# Study of Transmission Dynamics of Novel COVID-19 by Using Mathematical Model ☆

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**Abstract**

In this research work, we present a mathematical model for novel coronavirus -19 (NCOVID-19) which is consisted on three different compartments susceptible, infected and recovered classes abbreviated as under convex incident rate involving and emigration rate. We first derive the formulation of the model. Also, we give some qualitative aspects for the model including existence of equilibriums and its stability results by using various tools of nonlinear analysis. Then by mean of nonstandard finite difference scheme (NSFD), we simulate the results against the data of Wuhan city for the sixty days. By means of simulation, we show how protection, exposure, emigration, death and cure rates affect the susceptible, infected and recovered population with the passage of time involving emigration. On the basis of simulation, we observe the dynamical behavior due to emigration of susceptible and infected classes or one of these two.

*Keywords:* Mathematical model; Novel coronavirus -19; Nonstandard finite difference scheme; Emigration rate.

## 1. Introduction

Novel coronavirus-19 is a new chain of corona group of virus that had not been identified in humans history before December 2019. For the first time COVID-19 was found in Wuhan, China in December 2019, and has spread to various urban areas in China as well as round about 196 different countries of the world. It has since been declared as an outbreak by World Health Organization (WHO). According to the data reported by "WHO (World Health Organization)", by May 5, 2020, the reported laboratory confirmed, affected humans reached more than 3.5 million including more than 0.255 millions death cases has been recorded. Some researchers have also claimed that there

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are other sources of this corona virus including dogs, pangolin, etc. As per recoded data the death rate is different in different countries. Currently the highest death rate has been observed in Europe, USA and Iran. The number of confirmed cases growing on a very fast track on daily basis and has been declared a worldwide pandemic disease.

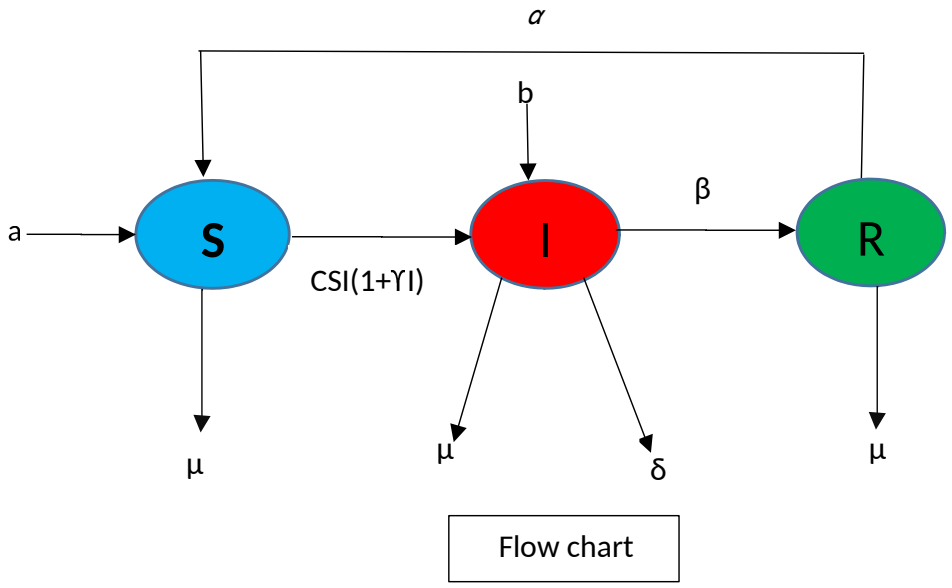
On 31st of December 2019, the WHO reported a novel corona virus (2019-nCoV) in Wuhan City, Hubei Province of China in humans, see [13, 14]. It was named as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by International Committee on Taxonomy of Viruses on 11th of February, 2020, for detail we refer [3, 5, 6, 7]. Firstly, this outbreak was identified in Wuhan with most early cases being reported in the city and later spread to the other countries in an alarming rate and became a lethal disease. There are different schools of thought behind the origin of the COVID-19 - some says that it might be bat origin (see [9]), some says that it might be related to a seafood market exposure (see [10]). If we observe International travel of any form has been a potential reason for the fast spread of the COVID-19 [8, 10, 11, 13]. So, emigration has a severe impact on the severity of spreading of the COVID-19. Recently, the whole world has been suffering due to a novel coronavirus pandemic and it was named by "Novel Coronavirus Infectious Disease (NCOVID-19)" which was claimed to outbreak first in Wuhan, central China (see [1]). It has been stated (fact) that the origin of NCOVID-19 is the transmission from animal to human as many infected cases claimed that they had been to a local fish and wild animal market in Wuhan in November (we refer [2]). Soon, some researchers confirmed that the transmission also happens person to person (see [13]). In present situation this pandemic has been produced very harmful effect on the health, economics and social life of the whole globe. In the whole world researchers, policy makers and doctors are struggling how to control this serious pandemic so that the lives of maximum people may be secured. They observed this disease from their own point of view. Also it is fact that most people infected with NCOVID-19 will experience mild to moderate respiratory illness. common symptoms: fever,tiredness,dry cough. Some peoples may experience: aches and pains, nasal congestion, runny nose, sore throat and diarrhoea.

