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Transmission-Term Inconsistencies in Vector-Borne Disease Models: Implications for Reproduction Numbers and Stability

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ABSTRACT

Host–vector models of mosquito-borne disease contain two cross-infection terms, and both must rest on one biting assumption and enter the equations as equal loss and gain terms. This study classified inconsistencies in these terms and derived their consequences for the basic reproduction number (R_0), the stability of the disease-free equilibrium and host-population conservation. A Ross–Macdonald-type benchmark model with susceptible, infectious and recovered hosts and susceptible and infectious mosquitoes was formulated under mosquito-limited biting. Its positivity, boundedness, R_0 and local stability were established with the next-generation matrix method and symbolic computation in SymPy. Six error types were defined in two groups. Formulation errors change the equations: mixed denominators in either cross-infection term (E1, E2), mass action with a per-mosquito biting rate (E3) and mismatched loss and gain terms (E4). Analysis and reporting errors leave the equations unchanged: an unstated R_0 convention (E5) and disagreement between analysed and simulated equations (E6). For each system analysed, the disease-free equilibrium was locally asymptotically stable when its computed R_0 was below one, so the threshold theorem did not reveal the error. Instead, E1 and E2 removed the dependence of R_0 on the mosquito-to-host ratio, E3 and E4 made R_0 depend on the unit in which populations are counted, and E4 broke host-population conservation. With parameter values derived from a published malaria model, R_0 was 7.16 for the benchmark, 2.35 under E1 and E2 and 0.77 under a consistent host-limited form. A six-point checklist for detecting these errors is given.

Keywords: Basic Reproduction Number, Host–Vector Model, Incidence Function, Model Consistency, Next-Generation Matrix, Vector-Borne Disease

INTRODUCTION

Mosquito-borne infections remain a large public health burden, and malaria is the largest of them. The World Health Organization (2025) estimated 282 million malaria cases and 610,000 malaria deaths worldwide in 2024, with 94% of the cases and 95% of the deaths in the WHO African Region. The same report lists Nigeria among the five countries that together accounted for more than half of all cases. Recent studies of malaria in Nigeria have used compartmental host–vector models to judge control measures by their effect on the reproduction number. Dambazau et al. (2025) estimated the effective reproduction number for Kano State and found that raising treatment coverage from 50% to 80% lowered it from 1.23 to 0.97. Adim et al. (2026) found that, in Rivers State, combining bed-net use of at least 80% with expanded artemisinin-based combination therapy brought the reproduction number to about 0.8. Duru et al. (2025) proved that the disease-free equilibrium of their model with vaccination, treatment and sterile insect technology is stable when $\mathcal{R}_0 < 1$ and used the sensitivity of $\mathcal{R}_0$ to assess the controls. Atokolo et al. (2023) analysed a host–vector model of vector-borne disease with vertical transmission, treatment and preventive measures, in which both cross-infection terms are bilinear mass-action terms of the form $\beta S_H I_M$ and $\beta S_M I_H$, and proved threshold and stability results from its next-generation reproduction number. In each case the recommendation rests on the value of a reproduction number computed from the model's transmission terms. These Nigerian studies are cited here as examples of how reproduction numbers are used to judge interventions; the analysis in this paper is general and is not a model of malaria in Nigeria.

The way the transmission terms are written determines whether such a model is valid. A model returns a reproduction number and a threshold theorem whenever its equations have a disease-free equilibrium and a next-generation matrix, and these results depend only on the algebraic form of the equations. They do not test whether the transmission terms describe a possible biting process, whether their parameters have the right units, or whether infected individuals leave one compartment at the rate at which they enter another. A model with inconsistent transmission terms can therefore produce a conventional-looking threshold theorem together with a reproduction number that has the wrong value or the wrong dependence on population sizes. This possibility is the motivation for the present study.

Deterministic compartmental equations remain the usual form of such models. In a scoping review of *Plasmodium vivax* transmission models, Anwar et al. (2024) found that 38 of the 48 included studies (79%) used deterministic compartmental models. Dinsa et al. (2024) reviewed eleven age-structured malaria transmission models published between 2019 and 2024, all deterministic and mostly compartmental systems of ordinary differential equations, and Ngwafor et al. (2024) found that 40% of the malaria control optimisation studies they reviewed used compartmental models to estimate health benefits. Grosso et al. (2025) describe compartmental models as the most widespread approach in infectious disease modelling and set out the Macdonald-style vector–host formulation on which

many malaria models are built. For dengue, Aguiar et al. (2022) carried out a ten-year systematic review of mathematical models, and Ogunlade et al. (2023) reviewed dengue transmission models to assess the effect of vector control. In a host–vector model, infection passes from vector to host when an infectious mosquito bites a susceptible host, and from host to vector when a susceptible mosquito bites an infectious host. Each route is written as a cross-infection term that couples the host and vector equations.

The basic reproduction number $\mathcal{R}_0$ is usually computed from these cross-infection terms with the next-generation matrix method of van den Driessche and Watmough (2002); Avram et al. (2024) review the theory of the next-generation matrix and its probabilistic interpretations. For the simplest host–vector models the next-generation matrix has a zero diagonal, so its spectral radius is the square root of the product of the vector-to-host and host-to-vector terms, whereas the expected number of hosts infected by one host through mosquitoes is the product itself. A reported value of $\mathcal{R}_0$ therefore depends both on the cross-infection terms and on the convention used to combine them.

