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Determining dengue infection risk in the Colombo district of Sri Lanka by inferencing the genetic parameters of *Aedes* mosquitoes

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Abstract

Background For decades, dengue has posed a significant threat as a viral infectious disease, affecting numerous human lives globally, particularly in tropical regions, yet no cure has been discovered. The genetic trait of vector competence in *Aedes* mosquitoes, which facilitates dengue transmission, is difficult to measure and highly sensitive to environmental changes.

Methods In this study we attempt, for the first time in a non-laboratory setting, to quantify the vector competence of *Aedes* mosquitoes assuming its homogeneity across both species; *aegypti* and *albopictus* and across the four Dengue serotypes. Estimating vector competence in relation to varying rainfall patterns was focused in this study to showcase the changes in this vector trait with respect to environmental variables. We quantify it using an existing mathematical model originally developed for malaria in a Bayesian inferencing setup. We conducted this study in the Colombo district of Sri Lanka where the highest number of human populations are threatened with dengue. Colombo district experiences continuous favorable temperature and humidity levels throughout the year creating ideal conditions for *Aedes* mosquitoes to thrive and transmit the Dengue disease. Therefore we only used the highly variable and seasonal rainfall as the primary environmental variable as it significantly influences the number of breeding sites and thereby impacting the population dynamics of *Aedes*.

Results Our research successfully deduced vector competence values for the four identified seasons based on Monsoon rainfalls experienced in Colombo within a year. We used dengue data from 2009 - 2022 to infer the estimates. These estimated values have been corroborated through experimental studies documented in the literature, thereby validating the malaria model to estimate vector competence for dengue disease.

Conclusion Our research findings conclude that environmental conditions can amplify vector competence within specific seasons, categorized by their environmental attributes. Additionally, the deduced vector competence offers compelling evidence that it impacts disease transmission, irrespective of geographical location, climate, or environmental factors.

Keywords Vector competence, Vectorial capacity, Rainfall, Bayesian, Parameter estimation

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Introduction

Dengue is a viral disease transmitted by *Aedes* mosquitoes, primarily *Aedes aegypti* and secondarily *Aedes albopictus*, reported predominantly in tropical and subtropical regions [1]. The tropical regions initially vulnerable to dengue fever are expected to expand rapidly due to climate change, accelerating the spread of the disease across new geographical areas and increasing its intensity [2]. As a result, this escalation increases the uncertainty of disease risk, leading to unprecedented outbreaks. For instance, Peru witnessed its largest outbreak in history coinciding with the Yaku Cyclone, while Pakistan experienced its outbreak in 2022 coinciding with record-breaking monsoon rainfall [3]. Located within the tropical zone where temperatures favor dengue transmission, Sri Lanka has been affected with the dengue viral disease since early 1960s, confirmed through serological testing [4]. After experiencing a notable surge in dengue instances, almost three times the figures recorded in 2021 and 2022, Sri Lanka received an outbreak alert, synchronized with the intense Southwest monsoon precipitation in 2024. The likelihood of dengue risk rapidly intensifying and extending the area of high-risk is expected to escalate exponentially, driven by erratic rainfall patterns and increasing temperatures attributed to climate change [5]. This could potentially overwhelm the healthcare system in a low-middle-income country like Sri Lanka.

Understanding disease transmission is a key component in moving forward with vector control, a crucial strategy to eradicate the disease. Mathematical models of disease transmission utilizing differential equations are frequently used to study how dengue spreads by taking into account the interactions between humans (hosts) and mosquitoes (vectors). Some models broaden their focus to incorporate external factors such as environmental conditions aiming to depict the transmission dynamics with greater accuracy. However, many studies overlooked internal factors of vectors, often simplifying complex nature of mosquito biology by assuming any mosquito encountering the dengue virus will inevitably become infected if the external factors are met, and, in turn, transmit the virus to another host [6–8]. i.e. for the events of A (*Aedes*), B (infection) and C (successful bite),

$$\begin{aligned} p(A,B|C) &= p(A|B,C)p(B|C) = p(A|B,C)p(B), \\ p(\text{infected } Aedes|\text{successful bite}) &= p(\text{infection}|\text{successful bite from } Aedes)p(Aedes), \end{aligned} \tag{1}$$

with $p(\text{infection}|\text{successful bite from } Aedes)$ is 100% while assuming B is independent of C. In other words, we define event C (a successful bite) as the condition where all external factors are satisfied. Evidently, vector genetic studies have indicated not all vectors possess equal capability of transmitting the virus violating the hypothesis made in Eq. (1) [9–11]. Therefore, relying on the hypothesis of definite infection post-exposure may yield only partially accurate results within the mathematical framework.

Dengue virus (DENV) is transmitted to vectors during a viremic blood meal from an infected host, and the virus then replicates inside the vector, enabling transmission to a new host via saliva. The genetic factors within both the mosquito and the virus determine this physiological capability, known as vector competence (ν_c). ν_c is determined by a complex interplay between genetic factors within the vector, characteristics of the pathogen (virus), and environmental conditions [12–14]. Upon acquisition, the virus must overcome several internal barriers within the mosquito, which significantly reduce its population size, diversity, and complexity. These barriers, known as bottlenecks, impact the mosquito's ν_c by affecting the virus's ability to transmit through saliva [15–18]. The strength of these barriers varies among mosquito populations, leading to different susceptibilities to virus transmission [15]. In a laboratory setting, researchers have attempted to assess ν_c by infecting mosquitoes with the DENV and subsequently measuring the virus concentration in the mosquito body parts [15, 19, 20]. Despite limited mathematical analysis outside laboratory settings, this study aims to estimate ν_c in relation to external factors by employing Vectorial Capacity (V_s) as a novel approach. V_s quantifies potential secondary infections from a single infected host, similar to the basic reproduction number (R_0), but includes vector, host, and pathogen interactions [21, 22].

