

Optimal voluntary and mandatory insect repellent usage and emigration strategies to control the chikungunya outbreak on Reunion Island

Sylvia RM Klein ¹, Alex O Foster ², David A Feagins ³, Jonathan T Rowell ⁴, Igor V Erovenko ^{Corresp. 4}

¹ Department of Mathematics, St. Mary's College of Maryland, St. Mary's City, Maryland, USA

² Department of Mathematics and Statistics, Coastal Carolina University, Conway, South Carolina, USA

³ Department of Mathematics, St. Mary's University, San Antonio, Texas, USA

⁴ Department of Mathematics and Statistics, University of North Carolina at Greensboro, Greensboro, NC, United States

Corresponding Author: Igor V Erovenko
Email address: igor@uncg.edu

In 2005, a chikungunya virus outbreak devastated the tropical island of Reunion, infecting a third of the total population. Motivated by the Reunion Island case study, we investigate the potential for two intervention measures under both voluntary and mandatory protocols to control a vector-borne disease when there is risk of the disease becoming endemic. The first measure uses insect repellent to prevent mosquito bites, while the second involves emigrating to the neighboring Mauritius Island to avoid infection. There is a threshold on the cost of using repellent above which both voluntary and mandatory regimes find it optimal to forgo usage. Below that threshold, mandatory usage protocols will eradicate the disease; however, voluntary adoption leaves the disease at a small endemic level. Emigrating from the island to avoid infection results in a tragedy-of-the-commons effect: while being potentially beneficial to specific susceptible individuals, the remaining islanders paradoxically face a higher risk of infection. Mandated relocation of susceptible individuals away from the epidemic is viable only if the cost of this relocation is several magnitudes lower than the cost of infection. Since this assumption is unlikely to hold for chikungunya, it is optimal to discourage such emigration for the benefit of the entire population.

1 **OPTIMAL VOLUNTARY AND MANDATORY INSECT REPELLENT**
 2 **USAGE AND EMIGRATION STRATEGIES TO CONTROL THE**
 3 **CHIKUNGUNYA OUTBREAK ON REUNION ISLAND**

4 SYLVIA R. M. KLEIN¹, ALEX O. FOSTER², DAVID A. FEAGINS³, JONATHAN T. ROWELL⁴,
 5 AND IGOR V. EROVENKO^{*4}

ABSTRACT. In 2005, a chikungunya virus outbreak devastated the tropical island of Reunion, infecting a third of the total population. Motivated by the Reunion Island case study, we investigate the potential for two intervention measures under both voluntary and mandatory protocols to control a vector-borne disease when there is risk of the disease becoming endemic. The first measure uses insect repellent to prevent mosquito bites, while the second involves emigrating to the neighboring Mauritius Island to avoid infection. There is a threshold on the cost of using repellent above which both voluntary and mandatory regimes find it optimal to forgo usage. Below that threshold, mandatory usage protocols will eradicate the disease; however, voluntary adoption leaves the disease at a small endemic level. Emigrating from the island to avoid infection results in a tragedy-of-the-commons effect: while being potentially beneficial to specific susceptible individuals, the remaining islanders paradoxically face a higher risk of infection. Mandated relocation of susceptible individuals away from the epidemic is viable only if the cost of this relocation is several magnitudes lower than the cost of infection. Since this assumption is unlikely to hold for chikungunya, it is optimal to discourage such emigration for the benefit of the entire population.

6 1. INTRODUCTION

7 Reunion Island is a tropical island located in the Indian Ocean 500 miles
 8 east of Madagascar and approximately 150 miles southwest of Mauritius. The
 9 island was devastated by a major chikungunya outbreak in 2005–2006, when
 10 approximately 266 thousand of the 785 thousand inhabitants were infected,

¹DEPARTMENT OF MATHEMATICS, ST. MARY'S COLLEGE OF MARYLAND, ST. MARY'S CITY, MD 20686, USA

²DEPARTMENT OF MATHEMATICS AND STATISTICS, COASTAL CAROLINA UNIVERSITY, CONWAY, SC 29528, USA

³DEPARTMENT OF MATHEMATICS, ST. MARY'S UNIVERSITY, SAN ANTONIO, TX 78228, USA

⁴DEPARTMENT OF MATHEMATICS AND STATISTICS, UNIVERSITY OF NORTH CAROLINA AT GREENSBORO, GREENSBORO, NC 27402, USA

Key words and phrases. Chikungunya, epidemiology, game theory, herd immunity, Nash equilibrium, Reunion Island.

*Corresponding author: igor@uncg.edu.

causing over 200 deaths [40]. In the aftermath of that outbreak, the chikungunya virus spread from Africa to Europe, USA, and Australia, and although the incidence levels of this disease remain low, its potential to cause future outbreaks in these areas is cause for concern. In this paper, we investigate the viability of voluntary participation in personal protective measures (mosquito repellent and emigration) against diseases like chikungunya on Reunion Island by constructing a game-theoretic model in which individual strategic payoffs are compared against the average population payoff.

Chikungunya virus (CHIKV) is an *Alphavirus* in the *Togaviridae* family, similar to Dengue fever and Zika virus [20, 25, 24]. It is a vector-borne virus spread through bites by the females of *Aedes aegypti* and *Aedes albopictus* mosquitoes. After a bite, there is a latency period for both humans and mosquitoes: it can take between 2 to 6 days for symptoms to develop and for an individual to become infectious [15]. The major symptoms associated with CHIKV are fever, rash, arthritis, headache, and nausea [15]. The defining characteristic of CHIKV is the persistence of arthritis for years after the initial infection [20]. A small percentage of people infected with CHIKV, however, never develop symptoms of the disease [12]. Humans are no longer infectious about a week and a half after the initial infection, but may still be symptomatic. Recovered individuals acquire lifelong immunity from future infections [9]. There is no vaccine to prevent or medicine to treat chikungunya virus [9]. The most effective way to prevent infection from CHIKV is to prevent mosquito bites, for example, by using insect repellent [8].

Chikungunya was first isolated in 1952–1953 in Tanzania [27]. The name translates to the native term for “that which bends up” [30]. There were limited outbreaks between the initial discovery of the disease and a worldwide outbreak that occurred in 2004–2005 [12]. This outbreak started in Kenya and spread to the surrounding islands including Mauritius, Rodrigues, The Seychelles, Mayotte, Madagascar and Reunion Island [26]. From these islands, it spread to other regions of the world—chikungunya virus is now present on every continent except Antarctica—most likely carried by tourists. The disease impacted Reunion Island most severely: a third of the population became infected, unusually severe forms were present, and the first occurrences of maternal-neonatal transmission were documented [4]. This severity of impact may be attributed to an increase in travel between islands and the climate of the region at the time of the epidemic [4, 35].

While the severity of the Reunion chikungunya outbreak may seem like an isolated event, vector-borne diseases such as malaria and dengue are becoming an increasingly prevalent public health issue in today’s society. In the United States there has been a 23-fold increase of vector-borne disease cases

51 in the past ten years [28]. There are now 16 vector-borne diseases widely dis-
52 tributed in the United States, all of which are resistant to control, and only one
53 of these (Yellow Fever) has an FDA-approved vaccine [28]. Even though there
54 are limited cases of CHIKV in the United States and its territories, the disease
55 is becoming more persistent: the number of national cases and distribution are
56 increasing, and the range of the mosquitoes that transmit CHIKV has spread
57 to 38 states as of 2016 [28].

58 Due to the transmission patterns of mosquito-borne diseases and the lack
59 of sufficient vector control to eradicate such diseases, individuals often have
60 to rely on voluntary participation in personal protection measures. Unfortu-
61 nately, individual self-interest in protection against an infectious disease does
62 not necessarily correspond to the desired outcome for society [16], namely
63 eradication of the disease. The effect of potentially selfish human behavior on
64 the spread of infectious diseases has only recently begun to receive atten-
65 tion, forming a new field of *behavioral epidemiology*; see [21] for a review of
66 behavioral epidemiology.

67 Originally designed for the field of economics [38], game theory has since
68 been used to model many biological phenomena [23, 18, 11, 37, 6], including
69 individual-level vaccination decisions [2]. In a vaccination game, a selfish indi-
70 vidual seeks to maximize its benefit, or rather to minimize the potential loss re-
71 sulting from either employing a potentially costly protective measure or facing
72 the consequences of the disease. As the likelihood of contracting the disease is
73 dependent upon the behavior of others within the at-risk population, the re-
74 sulting strategic interactions between individuals can be modeled using game
75 theory. Game-theoretic frameworks have been adopted to studying optimal
76 individual vaccination strategies for smallpox [3], influenza [16, 31], measles
77 [32], rubella [33], toxoplasmosis [34], Ebola [5], cholera [19], and meningitis
78 [22]. It has also been applied to other personal protective measures such as
79 insecticide-treated cattle [10], mosquito repellent [13], insecticide-treated bed
80 nets [7], clean water [19], and clean injecting equipment [29]. For an extensive
81 review of behavior-linked vaccination models, see [39].

