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A global-scale dataset of bat viral detection suggests that pregnancy reduces viral shedding

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Understanding viral infection dynamics in wildlife hosts can help forecast zoonotic pathogen spillover and human disease risk. Bats are important reservoirs of zoonotic viruses, and bat metapopulation dynamics, seasonal reproductive patterns and other life-history characteristics might explain temporal variation in the spillover of bat-associated viruses. Here, we analyse reproductive effects on viral dynamics in free-ranging bat hosts, leveraging a multi-year, global-scale viral detection dataset that spans eight viral families and 96 bat species from 14 countries. Bayesian models revealed that pregnancy had a negative effect on viral shedding across multiple data subsets, and this effect was robust to different model formulations. By contrast, lactation had a weaker influence that was inconsistent across models. These results are unusual for mammalian hosts, but given recent findings that bats may have high individual viral loads and population-level prevalence due to dampening of antiviral immunity, we propose that it would be evolutionarily advantageous for pregnancy to either not further reduce immunity or actually increase the immune response, reducing viral load, shedding and risk of fetal infection. This novel hypothesis would be valuable to test given its potential to help monitor, predict and manage viral spillover from bats.

1. Introduction

The emergence of Ebola [1,2], SARS-CoV [3], SARS-CoV-2 [4] and other zoonotic viruses has highlighted the urgent need to understand the processes that result in their spillover from animals to humans [5–9]. Because the exposure pathways that drive spillover are partially shaped by the prevalence and intensity of infection within animal hosts [6], understanding natural viral dynamics in wildlife may improve our ability to forecast and manage zoonotic disease risk. Bats are particularly important wildlife reservoirs of zoonotic viruses, including Marburg and other filoviruses, Nipah, Hendra, SARS-CoV, MERS-CoV and SARS-CoV-2 [4,10–13]. Furthermore, some bat-associated viruses, such as Nipah and Hendra, show seasonal patterns of infection in intermediate hosts and humans [13–15]. These findings imply there are predictable elements of spillover and suggest that studying viral infection and shedding in bats may be valuable in developing strategies to prevent human disease [16,17].

Seasonality of bat-associated viral outbreaks may be linked to features of bat life history and reproductive ecology, such as metapopulation structure, migration or synchronous birthing [12,13,18–20]. For example, some bat species form large maternity colonies, which increase local population density

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and intraspecific contact rates, thus increasing the potential for pathogen transmission and providing an ideal context for researchers to conduct viral sampling [18,21,22]. Viral infection in juvenile bats following the birthing period has attracted special scrutiny because the seasonal influx of susceptible juveniles could facilitate pathogen maintenance within bat populations [18,21–26]. Conversely, less attention has been given to maternal infection across reproductive states. Previous work has reported increased seropositivity [27–29] and viral detection [23,30] in pregnant and/or lactating female bats. However, such studies are often restricted to sampling a relatively small number of bat host species and typically consider only a single viral pathogen, limiting their generalizability. Our understanding of bat viral dynamics during the energetically demanding periods of pregnancy and lactation [31,32] would be enhanced by analysis of a broader range of host species and pathogens.

Here, we use Bayesian models to examine the effects of reproductive status on viral detection using a global-scale dataset from bats sampled across 14 countries. We analyse viral detection data from eight viral families across 96 bat species belonging to nine families. Leveraging this large dataset, we explicitly account for important sources of variation, such as geographic region, specimen type and different viral assays, to identify broad relationships between reproductive state and viral shedding across the order Chiroptera.

2. Methods

(a) Sample collection

The majority of viral data analysed here were from biological samples collected and tested as part of the PREDICT-1 project from 2009 to 2014 [33]. PREDICT-1 collaborated with partners in more than 20 countries throughout Asia, Africa and Latin America to conduct virus surveillance, operating with the primary goals of public health capacity strengthening and pathogen discovery [34]. Sampling focused on human–livestock–wildlife interfaces in emerging infectious disease hotspots that were thought to represent a high risk of viral spillover of public health relevance [35,36]. Field teams wearing appropriate personal protective equipment and trained in field biosafety protocols captured, sampled and released all animals under veterinary supervision, with guidance from relevant local authorities and an IACUC protocol from the University of California, Davis (protocol number 16048) [37]. Here, we only use data from sub-adult and adult female bats (electronic supplementary material, table S1, figure S1) [33].

At the time of sample collection, individual bats were identified to the lowest taxonomic specification possible and were sexed. Pregnancy and lactation status (yes/no) data were collected for female bats by gentle palpation of the abdomen and observation of milk production, respectively, following standard protocols [37,38]. Non-invasive oral swabs, faecal or rectal swabs, urine or urogenital swabs and blood samples were collected for later viral testing. Tissues (e.g. liver and spleen) were collected in rare cases when hunted bats were sampled or animals required euthanasia. As most specimens analysed in our study were swabs (oral, rectal or urogenital) or directly sampled excreta (faeces and urine) that are likely routes of viral exposure to humans and other hosts, we believe that our confirmed positive samples approximate the occurrence of bat viral shedding. For oral and rectal swabs, viral detection may not always indicate direct infection of hosts and could instead represent viral sequences derived from food items, but this scenario is likely to be rare. Samples were primarily collected into either NucliSENS Lysis Buffer (bioMérieux SA, Marcy-I'Étoile, France) or TRIzol [39,40], both of which inactivate viruses but allow for their molecular detection. Samples were frozen in liquid nitrogen in the field and safely transferred to in-country and US-based laboratories for storage at −80°C until viral testing.

(b) Viral testing

Broadly reactive consensus PCR (cPCR) was used to detect both known and novel viruses [41]. Consensus PCR assays were targeted at either viral genus or family (details given in electronic supplementary material, table S2), with multiple testing protocols sometimes employed for the same focal viral taxon (e.g. two different assays were used for coronavirus detection). All cPCR-positive samples were confirmed through sequencing and assigned a viral taxonomic designation by the PREDICT global laboratory team [40,42]. We acknowledge that the detection of viral nucleic acids through PCR does not necessarily indicate the presence of infectious virus, as could be established with alternative methods such as plaque assays [43,44]. The PREDICT project prioritized cPCR as an approach that enabled rapid, biosecure and low-cost viral detection across a range of viral families and laboratories around the world.

(c) Bayesian statistical modelling

We used Bayesian models to analyse the association between reproductive status and viral infection in female bats while controlling for multiple potential confounders that are inherent in a complex, multi-country surveillance dataset. Our outcome of interest, viral detection, was the result (positive or negative) of sequence-confirmed viral cPCR tests on field-collected bat specimens. We modelled the association between female reproductive status and viral detection using a binomial distribution with a logit link function (electronic supplementary material, figure S2).

We performed initial cleaning and filtration steps on viral testing data to ensure quality and consistency (electronic supplementary material, figure S1). For example, we used only data from wild bat samples and excluded data from animals sampled in other settings (e.g. wildlife markets) because the stress associated with captivity could alter viral dynamics. In addition, while multiple discovery approaches can generate valuable viral diversity data [41], we sought to reduce potential bias in

discovery efficacy by working solely with cPCR data, excluding results from high-throughput sequencing, real-time PCR or serology methods. We also excluded data on viral groups that never resulted in viral detection in female bats. While these data could have been incorporated into our modelling efforts, they were ultimately uninformative and would bias downwards our estimates of overall viral detection probability.

