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Dynamics of the Mathematical Model Related to COVID-19 Pandemic with Treatment

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Abstract COVID-19, declared as a pandemic worldwide, has different effects on people. Although there is still no specific vaccine or a single type of treatment for this disease, it is known that various treatment methods are used for this disease. This study is based on the idea that, contrary to the claims that herd immunity against COVID-19 can be achieved, most patients who respond to treatment may also lose immunity after recovery. To analyze the dynamic behavior of COVID-19 with a mathematical model, a new modified SIRS model with a treatment function is considered. Findings show that the disease presents a situation that leads to chaos.

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1. Introduction

In the process from the past to the future; war, famine, and infectious diseases are factors that make survival difficult [1]. In 1918-1920, the flu pandemic killed more than 100 million people [2]. Since 1981, AIDS has emerged as a global epidemic threatening the world's health [3]. In 2003, SARS [4] and today's disease COVID-19, which firstly started in the city of Wuhan, Hubei, China in early December of 2019, affect all humanity worldwide [5, 6]. As a result of this situation, researchers have done many studies to better understand and control diseases. Epidemic models are formulated to describe the extent of disease in a community [7–10]. Therefore, the dynamics of epidemic models are among the important study topics. The disease processes have been studied in detail by using mathematical models such as SI, SIS, SIR, SIRS, SEIR, SEIRS, MSEIR and MSEIRS in order to model, predict, control and treat epidemic diseases that have been tried to resist

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since the early 1900s. As many researchers examine the asymptotic behavior [7–9, 11, 12] and global behavior [13, 14] of epidemic models, most researchers examine bifurcation behavior [15–17].

When the literature is examined, we see the analysis of the epidemic models associated with COVID-19 disease since early 2020. In particular, it is tried to obtain results about COVID-19 by using mathematical models.

The world is facing the growing COVID-19 pandemic. The World Health Organization (WHO) leads and coordinates the global effort, supporting countries to prevent, detect and respond to the pandemic. To date, WHO have reported 40.455.651 confirmed cases of COVID-19 including 1.119.431 deaths in world-wide [18]. Although a total of 10.040.766.359 doses of vaccine were administered as of 1 February 2022, globally, as of 2 February 2022, there were 380.321.615 confirmed cases of COVID-19, including 5.680.741 deaths reported to WHO. Due to the rapidly increasing number of patients, adequate and necessary treatment may not be provided for patients in hospitals.

In diseases, vaccination is a preventive strategy and treatment is an important method to reduce the spread of diseases [15, 19, 20]. During an outbreak, if no vaccine or treatment is available for the disease, an alternative strategy is to quarantine suspected cases or isolate those diagnosed with the disease. Although no pharmaceutical product has been shown to be safe and effective for the treatment of COVID-19 in the early stages of the disease; in many countries, doctors have patients with COVID-19 drugs that are not approved for this disease.

Also, [21] gives information about drugs. Although there is no product approved by the U.S. Food and Drug Administration (FDA) to treat coronavirus disease 2019 (COVID-19), many medications are being tested. One investigation drug called remdesivir has been authorized by the FDA for emergency use during the COVID-19 pandemic. Remdesivir may be prescribed for people who are hospitalized with COVID-19. It's given through a needle in the skin (intravenously). In addition to remdesivir, other antiviral drugs being tested include favipiravir and merimepodib. A recent study found it reduced deaths by about 30% for people on ventilators and by about 20% for people who needed supplemental oxygen. The U.S. National Institutes of Health has recommended this drug for people hospitalized with COVID-19 who are on mechanical ventilators or need supplemental oxygen .

When people recover from COVID-19, their blood contains antibodies that their bodies produced to fight the coronavirus and help them get well. Antibodies are found in plasma, a component of blood. Convalescent plasma - literally plasma from recovered patients - has been used for more than 100 years to treat a variety of illnesses from measles to polio, chickenpox, and SARS. It is widely believed to be safe. In the current situation, antibody-containing plasma from a recovered patient is given by transfusion to a patient who is suffering from COVID-19. The donor antibodies may help the patient fight the illness, possibly shortening the length or reducing the severity of the disease. Though convalescent plasma has been used for many years, and with varying success, not much is known about how effective it is for treating COVID-19. A recent analysis of 35,000 hospitalized patients who received convalescent plasma to treat severe COVID-19 suggests that the therapy may reduce the risk of dying. The data comes from the ongoing Expanded Access Program (EAP) led by the Mayo Clinic. The researchers found that patients with (or at risk of) severe COVID-19 who received convalescent plasma within three days of

diagnosis were less likely to die than patients who received convalescent plasma later in their illness [22].

While the latest research suggests that antibodies against COVID-19 could be lost in just three months, a new hope has appeared on the horizon: the enigmatic T cell. The clues have been mounting for a while. First, scientists discovered patients who had recovered from infection with COVID-19, but mysteriously didn't have any antibodies against it. Next it emerged that this might be the case for a significant number of people. Then came the finding that many of those who do develop antibodies seem to lose them again after just a few months. In short, though antibodies have proved invaluable for tracking the spread of the pandemic, they might not have the leading role in immunity that we once thought. If we are going to acquire long-term protection, it looks increasingly like it might have to come from somewhere else. Several studies have shown that people infected with COVID-19 tend to have T cells that can target the virus, regardless of whether they have experienced symptoms. Hayday says: There's every evidence that the T cells can protect you, probably for many years. But when people get ill, the rug seems to be being pulled from under them in their attempts to set up that protective defense mechanism [18].

