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ANALYSIS OF A NEW SIR-M EPIDEMIC MODEL WITH INFECTION DURING TRANSPORT

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Abstract. In this paper, we propose a new epidemic SIR model with infection during transport, SIR-M. Under biologically motivated assumptions, we prove the positivity and boundedness of the solutions, calculate the basic reproduction number and the disease-free equilibrium. We prove that the disease-free equilibrium is globally asymptomatically stable if $\mathscr{R}_0$ is less than one, and then propose a generalized SIR-M model with n interacting populations. Finally, we numerically compare the behavior of the disease in different scenarios such as the perfect exit screening scenario, the mobility of infectious individuals from a single population, the prohibition of mobility (confinement), etc. We find that the use of certain health and precautionary measures such as screening, border control methods and containment, could significantly reduce the spread of the disease in both populations. On the other hand, the lack of control of the mobility of individuals may lead to a chaotic dispersion of the disease in both populations.

Keywords: epidemic SIR-M model; infection during transport; positivity and boundedness; basic reproduction number; global stability; numerical comparison.

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1. INTRODUCTION

The transmission of respiratory infectious diseases such as tuberculosis (TB), influenza, COVID-19, SARS, colds, etc., occurs through direct human-to-human or indirect contact with a contaminated intermediate objects, human-to-environment. Prolonged proximity of Susceptible-Infected individuals leads to increased spread of the disease in the population. Transport usually puts individuals in close proximity for long periods of time; it is likely that the probability of transmission during transport increases, especially with the duration of transport, so that long-distance travel would be prone to the transmission. Usually in most models analyzing the spread of infectious diseases, the transport component is usually not taken into account [1, 2, 3, 4]. However, knowing that more than 4.5 billion passengers traveled by plane in 2019. And it is estimated that humans travelled 23 billion kilometers in 2000 and that this will grow to an annual 105 billion kilometers by 2050[5, 6]. So it would be crucial to take the component of mobility into account in our models.

In the literature we find some articles examining the effect of mobility in the spread of disease. Brauer et al. have considered, in their paper [7], the impact of the arrival of already infected individuals, they proposed a model of evolution of the disease in a single population with immigration of infectious. Guo et al. [8] have studied the problems associated with the influx of individuals infected with tuberculosis. A few authors have considered the problem in the context of discrete delay differential equations, which make it possible to fix a precise travel time between locations[9, 10, 11].

In a framework of ordinary differential equations, Cui et al. [12] have formulated a susceptible-infectious-susceptible SIS model with infection during transport and investigated the local and global dynamics of these models. However, they assumed that all the parameters corresponding to the two patches are exactly the same. Arino et al. have revisited the model with different parameters in the two populations, [13].

After various authors have proposed different applications of compartmental models with mobility on infectious diseases [14] have proposed an application on dengue fever disease, [15] have measured the effect of confinement, lockdown, on the dynamics and spread of COVID-19. [16] have simulated a SEIR model with mobility representing the dispersion of COVID-19.[17]

Have analyzed the in-flight transmission and [18] have examined the contamination within cars In this article we propose a SIR-M model taking into account the transport component for the modeling of the spread of infectious diseases in the population. Our contribution is shared as follows: In the first section we formulate the SIR-M model, introduce the relevant parameters of the model and we prove the positivity and the boundlessness of the solutions. In the second section, we establish an analysis of the proposed model: we calculate the basic reproduction number as well as the disease-free equilibrium point, we investigate the global stability of the latter and we propose towards the end of this section a generalization of the SIR-M model with n interacting populations. In the last section, we propose simulations of four different scenarios, in order to measure the effect of mobility on the spread and dynamics of diseases in populations.

2. Model Formulation, Positivity and Boundedness

2.1. Model formulation. In our model we subdivide the total populations i and j at time t, denoted by $N_i(t)$ and $N_j(t)$, into three disjoint classes each: $S_i(t)$, $I_i(t)$, $R_i(t)$ and $S_j(t)$, $I_j(t)$, $R_j(t)$. With $S_i(t)$ denoting the number of susceptible individuals at time t, $I_i(t)$ the number of infective individuals and $R_i(t)$ the number of recovered individuals, in the population i. So that:

$$N_i = S_i + I_i + R_i \text{ and } N_j = S_j + I_j + R_j$$

We consider that individuals in the two populations and during transport mixes homogeneously, and that for each unit of time $t_i S_i$ susceptible individuals, $t_i I_i$ infectious individuals and $t_i R_i$ recovered individuals move from population i to population j using a specific mode of transport (train, bus, plane, etc.). Let's set α_i and α_j rates of contact between susceptible and infectious individuals in population i and j leading to a new infection during transport.