After filtration (per above), data remained heterogeneous, with samples collected across host species, time, space and other important dimensions. We account for this remaining variability in our models using varying effects. First, our primary statistical models included varying intercepts and slopes (for pregnancy and lactation effects) for each bat host species (electronic supplementary material, figure S2). This approach allowed us to explicitly account for the fact that baseline viral detection and reproductive effects on viral detection could vary across host species. Varying intercepts and slopes also help to provide generalizable inference since this model structure allows us to recover community-level estimates for the intercept, pregnancy effect and lactation effect parameters (i.e. these community-level parameter estimates represent the expected values for a typical bat species). Critically, the inclusion of varying slopes by host species prevents the reproductive effects observed in heavily sampled species from dominating inference. In essence, by fitting varying intercepts and slopes by host species, we make use of data from all bat species to gain insight on the bat community generally. We emphasize that with this model formulation, the model intercept corresponds to the expected viral detection probability in non-reproductive (i.e. non-pregnant and non-lactating) female bats.

In addition, our primary statistical models incorporated varying intercepts for the following data clusters: year of sample collection (which ranged from 2006 to 2014 because of the inclusion of archived specimens collected prior to the start of the PREDICT-1 project in 2009; see also [42]), country of sample collection, specimen type, the viral testing protocol used and the diagnostic laboratory conducting testing (electronic supplementary material, figure S2). Table 1 provides a justification for the inclusion of each varying intercepts cluster. All varying intercepts clusters were modelled using normal priors with a mean of zero, such that the varying intercept values represent deviations from the model's global mean [45]. Standard deviation parameters for these clusters were modelled using an exponential prior with a rate of 1. We implemented our varying intercepts clusters using a non-centred parameterization strategy to improve sampling efficiency and reduce bias [46]. This implementation recovers parameter estimates identical to those that could be obtained using a centred parameterization but improves the geometry of the posterior distribution for certain types of MCMC sampling problems. In sum, by incorporating these varying intercepts, we control for important sources of variation in our data (table 1), thereby making more robust general inferences about viral dynamics in female bats.

Our varying intercepts structure for viral testing deserves special attention given its importance to our modelling results. Briefly, every cPCR test we analysed is associated with both a general viral test type (i.e. the viral group that was tested for) and a specific viral testing protocol (i.e. the particular assay that was used). Using general viral test type as a control variable in our models would account for differences in detection across viral groups, but it would not account for differences in sensitivity or specificity associated with viral testing protocols. Therefore, our viral testing intercepts cluster consists of unique intercepts for each combination of general test type and specific testing protocol that was observed in the data (electronic supplementary material, table S2).

We chose to construct and fit a small suite of models that each contain the predictor variables that were considered relevant to our investigation *a priori* [47], namely the reproductive status variables. Conveniently, in a Bayesian setting, priors centred at zero have the same regularizing properties as shrinkage methods like ridge regression that are often used to control model complexity and reduce overfitting in large models [45,47,48]. This approach avoids some of the pitfalls associated with problematic ecological modelling strategies, including well-known issues with inference resulting from stepwise and all subsets model selection [48–51]. Thus, we initially fit the varying intercepts and slopes model using the full female bat viral detection dataset (i.e. the *All Viral Families* dataset; electronic supplementary material, table S1, figure S1). Next, to determine whether the effects of pregnancy and lactation on viral detection might differ depending on the viral taxa in question, we fit the varying intercepts and slopes model on data subsets representing test results from single viral families. We only fit single viral family data subsets when the subsets contained at least 200 viral tests from both pregnant and lactating bats to ensure relatively robust sample sizes across reproductive conditions. As a result, we fit additional models using data subsets for the viral families Coronaviridae and Paramyxoviridae. The Bayesian model we fit to the single viral family data subsets was identical to the model used to fit the *All Viral Families* dataset (electronic supplementary material, figure S2).

While we viewed the inclusion of varying intercepts and slopes as critical modelling components to appropriately control for variation in our data, we also constructed and fit simpler, alternative Bayesian models to verify our results were robust to model choice. First, we formulated a Bayesian model with varying intercepts but no varying slope effects (electronic supplementary material, figure S3). In this model, there are varying intercepts corresponding to each bat host species, but the pregnancy and lactation effects are fit by pooling information across all bat host species rather than by fitting species-specific pregnancy and lactation effects (electronic supplementary material, figure S3). In addition, we constructed a model without any varying effects (electronic supplementary material, figure S4). This model had only main effects of pregnancy and lactation on viral detection (electronic supplementary material, figure S4), and thus we stress that it does not control for any of the major sources of variation in our biosurveillance dataset (table 1). We fit these two alternative model types to all three datasets previously described (the *All Viral Families* dataset as well as the Coronaviridae and Paramyxoviridae data subsets).

We specified and fit all models using the Stan programming language [52] through the 'cmdstanr' package interface [53]. We set Stan's 'adapt_delta' and 'step_size' parameters to 0.99 and 0.5, respectively, to improve sampling of posteriors with difficult geometries at the expense of longer runtimes. For all model fits, we used four independent Markov chains, each with 3500 iterations. Given that 1000 iterations were used as warmup, we based our inferences on a total of 10 000 samples from each model (2500 post-warmup iterations each from four chains). We performed post hoc model diagnostics to ensure good model

Table 1. Variables included as varying intercepts clusters in Bayesian models of viral detection in female bats. Variables are listed along with the justification for their inclusion. Within each varying intercepts cluster, individual units were fit with a unique intercept. In general, this strategy can be used to model mechanistic drivers of the outcome under investigation or as a way to control for 'nuisance' variables. The numbers of distinct units within each varying intercepts cluster in the full female bat dataset are also shown. For technical details of the modelling approach, see the main text.

varying intercepts cluster	modelling relevance	number of units in cluster
year of sample collection	controls for temporal variability in viral detection	9
country of sample collection	controls for spatial variability in viral detection	14
specimen type	controls for variability in viral detection attributable to biological specimen type	7
viral test protocol	controls for variability in viral detection across viral families/genera, and controls for variability in efficacy of viral detection protocols	13
diagnostic laboratory conducting testing	controls for variability in viral detection efficacy among laboratories	15

fits. These checks included visual inspection of chains using trace plots and confirmed convergence of the $\widehat{R}$ statistic towards one for all parameters [54]. We used the 'cowplot' [55], 'dotwhisker' [56], 'ggplot2' [57], 'ggridges' [58] and 'rethinking' [45,59] packages to summarize and visualize model results. We report parameter estimates using posterior means and 95% highest posterior density intervals (HPDIs) to support model inference. In addition, where relevant, we draw attention to the proportion of a posterior's probability mass that has support for values below or above zero (corresponding to probability of support for a negative or positive influence of the predictor on the outcome, respectively).

3. Results

(a) Data summary

After data cleaning and filtering, our final female bat dataset contained 9694 cPCR test results from 1252 individuals of 96 different host species (electronic supplementary material, table S1, figures S1 and S5). These data represent 459 detections of 123 unique viral species across eight viral families: Adenoviridae, Astroviridae, Coronaviridae, Herpesviridae, Paramyxoviridae, Parvoviridae, Polyomaviridae and Rhabdoviridae.

In the female bat viral detection dataset, there were 28 bat host species for which we tested samples from both non-reproductive (not pregnant or lactating) and pregnant individuals. In 23/28 (82.1%) of those species, observed viral detection probability in samples from pregnant individuals was equal to or lower than in non-reproductive individuals (figure 1). There were 24 bat host species for which we tested samples from both non-reproductive and lactating individuals. In 21/24 (87.5%) of those species, observed viral detection probability in samples from lactating individuals was equal to or lower than in non-reproductive individuals (figure 1).

