Scientific report on a modified SEIR model applied to the data of COVID-19

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Abstract

The COVID-19 pandemic has resulted in a significant loss of human life around the world and it poses an unprecedented threat to public health, food systems, and the workplace. Since then, researchers from all around the world published a great number of COVID related papers and articles. In this scientific report we will study one of the papers which used a modified SEIR model applied to data of COVID-19 spread in Saudi Arabia. In this present study we will briefly analyze the structure of the model. We will look into such statistical simulation which further has been compared to real time data to test for accuracy. We conclude from these simulations the protocols through which we can reduce the outbreak. We will explain mathematical concept to have better understanding about the model methodology. Moreover, we will also calculate R_0 by using different method called next generation matrix method NGM to see the R_0 we have seen in the paper is same with other methods. Furthermore, we will discuss the local stability and sensitivity of the model at disease free equilibrium DFE. We will provide an extended model by adding a new parameter and calculate the corresponding reproduction number. Further examining the stability of R_0 following the technique mentioned in our assigned paper [7]. For comparison with changes of various embedded parameters we will plot different graphs to observe the changes more clearly. The whole idea of building compartmental models and running simulations is to avoid an epidemic and study how a disease behaves in long term.

Introduction

A Novel coronavirus was identified on 7 January 2020 and temporarily named "2019-nCoV" and it was subsequently named the "COVID-19 virus". Since then it has spread worldwide and affected substantial number of people. The 2019 Novel Corona Virus Infection (COVID 19) becomes a global public health crisis. Despite the fact that the outbreak represents a globally unprecedented threat, we still have a lot to learn about COVID 19 epidemiology, transmission dynamics, research methodologies, and governance.

Diseases are unpredictable which make them hard to control when there is an epidemic. An epidemic can be identified, interpreted and predicted through statistical simulations. A few of them have been listed in the paper [7].

Various factors affect the spread of a pandemic. Governments enacted various measures to curb the spread of COVID 19 such as: strictly limiting social interactions, closing down institutions, implementing lockdowns , restricting travels nationwide before the developments of vaccines. The interplay of several of these well-known factors have a significant impact on the spread of the virus. There are also some subtle and unknown factors that indirectly contribute to the spread of the virus. Nevertheless, even a simulation model based on a limited number of factors could be very useful for studying and analyzing COVID 19.

Compartmental modelling technique uses Ordinary differential equations to simulate how individuals categorized into different compartments(also dynamics) interact in a certain population. The interaction between various compartments is affected by various parameters. These kind of models are very helpful in epidemic simulations. The most basic form of a compartmental model is the SIR model. The model consists of three compartments where the S stands for susceptible, the I for Infected and the R for recovered, in a disease exposed population. The model is built on the assumption that out of a selected population, an individual transitions from a susceptible stage to an infected stage and then finally to the recovery.

Although there exist different models, our focus will be on the SEIR model where we have one more dynamic call the Exposed E. Here, a susceptible individual moves to an exposed stage before moving on to an infectious stage. For the sake of simplicity, different authors define these dynamics according to their purpose.

The purpose of this study is to employ the SEIR model to investigate COVID-19 dynamics and transmission. We will study and critically examine the SEIR model proposed in [7]. As we will study further the methodology, we will have a better understanding of why we use SEIR models to represent an infectious disease. We will compute and understand Equilibrium points. We will look into the basic reproduction number R_0(a well-known epidemiological concept for quantifying the transmission of an infectious disease) which is a key indicator of how a disease behaves in the long term. We will look at different methods of finding the R_0 and checking for stability of the model proposed. Furthermore we will be proposing an extended model which will help us understand more about COVID-19 and help us create protocols to reduce the virus outbreak.

Proposed Methodology

Formulation of the proposed modified SEIR model

The proposed model divides the populations (Fig 1) into four dynamic sub-populations which can be described with the following parameters of transmission rates.

- S(t) denotes the susceptible population.
- E(t) is the exposed population who are infected but who have not been detected by testing.
- I(t) denotes the people confirmed to have been infected and under treatment.
- R(t) is the population living in a secure zone or not affected by COVID-19.
- $\beta > 0$ is the transmission rate from a susceptible population to an infected population, which has not been detected.

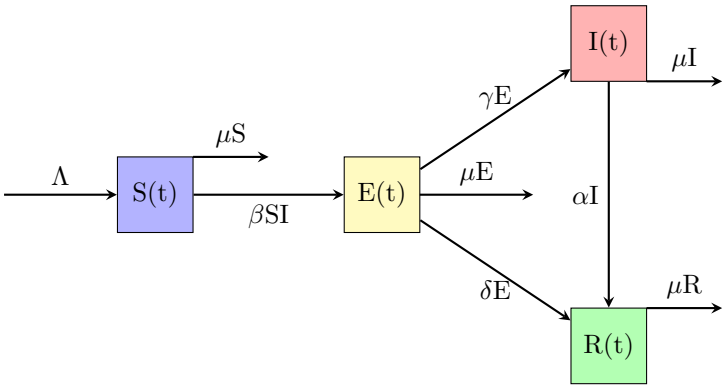

Fig 1. Flowchart of the proposed SEIR model

- $\Lambda > 0$ comprises new births and new residents per unit value of time.
- $\mu > 0$ is the rate of natural death.
- $\gamma > 0$ is the transmission rate of confirmed infected people from the exposed population ($\frac{1}{\gamma}$ is approximately the duration of the latent period).
- $\delta > 0$ is the transmission rate of recovery from the exposed population ($\frac{1}{\delta}$ is approximately the duration for which infection is suspected).
- $\alpha > 0$ is the transmission rate of recovery from the infected population (mean time spent in the "infectious" category of $\frac{1}{\alpha}$).

