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Research article

Equilibrium properties of a coupled contagion model of mosquito-borne disease and mosquito preventive behaviors

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Abstract: Although different strategies for mosquito-borne disease prevention can vary significantly in their efficacy and scale of implementation, they all require that individuals comply with their use. Despite this, human behavior is rarely considered in mathematical models of mosquito-borne diseases. Here, we sought to address that gap by establishing general expectations for how different behavioral stimuli and forms of mosquito prevention shape the equilibrium prevalence of disease. To accomplish this, we developed a coupled contagion model tailored to the epidemiology of dengue and preventive behaviors relevant to it. Under our model's parameterization, we found that mosquito biting was the most important driver of behavior uptake. In contrast, encounters with individuals experiencing disease or engaging in preventive behaviors themselves had a smaller influence on behavior uptake. The relative influence of these three stimuli reflected the relative frequency with which individuals encountered them. We also found that two distinct forms of mosquito prevention—namely, personal protection and mosquito density reduction—mediated different influences of behavior on equilibrium disease prevalence. Our results highlight that unique features of coupled contagion models can arise in disease systems with distinct biological features.

Keywords: coupled contagion; epidemiology; human behavior; ordinary differential equations; vector-borne disease

1. Introduction

Dengue virus is a mosquito-borne pathogen transmitted by *Aedes aegypti* and *Ae. albopictus* mosquitoes that poses a risk to approximately half of the world's population [1]. Currently, there is no treatment for dengue and only one moderately effective vaccine available, so interventions that

target mosquitoes are the primary means of dengue prevention [2]. These interventions span a variety of specific techniques, but they often boil down to either large-scale insecticide spraying, typically performed by government agencies, or in-home water container management, typically performed by residents and reinforced with educational campaigns [3,4]. Empirical studies suggest that "intersectoral" approaches combining spraying and community-driven control are more successful than spraying or community control strategies alone, although it is difficult to generalize across settings [5].

An important factor in the success of either of these strategies is human behavior, since interventions can only be effective if they are adopted in the first place. Behavioral choices can influence compliance with spraying campaigns and participation in mosquito larval habitat reduction, sometimes producing unexpected outcomes. For example, outdoor spatial spraying alone has been associated with a lower adoption of in-home water container management and higher entomological risk, as observation of outdoor spatial spraying can give a false sense of security [6–8]. Inclusion of educational programming in a campaign can counteract this effect, however [9]. Most importantly, though, clinical trials of community interventions have reported up to a 25% reduction in Aedes-borne diseases [10, 11], demonstrating that individual behavior can have a measurable impact on transmission.

Mathematical modeling has been used to elucidate the interplay between disease and behavioral dynamics for a variety of directly transmitted pathogens, particularly those impacted by vaccine hesitancy [12, 13] and changing contact patterns [14–16]. Forecasting models that account for individual behavioral change can produce significantly different forecasts than those that disregard adaptive behaviors [16], which could compromise forecast accuracy [17, 18]. There are comparatively few published models of mosquito-borne disease dynamics that include behavior, and much remains unknown about the relationships among mosquito density, disease prevalence, and preventive behaviors. Previous studies have explored relatively narrow questions around this topic, such as how changing mosquito preventive behaviors in the presence of a dengue vaccine and varying intervention effectiveness, can impact transmission [19,20]. Those studies have also provided insight into the impacts of information sharing across spatiotemporal scales and targeted public health messaging on mosquito-borne disease incidence [21, 22].

When disease-related behavior itself is conceptualized as an infectious entity, a "coupled contagion" model presents a useful framework for understanding the feedback between such behaviors and disease. This approach to modeling co-circulating contagious processes together, rather than independently, specifically considers how transmission is impacted as the contagions evolve and interact. For directly transmitted pathogens, reducing contact via social distancing or isolation are the primary forms of preventive behavior. Several studies coupling adaptive behaviors and directly transmitted pathogens found that even limited amounts of fear-driven self-isolation can drive multiple waves of infection in an epidemic scenario [23–27], while targeted public health messaging strategies could minimize outbreak size across a communication network [28]. Mosquito-borne diseases, in contrast, can be influenced by a wider range of preventive behaviors. In addition to reducing the mosquito biting rate through the use of a repellent, other preventive behaviors for mosquito-borne diseases include those with an indirect effect on transmission; namely, these are actions taken to reduce the mosquito population, rather than transmission itself. Likewise, mosquito-borne diseases are unique in that exposures to mosquito biting could prompt individuals to engage in preventive behaviors, independent of disease status.

In this study, we sought to establish fundamental principles for the coupled-contagion dynamics of a mosquito-borne disease and mosquito prevention behaviors. Our model allowed for two distinct

mosquito prevention behaviors: use of personal protection (which only confers direct protection) and reduction of the mosquito larval habitat (which confers indirect protection by reducing mosquito density). We considered three distinct influences that would prompt individuals to engage in these behaviors: encountering people experiencing disease, encountering people engaging in preventive behaviors, and encountering mosquitoes. Because our model with fully coupled dynamics of disease and behavior was not analytically tractable, we first performed separate analyses of the equilibria of each component model. We then explored the equilibria of the coupled model using numerical simulations, with a focus on understanding how the assumptions and parameters underpinning the uptake and impact of preventive behaviors affect the equilibrium prevalence of behavior and, ultimately, disease.

2. Methods

2.1. Single contagion models

To explore the interface between mosquito-borne disease and mosquito preventive behaviors, we developed two deterministic compartmental models for the standalone dynamics of disease and behavior. We established a constant human population size N,

$$N = S(t) + I_P(t) + R_P(t),$$

such that $N \equiv 1$, and leveraged this to characterize both single-contagion systems using analytical methods. We identified local equilibria using Mathematica 14.0 [29], and performed local stability analyses of those equilibria using the caracas R package (*version 2.0.1*) [30]. Calculations of the basic reproduction number were performed using the approach described in [31].

2.1.1. Disease model

We developed a susceptible-infected-recovered-susceptible (SIRS) model to describe disease dynamics, modeled after dengue. In reality, dengue viruses comprise four distinct serotypes that confer lifelong homologous immunity and temporary heterologous immunity [32]. Although the SIRS model we used does not capture the full complexity of these dynamics, it does capture the fact that waning heterologous immunity (as individuals transition from R to S) allows persistence of the four serotypes in aggregate. In this model, susceptible individuals S are subject to the force of infection for the pathogen, βI, such that

$$\frac{dS}{dt} = \xi R_P - \beta I_P S, \tag{2.1}$$

where the P subscript denotes that this model refers to infection with the pathogen. Infected individuals I_P recover from dengue at rate γ and lose immunity to dengue at rate ξ, or

$$\frac{dI_P}{dt} = \beta I_P S - \gamma I_P \tag{2.2}$$

$$\frac{dR_P}{dt} = \gamma I_P - \xi R_P. \tag{2.3}$$

Given that $\frac{dN}{dt} = 0$ and $N \equiv 1$, we reduced this system to

$$\frac{dS}{dt} = \xi(1 - S - I_P) - \beta I_P S \tag{2.4}$$

$$\frac{dI_P}{dt} = \beta I_P S - \gamma I_P \tag{2.5}$$

and performed our analyses thereon.