82 In this paper, we investigate the potential effects of voluntary and government-
83 mandated participation in utilizing the insect repellent as a protective measure
84 against a disease such as chikungunya on Reunion Island. We also analyze
85 the effect of emigration to a neighboring island (Mauritius) on the spread of
86 chikungunya among the remaining population of Reunion Island. We find that
87 the latter protocol has a paradoxically worsening of outcomes for the non-
88 participating population.

2. METHODS

We adopt a version of the epidemiological model of the chikungunya outbreak on Reunion Island by Yakob and Clements [40] by adding population dynamics (birth and death demographic processes) for both humans and mosquitoes and strategically-linked parameters so that the disease potentially may establish itself endemically. This assumption then permits us to use the framework of Dorsett et al. [13], Amaku [1], and Bauch and Earn [2]. All human inhabitants of the island (N) are divided into 5 compartments: individuals susceptible to chikungunya (S); exposed individuals (E), who had been bitten by an infected mosquito and acquired the disease; symptomatic infectious individuals (I), who developed the symptoms of the disease and became infectious to biting mosquitoes; asymptomatic infectious individuals (I_a), who became infectious but did not develop symptoms; and recovered individuals (R), who recovered from chikungunya and acquired immunity. The mosquito population is divided into 3 compartments: susceptible mosquitoes (X); exposed mosquitoes (Y), who bit an infected human and were exposed to the pathogen; and infectious mosquitoes (Z), who may infect humans by biting susceptible individuals. We did not consider in this model the full life-cycle of mosquitoes, such as egg and larval stages, because we did not incorporate mosquito population control as one of the measures to fight chikungunya.

New individuals enter the susceptible part of the population at a rate Λ_1 due to birth or immigration; there is a natural per capita human mortality μ_1 . Similarly, new mosquitoes are recruited into the susceptible compartment at a rate Λ_2 , and there is a natural per capita mosquito mortality μ_2 . We disregard the human disease-induced mortality because it is low, and doing so allows us to compute endemic equilibria analytically.

Susceptible humans who are bitten by infectious mosquitoes become exposed. The force of infection f_1 , which is the rate at which susceptible individuals move to the exposed class, depends on the density of susceptible humans S/N (i.e., the probability that an infectious mosquito bites a susceptible individual), the number of infectious mosquitoes Z , and the mosquito-to-human transmission coefficient β_1 . Similarly, susceptible mosquitoes who bite infectious humans become exposed. The force of infection f_2 , which is the rate at which susceptible mosquitoes move to the exposed compartment, depends on the density of infectious humans $(I + I_a)/N$ (i.e., the probability that a mosquito bites an infectious individual), the number of susceptible mosquitoes X , and the human-to-mosquito transmission coefficient β_2 .

Exposed humans become infectious (after a latency period) at a rate λ_1 ; a proportion ϕ of infectious individuals develop symptoms of the disease. Exposed mosquitoes become infectious at a rate λ_2 .

129 Infectious humans (both symptomatic and asymptomatic) recover at a rate
 130 γ and acquire immunity from future infections. The lifespan of a mosquito is
 131 too short to recover; an infectious mosquito remains as such until it dies.

132 Figure 1 shows a diagram for the chikungunya transmission model on Re-
 133 union Island. The parameters of the epidemiological model are summarized in
 134 table 1. The table includes the baseline value of the mosquito-to-human trans-
 135 mission parameter, denoted by β_1^0 . Later this parameter will be affected by an
 136 intervention measure (insect repellent), and hence it will become a function of
 137 the level of insect repellent usage, given by (7).

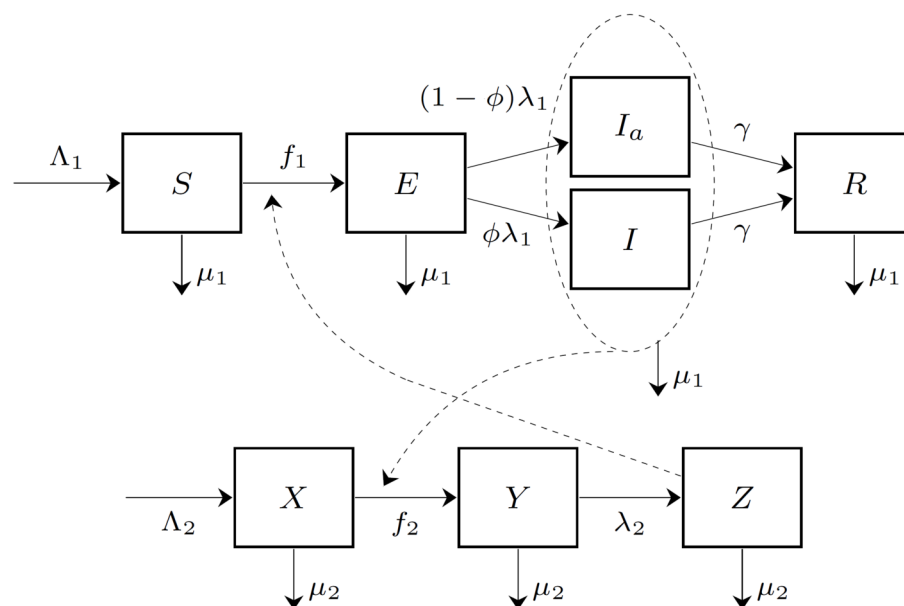


Figure 1. The compartment model of chikungunya virus transmission on Reunion Island. The human population is divided into five compartments: susceptible (S), exposed (E), symptomatic infectious (I), asymptomatic infectious (I_a), and recovered (R). The mosquito population is divided into three compartments: susceptible (X), exposed (Y), and infectious (Z). The forces of infection on human and mosquito populations, f_1 and f_2 , respectively, are population frequency-dependent functions of the state variables.

Table 1. Summary of the model parameters

Symbol	Meaning	Value	Source
β_1^0	Mosquito-to-human transmission	0.37	[14]
β_2	Human-to-mosquito transmission	0.37	[14]
γ	Human recovery rate	0.14	[40]
Λ_1	Human birth rate	3.58	Assumed
Λ_2	Mosquito birth rate	4.76×10^3	Assumed
λ_1	Rate of humans becoming infectious	0.5	[40]
λ_2	Rate of mosquitoes becoming infectious	0.5	[40]
μ_1	Human natural death rate	3.58×10^{-5}	Assumed
μ_2	Mosquito natural death rate	0.05	[40]
ϕ	Proportion of hosts that develop symptoms	0.97	[40]

138 The dynamics of the compartment model in figure 1 is described by the
 139 following system of differential equations:

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda_1 - \frac{\beta_1 SZ}{N} - \mu_1 S, \\
 \frac{dE}{dt} &= \frac{\beta_1 SZ}{N} - \lambda_1 E - \mu_1 E, \\
 \frac{dI}{dt} &= \phi \lambda_1 E - \gamma I - \mu_1 I, \\
 \frac{dI_a}{dt} &= (1 - \phi) \lambda_1 E - \gamma I_a - \mu_1 I_a, \\
 \frac{dR}{dt} &= \gamma(I + I_a) - \mu_1 R, \\
 \frac{dX}{dt} &= \Lambda_2 - \frac{\beta_2 X(I + I_a)}{N} - \mu_2 X, \\
 \frac{dY}{dt} &= \frac{\beta_2 X(I + I_a)}{N} - \lambda_2 Y - \mu_2 Y, \text{ and} \\
 \frac{dZ}{dt} &= \lambda_2 Y - \mu_2 Z.
 \end{aligned} \tag{1}$$

140 The disease-free equilibrium (DFE) of this system is given by

$$(S^0, E^0, I^0, I_a^0, R^0, X^0, Y^0, Z^0) = \left(\frac{\Lambda_1}{\mu_1}, 0, 0, 0, 0, \frac{\Lambda_2}{\mu_2}, 0, 0 \right). \tag{2}$$