Thus the total infection (in vehicles) per unit time during transport from population i to population j is : $\dfrac{\alpha_i t_i S_i I_i}{N_i}$

To model the transmission of the disease, from infectious individuals to susceptible individuals in population i or j, we use proportional incidence functions, $f(S_i, I_i)$ and $f(S_j, I_j)$. Defined as follow:

$$f_i(S_i, I_i) = \frac{\beta_i S_i I_i}{N_i} \text{ and } f_j(S_j, I_j) = \frac{\beta_j S_j I_j}{N_j}$$

With β_i the transmission rates between the infectious and susceptible individuals in population i and β_j the transmission rates between the infectious and susceptible individuals in population j

So we formulate the following two patch SIR epidemiological model to describe the transmission dynamics of the infectious diseases with transport.

$$
(1) \qquad \begin{cases}
\frac{dS_i}{dt} &= \Lambda_i - f_i(S_i,I_i) - \mu_i Si - t_i S_i + (1 - \frac{\alpha_j I_j}{N_j}) t_j S_j, \\
\frac{dI_i}{dt} &= f_i(S_i,I_i) - (\omega_i + \mu_i + t_i) I_i + (1 + \frac{\alpha_j S_j}{N_j}) t_j I_j, \\
\frac{dR_i}{dt} &= \omega_i I_i - (\mu_i + t_i) R_i + t_j R_j, \\
\frac{dS_j}{dt} &= \Lambda_j - f_j(S_j,I_j) - \mu_j Sj - t_j S_j + (1 - \frac{\alpha_i I_i}{N_i}) t_i S_i, \\
\frac{dI_j}{dt} &= f_j(S_j,I_j) - (\omega_j + \mu_j + t_j) I_j + (1 + \frac{\alpha_i S_i}{N_i}) t_i I_i, \\
\frac{dR_j}{dt} &= \omega_j I_j - (\mu_j + t_j) R_j + t_i R_i,
\end{cases}
$$

With

- α_i : The rate of contact between susceptible and infectious individuals in population i leading to a new infection,
- α_j : The rate of contact between susceptible and infectious individuals in population j leading to a new infection,
- t_i : The travel rate in population i,
- t_j : The travel rate in population j,

μ_i : The mortality rate in population i,

μ_j : The mortality rate in population j,

ω_i : The cure rate for population i,

ω_j : The cure rate for population j.

Λ_i : The recruitment of susceptible individuals through birth in population i,

Λ_j : The recruitment of susceptible individuals through birth in population j,

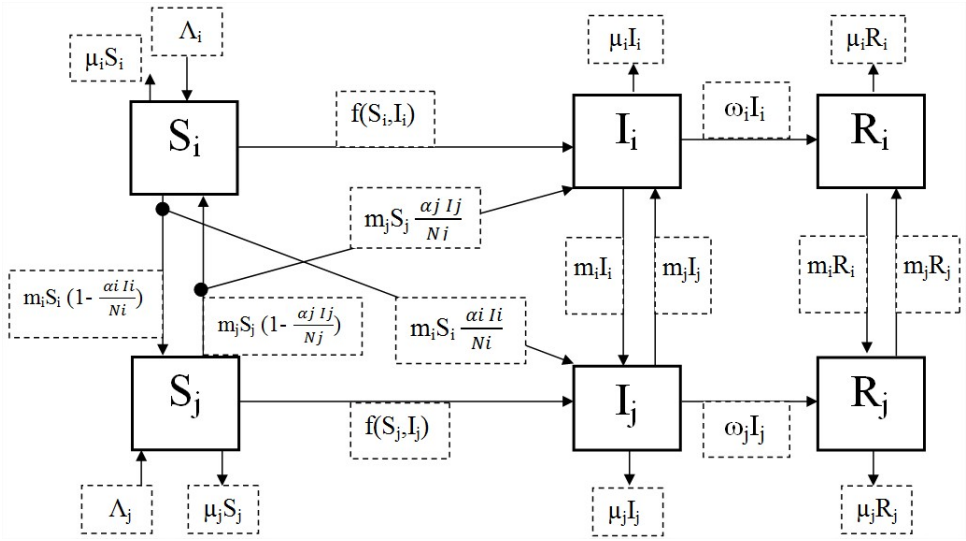

FIGURE 1. SIR model with mobility

Our SIR model with mobility is illustrated in the figure 1.

2.2. Positivity of solutions. Since the model monitors human populations, all its associated state variables must be positive and bounded. In addition, the following positivity result holds.

Theorem 1. *Let the initial data of the model be positive* $S_1(0) \geq 0$, $S_2(0) \geq 0$ $I_1(0) \geq 0$, $I_2(0) \geq 0$, $R_1(0) \geq 0$ *and* $R_2(0) \geq 0$, *then the solutions of the model* $S_1(t) \geq 0$, $S_2(t) \geq 0$, $I_1(t) \geq 0$, $I_2(t) \geq 0$, $R_1(t) \geq 0$ *et* $R_2(t) \geq 0$ *remain positive for all* $t > 0$.

Proof. Let us assume that $T = sup\{\tau \geq 0 \mid \forall 0 \leq t \leq \tau$ such that $S(t) \geq 0, I_1(t) \geq 0, I_2(t) \geq 0 R_1(t) \geq 0$ and $R_2(t) \geq 0\}$. Let us prove that $T = +\infty$.

