

Qualitative and Mathematical Analysis of COVID-19 with Relapse and Re-infection Rate: A Deterministic Modelling Approach

*¹Loyinmi, A.C., ²Ijaola, A.L., ¹Shittu, M.S., & ¹Ajala, A.S.

¹Department of Mathematics, Tai Solarin University of Education, Ogun State, Nigeria

²Department of Mathematics, Federal University of Agriculture, Abeokuta, Ogun State, Nigeria.

*Corresponding author email: loyinmiac@tasued.edu.ng

Abstract

According to the Centres for Disease Control and Prevention (CDC) and World Health Organisation (WHO), there is a high possibility of reoccurrence of COVID-19 in humans. In this study, we presented a modified SEIR model equipped with ordinary differential equations to demonstrate the dynamics of COVID 19 viral transmission, taking into account the rates of relapse and re-infection. Necessary qualitative, mathematical and sensitivity analysis were done to validate the propose model. Similarly, the model was found to be stable both locally and globally with respect to the reproduction number R_0 . Findings from the graphical solutions depict how these two factors affect the transmission dynamics, and how proper control measures will help in flattening out the transmission curve of the disease in the community at large.

Keywords: COVID 19, relapse rate, re-infected, stability, treatment rate, Sensitivity Analysis

Introduction

Pathogens that cause infectious diseases are still a major concern for worldwide public health and socioeconomic development (Loyinmi et al., 2024; Loyinmi et al., 2023; Zhu et al., 2020). Among them are coronaviruses and other worldwide dominant infectious illnesses such as TB, meningitis, sexually transmitted infections, and vector-borne illnesses. COVID-19 is a class of RNA viruses that mostly infect humans (other mammals). They can cause diseases of the respiratory, hepatic systems. Gastrointestinal and Neurological. The severe acute respiratory syndrome problem that causes the coronavirus disease 2019 (COVID-19) originated in China in December 2019 and was deemed a worldwide pandemic by the World Health Organization (WHO) on March 11, 2020. The COVID-19 epidemic is currently ongoing and continues to cause severe socioeconomic and public health problems around the globe (Gbodogbe, 2025; Huang et al., 2020; Li et al., 2020; Zhao et al., 2020; Khan & Atangana, 2020). On April 15, precisely in 2022, the infection had spread to over 220 nations and territories worldwide, resulting in over 550 million cases and over 6.5 million fatalities, six more human coronaviruses, including the Middle East respiratory syndrome, the severe acute respiratory syndrome coronavirus, are known to infect people in addition to SARS-COV-2 (Khan & Atangana, 2020). Because of cyclic spillover events and recurrent cross-species infections, coronaviruses are expected to reappear in the human population on a seasonal or annual basis. This is likely caused by the high frequency and extensive geographic spread of coronaviruses, the great diversity of persistent genome recombination genetic variation, and the rise in interactions between animals and humans. Together with non-pharmaceutical therapy (NPT) strategies (such as social distance, quarantine use of face mask, contact tracing, isolation, travel limitations, border and school security), the COVID-19 vaccinations successfully lessens the pandemic's effects, particularly in regard to case of severity and death (Eikenberry et al., 2020; Musa et al., 2020; Wai-Kit et al., 2020).

Some researchers conducted a qualitative study to investigate the COVID-19 outbreak utilizing the notion of fundamental reproduction ratio and stability theory of differential equations. In essence, they used some historical statistical data from Pakistan to predict the epidemic behavior and they used numerical methodologies like Nsf and Ode45 to figure out the research (Olumuyiwa et al., 2021). In addition to building the mathematical model to investigate the coronavirus's influence behavior, some scientist also talked about the stability of their suggested model (Annas et al., 2020; Ndairou et al., 2020). Many fractional order modeling to equally investigate the

transmission dynamics of infectious diseases abound (Loyinmi, 2024; Loyinmi & Ijaola, 2024a; Loyinmi & Ijaola, 2024b; Atangana & Araz, 2022) but three groups of researchers statistically examined the COVID-19 fractional model (Ighal & Karaca, 2021; Rahman et al., 2019; Suleman et al., 2018). The National Health Organization advised wearing surgical face masks and constantly using hand disinfectants in public areas to protect oneself from infection, as well as avoiding contact with sick people and animals that had fever or other breathing problems. Since the coronavirus is more prone to spread in confined spaces, social distance or less crowded areas can lower the danger of it spreading (Din et al., 2020; Hoehl et al., 2020). At some point, an SIR model was developed to estimate the coronavirus's final readings (Batista, 2020).

In most epidemic models, the spreading rate of infection or disease-free equilibrium point is considered to be endemic or stable if R_0 is between 0 and 1, otherwise it would be epidemic or unstable. Fundamental reproductive number is essentially determined from the mathematical proposed model. In general, it is more difficult to contain a disease epidemic when the values of R_0 are higher. Early on in the COVID-19 outbreak, Zhao et al., (2020) and Chen et al., (2020) provided a test approximation of the fundamental reproductive ratio. However, determining the precise expression for the fundamental reproductive ratio is not that simple, necessitating the use of more sophisticated methods, such as next-generation matrix method, in order to determine the reproduction ratio (Pauline & Watmough, 2008).

Researchers introduced the research of the fundamental reproduction ratio for the spread of epidemical virus. They also introduced the steady state and unpredictability of infection-free system according to reproductive rate (Liu et al, 2020; Pauline & Watmough, 2002). To estimate the transfer of parameters and predict the influencing behavior of the virus, epidemic computer simulation models are essential. These models have demonstrated their value in illustrating the pace at which viruses proliferate or decay over time, as well as in offering management strategies that may be modified to slow the disease's progress. Numerous applications and findings from the COVID-19 simulation models are being approved and make available online. In 2020 a Simulink computer application was built to track the COVID-19 virus. Using the Simulink technique, the researcher reproduced the results using the SIR and SEIRD epidemic models in the form of differential equations (algebraic) (Diekmann et al, 1990).

