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

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The discovery of a new form of corona-viruses in December 2019, SARS-CoV-2, commonly named COVID-19, has reshaped 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 more than one year. WHO (World Health Organization) is advertising vaccine as a safe and effective measure to fight off the virus. Saudi Arabia was the fourth country in the world to start to vaccinate its population. Even with the new simplified COVID-19 rules, the third dose is still mandatory. COVID-19 vaccines have raised many questions regarding in its efficiency and its role to reduce the number of infections. In this work,we try to answer these question and 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 numerical analysis of the model based on the Saudi case. Our developed model shows that the vaccine reduces the transmission rate and provides an explanation to the rise in the number of new infections immediately after the start of the vaccination campaign in Saudi Arabia.

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4   **Abstract**

5   The discovery of a new form of corona-viruses in December 2019, SARS-CoV-2, commonly
6   named COVID-19, has reshaped the world. With health and economy issues, scientists have been
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15   mathematical model with five compartments, including susceptible, vaccinated, infectious, asymp-
16   totic and recovered individuals. We provide theoretical results regarding the effective reproduction
17   number, the stability of endemic equilibrium and disease free equilibrium. We provide numerical
18   analysis of the model based on the Saudi case. Our developed model shows that the vaccine re-
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21   *keywords: COVID-19; mathematical model; vaccination; stability*

22 1 Introduction

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

32   The complex spreading patterns of COVID-19 and the various spread speed of its variants make
33   its containing and mitigating real challenges. The existing models vary in form and complexity, but
34   the common objective is to provide important information for global health decision makers about
35   the disease dynamics. The first control measure was lockdown and then health authorities imposed
36   mask wearing and social distancing. The authors of [18] showed, by extending the classical SEIR
37   model, that social distance is an important factor to reduce the reproduction number and this to
38   reduce the virus spread. Despite the *herd immunity* acquired via vaccine or infection, the social

distancing is still recommended as a public health measure. 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. The main contributions of this research are given as follows:

- Developing a novel mathematical model to predict the spread of COVID-19, with the presence of vaccinated and asymptotic compartments

- Analyzing the existence of endemic equilibrium point and the stability of disease-free equilibrium

- Investigating a real case study in Saudi Arabia, discussing the impact of vaccination on disease dynamics

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] [25][6] [32]. 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 [37] 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).

82 Recently, Lu *et. al* [23] studied the the SIRS epidemic model, the same considered in [2] but with
83 a generalized non-monotone incidence rate. The incidence rate is a function of the infection force of
84 a disease and the number of susceptible individuals. The given formula for the incidence rate models
85 the psychological pressure of some epidemic disease. The government is, in general, lead to take some
86 protective measures like lockdown when the infection number becomes very high. The authors showed
87 that the model has both repelling and attracting Bogdanov-Takens bifurcations. Moreover, from the
88 super-critical Hopf bifurcation, the authors concluded that a disease following this model presents
89 periodic outbreak, which is very important to understand its dynamics, in the real world.

90 The impact of treatment function was investigated in [10] using SIS model, where recovered in-
91 dividuals become again susceptible and the incidence rate is supposed bi-linear. In the considered
92 model, the treatment function is saturated, which results in the existence of backward bifurcation.
93 Thus, the eradication of the disease is not only related to the reproduction number but also to other
94 biological or epidemiological mechanisms, such as imperfect vaccine. The bifurcation analysis outlines
95 the necessary conditions to eliminate the disease. Zhang *et. al* [38] discussed the impact of the num-
96 ber of hospital beds on SIS epidemic model, by considering a nonlinear recovery rate. The authors
97 calculated the basic reproduction number corresponding to their model. This number determines the
98 condition for the disease-free equilibrium to be globally asymptotically stable.

99 The limitations of medical resources, mainly the availability of vaccinations, is modeled using a
100 piecewise-defined function for patient treatment in [35]. This function admits a backward bifurcation
101 with limited available medical resources. The variation of vaccination threshold affects the existence
102 of multiple steady states,crossing cycle, and generalized endemic equilibria. Similarly, Perez *et al*.
103 [29] considered nonlinear incidence rate for a generalized SIR model. Besides, the authors assumed
104 that the model has saturated Holling type II treatment rate and logistic growth. Non linear and
105 saturated functions allows to represent more accurately the dynamics epidemic diseases. Similar to
106 previous stated works, the authors revealed the importance of the basic reproduction number R_0,
107 whose value determines the existence of endemic equilibrium and the stability of the disease-free
108 equilibrium. Under some conditions related to the disease transmission rate and the treatment rate,
109 the model may undertake a backward bifurcation and a Hopf bifurcation. The above-mentioned
110 articles considered general disease models. In the literature, we can also find specific models targeting
111 particular disease such as avian influenza [15] and bacterial meningitis [5]. Since the declaration of
112 World Health Organization (WHO) of the Severe Acute Respiratory Syndrome Coronavirus (SARS-
113 CoV-2) as a pandemic on March 2020, the scientific community has been trying to understand the
114 dynamics of this virus.

