Lyapunov Stability Analysis of Covid 19 SIR Modeling

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Abstract: COVID-19 is an infectious disease which is spreading as a global pandemy to the whole world. This paper exlplores nonlinear compartemental dynamic models which is used to model the spread of Covid-19. The Susceptible-Infected-Removed (SIR) model is one of the compartmental dynamic models which can be used to simulate what happens when someone in the community catches a disease, like Covid-19. The classical SIR model assumes that the properties of individuals can be divided into three distinct compartments: S(t) is the total of susceptible persons, I(t) is the total of infected persons and R(t) is the total of recovered person from infection and are now immune against the disease. The model also includes the dedicated effort of the government, the decision makers, and the stakeholders. Theoretically, interventions such as social distancing, mass testing, and isolation of positive cases should slow the rate of the infection spreads. The analysis of equilibrium indicates that the model has two equilibriums. One of them isthe disease free equilibrium and the other one is the endemic equilibrium. If the effort level less than minimum level, than the spread of the virus becomes endemic and if the effort level more than minimum level, than the spread of the virus can be controlled. The basic reproduction number $\Re_0$ is used to be an indicator of the expected number of individuals directly infected by an infectious person in a population where all individuals are susceptible to infection. If $\Re_0 < 1$, then the disease free equilibrium point is stable, meaning that the virus spread can be controlled. If $\Re_0 > 1$, then the disease free equilibrium point is unstable, meaning that the virus spread will continue. By constructing suitable Lyapunov function for SIR covid-19 model, the stability of the disease free equilibrium state of the model is thereby established.

Keywords: SIR model, infectious disease, basic reproduction number, Lyapunov function

1. Introduction

At December 2019, a severe spread of respiratory diseases detected in Wuhan, China. The virus is “SARS-CoV-2,” and the new disease has been called “corona virus disease 2019” (abbreviated “COVID-19”). The virus has further spread to other parts of China, and then it is continuing to transmit globally around the world. On January 31, 2020, there were 9,776 cases and 213 deaths, and then the WHO declared the spread as an international public health emergency of (WHO, 2020). On February 9, 2020, the global death toll has risen to 811, surpassing the total death cases of the 2003 SARS epidemic, and the respiratory disease continued to spread globally. The virus grow quickly and China athority initiated a mitgation effort. To reduce and decrease the spread acceleration of the virus, there are some ways such as lockdown, traffic restriction, physical distancing, enhancing personal hygiene, and a reduction in crowd in the general community. These ways have been adopted by China government and other goverentments in the world (Chen, et. al., 2020).

The concealed and apparently unpredictable nature of infectious dynamics of COVID-19 poses a considerable challenge to the disease control. First of unpredictable nature, the intermediate source of origin and transfer to humans is not known, although it is widely speculated that wild animals such as bats, civets, and minks are responsible for starting the epidemic (Yang and Wang, 2020; Zhou, et al., 2020). Second, incubation time (time from

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exposure to the development of symptoms) of COVID-19 ranges from 2 to 14 days. In this period, they are capable of transmitting the disease to other people (Rothe, et al., 2020).

The ability to predict the potential for infectious disease spread will provide a mechanism for governments and health-care services to respond and prevent the spread of infectious disease (LaPorte, 1993; Wilson, 1994). The epidemiological modeling of infectious disease is a tool that has been used to predict the future trend of infectious disease spread and to evaluate strategies to control an epidemic (Halasa and Durr, eds. 2018). However, to pick out a reliable predictable model/method is far from simple, a rational evaluation of various possible choices is eagerly needed, especially under the severe threat of COVID-19 pandemics now (Yang, et. al., 2020).

In recent days, a number of researchers have studied epidemiological SIR model to forecast the spread of the COVID-19 and to simulate the global trend of the pandemic. Nesteruk (Nesteruk, 2020) used the popular SIR (Susceptible-Infectious-Removed) model to predict the spread of the corona virus with the use of statistical approach and hence predicted the number of infected, susceptible, and removed persons versus time. The model approach by Nesteruk (Nesteruk, 2020) has become fundamental in modelling disease control as used by several authors (Ming and Zhang, 2020; Oduwole and Kimbir, 2018).

