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# A comparison of bats and rodents as reservoirs of zoonotic viruses: are bats special?

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Bats are the natural reservoirs of a number of high-impact viral zoonoses. We present a quantitative analysis to address the hypothesis that bats are unique in their propensity to host zoonotic viruses based on a comparison with rodents, another important host order. We found that bats indeed host more zoonotic viruses per species than rodents, and we identified life-history and ecological factors that promote zoonotic viral richness. More zoonotic viruses are hosted by species whose distributions overlap with a greater number of other species in the same taxonomic order (sympatry). Specifically in bats, there was evidence for increased zoonotic viral richness in species with smaller litters (one young), greater longevity and more litters per year. Furthermore, our results point to a new hypothesis to explain in part why bats host more zoonotic viruses per species: the stronger effect of sympatry in bats and more viruses shared between bat species suggests that interspecific transmission is more prevalent among bats than among rodents. Although bats host more zoonotic viruses per species, the total number of zoonotic viruses identified in bats (61) was lower than in rodents (68), a result of there being approximately twice the number of rodent species as bat species. Therefore, rodents should still be a serious concern as reservoirs of emerging viruses. These findings shed light on disease emergence and perpetuation mechanisms and may help lead to a predictive framework for identifying future emerging infectious virus reservoirs.

## 1. Introduction

Emerging infectious diseases threaten global biodiversity and public health [1–3]. Most emerging and re-emerging infectious diseases of humans are zoonoses. Most zoonoses originate in wildlife and are increasing over time [3–5]; however, the relative importance of different groups of wildlife hosts in the emergence of zoonoses remains unclear, as do the mechanisms driving such differences.

Bats (Order Chiroptera) provide considerable ecosystem services, such as arthropod suppression, seed dispersal and pollination, across a vast range of regions and habitats. However, bats are receiving increasing attention as potential reservoirs for zoonotic diseases following recent identification of their involvement with severe acute respiratory syndrome-like coronaviruses, Ebola and Marburg filoviruses, as well as Hendra and Nipah paramyxoviruses [6]. Consequently, there has been repeated speculation that bats may be unique in their potential to harbour zoonotic viruses [6–8]. Traits that may make bats suited to hosting more viruses in general (zoonotic and non-zoonotic) include relatively long lifespans for their body size [9], which may facilitate viral persistence for chronic infections; the reliance of some on prolonged torpor, which can decrease both viral replication and immune function [10,11]; and flight, allowing movement and dispersal over long distances in some species. Additionally, many bat species are gregarious, some living in dense aggregations: for example, some Mexican free-tailed bat (*Tadarida brasiliensis mexicana*) colonies can reach densities of 3000 bats per square metre, in populations of up to a million individuals per roost [12,13]. Roosting sites can house diverse assemblages of multiple bat species [14,15]. High intra- and interspecific contact rates can facilitate rapid transmission of pathogens and large population sizes could sustain acute-immunizing infections. Additionally, there are some traits that may make bats more likely to host zoonotic viruses in particular and/or transmit them to humans. In evolutionary terms, bats are ancient mammals and it has been hypothesized that viruses which evolved in bats may use highly conserved cellular receptors, thus enhancing their ability to transmit viruses to other mammals [6]. Many species of bats have peridomestic habits, roosting in houses and other buildings, as well as trees in dense urban areas, leading to frequent human contact with bat excreta [16–18]. Bat–human contact is also increasing in recent decades owing to habitat encroachment and increased use of bats as bushmeat [17–21]. However, despite the speculation that bats are unusual in their potential to host zoonotic viruses, there are no quantitative comparative analyses to support this hypothesis.

Identifying reservoir species is key to controlling emerging infectious diseases, but there is currently no framework for characterizing the likely role a potential host species may play. Therefore, a general approach is needed for understanding how host–pathogen communities are broadly structured. A growing area of research in ecology relies on trait-based approaches to predict community assembly [22]. These approaches concentrate on traits of species in an attempt to find generalities in species interactions with each other and with the environment. Characterizing which traits are associated with pathogens and their reservoir hosts will contribute to understanding basic disease emergence and perpetuation mechanisms and may help to focus future research and disease mitigation efforts.