The total population $N(t)$ is defined as follow:

$$N(t) = S(t) + E(t) + I(t) + R(t) \tag{1}$$

The inflows and outflows in (Fig 1) are converted into first-order, ordinary non-linear differential equations by following the steps below.

$$\frac{dS(t)}{dt} = \Lambda - \beta S(t)I(t) - \mu S(t) \tag{2}$$

$$\frac{dE(t)}{dt} = \beta S(t)I(t) - \epsilon_1 E(t) \tag{3}$$

$$\frac{dI(t)}{dt} = \gamma E(t) - \epsilon_2 I(t) \tag{4}$$

$$\frac{dR(t)}{dt} = \delta E(t) + \alpha I(t) - \mu R(t) \tag{5}$$

Here $\beta S(t)I(t)$ is the number of individual who become infected per unit of time also called the force of infection and and $\epsilon_1 = (\gamma + \mu + \delta)$, $\epsilon_2 = (\alpha + \mu)$, and $\delta = 0$ lead to the usual SEIR model.

Discussion on modeling selection

To begin with the discussion of modeling of the infectious pandemic disease we can ask ourselves few questions for instance, Is a mathematical model able to represent a real world pandemic situation? If so then how can we compare among different models representing same pandemic situation? In search of answering such questions we have

learnt that an enormous amount of works have been done over the years by scientists and researchers and still continuing.

"A mathematical model is an imaginary microworld consisting of entities behaving according to precisely specified rules. Mathematics provides us with a language for formulating these rules of behaviour in a concise and unambiguous way, thus forcing and helping us to clearly state our assumptions. Once a mathematical model is constructed, mathematical analysis, often combined with computer simulations, helps us to investigate the global behaviour of the model, drawing out the consequences of the assumptions that we have made. [3]"

A mathematical model for the spread of an infectious disease in a population of hosts describes how the pathogen spreads among hosts based on patterns of infectious and susceptible individual contacts, the latency period between infection and infectiousness, the duration of infectiousness, the extent of immunity acquired following infection, and other factors.We can forecast the number of people who will be infected during an epidemic, the duration of the epidemic, the peak incidence, and, indeed, the entire epidemic curve, giving us with the expected number of cases at any point in time, if all of these parameters are defined in a model.

Having said that we must carry it in mind that there is no one perfect model to answer all the challenges arose during a pandemic. This is a continuous process to develop the model corresponding to the phase of the pandemic. Before a pandemic a reasonable way of making a model could be focused on planning and identifying the critical gaps, which area are more likely to catch the disease and would also be able to suggest the policy makers what sorts of actions to take to minimize the pandemic devastation. Similar way as the pandemic once take place a model should be able to suggest different aspects such as various intervention and control methods; projecting the rate of epidemic occurrence, hospitalization, and mortality; efficient use of the medical resources etc. The modeling for post pandemic rather focus on mostly on long term impact caused by the pandemic. [4]

The proposed modified SEIR model was submitted on 15 September,2020.The study was made on the time interval between 1 April,2020 and 5 August, 2020. Where the real data of COVID-19 spread shows that the infectious number from 1 April, 2020 it started rising and reached to peak on 17 June,2020 and then gradually it started falling.

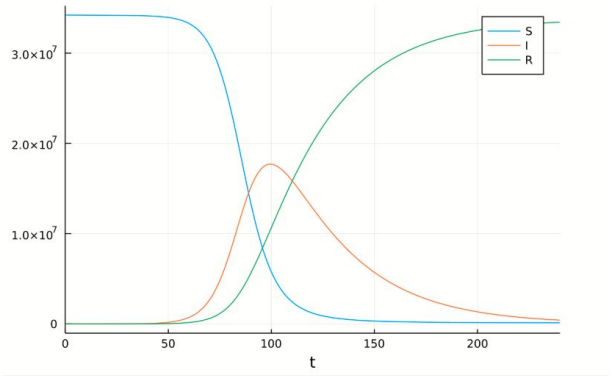

Fig 2. SIR model

The classical SIR model for epidemic modeling is consisted of three dynamics consecutively susceptible, infectious and recovered populations. In the proposed SEIR model it is consisted with one more which is exposed populations.

