ARTICLES
https://doi.org/10.1038/s41559-022-01723-0

nature ecology & evolution

Check for updates

Urban-adapted mammal species have more known pathogens

Gregory F. Albery[1]✉, Colin J. Carlson[2,3], Lily E. Cohen[4], Evan A. Eskew[5], Rory Gibb[6,7], Sadie J. Ryan[8,9,10], Amy R. Sweeny[11] and Daniel J. Becker[12]

The world is rapidly urbanizing, inviting mounting concern that urban environments will experience increased zoonotic disease risk. Urban animals could have more frequent contact with humans, therefore transmitting more zoonotic parasites; however, this relationship is complicated by sampling bias and phenotypic confounders. Here we test whether urban mammal species host more zoonotic parasites, investigating the underlying drivers alongside a suite of phenotypic, taxonomic and geographic predictors. We found that urban-adapted mammals have more documented parasites and more zoonotic parasites: despite comprising only 6% of investigated species, urban mammals provided 39% of known host–parasite combinations. However, contrary to predictions, much of the observed effect was attributable to parasite discovery and research effort rather than to urban adaptation status, and urban-adapted species in fact hosted fewer zoonotic parasites than expected on the basis of their total parasite richness. We conclude that extended historical contact with humans has had a limited impact on zoonotic parasite richness in urban-adapted mammals; instead, their greater observed zoonotic richness probably reflects sampling bias arising from proximity to humans, supporting a near-universal conflation between zoonotic risk, research effort and synanthropy. These findings underscore the need to resolve the mechanisms linking anthropogenic change, sampling bias and observed wildlife disease dynamics.

As the rate of infectious disease emergence continues to rise, it is becoming increasingly important to identify and understand the drivers of zoonotic risk in wild animals[1–3]. Humans are rapidly altering patterns of wildlife disease through a combination of climate change and land conversion, both of which are expected to drive increased spillover (that is, interspecific transmission of parasites from animals into humans[2–8]). Urban environments in particular are expected to facilitate the emergence of zoonotic pathogens in wildlife[3,7,9–11] because of a combination of impaired immune systems fed by anthropogenic resources[10,12] and greater pollution[13] as well as increased proximity of wild animals to humans[7,14]. This combination of factors is likely to become even more problematic in the future as the world's population continues to rapidly grow and urbanize[15–17].

Previous meta-analyses have uncovered elevated stressors and greater parasite burdens or parasite diversity in urban animals, with the general expectation that the urban environment weakens host immune responses[5,9,11]. However, these studies usually comprise relatively few examples spread across a small selection of animal species, reducing their ability to generally address the question of how urbanization affects zoonotic disease risk. Moreover, the results of such analyses have been equivocal, with both positive, negative and neutral effects of urban living on dimensions of wildlife disease[5,9,11]. Testing whether urban-adapted mammal species exhibit greater zoonotic risk in a broad-scale, pan-mammalian analysis could provide more general answers to this question, informing the design of parasite-sampling regimes and efforts to mitigate zoonotic disease risk in humans.

A recent pan-mammalian study used a literature review to build a database of mammal species' urban adaptation status (that is, their ability to live off urban resources[18]), which they then linked with species-level phenotypic traits. Although different traits were important for different mammalian orders, species with larger litters were generally more likely to be urban adapted. This relationship could explain the common observation that fast-lived host species (that is, those that favour reproduction over survival) tend to disproportionately source zoonotic parasites[3,14,19]. Complicating matters, a given species' observed parasite diversity depends inherently on the effort that has been directed towards examining it[20–23]. Such research effort is heterogeneously distributed in space[20,24,25] and across mammal species, particularly with regards to life history[14] and taxonomy[20,21]. As such, sampling bias could be important in mediating observed trends among urbanization, life history and zoonotic parasite diversity. In particular, urban mammal species may have more zoonoses (as a proportion of their known parasite richness) because historic contact with humans has allowed more parasites to spill over into humans and be observed. Although it has been shown that human-adjacent animals have both more parasite species and more zoonoses[5], it is unclear yet whether human contact has filtered them to produce disproportionately more observed zoonoses in urban species.

In this Article, we take a macroecological approach to investigate: (1) whether urban-affiliated mammal species have more zoonotic parasites and (2) whether urban-affiliated mammal species harbour more zoonotic parasites than expected given their overall

[1]Department of Biology, Georgetown University, Washington, DC, USA. [2]Center for Global Health Science and Security, Georgetown University Medical Center, Washington, DC, USA. [3]Department of Microbiology and Immunology, Georgetown University Medical Center, Washington, DC, USA. [4]Icahn School of Medicine at Mount Sinai, New York, NY, USA. [5]Department of Biology, Pacific Lutheran University, Tacoma, WA, USA. [6]Centre for Mathematical Modelling of Infectious Diseases, London School of Hygiene and Tropical Medicine, London, UK. [7]Centre on Climate Change and Planetary Health, London School of Hygiene and Tropical Medicine, London, UK. [8]Quantitative Disease Ecology and Conservation (QDEC) Lab Group, Department of Geography, University of Florida, Gainesville, FL, USA. [9]Emerging Pathogens Institute, University of Florida, Gainesville, FL, USA. [10]School of Life Sciences, University of KwaZulu-Natal, Durban, South Africa. [11]University of Edinburgh, Ashworth Laboratories, Edinburgh, Scotland. [12]Department of Biology, University of Oklahoma, Norman, OK, USA. ✉e-mail: gfalbery@gmail.com

