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Basic Analysis and Simulation of a SEIR Model for the Spread of Ebola Virus Disease

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Abstract
Despite its existence since 1976, Ebola virus is still present with the recent cases occurred in February 2021 and the largest outbreak was in March 2014, leading to the highest number of death cases compared to other viruses. This paper aims to model virus dynamics using an SEIR model in order to understand and analyse the Ebola outbreak transmission. The SEIR model consists of two equilibrium states which are disease-free equilibrium and endemic equilibrium. Routh-Hurwitz criterion is performed to study their stability criteria. The proposed epidemic model might be useful to estimate the severity of the Ebola virus by reviewing the dynamics of the virus in each compartment of the model (Susceptible, Exposed, Infected, and Recovered) as well as the value of the basic reproduction number, R_0 that is calculated using next-generation matrix method. Simulations of the proposed SEIR model were carried out including the observation when the certain parameter value is adjusted and then compared. In a nutshell, the R_0 obtained from the proposed parameter value is 2.52 indicating that the population is leading to an epidemic state since $R_0 > 1$. Thus, this result could be used to limit the spread of Ebola virus disease.
Keywords: Ebola virus disease; SEIR model; Stability analysis; Time series plots

1. Introduction

In 1976, in Sudan and Congo, the first outbreak of Ebola virus occurred in a community along the Ebola River located in Democratic Republic of the Congo [1]. Majority of people believe that the large bat branch is the initial cause of Ebola virus outbreak. People who came into contact with contaminated animal blood, excretions, organs, or other bodily fluids such as from fruit bats, monkeys and porcupines [2, 3] can be infected by Ebola virus. Health care workers are commonly infected with Ebola while treating suspected or confirmed Ebola patients.

The world's largest and most severe Ebola outbreak occurred in West Africa in March 2014. The death caused by Ebola virus is greater than the total number of deaths caused by other viruses. Furthermore, the virus went from country to country, beginning in Guinea and then spreading via the border to Sierra Leone and Liberia then by plane to travel to other countries [4].

During the coronavirus disease 2019 (COVID-19) pandemic, a new Ebola epidemic broke out in the Republic of Guinea in February 2021. The West African Ebola epidemic, which lasted from 2013 to 2016, was initiated in this region. Forest of Guinea was designated as a high-risk area for the virus. Conclusively, the focus of outbreak surveillance and contingency planning has shifted to prepare for the Ebola's reappearance in 2021 [5].

The latest instance of the Ebola virus disease, which has been around since 1976, was disclosed by the Democratic Republic of Congo's Ministry of Health in October 2021 [6]. Despite the fact that safety procedures have been made in the hopes of eliminating the virus's existence, new cases continue to emerge on a regular basis.

The use of a mathematical approach to the transmission of the Ebola virus disease is an important step in making predictions and setting expectations for the virus's future spread. The Susceptible-Exposed-Infected-Recovered (SEIR) model is a useful tool to understand disease propagation dynamics. Certain ways for controlling the disease can be proposed using this approach.

The scope of this study is to use differential equations to investigate the Ebola virus spreading. The Susceptible-Exposed-Infected-Recovered SEIR model, which is based on the compartmental dynamics of infectious diseases, is the core mathematical model that will be studied in this study. The model is solved using a set of ordinary differential equations (ODEs) and the disease's behaviours are investigated using stability analysis of the ODE system.

The basic reproduction number, R_0 is used to determine a potential of a disease transmission. It is the average number of secondary infections induced by a usual case of illness in a society where everyone is vulnerable. For two reasons, the basic reproduction number, R_0, must be established at the equilibrium point. First, the value of R_0 can be used to assess how quickly an illness spreads; if $R_0 > 1$, the infection has a chance of spreading to large populations, whereas if $R_0 < 1$, the infection has a chance of dying with a chance of 1. Second, knowing R_0 allows us to summarise the population's commitment to eradicate a virus. The stability of the system of ODEs is investigated and simulated using MATLAB.

This research aims to generate greater insight of the behaviour of the Ebola virus in order to develop preventive strategies to limit the disease spread. This model might be used to study the effects of the parameters on the population. Model analysis and simulation are performed to determine how the parameters function to stop the virus from spreading and reducing the chances of becoming infected with this infectious disease.