2.1.2. Behavior model

We used a susceptible-infected-susceptible (SIS) model to describe the transmission of mosquito preventive behavior. In the absence of disease, susceptible individuals S become infected with the preventive behavior at a rate $\lambda_B = \phi_M M + \phi_S I_B$, so that

$$\frac{dS}{dt} = \omega I_B - \lambda_B S, \tag{2.6}$$

where the B subscript denotes that this model refers to infection with the behavior. Adoption of behavior occurs at a rate equal to the sum of the current ratio of mosquitoes to humans M and the proportion of individuals already performing the behavior I_B, weighted by two parameters ϕ_M and ϕ_S, respectively. The ratio of mosquitoes to humans varies over time as the proportion of the population engaged in preventive behaviors does, such that $M = m(1 - (1 - \alpha_M)I_B)$, where $1 - \alpha_M$ represents the efficacy of the preventive behavior in reducing mosquito density. Infected individuals then "recover" from this behavior at rate ω,

$$\frac{dI_B}{dt} = \lambda_B S - \omega I_B, \tag{2.7}$$

at which time they revert back to the state of no longer engaging in the behavior. We conducted the same analysis of this two-dimensional system as was done for our disease model.

2.2. Coupled contagion model

Using our understanding of single contagion model equilibria and parameter relationships, we integrated the two models into a single ODE system capturing the joint transmission of mosquito-borne disease and mosquito preventive behavior (Figure 1).

Here, the coupled dynamics of disease and behavior were modeled as an SIRS-SIS system in which humans can be infected with behavior alone (denoted by subscript B), pathogen alone (denoted by subscript P), or co-infected with both pathogen and behavior (subscript PB). Simultaneous co-transmission of pathogen and behavior is not allowed under our model, given that it would further complicate the model and is likely to be an exceedingly rare event. The coupled nature of the model allows for dynamic feedback between disease and behavior, which can interact in ways that drive long-term disease dynamics [25].

As before, we assumed a constant human population without demographic change, disease-induced mortality, or serotype dynamics. We summarize human population attributes as follows, where N represents the entire population, N_B the proportion of the population engaged in preventive behaviors, and N_P the proportion of the population infected with the pathogen, implying

$$N = S + I_B + I_{PB} + I_P + R_P + R_{PB}$$

$$N_B = I_B + I_{PB} + R_{PB}$$

$$N_P = I_P + I_{PB}.$$

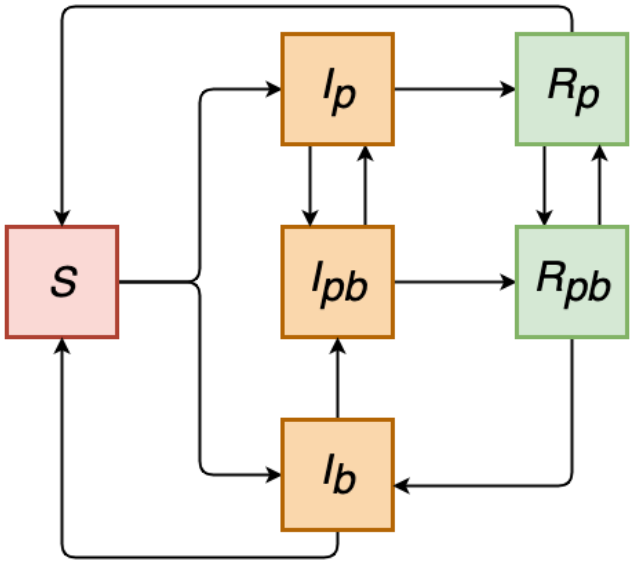

Figure 1. Diagram of the coupled contagion model for dengue and behavior transmission. Compartment labels refer to susceptible S, infected I, and recovered R classes, while subscripts indicate the contagion associated with each state: P for the pathogen alone, B for behavior alone, and PB for both pathogen and behavior.

Susceptible individuals (S) in the model are naive to both pathogen and behavior, and become infected with the pathogen at rate λ_P and behavior at rate λ_B,

$$\frac{dS}{dt} = \omega I_B + \xi R_P - \lambda_P S - \lambda_B S. \tag{2.8}$$

Those infected with the pathogen (I_P) or behavior (I_B) alone can then become co-infected (I_{PB}) at rate λ_B or λ_{PB}, respectively, so that singly infected states change via the contagious processes described in

$$\frac{dI_P}{dt} = \lambda_P S + \omega I_{PB} - \lambda_B I_P - \gamma I_P \tag{2.9}$$

and

$$\frac{dI_B}{dt} = \lambda_B S - \lambda_{PB} I_B - \omega I_B + \xi R_{PB}, \tag{2.10}$$

and contribute to the co-infected state

$$\frac{dI_{PB}}{dt} = \lambda_B I_P + \lambda_{PB} I_B - \gamma I_{PB} - \omega I_{PB}. \tag{2.11}$$

Once singly or co-infected, immunity to the pathogen and the behavior each wane at independent rates ξ and ω, respectively, as individuals recover from infection and cease performing the behavior, such that

$$\frac{dR_P}{dt} = \gamma I_P - \xi R_P - \lambda_B R_P + \omega R_{PB} \tag{2.12}$$

and

$$\frac{dR_{PB}}{dt} = \lambda_B R_P + \gamma I_{PB} - \xi R_{PB} - \omega R_{PB}. \tag{2.13}$$

While the high-level transmission processes in the coupled model are structurally consistent with our simpler, single contagion models, we have modified elements of both pathogen and behavior transmission to explicitly incorporate a mechanism for feedback between them. To do so, we first defined the force of infection for behavior, λ_B, to include the number of individuals infected with the pathogen N_P (i.e., $I_P + I_{PB}$), so that transmission of behavior is driven by a weighted sum of the proportion of the population infected with the pathogen, the adult mosquito prevalence, and the proportion of the population already engaged in the behavior, equal to

$$\lambda_B = \phi_D N_P + \phi_M M + \phi_S N_B, \tag{2.14}$$

where ϕ_D, ϕ_M, and ϕ_S are the weights for these three respective behavioral stimuli. We chose these three stimuli because they span the range of possible influences on mosquito preventive behavior uptake [33–35].

Table 1. Disease parameters, definitions, values, and sampling ranges for global sensitivity analysis. Parameters described as calibrated were tuned to achieve an outbreak matching published estimates of R_0 for dengue virus in temperate regions, approximately 2.0 [36, 37].

