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► **To cite this version:**

Kévin Jean, William R. Burnside, Lynn Carlson, Katherine Smith, Jean-François Guégan. An equilibrium theory signature in the island biogeography of human parasites and pathogens. *Global Ecology and Biogeography*, 2016, 25 (1), pp.107-116. 10.1111/geb.12393 . inserm-01351791

HAL Id: inserm-01351791

<https://inserm.hal.science/inserm-01351791>

Submitted on 4 Aug 2016

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

3

4 **Running title:** The island biogeography of human pathogens

5 **Key-words:** Infectious diseases, human, island biogeography, pathogen diversity, species-area
6 relationship, disease ecology

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44 **Words (abstract):** 300

45 **Words (main body):** 3785

46 **Number of references:** 36

47

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 21st 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: 5.768×10^{-3} , Multi-Host pathogens: 1.210×10^{-2} ,
248 Zoonotic pathogens: 3.788×10^{-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: 8.932×10^{-3} ,
251 Viruses: 1.486×10^{-2} , Fungi: 1.522×10^{-2} , Protozoans: 3.733×10^{-2} , Helminths: 2.416×10^{-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: 6.583×10^{-3} ;
274 1.737×10^{-2} and 3.536×10^{-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 $R^2_{\text{adj}} = 0.538$, $p < 10^{-3}$) (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 ($y = 8.769 \times 10^{-3} \cdot x + 2.060$, $R^2_{\text{adj}} = 0.045$, $p = 0.049$)
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

398 **ACKNOWLEDGEMENT**

399

400 We are thankful to François Guilhaumon from Institut de Recherche pour le Développement
401 (IRD) and Dov Sax from Brown University, for useful comments on an earlier draft. This
402 research has benefited from an “Investissement d’Avenir” grant managed by Agence
403 Nationale de la Recherche (CEBA ANR-LABX-10-2501). In addition, JFG received support
404 from both IRD and CNRS.

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
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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
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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.

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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.

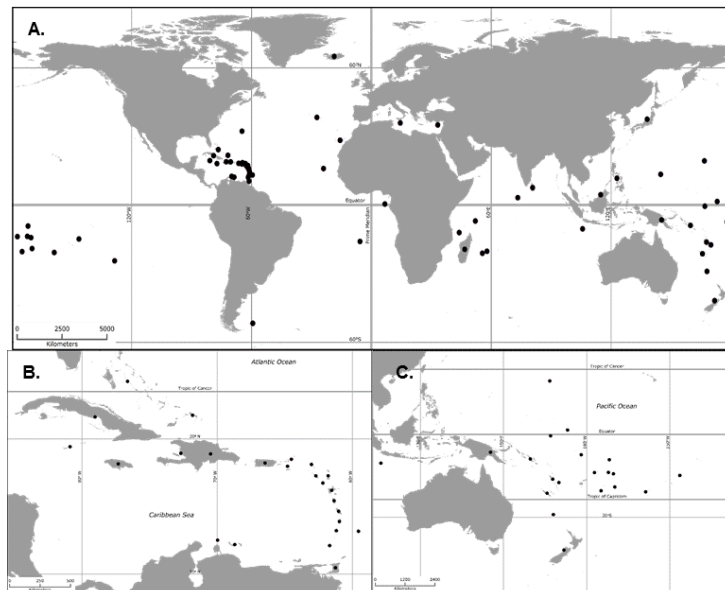
546 **Appendix S3:** Reproduced graphs with countries labeled.

