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Mathematical modeling of the effect of quarantine rate on controlling the infection of COVID19 in the population of Saudi Arabia

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Received 31 October 2021; revised 5 December 2021; accepted 14 December 2021
Available online 28 December 2021

KEYWORDS
COVID19; Epidemiological disease; Epidemic; Stability; Basic reproduction number

Abstract With the development of communications and transportation worldwide, the challenge of controlling epidemiological diseases becomes higher. The COVID19 has put all nations in a lethal confront with a severe disease that needed serious and painful actions. The sooner the actions, the less destructive the impact. In this paper, we incorporate what we believe is crucial but applicable to control the spread of COVID19 in the populations, that is, quarantine. We keep the model as simple as SI Kermack-McKendrick model with an additional compartment of quarantined patients. We established the system’s basic properties and studied the stability of the disease-free equilibrium and its relation to the basic reproduction number $R_0$ in which we calculated its formula. The focus of our study is to measure the effect of quarantine rate on controlling the spread of COVID19. We use the data collected from the Ministry of Health in Saudi Arabia. We studied three different values of the quarantine rate where newly infectious patients are detected and isolated within 14, 7, and 5 days. The simulations show a significant effect of the quarantine where COVID19 can be fully controlled if the newly infected patient enters the quarantine within five days. These results were proposed to the Public Health Authority in Saudi Arabia and approved by the Ministry of Health in which they applied promptly.

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1. Introduction

Humanity is still grappling with the Coronavirus disease-2019 (COVID-19), which is a deadly respiratory disease and caused by the Coronavirus SARS-CoV-2. Coronavirus are RNA viruses, enveloped non-segmented positive-sense, and belong to the family Coronaviridae and the order Nidovirales, which are widely distributed among humans and mammals [1]. In December 2019, the novel
Coronavirus appeared in China, with a geographical emphasis at Wuhan, the capital of Hubei Province [2]. The initial cases of infections were attributed to the seafood market in Wuhan, and then have widely spread all over the world. [3]. It has been found from clinical trials of patients in the hospital that those infected show symptoms consistent with viral pneumonia at the onset of COVID-19 [4], and these symptoms include fever, cough, sore throat, fatigue, impaired kidney and liver function, change in platelet counts and in addition lymphocytes counts.

On January 2, 2020, 41 confirmed cases of COVID-19 were admitted to hospital [6]. On January 30, 2020, China had about 7734 confirmed COVID-19 cases, while recorded 90 state in about 13 countries, covering Germany, United States, France, Canada, India and United Arab Emirates [3,6,7]. Since December 31, 2019 and as of November 28, 2021, confirmed cases reached 261,317,922 cases and 5,198,624 death cases of COVID-19, have been recorded [8].

Since the outbreak of the Covid-19 epidemic, governments in the world as well as the World Health Organization have been keen to take many strict structures to limit the spread of the epidemic. Scientists and researchers have been studying the pandemic behaviour intensively to understand and analyzing the disease. Many recent works of modeling COVID19 focused on different aspects of the disease. For instance, the recent papers of Sardar et al. [9], they focused on the lockdown effect on the transmission of COVID19. Also, the work of Ndaïrou et al. [10]. Other papers considered the undetected down effect on the transmission of COVID19. Also, the work of McKendrick (1927, 1932, 1933) [17,18]. The model classifies the behaviour of the infectious disease in human population are based on the early simple classic model of Kermack and McKendrick (1927, 1932, 1933) [17,18]. The model classifies three different classes of the population, susceptible S who are not infected but at the risk of getting infected, the infectious I who are infected and capable of transmitting the disease to others, and removed class R who recovered from the disease or died because of it. This classical model, often referred to as SIR model, has successfully matched many epidemics such as the Influenza outbreak in boarding school in England in 1978 [17], and the plague in Mumbai, India in 1905 [17]. The success of the model fittings is because of the relatively short period of these epidemics in the populations where they last for two to three years only before they self cleared. Therefore, there is no need to consider the growth rate of populations. Also, the model does not consider the incubation time from the time a susceptible interacts with an infectious patient until becoming infected. This is also relatively short in epidemics like Influenza, where it takes an average of three days only [4]. More recent disease outbreaks such as the SARS epidemic of 2002, the H5N1 influenza of 2005, the H1N1 influenza of 2009, and the Ebola outbreak of 2014 have been modeled with the reformulation of Kermack-McKendrick model, see [5].