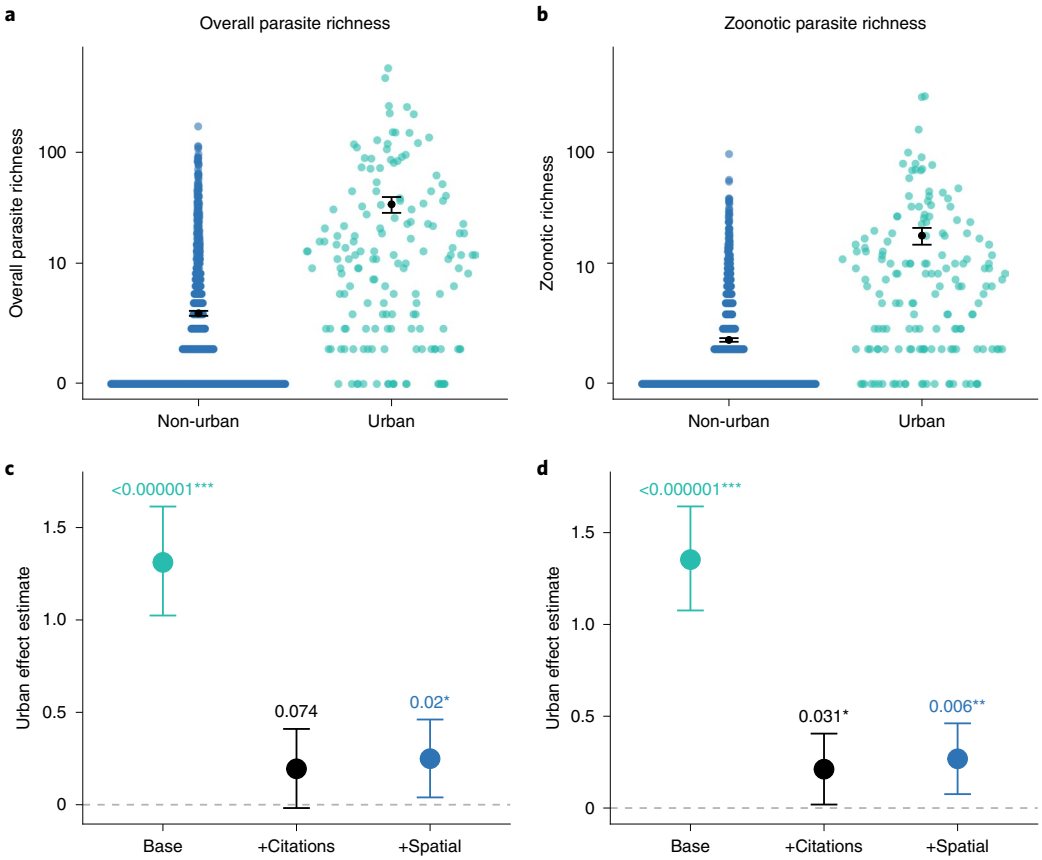

Fig. 1 | Urban-adapted mammals have more known parasites and zoonoses specifically. a,**b**, Parasite richness (**a**) and zoonoses richness (**b**) stratified by species that can capitalize on urban environments (Urban) and those that cannot (Non-urban). Each point represents a mammal species ($n = 2{,}792$ species). The *y* axis represents the species' known parasite diversity, on a $\log_{10}$ scale. Black dots and error bars are raw group means ± s.e.m. **c**,**d**, The urban adaptation effect for overall richness (**c**) and zoonotic richness (**d**) across multiple model formulations ($n = 2{,}792$ species). Base, model including all fixed effects except citation number; Citation, model including citation number; Spatial, model including both citation number and a spatially distributed SPDE random effect. Points are the mean of the posterior effect estimate distribution from the GLMMs and error bars are 95% CIs. Numbers above the error bars display *P* values, with asterisks denoting levels of significance (*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$).

parasite diversity. We anticipated that species capable of adapting to urban settings would host a higher diversity of known parasites, owing to greater susceptibility and more intense sampling effort, and that a disproportionately high number of these parasites would be known to be zoonotic as a result of their greater historical contact with humans. We further expected that urban adaptation status would account for some variation in the effects of life history traits on parasite richness, implying that fast-lived species more often transmit zoonotic parasites because they are more likely to inhabit urban environments in close proximity to humans[14].

Results

We ran a series of generalized linear mixed models (GLMMs) that broadly supported our prediction that urban-adapted mammals would have greater parasite richness. Our first model set examined parasite richness as a response variable, revealing that urban mammals have more known parasites (Fig. 1a and Supplementary Fig. 1), and more zoonoses specifically (Fig. 1b and Supplementary Fig. 2). This urban bias diminished substantially in magnitude when we added citation counts as an explanatory variable representing research effort (Fig. 1c); in the case of overall parasite richness, adding citation counts rendered the effect of urban adaptation non-significant ($P = 0.07$). Citation number was strongly positively associated with urban status, overall parasite richness and overall zoonotic richness (Figs. 1c and 2), as well as being significant for all parasite subgroups (Supplementary Figs. 4 and 5). We elaborated on these models by accounting for spatial patterns in parasite richness and sampling effort using a centroid-based stochastic partial differential equation (SPDE) effect. These effects improved model fit substantially (deviance information criterion threshold change (ΔDIC) > 150) and increased the magnitude and significance of the urban adaptation effects (Fig. 1c; $P = 0.018$ and $P = 0.006$). As such, we conclude that urban species have slightly higher parasite diversities when accounting for sampling effort and geographic heterogeneity.

To provide further insight into how histories of sampling may have shaped current patterns of observed pathogen richness across urban-adapted and non-urban species, we used our dataset to descriptively visualize historical pathogen discovery rates and publication effort trends between 1930 and 2015, following a recent study of mammalian viral discovery[26]. We find that fewer annual discoveries generally occur in urban species; however, because there are so few urban-adapted species (157 out of 2,792), these species have been, on average, much more intensely studied and with a higher parasite richness since the mid-1960s (Supplementary Fig. 7). Notably, differences in mean parasite richness between urban-adapted and non-urban species have continued to widen in the intervening years as the discrepancy in sampling effort has continued to grow (Supplementary Fig. 7). This finding suggests that higher observed parasite richness in urban-adapted species

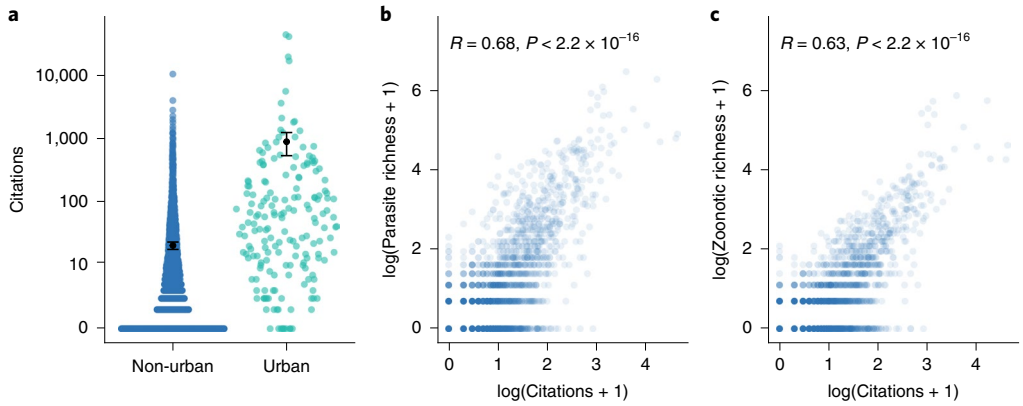

Fig. 2 | Citation numbers are higher in urban species and drive observed parasite and zoonotic parasite richness. a, Citation numbers in urban and non-urban species. Each coloured dot represents a species. *R* and *P* values are derived according to Spearman's rank correlations. Black dots and error bars are raw means ± s.e.m. ($n = 4{,}968$ species). **b**,**c**, Correlation between citation number and parasite richness (**b**) and zoonotic richness (**c**). See Fig. 1 for the slope estimates from the GLMMs for **b** and **c**.

is largely driven by long-term, accumulated differences in sampling effort.

