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Modeling the Spread of Coronavirus With Self-protection and Quarantine Effect

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Abstract

A nonlinear mathematical model to study the effect of transmission dynamics of COVID-19 virus in a population with variable size structure is proposed and analyzed. The model divides the total human population into five subclasses: susceptibles, self-protected susceptibles, infectives, quarantined infectives, and recovered population including a class representing cumulative density of coronavirus in the environmental reservoir. The model exhibits two equilibria, namely, the disease-free and the endemic equilibrium. Model analysis reveals the global dynamics of the spread of COVID-19 is completely determined by the basic reproduction number. If basic reproduction number is greater than one, the endemic equilibrium is locally asymptotically stable and is globally asymptotically stable under certain conditions showing that the disease becomes endemic. It is found that the infective population can be decreased if the individuals from susceptible population self protect themselves and do not come in direct contact with viral density deposited on surfaces or airborne droplets accumulated in the environmental reservoir. However, if higher number of individuals from infective class are quarantined at home or hospital, the spread of the disease can further be slowed down. Numerical analysis of the model is also performed to investigate the influence of certain key parameters on the spread of the disease and to support the analytical results.

Keywords: COVID-19; Quarantine; Self-protection; Reproduction number; Local stability; Global stability; Sensitivity

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1. Introduction

COVID-19, an infectious disease which has severely affected the entire world in different forms, is caused by the coronavirus SARS-CoV-2. An outbreak of the COVID-19 disease was first established in Wuhan, China and eventually invaded almost every nation due to fast modern air transportation. According to the World Health Organization (WHO) report, more than 131,472,605 people were infected worldwide and about 2,861,373 people were expired due to COVID-19 till April 4, 2021. In India, the first confirmed case was reported in March 2020, and as of April 4, 2021, approximately 12,392,260 cases were detected and 164,110 people died due to COVID-19 disease (Corona Virus Statistics (2020)).

The fast spread of the disease and lack of approved medicines made it a challenging problem for public health. Despite the fact that numerous vaccines were created in order to address the challenge, the inherent mutability of the coronavirus made the situation much more difficult to manage. Therefore, the focus was shifted to study the impact of non-pharmaceutical interventions such as lock down, isolation, social and physical distancing, avoidance of public gatherings, sanitization, quarantine of infectives, etc., to prevent further escalation of the disease.

Mathematical models play an important role in the study of transmission of any infectious disease for short-and long-term prediction of disease incidence. In the past few decades, several researchers have developed various mathematical models to investigate the transmission dynamics of infectious diseases and their control measures (Tripathi et al. (2007); Chavez and Song (2004); Brauer (1990); Aris (2012); Naresh et al. (2009); Singh et al. (2021)). These models try to accommodate the effects of various parameters on the spread of a disease, such as the presence of a disease vector, the phenomenon of relapse and reinfection, symptomatic and asymptomatic cases, analysis of the success of interventions with limited costs, etc., others and predicting the behavior of the epidemic and its short- and long-term effects.

Since COVID-19 is a recent pandemic and has rapidly spread in many countries across the globe, few mathematical studies have been conducted (Yang and Wang (2020); Ngonghala et al. (2020); Gurmu et al. (2020); Saldana et al. (2020); Sarkar et al. (2020); Obsu and Balcha (2020); Hu and Nie (2020); Pang et al (2020); Shao et al. (2020); Khanjanchi and Sarkar (2020); Bugalia et al. (2020); Chen et al. (2020); Sardar et al. (2020); Li et al. (2020)) to capture the transmission mechanism of COVID-19 and the effect of preventive measures. Chen et al. (2020) developed a Bats-Hosts-Reservoir-People transmission network model for simulating the potential transmission from the infection source (probably bats) to humans. Yang et al. (2020) proposed a mathematical model for COVID-19 incorporating multiple transmission pathways, including both human-to-human and environment-to-human transmission routes. They employed a bilinear incidence rate based on the law of mass action and fitted the model with Wuhan city (China) data and estimated the reproduction number. Li et al. (2020) proposed a model based on the transmission mechanism of COVID-19 in the population and implemented prevention and control measures. Ngonghala et al. (2020) developed a model of COVID-19 pandemic in US (particularly, in New York) for assessing the population-level impact of the mitigation strategies. The authors performed the rigorous analysis of the model and the impact of non-pharmaceutical intervention strategies. Garba et al.

(2020) proposed a compartmental model to analyze the dynamics of COVID-19 in South Africa. The model system (Sarkar et al. (2020)) was used to estimate the effect of mitigation strategies. Their study indicates the disease may die out if control measures are implemented early for a sustainable period of time. However, the effectiveness of self-isolation reduces the number of cases.

Sarkar et al. (2020) studied the dynamics of COVID-19 in India along with its 17 provinces and suggested that the contact rate between susceptible and infected individuals could be reduced by a strict isolation imposed on susceptible individuals. Moreover, the complete elimination of COVID-19 is possible via suitable combination of contact tracing and restrictive social distancing but it depends largely on how and when precautionary measures, isolation, and quarantine strategies are enforced. Bugalia et al. (2020) proposed a model to investigate the role of intervention strategies including lockdown on the transmission dynamics of COVID-19. They found that faster testing, to identify the infection quickly so that the infected individuals do not spread the disease further, can restrict the disease up to a certain level. Gurmu et al. (2020) analyzed a model and found the parameter which has high impact to decrease the disease in the community. In this direction, Sardar et al. (2020) also considered a mathematical model on COVID-19 to analyze the impact of social distancing and lockdown. Pang et al. (2020) presented a model to understand the transmission dynamics and control strategies of COVID-19 in Wuhan, China and shown that early control measures can prevent a larger outbreak of COVID-19. Obsu and Balcha (2020) performed the optimal control strategies for the transmission risk of COVID-19 and shown that the comprehensive impacts of prevention, intensive medical care and surface disinfection strategies outperform in reducing the disease epidemic with optimum implementation cost.

It may be noted that providing medical attention in hospitals in the era of pandemic to all COVID-19 affected persons was found to be a challenging task. It is, therefore, necessary that vulnerable individuals be advised to follow COVID-19 guidelines/Standard Operating Procedure (SOP) to self protect themselves. Thus, we have assumed that a large number of susceptibles follow COVID appropriate behavior and self protect themselves. This allows us to introduce a separate class of self-protected susceptibles who strictly follow the COVID protocal such as applying face cover/mask in public places, adopt social distancing, avoid public gatherings, wash hands frequently etc. and other guidelines issued from time to time. The objective of the present study is to understand the impact of self-protection, quarantine strategy and sanitization at public places towards minimizing the escalation of transmission of COVID-19.

The structure of the paper is organized as follows. In Section 2, we describe the model formulation, non-negativity and boundedness of solutions. The basic reproduction number with respect to model parameters is computed in Section 3. Section 4 provides the existence of equilibrium points and stability analysis. In Section 5, we present numerical simulations and discussions followed by conclusion in Section 6.

2. Mathematical Model

Consider a human population of size $N(t)$ at time t with constant immigration of susceptibles at a rate Λ. The total population size $N(t)$ is divided into five subclasses of susceptibles $X(t)$, self-protected susceptibles $Y(t)$, infectives $I(t)$, quarantined $Q(t)$ and the recovered class $R(t)$. Self-protected susceptibles $Y(t)$ constitute the segment of population who are assumed to strictly follow the COVID-19 guidelines/Standard Operating Procedure (SOP) such as applying face cover/mask in public place, maintaining social distancing, avoiding public gatherings, washing hands frequently. The infectives $I(t)$ are assumed to be infectious with strong infectivity but are not quarantined. The quarantined class $Q(t)$ consists of individuals who are diagnosed to be infected and have been quarantined either at home or at a hospital, due to the fact that they are isolated and, as a result, do not contribute to the spread of viruses. The class of recovered individuals from COVID-19 disease is represented by $R(t)$. The class $V(t)$ denotes the cumulative density of corona virus in the environmental reservoir.