The SIR model has two main equilibria, disease-free equilibrium and endemic equilibrium. To determine whether or not equilibrium points of SIR epidemic model is stable, a Lyapunov function is often employed. Constructing properties of SIR epidemic model using Lyapunov function is generally a nontrivial problem (Safi and Garba, 2012). This is due to the fact that there is no systematic method to construct Lyapunov function to model infectious diseases with a standard incidence rate (Vargas-De-Leon, 2011). Specifically, a Lyapunov function for SIR epidemic model was developed by Mena-Lorca and Hethcote (Mena-Lorca and Hethcote, 1992). Korobeinikov and Wake (Korobeinikov and Wake, 2002) found a symmetric-in-variables Lyapunov function, which was later extended to a broader epidemic models, including models with a larger of compartments (Guo and Li, 2006; Korobeinikov, 2004a; Korobeinikov, 2004b; Okuonghae and Korobeinikov, 2007) and models with nonlinear functional responses (Georgescu and Hsieh, 2006; Korobeinikov, 2006; Korobeinikov, 2007; Korobeinikov, 2009a; Korobeinikov, 2009b; Korobeinikov and Maini, 2004; Korobeinikov and Maini, 2005; O'Regan, et. al., 2010).

A nonlinear system may have isolated equilibrium point or non-isolated equilibrium point (a continuum of equilibria). They may have more than one isolated equilibrium point or more than one continuum of equilibria. Since every neighborhood of a non-isolated equilibrium point contains another equilibrium, a non-isolated equilibrium cannot be asymptotically stable (Bervoets and Faure, 2019; Bhat and Bernstein, 2003).. Therefore, we can not use asymptotic stabilicy concept for analyzing such kind of continuum of equilibria But, it is still relevant the question of whether the dynamics of the system will converge to limited points and whether these points are Lyapunov stable or not. This problem makes it is important to use the concepts of convergence and semistability (Bhat and Bernstein, 2003).

The concept of convergence incdicates that the dynamics of the system converges to a limit point. This point is an equilibrium point. Further, this point will depend on the initial conditions, In a system which is convergent, the limit points of dynamics of the system are not necessarily Lyapunov stable. Therefore, semistability requirement is needed to guarantee that the dynamics of the system converge to limit points which are Lyapunov stable. We call an equilibrium point semistable if and only if it is Lyapunov stable, and every dynamics begining around the equilibrium point will converge to a Lyapunov stable equilibrium point (Bhat and Bernstein, 2003). It can be seen that, for an equilibrium point, asymptotic stability implies semistability, while semistability implies Lyapunov stability.

Definition 1 : An an equilibrium point $x_e = 0$ is said to be pointwise asymptotic stable (Goebel and Sanfelice, 2018) or semistable if it is Lyapunov stable, and every trajectory

starting in a neighborhood of the equilibrium point converges to a (possibly different) Lyapunov stable equilibrium point.

2. Mathematical Model

Mathematical analysis and modelling has been employed to study infectious diseases epidemiology (Brauer and Castillo-Chavez, 2001) in order to reveal the most promising and realistic strategies that influence the transmission and control of these diseases. The fundamental ideas in epidemiology, that is, the transmission dynamic of infectious disease depends on the rate of contact between susceptible and infected individuals (Anderson and May, 1991; Hethcote, 2000). In particular, modeling the spread of infectious disease, a basic choice is to use compartmental models. Population is divided into compartments, with the assumption that every individual in the same compartment has the same characteristics (Blackwood and Childs, 2018; Mateus, et. al., 2018; Rachah and Torres, 2018). The three most commonly used compartmental dynamics are the susceptible population of size S, which includes those who are healthy and can catch a disease, the infectious population of size I, which includes those who are infected and can also transmit the disease, and the recovered (or removed) population of size R, which includes those who are recovered from the disease and are immune to it. The model is known as SIR model (Kermack and McKendrick, 1927).

The basic SIR model formulated by Kermack and McKendrick can be described by the following differential equations:

$$\begin{bmatrix} \dot{S} \\ \dot{I} \\ \dot{R} \end{bmatrix} = \begin{bmatrix} -\beta SI/N \\ \beta SI/N - \gamma I \\ \gamma I \end{bmatrix} \tag{1}$$

