



Article scientifique

Article

2025

Published version

Open Access

This is the published version of the publication, made available in accordance with the publisher's policy.

Leveraging Small Biodiversity Reserves to Prevent Zoonotic Disease: Insights from Dilution Effect and Pathogen Adaptation Theories

Arnal, Audrey; Gozlan, Rodolphe Elie; Charbonnel, Nathalie; Bouilloud, Marie; Chaves, Andrea; Gauthier-Clerc, Michel; Viguera-Galván, Ana L.; Arnathau, Céline; Roiz, David; Bento, Ana I.; Morand, Serge; Walzer, Chris; Suzán, Gerardo; Sarmiento Silva, Rosa Elena [and 1 more]

How to cite

ARNAL, Audrey et al. Leveraging Small Biodiversity Reserves to Prevent Zoonotic Disease: Insights from Dilution Effect and Pathogen Adaptation Theories. In: Disease biology, genetics, and socioecology, 2025, p. 6. doi: 10.53941/dbgs.2025.100006

This publication URL: <https://archive-ouverte.unige.ch/unige:184924>

Publication DOI: [10.53941/dbgs.2025.100006](https://doi.org/10.53941/dbgs.2025.100006)



Communication

Leveraging Small Biodiversity Reserves to Prevent Zoonotic Disease: Insights from Dilution Effect and Pathogen Adaptation Theories

Audrey Arnal^{1,2,3}, Rodolphe Elie Gozlan⁴, Nathalie Charbonnel⁵, Marie Bouilloud^{1,5}, Andrea Chaves^{6,7}, Manon Lounnas^{1,2}, Michel Gauthier-Clerc⁸, Ana L. Viguera-Galván^{2,3}, Céline Arnathau^{1,2}, David Roiz^{1,2}, Ana I. Bento⁹, Serge Morand^{10,11,12}, Chris Walzer^{13,14}, Gerardo Suzán^{2,15}, Rosa Elena Sarmiento Silva^{2,3,*}† and Benjamin Roche^{1,2,3,†}

¹ MIVEGEC, Université de Montpellier, IRD, CNRS, 34394 Montpellier, France

² International Joint Laboratory IRD/UNAM ELDORADO, Merida 97000, Mexico

³ Departamento de Microbiología e Inmunología, Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de México (UNAM), Ciudad de México 04510, Mexico

⁴ ISEM, University of Montpellier, CNRS, IRD, 34090 Montpellier, France

⁵ CBGP, INRAE, CIRAD, IRD, Institut Agro, Université de Montpellier, 34398 Montpellier, France

⁶ Centro Nacional de Innovaciones Biotecnológicas (CENIBiot), CeNAT, Conare, San José 1174-1200, Costa Rica

⁷ Escuela de Biología, Universidad de Costa Rica, San José 11501-206, Costa Rica

⁸ Faculté des Sciences, Université de Genève, 30 Quai Ernest-Ansermet, CH-1211 Geneve, Switzerland

⁹ Department of Public and Ecosystem Health, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853, USA

¹⁰ IRL Health DEEP, CNRS, Kasetsart University, Mahidol University Bangkok 10900, Thailand

¹¹ Faculty of Veterinary Technology, Kasetsart University, Bangkok 10900, Thailand

¹² Department of Social and Environmental Medicine, Faculty of Tropical Medicine, Mahidol University, Bangkok 10400, Thailand

¹³ Wildlife Conservation Society Southern Boulevard, Bronx, NY 10460, USA

¹⁴ Research Institute of Wildlife Ecology, University of Veterinary Medicine, 1210 Vienna, Austria

¹⁵ Departamento de Etología, Fauna Silvestre y Animales de Laboratorio, Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autónoma de México (UNAM), Ciudad de México 04360, Mexico

* Correspondence: rosass@unam.mx; Tel.: +52-554-449-7749

† Co-last authors.

How To Cite: Arnal, A.; Gozlan, R.E.; Charbonnel, N.; et al. Leveraging Small Biodiversity Reserves to Prevent Zoonotic Disease: Insights from Dilution Effect and Pathogen Adaptation Theories. *Disease Biology, Genetics, and Socioecology* **2025**, *1*(2), 6. <https://doi.org/10.53941/dbgs.2025.100006>.

