



Mathematical Model of COVID-19 Transmission Dynamics with Double Dose Vaccination

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Abstract

This research attempted to investigate the effects of double dose vaccination in a non-linear mathematical model of Covid-19 infections with special compartments class termed first and second dose vaccination. The basic reproduction number was obtained, the stability of the model was analyzed, and the sensitivity analysis was also carried out. Of interest is the numerical simulation of the model where the impacts of contact rate, first and second dose vaccination were studied. The obtained results recommended how to control the corona virus keeping in mind the contact rate and vaccination.

Keywords: Covid-19, Double dose Vaccination, Basic Reproduction Number, Global Stability.

Introduction

COVID-19 pandemic has been identified as a global threat. The causative agent was identified as a novel coronavirus known as Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), and the condition produced by the virus has been called Coronavirus Disease 2019 (COVID-19) by the World Health Organization (WHO 2020). It was confirmed by the WHO that the virus can also be breathed through normal breathing, resulting in new infections. COVID-19 has a 2–14-days incubation period, with approximately 97.5 per cent of infected patients developing symptoms by 11.5 days of infection (Del and Malani 2020, Li et al. 2020, Lai et al. 2020). In Nigeria, as of 4th June 2020, 11,516 cases had been confirmed, with 323 deaths (Adegboye et al. 2020).

In order to better understand how infectious diseases spread and how to prevent them, mathematical models are frequently

used. Several researchers have presented different mathematical models as methods of understanding the spread and control of COVID-19 virus. Das et al. (2021) presented a mathematical model that accounts for the transmission of the COVID-19 infections with a particular isolation class. The model was fitted with data from the ongoing pandemic scenario in India. Okuonghae and Omame (2020) with the aid of a mathematical model conducted an analysis of the spread of COVID-19 pandemic in Lagos, Nigeria. In the same vein, Ajisejiri et al. (2020) similarly conducted an analysis on the outbreak of COVID-19 in Nigeria. Advancing ways of curbing the spread of this deadly disease, Adegboye et al. (2020) presented a mathematical model which dealt with the early transmission of COVID-19 virus in Nigeria. A mathematical model of COVID-19 with vaccination and treatment was presented and analyzed by Diagne et al. (2021); they concluded in their research that

both therapeutic and non-therapeutic measures could later be ways of exterminating the spread of the deadly virus in future. Peter et al. (2021) presented a new mathematical model of COVID-19 using real data from Pakistan. The basic reproductive number that represents an epidemic indicator is obtained from the biggest eigenvalue of the next-generation matrix, and the model is examined qualitatively using the stability theory of differential equations. The disease-free equilibrium's global asymptotic stability criteria are determined. The study concluded that, with a regulated transmission rate, the methods used by the public health sector and the government to address the situation that is causing the pandemic to spread will be more effective.

With the arrival of vaccination, various countries have approved the implementation of safe and effective COVID-19 vaccines for human use, and these include AstraZeneca, Moderna and Pfizer (Gumel et al 2021). An analysis of a mathematical model of COVID-19 on ways of achieving herd immunity threshold was made by Gumel et al. (2021). In their paper, they stated the importance of two dose vaccination. It was emphasized that the first dose vaccine is particularly important to prime the immune system and the second dose is further needed to boost the immune system. The aim of this study was to investigate and analyze the effects of first and second dose vaccination in controlling the spread of COVID-19 pandemic using a mathematical model approach by extending the work of Das et al. (2021). Two additional compartments, namely individuals that are vaccinated with the first dose of vaccine $V_1(t)$ and individuals that are vaccinated with the second dose of vaccine $V_2(t)$ are incorporated. The $S(t)$, $E(t)$, $I(t)$, $Q(t)$, $R(t)$, represents the susceptible, exposed, infected, isolated and recovered classes, respectively. The force of infection is defined as $\lambda = \alpha SI$, where α is the effective

transmission between the susceptible and the infected individuals.

Materials and Methods

Formulation of model

The modeling frame work of this study is based on the previous research by Das et al. (2021), the first and second dose vaccination was incorporated into the previous model.

$$\begin{aligned} \frac{dS}{dt} &= A + \beta SI - \mu S \\ \frac{dE}{dt} &= \beta SI - (\mu + \alpha)E \\ \frac{dI}{dt} &= \alpha E - (\gamma + q + \mu)I \\ \frac{dQ}{dt} &= qI - (\theta + \mu)H \\ \frac{dR}{dt} &= \gamma I + \theta Q - \mu R \end{aligned} \tag{1}$$

We propose a deterministic mathematical model on the transmission dynamics of COVID-19, the population under consideration is divided into seven classes, based on the epidemiological status of individual in population. The total population size $N(t)$ is assumed to be constant and well mixed, that is,

$$N(t) = S(t) + V_1(t) + V_2(t) + E(t) + I(t) + Q(t) + R(t).$$

Hence,

$$\begin{aligned} \frac{dS}{dt} &= \beta - \alpha SI + \omega V_1 - (\mu + \eta)S \\ \frac{dV_1}{dt} &= \eta S - (\omega + \sigma + \mu)V_1 \\ \frac{dV_2}{dt} &= \sigma V_1 - (\mu + \wedge)V_2 \\ \frac{dE}{dt} &= \alpha SI - (\mu + \rho)E \\ \frac{dI}{dt} &= \rho E - (\mu + \delta + \tau + \phi)I \\ \frac{dQ}{dt} &= \phi I - (\mu + \delta + k)Q \\ \frac{dR}{dt} &= \tau I + kQ - \mu R + \wedge V_2 \end{aligned} \tag{2}$$

