Mathematical Modelling of Dynamics and Control of Coronavirus Disease 2019 (Covid-19) Transmission

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Abstract: Due to the massive economic and medical costs associated with infectious diseases, the World Health Organization's announcement of the global pandemic outbreak of the coronavirus COVID-19 has become one of the most concerning event in human history. The novel Covid-19, SARS-COV2, a seriously harmful infection that has made COVID-19 a lethal illness, an infection that target the human respiratory system is currently causing recurrent flare-ups of a Covid infection that the world is currently battling. Covid-19 specifically targets the human respiratory system. We are prepared to research the effects of carriers on the transmission dynamics of the Covid-19 disease by simulating the influence of carriers. By determining the positivity and boundedness of the realistic region for equilibrium, the analysis is carried out. Additionally, we ascertain the prerequisites that ensure its existence as well as the fundamental Reproduction number. Graphs were used to show and illustrate how regulating the dynamics affected the dynamics. The transmission models and regulation of Covid are explained in this paper. After using the SEIR model, we established the expectation that Covid-19 would persist but that transition could be managed. Therefore, our model predicts that while Covid won't be completely eradicated, the factors affecting its transmission will aid in illness forecasting and management.

Keywords: Covid-19, diseases, Mathematical Modelling, Modelling of Covid-19 Dynamics, Control of Covid 19 Transmission

MSC 2020 Classification: 92B05, 92D30

1. Introduction

For some infectious or contagious diseases, there are people who can transfer their disease yet won't show any side effects. These arrangements of individuals are called carriers and they play a crucial role in the spread of the infection. We can arrange carriers in two unique sorts. Hereditary transporters have the disease on their latent qualities. They can just exchange the sickness to their children and are not infectious. The significant piece of our review centers on irresistible illness transporters. Due to the massive economic and medical costs associated with infectious diseases, the World Health Organization's announcement of the global pandemic outbreak of the coronavirus COVID-19 has become one of the most concerning event in human history. The novel Covid-19, SARS-COV2, a seriously harmful infection that has made COVID-19 a lethal illness, an infection that target the human respiratory system is currently causing recurrent flare-ups of a Covid infection that the world is currently battling. Covid-19 specifically targets the human respiratory system.

Viral illness transmission associated to medical services is a serious problem with large financial costs and a potential death toll. In clinics all throughout the world, contaminations with the highly contagious SARS-CoV-2 infection have been shown to predominate. The number of patients crammed into a medical clinic bayou may have an impact on the spread of sickness. In this review, we analyze a numerical demonstrating and computational method to deal with, represent the transmission of SARSCoV-2 among hospitalized patients in order to examine this viewpoint. Understanding the mechanism of the diseases spread in the human population, such as COVID-19, requires the use of mathematical modeling. These models produce insights into the mechanisms of infectious disease transmission and help politicians and health professionals stop its wide spread. Over the past century, there has been a lot of research done on mathematical models of infectious disease dynamics [1]-[4]. These models are based on the SIR model.

To investigate and forecast the spread of Covid-19 in the French overseas department of Mayotte, Manou-Abi et al. [5] suggested a modified SEIR model. The dynamics of the pandemic may be understood and predicted using these models, which is highly intriguing. Studying the consequences of the various attenuation measurements through simulation is also crucial [6, 7].

As described by Safaret al. [8], alongside efforts to identify trustworthy resources, such as coronavirus vaccinations, the effects of the so-called non-pharmaceutical tactics are not insignificant [9] and [10]. We identify containment, isolation of proven cases, and mitigation by the use of masks and barrier gestures among these non-pharmaceutical approaches. The effects of non-pharmaceutical interventions on the spread of the illness are the subject of this essay. The reproduction value, which gauges a virus's ability for transmission, serves as the foundation for regulating the spread of viruses. When an infection infects less than one person and the speed of the disease is expected to stop, the value for spread differs from one virus to another (for example, value for influenza spread [11]). However, there is still limited information about the transmission potential of infectious diseases [12]-[14], and more theoretical work is required to connect the patterns of an epidemic. Several other authours who have made significant contributions includes but not limited to [15]-[22]In the current study, we revisit the work of Ebraheemet al. [23], modified the SIR model to include the effects of containment measures, and multiplied tests on the values of the basic reproduction number as well as latency time constants, which produced a remarkable accurate estimate of the real-time data for several countries.

2. Epidemic Model with Asymptomatic Carrier

The total human population at time t, denoted by $N_h(t)$, is split into a mutually exclusive sub-populations of susceptible humans (S(t)), Quarantine on exposed humans (Q(t)), asymptomatic infectious humans (A(t)), symptomatic infectious humans (I(t)), detected infectious humans via testing (and are isolated and in some form of hospitalization for prompt treatment) $(I_D(t))$ and recovered humans (R(t)). We assume that those in the I_D are completely isolated and do not come in contact with the general population.

