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# Urbanization and the ecology of wildlife diseases

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**Urbanization is intensifying worldwide, with two-thirds of the human population expected to reside in cities within 30 years. The role of cities in human infectious disease is well established, but less is known about how urban landscapes influence wildlife–pathogen interactions. Here, we draw on recent advances in wildlife epidemiology to consider how environmental changes linked with urbanization can alter the biology of hosts, pathogens and vectors. Although urbanization reduces the abundance of many wildlife parasites, transmission can, in some cases, increase among urban-adapted hosts, with effects on rarer wildlife or those living beyond city limits. Continued rapid urbanization, together with risks posed by multi-host pathogens for humans and vulnerable wildlife populations, emphasize the need for future research on wildlife diseases in urban landscapes.**

## Introduction

Urbanization is increasing at a global scale, with ecological consequences that extend beyond city boundaries (Box 1). Defined as growth in the area and numbers of people inhabiting cities, urbanization generates landscapes dominated by built-up structures for human use [1,2]. Most studies of the ecological impact of urbanization focus on patterns of biodiversity loss, with declines in species richness from rural areas towards the urban core documented across multiple taxonomic groups [3,4]. More recently, ecologists have begun to explore the mechanisms by which urbanization affects biodiversity, including processes related to resource competition, altered trophic interactions and disease [2,5].

An increasing number of studies point to links between human activity and the emergence of wildlife diseases [6–8], yet only a few address how wildlife–pathogen interactions respond to urban land use. However, urbanization can influence shifts in host geographical ranges and densities [9], interspecific interactions [5] and contamination of the environment with pathogens [10]. For example, across 176 foraging sites for wading birds in coastal Florida, a highly pathogenic nematode occurred only at sites disturbed by stream engineering and nutrient fluxes [10]. Many of the above processes have been studied in the context of agricultural land use or forest edge habitats, and are also relevant to pathogen spread in urbanized landscapes.

Here, we identify key hypotheses concerning the role of urbanization in the transmission and impacts of infectious diseases in wildlife populations. To capture a range of mechanisms and changes in their intensity, we consider patterns that occur across the urban–rural gradient (Box 1), in some cases focusing on wildlife species that inhabit both the urban core and surrounding suburban and rural areas. Understanding the ecology of wildlife pathogens in urban environments will become increasingly important for managing disease risks to wildlife and, in some cases, humans. Indeed, many pathogens are capable of infecting multiple host species [11,12], and some pose serious threats to human health and already vulnerable wildlife populations [13]. Finally, we emphasize several priorities for future research, including identifying those pathogens for which urbanization is likely to have the greatest impact.

## Wildlife communities in urban environments

Urbanization dramatically alters the composition of wildlife communities, leading to biodiversity loss [3,14] and increases in the abundance of species that thrive in urban areas. Indeed, one recent study exploring the patterns and causes of ‘biotic homogenization’ found a negative relationship among human population size, urban land use and species richness across all major taxonomic groups in North America [15]. This effect is due, in large part, to simplified habitat structures [2], increased resource availability [4] and altered trophic interactions [5].

Many animal species disappear from cities altogether, occur at low abundance, or are restricted to parks, forest fragments and other less intensely used areas. Because most wildlife parasites (especially those restricted to one or a few host species) will also be missing from urban centers, an important consideration is how relevant urbanization is to the ecology of wildlife diseases. The answer involves at least three crucial processes (Table 1). First, for diseases such as toxoplasmosis or rabies, which affect urban-adapted wildlife species, infection dynamics can change across a gradient of habitats, in some cases leading to increased prevalence in urban or suburban environments. Second, rarer wildlife species, such as many wood warblers or flying squirrels and other small rodents, which persist within city parks or surrounding natural areas, can be affected negatively by pathogens maintained in urban-adapted hosts (Box 2). The increased dominance of a few key host species, and conditions that favor interspecific contact rates, could cause declines of rarer wildlife through competition mediated by multi-host parasites [12,16].

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### Box 1. The scope and study of urbanization

Over 65% of the human population worldwide will reside in cities by 2025 and those areas will double in land coverage over the same period [49]. Most of this shift will occur in the developing world (Figure 1), where the urban population is expected to grow to four billion by 2030 [49]. Urban population growth and movement into cities closely mirrors social and economic reforms, such that shifts in human populations worldwide from rural to urban areas often reflect changes in employment opportunities, access to education and healthcare, and environmental degradation outside of cities.

The spread of urban centers results in rapid and dramatic landscape-level changes that are relatively permanent over ecologically significant time periods. Thus, urbanization signifies a drastic form of land conversion typified by dense human habitation, transportation, industry and associated infrastructure. In addition to greater human population density, changes that occur along urban-rural gradients include the loss of biota and natural habitat, increased densities of roads, buildings and other impervious surfaces, and microclimatic shifts (e.g. heat island effects).

Urban ecologists examine the interactions and feedbacks between human activities and ecological systems in urban landscapes [1,2]. One major challenge involves the need to categorize land-use types consistently based on quantifiable patterns that are relevant to ecological processes [50]. Often, urbanized areas are classified subjectively using terms such as urban, suburban, exurban and rural (see Ref. [50] for discussion). From a quantitative perspective, human population density and impervious surface coverage are commonly used to quantify the degree of urbanization and facilitate among-site comparisons. Census data on socioeconomic conditions at study sites can provide additional information relevant to underlying ecosystem processes. For example, higher family incomes and older buildings were strong predictors of plant species diversity in the Phoenix metropolitan area in Arizona, USA [51]. This association is probably

related to higher priority placed on gardening and landscaping among families with higher incomes, and was also a significant predictor of wildlife biodiversity in some taxonomic groups.

