Research Article

Modeling the Dynamics of an Epidemic under Vaccination in Two Interacting Populations

Ibrahim H. I. Ahmed, Peter J. Witbooi, and Kailash Patidar

Department of Mathematics and Applied Mathematics, University of the Western Cape, Private Bag X17, Bellville 7535, South Africa

Correspondence should be addressed to Peter J. Witbooi, pwitbooi@uwc.ac.za

We present a model for an SIR epidemic in a population consisting of two components—locals and migrants. We identify three equilibrium points and we analyse the stability of the disease free equilibrium. Then we apply optimal control theory to find an optimal vaccination strategy for this 2-group population in a very simple form. Finally we support our analysis by numerical simulation using the fourth order Runge-Kutta method.

1. Introduction

Mathematical modeling of the numerical evolution of infectious diseases has become an important tool for disease control and eradication when possible. Much work has been done on the problem of how a given population is affected by another population when there is mutual interaction. The mere presence of migrant people poses a challenge to whatever health systems are in place in a particular region. Such epidemiological phenomena have been studied extensively, described by mathematical models with suggestions for intervention strategies. The epidemiological effect of migration within the population itself was modeled for sleeping sickness in a paper [1] by Chalvet-Monfray et al. In the case of malaria, there is for instance a study [2] by Tumwiine et al. on the effect of migrating people on a fixed population. The latter two diseases are vector borne. Diseases that propagate without a vector spread perhaps more easily when introduced into a new region. Various studies of models with immigration of infectives have been undertaken for tuberculosis, see for instance [3] by Zhou et al., or the work [4] of Jia et al., and for HIV, see the paper [5] of Naresh et al.

A very simple compartmental model of an epidemic would be an autonomous system comprising a system of two or three differential equations, such as, for instance, the model of

Kermack and McKendrick. There are more sophisticated models that allow for an incubation period for the pathogen after entering the body of a host. One of the ways of dealing with this phenomenon is by way of delay differential equations, for instance, in the papers [6] of De la Sen et al. and [7] of Li et al. Another way of handling an incubation period is by introducing another compartment. A comparison of these two approaches can be found in the work [8] of Kaddar et al. Other models allow for certain entities such as force of infection or incidence rate to be nonconstant. Such a model, in both a deterministic and a stochastic version, is considered in [9] by Lahrouz et al.

In this paper, we study a disease of the SIR type, prevailing in a population that can be regarded as consisting of two subpopulations. We compare it with similar models existing in the literature. We study stability of equilibrium solutions and optimal roll out of the vaccination. Such a study, in the case of a homogeneous population, was done in [10] by Zaman et al. For more complex population structures, there is a study by Piccolo and Billings [11]. A model similar to that of Piccolo and Billings has been studied in a stochastic setting in the work [12] of Yu et al. In [12], such a population is being referred to as a *two-group* population. A model of SEIR type for such a diversified population was proposed in [4] by Jia et al. In the latter paper, they analyse stability of solutions, but they do not consider vaccination. Our paper aims to follow the approach of [4], but for the SIR case and to include vaccination. Some papers have addressed epidemic models with pulse vaccination, for instance, an SIR model with pulse vaccination strategy to eradicate measles is presented in [13] by Agur et al. A model of an SIR epidemic in a two-group population, separated by age, is presented in the paper [14] of Acedo et al. They present a vaccination strategy similar to that in [13]. Much work in pulse vaccination has been done following on and inspired by [13]. However, two different diseases of SIR type may require completely different strategies for effective control of the disease. In this paper, we cater for those diseases for which pulse vaccination is not the best solution. We will assume the so-called proportional vaccination. A very interesting control problem is solved in the paper [15] of Tchuenche et al. In [15], the control vector is 3-dimensional, providing for a two-dimensional control on vaccination and a control on treatment. In the current paper, the control problem and its solution follow more closely along the lines of [10]. We obtain a simplification over [10] by observing that some of the pivotal costate variables vanish.

This paper is organized as follows. In Section 2, we formulate the model by way of a system of six ordinary differential equations. Then, we analyse the disease-free equilibrium and derive the threshold parameters in Section 3. In Section 4, we consider the optimal control problem, controlling vaccination on both the locals and the migrants. The percentages of susceptibles being vaccinated are taken as the control variables. We include a simulation. Finally, Section 5 has concluding remarks and offers a brief outlook on further research possibilities.

