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An SIQVR Mathematical Model on COVID-19 with Virus Population in the Environment: A Case Study of India

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Abstract:- In the proposed SIQVR model, we examine the impact of vaccination and lockdown on COVID-19. Beginning with the outbreak in Wuhan in December 2019, India's nationwide lockdown on March 24, 2020, and vaccination campaign starting January 16, 2021, are analyzed. Using a five-dimensional mathematical model with nonlinear differential equations, our study reveals that complete eradication of the virus is possible only with total lockdown and universal vaccination. Without these measures, the virus will persist. Effective management includes contact tracing, quarantine, accelerating vaccination and targeted lockdowns.

Keywords*:* Mathematical model, COVID-19, Transmission, Equilibrium analysis, Numerical Simulation.

1. Introduction

Throughout the course of history, intermittent outbreaks of infectious diseases have had profound and lasting impacts on societies. These incidents have significantly shaped various aspects of human civilization, such as economics, politics, and social dynamics, with their effects often enduring for extended periods. These epidemic events have played a crucial role in influencing the foundational principles of modern medicine, leading the scientific community to develop essential concepts in epidemiology, disease prevention, immunization, and antimicrobial treatments [1,2]. Over the course of history, the globe has encountered various epidemics and pandemics, and one of the most significant was the Black Death outbreak, commonly referred to as the bubonic plague, in the year 1347. This specific pandemic is noteworthy for being the deadliest recorded in history, causing widespread devastation primarily in Europe [3,4,5]. The global outbreak of the Spanish flu from 1918 to 1920 was the first-ever worldwide pandemic and the first to occur in the era of modern medicine. Throughout this time, fields like infectious diseases and epidemiology extensively studied the nature of the diseases and the development of the pandemic [6,7,8]. The early 21st century witnessed a notable public focus on the onset of Severe Acute Respiratory Syndrome (SARS), triggered by the SARS Coronavirus (SARS-CoV-2) and emanating from China. The outbreak affected fewer than 10,000 individuals in China and Hong Kong initially, with additional cases reported in various countries. Due to the dedicated efforts of international public health systems, the outbreak was effectively controlled by mid-2003. In 2009, there was a revival of the 1918 "Spanish flu" pandemic, recognized as the 2009 H1N1 pandemic [9,10,11]. Commonly known as the "swine flu," this episode was thought to arise from the combination of avian, swine, and human influenza viruses. Fortunately, its impact was milder compared to the original Spanish flu. In December 2019, a pneumonia outbreak surfaced in Wuhan, a significant city in the central Chinese province of Hubei. Rapidly, it disseminated throughout the country. The Chinese Center for Disease Control and Prevention (CCDC) pinpointed a new beta-coronavirus, initially designated as 2019-nCoV, which is now officially acknowledged as Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) [12,13,14,15]. The third outbreak of a zoonotic coronavirus in the first two decades of the 21st century was triggered by SARS-CoV-2, the virus that leads to COVID-19. This virus facilitated the transmission of the disease from person to person, causing substantial worldwide health worries. The World Health Organization (WHO) expressed deep concern about the seriousness and swift global spread of this highly infectious illness, officially categorizing it as a pandemic on March 11, 2020 [16,17,18]. As per the WHO’s 57th report dated March 17,2020, there were a total 179112 confirmed cases worldwide, resulting in 7426 deaths, reflecting a mortality rate of approximately 4Director-General of the World Health Organization, commented that the world had possibly not witnessed such a devastating illness and emerging threat since the time before World War II. As of March 21, 2021, Uttar Pradesh had reported 607,050 confirmed cases of COVID-19, with 8,758 deaths, 2,774 active cases, and 595,518 recovered cases. By September 18, 2021, India had recorded a 2 total of 33,447,010 confirmed COVID-19 cases, including 447,166 (1As of March 21, 2022, Uttar Pradesh had documented 2,070,285 confirmed COVID-19 cases, with 23,492 deaths, 778 active cases, and 2,046,015 recovered cases [19,20,21,22]. By March 22, 2022, India had reported a total of 429,843,48 confirmed COVID-19 cases, including 516,574 (1Researchers globally have been exerting substantial efforts to track and prevent the spread of the disease [23,24,25,26]. They have been conducting experiments with innovative diagnostics and have successfully developed vaccines to curb the impact of the virus [27,28,29]. The potential of mathematical models lies in their ability to monitor and forecast the trajectory of an epidemic in various scenarios. Epidemiological mathematical models play a crucial role in predicting the peak in advance and evaluating the impact of different factors on controlling its spread. This study aims to introduce a flexible model that describes the flow of an epidemic, applicable to the specific conditions of a local city.

