

## An equilibrium theory signature in the island biogeography of human parasites and pathogens

Kévin Jean, William Burnside, Lynn Carlson, Katherine Smith, Jean-François Guégan

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1 **An equilibrium theory signature in the island biogeography of human parasites and**  
2 **pathogens**

3

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7 **Authors:** *Kévin Jean\**; *William R. Burnside\**; *Lynn Carlson*; *Katherine Smith*; *Jean-François Guégan*

8 \* both authors contributed equally to the work.

9

10 Kévin Jean: MRC Center for Outbreak Analysis, Department of Infectious Diseases  
11 Epidemiology, Imperial College London, St Mary's Campus, Norfolk Place,  
12 London, W2 1PG, UK.  
13 Center for Research in Epidemiology and Population Health, Team 4,  
14 INSERM U1018, 82 rue du Général Leclerc, 94276 Le Kremlin Bicêtre  
15 Cedex, France;  
16 [k.jean@imperial.ac.uk](mailto:k.jean@imperial.ac.uk)  
17  
18 William R. Burnside: National Socio-Environmental Synthesis Center (SESYNC); 1 Park Place,  
19 Suite 300; Annapolis, MD 21403, US.  
20 [bburnside@sesync.org](mailto:bburnside@sesync.org)  
21  
22 Lynn Carlson: Geological Sciences, Brown University, PO Box 1846 Providence, RI 02912,  
23 US.  
24 [Lynn\\_Carlson@brown.edu](mailto:Lynn_Carlson@brown.edu)  
25  
26 Katherine Smith: Department of Ecology and Evolutionary Biology; Brown University; 91  
27 Waterman St, Box G-W; Providence, RI 02912, US.  
28 [Katherine\\_Smith@brown.edu](mailto:Katherine_Smith@brown.edu)  
29  
30 Jean-François Guégan: UMR MIVEGEC IRD-CNRS-Université de Montpellier, Centre IRD de  
31 Montpellier, B.P. 64501, Montpellier, France; and International programme  
32 FutureEarth, ecoHEALTH initiative.  
33 [Jean-Francois.Guegan@ird.fr](mailto:Jean-Francois.Guegan@ird.fr)  
34  
35

36

37 Correspondence to: Kévin Jean,  
38 Imperial College London, Dept. of Infectious Diseases Epidemiology;  
39 St Mary's Campus  
40 Norfolk Place  
41 London, W2 1PG, UK  
42 [kevin.jean.lab@gmail.com](mailto:kevin.jean.lab@gmail.com),  
43

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48 **ABSTRACT**

49 **Aim:** Our understanding of the ecology and biogeography of microbes, including those that  
50 cause human disease, lags behind that for larger species. Despite recent focus on the  
51 geographic distribution of viruses and bacteria, the overall environmental distribution of  
52 human pathogens and parasites on Earth remains incompletely understood. As islands have  
53 long inspired basic ecological insight, we aimed to assess whether the micro-organisms that  
54 cause human disease in modern times follow patterns common to insular plants and animals.

55 **Location:** Global and regional.

56 **Methods:** Relying on the publically-accessible GIDEON database, we use the spatial  
57 distribution of nearly 300 human parasites and pathogens across 66 island countries and  
58 territories to assess the current predictive value of the “equilibrium theory” of island  
59 biogeography (MacArthur & Wilson 1967). The relationships between species richness and:  
60 i) island surface area and, ii) distance to the nearest mainland were investigated with linear  
61 regression, and ANCOVAs were used to test for differences in these relationships with  
62 respect to pathogen ecology and taxonomy.

63 **Results:** Pathogen species richness increases with island surface area and decreases with  
64 distance to the nearest mainland. The effect of area is more than 10 times lower than that  
65 usually reported for macro-organisms but is greater than the effect of distance. The strongest  
66 relationships are for pathogens that are vector-borne, zoonotic (with humans as dead-end  
67 hosts) or protozoan.

68 **Main conclusion:** Our results support the theory’s basic predictions: disease diversity is a  
69 positive function of island area and a negative function of island isolation. However,  
70 differences in the effects of area, distance, and pathogen ecology suggest that globalization,  
71 likely through human travel and the animal trade, has softened these relationships.  
72 Parasites that primarily target non-human species, whose distributions are more constrained  
73 by island life than are those restricted to human hosts, drive the island biogeography of  
74 human disease.

75

## 76 Introduction

77 Infectious diseases remain one of the chief causes of human morbidity and mortality  
78 worldwide, especially among the young and the poor (Lozano *et al.*, 2012). Understanding  
79 the drivers of human pathogen diversity, a key predictor of infectious disease prevalence, is  
80 a critical challenge of the 21<sup>st</sup> century (Dunn *et al.*, 2010). The diversity of infectious agents  
81 and the burden of disease vary dramatically across the globe, as they have throughout  
82 human history (Wolfe *et al.*, 2007; Dunn *et al.*, 2010). This disease burden exerts a profound  
83 effect on the economic fortunes of entire nations and world regions (Bloom & Sachs, 1998;  
84 Bonds *et al.*, 2012). However, our understanding of the biogeography of human disease is  
85 surprisingly limited. Less than ten infectious diseases are mapped comprehensively (Hay *et al.*,  
86 2013), and we know less about the distributions of many human parasites and pathogens  
87 than we do about those of most rare birds (Just *et al.*, 2014). As human populations grow  
88 and geographically change with urbanization and migration, exposing populations to novel  
89 social and ecological environments, there is an increasing need for first-order predictions to  
90 guide policy and future research.