We assume that susceptibles $X(t)$ become infected when they come in direct contact with infectives $I(t)$ with a transmission rate β as well as by exposure to viral density $V(t)$ deposited on surfaces/objects or airborne droplets accumulated in the environmental reservoir with transmission rate λ_1. The constant d is the natural mortality rate in all the classes, l is the rate by which susceptible individuals join the self-protected susceptible class whereas μ is the rate by which self-protected susceptibles loose their protection and again become susceptible to join the susceptible class. The increase in quarantined class is assumed to be directly proportional to the infective class where constant δ denotes the rate of transfer of infectives into quarantined class. The quarantined individuals after recovery move to recovered class with a rate constant η. It is also assumed that some of the infectives recover with self medication without being quarantined and join the recovered class with a rate ρ.

It is further assumed that some of the recovered individuals remain vulnerable to COVID-19 infection and become susceptible to increase the population of susceptibles with a rate constant ν. The constant α denotes the disease-induced death rate of infective and quarantined individuals. The growth of viral density $V(t)$ in the environmental reservoir is assumed to be directly proportional to the infectives where γ is the rate of increase of V. The constant σ is the rate by which viral density declines due to control/preventive measures like mass sanitization in the environment. The schematic diagram of disease transmission is shown in Figure 1.

With the above assumptions and considerations, the dynamics of viral transmission is assumed to be governed by the following system of nonlinear ordinary differential equations,

$$\frac{dX(t)}{dt} = \Lambda - \frac{\beta XI}{N} - \lambda_1 VX - dX - lX + \mu Y + \nu R, \tag{1}$$

$$\frac{dY(t)}{dt} = lX - (\mu + d)Y, \tag{2}$$

$$\frac{dI(t)}{dt} = \frac{\beta XI}{N} + \lambda_1 VX - (\delta + \rho + \alpha + d)I, \tag{3}$$

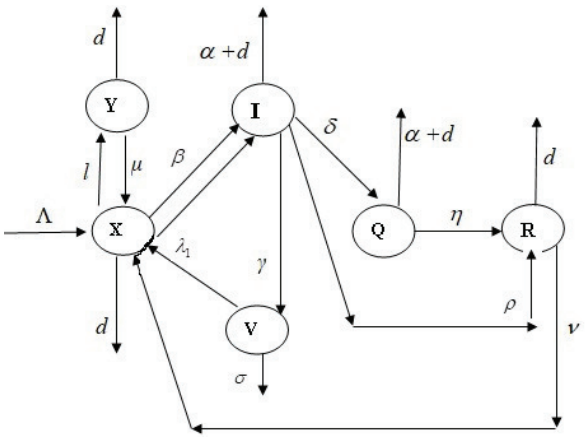

Figure 1. Schematic diagram of the model system (1) - (6)

$$\frac{dQ(t)}{dt} = \delta I - (\eta + \alpha + d)Q, \tag{4}$$

$$\frac{dR(t)}{dt} = \eta Q + \rho I - (\nu + d)R, \tag{5}$$

$$\frac{dV(t)}{dt} = \gamma I - \sigma V, \tag{6}$$

with initial conditions $X(0) = X_0 \geq 0, Y(0) = Y_0 \geq 0, I(0) = I_0 \geq 0, Q(0) = Q_0 \geq 0, R(0) = R_0 \geq 0$, and $V(0) = V_0 \geq 0$.

For convenience, let us take $b = (\mu + d)$, $c = (\delta + \rho + \alpha + d)$, $e = (\eta + \alpha + d)$. The above equations can now be written as follows, using $N = X + Y + I + Q + R$,

$$\frac{dN(t)}{dt} = \Lambda - dN - \alpha I - \alpha Q, \tag{7}$$

$$\frac{dY(t)}{dt} = l(N - Y - I - Q - R) - bY, \tag{8}$$

$$\frac{dI(t)}{dt} = (\frac{\beta I}{N} + \lambda_1 V)(N - Y - I - Q - R) - cI, \tag{9}$$

$$\frac{dQ(t)}{dt} = \delta I - eQ, \tag{10}$$

$$\frac{dR(t)}{dt} = \eta Q + \rho I - (\nu + d)R, \tag{11}$$

$$\frac{dV(t)}{dt} = \gamma I - \sigma V. \tag{12}$$

2.1. Non-negativity of Solutions

An important feature of any epidemiological model is to show that all the population variables are nonnegative for all $t \geq 0$, which implies that any trajectory starting with positive initial condition will remain positive for $t \geq 0$. The following lemma describes this fact,

Lemma 2.1.

If $X(0) \geq 0$, $Y(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0$ and $V(0) \geq 0$, the solution of $X(t)$, $Y(t)$, $I(t)$, $Q(t)$, $R(t)$ and $V(t)$ in the system (1) - (6) remain positive.

Proof:

We shall prove this lemma using contradiction by assuming that the total population $N(t) \neq 0$ for all $t \geq 0$.

Positivity of X(t):

Assume that $X(t_1) = 0$, $\frac{dX(t_1)}{dt} < 0$, $X(0) \geq 0$, $Y(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0$ and $V(0) \geq 0$, $0 \leq t \leq t_1$,

$$\frac{dX(t_1)}{dt} < 0,$$

$$\begin{aligned}\left(\frac{dX(t_1)}{dt}\right)_{t=t_1} &= \Lambda - \frac{\beta I(t_1)X(t_1)}{N(t_1)} - \lambda_1 V(t_1)X(t_1) - (d+l)X(t_1) + \mu Y(t_1) + \nu R(t_1) \\ &= \Lambda + \mu Y(t_1) + \nu R(t_1) \leq 0,\end{aligned}$$

which is a contradiction as $\Lambda + \mu Y(t_1) + \nu R(t_1) > 0$. Hence, it can be concluded that $X(t) \geq 0$ for $t \geq 0$.

Positivity of Y(t):

Assume that $Y(t_2) = 0$, $\frac{dY(t_2)}{dt} < 0$, $X(0) \geq 0$, $Y(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0$ and $V(0) \geq 0$, $0 \leq t \leq t_2$,

$$\frac{dY(t_2)}{dt} < 0,$$

$$\begin{aligned}\left(\frac{dY(t_2)}{dt}\right)_{t=t_2} &= lX(t_2) - (\mu + d)Y(t_2) \\ &= lX(t_2) \leq 0,\end{aligned}$$

which is a contradiction as $lX(t_2) > 0$. Hence, it can be concluded that $Y(t) \geq 0$ for $t \geq 0$.

Positivity of I(t):

Assume that $I(t_3) = 0$, $\frac{dI(t_3)}{dt} < 0$, $X(0) \geq 0$, $Y(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0$ and $V(0) \geq 0$, $0 \leq t \leq t_3$,

$$\frac{dI(t_3)}{dt} < 0,$$

$$\begin{aligned}\left(\frac{dI(t_3)}{dt}\right)_{t=t_3} &= \frac{\beta I(t_3)X(t_3)}{N(t_3)} + \lambda_1 V(t_3)X(t_3) - (\delta + \rho + \alpha + d)I(t_3) \\ &= \frac{\beta I(t_3)X(t_3)}{N(t_3)} + \lambda_1 V(t_3)X(t_3) \leq 0,\end{aligned}$$

which is a contradiction as $\frac{\beta I(t_3)X(t_3)}{N(t_3)} + \lambda_1 V(t_3)X(t_3) > 0$. Hence, it can be concluded that $I(t) \geq 0$ for $t \geq 0$.

Positivity of Q(t):

Assume that $Q(t_4) = 0$, $\frac{dQ(t_4)}{dt} < 0$, $X(0) \geq 0$, $Y(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0$ and $V(0) \geq 0$, $0 \leq t \leq t_4$,

$$\frac{dQ(t_4)}{dt} < 0,$$

$$\begin{aligned}\left(\frac{dI(t_4)}{dt}\right)_{t=t_4} &= \delta I(t_4) - (\eta + \alpha + d)Q(t_4) \\ &= \delta I(t_4) \leq 0,\end{aligned}$$

which is a contradiction as $\delta I(t_4) > 0$. Hence, it can be concluded that $Q(t) \geq 0$ for $t \geq 0$.

Positivity of R(t):

Assume that $R(t_5) = 0$, $\frac{dR(t_5)}{dt} < 0$, $X(0) \geq 0$, $Y(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0$ and $V(0) \geq 0$, $0 \leq t \leq t_5$,

$$\frac{dR(t_5)}{dt} < 0,$$

$$\begin{aligned}\left(\frac{dR(t_5)}{dt}\right)_{t=t_5} &= \eta Q(t_5) + \rho I(t_5) - (\nu + d)\, R(t_5) \\ &= \eta Q(t_5) + \rho I(t_5) \leq 0,\end{aligned}$$

which is a contradiction as $\eta Q(t_5) + \rho I(t_5) > 0$. Hence, it can be concluded that $R(t) \geq 0$ for $t \geq 0$.