How transmission scales with population size remains an open, partly empirical question. Tompros et al. (2022) compared transmission models for *Batrachochytrium salamandrivorans* in eastern newts, found that pure frequency dependence fitted best, and concluded that reducing host density may be an unreliable control strategy for that pathogen. Salako and Wu (2024) showed for reaction–diffusion epidemic models that whether restricting movement eliminates an infection depends on whether incidence follows mass action or standard incidence. For mosquito-borne disease the question is how biting scales with the numbers of hosts and mosquitoes. Chitnis et al. (2008) let the total number of mosquito–human contacts be half the harmonic mean of the number of bites mosquitoes seek and the number of bites humans can receive; the mosquito-limited and host-limited forms are the two limits of that function. Dahlin et al. (2024) placed temperature-dependent mosquito and parasite traits into two vector–host contact models and found that sustained transmission depends nonlinearly on host density, and that at very high host densities transmission cannot be sustained at any temperature. Dahlin et al. (2025) showed that under the usual assumption of one bite per gonotrophic cycle $\mathcal{R}_0$ increases linearly with the inverse of the cycle duration, and that heterogeneous and multiple biting can make this relationship saturating or concave.

The reliability of published models has also been questioned. Gallo et al. (2022) showed that when the parameters of a compartmental epidemic model cannot be identified from uncertain data, the model's forecasts become unreliable. Henderson et al. (2024) attempted to reproduce 100 randomly sampled infectious disease modelling studies released between January 2020 and August 2022 and could fully reproduce only 4 of them from the resources provided. Pokutnaya et al. (2023) noted that many infectious disease models are hard to reproduce and proposed a 22-element framework for describing such models so that others can share and reproduce them. These studies concern parameter estimation, code and reporting. They do not examine whether the equations of a model are internally consistent, and I found no estimate of how often host–vector models contain inconsistent transmission terms.

Five related but distinct problems can affect transmission terms and the results derived from them. The first is a choice between alternative incidence functions that are each internally consistent, such as mosquito-limited and host-limited biting; this is a modelling decision, and it is the subject of the studies of transmission scaling cited above. The second is an error in a compartmental transfer term, where an infection term leaves one compartment in one form and enters another in a different form. The third is a transmission parameter whose units do not match the term in which it is used. The fourth is the convention used to report$\mathcal{R}_0$. The fifth is disagreement between the equations analysed mathematically and the equations implemented in simulation code. The second to fifth problems are errors, because no single biological assumption produces them, and they are the subject of this study. Examples are a host-to-vector term divided by the vector total while the vector-to-host term is divided by the host total, or an infection term that leaves the susceptible class divided by the host total but enters the infected class without it. In the literature reviewed for this study, I did not find a classification of these errors for host–vector models together with their effects on the results that authors usually prove, namely $\mathcal{R}_0$ and the stability of the disease-free equilibrium. This is a statement about that literature search and does not establish that no such work exists.

This study aimed to classify common inconsistencies in the transmission terms of host–vector disease models and to determine their consequences for reproduction-number calculations, disease-free equilibrium stability, population conservation and the interpretation of epidemiological control thresholds.

MATERIALS AND METHODS

Benchmark Model

The host population is divided into susceptible (S_h), infectious (I_h) and recovered (R_h) classes, and the vector population into susceptible (S_v) and infectious (I_v) classes; mosquitoes do not recover. The model is

$$\frac{dS_h}{dt} = \Lambda_h - \lambda_h S_h - \mu_h S_h, \quad (1)$$

$$\frac{dI_h}{dt} = \lambda_h S_h - (\gamma + \mu_h) I_h, \quad (2)$$

$$\frac{dR_h}{dt} = \gamma I_h - \mu_h R_h, \quad (3)$$

$$\frac{dS_v}{dt} = \Lambda_v - \lambda_v S_v - \mu_v S_v, \quad (4)$$

$$\frac{dI_v}{dt} = \lambda_v S_v - \mu_v I_v, \quad (5)$$

With non-negative initial conditions and$N_h(0) > 0$. Here Λ_h and Λ_v are the constant recruitment rates of hosts and mosquitoes, μ_h and μ_v are their per capita death rates, γ is the recovery rate of infectious hosts, and λ_h and λ_v are the forces of infection on susceptible hosts and susceptible mosquitoes, defined in (7) and (9). All parameters are positive constants. There is no disease-induced death. The total host and vector populations are

$$N_h = S_h + I_h + R_h, \qquad N_v = S_v + I_v, \quad (6)$$

And these definitions are used in every derivation and simulation below. The parameters, their units, the values used in the simulations and their sources are given in Table 1.

Table 1: Parameters of Model (1)–(5) and the Values used in the Simulations. Values Taken from Chitnis Et Al. (2008) are those of their High-Transmission Baseline; the Symbol in Brackets is their Notation. The Computed Death Rates Use their Values $\mu_{1h} = 1.6 \times 10^{-5}$ Day^{-1}, $\mu_{2h} = 3.0 \times 10^{-7}$ Human^{-1} Day^{-1}, $\mu_{1v} = 0.033$ Day^{-1} and $\mu_{2v} = 2.0 \times 10^{-5}$ Mosquito^{-1} Day^{-1}

7	Description	Unit	Value	Source
a	Biting rate per mosquito	day^{-1}	0.50	Chitnis et al. (2008), Table 3 (σ_v)
b	Probability that a bite by an infectious mosquito infects a susceptible host	dimensionless	0.022	Chitnis et al. (2008), Table 3 (β_{hv})
c	Probability that a bite on an infectious host infects a susceptible mosquito	dimensionless	0.48	Chitnis et al. (2008), Table 3 (β_{vh})
γ	Host recovery rate	day^{-1}	0.0035	Chitnis et al. (2008), Table 3 (γ_h)
μ_h	Host death rate at the disease-free equilibrium	day^{-1}	1.729×10^{-4}	Computed as $\mu_{1h} + \mu_{2h} N_h^*$ from Chitnis et al. (2008), Table 3
μ_v	Mosquito death rate at the disease-free equilibrium	day^{-1}	0.13	Computed as $\mu_{1v} + \mu_{2v} N_v^*$ from Chitnis et al. (2008), Table 3
N_h^*	Host population at the disease-free equilibrium	hosts	523	Chitnis et al. (2008), Appendix A
N_v^*	Mosquito population at the disease-free equilibrium	mosquitoes	4850	Chitnis et al. (2008), Appendix A
m	Mosquito-to-host ratio	dimensionless	9.273	Computed from equation (10)
Λ_h	Host recruitment rate	hosts day^{-1}	$\mu_h N_h^*$	Set in this study so that $N_h \to N_h^*$
Λ_v	Mosquito recruitment rate	mosquitoes day^{-1}	$\mu_v N_v^*$	Set in this study so that $N_v \to N_v^*$

