IJFANS INTERNATIONAL JOURNAL OF FOOD AND NUTRITIONAL SCIENCES
ISSN PRINT 2319 1775 Online 2320 7876
 UGC CARE Listed (Group -I) Journal Volume 12, Iss 01, 2023

A SIQRV MATHEMATICAL MODEL ON COVID-19 INVESTIGATING THE COMBINED EFFECT OF VACCINATION AND LOCKDOWN TO CONTROL THE SPREAD OF COVID-19

Rajan Kumar Dubey[1], Rajesh Pandey[2]
Department of Mathematics and Statistics,
Deen Dayal Upadhyaya Gorakhpur University, Gorakhpur, U.P.(India), 273009.
[1]Email: rajankumardubey40@gmail.com
[2]Email: drrajesh1769@gmail.com, rajesh.math.stat@ddugu.ac.in

ABSTRACT

In this proposed model SIQRV, we will examine about the impact of vaccination and lockdown on COVID-19. In December 2019, novel Corona Virus disease was detected in Wuhan, China. Due to the outbreak scenario throughout the globe, On March 24, 2020, the government of India imposed a total lockdown on the nation and started the vaccination drive from 16 January 2021 to control the spread of COVID-19. We develop a five-dimensional mathematical model utilizing nonlinear ordinary differential equations to examine the effects of vaccination and lockdown on disease dynamics.

This study shows that disease can be vanished only if total lockdown is implemented and everyone is completely vaccinated otherwise this disease will always be present in the system. However, we can keep this disease under control by contact tracing, quarantine measures, increasing rate of vaccination and implementing partial lockdown.

Keywords: *System, Contact tracing, COVID-19, Transmission, Equilibrium analysis, Numerical simulation.*

I. INTRODUCTION

In a medical institute in Hubei province China, some patients with pneumonia of unknown reasons came to light in the month of December 2019. A special type of coronavirus was found as the innovatory reason for pneumonia and it was known as 2019-ncov. WHO termed this disease: COVID-19. Covid-19 has spread in maximum parts of the world within a very less time. It spreads very fast when an infected person comes into contact with another person i.e., human to human infection. The outbreak later inflicted more than 200 countries and the disease was declared a pandemic in March 2020 due to a large number of deaths. Infectious disease COVID-19 spread due to the movement of infected people from China to other countries. Since no vaccine or drug for treating the virus was available, China tried to control the outbreak in Wuhan by imposing a lockdown. A literature report in France (Barnett and Walker, 2008) says that lockdown is powerful because it decreases the rate of transmission of COVID-19 by 84%. Fraser et al. (Fraser et al.,2004) state: if a large number of infections are occurring due to asymptomatic cases as in the case of COVID-19, then quarantine becomes an important measure to control the outbreak. In the next study of SARS, the author states that this outbreak could be controlled by the isolation of asymptomatic individuals. A study by Chen et al. (Chen et al., 2006) states that we can control the SARS outbreak by isolating symptomatic patients and lesser contact tracing. The next research paper by A. S. Bhadauria, R. Pathak, and M. Chaudhary (A. S. Bhadauria, R. Pathak and M. Chaudhary, 2020)

studied the combined lockdown impact, quarantine, and contact tracing. In the reference of the research paper from serial number [20] to [29] contains different kinds of mathematical models based on COVID-19, but no work in the previous mathematical model could study the combined effect of lockdown, quarantine, and contact tracing and vaccination. We will expand this research paper on our findings.

Our study is dependent on the construction of a mathematical model to control the spread of COVID-19 by using vaccination with lockdown and observing their impact. The rest of this article is organized as follows: The proposed model is provided in a mathematical form, and its mathematical analysis was given after that. The result we got after the numerical simulation of the model is given in the numerical simulation section. Lastly, in the conclusion, section all results we obtained after that study are given.

II. MATHEMATICAL MODEL

We contemplate a five-dimensional model having susceptible population S(t), infected population I(t), quarantined population Q(t), recovered population R(t), and vaccinated population V(t). We supposed that the population is evenly distributed and disease is spreading in the system due to the direct contact with the susceptible class and infected individual class. Also due to the immigration of individuals the disease spreads in a susceptible population.

Hence, with immigration, we consider a SIQRV mathematical model. Under the region of consideration at any time t, we assume that N(t) denotes the total population. It can be obtained by adding the five subpopulations. The system gate-crashes due to Susceptible population having constant rate 'A' with immigration $(1-\theta)$ m. Here, m represents the number of migrants, while $theta $ represents the proportion of infected migrants. We suppose that a quotient of immigrants is infected which is responsible for spreading the virus. In the infected population, quotient θm is entered directly. Assume that k denotes the rate of contact tracing of the individual population. Thus, a quotient $(1-k)$ is culpable for diffusing infection, and the remaining are isolated. To model the communication between the susceptible and infected class we considered Holling type-II functional because it is responsible for that. The natural mortality rate is denoted by μ of the population in each class, α_1 and α_2 respectively denote death rates due to disease related to the infected and quarantined population, whereas δ_1 and δ_2 respectively denotes the recovery rate of the infected and quarantined population. Here σ denotes the transmission rate of infected to quarantined population. ξ denotes the vaccination rate of a recovered person. The vaccination rate of susceptible people who are neither infected nor quarantined is γ We take ψ as a positive constant. Thus, with this thought as system variables, we considered a five-dimensional epidemiological model by using susceptible S(t), infected I(t), quarantined Q(t), recovered R(t) and vaccinated V(t) population.

Figure 1 shows the flow diagram for the SIQRV COVID-19 model.

ISSN PRINT 2319 1775 Online 2320 7876
 UGC CARE Listed (Group -I) Journal Volume 12, Iss 01, 2023

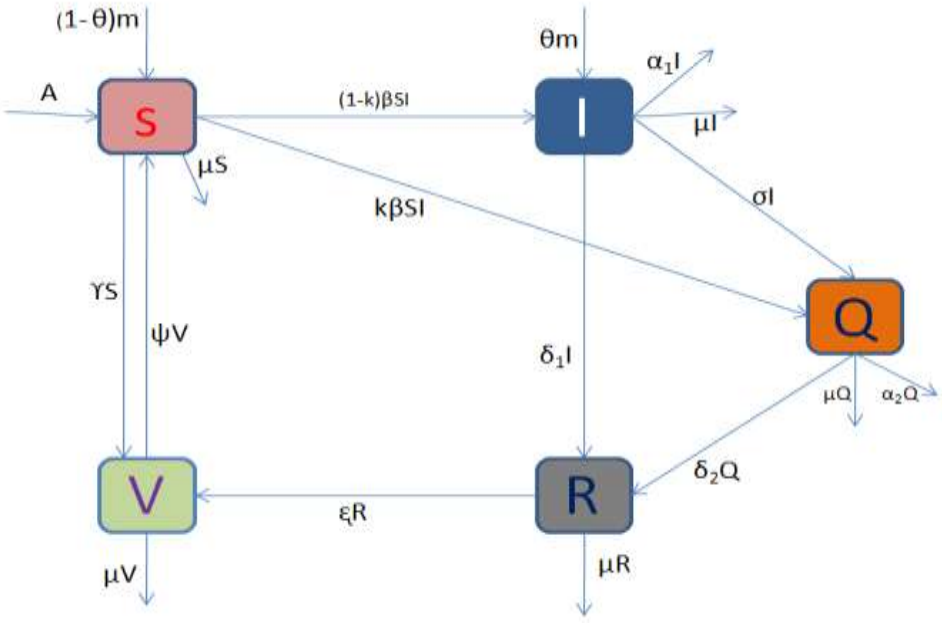

Figure 1: The SIQRV COVID-19 model flow diagram

Now,

$$\frac{dS}{dt} = A + (1-\theta)m - \beta\, SI - \mu S - \gamma S + \psi V, \qquad \ldots(2.1)$$

$$\frac{dI}{dt} = (1-k)\beta SI + \theta m - \alpha_1 I - \mu I - \sigma I - \delta_1 I, \qquad \ldots(2.2)$$

