GLOBAL STABILITY ANALYSIS OF TRANSMISSION DYNAMICS IN A COMPARTMENTAL MODEL OF CORONAVIRUS DISEASE WITH ENVIRONMENTAL RESERVOIRS

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ABSTRACT

This work proposes and analyzes a non-linear deterministic model which describes the outbreak of Coronavirus disease with fomites (environmental reservoirs) propagating the spread of the infection in human population. Suitable Lyapunov functions are constructed to demonstrate the Global Asymptotic Stability (GAS) of both the Endemic Equilibrium (EE) and Disease Free Equilibrium (DFE) of the system. The EE was also found to be epidemiologically relevant when the basic reproduction number of new infections is greater than unity.

KEYWORDS: Coronavirus, Global stability, Lyapunov function, Fomites, Infectious disease

INTRODUCTION

In Wuhan, central China, a city of 11 million people, a new deadly respiratory infection outbreak was reported late December, 2019. The novel COVID-19 (as it was eventually named) has so far ravaged the world for nearly three years now and has shown no signs of completely disappearing anytime soon. In fact, recent reports from the World Health Organization (WHO) and several independent researches verify the emergence of new strains of the deadly infectious disease continuously spreading even further to more geographical locations on the planet (Hossain et al., 2021; WHO, 2022a). Casualty figures worldwide, currently totals 6,287,117 as at May 31, 2022 with around 526,558,033 confirmed infection cases and counting (WHO, 2022b). The Coronavirus disease is one of the many respiratory infections in the Severe Acute Respiratory Syndrome (SARS) family. COVID-19 also known as Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is only the third zoonotic transmitted human coronavirus in recent times (Zhou et al., 2020), preceded by SARS-CoV-1 and Middle East Respiratory Syndrome Coronavirus (MERS-CoV) outbreaks of the year 2002 and 2012 respectively (Drosten et al., 2003; Peiris et al., 2003; Zaki et al., 2012). Specifically, SARS-CoV-2 is the virus that causes the respiratory illness responsible for the so called COVID-19 pandemic today. A typical symptomatic infected individual can go from mere dry coughs, running high fevers to experiencing breathing difficulties, multiple organ failures and eventually death in a matter of days. Owing to the severity of the outbreak, the WHO declared it a public health emergency of international concern January 30, 2020 and subsequently a pandemic on March 11, barely two months later. There are a few conflicting theories on the possible origins of the virus, however its genetic similarity to bat-borne coronaviruses suggest that its origins may not be far from human-bat interactions of some sort. Being a highly contagious single-stranded RNA virus, human to human transmission occurs when around 200 to 800 infectious SARS-CoV-2 virions are passed on. Like most respiratory infections, coronavirus spreads rapidly via respiratory droplets from infected individuals either during voluntary acts such as speaking, singing or even during involuntary actions such as sneezing, coughing and so on. These droplets being naturally defined as aerosols vary in their mid-air suspension lifetime according to size. Efforts to curb the spread of the virus have seen multiple mitigation measures including community lockdown, social distancing, face masking, vaccination, quarantine of infected individuals and so on. Some of these measures have affected the global economy in diverse ways. However, containing the outbreak and easing the spread of the virus is a task that the human society as a whole must tackle head-on and urgently too. The transmission of infectious diseases is a complicated diffusion process occurring in susceptible populations. Numerous factors play key roles in the said diffusion, from population growth rate to seasonal migration as a result of natural or unnatural causes. Environmental reservoirs are one of such factors which this work is focused on. These include different types of surfaces that can hold droplets on contact with infected individuals which are then picked up by unsuspecting susceptibles on contact with same surfaces. Examples of environmental reservoirs for Coronavirus spread include door knobs, bus rails, table surfaces, shared clothing etc. Thus motivated by some of the classical techniques in Dénes and Röst (2016), Gao et al. (2018), and Korobeinikov (2006), we propose and analyze an eco-epidemiological compartmental model with the disease infection spread via direct contacts and environmental reservoirs. In a nutshell, this work attempts to describe the outbreak and spread of coronavirus disease by highlighting the role fomites in the transmission dynamics. Global asymptotic stability analysis of both the endemic equilibrium and disease free equilibrium of the system is then demonstrated using suitable Lyapunov functions. Epidemiologically relevant thresholds for the basic reproduction number of new infections are noted and its connection to fomites presented.

