The Impact of Vaccination on Covid-19 Disease Transmission Patterns in a Human Population: A Theoretical Analysis

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Authors’ contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

Article Information

DOI: 10.9734/arjom/2021/v17i1030332

Editor(s):
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Complete Peer review History, details of the editor(s), Reviewers and additional Reviewers are available here: https://www.sdiarticle5.com/review-history/75356

Received 04 September 2021
Accepted 08 November 2021
Published 27 November 2021

Abstract

We construct a Mathematical model that describes the effect of vaccination on the dynamics of the transmission of COVID-19 disease in a human population. The model is a system of ordinary differential equations that describes the evolution of humans in a range of Covid-19 states due to emergence of an index case in a disease free region. The analysis of the model shows that effective vaccination can lead to disease eradication, where in the disease free state is locally asymptotically stable if the basic reproductive number, $R_0 < 1$ and unstable when $R_0 > 1$. The numerical simulations suggests the use of other social measures alongside vaccination in order to avert the possibility of the disease becoming endemic.

Keywords: Covid-19; modeling; vaccination; re-infection; symptomatic; asymptomatic; surface virus.
1 Introduction

The Coronavirus disease, known as Covid-19, is an Infectious disease caused by Severe Acute Respiratory Syndrome Coronavirus, which is a new virus of the same family as SARS-CoV2 [1], believed to have a zoonotic origin [2,3] and was identified and named by the World Health Organization (WHO) on January 10, 2020 following an earlier virus borne infection episode in Wuhan, China in December, 2019 [4]. The COVID-19 pandemic is considered as the biggest global threat worldwide because of thousands of confirmed infections, accompanied by thousands of deaths over the world [5]. Globally, as at 10th September 2021, there have been 223,022,538 confirmed cases of Covid-19 with 4,602,882 disease related deaths [6].

Nonlinear ordinary differential equations have been used to explore the complex mechanisms of the dynamics of various systems in multidisciplinary fields: for instance, they are used in economics [7], quantum physics [8], chaos [9], medicine [10] and health diseases [11]. These models aim to make an optimal predictive control of the parameters influencing the system dynamics.

In the work of [1], an epidemiological compartmental model that takes into account a super-spreading phenomenon of some individuals including fatality and hospitalized classes was proposed. The sensitivity analysis of their model shows that the most sensitive parameters to the basic reproduction number are infection rate of humans, the rate at which exposed humans become infectious and the disease related death rate. Increase in the infection rate and the rate at which exposed individuals become infectious increase the basic reproduction number, and in contrast, the disease related death rate and the basic reproduction number are inversely related.

A Bat – Reservoir population transmission model was proposed in [12], to understand and simulate potential transmission from zoonotic source to humans. They estimated the basic reproductive number \( R_0 \) as 2.4829. This value differs from 3.58, being the value estimated in [2]. The work of [1] suggests isolation and lockdown as a means of control of Covid-19 pandemic whereas some SIR models on Covid-19 have been proposed and carefully analyzed in [2,12,13,14]. Lotka-Volterra based models of COVID-19 have been proposed and analyzed in [15].

In the work of [4], the authors noted that, Covid-19 pandemic ravaging the world currently, will not end soon, as the result of their work shows damping oscillations. They aver that vaccination could be a possible remedy. Vaccination is substantially effective if we consider the other things that affect the disease such as wearing facemask, maintaining social distance, etc. [10].

Here, we include Vaccination and possible Re-infection of Recovered Covid-19 Patient alongside other features considered in [4]. We only consider human to human transmission of the disease following the introduction of an index case rather than infection from a Bat population. Brief introduction and model formulation are considered in Section 1 and Section 2 respectively. While in section 3, we present the model analysis, numerical simulations and discussions, followed by a brief conclusion in section 4.

2 Model Formulation

This model is intended to describe the progression of the disease over a period of infection following the introduction of an index case in an entirely susceptible population. As in other infectious disease models, we make assumptions concerning birth and natural death processes as well as other disease kinetics. We employ the principle of mass action including a correction term that describes the logistic population growth rate in the absence of the disease.

The total human population, \( H(t) \), is divided into 6 classes namely, a non-infectious susceptible class, \( P(t) \), a non-infectious latent class, \( E(t) \), an infectious symptomatic class, \( S(t) \), an infectious asymptomatic class \( A(t) \), a non-infectious Vaccinated class \( C(t) \) and a Recovered class \( R(t) \); Hence:

\[
H = P + E + S + A + C + R
\]
The State variables in the model are real and non-negative. Their description is given in Table 1 and the movement between compartments is summarized in Fig. 1, with the individual pathways discussed below.

