SABA Publishing

Journal of Mathematical Analysis and Modeling
jmam.sabapub.com
ISSN 2709-5924

J Math Anal & Model
(2025)6(1) : 46-67
doi:10.48185/jmam.v6i1.1276

Modeling the Impact of the AstraZeneca Vaccine on the Transmission Dynamics of COVID-19

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• *Received: 30 July 2024* • *Accepted: 10 April 2025* • *Published Online: 15 June 2025*

Abstract

COVID-19 continues to pose a significant global threat, with millions of cases reported worldwide and substantial mortality. This study develops a deterministic model to evaluate the impact of the AstraZeneca vaccine on the transmission dynamics of COVID-19. The disease-free equilibrium state of the model was determined, and the effective reproduction number for COVID-19, R_e, was computed using the next-generation matrix method. The analysis reveals that R_e is sensitive to vaccination parameters and establishes that the disease-free state is locally and globally asymptotically stable when R_e is less than 1 but unstable when R_e exceeds 1. The findings indicate that an increase in the vaccination rate (θ) and higher vaccination efficacy (γ) significantly reduce the number of COVID-19 infections. This, in turn, leads to a lower incidence of COVID-19 transmission. Enhanced vaccination coverage and effectiveness play a crucial role in preventing the spread of COVID-19, demonstrating the importance of robust immunization programs in public health.

Keywords: COVID-19, Modeling, AstraZeneca Vaccine, Transmission Dynamics, Impact.
2010 MSC: .

1. Introduction

COVID-19 is the disease caused by the SARS-CoV-2 coronavirus [13]. Since its emergence in Wuhan, China, in late 2019, COVID-19 has spread rapidly, leading to millions of infections and significant mortality across the globe [16]. The World Health Organization (WHO) declared COVID-19 a pandemic in March 2020, underscoring the urgent need for effective interventions to control the spread of the virus. COVID-19 primarily spreads through respiratory droplets when an infected person coughs, sneezes, or talks [14]. The virus can also spread by touching surfaces contaminated with the virus and then touching the face [12]. Symptoms of COVID-19 range from mild respiratory issues to severe pneumonia and can lead to acute respiratory distress syndrome (ARDS), multi-organ failure,

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and death, particularly among older adults and those with underlying health conditions [3]. The asymptomatic and pre-symptomatic transmission of the virus further complicates containment efforts [2].

Globally, around 1.5 million new COVID-19 cases and over 2500 deaths were reported from 10 July to 6 August 2023, marking an 80% rise in cases and a 57% drop in deaths compared to the previous period [4]. While most WHO regions saw declines in both, the Western Pacific Region reported more cases but fewer deaths [6]. As of 6 August 2023, over 769 million cases and 6.9 million deaths have been recorded worldwide [7]. Reduced testing and reporting mean current figures may not reflect true infection rates. During this period, 44% of countries reported at least one case, a figure that has been decreasing since mid-2022 [39].

Public health measures such as social distancing, mask-wearing, hand hygiene, and lockdowns have been implemented worldwide to mitigate the spread of the virus [34]. However, these measures, while effective in reducing transmission, have significant social and economic costs [31]. Thus, the development and deployment of vaccines have been seen as the most promising long-term solution to controlling the pandemic and returning to normalcy [33]. Vaccines serve multiple roles in combating infectious diseases: they reduce the susceptibility of individuals to infection, decrease the severity of disease among vaccinated individuals, and can potentially reduce transmission rates if they lower viral load and shedding [75]. Among the vaccines developed, the AstraZeneca vaccine, also known as ChAdOx1-S, has been widely administered across numerous countries.

The AstraZeneca COVID-19 vaccine, co-developed by the University of Oxford and Vaccitech, uses a replication-deficient chimpanzee adenovirus vector [73]. This vector, derived from a weakened chimpanzee cold virus, includes genetic material from the SARS-CoV-2 spike protein [71]. After vaccination, the spike protein is produced, enabling the immune system to recognize and fight the SARS-CoV-2 virus [69]. The AstraZeneca vaccine, an adenoviral vector vaccine, has demonstrated efficacy in preventing symptomatic COVID-19 and reducing severe outcomes, but its influence on transmission dynamics requires further exploration [60]. The AstraZeneca vaccine shows a 72% efficacy against symptomatic COVID-19, with efficacy increasing with longer intervals between doses [59]. The recommended administration consists of two intramuscular doses (0.5 ml each), spaced 8 to 12 weeks apart [58]. The full two dose regimen of this vaccine is believed to be more protective against variants of concern than a single dose alone [55]. The AstraZeneca vaccine is available for individuals who have recovered from COVID-19, though they may opt to postpone vaccination for 3 months after their infection [54]. However, people with a history of severe allergic reaction to any component of the vaccine should not take it [52]. The AstraZeneca vaccine is safe and effective in protecting individuals from the severe risks of COVID-19, including death, hospitalization, and serious illness [51].

The decision to focus on the AstraZeneca vaccine, rather than other vaccines, is based on several factors. Firstly, the AstraZeneca vaccine's widespread global distribution makes it a crucial component in the fight against COVID-19, particularly in low- and middle-income countries. Additionally, its relatively simple storage requirements compared to mRNA vaccines like Pfizer-BioNTech and Moderna make it more accessible in regions with limited cold chain infrastructure. Furthermore, the extensive clinical data available on the

AstraZeneca vaccine provides a robust foundation for analyzing its impact on transmission dynamics. Numerous mathematical models addressing infectious diseases use systems of differential equations to depict disease dynamics at various levels [42, 43, 44, 45, 46, 47]. Various mathematical models have been developed to analyze COVID-19 transmission dynamics, predict infection peaks, and propose mitigation strategies [5, 8, 9, 10, 15, 17, 18, 19, 20].