2. Model Formulation

To study the transmission of a disease in two interacting populations, we consider the total population with size N, as being divided into two subpopulations, the *migrant* subpopulation of size M, and the *local* subpopulation of size L. We assume that each subpopulation size is constant (the rate of birth equals the mortality rate) and that the population is uniform and homogeneously mixing. Divide each subpopulation into disjoint classes called the susceptible class (S), the infectious class (I), and the class of the removed (R). Thus, there will be three such classes for the local population and also three classes for the migrant population.
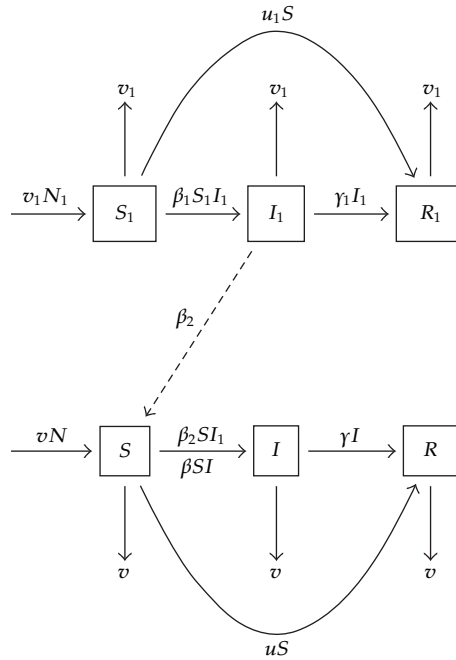
Figure 1: Flow chart of two interacting populations.

The sizes of these classes change with time and will be denoted by $S_0(t)$, $I_0(t)$, $R_0(t)$, $S_1(t)$, $I_1(t)$, and $R_1(t)$. Let us agree henceforth to suppress the subscript $(_0)$ for local population, writing simply $S(t)$ instead of $S_0(t)$, and so on.

The model is described by a system of six differential equations as follows. The schematic diagram depicted in Figure 1 illustrates the model and informs the differential equations. We note that the first three equations in (2.1) constitute an SIR model as, for instance, in the paper [10] by Zaman et al. Let us normalize the variables, using the new variables $s_1 = S_1/M$, $i_1 = I_1/M$, $r_1 = R_1/M$, $s = S/L$, $i = I/L$ and $r = R/L$. After normalizing our model, which we shall refer to as model (2.1) and (2.2), becomes as follows:

$$\frac{ds_1(t)}{dt} = v_1 - (v_1 + u_1(t))s_1(t) - \beta_1 i_1(t)s_1(t),$$

$$\frac{di_1(t)}{dt} = \beta_1 i_1(t)s_1(t) - (\gamma_1 + v_1)i_1(t), \tag{2.1}$$

$$\frac{dr_1(t)}{dt} = \gamma_1 i_1(t) - v_1 r_1(t) + u_1(t)s_1(t),$$

$$\frac{ds(t)}{dt} = v - (v + u(t))s(t) - \beta i(t)s(t) - \beta_2 i_1(t)s(t),$$

$$\frac{di(t)}{dt} = \beta i(t)s(t) + \beta_2 i_1(t)s(t) - (\gamma + v)i(t), \tag{2.2}$$

$$\frac{dr(t)}{dt} = \gamma i(t) - v r(t) + u(t)s(t).$$

Here v_1 and v are the mortality rate (equal to the birth rate) in the migrant subpopulation, and the local subpopulation, respectively. The functions $u_1(t)$ and $u(t)$ are the percentages of susceptible individuals being vaccinated in the respective subpopulations per unit time. Individuals enter the recovered compartment at rates γ_1 and γ for the respective subpopulations. Also β_1 and β are the transmission coefficients from the susceptible compartment into the infectious, for the migrant subpopulation and the local subpopulation, respectively. The transmission coefficient from migrants to locals is denoted by β_2. The term $\beta_2 i_1 s$ models the influence of the migrant subpopulation onto the locals as in the paper [4] of Jia et al.

In the normalized system above, the sizes of the two groups in the population are not visible. At least we should be aware of their relative sizes. In particular, the weighting constant c_0 must be in step with the ratio M/L. The feasible region for the system is the following set:

$$\Omega = \left\{ X \in \mathbb{R}_+^6 : X_1 + X_2 + X_3 = 1, \ X_4 + X_5 + X_6 = 1 \right\}. \tag{2.3}$$

3. Equilibria and Their Stability

Equilibrium points are time-independent solutions to the given system of equations. Therefore, in this subsection, we assume $u_1(t)$ and $u(t)$ to be constant functions, $u_1(t) \equiv u_1$ and $u(t) \equiv u$. Stability properties of the equilibria are closely linked with the numbers

$$K_1 = \frac{\beta_1 v_1}{(v_1 + u_1)(\gamma_1 + v_1)}, \qquad K = \frac{\beta v}{(v + u)(\gamma + v)}. \tag{3.1}$$

We shall prove that the basic reproduction ratio is the number $R_{u,u_1} = \max\{K, K_1\}$. This will follow from Proposition 3.3.

Notation 1. If u and u_1 are both identically zero, then R_{u,u_1} will be written as R_0. For an equilibrium point E, the coordinates will be denoted by E_s, E_{s_1}, and so on.

Remark 3.1. Suppose that in the model (1a, 1b) in [4] of Jia et al., we make the following modifications, transforming the model into SIR: replace the compartments E_M and I_M by a single compartment J_M, and similarly replace E_L and I_L by a single J_M,

Then the model takes the same form as our model (2.1) and (2.2), if in (2.1) and (2.2) we put $u(t) \equiv 0$, $u_1(t) \equiv 0$ and $v_1 = v$.

