From SARS-CoV and MERS-CoV to COVID-19
Pandemic: Challenges in Modeling the Transmission
Dynamics in South Africa

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Abstract
Two of the members (viz. SARS-CoV and MERS-CoV) of the coronavirus family are compared with the novel coronavirus SARS-CoV-2 for their clinical and nonclinical similarities and differences. This information is used to design a new mathematical model for the transmission dynamics of Coronavirus disease (COVID-19). It is an extension of the standard SEIR model, modified by the incorporation of four classes of individuals, which are differentiated by their infection status and severity. The model is also enriched by adding the class of contaminated environment to account for the indirect transmission. The associated reproduction number of the COVID-19 outbreak in South Africa is computed. From a good fit of the cumulative number of daily reported cases with the system plot, the model is used to assess the most effective measures in controlling the disease in South Africa. The impact of non-pharmaceutical interventions is assessed via a threshold analysis approach. More precisely, the social-distancing effectiveness parameter, the quarantine parameter, the lockdown starting time parameter, the attack rate of the pandemic, and the environmental infection coefficient are introduced and used to estimate the cumulative epidemic trajectories and future daily incidence for South Africa. Analysis of the model demonstrates that COVID-19 can be controlled effectively by reducing contact between individuals via social-distancing, monitoring close contacts, self-isolation and quarantining of suspected exposed individuals. Our findings suggest that early implementation of these measures and maintaining them for a sufficiently long period of time

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play an important role in mitigating the transmission. For instance, it is shown that implementing the lockdown measure one week later than the effective date of 26 March 2020 when it was done in South Africa would delay significantly the predicted date of mid July 2020 for peak time of the infection, and the currently predicted reduction of 39% in terms of cumulative numbers of cases and deaths would be lost. Similarly, it is shown that early quarantine of suspected individuals led to a 78% decrease in the number of symptomatic cases. Our findings also suggest that effective implementation of massive and quality testing as well as of contact tracing can contribute to address the challenge posed by the big unknown number of asymptomatic individuals. Furthermore, our predictions in terms of infection peak time and corresponding number of cases are consistent with those of South Africa’s government.

**Keywords:** Coronavirus, Social-distancing, Isolation, Quarantine, COVID-19, Control reproduction number.

# Introduction

A novel Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) was identified in January 2020 as the cause of an outbreak of a viral pneumonia in Wuhan (capital of Hubei, China). The disease, later named Coronavirus Disease 2019 (COVID-19), quickly spread to more than 188 countries across all regions causing in excess of 7.8 million infections with more than 430,000 deaths globally (as at 14 June 2020) [10, 59]. SARS-CoV-2 that causes this highly contagious infectious disease is closely related to the SARS virus. There are many different types of coronaviruses. Most of them circulate among animals. However, 7 types of coronavirus are known to jump from animals to humans (spillover) and thus causing illness in humans. Three of the seven human coronavirus infections can be much more severe (with mild to upper respiratory track illness that causes symptoms of the common cold) and have recently caused major outbreaks of deadly pneumonia [54]. These are:

(1) SARS-CoV, identified in 2002 as the cause of an outbreak of Severe Acute Respiratory Syndrome (SARS), that has flu-like symptoms.

(2) MERS-CoV, identified in 2012 as the cause of Middle East Respiratory Syndrome (MERS), that has flu-like symptoms.

(3) SARS-CoV-2, identified in late 2019 as the cause of an outbreak of Coronavirus 2019 (COVID-19), an acute respiratory illness that can be severe.

Studies in the literature suggest that SARS-CoV and MERS-CoV originated in bats, with further both circulating in civet cats and camels, respectively. The reservoir
Table 1: Most relevant clinical and nonclinical similarities and differences between SARS-CoV, MERS-CoV and SARS-CoV-2.

<table>
<thead>
<tr>
<th>Similarties &amp; differences</th>
<th>Characteristics</th>
<th>SARS-CoV</th>
<th>MERS-CoV</th>
<th>SARS-CoV-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Target receptors</td>
<td>ACE-2</td>
<td>ACE-2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N protein</td>
<td>IFN-λ inhibitor</td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>Ground glass</td>
<td>Bilateral, multilobar ground glass opacities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest CT scan</td>
<td>Lobar consolidation</td>
<td>Nodular opacities</td>
<td>No nodular opacities</td>
<td></td>
</tr>
<tr>
<td>Transmission</td>
<td>Contact with infected individual</td>
<td>Contact with infected individual</td>
<td>Contact with infected individual</td>
<td></td>
</tr>
<tr>
<td>$R_0$</td>
<td>0.4</td>
<td>3</td>
<td>1.4-2.5</td>
<td></td>
</tr>
<tr>
<td>Case fatality rate</td>
<td>9.6%</td>
<td>35%</td>
<td>(2.3%) (Now: 6.74%)</td>
<td></td>
</tr>
<tr>
<td>Prevention</td>
<td>Hand hygiene, cough etiquette</td>
<td>Hand hygiene</td>
<td>Hand hygiene cough etiquette</td>
<td></td>
</tr>
<tr>
<td>Animal reservoir</td>
<td>Bats and civet cats</td>
<td>Bats and camels</td>
<td>Unknown</td>
<td></td>
</tr>
<tr>
<td>Number infected cases</td>
<td>26 Countries</td>
<td>27 Countries</td>
<td>188 Countries</td>
<td></td>
</tr>
<tr>
<td>Symptoms</td>
<td>flu-like coughing &amp; fever</td>
<td>flu-like coughing &amp; fever</td>
<td>shortness of breath coughing &amp; fever</td>
<td></td>
</tr>
</tbody>
</table>

for SARS-CoV-2 is not known yet. In fact a lot is known about the dynamics of SARS-CoV and MERS-CoV, which contributed to contain and control the associated disease. On the contrary, the major challenge is that the knowledge we currently have on SARS-CoV-2 is scarce and not clinically proven. In the references [9, 38, 54], relevant similarities and differences are provided between SARS-CoV, MERS-CoV and SARS-CoV-2. For convenience, we give Table 1 which has more comparative facts than the table in [9] that deals with clinical similarities and differences.

However, it is believed that the novel SARS-CoV-2 is a highly diffusible virus, spread by droplets, via direct and indirect transmissions as follows: (a) direct contact with infectious individuals and (b) indirect contact with contaminated objects. In the current situation of absence of a rapid diagnostic test, vaccine or specific antiviral treatment, the main control measures against the infection are social-distancing, standard hygiene practice (e.g. using an alcohol based sanitizers, washing hands often with soap), wearing a face mask, quarantine and isolation of individuals feared exposed to or diagnosed with coronavirus [21, 37, 55, 59].

The established clinical symptoms of COVID-19 include fever, cough, shortness of breath, acute pneumonia, expectoration, hemoptysis often followed by renal failure.
The incubation period was estimated to be 5.2-5.5 days \[19\] and the serial interval (the time between the successive onset of symptoms in a chain of transmission) was 7.6 days \[3, 27, 43, 59\]. Since the flu season is coming in South Africa, there would be more confusion with the seasonal flu symptoms. The scale of COVID-19 is much higher and beyond expectation as compared to SARS and MERS diseases. The World Health Organization is concerned, among others, with the gaps in understanding the degree of transmissibility between people, possibility of ”super-spreaders” and potential for sustainable person-to-person transmission and spread \[58, 59\]. The super-spreaders are those who transmit the virus to more than 20 patients and have underlying respiratory diseases with a severe cough.

Mathematical modeling can be a useful tool for designing strategies to control rapidly spreading infectious diseases in the absence of an effective treatment, vaccine or diagnostic test. Such modeling has been used to assess the epidemic potential and control of SARS in Hong Kong \[13, 32\], Singapore \[13, 34\], Beijing \[57\], China \[65\] and Middle East \[55, 62\]. In the same vein, another deterministic model was designed to analyze the MERS-CoV outbreak in the Republic of Korea \[32, 59\]. The impact of the timing of control measures associated with a reduction of the transmission rate and diagnostic delays on the outbreak size and duration was assessed. The numerical analysis reveals that lack of self-protection sense and targeted control measures were the reasons of the outbreak spread quickly. However, it was reported that strengthening self-protection ability of susceptible and quickly isolating or monitoring close contacts are effective measures to control the disease \[59\]. Furthermore, partial correlation analysis shows that the infectivity and proportion of the asymptomatic infected cases have much influence on the disease spread.