where $S,\ I$ and R represent the density of the susceptible, infected, and recovered individuals, respectively, with the initial conditions $S(0) \geq 0,\ R(0) \geq 0,$ and $I(0) \geq 0.$ The term $\beta SI/N$ is a standard kinetic term, based on the idea that the number of new infected individuals per unit time corresponds to to the numbers of the infected and susceptible compartments. The total population N is large and constant, $S(t)+I(t)+R(t)=N.$ The recovered R can be formulated as $R=N-S-I.$ The coefficient $\beta>0,$ is the transmission coefficient, the effective contact rate that disease moves from infected individuals to susceptible individuals in the population. The coefficient $\gamma>0,$ is the recovery rate (or in other words, the average duration of infection $D=1/\gamma$). The classic SIR model assumes the individuals recover with immunity. Define $x_1=S/N,\ x_2=I/N,$ and $x_3=R/N,$ we can rewrite the SIR model into the form

$$\begin{bmatrix} \dot{x}_1 \\ \dot{x}_2 \\ \dot{x}_3 \end{bmatrix} = \begin{bmatrix} -\beta x_1 x_2 \\ \beta x_1 x_2 - \gamma x_2 \\ \gamma x_2 \end{bmatrix} \tag{2}$$

with $0 \leq x_1 \leq 1,\ 0 \leq x_2 \leq 1,\ 0 \leq x_3 \leq 1,$ and $x_1+x_2+x_3=1.$

Since the first two equations of Eqs. (2) do not depend on the third (the constant population size assumption), it is sufficient to consider the first two equations (Gui and Yong, 2017). Thus, we will focus our attention on the reduced model as shown in Eqs. (3).

$$\begin{bmatrix} \dot{x}_1 \\ \dot{x}_2 \end{bmatrix} = \begin{bmatrix} -\beta x_1 x_2 \\ \beta x_1 x_2 - \gamma x_2 \end{bmatrix} \tag{3}$$

and the closed set $\Gamma = \{(x_1, x_2) \in \Re_+^2 : x_1 + x_2 \le 1\}$.

The range of all states are between 0 and 1, this implies the existence of all the limits $x_1(\infty) = \lim_{t\to\infty} x_1(t)$, $x_3(\infty) = \lim_{t\to\infty} x_3(t)$, and thus $x_2(\infty) = \lim_{t\to\infty} x_2(t) = 1 - x_1(\infty) - x_3(\infty)$. Notice, it is easy to conclude that $x_2(\infty) = 0$ for all initial conditions, because if $0 < x_2 < 1$ then $x_3(\infty) = \infty$, this is a contradiction.

The basic reproduction number of the epidemic $\Re_0 = \dfrac{\beta}{\gamma}$, can be though of as the expected number of secondary infections generated by a single, typical infection in a population where all individuals are susceptible to infection (Fraser, et.al., 2009).
Equilibria are points where the states do not change with time, i.e.,

$$\begin{bmatrix} -\beta x_1 x_2 \\ \beta x_1 x_2 - \gamma x_2 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \end{bmatrix} \tag{4}$$

Observe that if $x_2 = 0$ then Eqs. (4) is satisfied. Therefore, there is a continuum of equilibria $(x_1, 0)$ with $x_2 = 0$ and arbitrary value of x_1 $(0 \le x_1 \le 1)$. The epidemiological interpretation requires the solution of Eqs. (4) with an initial value of $x_1 = 1$, that is, all individuals are free of disease (healthy). This state is an equilibrium point, $E_0 \Leftrightarrow [x_1 = 1, x_2 = 0]$, usually named as disease free equilibrium point. Another special equilibrium point is endemic equilibrium point, $E_+ \Leftrightarrow [x_1 = 0, x_2 = 0]$, such that $x_3 = 1$, since $x_1 + x_2 + x_3 = 1$. At this equilibrium point all individuals are removed or recovered. We conclude that the equilibria of the SIR system in Eqs, (2) are non-isolated.

Theorem 1 : The disease free equilibrium point, E_0, is unstable if $\Re_0 > 1$.

Proof : One formal method based of stability analysis uses the linearization of the SIR system to determine the local stability of the original system. To check the stability of disease free equilibrium point, $E_0 \Leftrightarrow [x_1 = 1, x_2 = 0]$, first, we will shift the states so that the equilibrium point is origin. Consider the change of variables

$$\begin{bmatrix} \xi_1 \\ \xi_2 \end{bmatrix} = \begin{bmatrix} x_1 - 1 \\ x_2 \end{bmatrix} \Leftrightarrow \begin{bmatrix} x_1 \\ x_2 \end{bmatrix} = \begin{bmatrix} \xi_1 + 1 \\ \xi_2 \end{bmatrix} \tag{5}$$

