Temporal and spatial limitations in global surveillance for bat filoviruses and henipaviruses

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Abstract

Sampling reservoir hosts over time and space is critical to detect epizootics, predict spillover, and design interventions. Yet spatiotemporal sampling is rarely performed for many reservoir hosts given high logistical costs and potential tradeoffs between sampling over space and time. Bats in particular are reservoir hosts of many virulent zoonotic pathogens such as filoviruses and henipaviruses, yet the highly mobile nature of these animals has limited optimal sampling of bat populations across both space and time. To quantify the frequency of temporal sampling and to characterize the geographic scope of bat virus research, we here collated data on filovirus and henipavirus prevalence and seroprevalence in wild bats. We used a phylogenetically controlled meta-analysis to next assess temporal and spatial variation in bat virus detection estimates. Our analysis demonstrates that only one in five studies sample bats longitudinally, that bat sampling efforts cluster geographically (e.g., filovirus data are available across much of Africa and Asia but are absent from Latin America and Oceania), and that reporting trends may affect some viral detection estimates (e.g., filovirus seroprevalence). Within the limited number of longitudinal bat virus studies, we observed high spatiotemporal variation. This suggests spatiotemporal sampling designs are essential to understand how zoonotic viruses are maintained and spread within and across wild bat populations, which in turn could help predict and preempt risks of viral spillover.

Introduction

Risks of pathogen spillover vary across time and space [1,2], in part because pathogen shedding from reservoir hosts is a dynamic spatiotemporal processes [3,4]. Metapopulation dynamics characterize many reservoir hosts [5], where connectivity among populations can determine the spatiotemporal distribution of a pathogen [6,7] and the degree of spatial synchrony (i.e., correlated fluctuations in time) structuring infection dynamics [8]. For example, panmixia of straw-colored fruit bats (*Eidolon helvum*) across Africa likely facilitates the widespread circulation of Lagos bat virus and henipaviruses [9]. Temporal pulses in infection prevalence driven by seasonality in birth, movement, and climate are also common across reservoir hosts [10,11]. Understanding how infection prevalence or intensity in reservoir hosts varies over space and time is thus a critical need for predicting and managing emerging infectious disease risks.

However, surveillance strategies for reservoir hosts often do not sample this underlying spatiotemporal process, as spatially and temporally explicit sampling designs present logistical challenges when studying mobile and gregarious species [3,12,13]. For many such hosts (e.g., wild birds and bats), surveillance is often opportunistic (e.g., outbreak responses) or relies on convenience sampling [14]. These non-probabilistic samples and often singular sampling events cannot characterize spatial and temporal fluctuations in infection; times or locations of high infection prevalence can be over- or under-represented, and lack of probabilistic sampling can result in non-randomly missing data [3,15]. These challenges to inference cannot be simply fixed with statistical modeling and can accordingly limit and bias estimates of population prevalence and important epidemiological parameters such as the basic reproductive number [14,16].

Given a fixed cost, difficult decisions must be made about how to allocate sampling efforts. Sampling over space facilitates detecting geographic clusters of disease and predictive risk mapping [17,18], while sampling over time can identify periods of intensive pathogen shedding and enable epidemiological inference about the dominant transmission routes within a reservoir host population [19,20]. Researchers often treat this as a tradeoff between sampling intensively over either time or space, rather than allocating effort to both [21]. The trend to sample populations at either one point in space and time or for trading off between spatial or temporal resolution likely reflects broader sampling limitations within ecology [22,23]. Yet implicit here is that the temporal component is constant over space or that the spatial component is constant over time, and such sampling designs result in no data to assess this assumption.

