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Pathogen biology

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

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Sampling reservoir hosts over time and space is critical to detect epizootics, predict spillover and design interventions. However, because sampling is logistically difficult and expensive, researchers rarely perform spatio-temporal sampling of many reservoir hosts. Bats are reservoirs 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. To quantify the frequency of temporal sampling and to characterize the geographical 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 shows that only one in four bat virus studies report data longitudinally, that 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 sampling designs and reporting practices may affect some viral detection estimates (e.g. filovirus seroprevalence). Within the limited number of longitudinal bat virus studies, we observed high heterogeneity in viral detection estimates that in turn reflected both spatial and temporal variation. This suggests that spatio-temporal sampling designs are important 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 zoonotic viral spillover.

1. Introduction

Risks of pathogen spillover vary across time and space [1,2], in part because pathogen shedding from reservoir hosts is a dynamic spatio-temporal processes [3,4]. Metapopulation dynamics and other spatial processes characterize many reservoir hosts [5], where populations connectivity can determine the spatio-temporal distribution of a pathogen [6,7] and degree of spatial synchrony structuring infection dynamics [8]. Temporal pulses of shedding driven by seasonality in birth and climate are also common [9,10]. Understanding how infection prevalence in reservoir hosts varies over space and time is thus a critical need for predicting and managing zoonotic disease risks.

However, surveillance strategies often do not sample this underlying spatio-temporal process, as spatially and temporally explicit designs present logistical challenges when studying mobile and gregarious species [3,11,12]. For hosts such as birds and bats, surveillance is often opportunistic or relies on convenience sampling [13]. These non-probabilistic and often single sampling events cannot characterize spatial and temporal fluctuations in

infection, can over- or under-represent times or locations of high prevalence, and can result in non-randomly missing data [3,14]. These challenges cannot be fixed with statistical modelling and can bias estimates of prevalence and epidemiological parameters such as the basic reproductive number [13,15].

Given a fixed cost, difficult decisions must be made about how to allocate sampling efforts. Sampling over space facilitates detecting geographical clusters of disease and predictive mapping [16,17], while sampling over time can identify periods of intensive pathogen shedding and enable inference about dominant transmission routes [18,19]. Researchers often treat this as a trade-off between sampling over either time or space, rather than allocating effort to both [20]. 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 quantify the temporal and spatial data limitations for two taxa of high-profile viruses of bats: the family *Filoviridae* and genus *Henipavirus*. Bats have been widely studied as reservoirs for zoonotic pathogens and host more viruses with zoonotic potential than other mammals [21,22]. Some henipaviruses and filoviruses (e.g. Marburg virus) can be shed from bats into the environment [23,24] and can cause fatal disease in humans by environmental exposure or from contact with intermediate hosts such as horses, wild primates or pigs [25–30]. Many filo- and henipaviruses show variable dynamics in space and time, including shedding pulses from bats [6,25,31–33], which implies spatio-temporal sampling are likely necessary to capture viral dynamics in bats. Yet while past efforts have focused on bat virus discovery [34], determinants of reservoir status [35] and experimental mechanisms of viral transmission [36], spatio-temporal studies of the bat–virus dynamics are rare [37]. This limits understanding how viruses are maintained and spread within and across bat populations and impairs improving future sampling designs and ecological interventions [20,38].

We here systematically collated data on filo- and henipavirus prevalence and seroprevalence in wild bats to (i) quantify the frequency of temporal studies and (ii) assess the geographical scope of current research. We used phylogenetic meta-analysis to (iii) quantify how sampling designs and reporting practices may influence viral detection estimates. Single snapshots could miss pulses of viral shedding from bats, whereas pooling data over time could under- or overestimate viral presence [18,20]. Lastly, we (iv) characterized the degree of temporal and spatial variation in bat virus detection estimates.

2. Methods

To systematically identify studies quantifying the proportion of wild bats positive for filoviruses and henipaviruses using PCR or serology, we searched Web of Science, CAB Abstracts and PubMed (see electronic supplementary material, figure S1). Our dataset included 1177 records from 68 studies. Viruses included not only Hendra, Nipah, Ebola and Marburg virus but also Lloviu and Reston virus. We grouped viruses by taxa given our sample sizes and known issues of serological cross-reactivity [39,40].

