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*Original Contribution*

# Viral Richness is Positively Related to Group Size, but Not Mating System, in Bats

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**Abstract:** Characterizing host traits that influence viral richness and diversification is important for understanding wildlife pathogens affecting conservation and/or human health. Behaviors that affect contact rates among hosts could be important for viral diversification because more frequent intra- and inter-specific contacts among hosts should increase the potential for viral diversification within host populations. We used published data on bats to test the contact-rate hypothesis. We predicted that species forming large conspecific groups, that share their range with more heterospecifics (i.e., sympatry), and with mating systems characterized by high contact rates (polygynandry: multi-male/multi-female), would host higher viral richness than species with small group sizes, lower sympatry, or low contact-rate mating systems (polygyny: single male/multi-female). Consistent with our hypothesis and previous research, viral richness was positively correlated with conspecific group size although the relationship plateaued at group sizes of approximately several hundred thousand bats. This pattern supports epidemiological theory that, up to a point, larger groups have higher contact rates, greater likelihood of acquiring and transmitting viruses, and ultimately greater potential for viral diversification. However, contrary to our hypothesis, there was no effect of sympatry on viral richness and no difference in viral richness between mating systems. We also found no residual effect of host phylogeny on viral richness, suggesting that closely related species do not necessarily host similar numbers of viruses. Our results support the contact-rate hypothesis that intra-specific viral transmission can enhance viral diversification within species and highlight the influence of host group size on the potential of viruses to propagate within host populations.

**Keywords:** Chiroptera, Contact-rate, Mating systems, Sociality, Viral transmission

## Introduction

Identifying host traits that influence pathogen diversity is important for understanding wildlife pathogen dynamics. Predicting viral diversity within hosts has gained recent attention because species with high viral richness may be

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more likely to initiate spillover events to humans or livestock (Nunn et al. 2005; Lindenfors et al. 2007; Turmelle and Olival 2009; Luis et al. 2013; Olival et al. 2017). Characterizing host traits that favor viral richness could help inform predictive models to forecast potential for zoonotic outbreak where pathogens are transmitted from animals to humans (Morse et al. 2012; Gortazar et al. 2014).

Viral establishment within a new host population involves two main steps (Antia et al. 2003). First, the virus needs to encounter the new host. Viruses can be introduced from other species (inter-specific transmission or spillover). Successful inter-specific transmission relies on physical opportunities for transmission such as occupying the same environment at the same time (Pedersen and Davies 2010). Novel viruses can also emerge within a host species via mutation owing, in part, to the rapid mutation rates of many viruses (Pulliam 2008; Plowright et al. 2015, Holmes and Drummond 2007). Second, once a virus has encountered a new host species, it needs to persist. Successful propagation and persistence can depend on viral characteristics, such as mutation rates (again rapid rates of mutation can enable rapid evolution of adaptations to novel environments in new host species), but host characteristics, including life-history traits or population density, are also important (Brierley et al. 2016). Persistence should be related to host population density because large populations may have many susceptible hosts with high contact rates compared to highly dispersed populations.

Once a virus has infected a new host species, epidemiological dynamics can be complex (Brierley et al. 2016), but similar principles that apply to inter-specific transmission also affect intra-specific transmission. The potential of a virus to persist within a species has primarily been attributed to host–pathogen characteristics (e.g., duration of the infectious period) that affect the number of secondary infections arising from an initial infection (i.e., the basic reproductive number, $R_0$). Epidemics occur when values of $R_0$ are greater than one (Dietz 1993; Antia et al. 2003). In addition to $R_0$, however, host traits that favor viral transmission should also favor viral establishment and an increase in viral diversification and richness within that host population. For most viruses, hosts of the same species must typically occupy the same physical environment at the same time for viruses to be propagated within a species.

Host geographic distribution is one characteristic of host ecology known to predict viral richness because distribution can facilitate, or limit, inter-specific transmission (Nunn et al. 2005). Host behavior is also important because it can mediate viral transmission (Antia et al. 2003), which could subsequently facilitate viral propagation and increase viral richness within a new host species. For example, in a recent meta-analysis, species with larger social networks had higher rates of pathogen transmission, but when the large social networks were subdivided into multiple small networks, transmission was reduced (Nunn et al. 2015). Group size, which often, but not always, correlates with contact and transmission rate (VanderWaal and Ezenwa 2016), is the most commonly examined behavioral predictor of viral richness. Species-specific group size is correlated with endo- (Lindenfors et al. 2007) and ectoparasite richness (Bordes et al. 2007), but the relationship between group size and viral richness is equivocal with variation in the direction of the relationship among host taxa (Ezenwa et al. 2006; Gay et al. 2014; Luis et al. 2015).

Mating system dynamics are another aspect of host behavior that could mediate contact rates and viral richness. Vertebrates generally display one of three types of mating systems: (1) monogamy (single male/single female); (2) polygyny (single male/multi-female); and (3) polygynandry (multi-male/multi-female) (Clutton-Brock 1989). These mating systems will differ in terms of contact rates among group members. For polygynous species that live in stable harem groups, transmission via bodily fluids or fomites should primarily occur among members of the same harem (Nunn et al. 2008). In contrast, social groups of polygynandrous species exhibit reduced stability and higher intra-specific contact rates, increasing potential transmission (Nunn et al. 2008).