We take advantage of the aforementioned equivalence in presenting our next theorem.

Theorem 3.2. *Let one consider the unvaccinated version of model* (2.1) *and* (2.2), *that is, with* $u(t) \equiv 0$ *and* $u_1(t) \equiv 0$, *and let us further assume that* $v_1 = v$.

If $R_0 < 1$, *then the disease-free equilibrium* F *with* $F_s = 1$ *and* $F_{s_1} = 1$ *exists and is globally stable.*

Proof. In view of Remark 3.1, this theorem is a direct consequence of [4, Theorem 1]. □

Turning to the more general model (2.1) and (2.2), with vaccination and without the assumption $v_1 = v$, we can identify three possible equilibrium points.

Proposition 3.3. (a) *If $R_{u,u_1} < 1$, then the disease-free equilibrium F is locally asymptotically stable and its coordinates are*

$$F_{s_1} = \frac{v_1}{v_1 + u_1}, \qquad F_{i_1} = 0, \qquad F_{r_1} = \frac{u_1}{v_1 + u_1},$$

$$F_s = \frac{v}{v + u}, \qquad F_i = 0, \qquad F_r = \frac{u}{v + u}. \tag{3.2}$$

(b) *If $K_1 < 1$ and $K > 1$, then there is a unique feasible equilibrium B with*

$$B_{s_1} = \frac{v_1}{v_1 + u_1}, \qquad B_{i_1} = 0, \qquad B_{r_1} = \frac{u_1}{v_1 + u_1},$$

$$B_s = \frac{\gamma + v}{\beta}, \qquad B_i = \frac{v}{\gamma + v}\left(1 - \frac{1}{K}\right), \qquad B_r = 1 - B_s - B_r. \tag{3.3}$$

(c) *The endemic equilibrium D has coordinates as follows:*

$$D_{s_1} = \frac{\gamma_1 + v_1}{\beta_1}, \qquad D_{i_1} = \frac{v_1}{\gamma_1 + v_1}\left(1 - \frac{1}{K_1}\right),$$

$$D_{r_1} = 1 - D_{s_1} - D_{r_1}, \tag{3.4}$$

D_s is a root x of the quadratic polynomial $P(x) = C_2 x^2 + C_1 x + C_0$ with

$$C_0 = \beta_1 v^2 v_1 + \beta_1 v^2 \gamma_1 + \beta_1 \gamma \gamma_1 v + \beta_1 \gamma v_1 v,$$

$$C_1 = C_0 + \beta_2 v_1 \gamma_1 \gamma + \beta_2 v_1^2 \gamma - \beta_2 v_1 \beta_1 v - \beta_2 v_1 \beta_1 \gamma - \beta_1 \gamma \gamma_1 u - \beta_1 \beta \gamma_1 v$$

$$+ \beta_2 u_1 v_1 v + \beta_2 v_1 \gamma_1 v + \beta_2 v_1^2 v - \beta_1 \beta v_1 v - \beta_1 v v_1 u - \beta_1 \gamma v_1 u - \beta_1 v \gamma_1 u$$

$$+ \beta_2 u_1 (v_1 \gamma + \gamma_1 \gamma + \gamma_1 v), \tag{3.5}$$

$$C_2 = \beta_1 \beta (\gamma_1 + v_1)(v + u),$$

$$D_i = \frac{v - (u + v)s}{v + \gamma}, \qquad D_r = 1 - D_s - D_i.$$

Proof. The given points $F, D, B \in \mathbb{R}^6$ clearly are equilibrium solutions, which may or may not be feasible.

(a) The Jacobian associated with the system (2.1) and (2.2) at point F is

$$W = \begin{pmatrix} a_1 & 0 & 0 & 0 & 0 & 0 \\ \beta_1 i_1 & b_1 & 0 & 0 & 0 & 0 \\ -u_1 & \gamma_1 & -v_1 & 0 & 0 & 0 \\ 0 & \beta_2 s & 0 & a - \beta_2 i_1 & -\beta s & 0 \\ 0 & \beta_2 s & 0 & \beta i + \beta_2 i_1 & c & 0 \\ 0 & 0 & 0 & u & \gamma & -v \end{pmatrix}, \tag{3.6}$$

where

$$a_1 = -v_1 - u_1 - \beta_1 i_1, \qquad b_1 = \beta_1 s_1 - \gamma_1 - v_1, \qquad c = \beta s - \gamma - v. \tag{3.7}$$

We set out to find the eigenvalues of W. This amounts to solving for λ in the equation,

$$q_1 \cdot (\lambda + v_1) \cdot q_2 \cdot (\lambda + v) = 0, \tag{3.8}$$

where $q_1(\lambda)$ and $q_2(\lambda)$ are the quadratic expressions below:

$$q_1 = (\lambda + v_1 + u_1 + \beta_1 i_1)(\lambda - \beta_1 s_1 + \gamma_1 + v_1) + \beta_1^2 i_1 s_1, \tag{3.9}$$

$$q_2 = (\lambda - \beta s + \gamma + v)(\lambda + v + u + \beta i + \beta_2 i_1) + \beta^2 s i + \beta\beta_2 i s. \tag{3.10}$$

