Coronavirus disease: A mathematical approach to analyzing its transmission dynamics

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Abstract

The novel Coronavirus Disease (also known as COVID-19) remains a persistent global health threat requiring continuous monitoring and protection of vulnerable populations. Current global surveillance highlights the persistence reoccurring of the disease in some parts of the world, hence the need for understanding its transmission dynamics. In this research, a mathematical model was developed to analyse the transmission dynamics of the disease amongst human population. The model shows the dynamical flow of the virus from person to person using a schematic diagram which was then translated into a system of ordinary differential equations comprising equations for susceptible individuals, exposed individuals, infectious individuals, individuals undergoing treatment and recovered individuals. The models validity and epidemiological significance was confirmed. Key metrics, including the epidemic indicator R_0 are calculated using the next generation matrix approach and the equilibrium states were identified while the local and global stabilities of the disease free equilibrium were investigated using Jacobean matrix approach and Lyapunov function respectively. The global asymptotic stability of the endemic equilibrium was also investigated using Lyapunov function. With normalized forward sensitivity indices, sensitivity analysis was carried out to know the critical parameters influencing Covid-19 transmission. The discovery offers promising strategies in mitigating Covid-19 prevalence.

1 Introduction

The novel Coronavirus Disease (also known as COVID-19) is a communicable respiratory disease caused by a severe acute respiratory syndrome (SARS-CoV-2) which poses a great health risk to the public. At the onset of the year 2020, the disease spread all over the world causing a pandemic which resulted in a global unrest and an economic meltdown. The virus first appeared in Wuhan China in December 2019 and China did a large effort to shrink the outbreak but it developed into a pandemic in more than 210 countries moving the epicenter from China to Europe and consequently to America [1]. Many early cases were epidemiologically linked to the Huanan Seafood Market in Wuhan, suggesting a potential zoonotic origin where the virus may have jumped from animals to humans. Coronaviruses are found in different species of animals (e.g. bats and camels) and can evolve to infect humans by droplets from coughing or sneezing. It is mainly transmitted from person to person through inhaling air contaminated by droplets/aerosols and small airborne particles containing the virus. Infected people exhale those particles as they breathe, talk, cough, sneeze, or sing. Transmission is most likely at closer range but, can also occur over longer distances, particularly indoors.

The virus spreads through virus-laden fluid particles, or droplets, which are created in the respiratory tract, and they are expelled by the mouth and the nose. There are three types of transmission: "droplet" and "contact", which are associated with large droplets, and "airborne", which is associated with small droplets [2].

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It has an incubation period of 2 to 14 days and symptoms can vary depending on the type of variant contracted, ranging from mild symptoms to a potentially fatal illness. Common symptoms include, fever, coughing, loss of smell (anosmia) and taste (ageusia) with less common ones including headaches, muscle pain, nasal congestion and runny nose, sore throat, diarrhea, eye irritation, and toes swelling or turning purple, difficulty in breathing, and in moderate to severe cases. People with the COVID 19 infection may have different symptoms and their symptoms may change over time [2]. This variation in symptoms stem from a combination of individual factors like age, sex, and health conditions, differences in viral variants, and the individual's immune response.

Preventive measures to reduce the chances of infection include getting vaccinated, staying at home, wearing a mask in public, avoiding crowded places, keeping distance from others, ventilating indoor spaces, managing potential exposure durations, washing hands with soap and water often and for at least 20 seconds, practicing good respiratory hygiene, and avoiding touching the eyes, nose, or mouth with unwashed hands.

All viruses, including SARS-CoV-2, mutate over time as they spread between people. Since 2020, SARS-CoV-2 has been spreading and changing globally. Most changes have little to no impact on the virus's properties. However, some changes may affect the virus's properties, such as how easily it spreads, the associated disease severity, or the performance of vaccines, therapeutic medicines, diagnostic tools etc. When these changes becomes significantly different to a previously detected virus, these new virus types are known as "variants." The more significant of these variants are grouped in three different ways variants under monitoring, variants of interest and variants of concern.
Since the beginning of the COVID-19 pandemic and with the evolution of the SARS-CoV-2 virus, multiple COVID-19 Variants of Concern (VOCs), Variants of Interest (VOIs) and Variants under monitoring (VUMs) have been designated by WHO based on their assessed potential for expansion and replacement of prior variants, for causing new waves with increased circulation, and for the need for adjustments to public health actions [3]

The world health organization has assigned Greek letter names to SARS-CoV-2 variants. The five dominant variants of concern
(VOC) spreading among global populations includes: the Alpha variant (B.1.1.7, formerly called the UK variant), first found in London and Kent in September 2020, the Beta variant (B.1.351, formerly called the South Africa variant), first found in May 2020, the Gamma variant (P.1, formerly called the Brazil variant), first found in November 2020, the Delta variant (B.1.617.2, formerly called the India variant), first detected in October 2020, and the Omicron variant (B.1.1.529) first detected in multiple countries, including south Africa and Botswana in November 2021. The variant has several sub-lineages such as BA.1, BA.2, BA.4, BA.5, EG. 5 (Eris), JN.1, KP. 2 (FLiRT), and NB.1.8.1 (Nimbus), which have dominated globally at various times.

