EcoHealth 16, 647–658, 2019
https://doi.org/10.1007/s10393-018-1336-3

EcoHealth

Original Contribution

Land-Use Change Alters Host and Vector Communities and May Elevate Disease Risk

Fengyi Guo,[1] Timothy C. Bonebrake,[1] and Luke Gibson[1,2]

[1]*School of Biological Sciences, University of Hong Kong, Hong Kong, China*
[2]*School of Environmental Science and Engineering, Southern University of Science and Technology, Shenzhen, China*

Abstract: Land-use change has transformed most of the planet. Concurrently, recent outbreaks of various emerging infectious diseases have raised great attention to the health consequences of anthropogenic environmental degradation. Here, we assessed the global impacts of habitat conversion and other land-use changes on community structures of infectious disease hosts and vectors, using a meta-analysis of 37 studies. From 331 pairwise comparisons of disease hosts/vectors in pristine (undisturbed) and disturbed areas, we found a decrease in species diversity but an increase in body size associated with land-use changes, potentially suggesting higher risk of infectious disease transmission in disturbed habitats. Neither host nor vector abundance, however, changed significantly following disturbance. When grouped by subcategories like disturbance type, taxonomic group, pathogen type and region, changes in host/vector community composition varied considerably. Fragmentation and agriculture in particular benefit host and vector communities and therefore might elevate disease risk. Our results indicate that while habitat disturbance could alter disease host/vector communities in ways that exacerbate pathogen prevalence, the relationship is highly context-dependent and influenced by multiple factors.

Keywords: biodiversity, deforestation, land-use change, dilution effect, disease, habitat loss

Introduction

Humans have transformed the terrestrial biosphere for thousands of years, yet only in the past few decades have land-use changes (such as deforestation, agriculture development, and urbanization) extended to the global scale and irreversibly altered the biosphere (Foley et al. 2005; Ellis 2011; Hansen et al. 2013). These drastic habitat changes have led to an accelerated decline in global biodiversity (Foley et al. 2005; Gibson et al. 2011; Newbold et al. 2015). Concurrently, infectious disease emergence and transmission are increasing in response to anthropogenic land-use changes (Patz et al. 2004, 2008; Murray and Daszak 2013; Gottdenker et al. 2014; Pienkowski et al. 2017), suggesting that such habitat disturbances, while generally causing biodiversity losses, might favor important disease hosts and vectors, typically habitat generalists with high growth and reproductive rates, thereby elevating disease risk (Keesing et al. 2010; Kilpatrick 2011; Liu et al. 2017; Ostfeld 2017). On the other hand, undisturbed habitats can sometimes support higher densities of disease pathogens and/or vectors, and are thus considered key sources of important infectious diseases such as leishmaniasis and malaria (re-

Published online: April 24, 2018

Correspondence to: Luke Gibson, e-mail: biodiversity@sustc.edu.cn

viewed in Wood et al. 2014). Despite the controversy, experimental studies on plant-fungus and plant-virus systems suggest that habitat disturbance generally increases community-level competence in pathogen transmission (and hence disease risk) by altering host/vector community composition (Lacroix et al. 2014; Liu et al. 2017).

Community competence in disease transmission is defined as the sum of abilities (e.g., susceptibility of amphibian hosts to trematode infection in Johnson et al. 2013a) of different host/vector species to support or transmit pathogens, each multiplied by their abundance (Johnson et al. 2015). Changes in host/vector community assembly following disturbance could drive disease outbreaks in different ways (see Table 1 with representative examples for each mechanism below). The first and most straightforward path is through the increase in absolute host/vector abundance (density) and the corresponding increase in density-dependent disease transmission intensity in disturbed areas (Keesing et al. 2006; Ryder et al. 2007). Alternatively, competence of each host/vector species might also be increased through changes in individual physiology such as body size, as larger-sized vectors have higher fecundity and hence greater infection potential (e.g., malaria vector *Anopheles arabiensis*, Ameneshewa and Service 1996). Moreover, larger bodied hosts provide more space and resources for ectoparasite colonization (e.g., shell disease of the European lobster, Davies et al. 2015), and greater energy requirements associated with larger body size also increase the chance of ingesting endoparasites (e.g., fungal parasite *Metschnikowia bicuspidata* of *Daphnia*, Hall et al. 2007).

Apart from direct changes in individual species abundance/competence, overall community competence in pathogen transmission could also be increased by raising the relative proportion of species that are more competent at disease transmission. The ''ubiquity-competence'' relationship suggests that highly competent hosts usually dominate in species-poor assemblages (Lacroix et al. 2014; Liu et al. 2016, 2017); therefore, declining biodiversity would indirectly intensify infection prevalence and disease risk, as predicted by the ''dilution effect'' hypothesis (Ostfeld and Keesing 2000a; Keesing et al. 2006, 2010; Johnson and Thieltges 2010; Ostfeld and Keesing 2012; Civitello et al. 2015; but also see Randolph and Dobson

Table 1. Mechanisms Explaining Disease Risk Associated with Host/Vector Community Composition.

Ecological metric	Mechanism	Representative example	References
Abundance (+)	Increased vector density	Increased frequency of dengue emergence (transmitted by *Aedes aegypti*) especially in regions with no effective mosquito control	Gubler (2011)
	Increased reservoir density or contact rates between hosts	Increased infection rate of *Ambystoma tigrinum* virus with higher infected host density	Greer et al. (2008)
Body size (+)	Increased vector fecundity	Increased survival and parous rate of larger-sized malaria vectors *Anopheles arabiensis*	Ameneshewa and Service (1996)
	Larger host surface area and more resources for colonization	Higher parasite load on larger *Rhabdomys pumilio*	Froeschke et al. (2013)
		Increased shell disease occurrence probability for larger European lobsters (*Homarus gammarus*)	Davies et al. (2015)
	Higher foraging-dependent transmission rate	Higher fungal parasite infection rates with increasing host (*Daphnia*) body size	Hall et al. (2007)
Diversity (−)	Reduced encounter between hosts and pathogens through transmission interference	Decreased foliar fungal disease severity with increasing host plant diversity while controlling for equal abundance	Liu et al. (2016)
	Constrained abundance of susceptible hosts through competition or predation from other species	Decreased proportion of most competent hosts with increasing amphibian species richness	Johnson et al. (2013a, b)

Direction of effect is shown in parentheses (correlation between ecological metrics and community competence to support infection).

