THE FREE RIDER PROBLEM IN VACCINATION POLICY AND IMPLICATIONS FOR GLOBAL ERADICATION OF INFECTIOUS DISEASES: A TWO-COUNTRY GAME DYNAMIC MODEL

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abstract. increasingly, an important obstacle to local elimination of vaccine preventable infectious disease is vaccine exemption by individuals under a voluntary vaccination policy, caused by free-riding on herd immunity or by inflated perceptions of vaccine risk. at a global level, this can also prevent global eradication of an infection, since any one country with endemic infection can act as a reservoir, seeding the rest of the world with infections. this also presents challenges to non-governmental third parties that fund and support global eradication efforts through vaccination campaigns, who must choose how to optimally deploy their funds to achieve global eradication in the face of vaccine exemption. behaviour-prevalence models capture the interplay between infection prevalence and disease transmission and have been applied to study free-riding behaviour in vaccination policy before. however, these obstacles to global eradication require a multi-country behaviour-prevalence model that incorporates travel between countries and also incentives by governments and nongovernmental donors. here, we develop a two-country model based on imitation dynamics for a pediatric infectious disease. we analyze the model numerically, showing a range of possible dynamic behaviours such as sustained oscillations in infection prevalence and the abundance of vaccinator strategists, and also parameter regimes where a certain budget allocation by the third party enables elimination in both countries. similar models may be useful in the future as planning tools for international organizations working on the global eradication of certain infectious diseases, such as measles and polio.

1 introduction since the rollout of universal vaccination programmes against many pediatric infectious diseases in the mid-twentieth century,
major strides have been made toward the reduction of disease burden [7]. Smallpox was declared eradicated in 1980, and polio may not be far behind [10, 15]. Ambitious goals have been made for the reduction of mortality due to measles, and many of these goals are being met or exceeded [33].

However, in many countries, the major threat to preventing elimination of vaccine-preventable infections is not public health infrastructure or funding, but deliberate non-vaccinating behaviour on the part of individuals; this is not uncommon in countries with voluntary vaccination policies, or mandatory policies with generous conditions for exemption [19, 27, 30]. Non-vaccinating behaviour could therefore conceivably prevent the global eradication of some diseases, especially measles for example. Individual choice may there be as important a factor to include in the analysis of vaccine uptake determinants as other factors such as logistics and vaccine supply. Conceivably, the incidence of some infectious diseases may eventually be lower in developing countries (where measles mortality is high) than in developed countries (where measles mortality is lower and often under-appreciated), leading to a situation where developed countries seed lesser-developed countries with measles cases through travel (case importation). A third determinant of vaccine uptake across multiple countries is the role of international donor organizations that supply funds to support vaccination programs in lesser-developed countries, such as the World Health Organization and the United Nations Children’s Fund [31].

Behaviour-prevalence models capture the interplay between disease prevalence and individual behaviour, and consist of both a transmission modelling component as well as a model of how individuals make decisions that can influence prevalence, such as whether or not to vaccinate. Modelling the inter-relationship between infection prevalence and individual vaccinating behaviour has attracted attention from both economists and epidemiologists [9, 16, 20], and recent years have seen significant growth in the study of this problem [2, 3, 5, 6, 8, 11, 13, 17, 18, 23, 24, 25, 26, 29, 32]. However, most of these models study behaviour-prevalence dynamics in a single population, and few are concerned with dynamics in separate subpopulations [11, 25]. The role of air transport in spreading infection worldwide is also receiving increasing attention from mathematical modellers [12, 22]. This is particularly true in the aftermath of Severe Acute Respiratory Syndrome (SARS), which spread relatively quickly worldwide by travelling individuals who were incubating the virus.

The increasing role of individual choice as a determinant of vaccine
uptake, and its potential impact on the feasibility of global eradication of infectious diseases, suggests a potential role for behaviour-prevalence models in this area. Using models to address the impact of vaccine exemption on the feasibility of global eradication requires capturing both the travel/immigration links through which case importation occurs, as well as behaviour-prevalence dynamics in multiple subpopulations (countries). Here we develop and numerically analyze a model that captures behaviour-prevalence dynamics in multiple countries, where an international donor has the option of allocating a certain part of their budget to vaccination programmes in any or all of the countries and where case importation can occur through links that represent travel and immigration.