Table 1: Description, parameters, values and references

Description	Parameters	Values	References
Recruitment rate	β	68,597.853	Estimated
Effective contact rate	α	1.12 days ⁻¹	Babaei et al. (2021)
Progression rate from Exposed to Infected	ρ	1/8 days ⁻¹	Li et al. (2020)
Progression rate from Infected to Isolated class	ϕ	1.923 × 10 ⁻³ days ⁻¹	Adewole et al. (2021)
Recovery rate	τ	1/10 days ⁻¹	Tang et al. (2020)
Treatment rate	k	0.0701	Garba et al. (2020)
Natural death rate	μ	0.0003205	Estimated
Covid-19 Induced death rate	δ	0.018	Garba et al. (2020)
Rate of first dose vaccine	η	0.4	Assumed
Rate at which vaccinated population move to Susceptible	ω	0.2	Assumed
Rate of second dose vaccine	σ	0.5	Assumed
Rate of population from V_2 to Recovery	\wedge	0.02	Assumed

Model analysis

We shall discuss and analyze some basic properties of the proposed model to check for the existence and uniqueness of the solution of the model. Since we wish to check the Covid-19 incidence and prevalence, we focus our attention on system of equations below.

$$\begin{aligned}
 \frac{dS}{dt} &= \beta - \alpha SI + \omega V_1 - (\mu + \eta)S \\
 \frac{dV_1}{dt} &= \eta S - (\omega + \sigma + \mu)V_1 \\
 \frac{dV_2}{dt} &= \sigma V_1 - (\mu + \wedge)V_2 \\
 \frac{dE}{dt} &= \alpha SI - (\mu + \rho)E \\
 \frac{dI}{dt} &= \rho E - (\mu + \delta + \tau + \phi)I \\
 \frac{dQ}{dt} &= \phi I - (\mu + \delta + k)Q
 \end{aligned}
 \tag{3}$$

Existence and uniqueness of solution

A Lipschitz criterion will be used to check for the existence and uniqueness of solution. Thus, if we recall from equation 3, let:

$$\begin{aligned}
 g_1 &= \beta - \alpha SI - \mu S - \eta S + \omega V_1 \\
 g_2 &= \eta S - (\omega + \sigma + \mu)V_1 \\
 g_3 &= \sigma V_1 - (\mu + \wedge)V_2 \\
 g_4 &= \alpha SI - (\mu + \rho)E \\
 g_5 &= \rho E - (\mu + \delta + \tau + \phi)I \\
 g_6 &= \phi I - (\mu + \delta + k)Q
 \end{aligned}
 \tag{4}$$

Theorem 1a: Let E^1 represent the region $0 \leq w \leq R$, then the system of equation in (3) has a

unique solution, provided that $\left| \frac{\partial f_i}{\partial x_i} \right|, i = 1, 2, \dots, 6$ are bounded and continuous.

Proof: From (4) we obtain the partial derivative given below.

For $g_1 = \beta - \alpha SI - \mu S - \eta S + \omega V$

$$\left\{ \begin{aligned} \left| \frac{\partial g_1}{\partial S} \right| &= |-\alpha I - \mu - \eta| < \infty, & \left| \frac{\partial g_1}{\partial V_1} \right| &= |\omega| < \infty, & \left| \frac{\partial g_1}{\partial V_2} \right| &= |0| < \infty, \\ \left| \frac{\partial g_1}{\partial E} \right| &= |0| < \infty, & \left| \frac{\partial g_1}{\partial I} \right| &= |0| < \infty, & \left| \frac{\partial g_1}{\partial Q} \right| &= |0| < \infty \end{aligned} \right\} \quad (5)$$

For $g_2 = \eta S - \omega V_1 - \delta V_1 - \mu V_1$

$$\left\{ \begin{aligned} \left| \frac{\partial g_2}{\partial S} \right| &= |\eta| < \infty, & \left| \frac{\partial g_2}{\partial V_1} \right| &= |-\mu - \omega - \sigma| < \infty, & \left| \frac{\partial g_2}{\partial V_2} \right| &= |0| < \infty \\ \left| \frac{\partial g_2}{\partial E} \right| &= |0| < \infty, & \left| \frac{\partial g_2}{\partial I} \right| &= |0| < \infty, & \left| \frac{\partial g_2}{\partial Q} \right| &= |0| < \infty \end{aligned} \right\} \quad (6)$$

For $g_3 = \sigma V_1 - \mu V_2 - \wedge V_2$

$$\left\{ \begin{aligned} \left| \frac{\partial g_3}{\partial S} \right| &= |0| < \infty, & \left| \frac{\partial g_3}{\partial V_1} \right| &= |0| < \infty, & \left| \frac{\partial g_3}{\partial V_2} \right| &= |-\wedge - \mu| < \infty, \\ \left| \frac{\partial g_3}{\partial E} \right| &= |0| < \infty, & \left| \frac{\partial g_3}{\partial I} \right| &= |0| < \infty, & \left| \frac{\partial g_3}{\partial Q} \right| &= |0| < \infty \end{aligned} \right\} \quad (7)$$

For $g_4 = \alpha SI - \rho E - \mu E$

$$\left\{ \begin{aligned} \left| \frac{\partial g_4}{\partial S} \right| &= |\alpha I| < \infty, & \left| \frac{\partial g_4}{\partial V_1} \right| &= |0| < \infty, & \left| \frac{\partial g_4}{\partial V_2} \right| &= |0| < \infty \\ \left| \frac{\partial g_4}{\partial E} \right| &= |-\mu - \rho| < \infty, & \left| \frac{\partial g_4}{\partial I} \right| &= |\alpha S| < \infty, & \left| \frac{\partial g_4}{\partial Q} \right| &= |0| < \infty \end{aligned} \right\} \quad (8)$$