Since mathematical models are powerful tools to understand the dynamics of real world phenomenon particulary the transmission of infectious disease. In literature large numbers of mathematical models of infectious disease have been studied, we refer few as [12, 15, 16, 17, 18, 19, 22]. By using mathematical models for understanding the transmission dynamics of a disease can help the researchers to make future prediction and to adopt some precautionary measure to save maximum papulation from lost. Also the mentioned tools help how to make strategies to control or eliminate the disease from society. Same as the case of current NCOVID-19, has been studied from different aspects in last few months, for detail see [23, 24, 25, 26]. Therefore motivated from the aforesaid discussion we observed that emigration has major roles in spreading the current disease in our society. It has been observed that due to emigration, this disease has been spread in the whole globe

within two three months. Therefore in this work we construct a modified SIR type model involving emigration rate to investigate the transmission dynamics of the aforementioned disease.

## 2. Model Formulation

This part of the paper is devoted to construct the mathematical model for our purposed problem. We take here three compartments; susceptible $S(t)$, infected $I(t)$, and recovered $R(t)$. Sine we construct the required model under convex incidence rate which is assumed to be a convex function with respect to the infective class due to host population. The benefit of using convex incidence rate is that it corresponds to an increased rate of infection because of two exposures over a small time period. Because a single contact tends catch infection at the rate $CIS$, while the new infective individuals arise from double exposures with $CI^2S$. It produce further chance that the recovered individual again may catch infection. Here we remark that the function $\Phi(S, I) = CI(t)S(t)(1 + \gamma I(t))$, where both $C, \gamma$ are positive constants. This is an interesting example for nonlinear incidence rate already used by some authors [15, 20, 21]. The Flow chart of the model is given as




The dynamics of the population are describe by the following differential equations:

$$\begin{aligned}
\frac{dS(t)}{dt} &= a - CI(t)S(t)(1 + \gamma I(t)) - \mu S(t) + \alpha R(t),\\
\frac{dI(t)}{dt} &= CI(t)S(t)(1 + \gamma I(t)) - (\beta + \mu + \delta - b)I(t),\\
\frac{dR(t)}{dt} &= \beta I(t) - (\alpha + \mu)R(t).
\end{aligned} \tag{1}$$

The parameters involved in model (1) are described as in Table 2.

| Parameters | The physical interpretation |
|---|---|
| $S(t)$ | Susceptible compartment |
| $I(t)$ | Infected compartment |
| $R(t)$ | Recovered compartment |
| $a$ | The recruitment rate |
| $\mu$ | Natural death |
| $\delta$ | Death due to corona |
| $b$ | The emigration rate of infected individuals |
| $\beta$ | Corona infection recovery rate |
| $C$ | infection rate |
| $\gamma$ | Rate at which recovered individuals lose immunity |
| $\alpha$ | rate of recovery from infection |

Table 1: The physical interpretation of the parameter.