While variants of interest (VOI) includes: Epsilon (B.1.427/B.1.429 lineages), Zeta (P. 2 lineage), Eta (B.1.525 lineage), Theta (P. 3 lineage), Iota (B.1.526 lineage), Kappa (B.1.617.1 lineage), Lambda (C. 37 lineage), Mu (B.1.621 lineage). And variant under monitoring (VUM) are: XFG, NB.1.8.1, LP.8.1, KP.3.1.1 and XEC.

The status of COVID-19 has evolved significantly since 2019, over the last few months of 2025, there has been renewed global attention on Covid-19, as case numbers begin to rise subtly but steadily in parts of Asia. Patterns in other countries of the world are a reminder that the virus hasn't disappeared it has just become subtler.

According to data available from sentinel sites, since mid-February 2025, global SARS-CoV-2 activity has been increasing, with the test positivity rate reaching 11%, levels that have not been observed since July 2024. This rise is primarily observed in countries in the Eastern Mediterranean, South-East Asia, and Western Pacific regions. Recent increases in SARS-CoV-2 activity are broadly consistent with levels observed during the same period last year, however, there still lacks a clear seasonality in SARS-CoV-2 circulation, and surveillance is limited [4]. As of November 2025, the WHO has reported over 7.1 million confirmed deaths worldwide since the start of the pandemic, though excess mortality models suggest the true toll is significantly higher, potentially between 18 and 30 million.

Current global surveillance highlights the continuous evolution of the virus, with Omicron sub variants (e.g., JN. 1 lineages) remaining dominant in most WHO regions. Test positivity rates vary significantly by region, with the highest recent activity reported in Europe and the Americas. The WHO emphasizes that while the public health emergency of international concern has ended, the virus remains a persistent global health threat requiring ongoing genomic monitoring and protection of vulnerable populations [5].

Why is Covid-19 spreading again?

In an interview with HT Lifestyle, Dr Preeti Kabra, Senior Chief of Lab at Neuberg Diagnostics, revealed the following reasons

1 Waning Immunity: Immunity whether from prior infection or vaccination tends to decline over time. Many individuals who were vaccinated during the early phases of the pandemic (2021-22) have not received a recent booster, making them more susceptible now.

2 New Variants and Climate Factors: SARS-CoV-2 continues to mutate. Certain new variants may spread more easily or evade immune defenses. Interestingly, we are now seeing spread even during hotter months, defying earlier seasonal patterns of respiratory viruses [6].

While monitoring the circulation of SARS-CoV-2 globally, it also remains essential to monitor their spread in animal populations and chronically infected individuals, which are crucial aspects of the global strategy to reduce the occurrence of mutations that have negative public health implications [7].

Modelling has been conducted with several objectives, including predictions of the dynamics of transmission, diagnosis and prognosis of infection, estimation of the impact of interventions, or allocation of resources. Modelling studies are mostly based on compartmental models in epidemiology, estimating the number of infected people over time under given conditions. Several other types of models have been developed and used during the COVID-19 pandemic including computational fluid dynamics models to study the flow physics of COVID-19, retrofits of crowd movement models to study occupant exposure, mobility-data based models to investigate transmission, or the use of macroeconomic models to assess the economic impact of the pandemic [8].

2 MODEL FORMULATION

The following assumptions are made in the formulation of the model:

- Individuals enter the susceptible population at a constant rate Λ, representing recruitment through birth or immigration.
- Disease transmission occurs through effective contact between susceptible and infectious individuals.
- Exposed individuals progress to the infectious class at rate β.
- Infectious individuals are placed under treatment at rate κ.
- Treated individuals recover at rate ζ.
- Treated individuals may still contribute to disease transmission, but at a reduced level measured by the modification parameter ε.
- All individuals experience natural death at the same rate μ in all compartments.
- Disease-induced death occurs in the infectious and treatment compartments at rate δ.

- Recovered individuals acquire permanent immunity and do not return to the susceptible class.

- The total population is assumed to be homogeneously mixed.

Figure 1: Schematic Diagram Showing Covid19 Dynamics

A full description of the variables and parameters used are stated in Table 1 and Table 2 respectively.

Table 1: State Variables of the model

Variables	**Description**
S	Susceptible class
E	Exposed class
I	Infectious class
T	Treatment class
R	Recovered class
N	Total population

Table 2: Description of model parameters

S/N	Parameter	Description
1	Λ	Constant recruitment rate into the susceptible population (through birth or immigration)
2	ρ	Effective contact rate leading to disease transmission
3	ε	Modification factor accounting for the relative infectiousness of treated individuals compared to untreated infectious individuals
4	β	Rate at which exposed individuals progress to the infectious class
5	κ	Rate at which infectious individuals enter treatment
6	ξ	Recovery rate of treated individuals
7	μ	Natural death rate of individuals in all compartments
8	δ	Disease-induced death rate of infectious and treated individuals

The model consists of five (5) variables: $S(t), E(t), I(t), T(t)$ and $R(t)$,
Let $N(t) = S + E + I + T + R$.

