STABILITY ANALYSIS OF A MATHEMATICAL MODEL FOR COVID-19 WITH WANING OF IMMUNITY AND REINFECTION

Abstract: In this paper a mathematical model that investigates reinfection and vaccination on the dynamics of COVID-19 was considered. The model particularly takes into account the waning rate of immunity after vaccination as well as administration of booster vaccine. Both disease free equilibrium and endemic equilibrium were determined as well as their stability analyzed using Routh Hurwitz stability criteria and Lyapunov stability. Numerical simulation was performed and we established that re-infection and waning rate of immunity contribute a lot in the disease staying in the population. In addition, results from numerical simulations show that booster vaccination increases the period of protection against the disease. Administration of booster vaccines is thus recommended for management of Corona Virus disease. The results show that reinfection, the waning rate of immunity after vaccination and the waning of immunity after infection contributes much on the diseases staying in the population. **Keywords:Vaccination, Reproduction number, COVID-19, Mathematical model, Stability analysis, Reproduction number** Mathematical model, Vaccination, Waning of immunity, Stability analysis, Reproduction number

1. Introduction

Corona Virus Disease of 2019 (COVID-19) is a novel corona virus that was first identified in December 2019 in China. It is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [5]. Covid-19 is likened to the severe acute respiratory syndrome(SARS) occurred in 2003 [7]. However covid-19 is more contagious than (SARS)of 2003 as within 3 months of outbreak there were more than 100,000 confirmed cases and more than 3000 death cases [7]. As much as the virus is a threat to everyone, symptoms vary from person to person. Most individuals experience fever, coughing and shortness of breath, while others may face savere symptoms such as damage to the lungs, acute respiratory failure [5] or others end up dying. Other symptoms include fatigue, muscle aches, headache, loss of taste, sore throat, nausea and a runny nose. The virus has since spread rapidly, resulting in approximately 779 million confirmed cases and 7 million deaths worldwide [20]. The recent data 28 days to June 28 2026 show that there are approximately 16,000 reported cases and 168 deaths[20]

Several interventions were put in place in order to reduce the spread of the disease. One potential solution that was proposed is the implementation of non-pharmaceutical measures which include; wearing face mask, public event bans, school and workplace closure, keeping social distance, public transport shutdowns, restrictions on internal movement, international travel controls and stay at home requirements [3, 7, 10]. Implementation of these measures have shown to be effective in containing the spread of other viruses, such as SARS-Cov [22]. And hence these measures can also be used to contain the spread of COVID-19 since they fall in the same group. However, implementation of these measures can negatively affect the economy and other health outcomes, including mental health and chronic conditions [1]. Vaccination is the common method that is relied on apart from the measures above. Vaccination not only provides protection for the individual it also provides it for the community at large since it keeps the effective reproduction rate below the level which would allow an epidemic to start, hence the so called 'herd immunity'[18]. As viruses are constantly changing, including the one that causes COVID-19, there is need for people to get vaccinated since the changes can lead to emergence of variants that can increase the risk of reinfection[6].

Many mathematical models for example in Ali M, George K and Kate F's research [2, 12, 15] have been used to describe the dynamics of COVID-19. The study done by Kate F [15] they first considered an SEIR model with no vaccine and then incorporated a vaccine compartment. The vaccine considered was imperfect meaning

individuals who are vaccinated can still contract the virus but at a reduced rate. The model considered in this paper not only incorpoarates vaccination but also administration of booster vaccination as well as waning of immunity after vaccination

2. MODEL EQUATION AND ANALYSIS

Note that the model to be analysed was formulated in [11] under the following assumptions

2.1. Assumptions

The population under study is divided into the following compartments S(t) which denote individuals who are susceptible to COVID-19 but not yet infected at time t, E(t) are exposed individuals who are infected but not yet infectious at time t, $I(t)$ are infective individuals, R(t) are vaccinated and recovered individuals, and V(t) are vaccinated individuals. For notational convenience we define the following variables;
$S(t) = S$, $E(t) = E$, $V(t) = V$, $I(t) = I$ and $R(t) = R$

We thus make the following assumptions for the model

(i) Individuals in each compartment are uniformly mixed.
(ii) Immunity induced by vaccination wanes and vaccines are imperfect, thus we introduce booster vaccination.
(iii) Infectivity rate of exposed individuals is less compared to infectivity of infected individuals.
(iv) Individuals in compartment V can be infected if they make contact with individuals in compartments E and I as the vaccines are imperfect.
(v) There is death due to COVID-19 in compartment I.
(v) Individuals in compartment R who were administered a booster vaccine are considered to have protective immunity at a longer period of time .
(vi) The total population size in consideration is a constant N.

The following parameters are used in the model

(i) β is the transmission rate of the disease, it describes the number of new cases that arise from each existing case.
(ii) λ_E is the infection rate of exposed individuals
(iii) η is the rate at which individuals are vaccinated while ρ is the efficacy of the vaccine
(iv) γ is the rate at which exposed members become infectious
(v) r_E and r_I are the rates at which exposed and infective members recover respectively, d is COVID-19 mortality rate, μ is the natural death rate in each compartment, b is rate of administering booster vaccine and ω is the rate at which individuals in R become susceptible to the disease again.

2.2. The Model

Using the above assumptions, parameters and basic concepts in infectious disease modeling the system of differential equations describing the model becomes

$$\begin{aligned}
\frac{dS}{dt} &= \phi - \beta S(\lambda_E E + I) - (\eta + \mu)S + \varepsilon V + \omega R, \\
\frac{dV}{dt} &= \eta S - (1-\rho)\beta V[\lambda_E E + I] - (b + \mu + \varepsilon)V, \\
\frac{dE}{dt} &= \beta S(\lambda_E E + I) + (1-\rho)\beta V[\lambda_E E + I)] - (\gamma + r_E + \mu)E, \\
\frac{dI}{dt} &= \gamma E - (r_I + d + \mu)I, \\
\frac{dR}{dt} &= r_E E + r_I I - (\omega + \mu)R + bV
\end{aligned} \tag{1}$$

Equation (1) is subject to the initial condition $S(0) \geq 0$, $V(0) \geq 0$, $E(0) \geq 0$, $I(0) \geq 0$, and $R(0) \geq 0$. Note that in [11] the properties which the model should satisfy to make it biologically reasonable were proved which include,

positivity and boundedness of solutions. In addition the disease free equilibrium as well as the basic reproduction number were also determined. Thus we will refer to them as soon as we need to make use of them.