Literatures on corona virus disease and its in-out flow transmissions dynamics showed sudden re-occurrence of infection have not been taken into account COVID 19. This can arise from either an exogenetic re-infection to a new strain of the virus or a relapse of the initial infection within a susceptible population. This study aims to demonstrate the effect of relapse and re-infection rate on the population density using a deterministic approach and also demonstrate the impact of proper control plan within the proposed model

Materials and Methods

Model formulation

Moving forward, In order to illustrate the dynamics of covid-19 transmission within the human populations, we built a deterministic model for the disease. The model makes some assumptions, of four human compartments which is equipped with biological parameters. The population as a whole is split into four classes: Susceptible = (S), Exposed (E), Infected = (I) and Recovered = (R). This is represented with a schematic flow below;

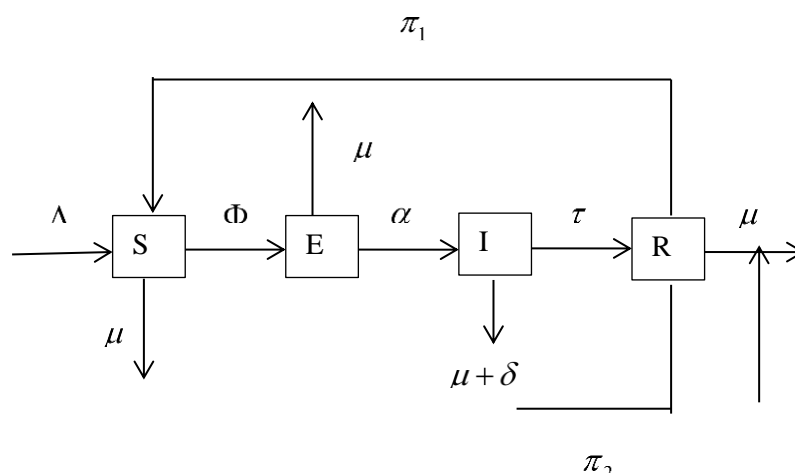


Figure 1: Schematic flow of the proposed model

Considering Figure 1, the rate of change in each class with respect to time is given as

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda - (\Phi + \mu)S + \pi_1 R \\ \frac{dE}{dt} &= \Phi S - (\mu + \alpha)E \\ \frac{dI}{dt} &= \alpha E - (\mu + \delta + \tau)I + \pi_2 R \\ \frac{dR}{dt} &= \tau I - (\mu + \pi_1 + \pi_2)R \end{aligned} \right\} \quad (1)$$

$$\Phi = \frac{\beta I}{1 + aI}$$

With the initial conditions

$$S(t) \geq 0, E(t) \geq 0, I(t) \geq 0 \text{ And } R(t) \geq 0$$

Λ = Rate of recruitment, Φ = incident rate which includes saturation factor a , the saturation factor is used as an inhibitory effect to lessen the infection severity while β is the contact rate of the disease, μ = Rate of natural death, α = Rate of progression from exposed to infectious, τ = Rate of progression from infected to recover, δ = Disease induced death, π_1 = Rate of relapse, π_2 = Rate of re-infection

Qualitative Analysis of the Model

Existence and Uniqueness of solution of the model.

Applying the Lipschitz criteria from the system of equations (1)

Let,

$$\left. \begin{aligned} C_1 &= \Lambda - \Phi S - \mu S + \pi_1 R \\ C_2 &= \Phi S - \mu E - \alpha E \\ C_3 &= \alpha E - (\mu + \delta + \tau)I + \pi_2 R \\ C_4 &= \tau I - (\mu + \pi_1 + \pi_2)R \end{aligned} \right\} \quad (2)$$

Theorem 1: Let ϕ represent the region $0 \leq \phi < \infty$, then system (2) exist, bounded and also has a unique solution

if and only if $\frac{\partial C_j}{\partial B_i}$ are continuous and bounded in ϕ , where B_i denote the state variables S, E, I, R for $j \neq i$

Proof

Partial derivative of system (2), each compartment needs to be verified in doing so we obtain;

$$\begin{aligned} \left| \frac{\partial C_1}{\partial S} \right| &= |-(\Phi + \mu)| < \infty, \quad \left| \frac{\partial C_1}{\partial E} \right| = |0| < \infty, \quad \left| \frac{\partial C_1}{\partial I} \right| = |0| < \infty, \quad \left| \frac{\partial C_1}{\partial R} \right| = |\pi_1| < \infty \\ \left| \frac{\partial C_2}{\partial S} \right| &= |\Phi| < \infty, \quad \left| \frac{\partial C_2}{\partial E} \right| = |-(\alpha + \mu)| < \infty, \quad \left| \frac{\partial C_2}{\partial I} \right| = |0| < \infty, \quad \left| \frac{\partial C_2}{\partial R} \right| = |\pi_2| < \infty \\ \left| \frac{\partial C_3}{\partial S} \right| &= |0| < \infty, \quad \left| \frac{\partial C_3}{\partial E} \right| = |\alpha| < \infty, \quad \left| \frac{\partial C_3}{\partial I} \right| = |-(\delta + \tau + \mu)| < \infty, \quad \left| \frac{\partial C_3}{\partial R} \right| = |0| < \infty \\ \left| \frac{\partial C_4}{\partial S} \right| &= |0| < \infty, \quad \left| \frac{\partial C_4}{\partial E} \right| = |0| < \infty, \quad \left| \frac{\partial C_4}{\partial I} \right| = |\tau| < \infty, \quad \left| \frac{\partial C_4}{\partial R} \right| = |\pi_1 + \pi_2 + \mu| < \infty \end{aligned}$$

From the derivative of the systems of equation (2), its solutions are bounded, exist and unique.

This ends the proof.