In this study, we make a first attempt to quantitatively address the hypothesis that bats are unique hosts of zoonotic viruses, and further, apply a trait-based approach to identify life-history, physiological and ecological traits that correlate with a species' propensity to host zoonotic viruses. Rodents (Order Rodentia) are a suitable comparison group as they are important reservoir hosts of a number of zoonotic viral pathogens with significant impacts on public health [23]. These pathogens include hantaviruses (causing hantavirus pulmonary syndrome and haemorrhagic fever with renal syndrome; [24]) and arenaviruses (causing, e.g. lymphocytic choriomeningitis, Lassa fever and Argentine, Bolivian, Venezuelan and Brazilian haemorrhagic fevers [25]). Rodents also share a number of characteristics with bats that have been hypothesized to affect reservoir potential; both taxonomic orders are evolutionarily ancient, diverse and include many species with peridomestic habits and species that commonly express torpor. Rodents are more diverse than bats in numbers of species and life-history strategies (the reproduction-longevity trade-off), which enables a more general examination of host correlates for viral richness across taxonomic orders. We also investigate a series of factors that may be important in pathogen sharing, such as host relatedness, geographical overlap and conservation status (which may be important in pathogen sharing, e.g. as in primates [26,27]). Finally, we examine the possibility of increased zoonotic viruses at low latitudes, as Jones *et al*. proposed a link between latitude and risk of zoonotic emergence [3].

In addition to host traits, viral traits affect spillover and emergence of zoonoses: RNA viruses are more likely to emerge than DNA viruses [28], and replication in the cytoplasm was the best predictor of cross-species transmission from livestock to humans [29]. Therefore, we also explore some basic characteristics of viruses found in bats and rodents.

## 2. Material and methods

### (a) Viral data

We compiled databases of viruses in bats or rodents and the species in which each has been detected by searching Thomson Reuters (formerly ISI) Web of Science (http://apps.webofknowledge.com/) for each rodent and bat genus 'AND virus' (under 'Topic') through the year 2011. Viruses were grouped at the species level, based on the International Committee on the Taxonomy of Viruses database. Host taxonomy conforms to Wilson & Reader's *Mammal Species of the World* [30]. These databases are included in the electronic supplementary material. Viruses were classified as zoonotic or non-zoonotic; RNA or DNA; replicating in the cytoplasm or nucleus, and whether they consist of a single segment or multiple segments. Viruses and hosts that were not identified to species were not included in the analyses. More than double the number of viruses are known for *Mus musculus* than for any other rodent species, as a consequence of its use as a laboratory animal and the donor of many cell lines. For example, minute virus of mice was discovered as a contaminant in the experimental stock of a different virus when grown in a mouse cell line [31]. Therefore, this host species was removed from analyses, because preliminary analyses identified it as a high leverage point.

### (b) Species trait data

For as many host species in our viral database as possible, we compiled data for the following traits (see the electronic supplementary material, table S14 and figures S4–S8): adult body mass, maximum longevity, number of litters per year, litter size, torpor use, migration (bats only), International Union for Conservation of Nature (IUCN) conservation status, species geographical distribution area, latitude of the midpoint (centroid) of the species distribution, number of other species in the same taxonomic order that are sympatric, number of citations on Web of Science. Data were obtained from an online database of mammalian traits (http://www.utheria.com) [32] on body mass, maximum longevity, number of litters per year and litter

**Table 1.** GLM rankings, with the number of zoonotic viruses identified in a species as the response variable (not considering host traits).

| model | AICc | d.f. | weight | *p*-value |
|---|---|---|---|---|
| ~ log(citations) + order | 1275.3 | 410 | 0.710 | $< 10^{-16}$ |
| ~ log(citations)*order | 1277.1 | 409 | 0.290 | $<10^{-16}$ |
| ~ log(citations) | 1301.5 | 411 | 0 | $<10^{-16}$ |
| ~ order | 1420.2 | 411 | 0 | 0.092 |
| ~ 1 | 1421.0 | 412 | 0 | |

size. Additional values were compiled from the literature (see the electronic supplementary material, table S14 for values and references) and the AnAge database (http://genomics.senescence.info/species/) [33]. Torpor expression was treated as a categorical variable with three categories: (i) no evidence of torpor use, (ii) some torpor use, but not true hibernation (minimum body temperature $\geq 11^{\circ}$C), and (iii) true hibernation (body temperature $< 11^{\circ}$C) [34].