91 Human parasites and pathogens interact both with their human hosts and with the  
92 broader environment, so their distributions should be a function of general ecological factors  
93 as well as of the specific ecology of *Homo sapiens*. Indeed, despite our sense of microbes'  
94 ubiquity, ecology still drives the worldwide distribution of human disease, the inspiration for  
95 the eponymous Baas-Becking hypothesis: "*Everything is everywhere, but the environment  
96 selects*" (Baas-Becking, 1934). As with species generally, the tropics have many more  
97 disease-causing species (Guernier *et al.*, 2004; Jones *et al.*, 2008; Peterson, 2008), and  
98 Earth can be divided into biogeographic human-disease regions (Just *et al.*, 2014).  
99 Considered broadly, our parasites and pathogens display patterns characteristic of animals  
100 and plants generally (Guernier *et al.*, 2004). At the same time, pestilence follows patterns of  
101 human dispersal and interaction. As anatomically modern humans migrated to new  
102 environments, such as from Africa to Eurasia and then to the Americas, our ancestors  
103 spread some pathogens, shed others, and acquired new ones along the way (Burnside *et al.*,  
104 2012). Historic and continuing changes in human population density, promoted by agriculture  
105 and then by industrialization, engendered and supported the "crowd-epidemic diseases,"  
106 such as seasonal influenza, measles and pertussis, that afflict urban residents (Bjørnstad &  
107 Harvill, 2005; Furuse *et al.*, 2010). With globalization, increasing travel, migration, and trade  
108 have spread pathogens and parasites specific to humans worldwide, though those with  
109 animals as their main reservoir and humans as secondary hosts remain more localized  
110 (Smith *et al.*, 2007). Illuminating the processes driving such large-scale epidemiological  
111 patterns is a growing focus of disease ecology (Guernier *et al.*, 2004; Dunn *et al.*, 2010;  
112 Bonds *et al.*, 2012).

113 A proven avenue for exploring the influence of spatial ecological and evolutionary  
114 processes is to study biodiversity patterns on islands. As Darwin argued, islands form natural  
115 laboratories where processes can be observed that are too complex to track on land masses  
116 (Darwin, 1859). MacArthur and Wilson formalized this insight in their influential "equilibrium  
117 theory of island biogeography" (MacArthur & Wilson, 1967). According to the theory, the  
118 number of species living on an island represents a dynamic equilibrium between species  
119 arriving from elsewhere (immigration) and those dying out some time after they arrive  
120 (extinction). The immigration rate declines with distance to the nearest mainland, the source

121 pool, while the extinction rate declines with island area, because larger islands can support  
122 larger populations with correspondingly lower probabilities of dying out. Once an island has  
123 reached ecological equilibrium, invasions will balance extinctions and the number of species  
124 will remain unchanged even though their composition may vary over time. The equilibrium  
125 theory of island biogeography has successfully explained a range of patterns of insular plant  
126 and animal species (Lomolino *et al.*, 2010) as well as of microbes with animal hosts (Bell *et*  
127 *al.*, 2005; Orrock *et al.*, 2011; Svensson-Coelho & Ricklefs, 2011), but its applicability to  
128 human pathogens is unclear. Previous research supports the existence of biogeographical  
129 patterns in microbes (e.g. Martiny, 2006; Hanson, 2012), but these studies were limited to  
130 free-living microbial taxa and not focused on host-associated pathogenic species. Recent,  
131 more-limited work on historic human populations supports the theorized effect of island size  
132 on the diversity of vector-borne pathogens (Cashdan, 2014), though the influence of distance  
133 and the effect of modern industrial lifestyles, with their enhanced mobility and access to  
134 medicine and public health, is less clear.

135 In this study, we use the Global Infectious Disease and Epidemiology Online Network  
136 Database (GIDEON) to examine whether the distribution of nearly 300 human pathogens  
137 occurring on different islands conforms to the general predictions of island biogeography  
138 theory, specifically that pathogen richness is a positive function of island size and a negative  
139 function of distance to the nearest mainland.

140

## 141 **Material & Methods**

### 142 *Data collection*

143 Analyses were based on a subset of data extracted and compiled from GIDEON  
144 (<http://www.gideononline.com/>). GIDEON provides clinical, geographical, and  
145 epidemiological information on 332 unique viruses, bacteria, fungi, protozoans, and  
146 helminths infecting humans in each of the 222 countries and administrative territories of the  
147 world. The database is updated regularly using publications from Medline based on a list of  
148 keywords and search information published by national Health Ministers, the World Health  
149 Organization (WHO), and the U.S. Centers for Disease Control and Prevention (CDC). As  
150 such, GIDEON is the most current, global database available for human infectious disease.

151 For simplicity, we use the term “pathogens” in this manuscript to cover both  
152 pathogens and parasites and consider disease names (e.g. measles) as synonymous with  
153 the infectious agents that cause them.

154 Following Guernier *et al.* (Guernier *et al.*, 2004) and Smith *et al.* (Smith *et al.*, 2007),  
155 we excluded pathogens causing infectious diseases that did not meet the following three  
156 criteria: (i) those with multiple etiological origins, (ii) those with major uncertainties  
157 surrounding national presence/absence, and (iii) vector- and reservoir-borne pathogens with  
158 imprecise information on hosts. The resulting database included 271 pathogens: 85 viruses,  
159 87 bacteria, 15 fungi, 64 helminths, and 20 protozoans.

160

161 We categorized pathogens three ways to assess the importance of different  
162 ecological and evolutionary processes: by host association, by transmission mode, and by  
163 taxonomy. We assigned host associations following Smith *et al.* (2007) as: human specific  
164 pathogens (n=83), which circulate exclusively in the human reservoir and are transmitted  
165 from person to person and hence are contagious, e.g. measles; (ii) zoonotic pathogens  
166 (n=152), which develop, mature, and reproduce entirely in non-human hosts but can still  
167 infect humans, who are then dead-end hosts, e.g. rabies; and (iii) multi-host pathogens  
168 (n=36), which can use both human and non-human hosts to complete their life-cycle, e.g.  
169 Ebola virus disease. We assigned pathogen transmission mode as follows: pathogens that  
170 spread through an arthropod vector (n=82) versus those not transmitted through a vector  
171 (n=189). Finally, we categorized pathogens by major taxonomic group: viruses, bacteria,  
172 fungi, protozoans, and helminths (including both helminth worms and nematodes).