With the outbreak of the COVID-19 pandemic, there has been a surge of mathematical modeling manuscripts and flattening the curve of the infection has become a popular expression among administrators, see for instance SIAM Blog \[47\] and the works in \[5, 20, 28, 29, 36, 48, 61\]. In particular, the following papers can be mentioned: Muzimoto and Chowell \[36\] designed a mathematical model to study the changes in COVID-19 transmission potential in the diamond princess as the outbreak progressed; Ferguson et al. \[20\] used agent-based model to investigate the impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand; Eikenberry et al. \[19\] proposed mathematics model to assess the potential impact of face masks use by the general public to curtail the COVID-19 pandemic; Ngonghala et al. \[41\] presented a mathematical model to assess the potential impact of non-pharmaceutical interventions on curtailing the 2019 novel Coronavirus.
Over the past two months COVID-19 cases and deaths have been increasing in a worrying manner in South Africa, viz. [15]: 48,285 cumulative total confirmed cases and 2.07% fatalities (on 5 June 2020) versus 1,380 cases and 0.37% fatalities (on 1 April 2020). The breakdown is particularly alarming for the Western Cape, Gauteng and Eastern Cape provinces that have been classified as hotspots or epicenters of the COVID-19. Hence this study. We construct a deterministic model for the transmission dynamics of COVID-19 pandemic in South Africa, taking into account the protocols and the guidelines for quarantine and isolation from COVID-19 exposure and infection [52], as designed and followed by the Government and health authorities. We also try to align our work to South Africa’s COVID-19 Modeling Consortium [50]. Hence our model is designed to assess the impacts of control measures such as social-distancing, quarantine and isolation strategies against the disease.

The remaining part of the paper is organized as follows. The model formulation together with the motivation and the manner in which the model fits in the literature are provided in Section 2 titled "methods". Section 3 is devoted to "materials and results" including estimation of the parameters and some analytical results such as the stability of the disease-free equilibrium set and the final size relation of the COVID-19. Section 4 provides numerical simulations, analyses and discussions regarding the the epidemiological dynamics of the infection and the impact of available control measures. Finally, conclusions are provided in Section 5.

2 Method

2.1 Model formulation

The total human population, $N(t)$, at time $t$ is divided into seven mutually exclusive compartments: Susceptible ($S(t)$), Exposed ($E(t)$), Asymptomatic ($A(t)$), Symptomatic infectious ($I(t)$), Quarantine ($Q(t)$), Isolated ($J(t)$) and Recovered individuals ($R(t)$). Thus

$$N(t) = S(t) + E(t) + A(t) + I(t) + Q(t) + J(t) + R(t).$$

This division of the total population aligns with South Africa’s guidelines for quarantine and isolation from COVID-19 exposure and infection [52]. In this context, it is worthwhile specifying the following regarding $Q$ and $J$ classes. Quarantine currently applies to [52]:

- An individual or group of persons who were in close contact with a person infected with coronavirus;

- Persons at high risk of having been exposed during international travel; and
• Symptomatic persons who have been identified as requiring testing or who have tested, but are awaiting test results. These persons can be discharged if they test negative.

Note that the compartment $Q$ includes mandatory quarantine in designated quarantine sites and self-quarantine at home subject to specific criteria. In any case, persons in quarantine are monitored regularly and transferred accordingly, as their infection status becomes clear. Though isolation is reserved for persons who are already sick and/or have tested positive for COVID-19 infections, it may in the context of the COVID-19 pandemic, include [52]:

• Isolation at a personal home known as self-isolation. This is the preferred option, subject to the person meeting the self-isolation criteria.

• Isolation in a health facility or at a designated isolation facility. People who cannot self-isolate at home should be considered for admission to such a facility.

As far as the compartments of human individuals are concerned, we use an extension of the standard $SEIR$ model modified by the incorporation of the $A$, $I$, $Q$ and $J$ classes differentiated by individuals’ infection status and severity. However, due to the clinical findings [59] and other works [11, 18, 51] which suggest that COVID-19 can be transmitted indirectly following contact with contaminated objects, surfaces and places often touched, we introduce a separate compartment of contaminated environment $P(t)$.

The population of susceptible individuals is decreased following infection with COVID-19 which can be acquired at the rate (force of infection)

$$\lambda = \frac{\beta(t)(\eta_1 A + \eta_2 Q + I + \eta_3 J)}{N} + \beta(t)\eta_4 P$$

in which the first term expresses direct transmission while the second reflects the indirect transmission. The relevance of the class $P$ of contaminated environment, which is the essence of the campaign to disinfect objects, surfaces, etc., is further strengthened by recent studies that show that infected patients shed SARS-CoV-2 in their stool (see for instance [11]). Here the parameter $\beta(t)$ is the time-dependent effective contact rate (contact, per person per unit time, capable of leading to COVID-19 infection). Due to the introduction of social-distancing policy (e.g. lockdown or stay at home), it is reasonable to assume that the contact rate will be a decreasing function of time. Unlike the exponential decay as in [19], we consider the following less fast decaying function, which is continuous:

$$\beta(t) = \begin{cases} 
\beta_0 & \text{if } t \in [0, \tau_0], \\
\beta_1 + \frac{\beta_0 - \beta_1}{1 + \omega(t - \tau_0)} & \text{with } \beta_1 << \beta_0 \text{ if } t \geq \tau_0, 
\end{cases}$$

(1)
where \( \tau_0 \) is the lockdown time. The parameter \( \omega > 0 \) is a measure of the compliance of the population with the interventions, mostly the social-distancing (and also the wearing of face-masks that has been introduced from 1 May 2020 in South Africa). The larger \( \omega \) is, the faster the contact rate \( \beta(t) \) decays to \( \beta_1 \), which represents the desired contact rate for COVID-19 to be controlled. Figure 1 illustrates the behaviour of the function \( \beta(t) \) for different values of \( \omega \).

Figure 1: The effective contact rate function with different values of the measure of compliance \( \omega \). Higher compliance with interventions results in faster decay of the contact rate.

Furthermore, the parameters \( 0 \leq \eta_1 \leq 1, 0 \leq \eta_2 \leq 1, 0 \leq \eta_3 \leq 1 \) and \( 0 \leq \eta_4 \leq 1 \) are modification parameters accounting for the assumed reduction in infectiousness of individuals in the asymptomatic (A), quarantined (Q), isolated (J) classes and contaminated environment (P), in comparison to infectious individuals in I class. Combining all the above facts, the rate of change of the susceptible population is given by

\[
\frac{dS}{dt} = -\lambda S.
\]

The population of individuals exposed to COVID-19 is increased by infection (at the rate \( \lambda \)). This population is decreased by the infection status of individuals (at a rate \( \sigma \) to be split into \((1-r)\sigma\) for asymptomatic and \(r\sigma\) for quarantined), so that

\[
\frac{dE}{dt} = \lambda S - \sigma E.
\]
The population of asymptomatic infectious individuals is increased by the progression of infected individual from the exposed class (at the rate $(1 - r)\sigma$), and it decreases by progression to symptomatic infectious class (at a rate $\gamma_3$). This gives

$$\frac{dA}{dt} = (1 - r)\sigma E - \gamma_3 A.$$  

The population of quarantined individuals increases, following the quarantine of individuals in the exposed class (at the rate $r\sigma$). Being carefully monitored, this population is decreased by testing of individuals with clinical symptoms (at a rate $\gamma_1$), isolation (at a rate $\gamma_2$) and recovery (at a rate $\psi$), so that

$$\frac{dQ}{dt} = r\sigma E - (\gamma_1 + \gamma_2 + \psi)Q.$$  

The parameter $r$ is the quarantine probability. Note that South African citizens repatriated from abroad during the lockdown are placed in quarantine, as per the Government’s guidelines [52]. Furthermore, the norm for COVID-19 is to move quarantined individuals tested with no clinical symptoms of the disease to the recovery class (at rate $\psi$).