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

$$\frac{dR}{dt} = \delta_1 I + \delta_2 Q - \mu R - \xi R, \qquad \ldots(2.4)$$

$$\frac{dV}{dt} = \gamma S + \xi R - \mu V - \psi V, \qquad \ldots(2.5)$$

with the initial conditions

$$S(0) > 0,\ I(0) \geq 0, Q(0) \geq 0,\ R(0) \geq 0 \text{ and } V(0) \geq 0\,.$$

The parameters are described in Table (1)

Table 1: Table of Description

Parameter	Description
A	Recruitment rate
β	Rate of infection due to transmission
M	Natural death rate
M	Number of migrants
Θ	Infected migrants rate
K	Rate of contact tracing
δ_1	Recovery rate of infected population
δ_2	Recovery rate of quarantined population
σ	Rate transmission from infected to quarantined population
α_1	Death rate of infected population due to disease
α_2	Death rate of quarantined population due to disease

ξ	Vaccinated rate of recovered person
ψ	A positive constant
γ	Vaccination rate of susceptible people who are neither infected nor quarantined

III. BOUNDEDNESS OF THE SYSTEM

Now we study the system variable boundaries as it is useful to prove some results by using the lemma given below. As a natural restriction boundedness of the system can be explicated to an uncertain increment of the infected population in the system. Due to various constraints either from natural conditions or from precautionary behavior like vaccination which is used by the population for protection from disease. We must demonstrate that the system's solutions are bounded in order to confirm the model's biological applicability.

1. Lemma:- Let $\Omega = \{(S, I, Q, R, V): 0 \leq N = S + I + Q + R + V \leq N_m\}$ be a field where every solution attracts which are initiating from the internal part of the positive octant,

Where, $N_m = \frac{A+m}{\mu}$.

Thus, $$\frac{dN}{dt} = A + (1-\theta)m - \beta SI - \mu S - \gamma S + \psi V + (1-k)\beta SI + \theta m - \alpha_1 I - \mu I - \sigma I - \delta_1 I + k\beta SI + \sigma I - \alpha_2 Q - \mu Q - \delta_2 Q + \delta_1 I + \delta_2 Q - \mu R - \xi R + \gamma S + \xi R - \mu V - \psi V$$

$$\frac{dN}{dt} = A + m - \mu(S + I + Q + R + V) - \alpha_1 I - \alpha_2 Q$$

$$\frac{dN}{dt} \leq A - \mu N + m. \qquad \ldots(3.1)$$

From comparison principle, it is observed that

$$N_m = \frac{A+m}{\mu}. \qquad \ldots(3.2)$$

IV. DYNAMIC MODEL WITHOUT LOCKDOWN

If we consider $m \neq 0$ then in this case system (2.1) to (2.5) posses no equilibrium point which is disease free. It has only pandemic equilibrium point. Hence, in the system if we allowed the immigration of population which involves some infected people then the disease will never tend toward zero but the spread of disease can be controlled in the system.

4.1 Equilibrium analysis

If there is no change with respect to time in a state of dynamical system then that point is called equilibrium point. Hence if a system starts from a point of equilibrium, then state of the system will always persist in equilibrium. Now our task is to find disease free equilibrium point. Then from equation (2.1) to (2.5), only pandemic equilibrium point $E^m(S^m, I^m, Q^m, R^m, V^m)$ exists.

The values of S^m, I^m, Q^m, R^m, and V^m are given as

$$S^m = \frac{(\alpha_1+\mu+\sigma+\delta)I^m - \theta m}{(1-k)\beta I^m},$$

$$Q^m = \frac{k(\alpha_1+\mu+\delta_1)I^m - k\theta m + \sigma I^m}{(1-k)(\alpha_2+\mu+\delta_2)},$$

$$R^m = \frac{1}{(\mu+\xi)}\left[\delta_1 I^m + \frac{\delta_2 k(\alpha_1+\mu+\delta_1)I^m - k\theta m + \sigma I^m)}{(1-k)\beta I^m}\right],$$

$$V^m = \frac{\gamma}{(\mu+\psi)}\left[\frac{(\alpha_1+\mu+\sigma+\delta)I^m - \theta m}{(1-k)\beta I^m}\right] + \frac{\xi}{(\mu+\xi)}\left[\delta_1 I^m + \frac{\delta_2 k(\alpha_1+\mu+\delta_1)I^m - k\theta m + \sigma I^m)}{(1-k)\beta I^m}\right]$$

and I^m is given by the root of the quadratic equation (4.1)

$$B_1 I^{m2} + B_2 I^m - B_3 = 0. \qquad \ldots(4.1)$$

where the coefficient of the above equation are

$$B_1 = -\beta\,(\alpha_1 + \mu + \sigma + \delta_1)\{\mu + \psi\}(\alpha_2 + \mu + \sigma + \delta_2)(\mu + \xi) + \beta\{\mu + \psi\}[\psi(1-k)(\alpha_2 + \mu + \delta_2) + \delta_2 k\,\xi(\alpha_1 + \mu + \delta_1)(\mu + \xi) + \sigma\,\xi(\mu + \xi)],$$

$$B_2 = [\{A + (1-\theta)m\}(1-k)\beta - (\mu + \gamma)(\alpha_1 + \mu + \sigma + \delta_1) - \theta\, m\,\beta][\{\mu + \xi\}(\mu + \xi)(\alpha_2 + \mu + \delta_2)] + (\mu + \xi)(\alpha_2 + \mu + \delta_2)\gamma\,(\alpha_1 + \mu + \sigma + \delta_1) - k\,\theta\, m\,\xi\,\delta_2\,(\mu + \xi)\beta\{\mu + \psi\},$$

and

$$B_3 = \{\mu + \xi\}(\alpha_2 + \mu + \delta_2)(\mu + \xi)\theta\, m\,(\mu + \gamma) + \gamma\,\theta\, m\,(\mu + \xi)\,(\alpha_2 + \mu + \delta\_2).$$

This shows that the uniqueness of positive equilibrium point E^m exist if and only if

$$\sqrt{\{B_2^2 + 4B_1B_2\}} > B_2. \qquad \ldots(4.2)$$

4.2 Analysis of local stability

In the system's equilibrium state the analysis of local stability at the point of equilibrium shows discernment into the affect of perturbations. To learn about the epidemic equilibrium's stability point, our system can be linearize with respect to the random equilibrium points $E^m(S^m, I^m, Q^m, R^m, V^m)$ and we obtain the corresponding matrix known as Jacobian matrix that is given by

$$J_E = \begin{bmatrix} b_{11} & b_{12} & 0 & 0 & b_{15} \\ b_{21} & b_{22} & 0 & 0 & 0 \\ b_{31} & b_{32} & b_{33} & 0 & 0 \\ 0 & b_{42} & b_{43} & b_{44} & 0 \\ 0 & 0 & 0 & b_{54} & b_{55} \end{bmatrix}$$

Where entries of the matrix J_E are given by

$b_{11} = -\beta I - (\mu + \gamma)$, $b_{12} = \beta S$, $b_{15} = \psi$,
$b_{21} = (1-k)\beta I$, $b_{22} = (1-k)\beta - (\alpha_1 + \mu + \sigma + \delta_1)$, $b_{31} = k\beta I$,
$b_{32} = k\beta S + \sigma$, $b_{33} = -(\alpha_2 + \mu + \delta_1)$, $b_{15} = \delta_1$,
$b_{43} = \delta_2$, $b_{44} = -(\mu + \xi)$, $b_{54} = \xi$,
$b_{55} = -(\mu + \psi)$.