2. Literature Review

2.1. Ebola Virus Disease

Ebola viruses are members of the genus Ebolavirus in the Filoviridae family of the order Mononegavirales. Their genomes are made up of a single, negatively polarised strand of ribonucleic acid (RNA) [7]. The Ebola virus has been known to health experts since 1976, but because of its history of isolated little outbreaks, they were sceptical that it could inflict widespread harm [8]. Only three of the five species in the Ebola virus genus which is the Bundibugyo, Ebola, and Sudan viruses have been linked to the disease outbreaks in human [9]. In both humans and primates, the Ebola virus causes a severe, perhaps deadly systemic illness [10]. A condition brought on by these viruses is distinguished by broad viral multiplication, immunological shutdown, severe electrolyte and fluid depletion, unusual inflammatory reactions and a high fatality rate [11].

2.1.1. Transmission

There has not been an exact proof of where the virus origins, naturally comes from animal such as fruit bats, non-human primates or porcupines [1, 2]. Human to-human transmission of the Ebola virus occurs when infected bodily fluids, such as breast milk or vomit, come into contact with broken skin or mucous membranes, as well as infected blood from an infected individual, whether living or dead [12].

Unlike the COVID-19, Ebola virus is not an airborne disease [13]. In order for the virus to spread, direct contacts are needed and that is the reason why health care workers are more prone to get infected since they were exposed to the infected patients and infectious materials while taking care of the patient [14]. During the outbreak, it was reported that most cases of infections were among close relatives which accumulated for about 70% of the transmission. Even after recovering, individuals that were infected are remain infectious since there is a possibility of the virus to remain in the body system for a few months after infection.

2.1.2. Symptoms

An individual is believed to not have the ability to spread the disease until they had shown symptoms which are developed from 2 to 21 days after infection occurred. The most common symptoms of being infected with the virus include fatigue, fever, headache, muscle pain and sore throat. This is also known as the mild stage which occurs in about three days from infection [13]. On the second stage, worse complication cases include diarrhoea, loss of appetite, abdominal pain, rash and vomiting can occur. Lastly, on the worst case, usually among individuals with an existing fatal disease, set between 7th to

12th days of infection, further complications such as organ failure, internal and external bleeding are to be expected [11].

2.1.3. Diagnosis

The early symptoms of Ebola virus disease are quite similar to the common diseases like dengue fever, malaria and meningitis. Due to that, this virus can be difficult to be recognised and several diagnostic methods are made to confirm the symptoms. The most common method for identifying the virus is polymerase chain reaction (PCR) which may detect low levels of the virus. However, when a person begins to recover, or when there was not enough virus in the blood, the approach becomes ineffective [14]. In addition, according to the World Health Organization (WHO), reverse transcriptase-polymerase chain reaction assay (RT-PCR), antibodycapture enzyme-linked immunosorbent assay (ELISA), serum neutralisation test and virus isolation by cell culture are also applicable. While currently, automated or semi-automated nucleic acid tests (NAT), rapid antigen detection tests and specimen diagnosis like blood in ethylenediaminetetraacetic acid (EDTA) are being used [11].

2.1.4. Treatments and Vaccines

As of now, there is no proven treatment other than providing care to those who have been infected, maintaining hydration with the aid of antibiotics and providing oxygen support while combating the virus in the body. Other drugs that help with blood clotting and decreasing temperature due to fever are also included [11]. Vaccines, on the other hand, have been introduced over the world to reduce the chances of infection. From the late 1970s, a few experimental vaccines had started to developed. However, in 2020, World Health Organization (WHO) approved the Ervebo vaccine for everyone aged 18 and older, excluding pregnant and breastfeeding women [15]. Zabdeno and Mvabea vaccines in a two-dose combination have also been approved and are in use [16].

Evidence suggested that recovered individuals from Ebola generate antibodies that last for at least ten years or more. It is unclear whether recovered individuals are protected permanently or susceptible to be infected again with a different Ebola strain. Long-term consequences, including as joint and visual difficulties have been reported in some survivors [17].

2.1.5. Prevention and Control

Effective epidemic control requires case management, surveillance and contact tracing as well as a strong laboratory service, safe burials and societal mobilisation. Community participation is critical to successfully control the epidemics. Raising public awareness of Ebola infection risk factors and protective actions that individuals can take, such as immunisation is an effective method to prevent spreading from other human [18].