The new state space can be rewritten as

$$\begin{bmatrix} \dot{\xi}_1 \\ \dot{\xi}_2 \end{bmatrix} = \begin{bmatrix} -\beta(\xi_1 + 1)\xi_2 \\ \beta(\xi_1 + 1)\xi_2 - \gamma\xi_2 \end{bmatrix} = \begin{bmatrix} -\beta\xi_1\xi_2 - \beta\xi_2 \\ \beta\xi_1\xi_2 + \beta\xi_2 - \gamma\xi_2 \end{bmatrix} = \begin{bmatrix} f_1 \\ f_2 \end{bmatrix} \tag{6}$$

Linearization of the system (6) at the origin is represented as follow

$$\begin{bmatrix} \dot{\xi}_1 \\ \dot{\xi}_2 \end{bmatrix} = \begin{bmatrix} \left.\dfrac{\partial f_1}{\partial \xi_1}\right|_{\xi=0} & \left.\dfrac{\partial f_1}{\partial \xi_2}\right|_{\xi=0} \\ \left.\dfrac{\partial f_2}{\partial \xi_1}\right|_{\xi=0} & \left.\dfrac{\partial f_2}{\partial \xi_2}\right|_{\xi=0} \end{bmatrix} \begin{bmatrix} \xi_1 \\ \xi_2 \end{bmatrix} = \begin{bmatrix} 0 & -\beta \\ 0 & \beta - \gamma \end{bmatrix} \begin{bmatrix} \xi_1 \\ \xi_2 \end{bmatrix} \tag{7}$$

The eigenvalues of the system (7) are $\lambda_1 = 0$ and $\lambda_2 = \beta - \gamma$. Here the eigenvalue of interest is $\lambda_2 = \beta - \gamma = \gamma(\Re_0 - 1)$. The application of Lyapunov's linearization method indicates this equilibrium point is locally unstable if $\beta > \gamma$, or in other word the disease free equilibrium point, E_0, is unstable if $\Re_0 > 1$.

Theorem 2 : The continuum equilibria are two groups, when $\Re_0 > 1$.

Proof : Since all of states and all parameters are positive, $\dot{x}_1$ is always negative and $\dot{x}_3$ is always positive. The value of $\dot{x}_2 = \beta x_2(x_1 - \frac{\gamma}{\beta}) = \beta x_2(x_1 - \frac{1}{\Re_0})$. If $\Re_0 > 1$ then the stability of the SIR system depend on $x_1(0) = x_{10}$ (the initial value of x_1). If $x_{10} > \frac{1}{\Re_0}$ then $\dot{x}_2 > 0$ or the SIR system will be unstable until $x_1 = \frac{1}{\Re_0} \Rightarrow \dot{x}_2 = 0$. If $\dot{x}_1 = -\beta x_1 x_2$ is still negative ($x_1 > 0$ and $x_2 > 0$) then x_1 will become less than $\frac{1}{\Re_0}$ or $x_1 < \frac{\gamma}{\beta}$. If $x_1 < \frac{\gamma}{\beta}$ then $\dot{x}_2 < 0$, or the dynamical SIR system is stable. In other word, if $\Re_0 > 1$ then $x_2(t)$ will increase until maximum, and then will decrease such as $x_2(\infty) = 0$, and $x_3(t)$ will increase such that we have an epidemic. Figure 1 shows a graph of the SIR model with $\Re_0 > 1$.

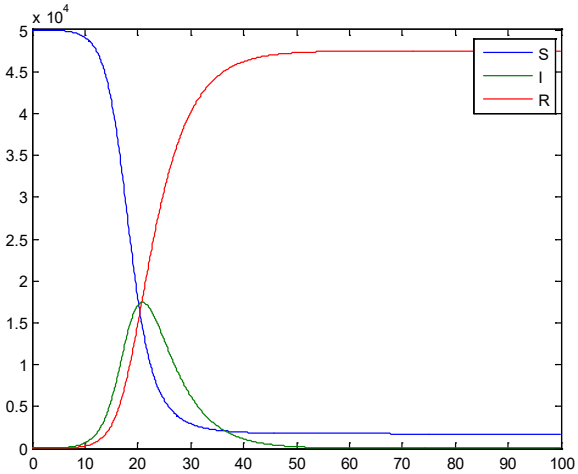

Figure 1. $\Re_0 = 3.5$; S(0) = 49995; I(0) = 5; R(0) = 0; $\beta = 0.7$; $\gamma = 0.2$;

We conclude that the continuum equilibria have two groups of stability, one group is unstable equilibria and another group is stable equilibria.