Proposition. Equilibrium point $E^m(S^m, I^m, Q^m, R^m, V^m)$ of the system (2.1) to (2.5) is locally asymptotically stable if $x_1 > 0$, $b_1 > 0$, $c_1 > 0$, $d_1 > 0$ *and* $e_1 > 0$.

Proof:- By dint of Jacobian matrix J_E we notice that the eigen value of $E^m(S^m, I^m, Q^m, R^m, V^m)$ are given by the root of the equation

$$\lambda^5 + x_1\lambda^4 + x_2\lambda^3 + x_3\lambda^2 + x_4\lambda + x_5 = 0. \qquad \ldots(4.3)$$

$$x_1 = -(b_{11} + b_{22} + b_{33} + b_{44} + b_{55}),$$

$$x_2 = b_{11}b_{22} + b_{11}b_{44} + b_{22}b_{44} + b_{44}b_{33} - b_{11}b_{33} - b_{22}b_{33} + b_{55}b_{11} + b_{55}b_{22} + b_{55}b_{33} + b_{55}b_{44} + b_{21},$$

$$x_3 = b_{11}b_{22}b_{33} - b_{11}b_{22}b_{44} + b_{11}b_{44}b_{33} + b_{22}b_{33}b_{44} - b_{55}b_{11}b_{22} - b_{55}b_{11}b_{44} - b_{55}b_{22}b_{44} - b_{55}b_{44}b_{33} + b_{55}b_{11}b_{33} + b_{55}b_{22}b_{33} - b_{21b_{33}} - b_{21}b_{44} - b_{21}b_{55},$$

$$x_4 = b_{33}b_{44} - b_{33}b_{55} - b_{44}b_{55} - b_{31}b_{43}b_{54} - b_{21}b_{42}b_{55},$$

$$x_5 = b_{11}b_{22}b_{33}b_{44}b_{55} - b_{33}b_{44}b_{55} - b_{31}b_{43}b_{54}b_{22} - b_{31}b_{43}b_{54}b_{33} + b_{21}\,b_{32}b_{44}b_{55}.$$

For local stability we use Routh Hurwitz stability criteria as:-

Now, from above equation we will form Routh Array as shown below

λ^5	x_0	x_2	x_4
λ^4	x_1	x_3	x_5
λ^3	b_1	b_2	0
λ^2	c_1	c_2	
λ^1	d_1	0	
λ^0	e_1		

Where,

$$b_1 = \frac{x_1x_2 - x_3}{x_1}, \qquad c_2 = \frac{b_1\ x_5 - 0}{b_1} = x_5,$$
$$b_2 = \frac{x_1x_4 - x_5}{x_1}, \qquad d_1 = \frac{c_1b_2 - b_1c_2}{c_1},$$
$$c_1 = \frac{b_1x_3 - b_2x_1}{b_1}, \qquad e_1 = \frac{d_1c_2}{d_1} = c_2.$$

Hence by Routh-Hurwitz criteria the system is said to be locally asymptotically stable if all the element in the first column are positive it means that there is no change in sign.

i.e. $x_1 > 0,\ b_1 > 0,\ c_1 > 0,\ d_1 > 0\ and\ e_1 > 0$

4.3 Persistence of the model

Now In this part, we will look upon the continuance of system and we will also find the condition by which a disease can persist in the system. 'Persistence' defines the long-term survival of each individual.

2.Lemma:- Suppose that $(\alpha_1 + \mu + \sigma + \delta_1) > (1 - k)\beta\, S_{min}$. The upper limit of population $S, I, Q, R\ and\ V$ is denoted by N_m. They are always greater than zero. Thus the system of equation (2.1) to (2.5) persevere.

Proof: According to the equation (2.1)

$$\frac{dS}{dt} \geq A + (1-\theta)m - (\beta N_m + \mu + \gamma) S + \psi V. \qquad \ldots(4.4)$$

then using boundedness and comparison principle, we have

$$S_{min} = \frac{\{A + \psi V_{min}}{(\beta N_m + \mu + \gamma)} \qquad \ldots(4.5)$$

Thus, S_{min} is always positive.

Now from equation (2.2), we have

$$\frac{dI}{dt} \geq \theta m + \{(\alpha_1 + \mu + \sigma + \delta_1) - (1-k)\beta S_{min}\} I. \qquad \ldots(4.6)$$

Again, using boundedness and comparison principle, we have

$$I_{min} = \frac{\theta m}{(\alpha_1 + \mu + \sigma + \delta_1) - (1-k)\beta S_{min}}. \qquad \ldots(4.7)$$

with condition $(\alpha_1 + \mu + \sigma + \delta_1) > (1 - k)\beta\, S_{min}$.

Thus I_{min} always remains positive. From the equation (2.3), we have

$$\frac{dQ}{dt} \geq k\beta S_{min} I_{min} + \sigma I_{min} - (\alpha_2 + \mu + \delta_2) Q. \qquad \ldots(4.8)$$

By using boundedness and comparison principle, we have

$$Q_{min} = \frac{k\beta S_{min} I_{min} + \sigma I_{min}}{(\alpha_2 + \mu + \delta_2)}. \qquad \ldots(4.9)$$

Thus Q_{min} always remains positive. From the equation (2.4), we have

$$\frac{dR}{dt} \geq \delta_1 I_{min} + \delta_2 Q_{min} - (\mu + \xi) R. \quad \ldots(4.10)$$

Again, using boundedness and comparison principle, we have

$$R_{min} = \frac{\delta_1 I_{min} + \delta_2 Q_{min}}{\mu + \xi}. \quad \ldots(4.11)$$

Thus Q_{min} always remains positive. From the last equation (2.5), we have

$$\frac{dS}{dt} \geq \gamma S_{min} + \xi R_{min} - (\mu + \psi)V. \quad \ldots(4.12)$$

By using boundedness and comparison principle, we have

$$V_{min} = \frac{\gamma S_{min} + \xi R_{min}}{(\mu + \psi)}. \quad \ldots(4.13)$$

Hence V_{min} always remains greater than zero.

Hence proved.

V. DYNAMIC MODEL WITH LOCKDOWN EFFECT

Now to understand the behavior of lockdown, consider (2.1) to (2.5) for m=0 in our model. It means exodus of the population is forcefully prohibited in system.

In that situation, the model can be written as the following system of equation (5.1) to (5.5).

$$\frac{dS}{dt} = A - \beta SI - \mu S - \gamma S + \psi V, \quad \ldots(5.1)$$

$$\frac{dI}{dt} = (1-k)\beta SI - \alpha_1 I - \mu I - \sigma I - \delta_1 I, \quad \ldots(5.2)$$

$$\frac{dQ}{dt} = k\beta SI + \sigma I - \alpha_2 Q - \mu Q - \delta_2 Q, \quad \ldots(5.3)$$

$$\frac{dR}{dt} = \delta_1 I + \delta_2 Q - \mu R - \xi R, \quad \ldots(5.4)$$

$$\frac{dV}{dt} = \gamma S + \xi R - \mu V - \psi V, \quad \ldots(5.5)$$

having the conditions that $S(0)$, $I(0)$, $Q(0)$, $R(0)$ and $V(0)$ are positive.