2. Mathematical Model

In this study we propose to analyze the flow of COVID-19 under five different conditions. In the following model S stands for susceptible, I stands for infected, Q stands for quarantined, V stands for virus population and R stands for recovered population. Susceptible population enter into the system with the constant rate A. β is the transmission coefficient of infection from infected human, ξ represent the transmission coefficient of infection directly from the virus present in the environment to the susceptible population. δ_1 and δ_2 are the rate at which population again becomes susceptible to infection after recovery. μ is the natural mortality rate and α_1, α_2 are disease related death rate of infected and quarantined population. σ is the rate of isolating infected population after tracing at the k. θ is the rate of tracing infected population and are quarantined on being suspected having infection. We assume that virus population is rising in the system at the constant rate r_0 proportional to the infected population present in the system and μ_0 is the rate of elimination of virus from the environment and ε is the recovery coefficient of quarantined population.

$$\frac{dS}{dt} = A - \beta SI - \xi VS + \delta_1 I + \delta_2 Q - \mu S \qquad .....(2.1)$$

$$\frac{dI}{dt} = (1-k)\beta SI + \xi VS + (1-\theta)mI - (\mu + \delta_1 + \alpha_1 + \sigma)I \qquad .....(2.2)$$

$$\frac{dQ}{dt} = k\beta SI + \theta mI + \sigma I - (\mu + \delta_2 + \alpha_2)Q \qquad .....(2.3)$$

$$\frac{dV}{dt} = r_0 I - \mu_0 V \qquad .....(2.4)$$

$$\frac{dR}{dt} = \varepsilon Q - \mu R \qquad .....(2.5)$$

With the initial conditions $S(0) > 0, I(0) \geq 0, Q(0) \geq 0, V(0) \geq 0$ and $R(0) \geq 0$.

