

The Impact of Waning Immunity on Covid-19 Dynamics: A Sqit Model with Contact Tracing and Quarantine

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Abstract

Waning immunity is a major driver of COVID-19 reinfection, yet most SQIT models assume permanent immunity in treated individuals. This study develops an SQIT model with contact tracing, quarantine, and a waning rate ϕ from treated class T back to susceptible class S . The model is formulated as a system of nonlinear ordinary differential equations. The disease-free equilibrium and basic reproduction number R_0 are derived using the next generation matrix method. Stability analysis shows the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$. Numerical simulations reveal that increasing ϕ accelerates the return treated individuals to the susceptible class, causing higher infection peaks and sustained transmission despite contact tracing and quarantine. Sensitivity analysis identifies ϕ , contact tracing rate, and house hold quarantine can control transmission only when ϕ is below a critical threshold. The study concludes that waning immunity must be incorporated in control strategies to avoid overestimating the impact of contact tracing and quarantine on COVID-19.

Keywords: COVID-19, SQIT model, Waning immunity, Contact tracing, Quarantine.

1. Introduction

COVID-19 is a respiratory disease caused by SARS-CoV-2, first identified in Wuhan, China, in late 2019. Transmission occurs mainly through respiratory droplets and aerosols from infected individuals, with contact transmission possible via contaminated surfaces. These droplets can land in the mouths or noses of people who are nearby or possibly be inhaled into the lungs. Spread is more likely when people are in close contact with one another [1]. The best way to prevent the transmission of infection is to avoid or limit contact with people who are showing symptoms of COVID-19 or respiratory infections. The next best thing is to practice good hygiene and physical distancing to prevent bacteria and viruses from being transmitted. Other prevention tips are to, wash hands frequently for at least 20 seconds at a time with warm water and soap, sanitizing of hands with an alcohol-based sanitizer, refuse touching one's face, eyes, nose, or mouth when hands are dirty, Staying at least 6 feet (2 meters) away from people, avoiding crowd gathering, covering of mouth with a tissue or the inside of one's elbow whenever there is sneeze or cough, disposing of used tissues immediately, and when at public places where it is difficult to maintain physical distancing, wear a face mask that cover both mouth and nose [1]. Contact tracing and quarantine reduced transmission when implemented promptly. In the UK, modeling estimated combined isolation and tracing reduced transmission by 50-60%, compared to 2-30% for self-isolation alone. A 7-day quarantine with a lateral flow test averted -50% of transmission, while 14-day quarantine without testing averted -59%. [2] found that

with $R_0 = 2.5$, control was probable if more than 50% of contacts were traced and isolated quickly.

Regional performance varied. In the WHO South-East Asia Region, contact tracing performed well when resourced and caseloads were low, but utility declined as incidence increased. As of 1 Feb 2023, WHO reported 10,120,175 cumulative cases and 1,472,174 deaths in the south-East Asia Region, and 757,599,137 cases and 6,843,908 death globally [4]. Africa reported lower infection rates at 0.011 but the highest cumulative case fatality rate at 0.020, compared to Europe's 0.326 infection rate and 0.009 fatality rate [4-5]. Mathematical studies confirm the effect. [7] estimated that intensive contact tracing, quarantine, and isolation in China could reduce the control reproduction number substantially, with travel restrictions cutting infections in Beijing by 91% over 7 days. [8] showed that tracing 40% or more of contacts flattened epidemic curves. [9] used an age-structured SEIR model for Canada and found that contact tracing and quarantine of exposed individuals reduced peak hospitalizations by 40-60%. [10] also developed an agent-based model for Boston and showed that manual contact tracing with 60% coverage could bring the effective reproduction number below 1. More recently, [11] derived generic formulae for the effective reproduction number under contact tracing and proved that tracing delay and coverage are the dominant factors controlling spread. [12] developed an SVEIHR model to evaluate how vaccination, hospitalization, and contact tracing interact in the transmission dynamics of COVID-19 in Nigeria.

The pandemic spread of COVID-19 in selected countries after the outbreak of SARS-CoV-2 in Wuhan City, China was considered by [14]. The authors estimated the infection rate and the initial number of infected individuals by using officially reported data from the early stages of the epidemic, employing the SIQRK model — Susceptible (S), Infectible (I), Quarantine (Q), and officially confirmed Recovered (RK) populations. The disease was expected to subside about six months after the first patients were found. [15] examined the emergence of COVID-19 in Wuhan, Hubei Province, China, in December 2019, and its spread to the rest of China and overseas. The authors employed population flow data between Wuhan and other cities/regions by 23 January 2020, and used maximum log likelihood estimation and the Akaike Information Criterion (AIC) to estimate a total of 8,679 (95% CI: 7,701–9,732) COVID-19 cases in Wuhan by that date. The study concluded that a comprehensive analysis based on different data sources is necessary to overcome biases inherent in any single source.

A new COVID-19 model with optimal control analysis incorporating a vaccination class was presented by [16]. The authors formulated a vaccine-free model, established stability results for $R_0 < 1$, and extended the model to include vaccination. Four control strategies were considered: prevention, vaccination control, rapid screening of exposed individuals, and identification of infected individuals without prior screening. Using the forward-backward Runge-Kutta method, the results demonstrated that optimal control strategies can significantly minimize infected individuals and improve population health outcomes. [17] carried out a mathematical analysis of COVID-19 via a new mathematical model that included a resistive class together with a quarantine class. The developed model consisted of four compartments: the susceptible class, the healthy (resistive) class, the infected class, and the quarantine class. The authors derived basic properties including boundedness and positivity within a biologically feasible region, and computed the threshold quantity to discuss local equilibria. Numerical simulations were

compared with available real data for Wuhan city, where the infection was initially originated.

A landmark study on quantifying SARS-CoV-2 transmission and the role of digital contact tracing was conducted by [13]. Published in Science, the study used mathematical modeling to determine whether contact tracing could control the COVID-19 epidemic. The authors found that a large proportion of COVID-19 transmission occurred before symptom onset (pre-symptomatic transmission), making traditional manual contact tracing insufficient on its own. They concluded that instantaneous digital contact tracing via smartphone applications would be essential to achieve epidemic control, particularly when combined with case isolation and quarantine. The study estimated that the basic reproduction number (R_0) ranged between 2 and 3, and that contact tracing would need to be near-instantaneous to be effective given the short serial interval of the virus. [18] examined the effectiveness of isolation and contact tracing in managing COVID-19 outbreaks in the United Kingdom. Using a stochastic individual-based transmission model, the authors evaluated the probability of outbreak control under different levels of contact tracing and self-isolation. Their results showed that combined strategies including contact tracing, self-isolation, and physical distancing could substantially reduce the reproduction number. However, the study found that contact tracing alone was insufficient unless more than 80% of contacts were identified and isolated promptly. The findings strongly supported the use of complementary non-pharmaceutical interventions (NPIs) alongside contact tracing programs. [3] investigated the feasibility of controlling COVID-19 outbreaks through isolation and contact tracing. Using a stochastic branching process model, the authors simulated COVID-19 outbreak scenarios under varying assumptions of transmissibility, tracing efficiency, and proportion of cases traced. The study found that the probability of outbreak containment was strongly dependent on the speed of contact tracing and the fraction of contacts successfully traced. For high-transmission scenarios ($R_0 > 2.5$), the model estimated that contact tracing alone had limited success even when 80% of contacts were traced. The study emphasized the need for rapid identification and a robust public health infrastructure to make contact tracing effective.