173 Our geographic choices are driven by island biogeography theory. From 222  
174 administrative territories recorded in GIDEON, we extracted data from 66 island countries  
175 and territories (Fig. 1.A), the largest being Madagascar and the smallest Tokelau (Appendix  
176 S1). Inclusion or exclusion of islands was driven by the completeness of information for a set  
177 of geographic, socioeconomic and demographic indicators based on previous,  
178 complementary work (Guégan & Broutin, 2009).

179 Since including all islands in this sample together could introduce confounding  
180 effects, such as those related to latitude, and because the equilibrium theory was originally  
181 elaborated for a group of islands within an archipelago, we extracted from the whole island  
182 dataset two regional island subsets, one for Caribbean islands (n=25, Fig. 1.B) and one for  
183 Pacific islands (n=21, Fig. 1.C). Territorial surface areas (in square-km) and total human  
184 population size were extracted from the 2010 *World Factbook*, published by the U.S. Central  
185 Intelligence Agency and updated yearly. ArcGIS software, version 9.3.1 (Esri, Redlands, CA,  
186 USA) was used to compute the centroid of each island and the distance, in km, from that  
187 centroid to the nearest mainland shoreline.

188

### 189 *Statistical analysis*

190 We used univariate linear regression models to investigate the relationship between  
191 the total number of pathogenic species (Species Richness, or SR) and both island surface  
192 area and distance from an island to the nearest mainland. SR and surface area variables  
193 were normalized by log-transformation. This linear relationship expressed in logarithmic  
194 space corresponds to the classical power model of the species-area relationship, generally  
195 expressed as  $SR=cA^z$ , where  $A$  is the surface area,  $c$  is the intercept, and  $z$  in the linear  
196 coefficient, or slope (Triantis *et al.*, 2012). The relatively small sample sizes prevented a  
197 reasonable use of multivariate analysis. Linear regression provided the most simple, robust  
198 method to test for monotony in the predicted relationships between pathogen diversity and  
199 the variables of interest. Although non-linear models may have explained more of the  
200 variation in some of the studied relationships, a comparison of models and discussion of their  
201 potential underlying mechanisms processes are beyond the scope of this research.

202 The analysis was first conducted on the whole set of island pathogen species and  
203 then on this set broken down by (i) host-requirement (human-only, zoonotic, multi-host), (ii)

204 transmission mode (vector-borne, directly transmitted) and (iii) taxonomy (bacteria, virus,  
205 fungi, protozoans, helminths). First, we calculated the SR for each of these three  
206 breakdowns. For transmission mode, for instance, we calculated SR for vector-borne  
207 pathogens and SR for directly-transmitted pathogens. Second, we estimated the linear  
208 relationship between these SR values and our covariates of interest, island surface area and  
209 distance to the nearest mainland. Finally, we assessed differences among these linear  
210 relationships and our covariates of interest using a generalized analysis of covariance  
211 (ANCOVA). For example, we tested for statistical difference in the linear relationship  
212 between SR and surface area (or distance to mainland) between vector-borne and directly-  
213 transmitted pathogens.

214 In the case of human-specific pathogens, one could consider the ultimate area  
215 occupied by a pathogen species as defined by the host population size. In order to test this  
216 hypothesis, we conducted a complementary analysis using univariate linear regression  
217 models to investigate the relationship between pathogen SR and island human population  
218 (log-transformed), hypothesizing that any relationship for the larger sample would be driven  
219 by that for human-only pathogens and that the relationship would be strongest for obligate  
220 human pathogens.

221 Analyses were conducted on the whole island dataset and then on both regional sub-  
222 datasets. Analyses were conducted using R software v2.15.1 (R Development Core Team,  
223 2005).

224

## 225 **Results**

### 226 *Species richness relationships with area and distance in the entire sample*

227 Our findings for the entire sample of island countries and territories supported  
228 predictions from the equilibrium theory of island biogeography, though the effect of area on  
229 pathogen diversity was much more pronounced than that of distance. Fig. 2 presents the  
230 island SR plotted against, respectively, surface area (Fig. 2.A) and distance to the mainland  
231 (Fig. 2.B). Larger islands support more species of pathogens, as shown in Fig. 2.A ( $y =$   
232  $1.695 \times 10^{-2} x + 2.022$ ,  $p < 10^{-3}$ ). Island surface area explained more than 40% of the total  
233 variance of pathogen SR ( $R^2_{\text{adj}}=0.407$ ). In turn, more-isolated islands tended to support fewer  
234 pathogen species, as shown in Fig. 2.B ( $y = - 6.394 \times 10^{-6} x + 2.087$ ,  $p=0.014$ ), though this  
235 relationship explains less than 10% of the total variance of SR ( $R^2_{\text{adj}}=0.0766$ ).

236

### 237 *Relationships between SR and host requirement, transmission pathway, and taxonomy*

238 Across all pathogen subcategories, SR increased with island surface area and  
239 decreased with distance to the nearest mainland. However, as Fig. 3 shows, the extent of  
240 these relationships, as indicated by differences among regression slopes, is driven by  
241 zoonotic status, vectorial transmission, and protozoan and helminthian taxonomy. Pathogens  
242 that infect humans obligately, those that do not require a vector for transmission, and those  
243 that are relatively small (viruses, bacteria) are affected much less by island biogeography.



