

**MATHEMATICAL MODELING
OF THE SPREAD OF CORONA VIRUS IN PRESENCE OF VACCINATION**

DILEEP SHARMA

Department of Mathematics, D.B.S. (P.G.) College, Dehradun-248001, India.

AGRAJ TRIPATHI

**Department of Mathematics,
Pranveer Singh Institute of Technology, Kanpur-209305, India.**

MANOJ KUMAR JADOUN*

Department of Mathematics, D.A.V. (P.G.) College, Dehradun-248001, India.

(Received On: 10-09-21; Revised & Accepted On: 07-10-21)

ABSTRACT

We propose a COVID-19 model with vaccination. We have divided the total population into three subclasses: the susceptible population, vaccinated population and infective population. A separate class $V(t)$ of cumulative density of corona virus in environmental reservoir is also taken into consider. Susceptibles are assumed to become COVID-19 infected via contacts with infectives and virus present in the reservoir. Model is analyzed using stability theory of differential equations. Both the infection-free and the endemic equilibria are found and their stability are investigated. Using Lyapunov functional approach, the sufficient conditions for global stability of the endemic equilibrium are obtained. It is shown that high rate of vaccination will help to reduce the infection in society. It is also found that the infective population can be decreased if susceptible do not come in direct contact with viral density deposited on surfaces/objects or airborne droplets accumulated in the environmental reservoir. Numerical simulations are also carried out to investigate the influence of key parameters on the spread of the disease, to support the analytical conclusion and illustrate possible behavioral scenario of the model.

Keywords: COVID-19, vaccination, susceptible, infectives, stability analysis.

1. INTRODUCTION

Corona viruses are family of viruses that circulate among animals and some times can also be found in humans. The disease was first identified December 2019 in Wuhan, China, and eventually invaded the world due to fast modern air transportation. The most general symptoms of COVID-19 are fever, fatigue, and dry cough. Some patients may have gripe and pains, nasal congestion, runny nose, or diarrhea. Some people become infected but don't develop any symptoms and don't feel illness. The COVID-19 pandemic is regard as the primus global threat worldwide because of thousands of confirmed infections, accompanied by thousands deaths over the world. According to the World Health Organization (WHO) situation report, worldwide, more than 20.4 Cr people were infected and about 43.1 Lack were expired due to COVID-19 virus infection between December 2019 and 12 August 2021 [28]. The extreme spread of the disease and lack of approved medicines made the disease a challenging problem for public health. Though some vaccine has been developed, it may not be equally effective on all strains of mutating corona virus, it is anticipated that the number of COVID-19 infections may still increase.

Corresponding Author: Manoj Kumar Jadoun*

Department of Mathematics, D.A.V. (P.G.) College, Dehradun-248001, India.

Mathematical models play an important role to understand the transmission dynamics of the epidemic and for short and long term prediction of epidemic incidence. Since the first case of COVID-19, various mathematicians around the world develop different mathematical models to understand the transmission dynamics of the virus, estimated the basic reproductive number and investigated effects of different intervention strategies. Several mathematical models have already been formulated for population dynamics of COVID-19 [2-9, 11-14, 20-27]. In Particular Okuonghae and Omame [5] studied the effect of control measure namely face mask and social distancing on the dynamics of the disease in detail. In Chu *et al.* [7], the authors have done a met analysis on the effectiveness of physical distancing, face mask, and eye protection in minimizing the human-to human transmission of the disease. In the paper by Ivorra *et al.* [2], a new mathematical model θ -SEIHRD model based on Be-CoDis model in Ivorra *et al.* [1] is proposed. This model considers existence of infectious undetected cases, control measures like sanitary conditions, isolation, quarantine, and tracing. It considers a novel method considering fraction of detected cases over actual total infected cases, which helps to understand the impact of COVID-19. Bandekar *et.al* [20], the authors performed an optimal control analysis with two control parameters to study the increase and decrease of the infected population with and without control. This study suggests that improved and rapid testing will help in identifying more infectives, thereby contributing in the decline of disease transmission rate. Sharma *et.al* [6] stress on follow the covid-19 protocol suggested by government like applying face mask or surgical masks, social distancing and proper sanitization at public places can help in controlling the spread. In Zhang *et al.* [26], it is clearly concluded that the spread of the disease could be controlled through effective contact tracing and by increasing the detection rates and quarantine of the individuals infectious to others. Khajanchi *et al.* [22] proposed a compartmental model with quarantine for the transmission dynamics of COVID-19 and calibrated the model with daily and cumulative cases for the four provinces of India. The authors have performed a detailed theoretical analysis in terms of the basic reproduction number and predicted the cumulative cases. Moreover, the study suggests that quarantine, unreported and reported individuals as well as intervention policies like social distancing, lockdown, and media effect can play an important role in controlling the transmission of COVID-19. Sarkar *et al.* [11] proposed a mathematical model that predicts the dynamics of COVID-19 in India along with its 17 provinces. The findings revealed the fact that the contact rate between susceptible and infected individuals could be reduced by a strict isolation imposed for susceptible individuals.

Our main contribution related with considering the effect of vaccination on the transmission dynamics of the Covid-19. This new class of vaccination, as compiled to any compartmental model, implies a number of analysis about absence of disease and endemic equilibrium point, which is also consider in this work. This paper is arranged as follows: we propose a model for COVID-19 in section 2. Section 3 describes basic reproduction number of model for COVID-19 and sensitivity analysis. Equilibrium analysis and stability analysis of equilibrium point discuss in section 4. Numerical simulations and Conclusions are describes in section 5 and in section 6 conclusion of the paper is presented respectively.

2. MATHEMATICAL MODEL

In the model, consider a population of size $N(t)$ at any time t and divided into three subclasses of susceptible population $X(t)$, vaccinated population $V(t)$ and infective population $I(t)$

- Susceptibles population $X(t)$, which denotes individuals who are susceptible to get the virus and become infectious.
- Vaccinated population $Y(t)$, which denote the population who has taken any vaccine of COVID-19
- Infective population $I(t)$, which denote the population who are infective of COVID-19 and can transmit the disease to other individuals

And a separate class $V(t)$ of cumulative density of corona virus in environmental reservoir is also taken into consider.