The disease free equilibrium point at initial point $S(0)$, $I(0)$, $Q(0)$, $R(0)$ and $V(0)$

Then, $A - 0 - \mu S - \gamma S + 0 = 0.$

Or,

$$S = \frac{A}{\mu + \gamma}. \quad \ldots(5.6)$$

5.1 Basic reproduction number

If each individual is susceptible in a population then the basic reproduction number is defined as a threshold digit that gives a value of secondary infections created by an infected individual class in total infection duration. We can calculate the basic reproduction number by a process known as the next-generation matrix. We partition the model into subpart R_1 and R_2 then system (5.1) to (5.5) takes the form as :-

$$X = R_1 - R_2. \quad \ldots(5.7)$$

Where,

$$R_1 = \begin{bmatrix} (1-k)\beta SI \\ k\beta SI \\ 0 \\ 0 \\ -\beta SI + A \end{bmatrix}, \qquad R_2 = \begin{bmatrix} (\alpha_1 + \mu + \sigma + \delta_1)I \\ -\sigma I + (\alpha_2 + \mu + \delta_2)Q \\ -\delta_1 I - \delta_1 Q + \mu R + \xi R \\ -\gamma S - \xi R + \mu V + \psi V \\ \mu S + \gamma S - \psi V \end{bmatrix}$$

and $X = \left[\frac{dI}{dt}, \frac{dQ}{dt}, \frac{dR}{dt}, \frac{dV}{dt}, \frac{dS}{dt}\right]$.

In the case of disease free equilibrium point the infected compartment $S, I, Q, R \; and \; V$.

$$\widetilde{R_1} = \left[\frac{\delta(R_1)_i}{\delta x_j}\right] \; \& \; \widetilde{R_2} = \left[\frac{\delta(R_2)_i}{\delta x_j}\right] \; for \; 1 \leq i,j \leq 4 .$$

Thus, $\widetilde{R_1} = \begin{bmatrix} \frac{(1-k)\beta A}{\mu+\gamma} & 0 & 0 & 0 \\ \frac{k\beta A}{\mu+\gamma} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$ &

$$\widetilde{R_2} = \begin{bmatrix} \alpha_1+\mu+\sigma+\delta_1 & 0 & 0 & 0 \\ -\sigma & \alpha_2+\mu+\delta_2 & 0 & 0 \\ -\delta_1 & -\delta_2 & \mu+\xi & 0 \\ 0 & 0 & -\xi & \mu+\xi \end{bmatrix}.$$

Here $\widetilde{R}_1 \geq 0$ and $\widetilde{R}_2$ is a matrix whose determinant is not equal to zero $(\widetilde{R}_2)^{-1} \geq 0$ and $\widetilde{R}_1 (\widetilde{R}_2)^{-1}$ as non negative matrix; $\widetilde{R}_1 (\widetilde{R}_2)^{-1}$ is the next generation matrix.

Now,

$$(\widetilde{R}_2)^{-1} = \begin{bmatrix} \frac{1}{\alpha_1+\mu+\sigma+\delta_1} & 0 & 0 & 0 \\ b_{21} & \alpha_2+\mu+\delta_2 & 0 & 0 \\ b_{31} & b_{32} & \frac{1}{\mu+\xi} & 0 \\ b_{41} & b_{42} & b_{43} & \frac{1}{\mu+\xi} \end{bmatrix}$$

Where,

$$b_{21} = \frac{-\sigma}{(\alpha_1+\mu+\sigma+\delta_1)(\alpha_2+\mu+\delta_2)},$$

$$b_{31} = \frac{\sigma\delta_2+\delta_1(\alpha_2+\mu+\delta_2)}{(\alpha_1+\mu+\sigma+\delta_1)(\alpha_2+\mu+\delta_2)(\mu+\xi)},$$

$$b_{32} = \frac{\delta_2}{(\alpha_2+\mu+\delta_2)(\mu+\xi)},$$

$$b_{41} = \frac{\xi[\sigma\delta_2+\delta_1(\alpha_2+\mu+\delta_2)]}{(\alpha_1+\mu+\sigma+\delta_1)(\alpha_2+\mu+\delta_2)(\mu+\xi)(\mu+\psi)},$$

$$b_{42} = \frac{\xi\delta_2}{(\alpha_2+\mu+\delta_2)(\mu+\xi)(\mu+\psi)},$$

$$b_{43} = \frac{\xi}{(\mu+\xi)(\mu+\psi)}.$$

In this case

$$\widetilde{R}_1 (\widetilde{R}_2)^{-1} = \begin{bmatrix} \frac{(1-k)\beta A}{(\mu+\gamma)(\alpha_1+\mu+\sigma+\delta_1)} & 0 & 0 & 0 \\ \frac{k\beta A}{(\mu+\gamma)(\alpha_1+\mu+\sigma+\delta_1)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

Then the spectral eigen value of the matrix is

$$\lambda = \frac{(1-k)\beta A}{(\mu+\gamma)(\alpha_1+\mu+\sigma+\delta_1)} \qquad \ldots(5.8)$$

As a result, the basic reproduction number is provided by

$$R_0 = \frac{(1-k)\beta\, A}{(\mu+\gamma)(\alpha_1+\mu+\sigma+\delta_1)} \qquad \ldots(5.9)$$

We conclude from the above expression that in disease free state R_0 is directly proportional to $\frac{A}{\mu+\gamma}$ which denotes susceptible population, $(\alpha_1+\mu+\sigma+\delta_1)$ which denotes infection period and β which denotes transmission rate. It is inversely proportional to the frequency of contact tracing k and with the rate of vaccination. Thus, by controlling the value of these parameters we can control spread of the disease.

5.2 Sensitivity analysis of basic reproduction number R_0

To calculate sensitivity of R_0 we studied about sensitivity analysis which depends on every parameter of R_0. Now to control the spread of corona virus, we have to make the value of $R_0 < 1$ by controlling the values of parameter involved in R_0 . By changing the parameter values we can study about rate of change of R_0. It can be estimated by change in h, where h is a parameter

$$\mathrm{SI[h]} = \frac{h}{R_0}.\frac{\partial R_0}{\partial h}.$$

The sensitivity indices that are normalized of the reproduction number regards to parameter k, μ, γ, α_1, β, σ, δ_1 are given by

$$\mathrm{SI[k]} = \frac{-k}{1-k} < 1,$$
$$\mathrm{SI[\mu]} = \frac{\mu\{(\alpha_1+\mu+\sigma+\delta_1)+(\mu+\gamma)\}}{(\mu+\gamma)(\alpha_1+\mu+\sigma+\delta_1)} < 1,$$
$$\mathrm{SI[\gamma]} = \frac{-\gamma}{\mu+\gamma} < 1,$$
$$\mathrm{SI[\alpha_1]} = \frac{-\alpha_1}{(\alpha_1+\mu+\sigma+\delta_1)} < 1,$$
$$\mathrm{SI[\beta]} = 1,$$
$$\mathrm{SI[\sigma]} = \frac{-\sigma}{(\alpha_1+\mu+\sigma+\delta_1)} < 1,$$
$$\mathrm{SI[\delta_1]} = \frac{-\delta_1}{(\alpha_1+\mu+\sigma+\delta_1)} < 1.$$

After we studies about above mathematical expression we observed that when β is changed slightly R_0 is very sensitive on it. If we increase the value of β it will increase the value of R_0 and remaining of parameter values are negative means that R_0 reduces if k, μ, γ, α_1, β σ and δ_1 increase. Thus, the value of R_0 increases as the transmission rate of infection increases and decreases with the rate of contact tracing, vaccinated rate of susceptible population who are neither infected nor quarantined, disease related death rate of quarantined population, transition rate from infected class to quarantined class, recovery rate of infected class and rate of natural death of every class of population.

