Mathematical COVID-19 model with vaccination: A case study in Saudi Arabia

Abeer D. Algarni[1], Aws Ben Hamed[2], Monia Hamdi[1], Hela ElMannai[1], Souham Meshoul[1]

[1] Department of Information Technology, College of Computer and Information Sciences, Princess Nourah bint Abdulrahman University, P.O.Box 84428, Riyadh 11671, Saudi Arabia

[2] Stability and Control Systems, College of Sciences, Sfax University, Tunisia

Abstract

The discovery of a new form of corona-viruses in December 2019, SARS-CoV-2, commonly named covid-19, has transformed the world. With health and economy issues, scientists have been focusing on understanding the dynamics of the disease, in order to provide the governments with the best policies and strategies allowing them to reduce the span of the virus. The world has been waiting for the vaccine for almost one year. In this work, we propose a new mathematical model with five compartments, including susceptible, vaccinated, infectious, asymptotic and recovered individuals. We provide theoretical results regarding the effective reproduction number, the stability of endemic equilibrium and disease free equilibrium. We provide a numerical analysis of the model based on the Saudi case.

keywords: COVID-19; mathematical model; vaccination; stability

1 Introduction

The outbreak of several pandemics such as COVID-19, requires the development of mathematical models in order to exhibit key epidemiological features, investigate transmission dynamics, and develop adequate control policies. Mathematical modelling, when dealing with infectious diseases, allows revealing inherent patterns and underlying structures that govern outbreaks. Simple models that contain the essential components and interactions are powerful tools to test different hypotheses and understand disease control for both short and long time. The stability analysis near the free disease equilibrium will show if the apparition of new infection cases will yield to disease outbreak. Some countries such Tunisia and Jordan registered zero cases for days in Summer 2020 but the introduction of new cases resulted in critical endemic situation by Autumn.

The complex spreading patterns of COVID-19 and the various spread speed of its variants make its containing and mitigating real challenges. The existing models vary in form and complexity, but the common objective is to provide important information for global health decision makers about the disease dynamics. Driven by the observed characteristics of COVID-19, we propose a mathematical model with two infectious states. It was reported by World Health Organization that one in three people who get COVID-19 do not show any symptoms. This is a challenging problem for health authorities as the asymptotic individuals carry the virus and may infect other people without knowing it. Moreover, consequent efforts were made worldwide since the authorisation of new vaccines by the end of 2020. By the end of November 2021, more than 50% of the world population has first dose administered and only 40% has second dose administered. In order to study the efficacy of vaccination to contain the virus spread and its negative consequences, our model include vaccinated state. The objective is to provide efficient public health policies in determining optimal vaccination strategies. Some questions have raised since the beginning of vaccination campaigns: how many individuals should

be vaccinated? Is the vaccine a solution to get rid of the disease permanently? These questions are related to financial and moral costs associated with the chosen governmental policy. This paper gives theoretical and numerical analysis associated with COVID-19 epidemic dynamics in order to answer these critical questions. Although we focus mainly on the Saudi case, the model structure is general and numerically adapted to any specific context without loss of validity of the qualitative results here shown.

In Section 2, we present the related works regarding epidemic modeling with a focus on COVID-19 control strategies and particularly population vaccination. Sections 3 and 4 include model description and analysis, respectively. The numerical results are given in Section 5 and Section 6 concludes this paper.

2   Related Works

The mathematical modeling in epidemiology started in England, in the 18th century, when Bernoulli analyzed the mortality of smallpox. Since then, a large variety of epidemiological models have been developed [13] [19][6] [25]. In this section, we present recent works proposed in this century, impacted by several outbreaks such as Ebola, Zika, and the swine flu [7].

Alexander and Moghadas [2] developed a Susceptible-Infected-Recovered-Susceptible (SIRS) epidemic model. The authors considered that the immunity acquired by the population after infection decreases over time. The dynamical behavior of the model is investigated using different types of bifurcation, including saddle-node, Hopf, and Bogdanov-Takens. The stability analysis based on the basic reproductive number and the rate of loss of natural immunity demonstrated the coexistence of two concentric limit cycles. These theoretical results have epidemiological implications such the determination of epidemic outbreak and the control the disease spread.

The authors of [30] investigated the Susceptible, Exposed, Infectious, Quarantine, Susceptible (SEIQS) epidemic model, with a nonlinear incidence rate. This model takes into consideration the communal sanitation measure of quarantine, aiming at avoiding broad infection. The authors provided a stability analysis using codimension-1 (transcritical,saddle-node, and Hopf) and codimension-2 bifurcations (Bogdanov-Takens).

Recently, Lu *et. al* [18] studied the the SIRS epidemic model, the same considered in [2] but with a generalized non-monotone incidence rate. The incidence rate is a function of the infection force of a disease and the number of susceptible individuals. The given formula for the incidence rate models the psychological pressure of some epidemic disease. The government is, in general, lead to take some protective measures like lockdown when the infection number becomes very high. The authors showed that the model has both repelling and attracting Bogdanov-Takens bifurcations. Moreover, from the super-critical Hopf bifurcation, the authors concluded that a disease following this model presents periodic outbreak, which is very important to understand its dynamics, in the real world.

The impact of treatment function was investigated in [10] using SIS model, where recovered individuals become again susceptible and the incidence rate is supposed bi-linear. In the considered model, the treatment function is saturated, which results in the existence of backward bifurcation. Thus, the eradication of the disease is not only related to the reproduction number but also to other biological or epidemiological mechanisms, such as imperfect vaccine. The bifurcation analysis outlines the necessary conditions to eliminate the disease. Zhang *et. al* [31] discussed the impact of the number of hospital beds on SIS epidemic model, by considering a nonlinear recovery rate. The authors calculated the basic reproduction number corresponding to their model. This number determines the condition for the disease-free equilibrium to be globally asymptotically stable.

The limitations of medical resources, mainly the availability of vaccinations, is modeled using a piecewise-defined function for patient treatment in [28]. This function admits a backward bifurcation

with limited available medical resources. The variation of vaccination threshold affects the existence of multiple steady states,crossing cycle, and generalized endemic equilibria. Similarly, Perez *et al.* [23] considered nonlinear incidence rate for a generalized SIR model. Besides, the authors assumed that the model has saturated Holling type II treatment rate and logistic growth. Non linear and saturated functions allows to represent more accurately the dynamics epidemic diseases. Similar to previous stated works, the authors revealed the importance of the basic reproduction number R_0, whose value determines the existence of endemic equilibrium and the stability of the disease-free equilibrium. Under some conditions related to the disease transmission rate and the treatment rate, the model may undertake a backward bifurcation and a Hopf bifurcation. The above-mentioned articles considered general disease models. In the literature, we can also find specific models targeting particular disease such as avian influenza [15] and bacterial meningitis [5]. Since the declaration of World Health Organization (WHO) of the Severe Acute Respiratory Syndrome Coronavirus (SARS-CoV-2) as a pandemic on March 2020, the scientific community has been trying to understand the dynamics of this virus.