2 Methods  Our behaviour-prevalence model is an extension of a previously published imitation dynamic model for a single population [3], and consists of a submodel for transmission dynamics in two countries connected through travel, and a submodel describing individual vaccinating behaviour occurring through a learning (imitation) process [21]. Also, we assume a third-party payer has a fixed budget for supporting vaccination programmes in the two countries, and the government of each country also has the option of supporting its own vaccination programme. We assume voluntary vaccination programmes for childhood diseases with lifelong, or long-term, natural immunity. We assume that individuals can choose either to vaccinate, or not to vaccinate. The players of the game are parents who decide when their child is born whether or not to vaccinate their baby. The payoff to vaccinate is denoted $e_v$ and the payoff not to vaccinate is $e_n(I)$ which is assumed to be a function of the current disease prevalence $I$ in the population. The payoffs are given by

\begin{align}
  e_v &= -r_v, \\
  e_n(I(t)) &= -r_i m I(t),
\end{align}

where $r_v$ is the perceived probability of significant morbidity from the vaccine, and the payoff for non vaccinators, $e_n(I(t))$ depends on the perceived probability of significant morbidity upon infection, $r_i$, and the probability of becoming infected which increases linearly with the current disease prevalence $I(t)$. The parameter $m$ quantifies the sensitivity of vaccinating behaviour to changes in prevalence. These equations assume a perfectly efficacious vaccine, and they also assume that individuals use a “rule of thumb” to determine their infection risk: the
probability of eventually being infected is proportional to current disease prevalence. Hence, this is a departure from the assumption of perfect rationality often assumed in games.

Individuals choose strategies through an imitation process. Each individual samples other members of the population at a constant rate and switches to their strategy with a probability proportional to the expected gain in payoff, if any, that is accrued by switching strategies. For instance, the payoff gain for a non vaccinator to switch to a vaccinator strategy is

\[ e_v - e_n = -r_v + r_i m I(t) \]  

Under these assumptions, it is possible to write down an equation representing the dynamics of \( x \), the proportion of the population adopting a vaccinator strategy at time \( t \):

\[ \frac{dx}{dt} = kx(1-x)(e_v - e_n(I)) \]

where \( k \) is the imitation (sampling) rate. This equation is explained and derived in more detail in [3].

The quantity \( I(t) \) can be supplied by the standard SIR (Susceptible-Infectious-Recovered) model with vital dynamics, where it is assumed that a fraction \( x \) of individuals born are vaccinated and become immune whereas the unvaccinated remainder enter the susceptible compartment. The resulting combined system of equations is

\[ \frac{dS}{dt} = \mu(1-x) - \beta SI - \mu S, \]

\[ \frac{dI}{dt} = \beta SI - \gamma I - \mu I, \]

\[ \frac{dR}{dt} = \mu x + \gamma I - \mu R, \]

\[ \frac{dx}{dt} = kx(1-x)(-r_v + r_i m I), \]

where \( S \) is the proportion of susceptible individuals, \( I \) is the proportion of infected individuals, \( R \) is the proportion of recovered individuals, \( \mu \) is the mean birth rate per capita, which is assumed to equal the mean death rate per capita, \( \beta \) is the transmission rate, and \( 1/\gamma \) is the mean
duration of infection. Since $R = 1 - S - I$, equation 8 can be eliminated from the system of equations, yielding

$$\frac{dS}{dt} = \mu(1 - x) - \beta SI - \mu S,$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I,$$

$$\frac{dx}{dt} = kx(1 - x)(-r_v + r_imI).$$

These equations are also explained and derived in greater detail in [3].

This basic model will now be extended to describe a situation where a country’s government supports the vaccination programme in its own country for instance through incentivizing vaccination. It will also be extended to describe two countries connected through travel (which can lead to case imports), and where a third-party payer has a fixed budget to devote to support vaccination programmes in either country, for instance through incentivizing vaccination.