For $g_5 = \rho E - (\tau + \phi + \mu + \delta)I$

$$\left\{ \begin{aligned} \left| \frac{\partial g_5}{\partial S} \right| &= |0| < \infty, & \left| \frac{\partial g_5}{\partial V_1} \right| &= |0| < \infty, & \left| \frac{\partial g_5}{\partial V_2} \right| &= |0| < \infty, & \left| \frac{\partial g_5}{\partial E} \right| &= |\rho| < \infty, \\ \left| \frac{\partial g_5}{\partial I} \right| &= |-\delta - \mu - \phi - \tau| < \infty, & \left| \frac{\partial g_5}{\partial Q} \right| &= |0| < \infty \end{aligned} \right\} \quad (9)$$

$$\left\{ \begin{aligned} \left| \frac{\partial g_6}{\partial S} \right| &= |0| < \infty, & \left| \frac{\partial g_6}{\partial V_1} \right| &= |0| < \infty, & \left| \frac{\partial g_6}{\partial V_2} \right| &= |0| < \infty, & \left| \frac{\partial g_6}{\partial E} \right| &= |0| < \infty, \\ \left| \frac{\partial g_6}{\partial I} \right| &= |0| < \infty, & \left| \frac{\partial g_6}{\partial Q} \right| &= |\delta + k + \mu| < \infty \end{aligned} \right\} \quad (10)$$

Clearly, all the partial derivatives are Lipschitz continuous and bounded. Hence, the system of equations (3) has a unique solution.

Invariant region

Theorem 1b: Assume that all variables and parameters are non-negative for $t \geq 0$. We

show that the region where the model (3) is sensible, remains positively invariant and attracting to the model for all $t \geq 0$. Thus, all the solution in Ω remains in Ω for all $t \geq 0$.

Proof: The total human population is defined as

$N(t) = S(t) + V_1(t) + V_2(t) + E(t) + I(t) + Q(t)$. Since human population changes with respect to

time. Hence, $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV_1}{dt} + \frac{dV_2}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dQ}{dt}$

$$\therefore \frac{dN}{dt} = \beta - \mu(S + V_1 + V_2 + E + I + Q) - \wedge V_2 - \delta(I + Q) \quad (11)$$

$$\therefore \frac{dN}{dt} \leq \beta - \mu N \quad (12)$$

Integrating both sides of equation (12) yield

$$\int_0^t \frac{dN}{\beta - \mu N} \leq \int_0^t dt \quad (13) \quad \Rightarrow \quad -\frac{1}{\mu} \ln(\beta - \mu N) \leq t \quad (14),$$

From (14), $N \leq \frac{\beta}{\mu} - \left[\frac{\beta - \mu N_0}{\mu} \right] e^{-\mu t}$ thus as $t \rightarrow \infty$ we have $N \leq \frac{\beta}{\mu}$

This implies that the proposed model in (3) can be studied in the feasible region

$$\Omega = \left\{ \left(S, V_1, V_2, E, I, Q \in \mathfrak{R}^6 : N \leq \frac{\beta}{\mu} \right) \right\} \quad (15)$$

Positivity of solution

Theorem 1c: Given $S_0 > 0, V_1 > 0, V_2 > 0, E > 0, I > 0, Q > 0$. Then the solutions

$$\left\{ \left(S, V_1, V_2, E, I, Q \in \mathfrak{R}^6 : N \leq \frac{\beta}{\mu} \right) \right\} \text{ are positively invariant for } t \geq 0.$$

Proof: From equation (3) introducing the force of infection λ for simplicity of the expression,

$$\frac{dS(t)}{dt} \geq -(\mu + \eta) S(t) \quad (16) \quad \Rightarrow \quad \frac{dS(t)}{S(t)} \geq -(\mu + \eta) dt \quad (17)$$

Solving equation (17) by separation of variables, the solution to (17) is obtained as

$$S(t) \geq S_0 e^{-(\mu + \eta)t} \geq 0. \quad (18)$$

Repeating the same procedure for the rest of the equations we obtain,

$$V_1(t) \geq V_{1_0} e^{-(\omega + \sigma + \mu)t} \geq 0 \quad (19)$$

$$V_2(t) \geq V_{2_0} e^{-(\mu + \wedge)t} \geq 0 \quad (20)$$

$$E(t) \geq E_0 e^{-(\mu + \lambda + \rho)t} \geq 0 \quad (21)$$

$$I(t) \geq I_0 e^{-(\mu + \delta + \tau + \phi)t} \geq 0 \quad (22)$$

$$Q(t) \geq Q_0 e^{-(\mu + \delta + k)t} \geq 0 \quad (23)$$

Hence, the solution of the model is positive. This completes the proof of the theorem. Having satisfied all the basic properties of an epidemiology model, we conclude that the proposed model is suitable to study COVID-19 in human population.

Equilibrium analysis

In this section, we discuss the disease free and endemic equilibria of the model, respectively.

Existence of disease free equilibrium

Recall from equation (3), at equilibrium that, $\frac{dS}{dt} = \frac{dV_1}{dt} = \frac{dV_2}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dQ}{dt} = 0$ (25)

By equating the RHS of equation (3) to zero, and setting E, I, Q to zero, and solving the equations we obtain the disease free equilibrium states below.

$$S^0 = \frac{(\omega + \sigma + \mu)\beta}{\mu(\eta + \omega + \sigma + \mu) + \eta\sigma}, \quad V_1^0 = \frac{\eta\beta}{\mu(\omega + \eta + \sigma + \mu) + \eta\sigma} \tag{26}$$

$$V_2^0 = \frac{\beta\sigma\eta}{\mu^2(\omega + \wedge + \eta + \sigma + \mu) + \mu(\omega \wedge + \eta \wedge + \wedge \sigma + \eta\sigma) + \wedge\eta\sigma}, \quad E = 0, Q = 0, I = 0 \tag{27}$$

Existence of endemic equilibrium state

When a disease persists in a population, it is termed endemic equilibrium. In this case E, I, Q is not equal to zero.