One of the measures to control the virus spread in to declare total or partial lockdown, forcing social distancing. The scientific community believes that the main cause of infection is the inhalation of virus droplets [14]. Gevertz *et al.* [11] modeled social distancing as a flow rate between susceptible and asymptomatic individuals. The model reveals the existence of of a critical implementation delay, when implementing social distancing mandates. A delay of two weeks is the critical threshold between infection containment and infection expansion.

Nadim and Chattopadhyay [21] investigated the effect of imperfect lockdown. In the adopted model, when the basic reproduction number, R_0 is less than unity, the stable disease free equilibrium coexists with a stable endemic equilibrium. This means that COVID-19 undergoes backward bifurcation. This phenomena was observed in the Kingdom of Saudi Arabia where the new cases were decreasing to reach 97 in 06, January 2021. Unfortunately, this rate reached 386 new cases, after one month, which obliged the Ministry of Health to declare partial lockdown for 10 days. The infection force is so high that the disease cannot be totally eradicated. The authors showed that under perfect lockdown, this backward bifurcation does not exist, but such condition is not possible in the real world. In [9], the authors included in their mathematical model, based on the classical SEIR, several prevention actions such as test campaign on the population and quarantining infected persons. The model took in consideration infection treatment efforts, such as vaccination and the therapy of induced cardio-respiratory complications. Besides the usual classes of the population, the authors considered two new classes, driven by specific characteristics of the virus: infected but asymptomatic patients and suspected infected individuals. The theoretical results, tuned using the Chinese case, were compared to United Kingdom case and the Italian case, showing the similarity between the model dynamics and the real epidemic behaviour. Another scientific aspect of COVID-19 is the possible transmission of the virus through contaminated surfaces. It is believed that the virus can survive several days on the surfaces depending on the material (wood, glass or plastic). Another issue faced by the governments is the awareness level of the population. Some individuals, deliberately, decide not to apply the precautionary measures, mainly wearing mask and respecting social distancing [16].

The issue of the efficiency of social distancing and rapid testing strategies against the pandemic was examined in [1], where the authors extended the standard SEIR model. The authors considered also the problem of undetected asymptomatic individuals, who have no symptoms but participate actively to virus spread. Furthermore, the limitation of medical resources was incorporated to the model. The theoretical findings emphasized the role of the basic reproduction number R_0 in the existence of stable COVID-19 free and COVID-endemic equilibrium points. This conclusion is contested by Mohd and Sulaiman [20], who studied the SIRS model with limited medical resources and false detection issues. The authors showed that the condition of reducing the basic reproduction number under the unity

132   value is necessary to eliminate the disease but not sufficient.

133   Since the authorization COVID-19 vaccines, several research works focused on giving insight to
134 mathematical characteristic of virus spread after population vaccination. Algehyne *et al.* [3] used
135 nonlinear functional analysis and fractal fractional derivative to model the evolution over time of four
136 compartments: susceptible, infected, infected positive tested, and recovered. The Spanish case was
137 investigated in [17] using also fractional derivatives. It is important to highlight that these works
138 do not consider vaccinated state as a separate compartment. They rather consider that the vacci-
139 nated individuals are moved from susceptible to recovered compartment.The vaccinated individuals
140 are considered to move also from exposed state in [29].

141   Different mathematical tools are used by Rajaei *et al.* [24] to compare the effect of vaccination
142 with social distancing and hospitalization. Extended Kalman filter (EKF) is used for state estimation
143 under uncertainty.

144   Most of the existing research works developing a relationship between infectious and asymptotic
145 individuals focus on estimating the model parameters using actual data [4] [11] [26] [12]. To the best
146 of our knowledge, our work is the first to provide to study mathematical stability of endemic and
147 disease free equilibrium.

148 3   Proposed model and effective reproduction number

149 Our objective is to derive the mathematical equations that better present the dynamics of covid-19
150 virus. The population is divided into five compartments: susceptible, vaccinated, infectious, asymp-
151 totic,and recovered; the numbers in these states are denoted by S(t), V(t), I(t), A(t), and R(t),
respectively. Fig. 1 depicted the flow diagram of the disease spread.

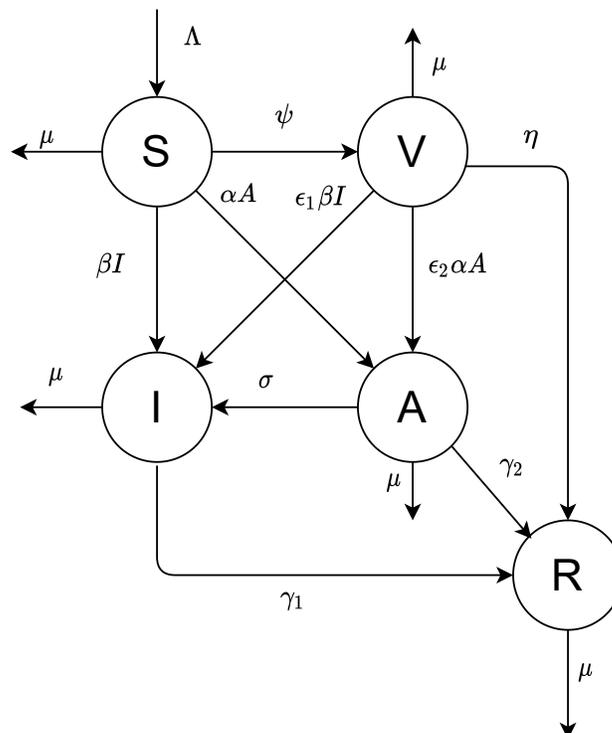



Figure 1: Proposed model.

152

153   All newborns are assumed to be susceptible.The natural recruitment and the natural death are
154 denoted by Λ and μ, respectively. The disease-induced death rate is ignored. Susceptible individuals
155 are vaccinated at rate constant ψ. The parameters α and β are the infecting rates of asymptotic and
156 infectious individuals, respectively. γ_1 and γ_2 are the rates that the infectious and asymptotic indi-
157 viduals become recovered and acquire temporary immunity, respectively. The vaccinated individuals
158 need a period of time to develop their immunity against the virus, represented by $\frac{1}{\eta}$.

159   The virus may infect vaccinated individuals but at a lower rate than susceptible individuals who
160 are not unvaccinated. Thus in this case, the transmission rates β and α are multiplied by a scaling
161 factor ϵ_1 and ϵ_2 ($0 \leq \epsilon_1$, $\epsilon_2 \leq 1$). Table 1 summarizes the different model parameters.