First, for the equilibrium of the model (1), we consider it's existence. Corresponding to some values of parameters there exists a disease-free equilibrium for system (1) denoted by $E_0 = (a/\mu, 0, 0)$. To compute the non-negative equilibrium, we have

$$\begin{aligned} a - CI(t)S(t)(1+\gamma I(t)) - \mu S(t) + \alpha R(t) = 0, \\ CI(t)S(t)(1+\gamma I(t)) - (\beta + \mu + \delta - b)I(t) = 0, \\ \beta I(t) - (\alpha + \mu)R(t) = 0. \end{aligned}$$

To find the Basic Reproduction Number $R_0$, let $x = (S(t), I(t))$, in model(1). Then

$$\frac{dx}{dt} = \mathcal{F} - \mathcal{V},$$

where

$$\mathcal{F} = \begin{pmatrix} CI(t)S(t)(1+\gamma I(t)) \\ 0 \end{pmatrix}$$

and

$$\mathcal{V} = \begin{pmatrix} (\mu - a)S(t) \\ (\beta + \mu + \delta - b)I(t) \end{pmatrix}$$

for the disease-free equilibrium Jacobian of $\mathcal{F}$ is

$$F = \begin{pmatrix} 0 & CS^0 \\ 0 & 0 \end{pmatrix}$$

and Jacobian of $\mathcal{V}$ to deduce the disease-free equilibrium is given by

$$V = \begin{pmatrix} \mu - a & 0 \\ 0 & \beta + \mu + \delta - b \end{pmatrix}.$$

Hence, for the model (1), by simple calculation, we have

$$FV^{-}1 = \begin{pmatrix} 0 & \frac{CS^0(\mu-a)}{\beta+\mu+\delta-b} \\ 0 & 0 \end{pmatrix}.$$

Hence the Basic Reproduction Number (reproductive rate) $R_0$ is

$$R_0 = \frac{ac}{\mu(\beta+\mu+\delta-b)}. \tag{2}$$

From (2), we clearly observe that

($i$) There is no positive equilibria of model (6), if $R_0 \leq 1$;

($ii$) A unique positive equilibrium also known as endemic equilibrium $E^*(t) = (S^*(t), I^*(t), R^*(t))$ exists under $R_0 > 1$.

The endemic equilibrium is given by

$$\begin{aligned} S^*(t) &= \left(\frac{\alpha\beta}{CI^*(1+\gamma I^*)+\mu-a}\right) I^* \\ I^*(t) &= \frac{-(\mu-a)(\beta+\mu+\delta-b)+C\beta\gamma\alpha+\sqrt{\Omega}}{2C\gamma(\beta+\mu+\delta-b)} \\ R^*(t) &= \frac{\beta}{\alpha+\mu} I(t)^*. \end{aligned}$$

The value of $\Omega$ is given as

$$\Omega = (\mu-a)\left((\delta+\beta-b+d)+C\beta\gamma\alpha\right)^2 - 4\alpha\beta c^2(\mu-a)(\delta+\beta-b+\mu). \tag{3}$$

Next, we will elaborate the characteristics of these equilibria and a global mathematical analysis of system (1).

## 3. Dynamical Behavior of the Model

To elaborate the dynamic of system (1), we have the following lemma.

**Lemma 3.1.** *The system (1) has invariant manifold of plane $S(t)+I(t)+R(t) = a/\mu$, which is fascinating in the $1^{st}$ octant.*

*Proof.* Adding up all the equations of system (1) and let $N(t) = S(t)+I(t)+R(t)$. Then

$$\begin{aligned} \frac{dS(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt} &= a - \mu S(t) - \mu I(t) - \mu R(t) + bI(t) - \delta I(t) \\ \frac{d}{dt}(S(t)+I(t)+R(t)) &= a - (\delta-b)I(t) - \mu(S(t)+E(t)+I(t)+R(t)) \end{aligned} \tag{4}$$

(4) implies that

$$\frac{dN(t)}{dt} = a - (\delta-b)I(t) - \mu N(t). \tag{5}$$

Hence for (5) we present the general solution as

$$N(t) = \frac{1}{\mu}\left[a - (\delta - b)I(t) - dN(t_0)e^{(t_0 - t)}\right].$$

Which complete our conclusion. □

Now furthermore we reduced the system (1), because obviously the limit set of (1) on plane $S(t) + I(t) + R(t) = \frac{a}{\mu}$ has limit set.

$$\begin{aligned} \frac{dI(t)}{dt} &= C\left(\frac{a}{\mu} - I(t) - R(t)\right)(1 + \gamma I(t)) - (\beta + \mu + \delta - b) \triangleq \omega, (I(t), R(t)) \\ \frac{dR(t)}{dt} &= \beta I(t) - (\mu + \alpha)R(t) \triangleq \xi(I(t), R(t)). \end{aligned} \tag{6}$$

We have the following theorem with regards to the none existence of cyclical shells in system (6), which show the none existence of cyclical shells of system (1) by Lemma (3.1).