**Table 1. List of model variable**

<table>
<thead>
<tr>
<th>State Variables Description</th>
<th>State Variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Human Population</td>
<td>H</td>
</tr>
<tr>
<td>Latent or Exposed non-infectious Human Population</td>
<td>E</td>
</tr>
<tr>
<td>Susceptible Human Population</td>
<td>P</td>
</tr>
<tr>
<td>Symptomatic infectious Human Population</td>
<td>S</td>
</tr>
<tr>
<td>Asymptomatic infectious Human Population</td>
<td>A</td>
</tr>
<tr>
<td>Recovered but Susceptible Human Population</td>
<td>R</td>
</tr>
<tr>
<td>Number of Viruses on Surfaces</td>
<td>V</td>
</tr>
<tr>
<td>Vaccinated human Population</td>
<td>C</td>
</tr>
<tr>
<td>time</td>
<td>t</td>
</tr>
</tbody>
</table>

Fig. 1. Pathway diagram of the COVID-19 model showing (a) the progression (solid) and transmission (dashed) of the disease between compartments; the variable names are listed in Table 1. The connecting arrows are labelled with the associated rate constants, where the natural death of each of the classes are not shown for clarity.

Susceptible humans get infected by contacting infectious humans and viruses from surfaces at a rates $\theta_1 \frac{S}{H} P, \theta_2 \frac{A}{H} P$ and $\nu V P$, where $\theta_1, \theta_2$ and $\nu$ are rate constants. The fractions $\frac{S}{H}$ and $\frac{A}{H}$ are the probabilities that the contacts are with symptomatic and asymptomatic humans. We note that humans in class $E$ are in the exposed stage of infection and are not infectious. Susceptible humans are recruited into the population through a constant birth rate, $\lambda_1$, with a correction term $\theta_3 H^2$, stopping the population from growing without limit in the absence of the disease, where $\theta_3$ is per capita resource availability for the human population. Incubating humans become infectious after a mean latency time, $\omega_1$, where a proportion, $k$, of them become asymptomatic. This assumption is different from that of [12], where they suggested two incubation period even though they meant a single incubation period.
Table 2. List of model parameter

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
<th>Value</th>
<th>Units</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\lambda_1$</td>
<td>Per capita birth rate</td>
<td>0.0000433</td>
<td>$Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\theta_1$</td>
<td>Infectious rate between Susceptible and symptomatic human population</td>
<td>0.05</td>
<td>$Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\theta_2$</td>
<td>Infectious rate between Susceptible and Asymptomatic human population</td>
<td>0.124</td>
<td>$Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\varphi_1$</td>
<td>Infectious rate between surface virus and Susceptible Human population</td>
<td>0.00000123</td>
<td>$Virus^{-1}Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>Recovery rate of Symptomatic human population.</td>
<td>0.0987</td>
<td>$Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\beta_2$</td>
<td>Recovery rate of Asymptomatic human population</td>
<td>0.854</td>
<td>$Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\theta_3$</td>
<td>Per capita Resources available for the human Population</td>
<td>0.00024</td>
<td>$Human^{-1}Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\mu_1$</td>
<td>Natural death rate</td>
<td>0.0000357</td>
<td>$Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\tau_1$</td>
<td>Rate of loss of Asymptomatic status</td>
<td>0.035</td>
<td>$Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$k$</td>
<td>Proportion of exposed Human becoming</td>
<td>0.005</td>
<td>Non-dimensional</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\alpha_1$</td>
<td>Contribution of Symptomatic humans to surface Viruses</td>
<td>0.0398</td>
<td>$VirusesHuman^{-1}Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\alpha_2$</td>
<td>Contribution of Asymptomatic humans to surface Viruses</td>
<td>0.001</td>
<td>$VirusesHuman^{-1}Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\varphi_2$</td>
<td>Infectious rate between surface virus and Susceptible Human population</td>
<td>0.00000123</td>
<td>$Virus^{-1}Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\omega_1$</td>
<td>Transition rate from exposed State to infectious State</td>
<td>0.000479</td>
<td>$Virus^{-1}Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\gamma_1$</td>
<td>Mortality rate of virus on surfaces</td>
<td>0.01</td>
<td>$Day^{-1}$</td>
<td>[12,4]</td>
</tr>
<tr>
<td>$\sigma_1$</td>
<td>Disease induced death rate</td>
<td>0.043</td>
<td>$Day^{-1}$</td>
<td>[16]</td>
</tr>
<tr>
<td>$\eta_1$</td>
<td>Rate at which susceptive humans are vaccinated.</td>
<td>0.0196</td>
<td>$Human^{-1}Day^{-1}$</td>
<td>Calculated from [6,17]</td>
</tr>
<tr>
<td>$\rho_1$</td>
<td>Rate at which recovered humans are been susceptible</td>
<td>0.084</td>
<td>$Day^{-1}$</td>
<td>assumed</td>
</tr>
<tr>
<td>m</td>
<td>Proportion of humans that were not vaccinated as a result of Conspiracy theory.</td>
<td>0.98</td>
<td>$Day^{-1}$</td>
<td>Calculated from [2]</td>
</tr>
<tr>
<td>$\eta_2$</td>
<td>Vaccine wearing out rate</td>
<td>0.08</td>
<td>$Day^{-1}$</td>
<td>assumed</td>
</tr>
<tr>
<td>$\beta_3$</td>
<td>Rate of vaccination of recovered humans</td>
<td>0.89</td>
<td>$Day^{-1}$</td>
<td>assumed</td>
</tr>
</tbody>
</table>
All human classes die naturally at per capita rate, $\mu$, while some individuals in the $S$ class die at an additional rate $\sigma_S$ from the disease. We also assume that recovered COVID-19 patients are recruited into the vaccinated class at a rate $\theta R$ or, become susceptible again at a rate $\rho R$ with $\theta$ and $\rho$ as rate constants. Surface viruses die at rate $\gamma V$ while symptomatic and asymptomatic humans contribute to the emergence of surface viruses at rates $\alpha S$ and $\alpha A$ respectively with $\alpha_1$ and $\alpha_2$ as rate constants. Susceptible Humans are vaccinated at a rate $\eta (1 - m)P$, where $\eta_1$ as rate constant and $m$ is the proportion of people unwilling to be vaccinated. We assume that vaccinated humans become susceptible at a rate $\eta_2 C$ as the effectiveness of the vaccine wears out. The proposed model consistent with the above assumptions is given as:

