# AN SADR EPIDEMIC MODEL WITH A TRANSPORT-RELATED INFECTION BETWEEN TWO REGIONS

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Abstract. If an infectious disease is eradicated in an area, the travelling of infected people can cause a re-epidemic of the disease in such area. Travelling is one of the influential factors in the spread of an infectious diseases. In this paper, we present an epidemic model to investigate the effect of travelling between two regions. We compute the basic reproduction number of the model and study the local and global stability of the equilibrium points. In addition, we perform numerical simulations to illustrate the impact of increasing travel parameters on disease dynamics. The results show that travel between areas will change the dynamics of the disease and spread the infection.

Keywords: Epidemic Model, Transport, Local Stability, Global Stability.

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#### 1. Introduction

Population dispersion is one of the factors that causes the transmission of many diseases such as influenza, SARS, and COVID-19 from one region to another region. In addition, other factors such as lifestyle, sexual behaviors, and increased international travel lead to outbreaks of infectious diseases in humans. The question is: what effect does travel between different regions have on the dynamics of the disease in each of the regions? Until the work of Cui et. al. [4], most studies have not examined transport contamination in infectious disease dynamics. For example, Rvanchev and Longini (1985) examined the global spread of influenza in air networks, and Ful Ford (2002) formulated a metapopulation model with age-structured, or Arino and Vanden (2003) studied a specific disease in humans that spread through person-to-person contact within a city or country, then moved from one city to another city. All these investigations ignore the possibility of individuals becoming infected during travel. However, in some regions, like some developing countries, the situation is different. Some developing countries have large populations on trains and public transport. Poor hygiene in vehicles and places of transport is an element in the transmission and spread of infectious diseases in these countries. Cui et. al., [4]

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Notation	Description of Notation
$S_i, i = 1, 2$	Susceptible individuals in region $i$
$A_i, i = 1, 2$	Asymptomatic infectious individuals and infectious individuals not detected
	by healthcare system in region $i$
$D_i, i = 1, 2$	Infectious individuals detected by the healthcare system, i.e., confirmed cases
	in region $i$
$R_i, i = 1, 2$	Recovered individuals in region i
β	Infection rate
$\alpha$	The rate at which susceptible individuals of region j leave for region i, $i, j = 1, 2, i \neq j$
$\gamma$	Infection rate of the travelling individuals
$\mu$	Natural death rate
$\nu$	Identification rate of infectious individuals
$\eta$	Rate of loss of immunity among recovered individuals
$\eta_1$	Rate of recovery of confirmed cases
$\delta_1$	Rate of exacerbation of symptoms of the disease
ξ	Rate of recovery of asymptomatic infectious individuals
m	Disease-induced mortality rate
Λ	Recruitment rate

Table 1. Description of Notations

proposed a SIS epidemic model to understand the effect of transport-related infection on disease spread. After that, many studies were done in this field. Liu and Takeuchi (2006) [14] studied the spread of the disease due to transport contamination by input tests on the SIQS model. Wan and Cui (2007) [30] studied transport-induced contamination in the SEIS model, Liu and Zhou (2009) [16] examined the transport-induced contamination in the SIRS model and Chen et. al., [3] studied the SIR model. It is reasonable to study the effect of transport-related infection in environments in which there is a relatively large displacement between two or more regions in such environment. In some multi-group models, the effect of displacement on the infection rate may be negligible and can be ignored due to the small amount of migration between different parts of the region. Detailed studies have been done on multi-group models without migration. See, for example, Li [10], Ottaviano et. al., [22, 23], Sottile et. al., [25] and Muroya et. al., [19].

An important feature of some infectious diseases, such as the Covid-19 pandemic, is the presence of asymptomatic individuals, which are people who can transmit the infection despite not showing symptoms. Asymptomatic carriers are usually detected late, which makes them easily transmit the disease to other people. As an example, in references [8, 18, 22, 23, 26], compartments for asymptomatic carriers are considered in their models. In this work, we consider a two-region SADR(Susceptible-Asymptomatic infectious-Detected infectious-Recovered-Susceptible) model based on the model presented in [18] where the authors considered direct and indirect transmission of COVID-19. Clearly, we only consider direct transmission and also the environment with two areas between which significant travel is done. We aim to study the impact of transport-related contamination on disease dynamics in this model. Two cases are considered in the model. First, we study the case where no travel is done between two regions, and then the situation in which people in all classes except the identified infected people can travel. We compute the basic reproduction number of the model and study the local and global stability of the equilibria. In addition, we perform numerical simulations to investigate better the impact of increasing travel parameters on disease dynamics.

### 2. Model formulation



Figure 1. The flowchart of the model

We consider two areas with a large population and a significant number of trips between them. As an example, in Iran, we can consider the city of Tehran and the northern provinces, both of which have a large population and also a lot of travel between them. In epidemic models, the population is divided into different classes and how the population moves between these classes forms the dynamics of the system.