The population of symptomatic infectious individuals (with clinical symptoms of COVID-19) in $I$ class increases, following the development of clinical symptoms by individuals in asymptomatic class (at the rate $\gamma_3$) and by quarantined individuals tested with clinical symptoms of the disease (at a rate $\gamma_1$). This population is decreased by isolation (at a rate $\kappa_1$), recovery (at a rate $\tau_1$), and COVID-19 induced mortality (at a rate $\delta_1$); this gives

$$\frac{dI}{dt} = \gamma_1 Q + \gamma_3 A - (\kappa_1 + \tau_1 + \delta_1)I.$$  

The population of individuals that are isolated or hospitalized, ($J(t)$), is generated by the isolation of infectious individuals with clinical symptoms of COVID-19 (at the rate $\kappa_1$) and isolation of quarantined individuals tested with clinical symptoms of the disease (at the rate $\gamma_2$). It is decreased by recovery (at a rate $\tau_2$) and disease induced death (at a rate $\delta_2$). Hence

$$\frac{dJ}{dt} = \kappa_1 I + \gamma_2 Q - (\tau_2 + \delta_2)J.$$  

The recovered population is generated by the recovery of individuals in $I$, $J$ and $Q$ classes (at the rates $\tau_1$, $\tau_2$ and $\psi$), respectively. This gives

$$\frac{dR}{dt} = \tau_1 I + \tau_2 J + \psi Q.$$  

The environmental contamination occurs by the activities of the individuals in $A$, $Q$, $I$ and $J$ classes on the environment at the rates $\xi_1$, $\xi_2$, $\xi_3$ and $\xi_4$, respectively. The virus
is decreased or cleared at the rate υ. Thus

\[ \frac{dP}{dt} = \xi_1 A + \xi_2 Q + \xi_3 I + \xi_4 J - \nu P. \]

In summary, the COVID-19 transmission model is given by the following system of nonlinear differential equations associated with the flow diagram in Figure 2 and the parameters described in Table 2.

\[ \begin{align*}
\frac{dS}{dt} &= -\lambda S, \\
\frac{dE}{dt} &= \lambda S - \sigma E, \\
\frac{dA}{dt} &= (1 - r)\sigma E - \gamma_3 A, \\
\frac{dQ}{dt} &= r\sigma E - (\gamma_1 + \gamma_2 + \psi)Q, \\
\frac{dI}{dt} &= \gamma_1 Q + \gamma_3 A - (\kappa_1 + \tau_1 + \delta_1)I, \\
\frac{dJ}{dt} &= \kappa_1 I + \gamma_2 Q - (\tau_2 + \delta_2)J, \\
\frac{dR}{dt} &= \tau_1 I + \tau_2 J + \psi Q, \\
\frac{dP}{dt} &= \xi_1 A + \xi_2 Q + \xi_3 I + \xi_4 J - \nu P.
\end{align*} \]

The system is appended with nonnegative initial conditions \( S(0) \geq 0, \ A(0) \geq 0, \ I(0) \geq 0, \ Q(0) \geq 0, \ J(0) \geq 0, \ R(0) \geq 0, \ P(0) \geq 0 \).

Adding the equations in model (2) that describe the evolution of humans yields the following conservation law:

\[ \frac{dN}{dt} = -\delta_1 I - \delta_2 J. \]
Figure 2: Flow diagram of the COVID-19 model

Table 2: Description of parameters of model (2)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\psi$</td>
<td>Removal rate from quarantined</td>
</tr>
<tr>
<td>$\beta$</td>
<td>Time-dependent effective contact rate</td>
</tr>
<tr>
<td>$\beta_0$</td>
<td>Effective contact rate before the lockdown</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>Targeted effective contact rate for disease control</td>
</tr>
<tr>
<td>$\omega$</td>
<td>Measure of social-distancing effectiveness</td>
</tr>
<tr>
<td>$\kappa_1$</td>
<td>Isolation rate of infectious individuals</td>
</tr>
<tr>
<td>$\eta_1, \eta_2, \eta_3$</td>
<td>Modification parameters for reduction in infectiousness of asymptomatic, quarantined &amp; isolated individuals in comparison to infectious individuals</td>
</tr>
<tr>
<td>$\eta_4$</td>
<td>Modification parameters for reduction in infectiousness of contaminated environment in comparison to infectious individuals</td>
</tr>
<tr>
<td>$\tau_0$</td>
<td>Starting day of lockdown</td>
</tr>
<tr>
<td>$\tau_1, \tau_2$</td>
<td>Recovery rates of symptomatic infectious and isolated individuals, respectively</td>
</tr>
<tr>
<td>$(1 - r)\sigma; r\sigma$</td>
<td>Progression rate of exposed individuals to asymptomatic infectious and quarantined classes, respectively</td>
</tr>
<tr>
<td>$r$</td>
<td>Probability of quarantine</td>
</tr>
<tr>
<td>$\gamma_1; \gamma_2$</td>
<td>Progression rate of quarantine individuals to infectious and Isolated classes</td>
</tr>
<tr>
<td>$\delta_1, \delta_2$</td>
<td>Disease-induced death rates for infectious and isolated individuals, respectively</td>
</tr>
<tr>
<td>$\xi_1, \xi_2, \xi_3, \xi_4$</td>
<td>Contamination rates of environment by infected individuals</td>
</tr>
<tr>
<td>$\nu$</td>
<td>Decay or clear rate of the virus on the environment</td>
</tr>
</tbody>
</table>
2.2 The new model in the literature

The proposed model extends some of the models on SARS and MERS in the literature as outlined below. We also indicate how our model fits in the current effort of modeling COVID-19.

(i) Unlike [12, 26, 40] who have incorporated demographic dynamics (e.g. birth and natural death rates) in their COVID-19 models, we did not do it. This is due to the fact that COVID-19 is a new disease, which is still growing. It is not clear if the disease is endemic and in fact there are many unknowns about this disease. Thus our focus is to understand the epidemiological dynamics of the disease.

(ii) In the SEIR-type model investigated in [32], there were only two sources of infection, namely the infectious and the isolated individuals. Here, we consider five sources including the contaminated environment, which is neither considered in [21, 55] nor in the papers [19, 41, 53] and [20] that deal with COVID-19. However, contaminated environment is considered in the recent paper [63], which is restricted to two sources of infection of COVID-19.

(iii) Unlike [23, 55] where the mass action formulation is considered throughout, we combined in our model both the standard incidence for human carriers of infection and the mass action principle for contaminated environment.

(iv) For the SARS model in [21], quarantined individuals are asymptomatic infective who develop symptoms and then move to the isolated class. However in our model, which follows the national protocol [52], some quarantined individuals develop symptoms and are transferred to infectious class, while others recovered by either treatment or acquiring natural immunity.

(v) The model assumes isolation of quarantined individuals with clinical symptoms of coronavirus. This was not considered in [55].

(vi) In a number of extended SEIR models for COVID-19 [41, 53, 64], the approach is to consider models with two or multiple groups depending on risk, age, quarantine or non-quarantine, etc., where each sub-population follows the same transition flows between compartments. The entire South African population being relatively young, and not affected as such by the elderly people-based risk criteria that is used in Europe and North America, we did not consider this approach.

3 Materials and Results

To the question ‘will COVID-19 ever disappear’, several sources suggest that the scenario of its complete disappearance is highly unlikely; ‘it just transmits too easily in the
human population’. As a matter of fact, China and some other countries are worried about second bad waves and new outbreaks. One of WHO’s top experts thinks that we might get into a period of cyclical waves or end up with low level endemic disease that we have to deal with (see [22]). The major challenge of suppression of COVID-19 in the absence of vaccine is also echoed in [20]. The above comment suggests that, like [63], we could embark into the full qualitative and quantitative analysis of the model. However, this will be done elsewhere in due course when more is known about the disease. At present, our focus is on the epidemiological dynamics of the infection. This is aligned with the current priority and strategy of the South African Government of mitigation, which focuses on slowing but not necessarily stopping the epidemic spread, in order to prepare ourselves and be ready with the infrastructure and facility needed by our hospitals and ICUs when the worse case scenario comes. We will fit the model using South African data and predict the evolution of the epidemic. To this end, we start the next subsection by estimating the epidemiological parameters of the model (2) relevant to COVID-19 data for South Africa obtained from the Johns Hopkins Medical University coronavirus resources center [10].

3.1 Estimation of parameters and model fitting

The National Institute for Communicable Diseases (NICD) confirmed South Africa’s first proven case of COVID-19 on Thursday 5 March 2020, which we consider to be the first day of the disease. A national state of disaster was declared on 15 March 2020, followed by a nationwide lockdown from 26 March 2020. We therefore take the time when the lockdown started to be \( \tau_0 = 25 \).