244 The positive relationship between SR and surface area was significant for every  
245 pathogen subcategory (each  $p < 10^{-3}$ , Table 1). However, as presented in Table 1, the  
246 strength of this relationship varied significantly across pathogen host-requirement categories  
247 (slope coefficients, Human Only pathogens:  $5.768 \times 10^{-3}$ , Multi-Host pathogens:  $1.210 \times 10^{-2}$ ,  
248 Zoonotic pathogens:  $3.788 \times 10^{-2}$ ; ; ANCOVA- $p < 10^{-3}$ ), transmission pathways (slope  
249 coefficients, Directly Transmitted pathogens: 0.0132, Vector-borne pathogens: 0.0469;  
250 ANCOVA- $p < 10^{-3}$ ), and taxonomic categories (slope coefficients, Bacteria:  $8.932 \times 10^{-3}$ ,  
251 Viruses:  $1.486 \times 10^{-2}$ , Fungi:  $1.522 \times 10^{-2}$ , Protozoans:  $3.733 \times 10^{-2}$ , Helminths:  $2.416 \times 10^{-2}$ ;  
252 ANCOVA- $p < 10^{-3}$ ).

253 The negative relationship between SR and distance to the nearest mainland was  
254 significant or at the limit of significance for nine of the ten categories we considered (for 5  
255 categories:  $p < 0.05$ ; for 4 categories:  $p < 0.10$ ; Table 1). The strength of this relationship varied  
256 significantly among pathogen transmission pathway categories (slope coefficients for Directly  
257 Transmitted and Vector-borne pathogens, respectively:  $-4.380 \cdot 10^{-6}$  and  $-2.310 \cdot 10^{-6}$ ;  
258 ANCOVA- $p = 0.0343$ ).

### 259 *Complementary analysis on regional sub-datasets*

260 As for the dataset as a whole, we found that larger islands supported greater  
261 pathogen diversity in the Caribbean and Pacific subsets ( $p < 10^{-3}$  and  $p = 0.001$ , respectively).  
262 However, a significant negative relationship between SR and distance to the nearest  
263 mainland was only observed for the Pacific islands ( $p = 0.02$ ).

264 The effect of island size was driven by zoonotic and vector-borne pathogens in both  
265 Caribbean and Pacific islands and, for Pacific islands only, by protozoans and helminths  
266 (Table 2). For both Caribbean and Pacific islands, we did not find significant differences  
267 across pathogens categories in the relationship between SR and distance to the nearest  
268 mainland.

269

### 270 *Species richness relationships with human population size and density*

271 Although results for the entire sample support the hypothesized positive effect of  
272 human population on SR, the relationship was not driven by human-only pathogens (slope  
273 coefficients for Human Only, Multi-Host and Zoonotic pathogens, respectively:  $6.583 \times 10^{-3}$ ;  
274  $1.737 \times 10^{-2}$  and  $3.536 \times 10^{-2}$ ; ANCOVA- $p < 10^{-3}$ ).

275 Together, our findings suggest the area of an island is more important than the  
276 population size of potential human hosts living there. Larger islands support more people ( $r =$   
277  $0.767$ , and more people support more species of pathogens ( $y = 1.742 \times 10^{-2} \cdot x + 1.986$ ,  
278  $R^2_{adj} = 0.538$ ,  $p < 10^{-3}$ ) (see Appendix S2). However, this relationship is largely a function of the  
279 relationship for more-populous island nations, corresponding to a “break” in the regression at  
280 a population of  $\sim 10^5$  and thus perhaps reflecting a threshold of urbanization or more-general  
281 intensification. Tellingly, though, the relationship between human population density and  
282 pathogen SR is relatively smooth and weak ( $y = 8.769 \times 10^{-3} \cdot x + 2.060$ ,  $R^2_{adj} = 0.045$ ,  $p = 0.049$ )  
283 (see Appendix S2), suggesting that human population size and pathogen SR are both  
284 responding to factors that vary with island area, such as environmental energy supply or the  
285 diversity of potential habitats.

286

## 287 Discussion

288 We have shown here that the distribution of known human pathogens on islands  
289 follows the main predictions of MacArthur and Wilson's equilibrium theory of island  
290 biogeography (MacArthur & Wilson, 1967): pathogen species richness increases with island  
291 area and decreases with distance to the nearest mainland. However, the relative influence of  
292 area is much greater than that of isolation, and the extent and strength of the associations  
293 vary by host requirement, transmission pathway, and pathogen taxonomy. Importantly,  
294 pathogens whose primary hosts are not humans are more strongly affected by island  
295 biogeography than are those that primarily afflict people.

296 A limitation of this study is the relatively small sample size, an inherent constraint of  
297 focusing on a relatively small subset of the larger GIDEON dataset. The resulting lack of  
298 statistical power did not allow us to account simultaneously for different categorical factors or  
299 to take into account other factors previously identified as important drivers of pathogen  
300 richness, such as climate (Guernier *et al.*, 2004; Dunn *et al.*, 2010). However, our purpose  
301 was not to identify and assess the relative influence of a large set of variables but rather to  
302 test how well an influential biogeographical theory describes a pattern of contemporary  
303 human ecology. Conducting a complementary analysis on regional sub-datasets (Caribbean  
304 and Pacific islands) was a way to control for shared characteristics of islands from the larger  
305 sample, such as latitude and regional biotic influences. The fact that the results of these  
306 regional analyses were similar to those for the whole dataset supports the validity of the  
307 relationships we found.

308 Another limitation is that GIDEON is an evidence-based database, so the data could,  
309 potentially, reflect a reporting bias. Indeed, wider sampling or research efforts on larger or  
310 less-isolated islands could contribute to the results described here. Hypothetically, although  
311 such a reporting bias for this island dataset could influence our findings, it is unlikely this bias  
312 would produce the patterns we observed across pathogen categories. Furthermore,  
313 healthcare expenditure is a poor predictor of human pathogen SR at the country-level (Dunn  
314 *et al.*, 2010) even if it does predict infectious pathogen prevalence. Thus, our results are  
315 likely independent of any reporting effect.