Immunity against COVID-19 is still uncertain. While it was observed that some patients recovered without producing antibodies, in others, even if antibodies were produced against the disease, these antibodies were found to disappear in the body after a certain period of time. Also; individuals infecting may become infected by exposure to low doses of viral particles. Each of these situations means that patients may be more likely to become re-infected if they are exposed to high doses of viral particles for the second time.

Plan of the paper as follows:

2. Mathematical Formulation and Existence of Positivity of (2.3)

Mathematical model of COVID-19 disease system of SIR are formulated in this section. Also, the existence and uniqueness and non-negative of the solution of the system (2.3) are investigated. This study aims to offer a new approach to COVID-19 disease by using a SIRS model. The basic epidemic models is an epidemic model subject to treatment (see [20]) without births and deaths. The model has the following form:

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\beta SI}{N} + \nu(N - S - I) \\ \frac{dI}{dt} &= I\left(\frac{\beta S}{N} - \gamma - \kappa\right) \\ \frac{dR}{dt} &= \gamma I - \nu(N - S - I) + \kappa I, \end{aligned} \tag{2.1}$$

where $S(0) \geq 0, I(0) \geq 0, R(0) \geq 0$ and $S(0)+I(0)+R(0) = N$. Thus, $S(t)+I(t)+R(t) = N$. The coefficient are as follows:

- β : average number of adequate contacts made by an infected individual per time.
- $\frac{\beta}{N}S$: average number of adequate contacts made by an infected individual NI resulting in an infection of a susceptible individual per time.

- $\frac{\beta}{N}SI$: number of infections caused by all infected individuals per time.
- γ : recovery rate, $\frac{1}{\gamma}$ = average infectious period.
- ν : rate of loss of immunity, $\frac{1}{\nu}$ = average length of immunity.
- N : total population size.
- κ : treatment rate.

The epidemic model is mentioned In [23]: Infectious diseases such as measles, mumps, rubella, and chickenpox are modeled by classifying individuals in the population according to their status with respect to the disease, healthy, infected, and immune. Diseases caused by viruses or bacteria are not modeled directly at the population level, only indirectly through the number of infected individuals. The disease states: S(susceptible); I(infected) and R(removed), are defined. The dynamics of SIRS model differ from the SIR model. It is not always the case that the epidemic dies out. The loss of immunity by immune individuals allows the disease to become endemic. The basic reproduction number is a threshold which determines whether the disease becomes endemic.

Since $R(t)$ can be obtained from $S(t)$ and $I(t)$, it is sufficient to consider only the variables S and I. The differential equations in S and I are given by

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\beta SI}{N} + \nu(N - S - I) \\ \frac{dI}{dt} &= I\left(\frac{\beta S}{N} - \gamma - \kappa\right). \end{aligned} \tag{2.2}$$

Using the forward Euler's Formula, we obtain as follows:

$$\begin{aligned} S_{t+1} &= S_t + \delta\left(\nu\left(N - S_t - I_t\right) - \frac{\beta S_t I_t}{N}\right) \\ I_{t+1} &= I_t + \delta I_t\left(\frac{\beta S_t}{N} - \gamma - \kappa\right). \end{aligned} \tag{2.3}$$

2.1. Existence and Uniqueness of the Solution

The sufficient condition for the existence and uniqueness of the solution of the system (2.3) is as follow:

Theorem 2.1. *For each initial condition which is positive, there exists a unique solution for the system (2.3).*

Proof. We prove a sufficient condition for the existence and uniqueness of the solutions of the system (2.3) in the region $\Delta \times (0, T]$ where

$$\Delta = \left\{(S_t, I_t) \in \mathbb{R}^2 : \max\left(|S_t|, |I_t| \leq \eta\right)\right\}.$$

The technique applied in [24] is implemented here. Consider $\mathbb{H}(x) = (\mathbb{H}_1(x), \mathbb{H}_2(x))$, a mapping is defined by

$$\begin{aligned} \mathbb{H}_1(x) =& S_t + \delta\left(\nu\left(N - S_t - I_t\right) - \frac{\beta S_t I_t}{N}\right) \\ \mathbb{H}_2(x) =& I_t + \delta I_t\left(\frac{\beta S_t}{N} - \gamma - \kappa\right). \end{aligned} \tag{2.4}$$

