

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

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*Corresponding author: igor@uncg.edu.

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

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

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

47 While the severity of the Reunion chikungunya outbreak may seem like
48 an isolated event, vector-borne diseases such as malaria and dengue are be-
49 coming an increasingly prevalent public health issue in today’s society. In the
50 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.

89

2. METHODS

90 We adopt a version of the epidemiological model of the chikungunya out-
91 break on Reunion Island by Jakob and Clements [40] by adding population dy-
92 namics (birth and death demographic processes) for both humans and mosquitoes
93 and strategically-linked parameters so that the disease potentially may estab-
94 lish itself endemically. This assumption then permits us to use the framework
95 of Dorsett et al. [13], Amaku [1], and Bauch and Earn [2]. All human inhabi-
96 tants of the island (N) are divided into 5 compartments: individuals susceptible
97 to chikungunya (S); exposed individuals (E), who had been bitten by an in-
98 fected mosquito and acquired the disease; symptomatic infectious individuals
99 (I), who developed the symptoms of the disease and became infectious to biting
100 mosquitoes; asymptomatic infectious individuals (I_a), who became infectious
101 but did not develop symptoms; and recovered individuals (R), who recovered
102 from chikungunya and acquired immunity. The mosquito population is di-
103 vided into 3 compartments: susceptible mosquitoes (X); exposed mosquitoes
104 (Y), who bit an infected human and were exposed to the pathogen; and infec-
105 tious mosquitoes (Z), who may infect humans by biting susceptible individuals.
106 We did not consider in this model the full life-cycle of mosquitoes, such as egg
107 and larval stages, because we did not incorporate mosquito population control
108 as one of the measures to fight chikungunya.

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

115 Susceptible humans who are bitten by infectious mosquitoes become ex-
116 posed. The force of infection f_1 , which is the rate at which susceptible individ-
117 uals move to the exposed class, depends on the density of susceptible humans
118 S/N (i.e., the probability that an infectious mosquito bites a susceptible indi-
119 vidual), the number of infectious mosquitoes Z , and the mosquito-to-human
120 transmission coefficient β_1 . Similarly, susceptible mosquitoes who bite infec-
121 tious humans become exposed. The force of infection f_2 , which is the rate
122 at which susceptible mosquitoes move to the exposed compartment, depends
123 on the the density of infectious humans $(I + I_a)/N$ (i.e., the probability that a
124 mosquito bites an infectious individual), the number of susceptible mosquitoes
125 X , and the human-to-mosquito transmission coefficient β_2 .

126 Exposed humans become infectious (after a latency period) at a rate λ_1 ; a
127 proportion ϕ of infectious individuals develop symptoms of the disease. Ex-
128 posed 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).

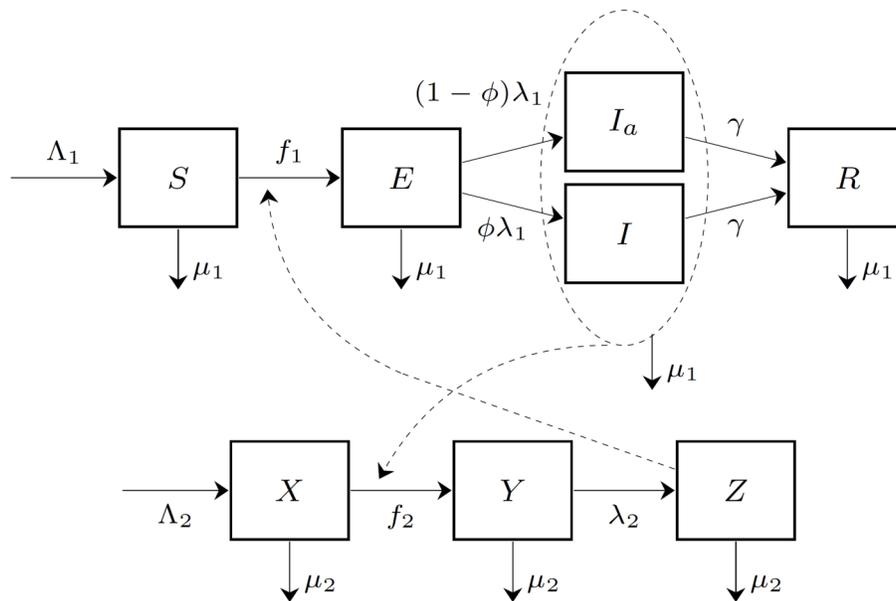


Figure 1. The compartment model of chikungunya virus transmission on Reunion Island. The human population is divided into five compartments: susceptible (S), exposed (E), symptomatic infectious (I), asymptomatic infectious (I_a), and recovered (R). The mosquito population is divided into three compartments: 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

