Systemic racism alters wildlife genetic diversity

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In the United States, systemic racism has had lasting effects on the structure of cities, specifically due to government-mandated redlining policies that produced racially segregated neighborhoods that persist today. However, it is not known whether varying habitat structures and natural resource availability associated with racial segregation affect the demographics and evolution of urban wildlife populations. To address this question, we repurposed and reanalyzed publicly archived nuclear genetic data from 7,698 individuals spanning 39 terrestrial vertebrate species sampled in 268 urban locations throughout the United States. We found generally consistent patterns of reduced genetic diversity and decreased connectivity in neighborhoods with fewer White residents, likely because of environmental differences across these neighborhoods. The strength of relationships between the racial composition of neighborhoods, genetic diversity, and differentiation tended to be weak relative to other factors affecting genetic diversity, possibly in part due to the recency of environmental pressures on urban wildlife populations. However, the consistency of the direction of effects across disparate taxa suggest that systemic racism alters the demography of urban wildlife populations in ways that generally limit population sizes and negatively affect their chances of persistence. Our results thus support the idea that limited capacity to support large, well-connected wildlife populations reduces access to nature and builds on existing environmental inequities shouldered by predominantly non-White neighborhoods.

urban evolution | redlining | effective population size | population isolation | Human Footprint Index

Historic and ongoing systemic racism and racial segregation have played a prominent role in the development and structure of cities in the United States (1, 2). One of the most direct causes of racial segregation was the government-sponsored practice of redlining, which graded neighborhoods based on desirability and systematically excluded racial and ethnic minorities from homeownership in better-ranked neighborhoods. During the suburb boom in the 1950s, discriminatory redlining policies and practices related to lending, insurance, zoning, and public housing collectively encouraged White Americans to move into new suburban communities and simultaneously pushed Black Americans and other racial and ethnic minorities to reside in urban cores (2). Lower-ranking redlined neighborhoods subsequently received less public investment and typically became densely populated, had more industrial infrastructure, and less green space (reviewed in 1, 2). Racial segregation and spatial isolation were often reinforced by physical barriers such as highways, railroad tracks, and sometimes walls (2). These practices, although outlawed in the Fair Housing Act of 1968, created a socially structured urban geography associated with socioeconomic and environmental inequality that persists in American cities today (2–4).

Ecological and Evolutionary Effects of Systemic Racism

Accumulating knowledge of the effects of systemic racism on the structure of urban environments now allows us to explore its effects on the ecology and evolution of urban wildlife. In a comprehensive review, Schell et al. (1) showed that socioeconomic decision-making and racial inequality have created environmental conditions that can alter the distribution and demography of wildlife in cities in ways that should cause evolutionary change (1, 5). However, these ideas have received little empirical attention. Residential segregation creates race-based disparities in natural resource availability, land use, pollution, and habitat connectivity, such that neighborhoods that historically excluded minorities tend to be better wildlife habitat (1). This means that local environmental carrying capacities for wildlife in cities are likely predicted by the racial makeup of neighborhoods. In general, larger, more connected populations have better chances of persisting because they are less strongly affected by demographic stochasticity and genetic drift and have higher genetic diversity. Cities are now the primary place where people interact with and benefit from nature (6), and their design is becoming increasingly important for the conservation of native biodiversity (7). Managing wildlife for conservation and human well-being requires a comprehensive

Significance

Residential racial segregation is associated with variation in habitat disturbance across cities in the United States. We repurposed open genetic data from 39 amphibian, bird, mammal, and reptile species sampled at 268 locations in cities across the United States and show that genetic diversity and connectivity are reduced in wildlife populations inhabiting minority neighborhoods. This suggests that these animal populations have slightly lower chances of persisting long term, limiting access to biodiversity in minority communities. Consistent, small effects across species indicate that the consequences of segregation on wildlife may be mitigated by more equitable urban development. By recognizing the ways human social factors negatively affect people and wildlife, we can change our behavior for the benefit of society.

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understanding of eco-evolutionary processes in cities, and this extends to identifying the ways that human social patterns shape evolution in urban wildlife.

