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Wildlife disease prevalence in human-modified landscapes

Grant Brearley[1,*], Jonathan Rhodes[1], Adrian Bradley[2], Greg Baxter[1], Leonie Seabrook[1], Daniel Lunney[3,4], Yan Liu[1] and Clive McAlpine[1]

[1] *Landscape Ecology and Conservation Group, Centre for Spatial Environmental Research, School of Geography, Planning & Environmental Management, The University of Queensland, Brisbane, 4072, Australia*
[2] *School of Biomedical Sciences, The University of Queensland, Brisbane, 4072, Australia*
[3] *Office of Environment and Heritage NSW, PO Box 1967, Hurstville, New South Wales 2220, Australia*
[4] *School of Biological Sciences and Biotechnology, Murdoch University Murdoch, Western Australia, 6150, Australia*

ABSTRACT

Human-induced landscape change associated with habitat loss and fragmentation places wildlife populations at risk. One issue in these landscapes is a change in the prevalence of disease which may result in increased mortality and reduced fecundity. Our understanding of the influence of habitat loss and fragmentation on the prevalence of wildlife diseases is still in its infancy. What is evident is that changes in disease prevalence as a result of human-induced landscape modification are highly variable. The importance of infectious diseases for the conservation of wildlife will increase as the amount and quality of suitable habitat decreases due to human land-use pressures. We review the experimental and observational literature of the influence of human-induced landscape change on wildlife disease prevalence, and discuss disease transmission types and host responses as mechanisms that are likely to determine the extent of change in disease prevalence. It is likely that transmission dynamics will be the key process in determining a pathogen's impact on a host population, while the host response may ultimately determine the extent of disease prevalence. Finally, we conceptualize mechanisms and identify future research directions to increase our understanding of the relationship between human-modified landscapes and wildlife disease prevalence. This review highlights that there are rarely consistent relationships between wildlife diseases and human-modified landscapes. In addition, variation is evident between transmission types and landscape types, with the greatest positive influence on disease prevalence being in urban landscapes and directly transmitted disease systems. While we have a limited understanding of the potential influence of habitat loss and fragmentation on wildlife disease, there are a number of important areas to address in future research, particularly to account for the variability in increased and decreased disease prevalence. Previous studies have been based on a one-dimensional comparison between unmodified and modified sites. What is lacking are spatially and temporally explicit quantitative approaches which are required to enable an understanding of the range of key causal mechanisms and the reasons for variability. This is particularly important for replicated studies across different host-pathogen systems. Furthermore, there are few studies that have attempted to separate the independent effects of habitat loss and fragmentation on wildlife disease, which are the major determinants of wildlife population dynamics in human-modified landscapes. There is an urgent need to understand better the potential causal links between the processes of human-induced landscape change and the associated influences of habitat fragmentation, matrix hostility and loss of connectivity on an animal's physiological stress, immune response and disease susceptibility. This review identified no study that had assessed the influence of human-induced landscape change on the prevalence of a wildlife sexually transmitted disease. A better understanding of the various mechanisms linking human-induced landscape change and the prevalence of wildlife disease will lead to more successful conservation management outcomes.

Key words: disease prevalence, disease transmission, fragmentation, habitat loss, host, human-modified landscapes, physiological stress.

* Address for correspondence (Tel: +61-7-3365-3535; Fax: +61-7-3365-6899; E-mail: g.brearley@uq.edu.au).

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I. INTRODUCTION

Human land use influences more than 75% of Earth's ice-free land surface (Ellis & Ramankutty, 2008), and few ecosystems remain undisturbed by some form of anthropogenic modification (Vitousek *et al*., 1997; Sanderson *et al*., 2002; Foley *et al*., 2005). This has led to a worldwide decline in biodiversity, with 37.8% of the world's wildlife identified as threatened in the 2008 IUCN Red List (Vié, Hilton-Taylor & Stuart, 2009).Human-modified landscapes put wildlife populations at risk of decline from higher mortality and lower fecundity rates due to direct and indirect interactions with human activities (Ferreras *et al*., 2001; Naves *et al*., 2003; Rhodes *et al*., 2011). This is particularly evident in highly modified landscapes, such as urban areas, where the high-contrast landscape significantly alters the biotic and physical attributes of the environment (Lugo, 2002). In addition, these areas also present multiple novel threats to the survival of wildlife, such as increased transport networks, traffic volumes (e.g. Forman *et al*., 2003; van der Ree *et al*., 2011), exotic predators (e.g. Butler, du Toit & Bingham, 2004; Manor & Saltz, 2004; Marks & Duncan, 2009) and increased susceptibility to persisting and/or novel diseases (e.g. Cleaveland *et al*., 2000; Millan *et al*., 2009; Lehrer *et al*., 2010). Human activities occur disproportionately more in fertile lowland regions, and species in these areas are often not well represented in protected areas. Consequently, we need to maximize survival and long-term persistence of these species within modified landscapes. Identifying the conservation needs of species facing multiple threats requires an understanding of the relative effects of individual, and often interacting, human activities (e.g. Lawler *et al*., 2002; Rhodes *et al*., 2011). It is important, therefore, to understand the causal mechanisms linking human-induced landscape change, wildlife disease prevalence, elevated wildlife mortality and reduced fecundity.

Human-induced landscape change is increasingly recognized as impacting the health and fitness of animals in habitat fragments (Cottontail, Wellinghausen & Kalko, 2009). Habitat loss and fragmentation reduce the amount and connectivity of suitable habitats (Fahrig, 2003). This affects population viability, lowers genetic diversity and causes inbreeding depression, potentially making individuals more susceptible to existing or novel diseases and stochastic environmental events (Smith, Acevedo-Whitehouse & Pedersen, 2009*a*; Clark *et al*., 2011). The contribution of infectious disease to the decline of susceptible wildlife populations may increase as the quantity and quality of suitable habitat declines (Smith *et al*., 2009*a*). The rising threat of disease epidemics in wildlife means that monitoring and understanding their prevalence (i.e. the number of total cases of a disease in a given population at a specific time) is an increasing conservation concern (Scott, 1988). Ultimately, there are a number of mechanisms to consider when assessing the impacts of human-induced landscape change on wildlife disease prevalence. Smith *et al*. (2009*a*) reviewed how human-induced drivers of disturbance can lead to environmental changes that may facilitate disease-mediated extinctions. These include habitat loss and alteration, climate change, overexploitation, invasive species, and environmental pollution. While each factor is important, wildlife disease prevalence will be guided by mechanisms linked to both disease transmission and host response.

Disease in wild populations requires specific consideration when trying to identify links between human-modified landscapes and changes in mortality rates. Infectious diseases play a major role in the dynamics of many natural populations and communities (Holmes, 1996; Lafferty & Holt, 2003) and may pose a threat to population viability (McCallum & Dobson, 1995). A previous review identified disease as either causing population declines or extinction in a range of wildlife, including amphibians, mammals

and birds (de Castro & Bolker, 2005). Disease-related extinction risks due to parasites may also be substantially underestimated, demonstrating the need to assess better the presence and impacts of infectious disease in wild animal populations (Leendertz *et al*., 2006; Pedersen *et al*., 2007). Understanding the influence of human-modified landscapes on disease prevalence is important in those landscapes that have already experienced increased mortality rates from multiple threatening processes, and where populations have been reduced below a critical size threshold for survival (Plowright *et al*., 2008).