**Theorem 3.2.** *There does not exist nontrivial periodic orbits corresponding to System (6).*

*Proof.* Consider a "Dulac function" and consider system(6) for $I(t) > 0$ and $R(t) > 0$.

$$D(I(t), R(t)) = \frac{1 + \gamma I(t)}{CI(t)}.$$

Then, we have

$$\begin{aligned} D\omega &= \left(\frac{a}{\mu} - I(t) - R(t)\right)(1 + \gamma I(t)) - \frac{(\beta + \mu + \delta - b)(1 + \gamma I(t))}{C} \\ D\xi &= \frac{\beta I(t)}{C}(1 + \gamma I(t)) - \frac{(\mu + \alpha)(1 + \gamma I(t))R(t)}{CI(t)} \end{aligned}$$

$$\begin{aligned} \frac{\partial(D\omega)}{\partial I(t)} &= -(1 + \gamma I(t))\left[1 + 2\gamma R(t) + 3\gamma I(t) - \frac{2\gamma a}{\mu}\right] - \frac{\gamma}{C}\left[\beta + \mu D - b\right] \\ \frac{\partial(D\xi)}{\partial R(t)} &= -\frac{(\mu + \alpha)(1 + \gamma I(t))}{CI(t)}. \end{aligned} \tag{7}$$

By adding all equations of (7), we have

$$\frac{\partial(D\omega)}{\partial I(t)} + \frac{\partial(D\xi)}{\partial R(t)} = -(1 + \gamma I(t))\left[1 + 2\gamma R(t) + 3\gamma I(t) - \frac{2\gamma a}{\mu}\right].$$

Hence

$$-\frac{\gamma}{C}\left[\beta + \mu\delta - b\right] - \frac{(\mu + \alpha)(1 + \gamma I(t))}{CI(t)} < 0.$$

Thus (8) proved the conclusion of the theorem. □

To study $S_0$ disease-free equilibrium and its properties, and also the endemic equilibrium $S^*$, we rascal (6) with

$$x = \frac{C}{\mu + \alpha} I(t),$$

$$\begin{aligned} y &= \frac{C}{\mu+\alpha}R(t), \\ \tau &= (\mu+\alpha)t. \end{aligned}$$

One can obtain from (6) as

$$\begin{aligned} \frac{dx}{d\tau} &= \frac{x}{1+qx}(B-x-y)-nx, \\ \frac{dy}{d\tau} &= px-y, \end{aligned} \tag{8}$$

with

$$\begin{aligned} p &= \frac{\beta}{\mu+\alpha} \\ n &= \frac{\beta+\mu+\delta-b}{\mu+\alpha} \\ B &= \frac{aC}{\mu(\mu+\alpha)} \\ q &= \frac{\gamma(\mu+\alpha)^2}{C^2}. \end{aligned}$$

**Note:** Keep in mind that $(0,0)$ may be obtained from system (8) infact the "disease-free equilibrium" $S_0$ of system(1) and $(x^*, y^*)$ of system (8) is the unique positive equilibrium is infact the endemic equilibrium $S^*$ of system (1) under the condition $n - B < 0$ with $x^* = \frac{B-n}{q-1}$ and $y^* = px^*$. In first glance, we investigate for $(0,0)$, the stability and topological type trivial equilibrium. At the point $(0,0)$, the Jacobian matrix of system (8) is given by

$$M_0 = \begin{pmatrix} B-n & 0 \\ p & -1 \end{pmatrix}. \tag{9}$$

The dynamic of system (8) is equivalent to (10). If $B-n=0$, then there exists a small neighborhood $N_0$ of (0, 0).

$$\begin{aligned} \frac{dx}{d\tau} &= -x-2y+O((x,y)^3) \\ \frac{dy}{d\tau} &= px-y. \end{aligned} \tag{10}$$

From (10) $(0,0)$ is a saddle-node. The next results is important.