Due to a 14-day incubation period, COVID-19 infection has a biologically realistic latency; newly infected persons may not be infectious while the virus is incubating in the body. For COVID-19, we distinguish between the latent period, which is the time

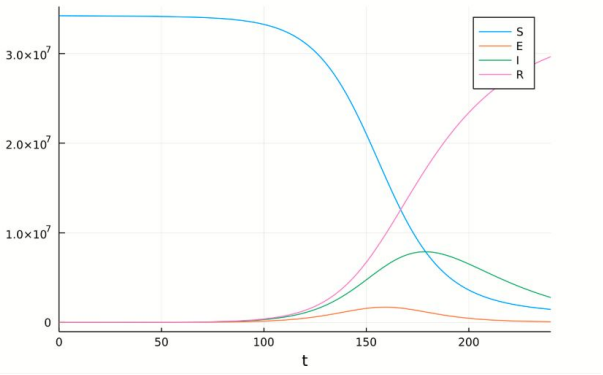

Fig 3. SEIR model

between when an individual is infected and when they become infectious, and the incubation phase, which is the time between when a someone is infected and when clinical symptoms, such as fever and coughing, occur. Infected people become infectious two days after symptoms appear in the case of SARS. As a result, the latent period for SARS is typically longer than the incubation period. In the case of COVID-19, data suggests that infected people can be infectious before symptoms appear, although the length of the latent phase is uncertain. The SEIR model has the advantage of being more biologically realistic when compared to the SIR model, but it also has the disadvantage of having two more unknown parameters: the latent time and the initial latent population. [5]

In the (Fig 2) and (Fig 3) we have assumed the same parameter value necessary for both SIR and SEIR model and comparing the output we can see that the SEIR model output gives us more closer value to the real data. In real data we have learned that the peak time was in middle of June and time interval between 1 April,2020 and 5 August, 2020. In (Fig 3) we can see the rise of infectious people begins around day 100(first week of April) and reach to peak around day(160-180) and then it starts to decrease. On the other hand SIR output in (Fig 2) neglect some important information about the exposed populations but this could be also useful to make people aware earlier while we all know prevention is better than cure.

Having said that the addition of exposed population to the model have some advantages but it could be considered in many different ways. For instance taking consideration of quarantine could be an important factor. Though in the proposed model natural death is taken under consideration but a dedicated compartment for death due to the pandemic could be more informative. Increasing all these factors would also make the model more difficult and complex as the number of unknown parameters would rise accordingly. Eventually we can add some parameters to the existing proposed SEIR model without increasing the number of components. For instance Study shows that less than 1% of recovered people got reinfected [6]. Some of the changes we will try to make in the model in upcoming sections.

Properties of the System

Before we move on to the solution of the system two theorems are needed to be considered carefully which are main conditions and also to check correctness of the solution.

- **Theorem 1** (Solutions are Never Negative.):
  Statement: "All the solutions of the proposed SEIR model with its initial condition are a subset in the interval $[0, \infty)$ and $\{S(t), E(t), I(t), R(t)\} \geq 0^{\frac{x-\mu}{\delta}}$ for all values $0 \geq t > \infty$".

  A simple explanation of the theorem is that at any given time t, values of all 4 dynamics $S(t), E(t), I(t), R(t)$ are always non-negative.Which makes sense because population can not be a negative number at any given time. The proof is quite simple and welly briefed in the main paper [7].

- **Theorem 2**(Solution domain):
  Statement:"All the solutions of the proposed SEIR model structure that initiate in $\mathbb{R}^4_+$ are bound within the region ψ defined by
  $\psi = \{(S, E, I, R) \in \mathbb{R}^4_+ : 0 \leq N(t) \leq \frac{\Lambda}{\mu}$ as $t \to \infty$."

  The solution always bounded by the region $[0, \frac{\Lambda}{\mu}]$. At any time t, N depends on the number of new residents (Λ) and inversely depends on the death rate by natural causes (μ). The total population will at most be the ratio of these two factors.

Epidemic Equilibrium And Reproduction Number

Epidemic equilibrium

In our model we have the first order non-linear differential equations (2),(3),(4) and (5). By setting all the derivatives equal to zero we will have,

$$S'(t) = E'(t) = I'(t) = R'(t) = 0 \to \{S, E, I, R\} \equiv constants \neq 0 \tag{6}$$

The equations (2),(3),(4) and (5) give us the following respectively

$$0 = \Lambda - \beta SI - \mu S \tag{7}$$

$$0 = \beta SI - \epsilon_1 E \tag{8}$$

$$0 = \gamma E - \epsilon_2 I \tag{9}$$

$$0 = \delta E - \alpha I - \mu R \tag{10}$$

Now solving with (7),(8),(9) and (10) we computed the value for I and we have the following:

$$I = \frac{\mu}{\beta}(\frac{\beta\gamma\Lambda}{\mu\epsilon_1\epsilon_2} - 1) \tag{11}$$

where

$$R_0 = \frac{\beta\gamma\Lambda}{\mu\epsilon_1\epsilon_2} \tag{12}$$

Basic reproduction number

In general, the basic reproduction number (R_0) is a well-known epidemiological concept for quantifying the transmission of an infectious disease. It is defined as the average number of secondary cases induced by a single main case in a population where no one is immune or vaccinated. It is so characterized at the beginning of an outbreak, and in particular before any public health measures are initiated.A value of R_0 greater than 1 suggests that the number of instances of the disease in the population will expand exponentially, whereas a value of R_0 less than 1 indicates that the outbreak will end. [10] [2]

Equilibrium by application of the Jacobian matrix

There are various ways to calculate R_0 However, the Jacobian matrix at the disease-free equilibrium is relatively tractable [8].