For any $x, \bar{x} \in \Delta$, it follows from (2.4) that

$$\begin{aligned}
&\|\mathbb{H}(x) - \mathbb{H}(\bar{x})\| = |\mathbb{H}_1(x) - \mathbb{H}_1(\bar{x})| + |\mathbb{H}_2(x) - \mathbb{H}_2(\bar{x})| \\
&= \left| S_t + \delta\nu N - \delta\nu S_t - \delta\nu I_t - \frac{\delta\beta S_t I_t}{N} + \overline{S_t} - \delta\nu N + \delta\nu\overline{S_t} + \delta\nu\overline{I_t} + \frac{\delta\beta\overline{S_t I_t}}{N} \right| \\
&\quad + \left| I_t + \frac{\delta\beta S_t I_t}{N} - \delta\gamma I_t - \delta\kappa I_t - \overline{I_t} - \frac{\delta\beta\overline{S_t I_t}}{N} + \delta\gamma\overline{I_t} + \delta\kappa\overline{I_t} \right| \\
&\|\mathbb{H}(x) - \mathbb{H}(\bar{x})\| \\
&= \left| \left(S_t - \overline{S_t}\right) - \delta\nu\left(S_t - \overline{S_t}\right) - \delta\nu\left(I_t - \overline{I_t}\right) - \frac{\delta\beta I_t}{N}\left(S_t - \overline{S_t}\right) - \frac{\delta\beta\overline{S_t}}{N}\left(I_t - \overline{I_t}\right) \right| \\
&\quad + \left| \left(I_t - \overline{I_t}\right) - \delta\gamma\left(I_t - \overline{I_t}\right) - \delta\kappa\left(I_t - \overline{I_t}\right) + \frac{\delta\beta I_t}{N}\left(S_t - \overline{S_t}\right) + \frac{\delta\beta\overline{S_t}}{N}\left(I_t - \overline{I_t}\right) \right| \\
&\leq \left[1 + \delta\nu + \frac{2\beta\eta}{N}\right]\left(S_t - \overline{S_t}\right) + \left[1 + \delta(\nu + \gamma + \kappa) + \frac{2\beta\eta}{N}\right]\left(I_t - \overline{I_t}\right) \\
&\|\mathbb{H}(x) - \mathbb{H}(\bar{x})\| \leq \Omega \|x - \bar{x}\|,
\end{aligned}$$

where $\Omega = \max\left\{1 + \delta\nu + \frac{2\beta\eta}{N}, 1 + \delta(\nu + \gamma + \kappa) + \frac{2\beta\eta}{N}\right\}$.

Thus $\mathbb{H}(x)$ satisfies the Lipschitz condition and hence it leads to the existence and uniqueness of solution of system (2.3). ∎

2.2. Non-Negativity of System (2.3)

Theorem 2.2. *The system (2.3) has positive solution, if the following conditions hold:*

(1) $\delta(\beta + \nu) < 1$, *then* $S_{t+1} > 0$, $\forall t$.
(2) $\delta(\beta - \gamma - \kappa) < 1$, *then* $I_{t+1} > 0$, $\forall t$.

Proof. The theorem is proved by direct proof and induction [25]. We know that $S_t \geq 0, I_t \geq 0, R_t \geq 0$ and $S_t + I_t + R_t = N$. Now consider the equation

$$\begin{aligned}
S_{t+1} =& S_t + \delta\left[\frac{\beta S_t I_t}{N} + \nu N - \nu S_t - \nu I_t\right] \\
=& S_t\left[1 - \delta\left(\frac{\beta I_t}{N} + \nu\right)\right] + \delta\nu(N - I_t).
\end{aligned}$$

We need to ensure that $1 - \delta\left(\frac{\beta I_t}{N} + \nu\right) > 0$. As $S_t \geq 0, I_t \geq 0, R_t \geq 0$ and $S_t + I_t + R_t = N$ leads to $0 \leq I_t \leq N$. Hence

$$\delta\left(\frac{\beta I_t}{N} + \nu\right) \leq \delta\left(\frac{\beta N}{N} + \nu\right) = \delta(\beta + \nu).$$

So if we consider

$$\delta(\beta + \nu) < 1$$

then

$$\delta\left(\frac{\beta I_t}{N} + \nu\right) \leq \delta(\beta + \nu).$$

Similarly, we can show that $I_{t+1} \geq 0$ if $\delta(\beta - \gamma - \kappa) < 1$. This completes the proof. ∎

3. The Existence and Stability of Equilibrium Points of System (2.3)

In this section, we consider the epidemic model (2.3). Firstly, we discuss the existence of equilibrium points for the model (2.3), and then study the stability of the equilibrium points by using the characteristic polynomial or the eigenvalues of the matrix evaluated at the equilibrium points. Now, let us give some necessary information.

Definition 3.1. [26] The following situations are valid for the equilibrium point (S, I) of any system

(i) If $|\lambda_1| < 1$ and $|\lambda_2| < 1$, it is a *sink point* and locally asymptotically stable;
(ii) If $|\lambda_1| > 1$ and $|\lambda_2| > 1$, it is a *source point* and locally unstable;
(iii) If $|\lambda_1| < 1$ and $|\lambda_2| > 1$ (or $|\lambda_1| > 1$ and $|\lambda_2| < 1$), it is a *saddle point*;
(iv) If $|\lambda_1| = 1$ or $|\lambda_2| = 1$, it is *non-hyperbolic*.