Landscape change can alter ecological and evolutionary processes, including those between host and parasite (Walsh, Molyneux & Birley, 1993; Chasar *et al*., 2009). Critically, human-induced landscape change and activities have been considered to be drivers of emergent infectious disease (EIDs) of wildlife (Plowright *et al*., 2008). Specifically, two primary factors resulting in EIDs, and often acting together, have been characterized by Daszak, Cunningham & Hyatt (2001) as follows: (*i*) 'spill-over' of pathogens from domestic animals, and (*ii*) anthropogenic movement of new pathogens into new locations, termed 'pathogen pollution'. Some examples of spill-over between domestic and wild animals are canine distemper virus (CDV) in African carnivores from domestic dogs (Cleaveland *et al*., 2000) and *Toxoplasma gondii* in woodchucks (*Marmotamonax*) from domestic and feral cats (Lehrer *et al*., 2010). There is a serious threat of multiple diseases (e.g. parvovirus, *Ehrlichia* spp., *Mycobacterium bovis* etc.) in the endangered Iberian lynx (*Lynx pardinus*) from reservoirs such as unvaccinated and unneutered domestic dogs and cats (Millan *et al*., 2009). Pathogen pollution has been linked to the introduction or spread of numerous wildlife EIDs, including parapoxvirus in the red squirrel (*Sciurus vulgaris*) following the introduction of the grey squirrel (*Sciurus carolinensis*) in the UK (e.g. Tompkins *et al*., 2002; Thomas *et al*., 2003; Tompkins, White & Boots, 2003), West Nile virus disease in US avian populations (e.g. Ezenwa *et al*., 2006; Gibbs *et al*., 2006; Bradley, Gibbs & Altizer, 2008), and chytridiomycosis in amphibians in many parts of the world (e.g. Rachowicz *et al*., 2005, 2006; Pauza, Driessen & Skerratt, 2010).

A potential link between human modification of landscapes and disease in wildlife that has received little attention is the effect of increased physiological stress on immune competence. By contrast, major pathways between stress and immune function in humans are well established. This process is outlined in detail by Hussain (2010). Following the perception of a stressor by the brain, the hypothalamus releases corticotropin-releasing hormone (CRH). CRH stimulates the pituitary gland to secrete adrenocorticotropic hormone (ACTH), which in turn stimulates the adrenal cortex to secrete glucocorticoids (GCs). GC stress hormones (e.g. cortisol or corticosterone in mammals) have been found to inhibit the production and activity of white blood cells. Cortisol also suppresses the capacity of white blood cells to produce chemical messengers, so that different varieties of immune cells become unable to communicate with each other. Cortisol can also signal too many immune cells to stop working (Talbott & Kreamer, 2007).

Ultimately, the influence of stress on immunity is considered the primary pathway through which stress influences infectious disease susceptibility in wildlife (Cohen & Williamson, 1991). A study on cotton rats (*Sigmodon hispidus*) found that individuals treated daily to induce stress had a significantly lower ability to resist a virus threat (McLean, 1982). Recent studies have provided further evidence that immunity is diminished during stress responses to enable redirection of resources towards activities that are more immediately valuable to survival (Sapolsky, Romero & Munck, 2000; Martin, 2009), although there may be a number of outcomes for the animal. In a review of stress and immunity, Martin (2009) found varying results relating to the duration of stressors. One of the most important conclusions of Martin (2009) was that sustained stressors (e.g. habitat modification) result in reductions of most immune processes, while short-term stressors (e.g. predation attempts) enhance most immune responses. Although still in its infancy, studies on birds (Fokidis, Orchinik & Deviche, 2009), reptiles (French, Fokidis & Moore, 2008) and more recently mammals (Johnstone, Lill & Reina, 2011; Johnstone, Lill & Reina, 2012; Brearley *et al*., 2012) have found varying elevated physiological stress responses to human-modified landscapes. These findings underline the importance of testing the potential three-way link between human-modified landscapes, physiological stress and disease prevalence in wildlife.

Mortality and disease-related infertility are crucial factors affecting the viability and dynamics of wildlife species (O'Keefe & Antovonics, 2002; Packer *et al*., 2003; Bairagi, Roy & Chattopadhyay, 2007; Su *et al*., 2009). In regions where habitat loss and other anthropogenic modifications are already reducing wildlife numbers, infectious disease may further affect population viability, leading to local extirpation (Plowright *et al*., 2008; Smith *et al*., 2009*a*). Although the interaction effects between human-induced landscape change and disease prevalence in wildlife populations are highly variable, there are areas which require further attention to understand this process better. Herein, we summarize the state of current research on the relationship between disease prevalence and human-induced landscape modification and specifically consider the influence of habitat loss and fragmentation on shifting disease prevalence. We synthesize these findings in relation to disease transmission types and host responses, two of the main mechanisms that determine disease prevalence. Finally, we conceptualize future research directions which will increase our knowledge of the relationship between human-modified landscapes and wildlife disease prevalence.

II. LITERATURE REVIEW

This review addresses two primary questions: (*i*) is there a consistent relationship between human-modified landscapes

and wildlife disease prevalence, and (*ii*) where are the significant knowledge gaps that require future research?

We considered approximately 70 research papers, book chapters and conference proceedings that assessed or discussed the influence of human-modified landscapes on wildlife disease prevalence. Literature was sourced from the ISI *Web of Science* database (http://www.isiwebofknowledge.com) using a combination of key word searches, including: disease, wildlife, fragmentation, habitat loss, urban, disease transmission, disease prevalence, parasite, pathogen, sexually transmitted disease, anthropogenic, infection, host, land-use, and environmental stressors. Additional literature was sourced from the citation lists of these papers.

On examination, more than 40 papers mentioned or briefly discussed the potential influence of human-induced landscape change on disease, however, their lack of field-based (e.g. experimentally designed studies in human-modified landscapes) or simulated (e.g. simulated environmental modification and disease change) analysis of disease change meant that they were excluded from our study. Further, as this review was focused on wildlife diseases, studies that had a focus on human disease, and with no impact on wildlife, were excluded from the final list; reference to wildlife in such studies was predominately as reservoirs and/or vectors of disease, e.g. hantavirus (Suzan *et al*., 2008, 2009; Armien *et al*., 2009; Dearing & Dizney, 2010). Similarly, although a number of studies on disturbance-related disease in marine environments were reviewed, our focus was on terrestrial systems, and due to the contrasting processes involved in the two ecosystems, marine studies were not incorporated into the final list.