An important consideration in urban landscape studies is the appropriate spatial scale for analysis. Conducting analyses at multiple scales is essential for detecting the relative importance of key landscape variables. In the case of infectious disease risk, host abundance and diversity, vector activity and pathogen survival in the environment will probably be affected by urban landscape measures at different spatial scales [52]; these scaling patterns will further depend on host dispersal abilities, host and vector habitat preferences, and the mode of parasite transmission.



Figure 1. The urban sprawl of Rio de Janeiro, Brazil.

Third, reduced biodiversity of urban wildlife can influence the transmission of some vector-borne diseases through a process termed the 'dilution effect' [17–19]. Here, high host species richness can lower parasite transmission if vectors feed on multiple host species varying in competence with respect to contracting, amplifying and transmitting the pathogen. The reverse situation could occur in urbanized areas if low host diversity increases the proportional abundance of key reservoir hosts.

Lyme disease, caused by the bacterium *Borrelia burgdorferi*, is the best studied example of the dilution effect. This pathogen is transmitted by *Ixodes scapularis* ticks that feed on a large number of mammal species. Studies in suburban environments of northeastern USA, characterized by high forest-edge and low mammalian biodiversity, indicate that a greater proportional abundance of the most competent reservoir for *B. burgdorferi* (the white-footed mouse, *Peromyscus leucopus*) is linked to increased infection prevalence in ticks, mice and humans [18,20]. Reduced host species diversity probably has a similar role in other wildlife pathogens (Table 1) such as West Nile virus (WNV) [21], although it is also possible that low host diversity in the extreme urban core could exclude competent reservoir hosts or vectors [22]. Further studies are needed to assess the general importance of the dilution effect and its significance for pathogens in urbanized areas.

#### Resource provisioning, host contact rates and susceptibility to infection

Many urban-adapted species occur at much higher densities in urban and suburban environments than in less-disturbed

areas [4]. Abundant resources not prone to seasonal fluctuations, either through accidental (e.g. household waste) or intentional (e.g. bird feeders) provisioning, support these populations. Such high population densities can elevate contact rates within and among wildlife species, and favor the transmission of parasites spread by direct contact or oral–fecal routes (Table 1). In response to resource provisioning, increased birth rates among urban-adapted species could provide further opportunities for parasite transmission by increasing the abundance of susceptible juvenile hosts (e.g. Ref. [9]).

The spatial distribution of resources also influences host aggregation and contact patterns. One study of macroparasite infections in wild raccoons *Procyon lotor* showed that experimentally clumping resources resulted in elevated host densities and increased prevalence of the raccoon roundworm *Baylisascaris procyonis*, particularly among juvenile animals [23]. This approach also provided evidence for greater parasite species richness per individual host under the clumped resource treatment, supporting a direct effect of aggregated resource distributions on within-species contact rates and parasite transmission.

Concentrated resources also influence host migration into urban landscapes and among-species contact rates, including contact between humans and wildlife hosts. One example is provided by the increasing number of red foxes *Vulpes vulpes* in European cities (Figure 1) [24]. Red foxes are the primary sylvatic reservoir of *Echinococcus multilocularis*, a tapeworm that causes liver disease in humans. The parasite is transmitted to intermediate hosts (typically rodents) by the ingestion of eggs deposited in fox

**Table 1. Examples and mechanisms illustrating effects of urbanization on the ecology of wildlife–parasite interactions**

