Title: Genetic and species-level biodiversity patterns are linked by demography and ecological opportunity

Running title: Geography of nuclear genetic diversity

Authors: Chloé Schmidt[1*], Stéphane Dray[2], Colin J. Garroway[1*]

Affiliations:

[1]Department of Biological Sciences, 50 Sifton Rd, University of Manitoba, Winnipeg, Manitoba R3T 2N2 Canada

[2]Univ Lyon, Université Claude Bernard Lyon 1, CNRS, Laboratoire de Biométrie et Biologie Evolutive, F-69100, Villeurbanne, France

*Correspondence to:

Chloé Schmidt
Department of Biological Sciences
50 Sifton Rd
University of Manitoba
Winnipeg, MB R3T 2N2
email: schmid46@myumanitoba.ca

Colin J Garroway
Department of Biological Sciences
50 Sifton Rd
University of Manitoba
Winnipeg, MB R3T 2N2
email: colin.garroway@umanitoba.ca

Abstract: Species richness and genetic diversity are the two most fundamental products of evolution. Both are important conservation targets—species richness contributes to ecosystem functioning and human wellbeing, while genetic diversity allows those species to respond to changes in their environment and persist in the long-term. Biogeographic patterns of species richness are well-described, but we know little about patterns of genome-wide genetic diversity at similar spatial scales. Further, despite considerable attention to latitudinal trends in species richness, we still do not have a solid empirical understanding of the various processes that produce them, how they interact, or how they affect genetic diversity. Here we show that genome-wide genetic diversity and species richness share spatial structure, however, species richness hotspots tend to harbor low levels of within-species genetic variation. A single model encompassing eco-evolutionary processes related to environmental energy availability, niche availability, and proximity to humans explained 75% of variation in gene diversity and 90% of the variation in species richness. Our empirical model of both levels of biodiversity supports theory and demonstrates the importance of carrying capacity and ecological opportunity at individual and species levels for generating continent-wide genetic and species diversity gradients.

Keywords: more individuals hypothesis, heterogeneity, Anthropocene, latitudinal diversity gradient, carrying capacity, macroecology

Introduction

Biodiversity patterns at the genetic and species levels form the foundation upon which higher-level diversity patterns emerge with the processes that generate diversity across these two base levels likely so entangled that they should be considered inseparable (Lowe et al. 2017; Pontarp et al. 2019). Biogeographic-scale variations in species-level diversity are among the best-described patterns in nature (Pontarp et al. 2019). The exploration of biogeographic patterns in genetic diversity across species has had to wait for technological advances in molecular genetics and the accumulation of data (Miraldo et al. 2016; Manel et al. 2020; Theodoridis et al. 2020). Regardless of research effort, our empirical understanding of the causes of diversity patterns at remains underdeveloped (Pontarp et al. 2019), likely in part due to a lack of integrated analyses of the causes of diversity at both levels. Here we produce a continent-scale map of nuclear genetic diversity for North American mammals and show that genetic diversity and species-level diversity are spatially correlated and likely have common environmental causes.

Existing hypotheses for species diversity patterns (Lomolino et al. 2016) generally fall into three broad categories: those related to evolutionary time for diversification, different diversification rates, and ecological limits on the number of species a region can support. Evolutionary time hypotheses predict that regions that have been colonized for the longest times should tend to have higher species richness than elsewhere due to diversification having taken place for longer periods (e.g., greater time for speciation in the tropics). Diversification rate hypotheses suggest that spatial variation in speciation or extinction rates (e.g., due variation in environmental conditions, mutation rates, and generation times) explain species richness patterns. Finally, ecological limits hypotheses posit that variation in resource availability sets a species-level

carrying capacity that limits the number of species able to coexist in a particular area. Here speciation, extinction, and colonization dynamics of species are analogous to the birth, death, and immigration dynamics that set population-level carrying capacities. There are at least 26 specific hypotheses that fall under these umbrella categories – detailed reviews can be found in (Mittelbach et al. 2007; Stein et al. 2014; Worm and Tittensor 2018; Pontarp et al. 2019).

Evolutionary time, evolutionary rates, and ecological limits hypotheses are often implicitly treated as competing ideas but speciation can clearly simultaneously be a product of both ecological and evolutionary processes (Pontarp and Wiens 2017). Indeed, recent modelling exercises suggest all categories of hypothesis can produce species richness gradients (Etienne et al. 2019). That said, the preponderance of theory suggests that carrying capacities limiting the supportable number of species in an environment produces the strongest and most stable species richness gradients (Vellend 2005; Worm and Tittensor 2018; Brodie 2019; Etienne et al. 2019). Etienne et al. (2019) used simulations to compare diversification rate, evolutionary time, and ecological limits hypotheses. Their models suggested that ecological limits on carrying capacity present the most parsimonious explanation for the latitudinal diversity gradient. There is also considerable empirical evidence in support of this theoretical work suggesting the likely importance of ecological limits in the formation of species richness patterns (Brodie 2019; Storch and Okie 2019). Taken together, there is good reason to consider ecological limits as a null expectation when exploring the causes of species richness patterns (Etienne et al. 2019).

We extended the consequences of processes related to ecological limits to explain multispecies population-level patterns of genetic diversity. If environments limit the number of species they

can support, they must also limit the population sizes of those species and thus the strength of genetic drift. Thus, demographic processes acting at the individual and species levels could simultaneously shape genetic and species-level biodiversity (Fig. 1). We focused on two prominent ecological limits hypotheses for species richness—the more individuals and environmental heterogeneity hypotheses. The more individuals hypothesis posits that energy availability imposes an upper limit on the number of individuals, and as a consequence, the number of species an area can support (Storch et al. 2018). According to the neutral theory of molecular evolution (Kimura 1983) and the neutral theory of biodiversity and biogeography (Hubbell 2001), diversity tends to increase with the number of individuals in an assemblage both in terms of genetic diversity within populations and the number of species in a community. We thus predicted positive relationships among genetic diversity, species richness, and energy availability. The habitat heterogeneity hypothesis suggests that environmental heterogeneity equates to niche availability, with heterogeneous areas able to support more specialized species, albeit at smaller population sizes because resources are divided (Kadmon and Allouche 2007; Allouche et al. 2012; Stein et al. 2014). As increasingly specialized populations diverge, genetic variation would be partitioned among locally adapted populations that may eventually no longer interbreed. These smaller populations will also lose genetic diversity due to genetic drift faster than large populations. We thus predicted that habitat heterogeneity would be positively associated with species richness and negatively associated with genetic diversity.

In addition to carrying capacity limits set by climatic factors and habitat complexity, a major contemporary environmental limitation on diversity is land transformation by humans. Habitat loss, fragmentation, and homogenization due to human activities such as urbanization reduce the

amount of habitat available to wild populations (McKinney 2006; Grimm et al. 2008) with consequences at genetic and species levels. Estimates suggest that within the last century, over 400 vertebrate species have gone extinct (Ceballos et al. 2020), vertebrate population sizes worldwide have shrunk by an average of 60% (WWF 2018), and intraspecific genetic diversity across taxa has declined by approximately 6% (Leigh et al. 2019). Contemporary rapid environmental change contributes to biodiversity patterns in addition to long-term processes. Because humans are known to influence both levels of biodiversity, our effects should be examined alongside natural factors. By reducing habitable area and environmental heterogeneity, we predicted that the effects of urbanization should also cause species richness and genetic diversity to decrease in more heavily disturbed areas.

Our objectives in this study were twofold. Biogeographic-scale correlations between nuclear genetic and species-level diversity patterns have not yet been established, so we first tested for shared spatial patterns at both levels of biodiversity. Having established shared patterns of variation we then tested for common environmental causes of genetic and species-level diversity using structural equation modelling (SEM). Structural equation modelling fits hypothesis networks that can accommodate multiple predictor and response variables within a hierarchical modelling framework. This allows the relative importance of multiple hypotheses to be assessed while accounting for species-level variation. Our data were repurposed publicly archived raw neutral nuclear genetic data for North American mammals spanning 801 sample sites, 38 species, and 34,841 individuals.