2.3. Stability Analysis

2.3.1. Local Stability of the DFE

For the DFE we determine its stability using the theorem below

Theorem 2.1. The DFE is locally asymptotically stable if $R_e < 1$ but unstable if $R_e > 1$

We prove this theorem with respect to our system

Proof. Recall that the DFE is given by

$$(S_o, V_o, E_o, I_o, R_o) = \left(\frac{\phi(b+\mu+\varepsilon)}{(b+\mu)(\eta+\mu)+\mu}, \frac{\phi\eta}{(b+\mu)(\eta+\mu)+\mu\varepsilon}, 0, 0, 0 \right)$$

We will denote it by $\epsilon_o = (S_o, V_o, E_o, I_o, R_o)$. The Jacobian matrix of system (1) is given by

$$A = \begin{pmatrix} -\beta(\lambda_E E + I) - k_1 & \varepsilon & -\beta\lambda_E S & -\beta S & \omega \\ \eta & -\psi\beta(\lambda_E E + I) - c & -\psi\beta\lambda_E V & -\psi\beta V & 0 \\ \beta(\lambda_E E + I) & \psi\beta(\lambda_E E + I) & \beta\lambda_E(S+\psi V) - k & \beta(S+\psi V) & 0 \\ 0 & 0 & \gamma & -(r+d+\mu) & 0 \\ 0 & b & r & r & -(\omega+\mu) \end{pmatrix}$$

Where we have let $c = (b+\mu+\varepsilon)$, $k = (\gamma+r+\mu)$, $k_1 = \eta+\mu$
Evaluating A at the DFE and letting $k_2 = \beta\lambda_E S_o$, $k_3 = \beta S_o$, $k_4 = b+\mu+\varepsilon$, $k_5 = \psi\beta\lambda_E V_o$, $k_6 = \psi\beta V_o$, $k_7 = \beta\lambda_E(S+\psi V) - (\gamma+r+\mu)$, $k_8 = \beta(S_o+\psi V_o)$, $k_9 = r+d+\mu$, $k_{10} = \omega+\mu$ we obtain;

$$A(\epsilon_o) = \begin{pmatrix} -k_1 & \varepsilon & -k_2 & -k_3 & \omega \\ \eta & -k_4 & -k_5 & -k_6 & 0 \\ 0 & 0 & k_7 & k_8 & 0 \\ 0 & 0 & \gamma & -k_9 & 0 \\ 0 & b & r & r & -k_{10} \end{pmatrix}$$

We determine the stability of ϵ_o by finding the eigenvalues of the matrix $A(\epsilon_o)$. That is, we solve

$$\begin{vmatrix} \lambda+k_1 & \varepsilon & -k_2 & -k_3 & \omega \\ \eta & \lambda+k_4 & -k_5 & -k_6 & 0 \\ 0 & 0 & \lambda-k_7 & k_8 & 0 \\ 0 & 0 & \gamma & \lambda+k_9 & 0 \\ 0 & b & r & r & \lambda+k_{10} \end{vmatrix} = 0$$

$$(\lambda+k_1)\begin{vmatrix} \lambda+k_4 & -k_5 & -k_6 & 0 \\ 0 & \lambda-k_7 & k_8 & 0 \\ 0 & \gamma & \lambda+k_9 & 0 \\ b & r & r & \lambda+k_{10} \end{vmatrix} - \eta\begin{vmatrix} \varepsilon & -k_2 & -k_3 & \omega \\ 0 & \lambda-k_7 & k_8 & 0 \\ 0 & \gamma & \lambda+k_9 & 0 \\ b & r & r & \lambda+k_{10} \end{vmatrix} = 0$$

$$(\lambda+k_1)(\lambda+k_{10})\begin{vmatrix} \lambda+k_4 & -k_5 & -k_6 \\ 0 & \lambda-k_7 & k_8 \\ 0 & \gamma & \lambda+k_9 \end{vmatrix} - \eta(\varepsilon(\lambda+k_{10}) - b\omega)\begin{vmatrix} \lambda-k_7 & k_8 \\ \gamma & \lambda+k_9 \end{vmatrix} = 0$$

$$(\lambda+k_1)(\lambda+k_{10})(\lambda+k_4)\begin{vmatrix} \lambda-k_7 & k_8 \\ \gamma & \lambda+k_9 \end{vmatrix} - \eta(\varepsilon(\lambda+k_{10}) - b\omega)\begin{vmatrix} \lambda-k_7 & k_8 \\ \gamma & \lambda+k_9 \end{vmatrix} = 0$$

$$\lambda-k_7)(\lambda+k_9) - \gamma k_8\left[(\lambda+k_1)(\lambda+k_{10})(\lambda+k_4) - \eta(\varepsilon(\lambda+k_{10}) - b\omega)\right] = 0$$

$\implies$

$$(\lambda - k_7)(\lambda + k_9) - \gamma k_8 = 0 \quad (2)$$
$$(\lambda + k_1)(\lambda + k_{10})(\lambda + k_4) - \eta(\varepsilon(\lambda + k_{10}) - b\omega) = 0 \quad (3)$$

From equations (2) and (3) we respectively obtain

$$\lambda^2 + (k_9 - k_7)\lambda - (k_7k_9 + k_8\gamma) = 0$$
$$\lambda^3 + (k_1 + k_4 + k_{10})\lambda^2 + (k_1k_4 + k_1k_{10} + k_4k_{10} - \eta\varepsilon)\lambda + k_1k_4k_{10} + \eta b\omega - \eta\varepsilon k_{10} = 0$$

We check the signs of the roots of the two latter equations using the Routh-Hurwitz stability criterion which we refer in [18**?**].
For

$$\lambda^2 + (k_9 - k_7)\lambda - (k_7k_9 + k_8\gamma) = 0 \quad (4)$$

the coefficients in the polynomial will make the first column of the Routh-Hurrwitz array and we can easily see that they are all non-zero. That is the first column will take the elements 1,$(k_9 - k_7)$ and $-(k_7k_9 + k_8\gamma)$. All we need to check are the signs of these elements.
Obviously $1 > 0$. We check $(k_9 - k_7)$. Let's assume $(k_9 - k_7) < 0 \implies k_9 < k_7$ Recalling k_7 and k_9 we have

$$r + d + \mu < \beta\lambda_E(S_o + \psi V_o) - (\gamma + r + \mu)$$

Which can be written as

$$\frac{r + d + \mu}{\gamma + r + \mu} < \frac{\beta\lambda_E S_o}{\gamma + r + \mu} + \frac{\beta\lambda_E\psi V_o}{\gamma + r + \mu} - 1$$