Bats (Chiroptera) provide a good model taxon to examine the relationship between mating system and viral richness. Bats display all mating systems described above, and social groups range in size across several orders of magnitude. Bats are also reservoir hosts for viruses of public health significance (Luis et al. 2013), such as Marburg, Hendra, and Nipah viruses (Daszak et al. 2006). This has led to recent prospecting to catalog novel viruses in bats, providing a database of adequate size for comparative analyses (Chen et al. 2014). Our objective was to use this published database to test what we term the intra-specific contact-rate hypothesis, that behavioral and ecological characteristics affecting intra- and inter-specific contact rates influence viral richness within bats. We predicted that: (1) species living in larger roosting groups exhibit greater viral richness than species in smaller groups; (2) polygynandrous bats host greater viral richness than polygynous bats because of a greater potential for viral transmission

during promiscuous mating; and (3) focal species with higher bat sympatry exhibit greater viral richness than species with lower bat sympatry.

## Methods

### Data Collection

We obtained values of viral richness for bats from 'the database of bat-associated viruses' (Chen et al. 2014). We omitted multiple instances of infection by different strains of the same viral species. For example, our search yielded 18 unique strains of rabies virus for species in our database, but we considered rabies as a single virus in our analyses. We included host–virus associations based on all detection methods used for studies in the database, including polymerase chain reaction and high-throughput sequencing methods. We also included zoonotic and non-zoonotic viruses as well as retroviruses. Our search of the database was conducted on January 8, 2017.

Previous research indicates that parasite richness is positively correlated with the amount of research conducted on a given host species (Nunn et al. 2005; Lindenfors et al. 2007), and research effort (i.e., number of citations) was shown to be highly correlated with viral species richness (Turmelle and Olival 2009; Luis et al. 2013; Luis et al. 2015). Therefore, we searched Web of Science for the Linnaean classification names of the bats examined in our analysis (and their synonyms; searches conducted on January 22, 2017), and we extracted the number of publications for each species in our database.

We quantified host-specific behavioral and ecological traits using literature searches. First, we categorized each bat species as either polygynous or polygynandrous following McCracken and Wilkinson (2000). Second, we quantified maximum group size for each species from mammalian species accounts and other peer-reviewed sources describing a given species' roosting behavior (see supplementary material for references). Third, we quantified the number of other bat species with overlapping geographic distributions (i.e., sympatry) (Luis et al. 2013). We also quantified the average latitude of the geographic range because latitude was correlated with viral richness in past analyses (Lindenfors et al. 2007). We calculated latitude and sympatry using data obtained through the International Union for the Conservation of Nature Red List of Threatened Species (IUCN 2012) as two-dimensional shapefiles. Sympatry for each species was determined by summing the total number of other bat species with overlapping distributions. We determined the centroid coordinate for all species-specific shapefiles, calculated the mean latitude, weighted based on the size ($km^2$) of all shapefiles associated with that species, and then used the species-specific absolute latitude value (i.e., regardless of longitude or hemisphere) as a covariate in the analyses. We also included body mass (g), extracted from the PanTHERIA database (Jones et al. 2009) and diet, extracted from the database of bat-associated viruses (Chen et al. 2014) in our models.

### Statistical Analyses

All analyses were conducted in R (R Development Core Team 2016). We followed previous comparative studies of parasite richness by correcting for host, but not viral, phylogeny using the mammalian super-tree (Jones et al. 2002; Bininda-Emonds et al. 2007) trimmed to match the bat species in our dataset (available as supplementary material). In cases where current bat taxonomy is under debate, we deferred to the Bininda-Emonds et al. (2007) super-tree and did not change branch lengths or split species into sub-species or very recently discovered 'sister' species, including the *Miniopterus* species complex (Tian et al. 2004). Including or excluding *Miniopterus schreibersii* had no effect on our results or conclusions so we retained *M. schreibersii* as a single species for our analyses.

We used phylogenetic generalized least squares (PGLS, R Package 'ape': Paradis et al. 2017) models based on maximum likelihood to account for phylogenetic signal ($\lambda$) (Blomberg and Garland 2002). Phylogenetic signal is a measure of trait divergence among species. Values of $\lambda$ range from zero to one, where $\lambda = 1$ means patterns are fully explained by phylogeny and a given trait is similar among closely related species, while values of $\lambda = 0$ mean no phylogenetic signal and closely related species do not share similar values of a given trait (Blomberg and Garland 2002). We also calculated Blomberg's *K*, a metric which estimates the phylogenetic signal of a given trait. Higher values of *K* mean stronger phylogenetic signal for that trait (for details, see Blomberg et al. 2003). We estimated Blomberg's *K* for viral richness as well as all explanatory variables (see below) using the 'phytools' package in R (Revell 2012) and present p values alongside values of Blomberg's *K* (see below).

The dependent variable for our analyses was $\log_{10}$-transformed total viral richness, which was obtained by

summing the unique number of viral species infecting a given bat species, as determined from our search of Chen et al.'s (2014) database. We $\log_{10}$-transformed the number of publications and group size to satisfy assumptions of our models. We then used PGLS models to test for effects of the number of publications ($\log_{10}$-transformed), group size ($\log_{10}$-transformed), mating system, sympatry, latitude, body mass ($\log_{10}$-transformed), and diet on $\log_{10}$-transformed viral richness. We used variance inflation factors (VIFs) to test for violations of multi-collinearity in our initial model. Our initial model suggested a violation of multi-collinearity, where latitude (VIF = 7.5) was correlated with all other variables so we removed latitude from subsequent analyses. We used the Akaike information criterion with small sample bias adjustment ($AIC_C$) to assess relative support for fixed effect variables in 26 PGLS models, including an intercept-only model. The model with the lowest $AIC_C$ was considered the most parsimonious. We inferred support for the best model by examining $AIC_C$ differences ($\Delta AIC_C$), $AIC_C$ weights ($w_i$), and cumulative $AIC_C$ weights ($accw_i$). We considered models with $\Delta$ $AIC_C$ values $\leq 2$ to have similar support (Burnham and Anderson 2002). Therefore, we assumed that the best model was the simplest model (i.e., with the smallest number of parameters) whose $AIC_C$ was less than 2 units greater than that of the model associated with the smallest $AIC_C$ (Burnham and Anderson 2002). A given predictor variable may falsely appear to have support because of the presence of other important variables in that model (Burnham and Anderson 2002, p. 131). $AIC_C$ weights sum to one across all models and reflect the probability a given model is the most parsimonious among the candidate models.

