

A Transmission Dynamics Model of COVID-19 With Consideration of the Vulnerability of a Population

Patricia Adjeley Laryea¹, F. T. Oduro², Christopher Saaha Bornaa³, Samuel Okyere⁴

¹ Department of Mathematics, C. K. Tedom University of Technology and Applied Sciences, Navrongo, Ghana

^{2,4} Department of Mathematics, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana

³ Department of Mathematics & ICT Education, C. K. Tedom University of Technology and Applied Sciences, Navrongo, Ghana, ORCID: <https://orcid.org/0000-0001-8730-1970>

Correspondence: F. T. Oduro, Department of Mathematics, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana. E-mail: ftoduro@gmail.com

Received: June 4, 2023 Accepted: September 30, 2023 Online Published: October 16, 2023

doi:10.5539/jmr.v15n5p47

URL: <https://doi.org/10.5539/jmr.v15n5p47>

Abstract

A well documented characteristic of COVID-19 is that whereas certain infected individuals recover without ever showing symptoms, others regarded as vulnerable, usually age with comorbidities tend to succumb to more or less severe symptoms. To address pertinent issues, we formulate an $SEI_A I_S RS$ Transmission Dynamics model of COVID-19 where I_A and I_S respectively represent asymptomatic and symptomatic classes thus allowing the inclusion of parameters which are vulnerability sensitive. We define a vulnerability factor, ϕ and show that the model is globally asymptotically stable at the disease-free equilibrium when $\mathcal{R}_0 < 1$ and ϕ is appropriately bounded above. We also show that the model is globally asymptotically stable at the endemic equilibrium when $\mathcal{R}_0 > 1$ and ϕ is appropriately bounded below. Finally, we employ numerical analysis using Ghana data, to further illustrate the effect of vulnerability related parameter values on the trajectories of key variables of the model. We thereby demonstrated that if a dominantly young population is of sufficiently low vulnerability then $\mathcal{R}_0 < 1$, and the Transmission Dynamics exhibits global asymptotic stability at the disease-free equilibrium.

Keywords: epidemiology, vulnerability, transmission dynamics, COVID-19

1. Introduction

A highly infectious disease known as Coronavirus disease 2019 (Covid-19) and caused apparently by a novel virus strain, originated in late November 2019, from Wuhan China. This virulent disease has spread rapidly and globally reaching virtually all countries and resulting in a pandemic (Gorbalenya, 2020).

Consequently, disease has, in many ways, adversely affected the world: it has extensively disrupted socioeconomic conditions, overwhelmed healthcare system capacities, and causing significant numbers of morbidities and deaths. The commonest symptoms of disease as presented in most of its variants are: fever, cough, fatigue, severe respiratory illness, just to mention a few (Huang, 2020). The virus is propagated directly via contact with respiratory droplets emanating from an infected individual or indirectly through the touching of surfaces contaminated with the virus (Riou and Althaus, 2020; Qun et al., 2020). Currently due to its novelty, there is no known cure for Covid-19. Clinical case management currently focuses on reducing disease symptoms to help support the immune system of the infected person in the fight against the virus.

Nevertheless, with the rapid and advancing development of vaccines and antivirals, treatment of the disease has been constantly improving. Several preventive and nonpharmaceutical measures have also been leveraged to help curtail the rapid spread of the disease such as social distancing requirements, the mandatory use of face masks, personal hygiene promotion through frequent washing of hands together with the use of hand sanitizers (Asamoah et al., 2020). These extraordinary control measures have been in place to combat all the variants of Covid-19 that have emerged on the course of time. In spite of strenuous efforts which have been made to combat the disease, Covid-19 remains endemic to date in many areas of the globe.

In past decades mathematical models (Yavuz and Haydar, 2022; Meyer and Lima, 2022; Olumuyiwa et al., 2021) have been employed to assess the rate and extent of spread and also prescribe effective control of infectious diseases involving

different pathogens. These models provide decision supports to clinicians and other professionals in fields such as public health policy making and emergency response planning. Others include health risk assessment and management, promotion and social marketing of health related issues and the controlling of related hazards (Al-Sheikh, 2013; Asamoah et al., 2021). Kermack and Mckendrick in 1927 successfully developed the Susceptible, Infected, Recovered (SIR) compartmental model to be used to mathematically model infectious epidemic diseases (Kermack and Mckendrick, 1927). They later introduced another compartmental class: Exposed (denoted E) to enhance the SIR modeling 1932 thereby obtaining the SEIR model. This was further elaborated by incorporating birth and death rates. Several studies can be cited which have used these mathematical models to investigate infectious diseases such as tuberculosis (Bowong and Kurths, 2010; Bowong and Jules, 2009), HIV/AIDS (Mukandavire et al., 2009), measles vaccination (Bauch et al., 2009; Widyaningsih et al., 2018), pertusis epidemiology (Pesco et al., 2014) and more recently COVID-19 (Iboi et al., 2020).

A distinct feature of the COVID-19 pandemic that has been observed all over the world is that persons with low immunity (Ega and Ngeleja, 2022) and/ or who are affected by Co-morbidities such as diabetes (Okyere and Ackora-Prah, 2022) and cardio-vascular diseases tend to be more vulnerable to severe infection than those without these conditions. The said conditions are strongly correlated with ageing: hence, it has been observed that severe Covid infection affects the aged (65+) far more than the young. Unfortunately, such vulnerability related issues have not been adequately dealt with in terms of transmission dynamics modeling and subsequent control implementation. The result has been the rather less than efficient public health interventions, both at the pharmaceutical (treatments and vaccines) and nonpharmaceutical (masks and quarantine) levels to combat the pandemic in many parts of the world.

In this paper, we attempt to address aspects of the forementioned problems by developing a transmission dynamics model of Covid-19 which allows the incorporation of vulnerability dependent parameters with a view to investigating the effect of vulnerability on the long term stability or persistence of the disease. The proposed model is developed in the next section, the theoretical analysis is presented in Section 3. Numerical simulations performed in Section 4 to support the theoretical results and the conclusion is presented in Section 5.

2. Formulation of the Model

2.1 Model Description

Everywhere the propagation of covid-19 has been characterised as having high transitivity and that susceptible (S) individuals, once exposed (E) quickly get infected with the virus but then subsequently become infectious and asymptomatic before possibly attaining an infectious and symptomatic status. We may thus regard individuals who though infectious, never exhibit symptoms as nonvulnerable to the disease. We therefore categorize individuals as being vulnerable if they become infectious and symptomatic almost immediately after being exposed.

The model we propose is thus simply a modification of the standard SEIR model in which the compartment of infectious individuals is split into two: namely, the asymptomatic infectious (I_A) and symptomatic infectious (I_S). In such an arrangement we are clearly able to identify parameters which depend on vulnerability.

The model of the total population at any time (t) is divided into five sub-population (compartments) with respect to disease status in the system.

The total population is represented by N and divided into sub-populations of Susceptible individuals (S), Exposed individuals (E), Infected asymptomatic individuals (I_A), Infected symptomatic individuals (I_S), and Recovered individuals (R). The total population at time t is given by:

$$N(t) = S(t) + E(t) + I_A(t) + I_S(t) + R(t). \tag{2.1.1}$$

Figure 1 shows the compartmental flow chart of the COVID-19 Model

Susceptible individuals, $S(t)$, include those that are at risk of being infected with COVID-19. Exposed individuals, $E(t)$, include those that are infected but not infectious (latent) and are within the environment of the disease (COVID-19). The infected asymptomatic compartment, $I_A(t)$, consists of individuals that have been infected and infectious but with no symptoms of the disease (COVID-19). The infected symptomatic compartment, $I_S(t)$, consists of those that have infection, are infectious and are showing symptoms of the disease (COVID-19). The recovered individuals, R , are those who have recovered from the COVID-19 disease with no permanent immunity.

The Susceptible group of individuals are recruited into the population at a rate Λ and acquire COVID-19 through droplets or direct contact of infected surfaces at the rate β . This class is reduced whenever the individuals are initially infected with the disease or die naturally. Those who recover from the infection at the rate ξ are with no permanent immunity and

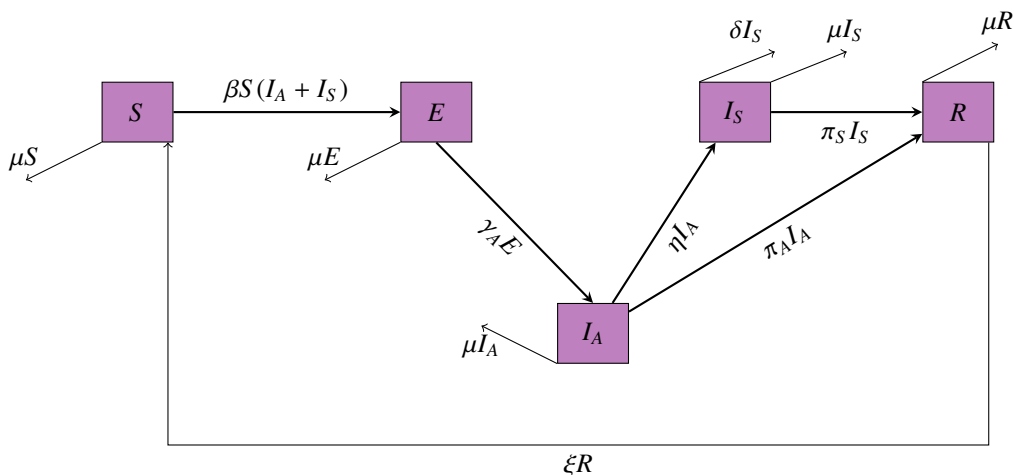


Figure 1. Flow Chart of COVID-19 Model

join the susceptible class at ξR . Thus

$$\frac{dS}{dt} = \Lambda + \xi R - \beta S(I_A + I_S) - \mu S$$

Contact with infectious surfaces and individuals, $(I_A + I_S)S \beta$, make the individuals exposed and therefore are moved from the susceptible class to the exposed compartment with a natural death rate μ . When the viral load increases the individuals become infectious, $\gamma_A E$, but show no symptoms and are therefore moved to the asymptomatic class. Thus

$$\frac{dE}{dt} = \beta S(I_A + I_S) - (\gamma_A + \mu) E$$

The infectious asymptomatic individuals, $\gamma_A E$, become symptomatic infectious at a rate η with natural death rate μ . This class of individuals can overcome the disease and recover at the rate π_A . Thus

$$\frac{dI_A}{dt} = \gamma_A E - (\eta + \pi_A + \mu) I_A$$

The infectious symptomatic individuals are recruited from the asymptomatic infectious class who become symptomatic infectious ηI_A and die due to the infection at a rate δ with natural death rate as μ . The class of individuals can also overcome the disease and recover at the rate π_S . Thus

$$\frac{dI_S}{dt} = \eta I_A - (\pi_S + \delta + \mu) I_S$$

The recovered class recruits from the asymptomatic infectious and symptomatic infectious classes at the rate π_A and π_S respectively as $\pi_A I_A + \pi_S I_S$ with natural death rate μ and they join the susceptible class at ξ . Thus

$$\frac{dR}{dt} = \pi_A I_A + \pi_S I_S - (\xi + \mu) R$$

The following system of nonlinear ordinary differential equations are therefore obtained as the Model equations:

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda + \xi R - \beta S(I_A + I_S) - \mu S \\ \frac{dE}{dt} &= \beta S(I_A + I_S) - (\gamma_A + \mu) E \\ \frac{dI_A}{dt} &= \gamma_A E - (\eta + \pi_A + \mu) I_A \\ \frac{dI_S}{dt} &= \eta I_A - (\pi_S + \delta + \mu) I_S \\ \frac{dR}{dt} &= \pi_A I_A + \pi_S I_S - (\xi + \mu) R \end{aligned} \right\} \tag{2.1.2}$$

Where

$a_1 = \gamma_A + \mu$, $a_2 = \eta + \pi_A + \mu$, $a_3 = \pi_S + \delta + \mu$, $a_4 = \xi + \mu$,
 then the model is transformed into;

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda + \xi R - \beta S(I_A + I_S) - \mu S \\ \frac{dE}{dt} &= \beta S(I_A + I_S) - a_1 E \\ \frac{dI_A}{dt} &= \gamma_A E - a_2 I_A \\ \frac{dI_S}{dt} &= \eta I_A - a_3 I_S \\ \frac{dR}{dt} &= \pi_A I_A + \pi_S I_S - a_4 R \end{aligned} \right\} \tag{2.1.3}$$

Table 1. Definition of variables and parameters of the $SEI_A I_S RS$ model

Variable/Parameter	Definition
S	Susceptible individuals
E	Exposed individuals
I_A	Infected asymptomatic individuals
I_S	Infected symptomatic individuals
R	Recovered individuals
Λ	Rate of recritment into the susceptible class
β	Transmission probability
η	Rate at which the asymptomatic become symptomatic
γ_A	Rate at which exposed become asymptomatic
π_A	Rate at which the asymptomatic recover
π_S	Rate at which the symptomatic recover
δ	Disease - induced death rate
μ	Natural Death rate
ξ	Rate at which recovered become susceptible due to loss of immunity

with nonnegative initial conditions $S(0) \geq 0$, $E(0) \geq 0$, $I_A(0) \geq 0$, $I_S(0) \geq 0$, $R(0) \geq 0$ and $N > 0$. It is assumed that all the parameters are nonnegative.

Clearly, the parameters γ_A and η are vulnerability dependent. So also are the parameters π_S and δ albeit in an a posteriori sense.

Now, let p_S be the probability that a vulnerable individual goes through the path $S - E - I_A - I_S - R$ as given in the flowchart of figure1. Then $p_S = \left(\frac{\beta}{a_1}\right)\left(\frac{\gamma_A}{a_2}\right)\left(\frac{\eta}{a_3}\right)$.

Similarly, let p_A be the probability that a less vulnerable individual goes through the path $S - E - I_A - R$ as given in the flowchart of figure1. Then $p_A = \left(\frac{\beta}{a_1}\right)\left(\frac{\gamma_A}{a_2}\right)$.

Hence the total probability is given by

$$p_S + p_A = \left(\frac{\beta}{a_1}\right)\left(\frac{\gamma_A}{a_2}\right)\left(\frac{\eta}{a_3}\right) + \left(\frac{\beta}{a_1}\right)\left(\frac{\gamma_A}{a_2}\right) \tag{2.1.4}$$

$$\begin{aligned} &= \left(\frac{\beta}{a_1}\right)\left(\frac{\gamma_A}{a_2}\right)\left(\left(\frac{\eta}{a_3}\right) + 1\right) \\ &= \left(\frac{\beta}{a_1}\right)\left(\frac{\gamma_A}{a_2}\right)\left(\frac{\eta + a_3}{a_3}\right) \end{aligned} \tag{2.1.5}$$

From the viewpoint of risk theory which requires that risk or the proximity of a hazard is the product of exposure and vulnerability, it becomes natural here to define a vulnerability factor ϕ as $\phi = \left(\frac{\gamma_A}{a_2}\right)\left(\frac{\eta+a_3}{a_3}\right)$ An exposure factor, ϵ , is now clearly defined as $\epsilon = \left(\frac{\beta}{a_1}\right)$.

Comparing the above equation 2.1.5 with equation 3.3.27, we notice \mathcal{R}_0 is related to the exposure and vulnerability factors ϵ and ϕ . Intact we have that $\mathcal{R}_0 = S \epsilon \phi$

Thus expressed this way, \mathcal{R}_0 may be regarded as a measure of the risk posed by the epidemic.

3. Qualitative Properties of the Model

3.1 Positivity and Boundedness of Solutions

We want to find non-negative answers in this part. Therefore, it is crucial to understand the circumstances in which the studied system of differential equations has non-negative solutions. If all solutions have non-negative initial data and remain non-negative throughout, the COVID-19 model would be epidemically well posed.

Theorem 3.1: Given that at $t = 0, S(0) \geq 0, E(0) \geq 0, I_A(0) \geq 0, I_S(0) \geq 0, R(0) \geq 0$ then $\Pi = \{ (S(t), E(t), I_A(t), I_S(t), R(t)) \in \mathbb{R}_+^5 \}$ for all $t > 0$ (i.e. positively invariant) and is bounded.

Proof. The total population of the model at any time (t) is given by: $N(t) = S(t) + E(t) + I_A(t) + I_S(t) + R(t)$

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_A}{dt} + \frac{dI_S}{dt} + \frac{dR}{dt}$$

$$\frac{dN}{dt} = (-E - I_A - S - I_S - R)\mu + \Lambda - \delta I_S$$

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I_S$$

Absence of excess mortality gives:

$$\frac{dN}{dt} \leq \Lambda - \mu N \tag{3.1.6}$$

Integrating

$$\int dN \leq \int \Lambda - \mu N dt$$

$$\int \frac{dN}{\Lambda - \mu N} \leq \int dt$$

multiply both sides by $-\mu$

$$\int \frac{-\mu dN}{\Lambda - \mu N} \geq \int -\mu dt$$

$$\ln(\Lambda - \mu N) \geq -\mu t + q$$

$$(\Lambda - \mu N) \geq e^{-\mu t + q}$$

$$(\Lambda - \mu N) \geq Q e^{-\mu t} \tag{3.1.7}$$

Applying the initial condition

$$N(0) = N_0$$

We obtain the relation for

$$(\Lambda - \mu N_0) = Q$$

$$\Lambda - \mu N(t) \geq (\Lambda - \mu N_0)e^{-\mu t} \tag{3.1.8}$$

As $t \rightarrow \infty, e^{-\mu t} \rightarrow 0$

$$\Lambda - \mu N(t) \geq 0$$

$$-\Lambda + \mu N(t) \leq 0$$

$$\mu N(t) \leq \Lambda$$

$$N(t) \leq \frac{\Lambda}{\mu} \tag{3.1.9}$$

Since

$N(t)$

is monotonically increasing starting from the initial state

$N(0)$

it approaches the upperbound.

Thus

$N(t) > N(0)$

for

$t > 0$

but

$N(0) \geq 0$.

Therefore

$N(t)$

is bounded below by

0.

In conclusion

$$0 \leq N(t) \leq \frac{\Lambda}{\mu}. \tag{3.1.10}$$

Hence,

$$\Pi = \left\{ (S, E, I_A, I_S, R) \in \mathbb{R}_S^5 : S + E + I_A + I_S + R \leq \frac{\Lambda}{\mu} \right\} \tag{3.1.11}$$

Where Π is a positively invariant set and bounded within zero and $\frac{\Lambda}{\mu}$.

Consider:

$$\frac{dS}{dt} = \Lambda + \xi R - \beta S(I_A + I_S) - \mu S \tag{3.1.12}$$

$(I_A + I_S) \leq \kappa$ since the population is bounded

$$\frac{dS}{dt} \geq \Lambda + \xi R - \kappa\beta S - \mu S$$

$$R = (N - S - E - I_A - I_S)$$

$$\frac{dS}{dt} \geq \Lambda + \xi(N - S - E - I_A - I_S) - \kappa\beta S - \mu S$$

$(S + E + I_A + I_S) \leq \kappa$ since the population is bounded and $N(t)$ is bounded below by 0.

$$\frac{dS}{dt} \geq \Lambda - \xi\kappa - (\kappa\beta + \mu)S$$

Let $(\Lambda - \xi\kappa) = \Lambda'$ and $(\kappa\beta + \mu) = \mu'$

$$\frac{dS}{dt} \geq \Lambda' - \mu' S$$

integrating

$$\int dS \geq - \int (\Lambda' - \mu' S) dt$$

$$\int \frac{dS}{(\Lambda' - \mu' S)} \geq \int dt$$

Multiply both sides by $-\mu'$

$$\int \frac{-\mu' dS}{(\Lambda' - \mu' S)} \leq \int -\mu' dt$$

$$\ln(\Lambda' - \mu' S) \leq (-\mu')t + c$$

$$(\Lambda' - \mu' S) \leq C e^{(-\mu')t}$$

where

$$C = e^c$$

At the initial time, $t=0$ and substituting into the inequality

$$C = (\Lambda' - \mu')S(0)$$

Thus, the inequality becomes;

$$\Lambda' - \mu' S(t) \leq (\Lambda' - \mu')S(0)e^{(-\mu')t}$$

As $t \rightarrow \infty, e^{(-\mu')t} = 0$

$$-\mu' S(t) \leq -\Lambda'$$

$$\mu S(t) \geq \Lambda'$$

$$S(t) \geq \frac{\Lambda'}{\mu'} \tag{3.1.13}$$

Since $S(t)$ is monotonically decreasing function it starts from its initial state $S(0)$ and decreasing towards its lower bound. Thus we have $S(t) \leq S(0)$. Hence $S(0)$ is an upper bound of $S(t)$ for all $t > 0$. In conclusion we have $\frac{\Lambda}{\mu} \leq S(t) \leq S(0)$.