Non-negativity (Positivity) of solution

Theorem 2: For all time $t > 0$, the differential equation of system (1) solution with positive initial condition remain non-negative

Proof

Using the Mittag-Leffler function also known as two parameter function

$$E_{\alpha,\beta} = \frac{Z^k}{\Gamma(\alpha k + \beta)}, \text{ where } \alpha = \beta = 1 \text{ and } k = 0, 1, 2, \dots \quad (3)$$

Replacing the total human population $N(t)$ with Z ;

$$E_{1,1} = \frac{N(t)^0}{\Gamma(1)} + \frac{N(t)^1}{\Gamma(2)} + \frac{N(t)^2}{\Gamma(3)} + \dots \quad (4)$$

Neglecting the higher terms on the right hand side of (4)

$$E_{1,1} \approx \frac{N(t)^0}{\Gamma(1)} + \frac{N(t)^1}{\Gamma(2)} \quad (5)$$

Recall that $\Gamma(1) = 1$, therefore (5) yields

$$E_{1,1} \approx 1 \quad (6)$$

It's obvious from (6) that the proposed population will remain positive (non-negative). Which establish Theorem 2

Presence of disease free equilibrium point

The term "disease-free equilibrium points" refers to stable-state situation when the model becomes immune to the corona virus infection.

From system (1)

For $S^0 \neq 0, E = 0, I = 0, R = 0$, then we have that

$$\Lambda - \mu S^0 = 0, S^0 = \frac{\Lambda}{\mu}$$

This provides us with IFE point for the system (1)

$$E^0 = (S^0, E^0, I^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right) \quad (7)$$

Reproduction Number

For an infectious model, the reproduction number R_0 is crucial. In a population that is entirely vulnerable, R_0 indicates the typical number of secondary infections in the exposed compartment that are caused by an infectious person who is already in the infected compartment. We implement the method in [16] to derive R_0

Using the method of Next generation Matrix

$$R_0 = \rho(PQ^{-1}) \quad (8)$$

Where P is the matrix of new infection and Q is the matrix of other transfer terms and ρ is the spectral radius of matrix of PQ^{-1}

From system (1) we have;

$$P = \begin{bmatrix} 0 & \beta S \\ 0 & 0 \end{bmatrix}, \quad Q = \begin{bmatrix} -(\mu + \alpha) & 0 \\ \alpha & -(\mu + \delta + \tau) \end{bmatrix} \quad (9)$$

Solving (9) using (8) we obtain

$$R_0 = \frac{\alpha\beta S}{(\mu + \delta + \tau)(\mu + \alpha)}$$

From infection free equilibrium $S = \frac{\Lambda}{\mu}$ which implies that

$$R_0 = \frac{\alpha\beta\Lambda}{(\mu + \delta + \tau)(\mu + \alpha)\mu} \quad (10)$$

Analysis of the reproduction number

- If $R_0 < 1$, then the infection is under control within the population
- If $R_0 = 1$, means that the infection is stable and will not lead to outbreak
- If $R_0 > 1$, then there is an outbreak within the population

Sensitivity Analysis of the model

The aim of sensitivity analysis is to ascertain how each parameters of the model affects the rate of reproduction of the disease, these aids in determining the parameters that have a significant influence on R_0 . The basic reproduction rate is often examined to determine whether or not effective treatment could help in the management of the disease in the system,. This technique is widely employed to test the resilience of model assumptions to variable values.

Using the normalized forward sensitivity index of a variable a , is given by:

$$\Pi_b^a = \frac{\partial a}{\partial b} x \frac{b}{a}$$

This depends on the differentiability of parameter b

This method of solution will be used in analyzing parameters that make up the reproduction number.

$$R_0 = \frac{\alpha\beta\Lambda}{(\mu + \delta + \tau)(\mu + \alpha)\mu}$$

Analyzing the derivatives of R_0 with respect to the contact rate β we obtain;

$$\Pi_{\beta}^{R_0} = \frac{\partial R_0}{\partial \beta} \cdot \frac{\beta}{R_0}, \quad \frac{\partial R_0}{\partial \beta} = 0.08 \frac{R_0}{\beta}, \quad \Pi_{\beta}^{R_0} = \frac{\partial R_0}{\partial \beta} \cdot \frac{\beta}{R_0} = 0.08 \frac{R_0}{\beta} \cdot \frac{\beta}{R_0}$$

Therefore $\Pi_{\beta}^{R_0} = 0.08$,

It is established that the recruitment rate Λ and the contact rate β has much influence on the rate at which people will get infected in the proposed model.

In the same manner all other parameters will be obtained

Table 1 contains the parameter evaluation indices

Parameters	Sensitivity Indices (SI)
Λ	+ve
β	+ve
μ	-ve
α	+ve
τ	+ve
δ	-ve

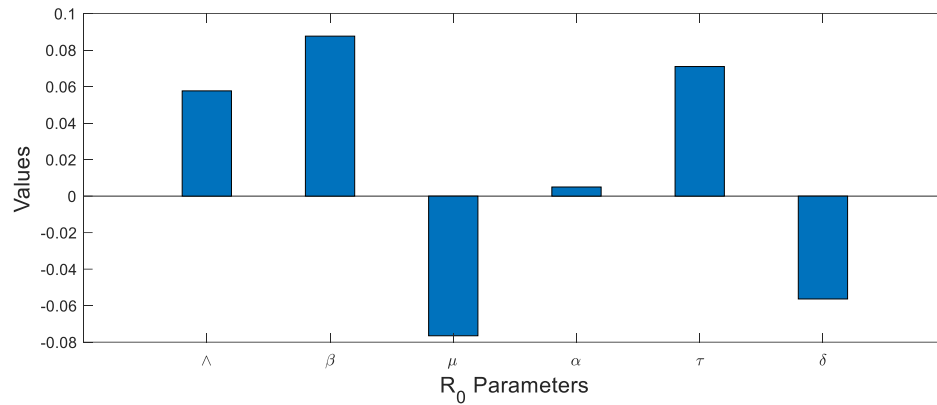


Figure 1: graphical representation of sensitivity analysis

Stability Analysis

Local Asymptotical Stability

Lemma 2: if $R_0 < 1$, then the system (1) is locally asymptotically stable and unstable otherwise

Proof.