Methods

Data assembly

Genetic diversity database. We used the database of genetic metrics in North America compiled by Schmidt et al. (2020a,b). This database repurposed raw microsatellite data from 34,841 individuals across 38 mammalian species sampled at 801 sites in the United States and Canada, and includes consistently calculated measures of gene diversity (Nei 1973) and population-specific F_{ST} (Weir and Goudet 2017). See Table S2 for a summary of the dataset. Microsatellite markers estimate genome-wide diversity well (Mittell et al. 2015). They are commonly used in wildlife population genetic stusdies because they are cost-effective and do not require a reference genome, which allowed us to maximize sample size. We chose to focus on North America to control for regional history. Detailed methods for assembling this dataset can be found in (Schmidt et al. 2020a). Briefly, we performed a systematic search for species names of native North American mammals with keywords “microsat*”, “single tandem*”, “short tandem*”, and “str” using the ‘dataone’ R package, which interfaces with the DataONE platform to search online open data repositories (Jones et al. 2017). We discarded search results that did not meet our criteria for inclusion and removed results where study design may have influenced genetic diversity. For example we excluded non-neutral data and samples taken after a recent bottleneck, translocations, managed or captive populations, or island populations. We additionally removed populations with fewer than 5 individuals sampled. Gene diversity estimates the richness and evenness of alleles in a population, and we used it here as our metric for genetic diversity because it is minimally affected by sample size (Charlesworth and Charlesworth 2010)(Fig. S1). Sample sites are treated as point locations.

Population size. Because species-level censuses are not generally available, we used body size as a proxy for species-level population size. The inverse relationship between body size and species

160 population size is well documented and is especially reliable in mammals (Damuth 1981, 1987).
161 Neutral genome-wide genetic diversity is also negatively correlated with body size (Frankham
162 1996; Romiguier et al. 2014), the most likely explanation being strong links between body size
163 and effective population size (Frankham 1996). We recorded mean adult body mass (g) for each
164 species using data from the PanTHERIA database (Jones et al. 2009). Mass was log-transformed
165 before analysis. There were no obvious outliers in these data.

166 *Species richness.* We downloaded range maps for terrestrial mammals native to North America
167 from the IUCN Red List database (IUCN 2019). We filtered these maps to retain ranges for
168 extant, native, resident, mainland species in ArcMap Desktop 10.3.1 (ESRI, Redlands, CA). To
169 generate a map of species richness coincident with genetic sample sites, we estimated species
170 richness at each site within a 10 km buffer. For the range-wide measure of species richness used
171 in our hierarchical structural equation models, we summed the number of ranges that overlapped
172 each of our 38 focal species' ranges. To correct for potential biases due to differences in range
173 size (e.g. species with large ranges tending to have more overlapping ranges), we divided the
174 number of overlapping ranges by the species' range area (km^2), giving us species richness per
175 square kilometer for each species.

176 *Environmental variables.* We used potential evapotranspiration as our measure of energy
177 availability (Currie 1991). Specifically, potential evapotranspiration measures the atmosphere's
178 ability to remove water from the Earth's surface and is an indicator of atmospheric energy
179 availability. Potential evapotranspiration is one of the strongest environmental correlates of
180 species richness in mammals (Currie 1991; Kreft and Jetz 2007; Fisher et al. 2011; Jiménez-
181 Alfaro et al. 2016). We estimated mean potential evapotranspiration (mm/yr) across each
182 species' range using annual potential evapotranspiration data from 1970-2000 available via the

CGIAR Consortium for Spatial Information (Trabucco and Zomer 2019). We used a global topography map (NOAA and U.S. National Geophysical Data Center) to record the range in elevation across focal species ranges to quantify environmental heterogeneity (Stein et al. 2015). As with species richness, we corrected elevation range for potential biases introduced by species range area, because larger ranges tended to encompass greater topographical heterogeneity. Finally, human influence was a site level variable estimated using the human population density within a 10 km zone around each site, following (Schmidt et al. 2020b) finding its strong effect on mammalian genetic diversity.

Analysis

Genetic diversity and species richness maps. All analyses were conducted in R version 3.6.1 (R Core Team 2019). Our first step was to identify spatial patterns in genetic diversity. We accomplished this using distance-based Moran's eigenvector maps (MEMs) in the R package 'adespatial' (Dray et al. 2017). MEMs detect spatial patterns in data from a modified matrix of distances between sites—a neighbor matrix—whose eigenvalues are proportional to Moran's I index of spatial autocorrelation (Borcard and Legendre 2002; Borcard et al. 2004; Dray et al. 2006). MEMs are spatial eigenvectors that represent relationships between sites at all spatial scales detectable by the sampling scheme and can be included in linear models because they are orthogonal. A total of 199 positive MEMs were detected. Next, we used the forward selection procedure described in (Blanchet et al. 2008) to select two sets of MEMs: one describing site-level spatial patterns in genetic diversity and the other describing site-level species richness. Thirteen MEMs explained important spatial variation in gene diversity. In order of increasingly fine spatial scales, significant patterns were MEMs 2, 3, 4, 5, 22, 27, 30, 31, 47, 49, 101, 145,

206 152. Forty-three MEMs were important predictors of species richness, and 8 of these patterns
207 were shared by genetic diversity (significant MEMs are listed in Fig. S3).

208 We then subset MEMs based on Moran's I to retain only those explaining broad-scale spatial
209 patterns (MEMs with Moran's I > 0.25). The cut-off for broad-scale MEMs was MEM 5 for
210 genetic diversity and MEM 11 for species richness. We then fit individual linear regression
211 models for species richness and genetic diversity with the broad-scale MEMs, and plotted the
212 predicted values on a map of North America.
213
214 *Variation partitioning.* We next quantified the extent to which genetic diversity and species
215 richness covary spatially. Because MEMs for species richness and genetic diversity were
216 computed from the same set of coordinates, they were directly comparable. This allowed us to
217 identify shared spatial MEMs that might be related to a common environmental cause. We used
218 linear regressions and variance partitioning to determine what fraction of the total variation in
219 species richness and genetic diversity could be attributed to: (1) non-spatial variation, (2) non-
220 shared spatial variation, and (3) shared spatial variation. We partitioned variation as follows:

$$y_{SR} \sim \alpha + \beta_{1S}(\mathrm{MEM}_{1S}) + \beta_{2S}(\mathrm{MEM}_{2S}) + \cdots + \beta_{iS}(\mathrm{MEM}_{iS}) + \epsilon$$

$$y_{GD} \sim \alpha + \beta_{1G}(\mathrm{MEM}_{1G}) + \beta_{2G}(\mathrm{MEM}_{2G}) + \cdots + \beta_{iG}(\mathrm{MEM}_{iG}) + \epsilon$$

221 Where α is the grand mean, and y_{SR} and y_{GD} are site-level metrics of species richness and genetic
222 diversity. MEM_{iS} and MEM_{iG} refer to the set of MEMs explaining spatial variation in species
223 richness and genetic diversity, respectively, and βs are their slopes. The coefficients of variation
224 (R^2) for these models gave us the total proportion of variation in each response variable

attributable to spatial variation. Subtracting these values from 1 gives the amount of non-spatial variation.

To determine the amount of shared variation, we used the set of MEMs shared between species richness and genetic diversity (MEM_{SG}) as predictors in the regressions below:

$$y_{SR} \sim \alpha + \beta_{1SG}(\mathrm{MEM}_{1SG}) + \beta_{2SG}(\mathrm{MEM}_{2SG}) + \cdots + \beta_{iSG}(\mathrm{MEM}_{iSG}) + \epsilon$$

$$y_{GD} \sim \alpha + \beta_{1SG}(\mathrm{MEM}_{1SG}) + \beta_{2SG}(\mathrm{MEM}_{2SG}) + \cdots + \beta_{iSG}(\mathrm{MEM}_{iSG}) + \epsilon$$

R^2 values from these models yielded the proportion of variation in genetic diversity and species richness explained by shared spatial variation. Subtracting these values from the total spatial variation in species richness and genetic diversity gives the proportion of non-shared spatial variation.