## Results

We identified 168 unique viruses that infected 51 bat species ($n$ = 27 polygynandrous and 24 polygynous) for which we also assigned a mating system ($N$ = 339 unique host–virus associations: Table 1). Mean total viral richness prior to $\log_{10}$-transformation was 6.5 ± 6.6 (SD, range = 1–33) viruses per bat species (Fig. 1). Prior to log-transformation, mean group size was $4.3 \times 10^5 \pm 2.7 \times 10^6$ (range = 1–$2.0 \times 10^7$, see supplementary materials). However, the distribution of group sizes in our dataset was significantly right-skewed with most species exhibiting group sizes < 10,000 individuals (median group size = 234; Fig. S1). After $\log_{10}$-transformation, mean group size was 2.7 ± 1.5 (SD, range for $\log_{10}$-transformed = 0–7.3). Prior to $\log_{10}$-transformation, the mean number of publications on a given bat species was 186 ± 264, but, again, ranged widely (range = 4–1425) with relatively few studies for most species (Fig. S2). After $\log_{10}$-transformation, the number of publications about a given species was 1.94 ± 0.57 (range for $\log_{10}$-transformed = 0.6–3.15; Fig. S2).

Based on the best-fit PGLS model, there was a positive logarithmic relationship between group size and viral richness, with species roosting in larger colonies hosting more viruses than species roosting in smaller colonies (Fig. 2a; ß = 0.1 ± 0.04, $t$ = 2.6, $p$ = 0.01). This effect was especially pronounced at relatively small colony sizes of < 10,000 bats such that, for each order of magnitude increase in group size, viral richness increases by approximately 25% (Fig. 2b, Table 2). We also found a positive relationship between number of publications and viral richness (Fig. 2c; ß = 0.36 ± 0.10, $t$ = 3.4, $p$ = 0.001). Mating system, body mass, and sympatry were present in several models with low $AIC_C$ values (Table 2) but including these variables did not improve model fit, suggesting that the data did not support these statistical associations. Diet did not appear in any top models (Table 2). For the best-fit PGLS model, $\lambda = 0$, indicating that phylogeny explained no residual variation in the relationship between viral richness and our predictor variables. Similarly, Blomberg's $K$ varied for our predictor variables. Phylogenetic signal was relatively weak for mating system ($K$ = 0.23) and number of publications ($K$ = 0.38), moderate for sympatry ($K$ = 0.52) and group size ($K$ = 0.64), and high for diet ($K$ = 1.03) and body mass ($K$ = 1.1).

## Discussion

We found support for the contact-rate hypothesis and our prediction that social group size would be positively correlated with viral richness (Fig. 2a). These results are consistent with the epidemiological assumption that, up to a point, larger group sizes result in higher contact rates and that high contact rates among hosts facilitate viral transmission (Nunn et al. 2015). Increased rates of viral transmission among group members presumably increase the likelihood of new viruses establishing within a population or species, thus increasing overall viral diversity.

The use of a continuous measure of group size enabled us to quantify a positive, logarithmic relationship between group size and viral richness that plateaued above group

**Table 1.** Summary of Virus Data Used for Analyses, Including the Number of Virus Species per Virus Family Detected in Bats as Well as the Number of Unique Host Bat Species Infected by a Given Virus Family.

| Virus family | Number of unique virus species detected in bats | Number of unique host bat species |
|---|---|---|
| Adenoviridae | 23 | 16 |
| Anelloviridae | 1 | 1 |
| Astroviridae | 15 | 12 |
| Bornaviridae | 2 | 2 |
| Bunyaviridae | 9 | 7 |
| Caliciviridae | 5 | 5 |
| Circoviridae | 7 | 6 |
| Coronaviridae | 62 | 30 |
| Filoviridae | 3 | 3 |
| Flaviviridae | 18 | 11 |
| Hepadnaviridae | 1 | 1 |
| Hepeviridae | 3 | 3 |
| Herpesviridae | 34 | 19 |
| Papillomaviridae | 7 | 7 |
| Paramyxoviridae | 41 | 27 |
| Parvoviridae | 14 | 9 |
| Picornaviridae | 15 | 8 |
| Polyomaviridae | 11 | 11 |
| Poxviridae | 1 | 1 |
| Reoviridae | 16 | 13 |
| Retroviridae | 7 | 6 |
| Rhabdoviridae | 44 | 30 |

sizes of about 10,000 bats. Past studies that have used categorical metrics of group size seem to have been unable detect this relationship (e.g., Gay et al. 2014; Luis et al. 2015). Our results suggest there is a threshold group size above which the increase in viral richness associated with group size begins to plateau (Ryder et al. 2007). This makes sense intuitively when considered in the context of density-dependent pathogen transmission in humans. The difference in potential for pathogen introduction and establishment is likely to be large when population density is relatively high, for example, in a human context, in large cities versus small towns or villages. However, the difference in pathogen transmission within large cities of, for example, 1 million people compared to even larger cities of 5 or 10 million people will be less pronounced. Our results suggest that similar dynamics are at play across bat species which, like different communities of humans, also display an enormous range in terms of aggregation sizes.