Species sampling intensity was represented by the logged number of Web of Science citations for the binomial species name (and commonly used synonyms) in quotations. IUCN conservation status, species distributions and longitude and latitude coordinates for the centroids of the distributions were obtained from the IUCN website (http://www.iucnredlist.org/technical-documents/spatial-data) [35]. The IUCN has seven conservation status categories: least concern, near threatened, vulnerable, endangered, critically endangered, extinct in the wild and extinct. The first three categories described all the bats in our analysis, and 'least concern' and 'vulnerable' described all the rodents in our analysis. Using the shape files from the IUCN website and the command 'over' from the R packages 'sp' and 'rgeos' [36,37], for each species in the analysis, we calculated how many other species in the same taxonomic order had species ranges that overlapped with its own, referred to here as sympatry. This included every species of bat or rodent for which IUCN had distribution shape files (1150 species of bat and 2216 of rodent).

We follow Fleming & Eby [38, p. 157] in defining migration in bats as 'a seasonal, usually two-way movement from one place or habitat to another to avoid unfavorable climatic conditions and/or to seek more favorable energetic conditions'. In most cases, these are regular annual movements that vary with predictable seasonal changes in temperature (for temperate zone bats) or rainfall (tropical bats). We did not consider records of one-way movements or dispersal as evidence for migration. We categorized migratory status of bats as: (i) species that can be broadly categorized as sedentary or only local (approx. less than 100 km) migrants, (ii) species that can be generalized as regional migrants (approx. 100–500 km), and (iii) species that can be categorized as long-distance migrants (greater than 500 km). This information was compiled from the literature, with species reported to be present in an area year-round considered evidence for category 1.

## (c) Analysis

We used generalized least squares (GLS) to examine host trait correlates of zoonotic viral richness per host species, while controlling for phylogeny as described below. To examine whether bats host a significantly greater number of zoonotic viruses per host species than rodents, with and without taking into account sampling intensity, we used generalized linear models (GLMs) with negative binomial errors.

Because many life-history traits are correlated, we performed principal components analyses (PCA) on the life-history traits: logged body mass, maximum longevity, number of litters per year and litter size. We performed three PCAs, one for bats ($PC_b$), one for rodents ($PC_r$) and one for the bats and rodents combined ($PC_{rb}$). The variables were rescaled to have unit variance before analysis in R using the 'prcomp' function [36], and these principal components were then used in subsequent analyses.

To determine if the number of zoonotic viruses or total number of viruses hosted by a species is significantly correlated to species traits, we performed GLS models for bats and rodents separately, then on the combined bat and rodent data. Because closely related species share traits, we tested for phylogenetic dependence using a GLS framework to allow for correlation structure in the error term. The 'APE' package [39] in R [36] was used to calculate a phylogenetic correlation matrix in which each entry was a pairwise correlation between each pair of species based on their shared branch lengths of a mammalian phylogenetic supertree [40]. The subsets of the phylogenetic tree that we used are shown in the electronic supplementary material, figures S2 and S3. The error term for the GLS was set to this correlation matrix multiplied by an additional parameter, Pagel's $\lambda$, that was estimated (using 'optim' in R) to determine the strength of phylogenetic dependence [41,42]. A $\lambda$ estimate of one indicates that the error structure of the model was directly proportional to the species shared branch lengths. A $\lambda$ estimate of zero indicates that the error structure of the model was not related to the species shared branch lengths (e.g. phylogeny does not explain any additional variation), and the correlation matrix is not included in the model. Models were ranked by their Akaike information criterion with a correction for finite sample sizes (AICc) values. Correlation coefficients (R) were obtained by using Pearson's product moment correlation comparing the observed number of viruses to model predictions.

Chi-squared ($\chi^2$) tests were used to examine which viral traits, such as type of nucleic acid, genomic segmentation and site of replication were associated with zoonotic infection and taxonomic order.

# 3. Results

Bats host, on average, significantly more zoonotic viruses per species than rodents (tables 1 and 2). The response variable, zoonotic viral richness (i.e. number of zoonotic viruses per host species), was significantly greater for bats than rodents after controlling for the significant effect of sampling effort (i.e. order and number of citations were in the best model by AICc; table 1). However, as there are approximately twice as many species of rodent as species of bat, the overall number of zoonotic viruses was fewer in bats (61) than in rodents (68). Twenty-four viruses were present in both bats and rodents, of which 21 were zoonotic. Viruses (both zoonotic and non-zoonotic) had a broader host range in bats, averaging 4.51 bat host species per virus, whereas rodent viruses averaged 2.76, which was significantly different by *t*-test ($t = 2.17$, $p = 0.031$; table 2).

