

## Global biogeographic regions in a human-dominated world: the case of human diseases

MICHAEL G. JUST,<sup>1,†</sup> JACOB F. NORTON,<sup>2</sup> AMANDA L. TRAUD,<sup>2</sup> TIM ANTONELLI,<sup>2</sup> AARON S. POTEATE,<sup>3,6</sup>  
GREGORY A. BACKUS,<sup>2</sup> ANDREW SNYDER-BEATTIE,<sup>2</sup> R. WYATT SANDERS,<sup>1</sup> AND ROBERT R. DUNN<sup>4,5</sup>

<sup>1</sup>*Department of Plant and Microbial Biology, North Carolina State University, Raleigh, North Carolina 27695 USA*

<sup>2</sup>*Department of Mathematics and Biomathematics Graduate Program, North Carolina State University,  
Raleigh, North Carolina 27695 USA*

<sup>3</sup>*Department of Sociology and Anthropology, North Carolina State University, Raleigh, North Carolina 27695 USA*

<sup>4</sup>*Department of Biological Sciences and Keck Center for Behavioral Biology, North Carolina State University,  
Raleigh, North Carolina 27695*

<sup>5</sup>*Center for Macroecology, Evolution, and Climate, Natural History Museum of Denmark, University of Copenhagen,  
2100 Copenhagen Ø, Denmark*

**Citation:** Just, M. G., J. F. Norton, A. L. Traud, T. Antonelli, A. S. Poteate, G. A. Backus, A. Snyder-Beattie, R. W. Sanders, and R. R. Dunn. 2014. Global biogeographic regions in a human-dominated world: the case of human diseases. *Ecosphere* 5(11):143. <http://dx.doi.org/10.1890/ES14-00201.1>

**Abstract.** Since the work of Alfred Russel Wallace, biologists have sought to divide the world into biogeographic regions that reflect the history of continents and evolution. These divisions not only guide conservation efforts, but are also the fundamental reference point for understanding the distribution of life. However, the biogeography of human-associated species—such as pathogens, crops, or even house guests—has been largely ignored or discounted. As pathogens have the potential for direct consequences on the lives of humans, domestic animals, and wildlife it is prudent to examine their potential biogeographic history. Furthermore, if distinct regions exist for human-associated pathogens, it would provide possible connections between human wellbeing and pathogen distributions, and, more generally, humans and the deep evolutionary history of the natural world. We tested for the presence of biogeographic regions for diseases of humans due to pathogens using country-level disease composition data and compared the regions for vectored and non-vectored diseases. We found discrete biogeographic regions for diseases, with a stronger influence of biogeography on vectored than non-vectored diseases. We also found significant correlations between these biogeographic regions and environmental or socio-political factors. While some biogeographic regions reflected those already documented for birds or mammals, others reflected colonial history. From the perspective of diseases caused by pathogens, humans have altered but not evaded the influence of ancient biogeography. This work is the necessary first step in examining the biogeographic relationship between humans and their associates.

**Key words:** colonial; human associates; pathogens.

**Received** 4 July 2014; revised 24 September 2014; accepted 26 September 2014; **published** 21 November 2014.  
Corresponding Editor: T. J. Davies.

**Copyright:** © 2014 Just et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited. <http://creativecommons.org/licenses/by/3.0/>

<sup>6</sup> Present address: Department of Anthropology, University of Oregon, Eugene, Oregon 97403 USA.

† **E-mail:** [mjust@ncsu.edu](mailto:mjust@ncsu.edu)

### INTRODUCTION

Biogeographic regions have been delineated

for many animal and plant taxa and are a cornerstone of large spatial scale biology. However, human associated species—such as pests,

domesticates, and pathogens—tend to be excluded from consideration when discerning biogeographic regions. These human associates are often expected to be ubiquitous and, on average, do indeed have larger geographic ranges than most other species (Dunn and Romdal 2005, Olden et al. 2006, Smith et al. 2007). Even relatively large human associates, such as rats and house flies, spread around the world with early western colonization (West 1951, He et al. 2009), just as West Nile virus and avian influenza H5N1 more recently spread (Centers for Disease Control and Prevention 2003, Spielman et al. 2004, Fauci 2005, Olsen et al. 2006). Delineation of biogeographic regions for taxa that are truly everywhere, such as what appears to be the case for aquatic protists (Fenchel and Finlay 2004), would not be particularly useful. It's possible that the easy and repeated spread of human associates might blur any interesting or useful biogeographic pattern in the composition of human associates. However, importantly, not all human associates have spread to all inhabited continents (Gonçalves et al. 2003), and some human-associated species, particularly those that live outdoors for part of their life cycles, are directly influenced by the presence of vectors, alternate hosts, climate or other environmental conditions (Wilcox and Colwell 2005), such that it is possible that historic and climatic influences might still lead to discrete biogeographic regions for these species.

While scientific curiosity and conservation have motivated most studies of biogeographic regions (Wallace 1876, Grenyer et al. 2006, Holt et al. 2013), the biogeography of human associates in general and pathogens in particular has the potential for direct human consequences. Others have suggested that diversity and prevalence of human pathogen species affect human politics, the likelihood of war (Thornhill et al. 2009), and religion (Fincher and Thornhill 2008) among other aspects of socio-politics (Nettle 2009), such that the biogeographic distribution of pathogens has the potential to pervasively affect human life and societies. If the limits of pathogen distributions are determined not only by climate and attempts at disease control but also the ancient biogeographic distribution of vectors and hosts (e.g., Stensgaard et al. 2013), the differences among regions in their pathogens (and conse-

quently religions, behaviors, and socio-politics) may be relatively persistent features of those regions.

To the extent that biogeographic regions for human pathogens exist, they might be expected to differ as a function of the biology of the pathogens. A priori it seems likely that vectored and non-vectored pathogens might differ in their biogeographic regions. Vectored pathogens require at least the presence of a suitable vector and, in some cases, a reservoir host. Non-vectored pathogens, on the other hand, many of which evolved relatively recently (Wolfe et al. 2007), can be transmitted either person to person or via contaminated water such that their global spread might be less likely to be influenced by environmental differences among regions of the world. We could have divided pathogens into other groupings, but we chose to focus on vectored/non-vectored taxa in as much as it allowed us to test a priori predictions (e.g., Smith and Guégan 2010), rely on relatively robust categorizations of pathogens (whether or not a pathogen is vectored tends to be reasonably well-known), and leads to groups with sample sizes sufficient to justify the development of biogeographic regions. If biogeographic regions exist for either grouping or overall, we hypothesize they might be due to differences in climate, biogeographic history (e.g., the movement of the continents, the chance dispersal of host lineages, etc.) or human history and culture.

Here we test whether human pathogens are distributed globally in distinct biogeographic regions. We first consider 301 human diseases caused by pathogens from the Global Infectious Diseases and Epidemiology Online Network (GIDEON; <http://gideononline.com>) database and then consider diseases caused by vectored ( $n = 93$ ) and non-vectored pathogens ( $n = 208$ ) separately. GIDEON defines vectors as the agent in which a pathogen is transmitted from one host to another. The GIDEON disease data are described in detail in Smith et al. (2007). They are not complete (diseases, particularly rare ones, can be missed), but they are, to our knowledge, the most complete disease data available. As with any global dataset, it is likely the data are poorer in regions with less well developed public health systems such that pathogens unique to those regions might be missed. This includes

both rare diseases that have not yet been detected in particular regions and emerging pathogens that have yet to be detected anywhere. As in other studies of the biogeography of non-human associates we use a hierarchical clustering algorithm (e.g., Wang et al. 2003, Xie et al. 2004, Oliver and Irwin 2008), specifically, Ward's hierarchical agglomerative method (Ward 1963), to evaluate if biogeographic regions exist. We then test the robustness of these biogeographic regions using a complementary and more computationally intensive approach: community detection (Lancichinetti and Fortunato 2009). Finally, once biogeographic regions were identified, we compared, individually, the environmental and socio-political variables associated with those regions for both vectored and non-vectored diseases caused by pathogens.

