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A generic arboviral model framework for exploring trade-offs between vector control and environmental concerns

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ABSTRACT

Effective public health measures must balance potentially conflicting demands from populations they serve. In the case of infectious disease risks from mosquito–borne infections, such as Zika virus, public concern about the pathogen may be counterbalanced by public concern about environmental contamination from chemical agents used for vector control. Here we introduce a generic framework for modeling how the spread of an infectious pathogen might lead to varying public perceptions, and therefore tolerance, of both disease risk and pesticide use. We consider how these dynamics might impact the spread of a vector-borne disease. We tailor and parameterize our model for direct application to Zika virus as spread by *Aedes aegypti* mosquitoes, though the framework itself has broad applicability to any arboviral infection. We demonstrate how public risk perception of both disease and pesticides may drastically impact the spread of a mosquito-borne disease in a susceptible population. We conclude that models hoping to inform public health decision making about how best to mitigate arboviral disease risks should explicitly consider the potential public demand for, or rejection of, chemical control of mosquito populations.

1. Introduction

The role humanity has taken as the ecosystem engineers of the environment in which we live is vast and complex. Making knowledge-based decisions to effectively influence our world necessitates the simplification of vastly interconnected systems into potentially manageable pieces. Frequently, these simplifications allow us to formulate plans in aid of targeted agendas, but then require consideration of their impact with respect to other unexplored dimensions. To be of actual help to policy makers, scientific practice must isolate and establish best practices for each desired goal for a given system, and then consider how best to balance multiple goals or constraints within the larger system without too drastically compromising those best practices. In the most challenging cases, the desired goals may be directly at odds with each other. Further complicating attempts to engineer appropriate solutions is the dynamic nature of demand for each goal over time. Public concern regarding, and therefore demand for safety from, infectious diseases led to the development of effective vaccines which were released without much concern for acceptance; in the 1950s parents in the United States were more scared of polio than of the potential side effects of a polio vaccine and there were relatively few challenges to vaccine uptake (Baicus, 2012). However, over time (and in no small part due to the success of vaccines themselves removing exposure to the experience of widespread outbreaks), the salient risks perceived by some parents began to

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shift away from fear to the observable consequences of disease outbreaks to fear of vaccines themselves. This has led to increasing rejection of vaccination programs and the recurrence of outbreaks of preventable childhood illnesses (Gangarosa et al., 1998; Dubé et al., 2015; Dredze et al., 2016; Blume, 2006). This single example highlights how differences in perception of relative risk can drive public demand for science to address its concerns in different ways.

Of course, vaccination programs focus on conflicts between two different potential health risks in direct opposition to each other (i.e. illness from the disease and side effects of the vaccine), but as with agriculture and erosion, or transportation and habitat fragmentation, efforts to manage public health risks can involve drastically different realms. Just as others have considered how public concern might impact policy interventions for vaccination (Bauch and Earn, 2004; Sharma et al., 2019; Brewer et al., 2007), in this paper, we will consider how shifting perceptions of risk may complicate the management of mosquito-borne infections in the context of concern for perceived environmental or public health risks from the application of pesticides.

As members of the public become aware of risks from the spread of newly emerging or reemerging infections there is a pattern of increased regional demand for public health agencies and policy makers to enact strategies to decrease risks of infection. One of our first and best lines of defense against dangerous vector-borne diseases such as dengue, Zika virus, and West Nile virus is to decrease the population of mosquitoes that act as the vector for transmission of infection to humans (Townson et al., 2005; Sikka et al., 2016).

Most mosquito control efforts rely on chemical treatment of the environment to decrease either survival of adult mosquitoes (i.e. via adulticides) or growth of their juveniles (i.e. via larvicides). The chemical pesticides used each have their own level of potential environmental effect beyond the targeted mosquito populations (in the case of the arboviral diseases mentioned, the target species are *Aedes albopictus, Aedes aegypti*, and *Culex pipiens*, but different vector-borne diseases involve targeting the appropriate vector and therefore incur their own insecticidal impacts). Historic control measures that targeted only mosquito populations accidentally posed direct health risks to humans (and the environment) from exposure to the chemicals used (e.g. malaria control using DDT; (Roberts et al., 1997; Hindrik et al., 2011; Henk, 2009)). Further, increased awareness of the fragility of insect biodiversity has led to public concern for non-human impacts from vector control efforts (Bonds, 2012; Bartlett et al., 2018; Fourrier et al., 2015). Although not all of these risks are associated with modern vector management strategies, especially when specifically targeted to limit transmission of Zika virus, public concern is likely to be shaped by gestalt awareness of harm from historical efforts, rather than limited to specific, exact measures (Morriss and Meiners, 2002; Gamble et al., 2010; Dhitinut Ratnapradipa PhD et al., 2015).

While these factors are already incredibly challenging, they are also embedded in a spatial and social landscape. Mosquito ecology, disease epidemiology, human population density, access to healthcare, perception of risks from outbreaks, concern for the environment, and even the municipal regions under the control of local public health, environmental protection, and vector control agencies are all affected by the features/actions of neighboring habitats and cities. Decisions are not made in isolation and the impacts from each management action can alter both the perceptions of risk and actual risk for neighboring regions. The responses by individuals may run counter to or in concert with existing regional plans. In a recent study in Ecuador, where active nationally coordinated city-level vector control operates through ultra-low volume (ULV) spraying, households were found to be spending around 10% of their income on personal abatement (including aerosols, liquid sprays, repellents, mosquito coils and unimpregnated bed nets) (Heydari et al., 2017). This points to a trade-off, or interplay between the scales at which abatement decisions are made, and the types of vector abatement used. The perception of personal agency and risk mitigation provides a backdrop to actionable decisions in the balance between personal protection against vector-borne disease and environmental contamination.