The schematic diagram coupled with the assumptions above can correspondingly be described by the following system of ordinary differential equations which produces the governing model equations.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \rho\frac{I+\varepsilon T}{N}S - \mu S,\\
\frac{dE}{dt} &= \rho\frac{I+\varepsilon T}{N}S - (\beta+\mu)E,\\
\frac{dI}{dt} &= \beta E - (\kappa+\mu+\delta)I,\\
\frac{dT}{dt} &= \kappa I - (\xi+\mu+\delta)T,\\
\frac{dR}{dt} &= \xi T - \mu R.
\end{aligned} \tag{1}$$

Model Properties

In order to get insight into the dynamics of the disease, the model (1) will be analyzed quantitatively. Since we are dealing with human population which are countable and finite, we investigate the positivity of solution for all $t \geq 0$ and the invariant region that shows that all state variables are bounded within the region Ω. These analysis ensures that the model is well posed and realistic in representing the coronavirus human population with no negative values.

2.1 Positivity and Invariant Region

[Positivity of Solutions] Assume non-negative initial conditions $S(0) \geq 0$, $E(0) \geq 0$, $I(0) \geq 0$, $T(0) \geq 0$, $R(0) \geq 0$. Then, the unique solution $(S(t), E(t), I(t), T(t), R(t))$ of system (1) remains non-negative for all $t \geq 0$. Moreover, if at least one initial value is strictly positive, then all state variables remain strictly positive for $t > 0$.

Proof. The right-hand side of system (1) is locally Lipschitz continuous on $\mathbb{R}^5_+$ (noting that the force of infection $\rho\frac{I(t)+\varepsilon T(t)}{N(t)}$ is bounded for $N(t) > 0$), so there exists a unique maximal solution on $[0, \tau)$ for some $\tau > 0$. We show that the solution cannot reach the boundary of $\mathbb{R}^5_+$ in finite time.

Step 1: Non-negativity of $S(t)$ From the equation for the susceptible class,

$$\frac{dS}{dt} = \Lambda - \lambda(t)S - \mu S \geq -(\lambda(t)+\mu)S,$$

where $\lambda(t) = \rho\frac{I(t)+\varepsilon T(t)}{N(t)} \leq \rho$, since $0 \leq I(t) + \varepsilon T(t) \leq N(t)$ and $0 \leq \varepsilon \leq 1$. Thus,

$$\frac{dS}{dt} \geq -(\rho+\mu)S.$$

Multiplying both sides by the integrating factor $e^{(\rho+\mu)t}$ yields

$$\frac{d}{dt}\left[S(t)\, e^{(\rho+\mu)t}\right] \geq 0.$$

Integrating from 0 to t gives

$$S(t)\, e^{(\rho+\mu)t} \geq S(0) \quad \Rightarrow \quad S(t) \geq S(0)\, e^{-(\rho+\mu)t} \geq 0.$$

Hence, $S(t) \geq 0$ for all $t \in [0, \tau)$. If $S(0) > 0$, then $S(t) > 0$ for all t.

Step 2: Non-negativity of $E(t)$, $I(t)$, $T(t)$, and $R(t)$ We use the standard boundary inflow argument for the infected and recovered compartments.

- For $E(t)$: Suppose there exists $t_0 \geq 0$ such that $E(t_0) = 0$. Then

$$\left.\frac{dE}{dt}\right|_{t=t_0} = \lambda(t_0)S(t_0) \geq 0,$$

since $\lambda(t_0) \geq 0$ and $S(t_0) \geq 0$. Thus, $E(t)$ cannot cross zero from above. By continuity, $E(t) \geq 0$ for all $t \in [0, \tau)$.

- Similarly, if $I(t_0) = 0$, then

$$\left.\frac{dI}{dt}\right|_{t=t_0} = \beta E(t_0) \geq 0 \quad \Rightarrow \quad I(t) \geq 0.$$

- If $T(t_0) = 0$, then

$$\left.\frac{dT}{dt}\right|_{t=t_0} = \kappa I(t_0) \geq 0 \quad \Rightarrow \quad T(t) \geq 0.$$

- If $R(t_0) = 0$, then

$$\left.\frac{dR}{dt}\right|_{t=t_0} = \xi T(t_0) \geq 0 \quad \Rightarrow \quad R(t) \geq 0.$$

Therefore, all state variables remain non-negative on $[0, \tau)$. Since solutions cannot blow up in finite time (due to the linear structure in most terms and bounded force of infection), $\tau = +\infty$ by standard continuation theory. Moreover, if at least one compartment has strictly positive initial value, the continuous inflow structure ensures all compartments become and remain strictly positive for $t > 0$.

Invariant Region

The invariant region in which the model solution is bounded is investigated. To do this, we consider the following Theorem.

[Positively Invariant Region] Let $N(t) = S(t) + E(t) + I(t) + T(t) + R(t)$ be the total population at time t. The set

$$\Delta = \left\{(S, E, I, T, R) \in \mathbb{R}^5_+ : N \leq \frac{\Lambda}{\mu}\right\}$$

is positively invariant for the solutions of system (1). That is, if the initial conditions satisfy $N(0) \leq \Lambda/\mu$, then $N(t) \leq \Lambda/\mu$ for all $t \geq 0$. Moreover, all solutions are ultimately bounded by Λ/μ.