We use the model introduced in [18] by the author and its compartments, the class  $S_i$ for susceptible individuals; the class  $A_i$  that consists of two groups of individuals in the community, asymptomatic infectious individuals, i.e., infected individuals who have no symptoms of the disease and individuals with symptoms of the disease that are not detected by the healthcare system; the class  $D_i$  for infectious individuals who are detected by the healthcare system, i.e., confirmed cases; and the class of recovered individuals  $R_i$ . The total population in the regions is:

$$
N_i(t) = S_i(t) + A_i(t) + D_i(t) + R_i(t), i = 1, 2.
$$
\n(1)

We assume that the related parameters, which assume non-negative values, are the same for both cities. The population of susceptible individuals in city  $i$  increases with the arrival of new people at the recruitment rate  $\Lambda$ , the loss of immunity among recovered individuals at the rate  $\eta$ , and the susceptible individuals of the city j leave for city i at the rate  $\alpha$ . It is decreased when the susceptible individuals are infected via effective contact with infectious individuals at rate  $\beta S_i A_i$ , by natural death at the rate  $\mu$ , and by susceptible individuals in city i leaving city j at the rate  $\alpha$ . In addition, it is decreased, when the fractions  $\alpha S_i$  and  $\alpha A_j$  of the people who are traveling from region j to i, will be infected with the disease, at infection rate  $\gamma$ , during the trip. This generates a flow of the form  $\gamma(\alpha S_i)(\alpha A_i) = \gamma \alpha^2 S_i A_i$ .

As it is mentioned in [14] from the biological point of view, the number of susceptible

during travel should be nonnegative, that is,  $\alpha S_j - \gamma \alpha^2 S_j A_j \geq 0$ . The population of asymptomatic infected individuals in the city  $i$  is increased by infection of susceptible individuals at the rate  $\beta S_i A_i$  and infection of traveling individuals in the city j to the city i at the rate  $\gamma \alpha^2 S_i A_j$ . It is reduced by travel to the city j at the rate  $\alpha$ , natural death at the rate  $\mu$ , and disease-induced mortality at the rate m. Also, it is reduced when asymptomatic infected individuals are identified in two ways, testing and the onset of symptoms at rates  $\nu, \delta_1$ .

The population of detected infectious individuals is increased by identifying infected individuals in two ways, testing and the onset of symptoms, at rates  $\delta_1, \nu$  respectively. It is decreased by natural death and death due to disease and identified individuals recovered at the rate  $\eta_1$ . In this model, it is assumed that the identified infected individuals are not allowed to travel, and as in [18], we assumed that the detected patients are either being treated in medical centers or are being treated and quarantined at home. Therefore, we have ignored the very small number of people who may be infected by contact with such patients.

The population of recovered individuals is increased when identified individuals recover and move to the recovered class, at the rate  $\eta_1$ , and when recovered individuals of city j leave for city i. Also, asymptomatic individuals recovered at the rate  $\xi$ . It is decreased by the loss of immunity at the rate  $\eta$ , by the natural death at the rate  $\mu$ , and when recovered individuals of the city i move to the city j at the rate  $\alpha$ .

Based on the flow diagram of the model depicted in Figure 1 and the explanation given above, the dynamic of the system is governed by the following equations:

$$
\begin{cases}\n\frac{dS_1}{dt} = \Lambda - \beta S_1 A_1 - \alpha S_1 + \alpha S_2 + \eta R_1 - \mu S_1 - \gamma \alpha^2 S_2 A_2, \\
\frac{dS_2}{dt} = \Lambda - \beta S_2 A_2 - \alpha S_2 + \alpha S_1 + \eta R_2 - \mu S_2 - \gamma \alpha^2 S_1 A_1, \\
\frac{dA_1}{dt} = \beta S_1 A_1 - (\mu + m) A_1 + \alpha A_2 - \alpha A_1 - (\delta_1 + \nu) A_1 + \gamma \alpha^2 S_2 A_2, \\
\frac{dA_2}{dt} = \beta S_2 A_2 - (\mu + m) A_2 + \alpha A_1 - \alpha A_2 - (\delta_1 + \nu) A_2 + \gamma \alpha^2 S_1 A_1, \\
\frac{dD_1}{dt} = (\delta_1 + \nu) A_1 - (\mu + m + \eta_1) D_1, \\
\frac{dD_2}{dt} = (\delta_1 + \nu) A_2 - (\mu + m + \eta_1) D_2, \\
\frac{dR_1}{dt} = \eta_1 D_1 - (\mu + \eta + \alpha) R_1 + \alpha R_2 + \xi A_1, \\
\frac{dR_2}{dt} = \eta_1 D_2 - (\mu + \eta + \alpha) R_2 + \alpha R_1 + \xi A_2.\n\end{cases} (2)
$$

In this model, infection is transmitted with the incidence rate  $\beta S_i A_i$ ,  $i = 1, 2$ , and when the individuals in the city  $i$  travel to the city  $j$ , infection is transmitted with the rate  $\gamma \alpha^2 S_i A_i$ ,  $i = 1, 2$ . Assuming that the travel time between two regions is short, we ignore birth, death, and recovery during the travel time.

In this article, we have considered two areas for travel. This model can be generalized by considering N regions. The analysis of N-region model is an open research problem.

#### 3. Model Analysis

We study the dynamics of this model in two cases. At first, we suppose no one is allowed to travel and then we suppose all people except the identified infected individuals are allowed to travel between regions.