Received: 22 February 2025

Revised: 12 March 2025

Accepted: 12 March 2025

Published: 2 April 2025

Abstract: In today's landscape of zoonotic pathogen outbreaks, the dilution effect theory, i.e., the theory that greater biodiversity can help curb pathogen transmission among wildlife, has gained significant attention. However, the positive link between animal diversity and pathogen richness urges us to apply this concept with caution. It is crucial to explore how conservation biology can safeguard human health by preventing the emergence of zoonotic diseases. By investigating the implications of conservation strategies on animal communities and pathogen transmission as well as the adaptive capabilities of pathogens, we propose that biodiversity conservation based on small reserves can effectively reduce pathogen spread in wildlife, provided certain measurable conditions are met. Given the urgent need to tackle both zoonoses disease emergence and biodiversity loss, these interventions should be prioritized and implemented without delay.

Keywords: conservation; biodiversity; dilution effect; zoonoses; prevention; one health



Copyright: © 2025 by the authors. This is an open access article under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

Publisher's Note: Scilight stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.

1. Introduction

The urgency to reverse the loss of biodiversity [1] has increased societal interest in environmental conservation, pushing it onto political agendas. However, many decision-makers remain hesitant to implement sustainable conservation programs [2]. The COVID-19 pandemic, driven by SARS-CoV-2, has highlighted the global threat of zoonotic diseases, which jump from animals to humans, causing significant health, social, and economic disruptions. No longer just a topic for debate, the emergence of zoonoses is now clearly linked to human-induced changes in our natural environments [3].

While geopolitical factors and financial resources often dominate discussions on international policies, short-term economic costs tend to overshadow the long-term impacts of these decisions. A key barrier is the lack of demonstrated synergies among crises that would maximize investment. To tackle this, we need a comprehensive cost-effectiveness analysis of how different habitat conservation strategies can mitigate disease emergence, encouraging authorities to engage more actively. Since the groundbreaking research by Ostfeld and Keesing (2000), numerous studies have highlighted the potential prophylactic effects of biodiversity conservation on infectious disease transmission [4]. This is evidenced by a frequent negative correlation between species richness and pathogen transmission [5–7]. However, the so-called ‘dilution effect’ is often non-linear and predicting the impact of biodiversity protection on zoonosis emergence in humans remains complex [8]. Thus, leveraging conservation biology tools to reduce the risk of emerging infectious diseases [9] demands a deeper understanding of how the dilution effect applies across various ecosystems [10–12] and processes, such as those involved in biodiversity loss [13]. Most studies focus on transmission dynamics within ecosystems, particularly whether altered pathogens are zoonotic [13,14]. However, pathogen circulation among animals is just the initial step toward spillover, overlooking critical animal-human interactions that biodiversity protection could influence. To safeguard human health, we must examine both the biological and sociological links between biodiversity loss and human exposure to zoonotic diseases, including direct human-animal interactions.

Recent studies linking conservation strategies and infectious diseases highlight a significant dependence on local contexts and the specific pathogens involved [15]. It is important to point out here that we use the term “pathogen” to represent any kind of parasitic microbe. Obviously, the host spectrum and its adaptive potential (which are key components for a potential dilution effect) will be extremely different according to the kind of microbes considered (e.g., viruses, bacteria, helminths, etc.). This variability of contexts and community assemblages complicates efforts to identify universally applicable win-win solutions for environmental protection and public health. Unlike research on the dilution effect, these studies often prioritize human disease burden [16]. Since human disease burden depends on pathogen circulation, socio-economic factors, and pathogen adaptation (whether partial or complete), this perspective falls short in explaining how biodiversity conservation reduces spillover risks [17]. While the general applicability of the dilution effect has garnered considerable support [6,8,12,13,18], it is evident that policies rooted in this effect cannot be universally applied across all diseases and contexts. A deeper mechanistic understanding is essential. Additionally, pathogens play a pivotal role in maintaining both vertebrate and invertebrate biodiversity—aligning with the Janzen-Connell hypothesis [19,20], which underscores the need for a multi-pathogen approach to this issue. In this light, it becomes crucial to identify the specific conditions and settings where broad-scale policies can effectively impact local ecosystems.

This study explores how conservation biology can reduce pathogen transmission among wildlife, ultimately lowering spillover risks to humans. Instead of focusing on direct human transmission, which is influenced by socio-economic and behavioral factors beyond this study, we concentrate on enzootic pathogen circulation, the early stage of potential epidemics. Our investigation examines global conservation strategies and their impact on wildlife pathogen transmission, pinpointing critical knowledge gaps for designing strategies with dual benefits for biodiversity and public health. By assessing the risks and rewards of each approach, we identify strategies that offer the safest public health co-benefits while minimizing zoonotic risk.