In this research, we are implementing the Kermack-McKendrick model for the reason that COVID19 dynamics in a population is similar to the behavior of Influenza. We are keeping the model’s simplicity because we only want to capture the effect of the quarantine in controlling COVID19 spread in the population. The reason for focusing on the quarantine impact is because it is more controllable than all other factors. The infection rate has the highest effect on any disease spread dynamics, and however, we can not control it easily. The other factor is the recovery rate, which we can not retain in COVID19 since it depends mainly on individuals’ immunity. Conversely, the quarantine rate can be forcibly applied by the authorities therefore fully managed and supervised. Of course, vaccination has a significant impact on an epidemic. Still, we do not consider it in this model because we focus on modelling the first phase of the outbreak before the production of vaccines.

We will use the data from the Saudi Arabia Ministry of Health to apply our model and measure the approximate number of infected patients in different scenarios of different quarantine rates. The model is considering three classes of the populations: susceptible S, infectious I, and quarantined patients Q using three-dimensional differential equations. Note that we are not considering the class of recovered patients nor the mortality class because these classes are not related to our aim of the study. Meaning that the scope of the analysis will only focus on the disease behavior in the population from the susceptible phase until the infectious phase. After that, patients will be either recovered or died and in both cases, they will exit the system. Moreover, we are not considering the case where recovered patients become infected again since the data we are implementing are from the first phase of the infection wave in Saudi Arabia and last for approximately 200 days, and the immunity gained from the first infection lasts for approximately 180 to 240 days [19]. Since we are studying the initial emergence of infection cases in the population of Saudi Arabia that took place in March 2020, we are not considering the vaccination effect as previously mentioned. The significant addition in our model is that we normalize the infection mass action rate by the total population denoting it as \(N(t) = S(t) + I(t) + Q(t)\). Where the previous models either did not normalize the infection rate by the total population as in [20], or considered it but as a fixed constant [10], we keep it as a function of \(t\). Another important consideration is the population growth rate and the mortality rate of all other causes than COVID19. Since we are applying the model to a large population, neglecting the population growth and death can affect simulating the solutions. The mathematical analysis in this paper will prove the positivity and boundedness of the system solutions. We also study the linear local stability of the disease-free by introducing the basic reproduction number \(R_0\). The mathematical formula of \(R_0\) can highlight the critical factors in the disease dynamics in the population. The parameters in the \(R_0\) formula can help reduce the value of \(R_0\) to become less than one, and in this case, the disease will eradicate even if it initially rapidly increases. When \(R_0\) is greater than one, the disease will be an epidemic which is the case in Saudi Arabia and most of the world. We also show that an endemic equilibrium exists when \(R_0 > 1\) and apply the numerical simulations based on the data collected from the Ministry of Health in Saudi Arabia to estimate the parameters values. The Numerical analysis corresponds with the theoretical analysis in predicting correctly the number of infected patients in the endemic steady state. The numerical analysis also confirms the importance of early detection and quarantine of infected patients in controlling the disease of COVID19. This study was proposed to the Public Health Authority on the 1st of April 2020 with recommendations of expanding the COVID19 test centers and enlarge the mass testing scale. It was approved and applied promptly by the Ministry of Health [21].
2. The model