**Table 2.** Summary of the viruses identified.

| | | total viruses | | zoonotic viruses | |
|---|---|---|---|---|---|
| order | mean no. hosts/virus | no. | mean/host (range) | no. | mean/host (range) |
| bats | 4.51 | 137 | 2.71 (1,15) | 61 | 1.79 (0,12) |
| rodents | 2.76 | 179 | 2.48 (1,20) | 68 | 1.48 (0,11) |

Species trait data were available for 66 species of bat and 81 species of rodent (out of 413 species totally). This subset of bat and rodent species had 46 and 53 zoonotic viruses, respectively, which accounted for more than 75 per cent of the total number of zoonotic viruses identified in both orders.

## (a) Species trait correlates of zoonotic infection in rodents

The first three principal components accounted for 93 per cent of the variance in rodent life-history strategies. $PC1_r$ separated r-selected species (larger litter size, more litters per year, shorter lifespan) from K-selected species (lower reproductive rates and greater mass and longevity; electronic supplementary material, figure S1a *x*-axis and table S1). $PC2_r$ separated reproductive strategies for a given number of offspring per year (litters per year versus litter size; electronic supplementary material, figure S1a *y*-axis and table S1). $PC3_r$ separated species with lower or higher values for all of the life-history traits (bigger, longer lived, higher reproduction; electronic supplementary material, table S1).

Two models tied for the best model by AICc in GLS analyses examining correlates of zoonotic viral richness in rodents (see the electronic supplementary material, table S2). Both models included the logged number of citations and rodent sympatry, with one model containing IUCN conservation status. The number of citations was positively correlated with viral richness. Rodent sympatry was also positively correlated to zoonotic viral richness, i.e. rodent species whose distributions overlapped with a greater number of other rodent species had more zoonotic viruses. Rodents with an IUCN status of 'vulnerable' hosted fewer zoonotic viruses than those listed as 'least concern'. Phylogeny did not explain additional variation: for every model, $\lambda < 1 \times 10^{-11}$. Figure 1*a* shows the ranking of variables by their ΔAICc values on removal or addition (compared with the best model).

## (b) Species trait correlates of zoonotic infection in bats

The first three principal components accounted for 88 per cent of the variance in bat life-history strategies. Negative values of $PC1_b$ indicated species with a greater litter size, and positive values indicated species with higher mass and longevity and more litters per year (see the electronic supplementary material, figure S1b *x*-axis and table S3). $PC2_b$ separated species with more litters per year from those with higher longevity (see the electronic supplementary material, figure S1b *y*-axis and table S3). $PC3_b$ separated species with larger litters and mass from those with higher longevity and litters per year (see the electronic supplementary material, table S3).

The best model examining correlates of viral richness in bats by AICc included (in order of importance by ΔAICc) bat sympatry, the logged number of citations, and $PC1_b$ accounting for 78.7 per cent of the model weight (see the electronic supplementary material, table S4). The positive coefficient of $PC1_b$ in the top ranked model indicated that bats with smaller litter size, larger body mass, greater longevity and more litters per year (see the electronic supplementary material, figure S1b, *x*-axis) are more likely to have more zoonotic viruses. Similar to our findings for rodents, sampling effort and sympatry were positively correlated to zoonotic viral richness. In models that did not include sympatry, phylogeny explained additional variation ($\lambda$ estimates ranged up to 0.29). For all the models that included sympatry, $\lambda < 0.001$, indicating phylogeny did not explain additional variation once sympatry was taken into account. The importance of the different variables ranked by ΔAICc is shown in figure 1*b*.

## (c) Species trait correlates of zoonotic infection across both bats and rodents

$PC1_{rb}$ of the combined data largely separated bats from rodents and accounted for 51.1 per cent of the variance in life-history traits (see the electronic supplementary material, figure S9 and table S5). $PC2_{rb}$, $PC3_{rb}$ and $PC4_{rb}$ largely described body mass, litters per year and longevity, and litter size and longevity, respectively (see the electronic supplementary material, figure S9 and table S5).