Host	Pathogen <sup>a</sup>	Locality	Effects on host or parasite biology	Refs
<b>Biodiversity loss and the dilution effect</b>				
White footed mouse <i>Peromyscus leucopus</i>	<i>Borrelia burgdorferi</i> (Lyme disease)	Northeast North America	Forest fragmentation, often near suburbs, linked with greater densities of infected ticks and white-footed mice; can result from loss of predators and less-competent hosts	[18,20]
Passeriformes and other vertebrate hosts	WNV	North America	Seroprevalence in wild songbirds higher in areas densely populated by humans; non-passerine bird diversity associated with lower infection rates in mosquitoes and humans	[21,64]
<b>Resource provisioning and contact rates within urban-adapted species</b>				
Raccoon <i>Procyon lotor</i>	<i>Baylisascaris procyonis</i> (raccoon roundworm); other endoparasites	Northeast USA	Higher raccoon abundance and birth rates in urban–suburban areas; clumped resources increase within-species contact rates, leading to higher parasite richness and increased <i>B. procyonis</i> prevalence	[9,23]
House finch <i>Carpodacus mexicanus</i>	<i>Mycoplasma gallisepticum</i> (mycoplasmal conjunctivitis)	East North America	Hosts more abundant in regions of high human population density; aggregation at bird feeding stations could increase contact rates and pathogen transmission	[65]
Red fox <i>Vulpes vulpes</i>	<i>Echinococcus multilocularis</i> (tapeworm)	European cities	Shifts in dietary behavior and lack of suitable intermediate hosts reduces prevalence in foxes inhabiting urban centers; risk to humans could increase owing to encounters with urban-dwelling foxes	[24,26]
Mule deer <i>Odocoileus hemionus</i>	CWD	CO, USA	Disease more prevalent in highly developed and residential areas, possibly owing to host crowding and aggregated food resources	[66]
<b>Interactions with reservoir hosts in or surrounding urbanized habitats</b>				
Gray fox <i>Urocyon cinereoargenteus</i>	CPV	San Francisco, CA, USA	Greater seroprevalence in wild canids captured in urban zone surrounding park; could be caused by direct or indirect contact with domesticated dogs	[28]
Allegheny woodrat <i>Neotoma magister</i>	<i>B. procyonis</i>	East North America	Declines in woodrat from fatal <i>B. procyonis</i> infections linked with exposure to raccoon feces; exposure could increase at the periphery of urban–suburban development	[46]
Red squirrel <i>Sciurus vulgaris</i>	Squirrel paramyxovirus	UK	Non-native gray squirrels introduced highly lethal virus; food provisioning in urban–suburban environments could increase squirrel contact rates and influence pathogen-mediated declines	[54,56]
<b>Environmental contaminants, host stress, and susceptibility to infection</b>				
Southern sea otter <i>Enhydra lutris nereis</i>	<i>Toxoplasma gondii</i> (meningoencephalitic disease)	West coast of USA	Infections higher in areas of maximum freshwater runoff associated with regions of high human density or activity; probably owing to exposure to cat feces via sewage contamination	[31]
Great tit <i>Parus major</i>	Stress biomarkers (no specific pathogen)	Southwest Sweden	Measure of oxidative stress affected by air pollution increased from rural–urban locations; greater stress and reduced plumage condition could be associated with susceptibility to disease	[67]
European blackbird <i>Turdus merula</i>	Acute stress response (no specific pathogen)	Munich, Germany	City-born hosts showed reduced acute stress response relative to forest-born conspecifics; indicates that species capable of evolutionary adaptation might thrive in urban environments and could be less affected by infectious diseases	[68]

<sup>a</sup>Abbreviations: CPV, canine parvovirus; CWD, chronic wasting disease; WNV, West Nile virus

feces. Although human cases are widely restricted to rural areas in northern Europe, where outdoor work and pet ownership are significant risk factors [25], migrations of infected foxes into urban areas might elevate human infection risk in cities. Current evidence suggests that there is little impact to humans living in urban areas; however, because *E. multilocularis* has a long incubation period (decades in some patients), long-term surveillance will be required to assess shifts in human infections associated with fox movements. The red fox–*E. multilocularis* system further emphasizes that multiple mechanistic drivers associated with urbanization can influence patterns of infectious disease risk. One recent study showed that, although fox populations are increasing in Zurich, Switzerland, the prevalence of *E. multilocularis* within those populations is not [26]. Transmission of *E.*

*multilocularis* could decline if foxes consume fewer intermediate hosts (as prey) in favor of human-generated resources in cities.

Resource provisioning in urban environments also affects host susceptibility and responses to both existing and introduced pathogens, mediated through effects on physical condition and immune defenses. For example, animals that are malnourished owing to low protein intake can become immunosuppressed, shedding more parasite eggs in feces and suffering higher rates of mortality following infection [27]. Thus, although elevated food resources for urban-adapted species could increase contact rates and pathogen transmission, supplemental feeding might also improve host condition, increase immunity to infection and decrease pathogen impacts on host survival and reproduction.

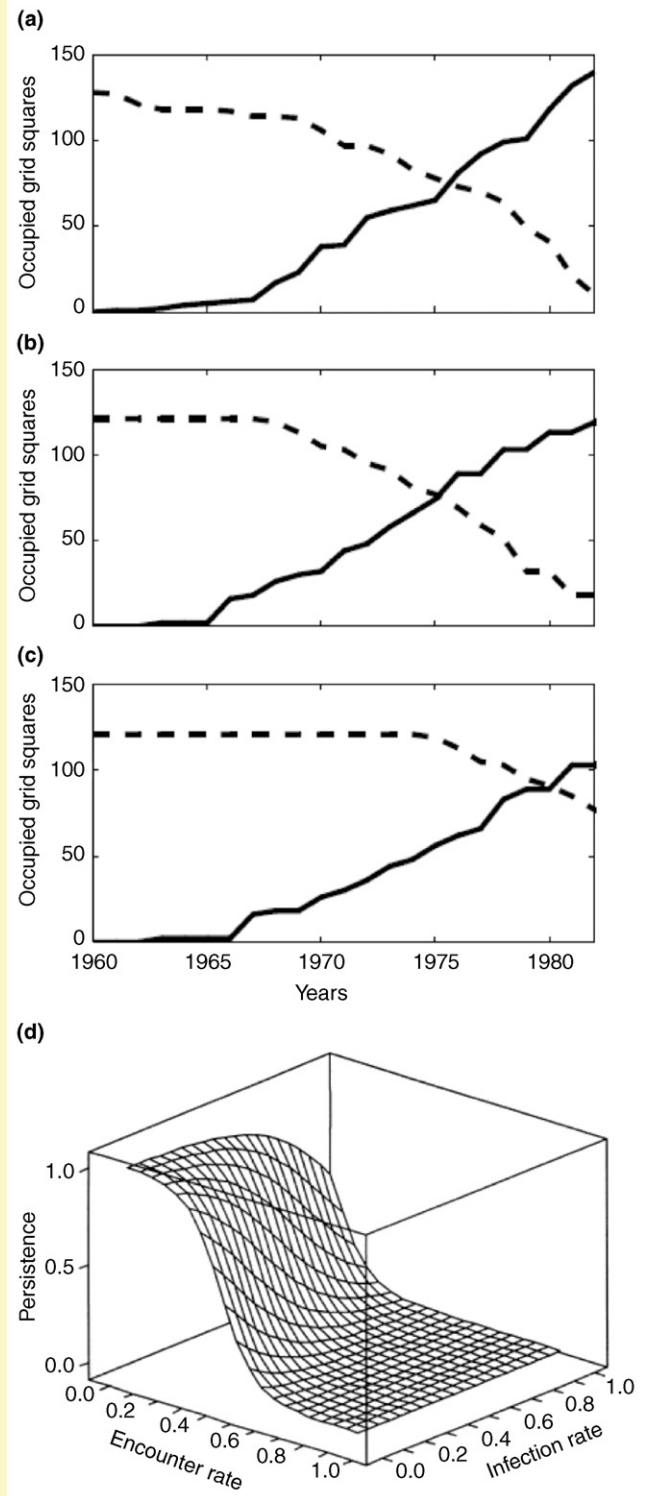
### Box 2. Red squirrel–gray squirrel paramyxovirus in the UK