The stability will be calculated at IFE (E^0)

$$J(S, E, I, R)$$

Let;

$$\begin{aligned} F_1 &= \Lambda - \Phi S - \mu S + \pi_1 R \\ F_2 &= \Phi S - \mu E - \alpha E \\ F_3 &= \alpha E - (\mu + \delta + \tau)I + \pi_2 R \\ F_4 &= \tau I - \mu R - \pi_1 R - \pi_2 R \end{aligned} \quad (11)$$

$$J = \begin{bmatrix} \frac{\partial F_1}{\partial S} & \frac{\partial F_1}{\partial E} & \frac{\partial F_1}{\partial I} & \frac{\partial F_1}{\partial R} \\ \frac{\partial F_2}{\partial S} & \frac{\partial F_2}{\partial E} & \frac{\partial F_2}{\partial I} & \frac{\partial F_2}{\partial R} \\ \frac{\partial F_3}{\partial S} & \frac{\partial F_3}{\partial E} & \frac{\partial F_3}{\partial I} & \frac{\partial F_3}{\partial R} \\ \frac{\partial F_4}{\partial S} & \frac{\partial F_4}{\partial E} & \frac{\partial F_4}{\partial I} & \frac{\partial F_4}{\partial R} \end{bmatrix} \quad (12)$$

At infection free equilibrium we obtain

$$J(E^0) = \begin{bmatrix} J_{11} & J_{12} & J_{13} & J_{14} \\ J_{21} & J_{22} & J_{23} & J_{24} \\ J_{31} & J_{32} & J_{33} & J_{34} \\ J_{41} & J_{42} & J_{43} & J_{44} \end{bmatrix} \quad (13)$$

Solving (18) we obtain the following Eigen values

$$J_{11} = -\mu, \quad J_{22} = -(\mu + \alpha), \quad J_{33} = -((\mu + \delta + \tau) - \frac{\alpha\beta S}{(\mu + \alpha)}), \quad \lambda_{44} = -\mu$$

From $J_{33} = -((\mu + \delta + \tau) - \frac{\alpha\beta S}{(\mu + \alpha)})$, we have that $J_{33} = -(\mu + \delta + \tau)(1 - \frac{\alpha\beta S}{(\mu + \delta + \tau)(\mu + \alpha)})$ which can be written as $J_{33} = -((\mu + \delta + \tau)(1 - R_0))$, it obvious that $R_0 < 1$ from J_{33}

Hence since all the Eigen values are negative (stable) and $R_0 < 1$ then system (1) is locally asymptotical stable (LAS)

This ends the proof.

Global Stability

Lemma 3: if $R_0 < 1$, then system (1) is Globally Asymptotical stable and unstable otherwise.

Proof

Splitting system (1) into two distinct parts;

$M = (S, R)$ and $N = (E, I)$ where M represent the set of uninfected sub-population and N represent the set infected sub-population

$$\frac{dM}{dt} = F(M, N) \text{ and } \frac{dN}{dt} = G(M, N), \quad G(M, 0) = 0$$

$$F(M, N) = \begin{bmatrix} \Lambda - \Phi S - \mu S + \pi_1 R \\ \alpha E - (\mu + \delta + \tau) \end{bmatrix} \quad \text{And} \quad G(M, N) = \begin{bmatrix} \Phi S - (\mu + \alpha)E + \pi_2 R \\ \alpha E - (\mu + \delta + \tau) \end{bmatrix} \quad (14)$$

With $G(M, 0) = 0$, and $E^0 = (M^0, 0)$ which represent IFE point of the sub-system

The conditions U_1 and U_2 needs to be verify for global stability to hold for system (1)

$$U_1 = \left(\frac{dM}{dt} \right) = F(M, 0) \text{ and } U_2 = G(M, N) = [BC - G^0(M, N) \geq 0 \forall \in \mathbb{R}_+^4]$$

Where B is a matrix which is diagonal entries are negative when differentiated with respect to the elements in matrix C, where $C = \begin{bmatrix} E \\ I \end{bmatrix}$, let's consider the reduced system U_1 i.e. where the infected component equal zero

$$U_1 = F(M, 0) = \begin{bmatrix} \Lambda - \mu S + \pi_1 R \\ -\mu R - (\pi_1 + \pi_2)R \end{bmatrix}, \quad M^* = (S, R) = \left(\frac{\Lambda}{\mu}, 0 \right) \text{ is the global asymptotic stability point for}$$

the reduced system U_1 , meanwhile U_1 has been verified earlier using the (Mittag-Leffler) function for positivity of solution hence the first condition holds

For U_2 , we have that

$$B = \begin{bmatrix} -(\mu + \alpha) & 0 \\ \alpha & -(\mu + \delta + \tau) \end{bmatrix}, \quad G^0(M, N) = \begin{bmatrix} \Phi(\frac{\Lambda}{\mu} - S) \\ \mu \\ 0 \end{bmatrix} \quad (15)$$

We have been able to establish the global convergence (stability) of our proposed model.

Numerical solutions

Using finite difference scheme, the set of differential equation used can be solved numerically to obtain numerical values

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda - \Phi S - \mu S + \pi_1 R \\ \frac{dE}{dt} &= \Phi S - \mu E - \alpha E \\ \frac{dI}{dt} &= \alpha E - (\mu + \delta + \tau) I + \pi_2 R \\ \frac{dR}{dt} &= \tau I - \mu R - \pi_1 R - \pi_2 R \end{aligned} \right\} \quad (16)$$

Applying the numerical method and decomposing (16) we obtain;

$$\left. \begin{aligned} \frac{S_{k+1} - S_k}{h} &= \Lambda - (\Phi + \mu) S_k + \pi_1 R_k \\ \frac{E_{k+1} - E_k}{h} &= \Phi S_k - (\mu + \alpha) E_k \\ \frac{I_{k+1} - I_k}{h} &= \alpha E_k - (\mu + \delta + \tau) I_k + \pi_2 R_k \\ \frac{R_{k+1} - R_k}{h} &= \tau I_k - (\mu + \pi_1 + \pi_2) R_k \end{aligned} \right\} \quad (17)$$

For ease of computation (17) can alternatively give as

$$\left. \begin{aligned} S_{k+1} &= S_k + (\Lambda - (\Phi + \mu) S_k + \pi_1 R_k) h \\ E_{k+1} &= E_k + (\Phi S_k - (\mu + \alpha) E_k) h \\ I_{k+1} &= I_k + (\alpha E_k - (\mu + \delta + \tau) I_k + \pi_2 R_k) h \\ R_{k+1} &= R_k + (\tau I_k - (\mu + \pi_1 + \pi_2) R_k) h \end{aligned} \right\} \quad (18)$$

Where h is the step size

Results

Numerical simulation

The values in Table 1 are used in the numerical simulation of the proposed modified model using MATLAB software with initial positive conditions. Susceptible = 1000, Exposed = 550, infected = 250, Recovered = 50. Are all assumed values.