Model Formulation

Dynamical systems whether physical, biological or social may be expressed conveniently or modeled in the form of differential equations. These equations provide vital insight into the overall behaviour of a system if they are formulated with underlying factors and assumptions governing the system. Our model in this work, is based on SEIIFR system of \( N(t) \) size of individuals at time \( t \) where individuals are
classified into different compartments: Susceptible \((S(t))\), Exposed \((E(t))\), Infectious \((I(t))\), and Recovered \((R(t))\), while Fomites \((F(t))\), is the class used to capture the contributions to the spread of the virus due to environmental reservoirs. An individual becomes infected when in contact with the virus through infected persons or infected surfaces (fomites). The model \((1)\) below is a nonlinear system of equations describing Covid-19 outbreak with all parameters as described in the Table 1.

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - (aI + \beta E + \gamma F)S - \mu S \\
\frac{dE}{dt} &= (aI + \beta E + \gamma F)S - (\mu + \delta + \beta_e)E \\
\frac{dI}{dt} &= \beta_e E - (\mu + \delta + \sigma)I \\
\frac{dF}{dt} &= \phi_e E + \phi_I - \eta F \\
\frac{dR}{dt} &= \sigma I - \mu R
\end{align*}
\] (1)

subject to the initial conditions:
\(S(0) = S_0 \geq 0, E(0) = E_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0\) and \(F(0) = F_0 \geq 0\).

Table 1: Parameters and their biological/ecological meaning

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Biological/Ecological meaning</th>
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<tbody>
<tr>
<td>(\Lambda)</td>
<td>Per Capita inflow rate of humans</td>
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<tr>
<td>(a)</td>
<td>Rate of individuals upon contact with infectious person becomes infected with Covid-19</td>
</tr>
<tr>
<td>(\beta)</td>
<td>Rate of individuals upon contact with exposed person becomes infected with Covid-19</td>
</tr>
<tr>
<td>(\gamma)</td>
<td>Rate of individuals upon contact with Fomites becomes infected with Covid-19</td>
</tr>
<tr>
<td>(\beta_e)</td>
<td>Rate of exposed individuals becoming infectious</td>
</tr>
<tr>
<td>(\sigma)</td>
<td>Fraction of infectious individuals upon effective treatment who are successfully recovered</td>
</tr>
<tr>
<td>(\mu)</td>
<td>Natural death rate</td>
</tr>
<tr>
<td>(\delta)</td>
<td>Covid-19 induced death rate</td>
</tr>
<tr>
<td>(\eta)</td>
<td>Rate of decontamination of Coronavirus from the Fomites</td>
</tr>
<tr>
<td>(\phi_e)</td>
<td>The rate at which exposed individuals contribute Coronavirus to the Fomites</td>
</tr>
<tr>
<td>(\phi_I)</td>
<td>The rate at which infectious individuals contribute Coronavirus to the environment</td>
</tr>
</tbody>
</table>

It is trivial to show that \(S, E, I, R \geq 0\) and are all ultimately bounded by \(\frac{A}{\mu}\) and also, \(0 \leq F \leq \frac{A(\phi_e + \phi_I)}{\eta\mu}\), showing that the system of equations of the model \((1)\) has unique solution and hence well-posed on the set:
\[\Gamma := \{(S, E, I, F, R) \in \mathbb{R}_+^5; 0 \leq S \leq \frac{A}{\mu}, 0 \leq F \leq \frac{A(\phi_e + \phi_I)}{\eta\mu}, R_+ = [0, \infty)\}\]

Equilibrium Analysis

Disease Free Equilibrium

The disease-free equilibrium point, \(e^0\), is given by:
\[e^0 = (S^0, 0, 0, 0, 0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)\] (2)