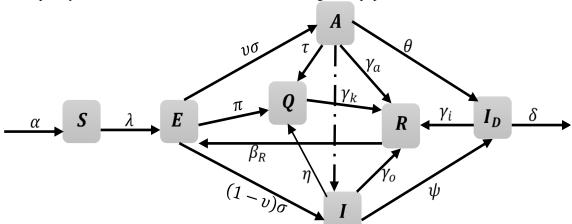


Figure 1: Schematic diagram of the model

Consider the interaction within the community as shown in the compartmental block shown in Figure 1, taking all the subclasses enumerated above into consideration, we assume the following:

- The migration rate to the community increases the total population
- Recovered population could still become susceptible
- Susceptible could become Asymptomatic, quarantine or symptomatic infectious
- Asymptomatic class could be quarantine, recovered, symptomatically-infectious humans or detected infectious humans,
- symptomatically-infectious humans could be quarantine, recovered or detected infectious humans,
- Detected infectious humans could recover or die due to the disease.
- We also assume that demographic parameters including births and natural deaths are excluded due to dynamics of an epidemic that is occurring within 1 14 days.

With the above assumptions, the model for COVID-19 transmission dynamics in a general population is given by the following system of deterministic non-linear differential equations given as

$$\frac{dS}{dt} = \alpha - \lambda(1 - \delta)(1 - \epsilon)S + (1 - \varphi)\beta_R R$$

$$\frac{dE}{dt} = \lambda(1 - \delta)(1 - \epsilon)S - \rho - (\sigma + \nu + \pi + \kappa)E + \varphi\beta_R R$$

$$\frac{dQ}{dt} = \pi E + \tau A + \eta I - (\gamma_k + d_Q)Q$$

$$\frac{dA}{dt} = \nu E - (\theta + \gamma_a + \mu + \tau + d_A)A$$

$$\frac{dI}{dt} = \sigma E - (\psi + \gamma_o + \eta + d_I)I + \mu A$$

$$\frac{dI_d}{dt} = \theta A + \psi I - (\gamma_i + d_{I_D})I_D + \kappa E$$

$$\frac{dR}{dt} = \gamma_i I_D + \gamma_a A + \gamma_o I + \gamma_k Q - \beta_R R$$

$$S(0) = S_0, E(0) = E_0, Q(0) = Q_0, A(0) = A_0, I(0) = I_0, I_d(0) = I_{d_0}, R(0) = R_0$$
(1)

It is imperative to state that, in the setting being considered in this work, the strict adoption of the use of face mask was being promoted well into the outbreaks.

3. Analysis of the Model

3.1Total Population

The total population N susceptible (S), Infected carriers (I_c) , the symptomatically infected (I) and those removed (R) from the population either by death, isolation or recovery from the disease etc. So than we have

$$N = S + Q + E + A + I + I_D + R (2)$$

Thus the total human population at time t, denoted by $N_h(t)$, is obtained by adding the number of susceptible humans (S(t)), Quarantine on exposed humans (Q(t)), asymptomatic infectious humans (A(t)), symptomatic infectious humans (I(t)), detected infectious humans via testing $(I_n(t))$ and recovered humans (R(t) from the disease). Thus we have,

$$N_h(t) = S(t) + E(t) + Q(t) + A(t) + I(t) + I_D(t) + R(t).$$
(3)

And so adding equations in the system (1), then we have
$$N_h^{'}(t) = S^{'}(t) + E^{'}(t) + Q^{'}(t) + A^{'}(t) + I^{'}(t) + I_D^{'}(t) + R^{'}(t)$$

$$=\alpha-\lambda(1-\delta)(1-\epsilon)S+(1-\varphi)\beta_RR+\lambda(1-\delta)(1-\epsilon)S-(\sigma+\pi+\kappa)E+\varphi\beta_RR+\pi E+\tau A+\eta I\\ -(\gamma_k+\kappa)Q+\nu\sigma E-(\theta+\gamma_a+\mu+\tau+\kappa)A+(1-\nu)\sigma E-(\psi+\gamma_o+\eta+\kappa)I+\mu A\\ +\theta A+\psi I-(\gamma_i+\kappa)I_D+\gamma_iI_D+\gamma_aA+\gamma_oI+\gamma_kQ-\beta_RR$$

$$= \rho - d_E E - d_S A - d_O Q - \delta I_D - d_I I - d_R R \tag{4}$$

Letting
$$\phi = \{d_E, d_A, d_Q, d_D, d_I, d_R\}$$
, we have
$$N'_h(t) \le -(S + E + Q + A + I + I_d + R)\phi \tag{5}$$

and then equating N_h' to 0 since both S', E', Q', A', I', I'_d and R' all approach 0 as $t \to \infty$, then

Which gives

$$N_h \le N_h(0)e^{-\emptyset t} \tag{6}$$

From

$$\frac{dS}{dt} = \alpha - \frac{\beta c(1-\delta)(1-\varepsilon)(\alpha A + I)}{N_h - I_D}S - d_s S$$