Similarly, positivity and boundedness can be shown for E, I_A, I_S and R

3.2 Determination of Equilibrium Points of the Model

3.2.1 Disease-free Equilibrium Point

The disease-free equilibrium, $\mathcal{E}^0 = (S, E, I_A, I_S, R)$, of the system of ordinary differential equations in (2.1.2) only exists when $E = I_A = I_S = R = 0$ and all other controls held constant. This is computed by setting the system of differential equations in 2.1.2 and the state variables $E = I_A = I_S = R = 0$. This is given as

$$\frac{dS}{dt} = \Lambda + \xi R - \beta S(I_A + I_S) - \mu S = 0$$

$$S = \frac{\Lambda}{\mu},$$

and all other state variables become zeros

Hence, the disease-free equilibrium is given by:

$$\mathcal{E}^0 = (S^0, E^0, I_A^0, I_S^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right) \tag{3.2.14}$$

3.2.2 Endemic Equilibrium Point

The endemic equilibrium point $\mathcal{E}^* = (S^*, E^*, I_A^*, I_S^*, R^*)$ is obtained by solving the system of equations 2.1.3 at a stationary point.

$$\Lambda + \xi R^* - \beta S^*(I_A^* + I_S^*) - \mu S^* = 0 \tag{3.2.15}$$

$$\beta S^*(I_A^* + I_S^*) - a_1 E^* = 0 \tag{3.2.16}$$

$$\gamma_A E^* - a_2 I_A^* = 0 \tag{3.2.17}$$

$$\eta I_A^* - a_3 I_S^* = 0 \tag{3.2.18}$$

$$\pi_A I_A^* + \pi_S I_S^* - a_4 R^* = 0 \tag{3.2.19}$$

Making E and I_S the subject of equations 3.2.17 and 3.2.18 we get

$$E^* = \frac{a_2 I_A^*}{\gamma_A} \tag{3.2.20}$$

$$I_S^* = \frac{\eta I_A^*}{a_3} \tag{3.2.21}$$

Substituting for E and I_S in equation 3.2.16

$$\beta S^* \left(I_A^* + \frac{\eta I_A^*}{a_3} \right) - \frac{a_1 a_2 I_A^*}{\gamma_A} = 0$$

$$\begin{aligned} \Rightarrow \beta S^* \left(1 + \frac{\eta}{a_3}\right) I_A^* - \frac{a_1 a_2 I_A^*}{\gamma_A} &= 0 \\ \Rightarrow \frac{\beta S^*}{a_3} (a_3 + \eta) I_A^* &= \frac{a_1 a_2 I_A^*}{\gamma_A} \\ \Rightarrow S^* &= \frac{a_1 a_2 a_3}{\beta \gamma_A (a_3 + \eta)} \end{aligned} \tag{3.2.22}$$

Substitute for I_S equation 3.2.21 in equation 3.2.19

$$\begin{aligned} \pi_A I_A^* + \pi_S \left(\frac{\eta I_A^*}{a_3}\right) - a_4 R^* &= 0 \\ \Rightarrow \left(\pi_A + \frac{\pi_S \eta}{a_3}\right) I_A^* &= a_4 R^* \\ \Rightarrow \frac{(\pi_A a_3 + \pi_S \eta) I_A^*}{a_3} &= a_4 R^* \\ R^* &= \frac{(\pi_A a_3 + \pi_S \eta) I_A^*}{a_3 a_4} \end{aligned} \tag{3.2.23}$$

Rearranging equation 3.2.15

$$\begin{aligned} \mu S^* - \Lambda &= \xi R^* - \beta S^* (I_A^* + I_S^*) \\ \Rightarrow \mu S^* - \Lambda &= \xi R^* - \beta S^* \frac{(a_3 + \eta) I_A^*}{a_3} \end{aligned}$$

Using equations 3.2.22 and 3.2.23 above, we now substitute S and R as follows

$$\begin{aligned} \mu \frac{a_1 a_2 a_3}{\beta \gamma_A (a_3 + \eta)} - \Lambda &= \xi \frac{(\pi_A a_3 + \pi_S \eta) I_A^*}{a_3 a_4} - \beta \frac{a_1 a_2 a_3}{\beta \gamma_A (a_3 + \eta)} \frac{(a_3 + \eta) I_A^*}{a_3} \\ \mu \frac{a_1 a_2 a_3}{\beta \gamma_A (a_3 + \eta)} - \Lambda &= \xi \frac{(\pi_A a_3 + \pi_S \eta) I_A^*}{a_3 a_4} - \frac{a_1 a_2}{\gamma_A} I_A^* \\ \mu \frac{a_1 a_2 a_3}{\beta \gamma_A (a_3 + \eta)} - \Lambda &= \left[\xi \frac{(\pi_A a_3 + \pi_S \eta)}{a_3 a_4} - \frac{a_1 a_2}{\gamma_A} \right] I_A^* \end{aligned}$$

Finding the L.C.M

$$\begin{aligned} \frac{\mu a_1 a_2 a_3 - \Lambda \beta \gamma_A (a_3 + \eta)}{\beta \gamma_A (a_3 + \eta)} &= \left[\frac{\xi \gamma_A (\pi_A a_3 + \pi_S \eta) - a_1 a_2 a_3 a_4}{a_3 a_4 \gamma_A} \right] I_A^* \\ \Rightarrow I_A^* &= \frac{\mu a_1 a_2 a_3 - \Lambda \beta \gamma_A (a_3 + \eta)}{\beta \gamma_A (a_3 + \eta)} \times \frac{a_3 a_4 \gamma_A}{\xi \gamma_A (\pi_A a_3 + \pi_S \eta) - a_1 a_2 a_3 a_4} \\ \Rightarrow I_A^* &= \frac{[\mu a_1 a_2 a_3 - \Lambda \beta \gamma_A (a_3 + \eta)] a_3 a_4}{[\xi \gamma_A (\pi_A a_3 + \pi_S \eta) - a_1 a_2 a_3 a_4] \beta (a_3 + \eta)} \end{aligned} \tag{3.2.24}$$

Let

$$\Gamma = [\mu a_1 a_2 a_3 - \Lambda \beta \gamma_A (a_3 + \eta)] a_3 a_4 \text{ and } \Gamma_1 = [\xi \gamma_A (\pi_A a_3 + \pi_S \eta) - a_1 a_2 a_3 a_4] \beta (a_3 + \eta)$$

Hence

$$I_A^* = \frac{\Gamma}{\Gamma_1} \tag{3.2.25}$$

The endemic equilibrium point is given as $\mathcal{E}^* = (S^*, E^*, I_A^*, I_S^*, R^*)$ where;

$$S^* = \frac{a_1 a_2 a_3}{\beta \gamma_A (a_3 + \eta)},$$

$$E^* = \frac{a_2 \Gamma}{\gamma_A \Gamma_1},$$

$$I_A^* = \frac{\Gamma}{\Gamma_1},$$

$$I_S^* = \frac{\eta \Gamma}{a_3 \Gamma_1}, \text{ and}$$

$$R^* = \frac{(\pi_A a_3 + \pi_S \eta) \Gamma}{a_3 a_4 \Gamma_1}.$$

3.3 Basic Reproduction Number of the Model

The concepts of Next Generation Matrix is applied here to establish the stability of the disease-free equilibrium (\mathcal{E}^0). The basic reproduction number is computed.

Using the Next Generation Matrix (van den Driessche and Watmough, 2002), we consider only the infective classes in the system of differential equations as:

$$\left. \begin{aligned} \frac{dE}{dt} &= \beta S (I_A + I_S) - a_1 E \\ \frac{dI_A}{dt} &= \gamma_A E - a_2 I_A \\ \frac{dI_S}{dt} &= \eta I_A - a_3 I_S \end{aligned} \right\} \tag{3.3.26}$$

The corresponding Jacobian matrix at disease free equilibrium is given as:

$$J = \begin{bmatrix} -a_1 & \beta S & \beta S \\ \gamma_A & -a_2 & 0 \\ 0 & \eta & -a_3 \end{bmatrix}$$

Where F and V is represented by

$$F = \begin{bmatrix} 0 & \beta S & \beta S \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} -a_1 & 0 & 0 \\ \gamma_A & -a_2 & 0 \\ 0 & \eta & -a_3 \end{bmatrix}$$

Thus

$$V^{-1} = \begin{bmatrix} -a_1^{-1} & 0 & 0 \\ -\frac{\gamma_A}{a_1 a_2} & -a_2^{-1} & 0 \\ -\frac{\gamma_A \eta}{a_1 a_2 a_3} & -\frac{\eta}{a_2 a_3} & -a_3^{-1} \end{bmatrix}$$

such that:

$$FV^{-1} = \begin{bmatrix} 0 & \beta S & \beta S \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} -a_1^{-1} & 0 & 0 \\ -\frac{\gamma_A}{a_1 a_2} & -a_2^{-1} & 0 \\ -\frac{\gamma_A \eta}{a_1 a_2 a_3} & -\frac{\eta}{a_2 a_3} & -a_3^{-1} \end{bmatrix}$$

$$= \begin{bmatrix} \frac{\beta S \gamma_A}{a_1 a_2} + \frac{\beta S \gamma_A \eta}{a_1 a_2 a_3} & \frac{\beta S}{a_2} + \frac{\beta S \eta}{a_2 a_3} & \frac{\beta S}{a_3} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

The reproduction number is the largest spectral radius (ρ) of $|FV^{-1}|$ and this is obtained as:

$$\rho(FV^{-1}) = \frac{\beta S \gamma_A (\eta + a_3)}{a_1 a_2 a_3}$$

Hence the Basic Reproduction (\mathcal{R}_0) is given as:

$$\mathcal{R}_0 = \frac{\beta S \gamma_A (\eta + a_3)}{a_1 a_2 a_3} \tag{3.3.27}$$

3.4 Sensitivity Analysis of the Model

The goal of sensitivity analysis is to measure the impact of parameter changes on the behaviour of the model. This is done to give more attention to parameters that are observed to play a significant role in the model behaviour. However these parameter values are sometimes unavailable or are not accurately measured. Sensitivity analysis plays a role by informing researchers to begin to pay attention to the model parameter values and measuring them more accurately.