Bite Balance and the Forces of Infection

Let κ_v be the number of bites one mosquito makes per day and κ_h the number of bites one host receives per day. A fraction I_v/N_v of the bites a host receives come from infectious mosquitoes, and a fraction I_h/N_h of the bites a mosquito makes land on infectious hosts, so the forces of infection are

$$\lambda_h = \kappa_h\, b\, \frac{I_v}{N_v}, \qquad \lambda_v = \kappa_v\, c\, \frac{I_h}{N_h}. \tag{7}$$

Mosquitoes make $\kappa_v N_v$ bites per day in total and hosts receive$\kappa_h N_h$. Every bite made is a bite received, which gives the bite-balance condition

$$\kappa_v N_v = \kappa_h N_h. \tag{8}$$

The benchmark assumes mosquito-limited biting: each mosquito bites at the fixed rate $\kappa_v = a$ whatever the host density, so by (8) each host receives $\kappa_h = aN_v/N_h$ bites per day. Substituting into (7) gives

$$\lambda_h = \frac{aN_v}{N_h}\, b\, \frac{I_v}{N_v} = \frac{ab\, I_v}{N_h}, \qquad \lambda_v = a\, c\, \frac{I_h}{N_h}. \tag{9}$$

Both forces of infection have units of day^{-1}, as a per capita rate must. The alternative host-limited assumption fixes the bites received per host, $\kappa_h = a$, so by (8) each mosquito bites at $\kappa_v = aN_h/N_v$, and (7) gives $\lambda_h = abI_v/N_v$ and $\lambda_v = acI_h/N_v$. Both assumptions satisfy (8) identically. The host-limited alternative is consistent, is labelled H below, and is not an error; it is included as a second reference case.

The two assumptions describe different biological situations. Mosquito-limited biting is appropriate when hosts are plentiful relative to mosquitoes, so that a host-seeking mosquito finds a host without delay and hosts are far from the number of bites they can tolerate. Host-limited biting is appropriate when mosquitoes are so numerous relative to hosts that the bites hosts can receive, limited for example by defensive behaviour, set the total. In the contact function of Chitnis et al. (2008) the total number of contacts is $\sigma_v N_v \sigma_h N_h/(\sigma_v N_v + \sigma_h N_h)$, where σ_v is the number of bites a mosquito seeks per day and σ_h the maximum number of bites a host can receive per day. Mosquito-limited biting is the limit $m\,\sigma_v/\sigma_h \to 0$ and host-limited biting the limit$m\,\sigma_v/\sigma_h \to \infty$, where m is defined in (10). Dahlin et al. (2024) found that host biting tolerance and host density together determine whether transmission can be sustained.

At the disease-free equilibrium (DFE) the totals are $N_h^* = \Lambda_h/\mu_h$ and$N_v^* = \Lambda_v/\mu_v$, and I write

$$m = \frac{N_v^*}{N_h^*} \tag{10}$$

For the mosquito-to-host ratio.

Classification of Errors

Six error types were defined, in two groups. Formulation errors E1 to E4 change the governing equations and so define a different dynamical system; their transmission terms are listed in Table 2. Analysis and reporting errors E5 and E6 leave the equations unchanged and concern how results are reported and checked.

Table 2: Transmission Terms in the Benchmark, in the Consistent Host-Limited Alternative H, and in Formulation Errors E1 to E4. The Loss Term Appears in Equation (1) or (4) and the Gain Term in Equation (2) or (5). The Limits of the Contact Function of Chitnis Et Al. (2008) are derived in the Numerical Methods Subsection

Case	Host infection: loss / gain	Vector infection: loss / gain	Description	Source
Benchmark	abI_vS_h/N_h / same	acI_hS_v/N_h / same	Mosquito-limited, balanced	Limit $\sigma_h \to \infty$ of Chitnis et al. (2008)
H	abI_vS_h/N_v / same	acI_hS_v/N_v / same	Host-limited, balanced	Limit $\sigma_v \to \infty$ of Chitnis et al. (2008)
E1	abI_vS_h/N_h / same	acI_hS_v/N_v / same	Vector term uses host-limited denominator	Defined in this study
E2	abI_vS_h/N_v / same	acI_hS_v/N_h / same	Host term uses host-limited denominator	Defined in this study
E3	abI_vS_h / same	acI_hS_v / same	Mass action with a still in day^{-1}	Mass-action form: Atokolo et al. (2023); Salako and Wu (2024); error defined in this study
E4	abI_vS_h/N_h / abI_vS_h	acI_hS_v/N_h / same	Loss and gain of host infection differ	Defined in this study

E1 and E2 mix the two biting assumptions. Reading κ_h and κ_v back from (7), E1 implies $\kappa_h = aN_v/N_h$ and$\kappa_v = aN_h/N_v$, so mosquitoes make aN_h bites per day while hosts receiveaN_v. E2 implies$\kappa_h = \kappa_v = a$, so mosquitoes make aN_v bites per day while hosts receiveaN_h. In both cases (8) fails unless$N_h = N_v$. E3 drops the denominators but keeps a as a biting rate per mosquito. E4 writes the host infection term with $1/N_h$ where it leaves S_h and without it where it entersI_h. E5 is reporting the product of the two cross-infection terms as$\mathcal{R}_0$, or not stating which convention is used. The next-generation matrix of van den Driessche and Watmough (2002) gives $\mathcal{R}_0$ as a spectral radius, which for a host–vector model is the square root of that product. E6 is proving threshold or stability results for one form of the transmission terms and simulating a different form.