We here quantified the temporal and spatial data limitations for two taxa of high-profile zoonotic viruses of bats: the family *Filoviridae* and genus *Henipavirus*. Bats have been widely studied as reservoirs for zoonotic pathogens and host more zoonotic viruses than other mammals [24,25]. Bat species such as *Pteropus alecto* and *Rousettus aegyptacius* have been confirmed as reservoirs for several henipaviruses (i.e., Hendra virus and Nipah virus) and Marburg virus, respectively [26,27], with bats implicated as reservoir hosts for other viruses within these taxa [28–32]. Many filoviruses and henipaviruses are shed from bats into the environment [33,34], and some can cause fatal disease in humans by environmental exposure (e.g., Marburg and Nipah viruses) or from contact with intermediate hosts such as horses or pigs (e.g., Hendra and Nipah viruses) [35–38]. Current evidence suggests that many filoviruses and henipaviruses show variable dynamics in space and time, including pulses of excretion from bats [6,30,35,39,40], which implies that spatiotemporal sampling is critical to capture viral dynamics in bat reservoirs. Yet while past efforts have focused on bat virus discovery [41], determinants of reservoir host status [42], and experimental mechanisms of viral transmission [33], spatiotemporal studies of

bat–virus dynamics are rare [43]. This limits understanding how zoonotic viruses are maintained and spread within and across bat populations and impairs improving future sampling designs. We here collated data on prevalence and seroprevalence in wild bats to (*i*) quantify the frequency of temporal reporting and (*ii*) assess the geographic scope of sampling. We next used phylogenetic meta-analysis to (*iii*) characterize temporal and spatial variation in virus detection estimates.

Methods

To systematically identify studies quantifying the proportion of wild bats positive for filoviruses and henipaviruses using PCR (RNA-based detection) or serology (antibody-based detection), we searched Web of Science, CAB Abstracts, and PubMed with the following string: (bat* OR Chiroptera*) AND (filovirus OR henipavirus OR 271 "Hendra virus" OR "Nipah virus" OR "Ebola virus" OR "Marburg virus" OR ebolavirus OR marburgvirus); we supplemented these searches by extracting data from references cited in identified studies (see Figure S1). Our dataset included 824 records from 56 studies (see Appendix). Viruses included not only Hendra virus, Nipah virus, Ebola virus, and Marburg virus but also Lloviu virus and Reston virus. We grouped viruses by taxa given our sample sizes and issues of serological cross-reactivity [44,45].

From each study, we defined sampling subunits: a sampling event of one bat species in one location per viral outcome. We classified each subunit into one of three sampling designs: pooled events over time, one sampling event, or multiple events. Records of a single prevalence or seroprevalence estimate from a bat population sampled over a period longer than one month were classified as pooled events, while records of virus estimates from a period less than or equal to one month were classified as single sampling events. Records of a given bat population over multiple monthly timepoints were classified as representing multiple events (i.e., longitudinal). For example, every monthly prevalence estimate per population of *Pteropus lylei* in Thailand would represent a unique sampling subunit, with the sampling design being classified as multiple events [46]. A conceptual schematic of these three sampling and reporting designs is provided in Figure 1A. One month was selected given that this timeframe was the lowest common temporal unit across studies and because bat shedding of these viruses can occur within a month [27,33]. Sampling design data were reported for most records (792/824 subunits; six publications did not always report temporal dimensions of their viral detection estimates). For each sampling subunit, we also recorded bat species (or only genus if available), virus taxon, virus detection outcome (prevalence or seroprevalence), sample size, the proportion of positive bats, sampling location, and country (recoded to the United Nations geoscheme); where possible, we also included data or derived viral detection estimates from online supplemental materials from each publication.

To assess how sampling and reporting practices relate to virus detection estimates, we used a phylogenetic meta-analysis to account for bat phylogeny, variable sampling effort, and the hierarchical nature of our dataset (i.e., subunits nested within studies). We first used the *metafor* package in R to calculate logit-transformed proportions and sampling variances [47]. We next used the rma.mv() function to fit a mixed-effects model with an interaction between sampling design, virus detection outcome (prevalence or seroprevalence), and virus taxa [48]. We included bat phylogeny (derived from the Open Tree of Life using the *rotl* and *ape* packages [49–51]) and subunit nested within study as random effects, and we used the estimated variance components to derive R^2 [52,53]. As our models account for bat phylogeny, we excluded subunits that pooled data across genera (n=52). Because some studies pooled data in a genus, we randomly selected one species from the genus to retain these samples. This dataset included 740 subunits from 48

studies and 196 species (Figure S2). To test if detection estimates varied over space and time, we fit a model with identical random effects to data from studies with multiple events (n=150). We fit an intercept-only model to quantify the contribution of true heterogeneity to total variance (I^2) and then included location as a fixed effect to test if viral data varied in longitudinal studies [54].