141 To compute the basic reproduction number R_0 , we use the next-generation
 142 matrix approach [36]. To simplify this computation, we temporarily combined
 143 the symptomatic and asymptomatic infectious compartments into one infec-
 144 tious compartment $I + I_a$: individuals in both I and I_a compartments have identi-
 145 cal contributions to the dynamics of chikungunya. We order the compartments

that contribute to new infections as follows: E , $I + I_a$, Y , and Z . Then the vector of the rates of appearance of new infections in these four compartments \mathcal{F} and the vector of the rates of transfer of existing infections between these four compartments \mathcal{V} are given by

$$\mathcal{F} = \begin{bmatrix} \frac{\beta_1 SZ}{N} \\ 0 \\ \frac{\beta_2 X(I+I_a)}{N} \\ 0 \end{bmatrix} \quad \text{and} \quad \mathcal{V} = \begin{bmatrix} \lambda_1 E + \mu_1 E \\ -\lambda_1 E + \gamma(I + I_a) + \mu_1(I + I_a) \\ \lambda_2 Y + \mu_2 Y \\ -\lambda_2 Y + \mu_2 Z \end{bmatrix}. \quad (3)$$

The matrices F and V are the Jacobians of \mathcal{F} and \mathcal{V} respectively, evaluated at DFE; they are given by

$$F = \begin{bmatrix} 0 & 0 & 0 & \beta_1 \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\Lambda_2 \beta_2 \mu_1}{\Lambda_1 \mu_2} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \lambda_1 + \mu_1 & 0 & 0 & 0 \\ -\lambda_1 & \gamma + \mu_1 & 0 & 0 \\ 0 & 0 & \lambda_2 + \mu_2 & 0 \\ 0 & 0 & -\lambda_2 & \mu_2 \end{bmatrix}. \quad (4)$$

The basic reproduction number is the spectral radius of the matrix FV^{-1} ; it is given by

$$R_0 = \frac{1}{\mu_2} \sqrt{\frac{\Lambda_2 \beta_1 \beta_2 \mu_1 \lambda_1 \lambda_2}{\Lambda_1 (\gamma + \mu_1) (\lambda_1 + \mu_1) (\lambda_2 + \mu_2)}}. \quad (5)$$

If $R_0 > 1$, then the system converges to the endemic equilibrium (EE) given by

$$\begin{aligned} S^* &= \frac{\Lambda_1 - (\lambda_1 + \mu_1)E^*}{\mu_1}, \\ E^* &= \frac{\Lambda_1 \Lambda_2 \beta_1 \beta_2 \mu_1 \lambda_1 \lambda_2 - \Lambda_1^2 \mu_2^2 (\lambda_1 + \mu_1) (\lambda_2 + \mu_2) (\gamma + \mu_1)}{\Lambda_2 \beta_1 \beta_2 \mu_1 \lambda_1 \lambda_2 (\lambda_1 + \mu_1) + \Lambda_1 \beta_2 \mu_1 \mu_2 \lambda_1 (\lambda_1 + \mu_1) (\lambda_2 + \mu_2)}, \\ I^* &= \frac{\phi \lambda_1 E^*}{\gamma + \mu_1}, \\ I_a^* &= \frac{(1 - \phi) \lambda_1 E^*}{\gamma + \mu_1}, \\ R^* &= \frac{\gamma \lambda_1 E^*}{\mu_1 (\gamma + \mu_1)}, \\ X^* &= \frac{\Lambda_1 \Lambda_2 (\gamma + \mu_1)}{\beta_2 \mu_1 \lambda_1 E^* + \Lambda_1 \mu_2 (\gamma + \mu_1)}, \\ Y^* &= \frac{\Lambda_2 \beta_2 \mu_1 \lambda_1 E^*}{(\lambda_2 + \mu_2) (\beta_2 \mu_1 \lambda_1 E^* + \Lambda_1 \mu_2 (\gamma + \mu_1))}, \quad \text{and} \\ Z^* &= \frac{\Lambda_2 \beta_2 \mu_1 \lambda_1 \lambda_2 E^*}{\mu_2 (\lambda_2 + \mu_2) (\beta_2 \mu_1 \lambda_1 E^* + \Lambda_1 \mu_2 (\gamma + \mu_1))}. \end{aligned} \quad (6)$$

155 In the game-theoretic models constructed in the next section, we will be
156 assuming that the system has reached an endemic equilibrium. In particular,
157 we will use the values from (6) for relevant compartment sizes.

158 3. RESULTS

159 We consider two intervention measures to fight the chikungunya outbreak
160 on Reunion Island: (1) using insect repellent to prevent mosquito bites, and (2)
161 emigrating to the neighboring Mauritius Island.

162 **3.1. Optimal levels of voluntary insect repellent usage.** We adopt a mod-
163 eling approach of [2, 13]. The strategy of an individual is the proportion of the
164 day $r \in [0, 1]$ the individual is protected from mosquito bites; the protection is
165 granted by insect repellent. We assume that the repellent provides complete
166 protection from mosquito bites while it is active. Since mosquitoes cannot
167 bites humans while they are protected by the insect repellent, the mosquito-
168 to-human transmission coefficient β_1 becomes a function of r . If no protection
169 is used ($r = 0$), then $\beta_1(0)$ is at its base value β_1^0 (given in table 1). If humans are
170 protected at all times ($r = 1$), then mosquitoes cannot bite these humans at all,
171 and hence they cannot infect humans: $\beta_1(1) = 0$. We therefore assume that the
172 mosquito-to-human transmission coefficient is a linear function of r given by

$$\beta_1 = \beta_1^0(1 - r). \quad (7)$$

173 If all susceptible humans in the population adopt the same strategy r_{pop} ,
174 then the basic reproduction number becomes a function of r_{pop} by substituting
175 the expression (7) for β_1 into (5). The graph of the basic reproduction number
176 as a function of the population strategy r_{pop} is shown in figure 2. The herd
177 immunity protection level r_{HI} is the population protection level that reaches
178 the threshold $R_0 = 1$ for disease eradication.

179 We define the utility function (expected payoff) of a susceptible individual
180 using strategy r in a population that adopted strategy r_{pop} as

$$E(r, r_{\text{pop}}) = -\pi(r, r_{\text{pop}})C_i - rC_p, \quad (8)$$

181 where C_i is the cost of infection, C_p is the cost of complete protection through
182 insect repellent, and $\pi(r, r_{\text{pop}})$ is the probability of infection. The latter depends
183 on the individual's strategy r because it determines how often mosquitoes may
184 bite the individual, and on the population strategy r_{pop} because it affects the
185 prevalence of the disease (e.g., the number of infected mosquitoes). The out-
186 come of a game does not change if the utility function is scaled, so we divide
187 the right-hand side of (8) by C_i to obtain

$$E(r, r_{\text{pop}}) = -\pi(r, r_{\text{pop}}) - rC, \quad (9)$$

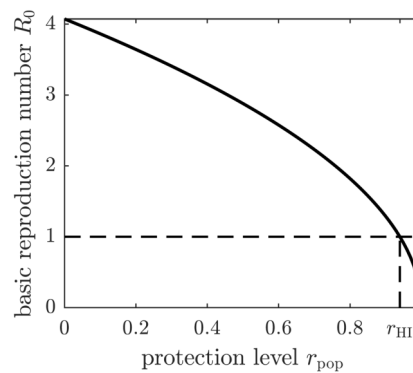


Figure 2. The graph of the basic reproduction number as a function of the population protection level r_{pop} . The basic reproduction number is at its maximum value—given by (5)—when no insect repellent is used by susceptible individuals ($r = 0$), and it becomes zero if the population employs complete protection from mosquito bites ($r = 1$). The threshold for disease eradication ($R_0 = 1$) is achieved at the herd immunity protection level r_{HI} .

188 where $C = C_p/C_i$ is the cost of complete protection relative to the cost of in-
189 fection.

190 We next compute the probability of getting infected and becoming symp-
191 tomatic as the transition probability from the susceptible compartment S to
192 the symptomatic infectious compartment I . This probability is the product of
193 the probability that a susceptible individual becomes exposed $f_1/(\mu_1 + f_1)$ multi-
194 plied by the probability that an exposed individual becomes symptomatically
195 infected $\phi\lambda_1/(\mu_1 + \phi\lambda_1 + (1 - \phi)\lambda_1) = \phi\lambda_1/(\mu_1 + \lambda_1)$:

$$\pi(r, r_{\text{pop}}) = \left(\frac{f_1(r, r_{\text{pop}})}{\mu_1 + f_1(r, r_{\text{pop}})} \right) \left(\frac{\phi\lambda_1}{\mu_1 + \lambda_1} \right), \quad (10)$$

196 where

$$f_1(r, r_{\text{pop}}) = \beta_1^0(1 - r) \frac{Z^*}{N^*} \quad (11)$$

197 is the force of infection, which depends on the individual protection level r
198 and on the population protection level r_{pop} . The individual protection level
199 r determines the rate at which mosquitoes bite the individual $\beta_1^0(1 - r)$. The
200 population protection level r_{pop} affects the prevalence of the disease in the pop-
201 ulation; it determines the size of the compartment Z^* via the substitution of
202 the expression $\beta_1^0(1 - r_{\text{pop}})$ for β_1 into (6). In particular, Z^* and N^* do not depend
203 on the individual protection level r .