To construct a mathematical model for COVID19 infection in population that consider the quarantine rate of the newly infected patients, we introduce the number of susceptible $S(t)$, the number of newly infectious patients who are not detected yet $I(t)$, and the number infectious patients who are detected and admitted to quarantine $Q(t)$ at time $t$ respectively. Our concern is to study the effect of quarantine in controlling COVID19 infection in population. Therefore, we are not considering the recovered patients nor the vaccinated class here. We model the infection rate using mass action normalized by total population density $N(t) = S(t) + I(t) + Q(t)$, with transmission coefficient denoted by $\beta$. We let $B$ be the overall (not per-capita) birth rate and $\mu$ be the per-capita mortality rate of causes different than COVID19. Moreover, deaths due to COVID19 has the per-capita rate $\mu_t$. We also take the rate in which the infectious patients detected and admitted to quarantine, either a home quarantine or at a health care facility, as $\delta$, where $1/\delta$ is the mean time from the infection until admission to quarantine. Per-capita recovery rate from COVID19 is $\alpha$ for patients not in quarantine and $\alpha_q$ for patients in quarantine. Therefore, we propose the following system of differential equations:

\[
\begin{align*}
\frac{dS(t)}{dt} &= B - \frac{\beta S(t)I(t)}{N(t)} - \mu S(t), \\
\frac{dI(t)}{dt} &= \beta \frac{S(t)I(t)}{N(t)} - (\delta + \alpha + \mu) I(t), \\
\frac{dQ(t)}{dt} &= \delta I(t) - (\alpha_q + \mu) Q(t).
\end{align*}
\] (2.1)

All parameters are strictly positive and initial conditions are $S(0), I(0), Q(0) \geq 0$ such that $S(0) + I(0) + Q(0) > 0$ since $N$ appears in the denominators of some terms.

3. Basic properties and linear stability

We begin the analysis by establishing the positivity and boundedness of the system’s solutions.

3.1. Positivity and boundedness

Proving the system’s positivity requires proving the non-negativity of solutions first. Then we will prove that solutions are strictly positive, meaning that solutions become and remain positive for all time.

Proposition 1. In system (2.1), suppose that $S(0), I(0), Q(0) \geq 0$ with $N(0) > 0$. Then for all $t > 0$ all variables remain non-negative.

Proof. From the equations of (2.1), we have $\frac{dS(t)}{dt} = B > 0$ when $S(t) = 0$. Then $S(t) > 0$ for all $t > 0$. All other equations have a similar property of non-negativity but not the strict positivity. Now we can apply Theorem 5.2.1 on page 81 of Smith [22], since the system has the necessary structure to do so. Non-negativity of each variable follows. □

We can also prove results on strict positivity of solutions but this depends on the initial data. The main sufficient conditions are that all disease variables $I$ and $Q$ become (and remain) strictly positive.

Proposition 2. Suppose that $S(0) > 0$ and that $Q(0) \geq 0$. Then $I(t)$ and $Q(t)$ are strictly positive for all $t > 0$ if and only if $I(0) > 0$.

Proof. Suppose that $S(0) > 0$ and that $Q(0) \geq 0$. From second equation on (2.1), we have

\[
\frac{dI(t)}{dt} = \left( \frac{\beta S(t)}{N(t)} - \delta \right) I(t),
\]

then, $I(t) = I(0) \exp \left[ \int_{0}^{t} \left( \frac{\beta S(s)}{N(s)} - \delta \right) ds \right] > 0$

for all $t > 0$ if and only if $I(0) > 0$.

From the third equation of (2.1), we have

\[
Q(t) = Q(0) e^{-\left(\alpha_q + \mu_q\right)t} + \delta \int_{0}^{t} e^{-\left(\alpha_q + \mu_q\right)(t-s)} I(s) ds \\
\geq \delta \int_{0}^{t} e^{-\left(\alpha_q + \mu_q\right)(t-s)} I(s) ds,
\]

by the strict positivity of $I(t)$ on $[0, \infty)$ proved above, then $Q(t) > 0$.

□

For boundedness of solutions, we need to find an invariant set $\Omega$ where solutions started in this set remain within it. One way to establish this is to prove that the total population $N(t)$ is bounded. The only condition for this property to be established depends on the initial condition $N(0) > 0$.

Theorem 1. Suppose that $N(0) > 0$. Then all solutions of the system (2.1) are bounded above.