## MATERIALS AND METHODS

### *Data collection*

*Human disease data.*—Data on the presence and absence of 301 diseases caused by pathogens (Appendix: Table A1) of humans in 229 countries (Appendix: Table A2) from GIDEON were the basis for the majority of our analyses. GIDEON also provided basic data on the life history of the pathogens that cause each disease, particularly whether or not each is vector borne. We excluded GIDEON entries from our analyses that were not recorded in the database as currently present in any country, were not associated with a pathogen, or were difficult to assign as vector- or non-vector-borne. We used the broader pathogen literature to guide our classification decisions such that we were able to identify vectors for *Mycobacterium ulcerans* as vector-borne (Marsollier et al. 2002), but none of the other *Mycobacteria* were classified as vector-borne. GIDEON's data have now been used in a number of papers on the biogeography of disease (Møller et al. 2009, Thornhill et al. 2009, Yang et al. 2012, Hay et al. 2013) and represent the highest quality data available at the global scale, except those for particular pathogens (e.g., malaria [Moyes et al. 2013]).

*Environmental and socio-political variables data.*—To compare different biogeographic regions, we focused on environmental variables known to be associated with the distribution of individual

pathogens. Toward this end, we extracted data on the minimum temperature, maximum temperature, daily precipitation frequency, annual precipitation volume, total precipitation volume in the month with the minimum amount of precipitation, and total precipitation volume in the month with the maximum amount of precipitation for each country from the Tyndall Center for Climate Change Research at the University of East Anglia (Mitchell et al. 2002). Each of these variables has been suggested to influence the distributions of at least some pathogens (Guernier et al. 2004, Jones et al. 2008, Bonds et al. 2012) or their vectors (Lafferty 2009). In addition, we used derived estimates of human population density (persons per square kilometer in 2010), gross domestic product (2012 US dollars), and land area (square kilometer) data per country from the Population Division of United Nations (United Nations 2013) and supplemented as needed from the CIA World Factbook (<http://www.cia.gov>). Human population density has the potential to influence the persistence of pathogens, but it has been suggested that there is also an association between the diversity of human pathogens and the diversity of birds and mammals, with areas with higher diversity of birds and mammals tending to have more diseases caused by pathogens (Dunn et al. 2010). For our analyses we chose to use only native mammal species richness data since global bird, mammal, and plant richness are highly correlated (Qian and Ricklefs 2008), and our qualitative result should be similar regardless of which of these variables we analyze. Mammal diversity estimates were based on the International Union for Conservation of Nature's native mammal species richness data (IUCN 2013).

### *Statistical analysis*

Using political boundaries as our unit of analysis, we demarcated biogeographic regions based on the composition of diseases caused by pathogens. We employed hierarchical clustering to identify potential biogeographic regions from three different schemas of the pathogen composition by country data: (1) full suite of diseases, (2) vector-borne diseases, and (3) non-vector-borne diseases. We then used a second statistical procedure, community detection, to validate our

findings. Ideally, one might consider fine grain data on human diseases, or data on individual taxa of pathogens (e.g., distinguishing the distribution patterns of strains of *Bartonella* rather than simply the presence of Bartonellosis) but such data exist only for a minority of pathogens. Amazingly, we remain more ignorant about the distribution of human pathogen taxa than we do about rare birds.

All of our analyses were performed in R 2.15.1 (R Core Team 2013) except where otherwise noted. Biogeographic regions were identified using Ward's agglomerative hierarchical clustering method (R function: `hclust`), as Ward's method is a preferred method in many biogeographic studies (Kreft and Jetz 2010). Regions were created by clustering countries based on their composition of diseases caused by pathogens (i.e., presence and absence). The major benefit to using Ward's method is that the algorithm joins groups while minimizing within-cluster variance. However, agglomerative hierarchical methods produce results (in the form of a dendrogram) without clear indication of the optimal number of biogeographic regions ( $k$ ). Therefore, we used a Mantel-based algorithm (Borcard et al. 2011) to determine  $k$  for each schema (i.e., vector-borne pathogens, non-vector-borne pathogens, and the full suite of 180 pathogens; R package: `cluster`; function: `daisy` [Maechler et al. 2013]). This method simply aims to maximize the correlation between the original (unclustered) distance matrix and the distance matrices computed by cutting the dendrogram at various levels (Borcard et al. 2011).

We validated the biogeographic regions found by hierarchical methods using community detection. In particular, we used a modularity maximization algorithm, Fast Unfolding (Blondel et al. 2008), to divide the network of countries into biogeographic regions, modularity being the fraction of connections that occur within regions minus the fraction expected given a particular network. In the network countries were connected if they shared the presence of a disease, and that connection was weighted by how many diseases they had in common. To compare our two sets of regions (i.e., clustering, community detection) we calculated a Rand similarity coefficient (Rand 1971). We assessed the strength of the match through comparison of the Rand

similarity coefficient to a post hoc distribution for the Rand similarity coefficient with randomization tests and then calculated the  $p$ -value directly from this distribution.

After determining the optimal number of biogeographic regions, we used multinomial and binomial models (R package: `nnet`) to assess how environmental and socioeconomic factors correlate with these biogeographic regions (Venables and Ripley 2002). Multinomial logistic regressions predict placement in a category, here biogeographic region, based on multiple independent variables, here environmental (number of native mammal species, maximum temperature, minimum temperature, temperature difference, precipitation frequency, total annual precipitation) and socio-political (GDP, population density) To corroborate the multinomial models, each schema and covariate combination was evaluated in isolation using a series of binomial logistic regressions in MATLAB.

## RESULTS

Using Ward's clustering algorithm and a Mantel optimality procedure, we determined the following number of biogeographic regions for each schema: vectored diseases ( $n = 7$ ), non-vectored diseases ( $n = 5$ ) and the full suite of 301 diseases ( $n = 2$  [Appendix: Table A2]). We discerned seven biogeographic regions when considering vectored diseases (Fig. 1A; Appendix: Fig. A1), but only five regions when considering just non-vectored diseases (Fig. 1B; Appendix: Fig. A3), implying that the former have more biogeographic structure. In addition, the differences among biogeographic regions for vectored diseases were much greater than those for non-vectored diseases (Appendix: Figs. A1, A2). The differences between the most distinct biogeographic regions for non-vectored diseases were akin to those found within the biogeographic regions of vectored diseases (Appendix: Figs. A1, A2). Though community detection methods discerned similar biogeographic regions, the exact number of regions was different for the two subset schemas. For example, three regions were found when the vector-borne diseases were considered using community detection, four less than found using Ward's clustering. However, the regions found using