Structural equation modeling. Next, we tested the hypothesis that differential carrying capacities and human disturbance simultaneously shape biodiversity patterns on genetic and species levels. To explore the common causes of genetic and species-level diversity, we fit our conceptual model integrating population genetics and ecological limits (Fig. 3a) to data using structural equation modelling. Using this approach we can examine cause-effect relationships within hypothesis networks that accommodate multiple predictor and response variables in a hierarchical modeling framework. Multiple hypotheses can be retained in a final model. Structural equation modeling is an extension of multivariate multiple regression where variables can be thought of as nodes in a network, and directional paths connecting nodes represent causal relationships. The strengths of paths are equal to regression coefficients (Shipley 2016). In addition to direct effects, you can quantify indirect effects between variables by multiplying direct effects over paths. Using standardized coefficients, we can compare the strength of

246 relationships and the relative support for retained hypotheses both within and across levels of
247 biodiversity. The appropriateness of links in the hypothesis network can be tested using tests of
248 directed separation (Shipley 2016), where the null hypothesis is that the two variables are
249 independent conditional on other predictors of either variable. This means that although we start
250 with a focus on ecological limits, the data can suggest the addition or removal of links
251 representing alternative hypotheses.
252
253 We have primarily focused on modeling broad-scale effects of the environment on continental
254 patterns of species richness and genetic diversity. We therefore focus here on hierarchical
255 modeling of patterns at the population and species level. Additionally, because the spatial
256 coverage of genetic sample sites in the data was not evenly distributed, some species ranges
257 could be oversampled if we considered site-level environmental variation, and thus
258 overrepresented compared to species ranges that contain fewer sampled populations. To capture
259 the broad spatial patterns depicted in Figure 2, and to avoid biasing our model as a result of
260 uneven sample site locations, we considered species richness, energy availability, and
261 heterogeneity at the species level in this analysis.

262 We implemented SEMs using the piecewiseSEM package (Lefcheck 2016; Lefcheck et al.
263 2019). PiecewiseSEM offers greater flexibility than other SEM software because it uses a local
264 estimation approach where each model is assessed individually (Lefcheck 2016). All variables
265 were scaled and centered before analysis.

266 We translated our conceptual model (Fig. 3a) into a series of 3 linear models with a single model
267 for each response variable (gene diversity, population size/body mass, and species richness). We
268 accounted for species-level differences in gene diversity using a linear mixed-effects model

269 controlling for species as a random effect within our structural equation model network.
270 Hierarchical models in piecewiseSEM were fit using the lme4 package (Bates et al. 2015).
271 Conceptually, a hierarchical model is a model of models—here, we are modelling gene diversity
272 within species and summarizing effects across species. Multiple linear regression models are fit
273 in base R.

274 Goodness-of-fit in SEM is determined by evaluating whether there are any missing links in the
275 causal structure, i.e. whether adding paths between pairs of variables would be more consistent
276 with the data. In piecewiseSEM missing links are tested using tests of directed separation
277 (Shipley 2016), where the null hypothesis is that the two variables are independent conditional
278 on other predictors of either variable. Starting with our conceptual model (Fig. 3a), we iteratively
279 updated models by adding links according to tests of directed separation until no further
280 biologically sensible links were suggested. We assessed model fit using the *p*-value for the
281 model network, where the null hypothesis is that the model is consistent with the data. Thus,
282 models with $p > 0.05$ are considered acceptable—we fail to reject our causal structure. We also
283 assessed fit using R^2 values for each response variable in the model network. For genetic
284 diversity, we used marginal (R^2_m) and conditional R^2 (R^2_c) values which respectively measure
285 the total variation explained by fixed effects, and the variation explained by both fixed and
286 random effects. We tested the residuals from component models for spatial autocorrelation using
287 Moran's tests and spatial correlograms.
288
289 *Effect of heterogeneity on population divergence.* After detecting a negative effect of
290 heterogeneity on intraspecific genetic diversity in our SEM, we performed a post hoc analysis to
291 test whether topographic heterogeneity also caused greater population differentiation within

292 species. A positive correlation between F_{ST} and heterogeneity, while controlling for distance,
293 would suggest that individuals move less between local environments, possibly due to niche
294 specialization. To test for differentiation we used population-specific F_{ST} (Weir and Goudet
295 2017) as a measure of genetic divergence, which was included in the genetic diversity database
296 (Schmidt et al. 2020a) where it was calculated in R using the 'hierfstat' package (Goudet and
297 Jombart 2015). Population-specific F_{ST} can be interpreted as a relative estimate of the time since
298 a population has diverged from a common ancestor. This metric requires at least 2 sampled
299 populations within a study to estimate, and due to this constraint 16 sites were excluded from this
300 analysis (n = 785). We controlled for isolation-by-distance by including MEMs significantly
301 related to F_{ST} to account for spatial structure. We scaled and centered all variables, then used a
302 linear mixed model controlling for species differences by including it as a random effect.
303
304 **Results**
305 *Spatial patterns in genetic diversity and species richness*
306 We detected spatial patterns at genetic and species levels of diversity. Sixty-five percent of the
307 total variation in species richness and 24% of variation in genetic diversity was spatially
308 structured (Fig. S2). Variance partitioning suggested that 85% of the total spatial variation in
309 genetic diversity, and 32% of spatial variation in species richness was accounted for by spatial
310 patterns shared at both levels of diversity (Fig. S2). We found no obvious relationship between
311 latitude and nuclear genetic diversity. Similar to patterns of species richness, a longitudinal
312 gradient in genetic diversity is the dominant pattern for North American mammals—however,
313 diversity gradients at the two levels trend in opposite directions. Nuclear genetic diversity

appears markedly lower in regions with high species richness, such as on the west and mid-Atlantic coasts, where there is high energy availability and topographic relief (Fig. 2).

Joint environmental causes of genetic diversity and species richness

Our conceptual model, updated according to tests of conditional independence among variables (directed separation), fit the data well (SEM p= 0.23, Fisher's C= 2.92; Fig. 3b, Table S1). Note that for structural equation models, $p > 0.05$ indicates that we fail to reject our model. There was no spatial autocorrelation in the body size model residuals, but genetic diversity and species richness models had statistically significant spatially autocorrelated residuals at very local scales (genetic diversity Moran's I = 0.025, species richness Moran's I = 0.029). These Moran's I values do not indicate strong spatial structure in the data, and we decided not to integrate it into our model. Positive spatial autocorrelation at such short distances is likely an artifact of irregular site locations and the hierarchical nature of the data. A lack of strong spatial autocorrelation in the model residuals suggests that the spatial structure of the diversity data was well captured by our model's environmental covariates (Fig. S3).

All predicted links in our conceptual model were supported (Fig 3a, b). Tests of directed separation suggested additional direct links from energy availability to species richness, genetic diversity to species richness, and heterogeneity to genetic diversity (Fig. 3b). Energy availability, niche heterogeneity, and human population density, acting both directly, and indirectly through species population size, explained 32% of the variation in genetic diversity. The species-level variation explained by the random effect for species brought the total variation in genetic diversity explained by our model to 75%. The same model explained 90% of the variation in

337 species richness. The strength of effects related to the more individuals hypothesis was most
338 prominent at the genetic level of diversity. The strength of the indirect effect of energy on
339 genetic diversity acting via population size was 0.13 compared to 0.02 for species richness (Fig.
340 3b, Table S1). Environmental heterogeneity, however, was the strongest single predictor of
341 species richness (path coefficient = 0.70 ± 0.01 SE), and a good predictor of genetic diversity
342 (path coefficient = -0.30 ± 0.07 SE). Directions of effects were as expected if greater niche
343 availability reduces population sizes, leading to increased genetic drift (Fig. 3, Table S1). Gene
344 diversity is not a measure of divergence so we tested whether environmental heterogeneity
345 predicted evolutionary divergence at the population level. Divergence increased in
346 heterogeneous environments ($\beta = 0.13 \pm 0.06$ SE). Finally, human population density both
347 directly and indirectly (via body mass/population size) affected species richness and genetic
348 diversity (Fig. 3b). Human population density had the strongest effect on population size/body
349 mass (path coefficient = -0.15 ± 0.03 SE), and relatively weaker direct effects on genetic
350 diversity and species richness (Fig. 3b, Table S1).
351
352 **Discussion**
353 We found striking content-wide spatial gradients in nuclear genetic diversity and show that these
354 patterns are negatively correlated with well-described biogeographic patterns in species richness
355 (Simpson 1964) (Fig. 2). Controlling for species-level variation, a considerable portion of the
356 variation in both genetic diversity and species richness patterns could be explained by just three
357 environmental factors – these were environmental energy availability, niche availability, and
358 human disturbance. Our model was consistent with the hypothesis that environmentally set
359 species-level carrying capacities simultaneously limit species population sizes, and consequently