The value of R_0 has to be unique thus the equilibrium will also be unique. Different R_0 for the same model depicts the model as unpredictable and hard to understand. To check the uniqueness of R_0 we need to apply the Jacobian Matrix which will give us the same R_0 at a diseases free equilibrium (DFE) in the form $E_0 = (\frac{\Lambda}{\mu}, 0, 0, 0)$. The Jacobian Matrix of the model takes the following form:

$$J_{E_0} = \begin{bmatrix} -\beta I - \mu & 0 & -\beta S & 0 \\ \beta I & -\epsilon_1 & \beta S & 0 \\ 0 & \gamma & -\epsilon_2 & 0 \\ 0 & \delta & \alpha & -\mu \end{bmatrix} \tag{13}$$

The Jacobian is linearized at DFE $E_0 = (\frac{\Lambda}{\mu}, 0, 0, 0)$ and thus we get new Jacobian Matrix.

$$J_{E_0} = \begin{bmatrix} -\mu & 0 & -\beta\frac{\Lambda}{\mu} & 0 \\ 0 & -\epsilon_1 & \beta\frac{\Lambda}{\mu} & 0 \\ 0 & \gamma & -\epsilon_2 & 0 \\ 0 & \delta & \alpha & -\mu \end{bmatrix} \tag{14}$$

If the matrix J_{E_0} is obtained from linearization and is the Jacoibian evaluated at equilibrium DFE $(E_0) = (\frac{\Lambda}{\mu}, 0, 0, 0)$, the condition $|J_{E_0}| \neq 0$ means that the equilibrium is isolated, so there is a disk around it that does not contain other equilibria. Therefore, from (14), we have

$$det(J_{E_0}) = \begin{vmatrix} -\mu & 0 & -\beta\frac{\Lambda}{\mu} & 0 \\ 0 & -\epsilon_1 & \beta\frac{\Lambda}{\mu} & 0 \\ 0 & \gamma & -\epsilon_2 & 0 \\ 0 & \delta & \alpha & -\mu \end{vmatrix} = \mu^2\epsilon_1\epsilon_2(\frac{\beta\gamma\Lambda}{\mu\epsilon_1\epsilon_2} - 1) \tag{15}$$

Thus it can be observed that the condition (15) is a unique equilibrium of the system. And the condition for unique equilibrium of the model is

$$\frac{\beta\gamma\Lambda}{\mu\epsilon_1\epsilon_2} - 1 \neq 0 \tag{16}$$

Hence $R_0 = \frac{\beta\gamma\Lambda}{\mu\epsilon_1\epsilon_2}$ is unique.

Computing R_0 by Next Generation Matrix method[NGM]

Epidemiologically, the linearization reflects that R_0 characterizes the potential for initial spread of an infectious agent when introduced into a fully susceptible population, and that we assume that the change in the susceptible population during initial spread is negligible. This linearized and reduced infected system is the starting point for our calculations.Where the vector of states are $\underline{x} = (E, I)$. We can give the reduced infected system this form.

$$\frac{d\underline{x}}{dt} = T(\underline{x}) - V(\underline{x}) \tag{17}$$

Where $T(\underline{x}) = \begin{bmatrix} \beta SI \\ 0 \end{bmatrix}$ and $V(\underline{x}) = \begin{bmatrix} -\epsilon_1 E \\ \gamma E - \epsilon_2 I \end{bmatrix}$. The system is linearized at the DFE $E_0 = (\frac{\Lambda}{\mu}, 0, 0, 0)$ so at any step S is always equal to $\frac{\Lambda}{\mu}$. Any linear ODE system is described by a matrix, usually called a Jacobian matrix, when derived by linearizing the original nonlinear ODE system. Our objective is to relate the structure of this matrix to the epidemiological interpretation. In particular, we explain how to determine R_0 by using the decomposed matrix (17), where T is the transmission part that describes the production of new infections and V is the transition part that describes state changes (death or immunization). Then, we calculate the eigenvalue of the matrix $-TV^{-1}$.

$$T = \begin{bmatrix} 0 & \beta S \\ 0 & 0 \end{bmatrix} \tag{18}$$

$$V = \begin{bmatrix} -\epsilon_1 & 0 \\ \gamma & -\epsilon_2 \end{bmatrix} \tag{19}$$

As we all know for V^{-1} to exist then the $det(V) \neq 0$. Which is true for this case. Then by Cayley–Hamilton method we can compute the inverse which will be the following:

$$V^{-1} = \begin{bmatrix} -\frac{1}{\epsilon_1} & 0 \\ -\frac{\gamma}{\epsilon_1 \epsilon_2} & -\frac{1}{\epsilon_2} \end{bmatrix} \tag{20}$$

The next step is to calculate $-TV^{-1}$ and finding the eigenvalues of the matrix.

$$A = -TV^{-1} = \begin{bmatrix} \frac{\beta S \gamma}{\epsilon_1 \epsilon_2} & \frac{\beta S}{\epsilon_2} \\ 0 & 0 \end{bmatrix} \tag{21}$$

The eigenvalues x of the matrix A are $x = 0$ or $x = \frac{\beta S \gamma}{\epsilon_1 \epsilon_2}$ from the characteristics polynomial $det(xI - A) = 0$. As $S = \frac{\Lambda}{\mu}$ we will have the dominant eigenvalue as $x = \frac{\beta \Lambda \gamma}{\mu \epsilon_1 \epsilon_2}$, which is the R_0 of the proposed model. The R_0 computed from NGM method is same as calculated previously in (12).