Positivity of V(t):

Assume that $V(t_6) = 0$, $\frac{dV(t_6)}{dt} < 0$, $X(0) \geq 0$, $Y(0) \geq 0$, $I(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0$ and $V(0) \geq 0$, $0 \leq t \leq t_6$,

$$\frac{dV(t_6)}{dt} < 0,$$

$$\begin{aligned}\left(\frac{dV(t_6)}{dt}\right)_{t=t_6} &= \gamma I(t_6) - \sigma V(t_6) \\ &= \gamma I(t_6) \leq 0,\end{aligned}$$

which is contradiction as $\gamma I(t_6) > 0$. Hence, it can be concluded that $V(t) \geq 0$ for $t \geq 0$. ■

3. Computation of Basic Reproduction Number

The basic reproduction number R_0, defined as the effective number of secondary infectives generated by a primary infected individual. We calculate R_0 by closely following the approach in Driessche and Watmough (2002, 2008). We first compute the new infectious matrix F and transfer matrix W (Diekmann et al. (2010)), according to formula

$$[F - W] = \begin{bmatrix} \frac{\partial(dI/dt)}{\partial I} & \frac{\partial(dI/dt)}{\partial V} \\ \frac{\partial(dV/dt)}{\partial I} & \frac{\partial(dV/dt)}{\partial V} \end{bmatrix}. \tag{13}$$

To calculate F and W, we only consider Equations (9) and (12), which correspond to the groups (I, V) capable of transmitting the disease. The non-negative matrix F, corresponding to new infections in the population at disease-free equilibrium is,

$$F = \begin{bmatrix} \frac{b\beta}{(l+b)} & \frac{\lambda_1 \Lambda}{d} \frac{b}{(l+b)} \\ 0 & 0 \end{bmatrix}. \tag{14}$$

The non-singular matrix W, corresponding to the transfer of individuals into and out of compartment is,

$$W = \begin{bmatrix} c & 0 \\ -\gamma & \sigma \end{bmatrix} \tag{15}$$

where W^{-1} is given by $W^{-1} = \frac{1}{c\sigma} \begin{bmatrix} \sigma & 0 \\ \gamma & c \end{bmatrix}$.

Here, FW^{-1} is the next generation matrix of the system (7) - (12).

$$FW^{-1} = \frac{1}{c\sigma} \begin{bmatrix} \frac{b\beta\sigma}{(l+b)} + \frac{\lambda_1 \Lambda}{d} \frac{b\gamma}{(l+b)} & \frac{\lambda_1 \Lambda}{d} \frac{cb}{(l+b)} \\ 0 & 0 \end{bmatrix},$$

$$\rho(FW^{-1}) = \frac{1}{c\sigma}\left(\beta\sigma + \frac{\lambda_1\Lambda}{d}\gamma\right)\frac{b}{(l+b)},$$

$$R_0 = \frac{\beta}{c}\frac{b}{(l+b)} + \frac{\lambda_1\Lambda}{d}\frac{\gamma}{c\sigma}\frac{b}{(l+b)}. \tag{16}$$

According to van den Driessche and Watmough (2002, 2008), the basic reproduction number (after putting value of b and c) of the system (7) - (12) is

$$R_0 = \left[\frac{\beta}{(\delta+\rho+d+\alpha)}\frac{(\mu+d)}{(l+\mu+d)} + \frac{\lambda_1\Lambda\gamma}{d(\delta+\rho+d+\alpha)\sigma}\frac{(\mu+d)}{(l+\mu+d)}\right]. \tag{17}$$

It is possible to rewrite the expression of R_0 to account for the source of infection as follows,

$$R_0 = R_{Infectives} + R_{virus}.$$

This expression shows infections resulting from two sources, namely, the infectives and the virus. Increasing the value of denominator reduces the reproduction number. Since the parameters α, d (disease-induced death rate due to COVID-19 and natural death rate respectively) and ρ (depends upon the immune system of the infectives) cannot be increased, the reproduction number can be reduced by increasing δ , the growth rate of quarantined class. This implies that if higher number of infectives are quarantined, the spread of COVID-19 infection can be reduced. Further, the reproduction number R_0 can also be decreased by increasing the values of l and σ. As a result, this suggests that if a greater number of vulnerable persons adhere to the COVID-19 criteria in a stringent manner, then the population of individuals who are self-protected will increase, which will result in a reduction in the transmission of infection. Moreover, with increase in the rate of elimination of viral density due to sanitization and other preventive measures, the reproduction number due to virus decreases and hence the spread of disease is reduced.

If $R_0 < 1$, then, on average an infected individual produces less than one infected individual over the course of its infectious period and hence infection cannot grow. Conversely, if $R_0 > 1$, then, on average an infected individual produces more than one new infection and the disease can invade the population.

3.1. Sensitivity Analysis

Sensitivity indices allow us to measure the relative change in a variable when a parameter changes. The derivatives are the rate of change of predictions with respect to a parameter. This work adopts the normalized forward sensitivity index to conduct the sensitivity analysis (Gurmu et al. (2020); Pang et al. (2020); Marino (2008)). The normalized forward sensitivity index of a variable with respect to a parameter is the ratio of relative change in the parameter. When variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial

derivative. For instance, the normalized forward sensitivity index is,

$$Y_M^{R_0} = \frac{\partial R_0}{\partial M} \times \frac{M}{R_0}.$$

The parameter values displayed in the Table 1 below are taken as the baseline and they are used to evaluate the sensitivity indices of some parameters which are responsible for the transmission dynamics of COVID-19 disease to four places of decimal in relation to the effective reproduction number R_0 using Equation (17). The results so obtained are given in Table 1,

$$R_0 = \left[\frac{\beta}{(\delta + \rho + d + \alpha)} \frac{(\mu + d)}{(l + \mu + d)} + \frac{\lambda_1 \Lambda \gamma}{d(\delta + \rho + d + \alpha)\sigma} \frac{(\mu + d)}{(l + \mu + d)} \right],$$

$$\frac{\partial R_0}{\partial \beta} \frac{\beta}{R_0} = \frac{\beta d \sigma}{\beta d \sigma + \lambda_1 \Lambda \gamma}, \quad \frac{\partial R_0}{\partial \lambda_1} \frac{\lambda_1}{R_0} = \frac{\lambda_1 \Lambda \gamma}{\beta d \sigma + \lambda_1 \Lambda \gamma}, \quad \frac{\partial R_0}{\partial \gamma} \frac{\gamma}{R_0} = \frac{\lambda_1 \Lambda \gamma}{\beta d \sigma + \lambda_1 \Lambda \gamma},$$

$$\frac{\partial R_0}{\partial \sigma} \frac{\sigma}{R_0} = -\frac{\lambda_1 \Lambda \gamma}{(\beta d \sigma + \lambda_1 \Lambda \gamma)}, \quad \frac{\partial R_0}{\partial \delta} \frac{\delta}{R_0} = -\frac{\delta}{(\delta + \alpha + d)}, \quad \frac{\partial R_0}{\partial l} \frac{l}{R_0} = -\frac{l}{(l + \mu + d)},$$

$$\frac{\partial R_0}{\partial \mu} \frac{\mu}{R_0} = \frac{l\mu}{(l + \mu + d)(\mu + d)}.$$

From Table 1, we can see that the positive indices, i.e., β, λ_1, γ and μ, show the great impact on expanding the disease in the population, since R_0 increases with increase in their values. Further, the parameter σ, δ and l, for which the sensitivity indices is negative, show that increasing σ, δ and l leads to decrease the basic reproduction number, which ultimately reduces the spread of disease in the population.

Table 1. Sensitivity indices

Parameter Symbol	Sensitivity indices
β	1.9x10^{-7}
λ_1	0.9999
γ	0.9999
σ	-0.9999
δ	-0.9861
l	-0.9705
μ	0.9695

4. Equilibria and Stability Analysis of the Model

4.1. Equilibria of the Model

The model (7) - (12) has two non-negative equilibria namely,

(i) $E_0\left(\frac{\Lambda}{d}, \frac{\Lambda l}{d(l+b)}, 0, 0, 0, 0\right)$, the disease-free equilibrium, which exists without any condition. This equilibrium implies that in the absence of any infection, the total population size remains at its equilibrium value Λ/d.