Which we can easily express the right hand side(RHS) in terms of R_e by adding

$$\frac{\gamma\beta S_o}{(\gamma + r + \mu)(r + \mu + d)} + \frac{\gamma\beta\psi V_o}{(\gamma + r + \mu)(r + \mu + d)}$$

of which the inequality still holds and we obtain $\frac{r+d+\mu}{\gamma+r+\mu} < R_e - 1$. The left hand side(LHS) is division of positive numbers which the quotient will be positive, thus for the inequality to hold $R_e > 1$ which implies that $k_9 - k_7 < 0$ if $R_e > 1$. This further implies $k_9 - k_7 > 0$ if $R_e < 1$. For $-(k_7k_9 + k_8)$ let's assume $-(k_7k_9 + k_8) > 0 \implies k_7k_9 + k_8 < 0$
Which also implies $k_7k_9 < -k_8$. Recalling k_7, k_9 and k_8 we have

$$[\beta\lambda_E(S + \psi V) - (\gamma + r + \mu)](r + d + \mu) < -\beta(S_o + \psi V_o)$$

$\implies$

$$\beta\lambda_E(S + \psi V) - (\gamma + r + \mu) < -\frac{\beta S_o}{(r + d + \mu)} - \frac{\psi V_o}{(r + d + \mu)}$$

Dividing through by $\gamma + r + \mu$ and rearranging terms we obtain

$$R_e - 1 < 0$$

$\implies -(k_7k_9 + k_8) > 0$ if $R_e < 1$ and $-(k_7k_9 + k_8) < 0$ if $R_e > 1$. We thus see that for $R_e < 1$ the elements are all positive but for $R_e > 1$ there is change in signs. What this means is that for $R_e < 1$ all the two eigenvalues will be negative while for $R_e > 1$ at least one of the eigenvalues will be positive. We now check for

$$\lambda^3 + (k_1 + k_4 + k_{10})\lambda^2 + (k_1k_4 + k_1k_{10} + k_4k_{10} - \eta\varepsilon)\lambda + k_1k_4k_{10} + \eta b\omega - \eta\varepsilon k_{10} = 0$$

The Routh-Hurrwitz array of this polynomial is

$$\begin{array}{c|cc} \lambda^3 & 1 & k_1k_4 + k_1k_{10} + k_4k_{10} - \eta\varepsilon \\ \lambda^2 & k_1 + k_4 + k_{10} & k_1k_4k_{10} + \eta b\omega - \eta\varepsilon k_{10} \\ \lambda^1 & \text{K} & \\ \lambda^0 & k_1k_4k_{10} + \eta b\omega - \eta\varepsilon k_{10} & \end{array}$$

Where

$$K = \frac{k_1k_4 + k_1k_{10} + k_4k_{10} - \eta\varepsilon - (k_1 + k_4 + k_{10})(\eta b\omega - \eta\varepsilon k_{10})}{k_1 + k_4 + k_{10}}$$

We see that all the elements in the first column are non-zero. All we need to do is check the sign of each element, 1 is trivial. We go to $k_1 + k_4 + k_{10}$ which is easily seen as $k_1 + k_4 + k_{10} > 0$ because k_1, k_2 and k_{10} are all positive. For K let's assume it's also positive that's

$$\frac{k_1k_4 + k_1k_{10} + k_4k_{10} - \eta\varepsilon - (k_1 + k_4 + k_{10})(\eta b\omega - \eta\varepsilon k_{10})}{k_1 + k_4 + k_{10}} > 0$$

Implying that

$$(k_1 + k_4 + k_{10})(k_1k_4 + k_1k_{10} + k_4k_{10} - \eta\varepsilon) - (\eta b\omega - \eta\varepsilon k_{10}) > 0$$

We observe that $\eta\varepsilon$ term is contained in (k_1k_2) with a positive sign hence they will cancel out. Also $\eta b\omega$ term is contained in $(k_1k_4k_{10})$ with a positive sign and they will cancel out. Thus we remain with positive constants which implies our assumption is correct. That is

$$\frac{k_1k_4 + k_1k_{10} + k_4k_{10} - \eta\varepsilon - (k_1 + k_4 + k_{10})(\eta b\omega - \eta\varepsilon k_{10})}{k_1 + k_4 + k_{10}} > 0$$

$\implies K > 0$. Finally we check $k_1k_4k_{10} + \eta b\omega - \eta\varepsilon k_{10}$. Since $\eta\varepsilon k_{10}$ is contained in $(k_1k_4k_{10})$ as positive they will cancel out thus obtaining $k_1k_4k_{10} + \eta b\omega - \eta\varepsilon k_{10} > 0$. All these results imply that the first column of the Routh-Hurrwitz array contains elements with same sign. Thus all the roots of the polynomial are negative. We see that for $R_e < 1$ all the eigenvalues are negative, thus the DFE is locally asymptotically stable.However, for $R_e > 1$ at least one eigenvalue is positive hence the DFE is unstable to small perturbations.The local stability of endemic equilibrium can also be determined in a similar manner.

2.3.2. Global Stability of DFE

We consider equation (5) below and state the theorem that will help us determine the global stability of the DFE of the general system and thus our system. This can be referred in [16]

$$\begin{aligned} \dot{x_i} &= -Tx - f(x, y) \\ \dot{y_j} &= g_j(x, y) \end{aligned} \tag{5}$$

Theorem 2.2. **(Castillo-Chavez, Feng and Huang)** If -T is a nonsingular M-matrix, $f(x, y) \geq 0$, $F \geq 0$ and if $R_o < 1$ then the DFE is globally asymptotically stable.