We constructed a path analysis, which showed that urban adaptation was not associated with greater zoonotic richness when accounting for a direct effect of parasite richness; in fact, the estimated effect was slightly negative (Fig. 3; $P = 0.024$). In contrast, the indirect effect of urban adaptation on zoonotic diversity acting through parasite diversity was positive, substantial and significant (effect + 0.401; 95% credibility interval (CI) 0.116–0.749; $P = 0.004$) (Fig. 3). Taken together, these results imply that positive effects of urban adaptation on zoonotic diversity act largely through greater overall known parasite diversity, rather than by disproportionately elevating zoonotic parasite richness specifically. We performed multiple additional analyses to examine several dimensions of urban adaptation and sampling bias that could affect our results. There was no improvement in model fit when urban status interacted with host order, suggesting that the effect of urban adaptation on parasite diversity and zoonotic risk did not vary between mammal orders (ΔDIC < 5 relative to the base model). We built a generalized additive mixed model (GAMM) to next examine whether citation numbers had different effects for urban and non-urban species, but found no support for the interaction (ΔDIC < 5). Similarly, multivariate models revealed concordance between estimates for the effect of urban adaptation across parasite subtypes and implied that the urban effects were not being driven by specific groups of parasites. Finally, we used zero-inflated GLMMs to account for mammal species with no recorded parasites, demonstrating strong urban biases for the count component (that is, the number of parasites a mammal species hosted) as well as the inflation component (that is, whether the mammal species had greater than zero known parasites) (Supplementary Fig. 6). This finding implies that our results are not disproportionately driven by excess zeros produced by the inclusion of pseudoabsences (that is, species without any evidence of parasites).

A GLMM with different spatial fields for urban and non-urban species was not an improvement over the overall SPDE model (ΔDIC = 14.35 relative to the SPDE model). This implies that the bias towards greater parasite richness in urban species is relatively evenly distributed across the globe, rather than being focused in certain areas. These findings imply that our results were robust to geographic variation in parasite richness and revealed strong spatial patterns (Fig. 4c). We found no effect of absolute latitude (Fig. 4b), but we observed substantial between-continent variation in parasite diversity (Fig. 4b): North America was associated with the greatest

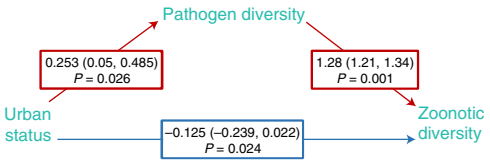

Fig. 3 | Path analysis revealed that urban-adapted mammals do not have more zoonoses than expected on the basis of their overall parasite diversity. Arrows denote hypothesized causal relationships. Red lines represent positive effects and blue lines represent negative effects. Other variables were included in the component linear models but are not displayed in this figure for clarity. Labels display the model effect estimates on the log-link scale, with 95% CIs in brackets and *P* values based on proportional overlap with zero.

parasite diversity, followed by Africa, then Eurasia, South America and Oceania.

Lastly, we also uncovered support for a range of other important species traits driving parasite richness (Fig. 4a). Most notably, faster life history was associated with greater (zoonotic) parasite diversity, according to the first principal component (PC1) (Fig. 4a). However, in the path analysis model, the effect of life history on zoonotic richness was supplanted by the inclusion of overall parasite richness (Supplementary Fig. 3). This finding reveals that, as with urban adaptation status, life history is associated with greater overall parasite richness rather than zoonotic richness specifically. There was substantial between-order variation in zoonotic and overall diversity (Supplementary Figs. 4 and 5), but adding a continuous phylogenetic similarity effect did not improve on the order-level effects (ΔDIC < 5). Diet diversity was positively associated with zoonotic richness but not with overall parasite richness (Fig. 4a). Phylogenetic distance from humans was negatively associated with zoonotic richness overall (Fig. 4a), with zoonotic richness of viruses and helminths and with overall richness of viruses and helminths; however, phylogenetic distance from humans was positively associated with overall richness of arthropods (Supplementary Figs. 4 and 5). Greater range area was associated with increased (zoonotic) parasite richness overall (Fig. 4a) and for many parasite subsets (Supplementary Figs. 4 and 5). Finally, domesticated species had more zoonotic helminths and protozoa (Supplementary Fig. 5) but did not differ in overall parasite richness from non-domesticated mammal species (Fig. 4a and Supplementary Fig. 4).

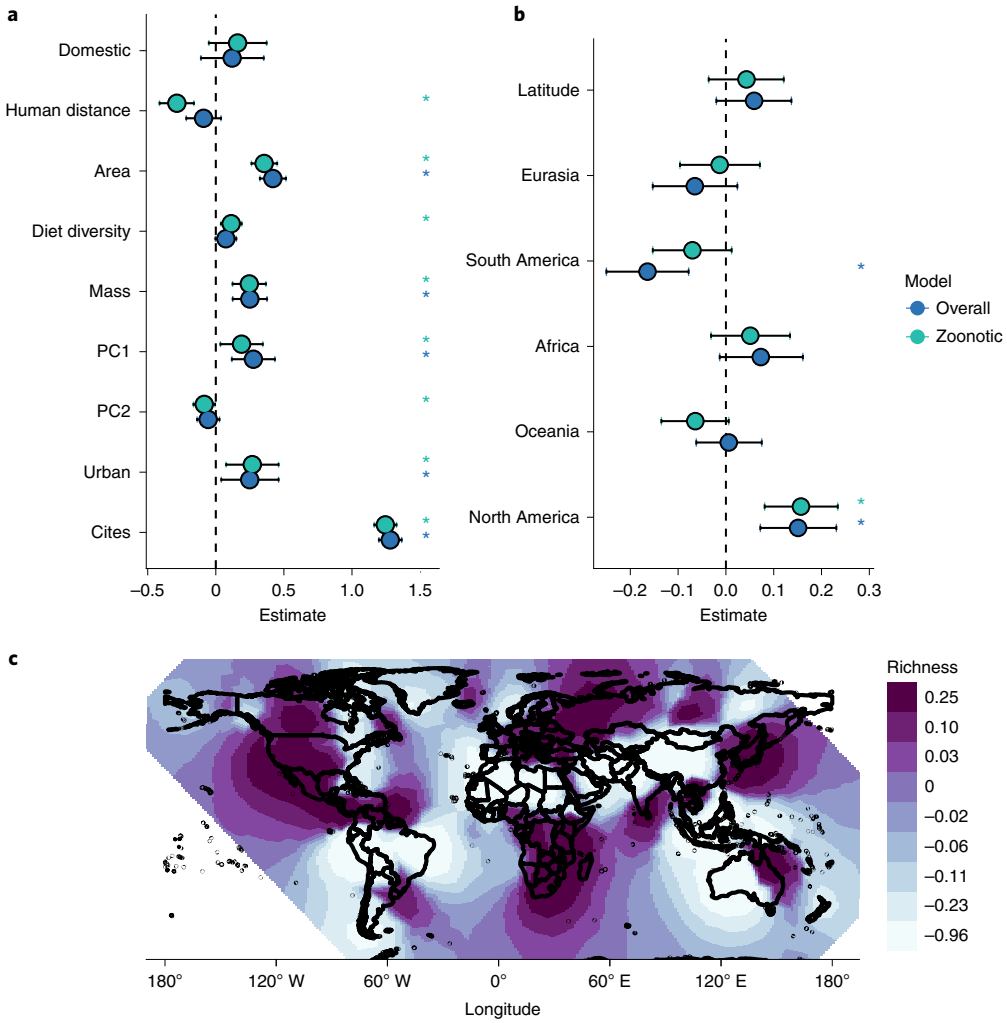

Fig. 4 | Model fixed-effect estimates and spatial effects on overall and zoonotic parasite richness. a, Fixed effects from the GLMMs for overall parasite richness and zoonotic richness, excluding order-level effects ($n = 2{,}792$). These models included an SPDE random effect to control for spatial autocorrelation. **b**, Fixed-effect estimates from the non-spatial GLMMs for overall parasite richness and zoonotic richness ($n = 2{,}792$). Points in **a** and **b** are the mean of the posterior effect estimate distribution from the GLMMs and error bars are the 95% CIs. Asterisks denote estimates that were significantly different from zero ($P < 0.05$). Order-level effects have been left out for clarity; see Supplementary Figs. 4 and 5 for full model effect estimates. **c**, Spatial distribution of the SPDE random effect, identifying hot and cold spots of parasite richness when non-spatial fixed effects (all effects except latitude and continent) are taken into account. Darker colours correspond to greater parasite richness. No marine data were used in our analysis; predictions that occur in the ocean are therefore largely extrapolations, but are necessary for model fitting to allow broad geographic modelling of spatial heterogeneity.