Proof. Since the total number of population is $N(t) = S(t) + I(t) + Q(t)$, by adding up equations of (2.1), we have

\[
\frac{dN(t)}{dt} = B - \mu S(t) - (\alpha + \mu) I(t) - (\alpha_q + \mu) Q(t) \\
= B - \mu N(t) - (\alpha + \mu) I(t) - (\alpha_q + \mu) Q(t) \\
\leq B - \mu N(t),
\]

hence,

\[
N(t) \leq N(0)e^{-\mu t} + \frac{B}{\mu}(1 - e^{-\mu t}) \\
\leq N(0) + B/\mu.
\]

Then $N(t)$ is bounded above and therefore all solutions $S(t), I(t)$ and $Q(t)$ are bounded and we can choose the invariant set $\Omega$ to be

\[
\Omega = (0, \mathcal{N}) \times (0, \mathcal{N}) \times (0, \mathcal{N}),
\]

where $\mathcal{N} = N(0) + B/\mu$. □

3.2. Linear stability of the disease-free equilibrium

Obviously, the system (2.1) has a disease-free equilibrium in which the $I$ and $Q$ variables are all zero while $S = S^0$, where
$S^0 = B/\mu$. We study the linear stability of this equilibrium by linearising the system about it. We introduce small perturbations (the tilde quantities) defined by

$$S = S^0 + \tilde{S}, \quad I = 0 + \tilde{I}, \quad Q = 0 + \tilde{Q}.$$  

First, for the case of no disease is present in the population, that is $I = 0$, and $Q = 0$, then $S(t)$ equation of the system (2.1) is linearized as follows:

$$\tilde{S}'(t) = B - \mu \left( S^0 + \tilde{S}(t) \right) = -\tilde{S}(t).$$

Then $\tilde{S}(t) \to 0$ as $t \to \infty$ and therefore $S(t) \to S^0$ as $t \to \infty$. Then the disease-free equilibrium is locally asymptotically stable.

Now, we introduce a general small perturbations (including small introductions of disease). Then the system (2.1) yields

$$\tilde{S}'(t) = B - \beta \left( \frac{(S^0 + \tilde{S}(t)) \tilde{I}(t)}{(S^0 + \tilde{S}(t)) + \tilde{I}(t) + \tilde{Q}(t)} \right) - \mu (S^0 + \tilde{S}(t)),$$

$$\tilde{I}'(t) = \beta \left( \frac{(S^0 + \tilde{S}(t)) \tilde{I}(t)}{(S^0 + \tilde{S}(t)) + \tilde{I}(t) + \tilde{Q}(t)} \right) - (\delta + \mu_e + \mu) \tilde{I}(t),$$

$$\tilde{Q}'(t) = \delta \tilde{I}(t) - (\eta + \mu_e + \mu) \tilde{Q}(t).$$

The linearization of the fractional term $\frac{S(t)}{N(t)}$ is dealt with as follows:

$$\frac{S(t)I(t)}{N(t)} = \frac{S^0}{S^0} \tilde{I}(t) \approx \tilde{I}(t) \left( 1 + \frac{1}{S^0} \left( \tilde{S}(t) + \tilde{I}(t) + \tilde{Q}(t) \right) \right)^{-1} \approx \tilde{I}(t) \left( 1 - \frac{1}{S^0} \left( \tilde{S}(t) + \tilde{I}(t) + \tilde{Q}(t) \right) \right)^{-1} \approx \tilde{I}(t).$$

keeping only linear terms. Therefore, at the disease-free equilibrium, the linearised system determining the small variables $\tilde{S}, \tilde{I}$, and $\tilde{Q}$

$$\tilde{S}'(t) = -\frac{\beta}{S^0} \tilde{I}(t) - \mu \tilde{S}(t),$$

$$\tilde{I}'(t) = \frac{\beta}{S^0} \tilde{I}(t) - (\delta + \mu_e + \mu) \tilde{I}(t),$$

$$\tilde{Q}'(t) = \delta \tilde{I}(t) - (\eta + \mu_e + \mu) \tilde{Q}(t).$$

With the trial solution $\left( \tilde{S}(t), \tilde{I}(t), \tilde{Q}(t) \right) = e^{it}(c_1, c_2, c_3)$ we have