Results

Only 21% of bat virus studies reported data longitudinally (5 for filoviruses, 7 for henipaviruses). Eight studies reported sampling wild bat populations 2–3 times while seven reported sampling bat populations over four times (Table 1). Half the studies (n=28) instead reported estimates across multiple timepoints as pooled proportions, where the number of days per pooled estimate ranged from 31–2191 ($\bar{x}$=603, SD=456). Bat sampling also showed geographic biases. Whereas filovirus data were available across much of Africa and Asia, no studies were from Latin America and Oceania (Figure 1). Although PCR and serology have been conducted per country for most regions, both diagnostics have not been used together in Europe and Western Africa (Table 1). Henipavirus sampling was more broadly distributed but was limited in Eastern Asia, Eastern and Middle Africa, and Europe (Figure 1), where henipavirus studies have not used both PCR and serology (Table 1). Geography was also associated with bat sampling design (χ^2=172.9, p=0.001). Longitudinal data were only reported from Central, Middle, and Eastern Africa for filoviruses and only reorted from Southeastern Asia and Oceania for henipaviruses (Table 1).

Our phylogenetic meta-analysis showed that viral detection estimates were associated with sampling design and reporting, although the effect depended on outcome and virus taxa (three-way interaction: Q_7=21.12, p=0.004, R^2=0.12). A post-hoc analysis with models fit to each virus–outcome dataset showed that sampling design was associated with filovirus seroprevalence (Q_2=11.53, p=0.003; Figure 2), with pooled detection estimates having the lowest proportions, likely by increasing zeros in the numerator. Sampling design had weak effects on henipavirus seroprevalence and no effect on prevalence for either viral taxon (Table S1). We also found high variation between and within longitudinal studies (Q_{149}=1606, p<0.0001, I^2=92%; Figure 2). Study contributed most (53.8%) to residual variance, suggesting high between-region variation. Yet subunit location was also predictive (Q_{20}=89, p<0.001), and a likelihood ratio test supported its inclusion over the intercept-only model (χ^2=57.9, p<0.001). This verifies high spatiotemporal variation in viral detection estimates, highlighting the need for spatiotemporal sampling designs.

Discussion

Our study provides a systematic synthesis of prevalence and seroprevalence for bat filoviruses and henipaviruses. Viral detection estimates varied significantly within and between longitudinal studies, indicating that spatiotemporal sampling is essential to make inferences about bat virus spillover, especially if a natural reservoir host species has already been identified. Yet few studies used spatiotemporal designs; only one in every five studies reported longitundinal data. Sampling design and reporting were also associated with some viral detection estimates. We therefore implore researchers to publish bat viral data at the lowest spatial and temporal resolution associated with sampling and to provide raw data at such resolutions when possible.

Geographic limitations were also evident for overall sampling effort and where longitudinal studies have been performed. This was especially evident for filoviruses; although the lack of studies in Latin America and Oceania likely reflect the absence of reported human

cases, bat reservoir hosts are predicted to occur in both regions [42]. Most studies also used either PCR or serology, although using both data streams may improve statistical inference about how zoonotic pathogens persist in reservoir host populations [19]. Rigorous case studies using explicitly spatiotemporal sampling in such understudied regions will be critical to improve understanding viral shedding dynamics in bats and how spillover risk varies over time and space.

Data availability

Data will be deposited in Dryad upon acceptance.