The endemic equilibrium point is given as

$$S^* = \frac{k_4 k_5}{\alpha\rho}, \quad V_1^* = \frac{\phi k_4 k_5}{\alpha\rho k_2}, \quad V_2^* = \frac{\sigma\eta k_4 k_5}{\alpha\rho k_2 k_3} \tag{28}$$

$$E^* = \frac{\alpha\beta k_2 \rho - k_1 k_2 k_4 k_5 + k_4 k_5 \omega\eta}{\alpha k_2 k_4 \rho}, \quad I^* = \frac{\alpha\beta k_2 \rho - k_1 k_2 k_4 k_5 + k_4 k_5 \omega\phi}{\alpha k_2 k_4 k_5} \tag{29}$$

$$Q^* = \frac{\phi(\alpha\beta k_2 \rho - k_1 k_2 k_4 k_5 + k_4 k_5 \omega\phi)}{\alpha k_2 k_4 k_5 k_6} \tag{30}$$

Where $k_1 = \mu + \eta, k_2 = \omega + \sigma + \mu, k_3 = \mu + \wedge, k_4 = \mu + \rho, k_5 = \mu + \delta + \tau + \phi$ and $k_6 = \mu + \delta + k$

Basic reproduction number

The basic reproduction number, conventionally denoted by R_0 is defined by Diekmann and Heesterbeek (2000) as the average number of secondary infections generated by a typical infectious individual during his or her entire period of infectiousness. We consider three compartments; E, I, Q .

$$\dot{E} = \alpha SI - (\mu + \rho)E, \quad \dot{I} = \rho E - (\mu + \delta + \tau + \phi)I, \quad \dot{Q} = \phi I - (\mu + \delta + k)Q \tag{31}$$

Hence, let

$$F = \begin{bmatrix} 0 & \alpha S & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}; \quad F_{DFE} = \begin{bmatrix} 0 & \alpha S^0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \tag{32}, \quad \text{and } V = \begin{bmatrix} (\mu + \rho) & 0 & 0 \\ -\rho & (\mu + \delta + \tau + \phi) & 0 \\ 0 & -\phi & (\mu + \delta + k) \end{bmatrix} \tag{33}$$

Thus

$$V^{-1} = \begin{bmatrix} \frac{1}{(\mu + \rho)} & 0 & 0 \\ \frac{\rho}{(\mu + \rho)(\mu + \delta + \tau + \phi)} & \frac{1}{(\mu + \delta + \tau + \phi)} & 0 \\ \frac{\rho\phi}{(\mu + \rho)(\mu + \delta + \tau + \phi)(\mu + \delta + k)} & \frac{\phi}{(\mu + \delta + \tau + \phi)(\mu + \delta + k)} & \frac{1}{(\mu + \delta + k)} \end{bmatrix} \tag{34}$$

The next generation matrix is defined as $G = F * V^{-1}$ and solving the Jacobian matrix above gives

$$R_0 = \frac{\alpha S^0 \rho}{(\mu + \rho)(\mu + \delta + \tau + \phi)}$$

Where $S^0 = \frac{(\omega + \sigma + \mu)\beta}{\mu(\eta + \omega + \sigma + \mu) + \eta\sigma}$

$$R_0 = \frac{\alpha\rho(\omega + \sigma + \mu)\beta}{(\mu + \rho)(\mu + \delta + \tau + \phi)[\mu(\eta + \omega + \sigma + \mu) + \eta\sigma]} \quad (35)$$

Stability analysis

Local stability analysis of diseases-free equilibrium

To examine the stability of the equilibrium of the model, the following outcomes are proven:

Lemma 1: The disease-free equilibrium of the model is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: We consider the Jacobian of the System of equation (3) which is given by

$$J(X^0) = \begin{bmatrix} -(\mu + \eta) & \omega & 0 & 0 & \alpha S^0 & 0 \\ \eta & -(\omega + \sigma + \mu) & 0 & 0 & 0 & 0 \\ 0 & \sigma & -(\mu + \lambda) & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\rho + \mu) & \alpha S^0 & 0 \\ 0 & 0 & 0 & \rho & -(\phi + \delta + \mu + \tau) & 0 \\ 0 & 0 & 0 & 0 & \phi & -(\delta + k + \mu) \end{bmatrix} \quad (36)$$

Using row reduction $\lambda_1 = -(\mu + \lambda)$, and $\lambda_2 = -(\delta + k + \mu)$ and the matrix reduce to 4×4

$$J(X^0) = \begin{bmatrix} -(\mu + \eta) - \lambda & \omega & 0 & -\alpha S^0 \\ \eta & -(\omega + \sigma + \mu) - \lambda & 0 & 0 \\ 0 & 0 & -(\rho + \mu) - \lambda & \alpha S^0 \\ 0 & 0 & \rho & -(\phi + \delta + \mu + \tau) - \lambda \end{bmatrix} \quad (37)$$

The remaining eigenvalues are obtained as

$$\left. \begin{aligned} \lambda_3 &= -\mu - \frac{1}{2}(\omega + \eta + \sigma) + \frac{1}{2}\sqrt{\eta^2 - 2\omega\eta - 2\eta\sigma + 4\eta\omega + \omega^2 + 2\omega\sigma + \sigma^2} \\ \lambda_4 &= -\mu - \frac{1}{2}(\omega + \eta + \sigma) - \frac{1}{2}\sqrt{\eta^2 - 2\omega\eta - 2\eta\sigma + 4\eta\omega + \omega^2 + 2\omega\sigma + \sigma^2} \\ \lambda_5 &= -\mu - \frac{1}{2}(\rho + \delta + \tau + \phi) + \frac{1}{2}\sqrt{\delta^2 + 2\delta\phi - 2\delta\rho + 2\delta\tau + \phi^2 - 2\phi\rho + 2\phi\tau + \rho^2 - 2\rho\tau + 4\rho\alpha S + \tau^2} \\ \lambda_6 &= -\mu - \frac{1}{2}(\rho + \delta + \tau + \phi) - \frac{1}{2}\sqrt{\delta^2 + 2\delta\phi - 2\delta\rho + 2\delta\tau + \phi^2 - 2\phi\rho + 2\phi\tau + \rho^2 - 2\rho\tau + 4\rho\alpha S + \tau^2} \end{aligned} \right\} \quad (38)$$

From the equation above, all roots have negative real part; hence, we concluded that the disease free equilibrium is asymptotically stable.

