On a Reaction-Diffusion Model of COVID-19

Rebecca Walo Omana∗, Issa Ramadhani Issa∗, Francis-Didier Tshianyi Mwana Kalala∗

Faculty of Sciences, Regional Center for Doctoral Education in Mathematics and Computer Science, University of Kinshasa, Kinshasa, Democratic Republic of the Congo

Email address:
rwal@yahoo.fr (Rebecca W. O.), issaramadhani41@gmail.com (Issa R. I.), tshiakal@gmail.com (Francis-Didier T. M. K.)
∗Corresponding author

To cite this article:
Rebecca Walo Omana, Issa Ramadhani Issa, Francis-Didier Tshianyi Mwana Kalala. On a Reaction-Diffusion Model of COVID-19. International Journal of Systems Science and Applied Mathematics. Vol. 6, No. 1, 2021, pp. 22-34. doi: 10.11648/j.ijssam.20210601.13

Received: November 23, 2020; Accepted: January 11, 2021; Published: March 26, 2021

Abstract: Nowadays mathematical models play a major role in epidemiology since they can help in predicting the spreading and the evolution of diseases. Many of them are based on ODEs on the assumption that the populations being studied are homogenous sets of fixed points (individuals) but actually populations are far from being homogenous and people are constantly moving. In fact, thanks to science progresses, distances are no longer what they used to be in the past and a disease can travel and reach out even the most remote places on the globe in a matter of hours. HIV and Covid-19 outbreaks are perfect illustrations of how far and fast a disease can now spread. When it comes to studying the spatio-temporal spreading of a disease, instead of ODEs dynamic models the Reaction-Diffusion ones are best suited. They are inspired by the second Fick’s law in physics and are getting more and more used. In this article we make a study of the spatio-temporal spreading of the COVID-19. We first present our SEIR dynamic model, we find the two equilibrium points and an expression for the basic reproduction number (\(R_0\)), we use the additive compound matrices and show that only one condition is necessary to show the local stability of the two equilibrium points instead of two like it is traditionally done, and we study the conditions for the DFE (Disease Free Equilibrium point) and the EE (Endemic Equilibrium point) to be globally asymptotically stable. Then we construct a diffusive model from our previous SEIR model, we investigate on the existence of a traveling wave connecting the two equilibrium thanks to the monotone iterative method and we give an expression for the minimal wave speed. Then in the last section we use the additive compound matrices to show that the DFE remains stable when diffusion is added whereas there will be appearance of Turing instability for the EE once diffusion is added. The conclusion of our article emphasizes the importance of barrier gestures and the fact that the more people are getting tested the better governments will be able to handle and tackle the spreading of the disease.

Keywords: Reaction-Diffusion, COVID-19, Traveling Wave, Upper-solution, Lower-solution, Turing Instability

1. Introduction

The COVID-19 was declared a pandemic by the WHO on the 30th Juinari 2020. The responsible agent is a coronavirus (SARS-Cov2) that spreads between people thanks to close contacts, usually via droplets produced by coughing, sneezing or talking. The droplets usually fall onto surfaces or to the ground rather than remaining in the air making it also possible for people to be infected by touching a contaminated surface or any contaminated object. According to the updated information available [27], incubation period ranges from 2 to 14 days and the main symptoms are fever, loss of appetite, shortness of breath, cough, fatigue, muscle aches and pain. The majority of the infected individuals are asymptomatic and tend not to be tested thou they do play a role in the spreading of the disease. The recovery time which usually ranges from 2 to 6 weeks differs from person to person and it happens that even after that period some people still complain to not be fully recovered.

Depending on the main purpose, dynamic models usually try to encapsulate as much as possible important features of the disease in the simplest way [3-6, 15-19] to provide a comprehensive view on the disease dynamic. That is why the majority of the current models on COVID-19 are very detailed in classes. For instance a quarantined class and/or
an hospitalization class are often taken into account leading to models with 5 to 8 classes [20-26]. Knowing how challenging it is to find front traveling waves for R-D models of three or more than three equations, we have chosen to build a simpler model with only four classes (the susceptibles, the asymptomatic infected individuals, the symptomatic infected individuals and the removed). Other approaches are regularly used to investigate on the existence existence of a traveling wave [5, 7] but here we use the monotone iterative method by setting up a pair of ordered super-solutions. We consider that no major action is taken to stop the spreading, therefore we have no quarantine and people are still free to move. The interactions between the four classes are given in the Figure 1

![Dynamic graph of COVID-19 transmission.](image)

Figure 1. Dynamic graph of COVID-19 transmission.

and the assumptions we make are the following:
1. Every new-born is susceptible i.e there are only horizontal transmission;
2. An asymptomatic infected individual is an infectious person presenting no or very few symptoms;
3. A symptomatic infected individual is an infectious person presenting symptoms of COVID-19;
4. Every contact with an infectious person does not always lead to a transmission of SARS-Cov2;
5. After an infectious contact there is always an incubating period but we do not take it into account here;
6. After a susceptible has been infected by either an asymptomatic infected individual or a symptomatic infected individual, he/she will go through an asymptomatic state he can remain into until he/she is totally healed or he/she will leave that state as soon as sufficient symptoms begin to appear;
7. A symptomatic infected individual can either die of COVID-19 or get healed;
8. We do not take into account reinfection by COVID-19.
9. The entire population has a per-capita death rate independent of COVID-19.