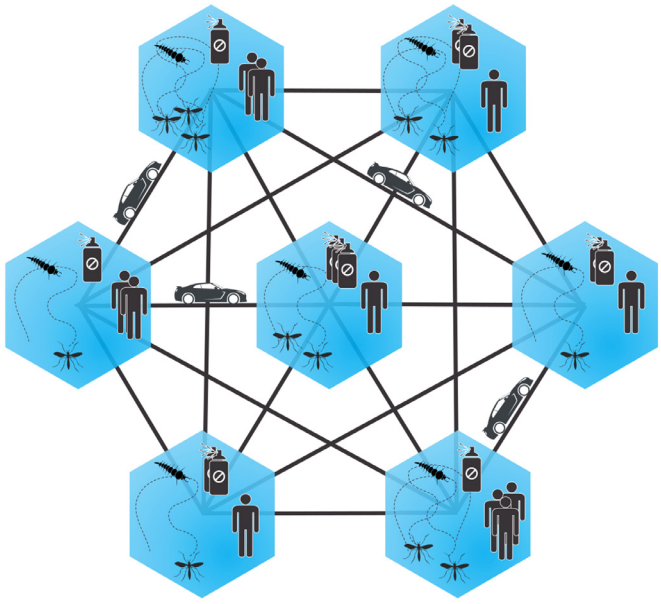


Fig. 1. Diagram. A schematic depicting elements included in our model: patches (i.e. city blocks) and their inter-connectivity, human density, juvenile and adult mosquito abundance, and application of insecticides.

We therefore consider a simplified model in which risk perception of a vector-borne disease outbreak, and therefore demand for mosquito control, is driven by a spatially explicit level of awareness of disease incidence. We incorporate a concern for environmental contamination that waxes and wanes as compared to the concern for disease mitigation. Using this model, we explore questions such as what is the effect on outbreak duration if concern is low versus high; what is the rate/pace at which abatement use increases given low or high severity of disease impact itself. In Section 2 we describe the details of the model and the meaning of each variable and parameter. We analyze the behavior of the variables as a function of time in Section 3.1, and the stationary state for different combination of parameters in Section 3.2. Final remarks can be found in Section 4.

2. Model

Our motivation is to analyze the interplay between the perceived level of threat from the infection and the willingness of the citizens to demand the usage of pesticides for vector control, to their municipalities. In order to achieve this, we developed a mathematical model that includes the most relevant aspects of this system. The model comprises N patches, each of which is occupied by a vector population (mosquitoes) and a host population (humans). A schematic of the elements in this model is depicted in Fig. 1. To get a better understanding of the model behavior, we first studied system-level dynamics of the model within an isolated patch (results in Appendix A).

In this model, the human population can be in any of the following three states: susceptible to infection by the virus (S^H), infected (I^H), or recovered (R^H), meaning that the individual can not get infected again. We also include terms that take into account

the natural birth rate (b^H) and death rate (μ^H) of people. The mosquito population is divided in the following categories: juvenile mosquitoes that are not infected and cannot transmit the virus (L^M), adult mosquitoes that are not infected with the virus but are susceptible to infection (S^M), and infected adult mosquitoes (I^M) that can transmit the pathogen by biting susceptible people. β^H and β^M are the rates at which susceptible people and mosquitoes become infected, respectively. An important characteristic of the model is the function accounting for the limited carrying capacity for larvae in each patch $f(M, K_j) = M(1 - (M/K_j))$ where K_j is the maximum carrying capacity for patch j and $M = S^M + I^M$ is the total number of adult mosquitoes that can lay eggs. The larvae transition to susceptible adult mosquitoes at rate ν. Finally, adult mosquitoes die at rate μ^M.

We can imagine that each patch represents an average city block of a US suburban area (although generalizations of the same model with more appropriate parameter choices would be applicable to other regions). Because of this, the human population is allowed to move from any patch to any other patch randomly, given that a person can travel from and to any house. The flux of people moving from patch i to patch j is expressed in the equations as the element $m_{i,j}$, where $m_{i,i} = 0$ and $\sum_{j=1}^{N} m_{i,j} = p$, where p is the probability of travelling. On the contrary, it is known that mosquitoes (and specifically *Aedes*) seldom travel large distances during their lifetimes (maximum flight range is usually reported at $50\,m$ ~ $100\,m$ but potentially much further at the extreme end of the distribution) (Reiter et al., 1995; Harrington et al., 2005). As a result, in our model the vectors can only move within the patch in which they are born, and the spread of the disease from one patch to another is determined by human mobility only. Within a patch disease is spread by infected mosquitoes which bite susceptible people. Each block has an initial population of S_j^0 humans, so the total initial population of the system is $S^0 = \sum_{j=1}^{N} S_j^0$.

A final key feature of this model is that people can demand for control methods, like adulticides and larvicides, to be applied on their own block or patch. The amount of control measures applied, in this model equivalent to the strength in the demand of the population, increases with the number of infected people in each patch. The demand for control is represented by C_j^M for adult mosquito control, and C_j^L for larval control. We assume that learning about infected individuals in the area will produce a fear of infection, which in turn will trigger the decision to demand action from the authorities. The model includes a percentage of severe outcomes that have a larger impact in the reaction to the disease than typical infected cases. On the other hand, the use of large quantities of pesticides can produce a state of fear in the population producing the opposite effect. Concern over the perceived public health risks of contaminants in the water and their own environment could discourage the demand for control measures from the authorities. Even if the pesticides utilized are innocuous for the environment, there can be negative public response out of fear of possible side effects. Though unfounded, these can still meaningfully compromise attempts to control the vectors, in this case the *Aedes aegypti* mosquito.