Local stability analysis of endemic equilibrium

Theorem 2a: Endemic equilibrium state is locally asymptotically stable if the determinant of a Jacobian matrix is greater than zero and the trace of the same matrix is less than zero (Nthiiri et al. 2016).

$$J^* = \begin{pmatrix} -(\alpha I^* + \eta + \mu) & \omega & 0 & 0 & -\alpha S^* & 0 \\ \eta & -(\omega + \sigma + \mu) & 0 & 0 & 0 & 0 \\ 0 & \sigma & -(\mu + \lambda) & 0 & 0 & 0 \\ \alpha I^* & 0 & 0 & -(\rho + \mu) & \alpha S^* & 0 \\ 0 & 0 & 0 & \rho & -(\phi + \delta + \mu + \tau) & 0 \\ 0 & 0 & 0 & 0 & \phi & -(\delta + k + \mu) \end{pmatrix}$$

To calculate the determinant, we assumed the following:

Let

$$\begin{aligned} A &= -(\alpha I^* + \eta + \mu), \quad B = -(\omega + \sigma + \mu), \quad C = -(\mu + \lambda), \\ D &= -(\rho + \mu), \quad E = -(\phi + \delta + \mu + \tau), \quad F = -(\delta + k + \mu), \\ G &= -\alpha S^*, \quad K = \alpha S^*, \quad M = \alpha I^* \end{aligned} \tag{39}$$

Therefore, J^* becomes

$$J^* = \begin{pmatrix} A & \omega & 0 & 0 & G & 0 \\ \eta & B & 0 & 0 & 0 & 0 \\ 0 & \sigma & C & 0 & 0 & 0 \\ M & 0 & 0 & D & K & 0 \\ 0 & 0 & 0 & \rho & E & 0 \\ 0 & 0 & 0 & 0 & \phi & F \end{pmatrix} \tag{40}$$

$$Det.(J^*) = (ABDE - ABK\rho + BGM\rho - DE\eta\omega + K\eta\omega\rho)CF$$

After the evaluation and substitution, the determinant of the above matrix becomes;

$$= \left[(\alpha I^* + \eta + \mu)(\omega + \sigma + \mu)(\rho + \mu)(\phi + \delta + \mu + \tau) - (\alpha I^* + \eta + \mu)(\omega + \sigma + \mu)(\alpha S^*) \right] (\mu + \lambda)(\delta + k + \mu) + \rho(\omega + \sigma + \mu)(\alpha S^*)(\alpha I^*) - (\rho + \mu)(\phi + \delta + \mu + \tau)\eta\omega + \alpha S^*\eta\omega\rho$$

That is, $det.(J^*) > 0$. Now, trace of (J^*) can be defined as the sum of the major diagonal elements. $Trace = A + B + C + D + E + F$ (41)

$$Trace\ of\ (J^*) = [-(\alpha I^* + \eta + \mu) - (\omega + \sigma + \mu) - (\mu + \lambda) - (\rho + \mu) - (\phi + \delta + \mu + \tau) - (\delta + k + \mu)]$$

That is, $- [(\alpha I^* + \eta + \mu) + (\omega + \sigma + \mu) + (\mu + \lambda) + (\rho + \mu) + (\phi + \delta + \mu + \tau) + (\delta + k + \mu)] < 0$

Hence, the trace of $(J^*) < 0$. Thus, the Jacobian matrix (J^*) has eigenvalues that contain negative real parts; therefore, we conclude that the endemic equilibrium is locally asymptotically stable.

Global stability at disease free equilibrium

To prove the global stability, we make use of Castillo-Chavez method (Castillo-Chavez and Song 2004). Consider a model of the form

$$\frac{dF}{dt} = F(x, I), \quad \frac{dI}{dt} = G(x, I), \quad G(x, 0) = 0 \tag{42}$$

Where $x \in \mathfrak{R}^m$ represents, individuals that are not infected in the population and $I \in \mathfrak{R}^n$ represents infected individuals. Following the above representation, the disease free equilibrium state can be written as $U_0 = (x^*, 0)$, the two conditions given below are used to verify the disease-free equilibrium is globally asymptotically stable.

$$(H_1). \text{ For } \frac{dx}{dt} = F(x, 0) \text{ is globally asymptotically stable.}$$

$$(H_2). \ G(x, I) = A\hat{I} - \hat{G}(x, I), \ \hat{G}(x, I) \geq 0 \text{ for all } (x, I) \in \Omega$$

Where $A = D_I G(x^*, 0)$ is an M-matrix (the off diagonal elements of A are non-negative) and Ω is the region where the model makes biological sense.

Lemma 2: The fixed point $U_0 = (x^*, 0)$ is globally asymptotically stable (g.a.s) equilibrium of (41) provided that $R_0 \leq 1$ and assumption that (H_1) and (H_2) are satisfied.