Now from (3.9) we can write q_1 in the form

$$q_1 = \lambda^2 + A_1 \lambda + A_2, \tag{3.11}$$

where A_1 and A_2 are the constants:

$$A_1 = v_1 + u_1 + \beta_1 i_1 - \beta_1 s_1 + \gamma_1 + v_1,$$
$$A_2 = (v_1 + u_1 + \beta_1 i_1)(\gamma_1 + v_1 - \beta_1 s_1) + \beta_1^2 i_1 s_1. \tag{3.12}$$

Substituting the equilibrium values (at the point F) of s_1, i_1, s and i, we can rewrite

$$A_1 = v_1 + u_1 - \frac{\beta_1 v_1}{v_1 + u_1} + \gamma_1 + v_1,$$
$$A_2 = (v_1 + u_1)\left(\gamma_1 + v_1 - \frac{\beta_1 v_1}{v_1 + u_1}\right). \tag{3.13}$$

The roots of q_1 have negative real parts if both A_1 and A_2 are positive. Now we note that A_2 is positive if and only if

$$\gamma_1 + v_1 - \frac{\beta_1 v_1}{v_1 + u_1} > 0, \tag{3.14}$$

that is,

$$K_1 = \frac{\beta_1 v_1}{(v_1 + u_1)(v_1 + \gamma_1)} < 1. \tag{3.15}$$

If $K_1 < 1$, then also $A_1 > 0$. From (3.10) we have q_2 as follows:

$$q_2 = \lambda^2 + (v + u + \beta i + \beta_2 i_1 - \beta s + \gamma + v)\lambda + (\gamma + v - \beta s)(v + u + \beta i + \beta_2 i_1) + \beta^2 si + \beta\beta_2 si.$$

(3.16)

Now let us define the coefficients Q_1 and Q_2 as

$$Q_1 = (v + u + \beta i + \beta_2 i_1 - \beta s + \gamma + v),$$

$$Q_2 = (\gamma + v - \beta s)(v + u + \beta i + \beta_2 i_1) + \beta^2 si + \beta\beta_2 si.$$

(3.17)

By applying a similar analysis as for q_1, we find that the roots of (3.16) have negative real parts if and only if both Q_1 and Q_2 are positive, which is equivalent to the condition

$$K = \frac{\beta v}{(v + u)(\gamma + v)} < 1.$$

(3.18)

Therefore, the disease-free equilibrium is locally asymptotically stable if $K_1 < 1$ and $K < 1$, that is, when $R_{u,u_1} < 1$.

((b) and (c)): The points are obtained by direct computation. Feasibility of B is clear when K and K_1 are as given in (b). □

We include a computational example of an endemic equilibrium point D.

Example 3.4. Let us choose parameter values:

$$(v, \beta, \gamma, u, v_1, \beta_1, \gamma_1, u_1, \beta_2) = (0.11, 0.40, 0.09, 0.5, 0.15, 0.55, 0.05, 0.25, 0.3).$$

(3.19)

Then we obtain $K_1 = 1.03125$ and D has coordinates:

$$D_{s_1} = 0.36364, \qquad D_{i_1} = 0.02273, \qquad D_s = 0.17726, \qquad D_i = 0.00936.$$

(3.20)

We note that $P(x)$ also has a root $x = 0.50866$, but this is not a feasible value for D_s since the corresponding D_i value $(= -1.00141)$ is negative.

In line with the terminology of [4], we shall refer to the point B as a *boundary* equilibrium. Stability analysis of the points B and D would take more effort than in the case of F, and could distract from the main purpose of this paper.

4. Optimal Vaccination Strategy

We wish to design optimal vaccination strategies $u^*(t)$ and $u_1^*(t)$, respectively, for the local population and the migrant population. We have six state variables $s_1(t)$, $s(t), \ldots, r(t)$. The variable $u(t)$ denotes the percentage of susceptible individuals being vaccinated per unit of time in the local population, and $u(t)$ is assumed to be bounded, $0 \leq u(t) \leq \alpha \leq 1$. A similar

interpretation holds for $u_1(t)$, and we assume that for some constant α_1, $0 \leq u_1(t) \leq \alpha_1 \leq 1$. Our optimal control problem amounts to minimizing the objective function below

$$J(u(t), u_1(t)) = \int_0^T \left[i(t) + c_0 i_1(t) + cu^2(t) + c_1 u_1^2(t) \right] dt, \tag{4.1}$$

where c_0, c, and c_1 are positive weighting constants. The integral in the objective function can be regarded as follows. The first two terms in the integrand represent the suffering, the lost working hours, the cost of hospitalization, and so on, due to infections. The other two terms represent the cost of vaccination. Similar objective functions are considered in the book [16] of Lenart and Workman and in, for instance, the paper [10] of Zaman et al. Our problem is then as follows.