5.3 Analysis of steady-state equilibrium and equilibrium stability

There are two equilibrium points of the System $(5.1)\ to\ (5.5)$. The first is known as the disease free equilibrium point, while the second is known as the pandemic equilibrium point.

Theorem. There is a unique disease free equilibrium point of the SIQRV model $(5.1) - (5.5)$. $E_0(\frac{A}{\mu+\gamma}, 0,\ 0,\ 0, 0)$ for each values of parameter. The above considered model has also another equilibrium point which is unique and known as pandemic equilibrium $\widetilde{E}(\tilde{S},\ \tilde{I},\ \tilde{Q},\ \tilde{R},\ \tilde{V})$. From equation $(5.1)\ to\ (5.5)$. we have,

$$\tilde{S} = \frac{\alpha_1+\mu+\delta_1}{(1-k)\beta},$$
$$\tilde{Q} = \left[\frac{k(\alpha_1+\mu+\sigma+\delta_1)+\sigma}{(\alpha_2+\mu+\delta_1)(1-k)}\right] I,$$

$$\tilde{R} = \frac{1}{\mu+\xi}\left[\delta_1 + \frac{\delta_2\{k(\alpha_1+\mu+\sigma+\delta_1)+\sigma\}}{(\alpha_2+\mu+\delta_1)(1-k)}\right] I,$$

$$\tilde{V} = \frac{1}{\mu+\psi}\left[\frac{1}{\gamma}(\alpha_1+\mu+\sigma+\delta_1)(1-k)\beta + \frac{\xi}{\mu+\xi}\left\{\delta_1 + \frac{\delta_2\{k(\alpha_1+\mu+\sigma+\delta_1)+\sigma\}}{(\alpha_2+\mu+\delta_1)(1-k)}\right\} I\right],$$

$$\tilde{I} = \left[\frac{\psi}{\mu+\psi}\left\{\frac{\xi}{\mu+\xi}\left\{\delta_1 + \frac{\delta_2\{k(\alpha_1+\mu+\sigma+\delta_1)+\sigma\}}{(\alpha_2+\mu+\delta_1)(1-k)}\right\}\right\} - \frac{\alpha_1+\mu+\sigma+\delta_1}{(1-k)}\right]^{-1}\left[(\mu+\gamma)\frac{(\alpha_1+\mu+\sigma+\delta_1)}{(1-k)\beta} + \frac{\psi(\alpha_1+\mu+\sigma+\delta_1)}{(\mu+\psi)(1-k)\beta} - A\right].$$

5.4 Analysis of local stability

We have to learn about the consistency of the distinct type of point of equilibrium for this Variational matrix is as follows:

$$J_E = \begin{bmatrix} a_{11} & a_{12} & 0 & 0 & a_{15} \\ a_{21} & a_{22} & 0 & 0 & 0 \\ a_{31} & a_{32} & a_{33} & 0 & 0 \\ 0 & a_{42} & a_{43} & a_{44} & 0 \\ 0 & 0 & 0 & a_{54} & a_{55} \end{bmatrix}$$

Where the entries of the matrix are given by

$a_{11} = -\beta I - (\mu + \gamma)$, $a_{12} = \beta S$, $a_{15} = \psi$,
$a_{21} = (1-k)\beta I$, $a_{22} = (1-k)\beta - (\alpha_1 + \mu + \sigma + \delta_1)$, $a_{31} = k\beta I$,
$a_{32} = k\beta S + \sigma$, $a_{33} = -(\alpha_2 + \mu + \delta_1)$, $a = \delta_1$,
$a_{43} = \delta_2$, $a_{44} = -(\mu + \xi)$, $a_{54} = \xi$,
$a_{55} = -(\mu + \psi)$.

5.4.1 Analysis of local stability in a disease-free equilibrium

From equation (5.1) *to* (5.5), E_0 is locally asymptotically stable if $x_1 > 0$, $b_1 > 0$, $c_1 > 0$, $d_1 > 0$ *and* $e_1 > 0$. It is clear from the previous finding in local stability analysis of pandemic equilibrium point.

5.4.2 Analysis of global stability in a disease-free equilibrium

Axiom :- If $R_0 < 1$ in the region R^{+2} of I-Q plane then system of equation (5.1) to (5.5), E_0 is globally asymptotically stable.

$$\frac{dI}{dt} = (1-k)\beta SI - (\alpha_1 + \mu + \sigma + \delta_1)I = f_1, \quad \ldots(5.10)$$

$$\frac{dQ}{dt} = k\beta SI + \sigma I - (\alpha_2 + \mu + \delta_2)Q = f_2 \quad \ldots(5.11)$$

Suppose $g(I,Q) = \frac{1}{IQ}$,

$$L(I,Q) = \frac{\partial}{\partial I}(gf_1) + \frac{\partial}{\partial I}(gf_2). \quad \ldots(5.12)$$

as $g(I,Q)$ is positive for each I, Q is positive so, we have

$$L(I,Q) = \frac{\partial}{\partial I}\left[\frac{1}{Q}(1-k)\beta S - \frac{1}{Q}(\alpha_1 + \mu + \sigma + \delta_1\right] + \frac{\partial}{\partial Q}\left[\frac{1}{Q}(k\beta S + \sigma) - \frac{1}{I}(\alpha_2 + \mu + \delta_2)\right]$$

$$= -\frac{1}{Q^2}(k\beta S + \sigma)$$

$$< 0.$$

i.e.,

$$L(I,Q) < 0. \quad \ldots(5.13)$$

Thus, we observed that there is no change in sign of $L(I,Q)$ and also in positive Quadrant of I,Q plane it is not equal to zero identically. Then in the same plane the criteria of Bendixon-Dulac says that there is no limit cycle. Therefore, when $R_0 < 1$ it is always locally asymptotically stable disease free equilibrium and in (I-Q) plane which is a part of region R^{+2} if $R_0 < 1$ is globally stable. Biologically this theorem can be explained as, when $R_0 < 1$ system will go under an equilibrium state which is disease free and it does not depend on size of the agitation or what we choose as the initial point to start it. The above model will always attain an equilibrium state which is disease free and disease tends toward zero from the system.

VI. NUMERICAL SIMULATION

Now, here we observed about quantitative nature of the above model during the lockdown period and after that the effect of vaccination to control the spread of COVID-19 in India. Using MATLAB, we defend the analytical calculations and carry a numerical simulation using mathematical software where once we allow immigration and once we don't allow the immigration of individuals in the system. The parameter used to perform numerical simulation is described in Table 2. Some of the parameters are assumed and most of them are taken from the previous literature.