Few restrictions can be made to reduce the infections from happening. For example, reducing the consumption of raw meats from the animals that have the possibility to carry the virus such as monkeys, bats and antelope. Proper ways of handling food such as thoroughly cooking the meats before consuming it [19]. Another method is to control the outbreak by distancing the healthy from the sick to avoid further transmission, as well as emphasising the significance of good hygiene and keeping the environment clean. Also, identifying individuals who may have come into touch with an Ebola patient and keeping track of their health for 21 days [20].

2.2. Mathematical Modelling Approach on Ebola Virus Disease

Mathematical models have become more important for public health experts to understand the repercussions of disease transmission in large metropolis areas. They can help to quantify the socio-economic effects of significant epidemics and forecast the effects of alternative intervention techniques. Mathematical modelling also entails evaluating the hard effects of initiatives, bringing together knowledge from several fields of study and investigating how health-related patterns emerge in dynamic structures [21].

2.2.1. Linear Regression

Regression models are used to evaluate and forecast the targeted variable using dependent variables. As a result, regression is considered to exhibit the number of confirmed instances over time and forecasting future development. However, linear regression could not be used for a long term estimation. Koch et al. compared the immune memory induced by natural infection and vaccination of Ebola virus disease. The study discovered that antibodies alone may not be enough to assess vaccination-induced antibody immunity [22]. A cohort study by Huttner et al. [23] observed the antibody perseverance after a single dose of vaccination where it is deemed to be important especially in countries where booster vaccinations are not practical.

2.2.2. Auto Regressive Integrated Moving Average (ARIMA) Model

The ARIMA model is a time series analysis model that is widely used. The model starts with time series data where the former data serves as the independent variable and the subsequent data as the dependent variable. From the case of Ebola virus disease during the year 2014 to 2016, this method was utilised by to make decisions and assess the accuracy of dying patient forecasting in the Republic of Liberia [24]. Peak *et al*. discussed the reduction of population mobility as a result of travel restrictions using mobile data [25].

2.2.3. Susceptible-Infected-Recovered (SIR) Model

SIR model is usually used to describe the behaviour of an infection. Mubayi *et al.* applied SIR model for analytical prediction of the transmission rate in terms of prevalence in which gave a good insight on the problem dynamics [26]. A study by Hayman *et al.* (2022) examined the unlikeliness of Ebola virus persistence in non-human primate using an SIR model [27].

2.2.4. Susceptible-Exposed-Infected-Recovered (SEIR) Model

A study in 2018 applied SEIR model to simulate the transmission of the Ebola virus Ebola virus with the effects of demographic and the factor of vaccination in the system [28]. Whilst, in 2021, Obeng-Kusi *et al*. published a study that focused at the dynamics of Ebola virus disease spread while taking vaccination factor into consideration and comparing it in different income countries [29].

3. Methodology

3.1. Model Formulation

The SEIR model for Ebola virus disease transmission is divided up into four compartments: Susceptible (S), Exposed (E), Infected (I), and Recovered (R). A set of differential equations can be used to represent the Ebola virus disease model such as

$$\frac{dS}{dt} = \delta - \beta SI - \delta S, \tag{1}$$

$$\frac{dE}{dt} = \beta SI - \mu E - \delta E, \tag{2}$$

$$\frac{dI}{dt} = \mu E - \gamma I - \delta I, \tag{3}$$

$$\frac{dR}{dt} = \gamma I - \delta R, \tag{4}$$

where β is transmission rate, μ is infection rate, γ is recovery rate and δ is birth or death rate. $S + E + I + R = N$ is assumed where N is the population constant [30].