204 To find the Nash equilibrium population protection level, we attempt to
205 maximize the utility function (9) of a focal individual. Observe that

$$f_1(r, r_{\text{pop}}) = (1 - r)f_1(0, r_{\text{pop}}), \quad (12)$$

206 and hence

$$\frac{\partial}{\partial r} f_1(r, r_{\text{pop}}) = -f_1(0, r_{\text{pop}}). \quad (13)$$

207 It follows that

$$\frac{\partial^2}{\partial r^2} E(r, r_{\text{pop}}) = \frac{2\mu_1 f_1(0, r_{\text{pop}})^2}{(\mu_1 + f_1(r, r_{\text{pop}}))^3} > 0. \quad (14)$$

208 Consequently, the utility function is a convex function of r , and thus it attains
209 its maximum value at one of the endpoints: $r = 0$ or $r = 1$.

210 This conclusion can be interpreted as follows. If the population repellent
211 usage is sufficiently high, then the probability of getting infected is very low.
212 A focal individual would rather bypass the potentially costly preventive mea-
213 sure and face the low morbidity risk instead. Hence individuals may improve
214 their payoff by deviating from the population strategy (they should stop using
215 repellent). On the other hand, if the population repellent usage is low, then the
216 probability of getting infected is too high, and a focal individual should prefer
217 to pay the cost of complete protection rather than face the high morbidity risk.
218 In this case, individuals may also improve their payoff by deviating from the
219 population strategy (they should use repellent 100% of the time).

220 So, if the population repellent usage is too high, then individuals would do
221 better if they stop using repellent, and hence the population repellent usage
222 will decrease. Conversely, if the population repellent usage is too low, then
223 individuals would do better if they start using repellent 100% of the time, and
224 hence the population repellent usage will increase. If the population repellent
225 usage is “just right” (Nash equilibrium), then individuals cannot improve their
226 payoffs by using repellent either less frequently or more frequently. We note
227 that there is a presumption of the population-wide adoption of treatment rates
228 in our model that is common to ESS-modeling; however, in situations such as
229 (14), there is a potential implication that the population should in fact sepa-
230 rate into distinct groups with different adoption rates. As it goes beyond the
231 framework discussed here, we leave that investigation for future research.

232 The Nash equilibrium protection level of the population r_{NE} is thus a solution
233 to the equation

$$E(0, r_{\text{NE}}) = E(1, r_{\text{NE}}) \quad (15)$$

234 or

$$\frac{f_1(0, r_{\text{NE}})}{\mu_1 + f_1(0, r_{\text{NE}})} \frac{\phi \lambda_1}{\mu_1 + \lambda_1} = C. \quad (16)$$

235 The graph of the optimal (Nash equilibrium) repellent usage as a function of the
236 relative cost of protection C is shown in figure 3a. The optimal repellent usage
237 r_{NE} reaches the herd immunity r_{HI} level only when the cost of the protective
238 measure relative to the cost of chikungunya infection is negligible (i.e., zero
239 mathematically). The optimal repellent usage remains very close to the herd

immunity level for a range of values of the relative cost C , and then drops off sharply. Once the relative cost of protection becomes too large (C_{\max}), then everyone stops using insect repellent because its high cost forces individuals to prefer to risk the cost of infection.

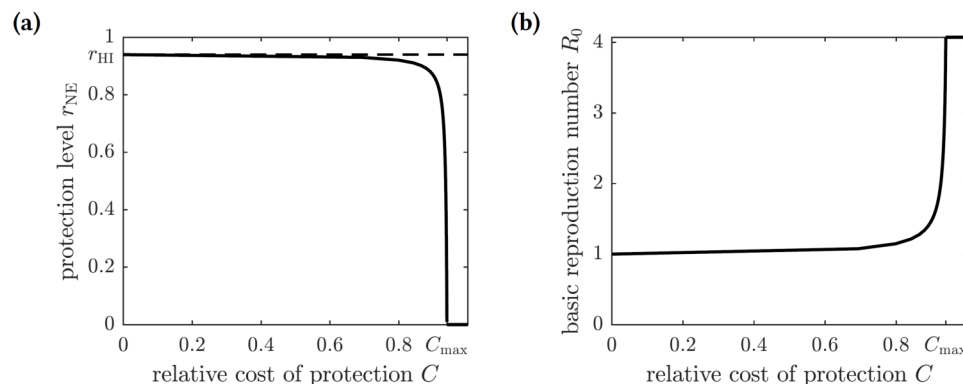


Figure 3. (a) The graph of the optimal level of population repellent usage r_{NE} as a function of the relative cost of protection. The optimal repellent usage reaches the herd immunity level only when $C = 0$. Everyone stops using repellent if its relative cost is too high: larger than the threshold value C_{\max} . (b) The graph of the basic reproduction number computed at the optimal population repellent usage level r_{NE} as a function of the relative cost of protection. When $C = 0$, the optimal protection level is equal to the herd immunity threshold, so $R_0 = 1$. When the relative cost of protection exceeds the threshold value C_{\max} , the population stops using repellent, and the basic reproduction number reaches its maximum value.

3.2. Optimal levels of mandatory insect repellent usage. We now consider a scenario where an organization (e.g., the government) enforces the use of insect repellent in the population to fight chikungunya. The organization must balance the cost of prevention of the disease in the population and the cost of treatment of symptomatically infected individuals. On the one hand, every individual who utilizes insect repellent 100% of the time results in a cost C_p (same as the cost of voluntary complete protection). On the other hand, every symptomatically infected individual results in a cost C_i .

The goal of the mandating organization is to find the repellent usage level for the population $r_{pop} \in [0, 1]$ so that the expected payoff (negative of the total cost)

$$E(r_{pop}) = -C_i I^* - r_{pop} C_p S^* \quad (17)$$

is maximal. (Note that the equilibrium values I^* and S^* depend on r_{pop} .) Here we analyze the case where the mandating organization addresses mosquito-to-human transmission by advising susceptible individuals to spray themselves with insect repellent. One may also consider an alternative scenario where

the infectious individuals are using insect repellent to reduce the human-to-mosquito transmission.

As before, we scale the payoff function and consider

$$E(r_{\text{pop}}) = -I^* - r_{\text{pop}}CS^*, \quad (18)$$

where $C = C_p/C_i$ is the relative cost of protection. The graphs of this function for different values of C are shown in figure 4. There are two possible outcomes: (1) the susceptible individuals should adopt the repellent usage level equal to that of the herd immunity threshold r_{HI} , leading to the eradication of the disease; or (2) no insect repellent should be used, and it is more cost-effective to treat symptomatically infected individuals only. The first outcome occurs for sufficiently low values of C (less than 0.00024), and the second outcome occurs for greater values of C (greater than 0.00024); the threshold value of C separating the two outcomes was found numerically.

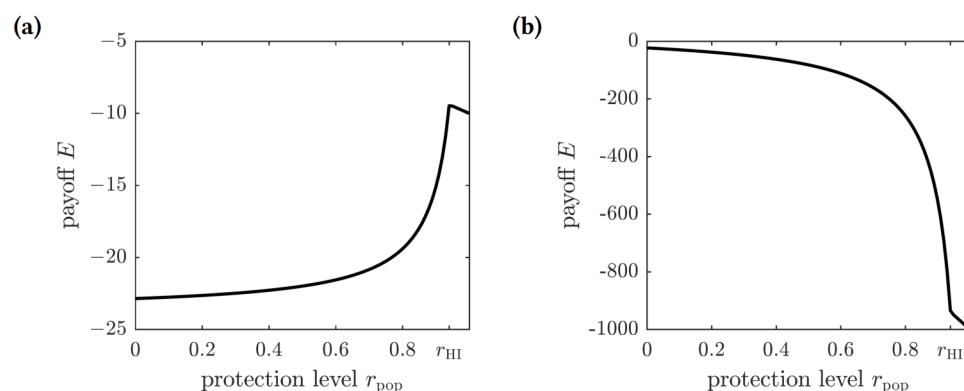


Figure 4. The graphs of the expected payoff function E given by the equation (18). (a) $C = 0.0001$, (b) $C = 0.01$. The graphs show two qualitatively different outcomes. If $C < 0.00024$ then mandating repellent usage necessary to reach the herd immunity threshold is most effective. If $C > 0.00024$ then no insect repellent should be used, and all efforts should be devoted to treating infected individuals.