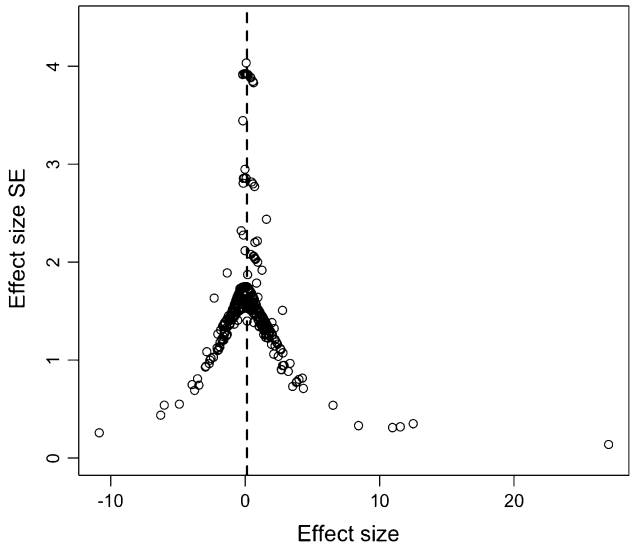

Figure 1. Funnel plot of effect size standard error plotted against effect size. The symmetric plot suggests there is no relationship between effect size and study size, and that those studies with small (or negative) effect sizes are not necessarily published at a lower frequency.

2012; Wood and Lafferty 2013). As synthesized in recent review papers (e.g., Wood et al. 2014; Johnson et al. 2015), the dilution effect can operate either through transmission interference (i.e., incompetent hosts serve to "distract" vectors/pathogens away from the most competent hosts and hence reduce encounter probability) or susceptible host regulation (i.e., incompetent hosts regulate competent host density through competition or predation). Based on the dilution effect hypothesis, reduced host diversity (even if the overall density remains constant) will increase the prevalence of frequency-dependent diseases in particular (where transmission is dependent upon the proportion of infected individuals), such as those transmitted by highly mobile vectors like mosquitoes (Keesing et al. 2006, 2010; Ostfeld and Keesing 2012; Civitello et al. 2015). Some well-studied examples of this phenomenon include Lyme disease (e.g., Ostfeld and Keesing 2000a; Allan et al. 2003; LoGiudice et al. 2003) and West Nile virus (e.g., Ezenwa et al. 2006; Swaddle and Calos 2008), as well as some plant fungal pathogens (Lacroix et al. 2014; Liu et al. 2016, 2017).

Here, we conduct a meta-analysis to investigate the effects of global land-use changes on infectious disease host/vector communities, and the implications for disease transmission risk. From an exhaustive literature search, we examined 331 pairwise data points from 37 studies comparing disease host and/or vector communities in both disturbed and undisturbed sites. Specifically, we compared host/vector species abundance, diversity, as well as individual body size between sites. We also inferred changes in disease risk based on the above relationships between three ecological metrics and community competence.

Methods

Data Compilation

We searched for relevant studies published between 1975 and May 2016 in all Web of Science databases using the search query (TS = [(clear-cutting* OR deforest* OR agriculture conversion* OR secondary forest* OR forest fragment* OR habitat loss* OR habitat conversion* OR pasture* OR logged*) AND (disease* OR vector* OR parasit* OR pathogen* OR dilution effect*) AND (abundance* OR density* OR diversity* OR richness*) AND (biodivers* OR ecosystem* OR ecology*) AND (bird* OR mammal* OR reptile* OR amphibia* OR arthropod* OR plants* OR lepidoptera* OR hymenoptera* OR arachnid* OR coleoptera* OR diptera* OR homoptera* OR isoptera* OR insect* OR rodent* OR primate* OR bat*)]). Additional studies from bibliographic references of the gathered articles were also reviewed if relevant.

We compiled data from 37 studies (out of 2103 papers from the initial search) that compared disease host or vector communities at multiple sites (where sample size > 1) in both disturbed and undisturbed habitats. Most studies were not included due to lack of replication (hence variance) in disturbed/undisturbed sites, or the absence of undisturbed control sites. The definition of undisturbed habitats varied by study, but most commonly was represented by continuous primary forests (Gibson et al. 2011). We classified land-use changes into 10 disturbance classes based on descriptions specified in the source literature: agroforestry, agriculture, clear-cut forests, disturbed/hunted forests, forest edges/ecotones, forest fragments, pastures, plantations, secondary forests, and villages.

For each study, we recorded the measured ecological metrics of disease hosts and/or vectors into three major categories: abundance (e.g., density, mean number of captures), diversity (e.g., species richness, Simpson and Shannon diversity indices), and size (e.g., body mass, length). When results were only reported in figures, we used DataThief (http://www.datathief.org) to extract data.

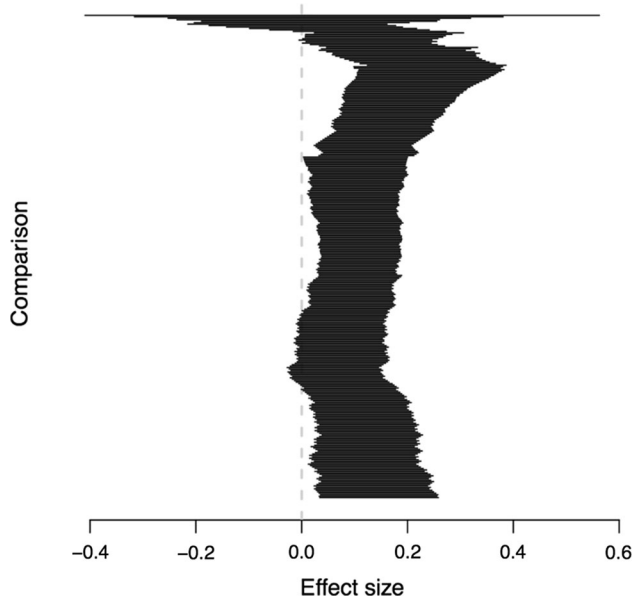

Figure 2. Cumulative meta-analysis of the dataset sorted by precision, from comparisons with small standard errors to those with large standard errors. At each comparison (n = 331), the cumulative effect size and 95% confidence intervals are plotted. The effect size is generally positive and does not overlap with 0 (gray dashed line) at any point after the first 257 comparisons. Overall, the addition of more imprecise comparisons (those with greater standard errors) causes little change to the cumulative effect size (and confidence intervals).

For each comparison, we recorded both the region (Africa, Asia, Europe, North America, Oceania, and South America) and the taxonomic group (arthropods, birds, mammals, plants, reptiles) of study subjects. When specified in the source literature, we also recorded the major type of pathogen (bacterium, fungus, macroparasite [including both endoparasites like helminths and ectoparasites like fleas], parasitoid, protozoan, virus) transmitted or sustained by the study subjects, as well as the associated diseases caused by these pathogens. For those organisms which can be the host/vector of multiple diseases, we logged only the focal pathogen transmitted/hosted by the study subjects as identified in the original study. For sampling of common hosts (e.g., rats) and vectors (e.g., mosquitoes, ticks), we included all comparative data even if the diseases and pathogens were not identified for each species.

