Results in Physics 19 (2020) 103586

Contents lists available at ScienceDirect

Results in Physics

journal homepage: www.elsevier.com/locate/rinp

Analysis of the outbreak of the novel coronavirus COVID-19 dynamic model with control mechanisms

Fatma Bozkurt [a,b], Ali Yousef [a], Thabet Abdeljawad [c,d,e,*]

[a] Department of Mathematics, Kuwait College of Science and Technology, Kuwait City 27235, Kuwait
[b] Department of Mathematics, Erciyes University, Kayseri 38039, Turkey
[c] Department of Mathematics and General Sciences, Prince Sultan University, 11586 Riyadh, Saudi Arabia
[d] Department of Medical Research, China Medical University, Taichung 40402, Taiwan
[e] Department of Computer Science and Information Engineering, Asia University, Taichung, Taiwan

ARTICLE INFO

Keywords:
Differential equations with piecewise constant arguments
Stability
Covid-19
Allee effect

ABSTRACT

The mathematical models of infections are essential tools in understanding the dynamical behavior of disease transmission. In this paper, we establish a model of differential equations with piecewise constant arguments that explores the outbreak of Covid-19 including the control mechanisms such as health organizations and police supplements for the sake of controlling the pandemic spread and protecting the susceptible population. The local asymptotic stability of the equilibrium points, the disease-free equilibrium point, the apocalypse equilibrium point and the co-existing equilibrium point are analyzed by the aide of Schur-Cohn criteria.

Furthermore and by incorporating the Allee function at time t, we consider the extinction case of the outbreak to analyze the conditions for a *strong Allee Effect*. Our study has demonstarted that the awareness of the police personal and the management of professional health organizations play a vital role to protect the susceptible class and to prevent the spreading. Numerical simulations are presented to support our theoretical findings. We end the paper by a describtive conclusion.

Introduction

Origin and evolution of coronaviruses

Coronaviruses are the largest group of viruses belonging to *Nidovirales* order, which includes *coronaviridae, Arterividae*, and *Roniviridae* families. The *coronaviridae* divided into four groups: the $\alpha-$coronavirus, $\beta-$coronavirus, $\gamma-$ coronavirus, and $\delta-$coronavirus. These groups are sorted based on serology but recently divided in terms of phylogenetic clustering [1]. All viruses of *Nidovirales* order are enveloped, non-segmented positive-sense RNA viruses, where within this, the *coronaviridae* has the most significant identified RNA genomes, containing approximately 30 kilobases (kb) genomes. On the other hand, all coronaviruses have animal origin [2,3]. Table 1 below shows that the four genera of coronaviruses originated from animals.

These coronaviruses were not seen as highly pathogenic to humans until the outbreak of SARS-CoV in 2002 and MERS-CoV in 2012. Both of them have the genera of $\gamma-$ coronavirus. SARS-CoV and MERS-CoV were transmitted directly from civets and dromedary camels (intermediate hosts) to the human hosts, respectively, and both viruses showed the same natural host of bats in the epidemic spread. Table 2 shows the animal origin of human coronaviruses.

The HCoV-NL63 and HCoV-229E are $\alpha-$coronaviruses that cause mild infections in humans. On the other hand, SADS-CoV, which has swine as an intermediate host role, does not show any evidence of infections in humans. HCoV-OC43 and HCoV-HKU1 are both $\beta-$coronaviruses and are also mostly harmless to the human body that has a rodent-borne origin. HCoV-229E and HCoV-OC43 were isolated nearly 50 years ago, while HCoV-NL63 and HCoV-HKU1 were identified in 2003. The same year a coronavirus that was again bat-borne appeared in China, which is known as SARS-CoV. This virus was transmitted to humans through market civets. In 2015, people faced with another infection in the Middle East known as MERS-CoV, that spilled over to dromedary camels. These viruses demonstrated severe respiratory syndromes in humans, including fever, dizziness, and cough [4–8].

All coronaviruses mentioned above were endemic in the human populations causing 15–30% of respiratory tract infections each year. However, in December 2019, a novel coronaviridae was reported again

* Corresponding author at: Department of Mathematics and General Sciences, Prince Sultan University, 11586 Riyadh, Saudi Arabia.
E-mail address: tabdeljawad@psu.edu.sa (T. Abdeljawad).

https://doi.org/10.1016/j.rinp.2020.103586
Received 25 September 2020; Received in revised form 31 October 2020; Accepted 4 November 2020
Available online 17 November 2020
2211-3797/© 2020 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Table 1
Genera of CoV and the pathogenic class.

Coronavirinae Genera	Pathogenic Class
α-CoV	Mammals
β-CoV	Mammals
γ-CoV	both non-mammal and mammals
δ-CoV	both non-mammal and mammals

Table 2
Natural-Intermediate and Human Host Transmission.

Natural Host	Intermediate Host	Human Host
Bats	unknown	HCoV-NL63
Bats	Camelids	HCoV-229E
Rodent	mammals	HCoV-OC43
Rodent	unknown	HCoV-HKU1
Bats	Civets	SARS-CoV
Bats	Dromedary Camels	MERS-CoV
Bats	Swine	SADS-CoV

in China (Wuhan). The outbreak was associated with intermediate hosts like reptilians, while the natural host was assumed as bats. This virus was designated later as COVID-19 by the WHO (World Health Organization). COVID-19 was characterized by two members of β-coronavirus; the human-origin coronavirus (SARS-CoV Tor2) and bat-origin coronavirus (bat-SL-CoVZC45). Intensive studies show that it was most closely related to the bat-origin coronavirus [9]. Thus, the primary assumption was set up as the natural host of COVID-19 spread by infected bats of genus *Rhinolophus* that are mainly in the area of Shatan River Valley. Domestic animals like snakes in the area were hunted for the food market in Wuhan, which had an intermediate host role in the transmission. Finally, this virus spill-over from the intermediate hosts to cause several diseases in human. A virus that started with an endemic pathogenic behavior in China (Wuhan) reaches somehow to a pandemic point worldwide with infections from human-to-human [1,9].

Apocalypse in pandemic form of COVID-19 and the role of health organizations and police personals

In December 2019, a novel coronavirus appeared in China (Wuhan). A few months later, the virus tends to spread worldwide. On the 16th of March 2020, the WHO upgraded the status of COVID-19 from epidemic to pandemic. To prevent the spread of infections, several fundamental actions such as quarantine, culling, and social distancing, should be considered [10]. Health institutions took high responsibilities to engage the public with '*healthy practices*' like hand washing and staying mainly at home. At the same time, nowadays, police play a crucial role in controlling the panic of civilians and supporting health institutions. Until the 31st of October 2020, the number of infections reaches around 46,008,997 cases, with 1,195,477 deaths and 33,300,518 recovered. The USA has 9,318,653 total cases, and the death rate reaches to 235,182. After that, we see India with the highest rate of deaths, which reaches around 121,681 and 8,137,119 total cases. On the other side, since December 2019 China showed 85,973 total cases, with 4634 deaths [11]. The spread of COVID-19 was seen first in China, but it is interesting to notice that the rate of infections and fatalities increased rapidly in the US, Europe and other countries. Therefore, in our study, we consider the coordination mechanisms to keep the public aware of the infection and in engaging them about fundamental protections. Furthermore, we want to show the essential role of police personals in controlling the spread.

Mathematical model

Mathematical models are crucial tools in analyzing the pandemic spread of the infection through populations. Many research areas in biology or medicine attract applied mathematics since mathematical modeling is essential to understand the dynamics of diseases and biological phenomena. Over the years, anatomical models have been formulated mathematically using integer-order differential equations [12–14]. However, it is seen that many problems in biology, as well as in other fields like engineering, finance, and economics, can be described successfully by differential equations with piecewise constant arguments [15–17]. It is noticed that there are some dynamics in the environment, which combine the tools of both differential-difference equations concerning time t. For such biological events, it is preferred to construct models of differential equations with both continuous and discrete-time. We refer herein to some literature that considered discrete and continuous-time effects as differential equations with piecewise constant arguments [18–23].

We consider here the pandemic infection that occurs when the virus is replaced in the human body and continues to spread from human-to-human. The proposed model constitutes of five equations. The first three equations of the model show an SI (susceptible-infected) model to explain the transmission from human-to-human. Where S is the susceptible class, I is the infected class, that does not know they are infected because of the late appeared symptoms of COVID-19 and Cshows the infected class that knows they are infected. In equation four, H denotes the hospitalized people in a quarantined place. While in equation five, M shows the police population who are knowledgeable and supportive of the health institutes. All equations in the model show a form of Pierre-Francois Verhulst's logistic population model. The police represent a predator–prey model of Holling Type II, while their role is to realize and guide the infected civilians to hospitals safely.

Thus, the mathematical model of this biological phenomena has the form:

$$\begin{cases} \dfrac{dS}{dt} = S(t)(r_1(K_1 - \alpha_1 S(t) - \alpha_2 S([\![t]\!])) - \beta_1 I([\![t]\!]) - \gamma_1 C([\![t]\!]) - \eta_1 S([\![t]\!])) \\ \dfrac{dI}{dt} = I(t)(K_2 - \alpha_3 I(t) - \alpha_4 I([\![t]\!]) + \beta_1(1-\varepsilon_1) S([\![t]\!]) - \theta I([\![t]\!]) - \eta_2 I([\![t]\!]) + \beta_2(1-\varepsilon_2) M([\![t]\!])) \\ \dfrac{dC}{dt} = C(t)(K_3 - \alpha_5 C(t) - \alpha_6 C([\![t]\!]) + \beta_1\varepsilon_1 S([\![t]\!]) + \beta_2\varepsilon_2 M([\![t]\!]) + \theta I([\![t]\!]) + \gamma_1 S([\![t]\!]) + \gamma_2 M([\![t]\!]) \\ \qquad - f([\![t]\!]) - \mu H([\![t]\!]) - \eta_3 C([\![t]\!])) \\ \dfrac{dH}{dt} = H(t)(K_4 - \alpha_7 H(t) - \alpha_8 H([\![t]\!]) + \mu C([\![t]\!]) + f([\![t]\!]) C([\![t]\!]) - \eta_4 H([\![t]\!])) \\ \dfrac{dM}{dt} = M(t)(K_5 - \alpha_9 M(t) - \alpha_{10} M([\![t]\!]) - \beta_2 I([\![t]\!]) - \gamma_2 C([\![t]\!]) - \eta_5 M([\![t]\!])) \end{cases} \tag{2.1}$$

where and

$$f([\![\mathrm{t}]\!]) = \frac{\delta_1 \mathrm{M}([\![\mathrm{t}]\!])}{1 + h\delta_1 \mathrm{C}([\![\mathrm{t}]\!])} \tag{2.2}$$

represents the Holling Type II function. All the parameters belong to $\mathbb{R}^+$ and $[\![\mathrm{t}]\!]$ denotes the integer part of $\mathrm{t} \in [0, \infty)$.

The susceptible S is composed of individuals that have not contacted the infection but can get infected through contacts from the human that does not know they are infected. S can get infected through I, who does not know they are infected. It is also possible that the class S noticed they were in touch with an infected person in class C. r_1 is the population growth rate of the susceptible population. α_1 and α_2 denotes the logistic rates in discrete and continuous time, while K_1 is the carrying capacity of S per year. The susceptible lost their class following contacts with infectives I and C at a rate of β_1 and γ_1, respectively. η_1 shows the natural death rate of the susceptible class S.

The I class does not know that they have COVID-19 because of the late appeared symptoms of the infection. In this equation α_3 and α_4 denote the logistic rates, while K_2 is the carrying capacity of I.The population of this class decreases after being informed about the virus from the health organizations and becomes aware of doing screening at a rate θ. Another possibility is that after the S-I contact and $M-I$ contact, the symptoms occur in early stages so that these classes noticed that they are infected, which is given with the rates ε_1 and ε_2, respectively. For the infected groups, we consider only the death rate caused by the infection, which is denoted by the parameter η_2.

C is the infected COVID-19 class, that has logistic rates in a continuous and discrete-time of α_5 and α_6, respectively and the carrying capacity is given by K_3. The population of this class increases with $\beta_1\varepsilon_1$ and $\beta_2\varepsilon_2$ who noticed from early appeared symptoms that they are infected. Another parametric increase comes from θ when the test is positive. Moreover, any contact between S andC is realized by the susceptible class with γ_1 that increases the population of the infected C group. We assume that the police personals are aware of the virus and know to protect themselves. However, we cannot ignore the possibility that during any contact with infected civilians, there might be an infection transmission to the police. Even if we consider this rate as very low, we include this case in the dynamical spread with γ_2. The C class loses its population with the Holling Type II function, which is an act of the police to guide the infected civilians to the hospitals. As we mentioned before, the hard work conditions of health institutions need support from the police to manage the spread. This cooperative organization will decrease the rate of infections and control the spread. μ denotes the rate of the people who realize they are infected through screening or the symptoms and decide to stay in the hospital, while η_3 represents the infectious death rate.

H represents the hospitalization under quarantine in hospitals, where α_5 and α_6 are the rates of the logistic function, while K_4 is the carrying capacity. The population of H increases through the people who noticed they are infected and prefer to stay in the hospital during the treatment process. $f([\![\mathrm{t}]\!])$ of Holling Type II increase the quarantine population in H. η_4 is the death rate caused by the infection.

M is composed of police personals to control the pandemic spread. The logistic rates in continuous and discrete-time are given as α_9 and α_{10}. K_5 is the carrying capacity of the police. This population loses its class after any symptom appears that show COVID-19 with a rate of γ_2. Further, it has the possibility that M gets infected through someone who still does not know it has the virus, which is given as β_2. The natural death rate of this class is η_5.

The Holling Type II function contains parameters of δ_1 and$h$, where it shows the rate of action and the rate of average time to guide the infected class to the hospitals, respectively. Table 3 provides brief descriptions of the parameters of the model.

Table 3
Parametric description of the dynamical model.