Discussion

Using a global pan-mammalian dataset of host species' traits and parasite associations, we found that urban-adapted mammal species have more known parasites, and in turn more zoonotic parasites, arising largely from research effort. This finding builds on recent work showing that wild animals with at least one known zoonotic parasite tend to inhabit human-managed landscapes[5], but we used a much broader dataset of urban-adapted mammals and applied a strict definition of urban adaptation based on long-term resource use and fitness in urban landscapes[18], while accounting for a correlated suite of phenotypic traits, research effort and geographic biases, including range size and phylogenetic relatedness to humans. Additionally, we were surprised to find that urban mammals' zoonotic richness was in fact lower than expected on the basis of their observed parasite richness. Our findings therefore do not support our main prediction that urban-adapted species host more known zoonotic parasites because they have had more historical contact with humans, creating more opportunities for the spillover of potentially zoonotic parasites[14]. Rather, urban species appear to have been preferentially sampled for non-zoonotic parasites, probably as a result of their proximity to humans and ease of sampling—that is, mammals in urban contexts might be more often spontaneously examined for parasites, while mammals in non-urban contexts are more likely to be examined specifically when they are suspected sources of zoonotic parasites. The reason for urban mammals' greater overall parasite richness remains uncertain, and many questions linger about the drivers of zoonotic diversity in urban wildlife. Most pressingly, why has human–wildlife contact not driven greater zoonotic diversity in urban species?

Sampling bias is one of few universal phenomena in ecological research[27,28], and understanding these biases is integral to designing interventions and predicting the consequences of global change. Our models revealed that urban-adapted species have been more thoroughly sampled for parasites than non-urban species, but in roughly similar patterns. Known urban status is highly geographically heterogeneous[18] and in a similar pattern to disease

surveillance[20,24,25], which we expected to be driving our perceived urban adaptation effect. The spatial patterns of parasite richness that we discovered mirror previously reported biases towards temperate, high-income countries[27,29] and were particularly high in North America, while being particularly low in South America, confirming that parasite biodiversity is substantially undersampled in the tropics[24]. This reflects the pattern of urban mammal diversity, which peaks at high latitudes and is low in South America, Southeast Asia and sub-Saharan Africa[18]. However, accounting for this heterogeneity in fact increased the urban bias estimate rather than decreasing it. Further, there was no significant interaction of urban adaptation with either the spatial effect or host order, implying minimal geographic and taxonomic bias in these urban-directed sampling processes. Finally, our temporal analysis revealed that urban and non-urban mammals have been subjected to similar trends in parasite discovery rate over the past century, with citation counts and parasite diversity following similar shapes throughout. The only analysis that implied a qualitatively different sampling trend in urban-adapted mammal species was our path analysis, which revealed that urban-adapted species have fewer known zoonotic parasites than expected from their observed parasite richness. Taken together, the evidence suggests that urban species are much better sampled for parasites than non-urban species, but with a stronger focus on non-zoonotic parasites, and this urban bias should be considered in future species-level analyses of zoonotic risk.

Even accounting for these layers of bias, our data retained a positive effect of urban status, suggesting that either: (1) urban mammals are subject to a specific sampling bias that could not be detected through our analyses, or (2) urban environments increase overall parasite diversity through effects on host immunity, behaviour and demography. Although these effects did not disproportionately increase zoonotic parasite diversity, urban mammals nevertheless host many zoonotic parasites as a result of their greater overall parasite richness, and therefore understanding this trend may be important for public health. Anthropogenic pollutants, altered nutrition and greater host densities in urban environments have been shown to weaken host immune systems and promote greater burdens and diversities of parasites when comparing hosts along urban–rural gradients[9,10]. Such intraspecific effects should accordingly scale up such that urban-adapted species have greater parasite richness than species that do not experience such immune impairments. Similarly, greater host densities and resource concentrations could facilitate elevated rates of density-dependent parasite transmission within and between species, rendering urban-affiliated species more likely to maintain parasites and resulting in greater observed parasite diversity[30]. However, there is some evidence that urban wildlife might exhibit stronger immunological resistance[31–33], which would be expected to have the opposite effect on parasite diversity, and a previous study found that some parasite groups are decreased in urban environments rather than increased[11]. Unfortunately, the field is generally lacking in large-scale cross-species analyses of immune function that would be required to differentiate these possibilities[14] (but see also refs. [34–36]). Ideally, future analyses incorporating life history, habitat preference, immunity and parasite diversity may be better able to differentiate the mechanisms underlying these species' zoonotic risk[14].

Achieving broad insights into the urban drivers of zoonotic risk may require finer-scale data than we had access to here. This study was conducted with a minimum compatibility filter: we considered a species as a host of a given parasite if it was observed with said parasite at any point in the literature, and richness was calculated as the sum of these associations across parasite subgroups. While studies of parasite diversity are common in macroecology, this deliberately narrow scope limits inference about a range of relevant processes including host competence (that is, species' ability to transmit parasites)[36], prevalence of the parasite in the host populations, host density and, therefore, the rate of spillover (that is, the number of animal-to-human transmission events per unit of time). These are all important components of a species' zoonotic risk, and some hosts undoubtedly present substantial zoonotic risk despite having relatively low known parasite diversity. For example, prairie dogs (*Cynomys ludovicianus*) only have five known parasites in our dataset, yet they are a widespread and abundant species and may play an important role in epizootic outbreaks of plague (*Yersinia pestis*) in North America[37]. Given this disparity, it remains unclear how closely a species' zoonotic diversity should correlate with the rate of spillover from these species; as such, we caution that our analysis does not necessarily offer insights into the relative frequency or rate of spillover events, or the potential severity of zoonotic outbreaks, in urban environments.

Providing a general answer to the question 'does urbanization increase the risk of zoonotic disease' may require datasets of individual-level or population-level infection status, using multiple hosts and parasites, distributed across a wide range of urbanization gradients. Higher-resolution datasets such as these would facilitate untangling of within-species and between-species confounders, as well as account for spatiotemporal covariates such as urban habitat composition[38]. These data are increasingly publicly available and are being used in large-scale analyses of disease dynamics (for example, refs. [8,39]); as such, these analyses may become increasingly possible in coming years. Regardless, in these and other analyses, correlated changes in the magnitude and shape of sampling biases (for example, towards zoonotic versus non-zoonotic parasites) should be taken into account when examining links among anthropogenic change, wildlife disease and zoonotic risk.