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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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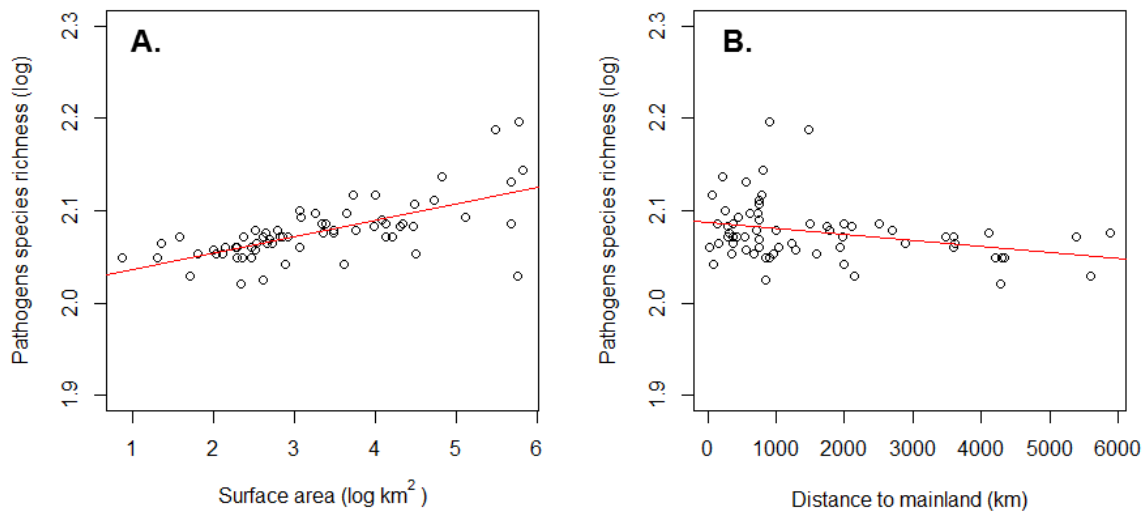
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555 **Fig. 2:** Pathogen species richness (log number of species) plotted against: **A)** island surface
556 area (log km²) and **B)** distance to the nearest mainland (km).