The advent of genome sequencing technologies has significantly advanced our understanding of transcriptomics, enabling detailed studies on messenger RNA expression across various organisms. This progression from analyzing mRNA expressions has revolutionized the field through methods like expressed sequence tags (ESTs) and quantitative RT–PCR to comprehensively analyze full transciptomes via bulk RNA-Seq [23]. A pivotal moment for ν_c research was the detailed publication of the *Aedes aegypti* transcriptome, offering high-resolution gene expression data at various developmental stages and conditions, which facilitated groundbreaking work in gene drive strategies, functional genomic studies, and the identification of genetic markers associated with ν_c [24]. Transcriptomic profiling has also shed light on genetic bases for differential susceptibility to the DENV among

Aedes aegypti strains, marking a significant advance in understanding mosquito immune responses and v_c [25]. Moreover, tissue-specific and single-cell transcriptomic analyses have deepened our knowledge of v_c, highlighting the genetic underpinnings that distinguish susceptible and refractory mosquito strains and suggesting new avenues for disease control and surveillance [26, 27]. Furthermore, metatranscriptomic profiling of mosquitoes offers a comprehensive view of their microbial ecosystems, providing valuable data for biosurveillance and understanding the genetic factors influencing v_c [28–30].

The influence of climate factors, particularly the temperature, on v_c have been experimented [14, 19]. Climate change significantly impacts mosquito genetics, with various factors driving evolutionary changes and adaptations. Furthermore, warmer temperatures and extended transmission seasons may favor genetic traits that enhance v_c and survival over longer periods [14]. Urban heat islands present unique adaptation challenges, potentially leading to genetic changes facilitating survival in urban environments [12]. Lastly, the increasing use of insecticides in response to changing mosquito distributions encourages the spread of resistance genes, showcasing a direct genetic impact of climate change [31]. Together, these factors underscore the complex interplay between climate change and mosquito genetics, highlighting the need for integrated approaches to vector control and disease prevention in a changing world. Studies show that V_s varies with environmental factors, such as temperature and vector genetics, and is influenced by climate change [32, 33]. Its variability depends on specific vector species and viral strains [34–36]. Studies have used metrics like Infection Rate (IR), Dissemination Efficiency (DE), and Transmission Efficiency (TE) to assess v_c, typically ranging from 0 to 1 [11, 15, 17].

All in all, we present in this paper on inferencing a genetic parameter of *Aedes* in order to gain insights on disease transmission. The rest of the paper is organized as follows. Model setup for parameter estimation section presents detailed discussion on the model and parameter estimation setup. Results are given in Results section followed by detailed discussion in Discussion section. Finally the paper concludes with Conclusions section stating future directions.

Model setup for parameter estimation

The literature predominantly presents two linear equations for V_s, of which, one is based on the vector's daily survival rate, while the other is based on vector mortality rate [22, 34]. Due to the unavailability of accurate vector survival rate data, we used the equation with the vector mortality rate,

$$V_s(t) = z(t)b^2 v_c \left(\frac{e^{-\mu_v EIP}}{\mu_v} \right). \tag{2}$$

The descriptions of parameters in Eq. (2) are given in Table 1. In this study, we aim to quantify for the first time a quantitative measure for the genetic factor of *Aedes* mosquito, v_c. The study is carried out based on Colombo district's Colombo Municipal Council (CMC) area and we assume CMC area is representable of Colombo district. The Colombo district, with its highest population density, standing at the greatest risk, especially as it is heavily affected by the Southwest monsoon. As highlighted in the introduction, climate change impact on monsoon specifically can overwhelm the district with the disease. Understanding the risk in this area can help greatly to reduce the burden on limited healthcare system of Sri Lanka. In the study area, we assumed homogeneity of v_c in *Aedes aegypti* and *Aedes albopictus*. Additionally, the results in this study majorly account for DENV–2 and DENV–3 serotypes as they are the most prevalent in Sri Lanka [37]. We verify the applicability of V_s formula in (2) for the dengue cases observed in Colombo. V_s, quantifies the risk of getting secondary humans infected by a single infected human. Therefore, the infected human population density, $I(t + \underline{h})$ can be considered proportionate to $V_s(t)$,

$$I(t + \underline{h}) \propto V_s(t), \tag{3}$$

where $I(t + \underline{h})$ is the infected human density at time $(t + \underline{h})$ with intrinsic incubation period $\underline{h}$. Since V_s is defined for a unit time, we consider the unit time as a week in our estimation procedure. Since the estimation of v_c is performed based on infected human population densities generated from successfully infected *Aedes*, the v_c estimate gives an account of the TE [15]. Moreover, we demonstrate here how seasonal variations in environmental factors can modulate v_c, leading to differing outcomes across seasons, warranting further investigations. In the Colombo district, environmental variables such as temperature and humidity consistently stay within

Table 1 Description of parameters & variables in Eq. (2)

Parameter	Description
z	Per-capita vector density
b	Vector biting rate
μ_v	Vector mortality rate
EIP	Extrinsic incubation period
v_c	Vector competence
V_s	Vectorial capacity
R	Rainfall

ranges favorable for dengue transmission throughout the year [38]. Furthermore, studies have shown immediate temperature variations do not significantly impact dengue cases [39]. For these reasons and rainfall being the only significant variable impacting seasonality in Colombo district, we have chosen rainfall as the primary factor influencing dengue transmission in Colombo, as it directly affects mosquito breeding habitats and the subsequent spread of the virus. Additionally, both commercial and administrative cities in Sri Lanka are located in the Colombo district, resulting in excessive human mobility. Consequently, Colombo is a significant hotspot for disease transmission. The dengue infected population density in Colombo shows a consistent pattern aligning with monsoons and therefore we carried out this study with respect to four such identified seasons [40]. To that end we have the following relationship to find seasonal per-capita vector density for four identified seasons,

$$z(t) = a_s R(t-\tau), \tag{4}$$

where a_s is the rainfall coefficient for season s and time lag denoted by τ. By combining the Eqs. (3) and (4), we can write the linear relation of infected dengue density and ν_c,

$$I(t+\underline{h}) = K a_s R(t-\tau) b^2 \nu_c \left(\frac{e^{-\mu_\nu EIP}}{\mu_\nu} \right). \tag{5}$$

In Eq. (5), the two parameters ν_c and K are not known. As the relationship between these two parameters is in a product form, accurately estimating unique values for ν_c becomes challenging when attempting to estimate both simultaneously. Therefore we first estimated lower bounds K for each season while fixing ν_c at its maximum value (i.e. $\nu_c = 1$). Subsequently, the accurate value of ν_c is estimated by setting K at its lower bound, thereby enabling the determination of a suitable estimate for ν_c up to 1.

