional $N$ values to represent the potential number of burdened hosts (mice and robins) infected hosts mice or robins as a mode of pathogen spread, calculated as (1) the potential number of infected mice or robins per patch available for dispersal ($N_{m \cdot inf}$, $N_{r \cdot inf}$), and (2) corresponding potential number of ticks that burden dispersing, infected mice or robins ($N_{t \cdot inf}$). Pathogen spread, the number of dispersal events was expected to be limited by the number of infected ticks, the number of available hosts, or the number of infected available hosts. In sum, pathogen spread was modeled in three paired scenarios: uninfected mice and robins burdened with infected ticks; infected mice and robins burdened with ticks; and infected mice and robins unburdened but dispersing the pathogen by infected-host movement alone.

Values of $k$ related to the minimum number of immigrant uninfected or infected ticks required for tick or pathogen population establishment in a destination patch, respectively. Larval- and nymphal-stage ixodid ticks introduced by mice or robins will likely drop off hosts and molt in the destination patch, while adult-stage gravid females introduced by deer will drop and lay eggs (3000 per female (Fish 1993)) in a destination patch. For *Ixodes* spread, I assumed tick establishment by passive dispersal of biting ticks that burden moving mice, robins, and deer. I calculated tick-spread $k$ values in terms of dispersing hosts by dividing the minimum assumed number of ticks for establishment ($k_t$, $k_{it} = 2$) by the average burden of infected ticks per host (published data, Table 2.7.2). The $k$ establishment parameters were therefore expressed as the
minimum number of dispersing hosts required to establish a minimum number of uninfected or infected ticks in a destination patch for ticks and the pathogen, respectively. Therefore, \( k \) values were parameterized in terms of the minimum number of burdened hosts (\( k_m, k_r, k_d \), for mice, robins, and deer, respectively) required to disperse at least two uninfected or infected ticks (Table 2.7.1).

Similarly, for pathogen spread, I parameterized pathogen establishment in terms of the minimum number of infected, burdened hosts (\( k_{im}, k_{ir} \), for mice and robins, respectively) required to disperse at least two infected ticks (Table 2.7.1), or in terms of the minimum number of infected hosts to establish a destination patch and infect ticks that feed on arriving hosts. All \( k \) values were kept constant per host and per establishment scenario, though additional \( k \) scenarios were run to test the sensitivity of minimum establishment on spread (\( k, k_{it} = 2; k, k_{it} = 10 \)) (Appendices 2.9.2.1 and 2.9.2.2).

I assumed a homogeneous distribution of tick parasitism among all model hosts and therefore equal probability of average tick burden for individuals of the same host species. Additionally, I assumed stable equilibria for all host populations (equal birth and death rates) and no interspecific interactions such as predation or competition. For ticks, I assumed that if at least two ticks are present on a host 100% of females successfully find a mate on-host if they have not already mated off-host in the source patch. Equal sex ratios are assumed for ticks and I assumed on-host blood feeding periods longer than the average duration of a single dispersal event for all hosts for successful introduction of ticks in a destination patch.

### 2.3.4 Observed and spatial data

Demographic parameters (abundance and infection prevalence) were calculated per patch in the network using values estimated from observed data sampled from the 12 sampled island patches in the landscape. Unsampled sites consisted of (1) 399 island patches, with minimum area of the smallest sampled island patches (1.7 ha); and (2) 67 mainland bank patches (US bank: 22 patches; Canadian bank: 45 patches). Unsampled patches in the region were included if they fell within the average maximum extent of deer movement (16 km) during the summer and fall seasons from the sampled islands (Porter et al. 2004). Sampled and unsampled patches were represented as polygons derived from CanMap RouteLogistics Ontario v2013.3 (DMTI 2013) using ArcGIS 10.2 (ESRI 2011). Mainland riverbank patches were represented as polygons,
designed as buffered contact points closest to patches in the river with a minimum area of the smallest sampled patch (1.7 ha). Overlapping mainland polygons were dissolved as continuous riverbank habitat.

I used deciduous forest habitat area (DMTI 2013) of each patch as a node weight in the network model. Deciduous forest provides suitable habitat and local conditions to promote growth and persistence of White-footed Mice populations (Nupp and Swihart 1996); ground-dwelling birds such as American Robins (Holmes and Sherry 2001); White-tailed Deer (Augustine and Jordan 1998); and the tick vector, *I. scapularis*. I estimated tick abundance in unsampled patches using a linear regressions between deciduous forest and total tick abundance per sampled patch ($R^2 = 0.774$). The average observed *B. burgdorferi* prevalence of the tick population per sampled patch (23.0%) was multiplied by the total estimated tick abundance for a value of total infected ticks per unsampled patch (Table 2.7.2). Observed mouse abundance values and infection prevalence (26.60%) were also estimated from sampled patches using this method. I used published estimates of robin and deer densities and used average dispersal behaviours for all host species. Because this landscape is made up of island patches separated by water, I used recorded swimming distances as single dispersal events among islands for mice (38.4m) and deer (2600m) and used robin dispersal distance estimates of seasonal breeding dispersal behaviours (135m) (Table 2.7.2).

### 2.3.5 Landscape metrics

Using the generalized connectivity model, I estimated the potential amount of reachable habitat (ARH) as the expected percentage of the total habitat area in the network that could be reached by a source population located randomly in a single patch within the habitat distribution. Potential ARH was given by an adapted version of probability of connectivity metric (Saura and Pascual-Hortal 2007) calculated as $PC_{gen}$, the overall generalized connectivity metric value for the entire landscape (Saura et al. 2014). I further partitioned the $PC_{gen}$ calculations as the percentage of total habitat resources in the landscape that could be reached per host: (a) amount of reachable habitat within the habitat patches ($PC_{intra}$ - intrapatch connectivity); (b) direct movement ($PC_{direct}$ - direct dispersal connectivity, not accounting for stepping stones); and (c) stepping stones ($PC_{step}$ - indirect dispersal connectivity, accounting for species reproduction and
further dispersal in intermediate patches acting as stepping stones across generations) (Saura et al. 2014). All ARH outputs were compared between the tick and pathogen models.

### 2.4 Results

For objective (1), I found discrepancy in the capacity of landscape spatial structure to mediate the potential amount of reachable habitat (ARH) for ticks than for the pathogen. ARH for tick and pathogen spread capacity does not necessarily increase linearly with increasing dispersal distance of hosts (Fig. 2.8.3), as the extent of tick spread by mice (18.0%) is relatively equal to that of robins (18.7%). Lower dispersal distance of mice compared to robins is compensated by the larger rodent population size, which yields larger quantities of potential tick-carrying dispersal events. Likewise, robins persist at lower densities than mice and have lower relative tick burdens than mammals in general. Deer support the greatest tick spread extent (ARH=82.0%) across the breadth of the patch network, while burdened, infected mice support the highest pathogen habitat reachability (19.8%), even if largely confined to within-habitat spread (Fig. 2.8.5).

Pathogen spread by infected, burdened mice was greater than spread by infected, unburdened mice (14%) or uninfected, burdened mice (13%) (Fig. 2.8.3). Overall, pathogen spread was limited to mice and robin dispersal, largely regulated by the relative densities of host and tick populations in source patches (Table 2.7.3, Fig. 2.8.3). Probability of tick spread for mice and deer was dependent on dispersal ability of tick-infested hosts and the relative abundance of hosts and ticks available to disperse from source patches. For both ticks and the pathogen, ARH was highly variable, given (a) relative host-tick abundance in source patches; (b) average host burden; (c) host dispersal capability to destination patches; and (d) configuration of the patch network (described in detail below). Host-contribution to spread was not sensitive to increasing $k$ values (Appendix 2.9.2.1, 2.9.2.2).

For objective (2), I quantified the contribution of the landscape heterogeneity on tick versus pathogen spread using partitioned landscape-level connectivity fractions (Saura et al. 2014): ‘$PC_{intra}$’ refers to proportion of connectivity contributed by within-habitat spread; ‘$PC_{direct}$’ refers to contributions by direct movement between pairs of patches; and ‘$PC_{step}$’ refers to contributions of stepping stone habitat to facilitate connectivity not possible by direct movement alone. Mice short-distance dispersal ability limits tick spread: (i) about one third of
the total ARH is confined within patches \((PC_{\text{intra}})\) and (ii) direct dispersal to other patches can only happen if they are very close to the source patch which makes the availability of stepping stone patches crucial to allow for several short-distance dispersal events finally determining a much larger amount of reached habitat \((PC_{\text{step}}\) being the fraction with the largest contribution, as illustrated in Fig. 2.8.5a). Effective tick spread to new patches by robins is relatively unlikely \((PC_{\text{intra}}\) being the dominant fraction) due to low tick burden and population densities (potential number of dispersing individuals) for this host. While the possibility of concatenating several effective (tick- or pathogen-spreading) interpatch movement steps using stepping stones was almost negligible, there was some chance of direct spread by robins to some other patches \((PC_{\text{direct}})\) (Table 2.7.3).

Tick spread by deer was largely favoured by the presence of stepping stones allowing for pathways consisting of multiple concatenated movement steps \((PC_{\text{step}})\); if stepping stones were removed from the landscape, the spatial extent of tick spread by deer would considerably decrease (<30% ARH by direct movement alone). Infected burdened mice spread the pathogen within-habitat more effectively than uninfected burdened mice, infected unburdened mice, and robins (Fig. 2.8.4b, Appendix 2.9.2.2). Stepping-stone habitat facilitated potential pathogen spread by infected, burdened mice most critically. Overall, potential spread of ticks and the pathogen by all hosts was less likely to occur by direct movement between patches alone, though in some clustered arrangements of patches this structural factor might facilitate local spread for short-distance dispersing hosts such as robins (Figs. 2.8.4 and 2.8.5).

2.5 Discussion

2.5.1 Landscape spatial heterogeneity mediates disease spread

The role of forest fragmentation and habitat heterogeneity has been established as having a positive influence on human Lyme disease risk (Brownstein et al. 2005), largely caused by altered demographic processes that can dilute or amplify disease within habitat fragments (LoGiudice et al. 2003b, Simon et al. 2014). Indeed, habitat fragmentation is also expected to influence the dispersal of hosts, and their associated ecto- and endo-parasites, by inhibiting or facilitating host movement between spatially-explicit habitat patches (Estrada-Peña 2003, Baguette and Van Dyck 2007). I support this hypothesis by quantifying how habitat spatial arrangement influences host dispersal capabilities leading to different indirect effects on vector
versus pathogen spread. I provided evidence that landscape spatial heterogeneity mediates tick spread differently than pathogen spread, relative to host dispersal processes. Landscape spatial heterogeneity was exhibited to have an influential effect on individual hosts’ capacity to facilitate the local invasion dynamics of tick-borne pathogens, acting as a filter that amplifies or reduces the relative contribution of different host species to disease spread according to their ability to disperse vectors and pathogens across fragmented landscapes. My work further develops our knowledge of the dynamic interactions between landscape structure and host dispersal, a critical relationship influencing the ecological processes contributing to disease spread, and can extend to my understanding of the role of vector-borne disease dependent on host movement for spread.

2.5.2 Landscape spatial heterogeneity and within-patch disease processes

As predicted by the generalized network connectivity model, small-bodied hosts (mammalian and avian) were more likely to contribute to tick and pathogen spread within patches and, in some cases, between adjacent patches. Conversely, large-bodied hosts (White-tailed Deer) contribute to tick spread among patches over larger scales, facilitated by stepping stones. Yet, dispersal potential was largely dependent on relative abundances of hosts, tick vectors, and relative prevalence of Borrelia in these respective populations per source patch (calculated by \( N \) and \( k \) values). I expect small-bodied hosts limited by movement between patches remain within patches and act to maintain the persistence of the tick and pathogen populations within source patches. Within-patch environmental conditions, such as percent deciduous forest cover, may therefore be more or less favourable for the potential dispersal of the tick vector and the pathogen to neighbouring patches. For example, low host-community richness and high densities of mice – a highly competent reservoir – have been shown to amplify \( B. \) burgdorferi among host and tick populations (Van Buskirk and Ostfeld 1995, Keesing et al. 2006). If degraded patches support lower host species diversity, disease prevalence may amplify in the host and tick populations, and consequently the probability of pathogen spread from source to destination patches by larger-bodied hosts. Conversely, if pristine, suitable source patches support higher host diversity, this may reduce the probability that a tick will become infected with the disease. In my study I assume a simplified host community, but in natural conditions greater diversity and
abundance of source-patch hosts may dilute the prevalence of the pathogen in both tick and host populations, reducing dispersal potential of Lyme disease from a given source patch.

2.5.3 Landscape spatial heterogeneity and between-patch disease processes

In addition to within-habitat processes, landscape spatial heterogeneity was expected to influence between-patch dispersal processes inherent in disease spread. I quantified how patch spatial arrangement and intermediate patches have a variable effect on host species’ influence on tick and pathogen dispersal potential. I established that geographic separation between any given pair of patches in the network limits the spread of ticks and the pathogen by smaller-bodied hosts with modest dispersal ability yet higher burdens and infection prevalence. I further quantified that stepping stone patches are critical landscape structural components in facilitating pathogen invasion via burdened, infected mice, and tick invasion via deer hosts. Short-distance and long-distance dispersal of propagules are critical processes in invasion success and rate of disease diffusion (Mollison et al. 1986, Lavorel et al. 1995).

Habitat isolation can inhibit establishment of invasive species and disease agents (With 2002). Yet, my results suggest that stepping stone patches may be important the spread of Lyme disease over multiple scales as observed between stepping-stone contributions for both burdened mice (short distance dispersal) and burdened deer (long distance dispersal). Mouse spread is generally limited to within-habitat movements but pathogen spread over short distances between island patches by infected mice is facilitated by stepping-stone patches. Likewise, ticks could spread over long distances by females feeding on dispersing deer that traverse fragmented habitat via stepping-stones. Finally, intermediate habitat may facilitate spread of ticks and the pathogen between two given patches for other dispersal-limited hosts in the host community more than direct movement alone (Apte et al. 2000, Saura et al. 2014).

2.5.4 Limited contributions of songbirds to disease spread

I found that robins do not effectively contribute to tick nor pathogen long-distance invasion relative to mice and deer, largely confined to within-patch spread. While songbirds have a likely role in the regional-scale northward migration of infected ticks into and across Canadian regions (Ogden et al. 2008), American Robins that forage on the forest floor and encounter nymphal-stage ticks relate to small burdens relative to White-footed Mice, on average (Giardina et al.
2000). For American Robin hosts, spread of ticks and the pathogen are generally limited by lower population densities and tick burdens instead of distance between patches (Anderson and Magnarelli 1984). The reservoir competence of ground-dwelling birds has been debated in the literature (LoGiudice et al. 2003b, Ginsberg et al. 2005, Newman et al. 2015). I suggest that American Robins have a reduced capacity to re-infect ticks and disperse *Borrelia* to vacant patches relative to highly abundant and competent rodent hosts due to low relative tick burdens and low densities in source patches. For simplicity, I assumed the same reservoir competence for American Robins as White-footed Mice; yet, demographic and density-dependent factors seemed to confine tick and pathogen spread by robins to within habitat movements. Furthermore, because American Robins breed in this sampling region they likely limit their dispersal distances due to nest protection and fidelity (Haas 1995), corresponding to an unintuitive dispersal limitation for an avian host (Wheelright and Mauck 1998).

2.5.5 Applications to terrestrial landscapes

The model outcomes were founded on a binary landscape where water is the primary barrier for dispersal and related Lyme disease spread. However, mainland landscapes are variable in resistance to species movement and can limit or favour the dispersal success of hosts and relative establishment success of their associated feeding vectors depending on the suitability of the inter-patch resistance matrix. For example, rural landscapes, consisting of forest-agriculture land cover, dominate large regions of northeast North America over which Lyme disease has spread over the past 50 years (Glass et al. 1995, Ostfeld et al. 1995, Schulze et al. 2005). High amounts of forest edge in agricultural or and suburban landscapes are positively associated with Lyme disease exposure risks where mouse, deer, and tick habitat overlap with recreation areas or properties (Jackson et al. 2006). White-footed Mice and deer are more likely to disperse larger distances between forest patches in agricultural regions than between islands in less resistant inter-patch matrix such as agricultural fields which could indirectly facilitate the spread potential for both tick vectors and tick-borne pathogens.

Similar landscape epidemiological dynamics are expected in suburban regions dominated by residential land cover. Fragmented suburban habitat can support high densities of White-tailed Deer as they benefit from the presence of edge habitat (Brownstein et al. 2005). This type of landscape mosaic has been shown to support higher tick densities than homogenous forested
landscapes because of extreme reservoir host abundances for tick feeding and reproduction (Ostfeld et al. 1995). Additionally, suburban environments can support high densities of White-tailed Deer as they benefit from the presence of edge habitat (Frank et al. 1998, Brownstein et al. 2005). If the arrangement of suburban habitat supports intermediate habitat, then stepping stones may facilitate the short-distance spread of tick and pathogen populations among small patches by White-footed Mice, posing potential human risks for suburban dwellers.

2.6 Conclusions

Landscape epidemiology is a growing field that focuses on how landscape change alters infectious disease dynamics and emergence. Understanding the spatiotemporal patterns of vector-borne pathogen spread is crucial to implement targeted awareness campaigns to mitigate human risks and possible vaccination strategies. Taking a landscape-epidemiology approach, my results shed light on the ability of landscape spatial structure to influence vector-borne disease emergence caused by the invasion of infected arthropod vectors and hosts. As has been suggested for Lyme disease (Simon et al. 2014), the unprecedented recent emergence of West Nile Virus, malaria, and Chagas disease in temperate zones of North America are linked to the regional latitudinal climatic release of arthropod vectors and mobile hosts (Eisen and Wright 2001, Mackinnon and Marsh 2010, Gottdenker et al. 2011, Harrigan et al. 2014). Fragmented habitat may alter the prevalence of the pathogen in localized vector populations and related host communities leading to the emergence of vector-borne disease in previously unestablished regions.

Demographic-dispersal connectivity models are a frontier in the prediction of vector-borne disease emergence over multi-regional scales where widespread sampling would be unfeasible. I present additional novelty in the parameterization of tick-host encounter rates and relative burdens represented in terms of $N$ and $k$ values which act as proxies for a more biologically realistic approximation of functional connectivity. I establish the ecological role of stepping stones in disease spread. Modeling connectivity by key stepping stones or patch clusters may improve spatially-explicit targeted monitoring (Killilea et al. 2008) or host vaccination efforts (Richer et al. 2014, Beasley et al. 2015) to reduce current human risks of vector-borne infections. Because arthropod vectors are so strongly linked to temperature and precipitation regimes, the spatially-explicit incorporation of generalized connectivity and climate models...
provide a more process-based modeling approach for vector-borne disease spread and establishment in future climate and landscape scenarios.
### 2.7 Tables

Table 2.7.1. Nomenclature and calculations of generalized connectivity model inputs. In the symbols below $m$, $r$ and $d$ refer to White-footed Mice, American Robins, and White-tailed Deer, respectively.

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Parameter</th>
<th>Calculation</th>
</tr>
</thead>
<tbody>
<tr>
<td>$t$</td>
<td>Total abundance of ticks per patch (sum all life stages)</td>
<td>Estimated from observed number of ticks per hectare</td>
</tr>
<tr>
<td>$t_\phi$</td>
<td>Potential tick infection prevalence</td>
<td>Infected ticks / total ticks (estimated from observed avg. prevalence= 23%)</td>
</tr>
<tr>
<td>$t_\phi \cdot \text{inf}$</td>
<td>Potential abundance of infected ticks per patch</td>
<td>$t \times t_\phi$</td>
</tr>
<tr>
<td>$N_m, N_r, N_d$</td>
<td>Density of host species (mice, robins, and deer, respectively)</td>
<td>Mice ha$^{-1}$ (estimated from observed data), robins and deer ha$^{-1}$ (published data)</td>
</tr>
<tr>
<td>$m, r, d$</td>
<td>Abundance of given host species</td>
<td>$(N_m, N_r, N_d) \times$ deciduous forest area (ha)</td>
</tr>
<tr>
<td>$B_m, B_r, B_d$</td>
<td>Average tick burden for mice, robins, and deer, respectively</td>
<td>Published data (Table 2)</td>
</tr>
<tr>
<td>$N_m, N_r, N_d$</td>
<td>Average abundance of hosts burdened with ticks</td>
<td>Mice: min ($N_m, B_m$); Robin: min ($N_r, B_r$); Deer: min ($N_d, B_d$)</td>
</tr>
<tr>
<td>$N_m, B_m, B_r, B_d$</td>
<td>Average infected-tick burden for mice or robins</td>
<td>($B_m, B_r, B_d) \times t_\phi$</td>
</tr>
<tr>
<td>$N_{m, \text{inf}}, N_{r, \text{inf}}$</td>
<td>Average abundance of hosts burdened with infected ticks</td>
<td>Mice: min ($N_m, B_{m, \text{inf}}$); Robin: min ($N_r, B_{r, \text{inf}}$)</td>
</tr>
<tr>
<td>$N_{m, \text{inf}}$</td>
<td>Abundance of infected mice</td>
<td>$m \times 0.266$ (avg. observed <em>Borrelia</em> prevalence in <em>Peromyscus leucopus</em>)</td>
</tr>
<tr>
<td>$N_{r, \text{inf}}$</td>
<td>Abundance of infected robins</td>
<td>$r \times 0.266$ (avg. assumed <em>Borrelia</em> prevalence in <em>Turdus migratorius</em>)</td>
</tr>
<tr>
<td>$k_t$</td>
<td>Number of ticks required to reach a vacant patch to allow for establishment of population</td>
<td>Assumed values: minimum $k_t = 2$</td>
</tr>
<tr>
<td>$k_{t, \text{inf}}$</td>
<td>Number of infected ticks required to reach a vacant patch to allow for establishment of infection</td>
<td>Assumed values: minimum $k_{t, \text{inf}} = 2$ (Additional $k$ values shown in Appendices 2.9.2.1 and 2.9.2.2.)</td>
</tr>
<tr>
<td>$k_m, k_r, k_d$</td>
<td>Number of hosts required to reach a vacant patch to allow for tick establishment</td>
<td>Mice: $k_t / B_m$; Robins: $k_t / B_r$; Deer: $k_t / B_d$</td>
</tr>
<tr>
<td>$k_{m, \text{inf}}, k_{r, \text{inf}}$</td>
<td>Number of hosts required to reach a vacant patch to allow for infected tick establishment</td>
<td>Mice: $k_{t, \text{inf}} / B_m$; Robins: $k_{t, \text{inf}} / B_r$</td>
</tr>
</tbody>
</table>
Table 2.7.2. Parameters used in the generalized model calculated from published data.

<table>
<thead>
<tr>
<th>Host</th>
<th>Density&lt;sup&gt;a&lt;/sup&gt; Mean (range)</th>
<th>Tick burden&lt;sup&gt;b&lt;/sup&gt; Average</th>
<th>Water-crossing dispersal&lt;sup&gt;c,d&lt;/sup&gt; Average (max)</th>
<th>Terrestrial dispersal&lt;sup&gt;e&lt;/sup&gt; Mean (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>White-footed Mice</td>
<td>32.75 ha&lt;sup&gt;-1&lt;/sup&gt; (22 - 43.5)</td>
<td>10.17</td>
<td>38.40 m (233.17 m)</td>
<td>75.7m (25-300m)</td>
</tr>
<tr>
<td>Peromyscus leucopus</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>American Robins</td>
<td>0.52 pairs ha&lt;sup&gt;-1&lt;/sup&gt; (0.03 - 1.15)</td>
<td>2.95</td>
<td>135 m (742 m)*</td>
<td>135 m (742 m)*</td>
</tr>
<tr>
<td>Turdus migratorius</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White-tailed Deer</td>
<td>0.42 ha&lt;sup&gt;-1&lt;/sup&gt; (0.08 – 0.91)</td>
<td>185</td>
<td>2 600 m (4000 m)</td>
<td>16 000 m (3.0-38 km)</td>
</tr>
<tr>
<td>Odocoileus virginianus</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Mice: (Sullivan 1977, Goundie and Vessey 1986); Robin (Bayne and Hobson 2001); Deer (Porter et al. 2004)

<sup>b</sup> Burdens averaged across tick life stages from published data (Anderson and Magnarelli 1984, LoGiudice et al. 2003)

<sup>c</sup> Mice: (Sullivan 1977, Jacquot and Vessey 1995); Robin: (Haas 1995); Deer: (Vellend et al. 2003)

<sup>d</sup> Mice: (Sheppe 1965, Sullivan 1977); Robin: (Haas 1995); Deer: (Schemnitz 1975)

<sup>e</sup> Mice: (Goundie and Vessey 1986); Robin (Haas 1995); Deer: (Porter et al. 2004)

* Estimated from agricultural gap-crossing behaviour
Table 2.7.3. Relative contribution (%) of host dispersal capacity on tick versus pathogen spread. Model-derived spread estimates are further partitioned into $\text{PC}_{\text{intra}}$ (within-habitat spread), $\text{PC}_{\text{direct}}$ (spread by direct movement between habitat patches), and $\text{PC}_{\text{step}}$ (spread facilitated by stepping-stone habitat patches).