Our results also have potential implications for our ability to predict and manage risk of zoonotic outbreaks. Most simplistically, species roosting in larger colonies, which tend to host more viruses, could have greater potential to initiate zoonotic outbreaks. For instance, Hendra virus is a zoonotic virus hosted by flying foxes (*Pteropus* sp.), which often roost in group sizes ranging from hundreds of thousands to million individuals (Daszak et al. 2006). Anthropogenic destruction of natural roosting habitat has increased the probability of flying fox aggregation in urban areas which could increase the chance of Hendra spillover (Plowright et al. 2015). Webber et al. (2016) used epidemiological models to show that colonies of *Eptesicus fuscus* that exhibit fission–fusion dynamics and are divided among multiple roost trees each day, differ from *E. fuscus* colonies roosting in buildings, where colonies are often larger (Lausen and Barclay 2006) and the entire colony is typically not subdivided into multiple roosts each day. Based on network epidemiological models, Webber et al. (2016) showed that social structure and organization of bat colonies in buildings without fission–fusion dynamics led to faster proliferation of a novel pa-

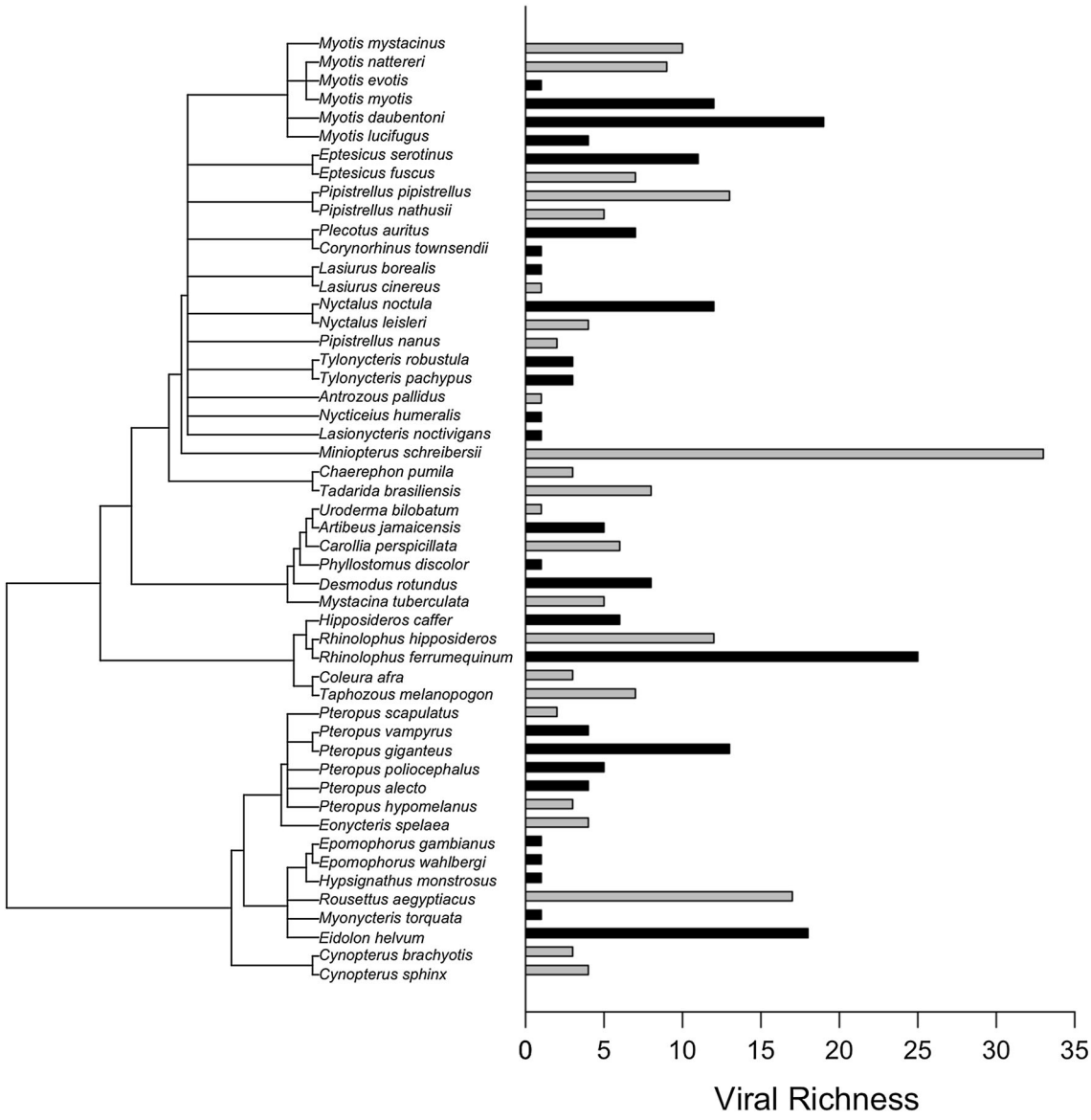


**Figure 1.** Phylogenetic tree of 51 bat species (Bininda-Emonds et al. 2007) and horizontal barplot of viral richness for each species. Gray bars indicate polygynous species, and black bars indicate polygynandrous species.

thogen than would occur in tree-roosting bats of the same species.