All four PCs were used in the GLS models that controlled for phylogeny to account for relatively small differences in life-history traits. The best model for the combined data included, in order of importance, citations, taxonomic order, taxon sympatry, the interaction between order and sympatry and torpor use (table 3 and figure 1*c*). Again, bats hosted more zoonotic viruses per species and the effect of sympatry for bats was 3.9 times higher than for rodents. Torpor use was negatively correlated to zoonotic viral richness (see the electronic supplementary material, table S7), and there was some weight for a negative effect of latitude (table 3). Phylogeny did not explain any additional variation ($\lambda < 0.01$).

## (d) Species trait correlates of total viral infections

The species trait correlates of total viral infections were similar to those for zoonotic infections. (see the electronic supplementary material, tables S8–S10.) The best rodent model included citations, rodent sympatry and torpor. The best bat model and the best model for the combined data were the same as for zoonotic viruses.

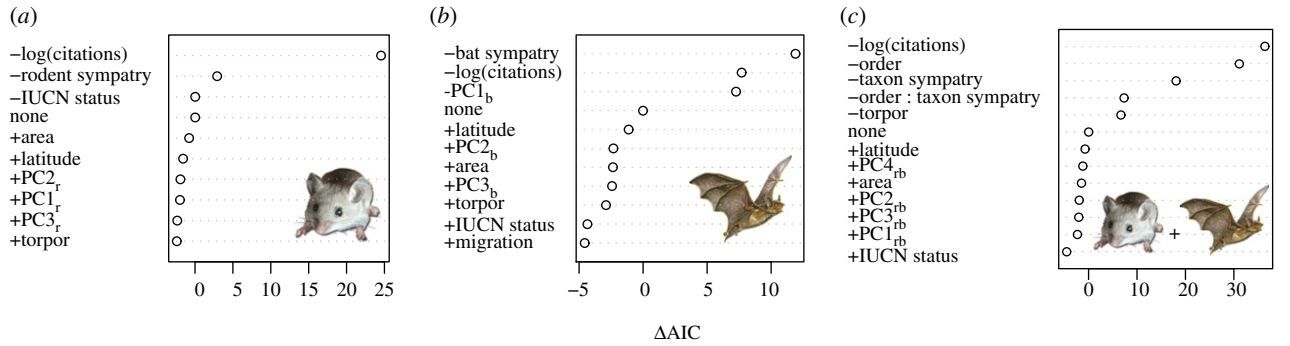


**Figure 1.** Ranking of variables from the GLS models (with phylogenetic correction) by ΔAICc: the change in AICc values when each variable is individually added (+) or removed (−) from the best model for (*a*) rodents (best model: number of zoonotic viruses ~ log(citations) + rodent sympatry + IUCN status), (*b*) bats (best model: number of zoonotic viruses ~ log(citations) + bat sympatry + $PC1_b$), and (*c*) combined rodent and bat data (best model: number of zoonotic viruses ~ log(citations) + order + taxon sympatry + order:taxon sympatry + torpor; where the colon represents the interaction). (Online version in colour.)

**Table 3.** A subset of the GLS rankings for rodents and bats together considering species traits. (The response variable is the number of zoonotic viruses identified in a species. λ shows the strength of the phylogenetic correction. See electronic supplementary material, table S6 for full set of models tested. Asterisks (*) indicate the two variables and their interaction and 'cit'. indicates logged citations.)