$$\frac{dP}{dt} = \lambda H + \rho_1 R + \eta_2 C - \left(\theta_1 \frac{S}{P} + \theta_2 \frac{A}{P} + \varphi V + \eta_1 (1 - m) + \mu_1\right)P - \theta_3 H^2$$  \hspace{1cm} (2.2)

$$\frac{dE}{dt} = \left(\theta_1 \frac{S}{P} + \theta_2 \frac{A}{P} + \varphi V\right)P - (\omega_1 + \mu_1)E$$  \hspace{1cm} (2.3)

$$\frac{dS}{dt} = (1 - k) \omega_1 E + \tau_1 A - (\beta_1 + \sigma_1 + \mu_1)S$$  \hspace{1cm} (2.4)

$$\frac{dA}{dt} = k \omega_1 E - (\beta_2 + \tau_1 + \mu_1)A$$  \hspace{1cm} (2.5)

$$\frac{dC}{dt} = \eta_1 (1 - m)P + \theta_4 R - (\eta_2 + \mu_2)C$$  \hspace{1cm} (2.6)

$$\frac{dR}{dt} = \beta_1 S + \beta_2 A - (\theta_4 + \rho_1 + \mu_3)R$$  \hspace{1cm} (2.7)

$$\frac{dV}{dt} = \alpha_1 S + \alpha_2 A - \varphi_2 VP - \gamma_1 V$$  \hspace{1cm} (2.8)

$$\frac{dH}{dt} = (\lambda_1 - \mu_4)H - \sigma_1 S - \theta_3 H^2$$  \hspace{1cm} (2.9)

Equation (2.9) is obtained by adding equations (2.2)-(2.7)

### 2.1 Parameter values and nondimensionalisation

Here we considered the various compartments as fractions of the total Population given by:

$$\bar{P} = \frac{P}{H}, \bar{E} = \frac{E}{H}, \bar{S} = \frac{S}{H}, \bar{A} = \frac{A}{H}, \bar{C} = \frac{C}{H}, \bar{R} = \frac{R}{H}$$

So that

$$\bar{P} + \bar{E} + \bar{S} + \bar{A} + \bar{C} + \bar{R} = 1$$

Let $\bar{v} = \frac{V}{V_0}, \bar{h} = \frac{H}{H_0}, \bar{t} = \frac{t}{t_0}$  \hspace{1cm} (2.10)