Methods

Data sources. *Phylogeographic data.* We used the PanTHERIA dataset[40] as a backbone for mammal taxonomy and phenotypic traits such as body mass. Phylogenetic data were derived from a mammalian supertree[41], as used for several host–virus ecology studies (for example, refs. [13,20,42]). The tree's phylogenetic distances between species were scaled between zero and one. Geographic data were taken from the International Union for Conservation of Nature (IUCN) species ranges[43]. For each species, we calculated total range area by adding together the areas for the 25 km raster cells in which they were present.

To derive a measure of study effort, which often explains substantial variation in parasite diversity[20,21], we conducted systematic PubMed searches to identify how many publications mentioned a given mammal species, following previous methodology[44]. Domestication status used a sensu lato definition based on whether a species has ever been partially domesticated, coded as a binary variable. For example, despite being widespread in the wild, the European red deer (*Cervus elaphus*) is coded as 'domestic' because it is often farmed, notably in New Zealand[45]. Because we were investigating spatial distributions of species (see above), fully domesticated species that do not exist in the wild (for example, cattle, *Bos taurus*) were generally excluded due to their absence from the IUCN species ranges. To investigate whether dietary flexibility could affect parasite diversity, following previous methodology[18], we derived diet diversity by calculating a Shannon index from the EltonTraits database proportional diet contents[46].

Life history data. To investigate how host life history variation affects parasite richness, we used a previously published, mass-corrected PC analysis of life history variation across mammal species[47]. The first two PCs from this analysis, which explained 86% of variation in six life history traits[47], were used as explanatory variables in our models. The six life history traits were gestation length, litter size, neonate body mass, interbirth interval, weaning age and sexual maturity age. PC1 explains 63% of the variance in the six traits, representing a generalizable slow–fast life history axis. PC2 explains 23% of variance in these traits and represents greater investment in gestation time and larger offspring. Both PCs were available for all mammals in our dataset. We coded the PCs such that increasing values corresponded to 'faster' life history (that is, favouring greater reproduction over survival).

Urban adaptation data. We identified each species' habitat preferences using a published database of long-term urban adaptation status in mammals[18]. This dataset was compiled using literature searches to identify species that were observed inhabiting urban environments; species are either coded as a 'visitor' or a 'dweller', on the basis of whether they rely fully on urban environments to survive and reproduce (dweller), or whether they continue to rely on non-urban resources (visitor). This approach distinguishes our analysis from previous studies

(for example, ref. [5]): we use a strict definition of 'urban-adapted' species, defining them as 'mammals that survive, reproduce, and thrive in urban environments', rather than basing urban status purely on survey records collected in urban settings. All species that were in PanTHERIA but were not in the urban adaptation dataset were coded as 'non-urban'. We used urban adaptation as a binary variable, coding species as zero or one, depending on whether it was in the urban adaptation dataset. Overall, 180 species in our dataset were coded as a one, denoting that they had been observed living off urban resources.

Host–parasite association data. The recently released CLOVER dataset[48] is the most comprehensive open-source dataset on the mammal–virus network. Here we use an expanded version of this dataset that encompasses all parasites, rather than restricting to viruses, to complete an analytical study of these taxonomically broad parasite data. This dataset was synthesized from four large-scale datasets of host–parasite associations, each collected through a combination of web scrapes and systematic literature searches[20,49–51]. These include the Enhanced Infectious Diseases Database (EID2[50]); the Host–Pathogen Phylogeny Project (HP3[20]); the Global Mammal Parasite Database (GMPD[49]); and a large-scale database on viruses and bacteria and their known hosts[51]. These contain a range of parasite groups, including viruses, bacteria, protozoa, fungi, helminths and arthropods. In this conjoined dataset, host–parasite associations were counted according to demonstrated compatibility: that is, if a host species had ever been discovered infected with a given parasite, it was coded as a one, and all undemonstrated associations were assumed absent. In addition to the taxonomic reconciliation underlying the CLOVER dataset, we cleaned the parasite names with the R package taxize[52], removing parasites that were not identified to species level and ensuring that no parasites existed under multiple identities. This ensured that no host–parasite associations were counted twice. We also combined the database with the VIRION dataset, a database of viruses that includes GenBank records[53], resulting in a total 18,967 unique host–parasite associations.

From this conjoined dataset, we derived the following traits for each mammal host species in our dataset: (1) total parasite richness: the number of unique parasite species known to infect a given host species; and (2) zoonotic parasite richness: the number of these parasites that has also been observed to infect humans in our dataset. All analyses were repeated for overall parasite numbers (for example, total number of zoonoses across all parasite groups) and for specific parasite subgroups (viruses, bacteria, protozoa, fungi, helminths and arthropods).

For each analysis, to facilitate model fitting, we eliminated species for which there were missing data and then removed all host orders for which there were fewer than 20 species or for which less than 1% of species had one or more known parasites. Leaving these taxa in did not notably alter fixed effects estimates generally but generated unlikely estimates for order-level effects. When combining the phenotypic, urban adaptation and parasite datasets, any species with no known parasite associations was coded as a zero (that is, a pseudoabsence), under the assumption that species with no known parasites are still informative of variables associated with low parasite richness[14].

Models. *Base model.* To analyse associations between urban adaptation status and parasite richness, we used GLMMs inferred using Integrated Nested Laplace Approximation (INLA)[54,55]. We used two response variables with a negative binomial distribution: total parasite richness and zoonotic parasite richness, where the second value was a subset of the first. Explanatory variables included: citation number ($\log(x+1)$-transformed); host order (7 levels: Artiodactyla, Carnivora, Chiroptera, Lagomorpha, Primates, Rodentia, Soricomorpha); urban adaptation status (binary, non-urban/urban); range area (continuous, log-transformed, defined above); phylogenetic distance from humans (continuous, scaled 0–1); body mass (continuous, log-transformed); domestication status (binary) and two life history PCs (PC1 and PC2; continuous, taken from ref. [47]). We also applied these models to each parasite subset to assess the generality of our parameter estimates. To examine how much of the observed urban effects were attributable to research effort, we first fitted a model without citation number as an explanatory variable and then added it to identify a change in the urban effect. Substantial reduction in the urban effect, accompanied by a strong effect of citation number, would imply that much of the urban effect occurs because urban-adapted species are better studied.

Urban:citation generalized mixed models. Because urban status and citation number were highly correlated and showed very different distributions, we fitted a generalized additive model (GAM) that was otherwise identical to our GLMMs, but with a smoothed term for citations that included an interaction with urban status.

Urban–order interaction model. We then compared the base model with one including an interaction between host order and urban adaptation status to investigate whether the effect of urban adaptation varied taxonomically. We used the DIC to measure model fit, with a $\Delta\text{DIC} < 5$ denoting competitive models.

Phylogenetic model. For each model, we fitted a phylogenetic similarity effect in place of the host order effect to estimate how phylogenetic relatedness between species contributed to similarity in parasite richness. We used DIC to identify whether this effect improved model fit in the same way as the interaction model.