The proof of this theorem can be found in [4]. All we need to check is if the conditions in Theorem 2.2 above are satisfied so that we make use of the theorem to determine the global stability of the DFE of our system. Recall that for our system in (1)

$F = \begin{pmatrix} \beta\lambda_E(S_o + \psi V_o) & \beta(S_o + \psi V_o) \\ 0 & 0 \end{pmatrix}$

V=$\begin{pmatrix} c_1 & 0 \\ -\gamma & c_2 \end{pmatrix}$

Where $c_1 = \gamma + \mu + r$, $c_2 = r + \mu + d$

$-T = -(F - V) = -\begin{pmatrix} \beta\lambda_E(S_o + \psi V_o) - c_1 & \beta(S_o + \psi V_o) \\ \gamma & -c_2 \end{pmatrix}$

$\implies -T = \begin{pmatrix} c_1 - \beta\lambda_E(S_o + \psi V_o) & -\beta(S_o + \psi V_o) \\ -\gamma & c_2 \end{pmatrix}$

Since the off-diagonal elements of -T are negative it implies -T is a nonsingular M-matrix. It is also clear that $F \geq 0$ and all that remains to be checked is if $f(x, y) \geq 0$. Recall that $f(x, y) = (F - V)x - F_i + V_i$. Since x

represents the disease compartments, $x = \begin{pmatrix} E \\ I \end{pmatrix}$ Thus

$$\begin{aligned}(F-V)x &= \begin{pmatrix} \beta\lambda_E(S_o+\psi V_o)-c_1 & \beta(S_o+\psi V_o) \\ \gamma & -c_2 \end{pmatrix}\begin{pmatrix} E \\ I \end{pmatrix} \\ &= \begin{pmatrix} E\beta\lambda_E(S_o+\psi V_o)-c_1E+\beta I(S_o+\psi V_o) & \gamma E-c_2I \end{pmatrix} \end{aligned} \tag{6}$$

Also from the definitions of F_i and V_i we have

$$-(F_i-V_i) = -\begin{pmatrix} \beta S(\lambda_E E+I)+\psi\beta V(\lambda_E E+I)-c_1E & -c_2I+\gamma I \end{pmatrix} \tag{7}$$

From equations (6) and (7) we have

$$\begin{aligned} f(x,y) &= \begin{pmatrix} \beta\lambda_E E(S_o+\psi V_o)+\beta I(S_o+\psi V_o)-\beta S(\lambda_E E+I)-\psi\beta V(\lambda_E E+I) & 0 \end{pmatrix} \\ &= \begin{pmatrix} (\beta I+\beta\lambda_E E)(S_o-S)+(\beta\psi\lambda_E E+\beta\psi I)(V_o-V) & 0 \end{pmatrix} \end{aligned}$$

Clearly $f(x,y) \geq 0$ since $S_o \geq S$ and $V_o \geq V$. Since all the conditions of Theorem 2.2 are satisfied, it implies that the DFE of our system is globally asymptotically stable. This implies that even if the disease is present in the population and $R_e < 1$ then the disease will be wiped out of the population since all the solutions will approach the DFE.

2.4. Stability of the Endemic Equilibrium

The endemic equilibrium(EE) means that the disease persists in the population. Thus it makes sense to know if the disease will stay in the population forever or it dies out. We start by determining the EE and then it's stability. We will assume that after vaccination and booster vaccination program are imposed, individuals stay for a long period of time in the recovery before being susceptible to the disease again thus we take $\omega << 1$ for the determination and further stability analysis of the EE. In fact from the expression of R_e, there is no dependence of R_e on the parameter ω. Therefore recalling system (1) and noting that R does not appear in the first four equations, we equate each of the differential equation to 0 and thus obtain a reduced system

$$\begin{aligned} 0 &= \phi-\beta S^*(\lambda_E E^*+I^*)-(\eta+\mu)S^*+\varepsilon V^* \\ 0 &= \eta S^*-\psi\beta V^*(\lambda_E E^*+I^*)-(b+\mu+\varepsilon)V^* \\ 0 &= \beta S^*(\lambda_E E^*+I^*)+\psi\beta V^*(\lambda_E E^*+I^*)-(\gamma+r+\mu)E^* \\ 0 &= \gamma E^*-(r+d+\mu)I^* \end{aligned} \tag{8}$$

Where (S^*, V^*, E^*, I^*) represent the endemic equilibrium values
From the last equation in (8) we obtain

$$I^* = \left(\frac{\gamma}{r+d+\mu}\right)E^* = k_1E^* \tag{9}$$

Using (9) in the other three equations of (8) and letting $k_2 = \frac{\lambda_E(r+d+\mu)+\gamma}{r+d+\mu}$
we obtain

$$\begin{aligned} 0 &= \phi-\beta k_2S^*E^*-(\eta+\mu)S^*+\varepsilon V^* \\ 0 &= \eta S^*-\psi\beta k_2V^*E^*-(b+\mu+\varepsilon)V^* \\ 0 &= \beta k_2S^*E^*+\psi\beta k_2V^*E^*-(\gamma+r+\mu)E^* \end{aligned} \tag{10}$$

The last equation of (10) gives

$$\left(\beta k_2S^*+\psi\beta k_2V^*-(\gamma+r+\mu)\right)E^* = 0$$

$E^* = 0$ will give us the DFE, we thus take

$$\beta k_2 S^* + \psi\beta k_2 V^* - (\gamma + r + \mu) = 0$$

$\implies S^* = k_3 - \psi V$ where

$$k_3 = \frac{\gamma + r + \mu}{\beta k_2} = \frac{(\gamma + r + \mu)(r + d + \mu)}{\beta\lambda_E(r + d + \mu) + \beta\gamma}$$

Adding the the last two equations of (10) to the first equation and using $S^* = k_3 - \psi V$ we obtain

$$\phi - (b + \mu)V^* - (\gamma + r + \mu)E^* - \mu(k_3 - \psi V^*) = 0 \tag{11}$$

From equation (11) we get

$$\begin{aligned} V^* &= \frac{\phi(1 - k_3)}{\mu(1 - \psi) + b} - \left(\frac{\gamma + r + \mu}{\mu(1 - \psi) + b}\right) E^* \\ V^* &= k_4 - k_5 E^* \end{aligned} \tag{12}$$

Thus

$$S^* = k_3 - \psi k_4 + \psi k_5 E^*$$

That is

$$S^* = k_6 + \psi k_5 E^* \tag{13}$$

We now use equations (12) and (13) into the second equation of (10) to get

$$\psi\beta k_2 k_5 {E^*}^2 + (\eta\psi k_5 + (b + \mu + \varepsilon)k_5 - \psi\beta k_2 k_4)E^* - ((b + \mu + \varepsilon)k_4 - \eta k_6) = 0$$