Theorem 2b: If $R_0 < 1$, the disease free equilibrium is globally asymptotically stable and unstable if $R_0 > 1$

Proof: The model equation (3) above is re-written as in form of (41) by setting $x = (S, V_1, V_2)$ and $T = (E, I, Q)$. the disease free equilibrium is given by

$$U_0(x^*, 0) = \left(\frac{(\omega + \sigma + \mu)\beta}{\mu(\eta + \omega + \sigma + \mu) + \eta\sigma}, \frac{\eta\beta}{\mu(\omega + \eta + \sigma + \mu) + \eta\sigma}, \frac{\beta\sigma\eta}{\mu^2(\omega + \lambda + \eta + \sigma + \mu) + \mu(\omega\lambda + \eta\lambda + \lambda\sigma + \eta\sigma) + \lambda\eta\sigma}, 0, 0 \right)$$

And the system $\frac{dx}{dt} = F(x, 0)$ becomes

$$\begin{cases} \dot{S} = \beta + \omega V_1 - (\mu + \eta)S \\ \dot{V}_1 = \eta S - (\omega + \sigma + \eta)V_1 \\ \dot{V}_2 = \sigma V_1 - (\mu + \lambda)V_2 \end{cases} \quad (43)$$

This equation has a unique equilibrium point

$$x^* = \left(\frac{(\omega + \sigma + \mu)\beta}{\mu(\eta + \omega + \sigma + \mu) + \eta\sigma}, \frac{\eta\beta}{\mu(\omega + \eta + \sigma + \mu) + \eta\sigma}, \frac{\beta\sigma\eta}{\mu^2(\omega + \lambda + \eta + \sigma + \mu) + \mu(\omega\lambda + \eta\lambda + \lambda\sigma + \eta\sigma) + \lambda\eta\sigma} \right)$$

Which is asymptotically stable, therefore, the condition (H_1) is satisfied.

For H_2 ; $G(x, T) = \begin{pmatrix} \alpha SI - (\mu - \rho)E \\ \rho E - (\mu + \delta + \tau + \phi)I \\ \phi I - (\mu + \delta + k)Q \end{pmatrix}$

$$D_I G(x^*, 0) = \begin{pmatrix} -(\mu + \rho) & \alpha S & 0 \\ \rho & -(\mu + \delta + \tau + \phi) & 0 \\ 0 & \phi & -(\mu + \delta + k) \end{pmatrix}$$

Clearly, $A = D_I G(x^*, 0)$ is a M-Matrix. On the other hand,

$$\hat{G}(x, T) = AI - G(x, T) = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix} \quad (44) \text{ Hence } \hat{G} = (x, T) \geq 0 \text{ for all } (x, T) \in \Omega; \quad \text{therefore,}$$

conditions (H_1) and (H_2) are satisfied. With theorem 3, the global stability of DFE is obtained and which completes the proof.

Global stability of endemic equilibrium point

Theorem 2b: If $R_0 > 1$, the endemic equilibrium point of the model equation (3) is globally asymptotically stable.

Proof: To establish the global stability of the endemic equilibrium of the model, we construct the following by Lyapunov function.

$$\left\{ \begin{aligned} V(S^*, V_1^*, V_2^*, E^*, I^*, Q^*) &= \left(S - S^* - S^* \ln \frac{S^*}{S} \right) + \left(V_1 - V_1^* - V_1^* \ln \frac{V_1^*}{V_1} \right) \\ &+ \left(V_2 - V_2^* - V_2^* \ln \frac{V_2^*}{V_2} \right) + \left(E - E^* - E^* \ln \frac{E^*}{E} \right) + \left(I - I^* - I^* \ln \frac{I^*}{I} \right) + \left(Q - Q^* - Q^* \ln \frac{Q^*}{Q} \right) \end{aligned} \right\} \quad (45)$$

The derivative of V along this solution of equation (45) by direct calculation gives:

$$\frac{dV}{dt} = \left(\frac{S-S^*}{S} \right) \frac{dS}{dt} + \left(\frac{V_1-V_1^*}{V_1} \right) \frac{dV_1^*}{dt} + \left(\frac{V_2-V_2^*}{V_2} \right) \frac{dV_2^*}{dt} + \left(\frac{E-E^*}{E} \right) \frac{dE}{dt} + \left(\frac{I-I^*}{I} \right) \frac{dI}{dt} + \left(\frac{Q-Q^*}{Q} \right) \frac{dQ}{dt} \quad (46)$$

where

$$\begin{aligned} \frac{dS}{dt} &= \beta - \alpha SI - (\mu + \eta)S + \omega V_1, \quad \frac{dV_1}{dt} = \eta S - (\omega + \sigma + \mu)V_1, \quad \frac{dV_2}{dt} = \sigma V_1 - (\mu + \wedge)V_2, \\ \frac{dE}{dt} &= \alpha SI - (\mu + \rho)E, \quad \frac{dI}{dt} = \rho E - (\mu + \delta + \tau + \phi)I, \quad \frac{dQ}{dt} = \phi I - (\mu + \delta + k)Q \end{aligned} \quad (47)$$

Then, we have

$$\begin{aligned} \Rightarrow &\left(\frac{S-S^*}{S} \right) [\beta - \alpha SI - \mu S - \eta S + \omega V_1] + \left(\frac{V_1-V_1^*}{V_1} \right) [\eta S - (\omega + \sigma + \mu)V_1] + \left(\frac{V_2-V_2^*}{V_2} \right) [\sigma V_1 - (\mu + \wedge)V_2] \\ &+ \left(\frac{E-E^*}{E} \right) [\alpha SI - (\mu + \rho)E] + \left(\frac{I-I^*}{I} \right) [\rho E - (\mu + \delta + \tau + \phi)I] + \left(\frac{Q-Q^*}{Q} \right) [\phi I - (\mu + \delta + k)Q] \end{aligned} \quad (48)$$