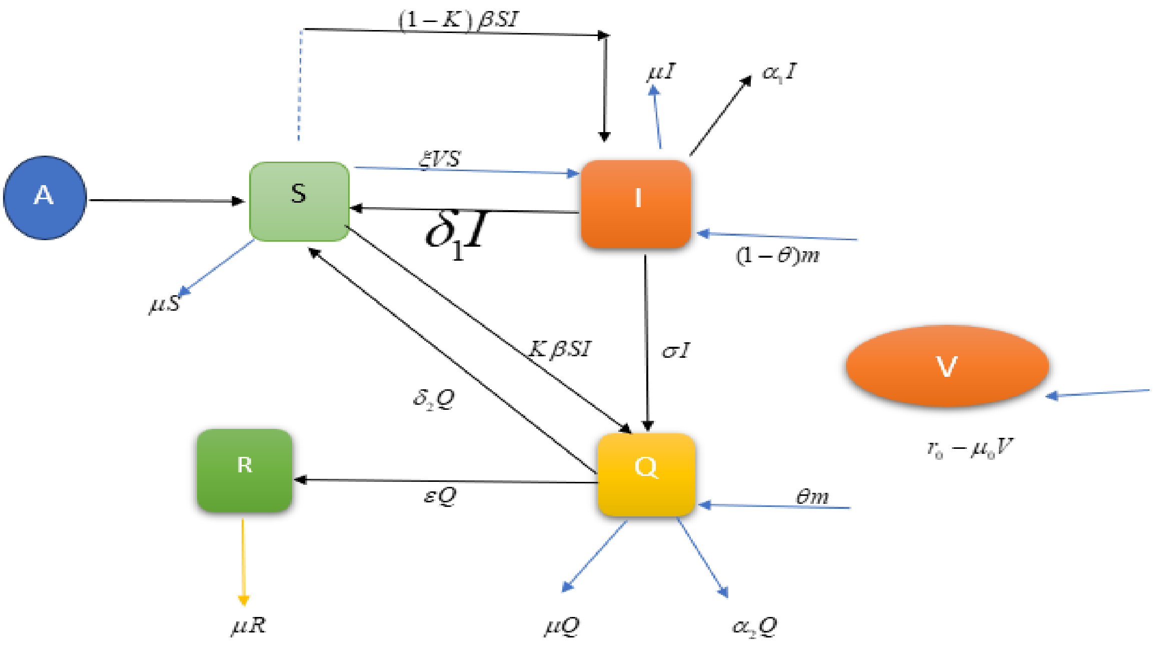

Figure 1: Flow diagram for a model system (2.1) to (2.5).

If we substitute $L_1=\mu+\delta_1+\alpha_1+\sigma$ and $L_2=\mu+\delta_2+\alpha_2$ then the equation (2.1) to (2.5) becomes:

$$\frac{dS}{dt}=A-\beta SI-\xi VS+\delta_1 I+\delta_2 Q-\mu S \qquad .....(2.6)$$

$$\frac{dI}{dt}=(1-k)\beta SI+\xi VS+(1-\theta)mI-L_1 I \qquad .....(2.7)$$

$$\frac{dQ}{dt}=k\beta SI+\theta mI+\sigma I-L_2 Q \qquad .....(2.8)$$

$$\frac{dV}{dt}=r_0-\mu_0 V \qquad .....(2.9)$$

$$\frac{dR}{dt}=\varepsilon Q-\mu R \qquad .....(2.10)$$

Table 1: Table of Description

Variable and Parameter	Description Susceptible
A	Recruitment Rate
β	Transmission rate of infection
ξ	Transmission coefficient of infection directly from the virus
μ	Natural mortality rate

m	number of migrants
θ	Rate of infected migrants
k	Rate of contact tracing
δ_1	Rate of recovery of infective population
δ_2	Rate of recovery of quarantined population
σ	Transmission rate from infective to quarantined population
α_1	Disease related death rate of infective population
α_2	Disease related death rate of quarantined population

3. Boundedness of the System:

The lemma provided specifies the region within which the system (2.1) to (2.5) is attracted.

Lemma 3.1 If the conditions outlined in section 2 are met, then the solutions to the system (2.1) to (2.5) will be confined within the specified set.

$$\Omega=\left\{(S,I,Q,V,R):S+I+Q+V+R\le\frac{A}{\mu},V\le\frac{r_0A}{\mu_0\mu}\right\}$$

Proof: Adding equation $(2.1),(2.2),(2.3),(2.5)$ and after doing some algebraic calculation, we have

$$\frac{dS}{dt}+\frac{dI}{dt}+\frac{dQ}{dt}+\frac{dR}{dt}=A-\beta SI-\xi VS+\delta_1 I+\delta_2 Q-\mu S+(1-k)\beta SI+\xi VS+(1-\theta)mI-(\mu+\delta_1+\alpha_1+\sigma)I$$

$$+k\beta SI+\theta mI+\sigma I-(\mu+\delta_2+\alpha_2)Q+\varepsilon Q-\mu R$$

$$\frac{dS}{dt}+\frac{dI}{dt}+\frac{dQ}{dt}+\frac{dR}{dt}=A-\mu S+mI-\mu I-\alpha_1 I-\mu Q-\alpha_2 Q+\varepsilon Q-\mu R$$

$$\frac{dS}{dt}+\frac{dI}{dt}+\frac{dQ}{dt}+\frac{dR}{dt}=A-(\alpha_1-m)I-(\alpha_2-\varepsilon)Q-\mu(S+I+Q+R)$$

Which implies that, $\lim\limits_{n\to\infty} Sup\left(S(t)+I(t)+Q(t)+R(t)\right)\le\frac{A}{\mu}$

Also, we have $\quad \frac{dV}{dt}\le\frac{r_0A}{\mu}-\mu_0 V$

Thus $\quad \lim\limits_{n\to\infty} Sup V(t)\le\frac{r_0A}{\mu_0\mu}$

Hence the prove lemma.