Meta-Analysis

We calculated the weighted average of the standardized difference between mean ecological metrics of host/vector communities in disturbed and undisturbed sites to measure the effect size of human-driven land-use changes. For each comparison, we calculated Hedges' g, the difference between disturbed and undisturbed group means, standardized using the pooled standard deviation of the two groups (Borenstein et al. 2009), defined as:

$$g = \frac{x_{\text{disturbed}} - x_{\text{undisturbed}}}{SD_{\text{pooled}}}$$

where

$$SD_{\text{pooled}} = \sqrt{\frac{(n_{\text{disturbed}} - 1) * SD^2_{\text{disturbed}} + (n_{\text{undisturbed}} - 1) * SD^2_{\text{undisturbed}}}{n_{\text{disturbed}} + n_{\text{undisturbed}} - 2}}$$

We used the conversion factor J to calculate the bias-corrected metric, g^*, defined as $g^* = Jg$ (Borenstein et al. 2009), where

$$J = 1 - \frac{3}{4 * (n_{\text{disturbed}} + n_{\text{undisturbed}} - 2) - 1}$$

We calculated effect sizes using the random-effects model, where effect sizes of individual comparisons are weighted by the inverse of within-study variance plus between-study variance (Borenstein et al. 2009).

The effect size was defined as positive when the ecological metric increased in disturbed sites. Alternatively, the effect size was negative when the measured disturbance led to a decrease in the ecological metrics of disease hosts/vectors. We are ultimately interested in the change in pathogen-transmitting competence at the community level (i.e., disease risk). However, because of the dilution effect, species diversity is predicted to affect community competence in the direction opposite to abundance and size. We therefore inverted the effect size for all diversity comparisons (n = 45), such that an increased diversity index was marked as a negative effect size, and a decreased diversity index was marked as a positive effect size. As such, positive effect sizes in our results imply increasing community competence in disturbed sites, while negative values suggest lower disease risk. We computed the median effect size and 95% confidence intervals for the full dataset, and for each subgroup of six variables (subject, pathogen type, taxonomic group, ecological metric, region, and disturbance type) by resampling 331 comparisons (with replacement) in each 10,000 bootstrap iteration. To address autocorrelation in the multiple comparisons, we used another resampling procedure based on 10,000 bootstrap samples (with replacement), in which only one row was sampled

Table 2. Effect Sizes by Six Core Variables.

	Comparisons	Studies	Mean ES	CI	Resample median	Resample CI
Overall	331	37	0.15	0.03, 0.26	0.15	0.04, 0.26
By study	331	37			0.19	− 0.12, 0.50
Subject						
Host	78	22	0.25	0.04, 0.46	0.25	0.03, 0.49
Vector	253	18	0.11	− 0.02, 0.25	0.11	− 0.02, 0.24
Pathogen type						
Bacterium	31	4	0.06	− 0.41, 0.54	0.06	− 0.43, 0.55
Fungus	3	1	1.26	− 2.77, 5.29	1.26	− 2.67, 3.80
Macroparasite	31	9	0.29	0.05, 0.54	0.29	0.09, 0.54
Parasitoid	10	4	0.19	− 0.61, 1.00	0.21	− 0.76, 1.01
Protozoan	86	10	− 0.28	− 0.51, − 0.04	− 0.27	− 0.51, −0.05
Virus	33	6	1.29	0.92, 1.67	1.29	0.92, 1.69
Taxonomic group						
Arthropods	270	24	0.09	− 0.04, 0.22	0.09	− 0.04, 0.22
Birds	8	4	1.02	− 0.17, 2.20	1.00	− 0.08, 4.43
Mammals	48	10	0.31	0.12, 0.49	0.30	0.13, 0.52
Plants	3	1	1.26	− 2.77, 5.29	1.26	− 2.67, 3.80
Region						
Africa	72	8	0.36	0.09, 0.62	0.36	0.11, 0.62
Asia	63	3	0.67	0.34, 0.99	0.67	0.35, 1.00
Europe	18	7	− 0.39	− 0.87, 0.10	− 0.37	− 1.01, 0.15
North America	33	7	0.62	0.15, 1.09	0.63	0.14, 1.15
Oceania	22	4	0.09	− 0.14, 0.32	0.09	− 0.16, 0.29
South America	123	8	− 0.16	− 0.31, 0	− 0.16	− 0.30, −0.01
Ecological metric						
Abundance	251	32	0.04	− 0.10, 0.17	0.03	− 0.10, 0.17
(−) Diversity	45	14	0.55	0.20, 0.90	0.55	0.21, 0.93
Size	35	5	0.39	0.16, 0.61	0.38	0.17, 0.63
Host abundance	46	18	0.10	− 0.21, 0.42	0.10	− 0.21, 0.44
Host abundance (density-dependent)	41	15	− 0.03	− 0.34, 0.27	− 0.03	− 0.34, 0.27
(−) Host diversity	13	7	0.90	0.15, 1.64	0.89	0.09, 1.65
Vector abundance	205	16	0.02	− 0.13, 0.17	0.02	− 0.13, 0.17
(−) Vector diversity	32	8	0.39	0.01, 0.77	0.39	0.05, 0.82
Disturbance type						
Agroforestry	8	4	− 0.05	− 0.86, 0.76	0.01	− 0.91, 0.95
Agriculture	46	8	0.59	0.19, 0.99	0.59	0.21, 1.04
Clear-cut forest	16	1	0.25	− 0.43, 0.94	0.24	− 0.44, 1.03
Disturbed/hunted	25	7	0.05	− 0.43, 0.53	0.05	− 0.46, 0.54
Forest edge	33	2	0.01	− 0.19, 0.21	0.01	− 0.15, 0.20
Forest fragment	42	8	0.50	0.28, 0.71	0.49	0.29, 0.75
Pastures	25	9	− 0.23	− 0.74, 0.29	− 0.23	− 0.81, 0.35
Plantation	4	2	0.91	− 0.97, 2.79	0.91	− 0.29, 9.06

Table 2. continued

	Comparisons	Studies	Mean ES	CI	Resample median	Resample CI
Secondary forest	38	7	0.21	− 0.08, 0.49	0.20	− 0.08, 0.48
Village	94	7	− 0.12	− 0.34, 0.10	− 0.12	− 0.33, 0.09

Average random-model effect sizes (''Mean ES,'' ''CI'') calculated for full dataset and by subject (host or vector), pathogen type, taxonomic group, region, ecological metric, and disturbance type. For each data subset, we repeated this procedure after resampling the random-model effect size calculations using 10,000 bootstrap samples (with replacement), from which we generated 95% confidence intervals (''Resample median,'' ''Resample CI''). To address potential spatial and temporal autocorrelation from studies that include many comparisons (e.g., multiple measurements of the same taxa, measurements of multiple taxa, measurements of multiple disturbance types), we repeated this procedure after resampling one row per study for only the full dataset, again using 10,000 bootstrap samples (''By study''). ''Comparisons'' and ''Studies'' represent the number of pairwise comparisons and studies for each group.

per study (thus 37 comparisons per iteration), to generate median effect size and 95% confidence intervals for the overall effect (Efron and Tibshirani 1991). The confidence intervals between the results of the two resampling methods were similar (Table 2). Therefore, the impact of duplicate comparisons appears negligible and we hereafter only report the results based on the first resampling procedure.