Notation	Description of Parameter
K_1	Carrying capacity of the susceptible class
K_2	Carrying capacity of the infected group who do not know they are infected
K_3	Carrying capacity of the infected of COVID-19
K_4	Carrying capacity of individuals in hospitals
K_5	Carrying capacity of police personals
α_1,α_2	Logistic rate of the susceptible class
α_3,α_4	Logistic rate of the infected group who do not know they are infected
α_5,α_6	Logistic rate of the infected group
α_7,α_8	Logistic rate of the hospitalized population
α_9,α_{10}	Logistic rate of the police personals
β_1	Infection rate from the $S-I$ interaction
β_2	Infection rate from the M $-I$ interaction
γ_1	Infection rate from the $S-C$ interaction
γ_2	Infection rate from the M $-C$ interaction
ε_1, ε_2	Recognition of infection
θ	Rate of screening
μ	Transmission rate to hospitalized of infected civilians and the police
δ_1	Rate of action of Holling Type II
h	Rate of average time to guide the infected class to hospitals
η_1	The natural death rate of the susceptible class
η_2	The infectious death rate of the individuals of class$I(t)$
η_3	The infectious death rate of the individuals of class C(t)
η_4	The infectious death rate of the individuals of class H(t)
η_5	The natural death rate of the police

Stability of the equilibrium points

By integrating model (2.1) over $[n, t)$ and taking, $t \to n+1$, we get a system of difference equations as follows:

$$\begin{cases} S(n+1) = \dfrac{S(n)\psi_1(n)}{\alpha_1 \mathrm{r}_1 \mathrm{S(n)} + (\psi_1(n) - \alpha_1 \mathrm{r}_1 \mathrm{S(n)})e^{-\psi_1(n)}} \\ I(n+1) = \dfrac{I(n)\psi_2(n)}{\alpha_3 I(n) + (\psi_2(n) - \alpha_3 I(n))e^{-\psi_2(n)}} \\ C(n+1) = \dfrac{C(n)\psi_3(n)}{\alpha_5 C(n) + (\psi_3(n) - \alpha_5 C(n))e^{-\psi_3(n)}} \\ H(n+1) = \dfrac{H(n)\psi_4(n)}{\alpha_7 H(n) + (\psi_4(n) - \alpha_7 H(n))e^{-\psi_4(n)}} \\ M(n+1) = \dfrac{M(n)\psi_5(n)}{\alpha_9 M(n) + (\psi_5(n) - \alpha_9 M(n))e^{-\psi_5(n)}} \end{cases} \tag{3.1}$$

where

$$\psi_1(n) = \mathrm{r}_1(K_1 - \alpha_2 \mathrm{S(n)}) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1 \mathrm{S(n)}$$

$$\psi_2(n) = K_2 - (\alpha_4 + \theta + \eta_2)\mathrm{I(n)} + \beta_1(1-\varepsilon_1)S(\mathrm{n}) + \beta_2(1-\varepsilon_2)M(\mathrm{n}),$$

$$\psi_3(n) = K_3 - (\alpha_6 + \eta_3)\mathrm{C(n)} + (\beta_1\varepsilon_1 + \gamma_1)S(\mathrm{n}) + (\beta_2\varepsilon_2 + \gamma_2)M(\mathrm{n}) + \theta \mathrm{I(n)} - f(\mathrm{n}) - \mu H(\mathrm{n}),$$

$$\psi_4(n) = K_4 - (\alpha_8 + \eta_4)\mathrm{H(n)} + \mu C(\mathrm{n}) + f(\mathrm{n})\mathrm{C(n)},$$

$$\psi_5(n) = K_5 - (\alpha_{10} + \eta_5)\mathrm{M(n)} - \beta_2 I(\mathrm{n}) - \gamma_2 C(\mathrm{n}),$$

and$f(n) = \frac{\delta_1 \mathrm{M(n)}}{1 + h\delta_1 \mathrm{C(n)}}$.

Hereafter, any global analysis of system (3.1) represents the behavior of (2.1), since (3.1) is a solution of (2.1) for $t \in [n, n+1)$ First, we need to find the equilibria of system (3.1), which also represents the critical points of system (2.1). These three essential equilibrium points in our

study, which are epidemic free-equilibrium point, apocalypse equilibrium point, and co-existing equilibrium point. These are given as follows;

Epidemic- free equilibrium point:

$$\Lambda_1 = \left(\overline{S}_1, 0, 0, 0, \overline{M}_1\right) = \left(\frac{r_1 K_1}{(\alpha_1+\alpha_2)r_1+\eta_1}, 0, 0, 0, \frac{K_5}{\alpha_9+\alpha_{10}+\eta_5}\right)$$

Apocalypse equilibrium point:

$$\Lambda_2 = \left(0, \overline{I}_2, \overline{C}_2, \overline{H}_2, 0\right) = \left(0, \frac{K_2}{\alpha_3+\alpha_4+\theta+\eta_2}, \frac{(\alpha_7+\alpha_8+\eta_4)\left(K_3+\frac{\theta K_2}{\alpha_3+\alpha_4+\theta+\eta_2}\right)-\mu\ (\alpha_5+\alpha_6+\eta_3)+\boldsymbol{\mu}\left(K_4+\frac{\theta K_2}{\alpha_3+\alpha_4+\theta+\eta_2}\right)}{\mu^2+(\alpha_5+\alpha_6+\eta_3)(\alpha_7+\alpha_8+\eta_4)}, \frac{}{\mu^2+(\alpha_5+\alpha_6+\eta_3)(\alpha_7+\alpha_8+\eta_4)}, 0\right)$$

$$\mu < (\alpha_7+\alpha_8+\eta_4)\left(K_3+\frac{\theta K_2}{\alpha_3+\alpha_4+\theta+\eta_2}\right)$$

Co-existing equilibrium point: $\Lambda_3 = \left(\overline{S}_3, \overline{I}_3, \overline{C}_3, \overline{H}_3, \overline{M}_3\right)$, where all classes are positive.

The Jacobian matrix around $\Lambda_1 = \left(\overline{S}_1, 0, 0, 0, \overline{M}_1\right)$ is given by

$$J(\Lambda_1) = \begin{pmatrix} a_{11} & a_{12} & a_{13} & 0 & 0 \\ 0 & a_{22} & 0 & 0 & 0 \\ 0 & 0 & a_{33} & 0 & 0 \\ 0 & 0 & 0 & a_{44} & 0 \\ 0 & a_{52} & a_{53} & 0 & a_{55} \end{pmatrix} \tag{3.2}$$

where

$$a_{11} = \frac{-(\alpha_2 r_1+\eta_1)+(\alpha_1 r_1+\alpha_2 r_1+\eta_1)e^{-\overline{\psi}_1}}{\alpha_1 r_1}, a_{12} = \frac{-\beta_1(1-e^{-\overline{\psi}_1})}{\alpha_1 r_1}, a_{13} = \frac{-\gamma_1(1-e^{-\overline{\psi}_1})}{\alpha_1 r_1}, a_{22} = e^{\overline{\psi}_2}$$

and

$$a_{33} = e^{\overline{\psi}_3}, a_{44} = e^{\overline{\psi}_4}, a_{52} = \frac{-\beta_2\left(1-e^{-\overline{\psi}_5}\right)}{\alpha_9}, a_{53} = \frac{-\gamma_2\left(1-e^{-\overline{\psi}_5}\right)}{\alpha_9}, a_{55} = \frac{-(\alpha_{10}+\eta_5)+(\alpha_9+\alpha_{10}+\eta_5)e^{-\overline{\psi}_5}}{\alpha_9}$$

The characteristic equation of the matrix (3.2) around the disease-free equilibrium point is given as;

$$(a_{11}-\lambda)(a_{22}-\lambda)(a_{33}-\lambda)(a_{44}-\lambda)(a_{55}-\lambda) = 0. \tag{3.3}$$

Theorem 3.1. *Let Λ_1 be the epidemic free equilibrium point of system (3.1). The following statements are true.*

(i) If

$$\overline{S}_1 \in \left(0, \frac{r_1 K_1}{\alpha_2 r_1+\eta_1}\right) \text{and} \overline{M}_1 \in \left(0, \frac{K_5}{\alpha_{10}+\eta_5}\right), \tag{3.4}$$

where $\delta_1 > \beta_2\varepsilon_2+\gamma_2$ and $K_5 < \frac{(\alpha_{10}+\eta_5)K_3}{\delta_1-\beta_2\varepsilon_2-\gamma_2}$. Then, system (3.1) is a local asymptotically stable for the $S-M$ class, while the remaining classes of infected individuals are unstable.

(ii) Let $K_2 = K_4 = 1$, $\varepsilon_1 = \varepsilon_2 = 1$ and $\delta_1 = \beta_2\varepsilon_2+\gamma_2$. If

$$\overline{S}_1 = \frac{r_1 K_1}{\alpha_2 r_1+\eta_1} \text{and} \overline{M}_1 = \frac{K_5}{\alpha_{10}+\eta_5} \tag{3.5}$$

$K_3 = 1 - \frac{(\beta_1+\gamma_1)r_1 K_1}{\alpha_2 r_1+\eta_1}$ and $K_1 < \frac{\alpha_2 r_1+\eta_1}{(\beta_1+\gamma_1)r_1}$, then Λ_1 of system (3.1) is non-hyperbolic.

Proof. ***We prove (i).*** *Let*

(a) $\lambda_1 = \frac{-(\alpha_2 r_1+\eta_1)+(\alpha_1 r_1+\alpha_2 r_1+\eta_1)e^{-\overline{\psi}_1}}{\alpha_1 r_1}$

(b) $\lambda_2 = e^{\overline{\psi}_2}$

(c) $\lambda_3 = e^{\overline{\psi}_3}$

(d) $\lambda_4 = e^{\overline{\psi}_4}$

(e) $\lambda_5 = \frac{-(\alpha_{10}+\eta_5)+(\alpha_9+\alpha_{10}+\eta_5)e^{-\overline{\psi}_5}}{\alpha_9}$.

(i) From (a) we obtain

$|\lambda_1| < 1 \Rightarrow \frac{-(\alpha_2 r_1+\eta_1)+(\alpha_1 r_1+\alpha_2 r_1+\eta_1)e^{-\overline{\psi}_1}}{\alpha_1 r_1} < 1 \Rightarrow \overline{\psi}_1 > 0$, which implies that $\overline{S}_1 \in \left(0, \frac{r_1 K_1}{\alpha_2 r_1+\eta_1}\right)$

We get from (b) the inequality $|\lambda_2| > 1 \Rightarrow e^{\overline{\psi}_2} > 1$, since $\overline{\psi}_2 > 0$. Considering (c), we have

$|\lambda_3| > 1 \Rightarrow e^{\overline{\psi}_3} > 1$

if $\delta_1 > \beta_2\varepsilon_2+\gamma_2$ and

$$\overline{M}_1 \in \left(0, \frac{K_3}{\delta_1-\beta_2\varepsilon_2-\gamma_2}\right) \tag{3.6}$$

From (d), we obtain

$|\lambda_4| > 1 \Rightarrow e^{\overline{\psi}_4} > 1 \Rightarrow \overline{\psi}_4 > 0.$

Finally, from the inequality in (e), we get

$|\lambda_5| < 1 \Rightarrow \frac{-(\alpha_{10}+\eta_5)+(\alpha_9+\alpha_{10}+\eta_5)e^{-\overline{\psi}_5}}{\alpha_9} < 1 \Rightarrow \overline{\psi}_5 > 0$, which implies that

$$\overline{M}_1 \in \left(0, \frac{K_5}{\alpha_{10}+\eta_5}\right) \tag{3.7}$$

Considering both (3.6) and (3.7) together, we obtain $K_5 < \frac{(\alpha_{10}+\eta_5)K_3}{\delta_1-\beta_2\varepsilon_2-\gamma_2}$. This completes the proof of (i).

(ii) Considering (a), we have

$|\lambda_1| = 1 \Rightarrow \frac{-(\alpha_2 r_1+\eta_1)+(\alpha_1 r_1+\alpha_2 r_1+\eta_1)e^{-\overline{\psi}_1}}{\alpha_1 r_1} = 1 \Rightarrow \overline{\psi}_1 = 0$, which is

$$\overline{S}_1 = \frac{r_1 K_1}{\alpha_2 r_1+\eta_1} \tag{3.8}$$

Thus, from the disease-free equilibrium point of the susceptible class, we obtain $\alpha_1 = 0$. This means that a non-hyperbolic behavior in the susceptible class happens if the logistic rate is only dependent on the discrete-time of t. Similarly, from (d) we obtain that $|\lambda_5| = 1$, if

$$\overline{M}_1 = \frac{K_5}{\alpha_{10}+\eta_5}, \tag{3.9}$$

then the equilibrium of class M is non-hyperbolic if the logistic rate is dependent only on the discrete-time of t. On the other side, if the recognition of the infection reaches $\varepsilon_1 = \varepsilon_2 = 1$ and the carrying capacity of class I is $K_2 = 1$, then $|\lambda_2| = 1$. Similarly, we have $|\lambda_4| = 1$ if $K_4 = 1$. Finally, we obtain that $|\lambda_3| = 1$, if

$$K_3 = 1 - \frac{(\beta_1 + \gamma_1)\mathrm{r}_1 K_1}{\alpha_2 \mathrm{r}_1 + \eta_1} \text{and} K_1 < \frac{\alpha_2 \mathrm{r}_1 + \eta_1}{(\beta_1 + \gamma_1)\mathrm{r}_1} \tag{3.10}$$

where $\delta_1 = \beta_2\varepsilon_2 + \gamma_2$. This completes the proof of (ii).

Remark 3.1. *Theorem* 3.1(*i*) *shows that S and M increase, if there is no loss of the classes through any infectional transmission. The remaining classes, which are I, C, and H have unstable behaviors, since the rate of action of Holling Type II is higher than the interaction rate of the infected classes to class M. This means that the police are highly aware of the transmission potential to reduce any of the infection possibilities.*

Theorem 3.1(ii) shows a non-hyperbolic behavior of system (3.1) around the disease-free equilibrium point. The susceptible class and the police are non-hyperbolic for the logistic rates in discrete-time t. If the recognition of infection is high in class I and C, and if the carrying capacity of the classes who do not know they are infected is low like the class of the hospitalized one, then both I and C classes are non-hyperbolic. The rate of action of Holling Type II is almost equal to the rate of infectives of class I and C to class$M$.

Thus, the theoretical results show that for any negligence of Sas well as M in '*healthy practices*,' both non-infected classes could reach to a non-hyperbolic behavior. The duties should have continuity in the management and control of civilians. In Theorem 3.1(ii), we emphasize that the main problem occurs in health protections if it is *not continuous*.