<table>
<thead>
<tr>
<th></th>
<th>Amount of reachable habitat (%)</th>
<th>Spread within habitat (%)</th>
<th>Spread by direct movement (%)</th>
<th>Spread by stepping stones (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(ARH)</td>
<td>38.4m</td>
<td>135m</td>
<td>2600m</td>
</tr>
<tr>
<td><strong>Tick</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White-Footed Mice</td>
<td>0.18</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>American Robins</td>
<td>0.11</td>
<td>0.13</td>
<td>-</td>
<td>90.83</td>
</tr>
<tr>
<td>White-tailed Deer</td>
<td>0.12</td>
<td>0.19</td>
<td>0.84</td>
<td>79.12</td>
</tr>
<tr>
<td><strong>Pathogen</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mice (burdened)</td>
<td>0.14</td>
<td>-</td>
<td>-</td>
<td>60.28</td>
</tr>
<tr>
<td>Infected mice (burdened)</td>
<td>0.20</td>
<td>-</td>
<td>-</td>
<td>67.75</td>
</tr>
<tr>
<td>Infected mice (unburdened)</td>
<td>0.13</td>
<td>-</td>
<td>-</td>
<td>30.01</td>
</tr>
<tr>
<td>Robins (burdened)</td>
<td>0.11</td>
<td>0.13</td>
<td>-</td>
<td>90.83</td>
</tr>
<tr>
<td>Infected robins (burdened)</td>
<td>0.03</td>
<td>0.03</td>
<td>-</td>
<td>94.02</td>
</tr>
<tr>
<td>Infected robins (unburdened)</td>
<td>0.01</td>
<td>0.01</td>
<td>-</td>
<td>0.01</td>
</tr>
</tbody>
</table>
2.8 Figures

Figure 2.8.1. Study region. Sampling occurred in nine islands and three mainland sites of the St. Lawrence River from 2009-2010 in Thousand Islands National Park, near Gananoque (Ontario, Canada). Unsampled islands in the region were included if they fell within the average maximum extent of deer movement (16 km) during the summer and fall seasons from the sampled islands (Porter et al. 2004). The circle represents a focused region of interest represented in Figure 2.8.5.
Figure 2.8.2. Conceptual framework of generalized network connectivity model, modified from Saura et al. (2014). Between any given pair of patches $i$ and $j$, the model was parameterized using three general factors: (a) $N$ values, calculated in patch $i$, to represent the number of potential dispersers; (b) $k$ values, calculated in patch $j$ to represent the minimum number of dispersers required for population establishment in patch $j$; and (c) dispersal kernel estimates calculated per host to determine likelihood of dispersal success by direct movement (solid arrow) or by the use of stepping stone patches (dashed arrows). Ultimately, $N$ and $k$ values are calculated in terms of the number of hosts required to disperse both the tick and pathogen population (see Table 2.7.1) between all possible pairs of patches in the network ($n = 438$).
Figure 2.8.3. Potential amount of reachable habitat (ARH) by (a) ticks (*Ixodes scapularis*) via burdened hosts; and (b) the pathogen (*Borrelia burgdorferi*) via infected ticks and infected hosts. ARH by ticks is a function of the tick burden per host, density of hosts, dispersal distance of a given host (indicated by vertical lines as average maximum distance per host, detailed in Table 2.7.2) and the spatial configuration of patches. For the pathogen, ARH is also dependent on the relative prevalence of the pathogen in the tick or host population. Values of $k$ relate to the minimum number of infected ticks ($k_{it}$) required for population establishment in a destination patch $j$ (see Table 2.7.1 for details). Grey shading indicates poor host capacity to disperse ticks or the pathogen beyond the given distance.
Figure 2.8.4. Contribution to the amount of reachable habitat by connectivity fractions: (a) habitat that can be reached within patches (PC_{intra}); (b) direct movement between source and destination patch without using any other intermediate patch (PC_{direct}); and (c) amount of connectivity between source and destination patches that is due to the contribution of stepping stones (PC_{step}). Grey shading indicates poor host capacity to disperse ticks or the pathogen beyond the given distance.
Figure 2.8.5. Illustration of relative role of landscape on tick or pathogen spread dependent on host dispersal host at a local scale (focus region shown in Fig. 2.8.1). Probability of connectivity is calculated in the model by within-patch spread (PC_{intra}, thick red lines); direct dispersal between pairs of patches (PC_{direct}, solid lines) and dispersal when accounting also for stepping stones between pairs of patches (PC_{step}, dashed lines). For infected, burdened (a) White-footed Mice, and (b) American Robins burdened by infected ticks, pathogen dispersal is largely confined to spread within patches. Conversely, tick spread by (c) White-tailed Deer is most contributed by stepping-stone patches over large distances. For short-distance host movements, stepping stones facilitate pathogen spread; for long-distance host movements, stepping stones facilitate tick spread.
2.9 Appendix

2.9.1 Tick dragging and diagnostic testing

Tick surveys were completed in June, August, and October 2009, and May, June, August, and October 2010, corresponding to periods of peak activity of each tick life stage (Daniels et al. 2000). Tick dragging occurred during daylight hours with no rainfall. A 1-m² flannel sheet was dragged through forest vegetation within one-hectare study plots per site for a total of two person hours per sampling session. Sheets were checked every two to five minutes and all ticks were removed. Ticks were placed in split-top vials in the field, then killed in 99% isopropyl alcohol and stored at -20°C until they were shipped to the Public Health Agency of Canada National Microbiology Laboratory in Winnipeg. All samples were stored at -80°C prior to testing. All ticks were counted and identified to species using a taxonomic key (Durden and Keirans 1996). Ticks identified as *I. scapularis* were tested for *B. burgdorferi* by polymerase chain reaction (PCR) (Bouchard et al. 2013, Werden et al. 2014). Samples were considered positive when they produced cycle threshold values < 40 with two different primer and probe sets on real-time PCR. If more than 30 ticks were collected during one sampling session, a minimum of 30 were arbitrarily selected for testing.


2.9.2 Figures

Figure 2.9.2.1. Amount of reachable habitat when $k = 10$. In the generalized connectivity model, $k$ values represented the minimum number of ticks required to establish a population in a destination patch. I performed analyses of (2.9.2.1) the amount of reachable habitat, and (2.9.2.2) partitioned connectivity fractions, using $k = 2$ (as shown in main text and Figures) $k = 10$ (as shown here) to identify how the role of the landscape may change per host per tick or pathogen scenario. I identified that robin dispersal is affected by increasing $k$ values in the tick and pathogen spread scenarios. Potential amount of reachable habitat is reduced for both tick and pathogen spread (2.9.2.1).

Tick and pathogen spread by robins was confined to within-habitat movement to a greater extreme than the $k = 2$ scenarios (2.9.2.2).
Figure 2.9.2.2. Refer to figure 2.9.2.2 for description.
Chapter 3

3 Urbanization, Grassland, and Diet Influence Coyote (*Canis latrans*) Parasitism Structure

3.1 Abstract

Land-use change can alter the ecological mechanisms that influence infectious disease exposure in animal populations. However, few studies have empirically integrated the environmental, spatial, and dietary patterns of wildlife epidemiology. I investigated how urbanization, habitat type, and dietary behaviour are associated with coyote (*Canis latrans*) parasitism structure along a gradient of rural to urban land cover using multivariate redundancy analyses. Coyote fecal samples were collected in eight urban and six rural sites in Calgary (Alberta, Canada). Parasite and diet components were identified using common flotation procedures and fecal dietary analysis, respectively. Redundancy analysis was used to identify the best land-cover, connectivity, and dietary predictors of variation in prevalence of fifteen parasite species among sites. I tested for significance using multiple permutation tests and ANOVAs. Significant factors affecting enteric parasite prevalence included dietary and land-cover factors (adj.-$R^2 = 0.413$, $p < 0.05$). Variation in dietary behaviour was observed between urban and rural sites (adj.-$R^2 = 0.471$, $p < 0.05$), as anthropogenic diet items (i.e., garbage, crabapples) were strongly influenced by urbanization. My research supports that developed habitat, grassland cover, and dietary choice interact to possibly influence the exposure of coyote hosts to enteric parasites and pioneers future investigation of disease ecology for natural populations in anthropogenic landscapes.

3.2 Introduction

An essential goal in contemporary ecology is to predict ecological responses to anthropogenic disturbance and land-use change. Ecologists need to quantify the consequences of landscape modification on the structuring processes of species richness and community assembly observed over multiple scales (Chase 2003). Cities represent gradients of landscape modification, and species interactions in urban habitat may be characterized by different biotic and abiotic factors than more rural or pristine habitat (Rodewald et al. 2014). Global urbanization trends are likely
to result in unprecedented and complex ecological responses in species interactions, densities, and movement, leading to influential effects on the transmission dynamics of infectious disease (Shochat et al. 2006, Bradley and Altizer 2007, Berg and Ellers 2010). Because the emergence and re-emergence of infectious disease in human populations is inextricably linked to animal ecology (Taylor et al. 2001, Patz et al. 2004, Estrada-Peña et al. 2014), it is now essential to quantify how urbanization alters the patterns, prevalence, and transmission of parasites in natural wildlife populations. Yet, few studies have applied community ecology methods to evaluate how landscape change influences parasite assemblages in natural populations. Here I quantify, using multivariate ordination analyses, how urbanization influences Coyote (*Canis latrans*) parasitism and diet in Calgary, Alberta, Canada.

Coyotes evolved and have persisted on the North American continent for over 1 million years (Wang and Tedford 2008). Their resilience and plasticity have allowed them to reclaim habitat, including remnants within cities, and to become ‘urban-adapted’ carnivores. Calgary coyotes have a diverse diet (Lukasik and Alexander 2012) and parasite infracommunity (Watts and Alexander 2012), and live within a complex and diverse habitat matrix. Rapid urban development has increased human and pet concentration in cities, and there has been an increase in reports of interactions between people and coyotes (Alexander and Quinn 2011). The ‘urban coyote’ phenomena has now turned to the potential for infection to manifest and transfer between wild and domestic canids (*Canis lupus familiaris*), and on to humans. Coyotes, therefore, are a representative urban wildlife species to examine the ecological intersection of landscape, parasitism, and public health.

Habitat fragmentation can profoundly affect host-parasite interactions by altering the niche breadth of both hosts and parasites (Daszak et al. 2001). Higher host densities could facilitate increased contacts between susceptible and infectious hosts at aggregated resource sites within isolated urban habitat patches (Wright and Gompper 2005). For coyote hosts, exposure may occur through contact with high densities of infected definitive (e.g., other coyotes, wolves, domestic canids) and intermediate hosts (e.g., small mammals), resulting in contaminated, isolated habitat (e.g., infected feces). Habitat fragmentation may then influence the abiotic environmental conditions necessary for parasites to persist outside of the host (Hagenaars et al. 2004). Many macroparasites have evolved life cycles that generally include stages off the host.
(e.g., helminths and arthropods) or in intermediate hosts (e.g., insects, amphibians, small mammals). Climate (Gems 2000) and vegetation (Pietrock and Marcogliese 2003) have been shown to influence the longevity of free-living parasite in the environment. Similarly, habitat degradation has been shown to increase the relative density of potential intermediate hosts such as rodents (Suzán et al. 2008). Therefore, urban development may impact host (definitive and intermediate), climatic, and vegetation factors on which parasite persistence depends (Deplazes et al. 2004).

Diet can also have an important role in carnivore parasite prevalence (Wirsing et al. 2007, Aguirre 2009). Generalist carnivores (i.e., coyotes) exist and disperse throughout urban areas, and feed on a variety of dietary items including animal species, vegetation, human sources (i.e., garbage), and infected intermediate hosts (Kellner et al. 2012). Definitive host carnivores that eat infected intermediate hosts (i.e., some rodent, amphibian, and insect species) are exposed to infection (Reperant et al. 2009). Dietary choice is likely different in rural versus urban habitat. Therefore, I expect that urbanization affects host dietary exposure risks and parasite prevalence.

Here, I seek to improve our understanding of the effect of urbanization on coyote parasite infracommunity structure (Bush et al. 1997) by examining the associations between habitat spatial heterogeneity, dietary behaviour, land cover, and the prevalence of parasites in the environment. I used redundancy analysis (RDA) to explore whether landscape and dietary components are associated with patterns of parasitism within a canid host metapopulation. My objectives were to describe variation in (1) coyote gastrointestinal parasite taxa using land-cover, connectivity, and dietary factors; and (2) coyote dietary assemblage using land-cover and connectivity factors. I expected that small-mammal diet items would have the strongest statistical association for Taenia-like spp. and Toxascaris leonina, because as intermediate hosts, small mammals are potential dietary sources of macroparasitic infection (Reperant et al. 2009) and coyotes that feed on these rodents are assumed to be more likely to be exposed to infection. I also predicted that urban cover and connectivity would explain the parasite assemblage best, assuming highly connected urban sites were more commonly occupied, thereby increasing transmission of infective larvae among other wild or domestic canid hosts. Finally, I predicted that urban cover would represent anthropogenic dietary choices (i.e., crabapples, domestic
animals, garbage) more significantly than rural cover, negatively influencing coyote parasite prevalence.

3.3 Methods

3.3.1 Sampling and laboratory analyses

Coyote fecal samples were collected opportunistically during weekly sampling days for 1 year (July 2009–June 2010) along transects within eight urban and six rural sites of Calgary, Alberta (51.083°N; 114.083°W) (Fig. 3.10.1). Sampling was standardized to 1 km transects known to be common defecation sites, consisting of defined paths and roads, described fully in Appendix 3.11.1.1. Despite having no link between fecal samples and individuals, greater numbers of fecal samples were assumed to provide an index of the parasitism shared by coyote groups (likely single denning sites) per sampling area. Edges of each transect were chosen at a minimum distance of 4 km from other transects in alternative sampling sites derived by average maximum reported home ranges of coyotes in North American cities (Grinder and Krausman 2001).

Samples were collected by two independent observers trained by one expert tracker. Coyote and domestic dog fecal samples were differentiated using morphological characteristics (Halfpenny 1986). I found coyote and domestic dog feces in situ were highly distinct morphologically. Weekly sampling was performed to improve detectability of fecal parasites which decay within four days of deposition in the free-living environment. However, samples were screened again prior to laboratory analysis. Fecal samples were frozen at -80°C in a freezer for 72 h to denature any infective-stage larvae, specifically *Echinococcus* spp. (Dryden et al. 2005).

Each of 256 collected fecal samples were analyzed first for parasitism and secondly for diet components (a paired analysis). I used a double-centrifugation fecal flotation (Zajac and Conboy 2012), a common method in carnivore fecal parasitism surveys (Gompper et al. 2003, Lesmeister et al. 2008, Liccioli et al. 2012) (Appendix 3.11.1.2). A total of 4 g of feces were extracted from equally distributed cross sections of each fecal sample and homogenized before centrifugation. Multiple microscope slides were prepared per homogenized sample. Parasites were identified to the lowest taxonomic level possible based on morphology, recorded as
prevalence per site (number of infected samples to total number of samples) for each parasite genus. I identified morphologically indistinguishable parasites to the genus-level instead of species-level, assuming that environmental and host associations with parasite groups are similar. Prevalence was measured to quantify a relative measure of the parasite assemblage in coyote subpopulations.

Dietary components were identified using a point-frame method (Chamrad and Box 1964) whereby fecal samples were separated into components over a 2.5 × 2.5 cm tabular grid (in total 25 cm²). Relative abundance of categorized diet items (Appendix 3.11.1.2) were then recorded as percentage according to the proportionate abundance in the fecal sample to other material. Percent by volume was measured for each dietary component on a 2.5 cm grid. Relative frequency of occurrence was determined per sampled site, as explained in Lukasik and Alexander (2012) (Tables 3.8.2 and 3.8.3).

3.3.2 Land cover and connectivity measurement

I used ArcGIS 10.0 (ESRI 2011) to extract land cover and connectivity measures (Tables 3.8.4 and 3.8.5) from CanMap RouteLogistics Alberta v2013.3 (DMTI 2013) and The City of Calgary Coyote database (2007–2011; Spatial and Numeric Data Services, University of Calgary). Land cover was categorized into vegetation (i.e., grassland, broadleaf forest, coniferous forest, mixed forest); urbanization (i.e., urban cover, developed cover, residential density, industrial land, agricultural land, roads/impervious surfaces); domestic dog park cover (i.e., off-leash, on-leash/path only, no dogs); and hydrology (i.e., water bodies, distance to water) (Table 3.8.1). Land-cover variables were extracted for each sample at two home range estimates to represent possible critical scales (Baguette and Van Dyck 2007) of published coyote dispersal variation: a 500 m radius, estimated average urban home range; and a 2000 m radius, estimated average rural home range (Grinder and Krausman 2001). All land-cover variables were verified in situ during sampling to ensure accuracy of remotely-sensed land-cover estimates. I then calculated percent land cover within each home range radius using Geospatial Modeling Environment 0.7.2.0 (Beyer 2012).

Structural and functional connectivity (Taylor et al. 1993) were measured to reflect fragmentation (Table 3.8.1) and have been applied to measure the effect of animal movement on
disease processes (Real and Biek 2007, Vander Wal et al. 2012). Structural connectivity is an index of the composition and configuration of habitat patches, not considering the dispersal ability of the study organism. This spatial predictor was measured using patch area, distance to nearest neighbour, and centrality. Patch centrality considered core and corridor location within a patch network, as was calculated using the LinkageMapper tool v. 0.9 (McRae and Kavanagh 2011). Functional connectivity, an index of landscape permeability dependent on dispersal limitations of the study organism, was measured using the probability (%) of connectivity (Saura et al. 2014). This calculation returned per-site dPC values, calculated per site at two dispersal thresholds (500 and 2000 m) according to urban and rural home ranges, respectively. I assumed a fat-tailed dispersal kernel, which was related to 5% of individuals surpassing the assumed average dispersal maxima.

3.3.3 Tests and representations of relationships between variables

The best predictors within each predictor category were chosen using criteria specified in previous research and detailed below (Peres-Neto et al. 2006). First, I excluded rare parasite species (<5% representation in the regional parasite community) and diet components (i.e., berries, unknown items) to prevent inflation of response variation. Using tests of multivariate homogeneity (Anderson 2006), no significance was identified among sites for parasitism ($p = 0.583$) and diet ($p = 0.619$) suggesting normality among observed prevalence and relative abundance data, respectively (Appendix 3.10.1.3). The proportion of species present relative to the number of fecal samples (prevalence) was Hellinger-transformed (Legendre and Gallagher 2001) to reduce the effect of extreme values and double-absences on matrix output and to account for small sample sizes within some sites. Finally, I transformed land-cover variables to approach normality (Table 3.8.1). Predictors with variation inflation factors (VIF) value of ~4 were excluded from the analysis to minimize collinearity (Zuur et al. 2009).

I used a constrained canonical ordination method (redundancy analysis RDA; Legendre and Legendre, 2012) to explain the variation of the prevalence of multiple enteric parasite species by multiple land-cover and dietary variables. Redundancy analyses regress multiple response variables on multiple explanatory variables to provide constrained, linear combinations of explanatory variables that explain a matrix of responses included simultaneously in the same
singular model. RDA is both a constrained form of PCA and constrained form of multivariate multiple regression ideal for ecological datasets representing uneven and small sample sizes among sites (Braak and Smilauer 1998, McArdle and Anderson 2001) and has been employed in previous investigations of environmental associations with host helminths communities (Chambers and Dick 2005, Aguirre-Macedo et al. 2007). Therefore, multivariate statistical methods were best suited for my analyses and empirical data to determine possible associations between supplementary environmental and dietary biological variables, and multiple enteric parasite infracommunity parameters. For parsimony, I limited redundancy models to five predictors (Legendre and Legendre 2012) (Appendix 3.10.1.4). The significance of the RDA models, canonical axes, and individual predictors was tested using a Monte Carlo permutation test (1000 permutations), with a significance level of $p < 0.05$ (Legendre et al. 2011). Multifactorial permutation tests are acceptable for small sample sizes on transformed data as long as sample sizes are, as a rule of thumb, >10 per site (Anderson and Legendre 1999, Anderson 2001). I performed variance partitioning on the top RDA model for each objective to quantify the variance explained by categorical groups of predictors (Zuur et al. 2009). An ANOVA was performed per model to test for significance of each predictor using the ‘adonis’ function in the vegan library in R (Venables and Smith 2011).

3.4 Results

Twelve months of surveys yielded 256 coyote fecal samples from eight urban and six rural sites in the region of Calgary, Alberta (Fig. 3.9.1). Number of samples per site was variable (Table 3.8.2). I identified two cestodes (*Dipylidium caninum* and *Taenia*-like spp.), five nematodes (*Capillaria* spp., *Toxascaris leonina*, *Toxocara canis*, *Trichuris* spp. *Uncinaria stenocephala*), two protozoa (*Cystoisospora* spp. and *Sarcocystis* spp.), and a trematode (*Alaria* spp.) (Watts and Alexander 2012) (Table 3.8.2). Variation in relative abundance of diet components was observed among all sites (Table 3.8.3).

RDA models were developed using landscape variables measured at 500 and 2000 m radii to represent critical scales of urban and rural home range size, respectively, but the strongest significance was at the 500 m urban home range scale (Tables 3.8.4 and 3.8.5). Neither structural nor functional connectivity metrics were included in the best model explaining parasite
prevalence. RDA models were significant, though they varied in significant values, percent of constrained ordination explained, and adjusted-$R^2$ values (Table 3.8.4).

Candidate models that explained parasitism using only diet were relatively poor in performance, adjusted-$R^2 = 0.180$ (Table 3.8.5). Models that included only land-cover and connectivity predictors had an increased significance, adjusted-$R^2 = 0.3348$, but remained relatively poor in describing the parasite infracommunity. By including land-cover, connectivity (Appendix 3.10.2.1) and diet components to predict parasite prevalence, the best model was parasite prevalence * grassland + agriculture + water + plant + domestic animals, adjusted-$R^2 = 0.4130$. Monte Carlo permutation tests confirmed the significance of the whole model ($p < 0.05$) on the first three primary axes ($p < 0.05$) in RDA. All but one factor were significant in the model; grassland, domestic animals, and parks/recreation had the strongest influence. This candidate model has the highest significance explaining the coyote gastrointestinal parasitism in my study area. Grassland was associated with *Taenia*-like spp., *Cystoisospora* spp., and *Toxascaris leonina*; agriculture was associated with *Trichuris* spp. and *Capillaria* spp.; water and plant diet components were associated with *Uncinaria* spp.; and domestic animals were most closely associated to *Sarcocystis* spp., *Dipylidium* spp., and *Toxocara* spp.

Using land-cover and connectivity factors to predict diet components (Objective 2), the best model was diet components * grassland + agriculture + water + coniferous forest + patch centrality, adjusted-$R^2 = 0.4712$. This model was the most significant of all four RDA analyses. Monte Carlo permutation tests confirmed the significance of the whole model ($p < 0.05$) on the first three primary axes ($p < 0.05$) in RDA (Fig. 3.9.2). All predictors were significant with the exception of ‘water.’ Grassland was associated with bird dietary components; agricultural land cover was associated with cattle, deer, and insect dietary components; coniferous forest was proximately associated with small mammals, mesomammals, and plant dietary components; and patch centrality and water were closely associated with garbage and crabapples. Patch centrality resulted in an indirect measure of distance to urban core (Table 3.9.4; Appendix 3.10.2.1).

Variance partitioning demonstrated that urbanization explained 28.1 and 40.6% of the variance for the best parasitism and diet models, respectively (Fig. 3.9.3). Independent from urbanization, grassland cover explained 19.9 and 11.0% of the variance for the best parasitism
and diet model, respectively. For the parasitism RDA only, diet explained 13.9% of the variation, while natural cover factors explained 15.8% of the variation in the diet assemblage model.

3.5 Discussion

Land cover and fragmentation can have cascading effects on natural communities (Chase 2003, Leibold et al. 2004). Yet, the influence of habitat spatial heterogeneity on parasite communities in host populations remains relatively unexplored. I demonstrated that combined land-cover and dietary factors best explained wild canid enteric parasitism infracommunity structure. Diet components alone did not perform as well as landscape factors in explaining parasitism structure; landscape factors alone had better explanatory power than diet components, but the best model combined these influences (Tables 3.8.4 and 5, Fig. 3.9.2). Partitioning of land-cover vs. dietary factors demonstrates the relative influence of urbanization (i.e., agriculture vs. urban recreation areas) versus specific landscape characteristics (i.e., grassland) independent from developed cover that are influential on both dietary behaviour and parasite exposure to hosts in natural populations (Fig. 3.9.3).

Parasite taxa recovered from my observed enteric parasite infracommunity were also recovered in subsequent coyote parasitism surveys in Calgary (Liccioli et al. 2012) and previous surveys in Canada (Holmes and Podesta 1968, Thompson et al. 2009). These enteric parasite species are common in canid hosts, likely having little effect on population mortality relative to viral infection (i.e., rabies, canine distemper) or intense sarcoptic mange infection, road mortality, or human population management. Therefore, I assume that coyotes act as reservoir hosts for the observed parasite assemblage. Only *Toxocara canis* poses potential zoonotic risks in free-living stages, though it is likely that recovered *Taenia*-like eggs were actually *Echinococcus* spp. eggs, a zoonotic cestode known to be prevalent in the Calgary coyote population (Catalano et al. 2012). On a regional scale, the urban and rural macroparasite assemblage was almost identical in structure. These commonalities demonstrate the persistence of a regional parasite species assemblage (urban and rural combined) over time, but variation in species prevalence among individual sites shows that local factors shape the coyote parasite infracommunity. The ecological associations between local land-cover, coyote behaviour, and parasitism-structuring processes that I observed were complex, as discussed below.
3.5.1 Dietary influence on parasite prevalence

The observed differences in urban-rural diet components reveal how landscape structure may influence exposure of coyotes to parasitism through dietary versus environmental factors. I found distinct dietary patterns between urban and rural sites (Objective 2, Fig. 3.10.2): agriculture (i.e., rural) was positively associated with deer, cattle, and insects and negatively associated with crabapple and garbage. In rural habitats, coyotes may forage on natural or livestock diet items, where natural items (i.e., intermediate hosts) are more likely sources of parasitism via consumption. In urban habitat, coyotes may preferentially forage on anthropogenic items such as garbage and crabapples. Ingestion of garbage and crabapples are not likely sources of gastrointestinal parasites. In urban habitat, it follows that diet may be a less probable source of exposure to parasite diversity than environmental transmission or transmission via alternate hosts.