Problem 1. Minimize $J(u(t), u_1(t))$ subject to the system (2.1) and (2.2) of differential equations, together with the initial conditions

$$s_1(0) = s_{10} \geq 0, \qquad i_1(0) = i_{10} \geq 0, \qquad r_1(0) = r_{10} \geq 0,$$
$$s(0) = s_0 \geq 0, \qquad i(0) = i_0 \geq 0, \qquad r(0) = r_0 \geq 0, \tag{4.2}$$

and terminal conditions, $s_1(T)$, $i_1(T)$, $r_1(T)$, $s(T)$, $i(T)$, and $r(T)$ are free, while the control variables are assumed to be measurable functions that are bounded above

$$0 \leq u(t) \leq \alpha \leq 1, \qquad 0 \leq u_1(t) \leq \alpha_1 \leq 1. \tag{4.3}$$

The Hamiltonian for this problem is as follows:

$$\begin{aligned}
H(t, s_1, i_1, r_1, s, i, r, u, \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6) = {}& i(t) + c_0 i_1(t) + cu(t)^2 + c_1 u_1(t)^2 \\
& + \lambda_1(t) \left(v_1 - (v_1 + u_1(t)) s_1(t) - \beta_1 i_1(t) s_1(t) \right) \\
& + \lambda_2(t) \left(\beta_1 i_1(t) s_1(t) - (\gamma_1 + v_1) i_1(t) \right) \\
& + \lambda_3(t) \left(\gamma_1 i_1(t) - v_1 r_1(t) + u_1(t) s_1(t) \right) \\
& + \lambda_4(t) \left(v - (v + u(t)) s(t) - \beta i(t) s(t) - \beta_2 i_1(t) s(t) \right) \\
& + \lambda_5(t) \left(\beta i(t) s(t) + \beta_2 i_1(t) s(t) - (\gamma + v) i(t) \right) \\
& + \lambda_6(t) \left(\gamma i(t) - v r(t) + u(t) s(t) \right).
\end{aligned} \tag{4.4}$$

In the theorem below, the controls, the state variables, and the costate variables are functions of time. However, notationally this dependence will be suppressed except when required explicitly. The upper dot denotes the time derivative.

Theorem 4.1. *An optimal solution for Problem 1 exists. An optimal solution satisfies the identity*

$$\lambda_3(t) = 0 = \lambda_6(t) \quad \forall 0 \leq t \leq T, \tag{4.5}$$

and also satisfies the following system of differential equations:

$$\dot{\lambda}_1 = \lambda_1(v_1 + u_1 + \beta_1 i_1) - \lambda_2 \beta_1 i_1,$$

$$\dot{\lambda}_2 = -c_0 + \lambda_1 \beta_1 s_1 - \lambda_2(\beta_1 s_1 - \gamma_1 - v_1) + \lambda_4 \beta_2 s - \lambda_5 \beta_2 s,$$

$$\dot{\lambda}_4 = \lambda_4(v + u + \beta i + \beta_2 i_1) - \lambda_5(\beta i + \beta_2 i_1),$$

$$\dot{\lambda}_5 = -1 + \lambda_4 \beta s - \lambda_5(\beta s - \gamma - v),$$

(4.6)

with transversality conditions:

$$\lambda_1(T) = 0, \quad \lambda_2(T) = 0, \quad \lambda_4(T) = 0, \quad \lambda_5(T) = 0. \tag{4.7}$$

Furthermore, the optimal vaccination strategy is given by

$$u^*(t) = \min\left(\max\left(\frac{\lambda_4^*(t)s^*(t)}{2c}, 0\right), \alpha\right),$$

$$u_1^*(t) = \min\left(\max\left(\frac{\lambda_1^*(t)s_1^*(t)}{2c_1}, 0\right), \alpha_1\right).$$

(4.8)

Proof. Existence of a solution follows since the Hamiltonian is convex with respect to $u(t)$ and $u_1(t)$. We check the first-order conditions for this optimization problem. We calculate the partial derivatives of the Hamiltonian with respect to the different state variables, in order to obtain the time derivatives $\dot{\lambda}_i(t)$ of the costate variables. Due to $s_1(T)$, $i_1(T)$, $r_1(T)$, $s(T)$, $i(T)$ and $r(T)$ being free, the following terminal conditions hold:

$$\lambda_1(T) = 0, \quad \lambda_2(T) = 0, \quad \lambda_3(T) = 0, \quad \lambda_4(T) = 0, \quad \lambda_5(T) = 0, \quad \lambda_6(T) = 0. \tag{4.9}$$

We start off by observing that,

$$\dot{\lambda}_3(t) = -\frac{\partial H}{\partial r_1} = -v_1 \lambda_3(t), \qquad \dot{\lambda}_6(t) = -\frac{\partial H}{\partial r} = -v \lambda_6(t). \tag{4.10}$$

This implies that $\lambda_3(t)$ and $\lambda_6(t)$ are of the form

$$\lambda_3(t) = Ae^{-v_1 t}, \qquad \lambda_6(t) = Be^{-vt}, \tag{4.11}$$

for some constants A and B, respectively. The terminal conditions $\lambda_3(T) = 0$ and $\lambda_6(T) = 0$, forces A and B to vanish. Therefore, λ_3 and λ_6 are identically zero, that is, $\lambda_3(t) \equiv 0$ and $\lambda_6(t) \equiv 0$ as claimed in the theorem.