Data of the study

All the identified variables and data of Eq. (5) are given in Table 2. Parameters common to *Aedes* vector and the dengue disease were extracted from the literature (see Table 2). We further assume there is no significant change observed in ν_c during the span of a single season i.e. ν_c is not a time dependent parameter within a season. For the parameter estimation we used annual average dengue data across the years from 2009 to 2022. We obtained the data from The National Dengue Control Unit (NDCU), Ministry of Health, Sri Lanka, with their permission to conduct the research, accompanied by a signed privacy policy, allowing us to use it for analysis and publication [41]. During this period, Colombo district experienced two outbreaks in 2017 and 2019. Outbreak years driven by unusually heavy rainfall that exceeds normal levels, can significantly alter the usual seasonal dengue incidence pattern. To avoid distorting this pattern, our study focuses on estimating ν_c under standard environmental conditions, excluding outbreak years. The rainfall coefficient of the four seasons were recalculated from [40] by omitting the outbreak years as well and are presented in Table 3. The rainfall data were obtained from the NASA power data access viewer [42]. The infected population density is calculated with respect to the total population in the Colombo district [43].

Table 2 The status of parameters and variables in the model (5). The definitions of the acronyms are given in Table 1

Description	Variables
Unknown parameter	ν_c
Known data (Observed)	I, R, K
Known data ([40, 44])	b, μ_ν, EIP, a_s

I is the reported infected human cases, K is the proportionality constant, and a_s is the rainfall coefficient for season s

Full Bayesian setup

In this section we setup the full Bayesian version for this model in Eq. (5). The Bayes' theorem yields,

$$p(\theta|y) = \frac{p(\theta)p(y|\theta)}{p(y)}, \tag{6}$$

where θ represents the vector of parameters and y denotes the vector of data/observables. The denominator in Eq. (6) is called the normalization constant and for the problem of parameter estimation, the denominator stays a constant. Therefore Eq. (6) reduces to the following after including all variables,

$$p\left(\nu_c | I(t+\underline{h}), K, R(t-\tau), \vec{P}, \sigma\right) = k_1 p(\nu_c|R) p(R) p(I(t+\underline{h})|\nu_c, K, R, \sigma), \tag{7}$$

where $\vec{P} = \{a_s, b, \mu_\nu, EIP\}$ is the vector of other available literature data and $1/k_1$ is the normalization constant. The time stamp of the variable $R(t-\tau)$ on the right hand side of Eq. (7) is ignored since time stamp is not relevant

Table 3 Seasonal rainfall coefficients in Eq. (4) calculated excluding the outbreak years of 2017 and 2019, deeming them as outlier points

Season (s)	Month	Seasonal rainfall coefficient (a_s)
1	April-August	7.495
2	August-October	1.782
3	October-January	5.596
4	January-April	2.189

to a probability distribution, i.e., prior distribution of R does not depend on a time stamp. A probability distribution was fitted to rainfall data from 2009 to 2022 to use as prior information of the rainfall distribution in the study area. The AIC suggested rainfall follows best with gamma distribution (from among normal, log normal, and gamma) and our result agrees with standard distribution for rainfall [45] (Fig. 1a). Here, $\alpha = 0.9461$ (shape parameter) and $\beta = 0.0219$ (rate parameter) in the gamma distribution,

$$p(R|\alpha,\beta) = \frac{\beta^{\alpha}}{\Gamma(\alpha)} R^{\alpha-1} \exp(-\beta R), \quad R > 0. \tag{8}$$

Based on the literature, the range for v_c in terms of TE ranges from 0 to 1 [10]. Consequently, in this study, we proposed two choices of beta distribution as prior distributions in the Bayesian setup to support both lower TE and higher TE. The choice of prior distribution was designed to encompass the range of v_c, including both low and high extremes providing a more meaningful framework to use prior knowledge. We were allowed to explore how powerful the observed data is to influence the initial prior assumptions of v_c to the final estimated values. The data reveal the extent to which these estimates are revised from their prior distributions. Although a gamma distribution can also accommodate to design a prior distribution, the range of v_c fits with the domain of a beta distribution - thus employing beta is most suitable. The two prior distributions are shown in Fig. 1b and c. Accordingly, a beta distribution with $\gamma = 1.2$ and $\delta = 10$ was chosen for a right skewed distribution (supportive prior) anticipating v_c ranges close to zero (low TE) while a left skewed distribution (unsupportive prior) with $\gamma = 10$ and $\delta = 1.2$ was chosen anticipating v_c close to 1 (high TE) (Eq. (13)),

$$p(v_c|\gamma,\delta) = \frac{\Gamma(\gamma+\delta)}{\Gamma(\gamma)\Gamma(\delta)} v_c^{\gamma-1}(1-v_c)^{\delta-1}, \quad v_c > 0. \tag{9}$$

The observed data are the infected dengue densities (dengue incidence densities) in Colombo district during 2009 – 2022 [41]. Let us assume the errors in dengue data against V_s are normally distributed, since there is no evidence to suggest otherwise. With the assumption that errors are linear, we can write the likelihood distribution for n observed data as,

$$p(I(t+\underline{h})|v_c,K,R,\sigma) = \prod_{i=1}^{n} \frac{1}{\sqrt{2\pi\sigma^2}} \exp\left(-\frac{1}{2\sigma^2}\left(I_{obs_i} - I(t_i+\underline{h})\right)^2\right) \tag{10}$$

assuming $\sigma_i = \sigma \ \forall \ i$. To avoid computational overflows, the log of the probability densities are taken.

$$\log p(I(t+\underline{h})|v_c,K,R,\sigma) = -\frac{n}{2}\log\left(2\pi\sigma^2\right) - \frac{1}{2\sigma^2}\left\|\left(I_{obs_i} - I(t_i+\underline{h})\right)\right\|^2 \tag{11}$$

By putting together the prior distributions and the likelihood distribution, we get the full log posterior