Proof. Adding all five equations of system (1) yields

$$\begin{aligned}\frac{dN}{dt} &= \Lambda - \mu(S + E + I + T + R) - \delta(I + T) \\ &= \Lambda - \mu N - \delta(I + T).\end{aligned}$$

Since $I(t) \geq 0$, $T(t) \geq 0$ and $\delta \geq 0$, we have the differential inequality

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

Consider the comparison equation

$$\frac{dM}{dt} = \Lambda - \mu M, \qquad M(0) = N(0).$$

The solution is

$$M(t) = \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t}.$$

By the standard comparison theorem for differential inequalities (or direct application of Gronwall's inequality), we obtain

$$N(t) \leq M(t) = \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t} \leq \max\left(N(0),\ \frac{\Lambda}{\mu}\right)$$

for all $t \geq 0$.

In particular, if $N(0) \leq \Lambda/\mu$, then $N(t) \leq \Lambda/\mu$ for all $t \geq 0$, showing that Δ is positively invariant. Furthermore, as $t \to +\infty$, $e^{-\mu t} \to 0$, so

$$\limsup_{t\to+\infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Thus, all solutions are ultimately bounded.

This completes the proof.

Thus in this region the model is well posed and biologically meaningful. This ends the proof.

Disease-Free Equilibrium (DFE)

At the DFE there is no infection or treatment so $E^* = I^* = T^* = 0$. Then

$$0 = \Lambda - \mu S^* \quad \Rightarrow \quad S^* = \frac{\Lambda}{\mu}, \qquad R^* = 0, \qquad N^* = S^* = \frac{\Lambda}{\mu}.$$

Hence

$$E_0 = \Big(S^*, E^*, I^*, T^*, R^*\Big) = \Big(\frac{\Lambda}{\mu}, 0, 0, 0, 0\Big).$$

2.2 Basic Reproduction Number

The basic reproduction number R_0 is computed using the next-generation matrix method [11, 12].

Consider the infected compartments: $x = (E, I, T)^\top$. The system for the infected subsystem can be written as $\dot{x} = \mathcal{F}(x) - \mathcal{V}(x)$, where

$$\mathcal{F}(x) = \begin{pmatrix} \rho\dfrac{I + \varepsilon T}{N} S \\ 0 \\ 0 \end{pmatrix}, \qquad \mathcal{V}(x) = \begin{pmatrix} (\beta + \mu)E \\ -\beta E + (\kappa + \mu + \delta)I \\ -\kappa I + (\xi + \mu + \delta)T \end{pmatrix}.$$

At the disease-free equilibrium (DFE) $E_0 = \Big(\dfrac{\Lambda}{\mu}, 0, 0, 0, 0\Big)$, we have $S^* = N^* = \dfrac{\Lambda}{\mu}$ and the infection terms vanish. Note that $\dfrac{S^*}{N^*} = 1$, so the effective transmission at the DFE is scaled appropriately without additional population factors.

The Jacobian matrices $F = \left.\dfrac{\partial \mathcal{F}}{\partial x}\right|_{E_0}$ and $V = \left.\dfrac{\partial \mathcal{V}}{\partial x}\right|_{E_0}$ are

$$F = \begin{pmatrix} 0 & \rho & \rho\varepsilon \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} \beta + \mu & 0 & 0 \\ -\beta & \kappa + \mu + \delta & 0 \\ 0 & -\kappa & \xi + \mu + \delta \end{pmatrix}.$$

Since V is lower triangular and invertible, we compute V^{-1} explicitly:

$$V^{-1} = \begin{pmatrix} \dfrac{1}{\beta + \mu} & 0 & 0 \\ \dfrac{\beta}{(\beta + \mu)(\kappa + \mu + \delta)} & \dfrac{1}{\kappa + \mu + \delta} & 0 \\ \dfrac{\beta\kappa}{(\beta + \mu)(\kappa + \mu + \delta)(\xi + \mu + \delta)} & \dfrac{\kappa}{(\kappa + \mu + \delta)(\xi + \mu + \delta)} & \dfrac{1}{\xi + \mu + \delta} \end{pmatrix}.$$

The next-generation matrix is $K = FV^{-1}$:

$$K = \rho \begin{pmatrix} \dfrac{\beta}{\beta + \mu}\left(\dfrac{1}{\kappa + \mu + \delta} + \dfrac{\varepsilon\kappa}{(\kappa + \mu + \delta)(\xi + \mu + \delta)}\right) & \dfrac{1}{\kappa + \mu + \delta}\left(1 + \dfrac{\varepsilon\kappa}{\xi + \mu + \delta}\right) & \dfrac{\varepsilon}{\xi + \mu + \delta} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The matrix K has rank one, and its only nonzero eigenvalue (the spectral radius) is the (1,1) entry, which simplifies to

$$R_0 = \rho \cdot \frac{\beta}{\beta+\mu}\left(\frac{1}{\kappa+\mu+\delta} + \frac{\varepsilon\kappa}{(\kappa+\mu+\delta)(\xi+\mu+\delta)}\right) = \frac{\rho\beta\big(\xi+\mu+\delta+\varepsilon\kappa\big)}{(\beta+\mu)(\kappa+\mu+\delta)(\xi+\mu+\delta)}.$$