3.3. Optimal levels of voluntary emigration. We are going to operate under the assumption that the chikungunya outbreak on Reunion Island did not affect the neighboring island of Mauritius Island, located 140 miles to the northeast of Reunion. Susceptible individuals—and only susceptible individuals, perhaps identified through a screening or quarantine procedure—may elect to emigrate from Reunion to Mauritius to protect themselves from the outbreak. To model the emigration as a potential personal protection measure against chikungunya, we allow residents of Reunion Island to leave the susceptible compartment of the epidemiological model at an emigration rate ω . This modification to (1) replaces the first equation describing the change in

the susceptible population with

$$\frac{dS}{dt} = \Lambda_1 - \frac{\beta_1 SZ}{N} - \mu_1 S - \omega S. \quad (19)$$

The DFE of the modified system is given by

$$(S^0, E^0, I^0, I_a^0, R^0, X^0, Y^0, Z^0) = \left(\frac{\Lambda_1}{\mu_1 + \omega}, 0, 0, 0, 0, \frac{\Lambda_2}{\mu_2}, 0, 0 \right), \quad (20)$$

and the corresponding basic reproduction number of the disease is

$$R_0 = \frac{1}{\mu_2} \sqrt{\frac{\Lambda_2 \beta_1 \beta_2 \lambda_1 \lambda_2 (\mu_1 + \omega)}{\Lambda_1 (\gamma + \mu_1) (\lambda_1 + \mu_1) (\lambda_2 + \mu_2)}}. \quad (21)$$

The graph of the basic reproduction number as a function of the emigration rate ω is shown in figure 5. It is an increasing function of ω , and hence emigrating susceptible individuals paradoxically make it worse for the remaining susceptible population. When the fresh blood supply is reduced due to emigration, susceptible mosquitoes are more likely to prey upon infectious humans, increasing the disease prevalence in the vector population. Consequently, the remaining susceptible human population is at an increased risk of contracting the disease from a mosquito bite. It follows that, while being potentially beneficial to specific individuals, voluntary emigration may result in a tragedy-of-the-commons effect for the remaining islanders.

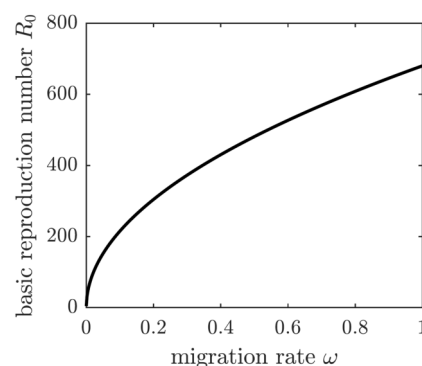


Figure 5. The graph of R_0 as a function of ω . If susceptible individuals emigrate from Reunion Island, then the remaining inhabitants face an increased spread of the disease.

To further investigate the effect of voluntary emigration on the chikungunya epidemic and whether (selfish) susceptible individuals should emigrate,

we compute the EE values of all compartments in the model with emigration:

$$\begin{aligned}
 N^* &= \frac{\Lambda_1 \mu_1 + \omega(\lambda_1 + \mu_1)E^*}{\mu_1(\mu_1 + \omega)}, \\
 S^* &= \frac{\Lambda_1 - (\lambda_1 + \mu_1)E^*}{\mu_1 + \omega}, \\
 I^* &= \frac{\phi \lambda_1 E^*}{\gamma + \mu_1}, \\
 I_a^* &= \frac{(1 - \phi) \lambda_1 E^*}{\gamma + \mu_1}, \\
 R^* &= \frac{\gamma \lambda_1 E^*}{\mu_1(\gamma + \mu_1)}, \\
 X^* &= \frac{\Lambda_2(\gamma + \mu_1)(\Lambda_1 \mu_1 + \omega(\lambda_1 + \mu_1)E^*)}{d + \mu_2(\gamma + \mu_1)(\Lambda_1 \mu_1 + \omega(\lambda_1 + \mu_1)E^*)}, \\
 Y^* &= \frac{\Lambda_2 \beta_2 \mu_1 \lambda_1 (\mu_1 + \omega)E^*}{(\lambda_2 + \mu_2)[d + \Lambda_1 \mu_1 \mu_2 (\gamma + \mu_1) + \mu_2 \omega(\lambda_1 + \mu_1)(\gamma + \mu_1)E^*]}, \text{ and} \\
 Z^* &= \frac{\Lambda_2 \beta_2 \mu_1 \lambda_1 \lambda_2 (\mu_1 + \omega)E^*}{\mu_2(\lambda_2 + \mu_2)[d + \Lambda_1 \mu_1 \mu_2 (\gamma + \mu_1) + \mu_2 \omega(\lambda_1 + \mu_1)(\gamma + \mu_1)E^*]},
 \end{aligned} \tag{22}$$

where

$$d = \beta_2 \lambda_1 \mu_1 (\mu_1 + \omega)E^*, \tag{23}$$

and E^* is the solution to the quadratic equation

$$aE^2 + bE + c = 0 \tag{24}$$

with coefficients

$$\begin{aligned}
 a &= -\mu_2 \omega (\lambda_1 + \mu_1)^2 (\lambda_2 + \mu_2) [\beta_2 \mu_1 \lambda_1 (\mu_1 + \omega) + \mu_2 \omega (\lambda_1 + \mu_1) (\gamma + \mu_1)], \\
 b &= -\Lambda_2 \beta_1 \beta_2 \mu_1^2 \lambda_1 \lambda_2 (\lambda_1 + \mu_1) (\mu_1 + \omega) - 2\Lambda_1 \mu_1 \mu_2^2 \omega (\lambda_1 + \mu_1)^2 (\lambda_2 + \mu_2) (\gamma + \mu_1) \\
 &\quad - \Lambda_1 \beta_2 \mu_1^2 \mu_2 \lambda_1 (\lambda_1 + \mu_1) (\lambda_2 + \mu_2) (\mu_1 + \omega), \text{ and} \\
 c &= \Lambda_1 \Lambda_2 \beta_1 \beta_2 \mu_1^2 \lambda_1 \lambda_2 (\mu_2 + \omega) - \Lambda_1^2 \mu_1^2 \mu_2^2 (\lambda_1 + \mu_1) (\lambda_2 + \mu_2) (\gamma + \mu_1).
 \end{aligned} \tag{25}$$

The biologically meaningful root of this equation is given by

$$E^* = \frac{-b - \sqrt{b^2 - 4ac}}{2a}. \tag{26}$$

Figure 6 shows the graphs of the number and proportion of symptomatically infectious individuals in the population as functions of the emigration rate ω . As more individuals leave the island, the overall population level declines, and hence there are fewer infected individuals. However, the infection spreads faster among the remaining inhabitants, resulting in a greater proportion of infected individuals in the population. The proportion of symptomatically infectious individuals grows with the migration rate, but it asymptotically

approaches the value

$$\lim_{\omega \rightarrow \infty} \frac{I^*}{N^*} = \frac{\phi \mu_1 \lambda_1}{(\gamma + \mu_1)(\lambda_1 + \mu_1)}. \quad (27)$$

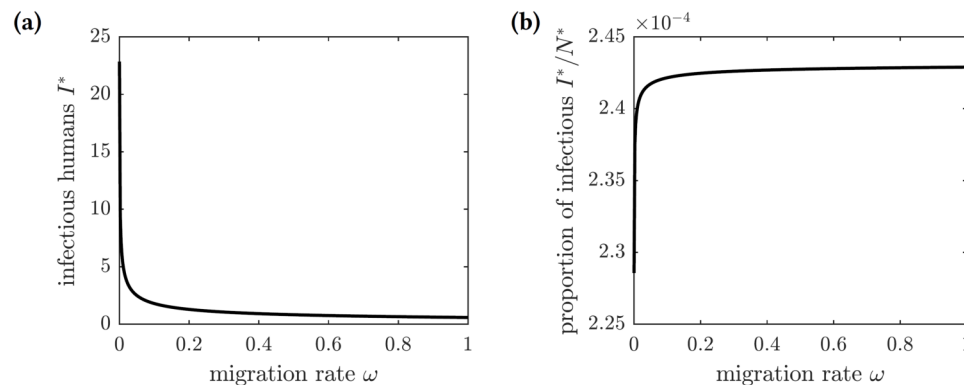


Figure 6. The graphs of the (a) number and (b) proportion of symptomatically infectious individuals in the population as functions of the emigration rate ω . Increased migration levels result in fewer infectious individuals overall but a greater proportion of infectious individuals in the population.