2. Potential Impacts of Landscape Conservation Strategies on Pathogen Transmission—The Role of Pathogen Adaptation and Habitat Connectivity

Since early research on the dilution effect [21,22], using conservation biology to reduce the risk of emerging infections has been considered. However, our understanding of the dilution effect, its mechanisms, and applicability was then underdeveloped. Since then, knowledge in conservation biology has significantly grown, highlighting the need to merge these fields.

Many reviews have outlined the conditions under which a dilution effect can be observed—such as significant variability in host competence, horizontal transmission, a link between host abundance and competence, and frequency-dependent transmission. Similarly, conservation strategies have been extensively explored, from

identifying key species to target, determining the optimal size of protected areas, and ensuring connectivity between habitats patches [23]. Rather than delving into an exhaustive review of this literature here, more detailed discussions are available in the supplementary materials. What remains clear is the pressing need to align these advances in conservation and disease ecology, paving the way for strategies that not only protect biodiversity but also mitigate the risk of pathogen spread.

In this study, we focus on the potential effects of concrete conservation strategies on host communities and, consequently, the expected circulation of pathogens within ecosystems (results are summarized in Table 1). To do so, we consider the balance between hazard (pathogen diversity, defined by the number of pathogen species, as a potential source of harm) and risk (actual exposure to a given pathogen through circulation), as described by Hosseini et al. (2017) [24]. Our focus is specifically on landscape selection strategies, particularly the debate over whether a single large reserve or several small reserves (i.e., the SLOSS debate) is more effective. It is important to note that we center our analysis on pathogens with minimal virulence in their wild hosts. We define here “virulence” as the infection cost for the host, for which a “minimal virulence” has a negligible impact on host abundance. As such, they do not significantly disrupt hosts community diversity or assembly—so as to avoid introducing complex host-pathogen dynamics. Additionally, we have chosen to maintain a broad perspective rather than focusing on a single pathogen to keep our findings widely applicable (for specific examples of how conservation strategies affect pathogen transmission, see Lambert et al., 2020) [25].

Table 1. Summary of different conservation strategies, their impact on animal communities, and their potential impact on pathogen transmission. We assume the conditions necessary for a dilution effect: (1) horizontally transmitted pathogen (i.e., no vertical transmission), and (2) animal communities with a high probability of extinction for low abundance species. As shown in Figure 1, ecosystem A assumes a perfect positive correlation between competence and species abundance (a perfect context for a dilution effect). Conversely, in ecosystem B we assume a perfect negative correlation between competence and species abundance (a perfect context for an amplification effect).

Conservation Strategies		Consequences on Animal Communities	Ref	Consequences on Pathogens Communities on the Ecosystem A	Consequences on Pathogens Communities on the Ecosystem B
<i>Several Small Reserves</i> Maximize regional diversity by combining small patches with several different species		Many patches, inter-connected, with low species richness in each	[26,27]	Rapid pathogen adaptation; high pathogen transmission within each reserve, which may lead to different pathogen adaptation within each patch (local speciation) Slow pathogen adaptation; hot and cold spots of transmission and adaptation Strong genetic drift effects may limit adaptation if interconnectivity between patches is strong	
<i>Intermediate Strategy</i> Maximize the time to population extinction		Few patches with a reasonably high species richness	[28,29]	Medium level of transmission. Determining the ideal patch size could be considered by looking at the pathogen communities	
<i>Single Large Reserves</i> Larger areas contain more species than smaller areas (species-area relationship theory and equilibrium theory of island biogeography) Decrease the probability of species extinction	Classic Reserves	One patch with high species richness	[30,31]	More pathogens species but less transmission (dilution effect)	More pathogens species and more transmission (amplification effect)
	Biodiversity Hotspots	One patch with high species richness	[32,33]	More pathogens species but less transmission (dilution effect)	More pathogens species and more transmission (amplification effect)
	Key Biodiversity Areas (KBAs)	Case-by-case	[34,35]	Host communities being heterogeneous between KBAs, it is difficult to extrapolate for pathogen communities	