We obtain

$$S(t) \leq \frac{\alpha}{(1-\delta)(1-\varepsilon)\lambda + d_s} \tag{7}$$

And hence the feasible region of our study for the equilibrium points is

region of our study for the equilibrium points is
$$\Omega = \{ (S, E, A, Q, I_D, I, R) \in R_+^4 | S \le \frac{\rho}{(1 - \delta)(1 - \epsilon)\lambda + d_S}, N_h \le N_h(0)e^{-\emptyset t}$$
(8)

It can be verified that Ω is positively invariant with respect to t

3.2Positivity and boundedness

For the model (1) to be epidemiologically meaningful, it is important to show that all its state variables are non-negative for all time (t) > 0 and that Ω is, indeed, bounded. We claim the following:

Theorem 1.Let the initial data for the model (1) be
$$S(0) \ge 0$$
, $S(0) \ge 0$, $E(0) \ge 0$,

Then the solutions (S, E, Q, A, I, I_D, R) of the model (1) are positive for all time t > 0.

Proof. Let

$$t_1 = \sup \sup\{t > 0: S > 0, E > 0, Q.0, A > 0, I > 0, I_D > 0, R > 0 \in [0, t]\}$$
 (9)

Thus, $t_1 > 0$.

We have, from the first equation of the system (1) that

$$\frac{dS}{dt} = \alpha - (\lambda(1 - \delta)(1 - \varepsilon) + d_s)S \tag{10}$$

where

$$\lambda = \frac{\beta c(p\beta_I I + \varepsilon_D \beta_D I_D)}{N}$$

which can be re-written as

$$\frac{dS}{dt} + (\lambda(1 - \delta)(1 - \varepsilon) + d_s)S = \rho \tag{11}$$

so that

$$S(t) = \exp \exp(-(\lambda + d_s)t) \left[\int_0^{t_1} \exp \exp((\lambda + d_s)t) \rho \, dt \right]$$

$$= \exp \exp(-(\lambda + d_s)t) \left[\rho \int_0^{t_1} \exp \exp((\lambda + d_s)\xi) \, d\xi \right]$$

$$= \exp \exp(-(\lambda + d_s)t) \left[\frac{\rho}{(\lambda + d_s)} \exp \exp((\lambda + d_s)\xi) \right]_0^{t_1}$$

$$= \frac{\rho}{(\lambda + d_s)} \exp \exp(-(\lambda + d_s)t) \left[\exp \exp((\lambda + d_s)t_1) - 1 \right]$$

$$S(t) = \frac{\rho}{(\lambda + d_s)} \left(1 - \exp \exp(-(\lambda + d_s)t) \right) + N_h(0)e^{-\theta t} \exp \exp(-(\lambda + d_s)t) \quad (12)$$

But

$$exp exp(-(\lambda + d_s)t) \equiv 1 \forall t, \lambda \geq d_s \geq 0$$

Therefore

$$S(t) \ge 0 \tag{13}$$

Similarly, it can be shown that: E > 0, A > 0, Q > 0, I > 0, $I_D > 0$, R > 0.

Lemma 1. The region $D = \{(S, E, Q, A, I, I_D, R) \in R_+^4 : N_h \le N_h(t)\}$ is positively-invariant for the model (8) and attracts all positive solutions of the model.

Proof. Adding all the equations of the model gives

$$N_h'(t) = S'(t) + E'(t) + Q'(t) + A'(t) + I'(t) + I_D'(t) + R'(t)$$
(14)

Recall

$$N_h'(t) = \rho - d_E E - d_S A - d_O Q - \delta I_D - d_I I$$

And that $\emptyset = \{d_E, d_A, d_Q, \delta, d_I\}$, which gives

$$N_h'(t) \le \rho - (S + E + Q + A + I + I_d + R)\phi \tag{15}$$

That is

$$N_h^{'} \leq \rho - N_h \phi$$

which can be re-written as

$$\frac{dN_h}{dt} + N_h \phi \le \rho \tag{16}$$

we let N be the solution of complementary part

$$\frac{dN}{dt} + N\phi = 0$$

$$N = \exp \exp(-\phi t)$$

Therefore,

$$N_h = u(t)N(t)$$

Hence

$$N_{h} \leq u(t)N(t) = \frac{\alpha}{\phi}(\exp\exp(\phi t) - 1)\exp\exp(-\phi t) = \frac{\alpha}{\phi}(1 - \exp\exp(-\phi t))$$

$$\Rightarrow N_{h} \leq \frac{\alpha}{\phi}(1 - \exp\exp(-\phi t)) + N_{h}\exp\exp(-\phi t) \quad (17)$$

 $N_h(t)$ approaches $\frac{\alpha}{\phi}$ as $t \to \infty$. Hence the region Ω attracts all solutions in R_+^7