In the process of COVID-19 spreading, the spreading among these three states is governed by the following assumptions. Λ is the rate of constant immigration of susceptibles and d is the natural mortality rate of all subclasses of human population. β_1 , β_2 are the contact rate of susceptibles become infected via contact with infective population and with the density of virus in the environmental reservoir respectively. The constant rate ϕ is the rate by which susceptible will join the vaccinated class but some vaccinated person will become susceptible again with rate ν due to wear off vaccine. The constant rate η is the rate by which infective will recover and join the susceptible class. The constant rate γ is the rate of increase of corona virus density emitted from infected individuals. The rate of elimination of corona virus density due sanitization or other precautionary measures is σ . The constant rate α is the disease related death rate of infective population.

Based on the above considerations, the pandemic spreading leads to dynamic transitions among the human population, shown in Figure 1.

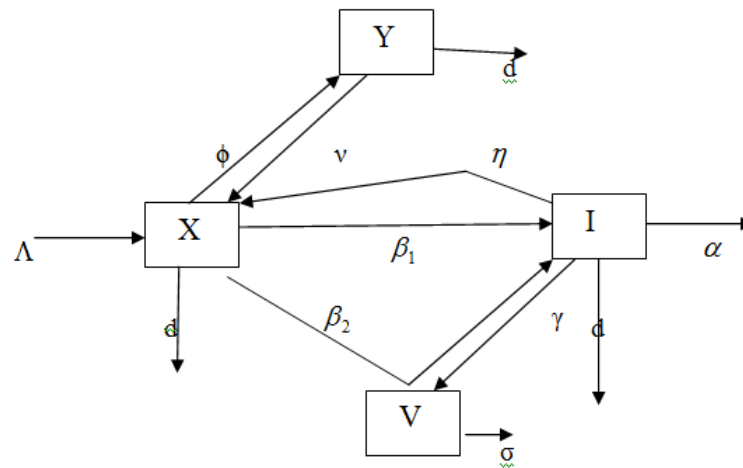


Figure-1: Schematic diagram of the model (2.1) - (2.4)

Taking into account the above assumptions and considerations, the dynamics of the COVID-19 outbreak is assumed to be governed by the following system of nonlinear ordinary differential equations,

$$\frac{dX(t)}{dt} = \Lambda - \beta_1 X(t)I(t) - \beta_2 V(t)X(t) - dX(t) - \phi X(t) + \eta I(t) + \nu Y(t) \tag{2.1}$$

$$\frac{dY(t)}{dt} = \phi X(t) - dY(t) - \nu Y(t) \tag{2.2}$$

$$\frac{dI(t)}{dt} = \beta_1 X(t)I(t) + \beta_2 V(t)X(t) - (\alpha + \eta + d)I(t) \tag{2.3}$$

$$\frac{dV(t)}{dt} = \gamma I(t) - \sigma V(t) \tag{2.4}$$

The total population $N(t)$ would be constant all the time in this model i.e.,

$$N(t) = X(t) + Y(t) + I(t)$$

We have the non negative initial conditions

$$X(0) = X_0 > 0, Y(0) = Y_0 > 0, I(0) = I_0 \geq 0, V(0) = V_0 \geq 0$$

2.1 Non-negativity of solutions

An important feature of any epidemiological model is to show that all the population variables are non-negative for all $t \geq 0$, which implies that any trajectory starting with positive initial condition will remain positive for $t \geq 0$ [8]. The following lemma describes this fact,

Lemma 2.1: If $X(0) \geq 0, Y(0) \geq 0, I(0) \geq 0$ and $V(0) \geq 0$, the solution of $X(t), Y(t), I(t)$ and $V(t)$ in the system (2.1) - (2.4) remain positive.

Proof: We shall prove this lemma using contradiction by assuming that the total population $N(t) \neq 0$ for all $t \geq 0$. We assume that there exists the time t_1, t_2, t_3 and t_4 respectively such that

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

$$\begin{aligned} \frac{dX(t_1)}{dt} < 0 \Rightarrow \left(\frac{dX(t_1)}{dt} \right)_{t=t_1} &= \Lambda - \beta_1 I(t_1)X(t_1) - \beta_2 V(t_1)X(t_1) - dX(t_1) - \phi X(t_1) + \eta I(t_1) + \nu Y(t_1) \\ &= \Lambda + \eta I(t_1) + \nu Y(t_1) \leq 0 \end{aligned}$$

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

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

$$\frac{dY(t_2)}{dt} < 0 \Rightarrow \left(\frac{dY(t_2)}{dt} \right)_{t=t_2} = \phi X(t_2) - (v + d)Y(t_2) \\ = \phi X(t_2) \leq 0$$

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

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

$$\frac{dI(t_3)}{dt} < 0 \Rightarrow \left(\frac{dI(t_3)}{dt} \right)_{t=t_3} = \beta_1 I(t_3) X(t_3) + \beta_2 V(t_3) X(t_3) - (\alpha + \eta + d)I(t_3) \\ = \beta_2 V(t_3) X(t_3) \leq 0$$

which is contradiction as $\beta_2 V(t_3) X(t_3) > 0$. Hence, it can be concluded that $I(t) \geq 0$ for $t \geq 0$.

Positivity of $V(t)$: Assume that $V(t_4) = 0, \frac{dV(t_4)}{dt} < 0, X(0) \geq 0, Y(0) \geq 0, I(0) \geq 0$ and $V(0) \geq 0, 0 \leq t \leq t_4,$

$$\frac{dV(t_4)}{dt} < 0 \Rightarrow \left(\frac{dV(t_4)}{dt} \right)_{t=t_4} = \gamma I(t_4) - \sigma V(t_4) \\ = \gamma I(t_4) \leq 0$$

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

3.1. COMPUTATION OF BASIC REPRODUCTION NUMBER

The basic reproduction number R_0 is defined as the effective number of secondary infections generated by a typical infected individual in an otherwise disease free population. It is very important in case of infectious disease. If $R_0 < 1$, then on average an infected individual produces less than one infected individual over the course of its infectious period and 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.