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

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

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

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

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

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

154 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

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

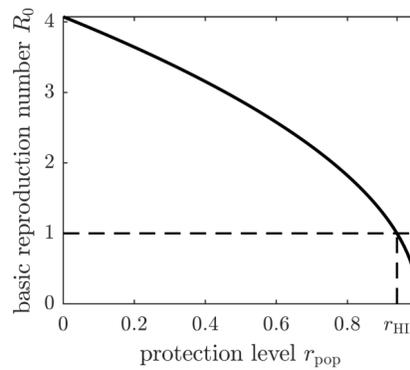


Figure 2. The graph of the basic reproduction number as a function of the population protection level r_{pop} . The basic reproduction number is at its maximum value—given by (5)—when no insect repellent is used by susceptible individuals ($r = 0$), and it becomes zero if the population employs complete 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

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

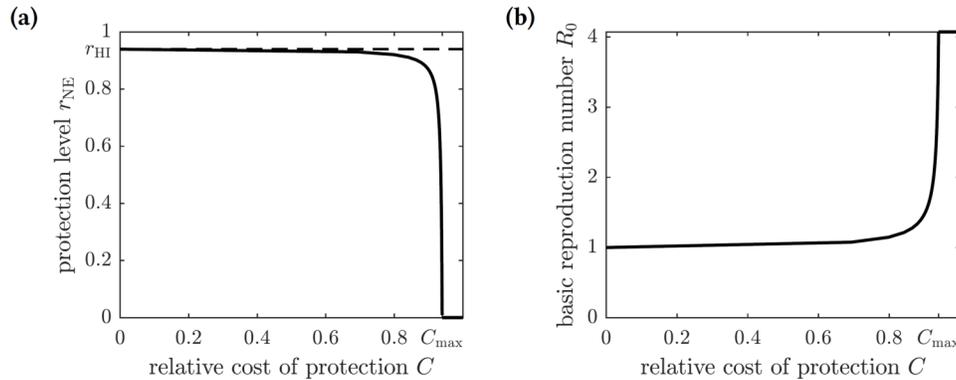


Figure 3. (a) The graph of the optimal level of population repellent usage r_{NE} as a function of the relative cost of protection. The optimal repellent usage reaches the herd immunity level only when $C = 0$. Everyone stops using repellent if its relative cost is too high: larger than the threshold 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.

244 **3.2. Optimal levels of mandatory insect repellent usage.** We now con-
 245 sider a scenario where an organization (e.g., the government) enforces the use
 246 of insect repellent in the population to fight chikungunya. The organization
 247 must balance the cost of prevention of the disease in the population and the
 248 cost of treatment of symptomatically infected individuals. On the one hand,
 249 every individual who utilizes insect repellent 100% of the time results in a cost
 250 C_p (same as the cost of voluntary complete protection). On the other hand,
 251 every symptomatically infected individual results in a cost C_i .

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

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

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

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

261 As before, we scale the payoff function and consider

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

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

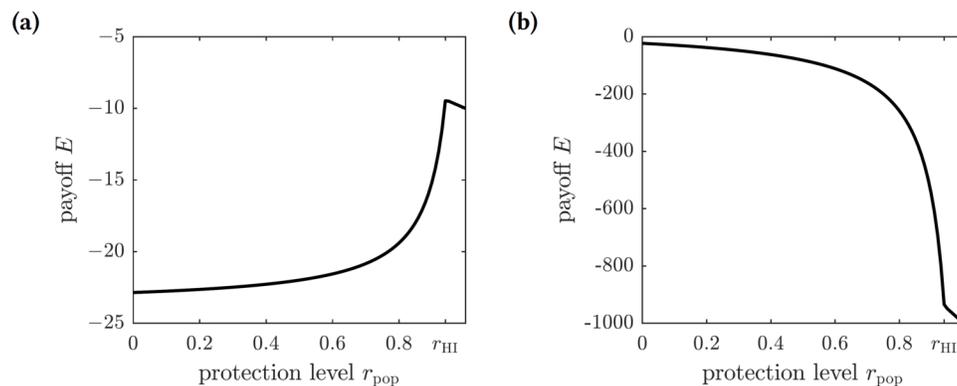


Figure 4. The graphs of the expected payoff function E given by the equation (18). (a) $C = 0.0001$, (b) $C = 0.01$. The graphs show two qualitatively different outcomes. If $C < 0.00024$ then mandating repellent usage necessary to reach the herd immunity threshold is most effective. If $C > 0.00024$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