(b) Bayesian statistical modelling

While the raw data summary suggests patterns of interest across reproductive categories, it does not account for important confounders in the virus surveillance dataset. Explicitly controlling for these factors motivated our use of Bayesian models to investigate reproductive effects on viral detection in female bats. For all fit Bayesian models, the $\widehat{R}$ statistic for all parameters approached 1.0 (i.e. were <1.01), and Markov chains mixed well (electronic supplementary material, figure S6). These diagnostics indicate model convergence. Furthermore, we confirmed that the varying intercepts and slopes model fit to the full *All Viral Families* dataset was able to make accurate in-sample predictions: observed test positivity in the full dataset and for each viral family data subset fell within the 50% HPDI of model-based predictions (electronic supplementary material, figure S7).

In the varying intercepts and slopes model fit to the full *All Viral Families* dataset, the posterior mean for the community-level intercept was negative (mean [95% HPDI]: −4.00 [−5.77, −2.26]), reflecting low viral detection overall (figure 2). The 95% HPDI for the community-level pregnancy effect on viral detection spanned only negative values (−0.76 [−1.40, −0.02]; 97.4% posterior support for negative values), indicating strong support for a reduction in viral detection in pregnant female bats compared with non-gravid individuals (figure 2). The community-level lactation effect's posterior mean was also negative (−0.68), and although the 95% HPDI overlapped zero (−1.64, 0.19), 93.9% of the parameter posterior had support for negative values (figure 2). In sum, these model estimates imply that modal viral detection probability in non-reproductive bats is approximately 1%, while viral detection from pregnant and lactating individuals is roughly half that (approx. 0.4% detection probability; figure 3). Estimates of species-specific reproductive effects closely conformed to the more general community-level estimates except in the case of heavily sampled species such as *Eidolon helvum* and *Pteropus giganteus* (electronic supplementary material, figure S8).

Among the varying intercepts clusters in the varying intercepts and slopes model, the viral test protocol cluster had more variation than any other (electronic supplementary material, figure S9). This reflects the fact that the probability of viral detection varies strongly according to the viral family or genus being tested for and the protocol used (electronic supplementary material, table S2). Year and country of sample collection had more limited influence on viral test outcome, while specimen type and laboratory where analyses were conducted had the least effect of all (electronic supplementary material, figure S9). However, due to non-independent sampling across the varying intercepts cluster variables—for instance, different countries

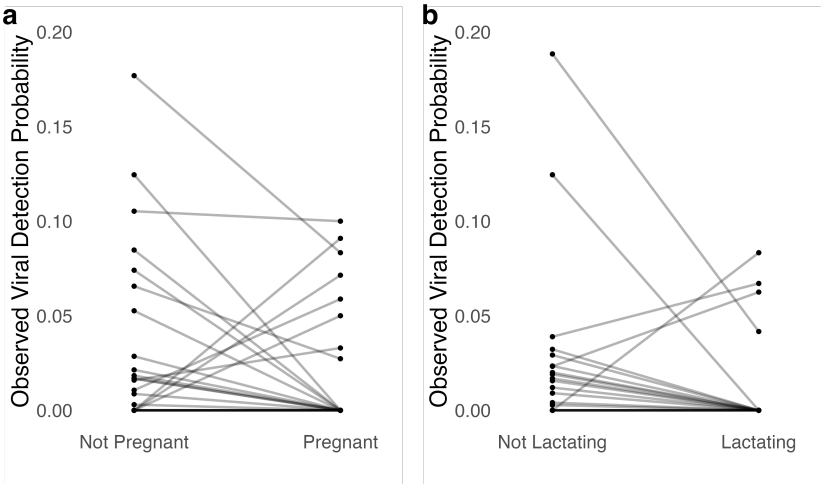

Figure 1. Observed effect of pregnancy (a) and lactation (b) on viral detection probability across bat host species. For visualization, the viral detection dataset was filtered to only include bat host species for which there were viral test data from both non-reproductive and reproductive conditions. All viral test data were pooled within host species and reproductive conditions. Lines connect data from the same host species and are displayed with transparency to emphasize areas of overlap in species trends.

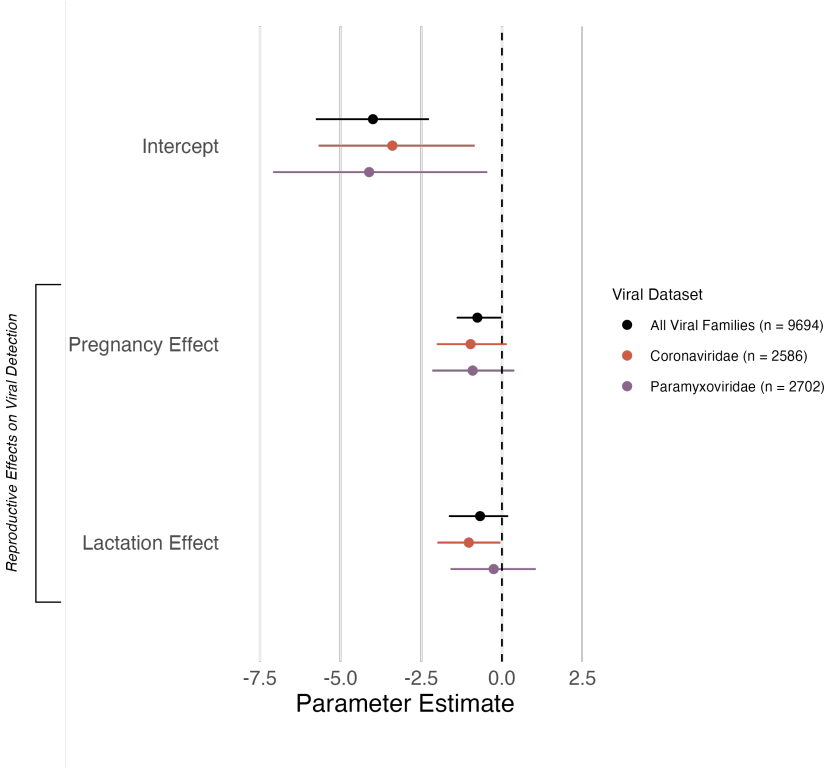

Figure 2. Dotchart showing parameter estimates from varying intercepts and slopes Bayesian models of viral detection in female bats, including community-level intercepts, pregnancy effects and lactation effects. Parameter means (dots) and 95% highest posterior density intervals are shown, and all parameter estimates are presented on the log-odds scale. The community-level intercept, pregnancy effect and lactation effect parameters are shown for the full *All Viral Families* dataset as well as the two data subsets composed of data from single viral families. Sample sizes (number of cPCR tests) are indicated in the figure legend. Detailed description of the model structure, predictor variables and model fitting is given in the main text and electronic supplementary material, figure S2.

participating in different sampling years and samples from a country only being tested in specific laboratories—the individual effects of the varying intercepts are not easily interpretable despite being appropriately controlled for.