3.3 Equilibrium Points

The equilibrium point of the system denotes a time when the rate of change of the population is zero. Thus, to obtain our equilibrium points we set

$$S'(t) = E'(t) = Q'(t) = A'(t) = I'(t) = I'_D(t) = R'(t) = 0$$
 (18)

and then solve the resulting system of non-linear equations below

$$\alpha - (1 - \delta)(1 - \varepsilon)\lambda S + (1 - \varphi)\beta_{R}R = 0$$

$$\beta c(1 - \delta)(1 - \varepsilon)\lambda S - \rho - (\sigma + \nu + \pi + \kappa + d_{E})E + \varphi\beta_{R}R = 0$$

$$\pi E + \tau A + \eta I - (\gamma_{k} + d_{Q})Q = 0$$

$$\nu E - (\theta + \gamma_{a} + \gamma_{k} + \tau + d_{A})A = 0$$

$$\sigma E - (\psi + \gamma_{o} + \eta + d_{I})I + \mu A = 0$$

$$\theta A + \psi I - (\gamma_{i} + \delta)I_{D} + \kappa E = 0$$

$$\gamma_{i}I_{D} + \gamma_{o}A + \gamma_{o}I + \gamma_{k}Q - (\beta_{R} + d_{R})R. = 0$$

Two cases may arise in an attempt to solve the above problem vis: Case

- (a): The Disease-Free Equilibrium Point (DFEP)
- (b): The Endemic Equilibrium Point (EEP)

Disease-Free Equilibrium Point: The DFEPwill occurs when $I = I_D = 0$. Therefore, applying system (19) become

$$\alpha - \lambda S + (1 - \varphi)\beta_{R}R = 0 \qquad (a)
\lambda S - (\sigma + \pi + d_{E})E + \varphi\beta_{R}R = 0 \quad b\pi E + \tau A + \eta I - (\gamma_{k} + d_{Q})Q = 0 \quad (b)
\nu\sigma E - (\theta + \gamma_{a} + \gamma_{k} + \tau + d_{A})A = 0 \qquad (c)
(1 - \nu)\sigma E + \gamma_{k}A - (\eta + \gamma_{o} + \psi + d_{A})I = 0 \qquad (d)
\theta A + \psi I + \kappa E - \gamma_{i} - d_{d}I_{D} = 0 \qquad (e)
\gamma_{o}I + \gamma_{a}A + \gamma_{k}Q + \gamma_{i}I_{D} - (\beta_{R} + d_{R})R. = 0 \qquad (f)$$

Equation (10) is identically satisfied by A = E = Q = R = 0 a becomes

$$S = \frac{\alpha}{(\lambda(1-\delta)(1-\varepsilon) + d_s)}$$
 (21)

Hence **DFE**

$$(S, E, Q, A, I, I_D, I, R) = \left(\frac{\alpha}{d_s}, 0, 0, 0, 0, 0, 0\right)$$
(22)

Case 2: The Endemic Equilibrium Point (EEP)

The necessary and sufficient condition for an endemic equilibrium

$$P^* = (S^*, E^*, Q^*, A^*, I_D^*, I^*, R^*)$$
(23)

to exist in the feasible region Ω is that

$$0 < S^* \le S$$

Where S and S^* are solutions from DFE and EEP respectively.

Now, endemic equilibrium point is obtained as solving the dynamics system of equations. We then have
$$S = \frac{\rho}{(\lambda + d_s)}, E = m_0 \frac{\lambda \rho}{(\lambda + d_s)} (1 + \Delta), A = m \frac{\lambda \rho}{(\lambda + d_s)} (1 + \Delta), I = n \frac{\lambda \rho}{(\lambda + d_s)} (1 + \Delta),$$

$$Q = n_1 \frac{\lambda \rho}{(\lambda + d_s)} (1 + \Delta), I_D = n_2 \frac{\lambda \rho}{(\lambda + d_s)} (1 + \Delta), R = \frac{\lambda \rho}{\beta_R (\lambda + d_s)} \Delta$$

where

$$\Delta = \frac{(\gamma_{o}n + \gamma_{k}n_{1} + \gamma_{i}n_{2} + n_{3})}{(d_{R} + (1 - \gamma_{o}n - \gamma_{k}n_{1} - \gamma_{i}n_{2} - n_{3})\beta_{R})}, m_{0} = \frac{1}{(\sigma + \pi + d_{E})}$$

$$m = \frac{v\sigma}{(\theta + \gamma_{a} + \mu + \tau + d_{A})(\sigma + \pi + d_{E})'}$$

$$n_{1} = \frac{\left(\pi + \eta n + \frac{v\sigma\tau}{(\theta + \gamma_{a} + \mu + \tau + d_{A})}\right)}{(\gamma_{k} + d_{Q})(\sigma + \pi + d_{E})}$$

$$n_{2} = \left(\frac{\theta v\sigma + \psi n(\theta + \gamma_{a} + \mu + \tau + d_{A})(\sigma + \pi + d_{E})}{(\theta + \gamma_{a} + \mu + \tau + d_{A})(\sigma + \pi + d_{E})(\gamma_{i} + \delta)}\right)$$

$$n_{3} = \frac{\gamma_{a}v\sigma}{(\theta + \gamma_{a} + \mu + \tau + d_{A})(\sigma + \pi + d_{E})}$$