Stability And Sensitivity Analysis Of Equilibira

Local stability

Furthermore lets look into the stability of the equilibrium(in our case the DFE(E_0)). The notion of stability(Lyapunov stability) of the equilibrium E_0 encapsulates the following property:

A solution remains close to E_0 in forwards time provided that it starts sufficiently close to E_0.

We will discuss more about the Lyapunov stability more in the next section.

In epidemiology stability can be inferred as an end of the epidemic or also getting back to the state of equilibrium.

Stability can be checked from the Jacobian matrix of the system (14), which is defined at DFE $(E_0) = (\frac{\Lambda}{\mu}, 0, 0, 0)$, by computing the characteristics equation and the eigen values(roots of the characteristics equation say x). Two of the roots are $x_1 = x_2 = -\mu$ and the other two are the solution of the following equation:

$$\begin{vmatrix} -\epsilon_1 - x & \frac{\beta\Lambda}{\mu} \\ \gamma & -\epsilon_2 - x \end{vmatrix} = 0 \tag{22}$$

which gives

$$(\epsilon_1 + x)(\epsilon_2 + x) - \frac{\gamma\beta\Lambda}{\mu} = 0 \tag{23}$$

The roots of the equation take the following forms:

$$x_3 = -\frac{1}{2}(\epsilon_1 + \epsilon_2) - \sqrt{(\epsilon_1 - \epsilon_2)^2 + 4\epsilon_1\epsilon_2 R_0} \tag{24}$$

$$x_4 = -\frac{1}{2}(\epsilon_1 + \epsilon)_2 + \sqrt{(\epsilon_1 - \epsilon_2)^2 + 4\epsilon_1\epsilon_2 R_0} \tag{25}$$

For the system to be asymptotically stable all the eigen values of the matrix J_{E_0} must have negative real part. In the above equations (19) and (20), the roots will be negative if $R_0 < 1$. Thus, if $R_0 < 1$, then the DFE E_0 is locally asymptotically stable. If $R_0 >= 1$ then DFE E_0 is locally asymptotically unstable.

Local sensitivity

The idea of Local sensitivity analysis simply says that how much it will change in the output values in terms of changing one parameter of input value. If the sensitivity or elasticity of quantity H and the parameter is P, it follows:

$$\wp_H^p = \frac{\frac{\partial H}{\partial P}}{\frac{H}{P}} = \pm\frac{\%\Delta H}{\%\Delta P} \tag{26}$$

It can be clearly noticed from this equation that the sensitivity of H with respect to P is positive if H is increasing with respect to P and negative if H is decreasing with respect to P.

Now we will observe how $R_0 = \frac{\beta\gamma\Lambda}{\mu(\gamma+\mu+\delta)(\alpha+\mu)}$ changes with the change in parameters(once at a time).

$$\wp_{R_0}^{\beta} = \frac{\frac{\partial R_0}{\partial \beta}}{\frac{R_0}{\beta}} = 1 > 0 \tag{27}$$

$$\wp_{R_0}^{\gamma} = \frac{\frac{\partial R_0}{\partial \gamma}}{\frac{R_0}{\gamma}} = \frac{\mu + \delta}{\epsilon 1} > 0 \tag{28}$$

$$\wp_{R_0}^{\mu} = \frac{\frac{\partial R_0}{\partial \mu}}{\frac{R_0}{\mu}} = -\left(1 + \frac{(\epsilon 1 + \epsilon 2)\mu}{\epsilon 1 \epsilon 2}\right) < 0 \tag{29}$$

$$\wp_{R_0}^{\delta} = \frac{\frac{\partial R_0}{\partial \delta}}{\frac{R_0}{\delta}} = -\frac{\delta}{\epsilon 1} < 0 \tag{30}$$

$$\wp_{R_0}^{\alpha} = \frac{\frac{\partial R_0}{\partial \alpha}}{\frac{R_0}{\alpha}} = -\frac{\alpha}{\epsilon 2} < 0 \tag{31}$$

From the equations (24) , (25) and (26) we can say that 1% increase in each one (μ, δ, α) will produce $\left(1 + \frac{(\epsilon 1+\epsilon 2)\mu}{\epsilon 1 \epsilon 2}, \frac{\delta}{\epsilon 1}, \frac{\alpha}{\epsilon 2}\right)$% decrease in R_0. From equation (23) 1% increase in γ will increase R_0 by $\frac{\mu+\delta}{\epsilon 1}$%. And finally from equation (22) 1% increae of β will increase the R_0 by 1%.

Lyapunov functions and global stability

In higher-dimensional systems, there are a variety of methods for determining the global stability of an equilibrium. One of the most commonly used functions is the Lyapunov function. Lyapunov functions are scalar functions that can be used to demonstrate the global stability of an equilibrium.