360 genetic diversity through their effects on the strength of genetic drift. Niche availability was the
361 strongest contributor to broad-scale patterns at both levels of diversity, followed by energy
362 availability, and then human disturbance. This is strong empirical evidence suggesting that
363 genetic diversity and species richness patterns emerge from the same processes thus jointly
364 forming the base of the biodiversity hierarchy.
365
366 In support of the more individuals hypothesis (solid lines in Fig. 3b), our data indicated that low
367 energy environments supported fewer species and smaller population sizes with lower genetic
368 diversity. High energy areas had greater species richness and larger, more genetically diverse
369 populations. However, effects related to the more individuals hypothesis were weaker than those
370 of environmental heterogeneity (dashed lines in Fig. 3b). Heterogeneity appeared to increase
371 species richness and facilitate coexistence through greater niche availability, however
372 partitioning resources among niches seemed to support smaller numbers of individuals from
373 those species, creating a negative relationship between species richness and genetic diversity. At
374 the genetic level, greater population divergence in more heterogeneous environments suggests
375 that genetic drift is strong and gene flow limited in these areas. Selection is more spatially
376 varying in heterogeneous environments, and coupled with low gene flow, this could create
377 sufficient conditions for local adaptation—which can happen even under relatively high levels of
378 genetic drift (Hämälä et al. 2018). At lower latitudes where small-bodied species with large
379 effective population sizes dominate, heterogeneity and spatially varying selection could be
380 efficient drivers of ecological speciation. These results lend support to the idea that there are
381 higher diversification rates in more complex environments because there are more opportunities
382 for speciation. We additionally speculate that the direct effect of energy on species richness we

383 detected even after accounting for population size and heterogeneity (Fig. 3b) may be related to
384 niche availability as well. This relationship has been noted elsewhere and has sometimes been
385 interpreted as refuting the more individuals hypothesis (Storch et al. 2018). Vegetation structure
386 may drive the link between species richness and temperature (Pautasso and Gaston 2005;
387 Jiménez-Alfaro et al. 2016), as complex, vegetation-rich habitats in warmer environments also
388 have greater niche availability. Because both links are retained in our model it seems clear that
389 this additional link does not negate the more individuals hypothesis, but rather is additive and
390 indeed more important in determining species richness than the more individuals effect.
391
392 The specific ways environments shape nuclear genetic- and species-level diversity will likely
393 differ across taxa. This carrying capacity-based interpretation of our results assumes that an
394 environmentally set equilibrium between speciation, immigration and extinction has been
395 reached. There is good evidence for this in North American mammals, where diversification
396 rates have slowed as diversity increased (Brodie 2019). It seems likely that processes other than
397 ecological limits will be more important for the diversity dynamics of taxa that may not have
398 reached or have been knocked out of equilibrium at the genetic or species levels. Speciation is a
399 product of both ecological and evolutionary processes, and it is unlikely ecological limits act in
400 isolation. Indeed, the underlying causes of species richness gradients—be they ecological limits,
401 evolutionary time, or diversification rates—have likely been debated for so long precisely
402 because several processes operating with different importance across the timeline of
403 diversification are capable of producing gradients (Etienne et al. 2019). Recent thinking (Pontarp
404 and Wiens 2017) advocates a more interconnected view, suggesting that time for speciation
405 should be most detectable more immediately following broad-scale environmental change. When

406 all locales are colonized, habitats that provide more opportunities for speciation should over time
407 become the most diverse. As diversity increases, diversification rates slow as regions approach
408 equilibrium (Brodie 2019). It follows that evolutionary time and diversification rates may have
409 each at different periods of history been the dominant driver of biodiversity, but both are
410 ultimately affected by variation in carrying capacity (Pontarp and Wiens 2017).
411
412 Contemporary drivers of biodiversity patterns are rarely modeled in a way that makes them
413 comparable to evolutionary scale causes. Understanding the ecological processes generating
414 gradients in genetic diversity and species richness has important implications for understanding
415 how biodiversity responds to human-caused environmental transformation. Cities are the world's
416 newest and most rapidly expanding biome, and it is clear that they have already had profound
417 effects on biodiversity patterns (Palumbi 2001; WWF 2018; Schmidt et al. 2020a). The negative
418 effect of human population density we detected on body size is consistent with previous findings
419 showing that urban communities tend to be made up of smaller species (Merckx et al. 2018).
420 Although it seems human presence and heterogeneity both have negative effects on genetic
421 diversity in our model, species richness was reduced in more urban environments (Fig. 3b). This
422 result suggests that cities reduce population sizes and gene flow (Schmidt et al. 2020a), but
423 currently do not support diverse communities. Because cities are relatively new habitat types and
424 they are still in the initial phase of colonization, we would not expect them to be in equilibrium.
425 At this stage processes related to evolutionary time will likely predominate until all available
426 niches are occupied. Indeed, there is some evidence that following an initial extinction debt after
427 rapid urbanization, older cities support more biodiversity (Aronson et al. 2014; Norton et al.

2016). Presently, a subset of species do well in cities (McKinney 2006), but the broader effects of habitat transformation remain to be seen in the long term.

It is notable that the negative correlation we find between species richness and nuclear genetic diversity contradicts relatively consistent positive correlations found between species richness and mitochondrial (mtDNA) genetic diversity (Miraldo et al. 2016; Manel et al. 2020; Millette et al. 2020; Theodoridis et al. 2020). This contrast warrants deeper exploration because it has implications for how we interpret biogeographical patterns in genetic diversity to advance general evolutionary knowledge, and how we apply this knowledge for the purposes of conservation and management. mtDNA has several idiosyncrasies associated with the specific biology of mitochondria that distinguish it from genetic diversity measured with neutral nuclear DNA. It is inherited as a single non-recombining locus, and has highly variable mutation rates which can vary 100-fold across species (Nabholz et al. 2008). Cellular metabolism within mitochondria produces reactive oxygen species which affect mutation rates in mtDNA, but these oxygen radicals do not cause oxidative damage in the nucleus (Hoffmann et al. 2004; Lanfear et al. 2007)—further decoupling mtDNA and nuclear DNA diversity. The most commonly used markers in mtDNA studies are the protein-coding genes *cytochrome oxidase I* and *cytochrome b*, which are involved in cellular respiration and very likely do not evolve under neutrality (Galtier et al. 2009). Unlike neutral nuclear DNA, it has been shown that mtDNA diversity is unrelated to life history, ecological traits, and census and effective population sizes (Bazin et al. 2006; Nabholz et al. 2008). mtDNA diversity is thus a very different quantity than the neutral nuclear diversity estimates we use here. Its lack of relationship with population size makes it unsuited for testing demographic hypotheses related to ecological limits or environmental stability, where

451 instability causes population size fluctuations which limit species richness. Using genetic
452 diversity metrics estimated from neutral nuclear DNA allows us to more clearly link
453 environments to species richness and genetic diversity through demography, population size, and
454 by extension, species life history traits which partly set the effective population size.
455
456 Given the above-described concerns about the suitability of mtDNA for detecting patterns of
457 interest, what then could drive the positive correlation between species richness and mtDNA
458 diversity? Such a relationship could be due to an analytical issue. Studies of mtDNA diversity –
459 species richness correlations tend to aggregate sequences within pre-defined geographic
460 sampling units, calculate mtDNA diversity for each species, then use the average diversity of all
461 species sampled in a spatial unit as their measure of genetic diversity in subsequent analyses
462 (Miraldo et al. 2016; Manel et al. 2020; Theodoridis et al. 2020). We suspect that this metric of
463 variation captures phylogenetic signals in mtDNA, and thus that it must be positively correlated
464 with species diversity because it reflects the accumulation of mutations following reproductive
465 divergence. Given the peculiarities of mtDNA noted above, we are concerned that conservation
466 recommendations for maintaining genetic diversity based on positive correlations between
467 mtDNA diversity and species diversity are misplaced (Miraldo et al. 2016; Theodoridis et al.
468 2020). Studies of mtDNA diversity – species richness correlations often interpret regions with
469 high mtDNA variation as indicative of a population's capacity to adapt, and thus warranting
470 conservation concern (Theodoridis et al. 2020). Regardless of whether one accepts the concerns
471 we describe, this too seems inappropriate. A population's capacity to adapt is defined as the
472 additive genetic variance underlying fitness (Fisher 1930). Though theory, based on strong
473 assumptions, suggests that neutral nuclear genetic diversity should predict additive genetic