Venkatesh and Rao [25] devised a mathematical model for the COVID-19 pandemic, incorporating intervention strategies and cost-effectiveness analysis. Their findings indicate that combining vaccination and treatment is the optimal and least expensive approach to reducing the spread of COVID-19 infections. Burch et al.[27] conducted a systematic review of mathematical models concerning COVID-19 vaccination in high-income countries. Their work emphasizes the importance of comprehensive models that prioritize outcome measures such as quality-adjusted life years, the population-level impacts of long COVID, and the cost-effectiveness of future policies.

Liang et al. [32] developed a mathematical modeling study on influenza and COVID-19 co-infection and vaccine effectiveness against severe cases. Their study found that to minimize the number of severe illnesses resulting from co-infection of influenza and COVID-19, vaccinations in the population are crucial, with particular priority given to the elderly. Paul et al. [35] conducted a mathematical analysis and simulation of a COVID-19 model focusing on a booster dose vaccination strategy in Bangladesh. Their findings indicate that increasing the first dose vaccination rate is more effective in reducing the number of asymptomatic and symptomatic cases than focusing on second and booster doses. Dickson et al.[36] developed a study on the transmission dynamics of the omicron variant of COVID-19 using nonlinear mathematical models. Their study found that symptomatic cases play a significant role in the spread and prevalence of Omicron infection within communities.

Omorogie et al. [48] present a non-linear deterministic mathematical model to investigate the population dynamics of COVID-19 in the context of vaccination. The study's findings indicate that concurrently increasing treatment rates and maintaining a relatively high vaccination rate will lead to a corresponding decline in COVID-19 cases within the population. Kavya et al. [49] explore the influence of vaccination and booster doses on the spread of Omicron through mathematical modeling. Their research emphasizes the importance of booster doses, given that the effectiveness of immunization can decrease over time. Chan et al. [50] developed a study which focuses on modeling geographic vaccination strategies for COVID-19 in Norway. The study's findings indicate that prioritizing vaccination early on could decrease COVID-19 related health outcomes by 8% to 20% compared to a baseline strategy that lacks geographic prioritization.

Idisi et al. [37] conducted a bifurcation analysis and created a model to study the dynamics of COVID-19 transmission, considering the impact of post-vaccination infections. Their findings suggest that enhancing vaccination coverage and reducing the rate at which vaccine efficacy wanes would significantly lower the reproduction number below one, potentially leading to the eradication of the disease from the population. Kambali et al.[40] developed a study on the nonlinear dynamic epidemiological analysis of effects of vaccination and dynamic transmission on COVID-19. Their study suggests that an increase in vaccination rates, enhancing vaccine efficacy, and enacting public measures to reduce both

static and dynamic transmission rates would effectively suppress oscillatory behavior and aid in the complete eradication of the disease. Sepulveda et al. [41] discuss the mathematical modeling of COVID-19 dynamics considering the effects of two vaccination doses and delays. Their research determined that increasing vaccination rates can significantly lessen the impact of the COVID-19 pandemic.

However, despite the extensive research on COVID-19 vaccination strategies, there is a noticeable gap in the literature regarding the specific impact of the AstraZeneca vaccine on the transmission dynamics of COVID-19. While many studies focus on general vaccination effects, booster doses, and co-infection scenarios, there is a lack of detailed mathematical models that isolate and analyze the effects of the AstraZeneca vaccine alone. Addressing this gap is crucial for developing targeted public health strategies and optimizing vaccination campaigns, particularly in regions where the AstraZeneca vaccine is predominantly used. This study aims to fill this gap by modeling the specific impact of the AstraZeneca vaccine on COVID-19 transmission dynamics, providing valuable insights into its effectiveness and implications for controlling the pandemic.

2. COVID-19 Model Formulation

In this section, a compartment model is constructed to illustrate the transmission dynamics of COVID-19 and the impact of the AstraZeneca vaccine within a population. The total population, denoted by $N(t)$, is divided into five compartments: Susceptible $S(t)$, individuals who are not infected but can become infected; Vaccinated $V(t)$, individuals with partial protection from the vaccine; Exposed, $E(t)$, individuals infected but not yet infectious; Infected $I(t)$, individuals who are currently infected and can transmit the virus; and Recovered $R(t)$, individuals with temporary immunity. Thus, $N(t) = S(t) + V(t) + E(t) + I(t) + R(t)$. The dynamics involve various transitions. The susceptible class $S(t)$ increases through new individuals at a rate of Λ. Susceptible individuals $S(t)$ become exposed $E(t)$ at a rate β upon contact with infected individuals $I(t)$. Vaccination occurs at a rate θ, moving individuals to the vaccinated class $V(t)$, providing partial protection with efficacy γ. This protection wanes over time, and vaccinated individuals revert to the susceptible class $S(t)$ at a rate η. Exposed individuals $E(t)$ become infectious at a rate σ, moving to the infected class $I(t)$. Infected individuals either recover at a rate ρ or die at a rate δ. Recovered individuals lose immunity and return to the susceptible class $S(t)$ at a rate ϕ. Mortality rates include natural deaths at a rate of μ. Table 1 shows the description of the parameters used in the model.

2.1. Compartmental Flow Diagram of COVID-19 Transmission Dynamics

A compartmental flow diagram illustrates disease transmission stages and immunity states within a population. Figure 1 depicts relationships between Susceptible, Exposed, Vaccinated, Infected, and Recovered individuals.