[8] analyzed the impact of delays on the effectiveness of contact tracing in COVID-19. Using a mathematical model, the authors demonstrated that testing and tracing delays of even a few days could substantially reduce the effectiveness of contact tracing programs. Their study showed that same-day test-trace strategies could reduce the effective reproduction number from values above 1 to values below the epidemic threshold, while delays of more than three days severely diminished the impact. These findings highlighted the importance of rapid and efficient testing frameworks as a prerequisite for successful contact tracing. [10] modeled the impact of testing, contact tracing, and household bubbles on the deconfinement period of the COVID-19 epidemic in Catalonia, Spain. Using a detailed age-structured compartmental model combined with mobility data, the authors found that maintaining an effective reproduction number below 1 after lockdown required a combination of moderate contact reductions, extensive testing, and contact tracing. The study underscored the role of household and workplace transmission dynamics in shaping the overall epidemic and found that targeting high-risk settings through contact tracing could yield disproportionate epidemiological benefits.

While quarantine and contact tracing have been incorporated in several COVID-19 transmission models, the limitation of many previous studies is that they either focused primarily on exposed-class frameworks (e.g., SEIR-type models), emphasized optimal control strategies, vaccination effects, or evaluated tracing effectiveness through stochastic and agent-based approaches without explicitly distinguishing the different epidemiological consequences of tracing susceptible individuals versus tracing infected individuals within a treatment framework.

The present study addresses this gap by formulating an SQIT (Susceptible–Quarantine–Infected–Treated) deterministic model in which contact tracing acts through two distinct pathways: susceptible individuals identified through tracing are transferred into quarantine, while infected individuals identified through tracing are moved directly into treatment. This modelling structure allows the simultaneous investigation of household quarantine, treatment, and differential contact tracing mechanisms within a unified framework. From a mathematical perspective, the study establishes the positivity and boundedness of solutions, derives the disease-free and endemic equilibrium states, computes the basic reproduction number, establishes local stability of the disease-free equilibrium using the Jacobian and Routh–Hurwitz criteria and quantifies parameter influence through normalized sensitivity analysis.

The epidemiological insight generated by this framework is that the effectiveness of contact tracing depends not only on its coverage but also on the pathway through which traced individuals are managed. The results demonstrate that tracing susceptible individuals increases quarantine uptake and interrupts transmission chains, whereas tracing infected individuals accelerates treatment initiation and shortens the infectious period. Sensitivity analysis further identifies the transmission rate, recruitment rate, and contact tracing parameters as dominant determinants of disease spread. Consequently, the findings suggest that combining rapid contact tracing with prompt quarantine and treatment is more effective in reducing COVID-19 transmission than interventions targeted solely at infectious individuals. Unlike previous studies that primarily investigated quarantine and contact tracing independently or within SEIR-based frameworks, this study distinguishes the effects of tracing susceptible and infected individuals in an SQIT structure and examines how these pathways jointly influence transmission dynamics, treatment uptake, and disease control. The aim of this study is to assess the impact of contact tracing and household quarantine on COVID-19 transmission using an SQIT compartmental model.

This study is organized as follows: Section 2 gives the model description, including the model's positivity and boundedness. Section 3 explains an in-depth mathematical analysis of the model, such as the basic reproduction number, local stability analysis of the disease-free equilibrium and also sensitivity analysis of the basic reproduction number. Section 4 gives the numerical simulation of the model, including results and discussions while Section 5 gives the concluding remarks of the findings.

2. Mathematical Methodology

2.1. Model Description and formulation

In this research, an (S, Q, I, T) epidemic model which focuses only on the dynamics of covid-19 outbreak. The model was formulated by splitting the total human population at time t , into four mutually exclusive compartment which are the susceptible $S(t)$, Quarantine $Q(t)$, Infected $I(t)$, and Treated population, with sizes denoted by $S(t)$, $Q(t)$, $I(t)$, and $A(t)$, respectively such that $N = S + Q + I + A$.

At susceptible class, Λ is the total population of a particular country and ε , are the influx of immigrants that enter into the neighborhood. A fraction of immigrants is susceptible at rate (ρ) Newly infected individuals infect susceptible at a rate (β) . During the COVID-19 lock down, those that refuses to adhere to rule of lock down by the government by staying indoor are traced via contact trace at rate (c_1) while some died naturally. At quarantine class, fraction of immigrant is quarantine at rate $(1-\rho)$. Those from susceptible class that refuses to obey lock down rule are traced and put under quarantine for isolation. Those that do not develop symptoms (Asymptomatic) returns back to susceptible at rate (ϕ) . Some also died naturally. Those that are symptomatic via contact tracing progressed to infected class immediately at rate (c_2) . At treated class, quarantined that develops symptoms are adequately undergoing treatment at rate (π) . The infected population also progressed immediately to treated class at rate (ω) . While at infected and treated classes, Some died naturally (μ) and some died due to disease death rate at (δ) . Some among the treated class became recovered and are allowed to be integrated back and join the susceptible due to vaccines wane-off at the rate of ϕ . Thus, considering all the above assumptions made, the model is presented by the following deterministic system of non-linear differential equations which capture the complex interactions between the compartments.

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda + \varepsilon\rho - \beta SI - \mu S - c_1 S + \sigma Q + \phi T \\ \frac{dQ}{dt} &= \varepsilon(1 - \rho) + c_1 S - \mu Q - \sigma Q - \pi Q \\ \frac{dI}{dt} &= \beta SI - \mu I - \delta I - c_2 I - \omega I \\ \frac{dT}{dt} &= \pi Q + c_2 I + \omega I - \mu T - \phi T \end{aligned} \right\} \quad (1)$$

$$S(0) = S_0 > 0, I(0) = Q_0 > 0, I(0) = I_0 > 0, T(0) = T_0 > 0 \quad (2)$$

Table 1: Definition of Parameters/Variables

Variables/Parameters	Description
Λ	Total population in a country
ε	Immigrant that comes into a new neighborhood
βSI	Contact rate of infected and susceptible
μ	Natural death rate
$c_1 S$	Contact trace of susceptible that do not stay at home during lockdown
σQ	Quarantine that did not develop symptoms
πQ	Quarantine that develop symptoms
δ	Disease death rate of infected people
c_2	Contact trace of infected people that moved to treated class
ϕ	Recovered class that finally become susceptible
ω	The progressive rate of moving infected to treated
$\varepsilon(1 - \rho)$	Fraction of the immigrant that are quarantined
ρ	Fraction of the immigrant that are susceptible

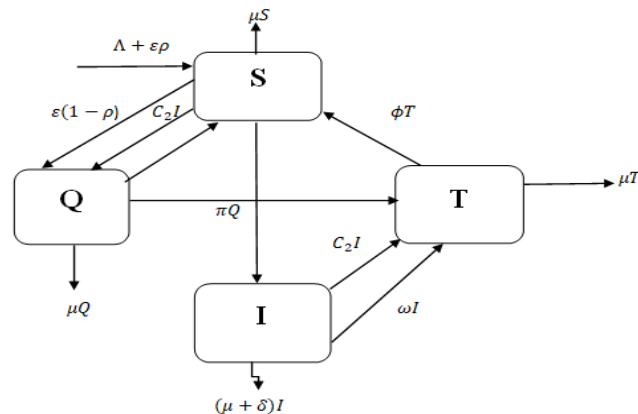


Figure 1: Schematic Diagram of the SQIT COVID-19 epidemic Model

2. 2 Positivity and boundedness of the model

In this section we shall show from model (1) that the state variables are non-negative and the solutions remain positive for all $t \geq 0$. Hence, the parameters in the model are assumed to be positive.

Theorem 1: Let the initial conditions or values of the state variables be such that $\{(S(0) \geq 0, Q(0) \geq 0, I(0) \geq 0, T(0) \geq 0, \epsilon \in \Omega)\}$, then the set $(S(t), I(t), A(t), T(t))$ is non-negative in Ω for all $t \geq 0$.