We test the hypothesis that systemic racism produces associations between neighborhood racial makeup and urban environments that alter population demography and thus evolutionary change in city-dwelling populations of amphibians, birds, mammals, and reptiles across the continental United States. It is now clear that urbanization and human land use generally affect the genetic composition of wildlife populations when compared to populations in more natural environments, although directions of effect can vary (8–10). How ecological and evolutionary processes shape genetic diversity within cities is less well understood. We predicted that levels of genetic diversity and connectivity among urban wildlife populations would vary with the racial composition of neighborhoods and increase in predominantly White, less environmentally disturbed areas. The effect of systemic racism on ecological and evolutionary change in urban wildlife will be likely mediated by differential resource distribution and habitat degradation (1). We explored this idea by testing the effects of the racial composition of neighborhoods on genetic diversity alone, and while statistically controlling for habitat quality measured by the Human Footprint Index (11). The Human Footprint Index and racial segregation broadly capture the intensity of urbanization, thus we predicted effects would be consistent across species. Other features of urbanization, as well as factors unrelated to urbanization, will also shape contemporary genetic diversity—often in species-specific ways. These species-specific questions were not our focus here.

Residential segregation has also contributed to the present marked wealth disparities across racial groups in the United States (12, 13). The ecological effects of wealth on wildlife can be notable. Wealth, or the luxury effect, alters habitat quality, population dynamics, and the species composition of urban wildlife communities. However, the strength of the luxury effect varies regionally and across taxa (1, 14, 15). Additionally, the greatest disparities in urban forest cover across the racial mosaic appear on public, rather than private land, reflecting the effect of biased municipal investment in communities rather than the effects of individual wealth (3). For these reasons, the environmental correlates of structural racism cannot be captured by neighborhood wealth alone, and here we focus on neighborhood habitat quality more generally.

Quantifying Genetic Diversity in Terrestrial Vertebrates

We tested the hypothesis that genetic diversity would decrease and population differentiation would increase with the proportion of non-White residents in neighborhoods by building a database of georeferenced publicly archived, raw, neutral microsatellite data sets [Fig. 1, *Materials and Methods*, and (10)]. We aggregated 7,698 individual genotypes from 8 amphibian, 14 bird, 15 mammal, and 3 reptile species native to North America. The median number of individuals sampled at each site was 23 (range, 5–233 individuals; *SI Appendix,* Table S1). Genetic diversity measured with microsatellites is strongly correlated with genome-wide diversity [$R^2 = 0.83$, (16)]. Due to a lack of suitable archived whole genome data, we focused on microsatellite data and conducted a systematic search for data in online data repositories in R (17) using a list of terrestrial vertebrate species native to North America (*Materials and Methods*). By repurposing raw data, we were able to consistently estimate our chosen metrics of genetic composition and environmental variation across the entire dataset. For each sample site, we estimated the effective population size of the
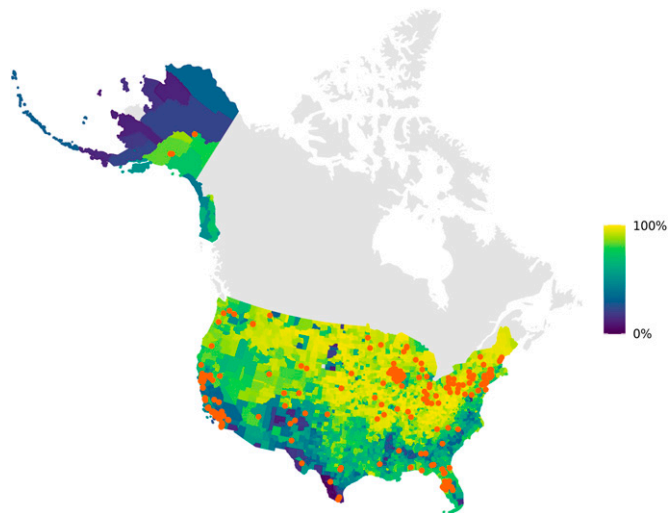