Although we cannot solve explicitly the SIR model (Weiss, 2013), we can find the maximum value of infected individuals ($x_{2\max}$). Let us evaluate

$$\frac{dx_1}{dx_2} = \frac{-\beta x_1 x_2}{\beta x_1 x_2 - \gamma x_2} = \frac{-\beta x_1}{\beta x_1 - \gamma} \tag{8}$$

This is can be solved by separating the variables and integrate

$$\int \frac{\beta x_1 - \gamma}{\beta x_1} dx_1 = -\int dx_2 \tag{9}$$

Hence, $-x_1 - x_2 + \frac{\gamma}{\beta}\log|x_1| = K.$ This means, for all $t \geq 0,$

$$x_1(t) + x_2(t) - \frac{\gamma}{\beta}\log|x_1(t)| = x_1(0) + x_2(0) - \frac{\gamma}{\beta}\log|x_1(0)|. \tag{10}$$

The value of $x_{2\max}$ will occur when $\dot{x}_2 = \beta x_1 x_2 - \gamma x_2 = 0,$ or in other word $x_1 = \gamma / \beta.$ To apply in Eqs. (10) yields

$$x_{2\max} = x_1(0) + x_2(0) - \frac{\gamma}{\beta}\log|x_1(0)| - \frac{\gamma}{\beta} + \frac{\gamma}{\beta}\log(\frac{\gamma}{\beta}). \tag{11}$$

It can be shown that for an initially fully susceptible population (Weiss, 2013), the maximum number of infected individuals is solely a function of $\Re_0,$

$$x_{2\max} = 1 - \frac{1}{\Re_0}(1 + \log \Re_0). \tag{12}$$

To describe the behavior of the SIR system, when $\Re_0 > 1,$ the phase plane of the system will be shown in Figure 2.

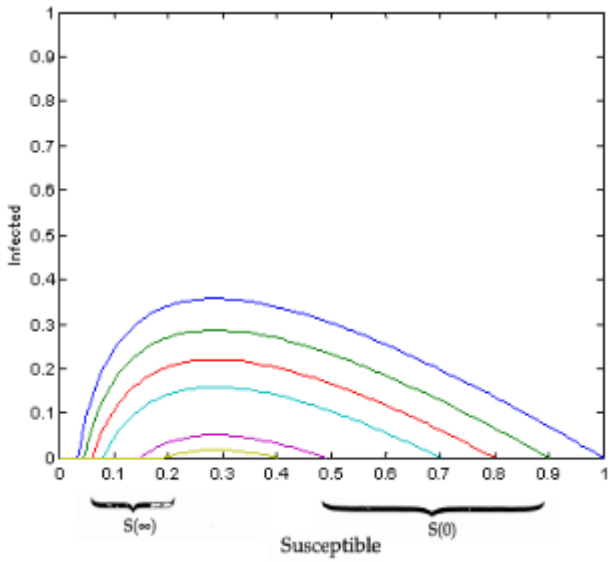

Figure 2. Phase plane of SIR with $\Re_0 = 3.5;\ \beta = 0.7;\ \gamma = 0.2;$

$S(0)$ denotes initial value of Susceptible population, while $S(\infty)$ denotes the value of Susceptible population after the epidemic event.

Therefore, we evaluate, that the direction of motion along the orbits and the limiting value of $x_1(\infty)$ of each orbit depends on the initial condition $x_1(0),$ but $x_1(\infty) > 0$ for all $x_1(0).$ Note that even in the general case of epidemic, not all individuals of the population become infected, a conclusions deducted from data for many epidemic cases (Brauer and Castillo-Chavez, 1994). The phase portrait shows that an orbit with initial condition $(x_{10}, 0)$ which is an unstable equilibrium point will go to a stable equilibrium point $(x_{1\infty}, 0).$

Theorem 3 : The disease free equilibrium point, $E_0,$ is semistable if $\Re_0 < 1.$