Definition: A scalar function V(x) such that $V : R^n \to R$ is called **radially unbounded** if $V(x) \to \infty$ if $\|x\| \to \infty$. One significant property of Lyapunov functions is that they are positive definite in the entire space.

Definition: Let V be a continuous scalar function, that is,$V : R^n \to R$. The function V is called positive definite on the entire space if

- $V(x^*) = 0$,
- $V(x) > 0$ for $x \neq x^*$

where x^* is an equilibrium of the autonomous system. [9]

Lyapunov stated that if a function $V(x)$ is globally positively definite and radially unbounded, and its time derivative is globally negative, $V(x) < 0$ for all $x \neq x^*$, then the equilibrium x^* is globally stable for the autonomous system $x' = f(x)$, and $V(x)$ is called the Lyapunov function [7] [9].

Theorem(Global stability):

Statement: The SEIR model DFE$(E_0) = (\frac{\Lambda}{\mu}, 0, 0, 0)$ is globally stable of the DFE under the condition $R_0 < 1$.

The proof of the theorem is explained in the paper [7] clearly. With the consideration of proposed SEIR model in space of the first three variables only (S, E, I). It is clear that if the DFE for the first three equations is globally stable, then $R \to 0$, and the DFE for the full SEIR model is globally stable. Based on that consideration a Lyapunov function V is introduced. Using the derivative in Lyapunov function V it can be showed that $V' \leq 0$ for every $(S(t), E(t), I(t)) \geq (\frac{\Lambda}{\mu}, 0, 0)$. which completes the proof.

Extension Of The Model

According to the assumptions of the case study we have discussed and computed the equilibrium and Basic Reproduction number. We will introduce a possible extension of the SEIR model. We will use the same model but with a new parameter and new mode of transmission of the virus. The new introduced parameter λ which is the rate of transmission from susceptible (S) to the exposed state (E). According to the defined exposed state(the exposed population who are infected but who have not been detected by testing.), we can actually interpret that the exposed are also likely to infect if they

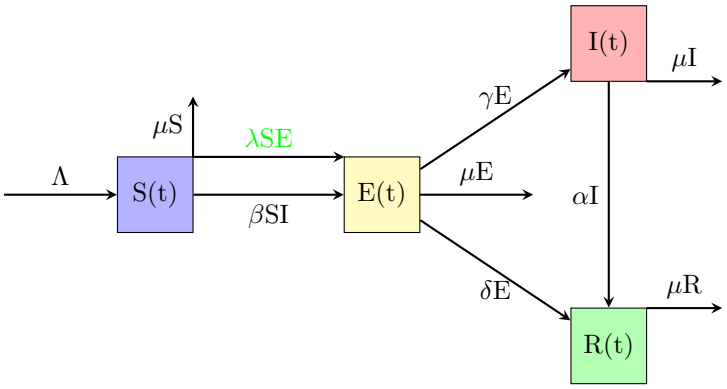

Fig 4. Flowchart of our modified SEIR model

have contact with the susceptible populous. Thus we can show this with a new compartmental model.

The inflows and the outflows give us the new first-order, ordinary non-linear differential equations are the following:

$$\frac{dS(t)}{dt} = \Lambda - \beta S(t)I(t) - \mu S(t) - \lambda S(t)E(t) \tag{32}$$

$$\frac{dE(t)}{dt} = \beta S(t)I(t) - \epsilon_1 E(t) + \lambda S(t)E(t) \tag{33}$$

$$\frac{dI(t)}{dt} = \gamma E(t) - \epsilon_2 I(t) \tag{34}$$

$$\frac{dR(t)}{dt} = \delta E(t) + \alpha I(t) - \mu R(t) \tag{35}$$

Lets derive the Base reproduction number from the system of equations. We will use he NGM method. Here the vector of states are $\underline{x} = (E, I)$. We will have $T(\underline{x}) = \begin{bmatrix} \beta SI + \lambda SE \\ 0 \end{bmatrix}$ and $V(\underline{x}) = \begin{bmatrix} -\epsilon_1 E \\ \gamma E - \epsilon_2 I \end{bmatrix}$. Thus we can give it the form of (17) and compute the eigen values of $-TV^{-1}$.

Where

$$T = \begin{bmatrix} \lambda S & \beta S \\ 0 & 0 \end{bmatrix} \tag{36}$$

and

$$V = \begin{bmatrix} -\epsilon_1 & 0 \\ \gamma & -\epsilon_2 \end{bmatrix} \tag{37}$$

We will solve the inverse of the matrix V by Cayley–Hamilton method. Which is the following:

$$V^{-1} = \begin{bmatrix} -\frac{1}{\epsilon_1} & 0 \\ -\frac{\gamma}{\epsilon_1 \epsilon_2} & -\frac{1}{\epsilon_2} \end{bmatrix} \tag{38}$$

Further,

$$B = -TV^{-1} = \begin{bmatrix} \frac{\lambda S}{\epsilon_1} + \frac{\beta S \gamma}{\epsilon_1 \epsilon_2} & \frac{\beta S}{\epsilon_2} \\ 0 & 0 \end{bmatrix} \tag{39}$$