474 variation (Falconer and Mackay 1996), it appears that this relationship is not strong enough to be
475 useful in practice (Mittell et al. 2015). There is no expectation of correlations between mtDNA
476 diversity and either neutral nuclear genetic diversity, or additive genetic variation (Mittell et al.
477 2015). mtDNA diversity thus cannot be interpreted as a population's capacity to adapt. Neutral
478 microsatellite variation does indeed reflect genome-wide variation well (Mittell et al. 2015).
479 Though not strongly correlated with the capacity to adapt, genome-wide variation is indicative of
480 the efficiency of selection through its link to effective population size and the scope for
481 inbreeding. Given that we have no reason to suspect that mtDNA reflects adaptive potential and
482 that mtDNA diversity trends opposite of nuclear genetic diversity, general management
483 strategies aimed at conserving high mtDNA genetic diversity regions would seem to have the
484 opposite effect of the conservation intent.
485
486 Ecosystem sustainability given environmental perturbations occurring more frequently due to
487 human causes, depends on the resiliency of landscapes, communities, and populations (Oliver et
488 al. 2015). Genetic diversity is crucial to a population's adaptive potential because the efficiency
489 with which selection can act is determined by the effective population size which sets the rate of
490 genetic drift. Yet genetic diversity is not equally distributed in space and indeed, in mammals,
491 appears to be lower in heterogeneous environments which exert greater spatially varying
492 selection. Knowledge of how natural environments shape population genetic composition is
493 fundamental to understanding how these natural patterns will shift with continued land
494 transformation by humans. Mammals are one of the best-studied taxa, however, rules applicable
495 to them may not generalize well across other groups. For instance, the relevance of the more
496 individuals hypothesis for ectotherms has been questioned because their energy usage is well

497 below that of endotherms (Buckley et al. 2008). Indeed, continental patterns of species richness
498 differ across taxa, which may stem from life history or physiology differences (Currie 1991). It
499 will be necessary to test the hypothesis developed here on other taxonomic groups and in
500 different regions to gain a more holistic understanding of the causes of biodiversity. The intimate
501 connections between the environment, species richness, and genetic diversity we find here
502 suggest that changes on one level can cascade throughout the system and profoundly reshape
503 biodiversity patterns across multiple biological levels in ways we do not yet fully grasp.
504
505 **Author contributions:** C.J.G. and C.S. conceptualized the study. C.S., S.D. and C.J.G. designed
506 the study and C.S. conducted the statistical analysis with input from S.D. and C.J.G. All authors
507 contributed to data interpretation. C.S. wrote the first draft of the manuscript and all authors
508 participated in editing subsequent manuscript drafts.
509
510 **Acknowledgements:** We would like to thank the Population Ecology and Evolutionary Genetics
511 group for their feedback on this manuscript. We are also grateful to the authors whose work
512 provided the raw data for this synthesis. C.S. and C.J.G. were supported by a Natural Sciences
513 and Engineering Research Council of Canada Discovery Grant to C.J.G. C.S. was also supported
514 by a U. Manitoba Graduate Fellowship, and a U. Manitoba Graduate Enhancement of Tri-council
515 funding grant to C.J.G.
516
517 **Data availability:** Synthesized genetic data is available from the Dryad Data Repository (DOI:
518 10.5061/dryad.cz8w9gj0c). Species range boundary files and environmental data are available
519 from open online sources (see Methods).

References

Allouche, O., M. Kalyuzhny, G. Moreno-Rueda, M. Pizarro, and R. Kadmon. 2012. Area-heterogeneity tradeoff and the diversity of ecological communities. Proc. Natl. Acad. Sci. U. S. A. 109:17495–17500.

Aronson, M. F. J., F. A. La Sorte, C. H. Nilon, M. Katti, M. A. Goddard, C. A. Lepczyk, P. S. Warren, N. S. G. Williams, S. Cilliers, B. Clarkson, C. Dobbs, R. Dolan, M. Hedblom, S. Klotz, J. L. Kooijmans, I. Kühn, I. MacGregor-Fors, M. McDonnell, U. Mörtberg, P. Pyšek, S. Siebert, J. Sushinsky, P. Werner, and M. Winter. 2014. A global analysis of the impacts of urbanization on bird and plant diversity reveals key anthropogenic drivers. Proc. R. Soc. B Biol. Sci. 281:20133330.

Bates, D., M. Mächler, B. Bolker, and S. Walker. 2015. Fitting linear mixed-effects models using lme4. J. Stat. Softw. 67.

Bazin, E., S. Glémin, and N. Galtier. 2006. Population size does not influence mitochondrial genetic diversity in animals. Science. 312:570–572.

Blanchet, G. F., P. Legendre, and Borcard. 2008. Forward selection of explanatory cariables. Ecology 89:2623–2632.

Borcard, D., and P. Legendre. 2002. All-scale spatial analysis of ecological data by means of principal coordinates of neighbour matrices. Ecol. Modell. 153:51–68.

Borcard, D., P. Legendre, C. Avois-Jacquet, and H. Tuomisto. 2004. Dissecting the spatial structure of ecological data at multiple scales. Ecology 85:1826–1832.

Brodie, J. F. 2019. Environmental limits to mammal diversity vary with latitude and global temperature. Ecol. Lett. 22:480–485.

Buckley, L. B., G. H. Rodda, and W. Jetz. 2008. Thermal and energetic constraints on ectotherm abundance: A global test using lizards. Ecology 89:48–55.

Ceballos, G., P. R. Ehrlich, and P. H. Raven. 2020. Vertebrates on the brink as indicators of biological annihilation and the sixth mass extinction. Proc. Natl. Acad. Sci. U. S. A. 117:13596–13602.

Charlesworth, B., and D. Charlesworth. 2010. Elements of evolutionary genetics. Roberts & Company Publishers, Greenwood Village, Colorado, USA.

Currie, D. J. 1991. Energy and large-scale patterns of animal- and plant-species richness. Am. Nat. 137:27–49.

Damuth, J. 1987. Interspecific allometry of population density in mammals and other animals: the independence of body mass and population energy-use. Biol. J. Linn. Soc. 31:193–246.

Damuth, J. 1981. Population density and body size in mammals. Nature 290:1980–1981.

Dray, S., G. Blanchet, D. Borcard, S. Clappe, G. Guenard, T. Jombart, G. Larocque, P. Legendre, N. Madi, and H. H. Wagner. 2017. adespatial: Multivariate Multiscale Spatial Analysis.

556 Dray, S., P. Legendre, and P. R. Peres-Neto. 2006. Spatial modelling: a comprehensive
557 framework for principal coordinate analysis of neighbour matrices (PCNM). Ecol. Modell.
558 196:483–493.

559 Etienne, R. S., J. S. Cabral, O. Hagen, F. Hartig, A. H. Hurlbert, L. Pellissier, M. Pontarp, and D.
560 Storch. 2019. A minimal model for the latitudinal diversity gradient suggests a dominant
561 role for ecological limits. Am. Nat. 194:E122–E133.

562 Falconer, D. S., and T. F. C. Mackay. 1996. Introduction to Quantitative Genetics. 4th ed.
563 Longman, Harlow, Essex, UK.

564 Fisher, J. B., R. J. Whittaker, and Y. Malhi. 2011. ET come home: Potential evapotranspiration
565 in geographical ecology. Glob. Ecol. Biogeogr. 20:1–18.

566 Fisher, R. A. 1930. The Genetical Theory of Natural Selection. Oxford University Press, Oxford.

567 Frankham, R. 1996. Relationship of Genetic variation to population size in wildlife. Conserv.
568 Biol. 10:1500–1508.