**Theorem 3.3.** *The trivial equilibrium point of the system (1) possess the following properties.*

(*i*) *As a result the system has a hyperbolic saddle, If* $n < B$.

(*ii*) *As a result the system has a saddle node, If* $n = B$.

(*iii*) *As a result the system has a stable hyperbolic node, If* $n > B$.

*Proof.* When $n - B < 0$, we study the topological type of endemic equilibrium $(x^*, y^*)$ and stability. From (8) at $(x^*, y^*)$, we have the Jacobian matrix

$$M_1 = \begin{pmatrix} \frac{B-2(1+p)x^*-n}{1+q} & \frac{-x^*}{1+q} \\ p & -1 \end{pmatrix},$$

where

$$\begin{aligned} det(M_1) &= \frac{2(1+p)x^* + n - B}{1+q} + \frac{px^*}{1+q} \\ &= \frac{(1+p)x^* + n - B + px^*}{1+q}. \end{aligned}$$

Thus $det(M_1)$ has not a unique sign due to

$$S_1 \triangleq (1+p)x^* + n - B + pX^*. \tag{11}$$

The relation (11) tells that $S1 > 0$ yields $det(M1) > 0$ and $(x^*, y^*)$ is a node (focus or a center). Also, for the stability of $(x^*, y^*)$, one can find the given results. □

**Theorem 3.4.** *The equilibrium $(x^*, y^*)$ of system (8) is locally stable in unique way and also it has a stable node if $n - B < 0$.*

*Proof.* From $tr(M_1)$ we examine $(x^*, y^*)$ for stability as:

$$\begin{aligned} tr(M_1) &= \frac{B - 2(1+p)x^* - n}{1+q} - 1 \\ &= \frac{B - 2(1+p)x^* - n - 1 - q}{1+q}. \end{aligned}$$

To determine $tr(M_1)$ in sign, we take

$$S_2 = -(2(1+p)x^* + q + 1 + n - B).$$

Let $S_2 = 0$. Then $n - B < 0$. Therefore $S_2 \neq 0$, which follows that $tr(M1) \neq 0$. Therefore for any positive values of parameters and $n - B < 0$ does not change the stability of $(x^*, y^*)$. Let $p = 1$, $q = 1$ and $B = 1$. Which implies that $tr(M1) = -1 < 0$. Due to the continuity of $tr(M_1)$ corresponding to parameters, as $tr(M1) < 0$ for $n - B < 0$. □

The following theorem summarized the results for the stability of the original system (1), in terms of the Basic Reproduction Number.

**Theorem 3.5.** *From (2) we define $R_0$.*

(*i*) *If $\mathcal{R}_0 < 1$, the model (1) has a unique disease-free equilibrium $E_0 = (\frac{a}{\mu}, 0, 0)$, which is a global attractor in the $1^{st}$ octant.*

(*ii*) *If* $\mathcal{R}_0 = 1$, *then model (1) has a unique disease-free equilibrium* $E_0 = (\frac{a}{\mu}, 0, 0)$ *which is a attracts of all orbits in the interior of the* $1^{st}$ *octant.*

(*iii*) *If* $\mathcal{R}_0 > 1$, *then model (1) has two equilibria, a disease-free equilibrium* $E_0 = (\frac{a}{\mu}, 0, 0)$ *and an endemic equilibrium* $E^*(t) = (S^*(t), I^*(t), R^*(t))$. *The endemic equilibrium* $E^*(t)$ *is a global attractor in the interior of the* $1^{st}$ *octant.*

## 4. Numerical Results and Discussion

We present numerical simulation for system (6) with the used values. We take different initial values corresponding to different classes involved in the model (6) for the month of March in four different localities in Pakistan. Here it is remarkable that for the numerical simulation, we have used the nonstandard finite difference scheme as already used in [15].
We use nonstandard finite difference (NSFD) scheme [28] to write the model in difference form as: consider first equation of model (6)

$$\frac{dS(t)}{dt} = a - CI(t)S(t)(1 + \gamma I(t)) - \mu S(t) + \alpha R(t) \tag{12}$$

which is decomposed in NSFD scheme as

$$\frac{S_{j+1} - S_j}{h} = a - CI_j(t)S_j(t)(1 + \gamma I_j(t)) - \mu S_j(t) + \alpha R_j(t) \tag{13}$$