3.2. Equilibrium Points

Setting equation (1) to equation (4) to 0 yields the equilibrium points of the system as follows

$$\frac{dS}{dt} = \delta - \beta SI - \delta S = 0, \tag{5}$$

$$\frac{dE}{dt} = \beta SI - \mu E - \delta E = 0, \tag{6}$$

$$\frac{dI}{dt} = \mu E - \gamma I - \delta I = 0, \tag{7}$$

$$\frac{dR}{dt} = \gamma I - \delta R = 0, \tag{8}$$

As a result, the system's disease-free equilibrium point (DFEP) is determined as $(S, E, I, R) = (1,0,0,0)$ and the endemic equilibrium point (EEP) is $(S^*, E^*, I^*, R^*) = \left(\frac{(\mu+\delta)(\gamma+\delta)}{\beta\mu}, \delta\left(\frac{1}{\mu+\delta} - \frac{\gamma+\delta}{\beta\mu}\right), \frac{\mu\delta}{(\mu+\delta)(\gamma+\delta)} - \frac{\delta}{\beta}, \frac{\mu\gamma}{(\mu+\delta)(\gamma+\delta)} - \frac{\gamma}{\beta}\right)$.

3.3. Stability Analysis

3.3.1. Endemic Equilibrium

To find the stability by Routh-Hurwitz criterion, consider the associated characteristic polynomial by solving the determinant of the Jacobian matrix with the formula $|J - \lambda I|$ given by

$$\begin{vmatrix} -\frac{\delta(\beta\mu - \mu\delta - \mu\gamma - \delta^2 - \delta\gamma)}{\mu\delta + \mu\gamma + \delta^2 + \delta\gamma} - \delta - \lambda & 0 & -\frac{\mu\delta + \mu\gamma + \delta^2 + \delta\gamma}{\mu} & 0 \\ \frac{\delta(\beta\mu - \mu\delta - \mu\gamma - \delta^2 - \delta\gamma)}{\mu\delta + \mu\gamma + \delta^2 + \delta\gamma} & -\mu - \delta - \lambda & \frac{\mu\delta + \mu\gamma + \delta^2 + \delta\gamma}{\mu} & 0 \\ 0 & \mu & -\gamma - \delta - \lambda & 0 \\ 0 & 0 & \gamma & -\delta - \lambda \end{vmatrix} = 0.$$

Therefore, the characteristic polynomial obtained is

$$f(\lambda) = A_0\lambda^4 + cA_1\lambda^3 + cA_2\lambda^2 + cA_3\lambda + cA_4 = 0, \tag{9}$$

where

$$c = \frac{1}{\mu\delta + \mu\gamma + \delta^2 + \delta\gamma},$$

$$A_0 = 1,$$

$$A_1 = \beta\mu\delta + \mu^2\delta + \mu^2\gamma + 4\mu\delta^2 + 5\gamma\delta\mu + \mu\gamma^2 + 3\delta^3 + 4\delta^2\gamma + \delta\gamma^2,$$

$$A_2 = \beta\mu^2\delta + 3\beta\mu\delta^2 + \beta\gamma\delta\mu + \mu^2\delta^2 + \mu^2\delta\gamma + 3\mu\delta^3 + 4\mu\delta^2\gamma + \mu\delta\gamma^2 + 2\delta^4 + 3\delta^3\gamma + \delta^2\gamma^2,$$

$$A_3 = 2\beta\mu^2\delta^2 + \beta\mu^2\delta\gamma + 3\beta\mu\delta^3 + 2\beta\gamma\delta^2\mu - \mu^2\delta^3 - 2\mu^2\delta^2\gamma - \gamma^2\delta\mu^2 - 2\mu\gamma^4 - 4\gamma\delta^3\mu - 2\mu\delta^2\gamma^2 - \delta^5 - 2\delta^4\gamma - \delta^3\gamma^2,$$

$$A_4 = \beta\mu^2\delta^3 + \beta\mu^2\delta^2\gamma + \beta\mu\delta^4 + \beta\gamma\delta^3\mu - \mu^2\delta^4 - 2\mu^2\delta^3\gamma - \gamma^2\delta^2\mu^2 - 2\mu\delta^5 - 4\gamma\delta^4\mu - 2\mu\delta^3\gamma^2 - \delta^6 - 2\delta^5\gamma - \delta^4\gamma^2.$$

By Routh-Hurwitz criterion, it is important to prove

$$A_i > 0 \ (i = 0,1, \dots, 4), \tag{10}$$

$$A_1A_2 - A_0A_3 > 0, \tag{11}$$

and

$$A_1A_2A_3 - A_1^2A_4 - A_0A_3^2 > 0, \tag{12}$$

to see if the eigenvalues have negative real parts and therefore stable. Seeing as proving the conditions in equation (10) to equation (12) is too complicated, the proving is done numerically in Section 4.