2. A Reaction Model of COVID-19

Consider a population with size $N$. We can divide it into sub-populations and denote their fractions by $S, E, I,$ and $R$ which respectively represent the fraction of susceptible, the fraction of asymptomatic infected individuals, the fraction of symptomatic infected individuals and the fraction of removed. Thus the sub-populations verify the identity: $S + E + I + R = 1$. Based on the assumptions made previously we can set a reaction model of COVID-19 as follows:

$$
\begin{align*}
S' &= \Lambda - \beta IS - \eta ES - dS \\
E' &= \beta IS + \eta ES - \epsilon E - \theta E - dE \\
I' &= \epsilon E - \mu I - \gamma I - dI \\
R' &= \gamma I + \theta E - dR.
\end{align*}
$$

(1)

The coefficients used into our model are explained in the following table:

| Coefficient | Meaning |
|-------------|---------|
| $\Lambda$   | The recruitment |
| $\beta$     | The infective contact rate symptomatic infected/susceptible |
| $\eta$      | The infective contact rate asymptomatic infected/susceptible |
| $\mu$       | The induced-disease death rate due to COVID-19 |
| $\epsilon$  | The transfer rate from asymptomatic infected to symptomatic infected |
| $d$         | The natural death rate out of $\mu$ |
| $\theta$    | The natural recovery rate of symptomatic infected individuals |
| $\gamma$    | The natural recovery rate of asymptomatic infected individuals |
The last equation in (1) does not intervene into the transmission of the disease, we can simplify our system by reducing it into three equations as follows:

\[
\begin{align*}
S' &= \Lambda - \beta IS - \eta ES - dS \\
E' &= \beta IS + \eta ES - (\epsilon + \theta)E - dE \\
I' &= \epsilon E - \mu I - \gamma I - dI
\end{align*}
\] (2)

To ensure the well posedness of the system we consider the proportion of the population in:

\[G = \{(S, E, I) \in \mathbb{R}_+^3 : S + E + I \leq 1\}.\] (3)

### 2.1. Equilibrium points and \( R_0 \)

The equilibrium points are

\[
\bar{u} = (S_0, 0, 0) = \left(\frac{\Lambda}{d\epsilon}, 0, 0\right)
\] (4)

for the disease free equilibrium (DFE) and

\[
u^* = \left(\frac{MN}{\epsilon \beta + \eta M}, \frac{\Lambda(\epsilon \beta + \eta M) - dMN}{N(\epsilon \beta + \eta M)}, -\frac{(\Lambda(\epsilon \beta + \eta M) - dMN)}{MN(\epsilon \beta + \eta M)}\right)
\] (5)

for the endemic equilibrium (E.E).

To find \( R_0 \) we use the next generation operator [18, 21]. Our DFE is given by \( \bar{u} = (\frac{\Lambda}{d\epsilon}, 0, 0) = (1, 0, 0) \) due to the fact that at this equilibrium point the entire population is susceptible i.e the fraction of the healthy people is 1. So

\[
J_u = \left(\begin{array}{ccc}
-d & \frac{-\eta\beta}{\epsilon} & \frac{-\beta\Delta}{\epsilon} \\
0 & \frac{\eta}{\epsilon} - (\epsilon + \theta + d) & \beta\frac{\Delta}{\epsilon} \\
0 & -d & -(\mu + \gamma + d)
\end{array}\right)
\] (6)

we obtain the following sub-matrices

\[
F = \left(\begin{array}{c}
\frac{\Lambda\Delta}{\epsilon d} \\
\frac{\Lambda\Delta}{\mu + \gamma + d}
\end{array}\right), \quad V = \left(\begin{array}{cc}
\frac{\Lambda\Delta}{\epsilon (\epsilon + \theta + d)} & 0 \\
0 & (\mu + \gamma + d)
\end{array}\right).
\] (7)

Then

\[
V^{-1} = \left(\begin{array}{c}
\frac{1}{\epsilon (\epsilon + \theta + d)} \\
(\mu + \gamma + d)
\end{array}\right)
\] and \( -FV^{-1} = \left(\begin{array}{c}
-d & 0 \\
0 & (\mu + \gamma + d)
\end{array}\right).
\] (8)

Hence the basic reproduction number is:

\[
R_0 = \rho(-FV^{-1}) = \frac{\Lambda(\epsilon \mu + \gamma + d) + \beta\epsilon}{d(\epsilon + \theta + d)(\mu + \gamma + d)} = \frac{\Lambda(\eta M + \beta\epsilon)}{dMN}.
\] (9)

**Theorem 2.1 (Existence of equilibria).** If \( R_0 \leq 1 \), the model (2) always has a disease-free equilibrium \( \bar{u} = (\frac{\Lambda}{d\epsilon}, 0, 0) \). If \( R_0 > 1 \), the model (2) has exactly one endemic equilibrium \( \nu^* = \left(\frac{\Lambda(\epsilon \beta + \eta M) - dMN}{N(\epsilon \beta + \eta M)}, \frac{\epsilon(\Lambda(\epsilon \beta + \eta M) - dMN)}{MN(\epsilon \beta + \eta M)}\right) \).

**Proof.** Let us suppose that \( R_0 \leq 1 \) then we have the existence of the EE

\[
u^* = (S^*, E^*, I^*) = \left(\frac{\Lambda(\epsilon \beta + \eta M) - dMN}{N(\epsilon \beta + \eta M)}, \frac{\epsilon(\Lambda(\epsilon \beta + \eta M) - dMN)}{MN(\epsilon \beta + \eta M)}\right).
\]

If \( R_0 := \frac{\Lambda(\eta M + \beta\epsilon)}{dMN} \geq 1 \) then \( \Lambda(\eta M + \beta\epsilon) = dMN \) therefore \( E^* = \frac{\epsilon(\Lambda(\epsilon \beta + \eta M) - dMN)}{N(\epsilon \beta + \eta M)} = 0 \) and \( I^* = \frac{\epsilon(\Lambda(\epsilon \beta + \eta M) - dMN)}{MN(\epsilon \beta + \eta M)} = 0 \).

