1 **ANTHROPOGENIC DISTURBANCE INCREASES DISEASE EMERGENCE RISK**

2 **THROUGH PREDICTABLE CHANGES IN PARASITE COMMUNITY STRUCTURE**

3 Guha Dharmarajan,[1,2*†] Pooja Gupta,[1,2] C. K. Vishnudas[3] and V. V. Robin[3†]

4 [1]Savannah River Ecology Laboratory, University of Georgia, Aiken, SC

5 [2]Indian Institute of Science Education and Research Kolkata, Mohanpur, West Bengal, India

6 [3]Indian Institute of Science Education and Research Tirupati, Tirupati, Andhra Pradesh, India

7 *Correspondence: E-mail: guha@srel.uga.edu

8 [†]Joint senior authors

9 ORCID: GD, 0000-0001-8500-0429; PG, 0000-0002-4241-8208; VVR, 0000-0003-3109-5498

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18 **CORRESPONDENCE:**

19 Guha Dharmarajan, Ph.D.
20 University of Georgia
21 Savannah River Ecology Laboratory
22 SRS, Building 737-A, Aiken, SC 29808
23 Phone: 803-640-0361; Fax: 803-640-0361
24 Email: guha@uga.edu
25

ABSTRACT

Niche theory predicts specialists will be more sensitive to environmental perturbation compared to generalists, a hypothesis receiving broad support in free-living species. Based on their niche breadth, parasites can also be classified as specialists and generalists, with specialists infecting only a few and generalists a diverse array of host species. Here, using avian haemosporidian parasites infecting wild bird populations inhabiting the Western Ghats, India as a model system, we elucidate how climate, habitat and human disturbance affects parasite prevalence both directly and indirectly via their effects on host diversity. Our data demonstrates that anthropogenic disturbance acts to reduce the prevalence of specialist parasite lineages, while increasing that of generalist lineages. Thus, as in free-living species, disturbance favors parasite communities dominated by generalist vs. specialist species. Because generalist parasites are more likely to cause emerging infectious diseases, such biotic homogenization of parasite communities could increase disease emergence risk in the Anthropocene.

INTRODUCTION

The Anthropocene has been characterized by large, and often rapid, alterations of natural environments driven by human-mediated factors acting at various scales – from local habitat modification to global climate change (Corlett 2015; Laurance 2019). Many species have been unable to adapt sufficiently to cope with the such anthropogenic changes to their environment leading to species loss at local and global scales (Young *et al.* 2016; Radchuk *et al.* 2019; Turvey & Crees 2019). However, community disassembly is not a random process, with species loss often following certain general principles with specialists being more sensitive to environmental perturbations (e.g., habitat loss and climate change) compared to generalists (Swihart *et al.* 2003; Kellermann *et al.* 2009; Slatyer *et al.* 2013), leading to the biotic homogenization of natural

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53   communities globally (McKinney & Lockwood 1999; Olden 2006; Olden & Rooney 2006;

54   Clavel *et al.* 2010; Li *et al.* 2020). While not universal (Williams *et al.* 2006; Colles *et al.* 2009),

55   the increased sensitivity of specialists to endangerment has been demonstrated in many taxa (Safi

56   & Kerth 2004; Kotiaho *et al.* 2005; Shultz *et al.* 2005; Walker 2006; Boyles & Storm 2007; Essl

57   *et al.* 2009; Saupe *et al.* 2015; White & Bennett 2015; Liang *et al.* 2019; Colléony & Shwartz

58   2020). Biotic homogenization is of particular concern because the generalist species that thrive in

59   human-dominated landscapes are also more likely to harbor greater diversity of parasites,

60   including those can infect humans (Gibb *et al.* 2020; Ostfeld & Keesing 2020). Thus, human-

61   mediated landscape modification can affect disease dynamics by favoring host communities that

62   are dominated by generalist species (Gibb *et al.* 2020; Ostfeld & Keesing 2020). However, the

63   effects of such human pressures on parasite community structure remains unclear.

64   Like free-living species, parasites can also be categorized as specialists and generalists,

65   based on their niche breadth, with specialist parasite lineages infecting only one or few related

66   host species, and generalists infecting broad diversity of hosts (Cooper *et al.* 2012; Gupta *et al.*

67   2019). This dichotomization of parasites is particularly important from the perspective of human

68   and wildlife health because generalist parasites are more capable of switching to new hosts, and

69   are thus more likely to be associated with emerging infectious diseases (EIDs) (Timms & Read

70   1999; Ewen *et al.* 2012; Hatcher *et al.* 2012; Farrell *et al.* 2013; Johnson *et al.* 2015a). While

71   there is some evidence that generalist parasites are less prone to extinction (Woolhouse *et al.*

72   2001; Cooper *et al.* 2012), and it has been hypothesized that generalist parasites are more likely

73   to adapt to relatively common generalist hosts (Ostfeld & Keesing 2020), the question of

74   whether human-mediated changes to the environment favor parasite communities dominated by

75   generalist vs. specialist species, as in the case of free-living communities, remains an open one.