It is important to say here that,

$$\Delta = \frac{(\gamma_o n + \gamma_k n_1 + \gamma_i n_2 + n_3)}{d_R + \beta_R (-1 + (\gamma_o n + \gamma_k n_1 + \gamma_i n_2 + n_3))}$$

$$R \ge 0 \ iff \quad d_R \ge (-1 + (\gamma_o n + \gamma_k n_1 + \gamma_i n_2 + n_3))\beta_R$$

That is

$$d_{R} \ge (-1 + (\gamma_{o}n + \gamma_{k}n_{1} + \gamma_{i}n_{2} + n_{3}))\beta_{R}$$

$$\beta_{R} \le \frac{d_{R}}{((\gamma_{o}n + \gamma_{k}n_{1} + \gamma_{i}n_{2} + n_{3}) - 1)}$$
(24)

Equation (24) implies the recovered population was prevented from exposure to the virus.

3.4The Basic Reproduction Number (BRN)

In epidemiology, the reproductive number, R_0 , is of utmost significance. It is the quantity of invectives a main infectious agent produces in a virgin population that is completely susceptible. It provides information about the disease's initial rate of growth over a generation. In other words, R_0 depends on the dynamics of the disease's transmission and assesses the infectious diseases capacity for growth ([24], [25]). Throughout the mechanics of the early epidemic growth, the value of R_0 is unchanged. Therefore, if $R_0 > 1$, the disease spreads

and finally becomes pandemic, if $R_0 < \overline{1}$, it eventually dies off and tends to zero, and if $R_0 = 0$, it is selfsustaining and the number of affected people stays constant.

Here, we demonstrate that R_0 is the fundamental proliferation number, defined by [5] as the normal number of optional contaminations causing by a single infectious in a completely helpless population during its entire period irresistible period, when the DFEP will be locally asymptomatically steady and the EEP will be temperamental i.e. the illness will cease to exist. However, when a strong individual will present more than one additional contamination during this time, the disease will continue to exist while the DFEP becomes shaky. When $R_0 = 1$, then the disease becomes endemic i.e the disease will remain dormant in the population at a constant rate. Thus the threshold quantity for eradicating the disease is to reduce the value of R_0 to be less than 1

Thus

$$0 < S^* < S$$

Implies

$$0 < 1 \le \frac{1}{S^*} \frac{\rho}{d_s} \Longrightarrow 0 < 1 \le R_0$$

$$S^* = \frac{\rho}{(\lambda(1-\delta)(1-\varepsilon) + d_s)}$$

Where

$$R_0 = \frac{1}{S^*} \frac{\rho}{d_s} = \frac{(\lambda(1-\delta)(1-\varepsilon) + d_s)}{d_s}$$

$$R_0 = \frac{(\lambda(1-\delta)(1-\varepsilon) + d_s)}{d_s}$$
(25)

Then the threshold quantity R_0 is then used to calculate the total number of equilibria.

Proposition 1. P_0 is the only equilibrium point in Ω if $R_0 \leq 1$, However if $R_0 > 1$ then there exist two equilibria, namely P_0 (which is the DFEP) and another unique equilibrium P^* which is the Endemic Equilibrium Point(EEP).

A quick check:

$$R_0 = \frac{(\lambda(1-\delta)(1-\varepsilon) + d_s)}{d_s} = \frac{\lambda(1-\delta)(1-\varepsilon)}{d_s} + 1 > 1$$
 (26)

That is $R_0 > 1$ which implies there will exist two equilibria.

4. Results and Discussions

4.1 Parameter Effects on the BRN

The carriers in the given system can have great effect on the basic reproduction number R_0 . The parameters λ , δ , ε and d_s are all related to the carrier class and they also appear in the BRN.

From equation (25), the rate of change of the dynamics with respect to
$$\lambda$$
 is computed as follows
$$\frac{\partial R_0}{\partial \lambda} = \frac{(1-\delta)(1-\varepsilon)}{d_{\varepsilon}} > 0 \text{ iff} \quad \delta, \varepsilon < 1 \text{ or } \delta, \varepsilon > 1$$
(27)

Otherwise,

$$\frac{\partial R_0}{\partial \lambda} < 0$$

Hence, increasing the rate of exposure λ will increase the BRN R_0 thus increasing the spread of the disease. Hence the analysis of the λ effect can be a useful control strategy in controlling the spread of

the effect of δ , in equation (25), we obtain (ii).