Analytical Methods

Positivity and boundedness were shown by the standard argument for the non-negative orthant and by solving the linear equations for the totals. The basic reproduction number was computed with the next-generation matrix method of van den Driessche and Watmough (2002), taking I_h and I_v as the infected compartments and the gain terms in equations (2) and (5) as new infections, and was defined as $\mathcal{R}_0 = \rho(FV^{-1})$, the spectral radius of the next-generation matrix. Local stability of the DFE was decided from the characteristic polynomial of the full Jacobian matrix at the DFE, using the Routh–Hurwitz criterion for its quadratic factor; global stability was not examined. All derivations were done symbolically in SymPy 1.14.0 under Python 3.13, for the benchmark, for H and for each of E1 to E4. The script checks that the DFE is an equilibrium of each system, computes the next-generation matrix and its spectral radius, computes and factorises the Jacobian matrix at the DFE, computes dN_h/dt, checks the bite-balance condition (8) for each case, and evaluates $\mathcal{R}_0$ at the values in Table 1. The script is provided as supplementary material.

Numerical methods

The values in Table 1 come from the high-transmission baseline of Chitnis et al. (2008), whose malaria model has the forces of infection

$$\lambda_h = \frac{\sigma_v N_v \sigma_h}{\sigma_v N_v + \sigma_h N_h}\,\beta_{hv}\frac{I_v}{N_v},$$

$$\lambda_v = \frac{\sigma_v \sigma_h N_h}{\sigma_v N_v + \sigma_h N_h}\left(\beta_{vh}\frac{I_h}{N_h} + \tilde{\beta}_{vh}\frac{R_h}{N_h}\right). \qquad (11)$$

As $\sigma_h \to \infty$ these tend to $\sigma_v\beta_{hv}I_v/N_h$ and $\sigma_v\left(\beta_{vh}I_h + \tilde{\beta}_{vh}R_h\right)/N_h$, which with $\tilde{\beta}_{vh} = 0$ are the forces of infection (9) with $a = \sigma_v$. The benchmark is therefore the mosquito-limited limit of their transmission terms. Letting $\sigma_v \to \infty$ instead gives $\sigma_h\beta_{hv}I_v/N_v$ and $\sigma_h\left(\beta_{vh}I_h + \tilde{\beta}_{vh}R_h\right)/N_v$, which with $\tilde{\beta}_{vh} = 0$ are the host-limited forms of case H with $a = \sigma_h$. For their baseline, $\sigma_h = 19$ day^{-1} and $\sigma_v = 0.5$ day^{-1} (Chitnis et al., 2008, Table 3), so $m\,\sigma_v/\sigma_h = 0.244$ and the contact rate of (11) is $1/(1 + 0.244) = 0.804$ times its mosquito-limited value; the mosquito-limited benchmark is the closer of the two limits at these values.

The parameter values were adapted to model (1)–(5) without recalibration. Rates and probabilities were taken directly; the density-dependent death rates of Chitnis et al. (2008) were replaced by their values at the disease-free equilibrium, as shown in Table 1; andΛ_h, Λ_v were set so that the totals in (6) tend to the disease-free population sizes of Chitnis et al. (2008). Their model also has exposed classes for hosts and mosquitoes, loss of immunity, disease-induced death, infectious recovered hosts and density-dependent death. Model (1)–(5) omits these, so its $\mathcal{R}_0$ is not expected to equal the value $\mathcal{R}_0 = 4.4$ that Chitnis et al. (2008) report for this baseline, and the numerical results below are not estimates of malaria transmission.

Model (1)–(5) with forces of infection (9), and its E4 variant, were integrated with the LSODA method of SciPy 1.18.1 (solve_ivp) with relative tolerance 10^{-9} and absolute tolerance10^{-9}, over the interval $0 \le t \le 10$ days. Both runs started from the initial conditions$S_h(0) = 523$, $I_h(0) = 0$, $R_h(0) = 0$, $S_v(0) = 4840$ and $I_v(0) = 10$, that is, the DFE with 10 of the susceptible mosquitoes moved to the infectious class. The benchmark run continued to $t = 10$ days; the E4 run was stopped by an event condition when N_h reached$50N_h^*$. The curves in Figure 1 were computed from the closed-form expressions for $\mathcal{R}_0$ in Table 3, and Figure 2 from the simulated trajectories. Figures were drawn with Matplotlib 3.11.2.

RESULTS AND DISCUSSION

Well-Posedness of the Benchmark model

Theorem 1

Suppose all parameters of model (1)–(5) are positive and the initial condition lies in $\mathbb{R}^5_{\ge 0}$ with$N_h(0) > 0$. Then, with forces of infection (9):

i. the solution stays in $\mathbb{R}^5_{\ge 0}$ for all $t \ge 0$;
ii. $N_h(t) \le \max\{N_h(0), N_h^*\}$ and $N_v(t) \le \max\{N_v(0), N_v^*\}$ for all $t \ge 0$, and $N_h(t) \to N_h^*$, $N_v(t) \to N_v^*$ as $t \to \infty$;
iii. the set

$$\Omega = \{(S_h, I_h, R_h, S_v, I_v) \in \mathbb{R}^5_{\ge 0} : N_h \le N_h^*,\ N_v \le N_v^*\} \qquad (12)$$

is positively invariant, and a solution that starts outside Ω approaches Ω as $t \to \infty$.