I expected specific associations between diet components and parasites due to transmission stages in rodents of some free-living parasites, but no association was identified. For example, I expected small mammals to be associated with *Taenia*-like spp., which often develop in rodent intermediate hosts, specifically voles and white-footed mice (Bowman and Georgi 2009, Reperant et al. 2009). Similarly, *Toxascaris leonina* can be maintained in intermediate mice or rat populations. However, statistical associations between taeniidae parasites and small or large mammals were not identified in my analyses, and in general I found that solely dietary components explained parasite prevalence weakly. Taeniidae helminths can infect a variety of wild or domestic livestock animals like sheep, goats, and pigs, though no small ungulate or swine production occurred on sampled agricultural areas (O’Connor et al. 2006). Similarly, due to identical morphology of some cestode eggs identified by flotation methods, *Taenia*-like spp. may also have been *Echinococcus* spp. (Catalano et al. 2012) which can be associated with rodent intermediate hosts (*E. multilocularis*) such as voles, or large-bodied ungulate hosts (*E. granulosus*) (McManus et al. 2003). Temporal delays between ingestion of infected diet components (e.g., intermediate rodent hosts) and infection in the coyote host may explain my poor correlation between diet and parasitism.
3.5.2 Land cover influence on parasite prevalence

Urban land cover and grassland had independent effects on parasite structure. First, land-cover types that represented urban-rural heterogeneity (i.e., parks/recreation, agriculture) were associated with urban vs. rural partitioning in parasite composition (Fig. 3.10.3). For example, agricultural land cover was intuitively associated with the parasite genera *Capillaria* spp. and *Trichuris* spp., parasites that commonly infect small-mammal rodents and multiple livestock hosts (Epe et al. 2004), respectively. Parks/recreation and grassland were associated with *U. stenocephala* and *T. canis*, species that commonly infect domestic canid hosts, representing urban exposure to parasitism. I expect that alternative definitive canid hosts, such as domestic canines, play an important role in exposure risks to coyote populations via parasite spillover. Exposure to parasitism during free-living parasite stages should not be density-dependent as a rule (Lafferty and Holt 2003). However, in fragmented urban environments where domestic canid population density is artificially high relative to what a habitat might naturally support, density may influence indirect transmission among wild and domestic canids. Therefore I suggest overlap in habitat use (i.e., domestic animals and coyotes in public parks; cross contamination through feces and prey) influences parasitism in natural populations, and these effects are likely amplified in urban core versus rural or pristine habitat. Recreational grasslands may also pose infection risks to human populations exposed to free-living stages of *Echinococcus* spp. or *Toxocara canis* via direct contact with infected canid feces.

I found a relationship between grassland and parasitism independent from urban-modified landscape change, and suggest that the convergence of host and parasite species on grassland predicts parasite prevalence. My results demonstrate associations between grassland and taeniidae parasites. Taeniids commonly develop in intermediate hosts, including domestic livestock (e.g., cattle, sheep, pigs) and ground-dwelling small mammals (e.g., rodents or rabbits) (Bowman and Georgi 2009). These animals represent different sources of rural or urban taeniid infection, but may be found near or within grasslands and agricultural areas with domestic livestock hosts. Grassland is known as preferred foraging and denning habitat for coyotes (Gese et al. 1988) and has been identified as high-risk land cover for suburban and urban feline tularemia infection risks (Raghavan et al. 2013). Additionally, grassland represents suitable habitat for small-mammal intermediate hosts such as rodents and rabbits (Jones et al. 2003).
Finally, I suggest that grassland vegetation (upland grasses and forbs) may enhance survival or transmission of free-living parasite stages, especially cestodes. Environmentally-mediated transmission is a vital component in metapopulation disease dynamics (Park 2012), and microhabitat suitability for free-living parasite stages in grasslands likely plays a significant role in parasitic exposure of wild or domestic hosts (Pietrock and Marcogliese 2003) independent from urbanization.

I expected structural and functional connectivity metrics (i.e., ability of landscape composition to facilitate coyote dispersal, Appendix 3.10.2.1) would positively influence parasite prevalence. Dispersing coyote hosts may forage in more connected habitat, especially in urban landscapes where inter-habitat matrix should limit movement. Yet, no functional connectivity predictors were found in my best RDA model. Temporal delay (latency of infection) between contact of a parasite species via ingestion or environmental contamination may explain a loss of some spatial structure in the coyote parasite infracommunity.

3.5.3 Limitations

My exploratory analysis was performed by non-invasive methods. These methods place predictive limitations on my models by an inability to link fecal sample—and parasite assemblage—to individual coyotes. I assumed that individual fecal samples represented subpopulations with similar infection prevalence, especially if individual sites represented family groups who likely share infections via frequent contacts within and around denning sites. I widened my non-invasive search effort to sampling transects contained within the entire urban–rural Calgary region to capture parasitism patterns over a large geographic area. This strategy compromised per-site sample sizes. I accounted for small sample sizes in my statistical analyses, and the observed parasite taxa in my study reflect the relative prevalence of similar parasitism surveys in the region (Holmes and Podesta 1968, Thompson et al. 2009, Liccioli et al. 2012). Therefore, I do not consider non-invasive sampling a significant limitation for my exploratory research questions.

I was also limited in my laboratory analyses: flotation methods limited parasite identification to genus level, and diet analyses were limited to observable diet groups, not taxonomic level. Nevertheless, non-invasive parasitological methods facilitated my landscape-
scale ecological investigation with significant results in a feasible, ethical manner. In future studies, observed knowledge of age, sex, and number of individuals using fecal genotyping will improve non-invasive parasitological investigations.

### 3.6 Conclusions

The functional relationship between landscape change, host behaviour, and parasite transmission remains complex yet fundamental to understanding infectious disease dynamics in changing landscapes. My work demonstrates how habitat spatial heterogeneity influences animal host ecology and behaviour, posing different exposure probabilities and variation in parasite prevalence. Rising global urbanization rates will likely bridge contacts between infected animal hosts, domestic hosts, and humans in the future. Considering the emergence of *E. multilocularis*, Lyme disease, and West Nile virus in Canada and many international cities (Catalano et al. 2012, Nelder et al. 2014, Wang et al. 2014), and the strong likelihood each of these infections will be additionally influenced by climate warming (Townroe and Callaghan 2014), I recommend more collaboration between ecologists, veterinarians, and public health scientists toward a mechanistic understanding of urban wildlife disease ecology.

### 3.7 Acknowledgements

I would like to thank the City of Calgary; Spatial and Numeric Data Services (SANDS), at the University of Calgary for spatial data. I would like to thank the Department of Geography, University of Calgary, NSERC, the Alberta Conservation Association for funding of this research. I would especially like to thank the Sandy Cross Conservation Area, Hamish Kerfoot, Big Hill Springs Provincial Park, the Inglewood Bird Sanctuary, and Fish Creek Provincial Park for the allowance of sampling on their land. I further thank Dr. Susan Kutz for the use of laboratory equipment and expertise for parasitological investigation.

### 3.8 Tables
Table 3.8.1. Environmental and spatial variable codes and descriptions for vegetation characteristics, urbanization proxies, domestic dog designations, hydrological features, and structural and graph-based connectivity variables.

<table>
<thead>
<tr>
<th>Category</th>
<th>Variables</th>
<th>Description</th>
<th>Variable Preprocessing</th>
<th>Variable acronyms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetation</td>
<td>Grassland</td>
<td>% (m²) native grasses, shrub; herbaceous vegetation with 20% minimum 20% ground cover</td>
<td>ln(p(1-p))</td>
<td>GR</td>
</tr>
<tr>
<td></td>
<td>Broadleaf forest</td>
<td>% (m²) broadleaf trees (aspen, balsam poplar, white birch) are 75% or more of crown closure</td>
<td>ln(p(1-p))</td>
<td>BF</td>
</tr>
<tr>
<td></td>
<td>Coniferous forest</td>
<td>% (m²) conifers (spruce, pine, fir, larch) are 75% or more of crown closure</td>
<td>ln(p(1-p))</td>
<td>CF</td>
</tr>
<tr>
<td></td>
<td>Mixed Forest</td>
<td>% (m²) neither coniferous or broadleaf trees account for 75% of crown closure</td>
<td>ln(p(1-p))</td>
<td>MF</td>
</tr>
<tr>
<td>Urbanization</td>
<td>Developed</td>
<td>% (m²) cover urban and built-up areas</td>
<td>ln(p(1-p))</td>
<td>DL</td>
</tr>
<tr>
<td></td>
<td>Industrial</td>
<td>% (m²) cover industrial sites</td>
<td>ln(p(1-p))</td>
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</tr>
<tr>
<td></td>
<td>Agricultural</td>
<td>% (m²) cover annually cultivated cropland, tame pastures, forage crops</td>
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</tr>
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<td></td>
<td>Residential</td>
<td>% (m²) cover moderate-to-high density residential area</td>
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<tr>
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<td>Parks and Recreation</td>
<td>% (m²) cover designated community, school, or public recreation</td>
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<td>Distance to roads</td>
<td>Distance (m) to nearest road or highway feature ≥ 40km/h</td>
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<tr>
<td>Hydrology</td>
<td>Water</td>
<td>% (m²) cover lakes, rivers, canals, and artificial water bodies</td>
<td>ln(p(1-p))</td>
<td>WC</td>
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<tr>
<td></td>
<td>Distance to water</td>
<td>Distance (m) to nearest hydrological feature (described above)</td>
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<td>DistW</td>
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<tr>
<td>Domestic Dog Designation</td>
<td>Off-leash</td>
<td>% (m²) cover parks designated for off-leash dog allowance</td>
<td>ln(p(1-p))</td>
<td>OffL</td>
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<td>On-leash/Patch only</td>
<td>% (m²) cover parks designated for on-leash, path-only dog allowance</td>
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<td>No Dogs</td>
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<td>Structural Connectivity</td>
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<td>Centrality</td>
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<td>Functional Connectivity</td>
<td>Probability of connectivity</td>
<td>Probability (%) of dispersal to or from a given patch, limited by dispersal ability</td>
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<td>dPC</td>
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Table 3.8.2. Prevalence (%, 95% confidence interval) of parasite species response variables in RDA multivariate analyses, categorized by parasite genera, as recovered from fecal flotation.

<table>
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<tr>
<td>Arbor</td>
<td>15</td>
<td>132</td>
<td>27.0±2.63</td>
<td>-</td>
<td>-</td>
<td>53.0±25.26</td>
<td>7.0±12.96</td>
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<td>-</td>
<td>20.0±20.4</td>
<td>-</td>
<td>7.0±12.96</td>
</tr>
<tr>
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<td>-</td>
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<td>35.0±20.9</td>
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<td>62.0±19.84</td>
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<td>-</td>
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<td>11.0±12.52</td>
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<td>15.11 (±20.26)</td>
<td>18.89 (±22.15)</td>
<td>20.33 (±22.77)</td>
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<td>0.04 (±0.82)</td>
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<td>7.50 (±12.52)</td>
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<td>BHS</td>
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<td>65.61 (±18.62)</td>
<td>6.67 (±9.78)</td>
<td>11.85 (±12.67)</td>
<td>4.02 (±7.7)</td>
<td>2.11 (±5.63)</td>
<td>6.37 (±9.57)</td>
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<td>-</td>
<td>0.04 (±0.78)</td>
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<tr>
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<td>HCR</td>
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<td>70.92 (±17.8)</td>
<td>-</td>
<td>10.83 (±12.18)</td>
<td>1.17 (±4.22)</td>
<td>0.17 (±1.61)</td>
<td>5.28 (±8.77)</td>
<td>-</td>
<td>-</td>
<td>2.81 (±6.48)</td>
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<td>Kerfoot</td>
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<td>0.89 (±3.76)</td>
<td>1.21 (±4.37)</td>
<td>12.13 (±13.06)</td>
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<td>7.32 (±10.42)</td>
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<td>0.18 (±2.01)</td>
<td>0.88 (±4.44)</td>
<td>4.12 (±9.45)</td>
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</table>
Table 3.8.4. Summary of redundancy analyses including (1) multiple dietary and landscape predictors explaining multiple coyote parasite species responses; and (2) landscape predictors explaining multiple coyote dietary item responses. Significance values of models were evaluated through a permutation (9999) test for small sample sizes. Significance values of individual predictors were evaluated through permutational multivariate ANOVAs. Significance codes: (***) 0.001 (**) 0.01 (*) 0.05.

<table>
<thead>
<tr>
<th>RDA Candidate Models</th>
<th>Constrained Variance</th>
<th>adj.-$R^2$</th>
<th>Predictors</th>
<th>RDA Axes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Parasites ~ Diet + Landscape</td>
<td>0.1758</td>
<td>0.4130**</td>
<td>Parks and recreation **</td>
<td>RDA 1 **</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Grassland ***</td>
<td>RDA 2 **</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Agriculture *</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Plant</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Domestic animals ***</td>
<td></td>
</tr>
<tr>
<td>2. Diet ~ Landscape</td>
<td>0.1107</td>
<td>0.4712**</td>
<td>Grassland **</td>
<td>RDA 1 **</td>
</tr>
<tr>
<td></td>
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<td>Agriculture ***</td>
<td>RDA 2 **</td>
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<td></td>
<td>Water</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Coniferous forest *</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Patch centrality *</td>
<td></td>
</tr>
</tbody>
</table>
Table 3.8.5. Summary of redundancy analyses including only (1) dietary or (2) landscape predictors of multiple coyote parasite species responses. Significance values of models were evaluated through a permutation (9999) test. Significance values of individual predictors were evaluated through permutational multivariate ANOVAs. Significance codes: (***) 0.001 (**) 0.01 (*) 0.05.

<table>
<thead>
<tr>
<th>RDA Candidate Models</th>
<th>Constrained Variance</th>
<th>adj.-$R^2$</th>
<th>Predictors</th>
<th>RDA Axes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Parasites ~ Diet</td>
<td>0.1364</td>
<td>0.1803*</td>
<td>Small mammals, Birds, Insects, Plant, Domestic animals</td>
<td>RDA 1 *, RDA 2</td>
</tr>
<tr>
<td>2. Parasites ~ Landscape</td>
<td>0.1643</td>
<td>0.3448**</td>
<td>Parks and recreation, Grassland, Agriculture, Coniferous forest, Distance to nearest neighbour</td>
<td>RDA 1 **, RDA 2 **</td>
</tr>
</tbody>
</table>
3.9 Figures

Figure 3.9.1. Study area. Sampling sites are represented within urban or rural parts of the Calgary landscape. Rural sites: PR: Priddis Ranch; HCR: Horse Creek Road; BHS: Big Hill Springs Provincial Park; RT: Sandy Cross Conservation Area (SCCA)-Rancher’s Trail; 16A: SCCA-Regional Road 16A; 5A: SCCA-Regional Road 5A. Urban sites: AL: Arbor Lake; NHP: Nose Hill Park; TCH: Tom Campbell Hill; SP: Stanley Park; IW: Inglewood Wildlands; WH: Weaselhead Natural Park; FCW: Fish Creek Provincial Park-West; FCE: Fish Creek Provincial Park-East.
Figure 3.9.2. Redundancy analyses for each objective: (a) RDA parasite ordination constrained by landscape combined with dietary components; (b) RDA dietary ordination constrained by landscape variables. Predictor codes: SM (small mammals); DomesticAn (domestic animals); Plant (plant materials); Bird (avian species); Insects (invertebrates); AG (agricultural land); GR (grassland); PR (parks and recreation); CF (coniferous forest); NN (distance to nearest neighbours); WC (water cover); CEN (patch centrality).
Figure 3.9.3. Variance partitioning for the best RDA models, categorized by thematic groupings of predictors: (a) RDA parasite ordination partitioned by urbanization (agriculture + parks/recreation); diet (domestic animals + plants); and grassland. (b) RDA diet ordination, partitioned by urbanization (agriculture + patch centrality); natural cover (coniferous forest + water cover); and grassland. Values < 0 are not shown.
3.10 Appendix

3.10.1 Appendix 3.10.1. Detailed description of methods

Site selection and sampling

Sites were chosen based on known coyote occupancy to maximize opportunistic sampling yield, while representing habitat differences. A preliminary investigation of sites verified the presence of coyote feces by walking all sites in a grid-like pattern along transects ranging from 1 to 4 km transects. I observed diminishing yields of fecal samples beyond ~1km sampling distances in high defection areas. Sampling was therefore standardized by surveying a fixed 1 km transect per site, consisting of defined paths and roads known by preliminary observation to be common defecation sites to maximize sample yields per site. Edges of each transect were chosen at a minimum distance of 4km from other transects in alternative sampling sites derived by an average maximum reported home ranges of coyotes in North American cities. Because individual coyotes were not being tracked or followed, it was assumed that these transects were common defecation areas for local, independent coyote subpopulations.

Laboratory analyses

I used a double-centrifugation fecal flotation, a common method in carnivore fecal parasitism surveys. While this method can be unreliable for some canid parasite species, such as specific *Taenia* spp. and *Echinococcus* spp., I considered this method satisfactory for my objectives because I identified morphologically indistinguishable parasites to the genus-level instead of species-level, assuming that environmental and host associations with parasite groups are similar. I performed preliminary sensitivity analyses of the flotation method to consider the sensitivity of egg oocyst recovery and identification using 2g, 4g, and 6g of feces per sample. Multiple flotations were performed on individual samples to address within-sample parasite detectability. In both preliminary analyses, no differences were observed in the detectability of parasite presence/absence at the genus level. Therefore, a total of 4 g of feces were extracted from equally distributed cross sections of each fecal sample and homogenized before centrifugation and multiple microscope slides were prepared per homogenized sample.
Dietary components were identified using a point-frame method. Hairs and bones were classified to the lowest observable taxonomic level (Kennedy and Carbyn 1981) and then aggregated by dietary category (e.g., small mammals, plants, etc.) for analysis. Insects and birds were identified to order, based on exoskeleton remains and feather barbules. Vegetation and anthropogenic items were identified by morphological characteristics, using a dissection microscope. Percent by volume was measured for each dietary component on a 2.5 cm grid.

Tests of normality

I tested for multivariate homogeneity using permutation tests of observed parasite and diet observations, including beta-adjusted pairwise distances to account for small sample sizes (Anderson 2006, Gijbels and Omelka 2012). No significance was identified among sites for parasitism \( p = 0.583 \) and diet \( p = 0.619 \) suggesting normality among observed prevalence and relative abundance data, respectively. I performed a second test of normality by skewness \( p = 0.89 \) and kurtosis \( p = 0.12 \) using Mardia’s multivariate normality test (Korkmaz et al. 2014), where parasite prevalence and dietary relative abundances were suggested to be normally distributed.

Multivariate parameter selection

I used a constrained canonical ordination method (redundancy analysis RDA; Legendre and Legendre 2012) to explain the variation in coyote enteric parasite prevalence. For parsimony, I limited redundancy models to five predictors (Legendre and Legendre 2012). Before the RDA analyses were performed, I selected the predictors by ranking models using the Leaps library in R (R Development Core Team 2011). Based on the adjusted \( R^2 \) values, I kept the top five variables (Peres-Neto et al. 2006). I chose the top RDA models according to: (a) overall model significance; (b) significance of axes 1 and 2; (c) significance of at least two of five predictors; and (d) ecological significance to this study system.


3.10.2 Figures

Figure 3.10.2.1. Structural connectivity of sampled sites in Calgary, Alberta. Urban sites seem to have higher centrality than rural sites.
Chapter 4

4 Urbanization Alters Wildlife Host-Parasite Interaction Structure

4.1 Abstract

The role of urban environments in the structuring processes of natural communities is a critical question for ecologists yet remains largely unknown. In particular, it is hypothesized that urban-modified habitat may alter the ecological processes that structure host-parasite interactions, carrying implications for disease dynamics and potential zoonotic risks. I used bipartite network models to evaluate the role of urbanization on the structure of host-parasite interactions in southwestern Ontario (Canada). Infected wildlife host and parasite data obtained from the Toronto Wildlife Centre ($n = 3073, 2007-2012$) were consolidated to construct bipartite networks using urban and natural land-cover indices. I compared distributions of interactions between (a) urban and natural gradients; (b) mammalian versus avian host species; and (c) between host and parasite functional groups (taxonomic class) using quantitative network metrics tested against null models. I identified significant asymmetry and nested structures of host-parasite interactions along urbanization and natural gradients to greater magnitude in avian than mammalian host species. Synanthropic, generalist hosts (i.e., pigeons and doves, small carnivores, small rodents and moles) maintained the most diverse and frequent observed parasitic interactions in suburban and urban sites. My results stress that urban-modified landscapes likely shape a host-parasite dominance hierarchy, favouring the persistence of a core, cohesive group of parasitic infections within the urbanized host community. I reinforce previous findings that intermediately-disturbed suburban environments may have a unique role in maintaining host parasitism. These original analyses provide improved perspectives concerning the impact of urbanization on ecosystem structure but carries possible consequences for zoonotic risks posed to human dwellers.

4.2 Introduction

It is well established among ecologists that rapid urbanization causes shifts in biodiversity patterns (Beissinger and Osborne 1982, Blair 1996, Prange and Gehrt 2004, McKinney 2008). Cities represent a mosaic of anthropogenic disturbance types, altering community structure and
species diversity towards the biotic homogenization of natural communities (McKinney 2006, Devictor et al. 2008). In particular, urbanization influences functional ecological interactions (Delgado-V and French 2012) such as pollination (Neil and Wu 2006), seed dispersal (Cheptou et al. 2008), as well as competition and predation among vertebrates (Faeth et al. 2005). Shifts in the frequency and type of interactions can directly and indirectly affect species abundance, community composition, and ecosystem functioning. Of recent interest to ecologists and epidemiologists is the functional influence of urbanization on the interaction structure between hosts and parasites because urban wildlife parasitism holds ecological and public health significance (Daszak et al. 2000, Bradley and Altizer 2007, Brearley et al. 2013).

Urbanization is expected to influence host-parasite interactions by altering the assembly processes and structure of host communities and their associated parasite communities (Patz et al. 2004, Thompson 2013). With relentless growth of cities at a global scale comes decreased natural habitat, increased built impervious surface area, and corresponding human population densities (Alberti 2005, Shochat et al. 2006). These land-use and land-cover changes can cause filtering pressures on wildlife communities whereby species sensitive to disturbance may reduce in abundance, become extirpated, or go locally extinct (Brearley et al. 2013). Conversely, opportunistic host species resistant to disturbance have the capability to persist in human-dominated habitat. Urban species can be highly competitive by utilizing resource-exploitation strategies (Hegglin et al. 2007, Shochat et al. 2010) and behavioural plasticity (McLeery 2009, Sol and González-Lagos 2013) to adapt and reproduce in developed, impervious urban land cover. With the loss of sensitive host species and the proliferation of generalist and synanthropic hosts, the overall composition and structure of host communities, and their associated parasitic infections, should theoretically shift along a gradient of urbanization (Bradley and Altizer 2007).

In practice, quantifying the complex species responses to habitat modification is impeded by a lack of analytical tools and large-scale empirical observations. In particular, complexity arises in understanding multi-host multi-parasite systems as a result of large number of different species that exist within a community and from the diverse nature and unequal strengths of the interactions among them (Poulin 2010, Gonzalez et al. 2011). Quantifying multi-host multi-parasite systems at large scales is challenging on a conceptual level, due to complexities in summarizing multiple parasitic interactions simultaneously over modified landscapes; and at a
practical level, whereby data acquisition of both host and parasite populations over large regions represents significant sampling difficulties (McCallum and Dobson 2002, Caron et al. 2012). These challenges have led to a scarcity in empirical, ecological investigations of multi-host multi-parasite systems (Carlsson-Granér and Thrall 2002, Kamo and Boots 2004, Rigaud et al. 2010, Anholt et al. 2012).

Recently, methods have been developed to allow the quantification of host-parasite interactions at the community scale: biotic communities can be conceptualized as networks of interacting species (i.e., predation, mutualism, parasitism) to reduce the complexities of ecological dynamics (Bascompte 2010). By considering all host and parasite species within a system as a network of interconnected entities, network analysis can quantify nested assembly (Krasnov et al. 2005, Graham et al. 2009), specialization (Vázquez et al. 2005), and structural heterogeneity of host-parasite interactions. Bipartite network graphs can shed light on biogeographical patterns in the structure of networks and whether or not the network properties of given host or parasite species are consistent across disturbance gradients (Tylianakis et al. 2007, Poulin 2010). Therefore, network analysis offers a robust statistical approach to uncover the complexity underlying interactions among multiple hosts and parasites within and between ecological communities.

I asked how the structure of host-parasite interactions changes along a gradient of urban landscape spatial heterogeneity. Using mutualistic bipartite networks, I investigated three objectives using an urban gradient compared to a natural gradient as a proxy of wildlife habitat: (1) quantify and visualize asymmetry in network structure for hosts and parasites at the species and functional level; (2) compare network structure between mammalian versus avian classes at the species level; and (3) quantify relative contribution of individual host and parasite species to overall network structure. For all objectives, I calculated quantitative metrics per bipartite network tested against null models and non-parametric statistics to evaluate the hypothesis that the distribution of asymmetry along the urbanization or natural gradients in host-parasite interaction networks is the result of non-random underlying distributions of infections among species.
It is expected that the frequency distribution of numbers of host species exploited by one or more parasite species is highly right-skewed, with more parasite species exploiting one or a few host species (Poulin 1992, Gregory et al. 1996, Vázquez et al. 2005). Compared to rural sites, I therefore predicted urban sites would express (1) non-random asymmetric distributions of infections due to higher abundance of generalist hosts and higher frequency of density-dependent interspecific interactions between species in cities; (2) greater diversity of parasitic interactions per host due to tolerance of generalist hosts to parasitism; and (3) greater nestedness caused by the filtering of abundant, generalist hosts in cities than less impervious rural or natural sites.