Here, we find the basic reproduction rate R_0 of the model (2.1)-(2.4), by using next generation matrix method [17, 18]. We first compute the new infectious matrix F and transfer matrix W [16], according to formula

$$[F - W] = \begin{bmatrix} \frac{\partial(dI/dt)}{\partial I} & \frac{\partial(dI/dt)}{\partial V} \\ \frac{\partial(dV/dt)}{\partial I} & \frac{\partial(dV/dt)}{\partial V} \end{bmatrix} \quad (3.1)$$

To calculate F and W , we only consider equations (2.3) and (2.4), 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 (disease free equilibrium is given in section 4.1) is,

$$F = \begin{bmatrix} \frac{\beta_1 \Lambda (v + d)}{d(\phi + v + d)} & \frac{\beta_2 \Lambda (v + d)}{d(\phi + v + d)} \\ 0 & 0 \end{bmatrix} \quad (3.2)$$

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

$$W = \begin{bmatrix} (\alpha + \eta + \mu) & 0 \\ -\gamma & \sigma \end{bmatrix} \quad (3.3)$$

W^{-1} is given by $W^{-1} = \frac{1}{(\alpha + \eta + \mu)\sigma} \begin{bmatrix} \sigma & 0 \\ \gamma & (\alpha + \eta + \mu) \end{bmatrix}$

FW^{-1} is the next generation matrix of the system (2.5)-(2.8). It follows that the spectral radius of matrix FW^{-1} is

$$FW^{-1} = \frac{\Lambda (v + d)}{d\sigma(\alpha + \eta + d)(\phi + v + d)} \begin{bmatrix} \beta_1 \sigma + \beta_2 \gamma & \beta_2 (\alpha + \eta + \mu) \\ 0 & 0 \end{bmatrix} \\ \rho(FW^{-1}) = \frac{\Lambda (v + d)(\beta_1 \sigma + \beta_2 \gamma)}{d\sigma(\alpha + \eta + d)(\phi + v + d)} \quad (3.4)$$

$$R_0 = \frac{\Lambda(v+d)(\beta_1\sigma + \beta_2\gamma)}{d\sigma(\alpha + \eta + d)(\phi + v + d)}$$

According to van den Driessche and watmough [17, 18], the basic reproduction number of the system (2.1)-(2.4) is

$$R_0 = \frac{\Lambda(v+d)(\beta_1\sigma + \beta_2\gamma)}{d\sigma(\alpha + \eta + d)(\phi + v + d)} \tag{3.5}$$

The increasing the value of denominator will reduce the reproduction number. ϕ , α , η and σ are the parameters which are only in the denominator. But we can not increase α (disease related death rate). Hence reproduction number can be reduce, by increasing ϕ , the vaccination rate of susceptible as the vaccination rate of susceptible will increase, reproduction rate will decrease. Hence, high rate of vaccination will help to reduce the infection in society. Reproduction rate will also decrease as the recovery rate of infectives will increase. It shows that if more and more infectives will recover soon then the reproduction rate will decrease. Reproduction number due to virus can be reduce by increasing the value of σ , the rate of elimination of virus density, by sanitization, social distancing and other precautionary measures.

3.2. SENSITIVITY ANALYSIS

Sensitivity analysis is crucial in determining the importance of various parameters in disease transmission. In Rodrigues *et al.* [10], a detailed explanation on sensitivity analysis for case of dengue is presented. It helps in determining the parameters with high and low impact on the reproduction number, thereby helping in focusing on various intervention strategies. 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 differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivative. From Chitnis *et al.* [15], the normalized forward sensitivity index of R_0 , that depends differentially on a parameter, is defined by

$$Y_{\lambda}^{R_0} = \frac{\partial R_0}{\partial \lambda} \times \frac{\lambda}{R_0}$$

The parameter values displayed in below table 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 infectious disease to four places of decimal in relation to the effective reproduction number R_0 , using equation (3.5), the result of which is presented in table 1 below

$$R_0 = \frac{\Lambda(v+d)(\beta_1\sigma + \beta_2\gamma)}{d\sigma(\alpha + \eta + d)(\phi + v + d)}$$

$$\frac{\partial R_0}{\partial \beta_1} \frac{\beta_1}{R_0} = \frac{\beta_1\sigma}{\beta_1\sigma + \beta_2\gamma} > 0, \quad \frac{\partial R_0}{\partial \beta_2} \frac{\beta_2}{R_0} = \frac{\gamma\beta_2}{\beta_1\sigma + \beta_2\gamma} > 0, \quad \frac{\partial R_0}{\partial \gamma} \frac{\gamma}{R_0} = \frac{\gamma\beta_2}{\beta_1\sigma + \beta_2\gamma} > 0$$

$$\frac{\partial R_0}{\partial \sigma} \frac{\sigma}{R_0} = -\frac{\gamma\beta_2}{(\beta_1\sigma + \beta_2\gamma)} < 0, \quad \frac{\partial R_0}{\partial \eta} \frac{\eta}{R_0} = -\frac{\eta}{(\alpha + \eta + d)} < 0, \quad \frac{\partial R_0}{\partial \phi} \frac{\phi}{R_0} = -\frac{\phi}{(\phi + v + d)} < 0$$

Sensitivity indices have given below for the parameter taken in the paper.

Table-1: Sensitivity index and indices Table

Parameter Symbol	Sensitivity indices
β_1	0.9091
β_2	0.0909
γ	0.0909
ϕ	0.8139
σ	-0.0909
η	0.3311

From table we can see that the positive indices i.e. β_1 , β_2 and γ show that they have great impact on expanding the disease in the population if their value increases R_0 increases, it means the number of secondary infections increases in the population. Further the parameter ϕ , η and σ for which the sensitivity indices is negative, shows that if ϕ , η and σ will increase the basic reproduction number will decrease, which minimize the disease in the population.