Then:

$$\frac{d\bar{P}}{dt} = t_0 \lambda_1 + t_0 \omega_1 \bar{R} + t_0 \eta_2 \bar{C} - t_0 [\theta_1 \bar{S} + \theta_2 \bar{A} + \varphi \bar{V} + \eta_1 (1 - m)] \bar{P} - t_0 \theta_3 \bar{H} \bar{P} + t_0 \sigma_1 \bar{S} \bar{P}$$

$$\frac{d\bar{E}}{dt} = t_0 (\theta_1 \bar{S} + \theta_2 \bar{A} + \varphi \bar{V} \bar{P}) - t_0 (\omega_1 + \lambda_1) \bar{E} + t_0 \theta_3 \bar{H} \bar{P} \bar{E} + t_0 \sigma_1 \bar{S} \bar{E}$$

$$\frac{d\bar{S}}{dt} = t_0 (1 - k) \omega_1 \bar{E} + t_0 \tau_1 \bar{A} - t_0 (\beta_1 + \sigma_1 + \lambda_1) \bar{S} + t_0 \theta_3 \bar{H} \bar{S} \bar{H} + t_0 \sigma_1 \bar{S}^2$$

$$\frac{d\bar{A}}{dt} = t_0 k \omega_1 \bar{E} - t_0 (\beta_2 + \tau_1 + \lambda_1) \bar{A} + t_0 \theta_3 \bar{H} \bar{R} \bar{A} + t_0 \sigma_1 \bar{A} \bar{S}$$
\[
\begin{align*}
\frac{dc}{dt} &= t_0 \eta_1 (1 - m) \hat{R} + t_0 \theta_4 \hat{R} - t_0 \eta_2 \hat{C} - t_0 \lambda_1 \hat{C} + t_0 \theta_3 H_0 \hat{H} \hat{C} + t_0 \sigma_1 \hat{C} \hat{S} \\
\frac{dR}{dt} &= t_0 \beta_3 \hat{S} + t_0 \beta_2 \hat{A} - t_0 (\rho_1 + \lambda_1 + \theta_4) \hat{R} + t_0 \theta_3 H_0 \hat{H} \hat{R} + t_0 \sigma_1 \hat{R} \hat{S} \\
\frac{d\bar{V}}{d\bar{t}} &= \frac{\tau_2 H_0 \alpha_2 \bar{S} \bar{H}}{V_0} \left( \frac{\tau_2 H_0 \alpha_2 \bar{A} \bar{H}}{V_0} \right) - t_0 H_0 \phi_2 \bar{H} \bar{R} - t_0 \gamma_2 \bar{R} \\
\frac{d\bar{B}}{d\bar{t}} &= t_0 (\lambda_1 - \mu_1) \bar{H} - t_0 \sigma_1 \bar{S} \bar{H} - t_0 \theta_3 H_0 H^2
\end{align*}
\]

Now we rescaled time with the rate of Vaccinated human population, which leads to the following dimensional parameters.

\[
t_0 = \frac{1}{\eta_1}, \lambda = \frac{\lambda_1}{\eta_1}, \rho = \frac{\rho_1}{\eta_1}, a = \frac{\theta_1}{\eta_1}, b = \frac{\theta_2}{\eta_1}, \mu = \frac{\mu_1}{\eta_1}, d = \frac{\phi_1 V_0}{\eta_1}, e = \frac{\beta_2}{\eta_1}, \sigma = \frac{\sigma_1}{\eta_1}
\]

\[
f = \frac{\tau_1 H_0 \eta_1}{\eta_1}, \tau = \frac{\tau_1}{\eta_1}, \omega = \frac{\omega_1}{\eta_1}, g = \frac{\alpha_1 H_0}{\eta_1 V_0}, \theta = \frac{\theta_3}{\eta_1}, \gamma = \frac{\gamma_1}{\eta_1}, \eta = \frac{\eta_2}{\eta_1}, \varphi = \frac{\phi_2 H_0}{\eta_1}
\]

\[
\beta = \frac{\beta_1}{\eta_1}
\]

Substituting (2.12) into (2.11) and dropping the hat, we have:

\[
\frac{dP}{dt} = \lambda (1 - P) + \rho R + \eta C - [a S + b A + d V + (1 - m)] P + f (P - 1) H + \sigma S P
\]

\[
\frac{dE}{dt} = (a S + b A + d V) P - (\omega + \lambda) E + f E H + \sigma E S
\]

\[
\frac{dS}{dt} = (1 - k) \omega E + \tau A - (\beta + \sigma + \lambda) S + f S H + \sigma S^2
\]

\[
\frac{dA}{dt} = k \omega E - (e + \tau + \lambda) A + f A H + \sigma A S
\]

\[
\frac{dC}{dt} = (1 - m) P + \theta R - (\eta + \lambda) C + f C H + \sigma C S
\]

\[
\frac{dR}{dt} = \beta S + e A - (\theta + \rho + \lambda) R + f R H + \sigma R S
\]

\[
\frac{d\bar{V}}{d\bar{t}} = g S H + h A H - \phi H V - \gamma V
\]

\[
\frac{d\bar{H}}{d\bar{t}} = (\lambda - \mu) H - \sigma S H - f H^2
\]