Considering the first compartment of (1)

$$\frac{dS}{dt} \geq -(\beta I + \mu + c_1)S$$

Using separable variable

$$\frac{dS}{S} \geq -(\beta I + \mu + c_1)dt$$

Integrate both sides

$$\int \frac{dS}{S} \geq - \int (\beta I + \mu + c_1)dt$$

$$\ln S(t) \geq -(\beta I + \mu + c_1)t + c$$

$$S(t) \geq e^{-(\beta I + \mu + c_1)t} \cdot e^c \quad \text{where } e^c = S_0$$

$$S(t) \geq S_0 e^{-(\beta I + \mu + c_1)t}$$

$$S(t) = S_0 e^{-(\beta I + \mu + c_1)t}.$$

$$S(0) = S_0$$

Since $S(t) \geq 0$, for all $t > 0$ provided that $S_0 \geq 0$.

Hence, $S(t) \geq 0$

Considering also the second compartment

$$\frac{dQ}{dt} = \epsilon(1 - \rho) + c_1 S - (\mu + \sigma + \pi)Q$$

$$\frac{dQ}{dt} \geq -(\mu + \sigma + \pi)Q$$

Using separable variable

$$\frac{dQ}{Q} \geq -(\mu + \sigma + \pi)dt$$

Integrate both sides

$$\int \frac{dQ}{Q} \geq - \int (\mu + \sigma + \pi) dt$$

$$\ln Q \geq -(\mu + \sigma + \pi)t + c$$

$$Q(t) \geq e^{-(\mu + \sigma + \pi)t} \cdot e^c \quad \text{where } e^c = Q_0$$

$$Q(t) \geq Q_0 e^{-(\mu + \sigma + \pi)t}$$

$$Q(t) = Q_0 e^{-(\mu + \sigma + \pi)t}$$

$$Q(0) = Q_0$$

Since $Q(t) \geq 0$, for all $t > 0$ provided that $Q_0 \geq 0$.

Hence, $Q(t) \geq 0$

It is possible to show using the same procedure for every other state variable,

$$S(t) \geq S_0 e^{-(\beta I + \mu + c_1)t}, Q(t) \geq Q_0 e^{-(\mu + \sigma + \pi)t} \geq 0, I(t) \geq T(0) e^{-(\mu + \delta + c_2 + \omega)t} \geq 0, T(t) \geq T(0) e^{-(\mu + \phi)t} \geq 0$$

This shows that all the solutions of equation (1) are positive for all $t \geq 0$. Therefore, the COVID-19 transmission model stated in (1) is both epidemiologically significant and numerically well posed in an attainable given region $\Omega \geq 0$

Theorem 2: Every solution in the region $\Omega = \{S(t), Q(t), I(t), T(t) \in \Omega^4: N(t) \leq \frac{D}{\mu} \text{ positively invariant with respect to the covid-19 model in the population.}$

The solutions for the system are contained and remain in the region Ω for all time $t \geq 0$.

Proof: Considering the equation of the model and adding up all the derivatives with respect to time t , we obtained. $N(t) = S(t) + Q(t) + I(t) + T(t)$

$$\begin{aligned} \frac{dN(t)}{dt} &= \Lambda + \varepsilon \rho - \beta SI - \mu S - c_1 S + \sigma Q + \phi T + \varepsilon - \varepsilon \rho + c_1 S - \mu Q - \sigma Q - \pi Q + \beta SI \\ &\quad - \mu I - \delta I - c_2 I - \omega I + \pi Q + c_2 I + \omega I - \mu T - \phi T \end{aligned}$$

$$\frac{dN(t)}{dt} = \Lambda + \varepsilon - (S + Q + I + T)\mu - \delta I$$

$$\text{Let } \Lambda + \varepsilon = D \text{ and } (S + Q + I + T) = N$$

$$\frac{dN(t)}{dt} = D - \mu N$$

$$I.F = e^{\int \mu dt} = e^{\mu t}$$

$$(e^{\mu t}) \frac{dN}{dt} + e^{\mu t} (\mu N) = D e^{\mu t}$$

$$\frac{d}{dt} (\mu N e^{\mu t}) = D e^{\mu t}$$

Integrating both sides

$$\int \frac{d}{dt} (\mu N e^{\mu t}) = \int D e^{\mu t}$$

$$N e^{\mu t} = \frac{D}{\mu} e^{\mu t}$$

$$N = \frac{D}{\mu} + k e^{-\mu t}$$

$$N(0) = N_0$$

$$N_0 = \frac{D}{\mu} + k$$

$$K = N_0 - \frac{D}{\mu}$$

$$N = \frac{D}{\mu} + \left[N_0 - \frac{D}{\mu} \right] e^{-\mu t}$$

$$N = \frac{D}{\mu} \text{ as } t \rightarrow \infty$$

As $t \rightarrow \infty$ the carrying capacity also reach a constant value

Where N_0 is the initial size of the population.

Therefore,

$$\lim_{t \rightarrow \infty} N(t) \leq \frac{\pi}{\mu} \quad (2)$$

This result implies that the COVID-19 model (1) has non-zero negative and bounded solution in the region Ω and all the solutions starting in Ω approach, enter or stay in Ω . Hence, it is sufficient to conclude that the model is epidemiologically well posed.

3. Mathematical Analysis of the Model

3.1.0 Disease free equilibrium point (DFE) represents a state where the disease is eliminated from the population, and the system reaches a steady state with zero infected individuals.

$$\frac{dS}{dt} = \Lambda + \varepsilon\rho - \beta SI - \mu S - c_1 S + \sigma Q + \phi T$$

$$\frac{dQ}{dt} = \varepsilon(1 - \rho) + c_1 S - \mu Q - \sigma Q - \pi T$$

$$\frac{dI}{dt} = \beta SI - \mu I - \delta I - c_2 S - \omega I$$

$$\frac{dT}{dt} = \pi Q + c_2 I + \omega I - \mu T - \phi T$$

At equilibrium point,

$$\frac{dS}{dt} = \frac{dQ}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = 0 \quad (3)$$

$$\Lambda + \varepsilon\rho - \beta SI - \mu S - c_1 S + \sigma Q + \phi T = 0$$

$$\varepsilon(1 - \rho) + c_1 S - \mu Q - \sigma Q - \pi T = 0$$

$$\beta SI - \mu I - \delta I - c_2 S - \omega I = 0$$

$$\pi Q + c_2 I + \omega I - \mu T - \phi T = 0$$

At DFE, we set $S \neq 0, Q = 0, I = 0, T = 0$ and solving (1), we obtained the DFE as

$$E^o = (S^o, Q^o, I^o, T^o) = \left(\frac{\Lambda + \varepsilon\rho}{\mu + c_1}, 0, 0, 0 \right) \quad (4)$$