Basic Reproduction Number

Consider the two vectors \(F_i = \left(\begin{array}{c} aI + \beta E + \gamma F \\ 0 \end{array}\right)\) and \(V_i = \left(\begin{array}{c} \eta(\mu + \delta + \sigma) \\ \eta\beta_e \end{array}\right)\) respectively, and thus, we have
\[\mathcal{R}_0 = \frac{1}{\eta(\mu + \delta + \sigma)(\mu + \delta + \beta_e)} \left(\begin{array}{cc} \eta(\mu + \delta + \sigma) & 0 \\ \eta\beta_e & \eta(\mu + \delta + \beta_e) \end{array}\right)\] (3)

where \(a = (\mu + \delta + \beta_e)(\mu + \delta + \sigma)\) and \(\varphi = \varphi_e(\mu + \delta + \sigma) + \beta_e\varphi_i\)

\[\mathcal{R}_0 = \frac{1}{\eta(\mu + \delta + \sigma)(\mu + \delta + \beta_e)} \left(\begin{array}{ccc} 0 & 0 & \theta \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{array}\right) \left(\begin{array}{c} \theta \\ \varphi_e \end{array}\right)\] (4)

where \(\theta = \beta\eta(\mu + \delta + \sigma)S^0 + \alpha\varphi_e(\mu + \delta + \sigma)\) and \(\varphi = \gamma(\varphi_e(\mu + \delta + \sigma) + \beta_e\varphi_i)\) and

\[\mathcal{R}_0 = \gamma(\mu + \delta + \beta_e)(\mu + \delta + \sigma)S^0\]

The characteristic equation \(\det(F - \lambda I) = 0\) implies
\[\lambda^2(\eta(\mu + \delta + \sigma)S^0 + \alpha\varphi_e(\mu + \delta + \sigma) + \lambda) = 0\] (5)

Therefore, the basic reproduction number \(\mathcal{R}_0\), is,
\[\mathcal{R}_0 = \frac{A(\beta\eta\gamma\varphi_e(\mu + \delta + \sigma) + (\alpha\eta + \gamma\varphi_i)\beta_e)}{\eta(\mu + \delta + \sigma)(\mu + \delta + \beta_e)}\] (6)

which may also be written as:
\[\mathcal{R}_0 = \frac{\beta\Lambda}{\mu(\mu + \delta + \beta_e)} + \frac{\alpha\Lambda\phi_e}{\mu(\mu + \delta + \sigma)(\mu + \delta + \beta_e)} + \frac{\gamma\Lambda(\varphi_e(\mu + \delta + \sigma) + \beta_e\varphi_i)}{\eta(\mu + \delta + \sigma)(\mu + \delta + \beta_e)}\] (7)

where the first two terms in \((7)\) reflect the contributions through the two transmission routes within humans (exposed to susceptibles and infected to susceptibles), while the last term captures the contributions via the fomites.
Existence of Endemic Equilibrium

The Endemic equilibrium point, \( e^* = (S^*, E^*, I^*, F^*, R^*) \) is obtained by setting the equations of the model to zero and solving them together to get:

\[
S^* = \frac{A}{\mu R_0}, \quad E^* = \frac{A}{(\mu + \delta + \beta_e)} \left( 1 - \frac{1}{R_0} \right),
\]

\[
I^* = \frac{\gamma_p R_0}{\mu (\mu + \delta + \beta_e)} \left( 1 - \frac{1}{R_0} \right),
\]

\[
R^* = \frac{\gamma_p (\mu + \delta + \beta_e)}{(\mu + \delta + \beta_e + \mu + \gamma_p)} \left( 1 - \frac{1}{R_0} \right)
\]

\[
F^* = \frac{1}{\eta} \left( \frac{\phi e}{(\mu + \gamma + \beta_e)} + \frac{\phi e}{(\mu + \gamma + \beta_e)} \right) \left( 1 - \frac{1}{R_0} \right)
\]

\( (8) \)