In examining parameter estimates from varying intercepts and slopes models fit to single viral family data subsets, the community-level intercepts for the Coronaviridae and Paramyxoviridae data subsets were similar to the estimate for the *All Viral Families* dataset (figure 2). However, these estimates had inflated 95% HPDIs relative to the *All Viral Families* estimate, as would be expected given smaller sample sizes in the data subsets. In the single viral family data subsets, the community-level pregnancy effect posterior means were both negative (figure 2). Although both posteriors overlapped with zero in the 95% HPDI, they both had >90% support for negative values (figure 2). Thus, there was support from both the Coronaviridae and Paramyxoviridae data subsets for a negative effect of pregnancy on viral detection. Estimates of the community-level lactation effect from the single viral family subsets were more heterogenous. In the Paramyxoviridae model, the 95% HPDI for the lactation effect parameter substantially overlapped zero (figure 2). However, in the Coronaviridae data subset, the

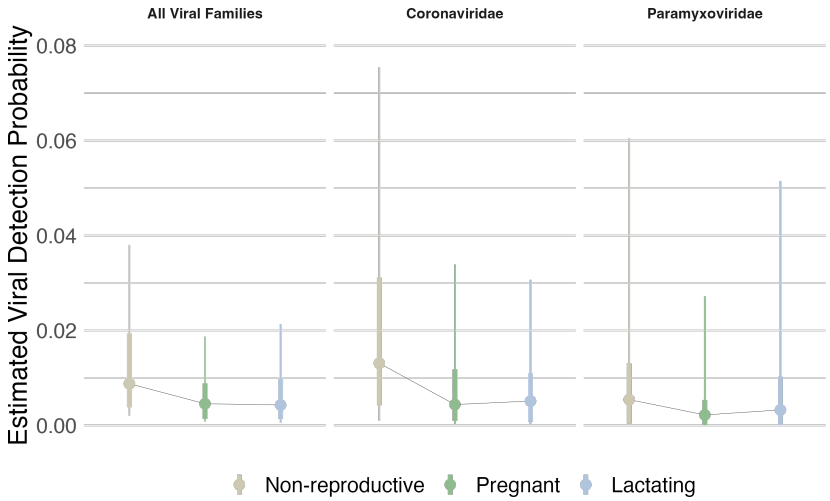

Figure 3. Model-based estimates of viral detection probability across reproductive conditions and viral datasets. Full parameter posterior distributions from varying intercepts and slopes Bayesian models were used to generate estimates of detection probability for each reproductive condition-viral dataset combination. Points represent distribution modes, thin vertical lines represent 80% highest posterior density intervals (HPDIs) and thick vertical lines represent 50% HPDIs. Thin black lines chart the changes in modal viral detection probability across the non-reproductive, pregnant and lactating conditions within each viral dataset. We elect to display modes rather than medians or means because for these highly right-skewed distributions, medians and means become unrepresentative of the location of the most concentrated probability mass. Furthermore, we show 80% HPDIs rather than wider intervals to help visualize changes in the areas of most concentrated probability mass (i.e. the 50% HPDIs).

community-level lactation effect posterior mean was negative (−1.03 [−2.00, −0.05], 98.4% posterior support for negative values). Overall, our varying intercepts and slopes models implied lower viral detection in pregnant bats relative to non-reproductive individuals while detection in lactating bats tended to be similar to or lower than non-reproductive bats, depending on the viral dataset in question (figure 3).

Fitting alternative Bayesian models to the viral datasets recovered consistent negative effects of pregnancy on viral detection, while the effects of lactation were more equivocal. When the *All Viral Families* dataset was fit using a varying intercepts Bayesian model, the 95% HPDI for the pregnancy effect only spanned negative values (−1.02 [−1.41, −0.63]), but the 95% HPDI for the lactation effect overlapped zero (−0.15 [−0.53, 0.23]; electronic supplementary material, figure S10). Use of the main effects Bayesian model and the *All Viral Families* dataset gave a broadly similar picture: in this analysis, the 95% HPDI for the pregnancy effect was strictly negative (−0.79 [−1.09, −0.50]), while the 95% HPDI for the lactation effect again overlapped zero (0.01 [−0.25, 0.27]; electronic supplementary material, figure S11).

4. Discussion

Our results highlight the important association between reproductive status and viral shedding in female bats. We consistently recovered a negative effect of pregnancy on viral detection in our global surveillance dataset after statistically accounting for other sources of variation inherent in this sampling design, including geographic and temporal heterogeneity. Lactation had a more uncertain influence on viral detection given that the strength of the lactation effect varied depending on the viral dataset and model used for analysis. These findings have implications for our general understanding of viral dynamics during reproduction in chiropteran and mammalian hosts and can help inform zoonotic pathogen surveillance efforts that seek to sample wildlife during time periods of highest pathogen prevalence.

(a) Study limitations

To foreground further interpretation of our results, we note that individual reproductive bats were necessarily captured during a broader reproductive season, when multiple aspects of bat ecology, such as movement and behaviour, may be altered in comparison to the non-reproductive season. Unfortunately, it is extremely difficult to statistically address this confounding given the need to define a reproductive season across the large suite of bat species included in our models [20,60], many of which were captured from multiple sites and in multiple countries. Furthermore, bat reproductive phenology and even the number of breeding seasons per year can vary across an individual bat species's range [61,62]. Thus, we lack the ability to cleanly separate individual reproductive status from reproductive season with our dataset. However, we highlight the fact that pregnancy and lactation are not completely confounded by reproductive season in our analyses given that non-reproductive females captured during the reproductive season are also included. Furthermore, our findings of reduced viral shedding associated with pregnancy do not align with general expectations about how bat ecology in the reproductive season (i.e. increased intraspecific contact) should impact viral dynamics (i.e. increased viral transmission and shedding) [20,21,60]. This suggests our results are recovering a signal of individual reproductive state as opposed to population-level or other concomitant ecological factors. Future work informed by multi-year, longitudinal studies across species should aim to dissect the unique contributions of individual-level reproductive status, population-level reproductive seasonality and community-level interactions to bat viral dynamics [63].

(b) Pregnancy and viral shedding

Pregnancy was the most important reproductive variable in our analyses, and model results across data subsets indicated that viral shedding is reduced in pregnant female bats. This finding contrasts with some previous studies that proposed pregnancy as a risk factor for viral infection in female bats [22,27–29,64]. However, these studies differ from ours in that they are of single bat populations or species and/or single viruses. Furthermore, inference from these studies is sometimes complicated by relatively small sample sizes and/or use of serology, which indicates prior, but not necessarily active, infection [27–29,64]. In contrast, our cPCR tests directly detect the presence of viral nucleic acid, indicative of infection and subsequent shedding. Furthermore, there are numerous reports of ambiguous associations between pregnancy and bat viral infection that also conflict with the prior studies that suggest a positive relationship between these two variables [23,24,30,65].

Parasite and pathogen dynamics during bat reproduction are often contextualized using the idea of life history trade-offs, which posit that the costs of reproduction negatively impact other aspects of organismal performance, including host defense [66,67]. Indeed, immune system depression is commonly invoked as an explanation when reproductive bats are found to have increased parasite prevalence or load [22,26,27,68]. However, the costs of reproduction vary across taxa [69], such that immunological trade-offs during reproduction in any given focal taxon may not generalize. For example, the greater mouse-eared bat demonstrated depressed T cell response in early pregnancy [68], whereas the same immunological parameter in Brazilian free-tailed bats was not affected by reproductive status [70]. Similarly, in Daubenton's bat, pregnant females had higher immunoglobulin G concentrations relative to non-reproductive individuals, while lactating females had higher hemolysis titers, suggesting no obvious trade-off between reproduction and measures of immunity [71]. These disparities are perhaps not surprising given that findings in mammalian reproductive immunology often depend upon the host species and immunological components considered, as well as study methodologies [72–74]. In sum, the linkages between reproductive status, immune system function and pathogen infection in bats are not currently well resolved. Our study, which leveraged a global biosurveillance dataset to broadly address bat–virus interactions during reproduction, helps to clarify this subfield and serves as a platform for additional research to more critically approach this topic.