Which can be written as

$$a_1 {E^*}^2 + a_2 E^* - a_3 = 0 \tag{14}$$

Where $a_1 = \psi\beta k_2 k_5$, $a_2 = (\eta\psi k_5 + (b + \mu + \varepsilon)k_5 - \psi\beta k_2 k_4)$ and $a_3 = ((b + \mu + \varepsilon)k_4 - \eta k_6)$
We note that $a_1 = \psi\beta k_2 k_5 > 0$ since $k_2 = \frac{\lambda_E(r+d+\mu)+\gamma}{r+d+\mu} > 0$ and
$k_5 = \left(\frac{\gamma+r+\mu}{\mu(1-\psi)+b}\right) > 0$.
Also using $R_o = \frac{\beta\lambda_E}{(\gamma+r+\mu)} + \frac{\gamma\beta}{(\gamma+r+\mu)(r+\mu+d)}$ we have $k_3 = \frac{(\gamma+r+\mu)(r+d+\mu)}{\beta\lambda_E(r+d+\mu)+\beta\gamma} = \frac{1}{R_o}$. We thus obtain

$$((b + \mu + \varepsilon)k_4 - \eta k_6) = \frac{1}{R_o}\left[\frac{\phi(\eta\psi + b + \mu + \varepsilon)}{\mu(1 - \psi) + b}(R_o - 1) - \eta\right] > 0$$

if $R_o > 1$. Thus $a_3 > 0$ if $R_o > 1$. With the fact that $a_1 > 0$ and $a_3 > 0$ for $R_o > 1$, equation (14) will have two distinct roots, one positive and the other negative, that is

$$E^*_{\pm} = \frac{-a_2 \pm \sqrt{a_2^2 + 4a_1 a_3}}{2a_1} \tag{15}$$

For the endemic equilibrium to be biologically relevant we take

$$E^*_{+} = \frac{-a_2 + \sqrt{a_2^2 + 4a_1 a_3}}{2a_1} \tag{16}$$

We use equation (16) in equations (8), (12) and (13) to obtain the expressions I^*, V^* and S^* respectively. That is

$$\begin{aligned} I^* &= k_1 E_+^* \\ V^* &= k_4 - k_5 E_+^* \\ S^* &= k_6 + \psi k_5 E_+^* \end{aligned} \tag{17}$$

Equation (16) and system (17) give us the endemic equilibrium
We are done with finding the endemic equilibrium and thus we proceed to determine it's stability. We are going to make use of Lyapunov function to help us determine the stability. Thus it will make sense if we proceed as follows;

Consider a system of differential equations given by

$$\dot{x_i} = f_i(x_1, x_2, ..., x_n) \tag{18}$$

Where $\dot{} = \frac{d}{dt}$ and $x_i = x_i(t)$. Let x_i^* be an equilibrium point of equation (18). A function L is said to be a Lyponov function of equation (18) if it satisfies the following

(i) $L(x_i^*) = 0$

(ii) $L(x_i) > 0$ for $x_i \neq x_i^*$

(iii) $\frac{dL}{dt} \leq 0$ over the solutions of equation (18)

As in [14, 19] we state the following principle which together with a suitable Lypunov function help in determination of the stability of the EE.

Lasalle Invariance Principle

Assume that L(x) is a Lyapunov function of equation (18) on a subset $G \subset R^n$, $n \geq 1$. Define a set $S = \{x \in G : \dot{L}(x) = 0\}$. Let M be the largest invariant set contained in S. Then for $t \geq 0$, every bounded trajectory of equation (18) that remains in G approaches the set M as $t \to \infty$

We need to make use of this principle to show the stability of the endemic equilibrium. Following [**?**] we let

$$\begin{aligned} L_1 &= S - S^* - S^* ln\frac{S}{S^*} \\ L_2 &= V - V^* - V^* ln\frac{V}{V^*} \\ L_3 &= E - E^* - E^* ln\frac{E}{E^*} \\ L_4 &= I - I^* - I^* ln\frac{I}{I^*} \end{aligned}$$

To use the principle above, we have to show that L given by $L = L_1 + L_2 + L_3 + L_4$ is a Lyapunov function for the system in equation (1)

Let $P^* = (S^*, V^*, E^*, I^*)$ denote the endemic equilibrium. Clearly $L(P^*) = 0$. To show the remaining conditions for a Lypunov function we use the inequality $1 - x + lnx \leq 0$ for $x > 0$ and the equality only holds if $x = 1$.

We can write L_1 as

$$\begin{aligned} L_1 &= S^* \left(\frac{S}{S^*} - 1 - ln\frac{S}{S^*} \right) \\ &= -S^* \left(1 - \frac{S}{S^*} + ln\frac{S}{S^*} \right) > 0 \end{aligned}$$

$$since\ 1 - \frac{S}{S^*} + ln\frac{S}{S^*} < 0$$

Similarly $L_2 > 0$, $L_3 > 0$, $L_4 > 0$ for $P^* \neq (S, V, E, I)$. Thus $L > 0$ for
$P^* \neq (S, V, E, I)$. We now determine the derivative of the Lyapunov function over the solutions of equation (1).

Differentiating each of $L_i,\ \ i = 1, 2, 3, 4$ and using equation (1) we have

$$\begin{aligned}
\dot{L}_1 &= \left(1 - \frac{S^*}{S}\right)\Big[\phi - \beta S(\lambda_E E + I) - (\eta + \mu)S + \varepsilon V + \omega R\Big] \\
\dot{L}_2 &= \left(1 - \frac{V^*}{V}\right)\Big[\eta S - \psi\beta V(\lambda_E E + I) - (b + \mu + \varepsilon)V\Big] \\
\dot{L}_3 &= \left(1 - \frac{E^*}{E}\right)\Big[\beta S\lambda_E E + \psi\beta V(\lambda_E E) - (r + \mu)E\Big] \\
\dot{L}_4 &= \left(1 - \frac{I^*}{I}\right)\Big[\beta SI + \beta VI - (r + d + \mu)I\Big]
\end{aligned}$$