The characteristics equation of the matrix B is $det(xI - B) = 0$, which will give us the dominant eigen value as $x = \Lambda(\frac{\lambda\epsilon_2+\beta\gamma}{\epsilon_1\epsilon_2\mu})$ at the DFE ($E_0 = (\frac{\Lambda}{\mu}, 0, 0, 0)$). As we know the dominant eigen value is the value of the Basic Reproduction Number (R_0). So our new R_0 in the system shown in Fig4 is as follows.

$$R_0 = \Lambda(\frac{\lambda\epsilon_2 + \beta\gamma}{\epsilon_1\epsilon_2\mu}) \tag{40}$$

We will check the stability of the R_0 calculated in (40). The first step is to calculate the Jacobian Matrix of the given system of equations at DFE ($E_0 = (\frac{\Lambda}{\mu}, 0, 0, 0)$). Then we will compute the eigen values from the characteristics polynomial $det(xI - J_0) = 0$ and get a sense of the stability of the SEIR model. We can say that equation (35) is redundent and we need not include it in the calculation of the Jacobian Matrix. Thus Making our matrix of the following form:

$$J_0 = \begin{bmatrix} -\mu & \frac{-\lambda\Lambda}{\mu} & \frac{-\beta\Lambda}{\mu} \\ 0 & \frac{-\epsilon_1\mu+\lambda\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} \\ 0 & \gamma & -\epsilon_2 \end{bmatrix} \tag{41}$$

The above mention Jacobian gives us one of the eigen values $x = -\mu$. The other roots of the characteristics polynomial are as follows:

$$x = \frac{1}{2}((\epsilon_1 + \epsilon_2 - \frac{\lambda\Lambda}{\mu}) \pm \sqrt{(\frac{\epsilon_1\mu + \epsilon_2\mu - \lambda\Lambda}{\mu})^2 - 4(\frac{\Lambda(\beta\gamma + \lambda\epsilon_2)}{\mu\epsilon_1\epsilon_2} - 1))} \tag{42}$$

Which can be also written as:

$$x = \frac{1}{2}((\epsilon_1 + \epsilon_2 - \frac{\lambda\Lambda}{\mu}) \pm \sqrt{(\frac{\epsilon_1\mu + \epsilon_2\mu - \lambda\Lambda}{\mu})^2 - 4(R_0 - 1))} \tag{43}$$

When we input the R_0 in the (42) we can see that for the roots to be real the R_0 needs to be less then 1. Which shows that if $R_0 < 1$, then the model is asymptotically stable and vice versa.

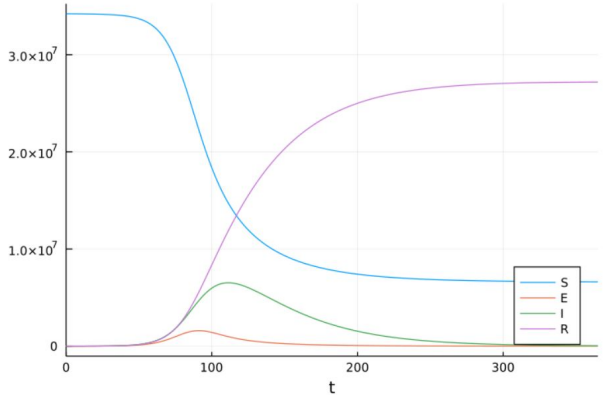

Fig 5. Extended SEIR Model

To have a better look at the extended model so the behaviour can be observed. A plot of the model has been shown in Fig5. The new parameter $\lambda \approx 0.1\text{x}10^{-7}$. As the number of exposed was much higher then the number of infectious at the initial stage we can assume the rate of transmission to be higher as well. Thus making our epidemic rise faster and consecutively drop fast too. It can be seen in 5 the all 4 dynamics reach

an equilibrium around day 200 depicting our extended model stable. It can be observed that the outbreak dies out before the value of susceptible get to 0. Thus making it possible for the infection rise again at a later stage. This is possible through other factors which can be added to the model.

Discussion

Findings and comparisons

To know how much similar the proposed model is to the real situation in Saudi Arabia it needs to be compared with real time data. The statistical data was acquired from Saudi Ministry of Health(MOH). The time line of the data is from 1st of April to 5 of August. Evidence of COVID-19 epidemic in Saudi Arabia was tested by March 3 but it was later till April 1st low number of positive cases were reported to increase. Thus 1st of April is considered the real start of COVID-19 epidemic in Saudi Arabia. The Predicted SEIR model has shown to be giving similar results as that of the data acquired by the Saudi MOH. But making further calculations checks and creating plots of the SEIR model it can be seen that is not the case.Lets Look into the infectious populous plot for the given parameter and initial values in [7].

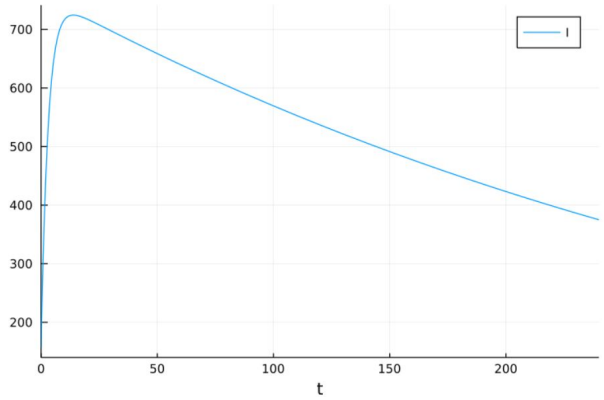

Fig 6. Predicted max infected population.