$$\frac{\partial R_0}{\partial \delta} = \frac{-\lambda(1-\varepsilon)}{d_s}$$

Since λ , ε and d_s are all positive numbers with $0 \le \varepsilon \le 1$, then $\varepsilon = 0$ imply

$$\frac{\partial R_0}{\partial \delta} = \frac{-\lambda}{d_s} < 0 \tag{28}$$

So it means that the rate of change R_0 with respect to δ decreases with for all values of λ and d_s The case when $\varepsilon = 1$ means R_0 is constant within the domain of existence

(iii). The effect of ε

$$\frac{\partial R_0}{\partial \varepsilon} = \frac{-\lambda (1 - \delta)}{d_{\varsigma}}$$

Since
$$\lambda$$
, δ and d_s are all positive numbers with $0 \le \varepsilon \le 1$, then $\delta \le 0$ imply
$$\frac{\partial R_0}{\partial \varepsilon} = \frac{-\lambda}{d_s} < 0 \tag{29}$$

So it means that the rate of change R_0 with respect to ε decreases with for all values of λ and d_s The case when $\varepsilon = 1$ means R_0 is constant within the domain of existence

The effect of d_s

$$\frac{\partial R_0}{\partial d_s} = -\frac{\lambda (1 - \delta)(1 - \varepsilon)}{d_s^2} \tag{30}$$

From the above, it could be seen that

$$\frac{\partial R_0}{\partial d_s} \le 0$$

4.2 Numerical simulations and results

In this section, we present numerical simulations for the model which were conducted using the rkf45 solvers coded in Maple programming Language and show the sensitive parameters on the dynamics of the disease. The main focus of the simulation study is to investigate the response of model parameters upon the covid-19 pandemic. The results will be shown graphically in order to investigate the dynamics of the disease and then proffer solution based on the numerical simulations as to which method will be more effective in eradicating the disease.

Baseline values of the parameters for model (1)

$$\alpha = 0.001, \varphi = 0.2. \cdot 0.6, \beta_R = 0.8, \pi = 1/120, \kappa = \frac{1}{60}, \mu = 0.02, \tau = 0.4, \delta = 0.1\beta c = 0.4236, p = \beta_I = 0.168, \varepsilon_D = \beta_D = 0.336, \varepsilon = 0.1, \eta = 1/14$$

$$\psi = 0.0088, \ \nu = 0.5, \gamma_k = 1/14, \gamma_i = 0.06, \gamma_a = \gamma_o = 0.13978, d_S = d_E = d_A = d_I = d_{I_d} = d_Q = d_R = 0.015, \theta = 0.00002, \sigma = \frac{1}{5.2},$$

$$(S(0), F(0), A(0), I(0), I(0), D(0), P(0)) = (3.67704, 50.30, 30.00)$$

$$(S(0), E(0), A(0), I(0), I_D(0), Q(0), R(0)) = (367704, 50, 30, 20, 0)$$

4.3 Discussions

Here, we discuss the behavior of the model. Under various computational simulations of the proposed model for state variables of interest, the transmission dynamics of an infectious disease can be adequately understood. The susceptible humans (S(t)), Quarantine on exposed humans (Q(t)), asymptomatic infectious humans (A(t)), symptomatic infectious humans (I(t)), detected infectious humans $(I_D(t))$ and recovered humans (R(t)) populations are investigated with different values of the parameters with an assumption that those in the I_D are completely isolated and do not come in contact with the general population.

In Figure 2, we discovered that an increase in the susceptible class triggers or boost the dynamics of Covid-19 while a decrease in the rate of $I_D(t)$ brings about declination to the dynamics of Covid-19. Figure 3 indicated that increase in the value of φ diminishes the rate of susceptible class. In Figure 4, an increase in the value of δ brings about an increase in susceptible class. Figures 5 depicts that an enhancement in the value of the parameters ρ enhances the rate of susceptible class while an increase in the value of λ in Figure 6 turns out to diminish the rate of susceptible humans while. We observed in Figure 7 that as ε increases, there is an increase in the rate of susceptible class.

In Figure 8, enhancing the value of ρ will bring about decrease in the rate of asymptomatic class while. In Figures 9 – 11, it is obvious that as the value of the parameters θ , μ and τ increases, the asymptomatic population decreases at each point while in Figures 12, an enhancement in the values of ρ declines the rate of infected

population. Increasing the value of μ in Figure 13 will enhance the rate of infected population. Figures 14 and 15 show that as $\psi and\eta$ increases, the infected population diminishes while figures 16 and 17 depicts that an enhancement in the value of $\theta and\psi$ increases the rate of detected-infected population.

Moreover, the impact of ρ and ν in figures 18 – 20 shows that as ρ , ν and π increases, the quarantine humans profile enhances but a decrease in the value of ρ , ν and π will diminish the rate of quarantine population. In figures 21, increasing the value of μ will bring about a declination in the rate of quarantine population. Figures 22, 23 and 25 indicated that an increase in the value of ρ , δ and β will bring about a decrease in the rate of recovery population while an enhancement in the value of μ in Figure 24 enhances the recovery population.