271 **3.3. Optimal levels of voluntary emigration.** We are going to operate un-
 272 der the assumption that the chikungunya outbreak on Reunion Island did not
 273 affect the neighboring island of Mauritius Island, located 140 miles to the north-
 274 east of Reunion. Susceptible individuals—and only susceptible individuals,
 275 perhaps identified through a screening or quarantine procedure—may elect
 276 to emigrate from Reunion to Mauritius to protect themselves from the out-
 277 break. To model the emigration as a potential personal protection measure
 278 against chikungunya, we allow residents of Reunion Island to leave the sus-
 279 ceptible compartment of the epidemiological model at an emigration rate ω .
 280 This modification to (1) replaces the first equation describing the change in

281 the susceptible population with

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

282 The DFE of the modified system is given by

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

283 and the corresponding basic reproduction number of the disease is

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

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

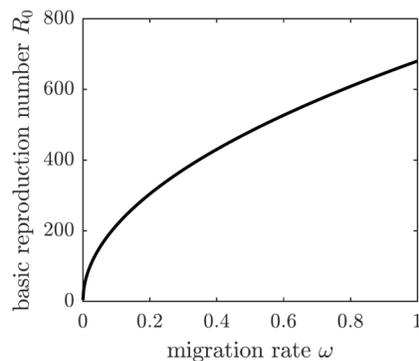


Figure 5. The graph of R_0 as a function of ω . If susceptible individuals emigrate from Reunion Island, then the remaining inhabitants face an increased spread of the disease.

294 To further investigate the effect of voluntary emigration on the chikun-
 295 gunya epidemic and whether (selfish) susceptible individuals should emigrate,

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

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

297 where

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

298 and E^* is the solution to the quadratic equation

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

299 with coefficients

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

300 The biologically meaningful root of this equation is given by

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

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

308 approaches the value

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

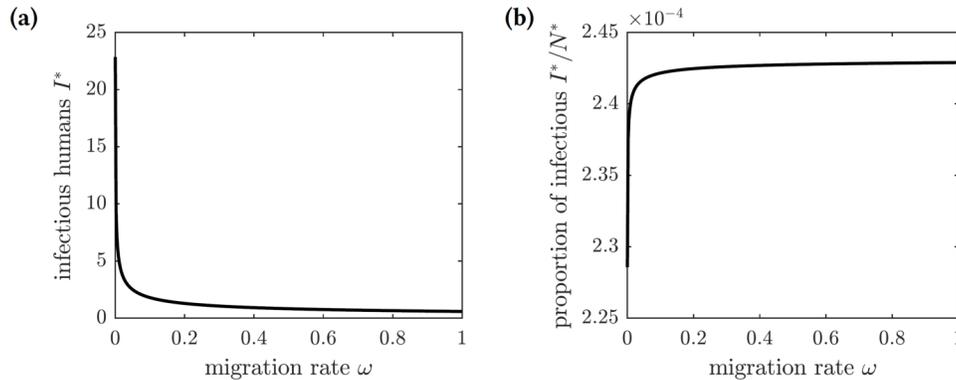


Figure 6. The graphs of the (a) number and (b) proportion of symptomatically infectious individuals in the population as functions of the emigration rate ω . Increased migration levels result in fewer infectious individuals overall but a greater proportion of infectious individuals in the population.

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

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

314 where C_b is the base (fixed) cost of migration, C_s is the scaling cost of migra-
 315 tion, C_i is the cost of the (symptomatic) chikungunya infection, and $\pi(\omega_{\text{pop}})$
 316 is the probability of getting infected given the population emigration rate ω_{pop} .
 317 We assume that the cost of emigration is an increasing function of the migra-
 318 tion rate because of the limited immigration potential of Mauritius: the more
 319 individuals migrate to Mauritius, the harder it becomes to find housing and
 320 jobs. For simplicity, we model the increasing emigration cost as a linear func-
 321 tion of the migration rate. The probability of getting infected and incurring the
 322 cost of a symptomatic chikungunya infection if remaining on Reunion Island
 323 is the transition probability from the susceptible class S to the symptomatically
 324 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.