The results Fig6 shows the max number of people predicted in the infectious dynamic. Comparing this plot to the real data acquired from Saudi MOH shown in Fig7 we see that the SEIR model proposed is giving us different values. Thus needing further investigations. According to the parameters suggested in (cite our paper) we calculated $R_0 \approx 0.5$. But the suggested R_0 is 2.008. These contradicting values are hard to examine because of lack of information on how the parameters were calculated.

Even more critical investigation shows there are some assumptions which have never been Mentioned. The proposed model assumes the population is immunized after getting recovered. Which was not true for COVID-19 as evidence over time proves patients who recover from it have a very good chance of getting infected by the virus.

The force of infection (β) suggested in the SEIR model is very small and thus making the outbreak very slow. One of the reasons such a value for β was assumed because during the time at which the initial conditions are assumed Saudi Arabia was under Lockdown. One of the values which has not been exmined is the death from the disease itself. This is important here because evidence shows COVID-19 has a high mortality rate. At the time under consideration total number of death cases was ≈ 3000.

It is understood that at the time under consideration there was not much information on COVID-19. Which maybe the reason of lack of concrete proof and

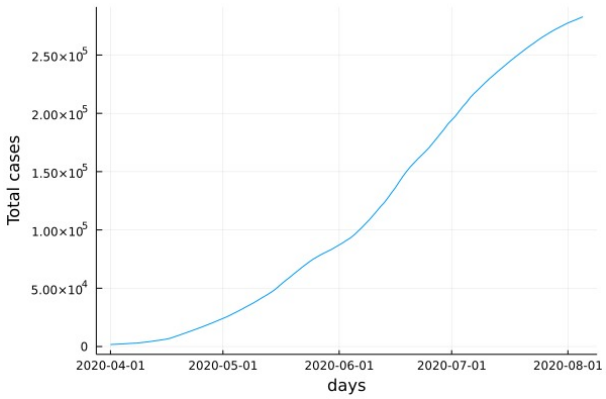

Fig 7. Actual max infected population.

assumptions on which the proposed model has been interpreted. But with some fine tuning the shortcomings of the model can be met.

Conclusion

The world has been under a partial lockdown ever since COVID-19 has been detected. Because of the unknown and unpredictable nature of the disease we give it a mathematical structure to make more sense of it. The purpose of the study was to understand and analyse the nature of the pandemic the world is facing and to rule out protocols and ways of intervention to reduce the spread of the virus. We have pointed out some Protocols which we believe can be helpful to reduce the pandemic.

- As the virus COVID-19 was new in nature it was critical that one of the first things needed was a way to test and detect individuals infected with it much faster.
- On successful detection of these individuals, quarantine and isolation protocol must be imposed so the infected does not spread the virus further.
- Because of the threatening nature of the virus more thorough lock-downs with controlled interaction should be implemented.
- Self quarantine of any untested individual with symptoms must be a priority.

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References

1. Qiu, Wuqi & Rutherford, Shannon & Mao, A. & Chu, Cordia. The Pandemic and its Impacts..(2017) Health, Culture and Society. 9. 1-11. 10.5195/HCS.2017.221.

2. Locatelli I, Trächsel B, Rousson V. Estimating the basic reproduction number for COVID-19 in Western Europe..(2021) PLoS ONE 16(3): e0248731.

3. A. Huppert and G. Katriel Mathematical modelling and prediction in infectious disease epidemiology (999-1005)..(2013) Clinical Microbiology and Infection

4. Aniruddha Adiga, Devadatt Dubhashi, Bryan Lewis, Madhav Marathe,Srinivasam Venkatramananand Anil vullikanti Mathematical Models for COVID-19 Pandemic: A Comparative Analysis 100,(793-807)(2020). Journal of the Indian Institute of Science

5. Roda, W. C., Varughese, M. B., Han, D., and Li, M. Y Why is it difficult to accurately predict the COVID-19 epidemic?. Infectious Disease Modelling, 5, 271–281 (2020).

6. Adnan I Qureshi, William I. Baskett, Wei Huang, Iryna Lobanova, S. Hasan Naqvi and Chi-Ren Shyu RReinfection With Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) in Patients Undergoing Serial Laboratory Testing. Clinical Infectious Diseases(2021)

7. Youssef,Hamdy M. and Alghamdi,Najat A. and Ezzat,Magdy A. and El-Bary,Alaa A. and Shawky,Ahmed M. A modified SEIR model applied to the data of COVID-19 spread in Saudi Arabia. AIP Advances(2020)

8. Heffernan, J. M., Smith, R. J., and Wahl, L. M. Perspectives on the basic reproductive ratio.. Journal of the Royal Society(2005), Interface, 2(4), 281–293.

9. Maia Martcheva An Introduction to Mathematical Epidemiology. Texts in Applied Mathematics Volume 61(Chapter 7)

10. O. Diekmann, J. A. P. Heesterbeek, and J. A. J. Metz. On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations. Journal of Mathematical Biology(1990), 28:365-382.