4.3 Methods

4.3.1 Study region

Observed data collected by the Toronto Wildlife Centre in Toronto (Ontario) from the southwestern Ontario region whereby individual animals were brought in by the public to the Centre for examination and possible rehabilitation (2007-2012) (Fig. 4.8.1). The region ranges from rural environments dominated by agriculture to highly urbanized city centres dominated by business, residential, and industrial land use (i.e., Toronto). Spatial locations of each intake individual were recorded as closest road intersection later converted into spatial points.

4.3.2 Landscape composition and urban/natural indices

To calculate a gradient of urbanization and natural habitat, I combined land-cover data using CanMap RouteLogistics Ontario v2013.3, DMTI (2013) and Southern Ontario Land Resource Information System (Smyth 2008) from my sampling region at a 1 km² grid cell (Fig. 4.8.1). The 1 km² cell size is assumed to capture the median home range value for avian and large terrestrial hosts in the region and is a commonly chosen scale of interest in previous studies examining urban community structure (Tewksbury et al. 1998, Rodewald and Bakermans 2006, Giraudeau et al. 2014, Rodewald et al. 2014). Only those cells that intersected points of infected observed individuals were used to extract landscape composition for a total of 699 cells, or ‘sites’. I then calculated landscape composition as percent cover per land-cover factor within each cell using Geospatial Modeling Environment 0.7.2.0 (Beyer 2012) (Table 4.7.1). I extracted a percent value of 11 land-cover types per grid cell: pasture, cropland, treed wetland, coniferous forest, mixed forest, deciduous forest, swamp, open water, urban greenspace, transportation, and built
impervious surface (Table 4.7.1). I also calculated a road-density index by summing the length of road segments contained within a grid cell, and weighted the sum of each road by the average speed limit per segment (Table 4.7.1).

I synthesized land-cover factors and a road-density index into the first two PCs that explained ~61% of the environmental variation cumulatively (see Appendix 4.9.2.2). PC1 represented the urban environmental variation which loaded positively and strongly (component loading > 69%) on the percentage of land covered by pasture, road density, and built impervious surface. I used PC1 as an ‘urban index’ to represent a gradient of increasing urban land cover and land use, ranging from -1.082 to 0.536. PC2 represented natural environmental variation which loaded positively and strongly (component loading > 58%) on the percentage of land covered by deciduous forest, coniferous forest, swamp, and urban greenspace. I used PC2 as a natural index to represent a gradient of decreasing natural cover per sampling unit, ranging from -0.725 to 0.580. This natural index was used for comparative network analyses against my hypotheses of urbanization effects on host-parasite interaction structure. Sampling-unit PC-values per urbanization or natural index were divided using natural breaks (jenks) to categorize sites in equal representations of the land-cover gradients and not biased to urbanized cover. Sites were classed as one of five urban or natural categories of land use to compare networks between rural, exurban, suburban, urban, and high-urban land cover categories (Hasse and Lathrop 2003, Atwood et al. 2004, Hansen et al. 2005). For the natural gradient, I correspondingly defined each category in terms of natural cover: full, high, moderate (mod.), low, or no cover.

4.3.3 Wildlife host and parasite data

All animals were examined upon intake by clinical veterinarians using standard intake protocols for parasitic infections, including ectoparasites, endoparasites (via fecal examination) and evidence of viral disease based on external examination of condition. Consistent protocols were used per host species, developing a controlled dataset of parasitism among all individuals examined.

I addressed detectability at both the level of the host and of the parasite. For hosts, I selected only parasitized individuals \( n = 3\,073 \) from the original dataset of total sampled individuals \( n = 26\,101 \) representing a sub-sampling of all observed wildlife species. For only
some infections (i.e., rabies, canine distemper virus, and heavy mange intensity) do I assume individuals are more likely to be sampled by the public due to apparent pathologic conditions and/or unusual behaviour caused by infection. To determine if there was a relationship between the number of hosts and human density, I performed a regression of number of remaining infected-host observations explaining variation in human density. While the regression was significant \((p < 0.0001)\) due to the large sample size, the percentage of variance explained was almost nil \((\text{adj.}-R^2 = 0.03)\). Therefore based on the result of this regression, I assumed that my samples were not confounded by a gradient of population density. All host species were then categorized according to functional group, generally grouped by taxonomic order.

For parasite detectability, I only selected observed infections if those parasitic infections were consistently tested upon intake examination by TWC veterinarians as part of standardized protocols (Appendix 4.9.1.2). Some parasitic infections required confirmation from the Canadian Wildlife Health Cooperative (CWHC) at Guelph University (i.e., rabies, leptospira, white nose syndrome). All parasite species were then categorized according to functional group, generally grouped by taxonomic order.

4.3.4 Bipartite networks and metrics

Bipartite networks are effective tools to investigate patterns in ecological parasitology because they provide both visual representation of complex ecological systems that influence disease cycles and a formal mechanism to test how the diversity of parasites and diseases are regulated in natural and modified ecosystems (Bascompte 2010, Poulin 2010). Variation in detailed structure of bipartite networks can shed light on how host-parasite communities are assembled made evident by uneven distributions among links (interactions) and nodes (species) (Vázquez et al. 2005, Canard et al. 2014). Therefore, I used bipartite networks to address my objectives.

For objective (1) I constructed a quantitative bipartite network per category of the urban and natural indices, including all observations of hosts and parasites to the level of taxonomic order (as a representation of functional host and parasite groups). A regional bipartite network was then constructed for the entire region at the species- and order-level (referred to herein as functional-group level) taxonomy to compare the change in interactions among host and parasite species and functional groups as compared to a total bipartite network for the entire region. For
objective (2), networks were further subdivided to compare parasitic interactions between mammalian and avian hosts across the urban and natural gradients. For objective (3), I calculate species-level network metrics using networks developed for objective (1). All graph visualization and analyses were performed using the bipartite library in R (Dormann et al. 2009, Venables and Smith 2011).

I summarized the structure of all bipartite networks using quantitative network metrics to evaluate host-parasite interaction asymmetry along the environmental gradients (Tylianakis et al. 2010). I defined asymmetry as the average mismatch between a focal species’ effect on its interaction partners and the reciprocal effect of the interaction partners on the focal species (Vázquez et al. 2007). Quantitative, weighted metrics calculate the relative contribution of host or parasite interactions taking into account the number of registered observations of a particular interaction (Bersier et al. 2002, Barrat et al. 2004, Banašek-Richter et al. 2009). Quantitative bipartite metrics are more robust than qualitative metrics because they evaluate the uneven magnitudes of true interactions in nature and are less sensitive to the sampling effort used to document interactions. First, I calculated connectance – a standardized measure of species combinations – to compare the overall network structure between functional and species-level networks. Then, asymmetry in network structure was characterized across urban and natural gradients using the following quantitative bipartite network metrics (Tylianakis et al. 2007, Dormann et al. 2009): (a) generality and (b) vulnerability, to quantify the weighted mean effective number of hosts per parasite (generality) and parasites per host (vulnerability), weighted by their marginal totals (row or column sums); (c) H2, to quantify a weighted network-level measure of specialization; and (d) Shannon’s diversity to quantify interaction diversity of interaction metrics described above.

I quantified (e) nestedness, a measure of species organization across sets of species assemblages that define the community, using a weighted-interaction nestedness estimator (WINE, Galeano et al. (2009)) which takes into account the weight or intensity of each observed interaction. A ‘WINE’ value of 0 represents a random distribution while 1 represents maximal nestedness. Significance was tested against a null model that constrains matrix fill to observed values and retains the distribution of number of events in the links whereby z-scores are calculated on how different the nestedness values deviate from average nestedness of randomly
generated matrices. To account for inflation of nestedness values caused by passive sampling (i.e., poorer likelihood to sample rare than dominant species in a given area) and dominant interactions, I also calculated nestedness for only dominant communities, defining dominance as host species that occupy all land-cover categories.

Finally, for objective (3) I evaluated host and parasite species’ relevance relative to the whole host and parasite community calculated using species strength. Species strength is quantified by the sum of dependencies of each species (Bascompte et al. 2006) per individual host and parasite functional groups to account for the pattern of total skewed dependencies and strong asymmetries that scale up to account for properties at the community level (Bascompte et al. 2006).

4.3.5 Null models and statistical analyses
To test asymmetric interactions and host specificity along an urban gradient, I used a null model of quantitative bipartite interactions (Vázquez et al. 2007, Dormann et al. 2009) constrained by the same marginal totals and connectance as the original observed network. Null models were implemented to quantify significant deviations of observed network structure values from expected means developed from a benchmark provided by randomized null models (1000 repetitions). I used Fisher’s tests for significance of difference between null and observed produced $p$-values per metric per network. I compared the variation in network structure between urban and natural gradients (objective 1) and between mammalian and avian host classes (objective 2) using the non-parametric Kruskal-Wallis one-way analysis of variance. For measures of nestedness, $p$-values were calculated as the probability of $z$-scores (of weighted-interaction nestedness values) equal to or greater than $Z$. $Z$ values below -1.65 or above 1.65 indicate approximate statistical significance at the 5% error level (one-tailed test). Values of $p<0.05$ indicate a significantly nested dataset.

4.4 Results

4.4.1 Objective 1: Urban versus natural gradients
I developed host-parasite quantitative bipartite networks using all observed interactions over the southwestern Ontario region (Fig. 4.8.2). Forty-four mammalian and avian species were
observed with at least one infection of the 23 observed parasite species. Distribution of interactions was highly skewed in all networks (Fig. 4.8.3). All network metrics were significant from expected means calculated by null models, demonstrating significant asymmetry in all networks (Fig. 4.8.4). Skewed distributions were also observed in all natural classes to different extents (Figs. 4.8.3 and 4.8.4). Along the urban gradient, at the functional level, the greatest realized proportion of possible links (i.e., connectance) was represented in the urban network (connectance = 0.560) whereas the lowest realized proportion of links was observed in the rural network (connectance = 0.345). Along the natural gradient, connectance was greatest in the no-cover network (0.60) whereas proportion of links is lowest in the full-cover network (0.417). Considering functional-groups, a core set of few generalist hosts (i.e., pigeons and doves; small carnivores; small rodents and moles) were parasitized by a core set of parasites (i.e., viral infections; ectoparasites). Occupancy of functional groups was inconsistent across the urban gradient as compared to the total regional set of interactions.

Nearly all quantitative bipartite metrics differed significantly from null models calculated per urban or natural category (Table 4.7.2, Fig. 4.8.4). Kruskal-Wallis tests of non-parametric one-way analyses of variance demonstrated no significant differences between the variability of network metrics over urban and natural gradients. Generality – a measure of the average number of hosts a given parasite species infects – decreased with increasing urbanization (3.958 – 2.161) to a greater extent than decreasing natural cover (species-level: 2.031 – 1.473; functional-level: 3.221 – 2.521). Conversely, vulnerability – a measure of the average number of parasites a given host is infected by – generally increased with urbanization. Additionally, vulnerability was nearly as high in suburban networks as it was in high-urban networks. Moderate-cover and no-cover sites also reflected relatively high vulnerability. Suburban and high-urban networks also represent the greatest Shannon’s diversity of interactions relative to all other high or low-urban or natural networks.

The nestedness value for the true regional host-parasite community (WINE = 0.483) was significantly larger than the mean values of the randomized community generated by the null models, but weighted nestedness scores increased from rural to high-urban networks (0.180 – 0.412) (Table 4.7.3, Fig. 4.8.5). Conversely, nestedness did not increase along the natural gradient. Nestedness calculated in the high-cover network (0.440) was nearly identical to that of
the no-cover network (0.456); whereas the low-cover network demonstrated the lowest
nestedness (0.276). Considering only dominant host species, nestedness differed from totally-
occupied host species (WINE = 0.298, z-score = 2.334, p = 0.009) as compared to nestedness
calculated for all host species (WINE = 0.483, z-score = 8.209, p <0.001) (Table 4.7.3). Along
the urban gradient, totally-occupied host-species nestedness generally increased as well (though
not necessarily linearly) in a parallel manner as that calculated for all host species (except high
nestedness in the rural category for fully-occupied hosts). The character of species interactions
did not differ dramatically for host and parasite species between the dominant nestedness values
versus all-species nestedness values (Appendix 4.9.2.1).

4.4.2 Objective 2: Mammalian versus avian hosts

I compared interactions between mammalian versus avian taxonomic groups. Asymmetric
bipartite network structure was observed in avian and mammalian interactions across the urban
gradient. In contrast to the consolidated host-parasite community (Objective 1, Fig. 4.8.3),
significant differences were observed in distribution of host-parasite interactions between
mammalian- versus avian-host networks (Fig. 4.8.6). Avian interactions demonstrate greater
generality, vulnerability, and Shannon diversity than mammalian interactions (Table 4.7.4, Fig.
4.8.7). Along both urban and natural gradients, vulnerability (Kruskal-Wallis $\chi^2 = 6.81$, df = 1, p-
value < 0.01), H2 specialization (Kruskal-Wallis $\chi^2 = 8.64$, df = 1, p-value = 0.00016), and
Shannon diversity (Kruskal-Wallis $\chi^2 = 3.94$, df = 1, p-value = 0.047) differed significantly.
Generality (Kruskal-Wallis $\chi^2 = 0.27$, df = 1, p-value = 0.60) and weighted-nestedness (Kruskal-
Wallis $\chi^2 = 0.04$, df = 1, p-value = 0.83) did not differ significantly between mammalian and
avian networks. Along the urban gradient, generality increases from rural to high-urban
mammalian networks, whereas maximum vulnerability (5.89) in avian interactions was observed
in the suburban and moderate-cover networks. Then along the natural gradient, mammalian and
avian interactions are relatively unchanged; whereas vulnerability varies more dramatically for
avian hosts. In many cases, H2 specialization was highest in the urban and low-cover networks
for both avian and mammalian networks, not necessarily in the most urbanized network.
4.4.3Objective 3: Species-level interactions

For host species, pigeons and doves represented the greatest avian-host species strength (3.33) whereas small rodents and moles represented the greatest mammalian-host species strength (1.04) (Fig. 4.8.8). Species strength was asymmetrical across all urban categories, but to a greater magnitude in high-urban network (Fig. 4.8.9). Species strength values for pigeons and doves increased along the urbanization gradient and represent the greatest species strength in the high-urban network. Songbirds have a relatively stronger contribution to network structure in the rural network which decreases with the urbanization gradient. Small carnivores contribute to network structure in exurban and suburban structure.

Considering parasite species, maximum species strength was observed in ectoparasites (5.89), followed by viral parasites (3.69) in the regional network (Fig. 4.8.8). Asymmetrical network complexity was consistently high in these dominant functional groups of parasite species (Fig. 4.8.9). Along the urban gradient, ecotoparasites maintained the greatest values of species strength but was variable in value. Maximum ectoparasite species strength was observed in the suburban areas. Viral species strength values were consist and relatively invariable compared to other parasite functional groups. The greatest magnitude of viral and ectoparasitic parasite species strength was observed in the suburban network. The greatest species strength per functional parasite group was observed in the suburban network. Nematode dependencies increased from rural to urban networks (0.33 – 1.06).

4.5Discussion

4.5.1Overall findings

Urban environments have been shown to alter ecological processes that influence the transmission of infectious diseases in wildlife (Bradley and Altizer 2007, Delgado-V and French 2012). As urbanization dramatically alters the diversity and abundance of natural populations, it is expected that the composition of wildlife host communities, and their associated parasites, will also shift (Shochat et al. 2006, Devictor et al. 2008). While asymmetry is generally found in many host-parasite interaction networks (Vázquez and Aizen 2004, Thébault and Fontaine 2008), urbanization is hypothesized to either magnify or temper the extent to which host-parasite interactions are unevenly distributed depending on the relative abundance of host populations,
parasite prevalence within those populations, and environmental conditions that favour transmission within and between host populations.

In this study, I provide evidence that habitat modification by urbanization alters the interaction structure of host-parasite relationships. I showed that network asymmetry and nested-hierarchical distributions of wildlife parasitism in more urban than rural areas of southwestern Ontario. In particular, I identified three generalized patterns. First, I found consistent asymmetric structure in regional, mammalian, and avian host communities, emphasizing skewed parasitic interactions between communities of wildlife hosts and parasites (discussed in section 4.5.2). Second, in highly-urbanized areas, I observed high specialization of host-parasite interactions among a smaller subset of host and parasite species, indicating a potential selection of generalist hosts and multi-host parasites in highly-urbanized environments (discussed in section 4.5.3). Third, suburban (moderate natural cover) and high-urban (no cover) networks demonstrated the greatest connectance, generalized breadth of parasitism among hosts, and diversity of host-parasite interactions (discussed in section 4.5.4). This collective evidence stresses the effect of urban, and especially suburban, land cover on the structuring processes of host communities and indirect effects on frequency of interactions among those communities. My results therefore corroborate previous evidence suggesting that urban development affects the community dynamics of disease systems in natural populations.

4.5.2 Asymmetric structure in host-parasite interaction networks

I observed structural heterogeneity among host-parasite interactions in all regional, mammalian, and avian host-parasite networks whereby a nested, core group of generalist hosts commonly interacts with a nested group of generalist or specialist parasites. Consistency in this gradient was observed in all bipartite networks which implies common ecological assembly processes among this regional community of hosts. These findings agree with skewed frequency distributions commonly observed in plant-animal networks (Bascompte et al. 2003, Vázquez and Aizen 2004) and in host-parasite networks (Vázquez et al. 2007, Poulin 2010, Tylianakis et al. 2010). Flexible life-history strategies among these core species likely drive this commonly observed dominant hierarchical community structure. Many urban hosts have adapted generalist traits, often identified by invasive and exotic species, relate to higher host abundances, rapid host

In tandem, generalist parasites that exploit multiple host species should thrive in high-density generalist host populations (Holt et al. 2003, Pedersen and Fenton 2007, Rigaud et al. 2010). I observed these patterns in my bipartite models: pigeons and doves (e.g., rock pigeons), small carnivores (e.g., raccoons and striped skunks), and small rodents and moles (e.g., eastern gray squirrels) represent a significant portion of the host-level interactions. At the parasite level, viral (e.g., AMPV-1, canine distemper virus) and ectoparasitic (e.g., fleas, mites) parasite groups represented the greatest diversity of hosts and frequency of infections. This core group of generalist hosts and parasites represent a nested dominance hierarchy which may act as a highly cohesive core of the host and parasite community on which other specialist hosts or parasites depend (Bascompte et al. 2003). Ecological bipartite networks are often described in their robustness and resilience dependent on critical interactions between species that provide critical ecosystem services (Bascompte 2010, Poulin 2010). Therefore, a nested, dense core of host-parasite interactions characterized by opportunistic host and parasite species may provide a functional role to support parasitic interactions under highly disturbed environments.

4.5.3 Urbanization alters the structure of host-parasite interactions

I argue that urbanization has a strong and functional influence on the structural heterogeneity of host-parasite interactions. My quantitative bipartite network analyses provide evidence that with increasing urban land cover, and decreasing natural vegetative cover, fewer host species share greater average frequency and diversity of parasitic interactions. Nestedness generally increased with increasing urban cover. I also observed similar patterns in mammalian and avian bipartite network structure. This combined evidence suggests that urbanization may reduce the overall number of host species but bequeaths a strengthening in the cohesion of the remaining core group of generalist host-parasite interactions.

I reason that filtering pressures caused by urban development select for resilient hosts capable of maintaining infections while persisting in disturbed habitat. A major hypothesis of urban ecology is that species richness declines with increasing urban cover (McKinney 2006). This hypothesis follows that urban environments may favour functional traits for species that
colonize readily in limited, nutrient-poor environments. Functional traits such as behavioural and
dietary plasticity allow for synanthropic, facultative species to thrive in high human densities
(Shochat et al. 2006). Conversely, species sensitive to highly-disturbed environments (Marzluff
et al. 2007, Francis et al. 2009) or shifts in food-web structure (Rodewald et al. 2014) may go
locally extinct or become isolated in habitat fragments surrounded by urban matrix (McKinney
and Lockwood 1999). Loss of sensitive host species likely contributes to the structural
heterogeneity and cohesive core of host-parasite networks because host-species losses should
reduce the number of potential hosts a given parasite can infect.

I extend this argument to suggest that urban-influenced host-parasite asymmetry may
affect disease transmission dynamics in wildlife populations. Indeed, I found evidence that
directly-transmitted parasites (viral- canine distemper virus, avian paramyxovirus; ectoparasitic:
fleas, ticks, mites) were more likely to be shared by species and occur at greater frequencies in
high-density urban or suburban than rural environments. This observation complements the
hypothesis that highly residential and impervious habitat favours density-dependent transmission
within and between species. High densities of generalist hosts should favour frequent
transmission of directly-transmitted parasites. As urbanization likely directly selects for
generalist host species, particular environmental characteristics in urban habitat may indirectly
select for parasite species to persist within high-density host populations at a greater prevalence.
For example, Raccoons that persist at high densities should experience more frequent contacts
through aggressive encounters or at resource aggregation sites (i.e., garbage cans). Intra-specific
contacts may transmit viral parasites such as canine distemper virus or rabies more readily
between one another (Wright and Gompper 2005). Therefore, specific conditions found in urban
environments may favour inter- and intra-specific transmission events, such as crowded
conditions in isolated habitat, roosting sites, or resource aggregation sites (Prange et al. 2004,
Wright and Gompper 2005).

Likewise, environmentally-mediated parasites (i.e., bacteria, protozoa) which often
contaminate habitats (i.e., nests, feces) may persist at greater abundance in urban than rural areas
(Giraudeau et al. 2014). Host-contact frequency to free-living parasitic stages may be more
common in these urban aggregated sites relative to rural or pristine habitat. For example, pigeons
that deposit bacterial parasites pose risks to other pigeons or birds sharing roosts in cities.
Raccoons that deposit the zoonotic parasite, *Baylisascaris procyonis* in feces pose environmental infection risks to other raccoons, other small carnivores, and potentially to humans, though recorded raccoon-human infections are considered very low (Sorvillo et al. 2002, Page et al. 2009). Furthermore, horizontally-transmitted parasites such as vector-borne parasites, bacterial parasites, or nematodes rely on contact with arthropods or free-living stages in the environment. Crowding may boost contamination levels and pose further infection risks for urban dwellers. Thus, shared habitat in urban areas may pose more environmentally-mediated exposure risks to wildlife than rural areas potentially altering the mechanisms that influence transmission dynamics within and between populations (Watts et al. 2015). These risks are not limited to wildlife species, and domestic animals or humans may also be at risk of environmental exposure to infection (Robertson et al. 2000, Thompson 2013, Mackenstedt et al. 2015).

Mammalian and avian infections differed in their response to urban landscapes. I observed differences in structural heterogeneity between mammalian versus avian networks along the urban gradient. In particular, while the trends in asymmetry were similar, the magnitude and extent of asymmetry was much greater in the avian bipartite networks compared to mammalian networks. For example, pigeons and doves maintain infections from every functional parasite group, and the majority of those infections are viral (namely AMPV-1), ectoparasitic, and protozoan in nature. In contrast, songbirds represent the greatest species strength in rural environments, a diverse group of avian hosts that may persist at lower densities in urban areas. Small carnivores (i.e., raccoons) tend to maintain viral infections; and small rodents and moles maintain ectoparasitic infections. These dominant host functional groups were also critical in maintaining host-parasite structural heterogeneity, but avian groups contributed differently than mammalian groups. Avian and mammalian habitat utilization is likely different (Croonquist and Brooks 1991) and probably corresponds to differences in transmission dynamics (Anderson and Magnarelli 1984). For example, songbirds have a strong diversity of parasitism in exurban or rural areas than urban areas, potentially linked to high inter-specific contact rates at resource sites such as bird feeders (Hall et al. 2007).

Structural heterogeneity in urbanized host-parasite networks may also support the persistence of wildlife reservoir populations. Urban synanthropic species have been shown to tolerate parasitism (Lehrer et al. 2010, Giraudeau et al. 2014), or experience minor expression of
disease symptoms caused by parasitism (Suzán and Ceballos 2005, Vander Wal et al. 2014), potentially favoring the persistence of reservoir host populations. Reservoirs of infection can be ecologically complicated structures comprising one or more interacting populations or species (Woolhouse et al. 2001, Haydon et al. 2002). Maintenance of multi-host parasite diversity within high-density host populations may produce conditions favourable for disease-reservoir populations. In my results, I identified high generality in number of hosts infected by a given parasite, in suburban and high-urban networks. Thus, while characterizing a defined reservoir population remains a challenge (Viana et al. 2014), it is intuitive to consider generalist host populations with greater frequency and diversity of infections in suburban or urban land cover as potential reservoirs. Wild reservoir populations are particularly critical in maintaining transmission dynamics, and urban structuring may facilitate the spillover transmission of parasites from maintenance reservoir host populations to susceptible target populations. Identifying and managing reservoir populations remains a practical and conceptual challenge (Viana et al. 2014) but is critical in monitoring and preventing zoonotic risks in human-dominated landscapes.

4.5.4 Suburban habitat may favour host-parasite interaction asymmetry

I found evidence that suburban habitat may have a particular role in structuring host-parasite interactions. In many of my networks, the suburban category expressed similar or maximum attributes of network structure (i.e., vulnerability, Shannon diversity, weighted nestedness) compared to the rural or highly-urbanized categories. Avian networks in particular had maximal vulnerability and diversity metric values in the suburban category. Average species strength, a measure of the sum of dependences per host or parasite, was also greatest in suburban networks for both hosts and parasites at the functional-level. These results reflect patterns observed in similar studies: Deplazes et al. (2004) identified spatial-overlap of intermediate and definitive hosts of the zoonotic fox tapeworm, *Echinococcus multilocularis*, in urban periphery of Zurich (Switzerland); feces infected with raccoon roundworm (*Baylisascaris procyonis*) was in greater densities in per-urban areas (Bauer 2013); and Lyme disease prevalence was found in suburban-dwelling birds in California (Newman et al. 2015) and in fragmented suburban residential white-footed mouse populations in Connecticut (Allan et al. 2003). These patterns of disease
prevalence are limited to single disease systems yet complement my observed patterns of host-parasite interaction diversity in suburban areas.