Proof. (a) On each face of the non-negative orthant the corresponding derivative is non-negative: $dS_h/dt = \Lambda_h > 0$ when $S_h = 0$, $dI_h/dt = \lambda_h S_h \ge 0$ when $I_h = 0$, $dR_h/dt = \gamma I_h \ge 0$ when $R_h = 0$, $dS_v/dt = \Lambda_v > 0$ when $S_v = 0$ and $dI_v/dt = \lambda_v S_v \ge 0$ when $I_v = 0$. Hence no trajectory leaves the orthant. (b) Adding equations (1) to (3) gives$dN_h/dt = \Lambda_h - \mu_h N_h$, because the infection terms cancel, so $N_h(t) = N_h^* + (N_h(0) - N_h^*)e^{-\mu_h t}$. This lies between $N_h(0)$ and N_h^* for all $t \ge 0$, which gives the bound, shows $N_h(t) \ge \min\{N_h(0), N_h^*\} > 0$ so that λ_h and λ_v stay defined, and gives $N_h(t) \to N_h^*$. Adding equations (4) and (5) gives $dN_v/dt = \Lambda_v - \mu_v N_v$ and the same conclusions forN_v. (c) If $N_h(0) \le N_h^*$ and $N_v(0) \le N_v^*$, the explicit solutions in (b) keep both inequalities for all $t \ge 0$, and with (a) this shows that Ω is positively invariant. If $N_h(0) > N_h^*$ or $N_v(0) > N_v^*$, the corresponding total decreases monotonically to its equilibrium value, so the distance from the solution to Ω tends to zero. ▫

Invariance of the non-negative orthant, part (a), and boundedness by the equilibrium totals, parts (b) and (c), are separate properties. The first holds for every non-negative initial condition; the second holds on Ω only when the initial totals do not exceed N_h^* andN_v^*.

Reproduction Number and Stability of the Benchmark

The DFE is$E_0 = (N_h^*, 0, 0, N_v^*, 0)$. With new infections $\mathcal{F} = (abI_vS_h/N_h,\ acI_hS_v/N_h)^T$ and transitions$\mathcal{V} = \left((\gamma + \mu_h)I_h,\ \mu_v I_v\right)^T$, the Jacobians at E_0 are

$$F = \begin{pmatrix} 0 & ab \\ ac\,m & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} \gamma + \mu_h & 0 \\ 0 & \mu_v \end{pmatrix},$$

$$FV^{-1} = \begin{pmatrix} 0 & ab/\mu_v \\ ac\,m/(\gamma+\mu_h) & 0 \end{pmatrix}. \quad (13)$$

The entry $ac\,m$ of F arises because $S_v/N_h = N_v^*/N_h^* = m$ at E_0. Define

$$\hat{\mathcal{R}}^2 = \frac{a^2bc}{\mu_v(\gamma+\mu_h)}. \quad (14)$$

Theorem 2

Suppose all parameters of model (1)–(5) are positive. The basic reproduction number of the benchmark model is

$$\mathcal{R}_0 = \rho(FV^{-1}) = \sqrt{\frac{a^2bc\,m}{\mu_v(\gamma+\mu_h)}} = \hat{\mathcal{R}}\sqrt{m}. \quad (15)$$

The DFE E_0 is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

Proof. The eigenvalues of FV^{-1} in (13) are $\pm\sqrt{a^2bc\,m/[\mu_v(\gamma+\mu_h)]}$, which gives (15). With the state variables ordered as $(S_h, I_h, R_h, S_v, I_v)$, the Jacobian matrix of (1)–(5) at E_0 is

$$J(E_0) = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & -ab \\ 0 & -(\gamma+\mu_h) & 0 & 0 & ab \\ 0 & \gamma & -\mu_h & 0 & 0 \\ 0 & -ac\,m & 0 & -\mu_v & 0 \\ 0 & ac\,m & 0 & 0 & -\mu_v \end{pmatrix}. \quad (16)$$

The first, third and fourth columns each contain only a diagonal entry, so expanding $\det(\lambda I - J(E_0))$ along these columns gives the factors$(\lambda+\mu_h)^2(\lambda+\mu_v)$. The remaining factor is the characteristic polynomial of the block of (16) in rows and columns I_h andI_v, so

$$p(\lambda) = (\lambda+\mu_h)^2(\lambda+\mu_v)[\lambda^2 + (\gamma+\mu_h+\mu_v)\lambda + \mu_v(\gamma+\mu_h)(1-\mathcal{R}_0^2)], \quad (17)$$

As confirmed in SymPy. Because all parameters are positive, the three eigenvalues $-\mu_h$ (twice) and $-\mu_v$ are negative. By the Routh–Hurwitz criterion the roots of the quadratic factor have negative real parts if and only if both of its coefficients are positive. The coefficient $\gamma+\mu_h+\mu_v$ is always positive and the constant term is positive if and only if$\mathcal{R}_0 < 1$. If $\mathcal{R}_0 > 1$ the quadratic factor is negative at $\lambda = 0$ and tends to $+\infty$ as$\lambda \to \infty$, so it has a positive real root and E_0 is unstable. ▫

The same conclusion follows from Theorem 2 of van den Driessche and Watmough (2002). Theorem 2 is a local result; it does not establish global stability ofE_0. Equation (15) shows that $\mathcal{R}_0$ increases with$m$, and that $\mathcal{R}_0 < 1$ exactly when$m < m_c = 1/\hat{\mathcal{R}}^2$. Writing $\mathcal{R}_{vh} = ab/\mu_v$ and $\mathcal{R}_{hv} = ac\,m/(\gamma+\mu_h)$ for the two non-zero entries ofFV^{-1}, $\mathcal{R}_0 = \sqrt{\mathcal{R}_{vh}\mathcal{R}_{hv}}$, whereas the product $\mathcal{R}_{vh}\mathcal{R}_{hv} = \mathcal{R}_0^2$ is the expected number of hosts infected by one infectious host through mosquitoes. This paper uses $\mathcal{R}_0 = \rho(FV^{-1})$ throughout.