The populations of red squirrels *Sciurus vulgaris* (Figure 1a) throughout the UK have declined following the introduction of gray squirrels *Sciurus carolinensis* (Figure 1b), a species endemic to North America [53]. Although reductions in red squirrel populations can result from direct competition for resources between the two species [53], gray squirrels also introduced a squirrel paramyxovirus that is highly lethal to red squirrels (but causes no discernible pathology in gray squirrels; [54]).

Results from a model developed by Tompkins *et al.* [55] support the conclusion that observed red squirrel declines are consistent with both direct and indirect competition with gray squirrels (Figure 1la–c). Using a stochastic individual-based model and applying this model to real landscapes, Rushton *et al.* [56] explored the role of the paramyxovirus virus in red squirrel population reductions. The results indicate that apparent competition between red and gray squirrels led to local loss of red squirrels under a large range of parameter values and assuming low levels (10%) of infection in gray squirrels. The population-level effects of the pathogen were most strongly influenced by interspecific encounter rates (Figure 1ld), suggesting that increased contact between infected and healthy individuals could raise the risk of disease-mediated population loss. Such encounter rates could increase around urban areas if food provisioning leads to greater contacts within and between host species. The introduction of this urban-adapted animal species and the subsequent spread of paramyxovirus in red squirrels illustrates the potential synergistic effects of urbanization and pathogen-mediated competition on the abundance of more vulnerable wildlife hosts.



**Figure 1.** Red squirrels (a) versus gray squirrels (b) in the UK.



**Figure 2.** The number of 5-km grid squares occupied by gray (solid line) and red (dashed line) squirrels between 1960 and 1982 in Norfolk, UK (a) Observed data adapted from Reynolds (1985). (b) Model predictions incorporating direct and indirect interspecific competition. (c) Model predictions assuming only direct competition. Model results shown in (ii) more closely replicate the field data both in timing and amplitude, providing evidence for the role of infectious disease in the population dynamics of both species. Model simulations (d) indicate that the persistence of red squirrels declines with increasing encounter rates between squirrel individuals and the rate of infection given an encounter occurs. Reproduced with permission from Ref. [55] (b) and Ref. [56] (c).



**Figure 1.** Many wildlife species can be attracted to urban centers, potentially bringing infectious disease with them. The European red fox, the major sylvatic host for *Echinococcus multilocularis*, has become increasingly urban-adapted in cities such as Zurich and Geneva, Switzerland. Many factors, including primary and intermediate host densities and host dietary behaviors, influence the prevalence of infection in urban centers, and infection risks for domestic animals and humans. These young European red foxes were born and raised in a residential garden in the south of England, an area outside of the endemic region for *E. multilocularis*. Reproduced with permission from Paul Cecil, University of Sussex, UK.

### Pathogen exposure, pollution, and stress

Cities serve as significant hubs of pathogen introductions (Box 3) and as sources of infection for wildlife that exist at the periphery of urban centers [28,29]. For example, infectious oocysts of the protozoan parasite *Toxoplasma gondii*, the causative agent of toxoplasmosis, are shed in the fecal material of domesticated and wild felids. This pathogen also causes infections in other mammals associated with both urban landscapes and more natural habitats (e.g. Ref. [30]). For example, *Toxoplasma* infections have been linked to southern sea otter *Enhydra lutris nereis* mortalities off the coast of California, USA [31]. Infection rates in otter populations were three times higher in areas of maximum freshwater runoff along the Californian shoreline, most of which were associated with regions of high human density. This association is probably a result of water contamination by cat feces and parasite amplification by benthic filter feeders that comprise a major component of sea otter diets [31].