Like 13, we can decomposed the model (6) in NSFD scheme and write the whole system as

$$\begin{aligned} S_{j+1} &= S_j + h\Big(a - CI_j(t)S_j(t)(1 + \gamma I_j(t)) - \mu S_j(t) + \alpha R_j(t)\Big) \\ I_{j+1} &= I_j + h\Big(CI_j(t)S_j(t)(1 + \gamma I_j(t)) + (\mu + \beta + \delta)I_j(t)\Big) \\ R_{j+1} &= R_j + h\Big(\beta I_j(t) - (\alpha + \mu)R_j(t)\Big). \end{aligned} \tag{14}$$

Using the scheme developed in (14), we present the numerical simulation of the model corresponding to the given values as In the presence of given rate of emigrant(s) in the Case I as $[0.098, 0.067, 0.0205, 0.0184]$, we present by graph according to the given data in Figure 1-3 to investigate the transmission dynamics of the various compartments of the considered model.

During the first thirty days in the presence of excessive rate of emigration the susceptible population is decreasing as shown in Figure 1. When the emigration rate is high, the decline in the papulation of uninfected (susceptible) people is deceasing, because they are exposing to infection and hence higher the emigration rate faster the growth rate of infected papulation and vice versa. As a results more death will occurs along with the recovery from the disease. Therefore the growth in the

| Parameters | physical description | Numerical value | Reference |
|---|---|---|---|
| $S(t)$ | Initial Susceptible compartment | $12.6 millions$ | [23] |
| $I(t)$ | Initial infected compartment | $0.84 millions$ | [23] |
| $R(t)$ | Initial recovered compartment | $0 millions$ | [23] |
| $a$ | The birth rate of infection | 0.1243 | [25] |
| $\mu$ | Natural death | 0.002 | [25] |
| $\delta$ | Death due to corona | 0.05 | [25] |
| $b$ | The emigration rate rate | 0.0205 | [26] |
| $\beta$ | Corona infection recovery rate | 0.09871 | [24] |
| $C$ | Infection rate | 0.580 | [25] |
| $\gamma$ | Rate at which recovered individuals lose immunity | 0.0003 | [26] |
| $\alpha$ | Rate of recovery | 0.854302 | [24] |

Table 2: The physical interpretation of the parameters and numerical values.

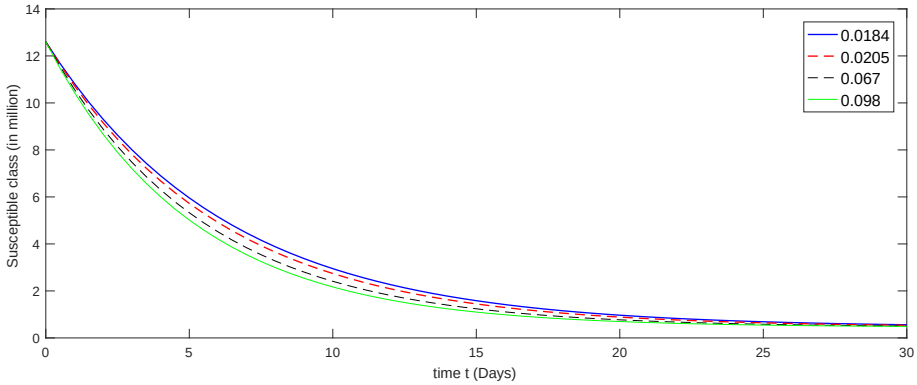



Figure 1: Dynamical behavior of susceptible class in the presence of given rate of emigrant(s) as Case I from 10 February to 10 March (2020).