316 According to the equilibrium theory of island biogeography, the positive relationship  
317 we found between human pathogen species richness and island area is due to lower  
318 extinction rates on larger islands. Larger islands contain larger habitat areas and a likelihood  
319 of greater habitat diversity. To human pathogens, larger habitat areas should support more  
320 host individuals, including more humans, and greater habitat diversity should support more  
321 species of alternative hosts. Indeed, nations with more people and more species of birds and  
322 mammals support more species of human pathogens (Dunn *et al.*, 2010), and habitat  
323 diversity drives the diversity of bacterial assemblages generally (Nemergut *et al.*, 2011). So  
324 the patterns we observed for human pathogens could simply mirror: (i) a species-human  
325 population relationship, in which the human population serves as the "area" that pathogen  
326 species occupy, and/or (ii) the species-area relationships for alternative insular host species.

327 However, the much greater effect of area on the species richness of zoonotics than  
328 on multi-host and human-only pathogens (Fig 2. A) suggests a strong mechanistic role for  
329 alternative insular hosts. In short, island animals for which pathogens are primary hosts help

330 drive the island biogeography of human disease. This is not surprising, because these  
331 animals are much more restricted in their ability to travel than are contemporary humans.  
332 This finding also corresponds with the strong relationship between pathogen diversity and  
333 bird and mammal species diversity (Dunn *et al.*, 2010) and the findings of a broader analysis  
334 that included continental nations (Smith *et al.*, 2007).

335 Greater immigration rates also likely moderate the effect of area on human-only  
336 pathogens through the rescue effect, a refinement of MacArthur and Wilson's theory. The  
337 rescue effect (Brown & Kodric-Brown, 1977) is the effect of immigration on extinction, which  
338 in the original theory was solely a function of island size. By continually bringing pathogens  
339 with us to islands when we migrate or travel, we "rescue" some pathogen species that might  
340 otherwise die out on smaller islands (i.e. when the number of susceptible individuals drops  
341 below a threshold necessary to sustain the disease, i.e. the Critical Community Size) (Rohani  
342 *et al.*, 1999).

343 Together, these features specific to human pathogens differentiate island microbes  
344 from other insular taxa. A positive relationship between island size and bacterial diversity  
345 holds in engineered systems (van der Gast *et al.*, 2005), supporting the generality of this  
346 biogeographic pattern and the importance of species-area/volume effects among microbes  
347 generally (Green & Bohannan, 2006). Yet mass-related limitations on active dispersal and  
348 exceptional rates of diversification (Martiny *et al.*, 2006), may explain the exceptionally low  
349 effect of area we observed: the value of the coefficient linking species richness and island  
350 surface (z-value) for insular human parasites and pathogens was about an order of  
351 magnitude lower than those of insular macro-organisms (Table 3).

352 The negative relationship we found between pathogen species richness and distance  
353 to the mainland is a function of varying immigration rates in MacArthur and Wilson's theory.  
354 Several factors may explain the relative weakness we found in the influence of isolation  
355 versus that of area. In the original theory, distance is that separating different islands to the  
356 same continental shore, viewed as the source of the same set of species. However, species  
357 considered here are pathogens of *Homo sapiens*, whose large-scale movement capacities  
358 have increased continuously, especially during the past five centuries (Smith & Guégan,  
359 2010). This increase in connectedness, which has profoundly lessened effective isolation,  
360 likely explains the much smaller effect of distance. One way to test this idea would be to  
361 explore the relationship between island pathogen diversity and transport connectedness  
362 (Colizza *et al.*, 2006).

363 The finding of a greater role for area than for human host population and for distance  
364 can be seen as supporting the Baas-Becking' hypothesis, which posits that, regarding  
365 microbes, "everything is everywhere, but the environment selects" (Baas-Becking, 1934) and  
366 a corresponding non-stochastic view of microbial community assembly (Barberán *et al.*,  
367 2014). In the case of zoonotics, the environment of interest is composed of non-human hosts  
368 and their habitats are not everywhere.

369 This work has several practical, broad-scale implications. The fact that our travel and  
370 trade swamps isolation so profoundly means there will remain few disease-free islands.  
371 However, the importance of area in supporting populations of vectors and alternative hosts  
372 means the proportion of island diseases that are zoonotic and vector-transmitted will tend to

373 decline with decreasing island size, with implications for public health management efforts.  
374 These implications should apply to current islands as well as to those, given time for  
375 equilibration, created or whose area or isolation is altered by rising sea levels. However,  
376 concluding that reducing or fragmentizing habitats is a viable public health strategy would  
377 misinterpret the broader lessons of ecology in the Anthropocene. The incursion of human  
378 populations into natural habitats is already associated with zoonotic outbreaks and  
379 emergence, and habitat loss would suppress biodiversity more broadly and have a  
380 disproportionate impact on larger taxa, such as mammals. As global biodiversity benefits  
381 human health and well-being in many ways, such a strategy would harm more than help.

382         Our results demonstrate how classic island biogeography theory applies to human  
383 pathogens, and our findings support the spirit of theoretical insight as much as the  
384 substance. Even if infectious diseases have been widely globalized because of large-scale  
385 human movements, area and isolation still affect macroscopic disease patterns. And the  
386 ways in which the results seem to show weak support—in the relative effect of isolation on  
387 human-only pathogens—highlights the importance of the underlying process, immigration,  
388 which is so strongly constrained by isolation. Globalization effectively increases pathogen  
389 immigration rates, reducing the historic barrier of isolation. Just as humans both follow and  
390 flout ecological patterns common to species generally (Burnside *et al.*, 2012), so do the  
391 parasites and pathogens that afflict us. And just as some aspects of biogeography are  
392 common to life generally, others may be unique to microbes. As it did for our understanding  
393 of assemblages of plant and animal communities, we hope that this test of “equilibrium  
394 theory” will be a stepping stone in the understanding of causal drivers behind global trends in  
395 human infectious disease and in the broader quest to understand the geography of life.