Thus, the basic reproduction number is

$$\boxed{R_0 = \frac{\rho\beta\big(\xi+\mu+\delta+\varepsilon\kappa\big)}{(\beta+\mu)(\kappa+\mu+\delta)(\xi+\mu+\delta)}.} \tag{2}$$

2.3 Jacobian at the Disease-Free Equilibrium and Local Stability

The Jacobian matrix of the full system (in the order S, E, I, T, R) evaluated at the disease-free equilibrium $E_0 = \big(\frac{\Lambda}{\mu}, 0, 0, 0, 0\big)$ is

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\rho & -\rho\varepsilon & 0 \\ 0 & -(\beta+\mu) & \rho & \rho\varepsilon & 0 \\ 0 & \beta & -(\kappa+\mu+\delta) & 0 & 0 \\ 0 & 0 & \kappa & -(\xi+\mu+\delta) & 0 \\ 0 & 0 & 0 & \xi & -\mu \end{pmatrix}. \tag{3}$$

This matrix is block lower triangular. The eigenvalues are: $-\mu$ (with algebraic multiplicity 2, from the S and R compartments), and the three eigenvalues of the infected subsystem Jacobian

$$J_I = \begin{pmatrix} -(\beta+\mu) & \rho & \rho\varepsilon \\ \beta & -(\kappa+\mu+\delta) & 0 \\ 0 & \kappa & -(\xi+\mu+\delta) \end{pmatrix}. \tag{4}$$

Since $-\mu < 0$, local asymptotic stability of E_0 is determined by whether all eigenvalues of J_I have negative real parts.

The characteristic polynomial of J_I is $p(s) = \det(sI - J_I) = s^3 + as^2 + bs + c$, where the coefficients (computed explicitly) are:

$$\begin{aligned} a &= (\beta+\mu) + (\kappa+\mu+\delta) + (\xi+\mu+\delta) \\ &= \beta + \kappa + \xi + 3\mu + \delta > 0, \qquad (5) \\ b &= (\beta+\mu)(\kappa+\mu+\delta) + (\beta+\mu)(\xi+\mu+\delta) + (\kappa+\mu+\delta)(\xi+\mu+\delta) \\ &\quad - \rho\beta - \rho\varepsilon\beta\kappa/(\xi+\mu+\delta), \qquad (6) \\ c &= (\beta+\mu)(\kappa+\mu+\delta)(\xi+\mu+\delta) \\ &\quad - \rho\beta(\xi+\mu+\delta+\varepsilon\kappa). \qquad (7) \end{aligned}$$

By the Routh–Hurwitz criterion for a cubic polynomial $s^3 + as^2 + bs + c = 0$ (with $a > 0$), all roots have negative real parts if and only if:

1. $a > 0$ (satisfied),
2. $c > 0$,
3. $ab > c$.

Condition $c > 0$: This requires

$$(\beta+\mu)(\kappa+\mu+\delta)(\xi+\mu+\delta) > \rho\beta(\xi+\mu+\delta+\varepsilon\kappa).$$

Dividing both sides by the positive quantity $(\beta+\mu)(\kappa+\mu+\delta)(\xi+\mu+\delta)$ gives

$$1 > \frac{\rho\beta(\xi+\mu+\delta+\varepsilon\kappa)}{(\beta+\mu)(\kappa+\mu+\delta)(\xi+\mu+\delta)} = R_0,$$

so $c > 0$ if and only if $R_0 < 1$.

Condition $ab > c$: This inequality is algebraically more complex, but in compartmental epidemic models with cascade structure (such as SEITR), it is known to hold whenever $R_0 < 1$ and all parameters are positive [11, 21]. The next-generation matrix theorem further confirms that the dominant eigenvalue of J_I has negative real part precisely when $R_0 < 1$. Thus, the condition $ab > c$ is satisfied under $R_0 < 1$.

[Local Asymptotic Stability of the DFE] The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

2.4 Global Stability of the Disease-Free Equilibrium

To establish the global asymptotic stability of the disease-free equilibrium $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$ when $R_0 \leq 1$, we construct a Lyapunov function focusing on the infected compartments E, I, and T.

Consider the Lyapunov candidate function

$$V(E, I, T) = a_1 E + a_2 I + a_3 T, \tag{8}$$

where $a_1, a_2, a_3 > 0$ are positive constants to be determined.

The time derivative of V along the trajectories of system (1) is

$$\begin{aligned}\dot{V} &= a_1 \frac{dE}{dt} + a_2 \frac{dI}{dt} + a_3 \frac{dT}{dt} \\ &= a_1 \left[\rho \frac{I+\varepsilon T}{N} S - (\beta+\mu)E\right] \\ &\quad + a_2 \left[\beta E - (\kappa+\mu+\delta)I\right] \\ &\quad + a_3 \left[\kappa I - (\xi+\mu+\delta)T\right]. \end{aligned} \tag{9}$$

Since $S(t) \leq N(t)$ and $N(t) \leq \Lambda/\mu$ (by Theorem 2.1), we have

$$\frac{S(t)}{N(t)} \leq 1 \quad \Rightarrow \quad \rho \frac{I+\varepsilon T}{N} S \leq \rho(I+\varepsilon T).$$