Results

We examined the funnel plot and plot of cumulative effect size to test for publication bias (Borenstein et al. 2009). The symmetric funnel plot (Fig. 1) suggests that studies with small effect sizes are not necessarily published at lower frequency. When sorting the data set by precision (Fig. 2), the addition of more imprecise comparisons (with higher standard error) does not cause substantial changes to the cumulative effect size and confidence intervals. Consequently, our results do not appear to be affected by publication bias.

The positive overall effect size (bootstrapped median: 0.15, 95% confidence interval: 0.04–0.26, $n = 331$) suggests increased community competence of pathogen transmission in disturbed habitats (Table 2). These impacts of measured land-use changes could be attributed to changes in three ecological metrics (Table 2, Fig. 3a). Not surprisingly, species diversity was the most sensitive metric (with greatest effect size) and declined dramatically in association with land-use changes (effect size: 0.55, 0.21–0.93, $n = 45$). This general pattern of diversity loss was consistent when separated by hosts (0.89, 0.09–1.65, $n = 13$) and vectors (0.39, 0.05–0.82, $n = 32$) (Table 2). In contrast, body size/mass of hosts and/or vectors generally increased in disturbed habitats (0.38, 0.17–0.63, $n = 35$). However, the most common metric, abundance, did not respond significantly to measured disturbances for both hosts and vectors (Table 2). As host abundance usually indicates transmission risk of density-dependent diseases, we isolated host abundance data primarily linked with those diseases, but the 95% confidence interval of effect size still straddled 0 (Table 2).

Combining different ecological metrics, the effect size also varied considerably by taxonomic group, pathogen type, region and disturbance type (Table 2, Fig. 3). Taxonomically, mammal host competence was strengthened by measured land-use changes, with an effect size of 0.30 (0.13–0.52, $n = 48$), while arthropods, which comprised 82% of species comparisons, did not respond directionally to disturbance (Table 2). Among the identified diseases and pathogens, hosts and/or vectors of viral (1.29, 0.92–1.69, $n = 33$) and parasitical (0.29, 0.09–0.54, $n = 31$) pathogens showed a substantial increase in disease transmission competence in disturbed habitats. On the other hand, protozoal hosts/vectors displayed declining competence from habitat disturbances (− 0.27, − 0.51 to − 0.05, $n = 86$), while bacterial hosts/vectors did not show a directional response (Fig. 3b).

Disease hosts and vectors from different regions showed mixed responses to habitat disturbance (Table 2, Fig. 3c). While the community competence generally increased on most continents including Africa (0.36, 0.11–0.62, $n = 72$), Asia (0.67, 0.35–1.00, $n = 63$), and North America (0.63, 0.14–1.15, $n = 33$) following habitat disturbance, the recorded land-use change in South America (− 0.16, − 0.30 to − 0.01, $n = 123$) was found to slightly reduce disease risk. No trend was observed in other continents (Fig. 3c). The type of habitat disturbance also had diverse impacts on disease hosts and vector communities (Table 2, Fig. 3d). Responses to most types of land-use changes appeared to be non-directional, except for the

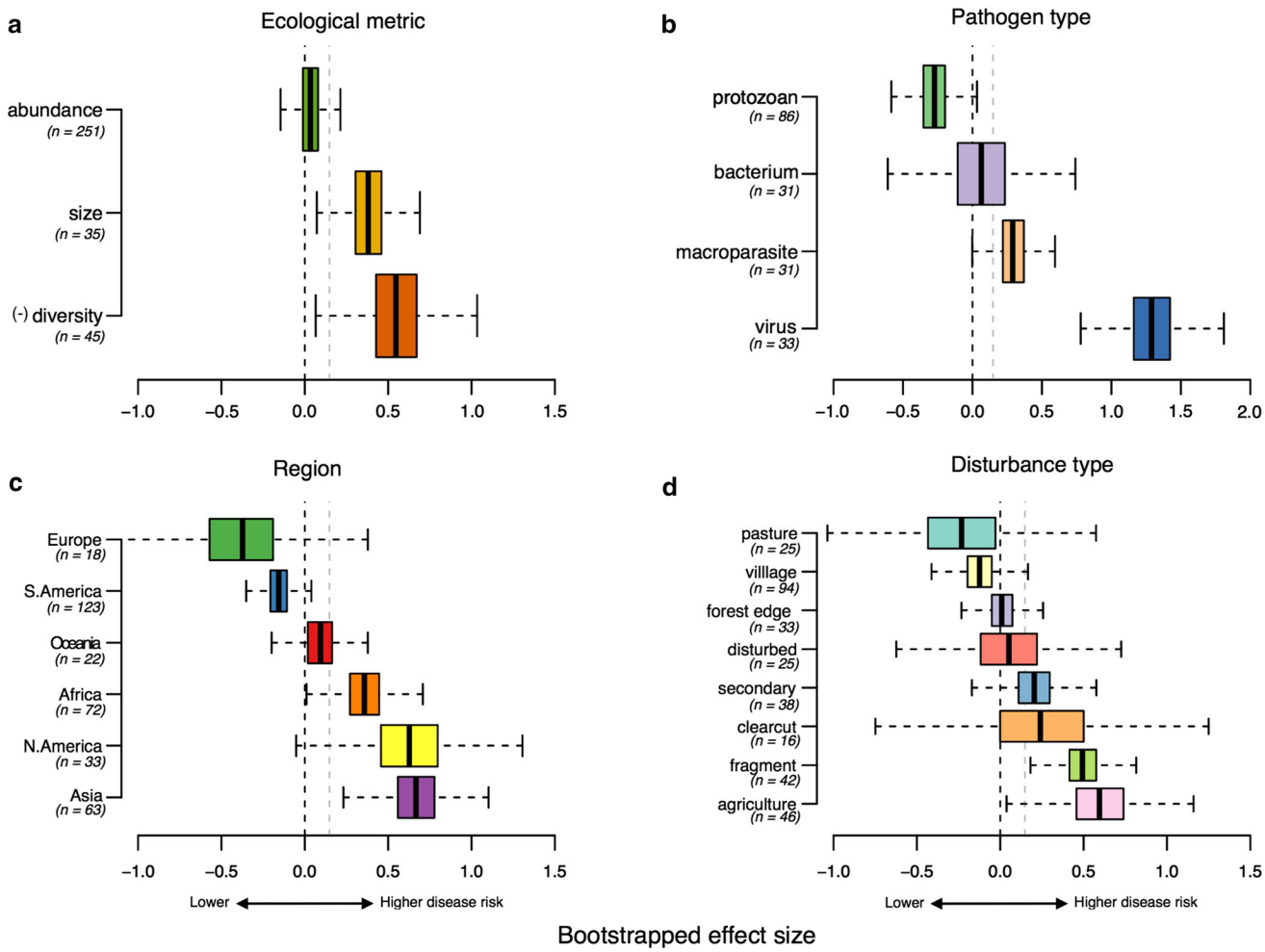

Figure 3. Box plots of bootstrapped effect sizes of disease hosts/vector competence based on **a** ecological metric; **b** pathogen type; **c** region; and **d** disturbance type. Boxes display the median values and interquartile ranges (IQR) of 10,000 resampled (with replacement) effect size calculations for each group; whiskers represent either (i) the most extreme upper or lower value or (ii) 1.5 times the IQR from the edge of the box. Gray dashed lines represent the median effect size for the entire dataset and black dashed lines show effect size of zero. Sample size is shown in parentheses (subgroups of less than 10 comparisons omitted). Note that scale bar for panel (**b**) is different from the other panels.

distinctive increase in host/vector community competence of forest fragments (0.49, 0.29–0.75, $n = 42$) and agricultural lands (0.59, 0.21–1.04, $n = 46$), which jointly made up 27% of total land-use change comparisons (Table 2, Fig. 3d).