Reproduction Number and Stability under Each Formulation Error

Theorem 3

Suppose all parameters are positive. For the host-limited case H and for formulation errors E1 to E4 defined in Table 2, E_0 is an equilibrium of the corresponding system, the next-generation matrix gives

$$\mathcal{R}_0^2 = \hat{\mathcal{R}}^2\,\phi, \quad (18)$$

with ϕ as listed in Table 3, and the characteristic polynomial of that system at E_0 has the form (17) with this value of $\mathcal{R}_0$. Hence, for each of these systems, E_0 is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

Proof. For each case the SymPy script substitutes E_0 into the right-hand side of the system and obtains zero, computes FV^{-1} from the gain terms in (2) and (5), and factorises the characteristic polynomial of the Jacobian matrix at E_0. In every case the polynomial has the factors $(\lambda+\mu_h)^2(\lambda+\mu_v)$ and a quadratic with linear coefficient $\gamma+\mu_h+\mu_v$ and constant term $\mu_v(\gamma+\mu_h)(1-\hat{\mathcal{R}}^2\phi)$. The argument of Theorem 2 then applies unchanged. ▫

Table 3: The Factor ϕ in $\mathcal{R}_0^2 = \hat{\mathcal{R}}^2\phi$ for each Case, Derived in Sympy, and How $\mathcal{R}_0$ Responds to the Mosquito-to-Host Ratio m

Case	ϕ	Response of $\mathcal{R}_0$ to m	Units of ϕ
Benchmark	m	Increases	Dimensionless
H	$1/m$	Decreases	Dimensionless
E1	1	None	Dimensionless
E2	1	None	Dimensionless
E3	$N_h^*N_v^*$	Not a function of m alone	individuals^2
E4	$N_v^* = mN_h^*$	Increases	individuals

Theorem 3 shows that none of the errors E1 to E4 changes the form of the threshold result. Each erroneous model still has a reproduction number that decides the stability of its DFE. What changes is the value of that number and what it depends on, and these changes are not visible from the stability theorem alone. A threshold theorem is therefore not evidence that the transmission terms of a model are biologically consistent.

Corollary 1 (E1 and E2)

Under E1 or E2, $\mathcal{R}_0 = \hat{\mathcal{R}}$, which does not depend on m, N_h^* or N_v^*. If $\hat{\mathcal{R}} > 1$, the DFE of the erroneous model is unstable at every vector density, whereas the DFE of the benchmark is locally asymptotically stable for $m < m_c$.

Proof. Under E1 the vector-infection term isacI_hS_v/N_v, so the corresponding entry of F is $ac\,S_v/N_v = ac$ at E_0 instead of $ac\,S_v/N_h = ac\,m$, and

$$FV^{-1} = \begin{pmatrix} 0 & ab/\mu_v \\ ac/(\gamma+\mu_h) & 0 \end{pmatrix}. \quad (19)$$

Its spectral radius is $\hat{\mathcal{R}}$. Under E2 the host-infection entry of F becomes $ab\,S_h/N_v = ab/m$ while the vector-infection entry stays$ac\,m$, so the two factors of m cancel in the product of the entries and again$\rho(FV^{-1}) = \hat{\mathcal{R}}$. The stability statement follows from Theorem 3. ▫

E1 and E2 sit between the two consistent assumptions. The benchmark gives $\mathcal{R}_0^2 \propto m$ and the host-limited case H gives $\mathcal{R}_0^2 \propto 1/m$; mixing one denominator from each removes m altogether. This is a mathematical consequence of the error, not a biological prediction: a study built on E1 or E2 would find that larviciding or adult mosquito control has no effect on the threshold, and that finding would be an artefact of the error.

Proposition 1 (E3)

Under E3, $\mathcal{R}_0^2 = \hat{\mathcal{R}}^2N_h^*N_v^*$. If both populations are counted in units of k individuals, $\mathcal{R}_0$ is divided by k.

Proof. The first statement is (18) with$\phi = N_h^* N_v^*$. Counting in units of k individuals replaces N_h^* and N_v^* by N_h^*/k and N_v^*/k, while a, b, c, γ, μ_h and μ_v are unchanged. ▫

The source of the inconsistency in E3 can be seen from the units of each term. The states S_h and I_v are numbers of individuals, a has units of day^{-1} and b is dimensionless, so the E3 term abI_vS_h has units of individual's^2 day^{-1}, whereas dS_h/dt has units of individual's day^{-1}. A reproduction number must be dimensionless and so cannot depend on the unit of counting, and Proposition 1 shows that E3 fails this test. Mass-action incidence is not itself an error. A mass-action term $\beta_h I_v S_h$ is consistent when β_h has units of individual^{-1} day^{-1}; for example, $\beta_h = ab/N_h^*$ and $\beta_v = ac/N_h^*$ give, in SymPy, the same $\mathcal{R}_0$ as the benchmark, and under a change of counting unit β_h and β_v are multiplied by k while the populations are divided by k, so $\mathcal{R}_0$ is unchanged. Such a transformation is a valid reparameterisation. E3 arises only when the mass-action form is combined with the definition of a as a biting rate per mosquito.

Proposition 2 (E4)

Suppose all parameters are positive. Under E4,

$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \frac{abI_vS_h}{N_h} + abI_vS_h = \Lambda_h - \mu_h N_h + ab\, I_vS_h\, \frac{N_h - 1}{N_h}$. (20)

If$N_h^* > 1$, the set Ω in (12) is not positively invariant for the E4 system. The reproduction number is$\mathcal{R}_0^2 = \hat{\mathcal{R}}^2 N_v^* = N_h^* \cdot \hat{\mathcal{R}}^2 m$, which is N_h^* times the benchmark value, and it changes with the unit of counting.