396  
397

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399

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405

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- 525

526

## 527 BIOSKETCH

528 Kévin Jean holds a PhD in Epidemiology and was initially trained in Ecology and Evolution.  
529 His work focuses on epidemiology and prevention of infectious diseases, with a constant  
530 effort to address comprehensively the biological, ecological, behavioral and social  
531 determinants of infectious transmission. For more information:  
532 <https://sites.google.com/site/kvnjean/home>

533 William Burnside, a postdoctoral fellow at the National Socio-Environmental Synthesis  
534 Center (SESYNC), applies methods from macroecology and functional ecology to test the  
535 ability of ecological theory to inform our understanding of human-environment systems.

536 Jean-François Guégan is a senior research scientist at the French Institute for Research in  
537 Developing Countries ([ww.ird.fr](http://ww.ird.fr)) and also a scientific adviser for the ecoHEALTH initiative  
538 from the international programme FutureEarth from the U.N. As a disease ecologist, his  
539 research interests focus on macroecology of infectious diseases and their hosts, and the  
540 links between climate change and biodiversity and emerging infections.

541

## 542 SUPPORTING INFORMATION

543 **Appendix S1:** Geographical characteristics of the 66 islands nations considered in the  
544 analysis.

545 **Appendix S2:** Species richness relationships with human population size and density.

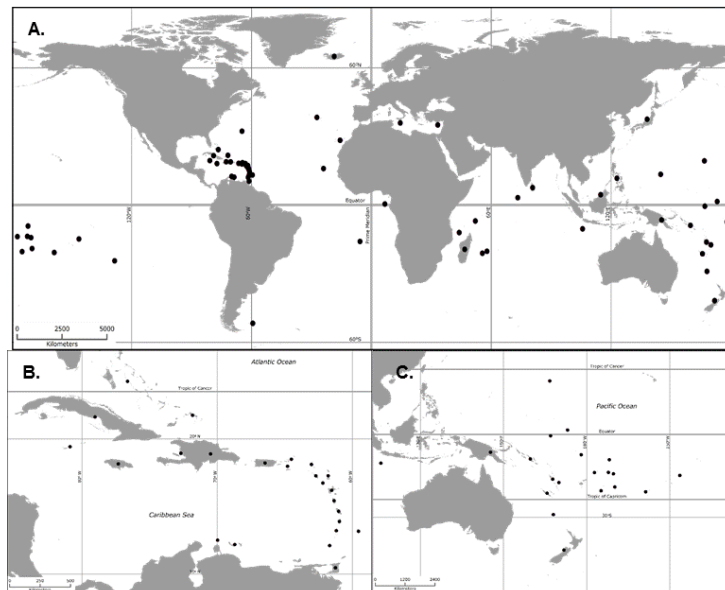
546 **Appendix S3:** Reproduced graphs with countries labeled.

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548

549 **Tables and Figures**

550 **Fig. 1:** Geographic location of the islands considered: **A)** whole dataset (n=66), **B)** Caribbean  
551 dataset (n=24) and **C)** Pacific dataset (n=21).



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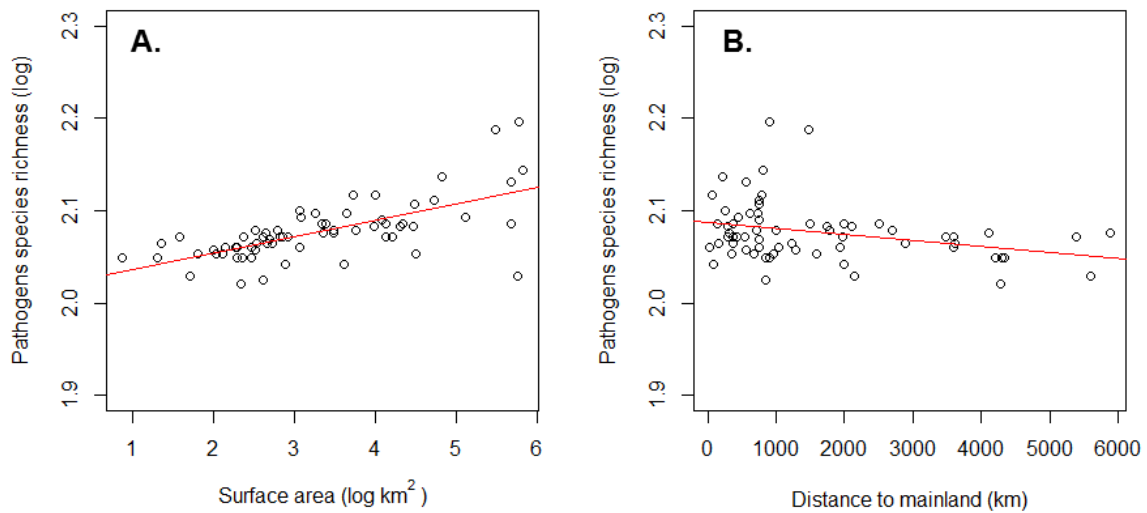
554

555 **Fig. 2:** Pathogen species richness (log number of species) plotted against: **A)** island surface  
556 area (log km<sup>2</sup>) and **B)** distance to the nearest mainland (km).

557 Linear regression parameters: **A)**  $y = 1.695 \times 10^{-2}x + 2.022$ ,  $R^2_{adj}=0.407$ ,  $p<0.0001$ ; **B)**  $y = -$   
558  $6.394 \times 10^{-6}x + 2.087$ ,  $R^2_{adj}=0.0766$ ,  $p=0.014$ . Total pathogen species considered:  $n=271$ .

559 Note that the influence of area is much stronger than that of distance ( $|1.695 \times 10^{-2}| \gg | -$   
560  $6.394 \times 10^{-6}|$ ). Appendix S3 includes these same graphs with the countries labeled.