$$\lambda c_1 e^{it} = -\frac{\beta}{S^0} c_2 e^{it} - \mu c_1 e^{it},$$

$$\lambda c_2 e^{it} = \frac{\beta}{S^0} c_2 e^{it} - (\delta + \mu_e + \mu) c_2 e^{it},$$

$$\lambda c_3 e^{it} = \delta c_2 e^{it} - (\eta + \mu_e + \mu) c_1 e^{it}.$$

So

$$\begin{pmatrix} \lambda + \mu & \beta c_2 e^{it} & 0 \\ 0 & \lambda - \beta + \delta + \mu_e + \mu & 0 \\ 0 & -\delta & \lambda + \eta + \mu_e + \mu \end{pmatrix} \begin{pmatrix} c_1 \\ c_2 \\ c_3 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}.$$  

Seeking non-trivial solutions for $(c_1, c_2, c_3)$, we find that $\lambda$ must satisfy the characteristic equation

$$(\lambda + \mu)(\lambda - \beta + \delta + \mu_e + \mu)(\lambda + \eta + \mu_e + \mu) = 0.$$  

Therefore, the disease-free equilibrium is locally asymptotically stable if $\beta < \delta + \mu_e + \mu$.

Then we may denote the basic reproduction number as $R_0 = \frac{\beta}{\delta + \mu_e + \mu}$. (3.4)

Now we can state the following Theorem.

Theorem 2. If $R_0 < 1$ where $R_0$ is defined by (3.4), then the disease free equilibrium $(S^0, 0, 0)$ is locally asymptotically stable to perturbations involving small introductions of disease.

3.3. The endemic equilibrium

If $R_0 > 1$ the disease-free equilibrium is no longer stable and the system (2.1) has an endemic equilibrium in which

$$\begin{pmatrix} \lambda + \mu & \beta c_2 e^{it} & 0 \\ 0 & \lambda - \beta + \delta + \mu_e + \mu & 0 \\ 0 & -\delta & \lambda + \eta + \mu_e + \mu \end{pmatrix} \begin{pmatrix} c_1 \\ c_2 \\ c_3 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}.$$  

4. Numerical simulations

In this section, we solve the system (2.1) numerically using Matlab with parameter values calculated based on the data of the Ministry of Health in Saudi Arabia [23], as illustrated in Table 1. The initial conditions are $S(0) = B/\mu = 3.4697e + 07$ (the approximate total population of Saudi Arabia), $I(0) = 1$, and $Q(0) = 0$. Based on the parameter values that reflect the current state of COVID19 in Saudi Arabia at
Table 1  Table of parameters.

| $S^0$ | $B$  | $\mu$ | $\beta$ | $\gamma$ | $\xi_q$ | $\mu_c$ |
|-------|------|-------|-------|--------|-------|--------|
| 3.4e+6 | 1603 | 4.62e−5 | 0.35   | 0.014  | 0.14  | 0.012  |

Fig. 1  Solution curve of $I(t)$ of system (2.1) where $R_0 = 3.6$ and the mean time to quarantine infectious patients is 14 days, $\delta = 0.07$. In this case the analytical value of $I^*$ is $I^* = 1.6685e + 4$ which matches the numerical value obtained from the numerical solutions.

Fig. 2  Solution curve of $I(t)$ of system (2.1) where $R_0 = 2.1$ and the mean time to quarantine infectious patients is 7 days, $\delta = 0.14$. In this case the analytical value of $I^*$ is $I^* = 9.6493e + 3$ which matches the numerical value obtained from the numerical solutions.
the time of this research (March 2020), the value of $R_0$ is larger than one. So we expect the solutions to tend to an endemic steady state. Due to the large scale of the total population $N$, this endemic steady state is hardly noticeable in the solution curve of $I(t)$ equation. Therefore, we will point this value out in the solution curve as shown in Figs. 1–3. The numerical value of the endemic steady state of the $I$ component we obtained from the numerical solution agrees with the analytical value calculated from $I$ formula in (3.5) using the same parameter values. We confirm this consistency using three different values of the parameter $\delta$, the quarantine rate.