Fig. 1. Map of 268 sample sites for 39 species of amphibian, bird, mammal, and reptile located in urban areas in the continental United States (points). Racial composition, measured by the proportion of the population identifying as White according to 2010 US census data, is depicted at the county level.

parental generation using a linkage disequilibrium method (18), gene diversity (19), standardized allelic richness, and genetic divergence using site-specific F_{ST} [fixation index, (20)]. The effective population size is an estimate of the strength of genetic drift a site experiences. Gene diversity and allelic richness are two measures of genetic diversity, and site-specific F_{ST} was our estimate of relative genetic differentiation among sites. We excluded sites not located within US Census-designated urban areas (21).

We measured the percentage of residents identifying as White in census blocks located within 0.5, 1, and 5 km of each sample site in our dataset using demographic data from the 2010 US Census (22). We present results from 0.5 km buffers here, but note results were consistent across all scales (*SI Appendix,* Fig. S1). We chose this metric of segregation because White Americans are the most racially separated demographic. According to the 2010 Census, the average White American lives in a predominantly White neighborhood, while other racial groups typically live in more diverse neighborhoods (23). Because the goal of redlining policies was to maintain homogeneous, predominantly White communities (2), the proportion of White residents in a neighborhood should broadly capture variation in environmental heterogeneity regardless of regional histories that have shaped the racial composition of neighborhoods in different ways. To test whether the well-established relationship between neighborhood racial composition and habitat disturbance held for our sample sites, we quantified disturbance at each site with the Human Footprint Index (11). The Human Footprint Index measures human-caused habitat transformation from the most wild to the most disturbed. It provides a broad index of habitat degradation by incorporating human population density, roads, railways, access to navigable rivers, built-up areas, land cover, and nighttime lights. The percentage of White residents in a neighborhood was negatively correlated with the Human Footprint Index at our sample sites (Pearson's $r = -0.52$; 95% confidence interval, -0.60 to -0.43), demonstrating that in our dataset, predominately non-White neighborhoods were located in more disturbed environments.

We tested the relationship between the racial composition of people and the genetic composition of species at sample sites using Bayesian hierarchical models [generalized linear mixed models (GLMM) and (24)]. We controlled for variation across taxonomic class and species using a random effect structure

with random intercepts for species nested in class and allowed slopes to vary across species (*Materials and Methods*). Here, random slope and intercept models estimate the effect of racial composition on each species, and the distribution of species-specific parameter estimates shrink toward an overall mean or the effect size across all species. This is a feature of hierarchical models that is valuable in a multispecies context. Shrinkage allows random effect groupings to inform each other, yielding more robust estimates of effect size, while estimating general effects across species that may be difficult to detect in single-species analyses (25). Moran's *I* tests detected no residual spatial autocorrelation in the models, indicating that our models captured spatial variation in genetic diversity well. This suggests there was no detectable spatial structure in model residuals caused by spatial variation in environments or regional histories. The whiteness of a neighborhood captures the environmental variation we are interested in here well.

Effects of Racial Segregation on Genetic Variation

We detected consistent relationships between the genetic composition of wildlife and the racial composition of neighborhoods (Figs. 2 and 3). Species tended to have larger effective population sizes, higher genetic diversity, and were less genetically differentiated in neighborhoods with higher proportions of White residents (Fig. 3, Table 1, and *SI Appendix*, Figs. S1 and S2). These patterns were visible from raw data comparing sites located in majority White (>50% White residents) vs. majority non-White residents (Fig. 2). For example, comparing species-level medians, gene diversity decreased by ~5% and effective population size by