We assume that a government supports vaccination in proportion to the current level of infection prevalence in the country, capturing the idea that governments are less pro-active when disease is rare, but make vaccination a priority when disease becomes highly prevalent. We also assume that the third-party payer devotes a fixed proportion of the budget to country 1 and the remainder to country 2, and that this is a constant level of support and does not depend upon short-term outcomes such current infection prevalence in either country. Therefore, the original payoff gain of equation 3 becomes

$$-r_{v1} + r_{r1}m_1I_1 + c_1r_{r1}I_1 + e_1D$$

for country 1, where $c_1$ represents the ability of the government of country 1 to incentivize vaccination, $D$ is the amount of the third-party payer budget devoted to support vaccination in country 1, $e_1$ is how successful a given third-party budget allocation to country 1 is in terms of supporting vaccination, and parameters $r_{r1}$, $r_{v1}$, $m_1$ and $I_1$ are defined as before except now the subscript 1 denotes that their values are specific to country 1. Equation 13 can be simplified to

$$-r_{v1} + \omega_1r_{r1}I_1 + e_1D$$

with $\omega_1 \equiv c_1m_1$. The corresponding equations for country 2 are

$$-r_{v2} + \omega_2r_{r2}I_2 + e_1(B - D)$$
where $B$ is the total budget available to the third-party payer, and where parameters $r_{i2}$, $r_{v2}$, and $I_2$ are defined as before except now the subscript 2 denotes that their values are specific to country 2.

Travel and immigration between countries is represented by a constant rate $b$ per capita. It is assumed that susceptible, infectious and recovered individuals travel at the same rate, although travel rates of infected persons would be lower, in practice, since typically only incubating individuals are well enough to travel. Finally, we also assume that there is a small, constant rate of infection $\alpha$ due to susceptible persons in country 1 and country 2 travelling to other countries that have not yet eliminated measles. The resulting equations for country 1, with the modifications of equations 14 and 15 and the travel terms, are given by

\begin{align}
\frac{dS_1}{dt} &= \mu_1(1 - x_1) - \beta_1 S_1 I_1 - \mu_1 S_1 - bS_1 + bS_2 - \alpha, \\
\frac{dI_1}{dt} &= \beta_1 S_1 I_1 - \gamma_1 I_1 - \mu_1 I_1 - bI_1 + bI_2 + \alpha, \\
\frac{dx_1}{dt} &= k_1 x_1(1 - x_1)(-r_{v1} + e_1 D + \omega_1 r_{i1} I_1) - b x_1 + b x_2,
\end{align}

where $b$ is the travel/immigration rate between country 1 and country 2, and all other parameters are as in Equations 10-12 except now specifically for country 1 (denoted by the subscript 1). Note that individuals in the $x$ compartment also move between countries, representing for instance the immigration or emigration of individuals and their current strategic beliefs about vaccination. The corresponding equations for country 2 are given by

\begin{align}
\frac{dS_2}{dt} &= \mu_2(1 - x_2) - \beta_2 S_2 I_2 - \mu_2 S_2 - bS_2 + bS_1 - \alpha, \\
\frac{dI_2}{dt} &= \beta_2 S_2 I_2 - \gamma_2 I_2 - \mu_2 I_2 - bI_2 + bI_1 + \alpha, \\
\frac{dx_2}{dt} &= k_2 x_2(1 - x_2)(-r_{v2} + e_2 (B - D) + \omega_2 r_{i2} I_2) - b x_2 + b x_1,
\end{align}

where parameters are similarly defined. The model equations 16–21 were analyzed to find out equilibrium and stability and also simulated numerically in MATLAB using ode45, an implementation of the fourth-order Runge-Kutta method.
3 Analysis  Here we characterize and analyze the stability of the model equilibria. As the case importation rate $\alpha$ in either of countries is very small (i.e., of order $10^{-8}$/day), we assume $\alpha = 0$ throughout the analysis.

3.1 Case I: $b = 0$ In this case, the model reduces to the following submodel:

\begin{align}
\frac{dS}{dt} &= \mu(1-x) - \beta SI - \mu S, \\
\frac{dI}{dt} &= \beta SI - \gamma I - \mu I, \\
\frac{dx}{dt} &= kx(1-x)(-r_v + E + \omega_r I),
\end{align}

where $E$ is equal to either $e_1 D$ or $e_2 (B - D)$ depending on country 1 or country 2 respectively.