This difference in social structure and pathogen risk between trees and buildings reflects realistic scenarios that could occur with urbanization or agricultural development in forests. Bats provide critical ecosystem services in temperate and tropical regions (e.g., Kalka et al. 2008; Boyles et al. 2011; Maine and Boyles 2015), and their conservation should be an important priority. Thus, we suggest that management actions maintain the abundance of high-quality, natural roosting habitat for bats. For example, minimizing urban or agricultural development in natural-ized bat habitats or, if development cannot be avoided, employing bat-proof construction in combination with replacement habitat that mimics natural roosting opportunities as in the original forest, could be beneficial (e.g., a large number of small bat houses, as opposed to a small number of very large bat houses, Webber et al. 2016). One implication of our detection of a plateau in the relationship between colony size and viral richness is that management efforts which avoid favoring unnaturally large aggregations of bats will be especially important and useful for species with small- to medium-sized colonies (Fig. 2b). For species with very large colonies of hundreds of thousands to mil-

**Table 2.** Summary of Top 10 PGLS Models Based on Akaike Information Criteria ($AIC_C$) to Test the Effects of $log_{10}$-Transformed Group Size, $log_{10}$-Transformed Publications (i.e., Pub), Sympatry, and Mating System (i.e., Mating), Body Mass and Diet on $log_{10}$-Transformed Viral Richness.

| PGLS model | $AIC_C$ | $\Delta$ $AIC_C$ | $w_i$ | $accw_i$ | Model $\lambda$ | Model $r^2$ |
|---|---|---|---|---|---|---|
| ~ pub + group size | 47.45 | 0 | 0.306 | 0.306 | 0 | 0.32 |
| ~ pub + group size + mating | 48.09 | 0.64 | 0.222 | 0.528 | 0 | 0.35 |
| ~ pub + group size + body mass | 49.06 | 1.61 | 0.137 | 0.665 | 0 | 0.33 |
| ~ pub + group size + mating + body mass | 50.20 | 2.75 | 0.077 | 0.742 | 0 | 0.35 |
| ~ pub + group size + mating + sympatry | 50.55 | 3.10 | 0.065 | 0.807 | 0 | 0.35 |
| ~ pub + mating | 50.84 | 3.39 | 0.056 | 0.863 | 0.71 | 0.32 |
| ~ pub + group size + sympatry + body mass | 51.41 | 3.96 | 0.042 | 0.905 | 0 | 0.34 |
| ~ pub | 51.52 | 4.07 | 0.04 | 0.945 | 0.60 | 0.27 |
| ~ pub + group size + mating + sympatry + body mass | 52.77 | 5.34 | 0.021 | 0.966 | 0 | 0.35 |
| ~ pub + mating + body mass | 52.80 | 5.36 | 0.021 | 0.987 | 0.79 | 0.33 |

For top models, lambda ($\lambda$) was 0, indicating little effect of residual phylogeny on the relationship between viral richness and the predictor variables. We included $\Delta$ $AIC_C$, model weight ($w_i$), and accumulated model weight ($accw_i$) to illustrate the importance of number of publications and group size

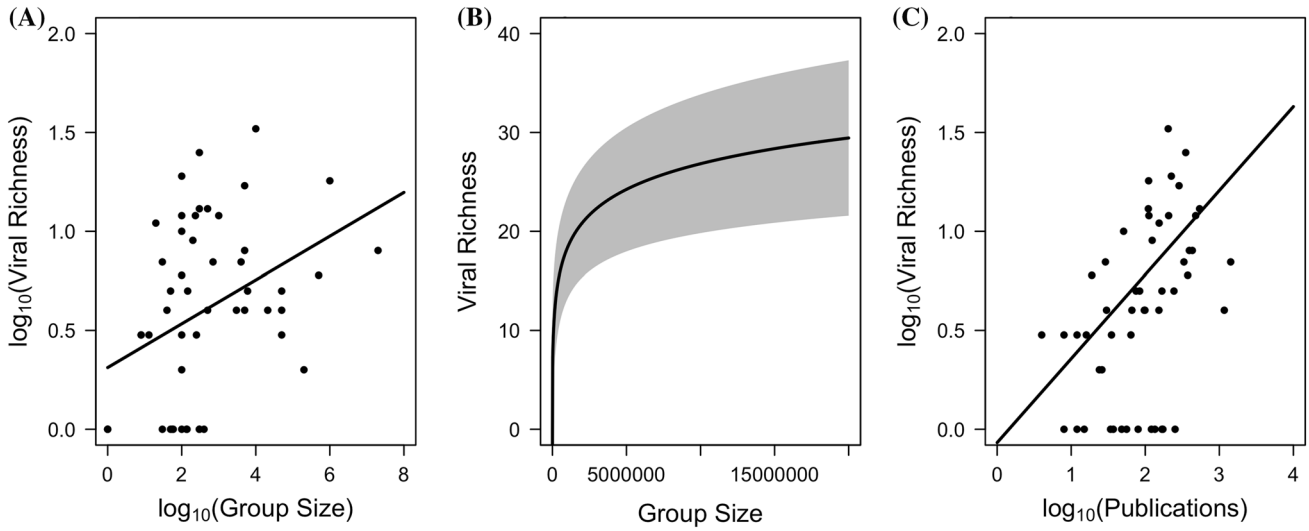


**Figure 2.** **a** Positive relationship between $log_{10}$-transformed viral richness and $log_{10}$-transformed group size for 51 bat species; **b** plateau relationship between untransformed viral richness and group size from Fig. 2a estimated based on coefficients extracted from our best-fit model (Table 2). Gray shading represents standard error around predicted values; **c** positive relationship between $log_{10}$-transformed viral richness and $log_{10}$-transformed number of publications on a given species. Trend lines for panels **a** and **c** generated from phylogenetic least-square model (see results for parameter estimates).

lions of bats, our results suggest that a further increase in colony size will have a relatively small impact on overall viral richness within that host species.