4. Equilibrium Analysis:

System (2.1) to (2.5) possesses two non-negative equilibrium points: the disease free equilibrium point $E^0\left(\frac{A}{\mu},0,0,0,0,0\right)$ and the endemic equilibrium point $E^n\left(S^n,I^n,Q^n,V^n,R^n\right)$. While the existence of the disease-free equilibrium point is self-evident, we will now explore the existence of the endemic equilibrium point as outline below:

We understand that if the equilibrium point of the model represented by equations (2.1) to (2.5) is denoted as $E^n\left(S^n,I^n,Q^n,V^n,R^n\right)$ it must be satisfy

$$A-\beta S^nI^n-\xi V^nS^n+\delta_1I^n+\delta_2Q^n-\mu S^n=0 \qquad .....(3.1)$$

$$(1-k)\beta S^nI^n+\xi V^nS^n+(1-\theta)mI^n-(\mu+\delta_1+\alpha_1+\sigma)I^n=0 \qquad .....(3.2)$$

$$k\beta S^nI^n+\theta mI^n+\sigma I^n-(\mu+\delta_2+\alpha_2)Q^n=0 \qquad .....(3.3)$$

$$r_0I^n-\mu_0V^n=0 \qquad .....(3.4)$$

$$\varepsilon Q^n-\mu R^n=0 \qquad .....(3.5)$$

Where $L_1=\mu+\delta_1+\alpha_1+\sigma$ and $L_2=\mu+\delta_2+\alpha_2$.

Now from equation (3.4), we obtain

$$V^n=\frac{r_0}{\mu_0}I^n$$

From equation (3.3) we have

$$Q^n=\frac{1}{L_2}\left[k\beta S^n+(\theta m+\sigma)\right]I^n$$

From equation (3.2) gives,

$$(1-k)\beta S^nI^n+\xi\frac{r_0}{\mu_0}I^nS^n+(1-\theta)mI^n-L_1I^n=0$$

$$S^n\left[(1-k)\beta+\xi\frac{r_0}{\mu_0}\right]I^n=\left[L_1-(1-\theta)m\right]I^n$$

This implies that

$$S^n=\frac{L_1-(1-\theta)m}{(1-k)\beta+\xi\frac{r_0}{\mu_0}}=\frac{A}{\mu}\times\frac{1}{R_0}$$

Where $$R_0 = \frac{A}{\mu}\left[\frac{(1-k)\beta+\xi\frac{r_0}{\mu_0}}{L_1-(1-\theta)m}\right]$$

From equation (3.1) we have

$$I^n = \frac{A-\mu S^n}{\beta S^n\left(1-\frac{\delta_2}{L_2}k\right)+\xi\frac{r_0}{\mu_0}S^n-\delta_1-\frac{\delta_2}{L_2}(\theta m+\sigma)}$$

$$= \frac{A\left(1-\frac{1}{R_0}\right)}{\Delta} = \frac{A}{R_0}\left(\frac{R_0-1}{\Delta}\right)$$

Where $$\Delta = \frac{\beta A}{\mu R_0}\frac{\left(\mu+\alpha_2+\delta_2(1-k)\right)}{L_2}+\xi\frac{r_0}{\mu_0}\frac{A}{\mu R_0}-\delta_1-\frac{\delta_2}{L_2}(\theta m+\sigma)$$

We observe that I^n exist if $R_0 > 1$ and $\Delta > 0$.

$$\Delta > 0 \text{ if } R_0 < \frac{A\left[\frac{\beta(\mu+\alpha_2+\delta_2(1-k)}{\mu+\delta_2+\alpha_2}+\xi\frac{r_0}{\mu_0}\right]}{\delta_1+\frac{\delta_2}{L_2}(\theta m+\sigma)}$$