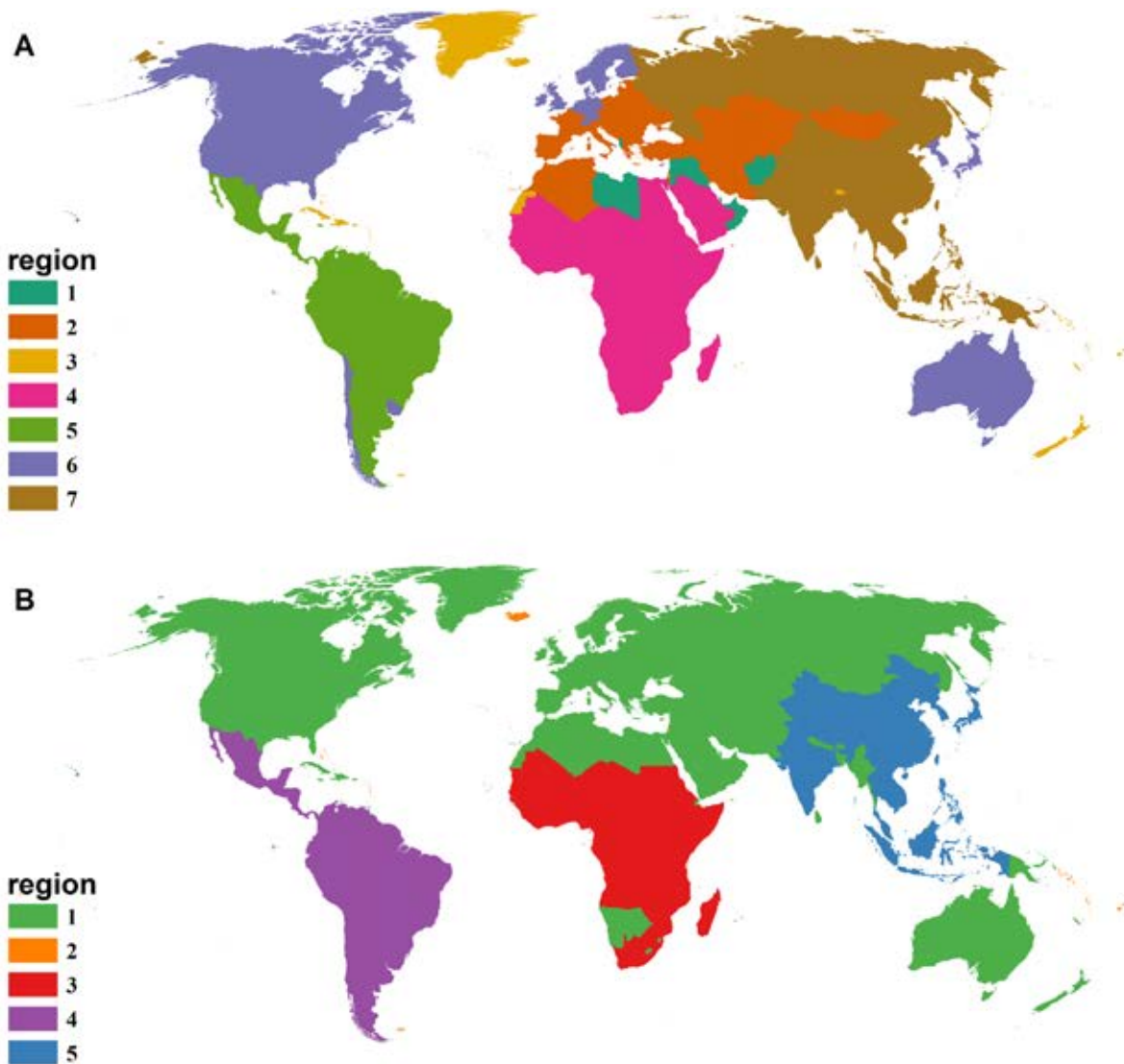


Fig. 1. (A) Map of biogeographic regions ( $n = 7$ ) delineated from vector-borne ( $n = 93$ ) human-associated diseases based on presence by country ( $n = 229$ ). (B) Regions ( $n = 5$ ) for non-vector-borne diseases ( $n = 208$ ). Regions were created by hierarchical clustering using Ward's distance.

community detection match the first branches in Ward's clustering (Appendix: Figs. A1, A2) and the regions detected using the two approaches were much more similar between the methods than expected by chance ( $p < 0.001$ ) on the basis of Rand similarity coefficients.

Vector-borne biogeographic regions were significantly correlated with climate, biodiversity, and social variables (Table 1). Non-vector-borne regions were correlated with all variables except minimum temperature (Table 1). Biogeographic re-

gions from the full suite of pathogens were correlated with native mammal species richness and GDP (Table 1).

## DISCUSSION

We have shown that biogeographic regions exist for human diseases caused by pathogens. Although those species that live on our bodies, in our homes, or in our backyards are among those with the most direct effects on our health and

Table 1. Z-statistic (full suite) values for each binomial regression and chi-squared ( $\chi^2$ ) values for each multinomial regression between each schema's biogeographic regions and individual environmental or socio-political variables.

Variable	Full suite	Vector	Non-vector
Native mammal species richness	-0.032*	152.49***	106.22***
Gross domestic product	$2.6 \times 10^{-5}$ ***	100.56***	44.70***
Population density	0.001	74.55***	23.44***
Maximum temperature	-0.051	67.85***	25.27***
Minimum temperature	-0.134	81.92***	9.23
Temperature difference	...	73.45***	56.40***
Precipitation frequency	$8.9 \times 10^{-4}$	55.20***	32.45***
Total annual precipitation	0.002	89.85***	50.56***
Minimum precipitation month	14.5	66.27***	14.07**
Maximum precipitation month	-0.017	70.53***	11.21*

Notes: The missing value for temperature difference for the full suite data schema is expected due to two interacting properties of the analysis: the optimal number of biogeographic regions ( $n = 2$ ) and the variable, temperature difference, is a linear combination of two other variables, maximum and minimum temperature.

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ .

well-being, the distribution of these organisms remains poorly understood, perhaps in part because they are assumed to live everywhere we do. For diseases and the pathogens that cause them this assumption is wrong. The biogeographic regions for diseases caused by pathogens are robust to the statistical approaches used and are as distinct as the biogeographic regions of, for example, vertebrates, or plants. In other words, not only do biogeographic regions exist for diseases and disease causing pathogens (and likely other human associates), they are comparable in their delineation to the other established biogeographic regions, regions typically described as existing due to biogeographic history before the actions and movement of humans.

The biogeographic regions for vectored diseases coincide in many respects with those recently derived for non-human vertebrates (Holt et al. 2013). Where it exists, the coincidence of these regions suggests that the same historic factors that influence the composition of wild birds, mammals, and amphibians also influence (whether directly or indirectly) which diseases are present in any particular region. While previous work has suggested strong links between climate and the diversity of pathogens or diseases globally (Guernier et al. 2004, Bonds et al. 2012), here we suggest something different: namely that, in addition to climate, history and geography have strong effects on which pathogens are where. That the impacts of history and geography are nearly as strong on diseases and their pathogens as on organisms such as mam-

mals, which are superficially less mobile between regions, is both novel and somewhat surprising. The biogeographic regions of birds, mammals and other animals are the result of the geographic position of landmasses, plate tectonics, and chance dispersal events (e.g., Holt et al. 2013). Ultimately, these same factors must also play a role in the distribution of the pathogens that cause diseases whether directly or via their effects on alternate hosts and vectors. As a result, the precise mix of vectored diseases in any particular place is the result of not only climate, human migration, and attempts at disease control, but also millions of years of tectonics and chance dispersal (or failure to disperse). So long as our attempts to control vectored pathogens are incomplete, human health and well-being, culture, and even political stability are likely to continue to be influenced by this ancient history.

However, the biogeographic regions for vectored diseases did depart from those of vertebrates in several interesting ways. For example, one of the biogeographic regions clearly defined for vectored diseases, Region 6, (Fig. 1) included portions of the Holarctic, but also included historically British colonized countries, such as Australia. From the perspective of vectored diseases, Australia is part of the same biogeographic region as England even though Australia is one of the most unique historical biogeographic regions in terms of birds or mammals (Wallace 1876, Holt et al. 2013). Similarly, India and Bangladesh, despite being climatically and his-

torically very different from any Holarctic region are part of a biogeographic region (Region 7) that is most similar to Region 6 (the region containing Britain [Appendix: Fig. A1]). Clearly, human history can and has altered the biogeographic regions of diseases, even if it has not fully obscured those ancient regions. The history of colonization from other regions also seems to influence the biogeographic regions, albeit somewhat less clearly. The composition of diseases in Italy, for example, was similar to that in its former colonies (e.g., Eritrea, Ethiopia, Somalia, etc.). The relationships between colonies and former colonies and states are doubtless very complex—the consequence of the layered influence of colonial movement, socio-politics and disease biology—and yet the existence of pathogen biogeographic regions that correspond to colonial histories is striking. The ancient biogeography of vectored pathogens can be altered and in as much as the Holarctic (including British) pathogen composition is a relatively benign one, altered to the ends of human benefit.