45% across all taxa in majority non-White neighborhoods (Fig. 2). Our GLMMs controlling for variation at the species and class levels reflected these patterns in the raw data: effects of segregation were consistently in the predicted direction (Fig. 3, Table 1, and *SI Appendix*, Table S3). We used the breadth and position of credible intervals in aggregate across species and for each species individually to assess the effects of racial segregation (Table 1, Fig. 3, and *SI Appendix*, Fig. S2). Effects were remarkably consistent across species, with clear general directions of effect, although species-specific effect sizes varied in strength and, rarely, in direction (Fig. 3 and *SI Appendix*, Fig. S2). Overall effects suggested the effects of segregation on genetic composition were weak (range of model slope coefficients: –0.21 to 0.11; Table 1 and Fig. 3). Our results provide evidence suggesting that demographic and evolutionary processes in urban wildlife vary within US cities in ways that make population persistence in minority neighborhoods more difficult. As expected, most variations in the genetic data were associated with species and class level variation captured in our random effects (conditional R^2, 34–89%; Table 1). Fixed effects associated with racial composition accounted for ~1–4% of variation in our models (marginal R^2; Table 1). These proportions of explained variation clearly show that variation in species and class-level demographic histories and species-specific interactions with environments are well controlled for in our models and are more important determinants of contemporary genetic diversity and differentiation than effects associated with racial segregation alone.

We then explored whether the effects of neighborhood racial composition might be mediated by habitat quality. To test this idea, we fit separate models relating the Human Footprint Index alone, and both the Human Footprint Index and the racial
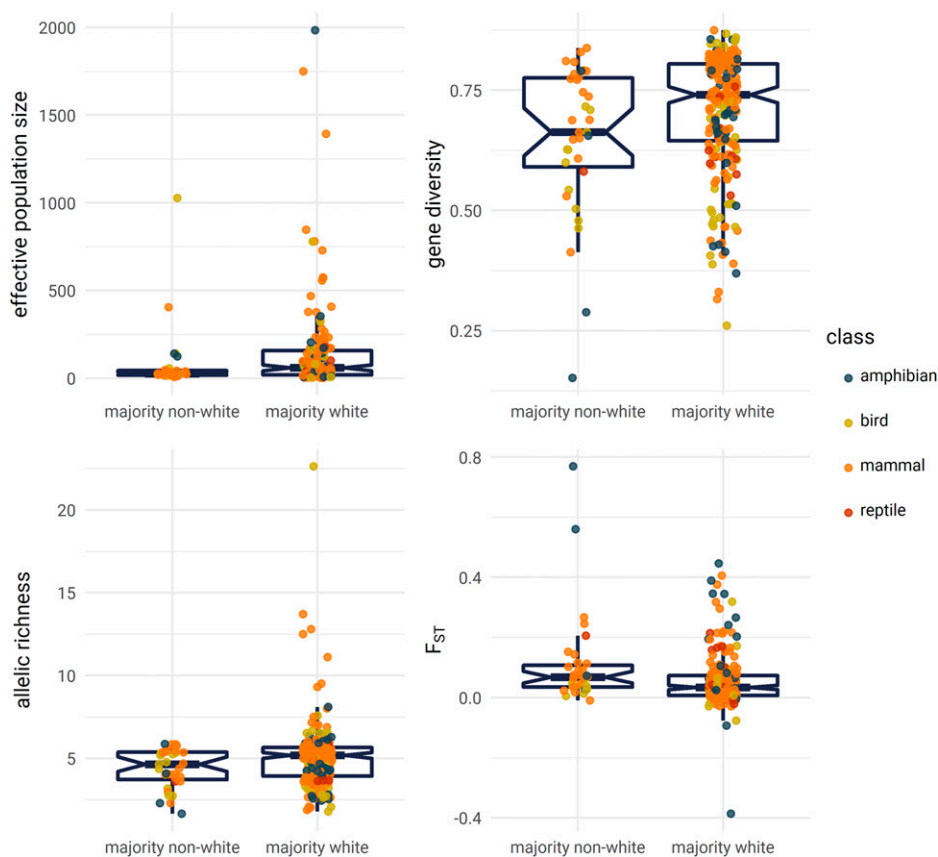