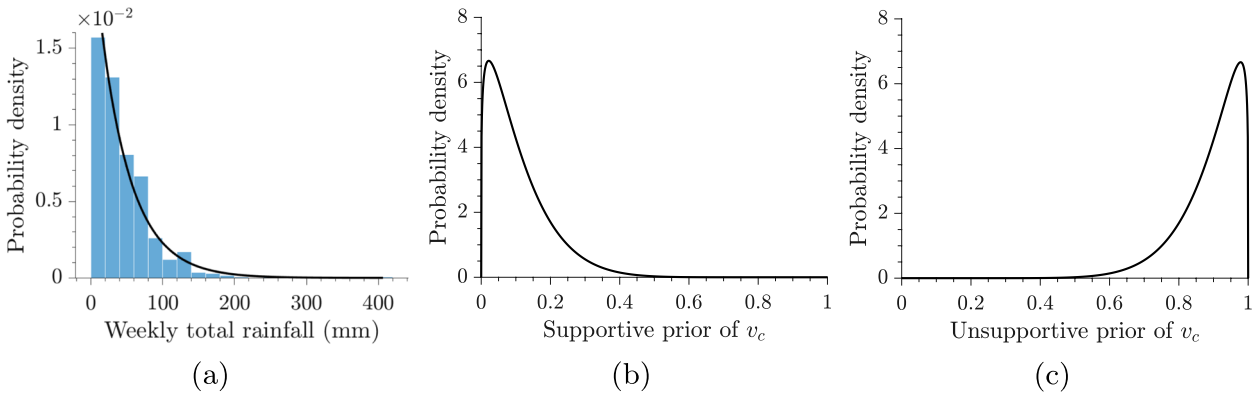

Fig. 1 Prior distributions: **a** gamma probability distribution for rainfall based on historical data in Colombo district, with $\alpha = 0.9461, \beta = 0.0219$ (histogram is blue and gamma distribution is in black) (**b**) anticipated right skewed beta distribution for v_c (supportive) enforcing small values highly probable. **c** anticipated left skewed beta distribution for v_c (unsupportive) enforcing large values highly probable

distribution (Eq. (12)). The prior distributions chosen are conjugate to the likelihood and thus no special treatment is needed in simulation step.

$$p\left(v_c|I(t+\underline{h},),K,R(t-\tau),\overrightarrow{P},\sigma\right) = k_1 p(v_c|\gamma,\delta)p(R|\alpha,\beta)p(I(t+\underline{h})|v_c,K,R,\sigma) \tag{12}$$

$$\begin{aligned}\log p\left(v_c|I(t+\underline{h},),K,R(t-\tau),\overrightarrow{P},\sigma\right) &= \log k_1 + \log\left(\frac{\Gamma(\gamma+\delta)}{\Gamma(\gamma)\Gamma(\delta)}\right) + (\gamma-1)\log v_c + (\delta-1)\log(1-v_c)\\ &+ \log\left(\frac{\beta^\alpha}{\Gamma(\alpha)}\right) + (\alpha-1)\log R - \beta R - \frac{n}{2}\log\left(2\pi\sigma^2\right) - \frac{1}{2\sigma^2}\left\|\left(I_{obs_i} - I(t_i+\underline{h})\right)\right\|^2\end{aligned} \tag{13}$$

With the built posterior distribution we estimated v_c for 4 seasons. After parameter estimation we also quantified the uncertainty of the parameters by calculating the 95% credible intervals of the simulated marginal distributions of v_c for each season. With these values of v_c the uncertainty of the dengue risk was calculated for each season and are presented in the next section.

Results

We simulated the unnormalized posterior density for each season established in Eq. (13). These simulations were carried out using MCMC toolbox of Delayed Rejection Adaptive Metropolis in MATLAB [46]. For this estimation, the per-capita vector density was extracted from a previous study in which the rainfall data were used to estimate the seasonal rainfall coefficients (a_s) [40]. These values for the four seasons are given in Table 3.

In our model, the sensitivity analysis was carried out by sequentially estimating the two parameters. In particular we allowed the value of v_c to have its upper bound in order to estimate the lowest possible K the model can handle. Afterward the actual value of v_c was estimated which was supported by the model. We further this sensitivity via prior distributions of v_c by allowing the model to locate its accurate value from the data. Although we did not use independent datasets to validate our model, we accomplished it within the parameter estimation mechanism. This procedure allowed us to find reasonable boundaries of v_c ensuring a comprehensive examination of the model thereby validating its reliability and performance.

Estimating lower bound for K

The first stage of parameter estimation aimed at determining the lower limit of K while setting the value of v_c to its upper limit. The point estimate of K was derived by conducting m simulations using 20 random initial conditions.

$$\log p\left(K|I(t+\underline{h},),v_c,R(t-\tau),\overrightarrow{P},\sigma\right) = \log k_1 + \log\left(\frac{\beta^\alpha}{\Gamma(\alpha)}\right) + (\alpha-1)\log R - \beta R - \frac{n}{2}\log\left(2\pi\sigma^2\right) - \frac{1}{2\sigma^2}\|\left(I_{obs_i} - I(t_i+\underline{h})\right)\|^2 \tag{14}$$

$$K^* = \frac{\sum_{j=1}^{m}\left(\frac{\sum_{i=1}^{w} K_{ij}}{w}\right)}{m} \tag{15}$$

where $m = 100000, w = 20$.

Estimating v_c with optimum lower bound of K

The seasonal v_c was estimated with the two beta prior distributions anticipated as mentioned in Model setup for parameter estimation section. For every season, MCMC was run 20 times starting from random initial value for v_c. Each of these simulations, MCMC was set to run 100000 sample generations, which was a sufficient amount of samples to observe the convergence of the chains. With the obtained convergent chains, 30% of samples were burnt to obtain the correct marginal distribution. The mean marginal distribution obtained from the 20 runs was then utilized to find v_c for each season. The point estimate for v_c is calculated from the mean of the marginal distribution similar to Eq. 15 (see Table 4). Additionally, the DRAM toolbox samples error variance with inverse gamma distribution based on an adaptation mechanism [46],

$$p(\sigma|r,q) = \frac{q^{-r}}{\Gamma(r)}\sigma^{(r-1)}\exp(-\sigma/q), \tag{16}$$

where $r = (1+N)/2, q = 2/(sd + \text{SSE})$ with sd, N and SSE respectively represent the standard deviation of dengue data, sample size and $\left\|\left(I_{obs_i} - I(t_i+\underline{h})\right)\right\|^2$. For the four seasons, the sampled marginal distributions are indicated in Fig. 2a–d. From these marginal distributions, 95% credible intervals were calculated and

Table 4 Estimated parameter values for proportionality constant, K and vector competence, v_c with supportive and unsupportive priors