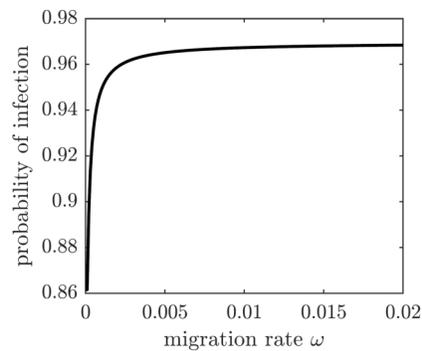


Figure 7. The probability of symptomatic chikungunya infection for a susceptible individual on Reunion Island is increasing with the emigration rate ω .

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

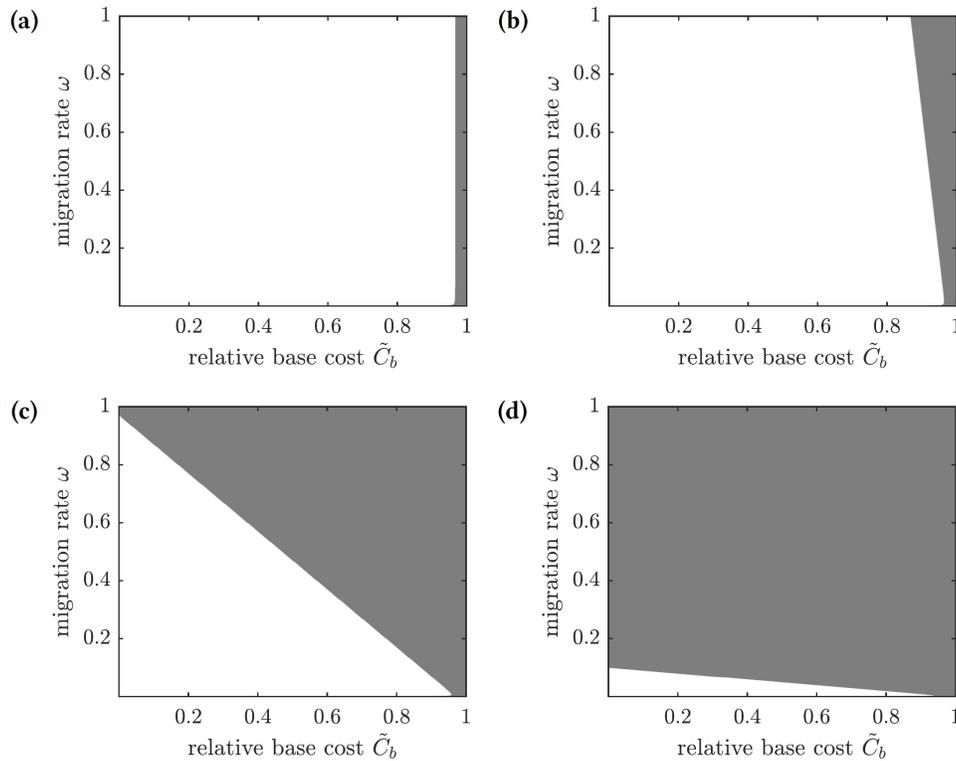


Figure 8. The regions in the (\tilde{C}_b, ω) -parameter space showing whether a focal susceptible individual should emigrate to Mauritius or remain on Reunion. Color code: white—emigrate, gray—stay. **(a)** $\tilde{C}_s = 0$, **(b)** $\tilde{C}_s = 0.1$, **(c)** $\tilde{C}_s = 1$, **(d)** $\tilde{C}_s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

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

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

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

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

- 359 1. For very low relative migration cost ($\tilde{C}_m \leq 0.00022$), higher migration
 360 rates result in smallest overall costs; however, the near-optimal costs
 361 are quickly achieved by small values of migration rate ($\omega = 0.01$)—see
 362 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.