Thus \( \nu^* = \left(\frac{MN}{\epsilon \beta + \eta M}, 0, 0\right) \). for the proportion of the population being entirely in the first component we have

\[
\frac{MN}{\epsilon \beta + \eta M} = 1 \iff \frac{\epsilon \beta + \eta M}{MN} = 1 \iff \frac{\Lambda(\epsilon \beta + \eta M)}{dMN} = \frac{\Lambda}{d} \quad \text{(which keeps the proportion since } \frac{\Lambda}{d} = 1)\]

Thus \( S^* = \frac{\Lambda}{d} \) and the unique equilibrium point in this situation is the disease free one.

If \( R_0 < 1 \) then \( \Lambda(\eta M + \beta\epsilon) < dMN \). Hence \( E^* = \frac{\epsilon(\Lambda(\epsilon \beta + \eta M) - dMN)}{N(\epsilon \beta + \eta M)} < 0 \) and \( I^* = \frac{\epsilon(\Lambda(\epsilon \beta + \eta M) - dMN)}{MN(\epsilon \beta + \eta M)} < 0 \). Both components must be positive to ensure the existence of an endemic equilibrium, therefore there is none.

Let us now suppose that \( R_0 > 1 \), then \( \Lambda(\eta M + \beta\epsilon) > dMN \). Let us check for positivity of the following components:

\[
E^* = \frac{\epsilon(\Lambda(\epsilon \beta + \eta M) - dMN)}{N(\epsilon \beta + \eta M)}, \quad I^* = \frac{\epsilon(\Lambda(\epsilon \beta + \eta M) - dMN)}{MN(\epsilon \beta + \eta M)}.
\]
\[
e^{(\Lambda(c+\eta M) - d MN)} > 0. \text{ Thus there exists an endemic equilibrium } u^* \text{ as defined in (5).}
\]

### 2.2. Stability of the Equilibria

**Theorem 2.2.** If

\[
\mathcal{R}_0 := \frac{\Lambda(\beta \epsilon + \eta (\mu + \gamma + d))}{d(\epsilon + \theta + d)(\mu + \gamma + d)} < 1
\]

then the DFE given in (4) is locally asymptotically stable in \( G \).

If

\[
\mathcal{R}_0 := \frac{\Lambda(\beta \epsilon + \eta (\mu + \gamma + d))}{d(\epsilon + \theta + d)(\mu + \gamma + d)} > 1
\]

then the E.E given in (5) is locally asymptotically stable in \( G \).

**Proof:** It suffices to show that the eigenvalues of the two Jacobian matrices at the two equilibria have real negative part. Next we use a property of the additive compound matrices to state:

**Theorem 2.3.** Let \( J_{0} \) and \( J_{u} \), be the Jacobian matrices at the DFE and the EE. If

\[
-|J_{0}| > 0
\]

then the DFE given in (4) is locally asymptotically stable in \( G \).

If

\[
-|J_{u}| > 0
\]

then the E.E given in (5) is locally asymptotically stable in \( G \).

**Proof:** To show that \( u \) is stable we must prove under which conditions \(-|J_{u}| > 0 \) and \( \mu(J_{u}^{[2]}) < 0 \). Where \( \mu \) is a Lozinsk\’u mesure on \( M_{n \times n} \),

\[
-|J_{u}| > 0 \iff d \begin{vmatrix} \Delta \eta & -N \\ \epsilon & -M \end{vmatrix} > 0
\]

\[
\iff d MN - \Lambda \epsilon \beta - \eta \Lambda M > 0
\]

\[
\iff d MN > \Lambda (\epsilon \beta + \eta M)
\]

\[
\iff \frac{\Lambda (\epsilon \beta + \eta M)}{d MN} < 1
\]

\[
\iff \mathcal{R}_0 < 1.
\]

The second compound matrix of \( J_{0} \) is given by:

\[
J_{u}^{[2]} = \begin{pmatrix}
\frac{\Delta \eta}{\epsilon} & -(N + d) & \frac{\Delta \beta}{\epsilon} \\
0 & -d + M & \frac{\Delta \beta}{\epsilon} \\
0 & 0 & \frac{\Delta \beta}{\epsilon} - (M + N)
\end{pmatrix}.
\]

(10)

Let us use \( \mu_1 \) as our Lozinsk\’u mesure with

\[
\mu_1(A) = \sup_k \left( Re(a_{kk}) + \sum_{i \neq k} |a_{ik}| \right), \quad A \in M_n(\mathbb{R}^n).
\]

(11)

From the first column we have:

\[
\frac{\Lambda \eta}{d} - (N + d) + \epsilon < 0 = \frac{\Lambda \eta}{d} - 2d - \theta - \epsilon < 0
\]

\[
\iff \frac{\Lambda \eta}{d} - 2d - \theta < 0
\]

\[
\iff \eta < 2d + \theta
\]

\[
\iff d > \eta - \theta
\]

(12)

We proceed the same way for the second and the third columns and find respectively:

\[
d > \beta - M \text{ and } d > \frac{\beta + 2\eta - (\mu + \gamma + \epsilon + \theta)}{2}
\]

Two conditions are necessary to the stability of the equilibrium \( \bar{u} \). The first one is the sign of the Jacobian \( J_{\bar{u}} \).

Indeed if \(-|J_{\bar{u}}| > 0 \) then \( \mathcal{R}_0 < 1 \) and this condition is necessary for the local stability of \( \bar{u} \). If \( \mu(J_{\bar{u}}^{[2]}) < 0 \), then we get a condition on the parameters and this has no much meaning and impact for our model.

**Theorem 2.4.** When \( \eta \leq \beta \) and \( \mathcal{R}_0 < 1 \), then disease-free equilibrium \( \bar{u} \) for (2) is globally asymptotically stable.