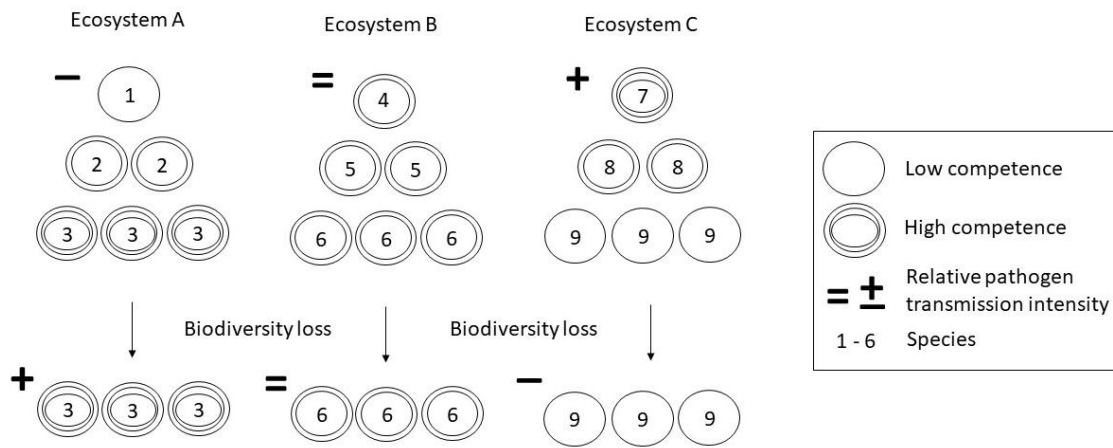


Figure 1. Examples of dilution and amplification effect. The competence (number of circles) and abundance (number of individuals per species) within animal communities, and their influence on pathogen transmission. Both ecosystems show a perfect positive (Ecosystem A), null (Ecosystem B) and negative (Ecosystem C) correlation between competence and abundance of each species (species 1 to 9). A dilution effect is expected in Ecosystem A, an amplification effect in ecosystem C while no impact on pathogen transmission is expected on Ecosystem B.

In a landscape-scale conservation strategy using multiple small reserves, two critical factors come into play: the adaptive capacity of pathogens and the connectivity between patches [36,37]. It is worth pointing out that pathogen adaptation is always challenging to forecast and can take many different forms. Nevertheless, the likelihood of pathogen adaptation (i.e., increased transmission in this case) is linked to its adaptation potential, which is the quantity that could be directly measured (e.g., pathogen mutation rate, pathogen substitution rate, etc.). When inter-patch connectivity is low, patch sizes are reasonable, and pathogens adapt quickly to their environment, this approach can foster local pathogens adaptation. In other words, each patch would host its own strain (i.e., a genotype), leading to low pathogen diversity at the patch level but high diversity across the entire region. Pathogen transmission would likely be high within individual patches, but low between them [38,39]. While the pathogen hazard remains high due to its wide geographical distribution, the risk of widespread transmission would be more contained.

On the other hand, if a pathogen adapts more slowly to its environment, we would expect a mosaic of ‘hot’ and ‘cold’ spots of adaptation [40]. In hotspots—where pathogens are well adapted—transmission would be high. But in cold spots, where environmental changes outpace pathogen adaptation, transmission would be low, possibly even leading to local pathogen extinction. Compared to fast adapting pathogens, this scenario would see a decrease in overall transmission and pathogen diversity at the landscape level. As a result, both the hazard and the risk of the remaining pathogens would likely be lower.

These impacts can be significantly influenced by increasing patch connectivity. When patches are fully connected, the dynamics resemble those of a single large population, where high pathogen adaptation and transmission are favored—though predicting exact outcomes becomes more challenging (see Table 1). On the other hand, with low or intermediate connectivity, pathogens may struggle to adapt due to conflicting pressures between local and regional environments [38]. This tension limits a pathogen’s ability to thrive in both contexts, potentially resulting in a global reduction in both hazard and risk. However, if connectivity becomes too strong, it can create a complex mosaic of adaptive responses, especially under high genetic drift, making patterns of pathogen adaptation difficult to predict.

To leverage this type of conservation strategy for reducing pathogen transmission, it is essential to strike the right balance—determining the ideal patch size and connectivity level to maintain this local-regional adaptation conflict. Doing so could help minimize transmission levels [22] and offer a clear benefit in terms of the hazard-risk trade-off. Such a threshold, when identified, can become a key tool to develop win-win strategies between biodiversity protection and human health.

This approach contrasts with the design of traditional large reserves, which are often based on specific conservation needs, such as protecting biodiversity hotspots or preventing area-sensitive species loss. While large reserves may offer ecological benefits, they often result in high species and pathogen richness, making their impact on pathogen circulation harder to predict. These areas might target Key Biodiversity Areas (KBAs), but due to the diversity of host communities within and between KBAs, the effects on pathogen dynamics remain uncertain. Some conservation strategies focus on specific species, like keystone species, to maintain high species richness.

In these cases, the outcome for pathogen transmission depends heavily on the makeup of surrounding animal communities—particularly the ratio of competent versus non-competent species in the ecosystem. Similarly, focusing on flagship species may boost conservation efforts but have little impact on pathogen dynamics unless that species plays a pivotal ecological role. Translocation strategies are also unlikely to affect pathogen transmission unless they significantly alter the structure of animal communities. In summary, developing large reserves to mitigate zoonotic risks may produce highly variable results, making it a less reliable option for protecting human health from zoonoses.