Discussion

The mixed responses in different subcategories (e.g., pathogen type, taxonomic group, region, and disturbance type) and the associated small effect sizes (Maher et al. 2013) suggest that the relationship between human land-use changes and disease transmission is complex and context-dependent (Young et al. 2013; Gottdenker et al. 2014). However, the positive overall effect size indicates the potentially increasing disease risk in disturbed habitats, consistent with other recent studies (Gottdenker et al. 2014; Pienkowski et al. 2017). When separated by ecological metric, there was a clear trend of increased host/vector size and decreased species diversity in disturbed habitats although host and/or vector abundance were similar between disturbed and undisturbed sites. These findings suggest that land-use changes trigger increases in body size of disease hosts and/or vectors, and a loss in host/vector diversity, possibly favoring human-tolerant species thriving at the cost of others (see below).

Referring to the dilution effect hypothesis (i.e., negative correlation between host diversity and disease prevalence; Keesing et al. 2006, 2010), the observed decline in

host and/or vector species diversity might indicate rising risk of infectious disease transmission in human-disturbed habitats. A meta-analysis on 61 parasite species by Civitello et al. (2015) provides strong supporting evidence for the dilution effect, whereby the reduction in frequency (i.e., relative proportion) of highly competent hosts was found to inhibit disease risk, independent of host density (i.e., absolute abundance). Other recent studies have provided mounting empirical evidence supporting the dilution effect (Johnson et al. 2013a, b; Rottstock et al. 2014; Lacroix et al. 2014; Parker et al. 2015; Liu et al. 2016, 2017). Vector diversity, on the other hand, is more problematically related to pathogen prevalence. As many tick-borne pathogens and plant DNA viruses are highly vector-specific, the greater vector diversity in undisturbed habitats might also lead to more diverse pathogen populations (Cumming and Guégan 2006; Power and Flecker 2008). Similar relationships have also been proposed between specialist pathogens and host richness (Dunn et al. 2010). Nonetheless, as reviewed in Johnson et al. (2015), pathogen diversity is not essentially equivalent to disease incidence and severity, but rather many empirical studies have reported the negative correlation between disease risk and parasite diversity, likely due to the competition between pathogens for shared resources and the ''transmission barrier'' in a more diverse community for specialist pathogens (Johnson et al. 2013b; Rottstock et al. 2014; Liu et al. 2016). Moreover, the ''susceptible host regulation'' principle in the dilution effect of hosts could still apply to vectors if the inclusion of less competent (in terms of pathogen transmission) vectors in undisturbed habitats indirectly reduced the abundance of the most competent vectors, and thus lessen disease transmission intensity.

One key assumption for the above deduction based on the dilution effect hypothesis is that those species eliminated in disturbed habitats are mostly incompetent hosts/vectors, while the highly competent species become dominant in species-poor assemblages (Ostfeld and Keesing 2000b; Keesing et al. 2006; Ostfeld 2017). Johnson et al. (2013a) provide empirical evidence for this assumption by comparing amphibian communities surveyed in 345 wetlands, focusing on the pathogen *Ribeiroia ondatrae*. In addition, in a manipulation experiment of artificial fertilization of natural alpine meadows in Tibet, Liu et al. (2017) demonstrated that the non-random pattern of plant species loss (host species with lower proneness to foliar fungal pathogens are extirpated first) led to increasing community competence along the disturbance gradient. However, estimating host/vector competence or assessing disease proneness for each species can be challenging as competence varies by locality (e.g., different mosquito species serve as primary vectors for the West Nile Virus in different countries; Kilpatrick 2011) and disease agent (e.g., deer was regarded as a ''diluting host'' for Lyme disease, but could be the reservoir host for Human Granulocytic Anaplasmosis; Randolph and Dobson 2012). Nevertheless, species that dominate species-poor assemblages often tend to invest less in immune defense and hence have higher competence in transmitting pathogens, and such trade-offs in investment between immunity and life history traits related to both reproduction and competition could be broadly applicable (Huang et al. 2013; Liu et al. 2017; Ostfeld 2017).

Although quantitative data for specific host/vector competence were unavailable for this meta-analysis, when compiling data from studies that focus on specific infectious diseases, we marked the focal host/vector species as specified in the original literature (available for approximately 80% of the studies). Very often, these identified species would be the most significant host/vector for the focal disease in each specific study context, with presumably highest competence (e.g., rodents for hantavirus, *Anopheles* for malaria, and *Ixodes* for Lyme disease). Therefore, despite data limitations, changes in hosts/vector metrics of different known pathogen types calculated in the meta-analysis likely indirectly reveal the responses of some high-competence hosts/vectors. The results are multidirectional: the beneficial effects of land-use changes on known hosts/vectors of macroparasites and pathogenic viruses suggest that habitat disturbance might enhance both direct and vector-borne disease transmission (Gottdenker et al. 2014). However, we also found negative impacts of disturbance on protozoal hosts/vector competence, somewhat contradictory to previous noted increases in protozoan pathogens under human impacts (Gottdenker et al. 2014). This might be because a single species could be the key host/vector for more than one pathogen (e.g., *Culex quinquefasciatus* for Japanese Encephalitis and nematodes; Thongsripong et al. 2013), and we were not able to cover all pathogen types associated with the focal organism in the meta-analysis. Thus, the observed pattern from identified host/vector community changes might not necessarily be the same as the overall effect on pathogens.

Apart from changes in diversity, the increase in host/vector body size in disturbed habitats also suggests rising disease risk based on increased individual competence within each species. In general, body size of hosts can relate

to the provisioning of shelter and food for parasites, and usually larger individuals harbor greater abundance of parasites with better resource quantity and quality, compared with conspecifics, which lead to larger-sized and longer-lived parasites (Poulin and Morand 2004; Nunn and Altizer 2006). And larger-sized parasites like fleas or ticks could further strengthen pathogen transmission by laying more eggs and giving birth to better quality offspring (Van der Mescht et al. 2013). Consequently, the increased body sizes of hosts/vectors in response to the habitat disturbances measured here will likely elevate the disease risks in these disturbed communities.