569 Galtier, N., B. Nabholz, S. Glémin, and G. D. D. Hurst. 2009. Mitochondrial DNA as a marker
570 of molecular diversity: A reappraisal. Mol. Ecol. 18:4541–4550.

571 Goudet, J., and T. Jombart. 2015. hierfstat: Estimation and Tests of Hierarchical F-Statistics.

572 Grimm, N. B., S. H. Faeth, N. E. Golubiewski, C. L. Redman, J. Wu, X. Bai, and J. M. Briggs.
573 2008. Global Change and the Ecology of Cities. Science. 319:756–760.

574 Hämälä, T., T. M. Mattila, and O. Savolainen. 2018. Local adaptation and ecological
575 differentiation under selection, migration and drift in *Arabidopsis lyrata*. Evolution.
576 72:1373–1386.

577 Hoffmann, S., D. Spitkovsky, J. P. Radicella, B. Epe, and R. J. Wiesner. 2004. Reactive oxygen
578 species derived from the mitochondrial respiratory chain are not responsible for the basal
579 levels of oxidative base modifications observed in nuclear DNA of mammalian cells. Free
580 Radic. Biol. Med. 36:765–773.

581 Hubbell, S. P. 2001. The Unified Neutral Theory of Biodiversity and Biogeography. Princeton
582 University Press, Princeton NJ.

583 IUCN. 2019. The IUCN Red List of Threatened Species. Version 2019-1.

584 Jiménez-Alfaro, B., M. Chytrý, L. Mucina, J. B. Grace, and M. Rejmánek. 2016. Disentangling
585 vegetation diversity from climate-energy and habitat heterogeneity for explaining animal
586 geographic patterns. Ecol. Evol. 6:1515–1526.

587 Jones, K. E., J. Bielby, M. Cardillo, S. A. Fritz, J. O'Dell, C. D. L. Orme, K. Safi, W. Sechrest,
588 E. H. Boakes, C. Carbone, C. Connolly, M. J. Cutts, J. K. Foster, R. Grenyer, M. Habib, C.
589 A. Plaster, S. A. Price, E. A. Rigby, J. Rist, A. Teacher, O. R. P. Bininda-Emonds, J. L.
590 Gittleman, G. M. Mace, and A. Purvis. 2009. PanTHERIA: a species-level database of life
591 history, ecology, and geography of extant and recently extinct mammals. Ecology 90:2648–
592 2648.

593 Jones, M. B., P. Slaughter, R. Nahf, C. Boettiger, C. Jones, J. Read, L. Walker, E. Hart, and S.

Chamberlain. 2017. dataone: R Interface to the DataONE REST API.

Kadmon, R., and O. Allouche. 2007. Integrating the effects of area, isolation, and habitat heterogeneity on species diversity: A unification of island biogeography and niche theory. Am. Nat. 170:443–454.

Kimura, M. 1983. The Neutral Theory of Molecular Evolution. Cambridge University Press, Cambridge.

Kreft, H., and W. Jetz. 2007. Global patterns and determinants of vascular plant diversity. Proc. Natl. Acad. Sci. 104:5925–5930.

Lanfear, R., J. A. Thomas, J. J. Welch, T. Brey, and L. Bromham. 2007. Metabolic rate does not calibrate the molecular clock. Proc. Natl. Acad. Sci. U. S. A. 104:15388–15393.

Lefcheck, J., J. Byrnes, and J. Grace. 2019. piecewiseSEM: Piecewise Structural Equation Modeling.

Lefcheck, J. S. 2016. piecewiseSEM: Piecewise structural equation modelling in r for ecology, evolution, and systematics. Methods Ecol. Evol. 7:573–579.

Leigh, D. M., A. P. Hendry, E. Vázquez□Domínguez, and V. L. Friesen. 2019. Estimated six per cent loss of genetic variation in wild populations since the industrial revolution. Evol. Appl. 12:1505–1512.

Lomolino, M., B. Riddle, and R. Whittaker. 2016. Biogeography: biological diversity across space and time. 5th ed. Oxford University Press.

Lowe, W. H., R. P. Kovach, and F. W. Allendorf. 2017. Population genetics and demography unite ecology and evolution. Trends Ecol. Evol. 32:141–152. Elsevier Ltd.

Manel, S., P. E. Guerin, D. Mouillot, S. Blanchet, L. Velez, C. Albouy, and L. Pellissier. 2020. Global determinants of freshwater and marine fish genetic diversity. Nat. Commun. 11:1–9. Springer US.

McKinney, M. L. 2006. Urbanization as a major cause of biotic homogenization. Biol. Conserv. 127:247–260.

Merckx, T., C. Souffreau, A. Kaiser, L. F. Baardsen, T. Backeljau, D. Bonte, K. I. Brans, M. Cours, M. Dahirel, N. Debortoli, K. De Wolf, J. M. T. Engelen, D. Fontaneto, A. T. Gianuca, L. Govaert, F. Hendrickx, J. Higuti, L. Lens, K. Martens, H. Matheve, E. Matthysen, E. Piano, R. Sablon, I. Schön, K. Van Doninck, L. De Meester, and H. Van Dyck. 2018. Body-size shifts in aquatic and terrestrial urban communities. Nature 558:113–116.

Millette, K. L., V. Fugère, C. Debyser, A. Greiner, F. J. J. Chain, and A. Gonzalez. 2020. No consistent effects of humans on animal genetic diversity worldwide. Ecol. Lett. 23:55–67.

Miraldo, A., S. Li, M. K. Borregaard, A. Florez-Rodriguez, S. Gopalakrishnan, M. Rizvanovic, Z. Wang, C. Rahbek, K. A. Marske, and D. Nogues-Bravo. 2016. An Anthropocene map of genetic diversity. Science. 353:1532–1535.

Mittelbach, G. G., D. W. Schemske, H. V. Cornell, A. P. Allen, J. M. Brown, M. B. Bush, S. P.

Harrison, A. H. Hurlbert, N. Knowlton, H. A. Lessios, C. M. McCain, A. R. McCune, L. A. McDade, M. A. McPeek, T. J. Near, T. D. Price, R. E. Ricklefs, K. Roy, D. F. Sax, D. Schluter, J. M. Sobel, and M. Turelli. 2007. Evolution and the latitudinal diversity gradient: Speciation, extinction and biogeography. Ecol. Lett. 10:315–331.

Mittell, E. A., S. Nakagawa, and J. D. Hadfield. 2015. Are molecular markers useful predictors of adaptive potential? Ecol. Lett. 18:772–778.

Nabholz, B., J. F. Mauffrey, E. Bazin, N. Galtier, and S. Glemin. 2008. Determination of mitochondrial genetic diversity in mammals. Genetics 178:351–361.

Nei, M. 1973. Analysis of gene diversity in subdivided populations. Proc. Natl. Acad. Sci. U. S. A. 70:3321–3323.

NOAA, and U.S. National Geophysical Data Center. n.d. TerrainBase, release 1.0. Boulder CO.

Norton, B. A., K. L. Evans, and P. H. Warren. 2016. Urban biodiversity and landscape ecology: patterns, processes and planning. Curr. Landsc. Ecol. Reports 1:178–192. Current Landscape Ecology Reports.

Oliver, T. H., M. S. Heard, N. J. B. Isaac, D. B. Roy, D. Procter, F. Eigenbrod, R. Freckleton, A. Hector, C. D. L. Orme, O. L. Petchey, V. Proença, D. Raffaelli, K. B. Suttle, G. M. Mace, B. Martín-López, B. A. Woodcock, and J. M. Bullock. 2015. Biodiversity and resilience of ecosystem functions. Trends Ecol. Evol. 30:673–684. Elsevier Ltd.

Palumbi, S. R. 2001. Humans as the world's greatest evolutionary force. Science. 293:1786–1790.

Pautasso, M., and K. J. Gaston. 2005. Resources and global avian assemblage structure in forests. Ecol. Lett. 8:282–289.