Season	K lower bound	v^c from Supportive Prior		v^c from Unsupportive Prior	
		$E(v_c)$	Credible Interval $\left(v_c^{L^*}, v_c^{U^*}\right)$	$E(v_c)$	Credible Interval $\left(v_c^L, v_c^U\right)$
1	1.37×10^{-06}	0.7077	(0.6676,0.7455)	0.9329	(0.9091,0.9540)
2	4.22×10^{-06}	0.6850	(0.6348,0.7310)	0.9286	(0.9029,0.9511)
3	1.61×10^{-06}	0.7609	(0.7280,0.7920)	0.9404	(0.9196,0.9588)
4	2.98×10^{-06}	0.7772	(0.7455,0.8067)	0.9425	(0.9228,0.9602)

$E(.)$ represents the expected value. $v_c^{L^*}$ and $v_c^{U^*}$ represent the lower and upper bounds of the estimated vector competence for supportive beta prior. v_c^L and v_c^U represent the lower and upper bounds of the estimated vector competence for unsupportive beta prior

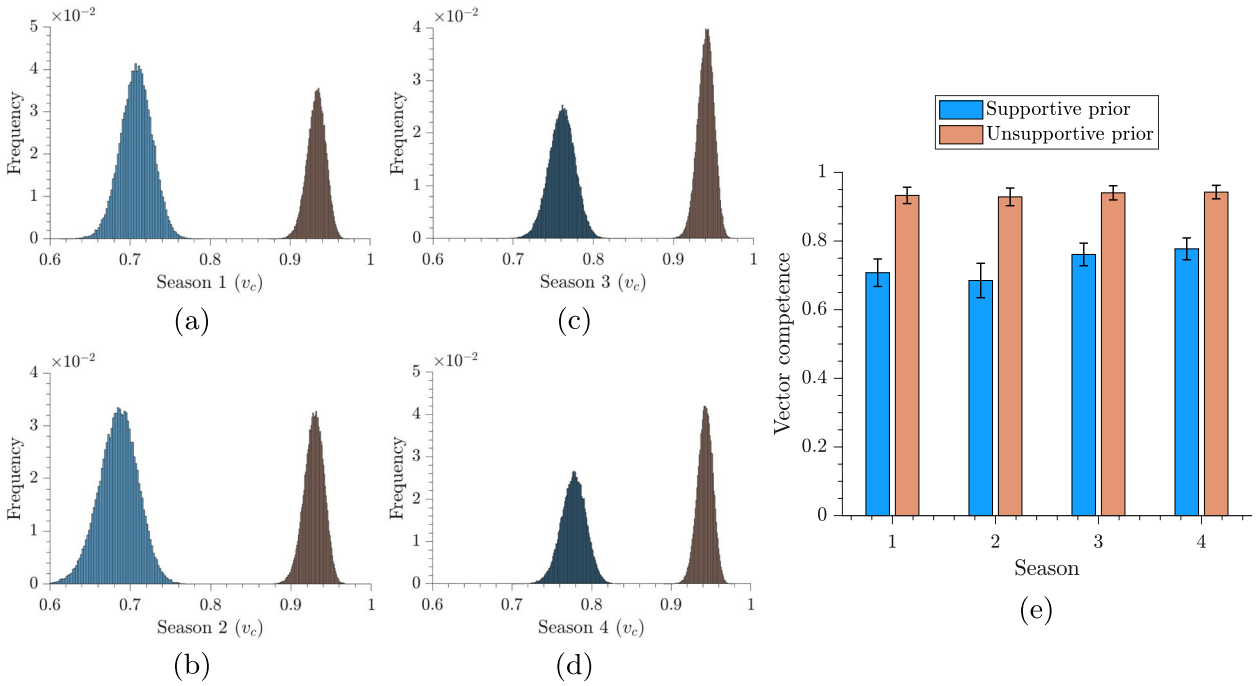

Fig. 2 The posterior distribution of vector competence against its chosen supportive and unsupportive beta prior distributions are showcased in (**a**), (**b**), (**c**), and (**d**) respectively for seasons 1, 2, 3, and 4. The estimates of vector competence (v_c) are shown in (**e**) with the error bars representing 95% credible intervals. In all the panels, results from the supportive and unsupportive priors are respectively shown in blue and orange colors

are shown in Table 4. This uncertainty in the estimates considers both the uncertainties in the model structure and the quality of the data. Note that when the supportive beta prior is used, i.e. when we suggest v_c possibly be lower in Colombo district, v_c was estimated between 63% and 75% for seasons 1 and 2 while v_c was estimated between 73% and 81% for seasons 3 and 4. In contrast, when the unsupportive beta prior is used, i.e. when a higher v_c is suggested, v_c was estimated between 90% and 96% for all the four seasons – which could be resulted from amplified influence for higher values of v_c from the unsupportive prior. Overall, it can be noted that v_c in the Colombo disrict is over 63% throughout the year.

Using these estimated parameter values for v_c, the estimated dengue density curves were obtained for the two cases of applying supportive and unsupportive beta priors (Figs. 3 and 4). Using the 95% credible intervals obtained for v_c for each season, the uncertainties of dengue cases were obtained at e, $2e$, $3e$ levels using the model in Eq. 2, where $e = I(E(v_c)) - I\left(v_c^U\right)$ when unsupportive prior is used and $e = I(E(v_c)) - I\left(v_c^{U^*}\right)$ when supportive prior is used. Here the symbol E represents the expected value. These confidence bands are shown in gray color in Figs. 3 and 4 with the