We propose a modified version of the Ross-Macdonald equations (Ross, 1911a; 1911b; MacDonald, 1950a; 1950b; 1957), to describe the dynamics of the spread of a vector-borne disease in a naive population, taking into account the idea of environmental concern over the use of pesticides for mosquito control, hereafter referred to as "environmental concern". Reflecting the particular case of Zika virus in *Aedes aegypti*, we construct a set of equations for each patch. So, for a given patch j we have,

$$\frac{dS_j^H}{dt} = b^H H_j - \beta^H I_j^M S_j^H - \mu^H S_j^H + \sum_i m_{ij} S_i^H - S_j^H \sum_i m_{ji} \tag{1}$$

Table 1
Initial conditions for each patch. Both susceptible populations are initialized by selecting a random number from a uniform distribution within the interval specified.

Variable	Description	Initial Value
S_j^H	Susceptible Humans	$\in$ [700; 800]
I_j^H	Infected Humans	1 in a random patch
R_j^H	Recovered Humans	0
D_j^H	Severe Outcomes	0
L_j^M	Mosquitoes Larvae	0
S_j^M	Susceptible Mosquitoes	$\in$ [1200; 1300]
I_j^M	Infected Mosquitoes	0
C_j^M	Mosquitoes control	0
C_j^L	Larvae control	0

$$\frac{dI_j^H}{dt} = \beta^H I_j^M S_j^H - rI_j^H - \mu^H I_j^H - \omega^H I_j^H + \sum_i m_{ij} I_i^H - I_j^H \sum_i m_{ji} \tag{2}$$

$$\frac{dR_j^H}{dt} = rI_j^H - \mu^H R_j^H + \sum_i m_{ij} R_i^H - R_j^H \sum_i m_{ji} \tag{3}$$

$$\frac{dD_j^H}{dt} = \delta I_j^H \tag{4}$$

$$\frac{dL_j^M}{dt} = f(\eta(S_j^M + I_j^M), K_j) - \nu L_i^M - C_j^L L_j^M \tag{5}$$

$$\frac{dS_j^M}{dt} = \nu L_i^M - \beta^M I_j^H S_j^M - \mu^M S_j^M - C_j^M S_j^M \tag{6}$$

$$\frac{dI_j^M}{dt} = \beta^M I_j^H S_j^M - \mu^M I_j^M - C_j^M I_j^M \tag{7}$$

$$\frac{dC_j^M}{dt} = \alpha^M D_j^H + \gamma^M I_j^H - \epsilon^M C_j^M \tag{8}$$

$$\frac{dC_j^L}{dt} = \alpha^L D_j^H + \gamma^L I_j^H - \epsilon^L C_j^L \tag{9}$$

Table 1 describes the variables in the model and values of the model parameters are shown in Table 2. Here, a unit of time represents one day. Note that Eqs. (1)–(4) represent the dynamics of the human population, and Eqs. (5)–(7) include the dynamics for the mosquito population. (For simplicity, we ignore the possibility of vertical transmission in the vector.) In Eqs. (8) and (9), the variables C_j^M and C_j^L indicate the amount of pesticides applied in patch j to control the adult mosquitoes and larvae populations, respectively. Both variables depend on the number of infected cases and severe outcomes inside each patch, and they decrease due to environmental concern, proportional to the amount of pesticides applied. We assume a linear dependence between the amount of pesticides applied and the environmental concern. The constant of proportionality is ϵ^M for adult mosquitoes and ϵ^L for larvicides. If these parameters are large enough, meaning that many people are reluctant to demand insecticidal control, the size of the epidemic can increase considerably.

For simplicity, we assume that $\gamma^M = \gamma^L = \gamma$, where γ represents the general strength of the control measures applied, regardless if we are considering larval or adult mosquitoes. In this investigation, we are going to make the simplified assumption that they behave in the same way, but this is done so in future models this condition can be relaxed. Similarly, we fix the values,

$$\alpha^M = \alpha^L = 100\gamma, \tag{10}$$

under the assumption that one severe outcome (e.g. such as infant microcephaly from Zika virus infection) has an effect on the

Table 2
Constant coefficients. We use the same values for each patch, except for the carrying capacities which may vary between patches. (Note: Assumed rates were chosen to reflect incidence and mortality outcomes observed in early Zika outbreaks reported in South America, however, their accuracy will not affect our results since our focus is on model scenario comparison and these rates were held constant across all scenarios.).

Parameter	Description	Value	Reference
β^H	Transmission rate for humans	1.5×10^{-4}	assumed
β^M	Transmission rate for mosquitoes	3.0×10^{-4}	assumed
μ^H	Natural death rate for humans	(8.6/1000)/365	(central, 2017)
μ^M	Natural death rate for mosquitoes	1/13	(Stone et al., 2017)
b^H	Human birth rate	(9/1000)/365	(Ellington et al., 2016)
r	Human recovery rate	0.037	(Gao et al., 2016)
ω^H	Human death rate from disease	0	assumed
δ	Composite rate	190/3, 474, 182	(Ellington et al., 2016)
ν	Maturation rate	1/7	(Stone et al., 2017)
η	Egg laying rate	10	(Stone et al., 2017)
p	Fraction of people that travel	0.2	assumed
α^M	Mosquito control motivation due to infected cases or severe outcomes	variable	
α^L	Larvae control motivation due to infected cases or severe outcomes	variable	
ϵ^M	Environmental concern demotivates mosquito pesticides usage	variable	
ϵ^L	Environmental concern demotivates larvae pesticide usage	variable	
K_j	Larvae carrying capacity for each patch	variable	