Changes in stress and pollution along the urban–rural gradient could also affect host susceptibility to infectious diseases (Table 1). Among vertebrate animals, chronic stress can lower resistance to infection and intensify the harmful effects of pathogens through effects on the host immune system [32,33], mediated, in part, by glucocorticoid hormones, such as cortisol. Increased interspecific competition in urban environments [34] has been linked to chronically elevated stress indices in wild bird populations, although further research is needed to evaluate associations among urbanization, immunity and stress in wildlife populations.

Furthermore, some heavy metal and pesticide pollutants become concentrated in the environment around developed areas and can be detrimental to vertebrate immune function [35,36]. A five-year field study of two amphibian

### Box 3. Exotic hosts, translocations, and commerce

As centers of transportation and trade, cities can function as ‘first points of entry’ for novel pathogens and, in some cases, might provide new opportunities for cross-species transmission. Anthropogenic introductions have expanded the geographical ranges of pathogens such as WNV in humans and wildlife [57], amphibian chytridiomycosis [58] and *Phytophthora ramorum*, the causative agent of sudden oak death [59]. Among wildlife populations, the 1999 introduction of WNV into New York City was particularly significant. The strain isolated from this outbreak belongs to the more virulent 1a clade of WNV and most closely resembles virus circulating in Israel, suggesting that human activity facilitated the movement of this agent into North America [60]. Once established around New York City, the virus spread rapidly across the continent and has caused >2800 cases of human disease and tens of thousands wild bird mortalities [57,61].

Cities also bring wildlife and their products in close proximity to humans and domesticated animals through live animal markets and the wildlife trade [62]. Examples of disease emergence events linked with the wildlife trade and live animal markets, including SARS-associated coronavirus [63], emphasize that as global commerce and travel increase, documenting pathogen introductions and the contacts among novel host species will become a more pressing concern in public health management. As such, periodic surveillance centered on cities with large human populations and commerce in wildlife products could represent a useful approach for detecting novel transmission events.

species, the marine toad *Bufo marinus* and whistling frog *Eleutherodactylus johnstonei*, found high levels of copper, cadmium and a byproduct of DDT decomposition in tissues samples, and further documented a decrease in B cell-mediated immunity and an increase in helminth infections [37]. More generally, however, the effects of environmental pollutants on host resistance to infectious diseases in wild animals are not well understood, and this area of research will become increasingly important as air, soil and water pollutants continue to accumulate around areas of human activity.

### Changes mediated by climate and seasonality

Research conducted in major metropolitan centers such as Tel Aviv, Israel and Phoenix, AZ, USA indicates that urban microclimates are typically warmer than outlying areas [38,39]. The urban heat island results from the increased retention of solar heat by impervious surfaces, radiant heat trapped by smog and a lack of shade vegetation. Several vector-borne diseases might respond to temperature changes associated with urbanization, particularly as these areas can also provide irrigated regions necessary for vector reproduction [2]. Specifically, more moderate winters and the dampened seasonality of urban centers could increase the survival, breeding success and activity of arthropod vectors that are essential for the transmission of many pathogens. In Stockholm, Sweden, for example, such conditions have lengthened the period of activity in the tick *Ixodes ricinus*, coinciding with an elevated incidence of tick-borne encephalitis in rodents and humans [40]. However, other factors could also have a role in this pattern, including human outdoor activity and higher rodent host densities; few studies have examined heat island effects on the ecology of vector-borne diseases.

Reduced seasonality in urban areas could also affect the persistence of parasite transmission stages in the

environment [41] and could favor the migration into cities of animals that serve as reservoir hosts for pathogens affecting humans [42]. Reduced seasonality can also increase population growth rates of some wildlife hosts by lengthening their breeding season, an effect that has been demonstrated for dark-eyed juncos *Junco hyemalis* inhabiting an urban site in California [43]. In such cases, a longer recruitment period could increase the probability of epidemics for pathogens that depend on susceptible hosts produced via new births [44]. By contrast, milder winter climates might reduce the individual-level impacts of infectious disease, especially if infected animals in harsher seasonal climates frequently die of secondary causes, such as exposure or starvation. Thus, similar to the effects of food provisioning, changes in the urban microclimate have multiple and, in some cases, opposite effects on pathogens affecting wildlife hosts.

### Consequences for wildlife conservation and public health

Although habitat loss and overexploitation are widely recognized as major causes of wildlife population declines, infectious diseases have become increasingly significant to animal conservation [6,7]. Beyond the direct impacts of urbanization on biodiversity, epidemiological processes altered by urban habitats can generate further challenges for wildlife populations. Of particular importance are multi-host pathogens that affect animals living at low population densities through interactions with other host species (Box 2; Table 1). For example, Cooper's hawks *Accipiter cooperii* nesting in urban areas experienced more than double the nest failure rate of hawks nesting in the suburbs. Most nestling mortalities were caused by trichomoniasis, a protozoan disease that can be transmitted through feeding on infected pigeons and doves [45]. Efforts targeted at lowering transmission among urban-adapted species (such as vaccination, treatment with anti-parasitic drugs or reducing supplemental food resources) could therefore limit pathogen transmission to less-abundant wildlife hosts.

Many wildlife species are absent from urban centers, and species that survive well in cities generally do not warrant conservation concern. However, several examples in Table 1 emphasize that processes occurring in cities and suburbs influence remnant wildlife populations within cities and can reach beyond the city limits. In at least one case, environmental contamination with *B. procyonis* by infected raccoons is directly linked to population declines in an endangered host species, the Allegheny woodrat *Neotoma magister* [46]. Decreasing risks to wildlife for this and other pathogens might require limiting the build up of environmental pathogen pollution, or reducing the population densities of reservoir hosts and tracking their movements from urban to more rural areas.