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

121 Nadim and Chattopadhyay [27] investigated the effect of imperfect lockdown. In the adopted
122 model, when the basic reproduction number, R_0 is less than unity, the stable disease free equilibrium
123 coexists with a stable endemic equilibrium. This means that COVID-19 undergoes backward bifur-
124 cation. This phenomenon was observed in the Kingdom of Saudi Arabia where the new cases were
125 decreasing to reach 97 in 06, January 2021. Unfortunately, this rate reached 386 new cases, after one
126 month, which obliged the Ministry of Health to declare partial lockdown for 10 days. The infection
127 force is so high that the disease cannot be totally eradicated. The authors showed that under perfect
128 lockdown, this backward bifurcation does not exist, but such condition is not possible in the real
129 world. In [9], the authors included in their mathematical model, based on the classical SEIR, several

130   prevention actions such as test campaign on the population and quarantining infected persons. The
131   model took in consideration infection treatment efforts, such as vaccination and the therapy of induced
132   cardio-respiratory complications. Besides the usual classes of the population, the authors considered
133   two new classes, driven by specific characteristics of the virus: infected but asymptomatic patients and
134   suspected infected individuals. The theoretical results, tuned using the Chinese case, were compared
135   to United Kingdom case and the Italian case, showing the similarity between the model dynamics and
136   the real epidemic behaviour. Lü *et. al* [24] proposed an epidemic model that distinguishes between
137   the first and the second waves od COVID-19 in China. The two-stage model includes *Contacts* com-
138   partment, besides the usual Susceptible, Infectious and Recovered compartments. The authors of [17]
139   raised the issue of undetected symptomatic and asymptomatic individuals. They also investigated the
140   effect of two control strategies that include the improvement of treatment for infected and isolated
141   individuals. Another scientific aspect of COVID-19 is the possible transmission of the virus through
142   contaminated surfaces. It is believed that the virus can survive several days on the surfaces depending
143   on the material (wood, glass or plastic). Another issue faced by the governments is the awareness level
144   of the population. Some individuals, deliberately, decide not to apply the precautionary measures,
145   mainly wearing mask and respecting social distancing [16].

146   The issue of the efficiency of social distancing and rapid testing strategies against the pandemic was
147   examined in [1], where the authors extended the standard SEIR model. The authors considered also
148   the problem of undetected asymptomatic individuals, who have no symptoms but participate actively
149   to virus spread. Furthermore, the limitation of medical resources was incorporated to the model. The
150   theoretical findings emphasized the role of the basic reproduction number R_0 in the existence of stable
151   COVID-19 free and COVID-endemic equilibrium points. This conclusion is contested by Mohd and
152   Sulaiman [26], who studied the SIRS model with limited medical resources and false detection issues.
153   The authors showed that the condition of reducing the basic reproduction number under the unity
154   value is necessary to eliminate the disease but not sufficient.

155   Since the authorization COVID-19 vaccines, several research works focused on giving insight to
156   mathematical characteristic of virus spread after population vaccination. Algehyne *et al*. [3] used
157   nonlinear functional analysis and fractal fractional derivative to model the evolution over time of four
158   compartments: susceptible, infected, infected positive tested, and recovered. The Spanish case was
159   investigated in [19] using also fractional derivatives. It is important to highlight that these works do
160   not consider vaccinated state as a separate compartment. They rather consider that the vaccinated
161   individuals are moved from susceptible to recovered compartment. The vaccinated individuals are
162   considered to move also from exposed state in [36].

163   Different mathematical tools are used by Rajaei *et al*. [31] to compare the effect of vaccination
164   with social distancing and hospitalization. Extended Kalman filter (EKF) is used for state estimation
165   under uncertainty.

166   Most of the existing research works developing a relationship between infectious and asymptotic
167   individuals focus on estimating the model parameters using actual data [4] [11] [33] [12]. To the best
168   of our knowledge, our work is the first to provide to study mathematical stability of endemic and
169   disease free equilibrium. Most relevant works in COVID-19 mathematical modeling are compared in
170   Table 1, in terms of disease control strategy, considered model and country of the case study.