From each study, we defined sampling subunits: a temporally defined sampling event of one bat species in one location per viral detection estimate. Each subunit is the lowest spatial, temporal and phylogenetic scale (of bats and their viruses) reported. We classified subunits into three sampling designs and reporting practices: one sampling event, multiple events or pooled events over time. Records of a single prevalence or seroprevalence estimate from a population sampled from a period less than or equal to one month were classified as single sampling events, whereas records of a population over multiple monthly time points were classified as spanning multiple events (i.e. a longitudinal study). For example, every monthly prevalence estimate per population of *Pteropus lylei* in Thailand would represent a unique subunit and be classified as longitudinal [41]. Records of a period longer than one month were classified as pooled events, where researchers may have sampled a population across more than one time point but reported data as a single viral detection estimate. A schematic of these categorizations is provided in figure 1*a*. One month was selected because this time frame was the lowest common temporal unit and because bat shedding of these viruses can occur within a month [36,42]. These data were reported for most records (1122/1177 subunits; three publications did not report these data and three additional publications did not always report such data for all records). For each subunit, we also recorded the bat species, virus taxon, coarse detection method (i.e. PCR or serology), number of bats sampled, proportion of bats positive, sampling time points, sampling location and country (recoded to the United Nations geoscheme for our descriptive analyses).

We quantified the proportion of studies using each sampling and reporting design, both across all data and stratified by virus taxon. To assess how the frequency of longitudinal studies (i.e. those with repeated sampling) has changed over time, we fit a generalized additive model with the *mgcv* package in R and a smooth term for publication year [43]. We also calculated the duration of repeat sampling for these longitudinal studies. For studies that pooled data over time, we quantified days represented per subunit. To describe geographical biases in bat virus studies, we assessed sampling gaps according to the region (United Nations geoscheme). We used a χ^2 test to assess if sampling designs and reporting practices were differently distributed across regions.

To assess the contribution of sampling designs and reporting practices to viral detection estimates and to quantify the degree of spatial and temporal variation in bat–virus interactions, we used the *metafor* package to calculate logit-transformed proportions and sampling variances and to fit hierarchical meta-analysis models [44,45]. To account for phylogenetic dependence, we included bat species as a random effect [46], for which the covariance structure used the phylogenetic correlation matrix; we obtained our phylogeny from the Open Tree of Life with the *rotl* and *ape* packages [47,48]. We excluded subunits that pooled data across or within bat genera ($n = 102$). As a small number of subunits ($n = 14$) pooled data across specified species in a genus, we randomly selected one species to retain these records. Our final dataset included 1075 subunits from 63 studies and 219 bat species (electronic supplementary material, figure S2). Our models also included subunit nested within the study as a random effect and weighting by sampling variances. To first assess heterogeneity among all viral detection estimates, we fit a random-effects model (REM; intercept only) and then stratified this analysis per viral taxon and detection method. We used restricted maximum likelihood to obtain unbiased estimates of the variance components, from which we derived I^2 to quantify the contribution of true heterogeneity to the total variance in viral detection estimates [49]. We used these estimates to partition variance attributed to each random effect; in the case of bat species, we derived phylogenetic heritability (H^2) as a measure of phylogenetic signal [46]. We used Cochran's Q to test if such heterogeneity was greater than expected by sampling error alone [50].