A better understanding of processes that impact wildlife–pathogen dynamics in urban landscapes should also point towards new approaches for limiting the risk of human exposure to zoonotic diseases. For example, information about population densities, contact rates and movements of skunks and raccoons have been used to predict urban sites at high risk for rabies outbreaks [47]. Similar

information for foxes harboring *E. multilocularis* can suggest strategies for concentrating urban disease control efforts, including baited vaccines or chemotherapy targeted at urban foxes (e.g. Ref. [48]) and limiting resource accumulation where animals might congregate near human dwellings.

Finally, urban planning represents a potential tool for altering habitats in ways that might reduce disease risks for both humans and wildlife hosts. Efforts to decrease impervious surface coverage, such as urban reforestation projects, could lower the potential for heat island effects on host reproduction, vector breeding and pathogen transmission. Because high wildlife biodiversity might reduce the net transmission of some multi-host vector-borne pathogens [19], increasing native vegetation and creating habitat corridors to facilitate reintroductions could also reduce pathogen prevalence and limit the risk of human exposure. We are not aware of any such strategies in use in urban settings to mitigate disease risk, but this represents an avenue for future collaboration among urban planners, veterinary biologists and wildlife ecologists.

### Challenges for future research

As human populations continue to migrate into cities and urban areas expand (Box 1), managing disease threats for humans and wildlife will depend on future research at the interface of two rapidly growing disciplines: urban ecosystem studies and infectious disease ecology. Surveillance programs targeted towards zoonotic agents would improve the ability of scientists to detect new agents entering cities and to document infections of pre-existing pathogens in novel host species. Also needed are studies that identify host–pathogen systems restricted to wildlife for which urbanization has significant impacts. Often overlooked for zoonotic diseases, these pathogens can have important consequences for animal species already threatened by other factors [13,16].

Perhaps most importantly, experimental and modeling approaches are needed to move beyond associational patterns and to tease apart the complex mechanisms by which urbanization affects hosts, pathogens and vectors. For example, to what degree do air pollution, noise and other environmental stressors influence wildlife susceptibility to infection, and are these effects counterbalanced by increased food resources or milder winter climates in cities? How does host immunity interact with physical landscape characteristics that alter host contact rates? In terms of vector-borne diseases, better information is needed regarding how biting arthropods are influenced by the heat island effect, distribution of breeding sites and shifts in host-species availability across urban–rural gradients.

To address these and other questions, manipulative field studies will become increasingly important for investigating wildlife–pathogen interactions in urban environments. Within the past decade, two cities in the USA were added to the Long-Term Ecological Research site network (<http://www.lternet.edu/>; funded by the National Science Foundation): Baltimore, MD and Phoenix, AZ [1]. Together with urban research sites in Europe, Asia and Latin America, these cities can serve as grounds for testing hypotheses

concerning the effects of host species diversity, food provisioning and environmental variables on the spread of wildlife diseases [20,22]. In turn, studies replicated across multiple locations can begin to explore how factors such as wildlife community composition, pollution and urban microclimates are affected by human population density, socioeconomic variables and land-use patterns.

### Concluding remarks

Our goal here was to identify key hypotheses concerning how wildlife–pathogen interactions will respond to urbanization and to highlight several examples that best illustrate these processes. Unlike other land-use changes that can influence wildlife disease emergence (e.g. forest fragmentation or agricultural intensification), the extreme changes that accompany urbanization probably cause declines or losses of most wildlife species and their associated parasites. However, the debate over positive versus negative effects of urbanization on the prevalence and impacts of wildlife diseases is likely to intensify as more research is published. Better understanding of the types of wildlife pathogens that do persist in urbanized areas, and mechanisms that cause increases in prevalence or impacts, can point to new strategies for limiting the risk of human and wildlife exposure in urban centers, and will improve our understanding of the ecological drivers behind spatial variation in pathogen occurrence.