3   Proposed model and effective reproduction number

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

Ref.	Control Measure	Model	Country of case study
[27]	lockdown	SEIR+Hospitalized+Lockdown	India, Mexico, South Africa and Argentina
[9]	test campaign and quarantine	SEIR+ Quarantined	UK, China, Italy
[11]	social distancing	SAIR	-
[16]	social distancing and mask wearing	SEIR+ Asymptotic	WHO
[1]	social distancing and rapid testing	SEIR+ Asymptotic	Indonesia
[4]	social distancing	SEIR+Quarantined+Hospitalized	Egypt and Ghana
[11]	social distancing	SAIR	WHO
[3]	vaccination	SIR	Brasil
[19]	vaccination	SEIRS+Death	Spain
[31]	vaccination	SEIR+Quarantined+Hospitalized	Canada
Our research	Vaccination	SAIR+Vaccinated	Saudi Arabia

Table 1: Most relevant studies in the field of COVID-19 prediction trend

Table 2 and Table 3 summarize the different model parameters and variables.

Parameters

Parameter	Description
Λ	Recruitment rate of susceptible humans
μ	Natural mortality 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 2: Model parameters and description.

Variables

Variable	Description
β	Transmission rate for Infectious
α	Transmission rate for Asymptotic
ψ	Vaccination coverage rate

Table 3: Model variables and description.

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

The virus may infect vaccinated individuals but at a lower rate than susceptible individuals who are not unvaccinated. Thus in this case, the transmission rates β and α are multiplied by a scaling

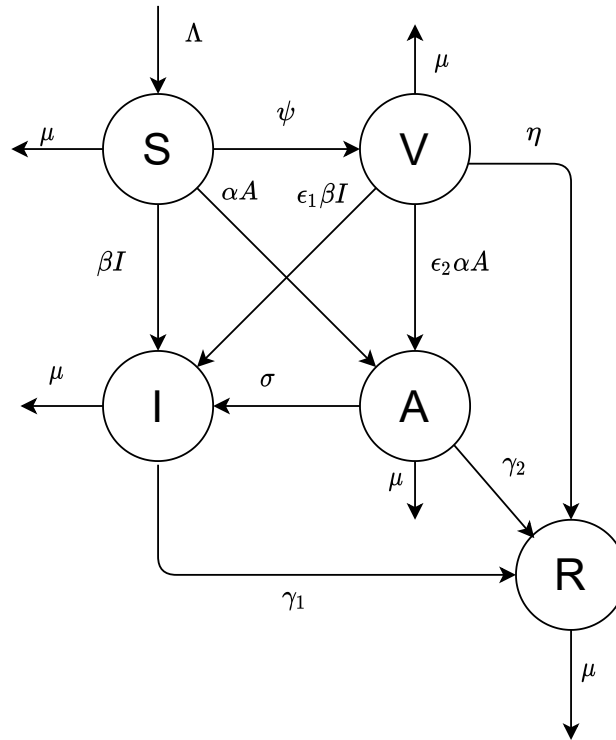



Figure 1: Proposed model.

factor ϵ_1 and ϵ_2 ($0\leq \epsilon_1,\ \epsilon_2 \leq 1$).

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

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

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

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

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

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

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

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 of appearance of new infections in each compartment. The progression from A to I is not considered to be new infection, but rather the progression of an infected individual through various infectious compartments.

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

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

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

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

Our developed model is similar to the two-strain model in [34] with two infectious compartments. FN^{-1}, the next generation matrix of system (3.1) has the two eigenvalues.

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

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

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:

$$\Lambda - \mu S - \psi S - \beta IS - \alpha AS = 0 \tag{4.1a}$$

$$-\mu V + \psi S - \eta V - \epsilon_1 \beta IV - \epsilon_2 \alpha AV = 0 \tag{4.1b}$$

$$-\mu I + \beta IS + \epsilon_1 \beta IV + \sigma A - \gamma_1 I = 0 \tag{4.1c}$$

$$-\mu A + \alpha AS - \sigma A + \epsilon_2 \alpha AV - \gamma_2 A = 0 \tag{4.1d}$$

$$-\mu R + \eta V + \gamma_1 I + \gamma_2 A = 0 \tag{4.1e}$$

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

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

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

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

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

226   We denote by:

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

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

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

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

$$P(I) = aI^2 + bI + c = 0 \tag{4.2}$$

232 .