Using $\phi = \beta S^*(\lambda_E E^* + I^*) + (\eta + \mu)S^* - \varepsilon V^*$, $\dot{L}_1$ becomes

$$\begin{aligned}
\dot{L}_1 &= \left(1 - \frac{S^*}{S}\right)\Big[\beta S^*(\lambda_E E^* + I^*) + (\eta + \mu)S^* - \varepsilon V^* - \beta S(\lambda_E E + I) - (\eta + \mu)S + \varepsilon V\Big] \\
&= -\mu\frac{(S - S^*)^2}{S} + \left(1 - \frac{S^*}{S}\right)\Big[\beta S^*(\lambda_E E^* + I^*) - \varepsilon V^* + \eta S^* - \eta S - \beta S(\lambda_E E + I) + \varepsilon V\Big] \\
&\leq \beta\lambda_E S^* E^*\left(1 - \frac{S^*}{S}\right)\left(1 - \frac{SE}{S^*E^*}\right) + \beta S^* I^*\left(1 - \frac{S^*}{S}\right)\left(1 - \frac{SI}{S^*I^*}\right) \\
&+ \varepsilon V^*\left(1 - \frac{S^*}{S}\right)\left(\frac{V}{V^*} - 1\right) + \eta S^*\left(1 - \frac{S^*}{S}\right)\left(1 - \frac{S}{S^*}\right) \\
&\leq \beta\lambda_E S^* E^*\left(1 - \frac{SE}{S^*E^*} - \frac{S^*}{S} + \frac{E}{E^*}\right) + \beta S^* I^*\left(1 - \frac{SI}{S^*I^*} - \frac{S^*}{S} + \frac{I}{I^*}\right) \\
&+ \varepsilon V^*\left(\frac{V}{V^*} - 1 - \frac{VS^*}{SV^*} + \frac{S^*}{S}\right) + \eta S^*\left(2 - \frac{S^*}{S} - \frac{S}{S^*}\right)
\end{aligned}$$

Let's assume that individuals who are vaccinated are equal to the individuals whose immunity wanes and become susceptible again. Then expressing each of the terms in brackets in terms of the inequality $1 - x + lnx \leq 0$ for any $x > 0$ then using the inequality we obtain

$$\begin{aligned}
\dot{L}_1 &\leq \beta\lambda_E S^* E^*\left(\frac{E}{E^*} - ln\frac{E}{E^*} + ln\frac{SE}{S^*E^*} - \frac{SE}{S^*E^*}\right) + \beta S^* I^*\left(\frac{I}{I^*} - ln\frac{I}{I^*} + ln\frac{SI}{S^*I^*} - \frac{SI}{S^*I^*}\right) \\
&+ \eta S^*\left(ln\frac{S}{S^*} - \frac{S}{S^*} + \frac{V}{V^*} - ln\frac{V}{V^*}\right)
\end{aligned}$$

Similarly using the inequality $1 - x + lnx \leq 0$ $\dot{L}_2$, becomes

$$\begin{aligned}
\dot{L}_2 &\leq \psi\beta\lambda_E V^* E^*\left(\frac{E}{E^*} - ln\frac{E}{E^*} + ln\frac{VE}{V^*E^*} - \frac{VE}{V^*E^*}\right) \\
&+ \psi\beta V^* I^*\left(\frac{I}{I^*} - ln\frac{I}{I^*} + ln\frac{VI}{V^*I^*} - \frac{VI}{V^*I^*}\right) \\
&+ \eta S^*\left(\frac{S}{S^*} - ln\frac{S}{S^*} + \frac{V}{V^*} - ln\frac{V}{V^*}\right)
\end{aligned}$$

For $\dot{L}_3$ we have

$$\begin{aligned}
\dot{L}_3 &\leq \beta\lambda_E S^* E^*\left(ln\frac{E}{E^*} - \frac{E}{E^*} + \frac{SE}{S^*E^*} - ln\frac{SE}{S^*E^*}\right) \\
&+ \psi\beta\lambda_E V^* E^*\left(ln\frac{E}{E^*} - \frac{E}{E^*} + \frac{VE}{V^*E^*} - ln\frac{VE}{V^*E^*}\right)
\end{aligned}$$

Finally $\dot{L}_4$ satisfies

$$\begin{aligned}\dot{L}_4 &\leq \beta S^* I^* \left(ln\frac{I}{I^*} - \frac{I}{I^*} + \frac{SI}{S^*I^*} + ln\frac{SI}{S^*I^*} \right) \\ &+ \psi\beta V^* I^* \left(ln\frac{I}{I^*} - \frac{I}{I^*} + \frac{VI}{V^*I^*} - ln\frac{VI}{V^*I^*} \right)\end{aligned}$$

Recalling the term $-\mu\frac{(S-S^*)^2}{S}$ in $\dot{L}_1$ and using the inequalities we have obtained for $\dot{L}_1, \dot{L}_2, \dot{L}_3 \; and \; \dot{L}_4$ we have

$$\frac{dL}{dt} \leq -\mu\frac{(S-S^*)^2}{S} \leq 0 \tag{19}$$

Clearly from equation (19) we see that the equality only holds at the EE. Therefore by the Lasalle's Invariance Principle above, the EE is the largest invariance subset of the system in equation (1). Thus all trajectories approach the EE, hence it is globally asymptotically stable. Biologically this means that as long as $R_e > 1$ then it is difficult to wipe the disease completely out of the population since all solutions will approach the endemic equilibrium which describes the presence of the disease in the population

3. Numerical Simulation

The parameter values in Table 1 below were used in the simulation

Table 1. *Parameter Values*

Parameter	Value	Source
ϕ	0.00005	[8]
β	0.5	[12]
λ_E	0.314	[12]
η	0.0005	Assumed
μ	0.00005	[21]
ω	0.0033	[9]
ε	0.0042	[17]
ψ	0.2	Assumed
b	0.0001	Assumed
γ	0.1667	[13]
r	0.1	[5]
d	0.0016	[12]

Figures 1 and 2 describe the dynamics of COVID-19 when there is no vaccine but 2 only describes how the disease compartments evolve with time. Similarly, Figures 3 and 4 describe the dynamics of the disease but only when the vaccine compartment is included.

We can see that from Figures 1 and 2 the disease spreads into the the population with the infective individuals increasing up to a peak of 0.1857 after about 65 days and the spread reduces to almost being depleted and then start increasing then decreasing to a level which the disease stays in the population. The increase up to the peak is due to the rapid spread of the disease caused by a high transmission rate($\beta = 0.5$) and $R_e > 1$ which causes a single infected individual to produce more than one secondary infections. The reduction is due to most individuals gaining protective immunity from the infection. The immunity is temporary and thus the increase and the disease staying in the population is mostly caused by the re-infection of individuals, the rate of administering booster vaccination is low and the rate at which individuals lose vaccine induced immunity is high (about 6 months), which contributes much on individuals becoming susceptible again even after vaccination.