Theorem 3.2. *[23] Let λ_1 and λ_2 be the roots of the characteristic equation of (2.3). If $|\lambda_{1,2}| < 1$ or $|Tr(J)| < 1 + det(J) < 2$, the equilibrium point (S, I) is locally asymptotically stable.*

Theorem 3.3. *[26] Let us take $F(\lambda) = \lambda^2 + B\lambda + C$ such that $F(1) > 0$. Also λ_1 and λ_2 are two roots of $F(\lambda) = 0$. So that the following are valid*

(i) If $F(-1) > 0$ and $C < 1$, $|\lambda_1| < 1$ and $|\lambda_2| < 1$;
(ii) If $F(-1) < 0$, $|\lambda_1| < 1$ and $|\lambda_2| > 1$ (or $|\lambda_1| > 1$ and $|\lambda_2| < 1$);
(iii) If $F(-1) > 0$ and $C > 1$, $|\lambda_1| > 1$ and $|\lambda_2| > 1$;
(iv) If $F(-1) = 0$ and $B \neq 0, 2$, $\lambda_1 = -1$ and $|\lambda_2| \neq 1$;
(v) If $B^2 - 4C < 0$ and $C = 1$, λ_1 and λ_2 complex root and $|\lambda_{1,2}| = 1$.

Then, we have the following Lemma and Theorem for model (2.3).

Lemma 3.4. *The existence of equilibrium steady states of the model (2.3) satisfies:*

(1) The disease free equilibrium steady state $E_0 = (N, 0)$ always exists.
(2) If $\beta > \gamma + \kappa$, then the unique positive endemic equilibrium steady state $E_1 = \left(\frac{N(\kappa+\gamma)}{\beta}, \frac{N\nu(\beta-\gamma-\kappa)}{\beta(\kappa+\nu+\gamma)}\right)$ exists.

Theorem 3.5. *Assume that $\beta > \gamma + \kappa$. We have the following conditions for the endemic equilibrium point E_1.*

(1) If $4 > 2\delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) + \delta^2\nu(\beta-\gamma-\kappa)$ and $\delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) > \delta^2\nu(\beta-\gamma-\kappa)$, then the endemic equilibrium point E_1 is a sink point.
(2) if $4 < 2\delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) + \delta^2\nu(\beta-\gamma-\kappa)$, then the endemic equilibrium point E_1 is a saddle point.
(3) If $4 > 2\delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) + \delta^2\nu(\beta-\gamma-\kappa)$ and $\delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) < \delta^2\nu(\beta-\gamma-\kappa)$, then the endemic equilibrium point E_1 is a source point.
(4) If $2\delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) + \delta^2\nu(\beta-\gamma-\kappa) = 4$ and $2\delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) \neq 2$, then the endemic equilibrium point E_1 is a Flip bifurcation point.

(5) If $(\beta+\nu)^2 < (\kappa+\nu+\gamma)^2(\beta-\gamma-\kappa)$ *and* $\delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) = \delta^2\nu(\beta-\gamma-\kappa)$, *then the endemic equilibrium point* E_1 *is a Neimark-Saker bifurcation point.*

Proof. Let us consider model (2.3), we have

$$f = S + \delta\left(\nu\left(N - S - I\right) - \frac{\beta SI}{N}\right)$$
$$g = I + \delta I\left(\frac{\beta S}{N} - \gamma - \kappa\right),$$

and the Jacobian matrix is obtained the following form

$$J(S,I) = \begin{bmatrix} 1+\delta\left(-\frac{\beta I}{N} - \nu\right) & \delta\left(-\frac{\beta S}{N} - \nu\right) \\ \delta\frac{\beta I}{N} & 1+\delta\left(\frac{\beta S}{N} - \gamma - \kappa\right) \end{bmatrix}.$$

We found as

$$J(E_1) = \begin{bmatrix} 1-\delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) & -\delta\left(\kappa+\gamma+\nu\right) \\ \delta\nu\left(\frac{\beta-\gamma-\kappa}{\kappa+\nu+\gamma}\right) & 1 \end{bmatrix}.$$

Jacobian matrix evaluated at equilibrium point. The trace and determinant of the Jacobian matrix can written

$$B = \text{Trace}[J(E_1)] = 2 - \delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right),$$
$$C = \text{Det}[J(E_1)] = 1 - \delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) + \delta^2\nu(\beta-\gamma-\kappa).$$

From $|J(E_1) - \lambda I| = 0$, the characteristic polynomial of the matrix is given by

$$F(\lambda) = \lambda^2 - \left[2 - \delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right)\right]\lambda + 1 - \delta\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right) + \delta^2\nu(\beta-\gamma-\kappa). \quad (3.1)$$

We can prove the results (1) - (5) by using Theorem 3.3. This completes the proof. ∎

4. Analysis of Neimark-Sacker Bifurcation and Its Chaos Control

The existence of Neimark-Sacker bifurcation and its chaos control are analyzed in this section. The following Lemma has equivalent to the Theorem 3.5 depending on the parameter δ. At, E_1, the Jacobian matrix is

$$J(E_1) = \begin{bmatrix} 1+\delta a_{11} & -\delta a_{12} \\ \delta a_{21} & 1 \end{bmatrix}. \quad (4.1)$$

Here $a_{11} = -\nu\left(\frac{\beta+\nu}{\kappa+\nu+\gamma}\right), a_{12} = (\kappa+\gamma+\nu)$ and $a_{21} = \nu\left(\frac{\beta-\gamma-\kappa}{\kappa+\nu+\gamma}\right)$. The characteristic equation is $F(\lambda) = \lambda^2 - T\lambda + D$, $T = 2 + \delta a_{11}$ and $D = 1 + \delta a_{11} + \delta^2 a_{12}a_{21}$. The eigen values are

$$\lambda_{1,2} = 1 + \frac{\delta U}{2} \pm \frac{\delta}{2}\sqrt{U^2 - 4V},$$

while $U = a_{11}$ and $V = a_{12}a_{21}$.