233   The number of endemic equilibria of the considered system depends on parameter values a, b,
234 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

235 $R_2 = R_{20}\frac{\mu+\eta+\epsilon_2\psi}{\mu+\psi}$

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

237   Since the model parameters A and I are positive, it follows that D >0 and a > 0. Furthermore, if
238 $R_2 > 1$, 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
239 $\psi < \psi_{crit}$, then $R_2 > 1$. We deduce that for $R_2 > 1$, P(I) has a unique positive root.
240   If $R_2 < 1$, we have $c>0$ and $\psi \geq \psi_{crit}$. Since $b(\psi)$ is an increasing function of ψ, if $b(\psi_{crit}) \geq 0$,
241 then $b(\psi) > 0$ for $\psi > \psi_{crit}$. In this case, P(I) has no positive real root and the system have no
242 endemic equilibrium.
243   We consider now the case where $b(\psi_{crit}) < 0$. We denote by $\Delta(\psi) \overset{def}{=} b^2(\psi) - 4ac(\psi)$. If $c(\psi_{crit}) = 0$,
244 $\Delta(\psi_{crit}) > 0$. Since $b(\psi)$ is an increasing linear function of ψ, there is a unique $\bar{\psi} > \psi_{crit}$ such that
245 $b(\bar{\psi}) = 0$. and $\Delta(\psi)$ has a unique root $\bar{\bar{\psi}}$ in $[\psi_{crit}, \bar{\bar{\psi}}]$.
246   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
247 no real positive root and the system (3.1) has no endemic equilibria for $\psi > \bar{\bar{\psi}}$.
248   If $R_2 = 1$, we have c=0. In this case, system has a unique endemic equilibrium for $b(\psi) < 0$ and
249 no endemic equilibrium for $b(\psi) > 0$.

250   4.2   Stability of disease-free equilibrium

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

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

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

254   The characteristic polynomial of the Jacobian matrix at DFE is given by $det(J_0 - \lambda I) = 0$, where
255 λ is the eigenvalue and I is 5×5 identity matrix. Thus, J_0 has eigenvalues given by:
256    $\lambda_1 = -\mu$
257    $\lambda_2 = -(\mu+\eta)$
258    $\lambda_3 = -(\mu+\psi)$
259    $\lambda_4 = -(\mu+\gamma_1) + \beta(S_0+\epsilon_1 V_0) = (\mu+\gamma_1)(R_1 - 1)$
260    $\lambda_5 = -(\mu+\sigma+\gamma_2) + \alpha(S_0+\epsilon_2 V_0) = (\mu+\sigma+\gamma_2)(R_2 - 1)$
261   All the eigenvalues are strictly negative except for λ_4 and λ_5. These eigenvalues depend the sign
262 of (R_2-1) and (R_1-1). The stability of the DFE represents the dynamics of disease free population

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

265 **Theorem 1** *Based on the Theorem of [34], we have the following results. If $R_1 > 1$ or/and $R_2 > 1$,*
266 *then λ_4 or/and λ_5 is/are strictly positive. In this case the DFE is unstable. If $R_1 < 1$ and $R_2 < 1$,*
267 *then λ_4 and λ_5 are strictly negative. The system is locally asymptotically stable.*

5 Numerical simulations

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

275 The death and birth rate for Saudi Arabia are estimated to be equal to 3.39 and 14.56 for 1000 per
276 year, respectively. The vaccination campaign started on 18/12/2020 with a vaccination coverage of the
277 total population of 0.02% to reach about 65% of the adult population fully vaccinated in November
278 2021. The vaccination rate is considered a the percentage of the total population that get vaccinated
279 per day. With approximately 45000 administrated doses per day and a total population of 35339000
280 in 2021, this rate is about 0.00 127. Is this rate enough to eradicate the disease? This what we are
281 trying to answer is this work.

282 The estimation of model parameters is very important to have accurate numerical results. One ap-
283 proach consists in calibrating the model by fitting it with reported data using, for example, least square
284 method [30]. In our paper, we used scientific reported facts about COVID-19 transmission mecha-
285 nisms. The research report [28] provides information about asymptotic individuals for COVID-19.
286 Most people, with no symptoms at the beginning, develop symptoms in 7-13 days, which corresponds
287 to the σ^{-1}. Recall that γ_1 is the recovery rate of infectious individuals. Interpreted as the expected
288 value of a Poisson process, γ_1 can be related to the needed time from the beginning of the infection
289 till recovery [11]. With average recovery duration equal to 10 days [8], the recovery rate of infectious
290 individuals is $\gamma_1 = 0.1$.