Author contributions

DJB and DEC designed the study, DEC collected data, DJB analyzed data, and all authors contributed to writing the manuscript.

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187 **References**

188 1. Plowright RK, Parrish CR, McCallum H, Hudson PJ, Ko AI, Graham AL, Lloyd-Smith JO.
189 2017 Pathways to zoonotic spillover. *Nat. Rev. Microbiol.* **15**, 502–510.

190 2. Schmidt JP, Park AW, Kramer AM, Han BA, Alexander LW, Drake JM. 2017
191 Spatiotemporal Fluctuations and Triggers of Ebola Virus Spillover. *Emerg. Infect. Dis.* **23**,
192 415–422. (doi:10.3201/eid2303.160101)

193 3. Hoye BJ, Munster VJ, Nishiura H, Klaassen M, Fouchier RAM. 2010 Surveillance of Wild
194 Birds for Avian Influenza Virus. *Emerg. Infect. Dis.* **16**, 1827–1834.
195 (doi:10.3201/eid1612.100589)

196 4. Plowright RK *et al.* 2015 Ecological dynamics of emerging bat virus spillover. *Proc R Soc B*
197 **282**, 20142124. (doi:10.1098/rspb.2014.2124)

198 5. Vergara PM, Saravia-Zepeda A, Castro-Reyes N, Simonetti JA. 2016 Is metapopulation
199 patch occupancy in nature well predicted by the Levins model? *Popul. Ecol.* , 1–9.
200 (doi:10.1007/s10144-016-0550-5)

201 6. Plowright RK, Foley P, Field HE, Dobson AP, Foley JE, Eby P, Daszak P. 2011 Urban
202 habituation, ecological connectivity and epidemic dampening: the emergence of Hendra
203 virus from flying foxes (Pteropus spp.). *Proc. R. Soc. B Biol. Sci.* **278**, 3703–3712.
204 (doi:10.1098/rspb.2011.0522)

205 7. Hess G. 1996 Disease in Metapopulation Models: Implications for Conservation. *Ecology*
206 **77**, 1617–1632. (doi:10.2307/2265556)

207 8. Bjørnstad ON, Ims RA, Lambin X. 1999 Spatial population dynamics: analyzing patterns
208 and processes of population synchrony. *Trends Ecol. Evol.* **14**, 427–432.

209 9. Peel AJ *et al.* 2013 Continent-wide panmixia of an African fruit bat facilitates transmission
210 of potentially zoonotic viruses. *Nat. Commun.* **4**, 2770. (doi:10.1038/ncomms3770)

211 10. Altizer S, Dobson A, Hosseini P, Hudson P, Pascual M, Rohani P. 2006 Seasonality and the
212 dynamics of infectious diseases. *Ecol. Lett.* **9**, 467–484.

213 11. Peel AJ, Pulliam JRC, Luis AD, Plowright RK, O'Shea TJ, Hayman DTS, Wood JLN, Webb
214 CT, Restif O. 2014 The effect of seasonal birth pulses on pathogen persistence in wild
215 mammal populations. *Proc. R. Soc. Lond. B Biol. Sci.* **281**, 20132962.
216 (doi:10.1098/rspb.2013.2962)

217 12. Kuiken T, Leighton FA, Fouchier R a. M, LeDuc JW, Peiris JSM, Schudel A, Stöhr K,
218 Osterhaus ADME. 2005 Pathogen Surveillance in Animals. *Science* **309**, 1680–1681.
219 (doi:10.1126/science.1113310)

220 13. Stallknecht DE. 2007 Impediments to Wildlife Disease Surveillance, Research, and
221 Diagnostics. In *Wildlife and Emerging Zoonotic Diseases: The Biology, Circumstances and*

Consequences of Cross-Species Transmission, pp. 445–461. Springer, Berlin, Heidelberg. (doi:10.1007/978-3-540-70962-6_17)

14. Nusser SM, Clark WR, Otis DL, Huang L. 2008 Sampling Considerations for Disease Surveillance in Wildlife Populations. *J. Wildl. Manag.* **72**, 52–60.