76 Here we test this question using avian haemosporidian parasites as a model system. Answering

77 this question is critical because generalist parasites are capable of rapidly switching to new host

78 species (Wells & Clark 2019), and thus the biotic homogenization of parasite communities will

79 increase the risk disease emergence.

80   Avian haemosporidian parasites are vector-borne pathogens of birds belonging to several

81 genera including *Haemoproteus* and *Plasmodium* (Valkiūnas 2005). We have previously shown

82 that in bird communities inhabiting an important biodiversity hotspot – the Western Ghats,

83 Southern India (Fig. 1a) – these genera differ in host breadth, with genetic lineages of

84 *Haemoproteus* being host-specialists while *Plasmodium* lineages are host-generalists (Gupta *et*

85 *al.* 2019), as seen in other bird communities (Fallon *et al.* 2005). Indeed, *Plasmodium* infections,

86 unlike *Haemoproteus*, have been responsible for epidemic mortalities in wild bird populations on

87 some oceanic islands (Warner 1968; Valkiūnas 2005; Niebuhr *et al.* 2016), because being

88 generalists *Plasmodium* lineages are more likely to emerge in novel host communities (Gupta *et*

89 *al.* 2019). Here, using data from a sample of >1000 birds of 28 species inhabiting the Western

90 Ghats (Table S1), we show that, the parasite community structure is predictably influenced by

91 anthropogenic disturbance, after controlling for effects associated with the environment (i.e.,

92 climate and terrain) and host community structure. Specifically, our results indicate that

93 anthropogenic pressure acts to reduce the prevalence of specialist parasite lineages, while

94 increasing the prevalence of generalist lineages. Such biotic homogenization of parasite

95 communities is a novel mechanism that can contribute to the increased risk of disease emergence

96 in human-dominated landscapes, and thus has broad implications of human and animal health.

METHODS

Field and laboratory methods

The study area was located in the southern 600 km of the Western Ghats (Fig. 1a). Field

sampling was carried out between 2011-2013, across the four major geographical regions

separated by three biogeographic barriers—Chaliyar River valley, Palghat Gap, and Shencottah

Gap (Fig. 1a). For our analyses we used samples from 28 species of birds (N = 1172) captured

during the pre-monsoon season (January-May) at 42 sites (700-2500 m above sea level) across

our study area (Fig. 1a; Supplementary Table 1). Procedures for bird mist-netting and blood

collection have been described previously (Gupta *et al.* 2019). Parasite detection was carried out

by sequencing 478 bp of the Haemosporidian cytochrome-b gene using genomic DNA extracted

from individual bird blood samples. All lab and field methods followed those described

previously in Gupta *et al.* (2019).

Environmental and ecological variables

One of the main objectives of this study was to elucidate how climate, landscape and human

disturbance affected parasite prevalence, both directly and through their effects on host diversity.

Environmental data were downloaded from publicly available datasets. We obtained the

bioclimatic variables from http://chelsa-climate.org/bioclim/. Landscape variables included: (a)

Terrain: Elevation, slope and roughness extracted from elevation data

(http://www.earthenv.org/DEM) using the R package RASTER (HIJMANS *ET AL.* 2015), and water

flow-accumulation from https://www.hydrosheds.org/page/hydrorivers; (b) Canopy height:

http://lidarradar.jpl.nasa.gov; (c) Landcover data: Obtained from authors of a published dataset

(Roy *et al.* 2015). We then used the package RASTER to calculate the proportions of 10 major

habitat types (i.e., cropland, degraded habitat, grassland, gregarious forest, locale-specific forest,

120 mixed forest, plantation, settlements, shrub and savannah and water); (d) Anthropogenic

121 disturbance: Included distance from protected areas (calculated as the distance from the nearest

122 boundary with negative values falling inside and positive outside protected areas, respectively)

123 using data from http://datasets.wri.org/dataset/64b69c0fb0834351bd6c0ceb3744c5ad. We also

124 used a Indian population dataset (http://www.ciesin.columbia.edu/data/india-census-grids/) to

125 calculate an index of human proximity following Alexander et al. (Alexander & Wint 2013).

126 Apart from the environmental variables we also used two host ecological variables, including

127 host phylogenetic and functional diversity. Host diversity measures were calculated using the

128 alpha diversity function implemented in the R package BAT (Cardoso *et al.* 2014), with

129 phylogenetic diversity using phylogenetic distances between the bird species and functional

130 diversity using the Gower distance between species based on ecological traits associated with

131 feeding strata, sociality, habitat, and genetic connectivity. Details of all variables used at given in

132 Table S2.

133 **Statistical analyses**

134 All statistical analyses were carried out using R ver. 4.0.0 (The R Foundation for Statistical

135 Computing 2019), and all statistical tests and reported *P* values are two-sided. A complete list of

136 R packages used for analyses are given in Table S3. All analyses were carried out at the scale of

137 the individual site (Table S1). We pooled samples across multiple years because we found no

138 significant effect of year (*Haemoproteus*: $\chi^2 = 9.281$; P = 0.158; *Plasmodium*: $\chi^2 < 0.001$; P =

139 1.000). Our major objective was to test for differences in the factors affecting infection risk with

140 specialist vs. generalist parasites. Thus, we first classified each Haemosporidian lineage as a

141 specialist or generalist using a randomization test to evaluate if the diversity of hosts the lineage

142 was found to infect was lower or higher than random expectation, respectively (see

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143   Supplementary Methods). All further analyses were carried out only using the parasite lineages

144   that were clearly classified as specialists or generalists.

