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Lyapunov Mappings and Analysis of a Nonlinear Spatio-temporal Epidemic Model

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Abstract

This paper focuses on the global asymptotic properties of the SIR diffusive model for infectious diseases. Using the analytic technique with Lyapunov functions, we developed conditions for the global attractor of a unique disease steady-state and the disease free equilibrium. The most eminent refuge to the model is the direct Lyapunov mapping. We investigate the global well-posedness of the mathematical model, determine conditions on R_o for which non-trivial equilibrium states exist, and examine their global stability. We are interested in finding the model's basic reproductive number, which determines whether the disease dies out or persists in the population. Finally, we consider a series of computational results to verify the theoretical results. The extensive numerical simulations show the dynamics of different population groups over time. The effects of different parameters on the compartments are shown in detail. The findings allude that the dynamics of the system are entirely estimated by the deterministic value R_o.

Keywords: Lyapunov function; uniform persistence; global analysis; SIR model; stability analysis.
AMS Subject Classification 2010: 92D30, 92D25, 93D05, 93D20, 93C20, 76E30.

I. Introduction

Infectious diseases have a remarkable impact on human population life, and thousands of people die of different communicable diseases. Controlling pandemic and endemic diseases has been an increasingly important issue to protect humans in the current decade. The susceptible class of individuals altering effectively during their life time. These variations are due to the grown up of the immune system. It has been expressed that a large number infectious diseases in nature incubate inside the innkeeper for a while before the hosts become infectious. In epidemiology, mathematical modeling is widely used to predict the results of an epidemic successfully [1, 2, 3].

In this decade, many scholars have considered spatio-temporal formation as a major factor because it influences both time and space dependent spreading of disease [4, 5, 6, 7]. In this study, we consider a time-space distributed problem, which is a dispersal version of the previously studied model [7, 8], where we assume the populated movements along all compartments. We strongly believe that our proposed model is a more general realistic biological and epidemiological model.

The model equations are

$$\begin{cases} \dfrac{\partial S(t,x)}{\partial t} = d_1\Delta S + a - mS - \dfrac{\beta IS}{1+\alpha I} + nR, & x\in\Omega, t\geq 0 \\ \dfrac{\partial I(t,x)}{\partial t} = d_2\Delta I + \dfrac{\beta IS}{1+\alpha I} - mI - \dfrac{\gamma IR}{1+R}, & x\in\Omega, t\geq 0 \\ \dfrac{\partial R(t,x)}{\partial t} = d_3\Delta R + \dfrac{\gamma IR}{1+R} - (m+n)R, & x\in\Omega, t\geq 0 \end{cases} \quad (1)$$

where $t\in[0,\infty]$ and $x\in\Omega$, with the homogeneous Neumann Boundary conditions

$$\frac{\partial S(t,x)}{\partial t} = \frac{\partial I(t,x)}{\partial t} = \frac{\partial R(t,x)}{\partial t} = 0, x\in\Omega, t\geq 0 \quad (2)$$

and the initial conditions

$$S(0,x)=\psi_1(x)\geq 0, I(0,x)=\psi_2(x)\geq 0, R(0,x)=\psi_3(x)\geq 0, x\in\Omega. \quad (3)$$

Where Ω is a domain with the smooth boundary $\partial\Omega$ and $\frac{\partial}{\partial\eta}$ denotes the outward normal derivative on $\partial\Omega$; $\psi_i(x)(i=1,2,3)$ are the non-negative Holder continuous bounded functions defined on Ω.

Assume the susceptible class $S(t,x)$, consists of the individuals who are capable of becoming infected, the infected class $I(t,x)$, consists of the individuals who are capable of transmitting the disease and the removed class $R(t,x)$, consists of individuals who have either died or recovered from the disease thereby becoming immune at time t and Ω. Here d_1, d_2, d_3 represent the diffusion coefficients for susceptible, infected and removal populations, respectively. Throughout this paper, we consider that the susceptible cells are recruited at a rate a. The parameter m is the natural death rate of the population, β is the transmission rate of infection, n is the rate of removal class individuals and return to S class, γ is the recovery rate of infected individuals, α is a non-negative constant and η is an outward normal vector on the boundary $\partial\Omega$.

A flow diagram of the model is shown in Figure 1

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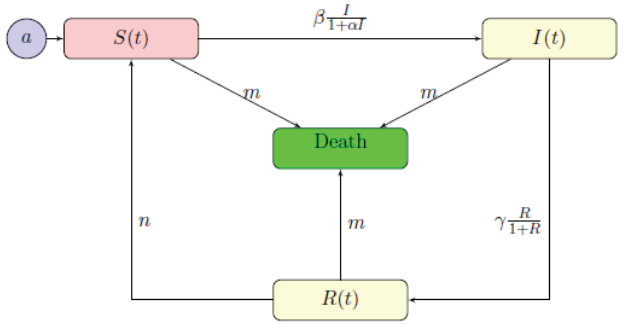

Fig. 1. Modeling scheme.

The paper is organized as follows. In Section 2, we prove the positivity and boundedness of the solution of the model. The disease-free (DFE) and endemic equilibrium (EE), respectively, are presented in this section. In addition, we present threshold values like the basic reproduction number in Section 2. For convenience, the existence-uniqueness of the solution of (1) are replaced over there. In section 3, we present local stability analysis along with responsible constraints. The global analysis steady states are replaced in Section 4. Section 5 presents computational results to verify the theoretical results using the Crank-Nicholson scheme. Finally, Section 6 discloses the concluding results.