3.1.1 Equilibria  The model system (22)–(24) has four equilibria:

- Disease-free, pure nonvaccinator equilibrium $E_0 = (0, 0, 0)$.
- Disease-free, pure vaccinator equilibrium $E_1 = (0, 0, 1)$.
- Endemic, pure nonvaccinator equilibrium $E = (\hat{S}, \hat{I}, 0)$, where $\hat{S} = \frac{R_0}{\beta + \delta}$ (say), $\hat{I} = \frac{\mu}{\mu + \gamma} \left(1 - \frac{1}{R_0}\right)$, which exists if $R_0 > 1$.
- Endemic, mixed state of vaccinator & nonvaccinator equilibrium $E^* = (S^*, I^*, x^*)$, where $S^* = \frac{1}{R_0}$, $I^* = \frac{\mu r_v E}{\omega_r}$, $x^* = 1 - \frac{1}{R_0} - \frac{\mu r_v}{\mu + \gamma} I^*$. So, the existence of $E^*$ requires

\begin{align}
R_0 &> \frac{\mu r_v}{\mu r_v - (r_v - E)(\gamma + \mu)} = 1 + \xi + \xi^2 + \cdots, \\
0 &< r_v - E < \frac{\mu r_v}{\gamma + \mu},
\end{align}

where $\xi = \frac{(r_v - E)(\gamma + \mu)}{\mu r_v}$.

3.1.2 Stability  The variational matrix of the linearized system is given by

\[
J = \begin{pmatrix}
-\beta I - \mu & -\beta S & -\mu \\
\beta I & \beta S - \gamma - \mu & 0 \\
0 & kx(1-x)\omega_r & k(1-2x)(-r_v + E + \omega_r I)
\end{pmatrix}.
\]
The characteristic equation for the disease-free equilibrium $E_0$ is

$$ (\lambda + \mu)(\lambda - \beta + \gamma + \mu)\{\lambda - k(-r_v + E)\} = 0. $$

So, $E_0$ is stable if $R_0 < 1$ and $r_v > E$.

Similarly, the characteristic equation for equilibrium $E_1$ is given by

$$ (\lambda + \mu)(\lambda + \gamma + \mu)\{\lambda + k(-r_v + E)\} = 0 $$

Hence, it is stable if $r_v < E$.

The characteristic polynomial around the endemic, pure nonvaccinator equilibrium $E$ is

$$ (\lambda - k(-r_v + E + \omega_r I))[\lambda^2 + \lambda(\beta I + \mu) + \beta I(\gamma + \mu)]. $$

So, $E$ is stable if $(-r_v + E + \omega_r I) < 0$, i.e., if

$$ 0 < r_v - E < \frac{\mu \omega_r}{\gamma + \mu} $$

and

$$ 1 < R_0 < \frac{\mu \omega_r I}{\mu \omega_r I - (r_v - E)(\gamma + \mu)} = 1 + \xi + \xi^2 + \cdots, $$

where $\xi = \frac{(r_v - E)I(\gamma + \mu)}{\mu \omega_r I}$.

The characteristic equation around the endemic equilibrium $E^*$ is

$$ \lambda^3 + a_2 \lambda^2 + a_1 \lambda + a_0 = 0 $$

where

$$ a_2 = (\beta I^* + \mu), $$
$$ a_1 = \beta^2 S^* I^*, $$
$$ a_0 = \mu \beta k I^* x^*(1 - x^*) \omega_r. $$

Note that $a_i > 0$ ($i = 1, \ldots, 3$) if (25)–(26) hold. Now $a_1 a_2 - a_0 = \beta I^*[\beta I^* + \mu]\gamma + \mu - \mu k x^*(1 - x^*) \omega_r]$, which is positive if

$$ x^* < \omega_0, $$

where $\omega_0 = \frac{\mu + \mu}{k \omega_r}$. Thus, by the Routh-Hurwitz stability criterion, $E^*$ is stable if the above conditions hold. Note that (31) reduces to

$$ R_0 < 1 + \xi + \xi^2 + \cdots $$
whenever $0 < \zeta < 1$, $\zeta = \frac{(r_v - E)(\gamma + \mu)}{\mu \omega (1 - \omega \gamma)}$. Equilibrium $E^*$ can destabilize through a Hopf bifurcation, for instance if $k$, $r_i$, or $\omega$ are sufficiently large, giving rise to oscillations in the number of vaccinators and the number of infected persons.

**Theorem 1.** The disease-free, pure nonvaccinator equilibrium $E_0 = (1, 0, 0)$ and the disease-free, pure vaccinator equilibrium $E_1 = (0, 0, 1)$ always exists. $E_1 = (0, 0, 1)$ is stable if $r_v < E$ and $E_0 = (1, 0, 0)$ is stable if $r_v > E$ and $R_0 < 1$. $R_0 > 1$ implies that the endemic, pure non-vaccinator equilibrium $E = (S, I, 0)$ exists and stable if $R_0 < 1 + \xi + \xi^2 + \cdots$, ($\xi < 1$ given by equation 26). Whenever $R_0 > 1 + \xi + \xi^2 + \cdots$, the system switches to the endemic and mixed state of vaccinator and non-vaccinator equilibrium and it is stable if $R_0 < 1 + \zeta + \zeta^2 + \cdots$, ($\zeta < 1$ given by equation 31). Note that $\zeta > \xi$, whenever $\omega_0 < 1$.