Thus, we conclude that I^n exists if $1 < R_0 < \frac{\frac{A}{\mu}\left[\frac{\beta(\mu+\alpha_2+\delta_2(1-k)}{\mu+\delta_2+\alpha_2}+\xi\frac{r_0}{\mu_0}\right]}{\delta_1+\frac{\delta_2}{L_2}(\theta m+\sigma)}$

From equation (3.5) we obtain

$$\mu R^n = \varepsilon Q^n$$

$$R^n = \frac{\varepsilon}{\mu}\times\frac{1}{L_2}\left[k\beta S^n+(\theta m+\sigma)\right]I^n$$

$$R^n = \frac{\varepsilon}{\mu L_2}\left[k\beta\frac{A}{\mu R_0}+(\theta m+\sigma)\right]I^n$$

5. Basic Reproduction Number

We determine the reproduction ratio using the next-generation matrix method as described by P. van den Driessche and Watmough. To achieve this, we divide the model into two compartments, R_1 and R_2 , resulting in the following system form, $X = R_1 - R_2$ where

$$R_1 = \begin{bmatrix} (1-k)\beta IS - \xi VS \\ k\beta IS \\ 0 \\ 0 \\ -\beta SI \end{bmatrix} \quad , R_2 = \begin{bmatrix} -(1-\theta)mI + L_1 I \\ -(\theta m + \sigma)I + L_2 Q \\ -r_0 I + \mu_0 V \\ -\varepsilon Q + \mu R \\ -A - \delta_1 I - \delta_2 Q + \mu S \end{bmatrix}$$

And $$X = \left[\frac{dI}{dt}, \frac{dQ}{dt}, \frac{dv}{dt}, \frac{dR}{dt}, \frac{ds}{dt}\right]$$

Given that the infected compartments are I, Q, and V, the Jacobian matrix $\overset{\square}{R}_1$ and $\overset{\square}{R}_2$ derived from R_1 and R_2 at the disease-free equilibrium are as follows:

$$\overset{\square}{R}_1 = \begin{bmatrix} (1-k)\beta\frac{A}{\mu} & 0 & \frac{\xi A}{\mu} & 0 \\ k\beta\frac{A}{\mu} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \& \quad \overset{\square}{R}_2 = \begin{bmatrix} -(1-\theta)mI + L_1 & 0 & 0 & 0 \\ -(\theta m + \sigma) & L_2 & 0 & 0 \\ -r_0 & 0 & \mu_0 & 0 \\ 0 & -\varepsilon & 0 & \mu \end{bmatrix}$$

Now, we have

$$\overset{\square}{R}_2^{\,-1} = \frac{1}{\left[L_1 - (1-\theta)m\right]L_2\mu_0\mu}\begin{bmatrix} L_2\mu_0 & \varepsilon Q & 0 & 0 \\ 0 & \mu_0\mu & 0 & 0 \\ 0 & r_0\mu & \mu\left[-(1-\theta)m + L_1\right] & 0 \\ L_2 r_0 & r_0\varepsilon & \varepsilon\left[-(1-\theta)m + L_1\right] & L_2\left[-(1-\theta)m + L_1\right] \end{bmatrix}$$

$$\therefore \qquad \mathrm{H} = \overset{\square}{R}_1 . \overset{\square}{R}_2^{\,-1}$$

$$\mathrm{H} = \frac{1}{\left[L_1 - (1-\theta)m\right]L_2\mu_0\mu}\begin{bmatrix} L_2\mu_0\left[(1-k)\beta\frac{A}{\mu}\right] & \varepsilon\mu\left[(1-k)\beta\frac{A}{\mu}\right] + r_0\xi A & \xi A\left[-(1-\theta)m + L_2\right] & 0 \\ L_2\mu_0 k\beta\frac{A}{\mu} & \varepsilon k\beta A & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