Parameter	Description
Λ	Recruitment rate of susceptible humans
μ	Natural mortality rate
β	Transmission rate for Infectious
α	Transmission rate for Asymptotic
ψ	Vaccination coverage rate
$1/\eta$	Immunity development period
γ_1	Recovery rate Infectious
γ_2	Recovery rate Asymptotic
$1/\sigma$	Period for asymptotic individuals to develop symptoms

Table 1: Model parameters and decription.

162   Based on the above assumptions and Fig. 1, we formulate the following model of differential
163 equations.

$$\frac{dS}{dt} = \Lambda - \mu S - \psi S - \beta IS - \alpha AS \tag{3.1a}$$

$$\frac{dV}{dt} = -\mu V + \psi S - \eta V - \epsilon_1 \beta IV - \epsilon_2 \alpha AV \tag{3.1b}$$

$$\frac{dI}{dt} = -\mu I + \beta IS + \epsilon_1 \beta IV + \sigma A - \gamma_1 I \tag{3.1c}$$

$$\frac{dA}{dt} = -\mu A + \alpha AS - \sigma A + \epsilon_2 \alpha AV - \gamma_2 A \tag{3.1d}$$

$$\frac{dR}{dt} = -\mu R + \eta V + \gamma_1 I + \gamma_2 A \tag{3.1e}$$

164   The basic reproduction number is defined as the number of secondary infections produced by a
165 single infectious individual during his or her entire infectious period. Since we introduce a vaccination
166 program in our model, it is called the effective reproduction number. The system (3.1) has always a
167 disease-free equilibrium, which is obtained by setting all the derivatives to zero with I = A = 0, that
168 yields to: $P_0 = (S_0, I_0, A_0, R_0, V_0) = (\frac{\Lambda}{\mu + \psi}, 0, 0, \frac{\eta \psi \Lambda}{\mu(\mu + \psi)(\mu + \eta)}, \frac{\psi \Lambda}{(\mu + \psi)(\mu + \eta)})$

169   Let $x = (I, A, V, R, S)^T$. System (3.1) can be rewritten as $x' = \mathcal{F}(x) - \mathcal{N}(x)$,, where $\mathcal{F}$ be the rate
170 of appearance of new infections in each compartment. The progression from A to I is not considered
171 to be new infection, but rather the progression of an infected individual through various infectious
172 compartments.

173
$$\mathcal{F}(x) = \begin{pmatrix} \beta I S + \epsilon_1 \beta I V \\ \alpha A S + \epsilon_2 \alpha A V \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{N}(x) = \begin{pmatrix} (\mu + \gamma_1)I - \sigma A \\ (\mu + \sigma + \gamma_2)A \\ (\mu + \eta)V - \psi S + \epsilon_1 \beta I V + \epsilon_2 \alpha A V \\ \mu R - \eta V - \gamma_1 I - \gamma_2 A \\ -\Lambda + (\mu + \psi)S + \beta I S + \alpha A S \end{pmatrix}.$$

174 The infected compartments are A and I, giving m=2. With A=I=0, the Jacobian matrices of $\mathcal{F}(x)$
175 and $\mathcal{N}(x)$ at the disease-free equilibrium P_0 are, respectively,

176
$$DF(P_0) = \begin{pmatrix} F & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad DN(P_0) = \begin{pmatrix} N & 0 & 0 & 0 \\ \epsilon_1 \beta V_0 & \epsilon_2 \alpha V_0 & \mu + \eta & 0 & -\psi \\ -\gamma_1 & -\gamma_2 & -\eta & \mu & 0 \\ \beta S_0 & \alpha S_0 & 0 & 0 & \mu + \psi \end{pmatrix},$$

177
$$F = \begin{pmatrix} \beta S_0 + \epsilon_1 \beta V_0 & 0 \\ 0 & \alpha S_0 + \epsilon_2 \alpha V_0 \end{pmatrix}, \quad N = \begin{pmatrix} \mu + \gamma_1 & -\sigma \\ 0 & \mu + \sigma + \gamma_2 \end{pmatrix}.$$

178 Our developed model is similar to the two-strain model in [27] with two infectious compartments.
179 FN^{-1}, the next generation matrix of system (3.1) has the two eigenvalues.

180
$$R_1 = \frac{\beta(S_0 + \epsilon_1 V_0)}{\mu + \gamma_1} = \frac{\beta\Lambda(\frac{1}{\mu+\psi} + \epsilon_1 \frac{\psi}{(\mu+\psi)(\mu+\eta)})}{\mu + \gamma_1} = \frac{\beta\Lambda(1 + \epsilon_1 \frac{\psi}{\mu+\eta})}{(\mu+\gamma_1)(\mu+\psi)}$$

181
$$R_2 = \frac{\alpha(S_0 + \epsilon_2 V_0)}{\mu + \gamma_2 + \sigma} = \frac{\alpha\Lambda(1 + \epsilon_2 \frac{\psi}{\mu+\eta})}{(\mu+\gamma_2+\sigma)(\mu+\psi)} = \frac{\alpha\Lambda(\mu+\eta+\epsilon_2\psi)}{(\mu+\gamma_2+\sigma)(\mu+\psi)(\mu+\eta)}$$

182 The effective reproduction number for the system is the maximum of the two.

4 Model analysis

4.1 Existence of endemic equilibrium point

In this section, we investigate the conditions for the existence of endemic equilibria of system (3.1).
Any equilibrium satisfies the following equations:

$$\Lambda - \mu S - \psi S - \beta I S - \alpha A S = 0 \tag{4.1a}$$
$$-\mu V + \psi S - \eta V - \epsilon_1 \beta I V - \epsilon_2 \alpha A V = 0 \tag{4.1b}$$
$$-\mu I + \beta I S + \epsilon_1 \beta I V + \sigma A - \gamma_1 I = 0 \tag{4.1c}$$
$$-\mu A + \alpha A S - \sigma A + \epsilon_2 \alpha A V - \gamma_2 A = 0 \tag{4.1d}$$
$$-\mu R + \eta V + \gamma_1 I + \gamma_2 A = 0 \tag{4.1e}$$

185 The equation (4.1d) gives the following expression:
186 $S = \frac{\mu+\sigma+\gamma_2}{\alpha} - \epsilon_2 V.$

187

188 The equation (4.1b) gives the following expression:
189 $V = \frac{\psi(\mu+\sigma+\gamma_2)}{\alpha(\mu+\eta+\epsilon_1\beta I+\epsilon_2\alpha A+\psi\epsilon_2)}.$

190

191 From Eq. (4.1c) and assuming $\epsilon_1 - \epsilon_2 = 0$, we deduce the following expressions:
192 $A = (\frac{\mu+\gamma_1}{\sigma} - \frac{\beta(\mu+\sigma+\gamma_2)}{\alpha\sigma})I = D\ I$
193 $S = \frac{\mu+\sigma+\gamma_2}{\alpha} \frac{(\mu+\eta+\epsilon_1\beta I+\epsilon_2\alpha A)}{(\mu+\eta+\epsilon_1\beta I+\epsilon_2\alpha A+\psi\epsilon_2)}$