We also found some outwardly unusual pairings of countries. For example, vectored Region 1, with member countries including Afghanistan, Malta, Monaco, Qatar, and others, seems unusual upon initial review as the climate, history, and biogeography of these nations within these regions is diverse. However, similarity indices, such as those used in our hierarchical clustering, tend to unite sites with low diversity (Boyle et al. 1990) and these countries were in the lowest 15% of vectored disease richness as is the case both in countries of small size, where sampling is likely to be incomplete or where, as in Qatar, climate is generally ill conducive to life. In other words, these regions share the attribute of hosting a relatively low diversity of recorded, vectored diseases.

Non-vectored diseases were clustered into fewer biogeographic regions than were vectored diseases regardless of our statistical approach and those regions were far less different from one another than were the biogeographic regions for vectored diseases. One of those regions was composed of the temperate Palearctic and Nearctic (together, Holarctic) regions of the world. The other four were largely comprised, respectively, of the countries that were islands (Region 2), large proportions of sub-Saharan Africa

(Region 3), Central and South America (Region 4), and Southeast Asia (Region 5). The links between colonial territories and states is dampened in the non-vectored regions compared to the vectored regions.

Like the pathogen subset schemas (vectored and non-vectored), the full suite of diseases (Appendix: Figs. A3, A4) is distributed into statistically significant biogeographic regions, but the nature of the regions is different from the subsets. We found just two biogeographic regions when considering the full set of diseases and these regions broadly reflected the division between Holarctic and Australia and the rest of the world. At the coarsest grain, in other words, the world can be divided, from the perspective of diseases, into just two pieces with many consequences, all of those associated with diseases, following from this division.

Biogeographic regions of vectored and non-vectored diseases were associated with both climatic and socio-political variables, suggesting that in addition to the influence of history, climate and socio-politics have influenced the distribution of diseases. Though much is understood about the individual natural histories of vectors (Qiu et al. 2002, Foley et al. 2007), less is known about general trends of the histories of the vectored diseases. Given that the biogeographic regions of vectored disease are associated with climate variables, it seems likely that the precise boundaries of these regions will shift as climate changes, as has been suggested to be the case for individual pathogens (Pascual and Bouma 2009), though just how they will shift is likely to be difficult to predict given that such shifts will represent the cumulative effect of the movement of hundreds of pathogen species, their vectors, and alternate hosts. Not surprisingly, given the less distinct biogeographic divisions of the full suite of diseases, they were not strongly associated with environmental and socio-political variables.

A key question that emerges from our work is why diseases, particularly those with vectors, can be grouped into biogeographic regions akin to those for birds and mammals. Clearly, climate influences the distribution of both vectors and pathogens, as we found here, and as shown in many other studies (e.g., Mordecai et al. 2013, Stensgaard et al. 2013). Yet, if climate were the

sole influence on the biogeographic regions of vectored pathogens, we would expect those regions to simply reflect the climatic zones of the world. They do not. Instead, they reflect both the influence of climate and, it appears, the influence of the biogeographic history of regions. This influence implies that many pathogens have been unable to disperse to all of the regions for which they and their vectors are climatically suited. In addition, for those pathogens that require alternate hosts (e.g., Chagas disease), the spread of pathogens may be limited by the need for dispersal of those hosts, the vector, and the pathogen. If true, these mechanisms bear obvious consequences for the future distributions of pathogens. As novel, non-vectored pathogens emerge, the relative lack of biogeographic regions for such pathogens suggests time, rather than climate and history are likely to influence where they will occur. Conversely, for vectored pathogens, it suggests that both of these barriers to dispersal may be more persistent.

More broadly, we have shown that diseases caused by pathogens can be persistently influenced by ancient evolutionary history and climate to such an extent as to cluster globally into biogeographic regions. But we suspect similar (though probably not identical) biogeographic regions exist for other human associates such as house guests, pests, and even mutualist species. Given that such species are those with the most direct effect on human fitness and some of the most direct effects on politics and economies, their biogeographic regions are fundamental to the human story. These regions are a reminder of the influences of evolutionary history and climate on our lives. We posit that among the most important factors influencing an individual's life expectancy and general fate is the disease biogeographic region in which they are born. But the influence of human history on the details of these regions is also a reminder that they can be changed.

#### ACKNOWLEDGMENTS

This work was supported by a grant from the USGS Southeast Regional Climate Science Center to R. R. Dunn. R. R. Dunn was also supported by Army Research Office Award W911NF-14-1-0556 and an NSF Career grant (0953390). We thank J. Osbourne, O. Ott, and M. Trautwein for their assistance with this project.

We also thank J.-P. Lessard, I. Kuznetsov, L. Bettine Kent, A.-S. Stensgaard, and anonymous reviewers for their comments on previous drafts of this manuscript.

#### LITERATURE CITED

- Blondel, V. D., J. Guillaume, R. Lambiotte, and E. Lefebvre. 2008. Fast unfolding of communities in large networks. *Journal of Statistical Mechanics: Theory and Experiment* 2008:P10008.
- Bonds, M. H., A. P. Dobson, and D. C. Keenan. 2012. Disease ecology, biodiversity, and the latitudinal gradient in income. *PLoS Biology* 10:e1001456.
- Borcard, D., F. Gillet, and P. Legendre. 2011. Cluster analysis. Pages 71–73 *in* *Numerical ecology with R*. Springer, New York, New York, USA.
- Boyle, T. P., G. M. Smillie, J. C. Anderson, and D. R. Beeson. 1990. A sensitivity analysis of nine diversity and seven similarity indices. *Research Journal of the Water Pollution Control Federation* 62:749–762.
- Centers for Disease Control and Prevention. 2003. Morbidity and Mortality Weekly Report. 52:241–248.
- Dunn, R. R., T. J. Davies, N. C. Harris, and M. C. Gavin. 2010. Global drivers of human pathogen richness and prevalence. *Proceedings of the Royal Society B* 277:2587–2595.
- Dunn, R. R., and T. S. Romdal. 2005. Mean latitudinal range sizes of bird assemblages in six Neotropical forest chronosequences. *Global Ecology and Biogeography* 14:359–366.
- Fauci, A. S. 2005. Emerging and reemerging infectious diseases: the perpetual challenge. *Academic Medicine* 80:1079–1085.
- Fenchel, T., and B. J. Finlay. 2004. The ubiquity of small species: patterns of local and global diversity. *BioScience* 54:777–784.
- Fincher, C. L., and R. Thornhill. 2008. Assortative sociality, limited dispersal, infectious disease and the genesis of the global pattern of religion diversity. *Proceedings of the Royal Society B* 275:2587–2594.
- Foley, D. H., L. M. Rueda, and R. C. Wilkerson. 2007. Insight into global mosquito biogeography from country species records. *Journal of Medical Entomology* 44:554–567.
- Gonçalves, M. L. C., A. Araújo, and L. F. Ferreira. 2003. Human intestinal parasites in the past: new findings and a review. *Memorias do Instituto Oswaldo Cruz* 98:103–118.
- Grenyer, R., C. D. L. Orme, S. F. Jackson, G. H. Thomas, R. G. Davies, T. J. Davies, K. E. Jones, V. A. Olson, R. S. Ridgely, and P. C. Rasmussen. 2006. Global distribution and conservation of rare and threatened vertebrates. *Nature* 444:93–96.
- Guernier, V., M. E. Hochberg, and J. Guégan. 2004.