The forward normalized sensitivity index would be used to perform the analysis.

Definition: It is defined as follows: Let \mathcal{R}_0 be a function that depends on x_i and it is differentiable, then the normalized forward sensitivity index of \mathcal{R}_0 relative to x_i is given by

$$\Pi_{\mathcal{R}_0}^{x_i} = \frac{\partial \mathcal{R}_0}{\partial x_i} \times \frac{x_i}{\mathcal{R}_0} \tag{3.4.28}$$

where $\mathcal{R}_0 = \frac{\beta S \gamma_A (\eta + a_3)}{a_1 a_2 a_3}$

This index measures the relative change in \mathcal{R}_0 due to relative changes in x_i . It shows the significance of each parameter in determining the spread of the infection. For an example, the sensitivity index of \mathcal{R}_0 with respect to β is given as

$$\begin{aligned} \Pi_{\mathcal{R}_0}^\beta &= \frac{\partial \mathcal{R}_0}{\partial \beta} \times \frac{\beta}{\mathcal{R}_0} = 1(\text{positive}) \\ \Pi_{\mathcal{R}_0}^\mu &= -\mu \left(\frac{1}{\mu} + \frac{1}{\gamma_A + \mu} + \frac{1}{\pi_S + \delta + \mu} \right) (\text{negative}) \\ \Pi_{\mathcal{R}_0}^{\gamma_A} &= \frac{\partial \mathcal{R}_0}{\partial \gamma_A} \times \frac{\gamma_A}{\mathcal{R}_0} = \frac{\gamma_A}{\gamma_A + \mu} (\text{positive}) \\ \Pi_{\mathcal{R}_0}^{\pi_S} &= \frac{\partial \mathcal{R}_0}{\partial \pi_S} \times \frac{\pi_S}{\mathcal{R}_0} = -\frac{\pi_S}{\pi_S + \delta + \mu} (\text{negative}) \\ \Pi_{\mathcal{R}_0}^\delta &= \frac{\partial \mathcal{R}_0}{\partial \delta} \times \frac{\delta}{\mathcal{R}_0} = -\frac{\delta}{\pi_S + \delta + \mu} (\text{negative}) \\ \Pi_{\mathcal{R}_0}^\Lambda &= \frac{\partial \mathcal{R}_0}{\partial \Lambda} \times \frac{\Lambda}{\mathcal{R}_0} = 1(\text{positive}) \\ \Pi_{\mathcal{R}_0}^{\pi_A} &= \frac{\partial \mathcal{R}_0}{\partial \pi_A} \times \frac{\pi_A}{\mathcal{R}_0} = -\frac{\pi_A}{\eta + \pi_A + \mu} (\text{negative}) \\ \Pi_{\mathcal{R}_0}^\eta &= \frac{\partial \mathcal{R}_0}{\partial \eta} \times \frac{\eta}{\mathcal{R}_0} = -\frac{\eta}{\eta + \pi_A + \mu} (\text{negative}) \end{aligned}$$

These expressions are evaluated with the values of the parameter that constitute \mathcal{R}_0 .

If the sensitivity index which is given by, $\Pi_{\mathcal{R}_0}^{x_i} = \frac{\partial \mathcal{R}_0}{\partial x_i} \times \frac{x_i}{\mathcal{R}_0}$, of a parameter is negative then a decrease (increase) in the value of the parameter will cause a decrease (increase) in \mathcal{R}_0 . However, if the sensitivity index is positive then an increase (decrease) in the value of the parameter will cause an increase (decrease) in \mathcal{R}_0 . The reproduction number, \mathcal{R}_0 , measures the average number of new infections caused by an infected individual in a population. Therefore an increase in the reproduction number will be detrimental to the survival of the population. Increases in β , γ_A , and Λ will lead to an increase in \mathcal{R}_0 and hence an increase in the transmission of the virus and π_S , δ , η , μ , and π_A have an inverse relation with \mathcal{R}_0 .

3.5 Stability Analysis of the Model

3.5.1 Local Stability Analysis

The stability analysis can be performed by considering the eigenvalues of the Jacobian matrix evaluated at a particular equilibrium point. Here we shall focus on the disease-free equilibrium point.

The relevant Jacobian matrix of model (2.1.3) is given by:

$$J = \begin{bmatrix} -\beta (I_A + I_S) - \mu & 0 & -\beta S & -\beta S & \xi \\ \beta (I_A + I_S) & -a_1 & \beta S & \beta S & 0 \\ 0 & \gamma_A & -a_2 & 0 & 0 \\ 0 & 0 & \eta & -a_3 & 0 \\ 0 & 0 & \eta & \pi_S & -a_4 \end{bmatrix} \tag{3.5.29}$$

The Jacobian matrix at the disease-free equilibrium point is written as:

$$J_{(DFE)} = \begin{bmatrix} -\mu & 0 & -\beta S & -\beta S & \xi \\ 0 & -a_1 & \beta S & \beta S & 0 \\ 0 & \gamma_A & -a_2 & 0 & 0 \\ 0 & 0 & \eta & -a_3 & 0 \\ 0 & 0 & \eta & \pi_S & -a_4 \end{bmatrix} \tag{3.5.30}$$

The characteristic equation of (3.5.30) is given by

$$|J_{(DFE)} - \lambda I| = (-\mu - \lambda)(-a_4 - \lambda) \begin{vmatrix} -a_1 - \lambda & \beta S & \beta S \\ \gamma_A & -a_2 - \lambda & 0 \\ 0 & \eta & -a_3 - \lambda \end{vmatrix} = 0 \tag{3.5.31}$$

Obviously $(-\mu)$ and $(-a_4)$ are negative terms and the stability of the model depends on

$$|J'_{(DFE)} - \lambda I| = \begin{vmatrix} -a_1 - \lambda & \beta S & \beta S \\ \gamma_A & -a_2 - \lambda & 0 \\ 0 & \eta & -a_3 - \lambda \end{vmatrix} = 0 \tag{3.5.32}$$

where $J'_{(DFE)}$ is the reduced form of $J_{(DFE)}$.

Simplifying and collecting terms of the above (3.5.32) we have

$$\sum_{n=0}^3 m_n \lambda^n = 0 \tag{3.5.33}$$

where

$$m_3 = 1,$$

$$m_2 = a_3 + a_2 + a_1,$$

$$m_1 = (a_2 + a_1) a_3 + a_1 a_2 - \beta S \gamma_A,$$

$$m_0 = a_1 a_2 a_3 - \beta S \gamma_A (\eta + a_3).$$

Theorem 3.2: (Routh-Hurwitz Criterion for third order polynomial) The polynomial $m_3 \lambda^3 + m_2 \lambda^2 + m_1 \lambda + m_0$ has all roots in the open left half of the complex plane if and only if $m_2 > 0$, $m_2 m_1 - m_0 m_3 > 0$ and $m_0(m_2 m_1 - m_0 m_3) > 0$ See Mahardika et al. (2019).

Theorem 3.3: The disease-free equilibrium point of the model is locally asymptotically stable whenever $\mathcal{R}_0 \leq 1$, otherwise it is unstable.

Proof. The expression $m_2 = a_3 + a_2 + a_1$, is obviously a positive term.

The expression $m_2 m_1 - m_0 m_3 = (a_3 + a_2 + a_1) ((a_2 + a_1) a_3 + a_1 a_2 - \beta S \gamma_A) - a_1 a_2 a_3 + \beta S \gamma_A (\eta + a_3)$. $m_2 m_1 - m_0 m_3 = (a_3 + a_2 + a_1) ((a_2 + a_1) a_3 + a_1 a_2 - \frac{\mathcal{R}_0 a_1 a_2 a_3}{\eta + a_3}) - a_1 a_2 a_3 + \mathcal{R}_0 a_1 a_2 a_3$ where

$$\beta S = \frac{\mathcal{R}_0 a_1 a_2 a_3}{\gamma_A (\eta + a_3)}$$

Since

$$\frac{\mathcal{R}_0}{\eta + a_3} < \frac{\mathcal{R}_0}{a_3}$$

we have now

$$m_2 m_1 - m_0 m_3 > (a_3 + a_2 + a_1) a_1 a_2 a_3 \left(\frac{1}{a_1} + \frac{1}{a_2} \right) + (a_3 + a_2 + a_1) a_1 a_2 a_3 \left(\frac{1}{a_3} - \frac{\mathcal{R}_0}{a_3} \right) - a_1 a_2 a_3 (1 - \mathcal{R}_0)$$

$$m_2 m_1 - m_0 m_3 = (a_3 + a_2 + a_1) a_1 a_2 a_3 \left(\frac{1}{a_1} + \frac{1}{a_2} \right) + (a_3 + a_2 + a_1) a_1 a_2 a_3 \left(\frac{1 - \mathcal{R}_0}{a_3} \right) - a_1 a_2 a_3 (1 - \mathcal{R}_0)$$

$$m_2 m_1 - m_0 m_3 = (a_3 + a_2 + a_1) a_1 a_2 a_3 \left(\frac{1}{a_1} + \frac{1}{a_2} \right) + a_1 a_2 [(a_3 + a_2 + a_1) (1 - \mathcal{R}_0) - a_3 (1 - \mathcal{R}_0)]$$

$$m_2 m_1 - m_0 m_3 = (a_3 + a_2 + a_1) a_1 a_2 a_3 \left(\frac{1}{a_1} + \frac{1}{a_2} \right) + a_1 a_2 [(a_2 + a_1) (1 - \mathcal{R}_0) + a_3 (1 - \mathcal{R}_0) - a_3 (1 - \mathcal{R}_0)]$$

$$m_2 m_1 - m_0 m_3 = (a_3 + a_2 + a_1) a_1 a_2 a_3 \left(\frac{1}{a_1} + \frac{1}{a_2} \right) + a_1 a_2 (a_2 + a_1) (1 - \mathcal{R}_0)$$

$$m_2 m_1 - m_0 m_3 = (a_3 + a_2 + a_1) a_3 (a_1 + a_2) + a_1 a_2 (a_2 + a_1) (1 - \mathcal{R}_0) \text{ Thus } m_2 m_1 - m_0 m_3 > 0 \text{ whenever } \mathcal{R}_0 \leq 1.$$