3.3.2. Disease-free Equilibrium

In order to test the stability using the Routh-Hurwitz criterion, the associated characteristic polynomial have be obtained by using the formula to solve the determinant of the Jacobian matrix in order to test. $|J - \lambda I|$ given by

$$\begin{vmatrix} -\delta - \lambda & 0 & -\beta & 0 \\ 0 & -(\mu + \delta) - \lambda & \beta & 0 \\ 0 & \mu & -(\gamma + \delta) - \lambda & 0 \\ 0 & 0 & \gamma & -\delta - \lambda \end{vmatrix} = 0.$$

Therefore, the characteristic polynomial obtained is

$$f(\lambda) = B_0\lambda^4 + B_1\lambda^3 + B_2\lambda^2 + B_3\lambda + B_4 = 0, \quad (13)$$

where

$$B_0 = 1,$$
$$B_1 = \gamma + 4\delta + \mu,$$
$$B_2 = 3\mu\delta + \gamma\mu + 5\delta^2 + 3\delta\gamma - \beta\mu,$$
$$B_3 = 3\mu\delta^2 + 2\gamma\delta\mu + 4\delta^3 + 3\delta^2\gamma - 2\beta\mu\delta,$$
$$B_4 = \mu\delta^3 + \mu\delta^2\gamma + \delta^4 + \delta^3\gamma - \beta\mu\delta^2.$$

By Routh-Hurwitz criterion, it is important to prove

$$B_i > 0 \ (i = 0,1, \dots ,4), \quad (14)$$
$$B_1B_2 - B_0B_3 > 0, \quad (15)$$

and

$$B_1B_2B_3 - B_1^2B_4 - B_0B_3^2 > 0, \quad (16)$$

to see if the eigenvalues have negative real parts and therefore stable. Seeing as proving the conditions in equation (14) to equation (16) is too complicated, the proving is done numerically in Section 4.

3.4. Basic Reproduction Number, R_0

The basic reproduction number influences the duration of the infective phase, the likelihood of infecting a susceptible individual during a single interaction, and the amount of newly susceptible persons contacted per unit of time.

R_0 is calculated using the next-generation matrix method. Only individuals who are capable of spreading infections should be concerned when using the next-generation matrix method. As a result, only E and I compartments are present in the system as follows

$$\frac{dE}{dt} = \beta SI - \mu E - \delta E,$$
$$\frac{dI}{dt} = \mu E - \gamma I - \delta I.$$

The matrices $\boldsymbol{F}$ and $\boldsymbol{V}$ at disease-free equilibrium can be represented as

$$\boldsymbol{F} = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix}$$

and

$$\boldsymbol{V} = \begin{bmatrix} \mu + \delta & 0 \\ -\mu & \gamma + \delta \end{bmatrix}.$$

The next-generation matrix is given by $\boldsymbol{FV^{-1}}$, where

$$\boldsymbol{V^{-1}} = \begin{bmatrix} \frac{1}{\mu + \delta} & 0 \\ \frac{\mu}{(\mu + \delta)(\gamma + \delta)} & \frac{1}{\gamma + \delta} \end{bmatrix},$$

giving

$$\boldsymbol{FV^{-1}} = \begin{bmatrix} \frac{\beta\mu}{(\mu + \delta)(\gamma + \delta)} & \frac{\beta}{(\gamma + \kappa)} \\ 0 & 0 \end{bmatrix}.$$

Therefore the R_0 obtained is

$$R_0 = \frac{\beta\mu}{(\mu+\delta)(\gamma+\delta)}.$$

4. Results and discussion

Numerical simulations of the Ebola virus disease in Sierra Leone can be carried out using actual parameter values. The initial conditions are $S(0) = 600000,\ E(0) = 88,\ I(0) = 30,\ R(0) = 1$, and the remaining parameter values are as shown in Table 1. The simulations are evaluated over a period of 100 days. The stability analysis is performed using the Routh-Hurwitz criterion by substituting the parameter values from Table 1 into the conditions as referred to in equation (10) to equation (12) and equation (14) to equation (16). As a result, at DFEP, the system is unstable, while at EEP, it is stable.