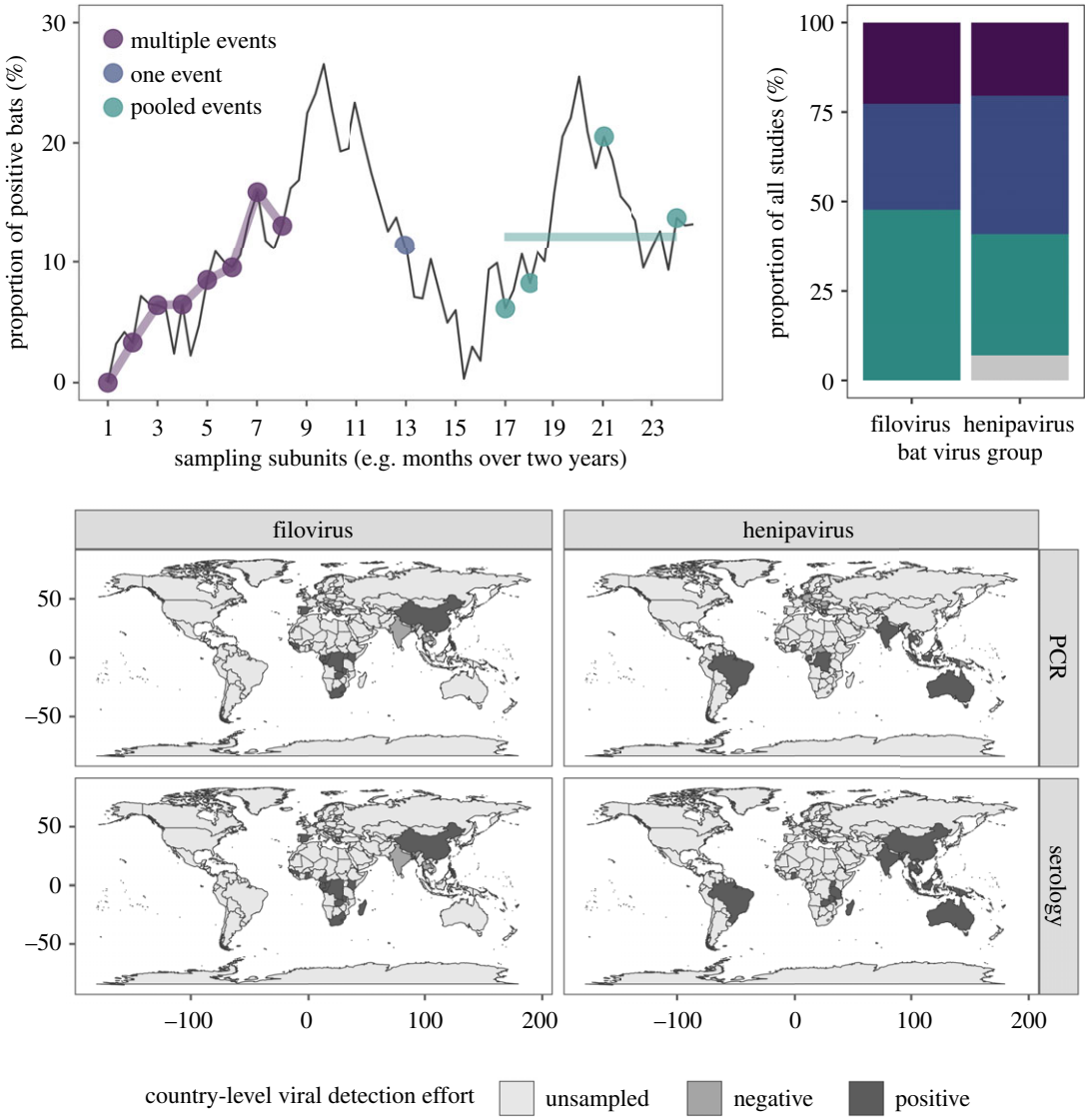

Figure 1. Top: conceptual schematic of how different sampling designs and reporting practices (coloured points and lines) capture the underlying temporal dynamics of infection (black line), followed by observed proportions for studies of bat filoviruses and henipaviruses (grey shows the proportion of studies not reporting these data). Bottom: countries sampled for bat filoviruses and henipaviruses and where wild bats have been found positive through PCR or serology. (Online version in colour.)

To next test how sampling designs and reporting practices may influence viral detection estimates ($n = 1020$), we fit a mixed-effects model (MEM) with the same random effects and an interaction between sampling design and reporting practices, detection method and virus taxon. We tested the significance of moderators using the Q test [44] and derived a pseudo-R^2 as the proportional reduction in the summed variance components compared with those of an REM [51].