In humans, pregnancy was historically thought to drive immunosuppression in the mother and, consequently, increased susceptibility to infectious disease [75–77]. This paradigm has increasingly been challenged, however, and pregnancy may be most accurately described as a period of immune system modulation, one that does not necessarily imply broad immunodeficiency [75,78]. For example, Kraus *et al*. [76] reported decreased activity of some immune cells, such as natural killer and T cells, during human pregnancy, but these shifts were accompanied by increased levels of defensin antimicrobial peptides and blood phagocytes. These findings can be reconciled with the fact that some diseases are known to be more severe during pregnancy [79,80] if pregnancy induces immune system reorganization that simultaneously strengthens barriers to initial pathogen establishment while weakening immune control of existing infections [76,77,81]. Furthermore, we now know of specific mechanisms, such as fetal-derived microRNAs, which can bolster maternal immune defenses during pregnancy [82]. This revised understanding of the immunology of pregnancy better matches evolutionary expectations, since there should be strong selective pressure for effective pathogen defense during the reproductive period as it is central to organismal fitness [75]. Collectively, these observations suggest that we should not assume that pregnancy strictly decreases immune defense and increases infection risk. Our statistical results, showing decreased viral detection in pregnant female bats, call for further integration of these perspectives into bat disease ecology in pursuit of a mechanistic understanding of wild bat immunology across reproductive states.

(c) Lactation and viral shedding

In contrast to strong pregnancy effects, we found more inconsistent evidence for an influence of lactation on bat viral detection. Although approximately 94% of posterior probability mass supported a negative value for the lactation effect when a varying intercepts and slopes Bayesian model was used to fit the full *All Viral Families* dataset, the Paramyxoviridae data subset had a lactation effect estimate very close to zero (figure 2). Furthermore, alternative Bayesian model formulations applied to the *All Viral Families* dataset did not suggest any substantial lactation effect on viral detection. Lactation is costly and can demand more energy than pregnancy [31]. Thus, one might hypothesize that physiological trade-offs may be more severe during lactation than pregnancy, necessitating lower investment in immune defenses and viral control. Indeed, lactation status has been linked to increased viral detection in some bat systems [23,30], but these results may be strongly confounded by reproductive behaviour (i.e. lactating individuals in some species are often found in dense maternity roosts). On the other hand, although lactation does incur an energetic cost for reproductively active females, it should also represent a shift back towards normal physiological and immunological status following pregnancy. Dietrich *et al*. [22] reported decreased paramyxovirus prevalence immediately following parturition in *Mormopterus francoismoutoui*, a finding they attribute to the reestablishment of typical immune function in lactating females. Furthermore, variation in the timing of sample collection within the lactation period (which differ in length across bat species) could have eroded any consistent signal of lactation on viral detection in our dataset. Regardless of the precise mechanisms at play, we find limited evidence that lactation reliably alters viral detection across the viral taxa considered, a result consistent with other recent work suggesting the likelihood of viral shedding during lactation is indistinguishable from non-reproductive periods [20].

(d) Bat reproduction, immunity and viral dynamics

Our findings point the way towards potentially fruitful avenues of investigation at the intersection of reproductive immunology and bat biology. In reproductive immunology, studies of humans and mice have begun to parse the complex immunological interactions between mother and fetus that contribute to a successful pregnancy, including fetal-derived antiviral factors that may influence maternal cells [82,83]. While there are some similarities across taxa in immunological strategies during reproduction, the mechanisms that allow for maternal tolerance of the fetus while also providing for both maternal and fetal pathogen defense throughout gestation are likely species-specific [83]. Concurrent with these developments in reproductive immunology, a recent literature has begun to describe the unique immunological characteristics of bats, from their potential evolutionary origins [84–86] to their genetic basis [87–90]. These investigations have highlighted the ways in which bat immune systems are broadly distinct from other animals. However, our results, and the reproductive immunology work in other mammals cited above, suggest that detailed study of bat immune responses in the context of reproduction is warranted.

Bats appear to have immune responses that enable a level of tolerance to viral infection that is unique among mammals [86]. Field sampling and experimental infection data suggest bat species can harbour far higher viral loads at the individual-level, and far higher viral prevalence at the population-level, than other mammals. In the small number of bat species examined thus far, specific immunological differences appear to account for these patterns. These include constitutive interferon (IFN) activation and greater combinatorial diversity in immunoglobulin genes that do not undergo substantial affinity maturation [88]. In other studies, bats were shown to have dampened STING and suppressed inflammasome pathways, both of which contribute to immune tolerance and ultimately allow higher viral loads and prevalence [86]. We propose that it would be evolutionarily advantageous for pregnancy in bats to either not lead to further reduced antiviral activity or actually increase the immune response, so as to reduce viral load, shedding and risk of fetal infection, concomitant with the findings in our study. This novel hypothesis would be valuable to test given its potential to help monitor, predict and manage viral spillover from bats. Our findings also have relevance for understanding the role that vertical transmission plays in the maintenance of important zoonotic viruses in bats (e.g. Ebola) [91]. Experimental researchers should investigate the immune system remodelling that may occur near the fetal–maternal interface in bats, with special attention to the unique mechanisms that have been previously implicated in bat immune defense [86–90]. Studies that track immune defenses and viral exposure within cohorts of wild, reproductive female bats would also be especially valuable as a means to validate the statistical relationships and mechanistic hypotheses put forward here [92,93].

5. Conclusions

Our results improve our understanding of viral dynamics in bat hosts and can be used to inform viral surveillance and zoonotic disease prevention efforts. Specifically, our findings suggest that the intense focus on sampling maternity colonies in bat infectious disease research may be less efficient during pregnancy periods than previously assumed (given reduced viral detection during pregnancy) and could bias pathogen discovery. The description of new, potentially high-consequence viruses in bats outside of maternity roosts [39] emphasizes that temporally, geographically and taxonomically broad sampling schemes will be key to comprehensive viral discovery and monitoring in bats [34]. Study designs incorporating multiple species and ecological contexts will also be necessary to completely characterize the viral diversity residing within mammalian host species, of which our knowledge remains incomplete [42,94,95]. Furthermore, careful attention to viral dynamics throughout the reproductive cycle in a wider variety of bat host species will help identify periods of increased zoonotic spillover risk to humans, since spillover is often associated with pulses of viral excretion in reservoir hosts [6,12,24,25]. Our work is congruent with recent, related studies that indicate the pup weaning period, not necessarily pregnancy or lactation periods, may be most strongly associated with viral shedding in bats [20,60]. Consideration of the interplay between reproductive biology and host defenses will deepen our understanding of virus–host dynamics and ultimately bolster our ability to prevent spillover from bats and other important wildlife hosts of viral pathogens.

Ethics. All animal work was conducted under an IACUC protocol from the University of California, Davis (protocol number 16048).

Data accessibility. Data and code are available on Zenodo [33].

Supplementary material is available online [96].