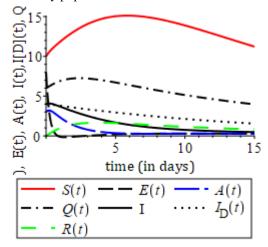


Figure 2: Covid-19 Dynamics with reference to subclass characteristics

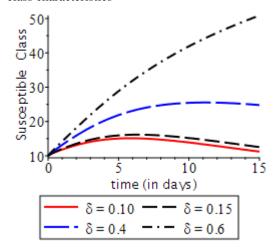


Figure 4: The different values of δ for the susceptible human sub-class

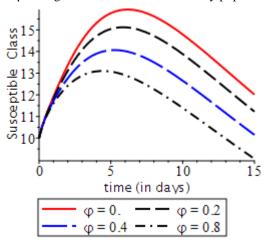


Figure 3: The different values of φ for the susceptible human sub-class

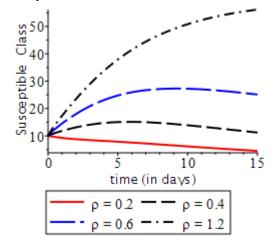


Figure 5: The different values of ρ for the susceptible human sub-class

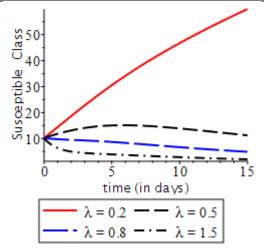


Figure 6: The different values of λ for the susceptible human sub-class

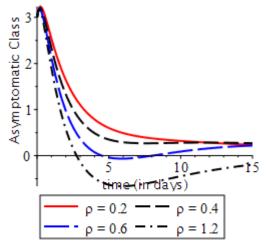


Figure 8: The different values of ρ for the asymptomatic human sub-class

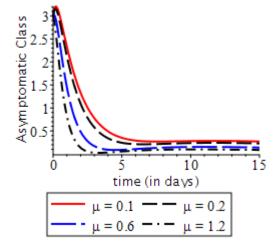


Figure 10: The different values of μ for the asymptomatic human sub-class

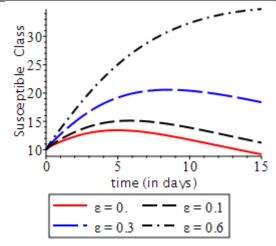


Figure 7: The different values of ε for the susceptible human sub-class

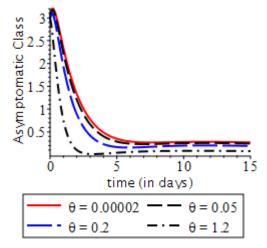


Figure 9: The different values of θ for the asymptomatic human sub-class

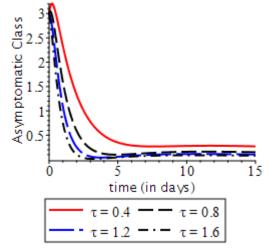


Figure 11: The different values of τ for the asymptomatic human sub-class

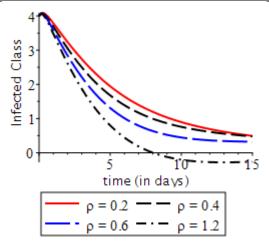


Figure 12: The different values of ρ for the infected human sub-class

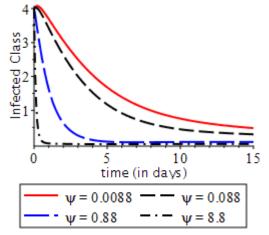


Figure 14: The different values of ψ for the infected human sub-class

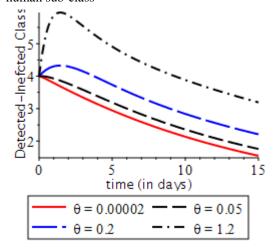


Figure 16: The different values of θ for the detected-infected human sub-class

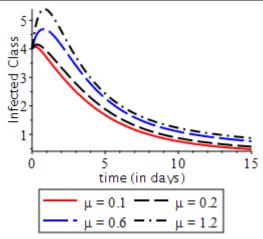


Figure 13: The different values of μ for the infected human sub-class

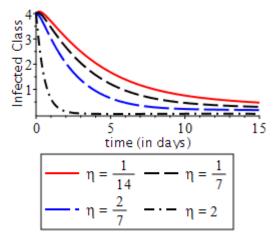


Figure 15: The different values of η for the infected human sub-class

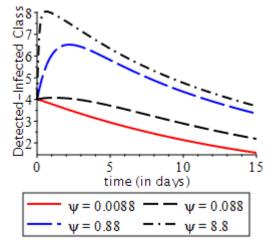


Figure 17: The different values of ψ for the detected-infected human sub-class

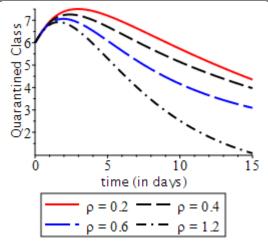


Figure 18: The different values of ρ for the quarantined human sub-class

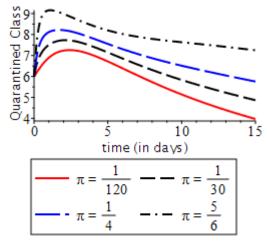


Figure 20: The different values of π for the quarantined human sub-class

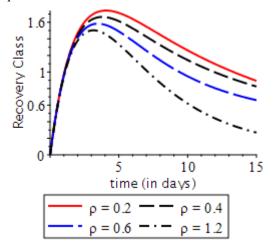


Figure 22: The different values of ρ for the recovery human sub-class

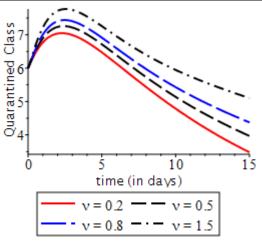


Figure 19: The different values of ν for the quarantined human sub-class

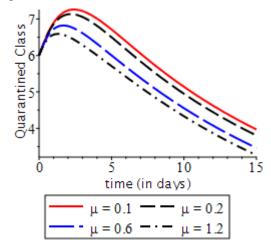


Figure 21: The different values of μ for the quarantined human sub-class

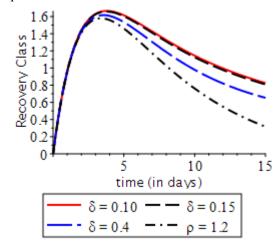
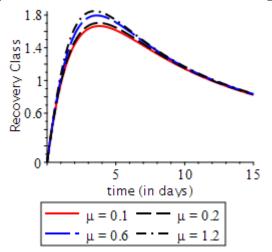


Figure 23: The different values of δ for the recovery human sub-class



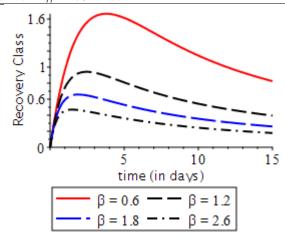


Figure 24: The different values of μ for the recovery human sub-class

Figure 25: The different values of β for the recovery human sub-class

5.1 Summary of findings

The SEIR model utilized in this research work shows the model of Covid infection transmission starting with one boundary of the populace then onto the next. This proposal recognized the impact of the model and how it uncovers that Covid won't be annihilated however the transmission elements will help our forecasts on control.