Obviously, the endemic equilibrium exists and is epidemiologically relevant when \( R_0 > 1 \). It also collapses to \( e^0 \) when \( R_0 = 1 \). Our attention will now be directed towards obtaining the conditions for global stability of the system equilibria. Several authors have analyzed and studied the global behaviour of epidemiological models (Dénes and Röst, 2016; Gao et al., 2018; Khan et al., 2015; Korobeinikov, 2006; Korobeinikov et al., 2015; Kryuchko and Blyuss, 2005). The approach for analyzing classical SIR epidemiological models is quite similar and applicable to SEIR, SEIS, SVEIR, and a number of other compartmental variants (Hethcote, 2000).

Proposition 1.0

If \( R_0 < 1 \), then the DFE of system (1) is globally asymptotically stable.

Proof. We will proceed by constructing a suitable Lyapunov function. We collapse the equations of the system to just three relevant classes namely \( S, E, \) and \( I \). It is verifiable that if the DFE for \( SEI \) is globally asymptotically stable, then with \( F(t) \) and \( R(t) \rightarrow 0 \), the DFE for \( SEI FR \) model is also globally asymptotically stable (Martcheva, 2015). Now, Let \( V \) be a Lyapunov function on \( \mathbb{R}^2 \), which clearly belongs to the positive orthant. Assuming it is given by:

\[
V = m(S - S^* - S^* \ln \frac{S}{S^*}) + \frac{1}{(\mu + \delta + \beta_e)} E + \frac{1}{\beta_e} I
\]

(9)

where \( m \) is a positive real number yet to be determined and \( S^* = \frac{A}{\mu} \). It is clear that \( V = 0 \) at the DFE. To see that \( V > 0 \) \( \forall (S, I, E) \) different from the DFE, it suffices to note that the first term of \( V \) which may be written as:

\[
m(S^* - \frac{S}{S^*} - 1 - \ln \frac{S}{S^*}) \]

for instance a function \( h(x) = x - 1 - \ln x \) has a global minimum at \( x = 1 \) i.e. \( h(1) = 0 \) and thus positive everywhere else for \( x > 0 \) and not 1. The last two terms of \( V \) are also clearly positive. Thus \( V \) is positive definite on the entire space and can be shown to be radially unbounded (i.e. \( V(x) \rightarrow 0 \) as \( \| x \| \rightarrow 0 \)). We now take its derivative w.r.t. \( t \).

\[
V' = m(-\frac{S^*}{S} S + \frac{1}{(\mu + \delta + \beta_e)} E + \frac{1}{\beta_e} I')
\]

\[
V' = m(\frac{1}{\mu - (aI + BE + \gamma F)S - \mu S} [\frac{1}{(\mu + \delta + \beta_e)} [(\alpha I + BE + \gamma F)S - (\mu + \delta + \beta_e)E] + \frac{1}{\beta_e}[\beta_e E - (\mu + \delta + \sigma) I])
\]

where \( m_1, m_2, m_3 \) are all positive real numbers to be determined. It is easy to see that \( L = 0 \) when \( (S, E, I) = \)


\[ L' = m_1 \left( -\frac{S^*}{S} + m_2 \frac{1}{1 - \frac{E^*}{E}} + m_3 \left( 1 - \frac{I^*}{I} \right) \right) \]

Substituting the equilibrium value for \( A \) from (1) into (15), we obtain

\[ L' = m_1 \left[ 1 - \frac{S^*}{S} \right] (aI^* + \beta E^* + \gamma F^*)S - (aI + \beta E + \gamma F)S - \mu S + m_2 \left[ (aI + \beta E + \gamma F)S - (aI + \beta E + \gamma F)S - \mu S \right] + m_3 \left( 1 - \frac{I^*}{I} \right) \beta E - (\mu + \delta + \sigma) I \]