Parameter	Definition	Value	Sampling range	Source(s)
α_B	Relative risk associated with use of personal protection and participation in behavior	0.569	0–1	[38]
1 - α_M	Efficacy of community-level preventive behaviors in reducing mosquito density	0.47	0–1	[10]
ξ	Waning rate of heterologous dengue immunity	180 days^{-1}	160–200 days^{-1}	[39, 40]
m	Baseline ratio of mosquitoes to humans	1.0	0.75–3.0	[41]
γ	Dengue infectious period	7 days^{-1}	5–9 days^{-1}	[42]
v	Extrinsic incubation period	14 days^{-1}	12–16 days^{-1}	[43]
g	Mosquito mortality rate	0.18 per day	0.13–0.23 per day	[44]
a	Mosquito biting rate	0.76 per day	0.6–0.9 per day	[45]
b	Probability of mosquito-to-human transmission given a bite by an infectious mosquito	0.3	0.2–0.4	Calibrated
c	Probability of human-to-mosquito infection given a bite on an infectious host	0.3	0.2–0.4	Calibrated

We define the force of infection of the pathogen for those not performing the behavior to be

$$\lambda_P = \beta N_P, \tag{2.15}$$

which features an expanded definition of the pathogen transmission rate β to include key components of mosquito biology and pathogen transmission [46]. By substituting the Ross-Macdonald model's

basic reproduction number expression for mosquito-borne pathogen transmission into our disease-only model's R_0 term, we can express the transmission rate as

$$\beta = \frac{Ma^2bce^{-gv}}{g}, \tag{2.16}$$

where M is the ratio of mosquitoes to humans, a is the blood-feeding rate, b is the probability that an infectious mosquito infects a susceptible human during blood-feeding, c is the probability that an infectious human infects a susceptible mosquito during blood-feeding, g is the mosquito mortality rate, and v is the extrinsic incubation period (Table 1). As in the behavior-only model, M is proportional to the population engaging in preventive behaviors, meaning that

$$M = m(1 - (1 - \alpha_M)N_B),$$

where $1-\alpha_M$ represents the reduction in mosquito density associated with preventive behaviors that result in mosquito larval habitat reduction, which provides the entire population with indirect protection [10].

In addition to these community-level effects on mosquito density, we assumed that individuals engaged in preventive behaviors also experienced some amount of direct, personal protection through actions such as the use of mosquito repellent. The parameter α_B encapsulates this as a relative risk of infection. This shows up in our model as a squared effect given that it modifies the blood-feeding rate, a, and mosquitoes blood-feed twice in a transmission cycle (Eq 2.16). Thus, the corresponding force of infection for those performing the behavior is

$$\lambda_{PB} = \alpha_B^2\lambda_P. \tag{2.17}$$

All behavioral parameters, definitions, and values are defined in Table 2.

Table 2. Behavioral parameters, definitions, values, and sampling ranges for global sensitivity analysis.

Parameter	Definition	Value	Sampling range	References
ϕ_D	Weight of disease on preventive behavior participation	0.01	0–1	Assumed
ϕ_M	Weight of mosquitoes on preventive behavior participation	0.01	0–1	Assumed
ϕ_S	Weight of social influence on preventive behavior participation	0.01	0–1	Assumed
ω	Waning rate of preventive behavior	15 days^{-1}	1–30 days^{-1}	Assumed

We used numerical analyses to approximate model equilibria based on the prevalence attained after five years of model simulation, under a range of behavioral conditions. Simulations were performed using R Statistical Software (*version 4.3.2, 2023-10-31*) and the deSolve R package (*version 1.38*) [47, 48].

We also performed a global, variance-based sensitivity analysis of the model with the Sobol method, using the sensobol R package (*version 1.1.5*) [49]. This allowed us to quantify the amount of variance

in disease and behavior equilibrium prevalence values that could be attributed to our input parameters and interactions between them. We included all disease and behavior parameters in this analysis and generated 16,000 parameter combinations of parameter values from the ranges given in Tables 1 and 2 using the Saltelli sampling scheme.

3. Results

3.1. Single contagion equilibria

3.1.1. Disease model

This model possesses two equilibrium solutions: a disease-free equilibrium, $(S^*, I^*) = (1, 0)$, and an endemic equilibrium $(S^*, I^*) = (\frac{\gamma}{\beta}, \frac{\xi(\beta-\gamma)}{\beta(\xi+\gamma)})$. Since an epidemic requires that $\frac{dI_P}{dt} > 0$, we substituted Eq 2.5 into this inequality and algebraically manipulated it until we obtained

$$\frac{\gamma}{\beta S} > 1.$$

Assuming a completely susceptible population, or $S = 1$, we found the expression for the basic reproduction number for the system, $R_0 = \frac{\beta}{\gamma}$. To assess the stability of this equilibrium, we formulated the Jacobian matrix

$$J = \begin{pmatrix} -\xi - \beta I_P & -\xi - \beta S \\ \beta I_P & \beta S - \gamma \end{pmatrix},$$

evaluated it at the disease-free equilibrium, and identified eigenvalues $\lambda_1 = \beta - \gamma$ and $\lambda_2 = -\xi$. Because λ_2 will always remain negative, values of λ_1 determine stability. Notably, $\beta/\gamma > 1$ and $\beta - \gamma > 0$ are always satisfied simultaneously, meaning that the value of R_0 determines equilibrium stability. These results are consistent with previous work [50–52] and provide a reference point for our more complex model that incorporates behavior, as well.

3.1.2. Behavior model

To analytically characterize behavioral contagion dynamics in the absence of disease, we leverage model population characteristics $\frac{dN}{dt} = 0$ and $N \equiv 1$ to reduce the system outlined in Eqs 2.6 and 2.7 to

$$\frac{dI_B}{dt} = \lambda_B(1 - I_B) - \omega I_B. \tag{3.1}$$

We then substituted in the previously mentioned expressions for λ_B and M to produce the expanded

$$\frac{dI_B}{dt} = \Big(\phi_M m(1 - (1 - \alpha_M)I_B) + \phi_S I_B\Big)\Big(1 - I_B\Big) - \omega I_B, \tag{3.2}$$

which is a Bernoulli differential equation. As such, it was possible to identify the closed-form solution

$$I_B(t) = \frac{1 + e^{-\lambda t + C_2}}{r_1 + r_2 e^{-\lambda t + C_2}}, \tag{3.3}$$

where

$$\lambda = A(r_1 - r_2),$$

$$A = \phi_M m,$$

$$B = -2\phi_M m + \alpha_M \phi_m m - \omega,$$

$$C = \phi_M m - \alpha_M \phi_M m - \phi_S,$$

$$r1 = \frac{-B + \sqrt{B^2 - 4AC}}{2A},$$

$$r2 = \frac{-B - \sqrt{B^2 - 4AC}}{2A},$$

and C_2 is an integration constant. We then found two equilibrium solutions,

$$I_B^* = \frac{-\omega - 2m\phi_M + m\alpha_M\phi_M + \phi_S \pm \sqrt{-4m\phi_M(m\phi_M - m\alpha_M\phi_M - \phi_S) + (-\omega - 2m\phi_M + m\alpha_M\phi_M + \phi_S)^2}}{2(-m\phi_M + m\alpha_M\phi_M + \phi_S)}.$$

Though this equilibrium formula is not intuitive, it provides the initial impression that the baseline mosquito to human ratio m and other related parameters—namely, those weighting mosquito density ϕ_M and larval habitat reduction efficacy α_M—are influential to the behavior equilibrium. This makes sense given the dynamic feedback between the mosquito population and behavior, which are necessarily more difficult to disentangle than the one-way effect of social reinforcement. We also note that parameter values must satisfy the condition

$$\phi_M m(1 - \alpha_M) \neq \phi_S.$$

When this condition is violated, the right-hand side of Eq 3.2 no longer contains a quadratic dependence on I_B, changing the structure of the model. Though there is no evident biological interpretation for this relationship, when parameter combinations do not meet this condition under the baseline model parameterization, we observe an intersection between the positive, stable solution and the negative, unstable solution, though there is no change in stability at these points (Figure A1). Additionally, the parameter weighting the influence of mosquito density, ϕ_M, must be positive for I_B to go from zero toward its non-zero equilibrium. When we examine Eq 3.2 with $\phi_M = 0$, it simplifies to

$$\frac{dI_B}{dt} = \phi_S I_B\Big(1 - I_B\Big) - \omega I_B.$$

In a population without behavior already present (i.e., with $I_B = 0$), this reduced equation will always equal zero. We can interpret this to mean that in this model, mosquitoes must be a behavior stimulus for the behavior to arise in the first place.