Without immigration, we found the system's equilibrium points when the government imposed a total lockdown. Here we discuss the equilibrium points of the system, first one is known as disease-free equilibrium point $(1.3964 \times 10^9, 0,0,0,0)$ and the second one is endemic equilibrium point $(1.3964 \times 10^9, 1.5158 \times 10^4, 2.12212 \times 10^5, 1.75 \times 10^2, 1.243 \times 10^6)$. We calculate the basic reproduction ratio R_0 which is equal to $.24$. By the above set of parameters, we verified every condition of asymptotic stability whether they are local or global. The requirement of local equilibrium point stability and model persistence is satisfied by the above set of parameters. On the infected population, we create population time series graphs. which are infected and study various parameters. The fluctuation in the graph of the infected population with respect to time and we observe the nature of the graph by changing the values of the transmission rate of infection which is shown in Figure 2. Figure 3 shows the impact of the rate of transition from the infected population to the quarantine population. We observed that by increasing in transition rate there is a decrease in the infected population. This shows that controlling the number of infected population σ plays a significant role and hence, also control the disease. On the infected population, the influence of contact tracking is shown in Figure 4. From the figure, we observed that if the number of immigrants increases in the system the number of infected individuals also increases in the system. Thus here we observe that for keeping the disease under control contact tracing, quarantine, and complete/partial lockdown play a very important role. If they are absent then the spread of COVID-19 cases will increase at a very high rate. From figure 5 we observed that as the transmission rate of infection 'β' decreases and the rate of vaccination 'γ' increases the infected population decreases very fast means the disease can be controlled by increasing the rate of vaccination 'γ'. Thus vaccination plays a significant role to control the disease.

The table(2) represents Details of parameters used.

Table 2: Details of the parameter

Parameter	Value	Reference
A	67302 person per day	Mohsen et al. (2020)
β	2.1×10^{-8}	Vega(2020)
μ	0.00002	Mandal et al. (2020)

ISSN PRINT 2319 1775 Online 2320 7876
 UGC CARE Listed (Group -I) Journal Volume 12, Iss 01, 2023

m	2500 persons	Marimuthu et al. (2020)
θ	0.9 per day	Assumed
k	0.5 per day	Assumed
δ_1	0.4 per day	Liang (2020)
δ_2	0.4 per day	Liang (2020)
σ	0.59 per day	Assumed
α_1	1.78×10^{-5}	Vega (2020)
α_2	1.78×10^{-5}	Vega (2020)
ξ	0.90	WHO
ψ	1	Assumed
γ	0.0002	Assumed

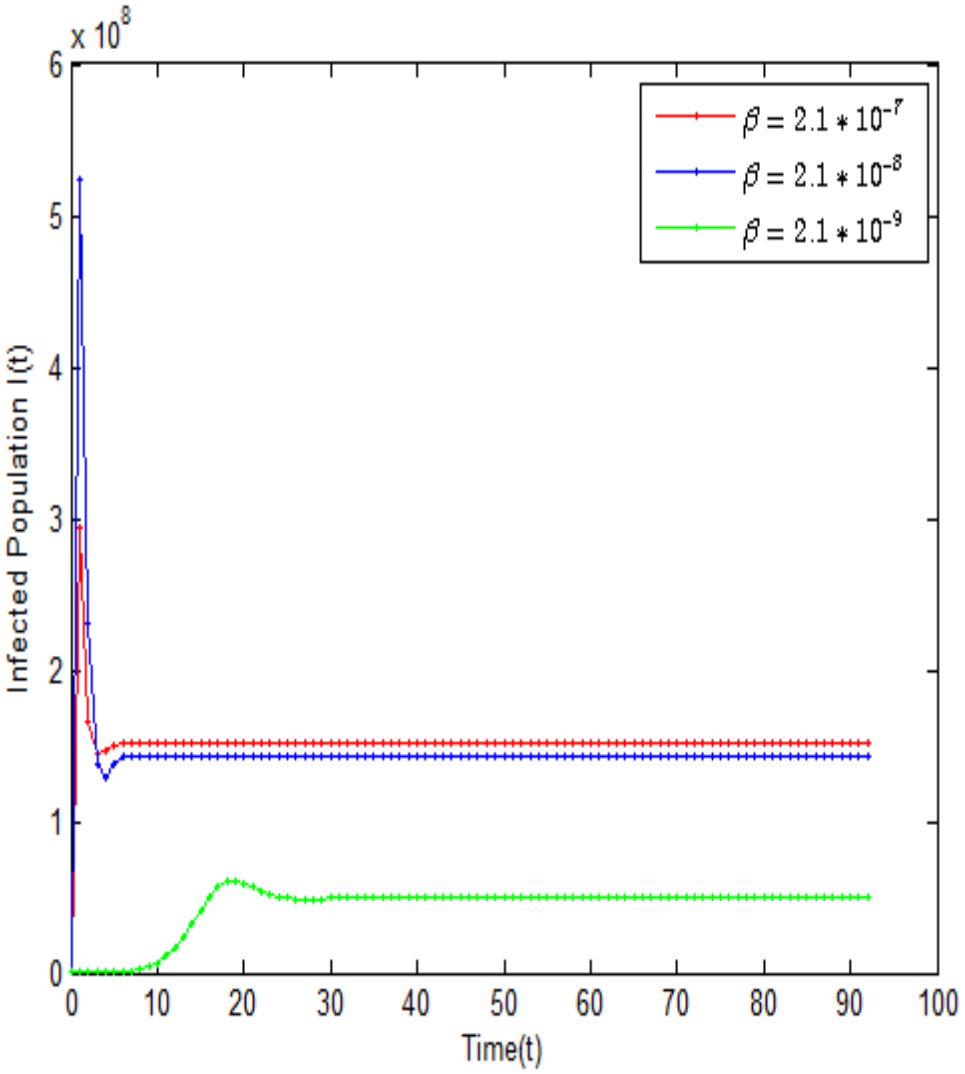

Figure 2: Variation in the infective population with transmission rate of infection.

ISSN PRINT 2319 1775 Online 2320 7876
 UGC CARE Listed (Group -I) Journal Volume 12, Iss 01, 2023

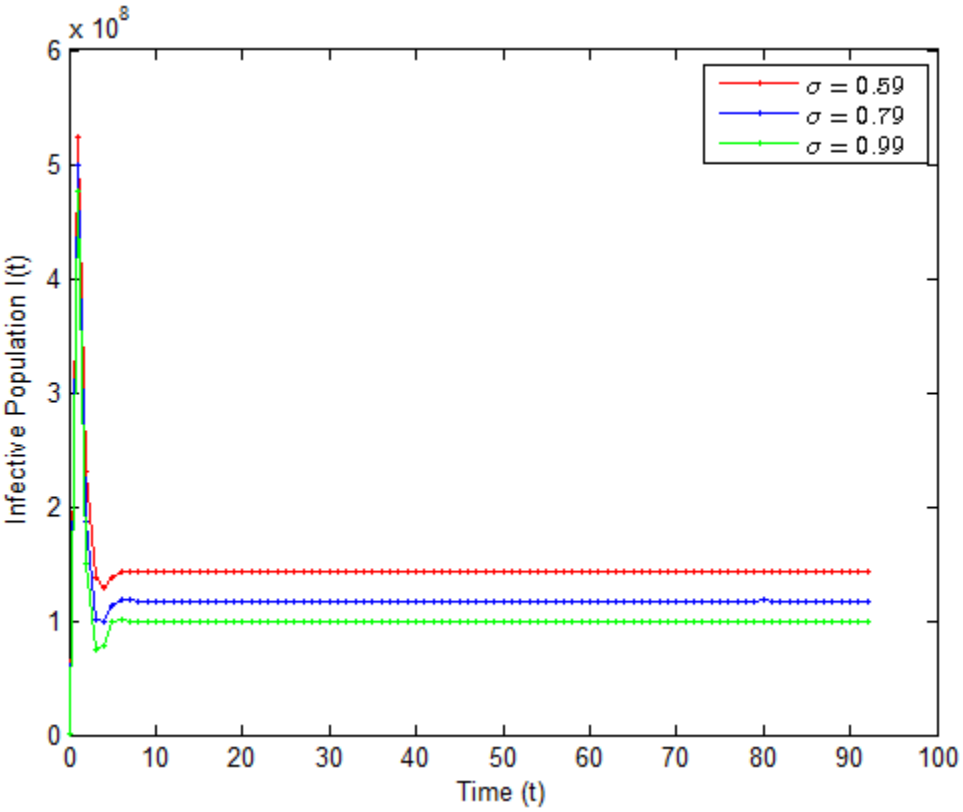

Figure 3: Outcome of transmission rate 'σ' with respect to infective population.