We next consider a game-theoretic model of individual migration decisions. Suppose that the population adopted the emigration rate ω_{pop} . A focal susceptible individual is presented with a choice to either migrate or not migrate. Each of the two strategic choices carries a corresponding payoff: E_m for migrate and E_{nm} for not migrate, given by

$$\begin{aligned} E_m(\omega_{\text{pop}}) &= -C_b - \omega_{\text{pop}} C_s, \text{ and} \\ E_{nm}(\omega_{\text{pop}}) &= -\pi(\omega_{\text{pop}}) C_i, \end{aligned} \quad (28)$$

where C_b is the base (fixed) cost of migration, C_s is the scaling cost of migration, C_i is the cost of the (symptomatic) chikungunya infection, and $\pi(\omega_{\text{pop}})$ is the probability of getting infected given the population emigration rate ω_{pop} . We assume that the cost of emigration is an increasing function of the migration rate because of the limited immigration potential of Mauritius: the more individuals migrate to Mauritius, the harder it becomes to find housing and jobs. For simplicity, we model the increasing emigration cost as a linear function of the migration rate. The probability of getting infected and incurring the cost of a symptomatic chikungunya infection if remaining on Reunion Island is the transition probability from the susceptible class S to the symptomatically infectious class I :

$$\pi(\omega_{\text{pop}}) = \frac{f_1(\omega_{\text{pop}})}{\mu_1 + f_1(\omega_{\text{pop}})} \frac{\phi \lambda_1}{\mu_1 + \lambda_1}. \quad (29)$$

325 This probability is an increasing function of the emigration rate because each
326 of the remaining susceptible individuals faces a higher risk of infection (cf. fig-
327 ure 5); the graph is shown in figure 7.

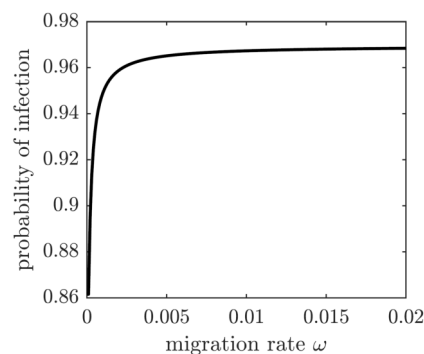


Figure 7. The probability of symptomatic chikungunya infection for a susceptible individual on Reunion Island is increasing with the emigration rate ω .

328 To find conditions when a focal susceptible individual should emigrate to
329 Mauritius or remain on Reunion, we scale the payoffs in equation (28) by $1/C_i$
330 and obtain

$$\begin{aligned} E_m &= -\tilde{C}_b - \omega_{\text{pop}} \tilde{C}_s, \text{ and} \\ E_{nm} &= -\pi(\omega_{\text{pop}}), \end{aligned} \quad (30)$$

331 where \tilde{C}_b and \tilde{C}_s are relative base and scaling costs of emigration, respectively.
332 A susceptible individual should emigrate when the relative cost of doing so is
333 less than the probability of getting infected: $\tilde{C}_b + \omega_{\text{pop}} \tilde{C}_s < \pi(\omega_{\text{pop}})$, and the
334 individual should remain on the island otherwise. The regions in the (\tilde{C}_b, ω) -
335 parameter space corresponding to the best choice for a focal individual for
336 several values of \tilde{C}_s are shown in figure 8. If the scaling cost of emigration
337 C_s is negligible (i.e., the cost of emigration does not depend on the number of
338 emigrating individuals), then the best strategy of a susceptible individual is to
339 emigrate as long as the relative base cost of emigration \tilde{C}_b is sufficiently small
340 (figure 8a). On the other hand, as the relative scaling cost of emigration \tilde{C}_s
341 grows, the individual's decision to emigrate starts to depend on the emigration
342 decisions of other individuals (figure 8b–c), until it becomes unprofitable to
343 emigrate regardless of the relative base cost of emigration if the emigration
344 rate is too high (figure 8d).

345 **3.4. Optimal levels of mandatory emigration.** Finally, we consider the po-
346 tential impacts on the chikungunya epidemic on Reunion Island of coordinated
347 emigration efforts. A mandating organization attempts to minimize overall
348 costs, which are comprised of the cost of treatment of symptomatically infected
349 individuals and the relocation costs of emigrating individuals. To estimate the

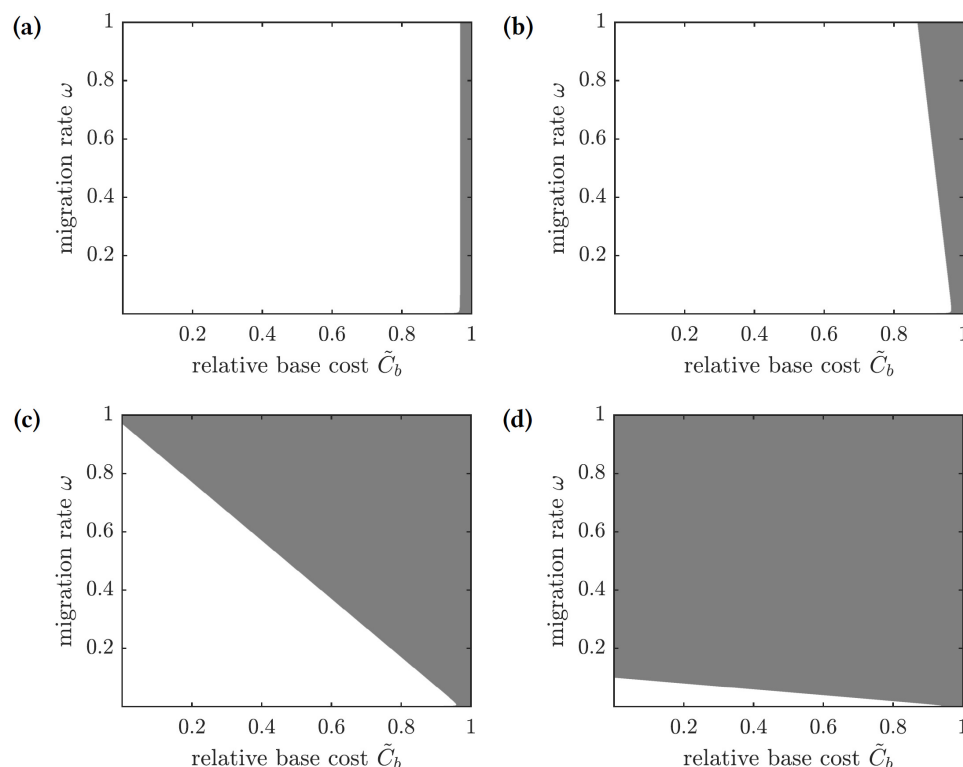


Figure 8. The regions in the (\tilde{C}_b, ω) -parameter space showing whether a focal susceptible individual should emigrate to Mauritius or remain on Reunion. Color code: white—emigrate, gray—stay. (a) $\tilde{C}_s = 0$, (b) $\tilde{C}_s = 0.1$, (c) $\tilde{C}_s = 1$, (d) $\tilde{C}_s = 10$.

number of emigrating susceptible individuals, we consider the difference between the total population size at equilibrium without emigration ($N^* = \Lambda_1/\mu_1$) and the total population size at equilibrium given the population migration rate ω (this expression is given in the first equation of (22)); we denote this difference by N_ω^* .

The payoff of the emigration policy with migration rate ω is given by

$$E(\omega) = -I^* - \tilde{C}_m N_\omega^*, \quad (31)$$

where $\tilde{C}_m = C_m/C_i$ is the cost of migration relative to the cost of infection. The graphs of this function for several values of \tilde{C}_m are shown in figure 9. There are three qualitatively different outcomes:

1. For very low relative migration cost ($\tilde{C}_m \leq 0.00022$), higher migration rates result in smallest overall costs; however, the near-optimal costs are quickly achieved by small values of migration rate ($\omega = 0.01$)—see figure 9a.

- 363 2. There is a small interval of the relative migration cost values ($0.00023 \leq$
364 $\tilde{C}_m \leq 0.00025$) where the optimal cost is achieved in the interior for very
365 small values of the migration rate ($\omega < 0.002$)—see figures 9b and 9c.
- 366 3. For all sufficiently large values of the relative migration cost ($\tilde{C}_m \geq$
367 0.00026), it is best to not allow individuals to emigrate from the island—
368 see figure 9d.

369 In practice, however, the cost of emigration (such as relocation from Reunion to
370 Mauritius) is usually comparable to or higher than the cost of the symptomatic
371 chikungunya infection. Therefore, the scenario shown in figure 9d is the most
372 realistic one: it is best to not allow susceptible individuals to leave the island
373 during the outbreak.