Multivariate models. To investigate whether urban adaptation status had different effects for the richness of different parasite types, we fitted two multi-response models using the MCMCglmm package[56]: one for overall richness and one for zoonotic richness. These models used each of the six parasite groups as response variables and included the same fixed effects, with different (but correlated) slopes for each response. Comparing the model's estimates for the effect of urban adaptation for each parasite allowed us to ask whether specific parasite groups are significantly more likely to be associated with urban adaptation status than others.

Zero-inflated models. To investigate whether pseudoabsences were disproportionately altering our results, we ran zero-inflated models of parasite and zoonotic richness, again using MCMCglmm to control for processes that specifically generate zero counts. These models generate two estimates for each explanatory variable: (1) the effect on the probability that a species' parasite count is greater than zero ('zero inflation') and (2) the effect on parasite count greater than zero when accounting for this effect ('Poisson'). We used uninformative priors. Importantly, the Poisson component of this model generates some zeros itself, which improves on similar models (for example, hurdle models) in which all zeros must be produced by the inflation term. This model allows us to identify whether, for example, urban species are simply more likely to have one or more known parasites, rather than having a greater overall known parasite richness, and whether our choice to code mammals with no known parasites as zero counts would influence the results.

Historical rates of parasite discovery. To investigate how differences between urban and non-urban wild mammals have accumulated over time, we analysed historical rates of parasite discovery and citation effort (from PubMed) between 1930 and 2020, following the methodology described in ref. [26]. Briefly, each unique host–parasite association was assigned a 'discovery date' (the year of the earliest reported association in our dataset, on the basis of either publication year, accession year or sampling year depending on the original data source; see ref. [26] for details). We accessed yearly counts of citations from the PubMed database per host species using the 'rentrez' package[57]. We visualized annual trends in novel parasite discovery and novel host–parasite association discovery in both urban and non-urban mammal species. We then fitted GAMs with a nonlinear effect of year (specified as a penalized thin-plate regression spline) to estimate the annual species-level mean publications, cumulative publications, parasites discovered and cumulative parasite richness, fitting separate models for urban-adapted ($n = 146$) and non-urban ($n = 1{,}365$) species in our host–parasite dataset. We visualized fitted trends in these metrics to examine how differences in yearly and cumulative publication effort and parasite discovery rates have varied between urban and non-urban species (Supplementary Fig. 7).

Path analysis. To investigate whether urban mammals had a disproportionately high zoonotic richness when accounting for overall parasite richness, we fitted a path analysis[58] with zoonotic richness as the ultimate response variable, log(overall richness + 1) as an explanatory variable, and every other explanatory variable described above. We took 1,000 random draws from the posterior distributions of: (1) the effect of urban affiliate status on overall parasite diversity, (2) the effect of urban affiliate status on zoonotic richness and (3) the effect of overall richness on zoonotic richness. This approach allowed us to identify whether urban adaptation had a significant positive effect on zoonotic richness when accounting for its effect on parasite richness as a whole, informing us as to whether a disproportionate number of urban mammals' known parasites are known zoonoses.

Spatial model. Observed parasite diversity in mammals is highly spatially heterogeneous at a global level[20,25,59], while the diversity of known urban-adapted species is heavily biased towards North America and Eurasia[18]. Both are driven by a combination of geographic variation in sampling effort as well as biotic and abiotic factors. To control for these spatial heterogeneities, we fitted spatial explanatory variables using three approaches using a SPDE effect in INLA[54,55]. This effect used species' geographic centroids in their IUCN ranges to control for spatial autocorrelation in the response variable according to Matern correlation, where species that were closer in space would be predicted to have similar numbers of known parasites as a result of sampling bias and biological factors. We first fitted one spatial field to the whole dataset to look for overall spatial structuring, and we then allowed this spatial effect to vary for urban and non-urban species to investigate whether the distribution of known richness varies between these hosts. Second, we incorporated species' presence on each of five continents (Eurasia, Africa, North America, South America and Oceania) as binary variables. Third, we added absolute latitude (that is, distance from the Equator). For the latter two approaches, we also fitted an interaction with urban adaptation to investigate whether the effect of urban adaptation status varied across space.

Reporting Summary. Further information on research design is available in the Nature Research Reporting Summary linked to this article.

Data availability

The CLOVER dataset is available at https://github.com/viralemergence/clover. The VIRION dataset is available at https://github.com/viralemergence/virion. All other ancillary data are available at https://github.com/viralemergence/UrbanOutputters.

Code availability

The code used here is available at https://github.com/viralemergence/UrbanOutputters.