2.2. Model Equations for the Transmission Dynamics of COVID-19 with the AstraZeneca Vaccine

These equations detail COVID-19 transmission dynamics, specifically addressing the AstraZeneca vaccine's impact. They model infection spread, including vaccination's effects

Table 1: Model parameters and their description

Parameter	Description	Value	Source
Λ	Humans recruitment rate	0.463603436	[1]
β	Humans effective contact rate	0.4531	[23]
μ	Humans natural death rate	0.0141784	[28]
θ	Humans vaccination rate	0.01	[20]
η	Rate of return to susceptible state	0.04	[23]
σ	Progression rate from exposed to infectious	0.200	Assumed
ρ	Recovery rate of infected humans	0.500	Assumed
γ	Vaccine efficacy	0.88	[26, 29]
ϕ	Rate of returning to susceptible state	0.011	[23]
δ	Disease-induced death rate	0.0119	[24]

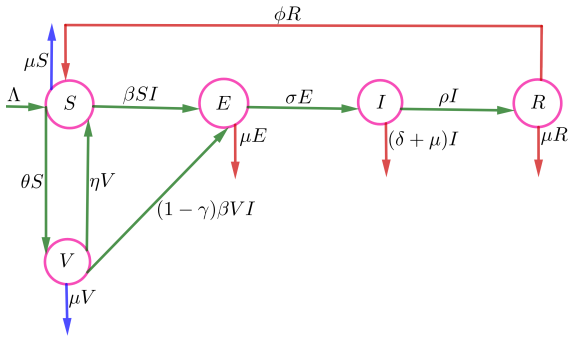

Figure 1: Dynamics of COVID-19 Transmission with AstraZeneca Vaccination

on transmission, progression, and control strategies. The model is represented by the following system of differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda + \eta V + \phi R - (\theta + \beta I + \mu)S \\
\frac{dV}{dt} &= \theta S - ((1-\gamma)\beta I + \eta + \mu)V \\
\frac{dE}{dt} &= \beta SI + (1-\gamma)\beta VI - (\alpha + \mu)E \\
\frac{dI}{dt} &= \alpha E - (\delta + \sigma + \mu)I \\
\frac{dR}{dt} &= \delta I - (\phi + \mu)R
\end{aligned} \tag{2.1}$$

3. Qualitative Analysis of the Model

3.1. Non-negativity of Solution

To ensure the ecological and epidemiological relevance of the model and to establish its mathematical robustness, it is crucial to demonstrate that all variables in the system, beginning with positive initial values, will remain non-negative as time progresses ($t \geqslant 0$).

Lemma 3.1. *Given the initial conditions* $\{S(0) > 0, V(0) \geqslant 0, I(0) \geqslant 0, C(0) \geqslant 0\}$ *and* $R(0) \geqslant 0$ *in* $\mathbb{R}^5_+$*, it ensures that the variables* $\{(S(t), V(t), E(t), I(t), R(t))\}$ *remain non-negative for all times* $t \geqslant 0$.

Proof. From the first equation of the model system (2.1), then we have;

$$\begin{aligned} \frac{dS}{dt} &= \Lambda + \eta V + \phi R - (\theta + \beta I + \mu)S \\ \frac{dS}{dt} &\geqslant -(\theta + \beta I + \mu)S \\ \frac{dS}{S} &\geqslant -(\theta + \beta I + \mu)dt \end{aligned} \tag{3.1}$$

By separating the variables, we have;

$$\int_{s(0)}^{s(t)} \frac{dS}{S} \geqslant -\int_0^t (\theta + \beta I + \mu)dt$$

$$\ln S(t) - \ln S(0) \geqslant -(\theta + \beta I + \mu)t$$

Upon simplifying further, we get;

$$S(t) \geqslant S(0)e^{-(\theta + \beta I + \mu)t} \tag{3.2}$$

Therefore, the solution $S(t)$ remains positive for all t provided that the initial susceptible population $S(0)$ is positive and the exponential term $e^{-(\theta+\beta I+\mu)t}$ stays non-negative. Conversely, it can be demonstrated that $V(0) > 0$, $E(0) \geqslant 0$, $I(0) \geqslant 0$, and $R(0) \geqslant 0$. Thus, all solution sets maintain positive values for $t \geqslant 0$, affirming the meaningfulness and well-posedness of the model. □

3.2. Invariant region

In this subsection, we investigate whether model variables have biological interpretation and a unique bounded solution that exists for all the time. From the model system (2.1), we have:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \Lambda - \mu N - \sigma I. \tag{3.3}$$

$$\frac{dN}{dt} \leqslant \Lambda - \mu N$$

Solving this, we obtain

$$0 \leqslant N(t) \leqslant \frac{\Lambda}{\mu} + N(0)\exp^{-\mu t}. \tag{3.4}$$

As $t \to \infty$, we have $0 < N(t) \leqslant \frac{\Lambda}{\mu}$. Hence, the model solution is feasible and positively invariant in the region

$$\Omega = \left\{ (S, V, E, I, R) \geqslant 0 \in \mathbb{R}^5_+ : S + V + E + I + R \leqslant \frac{\Lambda}{\mu} \right\}. \tag{3.5}$$

The presence of a feasible solution in the model, which remains positive in $\mathbb{R}^5_+$, indicates that the model system is both epidemiologically and mathematically well-defined. This characteristic of the model allows us to continue with additional mathematical analysis.