Suppose that $0 < T < +\infty$ then by continuity of solutions we have : $S_1(T) = 0$ or $I_1(T) = 0$ or $I_2(T) = 0$ or $R_1(T) = 0$ or $R_2(T) = 0$.

If $S(T) = 0$ then :

$$S(T) = 0 \Rightarrow \frac{dS(T)}{dt} = \lim_{t \to T^-} \frac{S(T) - S(t)}{T - t} = \lim_{t \to T^-} \frac{-S(t)}{T - t} \leq 0$$

However if we set susceptibles to zero,, $S(t) = 0$, in the first equation of the system (2) we get $\frac{dS(T)}{dt} = \Lambda > 0$. Similar proof for $I_1(t), I_2(t), R_1(t)$ and $R_2(t)$. Thus T could not be finite, which concludes the proof. □

2.3. Boundedness of solutions. In this subsection, we prove that all solutions of system (1) are bounded.

Proposition 2. *The set $\mathscr{G}$ is positively invariant, with:*

$$\mathscr{G} = \{(S_i, I_i, R_i, S_j, I_j, R_j) \in \mathbb{R}^6_+ \text{ such that } S_i + I_i + R_i + S_j + I_j + R_j \leq N_i^* + N_j^*\} \tag{2}$$

This proves that all solutions are bounded.

Proof. Let $N(t) = N_i(t) + N_j(t)$ be the total population. From the model, the differential equations governing the evolution of N_i and N_j are :

$$\frac{dN_i}{dt} = \Lambda_i - (m_i + \mu_i)N_i + m_j N_j$$

$$\frac{dN_j}{dt} = \Lambda_j - (m_j + \mu_j)N_j + m_i N_i$$

By calculating the limit of each, we find:

$$\lim_{t \to \infty} N_i(T) = \frac{\Lambda_i \mu_j + m_j(\Lambda_i + \Lambda_j)}{\mu_i \mu_j + m_i \mu_j + m_j \mu_i} = N_i^*$$

and

$$\lim_{t \to \infty} N_j(T) = \frac{\Lambda_j \mu_i + m_i(\Lambda_j + \Lambda_i)}{\mu_j \mu_i + m_j \mu_i + m_i \mu_j} = N_j^*$$

This implies the convergence of the total population N (t)

$$\lim_{t \to \infty} N(T) = N_i^* + N_j^* = \frac{\Lambda_i(m_i + m_j + \mu_j) + \Lambda_j(m_i + m_j + \mu_i)}{\mu_j \mu_i + m_j \mu_i + m_i \mu_j}$$

Then $\mathscr{G}$ is positively invariant. Since the set $\mathscr{G}$ is positively invariant, it suffices to consider the dynamics of the flow generated by model (1) in $\mathscr{G}$, where the usual existence, uniqueness and continuation results are verified for the system [19]. □

3. Model Analysis

3.1. Basic reproduction number. The basic reproduction number $\mathscr{R}_0$ is interpreted as the average number of new cases generated by an infectious subject in a susceptible population. To calculate it we use the next generation matrix FV^{-1} [20]:

$$\mathscr{R}_{\mathbf{0}} = \rho(\mathbf{FV^{-1}})$$

With $F = \mathscr{D}\mathscr{F}(x^*)$, $V = \mathscr{D}\mathscr{V}(x^*)$ and:

(1) $\mathscr{F}(x)$ represents the appearance of new infected, in the population. These are new infected, obtained by transmission of any kind. Horizontal, i.e., from individual to individual or vertical from mother to the child.

(2) $\mathscr{V}_i^+(x)$ represents what comes from other compartments by any other cause (displacement, healing, etc.).

(3) $\mathscr{V}_i^-(x)$ represents what leaves compartment i. For example by mortality, by change of epidemiological status, by movement etc.

(4) $\mathscr{V}_i(x) = \mathscr{V}_i^-(x) - \mathscr{V}_i^+(x)$

According to our model we get:

$$\mathscr{F} = \begin{pmatrix} f_i(S_i, I_i) + \dfrac{\alpha_j S_j m_j I_j}{N_j} \\ f_j(S_j, I_j) + \dfrac{\alpha_i S_i m_i I_i}{N_i} \end{pmatrix}, \mathscr{V} = \begin{pmatrix} (\omega_i + \mu_i + m_i) I_i - m_j I_j \\ (\omega_j + \mu_j + m_j) I_j - m_i I_i \end{pmatrix}$$

We have two infected compartments: I_i, I_j

$$\mathbf{F} = \begin{pmatrix} \beta_i & \alpha_j m_j \\ \alpha_i m_i & \beta_j \end{pmatrix}, \mathbf{V} = \begin{pmatrix} (\omega_i + \mu_i + m_i) & -m_j \\ -m_i & (\omega_j + \mu_j + m_j) \end{pmatrix}$$