In contrast to our prediction, we found no relationship between viral richness and sympatry in bats. While sympatry has been found to predict zoonotic viral richness in bats and rodents (Luis et al. 2013) and viral sharing in primates (Pedersen and Davies 2010), the relationship between sympatry and overall viral richness may be more complicated for bats than previously predicted. While sympatric species are more likely to share the same space due to their geographic overlap, many sympatric bats may not encounter one another due to extreme inter-specific variation in roosting behavior. For instance, some bats roost in ephemeral structures, such as foliage and leaf-tents, while others roost in large, permanent structures, such as

caves (Lewis 1995). This distinction could be important for two reasons. First, species roosting in ephemeral roosts may not encounter heterospecifics very frequently, thus limiting the potential for viral transmission and subsequent diversification among species. Second, large caves may house dozens of bat species, thus increasing the likelihood of heterospecific contact and the potential for viral transmission and diversification among sympatric hosts. The implications of this type of habitat partitioning among sympatric hosts could have implications not only for viral transmission among bat heterospecifics, but also for viral transmission among non-bat heterospecifics, such as rodents (Luis et al. 2013). We suggest that future studies attempt to delineate sympatry in the context of roosting niche and dietary niche overlap among species and determine potential influence of niche sharing on inter-specific viral transmission.

Although we predicted that polygynandrous species would have higher viral richness than polygynous species, we found no difference. One explanation could be that mating system may only predict richness for sexually transmitted viruses (Nunn et al. 2014). For instance, the prevalence of sexually transmitted diseases was higher for females from promiscuous primate species compared to females from species with polygynous or monogamous mating systems (Nunn et al. 2014). The same could be the case for bats. However, identifying which viruses are sexually transmitted in bats is difficult because most bats are highly cryptic and direct observation of copulation followed by viral testing is likely impossible for most bat species. Alternatively, this result could reflect a flaw in our assumptions about contact rates associated with different mating systems. For instance, harems can vary in the degree of stability among females, where some species have stable female group composition, and others are associated with ephemeral female group composition and females that frequently move among harems (McCracken and Wilkinson 2000). Classifying species as either polygynous or polygynandrous could therefore underestimate variation in contact rates associated with different mating systems (McCracken and Wilkinson 2000). We suggest that future studies attempt to quantify variation in sexual and non-sexual contact rates for bat species with different mating systems to help better explain variation in viral richness. While we found no effect of mating system on viral richness in bats, the influence of mating system on population social and genetic structure (Wilkinson 1985) could still be an important predictor of population-wide connectivity and contact rates, processes which can regulate pathogen dynamics and outbreaks (Altizer et al. 2003).

We found no influence of host phylogeny on viral richness. Although closely related species tend to share many of the same viruses (Luis et al. 2015), somewhat counterintuitively, our results, and those from previous studies (e.g., Turmelle and Olival 2009; Luis et al. 2013) suggest that phylogeny explains little variance in the relationship between viral richness and our predictor variables. One hypothesis is that although closely related species may host the same viruses, these shared viruses account for only a small proportion of total viral richness (Anthony et al. 2013). Alternatively, host species within a given clade may be likely to share a similar combination of viruses, but if some species within the clade are particularly suitable hosts, they may host greater viral richness than the rest of the clade, thereby obscuring possible phylogenetic signal in viral richness.

Previous studies assessing the influence of host behavior on viral richness have used population genetic structure (Turmelle and Olival 2009) and group size (Luis et al. 2015) as surrogates for contact rate. Our results, in combination with these studies, suggest the relationship between viral richness and host social behavior may be more complex than previously predicted and that direct estimates of contact rates, both within and across species, may be needed to better predict viral richness. To further disentangle effects of group size, and possibly mating system, on viral transmission, we suggest that future studies quantify contact rates for both polygynandrous and polygynous species. The most promising technology for quantifying contact rates involves proximity devices, which, if used to provide data for social network analyses, could be particularly useful (e.g., Hamede et al. 2009). Individual social network metrics may reflect the likelihood of viral acquisition or transmission among group members, while group-level social network structure may predict variation in contact rates between host species with different types of mating systems (Craft and Caillaud 2011). In addition, interactions among species within day roosts or during foraging could be particularly useful to elucidate potential transmission mechanisms among species (Serra-Cobo et al. 2013; Webber et al. 2016). A better understanding of bat community dynamics could therefore be valuable for understanding the influence of host social behavior on viral richness.

Our results suggest a link between group size and viral richness for bats but should be interpreted with caution

given the relatively small proportion of all bat species included in our analysis. Little is known about the roosting and social behavior for a large proportion of the approximately 1300 species of bats (Simmons 2005; Kerth 2008) and even less is known about host–virus dynamics for these species. Additional data on group sizes, roosting behavior, and viral richness of additional species would strengthen the analysis we conducted and improve the chance of detecting biologically important effects.

Our results provide support for the contact-rate hypothesis that rates of intra-specific transmission among hosts can influence rates of viral diversification and viral richness. The relationship between host behavior and pathogen risk is predicated on the assumption that individuals, population, and species that aggregate in groups have greater risk of acquiring, transmitting, and potentially retaining novel pathogens (Altizer et al. 2003; Tompkins et al. 2011), and our results support this assumption. However, our results also suggest the effect of colony size on viral richness is greatest for species that roost in small- to medium-sized colonies because the effect of group size on viral richness plateaus at extremely large group sizes. Our study highlights the importance of integrating behavioral traits in studies on viral richness with an emphasis on the behavioral mechanisms which could drive viral transmission and ultimately predict viral diversification and richness.