It is noted that $\overset{\square}{R}_1$ is a non-negative matrix, while $\overset{\square}{R}_2$ is a non-singular M-matrix with a non-negative inverse, and the product of $\overset{\square}{R}_1$ and $\overset{\square}{R}_2$ is also non-negative. Hence, as per H is identified as the next generation matrix. Consequently, the reproduction number is defined as:

$$R_0 = Max\left\{ \frac{L_2\mu_0(1-k)\beta\dfrac{A}{\mu}}{\left[L_1-(1-\theta)m\right]L_2\mu_0\mu}, \frac{\varepsilon\mu(1-k)\beta\dfrac{A}{\mu}+r_0\xi A}{\left[L_1-(1-\theta)m\right]L_1\mu_0\mu}, \frac{\xi A\left[-(1-\theta)m+L_2\right]}{\left[L_1-(1-\theta)m\right]L_2\mu_0\mu}, 0 \right\}$$

$$R_{0I} = \frac{(1-k)\beta A}{\mu^2\left[L_1-(1-\theta)m\right]} \qquad \text{and} \qquad R_{0V} = \frac{\xi r_0 A}{\left[L_1-(1-\theta)m\right]L_2\mu_0\mu}$$

R_{0I} represents the total number of secondary infections caused by infected individuals, while R_{0V} denotes the total number of secondary infections resulting from a viral load sufficient to spread the infection.

6. Local Stability Analysis:

Local stability analysis of the system reveals how small disturbances affect the system's equilibrium points. The local stability of these equilibrium points is assessed by examining the signs of the real parts of the eigenvalues of the variational matrix. To do this, we derive the general variational matrix of the system, which is obtained as follows:

$$V(\text{E}) = \begin{bmatrix} -\beta I-\xi V-\mu & -\beta S+\delta_1 & \delta_2 & -\xi S & 0 \\ (1-k)\beta I+\xi V & (1-k)\beta S+(1-\theta)m-L_1 & 0 & \xi S & 0 \\ k\beta I & k\beta S+\theta m+\sigma & -L_2 & 0 & 0 \\ 0 & r_0 & 0 & -\mu_0 & 0 \\ 0 & 0 & \varepsilon & 0 & -\mu \end{bmatrix}$$

....(6.1)

6.1 Local Stability Analysis of disease free Equilibrium Point

The variational matrix of the system at the disease-free equilibrium point $E_0\left(\dfrac{A}{\mu},0,0,0,0\right)$ is given by:

$$V(E_0) = \begin{bmatrix} -\mu & -\beta\dfrac{A}{\mu}+\delta_1 & \delta_2 & -\xi\dfrac{A}{\mu} & 0 \\ 0 & (1-k)\beta\dfrac{A}{\mu}+(1-\theta)m-L_1 & 0 & \xi\dfrac{A}{\mu} & 0 \\ 0 & k\beta\dfrac{A}{\mu}+\theta m+\sigma & -L_2 & 0 & 0 \\ 0 & r_0 & 0 & -\mu_0 & 0 \\ 0 & 0 & \varepsilon & 0 & -\mu \end{bmatrix} \qquad ....(6.2)$$

The eigenvalues of the matrix are $-\mu, -\mu_0, -L_2$ and the remaining two eigenvalues are determined from the quadratic equation:

$$\omega^2+\omega\left[\mu_0+L_1-(1-\theta)m\left\{1-\frac{(1-k)\beta\frac{A}{\mu}}{L_1-(1-\theta)m}\right\}\right]+\mu_0\left[-\xi\frac{Ar_0}{\mu\mu_0}+L_1-(1-\theta)m-(1-k)\beta\frac{A}{\mu}\right]=0$$

(6.3)

Based on equation (6.3), we can deduce that both eigenvalues are negative if $R_0<1$

Similarly, by using Lyapunov function we can show that our model system is Global asymptotically stable.

7. Numerical Simulation

In the preceding sections, we scrutinized our mathematical model through analytical means. To validate our analytical conclusions and observe the population's quantitative dynamics over time, we engage in numerical simulation using a specific set of hypothetical parameters.