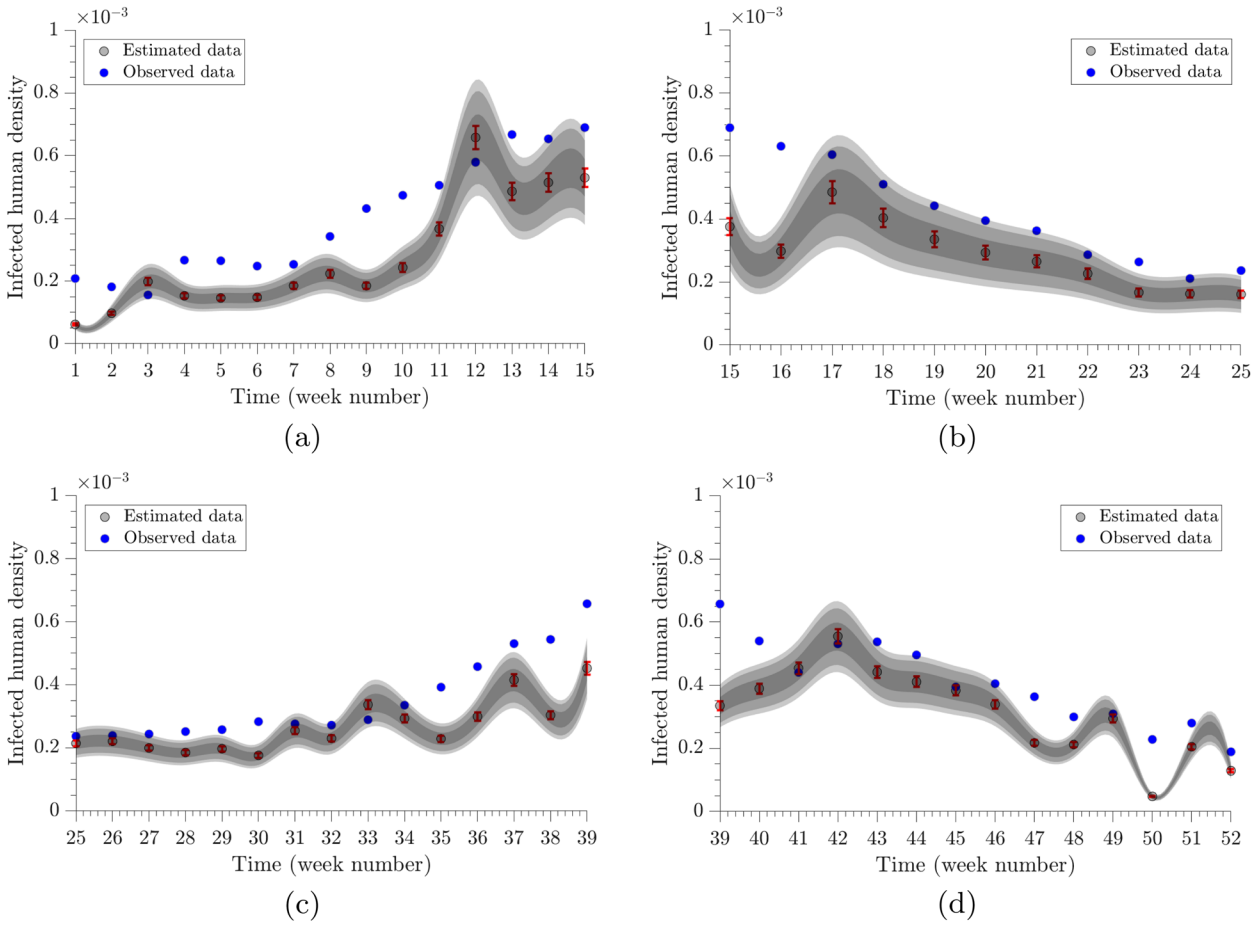

Fig. 3 Estimated dengue densities for the four seasons obtained using the supportive beta prior (right skewed) are shown where blue dots indicate the observed average dengue infected human population densities (I_{obs}) and gray dots indicate the estimated seasonal dengue risk ($I(t+\underline{h})$). The red error bars (e) represent the uncertainties of the respective estimated results. The gray color bands represent the uncertainties of the estimated results at e, $2e$, $3e$ levels. **a**, **b**, **c**, and **d** represent the seasons 1, 2, 3, and 4 respectively

respective error bars. The model presented in Eq. 2 reliably captures the underlying trend of the dengue data. However, it should be noted the underestimation of predictions from model to the observed data. This is because the nonlinearity of dengue incidences cannot be predicted within a linear framework. The predicted curves shows nonlinearity generated from rainfall only. Thus, It is advisable to explore additional external factors influencing vectorial capacity in Eq. 2, or opt for a different modeling approach if the primary aim is to estimate dengue infection counts accurately. Nevertheless, the vectorial capacity equation has proven sufficient for estimating the levels of intrinsic mosquito factors, given that it is the available formula available that integrates host, pathogen, and vector elements.

Discussion

Dengue, a vector-borne illness, has been spreading across tropical and subtropical regions worldwide for numerous decades [1]. However, these regions are anticipated to expand due to the effects of climate change, particularly based on rainfall patterns and temperatures [2]. The uncertainty of outbreaks has increased due to the uncertain complex nature of climate change influence on disease transmission, uncertainty in model frameworks and modeling assumptions, and data limitations leading to more uncertain situations in the future with respect to dengue spread [5, 47]. The Colombo district of Sri Lanka stands out as a densely populated area and has been under significant threat from dengue for an extended period. Specifically, the Colombo district offers favorable conditions for dengue transmission, including high population density, monsoon rains facilitating breeding grounds, suitable temperatures and humidity for mosquito proliferation. With the impact of climate change, alterations in rainfall patterns and rising temperatures are expected to render the Colombo district even more vulnerable and prone to unpredictable outbreaks, potentially overwhelming healthcare facilities. Dengue is