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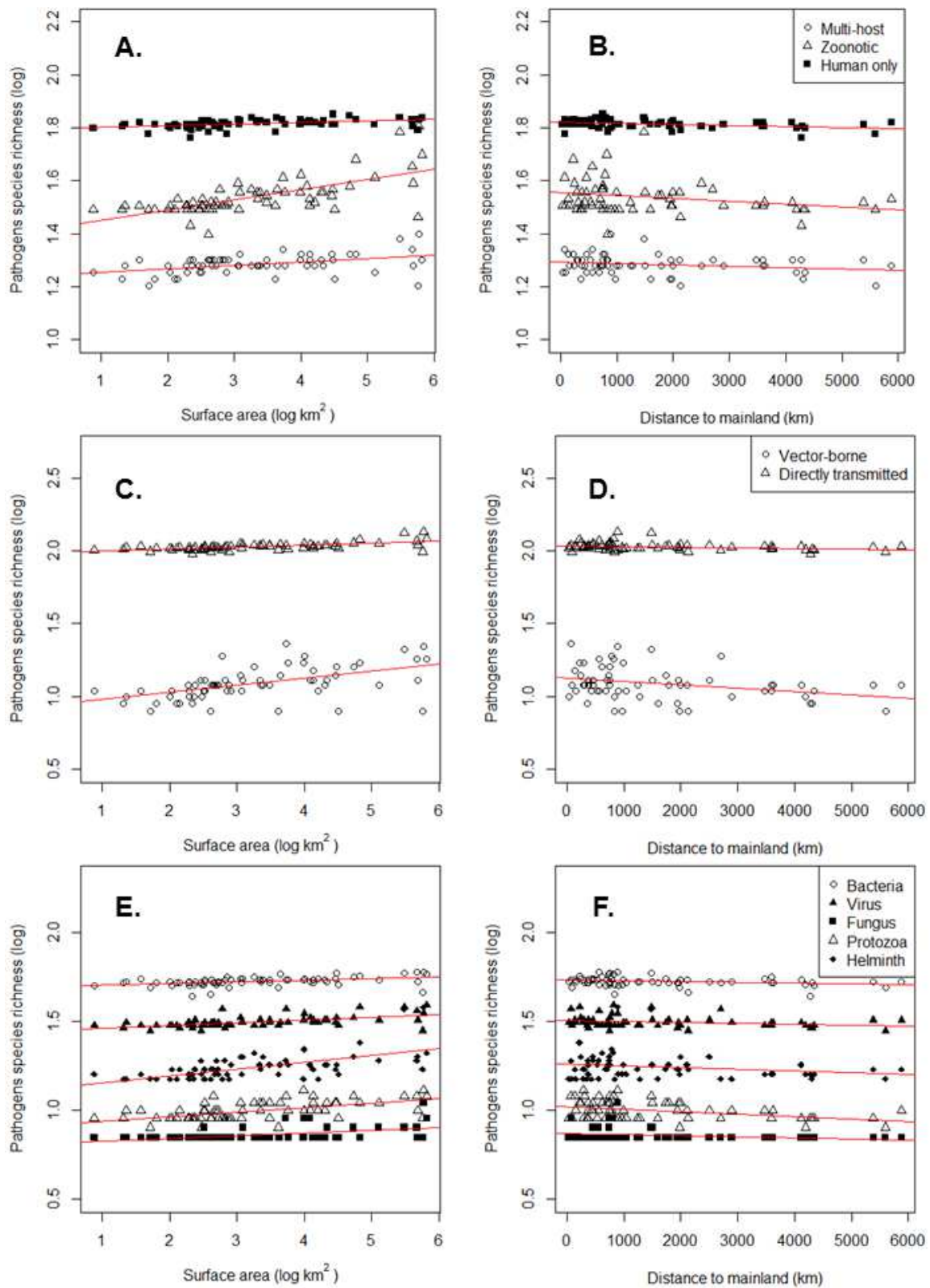
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571 **Fig. 3:** Pathogen species richness (log number of species) as a function of island surface  
 572 area (left) and distance to mainland (right) classified by host requirement (**A, B**), transmission  
 573 pathway (**C, D**), and taxonomy (**E, F**).



574

1 **Table 1: Results of univariate linear regressions of log number of pathogen species classified by host-requirement, transmission**  
2 **pathway, and taxonomy, as functions of a) island surface area (log) and b) distance to mainland in the total island sample (n=66).**  
3 **NB: The linear relationship between Species Richness and Island surface area expressed in logarithmic space corresponds to the**  
4 **classical power model of the species-area relationship, generally expressed as  $SR=cA^z$ , where  $A$  is the surface area,  $c$  is the intercept,**  
5 **and  $z$  in the linear coefficient, or slope.**

6

<i>Pathogen Species Richness classified by:</i>	a) Island surface area (log km <sup>2</sup> )					b) Distance to Mainland (km)				
	Slope (x10 <sup>-2</sup> )	Intercept	R <sup>2</sup> <sub>adj</sub>	p	ANCOVA p	Slope (x10 <sup>-6</sup> )	Intercept	R <sup>2</sup> <sub>adj</sub>	p	ANCOVA p
<b>Host-requirement</b>					<10 <sup>-3</sup>					0.354
Multi-host	1.21	1.25	0.178	<10 <sup>-3</sup>		-5.00	1.29	0.032	0.081	
Zoonotic	3.79	1.42	0.449	<10 <sup>-3</sup>		-10.99	1.56	0.043	0.051	
Human only	0.58	1.80	0.185	<10 <sup>-3</sup>		-4.10	1.82	0.13	0.002	
<b>Transmission pathway</b>					<10 <sup>-3</sup>					0.034
Vector-borne	4.69	0.94	0.277	<10 <sup>-3</sup>		-23.10	1.13	0.089	0.009	
Directly transmitted	1.32	1.99	0.392	<10 <sup>-3</sup>		-4.38	2.04	0.051	0.038	
<b>Taxonomy</b>					<10 <sup>-3</sup>					0.343
Bacteria	0.89	1.70	0.187	<10 <sup>-3</sup>		-4.62	1.73	0.063	0.024	
Viruses	1.49	1.45	0.337	<10 <sup>-3</sup>		-4.72	1.50	0.037	0.065	
Fungi	1.52	0.81	0.241	<10 <sup>-3</sup>		-5.79	0.87	0.039	0.061	
Protozoans	3.73	1.12	0.363	<10 <sup>-3</sup>		-13.88	1.02	0.154	0.001	
Helminths	2.42	0.92	0.329	<10 <sup>-3</sup>		-9.95	1.26	0.025	0.110	