291 Let ω denote the fraction of asymptotic individuals among positive cases. According to [28], and
292 based on 13 studies involving 21,708 people in 2020, $\omega = 0.17$. Using the same methodology as in [11]
293 $\gamma_2 = \frac{\omega}{1-\omega}\sigma \approx 0.2\sigma$. The asymptotic people are estimated to be 42% less contagious than symptomatic
294 individuals [28]. 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 4: Model parameters and values.

²⁹⁵ By 18/12/2020, considered as time 0 in this model, the number of recoveries is equal to 351722 ,
²⁹⁶ the number of active cases is equal to 3014. Assuming that $\omega = 0.17$, the number of initial infectious
²⁹⁷ with and without symptoms is equal to 2501 and 513, respectively.

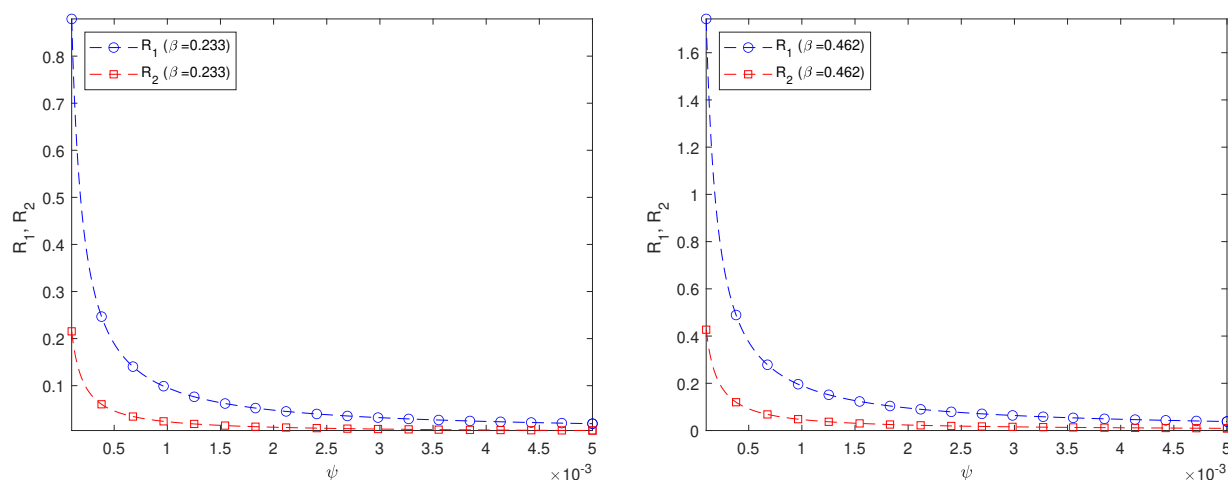
²⁹⁸ First, we investigate the effect of the vaccination rate on the effective reproduction number defined
²⁹⁹ as the maximum of the two entities R_1 and R_2. According to [8], COVID-19 transmission rate β ranges
³⁰⁰ between 0.233 and 0.462. Fig 2a and Fig 2b show the evolution of both R_1 and R_2 as a function of
³⁰¹ the vaccination rate ψ with virus transmission rate equal to 0.233 and 0.462, respectively. In both
³⁰² cases, R_1 corresponding to the strain of infectious individuals with symptoms is greater than R_2,
³⁰³ corresponding to the strain of asymptotic individuals. Thus the number of individuals infected by one
³⁰⁴ person currying the virus is mainly affected by individuals showing usual symptoms. Mathematical
³⁰⁵ theoretical result confirms that the vaccine reduces the spread of the virus among the population.
³⁰⁶ We would like to highlight the fact in our model that a vaccinated individual may be infectious with
³⁰⁷ or without symptoms. This result is very important as, till the end of 2021, an important portion
³⁰⁸ of worldwide population is still opposed to vaccine. In the case of high transmission rate and low
³⁰⁹ vaccination rate, R_1 is higher than 1. The disease free equilibrium is consequently unstable according
³¹⁰ 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,
³¹¹ respectively. When $beta$ is equal to 0.462, R_1 and R_2 are equal to 0.1580 and 0.0387, respectively. For
³¹² the Saudi Arabia, the effective reproduction number is less than 1, even for high transmission rate.
³¹³ 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 $\beta = 0.462$.