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### References

- Grimm, N.B. *et al.* (2000) Integrated approaches to long-term studies of urban ecological systems. *Bioscience* 50, 571–584
- Shochat, E. *et al.* (2006) From patterns to emerging processes in mechanistic urban ecology. *Trends Ecol. Evol.* 21, 186–191
- McKinney, M.L. (2002) Urbanization, biodiversity, and conservation. *Bioscience* 52, 883–890
- Marzluff, J.M. (2001) Worldwide urbanization and its effects on birds. In *Avian Ecology and Conservation in an Urbanizing World* (Marzluff, J.M. *et al.*, eds), pp. 19–38. Kluwer Academic Publishers
- Faeth, S.H. *et al.* (2005) Trophic dynamics in urban communities. *Bioscience* 55, 399–407
- Daszak, P. *et al.* (2000) Emerging infectious diseases of wildlife: threats to biodiversity and human health. *Science* 287, 443–449
- Lafferty, K.D. and Gerber, L.R. (2002) Good medicine for conservation biology: the intersection of epidemiology and conservation theory. *Conserv. Biol.* 16, 593–604
- Patz, J.A. *et al.* (2004) Unhealthy landscapes: policy recommendations on land use change and infectious disease emergence. *Environ. Health Perspect.* 112, 1092–1098
- Prange, S. *et al.* (2003) Demographic factors contributing to high raccoon densities in urban landscapes. *J. Wildl. Manage.* 67, 324–333
- Coyner, D.F. *et al.* (2002) Epizootiology of *Eustrongylides ingotus* in Florida: distribution, density, and natural infections in intermediate hosts. *J. Wildl. Dis.* 38, 483–499
- Cleaveland, S. *et al.* (2001) Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. *Phil. Trans. R. Soc. B* 356, 991–999
- Woolhouse, M.E.J. *et al.* (2001) Population biology of multihost pathogens. *Science* 292, 1109–1112
- Woolhouse, M.E.J. and Gowtage-Sequeria, S. (2005) Host range and emerging and reemerging pathogens. *Emerg. Infect. Dis.* 11, 1842–1847
- Pauchard, A. *et al.* (2006) Multiple effects of urbanization on the biodiversity of developing countries: the case of a fast-growing metropolitan area (Concepcion, Chile). *Biol. Conserv.* 127, 272–281
- Olden, J.D. *et al.* (2006) Forecasting faunal and floral homogenization associated with human population geography in North America. *Biol. Conserv.* 127, 261–271
- de Castro, F. and Bolker, B. (2005) Mechanisms of disease-induced extinction. *Ecol. Lett.* 8, 117–126
- Schmidt, K.A. and Ostfeld, R.S. (2001) Biodiversity and the dilution effect in disease ecology. *Ecology* 82, 609–619
- LoGiudice, K. *et al.* (2003) The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. *Proc. Natl. Acad. Sci. U. S. A.* 100, 567–571
- Keesing, F. *et al.* (2006) Effects of species diversity on disease risk. *Ecol. Lett.* 9, 485–498
- Allan, B.F. *et al.* (2003) Effect of forest fragmentation on Lyme disease risk. *Conserv. Biol.* 17, 267–272
- Ezenwa, V.O. *et al.* (2006) Avian diversity and West Nile virus: testing associations between biodiversity and infectious disease risk. *Proc. R. Soc. Lond. B. Biol. Sci.* 273, 109–117
- Kilpatrick, A.M. *et al.* (2006) West Nile virus epidemics in North America are driven by shifts in mosquito feeding behavior. *PLoS Biol.* 4, e82
- Wright, A.N. and Gompper, M.E. (2005) Altered parasite assemblages in raccoons in response to manipulated resource availability. *Oecologia* 144, 148–156
- Deplazes, P. *et al.* (2004) Wilderness in the city: the urbanization of *Echinococcus multilocularis*. *Trends Parasitol.* 20, 77–84
- Gottstein, B. (2001) Is high prevalence of *Echinococcus multilocularis* in wild and domestic animals associated with disease incidence in humans? *Emerg. Infect. Dis.* 7, 408–412
- Fischer, C. *et al.* (2005) *Echinococcus multilocularis* infections of rural, residential and urban foxes (*Vulpes vulpes*) in the canton of Geneva, Switzerland. *Parasite* 12, 339–346
- Ezenwa, V.O. (2004) Interactions among host diet, nutritional status and gastrointestinal parasite infection in wild bovines. *Int. J. Parasitol.* 34, 535–542
- Riley, S.P.D. *et al.* (2004) Exposure to feline and canine pathogens in bobcats and grey foxes in urban and rural zones of a national park in California. *J. Wildl. Dis.* 40, 11–22
- Suzan, G. and Ceballos, G. (2005) The role of feral mammals on wildlife infectious disease prevalence in two nature reserves within Mexico city. *J. Zoo Wildl. Med.* 36, 479–484
- Dubey, J.P. *et al.* (2004) Molecular and biologic characteristics of *Toxoplasma gondii* isolates from wildlife in the United States. *J. Parasitol.* 90, 67–71
- Miller, M.A. *et al.* (2002) Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*). *Int. J. Parasitol.* 32, 997–1006
- McCabe, P. *et al.* (2000) Animal models of disease. *Physiol. Behav.* 68, 501–507
- Padgett, D. and Glaser, R. (2003) How stress influences the immune response. *Trends Immunol.* 24, 444–448
- Ruiz, G. *et al.* (2005) Hematological parameters and stress index in rufous-collared sparrows dwelling in urban environments. *Condor* 104, 162–166
- Bernier, J. *et al.* (1995) Immunotoxicity of heavy metals in relation to Great Lakes. *Environ. Health Perspect.* 103, 23–34
- Krzystyniak, K. *et al.* (1995) Approaches to the evaluation of chemical-induced immunotoxicity. *Environ. Health Perspect.* 103, 17–22
- Linzey, D.W. *et al.* (2003) Role of environmental pollutants on immune functions, parasitic infections and limb malformations in marine toads and whistling frogs from Bermuda. *Int. J. Environ. Health Res.* 13, 125–148
- Saaroni, H. *et al.* (2000) Spatial distribution and microscale characteristics of the urban heat island in Tel-Aviv. *Israel. Lands. Urban Plan.* 48, 1–18
- Baker, L. *et al.* (2002) Urbanization and warming of Phoenix (Arizona, U.S.A.): impacts, feedbacks and mitigation. *Urban Ecol.* 6, 183–203