We assume the effective contact rate \( \beta = \beta_0 \) before the lockdown to be 0.40 (fitted). The desired minimum contact rate \( \beta \) to which the contact rate should decay is assumed to be \( \beta = \beta_1 = 0.155 \) (so as to achieve the target of bringing the control reproduction number to a value below unity: \( R_0 = 0.9697 < 1 \)). Following the study in the literature, which shows that the incubation period for COVID-19 ranges from 5-6 days [41], with about 70% of exposed individuals becoming infected, we assume the rate at which individuals exposed to COVID-19 are quarantined to be \( r \sigma = 0.4 \) per day. Similarly, we assume the rate at which exposed individuals become asymptomatically infectious to be \( (1 - r) \sigma = 0.6 \) per day. It should be noted that a challenge or big unknown in the modeling of COVID-19 is the portion of \( \sigma \) associated with the spread by asymptomatic individuals, as highlighted in [1, 26]. Since progression of individuals from asymptotically to symptomatically infectious class takes about 2-5 days [41], we set \( \gamma_3 = 1/2 \) per day. It is assumed that there is a short time period of about 7 days between the onset of disease symptoms in the quarantine class. Hence, we set the isolation rate of quarantined individuals (\( \gamma_2 \)) to be \( \gamma_2 = 1/7 \) per day. Similarly, the isolation
rate of symptomatic infected individuals ($\kappa_1$) is assumed to be $\kappa_1 = 1/7$ per day. Following [20], the progression rate of quarantined individuals to symptomatic class ($\gamma_1$) is assumed to be $\gamma_1 = 1/7$ per day. The National Institute of Communicable Diseases, South Africa [37], estimated the infection period for COVID-19 to range from 6-14 days, so we set the rates at which infectious and isolated individuals recover from COVID-19 ($\tau_1$ and $\tau_2$) to be $\tau_1 = 1/6$ per day and $\tau_2 = 1/10$ per day, respectively.

While some studies assumed the modification parameters ($\eta_1$ and $\eta_2$) for the relative infectiousness of asymptomatically infectious individuals in comparison to symptomatically infectious individuals to be $\eta_1 = \eta_2 = 0.5$ per day [20, 41], other studies [33] estimated the parameter to be in the range [0.42, 0.55]. Hence, we set modification parameters $\eta_1$, $\eta_2$, $\eta_3$ to be $\eta_1 = \eta_2 = \eta_3 = 0.5$ and $\eta_4$ to be in the range [0, 0.1]. Since data suggests that the COVID-19 case fatality rate in South Africa is about 0.04% [37], we assume the COVID-19 induced death rates ($\delta_1$ and $\delta_2$) to be $\delta_1 = \delta_2 = 0.0425$. Following [41], we set the proportion at which quarantined individuals recovered to be $\psi = 0.6$.

Contaminated environment is reported to be a substantial route for the transmission of SARS-CoV-2. This is the essence of the campaign to disinfect surfaces, buttons, hands, knobs and other places touched often, apart from scientific reports such as [11, 18, 51]. In fact, in South Africa, a number of hospitals have been closed and the scaling down of nationwide lockdown from level 5 to level 4 (recently to level 3) is subjected to schools and other facilities being thoroughly disinfected. Hence, the parameter $\upsilon$ is the rate at which the virus remains infective in the environment before decaying is estimated in the range [0, 1]. We assume shedding rates $\xi_1$, $\xi_2$, $\xi_3$ and $\xi_4$ of all infectious individuals to be in the range (0,0.5).

The cumulative number of disease-induced deaths denoted by $D = D(t)$ will be estimated from the following differential equation that results from recording death contributions in the model (2):

$$\frac{dD}{dt} = \delta_1 I + \delta_2 J.$$  

(4)

The values for the remaining parameters of the model, e.g. $\psi$, were estimated from the data [37]. The estimated parameters are recorded on Table 3. We now fit the model (2) using data obtained from Johns Hopkins Medical University Coronavirus Resources Center [10] for South Africa. Figure 3 shows a reasonably good fit for total confirmed cases and cumulative model predicted cases.
Table 3: Parameter values for the model (2)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Nominal value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\beta_0$</td>
<td>0.40 (0.002-0.75) per day</td>
<td>Fitted</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>0.155 (0.002-0.3) per day</td>
<td>Fitted</td>
</tr>
<tr>
<td>$\omega$</td>
<td>0.005 (0-1/7) per day</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\eta_1, \eta_2, \eta_3$</td>
<td>0.5 (0, 1)</td>
<td>[20, 33, 41]</td>
</tr>
<tr>
<td>$\eta_4$</td>
<td>0.000002[0, 1] per day</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\gamma_1, \gamma_2$</td>
<td>0.034 (0.01, 0.5) per day</td>
<td>[41]</td>
</tr>
<tr>
<td>$\gamma_3$</td>
<td>0.55 (0.01, 0.5) per day</td>
<td>[20, 41]</td>
</tr>
<tr>
<td>$\kappa_1$</td>
<td>1/14 (0, 0.5) per day</td>
<td>[41]</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>1 (0,1] per day</td>
<td>Estimated</td>
</tr>
<tr>
<td>$r$</td>
<td>0.4 (0,1) per day</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\tau_1$</td>
<td>1/6 (0,1) per day</td>
<td>[41]</td>
</tr>
<tr>
<td>$\tau_2$</td>
<td>1/10 (0,1) per day</td>
<td>[41]</td>
</tr>
<tr>
<td>$\delta_1, \delta_2$</td>
<td>0.0425 (0.01, 0.06) per day</td>
<td>[20, 41]</td>
</tr>
<tr>
<td>$\psi$</td>
<td>0.6 [0, 1]</td>
<td>[41]</td>
</tr>
<tr>
<td>$\xi_1, \xi_2, \xi_3, \xi_4$</td>
<td>(0, 0.5)</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\upsilon$</td>
<td>0.75 (0.4, 0.9)</td>
<td>Estimated</td>
</tr>
</tbody>
</table>

Figure 3: Time series plot showing a least square fit of the system (2) using South Africa COVID-19 reported cases for cumulative number of [10] infections. Parameter values used are as given in Table 3.

### 3.2 Analytical results

The task ahead of us is to compute the reproduction number. Since our model (2) includes quarantine and isolation arranged prior to the beginning of the epidemic, we call the corresponding reproduction number the control reproduction number instead
of using the classical terminology of basic reproduction number. Any point in the following manifold (line) is a disease-free equilibrium of the model (2):

\[ \mathcal{DFE} := \{(S, E, A, Q, I, J, R, P) = (S, 0, 0, 0, 0, 0, 0); 0 < S \leq N_0\}, \]  

where \(N_0\) is assumed to be very close or equal to the total population of South Africa. In what follows, we will systematically work with the largest disease-free equilibrium (DFE) point denoted by \(E_0\):

\[ E_0 = (S^*, E^*, A^*, Q^*, I^*, J^*, R^*, P^*) = (N_0, 0, 0, 0, 0, 0). \]

The linear stability of \(E_0\) can be established using the next generation operator on the system (2). We assume in this subsection that the contact rate \(\beta\) is a constant taking either the maximum value \((\beta = \beta_0)\) or the minimum value \((\beta = \beta_1)\). This assumption makes sense because in the absence of any interventions, the model (2) reduces to the one with \(\beta = \beta_0\), while for \(t\) large enough, it behaves like the model with \(\beta = \beta_1\) when all interventions are successfully implemented. The vector of appearance of new infections and that of the transfers out of and into the compartments are given by

\[
\mathcal{F} = \begin{bmatrix}
\frac{\beta S (\eta_1 A + I + \eta_2 Q + \eta_3 J)}{N} + \beta S \eta_4 P \\
0 \\
0 \\
0 \\
0 \\
0 \\
0 \\
0
\end{bmatrix}, \\
\mathcal{V} = \begin{bmatrix}
K_1 E \\
K_2 A - \sigma E \\
K_3 Q - \sigma E \\
K_4 I - \gamma_1 Q - \gamma_3 A \\
K_5 J - \gamma_2 Q - \kappa_1 I \\
v P - \xi_1 A - \xi_2 Q - \xi_3 I - \xi_4 J
\end{bmatrix},
\]

respectively, where, \(K_1 = \sigma, K_2 = \gamma_3, K_3 = \gamma_1 + \gamma_2 + \psi, K_4 = \kappa_1 + \tau_1 + \delta_1\) and \(K_5 = \tau_2 + \delta_2\).