5.2 Conclusion

This study gives an understanding on the transmission models and control of Covid. The SEIR model was utilized subsequently we set up an expectation that Covid-19 has come to remain however transmission can be controlled. The multiplication number was acquired by the utilization of cutting-edge grid and the investigation of soundness got for the mode the transmission elements of all boundary were viewed as which gives more understanding the rate at which individuals move from one populace boundary to the next concerning time.

5.3 Recommendation

More exposure and awareness on the preventive measures and control is required to diminish the spread and conceivably limit the impact of the infection within the briefest conceivable time.

Nomenclature: Description of variables and parameters in the model of this dissertation.

Parameter	Interpretation
S	Susceptible humans
E	Exposed humans (infected but not infectious and show no sign of disease)
Q	Quarantined humans' subclass
Α	Asymptomatically-infectious humans (undetected)
I	Symptomatically-infectious humans (undetected)
I_D	Detected infectious humans (asymptomatic and symptomatic) via testing
R	Recovered humans
σ	Progression rate from exposed state to infectious state
ν	Fraction of new infectious humans that are asymptomatic
α	Modification parameter that accounts for the reduced infectiousness of humans in the A class when compared to humans in the / class

γ_a	Recovery rates for individuals in the $A.I$ and I_D classes respectively
ψ	detection rate (via contact tracing and testing) for the I class
θ	detection rate (via contact tracing and testing) for the A class
$d_{()}$	Disease induced death rates for individuals in the/and I class respectively
$oldsymbol{eta}_c$	Effective transmission rate
$oldsymbol{eta}_{\scriptscriptstyle R}$	Rate of recovered that are exposed
$oldsymbol{eta}_{I}$	Rate of symptomatically infectious individuals that are exposed
$\boldsymbol{\beta}_{\scriptscriptstyle D}$	Rate of detected individuals that are exposed
π	Rate of exposed population that accept quarantine
μ	Fraction of exposed that became asymptomatic
τ	Rate of asymptomatically exposed to quarantined
δ	Proportion that maintains minimum distance
η	Rate of Infected symptomatic that are quarantined
λ	Force of infection
arepsilon	The rate of Quarantined individuals to become susceptible to the virus again
κ	Removal rate of infected detected cases
ho	detected individuals in relation to symptomatic (infected) individuals.
arphi	Rate at which the detected infected individuals move from severe to mild isolation
\mathcal{E}_D	The rate of Quarantined individuals that are detected with the virus.
p	The proportion at which infected symptomatic individual are generated from the exposed class.
()	S, E, A, I, I_D, Q, R

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