We vary the quarantine rate $\delta$ to study the effect of the prompt quarantine on the new cases of infection. Since the value of $1/\delta$ denotes the mean time to admit new cases of infect-

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**Fig. 3** Solution curve of $I(t)$ of system (2.1) where $R_0 = 1.5$ and the mean time to quarantine infectious patients is 5 days, $\delta = 0.2$. In this case the analytical value of $I$ is $I = 7.0854e + 3$ which matches the numerical value obtained from the numerical solutions.

**Fig. 4** The mean time to quarantine an infectious patient is 14, 7, and 5 days, with $\delta = 0.07, 0.14$, and 0.2, respectively. Noticeably, the best timing to control the spread of the COVID19 is to quarantine patients with an average of five days.
tious patients into quarantine, we evaluate \( \delta \) at 14.7 and 5 days, that is, \( \delta = 0.07, 0.14 \) and 0.2, respectively. The solution curves of the new infectious patients \( I(t) \) and the quarantined patients \( Q(t) \) equations are plotted for the three values of \( \delta \) in Fig. 4. By varying \( \delta \) and considering the three cases of the mean time to detect and quarantine patients, we notice the importance of the early detection and instant quarantine of infected patients. The analysis shows that to control the COVID-19 infection in the population, the best time to admit reported patients is a mean of five days from the time of infection. The outcomes of this study were presented to the Public Health Authority of Saudi Arabia in April 2020 [24]. We recommended that the Authority expands the COVID-19 testing centers and enlarge the scale of mass testing, especially in the highly crowded communities. These recommendations were approved and applied promptly by the Ministry of Health in Saudi Arabia [21].

5. Discussion and conclusions

Even though the Kermack-McKendrick model is a well-known mathematical model and lots of modifications have been done to improve it to fit different diseases, there is still more that can be done. In our model, we only aimed to measure the effect of quarantine on the COVID-19 dynamics in the population of Saudi Arabia. Therefore we kept the model simple and incorporate the compartment of quarantine patients on an SI model. As predicted, the sooner the infected cases were detected and isolated, the more control on the COVID-19 spread in the population. From the formula of \( R_0 \), one can conclude that the key factor parameters to decrease the value of \( R_0 \) in order to reduce the infection cases and control the disease are \( \beta, \delta, x, \mu \), and \( \mu_c \). However, not all these parameters are easy to control or ethical to increase. More precisely, the mortality rate due to COVID-19 \( \mu_c \) appears in the denominator, meaning that increasing this rate will reduce the \( R_0 \) and that will cause the COVID-19 to be eradicated from the population, but we do not wish for people to die.

On the other hand, the recovery rate parameter \( x \) can not be increased because it depends on individuals’ immunity and their reaction to the disease. For COVID-19, this rate is considerably high compared to the seasonal Influenza, but the spread of COVID-19 is as fast. The reason for this rapid and large infection scale is the high rate of infection, \( \beta \) in our model. Reducing the infection rate \( \beta \) can be done by applying social distancing and frequent hygienic washing of hands and surfaces although these applications are considered limited and can only reduce the rate to a certain level. Households can not apply these measures in living spaces where the probability of getting an infection is the highest. For all those reasons, we concluded that the most applicable procedure to imply is to increase the quarantine rate \( \delta \) by expanding COVID-19 test centers and enlarge the scale of mass testing to discover the new cases as soon as possible. The Ministry of Health instantly applied these recommendations in Saudi Arabia.

6. Future plans

Many modifications can be done to improve the COVID-19 modeling and consider more detailed aspects like age vulnerability in which we are working on in a separate paper by considering an age-structure model. Moreover, vaccination’s effect on the dynamics has an essential effect on eradicating COVID-19.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgement

The authors extend their appreciation to the Deanship of Scientific Research at University of Bisha Saudi Arabia for funding this work through COVID-19 Initiative Project under Grant No. (UB-COVID-133 –1441).

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