Now we calculate

$$\dot{\lambda}_1(t) = -\frac{\partial H^*}{\partial s_1}, \qquad \dot{\lambda}_2(t) = -\frac{\partial H^*}{\partial i_1}, \qquad \dot{\lambda}_4(t) = -\frac{\partial H^*}{\partial s}, \qquad \dot{\lambda}_5(t) = -\frac{\partial H^*}{\partial i}, \tag{4.12}$$

and we obtain the equations as asserted in the theorem.

We now turn to the final part of the proof, which is about the form of the controls, $u^*(t)$ and $u_1^*(t)$. The function $u^*(t)$ must optimize H. So we calculate

$$\frac{\partial H}{\partial u} = 2cu - \lambda_4 s. \tag{4.13}$$

Consider a fixed value of t. Now if $2cu(t) - \lambda_4(t)s(t)$ is zero for some value of $u(t)$ in $[0, \alpha]$, then the given value of $u(t)$ is optimal. If for every number $\overline{u} \in [0, \alpha]$, we have

$$2c\overline{u} - \lambda_4(t)s(t) \geq 0 \quad (\text{resp.,} \quad 2c\overline{u} - \lambda_4(t)s(t) \leq 0), \tag{4.14}$$

then we must choose $u(t) = 0$ (resp., $u(i) = \alpha$). Thus, we must have

$$u^*(t) = \min\left(\max\left(\frac{\lambda_4^*(t)s^*(t)}{2c}, 0\right), \alpha\right). \tag{4.15}$$

The function $u_1^*(t)$ also must optimize H, and by a similar argument we obtain the stated expression for $u_1^*(t)$. □

Numerical Simulation

We present two simulations in the examples below, and we use the Runge-Kutta fourth-order method. For both of these examples, we use the same parameter values, but the initial conditions on the state variables will be different. The parameter values are as follows:

$$c_0 = 1; \quad c = 0.3; \quad c_1 = 0.2; \quad g = 0.4; \quad d = 0.0222; \quad \beta = 0.09; \quad \beta_1 = 0.12; \quad \beta_2 = 0.02;$$

$$\alpha = 0.7; \quad \alpha_1 = 0.8; \quad \mu_1 = 0.0222; \quad \gamma_1 = 0.3; \quad T = 300. \tag{4.16}$$

The time horizon of a control problem in epidemiology is usually dependent on economic factors such as budgeting, biological, and medical considerations, or even maybe influenced by political dynamics. For the purpose of our illustrative examples, the chosen value of T is nominal.

Example 4.2. Consider the initial conditions

$$s(0) = 0.7; \quad i(0) = 0.28; \quad r(0) = 0.02; \quad s_1(0) = 0.7; \quad i_1(0) = 0.25; \quad r_1(0) = 0.05. \tag{4.17}$$

We note that if both groups have the infection on a significant scale, then the optimal strategy is to vaccinate in both groups on a comparable scale. The optimal vaccination rollouts for the two groups are similar in form (Figures 2, 3, and 4).

Example 4.3. In this case we assume at time $t = 0$ to have the local population to be infection-free. We consider the initial conditions

$$s(0) = 1; \quad i(0) = 0; \quad r(0) = 0; \quad s_1(0) = 0.7; \quad i_1(0) = 0.25; \quad r_1(0) = 0.05. \tag{4.18}$$
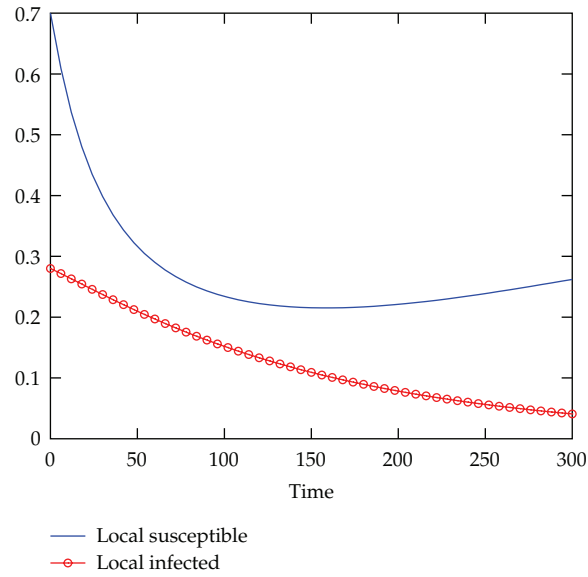
Figure 2: This plot shows the proportions of susceptible and infected individuals in the local subpopulation, according to Example 4.2.
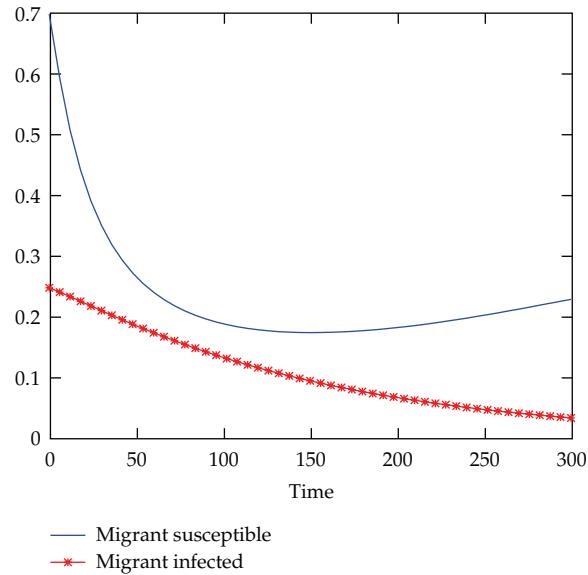