Using the notation in [56], the matrices \(F\) and \(V\), for the new infection terms and the remaining transfer terms, are, respectively, given by

\[
\mathcal{F} = \begin{bmatrix}
0 & \beta \eta_1 & \beta \eta_2 & \beta \eta_3 & \beta \eta_4 N_0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{bmatrix}, \\
\mathcal{V} = \begin{bmatrix}
K_1 & 0 & 0 & 0 & 0 \\
-(1 - r)\sigma & K_2 & 0 & 0 & 0 \\
-\sigma & 0 & K_3 & 0 & 0 \\
0 & -\gamma_3 & -\gamma_1 & K_4 & 0 \\
0 & 0 & -\gamma_2 & -\kappa_1 & K_5 \\
0 & -\xi_1 & -\xi_2 & -\xi_3 & -\xi_4 & v
\end{bmatrix}.
\]

It follows from [56] that the control reproduction number denoted by, \(R_0\), is given by \(R_0 = \rho(FV^{-1})\), where \(\rho(.)\) is the spectral radius of the involved matrix and the inverse
of the matrix $V$ is given by

$$V^{-1} = \begin{bmatrix} K_1^{-1} & 0 & 0 & 0 & 0 & 0 \\ \frac{\sigma}{K_1 K_2} & K_2^{-1} & 0 & 0 & 0 & 0 \\ \frac{r \sigma}{K_1 K_3} & 0 & K_3^{-1} & 0 & 0 & 0 \\ U_1 & \frac{\gamma_3}{K_2 K_4} & \frac{\gamma_1}{K_3 K_4} & \frac{K_4^{-1}}{K_4 K_5} & 0 & 0 \\ U_2 & \frac{\kappa_1 \gamma_3}{K_2 K_4 K_5} & \frac{K_4 \gamma_2 + \gamma_1 \kappa_1}{K_3 K_4 K_5} & \frac{\kappa_1}{K_4 K_5} & K_5^{-1} & 0 \\ \frac{R_0^{pv}}{\beta N_0 \eta_4} & U_3 & U_4 & \frac{K_5 \xi_3 + \kappa_1 \xi_4}{K_4 K_5 v} & \frac{\xi_4}{K_5 v} & v^{-1} \end{bmatrix}$$

(6)

with $R_0^{pv}$ defined below, and

$$U_1 = \frac{K_2 \gamma_1 r \sigma + K_3 \gamma_3 r \sigma}{K_1 K_2 K_3 K_4}, \quad U_2 = \frac{K_2 K_4 \gamma_2 r \sigma + K_2 \gamma_1 \kappa_1 r \sigma + K_3 \gamma_3 \kappa_1 r \sigma}{K_1 K_2 K_3 K_4 K_5},$$

$$U_3 = \frac{K_4 K_5 \xi_1 + K_5 \gamma_3 \xi_3 + \kappa_1 \gamma_3 \xi_4}{K_2 K_4 K_5 v}, \quad U_4 = \frac{K_4 K_5 \xi_2 + K_4 \gamma_2 \xi_4 + K_5 \gamma_1 \xi_3 + \gamma_1 \kappa_1 \xi_4}{K_3 K_4 K_5 v}.$$

Simple computations show that $R_0$ can be rewritten as the sum of two main contributions (viz. humans and environment) as follows:

$$R_0 = R_0^{ph} + R_0^{pv},$$

(7)

where

$$R_0^{ph} = \frac{\beta \eta_1 (1 - r) \sigma}{K_1 K_2} + \frac{\beta \eta_2 r \sigma}{K_1 K_3} + \frac{\beta K_6}{K_1 K_2 K_3 K_4} + \frac{\beta \eta_3 K_7}{K_1 K_2 K_3 K_4 K_5},$$

$$R_0^{pv} = \frac{\beta N_0 \eta_4 \xi_1 (1 - r) \sigma}{K_1 K_2 v} + \frac{\beta N_0 \eta_4 \xi_2 r \sigma}{K_1 K_3 v} + \frac{\beta N_0 \eta_4 \xi_3 K_6}{K_1 K_2 K_3 K_4 v} + \frac{\beta N_0 \eta_4 \xi_4 K_7}{K_1 K_2 K_3 K_4 K_5 v},$$

with

$$K_6 = (1 - r) \sigma \gamma_3 K_3 + r \sigma \gamma_1 K_2$$

$$K_7 = \gamma_2 r \sigma K_4 + \kappa_1 r \sigma \gamma_1 K_2 + \kappa_1 (1 - r) \sigma \gamma_3 K_3.$$

The threshold quantity, $R_0$, measures the average number of new COVID-19 cases that one infected case can generate if introduced into a population, where basic public health interventions (such as quarantine, isolation, social-distancing etc.) are implemented [41]. Using Theorem 2 in [56], the following result is established.

**Theorem 1** The DFE, $E_0$, of the model (2) is locally-asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$. 

16
Closely linked to the control reproduction number is the time-dependent effective reproductive number defined by $R_e(t) := R_0 S/N$. The effective reproduction numbers at 13th March 2020, for some countries with COVID-19 cases are given in Table 4.

Table 4: Estimate of COVID-19 effective reproduction numbers for some countries

<table>
<thead>
<tr>
<th>Country</th>
<th>Estimate reproduction number $R_0$</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hubei Province, China</td>
<td>6.49 (6.31, 6.66)</td>
<td>[35]</td>
</tr>
<tr>
<td>Bahrain</td>
<td>6.64 (5.20, 8.61)</td>
<td>[29]</td>
</tr>
<tr>
<td>Denmark</td>
<td>5.08 (4.60, 5.62)</td>
<td>[29]</td>
</tr>
<tr>
<td>Spain</td>
<td>5.17 (4.98, 5.37)</td>
<td>[29]</td>
</tr>
<tr>
<td>Qatar</td>
<td>5.38 (4.59, 6.34)</td>
<td>[29]</td>
</tr>
<tr>
<td>Austria</td>
<td>3.97 (3.56, 4.42)</td>
<td>[29]</td>
</tr>
<tr>
<td>Norway</td>
<td>3.74 (3.47, 4.04)</td>
<td>[29]</td>
</tr>
<tr>
<td>Portugal</td>
<td>3.68 (2.86, 4.75)</td>
<td>[29]</td>
</tr>
<tr>
<td>United States</td>
<td>3.29 (3.15, 3.43)</td>
<td>[29]</td>
</tr>
<tr>
<td>Canada</td>
<td>2.30 (2.07, 2.57)</td>
<td>[29]</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>2.90 (2.72, 3.10)</td>
<td>[29]</td>
</tr>
<tr>
<td>Italy</td>
<td>2.44 (2.41, 2.47)</td>
<td>[29]</td>
</tr>
<tr>
<td>Germany</td>
<td>3.29 (3.18, 3.40)</td>
<td>[29]</td>
</tr>
<tr>
<td>France</td>
<td>3.09 (2.99, 3.19)</td>
<td>[29]</td>
</tr>
<tr>
<td>Iran</td>
<td>2.00 (1.96, 2.03)</td>
<td>[29]</td>
</tr>
<tr>
<td>South Africa</td>
<td>2.2 (2.1, 2.8)</td>
<td>[37]</td>
</tr>
</tbody>
</table>

Remark 2. A comment is in order with Theorem 1. As far as data are concerned, all the effective reproduction numbers presented in Table 4 are higher than 2. Consequently, we have $R_0 > 1$ for the control reproductive number. Let us simulate the model (2) using the set of parameter values presented in Table 3 with $\beta = \beta_0 = 0.40$. We obtained the value of the control reproduction number for South Africa to be $R_0 = 2.5025$. The epidemiological implication of Theorem 1 is that COVID-19 can be eliminated from the population when $R_0 < 1$, provided that the initial size of the sub-populations of the model are in the basin of attraction of the DFE, $E_0$. Here, the ideal situation $R_0 < 1$ does not happen, as mentioned earlier and seen from Table 4. However, we obtained $R_0 = 0.9697$ for the case when $\beta = \beta_1 = 0.155$. Hence implementing, for a sufficiently long period of time, population-wide social-distancing (lockdown) combined with other strict interventions such as home isolation of cases and wearing of face-masks, has the potential to bring the control reproduction number below unity and thus to suppress transmission, as suggested in the reference [20]. This fact is made precise in the following result about the global asymptotic stability of the set of disease-free equilibrium point. For the definition of the global asymptotic stability of a set, we refer the reader to [31].
Theorem 3  The manifold of disease-free equilibrium points, \([DFE]\), of the model (2) is globally-asymptotically stable if \(R_0 < 1\), and unstable if \(R_0 > 1\).