3.1. No travelling allowed. At first, we assume that no travel takes place between two regions, i.e.,  $\alpha = 0$ . In this case, changes occur independently, and we have a system with the following form in each region,

$$
\begin{cases}\n\frac{dS}{dt} = \Lambda - \beta SA - \mu S + \eta R, \\
\frac{dA}{dt} = \beta SA - (\mu + m)A - (\delta_1 + \nu + \xi)A, \\
\frac{dD}{dt} = (\delta_1 + \nu)A - (\mu + m + \eta_1)D, \\
\frac{dR}{dt} = \eta_1 D + \xi A - (\mu + \eta)R.\n\end{cases}
$$
\n(3)

We compute the equilibrium points and basic reproduction number of the system. The system (3) has a disease-free equilibrium point (DFE),  $E_0 = \left(\frac{\Lambda}{\Lambda}\right)$  $\left(\frac{\Lambda}{\mu},0,0,0\right)$ . A classical way to compute the basic reproduction number  $R_0$  of a model, which is a key quantity in mathematical epidemiology, is the linearization theorem which uses the Jacobian matrix of the system in disease-free equilibrium point, see [Ch.5, 17].

**Lemma 3.1.** In the system (3), with  $R_0 = \frac{\beta \Lambda}{\sqrt{(\mu + m)} \Lambda}$  $\frac{\rho}{\mu(\mu+m+\delta_1+\nu+\xi)}$ . We have: 1. If  $R_0 < 1$ , then the DFE point is locally asymptotically stable. 2. If  $R_0 > 1$ , then the DFE point is unstable.

Proof. The Jacobian matrix for (3) is given by,

$$
J(E_0) = \begin{bmatrix} -\mu & -\beta \frac{\Lambda}{\mu} & 0 & \eta \\ 0 & \beta \frac{\Lambda}{\mu} - Z & 0 & 0 \\ 0 & (\delta_1 + \nu) & -(\mu + m + \eta_1) & 0 \\ 0 & \xi & \eta_1 & -(\mu + \eta) \end{bmatrix}
$$

where,  $Z = (\mu + m + \delta_1 + \nu + \xi)$ . This matrix has eigenvalues  $\lambda_1 = -\mu$ ,  $\lambda_2 = -(\mu + \xi)$  $\eta$ ),  $\lambda_3 = -(\mu + m + \eta_1)$  and  $\lambda_4 = \beta \frac{\Lambda}{\mu}$  $\frac{11}{\mu}$  – Z. Now  $\lambda_4$  < 0 is equivalent to  $R_0 =$  $\beta\Lambda$  $\frac{\mu(\mu+m+\delta_1+\nu+\xi)}{\mu(\mu+m+\delta_1+\nu+\xi)}$  < 1, and we obtain the claimed result. Furtheremore, when  $R_0 > 1$ , (3) has the unique endemic equilibrium point  $E(S^*, A^*, D^*, R^*)$ 

where,

$$
\begin{cases}\nD^* = \frac{(\delta_1 + \nu)}{(\mu + m + \eta_1)} A^* = p_1 A^*, \\
R^* = \frac{\eta_1 p_1 A^* + \xi A^*}{(\mu + \eta)} = \frac{(\eta_1 p_1 + \xi) A^*}{(\mu + \eta)} = q_1 A^*, \\
S^* = \frac{(\mu + m + \delta_1 + \nu + \xi)}{\beta} = \frac{\Lambda}{\mu R_0}, \\
A^* = \frac{\Lambda (1 - \frac{1}{R_0})}{\mu + m + \delta_1 + \nu + \xi - \eta q_1},\n\end{cases} \tag{4}
$$

with  $\mu + m + \delta_1 + \nu + \xi - \eta q_1 > 0$ .

3.2. All individuals except detected infectious ones can travel. Now we suppose all people except the detected infectious individuals are allowed to travel. Considering the positive invariance that we discuss in the following lemma, we can limit our study to this set.

Lemma 3.2. The set

$$
\Omega = \left\{ (S_1, A_1, D_1, R_1, S_2, A_2, D_2, R_2) : S_i \ge 0, A_i \ge 0, D_i \ge 0, R_i \ge 0, \right\}
$$
  

$$
\sum_{i=1}^{2} (S_i + A_i + D_i + R_i) \le \frac{2\Lambda}{\mu} \}
$$

is a positively invariant set for the system (2).

**Proof.** The total population  $N(t) = \sum_{i=1}^{2} (S_i(t) + A_i(t) + D_i(t) + R_i(t))$ , satisfies the relation,

$$
\frac{dN}{dt} = 2\Lambda - \mu N - \sum_{i=1}^{2} m(A_i + D_i) \le 2\Lambda - \mu N.
$$

Now by using comparison lemma, we have,

$$
N(t) \le N(0)e^{-\mu t} + \frac{2\Lambda}{\mu}(1 - e^{-\mu t}) \le \max\left(N(0), \frac{2\Lambda}{\mu}\right).
$$
\n<sup>(5)</sup>

All components of the solution of the system are continuously differentiable. Furthermore, if all compartments have nonnegative initial conditions and if any of the compartments are zero at time  $t = t_i \geq 0$ , then the derivatives are nonnegative. For example if  $A_1(t_1) = 0$ ,  $A_2(t_1) \geq 0, S_i(t_1) \geq 0, D_i(t_1) \geq 0$  and  $R_i(t_1) \geq 0$  for  $i = 1, 2$ , we get,

$$
\frac{dA_1(t_1)}{dt} \ge 0
$$

that implies  $A_1(t_1^+) \geq 0$ , and hence,  $A_1(t)$  is nonnegative for all time  $t \geq 0$ . The same reason is true for other variables, and as mentioned in [21] it can be concluded that all compartments are nonnegative at all times  $t > 0$ . Now this result with the relation (5) show the positive invariance of  $\Omega$ .  $\Box$ 

We study various properties of this system. At first, we compute the equilibrium points and basic reproduction number of the system. This system has a disease-free equilibrium point (DFE),  $E_0 = \left(\frac{\Lambda}{\mu}\right)$  $\frac{\Lambda}{\mu}, 0, 0, 0, \frac{\Lambda}{\mu}$  $\left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$ . To derive the basic reproduction number  $R_{0\gamma}$ , we use the next generation matrix approach, see [28].