### 3.2 Case II: $b \neq 0$

#### 3.2.1 Equilibria

The model equations (16)–(21) have following equilibria:

- Disease-free, pure non-vaccinator equilibrium $E_0 = (1, 0, 0, 1, 0, 0)$.
- Disease-free, pure vaccinator equilibrium $E_1 = (0, 0, 1, 0, 0, 1)$.
- Endemic, pure non-vaccinator equilibrium $E = (S, I, 0, S_2, I_2, 0)$.
- Endemic, mixed state of vaccinator & non-vaccinator equilibrium $E^* = (S_1, I_1, x_1, S_2, I_2, x_2)$.

Analytically it may be difficult to write down explicit expression for $S_1$, $S_2$'s etc., but we can find out them by numerically solving the differential equations (16)–(21).

#### 3.2.2 Stability

The jacobian around $E_0$ is:

$$
J(E_0) = 
\begin{pmatrix}
-\mu_1 - b & -\beta_1 & -\mu_2 & 0 & 0 & 0 \\
\beta_1 & \beta_1 - (\gamma_1 + \mu_1 + b) & 0 & 0 & 0 & 0 \\
0 & b & b & 0 & 0 & 0 \\
b & 0 & 0 & b & 0 & 0 \\
0 & b & 0 & b & 0 & 0 \\
-\mu_2 - b & -\beta_2 & -\mu_2 & 0 & 0 & 0 \\
0 & \beta_2 - (\gamma_2 + \mu_2 + b) & 0 & 0 & 0 & 0 \\
0 & 0 & k_2(-r_{v1} + e_2(B - D)) - b & b & b & b
\end{pmatrix}
$$
which gives the following characteristic equation

\[(32) \quad [\lambda^2 + \lambda(\mu_1 + \mu_2 + 2b) + b(\mu_1 + \mu_2) + \mu_1\mu_2]P_0Q_0 = 0,\]

where

\[P_0 = \lambda^2 + \lambda\{2b - (\beta_1 - \gamma_1 - \mu_1) - (\beta_2 - \gamma_2 - \mu_2)\} - \{b(\beta_1 - \gamma_1 - \mu_1) + \mu_1\mu_2\},\]

\[Q_0 = \lambda^2 + \lambda\{2b + k_1(r_v - e_1D) + k_2(r_v - e_2(B - D))\} + \{bk_1(r_v - e_1D) + bk_2(r_v - e_2(B - D)) + k_1k_2(r_v - e_1D)(r_v - e_2(B - D))\}.\]

So, \(E_0^1\) is stable if

\[\beta_1 < \gamma_1 + \mu_1, \quad \beta_2 < \gamma_2 + \mu_2 \quad \text{and} \quad r_v > e_1D, \quad r_v > e_2(B - D).\]

Again, the Jacobian around \(E_1^1\) is

\[
J(E_1^1) = \begin{pmatrix}
-\mu_1 - b & 0 & -\mu_1 & 0 \\
0 & -(\gamma_1 + \mu_1 + b) & 0 & 0 \\
b & 0 & -k_1(r_v - e_1D) - b & 0 \\
0 & b & 0 & b \\
0 & 0 & b & 0 \\
-\mu_2 - b & 0 & 0 & -\mu_2 \\
0 & -(\gamma_2 + \mu_2 + b) & 0 & 0 \\
0 & 0 & b & 0 \\
0 & 0 & 0 & -k_2(-r_v + e_2(B - D)) - b
\end{pmatrix}
\]

and this gives the following characteristic polynomial:

\[(33) \quad [\lambda^2 + \lambda(\mu_1 + \mu_2 + 2b) + b(\mu_1 + \mu_2) + \mu_1\mu_2]P_1Q_1 = 0,\]

where

\[P_1 = \lambda^2 + \lambda\{2b + (\gamma_1 + \mu_1) + (\gamma_2 + \mu_2)\}
+ \{b(\gamma_1 + \mu_1 + \gamma_2 + \mu_2) + (\gamma_1 + \mu_1)(\gamma_2 + \mu_2)\},\]

\[P_2 = \lambda^2 + \lambda\{2b + k_1(e_1D - r_v) + k_2(e_2(B - D) - r_v)\}
+ \{bk_1(e_1D - r_v) + bk_2(e_2(B - D)D - r_v)\}.
\[ + k_1 k_2 (e_1 D - r_{v1})(e_2 (B - D) D - r_{v2}) \].