Proof. Set

\[
P_{\text{max}} = \frac{(\xi_1 + \xi_2 + \xi_3 + \xi_4)N_0}{v}.
\]

It is easy to prove that the system (2) is a dynamical system on the compact set

\[
\Omega = \{(S, E, A, Q, I, J, R, P) \in \mathbb{R}^7_+ : N \leq N_0, P \leq P_{\text{max}} \}.
\]

Notice that the disease-free manifold \([DFE]\) is contained in the invariant domain \(\Omega\). Define on \(\Omega\) the Lyapunov function candidate,

\[
\mathcal{L} = E + a_0A + a_1Q + a_2I + a_3J + a_4P,
\]

where the positive constants \(a_0, a_1, a_2, a_3, a_4\) will be determined shortly. Then the directional derivative \(\dot{\mathcal{L}}\) of \(\mathcal{L}\) in the direction of the vector-function defined by the right-hand side of the system (2), \((i.e.\) the derivative along the trajectories), is given by

\[
\dot{\mathcal{L}} = \lambda S - K_1E + a_0((1 - r)\sigma E - K_2A) + a_1(r\sigma E - K_3Q) + a_2((1 - r)\sigma A + \gamma_1 Q - K_4I) + a_3(\kappa_1I + \gamma_2Q - K_5J) + a_4(\xi_1A + \xi_2Q + \xi_3I + \xi_4J - vP).
\]

Since \(S/N \leq 1\) and \(S \leq N_0\) in \(\Omega\), some lengthy computations lead the following estimate of \(\dot{\mathcal{L}}:\)

\[
\dot{\mathcal{L}} \leq \beta\eta_1A + \beta I + \beta\eta Q + \beta\eta_3J + \beta\eta_4N_0P - K_1E + a_0((1 - r)\sigma E - K_2A) + a_1(r\sigma E - K_3Q) + a_2((1 - r)\sigma A + \gamma_1 Q - K_4I) + a_3(\kappa_1I + \gamma_2Q - K_5J) + a_4(\xi_1A + \xi_2Q + \xi_3I + \xi_4J - vP) - a_0K_2A + a_1r\sigma E - a_1K_3Q + a_2\gamma_1Q + a_2\gamma_3A - a_2K_4I + a_3\kappa_1I.
\]

Grouping like terms yields

\[
\dot{\mathcal{L}} \leq E [a_0(1 - r)\sigma + a_1r\sigma - K_1] + A [\beta\eta_1 - a_0K_2 + a_2\gamma_3 + a_4\xi_1] + I [\beta + a_3\kappa_1 + a_4\xi_3 - a_2K_4] + Q [\beta\eta_2 + a_2\gamma_1 + a_4\xi_2 + a_3\gamma_2 - a_1K_3] + J [\beta\eta_3 + a_4\xi_4 - a_3K_5] + P [\beta\eta_4N_0 - a_4v].
\]

The constants \(a_0, a_1, a_2, a_3, a_4\) are then chosen such that

\[
\begin{cases}
\beta\eta_1 + a_2\gamma_3 + a_4\xi_1 + a_3 - a_0K_2 = 0 \\
\beta + a_3\kappa_1 + a_4\xi_3 - a_2K_4 = 0 \\
\beta\eta_2 + a_2\gamma_1 + a_4\xi_2 + a_3\gamma_2 - a_1K_3 = 0 \\
\beta\eta_3 + a_4\xi_4 - a_3K_5 = 0 \\
\beta\eta_4N_0 - a_4v = 0.
\end{cases}
\]
That is

\[
\begin{align*}
a_0 &= \frac{\beta \eta_1 + a_2 \gamma_2 + a_4 \xi_1}{K_2} \\
a_1 &= \frac{\beta \eta_2 + a_2 \gamma_1 + a_3 \gamma_2 + a_4 \xi_2}{K_3} \\
a_2 &= \frac{\beta}{K_4} + \frac{\beta \eta_3 \kappa_1}{K_4 K_5} + \frac{\beta \kappa_1 \xi_4 \eta_4 N_0}{v K_4 K_5} + \frac{\beta \xi_3 \eta_4 N_0}{v K_4} \\
a_3 &= \frac{\beta \eta_3}{K_5} + \frac{\beta \xi_4 \eta_4 N_0}{v K_5} \\
a_4 &= \frac{\beta \eta_4 N_0}{v}
\end{align*}
\]

This simplifies the above estimate of \( \dot{L} \) into

\[
\dot{L} \leq -K_1(1 - R_0)E,
\]

where the basic reproduction number is given in (7).

Assume as from now that \( R_0 < 1 \). Then it follows from (8) that

\[
\dot{L} \leq 0, \text{ with } \dot{L} = 0 \text{ if and only if } E = 0.
\]

Consider the set \( \mathcal{M} \) defined by

\[
\mathcal{M} = \{(S, E, A, Q, I, J, R, P) \in \Omega : \dot{L} = 0 \}
\]

and let \( \mathcal{B} \) be a compact invariant set contained in \( \mathcal{M} \). Denote by \( S(t), E(t), A(t), Q(t), I(t), J(t), R(t), P(t) \) the solution of the system (2) initiated at a point \( B \in \mathcal{B} \). Since \( \mathcal{B} \subset \mathcal{M} \) is invariant, we have \( (S(t), E(t), A(t), Q(t), I(t), J(t), R(t), P(t)) \in \mathcal{M} \), which by (9) implies that \( E(t) = 0 \) at any time. Substituting all of this in the model (2) shows that \( (S(t), E(t), A(t), Q(t), I(t), J(t), R(t), P(t)) \in [DFE] \). Since \( B \) and \( \mathcal{B} \) were taken arbitrarily, we have shown that the largest compact invariant set contained in \( \mathcal{M} \) is the set \([DFE]\). It follows, from LaSalle's Invariance Principle [31], that every solution to the system (2), with initial conditions in \( \Omega \), approaches the set \([DFE]\) as \( t \to \infty \).

Though each disease-free equilibrium point \( E \in [DFE] \) of the model (2) is locally asymptotically stable when \( R_0 < 1 \), the above result shows that the set \([DFE]\) is GAS under this condition (thus, COVID-19 will be effectively controlled or eliminated from the population).
With the expression of the control reproductive number computed in (7) for the constant contact rates \( \beta = \beta_0 \) and \( \beta = \beta_1 \), we are in a position to state an important result on the final sizes of the COVID-19 pandemic variables. The result is about the measure of the severity of the infection in two different ways. On the one hand, it predicts the number of susceptible individuals, who over the course of the COVID-19, will escape the pandemic. On the other hand, while the final sizes of variables with infection are zero, the result provides a measure of the severity of the epidemic in terms of its final size relations, as done in the study [2] that was recently applied to a COVID-19 model in [41]. The result reads as follows:

**Theorem 4** Denote \( N_\infty := \lim_{t \to \infty} N(t) \) and assume that \( S_0 = N_0 \). Let \( x \) denote the column vector \( x := (E, A, Q, I, J, P)^T \) and let \( b \) be the row vector defined by \( b := (0, \eta_1, \eta_2, 1, \eta_3, N_0 \eta_4) \). Then, we have the following:

1. The functions \( S(t) \) and \( N(t) \) satisfy the property
   \[
   \lim_{t \to \infty} S(t) = S_\infty > 0, \quad \text{so that} \quad 0 < S_\infty \leq N_\infty < N_0, \tag{10}
   \]
   where \( S_\infty \) is the unknown final size of the epidemic to be determined.

2. For the variables with infection in the solution of the COVID-19 pandemic model (2), we have
   \[
   x_\infty := \lim_{t \to \infty} x(t) = 0. \tag{11}
   \]

3. The final size relation for the COVID-19 pandemic is given by
   \[
   \ln \frac{S_0}{S_\infty} \geq R_0 \left( 1 - \frac{S_\infty}{S_0} \right) + \beta b V^{-1} x_0, \tag{12}
   \]
   where \( \beta = \beta_0 \) or \( \beta_1 \), the matrix \( V^{-1} \) is specified in Eq. (6), and \( x_0 = x(0) \).

Using the explicit expression of the matrix \( V^{-1} \) given in Eq. (6), and the fact that at the initial stage of the epidemic \( Q(0) = J(0) = 0 \), one obtains the following lower bound:

\[
\begin{align*}
b V^{-1} x_0 & \geq \eta_1 \left( E(0)(1 - r)\sigma + A(0)K_1 \right) + \frac{E(0)\eta_2 r\sigma}{K_1 K_3} + E(0)U_1 \\
&+ \left( A(0)\gamma_3 + I(0)K_2 \right) + \eta_3 \left( E(0)U_2 + \frac{A(0)\kappa_1 \gamma_3 + I(0)\kappa_1 K_2}{K_2 K_4 K_5} \right) \\
&+ N_0 \eta_4 \left( A(0)U_3 + \frac{E(0)R_0^{pv}}{\beta_0 N_0 \eta_4} + \frac{P(0)}{v} + \frac{I(0)(K_5 \xi_3 + \kappa_1 \xi_4)}{K_4 K_5 v} \right). \tag{13}
\end{align*}
\]
To simplify the final size relation, it is usual to set some of the initial conditions to be equal to zero \([2, 41]\). A typical choice is \(E(0) = A(0) = Q(0) = J(0) = P(0) = 0\) and \(I(0) > 0\). In this case, the final size relation (12)-(13) reduces to
\[
\ln \frac{S_0}{S_\infty} \geq R_0 \left( 1 - \frac{S_\infty}{S_0} \right) + \beta \left( \frac{1}{K_4} + \frac{\eta_3 \kappa_1}{K_4 K_5} + \frac{\eta_4 N_0 (K_5 \xi_3 + \kappa_1 \xi_4)}{K_4 K_5 v} \right) I(0).
\] (14)

For the analysis in the next section, Theorem 4 will be used as follows. The number \(\alpha := 1 - \frac{S_\infty}{S_0}\), called the ”attack rate or ratio” of the epidemic, is a measure of its severity, apart from the number \(S_\infty\) of susceptible individuals who escaped the epidemic [4]. The larger the attack rate is, the more severe the epidemic is, in terms of the cumulative total number \(S_0 - S_\infty\) of COVID-19 cases. Furthermore, Theorem 4 clarifies that the epidemic will stop, i.e. its curve will flatten, but not because of exhaustion of susceptible individuals [4].