Table 1 Parameter and values of model system (1-5)

Parameter Value unit

β	0.003 person−1day−1 [25]
ξ	0.002 viral load−1day−1
δ_1	0.01 day−1
δ_2	0.02 day−1
μ	0.03 day−1
k	0.4 day−1
θ	0.02 day−1
m	0.002 day−1
γ_1	0.04 day−1
σ	0.02 day−1
γ_2	0.05 day−1
r_0	0.04 day−1
μ_0	0.03 day−1

For these specified parameters, the basic reproduction number was determined to be 3.38. Additionally, we noted that the reproduction number attributable to the infected population, denoted as R_{0I} , is 1.36, while that stemming directly from the environment, denoted as R_{0v} , is 2.01. This suggests that the number of secondary infections caused by the virus present in the environment exceeds those generated by the infected population alone.

We generated Figures 2 to 6 to examine the temporal variation of different populations. Figure 2 illustrates the variation of all populations over time when considering the presence of the virus population in the environment. Conversely, Figure 3 illustrates this variation without factoring in the virus population in the environment. Our analysis of these figures reveals that when accounting for the virus population, the number of infected individuals is higher compared to scenarios where the virus population is not considered. This observation serves as evidence indicating that the actual number of infected individuals in the system surpasses the reported infected population, affirming our predictions regarding the infected population.

Figure 4 explores the temporal variation of the infected population under different transmission coefficients of infection from the virus and infected individuals [30,31,32]. It is evident that as both λ and β, the transmission coefficients, increase, the infected population also increases. However, the rate of increase in the infected population diminishes with higher values of ξ and β. This observation aligns with the concept of herd immunity, wherein once a certain portion of the population becomes infected, the rate of increase in infections decreases even with higher transmission rates. In Figure 5, the variation of the infected population over time is depicted for varying contact tracing rates of infected and immigrant individuals, represented by k and θ respectively. Notably, when contact tracing of immigrants and infected individuals is conducted at an equal rate, the infected population decreases. Remarkably, the graph reveals that the rate of contact tracing for immigrants significantly impacts the reduction of the infected population. Consequently, it's imperative for governments to enforce stringent measures to trace and quarantine infected immigrants to curb disease transmission. Figure 6 illustrates the variation of the infected population over time for different rates of population immigration. Here, it is observed that the infected population increases with higher rates of immigration of infected individuals. This implies that controlling the disease spread necessitates monitoring and regulating the immigration rate of the population.

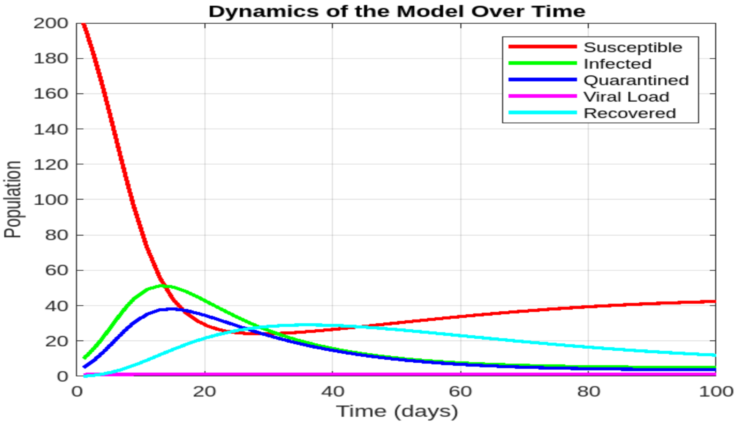

Figure 2. Variation of all population with time when presence of virus in the environment is considered

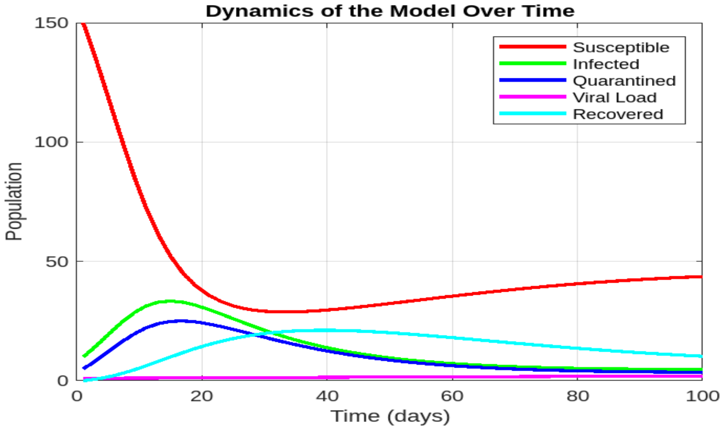

Figure 3. Variation of all population with time when population is not considered in the atmosphere