3.1.1 Endemic equilibrium points represents a steady state where the disease persists in the population, with a constant number of infected individuals. At endemic equilibrium, we set $S \neq 0, Q \neq 0, I \neq 0, T \neq 0$, thus

$$\Lambda + \varepsilon\rho - \lambda^* S^* - k_1 S^* + \sigma Q^* + \Phi T^* = 0 \quad (5)$$

$$\varepsilon(1 - \rho) + c_1 S^* - k_2 Q^* = 0 \quad (6)$$

$$\lambda^* - k_3 I^* = 0 \quad (7)$$

$$\pi Q^* + k_4 I^* - k_5 T^* = 0 \quad (8)$$

Where,

$$\lambda^* = \beta I^*$$

$$k_1 = (\mu + C)$$

$$k_2 = (\mu + \sigma + \pi)$$

$$k_3 = (\mu + \delta + c_2 + \omega)$$

$$k_4 = (c_2 + \omega)$$

$$k_5 = (\mu + \phi)$$

Solving equations 15-18 simultaneously, we obtained the endemic equilibrium points of the covid-19 model represented by $x^* = (S^*, Q^*, I^*, T^*)$ and obtained as:

$$S^* = \frac{(k_2 k_3 k_5) \Lambda + (k_2 k_3 k_5) \varepsilon \rho}{(k_2 k_3 k_5) (\lambda^* + k_1) - \sigma (\varepsilon (1 - \rho) + c_1 S^*) k_3 k_5 + \phi S^* (\varepsilon (1 - \rho) + c_1 (k_3 \pi) + k_2 k_4 \lambda^*)} \quad (9)$$

$$Q^* = \frac{\varepsilon (1 - \rho) + c_1}{k_2} \left(\frac{(k_2 k_3 k_5) \Lambda + (k_2 k_3 k_5) \varepsilon \rho}{(k_2 k_3 k_5) (\lambda^* + k_1) - \sigma (\varepsilon (1 - \rho) + c_1 S^*) k_3 k_5 + \phi S^* (\varepsilon (1 - \rho) + c_1 (k_3 \pi) + k_2 k_4 \lambda^*)} \right) \quad (10)$$

$$I^* = \frac{\lambda^*}{k_3} \left(\frac{(k_2 k_3 k_5) \Lambda + (k_2 k_3 k_5) \varepsilon \rho}{(k_2 k_3 k_5) (\lambda^* + k_1) - \sigma (\varepsilon (1 - \rho) + c_1 (k_3 k_5) + \phi (\varepsilon (1 - \rho) + c_1 (k_3 \pi) + k_2 k_4 \lambda^*))} \right) \quad (11)$$

$$T^* = \frac{\pi}{k_5} \left(\frac{(k_2 k_3 k_5) \Lambda + (k_2 k_3 k_5) \varepsilon \rho}{(k_2 k_3 k_5) (\lambda^* + k_1) - \sigma (\varepsilon (1 - \rho) + c_1 S^*) k_3 k_5 + \phi (\varepsilon (1 - \rho) + c_1 (k_3 \pi) + k_2 k_4 \lambda^*)} \right) \quad (12)$$

3.2 Basic Reproduction Number (R_0)

The computation of basic reproduction number is essential and this is defined as defined as the effective number of secondary infections caused by infected individuals in his entire period of infectiousness. To determine the next generation matrix for the model, the number of ways that new infectious can be transformed between compartment are put into considerations. F_1 and V_1 are computed as follows, using the approach of [23], where

$$f_i = \begin{pmatrix} 0 \\ \beta SI \end{pmatrix} \quad (13)$$

$$v_i = \begin{pmatrix} k_2 Q \\ K_3 I \end{pmatrix} \quad (14)$$

Where,

$$k_2 = (\mu + \sigma + \pi),$$

$$k_3 = (\mu + \delta + c_2 + \omega)$$

Let k be the next generation matrix, comparing of two parts F and v^{-1}
Where,

$$F = \left[\frac{\partial f_i(x_0)}{\partial x_j} \right], \quad V = \left[\frac{\partial v_i}{\partial x_j} \right] \quad (15)$$

The term f_i and v_i are computed as

$$F = \begin{bmatrix} \frac{\partial f_1}{\partial Q} & \frac{\partial f_1}{\partial I} \\ \frac{\partial f_2}{\partial Q} & \frac{\partial f_2}{\partial I} \end{bmatrix} = \begin{bmatrix} 0 & 0 \\ 0 & \beta S \end{bmatrix} \quad (16)$$

$$V = \begin{bmatrix} \frac{\partial v_1}{\partial Q} & \frac{\partial v_1}{\partial I} \\ \frac{\partial v_2}{\partial Q} & \frac{\partial v_2}{\partial I} \end{bmatrix} = \begin{bmatrix} k_2 & 0 \\ 0 & k_3 \end{bmatrix} \quad (17)$$

$$V^{-1} = \begin{bmatrix} \frac{1}{k_2} & 0 \\ 0 & \frac{1}{k_3} \end{bmatrix} \quad (18)$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 \\ 0 & \beta S \end{bmatrix} \begin{bmatrix} \frac{1}{k_2} & 0 \\ 0 & \frac{1}{k_3} \end{bmatrix} \quad (19)$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 \\ 0 & \frac{\beta S}{k_3} \end{bmatrix} \quad (20)$$

The basic R_0 which is the dominate eigenvalues of the product FV^{-1} is therefore obtain as $R_0 = \frac{\beta S}{k_3}$ but at dfe, $x_0 = \left(\frac{\Lambda + \varepsilon \rho}{\mu + c_1}, 0, 0\right)$.

Therefore,

$$R_0 = \frac{\beta(\Lambda + \varepsilon \rho)}{k_3(\mu + c_1)} \quad (21)$$

Where $k_3 = (\mu + \delta + c_2 + \omega)$

$$R_0 = \frac{\beta \Lambda + \beta \varepsilon \rho}{\mu^2 + \mu \delta + \mu c_2 + \mu \omega + \mu c_1 + c_1 \sigma + c_1 c_2 + \omega c_1} \quad (22)$$

3.2. Stability analysis of the Disease- free equilibrium

Theorem 3: The disease-free state is locally asymptotically stable if the basic reproduction number $R_0 < 1$ and unstable if otherwise.

Proof:

We evaluate the Jacobian matrix of the model at the disease- free equilibrium $\left(\frac{\Lambda + \varepsilon \rho}{\mu + c_1}, 0, 0, 0\right)$

$$J(x_0) = \begin{bmatrix} -G_1 & \sigma & -\beta q & \phi \\ c_1 & -G_2 & 0 & 0 \\ 0 & 0 & \beta q - G_3 & 0 \\ 0 & \pi & G_4 & -G_5 \end{bmatrix} \quad (23)$$

Where, $q = \frac{\Lambda + \varepsilon \rho}{\mu + c_1}$

$$G_1 = (\mu + c_1)$$

$$G_2 = (\mu + \sigma + \pi$$

$$G_3 = (\mu + \delta + c_2 + \omega)$$

$$G_4 = (c_2 + \omega)$$

$$G_5 = (\mu + \phi)$$

The characteristic equation is obtained from

$$\det(\lambda I - J(E_0)) = 0,$$

which can be written in the form

$$\lambda^4 p_0 + \lambda^3 p_1 + \lambda^2 p_2 + \lambda p_3 + p_4 = 0 \quad (24)$$

where

$$p_0 = 1$$

$$p_1 = (-\beta q + G_1 + G_2 + G_3 + G_5)$$

$$p_2 = (-\beta q G_1 - \beta q G_2 - \beta q G_5 - \sigma c_1 + G_1 G_2 + G_1 G_3 + G_1 G_5 + G_2 G_3 + G_2 G_5 + G_3 G_5)$$

$$p_3 = (\beta q \sigma c_1 - \beta q G_1 G_2 - \beta q G_1 G_5 - \beta q G_2 G_5 - \pi \phi c_1 - \sigma G_3 c_1 - \sigma G_5 c_1 + G_1 G_2 G_3 + G_1 G_2 G_5 + G_1 G_3 G_5 + G_2 G_3 G_5)$$

$$p_4 = (\pi \beta \phi q c_1 + \beta q \sigma G_5 c_1 - \beta q G_1 G_2 G_5 - \pi \phi G_3 c_1 - \sigma G_3 G_5 c_1 + G_1 G_2 G_3 G_5)$$

According to the Routh-Hurwitz criteria, all eigenvalues have negative real parts if

$$p_0 > 0, p_1 > 0, p_2 > 0, p_3 > 0, p_4 > 0, p_1 p_2 > p_3, p_1 p_2 p_3 > p_3^2 + p_1^2 p_4 \quad (25)$$

Substituting the expressions for p_0, p_1, p_2, p_3 and p_4 obtained above, we observe that all coefficients are positive whenever $R_0 < 1$.