(16)

next we combine +\( \mu S^* - \mu S \) with \( m_1 \left( 1 - \frac{S^*}{S} \right) \) which is the first term of the product in (16) and then we open up the brackets

\[ L' = -m_1 aI^* S^* + m_1 aI^* S^* + m_1 \beta E^* S^* - m_1 aIS \]

\[ -m_1 \beta ES - m_1 \gamma FS - m_2 \frac{\gamma F^2}{S} - m_1 \frac{\gamma F^2}{S} + m_1 aIS^* \]

\[ + m_1 \beta ES - m_1 \gamma FS + m_1 aIS + m_2 \beta ES - m_2 \gamma FS + m_2 \mu + \beta E)E \]

\[ -m_2 \mu + \beta E - m_2 \gamma FS + m_3 \beta E + m_2 \mu + \beta E)E + m_3 \beta E \]

\[ \cdot m_3 (\mu + \delta + \sigma) I - m_3 \beta E \frac{\gamma F}{S} + m_3 \mu + \delta + \sigma I \]

(17)

the last term in (18), \( m_3 (\mu + \delta + \sigma) I \) therefore becomes \( m_3 \mu + \delta + \beta E) \mu \) which then becomes \( m_2 (aI^* S^* + \beta E^* S^* + \gamma F^* S^*) \). Also the 15th term, \( m_2 (\mu + \delta + \beta E) \mu \) again becomes \( m_2 (aI^* S^* + \beta E^* S^* + \gamma F^* S^*) \). The 24th and 49th term may be collectively written as \( m_1 (aI^* S^* + \beta E^* S^* + \gamma F^* S^*) \).

The equation \( m_2 \beta E = m_2 \mu + \delta + \beta E \) is again deployed at the 18th term, thus \( -m_3 \beta E \frac{\gamma F}{S} \) becomes \( -m_2 (aI^* S^* + \beta E^* S^* + \gamma F^* S^*) \) \( \frac{\gamma F}{S} \). Bringing them all together and we have

\[ L' = -m_1 \mu \frac{1}{S} + m_1 aI^* S^* + m_2 (\mu + \delta + \beta E) \mu \]}

(19)

The last term on the RHS of (19), \( m_3 \beta E - m_2 (\mu + \delta + \beta E) \mu \) clearly vanishes from (19). The 3rd term may be further simplified as follows;

\[ m_1 aI^* S^* (1 + \frac{\beta E}{aI} + \frac{\gamma F}{aI}) - m_2 \frac{\mu + \delta + \beta E}{\beta E} (\mu + \delta + \sigma) I \]

we now set \( m_1 = m_2 \) so that \(-m_1 aI^* S^* - m_1 \beta E^* S^* - m_1 \gamma F S^* \) can cancel out with \(+m_2 aI^* S^* + m_2 \beta E^* S^* + m_2 \gamma F S^* \) then we multiply and divide certain fractions by equilibrium values

\[ L' = -\frac{m_1 (aI + \beta E + \gamma F)}{S} + m_1 aI^* S^* + m_1 \beta E^* S^* + m_1 \gamma F S^* \]

\[ -m_2 (\mu + \delta + \beta E) E - m_2 \frac{\gamma F}{S} \]

\[ + \frac{\gamma F}{S} \]

\[ - m_3 \beta E \frac{\gamma F}{S} + m_3 (\mu + \delta + \sigma) I \]

(18)

It is clear from the corresponding equilibrium equation in (1) that

\[ aI^* S^* + \beta E^* S^* + \gamma F^* S^* = (\mu + \delta + \beta E) \mu \]

also since \( m_1 = m_2 \), we then choose \( m_3 \) such that

\[ m_3 (\mu + \delta + \sigma) I = m_2 (\mu + \delta + \beta E) \mu \]

recall from the corresponding equilibrium equation in (1)

\[ \beta E \mu = m_2 (\mu + \delta + \beta E) \mu \]

\[ m_3 = \frac{(\mu + \delta + \beta E)}{\beta E} \]

Choosing \( m_2 = 1 \) we have that the 3rd term simplifies to

\[ S'(aI + \beta E + \gamma F) - \frac{(\mu + \delta + \beta E)(\mu + \delta + \sigma)}{\beta E} \]