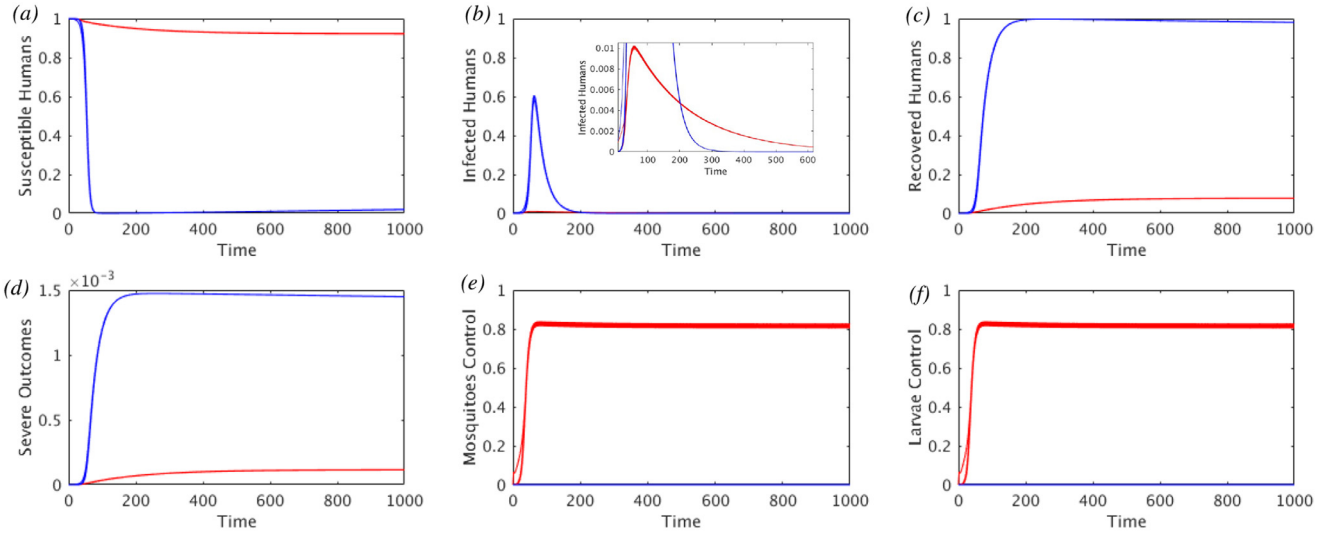


Fig. 2. *Scenario with low environmental concern and high control strength.* (Colour online) Example of a combination of parameters that leads to a small outbreak of the disease, where the environmental concern levels are low and the demand for control measures is high. Parameters: $N = 100$; $p = 0.2$; environmental concern: $\epsilon^M = \epsilon^L = \epsilon = 10$; control strength due to infected cases: $\gamma^M = \gamma^L = \gamma = 0.95$; control strength due to severe outcomes: $\alpha^M = \alpha^L = 100\gamma$. Each blue line represents the results obtained for the ground case, and each red line corresponds to the results for this particular combination of parameters γ and ϵ. A line for each patch has been plotted; however, most of the lines overlap. Inset in panel (b) is a zoom in the region where the red lines are significant.

population's perception of threat similar to a hundred regular infected cases. Finally, $\epsilon^M = \epsilon^L = \epsilon$ represents the general environmental concern. After these assumptions, Eqs. (8) and (9) can be rewritten as,

$$\frac{dC_j^M}{dt} = \gamma(100D_j^H + I_j^H) - \epsilon C_j^M \tag{8*}$$

$$\frac{dC_j^L}{dt} = \gamma(100D_j^H + I_j^H) - \epsilon C_j^L. \tag{9*}$$

We can now use γ and ϵ as the main parameters to describe and analyze a number of different possible scenarios. Note that Eqs. (8*) and (9*), are simply special cases of Eqs. (8) and (9), and we will solve the original equations, for this specific combination of parameters.

3. Results

3.1. Dynamics of the model

We solve the system of differential Eqs. (1)–(9) using an explicit Runge–Kutta (4,5) formula, based on the Dormand-Prince pair, detailed in Shampine and Reichelt (1997).

The simplest choice of combination of parameters is the case where no control measures are applied and, because of that, the results are independent of the level of environmental concern in the population. This will be our ground case and it will be used as a benchmark against which to compare all other combinations of parameters. In Figs. 2–4, each blue line represents the results obtained for the ground case, and each red line corresponds to the results with a combination of non-zero parameters γ and ϵ. A line for each patch has been plotted; however, because the dynamic of the epidemic are very similar in each patch, most of the lines overlap. In Fig. 2 we show the results for a combination of parameters with low environmental concern and high demand for control. Under this conditions the epidemic only spans over a small fraction of the population (less than 10%). Each plot shows the temporal dependence of one variable on the system of Eqs. (1)–(9). In this case we set model parameters to reflect a strong motivation to demand control measures $\gamma = 0.95$, and low environmental concern $\epsilon = 10$. Under these conditions, approximately only 10% of the population gets infected, although the outbreak persists for 2.4 times longer than the ground case (see inset of Fig. 2(*b*)). We see that, once the control measure begins, it increases rapidly and stays high un-