4. EQUILIBRIA AND STABILITY ANALYSIS OF THE MODEL

4.1 Equilibria of the model

In this subsection, we show the feasibility of all equilibria by setting the rate of change with respect to time t of all dynamical variables to zero. The model (2.1)-(2.4) has two non-negative feasible equilibria namely,

(i) $E_0 \left(\frac{\Lambda(v+d)}{d(\phi+v+d)}, \frac{\Lambda\phi}{d(\phi+v+d)}, 0, \right)$ the disease-free equilibrium, which exists without any condition. This equilibrium

implies that in the absence of any infection, the total population size (N) remains at its equilibrium value Λ/d .

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

$$X^* = \frac{\Lambda(v+d)}{d(\phi+v+d)R_0}, \quad Y^* = \frac{\phi\Lambda}{d(\phi+v+d)R_0}, \quad I^* = \frac{\Lambda(R_0-1)}{R_0(\alpha+d)}, \quad V^* = \frac{\gamma\Lambda(R_0-1)}{\sigma R_0(\alpha+d)}$$

From the above expression we can see that the endemic equilibrium will exist only if $R_0 > 1$ and if $R_0 < 1$ the disease will not persist in the population.

4.2 Local stability of the equilibria

To determine the local stability of E_0 , the following variational matrix of the system (2.1) – (2.4) is computed about E_0 as,

$$J(E_0) = \begin{bmatrix} -(\phi+d) & v & -\beta_1 X_0 + \eta & -\beta_2 X_0 \\ \phi & -(v+d) & -\xi & 0 \\ 0 & 0 & -\left(\frac{\beta_1 \Lambda(v+d)}{d(\phi+v+d)} + (\alpha + \eta + d) \right) & \frac{\beta_2 \Lambda(v+d)}{d(\phi+v+d)} \\ 0 & 0 & \gamma & -\sigma \end{bmatrix}$$

The two roots of the characteristic equation is determined by the equation

$$f(\lambda) = \lambda^2 + (\phi + v + 2d)\lambda + d(\phi + v + d) = 0$$

As there is no sign change, so by the descarte's rule of signs both roots of the equation are negative.

Another two roots of matrix is determined by the equation

$$f(\lambda) = \lambda^2 + \left(-\frac{\beta_1 \Lambda(\mu+v)}{\mu(\xi + \mu + v)} + (\alpha + \eta + \mu) + \sigma \right) \lambda - \frac{(\beta_1 \sigma + \beta_2 \gamma) \Lambda(\mu+v)}{\mu(\xi + \mu + v)} + (\alpha + \eta + \mu) \sigma = 0 \quad (4.1)$$

$$-\frac{(\beta_1 \sigma + \beta_2 \gamma) \Lambda(\mu+v)}{\mu(\xi + \mu + v)} + (\alpha + \eta + \mu) \sigma > 0, \quad (\alpha + \eta + \mu) \sigma > \frac{(\beta_1 \sigma + \beta_2 \gamma) \Lambda(\mu+v)}{\mu(\xi + \mu + v)}$$

$$1 > \frac{(\beta_1 \sigma + \beta_2 \gamma) \Lambda(\mu+v)}{\mu(\alpha + \eta + \mu) \sigma (\xi + \mu + v)}$$

$$1 > R_0$$

We can see if $R_0 > 1$, then $J(E_0)$ has at least one eigen value with positive real part. Hence, disease free equilibrium E_0 of the (2.1)-(2.4) is locally asymptotically stable if $R_0 < 1$. Therefore, the disease dies out i.e. infection does not persist in the population and under this condition the equilibrium E^* does not exist. It is unstable for $R_0 > 1$ and then E^* exists and the disease always persists in the population.

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

$$J(E^*) = \begin{bmatrix} -m_1 & v & -m_2 & -n_3 \\ \phi & -(v+d) & 0 & 0 \\ n_1 & 0 & -n_2 & n_3 \\ 0 & 0 & \gamma & -\sigma \end{bmatrix}$$

Where $m_1 = n_1 + \phi + d$, $m_2 = \beta_1 X^* - \eta$, $n_1 = \beta_1 I^* + \beta_2 V^*$, $n_2 = \frac{n_3 \gamma}{\sigma}$, $n_3 = \beta_2 X^*$

The roots of matrix is determined by the characteristic equation is given by

$$\lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4 = 0$$

Where,

$$a_1 = m_1 + n_2 + \sigma + v + d, \quad a_2 = m_1(v+d+n_2+\sigma) + (v+d)(n_2+\sigma) + n_2\sigma + m_2n_1 - v\phi - n_3\gamma$$

$$a_3 = m_1(v+d)(n_2+\sigma) + m_1n_2\sigma + n_2(v+d)\sigma + m_2n_1(\sigma+v+d) + m_3n_1\gamma - n_3\gamma(m_1+v+d) - v\phi(n_2+\sigma)$$

$$a_4 = m_1(v+d)n_2\sigma + v\phi n_3\gamma + m_2n_1(v+d)\sigma + m_3n_1\gamma(v+d) - n_3\gamma m_1(v+d) - v\phi n_2\sigma$$

The equilibrium E^* is locally asymptotically stable if and only if the following inequalities hold:

$$a_1 a_2 - a_3 > 0, a_1 a_2 a_3 - a_1^2 a_4 - a_3^2 > 0$$

4.3. Global Stability of the endemic Equilibrium

To show the global stability [6, 19] 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, stated below

Lemma 1: The biologically feasible region

$$\Omega = \left\{ (X, Y, I, V) \in R_+^4; 0 < X(t) + Y(t) + I(t) \leq \frac{\Lambda}{d}; 0 \leq V(t) \leq \frac{\gamma\Lambda}{\sigma d} \right\} \quad (4.2)$$

is a positive invariant a region and attracting region for the disease transmission model given by the system (2.1)-(2.4) with initial conditions equation.