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. E.A.E.: conceptualization, data curation, formal analysis, methodology, visualization, writing—original draft, writing—review and editing; K.J.O.: conceptualization, funding acquisition, methodology, project administration, supervision, writing—review and editing; J.A.K.M.: conceptualization, funding acquisition, project administration, supervision, writing—review and editing; P.D.: conceptualization, funding acquisition, project administration, supervision, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

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References

1. Baize S *et al*. 2014 Emergence of Zaire Ebola virus disease in Guinea. *N. Engl. J. Med.* **371**, 1418–1425. (doi:10.1056/nejmoa1404505)
2. Gire SK *et al*. 2014 Genomic surveillance elucidates Ebola virus origin and transmission during the 2014 outbreak. *Science* **345**, 1369–1372. (doi:10.1126/science.1259657)
3. Peiris JSM, Yuen KY, Osterhaus ADME, Stöhr K. 2003 The severe acute respiratory syndrome. *N. Engl. J. Med.* **349**, 2431–2441. (doi:10.1056/nejmra032498)
4. Zhou P *et al*. 2020 A pneumonia outbreak associated with a new coronavirus of probable bat origin. *Nature* **579**, 270–273. (doi:10.1038/s41586-020-2012-7)
5. 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)
6. Plowright RK, Parrish CR, McCallum H, Hudson PJ, Ko AI, Graham AL, Lloyd-Smith JO. 2017 Pathways to zoonotic spillover. *Nat. Rev. Microbiol.* **15**, 502–510. (doi:10.1038/nrmicro.2017.45)
7. Holmes EC. 2022 The ecology of viral emergence. *Annu. Rev. Virol.* **9**, 173–192. (doi:10.1146/annurev-virology-100120-015057)
8. Valero-Rello A, Sanjuán R. 2022 Enveloped viruses show increased propensity to cross-species transmission and zoonosis. *Proc. Natl Acad. Sci. USA* **119**, e2215600119. (doi:10.1073/pnas.2215600119)
9. Mollentze N, Streicker DG. 2023 Predicting zoonotic potential of viruses: where are we? *Curr. Opin. Virol.* **61**, 101346. (doi:10.1016/j.coviro.2023.101346)
10. Leroy EM *et al*. 2005 Fruit bats as reservoirs of Ebola virus. *Nature* **438**, 575–576. (doi:10.1038/438575a)
11. Hu B, Ge X, Wang LF, Shi Z. 2015 Bat origin of human coronaviruses. *Virol. J.* **12**, 221. (doi:10.1186/s12985-015-0422-1)
12. Plowright RK *et al*. 2015 Ecological dynamics of emerging bat virus spillover. *Proc. R. Soc. B* **282**, 20142124. (doi:10.1098/rspb.2014.2124)
13. Letko M, Seifert SN, Olival KJ, Plowright RK, Munster VJ. 2020 Bat-borne virus diversity, spillover and emergence. *Nat. Rev. Microbiol.* **18**, 461–471. (doi:10.1038/s41579-020-0394-z)
14. Luby SP *et al*. 2009 Recurrent zoonotic transmission of Nipah virus into humans, Bangladesh, 2001-2007. *Emerg. Infect. Dis* **15**, 1229–1235. (doi:10.3201/eid1508.081237)
15. Eby P, Peel AJ, Hoegh A, Madden W, Giles JR, Hudson PJ, Plowright RK. 2023 Pathogen spillover driven by rapid changes in bat ecology. *Nature* **613**, 340–344. (doi:10.1038/s41586-022-05506-2)
16. Morse SS, Mazet JAK, Woolhouse M, Parrish CR, Carroll D, Karesh WB, Zambrana-Torrelio C, Lipkin WI, Daszak P. 2012 Prediction and prevention of the next pandemic zoonosis. *Lancet* **380**, 1956–1965. (doi:10.1016/s0140-6736(12)61684-5)
17. Epstein JH *et al*. 2020 Nipah virus dynamics in bats and implications for spillover to humans. *Proc. Natl Acad. Sci. USA* **117**, 29190–29201. (doi:10.1073/pnas.2000429117)
18. George DB, Webb CT, Farnsworth ML, O'Shea TJ, Bowen RA, Smith DL, Stanley TR, Ellison LE, Rupprecht CE. 2011 Host and viral ecology determine bat rabies seasonality and maintenance. *Proc. Natl Acad. Sci. USA* **108**, 10208–10213. (doi:10.1073/pnas.1010875108)
19. Reed Hranac C, Marshall JC, Monadjem A, Hayman DTS. 2019 Predicting Ebola virus disease risk and the role of African bat birthing. *Epidemics* **29**, 100366. (doi:10.1016/j.epidem.2019.100366)
20. Montecino-Latorre D *et al*. 2022 Seasonal shedding of coronavirus by straw-colored fruit bats at urban roosts in Africa. *PLoS One* **17**, e0274490. (doi:10.1371/journal.pone.0274490)
21. Drexler JF, Corman VM, Wegner T, Tateno AF, Zerbinati RM, Gloza-Rausch F, Seebens A, Müller MA, Drosten C. 2011 Amplification of emerging viruses in a bat colony. *Emerg. Infect. Dis.* **17**, 449–456. (doi:10.3201/eid1703.100526)
22. Dietrich M, Wilkinson DA, Benlali A, Lagadec E, Ramasindrazana B, Dellagi K, Tortosa P. 2015 *Leptospira* and paramyxovirus infection dynamics in a bat maternity enlightens pathogen maintenance in wildlife. *Environ. Microbiol.* **17**, 4280–4289. (doi:10.1111/1462-2920.12766)
23. Gloza-Rausch F *et al*. 2008 Detection and prevalence patterns of group I coronaviruses in bats, northern Germany. *Emerg. Infect. Dis.* **14**, 626–631. (doi:10.3201/eid1404.071439)
24. 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)
25. 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)
26. Hoarau AOG, Köster M, Dietrich M, Le Minter G, Joffrin L, Ramanantsalama RV, Mavingui P, Lebarbenchon C. 2023 Synchronicity of viral shedding in molossid bat maternity colonies. *Epidemiol. Infect.* **151**, e47. (doi:10.1017/S0950268823000171)
27. Plowright RK, Field HE, Smith C, Divljan A, Palmer C, Tabor G, Daszak P, Foley JE. 2008 Reproduction and nutritional stress are risk factors for Hendra virus infection in little red flying foxes (*Pteropus scapulatus*). *Proc. R. Soc. B* **275**, 861–869. (doi:10.1098/rspb.2007.1260)
28. Breed AC, Breed MF, Meers J, Field HE. 2011 Evidence of endemic Hendra virus infection in flying-foxes (*Pteropus conspicillatus*)—implications for disease risk management. *PLoS One* **6**, e28816. (doi:10.1371/journal.pone.0028816)
29. Rahman SA *et al*. 2013 Risk factors for Nipah virus infection among pteropid bats, peninsular Malaysia. *Emerg. Infect. Dis.* **19**, 51–60. (doi:10.3201/eid1901.120221)
30. Annan A *et al*. 2013 Human betacoronavirus 2c EMC/2012–related viruses in bats, Ghana and Europe. *Emerg. Infect. Dis.* **19**, 456–459. (doi:10.3201/eid1903.121503)
31. Racey PA. 1982 Ecology of bat reproduction. In *Ecology of Bats* (ed. TH Kunz), pp. 57–104. New York, NY: Plenum Press. (doi:10.1007/978-1-4613-3421-7_2)
32. Wade GN, Schneider JE. 1992 Metabolic fuels and reproduction in female mammals. *Neurosci. Biobehav. Rev.* **16**, 235–272. (doi:10.1016/s0149-7634(05)80183-6)
33. Eskew EA, Olival KJ, Mazet JAK, Daszak P. 2025 Data and code supporting analyses of reproductive effects on viral detection in bats (Version 1.0.0). Zenodo. (doi:10.5281/zenodo.15013577)
34. PREDICT Consortium. 2014 *Reducing pandemic risk, promoting global health*. One Health Institute, University of California, Davis.
35. Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, Gittleman JL, Daszak P. 2008 Global trends in emerging infectious diseases. *Nature* **451**, 990–993. (doi:10.1038/nature06536)
36. Allen T, Murray KA, Zambrana-Torrelio C, Morse SS, Rondinini C, Di Marco M, Breit N, Olival KJ, Daszak P. 2017 Global hotspots and correlates of emerging zoonotic diseases. *Nat. Commun.* **8**, 1124. (doi:10.1038/s41467-017-00923-8)
37. PREDICT Consortium. 2020 *PREDICT standard operating procedures for One Health surveillance*. One Health Institute, University of California, Davis.
38. Kunz TH, Parsons S. 2009 *Ecological and Behavioral Methods for the Study of Bats*, 2nd edn. Baltimore, MD: Johns Hopkins University Press.