Expanding equation (47) above, we obtain;

$$\begin{aligned} = &-\frac{(S-S^*)^2}{S} \alpha (i-i^*)^2 - \frac{(S-S^*)^2}{S} (\mu + \eta) - \frac{(V_1-V_1^*)^2}{V_1} (\omega + \sigma + \mu) - \frac{(V_2-V_2^*)^2}{V_2} (\mu + \wedge) - \frac{(E-E^*)^2}{E} (\mu + \rho) \\ &- \frac{(I-I^*)^2}{I} (\mu + \delta + \tau + \phi) - \frac{(Q-Q^*)^2}{Q} (\mu + \delta + k) + \frac{(S-S^*)}{S} \beta + \frac{(S-S^*)}{S} \omega (V_1 - V_1^*) + \frac{(V_1-V_1^*)}{V_1} \eta (S-S^*) \\ &+ \frac{(V_2-V_2^*)^2}{V_2} \sigma (V_1 - V_1^*) + \frac{(E-E^*)}{E} \alpha (S-S^*) (I-I^*) + \frac{(I-I^*)}{I} \rho (E-E^*) + \frac{(Q-Q^*)}{Q} \phi (I-I^*) \end{aligned} \quad (49)$$

Collecting the positive and negative terms we obtain $\frac{dV}{dt} = M - N$, where M

$$\begin{aligned} = &\frac{(S-S^*)}{S} [\beta + \omega (V_1 - V_1^*)] + \frac{(V_1-V_1^*)}{V_1} \eta (S-S^*) + \frac{(V_2-V_2^*)^2}{V_2} \sigma (V_1 - V_1^*) (I-I^*) \\ &+ \frac{(E-E^*)}{E} \alpha (S-S^*) + \frac{(I-I^*)}{I} \rho (E-E^*) + \frac{(Q-Q^*)}{Q} \phi (I-I^*) \end{aligned} \quad (50)$$

Also

$$\begin{aligned} N &= \frac{(S-S^*)^2}{S} [\alpha (I-I^*) + (\mu + \eta)] + \frac{(V_1-V_1^*)^2}{V_1} (\omega + \sigma + \mu) + \frac{(V_2-V_2^*)^2}{V_2} (\mu + \wedge) + \frac{(E-E^*)^2}{E} (\mu + \rho) \\ &+ \frac{(I-I^*)^2}{I} (\mu + \delta + \tau + \phi) + \frac{(Q-Q^*)^2}{Q} (\mu + \delta + k). \end{aligned} \quad (51)$$

If $M < N$, then $\frac{dV}{dt}$ will be negative

$$\frac{dV}{dt} = 0; \text{ if and only if } S = S^*, V_1 = V_1^*, V_2 = V_2^*, E = E^*, I = I^* \text{ and } Q = Q^* \quad (52)$$

Thus, the largest compact invariant set is $\left\{ (S^*, V_1^*, V_2^*, E^*, I^*, Q^*) \in \Omega : \frac{dV}{dt} = 0 \right\}$ which is the singleton set $\{E^*\}$, hence the endemic equilibrium, by LaSalle's Invariant principles; it implies that E^* is globally asymptotically stable (GAS) in Ω if $M < N$.

Sensitivity analysis

The sensitivity indices with respect to the parameter values are given in form of:

$$\chi_{R_0}^\beta = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0}$$

$$R_0 = \frac{\alpha \rho (\omega + \sigma + \mu) \beta}{(\mu + \rho)(\mu + \delta + \tau + \phi)(\mu^2 + \mu \omega + \mu \eta + \mu \sigma + \eta \sigma)}$$

The sensitivity indices of R_0 to parameters for the model equation (3) are summarized in Table 2.

Table 2: Sensitivity indices of R_0 to parameters for the model equation (3)

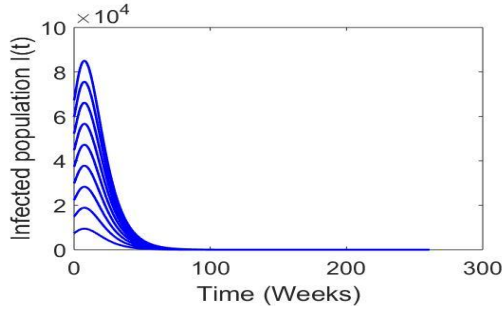
Parameters	α	β	ϕ	ω	σ
Sensitivity indices	1.000000	1.000000	-0.015993	0.285264	-0.285081
Parameters	ρ	δ	τ	η	μ
Sensitivity analysis	0.002558	-0.149696	-0.831646	-0.998879	-0.006526

In the sensitivity indices of R_0 , the most sensitive parameters are the effective contact rate (α) and the recruitment rate β . Another significant parameter is the second dose vaccine rate σ . The positive index indicates that the prevalence of the disease will rise with the increase in the parameter values and the one with negative index will decrease the spread of Covid-19 with increase in the parameter values.