Specific information was recorded from the reviewed literature, including focal species, geographic location, disease/s type, disturbance type, increase, decrease or varied change in disease prevalence and major study findings. For prevalence, varied change relates to a single study that has identified both an increase and decrease in disease prevalence between different hosts, diseases and/or landscape types. This information allowed an assessment of the directional influence of different human-modified landscapes on the prevalence of various disease types. Nineteen papers were selected for final analysis (Table 1).

III. SYNTHESIS OF CURRENT KNOWLEDGE

In an emerging field, it is important to recognize any potential trends, identify gaps in knowledge and provide future research directions. We recognize that inferring trends from a sample size of 19 studies has its limitations; however the results clearly indicate that the issue of wildlife disease in human-modified landscapes is complex and highly variable. Owing to the majority of past studies having been based on a one-dimensional comparison between unmodified and modified sites, identifying this variability, and outlining key areas for research, is an important function of this review.

(1) Human-modified landscapes and disease prevalence

Half (53%) of the papers reviewed indicated an increase in disease prevalence due to human-induced landscape change (Table 2). Of the remainder, 21% identified a decrease in disease prevalence, while 26% found that disease prevalence varied.

During the past decade, there has been an increase in the number of studies that have assessed the potential influence of habitat loss and fragmentation on disease prevalence (Chapman, Gillespie & Goldberg, 2005; Chasar *et al*., 2009; Evans *et al*., 2009). Studies focused on agricultural, plantation or cattle-grazing areas have shown that their influence on disease prevalence may vary considerably (Table 2). On the other hand, studies in urban landscapes (Fischer *et al*., 2005; Geue & Partecke, 2008; Friggens & Beier, 2010; Lehrer *et al*., 2010) tend to show an increase in disease prevalence (Table 2). Lehrer *et al*. (2010) found that the prevalence of the bacterium *Toxoplasma gondii* in the woodchuck was positively related to levels of urban land cover in excess of 70%. This was attributed to an overlap with higher densities of the definitive host in urban areas – domestic and feral cats. Consistent positive relationships have also been found between multiple host bird species and the presence of West Nile virus antibodies in the urban/suburban environment of Georgia, USA (Gibbs *et al*., 2006), probably due to increases in abundance of vector species in human-modified habitats. By contrast, studies of blood parasite infection of blackbirds (*Turdus merula*) in Munich, southern Germany, found fewer infected individuals in urban areas. This was considered to be due to a reduction in the frequency of appropriate vectors (Geue & Partecke, 2008). Furthermore, the prevalence of tapeworm (*Echinococcus multitocularis*) in the red fox (*Vulpes vulpes*) was found to decrease from rural and residential areas to dense urban areas in Geneva, Switzerland, due to lower contamination rates (Fischer *et al*., 2005). To gain a better understanding of the influence of human-modified landscapes on disease, it is essential to explore the potential mechanisms driving variation in disease prevalence in a range of different landscapes. This will be particularly important in urban landscapes in the future, as these areas are arguably the most damaging, persistent and rapidly expanding form of human landscape modification impacting biodiversity worldwide (Vitousek *et al*., 1997; McKinney, 2002; Miller & Hobbs, 2002; Foley *et al*., 2005).

Variability in disease outcomes typically reflects factors such as host species, transmission type, and disease. Variation in disease prevalence between host species were shown in studies on the red colobus (*Procolobusrufomitratus*) and black-and-white colobus (*Colobus guereza*) in Kibale National Park and surrounding edge habitats and agricultural fragments in Uganda (Chapman *et al*., 2006; Gillespie & Chapman, 2008). Human-modified habitats increased the likelihood of infection by gut parasites, but the prevalence and magnitude

Table 1. Summary of species, locality, disease/s, human-induced landscape disturbance, prevalence, major findings and study reference for each of the 19 studies chosen as relevant

Species	Locality	Disease/s	Disturbance	Prevalence	Major findings	References
Vector transmission						
Common fruit bat (*Artibeus jamaicensis*)	Panama, USA	Haemoparasite infections [trypanosomes and *Litomosoides* spp. (Nematoda)]	Human-made islands	Varied	Trypanosome (multi-host) prevalence increased in fragmented habitats due to a loss of bat species richness and changes to vegetation cover favouring transmission. *Litomosoides* spp. (Nematoda) prevalence showed no significant difference between habitats suggesting a greater host specificity. Negative relationship between trypanosome and number of bat species supported the dilution effect.	Cottontail *et al.* (2009)
Yellow-whiskered greenbul (*Andropadus latirostris*) and olive sunbird (*Cyanomitra olivacea*)	Cameroon, Africa	Avian malaria parasites (*Plasmodium* spp.) and other related haemosporidians (*Haemoproteus* and *Leucocytozoon* spp.)	Agriculture, logging	Varied	*Haemoproteus* spp. and *Leucocytozoon* spp. prevalence was greater in undisturbed sites. One morphospecies of *Plasmodium* sp. in *C. olivacea* showed increased prevalence in disturbed sites. Habitat change can affect host-parasite systems in various ways due to different modes of transmission (e.g. mosquito species responses to fragmentation).	Chasar *et al.* (2009)
Many small mammal species	Brazil, South America	*Trypanosoma cruzi*	Agricultural, cattle-raising	Increase	Higher prevalence of *T. cruzi* in fragments compared to a National Park reserve. Prevalence correlates with a loss of local small mammal diversity (decreasing the dilution effect = fewer potential hosts) and increase of vector population as a consequence of overall biodiversity maintenance and feeding opportunity (Schmidt & Ostfeld, 2001).	Vaz *et al.* (2007)
Blackbirds (*Turdus merula*)	Tunisia, Spain, Netherlands, UK, Germany, Poland, Czech Republic, Poland, Latvia, Estonia, France, (Europe)	Avian malaria and ticks	Small-scale and intensive agriculture	Decrease	Consistent reductions in tick prevalence and intensity in urban areas. Reductions in prevalence of avian malaria in most urban areas.	Evans *et al.* (2009)
Blackbirds (*Turdus merula*)	Germany, Europe	Blood parasites	Urban	Decrease	Infected individuals lower in urban areas. Blood-parasite exposure lower in urban areas.	Geue & Partecke (2008)
Wild songbirds	Georgia, USA	West Nile virus (WNV)	Residential, commercial, recreational	Increase	Antibody prevalence increased from rural to urban sites. Urbanization can influence patterns of this vector-borne viral disease. Prevalence likely to be related to increased densities of reservoirs in fragmented landscapes (e.g. white-footed mouse).	Bradley *et al.* (2008)

Table 1. (Cont.)