Proof : If $\Re_0 < 1$ the eigenvalues of the system (7) are $\lambda_1 = 0$ and $\lambda_2 = \beta - \gamma = \gamma(\Re_0 - 1) < 1,$ such that the dominant eigenvalue is 0, so we conclude that the linearization method is inconclusive. To prove the stability of $E_0,$ when $\Re_0 < 1,$ we will shift the states so that the equilibrium point is origin. Consider the change of variable $\xi_1 = x_1 - 1 \Leftrightarrow x_1 = \xi_1 + 1,$ such that the function (3) can be translated as shown in (13)

$$\begin{bmatrix} \dot{\xi}_1 \\ \dot{\xi}_2 \end{bmatrix} = \begin{bmatrix} -\beta(\xi_1 + 1)\xi_2 \\ \beta(\xi_1 + 1)\xi_2 - \gamma\xi_2 \end{bmatrix} \tag{13}$$

We consider candidate for the Lyapunov function:

$$V(\xi_1, \xi_2) = \frac{1}{2}(\xi_1 + 1 + \xi_2)^2 + \frac{1}{2}\xi_2^2 - \frac{1}{2} \tag{14}$$

The function in (14) is a positive definite. Evaluating the time derivative of $V(\xi_1, \xi_2)$ along Eqs. (13), which is applied to the disease free equilibrium point

$$\frac{dV}{dt} = (\xi_1 + 1 + \xi_2)\left(\frac{d\xi_1}{dt} + \frac{d\xi_2}{dt}\right) + \xi_2 \frac{d\xi_2}{dt} \tag{15}$$

$$\frac{dV}{dt} = (\xi_1 + 1)\left(\xi_2 - \frac{\gamma}{\beta}\right)\beta\xi_2 - \gamma\xi_2 - \gamma\xi_2^2 \tag{16}$$

Since $0 \le \xi_1 \le 1$ and $0 \le \xi_2 \le 1,$ the value of $\xi_2 - \frac{\gamma}{\beta} < 0$ when $\frac{1}{\Re_0} = \frac{\gamma}{\beta} > 1,$ such that $\frac{dV}{dt} < 0,$ for all $\xi_1 > 0$ and $\xi_2 > 0.$ It is shown that $\frac{dV}{dt}(\boldsymbol{\xi} = \mathbf{0}) = 0,$ this implies that $\frac{dV}{dt}(\boldsymbol{\xi})$ in (16) is a negative definite. Therefore, we may conclude that E_0 is stable when $\Re_0 < 1.$ But E_0 is a part of a continuum of equilibria, and therefore from definition 1, we can conclude that E_0 is a part of pointwise asymptotically stable equilibria or semistable equilibria, in other word, every dynamic starting around the equilibrium point will end to a stable equilibrium point which may be different.

3. Discussion

With a basic SIR models, we can forecast the dynamics of the COVID-19 pandemic and understand the effect of interventions on disease spread. To reduce the spread of disease before the presence of vaccine, it is very important to do physical distancing. The model illustrates the importance of social isolation (quarantine) for individuals who are infected. By staying in the quarantine room until they have fully recovered, it is very effective to reduce the number of individuals who are directly infected (Bloomfield, 2009). This protocol can decrease the size of an transmission by decreasing the probability of transmission of infectious disease to susceptible individuals. The effective physical distancing process in the SIR model is expressed by decreasing the value of β. The limit value, $\beta \to 0$, if there are antiviral drugs to treat the infectious disease and vaccines to prevent it.

To estimate the $\mathfrak{R}_0$ value, we use the SIR model based on the statistical virus spread data that is developed by Lisphilar (Lisphilar, 2020) and the COVID-19 SIR-based analytical model Python library, CovsirPhy (CovsirPhy Development Team, 2020). Analysis and estimation of R0 in this model are limited to the dataset until early December of 2020 only. To begin analyzing the dynamics of the R0 value regarding the COVID-19 spreading in Indonesia, we should take a look first at the SIR-based spreading pattern from time to time. The first case announced in Indonesia was around March 2020 in Jakarta city.