We found a non-directional response for abundance metrics (which comprised 75.8% of comparisons) and non-directional responses of arthropod hosts/vectors to land-use change (within arthropods, abundance was also the dominant response metric). This could indicate a negligible overall abundance response of hosts/vectors (especially arthropods) to disturbance, and thus could suggest minimal impacts on the transmission of density-dependent diseases (Keesing et al. 2006; Ryder et al. 2007). However, this does not take into account variation in host/vector competence and mobility. The decrease in abundance of certain species might be compensated or caused by the rise of others (Keesing et al. 2010; Thongsripong et al. 2013). Meyer Steiger et al. (2016), for example, found that even though overall mosquito abundance and species richness might be similar between disturbed and undisturbed habitats, the community structure could still vary considerably, especially for important disease vectors. Similarly, the manipulation study by Liu et al. (2016) also emphasized the importance of community composition rather than absolute abundance of hosts on disease risk.

Land-use types were also found to have multidirectional impacts on competence of host/vector communities, possibly as a consequence of the diverse microhabitats created by the various disturbance types (Patz and Olson 2006; Yasuoka and Levins 2007; Pienkowski et al 2017). The beneficial effects of forest fragmentation and agricultural lands suggest that partial disturbance can lead to thriving host/vector communities (Nupp and Swihart 1996; Malcolm 1997; Linard et al. 2007; Gibson et al. 2013; Rubio et al. 2014), probably due to the lack of top predators and competitors, as well as the novel resources that could be exploited by some synanthropic species (Patz et al. 2004; McKinney 2006). The seemingly inhibiting effects of pastures and villages (although confidence intervals overlapped with zero), on the other hand, indicate that even for generalists like those hosts/vectors, sometimes habitat disturbance might be too extreme for their survival (De Luca et al. 2003), or that they are eliminated through disease control and improvement in sanitation following urbanization (Wood et al. 2017). As for the neutral impacts and high within-group variation of most other land-use types, these might be explained by the high heterogeneity of microclimates created in different localities even under the same general disturbance type, as well as the diverse niche preferences by different host/vector species, which lead to highly diverse context-dependent responses (Yasuoka and Levins 2007).

Sampling bias and variation in group composition might be confounding factors that influenced some of the multidirectional results within categories. For example, South America and protozoal hosts/vector data (for which land-use change appeared to reduce disease risk) were rather dominated by comparisons to extreme disturbances like pastures and villages (65 out of 123 for South America and 54 out of 86 for protozoal pathogens), and most body size data (32 out of 35) were from comparisons with forest fragments. The impact of these confounding variables may not be trivial given the limited amount of data available. Another limitation, as pointed out by Gottdenker et al. (2014), is the oversampling of commonly studied diseases such as leishmaniasis and malaria. The dominance of such oversampled pathogens might largely skew trends identified in this meta-analysis (Gottdenker et al. 2014). Although our dataset covered several types of diseases and pathogens, additional studies on a greater variety of diseases in the future would enable a more representative analysis of disease host/vector responses to global land-use changes. Finally, throughout this project, we incorporated little data that directly measured disease transmission rate or pathogen prevalence (due to data scarcity), but instead inferred disease risk based on host/vector community changes in combination with studies linking the ecological metrics with parasite transmission risk (e.g., studies on dilution effects). Therefore, the implications on disease risk that we drew from all comparisons are highly dependent on the generality of those links between community composition and disease risk (e.g., dilution effect). It is also important to note that disease transmission could be affected by many other factors such as disease control efforts and climate change (Dunn et al. 2010; Zargar et al. 2015; Wood et al. 2017). We therefore call for more comparative studies with direct measurement of pathogen prevalence in disturbed

and pristine habitats to better investigate the relationship between land-use changes and infectious disease risk.

Conclusion

We found a general increase in community competence of disease hosts/vectors in response to human land-use changes. Elevation of disease risk could be a result of declining host/vector diversity and increasing body size of individuals in disturbed habitats, although the overall abundance remains unchanged. Other variables including pathogen and land-use type also contributed to the complexity in ecological responses. Overall, we conclude that the response of disease hosts and vectors to human land-use changes is complex, and is affected by multiple variables. Despite the undetermined interactive effects of each variable, this meta-analysis captures the general picture of how land-use changes alter disease host/vector communities in ways that might boost infectious disease emergence.

Acknowledgements

We thank several anonymous reviewers for constructive feedback on the manuscript. Research was supported by the Seed Funding Programme for Basic Research from the University of Hong Kong. We declare no competing interests for this project.

References

Allan B, Keesing F, Ostfeld R (2003) Effect of forest fragmentation on Lyme disease risk. *Conservation Biology* 17:267–272. https://doi.org/10.1046/j.1523-1739.2003.01260.x

Ameneshewa B, Service MW (1996) The relationship between female body size and survival rate of the malaria vector *Anopheles arabiensis* in Ethiopia. *Medical and Veterinary Entomology* 10:170–172. https://doi.org/10.1111/j.1365-2915.1996.tb00724.x

Borenstein M, Hedges LV, Higgins JPT, Rothstein HR (2009) *Introduction to Meta-analysis*, Chichester: Wiley

Civitello DJ, Cohen J, Fatima H, Halstead NT, Liriano J, McMahon TA, Ortega CN, Sauer LE, Sehgal T, Young S, Rohr JR (2015) Biodiversity inhibits parasites: broad evidence for the dilution effect. *Proceedings of the National Academy of Sciences* 112:8667–8671. https://doi.org/10.1073/pnas.1506279112

Cumming G, Guégan J (2006) Food webs and disease: is pathogen diversity limited by vector diversity? *EcoHealth* 3:163–170. https://doi.org/10.1007/s10393-006-0028-6

Davies CE, Johnson AF, Wootton EC, Greenwood SJ, Clark KF, Vogan CL, Rowley AF (2015) Effects of population density and body size on disease ecology of the European lobster in a temperate marine conservation zone. *ICES Journal of Marine Science* 72:i128–i138. https://doi.org/10.1093/icesjms/fsu237

De Luca A, Vasconselos H, Barrett T (2003) Distribution of sandflies (Diptera: Phlebotominae) in forest remnants and adjacent matrix habitats in Brazilian Amazonia. *Brazilian Journal of Biology* 63:401–410. https://doi.org/10.1590/s1519-69842003000300006

Dunn R, Davies T, Harris N, Gavin M (2010) Global drivers of human pathogen richness and prevalence. *Proceedings of the Royal Society of London B: Biological Sciences* 277:2587–2595. https://doi.org/10.1098/rspb.2010.0340

Efron B, Tibshirani R (1991) Statistical data analysis in the computer age. *Science* 253:390–395. https://doi.org/10.1126/science.253.5018.390