**Proof:** We use an approach given by Zhisheng Shuai and P. Van Den Driessche to construct our Lyapunov function [22, 29]. Let

\[
F = \begin{pmatrix}
\beta IS + \eta ES \\
0
\end{pmatrix} \geq 0, \quad V = \begin{pmatrix}
\frac{NE}{M} - \epsilon E
\end{pmatrix} \leq 0,
\]

(13)

\( F, V \) and \( V^{-1} \) defined like in (7) and (8).

\[
V^{-1} F = \begin{pmatrix}
\frac{\eta S_0}{MN} & 0 \\
\frac{\beta S_0}{N} & 0
\end{pmatrix} = \begin{pmatrix}
\epsilon S_0 & \frac{\beta S_0}{N} \\
\frac{\epsilon S_0}{MN} & \frac{\beta S_0}{MN}
\end{pmatrix}.
\]

(14)

If \( w^T = (x y) \) denotes the left eigenvector of \( V^{-1} F \) then we have

\[
(x y) \begin{pmatrix}
\frac{\eta S_0}{N} & \frac{\beta S_0}{N} \\
\epsilon S_0 & \frac{\beta S_0}{MN}
\end{pmatrix} = (x y) R_0
\]

(15)

and \( w^T = (1, 1) \).

If \( X = \begin{pmatrix}
E \\
I
\end{pmatrix} \) then we have

\[
w^T V^{-1} X = \begin{pmatrix}
1 \\
\frac{1}{MN}
\end{pmatrix} \begin{pmatrix}
1 & 0 \\
\frac{1}{M} & 1
\end{pmatrix} \begin{pmatrix}
E \\
I
\end{pmatrix}
\]

\[
= \begin{pmatrix}
(M + \epsilon) E + \frac{I}{M}
\end{pmatrix}
\]

*Algorithms on the calculation of \( \mu(J_{\bar{u}}^{[2]}) \) are given in [8-10]*
and the Lyapounov function is given by:

\[ L = \frac{1}{M} \left[ \frac{(M + \epsilon)E + I}{N} \right] = \frac{1}{(d + \mu + \gamma)} \left[ \frac{(d + \mu + \gamma + \epsilon)E + I}{(\epsilon + \theta + d)} \right]. \]  

(16)

We have

\[ L' = \frac{\partial L}{\partial E'} E' + \frac{\partial L}{\partial I'} I' = \frac{1}{M} \left[ \frac{(M + \epsilon)E' + I'}{N} \right]. \]

Therefore

\[ L' = -\frac{(M + \epsilon)}{MN} (S_0 - S)(\eta E + \beta I) + \frac{1}{MN} [(M + \epsilon)(\eta S_0 E - N E + \beta S_0 I) + \epsilon N E - MNI]. \]

Since \( \frac{(M + \epsilon)}{MN} (S_0 - S)(\eta E + \beta I) \ll 0 \) the expression

\[ (H) = \frac{1}{MN} [(M + \epsilon)(\eta S_0 E - N E + \beta S_0 I) + \epsilon N E - MNI] \]

\[ = \frac{\eta M + \epsilon \beta}{MN} S_0 E + \frac{\eta M + \epsilon \beta}{MN} S_0 I + \frac{\epsilon(\gamma - \beta)}{MN} S_0 E + \frac{M(\beta - \eta)}{MN} S_0 I - (E + I) \]

\[ = (R_0 - 1)(E + I) + \frac{S_0(\eta - \beta)}{MN} (\epsilon E - M I) \]

is negative.

From the hypothesis, \( R_0 \leq 1 \) and from (13) \( \epsilon E - M I \geq 0 \), thus with \( \eta \leq \beta \) we have \( (H) \leq 0 \) and therefore \( L' \leq 0 \).

**Theorem 2.5. [Global stability of the EE]**

The endemic equilibrium \( I^* \) for (2) is globally asymptotically stable when \( R_0 > 1 \).

**Proof.** Let

\[ L_1 = S - S^* - S^* \ln \frac{S}{S^*}, \quad L_2 = E - E^* - E^* \ln \frac{E}{E^*}, \quad \text{and} \quad L_3 = I - I^* - I^* \ln \frac{I}{I^*}. \]

\[ L_1' = \frac{\partial L_1}{\partial S'} S' = \frac{(S - S^*)}{S} (\beta S^* I^* + \eta S^* E^* + dS^* - \beta SI - \eta SE - dS) \]

\[ = (S - S^*) \left[ -d(S - S^*) + \beta(S^* I^* - SI) + \eta[S^* E^* - SE] \right] \]

\[ = -d \frac{(S - S^*)^2}{S} \left[ \beta S^* I^* - SI \right] + \eta S^* E^* \left[ \frac{1}{S} - \frac{SE^*}{S^*} \right] \]

\[ \leq \beta S^* I^* \left( \frac{1}{I^*} - \ln \frac{I}{I^*} \right) + \eta S^* E^* \left( \frac{1}{I^*} - \ln \frac{I}{I^*} \right) \]

\[ := a_{13} G_{13} + a_{12} G_{12}. \]

\[ L_2' = \frac{\partial L_2}{\partial E'} E' = -N \left( \frac{E - E^*}{E} \right)^2 + \frac{\beta}{E} (S - S^*) (SI - S^* I^*) + \eta \left( \frac{E - E^*}{E} \right) (SE - S^* E^*) \]

\[ \leq \beta S^* I^* \left( \frac{SI}{S^* I^*} - \ln \frac{SI}{S^* I^*} - \ln \frac{E}{E^*} \right) + \eta S^* E^* \left( \frac{SE}{S^* E^*} - \ln \frac{SE}{S^* E^*} - \frac{S}{S^*} + \ln \frac{S}{S^*} \right) \]

\[ := a_{21} G_{21} + a_{12} G_{12}. \]

and similarly

\[ L_3' = \frac{\partial L_3}{\partial I'} I' = \left( I - I^* \right) \left( \epsilon E^* + M I^* + \epsilon E - M I \right) \leq \epsilon E^* \left( \frac{E}{E^*} - \ln \frac{E}{E^*} - \frac{I}{I^*} + \ln \frac{I}{I^*} \right) \]

\[ := a_{32} G_{32}. \]

\[ a_{13} = \beta S^* I^*, \quad a_{12} = \eta E^* S^*, \quad a_{21} = \beta S^* I^*, \quad a_{12} = \eta E^* S^*, \quad a_{32} = \epsilon E^*. \]
Asymptotically the system (19) satisfies the following contacts with people able to diffuse. Then we can formulate way, we suppose that the symptomatic infectious still have

The associated weighted diagram given in Figure 2 has three vertices and two cycles. Along each cycle, $G_{21} + G_{32} + G_{13} = 0$ and $G_{21} + G_{12} = 0$. Then there exist $c_i$, $1 \leq i \leq 3$, such that $L = \sum_{i=1}^{3} c_i L_i$ is a Lyapounov function for (2).