3. Discussion and Perspectives

In this analysis, we explored the potential impact of conservation strategies on the enzootic circulation of pathogens, which is the critical first step before pathogens spillover into human populations. We highlighted that conservation strategies can have a broad range of effects on pathogen transmission—some positive, some negative. Notably, while some of these impacts may be anticipated, others remain unpredictable, raising concerns about the safety of their implementation. From our current understanding, establishing several small reserves with moderate interconnection between patches seems to offer the most reliable outcome. This approach hinges on the adaptive capacity of pathogens, a factor that can be measured, making it a more manageable strategy. Today, our knowledge is not sufficient to model quantitatively and provide forecast on the impact of conservation biology on the reduction of pathogen circulation. Therefore, it's essential to quantify both host species' competence, its evolution and patch connectivity to fine-tune the optimal reserve size, striking a balance between species richness and low pathogen transmission. Conceptually, a win-win scenario appears achievable with this strategy of several small reserves.

Our findings present a more optimistic perspective compared to earlier work, yet align with studies suggesting that the dilution effect often arises in the context of biodiversity loss [13]. The difference likely stems from the metrics we employed. Our focus was on enzootic circulation of pathogens, whereas the link between enzootic transmission, human exposure, and subsequent human-to-human spread involve distinct processes that require separate, careful consideration. While our study addresses a crucial gap by assessing how specific conservation strategies may affect pathogen transmission within animal communities, it does not provide direct recommendations for public health policy. However, we believe that fully understanding the mechanisms at play in each stage of pathogen transmission—from wildlife to humans—will be key to developing successful, win-win strategies that benefit both biodiversity and human health.

Indeed, the success of conservation approaches in supporting public health hinges on carefully balancing the risks, benefits, and hazards they introduce [24]. Achieving an ecosystem with high biodiversity, which also implies greater pathogens diversity, but low pathogen transmission—the ultimate goal—requires delicate balance. While this approach reduces risk (by preventing pathogen amplification), it increases hazard (more pathogens within the ecosystem [24]). Managing these hazards effectively calls for a better characterization of the interface between enzootic circulation and human exposure. It is also important to recognize potential tensions between the most ecologically beneficial conservation strategies and those aimed at reducing pathogen transmission. Identifying and navigating these trade-offs is essential to designing sustainable, locally tailored solutions that engage all stakeholders.

Conservation strategies are not solely about maximizing species richness; they may prioritize preserving genetic diversity or species' evolutionary potential [41]. In addition, social or ethical objectives often influence these strategies, such as reintroducing iconic species or eradicating invasive ones [42]. Socio-economic factors, like indigenous land rights or sustainable food production, also play a crucial role [43]. Reducing pathogen transmission is not without trade-offs either, as pathogens play a crucial role in shaping biodiversity [44] and habitat quality [45]. Therefore, efforts to limit pathogens should focus on those with zoonotic potential. Meanwhile, certain conservation strategies, like managing habitat matrices or establishing ecological corridors, are too context-dependent to predict their broader impact on pathogen transmission accurately. Strategies aimed at promoting the dilution effect or mitigating pathogen amplification could offer pathways to reducing pathogen transmission. Targeting species that contribute to dilution could help manage zoonotic outbreaks [46]. Ecological traits also matter: fast-living species, which often serve as disease reservoirs [47] tend to thrive in degraded landscapes, potentially increasing their number and elevating transmission risks. Developing surveillance systems in areas where human-wildlife interactions are frequent, like urban parks, could provide crucial insights into zoonotic pathogens transmission [18].

In conclusion, exploring 'win-win-win' strategies benefiting public health, biodiversity, and the economy is both feasible and essential. Mathematical models exist to test these ideas, and further research based on local data is needed to understand their effectiveness in diverse environments. While more data is required for safe

application of large reserves, our study demonstrates the potential of conservation strategies based on small reserves to limit pathogen circulation and reduce human exposure safely. However, caution is necessary, as pathogen transmission effects can be complex. Now more than ever, integrating conservation with public health strategies is crucial.

Funding

This research has not received any specific grant from any public, commercial, or nonprofit funding agency.

Institutional Review Board Statement

Not applicable.