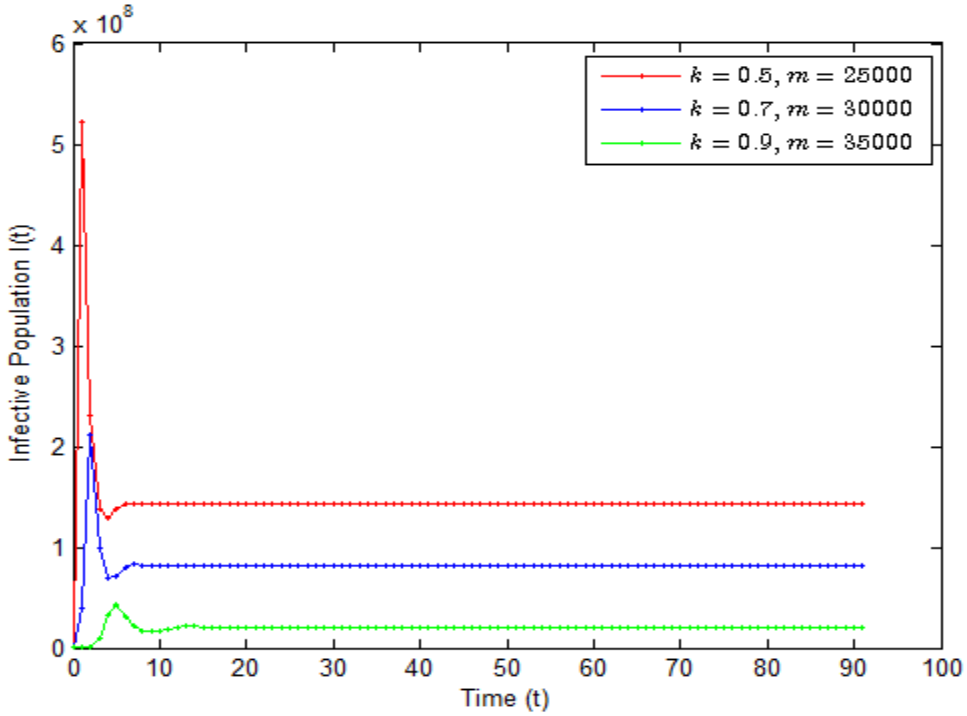

Figure 4: Outcome of contact tracing 'k' with respect to infective population.

ISSN PRINT 2319 1775 Online 2320 7876
 UGC CARE Listed (Group -I) Journal Volume 12, Iss 01, 2023

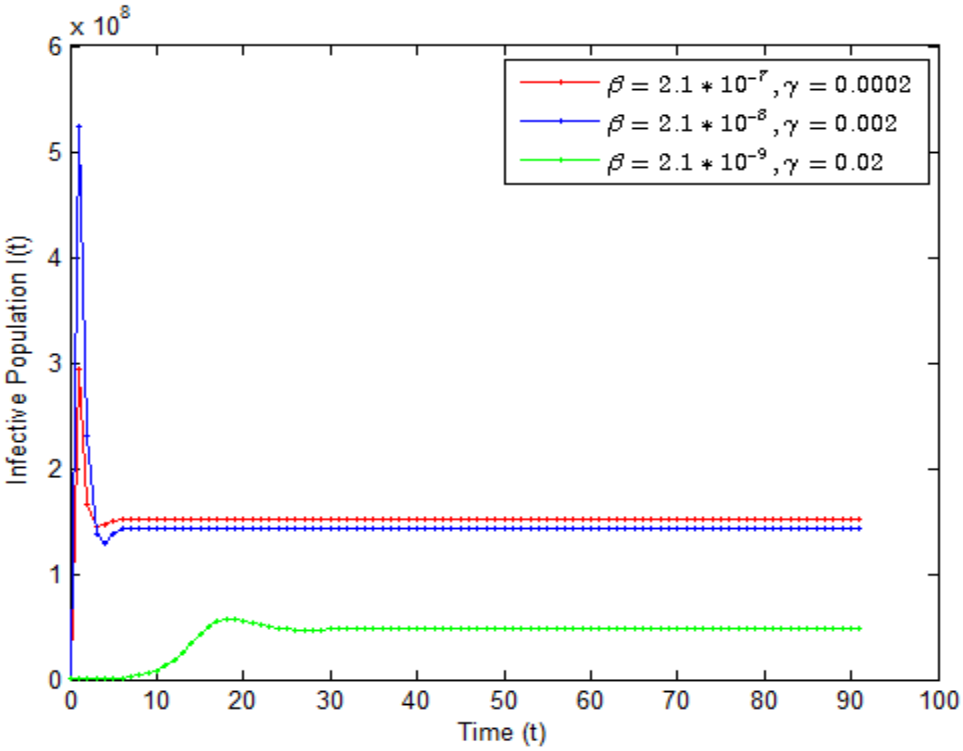

Figure 5: Effect of vaccination rate 'γ' with decrease in transmission rate of infection ' β ' on infective population.

VII. RESULT AND DISCUSSION

In this paper, we represented a non-linear mathematical model to learn about the influence of lockdown and vaccination on the dispense of COVID-19 which is based on different important precautions accepted by the Indian government to stop COVID-19 from spreading. The study of the above model is splintered into two parts; the first one is a complete lockdown state and the second one is a partial lockdown with a vaccination state. In both cases, we performed a qualitative analysis of the model and we calculate the basic reproduction ratio with the help of the next-generation matrix approach. We found that equilibrium is disease-free if the basic reproduction number is smaller than one also It is steady both locally and worldwide. Now pandemic equilibrium exists if the basic reproduction number is greater than one. The condition of persistence of the model and local stability of pandemic equilibrium in case of partial lockdown is determined by the vaccination state. Furthermore, we performed a numerical simulation to justify the analytical findings and draw the graphs. We found that the infected population increases when the transmission rate of infection rises and also observed that with the decrease in transmission rate infected population decreases, which means to control the disease we will have to reduce the rate of transmission. Thus, it is necessary to impose a strict lockdown to reduce the infection to zero. Moreover, to reduce the basic reproduction number below one we have identified some significant parameters. The vaccination rate of susceptible people who are neither infected nor quarantined and the transmission rate of infection is a very important factors to reduce the basic reproduction number to less than one. As we increase the vaccination rate basic reproduction number decreases very significantly below one. Thus, to control this disease vaccination is an essential measure accepted by the government. Thus, our study in this paper shows that for eliminating the disease to zero complete lockdowns are mandatory along with vaccinating the susceptible population that is neither infected nor quarantined controlling the immigration of

population density in the system is crucial for disease control. We can not impose a complete lockdown in a country where population density is very high (E.g. India) for a long time because it affects vaccination and may create financial issues. Thus lockdown is not the ultimate solution. Therefore, the government should prioritize additional preventative measures such as controlling the immigration of the infected population in the country, increasing the contact tracing rate quarantining the infected, and increasing the rate of vaccination. By doing the above we can control COVID-19.