II. Positivity and Boundedness of Solutions

An essential feature of an epidemiological relevant model is the positivity and boundedness of the solutions. Therefore, it is vital to prove that all the variables are non-negative for all time $t \geq 0$, implying that any solution with positive initial values will remain positive for all time $t \geq 0$.

Theorem 1. Assume $d_1 = d_2 = d_3$ in model (1). Then for any initial maps $(\psi_1(x), \psi_2(x), \psi_3(x)) \in X^+$ model (1) with the no-flux boundary conditions (2) and initial assumptions (3) has a unique, non-negative and consummately bounded solution $(S(t,x), I(t,x), R(t,x))$ defined on $[0,\infty)\times\Omega$.

Proof. It is obvious that the right side maps of problem (1) is locally Lipschitz for (S, I, R) in X^+ By the analogous reasons as in [7,8], together with [[9], Corollary 4], we can get the system (1) with states (2) and (3) obeys a unique local lenient solution $(S(t,x), I(t,x), R(t,x))$ prescribed on maximal presence interim $[0, S_{\max})$, where $0 < S_{\max} < \infty$. Moreover, resolution $(S(t,x), I(t,x), R(t,x))$ is a transcendent solution of system (1) . From [10], we furthermore can achive that $(S(t,x), I(t,x), R(t,x))$ is non-negative for $t \in [0, S_{\max})$. Defining $N(t,x) = S(t,x) + I(t,x) + R(t,x)$ when $d_1 = d_2 = d_3$ we have

$$\begin{cases} \dfrac{\partial N(t,x)}{\partial t} = d_1 \Delta N - mN + a \\ N_t = 0, x \in \partial\Omega, t \geq 0 \end{cases} \tag{4}$$

From Lemma 1 in [7], for any solution $N(t,x)$ of equation (4), we obtain $\lim_{x\to\infty} N(t,x) = \frac{a}{m}$ uniformly for $x \in \Omega$. Hence, $N(t,x)$ is also tied on $[0,\infty)\times\Omega$. It follows from the standard theory for semi-linear parabolic systems (see [11]) that $S_{\max} = \infty$. Which implies that the solution $(S(t,x), I(t,x), R(t,x))$ is prescribed for all $(t,x) \in [0,\infty)\times\Omega$ and also is unique and non-negative.

Since $\lim_{x\to\infty} N(t,x) = \frac{a}{m}$, then we have

$$N(t,x) \leq \frac{a}{m} + 1, \forall (t,x) \in [0,\infty)\times\Omega \tag{5}$$

Finally, from inequality (5), we gained that solution $(S(t,x), I(t,x), R(t,x))$ is consummately bounded, which finishes the proof.

Disease-free steady-state

To define the DFE (S_0, I_0, R_0) of the model, we present (1) as,

$$d_1 \Delta S_0 + a - mS_0 - \frac{\beta I_0 S_0}{1+\alpha I_0} + nR_0 = 0$$

$$d_2 \Delta I_0 + \frac{\beta I_0 S_0}{1+\alpha I_0} - mI_0 - \frac{\gamma I_0 R_0}{1+R_0} = 0$$

$$d_3 \Delta R_0 + \frac{\gamma I_0 R_0}{1+R_0} - (m+n) R_0 = 0$$

But for the DFE, we take the diffusion rates $d_i = 0$ for $i = 1,2,3$ and the count the infected cells $I_0 = 0$. Then we obtain,

Therefore, we have $\begin{cases} a - mS_0 + nR_0 = 0 \\ R_0 = 0 \end{cases}$

$$S_0 = \frac{a}{m}$$

And finally this gives the disease free equilibrium (DFE)

$$\boldsymbol{E}_0 = \left(\frac{a}{m}, 0, 0\right). \tag{6}$$

Disease equilibrium

When all compartments persist, then in this case of equilibrium point, we have the endemic equilibrium $\left(S^*, I^*, R^*\right)$, where the diffusion coefficients $d_i = 0, (i = 1,2,3)$. Here the infected individuals $I^* \neq 0$. Then we write

$$a - mS^* - \frac{\beta I^* S^*}{1+\alpha I^*} + nR^* = 0 \tag{7}$$

$$\frac{\beta I^* S^*}{1+\alpha I^*} - mI^* - \frac{\gamma I^* R^*}{1+R^*} = 0 \tag{8}$$

$$\frac{\gamma I^* R^*}{1+R^*} - (m+n)R^* = 0 \tag{9}$$

From equation (9), we have

$$\frac{\gamma I^* R^*}{1+R^*} - (m+n)R^* = 0$$

$$\therefore R^* = \frac{\gamma I^* - m - n}{m+n} \tag{10}$$

From equation (7), we have

$$a - mS^* - \frac{\beta I^* S^*}{1+\alpha I^*} + nR^* = 0$$

$$\Rightarrow S^*\left(m + \frac{\beta I^*}{1+\alpha I^*}\right) = a + nR^*$$

$$\therefore S^* = \frac{\left(1+\alpha I^*\right)\left(am + an + n\gamma I^* - mn - n^2\right)}{(m+n)\left(m + m\alpha I^* + \beta I^*\right)} \tag{11}$$