(ii) $E^*(N^*, Y^*, I^*, Q^*, R^*, V^*)$, the endemic equilibrium. The equilibrium values of different variables are given as,

$$Q^* = \frac{\delta(\Lambda - dN)}{\alpha\,(e+\delta)}, V^* = \frac{\gamma e(\Lambda - dN)}{\sigma\alpha(e+\delta)}, R^* = \frac{(\eta\delta + \rho e)\,(\Lambda - dN)}{(\nu + d)\,\alpha(e+\delta)}, I^* = \frac{e(\Lambda - dN)}{\alpha(e+\delta)},$$

$$Y^* = \frac{l}{(l+b)}\left[N - \left(e + \delta + \frac{\eta\delta + \rho e}{\nu + d}\right)\frac{(\Lambda - dN)}{\alpha\,(e+\delta)}\right], \tag{18}$$

and we get a quadratic equation in N,

$$\frac{b}{l+b}\left(\beta + \frac{\lambda\gamma}{\sigma}N\right)\left[N - \left(e + \delta + \frac{\eta\delta + \rho e}{\nu + d}\right)\frac{(\Lambda - dN)}{\alpha\,(e+\delta)}\right] - cN = 0. \tag{19}$$

To show the existence of E^*, Equation (19) can be written as,

$$F(N) = \frac{b}{l+b}\left(\beta + \frac{\lambda\gamma}{\sigma}N\right)\left[N - \left(e + \delta + \frac{\eta\delta + \rho e}{\nu + d}\right)\frac{(\Lambda - dN)}{\alpha\,(e+\delta)}\right] - cN = 0. \tag{20}$$

It would be sufficient if we show that $F(N)$ = 0 has one and only one positive root. To prove this, from Equation (20) we have,

$$F(0) = -\frac{\beta b}{l+b}\left(e + \delta + \frac{\eta\delta + \rho e}{\nu + d}\right)\frac{\Lambda}{\alpha\,(e+\delta)} < 0, \tag{21}$$

$$F\left(\frac{\Lambda}{d}\right) = c\left[\frac{b}{l+b}\left(\frac{\beta}{c} + \frac{\lambda\gamma}{\sigma c}\frac{\Lambda}{d}\right) - 1\right]\frac{\Lambda}{d} > 0,$$

$$F\left(\frac{\Lambda}{d}\right) = c\,[R_0 - 1]\,\frac{\Lambda}{d} > 0. \tag{22}$$

Thus, if $R_0 > 1$ then $F(\frac{\Lambda}{d}) > 0$. Using Equations (17) and (22), we can see that if l, the rate of increase of self- protected susceptibles is sufficiently large, the reproduction number $R_0 < 1$ and hence the endemic equilibrium does not exist,

$$F^{'}(N) = \frac{b}{l+b}\left[\begin{array}{r}\frac{\lambda\gamma}{\sigma}\left\{N - \left(e + \delta + \frac{\eta\delta+\rho e}{\nu+d}\right)\frac{(\Lambda - dN)}{\alpha(e+\delta)}\right\} \\ + \left(\beta + \frac{\lambda\gamma}{\sigma}N\right)\left\{1 + \left(e + \delta + \frac{\eta\delta+\rho e}{\nu+d}\right)\frac{d}{\alpha(e+\delta)}\right\}\end{array}\right] - c. \tag{23}$$

It is noted that $F'(N) > 0$, provided

$$\frac{b}{l+b}[\frac{\lambda\gamma}{\sigma}(N-(e+\delta+\frac{\eta\delta+\rho e}{\nu+d})\frac{(\Lambda-dN)}{\alpha(e+\delta)})+(\beta+\frac{\lambda\gamma}{\sigma}N)(1+\left(e+\delta+\frac{\eta\delta+\rho e}{\nu+d}\right)\frac{d}{\alpha(e+\delta)})] > c.$$

Thus, $F(N) = 0$ has exactly one root (say N^*) between 0 and $\frac{\Lambda}{d}$. Using the value of N^*, the values of Y^*, I^*, Q^*, R^* and V^* can be found from Equation (18).

4.2. Local Stability of the Equilibria

To determine the local stability of E_0, the following variational matrix of the system (7) - (12) is computed about E_0 as,

$$J(E_0) = \begin{bmatrix} -d & 0 & -\alpha & -\alpha & 0 & 0 \\ l & -(l+b) & -l & -l & -l & 0 \\ 0 & 0 & -(c-\beta w) & 0 & 0 & \frac{\lambda_1\Lambda w}{d} \\ 0 & 0 & \delta & -e & 0 & 0 \\ 0 & 0 & \rho & \eta & -(\nu+d) & 0 \\ 0 & 0 & \gamma & 0 & 0 & -\sigma \end{bmatrix},$$

where $w = \frac{b}{l+b}$,

The four roots of the characteristic equation are $\lambda = -d,\ \lambda = -(l+b)$, $\lambda = -(\nu+d)$, $\lambda = -e$. The other two roots of the variational matrix are determined by the following equation,

$$f(\lambda) = \lambda^2 + (c+\sigma-\beta w)\lambda + c\sigma - \sigma\beta w - \frac{\Lambda\lambda_1\gamma w}{d} = 0, \tag{24}$$

$$f(\lambda) = \lambda^2 + (c+\sigma-\beta w)\lambda + c\sigma(1-R_0) = 0. \tag{25}$$

We can see if $R_0 > 1$, then $J(E_0)$ has at least one eigenvalue with positive real part. Therefore, disease-free equilibrium E_0 of the system (7) - (12) is locally asymptotically stable if $R_0 < 1$. Under this condition disease dies out, i.e., infection does not persist in the population. For $R_0 > 1$ the disease free equilibrium is locally unstable and the endemic euilibriumm E^* exists, i.e., the disease always persists in the population.

Now the variational matrix corresponding to E^* is given by,

$$J(E^*) = \begin{bmatrix} -d & 0 & -\alpha & -\alpha & 0 & 0 \\ l & -(l+b) & -l & -l & -l & 0 \\ n_1 & -n_2 & -n_3 & -n_2 & -n_2 & n_4 \\ 0 & 0 & \delta & -e & 0 & 0 \\ 0 & 0 & \rho & \eta & -d_1 & 0 \\ 0 & 0 & \gamma & 0 & 0 & -\sigma \end{bmatrix},$$

$$n_1 = -\frac{\beta I^*(N^* - Y^* - I^* - Q^* - R^*)}{N^{*2}} + \frac{\beta I^*}{N^*} + \lambda V^*, n_2 = \frac{\beta I^*}{N^*} + \lambda V^*, d_1 = \nu + d,$$

$$n_3 = -\frac{\beta(N^* - Y^* - I^* - Q^* - R^*)}{N^*} + \frac{\beta I^*}{N^*} + \lambda V^* + c, n_4 = \lambda(N^* - Y^* - I^* - Q^* - R^*).$$

The roots of variational matrix are determined by the following characteristic equation,

$$f(\lambda) = \left(\lambda^6 + a_1\lambda^5 + a_2\lambda^4 + a_3\lambda^3 + a_4\lambda^2 + a_5\lambda + a_6\right) = 0, \tag{26}$$

$$a_1 = l + b + n_3 + e + d + \sigma + d_1,$$

$$\begin{aligned} a_2 = {} & (l+b)\left\{n_3 + e + d + \sigma + d_1)\right\} + n_3(e + d + \sigma + d_1) + e(d + \sigma + d_1) \\ & + d(\sigma + d_1) + (\sigma d_1 + n_2\delta + n_2 l) + n_1\alpha + n_2\rho - n_4\gamma, \end{aligned}$$

$$\begin{aligned} a_3 = {} & (l+b)\, n_3(e + d + \sigma + d_1) + (l+b)\, e(d + \sigma + d_1) + (l+b)\, d\,(\sigma + d_1) \\ & + (l+b)\,\sigma d_1 + n_3 e(d + \sigma + d_1) + n_3 d\,(\sigma + d_1) + n_3\sigma d_1 + e\sigma d_1 \\ & + ed\,(\sigma + d_1) + d\sigma d_1 + n_2\delta(d_1 + d + \sigma + l + b) + n_2\delta\eta \\ & + n_2 l(e + d_1 + d + \sigma + \rho + \delta - \alpha) + n_1\alpha(l + b + e + \sigma + d_1 - \delta) \\ & + n_2\rho(l + b + d + \sigma + e) - n_4\gamma(d_1 + e + d + l + b), \end{aligned}$$