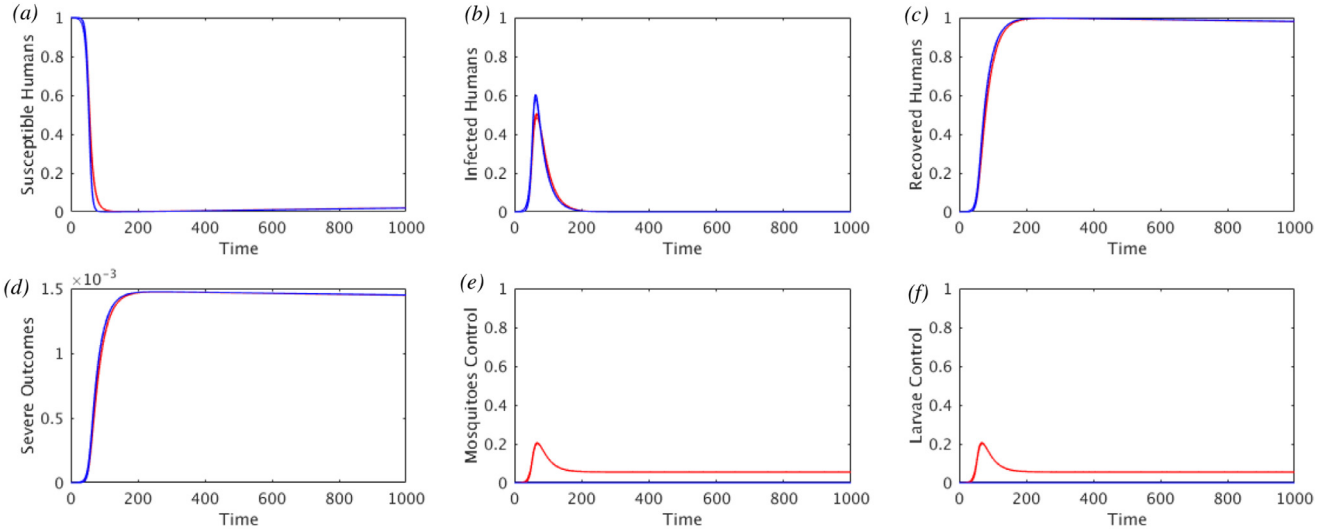


Fig. 3. *Scenario with high environmental concern and low control strength. (Colour online)* Example of a combination of parameters that leads to an epidemic outcome of the disease, where the environmental concern levels are high and the demand for control measures is low. Parameters: $N = 100$; $p = 0.2$; environmental concern: $\epsilon^M = \epsilon^L = \epsilon = 200$; control strength due to infected cases: $\gamma^M = \gamma^L = \gamma = 0.1$; control strength due to severe outcomes: $\alpha^M = \alpha^L = 100\gamma$. Each blue line represents the results obtained for the ground case, and each red line corresponds to the results for this particular combination of parameters γ and ϵ. A line for each patch has been plotted; however, most of the lines overlap.