194

195 The equation (4.1a) gives the following expression:
196 $\frac{\mu+\sigma+\gamma_2}{\alpha} \frac{(\mu+\eta+\epsilon_1\beta I+\epsilon_2\alpha D\ I)}{(\mu+\eta+\epsilon_1\beta I+\epsilon_2\alpha D\ I+\psi\epsilon_2)}[\mu + \psi + (\beta + \alpha D)I] = \Lambda$
197 We arrange the previous expression to get the following:
198 $(\epsilon_1\beta + \epsilon_2\alpha D)(\beta + \alpha D)I^2 + [(\mu+\eta)(\beta+\alpha D) + (\mu+\psi)(\epsilon_1\beta+\epsilon_2\alpha D) - \Lambda\alpha\frac{\epsilon_1\beta+\epsilon_2\alpha D}{\mu+\sigma+\gamma_2}]I + (\mu+\eta)(\mu+$

199 $\psi) - \Lambda\alpha\frac{\mu+\eta+\psi\epsilon_2}{\mu+\sigma+\gamma_2}$

We denote by:

$a = (\epsilon_1\beta + \epsilon_2\alpha(\frac{\mu+\gamma_1}{\sigma} - \frac{\beta(\mu+\sigma+\gamma_2)}{\alpha\sigma}))(\beta + \alpha(\frac{\mu+\gamma_1}{\sigma} - \frac{\beta(\mu+\sigma+\gamma_2)}{\alpha\sigma}))$

$b = [(\mu+\eta)(\beta+\alpha(\frac{\mu+\gamma_1}{\sigma} - \frac{\beta(\mu+\sigma+\gamma_2)}{\alpha\sigma})) + (\mu+\psi)(\epsilon_1\beta+\epsilon_2\alpha(\frac{\mu+\gamma_1}{\sigma} - \frac{\beta(\mu+\sigma+\gamma_2)}{\alpha\sigma})) - \Lambda\alpha\frac{\epsilon_1\beta+\epsilon_2\alpha(\frac{\mu+\gamma_1}{\sigma} - \frac{\beta(\mu+\sigma+\gamma_2)}{\alpha\sigma})}{\mu+\sigma+\gamma_2}]$

$c = (\mu+\eta)(\mu+\psi) - \Lambda\alpha\frac{\mu+\eta+\psi\epsilon_2}{\mu+\sigma+\gamma_2} = (\mu+\eta)(\mu+\psi)(1-R_2)$

The existence of endemic equilibrium is determined by the existence of positive solutions of the quadratic equation

$$P(I) = aI^2 + bI + c = 0 \qquad (4.2)$$

.

The number of endemic equilibria of the considered system depends on parameter values a, b, and c. This equation may have zero, one or two solutions. We denote $R_{20} = \frac{\alpha\Lambda}{(\mu+\gamma_2+\sigma)(\mu+\eta)}$ then

$R_2 = R_{20}\frac{\mu+\eta+\epsilon_2\psi}{\mu+\psi}$

We denote by $\psi_{crit} \overset{def}{=} \frac{(R_{20}-1)\mu + R_{20}\eta}{1 - \epsilon_2 R_{20}}$., where $R_2(\psi_{crit}) = 1$,

Since the model parameters A and I are positive, it follows that D >0 and a > 0. Furthermore, if $R_2 > 1$, then $c < 0$. Since $\frac{dR_2}{d\psi} = -R_{20}\frac{\eta + (1-\epsilon_2)\mu}{(\mu+\psi)^2} < 0$ Thus, R_2 is decreasing function of ψ and if $\psi < \psi_{crit}$, then $R_2 > 1$. We deduce that for $R_2 > 1$, P(I) has a unique positive root.

If $R_2 < 1$, we have $c > 0$ and $\psi \geq \psi_{crit}$. Since $b(\psi)$ is an increasing function of ψ, if $b(\psi_{crit}) \geq 0$, then $b(\psi) > 0$ for $\psi > \psi_{crit}$. In this case, P(I) has no positive real root and the system have no endemic equilibrium.

We consider now the case where $b(\psi_{crit}) < 0$. We denote by $\Delta(\psi) \overset{def}{=} b^2(\psi) - 4ac(\psi)$. If $c(\psi_{crit}) = 0$, $\Delta(\psi_{crit}) > 0$. Since $b(\psi)$ is an increasing linear function of ψ, there is a unique $\bar{\psi} > \psi_{crit}$ such that $b(\bar{\psi}) = 0$. and $\Delta(\psi)$ has a unique root $\bar{\bar{\psi}}$ in $[\psi_{crit}, \bar{\bar{\psi}}]$.

P(I) has two roots and the system 3.1 has two endemic equilibria for $\psi_{crit} < \psi < \bar{\bar{\psi}}$. and P(I) ha no real positive root and the system (3.1) has no endemic equilibria for $\psi > \bar{\bar{\psi}}$.

If $R_2 = 1$, we have $c = 0$. In this case, system has a unique endemic equilibrium for $b(\psi) < 0$ and no endemic equilibrium for $b(\psi) > 0$.

4.2 Stability of disease-free equilibrium

The Jacobian matrix with respect to the system (3.1) is given by:

$$J_0(P_0) = \begin{bmatrix} -(\mu+\gamma_1)+\beta(S_0+\epsilon_1 V_0) & \sigma & 0 & 0 & 0 \\ 0 & -(\mu+\sigma+\gamma_2)+\alpha(S_0+\epsilon_2 V_0) & 0 & 0 & 0 \\ -\epsilon_1\beta V_0 & -\epsilon_2\alpha V_0 & -(\mu+\eta) & 0 & \psi \\ \gamma_1 & \gamma_2 & \eta & -\mu & 0 \\ -\beta S_0 & -\alpha S_0 & 0 & 0 & -(\mu+\psi) \end{bmatrix}.$$

$\left|\lambda - J_0(P_0)\right| = 0.$

The characteristic polynomial of the Jacobian matrix at DFE is given by $det(J_0 - \lambda I) = 0$, where λ is the eigenvalue and I is 5×5 identity matrix. Thus, J_0 has eigenvalues given by:

$\lambda_1 = -\mu$

$\lambda_2 = -(\mu+\eta)$

$\lambda_3 = -(\mu+\psi)$

$\lambda_4 = -(\mu+\gamma_1)+\beta(S_0+\epsilon_1 V_0) = (\mu+\gamma_1)(R_1-1)$

$\lambda_5 = -(\mu+\sigma+\gamma_2)+\alpha(S_0+\epsilon_2 V_0) = (\mu+\sigma+\gamma_2)(R_2-1)$

All the eigenvalues are strictly negative except for λ_4 and λ_5. These eigenvalues depend the sign of (R_2-1) and (R_1-1). The stability of the DFE represents the dynamics of disease free population

237 when a small number of infected individuals introduced. Did the system stay disease free or an endemic
238 state may appear?