Ellis E (2011) Anthropogenic transformation of the terrestrial biosphere. *Philosophical Transactions of the Royal Society of London A: Mathematical, Physical and Engineering Sciences* 369:1010–1035. https://doi.org/10.1098/rsta.2010.0331

Ezenwa V, Godsey M, King R, Guptill S (2006) Avian diversity and West Nile virus: testing associations between biodiversity and infectious disease risk. *Proceedings of the Royal Society of London B: Biological Sciences* 273:109–117. https://doi.org/10.1098/rspb.2005.3284

Foley JA, DeFries R, Asner GP, Barford C, Bonan G, Carpenter SR, Chapin FS, Coe MT, Daily GC, Gibbs HK, Helkowski JH, Holloway T, Howard EA, Kucharik CJ, Monfreda C, Patz JA, Prentice IC, Ramankutty N, Snyder PK (2005) Global consequences of land use. *Science* 309:570–574. https://doi.org/10.1126/science.1111772

Froeschke G, van der Mescht L, McGeoch M, Matthee S (2013) Life history strategy influences parasite responses to habitat fragmentation. *International Journal for Parasitology* 43:1109–1118. https://doi.org/10.1016/j.ijpara.2013.07.003

Gibson L, Lee TM, Koh LP, Brook BW, Gardner TA, Barlow J, Peres CA, Bradshaw CJ, Laurance WF, Lovejoy TE, Sodhi NS (2011) Primary forests are irreplaceable for sustaining tropical biodiversity. *Nature* 478:378–381. https://doi.org/10.1038/nature10425

Gibson L, Lynam AJ, Bradshaw CJ, He F, Bickford DP, Woodruff DS, Bumrungsri S, Laurance WF (2013) Near-complete extinction of native small mammal fauna 25 years after forest fragmentation. *Science* 341:1508–1510. https://doi.org/10.1126/science.1240495

Gottdenker N, Streicker D, Faust C, Carroll C (2014) Anthropogenic land use change and infectious diseases: a review of the evidence. *EcoHealth* 11:619–632. https://doi.org/10.1007/s10393-014-0941-z

Greer AL, Briggs CJ, Collins JP (2008) Testing a key assumption of host-pathogen theory: density and disease transmission. *Oikos* 117:1667–1673. https://doi.org/10.1111/j.1600-0706.2008.16783.x

Gubler DJ (2011) Dengue, urbanization and globalization: the unholy trinity of the 21st century. *Tropical Medicine and Health* 39(4 Suppl):3–11. https://doi.org/10.2149/tmh.2011-S05

Hall SR, Sivars-Becker L, Becker C, Duffy MA, Tessier AJ, Cáceres CE (2007) Eating yourself sick: transmission of disease as a function of foraging ecology. *Ecology Letters* 10:207–218. https://doi.org/10.1111/j.1461-0248.2007.01011.x

Hansen MC, Potapov PV, Moore R, Hancher M, Turubanova S, Tyukavina A, Thau D, Stehman SV, Goetz SJ, Loveland TR, Kommareddy A, Egorov A, Chini L, Justice CO, Townshend

JRG (2013) High-resolution global maps of 21st-century forest cover change. *Science* 342:850–853. https://doi.org/10.1126/science.1244693

Huang ZY, de Boer WF, van Langevelde F, Olson V, Blackburn TM, Prins HH (2013) Species' life-history traits explain interspecific variation in reservoir competence: a possible mechanism underlying the dilution effect. *PLoS ONE* 8:e54341. https://doi.org/10.1371/journal.pone.0054341

Johnson PT, Thieltges DW (2010) Diversity, decoys and the dilution effect: how ecological communities affect disease risk. *Journal of Experimental Biology* 213:961–970. https://doi.org/10.1242/jeb.037721

Johnson PT, Preston DL, Hoverman JT, Richgels K (2013) Biodiversity decreases disease through predicTable changes in host community competence. *Nature* 494:230–233. https://doi.org/10.1038/nature11883

Johnson PT, Preston DL, Hoverman JT, LaFonte BE (2013) Host and parasite diversity jointly control disease risk in complex communities. *Proceedings of the National Academy of Sciences* 110:16916–16921. https://doi.org/10.1073/pnas.1310557110

Johnson PT, Ostfeld RS, Keesing F (2015) Frontiers in research on biodiversity and disease. *Ecology Letters* 18:1119–1133. https://doi.org/10.1111/ele.12479

Keesing F, Holt R, Ostfeld RS (2006) Effects of species diversity on disease risk. *Ecology Letters* 9:485–488. https://doi.org/10.1111/j.1461-0248.2006.00885.x

Keesing F, Belden LK, Daszak P, Dobson A, Harvell CD, Holt RD, Hudson P, Jolles A, Jones KE, Mitchell CE, Myers SS, Bogich T, Ostfeld RS (2010) Impacts of biodiversity on the emergence and transmission of infectious diseases. *Nature* 468:647–652. https://doi.org/10.1038/nature09575

Kilpatrick A (2011) Globalization, land use, and the invasion of West Nile virus. *Science* 334:323–327. https://doi.org/10.1126/science.1201010

Lacroix C, Jolles A, Seabloom EW, Power AG, Mitchell CE, Borer ET (2014) Non-random biodiversity loss underlies predictable increases in viral disease prevalence. *Journal of the Royal Society Interface* 11:20130947. https://doi.org/10.1098/rsif.2013.0947

Linard C, Lamarque P, Heyman P, Ducoffre G, Luyasu V, Tersago K, Vanwambeke S, Lambin E (2007) Determinants of the geographic distribution of Puumala virus and Lyme borreliosis infections in Belgium. *International Journal of Health Geographics* 6:15. https://doi.org/10.1186/1476-072x-6-15

Liu X, Lyu S, Zhou S, Bradshaw CJ (2016) Warming and fertilization alter the dilution effect of host diversity on disease severity. *Ecology* 97:1680–1689. https://doi.org/10.1890/15-1784.1

Liu X, Lyu S, Sun D, Bradshaw CJ, Zhou S (2017) Species decline under nitrogen fertilization increases community-level competence of fungal diseases. *Proceedings of the Royal Society of London B: Biological Sciences* 284:20162621. https://doi.org/10.1098/rspb.2016.2621

LoGiudice K, Ostfeld RS, Schmidt K, Keesing F (2003) The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. *Proceedings of the National Academy of Sciences* 100:567–571. https://doi.org/10.1073/pnas.0233733100

Maher JM, Markey JC, Ebert-May D (2013) The other half of the story: effect size analysis in quantitative research. *CBE-Life Sciences Education* 12:345–351. https://doi.org/10.1187/cbe.13-04-0082

Malcolm JR (1997) Biomass and diversity of small mammals in forest fragments. In: *Tropical Forest Remnants: Ecology, Management, and Conservation of Fragmented Communities*, Laurance WF, Bierregaard RO Jr (editors), Chicago: University of Chicago Press, pp 207–221