3.3. COVID-19-Free equilbrium Point

The equilibrium point of the model where there are no infected nodes is determined by setting the model system (2.1) to zero and establishing $S = 0$, $F = 0$, $E = 0$, $I = 0$, and $R = 0$. Hence, the disease-free equilibrium point, denoted as E^0 in a model system, is expressed as:

$$E^0 = (S^0, V^0, E^0, I^0, R^0) = \left(\frac{\Lambda(\eta+\mu)}{\mu(\eta+\mu)}, \frac{\Lambda\theta}{\mu(\eta+\mu)}, 0, 0, 0 \right) \tag{3.6}$$

3.4. Effective Reproduction Number

The effective reproduction number, R_e, signifies the average number of secondary infections that may arise when a single infected individual is introduced into a susceptible population, while certain interventions are in place to curb the spread of the disease [72]. Calculating R_e involves considering the disease compartments as follows:

$$\dot{X}_i = F_i(x) - V_i(x) \tag{3.7}$$

where F_i is the rate of appearance of new infection in compartment i and V_i is the transfer of infections from one compartment i to another. We employ the standard methodology proposed by Diekmann et al. [70, 74] and adopted by Mrope and Nyerere [68] to calculate the effective reproduction number, denoted as R_e, for model system (2.1) as:

$$FV^{-1} = \left[\frac{\partial F_i(E_0)}{\partial x_j} \right] \left[\frac{\partial V_i(E_0)}{\partial x_j} \right]^{-1} \tag{3.8}$$

Equation 2.1 delineates the disease compartments as follows:

$$\frac{dE}{dt} = \beta SI + (1-\gamma)\beta VI - (\alpha+\mu)E \tag{3.9}$$

$$\frac{dI}{dt} = \alpha E - (\delta+\sigma+\mu)I \tag{3.10}$$

Putting the equations in 3.7 in terms of the system of Equations in 3.11 as follows

$$\dot{X}_i = \begin{bmatrix} \beta SI + (1-\gamma)\beta VI \\ 0 \end{bmatrix} - \begin{bmatrix} (\alpha+\mu)E \\ (\alpha+\delta+\mu)I - \alpha E \end{bmatrix} \tag{3.11}$$

which implies that

$$\mathcal{F}_i = \begin{bmatrix} \beta SI + (1-\gamma)\beta VI \\ 0 \end{bmatrix}$$

and

$$\mathcal{V}_i = \begin{bmatrix} (\alpha+\mu)E \\ (\alpha+\delta+\mu)I - \alpha E \end{bmatrix}$$

Then the Jacobian matrix for $\mathcal{F}_i$ and $\mathcal{V}_i$ by using the system of Equations in (7) are given as

$$F = \frac{\partial \mathcal{F}_i}{\partial(E,I)} = \begin{bmatrix} 0 & \frac{\beta\Lambda(\eta+\mu)}{\mu(\eta+\mu)} + \frac{\beta(1-\gamma)\Lambda\theta}{\mu(\eta+\mu)} \\ 0 & 0 \end{bmatrix}$$

and

$$V = \frac{\partial \mathcal{V}_i}{\partial(E,I)} = \begin{bmatrix} \alpha+\mu & 0 \\ -\alpha & \alpha+\delta+\mu \end{bmatrix}$$

which implies that

$$V^{-1} = \frac{1}{(\alpha+\mu)(\alpha+\delta+\mu)} \begin{bmatrix} \alpha+\delta+\mu & 0 \\ \alpha & \alpha+\mu \end{bmatrix}$$

Following computation, the effective reproduction number was determined to be:

$$R_e = \frac{\alpha\beta\Lambda((1-\gamma)\theta+\mu+\eta)}{\mu(\alpha+\mu)(\sigma+\delta+\mu)(\theta+\mu+\eta)} \tag{3.12}$$

3.5. Local stability of DFE

In this part, eigen-value method is applied to investigate the local stability of the COVID-19-free equilbrium point for the model system 2.1.

Theorem 1. The disease free equilibrium for the model system(2.1) is locally asymptotically stable if $R_e < 1$ and unstable if $R_e > 1$.

Proof. We show that the variational matrix $J(E_0)$ of the COVID-19-free model system have only negative eigenvalues. The Jacobian matrix for the model system (2.1) is given by:

$$J(E_0) = \begin{bmatrix} -(\theta+\mu) & \eta & 0 & -\frac{\beta\Lambda(\eta+\mu)}{\mu(\eta+\mu)} & \phi \\ \theta & -(\eta+\mu) & 0 & \frac{\beta(1-\gamma)\Lambda\theta}{\mu(\eta+\mu)} & 0 \\ 0 & 0 & -(\alpha+\mu) & \frac{\beta(1-\gamma)\Lambda\theta}{\mu(\eta+\mu)} & 0 \\ 0 & 0 & \alpha & -(\delta+\sigma+\mu) & 0 \\ 0 & 0 & 0 & \delta & -(\phi+\mu) \end{bmatrix}$$

We observe that the Jacobian matrix $J(E_0)$ have five distinct eigenvalues given by $\lambda_1 = -(\theta+\mu)$, $\lambda_2 = -(\eta+\mu)$, $\lambda_3 = -(\alpha+\mu)$, $\lambda_4 = -(\delta+\sigma+\mu)$ and $\lambda_5 = -(\phi+\mu)$.
All the eigenvalues have negative real parts. In this case, all roots of the Jacobian matrix $J(E_0)$ are negative. Hence, the disease-free equilbrium E_0 is locally asymptotically stable at $R_e < 1$. □

3.6. Global stability of DFE

In this section, we delve into the global stability analysis of DFE, utilizing the method pioneered by Castillo-Chavez et al. [30] and adopted by Nyerere et al. [38] and Mrope and Kigodi [22].

Proof. To show the global stability of the disease-free equilibrium (DFE) of the given model, we begin by defining a Lyapunov function. Let us consider the Lyapunov function L given by

$$L = f_1 S + f_2 V + f_3 E + f_4 I + f_5 R,$$

where f_1, f_2, f_3, f_4, and f_5 are positive constants that will be determined later.