$$\begin{aligned} a_4 = {} & (l+b)\, n_3 e(d + \sigma + d_1) + (l+b)\, de(\sigma + d_1) + (l+b)\, d\sigma d_1 + n_2 l\delta\eta \\ & + (l+b)\, e\sigma d_1 + (l+b)\, n_3 d(\sigma + d_1) + n_3 d\sigma d_1 + (l+b)\, n_3\sigma d_1 \\ & + n_3 ed(\sigma + d_1) + n_3 e\sigma d_1 + ed\sigma d_1 + n_2\delta(l+b)(d + d_1 + \sigma) \\ & + n_2\delta\eta(l + b + d + \sigma) + n_2 l\left\{e(d + \sigma + d_1) + d_1(\sigma + d) + \sigma d\right\} \\ & + n_2 l\delta(\sigma + d + d_1) + l\, n_2\rho(\sigma + e + d) + n_2\delta(d(d_1 + \sigma) + d_1\sigma) \\ & - n_4\gamma\left\{ed_1 + d(e + d_1) + (d_1 + d + e)(b + l)\right\} - \alpha n_2 l(e + \sigma + d_1 \\ & - \alpha\delta n_1(d_1 + l + b + \sigma) + \alpha\delta n_2 l \\ & + \alpha n_1\left\{e(l + b + \sigma + d_1) + d_1(l + b + \sigma) + \sigma(l + b)\right\} \\ & + n_2\rho\left\{(l+b)(d + e + \sigma) + d(e + \sigma) + e\sigma\right\}, \end{aligned}$$

$$\begin{aligned} a_5 = {} & (l+b)\, n_3 ed\,(\sigma + d_1) + (l+b)\, n_3 d\sigma d_1 + (l+b)\, e\sigma dd_1 + n_3 ed\sigma d_1 \\ & + (l+b)\, e\sigma d_1 n_3 + n_2\delta\left\{d(d_1 + \sigma)(l+b) + (l+b)d_1\sigma + dd_1\sigma\right\} \\ & + n_2\delta\eta\left\{(\sigma + d)(l+b) + d\sigma\right\} + n_2 l\delta\eta(\sigma + d) \\ & - n_4\gamma\left\{ed_1(l+b) + ded_1 + d(e + d_1)(l+b)\right\} \\ & + n_2 l\delta\left\{d_1\sigma + d(d + \sigma)\right\} + n_2 l\left\{ed\sigma + ed_1(\sigma + d) + d_1\sigma d\right\} \\ & - \alpha n_2 l\left\{e(\sigma + d_1) + \sigma d_1\right\} + \rho n_2\left\{(\sigma + e)(l+b)d + (l+b)\sigma e + d\sigma e\right\} \\ & + n_2\rho l\left\{d\sigma + (d + \sigma)e\right\} + \alpha n_1\left\{ed_1(\sigma + l + b) + d_1\sigma(l+b) + e\sigma(l+b)\right\} \\ & + \alpha\delta n_2 l(\sigma + d_1) - \alpha\delta n_1\left\{(l+b)(d_1 + \sigma) + d_1\sigma\right\}, \end{aligned}$$

$$\begin{aligned} a_6 = {} & (l+b)\, n_3 ed\sigma d_1 + n_2\delta d\sigma(l+b)d_1 + n_2\delta\eta\sigma(l+b)\, d + n_2 led\sigma d_1 \\ & + n_2 l\delta\sigma dd_1 + n_2 l\delta\eta\sigma d + \alpha n_1 e(l+b)d_1 + \alpha\delta n_2 l\sigma d_1 + l\sigma n\rho n_2 d \\ & + n_2\rho e\sigma(l+b)d - n_4\gamma ed(l+b)d_1 - \alpha n_2 le\sigma d_1 - \alpha\delta n_1(l+b)\sigma d_1. \end{aligned}$$

Therefore, $a_i > 0$ for i = 1, 2, 3, 4, 5, 6. Thus, by Routh-Hurwitz criteria as stated below, the equilibrium E^* is locally asymptotically stable if the remaining conditions,

$$\begin{vmatrix} a_1 & 1 \\ a_3 & a_2 \end{vmatrix} > 0, \begin{vmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & a_4 & a_3 \end{vmatrix} > 0, \begin{vmatrix} a_1 & 1 & 0 & 0 \\ a_3 & a_2 & a_1 & 1 \\ a_5 & a_4 & a_3 & a_2 \\ 0 & 0 & a_5 & a_4 \end{vmatrix} > 0, \begin{vmatrix} a_1 & 1 & 0 & 0 & 0 \\ a_3 & a_2 & a_1 & 1 & 0 \\ a_5 & a_4 & a_3 & a_2 & a_1 \\ 0 & a_6 & a_5 & a_4 & a_3 \\ 0 & 0 & 0 & a_6 & a_5 \end{vmatrix} > 0,$$

are satisfied.

4.3. Global Stability of the Endemic Equilibrium

To show the global stability behavior of E^*, we need the bounds of dependent variables involved. For this, we find the region of attraction stated in the form of following lemma,

Lemma 4.1.

The region,

$$\Omega = \left\{ \begin{array}{c} (N, Y, I, Q, V, R)\,;\ 0 < N(t) \leq \overline{N};\ 0 \leq Y(t) \leq \overline{Y};\ 0 \leq (t) \leq \overline{I}; \\ 0 \leq Q(t) \leq \overline{Q};\ 0 \leq V(t) \leq \overline{V};\ 0 \leq R(t) \leq \overline{R} \end{array} \right\}, \tag{27}$$

is a region of attraction for the system (7)-(12),

where $\overline{N} = \frac{\Lambda}{d}, \overline{Y} = \frac{l\Lambda}{d(l+b)}, \overline{I} = \frac{\Phi\Lambda}{d}\frac{1}{(\Phi+c)}, \overline{Q} = \frac{\delta\bar{I}}{e}, \overline{R} = \frac{\eta\bar{Q}+\rho\bar{I}}{\nu+d}, \overline{V} = \frac{\gamma\Lambda}{\sigma d}$, and $\Phi = \left(\beta + \frac{\lambda_1\gamma\Lambda}{\sigma d}\right)$.

Proof:

From Equation (7),

$$\frac{dN(t)}{dt} = \Lambda - dN - \alpha I(t) - \alpha Q(t),$$

$$\frac{dN(t)}{dt} \leq \Lambda - dN.$$

Thus, $\lim_{t\to\infty} SupN(t) \leq \overline{N}$, where $\bar{N} = \frac{\Lambda}{d}$.

From Equation (8),

$$\frac{dY(t)}{dt} = l\left\{N(t) - Y(t) - I(t) - Q(t) - R(t)\right\} - bY(t),$$

$$\frac{dY(t)}{dt} \leq l\left\{N(t) - Y(t)\right\} - bY(t),$$

$$\frac{dY(t)}{dt} \leq lN(t) - (b+l)Y(t),$$

$$\frac{dY(t)}{dt} \leq l\frac{\Lambda}{d} - (b+l)Y(t).$$

Thus, $\lim_{t\to\infty} SupY(t) \leq \overline{Y}$, where $\bar{Y} = \frac{l\Lambda}{d(l+b)}$.

From Equation (12),

$$\frac{dV(t)}{dt} = \gamma I(t) - \sigma V(t),$$

$$\frac{dV(t)}{dt} \leq \gamma\frac{\Lambda}{d} - \sigma V(t),$$

since $I(t) \leq \mathrm{N}(t) \leq \overline{N}$.

Thus, $\lim_{t\to\infty} SupV(t) \leq \overline{\mathrm{V}}$, where $\bar{V} = \frac{\gamma\Lambda}{d\sigma}$.