To test if viral detection estimates showed spatio-temporal variation, we fit models with the same random effects to our data subset reporting multiple events ($n = 273$). We fit a REM to quantify I^2 for longitudinal studies. We then fit MEMs with location and month as univariate moderators to test if viral detection estimates varied across space and time. Because this subset of the data included many unique locations ($n = 28$) and months ($n = 12$), we did not use interaction terms and instead fit an additional set of MEMs to each viral taxon–detection method strata.

3. Results

Only 26% of bat virus studies reported data longitudinally (10 filo- and nine henipavirus studies; figure 1). However, the frequency of such studies has weakly increased over time (electronic supplementary material, figure S3, $\chi^2_1 = 2.75$, $p = 0.1$). Eleven studies reported sampling populations two to three times while 12 reported sampling populations over four times. The duration of longitudinal studies ranged from 150 days to over 10 years, on average spanning 2.5 years of repeat sampling (electronic supplementary material, figure S4). By contrast, half of our studies ($n = 35$) instead reported estimates across multiple time points as pooled proportions, which on average represented 643 days of temporally aggregated data (s.d. = 492; electronic supplementary material, figure S5).

Bat sampling showed geographical biases (figure 1 and table 1). Filovirus studies were conducted across much of Africa and Asia but not in Latin America and Oceania. PCR and serology have been used in the same region in most areas, but only one or the other have been used in Europe, Eastern and Middle Africa, and Eastern Asia for henipaviruses (table 1). Geography was also associated with sampling design and reporting ($\chi^2 = 365$, $p = 0.001$). Longitudinal data were only reported from Central, Eastern, Middle and Southern Africa for filoviruses and from South-eastern Asia, Eastern Africa and Oceania for henipaviruses (table 1).

Table 1. Summary of the temporal and spatial limitations for bat filovirus and henipavirus prevalence and seroprevalence data. Some studies had multiple diagnostic methods, sampling designs and reporting methods. Diagnostic mismatch refers to geographical regions (United Nations geoscheme) where either PCR or serology have been used (but not together).

		longitudinal virus studies	geographical sampling gaps	diagnostic mismatch	regions with longitudinal data
filoviruses	PCR	5/20	Latin America, Oceania		Central Africa, Eastern Africa
	serology	7/25			Central Africa, Eastern Africa, Middle Africa, Southern Africa
henipaviruses	PCR	4/13	Eastern Africa, Southern Africa, Eastern Asia	Europe, Eastern Africa, Middle Africa, Eastern Asia	Southeastern Asia, Oceania
	serology	5/27	Middle Africa, Southern Africa, Europe		Southeastern Asia, Oceania, Eastern Africa

We observed significant heterogeneity across viral detection estimates ($I^2 = 0.90$, $Q_{1074} = 7115$, $p < 0.001$). Bat species and study accounted for most variation ($I^2_{species} = 0.36$, $I^2_{study} = 0.35$, $H^2 = 0.40$; electronic supplementary material, table S1). We also found significant heterogeneity within each viral taxon–detection strata, although I^2 and H^2 values varied across these subsets (electronic supplementary material, table S1). Viral detection estimates for henipaviruses had much stronger phylogenetic signal than filoviruses.

Our MEM showed that viral detection estimates varied with detection method and virus taxa ($Q_1 = 7.75$, $p < 0.01$; seroprevalence was higher than prevalence, especially for henipaviruses) and that associations with sampling design and reporting weakly depended upon both virus taxa and detection method (three-way interaction: $Q_2 = 5.95$, $p = 0.05$, $R^2 = 0.02$; electronic supplementary material, table S2). A *post hoc* analysis with MEMs fit to each stratum showed sampling design and reporting were associated primarily with filovirus seroprevalence ($Q_2 = 10.84$, $p = 0.01$, $R^2 = 0.11$; figure 2), with longitudinal studies showing higher proportions of positive bats. Sampling design and reporting had no effects on henipavirus seroprevalence and weak effects on henipavirus prevalence (electronic supplementary material, table S3).