Let us find the relations between the $c_i$’s. $d^+(1) = 1 \implies c_2 a_{21} = c_1 a_{13}$, since $a_{21} = a_{13}$ then $c_2 = c_1$. $d^+(2) = 1 \implies c_3 a_{32} = c_2 a_{21} \implies c_3 = c_2 a_{32} / a_{21} = c_2 \beta S^* I^* / c E^*$. Our Lyapounov function is given by:

$$L = L_1 + L_2 + \beta S^* I^* / c E^*$$

(17)

3. A Reaction-Diffusion Model on COVID-19

Assume now that the individual in the population can move (diffuse) with the same diffusion coefficient. If the susceptibles and the asymptomatic infectious are free to move the same way, we suppose that the symptomatic infectious still have contacts with people able to diffuse. Then we can formulate our R-D model like:

$$\begin{align*}
S_t &= \Lambda + \triangle S - \beta S I - \eta E S - d S \\
E_t &= \triangle E + \beta I S + \eta E S - (\epsilon + \theta + d) E \\
I_t &= \triangle I + \epsilon E - (\mu + \gamma + d) I
\end{align*}$$

(18)

Using wave coordinates $\xi = x + ct$ in (18) yields:

$$\begin{align*}
0 &= \Lambda + S' - c S' - (\beta I + \eta E) S - d S \\
0 &= E'' - c E' + (\beta I + \eta E) S - N E \\
0 &= I'' - c I' + \epsilon E - M I
\end{align*}$$

(19)

Asymptotically the system (19) satisfies the following boundary conditions:

$$\begin{align*}
\begin{pmatrix} S \\ E \\ I \end{pmatrix} \bigg|_{(\xi) = \pm \infty} &= \begin{pmatrix} S^* \\ E^* \\ I^* \end{pmatrix}
\end{align*}$$

(20)

Linearizing (19) about $(\frac{\Lambda}{2}, 0, 0) = (1, 0, 0)$ we obtain:

$$\begin{align*}
0 &= \Lambda + S'' - c S'' - d S \\
0 &= E'' - c E' + (\eta - N) E \\
0 &= I'' - c I' - M I
\end{align*}$$

(22)

The second equation in (22) provides the speed of the wave. In fact its characteristic equation is:

$$r^2 - cr + (\eta - N) = 0.$$ 

To ensure the existence of real solutions we must have $c \geq 2\sqrt{\eta - N}$.

Hence the minimal speed is

$$c^* = 2\sqrt{\eta - N}$$

(23)

and the roots to the characteristic equation are:

$$r_{1,2} = \frac{c \pm \sqrt{c^2 - 4(\eta - N)}}{2},$$

(24)

and those of the third:

$$p_{1,2} = \frac{c \pm \sqrt{c^2 + 4M}}{2}.$$
4. Existence of a Traveling Wave

To prove the existence of a front traveling wave solution to (22) we shall use the monotone iterative method which relies on the following principle:

**Principle 4.1.** [Monotone Iterative Method] Consider the general second order ODE with Dirichlet boundary conditions given by:

\[
\begin{cases}
    u''(t) = f(t, u(t), u'(t)), & t \in I \equiv [a, b], \\
    u(a) = A, u(b) = B,
\end{cases}
\]

Hence the boundary conditions give:

\[
\begin{pmatrix}
    \bar{S}(\xi) \\
    \bar{E}(\xi) \\
    \bar{I}(\xi)
\end{pmatrix}
(\rightarrow{\infty}) = \begin{pmatrix}
    0 \\
    0 \\
    0
\end{pmatrix} \leq \begin{pmatrix}
    \frac{\Lambda}{d} S^* \\
    0 \\
    0
\end{pmatrix}
\]

\[
\begin{pmatrix}
    \bar{S}(\xi) \\
    \bar{E}(\xi) \\
    \bar{I}(\xi)
\end{pmatrix}
(\rightarrow{0}) = \begin{pmatrix}
    0 \\
    0 \\
    0
\end{pmatrix} \leq \begin{pmatrix}
    \frac{S^*}{E^*} \\
    \frac{E^*}{I^*}
\end{pmatrix}
\]

Thus \((\bar{S}(\xi), \bar{E}(\xi), \bar{I}(\xi)) = (0, 0, 0)\) is a lower-solution.

**Lemma 4.2.** Suppose that \(MN > \epsilon \beta\) and let \(\bar{X}(\xi) = (\bar{S}(\xi), \bar{E}(\xi), \bar{I}(\xi))\) be a function defined by:

\[
\begin{align*}
    \bar{S}(\xi) &= \frac{d}{\beta} S^* \forall \xi \in \mathbb{R}; \\
    \bar{E}(\xi) &= \begin{cases}
        E^* \epsilon^{q_1 \xi}, & \xi \leq 0 \\
        E^*, & \xi > 0
    \end{cases}; \\
    \bar{I}(\xi) &= \begin{cases}
        \frac{N}{\beta} E^* \epsilon^{q_1 \xi}, & \xi \leq 0 \\
        I^*, & \xi > 0
    \end{cases}
\end{align*}
\]

with \(q_1 = \frac{c + \sqrt{c^2 - 4d}}{2}\) the greater positive root of the equation \(q^2 - cq + \eta = 0\) and \(a_1 = c\) the non-zero positive root of the equation \(a^2 - ca = 0\). Then \(\bar{X}(\xi)\) is an upper-solution to (19).