Species	Locality	Disease/s	Disturbance	Prevalence	Major findings	References
Vector (*Culex* spp. mosquitoes) Host (multiple bird species)	Louisiana, USA	West Nile virus (WNV)	Various land-use/cover (e.g. developed land, forested upland, shrubland, wetland)	Varied	Infection prevalence decreased with increasing wetland land cover; however showed no significant difference when compared with developed land cover. Land-cover effects on disease distribution can be mediated by complex community-level processes that go beyond simple changes in single host or vector abundances.	Ezenwa *et al.* (2007)
Host (multiple bird species)	Georgia, USA	West Nile virus (WNV)	Urban, suburban	Increase	A weak positive correlation between antibodies and urban/suburban environment consistent throughout the study. Risks of endemicity appear to increase within urban/suburban areas compared to mountainous regions.	Gibbs *et al.* (2006)
Direct transmission						
Red colobus (*Procolobus ru fomitratus*), black-and-white colobus (*Colobus guereza*)	Uganda, Africa	Nematodes (e.g. *Strongyloides fulleborni*, *Strongyloides stercoralis*, *Oesophagostomum* sp., *Trichuris* sp., *Ascaris* sp., and *Colobenterobius* sp.). Cestode (*Bertiella* sp.). Protozoans (*Entamoeba coli*, *Entamoebahistolytica/dispar*, and *Giardia* sp.)	Small-scale agriculture	Varied	Prevalence and magnitude of infection was greater in red colobus living in fragmented forest. This pattern correlated with increased infective-stage colobus in fragments – demonstrating a greater infection risk. Infection was similar in black-and-white colobus living in fragmented and un-fragmented forests. Forest fragmentation can alter host-parasite dynamics and these can correlate with changes in host population size in forest fragments.	Gillespie & Chapman (2008)
Red colobus and black-and-white colobus	Uganda, Africa	Nematodes (*Trichuris* sp. (Superfamily Trichuroidea), *Oesophagostomum* sp. (Superfamily Strongyloidea), *Strongyloides fuelleborni* (Superfamily Rhabditoidea), and *Enterobius* (*Colobenterobius*) sp. (Superfamily Oxyuroidea) Protozoans (*Entamoebacoli* and *E. histolytica/dispar*)	Small-scale agriculture, plantations, edge habitat	Varied	Proportion of individuals in both species with multiple infections was greater in edge groups Prevalence of specific parasites also varied, particularly in the red colobus Less severe infections in the black-and white colobus suggests they raid agricultural crops and thus have established a better immune response Animals on edge may be nutritionally stressed – leading to elevated infection levels	Chapman *et al.* (2006)
Red colobus, red-tailed guenons (*Cercopithecusa scanius*) and black-and-white colobus	Uganda, Africa	*Cryptosporidium* sp. and *Giardia* sp.	Small-scale agriculture, plantations	Increase	Greater risk of infection of *Cryptosporidium a*nd *Giardia* spp. in disturbed habitats. Habitat disturbance may play a role in transmission or persistence of *Cryptosporidium* and *Giardia* spp.	Salzer *et al.* (2007)

Table 1. (Cont.)

Species	Locality	Disease/s	Disturbance	Prevalence	Major findings	References
Red colobus	Uganda, Africa	Gastrointestinal parasites (strongyle and rhabditoid nematodes)	Agriculture	Increase	Habitat degradation and human presence influence the prevalence of parasitic nematodes. Disease infection risk was higher in fragments with higher human impact (stump density).	Gillespie & Chapman (2006)
Red colobus	Uganda, Africa	Parasite (general) – model based	Simulated forest change	Increase	Resource clumping (rich sites) and landscape heterogeneity similar to patchy habitats contribute to disease spread. These characteristics are similar to nearby logged sections of forest.	Bonnell *et al.* (2010)
Woodchuck (*Marmota monax*)	Illinois, USA	*Toxoplasma gondii*	Urban, peri-urban	Increase	Prevalence was positively related to urbanization. All positive samples (containing antibodies) were from areas comprising urban land cover exceeding 70%. Increased prevalence most likely due to overlap with higher densities of the definitive host (in urban areas), domestic and feral cats.	Lehrer *et al.* (2010)
Balinese long-tailed macaques (*Macaca fascicularis*)	Bali, Indonesia	Numerous gastrointestinal parasites	Agriculture, anthropogenic change	Decrease	Increasing anthropogenic activity can lead to an overall lessening of the intensity and diversity of gut parasites. High interaction rates with humans provide a benefit to macaques *via* provisioning – ultimately reducing the need for foraging and the likelihood of exposure.	Lane *et al.* (2011)
Mule deer (*Odocoileus hemionus*)	Colorado, USA	Chronic wasting disease (CWD)	Residential	Increase	CWD prevalence was higher in disturbed sites and among males. Prevalence of CWD showed strong spatial heterogeneity among three study sites.	Farnsworth *et al.* (2005)
Frugivorous bats: *Artibeus jamaicensis*, *Artibeus lituratus*, *Sturnira lilum*, *Sturnira ludovici*	Villahermosa, Tabasco, Mexico	Alopecic syndrome	Urban	Increase	Higher prevalence in urban areas than in peri-urban areas. Possibly related to nutritional or endocrinal deficiencies. Anthropogenic activities may interfere with nutritional processes – therefore linking with the abovementioned.	Bello-Gutierrez *et al.* (2010)
Three frog species: *Litoria burowsae*, *Bryobatrachus nimbus*, *Criniatasmaniensis*	Tasmania, Australia	Chytridiomycosis, caused by pathogen *Batrachochytriumdendr obatidas* (Bd)	Anthropogenic	Increase	High incidence of Bd associated with human disturbance and activity (particularly gravel roads). Absence of Bd in remote, relatively undisturbed regions.	Pauza *et al.* (2010)
Red fox (*Vulpes vulpes*)	Geneva, Switzerland	Tapeworm (*Echinococcus multitocularis*)	Urban, residential	Decrease	Prevalence decreased from rural and residential areas to dense urban areas. Lower contamination in urban environments.	Fischer *et al.* (2005)
Sexually transmitted diseases (STDs) – No studies found						

For prevalence, 'varied' means that a single study identified both an increase and decrease in disease prevalence between different hosts, diseases and/or landscape types.

Table 2. Summary of the direction of disease prevalence change in response to different human-modified landscape types

	Disease prevalence change		
Landscape type/s	Increase	Decrease	Varied
Urban/suburban/peri-urban	6 (60%)	2 (50%)	1 (20%)
Agriculture/logging/cattle grazing	3 (30%)	2 (50%)	3 (60%)
Simulated landscape change	1 (10%)	—	—
Fragmented islands	—	—	1 (20%)
Total	10	4	5

The percentage (%) of the total studies showing the associated change in prevalence is shown in parentheses. 'Varied' means that a single study identified both an increase and decrease in disease prevalence between different hosts, diseases and/or landscape types.

of infection was greater in the red colobus in fragmented forest habitats, while the black-and-white colobus showed no change, or less severe infections, for a range of parasites (Chapman *et al*., 2006; Gillespie & Chapman, 2008). It was suggested that these changes may be due to the black-and-white colobus raiding nearby agricultural crops and, as a result, minimizing nutritional stress that may impede immune responses (Chapman *et al*., 2006). Such examples highlight the importance of looking beyond the influence of generalized human modification on disease to include detailed assessments of the impact of multiple factors at varying spatial and temporal scales. In addition, the implications of different transmission types and/or host responses must be considered. Su *et al*. (2009) noted that, although environmental heterogeneity has a profound effect on population dynamics and biological invasions, the spatial dynamics of disease invasions in host-parasite systemshave received little attention.