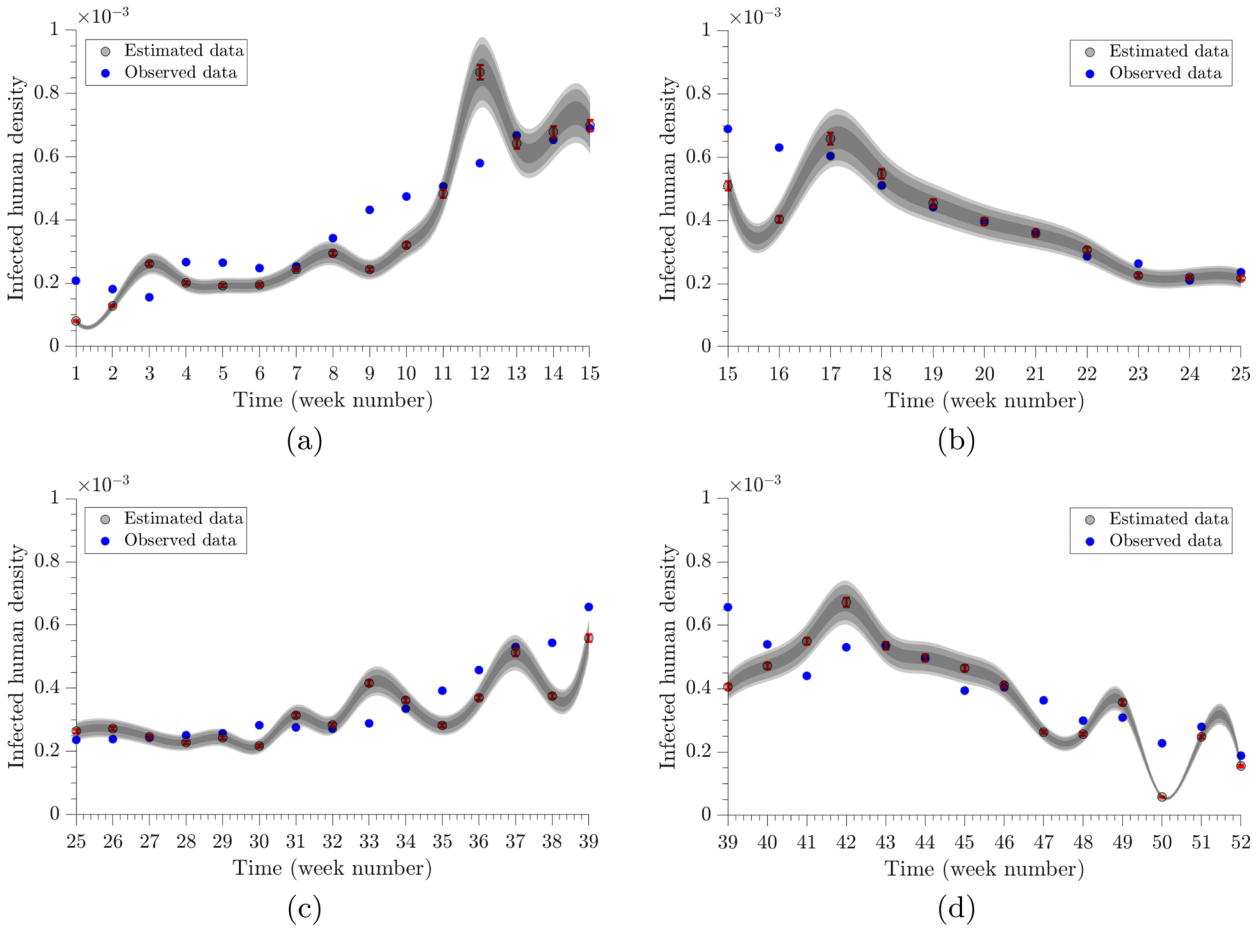

Fig. 4 Estimated dengue densities for the four seasons obtained using the unsupportive beta prior (left skewed) are shown where blue dots indicate the observed average dengue infected human population densities (I_{obs}) and gray dots indicate the estimated seasonal dengue risk ($I(t+\underline{h})$). The red error bars (e) represent the uncertainties of the respective estimated results. The gray color bands represent the uncertainties of the estimated results at e, $2e$, $3e$ levels. **a**, **b**, **c**, and **d** represent the seasons 1, 2, 3, and 4 respectively

influenced by both external and internal factors related to mosquitoes. However, the internal aspects of mosquito biology have often been overlooked, oversimplifying the intricate nature of the disease. Disease transmission compartmental models often disregard these internal factors, hypothesizing that mosquitoes have a 100% capability of becoming infected each time they bite an infected human [48–51]. In our study, we challenge this hypothesis by integrating the internal factors of mosquitoes into the model, aiming to estimate these factors based on observed dengue incidence data. Thus, we introduce a novel analysis to estimate the genetic trait known as vector competence (ν_c), which determines the mosquitoes' susceptibility to infection. Notably, this marks the first attempt to estimate ν_c outside of laboratory conditions using a mathematical framework. This estimation of ν_c helps better model the climate change impact on vector genetics in determining risk of future disease transmission. It further benefits for studying vector adaptation in the midst of climate change and vector control methods.

This mathematical model not only accounts for the combined influence of both external and internal factors but also captures the interplay between these factors. Dengue outbreaks in Sri Lanka are closely synchronized with rainfall seasons, prompting us to integrate rainfall data into our mathematical framework, given that temperature and humidity consistently remain at favorable levels. Moreover, due to the patterns observed in rainfall, we carried out the study for an average year broken down to four seasons [40]. Our developed model establishes a concurrent relationship between ν_c and rainfall, offering a holistic understanding of disease emergence from dual perspectives. The estimation process involved employing the formula of vectorial capacity (V_s) within a Bayesian framework. In this model (5), two unknowns

were identified: (1) the parameter of interest, ν_c, and (2) the proportionality constant, K. The parameter K serves solely as a scaling parameter, and therefore, its significance is negligible for the objectives of this study. Consequently, we determined a lower bound for K by setting ν_c at its upper bound, i.e., $\nu_c = 1$. Subsequently, these lower bounds of K were utilized in estimating ν_c by allowing the estimates of ν_c towards its upper bound as much as possible. Additionally, we endeavored to explore the lower and upper extremes of the estimates by employing supportive and unsupportive priors for ν_c (see the left skewed and right skewed beta distributions in Fig. 1). These dual mechanisms, (a) allowing to estimate ν_c near its upper bound and (b) subsequently attempting to push it towards both ends of the interval [0, 1], ensured the region of accurate estimates for ν_c.

One of the limitations of our study is the assumption of homogeneity in ν_c between *Aedes aegypti* and *Aedes albopictus*, which was necessary due to the lack of detailed data and information. One other limitation of this study is using mortality rate in V_s formula instead of survival rate data. This may oversimplify vector population dynamics overlooking potential variations in the transmission capabilities of these mosquito species. Moreover, while we included monsoon rainfall as the primary environmental variable, other factors such as urbanization, vector control measures, and socio-economic conditions could also significantly influence dengue transmission and may not be adequately represented in our model. Despite these limitations, our findings provide valuable insights into the influence of environmental factors on dengue transmission and underscore the importance of continuous model validation and improvement.