I reason that suburban patterns of host-parasite interaction diversity could be due to intermediate levels of disturbance. The intermediate disturbance hypothesis has a long history in ecology, positing that local species diversity is maximized when ecological disturbance is neither too rare nor too frequent (Menge and Sutherland 1987, Wilkinson 1999). It is assumed that urban landscapes cause the fragmentation, reduction, and loss of vegetative habitat, causing widespread species declines (Lizée et al. 2011). It should follow that loss of natural cover will also influence the abiotic conditions that favour or inhibit disease transmission. For example, specialist parasites tightly linked to particular host species may also become extirpated or locally extinct if the host species is sensitive to habitat loss, competitive exclusion, or urban disturbances (Bradley and Altizer 2007, Rodewald et al. 2014). Similarly, free-living parasite stages (i.e., some nematode, bacterial, or protozoan parasites), arthropod vectors, or ectoparasites are hypothesized to decrease in abundance with decreased vegetation (Evans et al. 2009, Delgado-V and French 2012). Urban pesticides and arthropod management practices could have additional extinction pressures on free-living parasite species, especially arthropods (Knudsen and Slooff 1992, Colwell et al. 2011). Thus, suburban environments that experience only moderate levels of fragmentation or forest loss may support a greater diversity of specialist hosts (and possibly their specialist parasites) while also supporting a core of generalist, synanthropic hosts.

Moreover, I observed no significant differences in the variability of host-parasite network structure between urban and natural gradients. This result demonstrates how the loss of natural cover and the increase of built cover has a similar effect on the structural heterogeneity of host-parasite interactions. This consolidated evidence supports my argument that intermediate-disturbed suburban environments may have a distinctive role in structuring host-parasite interactions by maintaining wide niche breadth host and parasite communities via diverse habitat and resource conditions.

4.5.5 Assumptions and limitations

The opportunistic sampling procedure of my dataset presents minor sampling biases at the level of the host and of the parasite. While I addressed these biases quantitatively (i.e., use of infected-
only observations, use of quantitative – as opposed to qualitative – metrics, and the selection of parasite species that are consistently tested for by TWC veterinarians), I recognize that the contributions of particular hosts (rock pigeons, raccoons, eastern gray squirrels) and parasites (AMPV-1, canine distemper virus, mites) to network structure could potentially bias results. However, these species thrive in many areas of southwestern Ontario and the subsampled data likely reflects true abundances and parasitic infections persisting in the region.

Furthermore, in some cases, diagnostics of parasites were limited to genus-level, not species-level. Particular diagnostics are required for many blood-borne or gastrointestinal parasites that likely exist in the region’s wildlife populations. For example, I was required to categorize fleas at the species-level given the data available although it is known that fleas as a group likely represent many different flea species. Similarly, Baylisascaris procyonis, commonly known as raccoon roundworm, is found in many raccoons in the region; yet, my results demonstrate a relatively lower B. procyonis prevalence in raccoons. This suggests that there may be inconsistencies in diagnostics for particular parasitic infections. However, I believe the value of such large-scale observations of multi-host multi-parasite interactions is powerful in conjunction with network-based analyses to be used as an indicator of wildlife-parasite interactions along urban and natural gradients.

4.6 Conclusions

The ecological consequences of tightly-linked host-parasite interaction structure in suburban and urban habitat is critical for the role of disease in species abundance, distribution, and extinction risks. Indeed, network structure and stability can be altered by various global environmental changes, and these alterations may have important ecosystem-level consequences (Tylianakis et al. 2010). It is critical that we continue to investigate how cities shape species interactions, community assembly patterns, and biodiversity. My argument that urbanization influences transmission dynamics by structuring host-parasite interactions stresses to the importance of urban landscapes in infectious disease ecology. The possibility that cities favour the persistence of reservoir populations merits future research as spillover risks have important veterinary and public health consequences. While I did not quantify zoonotic risks explicitly, I found greater prevalence of West Nile Virus, Leptospira spp., and definitive raccoon roundworm in the urban.
network where zoonotic risks posed to humans are greater by proxy. My work therefore fuels future targeted monitoring for zoonotic parasite prevalence in suburban and urban areas. With increasing rates of urbanization comes greater potential for domestic animal-wildlife and human-wildlife interactions. As global urbanization increases, wildlife-domestic and wildlife-human interactions are also expected to increase. Therefore, it is critical that parasitism is monitored in those wildlife host populations that are more likely to contact humans in suburban and urban areas.

4.7 Tables
Table 4.7.1. Description of land-cover metrics selected for landscape analysis. Land-cover variables were included in the development of principle components-based indices to represent the urbanization and natural gradients (PC1 and PC2, respectively). Data was accessed from Southern Ontario Land Resource Information System (SOLRIS), developed in April 2008. All variables, with the exception of road-density were extracted as percent cover per cell, and transformed by the ln(p/(1-p)) transformation per variable.

<table>
<thead>
<tr>
<th>Category</th>
<th>Variables</th>
<th>Description</th>
<th>MMU (Ha.)</th>
</tr>
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<tbody>
<tr>
<td>Agriculture</td>
<td>* Pasture</td>
<td>Areas of idle land characterized by hay or pasture or by a vegetative component in spring imagery (not within a woodland, wetland, or cropland class)</td>
<td>0.5</td>
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<tr>
<td></td>
<td>* Cropland</td>
<td>Areas considered agricultural production characterized by bare fallow fields in spring imagery</td>
<td>0.5</td>
</tr>
<tr>
<td>Vegetation</td>
<td>Grassland</td>
<td>Native grasses, shrub; herbaceous vegetation with 20% minimum 20% ground cover</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>Deciduous forest</td>
<td>&gt; 60% tree cover, deciduous tree species represent &gt;75% of the canopy cover</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>Coniferous forest</td>
<td>&gt; 60% tree cover, coniferous tree species represent &gt;75% of the canopy cover</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>Mixed forest</td>
<td>&gt; 60% tree cover, deciduous and coniferous tree species must both represent &gt;25% of the canopy cover</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>Swamp</td>
<td>Tree or shrub cover &gt;25% - dominated by hydrophytic shrub and tree species, water table seasonally or permanently at, near, or above substrate surface</td>
<td>0.5</td>
</tr>
<tr>
<td>Urbanization</td>
<td>Urban greenspace</td>
<td>Areas of vegetation found completely within the urban/infrastructure class (urban class not utilized in analyses)</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>Built-up Area – Pervious</td>
<td>Urban recreation areas, e.g., golf courses, playing fields</td>
<td>0.25</td>
</tr>
<tr>
<td></td>
<td>Built-up Area – Impervious</td>
<td>Residential, industrial, commercial, and civic areas</td>
<td>0.25</td>
</tr>
<tr>
<td>Transportation</td>
<td>Transportation</td>
<td>Highways, roads</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>Road density</td>
<td>Length of highways and roads per cell, multiplied by associated speed limit</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>Parks and Recreation</td>
<td>Cover designated as community, school, or public recreation</td>
<td>0.5</td>
</tr>
<tr>
<td>Hydrology</td>
<td>Open water</td>
<td>Large hydrological features, no macrophyte, vegetation, trees, or shrub cover</td>
<td>0.5</td>
</tr>
</tbody>
</table>

* Additional features (i.e., pasture, cropland) were developed by the Ontario Ministry of Natural Resources (OMNR) of the Government of Ontario for the 2012 Greenbelt Management Strategy (GBS).
Table 4.7.2. Network metrics for host-parasite interaction structure at the functional and species level along the urban and natural gradients. Networks were calculated per urban or natural values calculated using PCA-values per sampling unit.

<table>
<thead>
<tr>
<th>Urban Gradient</th>
<th>Region</th>
<th>-0.108</th>
<th>-0.52</th>
<th>0</th>
<th>0.203</th>
<th>0.54</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rural</td>
<td>Exurban</td>
<td>Suburban</td>
<td>Urban</td>
<td>High-urban</td>
<td></td>
</tr>
<tr>
<td><strong>Functional-level</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generality</td>
<td>3.042</td>
<td>3.958</td>
<td>3.558</td>
<td>2.949</td>
<td>2.160</td>
<td></td>
</tr>
<tr>
<td>Vulnerability</td>
<td>2.547</td>
<td>1.630</td>
<td>1.967</td>
<td>2.647</td>
<td>2.307</td>
<td>2.701</td>
</tr>
<tr>
<td>H2 specialization</td>
<td>0.390</td>
<td>0.612</td>
<td>0.402</td>
<td>0.382</td>
<td>0.476</td>
<td>0.410</td>
</tr>
<tr>
<td>weighted nestedness</td>
<td>0.642</td>
<td>0.533</td>
<td>0.350</td>
<td>0.577</td>
<td>0.600</td>
<td>0.715</td>
</tr>
<tr>
<td>Shannon diversity</td>
<td>2.381</td>
<td>2.392</td>
<td>2.277</td>
<td>2.550</td>
<td>2.301</td>
<td>2.082</td>
</tr>
<tr>
<td><strong>Species-level</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generality</td>
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<td>2.030</td>
<td>2.07</td>
<td>2.293</td>
<td>1.792</td>
<td>1.472</td>
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<tr>
<td>Vulnerability</td>
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<td>1.794</td>
<td>2.016</td>
<td>3.701</td>
<td>3.199</td>
<td>3.753</td>
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<td>H2 specialization</td>
<td>0.749</td>
<td>0.815</td>
<td>0.712</td>
<td>0.717</td>
<td>0.787</td>
<td>0.811</td>
</tr>
<tr>
<td>weighted nestedness</td>
<td>0.480</td>
<td>NA</td>
<td>0.179</td>
<td>0.403</td>
<td>0.379</td>
<td>0.402</td>
</tr>
<tr>
<td>Shannon diversity</td>
<td>2.986</td>
<td>2.776</td>
<td>2.876</td>
<td>3.187</td>
<td>2.877</td>
<td>2.553</td>
</tr>
<tr>
<td>Natural Gradient</td>
<td>Region</td>
<td>-0.73</td>
<td>-0.34</td>
<td>0</td>
<td>0.31</td>
<td>0.58</td>
</tr>
<tr>
<td></td>
<td>Full-cover</td>
<td>High-cover</td>
<td>Mod.-cover</td>
<td>Low-cover</td>
<td>No-cover</td>
<td></td>
</tr>
<tr>
<td><strong>Functional-level</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generality</td>
<td>3.042</td>
<td>3.220</td>
<td>3.192</td>
<td>3.248</td>
<td>2.870</td>
<td>2.520</td>
</tr>
<tr>
<td>Vulnerability</td>
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<td>2.294</td>
<td>1.920</td>
<td>2.679</td>
<td>2.027</td>
<td>2.663</td>
</tr>
<tr>
<td>H2 specialization</td>
<td>0.390</td>
<td>0.449</td>
<td>0.518</td>
<td>0.366</td>
<td>0.525</td>
<td>0.421</td>
</tr>
<tr>
<td>weighted nestedness</td>
<td>0.642</td>
<td>0.536</td>
<td>0.698</td>
<td>0.719</td>
<td>0.374</td>
<td>0.707</td>
</tr>
<tr>
<td>Shannon diversity</td>
<td>2.381</td>
<td>2.384</td>
<td>2.276</td>
<td>2.449</td>
<td>2.237</td>
<td>2.239</td>
</tr>
<tr>
<td><strong>Species-level</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generality</td>
<td>2.059</td>
<td>2.176</td>
<td>1.855</td>
<td>1.802</td>
<td>1.750</td>
<td>1.721</td>
</tr>
<tr>
<td>Vulnerability</td>
<td>3.541</td>
<td>3.080</td>
<td>2.475</td>
<td>3.569</td>
<td>2.385</td>
<td>3.750</td>
</tr>
<tr>
<td>H2 specialization</td>
<td>0.749</td>
<td>0.698</td>
<td>0.799</td>
<td>0.771</td>
<td>0.792</td>
<td>0.785</td>
</tr>
<tr>
<td>weighted nestedness</td>
<td>0.482</td>
<td>0.442</td>
<td>0.340</td>
<td>0.298</td>
<td>0.277</td>
<td>0.461</td>
</tr>
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<td>2.986</td>
<td>3.074</td>
<td>2.800</td>
<td>2.995</td>
<td>2.715</td>
<td>2.745</td>
</tr>
</tbody>
</table>
Table 4.7.3. Weighted nestedness scores for all hosts; only dominant species representing >5% of observed infections); and host species that occupy all urban categories. A ‘WINE’ value of 0 represents a random distribution while 1 represents maximal nestedness.

<table>
<thead>
<tr>
<th>All Hosts</th>
<th>WINE</th>
<th>z-score</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Urban Gradient</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Region</td>
<td>0.483</td>
<td>8.209</td>
<td>1.11E-16</td>
</tr>
<tr>
<td>Rural</td>
<td>0.180</td>
<td>2.897</td>
<td>0.0018</td>
</tr>
<tr>
<td>Exurban</td>
<td>0.181</td>
<td>2.938</td>
<td>0.0016</td>
</tr>
<tr>
<td>Suburban</td>
<td>0.406</td>
<td>6.082</td>
<td>5.9E-10</td>
</tr>
<tr>
<td>Urban</td>
<td>0.375</td>
<td>5.521</td>
<td>1.68E-08</td>
</tr>
<tr>
<td>High-Urban</td>
<td>0.411</td>
<td>5.617</td>
<td>9.67E-09</td>
</tr>
<tr>
<td><strong>Natural Gradient</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Region</td>
<td>0.483</td>
<td>7.891</td>
<td>1.44E-15</td>
</tr>
<tr>
<td>Full Cover</td>
<td>0.440</td>
<td>7.119</td>
<td>5.42E-13</td>
</tr>
<tr>
<td>High Cover</td>
<td>0.340</td>
<td>4.966</td>
<td>3.40E-07</td>
</tr>
<tr>
<td>Mod. Cover</td>
<td>0.301</td>
<td>4.335</td>
<td>0.000007</td>
</tr>
<tr>
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<td>0.275</td>
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<td>0.00005</td>
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<tr>
<td>No Cover</td>
<td>0.456</td>
<td>6.689</td>
<td>1.11E-11</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Co-occupied Host Species</th>
<th>WINE</th>
<th>z-score</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Urban Gradient</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Region</td>
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<td>0.0098</td>
</tr>
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<td>Rural</td>
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<tr>
<td>Exurban</td>
<td>0.225</td>
<td>2.769</td>
<td>0.0028</td>
</tr>
<tr>
<td>Suburban</td>
<td>0.474</td>
<td>5.041</td>
<td>2.30E-07</td>
</tr>
<tr>
<td>Urban</td>
<td>0.408</td>
<td>4.313</td>
<td>8.04E-06</td>
</tr>
<tr>
<td>High-Urban</td>
<td>0.510</td>
<td>5.354</td>
<td>4.28E-08</td>
</tr>
<tr>
<td><strong>Natural Gradient</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Region</td>
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<td>2.333</td>
<td>0.00980</td>
</tr>
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<td>Full Cover</td>
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<td>8.17E-11</td>
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<td>4.816</td>
<td>7.29E-07</td>
</tr>
<tr>
<td>No Cover</td>
<td>0.572</td>
<td>6.181</td>
<td>3.17E-10</td>
</tr>
</tbody>
</table>
Table 4.7.4. Network metrics for host-parasite interaction structure for avian- and mammalian-host communities along the urban and natural gradients. Networks were calculated per urban or natural values calculated using PCA-values per sampling unit.

<table>
<thead>
<tr>
<th>Urban Gradient</th>
<th>Region</th>
<th>-0.108</th>
<th>-0.52</th>
<th>0</th>
<th>0.203</th>
<th>0.54</th>
</tr>
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<tbody>
<tr>
<td>Avian</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generality</td>
<td></td>
<td>1.893</td>
<td>2.243</td>
<td>1.854</td>
<td>1.867</td>
<td>1.775</td>
</tr>
<tr>
<td>Vulnerability</td>
<td></td>
<td>5.164</td>
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<td>5.885</td>
<td>4.962</td>
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<td>0.543</td>
<td>0.412</td>
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<td>0.587</td>
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</tr>
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<td>0.257</td>
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<td>0.335</td>
</tr>
<tr>
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<td>2.454</td>
<td>2.467</td>
<td>2.539</td>
<td>2.712</td>
<td>2.485</td>
</tr>
<tr>
<td>Mammalian</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generality</td>
<td></td>
<td>1.704</td>
<td>1.646</td>
<td>1.966</td>
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<td>1.505</td>
</tr>
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<td>1.842</td>
<td>2.140</td>
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<td>0.903</td>
<td>0.671</td>
<td>0.685</td>
<td>0.780</td>
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<tr>
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<td>0.238</td>
<td>0.366</td>
<td>0.301</td>
</tr>
<tr>
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<td>2.154</td>
<td>1.853</td>
<td>2.283</td>
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<td>2.054</td>
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</table>

<table>
<thead>
<tr>
<th>Natural Gradient</th>
<th>Region</th>
<th>-0.73</th>
<th>-0.34</th>
<th>0</th>
<th>0.31</th>
<th>0.58</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avian</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generality</td>
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<td>1.429</td>
<td>1.842</td>
<td>1.657</td>
<td>1.857</td>
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<td>0.698</td>
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<td>1.233</td>
<td>1.585</td>
<td>1.649</td>
<td>1.516</td>
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<td>1.976</td>
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<tr>
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<td>0.723</td>
<td>0.702</td>
<td>0.805</td>
</tr>
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<td>0.522</td>
<td>0.278</td>
<td>0.311</td>
</tr>
<tr>
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<td>2.154</td>
<td>1.639</td>
<td>1.976</td>
<td>2.106</td>
<td>2.082</td>
</tr>
</tbody>
</table>
4.8 Figures

Figure 4.8.1. Study region and opportunistic sampling units. All opportunistically-sampled wildlife species were found in the southwestern Ontario region, but centralized to Toronto (Ontario, Canada). Sites where parasitic interactions were observed were categorized by (a) an urban index (PC1) and (b) a natural index (PC2), both calculated using land-cover data consolidated using principle components analysis. Sample sizes in maps represent number of opportunistic sampling sites.
Figure 4.8.2. Quantitative host-parasite bipartite interaction networks developed at the (a) species level and (b) functional-group level for all interactions observed at the regional scale. For each network, bars on the left represent wildlife host abundance and bars on the right represent parasite abundance. Linkage width represents frequency of a given host-parasite interaction. Asymmetric structure can be observed at both levels of interactions whereby a core group of generalist hosts or parasite species (or functional groups) maintain a significant quantity of the total sum of interactions.
Figure 4.8.3. Quantitative host-parasite bipartite interaction networks developed at the functional-group level for all interactions observed along the urbanization versus natural gradients. Variation in asymmetric structure can be observed along each gradient to a similar magnitude for urban and natural categories. Host and parasite species codes are given in Appendix Tables 4.9.1.1 and 4.9.1.2. Sample sizes indicate number of observed host-parasite interactions.
Figure 4.8.4. Quantitative (weighted) bipartite network metrics per urban and natural gradient category to quantify bipartite network structure. Solid lines represent significant differences between the urban and natural gradients using non-parametric Kruskal-Wallis tests, whereas dotted lines represent no significant differences. Solid symbols represent significant deviations of observed metrics from metrics calculated using constrained, randomized null bipartite networks.
Figure 4.8.5. Weighted-distance matrices to represent nestedness of host-parasite interactions along the urbanization and natural gradients. Dark red bands correspond to links of high relative importance in an extinction sequence when dealing with species distribution. All networks demonstrated significant nestedness from expected means, but nestedness was greater in urban and suburban networks. Tests of nestedness of fully-occupied host community were compared to the total host community (Appendix 4.9.2.1).
Figure 4.8.6. Quantitative host-parasite bipartite interaction networks developed for mammalian versus avian species along the urban gradient. I did not include visualization of networks developed over the natural gradient. Asymmetric structure is observed along the urban gradient for mammalian and avian host communities. Species codes are given in Appendices 4.9.1, 4.9.2. Sample sizes indicate number of observed host-parasite interactions.
Figure 4.8.7. Quantitative bipartite network metrics across an urbanization and natural gradient for mammalian and avian interactions (calculated at the species level). In contrast to the consolidated host-parasite community (Fig. 4.8.4), significant differences were observed in bipartite web structure between mammalian and host communities in the character and frequency of parasitic interactions.
Figure 4.8.8. Species-strength, representing species relevance across all partners, calculated as the sum of dependencies per species per host- and parasite functional group at the regional level. Species-strength values demonstrate asymmetric structure in the regional-scale networks for both host and parasite functional groups.
Figure 4.8.9. Species strength values calculated for host and parasite functional groups along the urban gradient. Pigeons and doves increase in species strength value from rural to high-urban areas, whereas songbirds decrease in species strength. For parasites, viral parasites are consistent across the urban gradient whereas ectoparasites maximize in species strength value in suburban areas.
### 4.9 Appendix

#### 4.9.1 Tables

Table 4.9.1.1 Reference key for hosts, including host code, scientific name, and common name.

<table>
<thead>
<tr>
<th>Code</th>
<th>Scientific Name</th>
<th>Common Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td><em>Anas rubripes</em></td>
<td>American Black Duck</td>
</tr>
<tr>
<td>2</td>
<td><em>Fulica americana</em></td>
<td>American Coot</td>
</tr>
<tr>
<td>3</td>
<td><em>Corvus brachyrhynchos</em></td>
<td>American Crow</td>
</tr>
<tr>
<td>4</td>
<td><em>Spinus tristis</em></td>
<td>American Goldfinch</td>
</tr>
<tr>
<td>5</td>
<td><em>Turdus migratorius</em></td>
<td>American Robin</td>
</tr>
<tr>
<td>6</td>
<td><em>Strix varia</em></td>
<td>Barred Owl</td>
</tr>
<tr>
<td>7</td>
<td><em>Eptesicus fuscus</em></td>
<td>Big Brown Bat</td>
</tr>
<tr>
<td>8</td>
<td><em>Cyanocitta cristata</em></td>
<td>Blue Jay</td>
</tr>
<tr>
<td>9</td>
<td><em>Branta canadensis</em></td>
<td>Canadian Goose</td>
</tr>
<tr>
<td>10</td>
<td><em>Quiscalus quiscula</em></td>
<td>Common Grackle</td>
</tr>
<tr>
<td>11</td>
<td><em>Accipiter cooperii</em></td>
<td>Cooper's Hawk</td>
</tr>
<tr>
<td>12</td>
<td><em>Canis latrans</em></td>
<td>Coyote</td>
</tr>
<tr>
<td>13</td>
<td><em>Phalacrocorax auritus</em></td>
<td>Double-crested Cormorant</td>
</tr>
<tr>
<td>14</td>
<td><em>Tamias striatus</em></td>
<td>Eastern Chipmunk</td>
</tr>
<tr>
<td>15</td>
<td><em>Sylvilagus floridanus</em></td>
<td>Eastern Cottontail</td>
</tr>
<tr>
<td>16</td>
<td><em>Sciurus carolinensis</em></td>
<td>Eastern Gray Squirrel</td>
</tr>
<tr>
<td>17</td>
<td><em>Bubo virginianus</em></td>
<td>Great Horned Owl</td>
</tr>
<tr>
<td>18</td>
<td><em>Picoles villosum</em></td>
<td>Hairy Woodpecker</td>
</tr>
<tr>
<td>19</td>
<td><em>Catharus guttatus</em></td>
<td>Hermit Thrush</td>
</tr>
<tr>
<td>20</td>
<td><em>Carpodacus mexicanus</em></td>
<td>House Finch</td>
</tr>
<tr>
<td>21</td>
<td><em>Passer domesticus</em></td>
<td>House Sparrow</td>
</tr>
<tr>
<td>22</td>
<td><em>Myotis lucifugus</em></td>
<td>Little Brown Bat</td>
</tr>
<tr>
<td>23</td>
<td><em>Anas platyrhynchos</em></td>
<td>Mallard</td>
</tr>
<tr>
<td>24</td>
<td><em>Microtus pennsylvanicus</em></td>
<td>Meadow Vole</td>
</tr>
<tr>
<td>25</td>
<td><em>Zenaida macroura</em></td>
<td>Mourning Dove</td>
</tr>
<tr>
<td>26</td>
<td><em>Cygnus olor</em></td>
<td>Mute Swan</td>
</tr>
<tr>
<td>27</td>
<td><em>Cardinalis cardinalis</em></td>
<td>Northern Cardinal</td>
</tr>
<tr>
<td>28</td>
<td><em>Accipiter gentilis</em></td>
<td>Northern Goshawk</td>
</tr>
<tr>
<td>29</td>
<td><em>Circus cyaneus</em></td>
<td>Northern Harrier</td>
</tr>
<tr>
<td>30</td>
<td><em>Aegolius acadicus</em></td>
<td>Northern Saw-whet Owl</td>
</tr>
<tr>
<td>31</td>
<td><em>Rattus norvegicus</em></td>
<td>Norway Rat</td>
</tr>
<tr>
<td>32</td>
<td><em>Hysticomorph hystricidae</em></td>
<td>Porcupine</td>
</tr>
<tr>
<td>33</td>
<td><em>Procyon lotor</em></td>
<td>Raccoon</td>
</tr>
<tr>
<td>34</td>
<td><em>Buteo jamaicensis</em></td>
<td>Red-tailed Hawk</td>
</tr>
<tr>
<td>35</td>
<td><em>Agelaius phoeniceus</em></td>
<td>Red-winged Blackbird</td>
</tr>
<tr>
<td>36</td>
<td><em>Vulpes vulpes</em></td>
<td>Red Fox</td>
</tr>
<tr>
<td>37</td>
<td><em>Sciurus vulgaris</em></td>
<td>Red Squirrel</td>
</tr>
<tr>
<td>38</td>
<td><em>Larus delawarensis</em></td>
<td>Ring-billed Gull</td>
</tr>
<tr>
<td>39</td>
<td><em>Columba livia</em></td>
<td>Rock Pigeon</td>
</tr>
<tr>
<td>40</td>
<td><em>Glaucops volans</em></td>
<td>Southern Flying Squirrel</td>
</tr>
<tr>
<td>41</td>
<td><em>Mephitis mephitis</em></td>
<td>Striped Skunk</td>
</tr>
<tr>
<td>42</td>
<td><em>Cygnus buccinator</em></td>
<td>Trumpeter Swan</td>
</tr>
<tr>
<td>43</td>
<td><em>Didelphis virginiana</em></td>
<td>Virginia Opossum</td>
</tr>
<tr>
<td>44</td>
<td><em>Aix sponsa</em></td>
<td>Wood Duck</td>
</tr>
</tbody>
</table>
Table 4.9.1.2 Reference key for parasites, including parasite code, scientific names, and common names.