- 40 Lindgren, E. and Gustafson, R. (2001) Tick-borne encephalitis in Sweden and climate change. *Lancet* 358, 16–18
- 41 Louis, V.R. *et al.* (2005) Temperature-driven *Campylobacter* seasonality in England and Wales. *Appl. Environ. Microbiol.* 71, 85–92
- 42 Parris, K.M. and Hazell, D.L. (2005) Biotic effects of climate change in urban environments: the case of the grey-headed flying-fox (*Pteropus poliocephalus*) in Melbourne, Australia. *Biol. Conserv.* 124, 267–276
- 43 Yeh, P.J. and Price, T.D. (2004) Adaptive phenotypic plasticity and the successful colonization of a novel environment. *Am. Nat.* 164, 531–542
- 44 Altizer, S. *et al.* (2006) Seasonality and the dynamics of infectious diseases. *Ecol. Lett.* 9, 467–484
- 45 Boal, C.W. and Mannan, R.W. (1999) Comparative breeding ecology of Cooper's hawks in urban and exurban areas of southeastern Arizona. *J. Wildl. Manage.* 63, 77–84
- 46 LoGuidice, K. (2003) Tropically transmitted parasites and the conservation of small populations: raccoon roundworms and the imperiled Allegheny woodrat. *Conserv. Biol.* 17, 258–266
- 47 Broadfoot, J.D. *et al.* (2001) Raccoon and skunk population models for urban disease control planning in Ontario, Canada. *Ecol. Appl.* 11, 295–303
- 48 Hegglin, D. *et al.* (2003) Anthelmintic baiting of foxes against urban contaminations with *Echinococcus multilocularis*. *Emerg. Infect. Dis.* 9, 1266–1272
- 49 United Nations Department of Economic and Social Affairs (2003) *World Population Prospects*, United Nations
- 50 Theobald, D.M. (2004) Placing exurban land-use change in a human modification framework. *Front. Ecol. Environ.* 2, 139–144
- 51 Hope, D. *et al.* (2003) Socioeconomics drive urban plant diversity. *Proc. Natl. Acad. Sci. U. S. A.* 100, 8788–8792
- 52 Ostfeld, R.S. *et al.* (2005) Spatial epidemiology: an emerging (or re-emerging) discipline. *Trends Ecol. Evol.* 20, 328–336
- 53 Wauters, L.A. and Grunell, J. (1999) The mechanism of replacement of red by grey squirrels: a test of the interference competition hypothesis. *Ethology* 105, 1053–1071
- 54 Tompkins, D. *et al.* (2002) Parapoxvirus causes a deleterious disease of red squirrels associated with UK population declines. *Proc. R. Soc. B* 269, 529–533
- 55 Tompkins, D.M. *et al.* (2003) Ecological replacement of native red squirrels by invasive greys driven by disease. *Ecol. Lett.* 6, 189–196
- 56 Rushton, S.P. *et al.* (2000) Modeling the spatial dynamics of parapoxvirus disease in red and grey squirrels: a possible cause of the decline in the red squirrel in the United Kingdom? *J. Appl. Ecol.* 37, 1–18
- 57 Marra, P.P. *et al.* (2004) West Nile virus and wildlife. *Bioscience* 54, 393–402
- 58 Rachowicz, L.J. *et al.* (2005) The novel and endemic pathogen hypotheses: competing explanations for the origin of emerging infectious diseases of wildlife. *Conserv. Biol.* 19, 1441–1448
- 59 Rizzo, D.M. and Garbelotto, M. (2003) Sudden oak death: endangering California and Oregon forest ecosystems. *Front. Ecol. Environ.* 1, 197–204
- 60 Lanciotti, R.S. *et al.* (2002) Complete genome sequences and phylogenetic analysis of West Nile virus strains isolated from the United States, Europe, and the Middle East. *Virology* 298, 96–105
- 61 Peterson, L.R. and Hayes, E.B. (2004) Westward ho? The spread of West Nile virus. *N. Engl. J. Med.* 351, 2257–2259
- 62 Karesh, W.B. *et al.* (2005) Wildlife trade and global disease emergence. *Emerg. Infect. Dis.* 11, 1000–1001
- 63 Xu, R.H. *et al.* (2004) Epidemiologic clues to SARS origin in China. *Emerg. Infect. Dis.* 10, 1030–1037
- 64 Gibbs, S.E.J. *et al.* (2006) Factors affecting the geographic distribution of West Nile virus in Georgia, USA: 2002–2004. *Vector-borne Zool. Dis.* 6, 73–82
- 65 Dhondt, A.A. *et al.* (2005) Dynamics of a novel pathogen in an avian host: *Mycoplasmal conjunctivitis* in house finches. *Acta Trop.* 94, 77–93
- 66 Farnsworth, M.L. *et al.* (2005) Human land use influences Chronic Wasting Disease prevalence in Mule Deer. *Ecol. Appl.* 15, 119–126
- 67 Isaksson, C. *et al.* (2005) Plasma glutathione and carotenoid coloration as potential biomarkers of environmental stress in Great Tits. *EcoHealth* 2, 138–146
- 68 Partecke, J. *et al.* (2006) Stress and the city: urbanization and its effects on the stress physiology in European blackbirds. *Ecology* 87, 1945–1952

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