The derivative of L with respect to time t is

$$\frac{dL}{dt} = f_1 \frac{dS}{dt} + f_2 \frac{dV}{dt} + f_3 \frac{dE}{dt} + f_4 \frac{dI}{dt} + f_5 \frac{dR}{dt}.$$

Using the system of equations provided, we have

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda + \eta V + \phi R - (\theta + \beta I + \mu) S, \\
\frac{dV}{dt} &= \theta S - [(1-\gamma)\beta I + \eta + \mu] V, \\
\frac{dE}{dt} &= \beta S I + (1-\gamma)\beta V I - (\alpha + \mu) E, \\
\frac{dI}{dt} &= \alpha E - (\delta + \sigma + \mu) I, \\
\frac{dR}{dt} &= \delta I - (\phi + \mu) R.
\end{aligned}$$

Substituting these into the derivative of L, we get

$$\begin{aligned}
\frac{dL}{dt} = {} & f_1 \left[\Lambda + \eta V + \phi R - (\theta + \beta I + \mu) S\right] \\
& + f_2 \left[\theta S - [(1-\gamma)\beta I + \eta + \mu] V\right] \\
& + f_3 \left[\beta S I + (1-\gamma)\beta V I - (\alpha + \mu) E\right] \\
& + f_4 \left[\alpha E - (\delta + \sigma + \mu) I\right] \\
& + f_5 \left[\delta I - (\phi + \mu) R\right].
\end{aligned}$$

Rearranging the terms, we get

$$\begin{aligned}
\frac{dL}{dt} = {} & f_1 \Lambda + f_1 \eta V + f_1 \phi R - f_1 (\theta + \beta I + \mu) S \\
& + f_2 \theta S - f_2 [(1-\gamma)\beta I + \eta + \mu] V \\
& + f_3 \beta S I + f_3 (1-\gamma)\beta V I - f_3 (\alpha + \mu) E \\
& + f_4 \alpha E - f_4 (\delta + \sigma + \mu) I \\
& + f_5 \delta I - f_5 (\phi + \mu) R.
\end{aligned}$$

To simplify, we choose f_1, f_2, f_3, f_4, and f_5 such that the coefficients of S, V, E, I, and R terms balance each other. Let us set:

$$f_1 = 1, \quad f_2 = \frac{\theta(1-\gamma)\beta}{\theta}, \quad f_3 = \frac{\beta}{\alpha}, \quad f_4 = \frac{\alpha}{\delta}, \quad f_5 = \frac{\delta}{\phi}.$$

Substituting these values, we get

$$\begin{aligned}\frac{dL}{dt} &= \Lambda + \eta V + \phi R - (\theta + \beta I + \mu)S \\ &\quad + \frac{\theta(1-\gamma)\beta}{\theta}\left[\theta S - [(1-\gamma)\beta I + \eta + \mu]V\right] \\ &\quad + \frac{\beta}{\alpha}\left[\beta SI + (1-\gamma)\beta VI - (\alpha+\mu)E\right] \\ &\quad + \frac{\alpha}{\delta}\left[\alpha E - (\delta + \sigma + \mu)I\right] \\ &\quad + \frac{\delta}{\phi}\left[\delta I - (\phi + \mu)R\right].\end{aligned}$$

Simplifying each term:

$$\begin{aligned}\frac{dL}{dt} &= \Lambda - \mu S - \beta SI + \left(\eta - \frac{\eta(1-\gamma)\beta}{\theta} - \frac{\mu(1-\gamma)\beta}{\theta}\right)V \\ &\quad + \left(\phi - \frac{\delta\phi}{\phi} - \frac{\delta\mu}{\phi}\right)R \\ &\quad + \left(\frac{\beta^2 SI}{\alpha} - \frac{\beta(\alpha+\mu)}{\alpha}E\right) \\ &\quad + \left(\frac{\alpha^2 E}{\delta} - \frac{\alpha(\delta+\sigma+\mu)}{\delta}I\right).\end{aligned}$$

Since $S(t) \leqslant N(t)$ for every $t \in \Omega$, we choose the constants f_1, f_2, f_3, f_4, and f_5 such that:

$$\begin{aligned}\eta - \frac{\eta(1-\gamma)\beta}{\theta} - \frac{\mu(1-\gamma)\beta}{\theta} &\leqslant 0, \\ \phi - \frac{\delta\phi}{\phi} - \frac{\delta\mu}{\phi} &\leqslant 0, \\ \frac{\beta^2}{\alpha} &\leqslant \beta\frac{(\alpha+\mu)}{\alpha}, \\ \frac{\alpha^2}{\delta} &\leqslant \alpha\frac{(\delta+\sigma+\mu)}{\delta}.\end{aligned}$$

After rearranging and solving these inequalities, we find that $L' \leqslant 0$ if $\mathcal{R}_e \leqslant 1$, where $\mathcal{R}_e$ is the effective reproduction number of the system. Hence, the largest invariant set where $L' = 0$ is the disease-free equilibrium E_0. By LaSalle's Invariance Principle, the given model system is globally asymptotically stable in Ω if $\mathcal{R}_e \leqslant 1$. Therefore, the DFE is globally asymptotically stable. □

3.7. Disease Endemic Equilbrium Point

The disease endemic equilbrium point of the model may be obtained by equating each equation of the model system (2.1) equal to zero, that is;

$$\begin{aligned}
\Lambda+\eta V^*+\phi R^*-(\theta+\beta I^*+\mu)S &= 0\\
\theta S^*-((1-\gamma)\beta I^*+\eta+\mu)V^* &= 0\\
\beta S^* I^*+(1-\gamma)\beta V^* I^*-(\alpha+\mu)E^* &= 0\\
\alpha E^*-(\delta+\sigma+\mu)I^* &= 0\\
\delta I^*-(\phi+\mu)R^* &= 0
\end{aligned}$$

Computing Endemic Equilbrium point (EE) from eqn above, we get;

$$\begin{cases}
S^* = \frac{\Lambda+\eta V^*+\phi R^*}{\theta+\beta I^*+\mu}\\
F^* = \frac{\theta S^*}{((1-\gamma)\beta I^*+\eta+\mu)}\\
E^* = \frac{\beta S^* I^*+(1-\gamma)\beta V^* I^*}{\alpha+\mu}\\
I^* = \frac{\alpha E^*}{\delta+\sigma+\mu}\\
R^* = \frac{\delta I^*}{\phi+\mu}
\end{cases}$$

3.8. Global Stability of Endemic Equilbrium Points

The global stability of the endemic equilibrium point (E^*) was examined using the Lyapunov function. In accordance with the Lyapunov function a point is considered to be asymptotically globally stable if the derivative of the function is negative.