Lemma 4.1. *The endemic equilibrium point E_1 is a*

(1) sink if one of the following conditions are satisfied:
  (i) $\mathbb{M}^* < 0$ and $\delta < \delta_3$,
  (ii) $\mathbb{M}^* \geq 0$ and $\delta < \delta_2$,
(2) source if one of the following conditions are satisfied:
  (i) $\mathbb{M}^* < 0$ and $\delta > \delta_3$,
  (ii) $\mathbb{M}^* \geq 0$ and $\delta > \delta_1$,
(3) saddle if $\mathbb{M}^* \geq 0$ and $\delta_2 < \delta < \delta_1$,
(4) non-hyperbolic if one of the following conditions are satisfied:
  (i) $\mathbb{M}^* < 0$ and $\delta = \delta_3$,
  (ii) $\mathbb{M}^* > 0$ and $\delta = \delta_1$ or $\delta = \delta_2$.

$\mathbb{M}^* = U^2 - 4V$ *and* $\delta_1 = \dfrac{-U + \sqrt{U^2 - 4V}}{V}, \delta_2 = \dfrac{-U - \sqrt{U^2 - 4V}}{V}, \delta_3 = -\dfrac{U}{V}$.

Let δ be the bifurcation parameter considered to analyzes Neimark-Sacker bifurcation. The occurrence of this bifurcation is ensured when the eigenvalues at endemic equilibrium states are complex conjugate with modulus equal to 1 [27]. The quadratic equation obtained from (4.1) is $F(\lambda) = \lambda^2 - (2 + \delta U)\lambda + (1 + \delta U + \delta^2 V)$.

From Lemma 4.1, if $\mathbb{M}^* < 0$ and $\delta = \delta_3$, then the eigenvalues are

$$\lambda_{1,2} = 1 - \frac{U^2}{2V} \pm i\frac{U}{2V}\sqrt{4V - U^2}.$$

Now we conclude the theorem for the existence of the Neimark-sacker bifurcation of system (2.3).

Theorem 4.2. *The Neimark-sacker bifurcation of system (2.3) occurs when $\mathbb{M}^* < 0$ and $\delta = \delta_3$ and*

$$|\lambda_{1,2}| = \left|1 - \frac{U^2}{2V} \pm i\frac{U}{2V}\sqrt{4V - U^2}\right| = 1.$$

Next hybrid controlled strategy (see [28]) is applied to control the chaos of the model (2.3) and is given by

$$\begin{aligned} S_{t+1} &= \alpha S_t + \alpha\delta\left(\nu\left(N - S_t - I_t\right) - \frac{\beta S_t I_t}{N}\right) + (1 - \alpha)S_t \\ I_{t+1} &= \alpha I_t + \alpha\delta I_t\left(\frac{\beta S_t}{N} - \gamma - \kappa\right) + (1 - \alpha)I_t. \end{aligned} \tag{4.2}$$

where $0 < \alpha < 1$. Parameter perturbation and feedback control are combined in (4.2) as control strategy and appropriate choice of α results in partial or completely elimination of Neimark sacker bifurcation. Jacobian of (4.2) at E_1 is

$$J(E_1) = \begin{bmatrix} 1 + \alpha\delta a_{11} & -\alpha\delta a_{12} \\ \alpha\delta a_{21} & 1 \end{bmatrix} \tag{4.3}$$

Here a_{11}, a_{12}, a_{21} are same as given in (4.1). The presence of the roots of the (4.3) in the unit disk ensure the asymptotic stability of E_1.

5. Numerical Simulation

Theoretical analysis is verified in this section and supported with appropriate examples by considering some special cases of system (2.3). Numerical simulations manifest clearly interesting rich complex dynamics behaviors. Moreover, the orbits of the solutions with phase plane diagrams for the COVID-19 model (2.3) are exhibited. Dynamic nature of the COVID 19 model (2.3) about the endemic equilibrium steady state under different sets of parameter values are presented.

Example 5.1. Consider the parameter values $N = 100, \delta = 3.6, \beta = 0.84, \nu = 0.265, \gamma = 0.1$ and $\kappa = 0.3$ with the initial conditions $(95, 5)$. Computation yields $(S^*, I^*) = (47.619, 20.8736)$. The Jacobian matrix is $J = \begin{bmatrix} -0.5854 & -2.394 \\ 0.6314 & 1 \end{bmatrix}$. Here $T = 0.4146$, $D = 0.9262$ and the eigen values are $\lambda_{1,2} = 0.2073 \pm i0.9398$ such that $|\lambda_{1,2}| = 0.9624 < 1$. The criteria for stability are satisfied. Hence the system (2.3) is stable, see Figure 1. The phase portrait in Figure 2.1 (b) shows a sink and spiraling of the trajectory towards the steady state (S^*, I^*).