**Table 2: Results of univariate linear regressions of log number of pathogen species classified by host-requirement, transmission pathway, and taxonomy as functions of a) island surface area (log) and b) distance to the nearest mainland for two regional island subsets, Caribbean islands (n=24) and Pacific islands (n=21).**

**NB: The linear relationship between Species Richness and Island surface area expressed in logarithmic space corresponds to the classical power model of the species-area relationship, generally expressed as  $SR=cA^z$ , where  $A$  is the surface area,  $c$  is the intercept, and  $z$  in the linear coefficient, or slope.**

	a) Island surface area (log km <sup>2</sup> )					b) Distance to Mainland				
	Slope (x10 <sup>-2</sup> )	Intercept	R <sup>2</sup>	p	ANCOVA p	Slope (x10 <sup>-6</sup> )	Intercept	R <sup>2</sup>	p	ANCOVA p
<b>Caribbean Islands (n=24)</b>										
<b>All pathogens</b>	2.12	2.01	0.54	<10 <sup>-3</sup>	-	-5.19	2.08	0.00	0.782	-
<i>Pathogen Species Richness classified by:</i>										
<b>Host-requirement</b>					<10 <sup>-3</sup>					<b>0.797</b>
Multi-host	0.97	1.26	0.11	0.112		1.68	1.29	0.00	0.93	
Zoonotic	4.51	1.38	0.56	<10 <sup>-3</sup>		-20.6	1.54	0.01	0.60	
Human only	1.20	1.78	0.35	0.002		0.21	1.82	0.00	0.99	
<b>Transmission pathway</b>					<b>0.038</b>					<b>0.581</b>
Vector-borne	6.03	0.91	0.28	0.007		-40.8	1.12	0.01	0.58	
Directly transmitted	1.62	1.97	0.61	<10 <sup>-3</sup>		0.54	2.02	0.00	0.97	
<b>Taxonomy</b>					<b>0.446</b>					<b>0.707</b>
Bacteria	1.66	1.67	0.36	0.002		-11.5	1.73	0.02	0.52	
Viruses	1.93	1.43	0.40	<10 <sup>-3</sup>		-13.5	1.50	0.02	0.50	
Fungi	2.50	0.79	0.29	0.006		-2.94	0.87	0.00	0.92	
Protozoans	2.92	1.15	0.22	0.020		35.1	1.22	0.03	0.39	
Helminths	3.51	0.88	0.54	<10 <sup>-3</sup>		-15.4	1.00	0.01	0.62	
<b>Pacific Islands (n=21)</b>										
<b>All pathogens</b>	1.21	2.03	0.56	<10 <sup>-3</sup>	-	-7.10	2.09	0.24	0.02	-
<i>Pathogen Species Richness classified by:</i>										
<b>Host-requirement</b>					<b>0.001</b>					<b>0.352</b>
Multi-host	1.39	1.24	0.34	0.006		-4.87	1.29	<0.01	0.324	
Zoonotic	2.53	1.45	0.64	<10 <sup>-3</sup>		-12.80	1.56	0.20	0.041	
Human only	0.43	1.80	0.16	0.078		-4.56	1.82	0.22	0.033	
<b>Transmission pathway</b>					<10 <sup>-3</sup>					<b>0.246</b>
Vector-borne	4.49	0.92	0.61	<10 <sup>-3</sup>		-18.90	1.11	0.13	0.103	
Directly transmitted	0.84	2.00	0.46	<10 <sup>-3</sup>		-5.65	2.04	0.26	0.019	
<b>Taxonomy</b>					<10 <sup>-3</sup>					<b>0.191</b>
Bacteria	0.79	1.70	0.19	0.046		-6.74	1.74	0.18	0.058	
Viruses	1.16	1.46	0.54	<10 <sup>-3</sup>		-5.97	1.51	0.18	0.056	
Fungi	0.62	0.83	0.25	0.021		-5.10	0.87	0.21	0.035	
Protozoans	2.26	1.15	0.80	<10 <sup>-3</sup>		-5.50	1.24	0.06	0.289	
Helminths	2.21	0.92	0.47	<10 <sup>-3</sup>		-16.90	1.04	0.34	0.005	

**Table 3 : Summary of z-values documented among different organisms for Island Species-Area Relationships.**

**z** corresponds to the coefficient of the power model  $SR=cA^z$ , where SR is the species richness and A the surface area. Equivalently, z corresponds to the slope of the log-linear relationship linking SR and A.

<b>Organisms</b>	<b>z-value</b>	<b>reference</b>
Plants	0.355	Triantis <i>et al.</i> , 2012
Invertebrates	0.323	Triantis <i>et al.</i> , 2012
Vertebrates	0.284	Triantis <i>et al.</i> , 2012
Bacteria	0.104 - 0.295	Green & Bohannan, 2006
Phytoplankton	0.134	Smith <i>et al.</i> , 2005
Fungi	0.20 - 0.23	Peay <i>et al.</i> , 2007
Human parasites and pathogens		<i>Present study</i>
<b>Overall</b>	<b>0.017</b>	
Bacteria	0.009	
Viruses	0.015	
Fungi	0.015	
Protozoans	0.037	
Helminths	0.024	