**Lemma 3.3.** The basic reproduction number of  $(2)$  is as follows:

$$
R_{0\gamma} = \frac{(\beta + \gamma \alpha^2) \frac{\Lambda}{\mu}}{\mu + m + \delta_1 + \nu + \xi}.
$$

**Proof.** The corresponding matrices  $\mathcal F$  and  $\mathcal V$  in the next generation matrix method have the following forms,

$$
\mathcal{F} = \begin{bmatrix} \beta S_1 A_1 + \gamma \alpha^2 S_2 A_2 \\ 0 \\ \beta S_2 A_2 + \gamma \alpha^2 S_1 A_1 \\ 0 \end{bmatrix}, F = \begin{bmatrix} \beta \frac{\Lambda}{\mu} & 0 & \gamma \alpha^2 \frac{\Lambda}{\mu} & 0 \\ 0 & 0 & 0 & 0 \\ \gamma \alpha^2 \frac{\Lambda}{\mu} & 0 & \beta \frac{\Lambda}{\mu} & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},
$$

and

$$
\mathcal{V} = \begin{bmatrix}\n(\mu + m + \delta_1 + \nu + \xi)A_1 + \alpha A_1 - \alpha A_2 \\
-(\delta_1 + \nu)A_1 + (\mu + m + \eta_1)D_1 \\
(\mu + m + \delta_1 + \nu + \xi)A_2 + \alpha A_2 - \alpha A_1 \\
-(\delta_1 + \nu)A_2 + (\mu + m + \eta_1)D_2\n\end{bmatrix},
$$

$$
V = \begin{bmatrix} X & 0 & -\alpha & 0 \\ -(\delta_1 + \nu) & (\mu + m + \eta_1) & 0 & 0 \\ -\alpha & 0 & X & 0 \\ 0 & 0 & -(\delta_1 + \nu) & (\mu + m + \eta_1) \end{bmatrix},
$$

in which  $X = (\mu + m + \delta_1 + \nu + \xi + \alpha)$ . Hence  $\begin{bmatrix} M & 0 & N & 0 \end{bmatrix}$ 

$$
FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ N & 0 & M & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \text{ where } M = \frac{(\beta X + \gamma \alpha^3) \frac{\Lambda}{\mu}}{X^2 - \alpha^2}, N = \frac{(\beta \alpha + \gamma \alpha^2 X) \frac{\Lambda}{\mu}}{X^2 - \alpha^2}.
$$

For computation of the spectral radius of  $FV^{-1}$ , we compute its characteristic polynomial,

$$
det(FV^{-1} - \lambda I) = \lambda^2((M - \lambda)^2 - N^2) = 0,
$$

which has the following roots,  $\lambda_1 = M - N$  and  $\lambda_2 = M + N$ . Hence,

$$
R_{0\gamma} = \rho (F V^{-1}) = M + N = \frac{(\beta X + \gamma \alpha^3) \frac{\Lambda}{\mu}}{X^2 - \alpha^2} + \frac{(\gamma \alpha^2 X + \beta \alpha) \frac{\Lambda}{\mu}}{X^2 - \alpha^2} = \frac{(\beta + \gamma \alpha^2) \frac{\Lambda}{\mu}}{\mu + m + \delta_1 + \nu + \xi}.
$$

The following result illustrates the threshold property of  $R_{0\gamma}$ .

**Theorem 3.1.** In system  $(2)$ , we have:

- 1. If  $R_{0\gamma} < 1$ , then the DFE point is locally asymptotically stable.
- 2. If  $R_{0\gamma} > 1$ , then the DFE point is unstable.

**Proof.** The Jacobian matrix of the system at  $E_0$  has the following form:

$$
J(E_0) = \left[ \begin{array}{cc} A & B \\ B & A \end{array} \right],
$$

where

$$
A = \begin{bmatrix} -\mu - \alpha & -\beta \frac{\Lambda}{\mu} & 0 & \eta \\ 0 & \beta \frac{\Lambda}{\mu} - X & 0 & 0 \\ 0 & (\delta_1 + \nu) & -(\mu + m + \eta_1) & 0 \\ 0 & \xi & \eta_1 & -(\mu + \eta + \alpha) \end{bmatrix}
$$

$$
B = \begin{bmatrix} \alpha & -\gamma \alpha^2 \frac{\Lambda}{\mu} & 0 & 0 \\ 0 & \gamma \alpha^2 \frac{\Lambda}{\mu} + \alpha & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha \end{bmatrix}.
$$