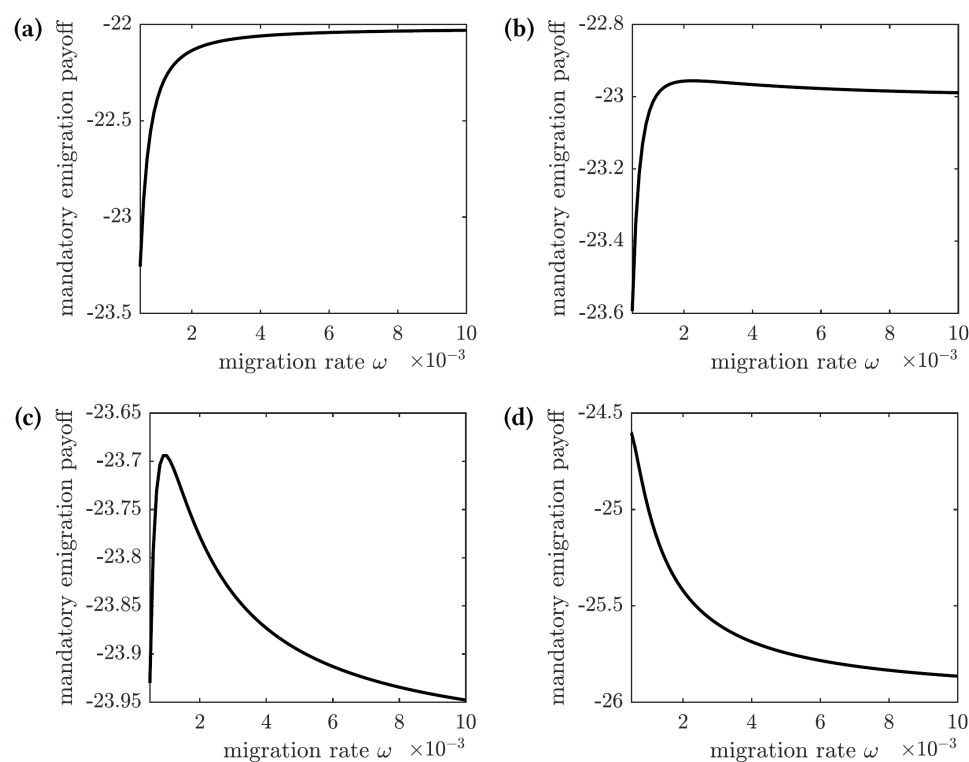


Figure 9. The overall cost of the mandated emigration policy as a function of the migration rate ω . (a) $\tilde{C}_m = 0.00022$, (b) $\tilde{C}_m = 0.00023$, (c) $\tilde{C}_m = 0.00024$, (d) $\tilde{C}_m = 0.00026$.

374

4. DISCUSSION

375 We investigated potential implications of both voluntary and mandatory in-
376 tervention measures to fight the chikungunya outbreak on Reunion Island.
377 Susceptible individuals may either prevent the infection by using insect re-
378 pellent and hence reduce the frequency of mosquito bites, or leave Reunion

Island and emigrate to neighboring Mauritius. We adopted a version of a previous epidemiological model of the chikungunya transmission on Reunion Island [40]. The epidemiological model informed the payoff functions in the game-theoretic models of individual and centralized decisions on the level of adoption of the protective measures. We found that the two protocols resulted in qualitatively different predictions concerning optimal allocations, with the latter measure creating an additional hazard for non-participants.

Voluntary participation in the two intervention measures produced opposite population-level effects. The more susceptible individuals spray themselves with insect repellent, the less likely the infectious mosquitoes generate new human infections before they die. Consequently, higher adoption levels of insect repellent usage in the population resulted in lower basic reproduction number values for the disease. Individuals using repellent provide (near) herd-immunity-effect benefits to the entire population. In contrast, if susceptible individuals vacated the island, then susceptible mosquitoes were more likely to bite infectious humans as a percentage of the remaining population, thus increasing the disease prevalence among mosquitoes. The remaining susceptible individuals subsequently faced an increased risk of contracting the infection from a mosquito bite. Increased migration levels resulted in drastically elevated basic reproduction number values. Thus, the impact of voluntary emigration is similar to the tragedy-of-the-commons effect: while being potentially beneficial to specific individuals, it hurts the remaining islanders.

The mandated repellent usage protocol resulted in the same outcome as the voluntary (i.e., selfishly rational) compliance scenario if the cost of the preventive measure relative to the cost of the disease was too high: it was best to bypass the repellent usage altogether. But if the relative cost of protection was sufficiently low, so that repellent usage was warranted, then the two scenarios effected different outcomes. In the voluntary compliance case, the population repellent usage fell short—albeit not by much—of the herd immunity threshold. In the mandated protocol case, reaching the herd immunity usage level and thus eradicating the disease was most effective.

That voluntary adoption of preventative measures against an infectious disease falls short of the herd immunity threshold has also been observed in other studies [17, 2, 13, 5, 19]. Yet looking at a mandated repellent usage scenario revealed that a mandatory protocol might have eliminated the epidemic if the relative cost of the preventive measure was sufficiently low.

Mandatory emigration from Reunion Island demonstrated that this preventive measure made sense for the public benefit only when the cost of relocation was significantly lower than the public cost of infection. Since this mathematical assumption is not likely to hold in practice, the model predicted that it

was best to avoid migration of susceptible individuals from the island. The potentially high cost of relocating susceptible individuals away from the epidemic was not compensated by the minimal decrease in the number of infected individuals.

The qualitative differences in optimal behavior under the two alternative treatment protocols invite further examination of our model's behavior and assumptions. Both evacuation/emigration of the human populace and the use of repellent reduce the pool of potential blood hosts for the mosquito population; however, they produce contrasting effects on the force of infection. A base assumption in the model is that each insect has a consistent average number of encounters with humans over a given time span. Repellent usage directly decreases the force of infection by deterring biting upon encounter—it is this feature of “wasted” encounters that permits the development of herd immunity. In contrast, reduction in the size of the standing human population elevates the force of infection by increasing the number of encounters an individual human experiences. Secondarily, this results in increased prevalence of the disease in the vector-population as their blood hosts are more likely to be infected. We hypothesize that distinct protocol results depend upon the presence of (1) a distinct vector population; (2) an assumption of constant predation encounters for vectors; (3) the proportional allocation of encounters across humans; (4) an inability of vectors to pre-judge encounters and thereby shift towards more palatable hosts; and (5) a secondary food source to support constant recruitment of new vectors. We propose a followup study to this paper that focuses specifically on the dynamic analysis of the force of infection as these assumptions are introduced or removed.

5. CONCLUSIONS

There are several additional directions in which our model can be improved. First, we are assuming that individuals possess complete information regarding the prevalence of the disease and the costs of protection relative to infection. But individuals rarely have access to the exact disease prevalence data, and hence they may only guess the relevant numbers. Second, the cost of intervention (such as using insect repellent) and the cost of the disease must be estimated individually. These costs include both direct costs such as paying for repellent or medical treatment, and indirect costs such as potentially harmful side effects of the chemicals in repellent or morbidity risks of the infection. Additionally, different individuals may have various opinions about the risk of using repellent or getting infected with chikungunya virus. Building these uncertainties into the model should allow a broader outlook at different strategies to combat such outbreaks.

Moreover, our model assumes that the population has reached an equilibrium with respect to the disease dynamics. But reaching this equilibrium usually occurs on a different timescale compared to individual preventive actions. For example, individuals could be more likely to participate in preventive efforts when the epidemic is at its peak rather than when the disease reached the endemic state. A dynamic model where susceptible individuals inform their preventive decisions on the current state of the prevalence of the disease which, in turn, affects the dynamics of the disease transmission, should present a more realistic analysis of selfish individual decisions to prevent the infection.

ACKNOWLEDGEMENTS

This research was conducted as part of a Research Experiences for Undergraduates program at the University of North Carolina at Greensboro in summer 2018, which was funded by the NSF grant DMS-1659646. SRMK, AOF, and DAF were undergraduate student participants, and JTR and IVE were faculty mentors. We thank J. Safley and P. Waiker, who served as graduate assistants during the REU program where this research was conducted.

CONFLICT OF INTEREST

All authors declare no conflicts of interest in this paper.

REFERENCES

- [1] M. Amaku, F.A.B. Coutinho, S.M. Raimundo, L.F. Lopez, M.N. Burattini, and E. Massad. A comparative analysis of the relative efficacy of vector-control strategies against dengue fever. *Bulletin of Mathematical Biology*, 76(3):697–717, 2014.
- [2] C.T. Bauch and D.J.D. Earn. Vaccination and the theory of games. *Proceedings of the National Academy of Sciences of the United States of America*, 101(36):13391–13394, 2004.
- [3] C.T. Bauch, A.P. Galvani, and D.J.D. Earn. Group interest versus self-interest in smallpox vaccination policy. *Proceedings of the National Academy of Sciences of the United States of America*, 100(18):10564–10567, 2003.
- [4] G. Borgherini, P. Poubeau, A. Jossaume, A. Gouix, L. Cotte, A. Michault, C. Arvin-Berod, and F. Paganin. Persistent arthralgia associated with Chikungunya virus: A study of 88 adult patients on Reunion Island. *Clinical Infectious Diseases*, 47(4):469–475, 2008.
- [5] A. Brettin, R. Rossi-Goldthorpe, K. Weishaar, and I.V. Erovenko. Ebola could be eradicated through voluntary vaccination. *Royal Society Open Science*, 5(1):171591, 2018.
- [6] M. Broom and J. Rychtář. *Game-Theoretical Models in Biology*. Chapman and Hall/CRC, 2013.
- [7] M. Broom, J. Rychtář, and T. Spears-Gill. The game-theoretical model of using insecticide-treated bed-nets to fight malaria. *Applied Mathematics*, 7:852–860, 2016.
- [8] Centers for Disease Control and Prevention. Chikungunya Virus: Prevention. <https://www.cdc.gov/chikungunya/prevention/index.html> (Accessed December 23, 2018).