Theorem 2. The COVID-19 has a unique endemic equilibrium point E^* for the model system that is globally asymptotically stable if $R_e > 1$ and unstable otherwise.

Proof. A Lyapunov function of the model system (2.1), as described by Vargas-De-León [64], Korobeinikov et al. [65], and Korobeinikov [67], was employed in this study. The Lyapunov function L is defined by

$$L(\mathbf{x}) = \sum_{i=1}^{n} \frac{1}{2}(\mathbf{x}_i - \mathbf{x}_i^*)^2,$$

Here, the population of the i-th compartment is denoted by $\mathbf{x}_i$, while $\mathbf{x}_i^*$ designates the endemic equilibrium point.
The model system (2.1) exhibits the following positive definite function

$$P(S,V,E,I,R) = \sum_{i=1}^{5} \frac{1}{2}(\mathbf{x}_i - \mathbf{x}_i^*)^2,$$

Then the above Lyapunov function of the MSD model system is written as:

$$L = \frac{1}{2}[(S-S^*)+(V-V^*)+(E-E^*)+(I-I^*)+(R-R^*)]^2.$$

Then, differentiation of the function $L(t)$ with respect to time results in:

$$\frac{dL}{dt} = [(S - S^*) + (V - V^*) + (E - E^*) + (I - I^*) + (R - R^*)]\frac{d}{dt}[S + V + E + I + R],$$
$$\frac{dL}{dt} = [S + V + E + I + R - (S^* + F^* + E^* + I^* + R^*)]\frac{d}{dt}[S + V + E + I + R].$$

However,

$$\frac{d}{dt}(S + F + E + I + R) = \Lambda - \sigma I - \mu N.$$

Furthermore,

$$\begin{aligned} \Lambda - \sigma I^* - \mu N^* &= 0, \\ \Rightarrow \Lambda - \sigma I^* - \mu(S^* + V^* + E^* + I^* + R^*) &= 0, \\ (S^* + V^* + E^* + I^* + R^*) &= \frac{\Lambda - \sigma I^*}{\mu}. \end{aligned}$$

Substituting into $\frac{dL}{dt}$ gives

$$\begin{aligned} \frac{dL}{dt} &= \left[N(t) - \frac{(\Lambda - \sigma I^*)}{\mu}\right][\Lambda - \sigma I - \mu N(t)]. \\ \frac{dL}{dt} &= \left[N(t) - \frac{(\Lambda - \sigma I^*)}{\mu}\right][-\mu(N(t) - (\Lambda - \sigma I))]. \\ \frac{dL}{dt} &= -\mu\left[N(t) - \frac{\Lambda}{\mu} + \frac{\sigma I^*}{\mu}\right]\left[N(t) - \frac{\Lambda}{\mu} + \frac{\sigma I}{\mu}\right]. \\ \frac{dL}{dt} &= -\mu\left[N(t) - \frac{\Lambda}{\mu}\right]\left[N(t) - \frac{\Lambda}{\mu}\right]. \\ \frac{dL}{dt} &\leqslant -\mu\left[N(t) - \frac{\Lambda}{\mu}\right]^2 < 0. \end{aligned}$$

Thus, it is clear that $\frac{dL}{dt} < 0$.
Therefore, the endemic equilibrium point (E^*) is globally asymptotically stable. □

4. Results and Discussion

4.1. Sensitivity Analysis

The partial rank correlation coefficient (PRCC) method was employed to evaluate the sensitivity of parameters to various variables. The PRCC is a global sensitivity analysis method that examines uncertainties in model parameters on a global scale, unlike local sensitivity methods. PRCC measures the strength of the relationship between parameters and variables using a correlation coefficient ranging from -1 to 1. Values approaching 1 and -1 indicate a strong positive and strong negative correlation, respectively, while values near 0 indicate a weak correlation. In this study, values between -0.3 and 0.3 have been labeled to indicate a weak correlation. Since PRCC is based on sampling, we employed Latin Hypercube Sampling (LHS), one of the Monte Carlo sampling schemes, with 1,000 samples, as shown in Figure 2. We assumed a uniform distribution for all sampled parameters.