We also detected high variation in viral detection estimates across longitudinal studies ($Q_{272} = 2866$, $p < 0.0001$, $I^2 = 0.94$; figure 2). Study contributed more to residual variance than phylogeny ($I^2_{species} = 0.27$, $I^2_{study} = 0.54$, $I^2_{subunit} = 0.14$). Across these data, location did not predict viral detection estimates ($Q_{27} = 17.67$, $p = 0.91$). Yet MEMs fit to each stratum showed high spatial heterogeneity for all data strata except filovirus prevalence, with location explaining up to 76% of the variation in viral detection estimates (electronic supplementary material, table S4). Month also had little predictive power across all longitudinal data ($Q_{11} = 6.95$, $p = 0.80$), but separate MEMs revealed that time explained up to 37% of the variation in filovirus seroprevalence and henipavirus prevalence (electronic supplementary material, table S5).

4. Discussion

Our study provides a systematic synthesis of prevalence and seroprevalence for bat filoviruses and henipaviruses that can guide future sampling. Only one in four studies reported longitudinal data, although the use of such approaches is increasing. Half of the studies instead pooled data over time (and space). Geographical limitations were also evident, especially for where longitudinal studies have been conducted. This was especially evident for filoviruses; although the absence of studies in Latin America and Oceania may reflect the lack of reported human cases, bat reservoirs are predicted to occur in both regions [35]. Many studies also used either PCR or serology, although using both may improve statistical inference about how zoonotic pathogens persist in hosts [18].

We found generally weak evidence that variation in sampling design and reporting affected viral detection estimates, although filovirus seroprevalence tended to be greatest from longitudinal studies. Serological surveys of Marburg and Ebola virus have found strong temporal dynamics that may reflect seasonality in bat reproduction or food availability [31,53,54]. Detection estimates could be higher with repeated sampling, as such studies are more likely to detect shedding pulses and pooling of data could increase zeros in the numerator (underestimating seroprevalence). The lack of a similar pattern for filovirus PCR data could result from the low prevalence and be biased by zero inflation. However, our low R^2, alongside high contributions of bat phylogeny and study random effects, suggests other aspects of bat ecology (e.g. seasonal birth [31,55]) or study idiosyncrasies (e.g. assay type, lethal versus live sampling, serological cut-offs [39,40]) likely play more critical roles in shaping viral detection estimates. The high H^2 for henipaviruses in particular also suggests that cladistic or trait-based analyses of viral shedding could be useful for guiding surveillance [35,56]. However, given at least some potential for sampling design and reporting practices to affect viral detection estimates, we encourage researchers to publish data at the lowest spatial, temporal and phylogenetic scale associated with sampling and to provide data at such scales to facilitate these future analyses.

Lastly, our analysis of longitudinal studies found significant spatial and temporal variation in some bat virus data. This implies spatio-temporal sampling is likely important to make inference about bat virus spillover. Although sampling over space and time is challenging, especially for highly mobile animals like bats, sampling can be informed by spatio-temporal variation in prevalence and seroprevalence and analyses of spatio-temporal autocorrelation [20,57].

