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Biodiversity Loss with Habitat and Risk of New Diseases †

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Abstract: Biodiversity is the number and variety of organisms found within a specified geographic region. It refers to the varieties of plants, animals and microorganisms, the genes they contain and the ecosystems they form. Approximately half of Earth's terrestrial surface is considered to be in a natural or semi natural condition. It relates to the variability among living organisms on the earth, including the variability within and between the species and that within and between the ecosystems. The degradation of nature is among the most serious issues that the world faces, but current targets and consequent actions amount, at best, to a managed decline. Required now are bold and well-defined goals and a credible set of actions to restore the abundance of nature to levels that enable both people and nature to thrive. Human population density strongly correlates with the risk of emergence for all major classes of emerging infectious disease. The maintenance of biodiversity is hypothesized to reduce pathogen prevalence and consequently human disease risk through the dilution effect. However, assuming microbial diversity correlates with that of all other life forms, there may be increased potential for novel pathogens to emerge from biodiverse regions. Here, we present a theoretical framework that exploits the species–area relationship (SAR) to link habitat biodiversity and fragmentation with the exposure to novel infectious diseases by exploiting ecological theory it is possible to identify high-risk areas for risk mitigation and mitigation measures that may simultaneously reduce risk and conserve biodiversity, a problem that has previously been described as both conceptually and practically challenging.

Keywords: biodiversity; microorganisms; degradation; infectious; habitat

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1. Introduction

The biological wealth of the planet has been declining rapidly. Important causes are: 1. Natural causes like floods, earthquakes and other natural disasters. 2. Habitat loss and fragmentation: This is the most important cause driving animals and plants to extinction. The most dramatic examples of habitat loss come from tropical rain forests. Once covering more than 14 per cent of the earth's land surface, these rain forests now cover no more than 6 per cent. Besides total loss, the degradation of many habitats by pollution also threatens the survival of many species. When large habitats are broken up into small fragments due to various human activities, mammals and birds requiring large territories and certain animals with migratory habits are badly affected, leading to decline of population. Habitat loss is caused by deforestation, over-population, pollution, global warming etc. [1]. Human population density strongly correlates with the risk of emergence for all major classes of emerging infectious disease [2]. Zoonotic infections are those among people that come from animal sources and biodiversity has been correlated with emergence of novel zoonotic infectious diseases at the macro-scale [3].

More specifically, the number of mammalian hosts for zoonotic infections increases with species richness among mammals [4,5]. Thus, human encroachment into species-rich habitats may simultaneously decrease biodiversity and increase exposure of people to novel microbes [8–10]. Zoonotic infections are those among people that come from animal

sources and biodiversity has been correlated with emergence of novel zoonotic infectious diseases at the macro-scale [6]. More specifically, the number of mammalian hosts for zoonotic infections increases with species richness among mammals [4,7]. Thus, human encroachment into species-rich habitats may simultaneously decrease biodiversity and increase exposure of people to novel microbes [8–10]. The maintenance of biodiversity is hypothesized to reduce pathogen prevalence and consequently human disease risk through the dilution effect [8]. However, assuming microbial diversity correlates with that of all other life forms, there may be increased potential for novel pathogens to emerge from biodiverse regions. Recent advances have linked anthropogenic land conversion to multi-host models for pathogen transmission between species in intact and degraded habitats [9], quantifying the changing infection risk across altered landscapes for multi-host pathogens. Given these results and that current disease control policies focus on rapid response to outbreaks [10]; models that link biodiversity with habitat structures and novel pathogen emergence are lacking. Here, we present a theoretical framework that exploits the species–area relationship (SAR) to link habitat biodiversity and fragmentation with the exposure to novel infectious diseases.

2. Experiments

2.1. Fragmentation

Habitat fragmentation can be considered as a product of non uniform habitat loss along habitat boundaries, meaning that habitat loss and fragmentation are concurrent in the majority of non-experimental scenarios [11]. Precisely altered experimental landscapes are required to disentangle these innately linked mech anism and their consequences on biodiversity [1], and usage of the terms 'fragmentation' and 'edge effects' often lack precision due to the inherently linked nature of habitat distributions, shapes and areas. Figure 1. Represents various effect of fragmentation.