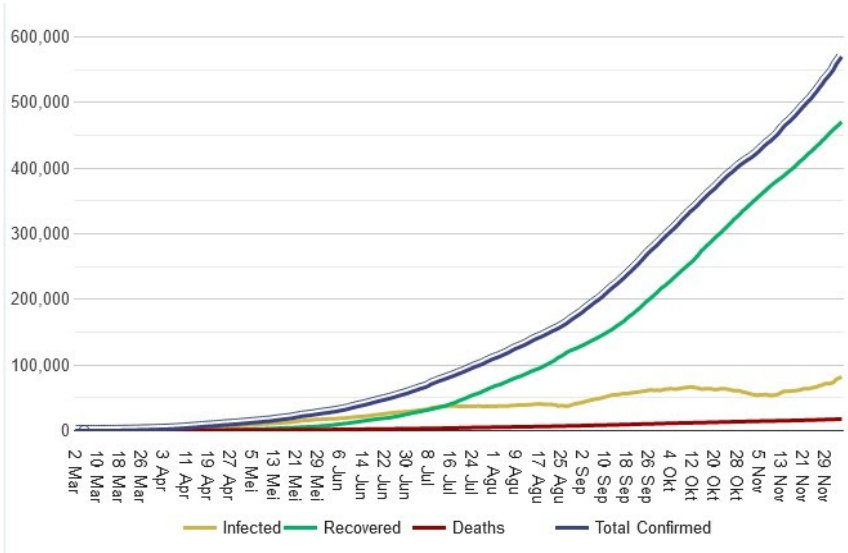

Figure 3. Total confirmed cases and S(t), I(t), R(t) cases overtime in Indonesia;

From the graph above, we can see that the COVID-19 cases in Indonesia have not shown a downward trend since the pandemic began. As of December 2020, Indonesia has confirmed about 569000 positive cases. The infected case seems to be exponentially increasing at some point around July, September, October, and late November. On the other hand, recovery cases also seem to be increasing in numbers from time to time. The increasing number for both of the confirmed and recovered cases are relatively at similar rates. Overall, at the moment, we can conclude that Indonesia is still in the first wave of the COVID-19 pandemic without any flattening trend so far.

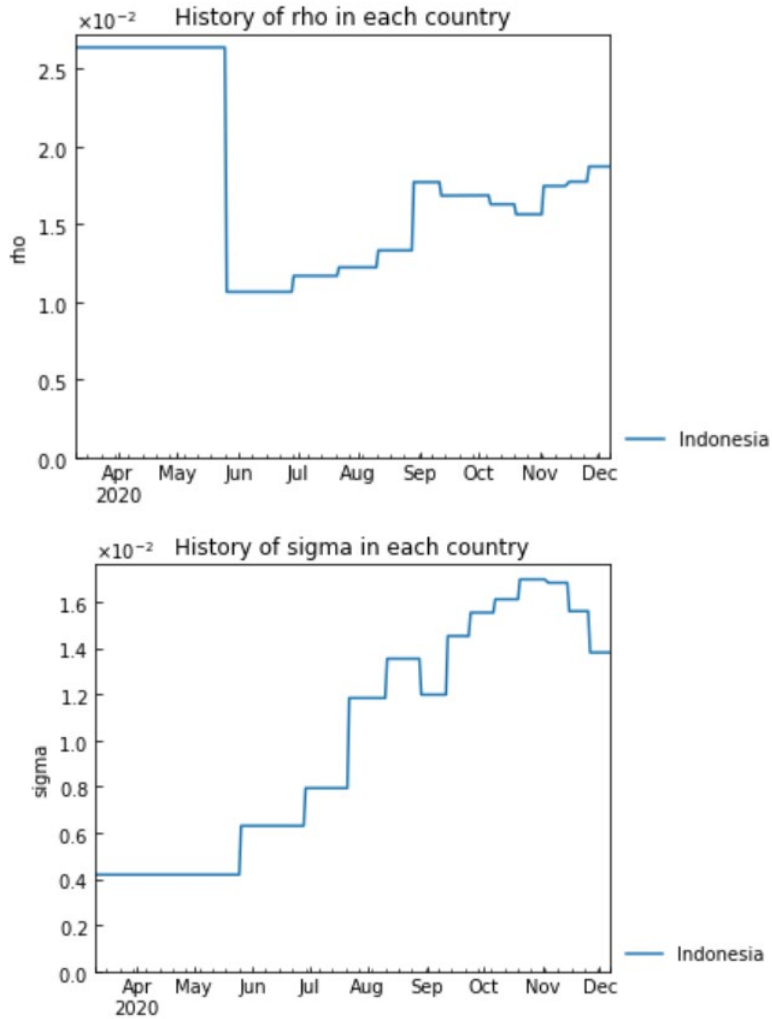

Figure 4. Contact rate (rho) and recovery rate (sigma) value overtime in Indonesia;

In line with the previous figure, where we could see the statistical graph of COVID-19 spread in Indonesia, the next figures represent the dynamics of contact and recovery rates from time to time since COVID-19 hit Indonesia. These two variables show how likely it is for subject analysis to move from the suspect compartment into infectious (contact rate) and from the infectious compartment into recovered (recovery rate) in the SIR model respectively.