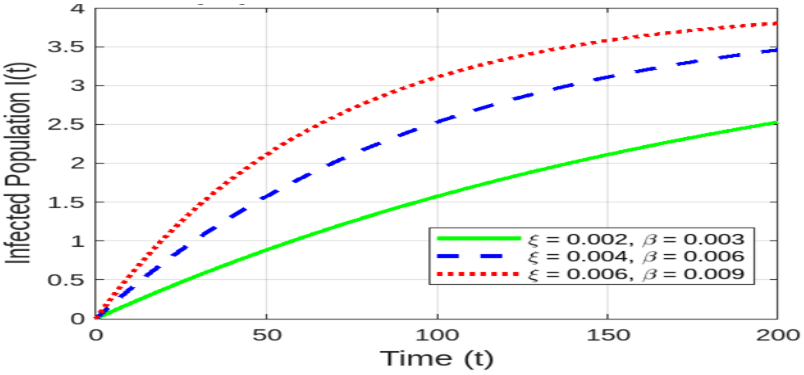

Fig. 4 Variation of infected population with time for different values of ξ and β.

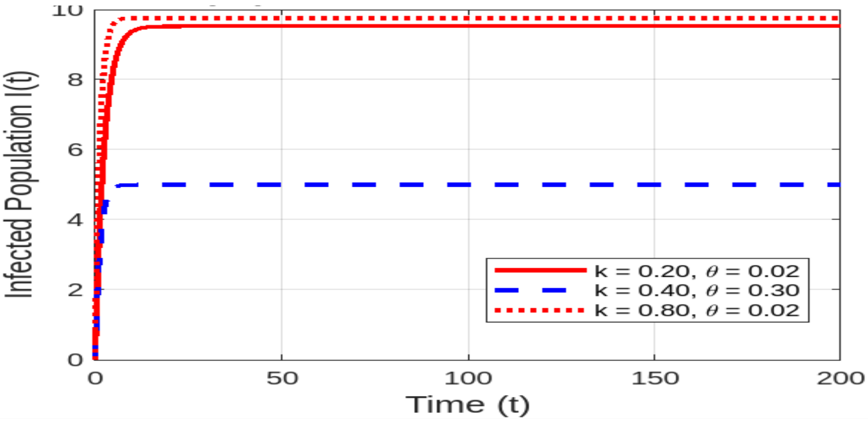

Fig. 5 Variation of infected population with time for different values of k and θ.

8. Results and Discussion

In this paper, we introduce a compartmental mathematical model for Covid-19 that accounts for the immigration of infected individuals and the influence of the virus population. We calculate the basic reproduction number for this model. Our findings indicate that the basic reproduction number consists of two components: one representing the number of secondary infections resulting from transmission by infected individuals, while the second term represents the number of secondary infections due to the transmission of the virus present in the environment. This implies that the virus's ability to survive in the environment plays a significant role in spreading the infection. We conduct an equilibrium analysis of the model, identifying the disease-free and endemic equilibrium points. We find that an endemic equilibrium exists if the basic reproduction number is between one and a specific constant. This suggests that if the basic reproduction number surpasses this constant, herd immunity occurs, leading to a decline in infection. Additionally, our stability analysis shows that the disease-free equilibrium is locally asymptotically stable if the basic reproduction number is less than one, and we identify the conditions for local asymptotic stability of the endemic equilibrium. We also determine the global stability conditions for both equilibrium states, observing that the disease-free equilibrium is globally stable if the basic reproduction number is less than one, while the endemic equilibrium is globally stable if it exceeds one.

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