Pontarp, M., L. Bunnefeld, J. S. Cabral, R. S. Etienne, S. A. Fritz, R. Gillespie, C. H. Graham, O. Hagen, F. Hartig, S. Huang, R. Jansson, O. Maliet, T. Münkemüller, L. Pellissier, T. F. Rangel, D. Storch, T. Wiegand, and A. H. Hurlbert. 2019. The latitudinal diversity gradient: novel understanding through mechanistic eco-evolutionary models. Trends Ecol. Evol. 34:211–223.

Pontarp, M., and J. J. Wiens. 2017. The origin of species richness patterns along environmental gradients: uniting explanations based on time, diversification rate and carrying capacity. J. Biogeogr. 44:722–735.

R Core Team. 2019. R: A Language and Environment for Statistical Computing. Vienna, Austria.

Romiguier, J., P. Gayral, M. Ballenghien, A. Bernard, V. Cahais, A. Chenuil, Y. Chiari, R. Dernat, L. Duret, N. Faivre, E. Loire, J. M. Lourenco, B. Nabholz, C. Roux, G. Tsagkogeorga, A. A. T. Weber, L. A. Weinert, K. Belkhir, N. Bierne, S. Glï¿½min, and N. Galtier. 2014. Comparative population genomics in animals uncovers the determinants of genetic diversity. Nature 515:261–263.

Schmidt, C., M. Domaratzki, R. P. Kinnunen, J. Bowman, and C. J. Garroway. 2020a. Continent-wide effects of urbanization on bird and mammal genetic diversity. Proc. R. Soc. B Biol.

671 Sci. 287:20192497.

672 Schmidt, C., M. Domaratzki, R. P. Kinnunen, J. Bowman, and C. J. Garroway. 2020b. Data
673 from: Continent-wide effects of urbanization on bird and mammal genetic diversity. Dryad
674 Data Repository.

675 Shipley, B. 2016. Cause and correlation in biology. 2nd ed. Cambridge University Press,
676 Cambridge.

677 Simpson, G. G. 1964. Species density of North American recent mammals. Syst. Zool. 13:57–73.

678 Stein, A., J. Beck, C. Meyer, E. Waldmann, P. Weigelt, and H. Kreft. 2015. Differential effects
679 of environmental heterogeneity on global mammal species richness. Glob. Ecol. Biogeogr.
680 24:1072–1083.

681 Stein, A., K. Gerstner, and H. Kreft. 2014. Environmental heterogeneity as a universal driver of
682 species richness across taxa, biomes and spatial scales. Ecol. Lett. 17:866–880.

683 Storch, D., E. Bohdalková, and J. Okie. 2018. The more-individuals hypothesis revisited: the role
684 of community abundance in species richness regulation and the productivity–diversity
685 relationship. Ecol. Lett. 21:920–937.

686 Storch, D., and J. G. Okie. 2019. The carrying capacity for species richness. Glob. Ecol.
687 Biogeogr. 28:1519–1532.

688 Theodoridis, S., D. A. Fordham, D. Nogues-Bravo, and S. C. Brown. 2020. Evolutionary history
689 and past climate change shape the distribution of genetic diversity in terrestrial mammals.
690 Nat. Commun. 1–11.

691 Trabucco, A., and R. Zomer. 2019. Global Aridity Index and Potential Evapotranspiration (ET0)
692 Climate Database v2. , doi: 10.6084/m9.figshare.7504448.v3.

693 Vellend, M. 2005. Species diversity and genetic diversity: parallel processes and correlated
694 patterns. Am. Nat. 166:199–215.

695 Weir, B. S., and J. Goudet. 2017. A Unified Characterization of Population Structure. Genetics
696 206:2085–2103.

697 Worm, B., and D. P. Tittensor. 2018. A theory of global biodiversity. Princeton University Press,
698 Princeton, New Jersey.

699 WWF. 2018. Living Planet Report - 2018: Aiming higher. WWF, Gland, Switzerland.

700

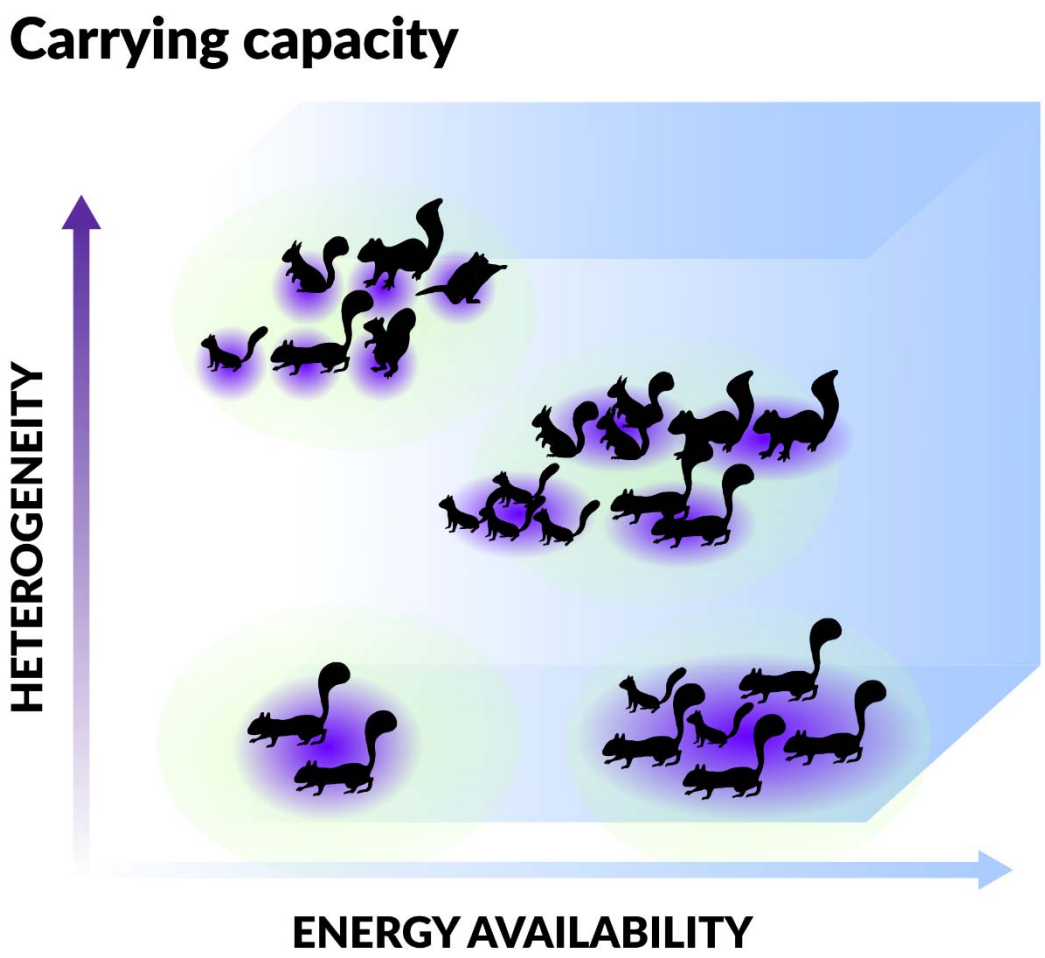
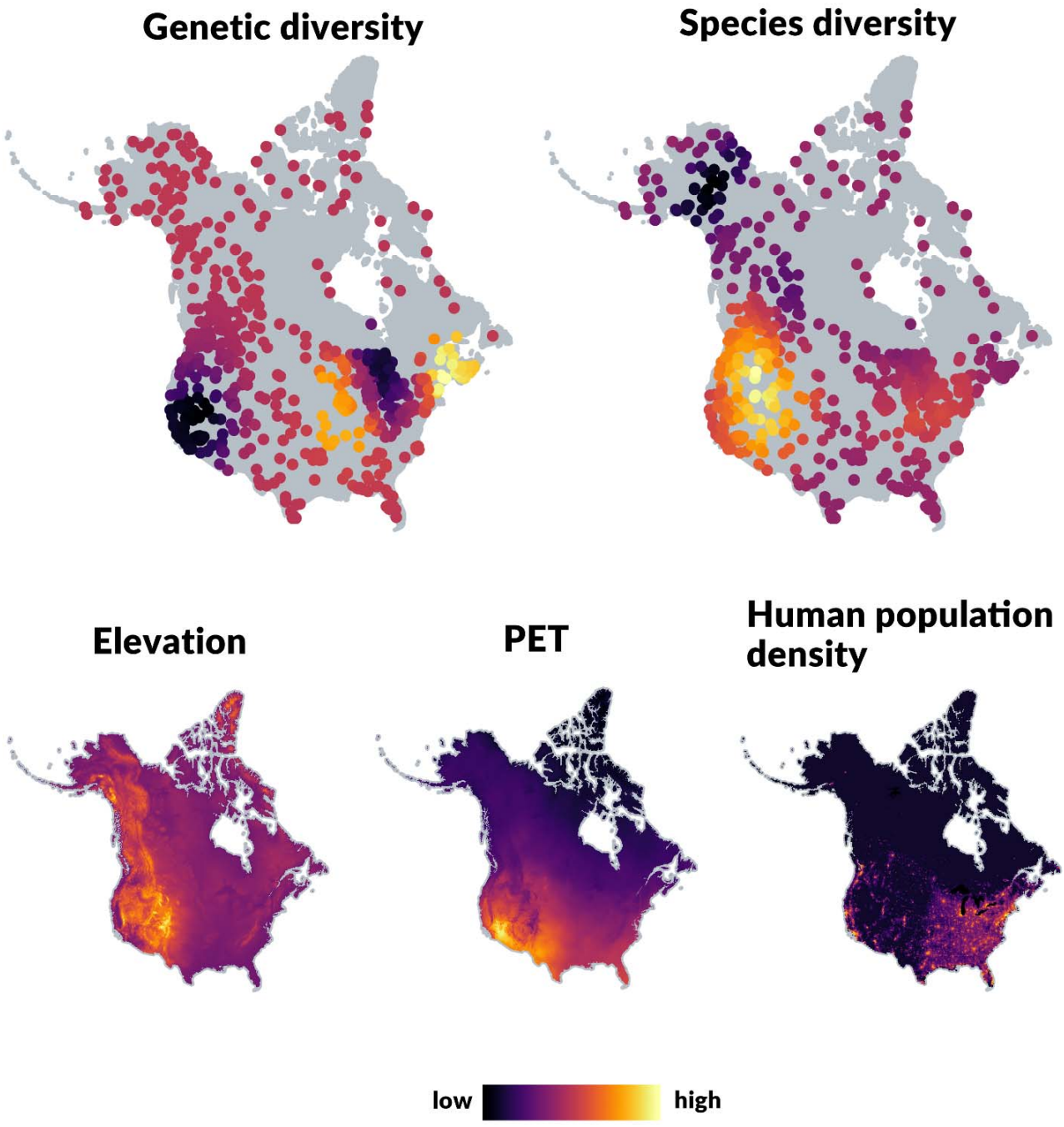