Again the expression $(m_2 m_1 - m_0 m_3) m_0$. By the properties of real numbers, If

$$m_2 m_1 - m_3 m_0 > 0$$

and

$$m_0 > 0,$$

then

$$(m_2 m_1 - m_3 m_0) m_0 > 0.$$

It has been shown above that

$$m_2 m_1 - m_3 m_0 > 0 \text{ is positive whenever } \mathcal{R}_0 \leq 1.$$

Now it is thus to be establish that

$$m_0 = a_1 a_2 a_3 - \beta S \gamma_A (a_3 + \eta).$$

Substituting

$$\beta S = \frac{\mathcal{R}_0 a_1 a_2 a_3}{\gamma_A (\eta + a_3)}$$

into m_0 gives

$$m_0 = a_1 a_2 a_3 - \mathcal{R}_0 a_1 a_2 a_3$$

$m_0 = (1 - \mathcal{R}_0) a_1 a_2 a_3$ is positive when $\mathcal{R}_0 < 1$ Thus $(m_2 m_1 - m_3 m_0) m_0$ is also positive whenever $\mathcal{R}_0 \leq 1$ The system is locally asymptotically stable at the disease free equilibrium point whenever $\phi < \frac{\mu a_1}{\Lambda \beta}$. *Proof.* $\mathcal{R}_0 < 1$ then $S \epsilon \phi < 1$

$$\Rightarrow \phi < \frac{1}{S \epsilon}$$

$$S = \frac{\Lambda}{\mu}, \epsilon = \frac{\beta}{a_1}$$

$$\phi < \frac{\mu a_1}{\Lambda \beta}.$$

3.5.2 Global Stability of the Disease-free Equilibrium

In the case of Local stability there exist a neighbourhood of the equilibrium point within which the system is stable. This neighbourhood is called the basin of attraction. If the basin of attraction is the entire space on which the model is valid i.e. \mathbb{R}_+^5 then the system is said to be globally stable. This require that the condition of the LaSalle-Lyapunov must hold.

As a result, we can draw the following conclusion concerning the stability of the disease-free equilibrium globally.

Theorem 3.4: The DFE is globally asymptotically stable in \square , the nonnegative orthant containing \mathbb{R}_+^5 , where $\mathcal{R}_0 \leq 1$.

Proof. Considering the candidate LaSalle-Lyapunov function we have:

$$L_{DFE}(t) = AE + BI_A + CI_S + DR \tag{3.5.34}$$

such that the variables A,B,C, and D are all nonnegative constants. It fulfills using its time derivative along the trajectories 2.1.2

$$\begin{aligned} \dot{L}_{DFE}(t) &= A\dot{E} + B\dot{I}_A + C\dot{I}_S + D\dot{R} \\ &= A(\beta S (I_A + I_S) - (\gamma_A + \mu)E), \\ &\quad + B(\gamma_A E - (\eta + \pi_A + \mu)I_A), \\ &\quad + C(\eta I_A - (\pi_S + \delta + \mu)I_S), \\ &\quad + D(\pi_A I_A + \pi_S I_S - (\xi + \mu)R) \\ &= (-A((\gamma_A + \mu) + B\gamma_A)E \\ &\quad + (A\beta S - B(\eta + \pi_A + \mu) + C\eta + D\pi_A)I_A \\ &\quad + (A\beta S - C(\pi_S + \delta + \mu) + D\pi_S)I_S \end{aligned}$$

$$-(D(\xi + \mu))R.$$

The coefficients of $E, I_A, I_S,$ and R are set to zero using the constants A, B, C and D . This is

$$\begin{aligned} &(-A(\gamma_A + \mu) + B\gamma_A)E \\ &-A(\gamma_A + \mu) + B\gamma_A = 0 \\ \Rightarrow B &= \frac{A(\gamma_A + \mu)}{\gamma_A} \end{aligned} \tag{3.5.35}$$

Now substitute B into the coefficient of I_A

$$\Rightarrow \left(A\beta S + A \left(\frac{\gamma_A + \mu}{\gamma_A} \right) (\eta + \pi_A + \mu) \right) I_A$$

The equation coefficients of C are equal

$$\begin{aligned} \Rightarrow A \left(\frac{\gamma_A + \mu}{\gamma_A} \right) (\eta + \pi_A + \mu) + C\eta &= -C(\pi_S + \delta + \mu) \\ \Rightarrow A \left(\frac{\gamma_A + \mu}{\gamma_A} \right) (\eta + \pi_A + \mu) + C\eta + C(\pi_S + \delta + \mu) &= 0 \\ \Rightarrow A \left(\frac{\gamma_A + \mu}{\gamma_A} \right) (\eta + \pi_A + \mu) + C(\eta + \pi_S + \delta + \mu) &= 0 \\ \Rightarrow C &= \frac{A \left(\frac{\gamma_A + \mu}{\gamma_A} \right) (\eta + \pi_A + \mu)}{(\eta + \pi_S + \delta + \mu)} \end{aligned} \tag{3.5.36}$$

Coefficients of I_S is same as I_A

$$\Rightarrow (I_A + I_S) \left(A\beta S^* + \left(\frac{A \left(\frac{\gamma_A + \mu}{\gamma_A} \right) (\eta + \pi_A + \mu)}{(\eta + \pi_S + \delta + \mu)} \right) (\pi_S + \delta + \mu) \right)$$

But

$$\beta S^* = \frac{\mathcal{R}_0 a_1 a_2 a_3}{\gamma_A (\eta + a_3)}$$

Then

$$\dot{L}_{DFE} = (I_A + I_S) \left(A \left(\frac{\mathcal{R}_0 a_1 a_2 a_3}{\gamma_A (\eta + a_3)} \right) - \left(\frac{A \left(\frac{\gamma_A + \mu}{\gamma_A} \right) (\eta + \pi_A + \mu)}{(\eta + \pi_S + \delta + \mu)} \right) (\pi_S + \delta + \mu) \right) \tag{3.5.37}$$

Let

$$Q = \left(\frac{\left(\frac{\gamma_A + \mu}{\gamma_A} \right) (\eta + \pi_A + \mu)}{(\eta + \pi_S + \delta + \mu)} \right) (\pi_S + \delta + \mu)$$

And

$$K = \left(\frac{a_1 a_2 a_3}{\gamma_A (\eta + a_3)} \right)$$

Where

$$a_1 = \gamma_A + \mu, a_2 = \eta + \pi_A + \mu, a_3 = \pi_S + \delta + \mu$$

So

$$K = \left(\frac{(\gamma_A + \mu)(\eta + \pi_A + \mu)(\pi_S + \delta + \mu)}{\gamma_A (\eta + \pi_S + \delta + \mu)} \right)$$

Comparing we observe that

$$K = Q$$

Thus we can write

$$K\mathcal{R}_0 - Q$$

Then we have

$$Q\mathcal{R}_0 - Q$$

Hence

$$\dot{L}_{DFE} = (I_A + I_S) (Q(\mathcal{R}_0 - 1)) \tag{3.5.38}$$

\Rightarrow when $\mathcal{R}_0 = 1$ then $\dot{L}_{DFE} = 0$ and when $\mathcal{R}_0 < 1$ then $\dot{L}_{DFE} < 0$. Therefore, globally asymptotically stable when $\mathcal{R}_0 \leq 1$.

3.5.3 Existence and Uniqueness of the Endemic Equilibrium

We here present the existence and uniqueness of the endemic equilibrium for the model 2.1.2. We shall make use of the basic reproduction number \mathcal{R}_0 .

Let $\mathcal{E}^* = (S^*, E^*, I_A^*, I_S^*, R^*)$ be the positive endemic equilibrium of model. Then, the positive endemic equilibrium can be obtained by setting the right hand side of equations in the model 2.1.2 equal to zero, giving

$$\begin{cases} \Lambda + \xi R^* - \beta S^*(I_A^* + I_S^*) - \mu S^* = 0 \\ \beta S^*(I_A^* + I_S^*) - a_1 E^* = 0 \\ \gamma_A E^* - a_2 I_A^* = 0 \\ \eta I_A^* - a_3 I_S^* = 0 \\ \pi_A I_A^* + \pi_S I_S^* - a_4 R^* = 0 \end{cases} \tag{3.5.39}$$

where

$$a_1 = \gamma_A + \mu, \quad a_2 = \eta + \pi_A + \mu, \quad a_3 = \pi_S + \delta + \mu, \quad a_4 = \xi + \mu.$$

Using the first, second, fourth and fifth equations of equation 3.5.39, one has

$$S^* = \frac{a_1 a_2 a_3}{\beta \gamma_A (a_3 + \eta)}, \quad E^* = \frac{a_2 \Gamma}{\gamma_A \Gamma_1}, \quad I_A^* = \frac{\Gamma}{\Gamma_1}, \quad I_S^* = \frac{\eta \Gamma}{a_3 \Gamma_1}, \quad R^* = \frac{(\pi_A a_3 + \pi_S \eta) \Gamma}{a_3 a_4 \Gamma_1}.$$

We now substitute the above expressions of S^* , E^* , I_S^* , and R^* into \mathcal{R}_0 , one obtains the following endemic equilibrium equations

$$\begin{cases} S = \frac{\mathcal{R}_0 a_1 a_2 a_3}{\beta \gamma_A (a_3 + \eta)}, \\ E^* = \frac{\beta S (\eta + a_3) \Gamma}{\mathcal{R}_0 a_1 a_3 \Gamma_1}, \\ I_S^* = \frac{\mathcal{R}_0 a_1 a_2 \Gamma}{\beta S \gamma_A \Gamma_1} - 1, \\ R^* = \frac{\mathcal{R}_0 a_1 a_2 a_3 (\pi_A + \pi_S) \Gamma}{\beta S \gamma_A a_3 a_4 \Gamma_1} \end{cases} \tag{3.5.40}$$

with

$$\Gamma = [\mu a_1 a_2 a_3 - \Lambda \beta \gamma_A (a_3 + \eta)] a_3 a_4,$$

$$\Gamma_1 = [\xi \gamma_A (\pi_A a_3 + \pi_S \eta) - a_1 a_2 a_3 a_4] \beta (a_3 + \eta),$$

$$\mathcal{R}_0 = \frac{\beta S \gamma_A (\eta + a_3)}{a_1 a_2 a_3},$$

Lemma *Provided $\mathcal{R}_0 > 1$, there exist solution to the system of equation 2.1.3 such that the model can attain endemic equilibrium.*

Proof. Using the Next Generation method we have shown that

$$\mathcal{R}_0 = \frac{\beta S \gamma_A (\eta + a_3)}{a_1 a_2 a_3}$$

$$S = \frac{\mathcal{R}_0 a_1 a_2 a_3}{\beta \gamma_A (a_3 + \eta)}.$$

$$(1) S = S^* \mathcal{R}_0 > S^* \mathcal{R}_0 - S^* = S^* (\mathcal{R}_0 - 1)$$

Thus S is positive (and therefore exists) only if $\mathcal{R}_0 > 1$.