| model | AICc | npar | weight | *p*-value | *R* | λ |
|---|---|---|---|---|---|---|
| ~order * taxon sympatry + cit. + torpor | 643.2 | 8 | 0.366 | $8.88 \times 10^{-16}$ | 0.66 | 0 |
| ~order * taxon sympatry + cit. + torpor + latitude | 643.8 | 9 | 0.269 | $1.55 \times 10^{-15}$ | 0.66 | 0 |
| ~order * taxon sympatry + cit. + $PC3_{rb}$ | 649.6 | 7 | 0.014 | $1.34 \times 10^{-14}$ | 0.63 | 0 |
| ~order * taxon sympatry + cit. | 649.9 | 6 | 0.012 | $9.77 \times 10^{-15}$ | 0.62 | 0 |
| ~order + cit. + taxon sympatry + torpor | 650.3 | 7 | 0.010 | $1.80 \times 10^{-14}$ | 0.63 | 0 |
| ~order + cit. + taxon sympatry | 659.3 | 5 | 0 | $5.30 \times 10^{-13}$ | 0.58 | 0 |
| ~order + cit. + taxon sympatry + $PC4_{rb}$ | 661.1 | 6 | 0 | $2.17 \times 10^{-12}$ | 0.58 | 0 |
| ~order + cit. + taxon sympatry + $PC1_{rb}$ | 661.4 | 6 | 0 | $2.58 \times 10^{-12}$ | 0.58 | 0 |
| ~order + cit. + taxon sympatry + IUCN | 663.5 | 7 | 0 | $1.04 \times 10^{-11}$ | 0.58 | 0 |
| ~cit. + taxon sympatry | 672.6 | 4 | 0 | $1.93 \times 10^{-10}$ | 0.52 | 0.001 |
| ~order + cit. | 680.5 | 4 | 0 | $9.80 \times 10^{-9}$ | 0.47 | 0 |
| ~order + taxon sympatry | 686.9 | 4 | 0 | $2.39 \times 10^{-7}$ | 0.44 | 0 |
| the null (intercept) model | 713.2 | 2 | 0 | | | 0.037 |

## (e) Viral traits associated with zoonotic infection

Using $\chi^2$ tests, we compared zoonotic with non-zoonotic viruses. The zoonotic viruses in our database were much more likely to be RNA viruses ($\chi^2 = 42.7$, $p < 0.001$), have multiple segments ($\chi^2$=12.3, $p < 0.001$) and replicate in the cytoplasm ($\chi^2 = 41.8$, $p < 0.001$; electronic supplementary material, table S11), compared with non-zoonotic viruses. From the host perspective, bats harbored a higher proportion of unsegmented genome viruses compared with rodents ($\chi^2 = 6.89$, $p = 0.008$; electronic supplementary material, table S12), but no significant differences were detected in replication site or nucleic acid, and no significant differences were present if comparing only zoonotic viruses of bats and rodents.

# 4. Discussion

It has been suggested that bats may be unique in hosting many emerging zoonotic viruses [6,7]. We found that bats indeed host a significantly greater number of zoonotic viruses per species compared with rodents. Additionally, using a trait-based approach, we identified important life-history and ecological predictors of zoonotic viral richness for both bats and rodents, and identified viral traits that were strongly associated with zoonotic infection.

Sympatry within taxonomic order appeared to be the most important host trait associated with zoonotic viral richness, other than sampling effort as reflected in number of citations. In previous studies, sympatry was also found to be an important predictor of sharing of rabies virus variants among bats [43] and viruses among primates [26]. We show that the effect of sympatry was 3.9 times stronger for bats than for rodents. Although there are fewer range overlaps in bats, perhaps a consequence of there being approximately half the number of bat species as rodent species, there appears to be a greater impact on the number of zoonotic viruses per host when sympatry does occur, suggesting that viruses may be transmitted more easily between sympatric bat species than between sympatric rodent species. One possible contributing factor is the level of interspecific contacts among bats when compared with rodents because many bat roosts have a diverse assemblage of bat species [14,15], whereas rodent species typically do not share communal nesting sites. However, high contact rates alone are

insufficient for cross-species transmission because host and/or virus traits also determine the ability of a virus to infect new host species. For example, the level of physiological similarity across sympatric bat species could affect the ability of viruses adapted to any of the sympatric species to spillover into others (which may be why phylogeny was also an important factor in viral sharing of rabies virus variants [43]). The generality of viral infection traits are probably also important and could allow non-specific viruses to take advantage of multiple host species in close contact. Here, we examined viral richness, but further examination of which viruses are shared among which hosts and their characteristics is warranted and may shed more light on this question.

The importance of $PC1_b$ for bats indicated that bat species with smaller litter size, greater body mass, longevity and more litters per year, tended to host more zoonotic viruses. Rodents have a broad range of reproductive strategies, but in bats, litter size is negatively correlated with the number of litters per year (Pearson's product moment correlation, $p = 0.024$). No bat species consistently has more than three offspring per year, but at lower latitudes (less than 20°), there is some variation as to how these offspring are distributed throughout the year. Our analyses suggest that species which spread births over the year host more zoonotic viruses. A potential physiological explanation is the trade-off between immune function and reproduction. Sex hormones can modulate immunocompetence and affect disease resistance genes and behaviour that may make individuals more susceptible to infection [44]. A potential ecological explanation is the replenishment of the susceptible pool from births. Immunizing, horizontally transmitted infections with a high $R_0$ (basic reproductive number) are vulnerable to 'burn-out' after an epidemic when the number of susceptible hosts drops below the level needed to sustain an epidemic. More litters per year could mean a more continual replenishment of susceptible individuals.