### 3.3 Testing and contact tracing

Along the lines of the estimation of the parameters done earlier in this section, there remains a major challenge with the whole COVID-19 pandemic, which impacts on the prediction of the dynamics of the disease. That is, no country in the world knows the number of people infected with COVID-19. The asymptomatic spread of COVID-19 is a big unknown, as highlighted in [1]. The WHO’s recommendation [60] to address the crisis is to conduct as many tests as possible because a greater degree of testing would provide a larger sample of people for which their infection status is known, thereby giving a less biased idea of the true prevalence of the virus. South Africa has subscribed to this strategy.

With a progression from 0.34 tests per thousand people on 25 March 2020 to 16.83 tests per thousand people as of 10 June 2020, South Africa is far ahead of other involved African countries, as illustrated on Figure 4 (A) drawn from Our World in Data [42]. From the same source, it cannot be disputed that conducting more tests increases the number of confirmed cases. For instance, on 10 June 2020, the following was recorded [42]: 998400 tests vs 52991 confirmed cases (South Africa), 102956 tests vs 2989 confirmed cases (Kenya), 82935 tests vs 13464 confirmed cases (Nigeria), 82239 tests vs 463 confirmed cases (Rwanda), and 56739 tests vs 4516 confirmed cases (Senegal).

However, South Africa is far below the set target of tests per day [39] i.e. 15,000 tests from 7 April 2020 to be up-scaled to 36,000 tests from end of April 2020. Figure 4 (B) drawn from Wits, NRF, iThemba Labs and Data Convergence dashboard [15] speaks for itself regarding the low numbers of tests per week conducted in South Africa from
Week 7 (15-16 February 2020) to Week 22 (25-29 May 2020).

It is essential and vital for South Africa not only to meet its target but to achieve beyond it, a campaign which can become a success in view of the easing of the lockdown measures and of the encouraging development of the past couple of days where more than 25,000 tests per day were conducted. In this regard, the Western Cape Province has been declared a hotspot COVID-19 province due to its high cumulative total numbers that read as follows on 12 June 2020 [15]: 36279 confirmed cases \textit{i.e.} 68\% of all national cases, and 891 deaths \textit{i.e.} 66\% of all deaths. Despite this fact that makes Western Cape the epicenter of COVID-19, the effective campaign of this province to conduct massive and quality tests, and contact tracing (cumulative total tests: 217534 \textit{i.e.} 20\% of all national tests) should be followed by other South African provinces and all countries in the continent if Africa is to better fight the COVID-19 pandemic, which is part of the sound recommendations in [25].
Figure 4: (A) Full list of cumulative total tests per thousand people in recorded African countries from 20 February to 11 June 2020; (B) Total conducted tests per week in South Africa from 15 February to 29 May 2020.

4 Numerical Simulations, Analysis and Discussion

In this section, numerical simulations and epidemiological analysis are carried out using the COVID-19 data for South Africa to assess the potential impact of the available intervention strategies.
4.1 Compliance with social-distancing measures

The effect of social-distancing coupled with other interventions such as quarantine and isolation is assessed by simulating the model (2) using the baseline parameter values presented in Table 3. The simulations show a decrease in the numbers of asymptomatic, quarantine, symptomatic and isolated individuals with increase in the social-distancing parameter $\omega$, as depicted on Figure 5 (A)-(D). This result is consistent with the fact that the social-distancing intervention reduces the number of cases in the USA [41]. Of great importance is also what the same Figure 5 (A)-(D) reveals regarding when to reach the peak of the pandemic in South Africa under the current strict social-distancing protocols. If 6 in 1000 people comply with the interventions per day, i.e the social-distancing effectiveness parameter $\omega = 0.006$, then the peak of the pandemic is expected to be attained around mid July 2020, with 90000 cumulative total confirmed cases at peak time, as seen on Figure 6 (A). On the contrary, a moderate or mild effectiveness level of social-distancing ($\omega = 0.004$) will delay the peak to September 2020, with 140000 cumulative total confirmed cases (see Figure 6 (A)). Furthermore, simulations show that the time to COVID-19 control (or elimination) using strict social-distancing protocol is shorter (expected to be attained by January 2021), compared to the mild effectiveness level of social-distancing which brings that time around late May 2021. Note that on 6 May 2020, South Africa’s government data modeling team [50] predicted the peak time of COVID-19 to be early July to early August 2020, with 80000-100000 total cases.

The next set of simulations presented on Figure 6 deals with cumulative total numbers of affected individuals. As the social-distancing parameter ($\omega$) increases, items (A), (B) and (C) of Figure 6 show a decrease in the cumulative numbers of infections, recoveries and deaths, respectively. Figure 6 (D) has a compelling message regarding the scenario were no social-distancing is implemented ($\omega = 0$). That is, the cumulative total number of COVID-19 induced mortality increases significantly with time. Thus, these simulations show that the implementation of strict social-distancing policy (popularly known as lockdown or stay at home) by South Africa’s Government has a significant community-wide impact in mitigating the transmission of COVID-19.
Figure 5: Simulations of the model (2), showing the decrease in numbers of COVID-19 infected individuals as the social-distancing parameter $\omega$ increases: (A) Asymptomatic, (B) Quarantined, (C) Symptomatic, and (D) Isolated individuals, respectively. Parameter values used are as given in Table 3 with various values of $\omega$. 
Figure 6: While the cumulative total number of deaths significantly increases in the absence of interventions i.e. $\omega = 0$ (D), there is a decrease in cumulative numbers of carriers of infections (A), recoveries (B), and deaths (D) as $\omega$ increases. Parameter values used are as given in Table 3 with various values of the compliance parameter $\omega$.

A further remark is in order about Figure 6 and the associated compelling message mentioned above. The nation-wide lockdown in South Africa, which started on 26 March 2020 at Alert 5 (i.e. most stringent restrictions on movement and economy activity) will be further down-scaled to Alert 3 (i.e. greater relaxing of restrictions) from 1 June 2020, after the earlier low-down to Alert 4 (i.e. retains most of the restriction of Alert 5)) that lasted from 1-31 May 2020. There is now strong uproar to completely lift the lockdown to Alert 1. The main challenge is, of course, to ensure that any easing of the lockdown measures does not erase the gains made so far in curtailing the pandemic. Hence, like [25, 41], this study strongly suggests that absolute caution should be exercised before terminating the current strict social-distancing protocols or lowering the COVID-19 alerts, so as to avoid the resurgence of the pandemic. Recent findings already showed that certain countries, such as South Korea and Hong Kong, that have relaxed the successfully-implemented social-distancing measures are
now experiencing a rebirth of COVID-19 [24]. Community lockdown surely induces severe burden and stress on the people. Nonetheless, it is a necessary measure to take to avert catastrophic loss of lives. Consequently, governments at all levels and private sectors have a responsibility to do everything they can to alleviate the sufferings of the people during lockdown.

4.2 The impact of quarantine

The effect of quarantine of individuals suspected of being exposed to COVID-19 is monitored by simulating the model (2) using parameter values given in Table 3 with various levels of effectiveness of quarantine ($r\sigma$). The results obtained, depicted in Figure 7 (A)-(D), show that quarantine of suspected individuals has a great impact in reducing the number of cases in a community. For instance, Figure 7 (C) suggests that early quarantine of suspected individuals could lead to averting of 12300 symptomatic cases i.e. a decrease by 78%, thereby reducing disease burden.
Figure 7: Simulations of the model (2), showing changes in the numbers of COVID-19 infected individuals, as the probability of quarantine \((r)\) varies: (A) Asymptomatic, (B) Quarantined, (C) Symptomatic, and (D) Isolated individuals, respectively. Parameter values used are as given in Table 3.