- Ecology drives the worldwide distribution of human diseases. *PLoS Biology* 2:e141.
- Hay, S. I., K. E. Battle, D. M. Pigott, D. L. Smith, C. L. Moyes, S. Bhatt, J. S. Brownstein, N. Collier, M. F. Myers, and D. B. George. 2013. Global mapping of infectious disease. *Philosophical Transactions of the Royal Society B* 368:20120250.
- He, W. M., Y. Feng, W. M. Ridenour, G. C. Thelen, J. L. Pollock, A. Diaconu, and R. M. Callaway. 2009. Novel weapons and invasion: biogeographic differences in the competitive effects of *Centaurea maculosa* and its root exudate ( $\pm$ )-catechin. *Oecologia* 159:803–815.
- Holt, B. G., J. Lessard, M. K. Borregaard, S. A. Fritz, M. B. Araújo, D. Dimitrov, P. Fabre, C. H. Graham, G. R. Graves, and K. A. Jönsson. 2013. An update of Wallace's zoogeographic regions of the world. *Science* 339:74–78.
- IUCN. 2013. IUCN Red List of Threatened Species (2013.1): terrestrial mammals. <http://goo.gl/DTSh29>
- Jones, K. E., N. G. Patel, M. A. Levy, A. Storeygard, D. Balk, J. L. Gittleman, and P. Daszak. 2008. Global trends in emerging infectious diseases. *Nature* 451:990–993.
- Kreft, H., and W. Jetz. 2010. A framework for delineating biogeographical regions based on species distributions. *Journal of Biogeography* 37:2029–2053.
- Lafferty, K. D. 2009. The ecology of climate change and infectious diseases. *Ecology* 90:888–900.
- Lancichinetti, A., and S. Fortunato. 2009. Community detection algorithms: A comparative analysis. *Physical Review E* 80:056117.
- Maechler, M., P. Rousseeuw, A. Struyf, M. Hubert, and K. Hornik. 2013. cluster: Cluster analysis basics and extensions. R package version 1.14.4. <http://cran.r-project.org/web/packages/cluster/index.html>
- Marsollier, L., R. Robert, J. Aubry, J.-P. Saint André, H. Kouakou, P. Legras, A. Manceau, C. Mahaza, and B. Carbonnelle. 2002. Aquatic insects as a vector for *Mycobacterium ulcerans*. *Applied and Environmental Microbiology* 68:4623–4628.
- Mitchell, T. D., M. Hulme, and M. New. 2002. Climate data for political areas. *Area* 34:109–112.
- Møller, A., C. Fincher, and R. Thornhill. 2009. Why men have shorter lives than women: effects of resource availability, infectious disease, and senescence. *American Journal of Human Biology* 21:357–364.
- Mordecai, E. A., K. P. Paaijmans, L. R. Johnson, C. Balzer, T. Ben-Horin, E. de Moor, A. McNally, S. Pawar, S. J. Ryan, T. C. Smith, and K. D. Lafferty. 2013. Optimal temperature for malaria transmission is dramatically lower than previously predicted. *Ecology Letters* 16:22–30.
- Moyes, C. L., W. H. Temperley, A. J. Henry, C. R. Burgert, and S. I. Hay. 2013. Providing open access data online to advance malaria research and control. *Malaria Journal* 12:161.
- Nettle, D. 2009. Ecological influences on human behavioural diversity: a review of recent findings. *Trends in Ecology & Evolution* 24:618–624.
- Olden, J. D., N. L. Poff, and M. L. McKinney. 2006. Forecasting faunal and floral homogenization associated with human population geography in North America. *Biological Conservation* 127:261–271.
- Oliver, M. J., and A. J. Irwin. 2008. Objective global ocean biogeographic provinces. *Geophysical Research Letters* 35:L15601.
- Olsen, B., V. J. Munster, A. Wallensten, J. Waldenström, A. D. Osterhaus, and R. A. Fouchier. 2006. Global patterns of *Influenza A* virus in wild birds. *Science* 312:384–388.
- Pascual, M., and M. J. Bouma. 2009. Do rising temperatures matter? *Ecology* 90:906–912.
- Qiu, W., D. E. Dykhuizen, M. S. Acosta, and B. J. Luft. 2002. Geographic uniformity of the Lyme disease spirochete (*Borrelia burgdorferi*) and its shared history with tick vector (*Ixodes scapularis*) in the northeastern United States. *Genetics* 160:833–849.
- Rand, W. M. 1971. Objective criteria for the evaluation of clustering methods. *Journal of the American Statistical Association* 66:846–850.
- R Core Team. 2013. R: A language and environment for statistical computing. Version 2.15.1. R Foundation for Statistical Computing, Vienna, Austria.
- Smith, K. F., and J. Guégan. 2010. Changing geographic distributions of human pathogens. *Annual Review of Ecology, Evolution, and Systematics* 41:231–250.
- Smith, K. F., D. F. Sax, S. D. Gaines, V. Guernier, and J. Guégan. 2007. Globalization of human infectious disease. *Ecology* 88:1903–1910.
- Spielman, A. et al. 2004. Outbreak of West Nile virus in North America. *Science* 306:1473–1475.
- Stensgaard, A.-S., et al. 2013. Large-scale determinants of intestinal schistosomiasis and intermediate host snail distribution across Africa: Does climate matter? *Acta Tropica* 128:378–390.
- Thornhill, R., C. L. Fincher, and D. Aran. 2009. Parasites, democratization, and the liberalization of values across contemporary countries. *Biological Reviews* 84:113–131.
- Qian, H., and R. E. Ricklefs. 2008. Global concordance in diversity patterns of vascular plants and terrestrial vertebrates. *Ecology Letters* 11:547–553.
- United Nations. 2013. World population prospects: The 2012 revision. United Nations, Department of Economic and Social Affairs, Population Division, New York, New York, USA.
- Venables, W. N., and B. D. Ripley. 2002. Modern applied statistics with S. Fourth edition. Springer, New York, New York, USA.

- Wallace, A. R. 1876. The geographical distribution of animals: With a study of the relations of living and extinct faunas as elucidating the past changes of the Earth's surface. Cambridge University Press, Cambridge, UK.
- Wang, H. G., R. D. Owen, C. Sánchez-Hernández, and M. De Lourdes Romero-Almaraz. 2003. Ecological characterization of bat species distributions in Michoacán, México, using a geographic information system. *Global Ecology and Biogeography* 12:65–85.
- Ward, Jr., J. H. 1963. Hierarchical grouping to optimize an objective function. *Journal of the American Statistical Association* 58:236–244.
- West, L. S. 1951. Introduction. Pages 1-19 in *The housefly: its natural history, medical importance, and control*. Comstock, Ithaca, New York, USA.
- Wilcox, B. A., and R. R. Colwell. 2005. Emerging and reemerging infectious diseases: biocomplexity as an interdisciplinary paradigm. *EcoHealth* 2:244–257.
- Wolfe, N. D., C. P. Dunavan, and J. Diamond. 2007. Origins of major human infectious diseases. *Nature* 447:279–283.
- Xie, Y., J. MacKinnon, and D. Li. 2004. Study on biogeographical divisions of China. *Biodiversity & Conservation* 13:1391–1417.
- Yang, K., J. LeJeune, D. Alsdorf, B. Lu, C. Shum, and S. Liang. 2012. Global distribution of outbreaks of water-associated infectious diseases. *PLoS Neglected Tropical Diseases* 6:e1483.