We further explored model behavior and parameter relationships via linear stability analysis of Eq 3.2. Evaluating the first derivative of this equation at the positive equilibrium across a range of plausible parameters produces all negative values, indicating that these parameter combinations produce only stable solutions. The solutions associated with this analysis are shown in Figure 2, where we see that as the waning rate of the behavior decreases (i.e., the duration of the behavior increases), the equilibrium prevalence of the behavior increases. In addition to the fact that this means that fewer individuals give up preventive behaviors in a given amount of time, more individuals in this category are gained as a result of a stronger influence of people taking up preventive behaviors due to social influence. We note that the sensitivity of I_B^* to ϕ_S becomes stronger in Figure 2 as ω decreases, which supports this interpretation.

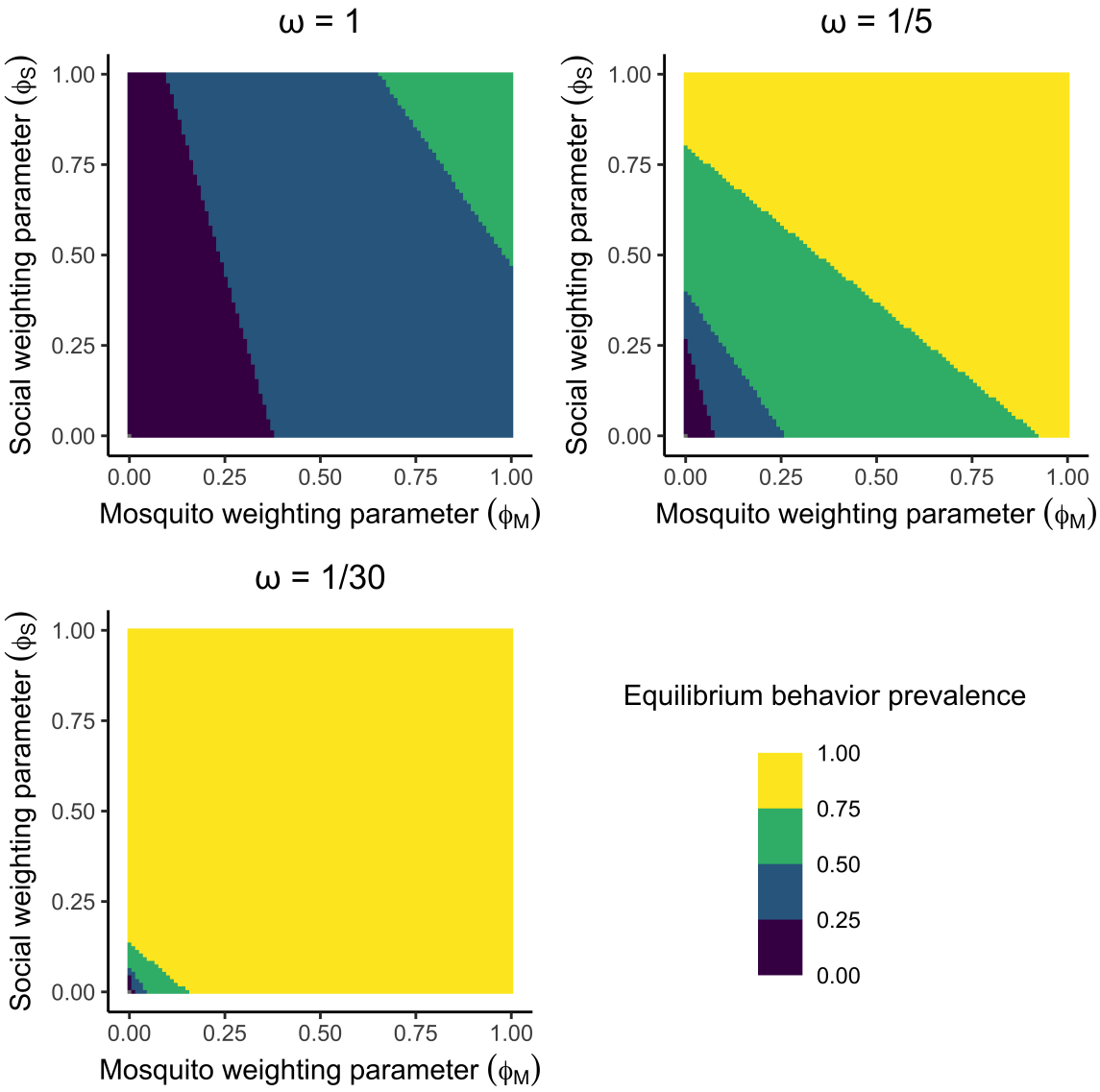

Figure 2. Prevalence of behavior under varying behavior waning rates ω. Fill color indicates the equilibrium prevalence of the behavior in a population. We assume the baseline value for mosquito control efficacy, $1 - \alpha_M = 0.47$, while varying weighting parameters for the mosquito population ϕ_M and social pressure ϕ_S.

3.2. Coupled contagion equilibria

We approached our analysis of the coupled contagion model numerically. Informed by our analysis of the single contagion model for behavior in Figure 2, we set all ϕ parameters to 0.01 to ensure that the magnitude of observed disease and behavior remained within reasonable bounds over the course of a simulation and to exert a measure of control on the relative importance of each stimulus. Furthermore, setting the baseline mosquito-to-human ratio m to 1 ensured that the product $\phi_M M$ was equal to ϕ_S and ϕ_D. These parameter choices established a baseline against which the effects of changes to ϕ parameters could be easily interpreted.

Simulating the model across a five-year period in the absence of preventive behaviors, we observed an equilibrium disease prevalence of 0.019, with a cumulative annual incidence of 971 per 1000 people for the final year of the simulation (Figure 3). These outcomes are plausible according to prior work on dengue epidemiology [36, 37, 53] and were most sensitive to the tuned transmission parameters b and c (Table 1). When we introduced behavior into the model, we observed a reduction in equilibrium disease prevalence from 0.019 to 0.015, with a corresponding reduction in cumulative annual incidence from 971 per 1000 people to 789 per 1000 people once equilibrium behavior prevalence was attained. In both scenarios, we found that the time-varying effective reproductive number declined from 2.0, the value to which it was calibrated initially, to approximately 1.0 by the end of the five-year simulation as the system reaches equilibrium (Figure A2).