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## References

Altizer S, Nunn C, Thrall P, et al. (2003) Social organization and parasite risk in mammals: Integrating theory and empirical studies. *Annual Review of Ecology Evolution and Systematics* 34:517–547. doi:10.1146/annurev.ecolsys.34.030102.151725

Anthony SJ, Epstein JH, Murray KA, et al. (2013) A strategy to estimate unknown viral diversity in mammals. *MBio* 4:e00598. doi:10.1128/mBio.00598-13.Editor

Antia R, Regoes RR, Koella JC, Bergstrom CT (2003) The role of evolution in the emergence of infectious diseases. *Nature* 426:658–661. doi:10.1038/nature02177.1

Bininda-Emonds ORP, Cardillo M, Jones KE, et al. (2007) The delayed rise of present-day mammals. *Nature* 446:507–513. doi:10.1038/nature05634

Blomberg S, Garland T, Ives AR (2003) Testing for phylogenetic signal in comparative data: Behavioral traits are more labile. *Evolution* 57:717–745

Blomberg SP, Garland T (2002) Tempo and mode in evolution: Phylogenetic inertia, adaptation and comparative methods. *Journal of Evolutionary Biology* 15:899–910

Bordes F, Blumstein DT, Morand S (2007) Rodent sociality and parasite diversity. *Biology Letters* 3:692–694. doi:10.1098/rsbl.2007.0393

Boyles JG, Cryan PM, Mccracken GF, Kunz TH (2011) Economic importance of bats in agriculture. *Science* 332(80):41–42. doi:10.1126/science.1201366

Brierley L, Vonhof MJ, Olival KJ, et al. (2016) Quantifying global drivers of zoonotic bat viruses: A process-based perspective. *American Naturalist* 187:E53–E64. doi:10.1086/684391

Burnham KP, Anderson DR (2002) *Model selection and multimodel inference, 2nd ed.*, New York: Springer

Chen L, Liu B, Yang J, Jin Q (2014) DBatVir: The database of bat-associated viruses. *Database* 2014:1–7. doi:10.1093/database/bau021

Clutton-Brock TH (1989) Mammalian mating systems. *Proceedings of the Royal Society of London B: Biological Sciences* 236:339–372. doi:10.1098/rspb.1989.0027

Craft ME, Caillaud D (2011) Network models: An underutilized tool in wildlife epidemiology? *Interdisciplinary Perspectives on Infectious Diseases*. doi:10.1155/2011/676949

Daszak P, Plowright RK, Epstein JH, et al (2006) The emergence of Nipah and Hendra virus: Pathogen dynamics across a wildlife-livestock-human continuum. In: *Disease ecology: Community Structure and Pathogen Dynamics*, pp 186–201

Dietz K (1993) The estimation of the basic reproduction number for infectious diseases. *Statistical Methods in Medical Research* 2:23–41. doi:10.1177/096228029300200103

Ezenwa VO, Price SA, Altizer S, et al. (2006) Host traits and parasite species richness in even and odd-toed hoofed mammals, Artiodactyla and Perissodactyla. *Oikos* 115:526–536. doi:10.1111/j.2006.0030-1299.15186.x

Gay N, Olival KJ, Bumrungsri S, et al. (2014) Parasite and viral species richness of Southeast Asian bats: Fragmentation of area distribution matters. *International Journal for Parasitology* 3:161–170. doi:10.1016/j.ijppaw.2014.06.003

Gortazar C, Reperant LA, Kuiken T, et al. (2014) Crossing the interspecies barrier: Opening the door to zoonotic pathogens. *PLoS Pathogens* 10:e1004129. doi:10.1371/journal.ppat.1004129

Hamede RK, Bashford J, McCallum H, Jones M (2009) Contact networks in a wild Tasmanian devil (Sarcophilus harrisii) population: Using social network analysis to reveal seasonal variability in social behaviour and its implications for transmission of devil facial tumour disease. *Ecology Letters* 12:1147–1157. doi:10.1111/j.1461-0248.2009.01370.x

Holmes EC, Drummond AJ (2007) The evolutionary genetics of viral emergence. In: *Wildlife and Emerging Zoonotic Diseases: The Biology, Circumstances and Consequences of Cross-Species Transmission*, Childs JE, Mackenzie JS, Richt JA (editors), Berlin, Heidelberg: Springer, pp 51–66

IUCN (2012) *IUCN Red List of Threatened Species*. Version 2012.1. http://www.iucnredlist.org. Accessed 1 Mar 2013

Jones KE, Bielby J, Cardillo M, et al. (2009) PanTHERIA: A species-level database of life history, ecology, and geography of extant and recently extinct mammals. *Ecology* 90:2648

Jones KE, Purvis A, MacLarnon A, et al. (2002) A phylogenetic supertree of the bats (Mammalia: Chiroptera). *Biological Reviews* 77:223–259

Kalka MB, Smith AR, Kalko EKV (2008) Bats limit arthropods and herbivory in a tropical forest. *Science* 320(80):71–72. doi:10.1126/science.1153352

Kerth G (2008) Causes and consequences of sociality in bats. *BioScience* 58:737–746. doi:10.1641/B580810

Lausen CL, Barclay RMR (2006) Benefits of living in a building: Big brown bats (*Eptesicus fuscus*) in rocks versus buildings. *Journal of Mammalogy* 87:362–370. doi:10.1644/05-MAMM-A-127R1.1