15. Venette RC, Moon RD, Hutchison WD. 2002 Strategies and statistics of sampling for rare individuals. *Annu. Rev. Entomol.* **47**, 143–174. (doi:10.1146/annurev.ento.47.091201.145147)

16. Hayman DTS, Suu-Ire R, Breed AC, McEachern JA, Wang L, Wood JLN, Cunningham AA. 2008 Evidence of Henipavirus Infection in West African Fruit Bats. *PLOS ONE* **3**, e2739. (doi:10.1371/journal.pone.0002739)

17. Peterson AT. 2006 Ecologic Niche Modeling and Spatial Patterns of Disease Transmission. *Emerg. Infect. Dis.* **12**, 1822–1826. (doi:10.3201/eid1212.060373)

18. Wang J-F, Stein A, Gao B-B, Ge Y. 2012 A review of spatial sampling. *Spat. Stat.* **2**, 1–14. (doi:10.1016/j.spasta.2012.08.001)

19. Plowright RK, Peel AJ, Streicker DG, Gilbert AT, McCallum H, Wood J, Baker ML, Restif O. 2016 Transmission or Within-Host Dynamics Driving Pulses of Zoonotic Viruses in Reservoir–Host Populations. *PLoS Negl. Trop. Dis.* **10**, e0004796. (doi:10.1371/journal.pntd.0004796)

20. Restif O *et al.* 2012 Model-guided fieldwork: practical guidelines for multidisciplinary research on wildlife ecological and epidemiological dynamics. *Ecol. Lett.* **15**, 1083–1094.

21. Bailey LL, Hines JE, Nichols JD, MacKenzie DI. 2007 Sampling design trade-offs in occupancy studies with imperfect detection: examples and software. *Ecol. Appl.* **17**, 281–290.

22. Estes L, Elsen PR, Treuer T, Ahmed L, Caylor K, Chang J, Choi JJ, Ellis EC. 2018 The spatial and temporal domains of modern ecology. *Nat. Ecol. Evol.* , 1. (doi:10.1038/s41559-018-0524-4)

23. Marsh DM, Trenham PC. 2008 Current trends in plant and animal population monitoring. *Conserv. Biol.* **22**, 647–655.

24. Luis AD *et al.* 2013 A comparison of bats and rodents as reservoirs of zoonotic viruses: are bats special? *Proc R Soc B* **280**, 20122753. (doi:10.1098/rspb.2012.2753)

25. Olival KJ, Hosseini PR, Zambrana-Torrelio C, Ross N, Bogich TL, Daszak P. 2017 Host and viral traits predict zoonotic spillover from mammals. *Nature* **546**, 646–650. (doi:10.1038/nature22975)

26. Amman BR *et al.* 2015 Oral shedding of marburg virus in experimentally infected egyptian fruit bats (rousettus aegyptiacus). *J. Wildl. Dis.* **51**, 113–124. (doi:10.7589/2014-08-198)

27. Halpin K *et al.* 2011 Pteropid bats are confirmed as the reservoir hosts of henipaviruses: a comprehensive experimental study of virus transmission. *Am. J. Trop. Med. Hyg.* **85**, 946–951. (doi:10.4269/ajtmh.2011.10-0567)

28. Drexler JF *et al.* 2009 Henipavirus RNA in African Bats. *PLOS ONE* **4**, e6367. (doi:10.1371/journal.pone.0006367)

29. Leroy EM, Epelboin A, Mondonge V, Pourrut X, Gonzalez J-P, Muyembe-Tamfum J-J, Formenty P. 2009 Human Ebola Outbreak Resulting from Direct Exposure to Fruit Bats in Luebo, Democratic Republic of Congo, 2007. *Vector-Borne Zoonotic Dis.* **9**, 723–728. (doi:10.1089/vbz.2008.0167)

30. Brook CE *et al.* 2019 Disentangling serology to elucidate henipa- and filovirus transmission in Madagascar fruit bats. *J. Anim. Ecol.* **0**. (doi:10.1111/1365-2656.12985)

31. Leroy EM *et al.* 2005 Fruit bats as reservoirs of Ebola virus. *Nature* **438**, 575–576.

32. Pernet O *et al.* 2014 Evidence for henipavirus spillover into human populations in Africa. *Nat. Commun.* **5**, 5342.