So, \( J(E^*_1) \) is stable if
\[ e_1 D > r_{v1} \quad \text{and} \quad e_2 (B - D) > r_{v2} \].

**Theorem 2.** The disease-free, pure nonvaccinator equilibrium \( E^*_0 = (1, 0, 0, 1, 0, 0) \) and the disease-free, pure vaccinator equilibrium \( E^*_1 = (0, 0, 1, 0, 0, 1) \) always exist. \( E^*_1 = (0, 0, 1, 0, 0, 1) \) is stable if \( e_1 D > r_{v1} \) and \( e_2 (B - D) > r_{v2} \). Whenever \( r_{v1} > e_1 D, \ r_{v2} > e_2 (B - D) \), \( E^*_0 = (1, 0, 0, 1, 0, 0) \) is stable if \( \beta_1 < \gamma_1 + \mu_1 \) and \( \beta_2 < \gamma_2 + \mu_2 \) hold.

The stability of other endemic equilibria may be found by numerically solving the model equations.

### 4 Results

The baseline parameter values for this system appear in Table 1. Initial conditions for all simulations were \( S_1 = S_2 = 0.95 \), \( I_1 = I_2 = 0.0001 \), and \( x_1 = x_2 = 0.2 \). We parameterize country 1 to represent the case of a typical developed country, and country 2 to represent the case of a typical developing country. The model structure and parameter values are not intended to be highly realistic, but rather are intended to illustrate certain principles and illustrate the dynamics these types of systems can exhibit. The relative magnitude of \( m_1 \) and \( m_2 \) was based partly on the ratio of healthcare spending between an example developed country (Canada) and an example developing country (Kenya) [35], with \( m_1 > m_2 \) since developed countries have better public health infrastructure and tolerance for disease prevalence is lower, for the same perceived infection risk. Similarly, we assumed \( e_1 < e_2 \) since a given budget buys less incentive in developed countries than developing countries due to differing consumer price index, and the values were based on relative GDP (PPP) for Canada versus Kenya [34]. Infection risk was greater in country 2 and country 1, but vaccine risk was similar since perceived vaccine risk is often inflated in some developed countries where vaccine exemption is common. Other epidemiological parameters were chosen to represent a measles-like infection [3]. In the following figures, baseline parameter values from Table 1 are assumed unless otherwise specified.

The model equations exhibit a wide range of dynamical behaviour, including oscillations in vaccinator strategists \( x \) and infection prevalence \( I \) (Figure 1). For instance, at the parameter values of Figure 1, the
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<td>$\omega_j$</td>
<td>sensitivity of individual vaccinating behaviour and government incentivizing behaviour to infection prevalence, country $j$</td>
<td>$c_1 = 10$, $m_1 = 1$; $c_2 = 0.2$, $m_2 = 1$</td>
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<tr>
<td>$b$</td>
<td>immigration/travel rate</td>
<td>$b = 0.001/yr$</td>
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<tr>
<td>$B$</td>
<td>third-party payer budget</td>
<td>$B = 1$</td>
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<tr>
<td>$D$</td>
<td>portion of budget allocated to country 1</td>
<td>$D = 0.5$</td>
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<tr>
<td>$k_j$</td>
<td>imitation rate, country $j$</td>
<td>$k_j = 6000/yr$</td>
</tr>
<tr>
<td>$e_j$</td>
<td>impact of a given budget allocation by third-party on vaccination incentives, country $j$</td>
<td>$e_1 = 0.000001$; $e_2 = 0.00002$</td>
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<td>$r_{ij}$</td>
<td>risk of significant morbidity upon infection, country $j$</td>
<td>$r_{i1} = 0.01$; $r_{i2} = 0.1$</td>
</tr>
<tr>
<td>$r_{vj}$</td>
<td>risk of significant morbidity upon vaccination, country $j$</td>
<td>$r_{v1} = 0.00001$; $r_{v2} = 0.000015$</td>
</tr>
<tr>
<td>$\beta_j$</td>
<td>transmission rate, country $j$</td>
<td>$\beta_j = 547.5/yr$</td>
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<td>$\gamma_j$</td>
<td>recovery rate, country $j$</td>
<td>$\gamma_j = 0.1/day$</td>
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<td>$\mu_j$</td>
<td>birth/death rate, country $j$</td>
<td>$\mu_j = 0.02/yr$</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>case import rate from other countries</td>
<td>$\alpha = 10^{-8}/day$</td>
</tr>
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**TABLE 1: Parameter values**