Recall that we had already established from (7) that;

\[ \frac{\alpha \beta E}{S} = R_0 - \kappa \]

where

\[ \kappa = \frac{\beta A}{\mu + \delta + \beta E} + \frac{\gamma A \phi_2}{\mu + \delta + \beta E} \]

thus we have

\[ \frac{(\mu + \delta + \sigma)}{\mu + \delta + \beta E} = \frac{\alpha A}{\mu (R_0 - \kappa)} \]

obviously we obtain

\[ \xi = \frac{\alpha A}{\mu (R_0 - \kappa)} \]

which yields
\[
\frac{\xi \mu (R_0 - \kappa) - \alpha A I}{\mu (R_0 - \kappa)}
\]

which results in a negative value if \( R_0 < \frac{\alpha A I}{\xi \mu} + \kappa \).

The 1st term in the RHS of (19) is obviously negative except at \( S = S^* \). The 2nd term may be written as follows;

\[
m_1 \alpha S^* \left(1 + \frac{\beta E^* + \gamma F^*}{\alpha I^*} \right) \left[3 - \frac{S^*}{S} - \frac{E^* IS^*}{E^* IS^* - IE^*} \right]
\]

as \( E \to E^*, I \to I^*, \) and \( F \to F^* \). To see that the term \( 3 - \frac{S^*}{S} - \frac{E^* IS^*}{E^* IS^* - IE^*} \) is indeed negative, let \( a_1 = \frac{S^*}{S}, a_2 = \frac{E^* IS^*}{E^* IS^* - IE^*}, a_3 = \frac{IE^*}{IE^*}\)

observe that \( a_1 a_2 a_3 = 1 \)

by Lemma 1.0 we have that;

\[
\left( a_1 + a_2 + a_3 \right) \geq \sqrt[3]{a_1 a_2 a_3}
\]

\[
= \frac{S^*}{S} + \frac{E^* IS^*}{E^* IS^* - IE^*} + \frac{IE^*}{IE^*} \geq 3
\]

therefore the term \( 3 - \frac{S^*}{S} - \frac{E^* IS^*}{E^* IS^* - IE^*} \) is indeed negative, let \( a_1 = \frac{S^*}{S}, a_2 = \frac{E^* IS^*}{E^* IS^* - IE^*}, a_3 = \frac{IE^*}{IE^*}\)

Thus, the derivative of our Lyapunov function ultimately results in

\[ L' \leq 0. \]

A quick application of Krasovkii-LaSalle theorem accommodates \( L' \) being equal to zero, as it is quite easy to see that the set \( \Gamma = \{ u \in \mathbb{R}_+^2; L(u) = 0 \} \) consists of the singleton \( (S^*, E^*, I^*) \).

**Conclusion**

The global asymptotic stability of the endemic equilibrium was demonstrated via a suitable candidate Lyapunov function. The basic reproduction number \( R_0 \), was obtained for the model (1) and the DFE was shown to be globally stable when \( R_0 < 1 \). This implies the population would eventually be free of the disease, as it will die out in due course. It was also noted that above the threshold value of 1, the EE is globally asymptotically stable, indicating persistence of the disease in the population. Our result shows that environmental reservoirs play a significant role in said persistence. This claim is validated by the last term in the expression for \( R_0 \) in (7) which clearly highlights the contributions to new infections in the population due to fomites. Frequent application of disinfectants to all surfaces that could potentially spread the viral disease would be an effective control measure, as such action would continuously reduce the term responsible for the spread via fomites until it eventually vanishes from \( R_0 \). This ultimately shifts the \( R_0 \) value further left of the critical threshold, thus slowing down the spread of the disease in the population from an endemic towards a disease free status. The persistence of the disease has also seen the emergence and evolution of new strains of the virus. Intensified global efforts to contain the virus have necessitated the use of huge amounts of disinfectants and antibiotics with rapid biological and environmental impacts on surface waters, wastewater, soils and sediments (Chen et al., 2021), thus posing global health threats on another dimension.

**REFERENCES**