Table 1: Parameters and Biological description

Parameters	Description	Values	Sources
Λ	Rate of human recruitment	0.20	[2]
Φ	Incident rate	0.5	Assumed
μ	rate of natural death	0.003465	[2]
α	Rate of progression from exposed to infectious	0.091193	[2]
τ	Rate of progression from infected to recover	0.030	[2]
δ	Disease induced death	0.0090	[3]
π_1	Rate of relapse	0-1 (calibrated)	Assumed
π_2	Rate of re-infection	0-1 (calibrated)	Assumed
β	Rate of contact	0.080	[3]

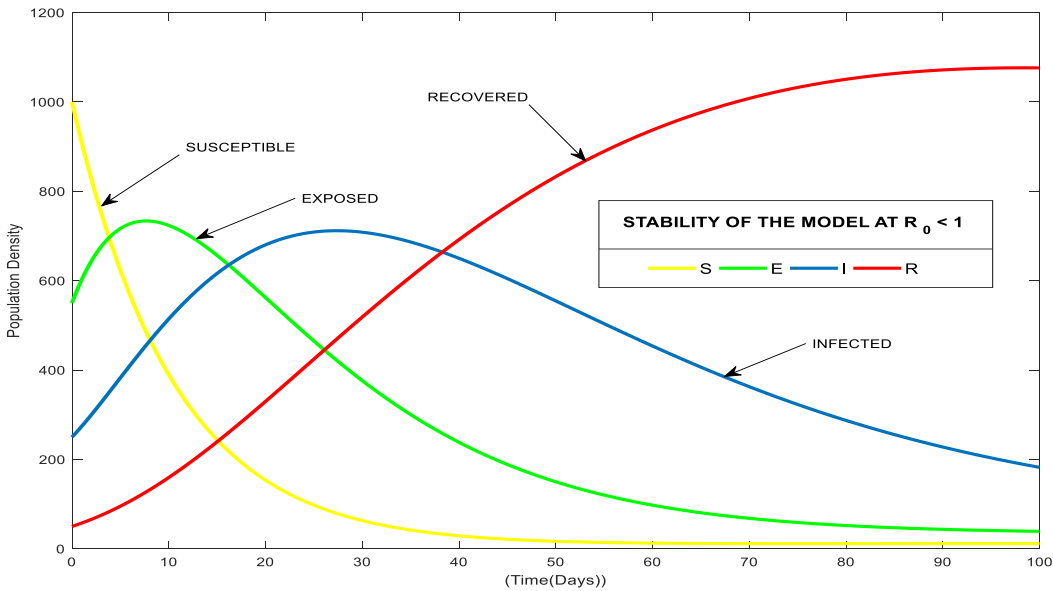


Figure 2.