(2) Landscape change and disease transmission

Transmission type is a key process in host-pathogen interactions (McCallum, Barlow & Hone, 2001). Therefore, understanding transmission dynamics is a major requirement for predicting a pathogen's impact on a host population (Greer, Briggs & Collins, 2008).The following conceptual model (Fig. 1) outlines key processes of the importance of transmission type in linking human-modified landscapes with changes in the prevalence of wildlife disease. The potential causal mechanisms contained within this conceptual model have been developed using the synthesis provided within this review.

Simple disease models assume that the rate of transmission will increase linearly with population density; i.e. density-dependent transmission (McCallum *et al*., 2001; Begon *et al*., 2002; Greer *et al*., 2008). An alternative model assumes that contact rates are independent of host population density (Ryder *et al*., 2007). In this model, frequency-dependent transmission is driven by the proportion of infected hosts within a population, rather than the density of the infected host (Begon *et al*., 2002; O'Keefe, 2005). Frequency-dependent transmission is typically applied to sexually transmitted disease (STDs) (Ryder *et al*., 2007), but can also occur due to territorial or social behaviour (Altizer *et al*., 2003). The problem with using such distinct groupings in natural systems is that the mechanisms involved in transmission are unlikely to be all of one type or another. It is more likely in natural systems that some combination of the two transmission processes will be at play. This has been recognized by a number of studies that have shown that both density-dependent and frequency-dependent transmission represent two extremes (Fenton *et al*., 2002), that the pattern of transmission is likely to shift along a continuum between the two (Fenton *et al*., 2002; Greer *et al*., 2008) and, in some cases, exhibit components of both (Ryder *et al*., 2007; Smith *et al*., 2009*b*).

Given the importance of transmission of wildlife disease, it is necessary to identify the mode of transmission when assessing the influence of habitat-modified landscapes on disease prevalence. For the purpose of this review, wildlife disease has been categorized based on three transmission modes: vector, direct and sexual (Fig. 1). This was chosen over density- and frequency-dependent transmission because these categories are not mutually exclusive (Ryder *et al*., 2007; Smith *et al*., 2009*b*).

(a) *Vector-transmitted disease*

Based on the current literature, vector-transmitted disease prevalence can be highly variable in human-modified landscapes, with examples showing increases, decreases and varied results (Fig. 1). The complexity of vector-transmitted diseases is well illustrated by the work of Friggens & Beier (2010) on fleas. They demonstrate that disease spread is a culmination of: (*i*) individual effects on host-parasite interactions, (*ii*) the habitat dependency of both the host and flea species, and (*iii*) the host specificity of fleas. This study illustrated the high variability in disease prevalence among and within vector types due to these three key factors. Their review on flea-borne diseases in 70 communities of small mammals from different geographic regions (including Africa, Asia, Australia, Europe, North America and South America) found that most measures of flea infestations increased with disturbance. Similarly, in the USA, decreasing habitat patch size has a positive influence on tick density and infection prevalence of Lyme's bacterium (*Borrelia burgdorferi*) (Allan, Keesing & Ostfeld, 2003; Brownstein *et al*., 2005). In Brazil, an increased prevalence of *Trypanosoma cruzi* in small mammals living in fragments, compared to in a National Park, correlated with a loss of local small mammal diversity and subsequent reduction in potential hosts (Vaz, D'Andrea & Jansen, 2007). Specifically, a decrease in abundance and richness of potential host species causes parasites to become more extensive, infecting a higher number of available host species (Poulin & Mouillot, 2005). On the other hand, in urban areas of Europe, a lower occurrence of avian malaria and other blood parasites in blackbirds