39. Goldstein T *et al*. 2018 The discovery of Bombali virus adds further support for bats as hosts of ebolaviruses. *Nat. Microbiol.* **3**, 1084–1089. (doi:10.1038/s41564-018-0227-2)
40. Anthony SJ *et al*. 2017 Global patterns in coronavirus diversity. *Virus Evol.* **3**, vex012. (doi:10.1093/ve/vex012)
41. Anthony SJ *et al*. 2015 Non-random patterns in viral diversity. *Nat. Commun.* **6**, 8147. (doi:10.1038/ncomms9147)
42. Anthony SJ *et al*. 2013 A strategy to estimate unknown viral diversity in mammals. *mBio* **4**, e00598-13. (doi:10.1128/mbio.00598-13)
43. Bae HG, Nitsche A, Teichmann A, Biel SS, Niedrig M. 2003 Detection of yellow fever virus: a comparison of quantitative real-time PCR and plaque assay. *J. Virol. Methods* **110**, 185–191. (doi:10.1016/s0166-0934(03)00129-0)
44. Puhach O, Meyer B, Eckerle I. 2023 SARS-CoV-2 viral load and shedding kinetics. *Nat. Rev. Microbiol.* **21**, 147–161. (doi:10.1038/s41579-022-00822-w)
45. McElreath R. 2020 *Statistical Rethinking: A Bayesian Course with Examples in R and Stan*, 2nd edn. Boca Raton, FL: CRC Press. (doi:10.1201/9780429029608)
46. Betancourt M. 2017 *Diagnosing biased inference with divergences*. See https://mc-stan.org/users/documentation/case-studies/divergences_and_bias.html.
47. Bolker B. 2018 *Multimodel approaches are not the best way to understand multifactorial systems*. See https://github.com/bbolker/discretization/blob/master/outputs/discrete.pdf.
48. Dahlgren JP. 2010 Alternative regression methods are not considered in Murtaugh (2009) or by ecologists in general. *Ecol. Lett.* **13**, E7–E9. (doi:10.1111/j.1461-0248.2010.01460.x)
49. Whittingham MJ, Stephens PA, Bradbury RB, Freckleton RP. 2006 Why do we still use stepwise modelling in ecology and behaviour? *J. Anim. Ecol.* **75**, 1182–1189. (doi:10.1111/j.1365-2656.2006.01141.x)
50. Mundry R, Nunn CL. 2009 Stepwise model fitting and statistical inference: turning noise into signal pollution. *Am. Nat.* **173**, 119–123. (doi:10.1086/593303)
51. Fieberg J, Johnson DH. 2015 MMI: multimodel inference or models with management implications? *J. Wildl. Manag.* **79**, 708–718. (doi:10.1002/jwmg.894)
52. Carpenter B *et al*. 2017 Stan: a probabilistic programming language. *J. Stat. Softw.* **76**, 1–32. (doi:10.18637/jss.v076.i01)
53. Gabry J, Češnovar R. 2022 cmdstanr: R interface to 'CmdStan'. See https://mc-stan.org/cmdstanr/.
54. Vehtari A, Gelman A, Simpson D, Carpenter B, Bürkner PC. 2021 Rank-normalization, folding, and localization: an improved R-hat for assessing convergence of MCMC (with discussion). *Bayesian Anal.* **16**, 667–718. (doi:10.1214/20-BA1221)
55. Wilke CO. 2020 cowplot: streamlined plot theme and plot annotations for 'ggplot2'. R package version 1.1.1. See https://cran.r-project.org/web//packages//cowplot/cowplot.pdf.
56. Solt F, Hu Y. 2021 dotwhisker: dot-and-whisker plots of regression results. R package version 0.7.4. See https://cran.r-project.org/web/packages/dotwhisker/dotwhisker.pdf.
57. Wickham H. 2016 *ggplot2: Elegant Graphics for Data Analysis*. New York, NY: Springer.
58. Wilke CO. 2022 ggridges: ridgeline plots in 'ggplot2'. R package version 0.5.4. See https://cran.r-project.org/web//packages/ggridges/ggridges.pdf.
59. McElreath R. 2023 rethinking: Statistical Rethinking book package. R package version 2.31. See https://rdrr.io/github/rmcelreath/rethinking/.
60. Montecino-Latorre D *et al*. 2020 Reproduction of East-African bats may guide risk mitigation for coronavirus spillover. *One Health Outlook* **2**, 2. (doi:10.1186/s42522-019-0008-8)
61. Lučan RK *et al*. 2014 Reproductive seasonality of the Egyptian fruit bat (*Rousettus aegyptiacus*) at the northern limits of its distribution. *J. Mammal.* **95**, 1036–1042. (doi:10.1644/14-mamm-a-035)
62. Racey PA, Entwistle AC. 2000 Life-history and reproductive strategies of bats. In *Reproductive Biology of Bats* (eds EG Crichton, PH Krutzsch), pp. 363–414. San Diego, CA: Academic Press. (doi:10.1016/B978-012195670-7/50010-2)
63. Sánchez CA *et al*. 2024 Advances in understanding bat infection dynamics across biological scales. *Proc. R. Soc. B* **291**, 20232823. (doi:10.1098/rspb.2023.2823)
64. 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)
65. Wacharapluesadee S, Boongird K, Wanghongsa S, Ratanasetyuth N, Supavonwong P, Saengsen D, Gongal GN, Hemachudha T. 2010 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)
66. Sheldon BC, Verhulst S. 1996 Ecological immunology: costly parasite defences and trade-offs in evolutionary ecology. *Trends Ecol. Evol.* **11**, 317–321. (doi:10.1016/0169-5347(96)10039-2)
67. Lochmiller RL, Deerenberg C. 2000 Trade-offs in evolutionary immunology: just what is the cost of immunity? *Oikos* **88**, 87–98. (doi:10.1034/j.1600-0706.2000.880110.x)
68. Christe P, Arlettaz R, Vogel P. 2000 Variation in intensity of a parasitic mite (*Spinturnix myoti*) in relation to the reproductive cycle and immunocompetence of its bat host (*Myotis myotis*). *Ecol. Lett.* **3**, 207–212. (doi:10.1046/j.1461-0248.2000.00142.x)
69. Hamel S, Gaillard J-M, Yoccoz NG, Loison A, Bonenfant C, Descamps S. 2010 Fitness costs of reproduction depend on life speed: empirical evidence from mammalian populations. *Ecol. Lett.* **13**, 915–935. (doi:10.1111/j.1461-0248.2010.01478.x)
70. Allen LC, Turmelle AS, Mendonça MT, Navara KJ, Kunz TH, McCracken GF. 2009 Roosting ecology and variation in adaptive and innate immune system function in the Brazilian free-tailed bat (*Tadarida brasiliensis*). *J. Comp. Physiol. B* **179**, 315–323. (doi:10.1007/s00360-008-0315-3)
71. Ruoss S, Becker NI, Otto MS, Czirják GÁ, Encarnação JA. 2019 Effect of sex and reproductive status on the immunity of the temperate bat *Myotis daubentonii*. *Mamm. Biol.* **94**, 120–126. (doi:10.1016/j.mambio.2018.05.010)
72. Xu YC, Yang DB, Wang DH. 2012 No evidence for a trade-off between reproductive investment and immunity in a rodent. *PLoS One* **7**, e37182. (doi:10.1371/journal.pone.0037182)
73. Flies AS, Mansfield LS, Flies EJ, Grant CK, Holekamp KE. 2016 Socioecological predictors of immune defences in wild spotted hyenas. *Funct. Ecol.* **30**, 1549–1557. (doi:10.1111/1365-2435.12638)
74. Downs CJ, Boan BV, Lohuis TD, Stewart KM. 2018 Investigating relationships between reproduction, immune defenses, and cortisol in Dall sheep. *Front. Immunol.* **9**, 105. (doi:10.3389/fimmu.2018.00105)
75. Mor G, Cardenas I. 2010 The immune system in pregnancy: a unique complexity. *Am. J. Reprod. Immunol.* **63**, 425–433. (doi:10.1111/j.1600-0897.2010.00836.x)
76. Kraus TA *et al*. 2012 Characterizing the pregnancy immune phenotype: results of the Viral Immunity and Pregnancy (VIP) study. *J. Clin. Immunol.* **32**, 300–311. (doi:10.1007/s10875-011-9627-2)
77. Kourtis AP, Read JS, Jamieson DJ. 2014 Pregnancy and infection. *N. Engl. J. Med.* **370**, 2211–2218. (doi:10.1056/nejmra1213566)
78. Veenstra van Nieuwenhoven AL, Heineman MJ, Faas MM. 2003 The immunology of successful pregnancy. *Hum. Reprod. Update* **9**, 347–357. (doi:10.1093/humupd/dmg026)
79. Mertz D, Geraci J, Winkup J, Gessner BD, Ortiz JR, Loeb M. 2017 Pregnancy as a risk factor for severe outcomes from influenza virus infection: a systematic review and meta-analysis of observational studies. *Vaccine* **35**, 521–528. (doi:10.1016/j.vaccine.2016.12.012)
80. Rogerson SJ, Hviid L, Duffy PE, Leke RFG, Taylor DW. 2007 Malaria in pregnancy: pathogenesis and immunity. *Lancet Infect. Dis.* **7**, 105–117. (doi:10.1016/s1473-3099(07)70022-1)
81. Sappenfield E, Jamieson DJ, Kourtis AP. 2013 Pregnancy and susceptibility to infectious diseases. *Infect. Dis. Obstet. Gynecol.* **2013**, 752852. (doi:10.1155/2013/752852)
82. Delorme-Axford E *et al*. 2013 Human placental trophoblasts confer viral resistance to recipient cells. *Proc. Natl Acad. Sci. USA* **110**, 12048–12053. (doi:10.1073/pnas.1304718110)
83. Ander SE, Diamond MS, Coyne CB. 2019 Immune responses at the maternal-fetal interface. *Sci. Immunol.* **4**, eaat6114. (doi:10.1126/sciimmunol.aat6114)
84. Zhang G *et al*. 2013 Comparative analysis of bat genomes provides insight into the evolution of flight and immunity. *Science* **339**, 456–460. (doi:10.1126/science.1230835)