**Proof**

Assume that \(\xi \leq 0\).

For the first equation of (19) we have:

\[
\begin{align*}
    \Lambda + \left(\frac{\Lambda}{d} S^*\right)'' - c \left(\frac{\Lambda}{d} S^*\right)' &= \left(\beta \frac{N}{\beta} E^* \epsilon^{q_1 \xi} + \eta E^* \epsilon^{q_1 \xi}\right) \frac{\Lambda}{d} S^* - d \frac{\Lambda}{d} S^* \\
    &= \Lambda - \left(NE^* \epsilon^{q_1 \xi} + \eta E^* \epsilon^{q_1 \xi}\right) \frac{\Lambda}{d} S^* - d \frac{\Lambda}{d} S^* \\
    &\leq \Lambda - \left(NE^* \epsilon^{q_1 \xi} + \eta E^* \epsilon^{q_1 \xi}\right) S^* - \Lambda S^* \\
    &\leq \Lambda - \left(NE^* \epsilon^{q_1 \xi} + \eta E^* \epsilon^{q_1 \xi}\right) S^* - \Lambda \\
    &\Rightarrow -E^* \left(NE^* \epsilon^{q_1 \xi} + \eta E^* \epsilon^{q_1 \xi}\right) S^* < 0
\end{align*}
\]

with \(f : I \times \mathbb{R}^2 \rightarrow \mathbb{R}\) a continuous function and \(A, B \in \mathbb{R}\). If in \(C^2(I)\) there exist \(\bar{U}(t)\) a lower solution to (25) and \(\bar{U}(t)\) an upper solution to (25) such that \(\bar{U}(t) \leq \bar{U}(t)\) on \(I\). Then the existence of a solution to the problem (25) lying between \(\bar{U}(t)\) and \(\bar{U}(t)\) is proved.

**Proof**

It is obvious the last two equations of (19) vanish and for the first one we have \(\Lambda \geq 0\).
For the first equation of (19) we have:

\[
\Lambda + \left( \frac{\Lambda}{d} S_* \right)' - e \left( \frac{\Lambda}{d} S_* \right) - (\beta I_* + \eta E_*) \frac{\Lambda}{d} S_* - d \frac{\Lambda}{d} S_* \leq \Lambda - (\beta I_* + \eta E_*) S_* - \Lambda \text{ car } \frac{\Lambda}{d} = 1 \text{ et } S_* \leq 1
\]

\[-(\beta I_* + \eta E_*) S_* < 0 \tag{32}\]

For the second equation of (19) we have:

\[
(\beta I_* + \eta E_*) \frac{\Lambda}{d} S_* - NE_* \leq (\beta I_* + \eta E_*) S_* - NE_* = 0. \tag{33}\]

For the third equation of (19) we have:

\[
MI_* - \epsilon E_* = 0 \tag{34}\]

**Proof**

Assume

\[
R_0 > 1 \text{ then } u^* = \left( \frac{MN}{e^2 + \eta M} - \lambda(e^2 + \eta M) - dMN + \epsilon \lambda(e^2 + \eta M) - dMN \right) \geq (0, 0, 0). \]

Hence the function \( \overline{X}(\xi) \) in (28) is well defined. By their definitions it obvious that:

\[
(S(\xi), E(\xi), I(\xi)) \leq (\overline{S}(\xi), \overline{E}(\xi), \overline{I}(\xi)).
\]

Then by principle on monotone iterative method we are ensured of the existence of a traveling wave \((S(\xi), E(\xi), I(\xi))\) solution to (18) that verifies

\[
(S(\xi), E(\xi), I(\xi)) \leq (S(\xi), E(\xi), I(\xi)) \leq (\overline{S}(\xi), \overline{E}(\xi), \overline{I}(\xi)).
\]

## 5. Turing Instability

When diffusion is added to a dynamic model it can radically change the nature of the equilibrium points and generate diffusion-driven (Turing) instabilities [12-14]. In this section we also use the additive compound matrices to investigate whether there will be appearance of Turing instability.

**Theorem 4.1**. Suppose \( \frac{N}{\lambda} < \frac{\epsilon^2}{M} < M \), the DFE will be locally asymptotically stable for all diffusion matrix \( D \succ 0 \).

**Proof**

The principal minor matrices to \( J_0 \) are

\[
J_1 = \begin{bmatrix} \eta - N & \beta \\ \epsilon & -M \end{bmatrix}, \quad J_2 = \begin{bmatrix} -d & -\beta \\ 0 & -M \end{bmatrix} \quad \text{and} \quad J_3 = \begin{bmatrix} -d & -\epsilon \\ 0 & N - \eta \end{bmatrix}
\]

\[
|J_1| > 0 \iff M(N - \eta) - \epsilon \beta > 0 \\
\iff M(N - \eta) > \epsilon \beta
\]

\[
|J_2| = dM > 0
\]

\[
|J_3| > 0 \iff d(N - \eta) > 0 \\
\iff N - \eta > 0 \\
\iff N > \eta
\]

\[
|J_4| = \frac{\epsilon^2}{N - \eta} < M \tag{a}
\]

\[
|J_5| = dM > 0
\]

\[
|J_6| > 0 \iff d(N - \eta) > 0 \\
\iff N - \eta > 0 \\
\iff N > \eta
\]

(4.8) and (4.9) are obtained due to the fact that \((S^*, E^*, I^*)\) is an equilibrium to (2).