Now by [4], the eigenvalues of  $J(E_0)$  are identical to those of  $(A+B)$  and  $(A-B)$ . And we have,

$$
A + B = \begin{bmatrix} -\mu & -(\beta + \gamma \alpha^2) \frac{\Lambda}{\mu} & 0 & \eta \\ 0 & \beta \frac{\Lambda}{\mu} - X + \gamma \alpha^2 \frac{\Lambda}{\mu} + \alpha & 0 & 0 \\ 0 & (\delta_1 + \nu) & -(\mu + m + \eta_1) & 0 \\ 0 & \xi & \eta_1 & -(\mu + \eta) \end{bmatrix},
$$

and

$$
A - B = \begin{bmatrix} -\mu - 2\alpha & -(\beta - \gamma \alpha^2) \frac{\Lambda}{\mu} & 0 & \eta \\ 0 & \beta \frac{\Lambda}{\mu} - X - \gamma \alpha^2 \frac{\Lambda}{\mu} - \alpha & 0 & 0 \\ 0 & (\delta_1 + \nu) & -(\mu + m + \eta_1) & 0 \\ 0 & \xi & \eta_1 & -(\mu + \eta + 2\alpha) \end{bmatrix}.
$$

,

Now  $\lambda_1 = -\mu$ ,  $\lambda_2 = -(\mu + \eta)$ ,  $\lambda_3 = -(\mu + m + \eta_1)$  and  $\lambda_4 = \beta \frac{\Lambda}{\mu} - X + \gamma \alpha^2 \frac{\Lambda}{\mu} + \alpha$  are  $\mu$ <sup>11</sup>  $\mu$ <sup>2</sup> $\mu$ eigenvalues of  $A + B$ . And  $\lambda'_1 = -(\mu + 2\alpha)$ ,  $\lambda'_2 = -(\mu + m + \eta_1)$ ,  $\lambda'_3 = -(\mu + \eta + 2\alpha)$ and  $\lambda'_4 = \beta \frac{\Lambda}{\mu}$  $\frac{\Lambda}{\mu} - X - \gamma \alpha^2 \frac{\Lambda}{\mu} - \alpha$  are eigenvalues of  $A - B$ . Since  $R_{0\gamma} < 1$  implies,  $\lambda_4 = \beta \frac{\Lambda}{\Lambda}$  $\frac{\Lambda}{\mu} - X + \alpha + \gamma \alpha^2 \frac{\Lambda}{\mu} < 0 \, \, {\rm and} \, \, \lambda_4' = \beta \frac{\Lambda}{\mu}$  $\frac{\Lambda}{\mu} - X - \gamma \alpha^2 \frac{\Lambda}{\mu} - \alpha < 0, \text{ hence } E_0 \text{ is lo-}$ cally asymptotically stable, and the proof is complete.  $\Box$ 

To determine whether the disease can invade the population, we study the global stability of the DFE equilibrium point.

**Theorem 3.2.** If  $R_{0\gamma} < 1$ , then the DFE point of (2) is globally asymptotically stable.

**Proof.** We use the function,  $V(t) = A_1 + A_2$  as a Lyapunov function. Its derivative along the solutions of the system is,

 $\dot{V}(t) = \frac{dV(X(t))}{dt} = A_1' + A_2' = \beta S_1 A_1 - (\mu + m + \delta_1 + \nu + \xi + \alpha) A_1 + \alpha A_2 + \gamma \alpha^2 S_2 A_2 +$  $\beta S_2 A_2 - (\mu + m + \delta_1 + \nu + \xi + \alpha) A_2 + \alpha A_1 + \gamma \alpha^2 S_1 A_1 = (\beta + \gamma \alpha^2)(S_1 A_1 + S_2 A_2) - (\mu + \alpha^2) S_1 A_1$  $m + \delta_1 + \nu + \xi (A_1 + A_2).$ 

This relation shows that, if  $A_1 = A_2 = 0$  then  $\dot{V}(t) = 0$ . Furthermore, since  $R_{0\gamma} < 1$ ,

$$
\dot{V}(t) \leq [(\beta + \gamma \alpha^2) \frac{\Lambda}{\mu} - (\mu + m + \delta_1 + \nu + \xi)](A_1 + A_2) \leq 0,
$$

which implies,

$$
L = \{ (S_1, A_1, D_1, R_1, S_2, A_2, D_2, R_2) / \dot{V}(t) = 0 \}
$$
  
= \{ (S\_1, A\_1, D\_1, R\_1, S\_2, A\_2, D\_2, R\_2) / A\_1 = 0, A\_2 = 0 \}.

Now by restricting the system on the set  $L$ , we have,  $\frac{d(D_1+D_2)}{dt} = -(\mu+m+\eta_1)(D_1+D_2)$ which implies  $\lim_{t\to\infty} D_1(t) = \lim_{t\to\infty} D_2(t) = 0$ , and,

$$
M \subset L_1 = \{ (S_1, A_1, D_1, R_1, S_2, A_2, D_2, R_2) / A_1 = A_2 = 0, D_1 = D_2 = 0 \},
$$

in which  $M$  is the largest positively invariant subset of  $L$ . Restricting the system to the set  $L_1$  results in,  $\frac{d(R_1 + R_2)}{dt} = -(\mu + \eta)(R_1 + R_2)$ , which implies,  $\lim_{t \to \infty} R_1(t) =$  $\lim_{t\to\infty} R_2(t) = 0$  and,

 $M \subset L_2 = \{(S_1, A_1, D_1, R_1, S_2, A_2, D_2, R_2) / A_1 = A_2 = 0, D_1 = D_2 = 0, R_1 = R_2 = 0\}.$ Finally, by restricting the system to the set  $L_2$ , the result is,

$$
\frac{d(S_1 + S_2)}{dt} = 2\Lambda - \mu(S_1 + S_2), \frac{d(S_1 - S_2)}{dt} = (-2\alpha - \mu)(S_1 - S_2).
$$