From equation (8), we obtain

$$\frac{\beta I^* S^*}{1+\alpha I^*} - mI^* - \frac{\gamma I^* R^*}{1+R^*} = 0$$

$$\Rightarrow \frac{\beta S^*}{1+\alpha I^*} - m - \frac{\gamma R^*}{1+R^*} = 0$$

$$\therefore S^* = \frac{\left(1+\alpha I^*\right)\left(mI^* + \gamma I^* - m - n\right)}{\beta I^*} \tag{12}$$

Equations (11) and (12) yields the following results

$$\frac{\left(1+\alpha I^*\right)\left(am + an + n\gamma I^* - mn - n^2\right)}{(m+n)\left(m + m\alpha I^* + \beta I^*\right)} = \frac{\left(1+\alpha I^*\right)\left(mI^* + \gamma I^* - m - n\right)}{\beta I^*}$$

$$\Rightarrow \left(I^*\right)^2\left[(m+n)\left(m\gamma\alpha + m^2\alpha + \beta\gamma + m\right) - \beta n\gamma\right] + I^*\left[(m+n)\left(m\gamma + m^2 - m^2\alpha - mn\alpha\right) - \beta\left(am + an + mn + m^2\right)\right] - m(m+n)^2 = 0 \tag{13}$$

To solve the equation (1.6) we get a real positive I^* and consequently, we find the disease equilibrium $E\left(S^*, I^*, R^*\right)$.

Threshold and Basic reproduction number

The threshold level defined by R_0 is arguably the most important quantity in infectious disease modeling. R_0 is defined as the average number of the new cases of an infection caused by one typical infected individual, in a population consisting of susceptible only. If $R_0 > 1$, the disease outbreak will occur. If $R_0 < 1$, the prevalence will toward the end asymptotically. In this paper, R_0 is deduced from the equation (1) using the next generation matrix method [6]. Let $Z = (I, R)^T$, then it follows from the system (1) that

$$\mathcal{F} = \begin{pmatrix} \frac{\beta IS}{1+\alpha I} \\ 0 \end{pmatrix}$$

$$\mathcal{V} = \begin{pmatrix} mI - \frac{\gamma IR}{1+R} \\ \frac{\gamma IR}{1+R} - (m+n)R \end{pmatrix}$$

the Jacobian matrix of matrices F and V are

$$F = \begin{pmatrix} \frac{\beta S}{(1+\alpha I)^2} & 0 \\ 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} m + \frac{\gamma R}{1+R} & \frac{\gamma I}{(1+R)^2} \\ -\frac{\gamma R}{1+R} & -\frac{\gamma I}{(1+R)^2} + m + n \end{pmatrix}$$

Substitute $S_0 = \frac{a}{m}, I_0 = 0, R_0 = 0$ we get

$$F = \begin{pmatrix} \frac{a\beta}{m} & 0 \\ 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} m & 0 \\ 0 & m+n \end{pmatrix}$$

The inverse of V is given by

$$V^{-1}=\begin{pmatrix}\frac{1}{m} & 0\\ 0 & \frac{1}{m+n}\end{pmatrix}$$

Thus, the next generation matrix for system 1 is

$$FV^{-1}=\begin{pmatrix}\frac{a\beta}{m^2} & 0\\ 0 & 0\end{pmatrix}$$

Since R_0 is the spectral radius of the matrix FV^{-1}, it follows that the basic reproduction number is

$$R_0=\frac{a\beta}{m^2}. \tag{14}$$

Existence and uniqueness of solution

We learning the algorithm of establishing the existence and uniqueness theorem from [12], now we will establish the existence and uniqueness of the solution of the governing system (1).

Consider the subset of $\mathbb{R}^3$ with vectors $x\geq 0$ as $\mathbb{R}^3_+$ and $\mathbb{X}:=C(\Omega,\mathbb{R})$ be a supremum norm $\|.\|_{\mathbb{X}}$. Let define $\mathbb{X}^+:=C\left(\Omega,\mathbb{R}^3_+\right)$, then $\left(\mathbb{X},\mathbb{X}^+\right)$ is a strongly ordered space. Assume that, $V_1(t),V_2(t),V_3(t):C(\Omega,\mathbb{R})\to C(\Omega,\mathbb{R})$ is the C_0 semi-groups connected with $d_1\Delta-m, d_2\Delta-m$ and $d_3\Delta-(m+n)$ subject to the no-flux boundary condition, respectively. Which follows that for any function $\varphi\in C(\Omega,\mathbb{R}), t\geq 0$,

$$\left(V_i(t)\varphi(x)\right)=e^{-mt}\int_\Omega \Gamma_i(x,y,t)\varphi(y)dx, i=1,2$$

$$\left(V_3(t)\varphi(x)\right)=e^{-(m+n)t}\int_\Omega \Gamma_i(x,y,t)\varphi(y)dx$$

where, Γ_i is defined as a Green function co-operated with $d_i\Delta, i=1,2,3,$ subject to the given no-flux boundary condition, respectively. It ensues that $V_i(t):C(\Omega,\mathbb{R})\to C(\Omega,\mathbb{R}),$ for all strictly positive t, is compact and strongly positive [13]. Especially, $V(t)=\left(V_1(t),V_2(t),V_3(t)\right):C(\Omega,R)\to C(\Omega,R),\forall t\geq 0$, is a sharply continuous semigroup.