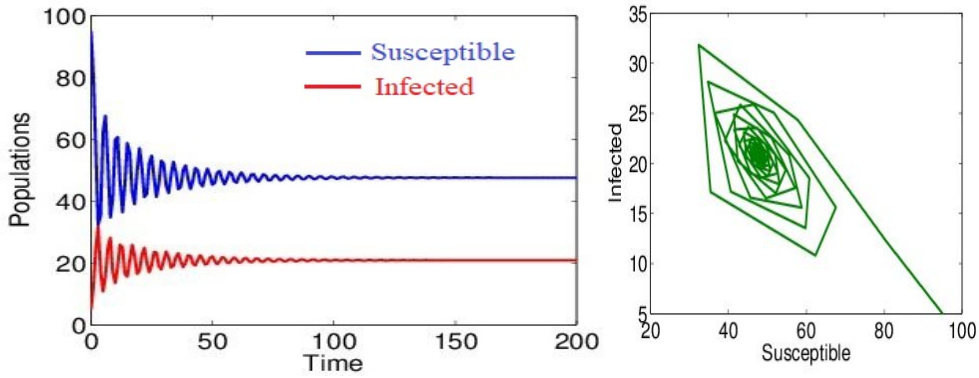

Figure 1. Time Series and Phase Plane of the Stability of the Model (2.3)

Example 5.2. Considering the values $N = 100, \delta = 4.1, \beta = 0.84, \nu = 0.265, \gamma = 0.1$ and $\kappa = 0.3$ with the initial conditions $(95, 5)$ yields the endemic equilibrium state $(S^*, I^*) = (47.619, 20.8736)$. The Jacobian matrix is $J = \begin{bmatrix} -0.8056 & -2.7265 \\ 0.7191 & 1 \end{bmatrix}$. Here $T = 0.1944$, $D = 1.1550$ and the eigen values are $\lambda_{1,2} = 0.0972 \pm i1.0703$ such that $|\lambda_{1,2}| = 1.0747 > 1$. The criteria for stability are not satisfied. Hence the system (2.3) is unstable. The trajectory spirals inwards but does not approach a point. The trajectory finally settles down as a limit cycle, see the phase portrait in Figure 2.

Example 5.3. Taking the values $M = 100, \beta = 0.84, \nu = 0.265, \gamma = 0.1, \kappa = 0.3$ and $\delta \in (3.7, 4.57)$ in the model (2.3) with the initial condition $S(0) = 95$ and $I(0) = 5$. This example is considered for Neimark-Sacker bifurcation. By simple calculation, unique positive endemic equilibrium steady state is estimated to be $(S^*, I^*) = (47.619, 20.8736)$. Moreover, the conditions of Lemma 4.1 are verified as follows: $U = -0.4404; V =$

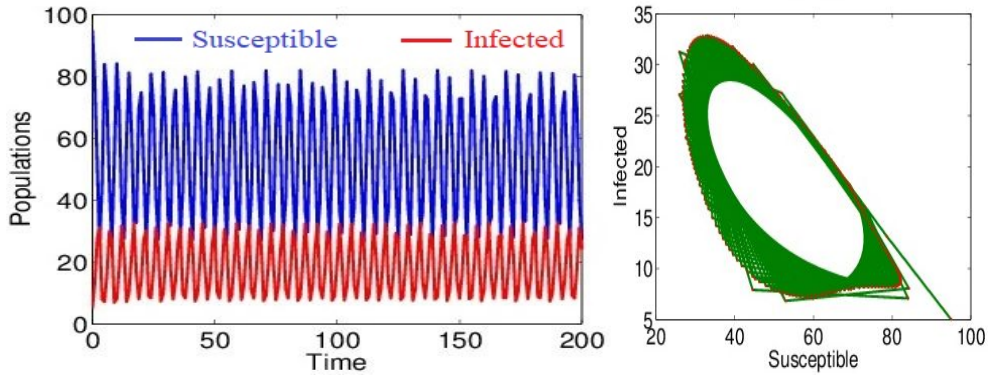

FIGURE 2. Time Series and Phase Plane of the Unstability of the Model (2.3)

$0.1166; M^* = -0.2724 < 0$ and $\delta_3 = 3.777$. Eigenvalues are $\lambda_{1,2} = 0.1683 \pm i0.9860$ with $|\lambda_{1,2}| = 1$. By Lemma 4.1, conditions for Neimark-Sacker bifurcation are obtained near the endemic equilibrium steady state E_1 at the bifurcation critical value δ_3.