McKinney M (2006) Urbanization as a major cause of biotic homogenization. *Biological Conservation* 127:247–260. https://doi.org/10.1016/j.biocon.2005.09.005

Meyer Steiger D, Ritchie S, Laurance S (2016) Mosquito communities and disease risk influenced by land use change and seasonality in the Australian tropics. *Parasites Vectors* 9:387. https://doi.org/10.1186/s13071-016-1675-2

Murray K, Daszak P (2013) Human ecology in pathogenic landscapes: two hypotheses on how land use change drives viral emergence. *Current Opinion in Virology* 3:79–83. https://doi.org/10.1016/j.coviro.2013.01.006

Newbold T, Hudson LN, Hill SL, Contu S, Lysenko I, Senior RA, Börger L, Bennett DJ, Choimes A, Collen B, Day J, et al. (2015) Global effects of land use on local terrestrial biodiversity. *Nature* 520:45–50. https://doi.org/10.1038/nature14324

Nunn C, Altizer S (2006) *Infectious Diseases in Primates*, Oxford: Oxford University Press

Nupp T, Swihart R (1996) Effect of forest patch area on population attributes of white-footed mice (*Peromyscus leucopus*) in fragmented landscapes. *Canadian Journal of Zoology* 74:467–472. https://doi.org/10.1139/z96-054

Ostfeld RS, Keesing F (2000) Biodiversity and disease risk: the case of Lyme disease. *Conservation Biology* 14:722–728. https://doi.org/10.1046/j.1523-1739.2000.99014.x

Ostfeld RS, Keesing F (2000) Biodiversity series: The function of biodiversity in the ecology of vector-borne zoonotic diseases. *Canadian Journal of Zoology* 78:2061–2078. https://doi.org/10.1139/z00-172

Ostfeld RS, Keesing F (2012) Effects of host diversity on infectious disease. *Annual Review of Ecology, Evolution, and Systematics* 43:157–182. https://doi.org/10.1146/annurev-ecolsys-102710-145022

Ostfeld RS (2017) Biodiversity loss and the ecology of infectious disease. *The Lancet Planetary Health* 1:e2–e3

Parker IM, Saunders M, Bontrager M, Weitz AP, Hendricks R, Magarey R, Suiter K, Gilbert GS (2015) Phylogenetic structure and host abundance drive disease pressure in communities. *Nature* 520:542–544. https://doi.org/10.1038/nature14372

Patz JA, Daszak P, Tabor GM, Aguirre AA, Pearl M, Epstein J, Wolfe ND, Kilpatrick AM, Foufopoulos J, Molyneux D, Bradley DJ (2004) Unhealthy landscapes: policy recommendations on land use change and infectious disease emergence. *Environmental Health Perspectives* 112:1092–1098. https://doi.org/10.1289/ehp.6877

Patz J, Olson S (2006) Malaria risk and temperature: influences from global climate change and local land use practices. *Proceedings of the National Academy of Sciences* 103:5635–5636. https://doi.org/10.1073/pnas.0601493103

Patz J, Olson S, Uejio C, Gibbs H (2008) Disease emergence from global climate and land use change. *Medical Clinics of North America* 92:1473–1491. https://doi.org/10.1016/j.mcna.2008.07.007

Pienkowski T, Dickens BL, Sun H, Carrasco LR (2017) Empirical evidence of the public health benefits of tropical forest conservation in Cambodia: a generalised linear mixed-effects model analysis. *The Lancet Planetary Health* 1:e180–e187

Poulin R, Morand S (2004) *Parasite Biodiversity*, Washington, DC: Smithsonian Books

Power A, Flecker A (2008) The role of vector diversity in disease dynamics. In: *Infectious DiseaseEcology: The Effects of Ecosystems on Disease and of Disease on Ecosystems*, Ostfeld RS, Keesing F, Eviner V (editors), Princeton: Princeton University Press

Randolph S, Dobson A (2012) Pangloss revisited: a critique of the dilution effect and the biodiversity-buffers-disease paradigm. *Parasitology* 139:847–863. https://doi.org/10.1017/s0031182012000200

Rubio A, Ávila-Flores R, Suzán G (2014) Responses of small mammals to habitat fragmentation: epidemiological considerations for rodent-borne hantaviruses in the Americas. *EcoHealth* 11:526–533. https://doi.org/10.1007/s10393-014-0944-9

Rottstock T, Joshi J, Kummer V, Fischer M (2014) Higher plant diversity promotes higher diversity of fungal pathogens, while it decreases pathogen infection per plant. *Ecology* 95:907–1917. https://doi.org/10.1890/13-2317.1

Ryder J, Miller M, White A, Knell R, Boots M (2007) Host-parasite population dynamics under combined frequency- and density-dependent transmission. *Oikos* 116:2017–2026. https://doi.org/10.1111/j.2007.0030-1299.15863.x

Swaddle J, Calos S (2008) Increased avian diversity is associated with lower incidence of human West Nile infection: observation of the dilution effect. *PLoS ONE* 3:e2488. https://doi.org/10.1371/journal.pone.0002488

Thongsripong P, Green A, Kittayapong P, Kapan D, Wilcox B, Bennett S (2013) Mosquito vector diversity across habitats in central Thailand endemic for dengue and other arthropod-borne diseases. *PLoS Neglected Tropical Diseases* 7:e2507. https://doi.org/10.1371/journal.pntd.0002507

Van der Mescht L, Le Roux PC, Matthee S (2013) Remnant fragments within an agricultural matrix enhance conditions for a rodent host and its fleas. *Parasitology* 140:368–377. https://doi.org/10.1017/S0031182012001692

Wood CL, Lafferty KD (2013) Biodiversity and disease: a synthesis of ecological perspectives on Lyme disease transmission. *Trends in Ecology & Evolution* 28:239–247. https://doi.org/10.1016/j.tree.2012.10.011

Wood CL, Lafferty KD, DeLeo G, Young H, Hudson P, Kuris A (2014) Does biodiversity protect humans against infectious disease? *Ecology* 95:817–832. https://doi.org/10.1890/13-1041.1

Wood CL, McInturff A, Young HS, Kim D, Lafferty KD (2017) Human infectious disease burdens decrease with urbanization but not with biodiversity. *Philosophical Transactions of the Royal Society B: Biological Sciences* 372:20160122. https://doi.org/10.1098/rstb.2016.0122

Yasuoka J, Levins R (2007) Impact of deforestation and agricultural development on anopheline ecology and malaria epidemiology. *The American Journal of Tropical Medicine and Hygiene* 76:450–460

Young H, Griffin R, Wood C, Nunn C (2013) Does habitat disturbance increase infectious disease risk for primates? *Ecology Letters* 16:656–663. https://doi.org/10.1111/ele.12094

Zargar U, Chishti M, Ahmad F, Rather M (2015) Does alteration in biodiversity really affect disease outcome? – A debate is brewing *Saudi Journal of Biological Sciences* 22:14–18. https://doi.org/10.1016/j.sjbs.2014.05.004