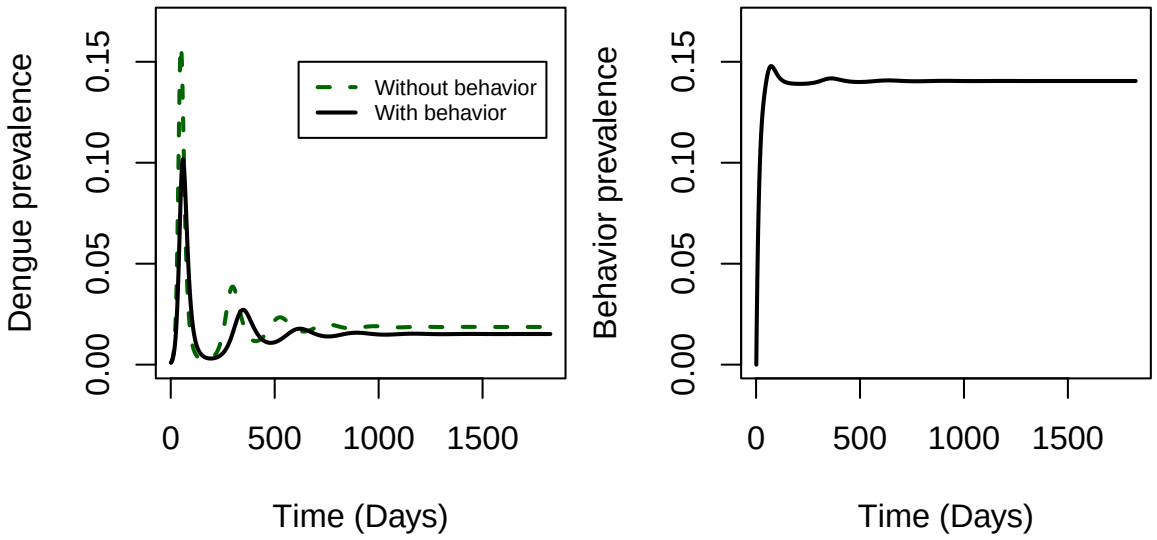

Figure 3. Disease and behavior dynamics over the course of a five-year simulation. Line color and type indicate the presence or absence of the behavioral contagion.

To understand the relative influence of different behavioral parameters, we systematically removed each influence on behavior participation one by one under our baseline model parameterization (Figure 4). Removing the influence of disease ($\phi_D = 0$) increased equilibrium disease prevalence only slightly, from 0.01517 to 0.01522. Removing the influence of social pressure ($\phi_S = 0$) increased disease prevalence somewhat more, up to 0.160. Most significantly, removing the influence of mosquitoes ($\phi_M = 0$) increased disease prevalence to 0.0186, which suggests that the influence of mosquitoes accounts for the majority of the reduction in disease prevalence when all influences on behavior are included. The extent of these reductions in disease correlate with the extent of change in the equilibrium behavior prevalence under each scenario about the ϕ parameters (Figure 4B). Ultimately, the relative importance of these influences on behavior owes to the frequency with which people susceptible to the behavior encounter them. Whereas everyone is bitten by mosquitoes, fewer are in contact with individuals engaged in preventive behavior, and very few are in contact with an infectious person at any given time.

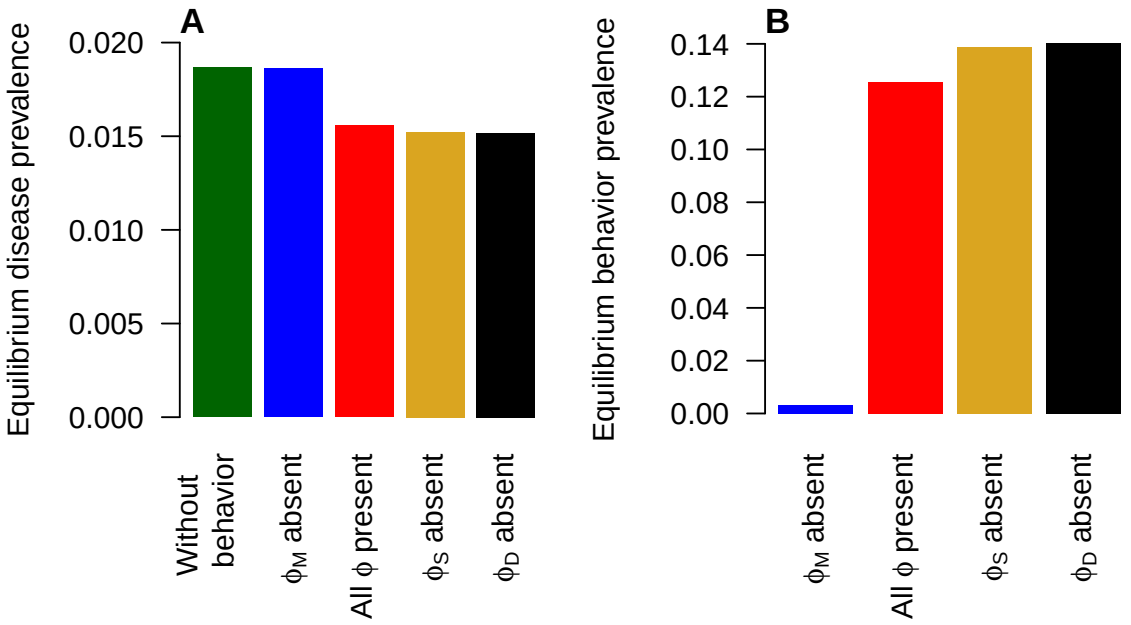

Figure 4. Equilibrium disease and behavior prevalence as influences on behavior participation are removed one by one. Fill color indicates the presence or absence of a given stimulus.

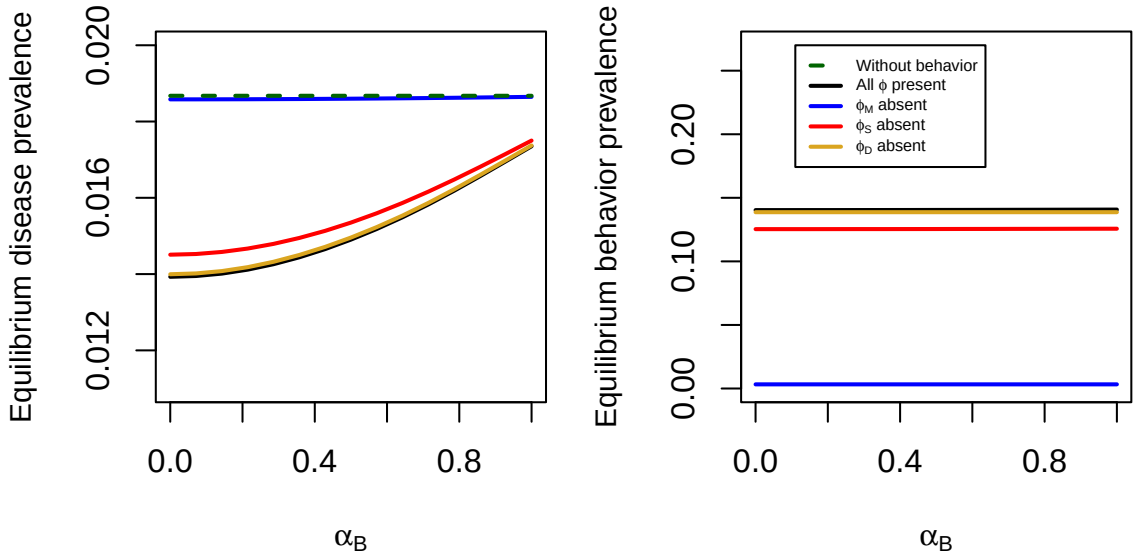

Figure 5. Equilibrium disease and behavior prevalence as relative risk associated with direct protection arising from preventive behavior α_B varies. Line colors indicate the behavior stimuli present or absent.