239 **Theorem 1** *Based on the Theorem of [27], we have the following results. If $R_1 > 1$ or/and $R_2 > 1$,*
240 *then λ_4 or/and λ_5 is/are strictly positive. In this case the DFE is unstable. If $R_1 < 1$ and $R_2 < 1$,*
241 *then λ_4 and λ_5 are strictly negative. The system is locally asymptotically stable.*

5   Numerical simulations

243 In this paper, we focus on vaccination analysis in Saudi Arabia. The presented numerical simulations
244 provide also general results that can be applied to any region. The data set is provided by King
245 Abdullah Petroleum Studies and Research Center (KAPSARC). It includes five classes: Tested, Cases,
246 Recoveries, Critical, Mortalities and Active and it spans the period from 04/03/2020 to 08/11/2021.
247 It includes also important events and measures such as international flights suspension and lockdown.
248 We use Simulink Tool in order to simulate different scenarios.

249   The death and birth rate for Saudi Arabia are estimated to be equal to 3.39 and 14.56 for 1000 per
250 year, respectively. The vaccination campaign started on 18/12/2020 with a vaccination coverage of the
251 total population of 0.02% to reach about 65% of the adult population fully vaccinated in November
252 2021. The vaccination rate is considered a the percentage of the total population that get vaccinated
253 per day. With approximately 45000 administrated doses per day and a total population of 35339000
254 in 2021, this rate is about 0.00 127. Is this rate enough to eradicate the disease? This what we are
255 trying to answer is this work.

256   The research report [22] provides information about asymptotic individuals for COVID-19. Most
257 people, with no symptoms at the beginning, develop symptoms in 7-13 days, which corresponds to
258 the σ^{-1}. Recall that γ_1 is the recovery rate of infectious individuals. Interpreted as the expected
259 value of a Poisson process, γ_1 can be related to the needed time from the beginning of the infection
260 till recovery [11]. With average recovery duration equal to 10 days [8], the recovery rate of infectious
261 individuals is $\gamma_1 = 0.1 =$.

262   Let ω denote the fraction of asymptotic individuals among positive cases. According to [22], and
263 based on 13 studies involving 21,708 people in 2020, $\omega = 0.17$. Using the same methodology as in [11]
264 $\gamma_2 = \frac{\omega}{1-\omega}\sigma \approx 0.2\sigma$. The asymptotic people are estimated to be 42% less contagious than symptomatic
265 individuals [22]. Thus, $\alpha = 0.42\ \beta$

Parameter	Range
Λ	14.56 per 1000 per year
β	[0.233, 0.462] [8]
α	0.42 β
ψ	model parameter
μ	3.39 per 1000 per year
$1/\eta$	14 days
γ_1	0.1
γ_2	0.2 σ
$1/\sigma$	[7,13] days

Table 2: Model parameters and values.

266   By 18/12/2020, considered as time 0 in this model, the number of recoveries is equal to 351722 ,
267 the number of active cases is equal to 3014. Assuming that $\omega = 0.17$, the number of initial infectious
268 with and without symptoms is equal to 2501 and 513, respectively.

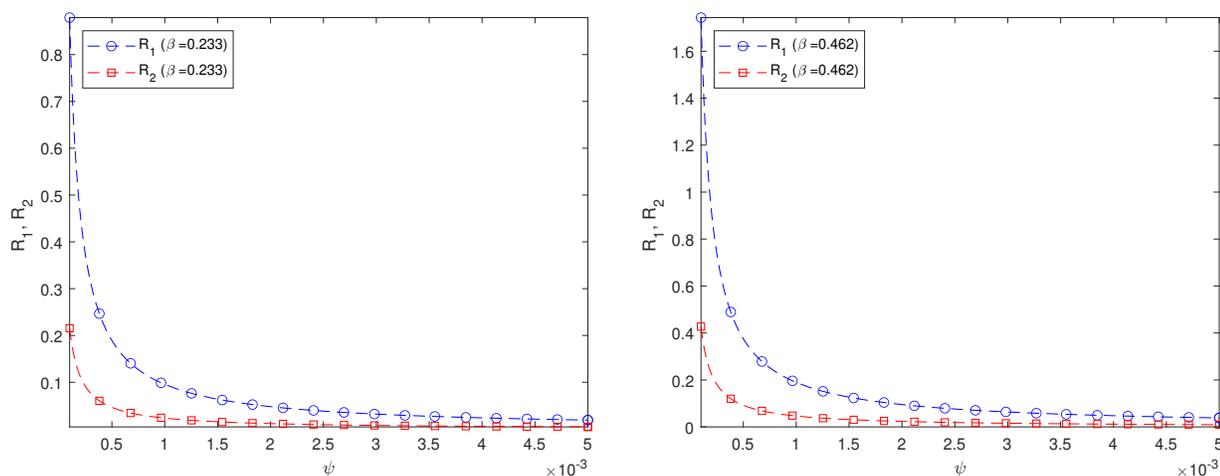
269   First, we investigate the effect of the vaccination rate on the effective reproduction number defined
270 as the maximum of the two entities R_1 and R_2. According to [8], covid-19 transmission rate β ranges
271 between 0.233 and 0.462. Fig 2a and Fig 2b show the evolution of both R_1 and R_2 as a function of
272 the vaccination rate ψ with virus transmission rate equal to 0.233 and 0.462, respectively. In both
273 cases, R_1 corresponding to the strain of infectious individuals with symptoms is greater than R_2,
274 corresponding to the strain of asymptotic individuals. Thus the number of individuals infected by one
275 person currying the virus is mainly affected by individuals showing usual symptoms. Mathematical
276 theoretical result confirms that the vaccine reduces the spread of the virus among the population.
277 We would like to highlight the fact in our model that a vaccinated individual may be infectious with
278 or without symptoms. This result is very important as, till the end of 2021, an important portion
279 of worldwide population is still opposed to vaccine. In the case of high transmission rate and low
280 vaccination rate, R_1 is higher than 1. The disease free equilibrium is consequently unstable according
281 to theorem 1. For the Saudi case, when $beta$ is equal to 0.233, R_1 and R_2 are equal to 0.0797 and 0.0195,
282 respectively. When $beta$ is equal to 0.462, R_1 and R_2 are equal to 0.1580 and 0.0387, respectively. For
283 the Saudi Arabia, the effective reproduction number is less than 1, even for high transmission rate.
284 This result is explained by the high vaccination rate.

(a) Varying R_1 and R_2 as a function of ψ, (b) Varying R_1 and R_2 as a function of ψ, $\beta = 0.462$.
$\beta = 0.233$.

Figure 2: Varying R_1 and R_2 as a function of vaccination rate for two virus transmission rates,
$\beta = 0.233$ and $\beta = 0.462$.