Proof. Equation (20) is the sum of the E4 forms of (1) to (3), confirmed in SymPy: the loss term abI_vS_h/N_h in (1) and the gain term abI_vS_h in (2) no longer cancel, and the spurious term $abI_vS_h(N_h - 1)/N_h$ remains. Take a point of Ω with$N_h = N_h^*$, $S_h > 0$ and$I_v > 0$. There $\Lambda_h - \mu_h N_h = 0$ and, since$N_h^* > 1$, the spurious term is positive, so $dN_h/dt > 0$ and the trajectory leavesΩ. The expression for $\mathcal{R}_0^2$ is (18) with$\phi = N_v^*$, and it is not invariant under the change of unit used in Proposition 1. ▫

The condition $N_h^* > 1$ itself depends on the unit of counting, which is a further sign that the E4 system is dimensionally inconsistent: the spurious term in (20) has units of individual's^2 day^{-1} in its gain part and individual's day^{-1} in its loss part. Proposition 2 means that the invariant region and positivity arguments usually given for host–vector models do not hold for a model with this error, even though the equations look like the benchmark at first reading. The same reasoning applies if the loss and gain terms of the vector infection differ.

Analysis and Reporting Errors

E5 and E6 differ from E1 to E4 because they do not define new dynamical systems, so they do not need a theorem. Under E5, the threshold at 1 is unchanged because $\mathcal{R}_0 > 1$ exactly when$\mathcal{R}_0^2 > 1$. The reported value, however, is $\mathcal{R}_{vh}\mathcal{R}_{hv} = \mathcal{R}_0^2$ instead of the spectral radius (15), so it cannot be compared with values computed under the other convention. Under E6, Theorem 3 applies to whichever system is analysed, and the stated threshold describes the simulated system only if the two systems are the same. Recomputing $\mathcal{R}_0$ from the simulation code detects E6, and it requires the code to be available; Henderson et al. (2024) could fully reproduce only 4 of 100 randomly sampled infectious disease modelling studies from the resources their authors provided.

Numerical Results

With the values in Table 1, $\hat{\mathcal{R}}^2 = 5.529$, so $\hat{\mathcal{R}} = 2.351$ and the critical ratio for the benchmark is $m_c = 0.181$. Table 4 gives $\mathcal{R}_0$ for every case at these values. At the source ratio $m = 9.273$ the benchmark gives$\mathcal{R}_0 = 7.161$, E1 and E2 give 2.351 and H gives 0.772, so the same parameter values place H below threshold and the other forms above it. Under E3 and E4 the value of $\mathcal{R}_0$ changes by a factor of 1000 and of about 31.6 when the populations are counted in thousands instead of individuals, as Propositions 1 and 2 predict.

Table 4. Basic Reproduction Number $\mathcal{R}_0 = \rho(FV^{-1})$ for Each Case at the Parameter Values in Table 1, Computed From (18) and Table 3, for E3 and E4 the Value is Given with Populations Counted in Individuals and in Thousands of Individuals

Case	$\mathcal{R}_0$ (Populations in Individuals)	$\mathcal{R}_0$ (Populations in Thousands)
Benchmark	7.161	7.161
H	0.772	0.772
E1	2.351	2.351
E2	2.351	2.351
E3	3745.0	3.745
E4	163.8	5.178

These comparisons change the transmission terms while keeping the parameter values fixed, without recalibrating the parameters for each form. They therefore show how sensitive $\mathcal{R}_0$ is to the structure of the transmission terms; they are not a like-for-like comparison of epidemiological forecasts and do not establish any difference in malaria transmission risk.

Figure 1 shows $\mathcal{R}_0$ against m for the benchmark, for E1 and E2, and for the host-limited case H. The benchmark crosses the threshold at m_c and H crosses it at $m = \hat{\mathcal{R}}^2 = 5.529$. The E1 and E2 line stays at 2.351 for every m. The three curves meet at $m = 1$, where the factors m, 1 and $1/m$ in Table 3 are equal; at that ratio a single numerical run cannot tell the three models apart.

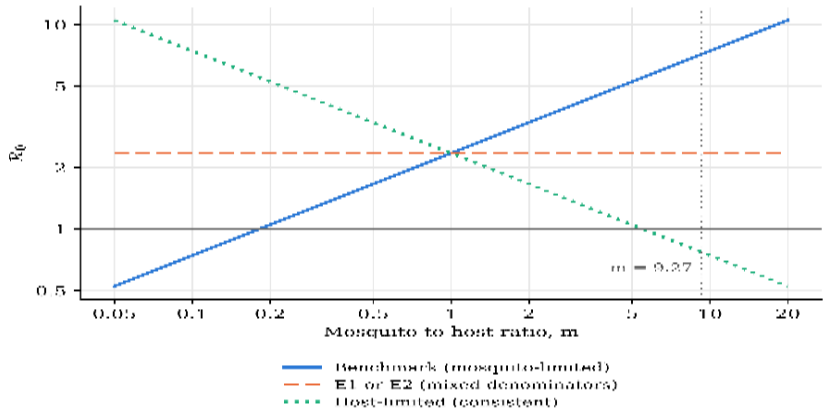


Figure 1: Basic reproduction number $\mathcal{R}_0$ against the mosquito-to-host ratio m for the benchmark model (solid line), error types E1 and E2 (dashed line) and the consistent host-limited case H (dotted line), computed from (18) and Table 3 with the other parameter values in Table 1. The horizontal line is at the threshold $\mathcal{R}_0 = 1$ and the vertical dotted line at the source ratio $m = 9.273$ of Chitnis et al. (2008). Both axes are logarithmic, so the benchmark and H appear as straight lines with slopes $1/2$ and $-1/2$

Figure 2 compares the host total $N_h(t)$ in the benchmark and in E4. In the benchmark N_h stays at $N_h^* = 523$, as Theorem 1 requires. In E4 the host population grows from the start and reached $50N_h^*$ at $t = 4.64$ days, when the event condition stopped the integration. This trajectory is specific to the deliberately inconsistent E4 system and to the parameter values used; it is not a biologically possible population trajectory, and it is shown only to illustrate the loss of conservation in (20).