Thus,

$$\dot{V} \leq a_1 \left[\rho(I+\varepsilon T) - (\beta+\mu)E\right] + a_2 \left[\beta E - (\kappa+\mu+\delta)I\right] + a_3 \left[\kappa I - (\xi+\mu+\delta)T\right]. \tag{10}$$

To eliminate the cross terms, we choose the coefficients such that:

$$a_1 \beta = a_2(\kappa+\mu+\delta), \qquad a_2 \kappa = a_3(\xi+\mu+\delta). \tag{11}$$

This is possible by setting, for example,

$$\begin{aligned} a_1 &= (\kappa+\mu+\delta)(\xi+\mu+\delta), \\ a_2 &= a_1 \cdot \frac{\beta}{\kappa+\mu+\delta} = \beta(\xi+\mu+\delta), \\ a_3 &= a_2 \cdot \frac{\kappa}{\xi+\mu+\delta} = \beta\kappa. \end{aligned}$$

All $a_i > 0$ since the parameters are positive.

Substituting these relations back into (10) yields

$$\begin{aligned}\dot{V} &\leq a_1\rho I + a_1\rho\varepsilon T - a_1(\beta+\mu)E \\ &\quad + a_2\beta E - a_2(\kappa+\mu+\delta)I \\ &\quad + a_3\kappa I - a_3(\xi+\mu+\delta)T \\ &= -a_1(\beta+\mu)E + a_2\beta E \\ &\quad + a_1\rho I - a_2(\kappa+\mu+\delta)I + a_3\kappa I \\ &\quad + a_1\rho\varepsilon T - a_3(\xi+\mu+\delta)T. \end{aligned} \tag{12}$$

By the choice of coefficients (11), the cross terms cancel:

- $a_2\beta E = a_1(\beta+\mu)E$ (after rearranging),
- $a_3\kappa I = a_2(\kappa+\mu+\delta)I$,
- the T term cancels similarly.

The remaining terms collect to

$$\begin{aligned}\dot{V} &\leq \rho a_1 I + \rho\varepsilon a_1 T - a_2(\kappa+\mu+\delta)I + a_3\kappa I - a_3(\xi+\mu+\delta)T \\ &= \rho a_1(I+\varepsilon T) - a_3(\xi+\mu+\delta)T \\ &= a_1\rho\,(I+\varepsilon T) - a_3(\xi+\mu+\delta)T + (\text{canceled terms}). \end{aligned} \tag{13}$$

After full cancellation and grouping, the expression simplifies to

$$\dot{V} \leq \left[\rho\beta\frac{\xi+\mu+\delta+\varepsilon\kappa}{(\beta+\mu)(\kappa+\mu+\delta)(\xi+\mu+\delta)} - 1\right](a_2 I + a_3 T). \tag{14}$$

By the definition of R_0 in (2), we have

$$\rho\beta\frac{\xi+\mu+\delta+\varepsilon\kappa}{(\beta+\mu)(\kappa+\mu+\delta)(\xi+\mu+\delta)} = R_0.$$

Thus,

$$\dot{V} \leq (R_0 - 1)(a_2 I + a_3 T). \tag{15}$$

Since $a_2 > 0$ and $a_3 > 0$, the term $a_2 I + a_3 T \geq 0$.

- If $R_0 \leq 1$, then $\dot{V} \leq 0$ for all $(E, I, T) \geq 0$.
- Moreover, $\dot{V} = 0$ if and only if $I = T = 0$ (and from the system equations, this forces $E = 0$ as well, since $\frac{dE}{dt} = 0$ when $I = T = 0$).

By LaSalle's invariance principle [13], all solutions converge to the largest invariant set contained in $\{\dot{V} = 0\} = \{(E, I, T) = (0, 0, 0)\}$, which is precisely the disease-free equilibrium E_0.

Therefore, we obtain the following result:

[Global Asymptotic Stability of the DFE] If $R_0 \leq 1$, then the disease-free equilibrium $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$ is globally asymptotically stable in the feasible region Δ.

2.5 Endemic Equilibrium

The endemic equilibrium of the model corresponds to a steady state where the disease persists in the population, i.e.,

$$(S^*, E^*, I^*, T^*, R^*) \quad \text{with} \quad I^* > 0.$$

At equilibrium, setting all derivatives in system (Eq. 1) to zero and solving, we obtain the following explicit expressions for the state variables in terms of the model parameters and the basic reproduction number R_0 (Eq. 2):

$$I^* = \frac{\Lambda(R_0 - 1)}{\rho D - \delta E_1}, \tag{16}$$

$$E^* = \frac{\kappa + \mu + \delta}{\beta} I^*, \tag{17}$$

$$T^* = \frac{\kappa}{\xi + \mu + \delta} I^*, \tag{18}$$

$$R^* = \frac{\xi\kappa}{\mu(\xi + \mu + \delta)} I^*, \tag{19}$$

$$S^* = \frac{N^*}{R_0}, \quad N^* = \frac{\Lambda - \delta E_1 I^*}{\mu}. \tag{20}$$

Here, the constants D and E_1 are defined as

$$D = 1 + \frac{\varepsilon\kappa}{\xi + \mu + \delta}, \quad E_1 = 1 + \frac{\kappa}{\xi + \mu + \delta}.$$

This formulation clearly shows that the endemic equilibrium exists if and only if $R_0 > 1$, and that the susceptible population at equilibrium is proportional to the total population via $S^* = N^*/R_0$. All other compartments scale linearly with the infectious population I^*, which is itself a function of the recruitment rate Λ, the disease parameters, and the mortality and treatment rates.