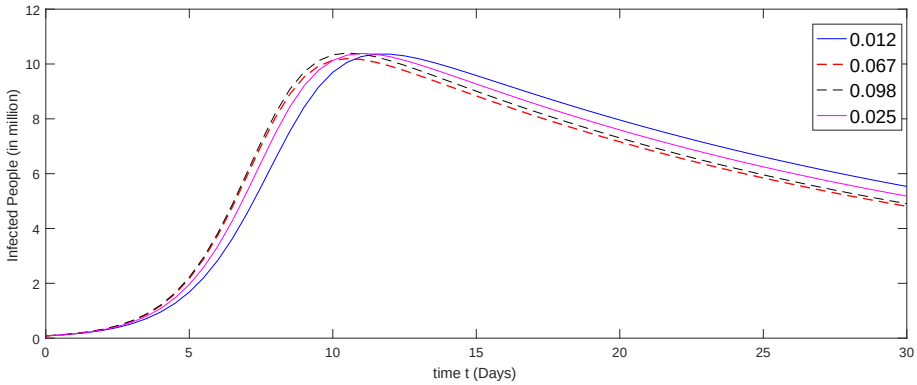



Figure 2: Dynamical behavior of infected class in the presence of given rate of emigrant(s) as Case I from 10 February to 10 March (2020).

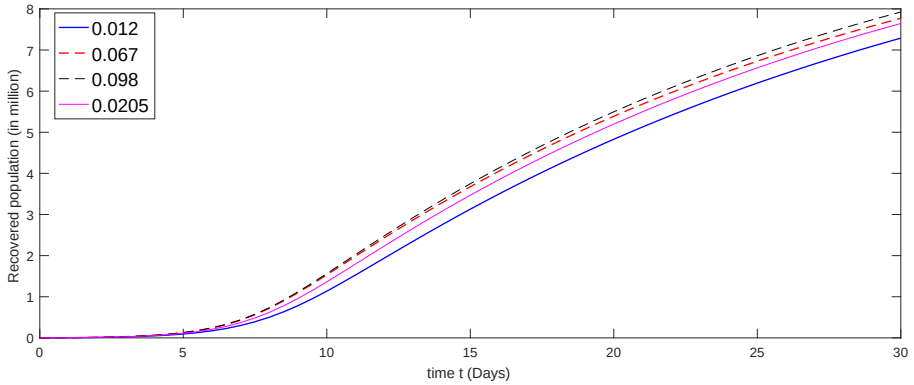



Figure 3: Dynamical behavior of recovered class in the presence of given rate of emigrant(s) as Case I from 10 February to 10 March (2020).

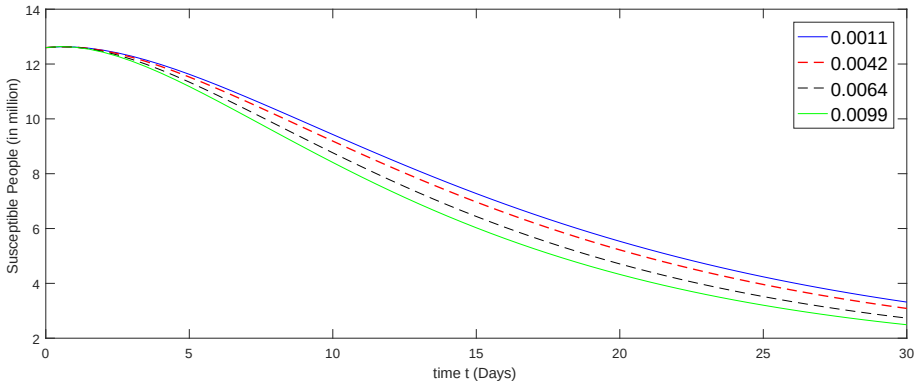



Figure 4: Dynamical behavior of susceptible class in the presence of given rate of emigrant(s) as Case II from 10 march to 10 April (2020).

recovery class is also different against different emigration. The concerned dynamics are presented by Figures 2 and 3 respectively.

Further we present by graphs in Figures 4-5 the dynamical behavior of the transmission dynamics corresponding to the second set of values of emigration rate assumed as $[0.0099, 0.0064, 0.0042, 0.0011]$ as Case II.