³¹⁴ Fig. 3 shows the weekly number of new active cases and recovered after infection in KSA, starting
³¹⁵ from 18/12/2020, the date when the vaccination starts. We can see that after 12 weeks, the number
³¹⁶ of cases raises. This behaviour was surprising for a population waiting to see the effect of vaccination.
³¹⁷ It's only after 31 weeks that the number of new cases start to decrease. The same phenomena was
³¹⁸ observed in both Fig. 4c and Fig. 5c. The theatrical results is conform to actual statistics. The effect
³¹⁹ of vaccination is not immediate; it needs several weeks to observe a decrease in the number of new
³²⁰ infectious cases.

³²¹ We compare the evolution in time of the five compartments (S, V, I, A ,and R) presented in

322 our model, for two different transmission rate and with the Saudi vaccination rate. With different
323 dynamics at the beginning, both scenarios show a convergence to a stable state. We observe almost
324 similar patterns for S, V. I and R. The number of susceptible individuals S decreases slowly at the
325 beginning and then, we observe a drastic decline. Obviously, the number of recovered follows the same
326 slow and then fast pace but in decrease. The number of vaccinated individuals V increases gradually
327 at the beginning and then it begins to fall down. The number of infectious individuals I remain stable
328 for a short period to witness an expansion followed by a decline. The number of asymptotic individuals
329 show different evolution patterns for two considered scenarios. When we set a low value for the virus
330 transmission rate, this number immediately shrinks. However, when we set a high value for the virus
331 transmission rate, this number increases before shrinking.

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

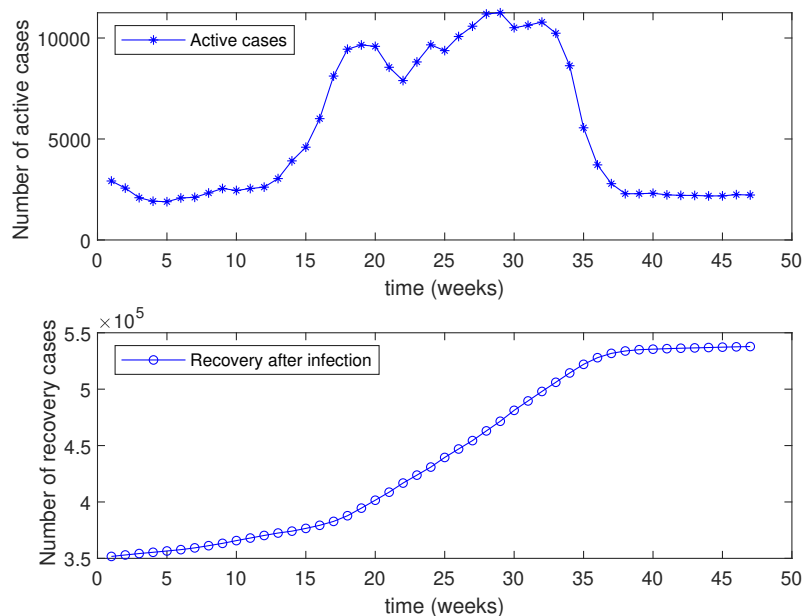
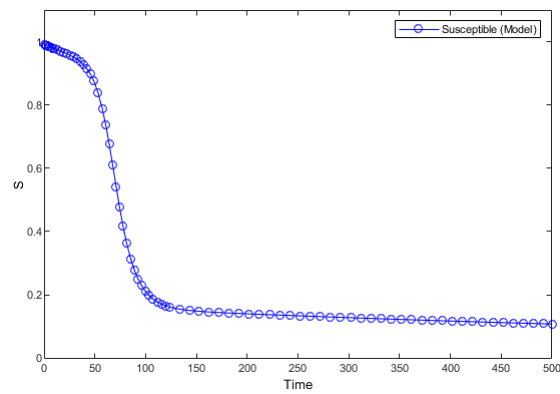
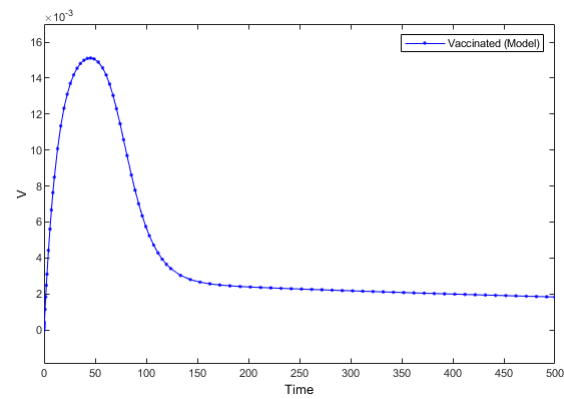
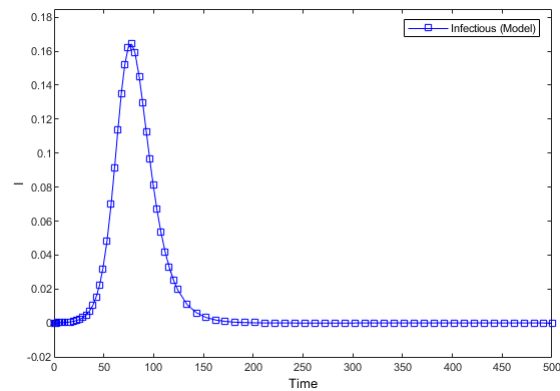
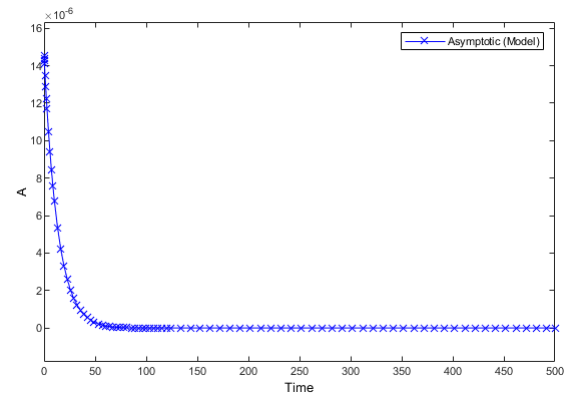