145   To test for the direct and indirect effects of the various predictor variables on parasite

146   infection risk we used a hybrid approach that combined random forests, a powerful machine

147   learning algorithm, and Structural Equation Models (SEMs). Thus, prior to building the SEM we

148   reduced the list of predictor variables using random forests models (RFMs) as suggested by

149   Duffy et al. (Duffy *et al.* 2016). Random forests models were implemented in the R package

150   RANGER (Wright & Ziegler 2017). All RFMs were run using 100,000 trees and we optimized the

151   model parameters (mTry, Min. node size, Split rule) using a 10-fold cross-validation procedure

152   implemented in the trainControl function from the R package CARET (Kuhn 2008). All RFMs

153   also included the number of birds sampled in each trap site as case weights to control for

154   potential sample size effects. We initially included all environmental variables for all RFMs, and

155   also added host-related variables for the parasite-related RFMs. Thus, initially environmental

156   factors (i.e., terrain, climate, habitat and disturbance) were included in the RFMs to predict host

157   ecological variables (host phylogenetic/functional diversity). In turn, parasite-related variables

158   (parasite phylogenetic diversity and infection risk) were assumed to be driven by direct effects of

159   environmental variables and the indirect effects of these variables through their effects on host

160   ecology. For each RFM, we first fit the full random forest model with all variables (Table S2),

161   and estimated the variable importance values (Wright & Ziegler 2017). We then used a forward

162   step-wise selection starting from the most important variable, and adding variables in decreasing

163   order of importance. At each step a variable was added if it's correlation with variables included

164   in the model until that step was ≤ 0.7 and if addition of the variable increased the coefficient of

165   determination (R^2) for the whole model. Model validation was done using three repetitions of a

166   10-fold cross validation procedure to maximize the r^2 value of the whole model using 75% of the

167   data to train the model and 25% to assess model prediction accuracy (Kuhn 2008). In order to

168   calculate the relative importance of each of the four main variable groups (i.e., terrain, climate,

169   habitat and ecology; Table S2) we calculated the weighted mean of the number of times

170   variables in each group were used to split trees in the random forest (see

171   https://stats.stackexchange.com/questions/92419).

172   To better understand the mechanisms driving the spatial variation in infection risk, we

173   modeled the direct and indirect effects between the variables identified by the RFMs using

174   structural equation models (SEMs), which are a powerful statistical approach wherein a set of

175   mutually interconnected equations are used to evaluate the causal relationships among a set of

176   variables (Shipley 2016). SEM analyses was implemented in the R package PIECEWISESEM

177   (Lefcheck & Freckleton 2015). Initially, we included as predictors all variables retained in the

178   final random forest models of host- and parasite-related variables (see details above). We

179   included both main and quadratic effects to model potential non-linear effects between the

180   variables, and z-transformed these variables to obtain standardized model coefficients. We then

181   sequentially dropped variables if dropping the variable from a specific model reduced the overall

182   Akaike Information Criterion (AIC) of the SEM model (Shipley 2013), and if the removed path

183   was not considered to be a significant missing path (Lefcheck & Freckleton 2015). Final model

184   acceptance was based on the Fisher's C statistic, with a model being accepted if the associated *P*-

185   value > 0.05 (Shipley 2016). Final path diagram was plotted using the R package diagram, and

186   the strength of specific paths was assessed visually using partial residual plots using the R

187   package VISREG (Breheny & Burchett 2017).

RESULTS

We identified a total of 47 unique parasite lineages infecting the birds in our study area, and found that 28 of 29 *Haemoproteus* lineages were host specialists and 2 of 18 *Plasmodium* lineages were host generalists, with the diversity of hosts infected by the remaining 17 lineages not differing significantly from random expectation (Table S4). All further analyses were carried out only using the 30 parasite lineages that were clearly classified as specialists or generalists. We found that the RFMs generally performed well, and the models for infection risk explained >70% of the variance amongst sampling sites in both parasites (*Haemoproteus:* $r^2= 0.794$; *Plasmodium*: $r^2= 0.707$; Fig. 1b and c; Table S5 and S6). Interestingly, in both *Haemoproteus* and *Plasmodium,* the majority of the total variance explained was due to host (61% and 49%, respectively) and landscape (21% and 26%, respectively) related factors, and least to climate (18% and 25%, respectively) (Fig. 1d). Our final RFMs revealed that there was considerable spatial variation in infection risk associated with *Haemoproteus* (Figure 1e) and *Plasmodium* (Figure 1f).