Data Availability Statement

We advocate for the sharing of research data by all authors contributing to publications in Scilight journals. In this section, authors may be asked to provide the raw data of their study together with the manuscript for editorial review and should be prepared to make the data publicly available if practicable. In any event, authors should ensure accessibility of such data to other competent professionals for at least 10 years after publication (preferably via an institutional or subject-based data repository or other data center), provided that the confidentiality of the participants can be protected and legal rights concerning proprietary data do not preclude their release. In instances where novel data were not generated or data remains inaccessible due to privacy or ethical considerations, a clear statement outlining these circumstances is mandatory.

Acknowledgments

We thank the French National Research Institute for Sustainable Development (IRD) for funding the International Joint Laboratory IRD/UNAM Merida ELDORADO. We also thank the Consejo Nacional de Humanidades Ciencia y Tecnología (CONAHCYT). We also thank the Consejo Nacional de Humanidades, Ciencia y Tecnología (CONAHCYT) for the postdoctoral fellowship awarded to ALVG with the number 2331314 CONV-2022 (1), 2022 (2) & 2022 (3).

Conflicts of Interest

There is no conflict of interest.

References

1. Ripple, W.J.; Wolf, C.; Newsome, T.M.; et al. World Scientists' Warning to Humanity: A Second Notice. *Bioscience* **2017**, *67*, 1026–1028. <https://doi.org/10.1093/biosci/bix125>.
2. Robinson, J.G. Conservation Biology and Real-World Conservation. *Conserv. Biol.* **2006**, *20*, 658–669. <https://doi.org/10.1111/j.1523-1739.2006.00469.x>.
3. Daszak, P.; Amuasi, J.; das Neves, C.G.; et al. *IPBES (2020) Workshop Report on Biodiversity and Pandemics of the Intergovernmental Platform on Biodiversity and Ecosystem Services*; IPBES Secretariat: Bonn, Germany, 2020. <https://doi.org/10.5281/zenodo.4147317>.
4. Ostfeld, R.S.; Keesing, F. Biodiversity and Disease Risk: The Case of Lyme Disease. *Conserv. Biol.* **2000**, *14*, 722–728. <https://doi.org/10.1046/j.1523-1739.2000.99014.x>.
5. Johnson, P.T.J.; Preston, D.L.; Hoverman, J.T.; et al. Biodiversity Decreases Disease through Predictable Changes in Host Community Competence. *Nature* **2013**, *494*, 230–233. <https://doi.org/10.1038/nature11883>.
6. Keesing, F.; Ostfeld, R.S. Impacts of Biodiversity and Biodiversity Loss on Zoonotic Diseases. *Proc. Natl. Acad. Sci. USA* **2021**, *118*, e2023540118. <https://doi.org/10.1073/pnas.2023540118>.
7. Ostfeld, R.S.; Keesing, F. Effects of Host Diversity on Infectious Disease. *Annu. Rev. Ecol. Evol. Syst.* **2012**, *43*, 157–182. <https://doi.org/10.1146/annurev-ecolsys-102710-145022>.
8. Halliday, F.W.; Rohr, J.R. Measuring the Shape of the Biodiversity–Disease Relationship across Systems Reveals New Findings and Key Gaps. *Nat. Commun.* **2019**, *10*, 5032. <https://doi.org/10.1038/s41467-019-13049-w>.
9. Khalil, H.; Ecke, F.; Evander, M.; et al. Declining Ecosystem Health and the Dilution Effect. *Sci. Rep.* **2016**, *6*, 31314. <https://doi.org/10.1038/srep31314>.
10. Ferraguti, M.; Martínez-De la Puente, J.; Jiménez-Clavero, M.Á.; et al. A Field Test of the Dilution Effect Hypothesis in Four Avian Multi-Host Pathogens. *PLoS Pathog.* **2021**, *17*, e1009637. <https://doi.org/10.1371/journal.ppat.1009637>.
11. Wood, C.L.; Lafferty, K.D.; DeLeo, G.; et al. Does Biodiversity Protect Humans against Infectious Disease? *Ecology* **2014**, *95*, 817–832. <https://doi.org/10.1890/13-1041.1>.