Figure 3: Number of active cases and recovered after infection in KSA. Starting from 18/12/2020.

(a) Susceptible

(b) Vaccinated

(c) Infectious

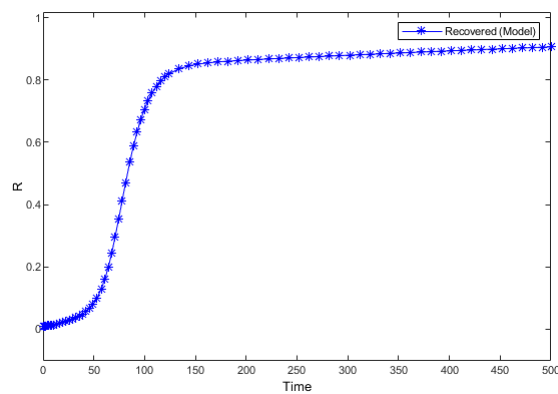
(d) Asymptotic

(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

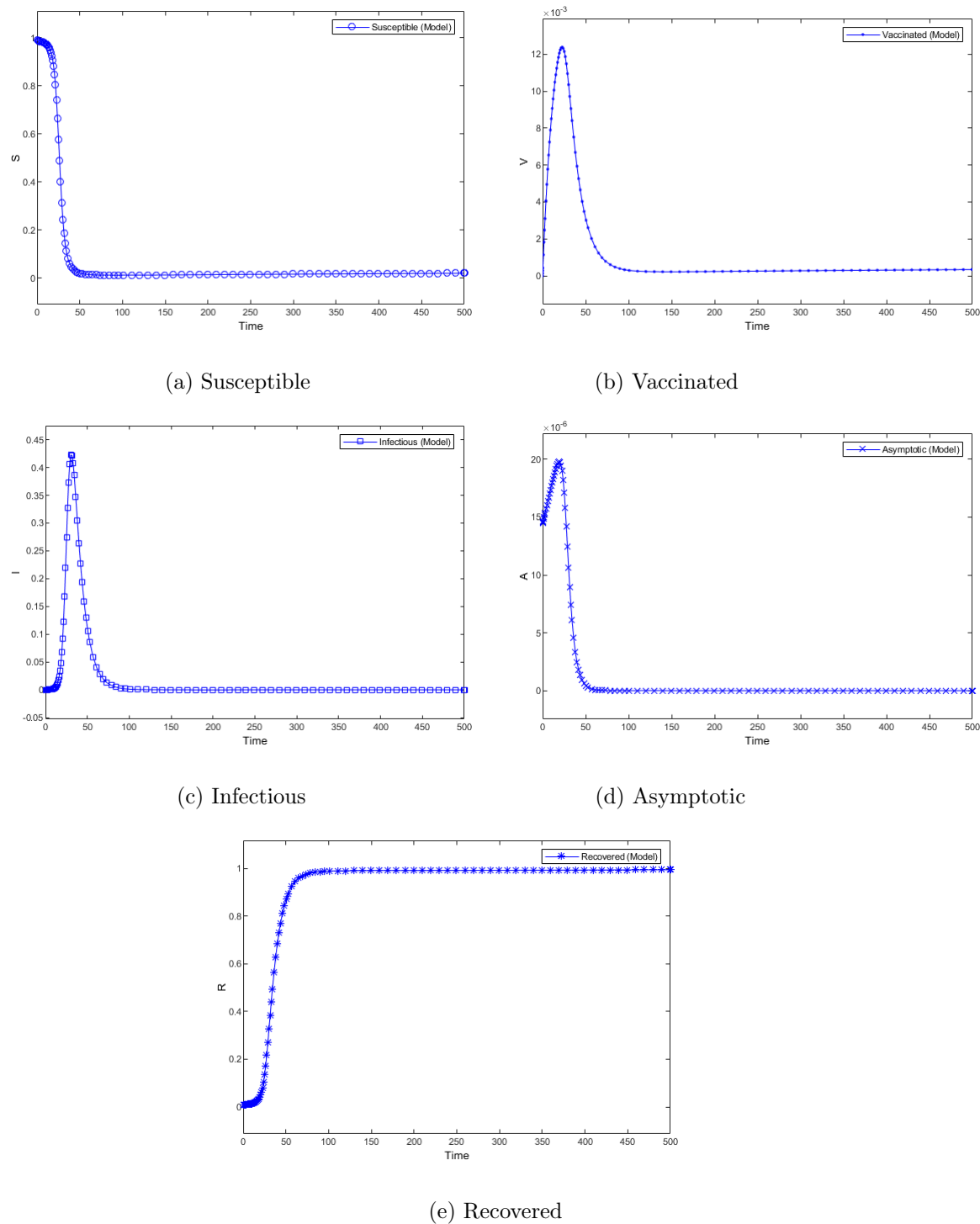
(d) Asymptotic

(e) Recovered

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

5.1   Managerial insights and practical implications

By March 2022, several countries, including Saudi Arabia, have simplified COVID-19 rules by relaxing some safe management measures such as social distancing and mask wearing. However, the Saudi ministry of health kept the condition of three vaccine doses. This decision is part of a public health strategy that continues to monitor virus variants, mainly Omicron and its sublineages BA.1 and BA.2. WHO's Technical Advisory Group requests countries to continue to be vigilant because of potential significant rise in the number of infections. Although these variants might resist neutralizing antibodies in the blood, the vaccine allows preventing severe illness and death. Unlike several countries, Saudi Arabia was spared from the lack of oxygen generators, the most important medical equipment for hospitalized patients. The main concern is, however, regulations regarding vaccination. The vaccine was made mandatory for individuals over 12 years old. The open question is the need for vaccine for children aged from 5 to 11 years. Therefore, managers need to simulate the effect of vaccination and make predictions of the virus spread. This work tries to provide them with an efficient tool that captures the specificity of the Saudi case.

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. The main findings of this work are as follows.