   To better understand the mechanisms driving the spatial variation in infection risk, we modeled the direct and indirect effects between the variables identified by the RFMs using an SEM. Our final SEM (i.e., reduced) model testing the hypothesized direct and indirect effects of our predictor variables fit the data well with no significant missing paths ($\Delta AIC_{REDUCED-FULL}$ = -875; Fisher's C = 130.651; DF = 166; *P* = 0.98; Table S7). The SEM revealed distinct paths affecting infection risk with specialist (*Haemoproteus*) vs. generalist (*Plasmodium*) parasites (Figure 2a and b; Table S8). Critically, human-mediated disturbance (distance to protected areas and human population proximity) affected infection risk in opposing ways through direct effects and mediation via host-related variables (Figure 2a and b).

211   To dissect the SEM results we focus on each of the primary variables in the model

212   sequentially. Our analyses revealed that that BIO05 (maximum temperature of warmest month)

213   had a negative effect on both functional host diversity ($\beta \pm SE = -0.261 \pm 0.120$; $P = 0.037$) and

214   phylogenetic host diversity ($\beta \pm SE = -0.306 \pm 0.139$; $P = 0.034$) (Fig 2a and b; Table S8). We

215   also found that anthropogenic disturbance, as measured by distance from protected areas, PA_{DIST}

216   reduced both functional ($\beta \pm SE = -0.229 \pm 0.113$; $P = 0.049$) and phylogenetic ($\beta \pm SE = -0.669$

217   ± 0.132; $P < 0.001$; Fig. 2c) host diversity. Thus, the biotic homogenization of host communities

218   occupying human-modified habitats is a characteristic of the bird communities in the Western

219   Ghats, as in other natural communities globally (McKinney & Lockwood 1999; Clavel *et al.*

220   2010).

221   Our analyses also revealed some interesting differences in factors affecting parasite

222   diversity. Thus, we found that BIO02 (mean diurnal temperature range) had significant non-

223   linear effects on the phylogenetic diversity of both *Haemoproteus* (Fig. 2d; Main $\beta \pm SE = -$

224   0.080 ± 0.199; $P = 0.689$, Quadratic $\beta \pm SE = -0.288 \pm 0.136$; $P < 0.041$) and *Plasmodium* (Fig.

225   2e; Main $\beta \pm SE = -0.528 \pm 0.162$; $P = 0.002$, Quadratic $\beta \pm SE = -0.657 \pm 0.118$; $P < 0.001$).

226   Alternatively, we found that BIO05 only affected the diversity of the generalist (*Plasmodium*)

227   parasites (Fig. 2f; Main $\beta \pm SE = -0.054 \pm 0.158$; $P = 0.736$, Quadratic $\beta \pm SE = -0.348 \pm 0.144$; P

228   < 0.021). Importantly, we found that host phylogenetic diversity had a positive effect on

229   phylogenetic diversity for specialist (*Haemoproteus*) parasites (Figure 2g; $\beta \pm SE = 0.683 \pm$

230   0.163; $P < 0.001$), but did not affect phylogenetic diversity for generalist (*Plasmodium*) parasites.

231   With respect to infection risk was we found a significant direct positive effect of parasite

232   diversity in the case of both *Haemoproteus* (Fig. 2h; $\beta \pm SE = 0.323 \pm 0.067$; $P < 0.001$) and

233   *Plasmodium* (Fig. 2i; $\beta \pm SE = 1.115 \pm 0.338$; $P = 0.001$). We also found that while the host

234 functional diversity had a significant direct negative effect on infection risk of the generalist

235 (*Plasmodium*) parasite (Fig. 2j; $\beta \pm SE = -0.789 \pm 0.289$; $P = 0.006$), but had no effect on

236 infection risk of the specialist (*Haemoproteus*) parasite. This difference between *Plasmodium*

237 and *Haemoproteus* could be because *Plasmodium*, as a generalist parasite, is likely affected by

238 host spatial proximity (and thus levels of niche overlap), but, as a specialist, *Haemoproteus* is

239 primarily affected by host phylogenetic relatedness (Gupta *et al.* 2019). Of most importance, we

240 also found opposite effects of anthropogenic disturbance on infection risk associated with

241 specialist and generalist parasites. In the case of the specialist parasite *Haemoproteus,* infection

242 risk was negatively affected by human population proximity (Figure 2k; $\beta \pm SE = -0.284 \pm$

243 0.111; $P = 0.011$) but a positive association was observed in the case of the generalist,

244 *Plasmodium* parasites (Figure 2l; $\beta \pm SE = 0.899 \pm 0.327$; $P = 0.006$).