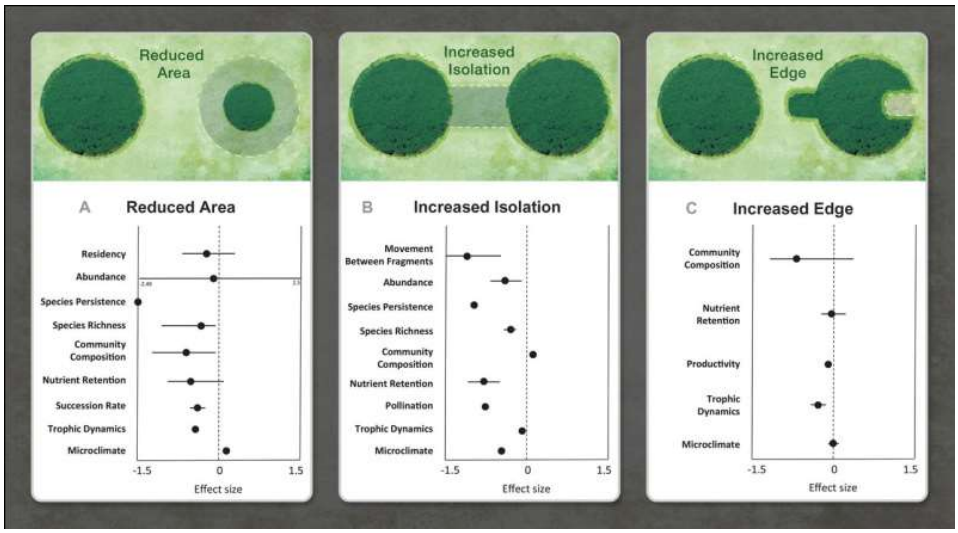

Figure 1. Habitate fragmenatation effect (**A**) Reduced area (**B**) Increased area (**C**) Increased edge [1].

2.2. Model of Biodiversity

Increasing contact through habitat encroachment and fragmentatio and the corresponding species diversity decline are likely to act antagonistically to affect hazards from novel pathogens. To explore this interaction, we first link the SAR to microbial diversity. The SAR predicts that at the landscape level the number of species, S, contained within similarly classed habitat fragments (or patches, i) scales with patch area, A. The exact SAR formulation is debated, here we use the power-law relationship [12] which has been extensively demonstrated for vertebrates, invert ebrates,

plants [13] and importantly microbial communities. Thus, the formulation of the SAR used states that the number of patch-associated species (Si) scales with patch area (Ai) following a power law with magnitude c and rate of decline z. $Si = cA^{z}{}_{i}$.

2.3. Emergence Model with Risk Regarding Infectious Disease

To model the risk (R) of infection emergence from the natural habitat into the expanding human population, we defined R as the product of the relative number of potential disease-causing agents within that habitat, which we assume to scale linearly with fragment biodiversity (Si), and the area over which the expanding human population comes into uniform contact with this habitat, which we assume is represented by the perimeter of the habitat (Pi). Initially, we assumed that infectious disease causing agents within the natural habitat correlate directly with all habitat biodiversity, i.e., that the total hazard from novel pathogens was proportional to patch biodiversity. Thus the risk for each patch i is: Ri = SiPi. To investigate division effects we compared k identically shaped and scaled fragments for differing SAR values of z while allowing total area to vary [14].

2.4. Encroachment Model

For complex pattern of fragmentation effect of encroachment stratagies with various parimeters and division effects we simulated the erosion of two-dimensional habitats to generate landscapes containing different numbers, shapes and sizes of patch to model the potential impact of fragmentation on risk in complex landscapes. We modelled the encroachment of an expanding population into a closed, homo generous habitat [15]. In simulations, the number of species (Si) contained within patch areas (Ai) was expressed between 0 and 1, and are a relative measure where 1 corresponds to the total number of species (S_0) contained within the natural habitat at time zero ($A_0 = 10^6$). So that equation can be expressed as $S_i\ cA^{z}{}_{i} = S_0$.

2.5. Cartographic Estimation of Risk Associated with African Tropical Forest Encroachment

To see how our model may have real-world applications we applied it to forest fragmentation in African tropical forest. The African forests were chosen because important global infectious diseases, such as EVD, falciparum malaria, HIV and Zika virus have emerged from them, and because they are highly diverse with relatively defined boundaries. A binary mask of forested areas was generated by selecting the following GlobCover classes; closed to open (greater than 15%) broadleaved evergreen and/or semi-deciduous forest (greater than 5 m), closed (greater than 40%) broadleaved deciduous forest (greater than 5 m), closed (greater than 40%) needle-leaved evergreen forest (greater than 5 m), closed to open (greater than 15%) mixed broadleaved and needle-leaved forest (greater than 5 m), closed (greater than 40%) broadleaved forest [16].