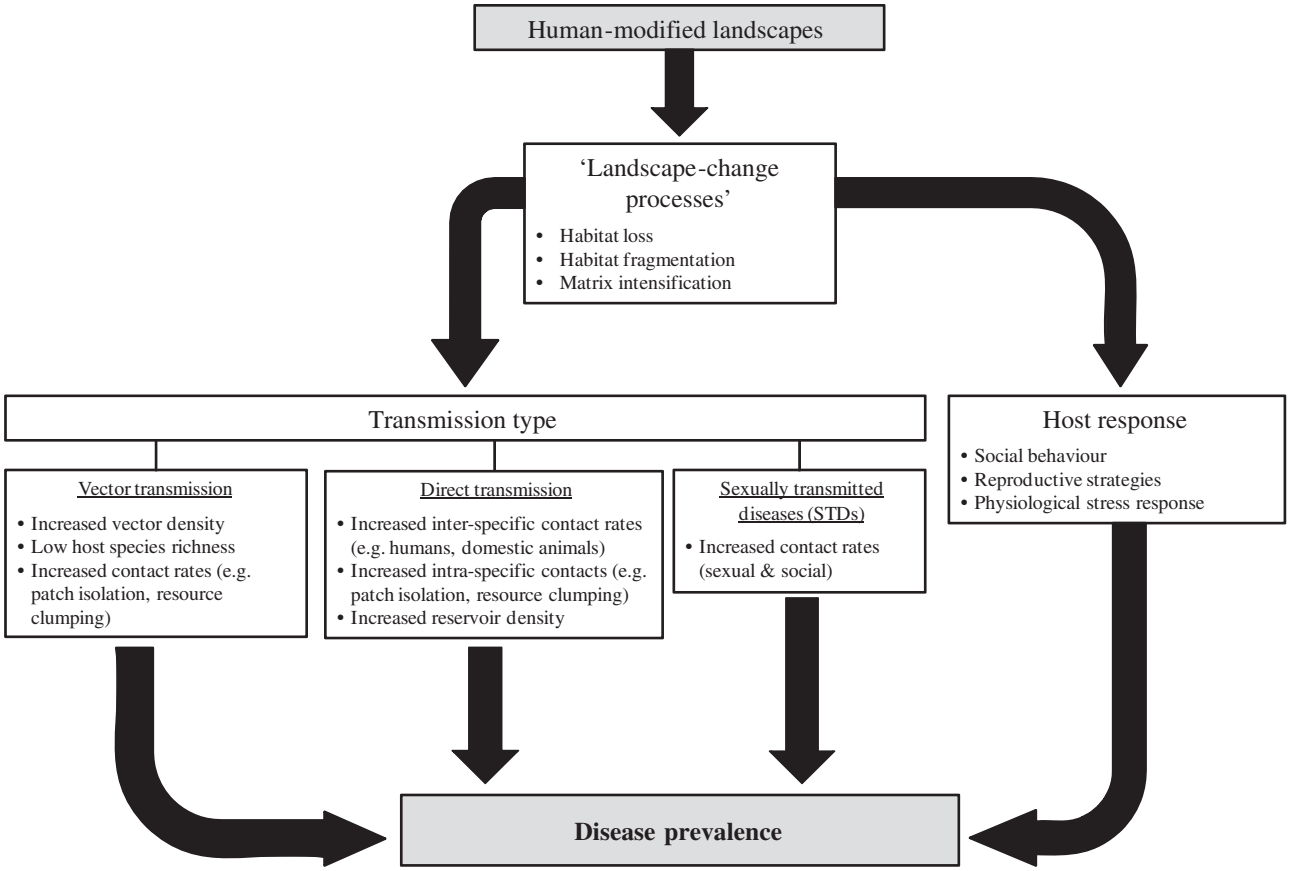

Fig. 1. Conceptual model illustrating key processes and examples linking human-modified landscape changes, disease transmission type, host response, and wildlife disease prevalence. Results of the review are provided for each transmission type, and are represented as the number of studies showing an increase (+), decrease (−) or varied (~) change in wildlife disease prevalence. For prevalence, 'varied' means that a single study identified both an increase and decrease in disease prevalence between different hosts, disease and/or landscape types.

(Geue & Partecke, 2008; Evans *et al*., 2009) was consistent with significant reductions in vector tick densities in these areas. Investigations into mosquito-transmitted diseases in the yellow-whiskered greenbul (*Andropadus latirostris*) and olive sunbird (*Cyanomitra olivacea*) of southern Cameroon found that variations in disease prevalence were most likely due to mosquito species responses to habitat fragmentation (Chasar *et al*., 2009). Likewise, Ezenwa *et al*. (2007) found that the infection prevalence of the West Nile virus from mosquitoes increased as a result of increasing wetland cover and not due to developed landscapes. A clear message from these studies is that the prevalence of vector-transmitted diseases in human-modified landscapes will vary, and be predominately related to the vector and/or hosts response to the landscape changes.

One common process that has received notable attention in vector-related transmission is the 'dilution effect'. This concept ties vector transmission to the presence and abundance of hosts (Lorange *et al*., 2005; Krasnov *et al*., 2006; Friggens & Beier, 2010), but disease and infection prevalence can either increase or decrease in modified landscapes. High host-species richness can lower parasite transmission if vectors feed on multiple host species that vary in their ability to contract, amplify and transmit the pathogen (Bradley & Altizer, 2007). An example is provided by the common fruit bat (*Artibeusja maicensis*) in the Barro Colorado Nature Monument in the Republic of Panama (Cottontail *et al*., 2009), where a negative relationship was found between the haemoparasite infection *Trypanosoma* sp. and the number of bat species in the area. Conversely, studies assessing the prevalence of Lyme's disease (Allan *et al*., 2003; LoGiudice *et al*., 2003) and West Nile virus (Bradley *et al*., 2008) have demonstrated that lower host diversity in human-modified landscapes creates situations where the proportional abundance of key reservoir hosts (e.g. white-footed mouse *Peromyscusleucopus*) can increase. These examples illustrate the variation in transmission processes of vector-transmitted diseases. The influence of habitat modification on vector-transmitted disease prevalence may vary among species (host and vector).

(*b*) *Directly transmitted disease*

Directly transmitted disease can show levels of variability in response to human-modified landscapes; but the current

data show a greater incidence of increase (Fig. 1). This increase may be a result of a more simplified transmission avenue requiring only the host response to human-modified landscapes, as opposed to responses from both host and vector in vector-transmitted disease. Diseases that are directly transmitted, through contact with infected hosts or their by-products, form a large proportion of wildlife diseases. Hantaviruses can be transmitted through direct transfer of infected bodily fluids (e.g. during aggressive behaviour and grooming), or indirect inhalation of contaminated excreta (Dearing & Dizney, 2010). Studies of directly transmitted disease have documented variations in disease prevalencein relation to landscape modification. Studies on *Toxoplasma gondii* in woodchucks (Lehrer *et al*., 2010) and chronic wasting disease in mule deer (*Odocoileus hemionus*) (Farnsworth *et al*., 2005) show that the prevalence increased in areas dominated by urbanization. Conversely, a study of Balinese macaques (*Macaca fascicularis*) found that increased anthropogenic activity led to an overall lessening of the intensity and diversity of gut parasites, most likely due to high contact with tourists and subsequent heavy provisioning (Lane *et al*., 2011).

Diseases that are transmitted directly between hosts typically follow a density-dependent transmission process, and therefore usually display a strong relationship with contact rates. In a simplified example, directly transmitted disease prevalence in species living in highly fragmented environments may be reduced due to a lack of connectivity reducing infection rates. However, in our review, the indication is that the isolation of habitats may in fact increase contact rates and subsequent transmission and prevalence, possibly due to clumping of resources and individuals. The number and duration of contacts per individual is positively associated with the probability of an individual becoming infected (Tompkins *et al*., 2011). However, a recent review of wildlife diseases by Tompkins *et al*. (2011) identified that a major challenge with contact dynamics and disease transmission lies in distinguishing the contacts that are potentially important to transmission from those that are not. A study on meerkats (*Suricata suricatta*) found that individuals that groomed others were more likely to be infected by *Mycobacterium bovis* than receivers of grooming. Conversely, individuals that were the subject of aggression, but not those initiating it, werealso the main drivers of infection of *M. bovis* (Drewe, 2010). A detailed understanding of host social and population dynamics is essential for host-pathogen dynamics of direct transmission to be understood.

(*c*) *Sexually transmitted diseases (STDs)*

This review identified no study that attempted to link human-modified landscapes with wildlife STD prevalence (Fig. 1). STDs were thought to be prime examples of frequency-dependent transmission, because sexual contact rates are believed to be independent of population density changes (Ryder *et al*., 2007; Greer *et al*., 2008; Smith *et al*., 2009*b*). Recent studies have found that STDs may be transmitted through social and other non-sexual contacts (Vitale *et al*., 2000; Cattani *et al*., 2003) and social, non-sexual contact will often incorporate a component of density dependence (Ryder *et al*., 2007). The transmission of a sexually transmitted mite, *Coccipolipus hippodamiae*, in the two-spot ladybird (*Adalia bipunctata*) depended more on the density of infected individuals in the study population than on the frequency of infection (Ryder *et al*., 2005). This supports the work of Antonovics, Iwasa & Hassell (1995) who indicated that a continuum of transmission dynamics exists between pure frequency and density dependence. Identifying a wildlife STD system that fits a density-dependent model that usually applies to other types of infectious disease (Ryder *et al*., 2005) has implications for species conservation. Management decisions based on pure frequency-dependent transmission in wildlife STD systems could yield unreliable results. This has implications for populations living in human-modified landscapes where the loss and fragmentation of habitat may reduce densities on a larger scale but may increase densities and contacts in isolated areas. Even a small degree of density dependence will result in increased parasite persistence with detrimental effects on host populations (Ryder *et al*., 2007). Even more critical is that only limited experimental investigations of STDs in wildlife populations have been conducted, and the majority are of invertebrates (Knell & Webberley, 2004; Webberley *et al*., 2006*a*,*b*; Nahrung & Clarke, 2007).