We see that as the emigration was slightly reduced during the thirty days from 10the March to 10the April. The decline in susceptible papulation at different rate is shown by Figure 4, while the corresponding dynamics of infectious and recovered class are presented via Figure 5 and 6 respectively. As the emigration rate is decreasing the susceptibility is deceases with slight rapid speed and consequently the infection rate is going on downing. The recovered papulation is also growing with faster speed when emigration rate is low. Because the chance of catching infection is deceasing.

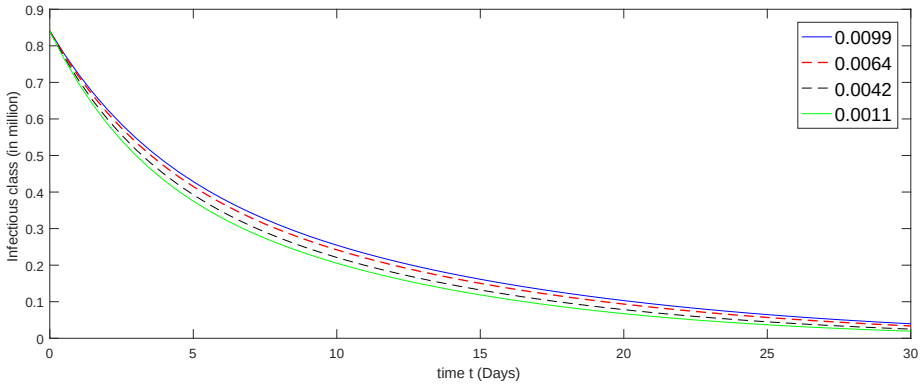



Figure 5: Dynamical behavior of infected class in the presence of given rate of emigrant(s) as Case II from 10 March to 10 April (2020).

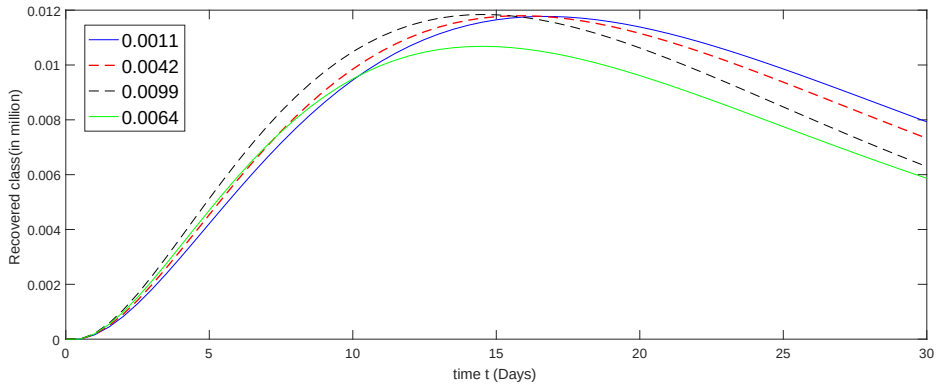



Figure 6: Dynamical behavior of recovered class in the presence of given rate of emigrant(s) as Case II from 10 March to 10 April (2020).

## 5. Conclusion

A mathematical model addresses the current novel coronavirus -19 under three compartments susceptible, infected and recovered has been studied. By nonlinear analysis the existence of global and local stability analysis has been demonstrated. On using nonstandard finite difference numerical method, we have simulated the results by using the real data of Wuhan city during the last sixty days from 10the February 2020 to 10the April 2020. Our model has been simulated for the fixed values of the parameters except emigration rate. In fist set of data we have simulated the model against highest values of emigration rate, we observed that due to this the infectious has rapidly transmitted from person to person during the first thirty days in the mentioned place. During this time more death have been occurred and recoded rate was also increased properly. After reducing the concerned emigration rate properly the dynamics greatly affected and the infection rate started on decreasing and the recovery rate also increased with different rate. Because the rate of emigration was different. On smaller emigration rate the rate of spread of infection is slow as compared at higher order and vice versa. We concluded that minimizing the emigration during this out break can cause the increase in protection rate. In other words avoiding unnecessary emigration of people will greatly helped in reducing or controlling this disease.

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## 6. Availability of data and materials

Not applicable.

## 7. Competing interests

The authors declare that they have no competing interests.

## Authors contributions

All authors contributed equally to this article. All authors read and approved the final manuscript

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