Thus FV^{-1} is given by:

$$\mathbf{FV^{-1}} = \begin{pmatrix} \dfrac{M_i}{det(V)} & \dfrac{\beta_i m_j + \alpha_j m_j (\omega_i + \mu_i + m_i)}{det(V)} \\ \dfrac{\beta_j m_i + \alpha_i m_i (\omega_j + \mu_j + m_j)}{det(V)} & \dfrac{M_j}{det(V)} \end{pmatrix}$$

With,

$$M_i = \beta_i(\omega_j + \mu_j + m_j) + \alpha_j m_1 m_2$$

$$M_j = \beta_j(\omega_i + \mu_i + t_i) + \alpha_i t_i t_j$$

$$det(V) = (\omega_i + \mu_i + t_i)(\omega_j + \mu_j + t_j) - t_i t_j$$

Therefore, the basic reproduction number of the system is:

$$\mathscr{R}_0 = \frac{M_i + M_j + \sqrt{\Delta}}{2det(V)} \tag{3}$$

With,

$$\Delta = (M_i - M_j)^2 + 4[\beta_i t_j + \alpha_j t_j(\omega_i + \mu_i + t_i)][\beta_j t_i + \alpha_i t_i(\omega_j + \mu_j + t_j)]$$

3.2. Global stability of the disease free equilibrium. In this section we will study the global stability of the disease free equilibrium, which is an equilibrium point, without infections i.e. $I_i^* = 0$ and $I_j^* = 0$.

Let $\mathscr{E}_0$ an equilibrium without disease, $\mathscr{E}_0 = (S_i^*, I_i^*, R_i^*, S_j^*, I_j^*, R_j^*) \in \mathscr{G}$ is such that:

$$\begin{cases} 0 &= \Lambda_i - f_i(S_i^*, I_i^*) - \mu_i S_i^* - t_i S_i^* + (1 - \dfrac{\alpha_j I_j^*}{N_j^*}) t_j S_j^*, \\ 0 &= f_i(S_i^*, I_i^*) - (\omega_i + \mu_i + t_i) I_i^* + (1 + \dfrac{\alpha_j S_j^*}{N_j^*}) t_j I_j^*, \\ 0 &= \omega_i I_i^* - (\mu_i + t_i) R_i^* + t_j R_j^*, \\ 0 &= \Lambda_j - f_j(S_j^*, I_j^*) - \mu_j S_j^* - t_j S_j^* + (1 - \dfrac{\alpha_i I_i^*}{N_i^*}) t_i S_i^*, \\ 0 &= f_j(S_j^*, I_j^*) - (\omega_j + \mu_j + t_j) I_j^* + (1 + \dfrac{\alpha_i S_i^*}{N_i^*}) t_i I_i^*, \\ 0 &= \omega_j I_j^* - (\mu_j + t_j) R_j^* + t_i R_i^*, \end{cases} \tag{4}$$

The proposed model has a single disease-free equilibrium (DFE) given by:

$$\mathscr{E}_0 = (S_i^*, I_i^*, R_i^*, S_j^*, I_j^*, R_j^*) = (\frac{\Lambda_i \mu_j + t_j(\Lambda_i + \Lambda_j)}{\mu_i \mu_j + t_i \mu_j + t_j \mu_i}, 0, 0, \frac{\Lambda_j \mu_i + t_i(\Lambda_i + \Lambda_j)}{\mu_i \mu_j + t_j \mu_i + t_i \mu_j}, 0, 0,) \tag{5}$$

Theorem 3. *if $\mathscr{R}_0 \leq 1$. Then the disease-free equilibrium point $\mathscr{E}_0$ is globally asymptotically stable in $\mathscr{G}$.*

Proof. To prove the global stability it is sufficient to prove that each positive solution $(S_i, I_i, R_i, S_j, I_j, R_j)$ tends to the disease-free equilibrium point $\mathscr{E}_0$, when t tends $+\infty$. i.e.:

$$\lim_{t\to\infty}(S_i(t), I_i(t), R_i(t), S_j(t), I_j(t), R_j(t)) = (\frac{\Lambda_i\mu_j + t_j(\Lambda_i+\Lambda_j)}{\mu_i\mu_j + t_i\mu_j + t_j\mu_i}, 0, 0, \frac{\Lambda_j\mu_i + t_i(\Lambda_i+\Lambda_j)}{\mu_i\mu_j + t_j\mu_i + t_i\mu_j}, 0, 0,)$$

From the second and the fifth ODE in the system 1, we obtain:

$$\begin{cases} \dfrac{dI_i}{dt} & \leq \quad \beta_i I_i - (\omega_i + \mu_i + t_i)I_i + (1+\alpha_j)t_j I_j \\ \\ \dfrac{dI_j}{dt} & \leq \quad \beta_j I_j - (\omega_j + \mu_j + t_j)I_j + (1+\alpha_i)t_i I_i \end{cases}$$

For $\mathscr{R}_0 \leq 1$ the eigenvalues of the matrix of coefficient F-V on the right side are in the left half-plane. So using a standard ODE comparison theorem, each positive solution of the second and the fifth ODE in the system 1 satisfies.