The proportion of vaccinators in country 1, $x_1$, is high and relatively stable except for some small oscillations whereas the infection prevalence $I_1$ exhibits extreme outbreaks approximately every 3 years. By comparison, $x_2$ is almost entirely stable and relatively low, while $I_2$ is almost endemic, exhibiting only small oscillations. The increased variability in country 1 relative to country 2 is attributable to the fact that $\omega_1 > \omega_2$. On average, infection is more prevalent (and vaccine coverage is lower) in country 2 than in country 1, but the infection dynamics are less stable in country 1 than in country 2. Despite the large outbreaks visible in country 1, the cumulative number of infections over time in country 2 is almost twice that in country 1.

Within a given country, this system exhibits dynamics similar to those observed in the corresponding 1-country system without additional incentives (Ref. [3]). For instance, increasing the imitation rates $k_1$ and $k_2$,
is destabilizing (produces oscillations), whereas increasing the transmission rates $\beta_1$ or $\beta_2$ is stabilizing (results not shown). Likewise, increasing the parameter $m_1$ representing the sensitivity to disease prevalence in country 1 increases vaccine coverage but also destabilizes dynamics, resulting in oscillations in infection prevalence and vaccinator strategists (Figure 2). Therefore, if individual and government reaction to disease prevalence is based only on current prevalence, higher sensitivity to infection prevalence for a given disease severity will lower long-term prevalence, but may also increase the chance of outbreaks.

![Figure 1: Time series of vaccinators and infection prevalence in both countries. Parameter values are $b = 0.0001/\text{yr}$, $m_1=9$, $k_1=9400/\text{yr}$, $\beta_1=700.5/\text{yr}$, $\gamma_1=0.2/\text{day}$, $m_2=0.5$, $k_2=8000/\text{yr}$, $\gamma_2=0.27/\text{day}$ with other parameters as in Table 1.](image)

The parameter $D$ controls how much of the third-party budget flows to country 1, and by varying its value we can understand how the budget allocation strategy of the third-party payer influences infection prevalence and vaccine uptake in the two countries. For instance, Figure 3 describes the infection prevalence and vaccinator abundance in both countries as a function of $D$. To capture the magnitude of oscillations, the figure depicts the average, minimum and maximum value for each outcome over a 500-year period after transient solutions have decayed. Changes in $D$ generally have more impact on coverage and prevalence in country 2 since $e_2 > e_1$. As $D$ is increased, resulting in support being
FIGURE 2: Time series of infection prevalence (top panel) and vaccinators (bottom panel) in country 1 for various $m_1$ values. Other parameters are as in Table 1. Initial conditions were $S_1 = 0.95$, $I_1 = 0.0001$, $x_1 = 0.2$.

shift away from country 2 and toward country 1, the abundance of vaccinators in country 2 declines from almost 100 % to almost 0 % and the infection prevalence climbs from 0 to 0.0005 (however, prevalence does not start to climb until $D = 0.25$, and the abundance of vaccinators likewise remains high until $D = 0.25$). By comparison, as $D$ is increased and more support flows to country 1, the impact on infection prevalence
and vaccinator abundance in country 1 is relatively small since the same budget allocation “buys” less incentive in country 1 due to its higher GDP per capita and living costs. However, there is a notable window of destabilization for country 1 for $D \in [0.25, 0.65]$. Minimum and maximum values in the left-hand panels of Figure 3 (country 1) for some regimes indicate the presence of oscillations. In other cases, such as most parameter values for the right-hand panels (country 2), the minimum and maximum values are indistinguishable from the average value because oscillations are small or non-existent.