If $H_i:G(H_i)\to\mathbb{X}$ is the originator of V_i, for all $i=1,2,3$ then $V(t)=\left(V_1(t),V_2(t),V_3(t)\right):\mathbb{X}\to\mathbb{X}$ is a semi-group generated by $H=(H_1,H_2,H_3)$ which is prescribed on $G(H):=G(H_{1})\times G(H_2)\times G(H_3)$. Now for any $\varphi=(\varphi_1,\varphi_2,\varphi_3)\in\mathbb{X}$, let us define $\mathcal{D}=(\mathcal{D}_1,\mathcal{D}_2,\mathcal{D}_3):\mathbb{X}^+\to\mathbb{X}$ by

$$\mathcal{D}_1(\varphi)(x)=a-m\varphi_1(x)-\frac{\beta\varphi_1(x)\varphi_2(x)}{1+\alpha\varphi_2(x)}+n\varphi_3(x),\forall x\in\Omega$$

$$\mathcal{D}_2(\varphi)(x)=\frac{\beta\varphi_1(x)\varphi_2(x)}{1+\alpha\varphi_2(x)}-m\varphi_2(x)-\frac{\gamma\varphi_2(x)\varphi_3(x)}{1+\varphi_3(x)},\forall x\in\Omega$$

$$\mathcal{D}_3(\varphi)(x)=\frac{\gamma\varphi_2(x)\varphi_3(x)}{1+\varphi_3(x)}-(m+n)\varphi_3(x),\forall x\in\Omega$$

Now, we can write (1) and $(2)-(3)$ as the following integral equation

$$\text{Where, } u(t)=V(t)\varphi+\int_0^t V(t-s)\mathcal{D}(u(s))ds$$

$$u(t)=\begin{pmatrix}S(t)\\ I(t)\\ R(t)\end{pmatrix},\quad V(t)=\begin{pmatrix}V_1(t) & 0 & 0\\ 0 & V_2(t) & 0\\ 0 & 0 & V_3(t)\end{pmatrix}$$

or it can be rewritten as the following abstract differential equation

$$\begin{cases}\dfrac{dw}{dt}=Hw+K(w), t>0\\ \qquad w_0=\varphi\in\mathbb{X}\end{cases} \tag{15}$$

where, $w=(S,I,R)$ and $\varphi=(S_0,I_0,R_0)$.

While $K(\varphi)$ is Lipschitz continuous on $\mathbb{X}^+$, it pursues that for any $\varphi\in\mathbb{X}^+$, (15) allows a unique continuous light solution $w(\cdot,t,\varphi)$ such that $w(\cdot,t,\varphi)\in\mathbb{X}$ for all t in its optimum boundary of existence. In addition, using Corollary 2.2.5 from [14] that $w(\cdot,t,\varphi)$ is a standard solution of (1) with respective boundary condition (2) for all $t>0$. Then using the parabolic maximum principle, it is observed from the equation (1) that $S(x,t), I(x,t)$ and $R(x,t)$ are all non-negative. Hence, we have the next fundamental results of the system (1)-(3).

Lemma 1. *For any IVF* $\varphi=(\varphi_1,\varphi_2,\varphi_3)\in\mathbb{X}^+$, *model* $(1)-(3)$ *has a unique result* $u(x,t,\varphi)$ *on* $[0,\sigma_\varphi)$ *with*

$u=\varphi$ *and* $u(\cdot,t,\varphi)\in\mathbb{X}^+$, *forallt* $\in[0,\sigma_\varphi)$.

In the following step, we prove that the solution of $(1)-(3)$ with IVF $\rho\in\mathbb{X}^+$ globally exists.

Consider the partial differential equation [7]

$$\begin{cases}\dfrac{\partial v}{\partial t}=D\nabla^2 v-dv(t,x)+A & (t,x)\in\omega \\ v_\eta=0 & t>0,x\in\partial\Omega\end{cases} \qquad (16)$$

where all parameters are positive.

Lemma 2. The problem (16) satisfied a unique positive equilibrium $u^*=\dfrac{A}{d}$ and the state is globally stable in $C(\Omega,R)$.

Theorem 2. *For all primary values* $\varphi\in\mathbb{X}^+$, *system* $(1)-(3)$ *has a unique value* $u(\cdot,t,\varphi)$ *on* $[0,\infty)$ *and the solution flow* $\varnothing=u(\cdot,t):\mathbb{X}^+\to\mathbb{X}^+, t\geq 0$, *has a global attractor.*

Proof. Following Lemma 1, the model $(1)-(3)$ has a solution $u(\cdot,t,\varphi)$ on $[0,\sigma_\varphi)$ is non-negative for any $t\in[0,\sigma_\varphi)$ over the domain. However, $\dfrac{a}{m}$ is a global solution by Lemma 2 for the differential equation as follows

$$\begin{cases}\dfrac{\partial v}{\partial t}=d_1\nabla^2 v+a-mv(t,x)+nv(t,x) & (t,x)\in\omega \\ v_\eta=0 & t>0,x\in\partial\Omega\end{cases}$$