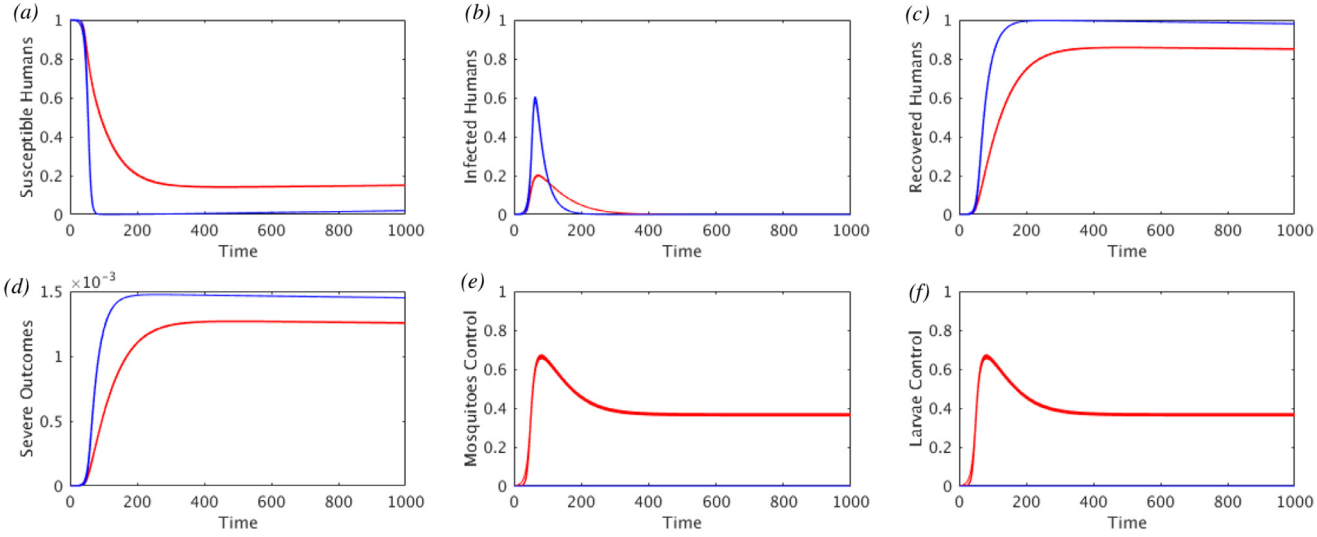


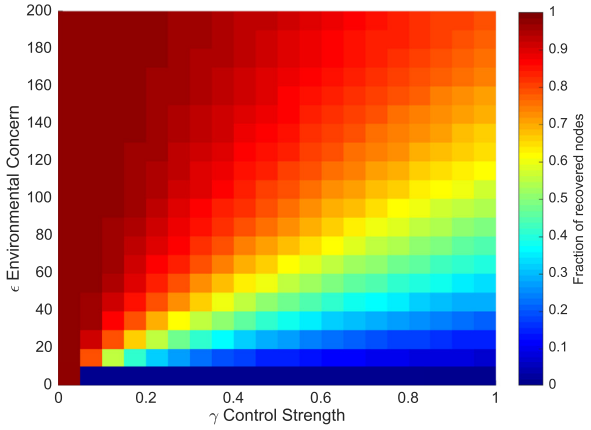
Fig. 4. *Scenario with intermediate values of environmental concern and control strength.* (Colour online) Example of a combination of parameters that leads to an epidemic outcome of the disease, where the environmental concern levels and the demand for control measures take intermediate values. Parameters: $N = 100$; $p = 0.2$; environmental concern: $\epsilon^M = \epsilon^L = \epsilon = 130$; control strength due to infected cases: $\gamma^M = \gamma^L = \gamma = 0.5$; control strength due to severe outcomes: $\alpha^M = \alpha^L = 100\gamma$. Each blue line represents the results obtained for the ground case, and each red line corresponds to the results for this particular combination of parameters γ and ϵ. A line for each patch has been plotted; however, most of the lines overlap.

til the end of the simulation, since there is no reason to decrease pesticide usage.

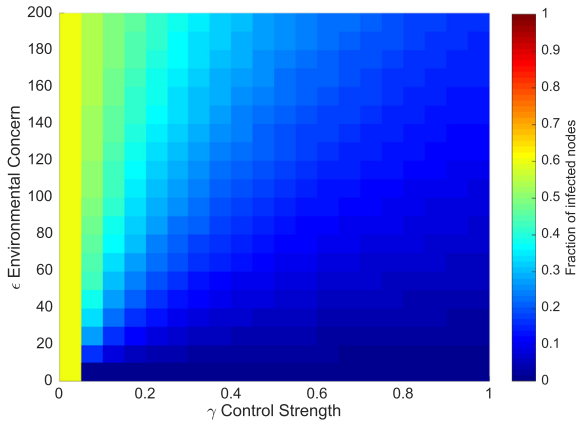
On the contrary, if we select a low motivation to demand pesticides, and a stronger environmental concern, we find that the control measures are not sufficient to stop the outbreak (see Fig. 3). By the end of the simulation 100% of the population is affected, because the efforts made to control the vector population were insufficient. Under these conditions, the dynamic of the outbreak is very similar to the one obtained from the ground case. The peak in applied control measures corresponds to the peak in infected individuals. However, the residual impact on perceived risk from severe outcomes continues to influence the demand for control even when the numbers of infections decrease.

There is also an interesting middle ground in which both parameters take intermediate values. As shown in Fig. 4, the fraction of recovered people at the end of the simulation is quite large (over 80%), but the fraction of infected individuals remains lower than the ground case (the peak of infected individuals is only 1/3 of the control case, Fig. 4(b)). The duration of the outbreak in this intermediate case is 1.8 times longer than the ground case. Even though the final number of recovered individuals is high (over 80%), this scenario may be considered preferable from a public health standpoint. Compared to the case shown in Fig. 3, which also affects the entire population, there are fewer simultaneous infected cases. This could allow municipal authorities more time to take preventive measures and ensure medical infrastructure is not overwhelmed by the number of simultaneous active cases.

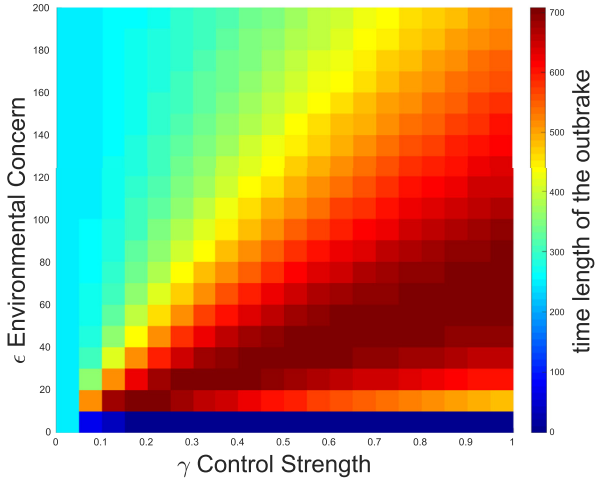
In Eqs. (8*) and (9*) we assumed that one severe outcome has an impact on the population's perception of threat similar to a hundred infected cases, see Eq. (10). Of course, this is an estimation of average risk perception across the population. In reality, risk perception will likely be both different for every person and difficult to measure. The effect of this parameter can be seen in panels (b), (e) and (f), of Figs. 2–4. In those plots we can clearly



(a) Fraction of recovered individuals at the end of the simulation.



(b) Maximum fraction of infected individuals reached during the simulation.



(c) Time duration of the outbreak.

Fig. 5. *Phase space exploring the trade off between environmental concern and control strength.* $N = 100$; $p = 0.2$ (fraction of human mobility). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

distinguish two different behaviors: at the beginning of the simulation, the demand for control measures increases proportionally to the number of infected cases; when the number of infected people reaches its maximum, we see a concomitant maximum in the strength of control measures. After the number of infected people goes back to zero, the cumulative number of severe outcomes remains constant, given that there are no new cases and therefore the strength of control measures applied also remains constant (since it is proportional to the number of severe cases). If we modify the relative effect that one severe outcome has on the perception of risk compared to the number of infected cases in Eq. (10), this will change the final constant value of the control strength, after the peak number of infected cases has passed.

3.2. Interplay between vector control and environmental concern

We can explore the complete space of (γ, ϵ) parameters and plot the fraction of recovered people at the end of the simulation to assess the size of the outbreak. In Fig. 5 we see how different combinations of these two parameters lead to very different outcomes. In Fig. 5(a) we show the fraction of individuals in the recovered state, at the end of the simulation. For a fixed value of the control strength γ, the fraction of recovered individuals increases when the environmental concern ϵ increases. In contrast, for a fixed value of the environmental concern, the size of the outbreak decreases when the control strength increases. Finally, we can see that if both parameters, γ and ϵ, are increased proportionally the fraction of recovered people remains constant.