Proof: Summing up the first three equations in system (2.1) to (2.3) and denoting

$$N(t) = X(t) + Y(t) + I(t)$$

We get

$$\begin{aligned} \frac{dN(t)}{dt} &= \Lambda - dN(t) - \alpha I(t) \\ \frac{dN(t)}{dt} &\leq \Lambda - dN(t) \end{aligned}$$

Thus, $\lim_{t \rightarrow \infty} \text{Sup}N(t) \leq \bar{N}$, here, $\bar{N} = \frac{\Lambda}{d}$

From equation (2.4),

$$\begin{aligned} \frac{dV(t)}{dt} &= \gamma I(t) - \sigma V(t) \\ \frac{dV(t)}{dt} &\leq \gamma \frac{\Lambda}{d} - \sigma V(t) \text{ since } I(t) \leq N(t) \leq \bar{N} \end{aligned}$$

Thus, $\lim_{t \rightarrow \infty} \text{Sup}V(t) \leq \bar{V}$, here, $\bar{V} = \frac{\gamma\Lambda}{d\sigma}$

Theorem 1: If the endemic equilibrium E^* exists, then it is globally asymptotically stable provided the following sufficient conditions are satisfied in Ω

$$(\nu + \phi)^2 \leq \frac{2}{3}(\phi + d)(\nu + d) \quad (4.3)$$

$$\left(\eta - \beta_1 X^* + k_2 \beta_1 I + k_2 \beta_2 V \right)^2 \leq \frac{4}{9}(\phi + d)(\alpha + \eta + d - \beta_1 X^*) \quad (4.4)$$

$$\left(k_2 \beta_2 X^* + k_3 \gamma \right)^2 \leq \frac{2}{3} k_3 \sigma (\alpha + \eta + d - \beta_1 X^*) \quad (4.5)$$

Where, k_2 is a constant and k_3 is given by $k_3 \geq \frac{3(\beta_2 X^*)^2}{2\sigma(\phi + d)}$

5. NUMERICAL SIMULATION AND DISCUSSION

To see the dynamical behavior of the model system, the system (2.1)-(2.4) is integrated numerically by fourth order Runge-Kutta method using the following set of parameters values:

$$\begin{aligned} \Lambda = 2000, \quad d = 0.02, \quad \alpha = 2, \quad \beta_1 = 0.015, \quad \beta_2 = 0.01 \quad \phi = 0.35, \quad \nu = 0.06 \quad \eta = 1, \\ \gamma = 0.03, \quad \sigma = 0.2, \end{aligned}$$

with initial values $X(0) = 100$, $Y(0) = 900$, $I(0) = 500$, and $V(0) = 100$.

The results of numerical simulation are displayed graphically in figs. (2-9). In fig. 2, the variation of infective population $I(t)$ with time t is shown for different values of β_1 , the rate of transmission of susceptibles to infective class through direct contact with infectives present in the population. It is seen that infective population increases with increase in the value of β_1 . In figs. 3 the variation of infective population $I(t)$ with time t is shown for different values of β_2 , the rate of transmission of susceptibles to infective class through direct contact with virus viral density deposited on surfaces/objects or airborne droplets accumulated in the environmental reservoir. It is seen that infective

population increases with increase in the value of β_2 . Thus, it suggested that the people should follow the COVID-19 guideline like apply facemask, social distancing etc. so that they do not come in the contact of viral density present in the reservoir. In figs. 4 the variation of infective population $I(t)$ with time t is shown for different values of σ , is the rate of elimination of corona virus density due sanitization or other precautionary measures. The rate depletion of transmission of susceptibles to infective class through direct contact with virus viral density deposited on surfaces/objects or airborne droplets accumulated in the environmental reservoir. It is seen that infective population decreases with increase in the value of σ . It shows that the proper sanitization at public place is helpful to control the spread of COVID-19. In figs. 5, the variation of infective population $I(t)$ is shown with time t for different values of η , the rate of recovery of infective individuals. It is noted that with increase in the recovery rate of infected individuals, their population decreases. Fig (6-7) shows the variation of vaccinated population, infective population with time t for different value of ϕ , the rate of vaccination of the susceptible. It is found that as the value of ϕ increase infective population decrease and vaccinated population increase. This indicates that if rate of vaccination of susceptible increases, the vaccinated population increases in turn infective population decreases. Fig (8-9) depicts the variation of vaccinated population, infective population with time t for different value of ν , the wear off rate of vaccine. It is found that as the value of ν increase vaccinated population decrease in turn infective population increase.

From the above discussion, it follows that if more and more susceptible individuals either vaccinate themselves or follow the COVID-19 guidelines do not come in contact with infectives and the viral density deposited on surfaces/objects or airborne droplets accumulated in the environmental reservoir, the spread of the disease can be controlled.