Fig. 2. Boxplots comparing data for genetic metrics sampled in sites with either majority (>50%) White or non-White residents. An outlier was omitted from effective population size for the purposes of visualization. Note that values for population-specific F_{ST} can be negative. Notches are non-overlapping for all genetic metrics, and patterns were consistent with outputs from GLMMs accounting for species variation (see Fig. 3).
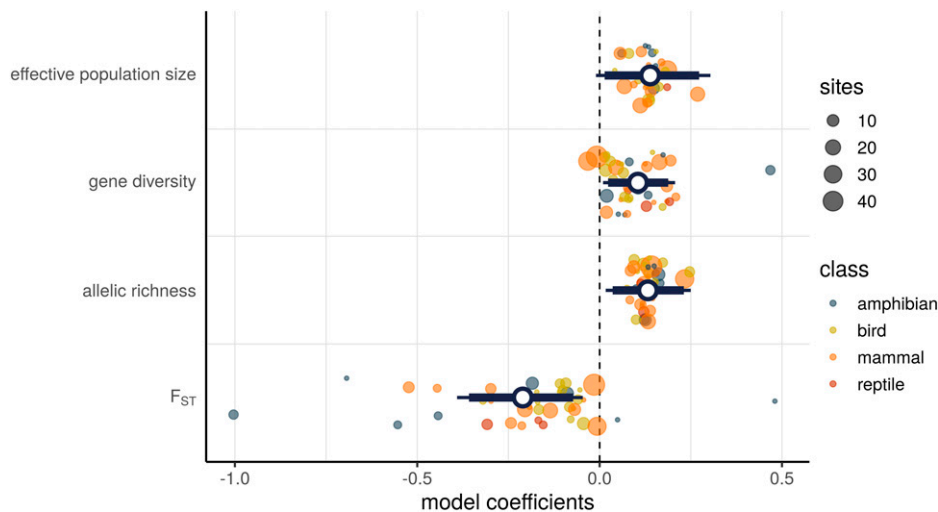
Fig. 3. Bayesian GLMM coefficients for the effect of racial segregation, measured as percent of White residents in a neighborhood, on genetic composition. Coefficient estimates (open circles) are shown with 90% (bold lines) and 95% (narrow lines) credible intervals. Species-specific slope estimates are shown by filled circles where the diameter is proportional to sample size (number of sites), and color corresponds to taxonomic class.

composition of neighborhoods together, to our measures of genetic composition. We used adjusted R^2 values, which account for different numbers of predictors, to determine whether models including both racial composition and the Human Footprint Index explained more variation than either covariate alone. The Human Footprint Index was negatively related to genetic diversity, effective population size, and connectivity (Table 1). For all genetic metrics, the proportion of variance explained by models including both racial composition and the Human Footprint Index was similar to the variation explained using only one of these covariates (Table 1). Thus, adding Human Footprint to our models did not improve explanatory ability. These results suggest that racial composition and the Human Footprint Index both capture the same underlying process. It seems likely that the combination of systemic racism and the development of urban landscapes are responsible for the patterns we find. The urban landscape is a dynamic product of human choices. By actively choosing to develop certain areas (2), forcing minorities to live in undesirable areas (2, 26), preferentially investing in greening majority White neighborhoods (3, 4, 27), or placing industrial facilities near communities of color (28), systemic racism contributes to the structure of the urban landscape, which in turn shapes wildlife demography in cities.

Our results suggest that neighborhoods that are largely non-White support smaller, more fragmented, less genetically diverse wildlife populations. Notably, the effects of racial composition and habitat degradation on genetic composition were consistent across the majority of taxa regardless of urban sample locality (Fig. 3). Previous multispecies work along urban–rural gradients suggested that mammal populations were generally negatively affected by increasing human disturbance, but responses in birds were species-specific with both increases and decreases in diversity detected (10), and no effects were detected across amphibians (29). When considering habitat variation within US cities, it appears urban wildlife populations, regardless of taxa or location, tend to be larger and harbor higher genetic diversity in the less disturbed habitat patches of predominantly White neighborhoods.