In general, we found that the effects of the ϕ parameters on disease were robust to values of other model parameters. Even so, the parameters governing the strength of the two forms of control played an important role in shaping these effects. First, changes in the relative risk of infection due to personal

protection (α_B) used by individuals in the behavior class had no effect on the prevalence of behavior but did affect disease prevalence (Figure 5). The curvilinear relationship between α_B and equilibrium disease prevalence reflects the quadratic effect of α_B on R_0, given that it affects both mosquito bites required for a complete transmission cycle. Second, changes in the efficacy of community-level larval habitat reduction ($1 - \alpha_M$) affected not only disease prevalence but also equilibrium behavior prevalence (Figure 6). This is a result of the fact that community-level control reduces mosquito density (M), which, in turn, reduces behavior prevalence. This reduction in behavior prevalence has the undesirable effect of reducing the use of personal protection. However, this undesirable effect is outweighed by the desirable effect of reducing mosquito density, resulting in a net reduction in disease prevalence.

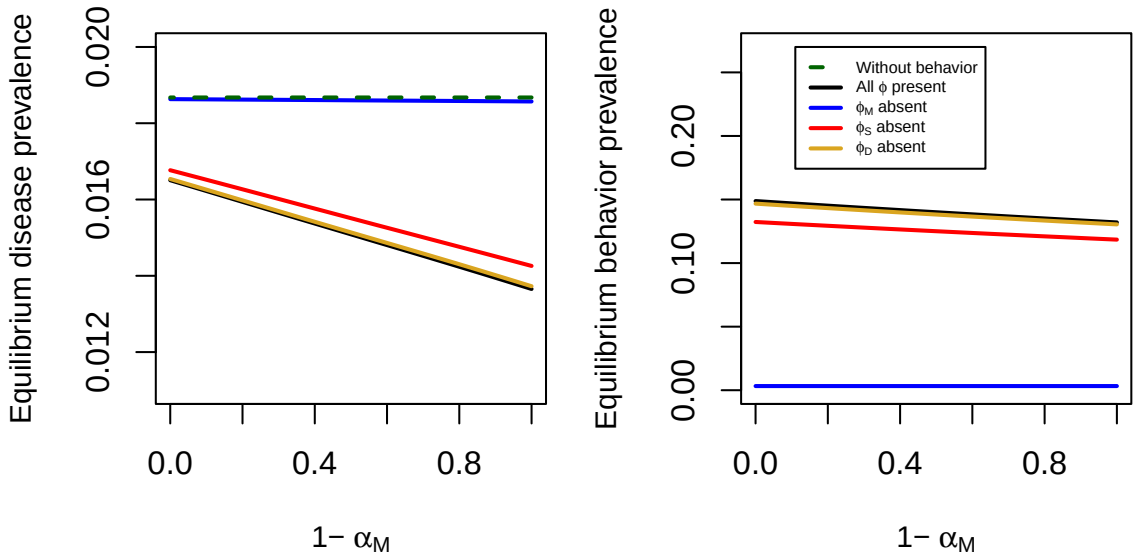

Figure 6. Equilibrium disease and behavior prevalence as the efficacy $1 - \alpha_M$ of community-level mosquito larval habitat reduction varies. Line colors indicate the behavior stimuli present or absent.

In addition to these one-at-a-time sensitivity analyses, we performed a global, variance-based sensitivity analysis of all model parameters to gain a more holistic view of the behavioral and epidemiological influences on long-term disease and behavior prevalence (Figure 7). We found that parameters directly related to disease transmission had the greatest effect on observed disease prevalence, particularly α_B, m, and γ, which refer to the relative risk of disease associated with direct protection arising from preventive behavior, the ratio of mosquitoes to humans in the population, and the disease recovery rate, respectively. While we observed that these parameters had relatively high first-order indices, which evidence a direct relationship between the parameter and disease prevalence, their notably higher total-order indices suggest that interactions with other parameters are a key driver of their influence on observed disease. Two behavioral parameters, the waning rate of behavior ω and the influence of mosquitoes on behavior ϕ_M, also have comparatively high total-order indices that again suggest involvement in higher-order interactions.

In contrast with the broad range of influences on disease prevalence, variation in behavior prevalence can be attributed almost entirely to ω, behavior waning rate, and ϕ_M, mosquito influence on behavior

uptake. Together, these two parameters are responsible for over 85% of the total variance in behavior prevalence observed. This result is consistent with our one-at-a-time analysis, for which the presence of the mosquito influence on behavior ϕ_M was required to observe non-negligible behavior outcomes (Figure 4). Further, evident model sensitivity to our choice of ϕ_M affirms our decision to assign identical values to all ϕ parameters to exert a measure of control and ensure that we are capturing the influence of behavior stimuli rather than individual behavior parameters.