## SUPPLEMENTAL MATERIAL

### APPENDIX

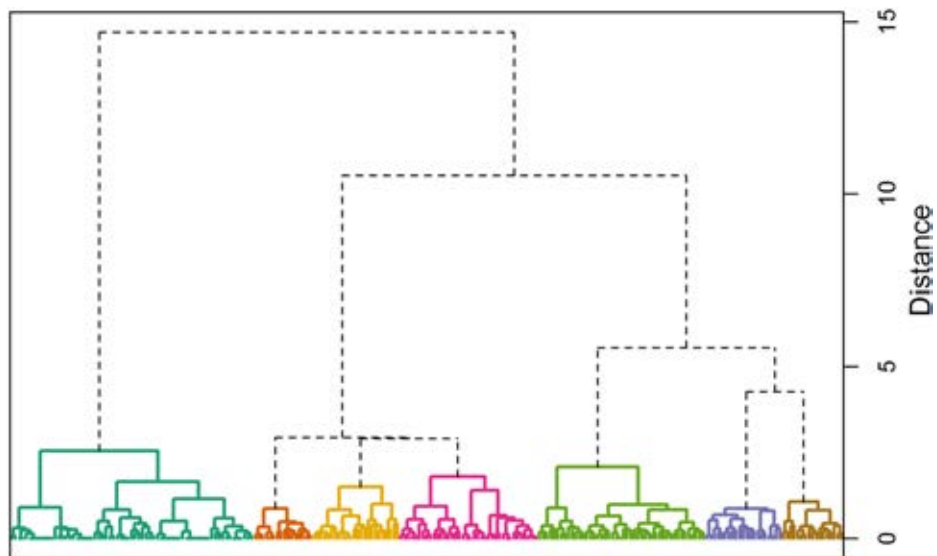


Fig. A1. Dendrograms from hierarchical clustering using Ward's distance for disease presence for countries ( $n = 229$ ) from the GIDEON database. The vertical axis represents Ward's distance between clusters. Our analyses resulted in 7 clusters using vector-borne disease ( $n = 93$ ) presence. Each differently colored cluster indicates a biogeographic region (colors match biogeographic regions from Fig. 1A).

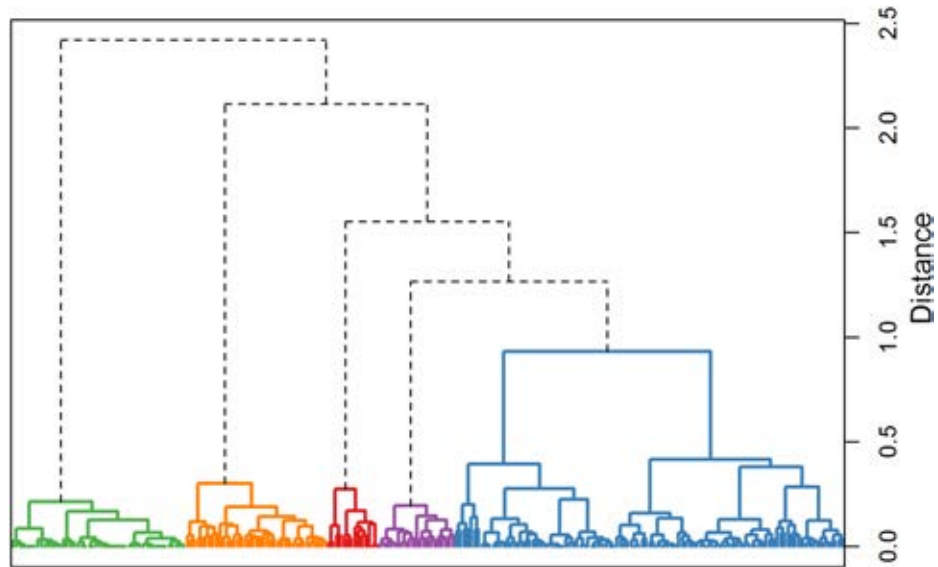


Fig. A2. Dendrograms from hierarchical clustering using Ward's distance for disease presence for countries ( $n = 229$ ) from the GIDEON database. The vertical axis represents Ward's distance between clusters. Our analyses resulted in 5 clusters using non-vector-borne disease ( $n = 208$ ) presence. Each differently colored cluster indicates a biogeographic region (colors match biogeographic regions from Fig. 1B).



Fig. A3. Map of biogeographic regions ( $n = 2$ ) delineated from the full suite of human-associated diseases ( $n = 301$ ) based on presence by country ( $n = 229$ ). Regions were created by hierarchical clustering using Ward's distance.

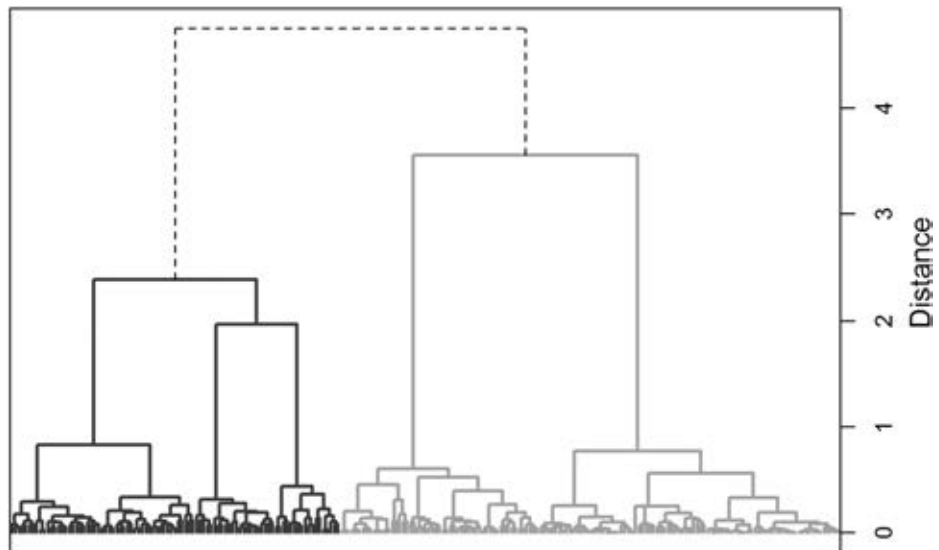


Fig. A4. Dendrogram from hierarchical clustering using Ward's distance for disease presence for countries from the GIDEON database. The vertical axis represents Ward's distance between clusters. Our analyses resulted in countries ( $n = 229$ ) being placed into 2 clusters using the full suite of diseases ( $n = 301$ ). Each differently colored cluster indicates a biogeographic region (colors match biogeographic regions from Appendix: Fig. A3).



Table A1. Diseases (n = 301) included from the GIDEON database for our analyses. Each diseases' group membership (V = vector-borne, NV = non-vector-borne) is also indicated.

Pathogen	Group membership
Actinomycosis	NV
Adenovirus	NV
Aeromonas hydrophila and marine Vibrio	NV
African tick bite fever	V
Alkhurma hemorrhagic fever	V
Amebic colitis	V
Amoeba, free living	NV
Amoebic abscess	V
Anaplasmosis	V
Angiostrongyliasis	NV
Angiostrongyliasis, abdominal	NV
Anisakiasis	NV
Anthrax	V
Argentine hemorrhagic fever	NV
Ascariasis	NV
Astrakhan fever	V
Babesiosis	V
Bacillary angiomatosis	V
Bacillus cereus food poisoning	NV
Balantidiasis	NV
Barmah Forest disease	V
Bartonellosis, cat borne	V
Bartonellosis, other systemic	V
Bartonellosis, South American	V
Bas-Congo virus	NV
Baylisascariasis	NV
Bertiella and Inermicapsifer	NV
Blastocystis hominis infection	NV
Bolivian hemorrhagic fever	NV
Botulism	NV
Brazilian hemorrhagic fever	NV
Brazilian purpuric fever	NV
Brucellosis	NV
Bunyaviridae infections, misc.	V
California encephalitis group	V
Campylobacteriosis	NV
Capillariasis, extraintestinal	NV
Capillariasis, intestinal	NV
Cercarial dermatitis	NV
Chancroid	NV
Chandipura and Vesicular stomatitis viruses	NV
Chikungunya	V
Chlamydial infections misc	NV
Chlamydophila pneumoniae infection	NV
Cholecystitis and cholangitis	NV
Cholera	NV
Chronic meningococemia	NV
Clonorchiasis	NV
Clostridial food poisoning	NV
Clostridial myonecrosis	NV
Clostridium difficile colitis	NV
Coenurosis	NV
Colorado tick fever	V
Coltivirus, Old World	V
Common cold	NV
Conjunctivitis, inclusion	NV
Conjunctivitis, viral	NV
Crimean-Congo hemorrhagic fever	V
Cryptosporidiosis	NV
Cutaneous larva migrans	NV
Cyclosporiasis	NV
Cysticercosis	NV
Cytomegalovirus infection	NV
Dengue	V
Dicrocoeliasis	NV
Dientamoeba fragilis infection	NV

Table A1. Continued.