From Equation (9),

$$\frac{dI(t)}{dt} = \left[\frac{\beta I(t)}{N(t)} + \lambda_1 V(t)\right]\{N(t) - Y(t) - I(t) - Q(t) - R(t)\} - cI(t),$$

$$\frac{dI(t)}{dt} \leq \left[\frac{\beta I(t)}{N(t)} + \lambda_1 V(t)\right]\{N(t) - I(t)\} - cI(t),$$

$$\frac{dI(t)}{dt} \leq \left[\beta + \lambda_1\frac{\gamma\Lambda}{d\sigma}\right]N(t) - (\phi + c)I(t),$$

$$\frac{dI(t)}{dt} \leq \phi\frac{\Lambda}{d} - (\phi + c)I(t).$$

Thus, $\lim_{t\to\infty} SupI(t) \leq \overline{I}$, where $\bar{I} = \frac{\phi\Lambda}{d(\phi+c)}$, and $\Phi = \left[\beta + \lambda_1\frac{\gamma\Lambda}{d\sigma}\right]$.

From Equation (10),

$$\frac{dQ(t)}{dt} = \delta I(t) - eQ(t),$$

$$\frac{dQ(t)}{dt} \leq \delta\bar{I} - eQ(t).$$

Thus, $\lim_{t\to\infty} SupQ(t) \leq \overline{Q}$, where $\bar{Q} = \frac{\delta\bar{I}}{e}$.

From Equation (11),

$$\frac{dR(t)}{dt} = \eta Q(t) + \rho I(t) - (\nu + d)\,R(t),$$

$$\frac{dR(t)}{dt} \leq \eta\bar{Q} + \rho\bar{I} - (\nu + d)\,R(t).$$

Thus, $\lim_{t\to\infty} SupR(t) \leq \overline{R}$, where $\bar{R} = \frac{\eta\bar{Q}+\rho\bar{I}}{(\nu+d)}$. ■

Theorem 4.1.

If the endemic equilibrium E^* exists, it is globally asymptotically stable provided the following sufficient conditions are satisfied in Ω,

$$\frac{9\alpha^2}{4de} < \frac{(\beta + \lambda_1 V^*)\, m_1}{\delta}, \tag{28}$$

$$3\eta^2\delta < \rho de, \tag{29}$$

$$[\lambda m_1(N^* - Y^*) + \gamma]^2 \leq \frac{4\sigma m_1}{5}\left[\lambda V^* + c - \beta\frac{N^* - Y^* - I^* - Q^* - R^*}{N^*}\right], \tag{30}$$

$$[m_1(\beta + \lambda_1 V^*) + k_1 l]^2 \leq \frac{(l+b)m_1 k_1}{2}\left[\lambda V^* + c - \beta\frac{N^* - Y^* - I^* - Q^* - R^*}{N^*}\right], \tag{31}$$

where

$$m_1 = \frac{\alpha}{\beta + \lambda_1 V^* - \beta\left[\frac{(N^* - Y^* - I^* - Q^* - R^*)I^*}{\bar{N}\, N^*}\right]},$$

and

$$k_1 \leq \min\left\{\frac{(l+b)dm_1}{3l^2}, \frac{(l+b)em_1(\beta + \lambda_1 V^*)}{3\delta\, l^2}, \frac{d(l+b)m_1(\beta + \lambda_1 V^*)}{4\rho\, l^2}\right\}.$$

Proof:

Consider the following positive definite function about E^* (Tripathi et al. (2007)),

$$\begin{aligned} P = {} & \tfrac{1}{2}(N - N^*)^2 + \tfrac{1}{2}k_1(Y - Y^*)^2 + \tfrac{1}{2}k_2(I - I^*)^2 + \tfrac{1}{2}k_3(Q - Q^*)^2 \\ & + \tfrac{1}{2}k_4(R - R^*)^2 + \tfrac{1}{2}k_5(V - V^*)^2. \end{aligned} \tag{32}$$

where k_i, i = 1,...,5 are constants to be chosen appropriately.

Differentiating P with respect to t, we get

$$\begin{aligned} \frac{dP}{dt} = {} & (N - N^*)\frac{dN}{dt} + k_1(Y - Y^*)\frac{dY}{dt} \\ & + k_2(I - I^*)\frac{dY}{dt} + k_3(Q - Q^*)\frac{dQ}{dt} \\ & + k_4(R - R^*)\frac{dR}{dt} + k_5(V - V^*)\frac{dV}{dt}. \end{aligned}$$

Using Equations (7) - (12) and simplifying, we get

$$
\begin{aligned}
\frac{dP}{dt} =& -d(N-N^*)^2 - k_1(l+b)(Y-Y^*)^2 - k_3e(Q-Q^*)^2 \\
&- k_4(\nu+d)(R-R^*)^2 - k_5\sigma(V-V^*)^2 \\
&- k_2[\frac{\beta I}{N} - \frac{\beta(N^*-Y^*-I^*-Q^*-R^*)}{N} + \lambda_1 V^* + c](Y-Y^*)^2 \\
&+ k_1 l(N-N^*)(Y-Y^*) - \alpha(N-N^*)(Q-Q^*) \\
&+ [-\alpha + \frac{k_2\beta I}{N} - \frac{k_2\beta(N^*-Y^*-I^*-Q^*-R^*)}{N} + k_2\lambda_1 V^*](N-N^*)(I-I^*) \\
&+ (-\frac{k_2\beta I}{N} - k_2\lambda_1 V^* + k_3\delta)(R-R^*)(I-I^*) \\
&- k_1 l(Q-Q^*)(Y-Y^*) - (\frac{k_2\beta I}{N} + k_1 l + k_2\lambda_1 V^*)(Y-Y^*)(I-I^*) \\
&- (\frac{k_2\beta I}{N} + k_1 l + k_2\lambda_1 V^*)(Y-Y^*)(I-I^*) \\
&- (\frac{k_2\beta I}{N} + k_2\lambda_1 V^* - k_3\delta)(Q-Q^*)(I-I^*) \\
&+ (\lambda_1 k_2(N-Y-I-Q-R) + k_5\gamma)(V-V^*)(I-I^*) \\
&- k_1 l(R-R^*)(Y-Y^*) + k_4\eta(Q-Q^*)(R-R^*),
\end{aligned} \tag{33}
$$

$$
\begin{aligned}
\tfrac{dP}{dt} =& -\tfrac{1}{2}a_{11}\left(N-N^*\right)^2 + a_{12}\left(N-N^*\right)\left(Y-Y^*\right) - \tfrac{1}{2}a_{22}\left(Y-Y^*\right)^2 \\
&-\tfrac{1}{2}a_{11}\left(N-N^*\right)^2 + a_{13}\left(N-N^*\right)\left(I-I^*\right) - \tfrac{1}{2}a_{33}\left(I-I^*\right)^2 \\
&-\tfrac{1}{2}a_{11}\left(N-N^*\right)^2 + a_{14}\left(N-N^*\right)\left(Q-Q^*\right) - \tfrac{1}{2}a_{44}\left(Q-Q^*\right)^2 \\
&-\tfrac{1}{2}a_{22}\left(Y-Y^*\right)^2 + a_{23}\left(Y-Y^*\right)\left(I-I^*\right) - \tfrac{1}{2}a_{33}\left(I-I^*\right)^2 \\
&-\tfrac{1}{2}a_{22}\left(Y-Y^*\right)^2 + a_{24}\left(Y-Y^*\right)\left(Q-Q^*\right) - \tfrac{1}{2}a_{44}\left(Q-Q^*\right)^2 \\
&-\tfrac{1}{2}a_{22}\left(Y-Y^*\right)^2 + a_{25}\left(Y-Y^*\right)\left(R-R^*\right) - \tfrac{1}{2}a_{55}\left(R-R^*\right)^2 \\
&-\tfrac{1}{2}a_{33}\left(I-I^*\right)^2 + a_{34}\left(I-I^*\right)\left(Q-Q^*\right) - \tfrac{1}{2}a_{44}\left(Q-Q^*\right)^2 \\
&-\tfrac{1}{2}a_{33}\left(I-I^*\right)^2 + a_{35}\left(I-I^*\right)\left(R-R^*\right) - \tfrac{1}{2}a_{55}\left(R-R^*\right)^2 \\
&-\tfrac{1}{2}a_{33}\left(I-I^*\right)^2 + a_{36}\left(I-I^*\right)\left(V-V^*\right) - \tfrac{1}{2}a_{66}\left(V-V^*\right)^2 \\
&-\tfrac{1}{2}a_{44}\left(Q-Q^*\right)^2 + a_{45}\left(R-R^*\right)\left(Q-Q^*\right) - \tfrac{1}{2}a_{55}\left(R-R^*\right)^2,
\end{aligned}
$$