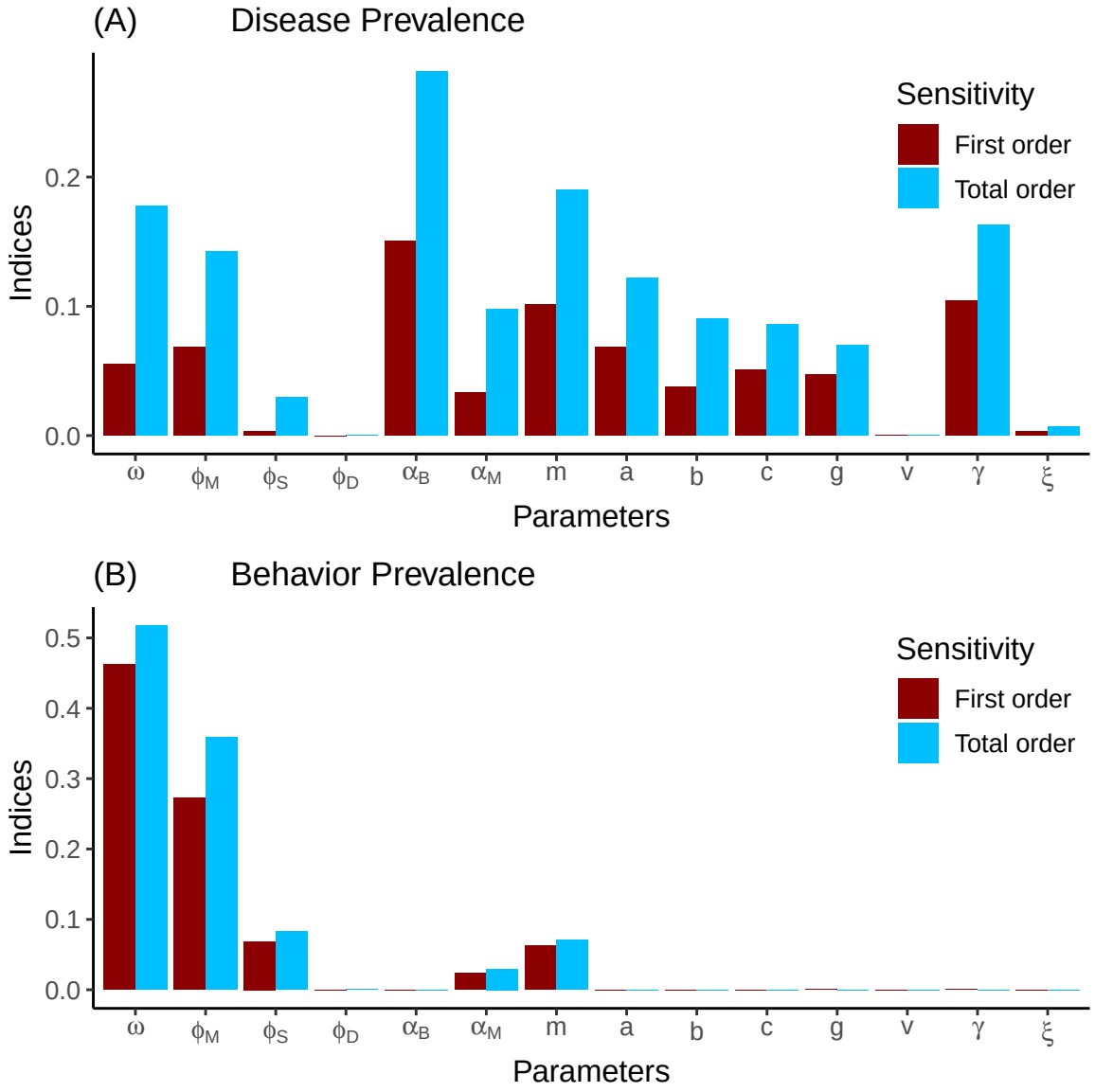

Figure 7. Variance-based sensitivity analysis of all model parameters for equilibrium disease prevalence (A) and equilibrium behavior prevalence (B). Fill colors indicate if indices are first or total order.

4. Discussion

In this study, we used a coupled contagion model of a mosquito-borne disease and mosquito preventive behaviors to gain insight into the primary drivers of feedbacks between disease and behavior for a mosquito-borne disease. We found that the coupling of disease and behavior contagions resulted in a lower equilibrium disease prevalence and a comparatively large equilibrium behavior prevalence. How long individuals maintain preventive behaviors was found to be a major driver of the equilibrium prevalence of behavior, which likewise affects the contribution of social pressure to behavior uptake. Interestingly, we found that the effects of contagion coupling on equilibrium prevalence of both contagions were most sensitive to the influence of mosquitoes in driving behavior uptake, followed by social pressure and, finally, disease itself. The relative importance of these influences reflects the frequency with which people encounter them. This finding highlights an important distinction between coupled contagion dynamics of mosquito-borne versus directly transmitted disease systems, given that mosquitoes are not a relevant influence to the latter. Another feature unique to coupled contagion dynamics for mosquito-borne disease systems is that behaviors that reduce the mosquito population can also reduce behavior uptake due to reduced contact with (and annoyance by) mosquitoes. Counterintuitively, this can result in a diminishment of the benefits derived from reducing the mosquito population, a phenomenon noted in some entomological field studies which observed that visible, community-wide insecticide spraying can lead to a decline in individual protective actions [6–8].

Beyond our study, epidemiological modeling studies that include preventive behaviors report measurable reductions in observed disease when such behaviors are introduced into the system [13,21,22,54,55]. This result is consistent with clinical trials and other field-based studies of community intervention performance [7, 56, 57]. Many of these modeling approaches include one or more parameters representing awareness of elements of the system (e.g., disease prevalence) as key drivers of participation in the behavior of interest. Increasing this awareness via a fixed parameter or via a weighting parameter for dynamic stimuli, as done here, is associated with more people engaging in behavior, regardless of specific behavioral mechanisms. At the same time, specific behavior- and disease-related outcomes are largely dependent on assumptions about stimulus strength and frequency, echoing real-world variation in dengue control behavior motivations and actions observed within and between communities [5]. In contrast with directly transmitted diseases, mosquito density adds complexity as both a stimulus and target of control, subject to both direct and indirect effects of preventive behaviors. While previous models of mosquito-borne disease and behavior do account for both community-level mosquito larval habitat reduction and individual-level personal protection, here we focus specifically on how they affect equilibrium prevalence of behavior and disease. In addition, we examined the effects of three distinct influences on the uptake of preventive behaviors. Our results reveal the consequences that even small changes to behavioral stimuli can have for equilibrium disease prevalence, highlighting the need for improved empirical understanding of these influences on the uptake of preventive behaviors.

In the absence of extensive empirical study, it can be difficult to explicitly quantify the attitudes and influences underpinning preventive behaviors. However, identifying the behavioral elements that drive effective mosquito-borne disease control — e.g., the success of interventions that incorporate community engagement and mobilization — could provide insight into a less-understood aspect of mosquito-borne disease control. Previous studies exploring community-based dengue interventions suggest that motivating factors are often time-varying [58], informed by socioeconomic and cultural

expectations [9,59], and shaped by inter- and intra-community dynamics [60,61]. However, these factors are further complicated by the bidirectional feedback between disease and behavior, which makes it difficult to disentangle the two. There is a pressing need for more intentional field study development and data collection to provide insight into these unknowns [12, 62]. With such data, future refinement of the mechanisms driving the dynamics of preventive behaviors can be advanced [54].

In this work, we relied on simplifying assumptions to hone in on behavioral processes of interest and maintain analytical tractability in the single contagion transmission models. One such assumption was the use of a static mosquito-human ratio, rather than implementing one of the many alternative mosquito population model structures available [63–65]. There are also many other plausible approaches to modeling behavior that would address aspects of the behavior-disease relationship not featured in this study. First, our model assumes that individuals engaging in preventive behaviors stop doing so at a constant rate over time. Expanding our model to allow the three behavioral stimuli to influence not just the uptake of preventive behaviors, but also their continuation, would be one way to relax this assumption. Similarly, allowing for heterogeneity in behavior uptake and efficacy could address differences within and among groups noted in clinical trials of community-based mosquito interventions, as well as possible differences in behavior participation driven by infection status [66, 67]. Lastly, our identification of mosquito biting as the primary influence on behavior uptake could be sensitive to our model's parameterization. Stimulus importance is context-dependent, though, and could be expected to vary under different conditions. For example, during an outbreak, people would likely find disease prevalence to be the most compelling influence toward control behaviors. Additionally, there could be a mismatch between the mosquitoes that influence preventive behaviors and those that pose a risk of infection [68].