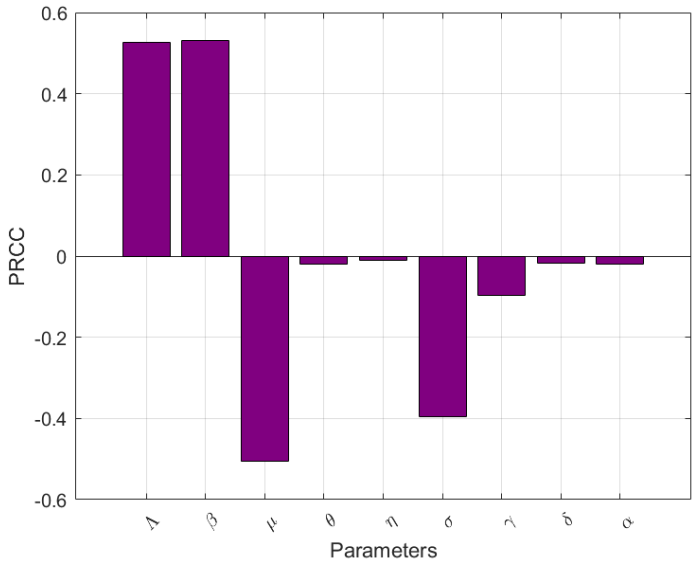

Figure 2: PRCC values of the model parameters for R_e of the system

4.2. Numerical Simulation

In this section, the mathematical model developed for analyzing the impact of the AstraZeneca vaccine on COVID-19 transmission is implemented using MATLAB. This involves using computational methods to simulate the complex dynamics of disease spread and vaccination effects. The parameter values provided in Table 1 are used as inputs for these simulations to ensure that the model accurately represents real-world conditions. The simulations start with initial conditions of $S = 1000, V = 500, E = 100, I = 50, R = 0$, which create a baseline scenario. These conditions are selected to reflect a population with varying levels of susceptibility, vaccination coverage, and infection rates, allowing for a detailed examination of how the vaccine influences disease dynamics over time.

4.2.1. COVID-19 case rates within the overall population

The incidence of the disease across the entire population refers to the number of new cases of COVID-19 that occur within a given population over a specific period. This measure is crucial for understanding how the disease spreads through the community. Figure 3 illustrates the disease trend over time in the whole population.

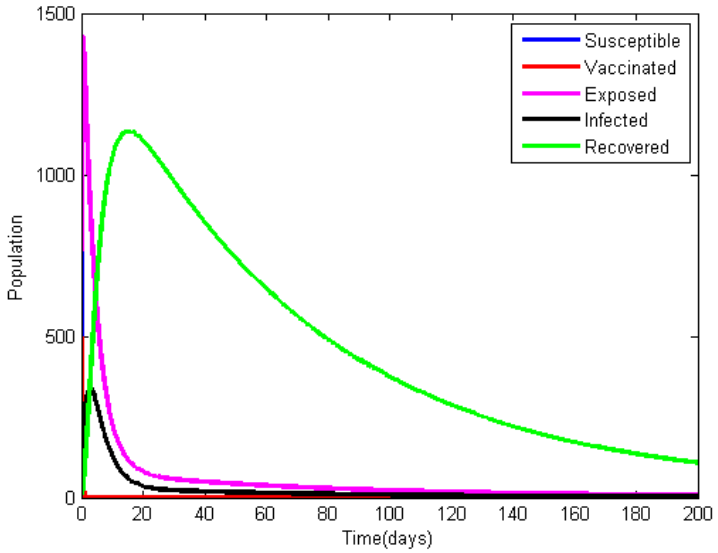

Figure 3: Dynamics of COVID-19 Transmission and Progression with AstraZeneca Vaccination

Initially, the susceptible population is high at 1000 but decreases over time as individuals transition to exposed and infected states. The exposed population rises, leading to a spike in infections before preventive measures like vaccination take effect. As vaccination progresses, with an efficacy of 0.88, new infections slow down and the vaccinated population grows. This helps reduce overall disease spread. Meanwhile, the number of recovered individuals increases, reflecting successful treatment and immunity development. This findings aligns with the outcomes of the study conducted by Ullah and Khan [66].

4.2.2. Incidences of COVID-19 Infection with and without AstraZeneca Vaccination

This simulation assesses the rate of COVID-19 infections among individuals, emphasizing the differences in infection rates between those who received the AstraZeneca vaccine and those who did not. Figure 4 shows the outcomes.

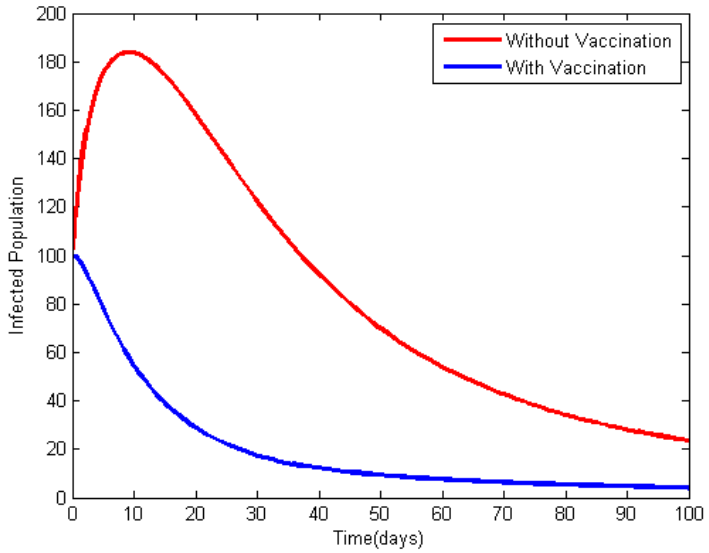

Figure 4: Incidences of HPV-infected population with vaccination and without vaccination

As depicted in the graph, without the AstraZeneca vaccine, COVID-19 infection rates rise significantly, highlighting the high susceptibility of individuals to the virus. In contrast, when the vaccine is administered, infection rates decrease markedly, indicating the vaccine's effectiveness in reducing COVID-19 transmission. This reduction in infections contributes to a lower overall burden of COVID-19 in the population. The graph underscores the importance of the AstraZeneca vaccine in controlling the spread of COVID-19 and supports the implementation of vaccination programs as a critical public health measure. These observations align with findings from recent studies, including those by Angeli et al. [62], Bandekar et al. [63] and Angelopoulou and Mykoniatis [61].

4.3. Impact of Varying Parameter Values

This section involves simulating how changes in parameter values affect the dynamics of COVID-19 within the model. The focus is on analyzing the impact of key parameters, particularly those with the most significant influence on the outcomes.

4.3.1. Effect of Vaccination Rates on Infected Population

The analysis examines how different vaccination rates (θ) affect the number of COVID-19 cases. This explores how varying levels of vaccination coverage influence the prevalence of infection within the population.