33. Schuh AJ *et al.* 2017 Modelling filovirus maintenance in nature by experimental transmission of Marburg virus between Egyptian rousette bats. *Nat. Commun.* **8**, 14446.

34. Middleton DJ, Morrissy CJ, van der Heide BM, Russell GM, Braun MA, Westbury HA, Halpin K, Daniels PW. 2007 Experimental Nipah Virus Infection in Pteropid Bats (Pteropus poliocephalus). *J. Comp. Pathol.* **136**, 266–272. (doi:10.1016/j.jcpa.2007.03.002)

35. Amman BR *et al.* 2012 Seasonal Pulses of Marburg Virus Circulation in Juvenile Rousettus aegyptiacus Bats Coincide with Periods of Increased Risk of Human Infection. *PLOS Pathog.* **8**, e1002877. (doi:10.1371/journal.ppat.1002877)

36. McFarlane R, Becker N, Field H. 2011 Investigation of the Climatic and Environmental Context of Hendra Virus Spillover Events 1994–2010. *PLOS ONE* **6**, e28374. (doi:10.1371/journal.pone.0028374)

37. Luby SP *et al.* 2006 Foodborne Transmission of Nipah Virus, Bangladesh. *Emerg. Infect. Dis.* **12**, 1888–1894. (doi:10.3201/eid1212.060732)

38. Pulliam JRC *et al.* 2012 Agricultural intensification, priming for persistence and the emergence of Nipah virus: a lethal bat-borne zoonosis. *J. R. Soc. Interface* **9**, 89–101. (doi:10.1098/rsif.2011.0223)

39. Pourrut X, Délicat A, Rollin PE, Ksiazek TG, Gonzalez J-P, Leroy EM. 2007 Spatial and Temporal Patterns of Zaire ebolavirus Antibody Prevalence in the Possible Reservoir Bat Species. *J. Infect. Dis.* **196**, S176–S183. (doi:10.1086/520541)

40. Páez DJ, Giles J, McCallum H, Field H, Jordan D, Peel AJ, Plowright RK. 2017 Conditions affecting the timing and magnitude of Hendra virus shedding across pteropodid bat populations in Australia. *Epidemiol. Infect.* , 1–11. (doi:10.1017/S0950268817002138)

41. Drexler JF *et al.* 2012 Bats host major mammalian paramyxoviruses. *Nat. Commun.* **3**, 796. (doi:10.1038/ncomms1796)

42. Han BA, Schmidt JP, Alexander LW, Bowden SE, Hayman DT, Drake JM. 2016 Undiscovered bat hosts of filoviruses. *PLOS Negl Trop Dis* **10**, e0004815.

43. Hayman DTS, Bowen RA, Cryan PM, McCracken GF, O'Shea TJ, Peel AJ, Gilbert A, Webb CT, Wood JLN. 2013 Ecology of Zoonotic Infectious Diseases in Bats: Current Knowledge and Future Directions. *Zoonoses Public Health* **60**, 2–21. (doi:10.1111/zph.12000)

44. Peel AJ *et al.* 2013 Use of cross-reactive serological assays for detecting novel pathogens in wildlife: Assessing an appropriate cutoff for henipavirus assays in African bats. *J. Virol. Methods* **193**, 295–303. (doi:10.1016/j.jviromet.2013.06.030)

45. Gilbert AT *et al.* 2013 Deciphering serology to understand the ecology of infectious diseases in wildlife. *Ecohealth* **10**, 298–313.