Table 1: Parameter value of SEIR model

Parameter	Definition	Estimated value	References
β	Transmission rate	0.45	[30]
μ	Infectious rate	0.08	[30]
γ	Recovery rate	0.1751	[30]
δ	Birth/ Death rate	0.001	[30]

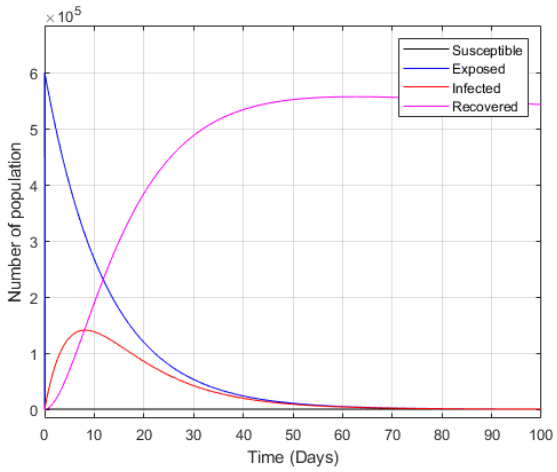

Figure 1 Simulation of the SEIR model for β = 0.45, μ = 0.08, γ = 0.1751, and δ = 0.001

Figure 1 displays the whole dynamic of the spread of Ebola virus disease with the proposed parameter values from a real-life data in Sierra Leone. The black curve represents the susceptible populations that instantly went from 600,000 to zero in less than a day. The exposed curve in blue peaked at 600,000 population and decreases over time until it reaches zero population. Next, the red curve describes the dynamic of infected populations that peaked around day eight and decreases to zero population over time. Lastly, the pink curve representing the recovered populations is the only curve that increases over 100 days. The basic reproduction number obtained from the proposed value is 2.52, indicating that virus spreading is approaching epidemic state and that the country's efforts to fight virus spread must be increased.