REFERENCES

[1]. Aparicio, J. P., & Hernandez, J. C.(2006*). Preventive treatment of tuberculosis through contact tracing*. Contemporary Mathematics, 410, 17-20.

[2]. Archana Singh Bhadauria, Rachana Pathak & Manisha Chaudhary. *A SIQ mathematical model on covid-19 investigating the lockdown effect*. Infectious Disease Modelling 6(2021), 244-257.

[3]. Arriola, L. M., & Hyman, J. M. (2003). *Forward and adjoint sensitivity analysis: With applications in dynamical systems.* Technical report.

[4]. Barnett, E. D., & Walker, P. F. (2008). *Role of immigrants and migrants in emerging infectious diseases*. Medical Clinics of North America, 92(6), 1447-1458.

[5]. Bhola, J., Venkateswaran, V. R., & Koul, M. (2020). *Corona epidemic in Indian context: Predictive mathematical modelling.* Medrxiv, 200471753.

[6]. Binti, H. F. A., Lau, C., Nazri, H., Ligot, D. V., Lee, G., Tan, C. L., et al. (2020). *Corona Tracker: World-wide COVID-19 outbreak data analysis and prediction, [preprint]*. Bull World Health Organ, E-pub, 19, 255695.

[7]. Brauer, F., & Castillo, C. C. (2001). *Mathematical models in population biology and epidemiology*. Berlin: Springer.

[8]. Calistus N. Ngonghala, Enahoro A. Iboi & Abba B. Gumel. *Will an imperfect vaccine curtail the COVID-19 pandemic in the U.S.* Infectious Disease Modelling 5 (2020) 510-524.

[9]. Carr, J. (1981). *Application of center manifold Theory.* Newyork: Springer Verlag.

[10]. Chavez, C. C., Feng, Z., & Huang, W. (2002). *On the computation of ro and its role on. Mathematical approaches for emerging and reemerging infectious diseases: An introduction,* 1, 229.

[11]. Chen, S. C., Chang, C. F., & Liao, C. M. (2006). *Predictive models of control strategies involved in containing indoor airborne infections*. Indoor Air, 16(6), 469-481.

[12]. Cowling, B. J., Park, M., Fang, V. J., Wu, P., Leung, G. M., & Wu, J. T. (2015). Preliminary *epidemiologic assessment of mers-cov outbreak in South Korea Maye-June 2015*. Euro Surveillance, 20(25), 21163.

[13]. Day, T., Park, A., Madras, N., Gumel, A., & Wu, J. (2006*). When is quarantine a useful control strategy for emerging infectious diseases.* American Journal of Epidemiology, 163(5), 479-485.

[14]. Driessche, P. V. D., & Watmough, J. (2002). *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission.* Mathematical Biosciences, 180, 2948.

[15]. Eunha, S. (2006). *A note on epidemic models with infective immigrants and vaccination*. Mathematical Biosciences and Engineering, 3(3), 557.

[16]. Fraser, C., Riley, S., Anderson, R. M., & Ferguson, N. M. (2004). *Factors that make an infectious disease outbreak controllable*. Proceedings of the National Academy of Sciences, 101(16), 6146-6151.

[17]. Jia, L., et al. (2020). *Prediction and analysis of coronavirus disease*. arXiv preprint arXiv:2003.05447.

[18]. Koonprasert, S., & Chananngam, N. (2017). *Global stability and sensitivity analysis of SEIQR worm virus propagation model with quarantined state in mobile internet*. Global Journal of Pure and Applied Mathematics, 13(7), 3833-3850.

[19]. Kwok, K. O., Arthur, T., Vivian, W. I., Wei, W. H., Park, E., Kiong, Y., \& Steven, R. (2019). *Epidemic models of contact tracing: Systematic review of transmission studies of severe acute respiratory syndrome and Middle East respiratory syndrome*. Computational and Structural Biotechnology Journal, 17, 186-194.

[20]. Liang, K. (2020). *Mathematical model of infection kinetics and its analysis for COVID-19, SARS and MERS*. Infection, Genetics and Evolution, 82, 104306.

[21]. Li, Q., Guan, X., Peng, W., Zhou, X., Tong, Y., et al. (2020). *Early transmission dynamics in Wuhan, China, of novel coronavirus-infected pneumonia*. New England Journal of Medicine, 1-9.

[22]. Mandal, M., Jana, S., Nandi, S. K., Khatua, A., Adak, S., & Kar, T. K. (2020). *A model based study on the dynamics of COVID-19: Prediction and control, Chaos (p.109889)*. Solitons and Fractals.
[23]. Marimuthu, Y., Nagappa, B., Sharma, N., Basu, S., & Chopra, K. K. (2020). *COVID-19 and tuberculosis: A mathematical model based forecasting in Delhi, India*. Indian Journal of Tuberculosis, 67(2). https://doi.org/10.1016/j.ijtb.2020.05.006.
[24]. Mohsen, M., Wraith, M. R., et al. (2020). *Time series modelling to forecast the confirmed and recovered cases of COVID-19 (p. 101742).* Travel Medicine and Infectious Disease.
[25]. Ndairou, F., Area, I., Nieto, J. J., & Torres, D. F. M. (2020). *Mathematical modeling of COVID-19 transmission dynamics with a case study of Wuhan*. Chaos, Solitons & Fractals, 135, 109846.
[26]. Nyabadza, F., & Hove-Musekwa, S. D. (2010). *From heroin epidemics to methamphetamine epiepidemics: Modelling substance abuse in a South African province*. Mathematical Biosciences, 225, 132140.
[27]. Pal, D., Ghosh, D., Santra, P. K., & Mahapatra, G. S. (2020). *Mathematical analysis of a COVID-19 epidemic model by using data driven epidemiological parameters of dseases spread in India*. MedRxiv.
[28]. Sherif Eneye Shuaib, Pakwan Riyapan & Arthit Intarasit. *A Mathematical model of COVID-19 Pandemic: A Case Study of Bangkok, Thailand*. Hindawi Computational and Mathematical Methods in Medicine 2021.
[29]. Spiteri, G., Fielding, J., Diercke, M., et al. (2020). *First cases of coronavirus disease 2019 (COVID-19) in the WHO European region, 24 January to 21 February 2020*. Euro Surveillance, 25(9), 200017.
[30]. Vega, D. I. (2020). *Lockdown, one, two, none, or smart. Modeling containing covid-19 infection: A conceptual model*. The Science of the Total Environment, 730, 138917.
[31]. Wang, W. (2004). *Population dispersal and disease spread. Discrete and Continuous Dynamical Systems*. Series B, 4(3), 797-804.
[32]. Wang, W., & Zhao, X. Q. (2006). *An epidemic model with population dispersal and infection period*. SIAM Journal on Applied Mathematics, 66(4), 1454-1472.
[33]. Yang, J. Y., Zhang, F. Q., \& Wang, X. Y. (2009). *SIV epidemic models with age of infection*. International Journal of Bio-mathematics, 2(1), 61-67.