46. Wacharapluesadee S, Boongird K, Wanghongsa S, Ratanasetyuth N, Supavonwong P, Saengsen D, Gongal G n., Hemachudha T. 2009 A Longitudinal Study of the Prevalence of Nipah Virus in Pteropus lylei Bats in Thailand: Evidence for Seasonal Preference in Disease Transmission. *Vector-Borne Zoonotic Dis.* **10**, 183–190. (doi:10.1089/vbz.2008.0105)

47. Viechtbauer W. 2010 Conducting meta-analyses in R with the metafor package. *J. Stat. Softw.* **36**, 1–48.

48. Konstantopoulos S. 2011 Fixed effects and variance components estimation in three-level meta-analysis. *Res. Synth. Methods* **2**, 61–76.

49. Michonneau F, Brown JW, Winter DJ. 2016 rotl : an R package to interact with the Open Tree of Life data. *Methods Ecol. Evol.* (doi:10.1111/2041-210X.12593)

50. Hinchliff CE *et al.* 2015 Synthesis of phylogeny and taxonomy into a comprehensive tree of life. *Proc. Natl. Acad. Sci.* , 201423041. (doi:10.1073/pnas.1423041112)

51. Paradis E, Claude J, Strimmer K. 2004 APE: analyses of phylogenetics and evolution in R language. *Bioinformatics* **20**, 289–290.

52. Becker DJ, Streicker DG, Altizer S. 2018 Using host species traits to understand the consequences of resource provisioning for host–parasite interactions. *J. Anim. Ecol.* **87**, 511–525. (doi:10.1111/1365-2656.12765)

53. Nakagawa S, Santos ES. 2012 Methodological issues and advances in biological meta-analysis. *Evol. Ecol.* **26**, 1253–1274.

54. Senior AM, Grueber CE, Kamiya T, Lagisz M, O'Dwyer K, Santos ESA, Nakagawa S. 2016 Heterogeneity in ecological and evolutionary meta-analyses: its magnitude and implications. *Ecology* **97**, 3293–3299. (doi:10.1002/ecy.1591)

328 **Tables**

329

330 Table 1. Summary of the temporal and spatial limitations for bat filovirus and henipavirus
331 prevalence and seroprevalence data. Some studies had multiple diagnostic methods and reporting
332 methods. Diagnostic mismatch refers to regions where either PCR or serology have been used.

		Longitudinal virus studies	Geographic sampling gaps	Diagnostic mismatch	Regions with longitudinal data
Filoviruses	PCR	2/15	Latin America, Oceania, Western Africa	Europe, Western Africa	Central Africa, Eastern Africa
	Serology	4/18	Latin America, Oceania, Europe		Central Africa, Eastern Africa, Middle Africa
Henipaviruses	PCR	3/10	Eastern Africa, Eastern Asia	Europe, Eastern Africa, Middle Africa, Eastern Asia	Southeastern Asia, Oceania
	Serology	4/27	Middle Africa, Europe		

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335 **Figures and legends**

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337 Figure 1. Characterizing studies of prevalence and seroprevalence for filoviruses and
338 henipaviruses in wild bats. Top: Conceptual schematic of how different sampling designs and
339 data reporting (colored points and lines) capture the underlying temporal patterns in viral
340 infection (black line), followed by observed proportions for field studies of bat filoviruses and
341 henipaviruses (grey shows the proportion of studies not reporting sampling designs). Bottom:
342 Countries sampled for filoviruses and henipaviruses and where wild bats have been found
343 positive (prevalence or seroprevalence greater than zero) by PCR or serology.