245   Taking into consideration direct and indirect effects, distance to protected areas had a

246 significant negative total effect on infection risk for *Haemoproteus* [$\beta_{TOT} = -0.027$; 95 %

247 confidence interval (CI): -0.059, -0.013] but a significant positive total effect on infection risk

248 for *Plasmodium* risk ($\beta_{TOT} = 0.014$; CI: 0.002, 0.036) (Fig. 3). Additionally, human population

249 proximity had a significant negative total effect on infection risk for *Haemoproteus* ($\beta_{TOT} = -$

250 0.057; CI: -0.172, -0.018), but a significant positive effect on *Plasmodium* risk ($\beta_{TOT} = 0.061$; CI:

251 0.037, 0.116) (Figure 3). These opposing effects of anthropogenic pressure on infection risk in

252 the case of specialist vs. generalist parasites is, at least partially, mediated by the opposing

253 effects of host diversity on infection risk. Thus, while host diversity had a positive total effect on

254 *Haemoproteus* infection risk ($\beta_{TOT} = 0.048$; CI: 0.025, 0.09), it had a negative effect in the case

255 of *Plasmodium* ($\beta_{TOT} = -0.046$; CI: -0.077, -0. 014) (Fig. 3). While all the SEM analyses were

256 carried out at the scale of each host population, qualitatively similar results were obtained with

257   lineage-level analyses also (Table S9; Fig. S1). Specifically, we found that as diversity of hosts a

258   parasite lineage could infect increased (i.e., as parasites became more generalist), the effect of

259   host diversity on observed parasite prevalence changed from being positive (i.e., an amplification

260   effect) to being negative (i.e., a dilution effect). Similar patterns were also found in the case of

261   the standardized prevalence calculated based on the host community composition (i.e., expected

262   prevalence). Thus, our data indicate that host species that tend to be found in high diversity host

263   communities (i.e., specialist hosts) are less competent hosts for generalist parasites (i.e.,

264   *Plasmodium*) compared to those in low diversity host communities (i.e., generalist hosts).

265   **DISCUSSION**

266   Darwin's "Tangled Bank" (Darwin 1909) is one of the most common images brought to mind

267   when envisioning ecological systems comprised of a complex network of interacting species, and

268   the functional integrity of this tangled bank can severely compromised by the recent and rapid

269   modifications to global environments (Lau & terHorst 2020). Parasites form an integral part of

270   all ecological networks, and comprise a large, albeit relatively uncharacterized, portion of global

271   biodiversity (Dobson *et al.* 2008; Okamura *et al.* 2018). Thus, there is an increasing recognition

272   of the need to incorporate them into biodiversity conservation plans (Gomez & Nichols 2013;

273   Dougherty *et al.* 2016). Indeed, parasites – especially specialist parasites that are closely

274   dependent upon a very narrow set of host species – may be especially prone to extinction risk

275   (Galetti *et al.* 2018; Thompson *et al.* 2018; Moir & Brennan 2020), and ignoring such species

276   coextinctions can lead to gross underestimation of global extinction rates (Koh *et al.* 2004;

277   Strona & Bradshaw 2018). Here we show that the loss of host diversity will negatively impact

278   the diversity of specialist, but not generalist, parasites. Thus, our data supports the idea that

279   specialist parasites can be especially sensitive indicators of ecosystem health (Hudson *et al.*

280 2006), because these parasites are not only more sensitive to environmental perturbations but

281 also to co-extinction caused by loss of specific host species on which they depend (Dunn *et al.*

282 2009; Colwell *et al.* 2012). The increased endangerment of specialist vs. generalist species is not

283 unexpected, and has been shown to be common across numerous free-living taxa globally. Such

284 non-random loss of species has critical impacts on ecosystem function through the biotic

285 homogenization of ecological communities (McKinney & Lockwood 1999; Olden 2006; Olden

286 & Rooney 2006; Clavel *et al.* 2010; Li *et al.* 2020).

287   Defaunation in the Anthropocene can have dramatic impacts on multiple ecosystem

288 services including nutrient and energy cycling, and can also directly impact disease dynamics

289 (Dirzo *et al.* 2014). Specifically, biotic homogenization due to non-random species loss has been

290 shown to alter disease dynamics by changing the relative abundance of competent vs. non-

291 competent hosts in a community (Johnson & Thieltges 2010; Johnson *et al.* 2013; Johnson *et al.*

292 2015b). For example, it has been found that the most competent host species (i.e., the ones most

293 likely to be infected) also tend to be generalists (i.e., the ones most likely to persist in low

294 diversity communities) in many well-characterized host-parasite systems, such as West Nile

295 virus infections in bird communities and *Borrelia burgdorferi* infections in small mammal

296 communities (Ostfeld & Keesing 2012). In such systems increasing host diversity reduces host

297 community competence – host species abundance weighted by its competence to transmit

298 infection (Johnson *et al.* 2015b) – leading to a strong dilution effect. Here we have shown that

299 such a dilution effect is more likely to be evidenced in the case of generalist vs. specialist

300 parasites. Specifically, our data reveal that loss of host biodiversity can increase infection risk

301 associated with the generalist parasite (*Plasmodium*) because low diversity communities are

302 more likely contain host species that are competent to harbor and transmit the parasite (Fig. S1c

303   and d). Consequently, as in other disease systems, we show that host species-specific traits (e.g.,

304   levels of niche specialization) can jointly affect endangerment and infection risk (Ostfeld &

305   Keesing 2012; Johnson *et al.* 2015b).

306   Interestingly, while the effects of anthropogenic disturbance on the structure of free-

307   living communities has received much attention, relatively little attention has been paid to

308   understanding how such disturbances impact parasite community structure. While our analyses