Figure 5 illustrates that increasing the vaccination rate (θ) leads to a significant reduction in the number of infected individuals, thus decreasing the overall burden of COVID-19. Conversely, lower vaccination rates result in higher infection rates. Additionally, higher vaccination rates are associated with shorter recovery times, while lower rates lead to prolonged illness.

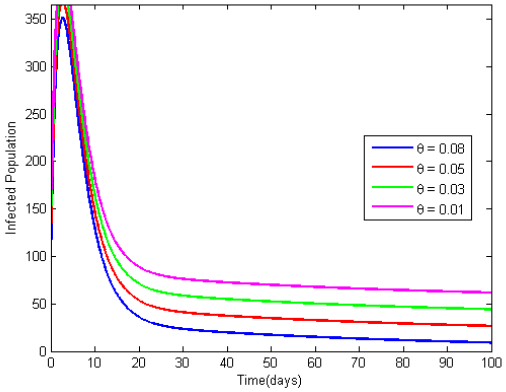

Figure 5: Incidences of COVID-19-infected population with different vaccination rates

4.3.2. Impact of Vaccination Efficacy on Infected Population

This simulation investigates how varying the efficacy of the AstraZeneca vaccine (γ) affects COVID-19 infection rates. Changes in vaccine efficacy directly impact the vaccine's ability to prevent transmission and reduce infection prevalence.

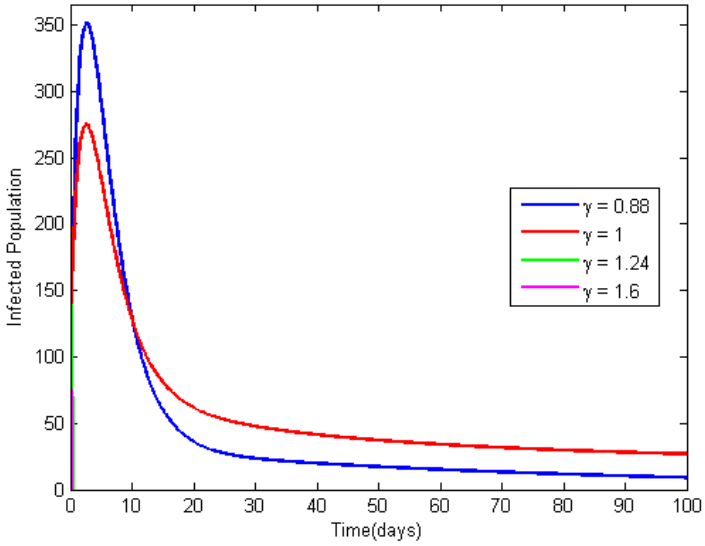

Figure 6: Incidences of COVID-19-infected population with varying vaccine efficacy rates

Figure 6 shows that when vaccine efficacy is set at 0.88, there is a modest decrease in infection rates. However, as efficacy increases from 0.88 to 1.24 and further to 1.6, the number of infections declines significantly. This decrease indicates that higher vaccine efficacy offers better protection against COVID-19, highlighting the importance of enhancing

vaccine effectiveness to control the spread of the virus.

5. Conclusion

This study develops a deterministic model for the AstraZeneca vaccine to examine its efficacy in reducing the transmission and progression of COVID-19 in the population. The model's fundamental properties, including the existence, positivity, and boundedness of solutions, were discussed. The stability of the model, including the disease-free equilibrium (DFE) and its stability properties, was also examined. The study found that the DFE is locally asymptotically stable when $R_e < 1$, indicating that the infection will die out over time if the basic reproduction number is less than one. Furthermore, the DFE is globally asymptotically stable under the same condition, suggesting that the disease will be eradicated regardless of the initial infection levels if $R_e < 1$.

In contrast, when $R_e > 1$, the model predicts the existence of an endemic equilibrium (EE), where the infection persists in the population. The global stability of the endemic equilibrium was analyzed and found to be globally asymptotically stable when $R_e > 1$. This implies that the infection will persist at a constant level in the population if the effective reproduction number exceeds one, regardless of initial conditions.

The findings from sensitivity analysis highlighted the human recruitment rate (Λ) and the effective humans contact rate (β) as the most positively sensitive parameters, indicating their critical role in influencing the spread of COVID-19. Conversely, the natural death rate (μ) from COVID-19 infections was identified as the parameter with the most negative sensitivity, suggesting that increasing the natural death rate can significantly reduce disease prevalence. MATLAB software was utilized for model simulation analysis, and the results indicated that effective implementation of vaccination strategies with high vaccine efficacy could potentially mitigate the spread of COVID-19 and reduce the incidence of severe disease. These findings provide critical insights into the role of vaccination in controlling COVID-19 transmission and highlight the importance of achieving and maintaining an effective reproduction number less than one to ensure the long-term eradication of the virus.

However, this study has limitations. The model assumes homogeneity in the population and does not account for variations in behavior, genetic factors, or differences in healthcare access. Additionally, the model's parameters were estimated based on available data, which may not fully capture the complexities of COVID-19 transmission dynamics in different regions. Future research should incorporate more detailed population data and consider heterogeneous mixing patterns to enhance the model's accuracy and applicability.

For future studies, incorporating heterogeneous mixing patterns and evaluating the impact of genetic variability on COVID-19 transmission dynamics should be considered. Additionally, exploring the cost-effectiveness of combined interventions and validating model predictions using empirical data would be beneficial.

Acknowledgment

We extend heartfelt appreciation to all those who made significant contributions to the successful completion of this project.

Data Availability

All data generated in this manuscript are included in the list of references.

Conflicts of Interest

The authors declare that they have no conflicts of interest associated with the publication of this paper.

Declaration of competing interest

Authors do not have any conflict of interest to disclose.

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