Increased zoonotic viral richness with host longevity is at odds with the 'pace of life' hypothesis, which proposes that short-lived animals put less energy into adaptive immunity in favour of more general immune responses (like broad bacterial recognition), which may make them more competent reservoir hosts [45]. The competency of Lyme disease hosts, for example, seems to follow this pattern [46], but there appears to be mixed evidence for this more generally [47]. This hypothesis also does not consider pathogen traits. Viruses that cause chronic or persistent infections would have higher fitness in a longer-lived host because of the increased infectious period. Therefore, how the pace of a host's life affects reservoir potential may be a function of traits of the virus, host and/or the virus–host interaction.

We hypothesized that torpor use would be positively correlated to viral richness since torpor expression was identified as a key factor in rabies perpetuation in big brown bats (*Eptesicus fuscus*) in Colorado [48], and viral titers can peak upon arousal from hibernation [10]. However, we found torpor was negatively correlated to viral richness. One potential explanation is reduced exposure to viruses owing to lower contact rates during torpor. More research is needed to determine the relationship between torpor, host competence as related to within-host viral persistence and population viral perpetuation processes.

For both bats and rodents, the number of citations was a positive indicator for viruses identified. It is commonly found that the number of pathogens recorded per species is positively correlated with sampling effort, even for well-studied species [26,49,50], indicating that the current estimates of viral richness are probably substantial underestimates. Hence, there may be many more viruses in both bats and rodents with the potential to spillover into humans. Although distribution area was not in the best models, this could be confounded by the finding that area was positively correlated to the number of citations (see the electronic supplementary material, figure S8)—widespread species are more often studied. Widespread species could also have more contact with humans, perhaps facilitating more frequent spillover of pathogens.

We did not see a significant effect of phylogeny in most models ($\lambda$ was near zero). However, $\lambda$ indicates only the amount of residual variation that can be explained by phylogeny after the variables are taken into account. Even though we found little effect of phylogeny in our overall models, when considered individually, all variables examined were correlated to phylogeny to some extent, in at least one of the two groups (see the electronic supplementary material, table S13), suggesting phylogenetic relationships are probably more important than indicated by the models.

Overall, our analyses have explained approximately 43 per cent of the variation seen in zoonotic viral richness among hosts. Although we show that zoonotic viral richness of bats and rodents is significantly different, a majority of the variance in the number of zoonotic viruses per host species is still unexplained, leaving room for multiple alternative explanations. Although sympatry is a good predictor of zoonotic viral richness, our findings suggest high species diversity alone [6] is not the reason for bats hosting a high number of zoonotic viruses. Rodents, the mammalian order with the greatest number of species (twice the number of bat species), were found to host only seven more zoonotic viruses than bats, and rodents host fewer zoonotic viruses per species than bats. It has been hypothesized that because bats are evolutionarily ancient mammals, viruses that evolved with bats may use cellular receptors that have been conserved in mammals, enhancing the ability to transmit to other mammals, including humans [6,51]. However, rodents are evolutionarily older than bats and more closely related to humans [52–54]. If cell receptor evolutionary patterns follow whole genome evolutionary patterns, cell receptors between humans and rodents should be more similar than between humans and bats. While it was beyond the scope of this paper to examine qualitative or quantitative differences in immunity between bats and rodents, such differences may play a role in viral establishment and perpetuation within host populations. We were also unable to address directly the hypothesis that flight helps disperse viruses [6]. However, we found that migration in bats did not predict a higher number of zoonotic viruses. One factor that we were not able to quantify but which is probably important for the ecology and evolution of viruses and other pathogens is the degree of sociality or coloniality of the host. Although many bat species are known to be colonial, a number of species are solitary or nearly so for at least part of the annual cycle. Moreover, the roosting behaviour and social structure of many other species is virtually unknown. Thus, we were not able to quantify coloniality reliably for the species in our analysis at this time, and we recommend future studies incorporate this variable.