Since

$$S_t\leq d_1\Delta S+a-mv+nv,\forall t\in[0,\sigma_\varphi),x\in\Omega \qquad (17)$$

It follows from the standard parabolic comparison theorem [13] that $S(\cdot,t.\varphi)$ is bounded on $[0,\sigma_\varphi)$; thus, there is a $S_0>0$ such that $S(\cdot,t,\varphi)\leq S_0,\forall t\in[0,\sigma_\varphi)$. Hence, it follows from (1) that

$$I_t\leq d_2\Delta I+\beta S_0-mI,\forall t\in[0,\sigma_\varphi),x\in\Omega. \qquad (18)$$

Again, by Lemma 2, $I(\cdot,t,\varphi)$ is bounded on $[0,\sigma_\varphi)$ such that $I(\cdot,t,\varphi)\leq I_0$, where $I_0>0$ for all $t\in[0,\sigma\varphi)$. Therefore, by the last equation of (1), we get

$$R_t\leq d_3\Delta R+\gamma I_0-(m+n)R,\forall t\in[0,\sigma_\varphi),x\in\Omega. \qquad (19)$$

As a result, $u(\cdot,t,\varphi)$ is bounded on $[0,\varphi)$. From (17), for any $\varphi\in\mathbb{X}^+$, it is seen that there exist $t_1=t_1(\varphi)>0$ such that

$$S\leq\frac{a}{m}+1:=h_1,\text{for all } t>t_1,x\in\Omega.$$

In a similar arguments, it is possible to show that there are $h_i>0$, and $t_i=t_i(\varphi)>0,i=1,2,3,$ such that

$$I\leq h_2,R\leq h_2,\text{for all } t>t_1,x\in\Omega.$$

So, the positive solution of $(1)-(3)$ is bounded in terms of the maximum norm. Which means, the solution flow $\Phi(t):\mathbb{X}^+\to\mathbb{X}^+$ presented by

$$(\Phi(t)\varphi)(x)=u(x,t,\rho),x\in,\Omega$$

is point evaporated. In comparison of [14,21], the function is compact for strictly positive time. Hence $\Phi(t):\mathbb{X}^+\to\mathbb{X}^+$, has a pervading compact in $\mathbb{X}^+$ and the proof is completed.

III. Local Stability Analysis

In the present potion, we want to explain the local stability of the steady state for the model 1 . And we state the following results.

Theorem 3.

i. When $R_0<1$, the DFE, E_0 of the system (1)-(3) is locally asymptotically stable;

ii. When $R_0>1$, the EE, E^* of the system (1)-(3) is locally asymptotically stable.

Proof: The Jacobean matrix of our system is

$$J=\begin{pmatrix}-m-\dfrac{\beta I}{1+\alpha I} & \dfrac{\beta S}{(1+\alpha I)^2} & n \\ \dfrac{\beta I}{1+\alpha I} & \dfrac{\beta S}{(1+\alpha I)^2}-m-\dfrac{\gamma IR}{1+R} & -\dfrac{\gamma IR}{1+R} \\ 0 & \dfrac{\gamma IR}{1+R} & \dfrac{\gamma IR}{1+R}-m-n\end{pmatrix}$$

The Jacobian matrix at $E_0=\left(\dfrac{a}{m},0,0\right)$ is

$$J=\begin{pmatrix}-m & \dfrac{a\beta}{m} & n \\ \dfrac{a\beta}{m} & -m+\dfrac{a\beta}{m} & 0 \\ 0 & 0 & -m-n\end{pmatrix}$$

The corresponding characteristic equation can be written as

$$|\lambda \mathrm{I} - \mathrm{J}| = 0$$

$$\Rightarrow \begin{vmatrix} \lambda + m & -\frac{a\beta}{m} & -n \\ -\frac{a\beta}{m} & \lambda + m - \frac{a\beta}{m} & 0 \\ 0 & 0 & \lambda + \mathrm{m} + \mathrm{n} \end{vmatrix} = 0$$

$$\Rightarrow (\lambda + m)(\lambda + \mathrm{m} + \mathrm{n})\left(\lambda + m - \frac{a\beta}{m}\right) = 0$$

To solve the equation, we get the eigenvalues

$$\lambda_1 = -m$$

$$\lambda_2 = -m - n$$

$$\text{and } \lambda_3 = -m + \frac{a\beta}{m}$$

Since we consider all the parameter are positive, so we have the first and last two eigenvalues are negative. Thus the DFE E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

IV. Global Stability Analysis

At this stage, we check the global solution of the equilibria E_0 and E^* in the bounded region such that (S, I, R) is a random and non-negative solution of (1).