12. Civitello, D.J.; Cohen, J.; Fatima, H.; et al. Biodiversity Inhibits Parasites: Broad Evidence for the Dilution Effect. *Proc. Natl. Acad. Sci. USA* **2015**, *112*, 8667–8671. <https://doi.org/10.1073/pnas.1506279112>.
13. Halliday, F.W.; Rohr, J.R.; Laine, A. Biodiversity Loss Underlies the Dilution Effect of Biodiversity. *Ecol. Lett.* **2020**, *23*, 1611–1622. <https://doi.org/10.1111/ele.13590>.
14. Mahon, M.B.; Sack, A.; Aleuy, O.A.; et al. A Meta-Analysis on Global Change Drivers and the Risk of Infectious Disease. *Nature* **2024**, *629*, 830–836.
15. Young, H.S.; Wood, C.L.; Marm Kilpatrick, A.; et al. Conservation, Biodiversity and Infectious Disease: Scientific Evidence and Policy Implications. *Philos. Trans. R. Soc. B Biol. Sci.* **2017**, *372*, 20160124.
16. Albers, H.J.; Lee, K.D.; Rushlow, J.R.; et al. Disease Risk from Human–Environment Interactions: Environment and Development Economics for Joint Conservation–Health Policy. *Environ. Resour. Econ.* **2020**, *76*, 929–944. <https://doi.org/10.1007/s10640-020-00449-6>.
17. Hopkins, S.R.; Lafferty, K.D.; Wood, C.L.; et al. Evidence Gaps and Diversity among Potential Win–Win Solutions for Conservation and Human Infectious Disease Control. *Lancet Planet Health* **2022**, *6*, e694–e705.
18. Gibb, R.; Redding, D.W.; Chin, K.Q.; et al. Zoonotic Host Diversity Increases in Human-Dominated Ecosystems. *Nature* **2020**, *584*, 398–402. <https://doi.org/10.1038/s41586-020-2562-8>.
19. Connell, J. On the Role of Natural Enemies in Preventing Competitive Exclusion in Some Marine Animals and in Rain Forest Trees. *Dyn. Popul.* **1971**, *298*, 312.
20. Janzen, D.H. Herbivores and the Number of Tree Species in Tropical Forests. *Am. Nat.* **1970**, *104*, 501–528. <https://doi.org/10.1086/282687>.
21. Allan, B.F.; Keesing, F.; Ostfeld, R.S. Effect of Forest Fragmentation on Lyme Disease Risk. *Conserv. Biol.* **2003**, *17*, 267–272. <https://doi.org/10.1046/j.1523-1739.2003.01260.x>.
22. Kilpatrick, A.M.; Salkeld, D.J.; Titcomb, G.; et al. Conservation of Biodiversity as a Strategy for Improving Human Health and Well-Being. *Philos. Trans. R. Soc. B Biol. Sci.* **2017**, *372*, 20160131. <https://doi.org/10.1098/rstb.2016.0131>.
23. Simberloff, D.; Abele, L.G. Refuge Design and Island Biogeographic Theory: Effects of Fragmentation. *Am. Nat.* **1982**, *120*, 41–50. <https://doi.org/10.1086/283968>.
24. Hosseini, P.R.; Mills, J.N.; Prieur-Richard, A.H.; et al. Does the Impact of Biodiversity Differ between Emerging and Endemic Pathogens? The Need to Separate the Concepts of Hazard and Risk. *Philos. Trans. R. Soc. B Biol. Sci.* **2017**, *372*, 20160129. <https://doi.org/10.1098/rstb.2016.0129>.
25. Lambert, S.; Gilot-Fromont, E.; Toigo, C.; et al. An Individual-Based Model to Assess the Spatial and Individual Heterogeneity of *Brucella Melitensis* Transmission in Alpine Ibex. *Ecol. Modell.* **2020**, *425*, 109009. <https://doi.org/10.1016/j.ecolmodel.2020.109009>.
26. Boecklen, W.J. Nestedness, Biogeographic Theory, and the Design of Nature Reserves. *Oecologia* **1997**, *112*, 123–142. <https://doi.org/10.1007/s004420050292>.
27. Skaggs, R.W.; Boecklen, W.J. Extinctions of Montane Mammals Reconsidered: Putting a Global-Warming Scenario on Ice. *Biodivers. Conserv.* **1996**, *5*, 759–778. <https://doi.org/10.1007/BF00051785>.
28. Etienne, R.S.; Heesterbeek, J.A.P. On Optimal Size and Number of Reserves for Metapopulation Persistence. *J. Theor. Biol.* **2000**, *203*, 33–50. <https://doi.org/10.1006/jtbi.1999.1060>.
29. Ovaskainen, O. Long-Term Persistence of Species and the SLOSS Problem. *J. Theor. Biol.* **2002**, *218*, 419–433. [https://doi.org/10.1016/S0022-5193\(02\)93089-4](https://doi.org/10.1016/S0022-5193(02)93089-4).
30. Connor, E.F.; McCoy, E.D. The Statistics and Biology of the Species–Area Relationship. *Am. Nat.* **1979**, *113*, 791–833. <https://doi.org/10.1086/283438>.
31. Diamond, J.M. The island dilemma: Lessons of modern biogeographic studies for the design of natural reserves. *Biol. Conserv.* **1975**, *7*, 129–146. [https://doi.org/10.1016/0006-3207\(75\)90052-X](https://doi.org/10.1016/0006-3207(75)90052-X).
32. Mittermeier, R.A.; Gil, P.G.; Hoffman, M.; et al. *Hotspots Revisited. Earth’s Biologically Richest and Most Endangered Terrestrial Ecoregions*; Cemex: San Pedro Garza Garcia, Mexico, 2004.
33. Mittermeier, R.A.; Turner, W.R.; Larsen, F.W.; et al. Global Biodiversity Conservation: The Critical Role of Hotspots. In *Biodiversity Hotspot*; Springer: Berlin/Heidelberg, Germany, 2011; pp. 3–22. ISBN 978-3-642-20991-8.
34. Eken, G.; Bennun, L.; Brooks, T.M.; et al. Key Biodiversity Areas as Site Conservation Targets. *Bioscience* **2004**, *54*, 1110–1118. [https://doi.org/10.1641/0006-3568\(2004\)054\[1110:kbaasc\]2.0.co;2](https://doi.org/10.1641/0006-3568(2004)054[1110:kbaasc]2.0.co;2).
35. Langhammer, P.F.; Bakarr, M.I.; Bennun, L.A.; et al. *Identification and Gap Analysis of Key Biodiversity Areas: Targets for Comprehensive Protected Area Systems*; IUCN: Gland, Switzerland, 2007.
36. VanAcker, M.C.; Little, E.A.H.; Molaei, G.; et al. Enhancement of Risk for Lyme Disease by Landscape Connectivity, New York, New York, USA. *Emerg. Infect. Dis.* **2019**, *25*, 1136. <https://doi.org/10.3201/eid2506.181741>.
37. McCallum, H.; Dobson, A. Disease, Habitat Fragmentation and Conservation. *Proc. R. Soc. London. Ser. B Biol. Sci.* **2002**, *269*, 2041–2049. <https://doi.org/10.1098/rspb.2002.2079>.