Received: 10 March 2021; Accepted: 3 March 2022;
Published online: 2 May 2022

References

1. Morse, S. S. et al. Prediction and prevention of the next pandemic zoonosis. *Lancet* **380**, 1956–1965 (2012).
2. Jones, K. E. et al. Global trends in emerging infectious diseases. *Nature* **451**, 990–993 (2008).
3. Keesing, F. et al. Impacts of biodiversity on the emergence and transmission of infectious diseases. *Nature* **468**, 647–652 (2010).
4. Carlson, C. J. et al. Climate change will drive novel cross-species viral transmission. Preprint at *bioRxiv* https://doi.org/10.1101/2020.01.24.918755 (2020).
5. Gibb, R. et al. Zoonotic host diversity increases in human-dominated ecosystems. *Nature* https://doi.org/10.1038/s41586-020-2562-8 (2020).
6. Loh, E. H. et al. Targeting transmission pathways for emerging zoonotic disease surveillance and control. *Vector Borne Zoonotic Dis.* **15**, 432–437 (2015).
7. Hassell, J. M., Begon, M., Ward, M. J. & Fèvre, E. M. Urbanization and disease emergence: dynamics at the wildlife–livestock–human interface. *Trends Ecol. Evol.* **32**, 55–67 (2017).
8. Cohen, J. M., Sauer, E. L., Santiago, O., Spencer, S. & Rohr, J. R. Divergent impacts of warming weather on wildlife disease risk across climates. *Science* **370**, eabb1702 (2020).
9. Murray, M. H. et al. City sicker? A meta-analysis of wildlife health and urbanization. *Front. Ecol. Environ.* **17**, 575–583 (2019).
10. Becker, D. J., Hall, R. J., Forbes, K. M., Plowright, R. K. & Altizer, S. Anthropogenic resource subsidies and host–parasite dynamics in wildlife. *Phil. Trans. R. Soc. B* **373**, 20170086 (2018).
11. Werner, C. S. & Nunn, C. L. Effect of urban habitat use on parasitism in mammals: a meta-analysis. *Proc. Biol. Sci.* **287**, 20200397 (2020).
12. Becker, D. J., Streicker, D. G. & Altizer, S. Linking anthropogenic resources to wildlife–pathogen dynamics: a review and meta-analysis. *Ecol. Lett.* **18**, 483–495 (2015).
13. Becker, D. J. et al. Macroimmunology: the drivers and consequences of spatial patterns in wildlife immune defense. *J. Anim. Ecol.* **89**, 972–995 (2020).
14. Albery, G. F. & Becker, D. J. Fast-lived hosts and zoonotic risk. *Trends Parasitol.* https://doi.org/10.1016/j.pt.2020.10.012 (2021).
15. Seto, K. C., Güneralp, B. & Hutyra, L. R. Global forecasts of urban expansion to 2030 and direct impacts on biodiversity and carbon pools. *Proc. Natl Acad. Sci. USA* **109**, 16083–16088 (2012).
16. Chen, G. et al. Global projections of future urban land expansion under shared socioeconomic pathways. *Nat. Commun.* **11**, 537 (2020).
17. Gao, J. & O'Neill, B. C. Mapping global urban land for the twenty-first century with data-driven simulations and shared socioeconomic pathways. *Nat. Commun.* **11**, 2302 (2020).
18. Santini, L. et al. One strategy does not fit all: determinants of urban adaptation in mammals. *Ecol. Lett.* **22**, 365–376 (2019).
19. Ostfeld, R. S. et al. Life history and demographic drivers of reservoir competence for three tick-borne zoonotic pathogens. *PLoS ONE* **9**, e107387 (2014).
20. Olival, K. J. et al. Host and viral traits predict zoonotic spillover from mammals. *Nature* **546**, 646–650 (2017).
21. Mollentze, N. & Streicker, D. G. Viral zoonotic risk is homogenous among taxonomic orders of mammalian and avian reservoir hosts. *Proc. Natl Acad. Sci. USA* **117**, 9423–9430 (2020).
22. Gutiérrez, J. S., Piersma, T. & Thieltges, D. W. Micro- and macroparasite species richness in birds: the role of host life history and ecology. *J. Anim. Ecol.* **88**, 1226–1239 (2019).
23. Teitelbaum, C. S. et al. A comparison of diversity estimators applied to a database of host–parasite associations. *Ecography* **43**, 1316–1328 (2019).
24. Jorge, F. & Poulin, R. Poor geographical match between the distributions of host diversity and parasite discovery effort. *Proc. R. Soc. B* **285**, 20180072 (2018).
25. Allen, T. et al. Global hotspots and correlates of emerging zoonotic diseases. *Nat. Commun.* **8**, 1124 (2017).
26. Gibb, R. et al. Mammal virus diversity estimates are unstable due to accelerating discovery effort. *Biol. Lett.* https://doi.org/10.1098/rsbl.2021.0427 (2022).
27. Hughes, A. et al. Sampling biases shape our view of the natural world. *Ecography* **44**, 1259–1269 (2021).
28. Estes, L. et al. The spatial and temporal domains of modern ecology. *Nat. Ecol. Evol.* **2**, 819–826 (2018).
29. Titley, M. A., Snaddon, J. L. & Turner, E. C. Scientific research on animal biodiversity is systematically biased towards vertebrates and temperate regions. *PLoS ONE* **12**, e0189577 (2017).
30. Lloyd-Smith, J. O. et al. Should we expect population thresholds for wildlife disease? *Trends Ecol. Evol.* **20**, 511–519 (2005).
31. Cummings, C. R. et al. Foraging in urban environments increases bactericidal capacity in plasma and decreases corticosterone concentrations in white ibises. *Front. Ecol. Evol.* **8**, 575980 (2020).
32. Hwang, J. et al. Anthropogenic food provisioning and immune phenotype: association among supplemental food, body condition, and immunological parameters in urban environments. *Ecol. Evol.* **8**, 3037–3046 (2018).
33. Strandin, T., Babayan, S. A. & Forbes, K. M. Reviewing the effects of food provisioning on wildlife immunity. *Phil. Trans. R. Soc. B* **373**, 20170088 (2018).
34. Downs, C. J., Dochtermann, N. A., Ball, R., Klasing, K. C. & Martin, L. B. The effects of body mass on immune cell concentrations of mammals. *Am. Nat.* **195**, 107–114 (2020).
35. Downs, C. J. et al. Extreme hyperallometry of mammalian antibacterial defenses. Preprint at *bioRxiv* https://doi.org/10.1101/2020.09.04.242107 (2020).
36. Becker, D. J., Seifert, S. N. & Carlson, C. J. Beyond infection: integrating competence into reservoir host prediction. *Trends Ecol. Evol.* **35**, 1062–1065 (2020).
37. Hanson, D. A., Britten, H. B., Restani, M. & Washburn, L. R. High prevalence of *Yersinia pestis* in black-tailed prairie dog colonies during an apparent enzootic phase of sylvatic plague. *Conserv. Genet.* **8**, 789–795 (2007).
38. Gecchele, L. V., Pedersen, A. B. & Bell, M. Fine-scale variation within urban landscapes affects marking patterns and gastrointestinal parasite diversity in red foxes. *Ecol. Evol.* **10**, 13796–13809 (2020).
39. Albery, G. F., Sweeny, A. R., Becker, D. J. & Bansal, S. Fine-scale spatial patterns of wildlife disease are common and understudied. *Funct. Ecol.* https://doi.org/10.1111/1365-2435.13942 (2021).
40. Jones, K. E. et al. PanTHERIA: a species-level database of life history, ecology, and geography of extant and recently extinct mammals. *Ecology* **90**, 2648–2648 (2009).
41. Fritz, S. A., Bininda-Emonds, O. R. P. & Purvis, A. Geographical variation in predictors of mammalian extinction risk: big is bad, but only in the tropics. *Ecol. Lett.* **12**, 538–549 (2009).
42. Albery, G. F., Eskew, E. A., Ross, N. & Olival, K. J. Predicting the global mammalian viral sharing network using phylogeography. *Nat. Commun.* https://doi.org/10.1038/s41467-020-16153-4 (2020).
43. *IUCN Red List of Threatened Species Version 2019-2* (IUCN, 2019); https://www.iucnredlist.org
44. Becker, D. J. et al. Optimising predictive models to prioritise viral discovery in zoonotic reservoirs. *Lancet Microbe* https://doi.org/10.1016/S2666-5247(21)00245-7 (2022).
45. Mason, P. Parasites of deer in New Zealand. *N. Zeal. J. Zool.* **21**, 39–47 (1994).
46. Wilman, H. et al. EltonTraits 1.0: species-level foraging attributes of the world's birds and mammals. *Ecology* **95**, 2027 (2014).
47. Plourde, B. T. et al. Are disease reservoirs special? Taxonomic and life history characteristics. *PLoS ONE* **12**, e0180716 (2017).
48. Gibb, R. et al. Data proliferation, reconciliation, and synthesis in viral ecology. *Bioscience* https://doi.org/10.1101/2021.01.14.426572 (2021).
49. Stephens, P. R. et al. Global mammal parasite database version 2.0. *Ecology* **98**, 1476 (2017).
50. Wardeh, M., Risley, C., Mcintyre, M. K., Setzkorn, C. & Baylis, M. Database of host–pathogen and related species interactions, and their global distribution. *Sci. Data* **2**, 150049 (2015).
51. Shaw, L. P. et al. The phylogenetic range of bacterial and viral pathogens of vertebrates. *Mol. Ecol.* **29**, 3361–3379 (2020).
52. Chamberlain, S. A. & Szöcs, E. taxize: taxonomic search and retrieval in R. *F1000Res* https://doi.org/10.12688/f1000research.2-191.v2 (2013).
53. Carlson, C. J. et al. The Global Virome in One Network (VIRION): an atlas of vertebrate–virus associations. *mBio* **13**, e0298521 (2022).
54. Lindgren, F. & Rue, H. Bayesian spatial modelling with R-INLA. *J. Stat. Softw.* **63**, 1–25 (2015).
55. Lindgren, F., Rue, H. & Lindstrom, J. An explicit link between Gaussian fields and Gaussian Markov random fields: the stochastic partial differential equation approach. *J. R. Stat. Soc. B* **73**, 423–498 (2011).
56. Hadfield, J. D. MCMC methods for multi-response generalized linear mixed models: the MCMCglmm R package. *J. Stat. Softw.* **33**, 1–22 (2010).
57. Winter, D. J. rentrez: an R package for the NCBI eUtils API. *R J.* **9**, 520–526 (2017).
58. Shipley, B. Confirmatory path analysis in a generalized multilevel context. *Ecology* **90**, 363–368 (2009).
59. Carlson, C. J., Dallas, T. A., Alexander, L. W., Phelan, A. L. & Phillips, A. J. What would it take to describe the global diversity of parasites? *Proc. R. Soc. B* **287**, 20201841 (2020).