309   do address this question, we lack data on the vectors that transmit the Haemosporidian parasites

310   in our study system. This remains a critical missing piece of the puzzle because *Haemoproteus*

311   and *Plasmodium* are transmitted by different vectors; biting midges (e.g., *Culicoides spp.*) and

312   mosquitoes (e.g., *Culex quinquefasciatus*), respectively (Valkiūnas 2005). Thus, differences in

313   the prevalence of these two parasites genera could be driven by underlying differences in the

314   effects of anthropogenic disturbance on their respective vector populations. However,

315   irrespective of the underlying mechanism, our data strongly supports the hypothesis that, as in

316   free-living species (McKinney & Lockwood 1999; Clavel *et al.* 2010), human-mediated

317   disturbance can favor parasite communities dominated by generalist vs. specialist species.

318   Clearly this finding has critical implications for human health also. For example, a recent study

319   has shown that species most likely to transmit pathogens to humans are also those that tend to

320   thrive in human-dominated landscapes (Gibb *et al.* 2020; Ostfeld & Keesing 2020). Thus,

321   anthropogenic disturbance can increase the risk of disease emergence in human populations. Our

322   results add another dimension to this idea, and reveals that anthropogenic disturbance also tends

323   to favor generalist vs. specialist pathogens. Because generalist parasites are more commonly

324   associated with emerging infectious diseases (Shaw *et al.* 2020), such biotic homogenization of

325   parasite communities could increase disease emergence risk in human-dominated landscapes.

326   To conclude, using data collected from >1000 birds belonging to 28 species across the

327   Western Ghats, we show that parasite community structure is strongly influenced by

328   anthropogenic disturbance and host community structure, after controlling for effects associated

329   with the environment (e.g., climate). Our data reveal that the effects of anthropogenic

330   disturbance favors parasite communities that are dominated by generalist vs. specialist species, in

331   keeping with expectations based on niche theory. These results have broad implications for our

332   understanding of disease dynamics in the Anthropocene. Specifically, the biotic homogenization

333   of parasite communities driven by anthropogenic disturbance has the potential to mediate some

334   ongoing debates in disease ecology. For example, high parasite diversity has been considered to

335   be a sign of a healthy ecosystem (Hudson *et al.* 2006), while at the same time disturbed

336   ecosystems are the ones with increased risk of disease emergence (Ostfeld & Keesing 2017;

337   Ostfeld & Keesing 2020). Our data resolves this apparent paradox by revealing that undisturbed

338   ecosystems are likely to have a high diversity of specialized parasites which are highly

339   susceptible to being lost because they can only infect one or a few related host species.

340   Alternatively, disturbed ecosystems tend to primarily retain generalist parasites, which are more

341   robust to disturbance because they can infect multiple species, a characteristic which also

342   increases their ability to cause EIDs. These results have critical implications for public health

343   policy because they provide a clear mechanistic understanding of why disease emergence risk is

344   highest in areas facing rapid human-mediated landscape modifications (Allen *et al.* 2017; Gibb *et*

345   *al.* 2020; Ostfeld & Keesing 2020).