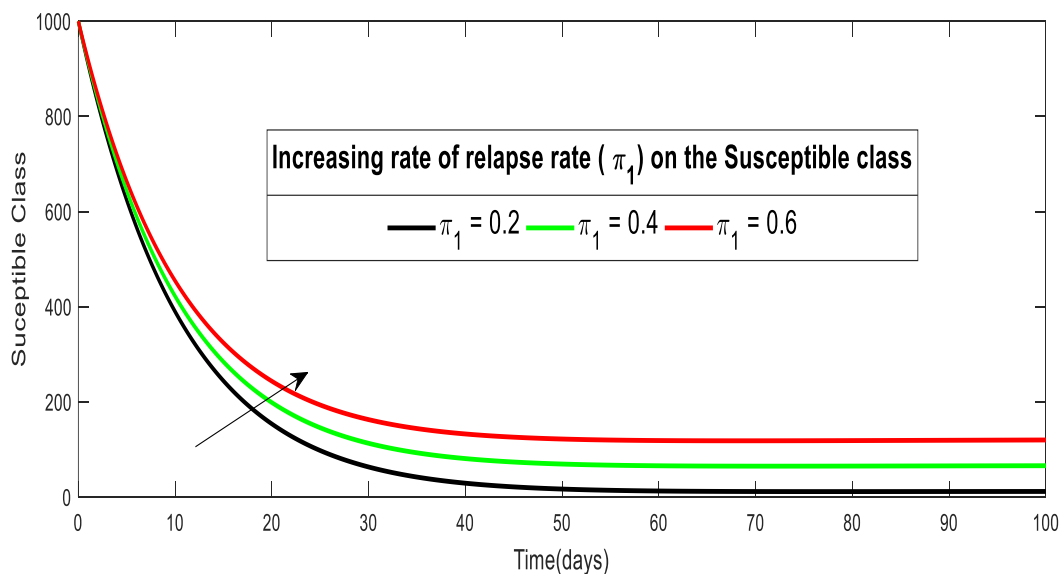


Figure 3

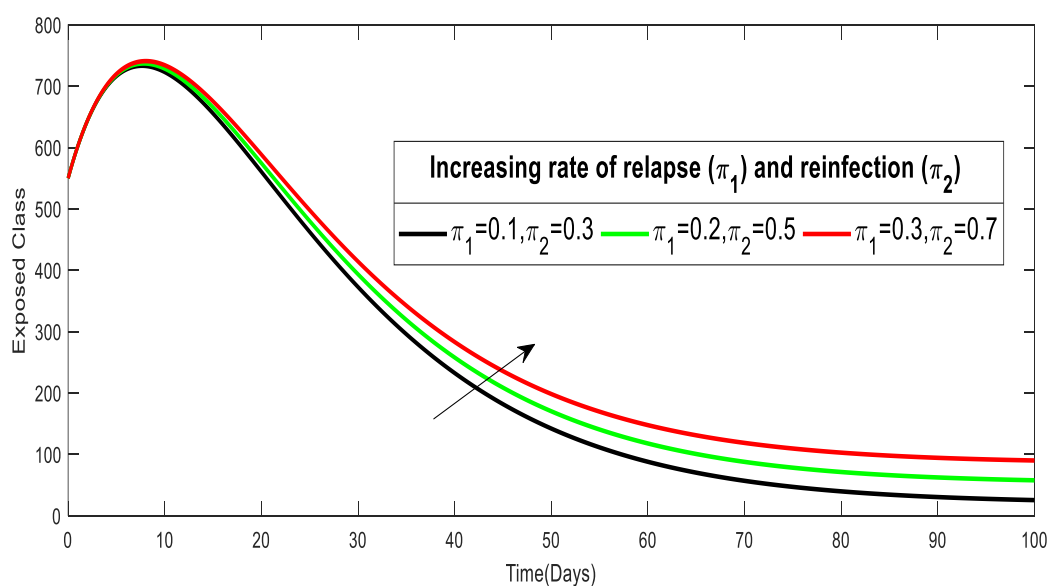


Figure 4

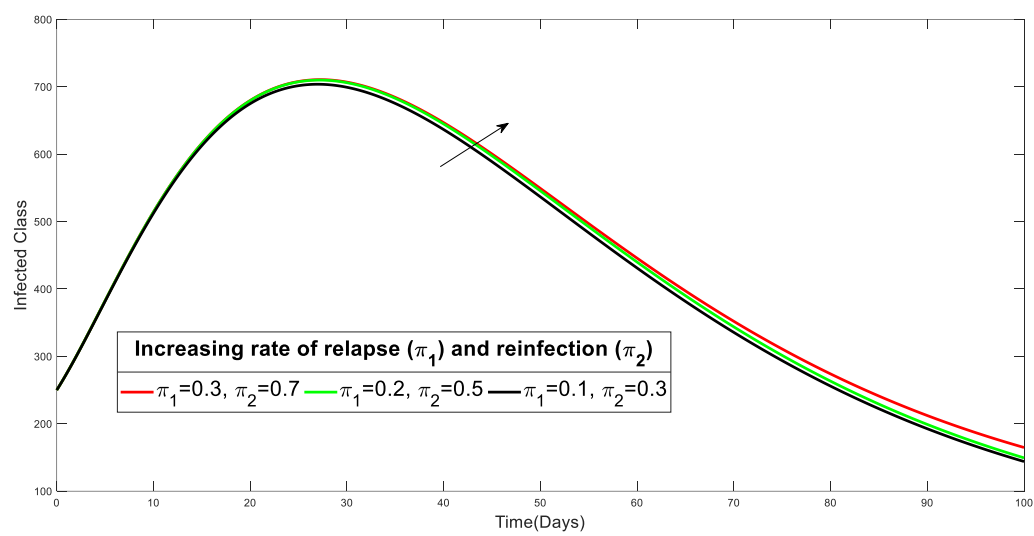


Figure 5

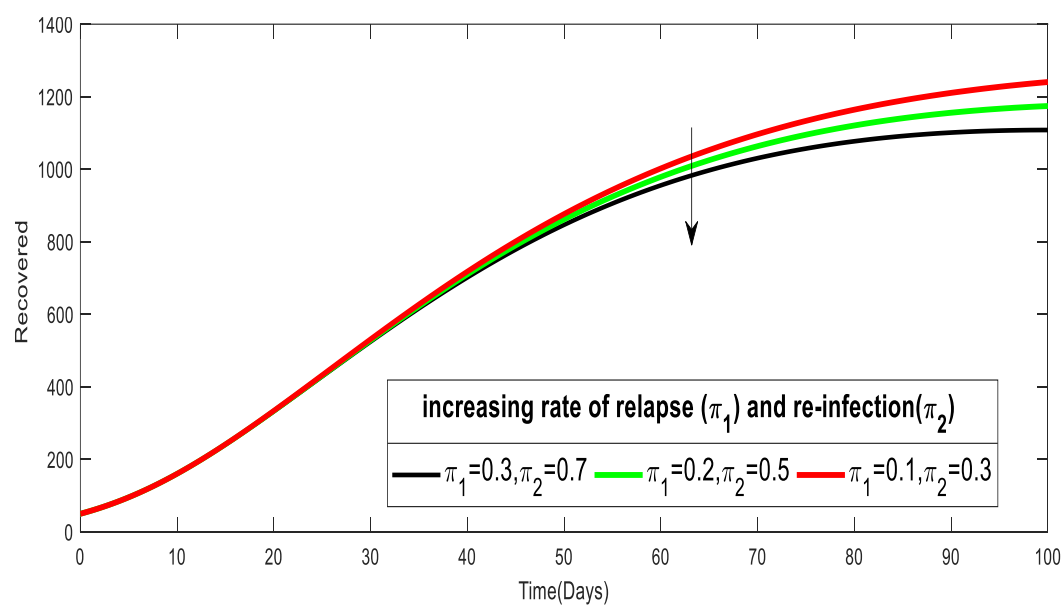


Figure 6

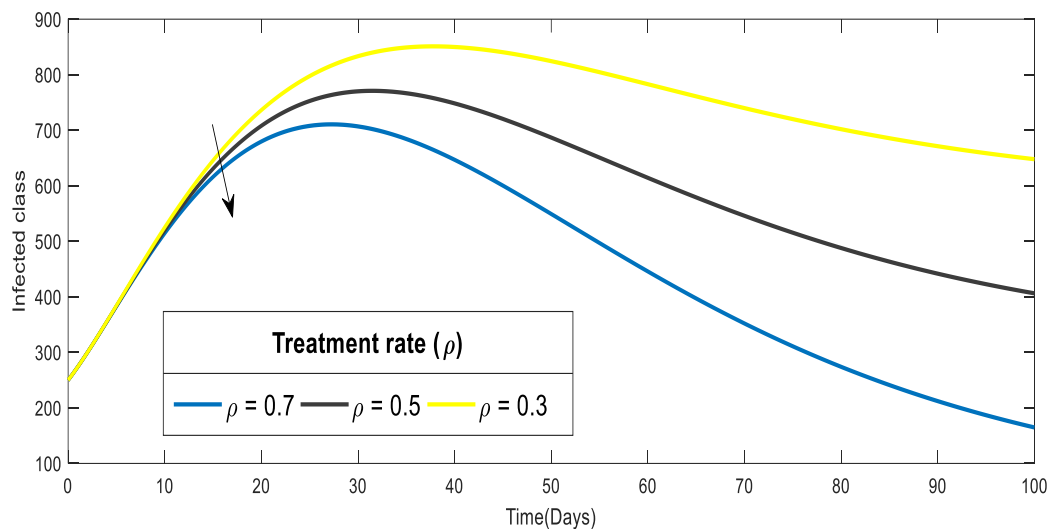


Figure 7

Discussion

The model stability for the reproduction number less than in unity (1) is demonstrated in figure 2, depicting the real life situation and mathematical accuracy of the proposed model. The center for disease control and prevention (CDC) and world health organization (WHO) predicts that there is possibility for COVID-19 to resurface in human population.

Figure 3-6's curves illustrate how this possibility is supported by data, demonstrating how "relapse and re-infection rate" might impact the population dynamics. It has been noted that a variety of factors may render a person who has recovered from the corona virus vulnerable, or even instantly contagious, for instance globules from an infected person which are often transmitted by cough or sneezing and not adhering to preventative measures. The consequences of ignoring the corona virus preventative procedures are shown in the aforementioned Figures 3–6. It instructs the recovered folks to refute the irrational assumption in a straightforward manner. In doing so, the disease will rapidly reach the point of disease free system, which is the aim of this study.

In Figure 3 the constant increase in the relapse rate in the susceptible class was demonstrated and its resultant effect could be felt in Figures 4 and 5 which makes it clear that the infectious and the exposed individuals will progressively grow if recovered individuals disregard or recklessly disregard the recommended preventive actions. As a result, the curves would get closer to the endemic state. The fact that there would be no evidence of a decline in the recovered class if there were constant "relapse and re-infection" in the population, as confirmed by Figure 6.