285   Fig. 3 shows the weekly number of new active cases and recovered after infection in KSA, starting
286 from 18/12/2020, the date when the vaccination starts. We can see that after 12 weeks, the number
287 of cases raises. This behaviour was surprising for a population waiting to see the effect of vaccination.
288 It's only after 31 weeks that the number of new cases start to decrease. The same phenomena was
289 observed in both Fig. 4c and Fig. 5c. The theatrical results is conform to actual statistics. The effect
290 of vaccination is not immediate; it needs several weeks to observe a decrease in the number of new
291 infectious cases.
292   We compare the evolution in time of the five compartments (S, V, I, A ,and R) presented in
293 our model, for two different transmission rate and with the Saudi vaccination rate. With different
294 dynamics at the beginning, both scenarios show a convergence to a stable state. We observe almost
295 similar patterns for S, V. I and R. The number of susceptible individuals S decreases slowly at the

²⁹⁶ beginning and then, we observe a drastic decline. Obviously, the number of recovered follows the same
²⁹⁷ slow and then fast pace but in decrease. The number of vaccinated individuals V increases gradually
²⁹⁸ at the beginning and then it begins to fall down. The number of infectious individuals I remain stable
²⁹⁹ for a short period to witness an expansion followed by a decline. The number of asymptotic individuals
³⁰⁰ show different evolution patterns for two considered scenarios. When we set a low value for the virus
³⁰¹ transmission rate, this number immediately shrinks. However, when we set a high value for the virus
³⁰² transmission rate, this number increases before shrinking.

³⁰³   The effect of the transmission rate can also be observed in the amplitude of each category of
³⁰⁴ individuals. When the models converge, the number of infectious and asymptotic individuals are zero.
³⁰⁵ We emphasize here our theoretical result, mentioned in Theorem 1, that states that if both R_1 and R_2
³⁰⁶ are less than one, the disease free equilibrium is stable. This result is consistent with the simulation
³⁰⁷ results. The difference between the two considered scenarios lies in the percentage of susceptible
³⁰⁸ and vaccinated individuals in the equilibrium. This percentage is very low when the transmission
³⁰⁹ rate is high. Although the percentages of vaccinated individuals are close, we observe a remarkable
³¹⁰ difference in the number of infectious individuals. When the transmission rate is high, almost 40% of
³¹¹ the population is infected, which rises public health issues.

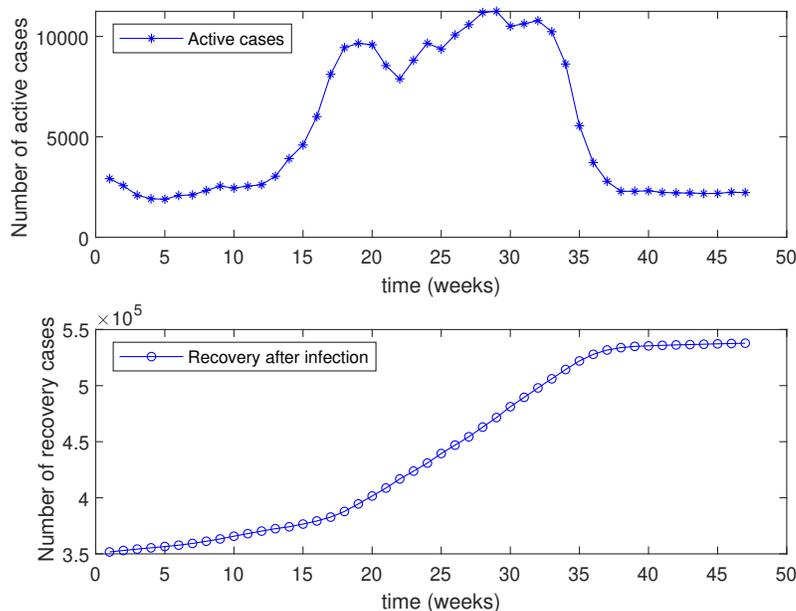
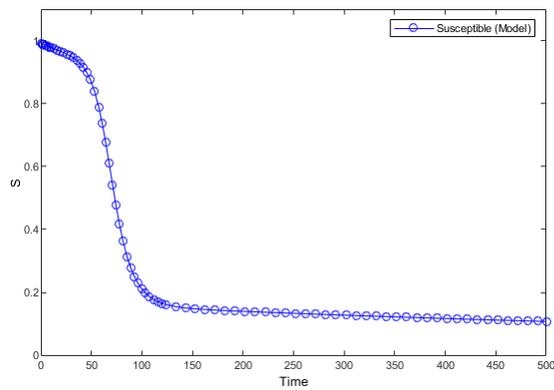
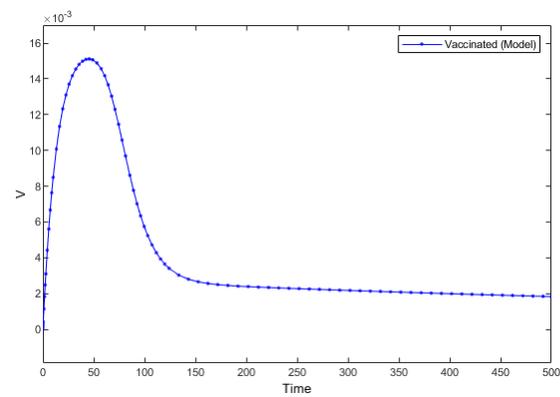
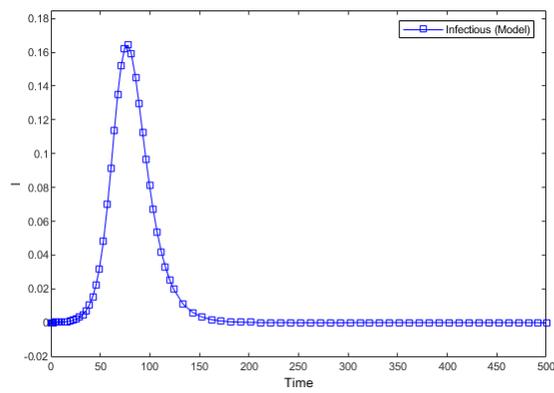
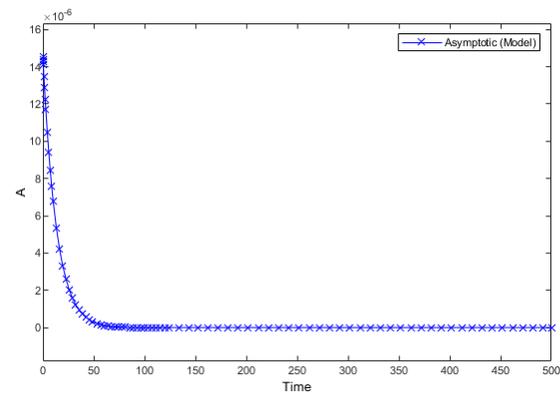
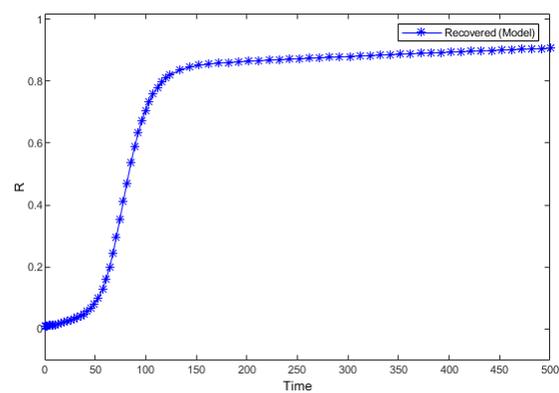
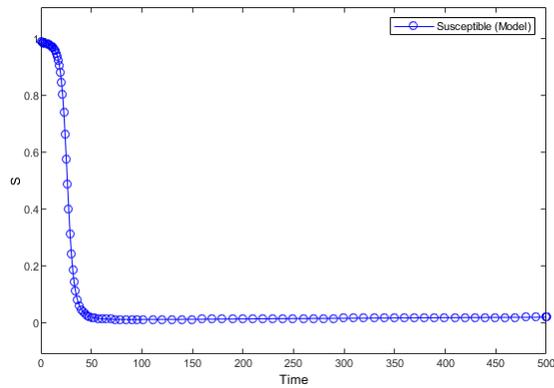
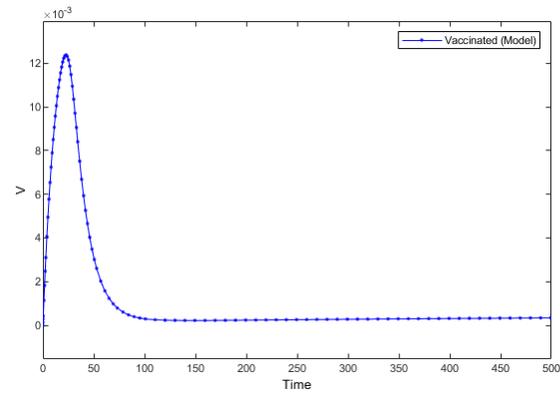
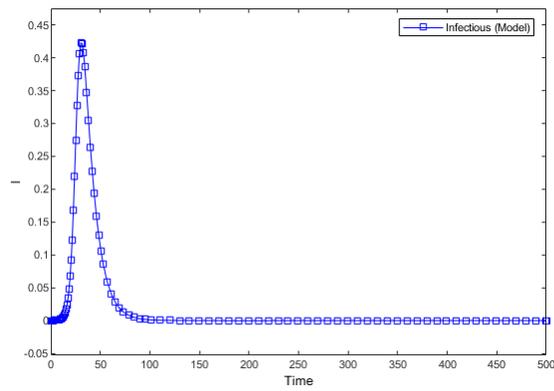
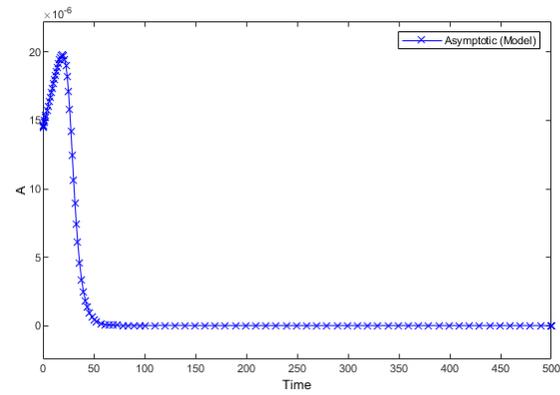