346 **ETHICAL APPROVAL**

347 This research was carried out in accordance with bird sampling permits granted by the Forest

348 Departments of Kerala and Tamil Nadu, and institutional animal ethics clearance from the

349 National Centre for Biological Sciences

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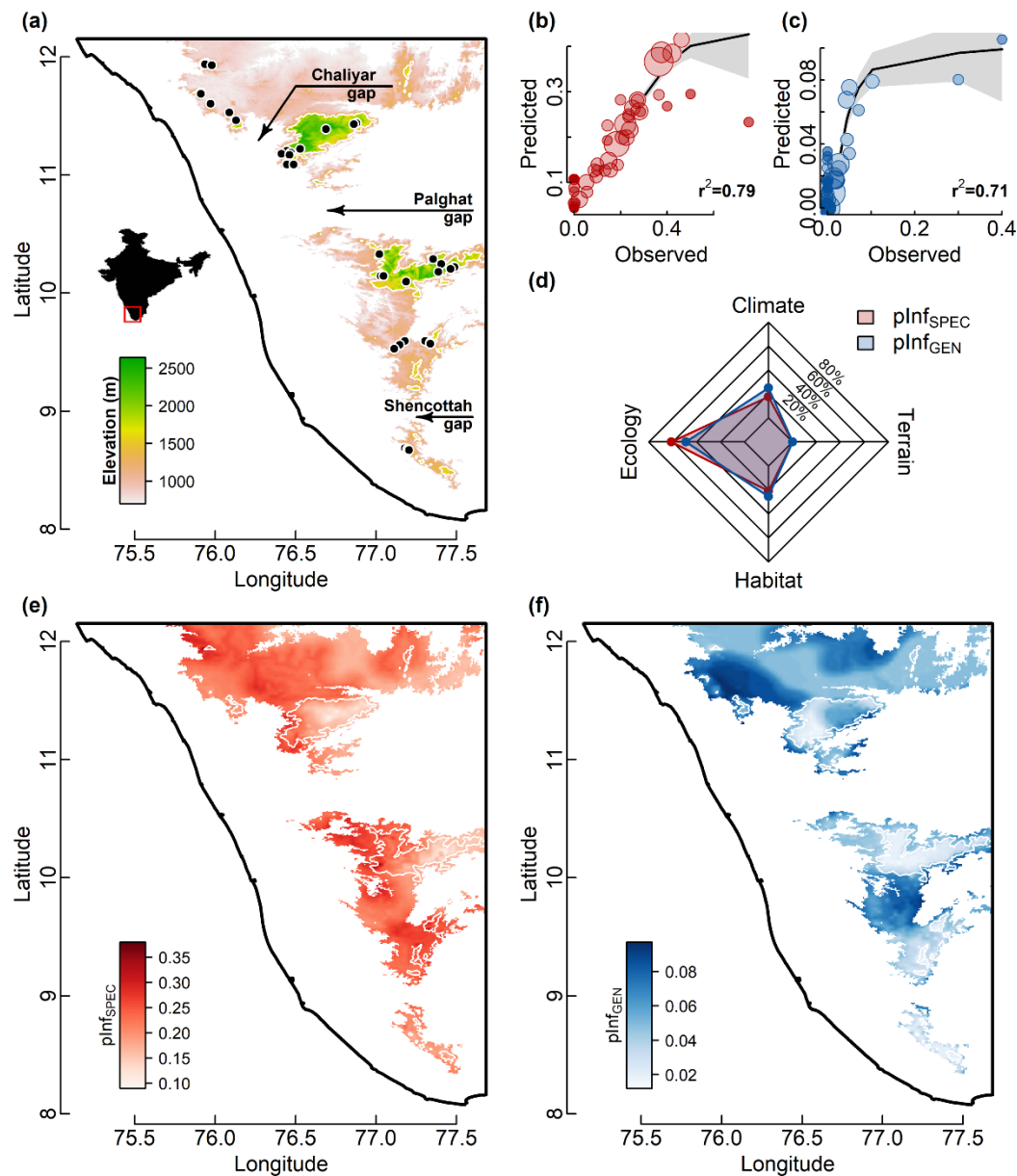
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FIGURES