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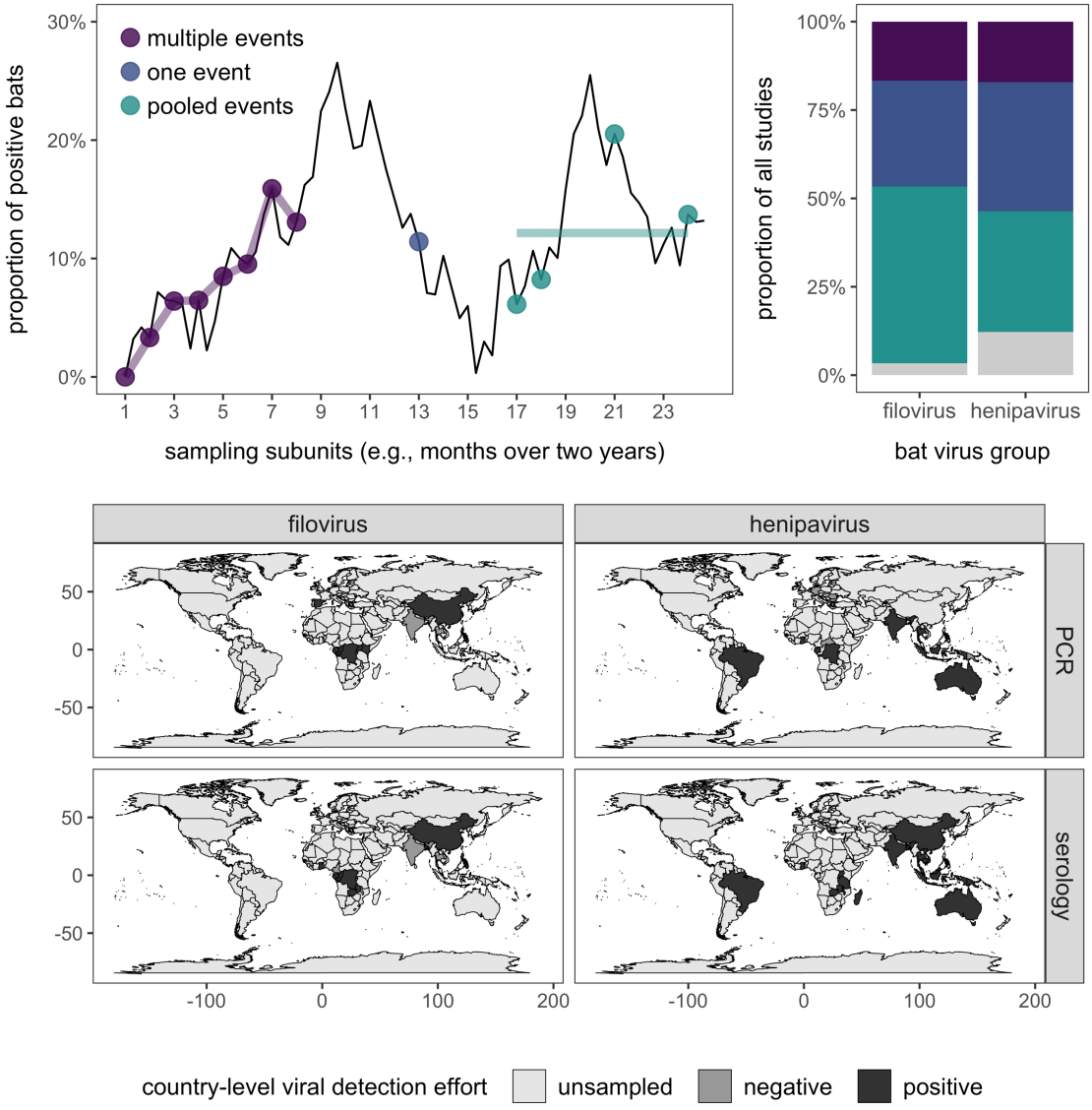

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Figure 2. Top: Influence of sampling design and reporting on virus detection estimates. Circles show proportions of positive bats per subunit and are colored by sampling design; lines and diamonds display back-transformed predicted means and 95% confidence intervals from the phylogenetic meta-analysis (limited to data reported per bat species or genus). Bottom: Spatiotemporal variation in viral detection estimates for studies that reported sampling across multiple months. Points represent each detection estimate per subunit and are shaped by sampling locations per subunit.