Pathogen	Group membership
Diocophyme renalis infection	NV
Diphtheria	NV
Diphyllobothriasis	NV
Dipyldiasis	NV
Dirofilariasis	V
Dracunculiasis	NV
Eastern equine encephalitis	V
Ebola	NV
Echinococcosis, American polycystic	NV
Echinococcosis, multilocular	NV
Echinococcosis, unilocular	NV
Echinostomiasis	NV
Ehrlichiosis, human monocytic	V
Endemic syphilis (bejel)	NV
Entamoeba polecki infection	NV
Enteritis necroticans	NV
Enterobiasis	NV
Enterovirus infection	NV
Epidural abscess	NV
Erysipelas or cellulitis	NV
Erysipeloid	NV
Erythrasma	NV
Escherichia coli diarrhea	NV
Fascioliasis	NV
Fasciolopsiasis	NV
Filariasis, Bancroftian	V
Filariasis, Brugia malayi	V
Filariasis, Brugia timori	V
Flinders Island spotted fever	V
Gardnerella vaginalis infection	NV
Gastrodiscoidiasis	NV
Gastroenteritis, viral	NV
Giardiasis	NV
Glanders	NV
Gnathostomiasis	NV
Gongylonemiasis	NV
Gonococcal infection	NV
Granuloma inguinale	NV
Group C virus fevers	V
Hantavirus infection, Old World	NV
Hantavirus pulmonary syndrome	NV
Hendra virus infection	NV
Hepatitis A	NV
Hepatitis B	NV
Hepatitis C	NV
Hepatitis D	NV
Hepatitis E	NV
Hepatitis G	NV
Herpes B infection	NV
Herpes simplex encephalitis	NV
Herpes simplex infection	NV
Herpes zoster	NV
Heterophyid infections	NV
HIV infection, initial illness	NV
Hookworm	NV
Human herpesvirus 6 infection	NV
Hymenolepis diminuta infection	NV
Hymenolepis nana infection	NV
Ilheus and Bussuquara	V
Infectious mononucleosis or EBV infection	NV
Influenza	NV
Intestinal spirochetosis	NV
Intracranial venous thrombosis	NV
Isosporiasis	NV
Israeli spotted fever	V
Japanese encephalitis	V
Japanese spotted fever	V

Table A1. Continued.

Pathogen	Group membership
Karelian fever	V
Kingella infection	NV
Kyasanur Forest disease	V
Lagochilascariasis	NV
Laryngotracheobronchitis	NV
Lassa fever	NV
Legionellosis	NV
Leishmaniasis, cutaneous	V
Leishmaniasis, mucocutaneous	V
Leishmaniasis, visceral	V
Leprosy	NV
Leptospirosis	NV
Listeriosis	NV
Liver abscess, bacterial	NV
Loiasis	V
Louping ill	V
Lyme disease	V
Lymphocytic choriomeningitis	NV
Lymphogranuloma venereum	NV
Malaria	V
Malignant otitis externa	NV
Mammomonogamiasis	NV
Mansonelliasis, <i>M. ozzardi</i>	V
Mansonelliasis, <i>M. perstans</i>	V
Mansonelliasis, <i>M. streptocerca</i>	V
Marburg virus disease	NV
Mayaro	V
Measles	NV
Melioidosis	NV
Meningitis, aseptic (viral)	NV
Meningitis, bacterial	NV
Metagonimiasis	NV
Metorchiasis	NV
Microsporidiosis	NV
Moniliformis and <i>Macracanthorhynchus</i>	NV
Monkeypox	NV
Mumps	NV
Murray Valley encephalitis	V
Mycobacteriosis, <i>M. marinum</i>	NV
Mycobacteriosis, <i>M. scrofulaceum</i>	NV
Mycobacteriosis, <i>M. ulcerans</i>	V
Mycobacteriosis, miscellaneous nontuberculous	NV
Mycoplasma (miscellaneous) infections	NV
<i>Mycoplasma pneumoniae</i> infection	NV
Myiasis	V
Nanophyetiasis	NV
Necrotizing skin or soft tissue infection	NV
Neutropenic typhlitis	NV
New World phleboviruses	V
Nipah and Nipah-like virus infection	NV
Nocardiosis	NV
North Asian tick typhus	V
Ockelbo disease	V
Oesophagostomiasis	NV
Old World phleboviruses	V
Omsk hemorrhagic fever	V
Onchocerciasis	V
O'nyong nyong	V
Opisthorchiasis	NV
Orf	NV
Ornithosis	NV
Oropouche	V
Otitis media	NV
Paragonimiasis	NV
Parainfluenza virus infection	NV
Parvovirus B19 infection	NV
Pediculosis	V

Table A1. Continued.

Pathogen	Group membership
Pentastomiasis, Armillifer	NV
Pentastomiasis, Linguatula	NV
Pericarditis, bacterial	NV
Pertussis	NV
Pharyngeal and cervical space infections	NV
Pharyngitis, bacterial	NV
Pinta	V
Plague	V
Pleurodynia	NV
Pneumonia - bacterial	NV
Pogosta disease	V
Poliomyelitis	NV
Powassan	V
Pseudocowpox	NV
Pyoderma (furunculosis impetigo etc)	NV
Pyomyositis	NV
Pythiosis	NV
Q-fever	NV
Queensland tick typhus	V
Rabies	NV
Rat bite fever, spirillary	NV
Rat bite fever, streptobacill	NV
Relapsing fever	V
Respiratory syncytial virus infection	NV
Rheumatic fever	NV
Rhinoscleroma and ozena	NV
Rhinospordiosis	NV
Rhodococcus equi infection	NV
Rickettsia felis infection	V
Rickettsia sibirica mongolotimonae infection	V
Rickettsialpox	V
Rift Valley fever	V
Rocio	V
Rocky Mountain spotted fever	V
Ross River disease	V
Rotavirus infection	NV
Rubella	NV
Salmonellosis	NV
Sarcocystosis	NV
SARS and hCoV-EMC	NV
Scabies	V
Scarlet fever	NV
Schistosomiasis, haematobium	NV
Schistosomiasis, intercalatum	NV
Schistosomiasis, japonicum	NV
Schistosomiasis, mansoni	NV
Schistosomiasis, mattheei	NV
Schistosomiasis, mekongi	NV
Sennetsu neorickettsiosis	V
Septicemia, bacterial	NV
Shigellosis	NV
Sindbis	V
Sinusitis	NV
Smallpox	NV
Sparganosis	NV
Spondweni	V
Spotted fevers, Old World	V
St. Louis encephalitis	V
Staphylococcal food poisoning	NV
Staphylococcal scalded skin syndrome	NV
Streptococcus suis infection	NV
Strongyloidiasis	NV
Suppurative parotitis	NV
Syphilis	NV
Taeniasis	NV
Tanapox virus infection	NV
Tetanus	NV



Table A1. Continued.