For simplicity, consider the followings

$$S = S(x,t), I = I(x,t), R = R(x,t).$$

Theorem 4. *If $R_0 \le 1$, then the disease-free equilibrium (DFE) $E_0(S_0, I_0, R_0)$ of the problem (1) is globally asymptotically stable.*

Proof. Let us define a Lyapunov function as

$$\text{Where} \qquad V_1(t) = \int_\Omega W_1(x,t)\,dx$$

$$W_1(x,t) = S_0\left(\frac{S}{S_0} - 1 - \ln\frac{S}{S_0}\right) + I + R$$

Simplifying the time-derivative of $W_1(x,t)$ with the solution of (1) implies

$$\frac{\partial W_1}{\partial t} = \left(1 - \frac{S}{S_0}\right)\frac{\partial S}{\partial t} + \frac{\partial I}{\partial t} + \frac{\partial R}{\partial t}$$

Thus from (1), we can write

$$\frac{\partial W_1}{\partial t} = \left(1 - \frac{S}{S_0}\right)\left(d_1\Delta S + a - mS - \frac{\beta IS}{1+\alpha I} + nR\right) + d_2\Delta I + \frac{\beta IS}{1+\alpha I} - mI - \frac{\gamma IR}{1+R} + d_3\Delta R + \frac{\gamma IR}{1+R} - (m+n)R$$

But as $a = mS_0$, we may write

$$\frac{\partial W_1}{\partial t} = \left(1 - \frac{S}{S_0}\right)\left(d_1\Delta S + mS_0 - mS - \frac{\beta IS}{1+\alpha I} + nR\right) + d_2\Delta I + \frac{\beta IS}{1+\alpha I} - mI - \frac{\gamma IR}{1+R} + d_3\Delta R + \frac{\gamma IR}{1+R} - (m+n)R$$

$$= \left(1 - \frac{S}{S_0}\right)\left\{mS_0\left(1 - \frac{S}{S_0}\right) - mS - \frac{\beta IS}{1+\alpha I} + nR\right\} + d_2\Delta I + \frac{\beta IS}{1+\alpha I} - mI + d_3\Delta R - (m+n)R$$

$$= \left(1 - \frac{S}{S_0}\right)d_1\Delta S + d_2\Delta I + d_3\Delta R + mS_0\left(2 - \frac{S}{S_0} - \frac{S_0}{S}\right) - \frac{mI}{1+\alpha I}(1 + \alpha I - \Re_0) + nR\left(1 - \frac{S}{S_0}\right) - (m+n)R$$

Introducing the Green's formula and given boundary conditions, we get

$$\int_\Omega \Delta S\,dx = \int_{\partial\Omega} \frac{\partial S}{\partial \eta}\,dS = 0 \tag{18}$$

$$\text{Similarly, } \int_\Omega \Delta I\,dx = \int_\Omega \Delta R\,dx = 0 \tag{19}$$

The following result is established by Green's formula and the no-flux boundary conditions as,

$$\int_\Omega \left(\frac{\Delta S}{S} - \frac{\Delta S^2}{S^2}\right)dx = \int_{\partial\Omega} \frac{1}{S}(\nabla S \Delta \eta)\,dS = 0$$

which ensure

$$\int_\Omega \frac{\Delta S}{S}\,dx = \int_\Omega \frac{\Delta S^2}{S^2}\,dx = 0$$

By the same arguments, we also can write

$$\int_\Omega \frac{\Delta I}{I}\,dx = \int_\Omega \frac{\Delta I^2}{I^2}\,dx = 0$$

$$\text{and} \int_\Omega \frac{\Delta R}{R}\,dx = \int_\Omega \frac{\Delta R^2}{R^2}\,dx = 0$$

Then using the above arguments, we have

$$\frac{\partial V_1}{\partial t} = -d_1 S_0 \int_\Omega \frac{\Delta S^2}{S^2} dx + mS_0 \int_\Omega \left(2 - \frac{S}{S_0} - \frac{S_0}{S}\right) dx + n\int_\Omega R\left(1 - \frac{S}{S_0}\right) dx$$

$$-m\int_\Omega \frac{I}{1+\alpha I}\left(1+\alpha I - \Re_0\right) dx - \left(m+n\right)\int_\Omega R dx$$

Thus, whenever $R_0 \le 1$, we get $\frac{\partial V_1}{\partial t} \le 0$.

While $S = S_0, I = R = 0$; we simplify, $\frac{\partial V_1}{\partial t} = 0$. As the end, the equilibirum E_0 is the largest compact invariant set in $\left\{(S,I,R) \in C\left(\Omega, \mathbb{R}^3_+\right) : \frac{\partial V_1}{\partial t} = 0\right\}$. Finally, recalling LaSalle's invariance principle [11], we obtain $\lim_{t\to\infty}\left(S(x,t), I(x,t), R(x,t)\right) \to E_0$; that measure, when $R_0 \le 1$, the DFE singleton $E_0 = \left(\frac{a}{m}, 0, 0\right)$ is globally asymptotically stable. Hence the proof is established.

V. Numerical Simulations

This section justified the theoretical results considering few numerical simulations. The results are presented graphically with multiple reproduction numbers of the model (1) using the MATLAB language. We consider the initial values as [2]:

$$\begin{cases} S^0(x) = 90\sin(x) + 400, & \text{in } \Omega \\ I^0(x) = 90\cos(x) + 400, & \text{in } \Omega \\ R^0(x) = 90\sin(0.5x) + 30, & \text{in } \Omega \end{cases}$$

and the no-flux boundary condition is:

$$S_\eta = I_\eta = R_\eta = 0 \text{on } \partial\Omega.$$

Example 1. We assume the parameters values as follows:

$a = 100; \alpha = 10; \beta = 0.000000005; \gamma = 0.8; m = 0.0009; n = 0.003$ with $d_i = 1, i = 1,2,3$. The established formula of R_0 (14) yields the reproduction number as $R_0 = 0.6$. Thus, the results of theorem (4) confirms that the selected parameters leading the disease-free equilibrium (DFE) which is designed in Figure 2.