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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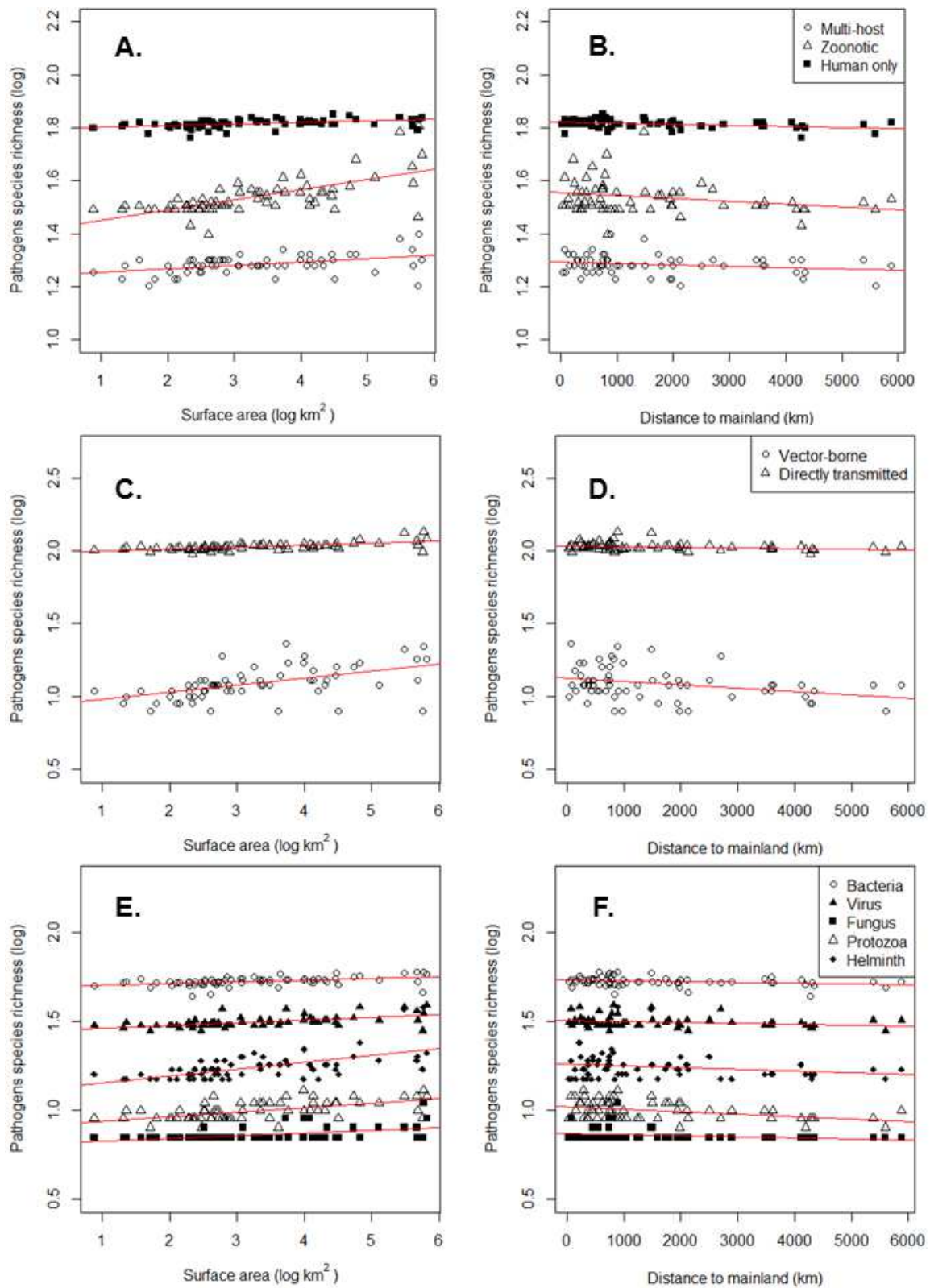
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571 **Fig. 3:** Pathogen species richness (log number of species) as a function of island surface
 572 area (left) and distance to mainland (right) classified by host requirement (**A, B**), transmission
 573 pathway (**C, D**), and taxonomy (**E, 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 ²)					b) Distance to Mainland (km)				
	Slope (x10 ⁻²)	Intercept	R ² _{adj}	p	ANCOVA p	Slope (x10 ⁻⁶)	Intercept	R ² _{adj}	p	ANCOVA p
Host-requirement					<10 ⁻³					0.354
Multi-host	1.21	1.25	0.178	<10 ⁻³		-5.00	1.29	0.032	0.081	
Zoonotic	3.79	1.42	0.449	<10 ⁻³		-10.99	1.56	0.043	0.051	
Human only	0.58	1.80	0.185	<10 ⁻³		-4.10	1.82	0.13	0.002	
Transmission pathway					<10 ⁻³					0.034
Vector-borne	4.69	0.94	0.277	<10 ⁻³		-23.10	1.13	0.089	0.009	
Directly transmitted	1.32	1.99	0.392	<10 ⁻³		-4.38	2.04	0.051	0.038	
Taxonomy					<10 ⁻³					0.343
Bacteria	0.89	1.70	0.187	<10 ⁻³		-4.62	1.73	0.063	0.024	
Viruses	1.49	1.45	0.337	<10 ⁻³		-4.72	1.50	0.037	0.065	
Fungi	1.52	0.81	0.241	<10 ⁻³		-5.79	0.87	0.039	0.061	
Protozoans	3.73	1.12	0.363	<10 ⁻³		-13.88	1.02	0.154	0.001	
Helminths	2.42	0.92	0.329	<10 ⁻³		-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 ²)					b) Distance to Mainland				
	Slope (x10 ⁻²)	Intercept	R ²	p	ANCOVA p	Slope (x10 ⁻⁶)	Intercept	R ²	p	ANCOVA p
Caribbean Islands (n=24)										
All pathogens	2.12	2.01	0.54	<10 ⁻³	-	-5.19	2.08	0.00	0.782	-
<i>Pathogen Species Richness classified by:</i>										
Host-requirement					<10 ⁻³					0.797
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 ⁻³		-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	
Transmission pathway					0.038					0.581
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 ⁻³		0.54	2.02	0.00	0.97	
Taxonomy					0.446					0.707
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 ⁻³		-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 ⁻³		-15.4	1.00	0.01	0.62	
Pacific Islands (n=21)										
All pathogens	1.21	2.03	0.56	<10 ⁻³	-	-7.10	2.09	0.24	0.02	-
<i>Pathogen Species Richness classified by:</i>										
Host-requirement					0.001					0.352
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 ⁻³		-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	
Transmission pathway					<10 ⁻³					0.246
Vector-borne	4.49	0.92	0.61	<10 ⁻³		-18.90	1.11	0.13	0.103	
Directly transmitted	0.84	2.00	0.46	<10 ⁻³		-5.65	2.04	0.26	0.019	
Taxonomy					<10 ⁻³					0.191
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 ⁻³		-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 ⁻³		-5.50	1.24	0.06	0.289	
Helminths	2.21	0.92	0.47	<10 ⁻³		-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.

Organisms	z-value	reference
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>
Overall	0.017	
Bacteria	0.009	
Viruses	0.015	
Fungi	0.015	
Protozoans	0.037	
Helminths	0.024	