Cities are new habitats, which means that declines in urban wildlife are recent and populations are unlikely to be at mutation-drift equilibrium. Genetic diversity loss following declines in abundance accumulates for many generations before it stabilizes (30), and rates of genetic diversity loss due to habitat loss and fragmentation tend to be nonlinear, with slow but increasing rates of genetic erosion through time (30, 31). Thus, we are currently at a point where signatures of genetic erosion are expected to be weak. If urban populations are out of mutation-drift equilibrium, we likely underestimate the eventual effect of urbanization and racial environmental disparities on genetic variation and population persistence. To place our findings in a more general context, an estimated 6% of genetic diversity has on average been lost from wildlife populations globally since the industrial revolution [~200 y ago and (9)], and the enactment of redlining occurred even more recently (~100 y ago). The small effect sizes we report suggest there is still time to make positive change with environmental interventions that promote gene flow from more genetically diverse populations across the urban racial mosaic.

Urban evolutionary ecology research is only beginning to explore the effects of spatial heterogeneity within cities generated by human social processes (1, 5). In our dataset, 87% of sites were located in predominantly White neighborhoods (>50% residents identifying as White). If we are to fully consider environmental heterogeneity within and across cities to understand the spectrum of ways humans affect their environments, more informed, comprehensive sampling of urban habitats is needed. Research in urban evolutionary ecology will become an increasingly important resource for decision-makers and city planners to make cities sustainable habitats for wildlife while meeting human needs (5). Environmental crises like climate change and biodiversity loss disproportionately affect non-White communities (1). Achieving environmental equity, and ultimately cities that support humans and wildlife alike, will require engagement and solutions from a diverse research community. Yet, racial diversity is enduringly low in ecology and evolution (32, 33), fostering blind spots that hold back progress in research that intersects with environmental justice (34).

We have shown that the well-described environmental patterns associated with historic and ongoing racial segregation in US cities (1, 2) have caused parallel patterns in wildlife demography detectable with genetic data. It is clear that systemic racism is altering the demography of urban wildlife populations on a national scale in ways that can shape the evolutionary processes acting on them and the probability of long-term persistence in cities. These results are concerning because urban biodiversity is important for human mental and physical well-being (35), and disparities in access to

nature build on existing health-related environmental disamenities in predominantly non-White neighborhoods (1, 36). Equitably distributing and increasing the amount and connectivity of natural habitat in cities can therefore benefit human well-being while simultaneously helping build resilience in urban wildlife.

Materials and Methods

Data Compilation. To create the database of genetic metrics (37), we performed three systematic searches of online data repositories between 2018 and 2020 using the DataONE interface for R (38) with the keywords "str," "microsat*," single tandem*," "short tandem*," and species name (e.g., *Alces alces*"). DataONE is a network providing access to multiple public data repositories, such as the Dryad Digital Repository (https://www.datadryad.org). We used existing datasets described in (10, 29) where detailed methods for dataset assembly can be found. We augmented this dataset in February 2019 with data from reptiles and in November 2020 with additional mammal data using the same inclusion criteria (*SI Appendix*, Tables S1 and S2). In brief, we retained search results with neutral microsatellite datasets sampled from native species located in North America where study design would not influence genetic diversity (e.g., island or managed populations). In using previously published data, we assumed that the authors of the original studies have selected appropriate markers for their work. We retrieved 68 total search results for reptiles, 28 of which were duplicates. In total, 11 datasets met our inclusion criteria. For additional mammal data, we obtained 37 search results, of which 10 were duplicates and 8 were added to our database. We measured effective population sizes, allelic richness, gene diversity, and population-specific F_{ST} for each sample site from raw microsatellite datasets. We estimated effective population size of the parental generation using the linkage disequilibrium method in NeEstimator (18). We were unable to estimate effective population size when sampling error overwhelmed signals of genetic drift, as is the case when too few individuals were sampled or populations were extremely large. We estimated allelic richness and gene diversity using the "allelic.richness" function in the hierfstat package (39) and the "Hs" function in the adegenet packages, respectively, in R (40). Allelic richness is sensitive to the number of sampled individuals, thus we standardized this measure to the minimum sample size across the entire dataset [five individuals and (41)]. Gene diversity (19) is a heterozygosity metric that is minimally affected by sample size variation (42). Finally, population-specific F_{ST} (20) is a relative measure of genetic differentiation that estimates how far populations have diverged from a common ancestor in a sample. We estimated this metric with the "betas" function in hierfstat and note that it can only be measured when at least two populations were sampled in the original dataset.