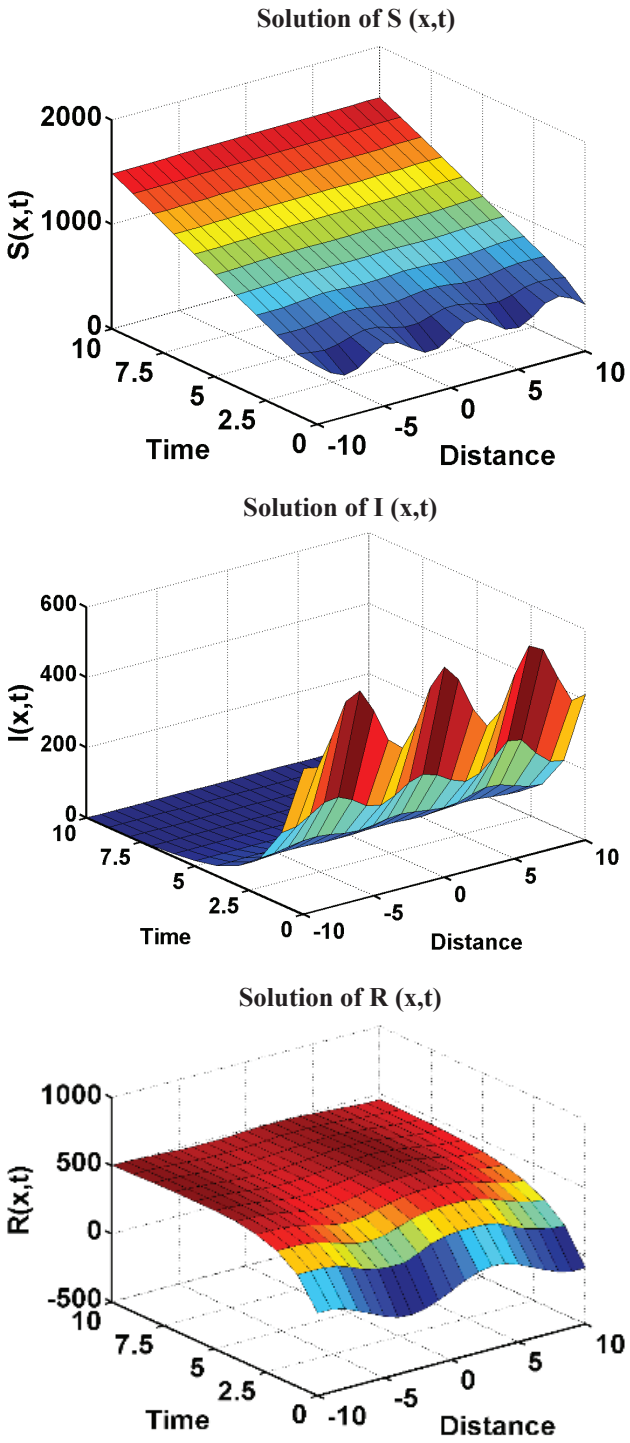

Fig. 2. DFE of the model (1) with time-space domain.

The formula (6) ascertain the analytic values of DFE singleton as $E_0(111111.1, 0, 0)$ and the graphical results are sounds to confirm the theory-computation managements.

Example 2. Now we consider the following system parameters as

$a = 1; \alpha = 10; \beta = 0.000005; \gamma = 0.8; m = 0.0005; n = 0.003$

As before, the term (14) gives the reproduction number, $R_0 = 19.6$, too large, ensure the analytical results presented in theorem and the accepted findings are decorated in Figure 3.