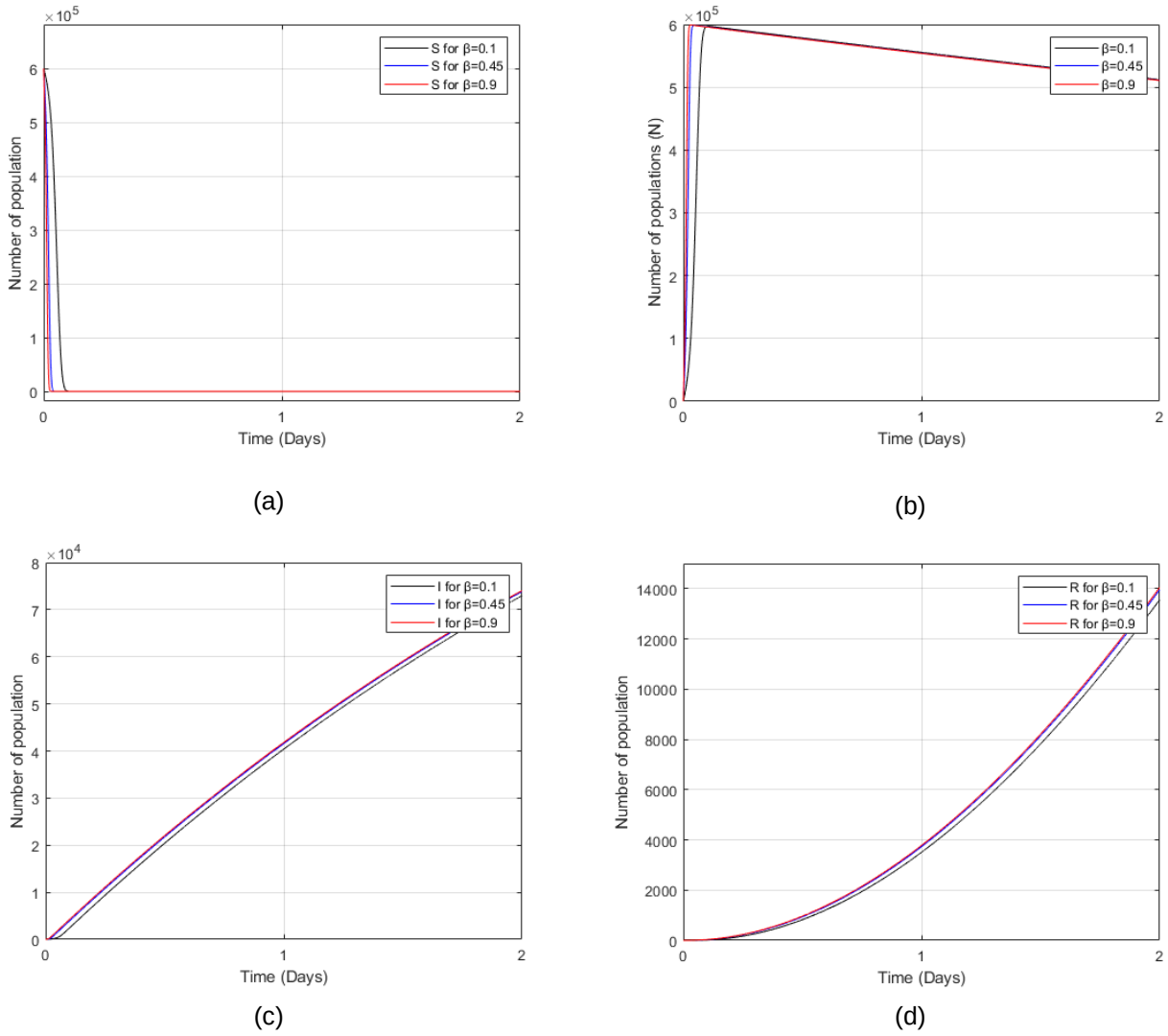

Figure 2 SEIR model with changes in β value where a) Susceptible, b) Exposed, c) Infected and d) Recovered

Figure 2 illustrates the comparison of the dynamics of the virus spreading in different values of transmission rate, β which is 0.1, 0.45 and 0.9. The graphs are separated in four compartments for a better insight of the spreading. However, the time period is minimised to see a clearer differences among the curves with different transmission rate values. It can be seen that in the susceptible graph, the curve with highest value of β converges to zero population faster than the others with lower values. Meanwhile in the exposed graph, the least value of β also is the fastest curve to converge to a value, however in this case, not reaching zero population but a decreasing number of population through time. Next, infected and recovered graphs both had quite similar characteristics where the position of the red curve is at the most top, blue curve in the middle and black curve on the bottom. However the difference between the two graphs is the difference of the number of population where the recovered graph had smaller range of population number in a period of two days. Generally for graphs c) and d), it can be deduced that the higher value of β, the more number of population obtained over time. The basic reproduction number, R_0 obtained for the β value of 0.1, 0.45 and 0.9 are 0.52, 2.52 and 5.05 respectively which can be deduced that only when the value of β is 0.1 that the disease will die out.