Fig. 1. Carrying capacities at population and species levels. Green areas represent total habitat area, and are all equal in size. Purple areas are niches, which increase in number with increasing heterogeneity (y axis), and increase in area with higher energy availability (x axis). In general, as energy availability increases, individual carrying capacities are higher, resulting in greater diversity at species and genetic levels (the more individuals hypothesis). As heterogeneity increases, species richness is higher due to the increased availability of niches. However, population sizes are reduced because niche area is smaller in more heterogeneous areas, generating a negative relationship between species richness and genetic diversity (heterogeneity hypothesis).



710

711 **Fig. 2.** Maps depicting spatial patterns of biodiversity and environmental factors. *(Top row)*
712 Points are the locations of 801 North American mammal populations for which raw
713 microsatellite data was available in public repositories. Point color indicates predicted values of
714 genetic diversity and species richness based on spatial patterns detected in the data. (*Bottom row*)
715 Maps showing the three environmental variables which we tested for simultaneous effects on
716 genetic diversity and species richness.

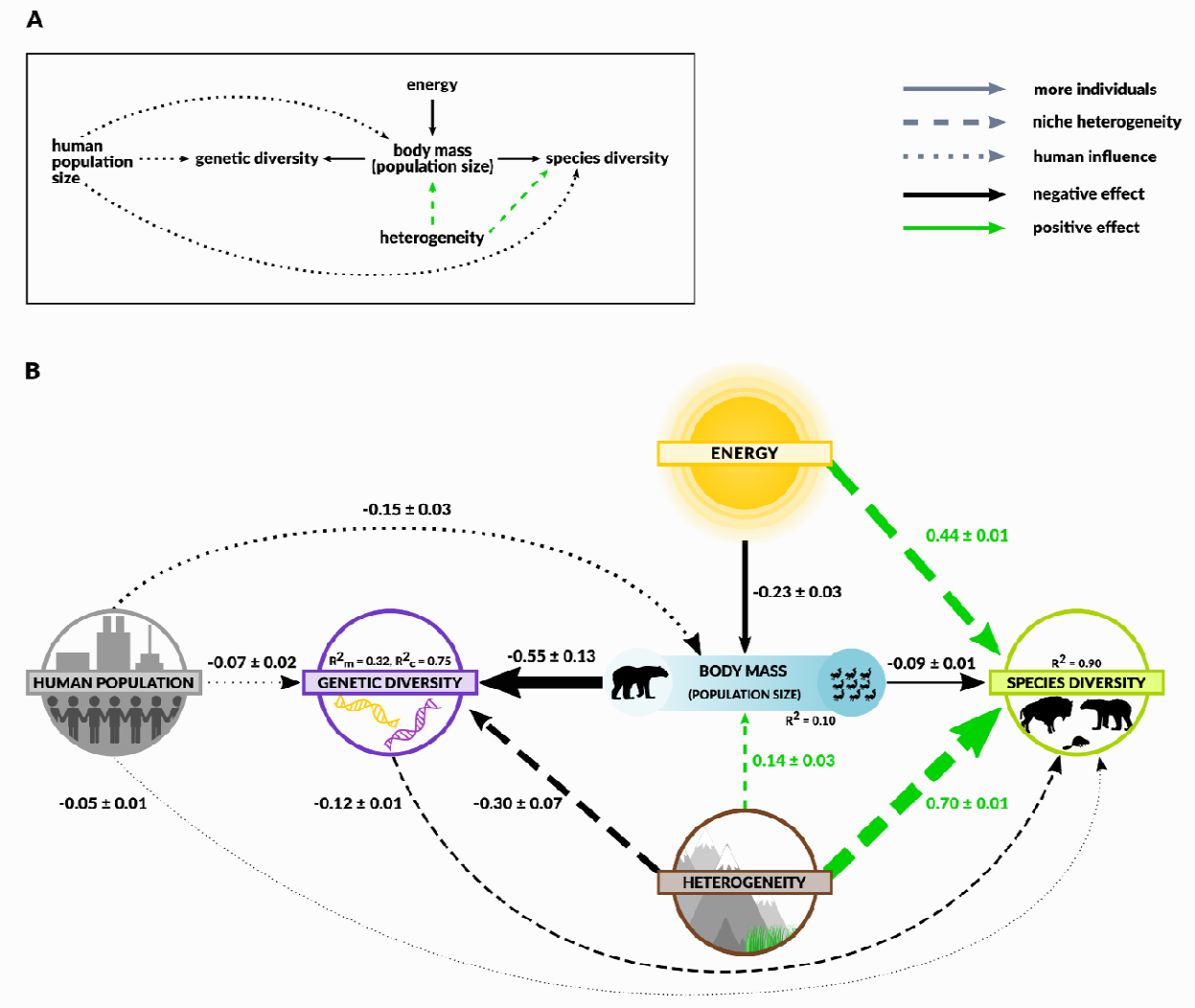


Fig. 3. Structural equation models. (a) Our conceptual hypothesis network combining the more individuals hypothesis (solid lines) with the effects of environmental heterogeneity (dashed lines) and human presence (dotted lines). Arrows represent unidirectional relationships between variables. (b) Structural equation model results. Green and black lines positive and negative relationships, respectively. Line widths reflect coefficient estimates, which are listed above each path with standard errors. R^2 values are the amount of variation explained for each response variable. Mass and species richness were measured at the species level, and genetic diversity was measured at the population level and fit with a random effect for species: R^2_m is the variation explained by fixed effects only, and R^2_c is the variation explained by fixed and random effects.