Figure 3: Number of active cases and recovered after infection in KSA. Starting from 18/12/2020.

(a) Susceptible

(b) Vaccinated

(c) Infectious

(d) Asymptotic

(e) Recovered

Figure 4: Percentage of susceptible, vaccinated, infectious. asymptotic and recovered individuals, $\beta = 0.233$, $\psi = 0.0012$.

(a) Susceptible

(b) Vaccinated

(c) Infectious

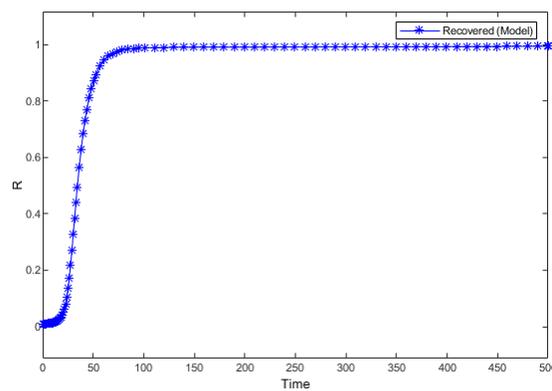
(d) Asymptotic

(e) Recovered

Figure 5: Percentage of susceptible, vaccinated, infectious. asymptotic and recovered individuals, $\beta = 0.462$, $\psi = 0.0012$.

6   Conclusion

In this paper, we present a mathematical model for covid-19, based on the virus behaviour. Our main target is to evaluate the effect of vaccination on the population. The presence of individuals presenting no symptoms and the immunity loss are the main characteristics that make this virus different from other known and already modeled diseases. We provide analytical expression of the effective reproduction number with is a key factor to determine necessary conditions for endemic and disease free equilibrium. We supported our theoretical findings with the numerical analysis applied to the Saudi case. Recently, the scientific community is observing the new variants that show each time different patterns. We aim in the future, to develop a new model with the new observed characteristics of variants such as beta and omicron.

Acknowledgment

The authors extend their appreciation to the Deputyship for Research & Innovation, Ministry of Education in Saudi Arabia for funding this research work through the project number PNU-DRI-RI-20-028.

References

[1] Dipo Aldila, Sarbaz H.A. Khoshnaw, Egi Safitri, Yusril Rais Anwar, Aanisah R.Q. Bakry, Brenda M. Samiadji, Demas A. Anugerah, M. Farhan Alfarizi GH, Indri D. Ayulani, and Sheryl N. Salim. A mathematical study on the spread of covid-19 considering social distancing and rapid assessment: The case of jakarta, indonesia. *Chaos, Solitons & Fractals*, 139:110042, 2020.

[2] M. E. Alexander and S. M. Moghadas. Bifurcation analysis of an sirs epidemic model with generalized incidence. *SIAM Journal on Applied Mathematics*, 65(5):1794–1816, 2005.

[3] Ebrahem A. Algehyne and Muhammad Ibrahim. Fractal-fractional order mathematical vaccine model of covid-19 under non-singular kernel. *Chaos, Solitons & Fractals*, 150:111150, 2021.

[4] Joshua Kiddy K. Asamoah, Zhen Jin, Gui-Quan Sun, Baba Seidu, Ernest Yankson, Afeez Abidemi, F.T. Oduro, Stephen E. Moore, and Eric Okyere. Sensitivity assessment and optimal economic evaluation of a new covid-19 compartmental epidemic model with control interventions. *Chaos, Solitons & Fractals*, 146:110885, 2021.