The Jacobian matrix around the apocalypse equilibrium point $\Lambda_2 = \left(0, \bar{I}_2, \bar{C}_2, \bar{H}_2, 0\right)$ is given as follows:

$$J(\Lambda_2) = \begin{pmatrix} a_{11} & 0 & 0 & 0 & 0 \\ a_{21} & a_{22} & 0 & 0 & a_{25} \\ a_{31} & a_{32} & a_{33} & a_{34} & a_{35} \\ 0 & 0 & a_{43} & a_{44} & a_{45} \\ 0 & 0 & 0 & 0 & a_{55} \end{pmatrix} \tag{3.11}$$

where

$$a_{11} = e^{\overline{\psi}_1}, a_{55} = e^{\overline{\psi}_5}, a_{21} = \frac{\beta_1(1-\varepsilon_1)(1-e^{-\overline{\psi}_2})}{\alpha_3}, a_{22} = \frac{-(\alpha_4+\theta+\eta_2)+(\alpha_3+\alpha_4+\theta+\eta_2)e^{-\overline{\psi}_2}}{\alpha_3}, a_{25} = \frac{\beta_2(1-\varepsilon_2)(1-e^{-\overline{\psi}_2})}{\alpha_3},$$

$$a_{31} = \frac{(\beta_1\varepsilon_1+\gamma_1)(1-e^{-\overline{\psi}_3})}{\alpha_5}, a_{32} = \frac{\theta(1-e^{-\overline{\psi}_3})}{\alpha_5}, a_{33} = \frac{-(\alpha_6+\eta_3)+(\alpha_5+\alpha_6+\eta_3)e^{-\overline{\psi}_3}}{\alpha_5}, a_{34} = \frac{-\mu(1-e^{-\overline{\psi}_3})}{\alpha_5}$$

$$a_{35} = \frac{\left(\beta_2\varepsilon_2+\gamma_2-\frac{\delta_1}{1+h\delta_1\bar{C}_2}\right)(1-e^{-\overline{\psi}_3})}{\alpha_5}, a_{43} = \frac{\mu(1-e^{-\overline{\psi}_4})}{\alpha_7}, a_{44} = \frac{-(\alpha_8+\eta_4)+(\alpha_7+\alpha_8+\eta_4)e^{-\overline{\psi}_4}}{\alpha_7}, a_{45} = \frac{\frac{\delta_1\bar{C}_2}{1+h\delta_1\bar{C}_2}(1-e^{-\overline{\psi}_4})}{\alpha_7}.$$

The characteristic equation of (3.11) is given by

$$(a_{11}-\lambda)(a_{22}-\lambda)(a_{55}-\lambda)\{(a_{33}-\lambda)(a_{44}-\lambda)-a_{34}a_{43}\} = 0. \tag{3.12}$$

Theorem 3.2. (*(Linearized Stability Theorem [22])*) Let

$$y_{n+1} = f(y_n, y_{n-1}), n = 0, 1, 2, \cdots \tag{3.13}$$

where for $p, q \in \mathbb{R}^+$. Then, the characteristic equation is

$$\lambda^2 - p\lambda - q = 0, \tag{3.14}$$

and the initial conditions are $y_{-1}, y_0 \in \mathbb{R}^+$.

If $|\lambda_{1,2}| < 1$, then the equilibrium $\bar{y}$ of equation (3.13) is locally asymptotically stable. Thus, $|\lambda_{1,2}| < 1$ if and only if

$$|p| < 1 - q < 2. \tag{3.15}$$

Theorem 3.3. *Let Λ_2 be the apocalypse equilibrium point of system* (3.1). *The following statements are true.*

(i) Assume that $R_0 < 1$, $\mathrm{r}_1 < \frac{\beta_1\bar{I}_2+\gamma_1\bar{C}_2}{K_1}$, $\bar{I}_2 < \frac{K_2}{(\alpha_4+\theta+\eta_2)}$, $\alpha_8 + \eta_4 > \mu$and $K_5 < \beta_2 I(\mathrm{n}) + \gamma_2 C(\mathrm{n})$ If

$$\overline{\psi}_3 < ln\left(\frac{(\alpha_5+\alpha_6+\eta_3)(1-R_0)}{(\alpha_6+\eta_3)(1-R_0)+\alpha_5}\right) \text{and} \overline{\psi}_4 > ln\left(\frac{\alpha_7+\alpha_8+\eta_4-\mu}{\alpha_8+\eta_4-\mu}\right), \tag{3.16}$$

then Λ_2 is a local asymptotically stable.

(ii) Assume that $R_0 > 1$, $\mathrm{r}_1 = \frac{\beta_1\bar{I}_2+\gamma_1\bar{C}_2}{K_1}$, $\bar{I}_2 = \frac{K_2}{(\alpha_4+\theta+\eta_2)}$, $\alpha_8 + \eta_4 > \mu$and $K_5 = \beta_2 I(\mathrm{n}) + \gamma_2 C(\mathrm{n})$ If

$$\overline{\psi}_3 \in \left(ln\left(\frac{\alpha_5+\alpha_6+\eta_3+\mu}{\alpha_6+\eta_3+\mu}\right), ln\left(\frac{\alpha_5+\alpha_6+\eta_3}{\alpha_6+\eta_3}\right)\right) \text{and} \overline{\psi}_4 \in \left(ln\left(\frac{\alpha_7+\alpha_8+\eta_4}{\alpha_8+\eta_4}\right), ln\left(\frac{\alpha_7+\alpha_8+\eta_4-\mu}{\alpha_8+\eta_4-\mu}\right)\right) \tag{3.17}$$

where

$$\overline{\psi}_3 + \overline{\psi}_4 = ln\left(\frac{(\alpha_5+\alpha_6+\eta_3)(\alpha_7+\alpha_8+\eta_4)(R_0-1)}{\alpha_5\alpha_7+(\alpha_8+\eta_4)\alpha_5}\right) \tag{3.18}$$

and $R_0 = \frac{\alpha_5+\alpha_6+\alpha_7+\alpha_8+\eta_3+\eta_4}{\alpha_6+\alpha_8+\eta_3+\eta_4}$, then Λ_2 of system (3.1) is a non-hyperbolic.

Proof.. (*(i)*) *From* (3.12), *we have the following*:

(a) $|\lambda_1| = e^{\overline{\psi}_1} < 1$, if

$$\mathrm{r}_1 < \frac{\beta_1\bar{I}_2+\gamma_1\bar{C}_2}{K_1}. \tag{3.19}$$

(b) $|\lambda_2| = \frac{-(\alpha_4+\theta+\eta_2)+(\alpha_3+\alpha_4+\theta+\eta_2)e^{-\overline{\psi}_2}}{\alpha_3} < 1$, if

$$\bar{I}_2 < \frac{K_2}{(\alpha_4+\theta+\eta_2)} \tag{3.20}$$

(c) $|\lambda_5| = e^{\overline{\psi}_5} < 1$, if

$$K_5 < \beta_2 I(\mathrm{n}) + \gamma_2 C(\mathrm{n}) \tag{3.21}$$

Moreover, from (3.12) we obtain a quadratic polynomial such as

$$\lambda^2 - (a_{33}+a_{44})\lambda + a_{33}a_{44}\left(1-\frac{a_{34}a_{43}}{a_{33}a_{44}}\right) = 0 \Rightarrow \lambda^2 - (a_{33}+a_{44})\lambda + a_{33}a_{44}(1-R_0) = 0 \tag{3.22}$$

where

$$R_0 = \frac{a_{34}a_{43}}{a_{33}a_{44}} \tag{3.23}$$

is the basic reproduction number of (3.22), which shows the potential transmission of the $C-H$ classes.

First of all, we want to obtain the conditions for $R_0 < 1$ to control the spread of infections. Thus, we obtain that R_0 is less than one, if

$$R_0 = \frac{a_{34}a_{43}}{a_{33}a_{44}} = \frac{\mu^2(1-e^{-\overline{\psi}_3})(1-e^{-\overline{\psi}_4})}{(-(\alpha_6+\eta_3)+(\alpha_5+\alpha_6+\eta_3)e^{-\overline{\psi}_3})((\alpha_8+\eta_4)-(\alpha_7+\alpha_8+\eta_4)e^{-\overline{\psi}_4})} < 1. \tag{3.24}$$

The denominator of (3.24) is positive when

$$\overline{\psi}_4 > ln\left(\frac{\alpha_7+\alpha_8+\eta_4}{\alpha_8+\eta_4}\right) \text{and} \overline{\psi}_3 < ln\left(\frac{\alpha_5+\alpha_6+\eta_3}{\alpha_6+\eta_3}\right) \tag{3.25}$$

Additionally, from (3.24), we have

$$\overline{\psi}_3 < ln\left(\frac{\alpha_5+\alpha_6+\eta_3+\mu}{\alpha_6+\eta_3+\mu}\right) \text{and} \overline{\psi}_4 > ln\left(\frac{\alpha_7+\alpha_8+\eta_4-\mu}{\alpha_8+\eta_4-\mu}\right) \tag{3.26}$$

Considering both (3.25) and (3.26), we obtain

$$\overline{\psi}_3 < ln\left(\frac{\alpha_5+\alpha_6+\eta_3+\mu}{\alpha_6+\eta_3+\mu}\right) < ln\left(\frac{\alpha_5+\alpha_6+\eta_3}{\alpha_6+\eta_3}\right) \text{and} \overline{\psi}_4 > ln\left(\frac{\alpha_7+\alpha_8+\eta_4-\mu}{\alpha_8+\eta_4-\mu}\right) > ln\left(\frac{\alpha_7+\alpha_8+\eta_4}{\alpha_8+\eta_4}\right), \tag{3.27}$$

where $\alpha_8+\eta_4 > \mu$. Applying the Linearized Stability Theorem to (3.22), we have

$$|a_{33}+a_{44}| < 1 + a_{33}a_{44}(1-R_0) < 2 \tag{3.28}$$

It is obvious that

$$a_{33}a_{44}(1-R_0) < 1,$$

since $R_0 < 1$, $a_{33} > 0$ and $a_{44} < 0$. Moreover, from (3.22), we can write

$$(\alpha_7+\alpha_8+\eta_4)\{(\alpha_6+\eta_3)(1-R_0)+\alpha_5\}e^{-\overline{\psi}_4} + (\alpha_5+\alpha_6+\eta_3)\{(\alpha_8+\eta_4)(1-R_0)+\alpha_7\}e^{-\overline{\psi}_3}$$

$$< \alpha_5\alpha_7 + (\alpha_6+\eta_3)\alpha_7 + \alpha_5(\alpha_8+\eta_4) + (\alpha_6+\eta_3)(\alpha_8+\eta_4)(1-R_0) + (\alpha_5+\alpha_6+\eta_3)(\alpha_7+\alpha_8+\eta_4)(1-R_0)e^{-\overline{\psi}_3}e^{-\overline{\psi}_4},$$

which holds for

$$\overline{\psi}_3 < ln\left(\frac{(\alpha_5+\alpha_6+\eta_3)(1-R_0)}{(\alpha_6+\eta_3)(1-R_0)+\alpha_5}\right) < ln\left(\frac{(\alpha_5+\alpha_6+\eta_3)\{(\alpha_8+\eta_4)(1-R_0)+\alpha_7\}}{(\alpha_6+\eta_3)(\alpha_8+\eta_4)(1-R_0)}\right) \tag{3.29}$$

Considering both (3.27) and (3.29), we obtain

$$\overline{\psi}_3 < ln\left(\frac{(\alpha_5+\alpha_6+\eta_3)(1-R_0)}{(\alpha_6+\eta_3)(1-R_0)+\alpha_5}\right) < ln\left(\frac{\alpha_5+\alpha_6+\eta_3+\mu}{\alpha_6+\eta_3+\mu}\right), \tag{3.30}$$

which completes the proof of (i).

(ii) To have a nonhyperbolic behavior, let us consider (3.12). Thus

(a) $|\lambda_1| = e^{\overline{\psi}_1} = 1$, if

$$\mathrm{r}_1 = \frac{\beta_1\overline{I}_2 + \gamma_1\overline{C}_2}{K_1}. \tag{3.31}$$

(b) $|\lambda_2| = \frac{-(\alpha_4+\theta+\eta_2)+(\alpha_3+\alpha_4+\theta+\eta_2)e^{-\overline{\psi}_2}}{\alpha_3} = 1$, if

$$\overline{I}_2 = \frac{K_2}{(\alpha_4+\theta+\eta_2)} \tag{3.32}$$

(c) $|\lambda_5| = e^{\overline{\psi}_5} = 1$, if

$$K_5 = \beta_2 I(\mathrm{n}) + \gamma_2 C(\mathrm{n}) \tag{3.33}$$

On the other side, conditions for $R_0 > 1$ implies that

$$R_0 = \frac{\mu^2(1-e^{-\overline{\psi}_3})(1-e^{-\overline{\psi}_4})}{(-(\alpha_6+\eta_3)+(\alpha_5+\alpha_6+\eta_3)e^{-\overline{\psi}_3})((\alpha_8+\eta_4)-(\alpha_7+\alpha_8+\eta_4)e^{-\overline{\psi}_4})} > 1, \tag{3.34}$$

if

$$ln\left(\frac{\alpha_5+\alpha_6+\eta_3+\mu}{\alpha_6+\eta_3+\mu}\right) < \overline{\psi}_3 < ln\left(\frac{\alpha_5+\alpha_6+\eta_3}{\alpha_6+\eta_3}\right) \text{and} ln\left(\frac{\alpha_7+\alpha_8+\eta_4}{\alpha_8+\eta_4}\right) < \overline{\psi}_4 < ln\left(\frac{\alpha_7+\alpha_8+\eta_4-\mu}{\alpha_8+\eta_4-\mu}\right), \tag{3.35}$$

where $\alpha_8+\eta_4 > \mu$. Moreover, from the quadratic form in (3.22), we obtain

$$\overline{\psi}_3 + \overline{\psi}_4 = ln\left(\frac{(\alpha_5+\alpha_6+\eta_3)(\alpha_7+\alpha_8+\eta_4)(R_0-1)}{\alpha_5\alpha_7+(\alpha_8+\eta_4)\alpha_5}\right), \tag{3.36}$$

where $R_0 = \frac{\alpha_5+\alpha_6+\alpha_7+\alpha_8+\eta_3+\eta_4}{\alpha_6+\alpha_8+\eta_3+\eta_4}$. This completes the proof of (ii).