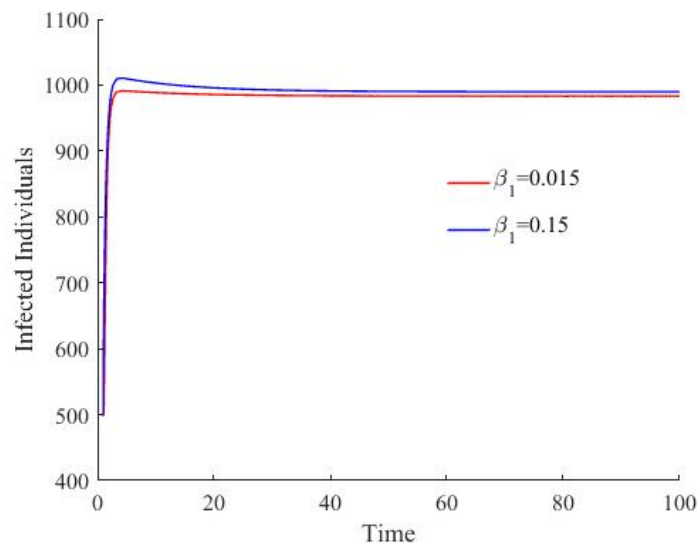


Figure-2: Variation of infected individuals for different values of β_1

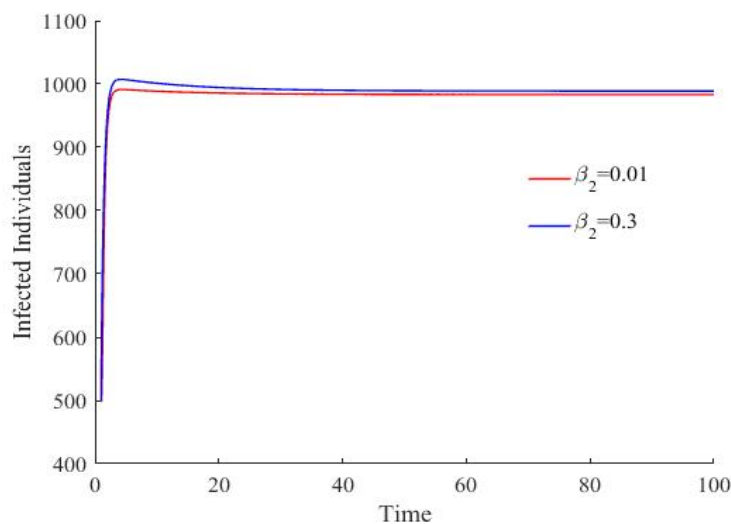


Figure-3: Variation of infected individuals for different values of β_2

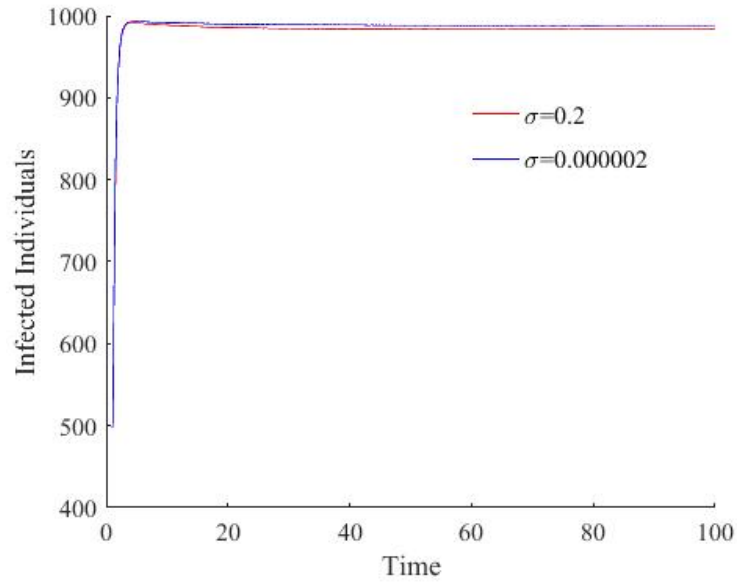


Figure-4: Variation of infected individuals for different values of σ

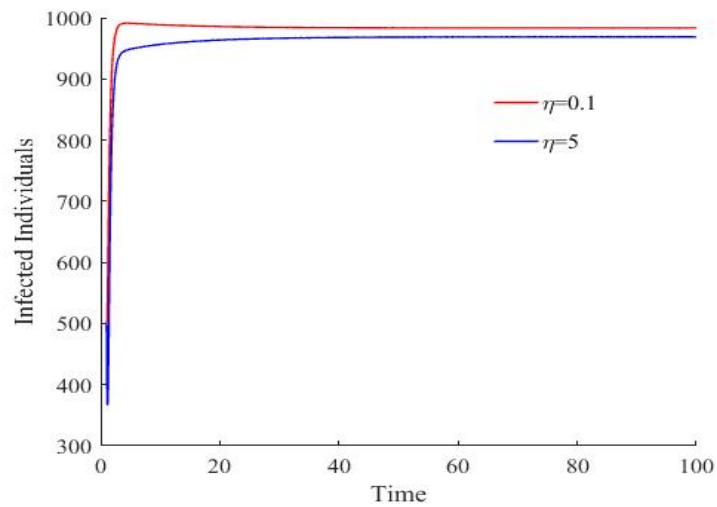


Figure-5: Variation of infected individuals for different values of η

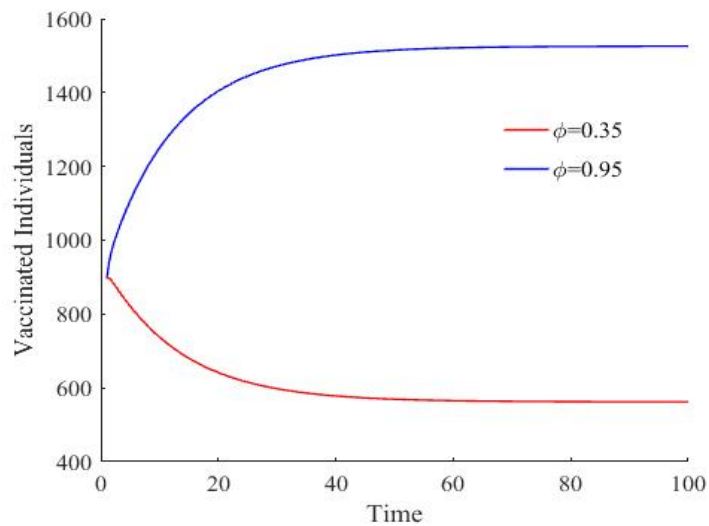


Figure-6: Variation of vaccinated individuals for different values of ϕ

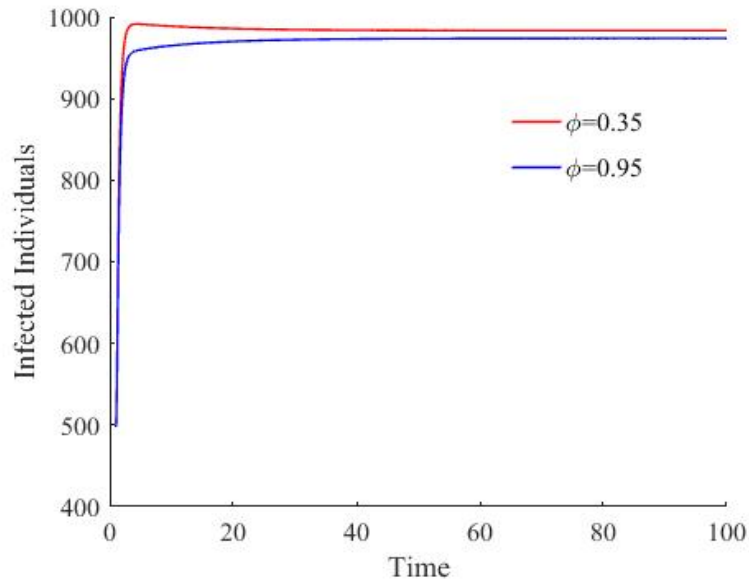


Figure-7: Variation of infected individuals for different values of ϕ

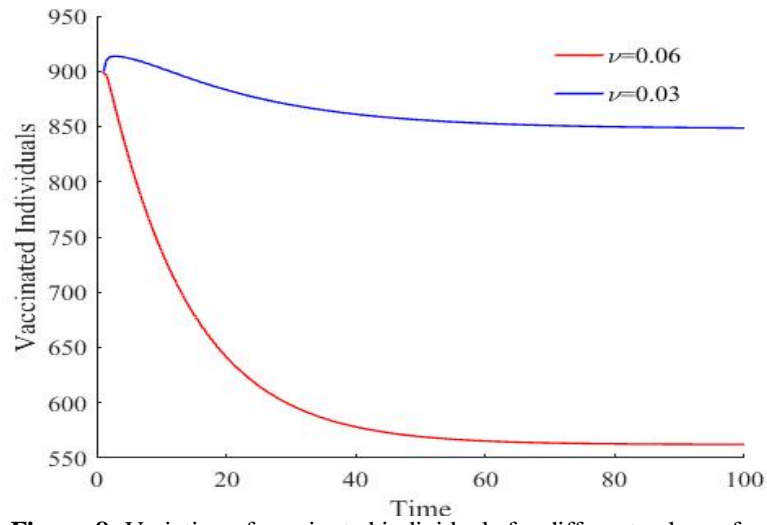


Figure-8: Variation of vaccinated individuals for different values of ν

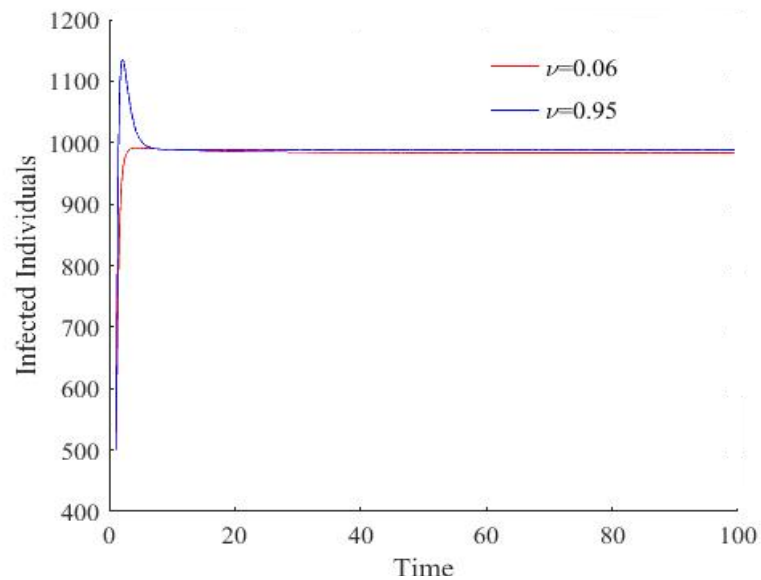


Figure-9: Variation of infected individuals for different values of ν

6. CONCLUSION

In this paper, a set of non linear differential equations are applied to describe the transmission dynamics of COVID-19 in presence of vaccination on the spread of corona virus in a population with variable size structure. The total human population is divided into three subclasses: the susceptible, the vaccinated persons and the infected persons a separate class of virus density present in the reservoir is taken into consider. The model exhibits two equilibria namely, the disease-free and the endemic equilibrium. The local and global stability results of these equilibria have been established. The analysis of the proposed model has been done using stability theory of differential equations and computer simulations. The reproduction number R_0 is found which shows that reducing the effective contact rates and improving the vaccination rate are very important to contain the spread of COVID-19. Sensitivity analysis of R_0 in terms of model parameters also demonstrates that the vaccination and the improvement of the recovery rate are two critical factors in fighting against COVID-19.

Finally, from numerical simulation, the results indicate once again that improving the vaccination rate are crucial to contain the spread of COVID-19, and early control measures can also effectively prevent a larger outbreak of COVID-19. Although improving the recovery rate can be realized by providing efficient confirmatory test kits, doctors and other medical resource and reducing the effective contact rate can be realized by following 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. 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.