For the boundary conditions we have:

\[
\left( \begin{array}{c} \Sigma \\ \overline{E} \\ \overline{I} \end{array} \right) (-\infty) = \left( \begin{array}{c} \frac{\Sigma}{\overline{E}} \\ \frac{\Sigma}{\overline{I}} \end{array} \right) \geq \left( \begin{array}{c} \frac{\Sigma}{\overline{E}} \\ \frac{\Sigma}{\overline{I}} \end{array} \right)
\]

\[
\left( \begin{array}{c} \Sigma \\ \frac{\Sigma}{\overline{E}} \end{array} \right) (+\infty) = \left( \begin{array}{c} \frac{\Sigma}{\overline{E}} \\ \frac{\Sigma}{\overline{I}} \end{array} \right) \geq \left( \begin{array}{c} \frac{\Sigma}{\overline{E}} \\ \frac{\Sigma}{\overline{I}} \end{array} \right)
\]

Hence, for both values \( \xi \succ 0 \) and \( \xi \leq 0 \), \( \overline{X}(\xi) \) is an upper-solution to (19).

**Theorem 5.1**. Suppose \( \frac{N}{\lambda} < \frac{\epsilon^2}{M} < M \), the DFE will be locally asymptotically stable for all diffusion matrix \( D \succ 0 \).
From (a) and (b) we know that $J_0$ satisfy the minor conditions if $0 < \frac{\epsilon \beta}{N} < M$ i.e under that condition the DFE will remain locally asymptotically stable even if diffusion is introduced in the Reaction model (2).

**Theorem 5.2.** There will always be a Turing instability at the EE defined in (5) for all diffusion matrix $D > 0$.

**Proof**

The principal minor matrices of $J_u^*$ are

$$K_1 = \left[ \begin{array}{cc} -\frac{\Lambda(\epsilon\beta + \eta M)}{MN} & \frac{\eta MN}{\epsilon\beta + \eta M} \\ \frac{\Delta(\epsilon\beta + \eta M) - dMN}{MN} & \frac{-\beta MN}{\epsilon\beta + \eta M} \end{array} \right] \quad K_2 = \left[ \begin{array}{cc} -\frac{\Lambda(\epsilon\beta + \eta M)}{MN} & -\frac{\beta MN}{\epsilon\beta + \eta M} \\ 0 & -M \end{array} \right] \quad \text{and} \quad K_3 = \left[ \begin{array}{cc} -\frac{\Lambda \epsilon \beta}{\epsilon\beta + \eta M} & \frac{\beta MN}{\epsilon\beta + \eta M} \\ \epsilon & -M \end{array} \right]$$

$$|K_1| > 0 \Leftrightarrow \frac{\eta MN}{\epsilon\beta + \eta M} \frac{(\Lambda(\epsilon\beta + \eta M) - dMN)}{MN(\epsilon\beta + \eta M)} - \frac{\epsilon \beta \Lambda N(\epsilon\beta + \eta M)}{MN(\epsilon\beta + \eta M)} > 0$$

$$\Leftrightarrow \frac{\Lambda(\epsilon\beta + \eta M)(\eta MN - \epsilon \beta \Lambda N)}{MN(\epsilon\beta + \eta M)} - d\eta (MN)^2 > 0$$

$$\Leftrightarrow \Lambda N(\epsilon\beta + \eta M)(\eta M - \Lambda \epsilon \beta) > d\eta (MN)^2$$

$$\Leftrightarrow \mathcal{R}_0 > \frac{\eta M}{\eta M - \Lambda \epsilon \beta}$$

$$|K_2| = \frac{M \Lambda(\epsilon\beta + \eta M)}{MN} = \frac{\Lambda(\epsilon\beta + \eta M)}{N} > 0$$

$$|K_3| = \frac{\epsilon \beta MN}{\epsilon\beta + \eta M} - \frac{\epsilon \beta MN}{\epsilon\beta + \eta M} = 0.$$
Table 2. Coefficients Values

| Coefficient | Estimated Values |
|-------------|-----------------|
| \( \Lambda \) | 0.0028           |
| \( N \)     | \( 12 \times 10^9 \) (The estimated population size of Kinshasa) |
| \( p \)     | 0.03            |
| \( \mu \)   | 0.03            |
| \( \epsilon \) | 0.3          |
| \( d \)     | 0.002           |
| \( \theta \) | 0.9             |
| \( \gamma \) | 0.8             |

![Figure 3](image1.png)

Figure 3. When \( \beta = 0.3 \) (\( s_1 = 10 \)) and \( \eta = 0.45 \) (\( s_2 = 15 \)), \( R_0 = 0.6579 \) and the trajectories are going to the DFE in fact \( S=1, E=I=R=0 \).

![Figure 4](image2.png)

Figure 4. If \( s_1 = 40 \) and \( s_2 = 55 \) (\( \beta = 1.2 \) and \( \eta = 1.65 \)), \( R_0 = 2.4546 \). We can see 3 different waves in the susceptible population and in the first weeks after the beginning of the infection the number of susceptibles dwindles quickly.
Figure 5. \( R_0 = 2.4546 \), we have 3 different waves of infections where the number of exposed increases for a certain length of time and reach a peak before a decrease and so on.

Figure 6. \( R_0 = 2.4546 \), we also have 3 different waves of infections and the number of symptomatic infected is always smaller than the number of asymptomatic infected.

Figure 7. \( R_0 = 2.4546 \), we also have 3 different waves where the number of recovered increases and decreases.
Figure 8. Here we can clearly see the interactions between the four classes when $R_0 = 2.4546$.

Figure 9. If $s_1 = 10$ and $s_2 = 55$ ($\beta = 0.3$ and $\eta = 1.65$), $R_0 = 1.0403$. We can see that even if the number of contacts for symptomatic infected has been strongly reduced, we still have lots of infections due to the asymptomatic infected contact number which is still high. We can also see it slows down the infection waves.

References

[1] Bing Li, Shengqiang Liu, Jing’an Cui and Jia Li: A Simple Predator-Prey Population Model with Rich Dynamics. Applied Science Journal, 16 May 2016.