### 3 Model Analysis

#### 3.1 Determining the basic reproduction number, \( R_0 \)

Using the next generation matrix approach [18, 19, 20], we consider the following linearised system

\[
\frac{dW}{dt} = FW - MW,
\]
where

\[ F = \begin{bmatrix} 0 & aP_0 & bP_0 & dP_0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad M = \begin{bmatrix} h_1 & 0 & 0 & 0 \\ -h_6 & h_2 & -\tau & 0 \\ -h_7 & 0 & h_3 & o \\ 0 & -h_4 & -h_5 & h_8 \end{bmatrix}, \quad W = \begin{bmatrix} E \\ S \\ A \\ V \end{bmatrix}. \]

Here, \( FW \) represents the emergence of new infections, \( MW \) the transition of these infections among compartments and \( W \) the reservoir of infection, where

\[
\begin{align*}
\lambda > \mu, \quad k < 1, \quad m < 1, \quad &r_1 = \lambda - \mu, \quad r_2 = 1 - k, \quad r_3 = 1 - m \\
&h_1 = \omega + \mu, \quad h_2 = \beta + \sigma + \mu, \quad h_3 = e + \tau + \mu, \\
&h_4 = \frac{gr_1}{\tau}, \quad h_5 = \frac{h_3}{\tau}, \quad h_6 = r_2\omega, \quad h_7 = k\omega, \quad h_8 = \frac{gr_3}{\tau} + \gamma
\end{align*}
\]

The largest eigenvalue of \( G = FM^{-1} \) is the basic reproduction number.

\[
G = \frac{1}{g_0} \begin{bmatrix} g_0 & 0 & 0 & 0 \\ K_2 & g_6 & K_6 & 0 \\ K_3 & 0 & K_7 & 0 \\ K_4 & K_5 & K_8 & B_8 \end{bmatrix}
\]

Where

\[
\begin{align*}
g_0 &= h_1h_3h_4, \quad g_9 = h_2h_3h_6, \quad K_2 = h_3h_4h_8 + \tau h_7h_8, \quad K_3 = h_2h_7\tau h_8 \\
K_4 &= h_2h_4 + h_3h_4h_6 + \tau h_4h_8, \quad K_5 = h_1h_3h_8, \quad K_6 = \tau h_4h_8, \quad K_7 = h_4h_2h_8, \\
K_8 &= h_1(h_2h_5 + \tau h_3), \quad B_7 = h_1h_2, \quad B_8 = B_8h_8, \quad g_0 = B_8h_8.
\end{align*}
\]

The highest eigenvalue of \( G \) in terms of \( \sigma^\star \) gives:

\[
\sigma^\star = R_0 = \frac{\omega P_0(\tau_l(\omega + \mu) + \tau_l(\beta + \sigma + \mu))}{(\omega + \mu)(\omega + \tau_l)(\beta + \sigma + \mu)}
\]

where \( L_1 = r_2(\sigma + r_1(\omega + d\mu)) \) and \( L_2 = k(bf + r_1(b\theta + dh)) \)

### 3.2 Positivity, existence and uniqueness of solution

The model is described in the domain

\[
\Omega \in \mathbb{R}^8 = \{ P, E, S, A, R, C, V, H: P \geq 0, E \geq 0, S \geq 0, A \geq 0, R \geq 0, C \geq 0, V \geq 0, P + E + S + A + C + R = 1 \}
\]

Suppose \( \forall t \geq 0 \) all variables are non-negative, it implies that \( P(0) + E(0) + S(0) + A(0) + C(0) + R(0) = 1 \) and \( V(0) = 0 \). If \( E = 0 \), and all other variables are in \( \Omega \), then \( \frac{dE}{dt} \geq 0 \), this is also the case for variables in (2.15) - (2.19). If \( H = 0 \), then \( \frac{dh}{dt} = 0 \). But if \( H > 0 \) and assuming \( \lambda > \mu \), then with suitable initial conditions, \( \frac{dh}{dt} > 0 \forall t > 0 \). It follows that the right-hand side of (2.15) - (2.20) is continuous with continuous partial derivatives. Thus, solutions exist and are unique.