Figure 3: For the case of Example 4.2, we show the proportions of susceptible and infected individuals in the migrant group.

Naively, one would expect to see that in such a case the optimal strategy should be to vaccinate the migrants at much higher ratios than the locals. Our simulation reveals that although the initial infection on locals is zero, it is optimal to immediately start on vaccination of the locals whenever there are infected migrants (Figure 5).
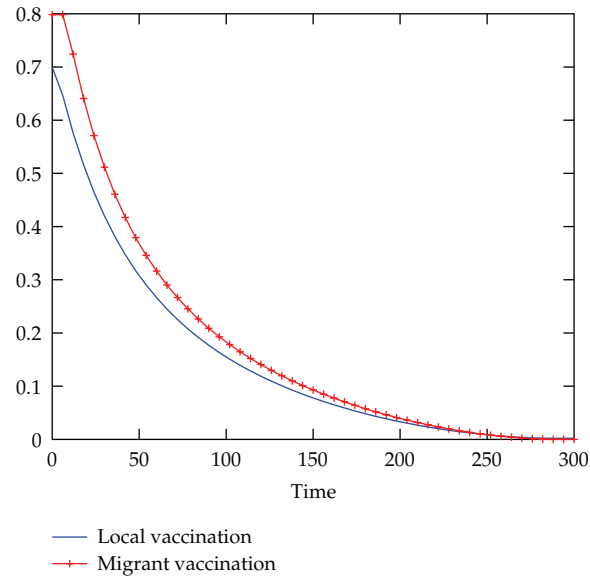
Figure 4: The vaccination strategies for the two groups, Example 4.2.
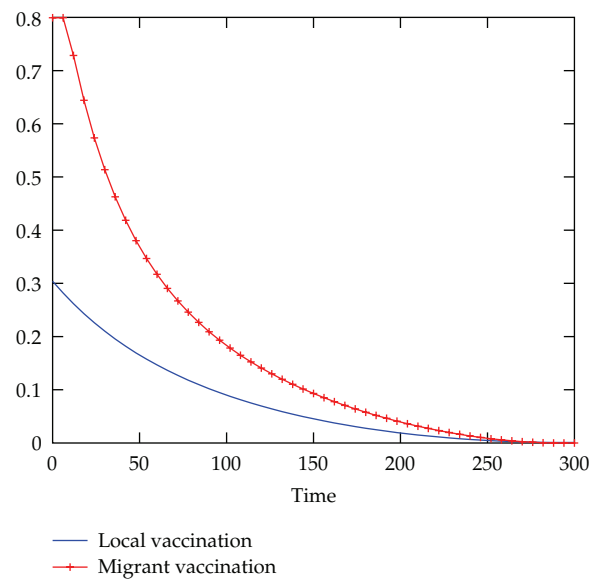
Figure 5: The vaccination strategies for the two groups, Example 4.3.

We conclude this section with a comparison of the values of the objective functional, comparing cases of contant vaccination with optimal (and variable) vaccination strategies.

Example 4.4. In Table 1 we use model parameters as for the simulations of Examples 4.2 and 4.3. Only the state variables and the controls are different. We take the values $s(0) = 1$ and $i(0) = 0$ as fixed (with $r = 1 - s - i$), and we select values for $s_1(0)$ and $i_1(0)$ as indicated in the table. For comparison with the case of optimal control, we consider the constant values

Table 1

s_1	i_1	$J(u^*, u_1^*)$	$J(\overline{u}, \overline{u}_1)$	$J(\widehat{u}, \overline{u}_1)$
0.45	0.50	0.74198	2.2439	3.0395
0.60	0.35	0.53076	2.2290	2.8290
0.75	0.20	0.31481	2.0092	2.6092

$\overline{u}_1 = 0.7$ and $\overline{u} = 0.7$ of $u_1(t)$, and $u(t)$, respectively, and in the last column we use the values $\overline{u}_1 = 0.7$ and $\widehat{u} = 0.3$.

The computations in the table show that the application of just a constant vaccination strategy would render excessively large values of the objective functional in comparison with the optimal vaccination strategy. The resulting benefit therefore makes it worth the effort of calculating the optimal control.