The temporal evolution of vaccinators and disease prevalence after a change in $D$ are also interesting. In the simulation experiment of Figure 4, $D$ is changed from 0 to 1 in the year 2000, representing a shift in support from country 2 to country 1. The impact on country 2 is significant: the abundance of vaccinators begins to decline immediately from 100% although it takes 80 years for the abundance to reach 20%. Initially, prevalence remains low. However, by year 2040, enough susceptible individuals have been recruited to make outbreaks possible. Severe outbreaks continue every 3-5 years thereafter but these eventually approach an endemic equilibrium. The start of outbreaks has little impact on the abundance of vaccinators at these parameter values, which continues to decline. By comparison, the impact of changing $D$ on country 1 is relatively small since the allocation budget of the third-party payer represents a small part of their GDP. However, there is nonetheless some change in the dynamics of country 1 which is apparent in the lower panel of Figure 4. In particular, when $D$ changes to 1 (thus shifting resources to country 1), the abundance of vaccinators builds slightly and infection prevalence declines slightly. This trend continues until the year 2042, when large outbreaks in country 2 begin to increase the magnitude of oscillations in country 1 (due to case importation from country 2 to country 1). Also, the abundance of vaccinators in country 1 begins to decline slightly at this point as a result of increased immigration of non-vaccinators from country 2. The broad conclusion to be drawn from Figure 4 is that, due to imitation dynamics and the need for the susceptible pool to build up before outbreaks are possible, a sudden change in $D$ has little short-term impact on vaccine uptake or prevalence, however, the long-term impacts are significant, especially for country 2 (the developing country).

This model system suggest the possibility for optimization upon $D$, for instance to minimize the overall prevalence of infection in either country. For a parameter regime away from the baseline parameter values, it is even possible for there to be intermediate values of $D$ where
infection is eliminated in both countries, whereas for higher or lower values outside this range, the disease is endemic in one country or the other. Figure 5 presents this case, where infection has been eliminated for $D \in [0.5, 0.6]$. For smaller values of $D$, infection is prevalent in country 1, and for larger values of $D$ infection is prevalence in country 2.
5 Discussion  In summary, we have developed and analyzed a two-country behaviour-prevalence model for voluntary vaccination of a vaccine-preventable disease, where each country’s government supports its vaccination programmes in proportion to the current infection prevalence, and where a third-party international organization has a budget which it can divide among the two countries to support their vaccination programmes. The two countries were parameterized to represent a typi-
The model exhibits a wide range of dynamic behaviour, including oscillations (Figure 1) and long-term dynamics in response to sudden changes in parameters (Figure 4). Changing parameters such as the budget allocated to country 1 ($D$) not only influences the average prevalence and abundance of vaccinator strategists, but also the stability...
FIGURE 5: Plots of average, minimum and maximum infection prevalence $I_1$ and $I_2$ in country 1 and 2, and vaccinator abundance $x_1$ and $x_2$ in country 1 and 2, as a function of budget allocation $D$ to country 1 in a parameter regime where elimination is possible. Parameter values are $e_1 = 0.00001$, $r_{v1} = 0.000005$, $r_{v2} = 0.000008$, $r_{i2} = 0.15$. Other parameters are as in Table 1.

of the dynamics. For certain parameter regimes, there is an intervals of values of $D$ for which elimination is possible in both countries, whereas for higher or lower values of $D$, the infection is endemic in one country or the other, and hence the country where infection is endemic can continue seeding country where elimination has been achieved.

Time series of case reports for many infectious diseases, including measles, exhibit recurrent, oscillatory outbreaks. Explaining these recurrent outbreaks has been the subject of much previous research, and it has been noted that basic compartmental measles models do not produce sustained oscillations unless additional mechanisms, such as time delays, demographic stochasticity, or seasonal variation in transmission rates, are included [1, 4, 14, 28]. This model illustrates how nonlinearities due to human behaviour are another mechanism by which oscillations may be sustained.

As with any model, this model has several limitations. The role of stochasticity near the elimination threshold cannot be captured with deterministic equations. Closer attention would have to be paid to parameterization in order for the model to be useful for policy decisions, and the model would have to be extended to multiple countries. Addi-
tionally, we relied upon numerical analysis because of the relatively high
dimension and nonlinearity of the system.

However, this preliminary analysis suggests it is possible to construct
models which can be used to address the feasibility of global eradication
of vaccine-preventable infections in situations where individual choice
is a driver of vaccine uptake in one or more countries, and where all
countries are interconnected through transportation. Such models can
be extended in ways that make them more realistic, or so that the models
could address closely related issues such as cross-border externalities.
Eventually, such models may also be useful to international decision-
makers for deciding how to optimize budget allocation among various
countries to achieve global eradication of a vaccine-preventable infection.

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