The model has mathematically and biologically relevant solutions in the domain \( \Omega \)

\( \forall t \in [0, \infty) \).
3.3 Steady state solution and stability analysis

The equilibrium point is \((P,E,S,A,R,V) = (P_0,0,0,0,C_0,0)\). At the disease free state, \(S = 0\) and \(A = 0\). Substituting these into the right hand side of (2.18), (2.19) gives \(R = 0\) and \(V = 0\). Further substitution of the values of \(S, A, R,\) and \(V\) into (2.14) gives \(E = 0\). Using \(S = A = R = E = V = 0\) in (2.17) and (2.12) gives \(C_0 = \frac{r_2}{\eta + \mu + r_2}\) and \(P_0 = \frac{\eta + \mu}{\eta + \mu + r_2}\) respectively. At the disease free state, all humans are entirely susceptible and we obtain from (2.20) the following logistic equation,

\[
\frac{dH}{dt} = r_1 H - fH^2
\]

With solution

\[
H(t) = \frac{KH_0}{H_0 + (K-H_0)e^{-r_1t}}
\]

Where \(r_1\) is as defined above and \(K = \frac{r_1}{f}\). As \(t \to \infty, H(t) \to K\), which is the carrying capacity of the environment.

Now, considering the Jacobian Matrix of the System at disease free state, \((P,E,L,S,A,R,V) = (P_0,0,0,0,C_0,0,0)\)

\[
J_{df} = \begin{bmatrix}
-h_9 & 0 & h_{10} & -bP_0 & \eta & \rho & -dP_0 \\
0 & -h_1 & aP_0 & bP_0 & 0 & 0 & dP_0 \\
0 & h_6 & -h_2 & \tau & 0 & 0 & 0 \\
0 & h_7 & 0 & -h_5 & 0 & 0 & 0 \\
r_3 & 0 & h_{11} & 0 & -h_{12} & 0 & 0 \\
0 & 0 & \beta & e & 0 & -h_{13} & 0 \\
0 & 0 & h_4 & h_5 & 0 & 0 & -h_9
\end{bmatrix}
\]

With the definitions \(h_i = 1, 2, 3, ... 8\) as defined above and

\[h_9 = r_3 + \mu, h_{10} = (\sigma - a)P_0, h_{11} = \sigma C_0, h_{12} = \eta + \mu, h_{13} = \theta + \rho + \mu\]

The characteristics polynomial equation of (3.8) with eigenvalue in terms of \(\lambda^*\) is given by:

\[
(\lambda^* + \theta + \rho + \mu)(\lambda^* + 2\mu)(\lambda^* + \eta + \mu + r_3)(\lambda^* + D_1\lambda^2 + D_2\lambda + D_3\lambda^* + D_4) = 0
\]

Clearly, equation (3.9) has three negative real roots. Thus we are left with the following quartic equation in term of the eigenvalue \(\lambda^*\)

\[
\lambda^* + D_1\lambda^* + D_2\lambda + D_3\lambda^* + D_4 = 0
\]

Where \(D_i\)'s are as defined in page (8).

**Lemma 3.1:** The disease free equilibrium is locally asymptotically stable if \(R_0 < 1\) and unstable if \(R_0 > 1\).

**Proof:**

We note that coefficients of (3.9) are all positive provided \(R_0 < 1\).

Applying the Ruth Hurwitz stability condition as stated in [7] and given in our case as satisfying the condition;

\[
\varphi = D_1D_2D_3 - (D_3^2 + D_2^2D_4) > 0
\]
We need to express $\varphi$ as a finite sum of positive terms involving the model parameters. The Maple result of the above algebra shows that $\varphi$ is indeed a sum of positive terms given by:
\[ \varphi = G_0(1 - R_0)^2 + G_1(1 - R_0) + G_0 + F, \]
where $F$ is the sum of the numerous positive terms involving the parameter space.

The maple file used to obtain the result is not included here but could be made available on request. The expressions of the constants are defined below.