38. Gandon, S.; Capowiez, Y.; Dubois, Y.; et al. Local Adaptation and Gene-for-Gene Coevolution in a Metapopulation Model. *Proc. R. Soc. B Biol. Sci.* **1996**, *263*, 1003–1009. <https://doi.org/10.1098/rspb.1996.0148>.
39. Rubio, A.V.; Ávila-Flores, R.; Suzán, G. Responses of Small Mammals to Habitat Fragmentation: Epidemiological Considerations for Rodent-Borne Hantaviruses in the Americas. *Ecohealth* **2014**, *11*, 526–533. <https://doi.org/10.1007/s10393-014-0944-9>.
40. Thompson, J.N. Specific Hypotheses on the Geographic Mosaic of Coevolution. *Am. Nat.* **1999**, *153*, S1–S14. <https://doi.org/10.1086/303208>.
41. Laikre, L. Genetic Diversity Is Overlooked in International Conservation Policy Implementation. *Conserv. Genet.* **2010**, *11*, 349–354. <https://doi.org/10.1007/s10592-009-0037-4>.
42. Courchamp, F.; Angulo, E.; Rivalan, P.; et al. Rarity Value and Species Extinction: The Anthropogenic Allee Effect. *PLoS Biol.* **2006**, *4*, e415. <https://doi.org/10.1371/journal.pbio.0040415>.
43. Fischer, J.; Abson, D.J.; Butsic, V.; et al. Land Sparing versus Land Sharing: Moving Forward. *Conserv. Lett.* **2014**, *7*, 149–157. <https://doi.org/10.1111/conl.12084>.
44. Hudson, P.J.; Dobson, A.P.; Lafferty, K.D. Is a Healthy Ecosystem One That Is Rich in Parasites? *Trends Ecol. Evol.* **2006**, *21*, 381–385.
45. Koltz, A.M.; Civitello, D.J.; Becker, D.J.; et al. Sublethal Effects of Parasitism on Ruminants Can Have Cascading Consequences for Ecosystems. *Proc. Natl. Acad. Sci. USA* **2022**, *119*, e2117381119. <https://doi.org/10.1073/pnas.2117381119>.
46. Keesing, F.; Ostfeld, R.S. Dilution Effects in Disease Ecology. *Ecol. Lett.* **2021**, *24*, 2490–2505. <https://doi.org/10.1111/ele.13875>.
47. Estavillo, C.; Weyland, F.; Herrera, L. Zoonotic Disease Risk and Life-History Traits: Are Reservoirs Fast Life Species? *Ecohealth* **2022**, *19*, 390–401. <https://doi.org/10.1007/s10393-022-01608-5>.