Which shows,

$$
\lim_{t \to +\infty} (S_1(t) + S_2(t)) = \frac{2\Lambda}{\mu}, \lim_{t \to +\infty} (S_1(t) - S_2(t)) = 0,
$$

hence,

$$
\lim_{t \to +\infty} S_i(t) = \frac{\Lambda}{\mu}, (i = 1, 2).
$$

Therefore,  $M = \{E_0\}$  and by the LaSalle theorem,  $E_0$  is globally asymptotically stable provided  $R_{0\gamma} < 1$ . This completes the proof.  $\Box$ 

Now we study the endemic equilibrium point of the model and analyze its global stability.

When  $R_{0\gamma} > 1$ , our system has the unique positive equilibrium  $E^*(S^*, A^*, D^*, R^*, S^*, A^*, D^*, R^*)$ , where

$$
\begin{cases}\nD^* = \frac{(\delta_1 + \nu)}{(\mu + m + \eta_1)} A^* = p_1 A^*, \\
R^* = \frac{\eta_1 p_1 A^* + \xi A^*}{(\mu + \eta)} = \frac{(\eta_1 p_1 + \xi) A^*}{(\mu + \eta)} = q_1 A^*, \\
S^* = \frac{(\mu + m + \delta_1 + \nu + \xi)}{\beta + \gamma \alpha^2} = \frac{\Lambda}{\mu R_{0\gamma}}, \\
A^* = \frac{\Lambda (1 - \frac{1}{R_{0\gamma}})}{\mu + m + \delta_1 + \nu + \xi - \eta q_1}.\n\end{cases} \tag{6}
$$

.

Now we extract sufficient conditions for the global stability of the endemic equilibrium point.

**Theorem 3.3.** Suppose 
$$
\mu + 2\alpha > max \left\{ \frac{\eta}{2} + \frac{2(\beta - \gamma \alpha^2)\Lambda}{\mu}, \frac{\eta_1 + \xi - \eta}{2} \right\}
$$
 and  $\mu + m + \frac{\eta_1}{2} > \delta_1 + \nu$  then the radius equilibrium point of (2) is absolutely asymptotically stable when

 $\frac{1}{2}$ , then the endemic equilibrium point of (2) is globally asymptotically stable when  $R_{0\gamma} > 1$ .

Proof. We consider the function,

$$
V(X) = V(S_1, A_1, D_1, R_1, S_2, A_2, D_2, R_2) =
$$
  
= 
$$
\frac{(S_1 - S_2)^2 + (A_1 - A_2)^2 + (D_1 - D_2)^2 + (R_1 - R_2)^2}{2}
$$

The time derivative of V along the solutions of the system,  $\dot{V}(t) = \frac{dV(X(t))}{dt}$ , have the following form,

 $\dot{V}(t) = (S_1 - S_2)[\beta(S_2A_2 - S_1A_1) + \eta(R_1 - R_2) - \mu(S_1 - S_2) - 2\alpha(S_1 - S_2) - \gamma\alpha^2(S_2A_2 - S_1A_1)]$  $[S_1A_1]$ ] +  $(A_1 - A_2)[\beta(S_1A_1 - S_2A_2) - (\mu + m + \delta_1 + \nu + \xi)(A_1 - A_2) - 2\alpha(A_1 - A_2)$  –  $\gamma\alpha^2(S_1A_1-S_2A_2)] + (D_1-D_2)[(\delta_1+\nu)(A_1-A_2)-(\mu+m+\eta_1)(D_1-D_2)]+(R_1-D_2)]$  $R_2$ [ $\eta_1(D_1 - D_2) + \xi(A_1 - A_2) - (\mu + \eta + \alpha)(R_1 - R_2) - \alpha(R_1 - R_2)$ ].

Hence,

$$
\dot{V}(t) = ((\beta - \gamma \alpha^2)(S_2 A_2 - S_1 A_1))(S_1 - S_2) + \eta (R_1 - R_2)(S_1 - S_2) - (\mu + 2\alpha)(S_1 - S_2)^2 + (\beta - \gamma \alpha^2)(S_1 A_1 - S_2 A_2)(A_1 - A_2) - (\mu + m + \delta_1 + \nu + \xi + 2\alpha)(A_1 - A_2)^2 + (\delta_1 + \nu)(A_1 - A_2)(D_1 - D_2) - (\mu + m + \eta_1)(D_1 - D_2)^2 + \eta_1 (D_1 - D_2)(R_1 - R_2) + \xi (A_1 - A_2)(R_1 - R_2) - (\mu + \eta + 2\alpha)(R_1 - R_2)^2.
$$
  
Now by using the inequalities,  $AB \leq \frac{1}{2}(A^2 + B^2)$  and

$$
S_2A_2-S_1A_1=S_2(A_2-A_1)+A_1(S_2-S_1)\leq \frac{\Lambda}{\mu}(|A_1-A_2|+|S_1-S_2|),
$$

we have

$$
\dot{V}(t) \leq \left[\frac{\eta}{2} + \frac{2(\beta - \gamma\alpha^2)\Lambda}{\mu} - (\mu + 2\alpha)\right](S_1 - S_2)^2 + \left[\frac{2(\beta - \gamma\alpha^2)\Lambda}{\mu} - (\mu + m + \frac{1}{2}\delta_1 + \frac{1}{2}\nu + \frac{1}{2}\xi + 2\alpha)\right](A_1 - A_2)^2 + \left[\frac{\delta_1 + \nu}{2} - (\mu + m + \frac{1}{2}\eta_1)\right](D_1 - D_2)^2 + \left[\frac{\eta_1 + \xi}{2} - (\mu + \frac{1}{2}\eta + 2\alpha)\right](R_1 - R_2)^2.
$$