<table>
<thead>
<tr>
<th>Code</th>
<th>Scientific Name</th>
<th>Common Name</th>
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<tbody>
<tr>
<td>A</td>
<td>Avian paramyxovirus-1</td>
<td>AMPV-1</td>
</tr>
<tr>
<td>B</td>
<td>Avipoxvirus</td>
<td>Avian Pox</td>
</tr>
<tr>
<td>C</td>
<td>Baylisascaris procyonis (definitive)</td>
<td>Baylisascaris (definitive)</td>
</tr>
<tr>
<td>D</td>
<td>Baylisascaris spp. (aberrant)</td>
<td>Baylisascaris spp. (aberrant)</td>
</tr>
<tr>
<td>E</td>
<td>Canine Distemper Virus</td>
<td>Canine Distemper Virus</td>
</tr>
<tr>
<td>F</td>
<td>Capillaria spp.</td>
<td>Capillaria</td>
</tr>
<tr>
<td>G</td>
<td>Circoviruses</td>
<td>Circovirus</td>
</tr>
<tr>
<td>H</td>
<td>Coccidia spp.</td>
<td>Coccidia</td>
</tr>
<tr>
<td>I</td>
<td>Siphonaptera spp.</td>
<td>Fleas</td>
</tr>
<tr>
<td>J</td>
<td>Hyppoboscidae spp.</td>
<td>Hyppoboscid Flies</td>
</tr>
<tr>
<td>K</td>
<td>Leptospira spp.</td>
<td>Leptospira</td>
</tr>
<tr>
<td>L</td>
<td>Phthiraptera spp.</td>
<td>Lice</td>
</tr>
<tr>
<td>M</td>
<td>Acari spp.</td>
<td>Mites</td>
</tr>
<tr>
<td>N</td>
<td>Notoedres spp.</td>
<td>Notoendric mite</td>
</tr>
<tr>
<td>O</td>
<td>Rabies</td>
<td>Rabies</td>
</tr>
<tr>
<td>P</td>
<td>Sarcoptes spp.</td>
<td>Sarcoptic Mites</td>
</tr>
<tr>
<td>Q</td>
<td>Salmonella spp.</td>
<td>Salmonella</td>
</tr>
<tr>
<td>R</td>
<td>Squirrel parapoxvirus</td>
<td>Squirrel Pox</td>
</tr>
<tr>
<td>S</td>
<td>Strongylidae spp.</td>
<td>Strongyles</td>
</tr>
<tr>
<td>T</td>
<td>Isodoidea spp.</td>
<td>Ticks</td>
</tr>
<tr>
<td>U</td>
<td>Trichomonas spp.</td>
<td>Trichomoniasis</td>
</tr>
<tr>
<td>V</td>
<td>West Nile Virus</td>
<td>West Nile Virus</td>
</tr>
<tr>
<td>W</td>
<td>Pseudogymnoascus destructans</td>
<td>White Nose Syndrome</td>
</tr>
<tr>
<td>Functional Group</td>
<td>General Description</td>
<td>Host Species</td>
</tr>
<tr>
<td>------------------</td>
<td>---------------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Bats</td>
<td>Insectivorous winged mammals capable of sustained flight</td>
<td>Big Brown Bat (<em>Eptesicus fuscus</em>)&lt;br&gt;Little Brown Bat (<em>Myotis lucifugus</em>)</td>
</tr>
<tr>
<td>Falcons and Hawks</td>
<td>Small to medium-sized diurnal birds of prey</td>
<td>Cooper's Hawk (<em>Accipiter cooperii</em>)&lt;br&gt;Northern Goshawk (<em>Accipiter gentilis</em>)&lt;br&gt;Northern Harrier (<em>Circus cyaneus</em>)&lt;br&gt;Red-tailed Hawk (<em>Buteo jamaicensis</em>)</td>
</tr>
<tr>
<td>Lagomorphs</td>
<td>Herbivorous mammals with fur, bear live young, have four limbs, and lactate.</td>
<td>Eastern Cottontail (<em>Sylvilagus floridanus</em>)</td>
</tr>
<tr>
<td>Large Carnivores</td>
<td>Large-bodied terrestrial, placental mammals defined by prominent canine teeth and simple stomachs adapted to digest meat</td>
<td>Coyote (<em>Canis latrans</em>)&lt;br&gt;Red Fox (<em>Vulpes vulpes</em>)</td>
</tr>
<tr>
<td>Large Rodents</td>
<td>Large-bodied mammals defined by single pair of unremittingly growing incisors in upper and lower jaws</td>
<td>Porcupine (<em>Hysticomorph hystricidae</em>)</td>
</tr>
<tr>
<td>Owls</td>
<td>Solitary and nocturnal birds of prey, defined by upright stance, a large broad head, binocular vision, binaural hearing, and feathers adapted for silent flight.</td>
<td>Barred Owl (<em>Strix varia</em>)&lt;br&gt;Great Horned Owl (<em>Bubo virginianus</em>)&lt;br&gt;Northern Saw-whet Owl (<em>Aegolius acadicus</em>)</td>
</tr>
</tbody>
</table>
| **Strigiformes** | Stout-bodied birds with variable plumage that feed on seeds, fruits, and plants. | Mourning Dove (*Zenaida macroura*)  
Rock Pigeon (*Columbia livia*) | Adapted to wide variety of habitat, with large natural ranges.  
Many persist in various open and semi-open environments, cliffs and rock ledges, roosting together in buildings or other built structures  
Distributed evenly on every continent except Antarctica | Seeds and fruit form major component of diet, but majority are granivorous and opportunistic foragers  
Often feed on ground in flocks |
| **Seabirds and Shorebirds** | Small-to-medium sized birds that forage and nest near aquatic habitat. | Double-crested Cormorant (*Phalacrocorax auritus*)  
Ring-billed Gull (*Larus delawarensis*)  
Raccoon (*Procyon lotor*)  
Striped Skunk (*Mephitis mephitis*)  
Virginia Opossum (*Didelphis virginiana*) | Breeding habitat near lakes, rivers, coasts  
Nest colonially on ground, faithful to nesting site  
Congregate in large numbers | Forage in flight, while swimming or diving (*Suliformes*), walking, or wading (*Charadriiformes*)  
Ominvorous: insects, fish, grain, eggs, earthworms, rodents  
*Charadriiformes*: opportunistic foragers (anthropogenic sources) |
| **Small Carnivores** | Small-to-medium sized terrestrial mammals defined by prominent canine teeth, simple stomachs, and mainly carnivorous diets | Eastern Chipmunk (*Tamias striatus*)  
Eastern Gray Squirrel (*Sciurus carolinensis*)  
Meadow Vole (*Microtus pennsylvanicus*)  
Norway Rat (*Rattus norvegicus*)  
Red Squirrel (*Sciurus vulgaris*)  
Southern Flying Squirrel (*Glaucomys volans*) | Originally adapted to deciduous and mixed forests, but adapted to extended range from mountainous, to coastal, to urban areas  
Dependent on vertical structures to climb to escape predation  
Nest in a variety of natural (tree hollows, rock crevices, ground dwellings) and unnatural (attics, chimneys, garages) locations | Forage on a variety of sources: invertebrates, plant materials, and some vertebrates  
Extremely omnivorous and high dietary plasticity |
| **Small Rodents and Moles** | Small-to-medium bodied mammals characterized by a single pair of unremittingly growing incisors | American Crow (*Corvus brachyrhynchos*)  
American Goldfinch (*Spinus tristis*)  
American Robin (*Turdus migratorius*)  
Blue Jay (*Cyanocitta cristata*) | Live and persist in a variety of terrestrial habitats; many depend on arboreal habitat for food and shelter  
Thrive in anthropogenically-altered habitat such as agricultural fields or urban areas  
Some persist in underground burrows | Omnivorous: rely on foods rich in protein, carbohydrates, and fats: nuts, seeds, plants, conifer cones, fruits, fungi, and green vegetation  
Can persist on anthropogenic food resources |
| **Songbirds** | Perching birds with complex vocalizations | American Crow (*Corvus brachyrhynchos*)  
American Goldfinch (*Spinus tristis*)  
American Robin (*Turdus migratorius*)  
Blue Jay (*Cyanocitta cristata*) | Variety of niche breadth where natural forest, shrub, or hedge is available for nesting, roosting, or foraging  
Many originally adapted to open habitat including grasslands, desert, and scrubland. Others are adapted to open woodland. | Primarily feed on nuts and seeds, but also soft fruits, berries, arthropods, and occasionally small vertebrates  
Most songbird species quickly naturalize in human-modified habitat such as agricultural or urban areas. |
### Waterfowl and Waders

Long-legged wading birds highly adapted for an aquatic life history

**Taxonomic orders:**
- Anseriformes
- Charadriiformes

| Common Grackle (*Quiscalus quiscula*) | American Black Duck (*Anas rubripes*) | Associated with wetland or coastal environments, can persist in grassland environments if adjacent to large water source |
| Hairy Woodpecker (*Picoides villosus*) | American Coot (*Fulica americana*) | Nests usually beside wetland or pond environments |
| Hermit Thrush (*Catharus guttatus*) | Canadian Goose (*Branta canadensis*) | Some species feed on terrestrial habitat adjacent (to some proximity) to aquatic habitat |
| House Finch (*Carpodacus mexicanus*) | Mallard (*Anas platyrhynchos*) | |
| House Sparrow (*Passer domesticus*) | Mute Swan (*Cygnus olor*) | |
| Northern Cardinal (*Cardinalis cardinalis*) | Trumpeter Swan (*Cygnus buccinator*) | |
| Red-winged Blackbird (*Agelaius phoeniceus*) | Wood Duck (*Aix sponsa*) | |

- Majority feed on small invertebrates, but variation in length of bills enables different food preferences
- Variety of feeding strategies adapted by the group

### References


<table>
<thead>
<tr>
<th>Parasites</th>
<th>Hosts (TWC Records)</th>
<th>Host Specificity</th>
<th>Etiology and Transmission</th>
<th>Environmental Factors</th>
<th>Pathology</th>
<th>Diagnostics</th>
<th>Zoonotic Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Endoparasites</strong></td>
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<tr>
<td><strong>Viruses</strong></td>
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</tr>
<tr>
<td>Rabies</td>
<td>Big Brown Bat</td>
<td>No specificity considered.</td>
<td>Lyssavirus: viral, transmitted by scratch, bite, or saliva from infected animal (direct, contact-based exposure)</td>
<td>No environmental link; transmission related to host densities and host species interactions</td>
<td>Fatal, debilitating: causes abnormal, aggressive behavior in hosts</td>
<td>Inconsistent; suspected by behaviour; CCWHC confirmed</td>
<td>Yes. Transmitted to humans by bite or scratch of infected animal. Early treatment necessary; likely fatal after showing symptoms.</td>
</tr>
<tr>
<td><strong>Circovirus</strong></td>
<td>Rock pigeon</td>
<td>No specificity considered.</td>
<td>Circoviridae: viral, direct or indirect transmission, via feather dander, feces, bodily fluids of infected birds</td>
<td>Long lived in the environment, can withstand freezing, remains in nests or nest boxes</td>
<td>Debilitating: disease of beak and feathers; immunosuppressive, could lead to susceptibility of other disease agents (ie. bacteria, fungi)</td>
<td>Consistent (condition)</td>
<td>No</td>
</tr>
<tr>
<td>Avian Pox</td>
<td>Northern Cardinal</td>
<td>No specificity considered. (Strains for gamebirds, songbirds, marine birds, etc.)</td>
<td>Avipoxvirus: viral, transmitted by direct contact with infected birds, ingestion of food and water contaminated by sick birds or carcasses, or contact with contaminated surfaces such as bird feeders and perches</td>
<td>Contamination of aquatic habitat, bird feeders, or perches</td>
<td>Debilitating: difficulty seeing, breathing, feeding, perching: weak, emaciated hosts</td>
<td>Consistent (condition)</td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>American Coot</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Mourning Dove</td>
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<tr>
<td></td>
<td>Wood Duck</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Common Grackle</td>
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<tr>
<td></td>
<td>Mallard</td>
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<tr>
<td></td>
<td>Rock Pigeon</td>
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<tr>
<td></td>
<td>American Robin</td>
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<tr>
<td></td>
<td>House Sparrow</td>
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<tr>
<td>Squirrel Pox</td>
<td>Eastern Gray Squirrel</td>
<td>No specificity considered.</td>
<td>Parapoxvirus: viral, transmitted by contact with infected lesions or contaminated crusts</td>
<td>Infected crusts resistant to drying; contaminated feeders or nests</td>
<td>Mildly debilitating. Rarely fatal (acquired immunity); can cause lethargy, lesions, scabs; can be debilitating for red squirrels</td>
<td>Consistent (condition)</td>
<td>No</td>
</tr>
<tr>
<td>Avian Paramyxovirus (APMV-1)</td>
<td>Double-crested Cormorant Rock Pigeon</td>
<td>No specificity considered. Many known serotypes; same genus as</td>
<td>Avulavirus: viral, direct contact between animals, indirect exposure by contaminated water sources, fomites</td>
<td>Contamination of water sources; feces of infected birds</td>
<td>Debilitating. Trembling, twisting of neck, wet/liquid feces, lethargy;</td>
<td>Consistent (condition)</td>
<td>Newcastle Disease can be transmissible to humans, but mild symptoms; mild conjunctivitis and</td>
</tr>
<tr>
<td><strong>Newcastle Disease</strong> (APMV-1)</td>
<td>paralysis of wings and legs</td>
<td>influenza-like symptoms; no significant hazard to human health</td>
<td></td>
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<tr>
<td>Canine Distemper Virus (CDV)</td>
<td>Coyote</td>
<td>No specificity considered (likely only the canid variant in Ontario)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Red Fox</td>
<td>Morbillivirus (Paramyxoviridae): viral, contagious; spread through air droplets or direct contact with infected animal</td>
<td></td>
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<tr>
<td></td>
<td>Raccoon</td>
<td>None.</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Striped Skunk</td>
<td>Fatal, especially for immunosuppressed; high fever, reddened eyes, watery discharge from nose and eyes, lethargy, anorexia</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Inconsistent (behaviour); CCWHC confirmed</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>No, but very transmissible to domestic canines</td>
<td></td>
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</tr>
<tr>
<td>West Nile Virus (WNV)</td>
<td>American Crow</td>
<td>No specificity considered</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Common Grackle</td>
<td>Flavivirus: vector-borne virus, transmitted between avian reservoirs and mosquito vectors</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td></td>
<td>Red-tailed Hawk</td>
<td>Vector-borne disease: related to presence of mosquito vector and mosquito habitat (stagnant water, wetlands, birdbaths, etc.)</td>
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<tr>
<td></td>
<td>Blue Jay</td>
<td>Relatively asymptomatic. Rarely, individuals lethargic.</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Northern Harrier</td>
<td>Inconsistent: CCWHC confirmed</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Great Horned Owl</td>
<td>Yes. Significant vector-borne disease</td>
<td></td>
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<tr>
<td></td>
<td>Owl</td>
<td></td>
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</tr>
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<td></td>
<td>House Sparrow</td>
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<tr>
<td>Parvovirus</td>
<td>Raccoon</td>
<td>No specificity identified.</td>
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<tr>
<td></td>
<td></td>
<td>Paroviridae: Raccoon parvovirus, similar to feline parvovirus: viral; highly contagious, transmitted by direct or indirect contact with infected feces, also by ingestion of virus-infected pig carcasses.</td>
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<td></td>
<td></td>
<td>Can persist in feces in free-living environment; virus can persist in ground soil for up to a year.</td>
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<tr>
<td></td>
<td></td>
<td>Usually sub-clinical in raccoons</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Inconsistent: CCWHC confirmed</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Nematodes</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Capillaria</td>
<td>Mourning Dove</td>
<td>No specificity considered, (though recognize impossibility in identification of various species and life histories).</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>American Black Duck</td>
<td>Nematoda (Trichurida) – endoparasite, often transmitted by ingestion of intermediate host (earthworm - C. annulata, C. contorta, C. caudiflata), but can be directly-transmitted by contact (C. obsignata, C. anatis).</td>
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<tr>
<td></td>
<td>Ring-billed Bull</td>
<td>Persists in earthworms (intermediate host).</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Red-tailed Hawk</td>
<td>Debilitating in young birds: weight loss, diarrhea, regurgitation, anaemia</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Mallard</td>
<td>Consistent (fecal flotation or necropsy); up to 2008?</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Rock Pigeon</td>
<td>No.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Trumpeter Swan</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strongyles</td>
<td>Mammalian</td>
<td>Yes. Mammalian and Avian categories, despite numerous subgroups of strongyle species.</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Little brown Bat</td>
<td>Nematoda (Strongylida) – endoparasite, known as threadworms, often transmitted by</td>
<td></td>
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<tr>
<td></td>
<td>Coyote</td>
<td>Persists in environments contaminated by infected feces (soil, water); seasonal;</td>
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</tr>
<tr>
<td></td>
<td>Red Fox</td>
<td>Often asymptomatic, can cause diarrhea in young animals</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Consistent (fecal flotation or necropsy); up to 2008?</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>No.</td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>Avian</strong></td>
<td><strong>Roundworm form found in natural hosts</strong></td>
<td><strong>Baylisascaris procyonis</strong> (raccoon roundworm); endoparasitic worm transmitted by oral ingestion of eggs in environment, feeding on animal infested with <em>Baylisascaris</em>, or contact with infected feces (skin absorption)</td>
<td><strong>Eggs, deposited in feces of infected animal, remain viable in environment for years; withstand heat and cold</strong></td>
<td><strong>Relatively asymptomatic in definitive hosts</strong></td>
<td><strong>Consistent (fecal flotation)</strong></td>
<td><strong>Yes. Though rare, people become infected by ingesting infectious eggs (most likely children); can cause nausea, tiredness, liver enlargement, blindness, coma.</strong></td>
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</tr>
<tr>
<td>Avian Red-winged Blackbird Canada Goose Ring-billed Gull Rock Pigeon American Robin House Sparrow Mute Swan Trumpeter Swan</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Raccoon Roundworm</strong> (definitive)</th>
<th><strong>Roundworm form found in natural hosts</strong> (raccoons).</th>
<th></th>
<th></th>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Raccoon</td>
<td></td>
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</tbody>
</table>

| **Raccoon Roundworm** (aberrant) | **Aberrant form of infection in secondary hosts; in the region it is likely that a skunk-specific species (*Baylisascaris columnaris*) exists** | **Baylisascaris spp.; infects a variety of other paratenic hosts transmitted by oral ingestion of eggs in environment, feeding on animal infested with *Baylisascaris*, or contact with infected feces (skin absorption)** | **Eggs, deposited in feces of infected animal, remain viable in environment for years; withstand heat and cold** | **Can cause gastrointestinal symptoms, visceral, ocular, or neural larval migrans) can be fatal in small-bodied paratenic hosts** | | **No.** |
| --- | --- | --- | --- | --- | | |
| Beaver Eastern Cottontail Coyote Red Fox Canada Goose Groundhog Porcupine Striped Skunk Eastern Gray Squirrel Red Squirrel | | | | | | |

<table>
<thead>
<tr>
<th><strong>Bacteria</strong></th>
<th><strong>No specificity considered.</strong></th>
<th><strong>Salmonella (up to 2000 bacteria species); endoparasite; transmitted through air as bacteria is shed from infected bird in the nasal or ocular secretions, fecal material, and feather dust</strong></th>
<th><strong>Spreads largely in crowded conditions (density dependent)</strong></th>
<th><strong>Often asymptomatic (sub-clinical); can cause lethargy, anorexia, diarrhea, arthritis, air sacculitis, acute fatal septicemia</strong></th>
<th><strong>Consistent (condition, fecal culture, or culture of abscesses)</strong></th>
<th><strong>Not considered dangerous for humans but may threaten infants, elderly, or those with immunosuppressive diseases</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Salmonella American Goldfinch Rock Pigeon House Sparrow</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
### Leptospira

<table>
<thead>
<tr>
<th>Hosts</th>
<th>Specificity Identified</th>
<th>Transmission</th>
<th>Disease Severity</th>
<th>Testing</th>
<th>Human Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Porcupine, Striped Skunk</td>
<td>No specificity identified</td>
<td>Leptospira (spirochete bacterium); many pathogenic and non-pathogenic species; transmitted by contact with urine of infected animal (primary or secondary hosts); or exposure by contaminated environment (water or soil)</td>
<td>Can persist in wet soil or water: muddy banks, ditches, guttles.</td>
<td>Mild to moderate: fever, vomiting, diarrhea, anorexia, weakness, muscle pain.</td>
<td>Inconsistent: opportunistic testing</td>
</tr>
</tbody>
</table>

### Coccidia

<table>
<thead>
<tr>
<th>Hosts</th>
<th>Specificity Identified</th>
<th>Pathogen Description</th>
<th>Disease Severity</th>
<th>Testing</th>
<th>Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mammalian</td>
<td>Yes. Mammalian and avian categories considered.</td>
<td>Coccidia (diverse protozoan parasites of genus <em>Eimeria</em>); endoparasite; transmission by exposure in free-living environment (eating or drinking) contaminated by infected feces (eggs or oocysts)</td>
<td>Eggs or oocysts deposited in feces by infected hosts develop in free-living environment (water, soil, moist surfaces)</td>
<td>Mildly debilitating (Coccidiosis): often animals develop immunity; can cause diarrhea, anorexia, anemia, lethargy, dehydration, increased susceptibility to other disease agents</td>
<td>Consistent (condition) up to 2008?</td>
</tr>
<tr>
<td>Avian</td>
<td></td>
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<tr>
<td>Virginia Opossum</td>
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</tr>
<tr>
<td>Red Fox</td>
<td></td>
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</tr>
<tr>
<td>Striped Skunk</td>
<td></td>
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<tr>
<td>Eastern Gray Squirrel</td>
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<tr>
<td>Red Squirrel</td>
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<tr>
<td>Mourning Dove</td>
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<tr>
<td>House Finch</td>
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<tr>
<td>Northern Goshawk</td>
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<tr>
<td>Common Grackle</td>
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<tr>
<td>Ring-billed Gull</td>
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<tr>
<td>Cooper’s Hawk</td>
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<tr>
<td>Red-tailed Hawk</td>
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<tr>
<td>Mallard</td>
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</tr>
<tr>
<td>Rock Pigeon</td>
<td></td>
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</tr>
<tr>
<td>House Sparrow</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trumpeter Swan</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Mute Swan</td>
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<tr>
<td>Common Grackle</td>
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<tr>
<td>Ring-billed Gull</td>
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<tr>
<td>Cooper’s Hawk</td>
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<tr>
<td>Red-tailed Hawk</td>
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<tr>
<td>Mallard</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Rock Pigeon</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>American Robin</td>
<td></td>
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</tbody>
</table>

### Trichonomas

<table>
<thead>
<tr>
<th>Hosts</th>
<th>Specificity Identified</th>
<th>Pathogen Description</th>
<th>Disease Severity</th>
<th>Testing</th>
<th>Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rock Pigeon American Robin</td>
<td>No specificity identified.</td>
<td><em>Trichomonas gallinae</em> (cosmopolitan protozoa): transmission most likely by birds feeding each other (regurgitation); through food or water</td>
<td>Cannot survive long periods outside the host.</td>
<td>Minor debilitation: lethargy, fluffed-plumage, drooling, regurgitation, difficulty swallowing</td>
<td>Consistent (condition)</td>
</tr>
<tr>
<td><strong>Fungal</strong></td>
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<tr>
<td><strong>White Nose Syndrome (WNS)</strong></td>
<td>Little Brown Bat</td>
<td>No specificity considered</td>
<td><em>Pseudogymnoascus destructans</em>: fungal; (independent fungus from <em>Geomyces</em> genus); Physical contact required between bats; possibly soil contamination</td>
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<td></td>
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<td></td>
<td>Some evidence for soil contamination of spores in caves/hibernacula</td>
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<tr>
<td></td>
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<td></td>
<td>Fatal; colonization of bat skin and nasal surfaces; loss of body fat; scarring of wing membranes</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Inconsistent (condition), opportunistic, CCWHC confirmed</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Ectoparasites</strong></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Sarcoptic Mange</strong></td>
<td>Coyote Red Fox Porcupine Raccoon Striped Skunk</td>
<td><em>Sarcoptes</em> spp.</td>
<td>Mite-associated skin disease: Acari, ectoparasite; transmitted by migration of arthropods between individuals or from the environment</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Can persist in environments near hosts (nests, burrows, dry built environments)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mildly debilitating: causes itching, skin inflammation, skin lesions, can cause susceptibility to other disease agents</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Consistent (condition)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>No.</td>
</tr>
<tr>
<td><strong>Notoedric Mange</strong></td>
<td>Porcupine Eastern Gray Squirrel Red squirrel</td>
<td><em>Notoedres centripela</em> (douglasi)</td>
<td>Mite-associated skin disease, particular to ear: Acari, ectoparasite; transmitted by migration of arthropods between individuals or from the environment</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Can persist in environments near hosts (nests, burrows, dry built environments)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mildly debilitating: causes itching, skin inflammation, skin lesions, can cause susceptibility to other disease agents</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Consistent (condition); no identification mites to genus- or species-level.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>No.</td>
</tr>
<tr>
<td><strong>Mites</strong></td>
<td>Mammalian Big Brown Bat Little Brown Bat Eastern Cottontail Red Fox Porcupine Raccoon Norway Rat Eastern Gray Squirrel Meadow Vole Avian Canada Goose Common Grackle Ring-Billed Gull Mallard Rock Pigeon</td>
<td>Yes: Mammalian and avian categories.</td>
<td>Arthropods (variety of species): ectoparasites, exposure by environmental transmission via migrating nymphs (greater exposure at higher host densities)</td>
</tr>
<tr>
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<td></td>
<td>Persist in nests, soil, dry environments (built environments such as buildings)</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>Mildly debilitating: skin irritation; rashes; itching behaviour (can cause secondary infection)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Consistent (condition); no identification mites to genus- or species-level.</td>
</tr>
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<td>No.</td>
</tr>
</tbody>
</table>
### Fleas