Because this study focuses on the effects of human demographics within cities, we excluded non-urban sites from this analysis based on whether they were located within the boundaries of census-designated urban areas (21). Data from

43 studies were ultimately included, and the final dataset consisted of 380 sites across all taxa (Table 1 and *SI Appendix*, Table S2). Of these, we were able to estimate gene diversity and allelic richness at all sites, site-specific F_{ST} at 373 sites, and effective population size at 285 sites. The datasets included a site-level measure of the Human Footprint Index (11) from previous analyses (10, 29). We then obtained demographic data from the United States Census Bureau through the IPUMS National Historical Geographic Information System (22). Demographic data are from census blocks, the smallest census geographic unit. For each site, we measured the percent of residents identifying as White within three buffer sizes: 0.5, 1, and 5 km. Note sample sizes differed across these scales when sites were not located near populated blocks within the designated buffer size (e.g., gene diversity $n_{0.5km} = 268$ sites, $n_{1km} = 283$ sites, and $n_{5km} = 296$ sites).

Statistical Analysis. All analyses were conducted in R version 3.6.3 (17). To test for the effects of residential racial segregation (% White residents in neighborhood) on the genetic diversity of wild populations we used Bayesian linear mixed models implemented in the brms package (24). We log-transformed effective population size and scaled and centered all variables prior to analysis.

Our modeling strategy incorporated a random effect structure to account for variation across taxonomic class and species. We included random intercepts for species nested in class, allowing slopes to vary among species. Random slope models provide more conservative parameter estimates due to shrinkage, where the distribution of group-level effects are drawn toward the overall mean effect (25). Shrinkage to the overall effect is strongest for groups with fewer observations, allowing them to borrow strength from better sampled-groups. In this way, knowledge is shared across grouping levels of a random factor because we assume they are drawn from a common statistical population–whereas in fixed-effect only models, groups are assumed to be independent. The benefits of shrinkage in random slope and intercept models are especially salient from a macrogenetics perspective. Species- or city-specific analyses often yield varying results [e.g., (43)], but when analyzing raw data aggregated across broader spatial or taxonomic contexts, random slope and intercept models can provide better estimates of general effects.

We treated previous results from a different dataset showing the effect of the Human Footprint Index on mammal gene diversity, allelic richness, effective population size, and population-specific F_{ST} (10) as suitable priors given the negative correlation between the percentage of White residents in a neighborhood and the Human Footprint Index. We therefore hypothesized that the direction of effect for the percentage of White residents would be positive for effective population size, gene diversity, and allelic richness, and negative for population-specific F_{ST}. Because all genetic metrics are centered and scaled, we predicted that effect sizes would fall between 0 and 1 for effective population size and diversity metrics, and between −1 and 0 for population-specific F_{ST}. We thus assigned slightly informative normally distributed priors with mean 0.5 and 0.25 SD for effective population size, gene diversity, and allelic richness. For site-

Table 1. Effects of racial segregation and environmental disturbance (the Human Footprint Index) on genetic composition