Remark 3.2. *Theorem* 3.3. *shows the local stability and non-hyperbolic criteria for the apocalypse equilibrium point* Λ_2. *Here we consider the case where neither the civilian nor the police personal are non-infected. In this situation, the positive equilibria exist only for the following infected classes. First, who do not know they are infected. Second, who is determined by COVID-19. Third, who is transferred to the hospitals. As a result, in Theorem* 3.3(*i*), *we obtained the conditions for a stable dynamical behavior of* Λ_2. *The basic reproduction number is considered for* $R_0 < 1$. *The apocalypse case occurred when the growth rate of the susceptible class becomes less than the infectious transmission rate from class I and C. This means that the susceptible class ignored the transmission risks and continue to be a host for the infection. Moreover, there was not enough prepared police personal for an epidemic or infection cases so that the control mechanisms did not work as expected. Therefore, even when the basic reproduction number is still less than one, professional human actions can prevent the spread of the infection. This scenario is considered for a case when the contribution of civilians and organizations are weak. Theorem* 3.3(*ii*) *shows the criteria when* Λ_2 *has a non-hyperbolic behavior, where* $R_0 > 1$. *We emphasized in this part that the basic reproduction number is depended on the logistic rates of the infected class C and the hospitalized class H for both discrete and continuous times, respectively. Additionally, the death rate caused by the infection is also a parametric value that affects the reproduction number in this part. Further, in this scenario, the susceptible class is still not active in keeping the rules, and they continue the have a host role in the spread of COVID-19. Despite that the capacity of the police personals increases according to the rate of infections, it does not make sense if they are not prepared for infection cases. Thus, while the police personals are trying to work on the field to keep the control of panic, also this class works after a time as a host group to spread the infection.*

The Jacobian matrix of the *co-existing equilibrium point* $\Lambda_3 = \left(\overline{S}_3, \overline{I}_3, \overline{C}_3, \overline{H}_3, \overline{M}_3\right)$ is given by

$$J(\Lambda_3)=\begin{pmatrix} a_{11} & a_{12} & a_{13} & 0 & 0\\ 0 & a_{22} & 0 & 0 & 0\\ a_{31} & a_{32} & a_{33} & a_{34} & a_{35}\\ 0 & 0 & a_{43} & a_{44} & a_{45}\\ 0 & a_{52} & 0 & 0 & a_{55}\end{pmatrix}, \tag{3.37}$$

where

$$\begin{aligned}
a_{11} &= \frac{-(\alpha_2 r_1+\eta_1)+(\alpha_1 r_1+\alpha_2 r_1+\eta_1)e^{-\overline{\psi}_1}}{\alpha_1 r_1}, a_{12}=\frac{-\beta_1(1-e^{-\overline{\psi}_1})}{\alpha_1 r_1}, a_{13}\\
&= \frac{-\gamma_1(1-e^{-\overline{\psi}_1})}{\alpha_1 r_1}, a_{22}=\frac{-(\alpha_4+\theta+\eta_2)+(\alpha_3+\alpha_4+\theta+\eta_2)e^{-\overline{\psi}_2}}{\alpha_3}, a_{31}\\
&= \frac{(\beta_1+\gamma_1)(1-e^{-\overline{\psi}_3})}{\alpha_5}, a_{32}=\frac{\theta(1-e^{-\overline{\psi}_3})}{\alpha_5}, a_{33}\\
&= \frac{-\left(\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)+\left(\alpha_5+\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)e^{-\overline{\psi}_3}}{\alpha_5}, a_{34}\\
&= \frac{-\mu(1-e^{-\overline{\psi}_3})}{\alpha_5}, a_{35}=\frac{\left(\beta_2-\frac{\delta_1}{1+h\delta_1\overline{C}_3}\right)(1-e^{-\overline{\psi}_3})}{\alpha_5}, a_{43}\\
&= \frac{\left(\mu+\frac{\delta_1\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)(1-e^{-\overline{\psi}_4})}{\alpha_7}, a_{44}\\
&= \frac{-(\alpha_8+\eta_4)+(\alpha_7+\alpha_8+\eta_4)e^{-\overline{\psi}_4}}{\alpha_7}, a_{45}=\frac{\frac{\delta_1\overline{C}_3}{1+h\delta_1\overline{C}_3}(1-e^{-\overline{\psi}_4})}{\alpha_7}, a_{52}\\
&= \frac{-\beta_2\left(1-e^{-\overline{\psi}_5}\right)}{\alpha_9}, a_{55}=\frac{-(\alpha_{10}+\eta_5)+(\alpha_9+\alpha_{10}+\eta_5)e^{-\overline{\psi}_5}}{\alpha_9}
\end{aligned}$$

$$\overline{\psi}_1\in\left(ln\left(\frac{(\alpha_1 r_1+\alpha_2 r_1+\eta_1)\left(\alpha_5+\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)+(\beta_1+\gamma_1)\gamma_1}{(\alpha_2 r_1+\eta_1)\left(\alpha_5+\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)+(\beta_1+\gamma_1)\gamma_1}\right), ln\left(\frac{\alpha_1 r_1+\alpha_2 r_1+\eta_1}{\alpha_2 r_1+\eta_1}\right)\right), \tag{3.40}$$

Due to the intensive information through WHO and the media, the scenario of the co-existing equilibrium point is constructed so that people are aware of the symptoms of COVID-19. Thus, we have $\varepsilon_1 = \varepsilon_2 = 1$. Moreover, in this scenario, the police personal is educated about the transmission risks, and therefore, there is no transmission contact through any infected person, who has identified already as COVID-19 host. This means that $\gamma_2 = 0$. However, still, there is a possibility that the police might be infected from the class who does not know they are infected.

The characteristic equation of (3.37) is as follows:

$$\begin{aligned}
&(a_{22}-\lambda)(a_{55}-\lambda)\left\{\lambda^3-(a_{11}+a_{33}+a_{44})\lambda^2+(a_{11}a_{33}+a_{11}a_{44}-a_{34}a_{43}\right.\\
&\left.-a_{31}a_{13})\lambda-a_{11}a_{33}a_{44}+a_{31}a_{13}a_{44}\right\}\\
&=0
\end{aligned} \tag{3.38}$$

Theorem 3.4. [23]. *The characteristic polynomial*

$$P(\lambda)=\lambda^3+a_2\lambda^2+a_1\lambda+a_0 \tag{3.39}$$

has all its roots inside $|\lambda|<1$ if and only if

(i) $P(1)>0$ and $(-1)^3P(-1)>0$.
(ii) $D_2^+ = 1+a_1-{a_0}^2-a_0a_2>0$.
(iii) $D_2^- = 1-a_1-{a_0}^2+a_0a_2>0$. □

Theorem 3.5. *Let Λ_3 be the co-existing equilibrium point of system (3.1) and assume that*

$\bar{I}_3<\frac{K_2}{\alpha_4+\theta+\eta_2}$ and $K_5>(\alpha_{10}+\eta_5)\overline{M}_3+\beta_2\bar{I}_3$. If

$$\overline{\psi}_3\in\left(0, ln\left(\frac{(\alpha_5+\alpha_6+\eta_3)\left(1+h\delta_1\overline{C}_3\right)^2-{\delta_1}^2 h\overline{M}_3}{(\alpha_6+\eta_3)\left(1+h\delta_1\overline{C}_3\right)^2-{\delta_1}^2 h\overline{M}_3}\right)\right), \tag{3.41}$$

and

$$\overline{\psi}_4\in\left(0, ln\left(\frac{\alpha_7+\alpha_8+\eta_4}{\alpha_8+\eta_4}\right)\right), \tag{3.42}$$

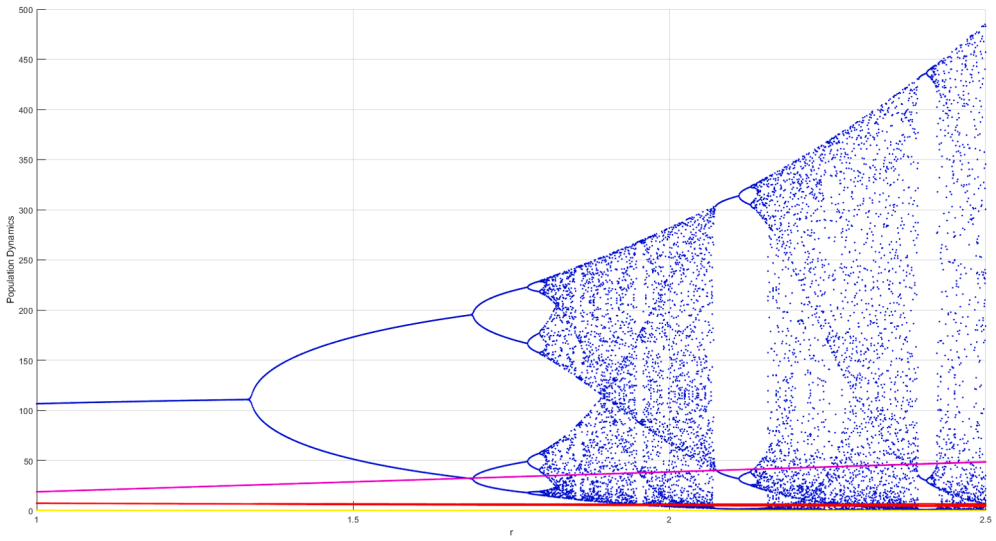

Fig. 1. Dynamical behavior of the population classes.

where $\overline{M}_3 < \frac{(\alpha_6+\eta_3)\left(1+h\delta_1\overline{C}_3\right)^2}{\delta_1{}^2h}$ and $r_1 > \frac{(\beta_1+\gamma_1)\gamma_1}{\alpha_1\alpha_5}$. Then the positive equilibrium point is local asymptotically stable.

Proof. *From the Schur-Cohn Criteria in Theorem* 3.4(*i*), *we can write*

$$1 + a_{11}a_{33} + a_{11}a_{44} - a_{34}a_{43} - a_{31}a_{13} > 0, \tag{3.43}$$

which holds for

$$\overline{\psi}_1 < ln\left(\frac{\alpha_1 r_1 + \alpha_2 r_1 + \eta_1}{\alpha_2 r_1 + \eta_1}\right) \tag{3.44}$$

$$\overline{\psi}_3 < ln\left(\frac{(\alpha_5+\alpha_6+\eta_3)\left(1+h\delta_1\overline{C}_3\right)^2 - \delta_1{}^2h\overline{M}_3}{(\alpha_6+\eta_3)\left(1+h\delta_1\overline{C}_3\right)^2 - \delta_1{}^2h\overline{M}_3}\right) \text{for} \overline{M}_3$$
$$< \frac{(\alpha_6+\eta_3)\left(1+h\delta_1\overline{C}_3\right)^2}{\delta_1{}^2h} \tag{3.45}$$

and

$$\overline{\psi}_4 < ln\left(\frac{\alpha_7+\alpha_8+\eta_4}{\alpha_8+\eta_4}\right) \tag{3.46}$$

Consider (ii) and (iii) together for an inequality such as

$$1 - (-a_{11}a_{33}a_{44} + a_{31}a_{13}a_{44})^2 > 0 \tag{3.47}$$

It is evident that $a_0 < 0$. Therefore (3.47) can be rewritten such as

$$1 + a_{31}a_{13}a_{44} > a_{11}a_{33}a_{44} \tag{3.48}$$

Which implies

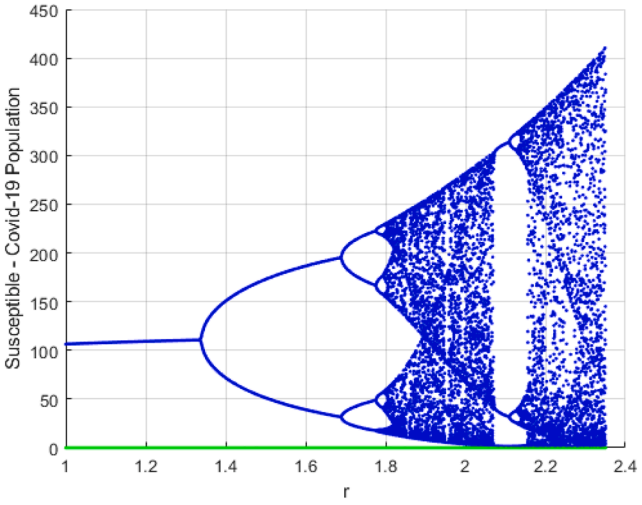

Fig. 2. Dynamical behavior of $S-I$ classes.

$$r_1\alpha_1\alpha_5\alpha_7 > (-(\alpha_8+\eta_4) + (\alpha_7+\alpha_8+\eta_4)e^{-\overline{\psi}_4})\left\{(\beta_1+\gamma_1)\gamma_1(1-e^{-\overline{\psi}_1})(1 - e^{-\overline{\psi}_3}) + (-(\alpha_2 r_1+\eta_1) + (\alpha_1 r_1+\alpha_2 r_1+\eta_1)e^{-\overline{\psi}_1})\left(-\left(\alpha_6 + \eta_3 - \frac{\delta_1{}^2h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right) + \left(\alpha_5+\alpha_6+\eta_3 - \frac{\delta_1{}^2h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)e^{-\overline{\psi}_3}\right)\right\} \tag{3.49}$$

The above inequality holds if $\overline{\psi}_4 > 0$ and

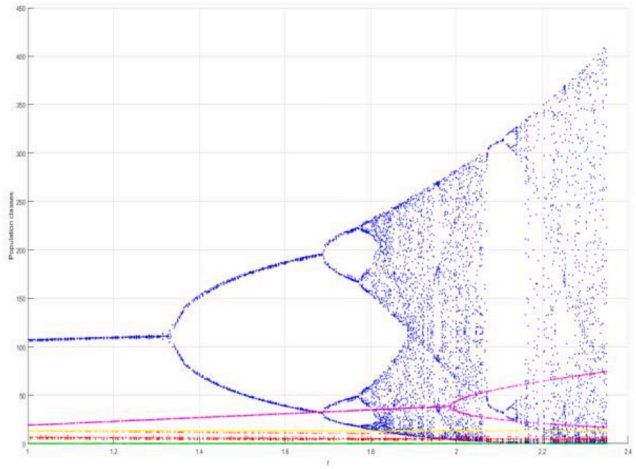

Fig. 3. Dynamical behavior of the population classes.

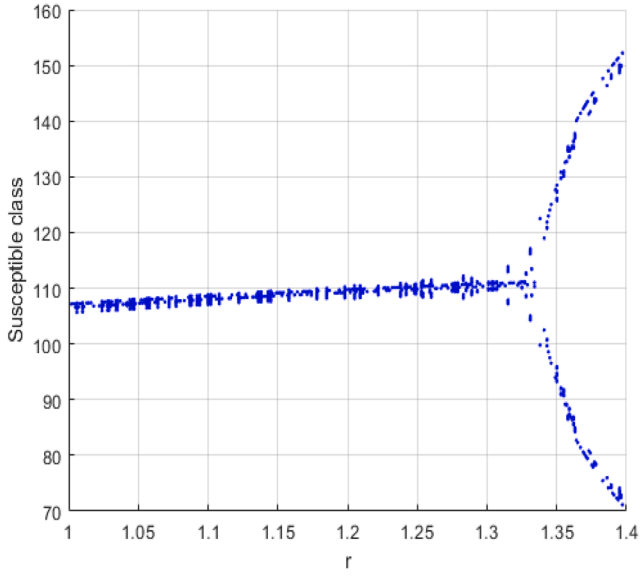

Fig. 4. Non-hyperbolic behavior of class.*S*

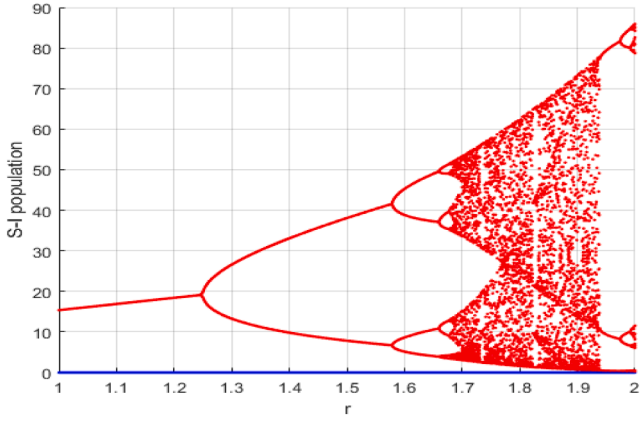

Fig. 5. Dynamical behavior of the population class *I* and the extinction of the susceptible population class.