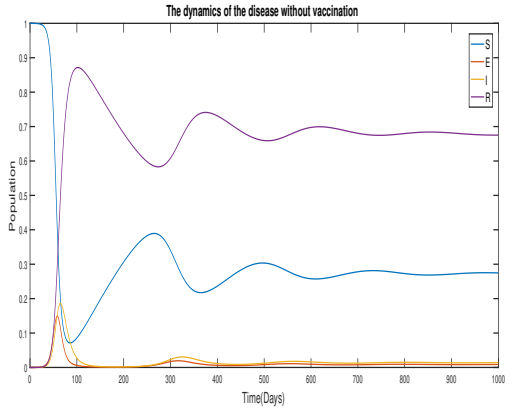

Figure 1. *Before vaccination*

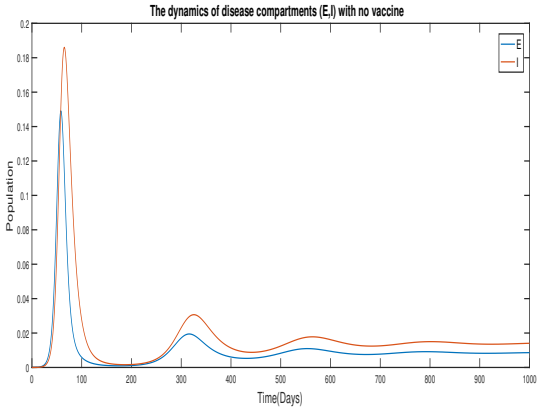

Figure 2. *E and I*

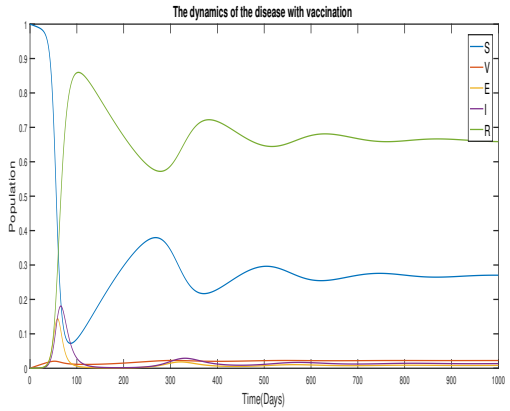

Figure 3. *After Vaccination*

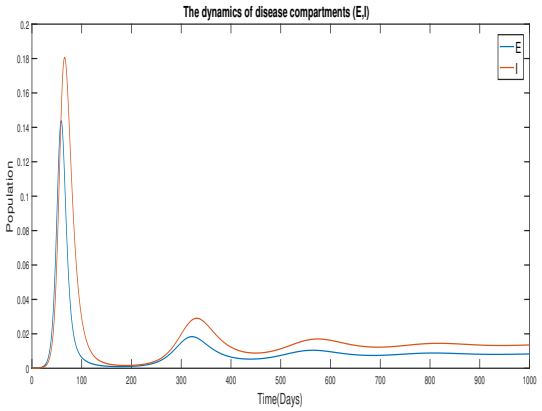

Figure 4. *E and I*

From Figure 4, we see that the peak is reduced to 0.18 after about 65 days. The curves look similar to the one in Figure 2 because of low vaccination coverage($\eta = 0.005$). Moreover, the rate of administering booster vaccination is low and the rate at which individuals lose vaccine induced immunity is high (about 6 months), which contributes much on individuals becoming susceptible again even after vaccination. Thus there is need to increase vaccination coverage and decrease the waning rate of immunity after vaccination.

In fact if the rate of vaccination is at the critical value $\eta_c = 0.0051$ we obtain

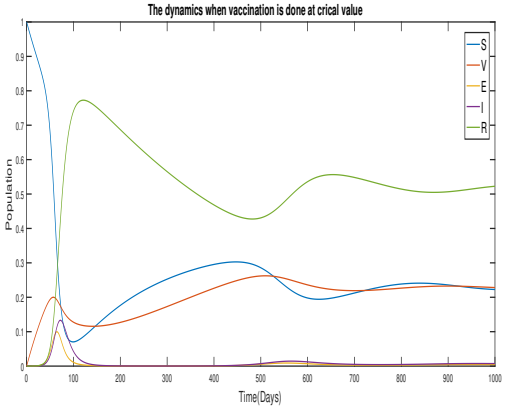

Figure 5. *Vaccination at η_c*

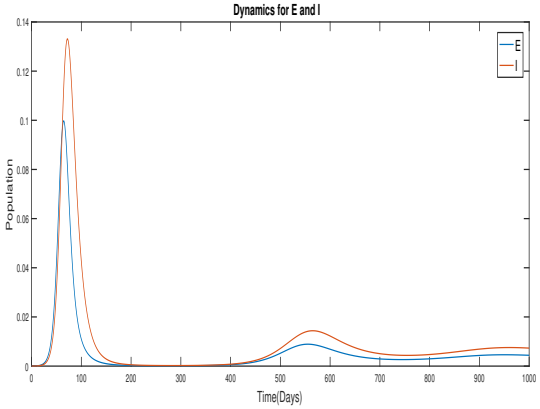

Figure 6. *E and I*

We observe that the number of individuals who stay in the population as vaccinated is much higher in Figure 5 than in Figure 3. Also comparing Figure 6 and Figure 3 we clearly see that the peak of infection is low (0.13) and delayed for about 15 days in Figure 6. In addition the number of infectives who stay in the population is low in Figure 6 since a lot of people are vaccinated and gain protective immunity for some time before the immunity start waning. We can see that even if the rate of vaccination is done at critical value $\eta_c = 0.0051$ the disease will not be completely eradicated from the population but the severity of the disease is reduced since we will have a small number of infected individuals staying in the population.

If there is no re-infection of individuals then the solution curves for E and I are as shown below.