\[ B_1 = h_2 h_3, \quad B_2 = h_2 h_6, \quad B_3 = h_4 h_6, \quad B_4 = h_4 h_7, \quad B_5 = h_5 h_3, \quad B_6 = h_1 + B_2 + B_3, \quad g_0 = B_9 h_8, \]
\[ g_1 = P_0 a h_4, \quad g_2 = P_0 a h_7, \quad g_3 = P_0 b h_2, \quad g_4 = P_0 d h_9, \quad g_5 = P_0 d h_9, \]
\[ g_6 = P_0 d h_2, \quad g_7 = h_1 h_3 h_5, \quad g_8 = h_1 h_3 h_5, \quad g_9 = h_2 h_3 h_6, \quad g_{10} = h_1 + h_2, \quad g_{11} = h_1 + h_3, \]
\[ g_{12} = h_2 + h_3, \quad g_{13} = h_3 + h_4, \quad C_1 = (g_7 + g_9 + B_9), \quad C_2 = (g_7 + g_9 + g_7 + g_6), \]
\[ C_3 = (g_7 + g_9 + g_7 + g_6), \quad g_{14} = g_{10} + B_9, \quad C_6 = B_4 + B_6, \quad C_7 = C_6 + \frac{C_4}{h_2}, \quad D_1 = g_{10} + g_{13}, \]
\[ D_2 = C_4(1 - R_0) + C_7, \quad D_3 = C_4(1 - R_0) + C_2 + C_3, \quad D_4 = g_6(g_1 - R_0), \]
\[ E_1 = 2g_7^2 + g_1 B_2 + g_2 B_3 + h_1(B_4 + g_7 + g_9)(B_1 + B_5 + B_7 + h_2 + h_1^2), \]
\[ E_2 = \frac{B_5 g_2 g_7}{g_5} + g_7(g_6 + g_7 + g_2 B_2 + B_3), \]
\[ E_3 = \frac{g_4}{h_3} + g_4 P_0 h_4 + 2B^2 h_3 P_0 d h_3, \]
\[ E_4 = \frac{g_5}{h_2} + g_5 B_2 + 2g_2 g_7 h_3 + 2g_4 h_3 + 3g_3 h_3 + 3B_2 h_3 + g_2 h_4, \]
\[ E_5 = B_7(g_6 + g_2 + g_3 + g_4) + g_6 h_4 P_0 + g_3 h_4 + \frac{g_2 g_7}{h_2}, \]
\[ E_6 = \frac{g_4}{d}(2B_2 + B_4) + \frac{2B_{3} B_2 d h_3}{g_5} + 2g_7 P_0 h_2 + g_7 h_2, \]
\[ E_7 = (g_1 + g_2 + g_3 + g_4 + g_6)(g_7 + g_9 + g_2 B_2 + B_3 + \frac{g_4}{h_3} + B_4 h_2), \]
\[ E_8 = (g_1 + g_2 + g_3 + g_4 + g_6)(g_2 g_5 + g_6 + \frac{2g_4}{h_3} + \frac{3g_3}{p_0 h_5} + \frac{4g_3}{p_0 h_5} + \frac{4g}{h_3}), \]
\[ E_9 = (g_1 + g_2 + g_3 + g_4 + g_6)(\frac{g_2 h_5}{h_3} + \frac{2g_5}{h_3}), \]
\[ E_{10} = (g_1 + g_2 + g_3 + g_4 + g_6)(\frac{2B_3 h_2 P_0 d h_3}{g_5 h_8} + \frac{g_4}{p_0 h_5} + \frac{2B_3 g_7 P_0 d}{g_5 h_3} + \frac{2h_2}{h_3}), \]
\[ E_{11} = (g_1 + g_2 + g_3 + g_4 + g_6)(\frac{g_2 b_2}{h_3} + \frac{b_2}{h_3}), \]
\[ E_{12} = (g_1 + g_2 + g_3 + g_4 + g_6)(\frac{g_3}{h_2 h_3 P_0} + \frac{g_4}{h_2 h_3 P_0}), \]
\[ E_{13} = (g_1 + g_2 + g_3 + g_4 + g_5)(\frac{g_3}{h_2 h_3 P_0} + \frac{g_4}{h_2 h_3 P_0}), \]
\[ E_{14} = (g_1 + g_2 + g_3 + g_4 + g_5 + g_6)(\frac{g_6}{h_3}), \]
\[ E_{15} = (g_1 + g_2 + g_3 + g_4 + g_5)(\frac{2g_6}{h_3} + \frac{2g_6}{h_3} + \frac{g_6}{h_2 h_3 P_0} + \frac{g_6}{h_2 h_3 P_0} + \frac{g_6}{h_2 h_3 P_0}). \]

Since $G_1 E_1$, $E_1$, $E_2$, $E_3$, $h_1$, $i = 1, 2, 3 > 0$, it follows that $\varphi > 0$, if $R_0 < 1$. Thus, the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$. If $R_0 > 1$, $D_1$ is positive and $D_4$ is negative, wherein the signs of $D_2$ and $D_3$ cannot be ascertained. However, we note that there is at least only one sign change depending on the signs of $D_2$ and $D_3$. Therefore the sequence of coefficients $D_1$, $D_2$, $D_3$, $D_4$ has only one sign change in the worst scenario. Thus, by the Descartes’ rule of sign, there exists at least one positive real eigenvalue, hence, we conclude that the disease free state is unstable if $R_0 > 1$. When $R_0 = 1$, (3.9) has one zero eigenvalue, which shows that $R_0 = 1$ is a bifurcation surface in $(\theta_1, \theta, \varphi_1, \omega_0, \gamma_2, \alpha_2, \beta_1, \beta_2, \lambda, \mu_1, \tau_1, k)$ parameter space.
3.4 Numerical solution