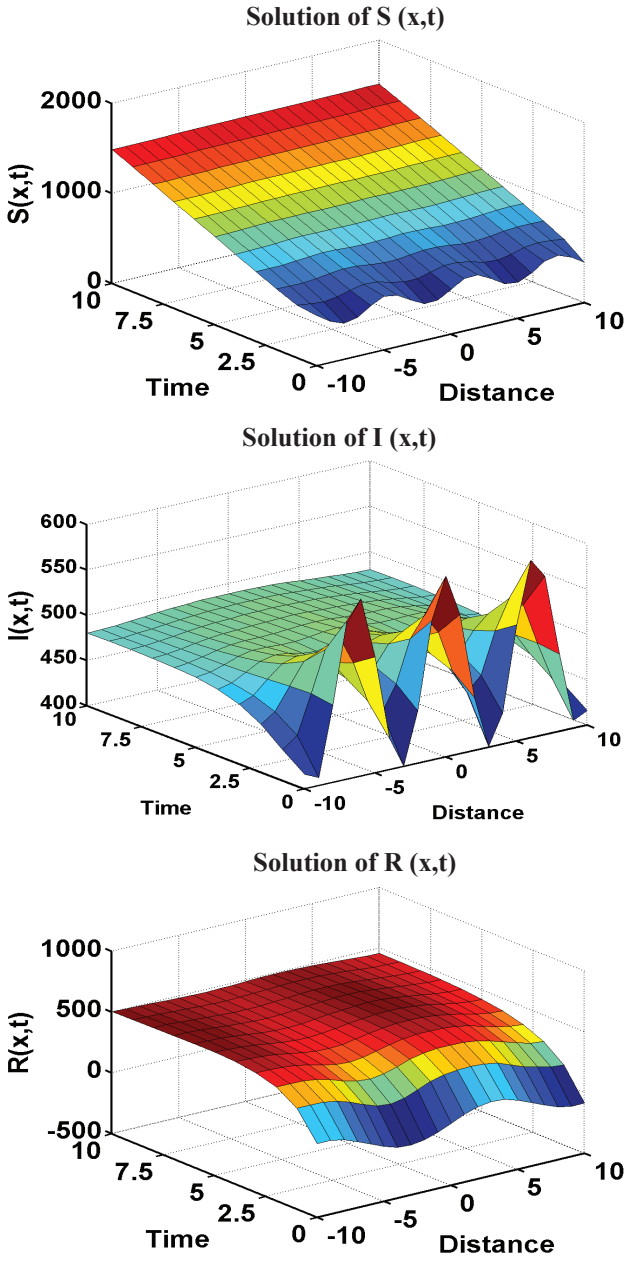

Fig. 3: Endemic equilibrium (EE) of the problem (1) with time-space domain.

Example 3. Finally we choose the following parameters values:

$a = 100; \alpha = 10; \beta = 0.0000006; \gamma = 0.01; m = 0.004; n = 0.003;$

Established formula in (14) yields the value of threshold number as $R_0 = 3.80$ which mean the disease is still not under control and confirms the theory as prescribed in earlier section. The results are decorated in Figure 4.

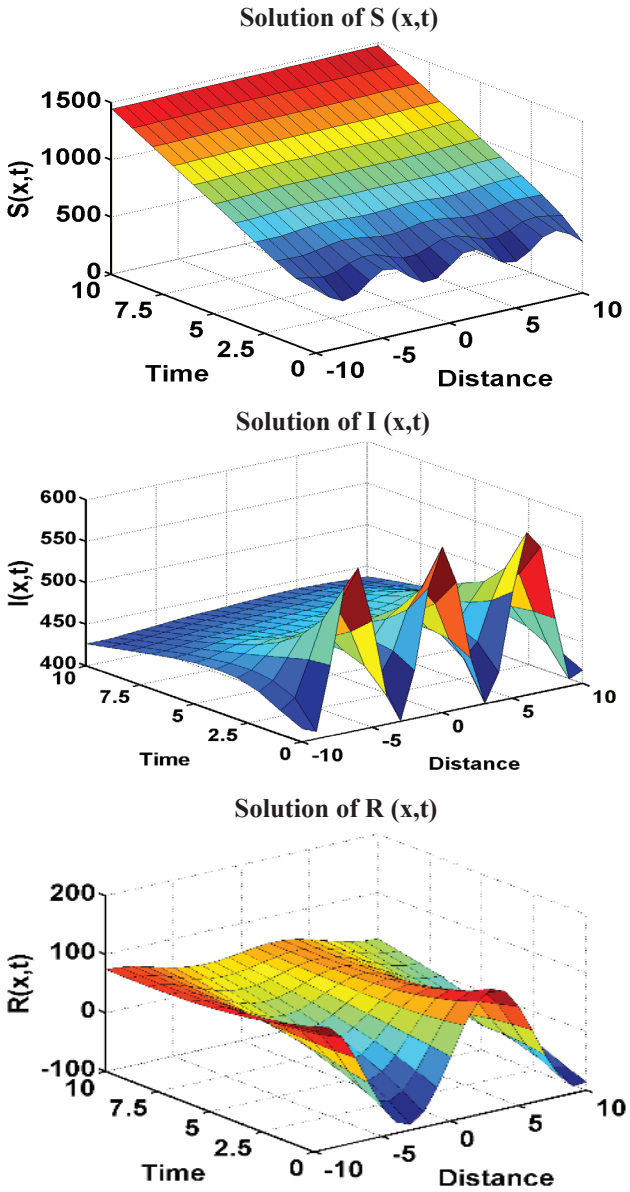

Fig. 4. Endemic equilibrium (EE) of the problem (1) with time-space domain.

VI. Concluding Remarks

This paper has studied a spatio-temporal SIR model proposed for various disease dynamics. We have analysed theoretical inter-locution of DFE steady-state, disease/endemic equilibrium, and threshold values at the primary state, which are used as auxiliary results throughout the paper. The solution of existence and uniqueness of the proposed model have been established. The positivity of solutions and boundedness are also established. The problem is investigated locally and globally for asymptotic behaviours observation. To prove our system's local and global stability, we configured the

worthy Lyapunov functions. We established the results for global stability of DFE and EE of the system by LaSalle's invariance principle. We present a series of numerical examples to check the theoretical results. It is proven that the numerical simulations agreed with the analytic outcomes of the model. The present study and analysis may help to forecast and predict the probable treatment strategies. It is also interesting to extend this study to a vaccination model against malignant diseases [22-29]. The bifurcation analysis and chaos theory analysis in this model are another open problem for future studies [30].

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Conflict of interest

The authors declare no conflict of interest.

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