The dynamical effect of promptly treating an infectious person is shown in Figure 7. It is recommended that governments and healthcare professionals take all necessary steps to expedite the treatment of coronavirus in various medical facilities. As long as the rate of relapse and re-infection is low or nonexistent, these should slow the spread of the disease. It is shown that the infectious curve would quickly flatten out after 100 days if the treatment rate ρ was substantially raised to 0.7 in the first instance. After 100 days, there wouldn't be any records of the disease in the afflicted population if everyone assumed responsibility for doing all the preventive measures. This implies that each contagious person will recover, as seen in Figure 7.

Conclusion

This study created a modified dynamical model. The needed properties like existence, uniqueness, boundedness and positivity (non-negativity) of the solution were obtained and consistently proved to be true analytically. Simultaneously, a comparison analysis was carried out which prompted the derivation of equilibrium points, demonstrating the presence and the steady state of the equilibriums, and utilizing the next generation matrix

approach to calculate the reproduction number, in which we are able to verify that the reproduction number R_0 is less than unity (1) from the stability analysis. Additionally the sensitivity analysis was carried out to determine the parameters that influence the rate of reproduction of the disease. The finite different scheme method was used in solving the set of differential equations used. The graphs curves provide a brief overview of potential risks that could arise from continuing to operate under the erroneous assumption. We also showed the impact of promptly treating contagious Individuals. Numerical validation and analyses of the proposed model showed that the practical method of lowering the propagation dynamics of the virus is by carefully implementing all preventative techniques.