As for the contact rate, we could see the spike increasing the contact rate in September 2020, which impacts the increasing numbers of new confirmed cases as seen in the first figure exponentially. As for the recovery rate, it seems that the recovery rate was gradually increasing from time to time, where we could see a significant increase in recoveries starting around August 2020. Since then, the recovery number keeps increasing along with the new confirmed cases. Referring to the graph of the progress of the COVID-19 case and the contact & recovery rate values above, we can estimate R0 from time to time, starting from the start of the pandemic.

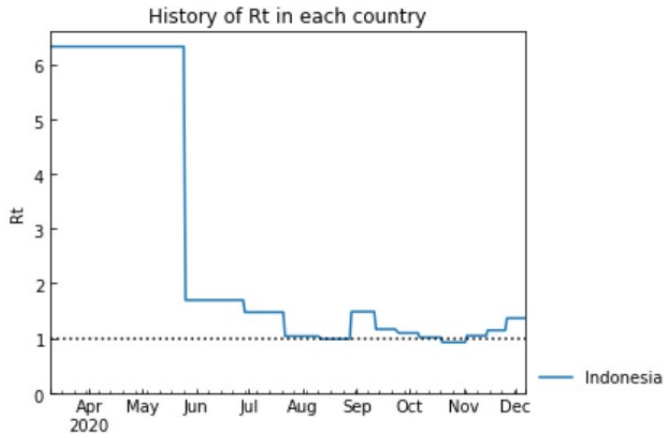

Figure 5. History of $\mathfrak{R}_0$ estimation value in Indonesia

From the graph above, we can see that the estimated value of $\mathfrak{R}_0$ has not shown a value below 1. The $\mathfrak{R}_0$ value shown in the graph until the beginning of December 2020 is at 1.36. The value of $\mathfrak{R}_0 > 1,$ also indicates that outbreaks can still occur, and the increase in COVID-19 cases is likely going to increase exponentially.

This analysis proves that the dynamics of COVID-19 in Indonesia have not reached the stability domain according to Lyapunov's function theory. From the past 9 months, Indonesia still hits an all-time-high in $\mathfrak{R}_0$ number each month. This phenomenon represents there are still so many policies and mitigation actions that the government has to make, especially in the effort to flatten the new confirmed cases, so that Indonesia's reproduction number could be shrunk down to below 1 and achieve the domain of stability. The way to decrease $\mathfrak{R}_0$ is to reduce the contact rate (beta) or increase the recovery rate (gamma). However, effective drugs and vaccines are still in development, so the only way Indonesia's government can do is reduce the contact rate by encouraging people to carry out the procedures and protocols for COVID-19 during the new normal by preventing crowd, maintaining distance, wearing masks all the time, washing hands, and continue to increase the number of COVID-19 test samples.

4. Conclusion

We discuss three theorems in this article. The three theorems explain the existence of infectious disease spread, the disease-free condition, and the endemic condition. The stability analysis of the disease-free equilibrium is undertaken by using Lyapunov function. If $\mathfrak{R}_0 < 1,$ the system has a group of continuum equilibrium points, such that the disease-free equilibrium point is stable, then the disease can be controlled means that the infectious disease will no longer spread. Meanwhile, if $\mathfrak{R}_0 > 1,$ the system has two groups of continuum equilibrium points, the disease-free equilibrium is unstable which means that the infectious disease will spread or the infected human will contaminate the disease to at least one individual in the population. Based on data-driven analysis in Indonesia, since the early of December 2020, $\mathfrak{R}_0$ is estimated to be about 1.36. This analysis proves that the dynamics of COVID-19 in Indonesia have not reached the stability domain according to Lyapunov's function theory. The way to decrease $\mathfrak{R}_0$ is to reduce the contact rate (beta) or increase the recovery rate (gamma). However, effective drugs and vaccines are still in development, so the only way Indonesia's government can do is reduce the contact rate by encouraging people to carry out the procedures and protocols for COVID-19 during the new normal by preventing crowd, maintaining distance, wearing masks all the time, washing hands, and continue to increase the number of COVID-19 test samples.

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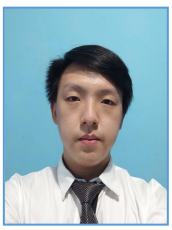

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