Therefore this relation in conjunction with the supposed inequalities implies that we can choose  $\lambda < 0$  such that  $V(t) \leq \lambda V(t)$ . And thus by Gronwall Lemma,  $V(X(t)) \leq$  $V(X(0))e^{\lambda t}$  for  $t > 0$ , which shows that for any solution  $X(t) = (S_1, A_1, D_1, R_1, S_2, A_2, D_2, R_2)$ of the system, we have

$$
\lim_{t \to \infty} V(X(t)) = 0. \tag{7}
$$

Now let  $p \in \mathbb{R}^8_{\geq 0}$  and  $q \in \omega(p)$ , in which  $\omega(p)$  is the  $\omega$ -limit set of the solution of the system which initiate at p, i.e.,  $X(t,p)$ . There exists  $\{t_m\}_{1}^{\infty}$ , with  $\lim_{m\to\infty}t_m = \infty$  and  $\lim_{m\to\infty} X(t_m, p) = q$ . Continuity of V implies,  $\lim_{m\to\infty} V(X(t_m, p)) = V(q)$ , this relation with (7) implies  $V(q) = 0$ . Hence,

$$
\omega(p) \subset M = \{q : V(q) = 0\}
$$

and

$$
\{q: V(q) = 0\} = \{(S_1, A_1, D_1, R_1, S_2, A_2, D_2, R_2) \in \mathbb{R}_{\geq 0}^8 | S_1 = S_2, A_1 = A_2, D_1 = D_2, R_1 = R_2\}.
$$

On the set M the relations  $S = S_1 = S_2, A = A_1 = A_2, D = D_1 = D_2, R = R_1 = R_2$ , leads to the following system,

$$
\begin{cases}\n\frac{dS}{dt} = \Lambda - (\beta + \gamma \alpha^2)SA - \mu S + \eta R, \\
\frac{dA}{dt} = (\beta + \gamma \alpha^2)SA - (\mu + m)A - (\delta_1 + \nu + \xi)A, \\
\frac{dD}{dt} = (\delta_1 + \nu)A - (\mu + m + \eta_1)D, \\
\frac{dR}{dt} = \eta_1 D + \xi A - (\mu + \eta)R.\n\end{cases} (8)
$$

This system is a special case of the SAIRS system introduced and analyzed in [22]. Hence the equilibrium point  $(S^*, A^*, D^*, R^*)$  of the above system is globally asymptotically stable when  $R_{0\gamma} > 1$  as it is proved in [22]. This shows that the endemic equilibrium is globally asymptotically stable.  $\Box$ 

### 4. Numerical Simulations

In this section we simulate the system using MATLAB, so that the solutions of the system and sensitivity of the solutions to traveling parameters can be seen numerically. We present two examples.



# TABLE 2. Parameters and initial values

# Example 1.

We use the parameters and initial values mentioned in Table 2. In this case,  $R_{0\gamma} < 1$ . In Figure 2, the plots A-D, show the sensitivity of  $A_i(t)$  with respect to  $\alpha, \gamma$ , and E,F show the trajectories.





## Example 2.

In this example, we use the set of parameters and initial values mentioned in Table 2, in which  $R_{0\gamma} > 1$ . The plots A-D, in Figure 3, show the sensitivity of  $A_i(t)$  with respect to  $\alpha$ ,  $\gamma$ , and E,F show the trajectories. The initial values show that there is no infectious individual in region 2 at  $t = 0$ . But the graph related to  $A_2(t)$  shows that the disease has spread and become endemic in region 2, and this is due to people traveling between the two regions.



FIGURE 3

### **CONCLUSION**

In this paper a new deterministic model on the effect of travel on the dynamic of infectious disease is proposed. Properties of the model equations such as feasible region, basic reproduction number of the model, equilibrium points and their local and global stability have been studied. We analized the case of 2 regions. The general case can have N regions, which we leave as an open problem. Sufficient conditions for the global stability of endemic equilibrium point derived in Th.3.4 shows that increasing the traveling parameters, i.e.,  $\alpha, \gamma$ , makes these conditions easy to satisfy. Also, the increasing of  $\alpha, \gamma$ 

causes the increase of  $R_{0\gamma}$ . Increasing  $R_{0\gamma}$  to values greater than 1 and establishing those sufficient conditions causes the global stability of the endemic point, and this is also a sign of the persistence of the disease.

This problem can also be seen in the numerical simulations. Figure  $3(c-j)$ , show the effect of the increase in  $\alpha, \gamma$  on the increase in peak height of asymptomatic patients  $A_i$  and confirmed cases  $D_i$ , as well as on the final value of these variables in both areas.

Figure 3(b) shows that, although there is no diseased person in area 2 at time 0, i.e.  $A_2(0) = D_2(0) = R_2(0) = 0$ , the disease spreads and becomes endemic in this area. And this shows the impact of travel between regions on the spread of the disease.