Furthermore,

$$p_1 p_2 - p_3 > 0, \text{ and}$$

$$p_1 p_2 p_3 - p_3^2 - p_1^2 p_4 > 0.$$

Hence all Routh-Hurwitz conditions are satisfied for $R_0 < 1$.

Therefore, every eigenvalue of $J(E_0)$ has negative real part and the disease-free equilibrium E_0 is locally asymptotically stable. Conversely, when $R_0 > 1$, at least one eigenvalue becomes positive and E_0 loses stability. Thus, E_0 is locally asymptotically stable if and only if $R_0 < 1$.

3.4. Sensitivity Analysis of the basic Reproduction number

This technique assesses how changes in model parameters affect the outcome of a mathematical model. It determines which parameters most influence the model's behavior, enabling researchers to focus on key factors. By evaluating the impact of parameter variations, sensitivity analysis informs decision-making and improves understanding of complex systems. Adapting the methodology introduced by [20], the normalized forward sensitivity index of a variable "K" that depends differentiable on a parameter "e" is defined as:

$$X_e^K := \frac{\partial K}{\partial e} * \frac{e}{K} \quad (29)$$

Following the approach used in [21], the sensitivity indices of the parameters relative to the basic reproduction number (32) is established. Therefore, the normalized forward sensitivity indices of the basic reproduction number (32) associated with parameters, τ , is calculated as

$$X_{\tau}^{R_0} := \frac{\partial R_0}{\partial \tau} * \frac{\tau}{R_0} \quad (30)$$

R_0 is therefore computed with respect to each of the parameters in table 2 and are seen in table 3 below:

Table 2: Parameter values, baseline values and source used in the model.

Parameters	Baseline Values	Sources
β	0.62×10^{-4}	Calculated from demographic data
Λ	0.25	Calculated from demographic data
c_1	0.06	Assumed
c_2	0.07	Assumed
ε	0.5	Flaxman et al 2020
δ	0.006	Verity et al 2020
ρ	0.3	Assumed
ω	0.01	Flaxman et al 2020
σ	0.1	Assumed
π	0.2	Hellewell J. et al 2020
μ	0.00039	World bank
ϕ	0.0135	Okuoghae 2020

Table 3: Sensitivity Indices of R_0 in terms of the Model's parameter baseline values

Parameter	Sensitivity index
Λ	+0.625000000
β	+1.000000000
ω	-0.1157541382
ε	+0.375000000
c_1	-0.9935419771
c_2	-0.8102789675
μ	-0.01097243424
ρ	+0.375000000
δ	-0.06945248293

3.5. Interpretation of Sensitivity Indices

The purpose of sensitivity analysis is to identify the most influential parameters on R_0 , which can inform disease control strategies. Parameters with positive sensitivity index means that an increase in the parameter value leads to an increase in R_0 , making the disease more transmissible. Conversely, parameters with negative sensitivity index imply that an increase in the parameter value leads to a decrease in R_0 , making the disease less transmissible. By identifying the most sensitive parameters, policymakers can target interventions to reduce disease transmission and control outbreaks more effectively.

The sensitivity indices of the basic reproduction number R_0 , relative to its parameters are summarized in table 3. $\beta, \Lambda, \varepsilon, \rho$ are parameters with positive indices

while parameters with negative indices are c_1, c_2, ω , and μ . For instance, $X_{\beta}^{R_0} = +1.0000$ means that increasing or decreasing β by 10% increases or (decreases) R_0 by 10% while $X_{c_2}^{R_0} = -0.8102789675$ means that increasing or (decreasing) R_0 by 8.102789675% as clearly observed from table 3.

4. Numerical Simulations and Discussion

A computational method used to solve complex mathematical models. In this study, numerical simulations are used to visualize and analyze the behavior of the COVID-19 transmission dynamics using Maple 18.0 mathematical software. The simulation demonstrates model equations and reveals the impact of those parameters on the model.

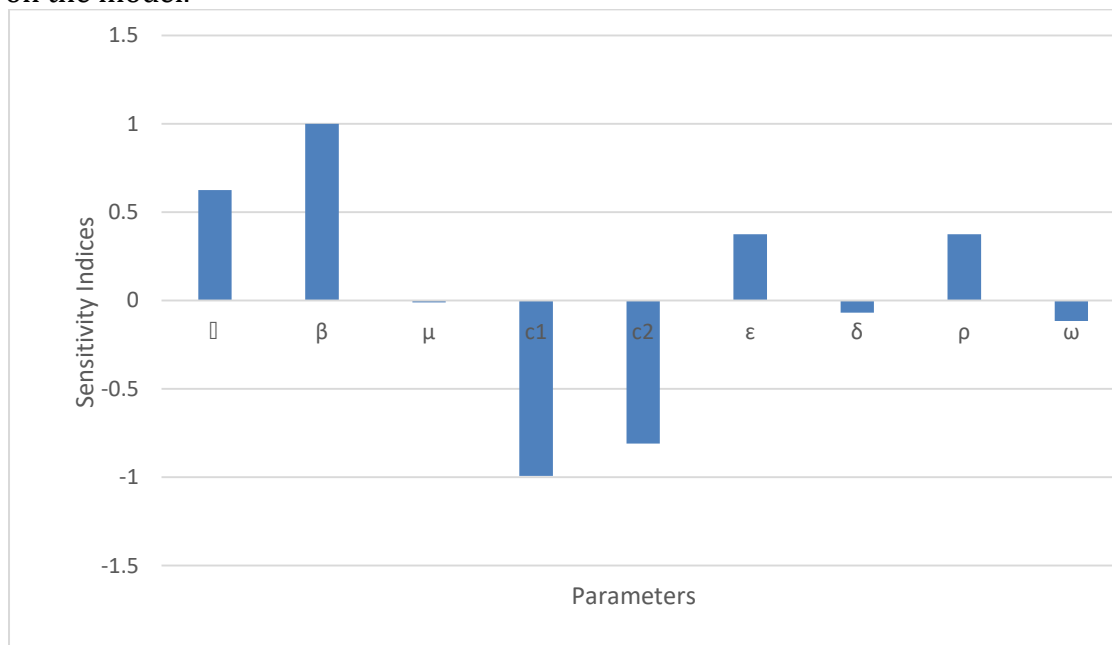


Figure 1: Graphical Representation of the sensitivity indices of R_0

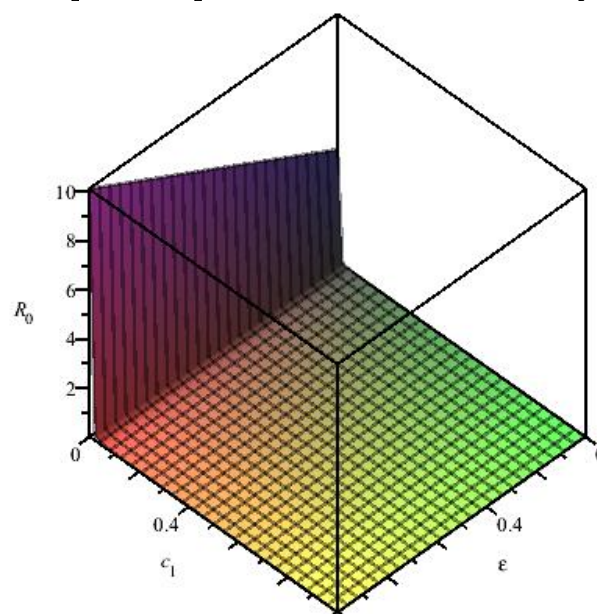


Figure 2: 3-D Plot showing the effect of c_1 and ϵ on the basic reproduction number (R_0)