4.3 Impact of starting time of the lockdown

Additional simulations were carried out to assess the population-level impact of early or late implementation of the lockdown policy. Using various values of the lockdown implementation dates \((\tau_0)\), Figure 8 (A)-(D) and Figure 9 (A)-(B) deal with total cases and cumulative total cases, respectively. They show that the early implementation of strict social-distancing protocols right from the very beginning of the COVID-19 pandemic in South Africa \((i.e. 25\ March\ 2020)\), has played an important role in terms of shifting and lowering the peak daily cases of COVID-19, and the fact that the pandemic is estimated to reach its peak around mid-July 2020. However, for the
scenario, where the lockdown is implemented at a later stage of the infection (assume one week later \textit{i.e.} 1 April, 2020), then the pandemic is expected to reach its peak in August 2020 with higher number of cases, with a longer time for disease control, as depicted on Figure 8 (A)-(D). The simulation presented in Figure 9 (A) shows that implementation of lockdown by the government one week earlier has helped in averting 250,000 cases of COVID-19, which represents a 39\% decrease in number of cases. Furthermore, our simulations showed that the early implementation of social-distancing measures in the country by one week has significantly decrease cumulative COVID-19 related mortality by about 11,000 (\textit{i.e.} 39\% reduction) nationwide as shown on Figure 9 (B).

Figure 8: Simulations of the model (2), showing changes in the number of COVID-19 infected individuals, as the parameter $\tau_0$ for the starting time of the lockdown varies: (A) Asymptomatic, (B) Quarantined, (C) Symptomatic, and (D) Isolated individuals, respectively. Parameter values used are as given in Table 3.
4.4 Severity of the COVID-19 pandemic

Numerical simulations of the model using the initial conditions $S_0 = 5.9 \times 10^7$ (population of South Africa), $E(0) = A(0) = Q(0) = P(0) = 0$ and $I(0) = 30$ are presented in Figure 10.

In the absence of any control measures, i.e. the contact rate is $\beta = \beta_0$, Figure 10 (A) shows that the number of susceptible individuals who will escape COVID-19 at the end of the pandemic is $S_\infty = 5.885 \times 10^7$. In the case when control measures are successfully implemented, so that $\beta = \beta_1$, the final size of susceptible individuals is $S_\infty = 5.899 \times 10^7$, as shown on Figure 10 (B). Furthermore, the corresponding attack rates ($\alpha$) are computed and found to be $\alpha = 0.0025$ and $\alpha = 0.0016$, respectively. Hence, the final size relation given in Theorem 4, in its simplified form (14), is satisfied since $R_0 = 2.5025$ for $\beta = \beta_0$ and $R_0 = 0.9697$ for $\beta = \beta_1$ (see Remark 2).

These results confirm that the infection is more severe for high contact rate $\beta$. Notice that, for the case when $\beta = \beta_0$, the total number of COVID-19 cases at the end of the pandemic is obtained to be $S_0 - S_\infty = 150,000$, which is higher than the number obtained for $\beta = \beta_1$. It also transpires from Figure 10 that the peak time and the flattening of the COVID-19 infection curve arise earlier when $\beta = \beta_1$ (i.e. mid July 2020) than when $\beta = \beta_0$, as already commented regarding Figure 6.
Figure 10: Simulations of the model (2) for the computation of the final size relations of the COVID-19 pandemic, namely the number $S_\infty$ of susceptible individuals who escaped the epidemic and the attack rate $\alpha$ of the epidemic. (A) In the absence of any control measures ($\beta = \beta_0$), (B) Under strict lockdown ($\beta = \beta_1$). Parameter values used are as given in Table 3, while initial conditions are $S_0 = N_0 = 59 \times 10^7$, $I_0 = 30$, $E_0 = Q_0 = A_0 = J_0 = P_0 = R_0 = 0$.

4.5 Impact of environmental contamination

Finally, the impact of environmental transmission is assessed by simulating the model (2) with various values of the transmission modification coefficient $\eta_4$ as depicted in Figure 11. When $\eta_4$ becomes larger than the baseline value, items (A) and (B) of the figure show continued increases in cumulative number of cases and deaths, respectively.
Figure 11: Simulations of the model (2), showing changes in the cumulative numbers of COVID-19 related cases (A) and deaths (B) as the environmental infection coefficient $\eta_4$ varies. Parameter values used are as given in Table 3.

5 Conclusions

Since the beginning of March 2020, South Africa has been hit by the COVID-19 pandemic in a more complex manner than China, where the disease started, as all cases were imported and culminated into challenging types of transmissions of the infection in super-spread events, hotspot transmission areas, community transmission areas, etc. In the current absence of treatment and vaccine that could eradicate the disease, South Africa’s government response to COVID-19 is to flatten the curve of infection early and to reduce the number of infections at the peak time, while expanding our healthcare capacity and better preparing our equipment in hospitals for the worse scenario to come [50].

Based on the authors’ experience with the outbreaks of the Severe Acute Respiratory Syndrome (SARS) [21] and the Middle East Respiratory Syndrome (MERS) [55], we have considered, for the transmission dynamics of COVID-19 in South Africa, an extension of the standard SEIR model in which the infected individuals are stratified into Asymptomatic, Symptomatic infectious, Quarantine, and Isolated compartments, respectively. We have also added a compartment of contaminated environment, in accordance with the campaign to disinfect surfaces, buttons, hands, knobs and other places touched often [51]. The model is used to assess the impact of various non-pharmaceutical measures on the control of the pandemic in South Africa [20].
The main findings of the study, which in a nutshell suggest that the COVID-19 can be controlled in South Africa provided that all the envisaged measures are implemented effectively, include the following:

(I) There is a good fit of the existing daily incidence of COVID-19 with the model system plot.

(II) We introduced a social-distancing effectiveness parameter $\omega > 0$ in terms of which threshold analysis led to the following result: The (cumulative total) numbers of COVID-19 infected individuals \textit{(i.e.} asymptomatic, quarantined, symptomatic, and isolated individuals\textit{)} decrease as $\omega$ increases. Furthermore, in the absence of social-distancing \textit{(i.e.} $\omega = 0$\textit{)}, the cumulative total number of COVID-19 induced mortality increases significantly with time.

(III) We introduced a starting time parameter $\tau_0$ for the lockdown measure in terms of which the following result was obtained: Early implementation of the lockdown intervention \textit{(i.e.} decreasing $\tau_0$\textit{)} results in considerable decrease in the numbers of COVID-19 infected individuals. In particular, the COVID-19 pandemic peak time \textit{(i.e.} mid-July 2020 for $\tau_0 = 25$\textit{)} can be delayed and its peak daily case become larger if $\tau_0 > 25$.

(IV) We introduced a quarantine parameter $r\sigma$, which as it varied, led to changes in numbers of COVID-19 infected individuals.

(V) We introduced an environmental infection coefficient $\eta_4$, which once increased kept both numbers of COVID-19 infected and death individuals increasing.

(VI) Our computation of the reproduction number showed that $R_0 = 2.5025$. This suggests that the outbreak will continue in South Africa. However, this threshold quantity could be brought to a value less than unity ($R_0 = 0.9697$) if the aforementioned control measures are effectively implemented.

(VII) We computed the final size relations of the COVID-19 pandemic, and the associated attack rate, which beside being a measure for the severity of the pandemic suggests that the lower the attack rate is the sooner the peak time of the COVID-19 would arise.

(VIII) Similar to [25, 41], this study suggests that caution should be exercised before easing or lowering the COVID-19 alerts, so as to avoid to erase the gains made so far in curtailing the pandemic.
This study suggests that massive and quality testing as well as contact tracing be effectively implemented in order to address the challenge posed by the big unknown number of asymptomatic individuals.

Our planned research project for the near future is to study the transmission dynamics of COVID-19 in the Western Cape Province. In the past few weeks and while finalizing this paper, we have observed alarming trends in the estimated (cumulative total) numbers of COVID-19 confirmed cases and deaths in the Western Cape Province and its capital Cape Town (65% of the entire country) [15]. Western Cape has been declared the epicenter of COVID-19. However, this province has been exemplary in conducting the highest number of tests in the country (a total of 194939 tests \(i.e\). 20%), and in identifying hotspot transmission areas or super-spread events. When designing the model for Western Cape, we need to take into account the framework of [50] in carefully sub-dividing the isolation compartment since hospitals, ICUs and other healthcare facility are already overwhelmed in the province (\(viz\). 1425 COVID-19 cases are hospitalized including 230 in ICUs).

Acknowledgements

The authors acknowledged the support of South African DST/NRF SARChI Chair in Mathematical Models and Methods in Bioengineering and Biosciences.

Conflicts of interest

The authors declare no conflict of interest.

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