- 498 [9] Centers for Disease Control and Prevention. Chikungunya Virus: Symptoms, Diagno-
499 sis, & Treatment. <https://www.cdc.gov/chikungunya/symptoms/index.html> (Ac-
500 cessed December 23, 2018).
- 501 [10] K. Crawford, A. Lancaster, H. Oh, and J. Rychtář. A voluntary use of insecticide-treated
502 cattle can eliminate African sleeping sickness. *Letters in Biomathematics*, 2(1):91–101,
503 2015.
- 504 [11] R. Cressman. *Evolutionary Dynamics and Extensive Form Games*. The MIT Press, 2003.
- 505 [12] L.G. Darrigo, A.M. de Sant’Anna Carvalho, and C.M. Machado. Chikungunya, Dengue,
506 and Zika in immunocompromised hosts. *Current Infectious Disease Reports*, 20(4):1–10,
507 2018.
- 508 [13] C. Dorsett, H. Oh, M.L. Paulemond, and J. Rychtář. Optimal repellent usage to combat
509 dengue fever. *Bulletin of Mathematical Biology*, 78(5):916–922, 2016.
- 510 [14] Y. Dumont, F. Chiroleu, and C. Domerg. On a temporal model for the Chikungunya dis-
511 ease: Modeling, theory and numerics. *Mathematical Biosciences*, 213(1):80–91, 2008.
- 512 [15] E.D. Fourie and J.G. Morrison. Rheumatoid arthritic syndrome after Chikungunya fever.
513 *South African Medical Journal*, 56(4):130–132, 1979.
- 514 [16] A.P. Galvani, T.C. Reluga, and G.B. Chapman. Long-standing influenza vaccination policy
515 is in accord with individual self-interest but not with the utilitarian optimum. *Proceedings*
516 *of the National Academy of Sciences of the United States of America*, 104(13):5692–5697,
517 2007.
- 518 [17] P.-Y. Geoffard and T. Philipson. Disease eradication: Private versus public vaccination.
519 *American Economic Review*, 87(1):222–230, 1997.
- 520 [18] J. Hofbauer and K. Sigmund. *Evolutionary Games and Population Dynamics*. Cambridge
521 University Press, 1998.
- 522 [19] J. Kobe, N. Pritchard, Z. Short, I.V. Erovenko, J. Rychtář, and J.T. Rowell. A game-theoretic
523 model of cholera with optimal personal protection strategies. *Bulletin of Mathematical*
524 *Biology*, 80(10):2580–2599, 2018.
- 525 [20] J.C. Leao, C.D.L. Marques, A.L.B.P. Duarte, O.P. de Almeida, S. Porter, and L.A. Gueiros.
526 Chikungunya fever: General and oral healthcare implications. *Oral Diseases*, 24(1–2):233–
527 237, 2018.
- 528 [21] P. Manfredi and A. D’Onofrio. *Modeling the Interplay Between Human Behavior and the*
529 *Spread of Infectious Diseases*. Springer, 2013.
- 530 [22] A Martinez, J. Machado, E. Sanchez, and I.V. Erovenko. Optimal vaccination strategies to
531 reduce endemic levels of meningitis in Africa. *Submitted*.
- 532 [23] J. Maynard Smith. *Evolution and the Theory of Games*. Cambridge University Press, 1982.
- 533 [24] J.C. Pile, E. A. Henschel, G.W. Christopher, K.E. Steele, and J.A. Pavlin. Chikungunya in a
534 North American traveler. *Journal of Travel Medicine*, 6(2):137–139, 1999.
- 535 [25] N.A. Prow, L. Liu, E. Nakayama, T.H. Cooper, K. Yan, P. Eldi, J.E. Hazlewood, B. Tang, T.T.
536 Le, Y.X. Setoh, A.A. Khromykh, J. Hobson-Peters, K.R. Diener, P.M. Howley, J.D. Hay-
537 ball, and A. Suhrbier. A vaccinia-based single vector construct multi-pathogen vaccine
538 protects against both Zika and Chikungunya viruses. *Nature Communications*, 9(1):1230,
539 2018.
- 540 [26] P. Renault, E. Balleydier, E. D’Ortenzio, M. Bâville, and L. Filleul. Epidemiology of
541 Chikungunya infection on Reunion Island, Mayotte, and neighboring countries. *Medecine*
542 *et Maladies Infectieuses*, 42(3):93–101, 2012.

- 543 [27] M.C. Robinson. An epidemic of virus disease in Southern Province, Tanganyika Territory,
544 in 1952–1953. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 49(1):28–
545 32, 1955.
- 546 [28] R. Rosenberg, N.P. Lindsey, M. Fischer, C.J. Gregory, A.F. Hinckley, P.S. Mead, G. Paz-
547 Bailey, S.H. Waterman, N.A. Drexler, G.J. Kersh, H. Hooks, S.K. Partridge, S.N. Visser,
548 C.B. Beard, and L.R. Petersen. Vital signs: Trends in reported vectorborne disease
549 cases—United States and Territories, 2004–2016. *Morbidity and Mortality Weekly Report*,
550 67(17):496–501, 2018.
- 551 [29] K. Scheckelhoff, A. Ejaz, and I.V. Erovenko. A game-theoretic model of optimal clean
552 equipment usage to prevent hepatitis C among injecting drug users. *Submitted*.
- 553 [30] S.L. Seneviratne, P. Gurugama, and J. Perera. Chikungunya viral infections: An emerging
554 problem. *Journal of Travel Medicine*, 14(5):320–325, 2007.
- 555 [31] E. Shim, G.B. Chapman, J.P. Townsend, and A.P. Galvani. The influence of altruism on
556 influenza vaccination decisions. *Journal of the Royal Society Interface*, 9(74):2234–2243,
557 2012.
- 558 [32] E. Shim, J.J. Grefenstette, S.M. Albert, B.E. Cakouros, and D.S. Burke. A game dynamic
559 model for vaccine skeptics and vaccine believers: Measles as an example. *Journal of The-
560 oretical Biology*, 295:194–203, 2012.
- 561 [33] E. Shim, B. Kochin, and A. Galvani. Insights from epidemiological game theory into
562 gender-specific vaccination against rubella. *Mathematical Biosciences and Engineering*,
563 6(4):839–854, 2009.
- 564 [34] D. Sykes and J. Rychtář. A game-theoretic approach to valuating toxoplasmosis vaccina-
565 tion strategies. *Theoretical Population Biology*, 105:33–38, 2015.
- 566 [35] S.D. Thiberville, V. Boisson, J. Gaudart, F. Simon, A. Flahault, and X. de Lamballerie.
567 Chikungunya fever: A clinical and virological investigation of outpatients on Reunion
568 Island, southwest Indian Ocean. *PLoS Neglected Tropical Diseases*, 7(1):e2004, 2013.
- 569 [36] P. van den Driessche and J. Watmough. Reproduction numbers and sub-threshold en-
570 demic equilibria for compartmental models of disease transmission. *Mathematical Bio-
571 sciences*, 180:29–48, 2002.
- 572 [37] T.L. Vincent and J.S. Brown. *Evolutionary Game Theory, Natural Selection, and Darwinian*
573 *Dynamics*. Cambridge University Press, 2005.
- 574 [38] J. von Neumann and O. Morgenstern. *Theory of Games and Economic Behavior*. Princeton
575 University Press, 1944.
- 576 [39] Z. Wang, C.T. Bauch, S. Bhattacharyya, A. d’Onofrio, P. Manfredi, M. Perc, N. Perra,
577 M. Salathé, and D. Zhao. Statistical physics of vaccination. *Physics Reports*, 664:1–113,
578 2016.
- 579 [40] L. Yakob and A.C.A. Clements. A mathematical model of Chikungunya dynamics and
580 control: The major epidemic on Réunion Island. *PLoS ONE*, 8(3):e57448, 2013.