The left column of Fig. 5(a) represents the state of the system without any control measures, and thus, there is no environmental concern about the use of pesticides. If we move horizontally in Fig. 5(a) increasing the strength of the control measures with no environmental concern (bottom row) we see that the outbreak can be stopped, even for small values of γ. This is due to the fact that the variables that represent the control measures C_j^M and C_j^L are monotonically increasing functions when $\epsilon = 0$. As we increase the environmental concern, the size of the epidemic becomes larger because the strength of control measures decreases when the number of infected cases is lower. When this is the case, it reflects that people are more concerned about exposure to pesticides than exposure to infection. We can think of this plot as the number of cases averted, given that in the ground case the fraction of infected people is 1. It is easy to estimate the fraction of people who did not get infected in scenarios with any other combination of parameters.

In Fig. 5(b) we can see the maximum fraction of infected individuals reached, i.e. the maximum value recorded for simultaneous infected individuals at any time in the simulation. We can measure the values in this plot by finding the maximum value of the

curves on plots Figs. 2(*b*), 3(*b*) or 4(*b*), for the whole space parameter. Limiting this quantity may be important due to throughput capacity for provision of adequate medical care. Fig. 5(c) shows the duration of the outbreak (in days) for different combination of parameters. It is clear from this plot that increasing control measures and decreasing environmental concern creates outbreaks of longer duration. When combining these results, we see the importance of keeping the environmental concern low. From Fig. 5(b), we see that when the environmental concern increases, the number of simultaneous infected people increases. From Fig. 5(a) we see that, when this happens, the faction of recovered individuals is higher, and from Fig. 5(c) we see that the outbreak is of shorter duration, creating a scenario with a large number of infected people at the same time, and limiting the time available to react.

4. Conclusion

In this paper we presented a mathematical model that extends standard epidemiological models for vector-borne disease dynamics to consider both the public demand for protection from mosquito-borne illness, and a simultaneous public concern about the environmental impacts of that protection. While still a highly simplified model, we demonstrated how public risk perception may impact the spread of a mosquito-borne disease in a naive population. Critically, our model provides a method for estimating the public health costs (i.e. the additional preventable cases of disease) associated with increased strength of public environmental concern (i.e. resistance to mosquito control efforts), even though the observed disease incidence was assumed to reinforce the public perception of health risks from infection, itself leading to demand for vector control. Although this study falls far short of providing all of the answers needed by policy makers, it provides a meaningful advance in consideration of the practical constraints in garnering public acceptance of vector-control interventions to mitigate the risks of outbreaks. Our hope is that these models may help guide planning and investment in public outreach and education campaigns that address not only the relevant infection risks, but also the risks from vector control itself.

There are, of course, many additional features of potential importance to public risk perception that our model does not explicitly address. For example, how the public is informed of the risks from exposure to both infectious pathogen and chemical pesticides may meaningfully shape the perception of these relative risks. This may in turn have a drastic impact on demand for, or rejection of, mosquito control strategies. Similarly, our model included direct observation of severe outcomes from infection but did not include explicit, observable negative outcomes to pesticide exposure. Inclusion of such effects in future studies will help shape explicit recommendations for policy makers who must address public concern about the methods used for mitigation of arboviral risks while still achieving effective outbreak control.

Author's contributions

All authors contributed equally to the design of the study, interpretation of the results, and preparation of the manuscript. GPS was responsible for model implementation and analysis. NF proposed, and secured funding for, the research. All authors have approved the final version of the manuscript.

Availability of data and material

Data sharing is not applicable to this article as no datasets were generated or analysed during the current study.

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Role of the funding source

The funding source had no involvement in the study design, in the collection, analysis and interpretation of data, in the writing of the report, or in the decision to submit this article for publication.

Declaration of Competing Interest

All authors contributed equally to the design of the study, interpretation of the results, and preparation of the manuscript. GPS was responsible for model implementation and analysis. NF proposed, and secured funding for, the research. All authors have approved the final version of the manuscript.

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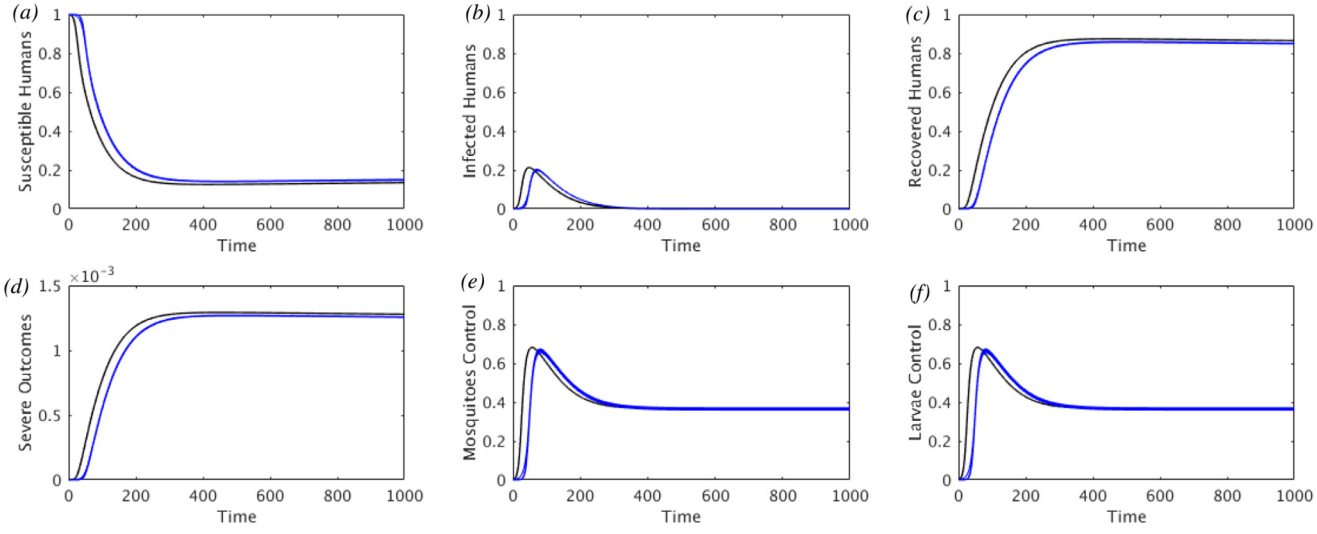