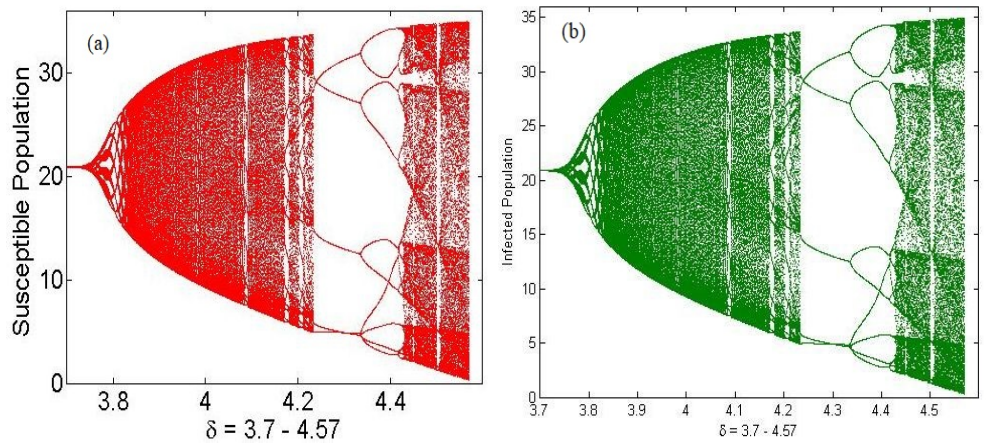

FIGURE 3. Neimark-Sacker Bifurcation of COVID-19 Pandemic Model of (2.3)

Neimark-Sacker bifurcation diagrams of the endemic equilibrium point E_1 of the model (2.3) in (δ, S) and (δ, I) planes are displayed in Figure 3 (a) & (b). Phase portraits of system (2.3) for different values of δ are presented in Figure 4. From Figure 3, it is observed that endemic equilibrium state of system (2.3) is locally asymptotically stable for $\delta < \delta_3 = 3.777$, lose its stability at $\delta = \delta_3$ and a stable invariant cycle bifurcates from E_1 for $\delta > \delta_3$. Also quasi-periodic orbits on the invariant cycle arise for $\delta > \delta_3$ and period orbits emerge in the period-windows, the orbits move towards chaos with the increasing of δ.

Phase plane portraits are also presented for the system with various values of δ in Figure 4. For $\delta = 3.75$, the solution curve spirals inwards and settles down indicating

stability and for δ from 3.8 to 4.2, the curve moves spirals inwards it settles down as limit cycle and indicating unstability. For $\delta = 4.3 - 4.4$, the solution curve spirals inwards but does not converge to a point. Finally for $\delta = 4.45 - 4.57$ the circle disappears and chaotic attractors appear. Comparing the bifurcation and phase plane diagrams, we justify our conclusions.

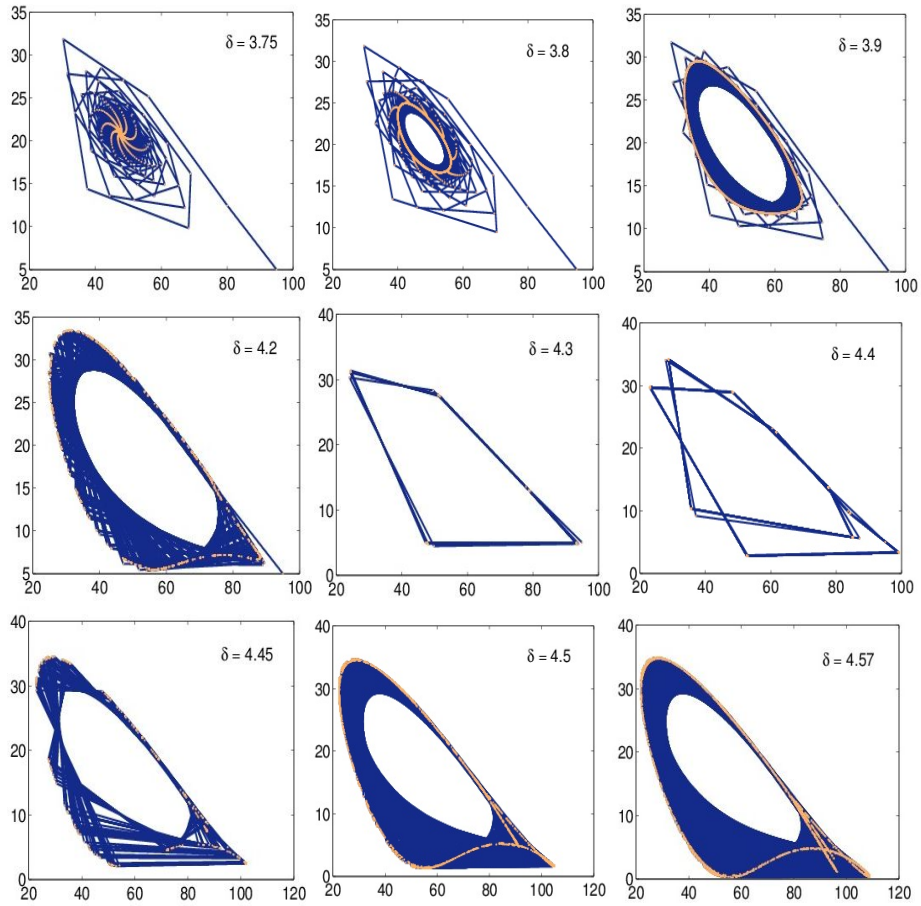

FIGURE 4. Phase Plane of the COVID-19 Pandemic Model of (2.3) for Different Values of δ

Example 5.4. Figure 4 exhibits a closed invariant circle appearing and unstability fixed point E^* with the set of parameter values $M = 100, \beta = 0.84, \nu = 0.265, \gamma = 0.1, \kappa = 0.3$ and takes $\delta = 3.78$ with the initial point $S(0) = 95$ and $I(0) = 5$.