Acknowledgements
This work was supported by funding to the Viral Emergence Research Initiative (VERENA) consortium, including National Science Foundation grant BII 2021909.

Author contributions
G.F.A. and D.J.B. conceived the study, and G.F.A. analysed the data and wrote the manuscript. C.J.C., L.E.C., E.A.E., R.G., S.J.R., A.R.S. and D.J.B. offered thoughts on the analysis and commented on the manuscript.

Competing interests
The authors declare no competing interests.

Additional information
Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s41559-022-01723-0.

Correspondence and requests for materials should be addressed to Gregory F. Albery.

Peer review information *Nature Ecology & Evolution* thanks Luis Escobar, James Hassell and the other, anonymous, reviewer(s) for their contribution to the peer review of this work.

Reprints and permissions information is available at www.nature.com/reprints.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

nature portfolio

Corresponding author(s): Gregory Albery

Last updated by author(s): 01/03/2022

Reporting Summary

Nature Portfolio wishes to improve the reproducibility of the work that we publish. This form provides structure for consistency and transparency in reporting. For further information on Nature Portfolio policies, see our Editorial Policies and the Editorial Policy Checklist.

Statistics

For all statistical analyses, confirm that the following items are present in the figure legend, table legend, main text, or Methods section.

n/a	Confirmed	
☐	☒	The exact sample size (*n*) for each experimental group/condition, given as a discrete number and unit of measurement
☐	☒	A statement on whether measurements were taken from distinct samples or whether the same sample was measured repeatedly
☐	☒	The statistical test(s) used AND whether they are one- or two-sided *Only common tests should be described solely by name; describe more complex techniques in the Methods section.*
☐	☒	A description of all covariates tested
☐	☒	A description of any assumptions or corrections, such as tests of normality and adjustment for multiple comparisons
☐	☒	A full description of the statistical parameters including central tendency (e.g. means) or other basic estimates (e.g. regression coefficient) AND variation (e.g. standard deviation) or associated estimates of uncertainty (e.g. confidence intervals)
☐	☒	For null hypothesis testing, the test statistic (e.g. *F*, *t*, *r*) with confidence intervals, effect sizes, degrees of freedom and *P* value noted *Give P values as exact values whenever suitable.*
☐	☒	For Bayesian analysis, information on the choice of priors and Markov chain Monte Carlo settings
☐	☒	For hierarchical and complex designs, identification of the appropriate level for tests and full reporting of outcomes
☐	☒	Estimates of effect sizes (e.g. Cohen's *d*, Pearson's *r*), indicating how they were calculated

Our web collection on statistics for biologists contains articles on many of the points above.

Software and code

Policy information about availability of computer code

Data collection	NA
Data analysis	The code used here is available at github.com/viralemergence/UrbanOutputters.

For manuscripts utilizing custom algorithms or software that are central to the research but not yet described in published literature, software must be made available to editors and reviewers. We strongly encourage code deposition in a community repository (e.g. GitHub). See the Nature Portfolio guidelines for submitting code & software for further information.

Data

Policy information about availability of data

All manuscripts must include a data availability statement. This statement should provide the following information, where applicable:

- Accession codes, unique identifiers, or web links for publicly available datasets
- A description of any restrictions on data availability
- For clinical datasets or third party data, please ensure that the statement adheres to our policy

The CLOVER dataset is available at github.com/viralemergence/clover. The VIRION dataset is available at github.com/viralemergence/virion. All other ancillary data are available in github.com/viralemergence/UrbanOutputters.

Field-specific reporting

Please select the one below that is the best fit for your research. If you are not sure, read the appropriate sections before making your selection.

☐ Life sciences ☐ Behavioural & social sciences ☒ Ecological, evolutionary & environmental sciences

For a reference copy of the document with all sections, see nature.com/documents/nr-reporting-summary-flat.pdf

Ecological, evolutionary & environmental sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description	The study examines species-level pathogen diversity in mammals and examines whether urban-adapted mammal species have more known pathogens, and whether a disproportionate number of these pathogens have been observed to infect humans. We also investigate the underlying geographic drivers and potential sampling biases.
Research sample	The research uses a series of publicly available databases. This includes the aggregated Clover database of host-pathogen associations (Gibb et al. 2021), which is the most comprehensive available dataset of mammal-virus associations, alongside the Global Mammal Parasite Database (GMPD Stephens et al 2017), Olival et al (2017), the Enhanced Infectious Diseases Database (EID2; Wardeh et al 2015); and Shaw et al (2020). Urbanisation status was derived from a recently published dataset of urban-adapted mammal species (Santini et al. 2019). Other phenotypic traits were taken from PanTheria (Jones et al 2009), phenotypic data from a supertree by Fritz et al (2009), and geographic data from the IUCN species ranges.
Sampling strategy	These datasets were chosen because they are the most comprehensive available datasets for species-level data of this nature.
Data collection	NA
Timing and spatial scale	The data are globally distributed, and were collected over the course of the last century of pathogen sampling and data sharing.
Data exclusions	Data were only excluded from the analysis if they were missing data for one or more of the variables; the 3004 species we list were those for which all columns were available (from a total ~5000 mammal species).
Reproducibility	NA
Randomization	NA
Blinding	NA

Did the study involve field work? ☐ Yes ☒ No

Reporting for specific materials, systems and methods

We require information from authors about some types of materials, experimental systems and methods used in many studies. Here, indicate whether each material, system or method listed is relevant to your study. If you are not sure if a list item applies to your research, read the appropriate section before selecting a response.

Materials & experimental systems

n/a	Involved in the study
☒	☐ Antibodies
☒	☐ Eukaryotic cell lines
☒	☐ Palaeontology and archaeology
☒	☐ Animals and other organisms
☒	☐ Human research participants
☒	☐ Clinical data
☒	☐ Dual use research of concern

Methods

n/a	Involved in the study
☒	☐ ChIP-seq
☒	☐ Flow cytometry
☒	☐ MRI-based neuroimaging