(2) Let $\mathcal{G} = \Gamma \gamma_A$ and $\mathcal{G}_1 = \Gamma_1 a_1 a_2$ then

$$I_A = \left(\frac{\mathcal{G}}{\mathcal{G}_1}\right) S$$

$$I_A = \left(\frac{\mathcal{G}}{\mathcal{G}_1}\right) S^* \mathcal{R}_0 = I_A^* \mathcal{R}_0$$

$$I_A = I_A^* \mathcal{R}_0 > I_A^* \mathcal{R}_0 - I_A^* = I_A^* (\mathcal{R}_0 - 1)$$

Thus I_A is positive (and therefore exists) only if $\mathcal{R}_0 > 1$.

(3) Since E , I_S , and R are each propotional to I_A , it follows similarly that E , I_S , and R are each positive (and therefore exists) only if $\mathcal{R}_0 > 1$.

The result follows since $(S^*, E^*, I_A^*, I_S^*, R^*)$ is the limit point of the considered neighbourhood. It is noted that a given initial value problem (IVP) has a unique solution.

3.5.4 Global Stability of the Endemic Equilibrium

In this section, we offer a finding pertaining to the presence and distinctiveness of the global asymptotic stability in the nonnegative orthant.

Theorem 3.5: When $\mathcal{R}_0 > 1$, the endemic equilibrium $\mathcal{E}^* = (S^*, E^*, I_A^*, I_S^*, R^*)$ is globally asymptotically stable in \mathbb{R}^5_+ .

Proof. When considering the system $\mathcal{R}_0 > 1$ there exists a unique endemic equilibrium $(S^*, E^*, I_A^*, I_S^*, R^*)$ given as in 2.1.3. The following Lyapunov function candidate is considered:

$$L_{EE}(t) = (S - S^* \ln S) + A_1(E - E^* \ln E) + A_2(I_A - I_A^* \ln I_A) + A_3(I_S - I_S^* \ln I_S) + A_4(R - R^* \ln R), \tag{3.5.41}$$

where A_1 is a constant that will later be established, followed by $A_2, A_3,$ and A_4 . With regard to time, this function can be differentiated to produce

$$\begin{aligned} \dot{L}_{EE}(t) &= \left(1 - \frac{S^*}{S}\right) \dot{S} + A_1 \left(1 - \frac{E^*}{E}\right) \dot{E} + A_2 \left(1 - \frac{I_A^*}{I_A}\right) \dot{I}_A + A_3 \left(1 - \frac{I_S^*}{I_S}\right) \dot{I}_S + A_4 \left(1 - \frac{R^*}{R}\right) \dot{R} \\ &= \left(1 - \frac{S^*}{S}\right) [\Lambda - \mu S - \beta S(I_A + I_S) + \xi R] \\ &\quad + A_1 \left(1 - \frac{E^*}{E}\right) [\beta S(I_A + I_S) - a_1 E] \\ &\quad + A_2 \left(1 - \frac{I_A^*}{I_A}\right) [\gamma_A E - a_2 I_A] + A_3 \left(1 - \frac{I_S^*}{I_S}\right) [\eta I_A - a_3 I_S] \\ &\quad + A_4 \left(1 - \frac{R^*}{R}\right) [\pi_A I_A + \pi_S I_S - a_4 R] \end{aligned} \tag{3.5.42}$$

where $a_1, a_2, a_3,$ and a_4 are defined as in 2.1.3. By considering 2.1.3, one has

$$\begin{cases} \Lambda = \mu S^* - \beta S^*(I_A^* + I_S^*) + \xi R^* \\ a_1 E^* = \beta S^*(I_A^* + I_S^*) \\ a_2 I_A^* = \gamma_A E^* \\ a_3 I_S^* = \eta I_A^* \\ a_4 R^* = \pi_A I_A^* + \pi_S I_S^* \end{cases} \tag{3.5.43}$$

with this in mind 3.5.42 becomes

$$\begin{aligned} \dot{L}_{EE}(t) &= \left(1 - \frac{S^*}{S}\right) [\mu S^* + \beta S^*(I_A^* + I_S^*) - \xi R^* - \mu S - \beta S(I_A + I_S) + \xi R] \\ &\quad + A_1 \left(1 - \frac{E^*}{E}\right) [\beta S(I_A + I_S)] - A_1 a_1 E + A_1 \beta S^*(I_A^* + I_S^*) \\ &\quad + A_2 \left(1 - \frac{I_A^*}{I_A}\right) [\gamma_A E] - A_2 a_2 I_A + A_2 \gamma_A E^* \\ &\quad + A_3 \left(1 - \frac{I_S^*}{I_S}\right) [\eta I_A] - A_3 a_3 I_S + A_3 \eta I_A^* \\ &\quad + A_4 \left(1 - \frac{R^*}{R}\right) [\pi_A I_A + \pi_S I_S] - A_4 a_4 R + A_4 \pi_A I_A^* + A_4 \pi_S I_S^* \\ &= -\mu \frac{(S - S^*)^2}{S} + \beta S^*(I_A^* + I_S^*) \left(1 - \frac{S^*}{S}\right) + \xi R^* \\ &\quad + [A_3 \eta + A_4 \pi_A - A_2 a_2 + \beta S^*] I_A + [-A_3 a_3 + A_4 \pi_S + \beta S^*] I_S \\ &\quad + [-A_1 a_1 + A_2 \gamma_A] E + [-A_4 a_4] R + A_2 \gamma_A E^* \left(1 - \frac{E}{E^*}\right) + A_3 \eta I_A^* \left(1 - \frac{I_A}{I_A^*}\right) \\ &\quad + A_4 \pi_A I_A^* \left(1 - \frac{I_A}{I_A^*}\right) + A_4 \pi_S I_S^* \left(1 - \frac{I_S}{I_S^*}\right) - A_1 \beta S^* \frac{E^*}{E} \frac{S}{S^*} (I_A^* + I_S^*) - \xi R^* \left(1 - \frac{R}{R^*}\right) \end{aligned} \tag{3.5.44}$$

Now, let $(x, y, z, v, w) = \left(\frac{S^*}{S}, \frac{E^*}{E}, \frac{I_A^*}{I_A}, \frac{I_S^*}{I_S}, \frac{R^*}{R}\right)$, we have

$$\begin{aligned} \dot{L}_{EE}(t) = & -\mu \frac{(S - S^*)^2}{S} + \beta S^* (I_A^* + I_S^*) (1 - x) + \xi R^* + [-A_1 a_1 + A_2 \gamma_A] E \\ & + [A_3 \eta + A_4 \pi_A - A_2 a_2 + \beta S^*] I_A + [-A_3 a_3 + A_4 \pi_S + \beta S^*] I_S + [-A_4 a_4] R \\ & + A_2 \gamma_A E^* (1 - y) + A_3 \eta I_A^* (1 - z) + A_4 \pi_A I_A^* (1 - z) \\ & + A_4 \pi_S I_S^* (1 - v) - A_1 \beta S^* \frac{y}{x} (I_A^* + I_S^*) - \xi R^* (1 - w) \end{aligned} \tag{3.5.45}$$

In order for the coefficients of $S(I_A + I_S)$, E , I_A , I_S , and R to be equal to zero, the nonnegative constants A_1, A_2, A_3 and A_4 are selected

$$\begin{cases} -1 + A_1 = 0, \\ -A_1 a_1 + A_2 \gamma_A = 0, \\ -A_4 a_4 + \xi = 0, \\ A_4 \pi_A + A_3 \eta - A_2 a_2 + \beta S^* = 0, \\ A_4 \pi_S - A_3 a_3 + \beta S^* = 0 \end{cases} \tag{3.5.46}$$

With the help of 3.5.39, we can easily state that the fifth equation of 3.5.46 is satisfied when the first and fourth equations of 3.5.46 are satisfied. As a result, in the following equations, we just take into account:

$$\begin{cases} A_1 = 1, \\ A_1 a_1 = A_2 \gamma_A, \\ -A_4 a_4 + \xi = 0, \\ A_4 \pi_A + A_3 \eta - A_2 a_2 + \beta S^* = 0. \end{cases} \tag{3.5.47}$$

The equation above when solved gives

$$A_1 = 1, A_2 = \frac{\gamma_A}{a_1}, A_4 = \frac{\xi}{a_4}, A_3 = \frac{A_2 a_2 - A_4 \pi_A - \beta S^*}{\eta} \tag{3.5.48}$$

Replacing the above expressions of A_1, A_2, A_3 , and A_4 in 3.5.45 one obtains

$$\begin{aligned} \dot{L}_{EE}(t) = & -\mu \frac{(S - S^*)^2}{S} + \beta S^* (I_A^* + I_S^*) (2 - x) + \xi R^* (1 - w) \\ & - A_1 \beta S^* (I_A^* + I_S^*) \frac{y}{x} + A_2 \gamma_A E^* (1 - y) \\ & + A_3 \eta I_A^* (1 - z) + A_4 \pi_A I_A^* (1 - z) + A_4 \pi_S I_S^* (1 - v) \end{aligned} \tag{3.5.49}$$

Now, using the fact that $A_1 = 1$, equation 3.5.49 becomes

$$\begin{aligned} \dot{L}_{EE}(t) = & -\mu \frac{(S - S^*)^2}{S} + A_1 \beta S^* (I_A^* + I_S^*) (2 - x) + \xi R^* (1 - w) \\ & - A_1 \beta S^* (I_A^* + I_S^*) \frac{y}{x} + A_2 \gamma_A E^* (1 - y) \\ & + A_3 \eta I_A^* (1 - z) + A_4 \pi_A I_A^* (1 - z) + A_4 \pi_S I_S^* (1 - v), \\ = & -\mu \frac{(S - S^*)^2}{S} + A_1 \beta S^* (I_A^* + I_S^*) \left(2 - x - \frac{y}{x}\right) + \xi R^* (1 - w) \\ & + A_2 \gamma_A E^* (1 - y) + A_3 \eta I_A^* (1 - z) + A_4 \pi_A I_A^* (1 - z) + A_4 \pi_S I_S^* (1 - v) \end{aligned} \tag{3.5.50}$$