[2] Alexei Pantchickine: Introduction aux systèmes dynamiques et à la modélisation. Institut Fourier, Université Grenoble-1, 2008.

[3] Xiao-Qiang Zhao: Fisher Waves In An Epidemic Model, Discrete And Continuous Dynamical Systems Series B Volume 4, Number 4 (Nov 2004), pp 1117-1128.

[4] Amin Boumerin and Nguyen Van Minh: Perron Theorem In The Monotone Iteration Method For Traveling Waves In Delayed Reaction-Diffusion Equation, Mathematics Subject Classification. 35K55, 35R10 (Feb 2008).

[5] Xiaojie Hou, Yi Li: Traveling Waves In A Three Species Competition-Cooperation System, math. AP (Jan 2013).

[6] W. Ding, W. Huang and S. Kansakar: Traveling wave solutions for a diffusive SIS epidemic model, PHD Thesis, Discrete Contin. Dyn. Syst. B, 18 (2013), 1291–1304.

[7] W. T. Li, G. Lin, C. Ma and F. Y. Yang, Traveling waves of a nonlocal delayed SIR epidemic model without outbreak threshold, Discrete Contin. Dyn. Syst. B, 19 (2014), 467–484.

[8] Dorothea Manika: Application of the Compound Matrix Theory for the computation of Lyapunov Exponents of autonomous Hamiltonian systems, M.Sc. Thesis, Thessaloniki University, September 2013.

[9] Xiaqing Ge, and Murat Arcak: A New Sufficient Condition for Ad-ditive D-Stability and Application to Cyclic Reaction-Diffusion Models, American Control Conference, 2009.
[10] James Muldowney: *Compound Matrices And Ordinary Differential Equations*, Rocky Mountain Journal of Mathematics, Volume 20, number 4, Fall 1990.

[11] M. Marcus and H. Minc: *A survey of Matrix Theory And Matrix Inequalities*, Allyn and Bacon, Boston, 1964.

[12] Liancheng Wang and Michael Y. Li: *Diffusion-Driven Instability in Reaction-Diffusion Systems*, Department of Mathematics and Computer Science, Georgia Southern University, November 1999.

[13] Karol Hajduk: *Turing Instability*, Faculty of Mathematics, Informatics and Mechanics, University of Warsaw, 2013.

[14] Kai Trepka: *Modifying Reaction Diffusion: A Numerical Model for Turing Morphogenesis, Ben-Jacob Patterns, and Cancer Growth*, Harvard University, Cambridge, MA, bioRxiv, 2010.

[15] Ansgar Jüngel: *Diffusive and non-diffusive population models*, Institute for Analysis and Scientific Computing, Vienna University of Technology, Wiedner Hauptstr. 8-10, 1040 Wien, 2010.

[16] R. Casten and C. Holland: *Stability properties of solutions to systems of reaction-diffusion equations*, SIAM J. Appl. Math. 33 (1977), 353-364.

[17] M. Y. Li, J. R. Graef, L. Wang, and J. Karsai: *Global dynamics of an SEIR model with varying total population size*, Math. Biosci. 160 (1999), 191-213.

[18] Zindoga Mukandavire, Prasenjit Das, Christina Chiyaka and Farai Nyabadza: *Global analysis of an HIV/AIDS epidemic model*, World Journal of Modelling and Simulation. Vol.6 (2010) No 3, pp. 231-240.

[19] W. Wang: *Backward bifurcation of an epidemic model with treatment*, Mathematical Biosciences, 201 (2006) 58-71.

[20] Qun Li, Prasenjit Das, Xuhua Guan, Peng Wu and Xiaoye Wang: *Early Transmission Dynamics in Wuhan, China, of Novel CORONAVIRUS-Infected Pneumonia*, The New England Journal of Medicine. DOI: 10.1056/NEJMoa2001316, January 29, 2020.

[21] G. Sallet: $\mathcal{R}_0$ EPICASA09, April 2010.

[22] Zhisheng Shuai and P. Van Den Driessche: *Global Stability of Infectious Disease Models Using Lyapunov Functions*, SIAM J. APPL. MATH. Vol. 73 No. 4, pp. 1513-1532, 2013 Society for Industrial and Applied Mathematics, April 2010.

[23] Anwar Zeb, Ebraheem Alzahrani, Vedot Suat Enturk and Gul Zaman: *Mathematical Model for Coronavirus Disease 2019 (COVID-19) Containing Isolation Class HIDAWI Article ID 345202, June 2020.*

[24] Faïcal Ndaïrou & Delfim F. M Torres: *Mathematical Model of COVID-19 Transmission Dynamics with a case Study of Wuhan ELSEVIER, Chaos, Soliton & Fractals volume 135, June 2020, 109846.*

[25] Mustapha Serhani & Hanane Libbardi: *Mathematical Model of COVID-19 Spreading with Asymptomatic Infected and Interacting peoples Journal of Applied Mathematics and Computing, August 2020.*

[26] D. Okuonghæ & A Oname: *Analysis of Mathematical Model for COVID-19 Population Dynamics in Lagos, Nigeria ELSEVIER, Chaos, Soliton & Fractals volume 135, October 2020 139: 110032.*

[27] Available online: https://www.worldometers.info/coronavirus (accessed on 1 May 2020).

[28] Ndondo Mboma Apollinaire, Walo Omana Rebecca, Maurice Yengo Vala-ki-sisa: *Optimal Control of a Model of Gambiense Sleeping Sickness in Humans and Cattle*, American Journal of Applied Mathematics 2016; 4 (5): 204-216.

[29] A. M. Ndondo, J. M. W. Munganga, J. N. Mwambakana, C. M. Saad-Roy, P. van den Driessche and R. O. Walo: *Analysis of a model of gambiense sleeping sickness in humans and cattle*, Journal of Biological Dynamics, 10: 1, 347-365, DOI: 10.1080/17513758.2016.1190873.