85. O'Shea TJ, Cryan PM, Cunningham AA, Fooks AR, Hayman DTS, Luis AD, Peel AJ, Plowright RK, Wood JLN. 2014 Bat flight and zoonotic viruses. *Emerg. Infect. Dis.* **20**, 741–745. (doi:10.3201/eid2005.130539)
86. Irving AT, Ahn M, Goh G, Anderson DE, Wang LF. 2021 Lessons from the host defences of bats, a unique viral reservoir. *Nature* **589**, 363–370. (doi:10.1038/s41586-020-03128-0)
87. Zhou P *et al*. 2016 Contraction of the type I IFN locus and unusual constitutive expression of IFN-α in bats. *Proc. Natl Acad. Sci. USA* **113**, 2696–2701. (doi:10.1073/pnas.1518240113)
88. Schountz T, Baker ML, Butler J, Munster V. 2017 Immunological control of viral infections in bats and the emergence of viruses highly pathogenic to humans. *Front. Immunol.* **8**, 1098. (doi:10.3389/fimmu.2017.01098)
89. Pavlovich SS *et al*. 2018 The Egyptian rousette genome reveals unexpected features of bat antiviral immunity. *Cell* **173**, 1098–1110.e18. (doi:10.1016/j.cell.2018.03.070)
90. Guito JC *et al*. 2021 Asymptomatic infection of Marburg virus reservoir bats is explained by a strategy of immunoprotective disease tolerance. *Curr. Biol.* **31**, 257–270.e5. (doi:10.1016/j.cub.2020.10.015)
91. Riesle-Sbarbaro SA *et al*. 2024 Selective replication and vertical transmission of Ebola virus in experimentally infected Angolan free-tailed bats. *Nat. Commun.* **15**, 925. (doi:10.1038/s41467-024-45231-0)
92. Bean AGD, Baker ML, Stewart CR, Cowled C, Deffrasnes C, Wang LF, Lowenthal JW. 2013 Studying immunity to zoonotic diseases in the natural host — keeping it real. *Nat. Rev. Immunol.* **13**, 851–861. (doi:10.1038/nri3551)
93. Mandl JN, Ahmed R, Barreiro LB, Daszak P, Epstein JH, Virgin HW, Feinberg MB. 2015 Reservoir host immune responses to emerging zoonotic viruses. *Cell* **160**, 20–35. (doi:10.1016/j.cell.2014.12.003)
94. Carroll D, Daszak P, Wolfe ND, Gao GF, Morel CM, Morzaria S, Pablos-Méndez A, Tomori O, Mazet JAK. 2018 The Global Virome Project. *Science* **359**, 872–874. (doi:10.1126/science.aap7463)
95. Carlson CJ, Zipfel CM, Garnier R, Bansal S. 2019 Global estimates of mammalian viral diversity accounting for host sharing. *Nat. Ecol. Evol.* **3**, 1070–1075. (doi:10.1038/s41559-019-0910-6)
96. Eskew EA, Olival KJ, Mazet JAK, Daszak P. 2025 Supplementary material from: A global-scale dataset of bat viral detection suggests that pregnancy reduces viral shedding. Figshare. (doi:10.6084/m9.figshare.c.7735462)