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Figure 1. Study area and random forests model results of infection risk. **(a)** Study area map showing sample site locations (circles) in four geographical regions which are separated by three biogeographic barriers (the Chaliyar, Palghat and Shencottah gaps). All analyses were based on a sample of birds (N = 1172) captured at the 42 sites (Table S1). Random forest model performance in predicting: **(b)** *Haemoproteus* infection risk (pInf$_{HAEM}$) and **(c)** *Plasmodium* infection risk (pInf$_{PLAS}$). **(d)** Relative importance of variables grouped into four major categories (climate-, terrain-, habitat-, and host-related factors) in predicting pInf$_{HAEM}$ (red polygon) and pInf$_{PLAS}$ (blue polygon). Random forest model predictions of the effects of climate, terrain, habitat and ecological factors on infection probability of: **(e)** *Haemoproteus* (pInf$_{HAEM}$) and **(f)** *Plasmodium* (pInf$_{PLAS}$).
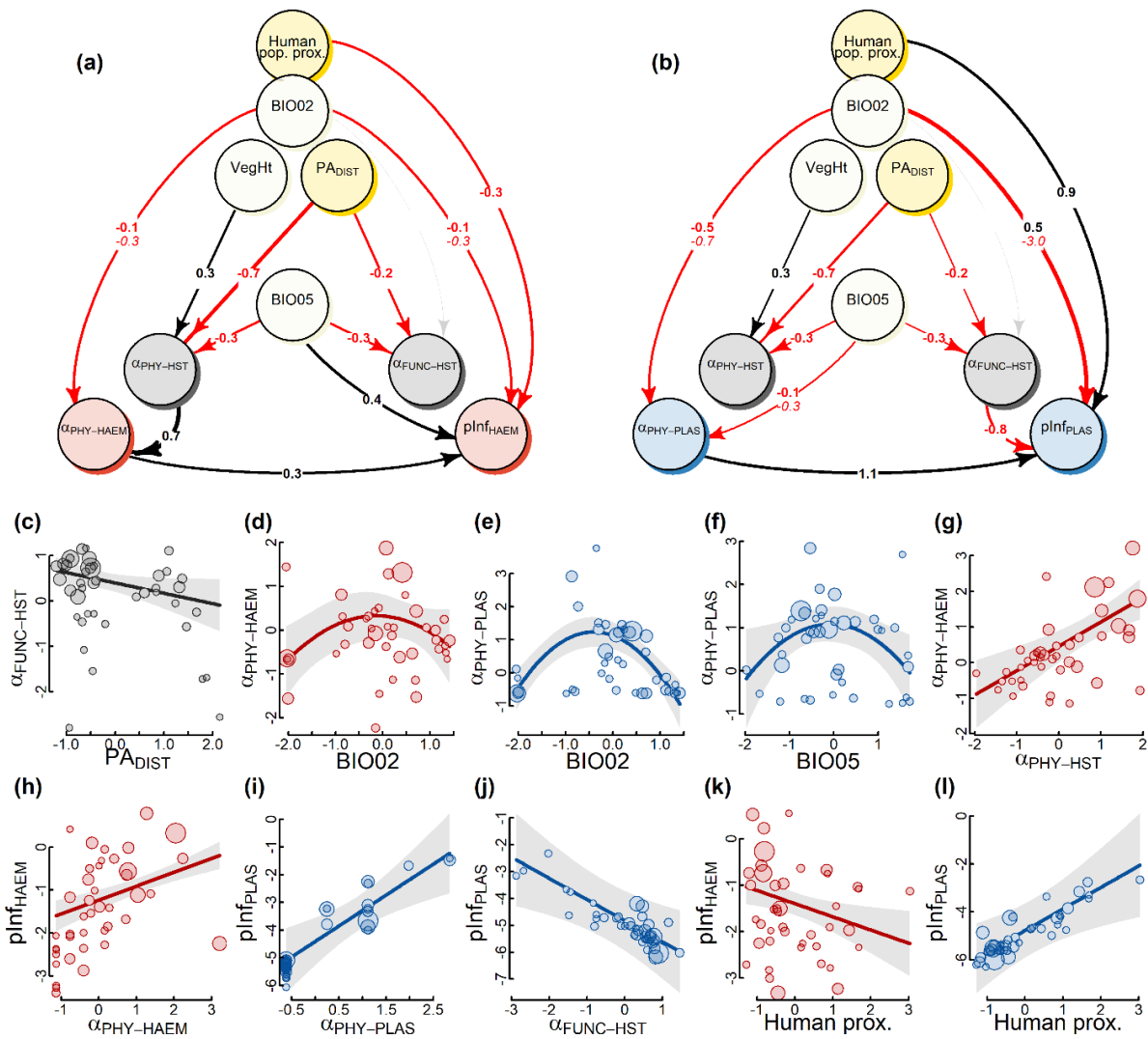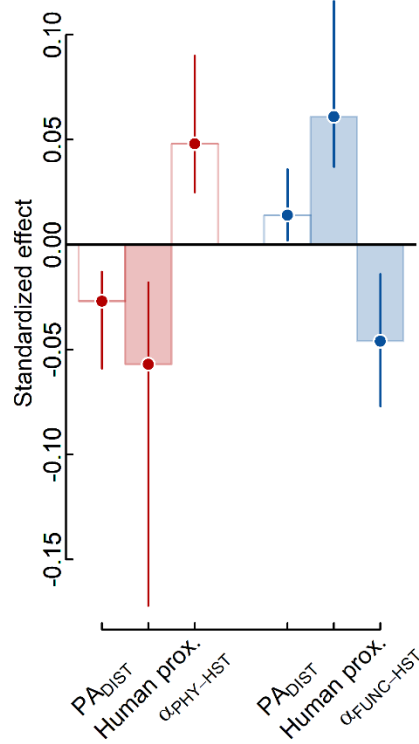
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Figure 2. Modeling the drivers of parasite infection risk. Structural equation model for: (**a**) *Haemoproteus* infection risk (pInf$_{HAEM}$), with direct or indirect effects of mean diurnal temperature range (BIO02), maximum temperature of warmest month (BIO05), vegetation height (VegHt), distance to protected areas (PA$_{DIST}$), human population proximity (Human pop. prox.), host phylogenetic diversity ($\alpha_{PHY\text{-}HOST}$), host functional diversity ($\alpha_{FUNC\text{-}HOST}$), and *Haemoproteus* phylogenetic diversity ($\alpha_{PHY\text{-}HAEM}$); (**b**) *Plasmodium* infection risk (pInf$_{PLAS}$), direct or indirect effects of BIO02, BIO05, VegHt, PA$_{DIST}$, Human pop. prox., $\alpha_{PHY\text{-}HOST}$, $\alpha_{FUNC\text{-}HOST}$, and *Plasmodium* phylogenetic diversity ($\alpha_{PHY\text{-}PLAS}$). In the flow diagrams, circles indicate variables (with model r^2 values, if applicable). Arrows indicate significant positive (black lines) or negative (red lines) relationships, with standardized coefficients for main (bold) and quadratic (italic) effects. (**c-n**) Partial residual plots for SEM paths for variables associated with the host (gray symbols), *Haemoproteus* (red symbols) and *Plasmodium* (blue symbols). Points represent trap sites scaled by relative sample size, with predicted fits (colored lines) and 95% confidence intervals (CIs; gray bands). All analyses were based on a sample of birds (N = 1172) captured at 42 sites across the study area (Figure 1A; Table S1).

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Figure 3. Indirect (white bars), direct (colored bars) and total (circles) effects of distance to
protected areas (PA$_{DIST}$), human population proximity (HPP), and either host phylogenetic ($\alpha_{PHY-HOST}$) or function ($\alpha_{FUNC-HOST}$) diversity on infection risk for *Haemoproteus* (red symbols) and
Plasmodium (blue symbols), respectively. Error bars are 95% CIs.