$$\lim_{t\to\infty}(I_i(t), I_j(t)) = (0,0).$$

As consequence:

$$\lim_{t\to\infty}(R_i(t), R_j(t)) = (0,0).$$

The limit system of the first and the fourth ODE of system 1 obtained when $\lim_{t\to\infty}(I_i(t), I_j(t)) = (0,0)$ and $\lim_{t\to\infty}(R_i(t), R_j(t)) = (0,0)$, is given by:

$$\begin{cases} \dfrac{dS_i}{dt} & \leq \quad \Lambda_i - \mu_i S_i - t_i S_i + t_j S_j \\ \\ \dfrac{dS_j}{dt} & \leq \quad \Lambda_j - \mu_j S_j - t_j S_j + t_i S_i \end{cases} \tag{6}$$

From 6 it is easy to verify that [21]:

$$\lim_{t\to\infty} S_i(t) = \frac{\Lambda_i\mu_j + t_j(\Lambda_i+\Lambda_j)}{\mu_i\mu_j + t_i\mu_j + t_j\mu_i}$$

$$\lim_{t\to\infty} S_j(t) = \frac{\Lambda_j\mu_i + t_i(\Lambda_i+\Lambda_j)}{\mu_i\mu_j + t_j\mu_i + t_i\mu_j}$$

Then the DFE is globally asymptotically stable in $\mathscr{G}$ if $\mathscr{R}_0 \leq 1$. □

3.3. Generalization of the model. The model we propose is a model representing the dynamics and evolution of the infection in any two interacting and mobile populations P_i and P_j.

In a general way we present the interactions between n different populations in the table 1.

	P_1	P_2	P_3	.....	P_i	P_n
P_1	$F(x_1)$	$F(x_1)+M(x_2)$	$F(x_1)+M(x_3)$	....	$F(x_1)+M(x_i)$	$F(x_1)+M(x_n)$
P_2	$F(x_2)+M(x_1)$	$F(x_2)$	$F(x_2)+M(x_3)$	....	$F(x_2)+M(x_i)$	$F(x_2)+M(x_n)$
P_3	$F(x_3)+M(x_1)$	$F(x_3)+M(x_2)$	$F(x_3)$	....	$F(x_3)+M(x_i)$	$F(x_3)+M(x_n)$
.....	.....	.....	.....	.....	.....	.....
P_i	$F(x_i)+M(x_1)$	$F(x_i)+M(x_2)$	$F(x_i)+M(x_3)$	....	$F(x_i)$	$F(x_i)+M(x_n)$
P_n	$F(x_n)+M(x_1)$	$F(x_n)+M(x_2)$	$F(x_n)+M(x_3)$	....	$F(x_n)+M(x_i)$	$F(x_n)$

TABLE 1. The general model of the interaction of n populations

Assuming the vector,

$$x_i = (S_i, I_i, R_i).$$

We write the system 1, in the following form.

For all $i \neq j$, $i \in \{1,...,n\}$ and $j \in \{1,...,n\}$:

$$\dot{x_i} = F(x_i) + M(x_j)$$

With the case $i = j$

$$\dot{x_i} = F(x_i).$$

4. NUMERICAL SIMULATION

In this section, we propose numerical simulations of different possible scenarios in order to show the impact of the mobility of individuals in the dispersion of the disease in the population. We will fix for this purpose the parameters, see 2, in a basic reproduction number close to 1.

Parameter	Value	Unit
Λ_i	9	$Individuals \times Days^{-1}$
Λ_j	8	$Individuals \times Days^{-1}$
$\beta_{\mathbf{i}}$	0.25	$Days^{-1}$
$\beta_{\mathbf{j}}$	0.15	$Days^{-1}$
$\alpha_{\mathbf{i}}$	0.4	$Days^{-1}$
$\alpha_{\mathbf{j}}$	0.3	$Days^{-1}$
$\mu_{\mathbf{i}}$	$1/365 \cdot 65$	$Days^{-1}$
$\mu_{\mathbf{i}}$	$1/365 \cdot 60$	$Days^{-1}$
$\omega_{\mathbf{i}}$	$1/7$	$Days^{-1}$
$\omega_{\mathbf{j}}$	$1/8$	$Days^{-1}$

TABLE 2. The parameter values used in the simulations

Case 1: Mobility with infection in both populations: In this case we assume that all individuals in both populations travel without constraints.

We note from the simulation shown in Figure 2, that the model converges to an equilibrium other than DFE, this point is the endemic equilibrium. This implies that infection during transport is more likely to cause an epidemic in both areas.

Case 2: Mobility without infection: Perfect exit screening scenario

During a pandemic, in some countries passengers are screened to check whether they show symptoms of an illness or meet certain health requirements such as vaccination against a particular illness. Screening can take place either on departure from a country (exit filtering) or on arrival in a country (entry filtering). For example, with the rapid international spread of

COVID-19. WHO has requested that all areas screen departing passengers for symptoms of COVID-19.