(3) Host response to human-modified landscapes

An increased disease prevalence is a threat to wildlife living in human-modified landscapes, because habitat loss, habitat fragmentation, and anthropogenic stressors restrict movement and dispersal (van der Ree *et al*., 2011), alter intra- and inter-species contact rates and the likelihood of disease spread (Smith *et al*., 2009*a*), and increase physiological stress (Brearley *et al*., 2010; Johnstone *et al*., 2011, 2012), which may lower immune responses (e.g. Sapolsky *et al*., 2000; Hussain, 2010) (Fig. 1). The mechanisms driving disease prevalence in human-modified landscapes may be influenced by how a species responds to these stressors, although they are poorly understood. In addition, the influence of human-modified landscapes on disease prevalence may be a result of changes to responses of disease carriers, such as vectors and reservoirs. For example, poor-quality or disturbed habitats are more likely to harbour a greater intensity and diversity of parasites relative to host populations in optimum habitats (Nunn & Altizer, 2006; Lane *et al*., 2011).

Transmission of many wildlife diseases depends on the rate at which individual hosts come into contact with one another (Wright & Gompper, 2005). In human-modified landscapes, loss and fragmentation of habitat change densities and social interactions of species through reduced connectivity or dispersal opportunities (reduce inter-patch contacts), create smaller patch sizes and/or resource clumping (increase contacts in small areas), and increase edge habitat (increase contacts with humans and/or exotic species). Resource clumping was found to be a key driver of contact rates and greater prevalence of parasites in raccoons (*Procyonlotor*)

(Wright & Gompper, 2005), while the risk of canid disease transmission in large carnivores (e.g. lions, panthers) is escalating due to increasing dog populations (Butler & du Toit, 2002). If the main driver is contact, then ecological factors that alter host contact rates will influence the structure and diversity of parasite assemblages (Wright & Gompper, 2005), and ultimately influence disease prevalence. Cowpox in voles is thought to be transmitted only through direct contact (Robinson & Kerr, 2001), but a range of factors may affect contact rates, such as breeding male and female territoriality, movement patterns of sub-adults, and dispersal that is seasonal and density dependent (Smith *et al*., 2009*b*). In addition to contact frequency and the type and direction of interactions (Drewe, 2010), it is imperative that host ecology and behaviour be considered when quantifying disease risk.

The ability of a species to adapt their physiological stress response to the many stressful perturbations in human-modified landscapes is likely to have implications for disease spread and transmission, particularly as increased physiological stress has been linked to a lowered immune response (McLean, 1982; Cohen & Williamson, 1991; Sapolsky *et al*., 2000; Martin, 2009). Although a small number of studies have assessed stress in response to landscape modification (e.g. Partecke, Schwabl & Gwinner, 2006; Brearley *et al*., 2012), there has been no attempt to determine the mechanisms linking landscape change, stress and disease. In addition to disease transmission type, it is likely that the tolerance of a host species towards human-induced landscape change, both ecologically and physiologically, will also play a role in determining the extent of disease transmission and its overall prevalence in a population.

IV. FUTURE RESEARCH DIRECTIONS

The studies we reviewed provide insight into the influence of human-induced habitat loss and fragmentation on wildlife disease, and we have identified three key areas requiring future research.

(1) Spatial and temporal effects on wildlife disease

Studies conducted at multiple spatial scales provide a more complete understanding of pattern-process relationships influencing species' distribution and abundance (Lindenmayer & Fischer, 2006). It stands to reason that characteristics present at a landscape-scale (e.g. habitat fragmentation, urbanization, total habitat) may have different impacts on wildlife disease compared to characteristics present at a patch-scale (e.g. edge intensity, patch size) or site-scale (e.g. habitat structure, resource availability).

Of the 19 studies reviewed, none assessed the influence of characteristics from multiple spatial scales on disease prevalence. While a large portion of studies included species-level parameters, the majority limited their analysis to a comparison between sites at a landscape level (e.g. those that were disturbed *versus* those that were not). This makes it difficult to determine which spatial scale has the greatest influence on wildlife disease. Studies conducted at a single spatial scale can explain only part of the overall impact of human-modified heterogeneous landscapes (Garden *et al*., 2006). The need to fill this gap in our knowledge is critical for wildlife conservation in human-modified landscapes, because wildlife and disease both may respond differently to the impacts of landscape modification at different spatial scales. Patch isolation and spatial configuration at a landscape scale may reduce disease transmission by limiting the dispersal of some wildlife species, whereas increasing patch size and spatial contagion of resources may influence host aggregations and contact patterns (Bradley & Altizer, 2007). The latter may ultimately result in increased disease transmission and prevalence. Although these are straightforward examples, they underline the importance of recognizing the potential influence of multiple spatial scales on wildlife disease prevalence for future studies to identify priority areas for conservation and management.

It is also important that future research incorporates temporal components of analysis into disease studies. Time-series assessments of landscape change and climate will provide a greater understanding of any lag effects on wildlife disease infection and prevalence. It is important to note that none of the reviewed studies linked temporal changes in human-modified landscapes with changes in wildlife disease prevalence. Establishing this link is essential because landscapes have temporal as well as spatial components (Marcucci, 2000; McAlpine *et al*., 2006), and associated wildlife responses to human-induced landscape change can be expected to occur over long periods of time (Tait, Daniels & Hill, 2005). It is likely that temporal parameters will influence wildlife disease systems, with changes in disease prevalence in a population lagging behind landscape modification. On the other hand, over time, the condition or quality of remnant habitats is likely to decline, whereas anthropogenic threats associated with the human-modified matrix will increase, as will intra- and inter-species competition. These processes are likely to create situations that favour disease transmission and prevalence, including increased physiological stress, increased contact rates and aggressive territorial behaviour due to resource clumping and limited availability as well as reduced dispersal opportunities.

(2) Links between habitat change, physiological stress and disease

While there is no evidence yet of a direct causal relationship between human-induced landscape change, physiological stress and disease, there are several reasons why the three maybe linked (see Johnstone *et al*., 2012). Many factors can act as stressors in animals. Physical and psychological stressors can include increased crowding, increased social

interaction, in adequate nutrition, dehydration, and temperature extremes. All of these stressors can be intensified in human-modified landscapes. The nutritional status of the host has long been associated with susceptibility to infectious disease and to its severity. The accepted model holds that inadequate nutrition impairs the functioning of the immune system, resulting in increased susceptibility to infection. It has even been suggested that the nutritional status of the host may have an influence beyond the host itself, such that the genome of a virus may be altered. Beck & Levander (2000) concluded that the oxidative stress status of mice due to a nutritional deficiency led to a non-virulent virus changing into a virulent form.