REFERENCES

1. B Ivorra, D Ngom, Ramos ÁM Be-CoDiS: a mathematical model to predict the risk of human diseases spread between countries—validation and application to the 2014–2015 ebola virus disease epidemic. *Bull Math Biol* 77(9) 2015, 1668–1704.
2. B Ivorra, M Ferrández, M Vela-Pérez, Ramos A Mathematical modeling of the spread of the coronavirus disease 2019 (COVID-19) taking into account the undetected infections. The case of china. *Commun Nonlinear Sci Numer Simul* (2020) 88:105303.
3. C. Yang, J. Wang, A mathematical model for the novel coronavirus epidemic in Wuhan, China, *Math. Biosci. Engg.*17(2020), 2708–2724.
4. C.-N. Ngonghala, E. Iboi, S. Eikenberry, M. Scotch, C.-R. MacIntyre, M.-H. Bonds, *et al.*, Mathematical assessment of the impact of non-pharmaceutical intervention curtailing the 2019 novel Corona virus, *Math. Biosci.* (2020), 108364.
5. D Okuonghae, A Omame, Analysis of a mathematical model for COVID-19 population dynamics in Lagos, Nigeria. *Chaos Solitons Fractals* (2020) 139:110032. [https:// doi. org/ 10. 1016/j. chaos. 2020, 110032](https://doi.org/10.1016/j.chaos.2020.110032).
6. D Sharma , A Tripathi and MK Jadoun (2021), Modeling the Spread of Corona Virus with Lockdown and Quarantine Effect, *International Journal of New Innovations in Engineering and Technology*, 17 (2) 2021, 78-96.
7. DK Chu, EA Akl, S Duda, K Solo, S Yaacoub, HJ Schünemann, DK Chu, EA Akl, A, Bognanni A El-harakeh, T Lotfi, M Loeb, A Hajizadeh, A Bak, A Izcovich, CA Cuello-Garcia, C Chen, DJ Harris, E Borowiack, F Chamseddine, F Schünemann, GP Morgano, GEUM Schünemann, G Chen, H Zhao, I Neumann, J Chan, J Khabsa, L Hneiny, L Harrison, M Smith, N Rizk, PG Rossi, P AbiHanna, R El-khoury, R Stalteri, T Baldeh, T Piggott, Y Zhang, Z Saad, A Khamis, M Reinap, S Duda, K Solo, S Yaacoub, HJ Schünemann (2020) Physical distancing, face masks, and eye protection to prevent person-to-person transmission of SARS-CoV-2 and COVID-19: a systematic review and meta-analysis. *The Lancet* 395(10242) (2020)1973–1987.
8. E. D. Gurmu, G. B. Batu and M. S. Wameko, Mathematical model of novel COVID 19 and its transmission dynamics. *Int. J. Math. Mod. Comp.* 10(02) 2020, 141-159.
9. F. Saldana, H. F. Arguedas, J. A. Camacho-Gutierrez, I. Barradas, Modeling the transmission dynamics and the impact of the control interventions for the COVID-19 epidemic outbreak, *Math. Biosci. Engg.* 17(2020), 4165–4183.
10. HS Rodrigues, MTT Monteiro, DFM Torres, Sensitivity analysis in a dengue epidemiological model. *Conf Papers Math* 2013:1–7. [https:// doi. org/ 10. 1155/ 2013/ 721406](https://doi.org/10.1155/2013/721406).
11. K. Sarkar, S. Khajanchi, J.-J. Nieto, Modeling and forecasting the COVID-19 pandemic in India, *Chaos Soliton Fract.* 139(2020), 110049.
12. Legesse Lemecha Obsu and Shiferaw Feyissa Balcha; Optimal control strategies for the transmission risk of COVID-19, *J. of Bio. Dyn.* 14(1) 2020, 590–607.

13. Lin Hu, Lin-Fei Nie, Dynamic modeling and analysis of COVID-19 in different transmission process and control strategies. *Math. Meth. Appl. Sci.* 2020, 1-14.
14. Liuyong pang, Sanhong Liu, Xinan Zhang, Tianhai Tian and Zhong Zhao. Transmission Dynamics and control strategies of COVID-19 in Wuhan, China. *J. Bio. Sys.* 28(3) 2020, 1-18.
15. N Chitnis, JM Hyman, JM Cushing, Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. *Bull Math Biol* 70(5) (2008):1272–1296.
16. O. Diekmann, J. A. P. Heesterbeek, M. G. Roberts, The construction of next-generation matrices for compartmental epidemic models, *J. R. Soc. Interface*,7(2010), 873–885.
17. P. van den Driessche, J. Watmough, further notes on the basic reproduction number. In: Brauer F van den Driessche P, Wu J, eds, *Mathematical Epidemiology, Lecture Notes in Mathematics Vol 1945*. Berlin: Springer; 2008, 159-178.
18. P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.* 180 (2002), 29-48.
19. R. Naresh, D. Sharma, A. Tripathi, Modelling the effect of tuberculosis on the spread of HIV infection in a population with density-dependent birth and death rate. *Math. Com. Mod.* 50(2009), 1154–1166.
20. S R Bandekar, M Ghosh, Mathematical modeling of COVID-19 in India and its states with optimal control, *Mode Earth Sys and Envir.* (2021) <https://doi.org/10.1007/s40808-021-01202-8>
21. S. Bugalia, P. Bajiya, J P Tripathi, M-T Li and G-Q Sun, Mathematical modeling of COVID-19 transmission: the role of intervention strategies and lockdown. *Math. Bio. and Engg.* 17(5)2020, 5961-5986.
22. S. Khajanchi, K. Sarkar, Forecasting the daily and cumulative number of cases for the COVID-19 pandemic in India, *Chaos.* 30(2020), 071101.
23. S.-M. Garba, J.-M. Lubuma, B. Tsanou, Modeling the transmission dynamics of the COVID-19 Pandemic in South Africa, *Math. Biosci.* (2020), 108441.
24. T.-M. Chen , J. R., Q.-P. Wang, Z.-Y. Zhao, J.-A. Cui and L. Yin, A mathematical model for simulating the phase-based transmissibility of a novel corona virus, *Infectious Diseases of Poverty*, 9 (2020), doi: 10.1186/s40249-020-00640-3.
25. T. Sardar, S.-S. Nadim, S. Rana, J. Chattopadhyay, Assessment of Lockdown Effect in Some States and Overall India: A Predictive Mathematical Study on COVID-19 Outbreak. *Chaos Soliton Fract.* 139(2020), 110078.
26. X-S Zhang, E Vynnycky, A Charlett, DD Angelis, Z Chen, W Liu (2021) Transmission dynamics and control measures of COVID19 outbreak in China: a modelling study. *Sci Rep.* <https://doi.org/10.1038/s41598-021-81985-z>
27. Y. Li, B. Wang, R. Peng, C. Zhou, Y. Zhan, Z. Liu et al., Mathematical modeling and epidemic prediction of COVID-19 and its significance to epidemic prevention and control measures, *Annals of Infectious Disease and Epidemiology*, 5(1) (2020) 1052.
28. World Health Organization Website. <https://www.who.int/>.

Source of support: Nil, Conflict of interest: None Declared.

[Copy right © 2021. This is an Open Access article distributed under the terms of the International Journal of Mathematical Archive (IJMA), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.]