5. Concluding Remarks

We observe the influence of the migrant subpopulation onto a given (local) population, and then determine an optimal vaccination strategy for the two-group population. In the model of Jia et al., the emphasis is on the high impact of migrants. The paper [4] of Jia et al., and also the work [2] of Tumwiine et al., together with some other papers, support the theory that migrants have considerable influence in transmission of most communicable diseases. Our model facilitates numerical illustration of these phenomena. Example 4.3, in particular, shows how optimal control theory informs the best strategy, eliminating the risk of naive decision making. With the results of this paper, we are now able to efficiently plan the rollout of the appropriate vaccination strategy on such a two-group population. Future research may include similar investigations on stochastic two-group SIR models such as the model in [12] of Yu et al. In particular, the approach of Lahrouz et al. [9] to the stochastic model, permits an SIR model in which the total population stays constant, while stochasticity prevails in the propagation of the disease. This method shows much promise in epidemiological modeling.

Acknowledgments

References

[1] K. Chalvet-Monfray, M. Artzrouni, J. P. Gouteux, P. Auger, and P. Sabatier, "A two-patch model of Gambian sleeping sickness: Application to vector control strategies in a village and plantations," *Acta Biotheoretica*, vol. 46, no. 3, pp. 207–222, 1998.

[2] J. Tumwiine, J. Y. T. Mugisha, and L. S. Luboobi, "A host-vector model for malaria with infective immigrants," *Journal of Mathematical Analysis and Applications*, vol. 361, no. 1, pp. 139–149, 2010.

[3] Y. Zhou, K. Khan, Z. Feng, and J. Wu, "Projection of tuberculosis incidence with increasing immigration trends," *Journal of Theoretical Biology*, vol. 254, no. 2, pp. 215–228, 2008.

[4] Z. W. Jia, G. Y. Tang, Z. Jin et al., "Modeling the impact of immigration on the epidemiology of

tuberculosis," *Theoretical Population Biology*, vol. 73, no. 3, pp. 437–448, 2008.

[5] R. Naresh, A. Tripathi, and D. Sharma, "Modelling and analysis of the spread of AIDS epidemic with immigration of HIV infectives," *Mathematical and Computer Modelling*, vol. 49, no. 5-6, pp. 880–892, 2009.

[6] M. De La Sen, R. P. Agarwal, A. Ibeas, and S. Alonso-Quesada, "On the existence of equilibrium points, boundedness, oscillating behavior and positivity of a SVEIRS epidemic model under constant and impulsive vaccination," *Informatica*, vol. 22, no. 3, pp. 339–370, 2011.

[7] Y. Li, L. Chen, and K. Wang, "Permanence for a delayed nonautonomous SIR epidemic model with density-dependent birth rate," *Discrete Dynamics in Nature and Society*, vol. 2011, Article ID 350892, 10 pages, 2011.

[8] A. Kaddar, A. Abta, and H. T. Alaoui, "A comparison of delayed SIR and SEIR epidemic models," *Nonlinear Analysis: Modelling and Control*, vol. 16, no. 2, pp. 181–190, 2011.

[9] A. Lahrouz, L. Omari, and D. Kiouach, "Global analysis of a deterministic and stochastic nonlinear SIRS epidemic model," *Nonlinear Analysis: Modelling and Control*, vol. 16, no. 1, pp. 59–76, 2011.

[10] G. Zaman, Y. Han Kang, and I. H. Jung, "Stability analysis and optimal vaccination of an SIR epidemic model," *BioSystems*, vol. 93, no. 3, pp. 240–249, 2008.

[11] C. Piccolo III and L. Billings, "The effect of vaccinations in an immigrant model," *Mathematical and Computer Modelling*, vol. 42, no. 3-4, pp. 291–299, 2005.

[12] J. Yu, D. Jiang, and N. Shi, "Global stability of two-group SIR model with random perturbation," *Journal of Mathematical Analysis and Applications*, vol. 360, no. 1, pp. 235–244, 2009.

[13] Z. Agur, L. Cojocaru, G. Mazor, R. M. Anderson, and Y. L. Danon, "Pulse mass measles vaccination across age cohorts," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 90, no. 24, pp. 11698–11702, 1993.

[14] L. Acedo, J.-A. Moraño, and J. Díez-Domingo, "Cost analysis of a vaccination strategy for respiratory syncytial virus (RSV) in a network model," *Mathematical and Computer Modelling*, vol. 52, no. 7-8, pp. 1016–1022, 2010.

[15] J. M. Tchuenche, S. A. Khamis, F. B. Agusto, and S. C. Mpeshe, "Optimal control and sensitivity analysis of an influenza model with treatment and vaccination," *Acta Biotheoretica*, vol. 59, no. 1, pp. 1–28, 2011.

[16] S. Lenhart and J. T. Workman, *Optimal Control Applied to Biological Models*, Mathematical and Computational Biology Series, Chapman & Hall/CRC, London, UK, 2007.