To understand how immunity can be affected by stress, it is important to consider aspects such as the nature, duration and intensity of the stressor, as well as the ecology of the target species, their coping responses and/or tolerance (Martin, 2009). Importantly, stressors which tend to occur for longer, such as those associated with habitat modification, mirror reductions of most immune processes, while short-term stressors, such as predation attempts, enhance most immune responses (Martin, 2009). As illustrated by Martin (2009), many of the studies of stress-immune interactions have excluded wild animals instead focusing on species typically bred for *in vivo* studies, such as rodents. It is not known whether the stress response in laboratory rodents occurs at the same magnitude and consequences in wild rodents, and whether there are similarities with other wild animal groups.

Over the past decade, evidence has shown increased physiological stress in response to human-induced landscape change and associated stressors in numerous species, including the squirrel glider *Petaurus norfolcensis* (Brearley *et al*., 2012), agile antechinus *Antechinus agilis* (Johnstone *et al*., 2011, 2012), African elephant *Loxodonta africana* (Ahlering *et al*., 2011), elk *Cervuselaphus* (Millspaugh *et al*., 2001), wolf *Canis lupus* (Creel *et al*., 2002), spotted hyena *Crocutacrocuta* (Van Meter *et al*., 2009), white-crowned sparrow *Zonotrichialeuco phrysoriantha* (Crino *et al*., 2011), and yellow-eyed penguins *Megadyptes antipodes* (Ellenberg *et al*., 2007). Many species are now confronted with novel and potentially stressful anthropogenic pressures, including human disturbance (Fernandez-Juricic, 2002), exotic predator species (Mooney & Hobbs, 2000), transport networks (Coffin, 2007; Rytwinski & Fahrig, 2007), residential developments (Baker & Harris, 2007; Brearley *et al*., 2010), exposure to pollutants (Burger *et al*., 2004), and changes to abiotic [e.g. warmer ambient temperatures (McLean, Angilletta & Williams, 2005)] and biotic conditions [e.g. vegetation structure and composition (Chen, Franklin & Spies, 1992; Brearley *et al*., 2011)]. Ultimately, over-activation of the acute stress response by an array of stressors has the potential to generate chronic stress and greater susceptibility to disease and reduced fecundity and survivorship (Sapolsky *et al*., 2000). With a growing recognition of the role of human-modified landscapes and increased physiological stress in wildlife, and the known link between stress and immune competency, it seems logical that the link between the three processes must be explored in future research.

(3) Sexually transmitted diseases

The lack of STD-based studies, including transmission dynamics, is well recognized (Ryder *et al*., 2005). A small number of studies have begun to address this limitation, but our review identified no study that directly assessed the influence of human-modified landscapes on a STD system in wildlife. With the exception of invertebrates, the only study that has been conducted on STDs is an investigation of chlamydiosis in the koala (*Phascolarctos cinereus*) (Augustine, 1998). This investigation involved stochastic population modeling with no experimental field validations, and the author recognized that the conclusions drawn were limited due to the lack of mechanistic transmission analysis. Furthermore, it only discussed the impacts of fragmentation and did not incorporate related landscape-change parameters into the model. As a result, it did not qualify for inclusion in this review.

Nonetheless, results from Augustine (1998), combined with additional theoretical discussions on koalas (Gordon, McGreevy & Lawrie, 1990; White & Timms, 1994), suggest that chlamydiosis may be a population regulator, and that the disease acting alone does not appear to limit population growth. In fact, it has been suggested that the greatest conservation concern would be a loss of this unique host-disease relationship due to disease extinction (Augustine, 1998). As a population regulator, chlamydiosismay limit population densities in unmodified or isolated environments where high densities of koalas have been associated with over-browsing (Phillips, 2000); however it contributes to population crashes in human-modified landscapes where there are multiple causes of unsustainable koala mortality (Rhodes *et al*., 2011). Augustine (1998) suggested that koala extinctions will only occur if other non-disease factors change birth and mortality rates. Regardless of the processes involved in largely intact landscapes, human-modified landscapes represent additional challenges to animal survival that need to be understood if we are to reduce the influence of disease on wildlife populations. Specifically for STD systems, the most important potential influence of human-induced landscape change will most likely be spatial- and temporal-scale impacts on contact rates, reproductive strategies and social behaviours.

In a study of koalas and climate change on the Liverpool Plains, north-west New South Wales, the prevalence of the disease chlamydiosis, which can lead to infertility, was found to increase markedly over a 2-year period which included intense heat waves during a drought (Lunney *et al*., 2012). Clinical chlamydiosis was historically absent or rare in this population, but its prevalence increased dramatically following intense stress from extreme hot weather in 2009. Lunney *et al*. (2012) also reviewed studies of chlamydial disease and population decline, and linked the disease to koala populations under stress from habitat loss and fragmentation. This example demonstrates the importance

of studying the interactions between wildlife disease and other threatening processes, including climate change.

V. CONCLUSIONS

(1) This review has highlighted that there are rarely consistent relationships between wildlife disease and human-modified landscapes. Instead, the potential link between human-induced landscape change and disease prevalence in wildlife populations is complex and variable. In addition, variation is evident between transmission types and landscape types.

(2) The greatest positive influence on disease prevalence appears to occur in urban landscapes and in directly transmitted disease systems. As a result, future research needs to take a spatial ecological view of the link between human-induced landscape change and wildlife disease prevalence by exploring mechanisms involved with disease transmission type and key host responses.

(3) The future research topics and conceptual models discussed provide a direction for further studies to allow us to understand the influence of human-induced landscape change on a range of wildlife diseases, and apply this understanding to conservation management.

(4) While the role of urban and landscape modification in human infectious disease is well established, much less is known about how spatially explicit, human-induced habitat loss and fragmentation influence wildlife-pathogen interactions. The current knowledge base provides a good indication that the potential influences of human-modified landscapes on wildlife disease are highly variable. However, most studies are based on a one-dimensional comparison between unmodified and modified sites without incorporating the spatially and temporally explicit quantitative approach required to understand this problem better. This is part of the challenge for future researchers.

(5) There are few studies that have attempted to separate the independent effects of factors such as habitat loss and fragmentation on wildlife disease, including causal factors operating at multiple scales. We conclude that this understanding is critical for conserving wildlife in human-modified landscapes, particularly in rapidly expanding urban areas where increases in wildlife disease prevalence have been recorded.

(6) There is a pressing need to understand better the potential causal link between the processes of human-modified landscapes and associated factors, such as matrix hostility and loss of connectivity, with physiological stress and disease susceptibility in animals. There is a good understanding of the influence of elevated stress on decreased immunity, but well-designed observational and experimental studies are needed to separate the cause-effect relationships in natural systems. Understanding the links between the processes of habitat loss, fragmentation, physiological stress and disease within a spatially and temporally explicit framework will enable a more targeted approach to undertaking wildlife-disease conservation in human-modified landscapes.

(7) There is a need to understand disease transmission processes better in human-modified landscapes. This is particularly important for STDs. In order to understand the potential influence of human-induced landscape change on a STD system, research questions should be directed towards a spatial and temporal analysis, with a focus on contact rates, reproductive strategies and social behaviours.

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