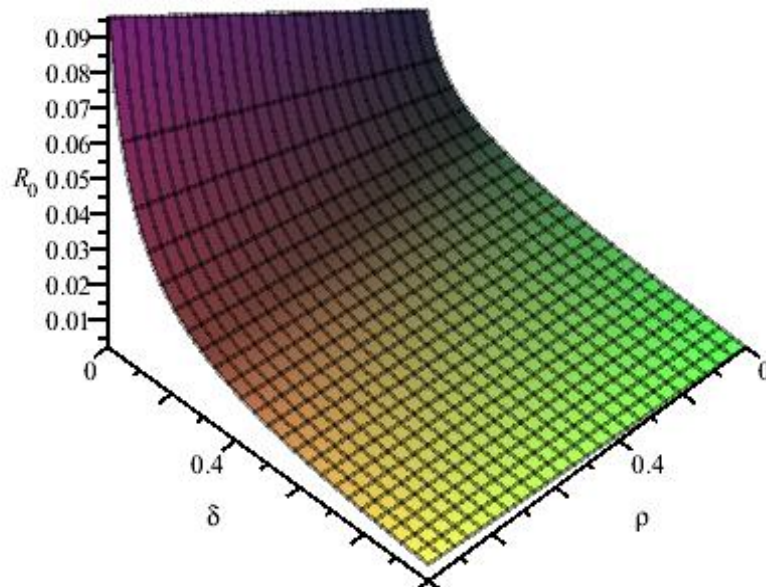


Figure 3: 3-D Plot showing the effect of δ and ρ on the basic reproduction number (R_0)

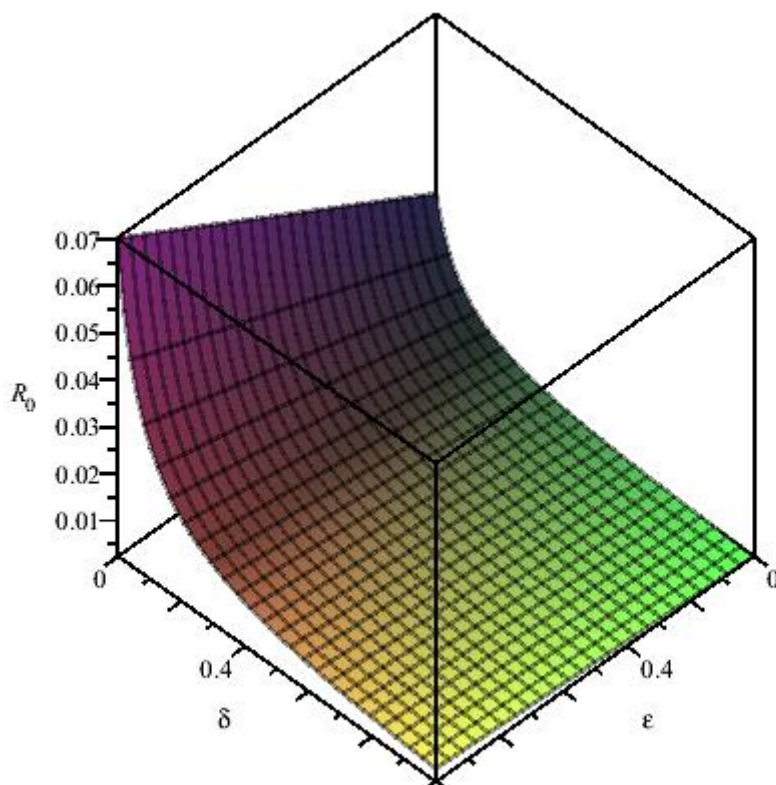


Figure 4: 3-D Plot showing the effect of δ and ϵ on the basic reproduction number (R_0)

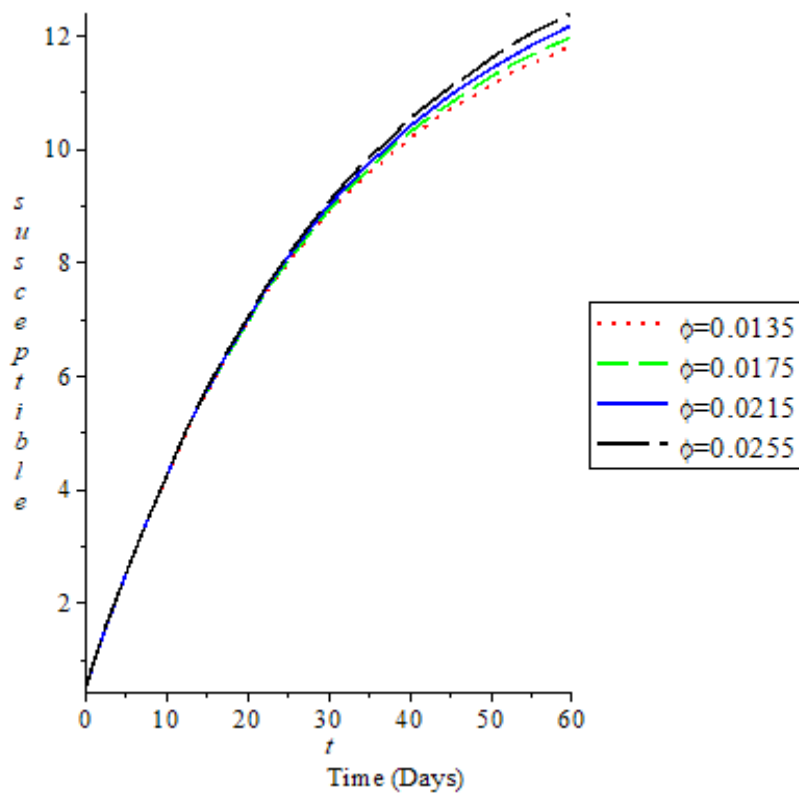


Figure 5: Graph of susceptible population against time for various values of ϕ

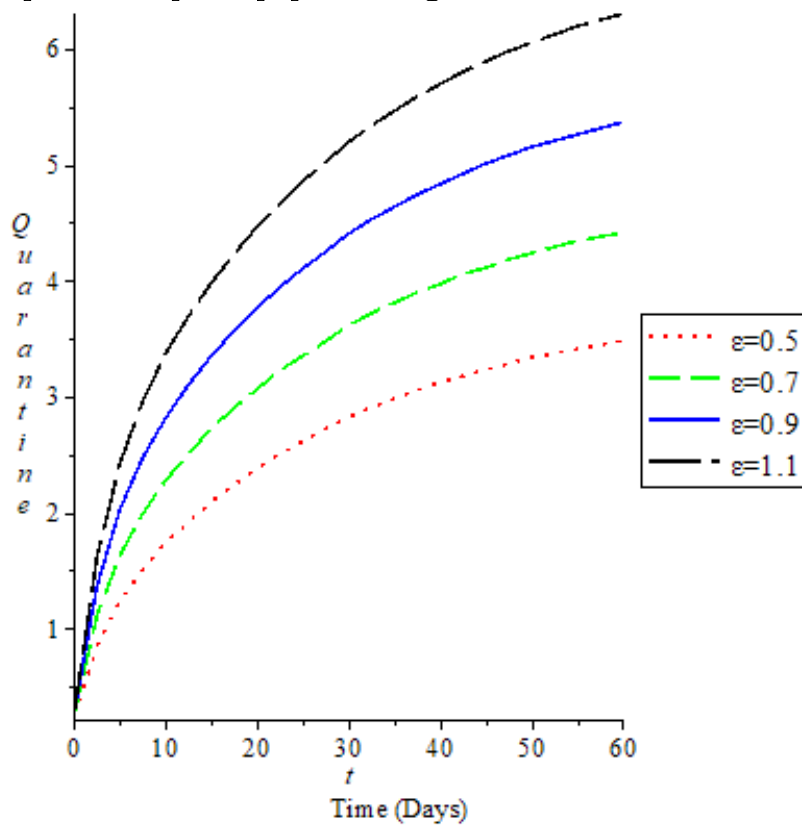


Figure 6: Graph of the Quarantine population against time for various values of ϵ