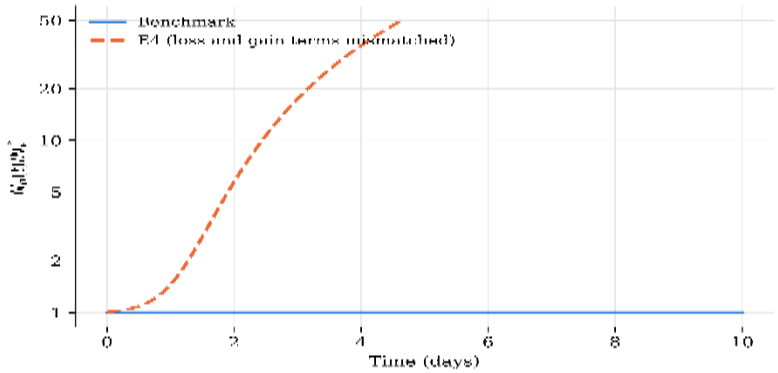


Figure 2: Host population normalised by its disease-free value, $N_h(t)/N_h^*$ with$N_h^* = 523$, for the benchmark model (solid line) and for error type E4 (dashed line), with the parameter values in Table 1 and initial conditions$S_h(0) = 523$, $I_h(0) = R_h(0) = 0$, $S_v(0) = 4840$, $I_v(0) = 10$. The vertical axis is logarithmic. The E4 run was stopped by an event condition when N_h reached$50N_h^*$, at $t = 4.64$ days

Checklist for Authors and Reviewers

Each error type in this study can be detected from a model's equations and code without fitting data. Table 5 lists six checks, the error each one detects and the evidence that shows the check has been passed.

Table 5: Six-Point Checklist for Transmission Terms in Host–Vector Models

Check	Error detected	Evidence that the check is passed
1. State the biting assumption and check that both cross-infection terms use the matching denominator	E1, E2	Bites made by mosquitoes equal bites received by hosts, condition (8), for all population sizes
2. Check the units of every transmission parameter and term	E3, E4	Every term in each equation has units of individuals per unit time, and $\mathcal{R}_0$ is unchanged when populations are counted in a different unit
3. Check that every infection term appears in identical form as a loss and as a gain	E4	dN_h/dt and dN_v/dt contain no infection terms unless disease-induced death is modelled
4. State the reproduction-number convention	E5	The text states whether $\mathcal{R}_0$ is $\rho(FV^{-1})$ or the product of the cross-infection factors
5. Recompute $\mathcal{R}_0$ symbolically from the equations implemented in the simulation code	E6	The recomputed expression is identical to the one stated in the text
6. Check the direction in which $\mathcal{R}_0$ responds to m	E1, E2	$\mathcal{R}_0$ increases with m under mosquito-limited biting and decreases under host-limited biting; an $\mathcal{R}_0$ independent of m indicates E1 or E2

Relation to Earlier Work and Limitations

The opposite responses of the benchmark and of case H to m mean that the same change in population sizes can appear to raise or lower $\mathcal{R}_0$ depending on the biting assumption: under the benchmark, lowering N_h^* raises m and so raises $\mathcal{R}_0$, while under H it lowers $\mathcal{R}_0$. Dependence of control conclusions on the transmission mechanism has also been reported by Tompros et al. (2022) and Salako and Wu (2024), and Dahlin et al. (2024) found that transmission in vector–host contact models depends nonlinearly on host density. The present results add that when the two assumptions are mixed within one model, as in E1 and E2, $\mathcal{R}_0$ loses its dependence on m entirely, which neither consistent assumption predicts.

The analysis has limits. It uses one base model, with no exposed classes, no disease-induced death and constant recruitment. Exposed classes and disease-induced death would change the expression for $\hat{\mathcal{R}}$, but the factor ϕ in Table 3 comes from the denominators of the transmission terms and would be expected to carry over; this was not checked here. Only the local stability of the DFE was treated. The effect of each error on endemic equilibria, on backward bifurcation and on global stability was not derived. The numerical comparisons use parameter values adapted from the more complex model of Chitnis et al. (2008) without recalibration, so they illustrate structural sensitivity and are not estimates of transmission. The six error types were demonstrated within the benchmark framework of this paper and are not an exhaustive classification of all possible errors in host–vector models. Finally, this study does not measure how often these errors occur in published models; that requires a systematic survey of the literature with a predefined sample.

Conclusion

This study classified six types of transmission-term inconsistency in host–vector disease models and derived their consequences for reproduction-number calculations, disease-free equilibrium stability and population conservation. The six types fall into two groups. The formulation errors change the equations: in a Ross–Macdonald-type model, mixing incompatible biting assumptions (E1, E2) removed the dependence of the reproduction number on the mosquito-to-host ratio, dimensionally inconsistent transmission terms (E3, E4) made the reproduction number depend on the unit in which populations are counted, and mismatched loss and gain terms (E4) violated host-population conservation and produced an impossible population trajectory. The analysis and reporting errors leave the equations unchanged: an unstated convention for the reproduction number (E5) prevents comparison of reported values, and disagreement between the analysed and simulated equations (E6) means that the stated threshold does not describe the simulated system. Each system analysed kept the usual local stability threshold under its own assumptions, so that threshold alone did not show whether the transmission terms were consistent. The six-point checklist proposed here is intended for model development and peer review; it has not been tested against a sample of published models. Future work should extend the analysis to more complex host–vector models, examine endemic equilibria and global stability, and measure how often these inconsistencies occur through a systematic review of published models.

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