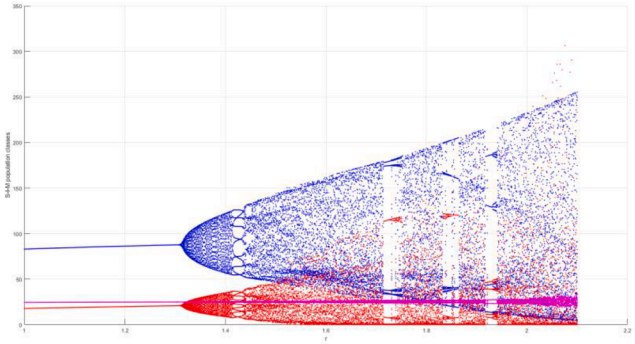

Fig. 6. Bifurcation diagram of the S-I-M classes.

$$(\beta_1+\gamma_1)\gamma_1+(\alpha_2 r_1+\eta_1)\left(\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)+\left\{(\alpha_1 r_1+\alpha_2 r_1+\eta_1)\left(\alpha_5+\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)+(\beta_1+\gamma_1)\gamma_1\right\}e^{-\overline{\psi}_1}e^{-\overline{\psi}_3}$$
$$<r_1\alpha_1\alpha_5+\left\{(\beta_1+\gamma_1)\gamma_1+\left(\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)(\alpha_1 r_1+\alpha_2 r_1+\eta_1)\right\}e^{-\overline{\psi}_1}+\left\{(\beta_1+\gamma_1)\gamma_1+(\alpha_2 r_1+\eta_1)\left(\alpha_5+\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)\right\}e^{-\overline{\psi}_3} \tag{3.50}$$

satisfies. From (3.50) we obtain the conditions as following;

$$\overline{\psi}_1>ln\left(\frac{(\alpha_1 r_1+\alpha_2 r_1+\eta_1)\left(\alpha_5+\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)+(\beta_1+\gamma_1)\gamma_1}{(\alpha_2 r_1+\eta_1)\left(\alpha_5+\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)+(\beta_1+\gamma_1)\gamma_1}\right) \tag{3.51}$$

and

$$\overline{\psi}_1<ln\left(\frac{(\beta_1+\gamma_1)\gamma_1+\left(\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)(\alpha_1 r_1+\alpha_2 r_1+\eta_1)}{(\alpha_2 r_1+\eta_1)\left(\alpha_6+\eta_3-\frac{{\delta_1}^2 h\overline{M}_3}{\left(1+h\delta_1\overline{C}_3\right)^2}\right)}\right) \tag{3.52}$$

where $r_1>\frac{(\beta_1+\gamma_1)\gamma_1}{\alpha_1\alpha_5}$. Considering both (3.50) and (3.51) together with (3.44), we have

$$\overline{\psi}_1 \in \left(ln \left(\frac{(\alpha_1 r_1 + \alpha_2 r_1 + \eta_1)\left(\alpha_5 + \alpha_6 + \eta_3 - \frac{\delta_1{}^2 h \overline{M}_3}{\left(1+h\delta_1 \overline{C}_3\right)^2}\right) + (\beta_1 + \gamma_1)\gamma_1}{(\alpha_2 r_1 + \eta_1)\left(\alpha_5 + \alpha_6 + \eta_3 - \frac{\delta_1{}^2 h \overline{M}_3}{\left(1+h\delta_1 \overline{C}_3\right)^2}\right) + (\beta_1 + \gamma_1)\gamma_1} \right), ln\left(\frac{\alpha_1 r_1 + \alpha_2 r_1 + \eta_1}{\alpha_2 r_1 + \eta_1}\right) \right) \tag{3.53}$$

Furthermore, from the characteristic equation in (3.38), we get $|a_{22}| < 1$ and $|a_{55}| < 1$, if

$\overline{\psi}_2 > 0 \Rightarrow \bar{I}_3 < \frac{K_2}{\alpha_4+\theta+\eta_2}$ and $\overline{\psi}_5 > 0 \Rightarrow K_5 > (\alpha_{10}+\eta_5)\overline{M}_3 + \beta_2 \bar{I}_3$. This completes the proof.

Remark 3.3. *In Theorem* 3.5 *we considered the local stability of the co-existing equilibrium point* Λ_3. *We assumed that intensive and continuous information of health organizations would reach to a fruitful awareness of civilian in recognizing the symptoms of COVID*-19. *Another vital organization was the police that is only responsible for controlling the panic of the civilians but shows now different roles in this pandemic scenario. Here, we assumed that this organization is well educated in infection cases and also knows to detect and protect the civilians as well as themselves. However, still, we keep the possibility that the police might be infected from the class who does not know they are infected.*

According to this scenario, we analyzed the stability criteria of the positive equilibrium point. We obtained that the increase of the screening rate will decrease the carrying capacity of the infected group who do not know they are infected. This I class is the most dangerous since they show host roles in the *human-to-human* transmission. They might have mild symptoms or could never show the symptoms. Therefore, the screening rate θ has a crucial role in I.

Additionally, we obtained that the carrying capacity of the police personal should be related to the infected class I and not C. However, this number is too high, and it might be difficult to lead all the personals to be responsible for the control mechanism. This mechanism is related to the function of Holling Type II, and this function showed that if there are specially educated police personals for infection cases, then the rate to act and the average time of these personals might be enough to have a stable dynamical system. There is no need to increase the carrying capacity of class M for this pandemic spread.

Example. *A series of simulations are conducted to verify the theoretical results using MATLAB* 2019. *The blue color denotes the susceptible class S, while the red color represents class I whose members do not know they are infected. The COVID*-19 *infected population who knows they are infected is shown in green color. We denote the hospitalized population in yellow and the class of police in purple.*

Figs. 1–4 present numerical simulations that are conducted to verify Theorem 3.1, which represents the stability of a non-infected population class of S and M. We obtain the dynamical behavior under the assumption that we stopped any transmission risk between the susceptible civilians and the infected groups. Additionally, the police are highly aware of the infection risks to protect themselves.

Thus, the values of the parameters are chosen according to the theoretical results of Theorem 3.1(i), which are $K_1 = 1.6, K_2 = K_3 = K_5 = 1, K_4 = 1.2, \alpha_1 = \alpha_9 = 0.012, \alpha_2 = \alpha_3 = \alpha_4 = \alpha_5 = \alpha_7 = \alpha_{10} = 0.05, \alpha_6 = \alpha_8 = 0.01, \beta_1 = 0.00134, \beta_2 = 0.001, \gamma_1 = 0.0004, \gamma_2 = 0.0001, \varepsilon_1 = \varepsilon_2 = 0.4, \theta = 0.1, \mu = 0.1, \eta_1 = \eta_5 = 0.002, \eta_2 = \eta_4 = 0.003, \eta_3 = 0.004, \delta_1 = 0.04$. Fig. 1 shows the dynamical behavior between the population classes, while Fig. 2 is a detailed graph of the $S-I$ interaction.

In considering the conditions for Theorem 3.1(ii), the parametric values are as following;

$K_1 = 1.6, K_2 = K_4 = K_5 = 1, K_3 = 0.947, \alpha_2 = \alpha_3 = \alpha_4 = \alpha_5 = \alpha_7 = \alpha_{10} = 0.05, \alpha_6 = \alpha_8 = 0.01, \beta_1 = 0.00134, \beta_2 = 0.001, \gamma_1 = 0.0004, \gamma_2 = 0.0001, \varepsilon_1 = \varepsilon_2 = 1, \theta = 0.1, \mu = 0.1, \eta_1 = \eta_5 = 0.002, \eta_2 = \eta_4 = 0.003, \eta_3 = 0.004, \delta_1 = 0.0011$. It is seen in Fig. 3 that, based on the results of Theorem 3.1(ii), we obtain a structure of non-hyperbolic behavior of the population classes. Fig. 4 gives a detailed diagram of a logistic rate of discrete-time.

Fig. 5 presents a dynamical structure of Theorem 3.3(i), where we focused on the apocalypse case that shows a dramatic increase of infected individuals from both civilians and police personals. The population rate per year for the susceptible class decreases since, in this apocalypse scenario, we assume that it is also difficult to have a non-infected offspring. The susceptible class and the police are not aware and educated, so that we expect a decrease in both classes.

Thus, for the chosen values $K_1 = 1, K_2 = K_3 = K_4 = 1.6, K_5 = 0.7, \alpha_1 = \alpha_9 = 0.012, \alpha_2 = \alpha_3 = \alpha_4 = \alpha_5 = \alpha_7 = \alpha_{10} = 0.05, \alpha_6 = \alpha_8 = 0.01, \beta_1 = 0.04, \beta_2 = 0.001, \gamma_1 = 0.001, \gamma_2 = 0.001, \varepsilon_1 = \varepsilon_2 = 0.4, \theta = 0.1, \mu = 0.1, \eta_1 = \eta_5 = 0.002, \eta_2 = \eta_4 = 0.003, \eta_3 = 0.004, \delta_1 = 0.04$, we obtain Fig. 5.

In Fig. 6, we show the bifurcation diagram of the susceptible class S, the group whose members do not know they are infected, and a class of the police personals according to the results of Theorem 3.5. The values are $K_1 = 1.4, K_2 = 1.2, K_3 = K_4 = 1, K_5 = 1.3, \alpha_1 = \alpha_3 = \alpha_5 = \alpha_7 = \alpha_9 = 0.012, \alpha_2 = \alpha_4 = \alpha_6 = \alpha_8 = \alpha_{10} = 0.01, \beta_1 = 0.04, \beta_2 = 0.001, \gamma_1 = 0.001, \theta = 0.1, \mu = 0.1, \eta_1 = \eta_5 = 0.002, \eta_2 = \eta_4 = 0.003, \eta_3 = 0.004, \delta_1 = 0.04$. This graph also represents that the infection spread might be repeated several times in the future. Therefore, the responsible control organizations in this pandemic spread should be careful of this infection case and need to continue to obstruct any spread of the novel coronavirus later on. The disquieting point is that whenever the infection appears again, it spreads fast and stays pandemic.

An analysis of the sensitive parameters in the control mechanisms

In this section, we analyze the periodic behavior of system (3.1) and prove that it does not show a periodic-two behavior. We show that a semi-cycle solution approaches the co-existing equilibrium pint. Moreover, it is obtained that the screening rate $\theta$and the average time of action δ are vital to control parameters for both health and police organizations in this pandemic spread.

Theorem 4.1. *Let* $\Phi = (S(n), I(n), C(n), H(n), M(n))$ *be a positive solution to system* (3.1). *If* $r_1 K_1 - \beta_1 I(n) - \gamma_1 C(n) \neq 0$ *and* $K_5 - \beta_2 I(n) - \gamma_2 C(n) \neq 0$ *for* $n = 0, 1, 2, \cdots$, *then system* (3.1) *does not show a period-two behavior.*

Proof. *Assume that*

$$\cdots, \upsilon, \omega, \upsilon, \omega, \cdots \tag{4.1}$$

solutions of period two to $(S(n))_{n=0}^{\infty}$ such that $\upsilon \neq \omega$. Thus, we can

write

$$\upsilon = \frac{\omega(\mathrm{r}_1(K_1 - \alpha_2\omega) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1\omega)}{\alpha_1\mathrm{r}_1\omega + (\mathrm{r}_1(K_1 - \alpha_1\omega - \alpha_2\omega) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1\omega)e^{-(\mathrm{r}_1(K_1-\alpha_2\omega)-\beta_1 I(\mathrm{n})-\gamma_1 C(\mathrm{n})-\eta_1\omega)}} \tag{4.2}$$

and

$$\omega = \frac{\upsilon(\mathrm{r}_1(K_1 - \alpha_2\upsilon) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1\upsilon)}{\alpha_1\mathrm{r}_1\upsilon + (\mathrm{r}_1(K_1 - \alpha_1\upsilon - \alpha_2\upsilon) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1\upsilon)e^{-(\mathrm{r}_1(K_1-\alpha_2\upsilon)-\beta_1 I(\mathrm{n})-\gamma_1 C(\mathrm{n})-\eta_1\upsilon)}}. \tag{4.3}$$

Note that

$$\begin{aligned}\alpha_1\mathrm{r}_1\omega\upsilon = {} & \omega(\mathrm{r}_1(K_1 - \alpha_2\omega) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1\omega) - \upsilon(\mathrm{r}_1(K_1 - \alpha_1\omega \\ & - \alpha_2\omega) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1\omega)e^{-(\mathrm{r}_1(K_1-\alpha_2\omega)-\beta_1 I(\mathrm{n})-\gamma_1 C(\mathrm{n})-\eta_1\omega)}\end{aligned} \tag{4.4}$$

and

$$\begin{aligned}\alpha_1\mathrm{r}_1\omega\upsilon = {} & \upsilon(\mathrm{r}_1(K_1 - \alpha_2\upsilon) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1\upsilon) - \omega(\mathrm{r}_1(K_1 - \alpha_1\upsilon \\ & - \alpha_2\upsilon) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1\upsilon)e^{-(\mathrm{r}_1(K_1-\alpha_2\upsilon)-\beta_1 I(\mathrm{n})-\gamma_1 C(\mathrm{n})-\eta_1\upsilon)}\end{aligned} \tag{4.5}$$

From (4.4) and (4.5), we have

$$\begin{aligned}&(\mathrm{r}_1K_1 - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}))(\omega - \upsilon) - (\alpha_2\mathrm{r}_1 + \eta_1)(\omega - \upsilon)(\omega + \upsilon) \\ &-(\mathrm{r}_1K_1 - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}))\big(\upsilon e^{-(\mathrm{r}_1(K_1-\alpha_2\omega)-\beta_1 I(\mathrm{n})-\gamma_1 C(\mathrm{n})-\eta_1\omega)} \\ &-\omega e^{-(\mathrm{r}_1(K_1-\alpha_2\upsilon)-\beta_1 I(\mathrm{n})-\gamma_1 C(\mathrm{n})-\eta_1\upsilon)}\big) + \omega\upsilon(\mathrm{r}_1(\alpha_1 + \alpha_2) + \eta_1) \\ &\big(e^{-(\mathrm{r}_1(K_1-\alpha_2\omega)-\beta_1 I(\mathrm{n})-\gamma_1 C(\mathrm{n})-\eta_1\omega)} - e^{-(\mathrm{r}_1(K_1-\alpha_2\upsilon)-\beta_1 I(\mathrm{n})-\gamma_1 C(\mathrm{n})-\eta_1\upsilon)}\big) = 0\end{aligned} \tag{4.6}$$

Since $\mathrm{r}_1K_1 - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) \neq 0$, (4.6) holds if $= \omega$, which contradicts our assumption. Similarly, we can obtain that $(\mathrm{I}(\mathrm{n}))_{\mathrm{n}=0}^{\infty}$, $(\mathrm{C}(\mathrm{n}))_{\mathrm{n}=0}^{\infty}$and $(\mathrm{H}(\mathrm{n}))_{\mathrm{n}=0}^{\infty}$ do not show a period-two behavior. Moreover, if

$K_5 - \beta_2 I(\mathrm{n}) - \gamma_2 C(\mathrm{n}) \neq 0$, then $(\mathrm{M}(\mathrm{n}))_{\mathrm{n}=0}^{\infty}$ is not a solution to period-two. This completes the proof.