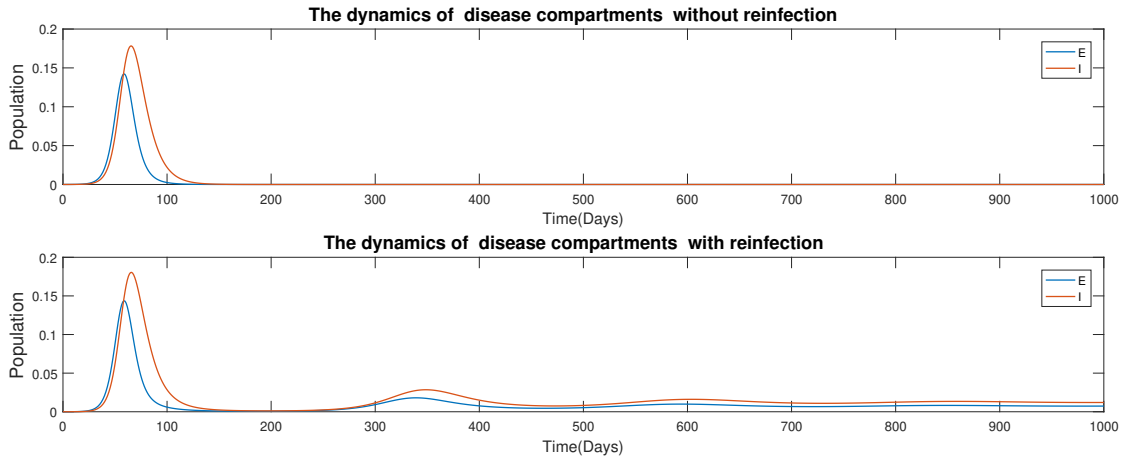

Figure 7. *Evolution for E and I with no re-infection*

We can easily observe that the disease will be wiped out completely as long as there is no re-infection. As re-infection is evident for COVID-19, it really plays a big role in the disease staying in the population even if vaccination program is in place just as seen in the bottom figure of Figure 7.

For waning of immunity after vaccination we have

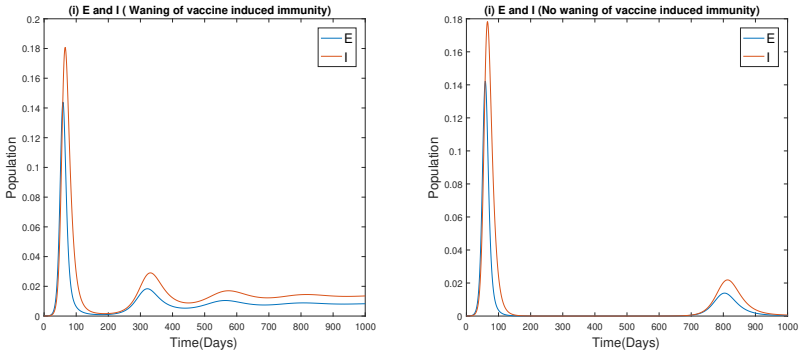

Figure 8. *The Effect of waning immunity after vaccination*

We easily observe in Figure 8 (i) that the disease is kept low in the population for some period of time in case the immunity induced by vaccine does not wane. But this is not enough to eradicate the disease completely out of the population.

For booster vaccination we have the following figure

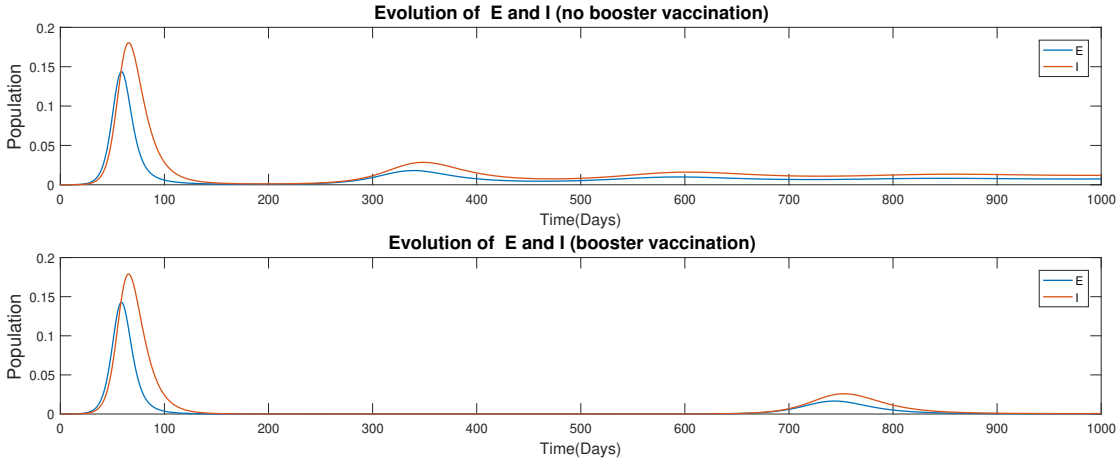

Figure 9. *Evolution for E and I with and without booster vaccination*

We observe that when booster vaccination is included, the infection is kept low in the population for some period of time before it starts increasing to a new peak. This shows that booster vaccination increases the protection period against the disease

4. Conclusion

The infection still persists even when vaccination program is in place. This is due to low vaccination coverage and temporary immunity gained from the vaccine. Higher vaccination coverage tends to eliminate the disease from the population but re-infection as well as waning rate of immunity after vaccination and infection play a major role

for the infection to start increasing again. Booster vaccination reduces the critical value needed to be vaccinated in order to contain the disease. Moreover, booster vaccination increases the period of protection against the disease. The disease staying in the population is due to waning rate of immunity after vaccination and infection, re-infection of individuals and low vaccination coverage.

5. Recommendation

Going for booster vaccination will increase the period of protection of individuals against the disease, individuals are therefore urged to go for booster vaccination. Further development of vaccines that improve the protection against the disease for a longer period is highly recommended. The study has not considered the risk level of infection in different geographical locations. Moreover time dependent waning immunity is more practical. A model considering these two aspects is recommended for future studies.

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Limitations

The model simplifies heterogeneous COVID-19 transmission by assuming homogeneous mixing and fixed parameter values. It does not distinguish between viral variants, age groups, geographical settings, risk categories or different vaccine types. Several simulation parameters are assumed rather than estimated from a single consistent dataset. Time-dependent changes in transmission, vaccination uptake and immunity are not incorporated. The vaccinated and recovered populations are represented through simplified immunity pathways, although their protection may differ. Initial conditions, numerical uncertainty and parameter sensitivity are not fully evaluated. Consequently, the simulations should not be interpreted as forecasts or as sufficient evidence for specific clinical or public-health recommendations.

Declaration of AI Use

This manuscript was prepared through the combined contributions of all author(s), including contributions to the study design, data, content development, results, interpretation, and related scholarly work. The author(s) acknowledge the use of Grammarly and ChatGPT to assist with grammar checking, language refinement, reference formatting. These AI-assisted tools were not used as authors and did not replace the intellectual contributions or scholarly judgment of the author(s). All AI-assisted outputs, including content, references, and interpretations, were carefully reviewed, revised, verified, and approved by the author(s). The author(s) accept full responsibility for the accuracy, integrity, and final content of the manuscript.