Lewis SE (1995) Roost fidelity of bats: A review. *Journal of Mammalogy* 76:481–496. doi:10.2307/1382357

Lindenfors P, Nunn CL, Jones KE, et al. (2007) Parasite species richness in carnivores: Effects of host body mass, latitude, geographical range and population density. *Global Ecology and Biogeography* 16:496–509. doi:10.1111/j.1466-8238.2006.00301.x

Luis AD, Hayman DTS, O'Shea TJ, et al. (2013) A comparison of bats and rodents as reservoirs of zoonotic viruses: Are bats special? *Proceedings of the Royal Society B* 280:20122753. doi:10.1098/rspb.2012.2753

Luis AD, O'Shea TJ, Hayman DTS, et al. (2015) Network analysis of host-virus communities in bats and rodents reveals determinants of cross-species transmission. *Ecology Letters* 18:1153–1162. doi:10.1111/ele.12491

Maine JJ, Boyles JG (2015) Bats initiate vital agroecological interactions in corn. *Proceedings of National Academy of Sciences* 112:12438–12443. doi:10.1073/pnas.1505413112

McCracken GF, Wilkinson GS (2000) Bat mating systems. In: *Reproductive Biology of Bats*, Chrichton EG, Krutzsch PH (editors), San Diego: Academic Press, pp 321–362

Morse SS, Mazet JAK, Woolhouse M, et al. (2012) Prediction and prevention of the next pandemic zoonosis. *Lancet* 380:1956–1965. doi:10.1016/S0140-6736(12)61684-5

Nunn CL, Altizer SM, Sechrest W, Cunningham AA (2005) Latitudinal gradients of parasite species richness in primates. *Diversity and Distributions* 11:249–256. doi:10.1111/j.1366-9516.2005.00160.x

Nunn CL, Jordan F, McCabe CM, et al. (2015) Infectious disease and group size: More than just a numbers game. *Philosophical Transactions of the Royal Society B: Biological Sciences* 370:20140111. doi:10.1098/rstb.2014.0111

Nunn CL, Scully EJ, Kutsukake N, et al. (2014) Mating competition, promiscuity, and life history traits as predictors of sexually transmitted disease risk in primates. *International Journal of Primatology* 35:764–786. doi:10.1007/s10764-014-9781-5

Nunn CL, Thrall PH, Stewart K, Harcourt AH (2008) Emerging infectious diseases and animal social systems. *Evolutionary Ecology* 22:519–543. doi:10.1007/s10682-007-9180-x

Olival KJ, Hosseini PR, Zambrana-torrelio C, et al. (2017) Host and viral traits predict zoonotic spillover from mammals. *Nature* 546:646–650. doi:10.1038/nature22975

Paradis E, Blomberg S, Bolker B, et al. (2017) *Package "ape": Analyses of Phylogenetics and Evolution*

Pedersen AB, Davies TJ (2010) Cross-species pathogen transmission and disease emergence in primates. *EcoHealth* 6:496–508. doi:10.1007/s10393-010-0284-3

Plowright RK, Eby P, Hudson PJ, et al. (2015) Ecological dynamics of emerging bat virus spillover. *Proceedings of the Royal Society B* 282:20142124

Pulliam JRC (2008) Viral host jumps: Moving toward a predictive framework. *EcoHealth* 5:80–91. doi:10.1007/s10393-007-0149-6

R Development Core Team (2016) *R: A Language and Environment for Statistical Computing*. R Foundation. https://www.r-project.org/.

Revell LJ (2012) phytools: An R package for phylogenetic comparative biology (and other things). *Methods in Ecology and Evolution* 3:217–223. doi:10.1111/j.2041-210X.2011.00169.x

Ryder JJ, Miller MR, White A, et al. (2007) Host-parasite population dynamics under combined frequency- and density-dependent transmission. *Oikos* 116:2017–2026. doi:10.1111/j.2007.0030-1299.15863.x

Serra-Cobo J, López-Roig M, Seguí M, et al. (2013) Ecological factors associated with European Bat lyssavirus seroprevalence in Spanish bats. *PLoS ONE* 8:e64467. doi:10.1371/journal.pone.0064467

Simmons NB (2005) Order chiroptera. In: *Mammal Species of the World: A Taxonomic and Geographic Reference*. John Hopkins University Press, pp 312–529

Tian L, Liang B, Maeda K, et al. (2004) Molecular studies on the classification of Miniopterus schreibersii (Chiroptera: Vespertilionidae) inferred from mitochondrial cytochrome b sequences. *Folia Zoologica* 53:303–311

Tompkins DM, Dunn AM, Smith MJ, Telfer S (2011) Wildlife diseases: From individuals to ecosystems. *Journal of Animal Ecology* 80:19–38. doi:10.1111/j.1365-2656.2010.01742.x

Turmelle AS, Olival KJ (2009) Correlates of viral richness in bats (Order Chiroptera). *EcoHealth* 6:522–539. doi:10.1007/s10393-009-0263-8

VanderWaal KL, Ezenwa VO (2016) Heterogeneity in pathogen transmission: Mechanisms and methodology. *Functional Ecology* 30:1606–1622. doi:10.1111/1365-2435.12645

Webber QMR, Brigham RM, Park AD, et al. (2016) Social network characteristics and predicted pathogen transmission in summer colonies of female big brown bats (Eptesicus fuscus). *Behavioral Ecology and Sociobiology*. doi:10.1007/s00265-016-2093-3

Wilkinson GS (1985) The social organization of the common vampire bat: II Mating system, genetic structure, and relatedness. *Behavioral Ecology and Sociobiology* 17:123–134