In the figure 3, we consider that the two populations follow a perfect protocol of exit border control implemented in a symmetric way, we fix to do this $\alpha_i = 0$, $\alpha_j = 0$, $m_i I_i = 0$ and $m_j I_j = 0$. As shown in the table 3.

	P_1	P_2	P_3	.....	P_i	P_n
P_1	$F(x_1)$	$F(x_1)$	$F(x_1)$	....	$F(x_1)$	$F(x_1)$
P_2	$F(x_2)$	$F(x_2)$	$F(x_2)$	....	$F(x_2)$	$F(x_2)$
P_3	$F(x_3)$	$F(x_3)$	$F(x_3)$	....	$F(x_3)$	$F(x_3)$
.....	.....	.....	.....	.....	.....	.....
P_i	$F(x_i)$	$F(x_i)$	$F(x_i)$	....	$F(x_i)$	$F(x_i)$
P_n	$F(x_n)$	$F(x_n)$	$F(x_n)$	....	$F(x_n)$	$F(x_n)$

TABLE 3. The general model of the interaction of n populations with mobility without infection

We notice from the figure 3 that the model converges asymptotically towards the disease-free equilibrium point, DFE, this implies a disappearance of the disease from the two populations over time. This result shows us the importance of border control methods and their impact on the spread of the disease.

Case 3: Mobility of infectious individuals from a single population

To show the impact of mobility on the spread of diseases, in a more lucid way, we consider that populations follow a perfect protocol of exit border control implemented asymmetrically. We assume that susceptible and covered from both populations and infectious from population 2 travel freely, while infectious from population 1 are prevented from traveling to population 2, i.e. $\alpha_i = 0$, $m_i I_i = 0$. The general case is detailed in the table 4

	P_1	P_2	P_3	.....	P_i	P_n
P_1	$F(x_1)$	$F(x_1)+M(x_2)$	$F(x_1)+M(x_3)$	....	$F(x_1)+M(x_i)$	$F(x_1)+M(x_n)$
P_2	$F(x_2)$	$F(x_2)$	$F(x_2)+M(x_3)$	....	$F(x_2)+M(x_i)$	$F(x_2)+M(x_n)$
P_3	$F(x_3)$	$F(x_3)$	$F(x_3)$	....	$F(x_3)+M(x_i)$	$F(x_3)+M(x_n)$
.....	.....	.....	.....	.....	.....	.....
P_i	$F(x_i)$	$F(x_i)$	$F(x_i)$	....	$F(x_i)$	$F(x_i)+M(x_n)$
P_n	$F(x_n)$	$F(x_n)$	$F(x_n)$	....	$F(x_n)$	$F(x_n)$

TABLE 4. The general model of the interaction of n populations with Traveler Infections from a single population

From the figure 4 we observe an oscillation in the infectious of population 1, with infection during transport, which means that the disease will persist in the population and will not disappear. While in population 2, without infection during transport, the disease disappeared in a very short time.

Case 4: Mobility ban (Containment) One of the most effective strategies to reduce and combat the chaotic spread of the disease is containment, which involves forcing, under pain of economic or criminal sanctions, a population to stay in their homes or in a specific place. In this case we propose a simulation which illustrates the application of confinement in two populations, in order to show its impact on the reduction of the propagation of a contagion. We assume that containment was applied after the 80th day of disease onset. According to the figure 5, we observe a considerable decrease in the numbers of infectious in the two populations from the 80th day. We thus conclude that containment is a very effective strategy to fight against the spread of contagion.