#### **REFERENCES**

- [1] Allen, L. J. S., An introduction to mathematical biology, Pearson education ltd., USA, (2007).
- [2] Arino, J., Van den Driessche, P., (2003), A multi-city epidemic model. Math. Popul. Stud., 10, 175-193. [3] Chen, Y., Yan, M., Xiang, Z., (2014), Transmission dynamics of a two-city SIR epidemic model with
- transport-related infections. J. App. Math., Article ID 764278.
- [4] Cui, J., Takeuchi, Y., Saito, Y., (2006), Spreading disease with transport-related infection. J. Theor. Biol., 239, 376-390.
- [5] Denphenedtnong, A., Chinviriyasit, W., (2013), On the dynamics of SEIRS epidemic model with transport-related infection. Math. Biosci., 245, 188-205.
- [6] Diekman, O., Heesterbeek, J. A. P., Metz, J. A. J., (1990), On the definition and the computation of the basic reproduction ratio R0 in the models for infection diseases in heterogeneous populations. J. Math. Biol., 28, 365-382.
- [7] Fulford, G. R., Roberts, M. G., Heesterbeek, J. A. P., (2002), The metapopulation dynamic of an infectious disease: tuberculosis in pissums. Theor. Popul. Biol., 61, 15-29.
- [8] Kemper, J.T., (1978), The effects of asymptomatic attacks on the spread of infectious disease: a deterministic model, Bull. Math. Biol., 40 (6), 707–718.
- [9] Kermak M., Mckendrick A., (1927), Contributions the mathematical theory of epidemics. Proc. Roy. Soc. A., 115, 700-21.
- [10] Li, M. Y., Zhisheng S., and Chuncheng W., (2010), Global stability of multigroup epidemic models with distributed delays. J. Math. Anal. Appl., 361.1, 38-47.
- [11] Liu, X., Chen, X., Takeuchi, Y., (2011), Dynamics of an SIQS epidemic model with transport-related infection and exit-entry screenings. J. Theor. Biol., 285, 25-35.
- [12] Liu, L., Liu, X., (2014), Global stability of a transport-related infection model with general incidence rate in two heterogeneous cities. Biosystem, 126, 41-51.
- [13] Liu, X., Stechlinski, P., (2013), Transmission dynamics of a switched multi-city model with transportrelated infections. Nonlinear Analysis: Real World Applications, 14, 264-279.
- [14] Liu, X., Takeuchi, Y., (2006), Spread of disease with transport-related infection and entry screening. J. Theor. Biol., 242, 517-528.
- [15] Liu, J., Wu, J., Zhou,Y., (2008), Modeling disease spread via transport-related infection by a delay differential equation, Rocky Mountain J. Math., 38(5), 1525-1540.
- [16] Liu, J., Zhou,Y., (2009), Global stability of an SIRS epidemic model with transport-related infection. Chaos, Solitons and Fractals, 40, 145-158.
- [17] Martcheva, M., An introduction to mathematical epidemiology, Springer, New York, 2015.
- [18] Memarbashi, R., Mahmoudi, S.M., (2021), A dynamic model for the COVID-19 with direct and indirect transmission pathways. Math. Meth. Appl. Sci., 44, 5873-5887.
- [19] Muroya, Y., Yoichi E., and Toshikazu K., (2013), Global stability for a multigroup SIRS epidemic model with varying population sizes. Nonlinear Analysis: Real World Applications, 14.3, 1693-1704.
- [20] Nakata,Y., (2011), On the global stability of a delayed epidemic model with transport-related infection. Nonlinear Analysis: Real World Applications, 12, 3028-3034.
- [21] K. Y. Ng and M. M. Gui, (2020), COVID-19: Development of a robust mathematical model and simulation package with consideration for ageing population and time delay for control action and resusceptibility. Physica D., 411, 132599.
- [22] Ottaviano, S., Sensi, M., and Sottile, S., (2022), Global stability of SAIRS epidemic models. Nonlinear Analysis: Real World Applications, 65, 103501.
- [23] Ottaviano, S., Sensi, M., and Sottile, S., (2023), Global stability of multi-group SAIRS epidemic models. Math. Meth. Appl. Sci., 65, 1-27.
- [24] Rvachev, L., Longini, I., (1995), A mathematical model for the global spread of influenza. Math. Biosci., 75, 3-22.
- [25] Sottile, S. and Liu, X., (2020), Time-varying epidemic transmission in heterogeneous networks and applications to measles, J. Bio. Sys., 28(4), 1-26.
- [26] Stilianakis, N.I., et. al., (1998), Emergence of drug resistance during an influenza epidemic: insights from a mathematical model, J. Infect. Dis., 177 (4), 863–873.
- [27] Takeuchi, Y., liu, X., Cui, J., (2007), Global dynamics of SIS models with transport-related infection. J. Math. Anal. Appl., 329, 1460-1471.
- [28] Van den Driessche, P., Watmough, J., (2002), Reproduction number and sub-threshold endemic equilibria for compartmental models of disease transmission. Math. Biosci., 180, 29-48.
- [29] Wallinga, J., Lipsitch, M., (2007), How generation intervals shape the relationship between growth rates and reproductive number. Proc. R. Soc. B., 274, 599-604.
- [30] Wan, H., Cui, J., (2007), An SEIS epidemic model with transport-related infection. J. Theor. Biol., 247, 507-524.



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