The numerical solution is obtained by using MATLAB’s ODE15s, variable order Runge-Kutta method with relative tolerance of $10^{-8}$ and absolute tolerance of $10^{-8}$. The dimensionless parameters used for the simulations are defined in (2.12) with numerical values: $\lambda = 0.0221, a = 2.551, b = 6.326, d = 0.0000628, \beta = 5.0357, e = 43.571, f = 0.01224, \mu = 0.00182, \eta = 4.081, r = 1.7857, g = 2.0306, h = 0.05102, \omega = 0.0244, \sigma = 2.1938, \rho = 4.286, \theta = 0.42857, \varphi = 0.0000628, \gamma = 0.5102$, with initial conditions $P = 0.98, E = 0.01, S = 0, A = 0, R = 0, C = 0.01, V = 0, H = 1$.

![Graphs showing the effect of vaccination on symptomatic, asymptomatic and recovered humans and Surface Virus](image)

Fig. 2. Results showing the effect of vaccination on symptomatic, asymptomatic and recovered humans and Surface Virus, where $t = 1$, represents approximately 5 days in real time. The initial conditions used are $P = 0.98, E = 0.01, S = 0, A = 0, C = 0.01, R = 0, V = 0, H = 1$ and the parameter values are given above.
Fig. 3. Result showing the effect of high contact rate between infectious and susceptible humans on the disease dynamics with $R_0 > 1$ and the values used for the simulations are the same as above with only $a = 10.551$, $b = 16.326$, $d = 0.628$ and $\omega = 0.244$.
3.5 Discussion

In this model, we describe the transmission of Covid-19 disease in an entirely susceptible human population due to the introduction of an index case and the effect of vaccination on the Disease dynamics. Using available data and with the introduction of vaccination, we obtain the Basic Reproduction Number, $R_0 = 0.3411$ different from the results of [4] and [9]. This value of $R_0$ suggests that the disease will likely die out due to vaccination as seen in Fig. 1a,b,c,d, where the symptomatic, asymptomatic, recovered human populations and viruses on environmental surfaces flatten out. This behavior is as a result of the impact vaccination in the system. This result agrees with the recommendation that was made in [4]. However, increasing the contact rates between infectious and susceptible humans will hinder the positive effect of vaccination. This agrees with the results of [21], which maintain that vaccination should be carried out in conjunction with other social measures that restrict contact rate between infectious and susceptible humans.

We note that the dimensionless parameters, $a$, $b$, $\omega$, and $d$ are key parameters in the Dynamics of the disease. An increase or decrease in these parameters will increase or decrease $R_0$ significantly, which implies, despite the introduction of vaccination, if the contributions of Symptomatic and asymptomatic humans, Surface virus and transition rate from latent period to infectiousness continue to increase without control, then there is every possibility that the disease will be endemic as shown in Fig. 2a,b,c, where the level of infection, and viruses on environmental surfaces pick and dropped to a steady state, implying an endemic situation. In Fig. 2f, the human population drops in a fast time scale due to the disease related death caused by high contact rate between infectious and susceptible humans in the midst of vaccination. Thus, in other to eradicate the disease, control measures like Social distancing, contact tracing, testing, quarantine, treatment, etc. are to be considered alongside vaccination.

4 Conclusion

In this work, we present a mathematical model on the dynamics of Covid-19 disease with vaccination. The model focuses on the effect of vaccination in the transmission dynamics of Covid-19 in a totally susceptible population due to the introduction of an index case. Analysis of the model shows that with the introduction of Vaccination the disease will likely die out. However, control measures like Social distancing, contact tracing, testing, quarantine disease case management should not be relaxed despite vaccination, as there exists the possibility of the disease becoming endemic with vaccination alone.

The model is a system of ordinary differential equations which may not capture some of the necessary features that drive the dynamics of the disease. The nature of the numerical solutions showing events happening in some fast timescale and others exhibiting long term behaviour suggests some other forms of analyses. The model does not account for the various variants of the disease and their possible effects on vaccination. Thus, further research work that will incorporate these features are highly recommended.

Competing Interests

Authors have declared that no competing interests exist.

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DOI: 10.3934/mbe.2020148


Available:https://doi.org/10.1016/j.chaos.2020.109846


DOI: 10.12785/amis/010104


Available:https://doi.org/10.1016/j.aej.2020.02.033


Available:https://doi.org/10.1063/5.0040301


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Peer-review history:
The peer review history for this paper can be accessed here (Please copy paste the total link in your browser address bar)
https://www.sdiarticle5.com/review-history/75356