The explicit dependence on R_0 highlights the classical threshold property: as $R_0 \to 1^+$, the endemic equilibrium approaches the disease-free equilibrium, and I^* vanishes continuously.

2.6 Global Stability of the Endemic Equilibrium

To study the global stability of the endemic equilibrium (EE) $(S^*, E^*, I^*, T^*, R^*)$, we construct a Volterra-type Lyapunov function commonly used in epidemiological models [14, 16]:

$$V(S, E, I, T, R) = S^* g\Big(\frac{S}{S^*}\Big) + E^* g\Big(\frac{E}{E^*}\Big) + I^* g\Big(\frac{I}{I^*}\Big) + T^* g\Big(\frac{T}{T^*}\Big) + R^* g\Big(\frac{R}{R^*}\Big), \tag{21}$$

where

$$g(x) = x - 1 - \ln x, \quad x > 0.$$

This function satisfies $V \geq 0$ and $V = 0$ if and only if $(S, E, I, T, R) = (S^*, E^*, I^*, T^*, R^*)$.

Derivative along trajectories

Differentiating V along the solutions of the system gives

$$\dot{V} = \left(1 - \frac{S^*}{S}\right)\dot{S} + \left(1 - \frac{E^*}{E}\right)\dot{E} + \left(1 - \frac{I^*}{I}\right)\dot{I} + \left(1 - \frac{T^*}{T}\right)\dot{T} + \left(1 - \frac{R^*}{R}\right)\dot{R}.$$

Substitute the model equations:

$$\begin{aligned}\dot{V} = &\left(1 - \frac{S^*}{S}\right)\left[\Lambda - \rho\frac{I + \varepsilon T}{N}S - \mu S\right] + \left(1 - \frac{E^*}{E}\right)\left[\rho\frac{I + \varepsilon T}{N}S - (\beta + \mu)E\right] \\ &+ \left(1 - \frac{I^*}{I}\right)[\beta E - (\kappa + \mu + \delta)I] + \left(1 - \frac{T^*}{T}\right)[\kappa I - (\xi + \mu + \delta)T] + \left(1 - \frac{R^*}{R}\right)[\xi T - \mu R].\end{aligned}$$

Algebraic manipulation

Using the equilibrium conditions

$$\begin{aligned}
0 &= \Lambda - \rho\frac{I^* + \varepsilon T^*}{N^*}S^* - \mu S^*,\\
0 &= \rho\frac{I^* + \varepsilon T^*}{N^*}S^* - (\beta+\mu)E^*,\\
0 &= \beta E^* - (\kappa+\mu+\delta)I^*,\\
0 &= \kappa I^* - (\xi+\mu+\delta)T^*,\\
0 &= \xi T^* - \mu R^*,
\end{aligned}$$

we can rewrite each term as a product of the equilibrium value and a logarithmic-type difference:

$$\left(1-\frac{S^*}{S}\right)\left[\Lambda - \rho\frac{I+\varepsilon T}{N}S - \mu S\right] = S^*\left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right) - S^*\frac{\rho(I-I^*)+\varepsilon\rho(T-T^*)}{N}\frac{S}{S^*},$$

and similarly for the other compartments. Combining all terms carefully (see [16, 14]) yields

$$\dot{V} \le 0, \quad \text{and } \dot{V} = 0 \text{ if and only if } (S,E,I,T,R) = (S^*,E^*,I^*,T^*,R^*).$$

Thus, by *LaSalle's invariance principle*, the endemic equilibrium is globally asymptotically stable in the interior of the feasible region.

Remark: The detailed algebra involves expressing differences like $S - S^*$, $E - E^*$, etc., and using inequalities such as $x - 1 - \ln x \ge 0$ for $x > 0$. These manipulations are standard in the literature;[16] for a complete derivation for SEIR-type models.

2.7 Sensitivity Analysis

Sensitivity analysis allows us to identify the most influential parameters by measuring the relative change in a state variable resulting from variations in model parameters. In this study, the sensitivity of the basic reproduction number R_0 to model parameters is investigated using the normalized forward sensitivity index. The normalized forward sensitivity index of a variable with respect to a parameter is defined as the ratio of the relative change in the variable to the relative change in the parameter.

When R_0 is a differentiable function of a parameter p, the sensitivity index can be expressed in terms of partial derivatives as

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p}\,\frac{p}{R_0}. \tag{22}$$

The sensitivity analysis is carried out with respect to the parameter set

$$P = \{\alpha, \beta, \alpha_1, \kappa, \delta, \zeta, \mu\}.$$

The computed sensitivity indices, evaluated at the baseline parameter values, are summarized in Table 3. For the baseline parameter set, the basic reproduction number is $R_0 = 1.348764$.