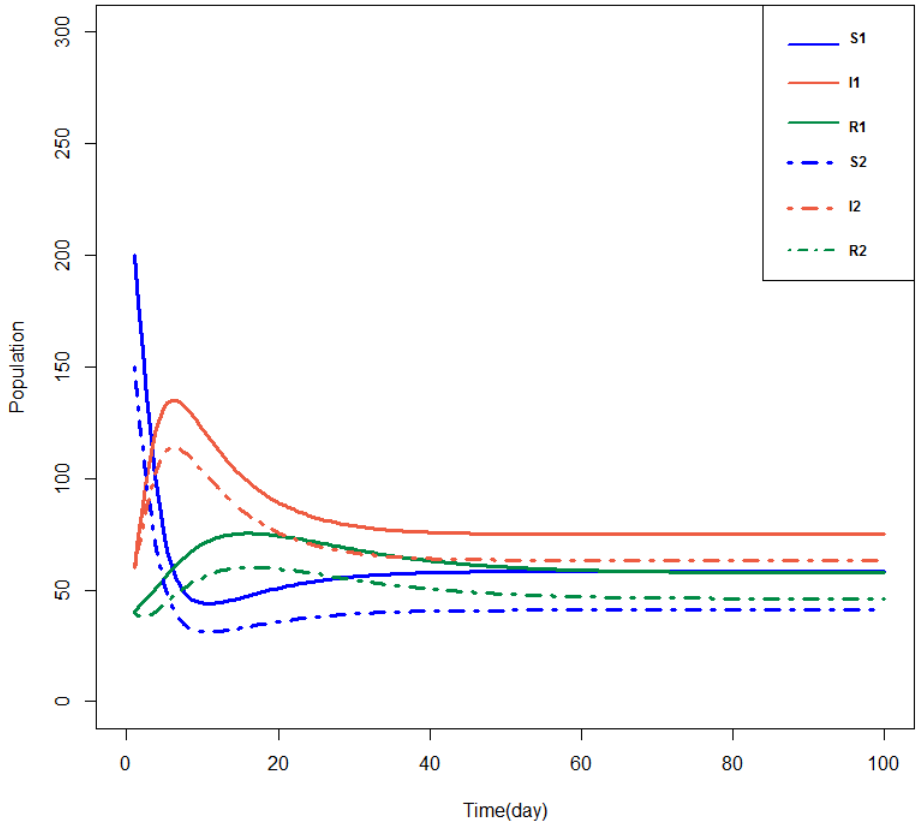

FIGURE 2. The behavior of the SIR-M model with infection

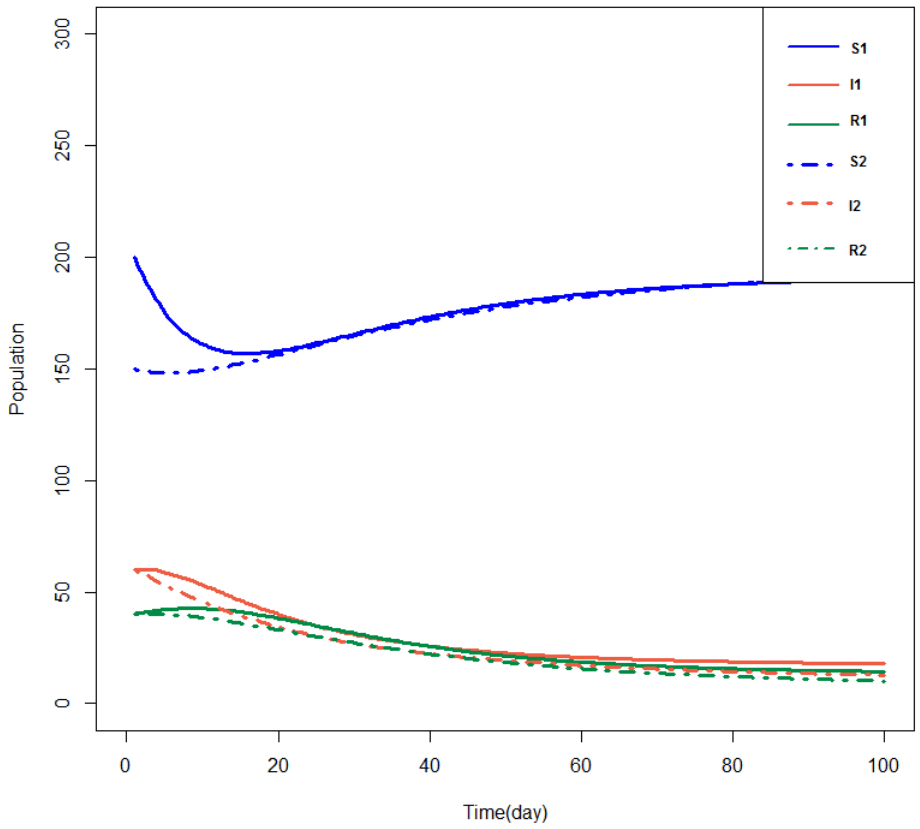

FIGURE 3. The behavior of the SIR-M model without infection

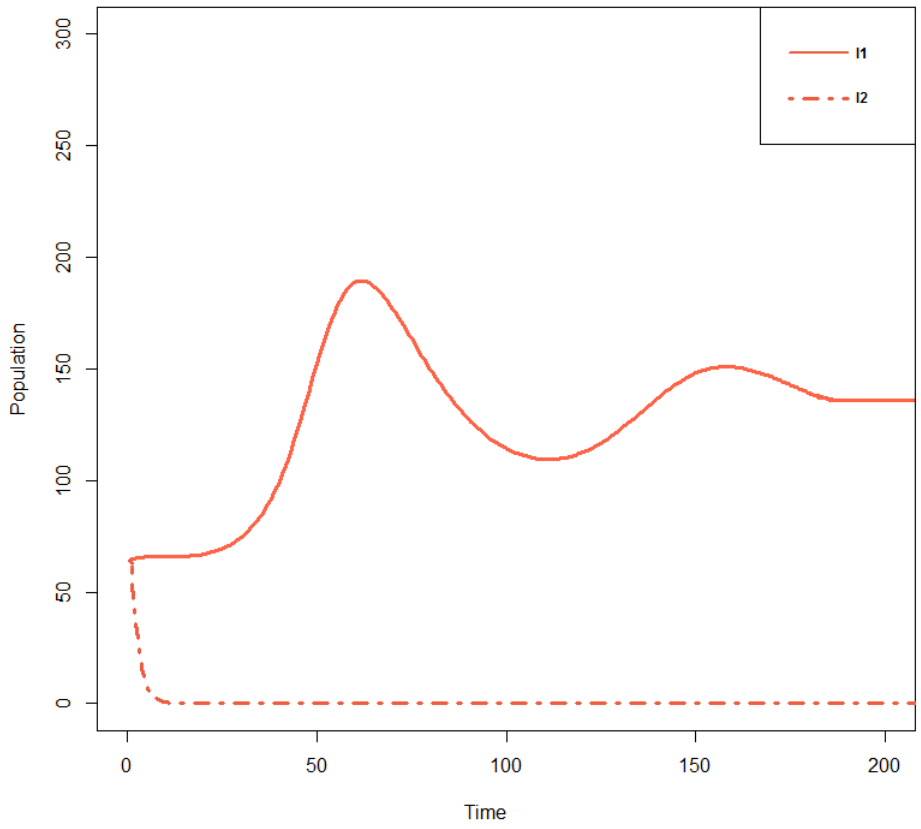

FIGURE 4. Behavior of infectious in populations 1 and 2 with a perfect border control protocol in an asymmetric way

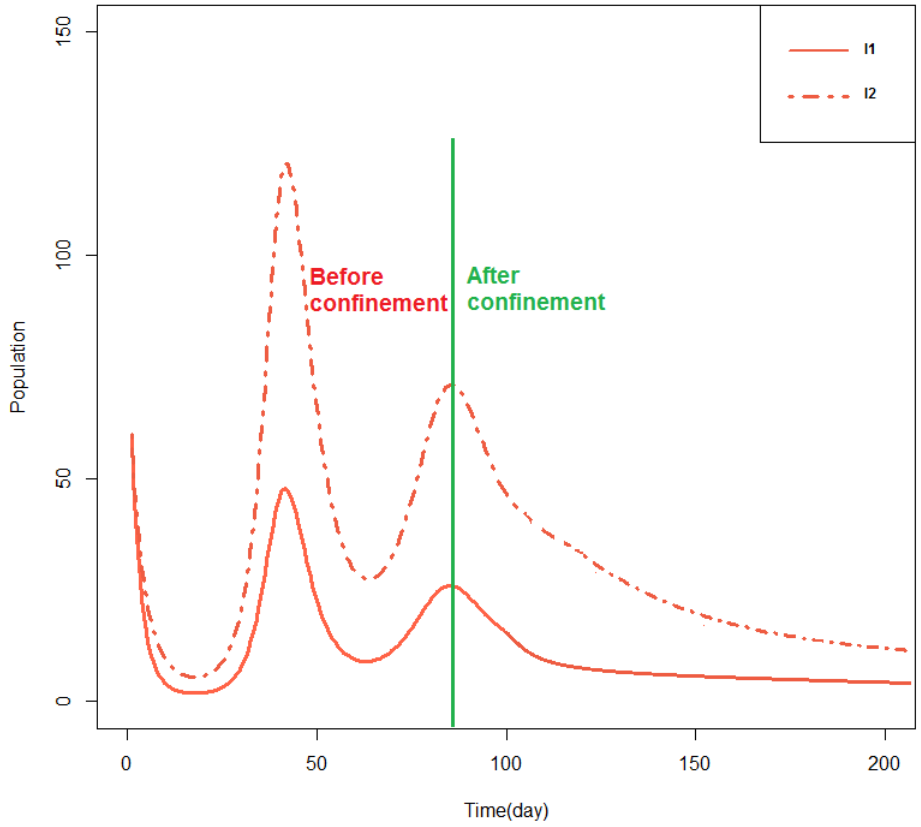

FIGURE 5. Behavior of infectious persons in populations 1 and 2 with application of total containment -Lack of mobility-

5. CONCLUSION

In this paper, we have proposed a model for the spread of diseases with infection during transport. We have considered a compartmental model SIR with mobility and different parameters in the different populations in interactions, SIR-M. We calculated the basic reproduction number as well as the disease-free equilibrium, DFE, we investigated the global stability of the DFE. and then we simulated different scenarios to show the impact of mobility on the spread of the disease. We assumed in a first case an interaction without limit, and we noticed the appearance of a new point of equilibrium of the model called the endemic point of equilibrium, in this case the disease persists in the two populations in interaction. In a second case, we simulated the

perfect screening scenario implemented in a symmetrical manner in the populations in interactions. We observed in this case an asymptotic stability of the equilibrium point without disease, which implies a disappearance of the disease, over time. We thus propose a case of the perfect screening scenario implemented in an asymmetric way. We have noticed in this case, the disappearance of the disease of the population applying entry filtering, screening, while the disease becomes more virulent in the population without border control. Finally, we simulated a case of dispersion before and after confinement in order to show the importance of confinement in the spread of the disease. This study allowed us to measure the impact of mobility, of individuals, on the spread of the disease. From the different simulated scenarios we were able to highlight the effectiveness of different strategies in the fight against the spread of the epidemic, such as screening, border control methods and containment.

CONFLICT OF INTERESTS

The author declares that there is no conflict of interests.

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