For these parametric values, the controlled system (4.2) can be written as

$$\begin{aligned} S_{t+1} &= S_t + \alpha\delta\left(\nu\left(N - S_t - I_t\right) - \frac{\beta S_t I_t}{N}\right) \\ I_{t+1} &= I_t + \alpha\delta I_t\left(\frac{\beta S_t}{N} - \gamma - \kappa\right). \end{aligned} \tag{5.1}$$

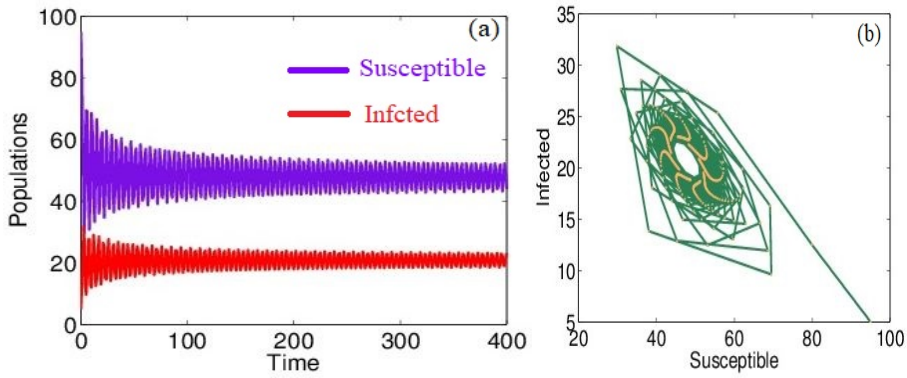

FIGURE 5. Time Series and Phase Plane for the Model (2.3)

where $M = 100, \beta = 0.84, \nu = 0.265, \gamma = 0.1, \kappa = 0.3$, $\delta = 3.78$ and $0 < \alpha < 1$. Jacobian of controlled model (5.1) evaluated at E_1 is $J(E_1) = \begin{bmatrix} 1 - 1.6647\alpha & -2.5137\alpha \\ 0.6630\alpha & 1 \end{bmatrix}$. The characteristic equation is $\lambda^2 - (2 - 1.6647)\lambda + 1.6666\alpha^2 + 1 - 1.6647\alpha = 0$. Then, the roots lie in the unit open disk if and only if $0 < \alpha < 0.9999$. Moreover, the plots for S and I of the controlled model (5.1) are exhibits in Figure 6 with $\alpha = 0.98$. It is clear that the endemic equilibrium E_1 is stable see Figure 6.

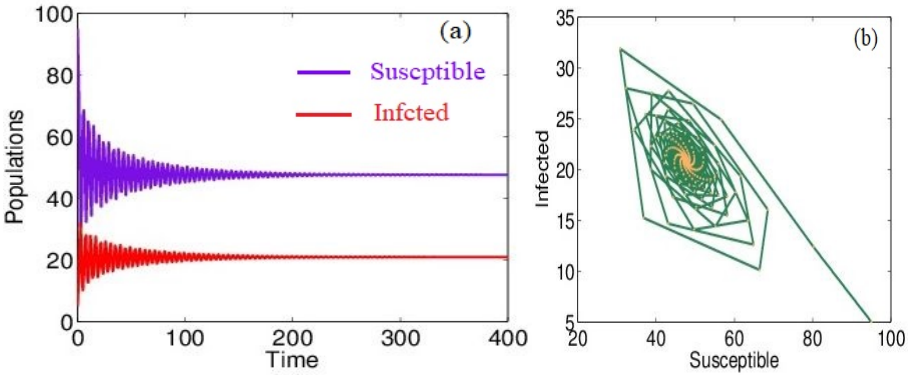

FIGURE 6. Time Series and Phase Plane for the Controlled Model (5.1)

6. CONCLUSION

COVID-19, a viral infectious disease that mainly occurs as fever and pneumonia, is known to give different symptoms depending on the person. In severe cases, antiviral and respiratory supportive therapies are the main treatment. Although there is no authorization by the FDA to use any drugs for the prevention or treatment of COVID-19, Remdesivir received an emergency use clearance from the FDA on May 1, 2020 based on preliminary data showing that hospitalized patients with severe disease have a faster

recovery time. From the perspective of clinical immunologists and rheumatologists, antiviral and supportive therapies are undoubtedly important in treating COVID-19 patients. While antiviral drugs containing hydroxyl-chloroquine and azithromycin are thought to be the best option to treat patients based on their presentation and symptoms, the main concern is that anti-inflammatory drugs such as corticosteroids may delay the elimination of the virus in those with compromised immune systems; and may increase the risk of secondary infection. At the same time, when studies on Covid-19 are examined, people produce a reasonable antibody response against the virus, but these antibodies decrease on most people in a short time and become undetectable in some cases. Since antibodies are the main defense feature of the immune system in fighting coronavirus, these findings suggest that they can re-infect people in seasonal waves and vaccines may not protect them for long.

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