$$
\begin{aligned}
&a_{11} = \tfrac{2d}{3}, \quad a_{22} = \tfrac{1}{2}k_1\left(l+b\right), \; a_{44} = \tfrac{1}{2}k_3e, \quad a_{55} = \tfrac{2}{3}k_4\left(\nu+d\right), \quad a_{66} = \tfrac{1}{2}k_5\sigma, \\
&a_{33} = \tfrac{2}{5}k_2\left[\tfrac{\beta I}{N} - \tfrac{\beta(N^*-Y^*-I^*-Q^*-R^*)}{N} + \lambda_1 V^* + c\right], \; , \\
&a_{12} = k_1 l, \quad a_{13} = \left(-\alpha + \tfrac{k_2\beta I}{N} - \tfrac{k_2\beta(N^*-Y^*-I^*-Q^*-R^*)}{N} + k_2\lambda_1 V^*\right), \\
&a_{14} = -\alpha, \; a_{23} = -\left(\tfrac{k_2\beta I}{N} + k_1 l + k_2\lambda_1 V^*\right) \; , \; a_{24} = -k_1 l, \quad a_{25} = -k_1 l, \\
&a_{34} = -\left(\tfrac{k_2\beta I}{N} + k_2\lambda_1 V^* - k_3\delta\right), \quad a_{35} = k_4\rho - \left(\tfrac{k_2\beta I}{N} + k_2\lambda_1 V^*\right) \; , \\
&a_{36} = \left(\lambda_1 k_2(N-Y-I-Q-R) + k_5\gamma\right), \; a_{45} = k_4\eta
\end{aligned}
$$

Choosing, k_5 = 1, and $k_1 \leq \min\left\{\frac{(l+b)dm_1}{3l^2}, \frac{(l+b)em_1(\beta+\lambda_1V^*)}{3\delta\, l^2}, \frac{(\nu+d)(l+b)m_1(\beta+\lambda_1V^*)}{4\rho\, l^2}\right\}$,

$$
k_2 = m_1\frac{\alpha}{\beta + \lambda_1 V^* - \beta\left[\frac{(N^*-Y^*-I^*-Q^*-R^*)I^*}{N\,N^*}\right]}, k_3 = \frac{\left(\beta+\lambda_1V^*\right)m_1}{\delta},
$$

$$k_4 = \frac{(\beta + \lambda_1 V^*)\, m_1}{\rho}.$$

Thus, $\frac{dP}{dt}$ is negative definite under the conditions stated in theorem showing that P is a Liapunov function, and hence, E^* is globally asymptotically stable inside the region of attraction Ω. ■

4.4. Transcritical Bifurcation

From the above discussion, we observe that the system (7) - (12) may undergo a transcritical bifurcation at E_0 when $R_0 = 1$. In this subsection, we establish the conditions using center manifold theory. Here, we omit the variable R as R does not play any role in the remaining five equations in system (7)-(12). We choose λ_1 as a bifurcation parameter. By solving $R_0 = 1$, we obtain

$$\lambda_1 = \lambda_1^* = \frac{\sigma d}{\Lambda \gamma}(\delta + \alpha + d) - \beta. \tag{34}$$

We can see that transcritical bifurcation exists only if $(\delta + \alpha + d) > \beta$.

It can easily be obtained that the Jacobian $J(E_0, \lambda_1^*)$ evaluated at E_0 and $\lambda_1 = \lambda_1^*$ has a simple zero eigenvalue and other eigenvalues have negative sign. Hence, E_0 is a non-hyperbolic equilibrium, when $\lambda_1 = \lambda_1^*$. Now, we calculate a right eigenvector $K = (w_1, w_2, w_3, w_4, w_6)$ and a left eigenvector $M = (v_1, v_2, v_3, v_4, v_6)$ associated to the zero eigenvalues. Here

$$w_1 = -\frac{\alpha}{d}\left(\frac{\sigma}{\gamma} + 1\right), w_2 = -l\left(\frac{\alpha}{d} + 1\right)\left(\frac{\sigma}{\gamma} + 1\right), w_3 = \frac{\delta\sigma^2}{e\gamma^2}, w_4 = \frac{\delta\sigma}{e\gamma}, w_6 = 1, \tag{35}$$

$$\nu_1 = 0, \nu_2 = 0 = 0, \nu_3 = \frac{\gamma}{(\delta + \alpha + d) - \beta}, \nu_4 = 0, \nu_6 = 1. \tag{36}$$

We need to calculate the bifurcation constants a and b. By choosing f_3 and calculating partial derivatives of f_3 (evaluated at E_0, $x_1 = N$, $x_2 = Y$, $x_3 = I$, $x_4 = Q$, $x_6 = V$), we obtain,

$a = 2\nu_3\,(w_1 w_3 \frac{\partial^2 f_3}{\partial N \partial I} + w_1 w_6\, \frac{\partial^2 f_3}{\partial N \partial V} + w_2 w_3\, \frac{\partial^2 f_3}{\partial L \partial I} + w_2 w_6\, \frac{\partial^2 f_3}{\partial L \partial V} + w_3 w_6\, \frac{\partial^2 f_3}{\partial I \partial V} + w_4 w_6\, \frac{\partial^2 f_3}{\partial Q \partial V} + w_3 w_4\, \frac{\partial^2 f_3}{\partial I \partial Q})$

$= 2\nu_3\,(w_1 w_3 \frac{\beta L_0}{N_0^2} + w_1 w_6\, \lambda_1 - w_2 w_3\, \frac{\beta}{N_0} - w_2 w_6\, \lambda_1 - w_3 w_6\, \lambda_1 - w_4 w_6\, \lambda_1 - w_3 w_4 \frac{\beta}{N_0})$,

$$a = -2\nu_3 \left\{ (w_3 + w_4)[\frac{\lambda(\mu + d)}{b}(1 + \frac{\alpha}{d}) - \frac{\beta l}{N_0}] + w_3 w_4 \frac{\beta}{N_0^2} \right\} < 0, \tag{37}$$

where $b = 2\nu_3\,(w_5 \frac{\partial^2 f_3}{\partial V \partial \lambda_1})$,

$$b = 2\nu_3 w_5 (N_0 - L_0) > 0. \tag{38}$$

Here, w_1, w_2 are negative and w_3, w_4 are positive (from Equation 35) so that a is negative and b is positive. Therefore, E_0 changes its stability from stable to unstable at $\lambda_1 = \lambda_1^*$ and there exists a positive equilibrium as $\lambda_1 = \lambda_1^*$ crosses its critical value. Hence, the system (7) - (12) undergoes a transcritical bifurcation at $\lambda_1 = \lambda_1^*$. Thus, the transmission rate λ_1 plays an important role in the disease spread. If λ_1 is less than the critical value then it is easy to control the disease but if the transmission rate λ_1 is above the critical value then the disease may reach endemic state.

5. Numerical Simulation and Discussion

To see the dynamical behavior of the model, the system (7) - (12) is integrated numerically by fourth order Runge - Kutta method using the following set of parameters values:

$\Lambda = 5$, $d = 0.00003$, $\alpha = 0.00011$, $\beta = 0.006$, $l = 0.1$, $\mu = 0.03$, $\lambda_1 = 0.002$,

$\eta = 0.01$, $\delta = 0.01$, $\gamma = 0.9$, $\sigma = 0.01$, $\rho = 0.8$

with initial values $N(0) = 1000$, $Y(0) = 0$, $I(0) = 1$, $Q(0) = 0$, $R(0) = 0$ and $V(0) = 1$.

The results of numerical simulation are displayed graphically in Figures (2-8). In Figure 2, the variation of infective population $I(t)$ with time t is shown for different values of λ_1, the rate of transmission of susceptibles to infective class through direct contact with viral density present in the environmental reservoir. It is seen that infective population increases with increase in the value of λ_1. This implies that if the individuals from susceptible population self protect themselves and do not come in direct contact with virus deposited on surfaces/objects or airborne droplets accumulated in the environmental reservoir, the infective population can be decreased.