Pathogen	Group membership
Thelaziasis	V
Thogoto	V
Tick-borne encephalitis	V
Tick-borne encephalitis: Russian spring-summer	V
Toxocariasis	NV
Toxoplasmosis	NV
Trachoma	V
Trichinosis	NV
Trichomoniasis	NV
Trichostrongyliasis	NV
Trichuriasis	NV
Trypanosomiasis, African	V
Trypanosomiasis, American	V
Tuberculosis	NV
Tularemia	V
Tungiasis	NV
Typhoid and enteric fever	NV
Typhus, endemic	V
Typhus, epidemic	V
Typhus, scrub	V
Vaccinia and cowpox	NV
Varicella	NV
Venezuelan equine encephalitis	V
Venezuelan hemorrhagic fever	NV
Vibrio parahaemolyticus infec.	NV
Wesselbron	V
West Nile fever	V
Western equine encephalitis	V
Whipple's disease	NV
Whitewater Arroyo virus infection	NV
Yaws	NV
Yellow fever	V
Yersiniosis	NV
Zika	V

Table A2. Biogeographic region membership by country and analysis schema. Vector-borne diseases (n = 93), non-vector-borne diseases (n = 208), and the full suite of diseases (n = 301) for regions created by hierarchical clustering using Ward's distance.

Country	Full suite	Non-vector	Vector
Afghanistan	1	1	1
Albania	1	1	1
Algeria	1	1	2
American Samoa	1	2	3
Andorra	1	1	3
Angola	2	3	4
Anguilla	1	2	3
Antigua Barbuda	1	2	3
Argentina	2	4	5
Armenia	1	1	2
Aruba	1	2	3
Australia	1	1	6
Austria	1	1	2
Azerbaijan	1	1	2
Azores	1	2	1
Bahamas	1	2	3
Bahrain	1	1	1
Bangladesh	2	1	7
Barbados	1	2	3
Belarus	1	1	2
Belgium	1	1	6
Belize	2	4	5
Benin	2	3	4
Bermuda	1	2	3
Bhutan	1	1	3
Bolivia	2	4	5
Bosnia and Herzegovina	1	1	2
Botswana	2	1	4
Brazil	2	4	5
British Virgin Islands	1	2	3
Brunei	1	1	3
Bulgaria	1	1	2
Burkina Faso	2	3	4
Burundi	2	3	4
Cambodia	2	5	7
Cameroon	2	3	4
Canada	1	1	6
Canary Islands	1	2	6
Cape Verde	1	1	3
Cayman Islands	1	2	3
Central African Republic	2	3	4
Chad	2	3	4
Chile	2	4	6
China	2	5	7
Christmas Island	1	2	3
Colombia	2	4	5
Comoros	1	1	3
Congo	2	3	4
Cook Islands	1	2	3
Costa Rica	2	4	5
Croatia	1	1	2
Cuba	1	1	3
Cyprus	1	1	6
Czech Republic	1	1	2
Democratic Rep of Congo	2	3	4
Denmark	1	1	6
Djibouti	2	1	4
Dominica	1	2	3
Dominican Republic	1	1	3
East Timor	2	5	7
Ecuador	2	4	5
Egypt	2	1	4
El Salvador	2	4	5
Equatorial Guinea	2	3	4

Table A2. Continued.

Country	Full suite	Non-vector	Vector
Eritrea	2	3	4
Estonia	1	1	2
Ethiopia	2	3	4
Falkland Islands	1	2	3
Fiji	1	2	3
Finland	1	1	6
France	1	1	2
French Guiana	2	4	5
French Polynesia	1	2	3
Gabon	2	3	4
Gambia	2	3	4
Georgia	1	1	2
Germany	1	1	6
Ghana	2	3	4
Gibraltar	1	2	1
Greece	1	1	2
Greenland	1	1	3
Grenada	1	1	3
Guadeloupe	1	2	3
Guam	1	1	3
Guatemala	2	4	5
Guinea	2	3	4
Guinea Bissau	2	3	4
Guyana	2	4	5
Haiti	1	1	3
Honduras	2	4	5
Hong Kong	1	1	3
Hungary	1	1	2
Iceland	1	2	3
India	2	5	7
Indonesia	2	5	7
Iran	1	1	2
Iraq	1	1	1
Ireland	1	1	6
Israel	1	1	2
Italy	1	1	2
Ivory Coast	2	3	4
Jamaica	1	1	5
Japan	2	5	6
Jordan	1	1	1
Kazakhstan	1	1	2
Kenya	2	3	4
Kiribati	1	2	3
Kuwait	1	1	1
Kyrgyzstan	1	1	2
Laos	2	5	7
Latvia	1	1	2
Lebanon	1	1	1
Lesotho	2	1	4
Liberia	2	3	4
Libya	2	1	1
Liechtenstein	1	1	6
Lithuania	1	1	2
Luxembourg	1	1	6
Macao	1	1	3
Macedonia	1	1	2
Madagascar	2	3	4
Malawi	2	3	4
Malaysia	2	5	7
Maldives	1	1	3
Mali	2	3	4
Malta	1	1	1
Marshall Islands	1	2	3
Martinique	1	2	3
Mauritania	2	3	4
Mauritius	1	1	3
Mexico	2	4	5

Table A2. Continued.

Country	Full suite	Non-vector	Vector
Micronesia	1	2	3
Moldova	1	1	2
Monaco	1	2	1
Mongolia	1	1	2
Montserrat	1	2	3
Morocco	2	1	2
Mozambique	2	3	4
Myanmar Burma	2	1	7
Namibia	2	1	4
Nauru	1	2	3
Nepal	2	1	7
Netherlands	1	1	6
Netherlands Antilles	1	2	3
New Caledonia	1	1	3
New Zealand	1	1	3
Nicaragua	2	4	5
Niger	2	3	4
Nigeria	2	3	4
Niue	1	2	3
Norfolk Island	1	2	3
Northern Marianas	1	2	3
Norway	1	1	6
Oman	2	1	1
Pakistan	2	1	7
Palau	1	2	3
Panama	2	4	5
Papua New Guinea	1	1	7
Paraguay	2	4	5
Peoples Dem Rep Korea	2	5	6
Peru	2	4	5
Philippines	2	5	7
Pitcairn Island	1	2	3
Poland	1	1	2
Portugal	1	1	2
Puerto Rico	1	1	3
Qatar	1	1	1
Republic of Korea	2	5	6
Reunion	1	1	3
Romania	1	1	2
Russian Federation	1	1	7
Rwanda	2	3	4
Samoa	1	2	3
San Marino	1	1	3
Sao Tome Principe	2	3	3
Saudi Arabia	2	1	4
Scotland	1	1	6
Senegal	2	3	4
Serbia and Montenegro	1	1	2
Seychelles	1	2	3
Sierra Leone	2	3	4
Singapore	1	1	3
Slovakia	1	1	2
Slovenia	1	1	2
Solomon Islands	1	2	3
Somalia	2	3	4
South Africa	2	3	4
Spain	1	1	2
Sri Lanka	2	1	7
St. Helena	1	2	3
St. Kitts Nevis	1	2	3
St. Lucia	1	2	3
St. Vincent Grenadines	1	2	3
Sudan	2	3	4
Suriname	2	4	5
Swaziland	2	1	4
Sweden	1	1	6
Switzerland	1	1	6



Table A2. Continued.

Country	Full suite	Non-vector	Vector
Syria	1	1	1
Taiwan	2	5	6
Tajikistan	1	1	2
Tanzania	2	3	4
Thailand	2	5	7
Togo	2	3	4
Tokelau	1	2	3
Tonga	1	2	3
Trinidad Tobago	2	1	5
Tunisia	1	1	2
Turkey	1	1	2
Turkmenistan	1	1	2
Turks and Caicos Islands	1	2	3
Tuvalu	1	2	3
Uganda	2	3	4
Ukraine	1	1	2
United Arab Emirates	1	1	1
United Kingdom	1	1	6
United States	1	1	6
Uruguay	2	4	6
Uzbekistan	1	1	2
Vanuatu	1	2	3
Venezuela	2	4	5
Vietnam	2	5	7
Virgin Islands US	1	2	3
Wake Island	1	2	3
Wallis and Futuna Islands	1	2	3
Western Sahara	1	1	3
Yemen	2	1	4
Zambia	2	3	4
Zimbabwe	2	3	4