<table>
<thead>
<tr>
<th>American Robin</th>
<th>Mute Swan</th>
<th>Hermit Thrush</th>
<th>Hairy Woodpecker</th>
</tr>
</thead>
<tbody>
<tr>
<td>Little Brown Bat</td>
<td>Eastern Cottontail Virginia Opposum Raccoon Striped Skunk Eastern Gray Squirrel Red Squirrel</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No specificity considered.</td>
<td>Siphonaptera (variety of arthropod insect species): ectoparasites, environmental transmission in dry, warm environments; jump between hosts (density dependent transmission)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Persists in dry, warm free-living environments, soil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mildly debilitating, skin irritation and itching behaviour of hosts</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consistent (condition)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>No.</td>
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</tbody>
</table>

### Lice

| Mammalian | Big Brown Bat Porcupine Raccoon Norway Rat Eastern Gray Squirrel |
| Yes, Mammalian and avian categories. | Phthiraptera (Louse): variety of arthropod insect species; obligate ectoparasites, avg. number of lice proportionate to host body size; moves between hosts directly |
| Rarely persists in free-living environment (wingless insects) |
| Mildly debilitating, skin irritation and itching, high burden can cause mortality |
| Consistent (condition) |
| No. |
Ticks

<table>
<thead>
<tr>
<th>Mammalian</th>
<th>Avian</th>
<th>Parasitiformes (arachnid species): ectoparasites; environmental transmission; can act as vectors for bacterial disease (ie. Lyme disease – Borreliosis, Babesia – Babesiosis, Bartonella – bartonellosis)</th>
<th>Persists, hibernates, and molts in soil, deciduous forest, river edge, etc.</th>
<th>Persistently debilitating, but very minor skin irritation. Typically ticks are not noticeable by hosts</th>
<th>Consistent (Condition)</th>
<th>No. But ticks can carry variety of vector-borne diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Raccoon</td>
<td>Hermit Thrush</td>
<td>Yes. Mammalian and avian categories.</td>
<td></td>
<td>No.</td>
<td>No.</td>
<td>No.</td>
</tr>
<tr>
<td>Striped Skunk</td>
<td>White-throated sparrow</td>
<td></td>
<td></td>
<td>No.</td>
<td>No.</td>
<td>No.</td>
</tr>
<tr>
<td>Eastern Gray Squirrel</td>
<td></td>
<td></td>
<td></td>
<td>No.</td>
<td>No.</td>
<td>No.</td>
</tr>
</tbody>
</table>

Hyppoboscid Flies

| Northern Cardinal                   | Hyppobosidae louse flies: ectoparasite; fly feeds on hosts (obligate parasite of birds); frequency-dependent transmission | No. | Debilitating. Mild to severe skin irritation; can be fatal for chicks | Consistent (condition) | No. |
| Cooper’s Hawk                       |                                                                              | No. | No.                                                                 | No.                                                                 | No. |
| Hawk                                |                                                                              | No. | No.                                                                 | No.                                                                 | No. |
| Great-horned Owl                    |                                                                              | No. | No.                                                                 | No.                                                                 | No. |

References


4.9.2 Figures

Figure 4.9.2.1. Weighted distances matrices to represent nestedness of host-parasite interactions for (a) all hosts represented in the regional community; and (b) only fully-occupied hosts in along the urban gradient. Dark red bands correspond to links of high relative importance in an extinction sequence when dealing with species distribution. All networks demonstrated significant nestedness from expected means, but nestedness was greater in urban and suburban networks. No significant differences in particular host-parasite interactions were identified between the total community versus the fully-occupied community.
Figure 4.9.2.2. PCA unconstrained ordination of environmental variables to develop PC indices of urban (BIS + RD + PAS) and natural (CF + DF + UG + SW) indices. PC1 explained 69% and PC2 explained 58% of the variability to the land cover factors. Environmental variable codes: BIS (built-impervious surface); RD (road density); UG (urban greenspace); CF (coniferous forest); DF (deciduous forest); SW (swamp); PAS (pasture).
Chapter 5

5 Host and Parasite Beta Diversity along an Urbanization Gradient

5.1 Abstract

It is widely accepted among ecologists that urbanization contributes to biotic homogenization of native communities, resulting in drastically altered community assemblages. Few studies, however, have evaluated the effect of urbanized landscapes on host and parasite diversity despite critical functional biotic and public health implications. I quantify, along an urbanization gradient near Toronto (Ontario, Canada) how alpha and beta diversity of host communities varies relative to their parasite communities. An urbanization index based on the first PCA of the land cover factors was characterized into 20 categories was computed on 1 km² grid cells ($n = 699$). Then I used the urbanization index to evaluate the diversity infected wildlife individuals ($n = 3073$) from the Toronto Wildlife Centre (2007-2012) from the southwestern Ontario region. Beta diversity was calculated as total variance in host and parasite community matrices. Further, beta diversity was partitioned into species contributions (SCBD) and sampling-unit contributions (LCBD) over the region. With urbanization, I found significant contributions of rural and highly-urbanized areas to beta diversity in both host and parasite communities, but not in suburban areas. Generalist hosts and parasites contributed most strongly to overall beta diversity. I argue that urban biotic homogenization of host communities, defined by dominant urban synanthropic species, provides invasive capacity for multi-host parasites to persist, transmit among urban wildlife populations, and potentially amplify in diversity. My work emphasizes the role of cities in structuring host and parasite community assembly via biotic homogenization of disease systems.

5.2 Introduction

As urbanization rapidly transforms landscapes worldwide, ecologists are required to investigate more clearly how urban environments affect patterns and processes of biodiversity (McKinney 2006, Shochat et al. 2006). With urban development comes radical changes in habitat structure, generally leading to a complete restructuring of vegetation, habitat composition, and connectivity (Faeth et al. 2005, Bierwagen 2007, Pardini et al. 2010, Scolozzi and Geneletti 2012). Intensive
shifts in urban habitat structure can homogenize ecological communities leading to biodiversity loss and increases in the abundance of species that thrive in urban areas (Shochat et al. 2006). However, anthropogenic habitat destruction, habitat fragmentation, and landscape simplification do not affect all species equally (Henle et al. 2004, Ewers and Didham 2006, Aronson et al. 2014) and urban biodiversity patterns are often described as non-linear relationships (Blair 2001, Crooks et al. 2004, Van Rensburg et al. 2009). Therefore, predicting ecosystem responses to urban perturbations still represents conceptual challenges because of the complex interactive dynamics between natural ecosystems that encompass many dimensions of the current biodiversity crisis (Luck and Smallbone 2011, Rodewald et al. 2014).

More recently, urban environments have been shown to influence host and parasite community composition towards altered disease dynamics (Watts et al. 2015). Because parasite species are dependent on host persistence and abundance, urban-environmental factors that shape host diversity should indirectly impact diversity patterns of parasites (Bradley and Altizer 2007, Brearley et al. 2013). Environmental factors that facilitate intra- and interspecific contacts among urban hosts should also favour the diversity of parasite infracomunities that reside within those populations (Wright and Gompper 2005, Lehrer et al. 2010). Conversely, unsuitable conditions that cause native species extinctions may cause the local extinction of their specialist parasites: specialist versus multi-host generalist parasites may respond to urban environments differently. Environmental shifts along an urbanization gradient such as temperature, precipitation, and vegetative conditions should also influence the persistence and transmissibility of vector-borne or free-living parasites (Sorvillo et al. 2002, Trejo-Macías et al. 2007). Therefore, quantifying the relative diversity of host versus parasite communities should shed light on the influence of urbanization on parasite ecology and disease dynamics (Martin and Boruta 2013, Giraudeau et al. 2014, Mackenstedt et al. 2015).

While many studies have investigated the role of human-modified landscapes on biotic and abiotic factors of parasite ecology (Sire et al. 2001, Gillespie and Chapman 2008, Püttker et al. 2008, Chasar et al. 2009), it is of increasing interest to ecologists and epidemiologists to investigate the particular impact of cities on host diversity relative to parasite diversity (Menalled et al. 1999, Jobet et al. 2000, Soriano et al. 2010). Parasites play a critical role in natural communities and ecosystem functioning (Hudson et al. 2006, Lefevre et al. 2009), and various studies have indicated that they are able to control host population size and demography.
Likewise, the functional role of biodiversity on disease transmission risk to humans remains a contentious debate. The hypothesis that transmission risk to humans and other species is generally reduced in communities with high biodiversity is both supported and disputed in the literature (Schmidt and Ostfeld 2001, Randolph and Dobson 2012, Ostfeld 2013, Pfäffle et al. 2015). Cities may reduce this potential ‘dilution effect’, whereby fewer host species in urban communities may indirectly amplify disease prevalence in remaining reservoir host or vector populations and, in effect, boost corresponding human infection risks (Keesing et al. 2010). Although this effect may not be generalizable to all disease systems, and is potentially limited to vector-borne disease dynamics, the debate remains a contemporary and vital frontier of disease ecology. Thus, the impact of cities on host and parasite biodiversity, though largely unknown, is a critical component of both disease ecology and epidemiology.

I investigated how urbanization influences the beta diversity of host communities as compared to their parasite communities. I quantified beta diversity of host and parasite communities along an urbanization gradient using a unique approach which partitions estimates of total beta diversity by specific contributions of species and sampling locations to overall diversity. I tested the hypothesis that urbanization has a negative effect on host and parasite diversity, caused by the biotic homogenization of natural communities and of the abiotic habitat in this study region. Biotic homogenization is defined as the replacement of local biotas with non-indigenous species, usually introduced by humans (McKinney and Lockwood 1999). I therefore expected that rural environments would reflect greater beta diversity because niche breadth and requirements in less disturbed, homogenized habitat should support a greater variation in species. I also tested the hypothesis that urban communities would be dominated by generalist hosts and parasites rather than specialist species. Urban environments should favour non-native, invasive species often defined by opportunistic, $r$-selected traits, rather than niche-specific species dependent on a small breadth of resources for survival and reproduction. Correspondingly, I expected that multi-host parasite species should thrive in high-density host populations often found in urban settings.
5.3 Methods

5.3.1 Study area and characterization of urban index

I obtained wildlife data from the Toronto Wildlife Centre in Toronto, Ontario (2007-2012). The southwestern Ontario region is defined by a gradient of development, from rural land cover dominated by agricultural and forested areas, to suburban land cover dominated by small forest patches and moderate-density residential dwellings, to highly urban land cover dominated by built, impervious surfaces and high-density residential dwellings. I approximated the spatial locations of each wildlife individual found across this region (Fig. 5.8.1) using the closest road intersection and subsequently converted into spatial points. I then used grid cells (1 km² cell size) across the southwestern Ontario region that captured these spatial points for a total of 699 sampling units. The standardized 1 km² cell size was assumed to capture regional avian and terrestrial median host ranges (Tewksbury et al. 1998, Rodewald and Bakermans 2006, Giraud et al. 2014, Rodewald et al. 2014).

For the urbanization categories, I extracted land-cover data per sampling unit from CanMap RouteLogistics Ontario v2013.3, DMTI (2013) and Southern Ontario Land Resource Information System (Smyth 2008) from the region (Fig. 5.8.1). I subsequently calculated landscape composition as the amount (%) of each land-cover factor per unit using Geospatial Modeling Environment 0.7.2.0 (Beyer 2012), and transformed values for principal component analyses (PCA) as: ln(p/(1-p)). I extracted the percent cover of eleven land-cover types per sampling unit: pasture, cropland, treed wetland, coniferous forest, mixed forest, deciduous forest, swamp, open water, urban greenspace, transportation, and built impervious surface. I calculated a road density index as the sum of road segment lengths × average speed limit, per sampling unit. I synthesized land-cover factors using principal components analysis (PCA), extracting PC1 as an urban index to approximate environmental variation across the study region (see Appendix 5.x.x). PC1 loaded positively and strongly (component loading > 69%) on pasture and built impervious land cover, as well as the road index, ranging in values from -1.082 to 0.536. PC values per unit were categorized using natural breaks (jenks) to categorize sampling units in equal representations of the land-cover gradient whereby individual units were classed as one of 20 categories of indexed land use (Hasse and Lathrop 2003, Atwood et al. 2004, Hansen et al. 2005). I chose to evaluate 20 categories because ~20 categories represented the maximum
number of categories to obtain as close as possible to 30 individual observations per category
distributed as evenly by natural jenks as possible along the rural-to-urban gradient.

5.3.2 Wildlife parasitism data

All wildlife individuals brought into the Toronto Wildlife Centre were examined upon intake. Standard intake protocols were consistent across clinical veterinarians to assess wildlife species identification and for parasitic infections. Ectoparasites and viral infections were identified using observed body condition based on external examination. Endoparasites (i.e., gastrointestinal parasitic infections) were identified using common fecal examinations. To address host sampling detectability biases along the urbanization gradient, I selected only parasitized individuals \( n = 3073 \) from the original dataset of the total individuals \( n = 26101 \) representing a random sub-sampling of all observed wildlife species (12% of total observations). I assumed that most animals were not brought to the Toronto Wildlife Centre due to disease-related body condition, though in some circumstances animals were likely detected due to apparent clinical symptoms caused by parasitic infections (i.e., rabies, canine distemper virus, heavy mange). I observed a significant yet almost nil relationship between sampling intensity and urbanization, as quantified using linear regression of number of observed infected wildlife explaining variation in human density (adj.-\( R^2 = 0.03, p < 0.0001 \)). To address parasite sampling detectability, I discarded observed data for parasites that did not fall into the standard protocols of intake examinations.

5.3.3 Quantification of alpha and beta diversity

I compared alpha and beta diversity measures of host and parasite communities at sampling unit and urban-category scales (‘levels’) to demonstrate how within-unit or category composition compares to community dissimilarity across an urbanization gradient. To quantify alpha diversity, I calculated species richness and Shannon’s diversity index per sampling unit or urban category. To quantify beta diversity, I utilized a recently developed method proposed by Legendre and Cáceres (2013) whereby total beta diversity is estimated by the total variance (\( S_{Total} \)) and dissimilarity (\( BD_{Total} \)) of a host or parasite community composition matrix.

First, \( S_{Total} \) (total sum of squares) was calculated where the squared differences from column means is calculated from Hellinger-transformed abundances of host and parasite species in a composition matrix. \( S_{Total} \) was further partitioned into species contributions to beta diversity (\( SCBD \)), again computed on data subjected to Hellinger transformation. Pre-hoc analyses used
both Hellinger and Chord transformation strategies and found values to be concordant. SCBD values indicate species that vary more (or less) than the mean across sites and/or exhibit large variations across the study area. SCBD values were then compared between sampling unit and urban-category scales for host versus parasite communities.

Second, $SS_{Total}$ was partitioned into local contributions of individual sampling units to beta diversity (LCBD). LCBD values indicate the sites that contribute more (or less) than the mean total beta diversity and are comparative indicators of sampling unit uniqueness. Large LCBD values indicate sites that have much different species compositions relative to other sites. LCBD indices were tested for significance by random permutations ($n = 999$) within the columns of the matrix to test whether the species are distributed independently of one another, among the sites. I evaluated the relationship between LCBD values (calculated at the urban-category scale only) and human population density using linear regression. Human population density was used here as a proxy of urbanization, independent of the urban index calculated using land cover and transportation factors.

5.4 Results

5.4.1 Overall diversity patterns

Variation in abundance and diversity was observed for both host and parasite community composition. Forty-four host species were identified and twenty-three parasite species were identified though in some cases hosts and parasites were not necessarily identified to the species-level. For urban-category scale, sampling units were consolidated into a category of urbanization given a PCA-derived index of urban land use. At the sampling unit scale, for hosts, $SS_{Total}$ was 515.3 and $BD_{Total}$ was 0.73; whereas for parasites, $SS_{Total}$ was 538.7 and $BD_{Total}$ was 0.77. Total beta diversity was significantly lower at the urban-category scale: for hosts, $SS_{Total}$ was 3.24 and $BD_{Total}$ was 0.17 and for parasites, $SS_{Total}$ was 2.73 and $BD_{Total}$ was 0.14 (Table 5.7.1).

5.4.2 Species contributions to beta diversity

At both scales, a subset of species contributed to total beta diversity greater than other species in host and parasite communities (Fig. 5.8.2). A greater number of species contributed to beta diversity at the urban-category scale than the sampling-unit scale for both host and parasite communities. A subset of the host community contributed to beta diversity well above the mean
of the 44 species (in decreasing order): Rock Pigeons (*Columba livia*), Eastern Gray Squirrels (*Sciurus carolinensis*), Raccoons (*Procyon lotor*), Striped Skunks (*Mephitis mephitis*), Red Foxes (*Vulpes vulpes*) and Big Brown Bats (*Eptesicus fuscus*) which had the highest SCBD index values. Many of these species, especially rock pigeons, are considered invasive urban adapters. Canine distemper virus, fleas, avian paramyxovirus-1 (AMPV-1), and sarcoptic mites contributed to parasite beta diversity as their SCBD values well surpassed the community average.

At the urban-category scale, Rock Pigeons have the greatest SCBD value (0.129) relative to the average SCBD for the host community. Similarly, for parasite community, sarcoptic mites have the highest SCBD value (0.086) contributing most to beta diversity relative to other species.

### 5.4.3 Local contributions to beta diversity

I tested the significance of local contributions to beta diversity by random, independent permutations within the community matrices. At the sampling-unit scale, the relative contributions of LCBD were in the range [0.0005 - 0.0024] for hosts versus [0.0006 - 0.0023] for parasites. No sites contributed significantly to overall beta diversity by host species at this scale, however, for parasite species, 18 sites were considered significant (*p* < 0.05) (Fig. 5.8.5a) and were in the range [0.002 – 0.022]. These values resided above the average sampling unit-scale parasite-LCBD value of 0.0014 and persist in the exurban and rural regions.

At the urban-category scale, LCBD values were in the range [0.014 – 0.125] for hosts versus [0.011 – 0.140] for parasites. Compared to alpha diversity measures, LCBD values for both hosts and parasites were inversely related to richness yet less related to Shannon’s diversity index (Fig. 5.8.3). Where species richness and Shannon’s diversity were lowest (i.e., rural areas, highly urbanized areas) LCBD values were often greatest. Host LCBD varied as a negative function of human population density (*n* = 20 PC categories, adj-*R*² = 0.456, *p* <0.001); a similar relationship was observed between parasite LCBD and human density (*n* = 20 PC categories, adj-*R*² = 0.375, *p* <0.001) (Fig. 5.8.4). For the parasite community, urban categories one, two, and four (related to relatively rural land cover) resulted in statistically significant LCBD-values in range of [0.106 – 0.140] (Fig. 5.8.5). These categories consisted of 60 total significant sites, although these sites were categorized by urban index and not by spatial association. For the host
community, urban categories one and four were significant based on LCBD values, consisting of 41 sites in the range [0.107 – 0.125].

5.5 Discussion

5.5.1 Overall findings

Urbanization causes the replacement of natural habitat with simplified habitat structures, increased resource availability, and altered trophic interactions, causing biotic homogenization of natural communities (Pickett et al. 1997, McKinney 2006, Smart et al. 2006, Devictor et al. 2008, Aronson et al. 2014). As geographically restricted native species with sensitive requirements maintain high extinction rates in urban areas, those widespread, broadly tolerant species that can live near humans, and benefit from their activities, will spread and become increasingly dominant (Brown 1989, Devictor et al. 2007, Ortega-Álvarez and MacGregor-Fors 2009). Corresponding shifts in urban wildlife community composition is expected to alter parasite infracomunities that reside within remaining synanthropic wildlife host populations relative to rural or more natural wildlife communities (Bradley and Altizer 2007, Lehrer et al. 2010, Watts and Alexander 2012). Subsequent patterns of disease caused by parasitism may impact the mortality and abundance of vulnerable populations. Though in some cases wildlife communities have been shown to reduce in diversity with urbanization (Smart et al. 2006, McKinney 2008), responses of parasite communities to anthropogenic environments is less well-known (Brearley et al. 2013, Martin and Boruta 2013).

My results provide evidence that urbanization affects wildlife-host beta diversity differently than parasite beta diversity. With increasing human density there was less contribution of host and parasite communities to beta diversity. Both communities demonstrated greater dominance by a few urban-exploiter species in areas that comprise human-dominated land cover. I argue that disturbed, impervious environments characteristic of urban areas facilitate the invasion of parasite species that can thrive at high abundances within and among high-density urban host populations. Cities create resource-rich conditions that facilitate introductions of generalist host species which may indirectly provide ‘habitat’ and resources for multi-host parasite generalists that can infect and thrive in unrelated host species (Woolhouse et al. 2001). Indeed, heterogeneous suburban landscape structure may permit the existence of both generalist and specialist hosts, indirectly affecting parasite community assembly processes.
These processes carry transmission risks as infected hosts often persist in close proximity to domestic-animal and human populations in suburban or urban environments. Therefore, if urbanization favours dominant traits in hosts, cities may have a significant role in the corresponding transmission dynamics and assembly processes of parasite communities that reside within generalist host populations. I describe these propositions below.

### 5.5.2 Biotic homogenization and host-parasite community assembly

I reason that urban environments foster biotic homogenization processes of generalist hosts and parasite communities, depending on non-native host invasion and density-dependent parasite transmission processes. I provide evidence that species contributions to total beta diversity for hosts and their parasites were skewed toward generalist species. High SCBD values indicate which species exhibit large variation across the study area. Wide variation in species diversity from rural to highly-urbanized environments is expected (Beissinger and Osborne 1982, Blair 1996, Devictor et al. 2007) and demonstrates a possible relationship between homogenization of environmental characteristics caused by human development and biodiversity patterns.

In my study, a significant number of the species that contributed to beta diversity (i.e., greater than average SCBD) are commonly found in cities. Rock Pigeons and Raccoons, for example, are synanthropic invasive wildlife species often infected by highly-infectious parasite species such as AMPV-1 and canine distemper virus, respectively. These host and parasite species were the most abundant in this dataset but were also dramatically variable in abundance across the region (as shown in chapter 4). Also, ectoparasites (i.e., fleas, mites) that are readily transmitted in high-density host conditions are common to wildlife and therefore contribute to overall beta diversity due to their wide variability in infecting multiple host species across the urbanization gradient. Urban environmental conditions that directly facilitate the invasion of non-native dominant, highly-abundant host generalists (McKinney and Lockwood 1999) may indirectly facilitate the transmission of parasite generalists within and between host populations (Havel et al. 2005). Therefore, processes of biotic homogenization of host and parasite assemblages could be caused by similar environmental pressures and altered community assemblages in residential, impervious habitat as opposed to agricultural or pristine habitat.
5.5.3 Urbanization and heterogeneity in host-parasite diversity patterns

My work supports the hypothesis that urban environments may have differential impacts on host versus parasite community assembly processes. In particular areas, I observed that the magnitude of variation in diversity was greater for parasite communities than for host communities. For example, in many urban categories (categories 12-18), I identified greater species richness and Shannon’s diversity from parasites than hosts suggesting that many hosts are infected with multiple parasites. Also, the gap between host and parasite richness and Shannon’s diversity values widens with urbanization, especially in the suburban-to-urban categories. These observations complement previous research: urbanization predicted greater poxvirus and coccidian infections in house finches (Giraudeau et al. 2014) and greater disease prevalence was observed in raccoon hosts with increased population densities (Prange et al. 2003).

Consequently, urban-induced processes of biotic homogenization may first impact wildlife host community structure, followed by cascading, population-level assembly processes for parasites transmitted within those host populations.

Furthermore, parasite communities may persist at higher diversity than host communities in urban environments. I observed that parasite groups which contributed to urban diversity often included viruses and ectoparasites, both of which likely transmit by direct contact and therefore most readily in high-density host populations. Urban conditions that sanction high-density host populations should also favour the transmission of multi-host, generalist parasites (Woolhouse et al. 2001, De Castro and Bolker 2005, Wright and Gompper 2005, Ferrari et al. 2011). Because parasites are dependent on hosts for resources, reproduction, and ultimately transmission to other hosts (Hoberg 1997, Morand and Krasnov 2010), it is expected that parasite communities will follow similar diversity patterns as their host communities. Indeed, ecological mechanisms in cities that favour transmission (i.e., inter- or intraspecific interactions, exposure to free-living parasites) also act as the underlying processes that structure parasite community assembly within and among host populations. However, generalist parasites may amplify in transmission rates, and consequently in diversity, via highly-frequent transmission in high host-density conditions (Woolhouse et al. 2001, Haydon et al. 2002, Buhnerkempe et al. 2015).