In Figures (3 - 4), the variation of infective population $I(t)$ and recovered population $R(t)$ respectively is shown with time t for different values of ρ, the recovery rate of infectives without being quarantined. It is observed that the infective population declines with increase in the recovery rate of infectives who are not quarantined but take self medication (Figure 3). However, the recovered population increases with recovery rate of infectives who do not quarantine themselves but take self medication (Figure 4). The variation of quarantined population $Q(t)$ is shown in Figure 5 with time t for different values of δ, the rate of transfer of infectives into quarantined class. This indicates that if rate of transfer of infectives into quarantined class increases, the population in quarantined class who are either isolated at home or hospital increases. Since this increased population of quarantined individuals is isolated, it does not contribute to viral transmission further, and hence, the spread of the disease can be lowered.

In Figures (6 - 7), the variation of quarantined population $Q(t)$ and recovered population $R(t)$ respectively is shown with time t for different values of η, the rate of recovery of quarantined individuals. It is noted that with increase in the recovery rate of quarantined individuals, their population decreases, (Figure 6) which ultimately increases the population of recovered individuals, (Figure 7) since the population of recovered individuals is directly proportional to that of quarantined individuals. In Figure 8, we have shown the variation of infective population $I(t)$ with time t for

different values of l, the rate of transfer of susceptibles to self-protected class. It is found that with increase in the value of l, the infective population decreases. This is due to the fact that if higher number of susceptibles opt to follow COVID-19 protocol and self protect themselves, the infective population declines.

From the above discussion, it follows that if more and more susceptible individuals either self protect themselves by following the COVID-19 guidelines or remain quarantined at home or hospital, the spread of the disease can be controlled.

6. Conclusion

In this paper, a nonlinear mathematical model has been proposed and analyzed to study the effect of self-protection and quarantine strategy on the spread of corona virus in a population with variable size structure.From the analysis of the proposed model it is found that if the individuals from susceptible population self protect themselves and do not come in direct contact with virus deposited on surfaces/objects or airborne droplets accumulated in the environmental reservoir, the infective population can be decreased. This decrease is further affected if the individuals from infective population recover from self medication without being quarantined. Moreover, if higher number of individuals from infective class are quarantined at home or hospital, the spread of the disease can be slowed down.

Finally from the analysis, it may be concluded that if more and more susceptible individuals follow COVID-19 protocol in the form of non-pharmaceutical interventions such as applying face cover/mask in public places, adopt social distancing, avoid public gatherings, etc., the spread of corona infection can be slowed down in the community. It is also observed that the viral density in the environmental reservoir decreases due to decreased number of infectives and through frequent sanitization of objects/surfaces which helps in keeping the epidemic under control.The implementation of these strategies can be more effective if there is a high level of individual awareness through media campaigns. If people are educated and made aware of the importance of such interventions, they would prefer to practice these preventive measures as a result of behavioral change due to media awareness. To understand the transmission dynamics of the disease and effect of theses intervention strategies, extensive research using mathematical models can be of vital importance. The present study can further be extended by incorporating the role of media awareness campaigns, sanitation efforts and their economic impact, effect of vaccination, age structure, etc.

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Appendix

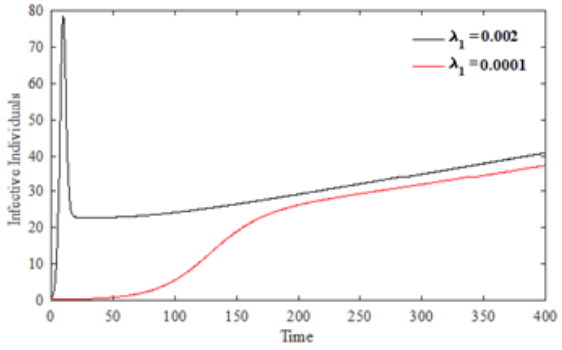

Figure 2. Variation of infective population for different values of λ_1

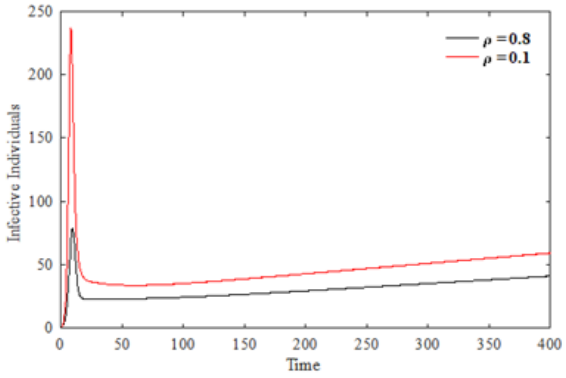

Figure 3. Variation of infective population for different values of ρ

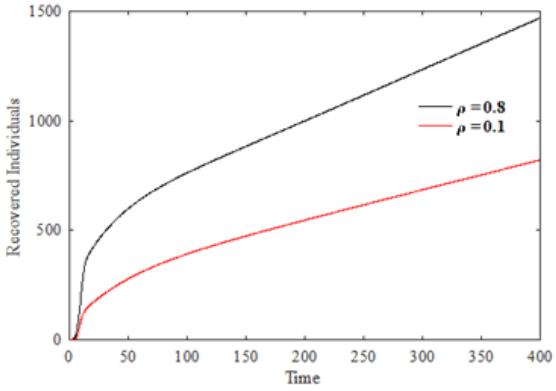

Figure 4. Variation of recovered population for different values of ρ

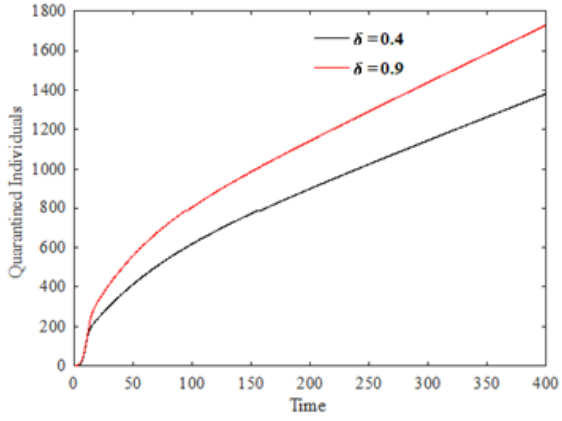

Figure 5. Variation of quarantined population for different values of δ

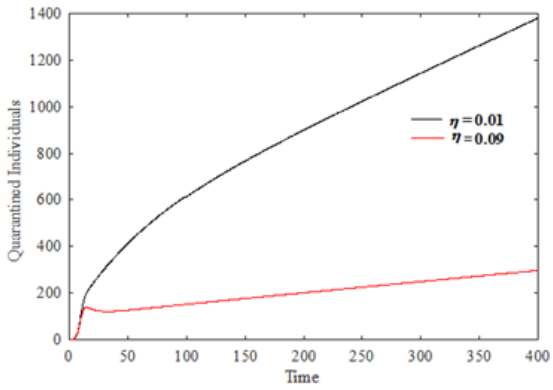

Figure 6. Variation of quarantined individuals for different values of η

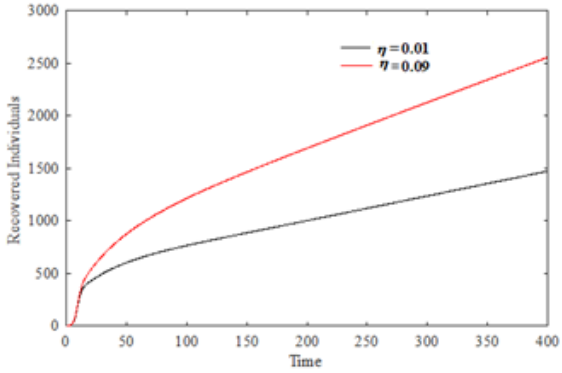

Figure 7. Variation of recovered population for different values of η

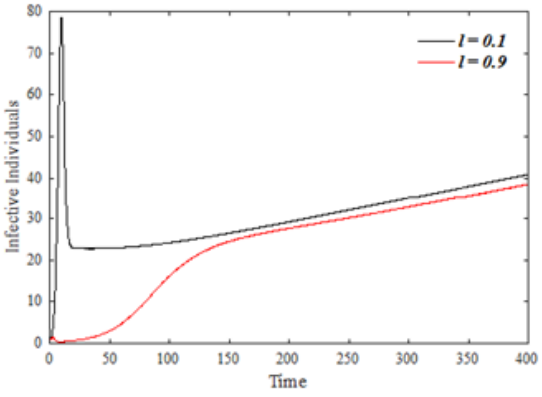

Figure 8. Variation of infective population for different values of l