Remark 4.1. *Theorem* 4.1. *shows that these two classes: who do not know they are infected and the population class who knows they are infected, have a dominant role in the carrying capacities of the susceptible and the police population. The non-periodic behavior is controlled by the interaction rates of class I and C.*

Theorem 4.2. *Assume that* $\Phi = (S(n), I(n), C(n), H(n), M(n))$ *is a positive solution to system* (3.1). *The upcoming statements hold.*

(i) If

$$\begin{aligned}&\psi_1(n) > \alpha_1\mathrm{r}_1\mathrm{S}(\mathrm{n}), \psi_2(n) > \alpha_3 I(n), \psi_3(n) > \alpha_5 C(n), \psi_4(n) \\ &\quad > \alpha_7 H(n) \text{and} \psi_5(n) > \alpha_9 M(n)\end{aligned} \tag{4.7}$$

then the solution of system (3.1) increases monotonically.

(ii) If

$$\begin{aligned}&0 < \psi_1(n) < \alpha_1\mathrm{r}_1\mathrm{S}(\mathrm{n}), 0 < \psi_2(n) < \alpha_3 I(n), 0 < \psi_3(n) < \alpha_5 C(n), \\ &0 < \psi_4(n) < \alpha_7 H(n) \text{and} < \psi_5(n) < \alpha_9 M(n)\end{aligned} \tag{4.8}$$

then the solution of system (3.1) decreases monotonically.

Proof. **(i)** *From* (3.1), *we obtain*

$$\begin{cases} \dfrac{S(n+1)}{S(n)} = \dfrac{\psi_1(n)}{\alpha_1\mathrm{r}_1\mathrm{S}(\mathrm{n}) + (\psi_1(n) - \alpha_1\mathrm{r}_1\mathrm{S}(\mathrm{n}))e^{-\psi_1(n)}} > 1 \\ \dfrac{I(n+1)}{I(n)} = \dfrac{\psi_2(n)}{\alpha_3 I(n) + (\psi_2(n) - \alpha_3 I(n))e^{-\psi_2(n)}} > 1 \\ \dfrac{C(n+1)}{C(n)} = \dfrac{\psi_3(n)}{\alpha_5 C(n) + (\psi_3(n) - \alpha_5 C(n))e^{-\psi_3(n)}} > 1 \\ \dfrac{H(n+1)}{H(n)} = \dfrac{\psi_4(n)}{\alpha_7 H(n) + (\psi_4(n) - \alpha_7 H(n))e^{-\psi_4(n)}} > 1 \\ \dfrac{M(n+1)}{M(n)} = \dfrac{\psi_5(n)}{\alpha_9 M(n) + (\psi_5(n) - \alpha_9 M(n))e^{-\psi_5(n)}} > 1 \end{cases} \tag{4.9}$$

for the conditions in (4.7), which completes the proof that the positive solutions of (3.1) are monotonic increasing.

(ii) In considering

$$\begin{cases} \dfrac{S(n+1)}{S(n)} = \dfrac{\psi_1(n)}{\alpha_1\mathrm{r}_1\mathrm{S}(\mathrm{n}) + (\psi_1(n) - \alpha_1\mathrm{r}_1\mathrm{S}(\mathrm{n}))e^{-\psi_1(n)}} < 1 \\ \dfrac{I(n+1)}{I(n)} = \dfrac{\psi_2(n)}{\alpha_3 I(n) + (\psi_2(n) - \alpha_3 I(n))e^{-\psi_2(n)}} < 1 \\ \dfrac{C(n+1)}{C(n)} = \dfrac{\psi_3(n)}{\alpha_5 C(n) + (\psi_3(n) - \alpha_5 C(n))e^{-\psi_3(n)}} < 1 \\ \dfrac{H(n+1)}{H(n)} = \dfrac{\psi_4(n)}{\alpha_7 H(n) + (\psi_4(n) - \alpha_7 H(n))e^{-\psi_4(n)}} < 1 \\ \dfrac{M(n+1)}{M(n)} = \dfrac{\psi_5(n)}{\alpha_9 M(n) + (\psi_5(n) - \alpha_9 M(n))e^{-\psi_5(n)}} < 1 \end{cases} \tag{4.10}$$

we obtain that (4.10) holds for the statement in (4.8). This completes the proof of (ii).

Theorem 4.3. *Let* $\Phi = (\mathrm{S}(\mathrm{n}), \mathrm{I}(\mathrm{n}), \mathrm{C}(\mathrm{n}), \mathrm{H}(\mathrm{n}), \mathrm{M}(\mathrm{n}))$ *be a positive solution of system* (3.1), *which has a single semi cycle. Moreover,* (4.7) *holds. If*

$$\begin{cases} \psi_1(n-1) < \ln\left(\dfrac{\overline{S}_3}{\mathrm{S(n-1)}}\right) \\ \psi_2(n-1) < \ln\left(\dfrac{\overline{I}_3}{\mathrm{I(n-1)}}\right) \\ \psi_3(n-1) < \ln\left(\dfrac{\overline{C}_3}{\mathrm{C(n-1)}}\right) \\ \psi_4(n-1) < \ln\left(\dfrac{\overline{H}_3}{\mathrm{H(n-1)}}\right) \\ \psi_5(n-1) < \ln\left(\dfrac{\overline{M}_3}{\mathrm{M(n-1)}}\right) \end{cases} \tag{4.11}$$

for all $n \in [1, \infty)$, then $(S(n), I(n), C(n), H(n), M(n))$ converges monotonically to the positive equilibrium point Λ_3.

Proof. *Suppose that* $0 < S(n-1) < \overline{S}_3$ *for all* $n \in [1, \infty)$. *Note that in this case, we have from (4.7) the inequality* $\mathrm{S(n-1)} < \mathrm{S(n)}$. *Moreover, we have*

$$S(n) < \overline{S}_3 \Rightarrow \frac{S(n-1)\psi_1(n-1)}{\alpha_1 r_1 S(n-1) + (\psi_1(n-1) - \alpha_1 r_1 S(n-1))e^{-\psi_1(n-1)}} < \overline{S}_3 \tag{4.12}$$

$$S(n+1) = \frac{S(n)(\mathrm{r_1}(K_1 - \alpha_2 \mathrm{S(n)}) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1 \mathrm{S(n)})}{\alpha_1 \mathrm{r_1 S(n)} + (\mathrm{r_1}(K_1 - \alpha_2 \mathrm{S(n)}) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1 \mathrm{S(n)} - \alpha_1 \mathrm{r_1 S(n)})e^{-(\mathrm{r_1}(K_1 - \alpha_2 \mathrm{S(n)}) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1 \mathrm{S(n)})}}$$

$$= \frac{(\mathrm{r_1}(K_1 - \alpha_2 \mathrm{S(n)}) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1 \mathrm{S(n)})S(n)e^{(\mathrm{r_1}(K_1 - \alpha_2 \mathrm{S(n)}) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1 \mathrm{S(n)})}}{\alpha_1 \mathrm{r_1 S(n)}(e^{(\mathrm{r_1}(K_1 - \alpha_2 \mathrm{S(n)}) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1 \mathrm{S(n)})} - 1) + (\mathrm{r_1}(K_1 - \alpha_2 \mathrm{S(n)}) - \beta_1 I(\mathrm{n}) - \gamma_1 C(\mathrm{n}) - \eta_1 \mathrm{S(n)})} < S(n)e^{\psi_1(n)}. \tag{4.19}$$

which holds for $S(n-1) < \overline{S}_3$ and

$$S(n-1) < \overline{S}_3 \cdot e^{-\psi_1(n-1)} \Rightarrow \psi_1(n-1) < ln\left(\frac{\overline{S}_3}{S(n-1)}\right) \tag{4.13}$$

Thus, we obtain
$0 < S(n-1) < S(n) < \overline{S}_3$ for all $n \in [1, \infty)$.
Similarly, as before, we can prove that

$$0 < I(\mathrm{n-1}) < \mathrm{I(n)} < \overline{I}_3 \mathrm{forall} \in [1, \infty), \mathrm{if} \psi_2(n-1) < \ln\left(\frac{\overline{I}_3}{\mathrm{I(n-1)}}\right) \tag{4.14}$$

$$0 < C(\mathrm{n-1}) < \mathrm{C(n)} < \overline{C}_3 \mathrm{forall} n \in [1, \infty), \mathrm{if} \psi_3(n-1) < \ln\left(\frac{\overline{C}_3}{\mathrm{C(n-1)}}\right) \tag{4.15}$$

$$0 < H(\mathrm{n-1}) < \mathrm{H(n)} < \overline{H}_3 \mathrm{forall} n \in [1, \infty), \mathrm{if} \psi_4(n-1) < \ln\left(\frac{\overline{H}_3}{\mathrm{H(n-1)}}\right) \tag{4.16}$$

and

$$0 < M(\mathrm{n-1}) < \mathrm{M(n)} < \overline{M}_3 \mathrm{forall} n \in [1, \infty), \mathrm{if} \psi_5(n-1) < \ln\left(\frac{\overline{M}_3}{\mathrm{M(n-1)}}\right) \tag{4.17}$$

This completes the proof.

Theorem 4.4. *Let* $\Phi = (S(n), I(n), C(n), H(n), M(n))$ *be a positive solution of system (3.1) and assume that (4.7) holds. Then the solution is bounded in the interval given by*

$$S(n) \in \left(0, S(0)e^{\sum_{i=1}^{n} \psi_1(i-1)}\right), I(n) \in \left(0, I(0)e^{\sum_{i=1}^{n} \psi_2(i-1)}\right), C(n) \in \left(0, C(0)e^{\sum_{i=1}^{n} \psi_3(i-1)}\right),$$

$$H(n) \in \left(0, H(0)e^{\sum_{i=1}^{n} \psi_4(i-1)}\right) \mathrm{and} M(n) \in \left(0, M(0)e^{\sum_{i=1}^{n} \psi_5(i-1)}\right) \tag{4.18}$$

Proof. *Consider first the susceptible class as following;*

From the iteration of the sequence in (4.19), we obtain

$$S(n) < S(0)e^{\sum_{i=1}^{n} \psi_1(i-1)} \tag{4.20}$$

Similarly, we can obtain the interval of the remaining population classes. This completes the proof.

Theorem 4.5. *Let* Λ_3 *be the co-existing equilibrium point of system (3.1) and assume that the conditions in Theorem* 3.5 *and Theorem* 4.2(*i*) *hold. If*

$$\begin{cases} K_1 < \dfrac{1}{\mathrm{r}_1}\left(\ln\left(\dfrac{2\overline{S}_3 - S(n)}{S(n)}\right) + (\mathrm{r}_1\alpha_2 + \eta_1)\mathrm{S(n)} + \beta_1 I(\mathrm{n}) + \gamma_1 C(\mathrm{n})\right) \\ K_2 < \ln\left(\dfrac{2\overline{I}_3 - I(n)}{I(n)}\right) + (\alpha_4 + \theta + \eta_2)I(\mathrm{n}) \\ K_3 < \ln\left(\dfrac{2\overline{C}_3 - C(n)}{C(n)}\right) + (\alpha_6 + \eta_3)C(n) - (\beta_1 + \gamma_1)S(\mathrm{n}) - \beta_2 M(\mathrm{n}) - \theta I(\mathrm{n}) + f(\mathrm{n}) + \mu H(n) \\ K_4 < \ln\left(\dfrac{2\overline{H}_3 - H(n)}{H(n)}\right) + (\alpha_8 + \eta_4)H(\mathrm{n}) - \mu C(\mathrm{n}) - f(\mathrm{n})C(\mathrm{n}) \\ K_5 < \ln\left(\dfrac{2\overline{M}_3 - M(n)}{M(n)}\right) + (\alpha_{10} + \eta_5)M(\mathrm{n}) + \beta_2 I(n) \end{cases} \tag{4.21}$$

Proof. *Consider a Lyapunov function V defined by*

$$V(n) = (\Phi(n) - \Lambda_3)^2, n = 0, 1, 2, \cdots \tag{4.22}$$

where $\Phi(n) = (S(n), \mathrm{I}(n), \mathrm{C}(n), H(n), M(n))$ and $\Lambda_3 = \left(\overline{S}_3, \overline{I}_3, \overline{C}_3, \overline{H}_3, \overline{M}_3\right)$. From

$$\Delta V(n) = V(n+1) - V(n) = (\Phi(n+1) - \Phi(n))(\Phi(n+1) + \Phi(n) - 2\Lambda_3), \tag{4.23}$$

we want to consider the conditions of the first equation of system (3.1) to obtain $\Delta V_1(n) < 0$. Simple computations reveal that

$$\Delta S(n) = S(n+1) - S(n) = \frac{S(n)(\psi_1(n) - \alpha_1 \mathrm{r}_1 \mathrm{S(n)})\left(1 - e^{-\psi_1(n)}\right)}{\alpha_1 \mathrm{r}_1 \mathrm{S(n)} + (\psi_1(n) - \alpha_1 \mathrm{r}_1 \mathrm{S(n)})e^{-\psi_1(n)}} > 0 \tag{4.24}$$

since (4.7) holds. To obtain the global asymptotic stability condition for the susceptible class, this means $\Delta V_1(n) < 0$, we need to get $S(n+1) + S(n) - 2\overline{S}_3 < 0$. This holds if

$$0 < \psi_1(n) < \ln\left(\frac{2\overline{S}_3 - S(n)}{S(n)}\right) \text{and} S(n) < \overline{S}_3, \tag{4.25}$$

which implies that $\lim_{n\to\infty} S(n) = \overline{S}_3$. Similarly, we can obtain the conditions

$$0 < \psi_2(n) < \ln\left(\frac{2\overline{I}_3 - I(n)}{I(n)}\right) \text{and} I(n) < \overline{I}_3 \text{for} \Delta V_2(n) < 0 \tag{4.26}$$

$$0 < \psi_3(n) < \ln\left(\frac{2\overline{C}_3 - C(n)}{C(n)}\right) \text{and} C(n) < \overline{C}_3 \text{for} \Delta V_3(n) < 0 \tag{4.27}$$

$$0 < \psi_4(n) < \ln\left(\frac{2\overline{H}_3 - H(n)}{H(n)}\right) \text{and} H(n) < \overline{H}_3 \text{for} \Delta V_4(n) < 0 \tag{4.28}$$

and

$$0 < \psi_5(n) < \ln\left(\frac{2\overline{M}_3 - M(n)}{M(n)}\right) \text{and} M(n) < \overline{M}_3 \text{for} \Delta V_5(n) < 0 \tag{4.29}$$

Thus, $\lim_{n\to\infty} I(n) = \overline{I}_3$, $\lim_{n\to\infty} C(n) = \overline{C}_3$, $\lim_{n\to\infty} H(n) = \overline{H}_3$ and $\lim_{n\to\infty} M(n) = \overline{M}_3$. Considering from Theorem 3.5 that we assumed $\varepsilon_1 = \varepsilon_2 = 1$ and $\gamma_2 = 0$, we get

$$\begin{cases} K_1 < \dfrac{1}{\mathrm{r}_1}\left(\ln\left(\dfrac{2\overline{S}_3 - S(n)}{S(n)}\right) + (\mathrm{r}_1\alpha_2 + \eta_1)\mathrm{S(n)} + \beta_1 I(\mathrm{n}) + \gamma_1 C(\mathrm{n})\right) \\ K_2 < \ln\left(\dfrac{2\overline{I}_3 - I(n)}{I(n)}\right) + (\alpha_4 + \theta + \eta_2)I(\mathrm{n}) \\ K_3 < \ln\left(\dfrac{2\overline{C}_3 - C(n)}{C(n)}\right) + (\alpha_6 + \eta_3)C(n) - (\beta_1 + \gamma_1)S(\mathrm{n}) - \beta_2 M(\mathrm{n}) - \theta I(\mathrm{n}) + f(\mathrm{n}) + \mu H(n) \\ K_4 < \ln\left(\dfrac{2\overline{H}_3 - H(n)}{H(n)}\right) + (\alpha_8 + \eta_4)H(\mathrm{n}) - \mu C(\mathrm{n}) - f(\mathrm{n})C(\mathrm{n}) \\ K_5 < \ln\left(\dfrac{2\overline{M}_3 - M(n)}{M(n)}\right) + (\alpha_{10} + \eta_5)M(\mathrm{n}) + \beta_2 I(n) \end{cases} \tag{4.30}$$

which completes the proof.