- People can observe that the vaccine helps to reduce the severity of symptoms. We gave a mathematical proof that the vaccine reduces the transmission rate.

- The vaccination campaign in Saudi Arabia was immediately followed by the rise in the number of infections. We have showed that this observation is mathematically justified and this rise is a necessary transition before the increase of new infections.

- By adjusting the model parameters based on collected data, we provide the decision-makers with the vaccination rate necessary for virus spread control.

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. As discussed in the previous section, several countries including Saudi Arabia did not face medical resource shortages. However, as highlighted by Lotfi [20], the management of medical waste is critical during COVID-19 pandemic. Moreover, the number of asymptomatic and untested symptomatic infections is uncertain. As future work, we propose to capture disease dynamic uncertainty and incorporate risk assessment, to alleviate the impacts of pandemic peak. Hybrid fuzzy, data-driven, robust optimization, and stochastics [21] [22] are examples of uncertainty analysis methods. Conditional value at risk (CVaR), which is the average shortfall beyond the VaR point, is a consistent and coherent risk assessment measure.

Data availability

The data set is publically available at this link: https://datasource.kapsarc.org/explore/dataset/saudi-arabia-coronavirus-disease-covid-19-situation/table/?disjunctive.daily_cumulative&disjunctive.indicator&disjunctive.event&disjunctive.city_en&disjunctive.region_en&sort=date

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.

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