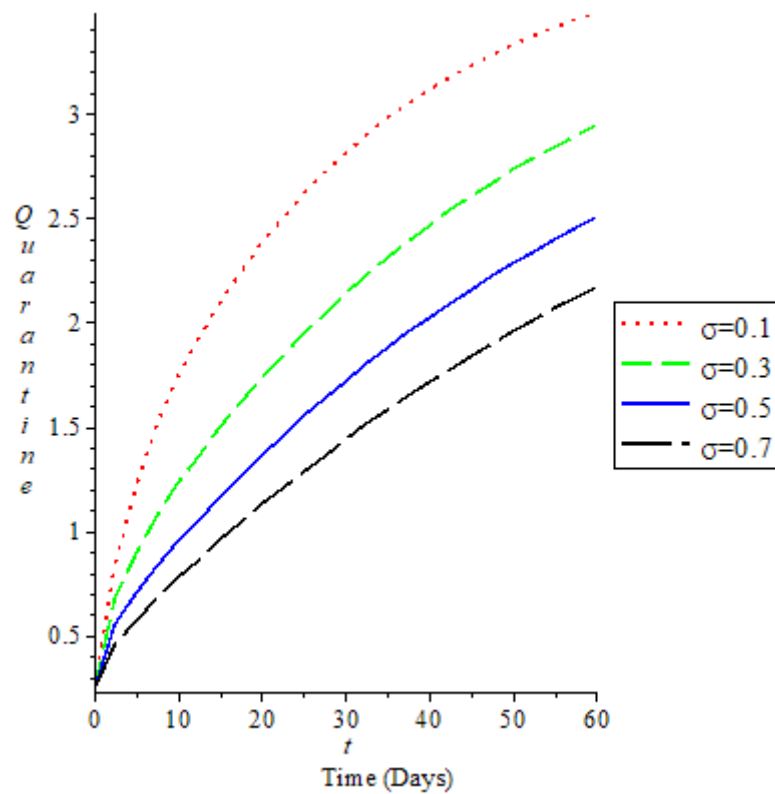


Figure 7: Graph of Quarantine population for various values of sigma (σ)

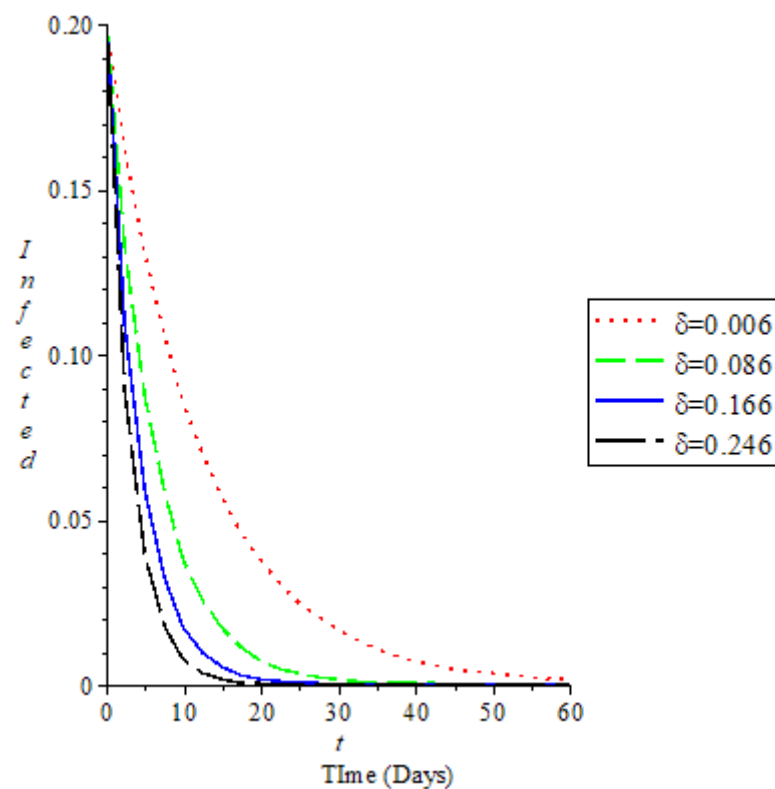


Figure 8: Graph of the Infected against time for various values of delta (δ)

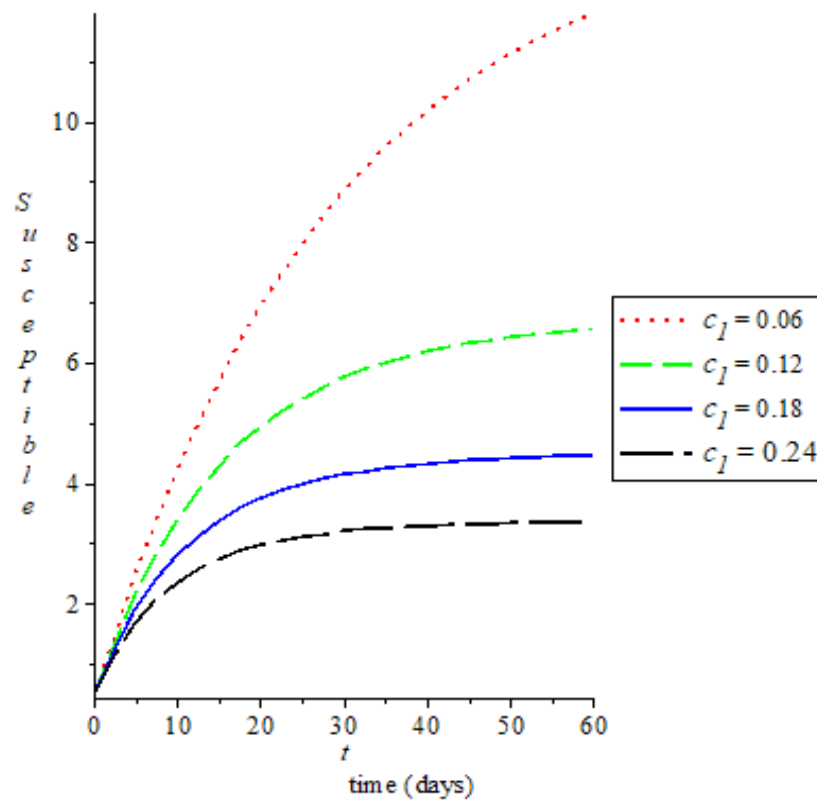


Figure 9: Graph of the Infected against time for various values of c_1 .