2.6. Pandemic Risk Associated with African Tropical Forest Encroachment

The pandemic potential of such agents following initial cross species transmission events (spillover) depends on subsequent human to human transmission and thus will be driven by human density) and connectivity. Understanding this for novel agents will help inform surveillance programmes. Therefore, we modelled potential spread and pandemic risk using network theory by converting the pixel grid into a network [17].To estimate the potential for pandemics froman emerging disease, such as EVD, we modelled disease spread between 3 km pixels across Africa.We assumed the potential for spread between adjacent pixels a and b was proportional to the product of the population densities, so that pandemics were likely to travel along paths of high population density. To estimate the relative chance of a source pixel image x resulting in spread to a destination pixel image y, we converted the pixel image to a network using 4-connectivity, with pixels representing nodes and edge weights between adjacent pixels a and b given by $d(a,b)\ 1/p_a p_b t$.

2.7. Ebola Virus Disease Modelling

We tested the predictive capacity of our cartographic model for an infectious disease system from African forests where index case data were available, namely EVD emergence. This system is not an ideal model for our system which aims to model the risk from pathogen diversity, however, there are several Ebola virus species and ebolaviruses have been linked to numerous host species, including bats and primates, and linked to high biodiversity areas and forest fragmentation [18].

3. Results

We assessed the impact of division effects by modelling k iden tically shaped and scaled fragments for differing SAR values of z while allowing total area to vary (Figure 2).When biodiversity is held constant eRIDE increases with k, for $z < 0.5$. Similarly, when eRIDE is held constant biodiversity necessarily decreases with k for $z < 0.5$ (electronic supplementary material, Figure S1). The point at which biodiversity and eRIDE are the same between k and 1 fragments tends towards $z < 0.5$ as k increases ¼ (electronic supplementary material, data S1; Figure 2). Analyses of invertebrate, plant, vertebrate and microbial systems suggest z does not reach 0.5 (electronic supplementary material, table S1). Thus, we predict that increased habitat division will result in increased relative RIDE for all biologically relevant scenarios where contact occurs at the habitat edge. We observed that increasing the dilution effect increased the extent to which division influenced eRIDE (Figure 2a). Strong correlation identified solidity, the ratio of the habitat's area to the area of its convex hull, as a good summary statistic for the proportion of core habitat in these simple habitat shapes (Figure 2c). In this context solidity as a measure of shape com plexity has the additional benefit of being a scale-independent.

Variable with no units. Area, and thus solidity, is independent of de Broglie's frequency and only affected byamplitude, while perimeter (and thus eRIDE) depends on both amplitude and frequency. Solidity demonstrated a strong negative correlation with eRIDE (Figure 2d). Predicted relationships between eRIDE and the four different classes of fragmentation effect (Figure 1) are summarized in Table 1.Our encroachment models generated heterogeneous landscape scenarios with large variance in metrics for all fragmentation classes. The eRIDE metric was seen to work best as a predictor of EVD emergence over smaller spatial scales; a 10–12-fold increase in eRIDE was observed in areas within 5 km, compared to a seven- to eightfold increase in areas within 5–60 km of known EVD outbreak cases.

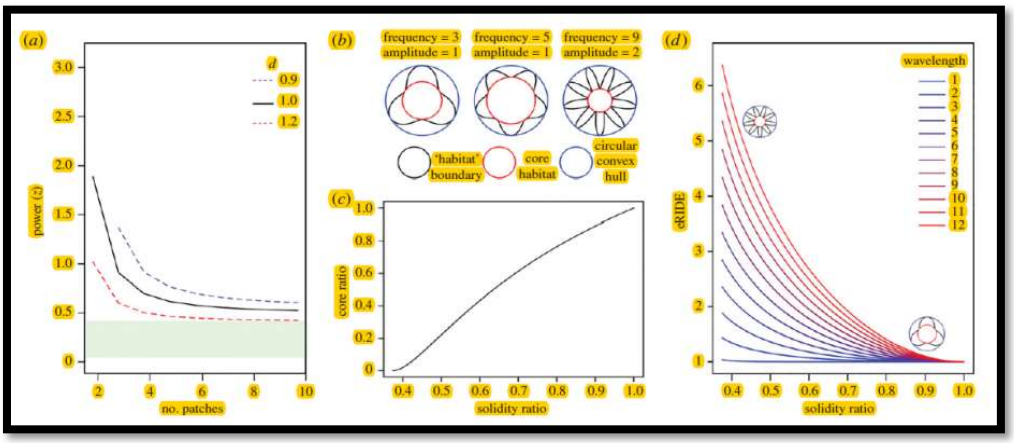

Figure 2. Linking fragmentation effect with (**a**) Patches (**b**) Habitat (**c**) with core ratio (**d**) eride.