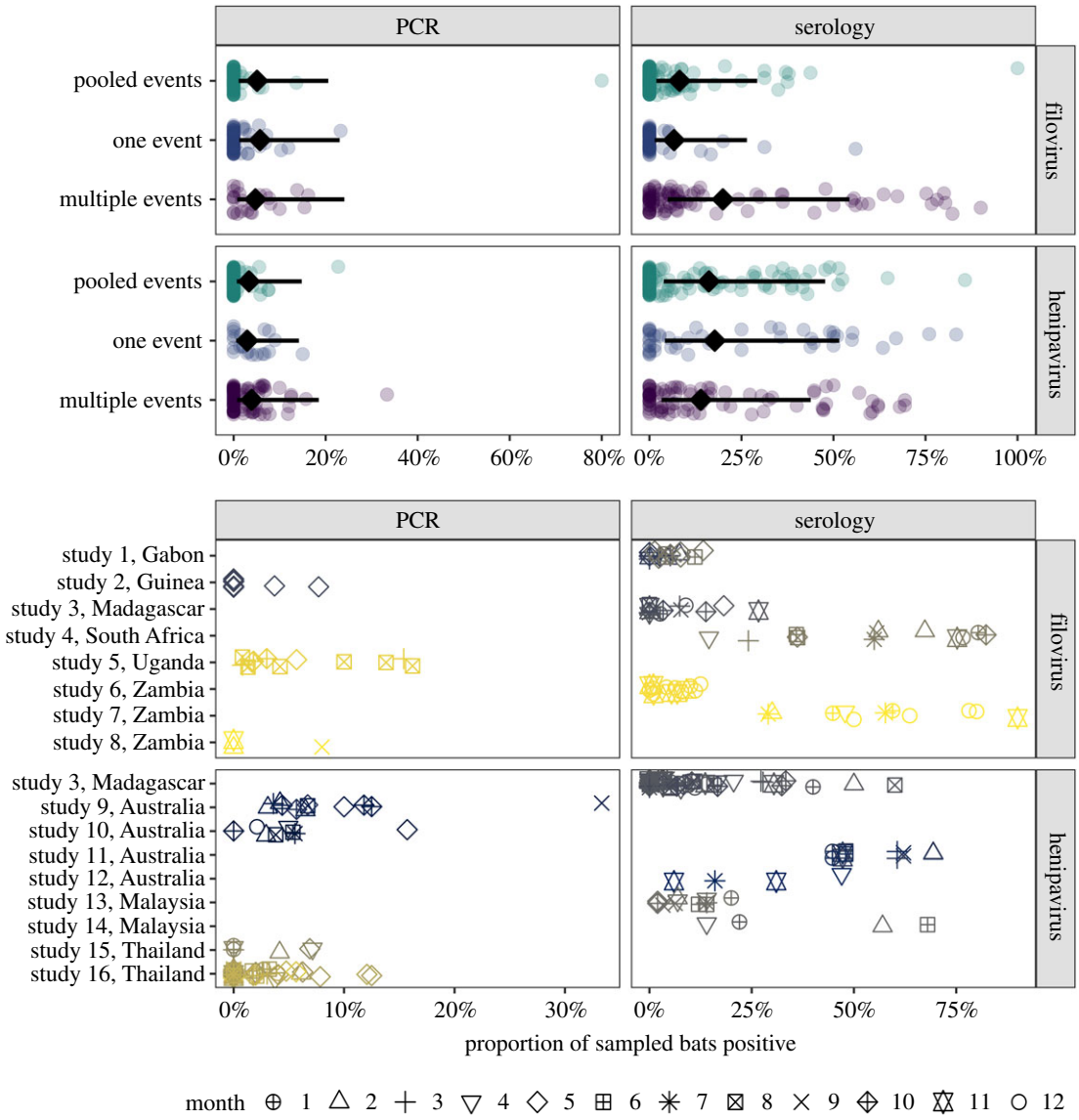

Figure 2. Top: influence of sampling designs and reporting practices on virus detection estimates. Points show proportions of positive bats per subunit; lines and diamonds display back-transformed predicted means and 95% confidence intervals from the MEM. Some overlap in 95% confidence intervals does not imply lack of statistical difference in mean estimates [52]. Bottom: spatio-temporal variation in viral detection estimates for longitudinal studies. Points represent subunit virus detection estimates and are coloured by locations and shaped by month. (Online version in colour.)

Greater variation over space can require more fine-scale spatial sampling, and greater variation over time can require more fine-scale temporal sampling. Spatio-temporal designs, such as stratified random sampling or rotating panels, can help capture spatial and temporal variation in virus shedding while also addressing some logistical challenges [13,58,59]. The increased use of such approaches, especially in the understudied regions identified from our analysis, will help improve understanding bat virus dynamics and how spillover risk varies over time and space.

Data accessibility. Data are available from the Dryad Digital Repository: https://dx.doi.org/10.5061/dryad.kkwh70s18 [60].

Authors' contributions. D.J.B., D.E.C., A.D.W. and R.K.P. designed the study, D.E.C. collected data and D.J.B. analysed data. All authors contributed to writing the manuscript. All authors agree to be held accountable for the content therein and approve the final version of the manuscript.

Competing interests. We declare we have no competing interests.

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