Variable	Covariate	Coefficient (95% CI)	R^2_m; R^2_c	R^2_{Adj}	Moran's I (P)
Allelic richness	Racial segregation (% White residents)	0.13 (0.02, 0.25)	0.02; 0.46	0.43	−0.01 (0.68)
$n = 268$ sites	Human Footprint Index	−0.12 (−0.25, 0.01)	0.02; 0.46	0.43	0.02 (0.80)
	Both	—	0.03; 0.47	0.43	−0.02 (0.82)
Gene diversity	Racial segregation (% White residents)	0.11 (0.01, 0.21)	0.01; 0.88	0.84	−0.00 (0.46)
$n = 268$ sites	Human Footprint Index	−0.13 (−0.24, −0.04)	0.02; 0.88	0.83	−0.01 (0.31)
	Both	—	0.02; 0.89	0.84	−0.01 (0.55)
Effective population size	Racial segregation (% White residents)	0.14 (−0.01, 0.30)	0.02; 0.34	0.24	−0.02 (0.73)
$n = 202$ sites	Human Footprint Index	−0.14 (−0.31, 0.03)	0.02; 0.36	0.25	−0.02 (0.77)
	Both	—	0.04; 0.37	0.24	−0.02 (0.81)
F_{ST}	Racial segregation (% White residents)	−0.21 (−0.39, −0.05)	0.04; 0.68	0.49	−0.02 (0.67)
$n = 264$ sites	Human Footprint Index	0.24 (0.03, 0.49)	0.05; 0.68	0.43	−0.02 (0.67)
	Both	—	0.06; 0.70	0.48	−0.02 (0.78)

Coefficient estimates are given with 95% credible intervals. Marginal R^2 (R^2_m) is the proportion of variation explained by fixed effects relative to that of random effects, and conditional R^2 (R^2_c) is that explained by fixed and random effects. Adjusted R^2 (R^2_{Adj}) is an estimate of variation explained taking into account model complexity. Moran's I index of spatial autocorrelation ranges between –1 and 1 with 0 indicating no autocorrelation; P values are given for permutation tests on model residuals.

specific F_{ST}, we assigned normal priors with mean –0.5 ± 0.25 SD. We tested model sensitivity to these priors by conducting parallel analyses with two additional, weaker priors for the effect of percentage of White residents (*SI Appendix,* Table S3). For effective population size, gene diversity, and allelic richness, we used a normal prior centered on mean 0 ± 1 SD; and a Student's *t* prior with mean 0.5 ± 0.25 SD and 3 degrees of freedom. Signs for prior distribution means were inverted for F_{ST}. Normal(0, 1) priors are generic and equally weight negative and positive parameter estimates; our priors with a Student's *t* distribution have the same mean and SD as our normal priors but have much heavier tails than a normal distribution and are therefore weaker than the Normal(0.5, 0.25) priors. We report prior quality ("informative" or "uninformative") based on Gelman's method in the "check_prior" function in the "bayestestR" package (44). Parameter estimates for the effect of percentage of White residents and 95% credible intervals were consistent across all priors for each genetic variable (*SI Appendix,* Table S3). We also report the probability of direction of effect measured with the function "pd" in bayestestR, which is the proportion of values in the posterior distribution that have the same sign as the median value. Across genetic response variables and prior distributions, all models had >89% probability that the effect was in the detected direction (*SI Appendix,* Table S3). Most models, including those reported in the main text, have >95% probability. Thus, models were not strongly affected by our choice of informative priors.

We ran all models with four chains and minimum 3,000 iterations. We tested for spatial autocorrelation in model residuals with Moran's *I* tests in the package adespatial (45). Here, spatially autocorrelated residuals would indicate the presence of residual dependencies in genetic composition potentially due to population structure (e.g., isolation by distance patterns) or spatial structure induced by the environment not captured by model predictors (i.e., effects specific to cities or regions or broader gradients in city structure). We used marginal and conditional R^2 to estimate the proportion of variation explained by fixed and fixed and random effects, respectively [Table 1 and (46)]. Next, we used the same modeling approach to test for the effects of Human Footprint Index alone, and the joint effects of racial segregation and the Human Footprint Index on genetic composition in another series of models. Finally, to compare explanatory ability between univariate models and models including both racial composition and Human Footprint Index, we used adjusted R^2 values. Adjusted R^2 for Bayesian models estimates the amount of variation explained using leave-one-out cross validation taking into account model complexity. If models including both covariates explain more variation than models with either covariate, this suggests that the effect of neighborhood racial composition and environmental disturbance on genetic composition are to some extent independent. If the opposite is true, it is more likely that racial composition affects genetic composition due to its correlation with environmental disturbance.

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