Fig. A.6. *One isolated patch.* Black line is the solution to Eqs. (1) to (9) for $N = 1$ patch. The results from Fig. 4 are included in blue for easy comparison. Parameters: environmental concern: $\epsilon^M = \epsilon^L = \epsilon = 130$; control strength due to infected cases: $\gamma^M = \gamma^L = \gamma = 0.5$; control strength due to severe outcomes: $\alpha^M = \alpha^L = 100\gamma$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

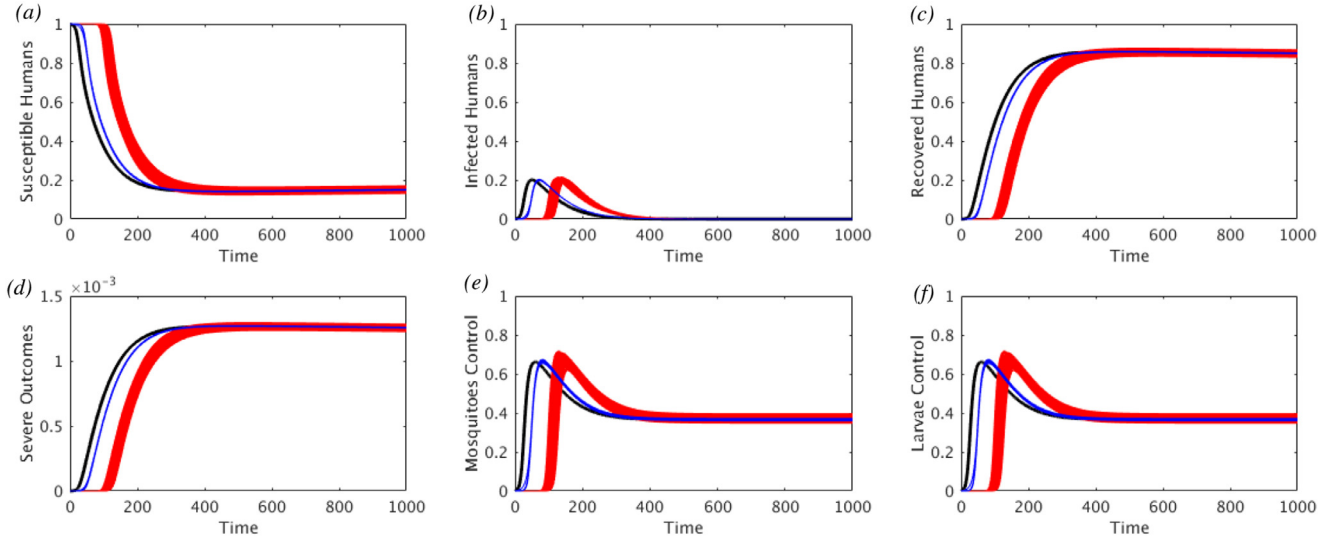

Fig. A.7. *Very limited mobility.* Solution to Eqs. (1) to (9) for $N = 100$ patches and fraction of people allowed to move from one path to another $p = 10^{-7}$. The black lines represent the results for the patch with the first infected patient. With a rate $p = 10^{-7}$ people can move to any other patch. The red lines represent the results for all the other patches. Finally, the results from Fig. 4 are included in blue for easy comparison. Parameters: environmental concern: $\epsilon^M = \epsilon^L = \epsilon = 130$; control strength due to infected cases: $\gamma^M = \gamma^L = \gamma = 0.5$; control strength due to severe outcomes: $\alpha^M = \alpha^L = 100\gamma$. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Appendix A. Model behavior for an isolated patch and very low mobility

For comparison, we include the outcome of our model for the case of a single isolated patch. In Fig. A.6 we can see the result of solving Eq. (1) to Eq. (9), using the same combination of parameters in Fig. 4 for the case of one patch. The black line is the result obtained for one isolated patch, the blue lines correspond to the results showed in Fig. 4. The general trend is similar to the one found using a fully connected network of $N = 100$ patches. Even though there is a shift in the time dependence, the final levels of infected and recovered people remains the same.

Also we compare the case with very limited mobility between patches, i.e. $p \to 0$. In this conditions, it takes more time for the disease to get to a neighboring patch. However, because all the patches are one step away from each other, the outbreak can still reach all other patches at the same time (see Fig. A.7). The black line corresponds to the results obtained for the patch with the first infected person. With a very small rate $p = 10^{-7}$, people can travel to neighboring patches. The red lines are the results obtained for all other patches. Once all the patches have infected individuals, the dynamics are similar to those when using larger values of p, but shifted in time. he results from Fig. 4, with $p = .2$, were included for comparison.

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