The result of multiplying the second equation of 3.5.43 by A_1 and the second equation of 3.5.47 by E^* is

$$\begin{cases} A_1 a_1 E^* = A_2 \gamma_A E^* \\ A_1 a_1 E^* = A_1 \beta S^* (I_A^* + I_S^*) \end{cases} \tag{3.5.51}$$

Hence, it clearly appears that

$$-A_1\beta S^*(I_A^* + I_S^*) + A_2\gamma_A E^* = 0 \tag{3.5.52}$$

Now, multiplying the preceding equation by $F_1(u)$ where $u = (x, y, z, v, w)^T$ and $F_1(u)$ a function to be calculated later, gives

$$-A_1\beta S^*(I_A^* + I_S^*)F_1(u) + A_2\gamma_A E^*F_1(u) = 0 \tag{3.5.53}$$

Additionally, if the fourth equation of 3.5.43 is multiplied by A_4 and the third equation of 3.5.47 is multiplied by R^* , the result is

$$\begin{cases} A_4a_4R^* = \xi R^* \\ A_4a_4R^* = A_4\pi_A I_A^* + A_4\pi_S I_S^* \end{cases} \tag{3.5.54}$$

Then, one can deduce that

$$-A_4\pi_A I_A^* - A_4\pi_S I_S^* + \xi R^* = 0 \tag{3.5.55}$$

Additionally, multiplying the previous equation by $F_2(u)$ where $u = (x, y, z, v, w)^T$ and $F_2(u)$ is a later-determined function, gives

$$-A_4\pi_A I_A^*F_2(u) - A_4\pi_S I_S^*F_2(u) + \xi R^*F_2(u) = 0 \tag{3.5.56}$$

Thus, after plugging equations 3.5.53 and 3.5.56 into equation 3.5.50, one obtains

$$\begin{aligned} \dot{L}_{EE}(t) = & -\mu \frac{(S - S^*)^2}{S} + A_1\beta S^*(I_A^* + I_S^*) \left(2 - x - \frac{y}{x} - F_1(u) \right) \\ & + \xi R^*(1 - w + F_2(u)) + A_2\gamma_A E^*(1 - y + F_1(u)) + A_3\eta I_A^*(1 - z - F_2(u)) \\ & + A_4\pi_A I_A^*(1 - z - F_2(u)) + A_4\pi_S I_S^*(1 - v - F_2(u)) \end{aligned} \tag{3.5.57}$$

In order to ensure that the coefficients of E^* and R^* are equal to zero the functions $F_1(u)$ and $F_2(u)$ are selected. In this scenario, one gets

$$F_1(u) = \left(\frac{1}{y} - 1 \right) \text{ and } F_2(u) = \left[\left(\frac{1}{z}, \frac{1}{v}, \frac{1}{w} \right) - 1 \right] \text{ for } I_A^*, I_S^*, \text{ and } R^* \text{ respectively.} \tag{3.5.58}$$

Then, we finally have

$$\begin{aligned} \dot{L}_{EE}(t) = & -\mu \frac{(S - S^*)^2}{S} + A_1\beta S^*(I_A^* + I_S^*) \left(3 - x - \frac{y}{x} - \frac{1}{y} \right) \\ & + A_3\eta I_A^* \left(2 - z - \frac{1}{z} \right) + A_4\pi_A I_A^* \left(2 - z - \frac{1}{z} \right) + A_4\pi_S I_S^* \left(2 - v - \frac{1}{v} \right) \end{aligned} \tag{3.5.59}$$

So, it can be seen from the arithmetic-geometric means inequality that $\dot{L}_{EE}(t) \leq 0$ is equal only if $S = S^*$ and $y = z = v = w$. The endemic equilibrium is globally asymptotically stable in Π , according to the LaSalle's invariance principle. The fact that Π is absorbing establishes the positive orthant's global asymptotic stability.

4. Numerical Analysis of the Model

Using Ghana data, we now perform numerical simulations of the model. Table 2 displays estate parameter values with relevant sources in the literature indicated.

The sensitivity indices of the model parameters influencing \mathcal{R}_0 are shown in Table 3 using the parameter values in Table 2 as a basis.

Table 2. Parameters estimates of the $SEI_A I_S RS$ model

Parameter	Value(per day)	Source
Λ	2.8452 (365days)	Okyere and Ackora-Prah (2022)
β	$0.83117941 \times 10^{-4}$	Okyere and Ackora-Prah (2022)
η	0.1200	Estimated
γ_A	0.2344×10^{-4}	Estimated
π_A	0.080	Estimated
π_S	0.563	Estimated
δ	0.00002442	Estimated
μ	0.4252912×10^{-5}	Estimated
ξ	0.0167	Akuka et al. (2022)

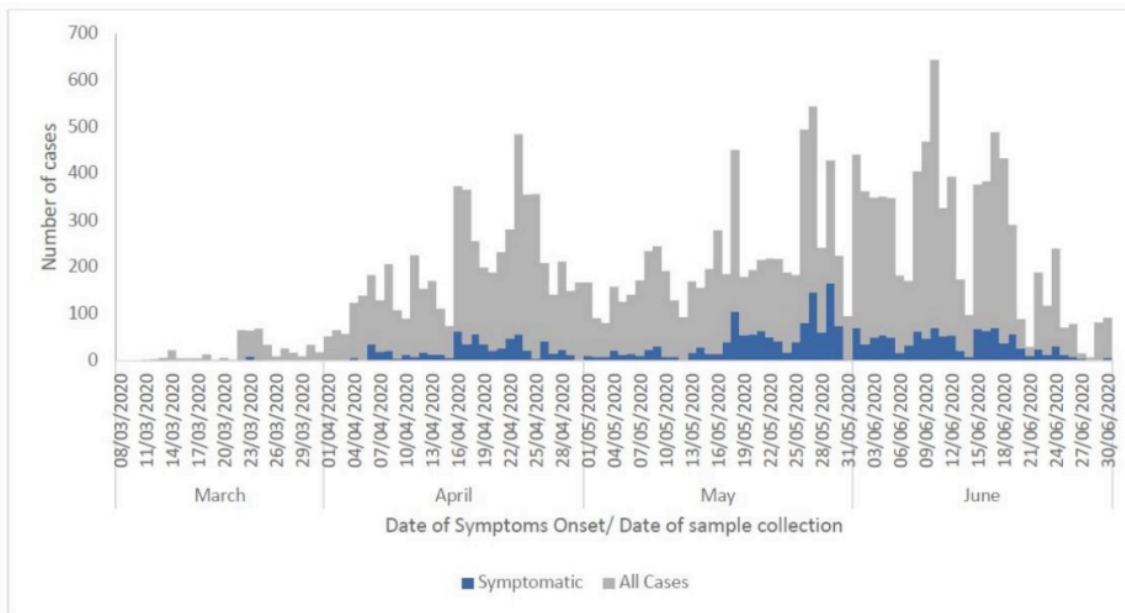


Figure 2. Epidemiological Curve of confirmed COVID-19 cases in Ghana, March 12, 2020 C June 30, 2020

Table 3. Sensitivity indices of the $SEI_A I_S RS$ model parameters

Parameter	Sensitivity index
Λ	+1.000
β	+1.000
η	-0.599
γ_A	+0.846
π_A	-0.399
π_S	-0.999
δ	-4.337×10^{-5}
μ	-1.000