The viral traits we found to be associated with zoonotic infection were consistent with those identified by studies

of other taxa. Zoonotic viruses in bats and rodents were overwhelmingly RNA viruses that have multiple segments and replicate in the cytoplasm. These results are similar to those previously published for domestic livestock: for example, the ability to complete replication within the cytoplasm was the single best predictor of whether livestock viruses can infect humans, with a multiple-segmented genome also being a good predictor [29]. Other studies have shown that RNA viruses are more likely to cause emerging infectious diseases than DNA viruses, whether from livestock or from other mammals, such as carnivores [28]. These viral traits shed light on mechanisms of pathogen emergence and spillover. Viruses with RNA and/or multiple-segmented genomes may be more likely to generate genetic diversity with replication, through mutation and reassortment, increasing the chance of zoonotic viral emergence. Additionally, the ability to replicate in the cytoplasm may allow greater chance of spillover to new hosts (including humans) through bypassing the need to interact with the complex cell machinery (which is probably highly host-specific) needed to enter the nucleus for replication.

As this study is based on a large literature search, there are necessarily constraints on inference, given different motivations for, and methods used during studies of both rodent and bat viruses through time. However, our analysis of citations and the interactions between order and citations suggest that the interaction only has 12 per cent weight (see electronic supplementary material, 4th model, table S6), thus suggesting that the effect of sampling effort was not substantially different between the two orders (with the exception of *Mus musculus*, removed from the analyses; see §2). Another potential source of bias may be reports of incidental or spillover hosts that are not important reservoirs but are treated with equal weight in these analyses. Furthermore, although we show that bats have more viruses per species, we cannot say with these analyses which species or orders are likely to be more important in spreading these viruses to humans. Disentangling these factors is difficult at present. Therefore, our results should be viewed with some caution. Recent advances in metagenomic and molecular studies may shed light on some of these issues and alter our understanding of human–rodent and human–bat cross-species transmission [55–57]. Since we did not examine other host groups that are important reservoirs of zoonotic viruses, e.g. primates, ungulates, carnivores and birds, the importance of bats in comparison with other groups remains an open question. We chose rodents as a suitable first comparison because bats and rodents are more similar in life-history traits than other host groups. For example, non-human primates are indeed important reservoir hosts, however, their close phylogenetic relationship with humans, less overlap of life-history traits with bats, and the multiple examples of humans transmitting viruses to primates, such as measles and mumps [58,59] add additional confounding variables. However, further comparative analyses examining a broader range of host groups are warranted.

This study provides evidence that bats are indeed special in at least one regard—they host more zoonotic viruses and more total viruses per species than rodents. However, because there is approximately twice the number of rodent species as bat species, the overall number of zoonotic viruses identified in bats was lower than in rodents. Therefore, rodents should remain a serious concern as reservoirs for future zoonotic disease emergence. This study additionally identifies several specific traits that appear to promote viral richness across taxonomic orders. Given the importance of sympatry in our analysis, future analyses should aim to determine the relative effects of phylogeny and sympatry more broadly in animal reservoirs of emerging zoonoses. Furthermore, our analyses support the theory that traits of zoonotic viruses are also important in determining probability of spillover. Both sympatry and viral traits may act together, with the ability to replicate in the cytoplasm and bypass additional host-specific cell machinery potentially allowing viruses to more easily pass between sympatric species in the same taxonomic order, which could be compounded by increased rates of interspecific contact. Our results, therefore, point to this as a newly hypothesized mechanism to explain, at least in part, how bats host more zoonotic viruses per species. Interspecific transmission may be more prevalent in bats than in rodents (or other orders). This is supported by the most recent molecular studies that indicate there has been a greater number of host switches of paramyxoviruses from bats to other mammals than from rodents, birds, primates, carnivores and cetartiodactyls [55]. Interspecific transmission and spillover is one of the least studied aspects of disease ecology and should therefore be a focus of further studies. Mechanisms of transmission of viruses among bat species may be different than transmission from bats to humans. The mechanisms of interspecific transfer of pathogens, particularly to humans, remain poorly understood, but in some cases are complex and involve intermediate hosts. Gaining understanding of actual mechanisms of such pathogen transfer should be an active area of research in order to develop evidence-based policies to minimize risks, while conserving bats and the irreplaceable ecosystem services they provide.

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