Additionally, synanthropic hosts are often resilient to parasitism as a trade-off for resource allocation and reproduction (Rohr et al. 2010). If urban hosts exhibit this
immunological trade-off, then hosts provide ample ‘habitat’ for multiple generalist parasites to readily invade. In effect, urban immunological tolerance could provide additional likelihood of parasite persistence and diversity within hosts. I therefore suggest that processes of biotic homogenization in cities may directly affect host communities but possibly carry indirect, augmented invasive responses to the assembly of respective parasite communities.

5.5.4 Suburban development and host-parasite community assembly

In particular, suburban environmental conditions may affect host and parasite beta diversity differently than rural or urban areas. My results demonstrated minimum LCBD values for sites and categories in suburban areas in indicating similar species compositions and weak contributions to regional beta diversity. At both the sampling unit- and urban-category scales, the only sites that contributed significant LCBD values were found in suburban, exurban, and rural areas. Additionally, the less unique the sampling unit or category in species composition, the greater the number of hosts that were parasitized in those suburban areas (Fig. 5.8.3a). Moderately-disturbed habitat (i.e., suburban environments) may favour high abundances of resource-exploiters while still allowing for the persistence of more sensitive, less abundant species (Blair 2001, Hansen et al. 2005, Merenlender et al. 2009, Luck and Smallbone 2011). With wider environmental heterogeneity assumed in suburban landscapes, diverse habitat conditions may provide a wider niche breadth for host communities, and, as a result, parasite communities, compared to rural or highly urban environments. Furthermore, suburban landscape structure possibly favours contacts between a variety of host species via shared wildlife resources or refugia (Bauer et al. 2013). Greater contacts among diverse host communities may homogenize the composition of parasite communities such as at forest edges or clustered resources such as garbage sites or bird feeders. Therefore, based on high community composition similarity for hosts and parasites within suburban areas, I argue that ecological processes that determine transmission in suburban habitat are different than rural or highly-urbanized areas.

5.6 Conclusions

In summary, the results of this study show that urbanization affects the variation in host and parasite diversity and patterns of community similarity. These patterns are most certainly the consequence of complex environment-host-parasite community assembly processes that vary over space and time and deserve closer attention. Directed surveys between different cities of
multi-host parasites in specific host communities would be valuable to investigate more mechanistic relationships between urbanization and wildlife disease dynamics (Buhnerkempe et al. 2015). In particular, the role of anthropogenic resources and resource provisioning has been recently highlighted as a significant factor linking urbanization to altered host-parasite interactions (Wright and Gompper 2005, Hartemink et al. 2014, Becker et al. 2015). Though this study did not take into consideration spatially-explicit dissimilarity measures of host nor parasite diversity, considerations of distance decay and community similarity between host and parasite assemblages may shed light on environmental versus taxonomic drivers of host-parasite community assembly and associated disease dynamics (Poulin 2003). Additionally, cities could favour the persistence of multi-host parasites in close vicinity to human populations. Because multi-host parasites often have zoonotic potential (Taylor et al. 2001, Anholt et al. 2012) with capability of infecting both animal and human populations, urbanization represents an emerging public health issue in the context of human-wildlife contact and transmission risks.

5.7 Tables

Table 5.7.1. Total beta diversity statistics for host and parasite communities in the southwestern Ontario region using $SS_{Total}$: the total sum of squares of the species composition data; and $BD_{Total}$: the index of beta diversity using a dissimilarity coefficient (Legendre and Cáceres 2013).

<table>
<thead>
<tr>
<th></th>
<th>Urban-category scale</th>
<th>Sampling-unit scale</th>
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<tr>
<td></td>
<td>$SS_{total}$</td>
<td>$BD_{total}$</td>
</tr>
<tr>
<td>Host</td>
<td>3.24</td>
<td>0.17</td>
</tr>
<tr>
<td>Parasite</td>
<td>2.73</td>
<td>0.14</td>
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5.8 Figures

Figure 5.8.1. Study area. Each 1 km² sampling unit ($n = 699$) was extracted from a grid of the study region southwestern Ontario (Canada) near Toronto. Each unit was provided an urban index value developed using PCA of land-cover values (%) quantified from the region, ranging from rural (category =1) to highly-urbanized land cover (category = 20). While Toronto represented the dominant urban area, urbanization values were also provided by other cities in the region.
Figure 5.8.2. Species contribution to total beta diversity (SCBD), ranked in decreasing order at the (a) sampling unit-scale, compared to (b) urban-category scale (1) for hosts and (2) parasites. SCBD values indicate which species exhibit large variations across the study area. At the urban-category scale, for both hosts and parasites, a few dominant species contributed to overall beta diversity relative to the rest of their respective communities.
Figure 5.8.3. Beta diversity (LCBD values) compared to alpha diversity metrics (richness (a) and Shannon’s diversity (b)) for host and parasite communities at the urban-category scale. Urban categories along the x-axis relate to increasing gradient of urbanization: highly rural (1) to highly urban (20). Species richness was generally greater in magnitude for parasite species than host species, especially in the moderate- to highly-urbanized categories. Shannon’s diversity also reflected this pattern. LCBD values, related to sites exhibiting strongly different species compositions, was greatest in rural to exurban categories, and lowest in suburban to urban categories.
Figure 5.8.4. Relationship between among-habitat beta diversity, calculated by LCBD, for (a) host species (circles) and (b) parasite species (diamonds). LCBD values are comparative indicators of site uniqueness (in this case, urban categories), and represent a partitioned statistic of the total sum-of-squares of the community composition data (Legendre and Cáceres 2013).
Figure 5.8.5. Maps for (a) sampling-unit versus (b) urban-category analyses showing the significant (<0.05) local contributions to beta diversity (LCBD) of the host and parasite community composition data. The size of circles indicates the overall LCBD values. At the sampling-unit scale, no sites were statistically significant derived from host community data. For the urban-category data, open circles represent significant sites derived by the parasite data only. Closed circles represent significant contributions shared by both hosts and parasites.
5.9 Appendix

Figure 5.9.1 PCA ordination of urbanization (BIS + RD + PAS) and natural cover (CF + DF + UG + SW) indices derived using land cover variables and a road density index extracted from individual sampling units. PC1 explained 69% and PC2 explained 58% of the variability to the land cover factors. Environmental variable codes: BIS (built-impervious surface); RD (road density); UG (urban greenspace); CF (coniferous forest); DF (deciduous forest); SW (swamp); PAS (pasture).
Chapter 6

6 Synthesis and Conclusions

6.1 Thesis synthesis

In this thesis, my goal was to investigate the overarching question: what is the spatial and ecological dependence of host-parasite interactions on landscape composition and configuration? Host-parasite interactions occur in specific locations at specific times and the nature, direction, intensity, and outcome of specific interactions depend upon their spatial and ecological contexts (Hess et al. 2002, Real and Biek 2007). While my work focused on three different disease systems (Lyme disease, coyote parasitism, and host-parasite communities), I consistently found variability in the spatial dispersion of host-parasite interactions. In these systems, spatial variability was often significantly associated with particular characteristics of landscape composition and configuration. Spatial and environmental heterogeneity have an impact on the structure, dynamics, and spread potential of host-parasite interactions by influencing the community and dispersal ecology of wildlife disease systems. My work also sheds light on two critical processes of disease ecology: the short versus long-distance dispersal of pathogens and the predominance of generalist – and potentially asymptomatic – versus specialist hosts and parasites in urban ecosystems. These ecological interactions are a fundamental driving force of host fitness, behaviour, immunity, population demographics, and species diversification (Holt et al. 2003, Hood 2003, Poulin and Mouillot 2003, Seabloom et al. 2015). Therefore, I have enriched our perception of the mediating ability of landscapes to shape the patterns and structuring processes of host-parasite interactions as well as the potential for the dispersal of parasitism throughout landscapes in natural populations. My work underscores the need to predict how host-parasite interactions and their associated transmission dynamics will become altered in the face of future habitat modifications. Below, I further discuss these contributions in the historical context of disease ecology and highlight individual contributions in the context of the current culture of ecology and epidemiology.
6.2 Contributions to disease ecology and landscape epidemiology

6.2.1 Historical context

A fundamental goal of ecology and evolutionary biology is to understand the role of the environment in the abundance and distribution of natural populations. As spatial structure of landscapes has a critical role in environmental heterogeneity, it is intuitive that landscape spatial heterogeneity should also shape the abundance and distribution of hosts and their parasites. Theoretical developments of spatial disease ecology are not new. In ‘The Ecology of Invasion of Plants and Animals, Charles Elton (1958) first describes how biological invasions can critically influence the abundance and persistence of natural populations using examples of viral, bacterial, or fungal infections. Extending from Elton’s foundational manuscripts, mathematicians Roy Anderson and Robert May further described that disease can have both stabilizing (Anderson and May 1978) and destabilizing (May and Anderson 1978) effects on populations under certain density-dependent and environmental conditions, extending to spatially-explicit disease control options (May and Anderson 1984). Robert Holt advanced his work on the role of parasite-mediated apparent competition (Holt 1977, Holt and Pickering 1985) by identifying the critical role of spatial heterogeneity in species coexistence (Holt 1984), later quantified in spatially-structured community-assembly processes in grassland ecosystems by Power and Mitchell (2004)). Nowadays, spatial dependence has become a fundamental aspect of understanding how and why variability in the frequency, character, and diversity of host-parasite interactions in natural and modified environments exists.

Several studies have highlighted both the role of environmental spatial heterogeneity and landscape structure on host-parasite dynamics. Yet in many early studies investigating landscape epidemiology have focused on climatic and landscape structure as determinants of disease patterns in natural populations. Langlois and Fahrig (2001) demonstrated that landscape structure has a greater effect on the pattern of distribution of hantavirus in Deer Mice than other ecological variables such as climate and seasonal change. Similarly, the prevalence and intensity of Baylisascaris procyonis, a Raccoon nematode, were significantly higher in a highly fragmented agricultural landscape (Page et al. 2001). Also, forest fragmentation and related landscape spatial heterogeneity was related to Lyme disease risk as a result of shifts in host community...
composition (Allan et al. 2003, Brownstein et al. 2005). More recently, literature reviews have highlighted the potential importance and promise of the field of landscape epidemiology in plant and forest systems (Plantegenest et al. 2007, Meentemeyer et al. 2012) and vector-borne diseases (Reisen 2010, Cumming et al. 2015) but still leaves room for the application of landscape epidemiology for infectious disease dynamics in vertebrate host populations (Lambin et al. 2010, Reisen 2010). This thesis therefore contributes to the field of landscape epidemiology by quantifying host-parasite interactions in vertebrate wildlife disease systems.

6.2.2 Landscape, dispersal processes, and disease spread

A central problem for disease ecology is to understand how changes in the degree of connectedness of individual hosts, different subpopulations of hosts, or communities of hosts influence the way in which diseases behave (Potterat et al. 1999, Brooks et al. 2008, McCallum 2008). Landscape composition and configuration has a significant role in facilitating or inhibiting connectivity and interaction among hosts. Functional connectivity models can be used to quantify and predict the degree to which environmental features facilitate or limit connectivity between host populations (Remais et al. 2010). The capability of pathogens to disperse over short versus long distances has critical implications for the rate of disease spread and capacity for invasion throughout a landscape (Lounibos 2002, Shigesada and Kawasaki 2002, Hoch et al. 2010). Introductions of parasites into naïve host populations is critical for parasite persistence. In chapter 2, I demonstrate the relative mediating capacity of short-distance dispersal of highly-competent rodent hosts (i.e., White-footed Mice) versus long-distance dispersal of poorly-competent ungulate hosts (i.e., White-tailed Deer) in the overall diffusion of Lyme disease across a fragmented landscape. Both components are critical in the eventual spread of the disease but are affected by landscape structure differently. Theoretically, stochastic invasion events can be predicted by random or rare introductions by long-distance dispersal (Duke-Sylvester et al. 2013, Papaix et al. 2014). In the ecology of Lyme disease, if susceptible tick or mice populations first established a distant destination patch, a random dispersal event of an infected rodent into this patch could lead to the persistence of the bacterium in this new location. Yet, it is more likely that the bacterium will slowly disperse through the landscape over multiple tick and mice generations via short-distance dispersal events.
6.2.3 Landscape, community assembly, and disease dynamics

Multi-host parasites have been implicated in the emergence of new diseases in wildlife and humans, yet little is known about the ecological factors that influence the host range of parasites in natural populations (Woolhouse 2001, Buhnerkempe et al. 2015). In my work, I highlighted the functional role of urban-related land-use and land-cover change on host and parasite community assembly and asymmetry in host-parasite interactions. I provided evidence that cities may functionally shift suburban and urban host and parasite communities towards dominance by generalist, reservoir hosts and multi-host parasites. Parasites that infect more than one host species are by definition likely to be encountered in several host populations, some of which may constitute infection reservoirs (Haydon et al. 2002). Often, wildlife reservoirs can allow for the persistence of multi-host parasites with little impact on population mortality or individual effects of disease. Conversely, specialist parasites that have adapted transmission between a specific host species are less likely to be encountered by multiple hosts. Spillover of parasites from robust reservoir host populations to susceptible hosts sensitive to mortality by infection represents ecological, epidemiological, and conservation concern (Thompson 2013). Determining how hosts enable persistence and which hosts are crucial for the persistence of these multi-host parasites is essential for the design of effective control measures (Wood et al. 2012, Viana et al. 2014). Therefore, in degraded environments, processes of biotic homogenization should favour the persistence of resilient generalist hosts and, indirectly, the persistence of multi-host parasites. Specialist host-parasite relationships may be sensitive to disturbance and become locally extinct or extirpated. As urban ecology is a frontier science, my work accentuates the potentially-homogenizing ecological processes that underlie disease transmission systems in urban landscapes.

Furthermore, many recent studies have demonstrated impacts of landscape spatial heterogeneity on host-parasite community assembly processes. In the mid-to-late 1990s, Richard Ostfeld and Felicia Keesing explored the role of host community composition on the prevalence of the Lyme disease-causative bacterium, *Borrelia burgdorferi*, in tick-vector and host populations (Schmidt and Ostfeld 2001, LoGiudice et al. 2003a, Keesing et al. 2006). These ecological investigations prompted a possible ‘dilution effect’ whereby greater host species richness often found in larger, pristine habitat patches should reduce the presence and impact of
disease than less species-rich communities often found in smaller, degraded habitat patches (Allan et al. 2003, Keesing et al. 2010). This ‘biodiversity-buffers-disease’ hypothesis has been met with both praise and scrutiny (Randolph and Dobson 2012, Johnson et al. 2013, Ostfeld 2013) but represents active debate in the field (Pfäffle et al. 2015) and is being explored in many spatial contexts (Estrada-Peña 2009, Werden et al. 2014, Huang et al. 2015). Ultimately, community composition and uneven patterns of generalist versus specialist host and parasite species are the foundations for the maintenance of reservoir populations of parasites. Future work should focus on sentinel host species (generalist) versus specialist host-parasite relationships to elucidate how urbanization and land-cover change may favour wildlife reservoir populations.

6.2.4 Network models and landscape epidemiology

Parasite dispersal is facilitated by both host and vector movements, both of which are mediated by the composition and configuration of the landscape (Madhav et al. 2004, Wang 2004, Baguette and Van Dyck 2007). Yet, monitoring the movement of wildlife hosts and their endoparasites or ectoparasites delivers exceptional sampling challenges. Landscape connectivity models can help elucidate dispersal dynamics (Bélisle 2005) especially in multi-host or vector-borne disease systems. I used network models in chapter 2 (generalized network connectivity model) and in chapter 4 (bipartite interaction network models) to quantify dispersal and interaction processes of hosts and parasites, respectively. Recently, network analysis has been used with increasing frequency for the study of parasitism in natural systems (Poulin 2010). Whether they have been used to predict the spread of disease through social or sexual contacts (Vander Wal et al. 2012, Zohdy et al. 2012) or to quantify food-web stability in host-parasite interaction webs (Tylianakis et al. 2007, Lafferty et al. 2008, Cagnolo et al. 2011), there has been a growing interest in the characterization of the spatial structure of populations in heterogeneous landscapes using network models (Hanski and Ovaskainen 2000, Brooks et al. 2008).

Network connectivity models are providing more flexible parameterization to include spatially-explicit demographic and environmental data for more biologically realistic measures of disease connectivity and spread modeling. In my work, hybridized demographic-dispersal connectivity models provided a robust, biologically-sensitive evaluation of the demographic,
environmental, and spatial dependencies of vector-borne disease spread potential. This hybrid network model could be readily applied to other disease systems where patches can be represented as spatially-discrete entities and where dispersal of hosts, vectors, or disease is a vital process of the system. Networks are especially applicable in quantifying the impact of connectivity on disease risk in metapopulations (Johst et al. 2002, Heard et al. 2015, Huang et al. 2015).

Similarly, in chapter 4, bipartite mutualistic networks provided an effective and holistic analysis of variation in host-parasite interaction structure over a geographic gradient. As disease systems are extremely complex, there remains significant prospects in the use of network models to predict how a host or parasite community will respond to perturbations such as invasion or removal of particular host or parasite species (Poulin 2010).

6.2.5 Applications to disease management

The need to integrate generalized ecological principles into disease management has become increasingly important in the past few decades as we are facing a rising incidence in disease emergence and re-emergence events across the globe (Daszak et al. 2000, Patz et al. 2004, Rhyan and Spraker 2010, Morse et al. 2012). Early detection of the range expansion of sentinel hosts, key vectors, and invasive parasites is necessary for implementation of prevention and control programs largely centered on public education and outreach (Narrod et al. 2012). From my work in chapter 2, I identified the role of stepping stones as functional structural characteristics in facilitating not only the diffusion of the hosts, but attached vectors and pathogens. In suburban or agricultural landscapes where human communities reside, stepping stone habitat patches may act as spatially-targeted management locations for monitoring (Remais et al. 2010, Hagenlocher et al. 2014), oral-baited vaccination of rodents (Tsao et al. 2012, Richer et al. 2014, Beasley et al. 2015), or intense public awareness and education (Lambin et al. 2010). Prospective work using spatially-explicit disease monitoring techniques will help target particular landscape composition or configuration characteristics that could act as proxies or indicators of particular disease processes.

Then, in chapters 3, 4, and 5, my results highlighted the strength of particular host species in maintaining potentially zoonotic pathogens (i.e., coyotes infected with *Echinococcus* spp. or
Toxocara spp.) and host-parasite interaction structure (i.e., pigeons, raccoons, eastern gray squirrels, songbirds) in developed and suburban-dominated habitat. Based on my results, disease managers and public health officials should focus monitoring efforts in exurban environments where rapid shifts in species interactions may be observed as rural or pristine habitat rapidly converted to high-density residential dwellings. Because cities may favour the persistence of reservoir host populations (Bradley and Altizer 2007, Brearley et al. 2013), the persistence of multi-host pathogens will likely also be favour in those hosts. Urban or suburban environments may therefore create greater potential for pathogen spillover to domestic animal or human hosts. Based on my work, these relative risks are likely quite low. However, in future urbanized habitat, pathogens may eventually reside in multiple wildlife host populations (Thompson 2013).

In consideration of altered trophic interactions and host-parasite relationships, urban planners and policy makers could incorporate ecological investigations to better predict how projected patterns and designs of settlement may affect host populations; and how disease patterns within- and between those populations may change along gradients of urbanization. If human-wildlife encounters also increase in with future urban settlements, there is a potential for greater zoonotic infection risks. As my work demonstrates such significant links between landscape structure and altered host-parasite interactions, I ultimately stress the inclusion of ecologically-informed, multi-scale research to further inform veterinarians, wildlife managers, urban planners, and public health officials in disease detection, monitoring, and surveillance.

6.3 Future prospects and recommendations

Considering the rapid emergence and re-emergence of infectious diseases in natural and human populations, there is a vital need for ecologically-grounded, multi-scale methodologies towards understanding the mechanisms that underlie patterns of host-parasite interactions in spatially-heterogeneous contexts. While my work has focused on global change by landscape modification, there will most certainly be additional and potentially synergistic effects of climate change on disease systems over multiple scales (Lafferty 2009, Molnar et al. 2013, Garza et al. 2014, Simon et al. 2014). It will be critical to therefore further develop dynamic models that consolidate laboratory-controlled investigations of isolated host-parasite-environment interactions with empirical observations from the environment (Cumming et al. 2015).
Increasingly accessible remotely-sensed data sets will help further link landscape structure, improvements in modeling complexity, and technological advances, landscape epidemiology represents a frontier discipline providing much opportunity for discovery and contributions to ecology. Though I did not explicitly address temporal factors in this thesis, spatio-temporal surveillance and data collection is vital in making more robust links between landscape change and shifts in host-parasite ecology (Waller et al. 2007, Jousimo et al. 2014).

As landscape connectivity clearly has a beneficial role in predicting disease spread, landscape genetics has provided robust insights on the genetic distance between host and parasite populations (Manel et al. 2003, Storfer et al. 2007). Landscape genetic methods are shedding light on the mediating effect of landscape structure on disease persistence and spread (Archie et al. 2009, Biek and Real 2010). Genetic approaches have also helped demonstrate the significant effect of agricultural barriers on the spread of rabies in a Raccoon population (Rioux Paquette et al. 2014) and the ability of rivers to impede the potential diffusion of chronic wasting disease in White-tailed Deer populations (Blanchong et al. 2008). Landscape genetics may therefore help predict the likelihood of short- versus long-distance dispersal events in the spread of parasites via abiotic or host movement.

Resource-based habitat approaches may prove useful for investigating the disease processes affected by environmental heterogeneity. The high abundance and predictability of food resources (i.e., bird feeders, supplemental feeding stations, accessible garbage/refuse sites) across space and time can make them accessible components of wildlife diets, leading to increased or decreased infection risk for wildlife and humans depending on the nature of provisioning and the particular host-parasite interaction (Hall et al. 2007, Becker et al. 2015). In Chapter 3, I demonstrate how dietary behaviour in coyotes shifts towards anthropogenic resources in urban sites versus rural sites. In chapters 4 and 5 I also demonstrate the role of suburban and urban land cover in structuring host-parasite interactions towards a skewed, core group of generalist host and parasites. Similarly, host and parasite communities were relatively similar in suburban areas relative to strictly rural or high-urban land cover. These results shed light on the likelihood that anthropogenic resources influence the spatial clustering of hosts towards high transmission potential within and between host populations.
Many research groups argue that a resource-based habitat concept (RBHC) is a better strategy to incorporate functional habitat and movement processes as a reliable mechanistic, spatially-explicit model for disease risk prediction and mapping (Becker and Hall 2014, Hartemink et al. 2014, Becker et al. 2015). The RBHC offers a framework to identify systematically the different ecological resources to multiple landscape features and other environmental factors. This approach assumes in a bottom-up manner that functional habitat is represented as an entity of functional space that results from the overlap of resources and scale of movements between them. Researchers argue that the RBHC framework may act as a bridge between existing mechanistic and statistical modelling approaches and may lend itself as a new approach in landscape epidemiology of vertebrate disease transmission systems (Becker et al. 2015).

Finally, the pursuit to bridge ecological and epidemiological modeling efforts to better predict potential dispersal of hosts and parasites is becoming increasingly popular. For example the basic reproduction number (\(R_0\)) - a critical epidemiological concept - describes the average number of secondary cases caused by one infectious individual in a closed population of susceptibles. As the likelihood of contacts between infected and susceptible is largely spatially-dependent, a value of \(R_0\) can be applied to landscape connectivity as a quantification of the likelihood an infected patch to maintain infected hosts and allow potential. Efforts in network modeling have shown that local spatial structure has a significant effect on disease invasion and how dependent \(R_0\) is on the structure of the network (Keeling 1999). This spatially-explicit value could be further incorporated into a quantification of the ‘force of infection’ (i.e., a measure of susceptibility of individuals or habitat patches to parasite invasion) (Dobson and Foufopoulos 2001, Balcan et al. 2009, Meentemeyer et al. 2008) and for spatially-targeted monitoring programs for sylvatic (naturally-cycling), zoonotic, or vector-borne diseases (Hartemink et al. 2008, Estrada-Peña et al. 2014, Papaix et al. 2014).

6.4 Final conclusions

The hypotheses and design of my thesis are founded by a long history of significant scientific achievements in the exploration of host-parasite interactions and disease emergence. By working on a variety of disease systems, I have elucidated some ways in which our changing landscapes
are affecting the assembly processes of interactions between hosts and their parasites and the potential dispersal of vector-borne disease agents into naïve landscapes. Infectious disease outbreaks are inextricably linked to ecological dynamics of complex disease systems. In the future, further ecological investigation can only advance our understanding of global change and infectious disease risks, and greater ecological sophistication is called for in epidemiological theory to explain zoonoses, pathogen spillover, and relationships between infectious diseases and biodiversity. While disease dynamics are established threads of the overall fabric that makes up the field of ecology, spatial elements of parasite ecology and evolution still need to be fully explored. Ultimately, while this concluding chapter has punctuated only a few milestones in the development and current state of disease ecology and landscape epidemiology, these critical advances have paved some foundational hypotheses of disease ecology as part of the ongoing crusade to establish a generalized theory of ecology.
References


Beyer, H. L. 2012. Geospatial Modelling Environment (Version 0.7. 2.1). Spatial Ecology, LLC.


Eisen, R., and N. Wright. 2001. Landscape features associated with infection by a malaria parasite (Plasmodium mexicanum) and the importance of multiple scale studies. Parasitology 122:507-513.


Estrada-Peña, A. 2009. Diluting the dilution effect: a spatial Lyme model provides evidence for the importance of habitat fragmentation with regard to the risk of infection. Geospatial Health 3:143-155.


disease emergence and its drivers: spillover of bat pathogens as a case study.  
Philosophical Transactions of the Royal Society of London B: Biological Sciences  
367:2881-2892.


Iowa.


network: tracking lice in a wild primate (Microcebus rufus) population to infer social  