[5] Joshua Kiddy K. Asamoah, Farai Nyabadza, Zhen Jin, Ebenezer Bonyah, Muhammad Altaf Khan, Michael Y. Li, and Tasawar Hayat. Backward bifurcation and sensitivity analysis for bacterial meningitis transmission dynamics with a nonlinear recovery rate. *Chaos, Solitons & Fractals*, 140:110237, 2020.

[6] Cristiane M. Batistela, Diego P.F. Correa, Atila M Bueno, and Jose Roberto C. Piqueira. Sirsi compartmental model for covid-19 pandemic with immunity loss. *Chaos, Solitons & Fractals*, 142:110388, 2021.

[7] Stelios Bekiros and Dimitra Kouloumpou. Sbdiem: A new mathematical model of infectious disease dynamics. *Chaos, Solitons & Fractals*, 136:109828, 2020.

[8] Seo Yoon Chae, KyoungEun Lee, Hyun Min Lee, Nam Jung, Quang Anh Le, Biseko Juma Mafwele, Tae Ho Lee, Doo Hwan Kim, and Jae Woo Lee. Estimation of infection rate and

predictions of disease spreading based on initial individuals infected with covid-19. *Frontiers in Physics*, 8:311, 2020.

[9] Paolo Di Giamberardino and Daniela Iacoviello. Evaluation of the effect of different policies in the containment of epidemic spreads for the covid-19 case. *Biomedical Signal Processing and Control*, 65:102325, 2021.

[10] Shifei Ding, Yanju Xiao, Weipeng Zhang, Guifeng Deng, and Zhehua Liu. Stability and bogdanov-takens bifurcation of an sis epidemic model with saturated treatment function. *Mathematical Problems in Engineering*, 2015.

[11] Jana L. Gevertz, James M. Greene, Cynthia H. Sanchez-Tapia, and Eduardo D. Sontag. A novel covid-19 epidemiological model with explicit susceptible and asymptomatic isolation compartments reveals unexpected consequences of timing social distancing. *Journal of Theoretical Biology*, 510:110539, 2021.

[12] Giulia Giordano, Franco Blanchini, Raffaele Bruno, Patrizio Colaneri, Alessandro Di Filippo, Angela Di Matteo, and Marta Colaneri. Modelling the covid-19 epidemic and implementation of population-wide interventions in italy. *Nature Medicine*, 26(6):855–860, Jun 2020.

[13] Abba B. Gumel, Enahoro A. Iboi, Calistus N. Ngonghala, and Elamin H. Elbasha. A primer on using mathematics to understand covid-19 dynamics: Modeling, analysis and simulations. *Infectious Disease Modelling*, 6:148–168, 2021.

[14] Mahesh Jayaweera, Hasini Perera, Buddhika Gunawardana, and Jagath Manatunge. Transmission of COVID-19 virus by droplets and aerosols: A critical review on the unresolved dichotomy. *Environmental Research*, 188:109819, September 2020.

[15] Xiaoyu Jiang, Yang Yu, Fanwei Meng, and Yancong Xu. Modelling the dynamics of avian influenza with nonlinear recovery rate and psychological effect. *Journal of Applied Analysis & Computation*, 10(3):1170–1192, 2020.

[16] Semu M. Kassa, John B.H. Njagarah, and Yibeltal A. Terefe. Analysis of the mitigation strategies for covid-19: From mathematical modelling perspective. *Chaos, Solitons & Fractals*, 138:109968, 2020.

[17] Pushpendra Kumar, Vedat Suat Erturk, and Marina Murillo-Arcila. A new fractional mathematical modelling of covid-19 with the availability of vaccine. *Results in Physics*, 24:104213, 2021.

[18] Min Lu, Jicai Huang, Shigui Ruan, and Pei Yu. Bifurcation analysis of an sirs epidemic model with a generalized nonmonotone and saturated incidence rate. *Journal of Differential Equations*, 267(3):1859 – 1898, 2019.

[19] Manotosh Mandal, Soovoojeet Jana, Swapan Kumar Nandi, Anupam Khatua, Sayani Adak, and T.K. Kar. A model based study on the dynamics of covid-19: Prediction and control. *Chaos, Solitons & Fractals*, 136:109889, 2020.

[20] Mohd Hafiz Mohd and Fatima Sulayman. Unravelling the myths of r0 in controlling the dynamics of covid-19 outbreak: A modelling perspective. *Chaos, Solitons & Fractals*, 138:109943, 2020.

[21] Sk Shahid Nadim and Joydev Chattopadhyay. Occurrence of backward bifurcation and prediction of disease transmission with imperfect lockdown: A case study on covid-19. *Chaos, Solitons & Fractals*, 140:110163, 2020.

[22] Bianca Nogrady. https://www.nature.com/articles/d41586-020-03141-3, 2020. [Online; accessed 11-November-2020].

[23] A.G.C. Perez, E. Avila-Vales, and G.E. Garcia-Almeida. Bifurcation analysis of an sir model with logistic growth, nonlinear incidence, and saturated treatment. *Complexity*, 2019, 2019.

[24] Arman Rajaei, Mahsa Raeiszadeh, Vahid Azimi, and Mojtaba Sharifi. State estimation-based control of covid-19 epidemic before and after vaccine development. *Journal of process control*, 102:1–14, Jun 2021.

[25] Piu Samui, Jayanta Mondal, and Subhas Khajanchi. A mathematical model for covid-19 transmission dynamics with a case study of india. *Chaos, Solitons & Fractals*, 140:110173, 2020.

[26] Novi Reandy Sasmita, Muhammad Ikhwan, Suyanto Suyanto, and Virasakdi Chongsuvivatwong. Optimal control on a mathematical model to pattern the progression of coronavirus disease 2019 (covid-19) in indonesia. *Global Health Research and Policy*, 5(1):38, Aug 2020.

[27] P. van den Driessche and James Watmough. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*, 180(1):29–48, 2002.

[28] A. Wang, Y. Xiao, and R. Smith. Multiple equilibria in a non-smooth epidemic model with medical-resource constraints. *Bulletin of Mathematical Biology*, 81(4):963–994, 2019.

[29] Phitchayapak Wintachai and Kiattisak Prathom. Stability analysis of seir model related to efficiency of vaccines for covid-19 situation. *Heliyon*, 7(4):e06812, 2021.

[30] NA YI, ZHIWU ZHAO, and QINGLING ZHANG. Bifurcations of an seiqs epidemic model. *International Journal for Information and Systems Sciences*, 5(3-4):296–310, 2009.

[31] Mengyun Zhang, J. Ge, and Z. Lin. The impact of the number of hospital beds and spatial heterogeneity on an sis epidemic model. *Acta Applicandae Mathematicae*, 167:59–73, 2020.