Remark 4.2. *Theorem* 4.5. *shows the conditions of global asymptotical stability of the co-existing equilibrium point* Λ_3, *where we assumed in Theorem* 3.5 *that the awareness of the susceptible class and the police personals are high so that* $\varepsilon_1 = \varepsilon_2 = 1$ *and* $\gamma_2 = 0$. *We obtained that global stability is depended on the harvest rate of each population class and their equilibrium points.*

Theorem 4.6. *Let* $\mathrm{F} \in C\left[(\mathbb{R}^+)^5, \mathbb{R}^+\right]$ *such that*

$\mathrm{F}(x, y, z, p, q) =$

$$\begin{cases} f_1(x,y,z,p,q) = \dfrac{x(\mathrm{r}_1(K_1-\alpha_2\mathrm{x})-\beta_1 y-\gamma_1 z-\eta_1\mathrm{x})}{\alpha_1\mathrm{r}_1\mathrm{x}+(\mathrm{r}_1(K_1-\alpha_1\mathrm{x}-\alpha_2\mathrm{x})-\beta_1 y-\gamma_1 z-\eta_1\mathrm{x})e^{-(\mathrm{r}_1(K_1-\alpha_2\mathrm{x})-\beta_1 y-\gamma_1 z-\eta_1\mathrm{x})}} \\ f_2(x,y,z,p,q) = \dfrac{y(K_2-(\alpha_4+\theta+\eta_2)\mathrm{y}+\beta_1(1-\varepsilon_1)x+\beta_2(1-\varepsilon_2)q)}{\alpha_3 y+(K_2-(\alpha_3+\alpha_4+\theta+\eta_2)\mathrm{y}+\beta_1(1-\varepsilon_1)x+\beta_2(1-\varepsilon_2)q)e^{-(K_2-(\alpha_4+\theta+\eta_2)\mathrm{y}+\beta_1(1-\varepsilon_1)x+\beta_2(1-\varepsilon_2)q)}} \\ f_3(x,y,z,p,q) = \dfrac{z\left(K_3-(\alpha_6+\eta_3)\mathrm{z}+(\beta_1\varepsilon_1+\gamma_1)x+(\beta_2\varepsilon_2+\gamma_2)q+\theta\mathrm{y}-\dfrac{\delta_1\mathrm{q}}{1+h\delta_1\mathrm{z}}-\mu p\right)}{\alpha_5 z+\left(K_3-(\alpha_5+\alpha_6+\eta_3)\mathrm{z}+(\beta_1\varepsilon_1+\gamma_1)x+(\beta_2\varepsilon_2+\gamma_2)q+\theta\mathrm{y}-\dfrac{\delta_1\mathrm{q}}{1+h\delta_1\mathrm{z}}-\mu p\right)e^{-\left(K_3-(\alpha_6+\eta_3)\mathrm{z}+(\beta_1\varepsilon_1+\gamma_1)x+(\beta_2\varepsilon_2+\gamma_2)q+\theta\mathrm{y}-\frac{\delta_1\mathrm{q}}{1+h\delta_1\mathrm{z}}-\mu p\right)}} \\ f_4(x,y,z,p,q) = \dfrac{p\left(K_4-(\alpha_8+\eta_4)\mathrm{p}+\mu z+\dfrac{\delta_1\mathrm{qz}}{1+h\delta_1\mathrm{z}}\right)}{\alpha_7 z+\left(K_4-(\alpha_7+\alpha_8+\eta_4)\mathrm{p}+\mu z+\dfrac{\delta_1\mathrm{qz}}{1+h\delta_1\mathrm{z}}\right)e^{-\left(K_4-(\alpha_8+\eta_4)\mathrm{p}+\mu z+\frac{\delta_1\mathrm{qz}}{1+h\delta_1\mathrm{z}}\right)}} \\ f_5(x,y,z,p,q) = \dfrac{q(K_5-(\alpha_{10}+\eta_5)\mathrm{q}-\beta_2 y-\gamma_2 z)}{\alpha_9 z+(K_5-(\alpha_9+\alpha_{10}+\eta_5)\mathrm{q}-\beta_2 y-\gamma_2 z)e^{-(K_5-(\alpha_{10}+\eta_5)\mathrm{q}-\beta_2 y-\gamma_2 z)}} \end{cases} \tag{4.31}$$

If $(\alpha_1-\alpha_2)\mathrm{r}_1 > \eta_1$ for $\alpha_1 > \alpha_2$, $\alpha_3 > \alpha_4+\theta+\eta_2, \alpha_5+\alpha_6+\eta_3 > \frac{\delta_1{}^2\mathrm{hq}}{(1+h\delta_1\mathrm{z})^2} > \alpha_6+\eta_3, \alpha_7 > \alpha_8+\eta_4$ and $\alpha_9 > \alpha_{10}+\eta_5$, then every oscillatory solution of (3.1) shows a semi cycle behavior with a length at most two.

Proof. *If* $\frac{\partial f_1}{\partial x} < 0, \frac{\partial f_2}{\partial y} < 0, \frac{\partial f_3}{\partial z} < 0$,$\frac{\partial f_4}{\partial p} < 0$ *and* $\frac{\partial f_5}{\partial q} < 0$, *then every positive oscillatory solution of* (3.1) *has a semi cycle of length at most two. We have* $\frac{\partial f_1}{\partial x} < 0$, *if* $(\alpha_1-\alpha_2)\mathrm{r}_1 > \eta_1$ *and* $\alpha_1 > \alpha_2$ *for fixed values of* y, z, p, q *in* $(0,\infty)$, *where*

$\mathrm{r}_1 K_1 - 2\alpha_2\mathrm{r}_1\mathrm{x} - \beta_1 y - \gamma_1 z - 2\eta_1\mathrm{x} > 0$ and $\mathrm{r}_1(K_1-\alpha_1\mathrm{x}-\alpha_2\mathrm{x}) - \beta_1 y - \gamma_1 z - \eta_1\mathrm{x} < 0$.

Moreover, we have $\frac{\partial f_2}{\partial y} < 0$, if $\alpha_3 > \alpha_4+\theta+\eta_2$ for fixed values of x, z, p, q in $(0,\infty)$, where

$K_2 - 2(\alpha_4+\theta+\eta_2)\mathrm{y} + \beta_1(1-\varepsilon_1)x + \beta_2(1-\varepsilon_2)q > 0$ and $K_2 - (\alpha_3+\alpha_4+\theta+\eta_2)\mathrm{y} + \beta_1(1-\varepsilon_1)x + \beta_2(1-\varepsilon_2)q < 0$.

We get similarly $\frac{\partial f_3}{\partial z} < 0$, if $\alpha_5+\alpha_6+\eta_3 > \frac{\delta_1{}^2\mathrm{hq}}{(1+h\delta_1\mathrm{z})^2} > \alpha_6+\eta_3$ for fixed values of x, y, p, q in $(0,\infty)$, where

$K_3 - 2(\alpha_6+\eta_3)\mathrm{z} + (\beta_1\varepsilon_1+\gamma_1)x + (\beta_2\varepsilon_2+\gamma_2)q + \theta\mathrm{y} - \frac{\delta_1\mathrm{q}}{1+h\delta_1\mathrm{z}} - \mu p + \frac{\delta_1{}^2\mathrm{hq}}{(1+h\delta_1\mathrm{z})^2} > 0$ and

$$K_3 - (\alpha_5+\alpha_6+\eta_3)\mathrm{z} + (\beta_1\varepsilon_1+\gamma_1)x + (\beta_2\varepsilon_2+\gamma_2)q + \theta\mathrm{y} - \frac{\delta_1\mathrm{q}}{1+h\delta_1\mathrm{z}} - \mu p < 0$$

Considering $\frac{\partial f_4}{\partial p} < 0$, we obtain $\alpha_7 > \alpha_8+\eta_4$ for fixed values of x, y, z, q in $(0,\infty)$, where

$K_4 - 2(\alpha_8+\eta_4)\mathrm{p} + \mu z + \frac{\delta_1\mathrm{qz}}{1+h\delta_1\mathrm{z}} > 0$ and $K_4 - (\alpha_7+\alpha_8+\eta_4)\mathrm{p} + \mu z + \frac{\delta_1\mathrm{qz}}{1+h\delta_1\mathrm{z}} < 0$.

Finally, we obtain $\frac{\partial f_5}{\partial q} < 0$, if $\alpha_9 > \alpha_{10}+\eta_5$ for fixed values of x, y, z, p in $(0,\infty)$, where

$K_5 - 2(\alpha_{10}+\eta_5)\mathrm{q} - \beta_2 y - \gamma_2 z > 0$ and $K_5 - (\alpha_9+\alpha_{10}+\eta_5)\mathrm{q} - \beta_2 y - \gamma_2 z < 0$.

This completes the proof.

Remark 4.3. *In Theorem* 4.6, *we obtain conditions for a positive oscillatory solution of a semi-cyle behavior with a length at most two. We see that in all population classes, the logistic rate at a continuous-time has a dominant role in both the logistic rate at discrete-time and the death rate (natural or infection). Additionally, class I oscillates if the screening rate is less than the logistic rate but still exists. If* θ, *then we see negative semi-cycle behavior, and the population size moves to class C. Otherwise, we have a positive semi-cycle behavior in class I. We noticed only in class C the effect of the Holling Type II function. We perceived that the rate of action and the rate of average time is restricted to the logistic rates and the death rate through infection as much as the logistic rate increases the transport to health institutions increases. The inverse ratio of the police class shows that increasing the size of class M, decrease the interval of classC.*

Strong and weak Allee effect of a spread of COVID-19

In 1838, Pierre Verhulst [24] considered the *logistic growth* function to explain mono-species growth. If $x(t)$ represents the population size at time t, then the logistic growth equation is

$$\frac{dx}{dt} = rx\left(1-\frac{x}{K}\right), \tag{5.1}$$

where r and K are positive numbers. However, in biological phenomena, many situations require modifications in the model to explain the growth of the population in low density-size, which is well known as *the Allee effect.* We can consider the Allee effect in two main types: (i) strong Allee effect and (ii) weak Allee effect. A population with a strong Allee effect will have a critical population size, which is the threshold of the population, and any size that is less than the threshold will go to extinction without any further aid. A population with a weak Allee effect

will reduce the per capita growth rate at lower population density or size [25–27].

In this section, we incorporate

$$a(I[\![t]\!]) = \frac{I([\![t]\!])}{E_0} - 1 \text{and} a(C[\![t]\!]) = \frac{C([\![t]\!])}{E_1} - 1 \tag{5.2}$$

to the infected classes I and Cin system (2.1) such as

$$\begin{cases} \dfrac{\mathrm{dS}}{\mathrm{dt}} = S(\mathrm{t})(\mathrm{r}_1(K_1 - \alpha_1 \mathrm{S}(\mathrm{t}) - \alpha_2 \mathrm{S}([\![\mathrm{t}]\!])) - \beta_1 I([\![\mathrm{t}]\!]) - \gamma_1 C([\![\mathrm{t}]\!]) - \eta_1 \mathrm{S}([\![\mathrm{t}]\!])) \\ \dfrac{\mathrm{d}I}{\mathrm{dt}} = I(t)a(I[\![t]\!])(K_2 - \alpha_3 I(t) - \alpha_4 \mathrm{I}([\![\mathrm{t}]\!]) + \beta_1(1-\varepsilon_1)S([\![\mathrm{t}]\!]) - \theta \mathrm{I}([\![\mathrm{t}]\!]) - \eta_2 \mathrm{I}([\![\mathrm{t}]\!]) + \beta_2(1-\varepsilon_2)M([\![\mathrm{t}]\!])) \\ \dfrac{\mathrm{d}C}{\mathrm{dt}} = C(t)a(C[\![t]\!])(K_3 - \alpha_5 C(t) - \alpha_6 \mathrm{C}([\![\mathrm{t}]\!]) + \beta_1\varepsilon_1 S([\![\mathrm{t}]\!]) + \beta_2\varepsilon_2 M([\![\mathrm{t}]\!]) + \theta \mathrm{I}([\![\mathrm{t}]\!]) + \gamma_1 \mathrm{S}([\![\mathrm{t}]\!]) + \gamma_2 M([\![\mathrm{t}]\!])) \\ \qquad\qquad - f([\![\mathrm{t}]\!]) - \mu H([\![\mathrm{t}]\!]) - \eta_3 \mathrm{C}([\![\mathrm{t}]\!])) \\ \dfrac{\mathrm{d}H}{\mathrm{dt}} = H(\mathrm{t})(K_4 - \alpha_7 H(t) - \alpha_8 \mathrm{H}([\![\mathrm{t}]\!]) + \mu C([\![\mathrm{t}]\!]) + f([\![\mathrm{t}]\!])\mathrm{C}([\![\mathrm{t}]\!]) - \eta_4 \mathrm{H}([\![\mathrm{t}]\!])) \\ \dfrac{\mathrm{d}M}{\mathrm{dt}} = M(\mathrm{t})(K_5 - \alpha_9 M(t) - \alpha_{10} \mathrm{M}([\![\mathrm{t}]\!]) - \beta_2 I([\![\mathrm{t}]\!]) - \gamma_2 C([\![\mathrm{t}]\!]) - \eta_5 \mathrm{M}([\![\mathrm{t}]\!])) \end{cases} \tag{5.3}$$

where E_0 and E_1 represent the threshold of the infected class whose members do not know they are infected and the class whose members know they are infected by COVID-19, respectively. If the population is below the threshold level, then the growth rate decreases to extinct. This phenomenon describes a *strong Allee effect*.