The estimates of ν_c from the supportive beta prior are all between (63%, 80%) for all four seasons across both species *albopictus* and *aegypti*. Although these results are obtained anticipating ν_c to be near its lower bound, the estimation gives higher values suggesting high ν_c in Colombo. Agreeing with these results, for the unsupportive beta prior, as anticipated, ν_c values are near its upper bound ranging between (90%, 96%) (see Fig. 2). When considered both supportive and unsupportive priors, the estimates for ν_c can lie in the bounds (63%, 75%) and (90%, 95%) respectively, for first two seasons where Colombo benefits from Southwest monsoon, known for its heavy rainfall. Similarly the range of ν_c estimates, respectively for supportive and unsupportive priors, can lie in the intervals (73%, 81%) and (92%, 96%) for last two seasons where Colombo benefit from Northeast monsoon characterized by comparatively lower rainfall. The intervals for seasons 1 and 2 (spanning 24 weeks) are comparatively narrower than those of seasons 3 and 4 (extending over 28 weeks). This difference could be linked to the decreased rainfall and prolonged warmer temperatures observed during the Northwest monsoon during seasons 3 and 4 (see Table A1 in Appendix A). This finding confirms that environmental conditions can enhance ν_c throughout a season, a period exceeding the lifespan of a mosquito. Obtaining different boundaries by different prior options justify the sensitivity of ν_c estimates to prior distributions. Further, these boundaries can provide insights into the stability of the ν_c estimates in the light of chosen prior as well as the accuracy of the values if a different prior is chosen.

The value of ν_c is often measured through experimental studies via three components, IR, DE, and TE. Since in this study, we estimate ν_c with respect to the reported infected human cases, our results reflect the TE. Several literature studies on ν_c based on experimental work are showcased in Table A2 in Appendix A. Studies conducted in Europe, Argentina and Uruguay present measurements of ν_c in subtropical and non-tropical environments [14, 15, 52]. These studies reveal lower values of ν_c ($5 - 10\%$ for serotype DENV–1, 20% and 42% for serotype DENV–2) and do not reflect the tropical settings in which ν_c is estimated in our study. Thus values from these regions do not relate and diverge from the estimates in our study. The experimental studies conducted in Brazil, Mexico, and Australia fall into the tropical setting. However, the Australian study was based on an Australian vector which is a different species than the vectors found in Sri Lanka [11]. Among the studies conducted in tropical regions, those in Mexico and Brazil demonstrate a range of moderate values for ν_c, varying between 11% and 62% for serotypes 1 and 4. Furthermore, the experiments quantify the interplay between mosquito genetics and pathogen characteristics through ν_c across different serotypes. For instance, DENV–2 has comparatively higher favorability, resulting in high ν_c values in facilitating increased disease transmission in Brazil. In Sri Lanka, where serotype 2 is most prevalent, our estimates of ν_c align with those from the Brazilian study, particularly for serotype 2, indicating higher values [20, 37]. Additionally, considering DENV–2's greater advantage against mosquito immunity while the tropical setting provides highly favorable external conditions, it becomes evident why Sri Lanka, particularly Colombo, has remained consistently threatened by dengue for many decades. On a different aspect, despite Brazil and Mexico sharing similar environmental conditions, the measurement of ν_c for serotype DENV–1 varies significantly. This suggests when favorable climate conditions are provided, disease transmission from the same serotype can be influenced by vector genetics alone. In summary, the literature studies in Table A2 in Appendix A provide clear evidence of

v_c affecting disease transmission, regardless of geography, climate setting, or environmental conditions. However, our findings in this study cannot be generalized to any vector species (e.g. *Aedes, Anopheles, Culex*, etc.) because the value of v_c depends on characteristics of the virus as well as the intrinsic characteristics of a vector. Thus we limit our findings in this study to Dengue, i.e., *Aedes aegypti* and *Aedes albopictus*.

Conclusions

This study has demonstrated v_c plays a significant role in the transmission dynamics of dengue, influenced by both internal and external factors. Our mathematical model, which incorporates the effects of environmental conditions such as rainfall, provides a comprehensive understanding of how these factors interact with mosquito genetics to affect v_c. The findings highlight favorable environmental conditions can enhance v_c throughout a season, extending beyond the lifespan of a mosquito. Notably, our results suggest even within the same serotype, disease transmission can be significantly influenced by vector genetics alone, emphasizing the importance of considering both genetic and environmental variables in disease prediction models. These insights underscore the need for continuous monitoring and adaptation of vector control strategies, especially in light of climate change and its potential to alter the patterns of dengue outbreaks. As vector-borne diseases continue to change, it will be essential to conduct further research on the variations in v_c across different contexts to develop effective control and prevention strategies.

Our study opens new avenues for future research to delve deeper into understanding the variations of v_c across different geographical areas, virus strains, vector species, and vector genetics. Additionally, further investigations could explore the potential impacts of v_c, offering insights into more effective strategies for dengue control and prevention in the face of evolving climate change and changing disease dynamics. The experimental data in Table A2 in Appendix A suggests mosquito genetics play a vital role in determining v_c for various serotypes. For instance, serotype DENV–2 interacts favorably with the mosquito immune system, resulting in high v_c values and facilitating increased disease transmission. Further, the model can be refined by incorporating additional environmental and socio-economic variables, using more specific data, and exploring non-homogeneous vector competence among different mosquito populations and dengue serotypes. Amidst these prospective avenues, v_c evolves in response to its influencing factors, underscoring the importance of comprehending its evolution - especially in the context of climate change. By continuing to refine our understanding of v_c and its implications for disease transmission, we can better prepare for and mitigate the impacts of dengue outbreaks in vulnerable regions.

Supplementary Information

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Supplementary Material 1.

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Authors' contributions
P. C. - Methodology, Investigation, Simulations, Data curation, Writing the original draft, and editing. L. S. P. - Investigation, Literature Survey and writing, Interpretation of results, Review, and proofreading. S. S. N. P. - Review and discussions. K. D. S.- Conceptualization, Investigation, Methodology, Data curation, Writing the original draft, and editing, Supervising. All authors read and approved the final manuscript.

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Availability of data and materials
The data that support the findings of this study are available from The National Dengue Control Unit, Ministry of Health, Sri Lanka but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are however available from the authors upon reasonable request and with permission of The national Dengue Control Unit, Ministry of Health, Sri Lanka.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
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Competing interests
The authors declare no competing interests.

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