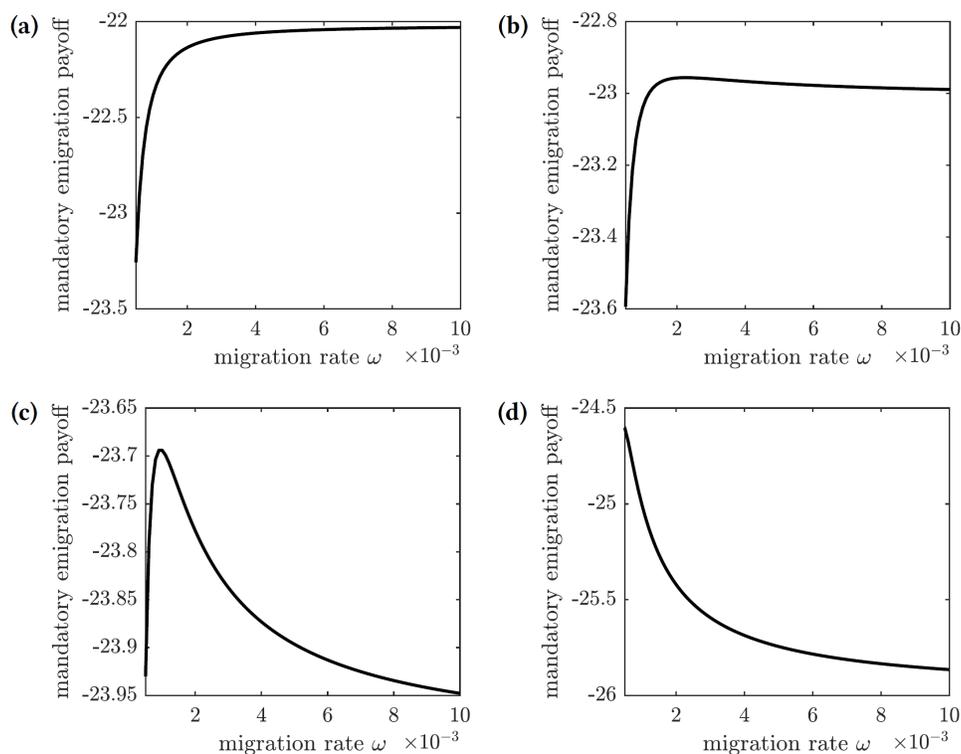


Figure 9. The overall cost of the mandated emigration policy as a function of the migration rate ω . (a) $\tilde{C}_m = 0.00022$, (b) $\tilde{C}_m = 0.00023$, (c) $\tilde{C}_m = 0.00024$, (d) $\tilde{C}_m = 0.00026$.

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 pellant and hence reduce the frequency of mosquito bites, or leave Reunion

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

386 Voluntary participation in the two intervention measures produced oppo-
387 site population-level effects. The more susceptible individuals spray them-
388 selves with insect repellent, the less likely the infectious mosquitoes generate
389 new human infections before they die. Consequently, higher adoption levels
390 of insect repellent usage in the population resulted in lower basic reproduc-
391 tion number values for the disease. Individuals using repellent provide (near)
392 herd-immunity-effect benefits to the entire population. In contrast, if suscep-
393 tible individuals vacated the island, then susceptible mosquitoes were more
394 likely to bite infectious humans as a percentage of the remaining population,
395 thus increasing the disease prevalence among mosquitoes. The remaining sus-
396 ceptible individuals subsequently faced an increased risk of contracting the
397 infection from a mosquito bite. Increased migration levels resulted in drasti-
398 cally elevated basic reproduction number values. Thus, the impact of volun-
399 tary emigration is similar to the tragedy-of-the-commons effect: while being
400 potentially beneficial to specific individuals, it hurts the remaining islanders.

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

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

415 Mandatory emigration from Reunion Island demonstrated that this preven-
416 tive measure made sense for the public benefit only when the cost of relocation
417 was significantly lower than the public cost of infection. Since this mathemat-
418 ical assumption is not likely to hold in practice, the model predicted that it

419 was best to avoid migration of susceptible individuals from the island. The
420 potentially high cost of relocating susceptible individuals away from the epi-
421 demic was not compensated by the minimal decrease in the number of infected
422 individuals.

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

444

5. CONCLUSIONS

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

458 Moreover, our model assumes that the population has reached an equilib-
459 rium with respect to the disease dynamics. But reaching this equilibrium usu-
460 ally occurs on a different timescale compared to individual preventive actions.
461 For example, individuals could be more likely to participate in preventive ef-
462 forts when the epidemic is at its peak rather than when the disease reached
463 the endemic state. A dynamic model where susceptible individuals inform
464 their preventive decisions on the current state of the prevalence of the dis-
465 ease which, in turn, affects the dynamics of the disease transmission, should
466 present a more realistic analysis of selfish individual decisions to prevent the
467 infection.

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475 CONFLICT OF INTEREST

476 All authors declare no conflicts of interest in this paper.

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