Results and Discussion

We presented in Table 1 the value of each of the parameters for the proposed model. We choose baseline parameter values that are consistent with COVID-19 infections and transmission. The disease-free equilibrium is locally asymptotically stable if the basic

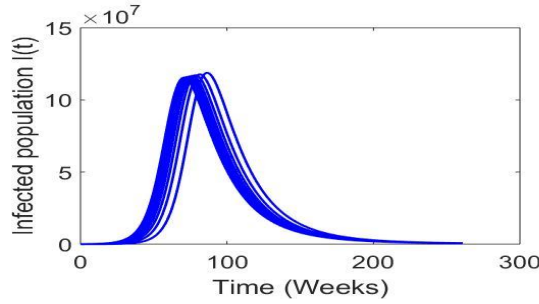
reproduction number is less than one, i.e., $R_0 < 1$. Furthermore, COVID-19 persists in the population if $R_0 > 1$. Figure 1 depicts the disease-free equilibrium's stability, i.e., when $R_0 < 1$. COVID-19 illness is eradicated in this case. Figure 2 depicts the endemic equilibrium's stability, i.e., when $R_0 > 1$, and COVID-19 disease endures in the population. The influence of transmission rate on COVID-19 prevalence is depicted in Figures 3 and 4. Increased vaccination coverage significantly reduced COVID-19 spread, as shown in Figure 4. Increase in second dose vaccine coverage will help slow the spread of the disease, as seen in Figure 5.



$$I(t) = 0, \beta = 68,597.863, \alpha = 1.12, \rho = \frac{1}{8}, \phi = 0.1923, \tau = 0.1, \kappa = 0.0701, \mu = 0.0003205, \delta = 0.018$$

$$\eta = 0.4, \omega = 0.2, \sigma = 0.5, \Lambda = 0.02$$

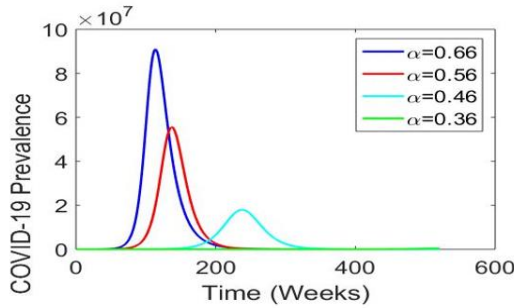
Figure 1: Diseases -free equilibrium: $R_0 < 1$. In this case, Covid-19 disease dies out.



$$I(t) = 0, \beta = 68,597.863, \alpha = 1.12, \rho = \frac{1}{8}, \phi = 0.1923, \tau = 0.1, \kappa = 0.0701, \mu = 0.0003205,$$

$$\delta = 0.018, \eta = 0.4, \omega = 0.2, \sigma = 0.5, \Lambda = 0.02$$

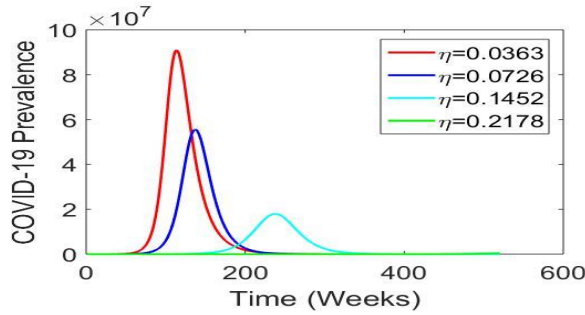
Figure 2: Endemic equilibrium: $R_0 > 1$. In this case Covid-19 disease persists in the population.



$$I(t) = 0, \beta = 68,597.863, \rho = \frac{1}{8}, \phi = 0.1923, \tau = 0.1, \kappa = 0.0701, \mu = 0.0003205, \delta = 0.018$$

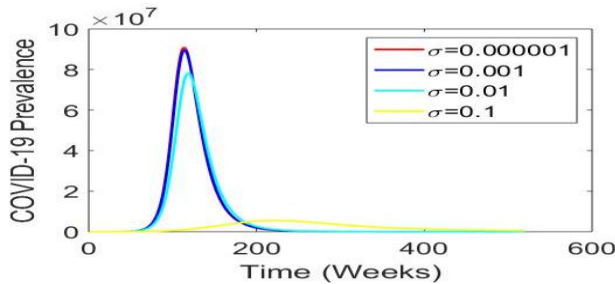
$$\eta = 0.4, \omega = 0.2, \sigma = 0.5, \Lambda = 0.02$$

Figure 3: Impact of transmission rate (effective contact rate) α on COVID-19 prevalence.



$$I(t) = 0, \beta = 68,597.863, \alpha = 1.12, \rho = \frac{1}{8}, \phi = 0.1923, \tau = 0.1, \kappa = 0.0701, \mu = 0.0003205, \\ \delta = 0.018, \omega = 0.2, \sigma = 0.5, \Lambda = 0.02$$

Figure 4: Impact of first dose vaccine η on COVID-19 prevalence.



$$I(t) = 0, \beta = 68,597.863, \alpha = 1.12, \rho = \frac{1}{8}, \phi = 0.1923, \tau = 0.1, \kappa = 0.0701, \mu = 0.0003205, \\ \delta = 0.018, \eta = 0.4, \omega = 0.2, \Lambda = 0.02$$

Figure 5: Impact of second dose vaccine σ on Covid-19 prevalence.

Conclusion and recommendation

A mathematical model on the transmission dynamics of COVID-19 with double dose vaccination was formulated and examined in this work. Sensitivity analysis was used to investigate the impact of model parameters, and the results revealed that the transmission rate has a significant impact on the spread of the Covid-19 epidemic. As a result, every effort should be made to reduce unnecessary transmission among COVID-19 infected and uninfected people. The importance of vaccination in regulating and preventing the spread of COVID-19 was also highlighted. The greatest strategy to stop a COVID-19 outbreak is to vaccinate the entire population. COVID-19 vaccine has a detrimental impact on the prevalence of the disease, according to statistical data. This finding suggests that increasing the vaccine coverage rate reduces COVID-19 dissemination. As a result, mass vaccination

should be promoted to cover the majority of the population in order to achieve a high level of herd immunity for the disease.

Based on the findings of this study, double dose vaccination is recommended as the best approach to use in the fight against the COVID-19 outbreak. As a result, if an intervention is to be adopted during a COVID-19 outbreak, this study suggests that mass vaccination of a double dose vaccine should be promoted to cover majority of the population, as this will reduce the disease's transmission rate.

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Declaration

The authors declare that there are no conflicts of interest concerning the publication of this paper.

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