References

- Annas, S., Pratama, M. I., Rifandi, M., & Sanusi, W. (2020). Stability analysis and numerical simulation of SEIR model for pandemic COVID-19 spread in Indonesia. *Chaos, Solitons & Fractals*, 139 (1). <https://doi.org/10.1016/j.chaos.2020.110072>.
- Atangana, A., & Araz, S.İ. (2022). Fractional stochastic differential equations: applications to covid-19 modeling. *Springer Nature*. <http://dx.doi.org/10.1007/978-981-19-0729-6>
- Batista, M. (2020). Estimation of the final size of the coronavirus epidemic by SIR model, *Research Gate*.
- Chen, T. M., Rui, J., Wang, Q. P., Zhao, Z. Y., Cui, J. A., & Yin, L. (2020). A mathematical model for simulating the phase-based transmissibility of a novel coronavirus. *Infectious Diseases of Poverty*, 9. <https://doi.org/10.1186/s40249-020-00640-3>
- Diekmann, O., Heesterbeek, J. A. P., & Metz, J. A. J. (1990). On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations. *J. Math. Biol.*, 28 (1990), 365–382. DOI, doi: 10.1007/BF00178324.
- Din, A., Li, Y., Khan, T., & Zaman, G. (2020). Mathematical analysis of spread and control of The novel corona virus (COVID-19) in China. *Chaos Solitons Fractals.*; 141, 110286. <https://doi.org/10.1016/j.chaos.2020.110286>.
- Eikenberry, S.E., Mancuso, M., Iboi, E., Phan, T., Eikenberry, K., Kuang, Y., Kostelich, E., & Gumel, A.B. (2020). To mask or not to mask: Modeling the potential for face mask use by the general public to curtail the COVID-19 pandemic. *Infect Dis Model*. 5,293–308.
- Gbodogbe, S. O. (2025). Harmonizing epidemic dynamics: A fractional calculus approach to optimal control strategies for cholera transmission. *Scientific African*, 27. <https://doi.org/10.1016/j.sciaf.2025.e02545>
- Hoehl, S., Rabenau, H., Berger, A., Kortenbusch, M., Cinatl, J., & Bojkova. (2020). Evidence of
- Huang, C., Wang, Y., Li, X., Ren, L., Zhao, J., & Hu, Y. (2020). Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *The Lancet*, 395(10223), 497–506.
- Idowu, O. K., & Loinmi, A. C. (2023). Qualitative analysis of the transmission dynamics and optimal control of covid-19. *EDUCATUM Journal of Science, Mathematics and Technology*, 10 (1), 54-70. <https://doi.org/10.37134/ejsmt.vol10.1.7.2023>.
- Iqbal, N., & Y. Karaca, Y. (2021). Complex fractional-order HIV diffusion model based on amplitude equations with Turing patterns and Turing instability, *Fractals*, 29(5), article 2140013
- Khan, M. A., & Atangana, A. (2020). Mathematical modeling and analysis of COVID-19: A study of new variant Omicron. *Physica A: Statistical Mechanics and its applications*. 599, 127452..<https://doi.org/10.1016/j.physa.2022.127452>
- Li, Q., Guan, X., Wu, P., Wang, X., Zhou, L., & Tong, Y. (2020). Early transmission dynamics in Wuhan, China, of novel coronavirus-infected pneumonia. *New Engl J Med* 382, 1199–1207. <http://dx.doi.org/10.1056/NEJMoa2001316>.
- Liu, Y., Gayle, A. A., Wilder-Smith, A., & Rocklöv, J. (2020). The reproductive number of COVID-19 is higher compared to SARS coronavirus. *Journal of travel medicine*, 27(2).
- Loinmi, A. C. (2024). A fractional-order model for Zika virus transmission dynamics: analysis, control strategies, and simulation insights. *FNAS Journal of Scientific Innovations*, 6(1), 84-108.
- Loinmi, A. C., & Ijaola, A. L. (2024a). Investigating the Effects of Some Controls measures on the Dynamics of Diphtheria Infection using Fractional Order Model. *Mathematics and Computational Sciences*, 5(4), 26-47. [10.30511/MCS.2024.2032110.1183](https://doi.org/10.30511/MCS.2024.2032110.1183).
- Loinmi, A. C., & Ijaola, A. L. (2024b). Fractional order model of dynamical behavior and qualitative analysis of Anthrax with infected vector and saturation. *Int. J of Math. Anal. And Modelling*, 7(2), 224-264.
- Loinmi, A. C., Ajala, A. S., & Alani, L. I. (2024). Analysis of the effect of vaccination, efficient surveillance and treatment on the transmission dynamics of cholera. *Al-Bahir journal for Engineering and Pure Sciences*, 5(2), 94–107. <https://doi.org/10.55810/2313-0083.1070>
- Loinmi, A. C., Gbodogbe, S. O., & Idowu, K. O. (2023). On the interaction of the human immune system with foreign body: mathematical modeling approach. *Kathmandu University Journal of Science, Engineering and Technology*, 17 (2), 1-17. <https://journals.ku.edu.np/kuset/article/view/137>

- Musa, S.S., Zhao, S., Wang, M.H., Habib, A.G., Mustapha, U.T., & He, D. (2020). Estimation of exponential growth rate and basic reproduction number of the coronavirus disease 2019 (COVID-19) in Africa. *Infect Dis Poverty*, 9(96). <http://dx.doi.org/10.1186/s40249-020-00718-y>.
- Ndaïrou, F., Area, I., Nieto, J. J., & Torres, D. F. (2020). Corrigendum to "Mathematical modeling of COVID-19 transmission dynamics with a case study of Wuhan" [*Chaos Solitons Fractals* 135 (2020), 109846].," *Chaos, Solitons and Fractals*, 141(1). <https://doi.org/10.1016/j.chaos.2020.110311>.
- Olumuyiwa, J. P., Qureshi, S., Yusuf, A., Al-Shomrani, M., & Abioye Idowu, A. (2021). A new mathematical model of COVID-19 using real data from Pakistan. *Results in Physics*, 24, article 104098. <https://doi.org/10.1016/j.rinp.2021.104098>
- Pauline, V. D., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*, 180(1), 29–48.
- Pauline, V. D., & Watmough, J. (2008). Further notes on the basic reproduction number. *Mathematical epidemiology*, 159–178.
- Rahman, J.U.I., Lu, D., Suleman, M., He, J. H., & Ramzan, M. (2019). He-Elzaki method for spatial diffusion of biological population. *Fractals*, 27(5),
- Suleman, M., Lu, D., He, J. H., Farooq, U., Hui, Y. S., & Rahman, J. U. (2018). Numerical investigation of fractional HIV model using Elzaki projected differential transform method. *Fractals*, 26(5), article 1850062.
- Wai-Kit, M., Huang, J., & Zhang, C.J.P. (2020). Breaking down of the healthcare system: mathematical modelling for controlling the novel coronavirus (2019-nCoV) outbreak in Wuhan, China. *Preprints*. doi: <https://doi.org/10.1101/2020.01.27.922443>.
- Zhao, S., Lin, Q., & Ran, J. (2020). Preliminary estimation of the basic reproduction number of novel coronavirus (2019-nCoV) in China, from 2019 to 2020: A data-driven analysis in the early phase of the outbreak. *International journal of infectious diseases*, 92, 214–217. DOI: [10.1016/j.ijid.2020.01.050](https://doi.org/10.1016/j.ijid.2020.01.050)
- Zhu, N., Zhang, D., Wang, W., Li, X., Yang, B., & Song, J. (2020). A novel coronavirus from patients with pneumonia in China. 2019. *New Engl. J. Med.*, 382, 727–33. DOI: [10.1056/NEJMoa2001017](https://doi.org/10.1056/NEJMoa2001017)