4. Discussion

By using the well-characterised species–area relationship from ecology, we have developed a model framework allowing us to predict that how people encroach into natural habitats determines how they experience the risk of novel infectious disease emergence. Overall, our model suggests that there is an argument for maintaining biodiversity and reducing encroachment for the benefit of human health through reduction in the emergence of novel infectious agents. This is in support of another recent analysis which employed a different strategy to suggest that the risk of pathogen spillover is highest at intermediatelevels of habitat loss [19]. Our model considers the total hazard across all habitat associated pathogens. We believe that this general framework may negate some of the need for single system modelling [20], as it may be applicable for all pathogens from macro- to micro parasites. Such an approach may inform land-use strategies inscenarios where little or no biological data are available and is therefore also pertinent regarding the emergence of 'Disease X' [30]. Index ranking of various country represented in Figure 3.

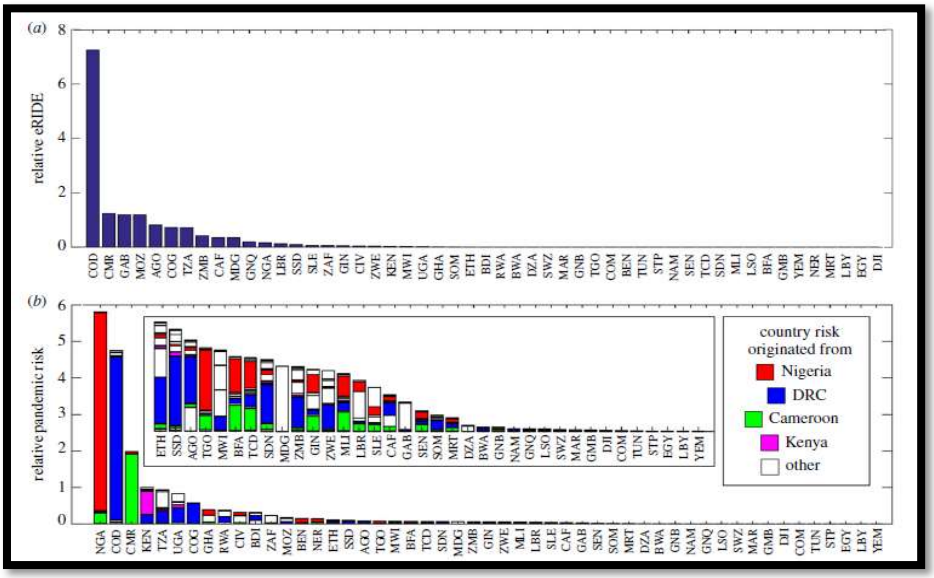

Figure 3. Index rankings per country. Numbers displayed are the sums of all (**a**) eRIDE and (**b**) pandemic risk index pixel values that fall within the country boundaries.

Our model assumes contact occurs at the edge of habitats and thus is more applicable to forest-like systems. The generalizability of our results and the inter action between fragmentation and eRIDE is likely robust until some contact diffusion threshold is reached, and thus if systems such as grasslands have different contact patterns we predict alternative scenarios may be more likely. In particular talking about production of antimicrobial drugs from microbes many scientists had done work on actinomycetes microbes [23–25]. These scenarios and measures of SAR-critical parameters such as c and nestedness require future detailed modeling and field studies. Future models of eRIDE integrating human movement data that account for internal and regional movements, along with seasonal variations, and across other regions will be valuable efforts [21,22].

5. Conclusions

In summary, our general model can be directly applied to establish optimal land-use strategies or to identify strategic sites for disease surveillance (e.g., Figure 6a). As human populations continue to expand into habitats, we propose that such general frameworks are essential for policymakers because they provide clear guiding principles that enable

common ground to be established between species conservation and novel disease emergence risk mitigation. The vast scale of the estimated pandemic-associated risk across Africa with respect to the emergence of EVD in Western Africa in 2014 clearly demonstrates that internationally coordinated efforts are required to avoid catastrophic events in the future. However, our model suggests that the implementation of smaller-scale land-use strategies linked with conservation efforts may help to improve the overall burden fromemerging infectious disease.

Author Contributions: DR read and approved the manuscript for publication. DR contributed to methodology, conceptualization, investigation, methodology, resources, literature review, and formal analysis of study data, draft, and edited the manuscript for intellectual content.

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Conflicts of Interest: Nil.

Abbreviations

The following abbreviations are used in this manuscript:

SAR	Species–area relationship
HIV	Human immunodeficiency virus

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