Our work sought to characterize the interplay between disease and behavior in a general way for mosquito-borne diseases. We found that these diseases may be distinct from others given the wider range of stimuli that influence the uptake of preventive behaviors. We also found that there are distinct ways in which the use of personal protection and mosquito density reduction affect coupled contagion dynamics, with the latter showing an interesting feedback in which mosquito density reduction results in fewer mosquitoes, which in turn reduces the use of personal protection and contributes to more disease. In this situation, indirect protection and direct protection work against each other via changes in the prevalence of behavior, meaning that the relative efficacies of these interventions will dictate their net effect on disease in real-world systems. Likewise, empirical quantification of how the frequencies of encounters with the three behavioral stimuli we considered translate into preventive behavior uptake is an important priority for future work. Together, these findings highlight the importance of developing coupled contagion models of disease and behavior in disease systems with different transmission modes and other characteristics.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors have no conflicts of interest to declare.

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Appendix

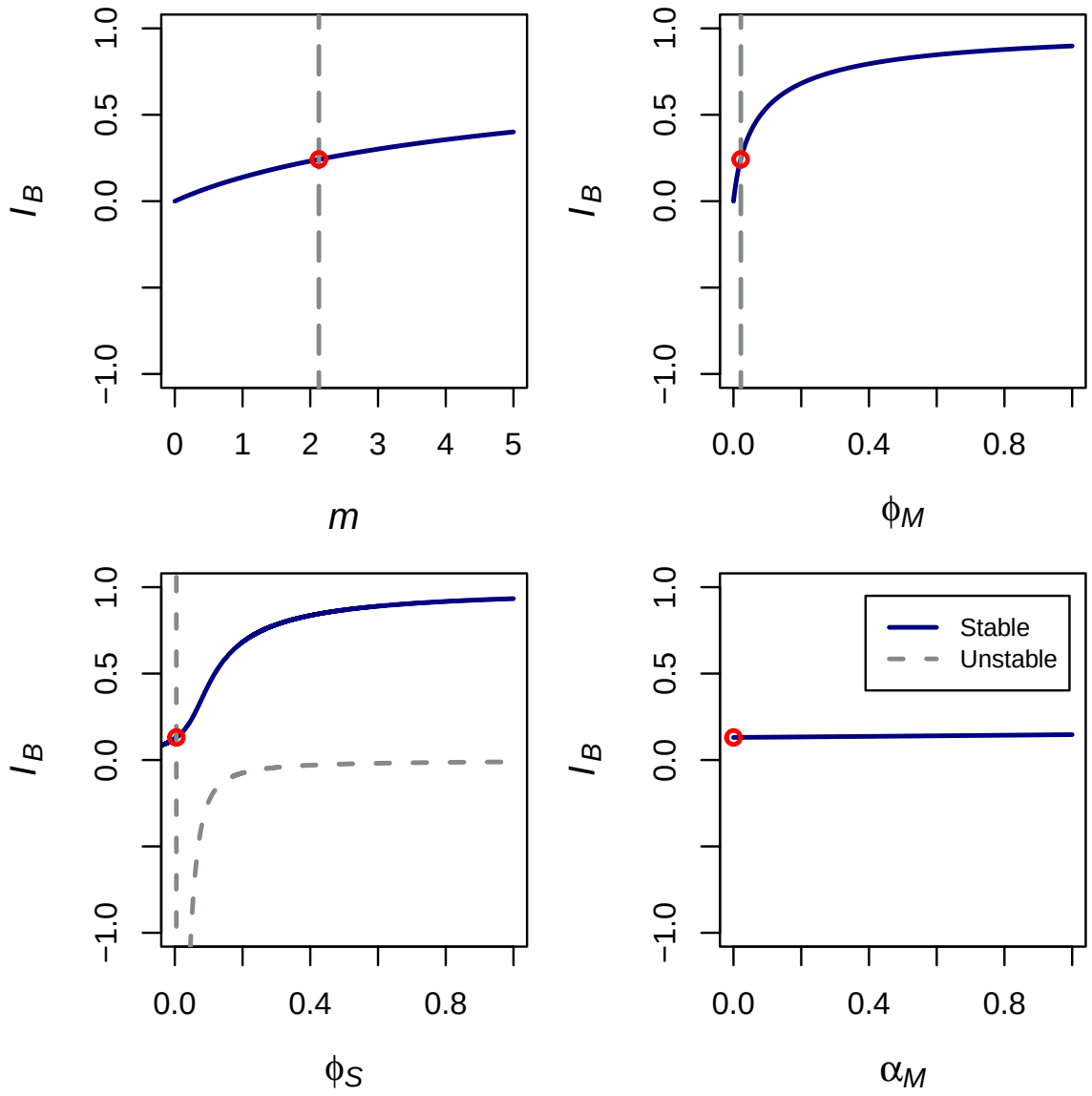

Figure A1. Equilibrium prevalence of behavior under varying behavior parameter values, for both positive, stable and negative, unstable solutions, in blue and gray, respectively. Red circles indicate plotted parameter values leading to a violation of solution conditions, when all other parameters are held at baseline values. Outcomes not valid for the population N = 1 are included for visualization purposes.

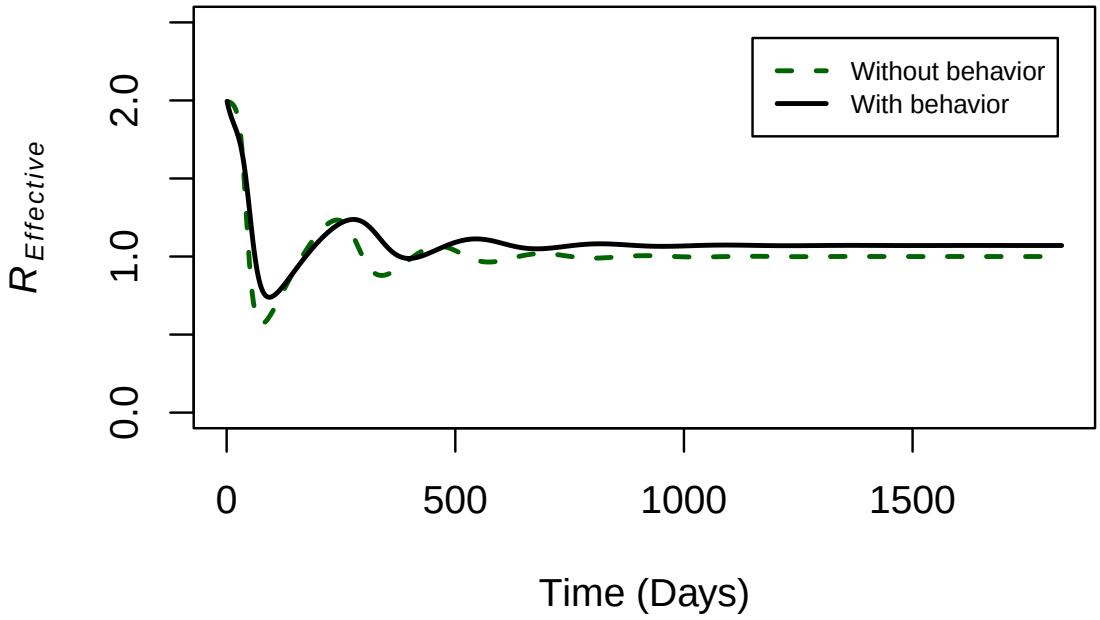

Figure A2. Effective disease reproduction number R_{Eff}, defined as the average number of new infections caused by an infected individual in a partially susceptible population, over the course of a five-year simulation. Line color and type indicate the presence or absence of the behavioral contagion.