Table 3: Baseline parameter values and normalized forward sensitivity indices of R_0.

S/N	Parameter	Baseline Value [Source]	Sensitivity Index
1	ρ	0.3 [17]	$+1.000000$
2	κ	0.3333 [18]	-0.653777
3	ε	0.0070 [19]	$+0.338924$
4	δ	0.0024 [20]	-0.185892
5	ξ	0.0021 [20]	-0.156401
6	μ	0.0000507 [19]	-0.006040
7	β	0.0240 [17]	$+0.002110$

INTERPRETATION OF SENSITIVITY ANALYSIS

Large values of the sensitivity index shows a strong influence of the chosen parameter on the reproduction number R_0. Table 3 shows the sensitivity indices and it is obvious that the values of parameters ρ is the largest which indicates that it is the most influential in determining the value of R_0, that is, increase in it's values while other values are constant will automatically expand the disease in a community. Same also goes with all other positive parameters like ε and β. Due to the fact that the basic reproduction number increases as there values increase, it means that the average number of secondary cases of infection will increase in the community. Additionally, while other parameters are kept constant, those with negative indices indicates that increase in their values can minimize the spread of the disease.

Figure 2: Sensitivity index by parameter

It shows the impact of each of the variables, in which the high "blue" bars are the variables that have strong effect in reducing the Reproduction number when there values are increased, and the high "Red" bars are the variables that have strong effect in increasing the Reproduction number when there values are reduced.

Sensitivity Analysis Radar Chart of R_0

Figure 3: Radar Chart (Normalized sensitivity indices)

This visualizes the normalized sensitivity of the basic reproduction number (R_0) to each parameter in a COVID-19 SEITR model, showing that the transmission rate (ρ) have the strongest positive influence on R_0

Figure 4: Shows transmission reduction at different levels

The intervention scenario by the use of mask and social distancing was simulated over a period of $350+$ days (x -axis). It can be seen that the infected fractions got to their peak on different days in which the 50% reduction reached its peak much later with less infected fraction than with the 15% and 30% reduction levels. This can be interpreted to mean that with more social distancing and use of mask, it will take longer days for the infected fraction to get to the peak, that is, infection will be slow and the number of the infected fraction will be less when there is an increase in the use of mask and social distancing. This makes sense and in agreement with reality.

Furthermore, there is an intercept after 200 days before the three levels of intervention gradually reduces with the 15% reduction coming down fastest than the 30% and 50% reductions.

Figure 5: Shows improvement on healthcare at different levels

This figure shows a situation where the healthcare is increased. The y -axis shows the percentage of the infected class. Here, the higher the treatment rate the faster the reduction of the number of infected individuals, and vice versa.

The piecharts shows the "fractions" for some of the intervention scenarios

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This figure shows a situation where the healthcare is increased. The y -axis shows the percentage of the infected class. Here, the higher the treatment rate the faster the reduction of the number of infected individuals, and vice versa.

Note: $0.02 = 2\%, 0.06 = 6\%$.

Same thing happens with an increase in treatment, but this time there is no interception of the three levels, meaning that the more the infected gets treated, the faster the disease fades away from the population.

Figure 6: Shows the baseline, transmission and health care improvement, with the combine strategies.

This figure shows that a reduction in transmission by the use of face mask and social distancing with an improvement in health care, the active Covid-19 cases fades away gradually while with the combination of the two strategies, the disease wears off at a faster rate and its peak at a very low level. The peak skyrocket when there are no interventions.

Figure 7: Shows the number of patients with and without intervention strategies

In the same vein, figure 7 shows a drastic reduction in the number of patients under treatment with improve health care, social distancing and the use of face mask. The active Covid-19 cases fades away gradually while with the combination of the two strategies, the disease fades off at a very fast rate and its peak at a very low level. Where there are no interventions, the number of patients under treatment is skyrocket to its peak few day after the disease was introduced into the population.

(a) Baseline (No Intervention)
Peak Active: Day 36 Total: 6857
Transmission Reduction (50%) Peak Active: Day
62 Total: 4405

Figure 8: Population distribution at the epidemic peak under varying intervention strategies.

The pie charts of figures 8(a)-8(d) shows the disease gets to its peak few days after it is introduced into a population when there are no interventions and longer days when there are intervention strategies. Also, when there is little treatment the exposed and the infected number of people are increased while when the treatment is high, the exposed and the infected number of people are reduced. This shows that the model is in agreement with reality.

Conclusion: This paper presents a deterministic mathematical model for analyzing the transmission dynamics of Coronavirus disease, it was shown that the state variables are positive and bounded within an invariant region Ω. Analysis of the model also showed that the disease free is locally asymptotically

stable when $R_0 < 1$ while the stabilities of the disease-free and endemic equilibriums were established using Lyapunov function. The sensitivity analysis of the basic reproduction number revealed that the recruitment rate and the contact rates are the most sensitive parameters indicating the need for intervention strategies to focus on reducing these parameters. Analysis also showed that increasing the rate at which the infectious are treated can control the
spread of the disease, emphasizing the importance of treatment as a target for control strategy in preventing the endemicity of the disease. The findings also emphasized the need for the use of face mask and social distancing in preventing the spread of the disease in a population.

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