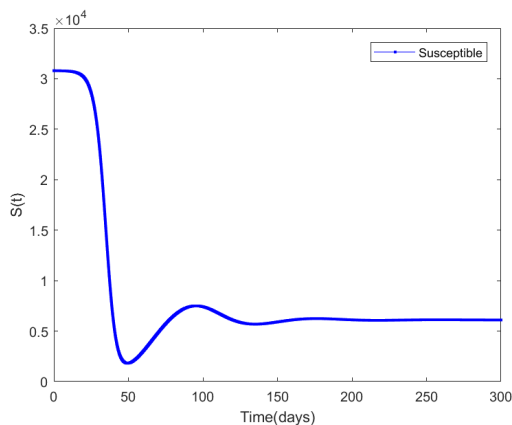


Figure 3. Susceptible

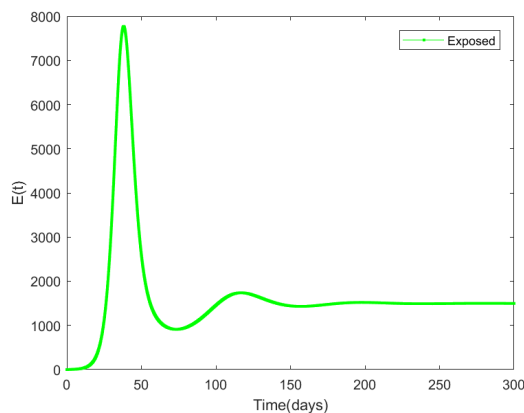


Figure 4. Exposed

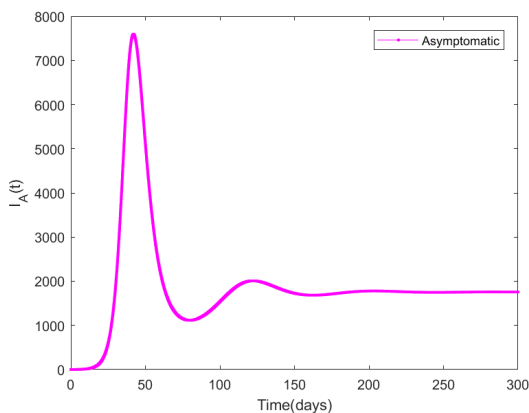


Figure 5. Infectious Asymptomatic

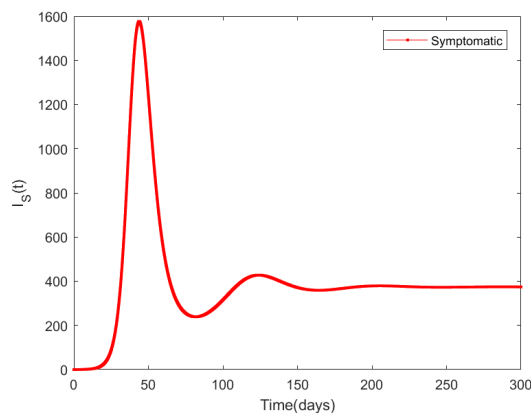


Figure 6. Infectious Symptomatic

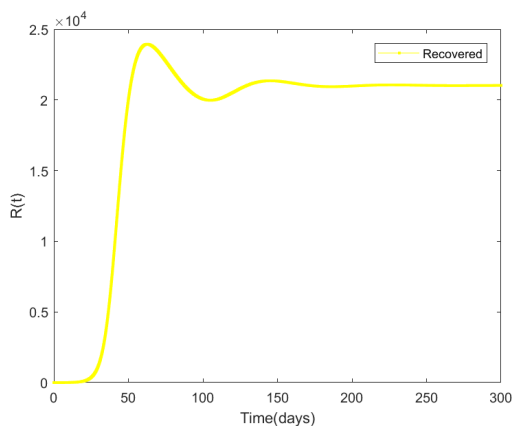


Figure 7. Recovered

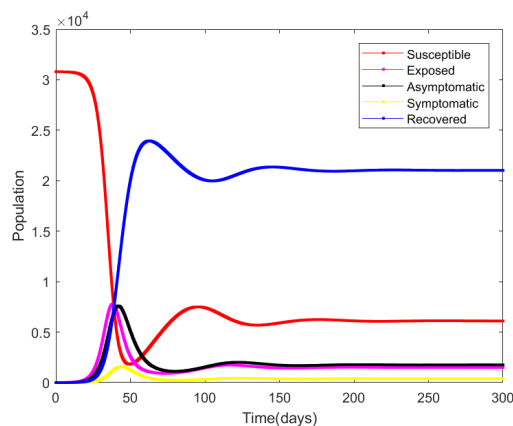


Figure 8. All variables

With positive sign of Λ , β and γ_A in Table 3, there is a positive correlation between each of these parameters and \mathcal{R}_0 which indicates that increasing (decreasing) any of these parameters would lead to increase (decreasing) in \mathcal{R}_0 , thus increasing (decreasing) the prevalence of COVID-19 disease. Also, with negative sign of η , π_A , π_S , δ and μ , there is a negative correlation between any of these parameters and \mathcal{R}_0 . This means that increasing (decreasing) any of these parameters would lead to decrease (increase) of \mathcal{R}_0 , thus decreasing (increasing) the prevalence of COVID-19 disease.

Based on these parameter values, we compute \mathcal{R}_0 and the vulnerability factor as follows: $\mathcal{R}_0 = 0.78216$, and $\phi = 1.42179 \times$

10^{-4} .

COVID-19 epidemic in Ghana therefore has a locally asymptotic stability at the disease free equilibrium point. Also, the result shows that the Ghana population has low vulnerability which is due to the relatively young population of that country. In fact, according to the 2021 Population and Housing census, the population of the country is 30.8 million and 3.14% of the population are 65 and above. Also 47.13% of the population are between the age 0-19 years (Service, 2021).

Inserting the parameter values in the model equations we have, for the model, $S(0) = 30,799,998$, $E(0) = 0.2$, $I_A(0) = 0.2$, $I_S(0) = 0$, $R(0) = 0$. The equations were numerically solved using MATLAB ode45 and plots of the trajectories of the variables S , E , I_A , I_S , and R obtained.

Figure 3 shows that the susceptible population monotonically decreases with time for the first 250 days. Figure 4, 5, and 6 indicate a peaking of infected populations and a tapering off by day 250. Considering Figure 4, 5, and 6 we observed that the number of exposed individuals turns to be greater than the number of infectious asymptomatic individuals which in turns is also greater than the number of infectious symptomatic individuals. From Figure 7 we see that the number of recovering individuals is higher than any of the other individuals. The above is summarized in Figure 8.

Epidemiological Curve of COVID-19 Cases, March-June 2020, the distribution of cases showed a propagated outbreak with multiple peaks of about one-month interval. The highest peak of confirmed cases as at June 30, 2020 was observed at June 10, 2020, (Kenu et al., 2020). The index cases were confirmed on March 12, 2020. Among symptomatic patients, cases were distributed over the four months with multiple peaks with the highest peak on April 29, 2020 (Figure 2).

5. Conclusion

We have formulated an $SEI_A I_S RS$ Transmission Dynamics model of COVID-19 and proved that it is epidemiological well posed, is globally asymptotically stable at the disease free equilibrium and at the endemic equilibrium when $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$ respectively. We also show that a vulnerability factor define via vulnerability dependent parameters, when appropriately bounded leads to stability at the disease free equilibrium.

Finally employing secondary clinical COVID-19 data on symptomatic and asymptomatic cases together with age structure census data from Ghana, we are able to demonstrate that the relatively low impact of the pandemic could be largely due to the youthfulness of the Ghanaian population. This evidently is the cause of a sufficiently low vulnerability so that $\mathcal{R}_0 < 1$. Thus the transition dynamics is necessarily globally asymptotically stable at the disease free equilibrium. Thus for this population, the disease can be eradicated and tends not to persist.

In a sequel to this paper we shall further explore issues raised here in the context of an age structured extension of our basic model.

References

- Akuka, P. N. A., Seidu, B., & Bornaa, C. S. (2022). Mathematical analysis of covid-19 transmission dynamics model in ghana with double-dose vaccination and quarantine. *Computational and Mathematical Methods in Medicine*.
- Al-Sheikh, S. (2013). Modeling and analysis of an seir epidemic model with a limited resource for treatment. *Global J. Sci. Frontier Res. Math. Decis. Sci.*, 12, 56–66.
- Asamoah, J. K. K., Bornaa, C., Seidu, B., & Jin, Z. (2020). Mathematical analysis of the effects of controls on transmission dynamics of sars-cov-2. *Alexandria Engineering Journal*, 59(6), 5069–5078.
- Asamoah, J. K. K., Jin, Z., Sun, G.-Q., Seidu, B., Yankson, E., Abidemi, A., Oduro, F. T., Moore, S. E., & Okyere, E. (2021). Sensitivity assessment and optimal economic evaluation of a new covid-19 compartmental epidemic model with control interventions. *Elsevier*.
- Bauch, C., Szusz, E., & Garrison, L. (2009). Scheduling of measles vaccination in lowincome countries: Projections of a dynamic model. *Vaccine*, 31(27), 4090-4098.
- Bowong, S., & Jules, T. J. (2009). Mathematical analysis of a tuberculosis model with differential infectivity. *Commun Nonlinear Sci Numer Simulat, Elsevier Ltd*, 14, 4010–4021.
- Bowong, S., & Kurths, J. (2010). Parameter estimation based synchronization for an epidemic model with application to tuberculosis in cameroon. *Phys. Lett. A*, 44(374), 4496-4505.
- Ega, T. T., & Ngeleja, R. C. (2022). Mathematical model formulation and analysis for covid-19transmission with virus transfer media and quarantineon arrival. *Hindawi Computational and Mathematical Methods*, 16.

- Gorbalenya, A. (2020). Severe acute respiratory syndrome-related coronavirus: the species and its viruses—a statement of the coronavirus study group. *bioRxiv*.
- Huang, C. (2020). Clinical features of patients infected with 2019 novel coronavirus in wuhan, china. *Lancet*, 395(10223), 497-506.
- Iboi, E., Ngonghala, C. N., & Gumel, A. B. (2020). Will an imperfect vaccine curtail the covid-19 pandemic in the u.s.? *Elsevier B.V. on behalf of KeAi Communications Co. Ltd.*
- Kenu, E., Odikro, M. A., Malm, K. L., Asiedu-Bekoe, F., Noora, C. L., Frimpong, J. A., Calys-Tagoe, B., & Koram, K. A. (2020). Epidemiology of covid-19 outbreak in ghana, 2020. *Ghana Med. Journal*, 54(4).
- Kermack, W. O., & Mckendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. *Proc. R. Soc. Lond.*, 115, 700–721.
- Mahardika, R., Widowati, & Sumanto, Y. (2019). Routh-hurwitz criterion and bifurcation method for stability analysis of tuberculosis transmission model. *Journal of Physics : Conference Series*.
- Meyer, J. F. C. A., & Lima, M. (2022). Relevant mathematical modelling efforts for understanding covid-19 dynamics: an educational challenge. *Springer, ZDM ÍC Mathematics Education*, (55), 49–63.
- Mukandavire, Z., Chiyaka, C., Garira, W., & Musuka, G. (2009). Mathematical analysis of a sex-structured hiv/aids model with a discrete time delay. *Nonlinear Anal.Theory Methods Appl.*, 3(71), 1082-1093.
- Okyere, S., & Ackora-Prah, J. (2022). A mathematical model of transmission dynamics of sars-cov-2(covid-19) with an underlying condition of diabetes. *Hindawi International Journal of Mathematics and Mathematical Sciences*, 15.
- Olumuyiwa, J. P., Qureshi, S., Yusuf, A., Al-Shomrani, M., & Idowu, A. A. (2021). A new mathematical model of covid-19 using real data from pakistan. *Results in Physics by Elsevier*.
- Pesco, P., Bergero, P., Fabricius, G., and Hozbor, D. (2014). Modelling the effect of changes in vaccine effectiveness and transmission contact rates on pertussis epidemiology. *Epidemics*, 7, 13-21.
- Qun, L., Guan, X., Wu, P., Wang, X., Zhou, L., & Tong, Y. (2020). Early transmission dynamics in wuhan, china, of novel coronavirus infected pneumonia. *NewEngl. J. Med.*, 382(13), 1199-1207.
- Riou, J., & Althaus, C. (2020). Pattern of early human-to-human transmission of wuhan2019 novel coronavirus (2019-nCoV), december 2019 to january 2020. *Eurosurveillance* 25(4), 5.
- Service, G. H. (2021). Covid-19 ghana's outbreak response management updates. <http://ghanahealthservice.org/covid19/archive.php>.
- van den Driessche, P., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.*, 180(1-2), 29-48.
- Widyaningsih, P., Saputro, D., & Nugroho, A. (2018). Susceptible exposed infected recovery (seir) model with immigration: Equilibria points and its application. *Proceedings of the AIP Conference Proceedings*, (1).
- Yavuz, M., & Haydar, W. Y. A. (2022). A new mathematical modelling and parameter estimation of covid-19: a case study in iraq. *AIMS Bioengineering*, 9(4), 420-446.

Copyrights

Copyright for this article is retained by the author(s), with first publication rights granted to the journal. This is an open-access article distributed under the terms and conditions of the Creative Commons Attribution license (<http://creativecommons.org/licenses/by/4.0/>).