Integrating both sides of (5.3) on $[n, t)$ and taking $t \to n+1$, we get a system of difference equations such as

$$\begin{cases} S(n+1) = \dfrac{S(n)\psi_1(n)}{\alpha_1 \mathrm{r}_1 \mathrm{S}(\mathrm{n}) + (\psi_1(n) - \alpha_1 \mathrm{r}_1 \mathrm{S}(\mathrm{n}))e^{-\psi_1(n)}} \\ I(n+1) = \dfrac{I(n)\psi_2(n)}{\alpha_3 I(n) + (\psi_2(n) - \alpha_3 I(n))e^{-a(I(n))\psi_2(n)}} \\ C(n+1) = \dfrac{C(n)\psi_3(n)}{\alpha_5 C(n) + (\psi_3(n) - \alpha_5 C(n))e^{-a(C(n))\psi_3(n)}} \\ H(n+1) = \dfrac{H(n)\psi_4(n)}{\alpha_7 H(n) + (\psi_4(n) - \alpha_7 H(n))e^{-\psi_4(n)}} \\ M(n+1) = \dfrac{M(n)\psi_5(n)}{\alpha_9 M(n) + (\psi_5(n) - \alpha_9 M(n))e^{-\psi_5(n)}}. \end{cases} \tag{5.4}$$

Let

$$G(n) = \frac{I(n+1)}{I(n)} = \frac{\psi_2(n)}{\alpha_3 I(n) + (\psi_2(n) - \alpha_3 I(n))e^{-a(I(n))\psi_2(n)}} \tag{5.5}$$

We obtain $\frac{\partial G(n)}{\partial I(n)} < 0$ if (4.7) holds and we have

$$\psi_2(n) < \frac{a(I(n))(\alpha_4 + \theta + \eta_2)}{\frac{da(I(n))}{dI}}. \tag{5.6}$$

Thus, the screening rate, the death rate caused by infection cases, and the logistic rate at discrete-time are essential to control parameters to support the Allee function in stabilizing the effect of the spread.

Furthermore, let

$$L(n) = \frac{C(n+1)}{C(n)} = \frac{\psi_3(n)}{\alpha_5 C(n) + (\psi_3(n) - \alpha_5 C(n))e^{-a(C(n))\psi_3(n)}} \tag{5.7}$$

We have $\frac{\partial L(n)}{\partial C(n)} < 0$ if (4.7) holds and we get

$$\psi_3(n) < \frac{a(C(n))\left(\alpha_6 + \eta_3 - \frac{\delta_1{}^2 \mathrm{hM(n)}}{(1+h\delta_1 \mathrm{C(n)})^2}\right)}{\frac{da(C(n))}{dC}} \text{for} \alpha_6 + \eta_3 > \frac{\delta_1{}^2 \mathrm{hM(n)}}{(1+h\delta_1 \mathrm{C(n)})^2} \tag{5.8}$$

The inequality in (5.8) shows that the strong Allee effect of class C is not only dependent on the logistic rate at discrete-time and the death rate caused by infection. The rate of action of the police personals increases and has a control role if the is no infection transmission from class C to class M. Professional control mechanisms stabilize the Allee effect so that class C decreases and extinct.

Theorem 5.1. *If* $\limsup_{t\to\infty} I(t) < E_0$, *then* $\lim_{t\to\infty} I(t) = 0$.

Proof. *Assume on the contrary that* $\lim_{t\to\infty} I(t) = \chi_1 > 0$. *The definition of* $\limsup_{t\to\infty} I(t)$ *implies that for any* $\varpi > 0$ *with*

$$0 < \varpi < E_0 - \limsup_{t\to\infty} I(t),$$

there is $t_\varpi > 0$ such that
$I(t) < \limsup_{t\to\infty} I(t) + \varpi$ for $t > t_\varpi$.
For $t > t_\varpi$ and $n \le t < n+1$, we obtain

$$I(t) = I(0)exp\left(\int_0^t \frac{1}{E_0}(K_2 - \alpha_3 I(s) - (\alpha_4 + \theta + \eta_2)\mathrm{I(n)} + \beta_1(1-\varepsilon_1)S(n) + \beta_2(1-\varepsilon_2)M(n))(I(n) - E_0)ds \right)$$

$$< I(0)exp\left(\int_0^t -(E_0 - \bar{I}_3 + \varpi)ds \right) < I(0)exp\left(-\left(E_0 - \bar{I}_3 + \varpi\right)t \right)$$

as $t \to \infty$, where we obtain a contradiction.

Theorem 5.2. *If* $\limsup_{t\to\infty} C(t) < E_1$, *then* $\lim_{t\to\infty} C(t) = 0$.

Proof. *The proof is similar to that of Theorem* 5.1.

Conclusion

We establish the outbreak of COVID-19 and the control mechanisms such as health organizations and the police supplements to control the pandemic spread and protect the susceptible class from the infection.

At first, we gave brief information about *Coronaviridae* and the host transmissions from animal-to-human and then from human-to-human. We emphasized that all human coronaviruses are animal origin, and the spread was seen endemic. The difference between the other coronaviruses and the novel coronavirus COVID-19 is that this *beta-coronavirus* has both human-origin coronavirus (SARS-CoV Tor2) and bat-origin coronavirus (bat-SL-CoVZC45) which spread pandemic

worldwide. We noticed that the infection cases or the death rates are higher than in China (Wuhan), while the spread of the infection started in December 2019 in this area. A united control mechanism was expected from civilians worldwide to stop the spread. However, it was seen that some countries were not prepared against any biological attack, whether it is virus or bacteria origin. Therefore, we established our model as a system of differential equations with piecewise constant arguments that is divided into five sub-classes;

- the susceptible class S,
- the infected class I, that does not know they are infected since specific symptoms do not appear or they have only a host role,
- the infected class C that knows they are infected because of some symptoms such as respiratory and intestinal infections, including fever, dizziness, and cough, appeared.
- the hospitalized people in a quarantined institute as H
- the police organization M to guide and control the panic of civilians and to support the health institutes in transporting the infected civilians.

In section 3, we obtained that the disease-free equilibrium point is stable if there are no transmission contacts between the susceptible civilians and infected groups. Moreover, we get that the rate of action of Holling Type II is higher than the interaction rate of the infected classes to the class of M. The results in Theorem 3.1 show that for any negligence of Sas well as M in '*healthy practices*,' both non-infected classes could reach to a non-hyperbolic behavior. The duties should have continuity in the management and control of civilians. In Theorem 3.1/(ii), we emphasize that the main problem occurs in health protections if it is *not continuous*. Figs. 1–4 show the numerical simulations that were considered in Theorem 3.1. The graphs represented the stability of a non-infected population class of S and M, were it was assumed that there is no transmission risk between the susceptible class and the infected compartment.

From Theorem 3.3, we obtained that the apocalypse case occurred when the growth rate of the susceptible class becomes less than the infectious transmission rate from class I and C. This scenario is considered for a case when the contribution of civilians and organizations are weak. Another scenario for Λ_2 was the non-hyperbolic behavior, where $R_0 > 1$. We emphasized in this part that the basic reproduction number is depended on the logistic rates of the infected class C and the hospitalized class H for both discrete and continuous times, respectively. Further, in this scenario, the susceptible class is still not active in keeping the rules, and they continue the have a host role in the spread of COVID-19. Fig. 5 presented the dynamical structure that shows a dramatic increase of infected individuals from both civilians and police personals. The population rate per year for the susceptible class decreases since, in this apocalypse scenario, we assume that it is also difficult to have a non-infected offspring. Thus, the susceptible class and the police are not aware and educated for a pandemic phenomena.

Finally we considered, at last, the local stability of the co-existing equilibrium point Λ_3. We assumed that intensive and continuous information of health organizations would reach to a fruitful awareness of civilian in recognizing the symptoms of the virus COVID-19. Another vital organization was the police that is only responsible for controlling the panic of the civilians but shows now different roles in this pandemic scenario. Here, we assumed that this organization is well educated in infection cases and also knows to detect and protect the civilians as well as themselves. However, still, we keep the possibility that the police might be infected from the class who do not know they are infected. Fig. 6 we demonstrated the bifurcation diagram of the susceptible class S, the group whose members do not know they are infected, and a class of the police personals according to the results of Theorem 3.5. We noticed that the increase of the screening rate will decrease the carrying capacity of the infected group who do not know they are infected. This I class is the most dangerous since they show host roles in the *human-to-human* transmission.

In section 4, we obtained that semi-cycle solutions approach the co-existing equilibrium pint. Moreover, it is obtained that the screening rate $\theta$and the average time of action δ are vital to control parameters for both health and police organizations in this pandemic spread. We proved in Theorem 4.1. that class I and class Cshow a dominant role on the carrying capacities of the susceptible and the police population. The non-periodic behavior is controlled by the interaction rates of class I and C. Moreover, we obtained that global stability is depended on the harvest rate of each population class and their equilibrium points. In Theorem 4.6, we obtained conditions for a positive oscillatory solution of a semi-cyle behavior with a length at most two. We see that in all the population classes, the logistic rate at a continuous-time has a dominant role in both the logistic rate at discrete-time and the death rate (natural or infection). Additionally, class I oscillates if the screening rate is less than the logistic rate but still exists.

Later in Section 5, we incorporated the Allee function at a discrete-time t for both class I and class C. We analyzed the strong Allee effect. We obtained essential control parameters to support the Allee functions in stabilizing the effect of the spread. Thus, for the Allee function of class I, we got that the screening rate, the death rate caused by infection cases, and the logistic rate at discrete-time are essential to control parameters to support the Allee function. While the Allee function of class C is not only dependent on the logistic rate at discrete-time and the death rate caused by infection, it also showed the vital role of the rate of action of the police personals.

In the end, numerical simulations, along with graphical illustrations, are presented to examine the validity of our theoretical findings. For the co-existing equilibrium case, we obtained a dramatic and disquieting point that shows a repeated form of the pandemic spread faster and appears the same time worldwide. Therefore, the responsible control organizations in this pandemic spread should be careful of this infection case and needs to continue to obstruct any spread of the novel coronavirus in the future.

CRediT authorship contribution statement

Fatma Bozkurt: Conceptualization, Methodology, Investigation, Visualization, Writing - original draft, Supervision. **Ali Yousef:** Conceptualization, Methodology, Investigation, Visualization, Writing - original draft, Supervision. **Thabet Abdeljawad:** Conceptualization, Methodology, Investigation, Visualization, Writing - original draft, Supervision.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Fehr AR, Perlman S. Coronaviruses: an overview of their replication and pathogenesis. Methods Mol Biol 2015;1282:1–12.
[2] Woo PCY, Lau SKP, Huang Yi, Yuen K-Y. Coronavirus diversity, phylogeny and interspecies jumping. Exp Biol Med (Maywood) 2009;234(10):1117–27.
[3] Su S, et al. Epidemiology, genetic recombination, and pathogenesis of coronaviruses. Trends Microbiol 2016;24:490–502.
[4] Forni D, Cagliari R, Clerici M, Sironi M. Molecular evolution of human coronavirus genomes. Trends Microbiol 2017;25:35–48.
[5] Howley PM, Knipe DM, Whelan S. Fields of virology: emergency viruses. China: Wolters Kluwer; 2020.
[6] Yousef A, Bozkurt F, Abdeljawad T. Qualitative analysis of a fractional pandemic spread model of the novel coronavirus (COVID-19). Computers, Mater Continua 2020;66(1):843–69.
[7] Dane J, Allali K, Hammouch Z. Mathematical analysis of a fractional differential model of HBV infection with antibody immune reponse. Chaos, Soliton Fractals 2020;136:1–6.

[8] Zaki AM, van Boheemen S, Bestebroer TM, Osterhaus AD, Fouchier RA. Isolation of a novel coronavirus from a man with pneumonia in SA. N Engl J Med 2012;367:1814–20.
[9] Wu F, et al. A new coronavirus associated with human respiratory disease in China. Nature 2020;1–10. https://doi.org/10.1038/s41586-02-2008-3.
[10] Lofgren ET, Collins KM, Smith TC, Cartwright RA. Equations of the end: teaching mathematical modeling using the zombie apocalypse. J Microbiol Biol Educ 2016;17(1):137–42.
[11] World Health Organization, Novel Coronavirus (2019-nCoV) Situation Reports, https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports/.
[12] Fatmawati MA, Khan E, Banyah Z, Hammouch EMS. A mathematical model of tumerculosis (TB) transmission with children and adult groups: a fractional model. AIMS Mathematics 2020;5(4):2813–42.
[13] Fend Z, Thieme HR. Recurrent outbreaks of childhood diseases revised: the impact of isolation. Math Biosci 1995;32:93–130.
[14] Liu X, Xiao D. Complex dynamic behaviors of a discrete-time predator–prey system. Chaos, Solitons Fractals 2007;32(1):80–94.
[15] Gopalsamy K, Liu P. Persistence, and global stability in a population model. J Math Anal Appl 1998;224:59–80.
[16] Bozkurt F, Ozturk I. A population model of two-strains tumors with piecewise constant arguments. Kuwait J Sci 2015;42(2):1–13.
[17] Cooke KL, Györi I. Numerical approximation of the solutions of delay differential equations on an infinite interval using piecewise constant arguments. Comp Math Appl 1994;28:81–92.
[18] Bozkurt F, Yousef A. Neimark-Sacker bifurcation of a chemotherapy treatment for glioblastoma multiform (GBM). Adv Differ Eq 2019;397(1):1–25.
[19] Bozkurt F. Modeling a tumor growth with piecewise constant arguments. Discr Dyn Nat Soc 2013;2013:1–8.
[20] Liu P, Gopalsamy K. Global stability and chaos in a population model with piecewise constant arguments. Appl Math Comput 1999;101(1):63–88.
[21] SO J-H, YU JS. Global stability in a logistic equation with piecewise constant arguments. Hokkaido Math J 1995;24(2):269–86.
[22] Kulenovic MRS, Ladas G, Prokup NR. A rational difference equation. Appl Math Comput 2001;41:671–8.
[23] Li X, Mou C, Niu W, Wang D. Stability analysis for discrete biological models using algebraic methods. Math Comput Sci 2011;5:247–62.
[24] Verhulst PF. Notice Sur la loi que la population pursuit das son avertissement. Correspondance Mathemat Phys 1838;10:113–21.
[25] Courchamp F, Berec L, Gascoigne J. Allee effects in ecology and conservation. Oxford: Oxford University Press; 2008.
[26] Bozkurt F, Yousef A. Flip bifurcation and stability analysis of a fractional-order population dynamics with Allee effect. J Interdiscip Mathem 2019;22(6):1009–29.
[27] Allee WC. Animal aggregations: a study in general sociology. Chicago, IL: University of Chicago Press; 1931.