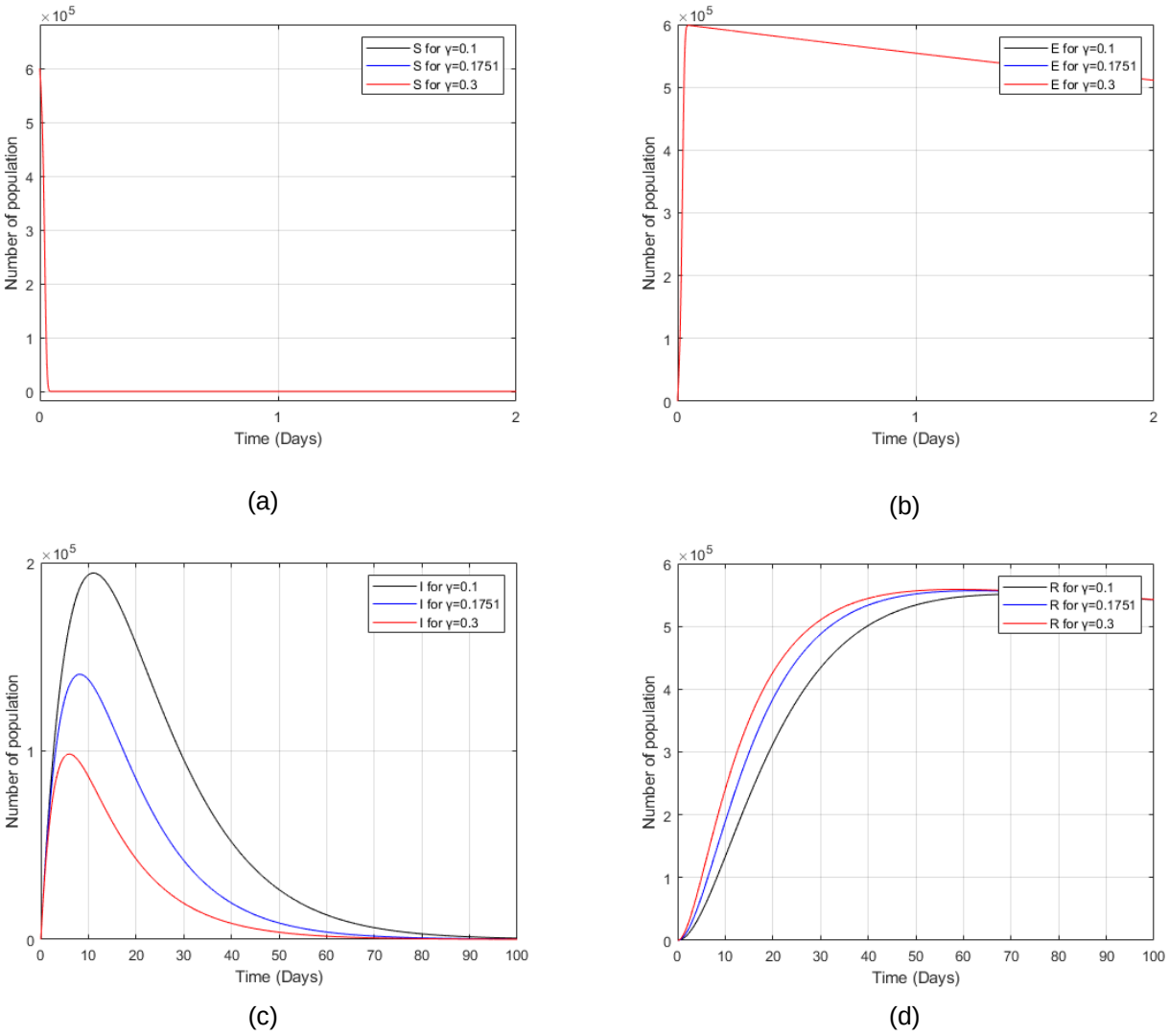

Figure 3 SEIR model with changes in γ value where a) Susceptible, b) Exposed, c) Infected and d) Recovered

Figure 3 describes the changes of value of recovery rate, γ affecting the number population over time. The value of infection rate used are 0.1, 0.1751 and 0.3. Graphs a) and b) were minimized into a range of two days since the changes of the curves are not significant. However, even with a smaller range of time, there is still no visible changes and the curves are overlapping with each other. For the infected graph, it can be clearly seen that the curve with the lowest peak at almost 100,000 population possessed the highest value of γ and the black curve with the lowest γ value had the highest peak among the curves. In contrary, the curves in recovered graph with the highest value of γ is the curve with the highest peak before converging to a value as the other curves. The R_0 obtained for the γ value of 0.1, 0.1751 and 0.3 are 4.40, 2.52 and 1.47 respectively. The suggested change of γ value does not lead to the disease dying out. However, the bigger the value of γ is, the smaller R_0 becomes.

Conclusion

The SEIR model that is used in this paper was a simple model which includes the demographic factors into consideration and not considering the effect of number of population into the model. Routh-Hurwitz criterion was used to examine the stability of the system and the time series plots for the numerical simulation were run by the external software which is MATLAB. As observed from the behaviour of the graph, the higher the transmission rate, the higher value of R_0 obtained which indicates that the disease is still spreading. Meanwhile, the higher the recovery rate, the lower value of R_0 obtained. The recovery

rate is deemed to be more highly responsive than the transmission rate due to the clear difference between the curves with different γ values.

With the value of R_0 obtained from the proposed parameter values being 2.52, it implies that the virus are still spreading, clearly a lot of improvements needed to be made in order to prevent this from happening. In the next research, it is suggested to include more factors into the system such as vaccination and quarantine factors especially when the world is already trying to overcome the COVID-19 situation. The additional factors are going to make the finding more accurate to the current situation.

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