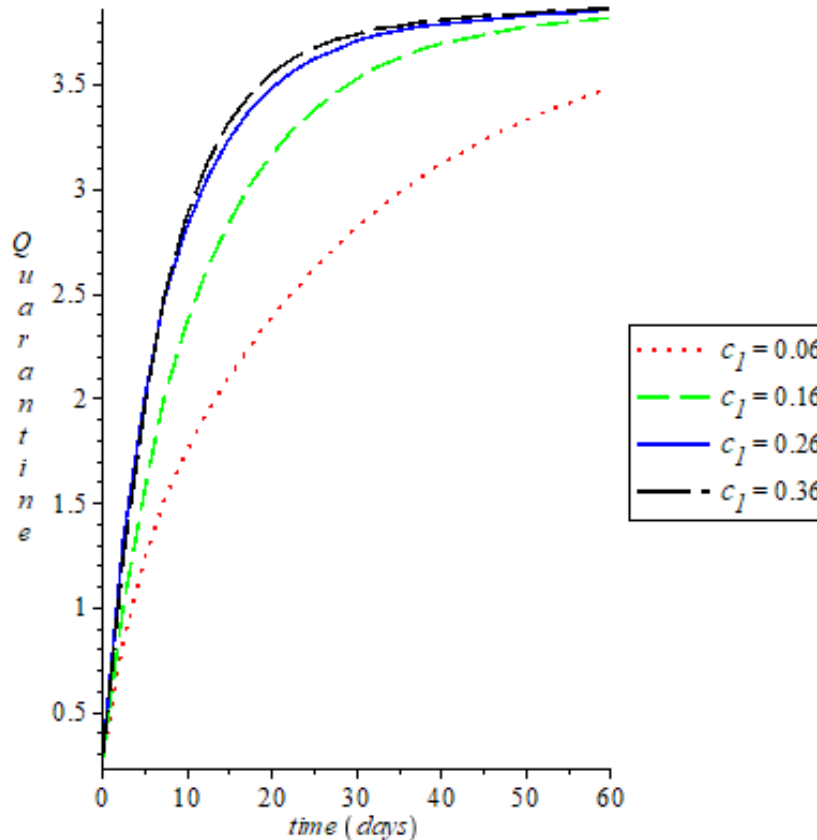


Figure 10: Graph of quarantine population against time for various values of c_1 .

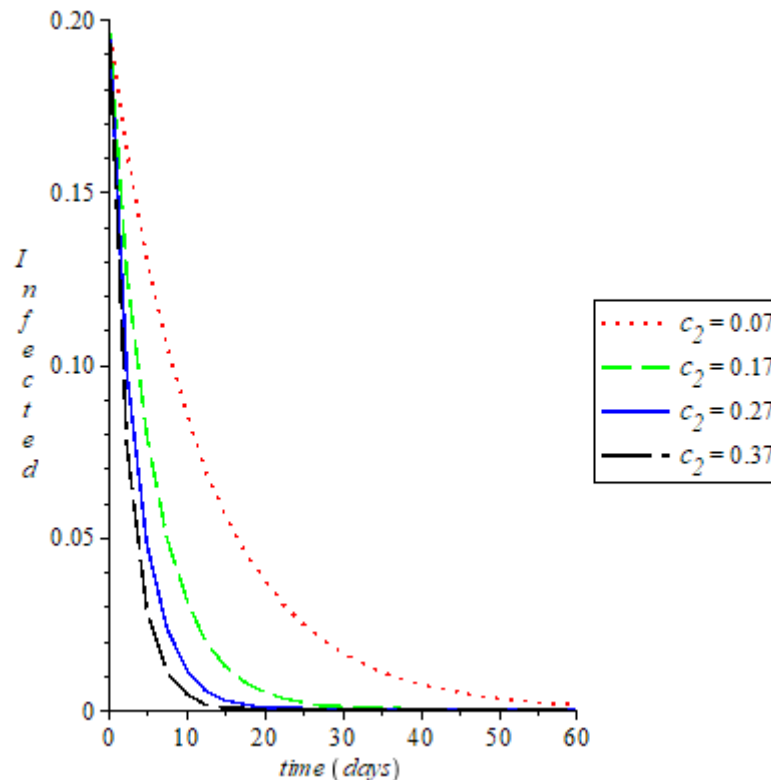


Figure 11: Graph of quarantine population against time for various values of c_2 .

4.2. Discussion

Figure 1: Illustrate the graphical representation of the basic reproduction number in a bar chart. The most sensitive parameters among others are β, c_1, c_2, Λ . It shows the influences of each parameter on the basic reproduction R_0 parameters $\beta, \Lambda, \varepsilon$, and ρ . are positively correlated and have a greater impact on R_0 , the parameters are ω, c_1, c_2, μ , and δ . negatively correlated with R_0 . R_0 will decrease rapidly as the parameters increases.

Figure 2 reveals the sensitivity of the basic reproduction number to the parameters c_1 and ε . An increase in both c_1 and ε , reduce the basic reproduction number. Also, in figure 3, an increase in parameter δ , resulted to a decrease in the basic reproduction number while an increase in ρ , increase the basic reproduction number.

Figure 4 reveals the sensitivity of the basic reproduction number to the parameters δ and ε . It also shows that an increase in δ decrease R_0 while increase in ε , show a corresponding increase in parameter ε .

Figure 5 shows that increasing the immunity-loss (or treatment-reversal) parameter ϕ increases the susceptible population over time. Epidemiologically, this indicates that when recovered or treated individuals return more rapidly to the susceptible class, the pool of individuals available for infection is continually replenished. Consequently, disease elimination becomes more difficult because transmission can be sustained even after treatment programs are implemented. This observation is consistent with the findings of Ahmed et al. [17], who reported that maintaining a large susceptible population can prolong disease persistence in compartmental epidemic models.

Figure 6 reveals that increasing (ε) , adds more immigrants directly into quarantine, so the quarantine population grows as ε rises continuously being identified and isolated. Higher (ε) values imply that more people end up in quarantine, showing that ε drives more individuals into quarantine compartment of the model.

Figure 7 shows the graph of quarantine population against time for various values of sigma (σ) . Increase in (σ) , moves asymptomatic quarantined people back to susceptible, thereby decrease quarantine population and adds to susceptible population.

Figure 8: demonstrates that increasing the disease-induced death rate $(\delta\backslash\delta\delta)$ decreases the infected population because infected individuals are removed from the transmission process more rapidly. However, disease-induced mortality is not a desirable control mechanism from a public-health perspective because it reflects severe disease outcomes rather than successful intervention.

In figure 9, An increase in the susceptible contact rate c_1 , leads to a larger quarantine population due to higher transfer from the susceptible compartment while in **figure 10** shows an increase in the infected contact tracing rate c_2 reduces the quarantine population because faster treatment of infected individuals' lower transmission, which in turn reduces the flow of people entering more susceptible are identified and moved into quarantine.

Figure 10 shows that increasing the tracing rate of infected individuals reduces the infected population through earlier treatment initiation. Together, these results indicate that contact tracing acts through two complementary pathways: preventing susceptible individuals from becoming infected and reducing the infectious period of already infected individuals. This finding agrees with the studies of [3], [13], and [8], who demonstrated that rapid and extensive contact tracing is one of the most effective non-pharmaceutical interventions for controlling COVID-19 outbreaks.

5. Conclusion

We analyzed an SQIT compartmental model to assess the role of quarantine, treatment, contact tracing, and disease-induced death in disease transmission. The study established positivity and boundedness of solutions, derived the disease-free and endemic equilibria, computed the basic reproduction number, and examine local stability using the Jacobian matrix and numerical simulations were used to evaluate the influence of model parameters. Sensitivity analysis showed that transmission rate, recruitment rate, and contact tracing rates are the key drivers of the system dynamics. Quarantine and contact tracing reduce disease spread by removing individuals from the transmission chain. Increasing contact tracing for susceptible shifts more individuals into quarantine, while increasing it for infected individuals accelerates movement to treatment and lower the infected population.

Overall, combining contact tracing with quarantine and treatment controls the outbreak more effectively than interventions that target only the infectious period. Overall, the results suggest that combining contact tracing with quarantine and treatment reduces the infected population more than focusing only on treatment during the infectious period.

6. Recommendation

Based on our research work and findings, the followings thrust areas are recommended for future studies:

1. Expand contact tracing to detect cases earlier and reduce transmission

2. Use shorter quarantine periods combined with testing to lower social and economic burden
3. Focus interventions on settings and groups with the highest transmission rates.
4. Improve data collection on contact tracing coverage to strengthen model predictions and policy decisions.

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